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ASSOCIATION BETWEEN THE FAMILY NUTRITION AND PHYSICAL ACTIVITY (FNPA) SCREENING TOOL AND CARDIOVASCULAR DISEASE RISK FACTORS IN 10-YEAR OLD CHILDREN

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By

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ABSTRACT

ASSOCIATION BETWEEN THE FAMILY NUTRITION AND PHYSICAL ACTIVITY SCREENING TOOL AND CARDIOVASCULAR DISEASE RISK FACTORS IN 10-YEAR OLD CHILDREN

By

Kimbo Edward Yee

PURPOSE: To examine the association of the Family Nutrition and Physical Activity (FNPA) screening tool, a behaviorally based screening tool designed to assess the obesogenic family environment and behaviors, with cardiovascular disease (CVD) risk factors in 10-year old children. METHODS: One hundred nineteen children were assessed for body mass index (BMI), percent body fat (%BF), waist circumference (WC), total cholesterol, HDL-cholesterol, and resting blood pressure. A continuous CVD risk score was created using total cholesterol to HDL-cholesterol ratio (TC:HDL), mean arterial pressure (MAP), and WC. The FNPA survey was completed by parents. The associations between the FNPA score and individual CVD risk factors and the continuous CVD risk score were examined using correlation analyses. RESULTS: Approximately 35% of the sample were overweight (19%) or obese (16%). The mean FNPA score was 24.6 ± 2.5 (range 18 to 29). Significant correlations were found between the FNPA score and WC ($r = -.35$, $p<.01$), BMI percentile ($r = -.38$, $p<.01$), %BF ($r = -.43$, $p<.01$), and the continuous CVD risk score ($r = -.22$, $p = .02$). No significant association was found between the FNPA score and TC:HDL ($r=0.10$, $p=0.88$) or MAP ($r=-0.12$, $p=0.20$). CONCLUSION: Children from a high-risk, obesogenic family environment as indicated with a lower FNPA score have a higher CVD risk factor profile than children from a low-risk family environment.

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CHAPTER 1

INTRODUCTION

Background and Rationale:

Childhood obesity is a major public health concern given the number of youth affected and its consequences. Currently, 18% of U.S. youth are obese and another 17% are overweight [1-3]. Among the immediate health concerns of obesity is the clustering of adverse cardiovascular disease (CVD) risk factors (i.e. dyslipidemia, hyperglycemia, and hypertension), which has been termed as the metabolic syndrome [4]. Recent studies have shown the emergence of the metabolic syndrome during childhood and adolescence [5-6] with data from the U.S. National Health and Nutrition Examination Survey (NHANES) III (1999-2002) indicating that 10% of adolescents possessed the metabolic syndrome [5]. Besides the immediate consequences, CVD risk factors track from childhood and adolescence into adulthood [6-9], which is of importance since the metabolic syndrome has been shown to be associated with the development of CVD and Type II diabetes in adulthood [10]. Thus, there is considerable interest in the prevention of obesity and CVD risk factors beginning in childhood.

It is well established that dietary behavior and physical inactivity are the two key risk factors influencing obesity and CVD risk factors [11-12]. Among children, the family environment plays an important role in determining dietary and physical activity behaviors [13-14]. Children are unique in that their dietary and physical activity behaviors are influenced directly by parents [13] and indirectly through the construct of the environment provided to them [15-16]. However, from a methodological standpoint it has been difficult to capture the shared family and child environment. Recently, the

Family Nutrition and Physical Activity (FNPA) Screening Tool, which assesses family environmental and behavioral factors (dietary, physical activity, sedentary time, and sleep) that influence children's risk for becoming overweight, was developed [17] and evaluated for its predictive validity of assessing risk for becoming overweight [18]. Results showed that the FNPA could significantly assess changes in BMI over a 1 year period after accounting for baseline BMI, parent BMI, and other demographic variables. The FNPA is the first instrument that combines information from a variety of behaviors (e.g. diet, physical activity and inactivity, sleep patterns, family structure) related to child obesity to evaluate family environments, and has potential for use by pediatricians, school nurses, and other health professionals for quickly assessing a child's family and home environment and his/her risk for becoming overweight. However, additional research using the FNPA is warranted to examine its utility in different settings and populations and other health outcomes besides overweight/obesity (e.g. CVD, metabolic syndrome, type II diabetes).

Purpose of the thesis:

The purpose of this study was to examine the association of the FNPA with a continuous CVD risk score in 10 year old children.

Significance of the Study:

The significance of this study will help expand upon the utility of the FNPA screening tool in assessing not only children's risk at overweight, but also their CVD risk. Also, this study will attempt to replicate the same findings of Ihmels et al., that the FNPA is associated with risk of overweight in children. If significant associations are found in this study then that could lead to future promising usage of the FNPA in smaller clinical

based settings and school based settings for predicting risk of becoming overweight and developing CVD in children.

Hypothesis:

It was hypothesized that the FNPA score will be inversely related to a continuous CVD risk score in 10 year old children.

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CHAPTER 2

LITERATURE REVIEW

Introduction

The problem of childhood obesity and associated cardiovascular and metabolic health has gained considerable interest in recent years. With the increasing burden of obesity and CVD, there is an urgent need to examine the etiology of the metabolic syndrome in order to improve efforts in its prevention, management, and treatment. Both genetic and environmental factors influence obesity and CVD risk factors in children; however, the focus of studies has been predominantly upon environmental or lifestyle influences (e.g. physical activity and diet). Since environmental/lifestyle factors of children are controlled primarily by parents, there is a need for screening tools to assess the family environment. The purpose of this literature review is to inform the reader on 1) CVD risk factors and the metabolic syndrome including its history, definitions, and prevalence in children and adolescents; 2) associations between physical activity, screen time, and diet with CVD risk factors/metabolic syndrome in children, and 3) the development of a screening tool to assess multiple dimensions of lifestyle in children and families.

1. Cardiovascular Disease Risk Factors and the Metabolic Syndrome

1a. The development of CVD risk factors in children

Even though the clinical manifestations of CVD occur in adulthood, various studies have shown atherosclerosis has its origins early in life [19-23]. This evidence stems from autopsy reports of coronary atherosclerotic lesions in children and adolescents, the prevalence of CVD risk factors in children and adolescents, the tracking

of CVD risk factors from childhood to adolescence to young adulthood, and the predictability of adult heart disease from childhood and adolescent CVD risk factors.

Much of our knowledge about CVD risk factor development in youth has been the product of the Bogalusa Heart Study and the Muscatine Study. The following section provides a brief overview of these research programs that have provided evidence for the early origins of atherosclerosis.

