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NEW PERSPECTIVES FOR UNDERSTANDING THE DEVELOPMENTAL

TRAJECTORY OF METABOLIC SYNDROME AND OBESITY

A Dissertation in

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ABSTRACT

Developmental frameworks and person-centered approaches have the potential to broaden our understanding of the nature and development of health status and disease risk in children. Few studies examining the etiology of metabolic syndrome and obesity during childhood have adopted these frameworks and approaches. The present research aims to address these limitations and provide new information about the development of obesity and metabolic syndrome across childhood and adolescence. Data used in the present research were from a longitudinal study of 197 girls and their parents, followed from daughters' ages 5 to 15 years. In the first study, we used a latent profile approach to identify a metabolic syndrome risk typology based on girls' values for six metabolic syndrome indicators. Statistical support was strongest for a four group solution: 1) Lower MetS Risk, 2) Dyslipidemia Risk, 3) Hypertension Risk, and 4) Higher MetS Risk. Examination of the antecedents of this risk typology revealed girls in the Higher MetS Risk group consumed significantly more sweetened beverages across ages 5 to 13 years and girls in the Dyslipidemia Risk group had the lowest levels of physical activity. These findings illustrate ways to identify girls at higher risk for chronic disease and point to potential opportunities for intervention during childhood to prevent the development of metabolic syndrome. In the second study, we used a growth mixture model approach to identify latent growth trajectories for girls' patterns of BMI change across ages 5 to 15 years. Statistical support was strongest for four patterns of BMI change: 1) Upward Percentile Crossing (UPC); 2) Delayed Downward Percentile Crossing (DDPC); 3) 60th Percentile Tracking (60PT) and 4) 50th Percentile Tracking (50PT). Diet and physical

activity patterns did not predict BMI trajectories, but girls in the UPC group had more overweight mothers, were breastfed for a shorter duration, under-reported dietary intake to a greater degree and presented the worst metabolic outcomes. These findings suggest future research is needed to explore factors other than self-reported diet and activity patterns that may distinguish among differing trajectories of childhood weight status. In the third study, we explored the maternal and psychosocial correlates of heterogeneity for girls' BMI trajectories. Our findings illustrated that girls exhibiting an upward percentile crossing trajectory developed in a distinct ecology compared to other girls in our sample, characterized by a combination of higher maternal weight status, weight concern, and higher levels of maternal restriction of daughter's diet, and more encouragement of daughter's weight loss. Overall, the results of the present research illustrate that girls do not follow a single pathway toward metabolic and weight status outcomes and the psychosocial influences associated with these pathways are multifactorial. Both mothers and daughters should be targeted for childhood obesity and metabolic syndrome prevention and intervention efforts, and our findings suggest several modifiable behaviors that may serve as successful targets for these efforts.

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Chapter 1

GENERAL INTRODUCTION

Obesity is a leading contributor to preventable mortality in U.S. adults, second only to tobacco use (1). Obesity is typically not a direct cause of death, rather contributes indirectly to mortality rates by increasing an individual's risk for developing the metabolic syndrome (defined as the clustering of dyslipidemia, hypertension, insulin resistance and abdominal obesity (2)), which further increases an individual's risk for life-threatening morbidities such as cardiovascular disease and type II diabetes (3). The emergence of obesity as a serious public heath concern has occurred rapidly, as evidenced by a more than doubling of the prevalence of obesity in adults between 1980 and 2004 (4). Additionally, the burden of this increase has been substantial, as demonstrated by corresponding increases in health care expenditures for treatment of obesity-related comorbidities (5-8). These trends illustrate the need to establish a more preventive approach to population health and point to the prevention of adult obesity as a top priority for these efforts.

The etiology of obesity and related comorbidities during childhood

The metabolic syndrome, type II diabetes and cardiovascular disease have traditionally been thought of as adult health concerns. Mounting evidence has suggested, however, that the study of child and adolescent metabolic health is a necessary starting

point for understanding the etiology of these conditions. While the clinical manifestations of cardiovascular disease typically appear in older adults, atherosclerotic plaques and other cardiovascular risk factors have been identified in youth (9). Additionally, the label of "adult-onset diabetes" is no longer appropriate for type II diabetes, as dramatic increases in the prevalence of type II diabetes have been seen in children and adolescents (10). Adult increases in obesity and the metabolic syndrome have been paralleled by dramatic increases in the prevalence of these conditions in child and adolescent populations (9-12) and community-based studies have shown that overweight, the metabolic syndrome, and cardiovascular risk factors track from late childhood and adolescence into adulthood (13, 14). Thus, early identification of youth at risk for subsequent morbidities such as cardiovascular disease and type II diabetes is possible (15) and understanding the development of the metabolic syndrome and obesity during childhood is a viable starting point for risk identification and targeted prevention efforts.

The metabolic syndrome in youth: Issues and research needs

There is currently a lack of consensus for how to best conceptualize the metabolic syndrome in youth; this state is a primary hindrance to progress in this field of research. For adults, several diagnostic definitions have been proposed and are widely used (16-18). For adolescents, researchers have created several different modifications of adult definitions on a per study basis (19-25). For both adults and adolescents, these definitions differ by the specific risk factors included and the cut-off values specified for each risk factor. The commonality of these definitions is their dichotomous categorization of risk:

individuals meeting a certain number of criteria are classified as having the metabolic syndrome.

Simply modifying adult metabolic syndrome definitions for adolescents may not provide an appropriate conceptualization of metabolic health risk in younger populations; in fact, it may actually be erroneous to assume that the development of the metabolic syndrome in younger populations is analogous to that of adults (26). Reaven, in his 1988 landmark Banting Lecture introducing the concept of metabolic syndrome, postulated that the clustering of cardiovascular risk factors comprising the metabolic syndrome (at the time labeled "insulin resistance syndrome") are secondary to the development of insulin resistance (27). Currently, researchers agree that the underlying pathogenesis of the metabolic syndrome in adults is still unclear, but is likely multidimensional and complex (28, 29).

More recently, the importance of obesity for the etiology of the metabolic syndrome has been recognized; excess adiposity is likely the cause of insulin resistance in many, but not all, individuals (30). Growth and puberty place children and adolescents in a metabolic state unique to that of adults, especially with respect to insulin resistance. For example, insulin sensitivity decreases and insulin secretion increases during puberty, independent of changes in body fat (31, 32). Additionally, children experience age-related increases in blood pressure and these increases are amplified during puberty (33). Thus, the etiology of the metabolic syndrome in adolescents may differ from that in adults due to the dynamic metabolic and hormonal fluctuations that occur during growth and puberty (26). Further research is needed to better understand how to conceptualize the metabolic syndrome in youth and this research should explore alternative analytical

approaches that avoid applying assumptions about the metabolic syndrome in adults to the study of the metabolic syndrome in youth.

Latent variable techniques for the study of the metabolic syndrome in youth

Person-centered, latent variable techniques, such as cluster analysis or latent profile analysis, are one possible alternative for exploring the presence of the metabolic syndrome in children and adolescents. Latent variables are not directly observed, rather inferred from variables that are directly observed or measured. Thus, *person-centered* latent variable techniques allow for identification of a multidimensional latent variable based on individuals' profiles for manifest indicator variables (34). Additionally, a key feature of these techniques is that they are data-driven, in that they do not use *a priori* assumptions to classify individuals (35). Subpopulations are identified by their distinct profiles for indicators of interest (36), not by predetermined classification criteria. This feature is particularly attractive for the study of metabolic health in adolescents because of the current lack of data on the etiology of the metabolic syndrome in youth. If *a priori* assumptions about how metabolic risk is manifest in this population can be avoided, a better understanding of the true nature of metabolic risk in youth may be obtained. Metabolic syndrome is, by definition, a latent variable identified by several manifest indicators of cardiovascular risk. Thus, a latent variable approach may be a more suitable way to conceptualize the metabolic syndrome than the dichotomous perspective imposed by the current diagnostic definitions (37).

The development of obesity influences the etiology of the metabolic syndrome during childhood and adolescence

Once there is a better understanding of how to conceptualize the metabolic syndrome in youth, a next step toward building an evidence-base for prevention efforts is to identify developmental factors during childhood that predict the etiology of metabolic syndrome. The presence of overweight and obesity is one of the strongest predictors of metabolic health in all age groups (25, 38). The prevalence of the metabolic syndrome is higher in overweight adolescents, compared to their normal weight peers (19, 25, 39, 40). Weight status is a stronger predictor of cardiovascular risk than insulin and hyperinsulinemia, regardless of whether risk is represented as individual physiological variables or as clustering of cardiovascular risk factors (39). This evidence is limited to secular trends and cross sectional evidence linking overweight to the metabolic syndrome.

To understand how the development of overweight affects the etiology of metabolic syndrome risk individual differences in childhood growth trajectories may be more informative predictors of metabolic syndrome risk however, this hypothesis needs empirical testing. Thoroughly understanding the physiological consequences of developmental trajectories of childhood weight status would provide important insights into the etiology of the metabolic syndrome during adolescence and adulthood.

Latent variable techniques for the study of obesity in youth

Latent variable approaches also hold promise for exploring differences in the developmental trajectories of childhood weight status. In particular, growth mixture modeling is a useful technique for identifying differing patterns of growth within a sample (41). In contrast to traditional growth curve modeling where change over time is modeled as a single developmental trajectory and individual variation around this trajectory is examined, growth mixture modeling allows for fitting of several developmental trajectories based on the distinct profiles of growth present within a sample. These latent trajectory classes, or latent risk trajectories based on a combination of manifest indicators, have the potential to indicate which particular patterns of change are important for which groups of individuals (42), and can be used to predict both proximal and distal outcomes (41). This technique can also examine interactions among measured covariates that may predict change parameters differently for different subgroups (36).

A major advantage of growth mixture modeling is that different groups of trajectories can be identified in cases where trajectory group membership is unknown or unobserved; thus, distinct trajectories within a sample are not specified *a priori,* rather are inferred from the data (36). Growth mixture modeling relaxes two assumptions held by traditional growth curve modeling: 1) that all members of a sample are from the same population; and 2) that covariates or causes of distinct developmental trajectories have the same influence on the growth factors of all individuals in the population. Relaxing these two assumptions is plausible for the study of the development of obesity, as the

onset of obesity can vary between children and the timing of obesity onset may influence, or be influenced by, differing factors.

Understanding lifestyle, psychosocial, and familial correlates of the development of childhood obesity and metabolic syndrome

If prevention of obesity is a first step toward prevention of the metabolic syndrome, then we need to understand the modifiable factors that influence the development of obesity. Overweight and obesity develop when an individual with a genetic predisposition for overweight encounters an "obesigenic" environment; an individual's gene-environment interactions work in concert to either promote or prevent energy intake in excess of energy expenditure (15). The implication for prevention science is that overweight and obesity are products of both genetics and modifiable behaviors. Obesity is a multifactorial problem with influences at the level of the individual (e.g., dietary intake, physical activity patterns, and sedentary behaviors), the family, home, and larger community. Thus, it is useful to know how the developmental trajectory of obesity affects metabolic outcomes, but in terms of prevention, it is also important to identify the specific characteristics of children and families that are associated with the developmental trajectory of overweight and obesity (43).

Influence of energy intake and expenditure on the development of obesity

A proximal determinant of weight gain is a positive energy balance, either due to excess energy intake or inadequate energy expenditure. The main behavioral

determinants of energy imbalance are an individual's patterns of dietary intake, physical activity and sedentary behavior. Thus, assessment of children's specific dietary intake and energy expenditure patterns, and identification of factors that contribute to excess caloric intake or insufficient energy intake, is a necessary first step for understanding the developmental trajectory of obesity during childhood.

However, associations between dietary intake, energy expenditure patterns and weight status have proven to be difficult to detect due to the presence of bias and error in self-reported data (44-47). This limitation arises with the assessment of dietary intake in particular. In fact, numerous authors have concluded overweight is due to low energy expenditure, rather than to overconsumption, because they are unable to link self-reported dietary patterns to weight change or weight status (48-50). Additionally, failures to find differences in the self-reported energy intakes of obese and non-obese individuals have led others to assert that overweight and obese individuals have altered energy requirements (51). An alternative explanation of the failure to note associations between dietary intake and weight status is that older children, adolescents and adults tend to under-report energy intake (46, 52-55), and the tendency to under-report energy intake is typically greater in individuals who are more overweight (47, 56, 57). This systematic bias substantially hinders our ability to accurately detect diet-disease relationships (58, 59). One possible strategy to circumvent this issue is to examine how other markers of obesigenic lifestyle patterns, such as maladaptive eating styles or self-perceptions, relate to developmental trajectories for childhood overweight and obesity.

Associations between diet- and weight-related psychosocial characteristics and obesity

Several psychosocial characteristics that that are related to eating behavior have consistently been related to adults', and more recently to children's, weight status (60-63), and may serve as informative, non-traditional markers of habitual eating behaviors and patterns. For example, dietary disinhibition (defined as the tendency to eat in response to emotional or contextual cues) has been consistently associated with overweight and obesity among both children (62-64) and adults (65-69). Eating in the absence of hunger is a behavioral measure of dietary disinhibition and is defined as the tendency to consume large amounts of palatable foods in a short period of time in the absence to hunger. Eating in the absence of hunger, has been proposed as a behavioral phenotype for obesity, given its implications for habitual overconsumption and consistent associations with overweight and obesity (70).

Additionally, in children, and specifically, in preadolescent and adolescent girls, the combination of dietary restraint (defined as the cognitive control over eating), disinhibition, high weight concerns, low body esteem, and frequent dieting has been implicated in the development of disordered eating patterns, such as anorexia, bulimia and binge-eating disorder, and overweight (71-73). These same findings have been reported for adults (74); however, at moderate levels dietary restraint, weight concern and healthy dieting is characteristic of successful weight loss maintenance across adulthood (75-78). Thus, based on this previous research, the constructs of weight concern and body esteem, as well as measures of eating styles, such as dietary disinhibition, restraint, and

dieting practices, all show promise as predictors of individual differences in weight status trajectories.

Associations among parent characteristics, parent-child interactions and obesity

For children, familial factors must be considered in the development of weight status and disease risk and may also provide non-traditional markers for habitual diet- and weight-related behavioral patterns. Parental characteristics transmit information to children about appropriate attitudes and values for appearance, diet and health (79) and children also develop beliefs about desirable weight status and health behaviors partially based on parental weight status, intake and activity patterns. For example, as early as age 5, a daughter's knowledge of and ideas about dieting are related to maternal dieting practices (80), suggesting that children are attuned to maternal attitudes about weight control and attempts to control food intake, and that this intergenerational influence can occur regardless of whether or not the parent is intentionally attempting to influence child behaviors or development. Thus, assessment of relevant parental behaviors may provide important insights into the etiology of children's diet- and weight-related behaviors and characteristics.

With respect to parent-child interactions, the ultimate goal of most parenting practices is to foster children's internalization of appropriate values and motivations to promote appropriate child behaviors (81). Additionally, children have a natural motivational propensity to acquire social values and behaviors, especially those of parents and family environments, and make them their own (82). Relationships

surrounding diet, eating and the development of health outcomes are no different than other socialized behaviors in that the interactions parents have with their children about food and eating can influence the development of children's regulatory abilities, eating competency or disturbance, food choice and preferences, and eventual weight status (79, 83-85).

With respect to children's eating, parents may restrict children's access to highly palatable, energy dense foods in an attempt to decrease children's preferences for these foods. The use of such practices tends to backfire; restriction can promote increased preference for those same foods, as well as over-consumption of those foods when available in the absence of parental controls (86-89). Parental use of pressure during feeding also negatively affects child intake; parents may pressure children to eat healthful foods, such as vegetables and fruits, however greater pressure from parents is actually associated with lower intakes of vegetables and fruit (90, 91) and higher intakes of dietary fat (90, 92) in children. Children with parents who use more controlling feeding practices tend to be more responsive to external eating cues, such as the presence of palatable food (93). This indicates that the internalization of self-regulatory abilities and healthy eating behaviors is not fostered when parenting practices are not supportive of and responsive to children's cues and needs. Thus, assessment of parental feeding practices may also provide insight into the etiology and development of habitual eating behaviors and patterns related to the developmental trajectory for overweight.

Proposed research

The studies that follow apply theoretical perspectives and analytical approaches that are new to the study of the etiology of obesity and the metabolic syndrome in youth. Few studies within this field have adopted developmental frameworks and even fewer have used person-centered, latent variable approaches to better understand the nature and development of health status and disease risk in children. The three studies that follow aim to address these limitations and provide new information about the development of obesity and metabolic syndrome across childhood and adolescence. Thus, overall goal of this research is to: 1) explore the conceptualization of the metabolic syndrome in youth, 2) explore how the development of overweight during childhood influences the development of the metabolic syndrome during adolescence, and 3) examine individual and familial factors are associated with individual differences in the development of overweight and the metabolic syndrome during childhood and adolescence.

Study 1, entitled *Risk profiles for metabolic syndrome in a non-clinical sample of adolescent girls[1]*, aims to explore how the metabolic syndrome in adolescent girls can be conceptualized. This study explores heterogeneity in metabolic syndrome risk in a sample of non-Hispanic, white adolescent girls. The objective of this study was to assess whether distinctly different profiles for the indicators of the metabolic syndrome exist within a sample of 13-year old girls. Mixture modeling, specifically, latent profile analysis was used to explore profiles based on participants' age 13 values for abdominal obesity, HDL, triglycerides, systolic and diastolic blood pressure and fasting blood glucose. A

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[1] This paper was published in *Pediatrics* in 2006.

secondary aim of this study was to evaluate the growth patterns, pubertal status, family characteristics, dietary patterns and physical activity behaviors that predict metabolic syndrome risk profiles.

Study 2, entitled *Developmental trajectories of childhood body mass index predict adolescent metabolic health outcomes*, focuses on how the development of overweight across ages 5-15 years influences metabolic health outcomes in a sample of non-Hispanic, white girls. The first aim of this study was to explore the possibility of heterogeneity in trajectories of weight status across childhood. Growth mixture modeling was employed to determine whether two or more distinct body mass index (BMI) trajectories exist within our sample of girls. The second aim of this study was to examine whether puberty, dietary intake, physical activity patterns and sedentary behaviors are associated these developmental BMI trajectories. The final aim of this study was to examine how heterogeneity in BMI trajectories associates with metabolic syndrome risk during adolescence.

Study 3, entitled *Maternal characteristics, attitudes, and feeding practices are associated with heterogeneity in daughters' weight status trajectories*, characterizes the family environment, in terms of maternal psychosocial characteristics, eating behaviors, attitudes and feeding practices, within which daughters' weight status trajectories develop. As Study 2 explores the connection between these trajectories and metabolic health outcomes during adolescence, the objective of Study 3 is to understand how maternal modeling of diet- and weight-related behaviors and attitudes, mother-daughter interactions and daughter behaviors and characteristics associate with the development of overweight across ages 5 to 15 years. The aim of this study was to explore the maternal

parenting behaviors, as well as characteristics of mothers and families, that distinguish

the developmental trajectory groups identified in Study 2.

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Chapter 2

**RISK PROFILES FOR METABOLIC SYNDROME IN A NON-CLINICAL
SAMPLE OF ADOLESCENT GIRLS**[2]

ABSTRACT

Objective: To describe risk profiles for metabolic syndrome in adolescence and identify the childhood antecedents for these profiles among a non-clinical sample of non-Hispanic, white girls.

Methods: Participants were part of a longitudinal study ($n = 154$) and assessed at 5, 7, 9, 11, and 13 years of age. At 13 years, girls were grouped based on values for the six metabolic syndrome (MetS) factors (blood pressure, HDL, triglycerides, waist circumference, blood glucose) using a mixture model approach. Fat mass was measured by DXA. Dietary intake was assessed by a 24-hour recall. Mothers reported family demographics and disease history. Girls' physical activity, sedentary behaviors and fitness levels were also assessed.

Results: Statistical support was strongest for a 4 group solution: (1) Lower MetS Risk ($n = 62$); (2) Dyslipidemia Risk ($n = 36$); (3) Hypertension Risk ($n = 33$); and (4) Higher MetS Risk ($n = 21$). At 13, the Hypertension and Higher MetS Risk groups had significantly higher weight status ($M \pm SD$ BMI %tile scores = 72.5±24.7 and 75.8±26.7, respectively) and percent body fat ($M \pm SD$ = 30.1±7.4 and 29.9±7.9, respectively) compared to the Lower MetS and Dyslipidemia Risk groups ($M \pm SD$ BMI %tile scores = 52.2±22.3 and 60.0±25.0; $M \pm SD$ %body fat = 24.2±4.8 and 25.3±4.9, respectively). Additionally, the Higher MetS and Hypertension Risk groups had greater increases in both BMI and fat mass across childhood. The Hypertension and Higher MetS Risk groups had significantly more family history of type II diabetes and obesity. The Higher MetS Risk group consumed significantly more servings of sweetened beverages during

childhood. The Dyslipidemia Risk group had the lowest physical activity participation during childhood and the Lower MetS Risk group had the highest fitness levels at age 13. *Conclusions***:** A risk typology consisting of four groups was identified based on the components of metabolic syndrome. Findings on the antecedents of this risk typology suggest ways to identify those at higher risk for chronic disease and point to potential opportunities for intervention during childhood to prevent the development of metabolic syndrome.

INTRODUCTION

Among U.S. adults, the prevalence of metabolic syndrome (defined as the clustering of dyslipidemia, hypertension, insulin resistance and abdominal obesity by the Adult Treatment Panel [ATP] III (1)) has risen from approximately 24% in 1988-1994 to 27% in 1999-2000 (2). Because the rates of obesity, insulin resistance and cardiovascular disease (CVD) are rising in children and adolescent populations (3-6), research has begun to examine the presence of metabolic syndrome among these younger groups, as well (7-11). In U.S. adolescents (12 to 19-years-of-age), the prevalence of metabolic syndrome is estimated to be approximately 6% (12); yet, there are few data on this issue because children and adolescents are not routinely screened for metabolic syndrome. Additionally, the definition and criteria for metabolic syndrome in adolescents are currently based on a modification of the adult ATP III definition, as age-appropriate definitions are still under investigation (13). However, as adolescence is characterized by accelerated growth and metabolic alterations, the development of metabolic syndrome during adolescence may differ from the development during adulthood (9). Thus, further research on metabolic syndrome risk in this age group is warranted.

Despite widespread use of metabolic syndrome as a construct in both research and clinical practice, there is still considerable debate about its definition and ultimate utility. In the literature on metabolic syndrome among adults, multiple definitions exist with different indicators and cut-offs (2, 3, 14, 15). Additionally, Kahn and colleagues (16) have questioned whether describing a syndrome adds to the understanding of the etiology

and consequences of certain CVD and diabetes risk factors beyond the information provided by the individual risk factors. Researchers have attempted to examine this issue using exploratory and confirmatory factor analysis to investigate whether the relevant indicators for metabolic syndrome reflect one underlying factor or several underlying factors (17-20), but a clear consensus has not yet been reached.

To respond to the need for novel methodological approaches for the study of metabolic syndrome, the present study uses the components of metabolic syndrome to examine a possible typology of risk among a non-clinical sample of non-Hispanic, white 13-year-old girls. The first aim of the present study was to use a data-driven approach to describe latent risk profiles based on the indicators of metabolic syndrome. Specifically, mixture models (21-23) were used to identify qualitatively distinct sub-populations within the sample based on values for abdominal obesity, high density lipoproteins (HDL), triglycerides, systolic and diastolic blood pressure and blood glucose. The second aim was to examine the developmental, lifestyle and family history variables associated with the different risk profiles. Across ages 5-13, developmental antecedents of the metabolic syndrome risk profiles at 13 were examined, including rate of growth, dietary patterns, physical activity, demographic factors and family history. Overall, this study aims to provide a thorough description of potential pathways leading to the emergence of metabolic syndrome and disease risk among a sample of non-Hispanic, white adolescent girls.

