8-28-2021

Growth, Maturation, and Weight Status: Insights from a Longitudinal Cohort of Nebraska Youth

John William Masker
University of Nebraska at Kearney, jmasker12@gmail.com

GROWTH, MATURATION, AND WEIGHT STATUS: INSIGHTS FROM A LONGITUDINAL COHORT OF NEBRASKA YOUTH

A Thesis

Presented to the

Graduate Faculty of the Kinesiology and Sports Sciences

Department and the Faculty of the Graduate College

University of Nebraska

In Partial Fulfillment

Of the Requirements for the Degree

Masters of Arts in Education

University of Nebraska at Kearney

by

John W. Masker

July 2021

THESIS ACCEPTANCE

Acceptance for the faculty of the Graduate College, University of Nebraska, in partial fulfillment of the requirements for the degree Master's of Arts in Education, University of Nebraska at Kearney.

Supervisory Committee

Name	Signature	Department
Kate Heelan		KSS
Joe Eisenmann		KSS
Todd Bartee		KSS
Jennie Hill		UNMC
Bryce Abbey		KSS

6/29/21

Supervisory Committee Co-Chair

6/29/21

Supervisory Committee Co-Chair

ABSTRACT

Pediatric obesity continues to be a major public health concern in the United States, with well-known short-term and long-term consequences. In efforts to combat pediatric obesity and identify children at high-risk for potential health problems, physicians and health professionals widely practice screening and classifying weight status using age-and-sex-specific body mass index (BMI) percentiles. However, some studies suggest the use of BMI for establishing weight status in relation to health risk in youth is problematic, especially during the period of the adolescent growth spurt. More importantly, maturation-related misclassification may result in overestimations of overweight prevalence among early-maturing youth, and underestimations of overweight prevalence among later-maturing youth.

Longitudinal data from 646 youth whose body mass and stature were measured from ages 8-14 during school health screenings were used for analysis. Age-and-sex specific BMI percentiles were calculated, and weight status was determined based on CDC growth charts. Height velocities (i.e. growth rates) were calculated to determine somatic maturity (biological age) based on age at peak height velocity. Overall, growth in stature, body mass, and BMI was described amongst weight status categories, while weight categorization was compared using standard procedures versus an approach adjusted for maturation.

As expected, children get taller and heavier with age, and significant differences in growth rate exist according to weight classification. Overall, 8.5% of children were re-classified into a lower weight category, with 22 (30.6%) overweight or obese boys and 14

(20.3%) overweight or obese girls reclassified into a lower weight category when adjusting for maturation.

Children grow and mature at different rates, and while the overall effects of maturational adjustment are relatively small, it should be considered when assessing adolescents in particular. Weight status assessments should be modified during the age range when maturational events occur.

ACKNOWLEDGEMENTS

First and foremost, I want to extend my most sincere appreciation to my advisor, Dr. Kate Heelan. During a time of uncertainty, you provided an incredible opportunity to better my education, develop professionally, and become a better man. The research assistant position was truly a prayer answered, and I am forever grateful. More importantly, your patience and leadership has allowed me to successfully complete this thesis. I also want to extend my most sincere appreciation to Dr. Joe Eisenmann. During your time here at UNK, you have not only been a great mentor, but a true friend. You have shown me how to work extremely hard, yet take a step back from work and enjoy life. I will forever cherish our times shooting hoops or playing catch. I also want to extend my thanks to Dr. Todd Bartee, Dr. Bryce Abbey, and Dr. Jennie Hill for serving on my committee and providing invaluable insight through this project. A thank you also goes out to the Physical Activity and Wellness Laboratory staff for all your friendship and support.

A very special thank you goes out to my parents Ken and Ann Masker. I am forever in debt to all the opportunities and experiences that you have provided in my life. You both have always placed your three boys first, and without your sacrifice, love, and guidance, we would not be in the positions we are today. Throughout my upbringing, you both have instilled strong morals and values that I will continue to hold close to my heart as I go through life. I love you Mom and Dad.

TABLE OF CONTENTS

THESIS ACCEPTANCE ii

ABSTRACT iii

ACKNOWLEDGEMENTS v

CHAPTERS

I. Introduction 1

II. Review of Literature 5
- Obesity Prevalence 5
- Anthropometry 7
- Growth Charts (CDC) 8
- Childhood Growth and Maturation 10
- Growth and Maturation of Obese Children 14
- The Problem with Anthropometry 16
- Purpose and Aims of the Study 18

III. Methodology 20
- Participants and Study Sites 20
- Anthropometry 20
- Calculation of Growth Rates and Determination of Peak Height Velocity 21
- Calculation of Biological Age 23
- Classification of Weight Status Based on Maturity 24
- Describing Growth Characteristics 24
- Statistical Analysis 25

IV. Results 27

V. Discussion 31

VI. Conclusion 44

REFERENCES 45

LIST OF TABLES

Table 1. Descriptive Statistics for Age, Stature, Body Mass, and BMI at each age. 61

Table 2. Average age at peak height velocity, height velocity, and biological age by maturity status. 62

Table 3. Comparison of weight classification according to standard CDC classification based on chronological age and biological age. 63

LIST OF FIGURES

Figure 1. Prevalence of Overweight & Obesity by Age. 64

Figure 2. Body Size of Midwestern Nebraska Children 65

2A. Boys 65

2B. Girls 66

Figure 3. Growth Velocity of Midwestern Nebraska Children 67

3A. Boys 67

3B. Girls 68

CHAPTER 1

Introduction

Obesity continues to be a major public health issue in the United States [1], given that it increases chronic disease morbidity [2] and mortality [3], including cardiovascular disease [4], type 2 diabetes [5], and certain cancers [6]. The recent estimate of the adult obesity prevalence was 42.4% in 2018 [7]. Obesity now accounts for 18% of deaths among Americans ages 40-85 years [8], which means it is comparable in severity to cigarette smoking as a public health hazard [9]. In addition to the serious health consequences, there are also substantial economic losses associated with obesity [10]. Economic losses arise from direct costs (e.g., increased medical spending per capita through outpatient and inpatient health services), as well as indirect costs (e.g., absenteeism, presenteeism, and higher insurance premiums) [11, 12]. In 2008, annual obesity-related medical costs totaled an estimated $147 billion [13]. The magnitude of this economic and societal burden, alongside the huge toll that excess weight takes on health and well-being have raised awareness that individuals, communities, and states must increase efforts to reverse the current trends in obesity.

Obesity not only affects adults, but children are also significantly impacted. Since 1990, the incidence of pediatric obesity has increased by 85% [14], with a current prevalence rate of 19.3% for 2-19 year-old children and adolescents [15]. Childhood obesity tracks into adulthood [16-20] and is associated with the premature development of chronic diseases (i.e., cardiovascular disease, diabetes) [21-24]. For most chronic diseases resulting from obesity, the risks depend partly on the age of onset and the

duration. Thus, obese children are more likely to remain obese into adulthood [17, 19, 25, 26], which increases the risk of premature death and disability [27]. Clearly, obese children and adolescents suffer from both short-term and long-term health consequences.

The American Academy of Pediatrics emphasizes the importance of routine assessment and recognition of excessive weight gain relative to linear growth in stature throughout childhood [28]. To prevent pediatric obesity and identify children at increased risk, many physicians and health professionals practice screening and classifying weight status [29]. Weight status classification is determined through simple anthropometry, which is the assessment of stature (i.e., standing height), and body mass (i.e., weight). These measures are then used to calculate the body mass index (BMI) by dividing body mass in kilograms by the square of stature in meters (kg/m^2) [30]. A child's weight status is then determined according to age-and-sex specific percentiles for BMI.

However, it is important to note that the BMI is not a diagnostic tool [31] and some experts acknowledge that the BMI is not a perfect measure of excess adiposity [32]. Due to age-related growth patterns and sex differences in body fat, there are precautions associated with using the BMI in children and adolescents [31, 33, 34]. BMI may not have the same relationship with body fatness in all demographics, and maturation-related misclassification may result in overestimations of overweight prevalence rates among early maturing youth, and underestimations among later maturing youth [35, 36]. Research suggests the use of BMI for establishing weight status in relation to health risk in youth is problematic, especially during the period of adolescence, and specifically, peak height velocity when stature changes dynamically [37]. The problems associated

with using BMI during adolescence were recently highlighted in a study by Gillison et al., [38] where weight classification was adjusted based on maturity status. The results indicated 32% and 15% of overweight girls and boys, respectively, were classified as overweight based on chronological age, but normal weight based on biological age. In addition, 11% of girls and 8% of boys classified as obese by BMI percentile were reclassified as overweight when maturity status was accounted for. Overall, this study found early maturing youth were almost five times more likely to be reclassified from overweight to normal weight than the 'on-time' maturers [38].

The pattern of anthropometric changes with age is generally similar in all children, but the size attained at a given age and the timing of maturational events vary considerably from child to child due to various factors. As a result, BMI changes substantially as children age due to stature and body mass changes during growth, development, and maturation [39-43]. There are normal age-and-maturity related increases in body fat during puberty; thus, it is likely that early maturing youth can be a normal weight at a higher BMI than their later maturing peers [38]. Since early-maturing youth may be at an increased risk of being misclassified for overweight, which in turn could be detrimental to their social and emotional well-being, they represent a susceptible group with whom health professionals should be particularly careful to minimize the potential unintended consequences of misclassifying weight status.

The purpose of the current investigation is to describe growth and maturity characteristics of 8-14 year-old boys and girls in a longitudinal study from a Midwestern

Nebraska community, with special attention on the role of biological maturity in the classification of weight status.

Primary Aim:

The primary aim of this investigation is to compare the weight categorization of a cohort of children using standard procedures (i.e., comparing BMI to age-and-sex specific CDC reference data) versus an approach adjusted for maturation status (i.e., relative to biological age instead of chronological age).

Secondary Aim:

The secondary aim of this investigation is to determine the growth rate of stature (cm/year), the growth rate of body mass (kg/year), and the growth rate of BMI (kg/m^2/year) among children classified as normal weight, overweight, or obese at age 12 years. Specifically, yearly anthropometric changes for ages 8 to 14 years were calculated and compared between weight classifications.

CHAPTER II

Review of Literature

Obesity Prevalence

Due to the significant increase in adult obesity rates over the last few decades [44], obesity has grown to epidemic levels and has emerged rapidly as a serious public health challenge [1, 45]. Obesity is a complex disease [46] involving an excessive amount of body fat, is largely preventable, and greatly increases the risk of chronic disease morbidity [2] –namely depression [47], cardiovascular disease [4], type two diabetes [5], certain cancers [6], and overall mortality [3, 48]. Globally, overweight and obesity currently affect over a third of the world's population [49, 50]. If trends in obesity remain consistent, by 2030 it is estimated 38% of the world's adult population will be overweight, and another 20% will be obese [51]. Specifically within the United States, projections based on secular trends also indicate over 85% of adults will be overweight or obese by 2030 [52]. More importantly, obesity rates have shown to continually climb among youth [14], as the percentage of children and adolescents affected by obesity has more than tripled in the United States since the 1970's [53]. Furthermore, since 1990, childhood obesity incidence has increased by 85%, with the current prevalence at 19.3%, which translates to 14.4 million 2-19 year-old youths [15]. A closer evaluation of age groupings demonstrates obesity prevalence is approximately 13.4% among 2-5 years, 20.3% among 9-11 years, and 21.2% among youth 12-19 years old [15].

Studies identifying the health risks associated with childhood obesity indicate that many bodily systems are negatively impacted [47, 54-60]. Childhood obesity can result in

hypertension and high cholesterol, which are risk factors for cardiovascular disease [61]. Childhood obesity may result in increased risk of impaired glucose tolerance and insulin resistance, which can progress into type two diabetes [22, 62]. Lastly, childhood obesity also creates risk for a variety of conditions, such as sleep apnea [63], asthma [64, 65], orthopedic complications (e.g., joint problems, musculoskeletal discomfort, mobility impairment) [55], non-alcoholic fatty-liver disease [66], gallstones [67], and gastro-esophageal reflux [68]. It even presents risk for psychological conditions such as depression and low self-esteem, behavioral problems, issues in school, and an overall low self-reported quality of life [47, 59, 69].

An important question to address about obesity in childhood is whether it is a fleeting or persistent health problem. It is acknowledged that children grow and mature at different rates [39-42], and a brief period of obesity may be no reason to worry; however, if obesity does become persistent, that is, firmly established and difficult to reverse, it can lead to the health concerns that manifest in adulthood [70, 71]. Findings from several studies have indicated weight status is established at early ages [72-74]. This means obesity in childhood can be difficult to reverse [20, 75], and children with obesity do not always reach a normal weight status [76]. In three U.S. metropolitan areas, it was found 65% of fifth graders with obesity retained their weight status through tenth grade [77]. Among children with an early onset of obesity, the majority were affected by persistent obesity regardless of sex, race, ethnicity, and socioeconomic status [70]. Obesity with onset during preschool or elementary school years is generally not a transient phenomenon, but one that can be a warning sign for long-term obesity, at least into

adolescence [70]. Evidence suggests a child's growth and maturation play a key role in their body composition and weight status [35, 78-91].

Anthropometry

To prevent pediatric obesity and identify children at increased risk, many physicians practice screening and classifying weight status [29]. Anthropometry is set of standardized techniques for methodically collecting measurements of the body, which involves carefully identifying bodily landmarks, specific subject positioning, and using appropriate instrumentation [37]. Through anthropometry health professionals are able to assess weight status, body composition, and follow growth. Stature and body mass are the two most often used measurements to assess growth. Stature, or standing height, is a linear measurement of the distance from the floor or standing surface to the top of the skull, and is measured in an upright posture without footwear. Body mass is a compound measurement of independently varying tissues like bone, muscle, and adipose, and is measured by a weight scale.

In addition to providing specific information, stature and body mass measurements can be related to one another to form a ratio. Ratios are influenced by two dimensions of the body, and the two dimensions are assumed to change in a linear manner [37]. The ratio between stature and body mass is most often expressed in the form of the body mass index (BMI). Body mass index is calculated by body mass in kilograms, divided by the square of stature in meters (kg/m^2) [30]. BMI is the most commonly accepted index for classifying adiposity in adults [31]. It is important to remember that BMI is a screening tool, and not a diagnostic tool [31], because it is only a

proxy measure of body fatness. Yet, it relates reasonably well to total-body fatness and lean tissue mass of the body measured by skinfold thickness and hydrostatic weighing [92-94].

For children, BMI is age-and-sex specific. BMI changes substantially as children age due to stature and body mass changes during growth, development, and maturation [39-43]. Weight status is determined using age-and-sex specific percentiles for BMI, to express anthropometry relative to other children of the same age and sex. Classifications according to BMI percentiles are determined as underweight (<5th percentile), normal weight (5th-84.9th percentile), overweight (85th-94.9th percentile), and obese (≥95th percentile) [31, 95, 96]. BMI-for-age allows health professionals to plot a measure of stature and body mass with age on the same chart, and is consistent with the index used for adult classification. BMI-for-age can be used continuously from 2 years of age into adulthood and to track body size throughout the life cycle. This is important because BMI-for-age in childhood is a determinant of adulthood BMI [18, 97, 98]. Finally, BMI-for-age compares reasonably well with both weight-for-stature measurements and correlates with subcutaneous and total body fatness in youth [31, 99]. Overall, BMI is a useful but imperfect proxy indicator of fat mass and subsequent health risk [100, 101]; still, its wide use among health professionals stems from its cost effectiveness and practicality.