In the 1970s, two major epidemiological studies, the Bogalusa Heart Study and the Muscatine Study, began to investigate the development of CVD risk factors in children and adolescents. The Bogalusa Heart Study initially began during the 1973-74 school year in Washington Parish, Louisiana, a political ward consisting of a bi-racial population (63% White, 37% Black). Since the initial cross-sectional survey, several follow-up surveys have been conducted (1976-77, 1978-79, 1981-82, 1984-85, 1987-1988, 1988-91, 1993-94, etc.).

The Muscatine Study began during the 1971-72 and 1972-73 academic years and thereafter included biennial surveys in school-aged children and adolescents and a follow-up survey between the ages of 20 and 34 yrs (Muscatine Young Adult Follow-Up Survey). The initial study population included school children from Muscatine, Iowa. Like the Bogalusa site, Muscatine was chosen due to the relative stability of the population and its proximity to the medical examining team (University of Iowa, Iowa City). In contrast to the Bogalusa Heart Study, the sample in the Muscatine Study consisted of a majority of Caucasians (approximately 96%).

The first published reports from both studies consisted of the descriptive epidemiology (age-, sex-, and race-associated variation) of CVD risk factors in preschool

children, school-aged children and adolescents [24-27]. During subsequent follow-up studies, the tracking of CVD risk factors has been determined over various time periods (i.e., 3-8 yr intervals [8, 28-30]. In general, tracking coefficients for blood pressure and blood lipids ranged from 0.30-0.60. However, tracking is often better at the extremes (i.e., upper quartile). In 2,446 subjects examined for total cholesterol between ages 8-18 yrs and re-examined between 20-30 yrs, 43% remained above the 90^{th} percentile, 62% remained above the 75^{th} percentile, and 81% remained above the 50^{th} percentile of reference values [31]. Along with the critical issue of the persistence of CVD risk factors and the predictability of adult CVD, another major component in establishing evidence in the early origins of atherosclerosis has been to determine the inter-relationships of CVD risk factors, environmental factors, and family history. An important finding from such analyses has been that CVD risk factors cluster in obese children and adolescents [32]. Furthermore, 61% of those subjects in the upper quartile of multiple risk factors during childhood remained in the upper quartile as young adulthood [29].

Familial studies have indicated that adverse CVD risk factors are more common in offspring of parents and relatives with hypertension, diabetes, obesity, hyperlipidemia, and history of myocardial infarction [33-34]. Besides the associations between familial history of CVD and CVD risk factors in offspring, genetic studies have also been conducted to determine candidate genes for CVD risk factors in these two studies [35-38].

Autopsy reports from the Bogalusa Heart Study have furthered the understanding of the relationship between childhood CVD risk factors and the extent of fatty streaks and fibrous plaques in the aorta and coronary arteries [21]. In 204 autopsy cases, 2-39 yrs of

age, 93 cases had data on risk factors. Results indicate that the body mass index (BMI), blood pressure (BP), total cholesterol (TC), triglycerides (TG), low-density lipoproteins (LDL), and high-density lipoproteins (HDL), as a group, are strongly associated with the extent of lesions (canonical correlation, r=0.70). The effect of multiple risk factors, or clustering of risk factors, on the percentage of intimal surface of the aorta or coronary arteries covered with fatty streaks indicates that risk factors during childhood or adolescence are strongly related to the extent of lesions in the aorta and coronary arteries of young adults, and that as the number of risk factors increases so does the severity of asymptomatic coronary and aortic atherosclerosis in young people.

Using a noninvasive method known as computerized beam tomography to detect the presence of atherosclerotic plaque in asymptomatic subjects with CVD, the Muscatine Study has also shown the relationship between childhood CVD risk factors and coronary calcification in young adulthood (29 to 37 yrs, mean age = 33 yrs) [22]. Results indicate that 31% of men and 10% of women have coronary artery calcification. Although CVD risk factors measured during the previous and most recent visits showed the strongest associations with coronary artery calcification, childhood (8-18 yrs, mean age=15 yrs) body weight was a significant predictor of adult coronary artery calcification (Odds ratio = 3.0). Systolic BP, diastolic BP, TC, and TG measured during childhood were not significantly different ($p>0.05$) between young adults showing the presence and absence of coronary artery calcification. Body weight, the BMI and triceps skinfold thickness were larger ($p<0.01$) in males, but not females, during childhood among those who displayed coronary calcification. Unfortunately, HDL, LDL, or apolipoprotein levels were not screened during childhood in this sample.

The Bogalusa Heart Study and the Muscatine Study have provided valuable information regarding the development and persistence of CVD risk factors from childhood through adolescence into young adulthood. As these studies continue, they will provide additional information on the long-term persistence of CVD risk factors into mid- and late-adulthood.

1b. History of the Metabolic Syndrome

The origins of what we now call the metabolic syndrome were first described in the 1920s by Kylin, as a condition involving hypertension, hyperglycemia, and hyperuricemia [39]. In the 1940s, Jean Vague, a pioneer in abdominal obesity research, described the terms "android" and "gynoid" obesity and how the distribution of body fat affects several health problems [40]. Vague particularly found that android obesity was prominent among patients with diabetes and CVD. In 1988, Gerald Reaven described the role of insulin resistance in human disease in the Banting Lecture at the American Diabetes Association conference [41]. In his lecture Reaven described the clustering of traits as "Syndrome X" and the clinical importance of how this clustering of metabolic abnormalities was linked to insulin resistance. Important to note in Reaven's lecture was the lack of obesity as a central factor in "Syndrome X" due to his argument that there were a number of non-obese diabetic individuals. Several other names (e.g. Insulin Resistance Syndrome, The Deadly Quartet, etc.) for this condition arose thereafter, but the term "metabolic syndrome" still remains today and is now accepted worldwide as the description of the constellation of metabolic abnormalities that relate to CVD and type II diabetes [42, 43].

1c. Definitions of the metabolic syndrome

As the clinical importance of the metabolic syndrome continued to grow during the end of the 20th century, health agencies and expert panels attempted to determine a unifying definition for the metabolic syndrome. Although there is a general consensus as to the components of the metabolic syndrome, the clinical cutpoints vary among definitions. The three most well known definitions were produced by the World Health Organization (WHO), International Diabetes Federation (IDF), and the National Cholesterol Education Program – Third Adult Treatment Panel (NCEP ATP III) [4, 42-43]. Table 1 summarizes the definitions from the organizations, and a brief description follows.

1c1. WHO Definition

The WHO definition was created in 1999 with an emphasis of insulin resistance being the major underlying factor contributing to the metabolic syndrome [42]. Insulin resistance was considered as the diagnosis of diabetes mellitus, impaired glucose tolerance (IGT), or impaired fasting glucose. Besides insulin resistance, two of four additional risk factors (hypertension, obesity, raised triglycerides, or low HDL-C) are required for diagnosis.