METHODS

Participants

The sample included, at study entry, 197 5-year-old girls and their parents. Families were reassessed every 2 years (ages 7, 9, 11, and 13); the final assessment included 168 families. Participating girls who did not provide a blood sample at age 13 (n =14) were not included in the final analyses, therefore, the final sample included 154 girls. Families were recruited for participation in the study using flyers and newspaper advertisements. In addition, families with age-eligible female children within a 5-county radius received mailings and follow-up phone calls (Metromail Inc.). Families were not recruited based on weight status or concerns about weight. Eligibility criteria for girls' participation at the time of recruitment included living with biological parents, the absence of severe food allergies or chronic medical problems affecting food intake, and the absence of dietary restrictions involving animal products.

Families were exclusively non-Hispanic, white and the average income for the sample ranged between $50,000 and $75,000, representing the demographics of the area surrounding the study site. Parents were relatively well educated, with fathers having a mean ± SD of 14.9 ± 2.7 years of education and mothers 14.8 ± 2.3 years. On average, parents were overweight; mothers' mean ± SD body mass index (BMI) was 28.3 ± 6.7, and fathers' mean ± SD BMI was 29.2 ± 4.4. With respect to girls' anthropometric and physiological characteristics at age 13, mean ± SD BMI percentile score was 62.7 ± 26.2. Twenty-seven percent (n=43) of girls were classified as either at-risk-for-overweight or

overweight (BMI percentile score ≥ 85). These proportions are similar to, but slightly lower than, the 2003-2004 NHANES estimates for 12 to 19-year-old non-Hispanic white girls (30.4%) (24). For the total sample, the mean ± SD blood glucose value was 81.9 ± 7.8 mg/dL, for triglycerides was 89.7 ± 44.4 mg/dL, for HDL was 47.8 ± 11.3, for waist circumference was 79.0 ± 11.5, and for blood pressure was 118.1 ± 12.9/66.0 ± 7.4 mmHg.

Measures

Background information. Mothers completed a questionnaire assessing family demographic characteristics, family history of chronic diseases and whether their daughter had been exclusively breastfed, exclusively formula fed, or fed a mix of breast milk and formula.

Weight status and adiposity. At each visit, mothers', fathers' and daughters' height and weight measurements were taken in the laboratory and were used to calculate BMI scores (weight (kg)/height (m)^2). Because girls were all the same age and gender, BMI scores, rather than BMI percentiles or z-scores, were used in both the cross-sectional and longitudinal analyses, following the suggestions of Cole et al (25). Age- and sex-specific BMI percentiles were also calculated and used to determine the prevalence of overweight in girls in this sample. Based on standardized reference criteria (26), a BMI percentile score ≥ the 85th percentile was used to classify girls as at-risk-for-overweight and ≥ the 95th percentile was used to classify girls as overweight. Girls' adiposity was measured at ages 9, 11 and 13 using Dual-Energy X-Ray Absorptiometry

(DXA) scans. Trained nurses measured girls' waist circumference at ages 7, 9, 11, and 13.

Pubertal Status. At ages 7, 9, 11 and 13, breast development was assessed by visual inspection by trained nurses using the Tanner rating scale (27).

Blood Lipids and Glucose. Girls' fasting blood lipid and glucose values were collected at age 13. Blood was collected via a finger prick and samples were analyzed by the Cholestech LDX enzymatic methodology with the Lipid Profile plus Glucose cartridge (Cholestech Corp, Hayward, CA).

Blood pressure. Seated blood pressure was measured after five minutes of quiet resting. On each occasion, two blood pressure readings were taken from the participant's right arm and an average was calculated.

Dietary Intake. Three 24-hour recalls were conducted to assess girls' dietary intake and the Minnesota Nutrition Data System for Research (NDS-R) version 4.06_34 (2003) was used to calculate nutrient intakes. At each assessment point, participants provided three 24-hour recalls within a two to three week period following their laboratory visit. The three recalls included two weekdays and one weekend day and were averaged to provide an assessment of typical daily consumption.

Sport Participation and Fitness. At each visit, girls completed an activity checklist that assessed organized sports participation (i.e. team or structured classes) over the past year. Girls' physical fitness was also measured at each visit using the Progressive Aerobic Cardiovascular Endurance Run (PACER) (28, 29).

TV Viewing and Computer Time. Sedentary behaviors were assessed at each visit by girls' reported number of hours of typical television and computers usage on both

school and non-school days. Girls' mean television viewing and mean computer time were derived by calculating mean hours per day from reported school day and non-school day television and computer use.

Statistical Analyses

The first aim of this study was to use the indicators of metabolic syndrome to find distinct subgroups of risk profiles among this non-clinical sample of non-Hispanic, white adolescent girls. A normal mixture model (or latent profile analysis) approach was used, which assumes that the full sample is composed of a heterogeneous mix of K sub-populations, each with their own mean levels on the risk indicators (30). In the current study, the six indicator variables were: 1) Waist Circumference, 2) Systolic Blood Pressure, 3) Diastolic Blood Pressure, 4) HDL, 5) Triglycerides, and 6) Blood Glucose. In preliminary analyses, two girls were extreme outliers on these indicators. Both met the criteria for metabolic syndrome and one, with a fasting glucose level of 127 mg/dL had been diagnosed with Type II diabetes. The second had a fasting glucose level of 158 mg/dL. Because the glucose levels for these girls were 6 and 10 standard deviations above the group mean, they were excluded from the mixture analysis, as they essentially represented their own clinical subgroup.

For the remaining 152 girls, the indicator variables were standardized and Mplus (30) was used to estimate normal mixture models (where the variables were independent within groups). For each possible number of subgroups (1, 2, 3 … etc) the statistical fit was compared based on the AIC and BIC (31) information criteria. Multiple runs were

conducted from multiple starting values to find the best fitting models. The analyses

showed that the four group solution presented below had the best fit. Posterior

probabilities of group membership were also used to classify girls into their most likely

group. The average posterior probabilities for the groups were $p = .89, .83, .89$ and $.90$

respectively.

Once risk subgroups were determined, the groups were compared on aspects of

their developmental history to assess whether the subgroups differed on variables

associated with metabolic syndrome and obesity. Analysis of Variance (ANOVA) with a

general linear model and Fisher's exact test LSD correction post hoc pair wise

comparison was used to describe group differences. All analyses comparing dietary

intake among groups were controlled for total caloric intake. Chi square analyses or

Fisher's exact tests (if cell sizes were smaller than 5) were used to determine group

differences on dichotomous variables. A mixed modeling approach was used to model

the growth curves for girls' BMI and fat mass change from age 5 to 13 y to determine

associations between group membership and growth trajectories.

RESULTS

Metabolic Syndrome Risk Subgroups

Comparisons of models' fit to the data using successively greater numbers of

classes revealed that a four group solution had the best fit based on information criteria

(31). Table 2.1 presents the mean (SD) values for the metabolic syndrome indicators for

each of the four subgroups, which were labeled based on their relative values for each of the metabolic syndrome indicators used for risk typology classification. As shown in Table 2.1, the first group, the "Lower Metabolic Syndrome (MetS) Risk" group, had mean values within the healthy ranges for all classification variables. The second group, the "HDL-Triglyceride (TG) Risk" group, had relatively higher TG levels and lower HDL levels, but relatively low values for waist circumference, and blood pressure. The third group was labeled the "Hypertension Risk" group, as they had higher blood pressure and waist circumference. The fourth group was labeled the "Higher Metabolic Syndrome (MetS) Risk" group because they had the highest values for waist circumference and blood pressure, and the lowest HDL value, relative to all other groups. Mean values of this group on the six indicator variables came close to or met the cut-off criteria for metabolic syndrome diagnosis in adolescents (13). Groups did not differ in mean blood glucose levels; means for all groups were well below the metabolic syndrome cutoff.

Cut-off Criteria for the Metabolic Syndrome Components

In the total sample, 8 girls were classified as having metabolic syndrome, defined by Cook and colleagues (13) as meeting at least three of the five age-adjusted cut-offs for values on measures of waist circumference, blood glucose, triglycerides, HDL, and blood pressure; 7 of these 8 girls were in the Higher MetS Risk group by the mixture model analysis, the other was in the Hypertension Risk group. The percentages of girls who met each age-adjusted cut-off are presented in Table 2.2.

Weight Status and Adiposity

Table 2.3 presents the anthropometric characteristics of each subgroup at age 13 and illustrates significant differences among the MetS risk groups on BMI (F [3, 147] = 14.57, $p<.001$), BMI percentile scores (F [3, 147] = 8.17, $p<.001$), fat mass (F [3, 131] = 14.52, $p<.001$) and percent body fat (F [3, 147] = 9.32, $p<.001$); the Higher MetS Risk and Hypertension Risk groups were consistent higher than the HDL-TG Risk and Lower MetS Risk groups on all of these variables. In the Lower MetS Risk group, only 5% had BMI percentiles greater than the 85th percentile. In the HDL-TG Risk group, 25% exceeded the 85th percentile. In the Hypertension Risk group 57% exceeded the 85th percentile and 90 % of the girls in the Higher MetS Risk group exceeded the 85th percentile.

Tanner staging of breast development was used as an indicator of pubertal status. There were significant differences among MetS risk groups on level of breast development (F [3, 138] = 5.34, $p<.01$). Post-hoc planned comparisons revealed that the Higher MetS Risk and Hypertension Risk groups had significantly higher scores ($M \pm$ SD = 3.9 $\pm$ 0.2 and 4.4 $\pm$ 0.2, respectively), indicating more advanced stages of breast development than the Lower MetS Risk and the HDL-TG Risk groups ($M \pm$ SD= 3.6 $\pm$ 0.1 and 3.6 $\pm$ 0.1, respectively; $p<.001$).

Developmental Antecedents

Weight and Adiposity Change. Figure 2.1 and Figure 2.2 illustrate differences in patterns of change from 5 to 13 years among the MetS risk groups on BMI and fat mass. At age 5, the Higher MetS Risk group had the highest BMI relative to all other groups (Figure 1; F [3, 148] = 3.95, p<.01). Additionally, the Higher MetS Risk and Hypertension Risk groups had greater changes in BMI between ages 5 and 13 than the Lower MetS Risk and HDL-TG Risk groups (F [3, 147] = 10.02, p<.001). Results were similar for changes in fat mass (Figure 2). The Higher MetS Risk group's fat mass at first measurement (age 9) was significantly higher than the values for the other 3 groups (F [3, 143] = 7.18, p<.01). The change in fat mass between 9 and 13 years of age was significantly greater for the Higher MetS Risk and the Hypertension Risk group than fat mass change for the Lower MetS Risk and the HDL-TG Risk groups (F [3, 143] = 6.51, p<.01).

Family History and Characteristics. No differences were found among groups for family history of hypercholesterolemia, hypertension, stroke, or heart attack. Family history of type II diabetes (F [3, 141] = 4.53, p<.01), obesity (F [3, 147] = 2.55, p=.05) and gestational diabetes (X^2 = 15.56, p<.001) differed significantly among groups. Post-hoc planned comparisons revealed the Higher MetS Risk and Hypertension Risk groups had significantly higher rates of type II diabetes ($M \pm SD$ = 2.2 ± 0.3 family members for both groups) than the Lower MetS Risk and HDL-TG Risk groups ($M \pm SD$ = 1.3 ± 0.2 and 1.2 ± 0.2 family members, respectively). This pattern was the same for family history of obesity: the Higher MetS Risk and Hypertension Risk groups had significantly higher

prevalence of obesity ($M \pm SD = 1.0 \pm 0.2$ and 1.2 ± 0.2 family members, respectively)

than the Lower MetS Risk and HDL-TG Risk groups ($M \pm SD = 0.7 \pm 0.3$ and 0.5 ± 0.3

family members, respectively). Additionally, family history of gestational diabetes

differed across groups; mothers of girls in the HDL-TG Risk (3 mothers) and Higher

MetS Risk (4 mothers) were the only groups that had reported gestational diabetes.

At study entry, girls in the Lower MetS Risk group had mothers (F [3, 141] =

4.69, p=.01) and fathers (F [3, 140] = 4.99, p<.01) with higher levels of education

(Mothers: $M \pm SD = 15.4 \pm 2.4$ years; Fathers $M \pm SD = 15.7 \pm 2.6$ years) than girls in

the Higher MetS Risk group (Mothers: $M \pm SD = 13.6 \pm 2.0$ years; Fathers $M \pm SD =$

14.1 ± 2.4 years). Additionally, girls in the Lower MetS Risk group had mothers (F [3,

147] = 10.64, p<.001) and fathers (F [3, 144] = 3.29, p<.05) with lower BMI (Mothers: M

$\pm SD = 23.8 \pm 3.6$; Fathers $M \pm SD = 27.3 \pm 4.0$) than girls in the Higher MetS risk group

(Mothers: $M \pm SD = 29.9 \pm 7.4$; Fathers $M \pm SD = 30.1 \pm 5.1$). No differences were found

among the four MetS risk groups on family income, birth weight or mothers'

retrospective reports of infant feeding (i.e. exclusive breastfed, exclusively formula fed or

fed a mix of both) or the duration of breastfeeding.

Dietary Intake. Dietary recall data were examined for group differences in

nutrient and food group intakes. The only significant difference among the four MetS risk

groups was in sweetened beverage intake (see Figure 2.3). A significant main effect of

group membership on sweetened beverage intake was seen in the repeated measures

ANOVA (F [3, 143] = 5.08, p<.01). At ages 5, 7, and 9 post hoc comparisons revealed

the Higher MetS Risk group had the highest daily sweetened beverage intake among all

groups (p<.001); this difference, over time was about a half to two-thirds of a serving (and between 40-75 calories) per day. No consistent differences among the MetS Risk groups were found for intakes of any other macronutrient, micronutrient, or food group, or in meal and snack frequency.

Physical Activity, Sedentary Behaviors and Fitness Levels. Across ages 5 to 11, the HDL-TG risk group was significantly lower than all other groups on activity participation (F [3, 147] = 4.70, p<.01). No differences were found among groups for average television or computer time. A significant effect of group membership was noted for fitness levels at age 13 (F [3, 147] = 2.95, p<.05); girls in the Lower MetS Risk group had significantly higher fitness scores than the other three groups (p<.001), the HDL-TG Risk, Hypertension Risk group and Higher MetS Risk groups were not significantly different on fitness scores.

DISCUSSION

The current study provided strong statistical support for four distinct risk profiles among a non-clinical sample of non-Hispanic, white adolescent girls and identified developmental and familial antecedents of these metabolic syndrome risk profiles. The four groups included: a Higher MetS risk group (14% of the sample); a Lower MetS Risk group (41% of the sample); a HDL-TG Risk group (24% of the sample); and a Hypertension Risk group (22% of the sample). At 13, these four groups also differed in BMI, fat mass, pubertal development and fitness levels. With respect to developmental antecedents, both the Higher MetS Risk and the Hypertension Risk groups had

significantly greater increases in weight and fat mass from 5 to 13 years. The Higher

MetS Risk and the Hypertension Risk groups also diverged from the HDL-TG Risk and

Lower MetS Risk groups in family history of obesity and type II diabetes. With respect to

food intake and physical activity, the Higher MetS Risk groups had the highest sweetened

beverage intakes, and Lower MetS risk the highest fitness levels. No differences were

found among groups on indices of sedentary behavior, specifically television viewing and

computer time.

Other research has used factor analysis to address the issue of whether metabolic

syndrome is a more useful conceptualization of risk compared to focusing on individual

indicators (16, 20, 32). Cluster analysis has also been used to address this issue by

looking for greater than chance clustering of dichotomous risk indicators (33). The

present approach instead posits an underlying typology and estimates parameters

associated with the latent groups. Although this mixture model analysis is similar to

factor modeling in accounting for covariation among observed variables by their

association with latent variables (34), there are some differences convenient for the

purposes of this study. Mixture model analysis provided the opportunity to construct

classes of individuals thought to differ qualitatively from one another. It was on the basis

of these classifications that differences among the four risk groups' developmental

antecedents could then be examined and described.

Longitudinal data on development lifestyle factors provided information on

several antecedents of risk classification. Changes in weight and adiposity were

developmental antecedents of interest as weight status, fat mass and change over time for

both of these variables from 5 to 13 were strong predictors of risk group membership.

This is consistent with research showing that overweight and obesity are intricately linked with the prevalence of metabolic syndrome, its various components and its consequences later in life (35-39). Before the 1990's, disease status such as Type II diabetes and cardiovascular disease were thought of as adult conditions, for which children were thought to be at very low risk. But, with the reports of dramatic increases in both cardiovascular disease and type II diabetes in children and adolescents (3, 5, 6, 40, 41), it has became apparent that susceptibility to and development of these chronic diseases can emerge as early as adolescence. Additionally, research has shown that the increased prevalence of type II diabetes and cardiovascular disease in adolescents may be partially attributable to the dramatic increases in overweight in both children and adolescents (10), as well as to patterns of weight change during childhood (42). The present findings reveal that elevated weight status and accelerated change in weight status during middle childhood are predictors of metabolic syndrome risk at age 13 and may serve as a key signal to clinicians and interventionists aimed at preventing metabolic syndrome and cardiovascular disease risk during early childhood.

Several aspects of girls' families were found to differ across risk groups. Girls classified as having higher risk for metabolic syndrome and the components of metabolic syndrome (hypertension, dyslipidemia) were more likely to have a family history of obesity, type II diabetes, and gestational diabetes, and to have parents who were more overweight. Additionally, the lower risk girls had parents who were more educated, although the mean educational levels of all four risk groups reflected 2 to 3 years of college education among parents. Similarities within families for the presence of metabolic syndrome and for traits related to metabolic syndrome have been attributed to

both genetic and shared environment factors (43-46). Thus, although it appears that some girls in the present study may be genetically predisposed to the development of metabolic syndrome, environmental factors, such as parent education level, were also predictive of risk status. Additionally, the difference in parental weight status between the groups is suggestive of both genetic and environmental influences, as overweight parents may be a marker for a less healthy parental eating and activity patterns. This supports the "obesigenic family" view (47-49), suggesting that there are certain environments created by parental behaviors that contribute to the development of overweight and co-morbidities (i.e. metabolic syndrome) in children.

With respect to lifestyle factors, the only dietary pattern that clearly distinguished the higher risk group from the other groups was early elevated intake of calorically sweetened beverages across ages 5 to 11 years. Relative to the Low MetS risk group, at ages 5, 7, and 9 years, the Higher MetS Risk group consumed 27%, 45% and 50% more sweetened beverage servings per day, respectively. This difference would result in an additional 40 to 75 calories per day for the Higher MetS Risk Group over time, which coincides with, and possibly accounts for, the greater weight and weight gain of the Higher MetS Risk group. For example, the consumption of an additional 50 kcal per day persisting over 4 years, assuming 50% of these additional calories are stored as fat, could result in an additional weight gain of about 10 lb (4.5 kg) over a 4 year period. This estimation is close to the difference in weight gain between ages 5 to 9 in the High MetS Risk group (17.62 kg) and the average weight gain in the other three groups (14.3 kg; data not shown). The differences among the groups in their sweetened beverage consumption disappeared by age 11, due to increases in sweetened beverage intake

among the other groups. This pattern suggests the possibility that consistently high intake

of sweetened beverages *early in life* may constitute a risk factor, for excessive weight

gain and increased metabolic syndrome risk. These findings are consistent with research

examining physiological responses to consuming high glycemic load carbohydrates (e.g.

sweetened beverages) which have shown that habitual consumption of these foods

contributes to the development of insulin resistance, especially when these high intakes

are accompanied by the consumption of higher than needed calories on a habitual basis

(50). Based on these findings, it is not unreasonable to expect that girls who have been

consuming higher amounts of sugar sweetened beverages from ages 5 to 11 would have

greater risk for insulin resistance or metabolic syndrome. Additionally, although a clear

consensus on the effect of high intakes of sweetened beverages on weight change and

health status has not been reached, children's consumption of caloric drinks has been

shown to predict change in BMI and overweight prevalence (51, 52). Thus,

epidemiological data associating childhood sweetened beverage intakes to weight change,

as well as data from the current and past studies associating childhood sweetened

beverage intakes to metabolic syndrome risk, suggests that this dietary factor may be an

important target for early lifestyle health promotion efforts.

Recently, the definition of metabolic syndrome and its clinical application has

been questioned, with a charge that the label "syndrome" offers no substantive clinical

utility (16). The critics contend that for a syndrome to have scientific force it must have

predictive utility greater than that offered by the individual components, must designate a

distinct underlying causal process, and must suggest a treatment strategy different from

merely treating the individual risk components (16). The present study does not

specifically address the clinical status of metabolic syndrome. To do so, higher risk for adverse health outcomes for the Higher MetS Risk group would need to be established. As this was a non-clinical sample of healthy, non-Hispanic, white 13-year-olds, the adverse health outcomes are not yet manifest. Concurrent associations between risk group membership and both higher body fat and BMI were seen, but whether this is reflective of a later morbidity remains to be tested in subsequent analyses using data from later points in development.

What this study does contribute is a novel approach to describing the clustering of the risk factors among a non-clinical sample of girls. This includes an estimate of the mean levels on the indicators associated with the subgroups of the girls in the study and a clear demonstration of developmental patterns showing consistent differences and a steady divergence from age 5 on measures of body weight and adiposity, which are associated with the clustering of the metabolic syndrome risk indicators. Additionally, this study was unique in that group means were estimated by the mixture modeling and not a priori considerations. Previous studies of metabolic syndrome in adolescents classify individuals based on modified adult values (13); in contrast, the current study allowed the data to determine profiles of metabolic syndrome risk based on the commonly used indicators.

Limitations of this study include a sample that is relatively small and homogeneous in both ethnicity and gender. Because a sample of non-Hispanic white girls was examined, results cannot be generalized to other ethnicities or to boys. Additionally, this study cannot currently assess future metabolic syndrome risk because data beyond age 13 have not yet been obtained. Data at future time points on the actual presence of

metabolic syndrome in this sample of adolescent girls are needed to assess the adequacy of the subgroups as indicators of risk, and to determine whether the three elevated risk groups (HDL-TG, Hypertension, and Higher MetS) will be associated with distinctly different patterns of comorbidities later in development.

Given the strong statistical support for the model, the similarities between the higher risk group in this model and the current conceptualization of metabolic syndrome in adolescents, and the various differences in the developmental precursors noted among the risk groups, this study provides support for a multi-faceted disease risk trajectory during adolescence. Additionally, one of the strengths of this study was the ability to utilize a rich, longitudinal dataset to examine developmental antecedents of risk subtypes. This study provided evidence that family history and persistent patterns of elevated sweetened beverage intake, accelerated weight gain and elevated fat mass accumulation during childhood were predictive of elevated risk for metabolic syndrome in early adolescence. These developmental antecedents of adolescent metabolic syndrome risk provide promising targets for preventive interventions.

Table 2.1: *Mean (s.d.) values for metabolic syndrome indicators in girls at age 13 by*

MetS risk group

	MetS Risk Group				MetS Criteria[1]
	Lower MetS (*n=62*)	HDL-TG (*n=36*)	Hypertension (*n=33*)	Higher MetS (*n=21*)	
Blood Glucose (mg/dL)	80.5 [a] (7.5)	81.7 [a] (7.2)	83.3 [a] (8.0)	84.0 [a] (9.1)	≥110
Triglycerides (mg/dL)	62.0 [a] (14.6)	120.3 [b] (27.9)	71.2 [a] (17.4)	147.9 [c] (65.0)	≥110
HDL (mg/dL)	53.5 [a] (11.0)	41.6 [b] (7.1)	49.4 [c] (9.7)	38.9 [b] (10.0)	≤40
Waist Circumference (cm)	73.3 [a] (6.9)	77.0 [b] (7.7)	83.1 [c] (8.9)	91.2 [d] (11.0)	≥ 90.7
Systolic Blood Pressure (mmHg)	110.4 [a] (9.7)	112.5 [a] (6.8)	130.0 [b] (8.3)	131.8 [b] (10.9)	≥118[2]
Diastolic Blood Pressure (mmHg)	61.3 [a] (5.3)	63.5 [b] (4.0)	73.0 [d] (5.6)	73.0 [d] (6.1)	≥76[2]

Note: Different superscripts across rows indicate significant differences among MetS

risk groups at $p<0.05$ in the Fisher's LSD comparison.

