

DIET AND MEDIA COMPONENTS RELATED TO CENTRAL ADIPOSITY IN
PREPUBERTAL CHILDREN

BY

NAIMAN A. KHAN

DISSERTATION

Submitted in partial fulfillment of the requirements
for the degree of Doctor of Philosophy in Nutritional Sciences
in the Graduate College of the
University of Illinois at Urbana-Champaign, 2012

Urbana, Illinois

Doctoral Committee:

Associate Professor Charles H. Hillman, Chair
Professor Sharon M. Donovan, Director of Research
Associate Professor Ellen M. Evans
Professor Kristen Harrison
Professor Craig Gundersen
Interim Director of EFNEP & SNAP-Ed Vicki Rowe

ABSTRACT

Overweight and obesity in childhood have tripled since 1980 and currently stand at 30% and 17% among 2-19-year-olds (Ogden et al., 2010), respectively. Excess fat mass in childhood is a strong predictor of obesity in adulthood and can lead to life-threatening medical conditions, such as diabetes, coronary heart disease, and certain cancers (Flegal et al., 2010). Previous studies have utilized BMI as the primary outcome and failed to measure diet and activity-related effects on actual fat mass. Abdominal adipose tissue (central adiposity) in children has been previously correlated with higher plasma lipids, elevated blood pressure, and insulin resistance (Daniels et al., 1999; Morrison et al., 1999; Krekoukia, 2007). Furthermore, children above the recommended values for waist circumference-for-age ($\geq 75^{\text{th}}$ percentile) and waist-to-height ratio (≥ 0.5) have elevated lipid profiles and higher insulin resistance (Savva, 2000). These studies suggest that central adiposity may be the most clinically relevant distribution of body fat in children, as is the case with adults. The overall objective of this research was to determine how media use and diet intake, independently or in concert, affect central adiposity in prepubertal children. Our central hypothesis was that poor diet quality is related to increased central adiposity, independent of aerobic fitness. The aims of our study were to determine the relationships between: 1) media use and central adiposity; and 2) diet and central adiposity.

Prepubertal children (113 females and 116 males) were recruited from an on-going NIH-funded afterschool physical activity research trial (FITKids). Pubertal staging was assessed using a modified Tanner Staging System (Tanner, 1962; Taylor, 2001). Parents reported their child's weekday and weekend television (TV) and videogame use. Maximal oxygen consumption ($\text{VO}_{2\text{max}}$) and one 24-hour recall were used to assess fitness level and dietary intake, respectively. Dual energy X-ray absorptiometry (DXA) was used to measure whole body percent fat mass

(WB %FM) and central adiposity (FM-abd). Gender-specific waist circumference-for-age percentiles (WC-age) and waist-to-height ratio (WHtR) were determined to further assess central adiposity.

Body composition results showed that females (N=105) had significantly higher WB %FM ($p<0.001$) and FM-abd ($p<0.001$) compared to males (N=109). Among females, pubertal staging was positively related to WB %FM ($r=.37$, $p<0.001$) and FM-abd ($r=.48$, $p<0.001$). Pubertal staging was not related to adiposity measures among males ($p>0.05$). However, fitness was inversely related to FM-abd among females ($r=-.59$, $p<0.001$) and males ($r=-.47$, $p<0.001$).

Weekend TV time among females was related to increased FM-abd ($r=.17$, $p=0.03$) and WHtR ($r=.22$, $p<0.001$), after controlling for fitness. Females who watched more than 2 hours of TV/day had significantly higher FM-abd ($p=0.03$). Females above the recommended WC-age cutoff ($\geq 75^{\text{th}}$ percentile) had significantly higher cholesterol-saturated fatty index (CSI) ($p=0.04$) and intake of added sugars ($p<0.001$). CSI was the only dietary variable related to increased FM-abd among females, after controlling for both fitness and energy intake ($r=.26$, $p=0.03$). However, no diet-body composition relationships remained significant after controlling for fitness and energy intake among males.

Standard linear regression models suggested that the strongest predictors of FM-abd among females were pubertal staging ($\beta=.418$, $p<0.001$) and fitness ($\beta=-.405$, $p<0.001$). The addition of energy intake, added sugars, and CSI accounted for an additional 10% of the variance in FM-abd. The same model explained 53% of the variance in WHtR. Among males, age was a positive predictor ($\beta=.306$, $p=0.005$) and fitness was a negative predictor ($\beta=-.453$, $p<0.001$) of FM-abd. Subsequent addition of diet variables only accounted for 0.5% of the variability in FM-abd. Fitness was the strongest predictor of WHtR ($\beta=.331$, $p=0.005$). Among females, pubertal

staging accounted for 13% of the variability in WB %FM. Energy from protein was a positive predictor of whole body adiposity ($\beta=.265$, $p=.007$). However, the contribution of energy from protein became non-significant when energy from fat was included in the model. The overall model accounted for 52% of the variability in WB %FM. Among males, age ($\beta=.335$, $p=.001$) and fitness ($\beta=-.427$, $p<0.001$) remained the strongest predictors of WB %FM.

In conclusion, females and males differed across all measures of adiposity, including the primary outcome of FM-abd. Weekend TV time appeared to influence energy intake, cholesterol and added sugars, however, these relationships attenuated once energy intake was controlled for. Added sugars and cholesterol-saturated fatty acid index were important predictors of central adiposity. After controlling for fitness and energy intake, the influence of these nutrients was non-significant among males. Collectively, these results suggest that dietary and sedentary behaviors may have differential health-related outcomes for prepubertal children based on gender. Body composition appeared to be significantly influenced by pubertal staging in girls and chronological age in boys. Therefore, our findings have implications for nutrition intervention efforts targeting obesity prevention during the critical period of preadolescence. Future work is needed to assess influence of diet and media on central adiposity as children transition into adolescence.

ACKNOWLEDGEMENTS

First, I would like to thank Dr. Robin Orr for taking a chance on me many years ago. Although Robin and I only worked together for a year, lessons from her brief mentorship will always be a source of motivation through my professional and personal endeavors. My training was unlike that of any other student I have known and I am very grateful for having a committee that allowed me the creative freedom to pursue my research interests. I would especially like to thank Dr. Sharon Donovan for acting as my advisor, and for her committed guidance and assistance towards the progress of my doctoral project. I learned a great deal about being a researcher and an educator by working closely with her over the last three years. I would also like to thank Dr. Ellen Evans for accepting me into the Bone and Body Composition Laboratory and for being a relentless proponent for my success. I also wish to thank Dr. Charles Hillman for involving me in the FITKids research trial and allowing me to utilize several aspects of a wonderful dataset. I would also like to thank Dr. Kristen Harrison and Dr. Craig Gundersen for serving on my committee, and for their encouragement and significant help with the conceptualization and analysis of my research project. In addition to the faculty members on my committee, I owe great gratitude to Dr. Vicki Rowe for her encouragement over the years and providing me with assistantship support throughout my doctoral studies.

I owe the success of this project to many who have assisted and supported me over the years. For teaching me everything I know about DXA, I would like to thank Dr. Mina Mojtahedi. For their help in collecting diet recalls and anthropometrics I would like to thank the members of the Neurocognitive Kinesiology Laboratory. I also had the good fortune to have a

strong circle of colleagues and friends over the years including Dr. Brittany Boyle, Brooke Schantz, Summer Porter, Dr. Chris Moulton, Jonathan Mun, and Matt Panasevich.

I would also like to thank my family members for their love and encouragement. My fiancée Hannah for supporting me through numerous challenges, personal and professional. My brothers: Saleem, Adnan, and Azeem for supporting and encouraging me. Finally, I am forever indebted to my parents for making tremendous sacrifices throughout their lives so that their children could pursue their goals and dreams.

TABLE OF CONTENTS

LITERATURE REVIEW.....	1
RESEARCH JUSTIFICATION.....	21
MATERIALS & METHODS.....	24
RESULTS.....	30
TABLES & FIGURES.....	37
DISCUSSION.....	68
CONCLUSIONS & FUTURE DIRECTIONS.....	75
APPENDICES.....	78
LITERATURE CITED.....	105

LITERATURE REVIEW

Definition and Prevalence of Obesity

Overweight and obesity are defined as excessive fat accumulation that may impair health (Caterson, 2002). Although there are several accurate methods of measuring fat mass/adiposity including magnetic resonance imaging (MRI) hydrostatic weighing, dual energy X-ray absorptiometry (DXA), the cost and need for trained personnel limits the widespread use of these techniques in both clinical and public settings. Furthermore, there are no well-accepted ranges for adiposity in children or adults (Flegal, 2006). Therefore, weight adjusted for height [expressed as body mass index (BMI), calculated as weight in kg divided by square of height in meters] is often used as a surrogate measure of adiposity. This index was first devised by the Belgian statistician Adolphe Quetelet (1797-1874), who applied it to adults. Subsequently, it was shown that if fat mass was independent of height, then BMI correlated highly with adiposity (Benn, 1970; Keys et al., 1972; Romero Corral, 2008). Among adults, the BMI cutoffs for overweight (BMI ≥ 25) and obese (BMI ≥ 30) are based on fixed values that are related to health-risk (Janssen, 2002). According to these definitions, worldwide obesity has more than doubled since 1980 with approximately 1.5 billion adults estimated to be overweight. This upward trend has been especially rapid in developed countries including the United States where 68% of the adult population is overweight and 32% are obese (Flegal et al., 2010).

Among children, BMI varies considerably with age. Therefore, a child's BMI is compared with that of a reference population of the same gender and age to assess adequate growth throughout childhood (Ebbeling, 2008). Both the World Health Organization (WHO) and the Centers for Disease Control (CDC) (2000) growth charts provide reference populations

for determining BMI-for-age percentiles. In the United States, the CDC (2000) charts are primarily used for growth assessment of children over 2 years. According to the CDC charts, obesity in childhood has tripled since 1980 and current estimates of overweight ($\geq 85^{\text{th}}$ percentile) and obese ($\geq 95^{\text{th}}$ percentile) prevalence among children stand at approximately 32% and 17%, respectively. Prevalence differs across race and ethnicity; 39% of Hispanic and African-American children (6-11 years) have a BMI at or above the 85th percentile compared to 28% in Caucasian (Ogden et al., 2010). Although prevalence of obesity among all 2-19-year-olds has remained stable in recent years, trend analysis among boys shows significant increases since 2000. Of further concern is that the BMI distribution has continually shifted to the right since the 1980's, suggesting that not only has the proportion of overweight children increased, but the degree to which children are overweight has increased substantially as well (Stifel, 2009).

However, it should be noted that the CDC growth charts were based on a reference population prior to the 1980's, when obesity prevalence was stable and not considered a public health threat (Ebbeling, 2008). Additionally, the BMI-for-age percentile cutoffs for overweight or obese children are not based on health risk due to the lack of clear risk-related criteria. In fact, there remains much debate as to which cutoffs and what charts need to be utilized. Although the CDC recommended using the sex- and age-specific 85th and 95th BMI percentiles as the arbitrary cutoffs to define childhood overweight and obesity in the United States (Flegal, 2006), the European Childhood Obesity Group favored the 90th and 97th percentiles (Poskitt, 1995), and the International Obesity Task Force (IOTF) proposed age and gender-specific cutoffs that would correspond to the widely used adult BMI values of 25 and 30 (Cole, 2000). These cutoffs vary in definition and reference populations resulting in wide-ranging prevalence estimates for overweight, obesity, or both (Flegal et al., 2001).

Obesity and Health

Obese adults are at increased risk for many serious health conditions, including coronary heart disease, hypertension, stroke, type 2 diabetes, and certain types of cancer (Ogden et al., 2010). Diseases associated with obesity accounted for 27% of the increases in U.S. medical costs between 1987 and 2001 (Thorpe et al., 2004). In 2006, obesity-related medical costs were estimated at \$147 billion with obese persons paying \$1,429 higher than persons of normal weight (Finkelstein, 2009). Obesity in adulthood is linked to comorbidities that have been attributed to estimates as high as 365,000 deaths annually in the United States (Mokdad, 2004). After adjusting for smoking status, annual death risk among nonsmokers increases by 12–40% among overweight and by 50–150% among those who are obese (Adams, 2006; Lawlor, 2006). Obesity also decreases life expectancy by as little as 0.8 (Reuser, 2008) to as much as 7 years (Peeters et al., 2003; Muennig, 2006) relative to their normal weight counterparts. However, the most staggering statistic highlighting the urgency needed to address this public health challenge is that obesity, and its related comorbidities, are the second leading cause of premature death in the United States after smoking (Biro, 2010).

Obesity in childhood often persists into adolescence and adulthood, especially for those children belonging to the highest BMI quartile (Guo et al. 1999) or those with an overweight parent (Whitaker et al. 1997). Furthermore, the pathological processes of obesity-related morbidities (e.g. cardiovascular disease and diabetes) begin in childhood (Biro, 2010). Therefore, the increase in prevalence and severity of overweight in children is of particular concern. The incidence of type 2 diabetes among adolescents, although not high, has increased by 10-fold since 1990 and may exceed that of type 1 diabetes among black and Hispanic adolescents (Fagot-Campagna, 2000). Fatty liver associated with excessive weight,

unrecognized in the pediatric literature prior to 1980, today occurs in about one in three obese children (Ebbeling, 2002). Among a nationally representative sample of 10,099 children (5-14 years), 39% of participants belonging to the obese category (BMI-for-age $\geq 95^{\text{th}}$ percentile) had elevated LDL ($\geq 90^{\text{th}}$ percentile) (Freedman et al., 2007). There is growing evidence demonstrating that early stages of atherosclerosis, the leading cause of cardiovascular and cerebrovascular events, can appear in utero, infancy, or throughout childhood (Napoli, 1997; Napoli, 1999). Risk factors such as hypertension, insulin resistance, diabetes, or dyslipidemia are aggravated by obesity and further hasten development of atherosclerosis (Bacha et al., 2003). Even more disconcerting is that obesity in the adolescent years is associated with increased risk of multiple comorbidities in adulthood regardless of whether the obesity persists or not (Must, 1992; Ludwig, 2007). Although the exact mechanisms of this phenomenon are unknown, one possible theory is that obesity in childhood may cause damage to the vasculature that may be irreversible even after subsequent weight normalization in adulthood (Barton, 2012).

In addition to chronic systemic diseases, which often take decades to manifest, overweight children endure multiple social and psychological challenges throughout childhood. Research shows that obese youth face weight stigma from multiple sources including peers, educators and parents (Puhl, 2007). They are more likely to be socially isolated, have high rates of disordered eating, anxiety and depression (Sjöberg and Sjoberg, 2005). It also appears that overweight children are subject to higher frequency of bullying compared to their normal weight counterparts (Janssen, 2004; Hayden Wade, 2005). In a longitudinal study, consequences of being overweight in adolescence included lower rates of marriage, lower education attainment, lower family income, and higher poverty 7 years later (Dietz, 1998).

Effects of Central Adiposity

Most studies use BMI as a measure for adiposity, because it is a quick and inexpensive method to screen for overweight or obesity. However, assessing obesity solely on the basis of BMI can be problematic because obesity is defined as a condition characterized by excess fat accumulation and not just excess weight (Flegal, 2010). BMI provides no information on fat distribution and does not distinguish between lean and fat mass. These can be significant limitations since measures associated with abdominal/central adiposity have been more strongly linked to disease risk than BMI (Grundy, 2004). Central adiposity is a major component of the metabolic syndrome, which is strongly related to development for CVD and diabetes (Eckel, 2005). Intra-abdominal/visceral fat tissue, in particular, is related to insulin resistance and subsequent alterations in glucose homeostasis (Després, 2006a). For any given amount of total body fat, individuals with excess visceral adipose tissue, have a substantially higher risk for development of insulin resistance and manifestation of metabolic syndrome (Després, 2006b). There are three possible mechanisms by which visceral adipose tissue may contribute to an insulin resistant state; 1) hypertrophied visceral adipose tissue becomes resistant to lipolytic effects of insulin resulting in impaired non-esterified fatty acid (NEFA) metabolism; 2) reduced adiponectin levels causing alterations in glucose flux and lipid catabolism; and 3) reduced capacity to store excess adipose subcutaneously, leading to increase in fat deposition around the liver, pancreas and heart (ectopic fat) (Després, 2006a) Both altered NEFA metabolism and endocrine function are causally implicated in the association between visceral adipose tissue and the development of metabolic syndrome, which is often absent in individuals without visceral adiposity.

Clinical signs and symptoms associated with the metabolic syndrome has been identified in children and adolescents and appears to persist into adulthood (Terry and Huang, 2007). Some indications are that central adiposity among children may be increasing, independent of whole body weight (McCarthy, 2005; Moreno, 2005). Although DXA does not distinguish between visceral and subcutaneous fat tissue, it provides an accurate estimate of total abdominal fat mass with a minimal dose of radiation. In children, central adiposity measured by DXA was associated with higher plasma lipid and lipoprotein concentrations, blood pressure and left ventricular mass (Daniels et al., 1999). However, there are no established cutoffs to define excess central adiposity. Among adults, waist circumference is incorporated into the diagnosis of metabolic syndrome and predicts plasma lipids, lipoproteins and insulin levels (Pouliot et al., 1994; Conway et al., 1997; Taylor et al., 1998). Among children, two arbitrary waist circumference cutoffs (>0.5 waist-height-ratio and $\geq 75^{\text{th}}$ percentile waist circumference-for-age) have been associated with increased insulin resistance and CVD risk factors (Savva, 2000). Savva and colleagues attempted to validate BMI, waist-to-height ratio, and waist circumference-for-age as predictors of cardiovascular disease risk factors in children. Among a large sample of 1037 boys and 950 females (mean age 11.40 ± 0.4), belonging to the highest quartile ($\geq 75^{\text{th}}$ percentile) for all measures was related to higher pathological values of blood low-density lipoproteins (LDL) and total triglycerides (TG). However, waist circumference-for-age and waist-to-height ratio were better predictors of LDL compared to BMI in a stepwise regression analysis. Nevertheless, these cutoffs have not been adopted by expert committees on childhood obesity assessment due to lack of comprehensive reference values. Nevertheless, assessment of central adiposity by imaging techniques or waist circumference may improve our understanding a child's future disease risk better than whole body fat mass or BMI.

Etiology of Obesity

The underlying causes of childhood obesity have been the subject of many studies in recent years and we are finally beginning to understand some of the complex genetic and environmental mechanisms involved. The most widely accepted theories implicate the interaction between genetic predisposition and an obesogenic environment that results in efficient energy storage (Moreno et al., 2010).

Parental influences on childhood obesity have been of interest since investigators have tracked obesity-related risk factors to earlier stages of development. Parental obesity appears to affect childhood adiposity through genetic predisposition. This was first confirmed by an adoption study in Danish children that showed a significantly higher correlation between the BMI of the adoptees and their biological parents compared to their adoptive ones (Stunkard, 1986; Sorensen et al., 1989; Sorensen et al., 1989). Another study among twins exposed to under-and over-feeding showed a considerable genetic component (~50%) in intake-related weight gain (Bouchard and Tremblay, 1997). Maternal obesity, in particular, appears to be strongly related to obesity in children. A European cross-sectional study of 3,306 children (5-7 years) and their parents showed that child BMI had a stronger correlation with maternal compared to paternal BMI ($r=0.25$ vs. 0.16 , $p<0.01$) (Danielzik et al., 2002). These findings have been supported by similar epidemiological studies across the globe including Japan, Australia, and China (Luo and Hu, 2002; Sekine et al., 2002; Wang, 2002).

Environment-gene interactions have been proposed as a possible mechanism by which parents may influence risk for obesity in their children. Epigenetic traits are those that result in heritable phenotypic changes without alterations in underlying DNA sequence. The impact of such interactions may occur during and after intrauterine development. Evidence from animal

studies suggests that maternal consumption of a high fat diet during gestation is related to subsequent excess fat accumulation in rat pups (Wu, 2006), independent of diet intake. Other perinatal factors such as low protein intake have also been linked to higher birth weight (Whitaker, 1997). In humans, infants born to women with gestational diabetes have significantly higher fat mass than infants of women without gestational diabetes (Catalano et al., 2003). Collectively, the aforementioned studies support the hypothesis that early nutrition programming may have considerable effects on subsequent obesity development in children.

Dietary Components and Obesity

Although genetic susceptibility affects propensity for excess fat gain, it appears that environmental factors play a substantial role throughout development. Of the numerous risk factors known to modulate obesity and its persistence into adulthood, diet and physical activity are the main modifiable determinants of energy balance (Rodriguez and Moreno, 2006). Numerous components of diet have been the subject of research. However, few have examined this relationship in preadolescent children (Bowman et al., 2004; Bradlee, 2010).

Childhood and adolescence are critical periods of development characterized by continuous body growth, psychological changes, and onset of dietary habits that are likely to continue into adulthood (Boot et al., 1997; Lake, 2006). However, the relationships between body composition and energy intake related behaviors are reciprocal (Rodriguez et al., 2004). Excess adiposity develops when energy intake exceeds expenditure over a long period of time leading to accumulation of fat tissue. However, it is not clear which specific aspects of the child's diet need to be targeted for prevention. The major dietary factors previously studied include excess total energy intake (Atkin and Davies, 2000; Alexy et al., 2004), excess percent

energy from fats (McGloin, 2002; Alexy et al., 2004) and contemporary diet patterns (Berkey et al., 2004; Bowman et al., 2004; Fisher et al., 2007).

Several studies have found a positive association between total energy intake and adiposity in children (Gillis et al., 2002; Jouret, 2007). Gillis et al. (2002) found that obese children and adolescents had significantly higher energy intake than their non-obese peers, independent of physical activity levels and other dietary factors. In a longitudinal study, further weight gain was reduced in children who decreased energy intake while increasing physical activity (Berkey et al., 2000). However, other studies found no relationship between energy intake and overweight or obese status (Maffeis et al., 1998; Andersen et al., 2005; Aeberli et al., 2007). The differences seen in studies may be attributed to a wide variation in measurement methods and instruments as well as underreporting of dietary intake by children (Livingstone, 2004). Epidemiological studies often rely on self-reported dietary intake without the help of a trained interviewer, therefore placing a larger burden on children to conceptualize portion sizes and recall food ingredients/preparation methods (Jain et al., 1996). In summary, much of the evidence supports the argument that excess energy intake is positively related to overweight and obesity (Rodriguez, 2010). However, the impact of energy intake on weight status, independent of physical activity or fitness, is still incompletely understood.

In addition to energy intake, the percent energy intake from fat and absolute fat intake have also been examined in relation to obesity (Magarey et al., 2001). One of the studies (Ortega et al., 1995) showed a positive association for percent energy from fat, but not with fat intake in absolute amounts, in a group of adolescents. Contrary to this finding, Gillis et al. (2002) reported a significant association between fat in grams and adiposity, but not with percent energy from fat. Magarey and colleagues conducted a prospective study that followed children from

birth to 15 years and found that fat, independent of total energy intake, was positively associated with subscapular skinfold thickness, but not with BMI or tricep skinfold thickness. Other studies have assessed the type of dietary fat, rather than the amount of total dietary fat. Obese children and adolescents have been shown to consume higher levels of saturated fat, independent of total energy intake and physical activity (Gillis et al., 2002). In addition, specific types of fats mediate weight gain. A study of 4-year-olds showed that children with adequate levels of dietary n-3 fatty acids were less likely to be obese compared to their counterparts who consumed low levels of n-3 fatty acids (Garemo et al., 2007). However, it is important to note that other studies have found no association between dietary fat intake and obesity (Davies, 1997; Maffeis et al., 1998; Berkey et al., 2000). Although total energy intake may play a more significant role in adiposity, it is important to examine the types of dietary fat that may have an effect on body composition independent of energy intake.

Positive associations between sucrose intake and BMI in young children have been previously documented (Garemo et al., 2007). Cross-sectional (Gibson and Neate, 2007) and prospective studies (Ludwig, 2001) have shown that consumption of sugar-sweetened beverages increases the risk for obesity (Olsen and Heitmann, 2009). It is proposed that sugar-sweetened beverages can lead to a disruption of the body's satiety mechanisms, while providing high amounts of calories in a short period of time (Malik et al., 2006). However, not all studies have found positive associations between sucrose consumption and obesity (Forshee et al., 2008).

