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**EDUCATION**

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**PUBLICATIONS**

- Pecukonis, E., Greeno, E., Hodorowicz, M., **Park, H.**, Ting, L., Moyers, T., ... & Wirt, C. (2016). Teaching Motivational Interviewing to Child Welfare Social Work Students Using Live Supervision and Standardized Clients: A Randomized Controlled Trial. *Journal of the Society for Social Work and Research*, 7(3), 479-505.
- Oke, M., Groza, V., **Park, H.**, Kalyanvala, R., & Shetty, M. (2015). The perceptions of young adult adoptees in India on their emotional well-being. *Adoption & Fostering*, 39(4), 343-355. doi: 10.1177/0308575915611776
- Groza, V., **Park, H.**, Oke, M., Kalyanvala, R., & Shetty, M. (2014). A study of adult adoptees in India placed through BSSK in Pune: Adoption and birth family issues. *Indian Journal of Social Work*, 75(2), 285-300.
- Park, H.**, Barth, R. P., & Harrington, D. (2013). Factor structure of adoptive parent-child relationship items from the national study of adoptive parents. *Journal of the Society for Social Work and Research*, 4(1), 20-30. doi: 10.5243/jsswr.2013.2

Liao, M., Dababnah, S., & **Park, H.** (accepted, 2017). Relationship between disabilities and adoption outcomes in African American children. *Journal of Child and Family Studies*.

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Pecukonis, E., **Park, H.**, Greeno, E., & Hodorowicz, M. (2016). *Teaching Motivational Interviewing to child welfare social work students using live supervision and standardized clients: A randomized control trial*. Paper accepted for presentation at Society for Social Work and Research 20<sup>th</sup> Annual Conference, Washington, D.C.

Pecukonis, E., **Park, H.**, Greeno, E., & Hodorowicz, M. (2015). *Teaching and assessing motivational interviewing using supervision and simulation: A randomized control trial*. Paper accepted for presentation at the Council on Social Work Education Annual Program Meeting, Denver, CO.

Ting, L., Greeno, E., & **Park, H.** (2015). *Training child welfare social work students in motivational interviewing: exploring empathy's role*. Paper accepted for presentation at the Council on Social Work Education Annual Program Meeting, Denver, CO.

Burry, C., Linsenmeyer, D., Wirt, C., Wade, K. & **Park, H.** (2014). *Field of dreams to reality: Evaluating MI in public child welfare education*. Paper presented at the Council on Social Work Education Annual Program Meeting, Tampa, FL.

Pecukonis, E., O'Reilly, N., & **Park, H.** (2013). *Incorporating interprofessional education in social work health curriculum*. Paper presented at the Council on Social Work Education Annual Program Meeting, Dallas, TX.

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**Park, H.** (2016). *Using motivational interviewing techniques for psychiatric rehabilitative program workers*. King Health Systems, MD.

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## TEACHING EXPERIENCE

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Master's foundation level course.  
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- Instructor for SOWK 670 Social Work Research (Hybrid course). Master's foundation level course.
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2014-Present                **Psychotherapist (LGSW)**  
King Health Systems, Baltimore, MD

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Case Western Reserve University Counseling Center,  
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2008-2009                **Social Work Intern**  
Cleveland Christian Home (School-based mental health),  
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2010-2011                **Student Representative**, Ph.D. Program Committee  
School of Social Work, University of Maryland, Baltimore

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- 2008–2010           **Director of Programming**, Mandel Student Council  
Mandel School of Applied Social Sciences, Case Western Reserve University
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Germaine Lawrence School (Residential treatment services), Boston, MA
- 2004-2005           **Mentor**  
Korean-American Students Association, Big Sibling/Little Sibling program, Hanover, NH
- 2002-2003           **Volunteer**  
Autism Society in New Hampshire, Lebanon, NH

#### **MEMBERSHIP IN PROFESSIONAL ASSOCIATIONS**

- 2008-Current        National Association of Social Work (NASW)
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## Abstract

Title of Dissertation: Assessing the Relationship between Adverse Childhood Experiences and Body Mass Index Trajectory of Children and Adolescents

Hyeshin Park, Doctor of Philosophy, 2017

Dissertation Directed by: Dr. Richard Barth, Professor and Dean, School of Social Work

**Background:** More than a third of American children and adolescents are overweight or obese. Because childhood obesity is a risk factor for various health, mental health, and socioeconomic problems in adulthood, health practitioners, policy makers, and researchers continue to identify growth trajectories and clarify risk factors for unhealthy growth trajectories. The purpose of this dissertation was to identify subcategories of children who follow different *body mass index* (BMI) trajectories, describe these groups, and explore whether adverse childhood experiences (ACEs) predict group membership.

**Methods:** A sample of children who participated in the Longitudinal Studies on Child Abuse and Neglect (LONGSCAN) study at the Eastern site (Baltimore, MD), and whose demographic and BMI data were collected at age four, were included in the study ( $n=201$ ). Latent Class Growth Analysis (LCGA) was used to examine longitudinal patterns of BMI growth over a span of 14 years (4 years – 18 years). Data were assessed and the optimal number of classes to describe the growth trajectories was selected. Bivariate and multivariate data analyses were used to describe the children in each group. Multinomial logistic regression was used to examine whether the number of cumulative preschool (age 4) or school-aged ACEs (ages 4 to 14) predicted group membership.

**Results:** Overall, the percentage of overweight/obesity increased with each additional wave. Based on z-BMI score, at age 4, 20.1% were overweight/obese. A marked

increase was identified when children were 12 years old (42.0%) and then at 18 years (49.4%). Three BMI growth trajectories were identified: *expected growth*, *emerging overweight*, and *increasing obesity*. Most children followed an *expected growth* trajectory (73.6%). However, about a fifth followed a trajectory with a steep increase in BMI over time (*emerging overweight* = 21.9%) and a small percentage of the children exhibited a high initial BMI as well as a high rate of increase (*increasing obesity* = 4.5%). Ages 8 to 12 and ages 16 to 18 had especially steep slopes when it came to BMI increase in the *emerging overweight* and *increasing obesity* trajectories. A higher preschool ACEs score was associated with a low odds ratio of being in the *emerging overweight* group compared to the *expected growth* group; school aged ACEs score did not predict membership to a particular class. Female children and those with a higher primary maternal caregiver BMI when the children were 4 years old predicted being in the *emerging overweight* group compared to the *expected growth* group.

**Implications:** The time periods that are especially sensitive to steeper weight gain are likely to be the time periods when interventions should be targeted for children in a low income, urban, largely African American community. The current study had results that were divergent from the hypothesis in that children who had higher ACEs at age four were less likely to have an obesity-prone BMI trajectory. Reasons and implications are discussed. The child's gender and the child's maternal caregiver's weight status should provide some guidance in intervention and treatment decisions.

Assessing the Relationship between Adverse Childhood Experiences and Body Mass  
Index Trajectory of Children and Adolescents

by  
Hyeshin Park

Dissertation submitted to the Faculty of the Graduate School of the  
University of Maryland, Baltimore in partial fulfillment  
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I thank my dissertation chair Dr. Richard Barth for not only guiding through the dissertation process but also providing me with immense support and insight over the last several years. My dissertation committee members were also crucial to my academic and personal development. Dr. Donna Harrington advised me throughout the doctoral program, teaching me analytical and research techniques, proofreading, and providing reassurance. Dr. Edward Pecukonis helped me to be mindful of the theoretical premise; he also allowed me to grow in other areas of teaching and conducting research through shared work experience in the Maternal Child Health Program and Motivational Interviewing research. Dr. Howard Dubowitz provided seamless access to the LONGSCAN Baltimore dataset, informed me of the detailed nature of the LONGSCAN project, and shared his expertise in childhood obesity as well as ACEs. Dr. Maureen Black shared her copious knowledge in youth obesity trends and interventions in this select population; she helped me to consider various implications for practice and research. Finally, I thank my family and friends for their patience, support, and prayers.

### **LONGSCAN Acknowledgement**

This proposal includes data from the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN), Baltimore site. LONGSCAN was funded by the Administration on Children and Families, U.S. Department of Health and Human Services. None of this document's content should be interpreted to indicate the support or endorsement by the funders of the original study. The content herein solely reflects the opinion of the author. For use of LONGSCAN Baltimore data, I was added to the existing IRB protocol at University of Maryland, Baltimore, where Dr. Howard Dubowitz is listed as PI.

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## **Chapter 1. Introduction: Relationship and Impact of Adverse Childhood Experiences (ACEs) and Body Mass Index Trajectory (BMI) Among Children**

Adverse childhood experiences (ACEs) are stress-inducing, traumatic events in childhood (Centers for Disease Control and Prevention [CDC], 2016). ACEs have gained significant attention in the public health sector because researchers have found linkage between ACEs and negative physical and mental health outcomes (Chartier, Walker, Naimark, 2009; Larkin, Felitti, & Anda, 2014; Wade et al., 2016). Researchers in many disciplines, including public health, medicine, nursing, social services, and criminal justice have extensively studied the relationship between ACEs and health outcomes (Lynch, Waite, & Davey, 2013). Among the likely health consequences of ACEs is obesity in childhood and adolescence (Burke, Hellman, Scott, Weems, & Carrion, 2011; Lynch et al., 2016).

The rising attention to the relationship between ACEs and obesity is, in part, due to the widely established importance of reducing childhood, adolescent, and adult obesity. Systematic reviews have found that childhood and adolescent obesity are associated with negative psychological outcomes such as low self-esteem and behavioral problems; negative health outcomes, such as increased risk for cardiovascular diseases, type 1 diabetes, asthma, premature mortality (Reilly & Kelly, 2005; Karnik & Kanekar, 2012); and adverse long term socioeconomic outcomes (Reilly, Methven, McDowell, Hacking, Alexander, Stewart, & Kelnar, 2003). There are economic costs as well. A recent systematic review calculated the direct cost of overweight and obesity; Tsai, Williamson, and Glick (2011) estimated the national aggregate cost of overweight and obesity for 2008 was 113.9 billion and reported that the cost of overweight and obesity

varied from 5% to 10% of total U.S. health care spending depending on the study that was examined. Wang, Beydoun, Caballero, and Kumanyika (2008) estimated that the total health care costs attributable to obesity and overweight in the United States will double every decade, ultimately accounting for about one-sixth of the total U.S. health care costs by 2030. Additionally, Finkelstein, Trogdon, Lohel, and Dietz (2009) found that after adjusting for demographic, economic factors, and smoking status, individuals who were obese incurred 42% more direct medical costs. Similarly, Arterbur, Maciejewski, and Tsevat (2005) reported an overall per capita health care expenditure for morbidly obese adults was 81% greater than for normal weight adults; it was 65% greater for overweight adults than for normal weight adults.

Due to the detrimental influences of obesity, researchers have attempted to better identify the etiology of obesity. The three main categories of factors that contribute to obesity are genetic, behavioral, and environmental (Chaput, Pérusse, Després, Tremblay, & Bouchard, 2014). Drong, Lindgren, and McCarthy (2012) reviewed studies that examine the impact of genetics on BMI based on genetic loci and DNA sequence; they reported that the variance in BMI explained by hereditary could vary from 2% (Speliotes et al., 2010) to about 20% (Yang et al., 2012). Other studies have confirmed that there is a positive correlation between parental obesity and child obesity or child's risk for becoming obese (He, Ding, Fong, & Karlberg, 2000; Hui, Nelson, Yu, Li, & Fok, 2003; Whitaker, 2004; Li, Law, Lo Conte, & Power, 2009).

With regards to behavioral factors, diet and exercise affect obesity. Lower fruit and vegetable intake (Epstein, Gordy, Raynor, Beddome, Kilanowski, & Paluch, 2001; Ledoux, Hingle, & Baranowski, 2010), higher fat-intake (Hooper, Abdelhamid, Moore,

Douthwaite, Skeaff, & Summerbell, 2012), and higher sugar-sweetened beverage intake increase the risk of obesity (Keller & Della Torre, 2015). Because weight gain is a function of calories consumed and calories expended, sedentary lifestyle and less physical activity are also related to obesity (Carlson, Crespo, Sallis, Patterson & Elder, 2012).

There are many environmental factors that appear to correlate with obesity outcomes. Feng, Glass, Curriero, Stewart, and Schwartz (2010) systematically reviewed literature concerning the relationship between the built environment and obesity; built environment encompassing access to fast food chains, restaurants, grocery stores, convenience stores, and facilities that enable physical activity (e.g., gyms, parks, green spaces, playgrounds) impact obesity. Feng et al. (2010) reported that most studies on this have found significant correlations between the built environment and obesity .

Van der horst, Oenema, Ferreira, Wendel-Vos, Giskes, van Lenthe, and Brug (2007) conducted a systematic review of environmental factors that correlated with obesity-related dietary behaviors. The authors found that parental diet and parenting education regarding diet were consistently related to children's fat and vegetable and fruit intake (Van de horst et al., 2007). In another study, parenting style, specifically authoritative parenting, was associated with healthier eating patterns, more physical activity, and lower BMI scores compared to permissive, authoritarian, and neglectful parenting (Sleddens, Gerards, Thijs, De Vries, & Kremers, 2011). Because more educated parents are likely to employ authoritative parenting (Benson & Haith, 2010), parental level of education may have confounded the findings.

Though health researchers have identified numerous factors that contribute to obesity, they agree that there are multiple deficiencies in understanding the etiology of obesity (Reilly & Hughes, 2015). Furthermore, the impact of any particular factor on obesity risk is difficult to partition because the genetic, environmental, and behavioral components are intricately intertwined (Drong, Lindgren, & McCarthy, 2012). The interlace is described by behavioral scientists as that “any given environment may have different effects on individuals who differ genetically, and genetic differences among individuals may create differences in the environments to which individuals are exposed” (Johnson, 2007). Thus, health and social researchers continue to better clarify and assess contributors to obesity.

In that vein, scholars have attempted to further understand the relationship between retrospectively recalled ACEs, representing several environmental factors, and obesity outcomes. Moreno, Pigeot, and Ahrens (2011) reviewed the literature that examined the relationship between ACEs including early maltreatment, and the development of obesity. They preliminarily concluded that those with more ACEs were more prone to obesity. They did, however, acknowledge that some studies reported contradictory results (Moreno et al., 2011). Moreno and colleagues (2011) reported that obesity appeared to develop more often in adulthood for those who had been exposed to childhood adverse events and urged researchers to examine whether weight gain develops earlier, such as in childhood or in adolescence. Danese and Tan (2014) also conducted a systematic review and a meta-analysis to examine the relationship between childhood maltreatment, a component of ACEs, and obesity. They found that even though adults who were maltreated in their childhood faced a higher risk of developing obesity in

adulthood, the association was not statistically significant in studies that examined obesity in childhood or adolescence. Both authors noted a dearth of studies examining the relationship between ACEs and obesity in childhood and adolescence.

In addition, there are very few studies that examine the cumulative impact of having multiple ACEs on obesity outcomes. According to a cumulative risk theory perspective, there may be a dose effect (Burke et al, 2011; Rutter 1979; Sameroff, 2000). Timing of ACEs may also impact the outcome (Flaherty et al., 2013). The relationship between cumulative risk and childhood obesity, especially occurring over time, has not been studied as extensively as cumulative risk and adult obesity at a single time point (cf. Burke et al., 2011; Shin & Miller, 2012). Hence, examination of whether ACEs at one point in time as well as over a period of time would impact obesity rates of trajectory is necessary. In addition, charting obesity risk or BMI longitudinally is especially important for children and adolescents because their weights naturally fluctuate (Guo, Huang, Maynard, Demerath, Towne, Chumlea, & Siervogel, 2000). Therefore, studying the relationship between ACEs and body mass index over time addresses a gap in our knowledge of whether ACEs contribute to childhood obesity.

This chapter presents the definition and prevalence of ACEs. Second, I provide the definition of obesity. Next, the chapter explores rates of obesity among children and adolescents, especially among urban, low-income, minority populations. Fourth, the social, economic, and health implications of obesity, and fifth, the link between ACEs and obesity and the relevance of this relationship to social work are discussed. Lastly, the specific aims of the study are outlined. The second chapter reviews the research and theory relevant to the problem. The third chapter describes the methods that are used to

explore the BMI trajectories, characteristics of children each trajectory groups and the impact of ACEs on pathways. The fourth chapter presents the results of the study. The fifth chapter involves a discussion of these results as well as implications for practice and policy.

### **Definition of Adverse Childhood Experiences**

Adverse Childhood Experiences (ACEs) are described as stressful or traumatic events. Even though the terms trauma and ACEs are often interchangeably used, there are some differences. Trauma broadly refers to an experience that is emotionally extremely distressing or painful. Herman (1992) said that traumatic events are extraordinary events because “they overwhelm the ordinary human adaptations to life” and “evoke helplessness and terror” (p. 33). For trauma that occurs in childhood, Terr (2003) defines it as the psychological result of an external adversity, whether sudden and single or repeated, that stresses the child beyond the child’s coping and defensive mechanisms. Although traumatic events that occur at any point in time may have detrimental psychological and physical effects, those occurring in childhood deserve particular attention because these uncontrollable and terrifying experiences may have the most profound effects when the nervous system and cognitive function are undergoing development (Van der Kolk, 2003).

If trauma is said to encompass a broader definition, the term, “adverse childhood experiences (ACEs),” is a more specific operationalization of traumatic events. ACEs focus on traumatic experiences that occurs in childhood and the term is used in research as a way to create an index of adverse events to include maltreatment, such as abuse and

neglect, and household dysfunction items, such as parental conflict, incarceration of a household member, and domestic violence (Felitti et al., 1998; SAMHSA, 2014).

Finkelhor, Shattuck, Turner, and Hamby (2013) recognized that there are many adversities that can be categorized as ACEs and that the current state of research is unable to clearly identify the exact ones that should be included and that the selection would depend on the purpose of the research. Nonetheless, studies have consistently focused on 8 to 10 variables that parallel the original ACEs study, which was conducted in collaboration between Kaiser Permanente's Health Appraisal Center and the U.S. Centers for Disease Control and Prevention. The original ACEs study contained 10 categories of adverse experiences: (1) physical abuse; (2) psychological abuse; (3) sexual abuse; (4) physical neglect; (5) emotional neglect; (6) parental psychopathology; (7) caregiver substance abuse; (8) marital conflict; (9) mother treated violently; and (10) criminal behavior in the household/incarceration of a household member (Felitti, Anda, Nordenberg, Williamson, Spitz, Edwards, & Marks, 1998; CDC, 2014). The first five are regarding child maltreatment and the latter five, household dysfunction. Numerous researchers have replicated these ACEs variables and assessed their relationship to behavioral, social, emotional, and health outcomes (Palusci, 2013).

### **Prevalence of ACEs**

The prevalence of ACEs varies by population and research study. CDC and Kaiser Permanente conducted the original study in order to examine the health implications of adverse childhood experiences (Felitti et al., 1998). The data were collected between 1995 and 1997 (Felitti et al., 1998) and included a sample of 17,337 participants. At the time of data collection, the participants were on average, 56 years for

women (54%) and 58 for men (46%). Participants were mainly from middle class families, well-educated, and insured under the Kaiser Health Plan in San Diego, California. They retrospectively answered questions about the above 10 adverse childhood experiences occurring during the first 18 years of life. The researchers found that 26% had experienced one ACE, 16% had experienced two, 10% had experienced three, and 13% had experienced four or more ACEs (CDC, 2014).

Since the original CDC-funded study, various researchers have endeavored to add information about the normative ACEs levels in different populations or samples. In the 2009 ACEs module of the Behavior Risk Factor Surveillance System (BRFSS), administered by the CDC and state health departments, 26,229 noninstitutionalized adults residing in Arkansas, Louisiana, New Mexico, Tennessee, and Washington responded to 11 questions about 8 categories of ACEs (verbal abuse, physical abuse, sexual abuse, household mental illness, household substance use, domestic violence, parental separation/divorce, and incarcerated family member). The sample was between 18 years and 98 years with a mean age of 47.1 years, 64% female, and 75% White (CDC, 2010). Overall, 41% reported having no ACE; 59% of the respondents stated that they had at least one type of ACEs and 10% reported that they had five or more. The most common ACE was household substance abuse at 29%.