[1] Criteria from the Cook et al. (12) adaptation of The National Cholesterol Education

Program (Adult Treatment Panel III) definition for adolescents.

[2] 90th percentile for age and gender.

Table 2.2: *Percent of girls meeting criteria for individual metabolic syndrome components[1]*

	MetS Risk Group				Total
	Lower MetS	HDL-TG	Hypertension	Higher MetS	Sample
	(n=62)	(n=36)	(n=33)	(n=21)	(n=152)
Blood Glucose	0	0	0	0	0
Triglycerides	0	55.6	0	66.7	22.3
HDL	9.7	41.7	12.1	57.1	24.3
Waist Circumference	0	2.8	21.2	47.6	11.8
Systolic Blood Pressure	21.0	19.4	90.0	85.7	44.7
Diastolic Blood Pressure	0	0	24.2	42.9	11.2
Median # of Criteria Met	0	1	1	3	1

[1] Criteria from the Cook et al. (13) adaptation of The National Cholesterol Education Program (Adult Treatment Panel III) definition for adolescents.

Table 2.3: *Mean (s.d.) values for girls' anthropometric characteristics at age 13 by MetS risk group*

	MetS Risk Group			
	Lower MetS (*n*=62)	HDL-TG (*n*=36)	Hypertension (*n*=33)	Higher MetS (*n*=21)
Height (cm)	159.5 [a] (7.2)	160.8 [ab] (5.8)	162.5 [b] (6.8)	162.2 [ab] (4.8)
Weight (kg)	49.4 [a] (6.8)	52.6 [a] (8.2)	59.8 [b] (11.5)	64.3 [b] (15.4)
BMI (kg/m^2)	19.4 [a] (2.0)	20.3 [a] (2.8)	22.7 [b] (4.4)	24.4 [b] (5.7)
BMI percentile score	52.2 [a] (22.3)	60.0 [a] (25.0)	72.5 [b] (24.7)	75.8 [b] (26.8)
Fat mass (kg)	11.8 [a] (3.5)	12.8 [a] (3.9)	18.3 [b] (7.8)	19.6 [b] (8.9)
Percent Body Fat	24.2 [a] (4.8)	25.3 [a] (4.9)	30.1 [b] (7.4)	29.9 [b] (7.9)

Note: Different superscripts across rows indicate significant differences among MetS risk groups at $p<0.05$ in the Fisher's LSD comparison.
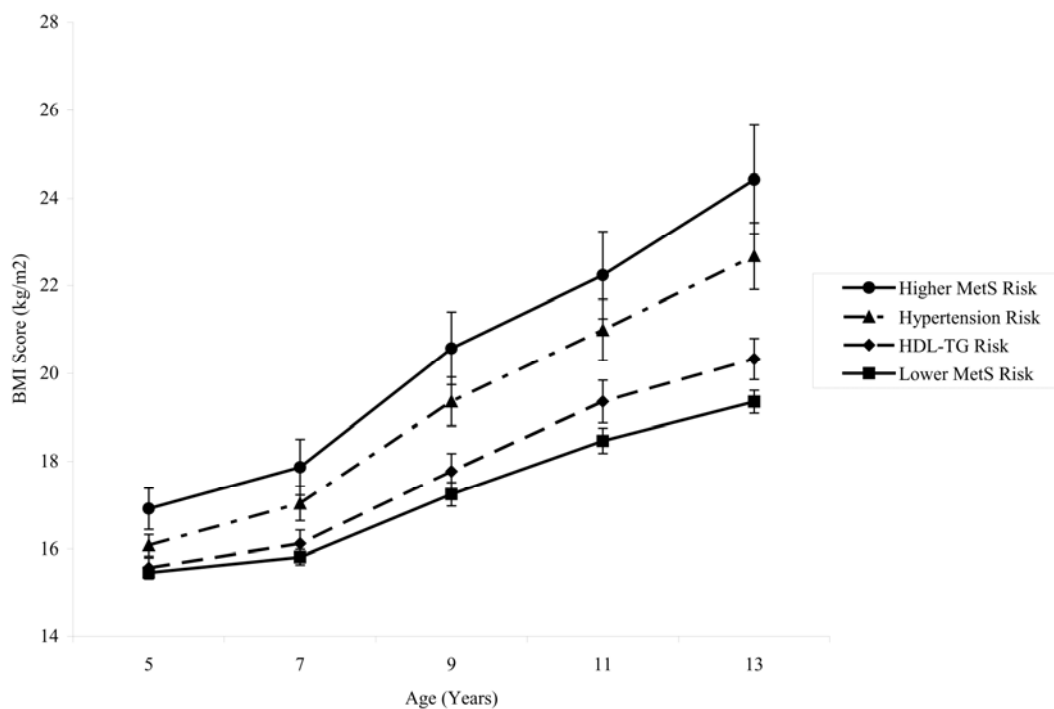
Figure 2.1: *BMI change from ages 5 to 13 years for MetS risk groups*

Plotted values are group mean ± standard error.

Between-group differences in intercept (BMI at age 5) were significant ($p<.05$).

The interaction of group membership with time was also significant ($p<.05$). The

higher MetS Risk group had a significantly steeper slope than the Lower MetS Risk

and HDL-TG Risk groups. The slope for BMI change in the Hypertension Risk group

was not significantly different from the Higher MetS Risk group.
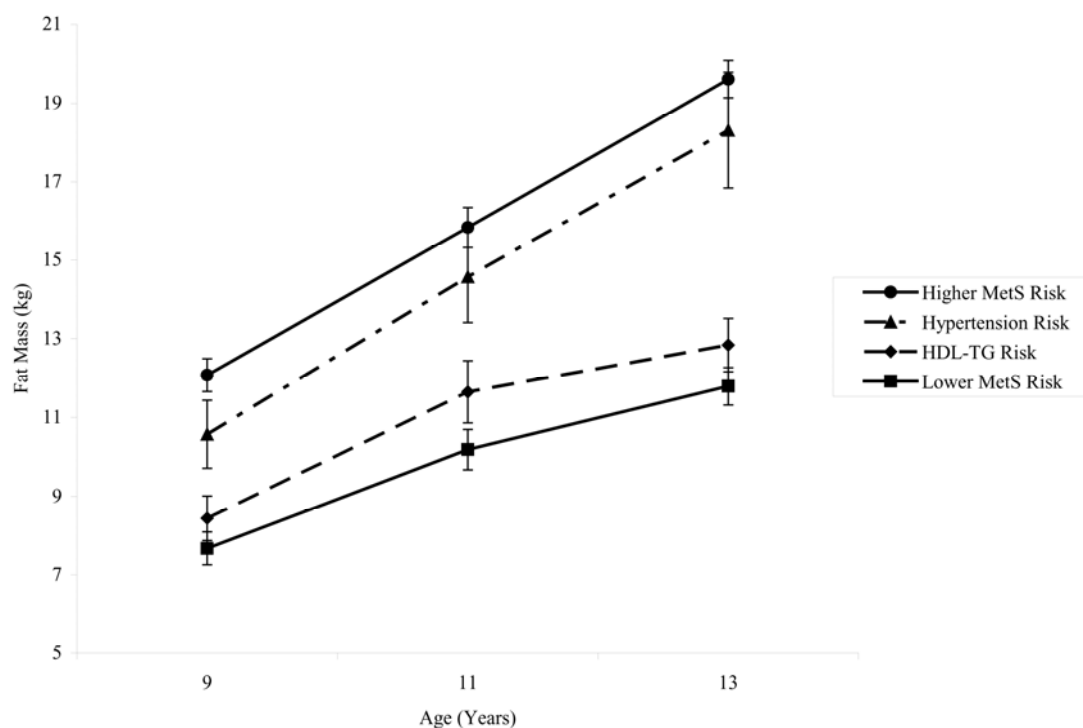
Figure 2.2: *Fat mass change from ages 9 to 13 years for MetS risk groups*

Plotted values are group means ± standard error.

Between-group differences in intercept (fat mass at age 9) were significant (p<.05).

The interaction of group membership with time was also significant at (p<.05). The

higher MetS risk group had a significantly steeper slope for fat mass change than the

Lower MetS Risk and HDL-TG Risk. The slope for fat mass change in the

Hypertension Risk group was not significantly different from the Higher MetS Risk
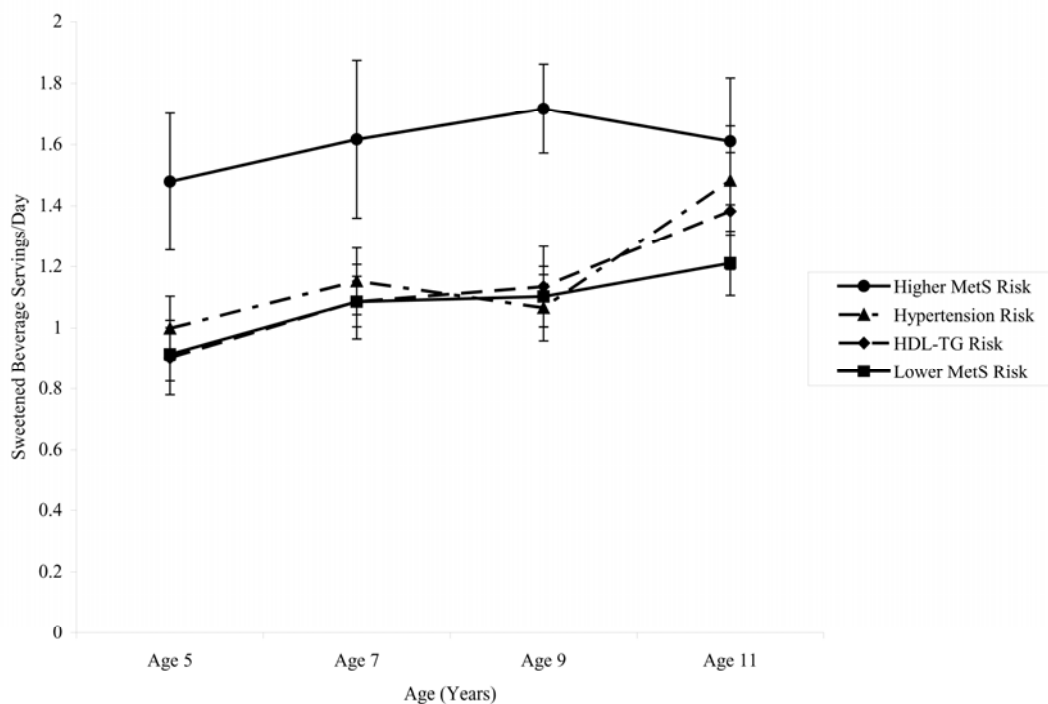
group.

Figure 2.3: *Mean servings per day of sweetened beverage from ages 5 to 11 years for MetS risk groups*

Plotted values are group means ± standard error.

Lower MetS Risk, HDL-TG Risk and Hypertension Risk were not significantly different at any age. Intake of the Higher MetS Risk group was significantly greater than all other groups at 5, 7, and 9 y of age (p<.001).

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Chapter 3

**DEVELOPMENTAL TRAJECTORIES OF CHILDHOOD BODY MASS INDEX
PREDICT ADOLESCENT METABOLIC HEALTH OUTCOMES**

ABSTRACT

Background: Increases in childhood obesity are linked to higher prevalence of metabolic health risk factors. We have few data on the developmental patterns of weight change during childhood underlying these secular trends.

Objective: To describe girls' body mass index (BMI) trajectories across childhood and adolescence.

Design: Participants were part of a longitudinal study of non-Hispanic, white girls (*n*=182) and their parents, assessed at daughters' ages 5, 7, 9, 11, 13 and 15 years. Height and weight were measured and used to calculate BMI. Dietary intake was assessed by 24-dietary recall interviews and cardiorespiratory fitness was assessed by the Progressive Aerobic Cardiovascular Endurance Run. Girls reported physical activity and television viewing patterns. Trained nurses assessed puberty by Tanner staging, and collected fasting venous blood draws, blood pressure, and waist circumference. Growth mixture models were used to model heterogeneity in girls' BMI trajectories over 10 years.

Results: Statistical support was strongest for four patterns of BMI change: 1) Upward Percentile Crossing (UPC; $n = 25$, 14%); 2) Delayed Downward Percentile Crossing (DDPC; $n = 37$, 20%); 3) 60th Percentile Tracking (60PT; $n = 52$, 29%) and 4) 50th Percentile Tracking (50PT; $n = 68$, 37%). Girls in the UPC group had mothers with the highest weight status compared to all other groups. Girls in the UPC and DDPC groups exhibited earlier pubertal timing compared to girls in the 60PT and 50PT groups. No longitudinal differences in dietary or activity patterns were found; however, significant dietary under-reporting bias was noted across ages 11-15 years and was higher for girls in

the UPC group. At age 15 years, girls in the UPC group had more metabolic risk factors than any other group.

Discussion: Through a novel approach for examining childhood weight status trajectories, several distinct patterns of BMI change were identified. Diet and physical activity patterns did not predict BMI trajectories, but girls with the most accelerated BMI trajectory had more overweight mothers, under-reported dietary intake to a greater degree and presented the worst metabolic outcomes. Future research is needed to explore factors other than self-reported diet and activity patterns that may distinguish among differing trajectories of childhood weight status.

INTRODUCTION

Increases in childhood obesity have been linked to higher prevalence of metabolic health risks during childhood and adolescence (1) and obesity during childhood is an important predictor of adult obesity, metabolic syndrome, type II diabetes and cardiovascular disease (2-4). We know little, however, about the developmental patterns of weight change *within children* that underlie secular increases in obesity or about the lifestyle correlates and metabolic consequences of interindividual differences in these trajectories. Understanding sources of heterogeneity in children's patterns of weight change is an important first step toward identification of the etiologic pathways of obesity onset and development (5).

Recent research has provided evidence for heterogeneity in the development of obesity by illustrating the presence of several distinct patterns of weight status across childhood and adolescence. Li and colleagues tracked a sample of children from age 2 to 12 and identified three patterns of weight status based on children's categorization as normal or overweight: 84% of children were consistently normal weight, 11% exhibited an early onset of overweight and 5% exhibited a later overweight onset (5). Using a similar categorical approach, Mustillo and colleagues tracked a sample of adolescents from age 9 to 16 and identified four patterns of weight status across this period: 73% of adolescents were never obese, 15% were chronically obese, 5% were obese at earlier ages, but normal weight at later ages and 7% were normal weight at earlier ages and obese at later ages (6).

These previous studies employed growth mixture modeling (GMM) techniques to model developmental trajectories of overweight and obesity (5, 6). In contrast to traditional growth curve modeling, where change over time is modeled as a single developmental trajectory, and individual variation around this trajectory is examined, GMM allows for fitting of several developmental trajectories based on the distinct profiles of growth present within a sample. Identification of these latent trajectory groups has the potential to illustrate which particular patterns of weight status change set children on the pathway to obesity (7). The present study also uses GMM, but adopts a slightly different approach by using a continuous indicator (body mass index [BMI]), rather than a categorical indicator (normal weight vs. overweight), to determine whether a set of distinct trajectories of weight status change can be identified. Additionally, as previous studies provided information on early life predictors (5) and psychiatric outcomes (6) associated with trajectories of overweight and obesity classification, the present study adds to these findings by examining specific modifiable behaviors and metabolic outcomes associated with trajectories of BMI change during childhood.

In sum, the objective of this study is to explore heterogeneity in developmental patterns of BMI change across childhood and adolescence in a non-clinical sample of non-Hispanic, white girls. This objective will be accomplished through three specific aims: 1) to assess whether distinctly different patterns of BMI change across ages 5 to 15 years exist within our sample; 2) to explore family demographic, dietary, physical activity, television viewing and fitness patterns that characterize the BMI trajectory groups identified; and 3) to describe the metabolic health outcomes at age 15 years associated with membership within each BMI trajectory group.

METHODS

Participants

Participants in a longitudinal study of the health and development of young girls were examined for this study. At study entry, the sample included 197 5-year old girls and their parents. Families were reassessed every 2 years (ages 7, 9, 11, 13, and 15 years); the final assessment included 166 families (84% of the original sample). Eligibility criteria for girls' participation at the time of recruitment included living with two biological parents, the absence of severe food allergies or chronic medical problems affecting food intake, and the absence of dietary restrictions involving animal products; the sample was not recruited based on parent or child weight status. Families were recruited for participation in the study using flyers and newspaper advertisements. Families with age-eligible female children within a 5-county radius also received mailings and follow-up phone calls (Metromail Inc.).

Measures

Background information. At study entry, parents reported family demographic characteristics, daughter birth weight and whether her daughter had been exclusively breastfed, exclusively formula fed, or fed a mix of breast milk and formula. Parents were also asked to retrospectively report their childhood weight status, with response options ranging from markedly underweight to markedly overweight.

Weight status and adiposity. For all participants, height and weight measurements were taken in the laboratory and were used to calculate BMI (weight [kg]/height [m]^2). Because girls were all the same age and gender, BMI, rather than BMI percentiles or z-scores, were used, following the suggestions of Cole and colleagues (8). Age- and sex-specific BMI percentiles were also calculated and used to determine the prevalence of overweight and obesity in girls in this sample. A BMI percentile ≥85th percentile was used to classify girls as overweight and ≥95th percentile was used to classify girls as obese. To estimate body composition (specifically, percent body fat), triceps and sub-scapular skinfold measurements were collected when girls were 5, 7, 9 and 11 years and Dual-Energy X-Ray Absorptiometry (DXA) scans were administered at 9, 11, 13 and 15 years. Trained nurses measured girls' waist circumference at age 15.

Pubertal Status and Timing. At ages 7, 9, 11 and 13 years, breast development was assessed by visual inspection by trained nurses using the Tanner rating scale (9).

Blood Assays. At age 15 years, girls fasted overnight for at least 11 hours prior to their laboratory visit and fasting blood samples were collected and processed for glucose, triglycerides, cholesterol and insulin. An index of insulin resistance was calculated according to the homeostasis assessment model (HOMA) formula (10): HOMA-IR=fasting insulin (μU/mL)*fasting glucose (mg/dL)/405.

Blood pressure. Seated blood pressure was measured after five minutes of quiet resting. On each occasion, two blood pressure readings were taken from the participant's right arm and an average was calculated.

Dietary Intake. At each visit, 24-hour recalls were conducted to assess girls' dietary intake and the Minnesota Nutrition Data System for Research (NDS-R) version

4.06_34 (2003) was used to calculate nutrient intakes. Girls provided three 24-hour recalls within a two to three week period following each laboratory visit. The three recalls included two weekdays and one weekend day and were averaged to provide an assessment of typical daily consumption.

To identify bias in the form of under- or over-reporting of energy intake, the method developed by Huang and colleagues was used (11, 12). A more detailed explanation of how bias in energy intake reporting is determined for our sample can be found elsewhere (see (13)). Briefly, gender and age group-specific ±1 SD cutoffs are created for reported energy intake (rEI) as a percent of predicted energy requirement (pER): (rEI/pER)*100 (11, 12, 14). pER is calculated for each individual girl based on the 2002 Dietary Reference Intakes (15) and the ±1 SD cutoffs were derived by propagating error variances for rEI, pER and estimated energy expenditure. rEI/pER*100 (hereon referred to as %rEI/pER) represents the extent to which reported energy intake matches predicted energy requirement; a value of 100% indicates perfect reporting accuracy, where reported energy intake exactly matches predicted energy requirement. Values below 100% illustrate under-reporting of energy intake and values over 100% indicate over-reporting of energy intake. Calculation of a ±1 SD cut-off for %rEI/pER plausibility provides an allowance for normal, daily variation in energy intake and energy expenditure.

Physical Activity Tendency. At ages 7, 9, 11 and 13 years the Children's Physical Activity (CPA) scale was used to assess girls' general tendency or inclination toward physical activity (e.g., "I would rather watch TV or play in the house than play outside" (16)). The CPA contains 15 items and uses a four-point response scale ranging from 1

(completely true) to 4 (completely false); scores were averaged to create an overall Tendency toward Physical Activities score.

Cardiorespiratory Fitness. Girls' cardiorespiratory fitness was measured at ages 9, 11 and 13 years using the Progressive Aerobic Cardiovascular Endurance Run (PACER) (17, 18). This progressive test provides an index of aerobic fitness as children run back and forth between markers spaced 20 m apart at a specified pace that progressively increases. Based on the number of laps completed and the total test time, girls are classified as being "fit" or "unfit" with respect to cardiorespiratory fitness level.

Television viewing. Starting at age 7 years, girls reported number of hours of typical television (TV) viewing on both school and non-school days.

Statistical Analyses

Descriptive information was generated for all variables of interest and each outcome variable was assessed for normality. To most accurately estimate girls' body fatness at ages 5 and 7 years, regression equations were created to describe the relationship between skinfold and DXA measurements at ages 9 and 11 years (when collection of these two measures overlapped). Based on previous research by Slaughter and colleagues (19), the equation created regressed DXA data on the sum and squared sum of triceps and sub-scapular skinfold measurements. The equation derived from this regression model was applied to the age 5 and 7 years skinfold data to calculate estimates of percent body fat at these assessment points.

Pubertal status from Tanner staging was regressed on chronological age and each girls' residual from this analysis was used to represent her pubertal timing (see (20)). This variable was calculated to illustrate how each girl's pubertal status related to the pubertal status of all other girls in the sample at a given age. Positive residuals indicate earlier pubertal maturation, negative residuals indicate later pubertal maturation and residuals close to zero indicate maturation at the sample norm for a given age.

BMI trajectory groups were identified by fitting latent growth mixture models (GMM) of repeated measures of BMI scores from ages 5, 7, 9, 11, 13 and 15 years using Mplus (version 4.1) (21) and AMOS (version 16.0). GMM is similar to conventional growth curve modeling in that it is a way to model developmental trajectories over time. However, GMM relaxes the single population assumption of conventional growth curve modeling to allow for differences across unobserved subgroups within a sample. We modeled a mixture of latent basis models (22), where basis coefficients for the growth factor are estimated from the data (as opposed to being assumed to be linear, quadratic etc.) Mplus estimates these models using maximum-likelihood, whereas the most recent version of AMOS implements a Bayesian algorithm. We found that the solutions in Mplus and AMOS were in close agreement, giving us added confidence in our results. In what follows we report the solution from Mplus.

Missing data were handled in Mplus with the assumption of missing at random; in our analysis, we excluded girls with less than three measurements, yielding a total of 182 girls. We estimated a mixture of latent basis models, with

loadings and factor means unconstrained across groups, but the variances and covariances of the factors were constrained to be equal across groups. One, two, three, four and five group solutions were examined and the number of latent groups (BMI trajectory groups) was determined by several model fit indices: (1) Akaike Information Criteria (AIC), (2) Bayesian Information Criteria, (3) Sample size adjusted BIC (SA-BIC), (4) Entropy, and (5) model interpretability. Smaller values for the AIC, BIC and SA-BIC indicate that a model with one less class (c = k-1) has to be rejected in favor of a model with at least k classes. After the best-fit solution was determined, girls were classified into their most probable groups based on the posterior probability.

