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Trajectories, Predictors, and Adolescent Health Outcomes of Childhood Weight Gain: A Growth Mixture Model

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Trajectories, Predictors, and Adolescent Health Outcomes of Childhood Weight Gain: A Growth Mixture Model

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Dedication

For my mothers

Julie, Ursula, Monika, Richard

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Trajectories, Predictors, and Adolescent Health Outcomes of Childhood Weight Gain: A Growth Mixture Model

Anne Bichteler, Ph.D.

The University of Texas at Austin, 2014

Supervisor: Elizabeth Gershoff

Obesity, as defined as BMI at or above the 95th percentile on the Centers for Disease Control and Prevention's growth charts, has increased almost 3-fold among children in the United States since 1980. Overweight in adolescence has been associated with increased fat retention and high blood pressure in adulthood, among other symptoms of metabolic syndrome. However, normative patterns of weight change in childhood have not been developed. Groups of children may follow different trajectory patterns of BMI change over time. If common trajectory patterns could be identified, and their risk factors and outcomes understood, more nuanced intervention with families and children at risk for obesity could be developed. This study used a national dataset of 1,364 children whose weight and length was measured 12 times from birth through 15 ½ years. Testing both latent class growth analysis and growth mixture modeling identified four distinct subgroups, or classes, of BMI growth trajectory from 24 months – 8th grade. These classes were compared on numerous demographic, biological, and psychosocial risk factors identified in previous research as related to obesity. Classes were differentiated primarily on the child's BMI at 15 months, the mother's BMI at 15 months,

birth weight for age, and percent increase in birth weight. Being male, Black, and lower SES were also related to membership in the higher-BMI trajectory classes. Of the psychosocial factors, maternal sensitivity, maternal depression, and attachment classification were also related to BMI class. Membership in these trajectories strongly predicted weight-related and blood-pressure outcomes at 15 ½ years over and above individual risk factors, demonstrating that patterns of change themselves are highly influential. The best-fitting models of weight-related outcomes at 15 ½ years included change trajectory in combination with biological, psychosocial, and SES risk factors from 0-24 months, with R^2 ranging from .31 = .50. Characteristics predicting adolescent overweight can be identified in the first years of life and should trigger the development and implementation of early intervention protocols in obstetrics and pediatrics.

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Introduction

Since the 1980's, the rapid increase in childhood obesity has been a public health concern in the United States. Seven percent of children were obese in 1980, increasing to 18% in 2012 (Figure 1). Although the incidence of obesity may now be dropping in the highest-risk populations (Centers for Disease Control and Prevention (CDC), 2012), well over a third of children and adolescents in the United States are overweight or obese. Childhood overweight is known to be associated with increased risk for type II diabetes, several cancers, bone and joint problems, sleep apnea, and poor socio-emotional outcomes (CDC, 2014). Federal health officials are now concerned that adolescent overweight is a risk for what is known as metabolic syndrome, the group of 5 risk factors associated with heart disease and stroke in adulthood, namely 1) large waist circumference/abdominal obesity, 2) high triglyceride level, 3) low HDL, or "good" cholesterol level, 4) high blood pressure, and 5) high fasting blood sugar (United States Department of Health & Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute, 2011).

Understanding if and how childhood weight gain influences disease in adolescence and adulthood requires longitudinal data. As the CDC's and World Health Organization's (WHO) healthy weight standards are developed on cross-sectional data sets (Kuczmarski et al., 2002), characterizations of developmentally normative change in body fat from birth to adolescence are lacking. Understanding the causes and outcomes of childhood weight gain requires the common patterns of change as a reference.

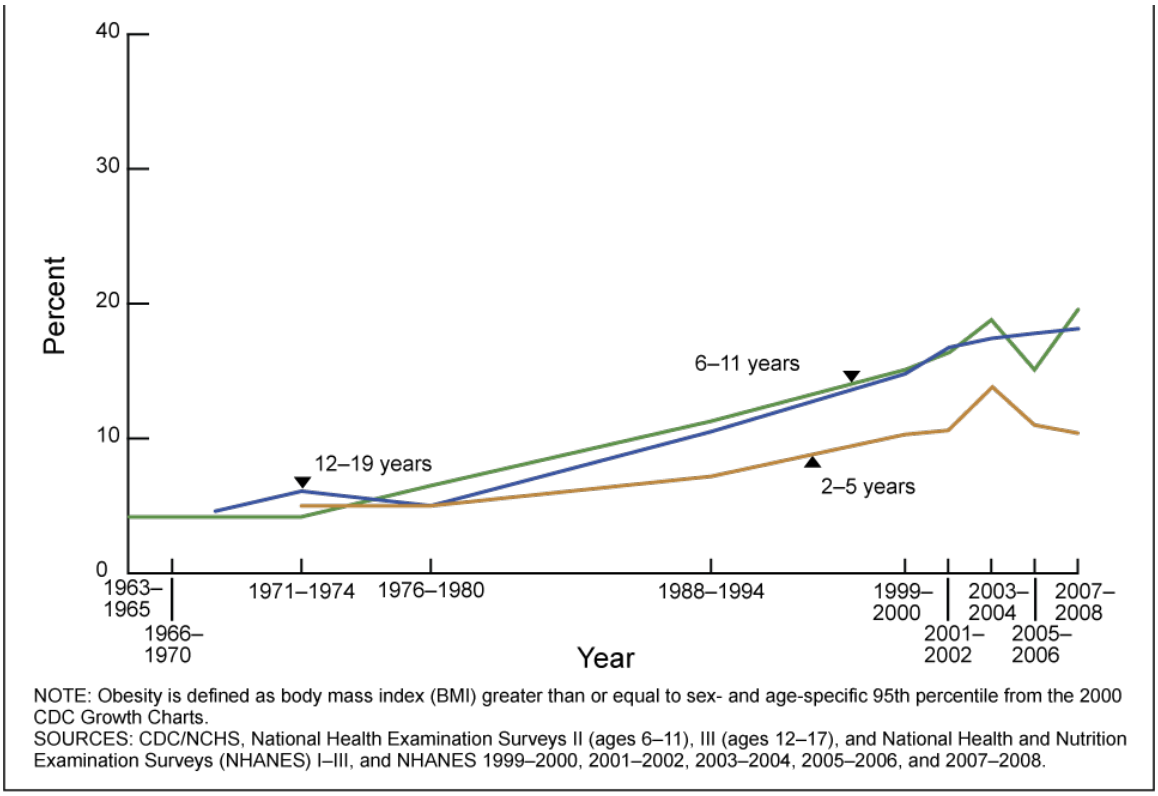

Figure 1. Trends in obesity among children and adolescents: United States, 1963-2008 (Ogden & Carroll, 2010).

Although the CDC's means and percentiles of body mass index (BMI) for given ages are termed "growth charts," they do not describe trajectories of change over time. Without mapping individuals' weight change from birth, common trajectory patterns cannot be identified, nor can risk be modeled for overweight and obesity.

The lack of understanding about what constitutes normative growth patterns of childhood weight is one reason that the etiology of obesity remains murky. On the one hand, research in developed countries has converged on a set of conditions promoting energy intake and discouraging energy expenditure, termed "obesogenic" influences, in the last several decades. Counted among obesogenic factors are an increase in relative poverty, an increase in fast food availability and consumption, the fall in price and

nutritional value of fast and prepared foods, the introduction of fast food into schools to offset cost, heavy marketing to children and lobbying by manufacturers of low-quality food, withdrawn funding from schools for physical education or after school sports, and unsafe sidewalks, playgrounds and neighborhoods for outdoor play (reviewed in Ebbeling, Pawlak & Ludwig, 2002). These have been concomitant with increases in sedentary activities, such as television viewing, video gaming and internet surfing. Overcoming these cultural, social, and political forces seems so daunting that researchers have focused most of their energy on discovering how and why individuals are differentially susceptible to them (Deckelbaum & Williams, 2001; Ebbeling, Pawlak & Ludwig, 2002). As risk for overweight may start as early as pre-conception, research is focused increasingly on children's genetic, environmental, and socio-emotional differences in the first few years of life, when intervention could be most effective.

The current study used the NICHD SECCYD longitudinal data set of 1364 children to address three main goals aimed at filling in several of the gaps in understanding the trajectories of weight gain across childhood and its sequelae, namely: 1) To use longitudinal data to define the shape and trajectories of typical healthy and unhealthy weight gain patterns over time; 2) To determine which biological and social risk factors in earliest childhood might predict weight change trajectories, and 3) To determine to what extent these childhood trajectories and indicators influence adolescent weight and symptoms of metabolic syndrome, as these have been shown to relate directly to risk for cardiovascular disease in adulthood.

CHILDHOOD OVERWEIGHT AND OBESITY

The Centers for Disease Control and Prevention (CDC) estimate a two-fold increase in obesity in children and a four-fold increase in adolescents in the United States over the last 30 years (CDC, 2014). Between 1980 and 2008, obesity increased from 5.0% to 10.4% among preschool children aged 2 to 5 years of age and from 6.5% to 19.6% among those aged 6-11. Among adolescents aged 12-19, obesity increased from 5.0% to 18.1% during the same period. It has increased in both genders and in all ethnic and socioeconomic groups (Ogden & Carroll, 2010), although rates of obesity in minority groups are consistently higher than in non-Hispanic whites, such that 19.8% of Black boys, 26.8% of Hispanic boys, and 16.7% of White boys are obese, while 29.2% of Black girls, 17.4% of Hispanic girls, and 14.5% of White girls are obese (Ogden & Carroll, 2010).

Childhood overweight and obesity, currently defined by the CDC and in this study as >= 85th and 95th percentiles of BMI respectively, are detrimental to both near-term and long-term physical health. Immediate health effects include high cholesterol, high blood pressure, prediabetes, bone and joint problems, and sleep apnea; long-term risks include adult obesity and its predicted increases in heart disease, type-2 diabetes, stroke, some cancers, and osteoarthritis (for an overview, see CDC, 2014). Modest correlations (r = .33 - .41) between BMI at 2-5 years old and adult BMI and adiposity (fat stored in the fatty tissue of the body) suggest a lifespan impact of overweight in the earliest years of life (Freedman, Khan, Serdula, Dietz, Srinivasan, & Berenson, 2005). Understanding trajectories of childhood overweight is particularly important, as both BMI and change in BMI percentile have separate influences on the components of

metabolic syndrome in adolescence and adulthood (Morrison, Aronson, & Gray-McGuire, 2007; Sun et al., 2008).

TRAJECTORIES OF OBESITY ACROSS CHILDHOOD

Although the CDC's BMI percentile charts are called "growth charts", they are in fact smoothed interpolations across cross-sectional measurements of different children at different ages. They are based on data collected primarily in the CDC's National Center for Health Statistics' (NCHS) National Health and Nutrition Examination Survey (NHANES) I, II, and III surveys and from supplemental data sources such as U.S. Vital Statistics and the Fels Longitudinal Study from the Fels Research Institute. Percentiles of children's weight-, length-, and BMI-for-age were grouped in one-month increments up to age 36 months, then in 6-month increments through age 20 years; these were then smoothed using a combination of linear weighted and polynomial regression techniques (Kuczmarski et al., 2002). These data, although presented as smoothed curves, do not inform us of different trajectories of growth, such as how children might vary from one another in their weight percentiles over time, what patterns of change might be normative vs. non-normative, at which ages children can be identified as being on one or another trajectory, whether certain critical periods are more influential in predicting weight outcomes in adolescence, or whether different sets or levels of risk factors predict different growth trajectories.

Analyses using longitudinal data to address these questions typically use a measure of percentile of body fat at a prior point in time to predict a measure or percentile of body fat at a later point. These analyses consider all children together,

relying on an assumption that their means and change over time are homogenous, which can result in ambiguous findings. High birth weight predicts obesity in early childhood (Dubois & Girard, 2006; Reilly et al., 2005); in a meta-analysis by Yu and colleagues (2011) examining associations between birth weight and subsequent risk of obesity, low birth weight was associated with a decreased risk of obesity, although not in studies of cohorts, studies with large sample sizes, or studies graded as higher quality. Differentiating circumstances may be present: rapid weight gain in the first 6 months to 1 year of life, particularly following low birth weight, predicts obesity in early childhood (Taveras et al., 2009; for reviews, see Fisher et al., 2006 and Monteiro & Victora, 2005), and on the other hand, catch-up weight gain may be important for small-for-gestational-age (SGA) infants to prevent neurodevelopmental deficiencies, and may not necessarily be associated with visceral adiposity and insulin resistance (Yeung, 2006; for a review, see Jain & Singhal, 2012). Clearly the change in weight for different children at different points of development may have different influences on health outcomes.

The risk for later adiposity in particular associated with the timing of childhood obesity is unclear. A review of literature published between 1985-1996 reported only weak associations among obesity statuses in childhood, adolescent, and adulthood (Power, Lake, & Cole, 1997). A review by Goran (2001) concluded that the risk for obesity later in life rose with increasing age at which the obesity first appeared; but studies on the risk associated with obesity timing since have found incidence in the earliest years to be associated with the greatest persistence into young adulthood (Cunningham et al., 2014). Fisher et al. (2006) reviewed the associations between infant

birth weight and growth and burden of disease in adulthood and with a couple of exceptions, such as the association between high birth weight and type 2 diabetes, were able only to conclude "that there is no single optimal pattern of infant growth that is associated with beneficial adult health outcomes" (p. 1208).

It may be that the trajectory of weight gain from infancy through adolescence is not the same for all children, and that studies such as those referenced above, which model means of entire study populations together, mask group differences in weight gain trajectories. A specific case of this possibility has already been identified, known as "adiposity rebound." Adiposity rebound refers to the point when children's BMI begins to rise again after its initial drop at around age 1 year. The sooner this rebound occurs, the faster and higher the rise in BMI over childhood (see Rolland-Cachera, Deheeger, Maillot, & Bellisle, 2006, for a review). If groups, or classes, of individuals, showed similar patterns of change over the period from birth through adolescence, risk and outcome models for these classes could identify different developmental trajectories for weight gain in childhood. Such different patterns of change could explain outcomes such as differences in risk of later adolescent obesity and symptoms of metabolic syndrome.

Research estimating different classes of obesity growth trajectories over time is rare. None yet exists estimating trajectories from birth through adolescence; two have estimated growth classes for large chunks of childhood, however. Pryor et al. (2011) modeled children from 5 months to 8 years, and Li et al. (2007) modeled them from 2 years to 12 years.

Pryor et al. (2011) used semiparametric mixture modeling with SAS Proc Traj to identify 3 classes of growth: a low-stable, a moderate, and a high-rising (Figure 2). Logistic regression was used to predict class membership in the high-rising vs. other groups. Starting at age 2.5 years, the high-rising trajectory diverged from the others and grew steadily; the other two groups' adiposity rebound did not begin until age 6 years and remained relatively flat. The authors note several limitations in this study: the use of maternal report of height and weight between 5 months and 5 years, the measure of maternal BMI available first at 17 months, and the inability to control for age due to

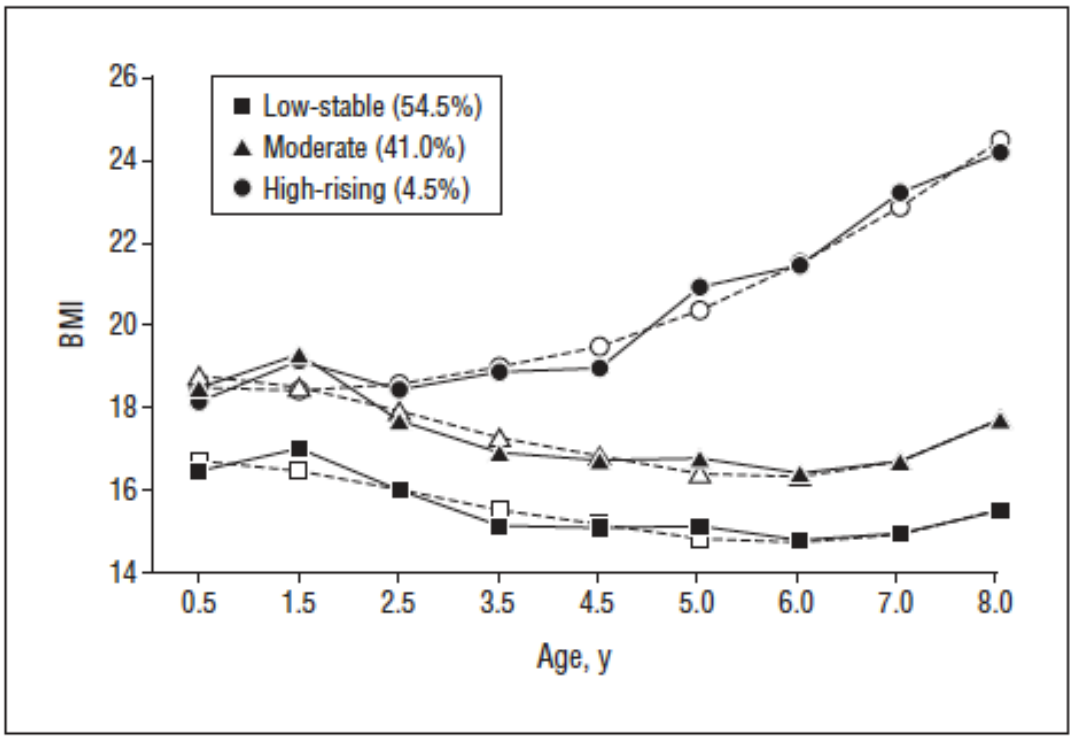

Figure 2. "Body mass index (BMI) trajectory groups (with BMI calculated as weight in kilograms divided by height in meters squared). Solid lines represent observed values, and dashed lines represent expected values." (Pryor, 2011, p. 909).

longitudinal attrition. Of additional note is that the study, rather than using percentile of BMI as recommended for analysis of children (CDC, 2014), relied on measured BMI, which tends to rise monotonically, obscuring differences within a population.

Li et al. (2007) used latent growth mixture modeling with BMI percentiles of a nationally representative sample of children from age 2 to12 years using the children of women in the NLSY79, a nationally representative sample first surveyed in 1979. Sampling weights were used to adjust for differential non-response and oversampling of Blacks, Hispanics, and economically disadvantaged non-black/non-Hispanic Whites.

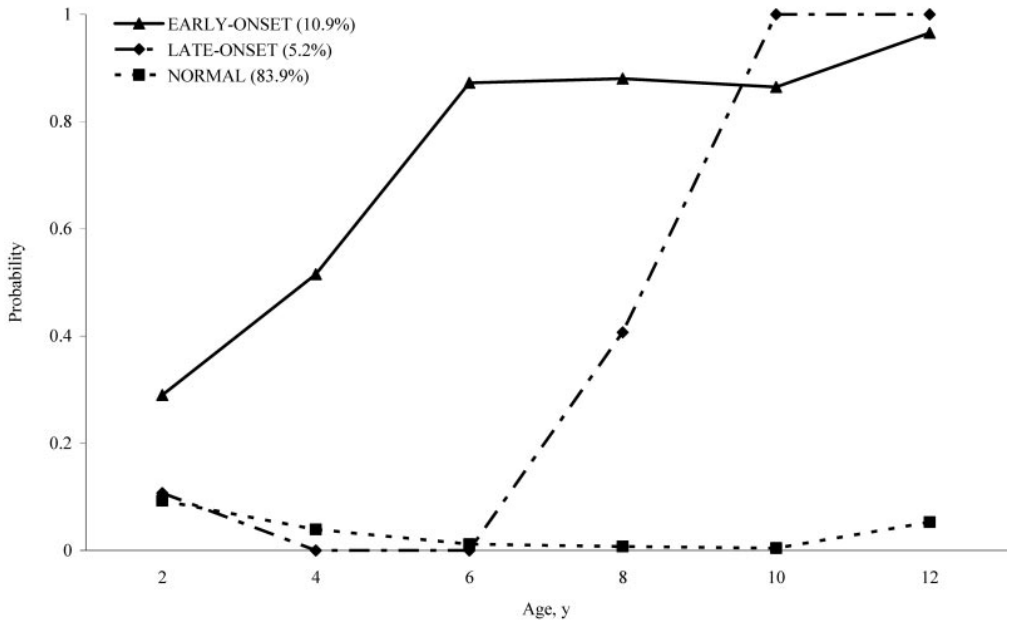

Figure 3. Probability of overweight in each of the 3 trajectory growth classes identified by latent growth mixture modeling in Li et al. (2007).

Latent growth mixture modeling allowed heterogeneity in trajectories and variance within and across trajectories, while estimating growth over time. Li et al. identified 3 classes of growth: early-onset, late-onset, and normal (Figure 3). Logistic analyses and group mean differences were used to characterize membership in each class.

The primary methodological weakness in this work was the absence of repeated measures before age 2 and after age 12. The early-onset trajectory was already on the rise by age 2, but differentiation by catch-up growth was not possible. Additional key risk factors for childhood weight gain were also omitted, such as maternal smoking. In addition, although the practice seems prevalent in the literature, the study's categorization of all continuous independent variables, such as birth weight or family income, reduces power, imposes breakpoints where they may not be supported ecologically, and does not take advantage of the capacity of growth mixture modeling to estimate trajectories of change with continuous variables.

Thus, while these two previous attempts to identify growth trajectories in BMI were successful in identifying classes of growth, each study had significant drawbacks. The current study addressed these by using height and weight measured by health care professionals from 0-15 ½ years and by modeling the BMI percentiles created by the CDC rather than raw scores. Measurements from 24 months through 8th grade were used to identify patterns of growth in BMI percentile over time. This study employed latent growth modeling, known for its usefulness in identifying homogenous sub-groups within larger heterogeneous populations (Jung & Wickrama, 2008), to answer the first research question:

Research Question 1

How many different classes of BMI growth trajectories from birth to adolescence exist in the national SECCYD dataset?

ETIOLOGY OF OVERWEIGHT/OBESITY

As overweight in very early childhood has been linked increasingly with adolescent and adult obesity (Cunningham, Kramer, & Narayan, 2014; Magarey, Boulton, & Cockington, 2003), attention has been shifting to the earliest years of life to identify associated risk factors. Many biological, environmental, and behavioral mechanisms have been identified which may contribute to weight gain prenatally and in the first few years after birth (see Ebbeling et al., 2002, for a review). Data have emerged to suggest influences of maternal and fetal factors during intrauterine growth as well as child growth during the first year of life on risk for later development of adult obesity and its comorbidities, findings which encourage a life-cycle approach to the etiology and inter-generational transmission of obesity from a biological perspective (Deckelbaum & Williams, 2001). Researchers are also exploring whether earliest emotional development, family relationships, and experienced adversity may explain some of the individual differences in weight trajectories (Thomas, Hyppönen, & Power, 2008; Davis et al., 2014; Ebbeling et al., 2002). Differences in demographic, biological and psychosocial factors between the trajectory groups identified in this study could provide insight into the relative strength of these risks for children's weight gain.

