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Christina McFadden

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Sharon L Hoerr RD, PhD
Major Professor's Signature

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A LONGITUDINAL GROWTH STUDY TO TRACK CHILDREN ON BMI-FOR-AGE GROWTH CHARTS

By

Christina McFadden

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ABSTRACT

A LONGITUDINAL GROWTH STUDY TO TRACK CHILDREN ON BMI-FOR-AGE GROWTH CHARTS

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Christina McFadden

Longitudinal growth measurements from age 6-18 years for 354 participants of the Motor Performance Study were plotted on the BMI-for-age growth charts. Most children (97%) fluctuated among major percentile growth channels and the majority fell within three or four channels. Those within four or more channels were 4.5 times more likely to be at risk for overweight (AROW) or overweight (OW) at age 16-18 years compared to those whose growth fell within fewer channels. Subjects AROW or OW at ages 6, 9, 12, or 15 years were more likely to be so at their last age than those who were underweight or normal weight at these ages and odds ratios for AROW or OW increased as age increased. Variations among three or fewer percentile channels along the BMI-for-age growth charts did not clearly identify children at risk for AROW or OW.

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I INTRODUCTION

Health professionals do not know how a normally growing child tracks along the new BMI-for-age growth charts, yet these new charts are recommended by the Centers for Disease Control and Prevention (CDC) for use to determine if children are growing properly or are at risk for underweight or overweight. The CDC asserts that a "normally growing child" will remain within the same percentile growth channel over time and that a child who moves up or down a percentile growth channel should be considered "at risk" for underweight or overweight. According to Gifford, "It has been determined that after age two, children tend to grow along a channel (percentile growth channel) due to their genetics and environment" (Gifford, 1980, pg. 7). However, this pattern has not been established longitudinally with the BMI-for-age growth charts, and the assumption and assertion that normal growth remains within percentile growth channels needs to be tested.

The number of children who are overweight is increasing dramatically in the United States and throughout the world (Ogden et al., 2001). Children who are overweight have increased risks for many serious health conditions including hypertension, impaired glucose tolerance and even Type 2 Diabetes (Whitaker et al., 1997; Freedman et al., 1999; Pinhas-Hamiel et al., 1996). It is important to identify children at risk for overweight to inform interventions that encourage healthy lifestyles. Interventions are more successful in the adoption of healthy lifestyle habits when implemented during childhood than adulthood (Story, 1999). Healthy lifestyle habits adopted during childhood could then prevent the onset of

disease in childhood or adulthood (Mei et al., 2002). There are currently no national health recommendations to intervene with children who are underweight according to the BMI-for-age growth charts (Kuczmarski et al., 2000). Accepted definitions of underweight have yet to be established (Roberts & Dallal, 2001).

Classifying children that are at a healthy weight as *overweight* or *at risk for overweight* can cause psychological damage and could lead to physiological damage, if such classification results in dieting, excessive exercise or the use any number of dangerous weight loss methods. Female adolescents report dangerous dieting practices to lose weight, even when they are not actually overweight (Krowchuk et al., 1998). While more common among females, dangerous dieting practices, eating disorders, and body dissatisfaction are becoming more common among males as well (Krowchuk et al., 1998). It is important to correctly identify a child's weight status to avoid doing harm physically or psychologically.

Before interventions can be implemented for children at risk for overweight, children at risk must be properly identified (Mei et al., 2002). The cutpoints on the BMI-for-age growth charts do not alone identify children at risk. The cutpoints used for the identification of *at risk for overweight (AROW)*, *overweight* (OW) and *underweight* (UW) are useful, but not perfect identifiers. These cutpoints have not been associated with specific health risk factors or diseases (Roberts & Dallal, 2001). In a normal distribution of the weight status of US children, some children will naturally fall into these risk categories with no adverse health risks. Additional methods to identify at risk children are necessary.

In order to identify children at risk for overweight, the association between weight status in childhood with that in adulthood has been studied. Such studies demonstrate that overweight children often become overweight adults; yet many thin children also become overweight adults and many overweight children become normal weight adults (Braddon et al., 1986; Guo et al., 1994; Must et al., 1992; Srinivasan et al., 1996; Stark et al., 1981; Thorkild et al., 1988; Whitaker et al., 1997). It is not known, however, whether or not growth patterns along the BMI-for-age growth charts can better predict adult overweight, than can childhood weight status at particular ages.

The studies on the new growth charts and construction of the growth charts themselves have been completed using mostly cross-sectional studies, with the exception of data from Ross Labs in Yellow Springs, Ohio (Guo et al., 2002). Most studies, which examined the relation of pediatric to adult weight and health status including the recent study by Guo et al., have used a childhood weight status at particular time points, but not examined the variability in children's growth percentiles or growth patterns. Because of the assumption that no variability in growth percentile channel indicates low health risk (CDC, 2000), variability in growth patterns is important to investigate. Thus, it is important to understand what growth patterns healthy children follow as their weight status is plotted on the BMI-for-age growth charts in order to properly identify those at risk for underweight or overweight. No published studies have been located which have reported the frequency of variability in BMI-for-age growth patterns of children over time.

This study will explore BMI status patterns on the BMI-for-age growth charts during children's growth. Growth charts will be plotted for 150 boys and 150 girls using selected longitudinal data from the Michigan State University Motor Performance Study (MPS) to examine how healthy, middle-income children track on the new BMI-for-age growth charts. The long-term goal of this analysis is to test the assumption or assertion that children of average BMI are those who remain in the same growth channel during growth. The aim is to help health professionals identify children at health risk, while doing no harm.

Specific Aims:

Aim 1. To compare BMI and fat status and the frequency of underweight ($\leq 5^{th}$ BMI-for –age percentile), at risk for overweight ($\geq 85^{th}$ – $<95^{th}$ BMI-for-age percentiles), overweight ($\geq 95^{th}$ BMI-for-age percentiles), and overfat ($\geq 95^{th}$ triceps and subscapular skinfold sum percentile) youth in the selected sample, to that of youth in the entire MPS and to that of a national sample of youth at ages 6, 9, 12, 15, and 18 years.

H1a: There will be no differences in BMI and fat status at each age among the selected sample and the MPS.

H1b: There will be no differences in the frequency of children underweight, at risk for overweight, overweight, and overfat at each age among the selected study, MPS, and NHANES III.

Aim 2. To define children's BMI-for-age growth patterns from ages 6-18 years[1] as: 1) staying in the same percentile growth channel; 2) increasing percentile growth channels; 3) decreasing percentile growth channels; or 4) increasing and decreasing percentile growth channels.

H2: Over half of the children will remain in the same growth channel from ages 6-18 years and the remainder will be distributed among the other growth pattern groups.

Aim 3. To identify the extent to which obesity (overweight plus overfat) and overweight at particular childhood ages measured by BMI-for-age percentile and sum of skinfold percentile and change in growth channels from age 6-18 years predicts overweight and overfat status at age 18 years.

H3a: Children who cross growth channels will have a higher risk for overweight, overfat, and obesity (overweight and overfat) at age 18 years than those who do not cross channels.

H3b: Children who cross growth channels the most times will have higher risk for overweight, overfat, and obesity (overweight and overfat) at age 18 years.

H3c: The ability of children's weight status (BMI-for-age percentile) at key ages to predict overweight at age 18 will increase with increasing age.

Aim 4. To identify the extent to which underweight in childhood and change in the growth channels from age 6-18 years predicts underweight at age 18 years.

H4a: Children who decrease growth channels will have a higher risk for underweight at age 18 years than those who do not decrease channels.

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[1] Growth patterns are defined up to age 18 years or up to the age that adult height is attained.

H4b: The ability of children's weight status (BMI-for-age percentile) at key ages to predict underweight at age 18 will increase with increasing age.

There has been only one longitudinal study to investigate the 2000 CDC BMI-for-age growth charts (Guo et al., 2000). Guo et al. studied the risks of childhood overweight with adult overweight and obesity using 347 subjects (48% male) from the Fels longitudinal data, comprised of middle-income subjects first enrolled in 1929. This proposed study aims to use more recent data than Fels and also examine variability in weight status overtime to understand the growth patterns children follow using the BMI-for-age growth charts. Such understanding should assist clinicians in their interpretation to distinguish between healthy children versus those at risk of adult overweight. For example, are children who cross percentile channels on the BMI-for-age channels really more likely to be overweight and overfat at age 18 than those who remain in the same percentile channel? Additionally, with a greater understanding of the growth charts, more clinicians may be likely to use the charts to identify children at nutritional risk without doing harm, as recommended by the Centers for Disease Control and Prevention (Kuczmarski et al., 2000).

GLOSSARY OF TERMS

Percentile: position of an individual on the BMI-for-age growth charts that indicates the percent of the reference population the individual equals or exceeds

Major percentile: selected percentiles (3^{rd}, 5^{th}, 10^{th}, 25^{th}, 50^{th}, 75^{th}, 85yh, 90^{th}, 95^{th}, 97^{th}) determined from smoothing techniques of weighted empirical percentile data points

Percentile growth channel: percentiles equal to or below a major percentile and above a lower major percentile

Growth pattern: path points follow when plotted on the BMI-for-age growth charts for individual children

At risk for overweight: $\geq 85^{th}$ and $< 95^{th}$ percentile on the BMI-for-age growth charts

Overweight: $\geq 95^{th}$ percentile on the BMI-for-age growth charts

Underweight: $< 5^{th}$ percentile on the BMI-for-age growth charts

Overfat: $\geq 95^{th}$ percentile of sum of skinfolds for age

Obese: both overweight and overfat

Tracking: the following of the growth of an individual child or group of children along the BMI-for-age growth charts by plotting longitudinal points during growth

Normal healthy growth: childhood and adolescent growth achieved when children are nourished and have no disease or deficiencies

Normal weight: BMI-for-age values between the 5^{th} and 84^{th} percentiles

Adult height: the height considered final adult height after three consecutive semiannual measurements were within 3mm

Prepubertal: stage at which a child has not yet begun the sexual maturation stages of puberty

Pubertal: stages at which a child has begun or finished puberty stages

II REVIEW OF THE LITERATURE

The topics reviewed here are those relevant to the research aims such as growth patterns of children and consequences of failure to identify abnormal growth. All literature reviewed relates to the weight status of American children, was published within the past 20 years, and is organized into the following eight subsections: 1) the trends in prevalence of childhood overweight, underweight, and stunting in the United States; 2) guidelines and recommendations for the assessment of growth status in children; 3) normal growth of children; 4) growth charts uses, development, strengths and limitations; 5) an analysis of BMI as an effective measure of adiposity in children and adolescents; 6) the health risks and disparities for the overweight child; 7) the health risks and disparities for the underweight child; 8) the prediction of overweight in adulthood from childhood weight status. Analysis of the literature on these topics will provide insight to the methods of data analysis and interpretation as well as to the significance of the research questions.

Trends in Childhood Overweight, Underweight and Stunting in the US

The prevalence of overweight, underweight and stunting among children is assessed to monitor secular trends and to make international comparisons. Underweight prevalence in the US decreased from 5.1% in the early 1970's to 3.3% by the 1990's (Wang et al., 2002). Stunting prevalence is currently estimated between 0.1% and 2.5% (Wheeler et al., 2004). Until the last 20 years, underweight and stunting have been of greater concern for early detection than

has overweight due to more immediate and severe health consequences (Wang et al., 2002). Underweight prevalence has since decreased, but the high and growing prevalence of childhood overweight has become a major public health concern in the United States and abroad because overweight and overfat status poses a threat to children's and adult's overall health (Freedman et al., 1999; Wang et al., 2002). Thus, literature reviewed in this section will focus on describing this growing increase in overweight among children as well as the importance of early identification.

According to NHANES III, 13% of US children ages 6-11 years and 14% of adolescents 12-19 years are overweight (National Center for Health Statistics, 2001; Strauss et al., 2001). This prevalence is even higher for some population subgroups, for example, 23.4% of Mexican American females aged 12-17 are overweight (Ogden et al., 2002; Adair et al., 2001). The prevalence of overweight children has increased among all race, sex, and age groups in the past four decades and preliminary data from NHANES IV indicates the prevalence continues to rise (Ogden et al., 2002; Strauss et al., 2001; Flegal & Troiano, 2000; Troiano et al., 1995).

Overweight children often become overweight adults and sometimes suffer medical complications, which will be discussed later (Whitaker et al., 1997; Freedman et al., 1999; Pinhas-Hamiel et al., 1996). Overweight interventions may yield long-term benefits (Mei et al., 2002; Epstein et al., 1990) and interventions aimed at children yield better long-term success than with adults (Epstein et al., 1998; Epstein et al., 1995; Story, 1999). In fact, the younger the

child at intervention, the more effective the intervention will likely be (Story, 1999). Therefore, it is important to identify children at risk for overweight and overweight as early as possible.

Assessing Growth Status in Children

Why do we measure children's growth status? Growth is monitored in children for two equally important reasons: 1) to monitor the health of a community to detect an abnormal prevalence of overweight, underweight or stunting, and 2) to screen individual children for health risks. Healthy People 2010 objectives address the first reason in their guidelines. Healthy People 2010 Objective 19.3 aims to reduce the proportion of overweight children and adolescents to 5% due to the alarming increasing trend in childhood overweight (HP2010, 2000). HP2010 Objective 19.4 aims to reduce growth retardation among low-income children under age 5 years from 8% to 5% (HP2010, 2000). This section focuses on the second reason as it relates to this study.

Normal growth is the most important single indicator of the health of a child (Lipman et al., 2000; Dietitians of Canada et al., 2004). In order to properly identify children with growth problems, the American Academy of Pediatrics produced guidelines for the frequency of measurements of children throughout the growing years shown in Table 1 (Lipman et al., 2000).

Table 1. Guidelines of the American Academy of Pediatrics on measurement frequency

Age	Measurement frequency
Up to 6 months	Every 2 months
6-18 months	Every 3 months
2-18 years	Yearly

(Lipman et al., 2000)

Normal growth in children is assessed clinically to help monitor health and detect growth problems early (Karlberg et al., 1999). Growth retardation or stunting is often the first sign of disease (Gotlin, 1984). In 1984 it was estimated that over 10 million children in the United States were evaluated for potential abnormal physical or sexual growth but most abnormal cases were found to be simple variations in normal growth patterns (Gotlin, 1984). Several endocrine or other diseases can be identified for treatment and risk factors can be recognized for preventative interventions when growth assessment is conducted correctly (Duck, 1996). For example, a slowly growing child may be assessed further for conditions such as renal disease, cystic fibrosis, Celiac disease, Rickets, or chromosomal disorders such as Prader-Willi Syndrome just to name a few (Duck, 1996). Along with growth problems that cause underweight in children, health professionals can screen for childhood overweight and obesity, a relatively new concern.

Normal Growth

In order to recognize growth problems in children, it is important to understand how children normally grow. Only with an understanding of normal growth can a health professional recognize abnormal growth, which is often a sign of disease or malnutrition. To search the literature on this topic, "normal growth", "sexual maturation stages" and "growth patterning" were searched on Medline; references of articles found to be relevant were collected; and the work of esteemed authors was searched, including that of Tom Lohman, Alex Roche and James Tanner.

Interpreting growth charts is difficult in the clinical setting, because not all children grow at the same rate or go through sexual maturation stages or height and weight spurts at the same time. The beginning of puberty can range from the ages of 9 to 15 years in youth. In a group of normally growing boys of the same age it is normal to find one boy who has finished his maturation cycle and another who has yet to begin. This variance in the timing of changes makes the interpretation of individual children's growth charts difficult, but the order in which these changes occur are generally the same among children of the same gender (Tanner, 1986). The general pattern of growth and the body composition changes during puberty will be reviewed in this section.

Infant growth is not reviewed because the BMI-for-age growth charts start at age two years. After the accelerated growth in infancy and prior to pubertal changes, childhood growth is fairly steady and includes gains in both height and weight. This review will focus primarily on the more relevant pubertal changes.

Linear growth, including gains in both height and weight occurs in healthy children and has traditionally been tracked along various growth charts developed in the recent past that indicate normal and abnormal growth (Kuczmarski et al., 2000). During linear growth, lean and fat mass is accumulated. Once puberty begins, marking the end of childhood, big changes occur in body composition, body size and sexual maturation. From ages 8-18 years, BMI generally increases and highly correlates with lean and fat components of the BMI; though annual changes are primarily driven by increases in the lean component (Wells, 2002; Siervogel et al., 2000). Changes in BMI during growth can be influenced by

changes in either fat or lean tissue and, unlike in adults, by changes in stature (Siervogel et al., 2000). The degree to which each component of the BMI influences the annual increases in BMI depends on the age, sex, and maturational status of the child (Siervogel et al., 2000).

Studying children in the Fels Longitudinal Study dataset Guo et al. found that total body fat (TBF) increased during growth in both males and females, but it increased at a much faster rate in females (Guo et al., 1998). TBF increased from 6.39kg at age 8 to 16.25kg at age 20 years in females (Guo et al., 1998). For males at these same ages TBF increased from 4.73kg to 9.72kg (Guo et al., 1998). Percent body fat (%BF) increased for females during growth (Guo et al., 1998). At age 20 years, females had an average %BF of 26% (Guo et al., 1998). Percent body fat also increased for boys, but only until age 14 years, when it declined until age 18 (Guo et al., 1998).

Fat free mass (FFM) increases by 10kg in both females and males between ages 8 and 14 years, but after age 14 increases in FFM are much greater in males than in females (Guo et al., 1998). Males gain 25kg of FFM from ages 14 to 18 years (Guo et al., 1998); females gain only 9kg of FFM. These findings are consistent with earlier studies that determined the gender differences in fat and lean tissue composition during puberty. Both genders increase lean body mass during puberty, but the rates and amounts are much greater in the male than in the female (Tanner 1986). Rapid increases in BMI during growth and, thus, rapid climbs along the BMI-for-age growth charts in both males and females can be due

to an unhealthy degree of fat deposition, rather than to deposition of lean body mass (Wells, 2002).

The age of pubertal events differs among children of the same sex, but the sequence of events is nearly always the same (Marshall & Tanner, 1975; Tanner, 1986). Peak height (PHV) and weight velocities (PWV) are sentinel growth events in puberty. At these times the gains in height (PHV) or weight (PWV) are accelerated over a 12-month period (Tanner, 1986). PHV indicates major body composition changes relevant to understanding growth charts. The BMI-for-age charts do not distinguish between fat and lean muscle tissue mass because body mass is composed of both. There are many changes in body composition taking place in males and females during puberty and without understanding the child's sexual maturation progression, it may be impossible to interpret their growth chart correctly. Further complicating this issue is the variance in the timing of the PHV among children of the same gender. PHV, on average, occurs anytime between ages 9.7 and 13.3 years in females and 11.7 and 15 years in males (Tanner & Davies, 1985). The average age for PHV in females is 12 years and about two years later, around age 14 years in males (Tanner, 1986).

Those who have their PHV early reach a higher peak, meaning more growth in inches per year, than those who have their PHV later (Tanner, 1986). Due to changes in body composition during growth spurts and the inability of BMI to distinguish between the lean and fat tissue compartments of the body, this rapid change in height and body mass could send the wrong signal to a health professional interpreting a child's growth chart. Fat mass accumulates prior to a

growth spurt to store provide adequate energy stores for growth. Fat storage will result in an increase in BMI due to the quickly obtained mass (Foster et al., 1977). Upon completion of the growth spurt, youth are often leaner because the energy they gained was spent on building more muscle, bone and organ mass. Because the largest changes occur in those who obtain their PHV the earliest, the charts of these children may display variability in BMI growth channels.