Once BMI trajectory group membership was determined for each girl, the remainder of analyses were conducted using the SAS version 9.0 program (23). Mixed model analyses were used to examine differences among BMI trajectory groups on patterns of change for BMI and percent body fat across ages 5 to 15 years. Repeated Measures Analysis of Variance (ANOVA) with a Bonferroni correction for *post hoc* pair-wise comparisons was used to examine BMI trajectory group differences on patterns of puberty, dietary intake, physical activity tendencies and TV viewing across ages 5 to 15 years. Chi-Squared analyses were used to assess associations between BMI trajectory group membership and fitness levels at ages 7, 9, 11 and 13 years. ANOVA with a general linear model and Bonferroni corrected *post hoc* pair-wise comparisons was used to assess BMI trajectory group differences for family demographics and parent weight status at study entry and for metabolic health outcomes at age 15 years. These outcomes included values for waist circumference, total cholesterol, low-density lipoproteins

(LDL), high-density lipoproteins (HDL), triglycerides, glucose, insulin, HOMA-IR and blood pressure.

RESULTS

Background Information

At study entry, girls' mean age was 5.3 ± 0.3 years, mothers' mean age was 35.4 ± 6.0 years and father's mean age was 37.5 ± 4.1 years. Two-thirds of parents reported a level of education higher than a high school diploma. All fathers and two-thirds of mothers were employed. Approximately equal proportions of families reported incomes within the following ranges: 1) below \$35,000; 2) between \$35,000 and \$50,000; and 3) over \$50,000. Based on our definitions for overweight (BMI $\geq 85^{th}$ percentile and $<95^{th}$ percentile) and obese (BMI $\geq 95^{th}$ percentile) in children, 14% (n=25) of girls were overweight and 5% (n=9) were obese at study entry. Fifty-three percent (n=96) of mothers and 75% (n=136) of fathers were overweight (defined as BMI > 25) at study entry. For daughters and mothers, these proportions were comparable to national statistics at that time, however, for fathers, overweight prevalence was higher than that reported for national data (24).

Growth Mixture Model Solution

A four-group solution was determined to be the best-fit model by the maximum likelihood (MPlus) approach and this best-fit solution was verified by the Bayesian

(AMOS) approach. For illustrative purposes, patterns of BMI change for the four groups were plotted on the CDC growth chart for girls age 2 to 20 years (25) (Figure 3.1). The groups were labeled based on how each groups' BMI trajectory related to the CDC growth chart percentile curves: 1) the Upward Percentile Crossing (UPC) group ($n = 25$, 14% of the sample); 2) the Delayed Downward Percentile Crossing (DDPC) group ($n = 37$; 20% of the sample); 3) the 60th Percentile Tracking (60PT) group ($n = 52$, 29% of the sample); and 4) 50th Percentile Tracking (50PT) group ($n = 68$, 37%).

Girls in the UPC group exhibited a consistent pattern of upward BMI percentile crossing across childhood (Figure 3.1); girls in this group had higher weight status at age 5 years and by age 15 years were, on average, classified as obese. Girls in the DDPC group also exhibited an upward BMI percentile crossing between ages 5 and 9 years, but diverged from this pattern at age 11 years, exhibiting a downward BMI percentile-crossing trajectory across ages 11 to 15 years. Although, on average, girls in the DDPC group were classified as overweight at age 9 and 11 years, by age 15 years these girls had BMI scores at the 60th percentile, placing them within the normal weight status range. Girls in the 60PT group tracked along the 60th percentile for BMI and the 50PT group tracked along the 50th percentile for BMI.

Family Demographic and Parent Weight Status differences among BMI Trajectory Groups

Parents of girls in the UPC and DDPC group reported lower education levels compared to parents of girls in the 60PT and 50PT groups (Table 3.1). No differences were seen among groups for family income or daughter birth weight; however girls in the

UPC group were breastfed for a significantly shorter duration. Mothers of girls in the UPC group retrospectively reported significantly higher weight as a child compared to mothers of girls in the 60PT and 50PT groups, while fathers reported significantly higher weight as a child compared to all other groups. At study entry, BMI for mothers of girls in the UPC group was significantly higher than for mothers of all other groups and, on average, mothers of girls in the UPC group were obese. Fathers of girls in the UPC group were only heavier than fathers of girls in the 50PT group.

Patterns of Percent Body Fat Change across ages 5 to 15 years for the BMI Trajectory Groups

Changes in BMI trajectory groups' percent body fat across ages 5 to 15 years were consistent with the BMI growth mixture solution obtained (Figure 3.2). Girls in the UPC and DDPC groups had similar percent body fat trajectories across ages 5 to 9 years; however, between ages 11 and 15 years the levels of percent body fat for the DDPC group decreased substantially. Girls in the UPC and DDPC groups had significantly higher percent body fat at age 5 years compared to girls in the 50PT group and girls in the UPC, DDPC, and 60PT groups all had greater increases in percent body fat across ages 5 to 15 years compared to girls in the 50PT group.

Patterns of Pubertal Status and Timing across ages 7 to 13 years for the BMI trajectory Groups

A significant main effect of group membership ($F[3, 139] = 15.51$, $p<.001$) and interaction between group membership and time ($F[9, 417] = 6.63$, $p<.001$) were noted for pubertal status; similar effects were found for pubertal timing (main effect: $F[3,139] = 14.88$, $p<.001$; group*time: $F[9, 417] = 6.62$, $p<.001$) (Table 3.2). *Post hoc* analyses revealed that, overall, girls in the UPC group matured earliest, followed closely by girls in the DDPC group, and the 50PT group matured the latest.

Patterns of Dietary Intake across ages 5 to 15 years for the BMI Trajectory Groups

At all ages, girls in our sample, on average, met or were slightly below the USDA Food Pyramid Guide's recommendations for intakes from the grains (6 servings/day), vegetables (2.5 servings/day), meats (5 servings/day), fruits (1.5 servings/day), and dairy (3 servings/day) (26) (Table 3.3). No significant differences among BMI trajectory groups were noted for self-reported dietary energy intake, grains, vegetables, fruits, dairy, meats, fats, sweets, added sugars, sweetened beverages, and eating in front of the TV; Figure 3.3 shows average daily values for these variables plotted across ages 5 to 15 years for each BMI trajectory group.

Reporting accuracy declined for all girls across ages 5 to 15 years ($F [5, 770] = 93.69$, $p<.001$; Figure 3.4). Across ages 5 to 9 years, girls tended to over-report energy intake, but were still within the 1 SD range for plausibility. Girls tended to under-report energy intakes to a greater degree as they entered into adolescence; at ages 13 the

majority of BMI trajectory groups, and age 15 all of BMI trajectory groups, were below

the 1 SD cut-off for plausibility. At ages 5 to 9 years, no significant differences in

reporting accuracy were seen among BMI trajectory groups; however, at age 11 years

girls in the UPC, DDPC and 60PT groups underreported to a greater extent than girls in

the 50PT group and at ages 13 and 15 years, girls in the UPC group under-reported to a

greater extent than girls in the 60PT and 50PT groups (F [3, 154] = 5.48, p<.001).

Patterns of Physical Activity, Fitness and TV Viewing for the BMI Trajectory Groups

No differences were noted for tendencies toward physical activity scores or for

TV viewing patterns; total sample values for these variables are presented in Table 3.3.

Based on the percent of each group who are classified as "unfit" by on their performance

during the PACER procedure, BMI trajectory groups were significantly different on

fitness levels at ages 9, 11 and 13 (Figure 3.5). Higher proportions of girls in the UPC

and DDPC groups were classified as "unfit" at age 9 compared to girls in the 60PT and

50PT groups (χ^2=15.87, p<.01). However, at age 11 higher proportions of girls in UPC

group were classified as "unfit" compared to girls in the DDPC, 60PT and 50PT

(χ^2=19.68, p<.001) groups. At age 13 almost all girls in the UPC group and the majority

of girls in the DDPC and 60PT groups were classified as "unfit" compared to a lower

percentage of girls in the 50PT group (χ^2=32.67, p<.001).

Metabolic Outcomes at age 15 for the BMI Trajectory Groups

Because girls in the UPC group had significantly higher BMI at age 15 compared to girls in all other groups, we hypothesized their metabolic health risk profile would differ from other girls in the sample. Thus, ANOVAs were conducted with *a priori* contrasts specified between the UPC group and all other groups. These analyses revealed that girls in the UPC group had significantly higher values for waist circumference, total cholesterol, LDL cholesterol, fasting insulin, HOMA-IR, triglycerides and blood pressure at age 15 years compared to girls in the DDPC, 60PT, and 50PT groups (Table 3.4).

DISCUSSION

Using a data-driven growth mixture modeling approach, we identified four distinct patterns of BMI change across childhood and early adolescence in a non-clinical sample of non-Hispanic, white girls. The four growth trajectories identified in this study were described in reference to the CDC BMI-for-age percentile curves: 1) Upward Percentile Crossing (UPC), 2) Delayed Downward Percentile Crossing (DDPC), 3) 60th Percentile Crossing (60PT) and 4) 50th Percentile Crossing (50PT). Although these patterns represent groups, not individuals, the upward percentile crossing pattern of BMI change that characterized the UPC group illustrated a trajectory that, if seen for an individual child, would likely be identified in a clinical setting as reflecting accelerated growth (27), a growth pattern that places children at elevated risk for obesity (28). The problematic nature of the UPC group's pattern of growth was confirmed by the finding that girls in this group, on average, accrued higher levels of body fat, had the earliest

onset of puberty, and had the highest metabolic health risk compared to all other girls in

this sample. Mothers of UPC group girls had the highest weight status, with a mean BMI

greater than 30. No group differences were evidence for diet and activity patterns;

however, girls in the UPC group were most likely to under-report their dietary intake.

Despite accelerated increases in BMI from ages 5 to 9 years, a pattern similar to that of

girls in the UPC group, the DDPC groups' metabolic outcomes at 15 were similar to girls

who tracked along the 60th and 50th percentiles.

Traditionally, growth has been used as an indicator of current health status and a

predictor of later health outcomes for infants and young children (27). Studies examining

the predictive ability of patterns of growth in infants and young children typically focus

on accelerated growth, defined as a weight change score above one standard deviation for

a sample of interest, and have linked accelerated weight gain during critical periods (e.g.,

infancy, adiposity rebound) to later obesity and metabolic risk factors (28-31). In the

present study, we took a similar stance by examining the implications of growth

trajectories during later childhood and adolescence. We chose to identify trajectories,

rather than quantify change scores, and this approach provided evidence for differing

patterns of growth across childhood that were linked to different metabolic health

outcomes. The present study is similar to research examining rapid growth in infants and

young children in that it attempts to link particular parameters of growth to differences in

subsequent health outcomes. However, by using growth mixture modeling techniques we

illustrate that early accelerated growth (starting at age 5 years) was only associated with

metabolic risk factors in adolescence when it was part of an overall trajectory of

accelerated growth across childhood and adolescence.

Two methodological aspects of this study are key in illustrating the contribution of the growth mixture model solution presented here: 1) this method summarized individual differences in the developmental progression of weight status in the form of four distinct trajectories; and 2) the form of the trajectories was not imposed *ex ante* but rather emerged from the data. Categorical grouping of individuals, as was achieved through our growth mixture model analysis, is a way to provide a heuristic summary of a complex developmental process (23, 46, 47). Through use of a person-centered technique (growth mixture modeling) we were able to illustrate the natural heterogeneity in the development of weight status across childhood and adolescence. By combining this person-centered approach with variable-centered methods we were able to explore correlates of individual differences in growth trajectories (32).

Across ages 5 to 9 years, two groups of girls (the UPC and DDPC groups) exhibited upward BMI percentile crossing; at age 9 their growth trajectories began to diverge. These two groups differed in familial factors, as mothers of girls in the UPC group breastfed their daughters for a shorter duration and had higher weight status at study entry. Thus, shorter breastfeeding duration and higher maternal weight at age 5 predicted which of these girls exhibited accelerated BMI change in early childhood and continued to have an upward percentile crossing BMI trajectory through age 15 years. It is well established that children of obese parents have a higher risk of becoming obese than children of normal weight parents (33, 34) and, although familial similarity in weight status is partially attributable to genetics (35), characteristics of parents and family environments also have a substantial influence on childhood weight status (36, 37). Thus, differences between the UPC and DDPC groups for maternal weight status and

related familial factors indicate that girls with higher BMI at age 5 years who also have heavier mothers may be important targets for intervention efforts. Future research is needed to further assess features of the family environment (specifically, maternal behaviors and indicators of maternal-child interactions) that are associated with childhood weight status trajectories; identification of these factors may highlight modifiable behaviors that can be the focus of these intervention efforts.

Overall, pubertal onset for our sample was comparable to national data, which illustrates that, on average, pubertal onset for non-Hispanic, white girls occurs around 9 to 10 years of age (35). However, our pattern of findings illustrates that membership in trajectory groups with higher initial weight and fat gain (the UPC and DDPC groups) predicted earlier pubertal development. In this same sample of girls, Davison and colleagues showed that higher fat mass at age 5 predicted earlier pubertal onset at ages 7 and 9 years (38). The fact that we found similar trends by using a different approach to conceptualize both weight status and pubertal timing is an additional validation for the BMI trajectory groups yielded by our data-driven analysis. Early menarche is associated with higher metabolic risk later in life and the combination of higher weight status and earlier puberty may be creating a cumulatively higher risk for metabolic health problems for these children (39). Although this hypothesis was not explicitly tested by the current study, our patterns of findings suggest that the culmination of higher weight status and earlier pubertal onset exhibited by girls in the UPC group did put these girls at higher metabolic risk at age 15 years.

Despite significant differences in growth among BMI trajectory groups, we could not identify any consistent group differences in dietary intake, physical activity, or

television viewing that corresponded with patterns of BMI change or explained the divergence seen in the trajectory for girls in the DDPC group. We did note, however, that based on our estimation of reporting bias in our dietary recall data, the tendency for all girls to under-report dietary intake increased with time and that, at later ages, bias was higher for girls in the UPC group. It is well established that the presence of bias in self-reported dietary data can mask relations between diet and health status (18, 41-44). Thus, dietary differences among BMI change groups that would explain the divergent trajectory of girls in the DDPC group and the upward percentile crossing trajectory of the UPC group may not have been detected due to the greater tendencies toward under-reporting bias at later ages.

Although fitness is determined by a combination of genetic and training influences (40) and our analyses cannot determine causality, an objective measure of cardiorespiratory fitness showed a pattern over time which suggested that the downward BMI trajectory trend of the DDPC group beginning at age 9 was associated with increases in fitness, suggesting differences between groups in physical activity levels. One limitation of the study was the use of self reported measures of physical activity and television viewing, as it is possible that the subjective data we collected for activity and sedentary behavior patterns were not sensitive enough or were also susceptible to reporting bias, thus masking lifestyle changes leading differing BMI trajectories. (41). The failure of these traditional measures of dietary intake, physical activity and television viewing to identify specific, modifiable lifestyle patterns associated with children's weight status trajectories suggests future research should explore how alternative markers

of obesigenic lifestyle patterns, such as maladaptive eating styles or self-perceptions, relate to weight change patterns across childhood.

Based on the Cook and colleagues criteria for metabolic syndrome (42), only five girls in our sample would be classified as having the metabolic syndrome at age 15 years, and all but one of those girls were members of the UPC group (data not shown). Another limitation of the study is that we did not collect data on indicators of metabolic health at all assessments, so we cannot assess whether girls in the DDPC group had a metabolic profile across ages 5 to 9 years that was similar to the UPC group but diverged as their trajectory diverged from the UPC group from ages 9 to 15 years. It is possible that these girls were in good metabolic health across all of childhood. However, given the strong evidence that higher weight status is associated with higher risk metabolic health profiles (i.e., lower HDL, higher LDL, triglycerides, glucose and blood pressure) (10-12, 34), it is likely that the metabolic profiles for girls in the DDPC group during their higher weight years would have been concerning. Further research is needed to explore these patterns more thoroughly.

Our findings have several implications for future research efforts. First, we have shown that children do not follow a single growth trajectory during childhood and adolescence. Recognition of heterogeneity in patterns of growth is an important first step toward developing a comprehensive understanding of the development of obesity. Second, we have shown that consideration of an individual's growth trajectory is necessary, which provides support for the potential usefulness of tracking individual children's BMI in clinical settings. Although girls in the DDPC group had similar growth trajectories to girls in the UPC group initially, the downward deflection of this group's

BMI trajectory during later childhood was associated with health outcomes at age 15 years. If data had been analyzed at a single point in time, or even by comparing two points in time, this pattern may have been overlooked and associations between weight status and metabolic health may have been underestimated. Finally, because elevated maternal weight status at study entry predicted accelerated growth, future research is needed to further explore other maternal, familial, and broader environmental factors associated with heterogeneity in the developmental trajectory of childhood weight status to develop a comprehensive understanding of the etiology of obesity and to identify effective targets for obesity prevention efforts.

Table 3.1: *Means (s.d.) for BMI trajectory group differences on family demographics at study entry*

	BMI Trajectory Group			
	UPC	DDPC	60PT	50PT
	(14%, *n*=24)	(20%, *n*=37)	(29%, *n*=52)	(37%, *n*=68)
Family income[1]	1.9 [a]	2.1 [a]	2.0 [a]	2.1 [a]
	(0.8)	(0.9)	(0.9)	(1.0)
Average parent education (years)	13.6 [a]	14.3 [a]	15.0 [b]	15.1 [b]
	(1.3)	(2.1)	(2.3)	(2.2)
Breastfeeding duration (months)	4.4 [a]	5.8 [b]	5.7 [b]	5.6 [b]
	(2.9)	(1.7)	(2.1)	(1.8)
Birth weight (pounds)	7.5 [a]	7.2 [a]	7.7 [a]	7.7 [a]
	(1.2)	(1.2)	(1.2)	(1.2)
Maternal Retrospective Reported Weight as Child[2]	3.4 [a]	2.9 [ab]	2.7 [b]	2.9 [b]
	(0.9)	(0.9)	(0.8)	(0.9)
Paternal Retrospective Reported Weight as Child[2]	2.9 [ac]	3.0 [b]	2.9 [b]	2.5 [c]
	(0.9)	(1.0)	(1.0)	(0.8)
Maternal BMI	31.4 [a]	26.5 [b]	26.4 [b]	24.6 [b]
	(8.9)	(5.9)	(5.3)	(4.2)
Paternal BMI	29.4 [a]	29.0 [a]	28.4 [a]	26.8 [b]
	(3.6)	(4.6)	(4.1)	(3.8)

[1]Variable coding: 0=less than $20,000; 1=$20-35,000; 2=$35-50,000; 3=over $50,000

[2]Variable coding: 1=Markedly Underweight to 5=Markedly Overweight

[a,b] Means in a row followed by the same superscript were not significantly different at $p<.05$ in the Bonferroni *post hoc* test.

Table 3.2: *Pubertal status and timing across ages 7 to 13 years by BMI trajectory group*

	Total Sample	BMI Trajectory Group			
		UPC (14%, *n*=24)	DDPC (20%, *n*=37)	60PT (29%, *n*=52)	50PT (37%, *n*=68)
Pubertal Status: Tanner Staging Score					
Age 7	1.1	1.4 [a]	1.2 [ab]	1.1 [b]	1.0 [b]
	(0.4)	(0.5)	(0.4)	(0.2)	(0.2)
Age 9	1.8	2.2 [a]	2.5 [a]	1.8 [b]	1.2 [c]
	(0.8)	(0.7)	(0.6)	(0.7)	(0.5)
Age 11	2.2	2.5 [a]	2.5 [a]	2.4 [a]	1.9 [b]
	(0.8)	(0.8)	(0.7)	(0.7)	(0.7)
Age 13	3.8	3.8 [a]	3.7 [a]	3.9 [a]	3.7 [a]
	(0.9)	(0.9)	(0.8)	(0.9)	(0.9)
Pubertal Timing: Residual Tanner Staging Score					
Age 7		0.3 [a]	0.1 [ab]	-0.1 [bc]	-0.1 [c]
		(0.5)	(0.4)	(0.2)	(0.2)
Age 9		0.5 [a]	0.7 [a]	0.0 [b]	-0.6 [c]
		(0.7)	(0.6)	(0.7)	(0.5)
Age 11		0.3 [a]	0.2 [a]	0.1 [a]	-0.3 [b]
		(0.8)	(0.7)	(0.7)	(0.7)
Age 13		0.1 [a]	-0.1 [a]	0.1 [a]	-0.1 [a]
		(0.9)	(0.8)	(0.8)	(0.9)

Note. Values are means (s.d.) for the total sample and for each BMI trajectory group.

[a,b,c] Means in a row followed by the same superscript are not significantly different at

p<.05 in the Repeated Measures ANOVA Bonferroni corrected *post hoc* test.

Table 3.3: *Means (s.d.) or percent (n) for dietary and physical activity variables for the total sample*

	Age					
	5	7	9	11	13	15
Dietary Intake						
Energy (kcal/day)	1528.3 (337.0)	1707.6 (346.4)	1819.5 (341.5)	1836.8 (453.7)	1691.5 (467.3)	1579.0 (414.9)
Grains (servings/day)	5.3 (1.7)	6.0 (1.7)	6.5 (1.7)	6.3 (2.0)	6.0 (2.0)	5.7 (2.0)
Vegetables (servings/day)	1.2 (1.1)	1.3 (1.0)	1.4 (1.0)	1.5 (1.2)	1.5 (1.1)	1.8 (1.2)
Fruits (servings/day)	1.7 (1.4)	1.6 (1.4)	1.5 (1.3)	1.3 (1.4)	1.2 (1.3)	1.3 (1.5)
Dairy (servings/day)	2.5 (1.2)	2.7 (1.1)	2.7 (1.1)	2.8 (1.3)	2.6 (1.4)	2.2 (1.2)
Meats (servings/day)	2.8 (1.3)	3.1 (1.4)	3.4 (1.5)	3.4 (1.6)	3.5 (1.7)	3.6 (2.1)
Fats (servings/day)	2.2 (1.4)	2.4 (1.5)	2.7 (1.5)	2.7 (1.6)	2.6 (1.7)	2.6 (1.7)
Sweets (servings/day)	1.0 (1.2)	0.9 (0.9)	1.0 (1.1)	1.0 (1.3)	0.7 (0.9)	0.7 (1.2)
Added sugars (grams/day)	76.3 (29.8)	88.5 (33.5)	91.3 (35.9)	96.4 (41.9)	81.2 (41.6)	67.3 (29.4)
Sweetened beverages (servings/day)	0.6 (0.5)	0.7 (0.6)	0.8 (0.8)	1.1 (1.0)	1.0 (1.0)	0.8 (0.8)
# Meals Eaten in front of TV			2.0 (2.0)	3.3 (2.7)	3.1 (2.3)	2.8 (2.4)
% Meals Eaten in front of TV			14.2 (14.2)	23.5 (18.7)	24.2 (17.6)	24.6 (19.8)

Table 3.3 (continued)

			Age			
	5	7	9	11	13	15

Physical Activity, Fitness and Sedentary Behavior

	5	7	9	11	13	15
Tendencies toward Physical Activity		2.8 (0.4)	2.9 (0.4)	3.0 (0.4)	2.8 (0.4)	
Fitness Level (% classified as "Unhealthy")			42.7 % (*n*=76)	35.3 % (*n*=61)	52.8 % (*n*=86)	
TV Viewing (hours/day)		2.3 (0.4)	2.1 (1.0)	1.9 (0.9)	2.0 (0.9)	1.9 (1.1)

Note. The Child Physical Activity (CPA) questionnaire was not administered at the age 5 and 15 assessments. The Progressive Aerobic Cardiovascular Endurance Run (PACER) protocol was not administered at the age 5, 7 and 15 assessments. Television (TV) Viewing was assessed from age 7 onward and eating during TV viewing was assessed from age 9 onward.