In 2010, the CDC added the ACEs module to the BRFSS study in 10 states and the District of Columbia. The ACEs were based on the same 11 questions that were asked in the 2009 BRFSS study. However, for the 2010 BRFSS study, the researchers separated household alcohol use and other drug use to create nine categories of ACEs. In a random sample from 10 states and DC of 53,998 participants, 41% reported no ACE,

44% reported 1 to 3 ACEs, 16% reported more than 4 ACEs (Gilbert et al., 2014).

Emotional abuse was the most prevalent type of ACE at 35%, followed by parental divorce/separation at 28% (Gilbert et al., 2014). In 2011, BRFSS included adult residents from Montana, Minnesota, Vermont, Washington and Wisconsin; 55.4% reported at least one ACE and consistent with the 2011 BRFSS study, 13.7% reported more than four ACEs (Campbell, Walker, & Egede, 2016).

The BRFSS studies are based on random digit dialing and thus the samples were representative of the population residing in the states that they examined. Random digit dialing was limited to landlines, thus those between the ages of 18 and 35 were underrepresented in the sample; the samples were weighted to address this issue. The BRFSS ACEs module suffers from similar issues as the original ACEs study. For both studies, the data collection relied on retrospective reports (CDC, 2010), which are susceptible to recall bias because either memory declines or the perception of one's past life experience changes with aging or problems and experiences in later life (McFarland, Ross, & Giltrow, 1992). Furthermore, the studies utilized cross-sectional designs. ACEs were more prevalent in the BRFSS study compared to the original CDC-ACEs study, most likely due to sample and methodological differences. Whereas the BRFSS study used a state representative sample, the original CDC-ACEs study used a middle-class sample with a higher average age.

The National Survey of Child and Adolescent Well-Being II (NSCAW II), a nationally representative longitudinal study that examined the well-being of 5,873 children (2 months to 17.5 years old) with substantiated or unsubstantiated abuse and neglect, included the ACEs variables in their study (Stambaugh, Ringeisen, Casanueva,

Tueller, Smith, & Dolan, 2013). Almost everyone had experienced at least one ACE (99%); 6% of the sample reported experiencing only one ACE, 19% reported two, 22% reported three, and 51% reported four or more ACEs. The higher prevalence of ACEs compared to the original ACEs study is not surprising because the sample for NSCAW II consisted of children involved with child welfare services. However, the authors of the study noted that these rates might be underestimated because the NSCAW children were young, implying that they still have years of childhood remaining, with possible exposure to future ACEs (Stambaugh et al., 2013).

Burke and colleagues (2011) examined the rates of ACEs in a low-income community pediatric clinic in California. The sample consisted of 54% females, 58% African American, 15% Hispanic, 13% Pacific Islander, 12% Other, and 3% White. Of 701 children and adolescents between the ages of 0 and 20.9 years who responded to nine ACEs categories, 33% had an ACE score of zero, 30% had one, 14% had two, 11% had three, and 12% had four or more. Wade et al. (2016) also assessed ACEs using the Philadelphia Adverse Childhood Experiences Survey (PHL ACE), which comprised 58% females, 45% White, 44% Black, and 30% who lived below the poverty level. Based on conventional, CDC driven ACEs categories, the study reported that 68% had experienced at least one ACE and 20% had experienced more than four ACEs. Both studies had a higher percentage of the sample reporting one or more ACEs (67% and 68% respectively) compared to the CDC-ACEs study (52%). Burke et al. (2011) reported that the difference in demographics of the sample (lower income vs. middle income) as well as the methodology of the study may have accounted for the differences in ACEs rates.

Thompson and colleagues (2015) used data from the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN) to examine the occurrence of ACEs. Data were collected from all five study sites and data collection occurred when the children were ages 4, 6, 8, 12, 14, and 16. The sample consisted of 802 participants; 56% were female, 26% were White, and 55% were African American (Thompson et al., 2015). The researchers identified three distinct developmental periods, early childhood (birth to age 6), later childhood (age 6 to age 12) and teenage years (age 12 to age 16) and assessed 8 types of ACEs (neglect, psychological maltreatment, physical abuse, sexual abuse, caregiver substance use, caregiver victimization, household criminal behavior, and caregiver depression (Thompson et al., 2015). On average, participants reported experiencing an average of 1.9 ACEs in early childhood, 1.5 in later childhood, 1.2 in teenage years, and 3.2 in their lifetime (some were experienced in more than one period). Compared to the original CDC-ACEs study, the LONGSCAN participants experienced more early adversities, even though the original CDC-ACEs study had a slightly wider time frame of the first 18 years for examination of ACEs. This discrepancy is most likely due to the fact that LONGSCAN participants were a prospective sample and included families at high risk for being referred to child welfare (Thompson et al., 2015).

In sum, ACEs scores vary depending on the characteristics of the sample and the study methodology. About two thirds experienced at least one ACEs in the BRFSS study and the CDC-ACEs study; on the other hand, studies employing higher risk samples report that virtually everyone had experienced at least one ACE (Stambaugh, Ringeisen, Casanueva, Tueller, Smith, & Dolan, 2013). Because ACEs scores vary by population, examining rates of ACEs in additional communities is important.

## **Prevalence and Definition of Childhood and Adolescent Obesity**

ACEs may perhaps increase the risk of childhood and adolescent obesity (Burke, Hellman, Scott, Weems, & Carrion, 2011). The terms obese and overweight are specific terms based on the person's body mass index (BMI), a function of weight and height ( $\text{kg}/\text{m}^2$ ). For adults, overweight is defined as a BMI between 25.0 and 29.9 and obesity is defined as BMI over 30 (CDC, 2015). For children, "overweight is defined as a BMI at or above the 85<sup>th</sup> percentile and below the 95<sup>th</sup> percentile for children and teens of the same age and sex. Obesity is defined as a BMI at or above the 95<sup>th</sup> percentile for children and teens of the same age and sex" (CDC, 2015). The percentiles were set based on children's BMI data from U.S. national surveys that were conducted from 1963-65 to 1988-94 (Kuczmarski et al., 2002). Although not a perfect measure, the BMI is the most widely used measure of obesity (Schneider & Brill, 2005; Ortega, Sui, Lavie, & Blair, 2016).

Approximately 17% of American children and adolescents between the ages of 2 and 19 are obese and an additional 15% are overweight (CDC, 2013). More concerning is the fact that the prevalence of obese children has more than doubled and that of obese adolescents has quadrupled in the past 30 years (Ogden, Carroll, Kit, & Flegal, 2014). This upward trend has leveled off and obesity rates have remained stable in the past decade, as evidenced by obesity prevalence rates from the National Health and Nutrition Examination Survey (NHANES), which was conducted from 2003 to 2012 in 2-year intervals (Ogden, Carroll, Kit, & Flegal, 2014; Xi et al., 2014). Nonetheless, the percentage of overweight obese children remains a major problem.

In the U.S., racial and ethnic disparities characterize obesity rates. African American, Hispanic, and Native American children exhibit the highest obesity rates among racial and ethnic groups (Ogden et al., 2006). (This review will focus on African American children because 95% of the sample in this study is African American.) In the most recent NHANES study that included BMI data from 2011 to 2012, 20% of Non-Hispanic Black and 22% of Hispanic children between the ages of 2 and 19 were obese and 35% of Non-Hispanic Black and 39% of Hispanic children were overweight or obese (Ogden, Carroll, Kit, & Flegal, 2014). The obesity rates were significantly higher when compared to the rates of non-Hispanic Asian and White children. For children between the ages of 2 and 5, a Non-Hispanic Black child is almost three times as likely to be obese (11%) as a White child that age (4%) (Ogden et al., 2014).

Studies have identified several possible explanations for the high prevalence of obesity among African American (and Hispanic) children. Lower income status plays a role in increased risk for obesity (Wang & Zhang, 2006; Wen, Gillman, Rifas-Shiman, Sherry, Kleinman, & Taveras, 2012). Additionally, low-income, urban neighborhoods where African Americans often reside tend to lack access to healthy foods (Morland, Wing, & Diez Roux, 2002; Morland, Wing, Diez Roux, & Poole, 2002; Powell, Auld, Chaloupka, O'Malley, & Johnston, 2007) and have few opportunities for physical activity (Powell, Slater, & Chaloupka, 2004). There are also early risk factors for obesity African American children are more inclined to experience. For example, African American children are more likely to have mothers with depression, to have been introduced to solid foods early, to have televisions in their rooms, and to have higher intake of sugar

sweetened beverages and fast foods compared to White children (Taveras, Gillman, Kleinman, Rich-Edwards, & Rifas-Shiman, 2010).

### **Impact of Childhood Obesity**

Childhood obesity has physical and psychological health implications. Reilly et al. (2003) conducted a systematic review to investigate the long-term health consequences of childhood obesity. They found that obese children are significantly more likely to have cardiovascular problems, asthma, type 1 diabetes, and obesity that persists through adulthood (Reilly et al., 2003). Moreover, they reported that some research indicated that obese children were prone to other physical conditions, such as low-grade systemic inflammation and increased serum C reactive protein (Reilly et al., 2003). Other conditions children with obesity are at risk include respiratory problems such as obstructive sleep apnea, Pickwickian syndrome, chronic snoring, and asthma; orthopedic problems such as Blount's disease and slipped capital femoral epiphysis; gastrointestinal problems such as gall bladder disease and steatohepatitis; cardiovascular problems such as dyslipidemias and hypertension; and endocrinologic problems such as insulin resistance, hyperinsulinism, impaired glucose tolerance, type 2 diabetes, polycystic ovarian syndrome, and menstrual irregularity (Schneider & Brill, 2005).

There are psychosocial concerns as well. Psychologically, obese children have a higher likelihood to have lower self-esteem and behavioral problems than non-obese children (Reilly et al., 2003). Janicke et al. (2007) found that in a sample of 96 children at risk for overweight, 75% had endured some type of peer victimization, and peer victimization was moderately associated with child depressive symptoms. Reilly et al. (2003) reported that studies have found associations between obesity in

adolescence/young adulthood and lower income level and educational attainment in young adulthood. Another long term consequence of childhood obesity may be that overweight children grow up to be overweight adults and have overweight children, due to genetic factors as well as dietary and lifestyle habits (Semmler, Ashcroft, van Jaarsveld, Carnell, & Wardle, 2009). For current and future physical and psychosocial health, childhood obesity needs to be addressed.

### **Relationship between ACEs and Childhood Obesity**

In the 1980s, Dr. Felitti ran a weight management clinic and noticed that patients in his clinic were disproportionately reporting maltreatment history from their childhood (Felitti & Anda, 2010; Steven, 2012; Williamson, Thompson, Anda, Dietz, & Felitti, 2002). Felitti then collaborated with Dr. Anda, an epidemiologist who was examining the relationship between behaviors and diseases, to assess whether ACEs would pose a lifelong impact on a person's health (Felitti & Anda, 2010). They developed 10 items that would define ACEs and spearheaded the original CDC-ACEs study. Since then, a number of studies have examined the link between ACEs and risk for obesity.

Several retrospective studies have confirmed the association between self-reported childhood maltreatment and adult obesity (Aaron & Hughes, 2007; Chartier, Walker, & Naimark, 2009; Dube, Cook, & Edwards, 2010; Felitti et al., 1998; Midei, Matthews, & Bromberger, 2010; Noll, Zeller, Trickett & Putnam, 2007; Williamson, Thompson, Anda, Dietz, & Felitti, 2002), yet the research is sparse when it comes to the relationship between childhood maltreatment and childhood obesity (Schneiderman, Smith, Arnold-Clark, Fuentes, Duan, & Palinkas, 2013). Furthermore, the relationship between the number of ACEs and risk for obesity is mixed (Burke et al., 2011;

Schneiderman et al., 2013). More investigation is necessary to clarify the relationship between number of ACEs and obesity among children, especially whilst measuring ACEs prospectively.

### **Aims and Objectives of the Dissertation**

The purpose of this study was to examine the BMI trajectories of children raised in urban, Baltimore neighborhoods and to assess whether the number of ACEs predicts high risk or low risk BMI trajectories. Ultimately, the findings of the study would help identify who should get further assessment, prevention, and intervention priority, thus guiding practitioner and public health actions. The study used data from the LONGSCAN Eastern (Baltimore, Maryland) site; the sample was a high-risk sample from families residing in a lower income, mainly African American, urban neighborhood. The longitudinal data were collected from 1991 to 2013 and the age of the children ranged from 4 to 18. The data were analyzed using latent class growth analysis (LCGA), a method used by other scholars to characterize BMI growth trajectories (Berlin, Parra, & Williams, 2014; Fuemmeler, Yang, Costanzo, Hoyle, Siegler, Williams, & Østbye, 2012; Hoekstra, Barbosa-Leiker, Koppes, & Twisk, 2011; Li, Goran, Kaur, Nollen, & Ahluwalia, 2007; Østbye, Malhotra, & Landerman, 2011). LCGA allows the exploration of distinct BMI trajectories of the children without imposing classification of the sample into pre-defined groups prior to the analysis (see Methods in Chapter 3 for more information). Once the distinct pathways were identified, the independent variables of trajectories were examined for their predictive ability. The specific aims of the study are stated below.

**Aim 1: Conduct a latent class growth analysis to identify trajectories of BMI for the LONGSCAN Baltimore participants.** The first aim of the study was to identify the number and shape of distinct BMI trajectories that characterize children over a 14-year period.

**Aim 2: Identify the risk factors, specifically the role of preschool ACEs (occurring at age 4) and school-aged ACEs (occurring between the ages of 4 and 14), which predict group memberships for different BMI trajectories.** Upon identifying the trajectory groups, multinomial logistic regression analyses were conducted to determine whether the number of ACEs in early childhood (at age 4, ‘preschool’) or through early adolescence (age 4 to 14, ‘school-aged’) predict membership in the trajectory groups. Other risk factors and important demographic variables were also included as covariates in the models.

## **Chapter 2:**

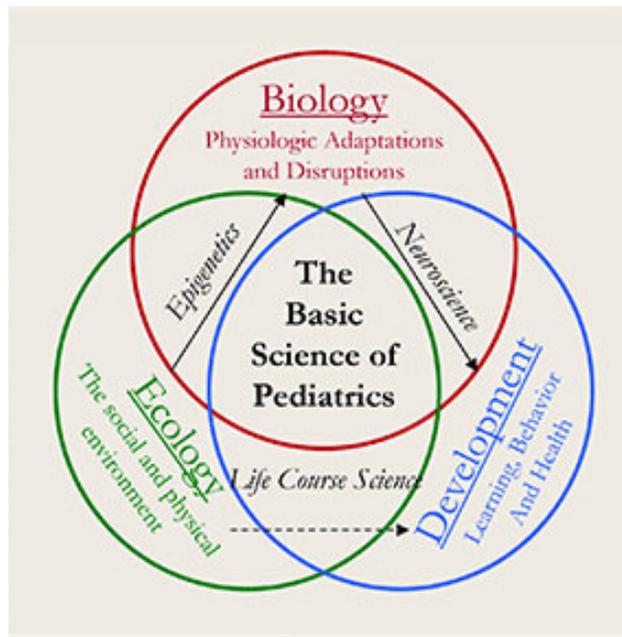
### **Theoretical Background and Literature Review**

The scholarship on ACEs and maltreatment has employed various theories to examine their causes as well as their impact on health and psychosocial outcomes. Some of the theories include the life course perspective, the biopsychosocial perspective, ecological theory, and the stress and coping framework (Anda et al., 2006; Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Larkin, Felitti, & Anda, 2014). This chapter provides an overview of the ecobiodevelopmental (EBD) framework (Shonkoff et al., 2012), cumulative risk theory (Sameroff, 2000), and stress and coping theory (Lazarus & Folkman, 1984) to guide the study. Potential pathways between ACEs and obesity are described based on these theories.

#### **Ecobiodevelopmental Theory**

Shonkoff and colleagues (2012) presented the EBD framework to urge pediatricians to think of disparities in health, learning, and behavior as stemming from an ongoing interaction between toxic stress, early adversities, brain architecture, and physical and mental health development. Figure 1 illustrates the ecobiodevelopmental (EBD) model of human health and disease; the framework purports that ecology or environmental factors affect biological factors and that the two together drive development across the lifespan (Shonkoff et al., 2012).

**Figure 1.** Ecobiodevelopmental Framework of Human Health and Disease (Shonkoff et al., 2012)



EBD builds on the ecological model and the biodevelopmental framework. The ecological model states multiple levels of social systems (including the microsystem, mesosystem, exosystem, and macrosystem) surround a child, and that these environmental factors influence the development of the child (Bronfenbrenner, 1979). The biodevelopmental framework proposes that early life experiences and interactions impact psychological, neurological, and physical development (Shonkoff, 2010).

Within the EBD, there is a biological element and it considers the impact of socioenvironmental factors on the physical aspects of a person. For example, when a child is exposed to ACEs, the body may activate the body’s stress response system for an extended time period, especially in the absence of a buffering mechanism such as a supportive relationship with a caregiver (Shonkoff et al., 2012). It should also be noted

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<sup>1</sup> Figure 1 was included with author’s permission.

that the child is also a participant in the process, absorbing as well as resisting biological, environmental, and developmental influences.

Stress in early childhood can have deleterious effects in terms of neurological development (Middlebrooks & Audage, 2008). Early childhood is an especially critical time period because the brain undergoes accelerated development between the prenatal period and the first few years of life (National Scientific Council on the Developing Child, 2012). The number of synapses reaches a life-time peak and the brain reaches two thirds of its adult size at three years of age (Bruer, 2009). Shonkoff and Phillips (2000) also stated that birth to five years of age is a critical period for brain and language development, as well as emotional, social, and regulatory advancements.

Hence, traumatic events occurring in early childhood, which the current study defines as the first four years of life, can have deleterious effects on the child's brain development (Middlebrooks & Audage, 2008; Shonkoff et al., 2012). Researchers who study the timing of ACEs and its impact on various developmental outcomes agree that adversities in early childhood years may herald more harmful effects than those in later years because unfavorable coping mechanisms and self-regulation methods developed in early childhood can impact numerous subsequent relationships (Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Sroufe, Carlson, Levy, & Egeland, 1999). Children learn to self-regulate and develop coping mechanisms via responsive interaction with primary caregivers and childhood maltreatment can impede this process (Benedetti, 2012).

According to the EBD, a child that experiences trauma from family or community interactions may face developmental challenges, both psychologically and physically. This can lead to obesity in the sense that external adversities such as childhood

maltreatment and household dysfunction occurring in early childhood can directly impact eating behaviors; the child may be subject to diminished parental regulation with regards to nutrition and physical activity. Moreover, ACEs can indirectly put children at risk for obesity by limiting the child's ability to cope with stress in a healthy manner, which can potentially encourage overeating in the future. Accordingly, the biological and developmental aspects are intertwined with environmental factors.

### **Cumulative Risk Theory**

Cumulative risk theory purports that the greater the accumulation of risk factors, the worse the outcome, regardless of the presence or absence of particular risk factors (Rutter, 1979; Sameroff, 2000). Rutter (1979) conducted an epidemiological study called the Isle of Wight study in England. Using a sample of children between the ages of 9 and 11, he found that the children who had two of the six risk factors (severe marital discord, low SES status, large family size, paternal criminality, maternal mental disorder, and foster placement) were four times more likely to have a mental disorder. Furthermore, children who had four risk factors were 10 times more likely to have a mental disorder compared to those who had none (Rutter, 1979). In a similar vein, Sameroff (2000) found in the Rochester Longitudinal Study (RLS) that having a larger number of risk factors (history of maternal mental disorder, high maternal anxiety, rigid parental attitudes/beliefs/values about child development, observations of few positive child-parent interactions, unskilled occupational status, low maternal educational status, disadvantaged minority status, single parenthood, stressful life events, and large family size) was associated with a higher chance of having negative psychological and academic outcomes. Overall, children who had more than 8 of the 10 risk factors were almost

seven times more likely to have negative academic outcomes compared to those with fewer than three risk factors (Sameroff, Bartko Baldwin, Baldwin, & Seifer, 1998).