1c2. NCEP ATP III Definition

Unlike the WHO definition, the ATP III definition did not revolve around the presence of a single underlying risk factor, but the presence of 3 of the following 5 risk factors: abdominal obesity, elevated triglycerides, reduced HDL-C, elevated blood pressure, and elevated fasting glucose [4]. Despite elevated fasting glucose being a contributing risk factor to having metabolic syndrome, the ATP III definition did not

necessarily require the presence of insulin resistance to be diagnosed with metabolic syndrome.

1c3. IDF Definition

In 2004 the IDF convened a workshop to establish a diagnostic definition of the metabolic syndrome to be used in clinical practices [43]. The focus of the IDF was much like the ATP III focus on abdominal obesity rather than insulin resistance. However, what distinguished the IDF definition from the ATP III definition was that central obesity needed to be present as the underlying risk factor for metabolic syndrome. Unlike the ATP III definition that used a set of sex-specific cutpoint for waist circumference to determine central obesity, the IDF definition uses sex- and –ethnic-group specific cut-points. Metabolic syndrome diagnosis required the presence of central obesity and two additional factors: elevated triglycerides, reduced HDL-C, raised blood pressure, and raised fasting plasma glucose.

Table 1. Definitions of the metabolic syndrome.

	WHO	NCEP ATP III	IDF
Underlying Criteria	Glucose intolerance, IGT, or diabetes and/or insulin resistance together with two or more of the following:	3 of 5 clinically identifiable risk factors listed below:	Central obesity together with two or more of the following:
Central Obesity	Males: Waist to hip ratio > 0.90 Females: Waist to hip ratio > 0.85 and/or BMI > 30 kg/m^2	Males: WC ≥ 102 cm Females: WC ≥ 88 cm	Defined as waist circumference with ethnicity specific values (if BMI >30 kg/m^2 then central obesity can be assumed)
Elevated Triglycerides	TG ≥ 150 mg/dL and/or Reduced HDL: Males - ≤35 mg/dL Females - ≤39 mg/dL	≥ 150 mg/dL	> 150 mg/dL or specific treatment for this lipid abnormality
Low High Density Lipoprotein Cholesterol (HDL-C)		Males: < 40 mg/dL Females: <50 mg/dL	Males: <40 mg/dL Females: <50 mg/dL or specific treatment for this lipid abnormality
Elevated Blood Pressure	≥ 140/90 mmHg	≥ 130/85 mmHg	SBP > 130 mmHg or DBP > 85 mmHg or treatment of previously diagnosed hypertension
Elevated Fasting Plasma Glucose	N/A	≥ 100 mg/dL	> 100 mg/dL or previously diagnosed Type II diabetes

Most recently in 2009, a joint scientific statement was released by the IDF, National Heart, Lung, and Blood Institute (NHLBI), AHA, World Heart Federation,

International Atherosclerosis Society, and International Association for the Study of Obesity on harmonizing the criteria used by individual health organizations to define the metabolic syndrome [44]. Organization members agreed that waist circumference would be used to determine central obesity, but that cutpoints would not be set for waist circumference due to variance in measures between gender and ethnicities along with organizations that still favor national or regional waist circumference cut points. However, members agreed upon cutpoints for triglycerides, HDL-C, blood pressure, and fasting glucose. Table 2 summarizes the definition/criteria from this joint statement.

Table 2. Criteria for clinical diagnosis of the metabolic syndrome.

Elevated Waist Circumference*	Population- and country-specific definitions
Elevated Triglycerides (drug treatment for elevated triglycerides is an alternate indicator¶)	≥ 150 mg/dL (1.7 mmol/L)
Low HDL-C (drug treatment for reduced HDL-C is an alternate indicator)	Males: <40 mg/dL (1.0 mmol/L) Females: <50 mg/dL (1.3 mmol/L)
Elevated blood pressure (antihypertensive drug treatment in a patient with a history of hypertension is an alternate indicator¶)	Systolic: ≥ 130 mmHg and/or ≥85 mmHg
Elevated fasting glucose£ (drug treatment of elevated glucose is an alternate indicator)	≥ 100 mg/dL

* It is recommended that the IDF cut points be used for non-Europeans and either the IDF or AHA/NHLBI cut points used for people of European origin until more data are available.

¶ The most commonly used drugs for elevated triglycerides and reduced HDL-C are fibrates and nicotinic acid. A patient taking 1 of these drugs can be presumed to have high triglycerides and low HDL-C. High dose w-3 fatty acids presumes high triglycerides.
£ Most patients with type 2 diabetes mellitus will have the metabolic syndrome by the proposed criteria.

1d. The Metabolic Syndrome in Children

With increasing rates of obesity among childhood [1], the cardiovascular and metabolic complications caused by obesity have led to interest in the metabolic syndrome

of children and adolescents. Several studies have emphasized the importance of the metabolic syndrome in children and the consequences that metabolic syndrome has on adult health status [45-46]. However, the prevalence of metabolic syndrome in children and adolescents varies between studies due to the lack of a universal definition and thus makes it difficult to draw conclusions. Several large population studies such as NHANES, the Bogalusa Heart Study, and the Young Finns study have determined the prevalence of metabolic syndrome in children [4, 16, 47]. The prevalence of metabolic syndrome has been found to be approximately 10% as compared to approximately 35% in adults (according to the NCEP ATP III criteria) [5]. In a study of 261 black preadolescent females and 240 ethnically-diverse preadolescent females, the prevalence of metabolic syndrome ranged from 0.4% to 24.6% when applying different criteria [48].

As indicated above, comparing results across studies has been difficult due to the lack of a universal definition of the metabolic syndrome in both children and adults. Several definitions have been proposed for children and adolescents [44]. Additional studies are needed to establish a universal definition of the metabolic syndrome in children. Once a universal definition is established, future research is warranted towards examining the genetic and lifestyle influences that affect the metabolic disorders in children that lead to an increase in the risk of type II diabetes and CVD.

1f. Continuous CVD risk factor/metabolic syndrome score:

In light of the lack of a universal definition for the metabolic syndrome in children, several researchers have devised the concept of using a continuous score that is representative of a composite CVD risk factor profile (i.e. the metabolic syndrome score) [7, 9, 49]. Risk factors used in calculating a continuous score have varied both in terms

of inclusion and assessment method (i.e. to include a measure of glucose or insulin or not; skinfolds, BMI, or waist circumference as the measure of obesity). Several authors have recommended using variables that are consistent with those used in the adult criteria for determining metabolic syndrome [45, 50]. Of those who have used or have developed a continuous score, Eisenmann [50] is the only one to use criteria that match with the adult criteria (waist circumference, HDL-C, triglycerides, mean arterial pressure, indicator of abnormal glucose metabolism). Statistical procedures to create the score involve standardizing the risk factors/variables chosen and regressing them onto demographic variables such as age, sex, and ethnicity. The standardized z-scores are then summed and derived as the continuous metabolic syndrome score with a higher score representative of an unhealthier metabolic profile. The advantage of using a continuous score is that it can detect associations of diseases/conditions with low prevalence rates in small sample sizes [50].