Table 3.4: *Mean (s.d.) values for age 15 metabolic outcomes by BMI trajectory group*

		BMI Trajectory Group				
		UPC	DDPC	60PT	50PT	
	Total Sample	(14%, *n*=24)	(20%, *n*=37)	(29%, *n*=52)	(37%, *n*=68)	F-test value[1]
Waist circumference (cm)	78.9 (10.1)	97.6 (8.6)	77.3 (8.5)	76.7 (6.5)	74.8 (5.5)	179.05***
Total Cholesterol (mg/dL)	158.4 (25.3)	175.1 (24.7)	155.3 (27.8)	157.0 (21.8)	154.7 (25.1)	11.62***
HDL (mg/dL)	50.6 (10.4)	48.7 (11.1)	50.6 (11.9)	49.7 (7.9)	52.0 (11.1)	0.76
LDL (mg/dL)	90.6 (23.3)	105.8 (22.0)	87.6 (26.8)	91.6 (17.9)	85.3 (23.9)	11.30***
Fasting Glucose (mg/dL)	84.4 (11.0)	83.9 (5.7)	83.1 (8.1)	85.4 (16.6)	84.4 (7.4)	.05
Fasting Insulin ((μU/mL)	9.1 (4.6)	14.3 (7.1)	8.0 (4.1)	8.0 (2.5)	8.4 (3.1)	40.60***

Table 3.4 (continued)

	Total Sample	BMI Trajectory Group				F-test value[1]
		UPC	DDPC	60PT	50PT	
Insulin Resistance	1.9	3.0	1.7	1.8	1.8	26.91***
(HOMA-IR)	(1.1)	(1.5)	(1.0)	(0.9)	(0.7)	
Triglycerides	86.4	103.7	85.1	78.5	86.9	4.17*
(mg/dL)	(42.9)	(39.8)	(32.9)	(27.1)	(56.2)	
Systolic Blood	101.3	105.5	101.3	100.5	100.4	4.28*
Pressure (mmHg)	(10.0)	(12.6)	(10.6)	(9.2)	(9.0)	
Diastolic Blood	64.9	68.9	64.8	64.2	64.0	8.92**
Pressure (mmHg)	(6.7)	(7.8)	(6.1)	(6.2)	(6.6)	

[1] *A priori* contrasts were specified for the UPC group vs. the DDPC, 60PT and 50PT groups.

*p<.05, **p<.01, ***p<.001
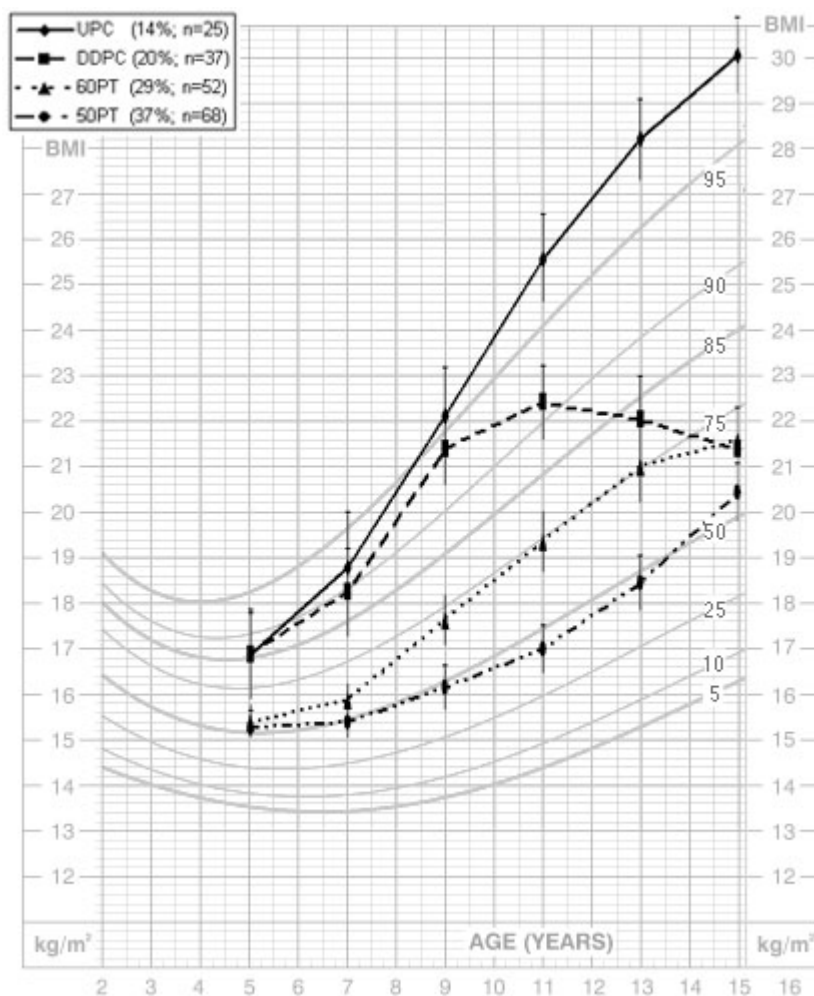
Figure 3.1: *Growth mixture model solution of BMI trajectory groups plotted on the CDC growth reference chart for girls age 2 to 20 years*

Intercepts for the UPC and DDPC group trajectories were significantly higher than the 50PT group trajectory (p<.001); the intercept for the 60PT group trajectory was not significantly different than the 50PT group trajectory. Slopes for the UPC and DDPC group trajectories were significantly greater than for the 50PT group trajectory (p<.01), but the slope for the 60PT group trajectory was no different than that for the 50PT group trajectory.
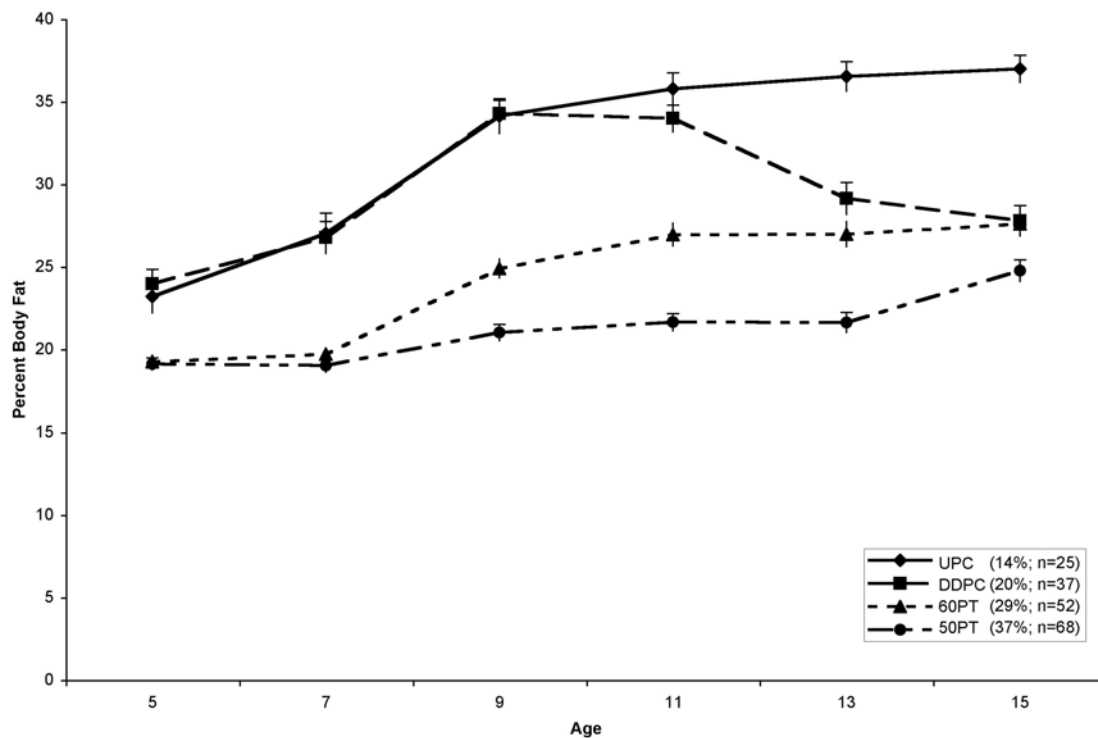
Figure 3.2: *Percent body fat across ages 5 to 15 years reflects patterns of BMI change for BMI trajectory groups*

Intercepts for the UPC and DDPC group percent body fat trajectories were

significantly different from the trajectory for the 50PT group (p<.001). The 60PT

group percent body fat trajectory intercept was not significantly different than that for

the 50PT group. Slopes for the UPC, DDPC, and 60PT group percent body fat

trajectories were significantly higher than that for the 50PT group (p<.001).
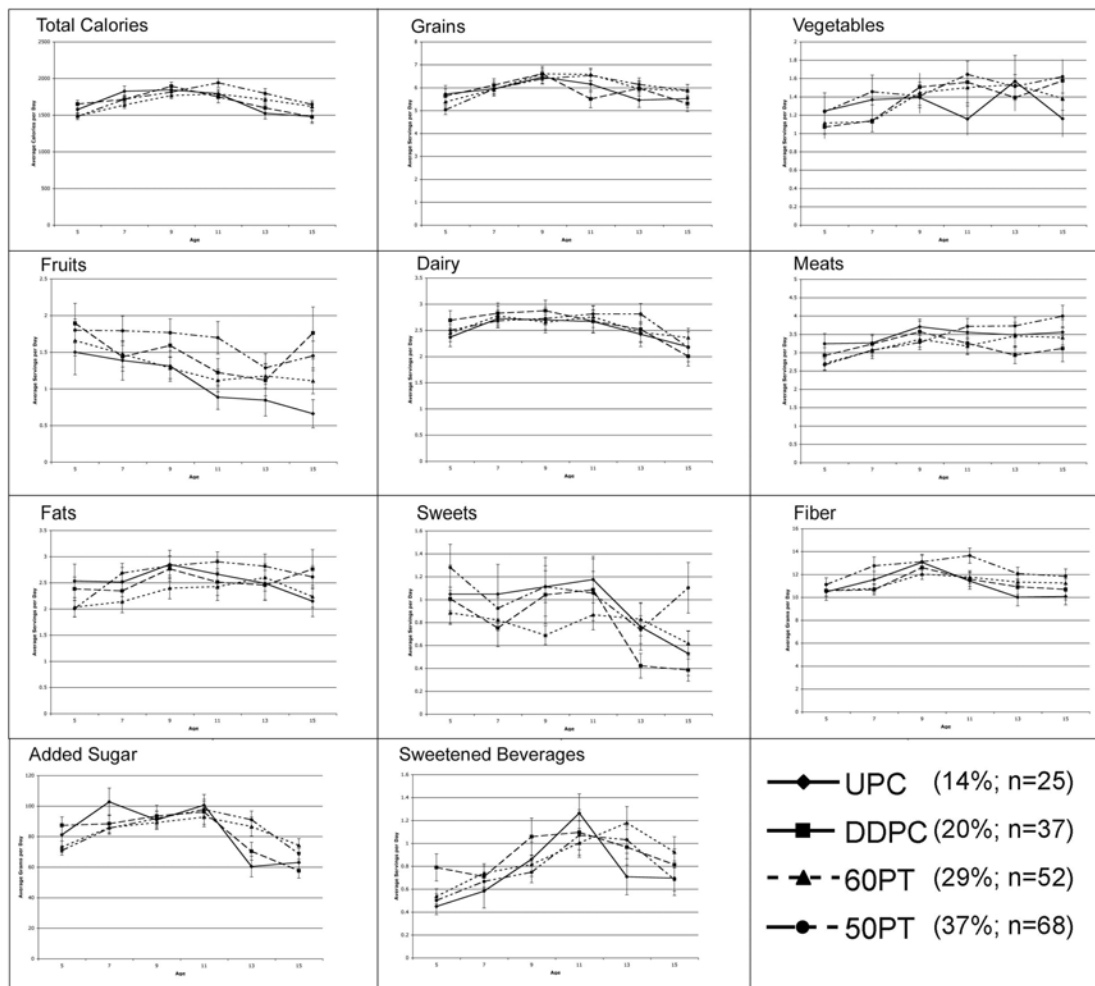
Figure 3.3: *Dietary intake patterns across ages 5 to 15 years did not differ among BMI trajectory groups*

No consistent, longitudinal differences among BMI trajectory groups were found for

dietary intake patterns.
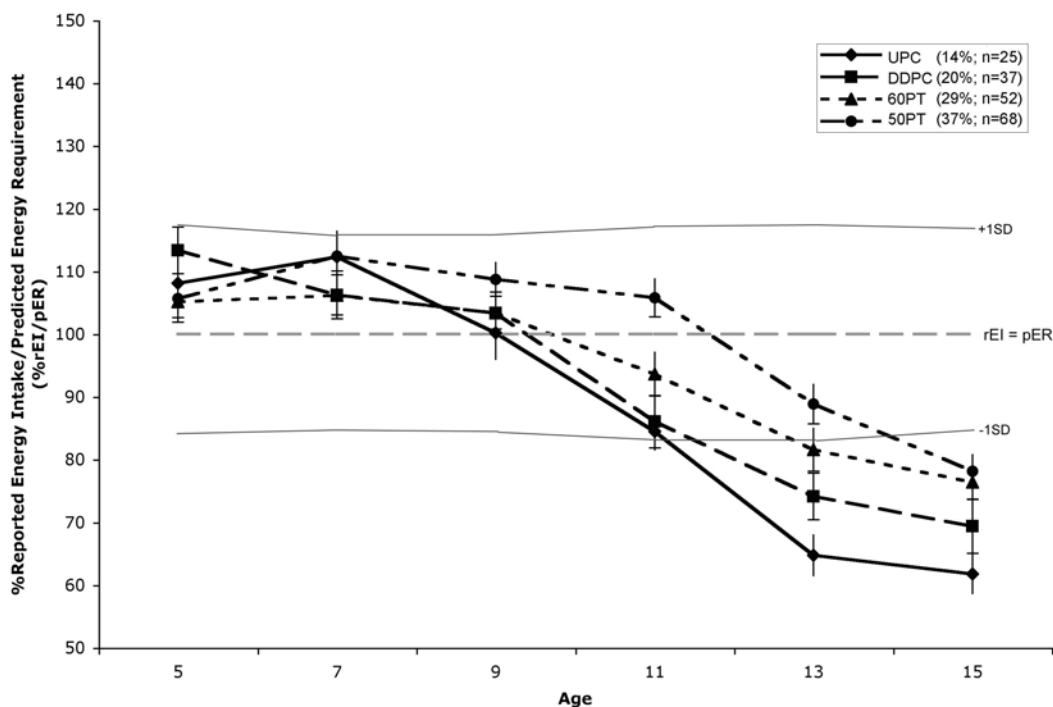
Figure 3.4: *Reporting accuracy declined for all BMI trajectory groups across ages 5 to 15 years*

rEI = reported energy intake, **pER** = predicted energy requirement, **SD** = standard deviation

Groups were not significantly different on reporting bias at ages 5, 7, or 9. At age 11, girls in the UPC, DDPC, and 60PT groups under-reported energy intake to a significantly greater extent than girls in the 50PT group (p<.001). At age 13 and 15, girls in the UPC group underreported to a significantly greater degree than girls in the 60PT and 50PT groups (p<.001).

Note: %rEI/pER=100 is the equivalent of perfect reporting accuracy. Values above the 1 standard deviation cut-off indicate over-reporting and values under the 1 standard deviation cut-off indicate under-reporting of energy intake.
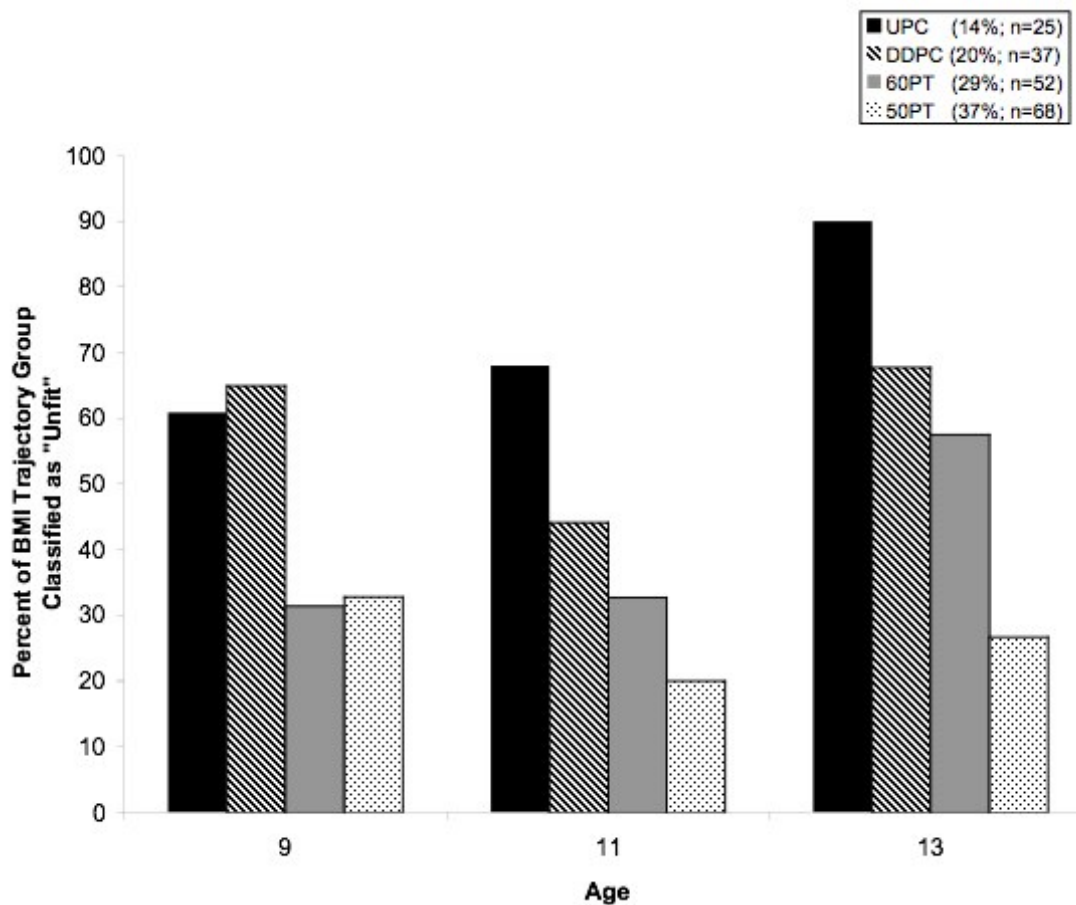
Figure 3.5: *Upward BMI percentile crossing associated with higher proportions of girls classified as "unfit" at ages 11 and 13 years*

Percent of girls classified as "unfit" by a progressive aerobic cardiovascular endurance run was not independent of BMI trajectory group membership (p<.01). At age 9 years, more girls in the UPC and DDPC groups were classified as unfit, but at ages 11 and 13 years, the UPC group had higher proportions of girls classified as unfit compared all other groups.

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Chapter 4

**MATERNAL CHARACTERISTICS, ATTITUDES, AND FEEDING PRACTICES
ARE ASSOCIATED WITH HETEROGENEITY IN DAUGHTERS' WEIGHT
STATUS TRAJECTORIES**

ABSTRACT

Background: Increases in the prevalence of childhood obesity have been well

documented, but information is needed about the developmental trajectories leading to

either overweight or normal weight, as well as the psychosocial characteristics of both

children and parents are associated with these trajectories.

Objective: To characterize the maternal and psychosocial correlates of heterogeneity in

girls' body mass index (BMI) trajectories across childhood and adolescence.

Design: Participants were part of a longitudinal study of non-Hispanic, white girls

(n=182) and their mothers, assessed biennially from 5 to 15 years. Girls' and mothers'

BMI scores were calculated from measured height and weight. Growth mixture model

techniques were used to model heterogeneity in girls' BMI trajectories over 10 years.

Questionnaires assessed mothers' and daughters' weight- and diet-related psychosocial

characteristics. Girls' disinhibited eating was assessed using a behavioral protocol to

measure eating in the absence of hunger.

Results: Statistical support was strongest for four BMI trajectories: (1) Upward Percentile

Crossing (UPC; n=25, 14%); (2) Delayed Downward Percentile Crossing (DDPC; n=37,

20%); (3) 60th Percentile Tracking (60PT; n=52, 29%) and (4) 50th Percentile Tracking

(50PT; n=68, 37%). Girls in the UPC group had mothers who had higher weight status

and higher levels of weight concern, perceived overweight and dietary disinhibition.

Mothers of girls in the UPC and DDPC group expressed more perceptions of and

concerns for their daughters' overweight status. During middle childhood, UPC group

mothers reported higher use of restrictive feeding practices, while DDPC group mothers

reported higher levels of monitoring of daughters intakes. By age 9, girls in the UPC

group reported higher levels of weight concern, dietary restraint and dieting, and lower

body esteem, but also exhibited greater tendencies toward eating in the absence of

hunger, compared to all other groups. Girls in the DDPC group had profiles of self-

perceptions, eating styles and dieting behaviors similar to the UPC group, but were less

extreme on all characteristics.

Discussion: These findings suggest future research should test intervention programs that

help overweight mothers foster healthy weight status in daughters through: 1) maternal

modeling of healthy diet- and weight-related attitudes and behaviors, 2) monitoring,

rather than restriction, of daughters' intakes, and 3) promotion of moderate levels of

dietary restraint and healthy dieting practices in daughters.

INTRODUCTION

Increases in the prevalence of overweight and obesity in all age groups have been well documented (1-3) and the association of obesity during childhood with increased risk for a wide array of undesirable physical and psychological health outcomes later in life has been established (4, 5). However, we lack information regarding the developmental trajectories that lead children to either become overweight or maintain a normal weight, and the characteristics of children, parents and parent-child interactions that are associated with these trajectories.

Children who become overweight do not follow a single, common pathway to this outcome. Some children are overweight for the entirety of their childhood, while other children's higher weight status may be limited to just early or later childhood (6-9). Recently, we used growth mixture modeling techniques, a novel approach for understanding the developmental trajectory of weight status change, to identify heterogeneity in trajectories of body mass index (BMI) across ages 5 to 15 years in a sample of girls (9). In contrast to traditional growth curve modeling, where change over time is modeled as a single developmental trajectory and individual variation around this trajectory is examined, growth mixture modeling allows for fitting of several developmental trajectories based on the possibility that distinct profiles of growth exist within a sample (10, 11).

In our previous study, we explored lifestyle patterns and metabolic outcomes associated with differing trajectories of BMI change, but also noted that maternal weight status was a significant predictor of these trajectories (9). Several early-life family demographic factors and maternal characteristics have also been identified by other research as predictors of developmental trajectories of overweight during childhood (6, 7). Also of interest are the psychosocial characteristics of mothers, children and mother-child interactions that are associated with heterogeneity in the developmental trajectory of childhood weight status.

Assessment of the family environment, and more specifically, maternal behaviors and indicators of mother-child interactions, may provide important insights into the etiology of children's diet- and weight-related behaviors and characteristics as they relate to the development of obesity. Mothers in particular have a strong influence on children's (especially daughters') diet- and weight-related psychosocial characteristics (12, 13). Mothers' concerns about their own weight status, dieting practices and eating styles are communicated both verbally and non-verbally to daughters (14, 15), and even at young ages, daughters exhibit similar dieting practices and attitudes about food and weight as are expressed by their mothers (16, 17). Thus, research to describe the family ecology within which girls' weight status trajectories develop should examine indicators of maternal modeling and maternal-daughter interaction focused on daughters' dietary intake and weight status.

The objective of the present study is to characterize maternal and daughter psychosocial characteristics associated with differing trajectories for girls' BMI change across childhood and adolescence. This objective will be accomplished through two specific aims: 1) to examine how maternal weight status and concern, eating styles, dieting practices, perceptions of daughter, and feeding practices are associated with girls' differing patterns of BMI change and 2) to examine how daughters' perceptions, eating styles, and dieting behaviors differ among girls with differing patterns of BMI change.