POTENTIAL PREDICTORS OF CHILDHOOD WEIGHT GROWTH PATTERNS

Childhood adversity. Reported effects of adverse experiences and relationships in childhood on health outcomes in adulthood, although robust, have been largely retrospective and cross-sectional in nature (see Danese & McEwen, 2012, and Uchino, 2009, for reviews). Recent availability of prospective longitudinal data, such as those used in the current study that include measures of quality of care and early relationships along with later socio-emotional, mental, and physical functioning, is allowing exploration of developmental associations between adverse events and relational stress in early childhood and later physical health outcomes (e.g. Moffitt et al., 2010). It is thought that adverse experiences in early childhood may trigger systemic psychological, neurological and endocrine changes that permanently alter stress physiology. For example, childhood abuse and family dysfunction have been linked with negative outcomes in adulthood such as alcoholism, smoking, suicide attempts, physical inactivity, severe obesity, heart disease, lung disease, and certain cancers (Felitti et al., 1998). In a 2011 review of cross-sectional and longitudinal studies predicting childhood obesity from psychosocial stressors, maternal mental and physical health, household strain and disruption, general and parenting stress and distress, housing and financial insecurity, and negative life events were all found to contribute to adversity-related weight outcomes (Gundersen, Mahatmya, Garasky, & Loohman, 2011).

Although the effects, or at least the recall, of childhood adversity appears to have an influence on adult health, stress appears to have only a negligible direct effect on overweight in children in prospective studies to date. Although repeated and/or chronic stress could be expected, based on theories and animal studies of HPA-axis activation and

neuro-endocrine responses, to trigger weight gain in children (Katz, Sprang, & Cooke, 2012), published research linking allostatic load in childhood with childhood weight gain has so far failed to find a link. In their review of psychosocial stress and childhood obesity, Gundersen et al. suggest that associations that may be attributed to stressors may in fact be the result of as-yet-unobserved traits among children that influence their coping strategies. Null findings with respect to general allostatic load may be because individuals develop different patterns of stress response very early, and some may be prone to obesogenic behaviors under stress and others not. Without differentiated trajectory classes of weight gain and time-varying measures of adversity, differences in response to stress could go unseen. Recent research has suggested that adults cope very differently with stress and have divergent patterns of overweight and obesity depending on how they represent their childhood (Bichteler & Jacobvitz, 2014, under review). In that study, adult attachment representation was shown to have a moderating influence on the impact of major life traumas on weight: dismissing speakers' weight increased and preoccupied speakers' fell with increasing numbers of severe lifetime adversities endured. The current project extended this research to children by using infant attachment classification and early childhood adversity in prospective longitudinal data.

Attachment security. Longitudinal studies of the effects and correlates of attachment security in childhood have demonstrated that the quality of a child's relationship with his caregiver at around 15 months, as assessed with Ainsworth's Strange Situation Procedure (SSP; Ainsworth, Blehar, Waters, & Wall, 1978), has a profound influence on later socio-emotional functioning (Waters et al., 2000; Main,

2000). The SSP stresses the child by leaving her both alone and alone with a stranger, and codes her response on being reunited with the caregiver after each of these separations. It is theorized that the child's ability to use the caregiver for soothing after this distress and to return to productive play with toys in the lab reveals her internalized sense of the caregiver's consistent availability and her own safety, so that the brief emergency can be recovered from and the child's attention turned to developmentally-appropriate engagement with her environment. Recent research is exploring whether the child's interaction with her mother this early in life may reflect an indelible pattern of stress response which will be exhibited across the life course, explaining individual differences in physical outcomes associated with stress.

It is possible that the SSP captures in the lab a young child's prevalent response or coping pattern to an immediate serious stressor – the mother leaving the child in a room alone. When the mother is reunited with the young child, the child's behavior is coded for proximity seeking, contact maintaining, avoidance of proximity and contact, and resistance to contact and comforting. A *secure* dyad is marked ultimately by the child's being effectively soothed and returning to the same level of play he was engaged in before the mother left the room. A child displays *insecure-avoidant* behavior by flat, withdrawn affect and avoiding the mother; the *insecure-resistant* category is marked by continued distress and the inability to be soothed or return to the same level of play reached before the separation. Consistent with John Bowlby's (1982) emphasis on the importance of experienced adversity, each of these infant attachment classifications is associated with observed parenting behavior: sensitive, contingent care; neglecting or

rejecting responses to the child's expressions of needs; and inconsistent care, respectively. It has been suggested that a secure relationship with the earliest primary caregiver fosters adaptive social regulation of stress neurobiology, and that frequent stress responded to with one of the insecure patterns of stress response may increase risk of physical and mental health problems, particularly in times of rapid brain development (Gunnar & Quevedo, 2007).

This idea was tested in a recent analysis of prospective assessment of earliest relationship functioning and its influence on adult health. In a rare longitudinal study, attachment security versus either of the insecure classifications at 18 months was shown to be a protective factor against physical illness at 32 years (Puig, Englund, Simpson, & Collins, 2013). However, the authors did not find a significant difference in the kind of symptoms reported by adults who had been classified anxious-resistant versus anxious-avoidant. In research on children's weight in particular, Anderson, Gooze, Lemeshow, & Whitaker (2012) found attachment insecurity as measured with the Attachment Q Sort (AQS; Waters & Deane, 1985) at 24 months predicted obesity in adolescence at 15 years. However, attachment insecurity at 15 and 36 months as assessed with the SSP, the gold standard for classifying infant attachment, did not predict obesity.

Theory and these recent findings suggest a couple of opportunities for exploring the effects of attachment security on weight and weight gain. Anderson et al. (2012) grouped all insecure children together, a practice which may mask very different stress apprehension and management strategies between the avoidant and anxious-resistant patterns. If the two insecure patterns do not have the same biological or psychological

patterns with respect to fat retention or eating, they might not differ in a common way from children with secure patterns of attachment. Puig et al. (2013) did test the different insecure classifications separately, but did not interact the patterns with the amount of stress the person had endured over her lifetime. If the insecure patterns differ in response to stress, then the amount of stress experienced could determine whether those differences emerge in weight outcomes. A recent study (Bichteler & Jacobvitz, under review) attempted to disaggregate insecure attachment patterns, stress response, and weight outcomes in adults. Adults' attachment representation was assessed in early adulthood, and their stress and weight outcomes in late-middle age. Insecure attachment classifications *dismissing* and *preoccupied*, conceptually similar to the infant classifications *–avoidant* and *–resistant* respectively, reflected very different patterns of stress response and weight gain under different levels of lifetime stress. Preoccupied speakers were heavier than secure and dismissing speakers under conditions of low stress and lost weight as stressful life events increased in number; dismissing speakers, who also reported using fewer coping strategies of any kind, gained weight under stress. The current study explored whether this interaction of experienced adversity and insecure attachment had the same influence on health outcomes during childhood.

To assess the strength of childhood adversity and attachment classification relative to known risks for obesity, it is necessary to include as many of these as possible in models predicting adiposity and change in adiposity over childhood. Biological, environmental, and socio-behavioral differences with robust predictive power should be

included in models testing the relatively new notion that different patterns of attachment behavior might moderate the influence of stress on health.

Biological mechanisms. As childhood obesity research and prevention emphases shift to the earliest months of life, biological mechanisms in pre-conception, perinatal, and postnatal periods have been identified. Biological factors shown consistently to predict risk of overweight and obesity when children are at the ages of 4.5 to 6 years include maternal smoking, maternal overweight, low socio-economic status, high birth weight, and rapid weight gain between 0 to 5 months (e.g. Dubois & Girard, 2006; Magarey et al., 2003; Li, Goran, Kaur, Nollen, & Ahluwalia, 2007). More controversial or ambiguous findings include ethnicity, gender, low birth weight and bottle feeding versus breast feeding in that set of risk factors (Dai, Labarthe, Grunbaum, Harrist, & Mueller, 2002; Wallace et al., 2013; Arenz, Rückerl, Koletzko, & von Kries, 2004). Dubois & Girard (2006) conducted a study on a representative sample of Canadian children born in 1998 (n = 2103) that incorporated a number of these biological factors simultaneously, in an effort to differentiate risks for obesity at age 4.5 years. Of the biological predictors at birth, child's gender, low birth weight, high birth weight, child's weight gain from 0-5 months, and maternal overweight remained significant when other environmental risk factors such as income were included. Prior ambiguous findings regarding low birth weight and bottle- versus breast-feeding may be confounded with the impact of early weight gain (Dietz & Gortmaker, 2001). Data sets including these variables should be exploited to supplement evidence (or not) of the influence of maternal prenatal nutrition and breast-feeding, as these questions have such cultural and

practical significance. The inclusion of known biological risk factors is important for determining not only their relative strengths, but also any effect an adversity-by-attachment classification term may have on childhood overweight/obesity.

Maternal overweight. Although maternal underweight and poor nutrition have been identified as risk factors for obesity, particularly in developing countries (Popkin, 1998), in the United States, maternal overweight is a more common concern. Prenatal over-nutrition may trigger permanent neuroendocrine and metabolic changes which account for the influence of maternal overweight on children's overweight over and above behavioral and environmental factors (Whitaker & Dietz, 1998). Even normal weight infants born to obese, overweight, or diabetic women have increased adiposity and risk for metabolic syndrome. Although the precise mechanisms are still unclear, it is thought that maternal inflammation and higher blood lipids activate proinflammatory pathways in the developing fetus permanently altering gene expression via epigenetic modifications, modifications that are maintained through feedback loops and create stable changes in the expression of metabolic genes and gene transcription (see Heerwagen, Miller, Barbour, & Friedman, 2010, for a review). Such developmental pathways are thought to be the primary route of adiposity transmission from mothers to children, rather than different attitudes or behaviors of obese mothers. In the one study associating overweight/obese mothers with infant feeding behavior, feeding behavior did not mediate the relation between maternal overweight/obesity and children's obesity (Lumeng et al., 2012). Whatever the mechanism, maternal overweight/obesity has been shown

consistently to predict childhood overweight/obesity, suggesting one route by which this epidemic propagates itself (e.g. Pryor et al., 2011; Magarey et al., 2003).

Birth weight for gestational age. Maternal overweight/obesity often, but not always, results in infant high birth weight for gestational age (> 90th percentile of weight for age; at full-term, defined as > 4000 grams / 8.8 pounds), a consistently significant predictor of overweight and obesity in childhood (Dubois & Gerard, 2006; see meta-analysis: Yu et al., 2011). High birth weight may be a sign of intra-uterine developmental changes that have programmed the infant's metabolic regulatory systems toward fat retention (see Heerwagen et al., 2010, for a review).

It is also true that the opposite, namely low birth weight, may be a risk for obesity. A few studies (but not all, c.f. Pryor et al., 2011; see Barker, 1997, for a review) have shown low birth weight for gestational age (< 10th percentile of weight for age; at full term, defined as < 2500 grams / 5.5 pounds) to be associated primarily with origins of heart disease, but also with later overweight/obesity. Two main theories are the fetal programming hypothesis, which states that growth restriction in the womb may program infants' physiology and metabolism to be susceptible to the symptoms of metabolic syndrome (Barker, 1997), and the fetal insulin hypothesis, which says that genetic variants influencing insulin secretion or action may both reduce fetal growth and trigger type 2 diabetes in later life (Hattersley & Tooke, 1999). A recent study evaluated these hypotheses by examining 37 potential genetic mediators that might disproportionately impact infants born small for gestational age and explain this phenomenon (Han et al., 2013). They found, however, that being small for gestational age itself was not

associated with obesity through age 11, but rather an interaction between the genetic variants and increasing BMI after birth did predict later obesity. This effect was found for both small- and average-for-gestational-age children, suggesting that poor outcomes associated with being small for gestational age had less to do with genetic variants and more to do with post-natal environmental factors. This recent work replicates findings reviewed by Ong & Loos (2006) detailing the mechanisms and risks for obesity associated with very early rapid weight gain.

Rapid weight gain. Infants in the upper quintiles of weight gain are more likely to be obese in early childhood, and then to have a higher risk of adult obesity, type 2 diabetes, and cardiovascular disease, both in the United States and in developing countries (Pryor et al., 2011; Dubois & Girard, 2006; Ong, Ahmed, Emmett, Preece, Dunger & ALSPAC Study Team, 2000; for reviews, see Jain & Singhal, 2012 and Ong & Loos, 2006). Precise mechanisms for this robust finding are still being identified, but generally it is suspected that the same neuroendocrine and metabolic changes observed in prenatal over-nutrition may account for the detrimental effects of postnatal over-nutrition as well.

Early adiposity rebound. "Adiposity rebound" refers to the normative, second rise in BMI that occurs between 5 and 7 years. An earlier adiposity rebound is associated with an increased risk of overweight, a robust phenomenon reviewed by Rolland-Cachera et al. (2006). Among obese children and adults, the mean age of the rebound is 3 years; among non-obese individuals, it is 6 years. The typical pattern associated with an early adiposity rebound is a low BMI followed by increased BMI level after the rebound

(Figure 4). Low BMI before the rebound may be the result of energy deficit at an early stage of growth caused by high-protein, low-fat diets. The protein triggers increased height, while the low-fat reduces energy intake. Similar to effects seen in infants small for gestational age, under-nutrition during the first years after birth may foster a "thrifty metabolism" that will exert adverse effects later in life, especially if the growing child is

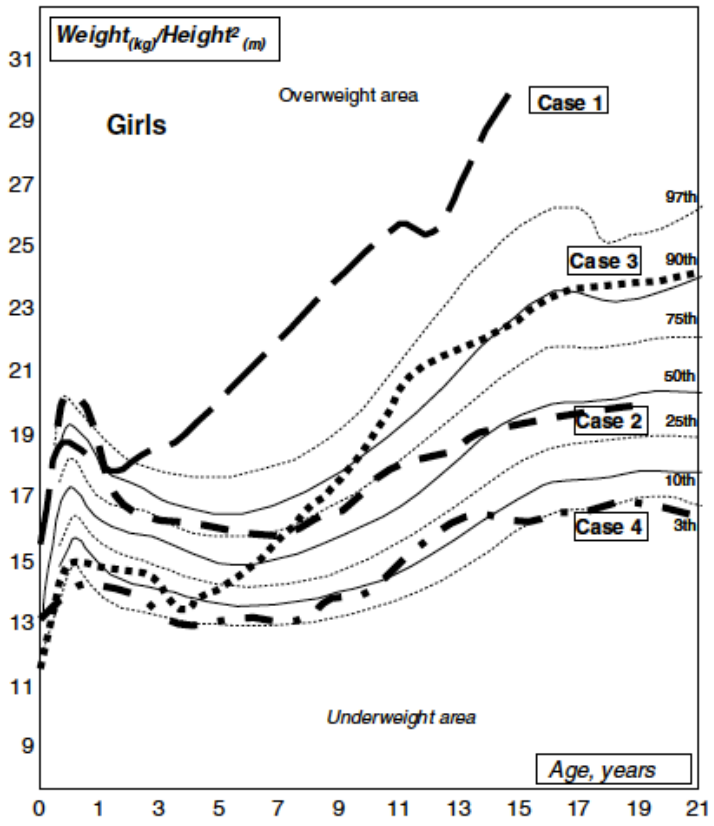

Figure 4. Four prototypical examples of BMI development plotted against French reference charts. Case no. 1, fat child at 1 year remained fat after an early adiposity rebound (2 years); case no. 2, fat child at 1 year did not stay fat after a late adiposity rebound (8 years); case no. 3, lean child at 1 year, became fat after an early adiposity rebound (4.5 years); case no. 4, lean child at 1 year, remained lean after a late adiposity rebound (8 years) (Rolland-Cachera et al., 2006, p. S12).

exposed to over-nutrition (Rolland-Cachera et al, 2006). This process may also explain some of the beneficial properties in high-fat, low-protein human milk versus formula.

Breast-feeding vs. bottle-feeding. Breast-feeding has been found to protect against later overweight and obesity in some, but not all, studies. It has been suggested that bottle-feeding triggers differences in macronutrient intake via hormonal mechanisms, and/or that higher protein and glucose concentrations could stimulate the development of fat cells and permanently alter glucose metabolism (see Ebbeling et al., 2002, for a summary). However, in studies where potential confounders such as maternal overweight, maternal smoking, catch-up weight gain, and socio-economic status are included, the influence of breast- versus bottle-feeding seems to disappear (Reilly et al., 2005; Dubois & Girard, 2006; Parsons, Power, & Manor, 2003). It may be that breast-feeding is protective in some trajectories of weight gain, but not all.

Maternal smoking. Maternal smoking in any amount during pregnancy has been shown consistently to be associated with risk for obesity in early childhood (Pryor et al., 2011; Reilly et al., 2005; see Behl et al., 2013 for a review). Its potential to differentiate a low birth weight and low SES trajectory classes makes maternal smoking an important covariate in weight modeling.

Environmental and social risk factors.

Socio-economic status (SES). The CDC reported that 1 in 3 low-income children surveyed were overweight or obese in 2009 (CDC, 2014). From 2003 to 2007, obesity prevalence increased by 10% for all U.S. children but increased by 23%–33% for children in low-education, low-income, and higher unemployment households (Singh, Siahpush, & Kogan, 2010). However, these figures did not control for parental overweight/obesity, which when included, weakens the relation between low SES in

early life and later obesity (for a review of longitudinal studies, see Parsons, Power, Logan, & Summerbell, 1999). Dubois & Girard (2006) did control for maternal BMI, and SES measured as household income retained its inverse relationship with childhood obesity at 4.5 years, as it did in the Li et al. (2007) longitudinal growth trajectory study. Pryor et al. (2011) found no effect for income or education, but this may be because the study population was not representative of the United States: the study was in Quebec and the population 93% white. The increasing prevalence of obesity in the United States among low-income, low-education, minority mothers (Ogden, Lamb, Carroll, & Flegal, 2010) makes SES a key covariate in any analysis including maternal BMI and ethnicity.

Race and Ethnicity. As the genetic make-up of all ethnic groups has been stable in recent history, and race/ethnicity itself is considered more a social than biological construct, it is included here as a risk in interaction with the increasingly obesogenic environment. The prevalence of childhood obesity among African Americans, Mexican Americans, and Native Americans exceeds that of other ethnic groups. The Centers for Disease Control and Prevention reported that in 2000 the prevalence of obesity was 19% of non-Hispanic black children and 20% of Mexican American children, compared with 11% of non-Hispanic white children (CDC, 2012). The increase since 1980 is particularly evident among non-Hispanic black and Mexican American adolescents.

Between NHANES III (1988-1994) and the 2007-2008 NHANES biennial, ethnic differences in the prevalence of obesity in middle childhood and adolescence emerged. Prevalence increased from 11.6% to 16.7% among non-Hispanic White boys; from 10.7% to 19.8% among non-Hispanic Black boys; and from 14.1% to 26.8% among

Mexican-American boys. Among girls, the significant differences between non-Hispanic Black and non-Hispanic White were retained between NHANES III (1988-1994) and the 2007-2008 NHANES biennial, with all levels rising: from 8.9% to 14.5% among non-Hispanic White girls; from 16.3% to 29.2% among non-Hispanic Black girls; and from 13.4% to 17.4% among Mexican-American girls (CDC, 2012).

The mechanisms of these differences associated with race/ethnicity are not well-understood, but may consist of stress induced by minority status and discrimination, low SES, difference in adipose tissue and fat retention, differences in pubertal maturation, and differences in parental obesity. Ethnic differences in weight gain in the earliest years of childhood are less well-documented, and given the influence of early BMI on later BMI, should be analyzed.

Gender. In Dubois & Girard's (2006) prospective cohort study, boys were more likely to have been born with high birth weight and more likely to be obese at 4.5 years. Other studies with similar methodology have found no difference between the genders (Pryor et al., 2011; Reilly et al., 2005). Gender differences in general and as predictors of different weight trajectory classes are important for determining pediatric risk models.

Maternal sensitivity. Anderson et al. (2012) showed that maternal insensitivity in the earliest years of life predicted adolescent obesity at 15 years, perhaps through its contribution to chronic stress. Their study tested maternal sensitivity in structured interactions at 15, 24, and 36 months in the NICHD SECCYD dataset. Maternal sensitivity at 15 and 24 months was the sum of ratings of sensitivity to nondistress,

positive regard, and intrusiveness (reverse coded); sensitivity at 36 months was the sum of supportive presence, respect for autonomy, and hostility (reverse coded).

Insensitivity during feeding itself has also been suggested as a means by which children's eating becomes dysregulated. Intrusiveness and assertive prompting during a standardized snack procedure at 15, 24, and 36 months have been associated both with overweight in children at 24 and 36 months and with the general maternal sensitivity ratings in the NICHD SECCYD data (Lumeng et al., 2012). It is possible that sensitive caregiving may be associated with learning satiety cues and managing stress adaptively.

Research Questions 2a and 2b

After latent classes with different growth curves of childhood weight gain are identified, of key interest will be the composition of these classes. The commonalities shared among members of higher-risk versus lower-risk classes could inform future risk modeling for different ages and identify when important predictors are the most influential and the best targets for prevention efforts. Extensive exploration of the composition of these classes will address these questions, as components of the second set of research questions:

2a. Do the robust risk factors for childhood obesity identified in prior research differentiate membership in the latent growth classes?

2b. Does any influence of early childhood adversity on membership in weight growth trajectories vary according to infant attachment classification?

METABOLIC SYNDROME

One of the main reasons childhood and adolescent overweight and obesity are of such concern is for their suspected influence on metabolic syndrome in adolescence and adulthood. Metabolic syndrome, associated with cardiovascular disease (CVD), is diagnosed when at least three of a cluster of these five abnormal conditions is present: 1) large waist circumference / abdominal obesity; 2) high triglyceride level; 3) low HDL, or "good" cholesterol level; 4) high blood pressure; and 5) high fasting blood sugar (United States Department of Health & Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute, 2011). Based on the Third National Health and Nutrition Examination Survey (NHANES III) in 1988-1994, the overall prevalence of metabolic syndrome among adolescents aged 12 to 19 years was 4.2%; males had significantly higher prevalence (6.1%) than did females (2.1%). Among obese and overweight adolescents, metabolic syndrome was present in 28.7% and 6.8% respectively. In contrast, it was present in only 0.1% of those with a BMI below the 85th percentile (Cook, Weitzman, Auinger, Nguyen, & Dietz, 2003). By the NHANES 1999-2000 biennial, prevalence had increased to 9.1% for boys and 3.7% for girls, with 32.1% of obese and 7.1% of overweight adolescents diagnosed. As the NHANES surveys use complex, multistage, probability sampling, a population weighted estimate can be calculated: >2 million adolescents had a metabolic syndrome phenotype at that time (Duncan, Li, & Zhou, 2004).