2. Lifestyle/environmental influences (PA, diet, screen time, family environment), and CVD risk factors/metabolic syndrome in children

2a. Physical Activity and Metabolic Syndrome in Children

Several authors have reviewed the role of physical activity on CVD risk factors and the metabolic syndrome in children [49, 51-56]. In general physical activity is inversely related to metabolic cardiovascular disease risk. For example, results from the European Youth Heart Study show a graded inverse association between physical activity measured via accelerometry and the clustering of metabolic risk factors [49]. When participants were grouped into quintiles based on physical activity counts, those in the most active quintile had less metabolic risk than those of the other quintiles. This study

was unique in that the clustering of risk factors was derived as a composite risk factor score (i.e. continuous score). Also from the European Youth Heart Study, Brage et al. examined the association between physical activity and a clustering of metabolic risk factors also using a composite risk factor score in Danish children [55]. Metabolic risk was found to be inversely related to physical activity measure via accelerometry ($p= 0.008$). Butte et. al [52] showed metabolic risk factors were negatively associated with total physical activity as well as the frequency of bouts of moderate to vigorous intensity levels of physical activity. However, Eisenmann states that the inverse association is weak to moderate at best [56], inferring that physical activity is just one of a multitude of influences on CVD risk factors

Overall, there is a great deal of importance in increasing physical activity levels to prevent the clustering of metabolic risk in children and to decrease any future burden of risk in adulthood. However, population studies examining the relationship between physical activity and clustered metabolic cardiovascular disease risk are limited due to the use of imprecise methods to measure physical activity in children and the lack of an absolute definition for the metabolic syndrome including the use of a continuous score. Future research using more precise, objectively measured physical activity and a universal definition for metabolic syndrome in youth is needed to further understand the influence of physical activity on metabolic syndrome and CVD risk factors in children.

2b. Dietary Behavior and CVD Risk Factors/Metabolic Syndrome in Children

Compared to studies of physical activity, fewer studies have examined the association between dietary behaviors and metabolic syndrome in children. In most

cases, the focus has been upon the effects of dietary intervention on metabolic syndrome/insulin sensitivity [10, 57-58].

ATP III recommendations for dietary modification in patients with metabolic syndrome include low intake of saturated fats and cholesterol, reduced consumption of simple carbohydrates, and increased intakes of fruits, vegetables, and whole grains (i.e. low fat, nutrient-dense diet) [10]. In a study by Chen et al. [57], children with the metabolic syndrome who underwent a 2-week intensive clinical intervention comprised of consuming a high-fiber, low-fat diet coupled with daily aerobic exercise reversed to not having the metabolic syndrome despite still being obese. The low energy dense, high-fiber diet showed that dietary modification aids in decreasing the prevalence of metabolic syndrome in children. Riccardi and Rivellese [58] found similar findings and concluded that the optimal diet for treatment of the metabolic syndrome was one low in saturated fat and high in fibrous/low-glycemic foods.

Overall, the findings of the aforementioned papers are similar to studies that have highlighted the beneficial effects of dietary modification on metabolic factors [59-62] Two of the studies highlighting the benefits from dietary modification focused on the effects of the Mediterranean diet [62-63]. The Mediterranean diet is one that consists of high consumption of fruits and vegetables as well as healthy grains. A staple of the Mediterranean diet is the consumption of large quantities of olive oil and fats in general; however, the ratio of monounsaturated fats to saturated fats is relatively high. The ATTICA study showed that Greek adults who had devoted themselves to the Mediterranean diet had 20% lower odds of having the metabolic syndrome regardless of age, sex, physical activity, lipids, and blood pressure levels [63].

In conclusion, despite the lack of evidence associating dietary behaviors with metabolic syndrome in children, it is clear that a diet that does not promote excessive weight gain, dyslipidemia, or insulin resistance represents the dietary lifestyle for both prevention and treatment of the metabolic syndrome. This diet mainly consists of a low intake of saturated fat and cholesterol and a high intake of nutrient dense foods such as fruits, vegetables, and whole grains. The development of the metabolic syndrome is influenced greatly by the dietary lifestyle of individuals.

2c. Screen Time and Obesity, CVD Risk Factors/Metabolic Syndrome in Children

Several studies have shown the positive association between television viewing and obesity in children and adolescents [51, 64-65]. Using NHANEs data between 1988-1994, Crespo et al. found that the prevalence of obesity was lowest among children who reported watching 1 hour or less of television per day while the prevalence of obesity was highest among children who watched 4 or more hours of television per day [66]. However, television does not account for total screen time as many studies have left out time spent using the computer and playing video games, which has been found to be associated with adverse health problems [65]. Thus, it is important to examine not only the effects of television viewing on obesity, but also computer and video game time as well.

The American Academy of Pediatrics has established guidelines for screen time (including television, computer, and video games) that recommend no more than 2 hours/day of screen time for children and adolescents [67]. However, these screen time guidelines were developed with a focus mainly on obesity and not other outcomes such as the metabolic syndrome. Only a few studies have examined the effects of television

viewing/screen time on metabolic syndrome in children and adolescents [67-68]. Mark and Janssen found that screen time was positively associated with the metabolic syndrome in a dose-response manner that was independent of physical activity [68]. However, Ekelund et. al found only a small association between television viewing and clustered metabolic risk, but determined the significance was due mainly to the association between television viewing and adiposity after controlling for percent body fat [69].

In summary, the positive association between the amount of screen time and obesity is significant, but low. Further studies need to define screen time as the combined amount of television viewing, computer usage, and video game usage. Due to the limited number of studies investigating the effects of screen time on metabolic risks and CVD risk in children, it is suggested that decreasing time spent in sedentary activities will lead to increased levels of physical activity that will in turn decrease children's risk of obtaining metabolic disorders.