Puberty includes sexual maturation, which correlates highly with linear growth (Gong & Heard, 1994; Tanner 1986). The sexual maturation stage can be determined using the Sexual Maturation Rating (SMR) stages developed by Tanner (Marshall & Tanner, 1970; Tanner, 1986). These stages can also be approximated based on the timing of the PHV (Tanner, 1986). Stage 1 is defined as prepuberty and stages 2-5 describe sexual development (Tanner, 1986). By stage 5, maturation is complete (Tanner, 1986). Sexual maturation stages for girls and boys are presented in **Tables 2 and 3,** respectively.

Table 2. Sexual maturation stages for girls

Stage	Mean Age & SD of Onset	Mean Age & SD of PHV	Breast Development	Pubic Hair Growth
1			Prepubertal; nipple elevation	Prepubertal; no pubic hair
2	11.0 ± 0.5		Small, raised breast bud	Sparse growth of hair along labia
3	11.8 ± 1.0	12.0 ± 0.9	General enlargement of raising of breast and areola	Pigmentation, coarsening and curling, with an increase in amount
4	12.4 ± 0.8		Further enlargement with projection of areola and nipple as secondary mound (Menarche begins)	Hair resembles adult type, but not spread to medial thighs
5	13.1 ± 0.8		Mature, adult contour; with areola in same contour as breast, and	Adult type and quantity, spread to medial thighs

			only nipple projecting	

Mean age & SD of Onset: Roche et al., 1995; white female participants of Fels Longitudinal Study; Tanner, 1986

Table 3. Sexual maturation stages for boys

Stage	Mean Age & SD of Onset	Mean Age & SD of PHV	Genital Development	Pubic Hair Growth
1			Prepubertal; no change in size or proportion of testes, scrotum, and penis from early childhood	Prepubertal; no pubic hair
2	11.2 ± 0.7		Enlargement of scrotum and testes; reddening and change in texture in skin of scrotum; little or no penis enlargement	Sparse growth of hair at base of penis
3	12.1 ± 0.8		Increase first in length then width of penis; growth of testes and scrotum	Darkening, coarsening and curling, increase in amount
4	13.5 ± 0.7	14.0 ± 0.9	Enlargement of penis with growth in breadth and development of glands; further growth of testes and scrotum, darkening of scrotal skin	Hair resembles adult type, but not spread to medial thighs
5	14.3 ± 1.1		Adult size and shape genitalia	Adult type and quantity, spread to medial thighs

Mean age & SD of Onset: Roche et al., 1995; white male participants of Fels Longitudinal Study; Tanner, 1986

In females, pubertal stages begin between the ages of eight and 14 years (Tanner, 1986). PHV occurs earlier in females than in males during the second stage of maturation and about one year before menarche (Tanner, 1975). During this growth spurt, females gain 3.5 inches of height, but the spurt itself can last for up to two years (Cohen, 1986). Most females will reach their adult height by age 16 years (Cohen, 1986). PHV occurs at the mean age of 12.1 years in females and PWV occurs three to six months after PHV at approximately age 12 years (Barnes, 1975; Tanner, 1986). Lean body mass increases 44%, but body fat mass increases 120% at this time (Frisch, 1983). This large increase in fat mass

accretion may be crucial for the normal development of menses and regular ovulatory cycles, though many adolescent girls do not enjoy this change in their bodies (Frisch & McArthur, 1974).

In males, pubertal stages begin later than in females, between the ages of 10 and 15 years (Tanner, 1986). Growth spurts also occur later during development in males than females (Tanner & Davies, 1985; Marshall & Tanner, 1970). The PHV will coincide with sexual maturation stage 4 at approximately 14.0 years of age (Tanner, 1986). Height increases from 2.2 to 5.1 inches a year after which growth is not complete, but progresses more slowly (Cohen, 1986). In males, PWV occurs simultaneously with PHV one to three years after Stage 2 maturation (Barnes, 1975; Cohen, 1986).

The range of ages for the timing of each of these maturation and growth stages in children is due mostly to genetics, but also the degree of fat storage (Karlberg, 2002). Fatter children are taller, developmentally more advanced, and even biochemically different than their leaner counterparts (Frisch, 1985). Fatness is thought to speed adolescent maturation and low fatness delay maturation (Frisch, 1985; Garn et al., 1986, Heald & Hollander, 1965). Sexually immature children may appear underweight, compared to mature children (Frisch, 1985).

This paragraph summarizes the key points relevant to interpretation of growth charts by health professionals. When using the BMI-for-age growth charts, maturation stage is an important factor in determining a child's health risk and normal growth pattern. Many factors influence the timing of puberty and children

of different ages can be in the same stage of maturation. Children's developmental age doesn't always match chronological age because children enter pubertal stages at different times. Fatter children will enter puberty before thinner children. As children's bodies are preparing for a growth spurt, they will gain weight and fat mass to provide the body energy for growth. At this time, it is crucial not to recommend weight loss in order to permit the full potential of the growth spurt. On the BMI-for-age charts, BMI does not equal weight, and the charts cannot distinguish between fat and lean tissue. Thus, weight loss is never recommended for prepubescent children. In adults, stature is stable, but in growing children it is not. Height and weight during childhood is not always proportionate, even in normally growing children. This further complicates the interpretation of the BMI-for-age growth charts. Health professionals should take caution in interpreting growth along these charts. Because of individual differences in growth and maturation, these charts may be problematic for use with individuals vs. for groups.

Growth Charts

Growth charts are used to plot age, height, weight, length and/or BMI of children as they grow. These charts are visual, graphic tools for clinicians, researchers and parents to help monitor growth status of children. The charts are used in clinical settings due to ease of use in screening for nutritional risks such as overweight, underweight, and stunting; however, many limitations of the growth charts exist. Clinicians are often rushed or improperly trained in measuring

heights and weights and often scales and stadiometers are not calibrated properly or correct equipment is not used (Lipman et al., 2000). Thus, there is a high potential for measurement, equipment or observer error when heights and weights are measured (Lipman et al., 2000). Other limitations relate to the reference tools and cutpoints. For example, there are problems in using BMI as an indicator of weight status in children, which will be discussed in detail later, thus the BMI-for-age growth chart cutpoints are limited as tools in detecting growth problems in children in a clinical setting (Garn et al., 1986; Maynard et al, 2001).

There are several benefits, however, in using growth charts to assess children's growth over time. While growth charts cannot be used to diagnose growth or other health problems, they can be used to screen for inadequacies. They are objective and economical compared to other assessment tools (Jelliffe & Jelliffe, 1989). Growth charts allow the tracking of a child's nutritional status throughout growth and into adulthood. The history, clinical use, and importance of growth charts used in the United States are discussed next along with a detailed look at the new CDC growth charts published in 2000.

National Center for Health Statistics (NCHS) Growth References

In 1977 the NCHS developed the first national growth charts to compare children in the United States of the same age and sex (Kuczmarski et al., 2000), which replaced the Harvard growth charts or Ten State Nutrition Survey data used previously as reference data for children's growth. The NCHS charts were constructed using several large United States survey populations and were

designed to identify children at nutritional risk, either overweight or underweight (Kuczmarski et al., 2000). For children from birth to three years, weight-for-age, length-for-age and weight-for-length charts were constructed using data from the Fels Longitudinal Study (Kuczmarski et al., 2000). The Fels study was a longitudinal growth study conducted by the Fels Research Institute from 1929 to 1975 (Hamill et al., 1977). A sample of 867 mostly white, middleclass, bottle-fed infants from Yellow Springs Ohio was recruited to participate and measured quarterly at precise intervals (Hamill et al., 1977). This sample was not representative of people living in the United States nor of breastfed infants, but it was the most complete dataset of longitudinal growth available for the construction of the first NCHS growth charts (Kuczmarski et al., 2000)[1].

The three charts for males and females aged 2-18yr were weight-for-age, stature-for-age and weight-for-stature. To construct these charts, data were compiled from three cross-sectional national surveys: 1) The National Health Examination Survey (NHES) cycle II for ages 6-11 years (1963–1965); 2) NHES cycle III for ages 12-17 years (1966-1970); and 3) The first National Health and Nutrition Examination Surveys (NHANES I) for ages 1-18 years (1971-1974). Thus those growth charts for older children and adolescents were representative of the US population, but were not comprised from longitudinal data (Kuczmarski et al., 2000).

When the 1977 growth charts were first published, the premise was that the charts would be revised periodically to include new data, modify population

[1] Fels data remain the most complete set of longitudinal growth data ever collected on infants, following many subjects into their 70's.

estimates, and improve statistical quality (Kuczmarski et al., 2000). Secular changes in growth patterns and lifestyle of American children imply that growth charts need to be updated and reevaluated periodically (Karlberg et al., 1999).

The first growth chart revisions were published in 2000 after limitations of the 1977 charts were investigated, reported and addressed. Most limitations of the older charts related to the infant sample. Limitations of both sets of charts are listed here. 1) The charts for ages 0-3 years were constructed from a small sample (1037) and were not nationally representative. 2) Observations for ages 0-3 years were recorded at three-month intervals, although one-month intervals were used in the growth charts. 3) Distributions of birth weights have changed since the Fels study was conducted. 4) Infants in the Fels study were predominantly formula fed, and, therefore were heavier and had different growth patterns than would a sample, including more breastfed infants (Kuczmarski et al., 2000). 5) Growth at extreme percentiles beyond the 5^{th} and 95^{th} percentiles could not be assessed. 6) There were age limitations of the weight-for-stature references, which did not go beyond puberty. 7) Growth after age 17 could not be assessed. 8) The most important limitation of the 1977 infant charts was that the recumbent length measurements used in the Fels study and the stature measurements used by NCHS data sets yielded inconsistent percentile estimations when transitions were made between the two types of measurements for children 24 and 36 months of age (Kuczmarski et al., 2000). All these limitations were addressed when the revised charts were constructed, though the new charts have limitations of their own, which will be discussed (Kuczmarski et

al., 2000) after the revisions in the 2000 growth charts are reviewed (Kuczmarski et al., 2000).

The CDC Growth Charts 2000

The Center for Disease Control and Prevention (CDC) revised the NCHS growth charts in 2000 (Kuczmarski et al., 2000). The data used to construct all 2000 growth charts were gathered by NCHS from 1963-1994 and included the following sources: National Health Examination Survey (NHES) II, III, NHANES I, II, III, US Vital Statistics, Wisconsin and Missouri Vital Stats, Fels Longitudinal Survey, and the Pediatric Nutrition Surveillance System (Kuczmarski et al., 2000). A detailed table of what surveys were used for each chart is described elsewhere (Kuczmarski et al., 2000; and online at: http://www.cdc.gov/growthcharts).

For the 0-3 year old growth charts data from the Fels Longitudinal Study were replaced with those for nationally representative surveys to eliminate disjunctions between curves for infants and children (Kuczmarski et al., 2000). All charts, except the infant charts, were extended from age 18 to age 20 years (Kuczmarski et al., 2000). The new growth charts included a Body Mass Index (BMI = kg/m^2)-for-age charts recommended for children 2-20yr (Kuczmarski et al., 2000). This allows for tracking of growth from childhood into adulthood and the old charts do not measure children after puberty, thus it is not possible to compare overweight and underweight prevalence between the charts of this age group. The new charts offer an opportunity to assess risk for overweight and overweight in children using the new BMI-for-age charts (Kuczmarski et al., 2000). The 3^{rd} and 97^{th} percentiles were added to all charts and the 85^{th} percentile was added to the

BMI-for-age and weight-for-stature charts (Kuczmarski et al., 2000). The weight-for-stature charts were revised to screen children only from ages 2-5 years (Kuczmarski et al., 2000).

The newer charts have slightly higher cutoff points, which may generate a lower prevalence of overweight among older children, but not for preschool aged children (Stauss & Pollack, 2001; Flegal et al., 2002). Strauss studied 6-12 year olds from the NHANES I data and found the prevalence to be 13.3% with the new charts vs. 15.6% with the old charts (Stauss & Pollack, 2001). Flegal et al. found contradictory results when comparing overweight prevalence of children ages 2-5 determined by the BMI-for-age and weight-for-stature charts (Flegal et al., 2002). In their study, more children were classified as overweight on the BMI-for-age charts, but fewer children were classified as underweight on the BMI-for-age charts (Flegal et al., 2002). The differences were more pronounced in girls than in boys (Flegal et al., 2002).

The old and the new charts are clearly not interchangeable. The age groups compared in the Strauss and Pollack (6-12 years) and the Flegal et al. (2-5 years) studies were different, which may explain the contradictory results in prevalence of overweight and underweight found using the old and new charts. It is possible that results of prevalence would differ by age on the two charts. Thus, at one age prevalence of either overweight or underweight would be greater on one chart than the other and at another age, prevalence would be lesser than the other.

BMI-for-age cutpoints

The cutpoints for nutritional risk according to the BMI-for-age charts are represented in **Table 4**. A consensus panel recommended these cutpoints in 1997 based on the rationale that the 95th percentile corresponds with a BMI of 30, and the 85th percentile corresponds with a BMI of 25 in a young adult (Barlow & Dietz, 1998). The term "overweight" and "at risk for overweight" were chosen versus "obese" and "overweight" because of the negative connotations associated with the word "obese" and also because weight reduction is usually contraindicated for growing children (Barlow & Dietz, 1998).

Table 4. Risk indicators for the 2000 CDC Growth Charts

Index	Cutpoint	Nutritional Status Indicator
BMI-for-age	≥95th	overweight
weight-for-stature	>95th	overweight
BMI-for-age	≥85th and <95th	at risk for overweight
BMI-for-age	<5th	underweight
weight-for-stature	<5th	underweight
height-for-age	<5th	short stature

(CDC, 2000)

The underweight cutpoint of <5th percentile was based on recommendations by the World Health Organization Expert Committee on Physical Status (World Health Organization, 1996). Obtaining consensus on cutpoints was challenging, however, because it was and is difficult to link weight status with chronic disease outcomes in children and youth (Troiano et al., 1995).

One study, reported by Lohman did find direct evidence linking a percent-fat level to the risk of coronary heart disease in children after a cardiovascular risk factor study was conducted as part of the Bogalusa Heart Study (Williams et al., 1992; Lohman, 1992). The indicator values were determined for children 5-18 years of age with the greatest risk for cardiovascular disease based on systolic and diastolic blood pressure, total cholesterol and lipoprotein cholesterol ratios

(Lohman, 1992). These values, represented in **Table 5**, are now used to detect overfat in children ages 5-18 years, but do not take sexual maturation stage into account (Lohman, 1992). The same percent fat values apply to adult men (25%), but for adult women, the value (32%) is higher than for girls (30%) (Lohman, 1992).

Table 5. Indicators of excess fat in children

	% Body Fat
Boys	>25%
Girls	>30%
	Skinfolds
Boys	≥95th percentile
Girls	≥95th percentile

(Lohman, 1992)

Validity of using BMI-for-Age to Identify Overweight or Underweight Children

Growth charts are used in clinics and pediatric offices to track growth in children and to determine if a child may be at nutritional risk. The CDC recommends using the BMI-for-age growth charts to screen for nutritional risk in children over 2 years of age (Kuczmarski et al., 2000). Although the CDC recommends replacing the weight-for-stature charts with the new BMI-for-age charts, Flegal et al. found that these charts are not interchangeable and do not produce the same results (Flegal et al., 2002).

It is important to accurately interpret BMI-for-age charts because most overweight identifications take place in clinical settings and interventions can be more effective in children than adults (Mei et al., 2002; Gotlin, 1984). A quick measure of adiposity that does not miss children with true excess stores of body fat and does not falsely mislabel healthy children as overweight or obese is ideal. The new BMI-for-age growth charts should increase in use, because they are

recommended by the CDC to screen children for underweight, at risk for overweight and overweight. Thus, an understanding of normal growth patterns compared to the growth chart channels and risk indicators of the charts is warranted.

The new charts were designed using mostly cross-sectional data although children are to be tracked longitudinally in clinical records (Kuczmarski et al., 2000). Cole suggested that using cross-sectional based charts to interpret longitudinal growth of children might be inappropriate because of possible misinterpretation (Cole, 1994). However, nationally representative longitudinal growth charts are not practical, because they would take much longer to complete and require more resources than cross-sectional charts (Karlberg et al., 1999). Although the BMI-for-age growth charts are recommended by the CDC to screen children, many limitations do exist in using BMI to screen for overweight and at risk for overweight. Many factors contribute to a child's predicted status on the growth chart, for example, stages of maturation and lean body mass, and must be considered when interpreting plots.

The BMI-for-age charts have been compared to the percent body fat indicators discussed by Lohman in a few studies. Mei et al. compared the BMI-for-age charts and the weight-for-stature charts to Lohman percent body fat indicators measured by DEXA and by skinfold percentiles. While this study investigated the accuracy of the two charts in identifying underweight and overweight children, the underweight data will not be discussed here because this study addresses the

15th percentile and the BMI-for-age growth charts identify a child as underweight at or below the 5th percentile.

Using NHANES III data the investigators found that both charts identified overweight equally well in children ages 3-5 years. For overweight, the sensitivities were 88.5 for both charts at this age group (Mei et al., 2002). The sensitivity measures the ability of the charts to correctly identify those that are overweight. If the sensitivity value was 100, that would mean that the chart correctly identified all of those that are overweight. The corresponding specificities were 79.4 for the BMI-for-age and 88.2 for the weight-for-height charts (Mei et al., 2002). The specificity measures the ability of the charts to correctly identify those that are not overweight. If the specificity value was 100, that would mean that the chart correctly identified all of those who are not overweight as being not overweight.

For children aged 6-19 years, BMI-for-age was better than weight-for-stature in identifying overweight using percent fat cutpoints measured by DXA as outcome criteria (Mei et al., 2002). The sensitivities of the BMI-for-age charts to identify overweight in this age group ranged from 98.6 to 100. The sensitivities for the weight-for-stature charts were between 95.8 and 100, but it is important to note here that these charts do not track children after puberty and special measures were taken to extend these charts to cover the ages of the BMI-for-age charts to make comparisons (Mei et al., 2002). The corresponding specificities for the BMI-for-age charts ranged from 67.7 to 72.2 and 74.7 to 70.8 for the weight-for-height charts (Mei et al., 2002).

Mei et al. supported using the BMI-for-age charts to assess overweight and underweight children age 2-19 years after comparing the charts to other body composition indexes to screen for underweight and overweight children (Mei et al., 2002). Their findings do not address the clinical utility of the 5th and 85th percentiles of BMI as cutpoints for risk, but only the validity of BMI as an indicator for body fatness (Mei et al., 2002). Nevertheless, they concluded that the BMI-for-age growth charts should help researchers and practitioners track overweight and underweight from early childhood into adulthood (Mei et al, 2002).

In a study conducted by Himes and Bouchard, the sensitivities were much lower than in the Mei et al. study. This study was conducted in 1989, prior to the BMI-for-age chart publication. This study was also conducted prior to Lohman's indicators of overweight, and instead the investigators used the 90th percentile of percent body fat for age and sex relative to distributions for Cincinnati youths (Himes & Bouchard, 1989). The sample consisted of Canadians of French descent. For male and female children and adolescents ages 8 to 19 years the sensitivities were 29% and 23%, respectively for BMI's >85th percentile (Himes & Bouchard, 1989). The matching specificities were 99% and 100% (Himes & Bouchard, 1989). This study was done before the BMI-for-age charts were constructed, but utilized a percentile category formulated in much the same way the new CDC charts were constructed. From this study, it appears that BMI-for-age was good at detecting those not overweight, but not very good at detecting those who were overweight. While the specificities for identifying children that are not overweight are high, the sensitivities are not, thus, many children may remain

overweight without identification and may miss out on beneficial interventions. The authors concluded that the triceps skinfold measure should be used to indicate overweight in boys, but that the BMI is the best single anthropometric indicator of overweight in girls (Himes & Bouchard, 1989). These findings conflict with the Mei et al. study discussed earlier.