Heart disease remains the leading cause of death in the United States (Hoyert & Xu, 2012). Both childhood overweight and change in weight percentile are associated with adult obesity and metabolic syndrome (Lloyd, Langley-Evans, & McMullen, 2012),

and metabolic syndrome is in turn a significant predictor of adult CVD. However, direct relations between childhood adiposity and adult CVD risk are weak (Freedman, Kahn, Dietz, Srinivasan, & Berenson, 2001), perhaps because different developmental trajectories of adiposity, metabolic syndrome, and CVD have not been established. Attempting to quantify the risk for metabolic syndrome in childhood and its utility in predicting future CVD risk is a relatively recent innovation (Sovio et al., 2013). In 2008, a small study of adults, those who had been diagnosed with pediatric metabolic syndrome experienced an incidence of CVD of 19.4%, compared with 1.5% for those without metabolic syndrome as children. In multivariate logistic regressions, both pediatric metabolic syndrome and age significantly predicted both CVD and type 2 diabetes (Morrison, Friedman, Wang, & Glueck, 2008). Pediatric metabolic syndrome has likewise been associated with type 2 diabetes in adulthood. The paucity of longitudinal data sets on children that include clinical outcomes has posed a particular challenge to modeling risk among children and adolescents.

Often, not all of the components of metabolic syndrome are present in adolescents, and a continuous metabolic risk score is assessed instead (Ekelund et al., 2012; Lloyd et al., 2012). In a study of metabolic risk score in late adolescence, rapid weight gain in earliest childhood again emerged as a strong, independent predictor and remained when models were adjusted for birth weight, maternal fat mass, SES, current fat mass, and weight gain in later childhood (Ekelund et al., 2012). Being small for gestational age has also been associated with metabolic syndrome as a whole as well as with the individual components waist circumference and blood pressure (see review in

Harville et al., 2012; Levy-Marchal & Jaquet, 2004). Clearly, understanding different trajectories of birth weight, early weight gain, and weight gain in middle childhood will be key to predicting metabolic risk factors in adolescence. I addressed the third research question to this end:

Research Question 3

Which classes of weight trajectory in childhood predict overweight and high blood pressure in adolescence?

THE CURRENT STUDY

The current study augmented existing research on overweight and obesity in childhood in several important methodological and analytical ways. Most prior research on patterns of weight gain through childhood has been cross-sectional in nature and has considered all children together as one homogenous group. This lack of differentiation has masked differences in developmental timing of critical weight-related changes, such as maternal BMI and weight gain after birth. This study included modeling prospective, longitudinal data from birth to age 15 ½ years old, generating comprehensive growth curves for different homogeneous childhood trajectories.

These trajectories of normative and non-normative change over time provided the basis for modeling that can differentiate risk profiles for different groups of children. Modeling behavioral factors, such as breastfeeding and smoking, with biological factors, such as birth weight for gestational age, weight increase, and maternal BMI, demonstrated the relative strength of these risks in predicting childhood weight gain. Pursuing a new avenue of individual differences, these analyses also included moderation

of the effects of childhood adversity on weight gain by infant attachment security classification. Conceptualizing the Strange Situation Procedure as a measure of both biological and behavioral stress response suggests new hypotheses regarding the heretofore ambiguous relations between childhood stress and weight gain. Testing adversity in the context of attachment classification along with other known risks to predict trajectory class membership yielded insights into impact of early life stress. Rather than limiting the scope of research to one or two constructs of interest, including as many of these factors as possible allowed exploration of early risk profiles as predictors of children's weight growth curves.

Finally, I sought to use these trajectory classes to contribute to an understanding of the etiology of metabolic syndrome and heart disease. Findings relating childhood BMI at various stages with adolescent symptoms of metabolic syndrome have remained ambiguous. Examples from the work reviewed here include findings that high birth weight is protective against later heart disease, but also predicts increase in obesity risk as well. Added to such complexities is recent lifespan research showing psychological patterns such as attachment anxiety to be associated with more inflammatory illness in later life. The current study, having defined different classes of growth trajectory and then coalesced the risk profiles for their membership, ultimately enabled prediction of two metabolic symptoms in adolescence – overweight and high blood pressure – significantly advancing risk modeling and informing future interventions to combat the leading cause of death in the U.S.

Meeting these goals was possible due to the rich dataset generated by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD). This dataset includes a wealth of complex and time-varying measures, such as numerous waves of BMI, demographic, and social status from birth to 15 ½ years, the Strange Situation Procedure, family stress and adversity, mental and physical health, sensitivity, childcare, and some metabolic symptoms at age 15 ½, among many others. This secondary data analysis using growth curve modeling took fullest advantage of these, and ANOVA, multinomial regression, and generalized linear modeling were brought to bear to analyze trajectory membership and health outcomes.

Method

A secondary data analysis using all four phases of data collected in the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD) addressed the research questions. The SECCYD is a comprehensive study of children and families from birth to age 15 ½ years, with the primary goal of evaluating child care experiences, characteristics of different child care setting, and children's developmental outcomes.

In 1991, a team of researchers enrolled 1,364 diverse children and families from 10 different sites across the nation. The sampling strategy focused on ensuring a sufficient size sample for factors of primary interest, e.g. maternal work outside the home, single mothers, minority representation. Analyses have shown that the data collected do reflect the natural distributions of these characteristics in the catchment and in the 1990 census data, but as a true probability sampling design was not used, the SECCYD data cannot be interpreted as representative of the general population. A total of 4 phases collected a total of 38 waves of data (Figure 5).

PARTICIPANTS

1,364 families were recruited from 24 hospitals at 10 sites across the United States just after the birth of a child. Conditional random sampling was designed to prevent selection bias and to ensure a proportion of minority and low-SES families were included. Detailed information on study design and procedures is available at http://www.nichd.nih.gov/research/supported/Pages/seccyd.aspx.

Year	Children's Ages or Grade	Children (N)	Data Collection Waves
1991-94	Phase I, ages 0-3	1,364	Home/Lab: 1, 6, 15, 24, and 36 Phone surveys every 3 months
1995-99	Phase II, through 1st Grade	1,226	Home/Lab: 54 months, first grade Phone surveys 42, 46, 50, 60, 66, K
2000-04	Phase III, through 6th Grade	1,061	Home/Lab: 3rd and 5th grades Lab only: 4th and 6th grades Phone surveys 2nd – 6th grades
2005-2007	Phase IV, through 9th Grade	1,009	Home/Lab: 9th grade Phone surveys 7-9th grade HPDA[1]: 12 ½, 13 ½, 14 ½, 15 ½

[1] Health and Physical Development Assessment

Figure 5. Overview of data collection phases and waves in the SECCYD.

Four phases of the study comprising 32 waves of data collection were completed. The first phase, from 1991-1994, evaluated children starting at one month of age and collected 12 waves of data over 3 years. Phase II (1995-1999) studied 1,220 of the original cohort from first grade, focusing on quantifying the effects of early child care across 10 waves of data collection. At Phase III (2000-2004) the cohort of over 1,110 enrolled children and families were followed with 6 waves of data collection through sixth grade, with an emphasis on middle childhood within the modern social ecology of work and family. A cohort of 1,056 children participated in Phase IV (2005-2008), which added measures of adolescent contexts, social relationships, health, and academic

performance to ongoing repeated measures of family relationships and social status in 4 annual waves. For the current study, only participants born at early term through late term who also had sufficient data available for modeling were included; details of sample selection and sample differences analysis are found at the beginning of the Results.

PROCEDURE

SECCYD measures analyzed for the current study were collected in multiple ways: by medical professionals, trained technicians or coders, by trained observers and interviewers, and by maternal report.

MEASURES

Body mass index (BMI). BMI is a simple weight to height ratio (kg/m^2). Raw body mass index is not recommended as a measure of children's central adiposity as it is not standardized for growth in early life and rises monotonically (CDC, 2014). Thus the CDC does not categorize childhood overweight and obesity by absolute cut-offs as it does for adults; rather, it has adopted the $>85^{th}$ percentile and $>95^{th}$ percentile thresholds for childhood overweight and obesity, respectively (CDC, 2014). The CDC's (2000) growth charts are used as the standard for BMI percentiles for age (Kuczmarski et al., 2000).

BMI is the most frequent assessment of body fat, and the World Health Organization and CDC base their standards against it. BMI has a sufficiently strong relation to body fat mass to be a reliable indicator of adverse levels of blood lipids in children and adolescents (Dai et al., 2011) and is a better predictor of children's and adolescents' insulin and blood pressure values than are estimates of percentage body fat

(Freedman, Horlick, & Berenson, 2013). High BMI percentile levels and changes in parameters of BMI curves in children are also linked to significant levels of risk for adult obesity at corresponding high percentile levels (see Duren et al., 2008, for a review of body composition measures).

Weight and length/height were measured by trained technicians to eliminate any bias in maternal reports. Gestational age, birth weight, and length were retrieved from birth certificates. BMI percentiles are not calculated by the CDC before 24 months, as the fluctuation before 2 years old is considered too great to be a reliable marker. Raw BMI at 15 months was used as a predictor of trajectory membership. BMI percentile measured at months 24, 36, 54, and at grades 1, 3, 5, 6, 7, and 8 were used for trajectory modeling.

Childhood adversities. Stressful family and social circumstances found to have contributed to adversity-related weight outcomes in prior research (Gundersen et al., 2011) guided the selection of survey items comprising childhood adversity in the current study. Particular emphasis was given to earliest childhood, when trajectories of weight gain were expected to be differentiated. A maternal total stress score indexed whether any of these events had occurred since the last interview: job loss of close friend or relative; illness/injury/addiction of close friend or relative; death of anyone close; any other major life event occurred; and the number of times household had moved. The average of mother-reported stresses from 0-15 months was used to predict the trajectory latent classes. A second stress item, a one-time comprehensive maternal report of total negative life events at 54 months, was used as well. Mother's general assessment of her

physical health was assessed via questionnaire at 1, 6, 15, and 24 months, as was the CES-D measure of depression (Radloff, 1977). Each of these assessments was tested for its association with trajectory class membership.

Infant attachment classification. The Infant Strange Situation Procedure (SSP) was administered in the lab to mother-child dyads at both 15 and 36 months. The SSP protocol is designed and validated for ages 9-18 months (Ainsworth et al., 1978), therefore the current study will focus on the attachment category identified at 15 months. Categories will not be merged into secure versus insecure; the insecure categories anxious-avoidant and anxious-ambivalent/resistant will be kept separate for analyses, as they are theorized to represent very different strategies for managing stressful situations.

Maternal overweight. There is no measure of maternal weight or height included in the SECCYD dataset until the study child reached age 15 ½. This analysis used weight status assigned by Lumeng et al. (2012) from coding mothers' videotaped images when children were 15 and 24 months old. Figure ratings based on pictorial scales have a high correlation with BMI (Cardinal, Kaciroti, & Lumeng, 2006). Lumeng et al. used 2 independent trained observers to code maternal figure using the Stunkard Figure Rating Scale from 1-9 (Stunkard, Sorensen, & Schulsinger, 1983), where higher scores represent higher BMI. Intraclass correlation coefficients exceeded .80 (Lumeng et al., 2012).

Birth weight for gestational age. Birth weight in grams and gestational age in weeks were recorded from birth records at time of recruitment. Birth weight for gestational age (birth weight in grams divided by gestational age in weeks) was used as a

continuous independent variable. The raw birth weight was adjusted by the modeled relation between birth weight and gestational age for the entire dataset.

Maternal prenatal smoking. Maternal smoking prenatally was assessed retrospectively at 24 months using the following response categories: 1) Did not smoke; 2) Smoked, but quit before pregnancy began; 3) Smoked, and stopped during first three months of pregnancy; 4) Smoked during first 3 months, stopped prior to birth; and 5) Smoked throughout the year. This variable was used as a scale from 1-5.

Rapid weight gain. Early weight gain was calculated by the percentage of birth weight for age gained between birth and 15 months. This change score will be modeled in its continuous form, so that the point at which the amount of weight gain becomes influential can be determined from the data, rather than defined at the outset.

Breast-feeding versus bottle feeding. Whether the mother was breast- or bottle-feeding the target child, and the month she had stopped breast-feeding if she had been previously, was assessed at the 1, 6, 15, 24, and 36 month interviews. However, maternal reports of duration varied wildly across waves. Thus, this variable was coded as a binary indicator of whether the mother had ever breastfed, as assessed at the 1-month wave.

Socioeconomic status (SES). The family's average income-to-needs ratio from birth – 24 months and maternal education level at 1 month were derived from mother reports and were used as indicators of socioeconomic status.

Race & Hispanic Ethnicity. Race was reported by the mother at time of recruitment as American Indian / Eskimo / Aleutian, Asian / Pacific Islander, Black or Afro-American, White, or Other. In both trajectory and outcome modeling, the only

contrast of significance to emerge was vis-à-vis the Black classification; thus a Black = 1 code was used to simplify analyses. The indicator for Hispanic was likewise binomial.

Maternal sensitivity. Maternal sensitivity was coded during structured interactions throughout childhood. In Phase I at 6, 15, and 24 months, mothers and children engaged in structured play interactions at home (6 months) or in a university laboratory (15 and 24 months), and mothers were coded for their sensitivity to non-distress, their positive regard, and their intrusiveness (reverse coded). Average sensitivity was calculated across these time points.

Markers for metabolic syndrome. Two markers for metabolic syndrome were available for modeling in the SECCYD dataset: central adiposity and blood pressure. Each was tested for associations with early life predictors and with growth class membership.

Central adiposity. Central adiposity at 15 ½ years was defined and tested in several ways: continuously as percentile BMI and waist circumference, and binomially as overweight (> 85th percentile BMI) and obese (> 95th percentile BMI).

High blood pressure (> 90th percentile). The U.S. National Heart, Lung, and Blood Institute's age-, gender- and height- specific tables for blood pressure percentiles in adolescents, with a floor of 120/80, were used to generate this binary term indicating pre-hypertension (National Heart, Lung, and Blood Institute, 2004).

SECCYD site. Children and families were drawn from 10 different sites in the United States for the SECCYD study. It is possible that growth trajectories and/or relations with predictors and outcomes of those trajectories differ across site. In an early

analysis of non-maternal care and child outcomes conducted by the steering committee of the SECCYD (NICHD, 1997), the authors recommended evaluating site as a potential confound and testing its association with independent variables to determine whether it should be included in models. In that study, site was significantly associated with the type of child care families chose, and was therefore controlled for in OLS regression models. In regressions using the logit link function, however, it was not, as the associated reduction in power was too great. Much of the most recent published developmental/psychological research using the SECCYD data (list found at http://www.icpsr.umich.edu/icpsrweb/ICPSR/series/233) does not control for site, (e.g. Laible, McGinley, Carlo, Augustine, & Murphy, 2013; Kamper & Ostrov, 2013; Anderson et al., 2012; Lumeng et al., 2012). This study will take the approach used by the NICHD steering committee, testing site for direct and moderating effects; sensitivity analysis based on the AIC and any relations with covariates will determine whether the added variable is required. As with the other independent variables, those without significant relations to each other or to class membership will be omitted from the final models, to prevent over-adjustment (Schisterman, Cole, & Platt, 2009).

DATA ANALYSIS PLAN

Addressing RQ 1: Identifying classes of BMI growth trajectories.

Determining the classes of trajectories of children's weight profiles over time was a multi-stage process. The end goal was to combine individuals' trajectories into the smallest number of homogenous but ecologically meaningful growth classes. The first step was to fit latent growth curves, with intercept, slope, quadratic, and possibly cubic

terms, to the entire sample. Latent growth curve modeling has an advantage over traditional repeated measures analyses in that it fits a line to all of the observed points to create individuals' developmental trajectories over time. Depending on the modeling strategy (latent class growth analysis or growth mixture modeling—see the distinction, below), individuals' intercepts, slopes, and variance may be allowed to differ from other individuals' values within the class to a greater or lesser degree. Forcing these values to be very similar typically results in a greater number of classes identified, but these classes may not be meaningfully different enough in terms of absolute change to warrant their being split. Allowing them to vary too much within class will not differentiate the classes sufficiently. The effort to strike this balance between homogeneity and ecological meaningfulness has encouraged a proliferation of model fit indices, statistical tests for the accuracy of modeled class membership, and model comparisons, but none of these is definitive; as with other types of cluster analysis, there remains some element of subjective judgment. In the current study, several methods of class determination and model testing (described below) were used until the most rational number of classes was identified.

Growth mixture modeling. Introduced by Muthén & Shedden (1999), *growth mixture modeling* (GMM) considers different classes of individuals to vary around different mean growth curves. Latent class variables, either categorical or continuous, are defined to capture these groups of individual growth trajectories, resulting in separate growth models for each class (cf. Muthén & Asparaouhov, 2006). When the latent class

variable is categorical, it represents the mixture of subpopulations where membership is inferred from the data.

Allowing growth factor variances to vary within classes allows greater heterogeneity within the class, which can result in fewer classes being identified. However, the modeling flexibility to allow separate growth models and unique estimates of variance for each class raises particular challenges for GMM. Mathematically modeling a distribution of mixtures of many sub-distributions is extremely computationally intensive, and prone to convergence issues due to likelihood estimation problems (although it has been done successfully in a study of children's BMI trajectories, see Li et al., 2007; see Jung & Wickrama, 2008, for an overview).

A group-based approach, which fixes the variance and covariance estimates for the growth factors within each class to zero, such as latent class growth analysis (LCGA), is considerably simpler to estimate. In this study, I used both GMM and LCGA and compared them, to arrive at the optimal solution.

Latent class growth analysis. Models assuming all individual growth trajectories within a class to be homogenous have been developed extensively by Nagin and colleagues, (see Nagin, 2005, for a review). In the SEM context, Muthén (2004) classified this approach as a simpler specification of GMM and labeled it *latent class growth analysis* (LCGA). Latent class growth analysis allows groups of individuals to have different mean growth curves, yielding different homogenous trajectory classes, but does not allow variation around those means. This approach may have the disadvantage that too many classes are identified because growth factor variances are not allowed to

vary within class. However, LCGA may also be used as a step prior to conducting GMM, as distinct classes can be identified, then explored to determine whether some may remain with the homogenous assumption, thus limiting computational intensity, and some allowed to merge, using the GMM parameterization (for a comparison of LCGA and GMM modeling, see Jung & Wickrama, 2008).

Model fit. As in other finite mixture modeling, models with increasing numbers of classes were fit to the data and then fit statistics were compared. No absolute measure of fit determined which model to retain; rather, as the number of classes increased, the fit statistics of the models were evaluated, until the most parsimonious, practical, but discriminating classes were defined. The following fit statistics were evaluated:

Bayesian Information Criterion (BIC). The most common fit statistic used to determine the number of latent classes is the BIC (Shwartz, 1978), which, similar to the Akaike Information Criterion (AIC), penalizes model fit as the number of parameters rises. This penalty is intended to prevent over-fitting a model and uses Bayesian estimation of the likelihood function.

Lo-Mendell-Rubin Likelihood Ratio Test (LMR LRT). A traditional chi-square difference test between a model of *k* classes vs. a model of *k*-1 classes will not be accurate, because the difference in the probabilities of class membership is not chi-square distributed when the likelihood of being in the *k*th class is zero when the *k*-1 class is being specified. The LMR LRT fit statistic is an improvement over the traditional chi-square difference test because it uses the correct distribution of the difference between the two models' log likelihoods (Lo, Mendell, & Rubin, 2001). Simulation testing has

established that the LMR LRT is more accurate at distinguishing the *k*- from the *k*-1 class model than the *k*- from the *k* + 1 class model. Thus, as the number of classes tested increases, and the *p*-value indicates a non-significant difference at *k* classes, we will interpret that there are at most *k*-1 classes in the optimal model (Nylund, Asparouhov, & Muthén, 2007).

Mean posterior probabilities. In the context of latent classes in SEM, the posterior probability refers to the probability of an individual's being grouped into each class, given the model parameters. If it is found that individuals with a high probability of being in one class also have a moderate probability of being in another class, for example, these classes may not be sufficiently distinguished (Muthén, 2002), and additional differentiation is required.

Entropy. Although not technically a fit statistic, entropy is a useful index of the quality of class cohesion. It uses a weighted average of posterior probabilities to quantify the strength of prediction of class membership given the observed indicators. Values range from 0 to 1, and high values (> 0.70) indicate that the latent classes are highly discriminative (Nagin, 2005).

Addressing RQ #2: Characterizing trajectory class membership. Once the trajectory classes and membership were identified, ANOVA tests and logistic regressions were used to determine how continuous and binomial variables, respectively, differed across class. To address question 2b regarding the influence of childhood adversity and any moderation by attachment classification, the sum of maternal stresses endured between 1 and 15 months was interacted with the three levels of organized attachment

classification in logistic regressions predicting membership in each class. (Children classified as disorganized were tested using their next organized classification, as the *disorganized* classification had no specific hypothesis associated with it and did not emerge significantly in modeling.)

The effect sizes for the logistic regressions were calculated using the R-squared measures for binomial GLMs proposed by Tjur (2009), which he termed the "coefficient of discrimination." Tjur's R-squared is calculated as the difference between the two means of the predicted probability for each category of a binomial variable and has been shown to equal the mean of the R-squared formulas based on the squared residuals.

Addressing RQ 3: Predicting symptoms of metabolic syndrome. In predicting adiposity at 15 ½ years, the continuous outcomes percentile BMI and waist circumference were modeled with linear regression, and the over-85th and over-95th binomial cut-offs with logistic regression. Systolic blood pressure was likewise modeled as high (=1) or not high. All the candidate early-life risks were used as predictors of metabolic syndrome along with weight trajectory, to determine the relative strengths of any effects; models were then refined based on the AIC and significance testing to generate the most parsimonious and explanatory models for the health outcomes.

Results

Data Preparation

The SECCYD (NICHD) dataset comprises 1364 newborns and families, enrolled from hospitals in 10 different sites across the United States. As this study was concerned primarily with identifying normative classes of weight change over time, only infants who were born early term through late term (37-42 weeks; Spong, 2013) were included for analysis; 56 children were omitted (54 born earlier than 37 weeks; 2 born after 42 weeks). Sixteen infants had no gestational age recorded; however, as their birth weights fell well within the range of the full term set, they were retained. Given the focus on growth trajectories, I required that children have two or more waves of data to be included in the analyses; 172 children were excluded for this reason, either because they dropped out directly after enrollment or because they did not have at least two measurements between 24 months and 8th grade. Four individuals were dropped during evaluation of the growth mixture modeling. These children had measurements at the first three time points only, and GMM optimization placed them into a fourth class of their own. As this class comprised only 0.35% of the population and could not describe a trajectory meaningfully due to the pattern of their missing data, the four individuals were dropped. These criteria resulted in the exclusion of a total of 232 children. Thus, the final sample for these growth analyses across nine time points, from 24 months to 8th grade, was 1,132 children.