2d. Family environment and Obesity/CVD Risk Factors in Children

One general area that has been studied in relation to pediatric overweight and CVD risk development is the area of the family environments and behaviors. Many studies have examined family factors such as parental feeding strategies, parental restriction and reward, family functioning, and household food insecurity in relation to pediatric overweight and CVD risk development in children [70-72]. In regards to feeding strategies, the general conclusion is that although there are several feeding strategies that may increase childhood overweight/obesity risk, there is limited evidence supporting any consistent conclusions. In a study by Melgar-Quinonez [70] and Kaiser,

cross-sectional survey analysis in low-income Mexican-American families showed that biological and socioeconomic factors were more associated with overweight than self-reported child feeding strategies. However, in a study that assessed the association of child-feeding practices and child adiposity, 15% of the variance in fat mass was explained by two practices, pressure to eat and concern for child's weight [71]. This study showed that child-feeding practices explained more variance in fat mass than biological markers such as energy intake.

A clearer area of the family environment's influence has been parental restriction foods and its influence on overweight/obesity risk. Several studies have shown that restriction by parents actually may lead to children to desire forbidden foods [73-74]. One such study by Fisher and Birch [73] evaluated the effects of maternal restriction of snack foods on children's intake of the snack foods when made available. In girls, child and maternal reports of restricting practices predicted snack food intake and higher levels of restriction predicted higher levels of snack food consumption. However, a majority of this research supporting this association has been conducted in non-Hispanic, white girls thus the conclusions may only apply to this population and not others.

Since the evaluation and assessment of family structures and behaviors is complex due to the variety of constructs involved in one individual family to another, the determination that there are family influences on pediatric overweight is difficult to interpret. With that said, is is suggested that parents work to maintain a positive home and family environment where parental control is balanced with child autonomy. Management strategies in relation to diet and physical activity should support the

development of child psychological and behavioral traits and support a positively emotional parent-child relationship.

3. The Family Nutrition and Physical Activity Screening Tool (FNPA)

3a. Development of the Family Nutrition and Physical Activity (FNPA) Screening Tool

The FNPA was developed at Iowa State University by Ihmels and colleagues [15] to assess family environment and behavioral factors that may be associated with children's risk of becoming overweight. The basis of the FNPA stems from an Evidence Analysis project supported by the American Dietetic Association (ADA). The ADA Evidence Analysis on Childhood Overweight is an ongoing project to determine and grade the strength of scientific evidence relating physical activity and dietary behaviors with the risk of becoming overweight and obese. The ADA Evidence Analysis identified ten primary factors (Table 3) that were positively associated with becoming overweight and obese; however, twenty-one questions were created for the survey to better assess the constructs of the FNPA. The questions are coded on a 2, 3, or 4 point Likert scale. The total scores for each construct are summed to provide an overall summary score. A higher FNPA summary score is indicative of a favorable, healthy family environment while a low FNPA summary score is indicative of a family environment that is high risk for children becoming overweight.

Table 3. 10 domains used to create the FNPA. Adopted from Ihmels et. al (2009) [16]

Domain	Example of question from the FNPA
Breakfast and family meal	Does the child eat breakfast and does the family eat a meal together?
Modeling of nutrition	Does the family watch TV while eating and do they eat fast food during the week?
Nutrient dense foods	Does the family eat prepackaged food or do they use fresh foods and fruits and vegetables?
High calorie beverages	Does the family drink soda and Kool-Aid or 100% fruit juices and low fat milk?
Restriction and reward	Does the family use food as a reward and do they restrict unhealthy foods?
Parent modeling physical activity	Do the parents participate in physical activity and does the family participate or play together?
Child's physical activity	Does the child participate in physical activity and organized sports?
Screen time	How many hours of screen time does the child get?
TV in bedroom	Does the child have a TV in his bedroom and do the parents monitor the screen time?
Sleep and schedule	How many hours does the child sleep and is there a bedtime routine?

3b. Validation of the FNPA

The FNPA was evaluated in an urban midwestern (Des Moines, IA) school district by examining the association between FNPA scores and BMI of 854 first grade children. The sample of first grade children were predominantly Caucasian (57.5%), with smaller percentages of African-Americans (15.3%), Hispanics (16.9%), Asians (5.6%), and other minorities (4.7%). The targeted schools varied in socioeconomic status (SES) with 5 schools in high SES (fewer than 33% of the students eligible for free or reduced lunch prices), 17 schools of middle SES status (33% to 66% of the students eligible for free or reduced lunch prices), and 15 schools of low SES (66% of more of the students eligible for free or reduced lunches). Differences in FNPA scores were also

analyzed across different socio-economic and ethnic populations. School nurses measured body mass and stature to determine BMI. BMI percentiles were then computed using the CDC SAS growth chart programs. BMI percentile was used to categorize the children as normal weight, at risk for overweight, and overweight (terminology used at that time). FNPA surveys were sent home to parents to be filled out and returned.

Results revealed that lower income families reported lower FNPA scores compared to higher income families. Factor analysis showed positive correlation between the constructs and child BMI; however, there were greater correlations shown between total FNPA score and child BMI. The average pairwise correlation between the ten constructs of the FNPA was relatively low ($r=0.24$, range, 0.07 to 0.66) signifying relation between the constructs yet a degree of independency between one another. The positive correlations between constructs represented the tendency of behaviors to group together in the home environment. Logistic regression was used to evaluate the construct validity of the FNPA by dividing the FNPA summary scores of the participants into tertiles to test for differences overweight. In the high risk group (i.e. low FNPA summary score) the FNPA significantly predicted BMI ($p = 0.026$). Children who had summary scores in the lowest tertile had an odds ratio of 1.7 (95% CI = 1.07 – 2.80) compared to children who had summary scores in the highest tertile; however, when accounting for parental BMI as a covariate, this association was reduced and no longer significant (indicating that parental BMI needs to be considered when examining the effectiveness of the FNPA).

The FNPA is the first instrument that combines information from a variety of behaviors (e.g. diet, physical activity and inactivity, sleep patterns, family structure)

related to child obesity to evaluate family environments. The FNPA has potential for use by pediatricians, school nurses, and other health professionals for quickly assessing a child's family and home environment and his/her risk for becoming overweight. However, additional research using the FNPA is warranted to validate its usage in different settings and populations and other health outcomes besides overweight/obesity (e.g. CVD risk factors, metabolic syndrome)

4. Summary and Conclusions

The clustering of obesity and CVD risk factors, now terms the metabolic syndrome, is a complex, multifactoral phenotype which is not only affecting adults, but children and adolescents as well. Lifestyle determinants that influence obesity, CVD risk factors, and the metabolic syndrome include physical activity behaviors, diet, and screen time and others.

The associations between these influential lifestyle determinants and CVD risk have been well studied. Physical activity, especially moderate to vigorous intensity activity, is inversely related to CVD risk factors in youth. Diets that are high in fat consumption and low in whole grain, fruit, and vegetable consumption lead to the promotion of obesity, hypertension, dyslipidemia, and/or insulin resistance. Screen time is positively associated with children's risk of developing the metabolic syndrome due to the possible effects of decreasing time spent in physical activity, disrupting healthy sleep patterns, and inducing children's consumption of unhealthy, low density energy foods.