Though both researchers support the cumulative risk theory, they have different theories about the relationship between the number of risk factors and the outcome. Rutter's (1979) cumulative risk theory argues that there is some threshold where with an increase in the number of risk factors, the level of maladaptive outcome increases in a quadratic fashion. On the contrary, Sameroff et al. (1998) contends that there is a linear relationship: as the number of risk factors increases, so does the chance of negative outcome. In either case, cumulative risk theory posits that the higher the number of risk factors the worse the outcome.

Cumulative risk theory has limitations. The variable "cumulative risk" is constructed by first dichotomizing each risk factor (1 = *exposure*; 0 = *no exposure*), and then summing them to generate a total score. Evans, Li, and Whipple (2013) reviewed the literature on cumulative risk and noted several weaknesses. The authors recognized that because risk factors are dichotomized, information on intensity, duration, and frequency of the exposure are lost (Evans et al., 2013). In the process of dichotomization, risk designation is often done arbitrarily, such as based on statistical distribution (e.g., upper quartile of exposure is considered a "risk"). Furthermore, the model is additive, and thus, the researcher is constrained in probing moderating variables for each particular risk factor (Evans et al., 2013).

Nonetheless, cumulative risk has advantages. Previous studies on early childhood adversities have found that the various types of ACEs are highly interrelated and frequently co-occur (Dong, Anda, Dube, Giles, & Felitti, 2003; Finkelhor, Shattuck,

Turner, & Hamby, 2012). Children who experience abuse or neglect in the home also often suffer from household dysfunction such as parental drug and alcohol use, parental mental illness, parental incarceration, and domestic violence (Anda, Croft, Felitti, Nordenberg, Giles, Williamson, & Giovino, 1999; Dong et al., 2003, Dube, Anda, Felitti, Edwards, & Croft, 2002; Dube et al., 2003, Dube et al., 2004; Felitti et al., 1998). In fact, Dong et al. (2004) reported that an individual who had experienced a single type of ACEs has a 75% likelihood of experiencing a second type of adversity. Thus, Larkin, Felitti, and Anda (2014) as well as Evans, Li, and Whipple (2013) contend that examining the influence of single ACEs variables can be misleading and that cumulative value of ACEs should be considered in order to better comprehend the impact of ACEs on outcomes.

Not only is the cumulative risk contextually intuitive but also statistically advantageous due to its parsimony. Measurement error is reduced (Ghiselli, Campbell, & Zedeck, 1981; Nunnally, 1978) and validity is enhanced (Brinberg & Kidder, 1982; Ghiselli et al., 1981). Using a single cumulative measure is useful in model building because it allows the model to converge more easily and produce stable estimates; additionally, a cumulative measure can allow for elevated statistical power compared to when multiple dichotomous variables are included in the model (Cohen, Cohen, West, & Aiken, 2003; Myers & Wells, 2003).

Cumulative risk theory recognizes the co-occurring nature of early adversities and how individuals with multiple risk factors are more likely to have impaired development (Sameroff, Seifer, Barocas, Zax, & Greenspan, 1987). Cumulative risk theory, in conjunction with the EBD framework (Shonkoff et al., 2012), supports the current study

aims and hypotheses that the children who have experienced a higher number of ACEs in early childhood are more likely to belong to a BMI trajectory that is prone to obesity.

### **Stress and Coping Theory**

Studies have demonstrated that the stress generated from early adversities can negatively influence the development of the hypothalamic-pituitary-adrenal (HPA) axis (Kalinichev et al., 2002; Liu et al., 2000). The HPA axis is a critical part of the neuroendocrine system that controls and regulates a person's response to stress. Danese and McEwen (2012) reviewed studies that examine the relationship between ACEs and allostatis (processes that attempt to maintain stability when changes occur), allostatic load, and age-related diseases. They cautiously concluded that ACEs occurring in sensitive developmental windows could chronically activate the allostatic system, thus "wearing and tearing" the system, resulting in antagonistic long-term health outcomes (Danese & McEwen, 2012).

When faced with stress, the body responds to reach homeostasis or to return to the normal state (Stephens & Wand, 2012). Children and adults use coping strategies to reach homeostasis. There are two types of coping, which are problem focused coping and emotion focused coping. Problem focused coping is a higher level of cognitive process, where the person attempts to master an aspect of the person or the environment, or to resolve a stressful relationship between the self and the environment (Lazarus & Folkman, 1984). Emotion focused coping involves palliating negative emotions that are brought about from stress (Lazarus & Folkman, 1984).

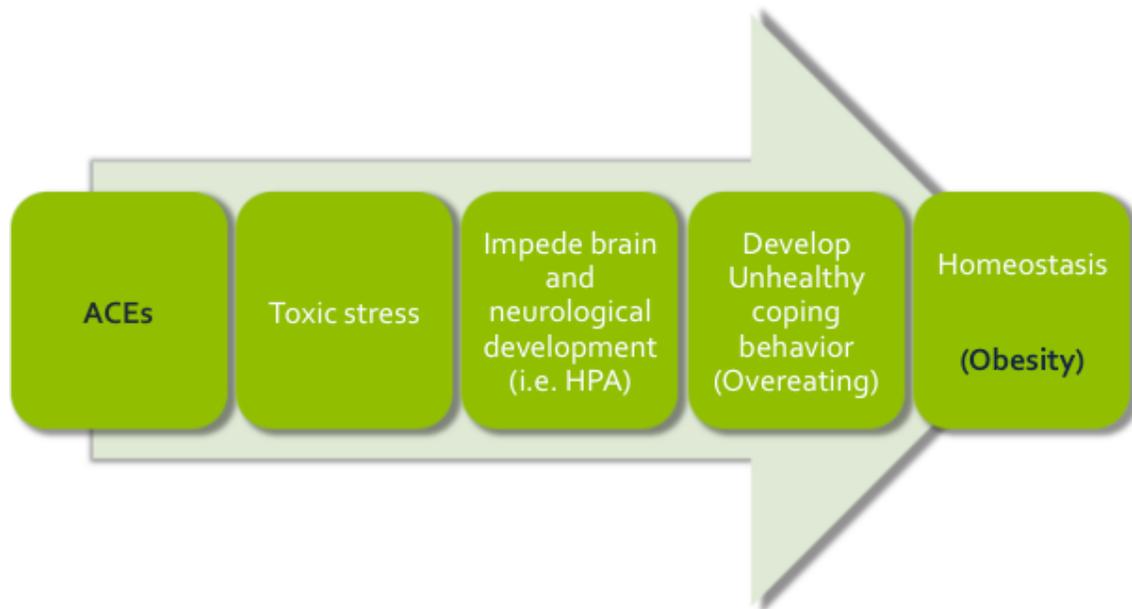
Based on cognitive and behavioral developmental stage, young children aged birth to five are unlikely to have the ability to assess their adverse circumstances or to

employ elaborate coping processes. Young children's stress response pathways are disrupted because they do not have the developmental ability to interpret and cope nor perhaps a caregiver who can guide them to cope; therefore, it is unsurprising that children with trauma commonly display negative affect and have inappropriate responses to situations (Chu & Lieberman, 2010). Their coping mechanisms may be limited to self-soothing behaviors, such as homeostatic eating and stress-related overeating, which contribute to excessive weight gain (Epel, Tojima, & Dallman, 2012).

Another potential pathway is that children exposed to ACEs may develop PTSD or depression, which may also lead to overeating. Several studies have confirmed the correlation between depression and overweight/obesity (Grundy, Cotterchio, Kirsh, & Kreiger, 2014; Heo, Pietrobelli, Fontaine, Stunkard, Faith, & Allison, 2003; Simon et al., 2008). Stunkard, Faith, and Allison (2003) reviewed articles on depression and obesity and reported that PTSD or depression may be a mediator/moderator for children with ACEs to develop obesity.

Figure 2 illustrates the theoretical pathway. It should be noted that this pathway is speculative and that the current study will not be testing the pathway from ACEs to obesity. However, the possible pathways may be helpful in explaining significant or nonsignificant results of the study as well as devising interventions that may reduce the risk of obesity among children with ACEs.

**Figure 2.** Theoretical pathway from ACEs to obesity.



### **BMI as Measure of Obesity and Importance of BMI Trajectory in Children**

There are several approaches to measurement of obesity. The most accurate measure to determine total body fat (TBF) and percent body fat (PBF) is done by dual energy x-ray absorptiometry. Brambilla, Bedogni, Heo, and Pietrobelli (2013) found that waist circumference-to-height ratio better assesses obesity compared to BMI or waist circumference. Compared to the more sophisticated measurements, BMI is imperfect; it is unable to disentangle fat and fat-free tissues and cannot take into account body fat distribution (Brambilla et al., 2013). Nonetheless, BMI is the most widely used tool to determine obesity, and it has been used extensively in scientific studies as well as clinical practice (Okorodudu et al., 2010).

Multiple studies have found that BMI is a valid measure in terms of detecting total body fat (TBF) and percent of body weight as fat (PBF) in children and adolescents when compared to other body-composition screening methods such as dual energy x-ray absorptiometry (Cole, Bellizzi, Flegal, & Dietz, 2000; Gallagher, Visser, Sepúlveda, Pierson, Harris, & Heymsfield, 1996; Lindsay, Hanson, Roumain, Ravussin, Knowler, & Tataranni, 2001; Mei, Grummer-Strawn, Pietrobelli, Goulding, Goran, & Deitz, 2001; Pietrobelli, Faith, Allison, Gallagher, Chiumello, & Heymsfield, 1998). BMI is also comparable to skinfold thickness in its ability to identify excess body fatness (Freedman, Ogden, Blanck, Borrud, & Dietz, 2013). Additionally, BMI is convenient and easy to administer, making it a common and appropriate tool to measure obesity.

Because of the changes that the child experiences via growth and hormonal changes, researchers recommend plotting BMI over time to track the shape of the BMI growth curve and predict obesity patterns (Black et al., 2010; Hillman, Corathers, & Wilson, 2009; Johnson et al., 2012). Measuring BMI at multiple time points and interpreting growth over time reduces the impact of measurement error; the sources of error include normal day-to-day variation and seasonal fluctuation (Himes, 2009). Overall, BMI increases rapidly during the first 9 to 12 months and then declines reaching a minimum at 4 to 6 years of age before beginning a gradual increase throughout adolescence and most of adulthood.

There are several variations of BMI that can be plotted: raw BMI, BMI %, BMI z-scores and centiles for sex and age (Cole, Faith, Pietrobelli, & Heo, 2005). Raw BMI is calculated using the mass and height of an individual  $(\text{kg})/(\text{m})^2$ , and is commonly used to investigate BMI growth trends (Danner, 2008; Mitchell, Pate, Beets, & Nader, 2013; Shin

& Miller, 2012). Some contend that raw BMI is the most appropriate for tracking obesity over time (Berkley & Colditz, 2007). Both z-scores and percentiles are also frequently used (Flegal & Ogden, 2011). BMI z-scores and centiles for sex and age are derived against a reference chart such as the 2000 CDC Growth Charts (Kuczmarski et al., 2000). Instead of defining weight status (obese, overweight, normal weight, or underweight) by specific BMI cut-offs, it is defined by charting the child's BMI on the growth chart, which takes into consideration that the healthy range of BMI changes by age and is different by gender (Lindsay et al., 2001; Kuczmarski et al., 2000; Pietrobelli et al., 1998; Reilly 2006;). BMI z scores is the "BMI of a child transformed into a scale comprising the number of SD units it is away from the mean of the referent population of the same age and gender" (Himes, 2009). BMI z scores indicate the severity of obesity that exceeds the BMI percentile chart (Himes, 2009). Lastly BMI % is the percentage difference from the median BMI [ $100 \log_e (\text{BMI}/\text{median BMI})$ ] (Cole, 2000). Though BMI % is not frequently used, Cole and colleagues (2005) found that there is benefit to using it because it captures the variability among overweight children.

Both the raw BMI and z-scores are used readily in research as well as clinical practice (Johnson et al., 2012); however, the current study uses the raw BMI score to assess the child's BMI status over time. Raw BMI score, rather than the standardized score, is recommended for the group based developmental trajectory approach because of its interpretability, sensitivity to change over time, and production of more valid effect estimates (Berkley & Colditz, 2007). Transformed scores have reduced variability, where one unit change can represent very different changes in BMI (Magee, Caputi, & Iverson, 2013). Using BMI z-scores longitudinally provides a change in an individual's

relative position within a population but does not allow the identification of distinct trajectory groups within a parsimonious model (Cole, Faith, Pietrobelli, & Heo, 2003). Furthermore, depending on the reference growth charts used, there may be differences in the interpretation of BMI growth data. Yasin and Filler (2013) investigated the differences between the two main growth charts, the WHO growth chart and the NHANES/CDC growth chart, and found that depending on the reference used to convert to z scores, there were discrepancies in the proportion of those labeled as obese. Specifically, almost twice as many individuals were categorized as obese, with a z score of 1.96 or higher, when WHO growth chart was used than when CDC growth chart was used as a reference (Yasin & Filler, 2013).

Researchers have found several different types of BMI growth trajectories. A table summarizing the literature is also presented (See Appendix A.1). The number of trajectories ranges from three to five. Three appears to be the most commonly occurring number of trajectories. Lane, Bluestone, and Burke (2013) used data from the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD) to examine longitudinal changes in BMI percentile in children from birth to age 11. The sample was drawn from 10 different regions in the U.S. and via conditional random sampling, 1,364 were selected to participate in the study. About a quarter of the sample consisted of minorities (13% African American, 6% Hispanic, 2% Asian/Native American, 3% Other) and 11% of the mothers had less than a high school education (average 14.4 years of education). Lane, Bluestone, and Burke (2013) conducted a latent class growth analysis and identified three trajectories of BMI percentile growth of 'elevated,' 'steady increase,' and 'stable.' The 'elevated' group

(25%) had an average BMI percentile of 58% at 24 months, and it increased through the preschool years until kindergarten, when the percentile leveled off at just above the 70<sup>th</sup> percentile. The slope was positive but non-significant ( $B = .10, p = .20$ ). BMI percentile in the ‘steady increase’ group (37%) increased from 36 months to age 6, when the average percentile leveled off near the 70<sup>th</sup> percentile; the average slope was positive ( $B = .14, p = .007$ ). Average BMI percentile remained stable over time at around 50% (non-significant slope) for the ‘stable’ group (39%).

Balistreri and Van Hook (2011) used the Early Childhood Longitudinal Study-Kindergarten Cohort (ECLS-K), a nationally representative sample of US kindergartners, to identify three distinct patterns of weight gain from kindergarten through eighth grade. They identified three trajectories: “gradual onset of overweight” (17.4%), “always overweight” (25.9%), and “normal” (56.7%).

Pryor et al. (2011) studied BMI changes in children from Quebec, Canada from when they were 5 months to 8 years old. The majority of the sample was white. Three trajectories were identified: low-stable (54.5%), moderate (41.0%), and high-rising (4.5%). The high-rising group was characterized by an increasing average BMI, which exceeded international cutoff values for obesity by age 8 years. A subsequent study using the same dataset was conducted to examine probabilities of overweight between 6 and 12 years (Pryor et al., 2015). Pryor and colleagues (2015) reported a different distribution of three classification of trajectories: “early-onset overweight” (11.0%), “late-onset overweight” (16.6%), and “never overweight” (72.5%).

Li, Goran, Kaur, Nollen, and Ahluwalia (2007) examined BMI changes among 1739 children from the National Longitudinal Survey of Youth 1979 (NLSY79), Child

and Young Adult file, which is a nationally representative longitudinal cohort study of children. They followed children's weights biannually from 2 years till 12 years and identified three trajectories: "early onset overweight" (10.9%), "late onset overweight" (5.2%), and "never overweight" (83.9%). The percentage of 'never overweight' was quite high and the author surmises that perhaps it is due to when the survey was conducted; obesity rates in youth started to increase in the 80s but the steeper slope came in the late 90s spiking in the 2000s (CDC, 2011).

Garden, Marks, Simpson, and Webb (2012) identified another set of classes of BMI trajectories from birth to 11.5 years using 370 children participants from the Childhood Asthma Prevention Study (CAPS) in Sydney Australia. The children were at risk for asthma at birth because one or more parents or siblings had current asthma or wheezing. Latent class growth analysis was used for boys and girls separately. The authors charted three classes for boys, which were 'normal,' 'early and persistent increase,' and 'late increase' trajectories. The 'normal' group (61%) tracked along the 50<sup>th</sup> percentile, the 'early and late increase in BMI' group (12%) started at 75<sup>th</sup> percentile at 2 years and continued to increase until moving above the 95<sup>th</sup> percentile at 11.5 years, the 'late increase in BMI' group (27%) were at 50<sup>th</sup> percentile from birth to 5 years but increased to the 85<sup>th</sup> by 8 years and the 90<sup>th</sup> percentile at 11.5 years. Girls displayed a similar but slightly different trend. Sixty two percent belonged in the 'normal' group. Twelve percent belonged in the 'early and persistently high BMI' group, where their BMI percentile increased to the 95<sup>th</sup> percentile at 3 years and subsequently reduced to be at the 85<sup>th</sup> percentile at 11.5 years. Finally, 26% belonged in the 'late increase in BMI':

these children charted 50th percentile from birth to 2 years, at which time the BMI increased to the 85th percentile at 8 years and the 95th percentile at 11.5 years.

Nonnemaker, Morgan-Lopez, Pais, and Finkelstein (2012) examined BMI trajectory patterns among 12 to 17 year olds from the 1997 National Longitudinal Survey of Youth (NLSY97). The survey used a (1) nationally representative sample of U.S. residents born between 1980 and 1984 ( $n=6,784$ ) and (2) an oversampling Hispanic, Latino, and African-Americans ( $n = 2,236$ ) (Nonnemaker et al., 2012). The authors identified four trajectories: (1) “high risk” for obesity, which consisted of children who were obese/overweight at age 12 and 72% with a BMI  $> 40$  by 23 years of age; (2) “moderate-to-high risk” for obesity, which consisted of children who were at risk of being obesity at age 12 and had been obese at some age (94%) but unlikely to have had a BMI  $\geq 40$  (4.4%); (3) “low-to-moderate risk” group had 8% of children who were obese at 12 years, 27% obese at 23 years, but their BMIs never went past 40 during the assessment period; (4) “low-risk” group had about 11% who were obese at some point over the age range but few who were obese at age 12 or 23. It should be noted that the authors’ definition of high BMI of 40 and above was higher than the CDC cut off of 30.

Stuart and Panico (2016) found four trajectories in a nationally representative sample of children at ages 3, 5, 7, and 11. The study was conducted in the United Kingdom and the sample included 9,699 children born in 2000-2002. A total of four trajectories were identified, two of which reflected that the children’s BMI remained in the normal range (low and mid normal, 85%), an overweight (14.4%) and an obese trajectory (3.1%).

Chen et al. (2016) analyzed data from Southeast Texas elementary school system, which included 45 elementary schools and 1,651 students. Data were collected biannually from kindergarten to the beginning of 5<sup>th</sup> grade. Half of the children were boys, 31.6% were non-Hispanic White, 22.7% were non-Hispanic Black, and 23.0% were Hispanic. The authors identified five different BMI trajectories when they categorized the children by normal vs. overweight/obese (BMI percentile above 85%): “persistently non-overweight/obese weight” (51.1 %), “early-onset overweight/obese” (9.2 %), “late-onset overweight/obese” (9.7 %), “becoming healthy weight” (8.2 %), and “chronically overweight/obese” (21.8 %). They also did another analysis using a categorical variable separating obese children (above 95<sup>th</sup> percentile) against those who were not and found three trajectories (persistently non-obese: 74.1 %, becoming obese: 12.8 %, and chronically obese: 13.2 %).

Apart from one study (Nonnemaker et al., 2012), which examined BMI changes from 12 to 17 years, the remaining studies examined growth in early and late childhood (birth to 12 years). No study has examined group trajectories occurring from early childhood through adolescence.