To date it is unclear how accurately the cutpoints on the BMI-for-age charts identify underweight and overweight children due to contradictory findings. The Himes and Bouchard study tested the sensitivities and specificities of Canadian children using the 90th percentile of Cincinnati children's percent body fat by age and sex. This sample cannot be used to represent American children, nor can it compare to the Mei et al. study because it utilizes a different definition of overweight than the Lohman percent body fat values, which were used in the Mei et al. study.

Another important consideration when using the BMI-for-age growth charts is interpreting the growth of early maturers and sporadic growth in height patterns (Wang & Adair, 2001). Early maturers tend to have their growth spurt earlier than others, which could complicate the interpretation of their BMI-for-age plots (Adair & Gorden-Larsen, 2001). A growth spurt is often preceded by weight gain just before an increase in height (Riumallo & Durnin, 1988). An early growth spurt may lead to misclassification as overweight status at an early age (Adair & Gorden-Larsen, 2001). Conversely, late maturers will have their growth spurt later and this may lead to underestimations of overweight among later maturing children (Adair & Gorden-Larsen, 2001). It is important to determine when the

growth spurt has occurred or will occur to correctly interpret BMI-for-age charts (Adair & Gorden-Larsen, 2001).

Additionally, some individuals' maturational and skeletal growth patterns may differ from the reference data populations (Wang & Adair, 2001). Careful evaluation of these growth patterns is necessary in order to avoid misclassifications of some youth (Wang & Adair, 2001).

Benefits and Implications of Using the BMI-for-age Charts in Clinical Settings

It is important to realize that one set of growth standards cannot be sufficient in diagnosing weight issues or identifying those with weight concerns because there are genetic and ethnic differences in growing children in the US (Kuczmarski et al., 2000). One growth standard such as the BMI-for-age charts cannot alone address these issues, but the differences are considered small enough that growth problems can be sufficiently identified using these charts as screening tools (Kuczmarski et al., 2000). A better understanding of how children track longitudinally along the BMI-for-age growth charts using a new data set may lead to improved interpretation of the charts and accurate identifications of those at risk for overweight or overweight in clinical settings.

BMI as measure of adiposity in children and adolescents

Acceptance of using BMI as an indicator for children and adolescents is increasing, because it captures changes in weight and height in relation to age and it can be used continuously from age 2 to age 20 (Flegal et al., 2002). BMI is

now generally considered the most convenient proxy for adiposity among adolescents (Himes & Bouchard, 1989; Adair et al., 2001).

There are special limitations for using BMI as a nutritional indicator in children and adolescents. Garn et al. found that BMI is not independent of stature for these ages, does not distinguish between fat and lean tissue, and is dependent on leg length, frame size, lean tissue and ethnicity (Garn et al., 1986).

BMI is considered independent of stature in adults, but is not so in children and adolescents because they are growing (Garn et al., 1986). Because growth occurs in children disproportionately, changes in fat-free mass (FFM), limb length and sexual maturation complicate BMI interpretation for children (Daniels et al., 1997; Guo et al., 1997). Maynard found correlations between BMI and percent body fat (%BF) in boys moderate to high, but lower for girls. This group also reported that BMI and FFM were more strongly correlated than BMI and %BF in girls age 8-13 (Maynard et al., 2001). Moreover, they found BMI and stature to be related for early adolescent boys, but the results were less clear for girls (Maynard et al., 2001). Therefore, young adolescents may have a large BMI due to tallness instead of due to adiposity. Children with increased adiposity are often taller than their shorter peers.

BMI changes in boys age 12-17 were found to be exclusively due to increases in FFM (Maynard et al., 2001). Adair also found that BMI and percent body fatness did not have the same relationship across all racial and ethnic groups (Adair et al., 2001). Daniels found that %BF was more strongly correlated with sexual maturation stage than with age (Daniels et al., 1997). Maturation

misclassifications may result in overestimations of overweight prevalence among early maturers and underestimation in late maturers (Adair et al., 2001). In Adair's study, early maturing girls were nearly twice as likely as average maturing girls to be determined overweight according to their BMIs (Adair et al., 2001).

Daniels demonstrated that the location of fat on the body must to be considered when using BMI (Daniels et al., 1997). Individuals with central adiposity will have a lower BMI than individuals with peripheral adiposity when they have equivalent percent body fat (Daniels et al., 1997). Finally, some are concerned with using BMI-for-age because it defines overweight by a statistical cutpoint without considering health-related consequences (Maynard et al., 2001).

Health risks and disparities for the overweight child

Many overweight children will not have health consequences until later in life if they remain overweight as adults (Must & Strauss, 1999), however, there are some complications that occur in some overweight and obese children. Psychological and social health can also be compromised along with the physical health in the overweight child. A detailed description of common and rare health risks and disparities that affect overweight children is presented here. To investigate the literature of this section, several review articles were located. From these reviews the original research articles were identified and several are presented here.

The physical health complications an overweight child may experience include cardiovascular, orthopedic, neurological, pulmonary, gastroenterological, and

endocrine (Must & Strauss, 1999). Of overweight children ages 5-11 years, 20-30% have an increased systolic or diastolic blood pressure (Figueroa-Colon et al., 1997). Previously, Rames et al. found that of all children 5-18 years, 1% had elevated blood pressure, and 60% of those were overweight (Rames et al., 1978). Adolescents above the 75th percentile for BMI are eight and a half times more likely to suffer from hypertension as adults than leaner adolescents (Srinirvasan et al., 1996). Total cholesterol and LDL cholesterol levels in adulthood that are dangerous to cardiovascular health are associated with adolescent obesity, especially in males (Lauer et al., 1988; Caprio et al., 1996). In 1997, Gutin et al. found that obesity is the main factor responsible for cardiovascular risk factors in the obese child (Gutin et al., 1997).

Orthopedic complications include slipped capital epiphyses, bilateral slipped capital epiphyses, and Blount's disease. Slipped capital epiphyses occurs in overweight children due to the pressure of their weight on their bones. The femoral head can be irreparably damaged when it is dislocated from the femoral growth plate (Must & Strauss, 1999). Between 50-70% of the people with this complication are obese (Kelsey et al., 1972; Wilcox et al., 1988) and this condition is found at younger ages in obese children than in non-obese children (Loder et al., 1993). Blount's disease is bowing of the legs and is caused by weight pressure or unequal weight bearing while the bones are growing (Must & Strauss, 1999). Eighty percent of children with Blount's disease are obese (Dietz et al., 1982).

Pseudotumor cerebri is a neurological syndrome most often found in middle-aged females, but sometimes found in overweight children (Must & Strauss, 1999). Symptoms of this intracranial hypertension are headaches, vomiting, blurred vision and diplopia (Must &Strauss, 1999). Corbett et al. found that 90% of the 57 patients reviewed with the condition were obese and 15% developed it before the age of 20 years (Corbett et al., 1982). Of children with pseudotumor cerebri, 30-80% are obese (Scott et al., 1997; Weisberg & Chutorian, 1977).

Though the relationship between weight and asthma is unclear, 30% percent of the obese children in a hospital-based weight control program suffer asthma (Unger et al., 1990). A similar prevalence of sleep apnea and abnormal sleep was found in studies by Mallory et al. and Marcus et al. (Mallory et al., 1989; Marcus et al., 1996). Of 41 severely obese children, one third had symptoms of sleep apnea and one-third had abnormal sleep patterns (Mallory et al., 1989). Silvesti et al. found the prevalence of abnormal sleep patterns to be much greater, 94%, in the obese children they studied (Silvesti et al., 1993). Along with ventilation and respiration problems, learning and memory function may also be affected in obese children with sleep apnea (Rhodes et al., 1995).

Pickwickian syndrome is a condition in which severe obesity is associated with hypoventilation, drowsiness, an abnormal increase in number of red blood cells, and ventricle failure due to hypertrophy (Must & Strauss, 1999). This condition is serious because it is related to pulmonary embolism and can cause sudden death in children, but the prevalence of this syndrome in children is unknown (Must & Strauss, 1999; Riley et al., 1976).

Gallstone formation is increased in obese individuals because the ratio of cholesterol to bile acid and phospholipids secreted in the bile is increased (Shaffer et al., 1977). In the Pima Indians, an obese population in the southwestern United States, the cholesterol content of bile increases after age 13 years, especially in females (Bennion et al., 1979). By age 30 years, the prevalence of gallstones is about 70% in this population (Sampliner et al., 1970). When other medical complications are not present, obesity accounts for the majority of gallstones in children (Must & Strauss, 1999).

Steatohepatitis, sometimes associated with liver fibrosis and liver cirrhosis, is found in an alarming prevalence in obese children (Must & Strauss, 1999). Twenty to 50% of obese children show evidence of steatohepatitis in ultrasound, transaminase, or laboratory testing (Kinugasa et al., 1984; Tominaga et al., 1995; Tazawa et al., 1997; Baldridge et al., 1995). Weight reduction can help normalize hepatic enzymes (Vajro et al., 1994).

Insulin resistance and hyperandrogenemia is found in obese children (Richards et al., 1985). Acanthosis nigricans is associated with glucose intolerance in overweight children and is found in as many as 25% of obese patients (Dietz, 1997). Higher levels of total cholesterol, low density lipoprotein (LDL) cholesterol, and triglycerides is associated with insulin resistance in obese children (Bergstrom et al., 1996; Steinberger et al., 1995; Jiang et al., 1995). Insulin resistance is also associated with non-insulin dependent diabetes mellitus (NIDDM) (Must & Strauss, 1999). Of those children whose BMI was above the 75th percentile in the Bogalusa Heart Study population, 2.4% developed NIDDM

before age 30 years, compared with none of the lean adolescents (Srinivasan et al., 1996).

Morbidity from gout in men is associated with adolescent overweight (Power et al., 1997). In a study done by Must and colleagues, men who were overweight adolescents were three times more likely to report having gout than men who were of an average weight in adolescence (Must et al., 1992).

Menstrual problems are often thought of in severely thin children, but they also occur in obese children (Must & Strauss, 1999). Polycystic ovary syndrome is associated with obesity in children and includes oligomnorrhea or amenorrhea, insulin resistance, hirsuitism, acne, and acanthosis nigricans (Must & Strauss, 1999). Polycystic ovary syndrome is common among obese adult women (Balen et al, 1995; Goldzeiher et al., 1962; Dunaif et al., 1988), but the prevalence in adolescents is unknown (Must & Strauss, 1999). Menstrual problems may be difficult to detect, but hormonal patterns associated with this syndrome are described more and more in obese adolescents (Lazar et al., 1995; Richards et al., 1985).

As discussed in the predicting overweight from childhood to adulthood section, sometimes, overweight children become overweight adults. Morbidity and mortality may be elevated in adults who were overweight or obese adolescents (Must et al., 1992; Hoffmans et al., 1988; Gunnell et al., 1988). In adulthood, overweight and obesity is a risk for many diseases and conditions such as cardiovascular disease, Type 2 Diabetes, hyperlipidemia, gall bladder disease, osteoarthritis, and some cancers (Burton et al., 1985).

Along with the physical complications found in overweight and obese children, social stigmatizations are also found. School children in the fifth and sixth grade prefer to play with an overweight child the least compared with children with other conditions such as being in a wheel chair (Latner & Stunkard, 2003; Richardson et al., 1961). Obese people are also stigmatized as having undesirable social behaviors such as being undisciplined, overindulgent, and social deviance behaviors by the public and even the scientific community (Johnston, 1985; DeJong, 1993).

A low self-esteem is not common among overweight and obese children, but it often develops in adolescence and continues into adulthood (Kaplan & Wadden, 1986; Sallade, 1973; Stunkard & Burt, 1967). This decrease in self-esteem may be due to early maturation, but results are unclear (Brooks-Gunn, 1988; Neumark-Sztainer et al., 1997). Neumark-Sztainer found that overweight boys are at greater risk of having suicidal tendencies than boys of an average weight (but not after controlling for socioeconomic variables) and severely overweight girls have a greater risk than moderately or average weight girls (Neumark-Sztainer et al., 1997). The investigators also found that the risk of having a poor emotional well-being was greater in severely overweight boys than average weight boys (Neumark-Sztainer et al., 1997).

Having a negative self-esteem due to a negative body image increases the risk of eating disorders in white girls (Attie & Brooks-Gunn, 1989). Pressure to be thin affects girls more than boys in the US (Rolls et al., 1991; Neumark-Sztainer et al., 1997). Obese girls are commonly concerned with their body image and these

feelings along with a fear of rejection may follow the adolescent into adulthood (Monello & Mayer, 1963; Stunkard et al., 1967; Neumark-Sztainer et al., 1997). Overweight youth are more likely to diet and to use unhealthy weight loss practices than their average weight peers (Neumark-Sztainer et al., 1995). Binge-eating and chronic dieting were reported more among moderately overweight and severely overweight youth than average weight youth, however vomiting was not associated with weight status (Neumark-Sztainer et al., 1995). The prevalence of binge-eating disorder in morbidly obese adolescent girls is similar to that of obese adult women, 30%, found in one study (Berkowitz et al., 1993). Eating disorders among overweight woman are increasing in prevalence (Yanovski, 1993).

Several studies have found that when women are obese adults, they often endure a decrease in social and economic status (Goldblatt et al., 1965; Gortmaker et al., 1993; Sobal & Stunkard, 1989). Discrimination towards overweight and obese persons is a factor in this decrease in status as well as the low self-esteem and lack of confidence that may manifest in these individuals (Must & Strauss, 1999). Even as youth, overweight girls are more likely to be concerned about future job expectations (Neumark-Sztainer et al., 1997).

Overweight children are often treated as much older than they are (Dietz, 1997). This may lead the child to feel inadequate in meeting expectations of others and the child may become socially isolated, especially with regards to adults outside the family (Dietz, 1997). The early maturation that leads to this treatment in young overweight children may also lead to eating disorders (Attie & Brooks-Gunn, 1989).

Of the several complications that can affect the obese or overweight child, some may be life threatening. These health risks raise serious concerns for American children and the economic future of the United States. Obesity and overweight is increasing and the prevalence of the above conditions is also expected to increase (Must & Strauss, 1999). More long and short-term consequences of childhood overweight and obesity may become apparent while prevention and treatment strategies, hopefully, follow. Early recognition of significant weight gain in children may help prevent some of these complications. As this epidemic expands, it is crucial to examine the tools available for the identification of these risks, such as the BMI-for-age growth charts.

Health risks and disparities for the underweight child

Several diseases and conditions can lead to a child's growth failure or faltering including those of endocrine or renal origin, liver diseases, systemic conditions, trace mineral deficiencies, anorexia nervosa, and skeletal problems. These can lead to growth failure, a drop in growth chart percentiles and, at times, severe health conditions. Psychological disparities can also occur in the underweight child. A detailed description of the most common conditions and diseases causing underweight in childhood will be discussed here.

In the United States, approximately 25% of hospitalized children are undernourished (Hendricks et al., 1995). Underfat children grow at a slower rate compared to normal fat children, and their sexual maturation is often delayed (Beunen et al., 1994). Though many persons strive for thinness in this country and

in many parts of the world, it is not the norm and may not always be socially acceptable. Restricting calories during childhood could result in growth failure or delayed physical maturity whether the child is overweight, normal weight or underweight (Pugliese et al., 1983). Caloric restrictions during growth limit fat stores, growth in stature and lean body mass growth. Deliberate calorie restriction, in athletes or those desiring to be thin, or malabsorption states may have this affect during childhood. Sexually immature children may appear underweight when compared to mature children, which is a problem with the BMI-for-age growth charts discussed earlier.

Restricting calories may be due to a fear of fat even in children. The fear of fat and of gaining weight begins as early as five years old (Feldman et al., 1988). An alarming percentage of adolescent girls, 83%, believe they are too fat when in fact they are at a normal weight (Feldman et al., 1986). Dieting is common even among children with 50% of third grade girls and almost 70% of adolescent girls reporting dieting (Moses et al., 1989; Maloney et al., 1989; Hill et al., 1994). Dieting schoolchildren have been found to be eight times more likely to develop an eating disorder when compared with those that do not diet (Patton et al., 1990).

Anorexia Nervosa (AN) and Bulimia are present in approximately 1-5% of Americans (Kurtzman et al., 1989). This percentage may be underreported. AN is due to the voluntary restriction of food intake often present with excessive exercise, relentless pursuit of thinness, food obsession, intense fear of fat, and distorted body image (Cohen, 1986). This disease may increase due to the

cultural value of thinness in the United States. The onset of AN is usually prepubertal, during adolescence or young adulthood (Cohen, 1986). Dieting sometimes follows a traumatic event or crisis as a way of exerting control on life (Bachar et al., 2002). Treatment of all eating disorders involves a team of professionals including physicians, dieticians and psychiatrists.

Systemic diseases and conditions, such as inflammatory bowel disease, can impair nutrient utilization results in weight loss and eventual impaired growth (Wine et al., 2004). Primary malnutrition can directly cause growth failure in children (Komrower, 1964). Malabsorption leads to poor weight gain in children causing nutrient and calorie loss resulting in secondary malnutrition and limited growth.

Intestinal diseases, such as Crohn's disease can cause anorexia, postprandial pain, malabsorption or the need for steroids (Wine et al., 2004). Each of these conditions could lead to impaired growth and maturation (Wine et al., 2004). The location of the affected bowels may further influence growth or determine the degree to which growth is affected (Wine et al., 2004).

Celiac disease is a common problem resulting in underweight in childhood (Catassi and Fasano, 2004). Inflammation due to the gluten intolerance leads to intestinal, pancreatic, and gall bladder dysfunction leading to diarrhea, malabsorption, and impaired growth (Catassi and Fasano, 2004). When gluten is eliminated from the diet and the diet is closely monitored, normal growth can be sustained.

Food allergies can have effects on growth similar to Celiac disease (Christie et al., 2002). Children allergic to milk and children with multiple food allergies are at the greatest risk for malnutrition (Christie et al., 2002). It is important that parents and children are able to clearly identify foods that contain and foods that do not contain specific allergens and thus, preventing growth problems (Christie et al., 2002).

Cystic Fibrosis is one of the most common lethal inherited disorder of individuals of Caucasian decent (CDC, 2004). If left untreated or undiagnosed, malnutrition due to malabsorption and digestion dysfunction are common problems (Hendricks et al., 1995). Pancreatic insufficiency is another common problem in those afflicted causing many patients to be deficient in fat-soluble vitamins (Baker et al., 2005).

Other fairly common diseases that can result in growth failure include renal disease, chromosome abnormalities, and hypothyroidism. End stage renal disease can result in severe growth failure (Nisset et al., 2004). Moderate catch-up growth is possible after renal transplants, yet final height is still reduced in some patients (Nisset et al., 2004). Down Syndrome, Turner's Syndrome, and Klinefelter Syndrome are the most common chromosomal abnormalities (Tyler and Edman, 2004). Down's Syndrome is often recognized at or even before birth, while the others are diagnosed later in life or not at all (Tyler and Edman, 2004). Syndrome-specific growth charts are recommended for use with these children because their growth is very different from those with normal chromosomes (Tyler and Edman, 2004). Physical and cognitive development is influenced by thyroid

hormone levels (Amer, 2005). Variants of these hormones lead to growth failure (Amer, 2005).

Predicting overweight in adulthood from childhood

To review the literature investigating the prediction of adult overweight from childhood overweight, the following keywords were searched on Medline First Search: *predicting overweight in adulthood*, *predicting overweight*, and *predicting overweight from childhood to adult*. The key references from some especially relevant studies were also searched further. The literature reviewed here is limited to studies that predict adult overweight from childhood weight status after age six. This was done for three reasons. First, many studies aim to predict adult weight status from infancy and early childhood (age one to five) weight status. Results have been poor, especially for males. Guo et al. found the correlation coefficients to be ≤ 0.3 between BMI values up to age four years and BMI values at age 35 years in boys and girls (Guo et al., 1994). At age six, the correlation was above 0.4 for girls, but did not rise to 0.4 until age 10 years for boys (Guo et al., 1994). After these ages, the correlations increased with age in both sexes (Guo et al., 1994). In a later study by Guo et al., the probabilities of adult obesity were found to be 0.24 and 0.15 for females and males respectively, above the 95th percentile on the BMI-for-age growth charts at age three, and 0.43 and 0.23 for females and males above the same percentile at age six (Guo et al., 2002).