Studying dietary patterns provides an important way of assessing combinations of nutrients, as well as, the frequency and environmental contexts in which foods are consumed (Quatromoni, 2002). Several interacting factors affect family and child food dietary patterns, including level of income, food availability, eating outside of home and family meal climate

(Finch et al., 2006; Moreno et al., 2010). However, the relative contribution of each of these as well as other factors on diet patterns is incompletely understood.

In adults, meal frequency and daily distribution of meals has been shown to have a relationship with overweight and obesity. Adults who report four or more eating episodes per day are less likely to be obese than those who report three or fewer episodes (Ma et al., 2003). This may also be the case with children (Toschke, 2005). In terms of daily energy intake distribution, obese children appear to eat less at breakfast, skip breakfast, and consume excess calories at dinner (Bandini et al., 1990; Livingstone and Robson, 2000; Serra Majem et al., 2003; Moreno et al., 2005). Decreases in breakfast frequency have been shown to prospectively predict BMI during adolescence and young adulthood (11-27 years) (Gillman et al., 2000; Niemeier et al., 2006). Positive eating habits such as consuming breakfast and the frequency of family dinners have been inversely related to fried foods and soft drink consumption (Gillman et al., 2000).

Assessment of typical snack choices of children suggests that they offer plenty of calories, but little nutritional value (Rodriguez et al., 2004; Moreno and Rodriguez, 2007; Ambrosini, 2009). Ambrosini et al. (2009) noted that “snacky” consumption pattern that was positively loaded for biscuits, buns, sweets, salted snacks, soft drinks, and nuts among 1631 Australian adolescents. This pattern was also inversely related to maternal level of education and positively related to TV viewing. In large longitudinal studies, snacking has been identified as an independent predictor of weight gain among children and adolescents as well (Field, 2004; Phillips et al., 2004).

Meeting nutrient needs from a variety of food groups is a component of overall diet quality. A recent study showed that diet quality is independently associated with weight status in

preadolescents (Jennings, 2011). Comparison of three types of diet quality indices (Diet Quality Index, Healthy Diet Indicator, and Mediterranean Diet Score) demonstrated that an inverse relationship exists between diet quality scores and obesity in 1,700 children between 9- and 10-years-of-age (Jennings, 2011). Both in adults and children, dietary variety that includes fruits and vegetables is associated with a healthy weight, while dietary variety from sweets, snacks and carbohydrates is associated with adiposity (Miller, 2008; Receveur, 2008). Understanding the behaviors that interact and influence dietary variety will be relevant to improving nutrition programs or interventions targeted towards children.

Measuring dietary intake in children can be challenging for multiple reasons, including poor conceptualization of portion sizes, inaccurate recall of foods eaten, lack of knowledge of food preparation methods, and selective under-/over-reporting of food intake (Livingstone and Robson, 2000). Furthermore, each of the three major instruments used in diet data collection (diet recalls, food frequency questionnaires, and diet records) have inherent measurement issues (Crawford et al., 1994). Children younger than 10-years have difficulty responding to food frequency questionnaires that require recalling intake over more than 1 day (Rodriguez, 2010). Using parents as proxies is not entirely reliable either, since they are often unaware of their children's dietary intake away from home.

Family Factors related to Obesity in Children

It is widely accepted that obesity is a complex and multifactorial condition influenced by genetic and environmental factors. Although the role of the home environment in the development of obesity has long been recognized, few studies have documented this relationship. The relationship between socioeconomic status (SES) and obesity remains inconclusive. It was

previously accepted that high SES groups in the United States are less likely to become overweight than their low-SES counterparts (Sobal, 1994; Sundquist, 1998; Wang, 2001). Recent trends show that an inverse relationship between SES and overweight status only exists for white children and a strong positive association existed in black adolescents (Wang and Zhang, 2006). Wang and Zhang (2006) also noted that the gap between ethnic groups became wider between 1971 and 2002, especially in adolescent girls. A review of the literature suggests that a third of the studies have found a positive relationship between SES and obesity, a third demonstrate a negative relationship and a third of the studies show no relationship at all (Strauss, 1999a). Variations may be due to the differences in ages of children studied and the measures of SES used. However, data from the Pediatric Nutrition Surveillance System indicate that the prevalence of obesity continues to increase in low-income preschool children among all ethnic groups (Mei, 1998). Studying specific aspects of households at different levels of SES may provide more relevant information for interventions.

Strauss (1999) examined the role of race, marital status, maternal education, and family income as well as standardized measures of home environment (using the Home Observation for Measurement of Environment Short Form) in the development of childhood obesity. Analysis of longitudinal data among 2913 normal weight children (0 to 8 years) participating in the National Longitudinal Survey of Youth (NLSY) (Strauss, 1999a) revealed that children raised in home environments with higher cognitive stimulation have the lowest rates of obesity independent of SES, race and maternal BMI. Lack of cognitive stimulation and emotional support has been shown to increase childhood overweight in other studies as well (Garasky et al., 2009). This suggests that specific stressors on the home environment may play a significant role in development of obesity.

Although a significant relationship appears to exist between parental and child weight the effect of parental level of education on childhood obesity is still unclear. Strauss (1999) found significant relationships between maternal education level, parental occupation, or marital status and incidence of obesity in their children. However, this relationship was not significant for the black children, whose obesity rates have increased the most over the past 3 decades. It is suggested that SES affects social and intellectual development by diminishing the capacity for supportive and consistent parenting (McLoyd, 1990). The personal resources of the mother such as here self-esteem, values, and occupational experience are reflected in the overall quality of the home environment (Menaghan, 1991) which is related to the cognitive stimulation of the child. Gundersen (2008) examined associations between food insecurity and maternal stressors with childhood overweight among low-income children. Analysis of 841 children from the 1999-2002 NHANES data revealed that younger children in food secure, low-income households experiencing higher levels of maternal stressors have a greater probability of being overweight than their food secure counterparts.

A household is considered 'food insecure' if it does not have the financial means to access enough food for all household members to sustain active and healthy living (Nord, 2010). Households that are more likely to be food insecure include those with low-income, headed by black non-Hispanic or Hispanic person, or headed by single parent (Nord, 2010). Some studies have found a positive relationship between food insecurity and child obesity (Casey et al., 2001; Dubois et al., 2006), while other studies have found either no relationship (Bhargava, 2008; Gundersen, 2008) or a negative one (Rose, 2006).

Although recent evidence shows no relationship between household food insecurity and child obesity, both conditions often coexist in low-income households (Gundersen, 2009).

Gundersen et al. (2009) analyzed 2,516 children (8-17 years old) from the 2001-2004 NHANES data and found that 56% of food insecure children were obese. There are many possible reasons why food insecure children could be obese including intake of cheaper calorically dense foods (Drewnowski and Specter, 2004), overeating when food is plentiful (Scheier, 2005), metabolic changes (Alaimo et al., 2001), parents overfeeding when food is plentiful (McIntyre et al., 2003) and maternal food insecurity during pregnancy (Laraia et al., 2006). Furthermore, the impact of food insecurity on child obesity appears to be different for preschool- versus school-aged children, with the former being more sensitive to it. It is possible that the nutritional outcomes of school-aged children (6-17 years) are less closely tied to family resources compared to preschool children. School-aged children may be supplementing their consumption away from home by eating at school, at “friends” or “neighbors”, or through income they earn through working (Bhattacharya, 2004).

In summary, there isn't sufficient evidence to support any single attribute of a child's diet or environment as an independent predictor of obesity. Dietary components that warrant further investigation include added sugars, types of fat, and overall energy intake. Often studies examining the nutrition-obesity relationship use BMI as an outcome and fail to measure actual fat mass or distribution. This is a significant limitation because whole body weight loss is often a daunting goal and focusing on a measure of fat distribution (e.g. waist circumference) may be a better therapeutic target (Després, 2006b). Additional research needs to be conducted to measure diet effects on direct measures of adiposity and fat distribution.

Media Components and Obesity

Media-related sedentary activities may include, but are not limited to, TV, computer and video game use (Henry J. Kaiser Family Foundation, 2010). Studies assessing video game and computer use have found little or no association with overweight or obesity in youth (Rey-López, 2008). Most studies have reported a positive association between TV viewing and adiposity. In adults, TV viewing predicts obesity, development of type 2 diabetes mellitus (Hu, 2001; Hu, 2003), and dyslipidemia (Fung, 2001). In children, TV viewing, independent of activity levels, has been associated with poor metabolic profiles (Sardinha, 2007). This relationship is strongest in children younger than 10-years-of-age suggesting that TV is a major risk factor for overweight and obesity in prepubertal children (Rey-López, 2008) In fact, reducing TV viewing has been an important part of successful obesity interventions among youth (Robinson, 1999; Epstein, 2000; Ludwig, 2004).

Current estimates of TV viewing indicate that youth watch an average of 4.5 hours of TV/day (Henry J. Kaiser Family Foundation, 2010). Child's access to video games increases daily screen time by 60 minutes for boys and 23 minutes for girls (Marshall, 2006). Computer use also increases screen time by 30 minutes/day. The high level of TV viewing is particularly troubling because children's TV exposure predicts overweight (Lumeng et al., 2006) and increases caloric consumption at the rate of 167 kilocalories per additional daily hour of viewing (Wiecha, 2006). This additional caloric consumption could be attributed to eating while watching TV, however, exposure to food advertising maybe have a more significant long-term contribution to excessive caloric intake. Zimmerman et al. (2010) studied associations between television content type and demonstrated that TV advertisement, rather than viewing time, is associated with obesity in children.

Children younger than 5 years see an average of 4000 television commercials (30 hours' worth) per year (Gantz, 2007). Several studies have shown that there is a lack of advertisements for nutritious foods on TV including programming geared to children (Wiecha et al., 2006). Analyzing nutrition content of foods advertised during weekend TV programming targeted at children (Saturday mornings) and adults (Sunday evenings) revealed that 97.5% of food commercials advertised unhealthy foods on Saturday mornings versus 78.3% on Sunday evenings (Kuribayashi et al., 2001). Foods were categorized as unhealthy if a serving of the food contained significantly high amounts of fat (>30% calories from fat), sugar (>30% calories from sugar), cholesterol (>35mg), or sodium (>360 mg). Children are influenced by food messaging, to the extent that food depictions affect food purchase requests (Donkin et al., 1993), their understanding of nutrition principals (Peterson, 1984), and their ability to self-regulate their intake of high calorie snacks (Dawson, 1988). It appears that no matter when children view TV, they are likely to be exposed to an abundance of advertisements for foods of low nutritional quality.

Previous studies have also shown a negative association between TV viewing and nutritional knowledge/beliefs. Signorielli and Staples (1997) provided 427 fourth- and fifth-graders with six pairs of foods and asked them which one of the choices was healthier. TV viewing predicted greater likelihood of selecting the unhealthy choice between the pairs, independent of child's gender, race, reading level, and parental education level. These studies suggest that children with higher TV viewing may have poor beliefs of nutritious foods. Harrison (2005) used a similar methodology to Signorielli & Staples (1997), with the additional measure of nutrition reasoning to study the impact of TV viewing in 134 first-third graders. In addition to asking children to identify the healthier food, they were asked to provide a reason for

why they thought that food was healthier. Results showed that TV viewing predicted subsequent decrease in both nutritional beliefs and reasoning. However, this relationship was strong only for diet-related foods. This was a meaningful find because it suggests that the link between TV viewing and concepts of weight loss/diet foods is stronger than the link between TV viewing and concepts of “regular foods” e.g. fruits, vegetables, and breads. Children are exposed to the numerous weight loss/diet-related food product advertisements meant for adults. Most of these lower calorie/fat/sugar items are marketed as “healthy” and can be a source of misinformation for children. This is cause for concern, because children, unlike adults, are still in the process of attaining peak bone and lean mass and what is considered “healthy” for adults is not always healthy for the growing child.

Dieting Behaviors and Obesity

Often childhood obesity studies neglect assessment of the psychosocial influences on children’s dietary choices and how they may be influenced by media. Evidence suggests that many adolescent girls who consider themselves overweight are actually of normal weight and are engaging in weight loss-related behaviors (Strauss, 1999b). These studies also reveal gender and ethnic differences in desire to be thin. Using the NHANES III data among 1932 adolescents, Strauss (1999) reported that the children’s own classification of whether they were overweight correlated poorly with medical definitions of overweight. Of the children who considered themselves overweight, 42% had a BMI less than the 85th percentile and 70% had a BMI less than the 95th percentile. Only 28% of the children who considered themselves overweight had a BMI between the 85th and 95th percentiles. Girls were more than twice as likely than boys to want to weigh less, while the majority of the boys desired to weigh more. A higher percentage

of white females, compared to black females, considered themselves to be overweight and have attempted to lose before they reach puberty. Adolescent black females also appear to be less concerned about being overweight (Wilson et al., 1994), feel less societal pressures to be thin, and are seven times more likely to report that they are not overweight (Kemper et al., 1994) compared to their white peers. Similarly, adolescent black males select larger ideal body sizes and are more likely to report being overweight less frequently than their white counterparts (Thompson, 1996). Only a few studies have examined desire to be thin in children prior to puberty. However, it has been reported that children acquire cultural values to beauty prior to adolescence and their desire to be thin precedes the desire to be beautiful (Feldman et al., 1988). In a cross-sectional study survey of first through third graders, 42% of females (6-7 years of age) preferred a body type different from their own (Collins 1991). Given that much of culture and media glorifies thinness, children and adolescents who feel that they are overweight may be engaging in harmful weight loss behaviors e.g., skipping meals and binge-eating (Neumark-Sztainer, 1999). These behaviors may produce the counterproductive effect of further weight gain (Field, 2003; Neumark Sztainer, 2007).

Fitness and Obesity

The reasons for the high rates of obesity in the U.S. children have been debated on the two sides of the energy balance equation: energy intake and energy expenditure (Schneider et al., 2007). Some researchers attribute obesity to excessive intake (Utter et al., 2003; Bleich et al., 2008), while others suggest that the current sedentary lifestyles of children and adolescents are primarily responsible for the increased obesity rates (Prentice, 1995). As discussed previously, dietary intake in children has been related to obesity in many studies, but not all. Therefore, it is

necessary to study sedentary behaviors in concert with diet to assess their collective influence on weight status. Both obesity and physical inactivity have similar associations with clinical risk factors (e.g. blood pressure, fasting blood glucose and inflammatory markers (Fagard, 1999; Wei et al., 1999; Church, 2002).

Furthermore, low fitness levels may be independently related to CVD risk among children. Results from 9-18-year-olds participating in the Quebec Family Study suggest that low levels of aerobic fitness were related to higher blood lipids compared to their higher fit counterparts within the same BMI category (Eisenmann, 2005). Multiple studies suggest that physical activity is related to decreased %body fat in children (Rowlands et al., 1999; Epstein, 2000). Rowlands and colleagues studied associations between children's activity (8-10-year-olds), aerobic fitness, and adiposity. Activity measured by pedometers correlated positively with fitness and negatively with adiposity. Epstein and colleagues utilized an intervention targeting sedentary behaviors in 8-12-year-olds. Results showed decreases in sedentary behaviors or increases in physical activity were associated with significant decreases in %body fat and improved fitness.

Majority of studies on obesity have failed to account for physical activity and cardiorespiratory fitness among their participants. The most common method of assessing physical activity involves relying on self-report questionnaires which are subject to over reporting and subsequent misclassification (Blair, 2004). Using maximal exercise tests to measure cardiorespiratory fitness provides an objective evaluation of an individual's activity patterns (Paffenbarger et al., 1993). Given that physical activity is closely related to obesity, there is a need to utilize accurate methods for assessment of both when studying the effects of behavioral factors on disease risk.

RESEARCH JUSTIFICATION

Targeting prevention in children is essential to reducing the life-long health burden associated with obesity. In order to design effective interventions, it is essential to understand the dietary and media use determinants that contribute to the development of central adiposity at critical developmental stages of childhood. Television viewing has long been studied as a physical activity-related practice (Eisenmann, 2008; Vicente-Rodríguez, 2008). However, emerging evidence suggests that this practice may also have significant nutritional consequences as well (Harrison, 2005; Miller, 2008; Zimmerman, 2010). In addition to predicting higher BMI-for-age, TV viewing is a source of nutrition misinformation and studying its influence on nutrition beliefs and reasoning may provide a link between children's dietary choices and media use (Signorielli and Staples, 1997; Harrison, 2005). Although several studies have assessed diet in relation to obesity, there is limited knowledge regarding the specific components that should be targeted. Furthermore, previous studies have failed to assess fitness and fat distribution when evaluating the role of behavioral factors in the development of childhood obesity.

Cardiorespiratory fitness, determined by maximal oxygen consumption (VO_{2max}), is stronger than self-reported physical activity as a predictor of health outcomes because it is less prone to misclassification (Blair, 2004). Additionally, abdominal adipose tissue (central adiposity) measured using DXA has been previously correlated with higher plasma lipids, elevated blood pressure, and insulin resistance in children (Daniels et al., 1999; Morrison et al., 1999; Krokoukia, 2007). There is mounting evidence that children with waist circumference-for-age and waist-to-height ratio above the 75th percentile or greater than 0.5, respectively, have dyslipidemia and higher insulin resistance than children below these cutoffs (Savva, 2000;

Ashwell, 2005). These studies suggest that central adiposity may represent the most-clinically relevant distribution of body fat to assess in children, as is the case with adults.

The research proposed here will examine associations between the prepubertal child's media use, diet and body fat distribution. Using DXA will provide an accurate method of assessing total body and central adiposity. The interaction between diet and media use will be examined in order to assess whether nutrition beliefs play a role in this relationship. Outcomes will be analyzed while considering the child's personal attributes e.g. age, gender, and pubertal staging. Finally, we will develop regression models that predict measures of central adiposity using diet and media use. There is a dearth of knowledge regarding behavioral factors related to central obesity among preadolescents. Studying these relationships during this stage of development is critical given that obesity persists into adulthood for a majority of obese adolescents (Dietz, 1997; Engeland, 2004). Additionally, identification of dietary and media use components related to central adiposity will provide important targets for behavioral intervention.

The *overall objective of this research* is to determine how media use and diet intake, independently or in concert, affect central adiposity in prepubertal children. Our *central hypothesis* is that poor diet quality is related to increased central adiposity, independent of cardiorespiratory fitness. **Figure 1** illustrates the aims and expected relationships between the major outcomes of the study.

Aim 1: Determine media use components related to central adiposity

The overall objective of this aim is to determine how total screen-time and components of media use (TV, computer or video game) relate to central adiposity in prepubertal children. *The working hypothesis* for this aim is that total screen-time and TV viewing are directly related to

central adiposity, independent of fitness level. It is also expected that children with high TV viewing will have lower overall nutrition beliefs.

Aim 2: Determine dietary components related to central adiposity

The overall objective of this aim is to determine how dietary intake relates to central adiposity in prepubertal children. *The working hypothesis* for this aim is that dietary components associated with lower diet quality (e.g., added sugars and saturated fat) will be related to increased central adiposity. It is expected that these relationships will remain significant after controlling for fitness and energy intake.

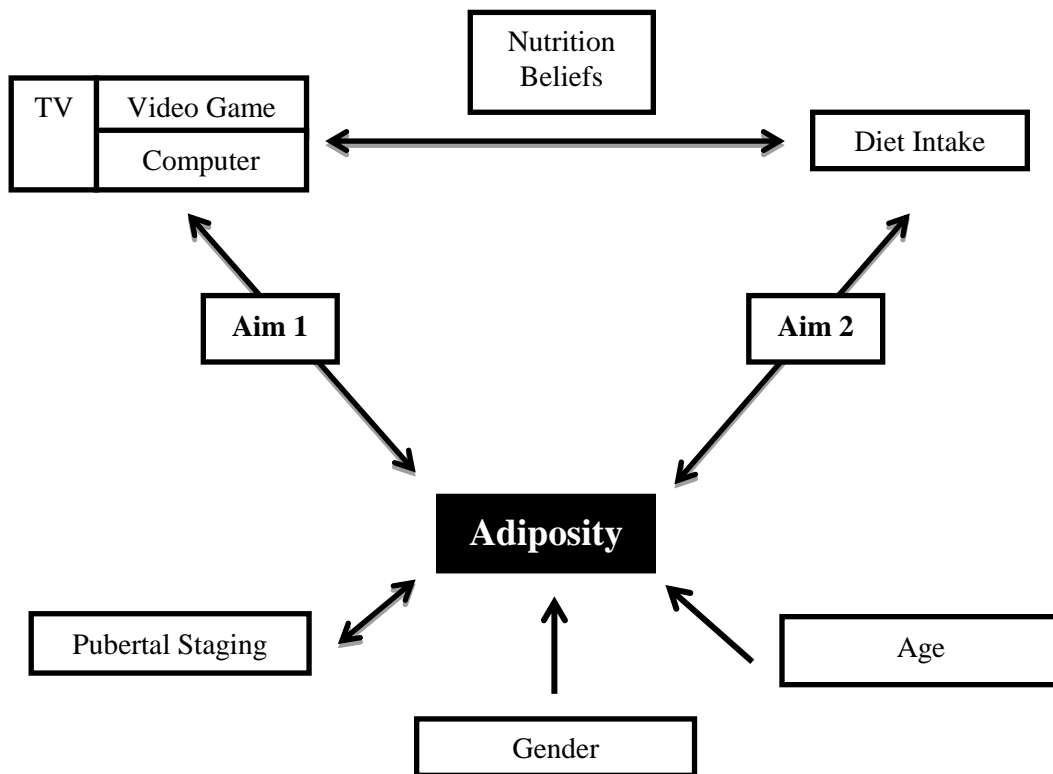


Figure 1: Relationships between media use, diet, and adiposity

MATERIALS & METHODS

Study Design and Participants

A four-year cross-sectional study (2008-2011) was undertaken to explore the relationships between demographic variables, diet, screen time, and central adiposity in a group of prepubertal children. Participants were recruited from an on-going NIH-funded after-school physical activity research trial (FITKids; HD055352; Charles Hillman, principal investigator).

Figure 2 illustrates the four waves of the study and sequence in which measures were added.

The study was approved by the Institutional Review Board of the University of Illinois and informed consent was attained from parent and child. All measures were assessed at baseline to avoid any effects of the intervention.

Measures

Data was collected over two days of testing. On day 1, either parent was asked to complete a questionnaire on family demographics and their child's media use (TV, computer, and video game viewing). Following a cardiorespiratory fitness test, the child was interviewed and asked to report his/her dietary intake using a 24-hour diet recall. Day 2 testing involved collection of child's anthropometric measures and body composition using DXA. Additional measures of diet and media use were included in the final year of the study (wave IV). Wave IV additions included Children's Eating Attitudes questionnaire (ChEAT), 3-day food journal, and a 2-day media log.

Demographic Information: Parents responded to a demographics and health history questionnaire (**Appendix A**) to report their level of education, household income and occupational status. Additional questions included child's race, birth weight and type of school lunch (free/reduced price/paid). SES was determined by creating an index based on: (1) participation in free or reduced-price meal program at school, (2) the highest level of education obtained by the mother and father, and (3) number of parents who worked full-time.

Media use: Over the first three waves of the study, child's media use was measured using a section of the demographical questionnaire (Appendix A) in which parents reported their child's weekday and weekend media use (TV, video games, and computer). However, a 2-day take-home media log was introduced for wave IV (**Appendix B**). The 2-day media log was adopted from a previous study (Henry J. Kaiser Family Foundation, 2010) and allows children to report upto 12 media-related activities over half hour segments of the day (6am-12pm).

Dietary Intake: With parental assistance, children reported intake of all foods and beverages by responding to one 24-hour recall (**Appendix C**). The Nutrition Data Systems for Research 2010 (Minneapolis, MN) was used to analyze all intake data. A three-day food record (**Appendix D**) was introduced in wave IV as an additional high quality measure of diet intake (Crawford, 1994).