The 232 children omitted from the study were compared to the included sample, to determine whether any significant differences in family income at birth, gender, ethnicity, or study site existed between the two sets. In binomial regressions, where

being dropped from the analysis was coded as 1, the association between being dropped and total family income was not significant (B = -2.7 e-07, p = 0.28). Gender was unrelated to being dropped (F (1, 1362) = 1.55, p = .21), as was ethnicity (F (4, 1359) = 1.43, p = .22) and site (F (9, 1354) = 1.31, p = .23). Tukey's Honest Significant Differences (Tukey's HSD) test demonstrated that none of the pairwise contrasts between ethnicities or sites differed significantly. Table 1 contains the demographic characteristics of the final sample.

Next the presence of outliers was investigated. Visual inspection of individual growth trajectories and an analysis of variation within individuals revealed a number of individuals with outlying data points. For example, one child's BMI plunged from the 60th percentile to the 0.1st percentile, while another's went from the 10th to the 85th percentile and back to the 10th again. Rather than drop such anomalous individuals entirely, a data trimming approach was taken; only the outlying data point was dropped.

Table 1.

Demographic characteristics of the total sample (N = 1,132) and broken out by race.

	Percent/average (range) total sample	Black/ AA	White	Asian/PI	AIEA	Other
Race		12.1 %	81.1 %	1.5 %	0.4 %	4.9 %
Hispanic	5.9%	1.5 %	4.8 %	17.6 %	60.0 %	27.3 %
Gender: Female	49.1 %	48.2 %	48.9 %	58.8 %	40.0 %	52.7 %
Income-to-need[1]	2.83 (.02 – 25.08)	1.2	3.1	3.0	1.4	2.5
Maternal education[2]	14.3 (7 – 21)	13.0	14.6	15.8	14.0	13.3

[1]Income-to-need ratio (range) is averaged from 0-24 months.
[2]Maternal education in years at child's birth.
Note. Black/AA = Black or Afro-American. Asian/PI = Asian or Pacific Islander. AIEA = American Indian, Eskimo, or Aleutian.

Outlying points were identified by fitting the highest order polynomial growth curve possible (depending on the number of data points present, including up to a cubic term) to each individual, then determining which standardized residual(s) fell above the 95th percentile. No individual was identified with more than one outlying point by this criterion. Taken together, 331 data points out of a total of 7,678 (4.3%) of the total, were dropped, allowing Mplus to execute its full information maximum likelihood (FIML) estimation for missing data in their place. This data trimming strategy allowed the retention of individuals with high variation in BMI percentile, as well as individuals with a data point which was in error or due to an unusual circumstance (e.g. illness). Trimming the data points resulted in more cohesive classes, as assessed by model entropy, compared to dropping the individuals altogether, indicating that the included individuals did contribute to the definition of normative classes. The inclusion of the data for these individuals also resulted in fewer GMM classes identified, rather than more, adding evidence that they were not outlying as a whole and should be retained.

FIML estimation was likewise used in the presence of missing data in all analyses reported below. The entire dataset was analyzed for attrition and patterns of missingness, which might have resulted in some time points or demographic characteristics being weighted more heavily in the growth curve analyses than others. Participation in BMI measurements decreased over time. For each year from 24 years to 8th grade, the measurements available at each time point dropped on average by approximately 3 (B = -0.51, SE =0.11, $p < .01$); there were 927 measurements at 24 months and 704 at 8th grade. Individuals' family income at birth, gender, ethnicity, and study site were analyzed with

respect to the number of measurements reported for them. Family income at enrollment was significantly negatively related to the number of time points the child participated in (B = -5.39 e-06, SE = 2.03e-06, $p < .01$), however the variance in missingness explained by family income was very small (adjusted $R^2 = 0.004$). Girls were more likely to have missing values than boys ($F(1, 1362) = 5.43$, $p < .05$). Site was related to time point missingness ($F(9, 1354) = 4.19$, $p < .001$). However, Tukey's HSD tests revealed that only four contrasts were significant out of the 45 tested: site 1 differed from sites 0, 3, and 5; site 3 also differed from site 4. Ethnicity was unrelated to time point missingness (F (4, 1359) = 1.27, p = .28).

Finally, the time points were centered around the mean time point in months, which eliminated collinearity between the growth factors and the time points. The time point intervals were then scaled by 1/100, to ease the calculation of the cubic term for the optimization algorithm. Thus, the intercept of the growth classes was not the starting point of the trajectory, but rather its mean BMI percentile at the mean time point.

RQ 1: Unconditional Models of Growth Trajectories

Data analysis began with latent growth modeling, a family of analyses which estimates latent variables that characterize individuals' growth, much as random and mixed effects models and cluster analysis do (Muthen, 2002). The latent variables in these models were factors common to each class – the intercept, slope, quadratic, and cubic terms – which defined the change over time, or "growth." An iterative process analogous to exploratory cluster analysis resulted in selecting a final growth model by striking a balance between the most mathematically coherent set of classes and the set of

classes with the most ecological meaning (Jung & Wickrama, 2008; Muthen, 2003). Growth mixture modeling (GMM) allows for differences in growth factors across unobserved subpopulations by identifying groups of individual growth trajectories (latent classes) which vary around different means. This method allows separate growth models to be estimated for each latent class. Latent class growth analysis (LCGA) is a restricted type of GMM in which the variance and covariance estimates for the growth factors defining each latent class are assumed to be zero. This approach is computationally easier; therefore a typical exploration of latent class analysis begins with LCGA.

Following this progression, in this study I started with LCGA, in which individuals were assumed to have homogeneous growth trajectories within each class, then moved to GMM, in which the variances were left free to be estimated, which yielded a better model fit and generated a smaller number of larger classes. The growth factors and variation in the best LCGA and GMM models were then reviewed against each other and the raw data to refine a combination of factor values, class membership, and variance estimation to yield a class solution with the best balance of parsimony and meaning (Muthen, 2003). All of the models used to identify classes were unconditional; that is, they did not include covariates or predictors of membership, only BMI percentiles at the nine time points between 24 months and 8th grade.

Growth trajectory class solutions were compared using several standard measures of relative fit. The log of the likelihood (LL) that the given model produced the observed BMI percentiles rises as models improve. However, a simple comparison of LLs across models is not a good measure of fit, as the LL must always rise as additional parameters

are estimated in a model. The Akaike Information Criterion (AIC) and the Bayesian Information Criterion (BIC) penalize the improvement in LL for additional parameters (AIC) and for sample size (BIC) and fall in value as models improve. A chi-square test of the difference in LL between two models provides a simple measure of improvement, but the Lo-Mendell-Rubin Likelihood Ratio Test (LMR LRT) takes into account that this difference is not normally distributed and provides a robust significance test for rejecting a model with the one less class (*k-1* classes) in favor of the tested model (*k* classes). When this test is not significant for the *k* class model, the *k-1* model is superior. The results of these tests are presented (Table 2) and discussed in the model refinement process described below.

Latent class growth analysis (LCGA)

In latent class growth analysis, a mean growth curve is calculated for each class, and no variation around those growth curves is estimated (i.e. they are defined at the outset of model estimation to be zero) (Muthen & Muthen, 2000). That is, every individual is assigned to a single class. As all of the individual growth trajectories within a class are assumed to be homogeneous, membership in the class is stricter than if variance of class members' growth trajectory parameters were allowed to vary. Stricter membership results in easier computation, more clearly differentiated classes, and higher entropy. However, because of this strict class definition, individual trajectories are required to be very similar in order to be classified together, and thus modeling typically yields more classes than if the classes were allowed to be more heterogeneous.

The fit statistics for the 2- through 7-class LCGA solutions are found in Table 2. Of first consequence was whether more than one growth class was identified, determining whether different patterns of change in BMI over time existed at all in this population of children. The LMR LRT for a two-class solution had a *p*-value of < .000, which indicated that a one-class solution was rejected in favor of (at least) 2 classes. The entropy for the LCGA solutions was quite good, indicating that the classes as defined by LCGA were good predictors of class membership. Although the AIC and BIC continued to fall as the number of classes estimated increased, their incremental decrease became small, providing informal support for a solution of no more than 6 classes. I determined the 5-class model to be the best of the LCGA models based on the LMR LRT, which rejected 4 classes in favor of 5 ($p = 0.012$), but did not reject 5 in favor of 6 ($p = 0.132$). As can be seen in Figures 6 and 7, the LCGA 4-class and 5-class solutions were very similar, containing consistently (5-class % of sample, and 4-class % of sample, respectively): a low stable trajectory class (14.0% – 15.0%); an average stable class (20.5% – 21.5%), named for its mean (43rd percentile), which fell just below the 50th percentile of the CDC 2000 growth charts; a median stable class (18.7% – 26.4%), named for its mean (67th percentile), which fell at the median for the entire sample (66th percentile); and a high rising trajectory class (35.3% – 37.1%). For the trajectory classes in common across the 4-class and 5-class solutions, class means were similar (4-class solution not shown; 5-class solution shown in Table 2). However, the LCGA 4-class solution lacked the low-to-high class (11.6%) that emerged in the 5-class solution.

Table 2.

LCGA, GMM, and final class model selected for BMI percentile trajectories, age 24 months – 8th grade.

Latent Class Growth Analysis (LCGA)						
Classes	Parameters	LL	AIC	BIC	Entropy	LMR LRT
2	26	3297.5	-6543.0	-6412.2	.88	$p < 0.000$
3	31	3807.1	-7552.3	-7396.2	.85	$p = 0.010$
4	36	4082.3	-8092.6	-7911.5	.84	$p = 0.001$
5	**41**	**4225.3**	**-8368.7**	**-8162.4**	**.84**	$\mathbf{p = 0.012}$
6	46	4342.0	-8592.1	-8360.6	.84	$p = 0.132$
7	51	4424.8	-8747.5	-8490.9	.82	$p = 0.231$
Growth Mixture Modeling (GMM) – Without Regression of Factors onto Intercepts						
2	**36**	**4555.8**	**-9039.7**	**-8858.5**	**.77**	$\mathbf{p = 0.001}$
3	41	4622.1	-9162.1	-8955.8	.78	$p = 0.069$
Growth Mixture Modeling (GMM) – With Regression of Factors onto Intercepts						
2	39	4770.1	-9462.1	-9265.9	.73	$p < .000$
3	**47**	**4933.2**	**-9772.4**	**-9535.9**	**.77**	$\mathbf{p = .003}$
4	55	5003.8	-9897.6	-9620.8	.77	$p = .202$
5	63	5003.8	-9889.6	-9564.5	.80	$p = .303$
Final Model						
4	**55**	**4926.8**	**-9765.6**	**-9560.3**	**.74**	$\mathbf{p = .016}$

Note: Best solutions are in bold. Final model retains the low-to-high rising class estimated by LCGA and allows the other 3 classes to be freely estimated (GMM), including the regression of growth factors onto intercepts.

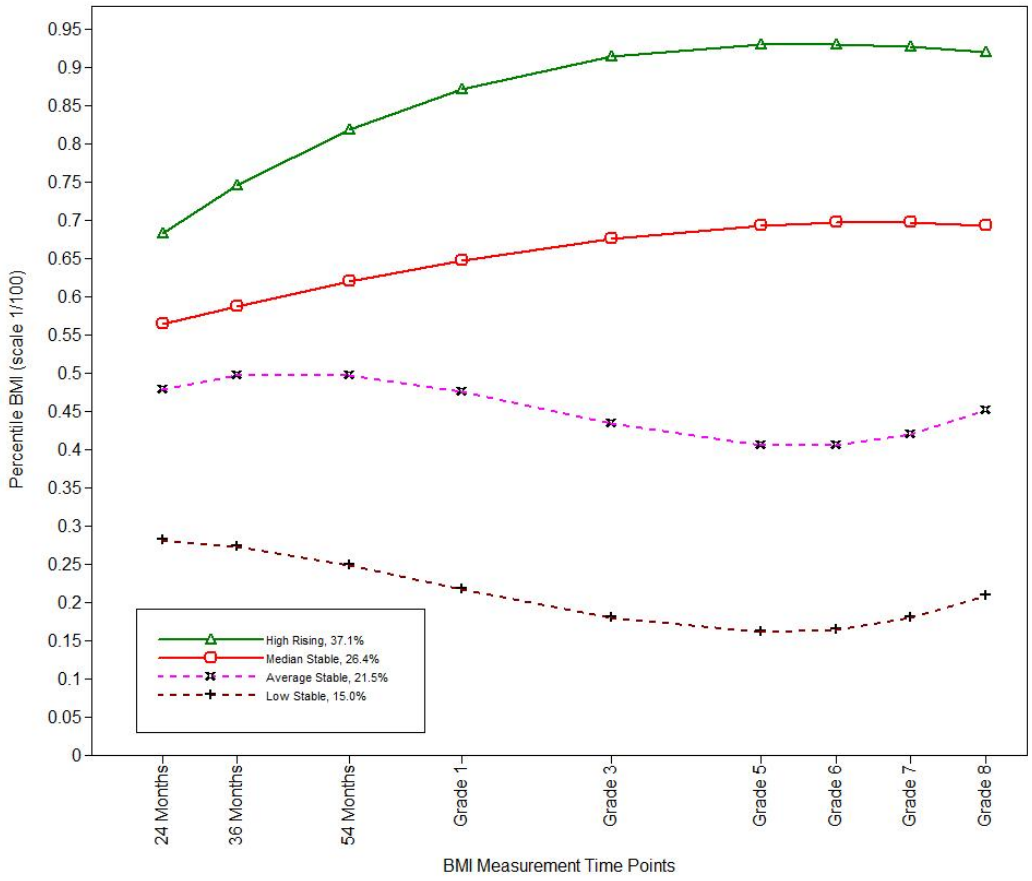

Figure 6. The LCGA 4-class solution.

The low-to-high rising class in the LCGA 5-class solution (Figure 7) was of particular interest, as it was the only class with such a rapid rise in BMI percentile. The cross-tabulation of class membership between the 4- and 5-class LCGA solutions (Table 3) revealed that all but one of the children in the new low-to-high class had been classified in either the median stable or high rising class in the 4-class solution. The LMR LRT showed the addition of the low-to-high class to be a statistically significant improvement over the solution without it, making that class key not only to the best-

fitting unconditional LCGA, but perhaps also to understanding rapid childhood weight gain in ~12% of the population.

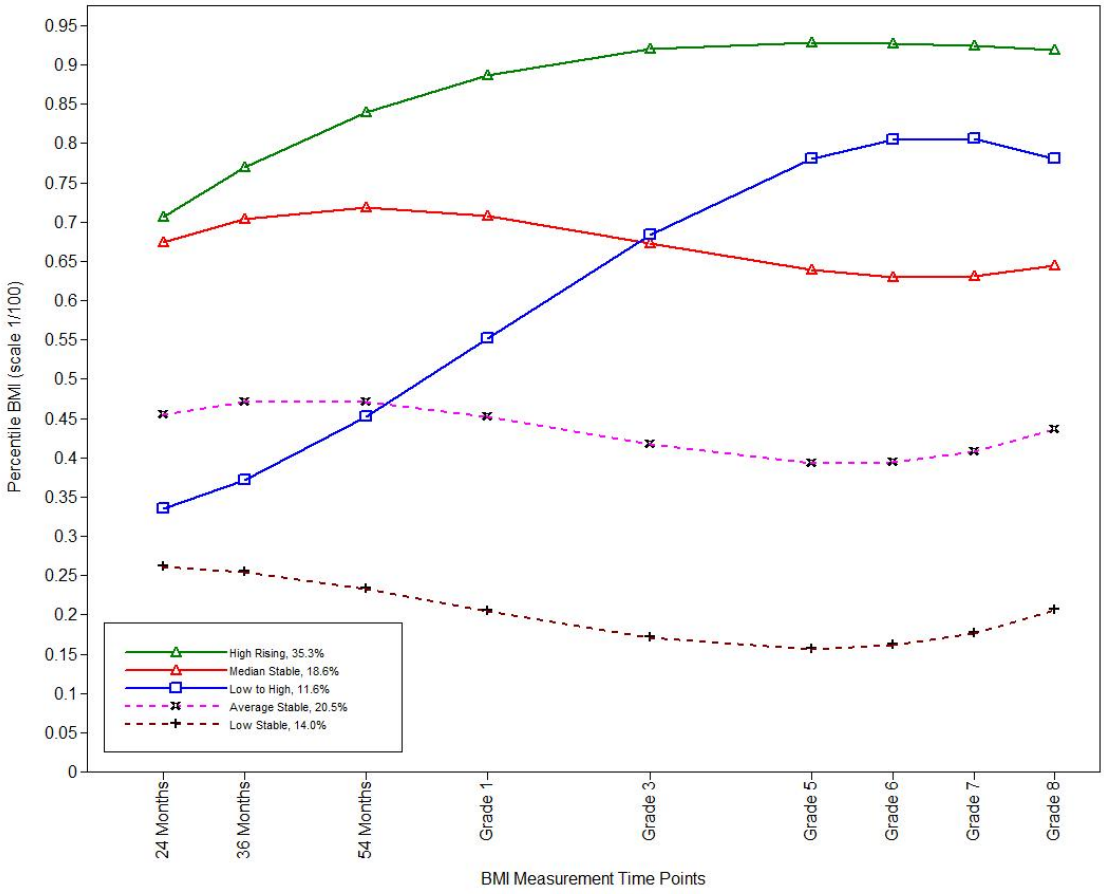

Figure 7. LCGA 5-class solution.

Visually examining Figure 7 also suggested that the average stable and low stable trajectories were of a consistently similar shape. The low stable trajectory appeared to decrease more rapidly between 24 and 54 months, but then the curves of the two trajectories mirrored each other. The average stable and low stable classes also had largely consistent membership between the 4- and 5- class models (Table 3), with 21 of 236 4-class average stable members moving to median stable in the 5-class solution, and

13 of 175 4-class low stable members moving to the low-to-high and average stable trajectories. The relatively flat trajectories, membership percentages, and below-median means of the average stable and low stable classes suggested that combining them should be explored in the final model refinement stage.

Table 3.

Cross-tabulation of individuals estimated in 4-classes and their distribution across the 5-class solution; low-to-high (LH) is the added class.

		5-class solution				
		HR	MS	LH	AS	LS
4-class solution	Totals					
HR	432	404	3	25		
MS	289	6	170	97	16	
AS	236		21		215	
LS	175			1	12	162
5-class Totals		410	194	123	243	162

Note: HR = High Rising, MS = Median Stable, LH = Low to High, AS = Average Stable, LS = Low Stable.

Next, however, the second, more generalized method of latent growth modeling, growth mixture modeling (GMM), was pursued, to explore whether allowing growth class variance to vary yielded different results. Although it would be more computationally intensive, the more relaxed class differentiation in growth mixture modeling was expected to explain more variance between trajectories and yield fewer classes.

Growth mixture modeling (GMM)

In GMM, class growth factors' variances are estimated, rather than constrained to zero, which adds complexity but also typically resolves to fewer and larger classes. As the variance of each growth factor – the intercept, linear, quadratic, and cubic terms – is

estimated rather than set to zero, groups of individual growth trajectories may vary around different means. This flexibility allows individuals' different trajectories to be classed together, as long as their factors' relative relationship is similar (Jung & Wickrama, 2008). The resulting growth classes are more heterogeneous, which typically lowers entropy, but allowing the model to estimate variance increases its accuracy, raising the log-likelihood.

In the initial GMM in this study, two growth classes were identified: an average-to-high rising and a low stable (Table 2, Growth Mixture Modeling (GMM) – Without Regression of Factors onto Intercepts). The LMR LRT did not reject the two-class for the three-class solution (LMR LRT $p = .069$). The two classes were so large, and varied so much around the trajectory means, that they did not differentiate sets of children well and masked categories of ecological interest (e.g. overweight children at the 85th percentile and higher, for example, or those with a more rapid increase). Individuals with a rising trajectory overall were grouped together in the large average-to-high rising class, which would not ultimately prove useful in risk assessment. In the case of a biological outcome such as BMI, trajectory growth may be closely related to the trajectory's starting point or the trajectory's mean, for example, and this relation should be called out.

One strategy for accomplishing greater differentiation by class intercept and change is further to parameterize the GMM model to allow these distinctions to emerge by class. In practical terms, the relation between the growth factors and intercept is added to class definition as part of the estimation process. Individual trajectories are then classed together not only by shape and variance but also by the relation between their

shape and their starting point. This relation is defined by adding a regression of the growth factors – slope, quadratic, and cubic terms – onto the class intercept (Muthén, 2009). In this study modeling BMI percentile change, trajectory intercepts were the trajectory means, as time points had been centered around the mean time point. By simultaneously solving for trajectories' growth factors and the influence of the trajectory's mean BMI percentile on those growth factors, I hoped that the remaining variance in change over time might emerge more clearly, in the form of additional differentiated classes.

The GMM models including the regressions of each class's growth factors onto its intercept fit better than those without them (Table 2, Growth Mixture Modeling (GMM) – With Regression of Factors onto Intercepts). Adding the slope, quadratic, and cubic terms regressed onto the intercept of a 3-class GMM increased the log likelihood by 312.6, a highly significant improvement ($\chi^2(626, 6)$, $p < .000$), and lowered the BIC from -8955.8 to -9685.2, compared to the 3-class model without the regressions. The 3-class GMM with the regressions was also clearly preferred to the equivalent a 2-class model (LMR LRT $p < .01$). Adding the regressions had thus yielded a significantly improved model that split one high rising class into two: a high rising and a median stable class. This differentiation produced more qualitatively meaningful trajectories for individuals with mean BMI percentiles > 50^{th} (Figure 8).