In order to assess these lifestyle and environmental factors that influence children's risk of obesity and adverse CVD and risk factors, a screening tool like the FNPA may prove useful in identifying risk. However, the FNPA has only been validated

in predicting risk of overweight and not other adverse health conditions like CVD or the metabolic syndrome. Future research is warranted to further examine the utility of the FNPA.

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CHAPTER 3

MANUSCRIPT

Introduction

Childhood obesity is a major public health concern given the number of youth it affects and its consequences. Currently, 18% of U.S. youth are obese and another 17% are overweight [1-3] . Among the immediate health concerns of childhood obesity is the development of adverse cardiovascular disease (CVD) risk factors (e.g. dyslipidemia, hyperglycemia, and hypertension) [4]. Besides the immediate consequences, CVD risk factors track moderately well from childhood and adolescence into adulthood [5-8] and CVD remains the leading cause of death in the U.S. Thus, there is considerable interest in the prevention of obesity and CVD risk factors beginning in childhood [9].

It is well established that diet and physical inactivity are the two determinants of obesity and CVD risk factors [9, 10]. Among children, the family environment plays an important role in determining dietary and physical activity behaviors [11, 12]as they can be influenced directly by parents [11] and/or indirectly through the construct of the environment provided to them [13]. However, from a methodological standpoint it has been difficult to capture the shared family and child environment. Recently, the Family Nutrition and Physical Activity (FNPA) Screening Tool, which assesses family environmental and behavioral factors that influence children's risk for becoming overweight, was developed [14] and evaluated for its predictive validity of assessing risk for becoming overweight [15]. Results showed that the FNPA was significantly associated with the prevalence of overweight at baseline [14] and 1 year change in BMI after accounting for baseline BMI, parent BMI, and other demographic variables [15].

The FNPA is the first instrument that combines information from a variety of behaviors (e.g. diet, physical activity and inactivity, sleep patterns, family structure) related to child obesity to evaluate family environments, and has potential for use by pediatrician, school nurses, and other health professionals for assessing a child's family and home environment and his/her risk for becoming overweight. However, additional research using the FNPA is warranted to examine its utility in different settings and populations, and if it has predictive validity for other health outcomes besides overweight/obesity (e.g. CVD, metabolic syndrome, type II diabetes). Therefore, the primary purpose of this study was to examine the association of the FNPA screening tool with CVD risk factors in 10 year old children. The secondary purpose of this study was to examine the FNPA by weight classification of children based on BMI percentile.

Methods

Participants.

The participants were fifth grade children that were part of the (S)Partners for Heart Health intervention, a CVD risk factor prevention and management program [16]. Participants were enrolled in one of two waves of data collection (Fall 2008 or Fall 2009). Since data collection occurred prior to the intervention, these data are cross-sectional. A total of 357 surveys (185 from Fall 2008; 172 from Fall 2009) were sent home to parents of the participants. A total of 171 surveys were returned (return rate of 49%). Six surveys were removed because one or more questions were not answered or answered incorrectly. Of the remaining 165 surveys returned, 46 participants were removed from data analysis due to lack of a key measure (35 declined fingerstick, 8 malfunctioned lipid assay, 2 declined waist circumference, 1 declined blood pressure).

Thus, the final sample size was 119 (60 girls, 59 boys). Participants were primarily Caucasian (>90%). Informed consent and assent were obtained from the participants and the study protocol was approved by the Michigan State University Institutional Review Board.

Anthropometry.

Height and body mass were measured according to standard procedures [17]. Height was measured to the nearest 0.1 cm using a portable stadiometer (Shorr Board stadiometer; Irwin Shorr, Olney, MD). Body mass, measured to the nearest 0.1 kg, and percent body fat were measured using a foot-to-foot bioelectric impedance device (Innerscan Body Composition Monitor BC-534, Tanita Corporation, Tokyo, Japan). Waist circumference was measured using a Gullick anthropometric tape with the anterior superior iliac spine to the nearest 0.1 cm. Body mass index (BMI) was calculated using the following equation: body mass in kg/height in m^2. Percentiles for height, body mass, and BMI were determined using SAS growth chart programs available on-line from the Center for Disease Control (http://www.cdc.gov/growthcharts/).

Blood lipids.

A non-fasting blood sample was obtained by fingerprick and collected in 35 microliter heparinized capillary tubes. The sample was immediately analyzed for total cholesterol (TC) and high-density lipoprotein-cholesterol (HDL) within 5 minutes by a portable analyzer according to the protocol of the manufacturer (Cholestech LDX System, Hayward, CA). Prior to and following daily data collection, the analyzer was calibrated with standard controls provided by the manufacturer. Previous studies have

shown the Cholestech LDX System to be a valid and reliable tool to measure blood lipids [18-19].

Blood pressure.

Resting blood pressure was assessed using a manual blood pressure device with the appropriate sized cuff on the subject's upper right arm following standardized procedures and recommendations [20]. Resting systolic (SBP) and diastolic (DBP) blood pressures were measured by auscultation after the subject was seated for 5 minutes. Mean arterial pressure (MAP) was calculated as DBP + (SBP-DBP/3). Measurements were taken in triplicate at 1-minute intervals. The mean of the three measurements was used for data analysis.

Determination of continuous cardiovascular disease risk score

The continuous CVD risk score was derived by first standardizing the individual CVD risk factors (waist circumference, MAP, and TC:HDL ratio) by regressing them onto age and gender to account for age and gender- related differences in these CVD risk factors (Z_WC, Z_MAP, and Z_TC:HDL). The standardized residuals (z scores) for the individual variables were then summed to create the continuous CVD risk score. A higher CVD risk score indicates a less favorable CVD risk profile.

Family Nutrition and Physical Activity Screening Tool.

The FNPA screening tool was developed by Ihmels and colleagues [14] through a comprehensive Evidence Analyses supported by the American Dietetic Association designed to determine the strength of evidence linking physical activity and diet behaviors with overweight/obesity. The Evidence Analyses identified ten primary factors (breakfast and family meals, modeling of nutrition, nutrient dense foods, high calorie

beverages, restriction and reward, parent modeling physical activity, child's physical activity, screen time, TV in the bedroom, sleep and routine schedule) that were positively associated with becoming overweight and obese. For this study the revised version of the original FNPA was used. The revised version consists of 10 questions assessing the ten constructs mentioned above (http://adaf.eatright-fnpa.org/public/partner.cfm). The revised version is considered more user-friendly and was developed as a result of the initial study as well as from feedback provided by parental focus groups. The score ranges from 10 to 30 with lower scores indicating an adverse, obesogenic family environment.