Nutrition Beliefs : Children responded to a Nutrition Beliefs and questionnaire (**Appendix E**) modified from previous work by Harrison (2005). Diet and fat-free versions of common foods are included to capture the child's beliefs and related to foods advertized as weight-loss or "healthier" options for adults.

Dieting Behavior: Children's dieting behaviors were assessed using children's healthy eating attitudes test (ChEAT) (Maloney et al. 1988). This is a self-report test that consists of subscales related to disturbed eating attitudes (Appendix F).

Pubertal Staging: Pubertal staging was determined by a modified Tanner Staging system (Appendix G) (Tanner, 1962; Taylor, 2001). Parents were allowed to complete the questionnaire in privacy or allowed to take home and return on a later date.

Anthropometrics: Child's barefoot standing height was measured to the nearest 0.1 cm with a stadiometer. Body weight was measured on a calibrated balance scale with participants wearing shorts and a t-shirt. *Waist and hip* circumferences were used to further assess the child's degree of central adiposity. Waist circumference was measured as the minimum circumference between the top of the iliac crest and the distal end of the rib cage along the midaxillary line and at the umbilicus. Hip circumference was measured as the maximal girth of the hips region (Maffeis et al., 2001). These measures were recorded in the Anthropometric form (Appendix H).

Body Composition: Body composition was assessed by DXA (Hologic QDR 4500, Bedford, Massachusetts). DXA measures included total and central adiposity. Central adiposity was defined as the amount of fat mass located between lumbar vertebral bodies L1 and L4 (Svendsen et al., 1993; Treuth et al., 1995). DXA adds minor risk to participants with minimal radiation exposure of one 1 mrem. At this dose level, no harmful effects of radiation have been documented and the risk is negligible.

Cardiorespiratory Fitness Assessment: Maximal oxygen consumption (VO_{2max}) was measured using a computerized indirect calorimetry system (ParvoMedics TrueMax 2400) with averages for oxygen uptake (VO_2) and respiratory exchange ratio (RER) assessed every 20 seconds. A modified Balke protocol (Gordon, 2009) was employed using a motor-driven treadmill at a constant speed with increases in grade increments of 2.5% every 2 minutes until volitional exhaustion. Heart Rate (HR) throughout the test and ratings of perceived exertion (RPE) was assessed every 2 minutes using the children's OMNI scale (Utter and UTTER, 2002). Relative peak oxygen consumption was expressed in ml/kg/min and was based upon maximal effort as evidenced by: 1) a plateau in oxygen uptake corresponding to an increase of less than 2 ml/kg/min despite an increase in exercise workload; 2) a peak heart rate (HR) ≥ 185 bpm (ACSM, 2006) and a HR plateau (Freedson and Goodman, 1993); 3) RER ≥ 1.0 (Bar Or, 1983); 4) ratings on the children's OMNI scale of perceived exertion ≥ 8 (Utter and UTTER, 2002). VO_{2max} percentile was determined according to previously provided normative data (Shvartz and Reibold, 1990).

Statistical Analyses

All data analyses were conducted using SPSS version 19.0 (SPSS, Inc., Chicago, IL). Normality in anthropometric, DXA, diet and media variables was determined using Q-Q plots and Shapiro-Wilk. Log transformations were utilized, as needed. Independent t-tests and one-way ANOVA were used to determine differences between groups. Males and females were grouped together when studying relationships between diet and media variables since there were no differences in overall screen-time or energy intake by gender. However, females and males differed significantly on multiple body composition variables including the primary outcome of

central adiposity, therefore, further analyses related to body composition were separated by gender.

Initial Pearson product-moment correlations were conducted between measures of body composition (i.e., central adiposity [FM-abd], whole body % fat mass [WB %FM], BMI, waist-to-height ratio [WHtR]), demographic factors (i.e., age, sex, race, socioeconomic status [SES], VO₂max percentile), and pubertal timing. Partial correlations were conducted between media and diet variables and body composition. The dietary components selected for analyses were total energy intake, macronutrients (protein, fat, and carbohydrates), saturated fats, added sugars, glycemic load, and cholesterol saturated fatty acid index. These components were selected based on prior studies in the literature implicating them in development of obesity in varying degrees. Finally, standard linear regression analyses were conducted for each gender to predict FM-abd, WHtR, and WB %FM. Pubertal timing, age and VO₂max percentile were included in the initial steps of all regression analyses due to their established relationship with adiposity in children. Diet and media measures were then added to the subsequent models. Data are expressed as mean ± standard deviation in text and tables. All significance tests were conducted at the alpha=0.05 level.

Figure 2. Outcomes assessed with each wave of the FITKids study

Wave I (2008)	Wave II (2009)	Wave III (2010)	Wave IV (2011)	N
Fitness				229
Body Composition				217
Media – Parent Reported				219
	↓ Diet Recalls			152
		↓ Nutrition Beliefs		96
			↓ Diet Records	32
			Dieting Behaviors	38
			Media log – Child Reported	28

RESULTS

Subject Demographics

Demographic characteristics from all participants (N=229; 113 female, 116 male) separated by gender are reported in **Table 1**. The majority of the participants were white and 40% were of lower SES. Over half of males and females belonged to Tanner stage 1 with a larger proportion of males at Tanner stage 2 compared to females (40% vs. 33%). However, there were differences in household income with a larger proportion of females belonging to households earning between \$80,000-99,999 and more males belonging to households earning between \$40,000-59,999.

Anthropometrics

Anthropometric characteristics from all participants (N=229; 113 female, 116 male) separated by gender are reported in **Table 2**. Although there was no difference in age, females had significantly higher weight ($p=0.02$), waist circumference ($p=0.01$), waist-to-height ratio ($p=0.02$), and BMI ($p=0.01$) than their male counterparts. Children were classified by BMI-for-age using the CDC (2000) growth charts and these results are summarized in **Table 3**. Although the proportion of normal weight children was similar across gender groups, a larger proportion of females were classified as obese compared to males (32% vs. 23%).

Body Composition by DXA

Utilization of DXA allowed for further classification based on WB %FM and the results are summarized in **Table 4**. Findings confirmed the group classifications made by BMI-for-age,

with a higher proportion of females being classified as obese compared to males (28% vs. 18%). Females had significantly higher whole body fat ($p<0.001$), total mass ($p=0.01$), and WB %FM ($p<0.001$) (**Figures 3 & 4**). Furthermore, females had significantly higher FM-abd ($p<0.001$) compared to males (**Figure 5**). Given the significant adiposity differences among females and males, additional analyses were conducted separately based on gender. Bivariate correlational analyses among females showed that pubertal staging is positively related to WB %FM ($r=0.37$, $p<0.001$) and FM-abd ($r=0.48$, $p<0.001$) (Table 5). Conversely, no statistically significant relationship was observed between pubertal staging and adiposity measures among males ($p=0.32$ and $p=0.08$) (**Table 6**). However, cardiorespiratory fitness was inversely related to FM-abd among females ($r=-0.59$, $p<0.001$) and males ($r=0.47$, $p<0.001$).

Fitness

There were no significant differences between male and female mean VO_{2max} percentile ($p=0.82$). VO_{2max} percentile comparisons between groups based on weight-status (BMI-for-age) are summarized in **Figure 6**. Normal weight children had significantly higher VO_{2max} percentile than both overweight ($p<0.001$) and obese ($p<0.001$) counterparts. However, the difference between overweight and obese children was not significant ($p=0.05$).

Media Use

Differences in media consumption between females and males are summarized in **Figure 7**. Males have higher weekly videogame and weekend screen time compared to females. However, there were no differences in TV and computer time between genders. Among females, weekend TV time was related to increased FM-abd ($r=0.17$, $p=0.03$) and WHtR ($r=0.22$,

$p < 0.001$), after controlling for the influence of fitness (**Table 7**). No media use variables showed significant correlations with measures of adiposity after controlling for fitness among males (**Table 8**). Within group differences in FM-abd were assessed based on the American Academy of Pediatrics recommendation of less than 2 hours of TV/day (**Figure 8**) (Strasburger, 2011). Females who watch more than 2 hours of TV/weekday had significantly higher FM-abd compared females who met the recommendation ($p = 0.03$). Differences in FM-abd were not significant among males across the TV use cutoff of 2 hours ($p = 0.09$). Media use was also assessed using 2-day media logs among wave IV participants. However, response rates were low with only 28 participants returning the logs on day 2 of testing. Furthermore, all media variables measured by media logs displayed deviations greater than their means and were therefore excluded from analyses.

Dietary Intake

Descriptive analyses of the 24-hour diet recalls are summarized in **Tables 9** and **10**. Intakes among both groups exceeded recommendations for calories from saturated fat and sodium. Additionally, children failed to meet their recommended intakes for several nutrients including protein, fiber, and potassium. Although there were no significant differences in macronutrient (protein, fat, and carbohydrates) intake between males and females (**Figure 9**), both groups reported consuming higher than recommended energy intake. **Table 11** summarizes comparisons between intake reported by 24-hour recalls and 3-day food records during wave IV of the study. There were no significant differences between intakes ($p > 0.05$) suggesting agreement between the two assessment methodologies.

Within gender differences in energy intake were assessed based on BMI-for-age classification as normal weight (5th-84th percentile) and overweight or obese (\geq 85th percentile). Overweight females had higher energy intake ($p=0.04$) compared to their normal weight peers (**Figure 10**). However, there were no significant differences in energy intake between overweight males and normal weight males ($p=0.84$) (**Figure 11**). Diet differences among females were further assessed by comparing those above or below the waist circumference-for-age cutoff (75th percentile) (**Figures 12 and 13**). Females above the cutoff had significantly higher CSI ($p=0.04$) and added sugars ($p<0.001$) compared to females below the cutoff. Partial correlations between diet variables and body composition are summarized in **Tables 12 and 13**. The CSI was the only dietary variable related to higher FM-abd among females. This relationship remained significant after controlling for both fitness and energy intake ($r=0.26$, $p=0.03$). However, no diet-body composition relationships remained significant after controlling for fitness and energy intake among males.

Relationships between diet and media use

Females and males were analyzed as one group when assessing relationships between media use and dietary intake. Bivariate correlation of media use and diet showed that weekday TV time was positively related to energy intake ($r=0.21$, $p<0.001$), cholesterol ($r=0.15$, $p=0.04$), added sugars ($r=0.20$, $p=0.02$) and CSI ($r=0.17$, $p=0.03$). Total screen time on weekdays was related to increased cholesterol ($r=0.17$, $p=0.02$), saturated fatty acids ($r=0.17$, $p=0.18$) and added sugars ($r=0.17$, $p=0.03$). However, after controlling for screen time and energy intake, weekend TV time was related to increased added sugars ($r=0.15$, $p=0.04$) and decreased CSI ($r=-0.15$, $p=0.04$).

Nutrition Beliefs

Reliability analysis of the nutrition beliefs questionnaire is summarized in **Table 14**. Overall, the questionnaire had moderate internal consistency based on the Cronbach alpha score of 0.63. After removing two items, the diet vs. high energy/low quality subscale (**Table 15**) had high internal consistency ($\alpha=0.86$) and was utilized in further analyses. The questionnaire was scored by awarding a point for selecting the nutrient dense/healthy food item in all comparisons. For comparisons between diet and high-energy/low-nutrient foods, participants were awarded one point for selecting the diet version of the food instead of the high-energy/low-nutrient option. Therefore, high overall scores on the questionnaire reflect increasing selection of healthy foods and low scores indicate increased selection of the high-energy/low-nutrient food items. Although there were no significant relationships with energy intake, bivariate correlations revealed that total nutrition belief scores were inversely related to screen time/weekday ($r=-0.27$, $p=0.04$) among females ($N=52$). Furthermore, nutrition belief scores were related to increased intake of vitamin D ($r=0.37$, $p=0.01$) and calcium ($r=0.42$, $p=0.003$) and reduced energy intake from fat ($r=-0.31$, $p=0.02$). Among males ($N=39$), increased scores on the diet vs. high-energy/low-nutrient subscale were inversely related to intake of energy from fats ($r=-0.35$, $p=0.02$) and saturated fats ($r=-0.30$, $p=0.03$). These analyses are summarized in **Tables 16** and **17**.

Dieting Behaviors

Only 38 participants responded to the questions corresponding to the dieting subscale of the ChEAT (Cronbach $\alpha=0.76$). Therefore, both genders were grouped together for further analyses. Bivariate analyses revealed that dieting subscale scores were related to increased FM-

abd, WHtR, WB %FM, and BMI. Diet data was only available for 30 of these respondents. Increasing scores on the dieting subscale were related to increased carbohydrate intake ($r=0.32$, $p=0.04$) and decreased intake of cholesterol ($r=-0.32$, $p=0.04$).

Regression Models

Given the significant differences in body composition between females and males, regression models were developed for each gender group. Standard linear regression models were performed to assess the independent effects of dietary variables on FM-abd, WHtR, and WB %FM. Age, pubertal staging and VO_{2max} percentile were included in the models of all regression analyses due to prior research indicating their relationship with adiposity in children. Dietary variables were then added to the subsequent models. The α level for statistical significance was set at 0.05.

Predictors related to FM-abd were analyzed and the complete results are displayed in **Tables 18 and 19**. Among females, pubertal staging alone accounted for 23% of the variance in FM-abd. However, addition of age did not significantly improve the model. The addition of fitness in model 3 significantly improved the model and explained an additional 22% of the variance. Model 6 includes energy intake, added sugars and CSI in addition to all prior variables and accounts for 53% of the variance in FM-abd. The strongest predictors of FM-abd were pubertal staging ($\beta=.418$, $p<0.001$) and fitness ($\beta=-.405$, $p<0.001$). Among males, pubertal staging was not a major contributor to FM-abd ($\beta=-.044$, $p=0.652$). However, age was a positive predictor ($\beta=.306$, $p=0.005$) and fitness was a negative predictor ($\beta=-.453$, $p<0.001$) of FM-abd. Subsequent addition of diet variables only accounted for an additional 0.5% of the variability in FM-abd.

Standard linear regression was also performed to determine the predictive ability of energy, added sugars, and CSI on WHtR. **Table 20** summarizes the findings among females. The results were similar to those developed for FM-abd with the overall model accounting for 53% of the variability in WHtR. Among males (**Table 21**), after addition of all variables, fitness was the strongest predictor of WHtR ($\beta=-.331$, $p=0.005$).

Regression analyses predicting WB %FM are summarized in **Tables 22** and **23**. Among females, pubertal staging alone accounted for 13% of the variability in WB %FM. Model 5 suggests that, energy from protein was a positive predictor of whole body adiposity ($\beta=.265$, $p=0.007$). However, when energy from fat is included in the model, the contribution of energy from protein becomes non-significant. The overall model accounted for 52% of the variability in WB %FM. Among males, age ($\beta=.335$, $p=.001$) and fitness ($\beta=-.427$, $p<0.001$) remained the strongest predictors of WB %FM.

TABLES & FIGURES

Table 1. Participant demographics and background

		Females (%)	Males (%)
Race	Asian	12	12
	Black	25	26
	White	47	47
	Other or Mixed	12	14
	Missing	4	1
Pubertal staging	Tanner Stage 1	64	58
	Tanner Stage 2	33	40
	Missing	3	2
Socioeconomic Status	Low	45	44
	Moderate	23	20
	High	31	35
	Missing	1	1
Household Income	≤19,999	21	15
	20,000-39,999	24	26
	40,000-59,999	11	24
	60,000-79,999	14	12
	80,000-99,99	16	8
	≥100,000	12	13
	Missing	2	2

Table 2. Anthropometric and fitness differences between females and males

	Females (N=113)	Males (N=116)
Birth weight (kg)	3.41 ± 0.80	3.47 ± 0.97
Age (years)	8.89 ± 0.59	8.80 ± 0.58
Weight (kg)	38.6 ± 11.3*	35.0 ± 11.57
Height (cm)	136.8 ± 6.93	134.9 ± 7.87
Waist Circumference (cm)	67.03 ± 10.24*	63.76 ± 8.54
Waist-to-height ratio	0.49 ± 0.06*	0.47 ± 0.065
BMI (kg/m ²)	19.9 ± 4.06*	18.40 ± 4.07
VO _{2max} percentile	19.14 ± 22.04	18.51 ± 20.38

All values presented as Mean ± SD

* Statistically significant difference between groups (p<0.05)

Table 3. Distribution of weight status by BMI-for-age percentiles

	Females (%) (N=113)	Males (%) (N=116)
Underweight (<5 th)	3	3
Normal Weight (5-84 th)	47	52
Overweight (85-94 th)	18	22
Obese (≥95 th)	32	23

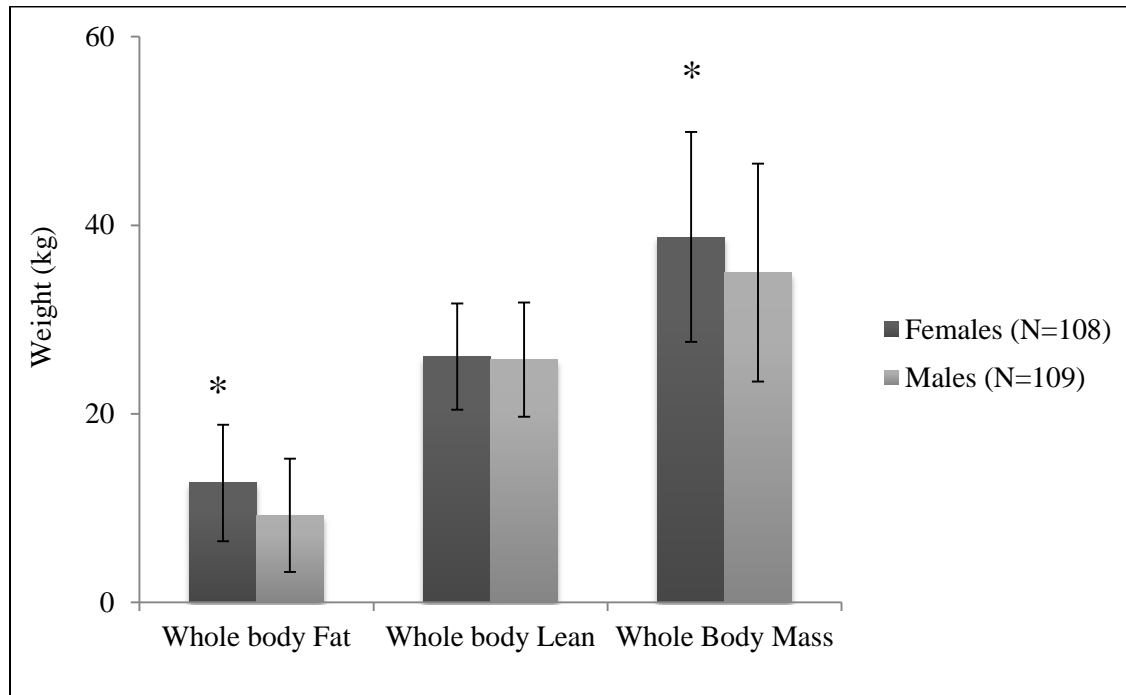
Participants were categorized using the Centers for Disease Control and Prevention growth charts (Kuczmarski et al., 2000).

Table 4. Classification whole body %adiposity as measured by DXA

	Females (%) (N=105)	Males (%) (N=109)
Low	1	0
Average	47	58
High	20	18
Obese	28	18
Missing	4	6

Participants were categorized based on previously provided whole body %adiposity reference values (Heyward, 2004)

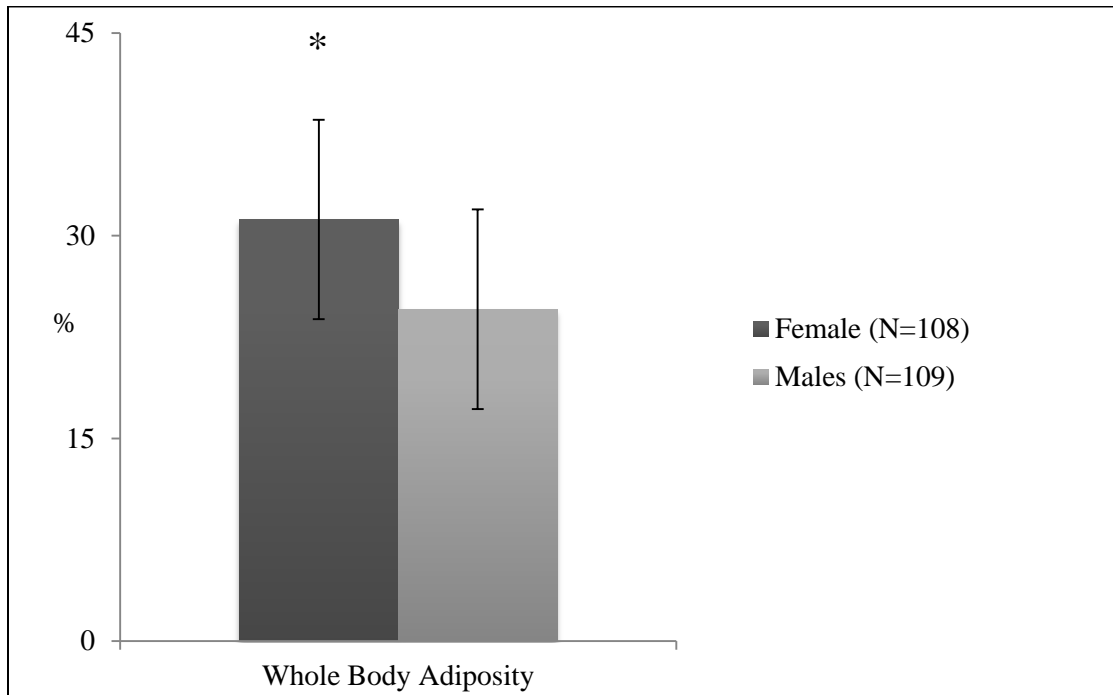
Figure 3. Females have higher whole body fat and total mass than males



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA.

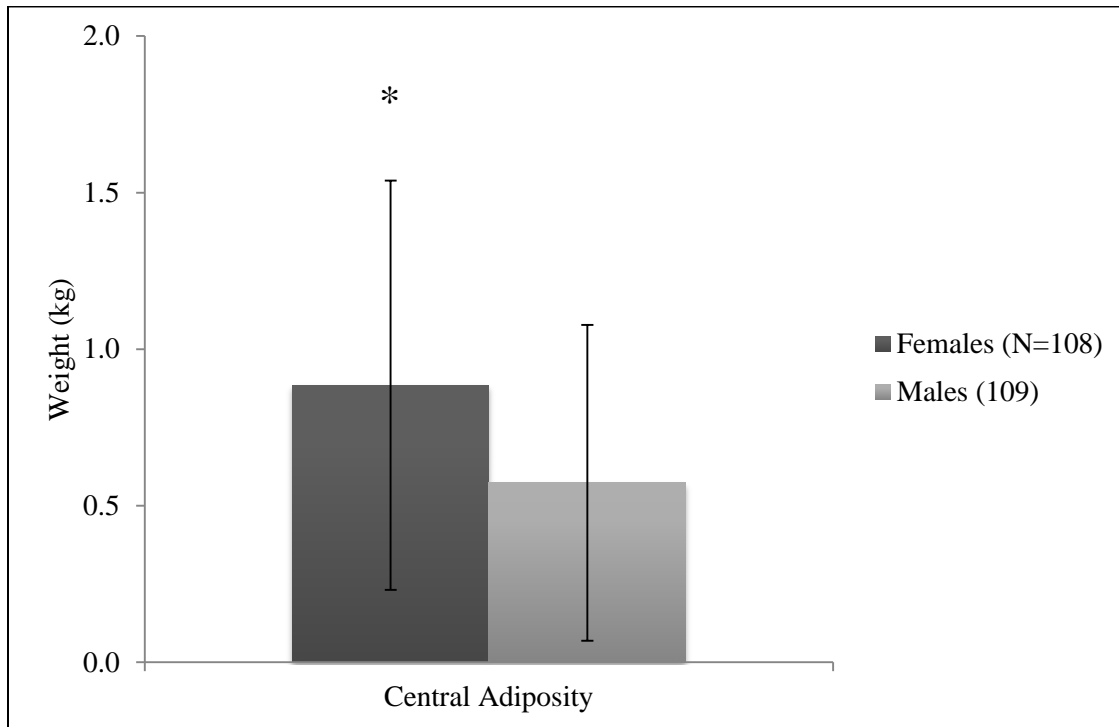
Figure 4. Females have higher whole body % adiposity than males



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA.