Second, the reason to limit this review to studies after age six years is that the correlations of childhood weight status with adult overweight and the odds ratios

for obesity in adulthood increase with increasing childhood age, especially after age six (Guo et al., 1994; Guo et al., 2002; Stark et al., 1981). Correlations between relative weights at age six and age 26 years were found to be 0.38 and 0.32 for females and males, respectively by Stark et al. (Stark et al., 1981). The correlations between relative weights at age 14 and 26 years rose to 0.64 and 0.52 for females and males and were even higher between relative weights at age 20 and 26 years (Stark et al., 1981).

Whitaker et al. found that the odds of becoming an obese adult was ten times higher for obese six to nine year olds than obese one to two year olds and two times higher for obese three to five year olds (Whitaker et al., 1997). The odds of becoming an obese adult increased rapidly after age six and remained high up to age 17 years (Whitaker et al., 1997). Among overweight children at any age interval, neither the age of onset nor the duration of overweight status in childhood increased the risk of adult obesity after adjusting for parental obesity or severity of childhood overweight (Whitaker et al., 1997).

Finally, beginning at age six, marked increases in the prevalence of childhood obesity occurs in the United States (Kuczmarski et al., 2000). Many studies predict adult overweight and obesity from childhood weight status beginning at ages six or seven years as does the present study (Powers et al., 1997; Magarey et al., 2003; Stark et al., 1981; Whitaker et al., 1997; Thorkild et al., 1988; Rolland-Cachera et al., 1989). These studies will be discussed here. Studies conducted prior to the year 2000 that investigated BMI and BMI-for-age values did not use the newly published CDC BMI-for-age charts.

The following studies reviewed are grouped and presented according to the type of analysis used to examine the relationship between childhood and adult obesity. First, studies using percentages and correlations are presented; then odds ratios and relative risks; finally regression and principal component analysis.

Percentages of sustained weight status from childhood to adulthood and correlations of childhood and adult weight status

The research has established that although not all overweight children become overweight adults, overweight children are more likely to become overweight adults than normal weight children (Rimm and Rimm, 1976; Guo et al., 1994; Abraham & Nordsieck, 1960; Abraham et al., 1970; Braddon et al., 1986; Miller et al., 1972; Must et al., 1992; Stark et al., 1981; Whitaker et al., 1997; Srinivasan et al., 1996; Magarey et al., 2003; Powers et al., 1997). Follow-up studies and retrospective studies have given the percentages of overweight or obese children that have become overweight or obese adolescents or adults. Zack et al. followed children's weight status into adolescence in two US Health Examination Surveys and found that 68-77% of the obese children became obese adolescents and 39-52% of lean children remained lean in adolescence (Zack et al., 1979). Though most overweight children will remain overweight in adolescence, lean children are also at risk of being overweight in adolescence. This is consistent with the findings that overweight in increasing in the US.

Srinivasan et al. conducted a follow-up study of the Bogalusa Heart Study and found that 58% of the 13-17 year old adolescents that were overweight in the

study were also overweight as adults aged 25-31 years (Srinivasan et al., 1996). Forty-one percent of lean adolescents remained lean as adults. Similarly, 13-18 year old adolescents were followed-up in adulthood and 52% of the surviving subjects who were overweight adolescents remained overweight as adults (Must et al., 1992). Compared to the study done by Zack et al., children that are overweight are likely to become overweight adolescents, but not as likely to be overweight in young adulthood. Children that are lean, however, remain lean in the same percentages from adolescence to adulthood.

Three studies followed children from childhood to young or middle adulthood (Magarey et al., 2003; Powers et al., 1997; Stark et al., 1981). Of the seven year olds who were overweight in the study by Stark and colleagues, 41-43% were overweight 26 year olds and 24-28% of the average weight seven year olds were also overweight adults (Stark et al., 1981). Nearly half of the overweight seven year olds were at an average weight by age 26 years (Stark et al., 1981). Magarey et al. found that 27-63% of overweight children became overweight 20 year olds (Magarey et al., 2003). Of those above the 95st percentile for BMI at ages 7-16 years in the study conducted by Powers and colleagues, 26-41% were overweight at age 33 (Powers et al., 1997). These studies are similar to those discussed earlier and further confirm the notion that the prediction of adult overweight is greater from adolescence than from childhood. It also confirms that not all overweight children become overweight adults and some average weight children will be overweight adults.

Magarey et al., Stark et al., Powers et al., and Williams et al. calculated correlation coefficients of childhood weight status with adult weight status (Magarey et al., 2003; Stark et al., 1981; Williams et al., 1999). Stark and colleagues found that overweight status at age seven was moderately correlated (0.35) with overweight at age 26 (Stark et al., 1981). Overweight at age seven was similar for 33 year olds in the study by Powers and colleagues, 0.33 for males and 0.37 for females (Powers et al., 1997). The correlations found by Williams and colleagues between ages seven and 21 were higher (0.57), but still only moderately correlated (Williams et al., 1999). Magarey and colleagues studied an Australian population born in the mid seventies and found that BMI values from age six years correlated >0.6 with BMI at age 20 years (Magarey et al., 2003). They suggested that the risk of becoming an overweight adult if overweight as a child might be increasing (Magarey et al., 2003). This is the only study found in the literature search that included subjects born after 1960.

The correlation coefficients between adult and childhood weight status increased with increasing childhood ages in all studies (Magarey et al., 2003; Powers et al., 1997; Stark et al., 1981; Williams et al., 1999). At age 14 years, the correlation with BMI at age 26 was 0.59 (Stark et al., 1981) and the correlation between BMI at age 13 with BMI at age 21 was 0.67 (Williams et al., 1999). These findings are comparable to the findings by Guo and colleagues (Guo et al., 1994).

Guo et al. correlated BMI values from ages one to 18 years to BMI values at an older adult age than the previously discussed studies (Guo et al., 1994). The

correlation coefficients for BMI's of seven year olds with BMI's at 35 years were between 0.32 and 0.55 (Guo et al., 1994). The correlations were stronger for females and increased with age (Guo et al., 1994). At age 18 years, the correlations of BMI values with values at age 35 years were between 0.6 and 0.8 (Guo et al., 1994). In a study that correlated approximate childhood BMI-for-age values with adult BMI values, correlations were found to increase with age from 0.52 at six years to 0.78 at 16 years (Rolland-Cachera et al., 1989).

Correlations between childhood and adult skinfold thicknesses show the same pattern in males compared to weight and BMI correlations. Hawk and Brook found that four combined skinfolds in males age seven moderately correlate (0.65) with adult measures and increase to 0.78 at age 14 years (Hawk and Brook, 1979). For females the same correlations are 0.62 and 0.51 respectively and thus do not display a similar increase with age (Hawk and Brook, 1979). Rolland-Cachera et al. also found that childhood skinfold thicknesses correlated with adult skinfold thicknesses and correlations for four skinfold measurements were as high as the correlation for the best single skinfold measure (Rolland-Cachera et al., 1989).

Odds ratios and relative risks of sustaining weight status from childhood to adulthood

Though not all overweight children will be overweight adults and some average weight children will be overweight adults, the odds for being overweight as an adult is higher for overweight children than average weight children. The

odds of being overweight at age 35 years was two to nine times higher for children above the 75th percentile of BMI from ages 8-18 years than for children at the 50th percentile (Guo et al., 1994). Whitaker et al. found that the odds of being an overweight adult was 10 times higher for overweight six to nine year olds than those not overweight of the same ages (Whitaker et al., 1997). The odds of being overweight as an adult increased with increasing childhood ages (Whitaker et al., 1997). This study also found that the more overweight a child was, the more likely he/she would be overweight as an adult (Whitaker et al., 1997).

Guo et al. studied the patterns of change in adiposity measured by BMI from ages 2-25 years (Guo et al., 2000). They found that generally BMI increases from age five to a maximum BMI obtained near age 22 years (Guo et al., 2000). A larger BMI during puberty increases a male's risk of becoming an overweight adult, and the risk is greater for a female (Guo et al., 2000).

Magarey et al, using a recent birth cohort, found the relative risks for overweight and obesity at age 20 years to be 3.5 for eight and eleven year olds, but higher (4.28) for 15 year olds (Magarey et al., 2003). Thorkild et al. studied the relative risks of being an overweight adult from ages seven and 13 years (Thorkild et al., 1988). They found the relative risks to be >100 for seven year olds with a BMI greater than 19 and for 13 year olds with a BMI greater than 26 (Thorkild et al., 1988). This study also determined that the risk of being in a higher BMI percentile at age 13 years was higher for children who increase or decrease percentiles from ages 7-13 years than those who did not (Thorkild et al., 1988).

These investigators consistently concluded that not all overweight children become overweight adults, but the odds and relative risk of becoming an overweight adult was greater for those overweight in childhood and even greater when overweight during adolescence than those not overweight. (Guo et al., 1994; Whitaker et al., 1997; Guo et al., 2000; Magarey et al., 2003; Thorkild et al., 1988). Odds ratios and relative risks are good statistical tests used to examine the relationship between childhood and adult overweight because they can be used to compare the odds and risks of becoming an overweight adult between those who are overweight in childhood and those who are not. The odds of becoming overweight in adulthood was greater in those who were overweight in childhood than those who were not overweight in childhood, yet some who were at a healthy weight in childhood became overweight in adulthood in the above studies. Relative risks directly compares the risks of becoming overweight between overweight and normal weight children. In the studies examine above, the risk was consistently greater among overweight children than normal weight children.

Results from these studies are consistent with the correlation studies. Overweight in childhood correlated greater with overweight in adulthood than normal weight in childhood. Odds and relative risks of becoming overweight in adulthood increased with age, as found in the correlation studies.

Multiple Regression

Zack et al. used multiple stepwise regression to predict skinfold measures for body fatness in adulthood from skinfolds at various childhood ages among other variables (Zack et al., 1979). They found that in males, skinfold thicknesses were more predictive of adult body fatness compared to family income, pubertal status, height and weight (Zack et al., 1979).

Rolland-Cachera et al. found that adiposity in adolescence could be predicted from the age of adiposity rebound (Rolland-Cachera et al., 1984). They found that the earlier the rebound occurred, the higher the child's adiposity at age 16 years (Rolland-Cachera et al., 1984). Similarly, Siervogel et al. found that children whose adiposity rebound occurred earlier were fatter at age 18 years than those whose adiposity rebound occurred later (Siervogel et al., 1990).

Cronk et al. studied the trends in BMI from childhood to adulthood with regression analyses (Cronk et al., 1982). The results showed that an upward BMI percentile shift and childhood BMI values correlated best with adult BMI values and adult body fat (Cronk et al., 1982). They also concluded that 35-60% of the variance in adult percent body fat, total body fat, and BMI was explained by childhood BMI values (Cronk et al., 1982).

Results from these multiple regression analyses agree with studies those from simple regression and relative risk studies. The studies discussed in this section present a relationship between childhood and adulthood weight statuses, but cannot exclusively explain the variance in adult overweight and obesity.

Table 6. Studies since 1958 which have predicted adult obesity from childhood weight and fat status

Article	Subjects	Ages	Findings
Predicting adult OW from childhood weight status			
Guo SS et al., 1994	277 male 278 female	Childhood ages 1-18y Adult age 35 ± y	1. OR of OW at 35yr was 2x's higher if >75th percentile at 8-18yr vs. the 50th percentile 2. OR of OW at 35yr inc w/inc age, w/most rapid inc after age 13yr
Stark O et al., 1981	5362 national survey of health and development males & females	Childhood ages 6, 7, 11, 14yr Adult ages 20, 26yr	1. 41-43% of OW 7yr olds were OW adults at age 26 vs. 24-28% average wt 7 yr olds 2. Nearly half of the overweight 7 year olds were of normal weight at age 26yr 3. OW at age 6 correlated with OW at age 20 (0.45) Correlations inc as age inc
Rimm IJ and Rimm AA., 1976.	73,532 females	Retrospective study, "were you considered a fat child?" adult ages compared: 20-29yr, 30-39yr, 40-49yr	Severely obese women were 2.4x's more likely to have been fat children vs. least obese women
Abraham S, Nordsieck M., 1960	100 Females	? 6 mos to 20-30y 10-13y 31y	OB girls were 4.4x's more likely to be obese adults than normal weight girls
Abraham S et al., 1970		Childhood ages 10-13y Adult age 31y	63% of the obese boys became obese adults, vs. 10% of the nonobese boys, relative risk 6.3 (Childhood Obesity, Epstein)

Must A et al., 1992	508 adolescents, lean or overwt males & females	Childhood ages 13-18yr 1988; 270 followed up at adult age	52% of the surviving subjects who were OW in adolescence were still OW at follow-up
Whitaker RC et al., 1997	854 Washington State 1965-1971 males & females	Childhood ages 1-17yr Adult ages 21-29yr	1. The probability of becoming an OB adult was over 50% for OB children vs. 10% for nonOB children after age 6yr & inc w/age for OB children 2. OR of OB as adult was 10x's higher for OB 6-9yr olds and OR inc w/inc age
Srinivasan SR et al., 1996	783 Bogalusa Heart Study 65% white 35% black 1976&1977 males & females	Childhood ages 13-17yr Adult ages 25-31 yr	1. 58% of OW adolescents remained OW as adults 2. 41% of lean adolescents remained lean as adults
Zack PM et al., 1979	2177 US health examination surveys males & females	Childhood ages 6-11 yr Adolescent ages 12-17 yr	1. 68-77% of OB children become OB adolescents 2. 39-52% of lean children become lean adolescents 3. Initial skinfold thickness & age are most predictive of subsequent body fatness in males
BMI-for-age predictions			
Guo SS et al., 2002	166 males 181 females	Childhood ages 3-20 yr Adult ages 30-39 yr	The probability of having a BMI >25 at 35y inc w/childhood & adolescent BMI %tile & w/age

Thorkild IA et al., 1988;	908 Danish males	7-13yr childhood 18-26yr adulthood	1. At age 7yr RR of being OB adult was 193 for those w/BMI>19 2. At age 13yr RR of being OB adult was 1450 for those w/BMI>26 3. Risk for children at higher BMI at 13 inc if percentile levels for children have inc or dec since age 7 4. Majority of obese children do not develop severe obesity in adulthood, only a few severely obese adults have been severely obese throughout childhood
Rolland-Cachera MF et al., 1989	135 French sample of long growth study 1953 males & females	1-17y Mean adult 21.2±2.5y	1. BMI in children sig. correlations w/adult values at all ages & inc w/age from 0.52 at age 6yr to 0.78 at age 16yr 2. SF sig. correl. w/adult values 3. From age 6, correlations for 4SF are as high as for the best predicted single SF
Cronk CE et al., 1982;	Six American longitudinal growth studies (Cronk et al. same issue) Males & females	Childhood ages 3mos – 18yr Adult age 30y (adult BMI, weight change) Adult ages 20-50yr Fels for %BF, TBF, FCN, FCS	<u>Longitudinal principal components analysis to summarize trends, multiple regression of BMI at 30yr with all components</u> An upward percentile shift and w/s2 values at each age group (3-9yr, 10-17y, 4-18y) correlated best with adult w/s2 values...better than %BF, TBF, etc... (10-17yr 0.58 for males; 4-18yr 0.69 for females) High levels of w/s2 or inc levels of w/s2 over long periods during childhood & adolescence predispose to high levels of BF in adulthood

			60% or less of variance of adult body fatness explained by childhood w/s2 %BF, TBF, and w/s2 at 30yr are best predicted by trends in childhood w/s2 with 35-60% of their variances explained
Fatness predictions			
Hawk LJ and Brook CGD, 1979	318 males 303 females families measured b/w 1961 & 1962	Childhood ages 2-15y Adult ages 17-30yr	High correlations for SF thickness from childhood to adulthood was not shown
Siervogel RM et al.,1990	250 boys 246 girls Fels	Long 2-18yr	The earlier children reached adiposity rebound, the earlier they inc adiposity & the fatter they were at age 18.
Rolland-Cachera MF et al., 1984	151 children French long study males & females	Birth to 14-16 yr	Age at rebound was predictive of future adiposity, earlier the rebound, the higher the adiposity level observed at 16 yr

III METHODS

Design

The study design is a secondary data analysis of a longitudinal growth study called the Motor Performance Study (MPS) that tracked children in physical growth and performance from ages 2-18 years. The BMI-for-age percentiles and fat status for children at ages 6, 9, 12, 15, and 18 years in the MPS were compared to those from a national sample. Using the MPS sample, risk for obesity, overweight and underweight at age 18 will be predicted from the number of times growth channels were crossed from ages 6-18 years, and by the type of growth pattern demonstrated over time.

Table 1 lists the specific aims, hypotheses and variables for this study. **Table 2** depicts the design for Aim 1. The design for Aim 3 is shown in **Figure 1**.

Table 1. Specific aims, hypotheses, and variables.

Specific Aims and Research Questions	Hypotheses	Dependent Variables	Independent Variables
Specific Aim 1. To compare BMI and fat status and the frequency of underweight, at risk for overweight, overweight, and overfat youth in the selected study sample, to that of youth in the entire MPS and to that of a national sample of youth at ages 6, 9, 12, 15, and 18 years.	H1a: There will be no differences in BMI and fat status at each age among the selected study and the MPS. H1b: There will be no differences in the frequency of children underweight, at risk for overweight, overweight and overfat at each age among the selected study, MPS, and NHANES III.	H1a: BMI for age percentile (X±SD) SFSum for age (X±SD) H1b: % UW at each age (≤5th) % at risk for OW at each age (≥85th - <95th) %OW at each age (≥95th) %OF at each age (≥95th) of the study sample, the MPS, and NHANES	
Specific Aim 2. To define children's BMI-for-age growth patterns from ages 6-18 years as: 1) staying in the same percentile growth channel; 2) increasing percentile growth channels; 3) decreasing percentile growth channels; or 4) increasing and decreasing percentile growth channels, in order to determine if children are distributed equally among the four growth patterns.	H2: Over half of the children will remain in the same growth channel from ages 6-18 years and the remainder will be distributed among the other growth pattern groups.	Percent of subjects in each of the four growth patterns	
Specific Aim 3. To identify the extent to which obesity and overweight in childhood and change in growth channels from age 6-18 years predicts overweight and overfat status at age 18[1] years.	H3a: Children who cross growth channels (Groups 2, 3, 4) will have a higher risk for overweight, overfat, and obesity (overweight and overfat) at age 18[1] years than those who do not cross channels.	Overweight, overfat, obesity (overweight and overfat) status at age 18[1] years	One of four growth pattern groups
	H3b: Children who lie in the most growth channels during growth will have higher risk for overweight, overfat, and obesity (overweight and overfat) at age 18[1] years.	Overweight, overfat, obesity (overweight and overfat) status at age 18[1] years	Number of channels subjects fall in during growth between ages 6-18 years
	H3c: The ability of children's weight status at key ages to predict overweight at age 18[1] will increase	Overweight, overfat, obesity (overweight and overfat) status at age 18[1] years	Overweight status at ages 6, 9, 12, 15

	with increasing age.		
Specific Aim 4. To identify the extent to which underweight in childhood and growth pattern from age 6-18 years predicts underweight at age 18 years.	H3a: Children who decrease growth channels (Groups 3) will have a higher risk for underweight at age 18[1] years than those who do not decrease channels.	Underweight status at age 18[1] years	One of four growth pattern groups
	H3b: The ability of children's weight status (BMI-for-age percentile) at key ages to predict underweight at age 18[1] will increase with increasing age.		Underweight status at ages 6, 9, 12, 15

[1]Age 18 or age growth was believed to be completed.