METHODS

Participants

Participants in a longitudinal study of the health and development of girls were examined for this study. At study entry, the sample included 197 5-year old girls and their mothers. Families were reassessed every 2 years (daughters' ages 7, 9, 11, 13, and 15 years); the final assessment included 166 families. Eligibility criteria for girls' participation at the time of recruitment included living with both biological parents, the absence of severe food allergies or chronic medical problems affecting food intake, and the absence of dietary restrictions involving animal products; the sample was not recruited based on parent or child weight status. Families were recruited for participation in the study using flyers and newspaper advertisements. Families with age-eligible female children within a 5-county radius also received mailings and follow-up phone calls (Metromail Inc.).

Maternal Measures

Background information. At study entry, mothers reported family demographic characteristics.

Weight status. At each visit, mothers' height and weight measurements were taken in the laboratory and were used to calculate Body Mass Index (BMI) scores (weight [kg]/height [m]^2). Based on the Centers for Disease Control and Prevention (CDC) reference criteria for adults, overweight was defined as a BMI $\geq$ 25 and obese was defined as a BMI $\geq$ 30 (18).

Weight Concerns. The Stanford Weight Concerns Scale was used to assess mothers' weight concerns (19). The Weight Concerns scale is a 5-item questionnaire that assesses fear of weight gain, worry about weight and body shape, the importance of weight, diet history, and perceived fatness. Higher scores indicate higher concerns for one's own weight status. The questionnaire was administered at five time points, daughters' ages 5, 7, 9, 11 and 15 years.

Restraint and Disinhibition. Mothers' restrained and disinhibited eating behaviors were assessed using the dietary restraint and disinhibition subscales from the Three Factor Eating Inventory developed by Stunkard and Messick (20). The dietary restraint subscale consists of 14 items that measure mothers' reported cognitive control of eating. The dietary disinhibition subscale consists of 16 items that measure mothers' tendency to eat in response to external factors such as the presence of food and emotional states. Higher scores indicate higher levels of dietary restraint and disinhibition. The

questionnaire was administered at five time points, daughters' ages 5, 7, 9, 11 and 15 years.

Dieting Practices. Mothers' dieting practices were assessed by the Weight Loss Behavior Scale (21). This scale was developed by French and colleagues and is intended to assess the different weight loss strategies engaged in by adults. The checklist consists of 24 items that are categorized into "healthy" and "unhealthy" dieting behaviors. Healthy dieting behaviors include: reducing caloric intake, eliminating snacks, increasing exercise, increasing fruit and vegetable intake, decreasing fat intake, eliminating sweets, reducing the amount of food consumed, changing the types of foods eaten, eating less meat, eating less high carbohydrate foods and eating low-calorie foods. Unhealthy dieting behaviors include: fasting, skipping meals, increasing cigarettes smoked, laxative use, diuretic use, appetite suppressant use, the use of diet pills, vomiting and liquid diets. Checklist items are summed to create total healthy and unhealthy dieting behavior use scores. This questionnaire was administered at all visits.

Child Feeding Practices and Attitudes. Maternal feeding practices and attitudes about child feeding were assessed by the Child Feeding Questionnaire (CFQ; (22)). This questionnaire contains 24 items that are divided into three attitudinal subscales: (1) perceived parent overweight status; and (2) perceived child overweight status; (3) concern for child overweight status or risk for becoming overweight; and four child feeding practices subscales: (1) restriction of child's eating; (2) pressuring child to eat; (3) monitoring of child eating; and (4) responsibility for child eating. Higher scores reflect higher perception or concern for overweight or higher use of specific feeding practices. The questionnaire was administered at daughters' ages 5, 7, 9, 11 and 13 years.

Maternal Encouragement of Daughters' Weight Loss. The Encouragement of Daughters' Weight Loss Scale was developed by our laboratory to measure the ways in which mothers encourage daughters to lose weight. Item examples include "Have you encouraged your daughter to exercise more in order to help her lose weight?" and "Have you ever put your daughter on a weight-loss diet?" Higher scores reflect higher levels of encouragement to lose weight. The questionnaire was administered at daughters' ages 9, 11 and 13 years.

Daughters' Measures

Weight status and adiposity. At each visit, daughters' height and weight measurements were taken in the laboratory and were used to calculate BMI. Because girls were all the same age and gender, BMI, rather than BMI percentiles or z-scores, were used in both the longitudinal and cross-sectional analyses, following the suggestions of Cole et al. (23). Age- and sex-specific BMI percentiles were also calculated and used to determine the prevalence of overweight and obesity in girls in this sample. A BMI percentile score above the 85th percentile was used to classify girls as overweight and above the 95th percentile was used to classify girls as obese.

Weight Concerns. For daughters, an amended version of the Stanford Weight Concerns Scale (19) assessed weight concerns. This questionnaire was administered at all visits.

Body Esteem. Girls' body dissatisfaction was assessed using the Body Esteem Questionnaire (24). This is a 24-item scale that assesses overall, nonspecific body esteem

(eg, "I like what I look like in pictures," "I'm proud of my body," and "I wish I were thinner"). Higher scores indicate higher levels of body esteem. This questionnaire was administered at all visits.

Perceived Parent Feeding Practices. Daughters' perceptions of maternal feeding practices and attitudes about child feeding were assessed by a child/adolescent version of the CFQ (22). This measure was adapted from the parent version of the CFQ and developed to assess girls' perceptions of the level of control that their mother exerts during feeding situations. For the purposes of these analyses, the Perceived Restriction and Perceived Pressure to Eat subscales were used. Higher scores on subscales indicate higher levels of perceived control of mothers by the daughter. The questionnaire was administered at five time points, daughters' age 5, 7, 9, 11, and 13 years.

Dutch Eating Behavior Questionnaire. Daughters' dietary restraint and disinhibition were measured using the original (for adults) Dutch Eating Behavior Questionnaire (DEBQ) created by Van Strien and colleagues (25). This scale was originally created for adults to measure dietary restraint and disinhibition, but was adapted for use with children. Higher scores indicate higher levels of restraint or disinhibition. This questionnaire was administered at all visits.

Eating in the Absence of Hunger. Girls' responsiveness to the presence of palatable foods in the absence of hunger was measured using a procedure developed in our laboratory, which has been previously described elsewhere (26). Briefly, approximately 20 minutes after eating lunch and reporting that they were full, girls were left alone in a room and were asked to taste and rate a set of palatable snack foods, such as potato chips, pretzels, chocolate bars and cookies. Each girl was told that she had a

few minutes alone and that after rating the foods she could eat as much or as little of the foods as she desired. Large amounts of the foods were available so that energy intake was not limited by availability. Other activities were available, including listening to music, looking at books and magazines and simple table games. Energy intake was calculated from gram weights obtained by pre- and post-weighing girls' food intake; manufacturers' information on energy content of foods was used to determine total energy intake, which was used as the indicator of eating in the absence of hunger. The variable of interest in this study was the proportion of energy consumed from the total energy available during the period in which the girl has free access to the snack foods. This procedure was administered at all visits.

Dieting Practices. An amended version of the French Weight Loss Behavior Scale (21) was used to assess daughters' dieting and dieting strategies. This measure was used to inquire about specific dieting practices of young girls, as well as where girls learn specific strategies. Girls were first asked whether they had ever dieted, then asked to indicate the specific dieting strategies they used. As noted above, the checklist consists of 24 items listing several "healthy" and "unhealthy" weight loss behaviors were summed to create total healthy and unhealthy dieting behavior use scores. This questionnaire was administered at all visits.

Statistical Analyses

Descriptive information was generated for all variables of interest and each outcome variable was assessed for normality. BMI trajectory groups were

identified by fitting latent growth mixture models (GMM) of repeated measures of BMI scores from ages 5, 7, 9, 11, 13 and 15 years using Mplus (version 4.1) (10) and AMOS (version 16.0); we described these analyses and reported these findings in more detail in a previous study (see (9)). Briefly, GMM is an extension of conventional growth curve modeling that allows for growth parameter (i.e., intercept and slope) differences across unobserved subgroups within a sample, rather than estimation of a single growth parameter for the entire sample. We modeled a mixture of latent basis models (27), where basis coefficients for the growth factor are estimated from the data (as opposed to being assumed to be linear, quadratic etc.) Missing data were handled in Mplus with the assumption of missing at random; in our analysis, we excluded girls with less than three measurements, yielding a total of 182 girls. The number of latent groups (BMI trajectory groups) was determined by comparison of model fit indices and model interpretability. After the best-fit solution was determined, each girl was classified into her most probable group based on the posterior probability, or probability of each girl belonging to each group.

Once BMI trajectory group membership was identified for each girl, the remainder of analyses were conducted using the SAS version 9.0 program (28). Repeated measures Analysis of Variance (ANOVA) was used to assess differences among BMI trajectory groups for maternal BMI across daughters' ages 5 to 15 years. For ease of interpretation and presentation, mothers' and daughters' psychosocial characteristics were organized into six categories: 1) *Maternal Diet- and Weight-Related Psychosocial Characteristics*, 2) *Maternal*

Perceptions of and Concerns for Daughters' Weight, 3) *Maternal Encouragement, Restriction and Monitoring during Feeding*, 4) *Daughters' Perceptions of Maternal Feeding, Weight Concerns and Body Esteem*, 5) *Daughters' Dietary Restraint, Disinhibition and Eating in the Absence of Hunger*, and 6) *Daughters' Dieting Practices*. Cross-sectional comparisons (at ages 5, 7, 9, 11, 13 and 15 years) of BMI trajectory groups on profiles for the above categories of parent and child psychosocial characteristics were made using Multivariate Analysis of Variance (MANOVA). Wilk's criterion was used to test overall significance of BMI trajectory group differences at each age. Fisher's exact test LSD correction was used for *post hoc* pair-wise comparisons to further explore differences among BMI trajectory groups on specific variables within the MANOVA profiles. Initial analyses revealed that the maternal responsibility over daughters' eating subscale did not vary among groups at any assessment point, thus this variable was excluded from the MANOVA analyses.

For illustrative purposes, MANOVA variables were standardized after all analyses had been conducted and separate plots of BMI trajectory group profiles across daughters ages 5 to 15 years were made for each category. These plots provide a presentation of the BMI trajectory group-specific profiles of indicator variables and how these indicator variable profiles change over time and differ among classes.

RESULTS

Background Information

At study entry, girls' mean age was 5.3 ± 0.3 years and mothers' mean age was 35.4 ± 6.0 years. Two-thirds of mothers reported a level of education higher than a high school diploma and approximately equal proportions of families reported incomes within the following ranges: 1) below \$35,000; 2) between \$35,000 and \$50,000; and 3) over \$50,000. Based on our definitions for overweight (BMI $\geq 85^{th}$ percentile and $<95^{th}$ percentile) and obese (BMI $\geq 95^{th}$ percentile) in children, 14% (*n*=35) of girls were overweight and 5% (*n*=9) were obese at study entry. Fifty-three percent (*n*=96) of mothers were overweight (defined as BMI > 25) at study entry. Overweight and obesity proportions for daughters and mothers were comparable to national statistics at the time of study entry (29).

BMI Trajectory Groups

A four-group solution was determined to be the best-fit model by the maximum likelihood (MPlus) and this best-fit solution was verified by the Bayesian (AMOS) approach. BMI trajectories across ages 5 to 15 years for the this solution were plotted on the CDC weight-for-age reference curves for girls age 2 to 20 years (30) (Figure 4.1). The groups were labeled based on their pattern of BMI change across ages 5 to 15 years in relation to the BMI percentile curves on the CDC reference chart: 1) the Upward Percentile Crossing (UPC) group (*n* = 25, 14%); 2) the Delayed Downward Percentile

Crossing (DDPC) group (n = 37; 20%); (3) the 60th Percentile Tracking (60PT) group (n = 52, 29%); and (4) 50th Percentile Tracking (50PT) group (n = 68, 37%). Girls in the UPC and DDPC groups had, on average, significantly higher BMI scores at study entry compared to girls in the 60PT and 50PT group. The UPC and DDPC groups had similar trajectories for BMI change across ages 5 to 9 years, but diverged at age 9 years in that the UPC group continued to exhibit upward BMI percentile crossing, while the DDPC group exhibited downward BMI percentile crossing. The 60PT and 50PT groups exhibited BMI trajectories that, on average, consistently tracked along the 60th or 50th percentile lines, respectively, across childhood and into adolescence.

Maternal Weight Status and Weight Change across Daughters' age 5 to 15 years by BMI Trajectory Group

Significant differences among BMI trajectory groups were noted for mothers' BMI across daughters' ages 5 to 15 years (Figure 4.2). Mothers of girls in the UPC group had significantly higher BMI at study entry (F[3, 153] = 7.88, p<.001) and across daughters' ages 5 to 15 years (F[3, 153] = 9.45, p<.001). No significant difference among BMI trajectory groups was found for maternal change in BMI across this 10 year period. On average, mothers of girls in the UPC group gained 6.1 kg, while gains of 6.6 kg, 5.7 kg and 4.8 kg were seen for mothers of girls in the DDPC, 60PT and 50PT groups, respectively.

Maternal Diet- and Weight-Related Psychosocial Characteristics by BMI Trajectory Group

Preliminary analyses revealed that maternal psychosocial characteristics were stable across daughters' ages 5 to 15 years, thus, average scores were created and a single MANOVA was conducted to assess differences among BMI trajectory groups on mean maternal psychosocial characteristics. A main effect for BMI trajectory group membership was noted, F(18, 490) = 1.62, p=.05. As shown in Table 4.1, *post hoc* comparisons revealed mothers of daughters in the UPC group had higher weight concerns than mothers of daughters in the DDPC and 50PT groups and higher perceived overweight compared to all other groups. Mothers of daughters in the UPC and 60PT had significantly higher dietary disinhibition scores than mothers of daughters in the 50PT group.

Maternal Perceptions of and Concerns for Daughters' Weight by BMI Trajectory Group

Significant differences among BMI trajectory groups were noted for maternal perceptions of and concerns for daughters' weight at all assessments: age 5: F(6, 352) = 4.56, p<.001; age 7: F(6, 354) = 7.51, p<.001; age 9: F(9, 426) = 12.17, p<.001; age 11: F(9, 402) = 11.94, p<.001; age 13: F(9, 385) = 11.70, p<.001. As shown in Figure 4.3, *post hoc* analyses revealed mothers of girls in the UPC and DDPC groups perceived their daughters to be significantly more overweight and were significantly more concerned about their daughters being overweight compared to the 60PT and 50PT groups across ages 5 to 11 years. At age 13, the UPC and DDPC were still significantly higher than the

60PT and 50PT groups on maternal perception of daughter overweight, but mothers of girls in the DDPC group reported significantly less concern for daughter overweight than girls in the UPC group. Additionally, at age 9, girls in the UPC and DDPC group were receiving more encouragement from mothers to lose weight than girls in the 60PT and 50PT groups, but at ages 11 and 13, girls in the UPC group were receiving the highest levels of encouragement to lose weight, followed by girls in the DDPC, then 60PT and 50PT groups.

Maternal Use of Encouragement, Restriction and Monitoring during Feeding by BMI Trajectory Group

Maternal feeding practices did not differ among BMI trajectory groups at age 5 or 13 years. A trend was noted at age 7: $F(9, 428) = 1.75$, p=.08 and maternal feeding practices did differ significantly at age 9: $F(9, 424) = 3.34$, p<.001 and age 11: $F(9, 402) = 4.01$, p<.001. As shown in Figure 4.4, *post hoc* analyses revealed that across ages 7 to 11 years, mothers of girls in the UPC and DDPC groups reported significantly lower pressuring of daughters to eat compared to mothers of girls in the 60PT and 50PT groups. At age 9, the deflection point for the DDPC group BMI trajectory, mothers of girls in the UPC group were using significantly more restrictive feeding practices while mothers of girls in the DDPC group reported significantly more monitoring of daughters' eating, compared to all other groups. At age 11, maternal restriction was still significantly higher for girls in the UPC group compared to all other groups, but group differences for monitoring were no longer significant.

Daughters' Perceptions of Maternal Feeding, Weight Concerns and Body Esteem by BMI Trajectory Group

Daughters' concerns and perceptions differed significantly at later ages: age 9: $F(12, 463) = 3.48$, p<.001; age 11: $F(12, 437)= 6.83$, p<.001; age 13: $F(12, 416) = 5.61$, p<.001; age 15: $F(12, 316) = 6.69$, p<.001. As shown in Figure 4.5, *post hoc* analyses revealed that across ages 11 to 13 years, girls in the UPC group perceived their mothers to be more restrictive of their dietary intake compared to all other groups. Across ages 9 to 15 years, girls in the UPC and DDPC groups had higher weight concerns compared to girls in the 60PT and 50PT groups. Girls in the UPC group had significantly lower body esteem across ages 9 to 15 years compared to all other groups, followed by girls in the DDPC group; by age 15 girls in the DDPC group did not differ from girls in the 60PT and 50PT groups on body esteem.

Daughters' Dietary Restraint, Disinhibition and Eating in the Absence of Hunger by BMI Trajectory Group

Starting at age 7, eating styles for BMI trajectory groups differed significantly: age 7: $F(9, 409) = 1.85$, p=.05; age 9: $F(9, 399) = 4.74$, p<.001; age 11: $F(9, 360) = 7.26$, p<.001; age 13: $F(9, 360) = 4.50$, p<.001; age 15: $F(9, 341) = 4.79$, p<.001. As shown in Figure 4.6, *post hoc* analyses revealed that across ages 7 to 15 years, girls in the UPC group had significantly greater levels of dietary restraint compared girls in the 60PT and 50PT groups. At ages 9, 13 and 15, girls in the DDPC group had greater levels of dietary restraint than girls in the 60PT and 50PT group and these levels were not significantly different from girls in the UPC group at ages 9 and 15. Levels of dietary disinhibition

only differed among groups at age 9 where girls in the 60PT group had higher reported

higher disinhibition compared to girls in the DDPC and 50PT group.

However, the behavioral measure of disinhibited eating, eating in the absence of

hunger, differed significantly among groups across ages 7 to 15 years. Across ages 7 to

15 years, girls in the UPC group exhibited significantly greater eating in the absence of

hunger, consuming more calories in the absence of hunger when presented with a wide-

array of palatable, energy-dense foods, compared to the 50PT group. At age 7, girls in the

DDPC and 60PT groups did not differ from girls in the UPC group and at age 9 only girls

in the DDPC group did not differ from girls in the UPC group. Across ages 11 to 15

years, the UPC group exhibited significantly higher levels of eating in the absence of

hunger than all other groups.

Daughters' Dieting Practices by BMI Trajectory Group

Dieting behaviors differed significantly among BMI trajectory groups at later

ages: age 9: $F(6, 354) = 7.59$, p<.001; age 11: $F(6, 336) = 9.17$, p<.001; age 13: $F(6, 320)$

$= 5.79$, p<.001; age 15: $F(6, 316) = 5.02$, p<.001. As shown in Figure 4.7, *post hoc*

analyses revealed that across ages 9 to 15 years, girls in the UPC group reported greater

use of healthy dieting behaviors compared to girls in all other groups. At ages 9 and 11

years, girls in the DDPC group reported higher use of healthy dieting behaviors compared

to girls in the 60PT and 50PT groups. With respect to unhealthy dieting behaviors, girls

in the UPC group reported higher use than all other groups across all ages. Girls in the

DDPC, 60PT and 50PT groups did not differ on unhealthy dieting at any age.

DISCUSSION

Using a data-driven methodology, we identified four patterns of BMI change within our sample. BMI trajectories were labeled based on the relation of each groups' pattern of BMI change to the age- and gender-specific BMI percentile curves on the CDC growth charts: 1) the Upward Percentile Crossing (UPC), 2) Delayed Downward Percentile Crossing (DDPC), 3) 60th Percentile Crossing (60PT), and 4) 50th Percentile Crossing (50PT), BMI trajectory groups. In a clinical setting, percentile crossing is an indicator of problematic growth for individual children (31); in particular, a child exhibiting an upward percentile pattern of BMI change across childhood would be identified as clinically at risk for later obesity and related co-morbidities. Although the four BMI trajectories presented in the current study represent groups, not individuals, identification of these trajectories illustrated the presence of a subset of girls within our sample that exhibited an upward percentile crossing BMI trajectory across childhood. Based on assessment of maternal and daughter psychosocial characteristics and interactions, this group of girls developed in a distinct ecology compared to other girls in our sample, characterized by a combination of higher maternal weight status, weight concern, and higher levels of maternal restriction of daughters' diet, and more encouragement of daughters' weight loss. Girls with differing BMI trajectories were no different on self-perceptions, eating styles and dieting behaviors at age 5, despite significant differences in weight status. However, differences emerged during later childhood and adolescence, possibly in response to patterns of BMI change and maternal influences, as girls exhibiting an upward percentile crossing BMI trajectory developed

higher weight concern, lower body esteem, greater dietary restraint and tendencies to eat in the absence of hunger, and higher use of healthy and unhealthy dieting practices, compared to other girls in the sample. Overall, these findings contribute to our understanding of the etiology and psychosocial correlates of obesity during childhood by illustrating how maternal and daughter characteristics associate with, likely as both a reaction to and result of, daughters' accelerated weight gain patterns across childhood and adolescence.

In a previous study, we were unable to detect associations between self-reported dietary intake and physical activity levels, and girls' BMI trajectories (9). We did note, however, that substantial bias was present in the self-reported dietary intake used to represent girls' habitual dietary intake, and this bias was greatest in the highest weight girls. Although it is possible that no association exists between girls' dietary intake and growth patterns, a more likely explanation is that the presence of bias in traditional measures of dietary intake and physical activity hindered our ability to detect associations between lifestyle patterns and weight status trajectories (32, 33). An approach we used to confront this issue was to use non-traditional markers of obesigenic lifestyle patterns, such as girls' specific combination of weight concerns, body esteem, dietary restraint, disinhibited eating, and dieting. Assessment of these more global measures of diet- and weight-related behavioral self-regulation (e.g., dietary restraint, dieting attempts, and eating in the absence of hunger) provided insights into the association between individual behaviors and weight status trajectories that we were not able to obtain with measures of self-reported dietary intake and physical activity patterns.

In the present study, similarities were seen between mothers' and daughters' patterns of weight status, weight concerns, dieting and eating styles. Mother-daughter similarities in diet- and weight-related psychosocial characteristics, and the influence of mothers on daughters' weight-related perceptions and concerns, have been well supported by past research (13, 14, 17, 34). For example, as early as age 5, daughters' knowledge of and ideas about dieting are related to maternal dieting practices and attitudes about weight and appearance (16), suggesting that daughters are attuned to maternal behaviors, regardless of whether or not mothers are intentionally attempting to influence daughters' behaviors or development. Thus, in the present study, maternal modeling of overweight, overweight perceptions, weight concern and disinhibited eating may have been an important factor influencing the concerns, self-evaluations, eating styles and dieting practices that emerged in daughters in the UPC group.

A more direct, and typically intended, way maternal behaviors are associated with daughters' eating and weight status is through the specific feeding practices mothers employ. Parent feeding practices (i.e. restriction (35), pressure to eat (36), and monitoring (37)) do causally influence children's eating behaviors (see (38)), but observational evidence has also shown that parent feeding practices are also exhibited in response to child characteristics (39). In the present study, mothers of girls in the UPC group consistently reported higher perception of and concern for daughters' overweight, likely in response to the higher weight status and accelerated BMI change seen for these girls. Our pattern of findings reveals that these mothers acted on these perceptions and concerns by restricting daughters' access to foods and encouraging their daughters to lose weight. These strategies were not effective in stopping their daughters' weight change

patterns, as evidenced by the finding that girls in the UPC group maintained an upward percentile crossing pattern of BMI change across childhood. Girls in the UPC group also developed greater tendencies toward eating in the absence of hunger across childhood. Both experimental and observational studies have consistently shown that restrictive feeding of children promotes over-consumption of restricted foods when available in the absence of parental controls, and is related to greater weight gain across childhood (26, 35, 40, 41). Thus, although maternal restriction may have also been in response to daughters' problems with self regulating their own intake, the tendency to eat in the absence of hunger that characterized girls in the UPC group may have developed in response to maternal use of restrictive feeding practices and further contributed to the accelerated BMI change patterns seen for this group.