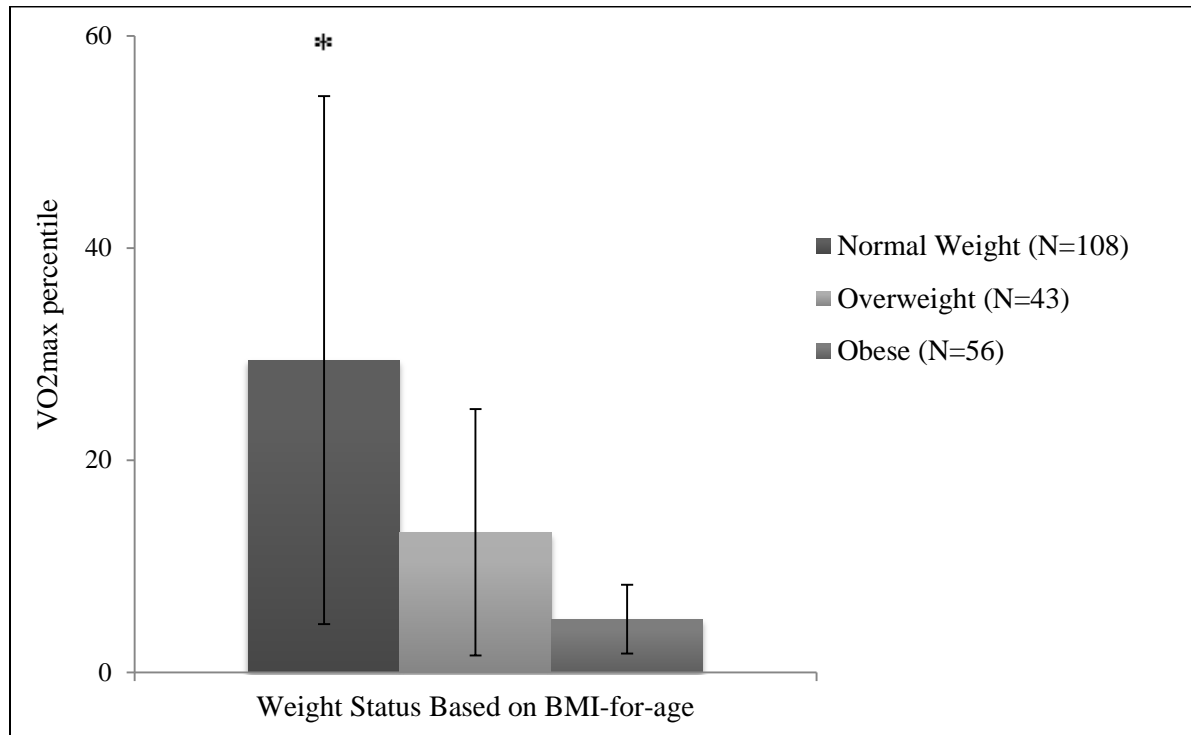
Figure 5. Females have higher central adiposity than males



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA.

Figure 6. Normal weight children have higher fitness than overweight and obese children



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA. Participants were categorized using the Centers for Disease Control and Prevention growth charts (Kuczmarski et al., 2000).

Table 5. Pubertal staging and age are related to increased central adiposity among females (N=106)

	1	2	3	4	5	6	7	8
1. Central Adiposity	—							
2. Waist-to-height ratio	.86**	—						
3. Whole body % Adiposity	.93**	.76**	—					
4. Body Mass Index	.86**	.85**	.76**	—				
5. VO _{2max} percentile	-.59**	-.50**	-.61**	-.54**	—			
6. Age	.19*	.06	.11	.16*	-.06	—		
7. Socioeconomic Status	-.10	-.21*	-.08	-.17*	.19*	-.01	—	
8. Pubertal Staging	.48**	.46**	.37**	.51**	-.29**	.29**	-.12	—

**significant at the 0.01 level (one-tailed)

*significant at the 0.05 level (one-tailed)

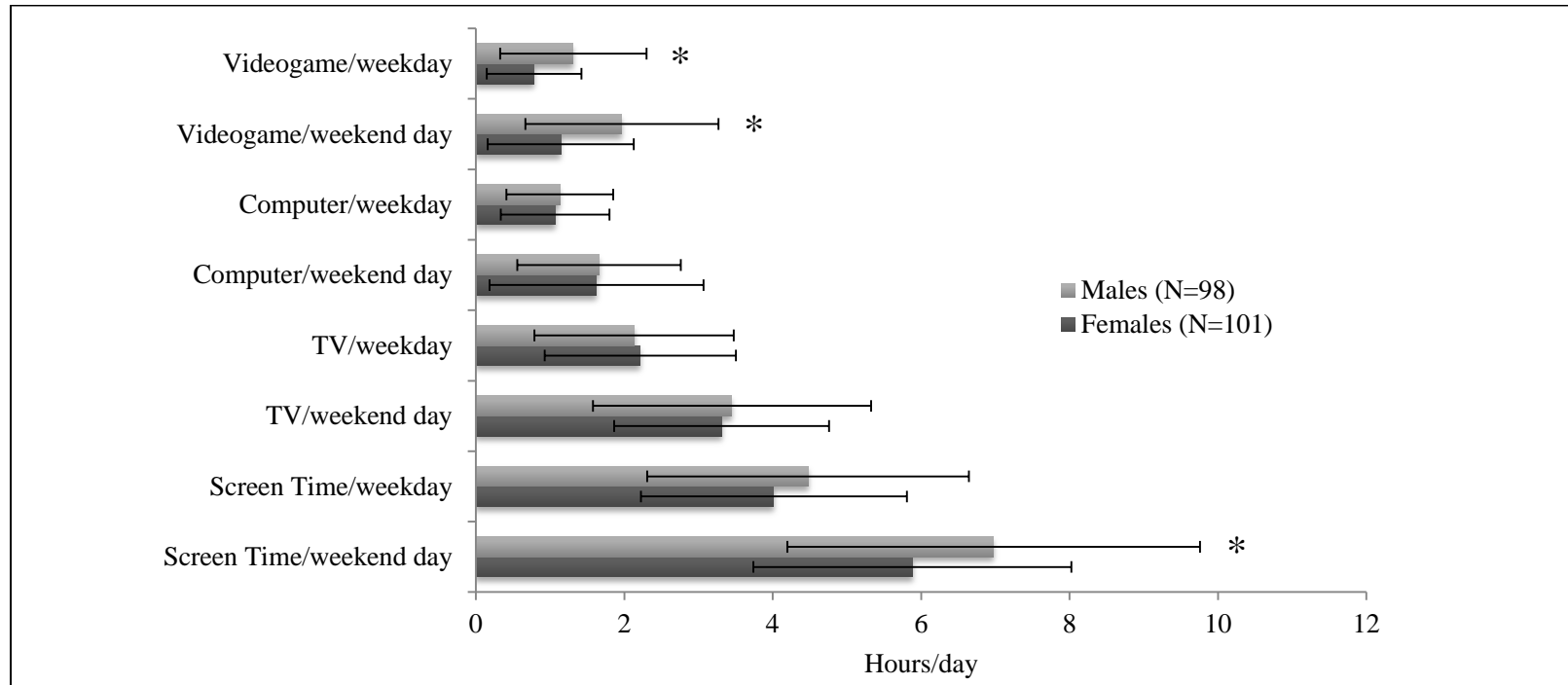
Table 6. Pubertal staging is not related to central adiposity among males (N=107)

	1	2	3	4	5	6	7	8
1. Central Adiposity	—							
2. Waist-to-height ratio	.84**	—						
3. Whole body % Adiposity	.94**	.80**	—					
4. Body Mass Index	.86**	.83**	.78**	—				
5. VO _{2max} percentile	-.48**	-.39**	-.53**	-.46**	—			
6. Age	.26**	.09	.20*	.20*	-.09	—		
7. Socioeconomic Status	-.09	-.11	-.10	-.10	.240**	-.07	—	
8. Pubertal Staging	-.05	-.12	-.15	-.02	.00	.18*	-.01	—

**significant at the 0.01 level (one-tailed)

*significant at the 0.05 level (one-tailed)

Figure 7. Males have higher videogame and weekend screen time than females



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA.

Table 7. Weekend TV time is related to increased central adiposity among females (N=99), after controlling for fitness

	1	2	3	4	5	6
1. Central Adiposity	—					
2. Waist-to-height ratio	.82**	—				
3. Whole Body % Adiposity	.88**	.72**	—			
4. Body Mass Index	.81**	.79**	.68**	—		
5. Television/weekday	.06	.13	.04	.15	—	
6. Television/weekend day	.17*	.22*	.07	.25*	.60	—

**significant at the 0.01 level (one-tailed)

*significant at the 0.05 level (one-tailed)

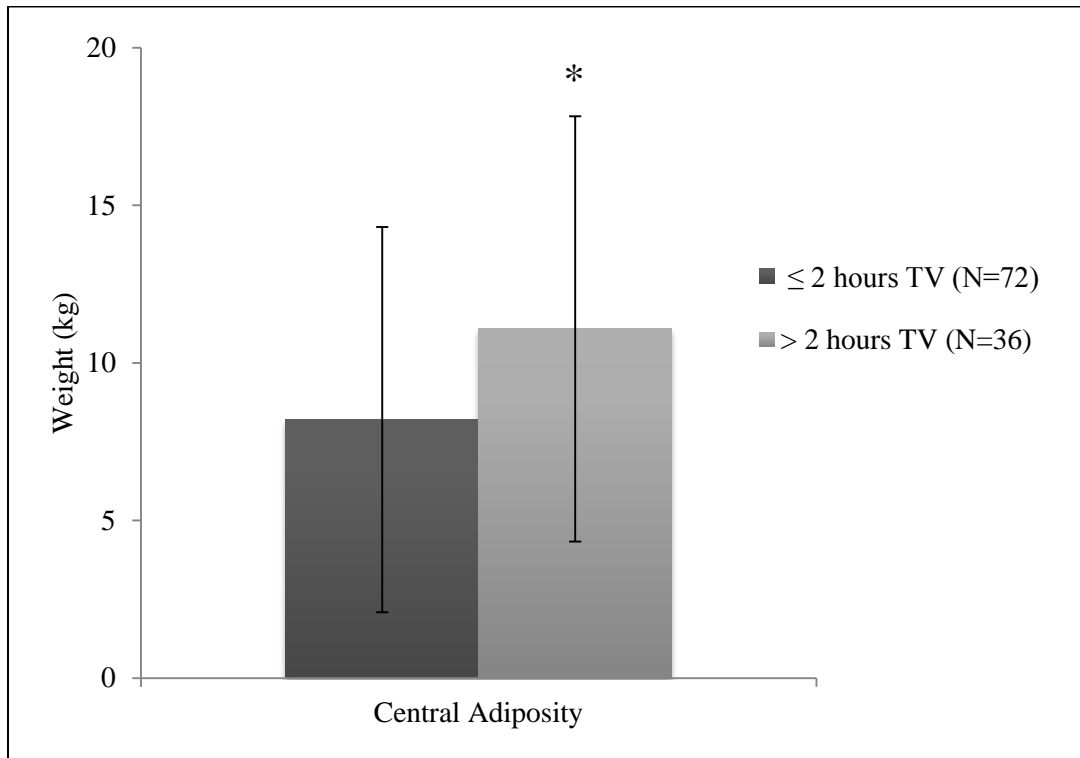
Table 8. TV viewing is not related to central adiposity among males (N=96), after controlling for fitness

	1	2	3	4	5	6
1. Central Adiposity	—					
2. Waist-to-height ratio	.78**	—				
3. Whole Body % Adiposity	.91**	.73**	—			
4. Body Mass Index	.80**	.78**	.68**	—		
5. Television/weekday	.07	-.04	-.02	.12	—	
6. Television/weekend day	.13	-.01	.10	.22	.60	—

**significant at the 0.01 level (one-tailed)

*significant at the 0.05 level (one-tailed)

Figure 8. Central adiposity is higher among females who watch more than 2 hours of TV/weekday



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA.

Participants separated based on the American Academy of Pediatrics recommendation to limit TV time to less than 2 hours per day (Strasburger, 2011)

Table 9. Diet intake reported by 24-hour recalls among females (N=71)

	Intake Reported	% of Energy	Recommendation
Energy (kcal)	1819 ± 560		1600-1700
Protein (g)	65.8 ± 21.9	14.9	20-30
Fat (g)	66.2 ± 26.4	32.3	25-35
Carbohydrate (g)	243.7 ± 85.4	52.7	45-65
Added Sugars (g)	70.6 ± 45.5	15.5	<25
Saturated Fatty Acids (g)	24.8 ± 11.6	12.2	<10
Cholesterol (mg)	197.6 ± 127.3		<300
Fiber (g)	13.6 ± 6.2		26
Sodium (mg)	2928 ± 1079		<1500
Potassium (mg)	2018 ± 700		4500mg
Calcium (mg)	948.3 ± 438.4		1300

All values presented as Mean ± SD

NDSR (2010) was used for all diet recall analyses

Table 10. Diet intake reported by 24-hour recalls among males (N=81)

	Intake Reported	% of Energy	Recommendation
Energy (kcal)	2001 ± 600		1700-1800
Protein	73.5 ± 24.2	15.5	20-30
Fat	71.8 ± 24.7	33.0	25-35
Carbohydrate	258.3 ± 85.3	51.5	45-65
Added Sugars	71.2 ± 42.7	14.2	<25
Saturated Fatty Acids	27.1 ± 12.3	12.2	<10
Cholesterol (mg)	211.2 ± 126.8		<300
Fiber (g)	14.4 ± 6.5		31
Sodium (mg)	3404 ± 1281		<1500
Potassium (mg)	2171 ± 863.8		4500mg
Calcium (mg)	1057 ± 502.4		1300

All values presented as Mean ± SD

NDSR (2010) was used for all diet recall analyses

Table 11. No difference in intake reported by 24-hour recall and 3-day food records (N=26)

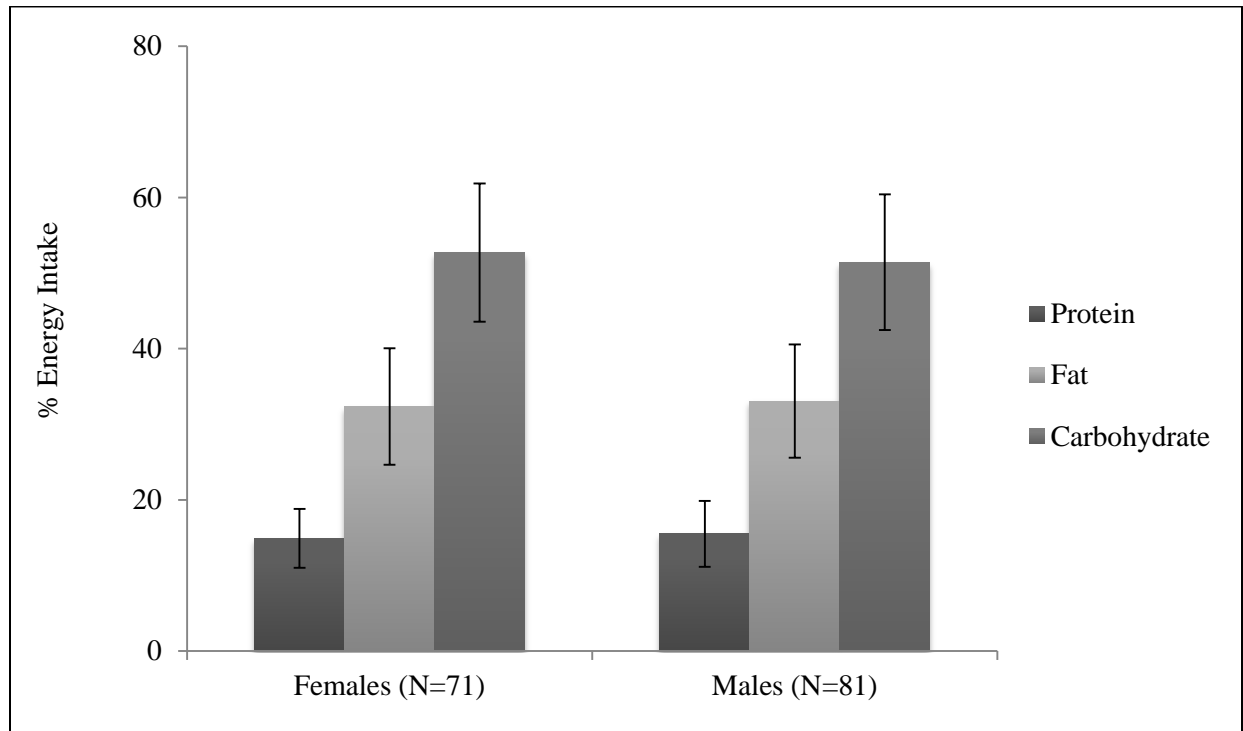
	Mean	P
Energy (kcal)	1928.19 ± 457.61 (recall) 1798.18 ± 372.84 (record)	0.28
Calories from Protein (%)	14.76 ± 2.94 15.29 ± 2.22	0.42
Calories from Fat (%)	32.93 ± 7.94 34.78 ± 5.64	0.24
Calories from Carbohydrate (%)	52.18 ± 8.57 49.94 ± 6.67	0.23
Cholesterol-Saturated Fatty Acid Index	36.40 ± 16.56 37.10 ± 1.46	0.84

All values presented as Mean ± SD

Differences between recalls and records were assessed using paired samples t-test

NDSR (2010) was used for all diet recall analyses

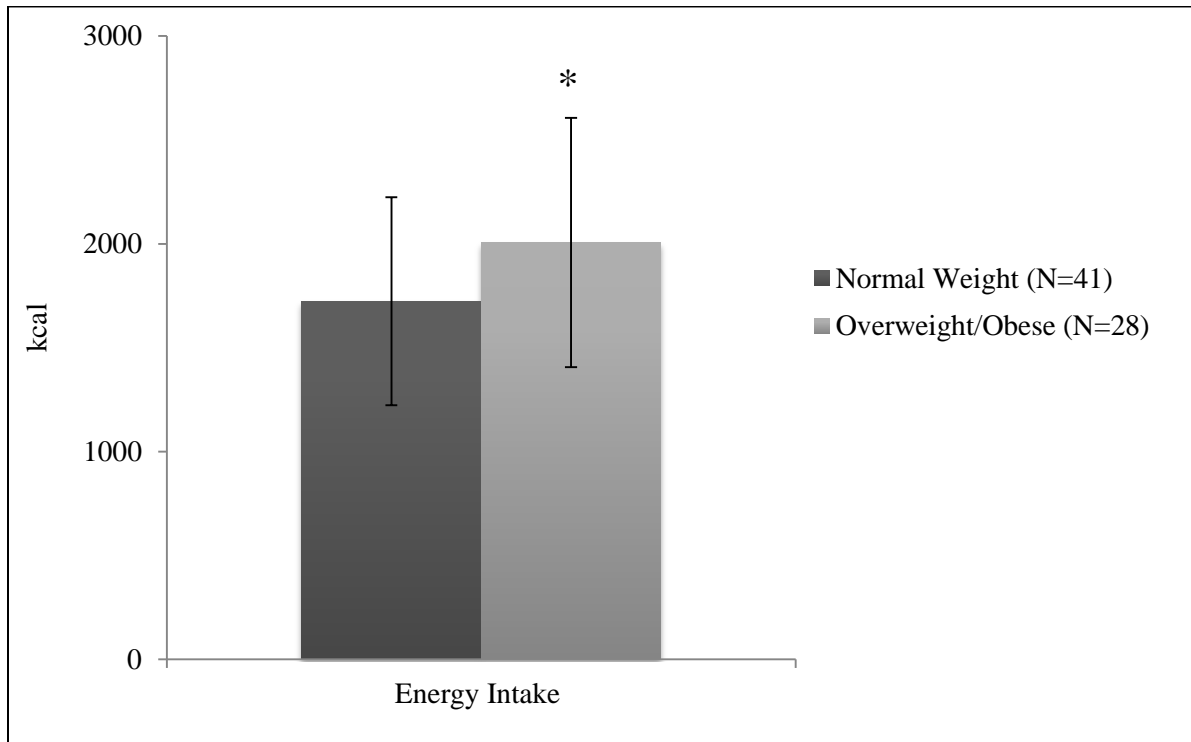
Figure 9. Females and males have similar macronutrient intake



All values presented as Mean \pm SD

Differences in groups assessed by one-way ANOVA

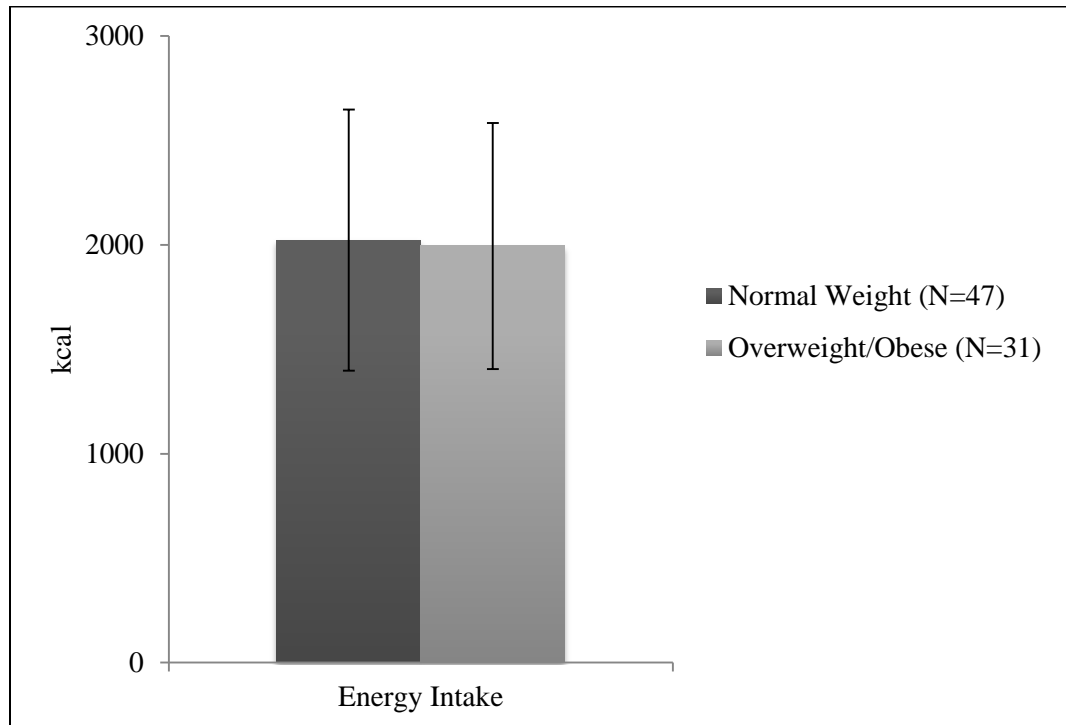
Figure 10. Overweight females have higher energy intake than normal weight females



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA. Participants were categorized using the Centers for Disease Control and Prevention growth charts (Kuczmarski et al., 2000).