Table 2. The study design for Aim 1 comparing mean anthropometric percentiles and frequencies exceeding risk cutpoints at key ages by gender across three samples.

	Sample		MPS[a]		NHANES III	
BMI-for-age Percentile						
At ages 6, 9, 12, 15, 18yr	X ± SD	F ≥85th-95th F ≥95th F ≤5th	X ± SD	F ≥85th-95th F ≥95th F ≤5th	X ± SD	F ≥85th-95th F ≥95th F ≤5th
TSF[b] + SSF[c] Percentile						
At ages 6, 9, 12, 15, 18yr	X ± SD	F[d] OF[e]	X ± SD	F OF	X ± SD	F OF

[a]MPS= MSU's Motor Performance Study
[b]TSF = triceps skinfold
[c]SSF = subscapular skinfold
[d]F = frequency
[e]OF = overfat

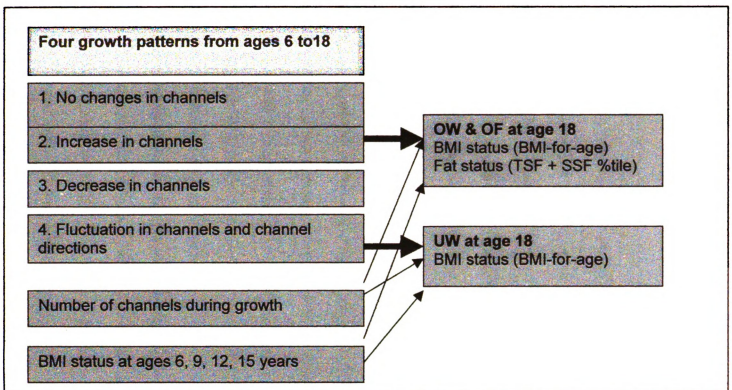


Figure 1. Study design for Aims 3 and 4 of BMI-for-age growth patterns, number of channels during growth, and BMI status at key ages to predict BMI and fat status at age 18 years.

OW=overweight; OF=overfat; TSF=triceps skinfolds; SSF=subscapular skinfolds

Subjects

Motor Performance Study

Thousands of children participated in the Motor Performance Study (MPS) physical activity program conducted by the Department of Kinesiology at Michigan State University, which ran from 1967 to 1999 (Haubenstricker, 1999) Most of these children were followed longitudinally and measured semi-annually with the aim of studying children's physical growth and motor skill development (Haubenstricker, 1999). Children were recruited after an article published in a local community newspaper, the Lansing State Journal, introduced the project to the greater Lansing area. Parents then called the Department of Kinesiology to volunteer their children to participate in the study (Haubenstricker, 1999). Participants were mostly white and from middle to upper class socioeconomic status households.

New enrollees were accepted at young ages each year (Haubenstricker, 1999). The subjects were volunteers, but their parents also paid a $10 program fee per quarter initially, which increased to $80 dollars by 1999 (Haubenstricker, 1999). These fees and the necessity of being on campus at certain times likely limited the participation of children from low income households (Haubenstricker, 1999). The participants benefited from instruction in several physical activities throughout their growing years, especially ages two to 13 years. Instruction was provided on Saturday mornings during the school year and during the first half of each summer (Haubenstricker, 1999). Examples of activities offered include ice-skating and team game skills (Haubenstricker, 1999).

Professors, graduate students, and other Kinesiology staff members collected data according to standardized instructions and protocols for measuring motor performance and physical growth. The procedures promoted consistency among data collectors (Haubenstricker, 1999). Thirteen physical growth and seven motor performance measures were taken semi-annually on each child (Haubenstricker, 1999). Physical growth measures included weight, standing height, sitting height, biacromial (shoulder) width, bicristal (hip) width, acrom-radiale (arm) length, radio-stylion (forearm) length, arm girth, thigh girth, calf girth, triceps skinfold, subscapular skinfold and umbilical skinfold (Haubenstricker, 1999). Some examples of the motor performance measures were flexed-arm hang, 30-yard dash and standing long jump.

Data Set

The data set for this study was extracted from the entire MPS dataset. Power analysis revealed that at least 275 subjects were needed for 80% power at a significance level of 0.05 to detect overweight status at age 18 from weight status at age nine years (Fleiss, 1981). The power calculation was conducted using the national prevalence of overweight of white children at risk for overweight at ages 9 (22%) and 18 (33%) (Kimm et al., 2002).

Subjects with the most complete data were extracted from the MPS dataset. Those with measurements of height and weight at each age from 6 to 15 years and a last measurement at either 16, 17 and/or 18 years were included in the study. The last age of measurement was determined by the completion of height

growth. Subjects no longer participated in the Motor Performance Study program, when it was determined that their height growth was complete. Height growth was considered to be completed when three consecutive semiannual measurements were within three millimeters of each other. It is possible that some females did attain more height at a later age after ending their participation (Taranger and Hagg, 1980).

The MPS data used in this study included gender, race, age, height, weight, BMI, triceps skinfold, and subscapular skinfold measurements for each child measured semiannually. The age range of 6-18 years was selected because this range captured the pre to post pubertal growth period and the years during which overweight increased. For example, when the 2000 BMI-for-age growth charts were constructed, data from NHANES III for children ages 6 and older were excluded because after age six there was an increase in prevalence of overweight compared to children from earlier NHANES (Kuczmarski et al., 2000).

There was a need to collapse the anthropometric data for meaningful comparisons between the study sample, the MPS and NHANES III to reduce the risk of committing a type II error. The ages of 6, 9, 12 and 15 years were selected as those from which to predict overfat and overweight at age 18, due to the body composition changes that generally occur at these key ages. At age six, the prevalence of overweight begins to increase (Kuczmarski et al., 2000; Kimm et al., 2001). At age nine most children are prepubescent, although some might enter puberty early, especially those overfat and/or overweight (Davison et al., 2003). Age 12 is often found to be the mean age of menarche in females

and developmental differences are evident between 12 and 13 year olds (Biro et al., 2001; Taylor et al., 2002). At age 15, most female adolescents are in a post-pubertal stage and most males have undergone their growth spurt (Biro et al., 2000).

Procedures

Human subject approval was obtained from the University Committee on Research Involving Human Subjects (UCRIHS) at Michigan State University. The dataset was requested from the Department of Kinesiology's Motor Performance Study investigators. The original data set was in SPSS format, and modified for this study to include only the identification code, race, gender, height, weight, triceps skinfold, subscapular skinfold, and age for each child at each age from 6-18 years.

The sum of the triceps and subscapular skinfolds was added and labeled as "SFsum." BMI-for-age percentiles according to Z-scores were calculated from measurements at each age. Each subject's growth was plotted on the BMI-for-age growth charts using Nutstat EpiInfo software (EpiInfo., 2002). NutStat was used to calculate Z-scores using the following equation: **Z-score = {[(X/M)^{L}] – 1}/(L x S)** in which L was the skewness of the chart curve, M was the mean, S was the standard deviation and X was the given BMI value. A new variable "BMI percentile = bmipctl" was then created in SPSS. Each growth channel was numbered consecutively with the first channel being $\leq 5^{th}$ percentile and the eighth channel being the $\geq 95^{th}$ percentile as shown in **Table 3**. The $\geq 85^{th}$ and

$\geq 90^{th}$ percentile channels were combined since both of these channels categorize children as "at risk for overweight." The assigned variable "channel number = channumb" was then be added to the dataset as an independent variable.

Table 3. Percentile channels and their corresponding Channel Numbers.

Channel Number	Growth Percentile Channel
1	$\leq 5^{th}$
2	$>5^{th}$ to $\leq 10^{th}$
3	$>10^{th}$ to $\leq 25^{th}$
4	$>25^{th}$ to $\leq 50^{th}$
5	$>50^{th}$ to $\leq 75^{th}$
6	$>75^{th}$ to $<85^{th}$
7	$\geq 85^{th}$ to $>95^{th}$
8	$\geq 95^{th}$

A variable "total change = totalchg" was developed to signify the number of semiannual changes in percentile channel. Channel crossings were calculated by subtracting the channel number at a given age from the channel number at the preceding age. For example, if a child was in the 70^{th} percentile at age six, and at the 40^{th} percentile at age seven, this was calculated as channel number 6 ($6 = 75^{th} > x > 50^{th}$) minus channel number 5 ($5 = 50^{th} > x > 25^{th}$) equaling one channel crossing. If the child stayed in the same channel between these two ages, the value given for the channel crossings was 0 because there were no channel crossings. In a similar fashion, the variable "growth channel = grthchan" was developed to signify the number of percentile channels subjects fell within during growth.

The age of peak height velocity (PHV) was identified for each individual to categorize subjects as before or after the average PHV age for sex to distinguish between earlier and later maturers. PHV marks the year in which the most height growth occurs during adolescence. On average, PHV occurs at age 12 years for females and at age 14 years for males. A variable "PHV category = phvcateg" was developed to categorize subjects as attaining peak height velocity before, at, or after the average age per sex. From PHV, it was not possible to identify the specific starting point of, stage or concluding point of sexual maturation, but it was a marker for age at a certain sexual maturation stage, which could assist in distinguishing between early, average, and late maturing children.

Analysis

Statistical Package for Social Science was used for the data analyses (version 10.0.5; SPSS Inc, Chicago, IL, 1999). Descriptive statistics described the sample size, gender, race, mean BMI-for-age percentile and mean sum of skinfolds percentile for each age from 6-18 years. Descriptive graphs were used for frequencies of growth pattern groups, total number of growth channels crossed and number of channels within during growth. Descriptive data for the mean BMI-for-age percentile and mean sum of skinfolds percentile were presented as a grand mean, and split by those whose last age of measurement was 16, 17, and 18 years to account for early and late maturers. The statistical analysis of each specific aim is discussed below (see Table 1).

Aim 1. At risk for overweight status at age 18 years included those above the 85th percentile and below the 95th percentile on the BMI-for-age growth charts. Overweight status at age 18 years included those above the 95th percentile on the BMI-for-age growth charts. Overfat status at age 18 included those above the 95th percentile on the TSF + SSF percentile charts. The BMI and fat status of the selected sample was compared to the MPS and a nationally representative sample, NHANES III, by age. The BMI and fat status of the MPS and the nationally representative sample was also compared by age at 6, 9, 12, 15, and 18 years.

H1a: Means and standard deviations of BMI-for-age percentiles at key ages (6, 9, 12, 15, and 18 years) were compared across the selected sample and the MPS using ANOVA.

H1b: The frequencies of children underweight, at risk for overweight, overweight, and overfat at each age were compared using Chi-Square test. Comparisons were across the three groups of those underweight, at risk for overweight, overweight, and overfat.

Aim 2. A growth pattern was determined for each individual in the sample and numbered one to four according to the growth pattern group into which they were categorized (see **Figure 1**). Growth pattern group one included those who did not change channels during growth from ages 6-18 years. Growth pattern group two included those who only increased channels at some point during growth from ages 6-18 years. Growth pattern group three included those who only decreased channels at some point during growth from ages 6-18 years.

Growth pattern group four included those who increased channels and decreased channels at some point during growth from ages 6-18 years.

H2: The frequency of individuals in each growth pattern was determined. Chi-Square analysis was used to determine if the observed frequencies of the four growth pattern groups differed significantly from expected frequencies.

Aim 3. Growth pattern frequencies, number of growth channels within and during growth from age 6 to18 years, and weight status at ages 6, 9, 12, 15, and the last age were determined.

H3a: The frequencies of each growth pattern group was then used to determine the odds ratios for each group of being overweight and overfat at 18 years of age, with group 1 (no changes in channels) used as the reference group.

H3b: The number of growth channels children were within during growth were determined. The odds ratios of being OW and OF at the last age were analyzed based on the number of growth channels within during growth. Odds ratios were calculated for each possible split of growth channels within controlling for PHV. The lower number of channels was used as the reference group in each case. **Table 4** depicts this analysis.

H3c: To predict obesity (overweight and overfat) at age 18 years from the BMI statuses (BMI-for-age percentile) at ages 6, 9, 12, and 15, odds ratios were run.

Aim 4. Growth pattern frequencies, number of growth channels within during growth from age 6 to18 years, and weight status at ages 6, 9, 12, 15, and the last age were determined.

H4a: The frequencies of each growth pattern group were used to determine the odds ratios for each group of being underweight at the last age, with group 1 (no changes in channels) used as the reference group.

H4b: To predict underweight at age 18 years from the BMI statuses (BMI-for-age percentile) at ages 6, 9, 12, and 15, odds ratios were run.

Table 4. Grouping for odds ratios analysis to determine the odds ratios of becoming overweight and overfat at the last age based on number of channels crossed during growth.

Number of Channels Crossed (Reference Group)	Number of Channels Crossed
0	≥1
0-1	≥2
0-2	≥3
0-3	≥4
0-4	≥5
0-5	≥6
0-6	≥7

Visual comparisons will be made between growth charts of overweight and average weight subjects in terms of their growth patterns to determine trends between the two sets of growth charts.

IV RESULTS

Descriptive Statistics

Three hundred and fifty-four participants were extracted from the original MPS dataset and included in the analysis. These subjects were all the subjects found to have complete growth data from ages 6 to 15 years and a measurement at 16, 17, and/or 18 years. When three consecutive semiannual height measurements were within three millimeters of each other, growth was determined to be complete and subjects no longer participated in the Motor Performance Study. As the last age of participation increased from 16 to 18 years, the percentage of females in the sample declined from 46% to 16% and the percentage of males increased from 54% to 84% at age 18 years (**Table 1**). This change in proportion of males and females was expected because females generally complete height growth earlier than males. It is possible, however, that some females grew slightly in height at a later age after ending their MPS participation (Taranger & Hagg, 1980).

Most all subjects in this analyses (n=345, 97.5%) were non-Hispanic white youth (**Table 1**). Eight (2.3%) were Asian and one (0.3%) was non-Hispanic black.

Table 1. Subjects characteristics – age, gender, race (percentages)

Age	6	9	12	15	16	17	18
	N=354	N=354	N=354	N=354	N=351	N=239	N=136
Gender							
Males	191	191	191	191	189	168	114
	(54)	(54)	(54)	(54)	(54)	(70)	(84)
Females	163	163	163	163	162	71	22
	(46)	(46)	(46)	(46)	(46)	(30)	(16)

Non-Hispanic White	345 (97.5)	345 (97.5)	345 (97.5)	345 (97.5)	342 (97.5)	233 (97.5)	133 (97.8)
Non-Hispanic Black	1 (0.3)	1 (0.3)	1 (0.3)	1 (0.3)	1 (0.3)	1 (0.4)	1 (0.7)
Asian	8 (2.3)	8 (2.3)	8 (2.3)	8 (2.3)	8 (2.3)	5 (2.1)	2 (1.5)

At each age, 6.3% to 11.2% of males and 0% to 9.8% of females exceeded the 85th percentile of the BMI-for-age growth charts (**Table 2**). The highest percentage of AROW/OW females occurred at six years old for females and progressively declined with increasing age. In contrast, the percentage of AROW/OW males increased with age with the highest percentage at age 16 years. Most subjects of both genders were in normal weight ranges and few measurements fell beneath the 5th percentile. The percentages closely resembled the expected normal distribution from which the BMI-for-age charts were composed, with just a slightly lower percentage of subjects in both risk categories of AROW/OW and UW.

Table 2. Number (and percentages) of children at various BMI-for-age percentiles by selected ages

		BMI-for-age Percentile		
Age		**<5th**	**≥5th to <85th**	**≥85th**
Males	**N**			
6	191	7 (3.7)	172 (6.3)	12 (6.3)
9	191	3 (1.6)	171 (89.5)	17 (8.9)
12	191	7 (3.7)	166 (86.9)	18 (9.4)
15	191	3 (1.6)	169 (88.5)	19 (9.9)
16	188	5 (2.7)	162 (86.2)	21 (11.2)
17	168	6 (3.6)	145 (86.3)	17 (10.1)
18	114	4 (3.5)	99 (86.8)	11 (9.6)
Females				

6	163	5 (3.1)	142 (87.1)	16 (9.8)
9	163	6 (3.7)	145 (89.0)	12 (7.4)
12	163	6 (3.7)	147 (90.2)	10 (6.1)
15	163	1 (0.6)	153 (93.8)	9 (5.6)
16	162	1 (0.6)	152 (93.8)	9 (5.6)
17	71	1 (1.4)	68 (95.8)	2 (2.8)
18	22	1 (4.5)	21 (95.5)	0 (0)

The average BMI-for-age percentiles ranged near the 50th percentile overall for both genders, with the percentiles slightly lower for females **(Tables 3 & 4)**. The sum of skinfold percentages were closer to the 25th percentile than the 50th in most cases, and as low as the 15th percentile for some females. Averages for both measurements appeared higher in those who exited the study at an earlier age than those who exited at a later age, especially in males. This was expected as early matures often have more body mass than those who mature later (Frisch, 1985). A greater difference between those that exited early versus late is found in the BMI-for-age percentile averages than in the sum of skinfold averages. The percentile differences are larger among the BMI-for-age values than among the sum of skinfold averages between these groups. This is likely due to a greater amount of muscle mass versus fat mass in the early maturers compared to the later maturers.