Growth Charts (CDC)

Through the scientific study of anthropometry, the present day growth charts for the United States were developed with data collected by the National Center for Health

Statistics using five cross-sectional, nationally representative health examination surveys spanning from 1963-1994 [102]. These surveys are known as the National Health and Nutrition Examination Survey (NHANES) conducted by the Centers for Disease Control and Prevention. Supplemental data was also incorporated from various scholarly sources such as the Fels Longitudinal Study, which is the longest running study of growth, maturation, and body composition in a sample of youth [103]. Complex, multistage, and stratified sampling procedures were used in selection to compile a sample which represented the noninstitutionalized civilian population of the United States [102]. A large number of children were included so the extreme percentiles (i.e., 5^{th} and 95^{th}) could be obtained by observation rather than estimation. All surveys consisted of a home interview and a physical examination of stature and body mass. The measurement procedures were consistent with the published recommendations for standardized anthropometric techniques [104], and age was recorded at the time of examination [102].

The data collected from these examinations are referred to as reference data. It is presented in the form of several curves representing different percentiles to fit the range of normal variability among children of the same age and sex. [37]. Data in the form of growth curves are solely a comparison for screening individuals or groups. These data are collected from a representative sample of clinically normal children and adolescents free from overt disease at the time of measurement. From this, the values are not necessarily ideal, normal, desirable, optimal, or the standard. Reference data indicate the statures, body masses, BMI's, and respective percentiles of children at different ages as they currently are, rather than what they should be. Although, while in use, the percentiles are

generally accepted as indicating the normal range of variation. For body mass, the circumstances are somewhat different due to the concern of increasing obesity prevalence in American youth [37]. Stature-for-age, weight-for-age, and BMI-for-age are all useful in evaluating the growth status of individual children, as well as in a sample of children. If a child is followed over time, their position on the growth charts can be noted. In using distance curves, the size attained by a child is evaluated relative to the growth data collected.

Childhood Growth and Maturation

The process of 'growing up' is the major event of young people in the first two decades of life. Growing up includes both physical growth and maturation, alongside cognitive and socio-cultural development. Specifically, biological growth is defined as a multi-factorial process that occurs due to a combination of hyperplasia (i.e., increase in number of cells), hypertrophy (i.e., increase in size of cells), and accretion (i.e., gradual accumulation through hyperplasia and hypertrophy) [105]. Simply stated, growth is the increase in size of the body or its parts. Growth during childhood and adolescence is controlled by many factors like genes and hormones, with a main role of the growth hormone, insulin-like growth factor-1 (IGF-1), thyroid hormones, and sex steroids [106, 107]. Several lines of evidence also support a major role of nutritional status in influencing childhood growth, as clearly supported by the association between short stature and malnutrition [108], as well as tall stature and overnutrition [109].

Maturation is defined as a series of biological events that progress toward and eventually end in a state of full maturity. The concept of maturation is operational, due to

the physiological processes that occur within the body cannot be measured directly. Growth and maturation are two terms that are often used synonymously and it is important to note that they are related, but separate constructs. Every human varies in the level of maturity attained at a given point in time (maturity status at a given age), in timing (when maturational events occur), and in tempo (rate of maturation) [39, 40, 43, 110]. The biological processes of maturation relate chronological time to biological time. Humans use chronological time to assess age, which is simply the number of years, months, and days alive since birth. While biological age is how old the body seems based on a number of physiological factors. A child's rate of biological maturation does not always proceed simultaneously with the calendar. Variation in biological age can be seen within a group of youth of the same chronological age and sex, as some children are biologically ahead of their chronological age, and some are biologically behind. Measurements of maturity vary according to the biological system that is examined. The most commonly used indicators of biological maturity in postnatal growth studies examine the skeletal system, reproductive system, and anthropometric growth patterns [37].

Examining the skeletal system (i.e., skeletal maturation) is suggested to be the best method of assessment for biological maturity [111]. Prenatally all children begin with a skeleton of cartilage, and fully develop a skeleton of bone by early adulthood. The skeletal structure of all humans progresses from cartilage to bone, so both the beginning and end points are known with this method. The skeleton is an ideal gauge of maturity because its maturation spans the entire period of growth. The rate at which the skeleton

progresses from cartilage, to initial bone formation, to shaping of bone, and eventually into adult structure varies among bones within an individual, as well as among individuals [37, 112].

Sexual maturation begins with the sexual differentiation of an embryo, through the end of puberty to full sexual maturity and fertility [37]. Assessing sexual maturity in growth studies is based on secondary sex characteristics, which are external indicators of sexual maturity at a given point in time [113]. These characteristics are breast development and menarche in girls, genital development in boys, and pubic hair for both sexes. Using secondary sex characteristics as indicators of maturity status and progress is limited to the pubertal phase of growth and development; thus, these indicators have limited applicability over the total course of growth [37, 114].

Both skeletal and sexual maturity are fairly invasive and impractical maturity assessment techniques. Because of this, the majority of studies use indicators of visible growth (i.e., somatic maturity/growth of the body). Somatic maturation can be measured using longitudinal anthropometric data that span into adolescence. Growth curves contain several parameters that provide information about maturity. There are two primary parameters, being takeoff, or initiation of the adolescent growth spurt, and peak height velocity, or maximum rate of growth during the spurt [37]. The inflection in the growth curve that signifies the adolescent growth spurt can be used to derive indicators of maturity such as age at onset of growth spurt, and age at peak height velocity (APHV) [108]. APHV is the most commonly used indicator of somatic maturity. Similarly, if

adult height is accessible, or can be estimated, the percentage of adult size attained at different ages throughout growth can also be used as a maturity indicator [115].

Longitudinal data are required to obtain precise information about growth and maturation patterns [116]; therefore, if a child were to be tracked over time, it would be noted the course of childhood growth in both stature and body mass from birth to 18 years of age follows a four-phase pattern. Growth begins with rapid gain in infancy and early childhood, steady gain during middle childhood, another period of rapid gain during adolescence, and finally slow gain until cessation with the attainment of adult size [37, 40, 43]. During infancy and early childhood, or the first three years of life, children tend to change positions on growth charts [117, 118], as the greatest gains in stature and body mass are seen. Crossing percentiles of stature and body mass is common in infancy and early childhood, as it reflects individual differences as each child moves toward their genetically determined growth percentiles.

During middle childhood around the ages of 6-12, growth becomes canalized, meaning following the same trajectory [37]. Children during this time will typically grow around 5-8 centimeters per year and will generally stay in the same percentile range [119]. In addition to stature gains, children will also gain about 4-10 pounds per year, increasing a little more every year from 6-10 years of age [119]. Conversely, steady growth is not seen during adolescence, as a child's position on growth charts often changes because of individual differences in the timing, tempo, and magnitude of the adolescent growth spurt and sexual maturation [37]. Once the spurt is complete, growth gradually slows down until full maturity and adult stature has been reached.

As many know, puberty is the main physiological event in human growth and maturation. Puberty is the transitional period between childhood and adulthood, and the appearance of secondary sex characteristics and the adolescent growth spurt are its most visible manifestations [120]. Puberty begins with the activation of the hypothalamic-pituitary-gonadal axis and ends with the attainment of reproductive capability and the acquisition of adult body composition [121, 122]. Usually there is a slowing of growth rate in stature prior to initiation, or take off, of the adolescent growth spurt [43]. Once youth enter their growth spurt, rates of growth in both stature and body mass accelerate. Typically, pubertal growth consists of a phase of acceleration that will eventually reach a maximum rate, followed by a phase of gradual deceleration, and then finally cessation of growth with the closure of the bone epiphyses [120]. Boys on average tend to be slightly taller and heavier than girls; however, during the early period of the growth spurt, girls are temporarily taller and heavier because of their earlier onset. For girls, their spurt begins around 9 or 10 years of age, peaks around 12, then ceases around 16. For boys, their spurt begins around 10 or 11 years of age, peaks around 14, then ceases around 18 [37]. According to the Centers for Disease Control and Prevention, based on the 50th percentile of youths, the onset of the adolescent growth spurt for girls occurs approximately around 11 years of age, and for boys around 13 years of age [123].

Growth and Maturation of Obese Children

The rising problem of childhood obesity has stimulated a growing interest in the relationship between body composition and the timing and tempo of puberty [124]. Although the biological mechanisms remain poorly understood [125], there is a growing

body of literature which seems to suggest a complex bidirectional causal relationship between early onset of puberty and obesity in females. A number of studies have shown that during pre-pubertal years, obese children present a higher growth velocity and accelerated bone age compared to lean subjects [126, 127].

Specifically for girls, the 'critical weight hypothesis' developed by Frisch (1970) states a certain amount or percentage of body fat is needed for the onset of menarche [82]. Most recently, data from a longitudinal school-based nationally representative sample in the United States found early maturing girls were almost twice as likely to be overweight than average-maturing girls [78]. It is purported that subcutaneous fat tissue acts as a secondary hormonal gland and adipose tissue influences the synthesis and release of hormones [81, 82, 128]. Rapid early weight gain has been linked to elevated insulin-like-growth factor-1 concentrations and insulin resistance, elevated adrenal androgen concentrations, exaggerated adrenarche, obesity, and consequently elevated concentration of hormones such as leptin [129]. It has been suggested that these factors could promote the activity of the gonadotropin-releasing hormone pulse generator, thereby influencing the timing of puberty, specifically menarche [130]. Other studies in this field have described similar results showing the additive effects of pre- and postnatal growth on timing of menarche [131-133].

In contrast, the relevance of early life risk factors for pubertal timing in boys in unclear, mainly because there is no major pubertal milestone. Age at menarche is one of the most significant pubertal milestones in a woman's life, and in comparison there is no corresponding event in boys. In fact, European studies have shown obesity is associated

with earlier puberty and voice break [134-138], while American studies have reported the opposite in that obesity is associated with delayed puberty in boys [139-142]. However, a large study following a birth cohort of approximately 7000 children from 1997, has suggested that infant weight growth may be associated with earlier pubertal onset even in boys [91, 143].

The Problem with Anthropometry

No objective measurement of anthropometry is perfect and most measures of a child's weight status fail to account for individual differences. Currently there are a multitude of BMI surveillance and screening programs utilized worldwide and provide data for monitoring population-level obesity; however, providing data as feedback has resulted in little retainment of weight management support [144], and has even isolated parents who were either angry or disbelieving of the information provided [144-146]. Research seeking the source of parental anger and rejection of objective anthropometry of their children has found that parents fear the risk of harm to their child's health and well-being is greater from labelling them as overweight (e.g., in undermining self-esteem and triggering eating disorder symptomology) than it is from physically being overweight [144-148]. More importantly, parents have argued the validity of BMI percentile assessments when children approach the stage of puberty [145]. The main argument posed by parents is that judgements of weight status which do not account for relative differences in pubertal development are not valid [145].

There is reliable evidence that an earlier onset of puberty is associated with greater risk of obesity, and thus the two may be somewhat conflated. Researchers have

also raised the question of whether it is appropriate to judge weight status based on BMI during puberty, when some increase in body fat is normal and healthy [149, 150]. Additionally the use of BMI-for-age for establishing weight status in relation to health risk in children has shown to be problematic, especially during the period of peak height velocity [38]. Work with DXA (dual-energy x-ray absorptiometry) scans to provide accurate measurements of body fat demonstrate a considerable normative increase in fat mass around the trunk region in both boys and girls in the lead up to the period of peak height velocity (i.e. the main event of puberty) regardless of physical activity and dietary intake [151, 152]. In summary, BMI for-age does not account for maturational status, which is a very critical shortcoming and should be considered when using BMI percentiles to screen children's weight status and possibly incorrectly label them as overweight or obese.

In order to determine the impact of adjustment for maturity status on population estimates of obesity, and the misclassification of risk for children who are advanced in maturity, Gillison et al. examined a cohort of 9-11 year old youth [38]. Comparisons were made of BMI against chronological age-matched and sex-matched reference charts, versus when estimated using reference charts matched to their biological age [38]. Maturity status was calculated by non-invasive means using the Khamis-Roche method [115]. The Khamis-Roche equation is based on the percentage of predicted adult stature that a child had attained at time of measurement. This method showed that among youth of the same age, those who were closer to their mature/adult stature were more advanced in biological maturity. This equation predicted adult stature from a child's age, stature,

body mass, and mid-stature of the biological parents [115]. Their results showed when BMI percentile was adjusted for maturation, there was a decrease in the proportion of youth classified as overweight (12.5% to 10.6%) and obese (12.5% to 11.8%). Overall, 11% of overweight or obese boys and 22% of overweight or obese girls were re-classified into a lower weight category [38].

Purpose and Aims of the Study

Since early maturing youth may be at an increased risk of misclassified for overweight, which in turn could be detrimental to a child's social and emotional well-being, they represent a susceptible group with whom health professionals should be particularly careful to minimize the potential unintended consequences of misclassifying weight status. Therefore, purpose of the current investigation is to describe growth and maturity characteristics of 8-14 year-old boys and girls in a longitudinal study from a Midwestern Nebraska community, with special attention on the role of biological maturity in the classification of weight status.

Primary Aim:

The primary aim of this investigation is to compare the weight categorization of a cohort of children using standard procedures (i.e., comparing BMI to age-and-sex specific CDC reference data) versus an approach adjusted for maturation status (i.e., relative to biological age instead of chronological age).

Secondary Aim:

The secondary aim of this investigation is to determine the growth rate of stature (cm/year), the growth rate of body mass (kg/year), and the growth rate of BMI

(kg/m^2/year) among children classified as normal weight, overweight, or obese at age 12 years. Specifically, yearly anthropometric changes for ages 8 to 14 years were calculated and compared between weight classifications.

CHAPTER III

Methodology

Participants and Study Sites

Youth in kindergarten through 8th grade from ten public elementary and two middle schools in Kearney, Nebraska (population ~33,000) participated in an annual health screening of body mass and stature between 2006-2020. The child's school associated identification number, date of birth, and date of screening were also collected and entered into a secure UNK BMI Reporter Web Application. Inclusion criteria for the current analysis included longitudinal data from 8-9 years of age through 13-14 years of age. De-identifiable data were exported from the web-based application for statistical analysis. This project was approved by the University of Nebraska at Kearney's Institutional Review Board (IRB) prior to program initiation (IRB number: #090612-1.)