In contrast, findings for the DDPC group provide support for previous research showing parental monitoring of children's intake is an effective way to help children make healthier dietary choices (37). A key difference between monitoring and restriction is the element of control: monitoring implies that mothers are aware of what daughters are eating, but are not necessarily restricting or attempting to control their intake of particular foods. Experimental data has shown that maternal monitoring elicits more desirable child reactions and food selection behaviors than maternal restriction (35, 37). Similar to mothers of girls in the UPC group, mothers of girls in the DDPC group had higher perceptions of and concerns for daughter overweight during early and middle childhood. These mothers, however, responded to these perceptions and concerns by monitoring daughters' intake and using more moderate encouragement of daughter weight loss. Although causality cannot be determined by the present study, the co-

occurrence of maternal monitoring of daughters intake and the emergence of daughters' downward percentile crossing BMI trajectory suggests that mothers of girls in the DDPC group may have chosen a more effective strategy for helping daughters achieve a healthy weight status in middle childhood.

The above associations between maternal feeding practices and attitudes and daughters' eating behaviors and weight status outcomes provide an illustration of and confirmation for Costanzo and Woody's theory of domain specific parenting (42). Costanzo and Woody proposed that parents tailor parenting practices to children based on several factors: (1) parental concerns for him- or herself, (2) parental concerns for child risk, and (3) parental perceptions of child risk (42). With respect to the development of children's eating behaviors and weight status outcomes, parents modulate feeding practices based on: (1) parental investment in weight and appearance, (2) parental perceptions of the child's own health status and eating behaviors and (3) parental perceptions of the child's risk for overweight or related comorbidities. Based on these ideas, Costanzo and Woody asserted that parents, or as illustrated by the present study, mothers of girls in the UPC group, who have issues with their own eating patterns and weight status tend to make more attempts to control their children's intake and weight.

Although tailoring of parenting practices to children's needs has potential to be beneficial for a child's development, Costanzo and Woody assert, and the present study illustrates, that when these well-intentioned parenting practices are over-controlling they hinder children's ability to develop their own self-regulatory abilities and may lead children to develop maladaptive eating styles (e.g., higher levels of dietary restraint, eating in the absence of hunger and dieting), self-perceptions (e.g., higher weight

concerns and lower body esteem) and weight status (e.g., an upward percentile crossing BMI trajectory).

In adults, moderate vigilance of weight status has been shown to be an effective weight maintenance strategy (43). For individuals who are of higher weight status, moderate levels of weight concern, dietary restraint and healthy dieting behaviors may help with achievement of a healthier weight status and is associated with long-term weight maintenance (44). Girls in the DDPC group showed a similar pattern of behaviors. Girls in this group were concerned with their weight status from early on and this concern persisted into adolescence. Additionally, girls in the DDPC group exhibited higher levels of restraint than the 60PT and 50PT groups, but this level was not consistently equal to that of the UPC group and was not accompanied by higher levels of disinhibited eating, as was seen in the UPC group. Girls in the DDPC groups also exhibited moderate levels of healthy dieting behaviors at ages 9 and 11. Taken together, this suggests that part of the downward trend in the BMI trajectories for girls in the DDPC group may have been attributable to their moderate concern, dietary vigilance, and dieting behavior; the presence of these behaviors in these girls was not coupled with high disinhibition and eating in the absence of hunger or low body esteem, which suggests that these behaviors were a healthy reaction to higher weight status and not part of an axis of disordered eating and body-esteem issues.

The main objective of this study was to characterize the maternal and daughter psychosocial qualities that distinguished among four trajectories of childhood BMI change. A limitation of the present study is that direction of influence and causality cannot be determined by the findings presented here and many of the differences noted

among BMI trajectory groups could reflect either mothers' or daughters' *reactions* to daughters' weight status or mothers' or daughters' *influences* on daughters' weight status. However, while our study design does not allow us to determine causality, many studies in this field have illustrated that associations between parent and child diet- and weight-related characteristics are characterized by bidirectional influences (38), which makes causality difficult to determine with any study design. Despite this limitation, the present research has provided a comprehensive, longitudinal examination of how maternal characteristics, behaviors and interactions with daughters differ among girls with differing patterns of BMI change, and how these relationships may change over time. Additionally, our results further illustrate that the development of childhood obesity is complex: girls do not follow a single pathway toward normal weight or overweight outcomes and the psychosocial influences associated with these pathways are multifactorial.

To achieve the ultimate goal of understanding the characteristics of both children and families that are associated with the etiology and development of childhood weight status trajectories, future research should further explore how the familial, maternal and daughter characteristics identified here as significant predictors of BMI change trajectories are causally related to the etiology and development of obesity across childhood and adolescence. Causal evidence supporting this data would indicate that overweight mothers should be educated about fostering healthy weight status in higher weight daughters through: 1) maternal modeling of healthy diet- and weight-related attitudes and behaviors, 2) monitoring, rather than restriction, of daughters' intakes, and

3) promotion of moderate levels of dietary restraint and healthy dieting practices in

daughters.

Table 4.1: *Means (s.d.) for BMI trajectory group differences on maternal diet- and weight-related psychosocial characteristics*

	BMI Trajectory Group				
	UPC	DDPC	60PT	50PT	
	(14%, *n*=24)	(20%, *n*=37)	(29%, *n*=52)	(37%, *n*=68)	F-, p-value
Weight	2.0^a	1.6^b	1.7^{ab}	1.5^b	3.48, p<.05
concerns	(0.6)	(0.7)	(0.7)	(0.6)	
Perceived	3.5^a	3.2^b	3.0^b	3.1^b	5.46, p<.01
Overweight	(0.4)	(0.6)	(0.5)	(0.4)	
Dietary	9.2^a	8.0^a	8.7^a	7.4^a	1.55, p>.05
Restraint	(4.4)	(4.3)	(4.2)	(4.0)	
Dietary	8.3^a	6.8^{ab}	7.1^a	5.7^b	4.46, p<.01
Disinhibition	(3.4)	(3.5)	(3.4)	(3.1)	
Healthy	5.4^a	4.5^a	4.8^a	4.3^a	1.56, p>.05
Dieting	(2.3)	(2.6)	(2.3)	(2.2)	
Unhealthy	1.1^a	0.9^a	0.9^a	0.7^a	0.98, p>.05
Dieting	(1.0)	(1.0)	(1.0)	(0.9)	

Note: For mothers, all of the above variables were highly stable over time. Thus, for mothers only, these values presented are averages for each variable across daughters' age 5-15 years.

[a,b] Means in a row followed by the same superscript were not significantly different at p<.05 in the Fisher's LSD *post hoc* test.
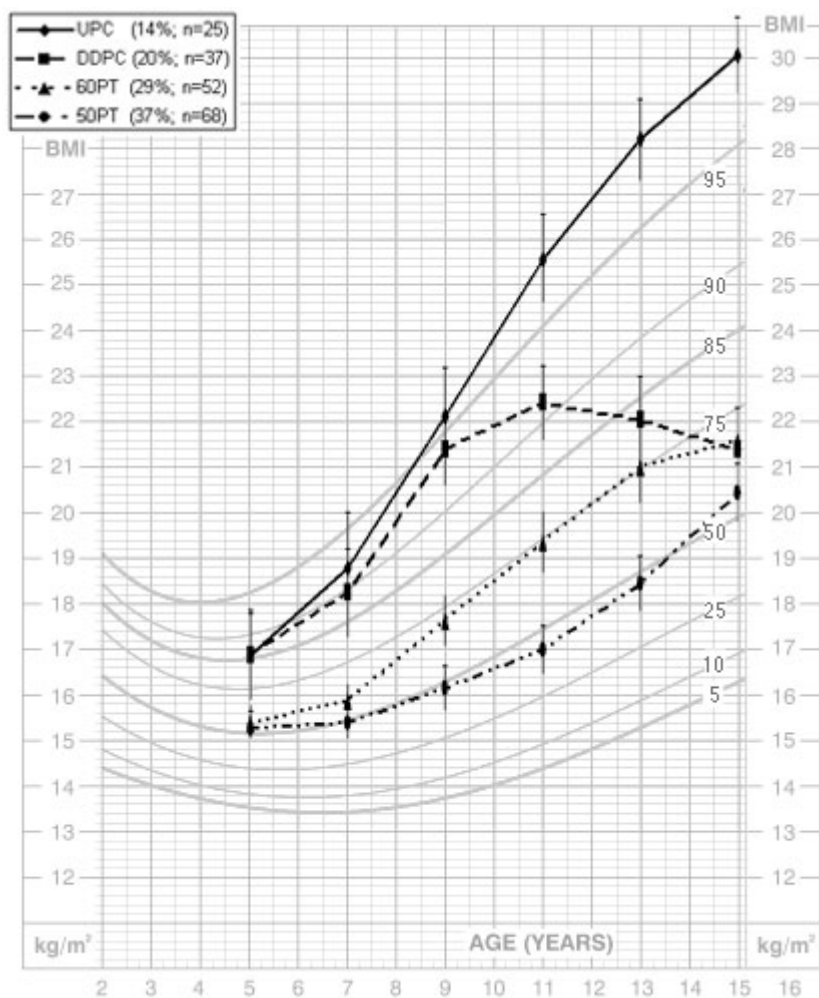
Figure 4.1: *Growth mixture model solution for BMI trajectory groups plotted on the CDC Growth Reference Chart for Girls 2 to 20 years*

Intercepts for the UPC and DDPC group trajectories were significantly higher than

the 50PT group trajectory (p<.001); the intercept for the 60PT group trajectory was

not significantly different than the 50PT group trajectory. Slopes for the UPC and

DDPC group trajectories were significantly greater than for the 50PT group trajectory

(p<.01), but the slope for the 60PT group trajectory was no different than that for the
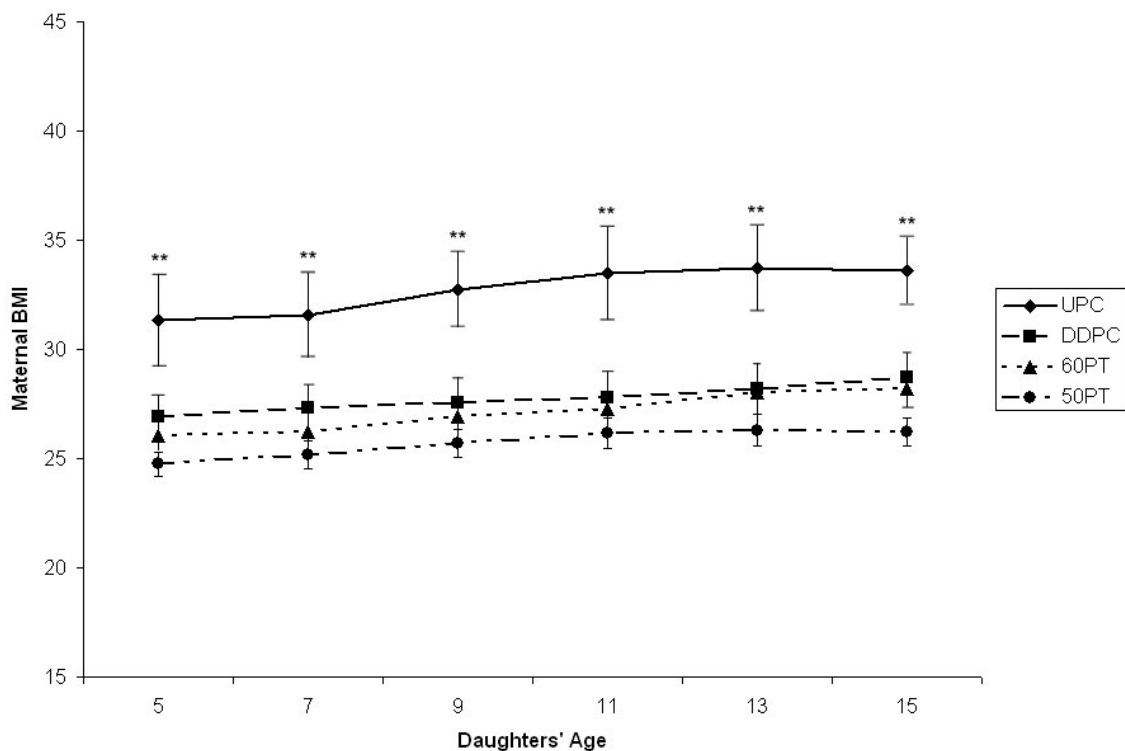
50PT group trajectory.

Figure 4.2: *Maternal weight status and change in weight status across daughters' age 5 to 15 years by BMI trajectory group*

Maternal BMI was significantly greater for girls in the UPC group at study entry (F[3, 153] = 7.88, p<.001) and across ages 5 to 15 years (F[3, 153] = 9.45, p<.001), compared to all other groups. Differences among BMI trajectory groups for maternal BMI change across this period were not significant.

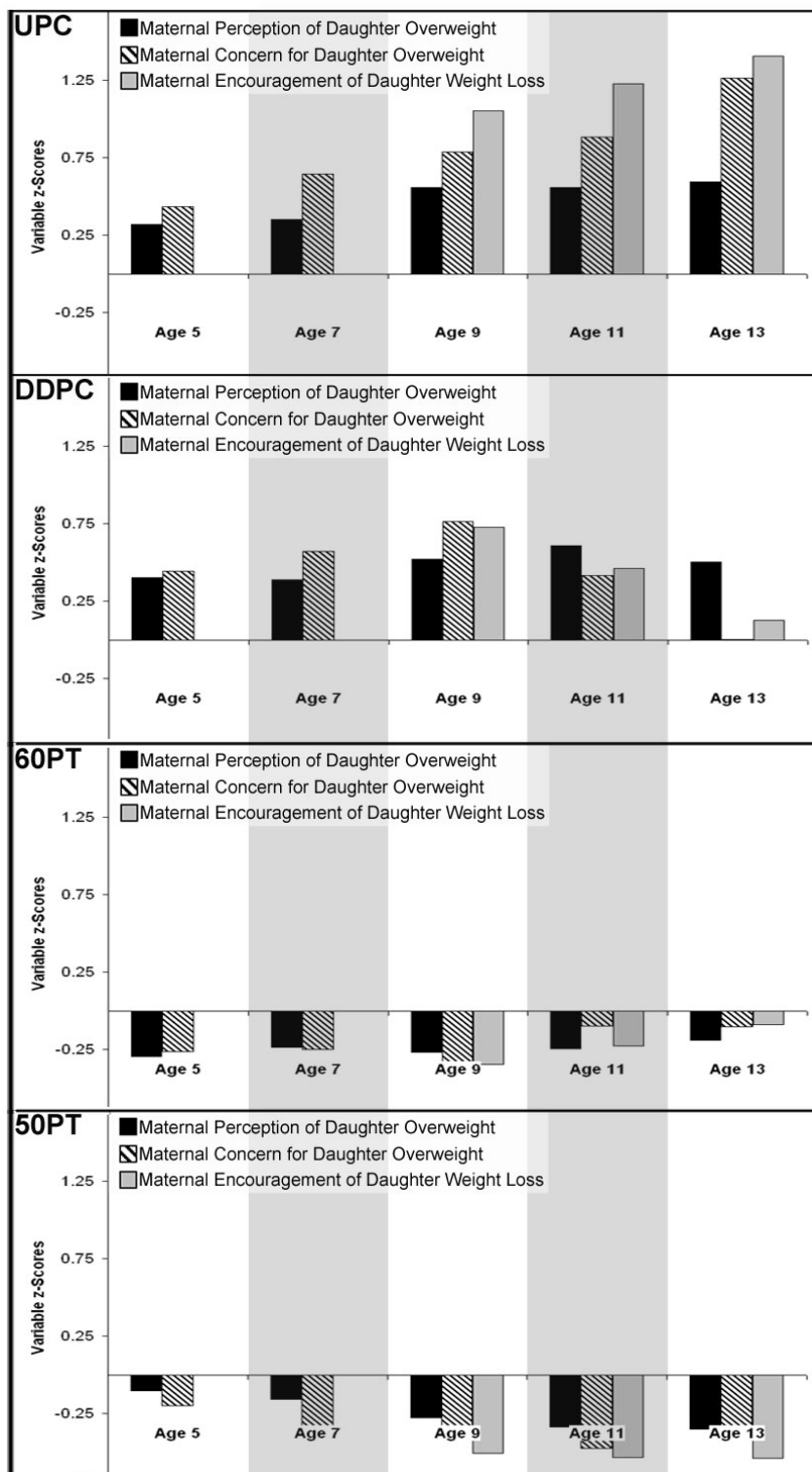Figure 4.3: *Maternal perceptions of and concerns for daughters' weight across ages 5 to 13 years by BMI trajectory group*