As the model fit was consistently superior with the growth factors regressed on the intercepts, all GMM modeling reported here included the class-specific linear, quadratic, and cubic growth factors regressed onto the class's intercept. The advantages

of GMM were substantial, as expected: the successful estimation of variance and the parsing out of the influence of the intercept on growth yielded a best model higher in log-likelihood (4933.2 vs. 4225.3) and lower in the BIC fit statistic (-9535.9 vs. -8162.4) than the best LCGA model. The classification quality of the best GMM model as measured by entropy, an index summarizing the overall precision of classes based on the posterior

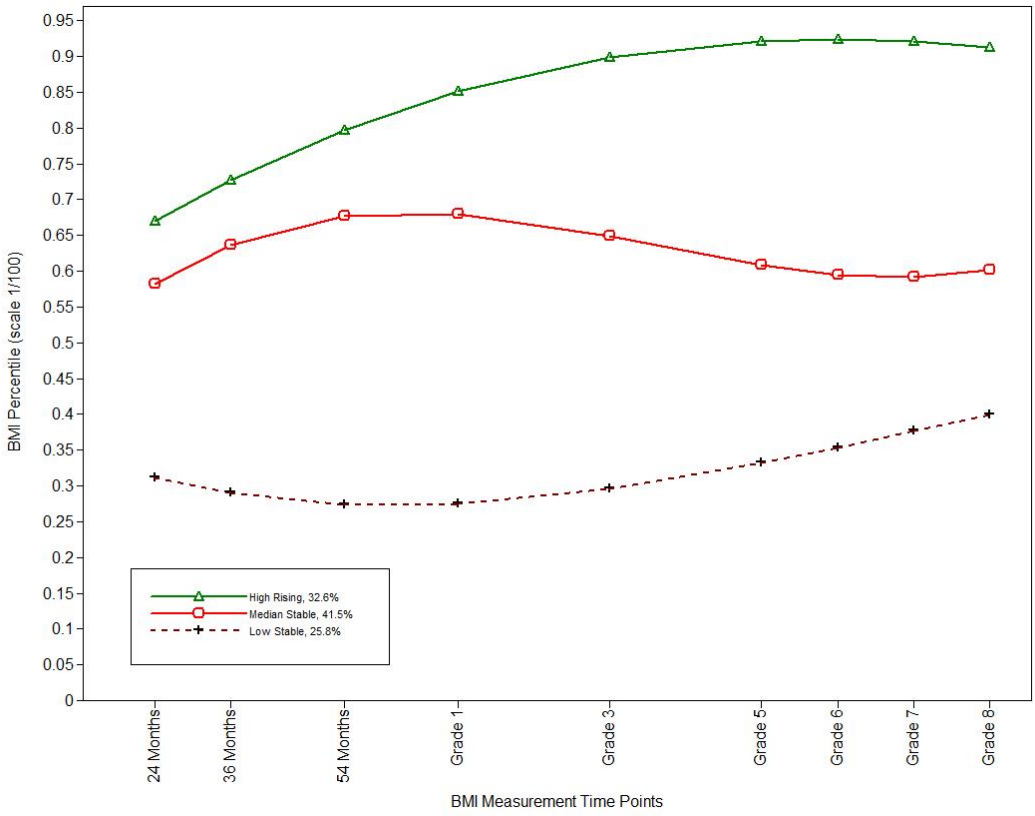

Figure 8. Best GMM solution, with each class's growth factors regressed onto class intercept (mean BMI percentile).

probabilities, was not as high as in the best-fitting LCGA (entropy .77 vs. .84); this drop was also expected, due to the greater heterogeneity in GMM classes. However, each of the three classes in the best GMM model exceeded the posterior probability cut-off of .70

suggested by Nagin (2005) as qualifying for good class distinction. The best GMM solution is reported in Table 4.

In spite of its broader class definition relative to LCGA, the final 3-class GMM also strongly supported the existence of different sub-groups, or classes, of BMI percentile trajectory. If the growth factors themselves were the only objects of interest, the 3-class GMM would be best, as the fit statistics were superior to the LCGA model across the board. But there were also ecological and clinical considerations. Notably, the low-to-high rising class in the best LCGA solution, a class that might point to a very specific risk profile and/or constellation of outcomes, was absent.

Table 4.

GMM 3-class solution: Growth factor coefficients, variance, and significance by class.

Growth factors by class	Estimate	(SE)	*p*-value
High Rising (32.6% of sample)			
intercept	0.893	0.008	0.000
linear	0.159	0.019	0.000
quadratic	-0.221	0.019	0.000
cubic	0.031	0.034	0.368
High Stable (41.5% of sample)			
intercept	0.655	0.015	0.000
linear	-0.166	0.032	0.000
quadratic	-0.123	0.032	0.000
cubic	0.398	0.079	0.000
Low Stable (25.8% of sample)			
intercept	0.292	0.021	0.000
linear	0.115	0.054	0.032
quadratic	0.145	0.053	0.006
cubic	-0.092	0.118	0.439

Note. Estimate = mean coefficient of the growth factor; SE = standard error; *p*-values are two-tailed. Linear, quadratic, and cubic growth factors are regressed onto class intercept.

Final growth class model

The best distillation of growth classes would retain the low-to-high distinction of the LCGA 5-class solution and the computational rigor of GMM's variance estimation to approximate an ideal of meaning and fit. Thus I defined the low-to-high class, using the growth factor values generated by the best LCGA model, at the outset of the final model estimation. I then allowed GMM estimation of the remaining 3 classes. This last BMI percentile growth class model (Table 2, Final Model) was both more parsimonious and better fitting than the best LCGA model, yet retained the ecologically meaningful low-to-high rising class, which GMM alone had eliminated. The final model was superior in fit to the LCGA 5-class model, by the log likelihood and the BIC fit statistic (LL 4926.8 vs. 4225.3 and BIC -9560.3 vs. -8162.4, respectively), and comparable to the GMM 3-class model (LL 4926.8 vs. 4933.2 and BIC -9560.3 vs. -9535.9, albeit with the use of 8 additional parameters). Entropy of the final model remained respectable, at .74.

Table 5 records the parameters of the final solution; Figure 9 graphs it. Individuals were classified into their most-likely trajectory class based on the posterior probability of their membership in these classes. The four classes were very different: fully 63% of the children in the high rising class had been classifiable as obese at least once during their childhood, vs. 21% in the median stable class; 69% of the median stable class had been overweight, but only 6% of the low stable class. With individuals assigned to their growth classes, characterizing inter-individual differences distinguishing these classes could begin.

Table 5.

Final BMI trajectory class solution: Growth factor coefficients, variance, significance, posterior probability, and overall weight statistics by class.

Trajectory characteristics				PP	Mean BMI %-ile	% Overweight, one or more time point	% Obese, one or more time point
Growth factors by class	Est.	(SE)	*p*-value				
High Rising (30.7% of sample)				.88	86th	91%	63%
intercept	0.90	(0.01)	0.000				
linear	0.14	(0.02)	0.000				
quadratic	-0.21	(0.02)	0.000				
cubic	0.05	(0.04)	0.178				
Median Stable (29.0% of sample)				.82	68th	69%	21%
intercept	0.71	(0.02)	0.000				
linear	-0.13	(0.06)	0.025				
quadratic	-0.14	(0.03)	0.000				
cubic	0.28	(0.09)	0.003				
Low to High[1](11.8% of sample)				.81	55th	44%	14%
intercept	0.67	(0.03)	0.000				
linear	0.52	(0.04)	0.000				
quadratic	-0.25	(0.06)	0.000				
cubic	-0.45	(0.09)	0.000				
Low Stable (28.4% of sample)				.88	32nd	6%	1%
intercept	0.29	(0.02)	0.000				
linear	-0.20	(0.05)	0.000				
quadratic	0.17	(0.03)	0.000				
cubic	0.49	(0.08)	0.000				

[1]Growth factor estimates defined from the final LCGA 5-class model.

Note. Est. = estimated mean coefficient of the growth factor; SE = standard error; *p*-values are two-tailed. PP = average posterior probability for the trajectory; % Overweight = BMI percentile >= 85th; % Obese = BMI percentile >= 95th. Linear, quadratic, and cubic growth factors are regressed onto class intercept for all but the low-to-high rising class.

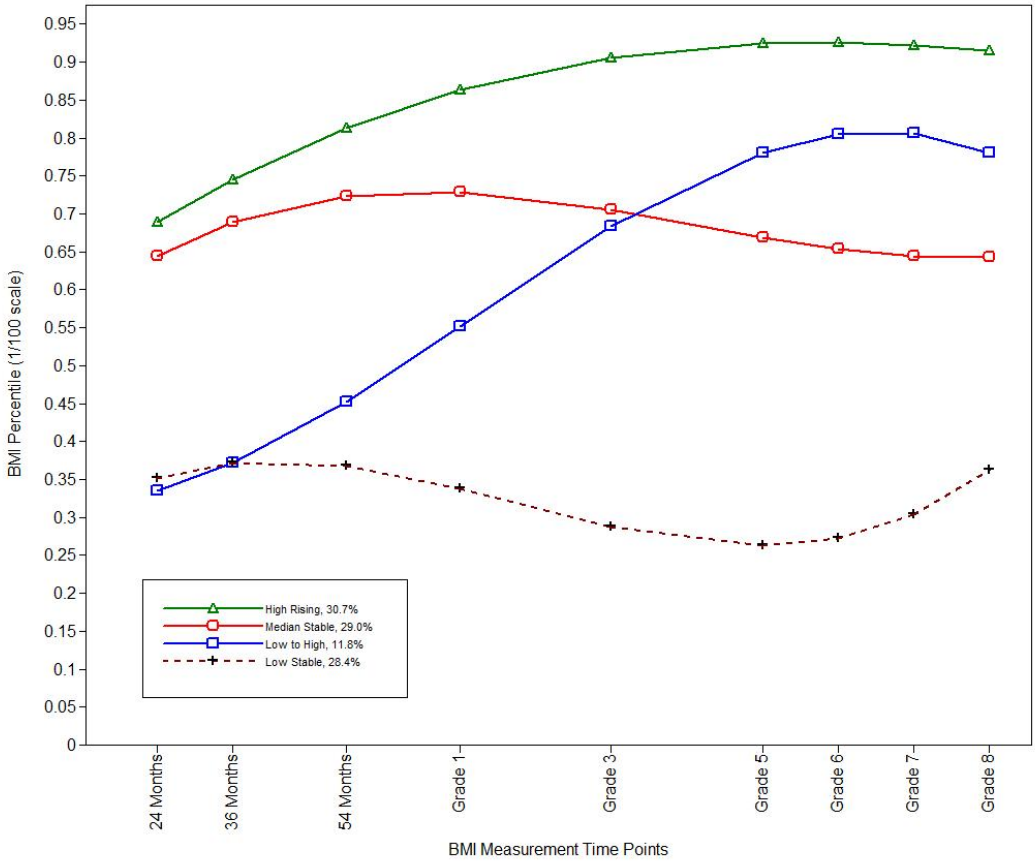

Figure 9. Final BMI trajectory growth class solution: Low-to-high class defined from best LCGA model; remaining 3 classes freely estimated with growth factors regressed onto intercept (GMM).

RQ 2A: CHARACTERIZING TRAJECTORY MEMBERSHIP: CHILDHOOD RISK FACTORS

My review of the literature reporting links between various biological and social factors associated with childhood weight gain had generated a robust list of potential predictors for membership in these BMI trajectories, many of which, with a few notable exceptions, were either present in the SECCYD dataset or could be approximated from what was measured in that study. These could now be assessed for the degree to which they were associated with each class. Descriptive statistics of the variables tested for associations with BMI trajectories are in Table 6.

Table 6.

Mean (standard deviation) and range of early continuous childhood predictors in the sample population (N = 1,132).

	Mean (SD) or Percentage	Range
Continuous variables		
Birth weight for age (kg)	3.52 (0.45)	2.01 – 5.57
Ratio weight increase, 0-15 mo.	2.10 (0.42)	1.05 – 4.57
Child BMI, 15 mo.	17.31 (1.42)	19.00 – 30.61
M average sensitivity, 6-24 mo.	9.31 (1.32)	4.55 – 12.00
M sum of stresses, 0-15 mo.	2.16 (1.75)	0 – 10
M BMI, Stunkard Scale, 15 mo.	4.37 (1.54)	1 – 9
M BMI, Stunkard Scale, 24 mo.	4.44 (1.55)	1 – 9
M prenatal smoking (1-5 scale)	1.62 (1.28)	1 – 5
Negative life events, 54 mo.	3.21 (3.36)	0 – 27
M health, average 0-24 mo.	3.27 (0.54)	1.25 - 4
M depression, 01 mo.	11.12 (8.76)	0 – 53
M depression, 06 mo.	9.00 (8.28)	0 – 52
M depression, 15 mo.	9.05 (8.08)	0 – 51
M depression, 24 mo.	9.44 (8.50)	0 – 51
Breastfed: Yes	81%	
SS: Avoidant	15%	
SS: Resistant	14%	
SS: Secure	71%	

Note: BMI = body mass index; M = maternal; SS = Strange Situation Protocol.

Comparing mean differences on early childhood variables risk factors across the classes indicated whether BMI growth trajectories could be differentiated by as early as the period from birth to 24 months. One-way analyses of variance (ANOVA) and Tukey's Honest Significant Differences test identified mean differences across the classesin continuous variables (Table 7). Binomial predictors were tested for associations

with class membership in logistic regressions using the low stable class as the baseline contrast (Table 8).

The low stable class differed significantly from the high rising in almost every continuous risk variable. The strongest associations were in the biological measures, e.g. birth weight for age, percent increase in weight from 0-15 months, and mother's BMI at 15 months. Mothers of children in the low stable class were also significantly higher in sensitivity, the only continuous relational variable tested. The socio-economic status-related variables, average income-to-needs ratio from 0-24 months and maternal education at 1 month, were higher in the low stable and median stable classes than in the high rising class. Lower maternal depression at 6, 15, and 24 months was associated with membership in the low stable vs. high rising class, but the opposite relation was present at 1 month. Among the categorical variables, girls were less likely to be in the high-rising class than the low-stable class, and identifying as Black was related to greater probability of membership in all classes but the low stable class. Although many of these coefficients were highly significant, their individual effect sizes (μ^2 for the one-way ANOVAs and coefficient of discrimination, *D* for the logistic regressions) were small.

Table 7. Mean differences (ANOVA tests) in early continuous childhood predictors across growth trajectories.

	Low Stable	Low-to-High	Median Stable	High Rising	*F*	*p*-value	μ^2	Significant Differences
Continuous variables	Mean (SE)	Mean (SE)	Mean (SE)	Mean (SE)				
Birth weight for age (kg)	3.40 (0.43)	3.46 (0.41)	3.58 (0.46)	3.60 (0.47)	14.01	5.72E-09***	0.04	LS < MS, HR LH < MS†, HR
Ratio weight increase, 0-15 mo.	2.03 (0.39)	2.02 (0.40)	2.12 (0.41)	2.18 (0.46)	9.55	3.13E-06***	0.02	LS < MS, HR LH < HR
Child BMI, 15 mo.	16.61 (1.20)	16.66 (1.03)	17.62 (1.27)	17.86 (1.48)	69.46	< 2E-16***	0.16	LS, LH < MS, HR MS < HR†
M average sensitivity, 6-24 mo.	9.45 (1.20)	9.29 (1.22)	9.43 (1.40)	9.10 (1.35)	5.48	0.001***	0.01	LS, MS > HR
M sum of stresses, 0-15 mo.	2.08 (1.71)	1.94 (1.49)	2.19 (1.79)	2.27 (1.83)	1.39	0.246	<0.01	
M BMI, 15 mo.	3.93 (1.28)	4.54 (1.48)	4.20 (1.48)	4.87 (1.67)	24.91	1.30E-15***	0.06	LS < LH, HR MS < HR
M BMI, 24 mo.	4.05 (1.41)	4.43 (1.60)	4.33 (1.44)	4.90 (1.63)	19.19	3.86E-12***	0.05	LS, LH, MS < HR LS < LH†, MS†
M prenatal smoking (1-5 scale)	1.53 (1.19)	1.66 (1.32)	1.50 (1.18)	1.78 (1.42)	3.43	0.0165*	0.01	LS, MS < HR
Income-to-need ratio, average 0-24 mo.	3.55 (2.47)	3.30 (2.95)	3.81 (2.79)	3.03 (2.53)	5.45	1.00E-03**	0.01	LS, MS > HR
Negative life events, 54 mo.	2.94 (2.95)	2.90 (3.06)	3.24 (3.28)	3.53 (3.83)	2.20	0.086†	0.01	LS < HR†
M education, yrs.	14.60 (2.57)	14.18 (2.45)	14.58 (2.48)	13.95 (2.43)	5.41	0.001**	0.01	LS, MS > HR
M health, average 0-24 mo.	2.64 (13.1)	3.26 (0.60)	3.27 (0.51)	2.54 (12.6)	0.41	0.75	<0.01	
M depression, 01 mo.	9.93 (7.65)	10.12 (8.25)	11.78 (8.90)	12.06 (9.58)	4.56	0.004**	0.01	LS < HR LS < MS
M depression, 06 mo.	8.07 (7.46)	7.69 (7.67)	9.50 (8.58)	9.73 (8.79)	3.69	0.012*	0.01	LS < HR
M depression, 15 mo.	8.23 (7.29)	8.00 (7.67)	9.21 (8.30)	9.98 (8.60)	3.47	0.016*	0.01	LS < HR
M depression, 24 mo.	8.08 (7.44)	9.08 (8.57)	9.89 (8.90)	10.40 (8.90)	4.85	0.002**	0.01	LS < MS, HR

† $p < .10$ *$p < .05$ **$p < .01$ ***$p < .001$; two-tailed.

Note. M = maternal. LS = low stable; LH = low to high; MS = median stable; HR = high rising. Significant group differences identified by Tukey's HSD test.

Table 8.

Percentages of binomial variables and odds ratios (OR) relating them to the three upper trajectories vs. the low stable (LS) trajectory. Effect sizes calculated as the coefficient of discrimination, *D*.

	LS	Low-to-High			Median Stable				High Rising				R^2
	%	%	OR	(CI)	*z*	%	OR	(CI)	*z*	%	OR	(CI)	*z*	
Categorical variables														
Gender: Female	55	44	0.67	(0.43 – 1.03)	-1.83 †	48	0.77	(0.56 – 1.04)	-1.70 †	46	0.72	(0.54 – 0.97)	-2.12 *	0.01
Race: Black	7	14	2.01	(1.02 – 3.98)	2.01 *	12	1.73	(1.02 – 2.92)	2.03 *	16	2.35	(1.43 – 3.85)	3.39 ***	0.01
Hispanic: Yes	7	4	0.55		-1.08	7	1.11		0.32	5	0.74		-0.92	< 0.01
Breastfed: Yes	82	79	0.83		-0.67	83	1.14		0.64	80	0.87		-0.70	< 0.01
SS: Avoidant	14	22	1.81	(1.04 – 3.13)	2.11 *	14	1.06		0.25	16	1.81		0.72	< 0.01
SS: Resistant	15	8	0.51	(0.24 – 1.07)	-1.78 †	13	0.87		-0.64	14	0.93		-0.35	< 0.01
SS: Secure	71	70	0.92		-0.35	72	1.05		0.30	70	0.95		-0.29	< 0.01

† $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$; two-tailed.

Note. CI = confidence interval. CI provided when association with trajectory is significant at <= .10. Breastfed = ever breastfed, without regard for duration. SS = Strange Situation protocol classification. R^2 = Tjur's R^2 for logistic regressions (Tjur, 2009).

RQ 2B: Moderation by Infant Strange Situation Classification

This study also explored specific hypotheses with respect to the quality of the mother-child relationship as measured by the Infant Strange Situation Protocol (SSP). Recent research has suggested that stress management patterns displayed by infants during the SSP might persist through the life span. It has been speculated that individual differences in stress management strategy accounts for the ambiguous results to date attempting to link stress and weight gain; i.e. some strategies may lead to weight gain under stress and some to weight loss (Bichteler & Jacobvitz, under review).

Considering the high-rising weight trajectory to be the least healthy (as over 63% of the individuals classified in it exceeded the 95th percentile cut-off for obesity at least once during childhood), membership in this class was modeled in logistic regression using all of the variables found to vary at least marginally across the weight trajectories (Tables 7 and 8), with the addition of an interaction term between maternal sum of stresses from 0-15 months and the dyad's 3-way attachment classification coded at 15 months. Although neither of these variables had been associated independently with the high-rising trajectory, it had been hypothesized that their effects might only be observed in the context of each other, as had been found in adults (Bichteler & Jacobvitz, under review). The full model was submitted to backward stepwise reduction based on the AIC, which, for every iteration, tests the deletion of each variable against an improvement (an absolute decrease) in the AIC, then deletes the variable if the resulting model is superior. The AIC criterion often yields models including non-significant

independent variables; only those with at least a marginal (at $p <= .10$) were retained.

Results of the full and best-fitting reduced models are in Table 9.

Table 9.

Membership in the high-rising weight trajectory predicted by all risk factors (Full Model) and by the best-fitting set of variables with at least marginal significance (Reduced Model).

	Full Model			Reduced Model		
Early childhood risk factors	OR	(CI)	*p*	OR	(CI)	*p*
Birth weight for age (g)	1.00	(1.00 – 1.00)	***	1.001	(1.001 – 1.002)	***
Percent weight increase, 0-15 mo.	3.96	(2.12 – 7.37)	***	3.97	(2.13 – 7.38)	***
Child BMI, 15 mo.	1.26	(1.09 – 1.46)	**	1.26	(1.09 – 1.46)	**
Maternal BMI, 15 mo.	1.22	(1.06 – 1.39)	**	1.22	(1.07 – 1.40)	**
Maternal BMI, 24 mo.	1.15	(1.01 – 1.31)	*	1.15	(1.01 – 1.31)	*
Maternal prenatal smoking	1.15	(1.03 – 1.29)	*	1.17	(1.05 – 1.30)	**
Gender: Female	1.33	(0.99 – 1.77)	†	1.31	(0.98 – 1.75)	†
Race: Black	1.36	(0.85 – 2.18)		1.48	(0.95 – 2.30)	†
Maternal sum of stresses, 0-15 mo.	1.04	(0.94 – 1.14)		1.05	(0.96 – 1.15)	
Strange Situation: Avoidant	0.99	(0.51 – 1.89)		1.009	(0.53 – 1.92)	
Strange Situation: Resistant	2.10	(1.10 – 4.03)	*	2.13	(1.11 – 4.07)	*
Sum of stresses x Avoidant	0.98	(0.78 – 1.23)		0.98	(0.78 – 1.22)	
Sum of stresses x Resistant	0.77	(0.59 – 1.00)	†	0.76	(0.59 – 0.99)	*
Income-to-need ratio, average 0-24 mo.	0.99	(0.93 – 1.06)				
Maternal average sensitivity	0.96	(0.84 – 1.10)				
Maternal education, yrs.	0.98	(0.91 – 1.05)				
Maternal depression, 1 mo.	1.00	(0.98 – 1.02)				
Maternal depression, 6 mo.	1.00	(0.98 – 1.02)				
Maternal depression, 15 mo.	1.00	(0.98 – 1.02)				
Maternal depression, 24 mo.	1.00	(0.98 – 1.02)				
Model fit						
Tjur's R^2		0.169			0.167	
Residual deviance (df)		1226.1 (1102)			1227 (1109)	
AIC		1286.1			1273.3	

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. Secure is the baseline Strange Situation contrast. Study site was controlled for in both models, not shown.