Anthropometry

Body mass and stature measurements took place in private rooms within each school as part of the annual school health screenings. Body mass was measured to the nearest 0.1 lb. using a Befour Platform Scale (PS6600, Befour Inc., Saukville WI). Stature was measured to the nearest 0.25 inch using a seca Portable Stadiometer (Model 213, seca North America Inc., Chino CA). Both measurements were obtained without shoes and excess clothing and accessories. Upon measurement and data entry, the UNK BMI Reporter Web Application calculated a chronological age (observation date – date of birth) to the nearest 0.1 year, a BMI (kg/m^2), and an age-and-sex-specific BMI percentile for each child based on CDC growth charts [102]. Weight status was

determined based on each child's respective BMI percentile being either normal weight (5^{th}-84.9^{th} percentile), overweight (85^{th}-94.9^{th} percentile), or obese ($\geq 95^{th}$ percentile) [102]. The UNK BMI Reporter Web Application tracks students based on their school associated identification number, which allowed for an export of participants with data from ages 8-14 years. Yearly exported data included school identification number, chronological age, sex, body mass, stature, BMI, BMI percentile, and date of screening. The investigators then merged the yearly data by identification number.

Calculation of Growth Rates and Determination of Peak Height Velocity

The primary aim of this investigation was to compare the weight categorization of a cohort of children using standard procedures (i.e., comparing BMI to age-and-sex specific CDC reference data) versus an approach adjusted for maturation status (i.e., relative to biological age). In order to adjust BMI for maturity status, the biological age of the individual child needed to be determined. This was a two-step processes: first determining the age at peak height velocity (APHV), then calculating the biological age.

To determine the APHV, or the age at which maximal rate of growth occurs during adolescence, annual growth rates of height velocities (cm/year) were calculated as outlined in the Example Table and described here: To calculate height velocities, approximate yearly stature increments were calculated by subtracting consecutive measurements (e.g.164.0 cm – 162.9 cm = 1.1 cm increment). Then, the intervals (days) between consecutive measurements were calculated using the same method (e.g. 6-12-09 minus 10-8-08 = 257 days). To derive a height velocity, stature increments were divided by the intervals and multiplied by 365 days (e.g. 1.1 cm / 257 days *365 days/year = 1.56

cm/year). It is important to mention all participants' true chronological ages were recorded at each measurement session, and mid-age (the midpoint between the age of consecutive annual screenings) was also calculated to align with the height velocity given that growth is a continuous, dynamic process between observation dates.

Using the calculated height velocities, growth curves were constructed for each individual by graphing the mid-age on the x-axis (years), and height velocity on the y-axis (cm/year). The growth velocity curves were fitted using a nonparametric cubic spline interpolation to assess peak height velocity (PHV) and the corresponding APHV [153]. Given the age range of the subjects and that girls mature about two years in advance of boys, investigators were able to derive these parameters in every girl, but not in every boy. The spline interpolation was only able to ascertain APHV in the early maturing and a small number of average maturing boys due to the later onset of pubertal events in boys. The interpolation was not able to ascertain APHV in most average and all late maturing male participant cases; therefore, only a select sample of early and average maturing boys were included for analysis. The same growth velocity procedure was done for body mass and BMI.

Example of Height Velocity Calculations

Date	Chronological Age (years)	Stature (cm)	Mid-age (years)	Increment (cm)	Interval (days)	1-yr height velocity (cm/year)
10-8-08	13.46	162.9	-	-	-	-
6-12-09	14.17	164.0	13.8	1.1	257	1.56
7-26-10	15.29	174.1	14.7	10.1	409	9.01
8-15-11	16.34	179.9	15.8	5.8	385	5.50
7-17-12	17.26	181.3	16.8	1.4	336	1.52

Calculation of Biological Age

To determine a biological age, the APHV was compared with nationally representative reference standards [154], which indicated an average APHV of 11.49 ± 1.17 years for girls and 13.57 ± 1.11 years in boys. Using the participant APHV, an estimated biological age was determined as: estimated biological age = chronological age + (average APHV – calculated APHV) [36]. This equation estimates each participant's biological age as their chronological age plus the differences between the mean APHV in the nationally representative samples and the ascertained individual APHV.

Female participants in the current study that attained PHV prior to 10.32 years of age (11.49 – 1.17 = 10.32 years) were deemed an early maturer, between 10.32-12.66 years an average maturer, and after 12.66 years a late maturer (11.49 + 1.17 = 12.66). For

male participants, attaining a PHV prior to 12.46 years was deemed an early maturer and between 12.46-14.68 years an average maturer, Again, because of the age range in the current study, several average and all late maturing boys were not able to be classified.

Classification of Weight Status Based on Maturity

Based on the biological age for each participant, it was determined how many participants were misclassified when their weight status is referenced using biological age instead of chronological age. To exemplify this concept, a chronologically aged 12.0 year old, overweight girl has reached PHV at 10.3 years. From this, it is known that she is an early maturer and roughly 1.19 years ahead (biologically) of her chronological age based on the methodology described above [12.0 + (11.49 – 10.3) = 13.19]. The biological age of this girl is 13.19 years and when her weight status is referenced by biological age, it is found that she is normal weight status instead of overweight.

Describing Growth Characteristics

The secondary aim of this investigation was to determine both the growth status and growth rates for stature, body mass, and BMI gain among children classified as normal weight, overweight, or obese at age 12 years. Yearly stature, body mass, and BMI velocities from ages 8 to 14 were calculated using the same procedure for the individual height velocities required in the primary aim. Then, the anthropometric velocities were averaged according to sex and weight classification. The yearly average velocities were then compared between weight classifications. Growth curves illustrating the average anthropometric velocities and the differences in rate of stature, body mass, and BMI gain between weight classifications over time were constructed as a visual. Additionally, to

gain a more comprehensive understanding of growth in the sample, distance curves illustrating the means for stature, body mass, and BMI status over time were plotted for each sex and weight class.

Statistical Analysis

Microsoft Excel (2016, Redmond WA) and Statistical Analysis Software for Windows (SAS Version 9.4, 2021, Cary NC) were utilized mutually for all calculations, graphing, and any subsequent analysis. Descriptive statistics were calculated by sex and chronological age.

Primary Aim

BMI percentiles were calculated twice using coding from the Centers for Disease Control and Prevention in SAS. They were calculated using chronological age and biological age. Weight status was then determined based on BMI percentile categories being either normal weight (5^{th}-84.9^{th} percentile), overweight (85^{th}-94.9^{th} percentile), or obese ($\geq 95^{th}$ percentile). Investigators assigned a value to indicate misclassification (i.e., -1 indicated a lower weight category, 0 indicated same weight category, and +1 indicated a higher weight category). It was examined whether the classification of weight status differed significantly when using chronological versus biological age using a Chi Square (χ^2) test. A p-value of <0.05 indicated statistical significance.

Secondary Aim

In order to evaluate the secondary aim; stature, body mass, and BMI velocities were compared using a general linear regression model with post hoc analysis to

determine statistical significance ($p<0.05$) in growth velocities among the different weight categories and sex at each age.

CHAPTER IV

Results

Descriptive statistics for anthropometric characteristics for the total sample (N=646) of 8-14 year old children by age and sex (n=358 boys, n=288 girls) are shown in Table 1. Nine children were classified as underweight (BMI <5th percentile) were omitted from the sample due to underrepresentation, and also because they were not a main focus for this study. As expected, boys and girls gain in height, body mass, and BMI with age. The average height across all ages for boys approximates the 70th percentile and weight and BMI approximates the 80th percentile. The average height across all ages for girls approximates the 60th percentile and weight and BMI approximates the 75th percentile. In general, the prevalence of combined overweight and obesity ranged from 26-36% across ages for both sexes (Figure 1). Boys consistently had a higher total prevalence of overweight and obesity compared to girls, with the highest prevalence (36%) at age 12 years. In girls, the prevalence of combined overweight and obesity was about 25-30% with the highest prevalence occurring at age 14 years (31%). Small fluctuations of overweight and obesity prevalence were observed through time with no clear patterns or trends.

The age-specific means for stature, body mass, and BMI according to weight status (normal weight, overweight, and obese) are plotted relative to selected CDC reference values in Figures 2A & 2B. On average, obese children were slightly taller and heavier than the overweight and normal weight children for both sexes, and they were consistently above the age-specific 95th percentile for body mass and BMI. Overweight

children remained between the 75th-94th percentiles for all anthropometric measurements, with the exception of stature for girls. The mean statures for obese and overweight girls fell approximately to the 50th percentile by age 14 years to match that of normal weight girls. Whereas for boys, the mean statures maintained a steady trajectory, and remained above 50th percentile by age 14 years for all weight categories. Normal weight children approximated the 50th percentile for height, body mass and BMI across the age range. Average statures between weight classes for girls differed by 2 cm for girls, and 3 cm for boys at age 14 years.

Mean growth rates for height, body mass, and BMI are shown in Figures 3A & 3B. Obese children experienced the greatest rate of weight gain, while normal weight children experienced the smallest rate of weight gain. Obese children from ages 8-11 years experienced the greatest rate of gain in height; however, this gain decreased through ages 12-14 years. All children experienced similar rates of growth for height, weight, and BMI by age 14 years. There were significant differences ($p<0.05$) in growth rates for height, body mass, and BMI, among the different weight categories for both sexes. The significant differences between weight categories at different ages further emphasize the variability in growth and maturation among children, especially during puberty and the adolescent spurt (Figures 3A & 3B).

The primary aim of the current investigation is to compare the weight categorization using standard procedures (i.e., comparing BMI to age-and-sex specific CDC reference data) versus an approach adjusted for maturation status (i.e., matching relative to biological age). In this analysis, 213 (33% of original sample) children (176

boys, 37 girls) were further omitted from the sample for a multitude of reasons. A large number of males (n=156, 24% of original sample) were discarded due to a later onset of the adolescent growth spurt and the inability to ascertain APHV; thus not able to calculate biological age. Other participants (n=57, 9% of original sample) were discarded due to the inability to distinguish a clear PHV. Thus, the analytic sample for this aim included 433 individuals (58% girls, mean age was 12.0 ± 0.2 years, range 11.6-12.7). The mean APHV for girls was 11.4 ± 1.0 years with 16.3% classified as early maturers, 74.1% average, and 9.6% as late. The mean APHV for all boys cannot be reported based on the limitations described above. For the early maturing boys, the average APHV was 11.6 ± 0.7 years and for average maturing boys was 12.9 ± 0.3 years. Table 2 represents the frequency and means for somatic maturity parameters for early, average, and late maturing girls and boys. For girls, the mean difference between chronological age and biological age was 0.1 ± 0.8 years with a range of -1.9 to 2.9. For boys, the mean difference between chronological age and biological age was 1.3 ± 0.6 years with a range of -0.4 to 3.9.

At age 12 years for boys, according to CDC chronological age and-sex-specific percentiles, 15.9% children were classified as overweight and 16.6% were obese with more boys than girls being overweight or obese (36% vs 28%, respectively). Table 3 shows the comparison of weight classification according to standard CDC classification based on chronological age and biological age. When BMI percentiles were referenced using biological age versus chronological age, there was a small increase in the proportion of 12 year old children classified as overweight (from 15.9% to 16.4%), and

small decrease in the proportion of children classified as obese (from 16.6% to 13.2%). This is related to a small difference in the mean BMI percentile within the sample; chronological versus biological mean, BMI percentile=64.9 ± 27.8 versus 61.7 ± 28.7. For those who shifted from the overweight category based on chronological age to the normal weight category based on biological age, the mean BMI percentile decreased from 87.9 ± 2.0 to 81.3 ± 2.7; and, for those who shifted from the obese category based on chronological age to the normal weight category based on biological age the mean BMI percentile decreased from 96.1 ± 0.6 to 93.0 ± 1.2. Overall, 22 (30.6%) overweight or obese boys and 14 (20.3%) overweight or obese girls were reclassified into a lower weight category (χ^2 =527.5, p<0.001). Underestimations of obesity were also seen with 8 total children reclassified into a higher weight category. The results of the χ^2 test indicate there were statistically significant differences in the number of children reclassified.

Of the 132 early maturers, 52.3% were normal weight using standard CDC classification based on chronological age (18.2% overweight, 29.6% obese), compared with 60.1% using biological age (19.7% overweight, 18.9% obese). This represents misclassification of 19 overweight or obese boys and 7 overweight or obese girls. Of the 277 average maturers, 72.9% were classified as normal weight using standard CDC classification based on chronological age (15.2% overweight, 11.9% obese), compared to 74.0% using biological age (14.4% overweight, 11.6% obese). Last, of the 24 late maturing girls, 88% were classified as normal weight using standard CDC classification based on chronological age (12% overweight, 0% obese), compared to 79.2% using biological age (20.8% overweight, 0% obese).

CHAPTER V

Discussion

The purpose of the current investigation was to describe growth and maturity characteristics of 8-14 year-old boys and girls in a longitudinal study from a Midwestern Nebraska community, with special attention on the role of biological maturity in the classification of weight status. As expected, children get taller and heavier with age; however, significant differences exist in growth according to weight classification. Furthermore, biological maturity may impact the classification of adolescent children, specifically those classified as overweight and obese.

Children grow and mature at different rates, and the three key determinants of childhood and adolescent growth and maturation are genes, hormones, and nourishment [37]. Although several childhood growth studies have documented the negative consequences of undernutrition [155], there are very few longitudinal studies that have examined the impact of overnutrition on the growth rates and maturation of overweight and obese children [156, 157]. Excess adiposity during childhood can affect the process of growth and puberty. Specific information related to the relationship between biological maturation and adiposity are discussed later in this section.

In the current study, obese boys and girls were taller and heavier than the overweight and normal weight boys and girls between the ages of 8-11 years. These findings are concordant with other studies that show childhood obesity is related to an acceleration in linear growth prior to puberty. Rapid weight gain during the pre-pubertal/mid-to-late childhood years is often accompanied by a higher growth velocity

and advanced bone age compared to lean subjects [126, 127, 158, 159]. Advanced skeletal maturity (i.e., bone age) has been shown to precede linear growth acceleration in overweight individuals, as well as subsequent growth velocity reduction due to a lesser growth spurt during puberty [156, 158-160]. The temporary increase in height gain during mid-to-late childhood is compensated by an earlier onset of puberty and subnormal height gain during adolescence [156]. More specifically, He & Karlberg [156] found that a higher childhood BMI was related to increased height gain during the same period, an earlier onset of puberty, and reduced height gain during adolescence. It has been concluded that overnutrition (e.g., obesity) in childhood may not result in a difference in final adult stature as obese and non-obese children show similar adult stature [134, 161].

In the current study, the mean stature for obese girls fell from approximately the 90th percentile at age 11 years approximately to the 50th percentile by age 14 years, which resulted in similar heights between weight classifications. Obese boys remained taller than the overweight and normal weight boys, as the mean stature approximated the 75th percentile at 14 years; however, this may not reflect the final stature given that boys do not reach adult height until a later age than girls. Our findings suggest the obese girls entered puberty earlier than average, resulting in a visible manifestation of a respective early growth spurt. At age 12 years, 31% of the obese girls were classified as an early maturer based on APHV. For boys, an inflection in height velocity was observed for the normal weight and overweight categories, but not for the obese category. This inflection signifies the initiation of the boys adolescent growth spurt, and on average occurred

around 11.5 years of age in our sample, which is concordant with the established literature. However, since no distinct inflection in height velocity was observed for the obese boys, it is more difficult to conclude their maturity status. This finding seems to further highlight the subnormal aspect of height gain among obese children during adolescence.