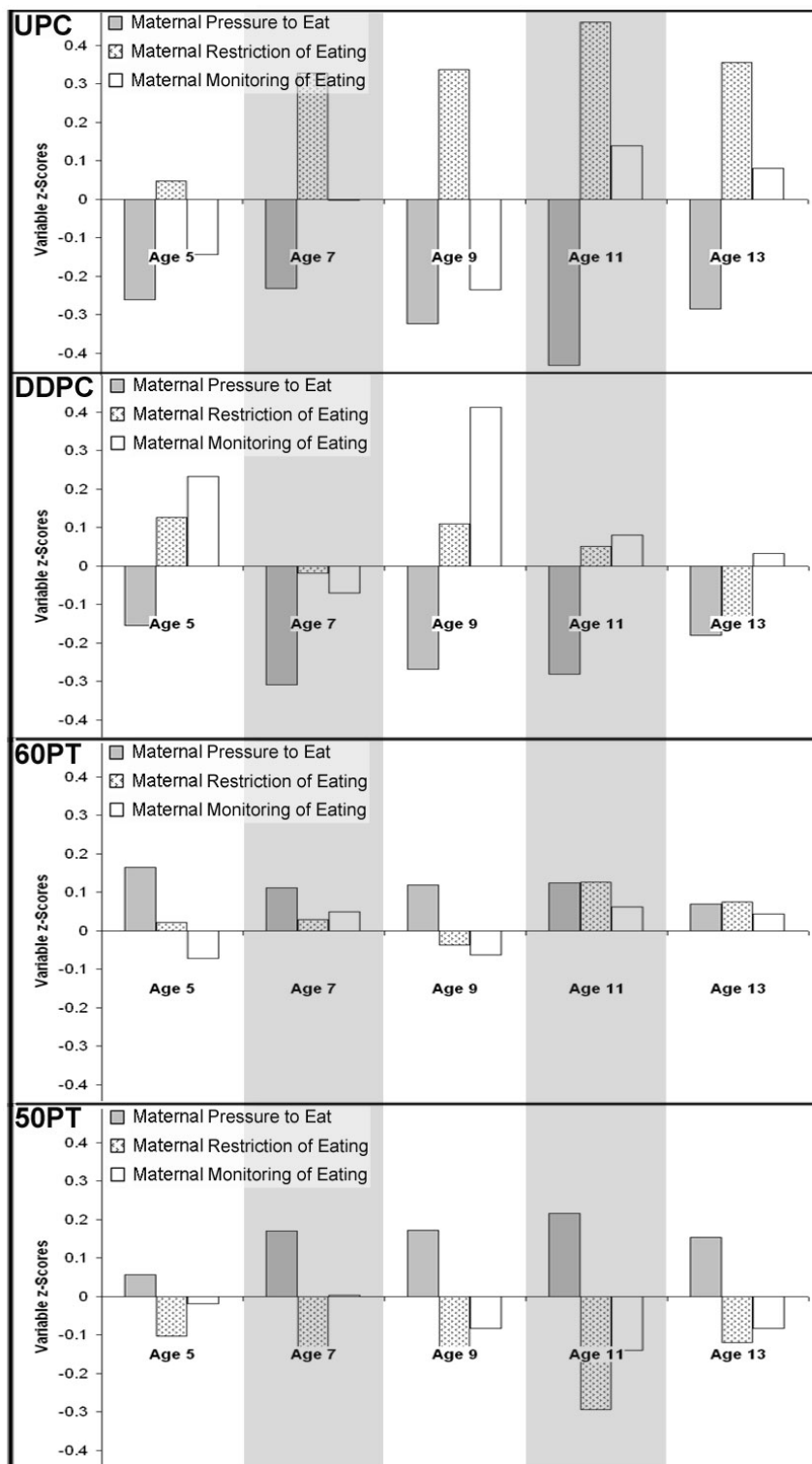Figure 4.4: *Maternal pressure, restriction and monitoring during feeding across ages 5 to 13 years by BMI trajectory group*
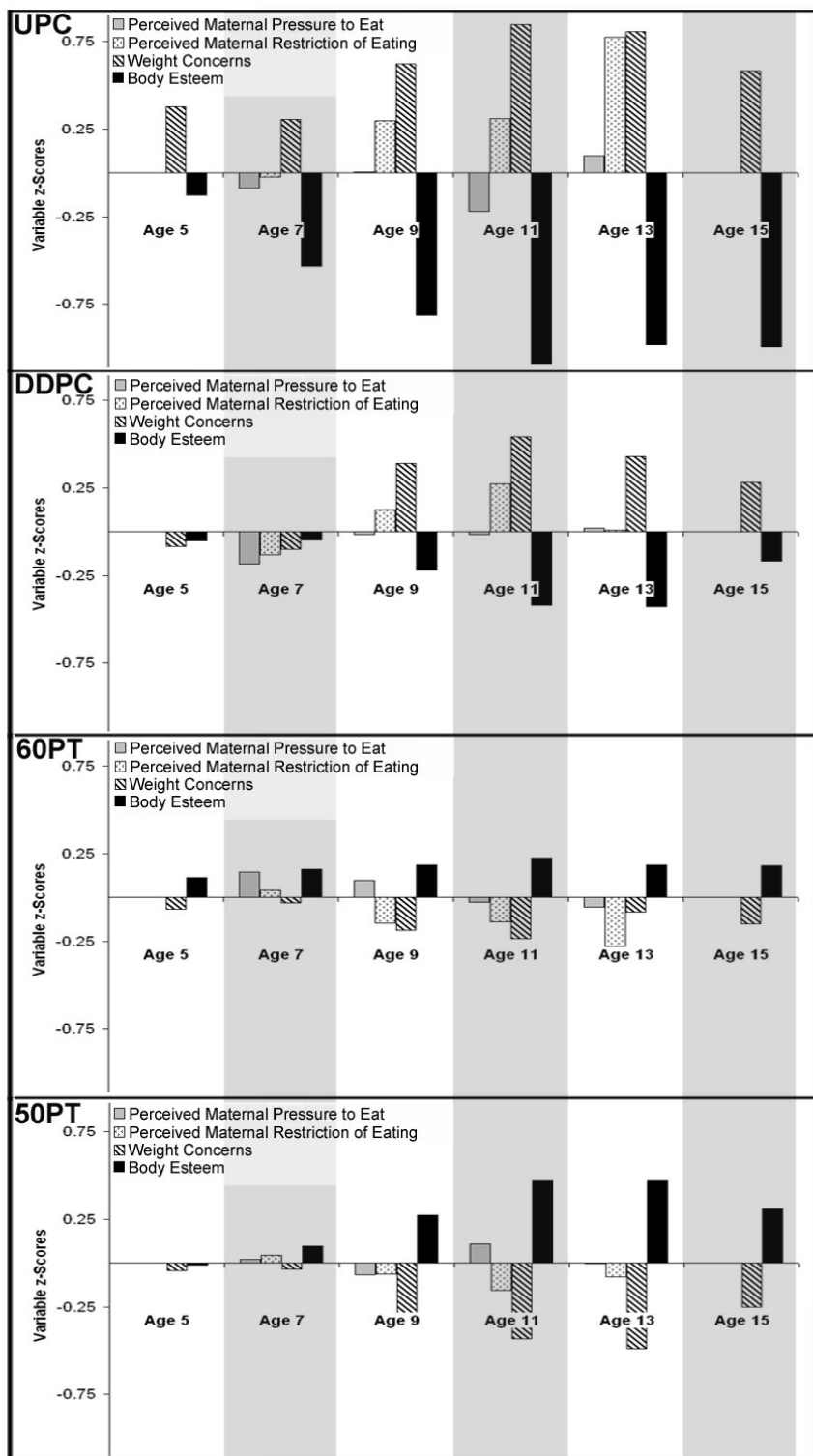
Figure 4.5: *Daughters' perceptions of maternal feeding, weight concerns and body esteem across ages 5 to 15 years by BMI trajectory group*
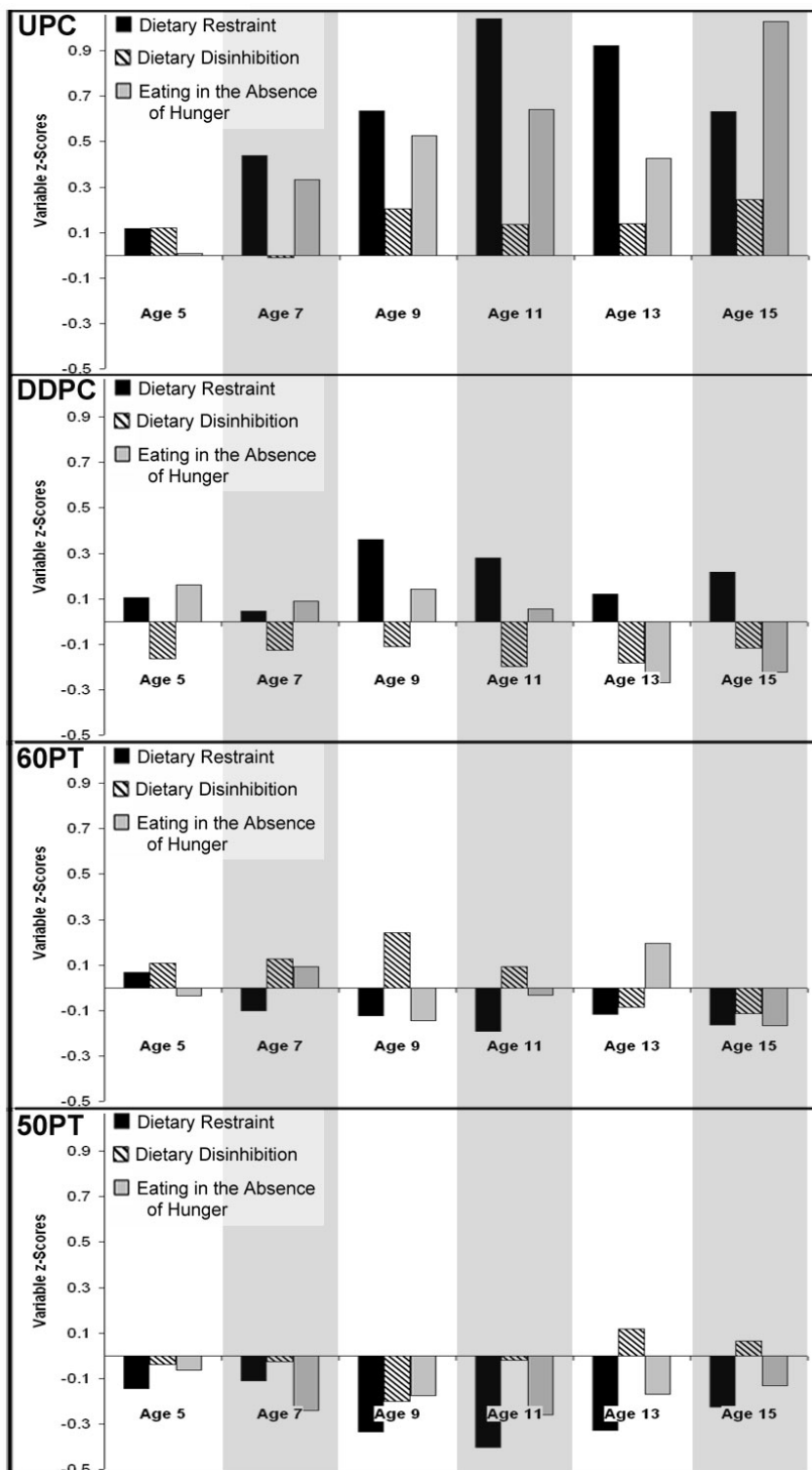
Figure 4.6: *Daughters' dietary restraint, disinhibition and eating in the absence of hunger across ages 5 to 15 years by BMI trajectory group*
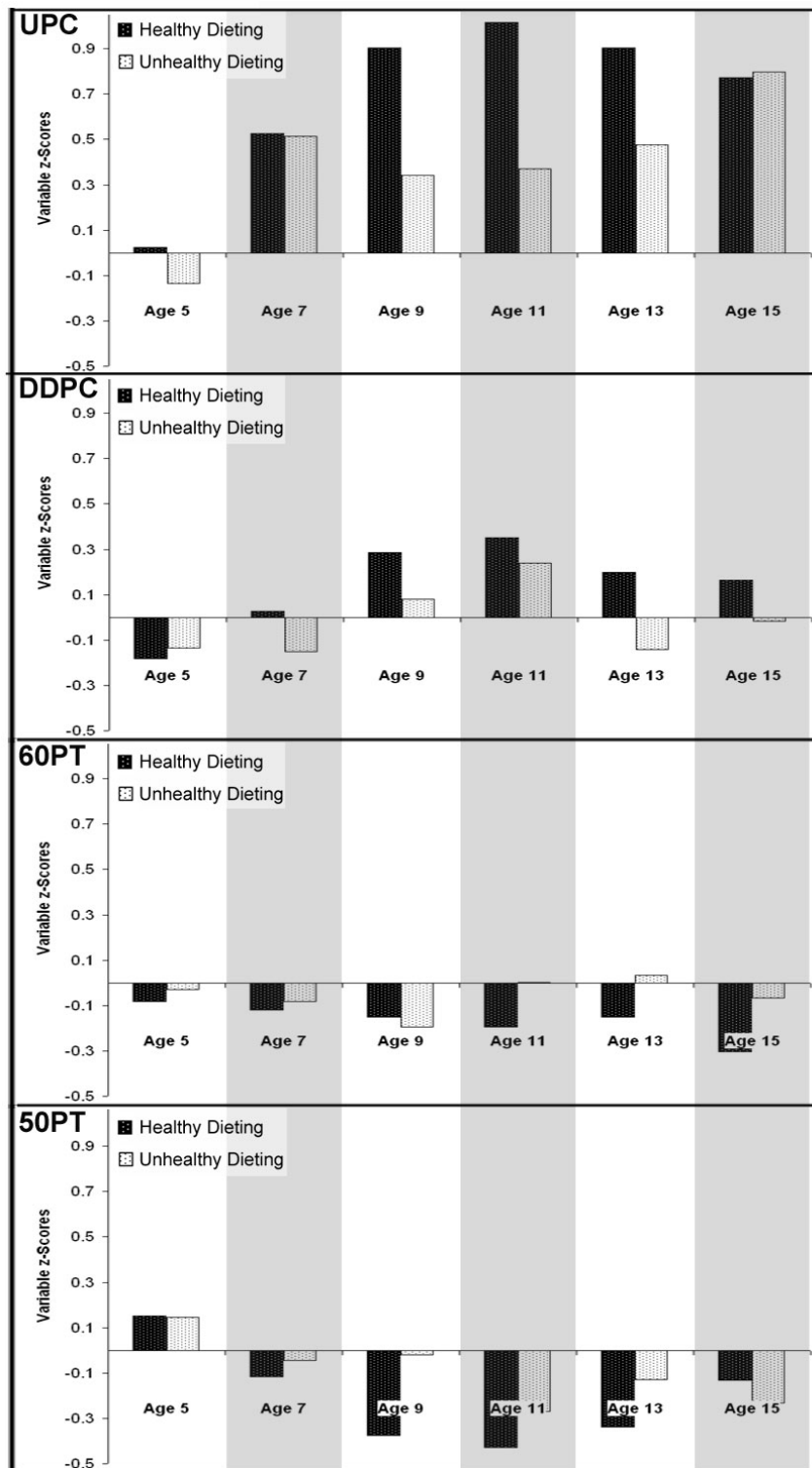
Figure 4.7: *Daughters' dieting practices across ages 5 to 15 years by BMI trajectory group*

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Chapter 5

CONCLUSIONS

The three studies that comprise this dissertation have applied theoretical

perspectives and analytical approaches that are novel to the study of the metabolic

syndrome and obesity. These studies differ from each other in that each uses unique

perspectives and analytical approaches to understand the development of metabolic

syndrome and obesity during childhood, but in combination these studies also illustrate

an overarching message: we can obtain a comprehensive understanding of the etiology of

metabolic health risk during childhood by exploring 1) the developmental trajectories

children follow to reach weight status and metabolic health outcomes; 2) the

heterogeneity that exists in the manifestation of metabolic health risk; and 3) the

individual and familial factors that are associated with the aforementioned trajectories

and outcomes.

In Study 1, we explored two perspectives. The first is that metabolic syndrome

risk is more complex than the dichotomous view imposed by current metabolic syndrome

definitions, where an individual is labeled as "having" or "not having" the metabolic

syndrome based on whether or not he or she met a certain number of criteria. The second

is that risk for metabolic syndrome in adolescent girls can be modeled using a data-driven

analytical approach that does not classify girls based on *a priori* assumptions or

definitions. The application of these perspectives is based on the observation that the

prevalence of the metabolic syndrome in U.S. adolescents is estimated to be

approximately 6% (1), yet, little is known about the development of the metabolic syndrome in youth because children and adolescents are not typically screened for cardiovascular risk factors. Thus, there is currently a lack of consensus for how to best conceptualize the metabolic syndrome in adolescents. Additionally, current definitions for metabolic syndrome in adolescents are based on modifications of adult definitions, as age-appropriate definitions are still under investigation (2).

The objective of Study 1 was to describe a risk typology for the metabolic syndrome in 13-year old girls and to examine the developmental antecedents of this typology. To do this, we used latent profile analysis, an analytical approach novel to this field, and identified the presence of four distinct profiles of metabolic syndrome risk factors. The groups identified were a lower metabolic syndrome risk group (40% of the sample), a dyslipidemia risk group (24%), a hypertension risk group (22%), and a higher metabolic syndrome risk group (14%). Girls in the hypertension risk group and higher metabolic syndrome risk group had higher weight status and adiposity and greater changes in weight status and adiposity across ages 5 to 13 years. Several developmental antecedents across ages 5 to 13 years predicted this metabolic risk typology at age 13, as girls in the higher metabolic syndrome risk group consumed more sweetened beverages across ages 5 to 13 years and girls in the higher dyslipidemia risk group had lower physical activity participation across childhood. Overall, these findings suggest that metabolic health risk is not a dichotomous variable, as we found that several subtypes of risk profiles were present within our sample of girls. These risk profiles were related to other clinically relevant markers of metabolic health, such as adiposity across childhood. Based on these findings, future research should test the effectiveness of targeting the

antecedents identified by this study (for example, decreasing sweetened beverage intakes or increasing physical activity participation) within programs to prevent the development of the metabolic syndrome in youth.

In Study 2, we explored the perspective that heterogeneity exists for childhood growth trajectories and by understanding sources of this heterogeneity we may gain important insights into the etiology of the metabolic syndrome and obesity during childhood. The application of this perspective is based on the observation that the prevalence of childhood overweight obesity has risen dramatically over the past three decades (3), and these increases have been linked to higher prevalence of metabolic health risks during childhood and adolescence (4), yet little is known about the developmental patterns of weight change *within children* that underlie these increases in obesity. Recent research has shown that children (5) and adolescents (6) do not follow a common pathway to weight status outcomes, however, more research is needed to further understand the correlates and outcomes associated with differing weight status trajectories.

The objective of Study 2 was to describe patterns of body mass index (BMI) change across childhood and adolescence and to examine metabolic outcomes, as well as the familial demographic, dietary intake, physical activity and television viewing patterns that are associated with these trajectories. To do this, we employed growth mixture models, another analytical approach novel to this field, and identified the presence of four distinct patterns of BMI change across ages 5 to 15 years. When described in relation to the CDC growth reference BMI percentile curves, we found that one subgroup of girls exhibited upward percentile crossing change in BMI (14% of the sample), another

subgroup of girls exhibited a delayed downward percentile crossing (20%), and a third and fourth group exhibited percentile tracking, one along the 60th percentile (29%) and the other along the 50th percentile (37%). Girls with upward percentile crossing and delayed downward percentile crossing BMI trajectories had greater increases in percentile body fat across ages 5 to 9 years, as well as earlier pubertal timing, compared to girls who tracked along percentile lines. However, only girls exhibiting upward percentile crossing across all of childhood had more metabolic risk factors at age 15, compared to all other girls in this sample. With respect to predictors and correlates of these trajectories, maternal BMI at study entry was significantly higher for girls exhibiting an upward percentile crossing BMI trajectory compared to all other groups. We could not identify dietary and physical activity patterns associated with differing BMI trajectories, however, for all girls in our sample we noted increases across childhood in tendencies to under-report energy intake. These tendencies were greater for girls exhibiting an upward percentile crossing BMI trajectory. Thus, systematic bias may have hindered our ability to detect links between dietary intake and patterns of BMI change in our sample. These findings suggest that alternative measures of diet and physical activity should be examined to confront the possibility that reporting bias hindered our ability to link health behaviors to health outcomes in this study.

Study 3 was designed to build upon the conclusions of Study 2. In Study 3, we explored the perspective that assessment of maternal influences and more global measures of daughters' diet- and weight-related self-regulation (e.g., dietary restraint, dieting attempts, and eating in the absence of hunger) will provide insights into the association between individual behaviors and weight status trajectories. Thus, the

objective of Study 3 was to examine the mother and daughter psychosocial correlates of heterogeneity in trajectories for BMI change in daughters. Using the same trajectory groups identified in Study 2, we found girls who exhibited a persistent upward BMI percentile crossing across age 5 to 15 years developed in a distinct ecology, characterized by 1) higher maternal weight status, weight concerns and patterns of disinhibited eating, and 2) higher maternal encouragement of their daughters to lose weight coupled with use of restrictive feeding practices in response to concerns for their daughters' weight status. Mothers of girls who exhibited a downward deflection in their BMI percentile trajectory during middle childhood showed similar levels of concern about their daughters' early accelerated BMI trajectory compared to mothers of girls exhibiting upward BMI percentile crossing. These two groups of mothers differed in that the former reacted to this concern by applying moderate levels of encouragement to lose weight and by monitoring, rather than restricting, their daughters' intakes during middle childhood. Starting at age 9, these daughters exhibited moderate levels of weight concern, dietary restraint and healthy dieting behaviors, which may have been a healthy reaction to their higher weight status across ages 5 to 9 years and an effective strategy for achieving a healthy weight during later childhood and adolescence. Overall, findings from Study 3 suggest future research should test intervention programs that help overweight mothers foster healthy weight status in daughters through: 1) maternal modeling of healthy diet- and weight-related attitudes and behaviors, 2) monitoring, rather than restriction, of daughters' intakes, and 3) promotion of moderate levels of dietary restraint and healthy dieting practices in daughters.

These three studies were exploratory in nature with the overarching goal of applying new perspectives and approaches to understand metabolic syndrome and obesity risk in children. However, a commonality that emerged from the culmination of these findings is that these studies illustrated metabolic health and growth patterns that have been widely recognized in clinical settings using predetermined criteria and thresholds. These clinically observed patterns include: 1) the metabolically obese, normal weight phenotype in adults; 2) accelerated growth as a predictor of later obesity and metabolic risk; and 3) weight maintenance during linear growth as a goal for overweight children to achieve a healthy weight status.

With respect to the metabolically obese, normal weight phenotype, a common observation among physicians is the presence of a subgroup of adults who are labeled "metabolically obese" because they exhibit insulin resistance, dyslipidemia or hypertension (characteristics associated with obesity), yet are not overweight or obese (7). In both clinical and research settings, this subgroup of individuals is typically classified *a priori* by assessing whether normal weight individuals exceed clinical thresholds for metabolic abnormalities (8, 9). Although evidence in pediatric populations on this subtype of individuals is limited, recent research has used similar *a priori* classification methods to identify the presence of metabolically obese normal weight children (10).

In Study 1, we did not use *a priori* cut-offs to classify girls as metabolically obese and we did not conduct our analysis with the assumption that a subset of girls would exhibit a metabolically obese, normal weight phenotype. However, through our data-driven identification of a metabolic syndrome risk typology, we identified a

subgroup of girls who had a normal weight status but, on average, met or were close to thresholds for abnormal triglyceride and HDL values. We labeled this group the dyslipidemia risk group, but could have also labeled them the metabolically obese, normal weight girls.

An important consideration that has been recognized in the adult medical literature is that this subgroup of at-risk individuals may not be detected by current screening practices, which often focus on the weight status of an individual as a main indicator of health risk (8-10). In adults, broadening our understanding of obesity to include classification of individuals who are metabolically obese has important implications for therapeutic efforts, as these individuals need nutritional and lifestyle intervention despite their normal weight status (8). In pediatric populations, metabolically obese, normal weight children may be especially unlikely to be detected as at higher risk for metabolic syndrome and related co-morbidities because of their normal BMI and young age. Study 1 supports the presence of a metabolically obese, normal weight phenotype in a sample of adolescence girls and these findings suggest monitoring of metabolic risk factors is warranted in youth so these at-risk children can be targeted by prevention and therapeutic efforts.

With respect to associations between accelerated growth and later metabolic health risk, the correspondence between the present research and the use of upward percentile crossing as a clinical indicator of problematic growth was discussed in both Study 2 and 3 thus will only briefly be discussed here. BMI tracking and percentile crossing are used in clinical settings to identify individual children at risk for overweight and obesity (11-13). In correspondence with earlier research linking accelerated weight

gain during infancy and early childhood with higher risk for obesity and undesirable metabolic outcomes (14-16), we found that girls exhibiting accelerated growth across childhood had more metabolic risk factors during adolescence. However, it is also important to note that a different subgroup of girls exhibited upward percentile crossing limited to ages 5 to 9 years and these girls did not present metabolic risk factors at age 15. In a recent statement regarding the prevention, assessment and treatment of child and adolescent overweight and obesity, the American Academy of Pediatrics recommended that "physicians and allied health care providers perform, at a minimum, a yearly assessment of weight status for all children and that this assessment include calculation of height, weight (measured appropriately), and BMI for age and plotting of those measures on standard growth charts (p. S186, (13))." Our findings also support the potential usefulness of tracking individual children's BMI in clinical settings, but add support for the need to consider a child's entire growth trajectory and not just early accelerated weight gain, as we found that only girls with accelerated growth (i.e., upward BMI percentile crossing) across *all* of childhood had undesirable metabolic outcomes at age 15 years.

With respect to weight maintenance during linear growth, pediatricians recommend that this growth pattern be the goal of therapeutic efforts to reverse childhood overweight and prevent adolescent and adult obesity. The rationale behind this recommendation is the notion that growing children have the potential to "grow into" their higher weight status and promotion of weight loss in young children may lead to eating disorders or stigma (13). In Study 2, the girls who exhibited a delayed downward percentile crossing BMI trajectory across ages 11 to 15 years illustrated this weight

maintenance during linear growth, which allowed these girls to achieve a healthy weight status by age 15 years. Although body weight data was not presented in Study 2, these girls, on average, gained 20 kg across ages 5 to 9 years, which was similar to the weight gain across this same period (21 kg) for girls exhibiting a persistent upward BMI percentile crossing trajectory. However, across ages 11 to 15 years, girls exhibiting a delayed downward percentile crossing trajectory gained an average of 6 kg, which was less than the average weight gain across this same period for girls who tracked along the 60th percentile (15 kg) and 50th percentile (19 kg), as well as for girls exhibiting a persistent upward percentile crossing (22 kg).

In Study 3, we presented evidence that weight maintenance during linear growth was not simply attributable to girls naturally "growing out" of their overweight status. Rather, this growth pattern was likely a function of maternal and daughter behaviors and interactions. These girls had mothers who were concerned about the weight status of their daughters, but expressed this concern through moderate encouragement of weight loss and monitoring of daughters' dietary intake during middle childhood. These daughters also exhibited moderate levels of weight concern, dietary restraint and healthy dieting during middle childhood, all of which have been identified as effective weight maintenance strategies in adults (17, 18). Our data suggest that weight maintenance during linear growth is achievable in a naturalistic setting and illustrates several maternal and daughter behaviors that therapeutic efforts can promote to achieve this weight status trajectory. Specifically, pediatricians and intervention programs that recommend that families should strive to help overweight daughters maintain their body weight during linear growth should also recommend that mothers 1) model healthy weight status and

eating behaviors, 2) monitor, not restrict, daughters' intakes, and 3) apply moderate encouragement of daughters' weight maintenance. Additionally, these programs should promote, or help families promote, moderate levels of dietary restraint and healthy dieting behaviors in these overweight girls to help them achieve weight maintenance and, ultimately, healthy weight status.

Overall, the culmination of findings presented within this dissertation provides support for the natural presence of clinically relevant metabolic profiles and both maladaptive and adaptive growth patterns in children. This correspondence provides further validation for the utility of the data-driven approaches we employed. Because the present research also examined how patterns of dietary intake, physical activity, cardiorespiratory fitness, eating styles, psychosocial characteristics and mother-child interaction may influence these clinically relevant metabolic profiles and growth patterns, the conclusions drawn from the present research highlight have implications for future metabolic syndrome and obesity prevention and intervention efforts.

Future Directions

A strength of the present research is that we made use of a rich, longitudinal dataset, which allowed us to explore a wide array of factors influencing the development of metabolic syndrome and obesity. However, this research is not without limitations and these limitations suggest avenues for future studies. Our sample was a racially and demographically homogeneous sample of girls and their parents; despite this homogeneity, we found heterogeneity in both metabolic health risk and developmental weight status trajectories. Thus, the perspectives and analytical approaches applied here should be further explored in more diverse samples to understand the true depth of this

heterogeneity. Additionally, many of the behavioral measures examined were self-reported, which may have limited our ability to find associations between individual behaviors and outcomes; although this limitation was addressed in Study 3, future research should further explore the use of alternative behavioral markers for habitual consumption and expenditure patterns. Our findings were exploratory and correlational by nature, which limits our ability to infer causality. Future research should attempt to establish causal relations between child behaviors, parent influences and child weight status and metabolic outcomes; one strategy for conducting this causal research is through intervention studies where promising modifiable behaviors can be tested as intervention components against a standard or no treatment control.

The research presented here suggests several potential targets for these future intervention studies or prevention efforts. Overall, our evidence suggests that decreasing sweetened beverage consumption in young girls and promotion of physical activity and physical fitness across childhood may decrease dyslipidemia and metabolic syndrome risk during adolescence. Our evidence also suggests that both mothers and daughters should be targeted for childhood obesity and metabolic syndrome prevention and intervention efforts, as maternal modeling and interactions with daughters surrounding dietary intake and weight status patterns were associated with daughters' growth trajectories, as well as reactions to and perceptions of weight status. Future research should continue to apply the perspectives and analytical approaches presented here to further explore and understand the developmental trajectories children follow to reach weight status and metabolic health outcomes, the heterogeneity that exists in the

manifestation of metabolic health risk, and the individual and familial factors that are associated with these differing trajectories and outcomes.

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