With regards to trajectory groups, the number varied from three to five trajectory groups. Generally, the largest percentage of children belonged to the normal or expected growth groups. Initial BMI at time of measurement (e.g. Lane, Bluestone, & Burke, 2012), timing of onset of overweight/obesity (e.g. Garden, Marks, Simpson, & Webb, 2012) or level of risk of overweight/obesity (e.g. Li et al., 2007) differentiated other growth trajectory groups. All studies used a type of growth mixture modeling.

Understanding patterns of growth through tracking BMI in children is crucial in identification and prevention of overweightness and obesity in children (Voelker, 2007). However, in practice many pediatricians do not track BMI (Voelker, 2007) and in ACEs research, perhaps because of attrition rates in longitudinal studies, there is a limited number of studies that actually examine the longitudinal changes in BMI in relation to trauma (Shin & Miller, 2012). To date, there is no prospective evidence that supports the link between ACEs and subsequent development of obesity in children.

### **Adverse Childhood Experiences and Obesity**

A literature review was conducted to understand the state of literature regarding adverse childhood experiences and obesity in children and adolescents. The review includes articles and texts regarding whether different types of adverse childhood experiences or the cumulative value of ACEs impact a person's obesity outcome and/or BMI trajectory. The majority of studies to date have looked at obesity as an outcome at a single point in time rather than as a trajectory.

An electronic search was conducted in the following databases: Academic Search Premier, CINHALL, Google Scholar, PsycINFO, Pubmed, SocIndex, and Web of Science. Various combinations of three categories of search terms were used: 1) adolescen\* or child\* or pediatr\* or teen\* or youth AND 2) early advers\* or ACE\* or maltreat\* or abuse or neglect or trauma AND 3) adiposity or body weight or body mass index or obesity or obese or overweight\* or over weight\*. Relevant articles using the LONGSCAN data were identified and obtained through its website (<http://www.unc.edu/depts/sph/longscan/>). The literature review focuses on findings for children and adolescents mainly; because articles about the relationship between ACEs

and childhood obesity outcomes were not abundant, studies that examined ACEs and adult obesity outcomes were also included in the review.

**Maltreatment and obesity.** Several studies have found a link between history of maltreatment and adult obesity (Boynton-Jarrett, Rosenberg, Palmer, Boggs, & Wise, 2012; D'Argenio, Mazzi, Pecchioli, Lorenzo, Siracusano, & Triosi, 2009; Felitti et al., 1998). Some, although limited, research describes the relationship between childhood maltreatment and childhood obesity (Schneiderman, Mennen, Negriff, & Trickett, 2012). The relationship appears stronger for children with experiences of neglect and child sexual abuse among different types of maltreatment; however, the findings are not consistent across the board (Feummeler et al., 2009; Moyer et al, 1997; Shin & Miller, 2012). Sample and methodological differences as well as types of maltreatment studied appear to drive the differences in findings (Danese & Tan, 2014; Shin & Miller, 2012; Whitaker, Phillips, Orzol, & Burdette, 2007).

Danese and Tan (2014) conducted a systemic review and a meta-analysis of 41 studies to test the relationship between childhood maltreatment and obesity among children, adolescents, and adults. Danese and Tan (2014) found a significant association between childhood maltreatment and obesity regardless of assessment modalities of both the independent and the dependent variable (prospective or retrospective report of maltreatment; questionnaire, interview or records of maltreatment; self-report or exam measurement of obesity; categorical or continuous measure of BMI or waist circumference). Overall, the authors concluded that the odds of being obese was 36% more for maltreated individuals than those without a history of maltreatment (CI = 1.26-1.47) (Danese & Tan, 2014). Various childhood maltreatment types predicted obesity,

including sexual abuse, physical abuse, physical neglect, and emotional abuse; only emotional neglect did not predict obesity (Danese & Tan, 2014). Danese and Tan (2014) identified current depression as a potential mediating factor for the relationship between childhood maltreatment and obesity; when they adjusted for depression, the correlation between childhood maltreatment and obesity became nonsignificant.

Some positive relationship between childhood maltreatment and BMI was found in another study but was specific to experiencing a combination of physical abuse and neglect or only neglect (Shin & Miller, 2012). Using the Add Health data, Shin and Miller (2012) examined the association between childhood maltreatment and longitudinal growth trajectories of BMI from adolescence (7<sup>th</sup>-12<sup>th</sup> grade) to young adulthood. They found that participants who had experienced both neglect and physical abuse had a significantly higher average initial BMI by .39 points compared to those without maltreatment experience (Shin & Miller, 2012). However, the BMI growth trajectory over time was similar between the group that experienced both neglect and abuse when compared to the group that did not experience any type of child maltreatment (Shin & Miller, 2012). On the other hand, those who had experienced only neglect had similar initial BMI as those who did not experience any childhood maltreatment; when it came to BMI growth trajectory, those who had experienced only neglect had a statistically significantly faster, though perhaps not clinically significant, BMI growth rate of .03 points per year than those who did not experience any childhood maltreatment (Shin & Miller, 2012). A counterintuitive finding was that experiencing all three types of maltreatment, thus having multiple risk factors, was not predictive of either higher

baseline BMI or faster growth rate of BMI compared to those without any maltreatment (Shin & Miller, 2012).

Whitaker, Phillips, Orzol, and Burdette (2007) sought to determine whether child maltreatment (neglect, corporal punishment, psychological aggression) was associated with childhood obesity at age three. Children who had experienced neglect in the prior year had an increased risk of obesity compared to those who did not experience neglect in the prior year; the odds of being obese was 56% higher for those who had experienced neglect. There was not a significant relationship between obesity and exposure to corporal punishment or psychological aggression.

Some studies focused only on neglect and its relationship to childhood obesity. Lissau and Sorenson (1994) found that children who suffer from neglect had higher risks of becoming obese in adolescence. Knutson, Taber, Murray, Valles, and Koepl (2010) examined whether care or supervisory neglect was associated with obesity in a sample of high risk children, who were neglected, physically abused, or from families with domestic violence. Knutson et al. (2010) found that care neglect, which included “poor hygiene, exposure to household environmental hazards, and inadequate health care” (p. 524), was significantly associated with higher BMI among younger children (three to five years) but not older children (six to nine years); on the contrary, supervisory neglect, which entailed “parental lack of awareness of child activities, personal preferences, and the child’s engagement in risky or deviant behaviors” (p. 524) was significantly related to higher BMI among older children but not younger children. The article did not clarify the rate occurring for each type of neglect; hence, there is some suspicion that care

neglect is more common for young children and supervisory neglect is more of an issue for school aged children.

Bennett, Sullivan, Thompson, and Lewis (2010) followed two groups of urban, African American children annually: the first group was between 4 and 7 years old and the second group was between 6 and 9 years old. Chronicity of neglect was related to lower BMIs for children at 8 ( $r(73) = -.24, p \leq .05$ ) and 9 years ( $r(50) = -.24, p \leq .10$ ) but was unrelated to BMI at earlier ages (Bennett et al., 2010). Assessing the relationship between neglect status and obesity outcomes, the authors found that overall, neglected children were not at increased risk of being underweight or overweight relative to non-neglected children (Bennett et al., 2010). In fact, at one time point, 8 years, neglected children were at a lower risk of high BMI than non neglected children (Bennett et al., 2010). Some researchers speculate that parents who neglect their children fail to provide adequate nutrition to their children, thus leading the children to be undernourished and not have adequate weight gain (Block et al., 2005).

Several researchers found that childhood sexual abuse (CSA) was related to obesity. One study found a positive relationship between history of CSA and later obesity risk among men. Fuemmeler, Dedert, McClernon, and Beckham (2009) investigated the relationship between childhood abuse and obesity in young adulthood using a sample drawn from the National Longitudinal Study of Adolescent Health (Add Health), which utilized a nationally representative sample. They built separate models by gender and found that none of the abuse categories (sexual abuse, physical abuse, neglect) was predictive of overweight/obesity in women. For men, the odds of becoming obese ( $BMI \geq 30$ ) were 1.63 times higher (95% CI [1.03, 2.58]) for those with CSA than

those without; their odds of becoming obese/overweight ( $BMI \geq 25$ ) were 1.46 times higher (95% CI [1.02, 2.08]) for those with CSA than those without. When the logistic regression model included all three types of maltreatment, CSA was still a significant predictor of obesity (OR=1.66, 95% CI [1.03-2.70]) as well as of overweight/obesity (OR=1.51, 95% CI [.76, 1.18]).

Other researchers focused on child sexual abuse histories among women only and the findings varied. Holmberg and Hellburg (2010) studied the association between sexual abuse in childhood and BMI outcomes in adolescence for females only. They found that the group that experienced sexual abuse and the group that did not experience sexual abuse had comparable BMIs, concluding that there was no significant relationship between experience of childhood sexual abuse and obesity (Holmberg & Hellburg, 2010). Similarly, Moyer, DiPietro, Berkowitz, and Stunkard (1997) did not find significant differences in BMI between those with sexual abuse history and those without in girls between the ages of 14 and 18.

However, another study found that a positive relationship between child sexual abuse history and obesity in women, but only when the sexual abuse was penetrative rather than non-penetrative (Mamun, Lawlor, Callaghan, Bor, Williams, & Najman, 2007). The study did not find a significant relationship between sexual abuse and obesity in men (Mamun et al., 2007). In a similar vein, positive findings were reported in a longitudinal prospective study that examined the link between child sexual abuse and obesity (Noll, Zeller, Trickett, & Putnam, 2007). They compared BMI scores between a sample of 84 female subjects with substantiated childhood sexual abuse and 89 demographically similar comparison female subjects without childhood sexual abuse.

They found that during early childhood and adolescence (ages 6-14 years), having a history of sexual abuse was not predictive of being at an increased risk for obesity. Women with sexual abuse histories were 2.85 times more likely to be obese (95% CI [1.06, 4.64],  $p=.009$ ) than those without a history of sexual abuse; nonetheless, this finding should be interpreted with caution because the confidence interval was wide, indicating that the effect is unclear. On the contrary, obesity rates between the two groups were comparable in early adolescence. In terms of trajectory, those who had experienced sexual abuse acquired body mass at a significantly steeper rate from childhood to young adulthood than in comparison females.

Overall evidence suggests the relationship between childhood maltreatment and childhood obesity remains unclear. Authors postulated that the inconsistencies in the findings may be due to the poor quality of some of the studies in terms of measurement; instead of measuring height and weight directly, which is more valid and reliable (Akerman, Williams, & Meunier, 2007; Merrill & Richardson, 2009), some studies used self or parent reported height and weight, which have been found to be inaccurate (Dubois & Girad, 2007; Sherry, Jefferds, & Grummer-Strawn, 2007). Schneiderman et al. (2012) concur that the overall relationship between maltreatment and childhood obesity, as well as specific relationships between types of maltreatment and obesity is unclear. Nonetheless, it should be noted that Danese and Tan's (2014) study with the strongest methodological rigor (systemic review and meta analysis) found a positive correlation between childhood maltreatment and obesity.

**ACEs and obesity.** Few researchers attempted to assess the association between early adversities and early trauma in general on obesity outcomes. Heerman,

Krishnaswami, Barkin, and McPheeters (2016) examined the relationship between adverse family experiences (AFE) and childhood overweight/obesity using a nationally representative data from the 2011-2012 National Survey of Children's Health (NSCH). AFE encompassed ACEs variables from the original CDC study as well as potentially destructive and stressful life course events in childhood. The researchers found that 20.4% of the children with 2 or more AFE were obese while 15.6% of those with 1 AFE were obese and 12.5% of children without AFE were obese.

Similarly, other studies have found a dose relationship between ACEs and obesity risk. Burke, Hellman, Scott, Weems, and Carrion (2011) conducted a retrospective, cross-sectional, chart review and assessed the impact of ACEs on overweight outcomes in 701 youth between the ages of 0 and 20.9 years ( $M=8.13$ ,  $SD=5.47$ ) residing in an urban neighborhood in CA. Having one or more ACEs was not associated with being overweight, but having four or more ACEs was related to being overweight. Gunstad, Paul, Spitznagel, Cohen, Williams, Kohn, and Gordon (2006) investigated the relationship between 19 different early life stressors among 696 adult participants whose age ranged from 18 years to 82 years ( $M=36.59$ ,  $SD=16.32$ ). They found that exposure to the total number of early life stressors was significantly related to adult BMI; obese men were more likely to have had greater total exposure to early life stressors and overweight/obese men were more likely to have been bullied and emotionally abused than normal weight men (Gunstad et al., 2006). The relationship between BMI and life stressors was not found in women (Gunstad et al., 2006). Both studies found that higher number of ACEs is related to higher likelihood of obesity (Burke et al., 2011; Gunstad et al., 2006).

Contrary to other studies that have found adverse health outcomes among children with ACEs, Marie-Mitchell and O'Connor (2013) found that obesity was in fact less common in the group with higher number of ACEs. The authors used a six item ACEs score (suspected maltreatment, domestic violence, substance use, mental illness in household, criminal behavior in household, single parenthood) and a seven item ACEs score (no high school or GED of mother in addition to the six ACEs items) to assess whether early adversities were predictive of obesity among four and five year old children (Marie-Mitchell & O'Connor, 2013). The authors suspected that this finding was due to a high percentage of low birth weight and FTT infants among high risk families; they are still catching up with regards to growth and thus they may not be prone to obesity in young childhood.

Studies that examine obesity in foster care children or children involved with the child welfare system were also reviewed to examine the relationship between ACEs and obesity. By definition, children in child welfare have been subject to, often multiple, adversities such as neglect, abuse, domestic violence, parental substance abuse, unstable living conditions, poverty, and/or mental illness (Steele & Buchi, 2008). Some studies have found that the rate of obesity in the population is similar to the national rate. Steele and Buchi (2008) conducted an analysis of the health conditions of children under 18 who were in foster care in Utah. They found that among children between the ages of 3 and 18, 17% were overweight and 18% were obese. Schneiderman, Leslie, Arnold-Clark, McDaniel and Xie (2011) found that among children who received child welfare services in Los Angeles between ages two and six, 14% were obese and 33% were

overweight/obese. A variant finding was that in their study, Schneiderman et al. (2011) found that 6% were underweight.

Schneiderman, Mennen, Negriff, and Trickett (2012) compared overweight and obesity among children who had been referred for child welfare services for maltreatment ( $n=303$ ) versus comparison children who had no previous or ongoing involvement with child welfare ( $n=151$ ); they resided in Los Angeles and were aged between 9 and 12. Among the foster care sample, 76% experienced more than one type of maltreatment (Schneiderman et al., 2012). Overall, the comparison group reported a higher rate of overweight/obesity of 26.5% versus 19.1% in the maltreated group; obesity percentage was 34.4% in the comparison group and 27.1% for the maltreated group. In general, there was a high percentage of obesity among the participants; the authors speculated that it was because the sample consisted of Los Angeles residents, where obesity is more prevalent than in other areas of the US (Mennen et al., 2012). Furthermore, the sample consisted of 88% minorities (Hispanic, African Americans, and biracial) and studies have found that Hispanics and African Americans especially are more likely to have a higher BMI compared to White counterparts (Ogden et al., 2010). There was no main effect of maltreatment status on BMI group, nor an interaction between maltreatment group and gender. Among maltreated children, being physically abused (OR = .14; 95% CI = .04–.49) or sexually abused (OR = .24; 95% CI = .06–.996) reduced the odds of being in the obese group for girls only; among maltreated children, being neglected (OR = .46; 95% CI = .25–.86) also reduced the odds of being obese for both genders compared to maltreated children without neglect (Schneiderman et al., 2012).

The relationship between ACEs and weight status may change over time, with ACEs being associated with lower weight in young childhood and obesity by adulthood. This is a particularly important perspective to keep in mind when doing anticipatory guidance with young children. Screening for ACEs may be a way to identify those healthy-weight children who are at greatest risk for obesity in adulthood.

**Failure to thrive and obesity.** Because the current sample had a subsample of children with failure to thrive (FTT), a literature review of the link between FTT and long-term growth was conducted. FTT is a term that describes children who grow inadequately when compared to age and gender specific growth charts (Kessler & Dawson, 1999). There have been studies that examine whether children who are identified as FTT in infancy or early childhood are able to reach sufficient growth in the latter years.

Winick, Meyer, and Harris (1975) assessed growth in female children adopted from Korea and placed in adoptive families before they turned three. All children were born full-term. Based on their growth prior to age two, the children were divided into three groups: malnourished (below 3<sup>rd</sup> percentile for both weight and height), moderately nourished (from 3<sup>rd</sup> through 24<sup>th</sup>), and well-nourished (above 25<sup>th</sup> percentile). After at least 6 years, when the children reached elementary school age (grade 1 to 8), the authors compared the children's height and weight among the three groups. Those who were malnourished were more likely to be shorter than the control group, but the weight differences among the groups had dissipated.

Lien, Katchadurian, and Winick (1977) used the same data set to assess whether age at adoption impacts the growth status. They reported that by the American children

on the Harvard reference standards, the three groups did not reach the 50<sup>th</sup> percentile in height and weight; however, all three groups of children surpassed the 50<sup>th</sup> percentile in height and weight when compared to the Korean reference standards (Lien, Katchadurian, & Winick, 1977).

Rudolf and Logan (2005) conducted a systematic review to assess the long term growth (at 3-9 years) among children who were termed as failure to thrive in infancy. All studies with a comparison group in the systematic review showed that children with FTT were lighter and shorter than comparison children at follow up, though in general, growth normalized over time and only a few children remained below the third percentile in both height and weight.

A subset of the LONGSCAN Baltimore sample included a community sample of children with FTT and a community sample of children without growth deficiencies in the first two years of life. The FTT subgroup was recruited during infancy and several studies have been published to examine the longitudinal growth of these children over time (Black & Krishnakumar, 1999; Black, Dubowitz, Krishnakumar, & Starr, 1999; Kim & Furman, 2014). Black and Krishnakumar (1999) compared growth curve models of children with FTT to children without FTT. Children in the FTT group received growth monitoring, nutrition counseling, and interaction coaching regarding mealtime behavior (Black, 1995) through a two year long Growth and Nutrition Clinic. Black and Krishnakumar (1999) reported that though children in the FTT group were not as heavy or tall as the children in the community group, most of the children in the FTT group caught-up, leaving only 3% of the FTT children stunted or wasted. Even though most children with FTT moved to the normal growth trajectory, the researchers recognized that

the FTT group gained both height and weight at a slower rate than that of the community group (Black & Krishnakumar, 1999).

Black, Dubowitz, Krishnakumar, and Starr (2007) compared the growth between the FTT group and the average growth (AG) group at age eight. The FTT group was randomized into a clinical intervention group (FTT-CO) or a clinical intervention and home intervention group (FTT-HI). Unadjusted analyses of BMI comparison revealed that the AG group had a higher BMI than either of the FTT groups, though the researchers observed a linear trend where the FTT-HI group sustained an intermediate position between the FTT-CO and AG group. Upon adjusting for maternal education, public assistance, and maternal anthropometry, the researchers found that the BMI for the AG and FTT-HI groups did not differ significantly; however, the FTT-CO group had significantly lower BMI than the AG group.

Even though both studies using the same dataset reported that children with FTT tend to have lower BMI compared to those without FTT, a study by Kim and Furman (2014) found that there are a few anomalies who despite their FTT status, grew up to be obese. Using the same LONGSCAN Baltimore sample, Kim and Furman (2014) identified 11 children whose BMI were over the 95<sup>th</sup> percentile for their age and gender after 2 years of age; they evaluated 6 children in their article. The children were obese by 5 to 13 years of age, and the authors speculated that perhaps the FTT intervention may have allowed children to overcompensate for their slow growth in the beginning. The sample size was small, but it is important to note that FTT children, who generally experience stunted growth, may also become obese in later childhood.