The epidemic of pediatric obesity has stimulated an interest in the relationship between body composition and the timing and tempo of puberty [124]. Although the biological mechanisms remain poorly understood [125], there is a growing body of literature which seems to suggest a complex bidirectional causal relationship between the early onset of puberty and obesity in females. There are several studies which have reported a prediction of age at pubertal onset in overweight and obese girls, and the established observation is that overweight girls tend to mature earlier [162]. Although there is an established relationship, the studies that show a relationship between early puberty and obesity in girls do not answer the question of whether increased body fat predisposes girls to earlier puberty, or if earlier puberty in some girls leads to estrogen-mediated increase in body fat [163]. A review of the effects of gonadal steroids on body composition in adults concluded that estrogens, and possibly progesterone, largely account for the degree of body fatness in women as opposed to men [163]. This is because these hormones seem to work together to favor the storage of excess calories as fat, with estrogens promoting the accumulation of fat in peripheral adipose tissue depots [164]. Therefore, it is possible that the early pubertal girl produces enough gonadal

steroids to result in greater BMI and greater body fat than would be found in age-matched pre-pubertal girls [164].

In contrast, two other longitudinal studies have a different physiological explanation for the relationship between obesity and early maturation. One study [165] found that girls with a higher percentage of body (as measured by skinfold thickness) or higher BMI percentiles at age 7 years were significantly more likely to be classified as having earlier pubertal development at or by age 9 years. Pubertal development was measured by stage of breast development (i.e., Tanner Stages), serum estradiol levels, and a parental assessed pubertal development scale [165]. The same research group also found that an increased BMI as early as age 3 years, plus the increase of BMI between the ages 3 to 6 years, were significant risk factors for whether a girl would be earlier than average in entering puberty [166].

It is purported that subcutaneous fat tissue acts as a secondary hormonal gland and adipose tissue influences the synthesis and release of hormones [81, 82, 128]. Several cross sectional and longitudinal studies, have shown a marked rise in serum leptin concentrations in young girls, beginning as early as age 7 years and continuing as they progress through puberty at least until age 15 years [166-168]. These changes in leptin are mirrored by increasing body fat during female puberty. In one study, the rise in leptin was well established two years prior to clear increases in key pubertal hormones (luteinizing hormone, estradiol) were observed [167]. This is consistent with the hypothesis that higher leptin levels are one of the factors that are critical in allowing puberty to progress, rather than a result of the hormonal increases of puberty.

Contrary to girls, little is known about the possible relationships between obesity and pubertal maturation in boys, as the evidence is less clear with conflicting results [129]. To make this matter more complex, European studies have shown obesity is associated with earlier puberty and voice break (an indirect marker of puberty) [134-138], while American studies have reported the opposite in that obesity is associated with delayed puberty in boys [139-142]. One potential explanation for the conflicting findings between European and American studies is that a variety of maturity assessment methods were used. For example, in one European study maturity was assessed by stages of secondary sex characteristics (i.e., Tanner stages), while in one American study maturity was assessed through APHV. In comparison to girls, where there is a major milestone of puberty (i.e., age at menarche), there is no such corresponding event in boys, causing it to be much more difficult to study the relationship. To fully assess maturity effectively, a large sample of healthy boys with annual or semi-annual recorded statures and body masses, alongside a physical examination with Tanner staging [169] done by experienced personnel, or longitudinal growth records detailed enough to allow one to determine accurately the time of the pubertal growth spurt are necessary. Very few growth studies are suited for obtaining this information; thus, direct assessments of biological maturity (i.e., sexual and skeletal) are limited and have only been performed in a few studies, whereas others have been based on proxy markers of pubertal onset and progression (i.e., maturity offset or age at voice break).

Another key aspect when examining a potential relationship between adiposity and pubertal maturation is the use of BMI. In boys, the BMI as a proxy of body fat may

be misleading, because the correlation between BMI and body fat is much lower in boys than in girls [80]. This may be because during male puberty, the increasing muscle mass related to the anabolic effect of rising testosterone levels will cause an increase in total body mass and BMI independent of any increase in body fat [163]. In the current study, peak height velocity and the respective APHV was determined using longitudinal growth records. Age at peak height velocity is the most commonly used indicator of maturity in longitudinal studies of adolescence [37]. While it is possible that the obese boys were experiencing delayed puberty, given the age range of the data, the findings in relation to maturity status remain inconclusive. Moving forward, future growth and maturational studies must ensure consistent pubertal assessment methodology, rigorous training to ensure accuracy, and a data set that fully encompasses maturational events.

As mentioned previously, obese boys and girls for both sexes were taller and heavier than their normal and overweight peers. In the current study, once a weight status was established, many of the children remained in the same category through time. This concept is referred to as tracking [170], which is the stability of a trait over time. Our findings support the literature [20, 70, 72, 75] that once obesity is established, it can be very difficult to reverse. At age 8, 179 participants were classified as either overweight or obese, which 144 (80.5%) were classified as either overweight or obese by age 14 years. This raises an important question about obesity in childhood. Childhood obesity has often been discussed whether it is a transient or persistent health problem [70]. A brief period of obesity may be no reason to worry; however, if obesity does become persistent, meaning, firmly established and difficult to reverse, it can lead to health concerns that

manifest in adulthood [55, 56] or even earlier. Findings from several studies have indicated weight status is established at early ages [73-75], and children with obesity do not always reach a normal weight [77]. Epidemiologic studies indicate the most significant increase in overweight status occurs between preschool (ages 2-5 years) and school age (ages 6-11 years) [171], which is highly predictive of progressing to obesity in subsequent developmental periods [172]. Persistent obesity can be primarily attributed to the adiposity rebound, which is the second rise in adiposity that occurs between ages 3-7 years [173]. The age at which the adiposity rebound occurs predicts greater fatness into adolescence [174] and adulthood [175-177]. This increase in overweight/obesity prevalence, alongside little fluctuation in weight status categories through time provides insight that obesity can be hard to reverse.

In the current study, significant differences in yearly weight velocity from the ages of 8-11 were observed. More specifically, obese children were gaining more than two times the amount of weight compared to normal weight children during this period. As an example, obese boys and girls at the age of 10 years were gaining 8.4 kg/year and 9.1 kg/year, respectively, while the normal weight boys and girls were gaining 3.7 kg/year and 4.5 kg/year, respectively. After 11 years of age, the significant differences in weight velocity were no longer observed as children were gaining close to the same amount of weight regardless of weight classification (i.e., 14 years obese boys and girls were gaining 7.7 kg/year and 4.9 kg/year, respectively, while normal weight boys and girls were gaining 6.7 kg/year and 4.7 kg/year, respectively. It is important to mention that while weight gain was much greater among the obese children, the yearly height

velocities were very similar. If weight gain would have been reduced through the earlier ages of 8-11 years (or younger), it is likely the obese children would have grown out of the condition. Given the findings that obesity can persist into adolescence and adulthood, an emphasis must be placed on prevention at earlier ages. Indeed, there is agreement among leading experts that obesity prevention programs should begin early in life and involve the family to promote lifestyle behavioral change that leads to a healthy weight across the lifespan [31, 178, 179].

The primary aim of the current investigation was to compare the weight categorization of a cohort of children using the standard procedure of weight classification using the age-and-sex specific CDC reference data versus an approach adjusted for maturation status (i.e., relative to biological age). Although the BMI and the respective age-and-sex specific percentiles have long been utilized in establishing weight status in children, maturation-related misclassification may result in overestimations of overweight prevalence in early maturing youth, and underestimations among later maturing youth [35, 36] as shown here. Overall, 8.5% of children were misclassified into a lesser category (e.g. from overweight to normal weight), and of these children who were misclassified 73% were classified as early maturers. This represented about 31% of overweight and obese boys, and 20% of overweight and obese girls. Only eight children (1.9%) were reclassified into a higher weight category. To my knowledge, only one other study has utilized a similar approach of examining the impact of biological maturity on the accuracy of weight classifications [38]. Gillison et al. [38] found that 32% of overweight girls and 15% of overweight boys (based on age-specific cut points) were

reclassified as normal weight when the classification approach was adjusted for maturity status. Additionally, 11% and 8% of obese girls and boys, respectively, were reclassified as overweight when the classification approach was adjusted for maturity [38]. These findings lend support to those of the current investigation.

To further highlight the impact of maturation on weight classification, a sensitivity and specificity analysis of BMI in classifying obesity (as measured by body fat mass via DXA scans) in New Zealand adolescents (maturity assessed by Tanner stages) reported 6%-12% misclassification of weight status [180]. Nonetheless, the study conducted by Gillison et al. and the current investigation demonstrate how weight classification may account for children's maturity status in addition to age and sex when plotting BMI against growth reference charts, and report on the likely effects in terms of changes to weight classifications. The findings of this study indicate that 22.9% of overweight girls and 35.3% of overweight boys (standard CDC classification based on chronological age) were reclassified as normal weight when the approach was adjusted using biological age. Also, 17.7% and 22.2% of obese boys and girls, respectively, were reclassified as overweight when the approach was adjusted using biological age. The findings suggest that early-maturing children are particularly at risk for weight status misclassification. While the overall effects of maturational adjustment are relatively small (8.5% of total sample), it should be considered when assessing young adolescents for weight status.

These results have implications for BMI surveillance and screening programs that are widely utilized worldwide to provide data for monitoring population-level obesity.

Providing BMI data as feedback has resulted in little retainment of weight management support [144], and may even isolate parents who are either angry or disbelieving of the information provided [144-146]. Research seeking the source of parental anger and rejection of objective anthropometry has found parents argue the validity of BMI percentile assessments when children approach puberty [145]. Also, parents pose the argument that judgements of weight status that do not account for relative differences in pubertal development are not valid [145]. Thus, accounting for maturity could alleviate some of these issues.

More importantly, assessments of weight status based upon chronological age may place children at harm. Although the physical ills of pediatric obesity are often focused upon, the myriad of psychological and social conditions like depression, low self-esteem, behavioral problems, issues in school, and an overall low self-reported quality of life are not emphasized enough [47, 59, 69]. Obese youth are at greater risk of poor mental health [181-183] and may be susceptible to the negative impact of an objective weight assessment, as they generally hold more negative physical self-perceptions (i.e., lower perceptions of attractiveness, sports competence, and fitness) [184]. As such, early-maturing youth represent a vulnerable group with whom health professionals should be particularly careful to minimize the potential unintended negative consequences of misclassifying weight status.

The findings highlight two key implications for clinical practice and public health. First, they raise the question of whether health professionals should adjust for maturity when determining whether youth are overweight. Given the lack of evidence that weight

monitoring results in positive effects on children's health and health behaviors [144, 185], and even such screening could undermine their well-being and self-concept [145, 146], adjusting for biological maturity when classifying weight status will result in little to no harm (i.e., children are not identified, and do not receive effective help). Conversely, this practice could be of benefit if health professionals are better able to raise awareness and engage with parents whose children remain classified as overweight or obese following adjustment, as a result of showing that the classification has been tailored to the child's level of biological maturity. The maturity adjustment approach using longitudinal data in the current study may be feasible, yet challenging for some health professionals (i.e., physicians, school nurses) to adopt and implement when assessing weight status. However, if obtaining and keeping record of longitudinal data is not feasible, there are alternative methods to determine maturity such as 1) the Khamis-Roche equation [115] to predict adult stature and derive a percentage of predicted adult stature, and 2) the maturity offset equation to predict years from peak height velocity [186].

Although this study primarily highlights the shortcomings of the BMI in using chronological age-based BMI reference charts to classify weight status, it must be acknowledged that it is sufficient for what it was originally created and intended to do, which is measure rates of obesity in a population. The BMI is solely a screening tool and not a diagnostic tool [31], because it is only a proxy measure of body fatness. Because it is a general measure of obesity that works well in most people, looking at changes in BMI allow health professionals to gauge how rates of overweight and obesity have

changed through time. In summary, the BMI is a useful but imperfect proxy indicator of fat mass and subsequent health risk [101, 102]; yet, its widespread use among health professionals stems from its cost effectiveness and practicality.

A major strength of this study lies within collecting anthropometric data longitudinally to gain a better understanding of the growth and maturational characteristics, as few longitudinal growth studies are available. More specifically, a unique aspect of this study was the comparison of growth across normal weight, overweight, and obese youth. Obtaining longitudinal data permits the calculation of growth rates, which further allows for the ascertainment of APHV. With longitudinal data available, assessing APHV is a very feasible gauge of somatic maturity. Using APHV to calculate a subsequent biological age is also unique to maturational adjustment approaches.

As with any study, there are limitations within this study that must be addressed. First, a major limitation is that the age range was restricted from ages 8 to 14 and does not encompass puberty to its entirety in some subjects. Many longitudinal growth studies acquire data during the entire age range of childhood and adolescence (approximately 6 to 18 years of age). Some growth studies even acquire anthropometric/biological data from birth, through childhood and puberty, with a period of follow-up in adulthood [187]. To enhance studies addressing maturity adjustment of weight status moving forward, longitudinal data should be collected from ages 8-18 years to fully capture puberty and the adolescent growth spurt. Additionally, it has been recommended the interval between two measurements should not be less than 0.85 years (310 days) and not exceed 1.15

years (420 days) for proper growth velocities [188]. Given the feasibility of working in a school environment, many growth velocities were outside this range. Second, participants were predominately white children from central Nebraska, which limits our ability to generalize the results to children of other ethnicities [189, 190]. Third, a variety of potential problems resulted in the discarding of inadequate growth velocities. These problems included measurement error, lack of precision, data entry error, insufficient training, and lack of interrater reliability. Staff trained on measurement protocols were instructed to measure stature to the nearest 0.25 inch, whereas many growth studies measure subjects to the nearest 0.1 of an inch. Finally, one last potential problem is interrater reliability.

CHAPTER VI

Conclusion

The goal of the current investigation was to describe growth characteristics of central Nebraska youth, while attempting to quantify the difference that adjusting classifications of weight status by a child's level of maturity could have on both population estimates of childhood overweight/obesity, as well as the treatment of individual children. The results of this study further highlight the variability of growth and maturation among children, especially during pubertal growth. It seems weight status at an early age can also influence the processes of growth and maturation, as the findings show obese youth are taller and heavier at younger ages, and furthermore experience a lesser growth spurt compared to their peers. Yet, it is important to remember that there are a variety of factors that influence the manner in which children grow, develop, and mature.

While the overall effects of adjustment are small (8.5% of the total sample reclassified into a lesser weight category), maturation status should be considered, particularly when assessing young adolescents when maturational events are likely to occur. Given that 31% of overweight and obese boys, and 20% of overweight and obese girls were reclassified into a lower weight category, there are serious implications for weight status being misclassified due to maturity. Moving forward, health professionals throughout the world should seriously consider tailoring judgements of weight status for maturity.