The last seven variables listed in Table 9, although they were significantly higher in the high-rising trajectory than in the low stable trajectory (Table 7), were not significant predictors in logistic regression modeling, nor were they retained in the reduced model minimizing the AIC. The stress-by-attachment classification interaction, however, was marginally significant in the full model, and significant at $p < .05$ in the reduced model, particularly with respect to the contrast between secure and anxious-resistant dyads. Children with anxious-resistant attachment patterns at 15 months were more likely to be in the high-rising trajectory when under less stress and less likely under greater stress (Figure 10).

RQ 3: Predicting Symptoms of Metabolic Syndrome at 15 ½ Years

Finally, the utility of the four weight change trajectory classes in predicting adolescent weight and blood pressure was tested. Although different weight change trajectories had clearly emerged, it was unclear whether they would add explanatory power to health outcomes at 15 ½ years. Multivariate regression models including all demographic, biological and psychosocial risk factors, but without trajectory class membership, were tested, to determine which set of early childhood risk factors best predicted each health outcome of interest. These models are reported in the Appendix. I then tested the full set of risk factors plus trajectory class membership to determine the best-fitting model for each health outcome that included individuals' BMI trajectory. To quantify any increase in variance explained by modeling with trajectory membership, these two models were compared, for each of the five health outcomes at 15 ½ years: percentile BMI, waist circumference, overweight (>= 85th percentile = 1), obesity (>=

Strange Situation Classifications: A = Avoidant; B = Secure; C = Anxious/Resistant

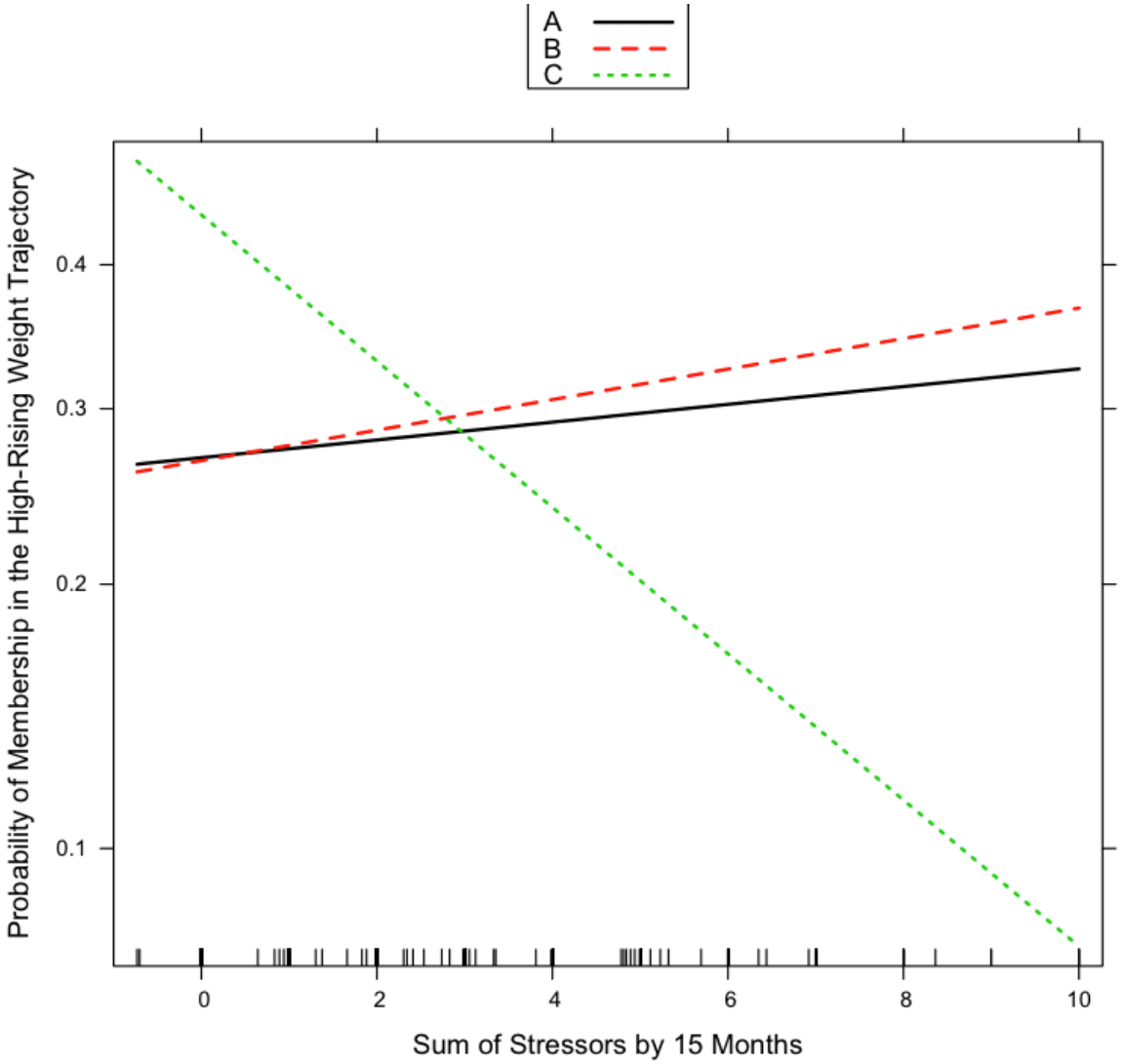

Figure 10. Strange Situation classification moderates the effect of stress on membership in the high-rising weight trajectory.

95th percentile = 1), and blood pressure (modeled as high = 1). The sample means/percentages for these variables are in Table 10. The analyses explored whether the rate and trajectory of change over the course of childhood predicted health outcomes at age 15 ½ over and above the early childhood risk factors identified.

All early childhood risk factors were entered into linear or logistic regressions, as appropriate. Two interaction terms were also included a priori: birth weight for age by percent increase in weight from birth-15 months (testing rapid weight gain after birth), and the sum of stresses from 0-15 months with strange situation classification at 15 months (RQ 2b). The subsequent models were subjected to stepwise backwards

Table 10.

Mean (standard deviation) and range of health outcomes at 15 ½ years (N = 1132).

	Mean or Percentage	(SD)	Range
Percentile BMI	0.66	(0.27)	0.00 – 1.00
Waist circumference (cm)	75.78	(12.23)	25 – 133.35
Overweight (>= 85th percentile)	31.1%		
Obese (>= 95th percentile)	15.6%		
High blood pressure (=1)	28.7%		

elimination based on the AIC: each variable was tested iteratively for whether its inclusion benefited model fit; if not, it was eliminated, but retested at every iteration. This stepwise AIC-based reduction results in generous variable inclusion; therefore, the final model refinement was performed manually, testing for at least marginal significance. During this phase, a fourth interaction was found to emerge in several models: Hispanic origin by maternal education. This interaction was likewise retained where it contributed at least marginally significantly to outcome variance.

Regression analyses showed that earliest childhood risk factors, all measured at 24 months of age or younger, significantly predicted the weight and blood pressure

symptoms under study. Further, in every case, the trajectories of change from 24 months to Grade 8 predicted the outcomes above and beyond the simple effects, sometimes doubling or more the explained variance (Tables 11 – 15). Graphs of any significant interactions follow the table (Figure 11). Table 16 summarizes the contrasts between classes in predicting the health outcomes. Class trajectory membership was the strongest single predictor of weight outcomes at 15 ½ years, indicating that the path children are on as early as 54 months has a direct influence on their adolescent, and research would suggest adult, health.

The linear regression analyses of continuous outcomes percentile BMI and waist circumference at 15 ½ years yielded models with more significant predictors and greater variance explained than did the logistic regressions of the binary outcomes; this was to be expected, as substantial information is lost when continuous variables such as percentile are dichotomized (into overweight/not overweight, for example). Birth weight for age, percent weight increase from 0-15 months, and average maternal sensitivity from 6-24 months (inversely related) were all significant predictors of greater BMI percentile and waist circumference at 15 ½ years. These variables were not significant in the logistic regressions, however. Maternal BMI, usually at 15 months, predicted all but blood pressure. Greater maternal education was negatively associated with both overweight and obesity. The profile of related early childhood risk factors varies for each outcome; most notable perhaps are some of the null findings in the presence of trajectory membership: race, Hispanic origin, child BMI at 15 mo., maternal stresses, maternal physical health, maternal depression, and breastfeeding.

Table 11.

Best linear regression model predicting percentile BMI at 15 ½ years from early childhood risk factors and BMI change trajectory across childhood.

	B (SE)	*b*	t-value	*p*
Intercept	0.75 (0.21)		3.577	***
High rising trajectory vs. low stable	0.43 (0.02)	1.65	22.553	***
Median stable trajectory vs. low stable	0.22 (0.02)	0.45	11.751	***
Low-to-high trajectory vs. low stable	0.25 (0.02)	1.47	10.38	***
Gender: female	0.03 (0.01)	0.05	1.831	†
Birth weight for age	-0.00 (0.00)	-0.00	-1.41	
SS: Avoidant vs. secure	-0.01 (0.02)	-0.07	-0.781	
SS: Anxious-resistant vs. secure	-0.04 (0.02)	-0.43	-2.025	*
Maternal BMI 15 mo.	0.01 (0.00)	0.05	2.876	**
Percent weight increase, 0-15 mo.	-0.16 (0.09)	-0.59	-1.767	†
Maternal average sensitivity, 6-24 mo.	-0.02 (0.01)	-0.03	-2.753	**
Income-to-need ratio average, 0-24 mo.	-0.01 (0.00)	-0.04	-2.317	*
Birth weight for age x percent weight increase	0.00 (0.00)	0.00	2.048	*
Model fit with trajectory membership				
R^2	0.50			
AIC	-373.7			
Model fit without trajectory membership (Table A1, Appendix)				
R^2	0.19			
AIC	12.06			

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. SS = strange situation protocol. Variables with at least marginal significance were retained. Best-fitting model without trajectory membership is in Table A1, Appendix.

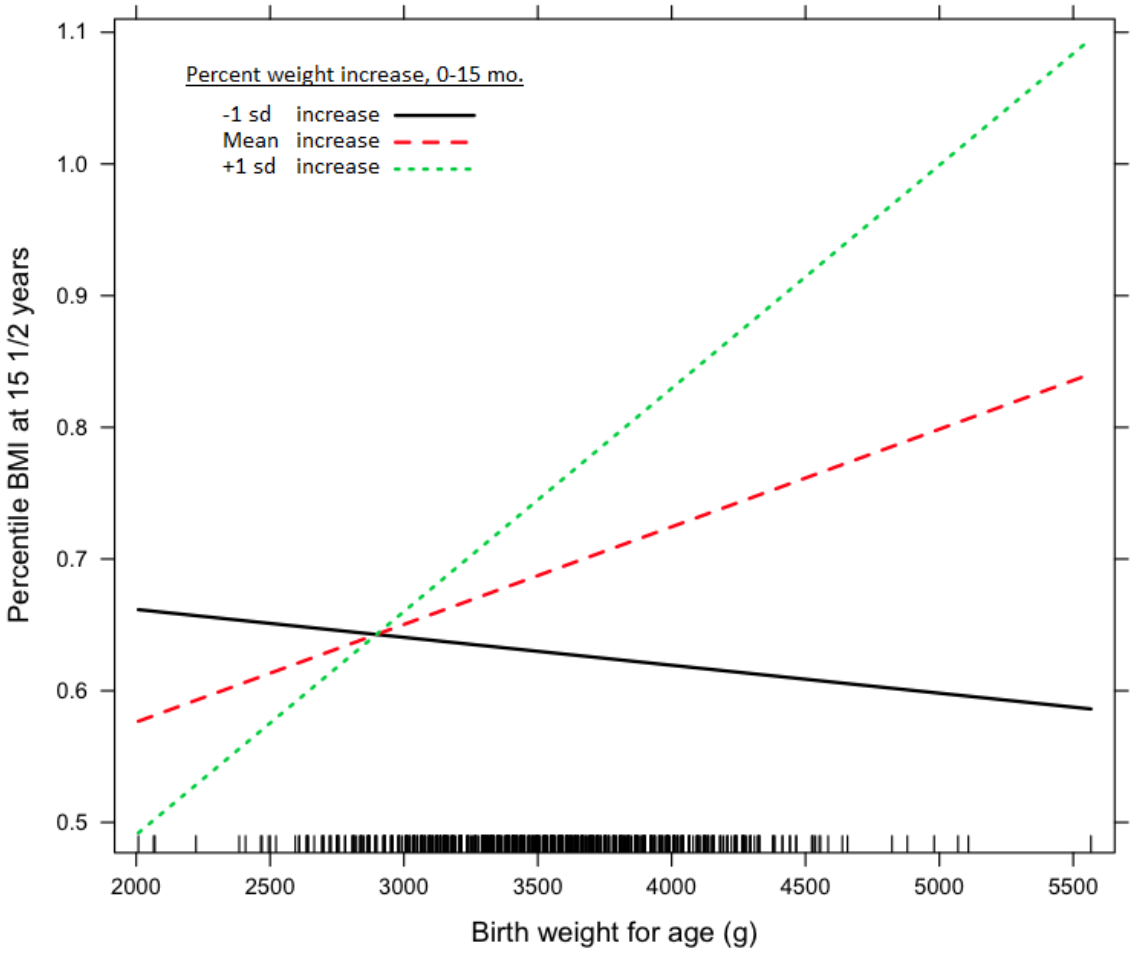

Figure 11. Percent increase in weight from 0-15 months moderates the influence of birth weight for age on percentile BMI at 15 ½ years.

Table 12.

Best linear regression model predicting waist circumference at 15 ½ years from early childhood risk factors and BMI change trajectory across childhood.

	B (SE)	*b*	t-value	*p*
Intercept	56.032 (6.47)		8.66	***
High rising trajectory vs. low stable	15.216 (0.95)	1.25	15.98	***
Median stable trajectory vs. low stable	2.972 (0.91)	0.12	3.27	**
Low-to-high trajectory vs. low stable	4.877 (1.17)	182.88	4.18	***
Gender: Female	-4.204 (0.69)	-0.54	-6.08	***
Birth weight for age	0.003 (0.00)	0.00	2.88	**
Maternal BMI 15 mo.	1.114 (0.23)	0.01	4.91	***
Percent weight increase, 0-15 mo.	3.600 (1.18)	0.09	3.06	**
Maternal average sensitivity, 6-24 mo.	-0.764 (0.28)	-0.06	-2.71	**
Model fit with trajectory membership				
R^2		0.44		
AIC		5746.7		
Model fit without trajectory membership (Table A2, Appendix)				
R^2		0.25		
AIC		5988.5		

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. Variables with at least marginal significance were retained. Best-fitting model without trajectory membership is in Table A2, Appendix.

Table 13.

Best logistic regression model predicting overweight (>= 85th percentile BMI) at 15 ½ years from early childhood risk factors and BMI change trajectory.

	OR	(CI)	*p*
High rising trajectory vs. low stable	62.25	(28.99 – 133.70)	***
Median stable trajectory vs. low stable	4.88	(2.19 – 10.88)	***
Low-to-high trajectory vs. low stable	9.17	(3.85 – 21.86)	***
Maternal BMI 24 mo.	1.24	(1.09 – 1.41)	***
Income-to-need ratio average, 0-24 mo.	0.90	(0.82 – 1.00)	*
Maternal education, yrs.	0.92	(0.83 – 1.01)	†
Model fit with trajectory membership			
Tjur's R^2		0.42	
Residual deviance (df)		632 (798)	
AIC		648.3	
Model fit without trajectory membership (Table A3, Appendix)			
Tjur's R^2		0.18	
Residual deviance (df)		850 (789)	
AIC		884.0	

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. Variables with at least marginal significance were retained. Female gender was significantly related (OR = 0.62) and retained, not shown. Best-fitting model without trajectory membership is in Table A3, Appendix.

Table 14.

Best logistic regression model predicting obesity (>= 95th percentile BMI) at 15 ½ years from early childhood risk factors and BMI change trajectory.

	OR	(CI)	*p*
Median stable trajectory vs. high rising	0.08	(0.04 – 0.17)	***
Low-to-high trajectory vs. high rising	0.22	(0.10 – 0.45)	***
Low stable vs. high rising[1]	-	-	-
Maternal BMI, 15 mo.	1.48	(1.28 – 1.71)	***
Maternal education, yrs.	0.88	(0.79 – 0.97)	*
Model fit with trajectory membership			
Tjur's R^2		0.31	
Residual deviance (df)		451 (800)	
AIC		463.22	
Model fit without trajectory membership (Table A4, Appendix)			
Tjur's R^2		0.19	
Residual deviance (df)		561 (790)	
AIC		593.3	

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

[1] No obese children were classified in the low stable trajectory.

Note. Variables with at least marginal significance were retained. Best-fitting model without trajectory membership is in Table A4, Appendix.

Table 15.

Best logistic regression model predicting high blood pressure at 15 ½ years from early childhood risk factors (0-24 months) and BMI change trajectory across childhood.

	OR	(CI)	*p*
High rising trajectory vs. low stable	4.49	(2.82 – 7.14)	***
Median stable trajectory vs. low stable	1.73	(1.05 – 2.84)	*
Low-to-high trajectory vs. low stable	1.28	(0.64 – 2.54)	
Maternal prenatal smoking	1.13	(0.99 – 1.29)	†
Model fit with trajectory membership			
Tjur's R^2		0.18	
Residual deviance (df)		792.8 (802)	
AIC		822.8	
Model fit without trajectory membership (Table A5, Appendix)			
Tjur's R^2		0.15	
Residual deviance (df)		820.2 (797)	
AIC		860.2	

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. The low stable trajectory is the baseline trajectory contrast. Variables with at least marginal significance were retained. Female gender was significantly related (OR = 0.25) and retained, not shown. Of the 45 contrasts between sites, one study site was significantly different from four others, ($p < .05$); therefore site was retained, but is not shown. Best-fitting model without trajectory membership is in Table A5, Appendix.

Table 16.

Significance of all class contrasts by health outcome at 15 ½ years.

Trajectory contrasts	Percentile BMI	Waist Circumference	Over-weight	Obese	High blood pressure
High rising vs. median stable	***	***	***	***	***
High rising vs. low-to-high	***	***	***	***	***
High rising vs. low stable	***	***	***	***	***
Median stable vs. low-to-high	n.s.	n.s.	- *	- *	n.s.
Median stable vs. low stable	***	**	***	***	*
Low-to-high vs. low stable	***	***	***	n.s.	n.s.

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.
Note. All significant contrasts have positive coefficients except for the median stable vs. low-to-high contrast testing overweight and obesity. Low stable had the lowest BMI across time.

These findings, along with the robust interaction between birth weight for age and percent increase from 0-15 months predicting percentile BMI at 15 ½ years, are discussed in greater detail, below. The nature and rate of change in BMI over time, as reflected in trajectory class membership, and maternal BMI were the most consistent and strongest predictors of fat-related and blood pressure outcomes at 15 ½ years. Modeling so many demographic, biological, and psychosocial factors together offered a clear advantage in determining relative risk. Taken together, these results point to strong predictive power of risk factors present between 0-24 months of life, with important clinical implications.

Discussion

This study capitalized on data from birth to 15 ½ years in the NICHD SECCYD longitudinal dataset to explore children's weight change over time. Key biological and social variables were available across 12 time points, creating an opportunity to observe different trajectories of weight change, compare them, and build prediction models for health outcomes at age 15 ½. Long-standing questions regarding the heterogeneity of children's patterns of change, the relative influence of factors such as birth weight, breast-feeding, and socioeconomic status, and whether these patterns or factors pose any risk to adolescent weight and cardiac health could be addressed. A total of 1,132 children from the SECCYD sample, which reflected the national distribution on maternal employment, marriage status, and racial identification, were included in these analyses. This large sample size allowed a detailed exploration of three research questions: 1) Were there different trajectories of weight gain across childhood? 2) Did these trajectories differ in composition on biological and social risk factors? 3) To what degree did membership in different classes of change trajectory predict adolescent weight and blood pressure?

RQ 1: Unconditional Models of Growth Trajectories

The SECCYD dataset alone indicated a major shift in U.S. children's overall weight since the 1970's, when the bulk of the children were measured for the CDC's growth charts. This study, like others using more recent data (e.g. Ogden & Carroll, 2010), identified population-level shifts upward in weight. The 50th percentile of the SECCYD BMI percentiles was the 69th percentile of the CDC population; that is, 50% of

the SECCYD sample was at or exceeded the 69th percentile in BMI at 8th grade – a 19% increase across the board. Seventeen percent of the SECCYD population exceeded the 95th percentile cut-off for obesity at 8th grade – more than tripling the number of obese children since the 1970's. The CDC growth charts were compiled from a combination of data sources, among them (NHANES) I, II, and III surveys, U.S. Vital Statistics, the FELS longitudinal study, all collected in the period 1963-1994. Each of these datasets was collected in the period from 1963 to 1994, and thus before the steep gains in obesity rates across all age groups that occurred from 1994 on. One of the first implications for researchers and clinicians of these more recent data is that the CDC growth charts may no longer be considered normative. Health scientists may choose to use them as a target for the population distribution of children's BMI in the United States, but they do not reflect the current distribution.

Above and beyond the population-level shift in children's BMI, this study was concerned with how BMI change occurs over childhood. Raw BMI may generally increase, but whether a person remains at a healthy percentile, an unhealthy percentile, or fluctuates, and whether groups of individuals follow similar patterns, have been unknown. If different common patterns of change could be identified in early childhood, and the biological and social variables associated with the unhealthy patterns understood, sub-populations at risk could be more easily identified and treated. Early childhood variables were emphasized in these analyses, as the earlier families could be identified as at-risk for overweight, the earlier and more successful interventions would be.

To determine whether children gained weight over childhood at different time points and/or rates, two methods of growth class analysis were tested. Latent class growth analysis (LCGA), which keeps variance across the growth factors constant, was the starting point, as recommended for exploratory growth analysis (Jung & Wickrama, 2005; Muthen, 2009). LCGA is a specific case of growth mixture modeling (GMM); GMM does not keep variance constant, but allows it to vary and be estimated by the modeling process. GMM was also attempted, and the best LCGA and GMM models compared; a final model was then refined using advantages from each.

Clear, separate patterns of change over time were identified using the growth class modeling. The best LCGA model yielded 5 classes: high rising, median stable, average stable, low stable, and low-to-high rising. The low-to-high rising class was of particular interest, as it seemed to reflect a sharp and steady increase in BMI between 36 months and 6th grade that the other trajectories lacked. The best GMM model yielded 3 classes: high rising, median stable, and low stable; although these classes were larger, estimating the variance had also improved model fit – the main advantage of GMM. Thus I retained the low-to-high rising class distinguished by the LCGA model and used GMM for the remaining three classes, yielding a final 4-class solution: high rising, median stable, low-to-high rising, and low stable. These four patterns of change had highly significant and different intercepts (BMI percentile means), slopes, quadratic, and cubic terms, with good class posterior probabilities and entropy, establishing that in the SECCYD sample, BMI change was not homogeneous.