Table 3. Grand means and stratified means ± SD at the last ages of 16, 17, 18 years of BMI-for-age percentiles and skin fold sums for males

	Ages						
	6	**9**	**12**	**15**	**16**	**17**	**18**
N	191	191	191	191	188	168	114
Grand Means							
BMI Percentile	47.6 ± 26.3	48.5 ± 24.9	46.1 ± 25.8	52.5 ± 25.3	52.1 ± 25.3	49.6 ± 26.4	47.3 ± 26.1
Sum of Skinfolds (mm)	13.8[1] ± 3.5	16.4[1] ± 6.7	19.0[1] ± 8.8	17.5[1] ± 6.1	18.5[1] ± 7.1	19.0[1] ± 9.2	19.7[2] ± 7.9
Means for Last Age of 16 Years							
N	22	22	22	22	22		
BMI Percentile	52.3 ± 30.6	55.7 ± 30.0	57.0 ± 26.2	63.4 ± 20.9	59.6 ± 23.0	N/A	N/A
Sum of Skinfolds (mm)	14.8[1] ± 3.5	17.8[1] ± 7.2	19.8[1] ± 8.6	19.9[1] ± 5.3	20.0[1] ± 7.6	N/A	N/A
Means for Last Age of 17 Years							
N	55	55	55	55	54	55	
BMI Percentile	50.6 ± 25.0	54.0 ± 23.7	54.3 ± 24.9	60.2 ± 22.7	58.9 ± 24.6	55.3 ± 27.2	N/A
Sum of Skinfolds (mm)	14.0[1] ± 3.0	17.3[1] ± 7.2	20.7[1] ± 11.9	20.0[1] ± 11.4	20.6[1] ± 12.5	20.6[1] ± 11.7	N/A
Means for Last Age of 18Years							
N	114	114	114	114	113	113	114
BMI Percentile	45.3 ± 26.0	45.0 ± 23.9	40.1 ± 24.5	46.6 ± 23.7	47.5 ± 25.0	46.8 ± 25.7	47.3 ± 26.1
Sum of Skinfolds (mm)	13.4[1] ± 3.4	15.1[1] ± 5.5	17.7[1] ± 6.5	16.5[1] ± 5.8	17.3[1] ± 6.2	18.0[1] ± 7.1	19.6[2] ± 7.7

[1] 50th percentile for age (age 6 = 13.0 – 15.2mm; age 9 = 14.0 – 19.0mm; age 12 = 17.0 – 24.0mm; age 15 = 15.0 – 21.0mm; age 16 = 16.0 – 22.5mm; age 17 = 16.0 – 22.0mm; age 18 = 21.0 – 30.0mm)

[2] 25th percentile for age (age 6 = 10.5 – 13.0mm; age 9 = 11.0 – 14.0mm; age 12 = 12.5 – 17.0mm; age 15 = 12.0 – 15.0mm; age 16 = 13.0 – 16.0mm; age 17 = 13.0 – 16.0mm; age 18 = 15.0 – 21.0mm)

Table 4. Grand means and stratified means ± SD at the last ages of 16, 17, 18 years of BMI-for-age percentiles and skin fold sums for females

	Ages						
	6	**9**	**12**	**15**	**16**	**17**	**18**
N	163	163	163	163	162	71	22
Grand Means							
BMI Percentile	45.3 ± 25.1	42.9 ± 25.2	41.6 ± 26.3	45.8 ± 23.0	45.7 ± 22.7	42.5 ± 21.7	39.3 ± 21.2
Sum of Skinfolds (mm)	15.7[1] ± 3.5	17.9[2] ± 5.7	20.1[2] ± 7.8	23.6[1] ± 8.3	24.8[1] ± 7.6	25.5[1] ± 7.6	25.2[2] ± 6.6
Means for Last Age of 16 Years							
N	92	92	92	92	92		
BMI Percentile	47.6 ± 26.6	46.0 ± 26.0	45.6± 26.4	46.7± 24.2	46.8 ± 23.2	N/A	N/A
Sum of Skinfolds (mm)	16.1[1] ± 3.6	18.7[2] ± 6.3	20.6[2] ± 8.6	23.6[2] ± 8.6	24.9[2] ± 8.2	N/A	N/A
Means for Last Age of 17 Years							
N	49	49	49	49	49	49	
BMI Percentile	43.2 ± 23.6	40.9 ± 24.9	40.1 ± 25.5	47.1± 22.0	46.4 ± 22.9	45.5± 22.8	N/A
Sum of Skinfolds (mm)	15.1[2] ± 3.2	17.2[2] ± 5.0	19.9[2] ± 6.8	24.7[2] ± 8.4	25.1[2] ± 7.1	26.3[2] ± 8.4	N/A
Means for Last Age of 18Years							
N	22	22	22	22	22	22	22
BMI Percentile	40.6 ± 21.9	34.9 ± 20.8	28.4 ± 24.0	39.3 ± 20.1	39.9 ± 19.8	35.8 ± 17.9	39.4 ± 21.2
Sum of Skinfolds (mm)	15.1[2] ± 3.9	16.1[2] ± 4.3	18.3[2] ± 6.3	21.4[2] ± 5.8	23.5[3] ± 6.5	23.8[2] ± 5.2	25.2[2] ± 6.6

[1] 50th percentile for age (age 6 = 15.5 – 18.5mm; age 9 = 19.0 – 25.5mm; age 12 = 23.0 – 31.0mm; age 15 = 26.5 – 34.5mm; age 16 = 30.0 – 39.5mm; age 17 = 31.0 – 42.0mm; age 18 = 32.0 – 44.0mm)
[2] 25th percentile for age (age 6 = 12.5 – 15.5mm; age 9 = 14.5 – 19.0mm; age 12 = 18.0 – 23.0mm; age 15 = 20.5 – 26.5mm; age 16 = 24.0 – 30.0mm; age 17 = 23.0 – 31.0mm; age 18 = 24.0 – 32.0mm)
[3] 15th percentile for age (age 6 = 11.0 – 12.5mm; age 9 = 12.5 – 14.5mm; age 12 = 15.0 – 18.0mm; age 15 = 18.0 – 20.5mm; age 16 = 21.5 – 24.0mm; age 17 = 20.0 – 23.0mm; age 18 = 21.0 – 24.0mm)

Average BMI values and average sum of skinfolds are better correlated for females than for males (**Tables 5 & 6**). This is expected because females have a higher percentage of body fat than males. The best correlations are at ages 9 and 12 for both males and females, probably indicating the time of weight gain and fat storage in preparation for a height spurt.

Table 5. Correlations between average BMI's and average sum of skinfolds for males at key ages

Age	N	Correlation
6	191	0.500*
9	191	0.622*
12	191	0.632*
15	191	0.506*
16	188	0.499*
17	168	0.488*
18	114	0.568*

*p<0.001

Table 6. Correlations between average BMI's and average sum of skinfolds for females at key ages

Age	N	Correlation
6	163	0.689*
9	163	0.797*
12	163	0.765*
15	163	0.710*
16	162	0.680*
17	71	0.681*
18	22	0.759*

*p<0.001

Specific Aim 1. To compare BMI and fat status and the frequency of underweight, at risk for overweight, overweight, and overfat youth in the selected study sample, to that of youth in the entire MPS and to that of a national sample of youth at ages 6, 9, 12, 15, and 18 years.

The study sample included too few underweight and overfat youth for meaningful statistical comparisons of the prevalence of these variables to NHANES data. The prevalence of AROW/OW subjects in the study sample was compared to three NHANES datasets (**Table 7**), which spanned the years encompassed by the years of the study sample data collection (1967-1999). There were significantly fewer AROW/OW subjects in the study sample compared to each of the NHANES datasets for both genders. Fifteen percent would have been expected to be in this risk category based on the normally distributed nature of the growth charts. The study sample is therefore not a nationally representative sample of this risk category.

Table 7. Prevalence of $\geq 85^{th}$ BMI-for-age percentile in the study sample compared to NHANES I, II, and III.

Age	N	Research Dataset	NHANES I (1971-1974)	NHANES II (1976-1980)	NHANES III (1988-1994)
Males					
6	12	6.3	12.1	15.8	23.3
9	17	8.9	17.6	17.4	27.8
12	18	9.4	14.1	15.6	29.4
15	19	9.9	14.7	14.4	23.2
18	11	9.6	18.6	12.2	16.0
Females					
6	16	9.8	11.0	12.5	23.3
9	12	7.4	14.5	16.1	25.6
12	10	6.1	22.3	17.1	30.9
15	9	5.5	17.3	13.3	23.0
18	0	0	10.1	12.6	18.5

Specific Aim 2. To define children's BMI-for-age growth patterns from ages 6-18 years as: 1) staying in the same percentile growth channel; 2) increasing percentile growth channels; 3) decreasing percentile growth channels; or 4)

increasing and decreasing percentile growth channels, in order to determine if children are distributed equally among the four growth patterns.

The growth charts were plotted over time for each child and examples are in Appendix A. When these data were summarized, most subjects fluctuated among percentile channels during growth from ages 6 to 18 years (**Figure 1**). Very few subjects solely increased or decreased in channels over time and even less remained in the same percentile growth channel during growth. This later finding was unexpected. Most subjects crossed centile lines several times, up to a maximum of 17 crosses during growth (**Figure 2**). To adjust for those that grew right along a centile line, but jumping a little above and below the line several times within the 10 to 12 years of growth studied, the total number of BMI-for-age percentile channels in which the subjects were *within* was also analyzed (**Figure 3**). The majority of subjects were still found to lie within 3 to 4 growth channels while growing during these years. A few children were found to lie within six and seven BMI-for-age channels during growth.

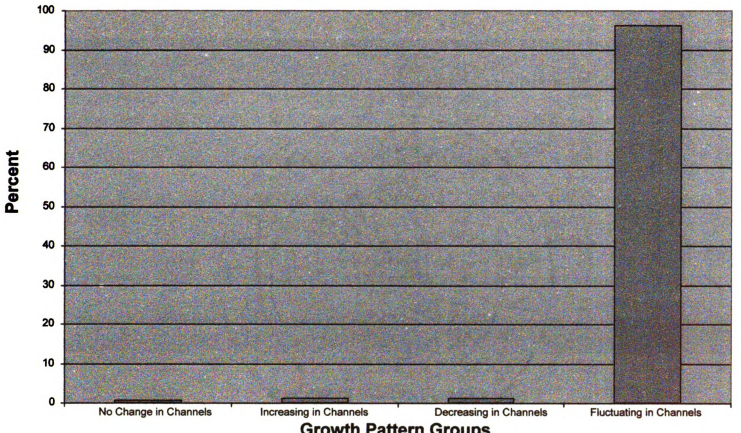


Figure 1. Frequencies of growth pattern groups within the study sample. Groups include 1) No change in BMI-for-age percentile channels, 2) Increasing only in channels, 3) Decreasing only in channels, and 4) Fluctuating in channels during growth from ages 6 to 16, 17 or 18 years, N=354.

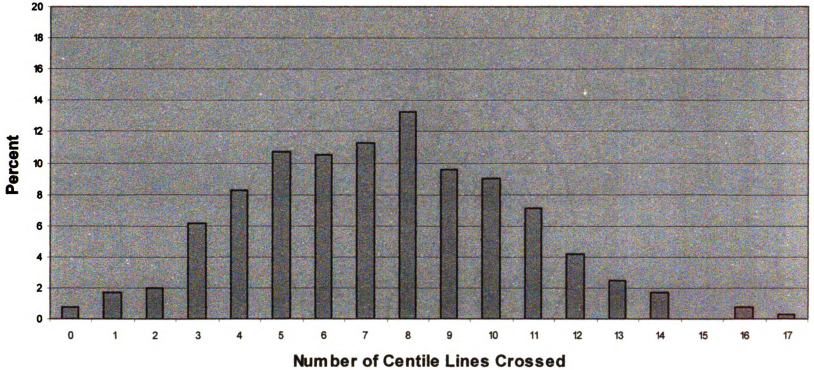


Figure 2. Frequencies in which BMI-for-age percentile channel lines (example: 10^{th}, 25^{th}, 50^{th} percentile lines) are **crossed** during growth from ages 6 to 16, 17 or 18 years for both males and females, N=354.

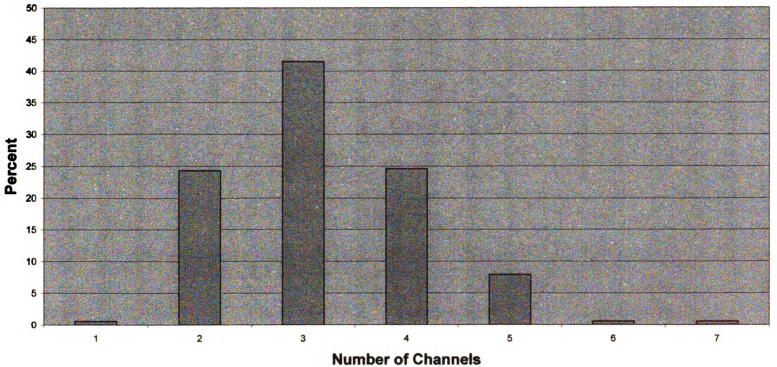


Figure 3. Number of BMI-for-age percentile channels subjects fall *within* during growth from ages 6 to 16, 17 or 18 years for both males and females, N=354.

Specific Aim 3. To identify the extent to which obesity and overweight in childhood and change in growth channels from age 6-18 years predicts overweight and overfat status at age 18 years.

H3a: This analysis could not be run because 97% of subjects were within group 4. That is, most children's growth patterns demonstrated fluctuation among channels and in channel directions.

H3b: The odds ratios were analyzed for the number of BMI-for-age growth channels children fell into during growth to avoid overestimation of growth channel changes in those that jumped a little above and below a main percentile *line* versus among several percentile *channels*. In these cases, subjects did not make big changes in percentiles during growth, but only changes in percentile channels.

Those youth who fell within four or more channels during growth were found to be 4.5 times more likely to be AROW and OW at their last age compared to those who fell within less than four channels during growth (**Table 8**). Gender and PHV were controlled, but when PHV was not controlled, the results were unchanged. The other cutpoints for channels within during growth that were tested were not significant. When observing the BMI-for-age growth charts of those AROW and OW, 64% (N = 21 and 33) were found to be within four channels during growth. Only five of the 33 AROW/OW subjects were within five

growth channels, but none were in more than five. Few were within two or three channels, but only one male grew continuously above the 95th percentile channel throughout growth. The majority of subjects of both genders were within less than 4 growth channels during growth (**Table 9**).

Of those within four or more growth channels, 25% of males and 9.8% of females were AROW or OW and 75% of males and 90.2% of females were UW or NW. Yet, only 6.5% of males and 2.7% of females within less than 4 growth channels were AROW or OW while 93.5% of males and 97.3% of females were UW or NW. Thus, a greater proportion of AROW and OW subjects were within greater than four channels than within less than four channels. For those UW and NW, a greater proportion was within less than four channels than within four or more channels. AROW and OW subjects often were not above the 85th percentile at an early age, but eventually increased to this channel requiring them to cross over major percentile channels during growth. This was especially true with males as they likely gained lean body mass and, therefore, their BMI's increased. This gain in body mass is attributed to lean body mass because few subjects were overfat in this study sample.

It is interesting that of the females within four or more channels, many more were UW and NW compared to the males. This suggests that normally growing females cross more channels than normally growing males. However, fewer AROW and OW females were included in this study than AROW and OW males. There were no AROW or OW females at age 18, and less than half the

percentage of AROW and OW females at ages 16 (5.6%) and 17 (2.8%) years (last ages) than males at these ages (11.2% and 10.1 % respectively).

Table 8. Odds of being AROW/OW at the last age by number of channels within during growth.

Variables	N	Odds Ratio (CI)
≥4 Channels During Growth	354	4.5* (2.1, 9.8)
<4 Channels During Growth (ref)		

*p<0.001
No other channel division yielded significant results. Controlled for gender and age of PHV.

Table 9. Number of BMI-for-age percentile channels within during growth by weight status.

		Weight Status	
Number of Channels in During Growth	N	UW & NW	AROW & OW
Males			
≥4 Channels	68	75.0%	25.0%
<4 Channels	123	93.5%	6.5%
Females			
≥4 Channels	51	90.2%	9.8%
<4 Channels	112	97.3%	2.7%

AROW = at risk for overweight (≥85th and <95th BMI-for-age percentile); OW = overweight (≥95th percentile); NW = normal weight (≥5th and <85th percentile); UW = underweight (<5th percentile).

H3c: Odds ratios were analyzed at each specified age (6, 9, 12, 15) to predict weight status at the last age from weight status at these ages. As expected from

the literature search, subjects who were AROW and OW at these ages were more likely to be AROW and OW at their last age compared to those who were NW at these ages. In fact, if OW at age 12, the odds were 100 of being OW at the last growth age. Similarly, those that were NW at these ages were more likely to be NW at their last age compared to those who were AROW and OW at these ages. As the initial age of weight status increased, generally, the odds of being this weight status at the last age increased. Each value was significant except for the odds of being OW at the last age predicted from OW status at age 15 years.

Table 10. Odds of being AROW and OW at the last age by weight status at key ages 6, 9, 12, 15.

Variables	N	Odds Ratio (CI)
Age 6	334	
AROW	21	8.4* (2.7, 26.3)
OW	6	60.0* (6.4, 566.5)
NW (ref)	307	
Age 9	339	
AROW	23	22.6* (8.1, 62.8)
OW	6	28.5* (4.6, 175.0)
NW (ref)	310	
Age 12	331	
AROW	21	63.4* (19.9, 202.3)
OW	6	>100*
NW (ref)	304	
Age 15	338	
AROW	23	42.9* (14.8, 124.1)
OW	3	>100
NW (ref)	312	

*p<0.001
N sizes vary due to varying weight statuses of individual subjects at different ages.
AROW = at risk for overweight (≥85th and <95th percentile); OW = overweight (≥95th percentile); NW = normal weight (≥5th and <85th percentile). Controlled for gender.

Table 11. Odds of Being NW at the Last Age by Weight Status at Key Ages 6, 9, 12, 15.

Variables	N	Odds Ratio (CI)

Age 6	334	
NW	307	13.5* (5.0, 36.3)
AROW/OW (ref)	27	
Age 9	339	
NW	310	23.7* (9.3, 60.4)
AROW/OW (ref)	29	
Age 12	331	
NW	304	73.1* (24.6, 216.7)
AROW/OW (ref)	27	
Age 15	338	
NW	312	53.6* (19.0, 151.8)
AROW/OW (ref)	26	

*p<0.001

N sizes vary due to varying weight statuses of individual subjects at different ages. AROW = at risk for overweight (≥85th and <95th percentile); OW = overweight (≥95th percentile); NW = normal weight (≥5th and <85th percentile). Controlled for gender.

Specific Aim 4. To identify the extent to which underweight in childhood and growth pattern from age 6-18 years predicts underweight at age 18 years.

These analyses were not run because only one to seven youth were found to be underweight at any age. The majority of youth who were underweight were generally below the 25th percentile throughout growth.

V DISCUSSION

This study is unique in demonstrating the great variability of growth of normally growing children across growth channels on the BMI-for-age growth charts released by CDC in 2000. Few children tracked consistently along a major percentile growth channel between the ages of 6 and 18 years. Those children whose percentiles did fall within four or more growth channels, however, were more likely to be AROW or OW at their last age (16-18 years) than those whose BMI-for-age percentiles fell within less than four channels. As other studies have shown, however, weight status at key ages 6, 9, 12, and 15 years was predictive of weight status at the last age (Guo et al., 1994; Guo et al., 2002; Stark et al., 1981; Whitaker et al., 1997; Magarey et al., 2003; Thorkild et al., 1988).

The frequency of AROW and OW subjects in this study sample was significantly lower compared to each of the NHANES datasets for both genders where at least 15% would be expected to be AROW or OW (Kuczmarski et al., 2000). This finding is not surprising because the current sample was comprised mostly of white middle-income children enrolled by their parents to regularly participate in fitness and physical performance sessions (Haubenstricker, 1999). It has been well documented that the prevalence of overweight and at risk for overweight is highest among those from ethnic minority families and those having limited incomes compared to white and higher income populations (Boumtje et al., 2005; Ogden et al., 2002; Adair et al., 2001). Data were collected over a period of 30 years and the time frame of each subject's participation was unknown, therefore, it was possible that many children having complete data had

entered the MPS program in the earlier years, that is the 1960's and 70's when the prevalence of childhood overweight was less than in the later years of the program (Ogden et al., 2002; Strauss et al., 2001; Flegal & Troiano, 2000; Troiano et al., 1995). The MPS study was continuous from 1967 to 1999 with subjects entering and completing the program in various decades.

Findings from the current study do not support the assertion that a "normally growing child" will remain within the same percentile growth channel over time and that a child who moves up or down a percentile growth channel should be considered "at risk" for underweight or overweight (CDC, 2000). According to Gifford, "It has been determined that after age two, children tend to grow along a channel (percentile growth channel) due to their genetics and environment" (Gifford, 1980, pg. 7). Instead, these findings demonstrated that very few children tracked longitudinally along the same percentile growth channel during growth, and, 97%, fluctuated between channels numerous times. In a similar study, Mei et al. tracked children up to five years old longitudinally on the BMI-for-age growth charts (Mei et al., 2004), but only 8 to 15% of subjects crossed two major BMI-for-age percentiles between the ages of 2 and 5 years (Mei et al., 2004). Nevertheless, Mei et al.'s findings support ours because the number of fluctuations in growth channels would be expected to increase within a longer age span. Thus, normally growing children may fall within several BMI-for-age growth channels during growth from ages 6 to 18 years of age.

This study did find that subjects whose BMI-for-age percentiles fell within the most growth channels during growth from ages 6 to 18 years had a higher risk of

being AROW or OW (4.5 times) at their last age compared to those whose BMI-for-age percentiles fell within less growth channels. This finding is somewhat unique to this study, because the odds ratios of weight status upon growth completion based on the number of BMI-for-age growth channels within during growth have not been studied previously. In this study, as in others, at each key age AROW and OW children were more likely to be AROW and OW at their last age compared to UW and NW children and vice versa. Odds ratios for AROW and OW at last ages increased with increasing childhood age, as other studies have shown (Guo et al., 1994; Guo et al., 2002; Stark et al., 1981). These expected results were similar to those found by other researchers (Guo et al., 1994; Guo et al., 2002; Stark et al., 1981; Whitaker et al., 1997; Magarey et al., 2003; Thorkild et al., 1988).