REFERENCES

1. Ayton, A. and A. Ibrahim, *Obesity is a public health emergency.* BMJ, 2019. **366**: p. l5463.

2. Must, A., et al., *The disease burden associated with overweight and obesity.* Jama, 1999. **282**(16): p. 1523-9.

3. Calle, E.E., et al., *Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults.* New England Journal of Medicine, 1999. **341**(15): p. 1097-1105.

4. Lavie, C.J., R.V. Milani, and H.O. Ventura, *Obesity and Cardiovascular Disease.* Risk Factor, Paradox, and Impact of Weight Loss, 2009. **53**(21): p. 1925-1932.

5. Gómez-Ambrosi, J., et al., *Body Adiposity and Type 2 Diabetes: Increased Risk With a High Body Fat Percentage Even Having a Normal BMI.* Obesity, 2011. **19**(7): p. 1439-1444.

6. Calle, E.E. and R. Kaaks, *Overweight, obesity and cancer: epidemiological evidence and proposed mechanisms.* Nat Rev Cancer, 2004. **4**(8): p. 579-91.

7. Hales, C.M., et al. *Prevalence of obesity and severe obesity among adults: United States, 2017-2018.* . [Online] 2020 February 27th, 2020 [cited 2020. May 11]; NCHS Data Brief, no 360.:[Available from: https://www.cdc.gov/nchs/products/databriefs/db360.htm.

8. Masters, R.K., et al., *The impact of obesity on US mortality levels: the importance of age and cohort factors in population estimates.* Am J Public Health, 2013. **103**(10): p. 1895-901.

9. *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General.* , N.C.f.C.D.P.a.H. Promotion, Editor. 2014, United States Department of Health and Human Services: Atlanta, GA.

10. Tremmel, M., et al., *Economic Burden of Obesity: A Systematic Literature Review.* Int J Environ Res Public Health, 2017. **14**(4).

11. Hammond, R.A. and R. Levine, *The economic impact of obesity in the United States.* Diabetes Metab Syndr Obes, 2010. **3**: p. 285-95.

12. Trogdon, J.G., et al., *Indirect costs of obesity: a review of the current literature.* Obes Rev, 2008. **9**(5): p. 489-500.

13. Finkelstein, E.A., et al., *Annual medical spending attributable to obesity: payer-and service-specific estimates.* Health Aff (Millwood), 2009. **28**(5): p. w822-31.

14. Hales, C.M., et al., *Trends in Obesity and Severe Obesity Prevalence in US Youth and Adults by Sex and Age, 2007-2008 to 2015-2016.* JAMA, 2018. **319**(16): p. 1723-1725.

15. Fryar, C.D., M.D. Carroll, and J. Afful, *Prevalence of overweight, obesity, and severe obesityamong children and adolescents aged 2-19 years: United States, 1963-1965 through 2017-2018.*, in *NCHS Health E-Stats*. 2020, Centers for Disease Control and Prevention.

16. Clarke, W.R. and R.M. Lauer, *Does childhood obesity track into adulthood?* Crit Rev Food Sci Nutr, 1993. **33**(4-5): p. 423-30.

17. Gordon-Larsen, P., N.S. The, and L.S. Adair, *Longitudinal trends in obesity in the United States from adolescence to the third decade of life.* Obesity (Silver Spring), 2010. **18**(9): p. 1801-4.

18. Guo, S.S. and W.C. Chumlea, *Tracking of body mass index in children in relation to overweight in adulthood.* Am J Clin Nutr, 1999. **70**(1): p. 145s-148s.

19. Herman, K.M., et al., *Tracking of obesity and physical activity from childhood to adulthood: the Physical Activity Longitudinal Study.* Int J Pediatr Obes, 2009. **4**(4): p. 281-8.

20. Singh, A.S., et al., *Tracking of childhood overweight into adulthood: a systematic review of the literature.* Obes Rev, 2008. **9**(5): p. 474-88.

21. Cote, A.T., et al., *Childhood obesity and cardiovascular dysfunction.* J Am Coll Cardiol, 2013. **62**(15): p. 1309-19.

22. Lloyd, L.J., S.C. Langley-Evans, and S. McMullen, *Childhood obesity and risk of the adult metabolic syndrome: a systematic review.* International journal of obesity (2005), 2012. **36**(1): p. 1-11.

23. Bacha, F. and S.S. Gidding, *Cardiac Abnormalities in Youth with Obesity and Type 2 Diabetes.* Curr Diab Rep, 2016. **16**(7): p. 62.

24. Pollock, N.K., *Childhood obesity, bone development, and cardiometabolic risk factors.* Mol Cell Endocrinol, 2015. **410**: p. 52-63.

25. Bass, R. and I. Eneli, *Severe childhood obesity: an under-recognised and growing health problem.* Postgrad Med J, 2015. **91**(1081): p. 639-45.

26. Jensen, M.D., et al., *2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society.* Circulation, 2014. **129**(25 Suppl 2): p. S102-S138.

27. Durazo-Arvizu, R.A., et al., *Mortality and optimal body mass index in a sample of the US population.* Am J Epidemiol, 1998. **147**(8): p. 739-49.

28. *Prevention of Pediatric Overweight and Obesity.* Pediatrics, 2003. **112**(2): p. 424.

29. Gulati, A.K., D.W. Kaplan, and S.R. Daniels, *Clinical tracking of severely obese children: a new growth chart.* Pediatrics, 2012. **130**(6): p. 1136-40.

30. Keys, A., et al., *Indices of relative weight and obesity.* Journal of Chronic Diseases, 1972. **25**(6): p. 329-343.

31. Barlow, S.E., *Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report.* Pediatrics, 2007. **120 Suppl 4**: p. S164-92.

32. Cornier, M.A., et al., *Assessing adiposity: a scientific statement from the American Heart Association.* Circulation, 2011. **124**(18): p. 1996-2019.

33. Hammer, L.D., et al., *Standardized percentile curves of body-mass index for children and adolescents.* Am J Dis Child, 1991. **145**(3): p. 259-63.

34. Pietrobelli, A., et al., *Body mass index as a measure of adiposity among children and adolescents: a validation study.* J Pediatr, 1998. **132**(2): p. 204-10.

35. Himes, J.H., *Maturation-related deviations and misclassification of stature and weight in adolescence.* Am J Hum Biol, 1999. **11**(4): p. 499-504.

36. Wang, Y. and L. Adair, *How does maturity adjustment influence the estimates of overweight prevalence in adolescents from different countries using an international reference?* Int J Obes Relat Metab Disord, 2001. **25**(4): p. 550-8.

37. Malina, R.M., C. Bouchard, and O. Bar-Or, *Growth, maturation, and physical activity*. 2004: Human kinetics.

38. Gillison, F., et al., *Assessing the impact of adjusting for maturity in weight status classification in a cross-sectional sample of UK children.* BMJ open, 2017. **7**(6): p. e015769-e015769.

39. Eveleth, P.B. and J.M. Tanner, *Worldwide Variation in Human Growth*. 1991, Cambridge: Cambridge University Press.

40. Falkner, F.T. and J.M. Tanner, *Human Growth: A Comprehensive Treatise*. 1986: Plenum Press.

41. Mahan, L.K. and J.M. Rees, *Nutrition in Adolescence*. 1984: Times Mirror/Mosby College Pub.

42. Morabia, A. and M.C. Costanza, *International variability in ages at menarche, first livebirth, and menopause. World Health Organization Collaborative Study of Neoplasia and Steroid Contraceptives.* Am J Epidemiol, 1998. **148**(12): p. 1195-205.

43. Tanner, J.M., *A history of the study of human growth*. 1981, Cambridge: Cambridge University Press.

44. Ogden, C.L., et al., *Prevalence of overweight and obesity in the United States, 1999-2004.* Jama, 2006. **295**(13): p. 1549-55.

45. Murray, C.J., et al., *The state of US health, 1990-2010: burden of diseases, injuries, and risk factors.* Jama, 2013. **310**(6): p. 591-608.

46. Allison, D.B., et al., *Obesity as a disease: a white paper on evidence and arguments commissioned by the Council of the Obesity Society.* Obesity (Silver Spring), 2008. **16**(6): p. 1161-77.

47. Mustillo, S., et al., *Obesity and psychiatric disorder: developmental trajectories.* Pediatrics, 2003. **111**(4 Pt 1): p. 851-9.

48. Hruby, A. and F.B. Hu, *The Epidemiology of Obesity: A Big Picture.* PharmacoEconomics, 2015. **33**(7): p. 673-689.

49. Ng, M., et al., *Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: a systematic analysis for the Global Burden of Disease Study 2013.* Lancet, 2014. **384**(9945): p. 766-81.

50. Stevens, G.A., et al., *National, regional, and global trends in adult overweight and obesity prevalences.* Popul Health Metr, 2012. **10**(1): p. 22.

51. Kelly, T., et al., *Global burden of obesity in 2005 and projections to 2030.* Int J Obes (Lond), 2008. **32**(9): p. 1431-7.

52. Wang, Y., et al., *Will all Americans become overweight or obese? estimating the progression and cost of the US obesity epidemic.* Obesity (Silver Spring), 2008. **16**(10): p. 2323-30.

53. Fryar, C.D., M.D. Carroll, and C.L. Ogden, *Prevalence of Overweight, Obesity, and Severe Obesity Among Children and Adolescents Aged 2-19 Years: United States, 1962-1963 through 2015-2016.* 2018, Centers for Disease Control and Prevention.

54. Kumar, S. and A.S. Kelly. *Review of childhood obesity: from epidemiology, etiology, and comorbidities to clinical assessment and treatment*. in *Mayo Clinic Proceedings*. 2017. Elsevier.

55. Taylor, E.D., et al., *Orthopedic complications of overweight in children and adolescents.* Pediatrics, 2006. **117**(6): p. 2167-74.

56. Halfon, N., K. Larson, and W. Slusser, *Associations between obesity and comorbid mental health, developmental, and physical health conditions in a nationally representative sample of US children aged 10 to 17.* Acad Pediatr, 2013. **13**(1): p. 6-13.

57. Morrison, K.M., et al., *Association of depression & health related quality of life with body composition in children and youth with obesity.* J Affect Disord, 2015. **172**: p. 18-23.

58. Schwimmer, J.B., T.M. Burwinkle, and J.W. Varni, *Health-Related Quality of Life of Severely Obese Children and Adolescents.* JAMA, 2003. **289**(14): p. 1813-1819.

59. Taylor, V.H., et al., *The impact of obesity on quality of life.* Best Pract Res Clin Endocrinol Metab, 2013. **27**(2): p. 139-46.

60. Kelly, A.S., et al., *Severe obesity in children and adolescents: identification, associated health risks, and treatment approaches: a scientific statement from the American Heart Association.* Circulation, 2013. **128**(15): p. 1689-712.

61. Freedman, D.S., et al., *Cardiovascular risk factors and excess adiposity among overweight children and adolescents: the Bogalusa Heart Study.* J Pediatr, 2007. **150**(1): p. 12-17.e2.

62. Han, J.C., D.A. Lawlor, and S.Y. Kimm, *Childhood obesity.* Lancet, 2010. **375**(9727): p. 1737-48.

63. Narang, I. and J.L. Mathew, *Childhood obesity and obstructive sleep apnea.* J Nutr Metab, 2012. **2012**: p. 134202.

64. Mohanan, S., et al., *Obesity and asthma: pathophysiology and implications for diagnosis and management in primary care.* Exp Biol Med (Maywood), 2014. **239**(11): p. 1531-40.

65. Sutherland, E.R., *Obesity and asthma.* Immunology and allergy clinics of North America, 2008. **28**(3): p. 589-ix.

66. Africa, J.A., K.P. Newton, and J.B. Schwimmer, *Lifestyle Interventions Including Nutrition, Exercise, and Supplements for Nonalcoholic Fatty Liver Disease in Children.* Digestive diseases and sciences, 2016. **61**(5): p. 1375-1386.

67. Koebnick, C., et al., *Pediatric obesity and gallstone disease.* J Pediatr Gastroenterol Nutr, 2012. **55**(3): p. 328-33.

68. Malaty, H.M., et al., *Obesity and gastroesophageal reflux disease and gastroesophageal reflux symptoms in children.* Clin Exp Gastroenterol, 2009. **2**: p. 31-6.

69. Rankin, J., et al., *Psychological consequences of childhood obesity: psychiatric comorbidity and prevention.* Adolesc Health Med Ther, 2016. **7**: p. 125-146.

70. Cunningham, S.A., et al., *Entrenched obesity in childhood: findings from a national cohort study.* Annals of epidemiology, 2017. **27**(7): p. 435-441.

71. Goldhaber-Fiebert, J.D., et al., *The utility of childhood and adolescent obesity assessment in relation to adult health.* Medical decision making : an international journal of the Society for Medical Decision Making, 2013. **33**(2): p. 163-175.

72. Stuart, B. and L. Panico, *Early-childhood BMI trajectories: evidence from a prospective, nationally representative British cohort study.* Nutrition & Diabetes, 2016. **6**(3): p. e198-e198.

73. Ziyab, A.H., et al., *Developmental trajectories of Body Mass Index from infancy to 18 years of age: prenatal determinants and health consequences.* J Epidemiol Community Health, 2014. **68**(10): p. 934-41.

74. Franzese, A., et al., *Onset of obesity in children through the recall of parents: relationship to parental obesity and life events.* J Pediatr Endocrinol Metab, 1998. **11**(1): p. 63-7.

75. Kamath, C.C., et al., *Clinical review: behavioral interventions to prevent childhood obesity: a systematic review and metaanalyses of randomized trials.* J Clin Endocrinol Metab, 2008. **93**(12): p. 4606-15.

76. Hernandez, R.G., et al., *Predictors of favorable growth patterns during the obesity epidemic among US school children.* Clin Pediatr (Phila), 2015. **54**(5): p. 458-68.

77. Schuster, M.A., et al., *Changes in obesity between fifth and tenth grades: a longitudinal study in three metropolitan areas.* Pediatrics, 2014. **134**(6): p. 1051-8.

78. Adair, L.S. and P. Gordon-Larsen, *Maturational timing and overweight prevalence in US adolescent girls.* Am J Public Health, 2001. **91**(4): p. 642-4.

79. Beunen, G.P., et al., *Adiposity and biological maturity in girls 6-16 years of age.* Int J Obes Relat Metab Disord, 1994. **18**(8): p. 542-6.

80. Daniels, S.R., P.R. Khoury, and J.A. Morrison, *The utility of body mass index as a measure of body fatness in children and adolescents: differences by race and gender.* Pediatrics, 1997. **99**(6): p. 804-7.

81. Frisch, R.E. and J.W. McArthur, *Menstrual cycles: fatness as a determinant of minimum weight for height necessary for their maintenance or onset.* Science, 1974. **185**(4155): p. 949-51.

82. Frisch, R.E. and R. Revelle, *Height and weight at menarche and a hypothesis of critical body weights and adolescent events.* Science, 1970. **169**(3943): p. 397-9.

83. Garn, S.M., et al., *Maturational timing as a factor in female fatness and obesity.* The American Journal of Clinical Nutrition, 1986. **43**(6): p. 879-883.

84. Guo, S.S., et al., *Age- and maturity-related changes in body composition during adolescence into adulthood: The Fels Longitudinal Study.* International Journal of Obesity, 1997. **21**(12): p. 1167-1175.