**HIV risk/in utero substance exposure and obesity.** Children who are exposed to HIV in utero, infected or not, have been found to be associated with low birth weight and slower early growth (Zunza, Mercer, Thabane, Esser, & Cotton, 2014). Researchers have found that perinatal problems are increased due to HIV's impact on the mother and the baby's immune function (Pattinson, Hulsbergen, & Van Hoorick, 2010). HIV exposed but uninfected children are subject to staggered growth also because they face multiple household adversities, such as economic hardship, parental mental health issues, neglect, exposure to violence, among others (Sherr et al., 2014).

Behnke, Smith, COMMITTEE ON SUBSTANCE ABUSE, and COMMITTEE ON FETUS AND NEW BORN (2013) reviewed literature on the association between prenatal substance use and the child's long term growth. The review found that marijuana and opiates are not associated with abnormal long term growth; the relationship between cocaine and growth was deemed inconclusive because there was a dearth of studies that examined this particular relationship; finally, they reported that prenatal nicotine exposure had an inconclusive effect on growth, as some have found no relationship and others have found that children exposed to nicotine are more likely to become obese than those who had not been exposed to nicotine in utero (Behnke et al., 2013).

**Maternal weight and obesity.** Studies have found that maternal weight or BMI is positively linked to the child's BMI most likely due to similar genetic disposition and/or behavioral/environmental factors, such as feeding habits and physical activity (Parsons, Power, Logan, & Summerbelt, 1999). Lake, Power, and Cole (1997) assessed the relationship between parental BMI and child's BMI at age 7, 11, 16, and 23. The

researchers found that at each age of follow up, the mean BMI of the children increased as the parental BMI increased. Whitaker, Wright, Pepe, Seidel, and Dietz (1997) found that having an obese parent gradually increased the obesity risk for the child as the child moved from toddlerhood to early childhood. Gibson et al. (2016) also found that maternal BMI was a significant predictor of child BMI z-score for children in their middle to late childhood. Higher maternal BMI score was predictive of higher child BMI z-scores in a sample of community children; however, maternal BMI was not a significant predictor in a clinically derived sample of only obese and overweight children (Gibson et al., 2016). Parsons, Power, Logan, and Summerbell (1999) conducted a systematic review to identify childhood predictors of adult obesity; they found that maternal obesity is a consistent predictor of the child's risk of obesity or overweight. Agras, Hammer, McNicholas, and Kraemer (2004) conducted a prospective study from birth to 9.5 years regarding risk factors for childhood overweight and found that parental overweightedness was the strongest risk factor.

**Other demographic factors and obesity.** A wealth of literature demonstrates various demographic factors related to obesity and that there are disproportionalities that exist in obesity. Socioeconomic status, race, ethnicity, and gender have been individually associated with obesity (Moore, Howell, & Treiber, 2002; Must & Tybor, 2005; Reilly, Ness, & Sherriff, 2007). Children from lower socioeconomic status or lower household income are more prone to obesity because limited income and lower education can prevent families from purchasing healthier, fresher foods and participating in healthy behaviors (Omar, Coleman, & Hoerr, 2001). Lower maternal educational attainment is related to higher BMI in children; a likely explanation is that mothers play a critical role

in terms of deciding the quality and quantity of foods consumed, where and when the intake happens (Zhang & McIntosh, 2011), and the amount and type of exercise the children engage (Einsberg, Radunovich, & Brennan, 2007). Mother's lack of knowledge about nutrition and healthy feeding or eating behaviors may prevent the children from building healthy dietary and physical habits for children.

There is also difference by racial and ethnicity status. Minority children, specifically African Americans and Hispanics, are more likely to be overweight (Delva et al., 2007; Freedman, Khan, Serdula, Ogden, & Dietz, 2006). In the adult population, African American women, regardless of income level, have been found to be disproportionately affected by obesity; 33% of men and less than 20% of women have normal BMIs regardless of income level (Kumanyika et al., 2007). In children, African American girls have the highest risk of obesity among children of various races and ethnicities even after controlling for socioeconomic status (U.S. Department of Health & Human Services, 2012; Wang & Zhang, 2006).

### **Gaps in the Literature**

Although there are a number of researchers who have studied the impact of childhood maltreatment and ACEs on adult obesity, there are only a few studies that examined the link for childhood obesity (Heerman, Krishnaswami, Barkin, & McPheeters, 2016; Schneiderman et al., 2013). Furthermore, there is no prospective, longitudinal study that assesses the impact of ACEs in early childhood on BMI trajectory over a developmental period. The relationship between ACEs and obesity outcome is not completely clear, because some studies have found significant relationships and others did not. Potentially, gaining knowledge on the interrelationship between ACEs and BMI

trajectories will aid in developing effective prevention as well as intervention strategies for childhood obesity.

### **Research Aims**

The research aims for this study are outlined and for each aim, research questions and hypotheses are provided. The ecobiodevelopmental framework and cumulative risk theory provide the theoretical framework for these research questions.

**Aim 1: Conduct a latent class growth analysis to identify trajectories of BMI for the LONGSCAN Baltimore participants.** The first aim of the study was to identify the number and shape of distinct BMI (BMI) trajectories that subgroups of young children (4 year olds) follow over a period of 14 years.

- 1) How many distinct trajectories of BMI do subgroups of these young children follow? Assuming there are more than two trajectories, what shape are these trajectories (i.e., linear, cubic, quadratic)? What proportions of children follow each trajectory?

The hypothesis was that some of these young children would have steady expected BMI development. However, some would be at high risk for obesity as identified by steep increase in their BMIs over time, whereas others would be at low/moderate risk and show slower rate of BMI increase over time. There may be a group of children that experience a decline in BMI as well.

**Aim 2: Assess whether the number of ACEs, as well as other demographic risk factors, predict group membership of the BMI trajectories.** Upon identifying the trajectory groups, multinomial logistic regression analyses were conducted to determine whether the number of ACEs predicts membership to the trajectory groups. Other risk

factors and important demographic variables were included as covariates in the model; these include FTT/not FTT during infancy, HIV risk/no risk in infancy, primary caregiver's/maternal level of education, and household income as defined by household poverty status as defined by household receipt of AFDC when the children were age 4.

- 1) Does the number of ACEs predict membership in BMI trajectory groups, after controlling for other demographic factors? What other demographic risk factors predict membership in each BMI group in multivariate analysis?

Based on cumulative risk theory, the hypothesis was that children with a high number of ACEs would be more likely to gain BMI at a steeper rate than those with few or no ACEs.

### **Chapter 3: Study Method**

This chapter describes the study method for the dissertation and includes an overview of the main statistical technique, Latent Class Growth Analysis (LCGA). The study examines BMI growth patterns of children from the LONGSCAN Baltimore data from the age of 4 to 18 and examines the predictive value of preschool (4 years) as well as school-aged (between 4 and 14) ACEs (Flaherty et al., 2013) on the BMI trajectories.

#### **Data Source and Original Data Collection**

The dissertation is a secondary data analysis of data from the Longitudinal Studies of Child Abuse and Neglect (LONGSCAN), which was initially formed with an intention to address antecedents and consequences of child maltreatment, as well as factors that mediate and/or moderate those processes (Runyan, Curtis, Hunter, Black, Kotch, Bangdiwala, & Dubowitz, 1998). There were five independent sites to LONGSCAN, one in each of the following regions: East, Midwest, South, Southwest, and Northwest. The five sites shared objectives, measures, data collection strategies, and data management and were coordinated through a single center at the University of North Carolina, Chapel Hill (Runyan, Dubowitz, English, Kotch, Litrownik, Thompson, & The LONGSCAN Investigator Group, 2011).

Only the Eastern site (Baltimore, MD) was considered in the current dissertation proposal because it is the only site that tracked weight and height data at all time points during the face-to-face interviews. For this particular region, an earlier event cohort study later joined LONGSCAN when the children were between 4 and 6. The 4-year-old sample was recruited from an inner city pediatric clinic, serving children from low-income families starting in 1991.

Consent procedures for interviews from the children and the parents were developed prior to the data collection and were approved by the IRB at the University of Maryland, Baltimore. Consent was obtained from parents or primary caregivers and assent from children were obtained once the children were old enough to provide assent. In order to maintain contact, brief telephone interviews were conducted and information about service utilization and life events were obtained in the years when face-to-face interviews were not conducted.

### **Sample**

The current study used data from the Baltimore site of LONGSCAN. The children participants were from inner city Baltimore pediatric clinics ( $N=333$ ) and were born between 1988 and 1991. The sample consisted of three groups (Hunter & Knight, 1999). The first group included 129 children who were diagnosed with non-organic failure-to-thrive (FTT). Eligibility criteria were that (1) the children's weight-for-age was below the 5<sup>th</sup> percentile based on the National Center for Health Statistics growth charts at the time of recruitment, when the children were less than 25 months old; (2) gestational age was at least 37 weeks; (3) birth weight was appropriate for gestational age; (4) no history of perinatal complication; and (5) no history of congenital disorders or chronic illnesses that could impede growth. The second group consisted of 83 children recruited from a clinic serving children of women who were infected with HIV or at a high risk for HIV due to substance use. The third group consisted of 121 inner city children whose families did not have risk factors other than poverty. These children were similar to the first two groups in terms of age, gender, and race. At the first wave of LONGSCAN, which occurred when the children were four years old, the sample size was 237. At the

second wave when the children were six years old, an additional 45 subjects from the original sample were included, bringing the total Baltimore LONGSCAN sample to  $n=282$  (103 with FTT; 68 with HIV risk; 111 with no extra risk).

For the current study, the sampling frame included children who participated in the first wave of data collection at age 4 ( $N=237$ ). Eligibility criteria were established based on the number of BMI data points available for each individual. Researchers recommend at least three repeated observations per individual (Comer & Kendall, 2013; Curran, Obeidat, & Losardo, 2010), because using two time points would result in not only statistical underidentification but also an inability to evaluate shape of the growth trajectory. Having several repeated data points is better, because it helps in statistical identification, modeling flexibility, and statistical power of analyses (Muthen & Curran, 1997). Moreover, considering BMI trajectory can be nonlinear, I decided to use only cases with at least two data points from childhood (4, 6, 8) and at least a single data point from adolescence (12, 14, 16, 18) (H. Dubowitz, personal communication, September 17, 2015). Based on these inclusion criteria, the final sample size was 201.

**Descriptive data.** Table 3.1 and Table 3.2 present descriptive statistics for the sample. Children in the current study were between 3 and 4 years old ( $M=3.84$ ,  $SD=.37$ ) at Wave 1 and they were 18 years old ( $M=18.61$ ,  $SD=.44$ ) at Wave 7 of the study. Slightly over half of the children were male (54.7%) and the majority of the children were black (93.5%). A third of the children were categorized as failure to thrive (FTT) and a quarter were considered at risk for HIV (24.9%).

Primary maternal caregivers' (biological, foster, adoptive mothers) demographics were analyzed. Over three quarters (77.5%) of the primary maternal caregivers were

receiving AFDC at the first wave of the study. Average maternal weight was 73.1 kg ( $SD=19.2$ ) and average level of education of the maternal caregivers was 11.39 years ( $SD=1.49$ ). Child and primary maternal caregiver demographics were compared for the sample that was used for the study to the sample that was excluded for the study; chi-squares and t-tests were performed and no significant differences were detected (see Table 3.1 & Table 3.2).

**Table 3.1.** Demographics of children and families at Age 4 of the LONGSCAN

Baltimore study

	<b>Total</b>	<b>Included</b>	<b>Excluded</b>	<b>Chi-square</b>	<b><i>p-value</i></b>
<b>Child Demographics</b>	(n=237)	(n=201)	(n=36)		
Gender	%	%	%		
Female	46.0	45.3	50.0	.28	.60
Male	54.0	54.7	50.0		
Race					
African American	92.4	93.5	86.1	2.40	.12
Other	7.6	6.5	13.9		
Failure to thrive	35.0	33.3	44.4	1.66	.20
At risk for HIV	24.1	24.9	19.4	.49	.48
<b>Caregiver Demographics</b>	(n=236)	(n=200)	(n=36)		
Receipt of AFDC	76.7	77.5	72.2	.48	.49

**Table 3.2.** Primary maternal caregiver demographics at Age 4 of the LONGSCAN

Baltimore study

	<b>Total</b>	<b>Included</b>	<b>Excluded</b>	<b>t-test</b>	<b><i>p-value</i></b>
<b>Caregiver Demographics</b>	(n=237)	(n=200)	(n=36)		
Mother's education level (# of years)	$M=11.34$ $SD=1.56$	$M=11.39$ $SD=1.49$	$M=11.08$ $SD=1.86$	-1.09	.28
Maternal weight (kg)	(n=202) $M=73.17$ $SD=19.81$	(n=170) $M=73.07$ $SD=19.19$	(n=32) $M=73.71$ $SD=23.14$	.17	.87

## **Measurement: LONGSCAN Measures and Construction of Variables**

All measures and variables for the current study came from the LONGSCAN Baltimore's data collected from children, caregivers, and caseworker records. The BMI (height and weight) measures and covariates and predictors used in the study are explained below. Table 3.4 provides a summary of the measures.

**BMI (outcome variable).** The primary outcome was the raw BMI score. Raw BMI was calculated by dividing weight in kilograms by the squared value of the height in meters  $(\text{kg})/(\text{m})^2$ . Trained nurses measured height in meters using a tape measure by standing the child on a hard surface floor and placing a wooden ruler on top of the head, perpendicular to the wall. A Tanita BF-578 Body Fat Scale, which was calibrated on a regular basis, was used on hard surface floor to measure weight (kg).

**Adverse Childhood Experiences (ACEs) Score.** Dr. Anda and his colleagues developed the original CDC-ACEs questions and score after reviewing the literature and consulting experienced researchers in the field; the questions and score have good test-retest reliability (Dube, Williamson, Thompson, Felitti, & Anda, 2004). Based on the original CDC-ACEs study, LONGSCAN researchers developed an ACEs scoring system (Flaherty, Thompson, Dubowitz, Harvey, English, Proctor, & Runyan, 2013; Thompson, Flaherty, English, Litronik, Dubowitz, Kotch, & Runyan, 2015). They included four categories of maltreatment (psychological maltreatment, physical abuse, sexual abuse, and neglect) and four measures of household dysfunction (caregiver's substance use/alcohol abuse, caregiver's depressive symptoms, caregiver being treated violently, and criminal behavior in household) (Flaherty et al., 2013; Thompson et al., 2015).

For the current study, ACEs were measured in two ways. First, a cumulative ACEs score of preschool ACEs when the children were four years old was calculated. Second, a cumulative ACEs score based on a previous study (Flaherty et al., 2013), hereafter referred to as school aged ACEs, was calculated based on ACEs occurring between ages 4 to 14.

***Preschool ACEs score.*** The ACEs score included seven variables and was construed as a continuous variable ranging from zero to seven. The variables were akin to previous studies that examine the impact of ACEs using the LONGSCAN data. The items consisted of four child maltreatment variables (physical abuse, sexual abuse, psychological maltreatment, and neglect) and three household dysfunction variables (caregiver's alcohol abuse, caregiver's depressive symptoms, and caregiver being treated violently). The household dysfunction variable that reflects criminal behavior in household was not included in the current study because it was not measured when the participating children were four years old. Caregiver's use of alcohol was assessed; only the use of alcohol rather than the use of other substances was included, because other substance use when the children were four years old was not assessed for the entire sample. All ACEs variables were dichotomized (1 = *present*, 0 = *absent*) and then summed to produce a cumulative score.

***Childhood maltreatment.*** The current study incorporated four childhood maltreatment variables for the ACEs score. They were psychological maltreatment, physical abuse, sexual abuse, and neglect. The variables were operationalized the same way that previous researchers had done (Flaherty et al., 2013; Thompson et al., 2015).

LONGSCAN coded Child Protective Services (CPS) administrative records of substantiated and unsubstantiated childhood maltreatment reports using the Modified Maltreatment Classification System (MMCS), which was developed by English and the LONGSCAN Investigators (1997) based on the Maltreatment Classification System of Barnett, Manly, and Cicchetti (1993). This decision was based on research that suggests differentiating substantiated and unsubstantiated reports is not useful (Drake et al., 2011; English et al., 2005; Hussey et al., 2005; Kohl, Jonson-Reid, & Drake, 2009). Thus, the current study construed both alleged and substantiated cases as indicative of abuse.

The four types of maltreatment were defined as follows and dichotomized. *Psychological maltreatment* was defined as threats to psychological safety and security, lack of acceptance and threats to self-esteem, or failure to allow age-appropriate autonomy. *Physical abuse* was defined as any deliberate infliction of injury such as blows or injury to the head, torso, buttocks, or limbs; and violent handling, choking, burning, shaking, or nondescript injury. *Sexual abuse* was defined as any sexual exposure, exploitation, molestation, or penetration between responsible adult and the child. *Physical and supervisory neglect* was defined as failure to meet the child's physical needs by providing adequate supervision to ensure the child's safety. Interrater reliability of the coding has been proven to be high, with kappas ranging from .73 for psychological abuse to .87 for physical abuse (Thompson et al., 2015). All four variables were coded as (1 = *present*, 0 = *absent*).

*Household dysfunction.* Household dysfunction consisted of three items for preschool ACEs: (1) caregiver depression, (2) caregiver alcohol use, (3) caregiver being treated violently. Caregiver depression was assessed using the Center for Epidemiologic

Studies Depression Scale (CES-D), a 20 item questionnaire that captures six major dimensions of depression: depressed mood, feelings of worthlessness, feelings of helplessness and hopelessness, psychomotor retardation, loss of appetite, and sleep disturbance. Data were collected from the primary caregivers when the children were 4 years old. Total score ranges from 0 to 60; based on Radloff's (1977) recommendation, scores 0 - 15 were coded as 0 (*not depressed*) and 16 and above were coded as 1 (*depressed*).

Alcohol use by the primary maternal caregiver was assessed using the CAGE Questionnaire (Ewing, 1984; Ewing & Rouse, 1970). It is a brief screening tool of alcohol use and consists of four questions (Cut down, Annoy, Guilty, and "Eye-opener"). The interviewer administered the CAGE Questionnaire to the parent when the children were four years old. The scores range from 0 to 4, where higher scores indicate greater risk for alcoholism. Bradley et al. (1998) recommended that the cutoff point for the CAGE to be lowered from two to one or more positive responses in order to increase sensitivity by as much as 20%, up to a 10% maximum reduction in specificity. Hence, presence of any positive responses in the CAGE was recorded as (1 = *alcohol use*) and no positive responses in the CAGE as (0 = *without alcohol use*).

Presence of "caregiver treated violently" was identified using a two-step process using the History of Loss and Harm (Hunter & Everson, 1991) scale and the Autonomy and Relatedness Inventory ([ARI]; Hall & Kiernan, 1990) for the age 4 variable. History of Loss and Harm (Hunter & Everson, 1991) is an 18-item measure, which was designed to assess the person's lifetime experience of physical and sexual abuse, and separation from/loss of primary caregiver in childhood. In the LONGSCAN study, it was assessed

when the children were 4 years old. Presence of violence was indicated by affirming to the following statement: “Since you’ve been an adult, have you ever been hit, slapped, beaten, or pushed around by someone?” Mitchell (2008) reported that 88% of LONGSCAN (including all regions) respondents reported that the perpetrator was their husband or their partners. An affirmative response to the statement indicated presence of experience of violence (1 = *caregiver treated violently*; 0 = *caregiver was not treated violently*).

***School-aged ACEs score.*** Eight categories of adversities were considered (Flaherty et al., 2013; Thompson et al., 2015). Four child maltreatment variables (physical abuse, sexual abuse, psychological maltreatment, and neglect) were included and these were measured identically as the aforementioned preschool ACEs index. Presence of each of the four types of abuse at any data point between 4 and 14 years was coded as present or not present (1 = *present*; 0 = *not present*).

However, there were some differences in the measurement of household dysfunction from the preschool ACEs score. Caregiver’s depression was the only item measured in the same way as it was for age 4. Caregiver’s substance abuse included not only alcohol but also other illegal substances. Caregiver being treated violently was measured using a different measurement, and criminal behavior by household member was included in this index. Again, all adversity was dichotomized and summed to produce an overall ACEs score that ranged from 0 to 8.