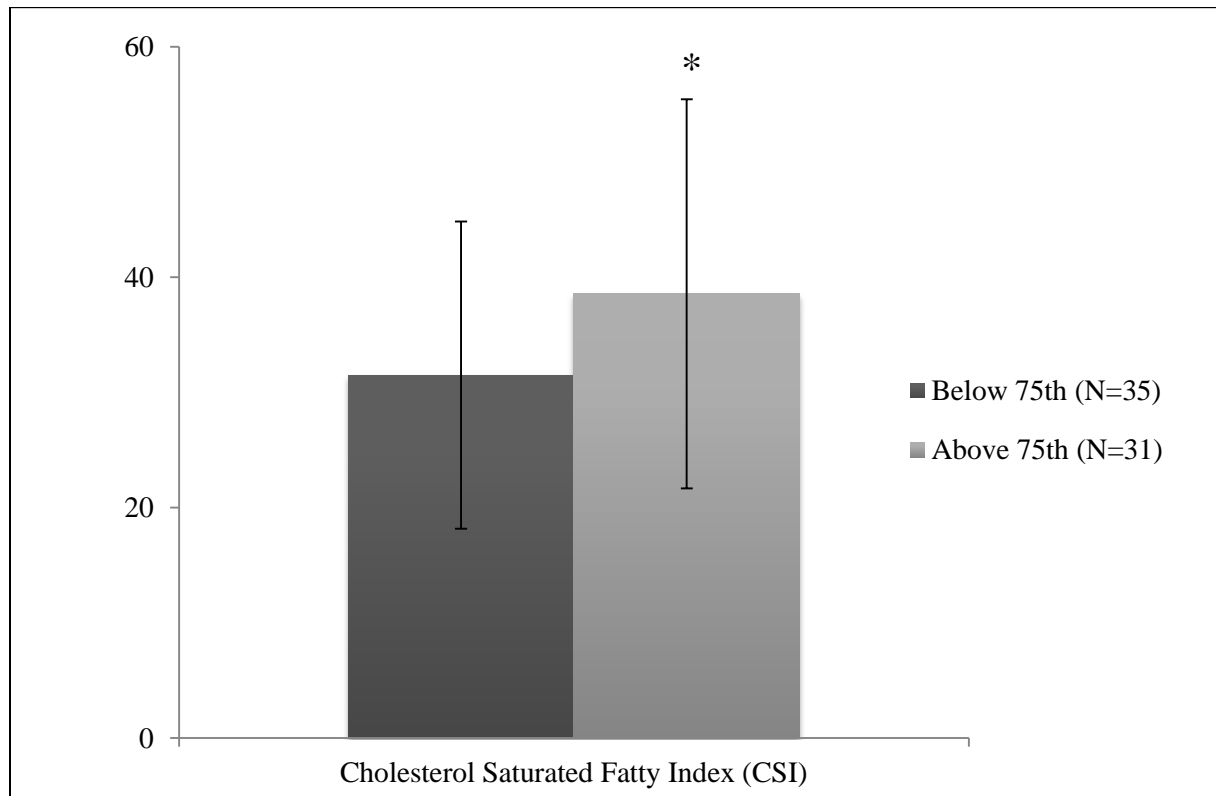
Figure 11. No differences in energy intake between normal weight and overweight males



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA. Participants were categorized using the Centers for Disease Control and Prevention growth charts (Kuczmarski et al., 2000).

Figure 12. Females above the waist circumference-for-age cutoff consume a diet with a higher cholesterol-saturated fatty acid index¹



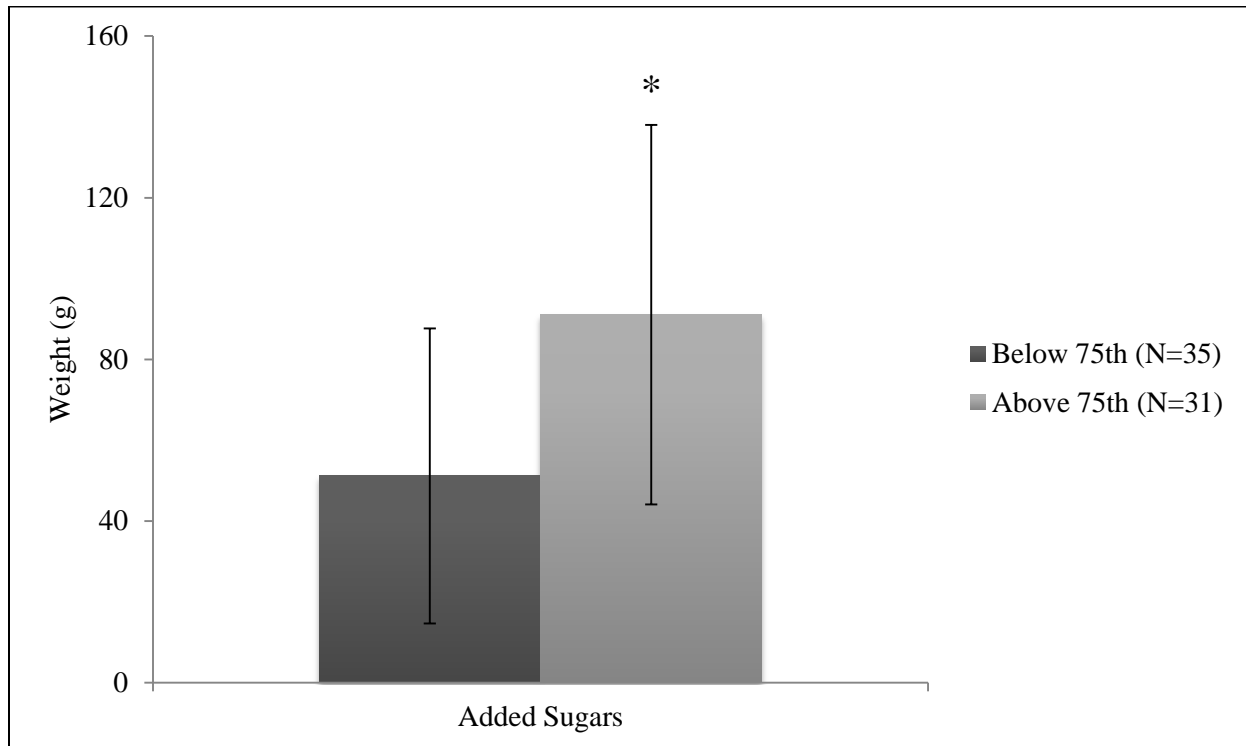
All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA.

¹Cholesterol-saturated fatty acid index (CSI) = $1.01 (\text{g SFA}) + .05 (\text{mg Cholesterol})$

Participants were categorized using waist circumference-for-age percentile references provided by Fernandez et al. (2004)

Figure 13. Females above the waist circumference-for-age cutoff consume higher added sugars¹



All values presented as Mean \pm SD

* Statistically significant difference between groups ($p < 0.05$) as determined by one-way ANOVA. Participants were categorized using waist circumference-for-age percentile references provided by Fernandez et al. (2004)

¹Foods classified as added sugars by NDSR (2010) include: white sugar (sucrose), brown sugar, powdered sugar, honey, molasses, pancake syrup, corn syrups, high fructose corn syrups, invert sugar, invert syrup, malt extract, malt syrup, fructose, glucose (dextrose), galactose, and lactose.

Table 12. Cholesterol-Saturated Fatty Index related to increased central adiposity among females (N=66), after controlling for fitness and energy intake

	1	2	3	4	5	6
1. Central Adiposity	—					
2. Whole Body %Adiposity	.87**	—				
3. Added Sugars	.11	-.03	—			
4. Cholesterol	.22*	.17	-.16	—		
5. Saturated Fatty Acids	.18	.13	.13*	.20*	—	
6. Cholesterol-Saturated Fatty Acid Index	.26*	.19	.01	.68**	.85**	—

**significant at the 0.01 level (one-tailed)

*significant at the 0.05 level (one-tailed)

Table 13. Cholesterol-Saturated Fatty Index not related to central adiposity among males (N=75), after controlling for fitness and energy intake

	1	2	3	4	5	6
1. Central Adiposity	—					
2. Whole Body % Adiposity	.92**	—				
3. Added Sugars	-.02	-.03	—			
4. Cholesterol	.12	.11	-.14	—		
5. Saturated Fatty Acids	.06	.10	-.25*	.37**	—	
6. Cholesterol Saturated Fat Index	.10	.12	-.25*	.72*	.92*	—

**significant at the 0.01 level (one-tailed)

*significant at the 0.05 level (one-tailed)

Table 14. Reliability analysis of nutrition beliefs questionnaire

Apple vs. Fat-free fruit snack vs. Regular fruit snack
Cottage cheese vs. Fat-free ice cream vs. Regular ice cream
Baked sweet potato vs. Baked potato chips vs. Potato fries
Baked chicken nuggets vs. Deli sliced chicken vs. Fried chicken nuggets

Cronbach alpha = .63

Table 15. Reliability analysis of diet vs. high-energy/low-nutrient subscale

Fat-free cookie or regular cookie
Regular coke or Diet coke
Fat-free- fruit snack or Regular fruit snack
Regular ice cream or Fat-free ice cream

Cronbach alpha = .86

The diet vs. high-energy/low-nutrient subscale was determined by conducting exploratory factor analysis. Two items were removed to achieve the highest possible internal consistency.

Table 16. Nutrition Belief Score related to increased vitamin D, calcium, and reduced calories from fat among females (N=40)

	1	2	3	4	5	6	7	8
1. Total Nutrition Belief Score	—							
2. Diet vs. high-energy/low-nutrient Subscale	.55**	—						
3. Vitamin D	.37*	.27*	—					
4. Total Fat	-.01	.15	-.06	—				
5. Total Saturated Fatty acids	.21	.23	-.05	.83**	—			
6. %Calories from Fat	-.31*	.06	-.28*	.61**	.41**	—		
7. %Calories from Saturated Fatty Acids	.18	.18	-.12	.51**	.81**	.57**	—	
8. Calcium	.42**	.20	.75**	.24	.30*	-.11	.23	—

**significant at the 0.01 level (one-tailed)

*significant at the 0.05 level (one-tailed)

Table 17. Diet vs. high-energy/low-nutrient subscale related to decreased intake of calories from total and saturated fat among males (N=39)

	1	2	3	4	5	6	7	8
1. Total Nutrition Beliefs Score	—							
2. Diet vs. high-energy/low-nutrient	.66**	—						
3. Vitamin D	-.09	-.11	—					
4. Total Fat	-.19	-.28*	.45**	—				
5. Total Saturated Fatty acids	-.08	-.22	.54**	.83**	—			
6. %Calories from Fat	-.13	-.35*	-.04	.42**	.35*	—		
7. %Calories from Saturated Fatty Acids	-.08	-.30*	.30*	.32*	.66**	.67**	—	
8. Calcium	-.08	-.07	.76**	.53**	.56**	.03	.30*	—

**significant at the .01 level (one-tailed)

*significant at the .05 level (one-tailed)

Table 18. Predictors of central adiposity among females

	β	P	Model R ²
Model 1: Pubertal staging	.482	.00	.225
Model 2: Pubertal staging	.468	.00	.220
Age	.047	.60	
Model 3: Pubertal staging	.319	.00	.443
Age	.060	.432	
Fitness	-.495	.00	
Model 4: Pubertal staging	.409	.00	.501
Age	.106	.262	
Fitness	-.402	.00	
Energy	.097	.297	
Model 5: Pubertal staging	.415	.00	.502
Age	.089	.365	
Fitness	-.408	.00	
Energy	.861	.393	
Added Sugars	-.018	.895	
Model 6: Pubertal staging	.418	.00	.533
Age	.050	.613	
Fitness	-.405	.00	
Energy	-.057	.727	
Added Sugars	-.018	.895	
CSI	.249	.056	

Table 19. Predictors of central adiposity among males

	β	P	Model R ²
Model 1: Pubertal staging	-.044	.652	.002
Model 2: Pubertal staging	-.092	.339	.073
Age	.271	.005	
Model 3: Pubertal staging	-.080	.348	.276
Age	.228	.009	
Fitness	-.453	.00	
Model 4: Pubertal staging	-.062	.538	.279
Age	.310	.003	
Fitness	-.413	.00	
Energy	.034	.734	
Model 5: Pubertal staging	-.064	.528	.279
Age	.304	.004	
Fitness	-.415	.00	
Energy	.038	.740	
Added Sugars	.001	.996	
Model 6: Pubertal staging	-.052	.623	.281
Age	.306	.005	
Fitness	-.418	.00	
Energy	-.009	.957	
Added Sugars	.004	.977	
CSI	.074	.601	

Table 20. Predictors of waist-to-height ratio among females

	β	P	Model R ²
Model 1: Pubertal staging	.459	.00	.211
Model 2: Pubertal staging	.482	.00	.216
Age	-.080	.385	
Model 3: Pubertal staging	.363	.00	.361
Age	-.069	.404	
Fitness	-.397	.00	
Model 4: Pubertal staging	.459	.00	.449
Age	-.099	.320	
Fitness	-.315	.002	
Energy	.156	.113	
Model 5: Pubertal staging	.477	.00	.478
Age	-.142	.159	
Fitness	-.323	.002	
Energy	.140	.311	
Added Sugars	.036	.800	
Model 6: Pubertal staging	.480	.00	.531
Age	-.183	.069	
Fitness	-.320	.001	
Energy	-.064	.694	
Added Sugars	.031	.822	
CSI	.312	.018	

Table 21. Predictors of waist-to-height ratio among males

	β	P	Model R ²
Model 1: Pubertal staging	-.116	.234	.013
Model 2: Pubertal staging	-.136	.169	.026
Age	.114	.248	
Model 3: Pubertal staging	-.126	.170	.167
Age	.078	.395	
Fitness	-.378	.00	
Model 4: Pubertal staging	-.060	.583	.143
Age	.103	.346	
Fitness	-.353	.002	
Energy	.078	.481	
Model 5: Pubertal staging	-.070	.526	.150
Age	.100	.365	
Fitness	-.358	.002	
Energy	.048	.698	
Added Sugars	.082	.501	
Model 6: Pubertal staging	-.118	.302	.167
Age	.137	.230	
Fitness	-.331	.005	
Energy	.022	.897	
Added Sugars	.123	.357	
CSI	-.097	.522	

Table 22. Predictors of whole body % adiposity among females

	β	P	Model R ²
Model 1: Pubertal staging	.365	<.001	.133
Model 2: Pubertal staging	.365	<.001	.133
Age	-.001	.991	
Model 3: Pubertal staging	.200	.018	.406
Age	.013	.871	
Fitness	-.547	<.001	
Model 4: Pubertal staging	.320	.004	.452
Age	.071	.472	
Fitness	-.460	<.001	
Energy	.088	.369	
Model 5: Pubertal staging	.378	<.001	.512
Age	.023	.807	
Fitness	-.447	<.001	
Energy	.150	.118	
Protein (% Energy)	.265	.007	
Model 6: Pubertal staging	.390	.001	.524
Age	.005	.961	
Fitness	-.441	<.001	
Energy	.118	.423	
Protein (%Energy)	.397	.587	
Fat (%Energy)	.396	.768	
CSI	.004	.982	

Table 23. Predictors of whole body % adiposity among males

	β	P	Model R ²
Model 1: Pubertal staging	-.135	.164	.009
Model 2: Pubertal staging	-.174	.072	.067
Age	.223	.022	
Model 3: Pubertal staging	-.161	.054	.313
Age	.176	.036	
Fitness	-.499	<.001	
Model 4: Pubertal staging	-.139	.149	.349
Age	.297	.002	
Fitness	-.474	<.001	
Energy	-.010	.917	
Model 5: Pubertal staging	-.155	.106	.370
Age	.307	.002	
Fitness	-.439	<.001	
Energy	-.051	.607	
Protein (% Energy)	-.154	.125	
Model 6: Pubertal staging	-.157	.116	.388
Age	.335	.001	
Fitness	-.427	<.001	
Energy	-.194	.213	
Protein (%Energy)	-.204	.064	
Fat (%Energy)	-.028	.809	
CSI	.169	.278	

DISCUSSION

Central adiposity has emerged as an important predictor for metabolic complications and adverse health effects (metabolic syndrome, type 2 diabetes, and CVD) among adults and adolescents (Bjorntorp, 1992; Carey et al., 1997). Among adults, the surrogate measure of waist circumference is a critical component of the National Cholesterol Education Program Adult Treatment Panel III and the International Diabetes Federation's criteria for diagnosis of metabolic syndrome (Goran and Gower, 1999; Esmailzadeh et al., 2006). Although, limited data among children has thus far prevented the establishment of anthropometric cutoffs for abdominal obesity, growing evidence suggests markedly different metabolic profiles can be seen between children above and below the waist circumference-for-age cutoff of 75th percentile (Savva, 2000) and waist-to-height ratio cutoff of 0.5 (Li, 2006). The present study aimed to determine diet and media use factors that are related to increased central adiposity in prepubertal children. Utilization of DXA allowed for assessment of central adiposity as a continuous variable. Additionally, we compared behavioral differences across waist circumference cutoffs that are related to CVD risk factors in children.

Secular trends in activity-related behaviors suggest that 8-18-year-olds spend more time with media (an average of 7.5 hours/day) than any other sedentary activity, besides sleep (Henry J. Kaiser Family Foundation, 2010). Although the use of mobile devices (e.g. cell phones, handheld videogames, mp3 players) has been on the rise, TV viewing is the most widely studied media use variable. Several studies have examined the effects of TV on weight status in children. In a large longitudinal study of 8000 Scottish children, watching more than 8 hours of TV/week at age 3 was related to obesity at age 7 (Reilly, 2005). In another study, each

additional hour of TV viewing at age 5 increased the risk of being obese in adulthood by 7% (Viner, 2005). Findings from several other studies implicate increased TV viewing in obesity development (Jordan, 2007; Dennison, 2008). However, the present study was unique in three ways: 1) control for cardiorespiratory fitness; 2) control for energy intake; and 3) utilization of direct and anthropometric measures of whole body and regional adiposity. Cardiorespiratory fitness and energy intake have both been independently implicated in development of obesity (Blair, 2004; Rodriguez, 2010)

After controlling for fitness and energy intake, our data showed that weekend TV viewing was related to increased waist-to-height ratio and BMI among females. Furthermore, females who watched more than 2 hours of TV/weekday had significantly higher central adiposity compared to females who watched less than 2 hours of TV/weekday. Among males, weekday TV time was related to increased BMI whereas weekly videogame use and weekend screen-time were related to increased whole body %adiposity. Utilizing whole body measures of adiposity (whole body %adiposity and BMI) as well as fat distribution (central adiposity and waist-to-height- ratio) allowed us to identify which measures were more strongly associated with specific media use components. However, it should be noted that relationships between media use and central adiposity, the primary outcome of this study, were only significant for females and not males. Nevertheless, this was the first study to assess media use among preadolescents and relate it to central adiposity as defined by DXA.

Although some studies have examined the effect of dietary intake in the development of central adiposity, evidence in children remains inadequate. Among adults, higher intake of vegetables, dairy, whole grains and lower intake of processed red meat and soda was related to smaller gains in BMI and waist circumference (Newby et al., 2003). Among a large NHANES

sample of 5-11-year-olds, Bradlee et al. (2009) showed that intakes of dairy and whole grains were inversely related to adiposity. Energy intake and energy from fat have also been shown to be positively related to whole body adiposity (Tucker, 1997). However, the aforementioned studies used surrogate measures of whole body adiposity and fat distribution. To our knowledge, the present study is the first to examine relationships between nutrient intakes and central adiposity measured by DXA among preadolescents. Our finding that added sugars were related to central adiposity may be consistent with previous studies implicating sugar sweetened beverages in childhood obesity (Ludwig, 2001; Berkey, 2004). Additionally, added sugar intake was higher among females who had a waist circumference-for-age above the 75th percentile. However, similar to Berkey et al (2004), the influence of added sugars in our data was attenuated once total energy was controlled for. Although energy intake was related to central adiposity, our results differed from Tucker et al. (1997) since calories from fat were not associated with obesity. Rather, saturated fatty acids and CSI were related to central adiposity even after controlling for fitness and energy intake. These findings may be consistent with Newby et al. (2003) given that processed red meats are often high in cholesterol and saturated fatty acids. However, our diet analyses were limited to the nutrient level and we cannot draw conclusions on food group associations with adiposity. It is noteworthy, however, that we observed differences in diet intake based on waist circumference-for-age cutoffs, similar to findings by Bradlee and colleagues. Future research is needed to further assess the efficacy of using waist circumference cutoffs to assess differences in behavioral factors related to obesity in children.

Several studies among children and teenagers have shown that TV viewing is related to increased intake of energy, fat, and sodas and lower intake of fruits and vegetables (Wechsler, 2003; Wiecha, 2006; Miller, 2008). However, fewer studies have assessed the mediating factors

in the media-diet relationship. In our study nutrition belief scores were inversely related to total screen time and positively related to vitamin D and calcium intake among females. Although the diet vs. high-energy/low-nutrient subscale was not related to screen time, scores among males were inversely related to energy from fats and saturated fats. It appears that nutrition belief scores were influenced by overall media use among females, such that higher scores were associated with dietary intake of nutrients that have known positive associations with healthy growth outcomes in children. These findings suggest that media use may play a role in what foods children think are healthy for them. However, we are unable to implicate any particular medium (e.g. TV or computer use) in nutrition beliefs since we did not assess the specific content that children were viewing. Given that children are exposed to a plethora of food advertisements, majority of which are for foods of low nutritional quality (Gantz, 2007), it is important that future studies examine how media exposure affects children's nutrition beliefs and subsequent diet choices.

Although associations between dieting behaviors and media use have been the subject of research for several years. Recent evidence suggests that dieting behaviors are more prevalent among overweight and obese children compared to their normal weight counterparts (Neumark-Sztainer, 2002). Behaviors such as skipping meals and subsequent binge eating may have the counterproductive effect of weight gain among children attempting to lose weight (Field, 2003; Neumark Sztainer, 2007). In our smallest subgroup of participants, dieting behaviors were related to increased central adiposity, waist-to-height ratio, whole body %adiposity, and BMI. Examining diet data showed that increasing scores on the dieting subscale were related to increased carbohydrate intake and decreased intake of cholesterol. However, the small sample size and inability to separate the groups by gender limits the conclusions that can be drawn from

these correlations. Nevertheless, future studies are needed to examine the directionality of the obesity-dieting relationship and account for the mediating variables which may include but are not limited to media use.

There is a dearth of information on dietary factors related to central adiposity in prepubertal children. Berkey and coworkers (2004) developed separate models for females and males to predict gains in BMI using different beverages (milk, sugar-sweetened, diet soda, and fruit juices). However, none of the beverages significantly improved the model after adjusting for energy intake. Modeling by Tucker et al. (1997) predicted whole body %adiposity (measured by skinfold thickness) using macronutrient intake, total energy and fiber. Although Tucker and colleagues controlled for fitness, they did not assess effects on fat distribution or central adiposity. Regression models developed from this study are the first in the literature to predict central adiposity using specific nutrients while controlling for the effects of fitness and energy intake. Although pubertal staging and fitness were the main contributors to central adiposity measured by DXA and waist-to-height ratio, dietary variables including energy, added sugars, and CSI explained an additional 10% of the variance among females. The same diet variables explained an additional 17% of the variability of waist-to-height ratio among females. We developed models to predict waist-to-height ratio because it is an anthropometric measure with potential for wide scale application. Waist-to-height ratio also adjusts for participant height and may provide a more clinically relevant outcome compared to a weight measure of central adiposity measured by DXA. Fitness was the strongest predictor of whole body %adiposity followed by pubertal staging. The finding that energy from protein was related to increased whole body %adiposity is consistent with Tucker and co-workers (1997). However, the addition of energy from fat to the model attenuates the effect on energy from protein suggesting

mediating effects of fat intake. Although media use, weekend TV time in particular, appeared to be related to central adiposity, this relationship attenuated after the addition of energy intake to the models. Our data is consistent with other studies that suggest that the media-obesity relationship is mediated by energy intake (Zimmerman, 2010). The lack of relationships between behavioral factors and adiposity in males is worth noting. In a large population-based sample of 9-10-year-olds (N=1700), Jennings and colleagues adjusted for gender, energy density, and physical activity and observed an inverse relationship between overall diet quality scores and BMI Z-score, waist-to-height ratio, and waist circumference. Therefore, it is likely that diet has an influence on anthropometric measures of central adiposity among males as well.