85. Jaruratanasirikul, S., L. Mo-suwan, and L. Lebel, *Growth pattern and age at menarche of obese girls in a transitional society.* J Pediatr Endocrinol Metab, 1997. **10**(5): p. 487-90.

86. Kaplowitz, P.B., et al., *Earlier onset of puberty in girls: relation to increased body mass index and race.* Pediatrics, 2001. **108**(2): p. 347-53.

87. Kemper, H.C., R. Verschuur, and J.W. Ritmeester, *Longitudinal development of growth and fitness in early and late maturing teenagers.* Pediatrician, 1987. **14**(4): p. 219-225.

88. Morrison, J.A., et al., *Sexual maturation and obesity in 9- and 10-year-old black and white girls: The National Heart, Lung, and Blood Institute Growth and Health Study.* The Journal of Pediatrics, 1994. **124**(6): p. 889-895.

89. van Lenthe, F.J., C.G. Kemper, and W. van Mechelen, *Rapid maturation in adolescence results in greater obesity in adulthood: the Amsterdam Growth and Health Study.* Am J Clin Nutr, 1996. **64**(1): p. 18-24.

90. Wellens, R., et al., *Body size and fatness in young adults in relation to age at menarche.* Am J Hum Biol, 1992. **4**(6): p. 783-787.

91. Ribeiro, J., et al., *Association between overweight and early sexual maturation in Portuguese boys and girls.* Ann Hum Biol, 2006. **33**(1): p. 55-63.

92. Freedman, D.S., M. Horlick, and G.S. Berenson, *A comparison of the Slaughter skinfold-thickness equations and BMI in predicting body fatness and cardiovascular disease risk factor levels in children.* Am J Clin Nutr, 2013. **98**(6): p. 1417-24.

93. Garrow, J.S. and J. Webster, *Quetelet's index (W/H2) as a measure of fatness.* Int J Obes, 1985. **9**(2): p. 147-53.

94. Wohlfahrt-Veje, C., et al., *Body fat throughout childhood in 2647 healthy Danish children: agreement of BMI, waist circumference, skinfolds with dual X-ray absorptiometry.* Eur J Clin Nutr, 2014. **68**(6): p. 664-70.

95. Force, U.P.S.T., *Screening for Obesity in Children and Adolescents: US Preventive Services Task Force Recommendation Statement.* JAMA, 2017. **317**(23): p. 2417-2426.

96. World Health, O., *Physical Status: The Use and Interpretation of Anthropometry.* Geneva: World Health Organization, Technical Report Series, 1995. **No. 854**.

97. Guo, S.S., et al., *The predictive value of childhood body mass index values for overweight at age 35 y.* Am J Clin Nutr, 1994. **59**(4): p. 810-9.

98. Whitaker, R.C., et al., *Predicting obesity in young adulthood from childhood and parental obesity.* N Engl J Med, 1997. **337**(13): p. 869-73.

99. Eisenmann, J.C., K.A. Heelan, and G.J. Welk, *Assessing body composition among 3- to 8-year-old children: anthropometry, BIA, and DXA.* Obes Res, 2004. **12**(10): p. 1633-40.

100. Freedman, D.S. and B. Sherry, *The validity of BMI as an indicator of body fatness and risk among children.* Pediatrics, 2009. **124 Suppl 1**: p. S23-34.

101. Mei, Z., et al., *Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents.* Am J Clin Nutr, 2002. **75**(6): p. 978-85.

102. Kuczmarski, R.J., et al., *2000 CDC Growth Charts for the United States: methods and development.* Vital Health Stat 11, 2002(246): p. 1-190.

103. Wadsworth, M.E.J., *Growth, maturation and body composition: the Fels longitudinal study 1929-1991*, in *J Epidemiol Community Health*. 1993. p. 251-2.

104. Lohman, T.G., A.F. Roche, and R. Martorell, *Anthropometric standardization reference manual*. 1988, Champaign, IL: Human Kinetics Books.

105. Lloyd, R.S., Oliver, J. L. , *Strength and Conditioning for Young Athletes*. 1st ed. 2014, London, England: Routledge. 256.

106. Patel L., C.P., *Normal and Disordered Growth*, in *Clinical Pediatric Endocrinology*. 2005. p. 90-112.

107. Wit, J.M. and C. Camacho-Hübner, *Endocrine regulation of longitudinal bone growth.* Endocr Dev, 2011. **21**: p. 30-41.

108. Rogol, A.D., P.A. Clark, and J.N. Roemmich, *Growth and pubertal development in children and adolescents: effects of diet and physical activity.* Am J Clin Nutr, 2000. **72**(2 Suppl): p. 521s-8s.

109. Forbes, G.B., *Nutrition and growth.* J Pediatr, 1977. **91**(1): p. 40-2.

110. Marceau, K., et al., *Individual differences in boys' and girls' timing and tempo of puberty: modeling development with nonlinear growth models.* Dev Psychol, 2011. **47**(5): p. 1389-1409.

111. Manzoor Mughal, A., N. Hassan, and A. Ahmed, *Bone age assessment methods: a critical review.* Pakistan journal of medical sciences, 2014. **30**(1): p. 211-215.

112. DeLuca, F. and J. Baron, *Skeletal Maturation.* The Endocrinologist, 1999. **9**(4): p. 286-293.

113. Susman, E.J., et al., *Longitudinal Development of Secondary Sexual Characteristics in Girls and Boys Between Ages 9½ and 15½ Years.* Archives of Pediatrics & Adolescent Medicine, 2010. **164**(2): p. 166-173.

114. Luciano, A.P., et al., *Median ages at stages of sexual maturity and excess weight in school children.* Reproductive health, 2013. **10**: p. 56-56.

115. Khamis, H.J. and A.F. Roche, *Predicting adult stature without using skeletal age: the Khamis-Roche method.* Pediatrics, 1994. **94**(4 Pt 1): p. 504-7.

116. Tanner, J.M., *Growth at adolescence, 2nd ed*. Growth at adolescence, 2nd ed. 1962, Thomas: Springfield, Ill.

117. Buschang, P.H., R. Tanguay, and A. Demirjian, *Growth instability of French-Canadian children during the first three years of life.* Can J Public Health, 1985. **76**(3): p. 191-4.

118. Roche, A.F. and J. Li, *Changes in percentile levels for growth.* Pediatric Forum, 1998. **9**: p. 30-34.

119. Roche, A.F., *Growth, maturation, and body composition: the Fels Longitudinal Study 1929-1991*. 1992: Cambridge University Press.

120. Abbassi, V., *Growth and normal puberty.* Pediatrics, 1998. **102**(2 Pt 3): p. 507-11.

121. Beunen, G.P., A.D. Rogol, and R.M. Malina, *Indicators of biological maturation and secular changes in biological maturation.* Food Nutr Bull, 2006. **27**(4 Suppl Growth Standard): p. S244-56.

122. Falkner, F., Marshal, WA, Tanner JM. , *Human Growth II. Postnatal Growth.* 1986, New York, NY.: Plenum Press.

123. Prevention, C.f.D.C.a., *Data Table of Stature-for-age Charts.* 2001.

124. Ahmed, M.L., K.K. Ong, and D.B. Dunger, *Childhood obesity and the timing of puberty.* Trends Endocrinol Metab, 2009. **20**(5): p. 237-42.

125. Wadden, T.A. and G.A. Bray, *Handbook of obesity treatment*. 2018: Guilford Publications.

126. Vignolo, M., et al., *Growth and development in simple obesity.* Eur J Pediatr, 1988. **147**(3): p. 242-4.

127. Davison, K.K., E.J. Susman, and L.L. Birch, *Percent body fat at age 5 predicts earlier pubertal development among girls at age 9.* Pediatrics, 2003. **111**(4 Pt 1): p. 815-21.

128. Frisch, R.E., *The right weight: body fat, menarche and fertility.* Proc Nutr Soc, 1994. **53**(1): p. 113-29.

129. De Leonibus, C., M.L. Marcovecchio, and F. Chiarelli, *Update on statural growth and pubertal development in obese children.* Pediatric reports, 2012. **4**(4): p. e35-e35.

130. Dunger, D.B., M.L. Ahmed, and K.K. Ong, *Early and late weight gain and the timing of puberty.* Mol Cell Endocrinol, 2006. **254-255**: p. 140-5.

131. Adair, L.S., *Size at birth predicts age at menarche.* Pediatrics, 2001. **107**(4): p. E59.

132. Sloboda, D.M., et al., *Age at menarche: Influences of prenatal and postnatal growth.* J Clin Endocrinol Metab, 2007. **92**(1): p. 46-50.

133. Tam, C.S., et al., *Opposing Influences of Prenatal and Postnatal Growth on the Timing of Menarche.* The Journal of Clinical Endocrinology & Metabolism, 2006. **91**(11): p. 4369-4373.

134. He, Q. and J. Karlberg, *Bmi in childhood and its association with height gain, timing of puberty, and final height.* Pediatr Res, 2001. **49**(2): p. 244-51.

135. Juul, A., et al., *Age at voice break in Danish boys: effects of pre-pubertal body mass index and secular trend.* International Journal of Andrology, 2007. **30**(6): p. 537-542.

136. Monteilh, C., et al., *Timing of maturation and predictors of Tanner stage transitions in boys enrolled in a contemporary British cohort.* Paediatr Perinat Epidemiol, 2011. **25**(1): p. 75-87.

137. Vizmanos, B. and C. Martí-Henneberg, *Puberty begins with a characteristic subcutaneous body fat mass in each sex.* Eur J Clin Nutr, 2000. **54**(3): p. 203-8.

138. Ong, K.K., et al., *Timing of Voice Breaking in Males Associated with Growth and Weight Gain Across the Life Course.* The Journal of Clinical Endocrinology & Metabolism, 2012. **97**(8): p. 2844-2852.

139. Euling, S.Y., et al., *Examination of US puberty-timing data from 1940 to 1994 for secular trends: panel findings.* Pediatrics, 2008. **121 Suppl 3**: p. S172-91.

140. Wang, Y., *Is obesity associated with early sexual maturation? A comparison of the association in American boys versus girls.* Pediatrics, 2002. **110**(5): p. 903-10.

141. Lee, J.M., et al., *Body mass index and timing of pubertal initiation in boys.* Arch Pediatr Adolesc Med, 2010. **164**(2): p. 139-44.

142. Buyken, A.E., N. Karaolis-Danckert, and T. Remer, *Association of prepubertal body composition in healthy girls and boys with the timing of early and late pubertal markers.* Am J Clin Nutr, 2009. **89**(1): p. 221-30.

143. Hui, L.L., et al., *Infant growth and onset of puberty: prospective observations from Hong Kong's "Children of 1997" birth cohort.* Ann Epidemiol, 2012. **22**(1): p. 43-50.

144. Falconer, C.L., et al., *The benefits and harms of providing parents with weight feedback as part of the national child measurement programme: a prospective cohort study.* BMC Public Health, 2014. **14**: p. 549.

145. Gillison, F., F. Beck, and J. Lewitt, *Exploring the basis for parents' negative reactions to being informed that their child is overweight.* Public Health Nutr, 2014. **17**(5): p. 987-97.

146. Grimmett, C., et al., *Telling parents their child's weight status: psychological impact of a weight-screening program.* Pediatrics, 2008. **122**(3): p. e682-8.

147. Borra, S.T., et al., *Developing health messages: qualitative studies with children, parents, and teachers help identify communications opportunities for healthful lifestyles and the prevention of obesity.* J Am Diet Assoc, 2003. **103**(6): p. 721-8.

148. Syrad, H., et al., *Health and happiness is more important than weight': a qualitative investigation of the views of parents receiving written feedback on their child's weight as part of the National Child Measurement Programme.* J Hum Nutr Diet, 2015. **28**(1): p. 47-55.

149. Bini, V., et al., *Body mass index in children and adolescents according to age and pubertal stage.* Eur J Clin Nutr, 2000. **54**(3): p. 214-8.

150. O'Dea, J. and S. Abraham, *Should body-mass index be used in young adolescents?* Lancet, 1995. **345**(8950): p. 657.

151. Sherar, L.B., et al., *Relationship between trajectories of trunk fat mass development in adolescence and cardiometabolic risk in young adulthood.* Obesity (Silver Spring), 2011. **19**(8): p. 1699-706.

152. Sherar, L.B., et al., *Age and gender differences in youth physical activity: does physical maturity matter?* Med Sci Sports Exerc, 2007. **39**(5): p. 830-5.

153. Moore, S.A., et al., *Enhancing a somatic maturity prediction model.* Medicine & Science in Sports & Exercise, 2015. **47**(8): p. 1755-1764.

154. Berkey, C.S., et al., *Longitudinal height velocity standards for U.S. adolescents.* Statistics in Medicine, 1993. **12**(3-4): p. 403-414.

155. Martorell, R. *Long-term consequences of undernutrition on physical and cognitive development*. 2005.

156. He, Q. and J. Karlberg, *BMI in childhood and its association with height gain, timing of puberty, and final height.* Pediatric research, 2001. **49**(2): p. 244-251.

157. Denzer, C., et al., *Pubertal development in obese children and adolescents.* International Journal of Obesity, 2007. **31**(10): p. 1509-1519.

158. Akaboshi, I., et al., *Taller stature after postnatal rapid weight gain in early infancy predicts overweight status at age 3.* Acta Paediatr, 2008. **97**(10): p. 1460-4.

159. Sopher, A.B., et al., *Bone age advancement in prepubertal children with obesity and premature adrenarche: possible potentiating factors.* Obesity (Silver Spring), 2011. **19**(6): p. 1259-64.

160. Stovitz, S.D., et al., *Growing into obesity: patterns of height growth in those who become normal weight, overweight, or obese as young adults.* Am J Hum Biol, 2011. **23**(5): p. 635-41.

161. Denzer, C., et al., *Pubertal development in obese children and adolescents.* Int J Obes (Lond), 2007. **31**(10): p. 1509-19.

162. Garn, S.M. and J.A. Haskell, *Fat and growth during childhood.* Science, 1959. **130**(3390): p. 1711-1712.

163. Kaplowitz, P.B., *Link between body fat and the timing of puberty.* Pediatrics, 2008. **121 Suppl 3**: p. S208-17.

164. Rosenbaum, M. and R.L. Leibel, *Role of Gonadal Steroids in the Sexual Dimorphisms in Body Composition and Circulating Concentrations of Leptin1*. The Journal of Clinical Endocrinology & Metabolism, 1999. **84**(6): p. 1784-1789.

165. Davison, K.K., E.J. Susman, and L.L. Birch, *Percent body fat at age 5 predicts earlier pubertal development among girls at age 9.* Pediatrics, 2003. **111**(4 Pt 1): p. 815-821.
166. Lee, J., et al., *Weight Status in Young Girls and the Onset of Puberty.* Pediatrics, 2007. **119**: p. e624-30.

167. Garcia-Mayor, R.V., et al., *Serum leptin levels in normal children: relationship to age, gender, body mass index, pituitary-gonadal hormones, and pubertal stage.* J Clin Endocrinol Metab, 1997. **82**(9): p. 2849-55.