Guo et al. reported that the odds of being overweight at age 35 years was 2 to 9 times higher for children above the 75th percentile of BMI from ages 8-18 years than for children at the 50th percentile (Guo et al., 1994). The last age in thiat study was 35 years compared to a much younger final age of 16-18 years in the present study. Thus, due to the smaller time difference between the predicted age and the last age in this study compared to the Guo et al. study, it is expected that the odds ratios of the AROW and OW weight status in this study would be greater than the odds found by Guo et al.

Whitaker et al. found that the odds of being an overweight adult at ages 21 to 29 years was 10 times higher for overweight 6 to 9 year olds than those not overweight at the same ages (Whitaker et al., 1997). The odds of being

overweight as an adult increased with increasing childhood ages (Whitaker et al., 1997). Whitaker et al. also found that the more overweight a child was, the more likely he/she would be overweight as an adult (Whitaker et al., 1997). The odds found by Whitaker and colleagues were smaller than those found in the present study, but this again could be because of the older last age.

In a similar study using relative risks, results were consistent with those in the present study. Magarey et al., using a recent birth cohort, found the relative risks for overweight and obesity at age 20 years to be 3.5 for eight and 11 year olds, but higher (4.3) for 15 year olds (Magarey et al., 2003). Though Magarey et al. studied relative risks, their findings can be compared to our odds ratios. Our odds were greater than their relative risks for those at similar ages to become overweight at a similar last age. However, many of our subjects, especially females had completed their growth by age 16 years, which could explain, in part, the difference in odds ratio values compared to relative risks for a last age of 20 years.

Investigators have consistently concluded that not all overweight children become overweight adults, but the odds and relative risks of becoming an overweight adult was greater for those overweight in childhood than those not overweight. Many investigators have also found, as we did, that the odds and relative risks are even greater for those overweight during adolescence than for those overweight at younger ages (Guo et al., 1994; Whitaker et al., 1997; Guo et al., 2000; Magarey et al., 2003; Thorkild et al., 1988). In comparing the results from this study to other studies, our odds ratios are higher, but this might be

overstated in part due to having a younger last age as well as small homogenous sample.

Similar analyses predicting UW status were not run because the very small number of subjects who were underweight was inadequate to run meaningful analyses. This finding was not surprising because the prevalence of underweight in the US has decreased from 5.1% in the early 1970's to 3.3% by the 1990's (Wang et al., 2002).

Conclusion

This study found that BMI-for-age percentiles of healthy children as well as children at risk of overweight lie within many percentile channels during growth. Thus, variable patterns of growth along the BMI-for-age growth charts might not be an especially good indicator of risk for overweight in children and youth. The number of channels a child's BMI-for-age percentiles lie within during growth might give, however, some indication of risk, when the number of channel crossings exceeds four. Additional studies are needed to confirm this. Clearly this study supports the established findings that AROW and OW children are more likely to be of the same weight status as adolescents and adults than their NW counterparts and vice versa. In addition, the older the AROW or OW child is, the more likely he or she will be OW at a later age. The study sample was not nationally representative, but was fairly homogeneous in race and socioeconomic status.

Strengths of Study

A major strength of this study was use of a longitudinal dataset, more recent than the Fels study from the 1930's, to track children along the 2000 BMI-for-age growth charts. The tracking of children's BMI over time had only been done previously using the Fels data, and using different methods than those used here. This study is unique in using a longitudinal investigation to determine the patterns of children's growth along the chart's channels. Also, the selected sample was a good population to initially investigate growth patterns due to its homogeneity in ethnicity, social economic status and assumed health. Another strength of the study was that standardized procedures for anthropometrics were used when the Motor Performance Study data were collected.

Limitations

Some of the limitations of the study include the inability to report the reliability and the validity of the measurements, due to using a secondary dataset where these data were not reported. On the other hand, graduate students and faculty trained in anthropometric measurements using standardized procedures did the measuring. Due to the location and timing of the original data collection, the sample consisted of mostly white middle and upper class children. Thus, while in some ways a strength, the sample used in this study is not nationally representative, nor can differences by ethnicity and income be examined.

Sexual maturation stages were not assessed upon data collection. This is a limitation because sexual maturation should be controlled when using BMI to

predict weight and fat status. Sexual maturation stage (SMA) is a better indicator of pubertal growth in terms of BMI than is age (Adair et al., 2001). However, from this dataset, it was possible to determine peak height velocity (PHV), which was used to categorize early and late maturers for both genders, though no change in results was found with this categorization, in addition to completed growth indications. Those who discontinued participation in the MPS at an early age, like 16 years, were assumed to be earlier maturers than those whose participation ended at a 18 years. Some subject's height growth, and therefore participation, was complete at age 16 years. PHV occurs at sexual maturation stage (SMA) two for girls and at SMA four for boys.

This study captured growth channel changes that occur semiannually, but did not catch quarterly changes or more frequent changes. In addition, some changes might have occured due to physical conditioning and training during the school year or while participating in competitive sports. It is not possible to determine the cause of BMI-for-age channel crossings, only to study the patterns along the channels during growth and deduce possible implications. Finally, no historical growth data were available for the children's parents that would have been useful in interpreting the growth patterns as well.

VI IMPLICATIONS

In order to identify children at risk and in need for intervention using the new BMI-for-age growth charts, health professionals must first understand how healthy children track along the growth charts. Little research on growth during adolescence has been conducted to provide insight about how a healthy growing child would track along the BMI-for-age growth charts, much less how to clearly identify a child at risk using the charts. Nevertheless, the CDC advised that deviation from a growth channel indicates risk for OW in children. This study is the first to show that healthy children as well as children at risk of overweight cross many percentile channels during growth. In fact, only 3% of this study sample followed the same channel during growth from ages 6 to 18 years, and most fluctuated 3 – 4 times.

Another major finding was that those whose BMI-for-age percentiles fell within four or more growth channels during growth were 4.5 times more likely to be AROW or OW at growth completion compared to those whose BMI-for-age percentiles fell into fewer channels. Such findings are limited however, due to the small number of AROW and OW subjects in the study and the predominantly white, middle class subject participation. The sample was not nationally representative, but still provides insight about how healthy children track along the BMI-for-age growth charts.

BMI-for-age percentile points of normally growing children might fall within several major percentile channels during growth up to age 18 years. Clinicians, as well as parents should not be unnecessarily alarmed when a child increases

or decreases BMI-for-age percentile channels. Instead, other factors are just as, if not more, important in determining whether a child is at risk of underweight or overweight or not. For example, with children the height changes disproportionately to weight during growth spurts. Thus, unlike what is seen with adults, during growth children's height also influences the BMI. In addition, BMI does not distinguish between fat and lean tissue mass.

Other indicators of normal or at risk growth include the parents' body sizes and shapes, children's maturational stages, degree of fatness, and food and activity patterns. Children will often have similar body sizes and shapes to one or both parents with growth and body size being genetically driven to some degree. A high BMI-for-age percentile channel for a child could also reflect the food and activity patterns of the entire family. Thus, examination of the parents' weight histories and family food and activity patterns is important to interpreting children's BMI-for-age. Parents' behaviors and attitudes about food may also influence a child's eating, and therefore, growth patterns.

These research findings should convince health professionals that the BMI-for-age growth charts should never be used in isolation to assess a child's health risk. BMI-for-age growth patterns are important to understand, but they provide little information on nutritional status and health when used alone. They can be useful, however, when used in combination with other sources of information to assess a child's health status.

VII FURTHER RESEARCH

The present sample was limited by having few AROW and OW children and adolescents. A sample containing more such subjects would likely produce slightly different results in terms of growth pattern group distribution and risk for OW. The sample was also limited in ethnic and economic diversity. The majority were white, middle class children. More research needs to be conducted with a more sociodemographically diverse sample. It would also be beneficial to include at least two days of dietary recalls to identify eating patterns, inquire about parent's pubertal growth patterns and current size, and the family's food and activity patterns.

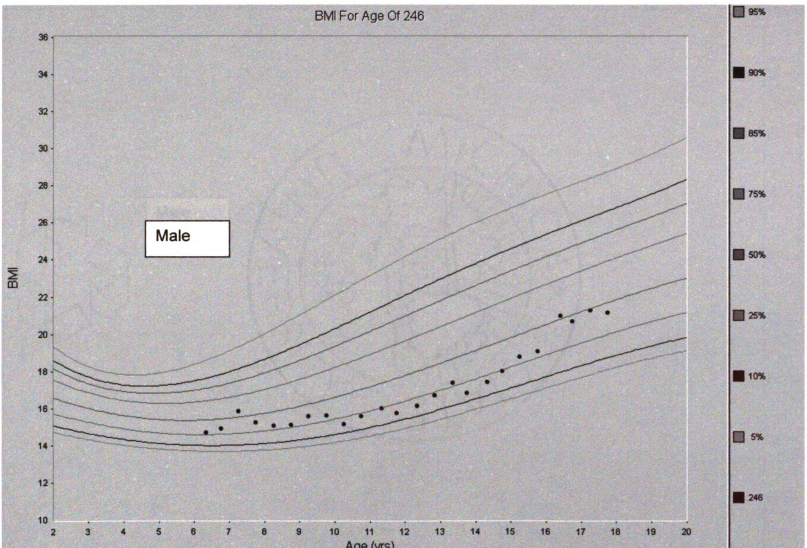
Such requirements for a study would be expensive, but each factor alone would contribute to the knowledge needed to fully understand how normally growing children track along the BMI-for-age growth charts. Children at risk of health disparities might be identified more easily if these patterns were better understood. Understanding these patterns may also reassure parents and cause less harm to children who are misclassified as "at risk" for overweight according to current recommendations.

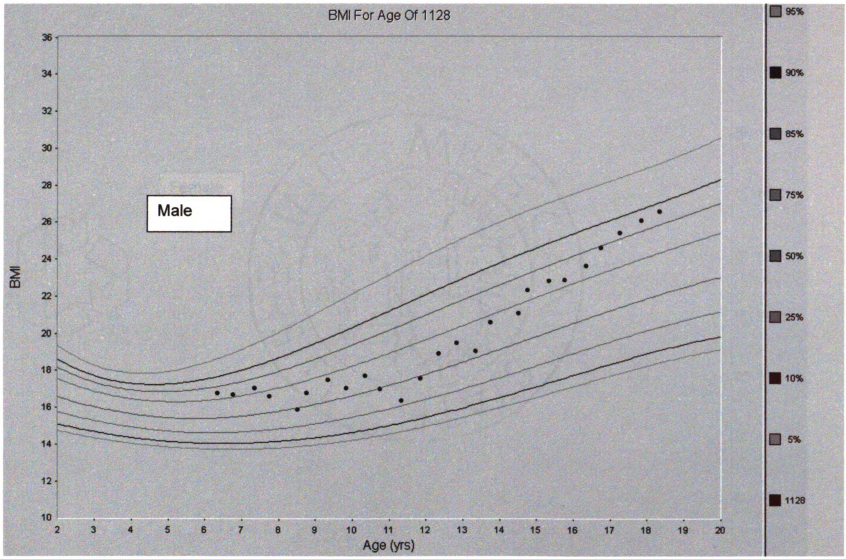
The ideal dataset for investigating growth patterns along the BMI-for-age growth charts would include a nationally representative, ethnically diverse sample with linear growth and sexual maturation measurements tracked longitudinally from birth to maximum height growth. The US Department of Health and Human Services and the Environmental Protection Agency are currently developing the National Children's Study (First Gov, 2005). This study

is expected to be the largest long-term study of human health ever conducted in the United States (First Gov, 2005). Researchers plan to follow 100,000 children from before birth to age 21 (First Gov, 2005). Variables will include growth data, pubertal data, children's dietary intake, and parental biological markers, as well as air, water, and house dust, neighborhood safety, and how often children see a doctor (First Gov, 2005). This study could contain the majority, if not all of the variables necessary to comprehensively evaluate growth interpretation along the BMI-for-age growth charts.

APPENDIX

BMI For Age Of 4
Male
BMI
Age (yrs)
95%
90%
85%
75%
50%
25%
10%
5%
4


BMI For Age Of 246
Male
BMI
Age (yrs)
95%
90%
85%
75%
50%
25%
10%
5%
246


BMI For Age Of 1128
Male
BMI
Age (yrs)
95%
90%
85%
75%
50%
25%
10%
5%
1128

BMI For Age Of 835
Female
BMI
Age (yrs)
95%
90%
85%
75%
50%
25%
10%
5%
835

BMI For Age Of 849
Female
BMI
Age (yrs)
95%
90%
85%
75%
50%
25%
10%
5%
849

BMI For Age Of 1222
Male
BMI
Age (yrs)
95%
90%
85%
75%
50%
25%
10%
5%
1222

REFERENCES

Abraham S, Collins G, Nordsieck M. Relationship of childhood weight status to morbidity in adults. *HSMHS Health Report*. 1971; 86:273-284.

Abraham S, Nordsieck M. Relationship of excess weight in children and adults. *Public Health Rep*. 1960; 75:263-273

Adair LS, Gordon-Larsen P. Maturational timing and overweight prevalence in US adolescent girls. Am J Pub Health. 2001; 91(4):642-644.

Attie I and Brooks-Gunn J. Development of eating problems in adolescent girls: a longitudinal study. *Dev Psychol*. 1989; 25:70-79.

Bachar E, Latzer Y, Canetti L, Gur E, Berry EM, Bonne O. Rejection of life in anorexic and bulimic patients. Int J Eat Disord. 2002; 31:43-48.

Baker SS, Borowitz D, Baker RD. Pancreatic exocrine function in patients with cystic fibrosis. Curr Gastroenterol Rep. 2005 Jun;7(3):227-33.

Baldridge AD, Perez-Atayde AR, Graeme-Cook F, Higgins L, Lavine JE. Idiopathic steatohepatitis in childhood: A multicenter retrospective study. *J* Pediatr. 1995; 127:700-704.

Balen AH, Conway GS, Kaltsas G, Techatraisak K, Manning PJ, West C, Jacobs HS. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. *Hum Reprod*. 1995; 10:2107-2111.

Barlow SE, Dietz WH. Obesity evaluation and treatment: expert committee recommendations. *J Pediatr*.1998; 102:e29.

Barnes HV. Physical growth and development during puberty. Med Clin North Am. 1975; 59:1305-17.

Bennion LJ, Knowler WC, Mott DM, Spagnola AM, Bennet PH. Development of lithogenic bile during puberty in Pima Indians. *N Engl J Med*. 1979; 300:873-876.

Bergstrom E, Hernell O, Persson LA, Vessby B. Insulin resistance syndrome in adolescents. *Metabolism*. 1996; 45:908-914.

Berkowitz R, Stunkard AJ, Stallings VA. Binge-eating disorder in obese adolescent girls. *Ann NY Acad Sci*. 1993; 699:200-206.

Biro FM, McMahon RP, Striegel-Moore R, ,Crawford PB, Obarzanek E, Morrison JA, Barton BA, Falkner F. Impact of timing of pubertal maturation on growth in

black and white female adolescents: The National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr.* 2001; 138:636-643.

Braddon FEM, Rodgers B, Wadsworth MEJ, ,Davies JMC. Onset of obesity in a 36 year birth cohort study. *Br Med J.* 1986; 193:299-303.

Brooks-Gunn J. Antecedents and consequences of variations in girls' maturation timing. *J Adolesc Health Care.* 1988; 9:365-383.

Burton BT, Foster WR, Hirsch J, VanItallie TB. Health implications of obesity: An NIH consensus development conference. *Int J Obes.* 1985; 9:155-169.

Caprio S, Hyman LD, McCarthy S, et al. Fat distribution and cardiovascular risk factors in obese adolescent girls: importance of the intra-abdominal fat depot. *Am J Clin Nutr.* 1996; 64:12-17.

Centers for Disease Control and Prevention. Interpreting growth charts. 2000. Available at: http://www.cdc.gov/nccdphp/dnpa/growthcharts/00binaries/growthchart.pdf Accessed February 28, 2003.

Centers for Disease Control and Prevention. What is EpiInfo? 2002. Available at: http://www.cdc.gov/epiinfo/ Accessed April 24, 2003.

Cohen SA. *The Underweight Infant, Child, and Adolescent.* East Norwalk, CT: Appleton-Century-Crofts; 1986.

Cole TJ. Growth charts for both cross-sectional and longitudinal data. *Stats Med.* 1994; 13:2477-2492.

Corbett JJ, Savino PJ, Thompson S, Dansu T, Schatz NJ, Orr LS, Hopson D. Visual loss in pseudotumor cerebri. *Arch Neurol.* 1982; 39:461-474.

Cronk CE, Roche AF, Kent R, et al. Longitudinal trends of weight/stature2 in childhood in relationship to adulthood body fat measures. *Hum Biol.* 1982; 54:751-764.

Daniels SR, Khoury PR, Morrison JA. The utility of body mass index as a measure of body fatness in children and adolescents: differences by race and gender. *Pediatrics.* 1997; 99:804-807.

Davison KK, Susman EJ, Birch LL. Percent body fat at age 5 predicts earlier pubertal development among girls at age 9. *Pediatrics.* 2003; 111(4):815-821.

DeJong W. Obesity as a chartacteristical stigma: The issue of responsibility and judgments of task performance. *Psychol Rep.* 1993; 73:963-970.

Dietz WH, Gross WL, Kirkpatrick JA. Blount disease (tibia vara): another skeletal disorder associated with childhood obesity. *J Pediatr.* 1982; 101:735-737.

Dietz WH. Childhood weight affects adult morbidity and mortality. *J Nutr.* 1998; 128:411S-414S.

Dietz WH. Health consequences of obesity in youth: Childhood predictors of adult disease. *Pediatrics.* 1998; 101:518-525.

Duck SC. Identification and assessment of the slowly growing child. *Am Fam Physician.* 1996; 53(7):2305-2312.

Dunaif A, Mandeli J, Fluhr H, Dobrjansky A. The impact of obesity and chronic hyperinsulinemia on gonadatropin release and gonadal steroid secretion in the polycystic ovary syndrome. *J Clin Endocrinol Metab.* 1988; 66:131-139.

Epstein LH, Valoski A, Wing RR, and McCurley J. Ten years follow-up of behavioral family-based treatment for obese children. *JAMA.* 1990; 2519-2523.

Epstein LH, Valoski AM, Kalarchian MA and McCurley J. Do children lose and maintain weight easier than adults: a comparison of child and parent weight changes from six months to ten years. *Obes Res.*1995; 3:411-417.

Epstein LH, Myers MD, Raynor HA, Saelens BE. Treatment of Pediatric Obesity. *Pediatrics.* 1998;101(3):554 - 570.

Evelyth PB, Tanner JM. Worldwide variation in human growth. 2nd ed. NY:Cambridge University Press. 1990.

Feldman W, Feldman E, Goodman JT. Culture *versus* biology: Children's attitudes toward fatness and thinness. *Pediatrics.* 1988; 81:190-194.

Figueroa-Colon R, Franklin FA, Lee JY, Aldridge R, Alexander L. Prevalence of obesity with increased blood pressure in elementary school-aged children. *South Med J.* 1997; 90:806-813.

First Gov. The National Children's Study. Available at: http://www.nationalchildrensstudy.gov/ Accessed November 1, 2005.

Flegal KM and Troiano RP. Changes in the distribution of body mass index of adults and children in the US population. *Int J Obes.* 2000; 24:807-818.

Flegal KM, Wei R, Ogden C. Weight-for-stature compared with body mass index-for-age growth charts for the United States from the Center of Disease Control and Prevention. *Am J Clin Nutr.* 2002; 75:761-766.

Fleiss JL. *Statistical Methods for Rates and Proportions*. 2nd Ed. New York, NY: John Wiley & Sons; 1981.