Statistical analysis.

Descriptive statistics were calculated for all variables and sex differences were examined by independent t-tests for continuous variables. The associations between the FNPA score and the individual CVD risk factor residuals (Z_WC, Z_MAP, and Z_TCHDL), and the continuous CVD risk score (Z_CVD were examined using Pearson correlation coefficients. Differences in the CVD risk factors were also examined by median split of the FNPA by independent t-tests. Differences in the FNPA score by weight status (normal weight, overweight, and obese) were examined using analysis of variance (ANOVA). Statistical analyses were conducted using SPSS version 17.0 (Chicago, IL).

Results

Prior to data analysis of the entire sample, differences between respondents and non-respondents to the survey, and those with incomplete data were examined. There were no significant differences in body mass, BMI, BMI percentile, TC, HDL, TC:HDL,

MAP, WC, or self-reported PA and fruit and vegetable consumption between subjects who returned the FNPA and subjects who did not return it. Subjects that did not return the FNPA had a significantly higher amount of total screen time compared to those who did return the FNPA (4.1 hours vs. 3.0 hours, respectively). There were no significant differences in body mass, BMI, BMI percentile, TC, HDL, TC:HDL, MAP, WC, or self-reported PA and fruit and vegetable consumption between the subjects removed from data analysis (due to an incorrect/incomplete FNPA or from lacking a CVD risk factor measure) and subjects who returned the FNPA.

Table 4 provides the descriptive characteristics of the sample. Mean values for body size and composition variables were not significantly different between sexes ($p>0.05$); however, body mass and BMI were slightly higher in males that resulted in a significantly higher mean BMI percentile and percent of males classified as combined overweight and obese ($p<0.05$). There were no significant sex differences in CVD risk factors or the FNPA score. The percent of subjects classified as overweight or obese was 32.0%, and the percent of subjects with adverse TC, HDL-C, TC:HDL-C, and BP were 25.2%, 28.6%, 39.5%, and 10.1%, respectively.

Pearson correlations between the FNPA score, the standardized residuals of individual CVD risk factors and the continuous CVD risk score are shown in Table 5. The correlations between the FNPA score and Z_TCHDL, Z_WC, and Z_MAP were 0.014, -0.355, and -0.123, respectively, with the correlation between the FNPA score and Z_WC found to be significant ($p<0.05$). The correlations between the FNPA score and other indicators of adiposity – BMI, BMI percentile, and percent body fat (-0.425, -0.377, and -0.430, respectively) – were significant ($p<0.05$) and of similar magnitude as found

with WC. When controlling for Z_WC, the correlations between the FNPA score and Z_TCHDL and Z_MAP were 0.146 and -0.006, respectively.

To further explore the association between the FNPA and CVD risk factors, the sample was split based on the median FNPA score (≤25 and >25) (Table 6). The prevalence of overweight and obese and the mean values for BMI, and percent body fat were significantly higher in children with a FNPA score ≤25 when compared to those with a FNPA score >25 ($p<0.05$). The continuous CVD risk score was not significantly different between these two groups. The mean FNPA score by weight classification is shown in table 7. Overweight and obese children had a significantly lower mean FNPA score when compared to normal weight children ($p<0.05$).

Discussion

This study shows the association between the FNPA screening tool for assessing modifiable home environments and behaviors and CVD risk factors in children. The FNPA score explained about 5% of the total variance in the continuous CVD risk factor score of 10-year old children. The results were stronger for adiposity measures alone.

This study is the first to report the use of the FNPA since the original investigations by Ihmels and colleagues [14, 15], which included cross-sectional and 1-year follow-up of first grade students from Des Moines, IA, USA and examined BMI as the outcome. Compared to the sample in the original investigators, our sample size was considerably smaller and older (119 fifth graders vs. 854 first graders). The magnitude of the correlation found between the FNPA score and child BMI was higher in our study (-0.425 vs. -0.173). When comparing children's FNPA score by median split (≤25 or >25), the prevalence of overweight and obese was greater in children with a score ≤25

compared to children with a score of >25 (43.1% vs. 14.9%). We also found that overweight and obese children had a significantly lower FNPA score compared to normal weight children. Likewise, Ihmels et al. [14] found that children who had a FNPA score in the lowest tertile had an increased odds of overweight (1.7, 95% CI = 1.07 – 2.80) compared to children who had a FNPA score in the highest tertile. On the other hand, the FNPA was not significantly related to TC:HDL or MAP in the current study; therefore, the association with the continuous CVD risk score is probably due to WC as it was significantly correlated with the other CVD risk factors included in the CVD risk score.

Several studies have examined the association between the individual lifestyle factors included in the FNPA (i.e. physical activity, diet, screen time, etc.) and CVD risk factors [21-24]. For example, results from the European Youth Heart Study [21] showed an inverse correlation (r=-0.18) between moderate-to-vigorous physical activity and a continuous CVD risk score. In a study examining the influence of physical activity and total screen time on childhood overweight [22] the correlations between physical activity and total screen time with BMI were -0.25 and 0.22, respectively. A study by Frank et al. [23] investigating the association between diet and CVD risk factors in 10-year old children found low correlations [range: -0.19 to 0.18] between dietary components (protein, fat, carbohydrate, sodium, cholesterol, etc.) and individual CVD risk factors (total cholesterol, systolic blood pressure, skinfolds, etc.). Laurson et al. [24] have also investigated behavioral lifestyle factors using a composite score and its association with BMI, with results showing low correlations (r <0.20) between individual behavioral factors (PA, screen time, dietary variables, family eating) and BMI as well as the composite behavioral score and BMI (r=-0.019). Thus, our results are similar to the

general consensus that behavioral lifestyle factors show low correlations ($r<0.30$) with adiposity and CVD risk factors in children.

Since, the FNPA score explained only 5% of the total variance in CVD risk score, it is important to consider other factors not measured in this study (e.g. genetics, race, and stress) that influence CVD risk factors. Several review papers [25, 26] have outlined the importance of genetic polymorphisms in the pathogenesis of CVD. Using data from the Framingham Heart Study, McQueen et al. [27] found the heritability of a composite metabolic score to be 0.61. Additionally, the general x environmental interaction on CVD risk factors is a topic of great interest [28]. Future studies should examine the combined influence of genetic factors and shared behavioral environment factors determined by the FNPA on CVD risk factors in children.

Although this study showed the association between the FNPA screening tool and CVD risk factors in children, WC appears to driving the association in terms of the continuous CVD risk score. The only significant association between the CVD risk factors and the FNPA score was with WC ($r=-0.355$). Also, when examining the associations amongst the selected CVD risk factors, both TC:HDL and MAP were found to be significantly correlated with WC. These results along with those of Ihmels et. al suggest that the utility of the FNPA is best suited for predicting a child's risk for overweight/obesity. However, since obesity is closely related to CVD risk factors in youth [29], the FNPA screening tool can be used to identify CVD risk by determining risk for overweight. Additional work is needed to further refine and improve the FNPA in both clinical and research settings.