168. Ahmed, M.L., et al., *Longitudinal study of leptin concentrations during puberty: sex differences and relationship to changes in body composition.* J Clin Endocrinol Metab, 1999. **84**(3): p. 899-905.

169. Gardner, L.I., *Endocrine and genetic diseases of childhood and adolescence.* 1975.

170. Malina, R.M., *Tracking of physical activity and physical fitness across the lifespan.* Res Q Exerc Sport, 1996. **67**(3 Suppl): p. S48-57.

171. Ogden, C.L., et al., *Prevalence of Overweight and Obesity in the United States, 1999-2004.* JAMA, 2006. **295**(13): p. 1549-1555.

172. Nader, P.R., et al., *Identifying risk for obesity in early childhood.* Pediatrics, 2006. **118**(3): p. e594-601.

173. Cole, T.J., *Children grow and horses race: is the adiposity rebound a critical period for later obesity?* BMC Pediatrics, 2004. **4**(1): p. 6.

174. Rolland-Cachera, M.F., et al., *Early adiposity rebound: causes and consequences for obesity in children and adults.* Int J Obes (Lond), 2006. **30 Suppl 4**: p. S11-7.

175. Rolland-Cachera, M., M. Deheeger, and F. Bellisle, *ed. Obesity in childhood and adolescence*. 2002.

176. Rolland-Cachera, M.F., et al., *Tracking the development of adiposity from one month of age to adulthood.* Ann Hum Biol, 1987. **14**(3): p. 219-29.

177. Whitaker, R.C., et al., *Early adiposity rebound and the risk of adult obesity.* Pediatrics, 1998. **101**(3): p. E5.

178. August, G.P., et al., *Prevention and treatment of pediatric obesity: an endocrine society clinical practice guideline based on expert opinion.* J Clin Endocrinol Metab, 2008. **93**(12): p. 4576-99.

179. McGuire, S., *Institute of Medicine (IOM) Early Childhood Obesity Prevention Policies. Washington, DC: The National Academies Press; 2011.* Adv Nutr, 2012. **3**(1): p. 56-7.

180. Taylor, R.W., et al., *Identifying adolescents with high percentage body fat: a comparison of BMI cutoffs using age and stage of pubertal development compared with BMI cutoffs using age alone.* Eur J Clin Nutr, 2003. **57**(6): p. 764-9.

181. Niven, A.G., et al., *Maturational differences in physical self- perceptions and the relationship with physical activity in early adolescent girls.* Pediatr Exerc Sci, 2007. **19**(4): p. 472-80.

182. Kaltiala-Heino, R., et al., *Early puberty is associated with mental health problems in middle adolescence.* Soc Sci Med, 2003. **57**(6): p. 1055-64.

183. Laitinen-Krispijn, S., et al., *Pubertal maturation and the development of behavioural and emotional problems in early adolescence.* Acta Psychiatrica Scandinavica, 1999. **99**(1): p. 16-25.

184. Cumming, S.P., et al., *Physical Activity, Physical Self-Concept, and Health-Related Quality of Life of Extreme Early and Late Maturing Adolescent Girls.* The Journal of Early Adolescence, 2012. **32**(2): p. 269-292.

185. Nihiser, A.J., et al., *BMI measurement in schools.* Pediatrics, 2009. **124 Suppl 1**: p. S89-97.

186. Mirwald, R.L., et al., *An assessment of maturity from anthropometric measurements.* Med Sci Sports Exerc, 2002. **34**(4): p. 689-94.

187. Baxter-Jones, A.D.G., et al., *A longitudinal study of the relationship of physical activity to bone mineral accrual from adolescence to young adulthood.* Bone, 2008. **43**(6): p. 1101-1107.

188. Soliman, A., et al., *Advances in pubertal growth and factors influencing it: Can we increase pubertal growth?* Indian J Endocrinol Metab, 2014. **18**(Suppl 1): p. S53-62.

189. Isong, I.A., et al., *Racial and Ethnic Disparities in Early Childhood Obesity.* Pediatrics, 2018. **141**(1): p. e20170865.

190. Caprio, S., et al., *Influence of race, ethnicity, and culture on childhood obesity: implications for prevention and treatment: a consensus statement of Shaping*

America's Health and the Obesity Society. Diabetes care, 2008. **31**(11): p. 2211-2221.

Table 1. Descriptive Statistics for Age, Stature, Body Mass, and BMI at each age.

Total (n=646)					Boys (n=358)				Girls (n=288)			
Age Category	Age (years)	Stature (cm)	Body mass (kg)	BMI (kg/m²)	Age (years)	Stature (cm)	Body Mass (kg)	BMI (kg/m²)	Age (years)	Stature (cm)	Body Mass (kg)	BMI (kg/m²)
8	8.3 ± 0.2	130.4 ± 5.9	30.0 ± 6.5	17.5 ± 2.8	8.5 ± 0.2	130.9 ± 5.9	30.4 ± 6.5	17.6 ± 2.9	8.3 ± 0.2	129.6 ± 5.8	29.5 ± 6.5	17.4 ± 2.8
9	9.3 ± 0.2	136.2 ± 6.3	34.4 ± 8.2	18.6 ± 3.3	9.3 ± 0.3	136.7 ± 6.3	34.7 ± 8.1	18.4 ± 3.3	9.3 ± 0.2	135.6 ± 6.3	33.9 ± 8.2	18.3 ± 3.4
10	10.3 ± 0.3	141.9 ± 6.8	38.6 ± 9.5	19.0 ± 3.6	10.3 ± 0.3	142.1 ± 6.7	38.9 ± 9.5	19.1 ± 3.7	10.3 ± 0.2	141.6 ± 6.9	38.3 ± 9.5	18.9 ± 3.8
11	11.3 ± 0.2	147.7 ± 7.3	43.6 ± 11.1	19.8 ± 3.9	11.3 ± 0.3	147.6 ± 7.3	43.7 ± 11.2	19.9 ± 4.0	11.2 ± 0.2	147.9 ± 7.5	43.5 ± 11.0	19.7 ± 3.8
12	12.0 ± 0.2	151.8 ± 7.7	47.6 ± 12.3	20.6 ± 4.1	12.0 ± 0.3	151.7 ± 7.9	47.8 ± 12.5	20.6 ± 4.3	11.9 ± 0.2	152.0 ± 7.4	47.4 ± 12.0	20.3 ± 4.1
13	13.0 ± 0.3	158.2 ± 8.0	53.6 ± 13.6	21.3 ± 4.5	13.0 ± 0.3	158.8 ± 8.7	54.1 ± 14.2	21.3 ± 4.6	12.9 ± 0.2	157.4 ± 6.8	53.1 ± 12.9	21.3 ± 4.4
14	14.0 ± 0.3	164.0 ± 8.3	59.6 ± 14.5	22.1 ± 4.6	14.0 ± 0.3	166.3 ± 8.7	61.1 ± 15.3	22.0 ± 4.7	13.9 ± 0.2	161.1 ± 6.7	57.8 ± 13.2	22.2 ± 4.5

Values indicate mean ± standard deviation. Age Category based on whole year: 8 = 8.0-8.99; 9 = 9.0-9.99; etc.

Table 2. Average age at peak height velocity, height velocity, and biological age by maturity status.

	Boys (n=182)				Girls (n=251)			
Maturity Status	**n %**	**APHV (years)**	**PHV (cm/year)**	**Bio Age (years)**	**n %**	**APHV (years)**	**PHV (cm/year)**	**Bio Age (years)**
Early	91, 50%	11.6 ± 0.7	10.7 ± 2.1	13.9 ± 0.8	41, 16.3%	9.9 ± 0.4	9.4 ± 1.5	13.5 ± 0.4
Average	91, 50%	12.9 ± 0.3	9.9 ± 1.8	12.8 ± 0.3	186, 74.1%	11.5 ± 0.7	8.7 ± 1.5	11.9 ± 0.8
Late	/	/	/	/	24, 9.6%	12.6 ± 1.6	8.1 ± 1.9	10.8 ± 0.4

Values indicate n and (%) mean ± standard deviation. APHV = Age at Peak Height Velocity; PHV = Peak Height Velocity; Bio Age = Biological Age.

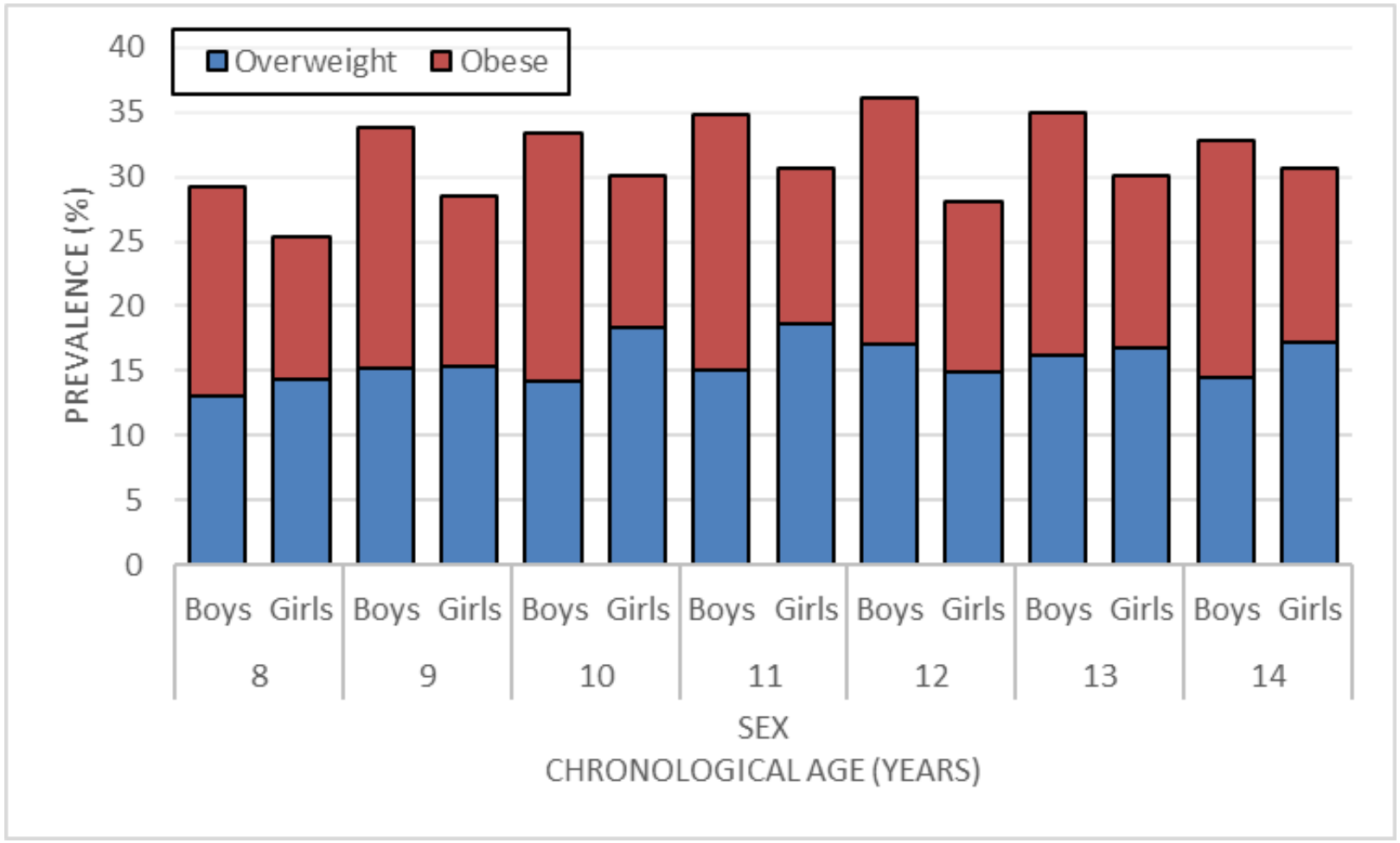
Maturity Status. For girls: Early = Peak height velocity was attained prior to 10.32 years. Average = Peak height velocity was attained between 10.32 – 12.66 years. Late: Peak height velocity was attained after 12.66 years. **For boys:** Early: Peak height velocity was attained prior to 12.46 years. Average: Peak height velocity was attained between 12.46 – 14.68 years.

Table 3. Comparison of weight classification according to standard CDC classification based on chronological age and biological age.

Standard Weight Classification Using Chronological Age	Adjusted Weight Classification Using Biological Age				
	Normal Weight (%)	Overweight (%)	Obese (%)	Underweight (%)	Total n
Total χ^2 (df=6)=527.50, p<0.0001					
Normal Weight	284 (97.3)	7 (2.4)	0	1 (0.3)	292
Overweight	20 (29.0)	48 (69.6)	1 (1.5)	0	69
Obese	0	16 (22.2)	56 (77.8)	0	72
Boys χ^2 (df=6)=218.52, p<0.0001					
Normal Weight	109 (99.1)	0	0	1 (0.9)	110
Overweight	12 (35.3)	22 (64.7)	0	0	34
Obese	0	10 (26.3)	28 (73.7)	0	38
Girls χ^2 (df=4)=314.98, p<0.0001					
Normal Weight	175 (96.2)	7 (3.9)	0	0	182
Overweight	8 (22.9)	26 (74.3)	1 (2.9)	0	35
Obese	0	6 (17.7)	28 (82.4)	0	34

Values indicate n(%).

Figure 1. Prevalence of Overweight & Obesity by Age.