Given that our results displayed a significant contribution of pubertal staging among females and age among males, it is important to understand our findings in the context of typical growth prior to and after the onset of puberty. Early onset of puberty among both males and females has been previously linked to childhood obesity (Davison, 2003; Ahmed, 2009). However, research from the Fels Longitudinal Study suggests that early menarcheal timing (before 11.9 years) is related to greater cardiovascular risk factors (elevated blood pressure and glucose intolerance) independent of body composition (Remsberg, 2005). Rico and colleagues assessed cross-sectional differences in body composition between preadolescents and adolescents using DXA and Tanner stage methodology (Rico, 1993). Stage 4 and 5 children (females and males) had significantly higher fat and fat free mass compared to children in stages 1 and 2. However, females had higher fat mass in stages 4 and 5 compared to males. A similar cross-sectional study showed that females had significantly higher whole body %adiposity than males at all stages of development (Boot et al., 1997). Boot and colleagues also found that, among males, whole body %adiposity was highest in stage 3 with subsequent decreases after the

onset of puberty in stages 4 and 5. Although Tanner staging appeared to be related to body composition in both studies, some key differences are worth noting. Rico et al. (1993) did not observe gender differences in whole body %adiposity at stages 1 and 2 while Boot and coworkers (1997) showed sex differences across all Tanner stages. Furthermore, while Rico et al. (1993) observed increased fat mass between later and earlier stages of development among both genders, Boot and colleagues (1997) found that stage 4 and 5 males had lower fat mass and whole body %adiposity than those in stage 3. Our study results are consistent with those from Boot and colleagues (1997), since we observed significant differences in both total fat mass and whole body %adiposity between females and males in Tanner stages 1 and 2. It appears that studies are consistent in their findings for post pubertal somatic changes in children. However, additional research is needed among prepubertal children since results have been equivocal thus far. Collectively, outcomes from multiple body composition studies suggest that males continue to accrue fat free mass whereas females increase in both fat free and fat mass as they go through puberty. The additional impact of specific diet and activity-related behaviors on excess adiposity during this transitional phase of development needs to be further assessed.

CONCLUSIONS & FUTURE DIRECTIONS

Although growth occurs throughout childhood, preadolescence represents an important transition phase from childhood to adolescence and is characterized by significant body compositional changes (Boot et al., 1997; Maggs et al., 1997). However, preadolescents are an understudied population group and there is limited research on behavioral factors related to excess fat accumulation during this critical period of development. Given its causal link to insulin resistance, it is concerning that much of the childhood obesity literature has failed to assess outcomes related to central adiposity. Therefore, we sought to assess the independent effects of media use and nutrient intake on central adiposity among preadolescent children. Previous studies have varied in their assessment of obesity and behaviors related to energy balance. Our study utilized high quality measures to assess cardiorespiratory fitness and measures of whole body and central adiposity. Furthermore, we assessed the key behavioral factors of diet and media use that have been previously shown to contribute to excess body weight. **Figure 14** illustrates the observed relationships between the main outcomes of our study.

It is important to recognize the role of gender in our findings. Initial body composition analyses revealed that females and males differed across all measures of adiposity, including the primary outcome of central adiposity. Therefore, separate conclusions were drawn based on gender groups. The effects of media use appear to differ by medium and day type. After controlling for fitness, weekend TV and video game use were related to measures of whole body, but not central adiposity. Additionally, weekend TV time appeared to influence energy intake, cholesterol and added sugars, however, these relationships attenuated once energy intake was

controlled for. Our overall media findings suggest that the influence of TV time on central adiposity is not independent and may be mediated by fitness and diet intake among children. Furthermore, the relationship between diet and TV time was independent of total nutrition belief scores. This finding was in contrast to our expectations as suggested in **Figure 1**. However, nutrition beliefs appeared to mediate the interaction between diet and central adiposity. Future research needs to consider non media factors (e.g. parental and peer influencers) on diet intake. Nutrient analyses implicated intake of energy, added sugars, and cholesterol-saturated fatty acid index as an important predictors of central adiposity. After controlling for fitness and energy intake, the influence of these nutrients was non-significant among males. Collectively, these results suggest that dietary and sedentary behaviors may have differential health-related outcomes for prepubertal children based on gender. These findings have implications for nutrition education or intervention efforts that may need to be modified to account for age and pubertal staging when targeting obesity prevention during the critical period of preadolescence.

Limitations

There were several limitations to this study. These results were based on a secondary data analysis and participants were recruited for a physical activity trial and not a body composition study. Therefore, the sample selected may not accurately represent the general population of preadolescents. The use of a single day's dietary recall may limit the precision of estimated dietary intakes of individuals. Additionally, there is the possibility that children may have underreported their dietary intake. As with any cross-sectional analysis, cause and effect cannot be determined and assumptions were made regarding directionality of relationships.

Future Directions

Although our study was limited by its cross-sectional design, we were able to draw important behavioral and development associations with central adiposity for future research. Longitudinal studies are needed to assess changes in central adiposity vs. whole body adiposity during the transitional phase from preadolescence to adolescence. Given that nutrients are scarcely consumed in isolation, the influence of food groups and diet patterns on central adiposity needs to be determined. Although we found significant associations between nutrition beliefs and diet intake, the moderate internal consistency of the questionnaire suggests need for modification and further testing. Finally, studies with different dietary and sedentary activity intervention targets for preadolescent females and males will greatly improve our understanding of obesity in this age group.

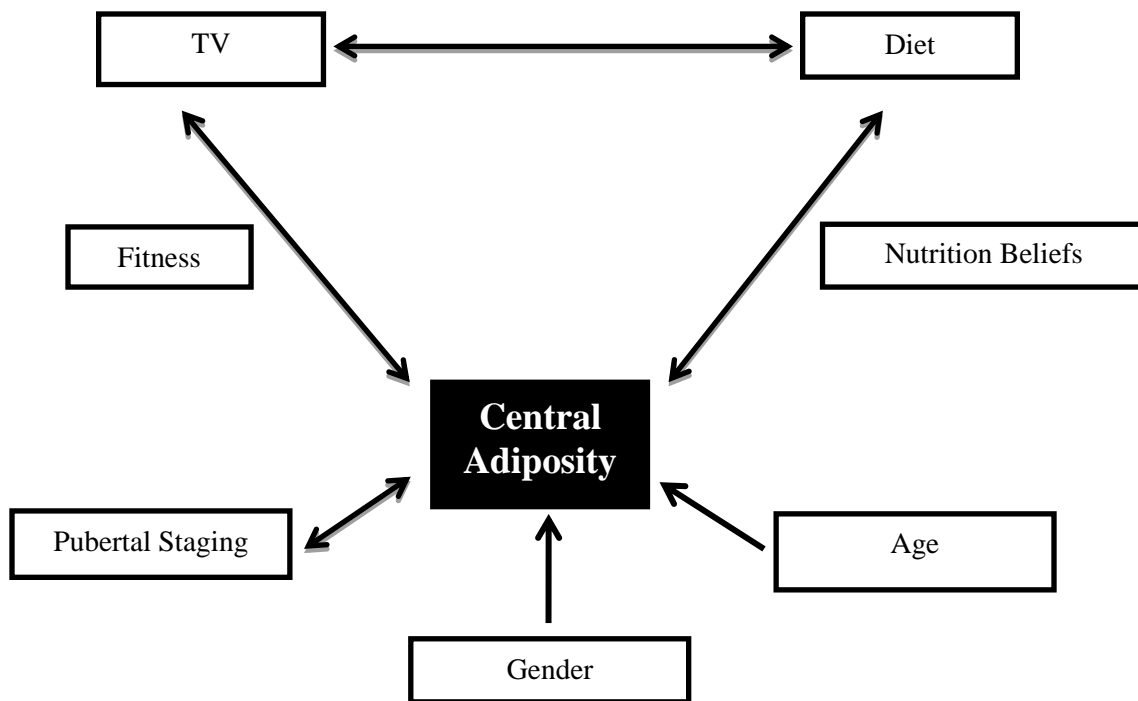


Figure 14. Summary of findings demonstrating interactions between media, diet, and central adiposity

APPENDICES

APPENDIX A

PARTICIPANT DEMOGRAPHICS QUESTIONNAIRE

General Information

1. What was your child's date of birth? _____ / _____ / _____
2. Was your child born before 38 weeks of pregnancy? Yes No
If yes, how early was your child born? _____
3. What was your child's birth weight? _____ lbs _____ oz
4. Did the mother of your child suffer from any medical condition while she was pregnant?
Yes No If yes, what condition?
5. What is your child's current age? _____
6. What is your child's current (or recently completed) Grade Level? _____
7. What is your child's sex? Male Female
8. Which is your child's dominant hand? Right Left No Preference
9. Is your child color blind? Yes No
10. Does your child wear contacts or glasses? Yes No
If yes, what was their prescription for?

Demographics

1. Does your child live with their biological parents? Yes No
2. Does your child live in a single parent/guardian household? Yes No
3. Does your child live with their Mother or a Female guardian? Yes No
4. Does your child's Mother/Female guardian work? Yes No
5. What is the highest level of education obtained by your child's Mother/Female guardian?
 - a) Did not complete high school
 - b) High School Graduate
 - c) Some College
 - d) Bachelor Degree
 - e) Advanced Degree
6. Does your child live with their Father or a Male guardian? Yes No
7. Does your child's Father/Male guardian work? Yes No
8. What is the highest level of education obtained by your child's Father/Male guardian?
 - a) Did not complete high school
 - b) High School Graduate
 - c) Some College
 - d) Bachelor Degree
 - e) Advanced Degree

9. How many other children (under the age of 18) live with your child? _____
 How old are they? _____
 What is their sex? _____
10. How many biological siblings does your child have? _____
11. Does your child receive free or reduced-price school lunch? Yes No
12. Do you consider yourself to be Hispanic or Latino (*A person of Mexican, Puerto Rican, Cuban, South or Central American, or other Spanish culture or origin, regardless of race*)?
 Yes No
13. What race / ethnicity do you consider your child?
- | | |
|-------------------------------------|--|
| a) American Indian or Alaska Native | d) Native Hawaiian or other Pacific Islander |
| b) Asian | e) White or Caucasian |
| c) Black or African American | f) Mixed or Other |
14. What is your approximate household income?
- | | |
|------------------|-------------------|
| a) <10,000 | g) 61,000-70,000 |
| b) 10,000-20,000 | h) 71,000-80,000 |
| c) 21,000-30,000 | i) 81,000-90,000 |
| d) 31,000-40,000 | j) 91,000-100,000 |
| e) 41,000-50,000 | k) 100,000+ |
| f) 51,000-60,000 | |

Activities

1. Does your child participate in musical activities? Yes No
 If yes:
 Does your child play an instrument? Yes No
 If so, what instrument(s)?
 Does your child participate in choir? Yes No
 How many hours a week does your child spend participating in musical activities?
2. Does your child participate in religious activities? Yes No
 If yes, how many hours a week does your child spend participating in religious activities?
3. Does your child participate in sports activities? Yes No
 If yes:
 Does your child participate in formal youth sports? Yes No

In what activities does your child participate?

4. Has your child attended regular afterschool care outside of your home in the last year?

Yes No

Habits

1. How much time does your child spend watching television on an average day during the week?

- | | |
|-------------------------|-------------------------|
| a) < 1 Hour per Day | f) 5 to 6 Hours per Day |
| b) 1 to 2 Hours per Day | g) 6 to 7 Hours per Day |
| c) 2 to 3 Hours per Day | h) 7 to 8 Hours per day |
| d) 3 to 4 Hours per Day | i) > 8 Hours per Day |
| e) 4 to 5 Hours per Day | |

2. How much time does your child spend watching television on an average day during the weekend?

- | | |
|-------------------------|-------------------------|
| a) < 1 Hour per Day | f) 5 to 6 Hours per Day |
| b) 1 to 2 Hours per Day | g) 6 to 7 Hours per Day |
| c) 2 to 3 Hours per Day | h) 7 to 8 Hours per day |
| d) 3 to 4 Hours per Day | i) > 8 Hours per Day |
| e) 4 to 5 Hours per Day | |

3. How much time does your child spend on a computer on an average day during the week?

- | | |
|-------------------------|-------------------------|
| a) < 1 Hour per Day | f) 5 to 6 Hours per Day |
| b) 1 to 2 Hours per Day | g) 6 to 7 Hours per Day |
| c) 2 to 3 Hours per Day | h) 7 to 8 Hours per day |
| d) 3 to 4 Hours per Day | i) > 8 Hours per Day |
| e) 4 to 5 Hours per Day | |

4. How much time does your child spend on a computer on an average day during the weekend?

- | | |
|-------------------------|-------------------------|
| a) < 1 Hour per Day | f) 5 to 6 Hours per Day |
| b) 1 to 2 Hours per Day | g) 6 to 7 Hours per Day |
| c) 2 to 3 Hours per Day | h) 7 to 8 Hours per day |
| d) 3 to 4 Hours per Day | i) > 8 Hours per Day |
| e) 4 to 5 Hours per Day | |

5. How much time does your child spend playing video games on an average during the week?
- a) < 1 Hour per Day
 - b) 1 to 2 Hours per Day
 - c) 2 to 3 Hours per Day
 - d) 3 to 4 Hours per Day
 - e) 4 to 5 Hours per Day
 - f) 5 to 6 Hours per Day
 - g) 6 to 7 Hours per Day
 - h) 7 to 8 Hours per day
 - i) > 8 Hours per Day
6. How much time does your child spend playing video games on an average during the weekend?
- a) < 1 Hour per Day
 - b) 1 to 2 Hours per Day
 - c) 2 to 3 Hours per Day
 - d) 3 to 4 Hours per Day
 - e) 4 to 5 Hours per Day
 - f) 5 to 6 Hours per Day
 - g) 6 to 7 Hours per Day
 - h) 7 to 8 Hours per day
 - i) > 8 Hours per Day
7. How much time does your child spend being physically active on an average during the week?
- a) < 1 Hour per Day
 - b) 1 to 2 Hours per Day
 - c) 2 to 3 Hours per Day
 - d) 3 to 4 Hours per Day
 - e) 4 to 5 Hours per Day
 - f) 5 to 6 Hours per Day
 - g) 6 to 7 Hours per Day
 - h) 7 to 8 Hours per day
 - i) > 8 Hours per Day
8. How does your child travel to school?
- a. Walking
 - b. Bicycle
 - c. Public Transport
 - d. Bus
 - e. Car
9. How much time does your child spend being physically active on an average during the weekend?
- a) < 1 Hour per Day
 - b) 1 to 2 Hours per Day
 - c) 2 to 3 Hours per Day
 - d) 3 to 4 Hours per Day
 - e) 4 to 5 Hours per Day
 - f) 5 to 6 Hours per Day
 - g) 6 to 7 Hours per Day
 - h) 7 to 8 Hours per day
 - i) > 8 Hours per Day

10. How much sleep does your child regularly get?

- a) < 5 Hours per Day
- b) 5 to 6 Hours per Day
- c) 6 to 7 Hours per Day
- d) 7 to 8 Hours per Day
- e) 8 to 9 Hours per Day
- f) 9 to 10 Hours per Day
- g) > 10 Hours per Day

11. How much sleep did your child get last night?

- a) < 5 Hours
- b) 5 to 6 Hours
- c) 6 to 7 Hours
- d) 7 to 8 Hours
- e) 8 to 9 Hours
- f) 9 to 10 Hours
- g) > 10 Hours

12. Does your child have any of the following in his/her bedroom

- a. TV
- b. Computer
- c. Gaming console

13. How many caffeinated soft drinks does your child regularly drink in a day?

- None One Two Three or more

14. How many cups of tea does your child regularly drink in a day?

- None One Two Three or more

15. How often would you rate your child's stress level as HIGH?

- Occasionally Frequently Constantly

When was the last time your child:

Had a caffeinated substance?

Ate a meal or a snack?

What did s/he have to eat?

Exercised?

What type of exercise?

How long did s/he exercise for?

How intense did s/he work-out?

General Health

1. When was the last time your child saw a doctor? _____
2. Does your child have any allergies? Yes No
3. Has your child ever been diagnosed with dyslexia? Yes No

4. Has your child ever been diagnosed with an attentional disorder? Yes No

5. Has your child ever been diagnosed with asthma? Yes No

6. Is your child epileptic? Yes No

7. Is your child diabetic? Yes No

If so please explain:

8. Has your child been diagnosed with any kind of cancer? Yes No

If so please explain:

9. Does your child have hearing loss or wear a hearing aid? Yes No

10. Has your child been hospitalized within the last 6 months? Yes No

If so please explain:

11. Has your child ever lost consciousness as a result of hitting their head? Yes No

If yes:

When did this occur?

Where did s/he hit his/her head?

How long was s/he unconscious?

12. Has your child ever lost consciousness as a result of any other type of injury or seizure?

Yes No

If yes:

When did this occur?

How long was s/he unconscious?

Medications/Supplements

Medications: Is your child presently taking or have they taken any of the following medications within the past two months? Please circle your answer.

Asprin, Bufferin, Anacin

Blood Pressure pills

Cortisone

Cough Medicine

Digitalis

Hormones

Insulin or Diabetic pills

Tranquilizers

Weight reducing pills

Blood thinning pills

Dilantin

Allergy Shots

Water pills

Antibiotics

Iron or poor blood medications

Barbiturates

Laxatives

Phenobarbital

Sleeping pills

Thyroid medicine

Other(s): _____

1. Does your child take Ginkgo Biloba supplements? Yes No

If yes:

When was the last time they took the supplement?

What dose of the supplement did they take?

2. Does your child take Iron supplements? Yes No

If yes:

When was the last time they took the supplement?

What dose of the supplement did they take?

3. Does your child take any stimulants or sedatives? Yes No

If yes:

What do they take?

When was the last time they took it?

What dose of it did they take?

Cardiovascular Health

Does your child have any of the following:

1. Yes No Pain or discomfort in the chest, neck, jaw, arms, or other areas that may be related to poor circulation.
2. Yes No Heartbeats or palpitations that feel more frequent or forceful than usual or feeling that your heart is beating very rapidly.
3. Yes No Unusual dizziness or fainting.
4. Yes No Shortness of breath while lying flat or a sudden difficulty in breathing that wakes them up while sleeping.
5. Yes No Shortness of breath at rest or with mild exertion (such as walking two blocks).
6. Yes No Feeling lame or pain in the legs brought on by walking.
7. Yes No A known heart murmur.

8.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Unusual fatigue with usual activities.
9.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Has any male in your immediate family had a heart attack or sudden death before the age of 55?
10.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Has any female in your immediate family had a heart attack or sudden death before the age of 65?
11.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Do you have family history of heart disease?
12.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Do you have family history of lung disease?
13.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Do you have family history of diabetes?
14.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Do you have family history of strokes?
15.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Has your child been diagnosed with a past or present cardiovascular disease?
16.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Does your child have any significant heart rhythm disorder?
17.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Has your child been diagnosed with hypertension?
18.	<input type="checkbox"/> Yes <input type="checkbox"/> No	Has your child been diagnosed with peripheral vascular disease?
<i>Other</i>		
Is there anything else you feel we should know about your child's current/past health?		

APPENDIX B
TWO-DAY MEDIA LOG

PLEASE FILL OUT THIS DIARY FOR ONE WEEKDAY AND ONE WEEKEND DAY

This diary will record your activity for two days (one weekday and one weekend day). Each section in the diary contains an Activities Grid and one end of day question. Please fill in the Activities Grid throughout the day. At the end of the day, before you go to sleep, please answer the question at the end of the section and make sure the Activities Grid is complete.

START DATE:

Before you begin to use the Activities Grid, please fill in the date you are beginning your diary on the next page.

ACTIVITIES GRID:

The Activities Grid is designed to help you keep track of the different kinds of media you have used through out the day. Each column is for a different half hour period of the day. There are three different questions.

1. WERE YOU DOING ANY MEDIA ACTIVITIES FOR AT LEAST 15 MINUTES?

(CIRCLE "YES" OR "NO". PLEASE DO NOT LEAVE BLANK)

YES – Answer the questions below.

NO – Go to the next time slot.

2. WHAT WAS YOUR MAIN MEDIA ACTIVITY? (CIRCLE ONLY

- | | |
|---|-------------------------------|
| 1. Listening to music | 7. Playing computer games |
| 2. Watching TV | 8. Doing homework on computer |
| 3. Watching Videotapes/DVDs | 9. Instant messaging |
| 4. Watching a movie (in theater) | 10. Emailing |
| 5. Reading for fun (books, magazines, etc) | 11. Visiting websites |
| 6. Playing video games (handheld or player) | 12. Other computer activities |

ONE ANSWER)

Please circle the number that matches the one media activity that you were paying most attention to. Then answer the next question about the media activity you circled.

3. WHAT ELSE WERE YOU DOING? (CIRCLE AS MANY ANSWERS AS YOU NEED)

- | | |
|---------------------------------|---|
| 1. Nothing else | 9. Reading |
| 2. Chores | 10. Playing video games |
| 3. Eating | 11. Playing computer games |
| 4. Talking on the phone | 12. Instant messaging |
| 5. Homework (not on computer) | 13. Emailing |
| 6. Homework (on computer) | 14. Visiting websites |
| 7. Listening to music | 15. Other computer activities |
| 8. Watching TV, videos, or DVDs | 16. Something else (write in activity: _____) |

Please circle the number or numbers that match the other things you were doing when you were reading, listening to music, playing a videogame, watching TV or a movie, or using the computer. If you were doing "Something else", please write in your answer.

4. WHERE WERE YOU? (CIRCLE ONLY ONE ANSWER)

- | | |
|-------------------------|------------------------|
| 1. My bedroom | 5. Before/after school |
| 2. Another room at home | 6. Car or bus or train |
| 3. A friend's home | 7. Someplace else |
| 4. School | |

Please circle the number that matches the place where you were when you were doing this activity.

5. WHO WAS WITH YOU? (CIRCLE AS MANY ANSWERS AS YOU NEED)

- | | |
|----------------------|--------------------|
| 1. I was mainly home | 5. Sitter or nanny |
| 2. Mother or father | 6. Grandparent |
| 3. Brother or sister | 7. Teacher |
| 4. Friend | 8. Someone else |

Please circle the number or numbers that match the people that were with you when you were doing the activity.

THANK YOU FOR HELPING US WITH THIS IMPORTANT PROJECT!

PLEASE RETURN THIS DIARY TO A FITKIDS STAFF MEMBER

DAY OF THE WEEK
(CIRCLE ONE ANSWER ONLY)

- SUNDAY..... 1
- MONDAY..... 2
- TUESDAY..... 3
- WEDNESDAY..... 4
- THURSDAY..... 5
- FRIDAY..... 6
- SATURDAY..... 7

TODAY'S DATE
(WRITE ANSWER BELOW)

_____ MONTH _____ DAY _____ YEAR

TODAY, HOW MUCH TIME DID YOU SPEND DOING THE FOLLOWING ACTIVITIES?
(CIRCLE ONE ANSWER NEXT TO EACH STATEMENT – A THROUGH F)

	None	30 minutes or less	1 hour	2 hours	3 hours	4 hours or more
A. Being in school	0	1	2	3	4	5
B. Working at a job	0	1	2	3	4	5
C. Doing chores	0	1	2	3	4	5
D. Doing homework	0	1	2	3	4	5
E. Participating in a sports team, hobby, or club	0	1	2	3	4	5
F. Being in a Child-Care or Before/After School Program	0	1	2	3	4	5

(Henry J. Kaiser Family Foundation, 2010)

APPENDIX D

3-DAY FOOD RECORD

Instructions for Completing the 3-Day Food Record

1. Record everything your child ate or drank (including supplements) during the 24-hour time period indicated (12:01 a.m. to midnight). Repeat this for a total of 3 days (2 week days, 1 weekend day).
2. To the best of your ability, describe combination or mixed dishes that were eaten. For example, what ingredients were included on that piece of pizza? Was it thick or thin crust? Include brand names if known.
3. Describe the amounts consumed in terms appropriate for that item. For example: ounces (cups) of milk, tablespoons of French dressing, slices of bread, pieces of fruit, etc. If you had a piece of pizza, how big was it in inches or sections, etc.? Record exact amounts to the best of your ability.