Foster TA, Voors AW, Webber LS, Freerichs RR, Berenson GS. Anthropometric and maturation measurements of children, ages 5 to 14 years, in a biracial community – The Bogalusa Heart Study. *Am J Clin Nutr*. 1977; 30:582-591.

Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: The Bogalusa Heart Study. *Pediatrics*. 1999; 103(6):1175-1182.

Frisch RE. Fatness, menarche, and female fertility. *Perspectives in Biology and Medicine*. 1985; 28(4):611-633.

Frisch RE. Fatness, puberty, and fertility: The effects of nutrition and physical training on menarche and ovulation. In: Brooks-Gunn J, Peterson AC, eds. *Girls at puberty: biological and psychosocial perspectives*. New York, NY: Plenum Press; 1983:29-49.

Frisch RE and McArthur JW. Menstrual cycles: fatness as a determinant of minimum weight fro height necessary for their maintenance or onset. *Science*. 1974; 185:949-951.

Garn SM, LaVelle M, Rosenberg KR, Hawthorne VM. Maturational timing as a factor in female fatness and obesity. *Am J Clin Nutr*. 1986; 43:879-883.

Garn SM, Leonard WR, Hawthorne VM. Three limitations of the body mass index. Am J Clin Nutr. 1986; 44:996-997.

Goldblatt PB, Moore ME, Stunkard AJ. Social factors in obesity. *JAMA*. 1965; 192:1039-1044.

Goldzeiher JW and Green JA. The polycystic ovary syndrome. I. Clinical and histological features. *J Clin Endocrinol Metab*. 1962; 22:325-331.

Gong EJ and Heard FP. Diet, nutrition and adolescence. In: Shils ME, Olson JA, Shike M, eds. *Modern nutrition in health and disease*, 8th ed. Philadelphia, PA: Lead & Febiger; 1994:759-769.

Gortmaker SL, Must A, Perrin JM, et al. Social and economic consequences o foverweight in adolescence and young adulthood. *N Engl J Med*. 1993; 329:1008-1012.

Gotlin R. Assessing normal growth. *Primary Care*. 1984; 11:549-566.

Guo SS, Chumlea WC, Roche AF, Siervogel RM. Age-and maturity-related changes in body composition during adolescence into adulthood: The Fels Longitudinal Study. *Int J Obes*. 1997; 21:1167-1175.

Guo SS, Huang C, Maynard LM, et al. BMI during childhood, adolescence, ,and young adulthood in relation to adult overweight and adiposity: the Fels Longitudinal Study. *Int J Obes Relat Metab Disord*. 2000; 24:1628-1635.

Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35y. *Am J Clin Nutr*. 1994; 59:810-819.

Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *Am J Clin Nutr*. 2002; 76:653-658.

Gunnell DJ, Frankel SJ, Nanchahai K, Peters TJ, Smith GD. Childhood obesity and adult cardiovascular mortality: a 57 year follow-up study based on the Boyd Orr cohort. *Am J Clin Nutr*. 1998; 67:1111-1118.

Gutin B, Owens S, Teiber F, Islam S, Karp W, Slavens G. Weight-independent cardiovascular fitness and coronary risk factors. *Arch Pediatr Adolec Med*. 1997; 151:562-565.

Hamill PV, Drizd TA, Johnson CL, Reed RB, Roche AF. NCHS growth curves for children birth-18 years, United States. Vital Health Stat. 1977; 11:165.

Haubenstricker JL. History of the Motor Performance Study and Related Programs. Haubenstricker JL, Feltz DL. 100 Years of Kinesiology; History, Research, and Reflections. East Lansing, MI. Michigan State University Printing Services. 1999. pg 103-115.

Hawk LJ, Briook CGD. Influence of body fatness in childhood on fatness in adult life. *Br Med J*. 1979; 1:151-152.

Heald FP and RJ Hollander. The relationship between obesity in adolescence and early growth. *J Pediatr*. 1965; 67(1): 35-38.

Hill AJ, Oliver S, Rogers PJ. A weight on children's mids: body shape dissatisfaction at 9 years old. *Int J Obes*. 1994; 18:383-389.

Himes JH, Bouchard C. Validity of anthropometry in classifying youths as obese. *Int J Obes*. 1989; 13:183-193.

Hoffmans MDAF, Kromhout D, de Lezenne Coulander C. The impact of body mass index of 78612 Dutch men on 32-year mortality. *J Clin Epidemiol.* 1988; 41:749-756.

Jelliffe DB, Jelliffe EFP. Community Nutritional Assessment. NY: Oxford University Press.1989.

Jiang X, Srinivasan SR, Webber LS, Wattigney WA, Berenson GS. Association of fasting insulin level with serum lipid and lipoprotein levels in children, adolescents and young adults: The Bogalusa Heart Study. *Arch Intern Med.* 1995; 155:190-196.

Johnston FE. Health implications of childhood obesity. *Ann of Int Med.* 1985; 103:1068-1072.

Kaplan KM and Wadden TA. Childhood obesity and self-esteem. *J Pediatr.* 1986; 109:367-370.

Karlberg J. Secular trends in pubertal development. *Horm Res.* 2002; 57 Suppl 2: 19-30.

Karlberg J, Cheung YB, Luo ZC. An update on the update of growth charts. *Acta Paediatr.* 1999; 88:797-802.

Kelsey JL, Acheson RM, Keggi KJ. The body builds of patients with slipped capital epiphyses. *Amer J Dis Child.* 1972; 124:276-281.

Kimm SY, Barton BA, Obarzanek E, McMahon RP, Kronsberg SS, Waclawiw MA, Morrison JA, Schreiber GB, Sabry ZI, Daniels SR. Obesity Development During Adolescence in a Biracial Cohort: The NHLBI Growth and Health Study. *Pediatrics.* 2002; 110(5):54-58.

Kimm SY, Barton BA, Obarzanek E, McMahon RP, Sabry ZI, Waclawiw MA, Schreiber GB, Morrison JA, Similo S, Daniels SR. Racial divergence in adiposity during adolescence: The NHLBI Growth and Health Study. *Pediatrics.* 2001; 107(3):E34.

Kinugasa A, Tsunamoto K, Furukawa N, Sawada T, Kusunoki T, Shimada N. Fatty liver and its fibrous changes found in simple obesity of children. *J Ped Gastroenterol Nutr.* 1984; 3:408-414.

Krowchuk DP, Kreiter SR, Woods CR, Sinal SH, DuRant RH. Problem dieting behaviors among young adolescents. *Arch Pediatr Adolesc Med.* 1998; 152:884-888.

Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, Flegal KM, Guo SS, Wei R, Mei Z, Curtin LR, Roche AF, Johnson CL. CDC growth charts: United States. Adv Data 2000; 314:1-28.

Kurtzman FD, Yager J, Landsverk J, Wiesmeier E, Bodkurka DC. Eating disorders among selected female student populations at UCLA. *J Am Dietetic Assoc.* 1989; 89:45-53.

Latner JD and Stunkard AJ. Getting worse: The stigmatization of obese children. *Obes Res.* 2003; 11 (3):452-456.

Lauer RM, Lee J, Clarke WR. Factors affecting the relationship between childhood and adult cholesterol levels: the Mustatine Study. *Pediatrics.* 1988; 82:309-318.

Lazar L, Kauli R, Bruchis C, Nordenberg J, Glaatzer A, Perzelean A. Early polysystic ovary-like syndrome in girls with central precocious puberty and exaggerated adrenal response. *Eur J Endocrinol.* 1995; 133:403-406.

Lipman TH, Hench K, Logan JD, DiFazio DA, Hale PM, Singer-Granick C. Assessment of growth by primary health care providers. *J Pediatr Health Care.* 2000; 14:166-171.

Loder RT, Aronson DD, Greenfield ML. The epidemiology of bilateral slipped capital femoral epiphysis: A study of children in Michigan. *J Bone Joint Surg.*1993; 75:1141-1147.

Lohman TG. *Advances in body composition assessment.* Champaign, IL: Human Kinetics Publishers; 1992.

Magarey AM, Daniels LA, Boulton TJ, Cockington RA. Predicting obesity in early adulthood from childhood and parental obesity. *Int J Obes.* 2003; 27:505-513.

Mallory GB, Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. *J Pediatr.* 1989; 115:892-897.

Maloney MJ, McGuire J, Damniels SR, Specker B. Dieting behavior and eating attitudes in children. *Pediatrics.* 1989; 84:482-489.

Marcus CL, Curtis S, Koerner CB, Joeffe A, Serwint JR, Loughlin GM. Evaluation of pulmonary function and polysomnography in obese children and adolescents. *Pediatr Pulmonol.* 1996; 21:176-183.

Marshall WA and JM Tanner. Variations in the Pattern of Pubertal Changes in Boys. *Archives of Disease in Childhood.* 1970; 45:13-23.

Maynard LM, Wisemandle W, Roche AF, Chumlea WC, ,et al. Childhood body composition in relation to body mass index. *Pediatrics*.2001; 107(2):344-350.

Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, Dietz WH. Validity of body mass index compared with other body composition screening indexes fro the assessment of body fatness in children and adolescents. *Am J Clin Nutr*. 2002; 75:979-985.

Miller FJW, Billwewicz WZ, Thomson AM. Growth from birth to adult life of 442 Newcastle-upon-Tyne children. *Br J Prev Soc Med*. 1972; 26:224-230.

Monello LF and MayerJ. Obese adolescent girls: unrecognized 'minority' group? *Am J Clin Nutr*. 1963; 13:35-49.

Moses N, Banlivy MN, Lifshitz F. Fear of obesity among adolescent girls. Pediatrics. 1989; 83:393-398.

Must A and Strauss RS. Risks and consequences of childhood and adolescent obesity. *Intl J of Obes*. 1999; 23(Suppl 2); S2-S11.

Must A, Jacques PF, Dallai GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents: A follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med*. 1992; 327:1350-1355.

National Center for Health Statistics, Centers of Disease Control and Prevention. More American Children and Teens are Overweight. Accessed 2.27.2003. Available online at: http://www.cdc.gov/nchs/releases/01news/overwght99.htm

Neumark-Sztainer D, Butler R, Palti H. Dieting and binge eating: Which dieters are at risk? *J Am Diet Assoc*. 1995; 95:586-589.

Neumark-Sztainer D, Story M, French SA, Hannan PJ, Resnick MD, Blum RW. Psychosocial concerns and health-compromising behaviors among overweight and nonoverweight adolescents. *Obes Res*. 1997; 5:237-249.

Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999-2000. *JAMA*. 2002:288(14):1728-1732.

Patton GC, Johnson-Sabine E, Wood K, Mann AH, Wakeling A. Abnormal eating attitudes in London schoolgirls - a prospective epidemiological study. *Psych Med*. 1990; 20:382-394.

Pinhas-Hamiel O, Dolan LM, Daniels SR, Standiford D, Khoury PR, Zeitler P. Increased incidence of non-insulin-dependent diabetes mellitus among adolescents. *J Pediatr*.1996;128:608-615.

Power C, Lake JK, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. *Int J Obes.* 1997; 21:507-526.

Power C, Lake JK, Cole TJ. Body mass index and height from childhood to adulthood in the 1958 British born cohort. *Am J Clin Nutr.* 1997; 66(5):1094-1101.

Pugliese MT, Lifshitz F, Grad G, Fort P, Marks-Katz M. Fear of obesity: a cause of short stature and delayed puberty. *N Engl J Med.* 1983; 309:513-518.

Rames LK, Clarke WR, Connor WE, et al. Normal blood pressures and the elevation of sustained blood pressure elevation in childhood: the Muscatine study. *Pediatrics.* 1978; 61:245-251.

Rhodes SK, Shimoda KC, Waid LR, Mahlen P, Oexmann MJ, Collop NA, Willi SM. Neurocognitive deficits in morbidly obese children with obstructive sleep apnea. *J Pediatr.* 1995; 127:741-744.

Richards GE, Cavalli A, Meyer WJ, Prince MJ, Peters EJ, Stuart CA, Smith ER. Obesity, aconthosis nigricans, insulin resistance and hyperandrogemia: pediatric perspectives and natural history. *J Pediatr.* 1985; 107:893-897.

Richardson SA, Goodman N, Hastorf AH, Dornbusch SM. Cultural uniformity in reaction to physical disabilities. *Am Sociol Rev.* 1961; 26:241-247.

Riley DJ, Santiago T, Edelman NH. Complications of obesity-hypoventilation syndrome in childhood. *Am J Dis Child.* 1976; 130:671-674.

Rimm IJ and Rimm AA. Association between juvenile onset obesity and severe adult obesity in 73,532 women. *Am J Pub Health.* 1976; 66(5):479-481.

Riumallo J and Durnin JV. Changes in body composition in adolescent boys. *Eur J Clin Nutr.* 1988 Feb; 42(2):107-12.

Roberts SB and Dallal GE. The new childhood growth charts. *Nutrition Reviews.* 2001; 59(2):31-36.

Roche AF, Wellens R, Attie KM, Siervogel RM. The timing of sexual maturation in a group of US white youths. *J Pediatr Endocrin & Metab.* 1995; 8(1):11-18.

Rolland-Cachera MF, Bellisle B, Sempe M. The prediction in males and females of the weight/height2 index and various skinfold measurements in adults: a two-decade follow-up study. *Int J Obesity.* 1989; 13:305-311.

Rolland-Cachera MF, Deheeger M, Bellisle F, Sepre M, Guilloud-Bataille M, Patois E. Adiposity rebound in children: a simple indicator for predicting obesity. *Am J Clin Nutr.* 1984; 39:129-135.

Sallade J. A comparison of the psychological adjustment of obese vs. non-obese children. *J Psychosom Res.* 1973; 17:89-96.

Sampliner RE, Bennet PH, Comess LJ, Rose FA, Burch TA. Gallbladder disease in Pima Indians demonstration of high prevalence and early onset by cholecystography. *N Engl J Med.* 1970; 283:1358-1364.

Scott IU, Siatkowski RM, Eneyni M, Brodsky MC, Lam BL. Idiopathic intracranial hypertension in children and adolescents. *Am J Opth.* 1997; 124:253-255.

Shaffer EA, Small DM. Biliary lipid secretion in cholesterol gallstone disease: the effect of cholecystectomy and obesity. *J Clin Invest.* 1977; 59:828-840.

Siervogel RM, Maynard LM, Wisemandle WA, Roche AF, Guo SS, Chumlea WC, Twone B. Annual changes in total body fat and fat-free mass in children from 8 to 18 years in relation to changes in body mass index. The Fels Longitudinal Study. *Ann NY Acad Sci.* 2000; 904:420-423.

Siervogel RM, Roche AF, Guo S, Mukherje ED, Chumlea C. Patters of change in weight/stature2 from 2 to 18 years: findings from long-term serial data of children in the Fels longitudinal growth study. *Int J Obes.* 1991; 15:479-785.

Silvesti JM, Weese-Mayer DE, Bass MT, Kenny AS, Hauptmann SA, Pearsall SM. Polysomnography in obese children with a history of sleep-associated breathing disorders. *Pediatr Pulmonol.* 1993; 16:124-129.

Sobal J and Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull.* 1989; 105:260-275.

Srinirvasan SR, Bao W, Wattigney WA, Berenson GS. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: the Bogalusa Heart Study. *Metabolism.* 1996; 45:235-240.

Stark O, Atkins E, Wolff OH, Douglas JWB. Longitudinal study of obesity in the National Survey of Health and Development. *Br Med J.* 1981; 283:13-17.

Statistical Package for the Social Science, Version 10.0.5. 1999. SPSS Inc. Chicago, IL.

Steinberger J, Moorehead C, Katch V, Rocchini AP. Relationship between insulin resistance and abnormal lipid profile in obese adolescents. *J Pediatr.* 1995;126:690-695.

Story M. School-based approaches for preventing and treating obesity. *Int J Obesity*. 1999; 23S2:S43-S51.

Strauss RS. Self-reported weight status and dieting in a cross-sectional sample of young adolescents. *Arch Pediatr Adolesc Med*. 1999; 153:741-747.

Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986-1998. *JAMA*. 2001; 286(22):28452848.

Stunkard A and Burt V. Obesity and the body image: II. Age at onset of disturbance in the body image. *Am J Psychiat*. 1967; 123:1443-1447.

Tanner JM. Normal growth and techniques of growth assessment. *Clinics in Endocrinology and Metabolism*. 1986; 15(3):411-451.

Tanner JM, Davies PSW. Clinical longitudinal standards for height and height velocity for North American children. *J. Pediatr*. 1985; 107(3):317-329.

Taranger J and U Hagg. The timing and duration of adolescent growth. *Acta Odontol Scand*. 1980;38(1):57-67.

Taylor WC, Chan W, Cummings SS, Simons-Morton BG, Day RS, Sangi-Haghpeykar H, Pivarnik JS, Mueller WH, Detry MA, Wei II, Johnson-Masotti AP, Hsu HA. Healthy Growth: project description and baseline findings. *Ethn Dis*. 2002; 12(4):567-577.

Tazawa Y, Noguchi H, Nishinomiya F, Takadda G. Serum alanine aminotransferase activity in obese children. *Acta Paeditr*. 1997;86:238-241.

Thorkild I, Sorensen A, Sonne-Holm S. Risk in childhood of development of severe adult obesity: retrospective, population-based case-cohort study. *Am J Epidemiol*. 1988; 127(1):104-113.

Tominaga K, Kurata JH, Chen YK, Fujimoto E, Miyagawa S, Abe I, Kusano Y. Prevalence of fatty liver in Japanese children and relationship to obesity. *Dig Dis Sci*. 1995; 40:2002-2009.

Troiano RP and Flegal KM. Overweight children and adolescents: Description, epidemiology, and demographics. *Pediatrics*. 1998; 101:497-504.

Troiano RP, Flegal KM, Kuczmarski RJ, ,Campbell SM, Johnson CL. Overweight prevalence and trends for children and adolescents. *Arch Pediatr Adolesc Med*. 1995; 149:1085-1091.

Unger R, Kreeger L, Christoffel KK. Childhood obesity: medical and familial correlates and age of onset. *Clin Pediatr.* 1990; 29:368-373.

U.S. Department of Health and Human Services. *Healthy People 2010: Objectives for Improving Health.* Washington, D.C.: U.S. Government Printing Office. 2000.

Vajro P, Fontanella A, Perna C, et al. Persistent hyperaminotransferasemia resolving after weight reduction in obese children. *J Pediatr.* 1994; 125:239-241.

Wang Y, Adair L. How does maturity adjustment influence the estimates of overweight prevalence in adolescents from different countries using an international reference? *Int J Obes.* 2001; 25:550-558.

Wang Y, Monteiro C, Popkin BM. Trends of obesity and underweight in older children and adolescents in the United States, Brazil, China, and Russia. *Am J Clin Nutr.* 2002; 75:971-977.

Weisberg LA and Chutorian AM. Pseudotumor cerebri of childhood. *Am J Clin Nutr.* 1977; 131:1243-1248.

Wells JCK. Body composition in childhood: effects of normal growth and disease. *Proceedings of the Nutrition Society.* 2003; 62:521-528.

Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med.* 1997; 37(13):869-873.

Wilcox PG, Weiner DS, Leighley B. Maturation factors in slipped capital femoral epiphyses. *J Pediatr Orthop.* 1988; 8:196-200.

Williams S, Davie G, Lam F. Predicting BMI in young adults from childhood data using two approaches to modeling adiposity rebound. *Int J Obes.* 1999; 23:348-354.

World Health Organization Expert Committee on Physical Status. The Use and Interpretation of Anthropometry. *Physical Status: Report of a WHO Expert Committee: WHO Technical Report Series 854*, WHO, Geneva, 1996.

Yanovski SZ. Binge eating disorder: current knowledge and future directions. *Obes Res.* 1993; 1:306-324.

Zack PM, Harlan WR, Leaverton PE, Cornoni-Huntley J. A longitudinal study of body fatness in childhood and adolescence. *J Pediatr.* 1979; 95(1):126-130.