2A. Boys

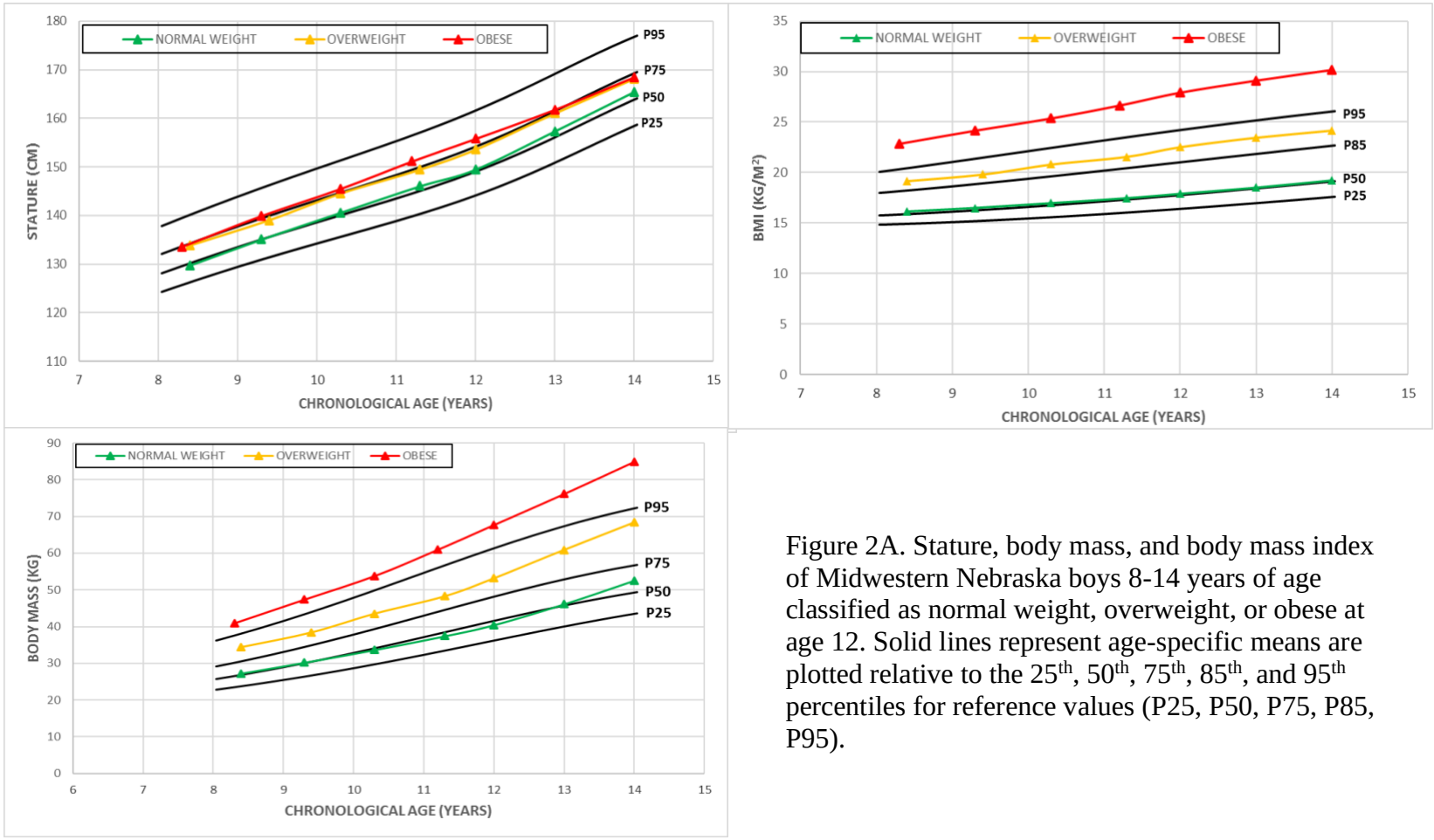


Figure 2A. Stature, body mass, and body mass index of Midwestern Nebraska boys 8-14 years of age classified as normal weight, overweight, or obese at age 12. Solid lines represent age-specific means are plotted relative to the 25th, 50th, 75th, 85th, and 95th percentiles for reference values (P25, P50, P75, P85, P95).

2B. Girls

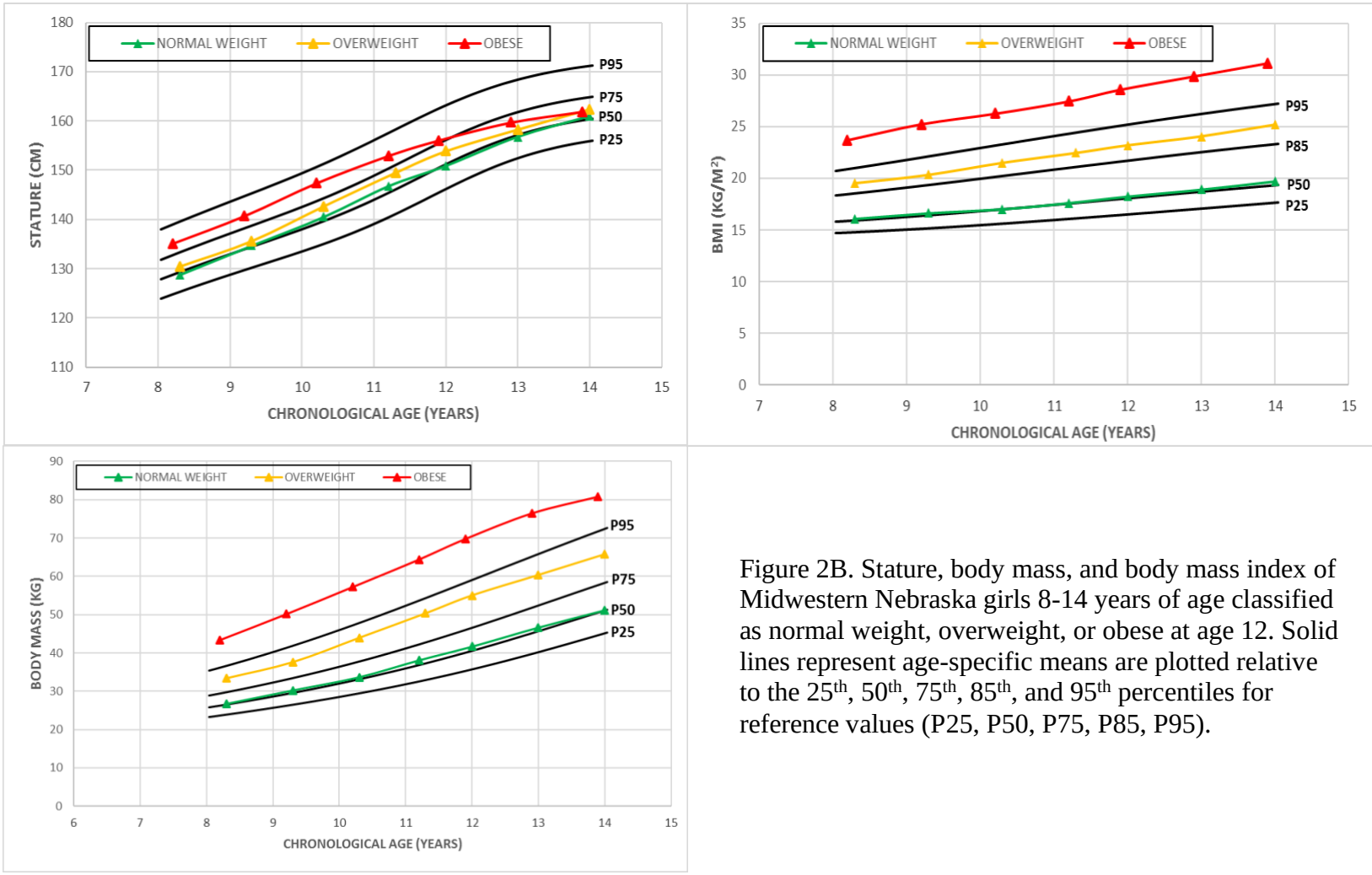
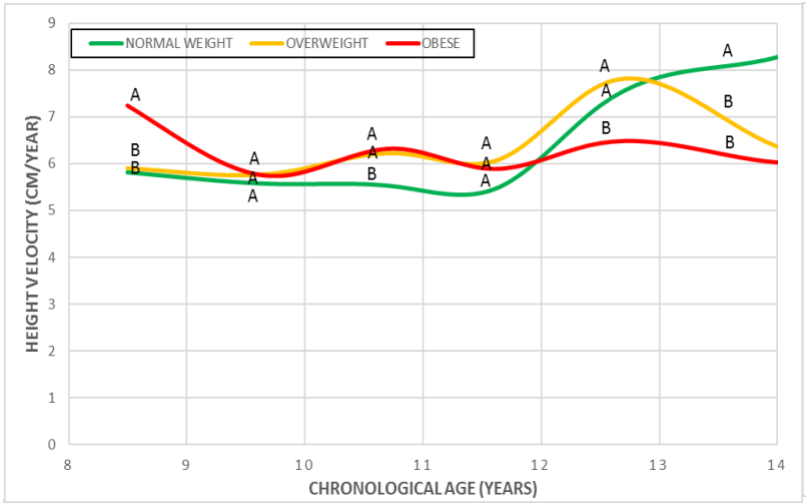
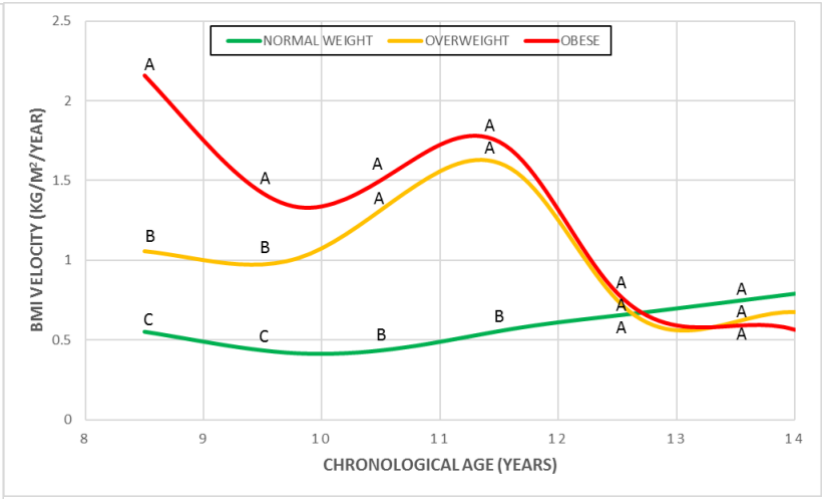


Figure 2B. Stature, body mass, and body mass index of Midwestern Nebraska girls 8-14 years of age classified as normal weight, overweight, or obese at age 12. Solid lines represent age-specific means are plotted relative to the 25th, 50th, 75th, 85th, and 95th percentiles for reference values (P25, P50, P75, P85, P95).

Growth Velocity of Midwestern Nebraska Children

3A. Boys





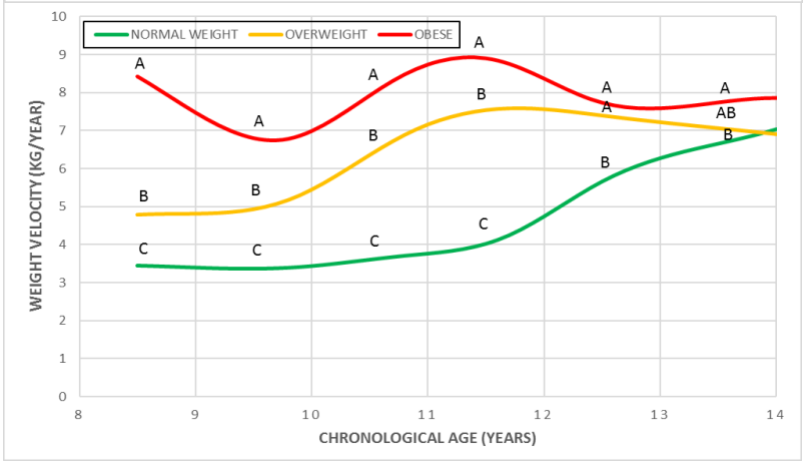
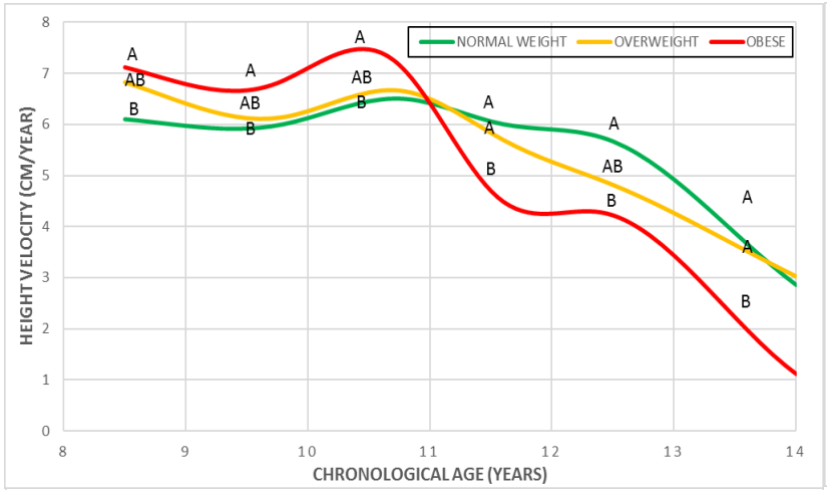
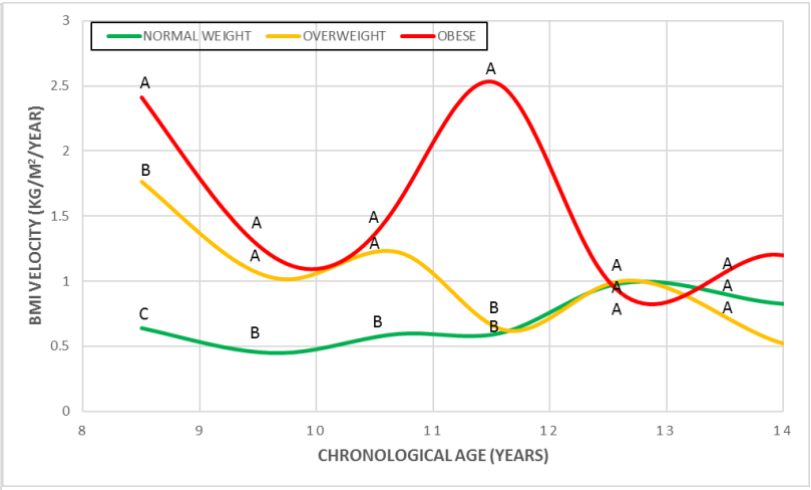


Figure 3A. Height, weight, and BMI velocity of Midwestern Nebraska boys 8-14 years of age classified as normal weight, overweight, or obese at age 12. Different letters indicate significant differences ($p<0.05$) in growth rates at each age interval.

3B. Girls





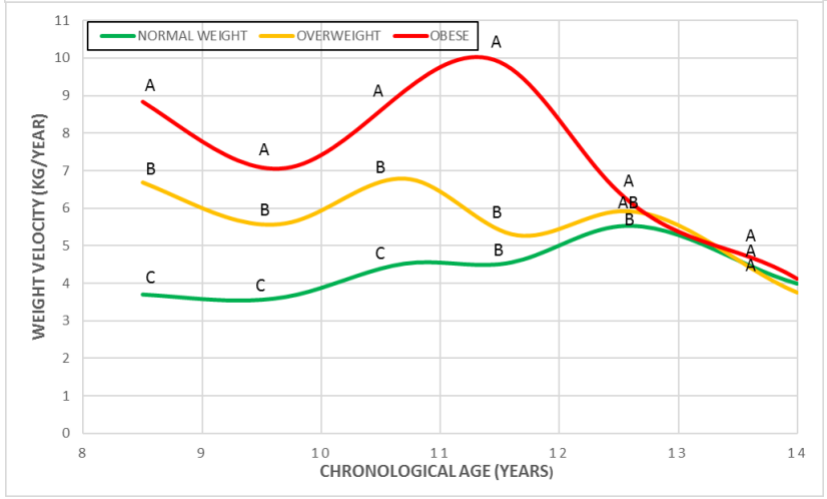


Figure 3B. Height, weight, and BMI velocity of Midwestern Nebraska girls 8-14 years of age classified as normal weight, overweight, or obese at age 12. Different letters indicate significant differences ($p<0.05$) in growth rates at each age interval.