A limitation of this study was the small sample size. Another limitation of the study was the cross sectional design. Similar to the study by Ihmels et al. [15] that used the FNPA to predict change in BMI over 1 year, future studies should use the FNPA to predict change in CVD risk factors using a longitudinal design. Despite the validation of the FNPA screening tool, no studies have yet shown the reliability of the FNPA. Future studies are in development to address this issue. FNPA scores in this study ranged from 18-29 with more than half of the scores $\geq$25. Given the distribution of scores on the higher end, the evaluation of higher risk environments (i.e. a higher percentage of FNPA scores ranging from 10-18) was not possible. In comparison, the only other usage of the revised version of the FNPA had scores ranging from 11-30 with a mean FNPA score of 16 in a sample of obese children seeking treatment [30]. Some FNPA surveys were returned shortly after the start of the intervention; however, no differences in body size, CVD risk factors, FNPA score, or self-reported physical activity, nutrition, and screen time were found when comparing children who returned surveys after the start of the intervention to those who returned surveys before the start of the intervention. A limitation with the continuous CVD risk score used in this study was that it was created using only 3 risk factors (TC:HDL, WC, and MAP) and did not include a measure of insulin resistance. With the use of a continuous CVD risk score, it should be noted that the score itself is sample specific and thus any comparison with another study cannot be made unless demographic variables are similar. Another limitation of this study was the low prevalent rates of CVD risk factors as indicated by the small percentage of children with adverse CVD risk factors.

In conclusion, the role of the family environment on CVD risk factors in children is important to consider due to the influence that the family environment has on children's behaviors (e.g. physical activity and dietary behaviors, sedentary time) related to CVD risk. The FNPA screening tool has the potential to identify children that may be at risk for overweight and the development of CVD risk factors. For clinical and health professionals, the FNPA screening tool can be used for a quick assessment of a child's family environment and behaviors and their risk for becoming overweight and developing adverse CVD risk factors. Interventions designed to modify the family home environment can use the FNPA to determine what changes to implement to improve obesity and CVD risk factors in children. Future research is needed to further examine the utility of the FNPA screening tool in other populations and in longitudinal studies examining both child overweight and CVD risk.

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APPENDIX

Table 4. Descriptive characteristics for males, females, and the total sample.

	Males (n=59)	Females (n=60)	Total (N=119)
Age (yrs)	10.5 (0.4)	10.5 (0.4)	10.5 (0.4) 9.0 – 11.5
Height (cm)	143.9 (6.9)	143.1 (6.4)	143.4 (6.6) 124.6 – 161.8
Weight (kg)	42.1 (11.5)	39.0 (9.8)	40.3 (10.6) 24.9 – 76.7
BMI (kg/m^2)	20.0 (4.1)	18.9 (3.7)	19.4 (3.9) 13.5 – 32.1
BMI Percentile	71.1 (26.0)*	59.3 (29.1)	64.3 (28.3) 2.0 – 99.4
%Overweight (%)	16.0%	23.2%	20.2%
%Obese (%)	20.0%	5.8%	11.8%
WC (cm)	68.5 (12.6)	65.4 (11.1)	66.7 (11.8) 50.1 – 111.3
Percent Body Fat	22.4 (7.9)	23.3 (7.5)	22.9 (7.7) 9.9 – 45.2
TC (mg/dL)	155.5 (27.5)	152.1 (23.6)	153.6 (25.3) 100 – 229
%Adverse - TC	30%	21.7%	25.2%
HDL-C (mg/dL)	48.0 (14.4)	47.8 (16.3)	47.9 (15.5) 21 - 133
% Adverse – HDL-C	28%	29%	28.6%
TC:HDL Ratio	3.6 (1.5)	3.4 (1.0)	3.5 (1.3) 1.4 – 8.8
%Adverse – TC:HDL Ratio	36%	42%	39.5%
SBP (mmHg)	102.7 (12.0)	102.0 (9.9)	102.3 (10.7) 59.5 – 129.0
DBP (mmHg)	67.6 (8.9)	67.6 (8.0)	67.6 (8.4) 60 – 88.5
%Adverse – BP	12%	8.7%	10.1%
MAP (mmHg)	79.4 (8.9)	79.1 (8.0)	79.2 (8.4) 58.0 – 99.3
Z_CVD	-0.00 (2.6)	-0.00 (1.8)	-0.00 (2.2) -4.4 – 5.5
FNPA Score	24.7 (2.5)	24.5 (2.5)	24.6 (2.5) 18-29

Values are mean (SD) for boys and girls. Minimum and maximum (range) also shown for the total sample. * $P<0.05$ for sex difference

%Adverse TC: ≥200 mg/dL; %Adverse HDL-C: <40 mg/dL; %Adverse TC:HDL Ratio: >3.5; %Adverse BP: ≥95 age-, sex-, and height-specific percentile for SBP or DBP

Table 5. Correlations between FNPA score, standardized residuals for TC:HDL ratio, WC, MAP, and CVD risk score in 10 year old children.

	FNPA Score	Z_TCHDL	Z_WC	Z_MAP	Z_CVD
FNPA Score	-	0.014	-0.355**	-0.123	-0.216*
Z_TCHDL	-	-	0.325**	0.149	0.686**
Z_WC	-	-	-	0.330**	0.771**
Z_MAP	-	-	-	-	0.689**
Z_CVD	-	-	-	-	-

** Correlation is significant at the 0.01 level (2-tailed)

* Correlation is significant at the 0.05 level (2-tailed)

Table 6. Differences in body size and CVD risk factors by median split of the FNPA score (≤25 and >25) in 10 year old children.

	FNPA >25	FNPA ≤25
Overweight (%)	10.6%	26.4%*
Obesity (%)	4.3%	16.7%*
Overweight and Obesity (%)	14.9%	43.1%*
BMI (kg/m2)	18.0 (2.7)	20.3 (4.3)*
Percent Body Fat	20.1 (5.7)	24.8 (8.3)
Z_CVD	-0.51 (1.9)	0.33 (2.3)

* Significant difference between groups ($p<0.05$)

Table 7. Differences in the FNPA by weight in 10 year old children.

Classification	Percentage of sample	FNPA score
Normal weight	68.0%	25.1 (2.2)
Overweight	20.2%	23.9 (2.5)*
Obese	11.8%	22.5 (2.7)*

* Significantly different from normal weight category ($p<0.05$).