Sample Breakfast

Raisin bran cereal	1 cup.
2% milk	6 oz.
Orange	1 medium size, 3" diameter
Toast (whole wheat)	1 slice
with butter	1 teaspoon
with strawberry jam	1 tablespoon
Black coffee	1 cup (8 oz.)
with sugar	2 teaspoons

4. Remember to include beverages, and anything your child may add to them, such as milk or sweetener.
5. Remember to include anything added to a food after it is prepared, such as margarine, salt, catsup, mustard and the estimated amount.
6. If you need additional space, use the back of the paper or attach additional sheets.
7. Answer the question at the bottom of the day's record. (Does this day's record represent your child's usual food intake? ___ Yes ___ No). If your answer is no, explain why it wasn't representative. Was your child ill or was he/she on a special diet? Did you have unexpected guests and you took them out to dinner?
8. If you have any other questions concerning the food diary, please call Naiman at 217-244-2962. Or email Naiman at nakhan2@illinois.edu

1. Has food intake declined over the past 3 months due to loss of appetite, digestive problems, chewing or swallowing difficulties?

0= severe loss of appetite

1= moderate loss of appetite

2= no loss of appetite

Response: ____

2. Have you suffered any psychological stress or acute disease in the past 3 months?

0= yes

2= no

Response: ____

APPENDIX E

NUTRITION BELIEFS QUESTIONNAIRE

Now I'm going to ask you about different foods. Sometimes you hear teachers and parents talking about which foods are good for you and which foods are not. I'm going to ask you which of two foods is better for helping you grow up strong and healthy.

1) Whole wheat bagel or Fat-free cookie

Reason:

2) Diet Coke or 2% Milk

Reason:

3) Apple or Fat-free fruit snack

Reason:

4) Fat-free ice cream or Cottage Cheese

Reason:

5) Baked sweet potato or Baked potato chips

Reason:

6) Baked chicken nuggets or Deli sliced chicken

Reason:

7) Fat-free cookie or Regular cookie

Reason:

8) Regular Coke or Diet Coke

Reason:

9) Fat-free- fruit snack or Regular fruit snack

Reason:

10) Regular ice cream or Fat-free ice cream

Reason:

11) Baked potato chips or Potato fries

Reason:

12) Fried chicken nuggets or Baked chicken nuggets

Reason:

13) Whole wheat bagel or Regular cookie

Reason:

14) Regular Coke or 2% milk

Reason:

15) Apple or Regular fruit snack

Reason:

16) Regular ice cream or Cottage cheese

Reason:

17) Baked sweet potato or Potato fries

Reason:

18) Fried chicken nuggets or Deli sliced chicken

Reason:

APPENDIX F

CHILDREN'S EATING ATTITUDES TEST

These questions ask about your eating habits and attitudes. Please answer as quickly and as honestly as possible by circling/underlining one response for each statement.

1. I am scared about being overweight.

Never Rarely Sometimes Often Usually Always

2. I stay away from eating when I am hungry.

Never Rarely Sometimes Often Usually Always

3. I think about food a lot of the time.

Never Rarely Sometimes Often Usually Always

4. I have gone on eating binges where I feel that I might not be able to stop.

Never Rarely Sometimes Often Usually Always

5. I cut my food into small pieces.

Never Rarely Sometimes Often Usually Always

6. I am aware of the energy (calorie) content in foods that I eat.

Never Rarely Sometimes Often Usually Always

7. I try to stay away from foods such as breads, potatoes, and rice.

Never Rarely Sometimes Often Usually Always

8. I feel that others would like me to eat more.

Never Rarely Sometimes Often Usually Always

9. I throw up after I have eaten.

Never Rarely Sometimes Often Usually Always

10. I feel very guilty after eating.

Never Rarely Sometimes Often Usually Always

11. I think a lot about wanting to be thinner.

Never Rarely Sometimes Often Usually Always

12. I think about burning up energy (calories) when I exercise.

Never Rarely Sometimes Often Usually Always

13. Other people think that I am too thin.

Never Rarely Sometimes Often Usually Always

14. I think a lot about having fat on my body.

Never Rarely Sometimes Often Usually Always

15. I take longer than others to eat my meals.

Never Rarely Sometimes Often Usually Always

16. I stay away from foods with sugar in them.

Never Rarely Sometimes Often Usually Always

17. I eat diet foods.

Never Rarely Sometimes Often Usually Always

18. I think that food controls my life.

Never Rarely Sometimes Often Usually Always

19. I can show self-control around food.

Never Rarely Sometimes Often Usually Always

20. I feel that others pressure me to eat.

Never Rarely Sometimes Often Usually Always

21. I give too much time and thought to food.

Never Rarely Sometimes Often Usually Always

22. I feel uncomfortable after eating sweets.

Never Rarely Sometimes Often Usually Always

23. I have been dieting.

Never Rarely Sometimes Often Usually Always

24. I like my stomach to be empty.

Never Rarely Sometimes Often Usually Always

25. I enjoy trying new rich foods.

Never Rarely Sometimes Often Usually Always

26. I have the urge to throw up after eating.

Never

Rarely

Sometimes

Often

Usually

Always

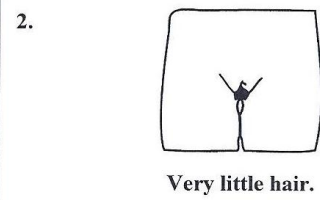
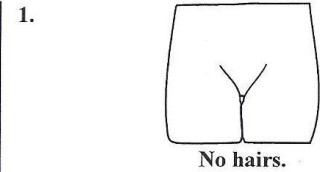
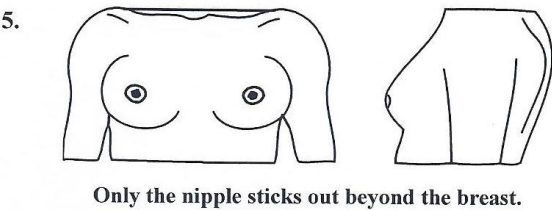
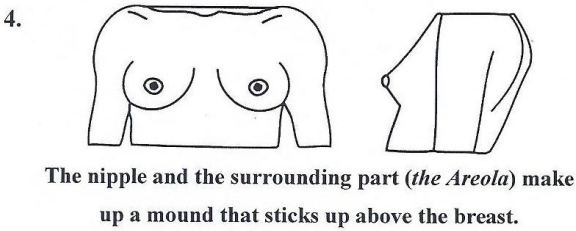
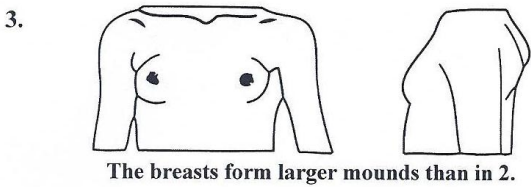
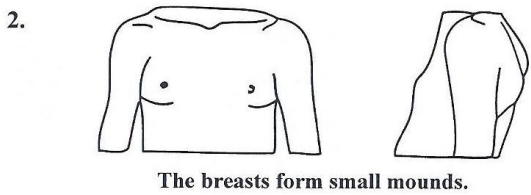
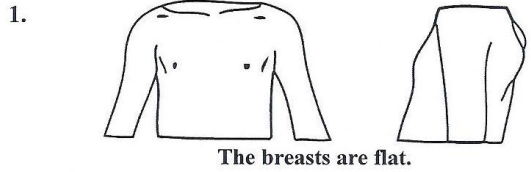
(Maloney et al., 1988)

APPENDIX G

TANNER STAGING SYSTEM

Tanner Staging Questionnaire

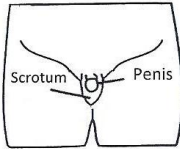
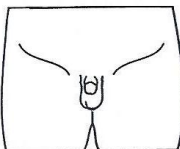
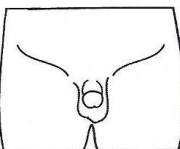
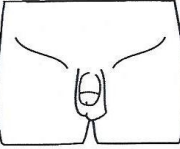
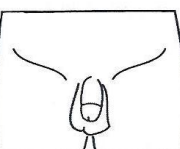
On each side of the line, please circle the number that *best represents* your child's pubertal status.

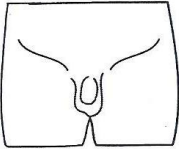
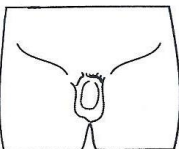

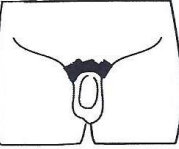
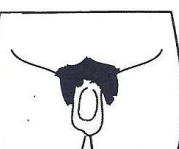


(Tanner, 1962)

Tanner Staging Questionnaire

On each side of the line, please circle the number that *best represents* your child's pubertal status.

1. 
Scrotum and penis are the same size.
2. 
The scrotum has lowered a bit and the penis is a little larger.
3. 
The penis is longer and the scrotum is larger.
4. 
The penis is longer and wider; the scrotum is darker and bigger than before.
5. 
The penis and scrotum are the size and shape of an adult.

1. 
No hairs.
2. 
Very little hair.
3. 
Quite a lot of hair.
4. 
The hair has not spread over the thighs.
5. 
The hair has spread over the thighs.

(Tanner, 1962)

APPENDIX H
ANTHROPOMETRIC FORM

Measures	Trial 1	Trial 2	Trial 3	Comments
Body Mass (kg)				
Height				
BMI (kg/m ²)				
Circumferences (cm):				
Waist - natural				
Waist – umbilicus				
Hip				

LITERATURE CITED

- Adams, K. F. 2006. Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. *N. Engl. J. Med.* 355(8): 763.
- Aeberli, I., M. Kaspar, and M. B. Zimmermann. 2007. Dietary intake and physical activity of normal weight and overweight 6 to 14 year old swiss children. *Swiss Med. Wkly.* 137(29-30): 424-430.
- Ahmed, M. L. 2009. Childhood obesity and the timing of puberty. *Trends in Endocrinology and Metabolism.* 20(5): 237.
- Alaimo, K., C. M. Olson, and E. A. Frongillo Jr. 2001. Low family income and food insufficiency in relation to overweight in US children: Is there a paradox? *Arch. Pediatr. Adolesc. Med.* 155(10): 1161-1167.
- Alexy, U., W. Sichert-Hellert, M. Kersting, and V. Schultze-Pawlitschko. 2004. Pattern of long-term fat intake and BMI during childhood and adolescence--results of the DONALD study. *Int. J. Obes. Relat. Metab. Disord.* 28(10): 1203-1209.
- Ambrosini, G. L. 2009. Adolescent dietary patterns are associated with lifestyle and family psycho-social factors. *Public Health Nutr.* 12(10): 1807.
- Andersen, L. F., I. T. Lillegaard, N. Overby, L. Lytle, K. I. Klepp, and L. Johansson. 2005. Overweight and obesity among norwegian schoolchildren: Changes from 1993 to 2000. *Scand. J. Public Health.* 33(2): 99-106.
- Ashwell, M. 2005. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. *Int. J. Food Sci. Nutr.* 56(5): 303.
- Atkin, L. M., and P. S. Davies. 2000. Diet composition and body composition in preschool children. *Am. J. Clin. Nutr.* 72(1): 15-21.
- Bacha, F., R. Saad, N. Gungor, J. Janosky, and S. A. Arslanian. 2003. Obesity, regional fat distribution, and syndrome X in obese black versus white adolescents: Race differential in diabetogenic and atherogenic risk factors. *J. Clin. Endocrinol. Metab.* 88(6): 2534-2540.
- Bandini, L. G., D. A. Schoeller, H. N. Cyr, and W. H. Dietz. 1990. Validity of reported energy intake in obese and nonobese adolescents. *Am. J. Clin. Nutr.* 52(3): 421-425.
- Bar Or, O. 1983. *Pediatric sports medicine for the practitioner: From physiologic principles to clinical applications.* . New York, Springer-Verlag.
- Barton, M. 2012. Childhood obesity: A life-long health risk. *Acta Pharmacol. Sin.* 33(2): 189.

- Benn, R. T. 1970. Indices of height and weight as measures of obesity. *J. Epidemiol. Community Health.* 24(1): 64.
- Berkey, C. S. 2004. Sugar-added beverages and adolescent weight change. *Obesity.* 12(5): 778.
- Berkey, C. S., H. R. Rockett, A. E. Field, M. W. Gillman, and G. A. Colditz. 2004. Sugar-added beverages and adolescent weight change. *Obes. Res.* 12(5): 778-788.
- Berkey, C. S., H. R. Rockett, A. E. Field, M. W. Gillman, A. L. Frazier, and C. A. Camargo Jr. 2000. Activity, dietary intake, and weight changes in a longitudinal study of preadolescent and adolescent boys and girls. *Pediatrics.* 105(4): E56.
- Bhargava, A. 2008. Socio-economic, behavioural and environmental factors predicted body weights and household food insecurity scores in the early childhood longitudinal study-kindergarten. *Br. J. Nutr.* 100(02): 438.
- Bhattacharya, J. 2004. Poverty, food insecurity, and nutritional outcomes in children and adults. *J. Health Econ.* 23(4): 839.
- Biro, F. M. 2010. Childhood obesity and adult morbidities. *Am. J. Clin. Nutr.* 91(5): 1499S.
- Björntorp, P. 1992. Abdominal fat distribution and the metabolic syndrome. *J. Cardiovasc. Pharmacol.* 20 Suppl 8: S26-S28.
- Blair, S. N. 2004. The fitness, obesity, and health equation. *JAMA (Chicago, Ill.).* 292(10): 1232.
- Bleich, S., D. Cutler, C. Murray, and A. Adams. 2008. Why is the developed world obese? *Annu. Rev. Public Health.* 29: 273-295.
- Boot, A. M., J. Bouquet, M. A. de Ridder, E. P. Krenning, and S. M. de Muinck Keizer-Schrama. 1997. Determinants of body composition measured by dual-energy X-ray absorptiometry in dutch children and adolescents. *Am. J. Clin. Nutr.* 66(2): 232-238.
- Bouchard, C., and A. Tremblay. 1997. Genetic influences on the response of body fat and fat distribution to positive and negative energy balances in human identical twins. *J. Nutr.* 127: S943-S947.
- Bowman, S. A., S. L. Gortmaker, C. B. Ebbeling, M. A. Pereira, and D. S. Ludwig. 2004. Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. *Pediatrics.* 113(1 Pt 1): 112-118.
- Bradlee, M. L. 2010. Food group intake and central obesity among children and adolescents in the third national health and nutrition examination survey(NHANES III). *Public Health Nutr.* 13(06): 797.

- Carey, V. J., E. E. Walters, G. A. Colditz, C. G. Solomon, W. C. Willett, B. A. Rosner et al. 1997. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. the nurses' health study. *Am. J. Epidemiol.* 145(7): 614-619.
- Casey, P. H., K. Szeto, S. Lensing, M. Bogle, and J. Weber. 2001. Children in food-insufficient, low-income families: Prevalence, health, and nutrition status. *Arch. Pediatr. Adolesc. Med.* 155(4): 508-514.
- Catalano, P., A. Thomas, L. Huston Presley, and S. Amini. 2003. Increased fetal adiposity: A very sensitive marker of abnormal in utero development. *Obstet. Gynecol.* 189(6): 1698-1704.
- Caterson, I. D. 2002. Obesity: Epidemiology and possible prevention. *Baillière's Best Practice Research. Clinical Endocrinology Metabolism.* 16(4): 595.
- Church, T. S. 2002. Associations between cardiorespiratory fitness and C-reactive protein in men. *Arterioscler. Thromb. Vasc. Biol.* 22(11): 1869.
- Cole, T. J. 2000. Establishing a standard definition for child overweight and obesity worldwide: International survey. *BMJ. British Medical Journal.* 320(7244): 1240.
- Conway, J. M., F. F. Chanetsa, and P. Wang. 1997. Intraabdominal adipose tissue and anthropometric surrogates in african american women with upper- and lower-body obesity. *Am. J. Clin. Nutr.* 66(6): 1345-1351.
- Crawford, P. B. 1994. Comparative advantage of 3-day food records over 24-hour recall and 5-day food frequency validated by observation of 9- and 10-year-old girls. *J. Am. Diet. Assoc.* 94(6): 626.
- Crawford, P. B., E. Obarzanek, J. Morrison, and Z. I. Sabry. 1994. Comparative advantage of 3-day food records over 24-hour recall and 5-day food frequency validated by observation of 9- and 10-year-old girls. *J. Am. Diet. Assoc.* 94(6): 626-630.
- Daniels, S. R., J. A. Morrison, D. L. Sprecher, P. Khoury, and T. R. Kimball. 1999. Association of body fat distribution and cardiovascular risk factors in children and adolescents. *Circulation.* 99(4): 541-545.
- Danielzik, S., K. Langnase, M. Mast, C. Spethmann, and M. Muller. 2002. Impact of parental BMI on the manifestation of overweight 5-7 year old children. *Eur. J. Nutr.* 41(3): 132-138.
- Davies, P. S. 1997. Diet composition and body mass index in pre-school children. *Eur. J. Clin. Nutr.* 51(7): 443-448.
- Davison, K. K. 2003. Percent body fat at age 5 predicts earlier pubertal development among girls at age 9. *Pediatrics.* 111(4): 815.

- Dawson, B. L. 1988. Television food commercials' effect on children's resistance to Temptation1. *J. Appl. Soc. Psychol.* 18(16): 1353.
- Dennison, B. A. 2008. The role of television in childhood obesity. *Prog. Pediatr. Cardiol.* 25(2): 191.
- Després, J. P. 2006a. Abdominal obesity and metabolic syndrome. *Nature.* 444(7121): 881.
- Després, J. P. 2006b. Is visceral obesity the cause of the metabolic syndrome? *Annals of Medicine (Helsinki).* 38(1): 52.
- Dietz, W. H. 1998. Health consequences of obesity in youth: Childhood predictors of adult disease. *Pediatrics.*101:518-525.
- Dietz, W. H. 1997. Periods of risk in childhood for the development of adult obesity--what do we need to learn? *J. Nutr.* 127(9): 1884S-1886S.
- Donkin, A. J., R. J. Neale, and C. Tilston. 1993. Children's food purchase requests. *Appetite.* 21(3): 291-294.
- Drewnowski, A., and S. E. Specter. 2004. Poverty and obesity: The role of energy density and energy costs. *Am. J. Clin. Nutr.* 79(1): 6-16.
- Dubois, L., A. Farmer, M. Girard, and M. Porcherie. 2006. Family food insufficiency is related to overweight among preschoolers'. *Soc. Sci. Med.* 63(6): 1503-1516.
- Ebbeling, C. B. 2008. Tracking pediatric obesity. *JAMA (Chicago, Ill.).* 299(20): 2442.
- Ebbeling, C. B. 2002. Childhood obesity: Public-health crisis, common sense cure. *Lancet.* 360(9331): 473.
- Eckel, R. H. 2005. The metabolic syndrome. *Lancet.* 365(9468): 1415.
- Eisenmann, J. C. 2008. Combined influence of physical activity and television viewing on the risk of overweight in US youth. *Int. J. Obes.* 32(4): 613.
- Eisenmann, J. C. 2005. Aerobic fitness, body mass index, and CVD risk factors among adolescents: The quebec family study. *Int. J. Obes.* 29(9): 1077.
- Engeland, A. 2004. Obesity in adolescence and adulthood and the risk of adult mortality. *Epidemiology.* 15(1): 79.
- Epstein, L. H. 2000. Decreasing sedentary behaviors in treating pediatric obesity. *Archives of Pediatrics Adolescent Medicine.* 154(3): 220.

- Esmailzadeh, A., P. Mirmiran, and F. Azizi. 2006. Clustering of metabolic abnormalities in adolescents with the hypertriglyceridemic waist phenotype. *Am. J. Clin. Nutr.* 83(1): 36-46.
- Fagard, R. H. 1999. Physical activity in the prevention and treatment of hypertension in the obese. *Med. Sci. Sports Exerc.* 31(11 Suppl): S624-S630.
- Fagot-Campagna, A. 2000. Type 2 diabetes among north adolescents: An epidemiologic health perspective. *J. Pediatr.* 136(5): 664.
- Feldman, W., E. Feldman, and J. T. Goodman. 1988. Culture versus biology: Children's attitudes toward thinness and fatness. *Pediatrics.* 81(2): 190-194.
- Field, A. E. 2004. Snack food intake does not predict weight change among children and adolescents. *Int. J. Obes.* 28(10): 1210.
- Field, A. E. 2003. Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics.* 112(4): 900.
- Finch, M., R. Sutherland, M. Harrison, and C. Collins. 2006. Canteen purchasing practices of year 1?6 primary school children and association with SES and weight status. *Aust. N. Z. J. Public Health.* 30(3): 247-251.
- Finkelstein, E. A. 2009. Annual medical spending attributable to obesity: Payer-and service-specific estimates. *Health Aff.* 28(5): w822.
- Fisher, J. O., Y. Liu, L. L. Birch, and B. J. Rolls. 2007. Effects of portion size and energy density on young children's intake at a meal. *Am. J. Clin. Nutr.* 86(1): 174-179.
- Flegal, K. M. 2010. High adiposity and high body mass index-for-age in US children and adolescents overall and by race-ethnic group. *Am. J. Clin. Nutr.* 91(4): 1020.
- Flegal, K. M. 2006. Overweight in children: Definitions and interpretation. *Health Educ. Res.* 21(6): 755.
- Flegal, K. M., C. L. Ogden, R. Wei, R. L. Kuczmarski, and C. L. Johnson. 2001. Prevalence of overweight in US children: Comparison of US growth charts from the centers for disease control and prevention with other reference values for body mass index. *Am. J. Clin. Nutr.* 73(6): 1086-1093.
- Flegal, K. M., M. D. Carroll, C. L. Ogden, and L. R. Curtin. 2010. Prevalence and trends in obesity among US adults, 1999-2008. *JAMA.* 303(3): 235-241.
- Forshee, R. A., P. A. Anderson, and M. L. Storey. 2008. Sugar-sweetened beverages and body mass index in children and adolescents: A meta-analysis. *Am. J. Clin. Nutr.* 87(6): 1662-1671.

- Freedman, D. S., Z. Mei, S. R. Srinivasan, G. S. Berenson, and W. H. Dietz. 2007. Cardiovascular risk factors and excess adiposity among overweight children and adolescents: The bogalusa heart study. *J. Pediatr.* 150(1): 12-17.e2.
- Fung, T. T. 2001. Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. *Am. J. Clin. Nutr.* 73(1): 61.
- Gantz, W. 2007. Food for thought: Television food advertising to children in the united states. . Menlo Park, CA, Kaiser Family Foundation.
- Garasky, S., S. D. Stewart, C. Gundersen, B. J. Lohman, and J. C. Eisenmann. 2009. Family stressors and child obesity. *Soc. Sci. Res.* 38(4): 755-766.
- Garemo, M., R. A. Lenner, and B. Strandvik. 2007. Swedish pre-school children eat too much junk food and sucrose. *Acta Paediatr.* 96(2): 266-272.
- Gibson, S., and D. Neate. 2007. Sugar intake, soft drink consumption and body weight among british children: Further analysis of national diet and nutrition survey data with adjustment for under-reporting and physical activity. *Int. J. Food Sci. Nutr.* 58(6): 445-460.
- Gillis, L. J., L. C. Kennedy, A. M. Gillis, and O. Bar-Or. 2002. Relationship between juvenile obesity, dietary energy and fat intake and physical activity. *Int. J. Obes. Relat. Metab. Disord.* 26(4): 458-463.
- Gillman, M. W., S. L. Rifas-Shiman, A. L. Frazier, H. R. Rockett, C. A. Camargo Jr, A. E. Field et al. 2000. Family dinner and diet quality among older children and adolescents. *Arch. Fam. Med.* 9(3): 235-240.
- Goran, M. I., and B. A. Gower. 1999. Relation between visceral fat and disease risk in children and adolescents. *Am. J. Clin. Nutr.* 70(1 Part 2): 149S-156S.
- Gordon, N. F. 2009. ACSM's guidelines for exercise testing and prescription.
- Grundy, S. M. 2004. Definition of metabolic syndrome. *Circulation.* 109(3): 433.
- Gundersen, C. 2009. Food insecurity is not associated with childhood obesity as assessed using multiple measures of obesity. *J. Nutr.* 139(6): 1173.
- Gundersen, C. 2008. Child-specific food insecurity and overweight are not associated in a sample of 10-to 15-year-old low-income youth. *J. Nutr.* 138(2): 371.
- Harrison, K. 2005. Is "fat free" good for me? A panel study of television viewing and children's nutritional knowledge and reasoning. *Health Commun.* 17(2): 117-132.
- Hayden Wade, H. A. 2005. Prevalence, characteristics, and correlates of teasing experiences among overweight children vs. non-overweight peers ast. *Obesity.* 13(8): 1381.