Over 30% of the sample was classified into the high-rising class, whose mean BMI percentile across the trajectory was over the 90th percentile, and 63% of whom were classified as obese (>= 95th percentile) at least once during childhood. I considered the low stable class the healthiest, with a mean BMI at the 29th percentile. The median stable and low-to-high rising classes had similar mean BMI percentiles (71st and 67th, respectively), but it was the median stable class that had the greatest number of children who had ever been overweight in childhood (69% vs. 44%). Whether there was a meaningful difference in health outcomes distinguishing these classes remained an empirical question for subsequent analyses.

The clinical implications of such heterogeneous weight change trajectories are significant. Today, the CDC growth charts (example, Figure 12A) are used to determine the percentile BMI a child falls into based on their gender, age, and raw BMI. A boy aged 36 months with raw BMI 15.75 falls approximately at the 35th percentile for BMI. The same child at 54 months with raw BMI 15.5 is in the 50th percentile. Without knowledge of patterns of change over time, neither of these percentiles would trigger concern for his weight status. Using the weight change trajectory classes (Figure 12B), however, would prompt immediate attention: a 15%-ile increase is large between these ages, and is associated with the low-to-high rising class, which is in turn associated with a much higher BMI across childhood than the low stable class. The same differentiation is observed between the median stable and high rising classes in the 36 month – Grade 1 window, with cause for even greater concern, perhaps, as these trajectory classes are much more common.

Upon visual inspection of the growth classes, the stable trajectories appear to start to diverge from the rising classes at 36 months and are clearly distinguishable by Grade 1, suggesting that this period may be critical for evaluating children's risk for membership in high-BMI trajectories. This finding is squarely in line with prior research on adiposity rebound, defined as weight gain following the initial drop at around 1 year of age. The sooner this rebound occurs, the faster and higher the rise in BMI over childhood (see Rolland-Cachera, Deheeger, Maillot, & Bellisle, 2006, for a review).

A. B.

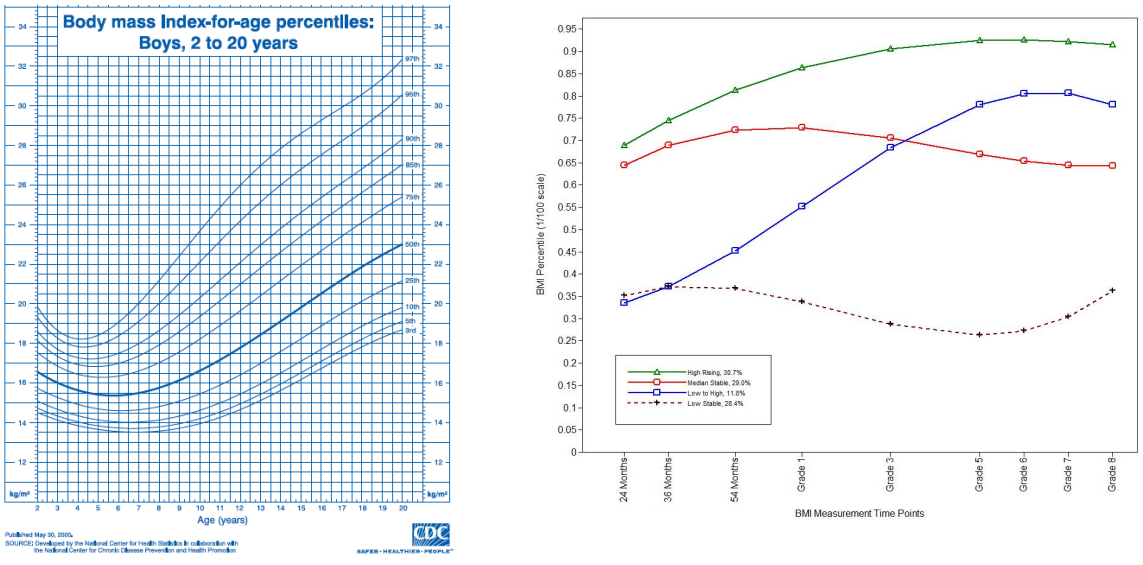

Figure 12. An example CDC 2000 BMI growth chart (A) vs. the final model of BMI percentile growth trajectories identified in the SECCYD data set (B).

Early adiposity rebound is defined as starting at around 36 months, as is also reflected here in the low-to-high rising and high rising classes, whose members appear to begin their rapid rise in BMI percentile at 36 months, with a divergence from the nearest

stable class observable by 54 months[1]. By contrast, in the stable classes, children are staying within about 10 BMI percentile points across childhood, maintaining their relative place in the BMI distribution of their peers. This additional evidence of rate of change-related risk should be of first interest to pediatricians and parents, as it appears independent of the initial BMI percentile starting point.

The next phase of analysis sought to determine just which early-life variables might put children at risk for membership in the high-BMI growth classes.

RQ 2A: CHARACTERIZING GROWTH TRAJECTORIES

Once individuals had been classified into their most likely growth trajectory class, the classes could be compared on the demographic, biological, and social differences between them. Using analysis of variance (ANOVA) tests, I documented all contrasts, but for this discussion focus primarily on differences between the low stable and high rising classes, as these represent the extremes of weight trajectory outcomes. Unexpected findings with respect to the other two classes which warrant consideration or future attention are also discussed.

Demographic variables. Of the three demographic variables tested – gender, identifying as Black, and identifying as Hispanic – only the first two were related weight trajectory membership. Boys were significantly more likely than girls to be in the high rising than in the low stable class, a finding consistent with recent prevalence analyses of the nationally representative NHANES datasets from 1988 – 2008 (Ogden & Carroll,

[1] In post-hoc regression of percentile BMI at 15 ½ years onto the first five weight/BMI measurements together (birth weight for age, and BMI percentile at 15, 24, 36, and 54 months), only BMI percentile at 54 months was significantly related to BMI percentile at 15 ½ years ($b = 0.63$, $p < .001$, not shown).

2010). Also consistent was the finding that children identified as Black were more likely than non-Blacks to be in all but the low stable class. Increased prevalence of obesity in the Hispanic population has also been documented in the same NHANES reviews, but only since 1994. The current study may not have found an increase associated with Hispanic origin because the SECCYD children were recruited in 1991, before the difference between Hispanic and non-Hispanic was significant, or because nationally representative stratified sampling was not employed in the SECCYD methodology. As a whole, trajectory membership of the SECCYD children from 1991-2006 appeared to be following the trends found in the U.S. national surveys.

Biological variables. The greatest distinction between the BMI growth classes was explained by the child's raw BMI at 15 months ($F = 69.5$, $p < .001$, $\mu^2 = .16$). Although the CDC does not consider BMI to be stable enough at this age to generate normative percentiles, BMI is an effective estimate of central adiposity in children of all ages (Duren, et al., 2008). Children in the low stable and low-to-high rising classes had lower BMI at 15 months than those in the median stable and high rising classes, suggesting that the 15-month time point might itself be included in future trajectory modeling. However, it did not distinguish between the low stable and the low-to-high rising, nor between the median stable and the high rising. In other words, BMI at 15 months appears associated with the starting points of the trajectories at 24 months, but is not associated with the very different rates of change, nor with the very different mean BMI percentiles, suggesting that there are other factors associated with the divergence of those classes.

The next greatest difference between classes does differentiate low stable from low-to-high rising and median stable from high rising: maternal BMI at 15 months. (Maternal BMI at 24 months is almost as strong, although it does not differentiate low stable from low-to-high rising.) Maternal obesity has been found consistently elsewhere to be related to infant obesity, but the mechanism of this transfer from mother to child remains unclear. The hypothesis, or myth, that obese mothers use food in unhealthy ways, by emotional feeding, as a reward, or in over-assertive prompting, has been debunked (e.g. Wardle, Sanderson, Guthrie, Rapoport, & Plomin, 2002). Lumeng et al. (2012) found no difference in the feeding behaviors of obese vs. normal-weight mothers in the SECCYD dataset. Other possibilities should be explored regarding the apparent transmission of overweight from mother to child. Biological mechanisms may be at work, such as prenatal maternal over-nutrition leading to changes in fetal metabolism, insulin, and endocrine system functioning (Whitaker & Dietz, 1998), preprogramming the child's cells toward fat retention. Environmental factors unaccounted for here, such as neighborhood quality and availability of fresh produce, could be affecting whole families. Shared practices of eating, physical activity, cooking at home, and television advertisement viewing could all be contributing to the normalization of overweight in the home.

Although the SECCYD dataset does not include maternal prenatal weight or measured BMI at 15 months, it has been supplemented since by ratings of maternal BMI at 15 and 24 months using the Stunkard Figure Rating Scale (Lumeng et al., 2012) on videoed observations of the mothers. Women may be expected to be heavier 15 months

post-partum than pre-conception, but if that trend is largely universal, maternal BMI at 15 months may serve as a relative measure of women's BMI during pregnancy. To the extent that we can consider a mother's BMI at 15 months to be a proxy for her weight status during pregnancy, these findings may support the prenatal over-nutrition theory: that it is intra-uterine developmental changes that have programmed the infant's metabolic regulatory systems toward fat retention (as reviewed in Heerwagen et al., 2010).

Prenatal over-nutrition may thus explain why maternal overweight/obesity in turn usually results in infant high birth weight for gestational age, a consistently significant predictor of overweight and obesity in childhood (Dubois & Gerard, 2006; see meta-analysis: Yu et al., 2011). This study found mean infant birth weight for age to differ significantly across the classes, with the members of the high rising class having higher birth weights than those in the low stable and low-to-high rising classes. In terms of clinical application, however, this finding is somewhat ambiguous. Birth weight for age alone does not distinguish between the median stable and the much higher-BMI high rising classes, nor between the low stable and the low-to-high rising classes. As with child's BMI at 15 months, birth weight seems to differentiate the trajectory classes by starting point – low-starters from high starters – but does not pre-determine the child's weight status across childhood. In practical terms, then, a high birth weight should trigger immediate attention to proper nutrition in the first few years of life, to ensure that the child is not beginning on a high rising trajectory. That said, low birth weight is not protective for the ~ 12% of children in the low-to-high rising class.

Very early increase in BMI, in this study measured by the percentage of weight gained between birth and 15 months, had a similar profile in distinguishing the weight trajectory classes as birth weight. The low stable class increased less during that period than the median stable or high rising classes, but was not distinguished from the low-to-high rising class. During this earliest period, the low-to-high rising class was only distinguished from the high rising class in having a smaller increase; this is no doubt a result of the low-to-high rising class experiencing its rapid increase in later childhood. However, again this variable did not distinguish the low stable from the low-to-high rising trajectory; differentiating those classes with biological pre-trajectory variables remained difficult.

The final biological variable to differentiate the classes was maternal smoking. This measure, collected from maternal report about her prenatal behavior at 24 months, was treated as a scale: 1) Did not smoke; 2) Smoked, but quit before pregnancy began; 3) Smoked, and stopped during first three months of pregnancy; 4) Smoked during first 3 months, stopped prior to birth; and 5) Smoked throughout the year. Given the social pressure against smoking during pregnancy, and the subsequent tendency of mothers significantly to under-report both smoking at all and amount smoked (Ford, Tappin, Schluter, & Wild, 1997), it is a testament to the strength of the effect of smoking that this item was significantly different across the growth classes. Mothers of children in the low stable and median stable classes reported significantly less smoking than those of children in the high rising class, supporting prior findings that maternal smoking in any amount during pregnancy is associated with risk for obesity in early childhood (Pryor et

al., 2011; Reilly et al., 2005; see Behl et al., 2013 for a review). The low-to-high rising class had higher levels of maternal smoking, but non-significantly; that null finding could be a result of low statistical power or high variability, or because the much later increase in weight in childhood in that class is not associated with prenatal smoking.

Breastfeeding. One null finding among the biological variables was of particular interest: whether the child had ever been breastfed. Although breastfeeding has been found to be protective against later obesity in prior studies, primarily in univariate analyses (Armstrong & Reilly, 2002; Reilly et al., 2005), these effects seem to disappear when confounders such as maternal overweight, maternal smoking, child's catch-up weight gain, and socio-economic status are included. In this study, univariate logistic regression of trajectory class onto the binomial ever-breastfed variable did not identify any difference in breastfeeding across the classes. Both Bogen, Hanusa, & Whitaker (2004) and Reilly et al. (2005) found an interaction between maternal smoking and maternal breastfeeding, such that breastfeeding was protective against obesity only for children of non-smokers. In post-hoc analyses I also tested this interaction for any univariate association with growth class membership or weight and blood pressure outcomes; no significant relations were present. Neither breastfeeding alone nor in interaction with maternal smoking predicted weight and blood pressure outcomes at 15 ½ years in logistic and regression analyses which included the above confounds.

Recent research has suggested that the effect of breastfeeding might be mediated by the development of the child's palate, i.e., that the different flavors that come through in breast milk foster an early tolerance for different flavors, particularly vegetables. As a

child's food preferences and diet in the first year of life affects her food choices later in childhood, and thereby her weight, breastfeeding may start a child on a more healthful path (reviewed in Saint Louis, 2014). This study did not include a reliable measure of breast-feeding duration, nor dietary information, so this hypothesis could not be tested.

Psychosocial variables. In a 2011 review of cross-sectional and longitudinal studies modeling childhood obesity from psychosocial stressors, the stressors of maternal mental and physical health, general stress, housing and financial insecurity, and negative life events, were found to contribute to adversity-related weight outcomes (Gundersen et al., 2011). The current study tested maternal depression (at 1, 6, 15, and 24 months), maternal physical health (average maternal report 0-24 months), the sum of maternal stresses (0-15 months), the average income-to-need ratio (0-24 months), and negative life events (54 months) for differences across the BMI growth trajectories in the effort to replicate these findings. Average maternal sensitivity (6-24 months) as assessed in mother-child interactions in a laboratory setting was also tested.

Membership in both the low stable and median stable classes was associated with a greater income-to-need ratio than that experienced by members of the high rising class. The finding that stability in BMI percentile and in lower overall BMI were both associated with greater relative wealth mirrors prior findings in univariate analyses that obesity has increased the fastest among low-income children (CDC, 2014). Although controlling for parental overweight/obesity has been found to weaken the relation between low SES in early life and later obesity (see Parsons, Power, Logan, & Summerbell, 1999), its direct effect remains (Dubois & Girard, 2006; Li et al., 2007).

Potential confounding by low-income mothers' overweight made multivariate analysis of the health outcomes in this study, discussed below, of particular interest.

Maternal depression has been associated with greater likelihood of having overweight and obese children, and a lesser likelihood of limit-setting, restricting their child's intake, and modeling healthy eating (Gross, Velzco, Briggs, & Racine, 2013). In this study, maternal depression significantly distinguished the low stable from the high rising class in the expected direction (greater maternal depression associated with greater weight gain), albeit with small effect sizes ($\mu^2 = .01$).

However, clues as to what constitutes maternal sensitivity and the mechanisms by which sensitivity might affect weight remain scarce. Maternal sensitivity distinguished the low stable and median stable groups from the high-rising group, which may be a sign that sensitivity is related less to BMI percentile itself than to change in that percentile. Sensitivity was related to stability and avoidant attachment related to the low-to-high rising class, suggesting that some common hallmarks of sensitivity and attachment security, such as contingent, available, and non-intrusive care, may supply the young child with stress management skills other than emotional eating.

That interpretation of course assumes that stress and stress management are influential on weight gain. In this study, stress in earliest childhood was taken directly from the mother's report of her stress, under the assumption that her stresses and stress management would directly influence her infant's experience of sensitive care and safety. The earliest measures of maternal stress available were assessed at 6, 12, and 15 months; this study used the accumulation of these endorsed events: job loss of close friend or

relative, illness/injury/addiction of close friend or relative, death of anyone close, any other major life event occurred, and the number of times household had moved. Neither the sum of these stresses nor the number of negative life events endured at 54 months were significantly related to BMI growth class membership. However, this null finding was not necessarily unexpected. If there are individual differences in stress management and in the effects of stress on eating behavior, a direct effect of stress on weight would not appear. This study rather tested the hypothesis that attachment classification, as a reflection of a child's stress management strategy, would moderate the effect of stress on weight trajectory.

RQ 2B: Stress, Attachment Security, and BMI Change

Children in dyads classified as avoidant were more likely to be in the low-to-high rising class than in the low stable class; the opposite relation was marginally significant for children in anxious-resistant dyads. Children in avoidant dyads do not approach their primary care-giver for comfort or a secure base as secure children do. They are often identified in preschool and middle childhood as having trouble with peer relationships due to anger and hostility. If they are less well-adapted and relate poorly to others as they enter preschool and primary school, could that stress, unmeasured here, trigger food-related coping? Clearly more research is called for.

This study also explored the possibility that attachment classification moderated the influence of stress on overweight/obesity. Stress alone was not expected to trigger overweight, as some individuals might gain weight along HPA-axis inflammatory pathways, but some might lose weight, if adrenaline or anxiety reduced appetite, for

example. I hypothesized that children's attachment security, as demonstrated in the Infant Strange Situation protocol, might be considered a classification of stress management pattern. Avoidant children might not be able to engage with stress actively, leading them to use less-adaptive coping strategies, like eating; anxious-resistant children might be heavier in general, using food to soothe anxiety, but in conditions of stress might lose weight, as the increasing anxiety over their baseline made eating a source of control or discomfort or both, analogous to the findings among preoccupied adults.

This hypothesis was partially supported in the current study. Avoidant children were no more likely than secure children to be in the high rising BMI percentile trajectory class under conditions of greater stress. Anxious-resistant children, however, were more likely to be in the high rising trajectory class under lower stress and less likely under higher stress. To the extent that children's anxious-resistant behavior is analogous to adults' preoccupied speech, this finding mirrors a recent waist circumference finding in Bichteler & Jacobvitz (under review; Figure 13) in which adult attachment classification as measured with the Adult Attachment Interview (AAI) moderated the influence of the accumulation of major lifetime stresses (terrible events) on adiposity. The two studies had very different samples, covariates, and attachment measures, but the results of each suggest that attachment behavior and states of mind may help explain why, although biological stress reactivity pathways are so well-documented and promote fat retention, measured stress has had such a negligible association with weight outcomes in human subjects research. The apprehension and management of stress may not be

homogenous in the population; individuals of all ages may employ mental patterns formed from earliest experiences of care in their response to stress.

In summary of RQ2 regarding characteristics of the weight trajectory classes, analyses demonstrated that significant individual differences differentiate the BMI weight trajectory classes. Biological factors such as child and mother BMI at 15 months, birth

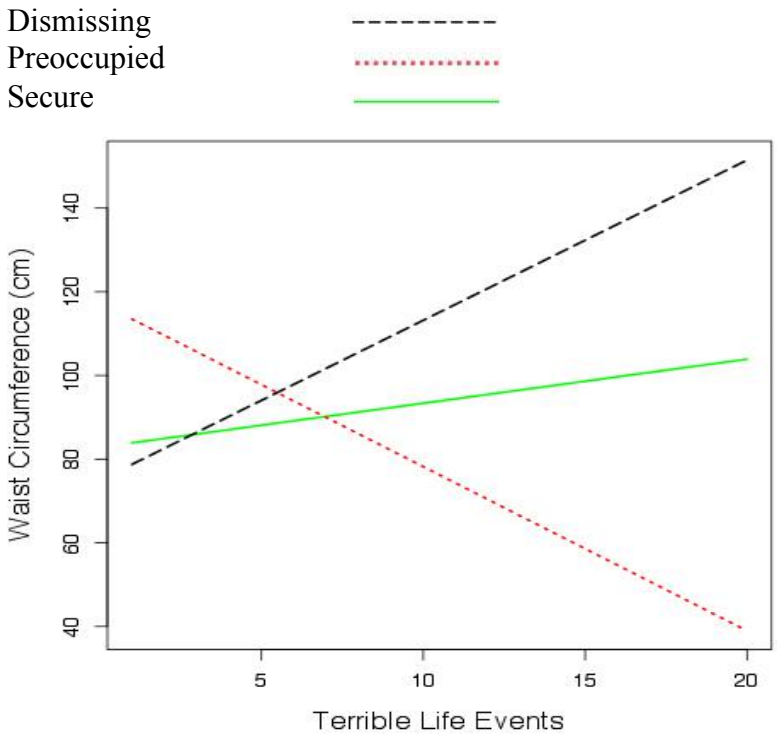

Figure 13. Attachment-related speech measured with the Adult Attachment Interview moderates the association between the number of terrible life events endured across the lifespan and waist circumference in middle age (Bichteler & Jacobvitz, under review).

weight for age, and percent increase in weight from 0-15 months explained the greatest differences; to a lesser extent psychosocial factors maternal sensitivity, income-to-need

ratio, maternal depression, and attachment security also separated the low stable from high rising weight trajectory classes. Race (Black/not Black) was also associated with every class, and particularly the high rising class, over against the low stable class, replicating prior findings on minority status and weight in the United States.

It is worth noting that few of the class differences in risk identified by the ANOVA and logistic regression analyses were of a meaningful size. Most had effect size μ^2 or R^2, depending on the statistical test used, of < .05. Although heterogeneous trajectories had emerged quite clearly in the latent growth analysis, the variables tested across the classes did not explain much of their separateness, all told. Some important candidate variables that might have accounted better for class differences were not present in the SECCYD dataset, such as feeding practices, food preferences, exercise vs. sedentary lifestyle, sleep duration, and timing of puberty. Differences in their composition were not well-demonstrated by the variables in this study, but the classes themselves proved very powerful in predicting adiposity and blood pressure outcomes in adolescence.

RQ 3: PREDICTING ADIPOSITY AND BLOOD PRESSURE AT 15 ½ YEARS

Latent class growth modeling had determined to which BMI percentile change class each child belonged. Linear and regression modeling then determined how much a child's membership in his change class influenced his adiposity and blood pressure at 15 ½ years old. I refined best-fitting explanatory models, one that included growth class membership and one that didn't, for each of the five health outcomes: BMI percentile, waist circumference, overweight (1 = yes), obesity (1 = yes), and high blood pressure (1

= yes). Including early childhood risk factors with trajectory class membership in the best-fitting models determined which factors retained a direct influence on health outcomes over and above a child's pattern of weight change.

The linear regressions of continuous BMI percentile and waist circumference yielded the most nuanced models of adiposity at 15 ½ years. Membership in the low stable weight change trajectory, relatively flat, with a mean of the 29th percentile BMI, significantly negatively predicted both outcomes vis-à-vis all other classes. Class membership increased the variance explained in percentile BMI and waist circumference over the best model without class membership by 31% and 19%, respectively. However, several early childhood risk factors also predicted these outcomes directly, over and above a child's change trajectory.