***Household dysfunction.*** Household dysfunction consisted of four items for school aged ACEs: (1) caregiver depression, (2) caregiver’s substance use, (3) caregiver being treated violently, and (4) criminal behavior by household member.

Caregiver depression was assessed using the CES-D and data were collected from the primary caregivers when the children were 4, 6, 12, and 14 years old. If the caregiver reported a score equal or above 16 at any of the four data points, caregiver depression variable was coded as 1 (*depressed*). If the CES-D score was 15 or below at all available four data points, this variable was coded as 0 (*not depressed*).

For the substance use variable, two different measures were considered for different data points. When the children were 4, CAGE was administered to caregivers. When the children were 8, 12, and 14, the Caregiver Substance Use measure, developed by LONGSCAN, was administered to caregivers. The questions asked about the caregiver's use of common legal (tobacco and alcohol) and illegal substances (marijuana, cocaine, hallucinogens, heroin, and stimulants). Positive response on the CAGE and current use of any illegal substances and/or "daily" use of alcohol at any of the four time points were coded as 1 (*substance use present*) while a negative response at all available time points was coded as 0 (*without substance use*).

When the child was 6, 8, 12, and 14, the partner-to-partner Conflict Tactics Scale (Straus, 1979) was administered to maternal caregivers. Any positive responses to kicking, biting, punching, hit with an object, being beaten up, threatened with a knife or a gun, or the victim of a knife or a gun in the previous 3 months, for any given time point, were coded as 1 = *caregiver treated violently*; negative responses were coded as 0 = *caregiver was not treated violently*.

Finally, criminal behavior by household member was assessed using the Child Life Events measure, developed by LONGSCAN. The caregivers were asked whether anyone in the child's household was jailed or imprisoned in the past year. The instrument

was administered to caregivers when the children were 6, 8, 12, and 14. Affirmative responses at any of the four time point were coded as 1 = *criminal behavior in the household* and nonaffirmative responses at all time points as 0 = *no criminal behavior in the household*.

***Demographic control variables.*** The data for demographic variables were derived from Age 4 of the LONGSCAN Baltimore study. Child gender was coded as 1 = *male* and 2 = *female*. Race and ethnicity were excluded from analyses because the sample was mostly African Americans (93.5%) (5% Caucasian, .4% Hispanic, and other 1.8% at Age 4) and there were no differences in mean BMI by race (see Table 3.3); thus race was not included as a control variable. Receipt of AFDC (1 = *receive AFDC*, 0 = *do not receive AFDC*) was included to distinguish levels of economic status of children at age four. Primary maternal caregiver's education (# of years of education) when the children were four years old was also included. Finally, because the sample included failure to thrive (1 = *failure to thrive*, 0 = *normal growth*) and HIV risk groups (1 = *HIV risk*, 0 = *without HIV risk*), those terms were added as demographic control variables to assess whether group membership impacts identification with certain BMI trajectories.

**Table 3.3.** Comparison of Average BMI by Race

BMI	Non black			Black			<i>t</i> -test	<i>p</i> -value
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>		
Wave 1 (4yrs)	13	14.96	1.21	186	15.80	1.83	-1.63	.10
Wave 2 (6yrs)	13	15.02	1.47	167	16.15	2.45	-1.65	.10
Wave 3 (8yrs)	13	16.42	2.84	172	17.54	3.81	-1.03	.30
Wave 4 (12yrs)	10	20.41	5.61	140	21.15	5.95	-.38	.70
Wave 5 (14 yrs)	11	23.84	6.20	137	22.69	5.94	.62	.54
Wave 6 (16 yrs)	3	22.56	1.43	68	25.39	5.61	-.25	.80
Wave 7 (18 yrs)	12	28.93	8.89	148	26.89	7.41	.90	.37

**Table 3.4.** Variables and Measurement for the Proposed Study

<b>Concept / Variable</b>	<b>Measurement</b>	<b>Source of Information</b>	<b>Children's Age</b>
<i>Outcome Variable</i>			
Body Mass Index	Height (meter) and weight (kilogram) measured. BMI = Weight (Kg)/ Height (m <sup>2</sup> ) Continuous variable	Child, administered by trained nurse or research assistant	4, 6, 8, 12, 14, 16, 18
<i>Independent Variables for the Multinomial Regression Analyses</i>			
Gender	1 = Male 2 = Female Dichotomous variable		
FTT or Not	1 = FTT 0 = Normal Growth (NG) Dichotomous variable	Parent or caregiver	4
HIV Risk or Not	1=HIV risk 0=No risk Dichotomous variable	Parent or caregiver	4
Maternal Weight	Weight in kg Continuous variable	Parent or caregiver	4
Maternal level of education	Years of education Continuous variable	Parent or caregiver	4
Household income	1 = Receive AFDC 0= Do not receive AFDC	Parent or caregiver	4
Preschool ACEs (occurring at age 4)	Combined score for (1) physical abuse; (2) sexual abuse; (3) psychological maltreatment; (4) physical and supervisory neglect; (5) caregiver depression, (6) caregiver's alcohol use, (7) caregiver treated violently Continuous variable (sum of seven dichotomous variables, coded as 0 or 1)	(1-4) CPS records; (5-7) Parent or caregiver	4
School-aged ACEs (occurring at age 4-14)	Combined score for (1) physical abuse; (2) sexual abuse; (3) psychological maltreatment; (4) physical and supervisory neglect; (5) caregiver depression, (6) caregiver's substance use, (7) caregiver treated violently, (8) criminal behavior by household member Continuous variable (sum of eight dichotomous variables, coded as 0 or 1)	(1-4) CPS records; (5-8) Parent or caregiver	4, 6, 8, 12, 14

## **Data Analysis Plan**

The current study was conducted in two phases. The first phase used a type of growth mixture modeling (GMM): latent class growth analysis (LCGA). LCGA was conducted to identify distinct BMI trajectories in the Baltimore LONGSCAN population. The second phase entailed running multinomial logistic regression analyses to assess whether the number of ACEs as well as other demographic factors can predict a child's membership to a particular BMI trajectory. This section provides detailed information on the analytical strategies for considering the complex nature of the analyses.

**Latent class growth analysis.** LCGA was conducted to identify distinct BMI trajectories in this Baltimore LONGSCAN sample using the participants' raw BMI score. LCGA combines mixed effects multilevel modeling and latent class modeling procedures; it produces random slopes and intercepts, which are continuous latent variables and trajectory classes, which are categorical latent variables (Muthén, 2001).

LCGA is different from the conventional latent growth curve model; whereas the conventional latent growth curve model assumes that a single trajectory that takes an average can sufficiently describe the entire population, LCGA recognizes the heterogeneity in the population and identifies several latent subgroups that follow distinct trajectories over time for a repeatedly measured specific outcome (Nagin, 2005). As a person-centered statistical approach, LCGA does not rely on pre-determined groups (Jung & Wickrama, 2008). LCGA will cluster individuals with similar trajectories and place them in separate groups, and within each group, the children share developmental trajectories of the target outcome (Jung & Wickrama, 2008).

Hence, LCGA first identifies heterogeneous classes within each outcome of interest based on their distinct trajectories over time. Then, LCGA estimates the optimal number of classes/subgroups of individuals within the population who follow similar trajectories over time, the intercepts (initial value) and slopes (change over time) for these subgroups, and the proportion of individuals belonging to each group. Trajectories can be modeled to follow linear, quadratic, or cubic trajectories. Because the children in a particular class will share a single trajectory over time, within a class, the random effects for intercept, slope, and quadratic term are set to zero (Fitzmaurice, Davidian, Verbeke, & Molenberghs, 2008). The fact that random effects are set to zero can be considered a shortcoming of LCGA; LCGA cannot estimate random effects within each class or allow variation across individuals within classes (Jung & Wickrama, 2008). Nonetheless, holding the variance of intercept, slope, and quadratic term at zero allows modeling to be simpler and less likely to have convergence problems (Nagin, 2005).

LCGA is appropriate for the study because even though the children were from similar socioeconomic and geographic backgrounds, there was diversity in the sample especially considering that it included three cohorts of children who were identified as FTT, at risk for HIV due to mothers with substance use, and without risks other than poverty. The heterogeneity of the sample portends a more diverse growth trajectory than if the sample were homogeneous. LCGA identifies children following distinct BMI trajectories, whether “low risk” or “high risk”; moreover, it allows to describe the size and shape of each trajectory.

***Specifying the LCGA model.*** For the current study, *Mplus* Version 7.3.1 (Muthén & Muthén, 2015) was used to model distinct BMI trajectories of young children.

BMI was charted at ages 4, 6, 8, 12, 14, 16, and 18 (7 Waves). After preparing and importing BMI data for the seven time points into *Mplus*, I ran a series of unconditional LCGA models to estimate the optimal number of classes and form of BMI trajectories. The time points in the model were plotted at 0, 1, 2, 4, 5, 6, 7, and 8. A time varying covariate (TVC) was used because the distance between Waves was not consistent; time point 3 was skipped in order to recognize that data were not collected when the participants were 10 years old, and thus there was a longer period of time in between 8 and 12 years (Wave 3 and Wave 4). Using a time variant covariate is especially important in BMI trajectory modeling during this time, because children go through significant growth during pubertal years.

Linear models were run first and then another set of models were run specifying a quadratic term, based on some evidence that BMI growth in childhood and adolescence may be curvilinear (Cecil-Karb & Grogan-Kaylor, 2009; Heo, Faith, Mott, Gorman, Redde, & Allison, 2003). A cubic term was not used because the minimum number of data points for each case was three and a cubic shape would require at least four data points for accurate modeling.

In order to estimate the number of classes, I modeled a range of class numbers and examined which best fits the data sets. Four linear LCGA models were specified (1, 2, 3, and 4 class models) for BMI. First, I fitted an unconditional one-class latent class growth curve model. Because in theory, at least two trajectories, one that is “high risk” because BMI increases at a steep rate over time and one that is “no risk” where BMI stays in the healthy range over time, were expected to emerge, I tested a 2 class model subsequently. A 3-class model was tested based on Lane, Bluestone, and Burke’s (2013)

study that identified three trajectories of BMI percentile growth of ‘elevated,’ ‘steady increase,’ and ‘stable,’ as well as Garden, Marks, Simpson, and Webb’s (2012) study that charted ‘normal,’ ‘early and persistent increase,’ and ‘late increase’ trajectories. A 4-class model was tested, as Nonnemaker et al. (2012) identified four trajectories: “high risk” for obesity, “moderate-to-high risk,” “low-to moderate,” and “low-risk” for obesity. Models with more than four classes were not considered because Monte Carlo studies have illustrated that there is a flattening out of log likelihood values when moving from four to five classes and when moving from four to six classes for growth mixture models (GMM) and latent class analysis models (Nylund, Asparouhov, & Muthén, 2007). This trend was observed regardless of the sample size, which ranged from 200 to 1000 (Nylund, Asparouhov, & Muthén, 2007). Furthermore, given the sample size of the current study, class sizes larger than four would yield classes with insufficient sample sizes. The models were run again with a quadratic term, thus bringing the total number of models using the study sample to eight.

Additionally, the models were run with and without the non African American sample in order to test for race effects. Finally, models were run with gender as a known class, as some studies have found differences in the impact of ACEs on BMI trajectory by gender (Fuemmeler et al., 2009; Noll et al., 2007). It is common practice to separate BMI trajectories by gender, because their growth patterns differ (Garden et al., 2012). Sample size will be smaller if trajectories are separated by gender. However, Muthén and Muthén (2002) emphasized that more important than sample size is the specifics of the situation and that they had successfully implemented mixture models with 30 subjects (Muthén, 2013).

*Selecting the optimal LCGA model by assessing model fit.* In order to select the optimal LCGA model, the output for each of four linear and four quadratic models were assessed and compared. When a quadratic term was added and the models were run, the models did not converge, most likely due to computational burden (Jung 2007). Thus only linear models were considered.

The covariance coverage in this data set ranged from 0.597 to 0.990, which met the minimum requirement (.10) for model estimation (Muthén & Muthén, 1998-2010). A series of fit indices, average posterior probability of group membership, and entropy, were examined to identify the optimal model. In addition, graphic output was visually examined to help understand how well the models fit the data.

The Bayesian Information Criterion (BIC) was examined to determine the optimal number of classes and the best fit model. BIC is a goodness-of-fit measure and is defined as “the log-likelihood evaluated at the maximum likelihood estimate less one-half the number of parameters in the model times the log of the sample size” (Jones, Nagin, & Roeder, 2001, p. 390). BIC penalizes model complexity, such as the number of parameters in the model (Bauer & Curran, 2004; D’Unger, Land, McCall, & Nagin 1998; Kass & Raftery, 1995), which is useful in identifying the best model in large samples (Barron & Cover, 1991). However, Barron and Cover (1991) criticized BIC for being inclined to choose too simple of a model when the sample size is small. Nonetheless, simulation studies have confirmed the utility of BIC in identifying the best number of classes in LCGA (Nagin, 2005; Nylund, Asparouhov, & Muthén, 2007). The model with the BIC statistic that is the least negative and closest to zero should be selected, because

it is most likely to be correctly specified (Nagin, 2005). For *Mplus*, Muthén (2008) recommends to select the model with the lowest BIC.

Akaike's Information Criteria (AIC) is another information criterion that was examined. AIC is similar to the BIC but does not vary based on sample size (Nagin, 2005). AIC is susceptible to overfitting, selecting models with additional classes (Nagin, 2005, Nylund, Asparouhov, & Muthén, 2007). AIC and BIC were examined in conjunction with each other; lower AIC and BIC indicated the best fit model.

The Lo, Mendell, Rubin statistic (LMR) and Bootstrap likelihood ratio test were also used to identify the best model. They complement the BIC, which tends to favor models with fewer classes (Bauer & Curran, 2004). LMR can compare mixture models with different numbers of latent classes (Lo, Mendell, Rubin, 2001). The LMR statistic tests two neighboring class models ( $k$  classes against  $k-1$  classes). The  $p$ -values are examined; a significant chi-square value ( $p < .05$ ) indicates that the  $k-1$  class model has to be rejected in favor of the  $k$  class model (Nylund et al., 2007).

Nylund et al. (2007) conducted simulation studies and reported that the bootstrap likelihood ratio test (BLRT) performed best in identifying the best fit model. BLRT uses bootstrap samples to estimate the distribution of the log likelihood difference test statistic; it compares the model fit between  $k-1$  classes and  $k$  classes, similar to the LMR statistic, a significant  $p$  value indicates that  $k-1$  class model has to be rejected in favor of the  $k$  class model (Nylund et al., 2007).

Another criteria that I examined considered the parsimony of the model. Little (2013) states that typically the candidate models have numbers of classes that are different by one (e.g., three-class vs. four-class models). He recommends that the

researcher choose the model that is the most parsimonious and with an adequate overall goodness of fit (Little, 2013). Thus, I also considered this criterion while assessing the interpretability of the classes.

After selecting the final model, posterior probabilities and entropy were critiqued to assess the distinctiveness of classes in the final model (Jedidi et al., 1997; Jung & Wickrama, 2008). Posterior probability determines the most likely class for a given child; it notates the probability a given child is in a particular class, where a maximum probability represents the highest probability of group membership for the child. For example, in a three-class model, if a child's most likely class membership to class A is .1, class B is .8, and class C is .1, the child will be assigned to class B. Average posterior probability of class membership was also examined to assess the degree of classification error and to check the adequacy of the model (Nagin, 2005).

Entropy measures classification uncertainty and it is based on posterior probability. *Mplus* reports relative entropy of a model; this reported entropy ranges from zero to one, and a higher entropy value close to one is preferred. Because entropy is a function of the number of classes, a model with as many classes as observations would have an entropy value of one. Therefore, entropy values close to one indicate a high certainty in classification.

Lastly, the author visually examined the data through graphic output to determine whether the classes in the final model are distinct and meaningful. The estimated and actual means of the trajectories were visually compared as well to assess the relative difference. In addition, individual trajectories for children assigned to each class were examined to study how well they fit the estimated trajectory group line. In summary,

model selection was based on better model fit (AIC, BIC, LMR, BLMR), models that do not have classes with a low proportion of participants in a given class, and higher entropy. Upon identifying the best-fitting model, the output of “most likely class membership” was exported from *Mplus* and they merged with the SPSS file.

**Bivariate analyses.** Bivariate analyses were conducted to describe the characteristics of children (gender, HIV risk, FTT, ACEs) and their caregivers (maternal weight, receipt of AFDC, maternal level of education) in each trajectory group that emerged from the data through LCGA. SPSS 22.0 was used. *P-value* that is less than .05 are considered statistically significant; for the current study, results that yielded *p-value* between .05 and .1 are also reported, but these results should be interpreted with caution.

**Multinomial logistic regression.** Multinomial logistic regression analyses were conducted using SPSS 22.0 to assess whether the number of ACEs predicted a child’s membership to a particular BMI trajectory. Demographic variables (gender, maternal level of education, HIV risk through having mothers with substance abuse/no risk, FTT/not FTT, and AFDC/Medicaid receipt) were entered first as control variables and then the number of ACEs, which was the main independent variable of interest. The adjusted Wald F, which is more conservative than Wald, as well as the *p-value* for the Wald F test were reported from the analyses. Odds ratios and 95% confidence intervals for each significant variable were reported. Statistical significance was set at .05 (*p-value*), but similar to bivariate analyses, results that had *p-value* of .05 and .1 were also reported. The latter findings should be interpreted with caution.

**Handling of missing data.** Because LONGSCAN is a longitudinal dataset that has been tracking children over approximately 14 years, there are missing data due to

participant attrition. The amount of missing data generally increased with Wave, with a significant decrease at age 16 (Wave 6).

***LCGA missing data.*** In order to ensure that the output models would accurately represent the data, the researcher excluded cases with fewer than three BMI data points. Additionally, each individual had to have at least two data points from childhood years (Ages 4 – 8) and at least one data point from adolescence (Ages 12-18) to be included in the dataset. This was done so that the modeled trajectory would be across the childhood/adolescent life span. Ensuring each case has enough data points was important because BMI changes unpredictably from one wave to another and from one individual to another; without sufficient data points, estimating missing data correctly is difficult.

I assumed that the missing data were missing at random (MAR), which means that “the probability of missing data on a variable is related to other variables, but not to the would-be values of the incomplete variable” or missing completely at random (MCAR), which means that the “propensity for missing data on a particular variable is unrelated to other measured variables and to the would-be values of that variable” (Enders, 2011). In the LCGA analysis, full information maximum likelihood (FIML) estimation was used to handle the missing data. *Mplus* estimates parameters using FIML to build models whilst making use of all available data points and without discarding incomplete cases or imputing missing values (Enders, 2011; Muthén, 2004; Schafer & Graham, 2002). *Mplus* provides a covariance coverage matrix that shows the proportion of observations available for each pair of variables and *Mplus* recommends a minimum coverage proportion of 10%. FIML adjusts the standard errors and scales the chi-square statistic to account for nonnormally distributed data (Little et al., 2014). Alternative

approaches to handling missing MAR data, such as multiple imputation (MI) and maximum likelihood (ML) were considered; however, these methods were not used because they are not available within a mixture modeling framework or would not allow estimation of model comparison likelihood test, thus impeding the ability to choose the optimal number of classes.

*ACEs and covariate missing data.* With regards to ACEs variables, there were no missing data. The sample composed of only those with data available at Age 4 (Wave 1); hence, there was no missing data for the categorical variables/ACEs categories. Whether the child was considered FTT or at risk for HIV was also determined at time of recruitment; hence, there were no missing data for these variables either. Missing data for the receipt of AFDC and maternal level of education were minimal (<.01%). However, 14.8% of the data were missing for maternal weight. Multiple imputation (MI) based on log-linear modeling was used because simulation studies have confirmed that it produces unbiased statistical inference and is robust against deviations from the assumed imputation model (Ezzati-Rice et al., 1995; Schafer & Graham, 2002; Vermunt, Ginkel, Der Ark, Andries, & Sijtsma, 2008). The MI procedure generated 10 complete data sets and pooled estimates are reported in the results.