- Henry J. Kaiser Family Foundation. 2010. Profiles of generation M2: Media in the lives of 8-to 18-year-olds. Available at <http://www.kff.org/entmedia/upload/8010.pdf>
- Heyward, V. H. 2004. Applied body composition assessment. Champaign, IL: Human Kinetics, 2004.
- Hu, F. B. 2003. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA (Chicago, Ill.)*. 289(14): 1785.
- Hu, F. B. 2001. Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. *Arch. Intern. Med.* 161(12): 1542.
- Jain, M., G. R. Howe, and T. Rohan. 1996. Dietary assessment in epidemiology: Comparison of a food frequency and a diet history questionnaire with a 7-day food record. *American Journal of Epidemiology*. 143(9): 953-960.
- Janssen, I. 2004. Associations between overweight and obesity with bullying behaviors in school-aged children. *Pediatrics*. 113(5): 1187.
- Janssen, I. 2002. Body mass index, waist circumference, and health risk: Evidence in support of current national institutes of health guidelines. *Arch. Intern. Med.* 162(18): 2074.
- Jennings, A. 2011. Diet quality is independently associated with weight status in children aged 9-10 years. *J. Nutr.* 141(3): 453.
- Jordan, A. B. 2007. Heavy television viewing and childhood obesity. *Journal of Children and Media*. 1(1): 45.
- Jouret, B. 2007. Factors associated with overweight in preschool-age children in southwestern france. *Am. J. Clin. Nutr.* 85(6): 1643.
- Kemper, K. A., R. G. Sargent, J. W. Drane, R. F. Valois, and J. R. Hussey. 1994. Black and white females' perceptions of ideal body size and social norms. *Obes. Res.* 2(2): 117-126.
- Keys, A., F. Fidanza, M. J. Karvonen, N. Kimura, and H. L. Taylor. 1972. Indices of relative weight and obesity. *J. Chronic Dis.* 25(6): 329-343.
- Krekoukia, M. 2007. Elevated total and central adiposity and low physical activity are associated with insulin resistance in children. *Metabolism, Clinical and Experimental*. 56(2): 206.
- Kuczmarski, R. J., C. L. Ogden, L. M. Grummer Strawn, K. M. Flegal, S. S. Guo, R. Wei et al. 2000. CDC growth charts: United states. *Advance Data from Vital Health Statistics of the National Center for Health Statistics*. (314): 1-27.
- Kuribayashi, A., M. C. Roberts, and R. J. Johnson. 2001. Actual nutritional information of products advertised to children and adults on saturday. *Children's Health Care*. 30: 309-322.

- Lake, A. A. 2006. Longitudinal change in food habits between adolescence (11–12 years) and adulthood (32–33 years): The ASH30 study. *Journal of Public Health*. 28(1): 10.
- Laraia, B. A., A. M. Siega-Riz, C. Gundersen, and N. Dole. 2006. Psychosocial factors and socioeconomic indicators are associated with household food insecurity among pregnant women. *J. Nutr.* 136(1): 177-182.
- Lawlor, D. A. 2006. Reverse causality and confounding and the associations of overweight and obesity with mortality. *Obesity*. 14(12): 2294.
- Li, C. 2006. Recent trends in waist circumference and waist-height ratio among US children and adolescents. *Pediatrics*. 118(5): e1390.
- Livingstone, M. B. 2004. Issues in dietary intake assessment of children and adolescents. *Br. J. Nutr.* 92(S2): S213.
- Livingstone, M. B., and P. J. Robson. 2000. Measurement of dietary intake in children. *Proc. Nutr. Soc.* 59(2): 279-293.
- Ludwig, D. S. 2001. Relation between consumption of sugar-sweetened drinks and childhood obesity: A prospective, observational analysis. *The Lancet*. 357: 505-508.
- Ludwig, D. S. 2007. Childhood obesity—the shape of things to come. *N. Engl. J. Med.* 357(23): 2325.
- Ludwig, D. S. 2004. Programming obesity in childhood. *The Lancet*. 364(9430): 226.
- Lumeng, J. C., S. Rahnema, D. Appugliese, N. Kaciroti, and R. H. Bradley. 2006. Television exposure and overweight risk in preschoolers. *Arch. Pediatr. Adolesc. Med.* 160(4): 417-422.
- Luo, J., and F. Hu. 2002. Time trends of obesity in pre-school children in china from 1989 to 1997. *Int. J. Obes.* 26(4): 553-558.
- Ma, Y., E. R. Bertone, E. J. Stanek 3rd, G. W. Reed, J. R. Hebert, N. L. Cohen et al. 2003. Association between eating patterns and obesity in a free-living US adult population. *Am. J. Epidemiol.* 158(1): 85-92.
- Maffei, C., A. Pietrobelli, A. Grezzani, S. Provera, and L. Tato. 2001. Waist circumference and cardiovascular risk factors in prepubertal children. *Obes. Res.* 9(3): 179-187.
- Maffei, C., G. Talamini, and L. Tato. 1998. Influence of diet, physical activity and parents' obesity on children's adiposity: A four-year longitudinal study. *Int. J. Obes. Relat. Metab. Disord.* 22(8): 758-764.

- Magarey, A. M., L. A. Daniels, T. J. Boulton, and R. A. Cockington. 2001. Does fat intake predict adiposity in healthy children and adolescents aged 2--15 y? A longitudinal analysis. *Eur. J. Clin. Nutr.* 55(6): 471-481.
- Maggs, J. L., J. Schulenberg, and K. Hurrelmann. 1997. Developmental Transitions during Adolescence: Health Promotion Implications. Pages 522-546 in *Health Risks and Developmental Transitions during Adolescence*. New York, NY, US: Cambridge University Press,
- Malik, V., M. Schulze, and F. Hu. 2006. Intake of sugar-sweetened beverages and weight gain: A systematic review. *Am. J. Clin. Nutr.* 84(2): 274-288.
- Maloney, M. J., J. B. McGuire, and S. R. Daniels. 1988. Reliability testing of a children's version of the eating attitude test. *J. Am. Acad. Child Adolesc. Psychiatry.* 27(5): 541-543.
- Marshall, S. J. 2006. A descriptive epidemiology of screen-based media use in youth: A review and critique. *J. Adolesc.* 29(3): 333.
- McCarthy, H. D. 2005. Trends in waist circumferences in young british children: A comparative study. *Int. J. Obes.* 29(2): 157.
- McGloin, A. 2002. Energy and fat intake in obese and lean children at varying risk of obesity. *Int. J. Obes.* 26(2): 200-7.
- McIntyre, L., N. T. Glanville, K. D. Raine, J. B. Dayle, B. Anderson, and N. Battaglia. 2003. Do low-income lone mothers compromise their nutrition to feed their children? *CMAJ.* 168(6): 686-691.
- McLoyd, V. C. 1990. The impact of economic hardship on black families and children: Psychological distress, parenting, and socioemotional development. *Child Dev.* 61(2): 311.
- Mei, Z. 1998. Increasing prevalence of overweight among US low-income preschool children: The centers for disease control and prevention pediatric nutrition surveillance, 1983 to 1995. *Pediatrics.* 101(1): e12.
- Menaghan, E. G. 1991. Determining children's home environments: The impact of maternal characteristics and current occupational and family conditions. *Journal of Marriage and Family.* 53(2): 417.
- Miller, S. A. 2008. Association between television viewing and poor diet quality in young children. *International Journal of Pediatric Obesity.* 3(3): 168.
- Mokdad, A. H. 2004. Actual causes of death in the united states, 2000. *JAMA (Chicago, Ill.)*. 291(10): 1238.

- Moreno, L. A. 2005. Secular trends in waist circumference in spanish adolescents, 1995 to 2000–02. *Arch. Dis. Child.* 90(8): 818.
- Moreno, L. A., M. Kersting, S. de Henauw, M. Gonzalez-Gross, W. Sichert-Hellert, C. Matthys et al. 2005. How to measure dietary intake and food habits in adolescence: The european perspective. *Int. J. Obes. (Lond).* 29 Suppl 2: S66-77.
- Moreno, L. A., and G. Rodriguez. 2007. Dietary risk factors for development of childhood obesity. *Curr. Opin. Clin. Nutr. Metab. Care.* 10(3): 336-341.
- Moreno, L. A., I. Pigeot, and W. Ahrens. 2010. Childhood obesity: Etiology-synthesis part II: Epidemiology of Obesity Children and Adolescents. 2(2):483-492.
- Morrison, J. A., D. L. Sprecher, B. A. Barton, M. A. Waclawiw, and S. R. Daniels. 1999. Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: The national heart, lung, and blood institute growth and health study. *J. Pediatr.* 135(4): 458-464.
- Muennig, P. 2006. Gender and the burden of disease attributable to obesity. *Am. J. Public Health.* 96(9): 1662.
- Must, A. 1992. Long-term morbidity and mortality of overweight adolescents. *N. Engl. J. Med.* 327(19): 1350.
- Napoli, C. 1999. Influence of maternal hypercholesterolaemia during pregnancy on progression of early atherosclerotic lesions in childhood: Fate of early lesions in children (FELIC) study. *Lancet.* 354(9186): 1234.
- Napoli, C. 1997. Fatty streak formation occurs in human fetal aortas and is greatly enhanced by maternal hypercholesterolemia. intimal accumulation of low density lipoprotein and its oxidation precede monocyte recruitment into early atherosclerotic lesions. *J. Clin. Invest.* 100(11): 2680.
- Neumark Sztainer, D. 2007. Why does dieting predict weight gain in adolescents? findings from project EAT-II: A 5-year longitudinal study. *J. Am. Diet. Assoc.* 107(3): 448.
- Neumark-Sztainer, D. 2002. Ethnic/racial differences in weight-related concerns and behaviors among adolescent girls and boys: Findings from project EAT. *J. Psychosom. Res.* 53(5): 963.
- Neumark-Sztainer, D. 1999. Sociodemographic and personal characteristics of adolescents engaged in weight loss and Weight/Muscle gain behaviors: Who is doing what? *Prev. Med.* 28(1): 40.

- Newby, P. K., D. Muller, J. Hallfrisch, N. Qiao, R. Andres, and K. Tucker. 2003. Dietary patterns and changes in body mass index and waist circumference in adults. *Am. J. Clin. Nutr.* 77(6): 1417-1425.
- Niemeier, H. M., H. A. Raynor, E. E. Lloyd-Richardson, M. L. Rogers, and R. R. Wing. 2006. Fast food consumption and breakfast skipping: Predictors of weight gain from adolescence to adulthood in a nationally representative sample. *J. Adolesc. Health.* 39(6): 842-849.
- Nord, M. 2010. Household food security in the United States (2008). Washington, DC: US Department of Agriculture, Economic Research Service; November 2010.
- Ogden, C. L., M. D. Carroll, L. R. Curtin, M. M. Lamb, and K. M. Flegal. 2010. Prevalence of high body mass index in US children and adolescents, 2007-2008. *JAMA.* 303(3): 242-249.
- Olsen, N. J., and B. L. Heitmann. 2009. Intake of calorically sweetened beverages and obesity. *Obes. Rev.* 10(1): 68-75.
- Ortega, R. M., A. M. Requejo, P. Andres, A. M. Lopez-Sobaler, R. Redondo, and M. Gonzalez-Fernandez. 1995. Relationship between diet composition and body mass index in a group of spanish adolescents. *Br. J. Nutr.* 74(6): 765-773.
- Paffenbarger, R. S., S. N. Blair, I. M. Lee, and R. T. Hyde. 1993. Measurement of physical activity to assess health effects in free-living populations. *Med. Sci. Sports Exerc.* 25(1): 60-70.
- Peeters, A., J. Barendregt, F. Willekens, J. Mackenbach, A. Al Mamun, and L. Bonneux. 2003. Obesity in adulthood and its consequences for life expectancy: A life-table analysis. *Ann. Intern. Med.* 138(1): 24-32.
- Peterson, P. E. 1984. How pronutrition television programming affects children's dietary habits. *Dev. Psychol.* 20(1): 55.
- Phillips, S. M., L. G. Bandini, E. N. Naumova, H. Cyr, S. Colclough, W. H. Dietz et al. 2004. Energy-dense snack food intake in adolescence: Longitudinal relationship to weight and fatness. *Obes. Res.* 12(3): 461-472.
- Poskitt, E. M. E. 1995. Defining childhood obesity: The relative body mass index (BMI). *Acta Pædiatrica.* 84(8): 961.
- Pouliot, M. C., J. P. Desprs, S. Lemieux, S. Moorjani, C. Bouchard, A. Tremblay et al. 1994. Waist circumference and abdominal sagittal diameter: Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *Am. J. Cardiol.* 73(7): 460-468.
- Prentice, A. M. 1995. Obesity in britain: Gluttony or sloth? *BMJ. British Medical Journal.* 311(7002): 437.

- Puhl, R. M. 2007. Stigma, obesity, and the health of the nation's children. *Psychol. Bull.* 133(4): 557.
- Quatromoni, P. A. 2002. The internal validity of a dietary pattern analysis. the framingham nutrition studies. *J. Epidemiol. Community Health.* 56(5): 381.
- Receveur, O. 2008. Consumption of key food items is associated with excess weight among elementary-school-aged children in a canadian first nations community. *J. Am. Diet. Assoc.* 108(2): 362.
- Reilly, J. J. 2005. Early life risk factors for obesity in childhood: Cohort study. *BMJ. British Medical Journal.* 330(7504): 1357.
- Remsberg, K. E. 2005. Early menarche and the development of cardiovascular disease risk factors in adolescent girls: The fels longitudinal study. *J. Clin. Endocrinol. Metab.* 90(5): 2718.
- Reuser, M. 2008. The burden of mortality of obesity at middle and old age is small. A life table analysis of the US health and retirement survey. *Eur. J. Epidemiol.* 23(9): 601.
- Rey-López, J. P. 2008. Sedentary behaviour and obesity development in children and adolescents. *NMCD. Nutrition Metabolism and Cardiovascular Diseases.* 18(3): 242.
- Rico, H. 1993. Body composition in children and tanner's stages: A study with dual-energy x-ray absorptiometry. *Metabolism, Clinical and Experimental.* 42(8): 967.
- Robinson, T. N. 1999. Reducing children's television viewing to prevent obesity. *JAMA (Chicago, Ill.).* 282(16): 1561.
- Rodriguez, G. 2010. Food patterns and nutrient intake in relation to childhood obesity.: 329.
- Rodriguez, G., and L. A. Moreno. 2006. Is dietary intake able to explain differences in body fatness in children and adolescents? *Nutr. Metab. Cardiovasc. Dis.* 16(4): 294-301.
- Rodriguez, G., L. A. Moreno, M. G. Blay, V. A. Blay, J. M. Garagorri, A. Sarria et al. 2004. Body composition in adolescents: Measurements and metabolic aspects. *Int. J. Obes. Relat. Metab. Disord.* 28 Suppl 3: S54-8.
- Romero Corral, A. 2008. Accuracy of body mass index in diagnosing obesity in the adult general population. *Int. J. Obes.* 32(6): 959.
- Rose, D. 2006. Household food insecurity and overweight status in young school children: Results from the early childhood longitudinal study. *Pediatrics.* 117(2): 464.
- Rowlands, A. V., R. G. Eston, and D. K. Ingledew. 1999. Relationship between activity levels, aerobic fitness, and body fat in 8- to 10-yr-old children. *J. Appl. Physiol.* 86(4): 1428-1435.

- Sardinha, L. B. 2007. Objectively measured time spent sedentary is associated with insulin resistance independent of overall and central body fat in 9-to 10-year-old portuguese children. *Diabetes Care*. 31(3): 569.
- Savva, S. C. 2000. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. *Int. J. Obes*. 24(11): 1453.
- Scheier, L. M. 2005. What is the hunger-obesity paradox? *J. Am. Diet. Assoc.* 105(6): 883-4, 886.
- Schneider, M., G. F. Dunton, and D. M. Cooper. 2007. Media use and obesity in adolescent females. *Obesity (Silver Spring)*. 15(9): 2328-2335.
- Sekine, M., T. Yamagami, S. Hamanishi, K. Handa, T. Saito, S. Nanri et al. 2002. Parental obesity, lifestyle factors and obesity in preschool children: Results of the toyama birth cohort study. *Journal of Epidemiology*. 12(1): 33-39.
- Serra Majem, L., L. Ribas Barba, J. Aranceta Bartrina, C. Perez Rodrigo, P. Saavedra Santana, and L. Pena Quintana. 2003. Childhood and adolescent obesity in spain. results of the enKid study (1998-2000)]. *Med. Clin. (Barc)*. 121(19): 725-732.
- Shvartz, E., and R. C. Reibold. 1990. Aerobic fitness norms for males and females aged 6 to 75 years: A review. *Aviat. Space Environ. Med.* 61(1): 3-11.
- Signorielli, N., and J. Staples. 1997. Television and children's conceptions of nutrition. *Health Commun.* 9
- Sjöberg, R. L., and Sjöberg. 2005. Obesity, shame, and depression in school-aged children: A population-based study. *Pediatrics*. 116(3): e389.
- Sobal, J. 1994. Social and economic consequences of overweight in adolescence. *N. Engl. J. Med.* 330(9): 647.
- Sorensen, T., R. Price, A. Stunkard, and F. Schulsinger. 1989. Genetics of obesity in adult adoptees and their biological siblings. *Br. Med. J.* 298(6666): 87-90.
- Stifel, D. C. 2009. Childhood overweight in the united states: A quantile regression approach. *Economics and Human Biology*. 7(3): 387.
- Strasburger, V. C. 2011. Children, adolescents, obesity, and the media. *Pediatrics*. 128(1): 201.
- Strauss, R. S. 1999a. Influence of the home environment on the development of obesity in children. *Pediatrics*. 103(6): e85.

- Strauss, R. S. 1999b. Self-reported weight status and dieting in a cross-sectional sample of young adolescents: National health and nutrition examination survey III. *Arch. Pediatr. Adolesc. Med.* 153(7): 741-747.
- Stunkard, A. J. 1986. An adoption study of human obesity. *N. Engl. J. Med.* 314(4): 193.
- Sundquist, J. 1998. The influence of socioeconomic status, ethnicity and lifestyle on body mass index in a longitudinal study. *Int. J. Epidemiol.* 27(1): 57.
- Svendsen, O. L., C. Hassager, I. Bergmann, and C. Christiansen. 1993. Measurement of abdominal and intra-abdominal fat in postmenopausal women by dual energy X-ray absorptiometry and anthropometry: Comparison with computerized tomography. *Int. J. Obes.* 17(1): 45-51.
- Tanner, J. M. 1962. *Growth at adolescence.* . Oxford, Blackwell Scientific Publications.
- Taylor, R. W., D. Keil, E. J. Gold, S. M. Williams, and A. Goulding. 1998. Body mass index, waist girth, and waist-to-hip ratio as indexes of total and regional adiposity in women: Evaluation using receiver operating characteristic curves. *Am. J. Clin. Nutr.* 67(1): 44-49.
- Taylor, S. J. C. 2001. Performance of a new pubertal self-assessment questionnaire: A preliminary study. *Paediatr. Perinat. Epidemiol.* 15(1): 88.
- Terry, T. K., and Huang. 2007. Metabolic syndrome in youth: Current issues and challenges. *Applied Physiology, Nutrition, and Metabolism.* 32(1): 13.
- Thompson, S. H. 1996. Black and white adolescent males' perceptions of ideal body size. *Sex Roles.* 34(5-6): 391.
- Thorpe, K., C. Florence, D. Howard, and P. Joski. 2004. The impact of obesity on rising medical spending. *Health Aff. Suppl Web Exclusives:* W4-480.
- Toschke, A. M. 2005. Meal frequency and childhood obesity. *Obes. Res.* 13(11): 1932.
- Treuth, M. S., G. R. Hunter, and T. Kekes Szabo. 1995. Estimating intraabdominal adipose tissue in women by dual-energy X-ray absorptiometry. *Am. J. Clin. Nutr.* 62(3): 527-532.
- Tucker, L. 1997. Body fat percentage of children varies according to their diet composition. *J. Am. Diet. Assoc.* 97(9): 981.
- Utter, A. C., and UTTER. 2002. Children's OMNI scale of perceived exertion: Walking/running evaluation. *Med. Sci. Sports Exerc.* 34(1): 139.
- Utter, J., D. Neumark-Sztainer, R. Jeffery, and M. Story. 2003. Couch potatoes or french fries: Are sedentary behaviors associated with body mass index, physical activity, and dietary behaviors among adolescents? *J. Am. Diet. Assoc.* 103(10): 1298-1305.

- Vicente-Rodríguez, G. 2008. Television watching, videogames, and excess of body fat in spanish adolescents: The AVENA study. *Nutrition*. 24(7-8): 654.
- Viner, R. M. 2005. Television viewing in early childhood predicts adult body mass index. *J. Pediatr*. 147(4): 429.
- Wang, Y. 2001. Cross-national comparison of childhood obesity: The epidemic and the relationship between obesity and socioeconomic status. *Int. J. Epidemiol*. 30(5): 1129.
- Wang, Y., and Q. Zhang. 2006. Are american children and adolescents of low socioeconomic status at increased risk of obesity? changes in the association between overweight and family income between 1971 and 2002. *Am. J. Clin. Nutr*. 84(4): 707-716.
- Wang, Z. 2002. Association between overweight or obesity and household income and parental body mass index in australian youth: Analysis of the australian national nutrition survey, 1995. *Asia Pac. J. Clin. Nutr*. 11(3): 200.
- Wechsler, R. 2003. Television viewing and its associations with overweight, sedentary lifestyle, and insufficient consumption of fruits and vegetables among US high school students: Differences by race, ethnicity, and gender. *J. Sch. Health*. 72(10): 413-21.
- Wei, M., L. W. Gibbons, T. L. Mitchell, J. B. Kampert, C. D. Lee, and S. N. Blair. 1999. The association between cardiorespiratory fitness and impaired fasting glucose and type 2 diabetes mellitus in men. *Ann. Intern. Med*. 130(2): 89-96.
- Whitaker, R. C. 1997. Predicting obesity in young adulthood from childhood and parental obesity. *N. Engl. J. Med*. 337(13): 869.
- Wiecha, J. L. 2006. When children eat what they watch: Impact of television viewing on dietary intake in youth. *Archives of Pediatrics Adolescent Medicine*. 160(4): 436.
- Wiecha, J. L., K. E. Peterson, D. S. Ludwig, J. Kim, A. Sobol, and S. L. Gortmaker. 2006. When children eat what they watch: Impact of television viewing on dietary intake in youth. *Arch. Pediatr. Adolesc. Med*. 160(4): 436-442.
- Wilson, D. B., R. Sargent, and J. Dias. 1994. Racial differences in selection of ideal body size by adolescent females. *Obes. Res*. 2(1): 38-43.
- Wu, Q. 2006. Parental obesity and overweight affect the body-fat accumulation in the offspring: The possible effect of a high-fat diet through epigenetic inheritance. *Obesity Reviews*. 7(2): 201.
- Zimmerman, F. J. 2010. Associations of television content type and obesity in children. *Am. J. Public Health*. 100(2): 334.