In light of prior findings that even normal-weight infants born to obese or overweight women have increased adiposity and risk for metabolic syndrome, it is noteworthy that this study found maternal BMI not only to be associated with differences between the weight trajectory classes, but also directly to predict BMI percentile and waist circumference at 15 ½ years, even when birth weight for age is controlled for. This study thus supports the hypothesis reviewed in Heerwagen et al. (2010) that intra-uterine inflammation and higher blood lipids permanently alter the fetus's gene expression via epigenetic modifications that are then maintained through feedback loops and permanently change the expression of metabolic genes and gene transcription.

Strong evidence also exists for an interaction effect between birth weight for age and percent increase in weight from 0-15 months, which further supports the theory that

rapid weight gain at any time during early development is detrimental, and particularly when birth weight is already high. Conversely, *not* gaining weight rapidly appears to be protective against the influence of high birth weight: regardless of birth weight, children who do not gain weight rapidly between 0-15 months are not at risk for high BMI at 15 ½ years. That this interaction persists, over and above the high rising versus median stable trajectory split which is already accounted for, demonstrates its diagnostic utility across the range of weight and weight trajectories.

Implications for triggers and timing of clinical intervention of these two findings are profound: mothers' BMI, children's birth weight for age, and rapid increase in their weight are all variables directly predicting ongoing weight gain throughout childhood, high BMI at 15 ½ years, and greater waist circumference, and are all identified at 15 months or younger. Improvement in these causal factors at the earliest opportunity should be a priority for health care providers.

Among the early childhood psychosocial variables, greater maternal sensitivity measured between 6-24 months was directly negatively related to both BMI percentile and waist circumference at 15 ½ years. Not only did it distinguish the stable classes from the high rising class in the ANOVA test, it exerted a separate influence on health outcomes when those classes were present in regression modeling. One plausible mechanism for this relation is the negative relation between maternal sensitivity and assertiveness and intrusiveness in feeding in the SECCYD sample (Lumeng et al., 2012), feeding behaviors which were associated with childhood obesity. Contingent care,

recognized by a mother's meeting her child's needs promptly and accurately, may also minimize confusion of food for other needs.

The effect of attachment security on BMI percentile at 15 ½ years is difficult to assess when maternal sensitivity is controlled for. It appears that when maternal sensitivity is accounted for, secure children's percentile BMI is likely to be higher than anxious-resistant children's. This could be due to other factors in common with anxious-resistant children, such as greater anxiety. Of course, this confounding may operate in the other direction: as maternal sensitivity is known only partially to account for attachment security; does this model then demonstrate an effect of sensitivity *other* than that already captured by attachment classification? For attachment researchers, pursuing lines of enquiry exploring different health outcomes of sensitivity vs. security may help disentangle the two.

The best logistic regression models of overweight, obesity, and high blood pressure were considerably simpler. What was lost by dichotomizing these continuous outcomes could be considered gained in simplicity of interpretation. Overweight and obesity at 15 ½ years were predicted by membership in all classes relative to the low stable class. Low-to-high rising class members were in a significantly higher BMI percentile at 15 ½ years than the median stable class: compelling evidence that stability, even stability around the 71st percentile of BMI, predicts lower BMI than starting low but rapidly increasing throughout childhood. The rate of change as reflected in the trajectory classes added 24% and 12% to the variance explaining overweight and obesity, respectively.

Maternal education (negative relation) and maternal BMI were consistently related to both dichotomous weight outcomes over and above the change trajectory's influence; income-to-need ratio and female gender also predicted overweight. But important predictors in the continuous regressions were absent: birth weight, weight increase, and BMI at 15 months, for example. The change trajectories seemed to account for these biological factors fully, leaving only SES-related variables (but not Black race!) as significant direct effects. An important methodological lesson learned in this study should be that choices to dichotomize weight-related outcome variables should be made carefully and reported mindfully, as modeling results, and therefore clinical implications, depend heavily on this choice.

High blood pressure was the health outcome the least well-explained by either early childhood risk factors' direct effects or by weight change trajectories, with only 18% of the variance in high blood pressure at 15 ½ explained by both sets of factors. Membership in the high rising and median stable trajectories, but not the low-to-high rising, versus the low stable trajectory predicted high blood pressure at 15 ½ years, as did being male, and (marginally) maternal pre-natal smoking. In a model controlling for gender, BMI percentile at 15 ½ years was strongly related to high blood pressure at that age (OR = 16.97, $p < .001$, CI (8.01 – 35.96), Tjur's $R^2 = 0.16$), suggesting that the childhood trajectory classes and early childhood covariates may have an indirect association with blood pressure through their associations with BMI. Overweight at the same time point is clearly related to high blood pressure, but the pattern of reaching that overweight, and the earliest childhood predictors of overweight, are not strongly

implicated as causes in this study. Clearly there are other mechanisms, unmeasured or untested here, at work in the development of high blood pressure in adolescents.

LIMITATIONS & STRENGTHS

Despite the success of the models described above, this study did have some limitations. First, the modest relations between earliest childhood risk factors, weight trajectories, and high blood pressure in adolescence point to missing covariates and/or explanatory factors in the reported models. Although high BMI was predicted strongly (R^2 = 0.50) by the included risk factors and trajectories, and high BMI was associated with high blood pressure, the risk factors at the time points available did not explain high blood pressure well (R^2 = .18), suggesting that other factors or changes in them across childhood unmeasured in this study would be needed to explain blood pressure at 15 ½. Second, as the focus of the study was on earliest childhood predictors, the trajectory modeling did not include time-varying covariates throughout childhood. A future study that includes the key variables that emerged as important predictors in this study measured across all data points and not just early childhood will be an important next step in understanding the associations revealed in this study. Third, the SECCYD dataset does not include several variables that have been linked with childhood weight and obesity in previous research, including children's eating habits and nutrition, sleep duration, TV watching, exercise, pubertal timing (which was only available for a small subset of children in the SECCYD and so not used). These variables may be needed to increase the percent of variance explained in the 15 ½ year outcomes.

The current study also has a number of strengths. First, the SECCYD dataset contained myriad weight-related variables from birth to adolescence, for a relatively large sample of 1,364 children in 10 different U.S. regions, which sets it apart from previous research. The typical study on correlates of children's weight focuses on a cross-section of a sample population and often a cross-section of time. The SECCYD afforded the examination of growth trajectories across 12 data points, the large number of which gave added precision to the growth trajectories. Second, the presence of demographic, biological, and psychosocial factors measured for the same children across time allowed testing of all of these factors together and the opportunity to understand their relative importance in the prediction of adolescent health outcomes. Third, the large sample size in this study eliminated the usual issues in growth modeling that can arise from skewed data and used two sophisticated analytic techniques, namely growth curve modeling and latent class modeling, to build a clear picture of change over time. The result of such modeling was a significant refinement in our understanding of longitudinal change in BMI percentiles across different sub-groups of children and their earliest risk factors for weight gain.

CONCLUSION

An analysis of the SECCYD longitudinal data from birth to mid-adolescence, with its many waves of BMI and complex social, demographic, and relational measures, had many benefits. Trajectories of weight gain over that entire period were estimated, yielding new information about developmental change that may be used to model paths of overweight and obesity. These trajectories demonstrated conclusively that weight

change is not homogeneous across all children, and that it is a combination of starting point and rate of increase that predicts risk. Furthermore, the diagnostic combination of trajectory-related risks can be observed as early as 36-54 months. The wealth of environmental influences included in SECCYD dataset allowed prediction with both known and ambiguous risk factors, which better inform our understanding of which of these factors exert and retain their influence in complex multivariate modeling. The rare inclusion in such a large dataset of the time-intensive Strange Situation Procedure also offered the opportunity to explore physical health outcomes of early infant attachment patterns, particularly in the context of adversity.

The nature of BMI change over time as defined by the BMI percentile growth classes was powerfully related to weight at 15 ½ years. We were able to ascertain which trajectories of overweight in childhood contributed to longer-term risks to weight and heart health. Children who gain weight rapidly after birth are at highest risk for later fatness. Although children in the low-to-high rising class have low birth weight, their steady increase in BMI starting at 36 months and continuing over childhood means that they end up with higher BMI at 15 ½ years than do children in the median stable class, whose birth weights were on average 30 percentage points higher. Birth weight and rapid early increase in BMI can now provide critical diagnostic data well before a child has been classified as overweight (the 85th percentile of BMI), when it may be much more difficult to intervene. Determinants of trajectory membership remain less clear, as factors such as maternal BMI, maternal sensitivity, and SES appear to play only a small role.

That the risk factors for class membership appear already between birth and 15 months should encourage more research into the predictors of membership in trajectory growth classes. Identifying and intervening with families and children in the high and rapidly rising weight trajectories should be the focus of obesity prevention efforts for the highest value to public health.

Appendix

Table A1.

Best linear regression model predicting percentile BMI at 15 ½ years from early childhood risk factors (0-24 months), without change trajectory membership.

	B (SE)	*b*	t-value	*p*
Intercept	0.13 0.28		0.48	
Gender: female	0.03 0.02	0.07	1.93	†
Birth weight for age	-0.00 0.00	-0.04	-0.32	
Maternal BMI 15 mo.	0.04 0.01	0.21	6.20	***
Percent weight increase, 0-15 mo.	-0.13 0.11	-0.21	-1.14	
Maternal average sensitivity	-0.02 0.01	-0.09	-2.29	*
Income-to-need ratio average, 0-24 mo.	-0.01 0.00	-0.07	-1.75	†
Hispanic: Yes	0.46 0.22	0.40	2.07	*
Maternal education, yrs.	-0.01 0.00	-0.05	-1.16	
Child BMI, 15 mo.	0.03 0.01	0.13	2.67	**
Hispanic: Yes x Maternal education, yrs.	-0.04 0.02	-0.07	-2.37	*
Birth weight for age x Percent weight increase, 0-15 mo.	0.00 0.00	0.13	2.15	*
Model fit				
R^2	0.19			
AIC	12.06			

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. Variables with at least marginal significance were retained. Figure of the Hispanic by maternal education moderation effect is in Figure A1. The birth weight for age by percent weight increase moderation effect mirrors that found in Figure 11, i.e. greater weight increase is associated with the highest BMI percentile at 15 ½ years among those born the heaviest, and lowest weight increase buffers the effect of high birth weight.

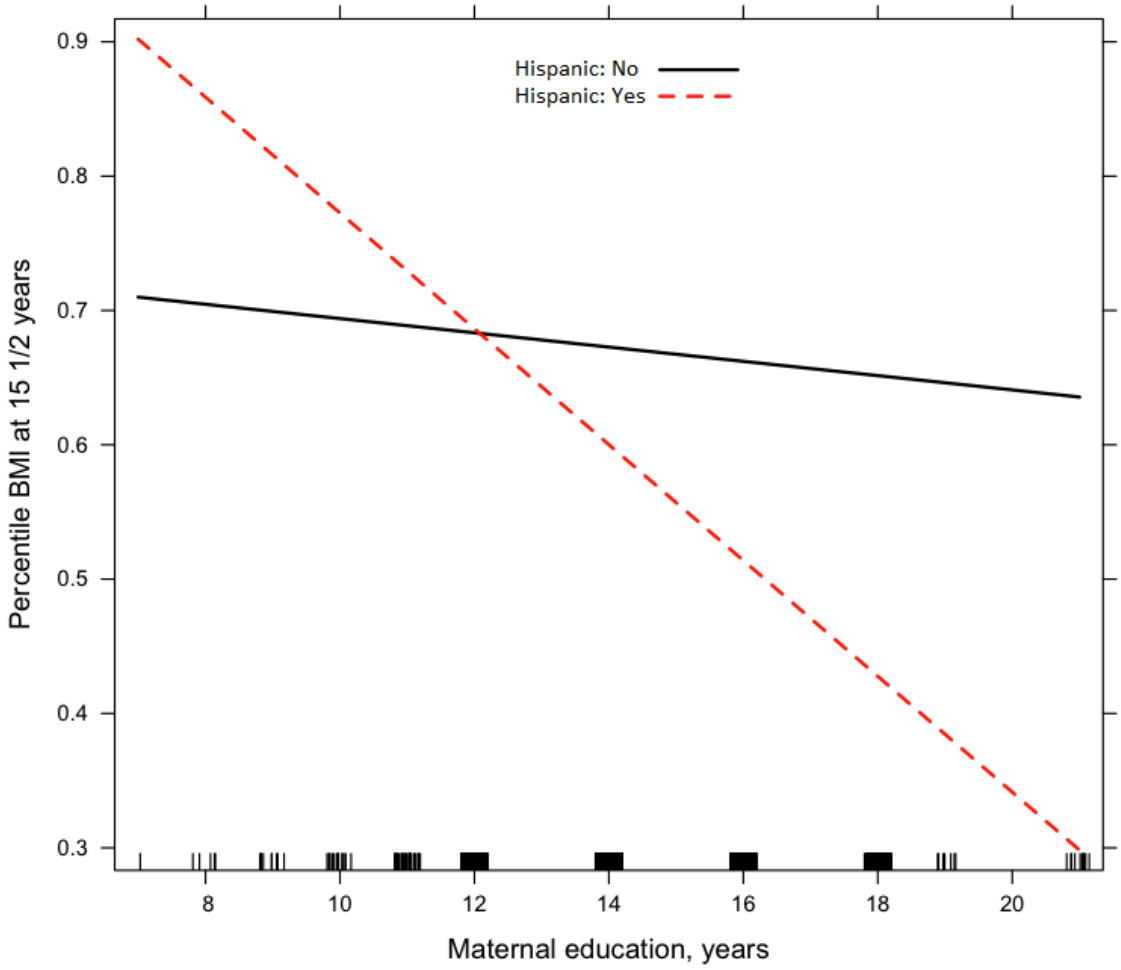

Figure A1. Education moderates the influence of Hispanic heritage on percentile BMI at 15 ½ years: lower education is associated with greater BMI and higher education with less.

Table A2.

Best linear regression model predicting waist circumference at 15 ½ years from early childhood risk factors (0-24 months), without change trajectory membership.

	B (SE)	*b*	t-value	*p*
Intercept	25.71 (7.39)		3.48	***
Gender: female	-3.87 (0.80)	-0.16	-4.84	***
Hispanic: Yes	18.28 (9.88)		1.85	†
Maternal education, yrs.	-0.11 (0.19)		-0.59	
Birth weight for age	0.01 (0.00)	0.00	7.46	***
Maternal BMI, 15 mo.	1.94 (0.26)	72.64	7.39	***
Maternal prenatal smoking	0.57 (0.33)	0.07	1.73	†
Percent weight increase, 0-15 mo.	9.28 1.28	0.96	7.27	***
Maternal average sensitivity	-0.67 (0.35)	-0.02	-1.91	†
Hispanic: Yes	18.23 (9.88)	1.95	1.85	†
Maternal education, yrs.	-0.11 (0.19)	-0.00	-0.59	
Hispanic: Yes x Maternal education, yrs.	-1.39 (0.72)	-0.28	-1.95	†
Model fit				
R^2		0.25		
AIC		5988.5		

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. Variables with at least marginal significance were retained. Of the 45 contrasts between sites, one study site was significantly different from two others, ($p < .05$), therefore site was retained, but is not shown.

Table A3.

Best logistic regression model predicting overweight (>= 85th percentile BMI) at 15 ½ years from early childhood risk factors, without change trajectory membership.

	OR	(CI)	*p*
Birth weight for age	1.00	(1.00 – 1.00)	
Maternal BMI, 15 mo.	1.44	(1.29 – 1.61)	***
Percent weight increase, 0-15 mo.	0.27	(0.03 – 2.42)	
Income-to-need ratio average, 0-24 mo.	0.90	(0.82 – 0.99)	*
Maternal education, yrs.	0.91	(0.84 – 1.00)	*
Child BMI, 15 mo.	1.17	(0.98 – 1.40)	†
Birth weight for age x Percent weight increase, 0-15 mo.	1.00	(1.00 – 1.00)	†
Model fit with trajectory membership			
Coefficient of discrimination *D*		0.18	
Residual deviance (df)		850 (789)	
AIC		884.0	

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. The low stable trajectory is the baseline trajectory contrast; secure is the baseline attachment classification contrast. Variables with at least marginal significance were retained. The birth weight for age by percent weight increase moderation effect mirrors that found in Figure 11, i.e. greater weight increase is associated with the highest BMI percentile at 15 ½ years among those born the heaviest, and lowest weight increase buffers the effect of high birth weight. Of the 45 contrasts between sites, two were significantly related to obesity ($p < .05$), therefore site was retained, but is not shown.

Table A4.

Best logistic regression model predicting obesity (>= 95th percentile BMI) at 15 ½ years from early childhood risk factors (0-24 months, without change trajectory membership.

	OR	(CI)	*p*
Birth weight for age	1.00	(1.00 – 1.00)	
Maternal BMI, 15 mo.	1.72	(1.50 – 1.98)	***
Maternal prenatal smoking	1.16	(0.98 – 1.37)	†
Percent weight increase, 0-15 mo.	0.65	(0.05 – 7.66)	
Maternal education, yrs.	0.89	(0.81 – 0.99)	*
Birth weight for age x Percent weight increase, 0-15 mo.	1.00	(1.00 – 1.00)	†
Model fit with trajectory membership			
Coefficient of discrimination *D*		0.19	
Residual deviance (df)		561 (790)	
AIC		593.3	

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. The low stable trajectory is the baseline trajectory contrast; secure is the baseline attachment classification contrast. Variables with at least marginal significance were retained. Of the 45 contrasts between sites, four were significantly related to obesity ($p < .05$), therefore site was retained, but is not shown.

Table A5.

Best logistic regression model predicting high blood pressure at 15 ½ years from early childhood risk factors (0-24 months), without change trajectory membership.

	OR	(CI)	*p*
Gender: Female	0.29	(0.20 – 0.42)	***
Birth weight for age	1.00	(1.00 – 1.00)	*
SS: Avoidant vs. secure	1.60	(0.75 – 3.43)	
SS: Anxious-resistant vs. secure	1.77	(0.81 – 3.88)	
Maternal sum of stresses, 0-15 mo.	1.07	(0.95 – 1.19)	
Maternal BMI, 15 mo.	1.16	(1.04 – 1.30)	**
Maternal prenatal smoking	1.15	(1.01 – 1.32)	*
Percent weight increase, 0-15 mo.	2.30	(1.34 – 3.95)	**
Sum of stresses x Avoidant vs. secure	0.86	(0.64 – 1.16)	
Sum of stresses x Resistant vs. secure	0.73	(0.53 – 1.00)	†
Model fit with trajectory membership			
Coefficient of discrimination *D*		0.15	
Residual deviance (df)		820.2 (797)	
AIC		860.2	

† $p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$; two-tailed.

Note. SS = strange situation protocol. Variables with at least marginal significance were retained. Of the 45 contrasts between sites, one study site was significantly different from four others, ($p < .05$), therefore site was retained, but is not shown.

Strange Situation Classifications: A = Avoidant; B = Secure; C = Anxious/Resistant

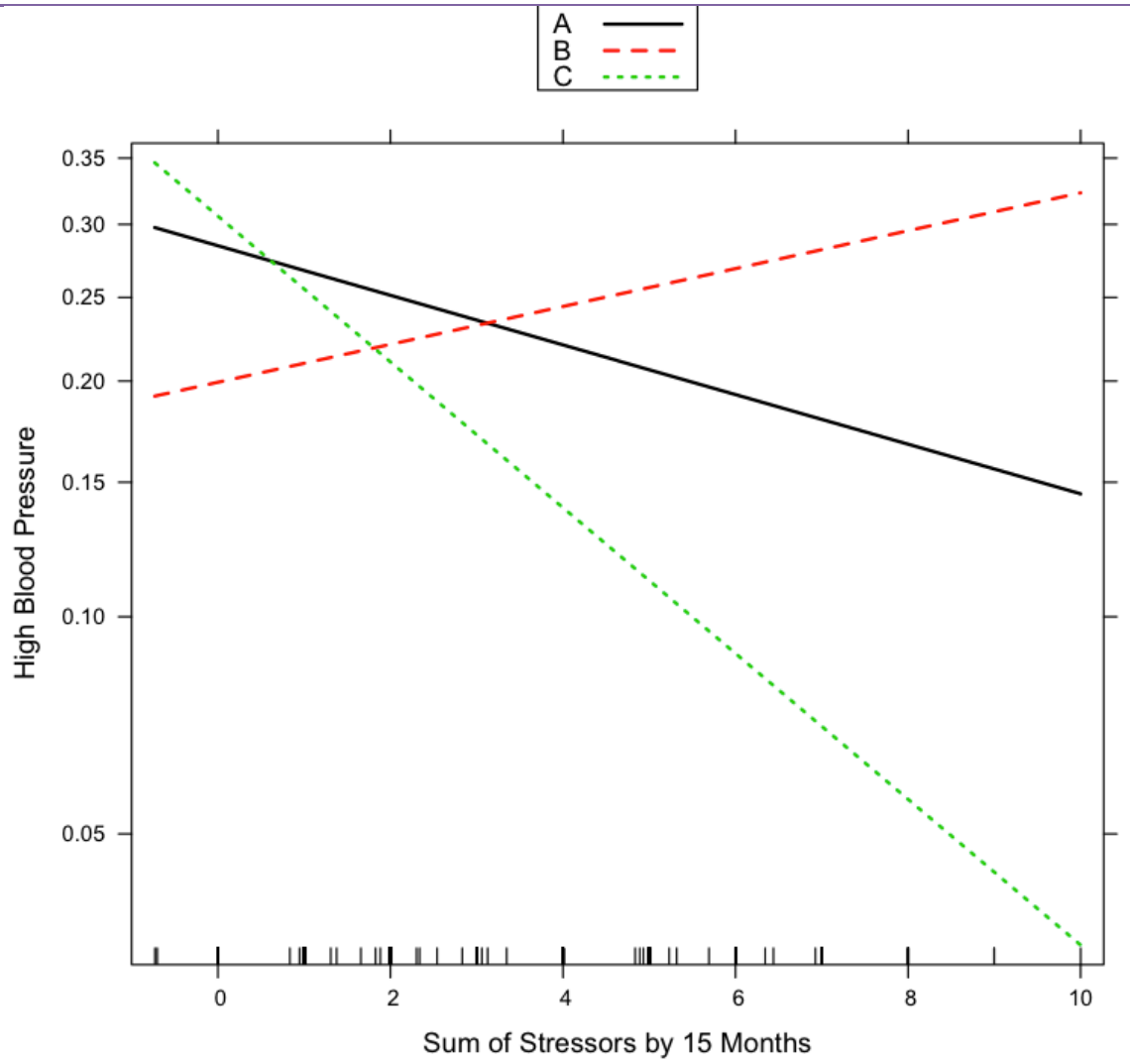

Figure A2. Strange situation classification at 15 months moderates the influence of stresses on high blood pressure at 15 ½ years.

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