## Chapter 4: Results

Results are presented in three main parts. First, BMI descriptive statistics are presented. Second, the first aim is addressed; results from the LCGA analyses are presented, including model selection, BMI trajectories, and description of the subgroups. Third, the second aim is addressed; results from multinomial logistic regression are evaluated to assess the relationship between preschool ACEs and school-aged ACEs, as well as other demographic characteristics, and class membership.

### BMI Descriptive Statistics

Foremost, average BMI scores were compared between the final sample of children who were included in the analysis based on the inclusion criteria of having two BMI data points in childhood and one in adolescence and the excluded sample of children. No significant differences were detected (See Table 4.1).

**Table 4.1.** Children’s BMI by Age: Comparison of Included and Excluded

BMI	Included			Excluded			<i>t-test</i>	<i>p-value</i>
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>		
Wave 1 (4yrs)	199	15.75	1.81	34	15.56	1.95	-.60	.55
Wave 2 (6yrs)	180	16.07	2.41	19	15.69	2.59	-.66	.51
Wave 3 (8yrs)	185	17.46	3.75	14	16.29	2.61	-1.15	.25
Wave 4 (12yrs)	150	21.10	5.92	1	15.55	-	.*	-
Wave 5 (14 yrs)	148	22.78	5.95	5	22.92	5.17	.05	.96
Wave 6 (16 yrs)	71	23.35	5.50	1	28.82	-	.*	-
Wave 7 (18 yrs)	160	27.04	7.52	8	27.77	5.89	.27	.79

\*Cell sample size was too small to conduct a t-test analysis

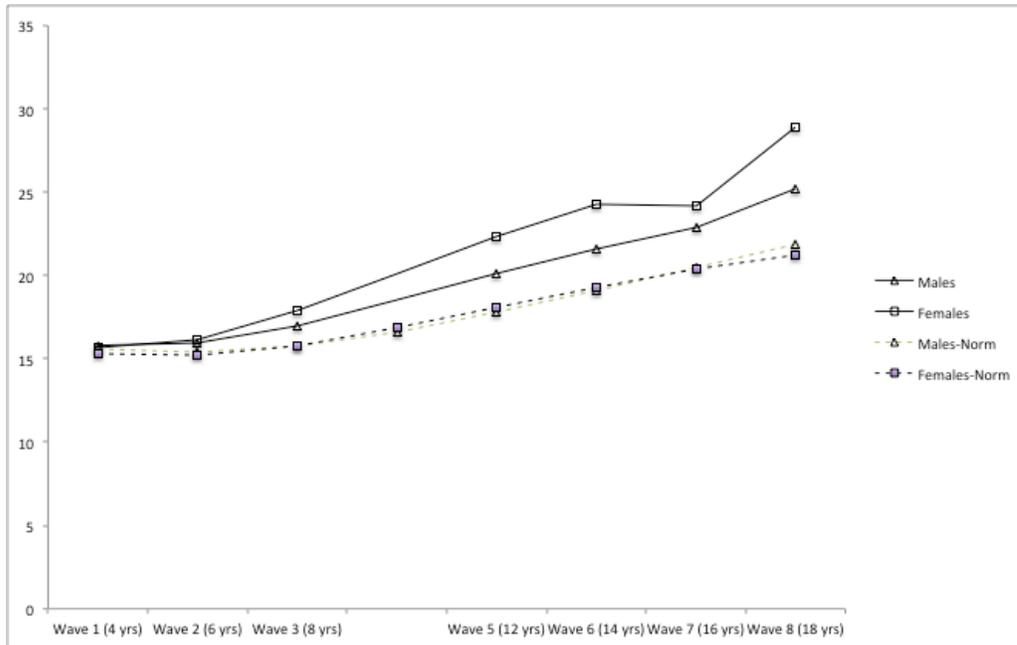
Table 4.1 also reports the mean BMI at each wave in the analytical (included) sample. Mean BMI increased with each subsequent wave and ranged from 15.75 at age 4 (Wave 1) to 27.04 at age 18 (Wave 7). The overall change in BMI over the waves demonstrates expected growth; average BMI is more or less steady between Wave 1 (age 4) and Wave 2 (age 6) and then increases through adolescence.

Average BMIs by Wave by gender were also compared (see Table 4.2). T-tests were conducted; at Waves 4, 5, 7, female average BMI was significantly higher than male average BMI ( $p < .05$ ) by 2.22, 2.63, and 3.75 points respectively (See Table 4.2). Figure 3 also depicts average BMIs by gender but also includes normal BMI (50<sup>th</sup> percentile as defined by CDC growth charts) by gender for each corresponding age (CDC, 2010). The “normal weights” are lower for both males and females; the discrepancies are larger for females.

**Table 4.2.** Average Body Mass Index (BMI) for children by wave by gender

BMI	Males			Females			<i>t-test</i>	<i>p-value</i>
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>		
Wave 1 (4yrs)	109	15.81	1.86	90	15.67	1.76	0.54	0.59
Wave 2 (6yrs)	98	15.98	2.14	82	16.18	2.71	-0.57	0.57
Wave 3 (8yrs)	96	17.02	3.23	89	17.92	4.22	-1.64	0.10
Wave 4 (12yrs)	84	20.12	5.13	66	22.34*	6.62	-2.32	0.02*
Wave 5 (14 yrs)	83	21.62	5.4	65	24.25*	6.33	-2.73	0.01*
Wave 6 (16 yrs)	46	22.91	5.77	25	24.17	4.98	-0.92	0.36
Wave 7 (18 yrs)	81	25.19	5.94	79	28.93*	8.47	-3.25	0.00*

Figure 3. Comparison of average BMIs by gender (sample BMI vs. 50<sup>th</sup> percentile based on CDC growth charts)



**Aim 1: Conduct a latent class growth analysis to identify trajectories of BMI for the LONGSCAN Baltimore participants.**

Using the BMI data, a series of LCGA models were conducted to identify the optimal number of classes and shape of BMI growth trajectories that the children from the LONGSCAN Baltimore site followed over a span of 14 years. Results are summarized for BMI growth trajectories in the subsequent sections, including model comparison and selection, description of the growth trajectories, and characteristics of the subgroups.

**LCGA model selection.** Model fit statistics for the linear LCGA models for 1, 2, 3, and 4, and classes are presented in Table 4.3. The single model provided a baseline from which I could explore unobserved trajectories. LMR and BLRT statistics are not calculated for single class models in *Mplus*.

**Table 4.3.**

Latent class growth analysis model fit statistics for BMI growth trajectories (N=201)

Number of classes	Log-likelihood	LMR	LMR Adj	BLRT	AIC	BIC	Sample size adj BIC	Entropy
Linear Models								
1	-3063.210	-	-	-	6144.420	6174.150	6145.637	-
2	-2803.355	519.710 <i>p</i> =.118	488.976 <i>p</i> =.131	519.710 <i>p</i> <.000	5630.710	5670.350	5632.332	0.959
3	<b>-2722.099</b>	<b>162.513</b> <i>p</i> =.029	<b>152.902</b> <i>p</i> =.032	<b>162.513</b> <i>p</i> <.000	<b>5474.198</b>	<b>5523.747</b>	<b>5476.225</b>	<b>0.940</b>
4	-2671.717	100.763 <i>p</i> =.315	94.805 <i>p</i> =.336	101.749 <i>p</i> <.000	5379.434	5438.894	5381.867	0.887

**Bold with gray shading** highlights the model that fits the data best (the 3 class linear model) as indicated by LMR, LMR adjusted, and BLRT tests.

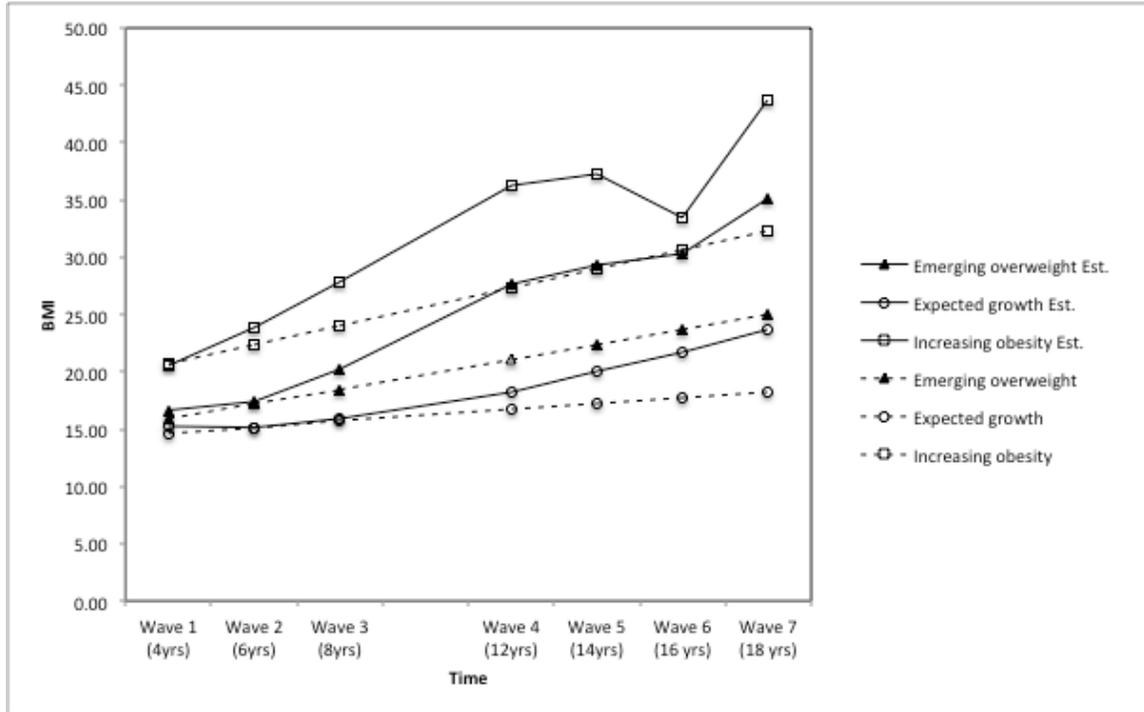
With every additional class, the Akaike information criterion (AIC) and Bayesian information criterion (BIC) decreased. AIC and BIC decreased by the largest amount when going from a 1-class model (AIC=6144.420, BIC=6174.150) to a 2-class model (AIC=5630.710, BIC=5670.350). However, LMR (519.710, *p*=.118) nor adjusted LMR (488.976, *p*=.131) were significant for the 2 class model. On the other hand, the 3 class linear TVC model had significant LMR (162.513, *p*=.029) and LMR adjusted statistics (152.902, *p*=.032), in addition to a significant BLRT statistic (162.513, *p*<.000); these statistics demonstrated that the 3-class model better fit the data than the 2-class model. Entropy was slightly higher for the 2-class model (.959) compared to the 3-class model (.940) but the difference was minute (.02).

Four-class linear TVC model was also considered. Though the AIC and BIC scores for the 4-class model (AIC=5379.434, BIC=5438.894) were the lowest, the level of decrease of both statistics were larger when going from 2-class model (AIC=5630.710, BIC=5670.350) to a 3-class model (AIC=5474.198, BIC=5523.747) than from a 3-class

model to a 4-class model (AIC=5379.434, BIC=5438.894), indicating that the model fit improved more when going from a 2-class model to a 3-class model. Four-class model also did not have significant LMR (100.763,  $p=.315$ ) nor a significant adjusted LMR (94.805,  $p=.336$ ). BLRT statistics were significant for all models, so they were unable to distinguish better fit; nonetheless, a significant BLRT for the 3-class model supported that the 3-class model fit the data well. Based on these logics, the 3-class model was selected as the best-fitting model.

The three class linear TVC LCGA model estimates that the high-risk children who were predominantly African American and from lower socioeconomic background are likely to follow one of three different BMI trajectories (Figure 4). In addition to estimated average BMI, Figure 4 also depicts actual average BMI by age/wave. The black lines represent the *emerging overweight* group, the green lines represent the *increasing obesity* group, and the red lines represent the *expected growth* group. There are two lines for each color, because one shows the actual data and the other shows the estimated data based on LCGA. The squares represent estimates whereas the circles represent actual data. Table 4.4 provides estimates for the intercepts and slopes for each of the trajectories.

**Figure 4.** BMI trajectories by subgroups (Actual vs. Estimate)



**Table 4.4.**

Body mass index: Three class linear LCGA model estimates for intercepts and slopes

Latent Class	Intercept				Slope			
	Est.	SE	Est/SE	p-value	Est.	SE	Est/SE	p-value
Emerging overweight (n=44)	15.861	0.283	56.097	0.000	1.298	0.144	9.040	0.000
Expected growth (n=148)	14.635	0.145	101.134	0.000	0.521	0.029	18.098	0.000
Increasing obesity (n=9)	20.674	0.848	24.365	0.000	1.657	0.143	11.626	0.000

The first group comprised 21.9% of the sample (n=44). The estimated slope suggested that the average BMI of this group increased at a high rate of 1.30 ( $p<.001$ ) every two years. This group was termed the *Emerging Overweight* group because the subgroup of children started with a normal BMI but increased at a steep rate. The actual mean BMI score revealed that by age 12 (Wave 4), the average BMI of this group was 27.64 (See Table 4.5). Average BMI stayed steady and close to 30 at age 14 and 16

(Wave 5 and 6); however, by age 18, the average BMI increased to 35.19, which is considered obese by CDC definition (CDC, 2016).

The second group followed a normal trajectory, and thus was termed as *Expected Growth*. It was the largest group (73.6%,  $n=148$ ) and the estimated slope showed that the average BMI would increase by .52 every two years. The actual mean BMI of this group continued to remain in a healthy range.

The third group was the smallest, comprising 4.5% of the sample ( $n=9$ ). The estimated intercept (20.67) and slope (1.66,  $p<.001$ ) were both the highest for this group. Actual mean BMI started out with an average BMI of 20.56; the largest increase happened between age 8 (Wave 3) and age 12 (Wave 4), where the average BMI grew to 36.31. At age 18 (Wave 7), BMI increased to 43.72. Because BMI escalated, this group was labeled the *Increasing Obesity* group. Table 4.5 illustrates changes in BMI by age/Wave.

**Table 4.5.**

Actual Body mass index: Average BMI by group by age/wave

	Latent Class								
	Emerging overweight			Expected growth			Increasing obesity		
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>
Wave 1 (4yrs)	44	16.66	1.31	146	15.18	1.22	9	20.56	2.83
Wave 2 (6yrs)	42	17.47	1.58	130	15.14	1.27	8	23.84	2.77
Wave 3 (8yrs)	40	20.26	2.91	136	15.95	2.15	9	27.79	2.55
Wave 4 (12yrs)	32	27.64	3.23	111	18.26	2.83	7	36.31	4.61
Wave 5 (14yrs)	34	29.32	5.57	109	20.07	2.77	5	37.18	4.15
Wave 6 (16 yrs)	10	30.34	6.60	58	21.63	3.14	3	33.49	9.86
Wave 7 (18 yrs)	33	35.19	35.19	119	23.66	3.76	8	43.72	5.88

Posterior probabilities, which indicates how likely it is that each child belongs to one of the three groups was calculated for each child. The average posterior probability was high: .93 for *Emerging Overweight*, .98 for *Typical Growth*, and .99 for *Increasing*

*Overweight*, thereby suggesting that the individual children are very likely to belong to their assigned group.

**Aim 2: Identify the risk factors, specifically the role of preschool ACEs and school-aged ACEs, which predict group memberships for the subgroups of children following distinct BMI trajectories.**

LCGA identified three classes of growth trajectories, which were termed as *emerging overweight*, *expected growth*, and *increasing obesity*. Descriptive analyses of these groups are performed to understand the groups. To assess whether the demographic characteristics were related to class membership, bivariate analyses were run. Finally, to test whether ACEs had predictive value in determining class membership, I regressed class membership on ACEs and covariates in two separate multinomial logistic regression (MLR) analyses on the modeled classes. The first MLR model included ACEs score based on age 4 and the second MLR model included ACEs score based on ages 4 through 14. A summary of the variables entered in each of the models is provided in Chapter 3, Table 3.4. Results of all analyses are reported and significant relationships ( $p < .05$ ) as well as those that approached significance ( $p < .1$ ) are highlighted with asterisks.

#### **Characteristics of children and caregivers in each trajectory group.**

Demographic characteristics by class are described in Table 4.6. Demographic characteristics of children and primary maternal caregivers were compared based on class membership (See Table 4.7 and Table 4.8). Gender was a significant independent variable in its relationship to class membership ( $\chi^2=5.37, p=.07$ ); there were more females in the *emerging overweight* group and more males in the *expected growth* group.

Analysis of variance (ANOVA) revealed that maternal weight different by class membership ( $F = 5.20, p=.006$ ). The *emerging overweight* group had a significantly higher average maternal weight of 81.21kg, which was almost 10kg heavier than the average maternal weight of 71.55kg in the *expected growth* group and about 20 kg heavier than the average maternal weight of 61.67kg in the *increasing obesity* group. Race, whether the child was termed as failure to thrive or at risk for HIV at time of recruitment, receipt of AFDC, or primary caregiver’s education level were not significantly different by latent classes.

**Table 4.6.** Baseline characteristics of children in BMI trajectory groups (descriptive analysis) ( $n=201$ )

Variable	Total %	Emerging Overweight %	Expected Growth %	Increasing Obesity %
% based on most likely group membership	( $N=201$ )	( $n=44$ )	( $n=148$ )	( $n=9$ )
<b>Child Demographics</b>				
Gender				
Female	45.3	56.8	40.5	66.7
Male	54.7	43.2	59.5	33.3
Race				
African American	93.5	88.6	94.6	100
Other	6.5	11.4	5.4	0
Failure to thrive	33.3	29.5	35.1	22.2
At risk for HIV	24.9	20.5	25.0	44.4
<b>Caregiver Demographics</b>				
Receipt of AFDC	77.5	77.3	77.6	77.8

**Table 4.7.** Baseline characteristics of children and families in BMI trajectory groups and bivariate analyses (Chi-square)

	<b>Emerging Overweight</b>	<b>Expected Growth</b>	<b>Increasing Obesity</b>	<b>Chi-square</b>	<b>p-value</b>
<b>Child Demographics</b>	(n=44)	(n=148)	(n=9)		
Gender	%	%	%		
Female	27.5	65.9	6.6	5.37	.07*
Male	17.3	80.0	2.7		
Race					
African American	20.7	74.5	4.8	2.64	.27
Other	38.3	61.5	0.0		
Failure to thrive					
Normal Growth	23.1	71.6	5.2	1.00	.61
Failure to thrive	19.4	77.6	3.0		
At risk for HIV					
Not at Risk	23.2	73.5	3.3	2.31	.32
At risk	18.0	74.0	8.0		
<b>Caregiver Demographics</b>	(n=236)	(n=200)	(n=36)		
Receipt of AFDC					
Do not Receive	22.2	73.3	4.4	.00	1.0
Receive	21.9	73.5	4.5		

\* $p < .1$  \*\* $p < .05$

**Table 4.8.** Baseline characteristics of families in BMI trajectory groups and bivariate analyses (ANOVA)

	<b>Emerging Overweight</b>	<b>Expected Growth</b>	<b>Increasing Obesity</b>	<b>ANOVA</b>	<b>p-value</b>
<b>Maternal Demographics</b>	(n=44)	(n=147)	(n=9)		
Mother's education level (# of years)	$M=11.30$ $SD=1.34$	$M=11.40$ $SD=1.55$	$M=11.67$ $SD=1.41$	$F = .25$	.78
Maternal weight (kg)	(n=35) $M=81.21$ $SD=21.81$	(n=127) $M=71.55$ $SD=18.10$	(n=8) $M=61.67$ $SD=4.50$	$F = 5.20^*$	.006**

\* $p < .1$  \*\* $p < .05$

Preschool ACEs and school-aged ACEs were compared by classes. Regardless of the time frame encompassed, did not differ significantly by trajectory subgroups (Table 4.9). Percentage of each ACE experienced for each class is presented in Table 4.10. Bivariate analyses were conducted for different ACEs experienced by class (Table 4.11). Chi-square analyses were not conducted for some of the ACEs components, because of

the small sample size. The recommended minimum value of cell expected (row total n \* column total n / grand total n) should be 5 or more in at least 80% of the cells (Bewick, Cheek, & Ball, 2003; McHugh, 2013); oftentimes this recommendation could not be met. Furthermore, even though presence of depression in the maternal caregiver when the children were 4 years old ( $p=.07$ ), caregiver substance use at ages 4 through 14 ( $p=.09$ ), and caregiver criminal behavior at ages 4 through 14 ( $p=.06$ ) showed some trend towards significance, given the small cell size, the results should be interpreted with caution.

**Table 4.9.** Number of ACEs in BMI trajectory groups and bivariate analyses (n=201)

Variable	Total %	Emerging Overweight %	Expected Growth %	Increasing Obesity %	<i>F-test</i>	<i>p-value</i>
% based on most likely group membership	(n=201)	(n=44)	(n=148)	(n=9)		
<b>Number of ACEs</b>						
<b>Preschool ACEs</b>						
0	36.8	40.9	35.1	44.4		
1	27.9	34.1	26.4	22.2		
2	19.9	18.2	21.6	0		
3	9.0	6.8	8.8	22.2		
4	5.0	0	6.1	11.1		
5	1.5	0	2	0		
Mean, <i>SD</i>	<i>M</i> =1.22 <i>SD</i> =1.25	<i>M</i> =.91 <i>SD</i> =.94	<i>M</i> =1.30, <i>SD</i> =1.31	<i>M</i> =1.33, <i>SD</i> =1.58	1.74	.18
<b>School-aged ACEs</b>						
0	7.5	4.5	8.8	0.0		
1	14.4	20.5	12.2	22.2		
2	20.4	25.0	18.9	22.2		
3	22.9	29.5	20.9	22.2		
4	13.9	4.5	17.6	0.0		
5	9.5	11.4	8.8	11.1		
6	6.0	0.0	7.4	11.1		
7	5.5	4.5	5.4	11.1		
Mean, <i>SD</i>	<i>M</i> =3.01, <i>SD</i> =1.84	<i>M</i> =2.66, <i>SD</i> =1.63	<i>M</i> =3.09, <i>SD</i> =1.88	<i>M</i> =3.33, <i>SD</i> =2.18	1.09	.34

\* $p<.1$  \*\* $p<.05$

**Table 4.10.** Description of ACEs in BMI trajectory groups (n=201)

Variable	Total %	Emerging Overweight %	Expected Growth %	Increasing Obesity %
% based on most likely group membership	(n=201)	(n=44)	(n=148)	(n=9)
<b>Preschool ACEs</b>				
Childhood maltreatment				
Physical abuse	6.0	4.5	6.1	11.1
Sexual abuse	0.5	0	.7	0.0
Neglect	20.4	15.9	22.3	11.1
Emotional abuse	5.0	2.3	5.4	11.1
Household dysfunction				
Drink alcohol	21.4	11.4	24.3	22.2
Mother treated violently	31.3	34.1	29.7	44.4
Mother depressed	37.3	22.7	41.9	33.3
<b>School-aged ACEs</b>				
Childhood maltreatment				
Physical abuse	31.3	31.8	30.4	44.4
Sexual abuse	15.4	11.4	16.2	22.2
Neglect	33.3	22.7	37.2	22.2
Emotional abuse	40.8	38.6	39.9	66.7
Household dysfunction				
Caregiver substance use	37.3	43.2	33.8	66.7
Caregiver violence	37.8	31.8	34.2	44.4
Caregiver depression	63.2	52.3	66.9	55.6
Caregiver crime	41.8	34.1	45.9	11.1

**Table 4.11.** Description of ACEs in BMI trajectory groups and bivariate analyses (n=201)

Variable	Emerging Overweight %	Expected Growth %	Increasing Obesity %	Chi-Square	<i>p-value</i>
% based on most likely group membership	(n=44) 21.9%	(n=148) 73.6%	(n=9) 4.4%		
<b>Preschool ACEs</b>					
Childhood maltreatment					
Physical abuse	16.7	75.0	8.3		
w/o Physical abuse	22.2	73.5	4.2		
Sexual abuse	0.0	100.0	0.0		
w/o Sexual abuse	22.0	73.5	4.5		
Neglect	17.1	80.5	2.4	1.35	.51
w/o Neglect	23.1	71.9	5.0		
Emotional abuse	10.0	80.0	10.0		
w/o Emotional abuse	22.5	73.3	4.2		
Household dysfunction					
Drink alcohol	11.6	83.7	4.7		
No alcohol	24.7	70.9	4.4		
Mother treated violently	23.8	69.8	6.3		
Not treated violently	21.0	75.4	3.6		
Mother depressed	13.3	82.7	4.0	5.39	.07*
Not depressed	27.0	68.3	4.8		
<b>School-aged ACEs</b>					
Childhood maltreatment					
Physical abuse	22.2	71.4	6.3	.78	.68
w/o Physical abuse	21.7	74.6	3.6		
Sexual abuse	16.1	77.4	6.5		
w/o Sexual abuse	22.9	72.9	4.1		
Neglect	14.9	82.1	3.0	3.70	.16
w/o Neglect	25.4	69.4	5.2		
Emotional abuse	20.7	72.0	7.3	2.63	.27
w/o Emotional abuse	22.7	74.8	2.5		
Household dysfunction					
Caregiver substance use	25.3	66.7	8.0	4.75	.09*
	19.8	77.8	2.4		
Caregiver violence	18.4	76.3	5.3	.96	.62
	24.0	72.0	4.0		
Caregiver depression	18.1	78.0	3.9	3.35	.19
	28.4	66.2	5.4		
Caregiver crime	17.9	81.0	1.2	5.61	.06*
	24.8	68.4	6.8		

Chi-square values are only reported for groups with *expected cells* larger than 5

\* $p < .1$  \*\* $p < .05$

**Multinomial logistic regression to predict BMI trajectory groups.** The relationship between ACEs and other demographic factors and BMI trajectory group membership was tested by entering seven independent variables into several multinomial logistic regression (MLR) models (See Chapter 3 for a summary of the variables entered in the models). Primary maternal caregiver's weight was imputed using multiple imputation (MI). Results with MI are presented because MI allows for higher statistical power and less bias. Results of the original data without MI are presented in Appendix (See Table B.1. and B.2.).

The *expected growth* group was used as the referent group. Two separate MLR models were run to assess whether ACEs and six other demographic variables would predict membership to the BMI trajectory groups. Model 1 takes all the demographic variables into account as well as ACEs score at age 4. Model 2 also includes all the demographic variables and ACEs score for ages 4 through 14.

The first model included preschool ACEs. Compared to the *expected growth* group, children in the *emerging overweight* group were more likely to be female and have maternal caregivers who weighed more (Table 4.12). The odds of being in the *emerging overweight* group versus the *expected growth* group were 53% lower for boys than girls ( $p=.04$ ). Every one kilogram increase in the child's maternal caregiver's weight when the child is four years old was associated with a .02 increase in the odds ratio for being in the *emerging overweight* group relative to the *expected growth* group when the other variables in the model are held constant ( $p=.07$ ). Every one point increase in the preschool ACEs score was associated with a .27 decrease in the odds ratio for being in the *emerging overweight* group relative to the *expected growth* group when the other

variables in the model are held constant ( $p=.07$ ). Other covariates including, whether the child was termed as FTT or at risk for HIV, the level of education of the primary maternal caregiver, and the receipt of AFDC did not predict class membership to *emerging overweight*. Preschool ACEs score and none of the covariates predicted class membership to the *increasing obesity* group.

**Table 4.12.** Multinomial logistic regression identifying demographic factors and Preschool ACEs (4 yrs) that predict membership in BMI trajectory groups ( $n=201$ )

Variable	<i>B</i>	SE of <i>B</i>	<i>p</i> -value	Exp( <i>B</i> )	95% CI for OR (Lower bound)	95% CI for OR (Upper Bound)
<b>Emerging Overweight (<math>n=35</math>) (Ref: Expected Growth, <math>n=126</math>)</b>						
Child characteristics						
Gender (ref = female)	-0.75	0.37	0.04*	0.47	0.23	0.98
Normal growth (ref = FTT)	0.28	0.42	0.51	1.32	0.58	3.01
No HIV risk (ref = HIV risk)	0.23	0.50	0.64	1.26	0.48	3.33
Preschool ACEs	-0.31	0.17	0.07*	0.73	0.53	1.02
Parent Characteristics						
Maternal Weight (kg)	0.02	0.01	0.07*	1.02	1.00	1.05
Years of education	-0.05	0.13	0.71	0.95	0.74	1.23
Household income (ref = AFDC Receipt)	-0.10	0.45	0.82	0.90	0.37	2.19
<b>Increasing Obesity (<math>n=8</math>) (Ref: Expected Growth, <math>n=126</math>)</b>						
Child characteristics						
Gender (ref = female)	-0.92	0.75	0.22	0.40	0.09	1.72
Normal growth (ref = FTT)	0.34	0.96	0.73	1.40	0.21	9.27
No HIV risk (ref = HIV risk)	-0.52	0.87	0.55	0.60	0.11	3.24
Preschool ACEs	0.00	0.29	1.00	1.00	0.56	1.78
Caregiver Characteristics						
Maternal Weight (kg)	-0.03	0.03	0.28	0.97	0.91	1.03
Years of education	0.06	0.24	0.81	1.06	0.66	1.72
Household income (ref = AFDC Receipt)	0.22	0.91	0.81	1.25	0.21	7.43

A similar pattern was noticed in the second model where school-aged ACEs score was included (Table 4.13). The odds of being in the *emerging overweight* group versus the *expected growth* group were 51% lower for boys than girls ( $p=.05$ ) given the other variables in the model are held constant. Every one kilogram increase in the child's maternal caregiver's weight when the child is four years old was associated with a .02 increase in the odds ratio for being in the *emerging overweight* group versus the *expected growth* group when the other variables in the model are held constant ( $p=.03$ ). However,

school-aged ACEs score was not a significant predictor of class membership. Whether the child was termed as FTT or at risk for HIV, the level of education of the primary maternal caregiver, and the receipt of AFDC did not predict class membership to *emerging overweight*. School-aged ACEs score and none of the covariates predicted class membership to the *increasing obesity* group.

**Table 4.13.** Multinomial logistic regression identifying demographic factors and school-aged ACEs (4-14 yrs) that predict membership in BMI trajectory groups (n=201)

Variable	<i>B</i>	SE of <i>B</i>	<i>p</i> -value	Exp( <i>B</i> )	95% CI for OR (Lower bound)	95% CI for OR (Upper Bound)
<b>Emerging Overweight (n=35) (Ref: Expected Growth, n=126)</b>						
Child characteristics						
Gender (ref = female)	-0.72	0.36	0.05*	0.49	0.24	0.99
Normal growth (ref = FTT)	0.35	0.42	0.40	1.42	0.63	3.21
No HIV risk (ref = HIV risk)	0.38	0.48	0.43	1.46	0.57	3.74
School-aged ACEs	-0.12	0.10	0.24	0.89	0.73	1.08
Parent Characteristics						
Maternal Weight (kg)	0.02	0.01	0.03*	1.02	1.00	1.04
Years of education	-0.03	0.13	0.84	0.98	0.76	1.25
Household income (AFDC Receipt)	0.01	0.44	0.99	1.01	0.42	2.39
<b>Increasing Obesity (n=8) (Ref: Expected Growth, n=126)</b>						
Child characteristics						
Gender (ref = female)	-0.91	0.75	0.23	0.40	0.09	1.75
Normal growth (ref = FTT)	0.36	0.96	0.71	1.43	0.22	9.34
No HIV risk (ref = HIV risk)	-0.45	0.85	0.60	0.64	0.12	3.40
School-aged ACEs	0.03	0.19	0.89	1.03	0.71	1.48
Caregiver Characteristics						
Maternal Weight (kg)	-0.03	0.03	0.19	0.97	0.92	1.02
Years of education	0.05	0.25	0.85	1.05	0.65	1.70
Household income (ref = AFDC Receipt)	0.20	0.90	0.82	1.22	0.21	7.15

The findings confirmed, with caution ( $p$  value was .07) that the study hypotheses that preschool ACEs would be indicative of class membership in the *emerging overweight* trajectory vs. the *expected growth* trajectory. School-aged ACEs score did not, however, predict class membership. Gender and maternal average weight when the children were four years old also predicted membership in *emerging overweight* trajectory to *expected growth* trajectory.

## Chapter 5. Discussion

The current study identified three BMI growth trajectories among primarily African American, high-risk children residing in an urban, low-income area. These trajectories are compared to those identified in other research studies. Additionally, sensitive periods of vulnerability to obesity are identified and discussed. Gender of the child, average maternal caregiver weight, and ACEs when the children were four years old were predictive of membership in the *emerging overweight* trajectory, compared to the *expected growth* trajectory. These results are discussed below in relation to prior research and aforementioned theories. Strengths and limitations of the current study, implications for practice, policy, research, and theory, and finally, conclusion, are presented.

### Identification of Distinct BMI Trajectories of Children

In this study, LCGA, a growth mixture modeling approach, was used to identify distinct BMI trajectories between the ages of 4 and 18. The sample was a predominantly low income, mostly African American, high-risk group of children from inner city Baltimore, Maryland. As predicted, the majority of children (73.6%) followed an *expected growth* trajectory, where their BMI increased at a steady rate and edged up to overweight; about a third fell into the overweight/obese category by age 18. However, the average BMI fell in the normal range at age 18. *Emerging overweight* was the second largest group (21.9%) and though the average BMI at age 4 was normal, it increased at a steep rate over time. Lastly, there was a small group termed *increasing obesity* (4.5%) whose BMI started high and increased over time.

The current findings that portray three distinct trajectories are similar to results from other studies that track BMI growth in children. Though studies have identified anywhere from three to five trajectories, the majority of studies appear to report three distinct subgroups of growth trajectories (e.g., Balistreri & Van Hook, 2011; Li, Goran, Kaur, Nollen, & Ahluwalia, 2007; Pryor et al., 2011; Pryor et al., 2015). In all of the studies that examine BMI growth, the largest subgroup is the “normal” or “never overweight” trajectory that parallels the current study’s *expected growth* group. Similar to the *emerging overweight* group, other studies have also found what they termed as the “gradual onset of overweight” (Balistreri & Van Hook, 2011) or the “increased probability of overweight” group (Stuart & Panico, 2016). There are some variations of this category depending on whether the authors place emphasis on the degree of weight gain or the time frame of the weight gain. The *increasing obesity* group that came about in the current study reflected the “always overweight” or “high rising” group identified in other studies (Pryor et al., 2015; Stuart & Panico, 2016). A trend that was not distinguished in the current study was a group that started out with a high BMI and decreased to a healthy range. This was not an alarming outcome given that the study sample was prone to start with a low BMI, with a significant percentage of children being categorized as failure to thrive.

### **Ages Prone to Weight Gain**

For both the *emerging overweight* and *increasing obesity* classes, the largest positive change in average BMI occurred between ages 8 and 12 and 16 and 18. There may be sensitive periods of growth when some children are prone to rapid weight gain. Hormonal changes and puberty may account for some of this change (Daniels, 2016).

Other studies have identified time periods of development that appear more sensitive to weight gain. Li et al. (2007) identified a “late onset overweight” group where the largest increase in BMI occurred between 6 years to 10 years of age. Although there is a shortage of studies that examine BMI changes specifically during late adolescence (Yang, Turk, Allison, James, & Chasens, 2014), national trend data suggests that this transitional period from adolescence to adulthood is a sensitive time period for gaining weight (Gordon-Larson, Adair, Nelson, & Popkin, 2004; Ogden et al., 2006). These findings call for research and practice implications, which are discussed below.

### **Divergence of Trajectory**

The trajectories show that the children show divergence in their growth trajectory in early childhood (ages 6 and 8), suggesting that their growth trajectories may be determined rather early. Other researchers have similarly identified an “early onset overweight” group (Li et al., 2007) and in addition, early-childhood obesity has been found to be disproportionately prevalent among low-income, minority, urban children (Freedman et al, 2006; Stettler et al., 2005). This finding suggests that perhaps positive dietary and activity behaviors should be modeled and taught in early childhood to prevent obesity (Perryman, 2011).

### **Significant Predictors of *Emerging Overweight* Group**

**Gender.** Compared with males, females were more prone to be in the *emerging overweight* group compared to the *expected growth* group. This is unsurprising because studies have consistently found a higher obesity rate among African American females compared to their male counterparts as well as compared to White females (Fryar, Carroll, & Ogden, 2014). Ogden et al. (2014) also reported that the data from the National Health

and Nutrition Examination Survey (NHANES) showed that though obesity rates among African American and White girls between the ages of 2 and 11 were similar, by adolescence and adulthood, there was a racial discrepancy; the rate of obesity increased at a significantly higher rate among African American girls than among White girls. By 19 years, 42.5% of African American females were obese whereas 33.8% of White girls were obese (Ogden et al., 2014). Similarly, in the current study, 38.0% of girls were obese and an additional 19.0% of girls were overweight.

Another explanation could be related to the children's socioeconomic backgrounds. For example, Balisteri and Hook (2011) found that among girls, having parents with less education or lower socioeconomic status increased the risk of being in the "gradual onset of overweight group." On the other hand, for boys, only parental education, but not parental income, was related to a reduction in obesity risk. The authors speculated that the explanation for this phenomenon is complex, entailing gender and parenting practices; for example, girls from lower SES backgrounds may experience decreased pressure to maintain a healthy weight. Because the current study sample was also from a disadvantaged community in terms of SES, more females than males could have been prone to belong in a latent class that experienced steeper weight gain over time.

**Maternal Weight.** Another significant predictor of being in the *emerging overweight* group compared to the *expected growth* group was maternal weight as recorded when the children were four years old. A higher maternal weight was predictive of membership in the *emerging overweight* group. This is consistent with findings in Pryor et al. (2015); they reported that having two overweight parents (OR = 7.29) or having one overweight parent (OR = 2.22) were significant predictors of class

membership in the “early onset overweight” group compared to the “never overweight” group. Li et al. (2007) also indicated that having a high maternal BMI is one of the most notable risk factors for early-onset obesity among a racially diverse group of children who were followed from younger than 2 years of age to the ages of 12 and 13. The linkage between parent obesity and child obesity is further corroborated by the ecobiodevelopmental theory (Shonkoff et al., 2012) and studies that recognize that the combination of genetic disposition and family environmental factors, such as the parents’ level of knowledge and ability to support a healthy diet and positive lifestyle choices, may influence the children’s obesity outcomes (Anderson & Whitaker, 2010).

Unexpectedly, maternal weight was not predictive of group membership to the *increasing obesity* group compared to the *expected growth* group. In fact, the average maternal weight for this group was the lowest ( $M=61.67$  kg,  $SD=4.50$ ). The reason is difficult to explain and given the small sample size pertaining to this analysis, the findings should be interpreted with caution.

### **ACEs**

I hypothesized that based on the cumulative risk model (Sameroff et al., 1987) and the stress and coping model (Lazarus & Folkman, 1984), a higher number of ACEs would portend a high risk BMI growth trajectory group. On the contrary, having a higher preschool ACEs (4 years) led to a lower chance of being in the *emerging overweight* group relative to the *expected growth* group. The finding should be interpreted with caution because the  $p$  value was .07. School-aged ACEs (4-14 years) did not predict class membership.

One explanation for this finding of the relationship between ACEs at 4 years and obesity trajectory group pertains to what is considered ‘statistically significant’. I used a more lenient  $p$  value to assess multivariate relationships ( $p < .1$ ) because statisticians have noted that setting statistical significance at .05 is somewhat arbitrary; in addition, they admonish readers about assuming  $p > .05$  as a confidence statement in support of the null hypothesis or a  $p < .05$  as a proof of an effect (Gelman 2013). However, Gelman and Hill (2007) also warn about drawing conclusions when interpreting results with  $p$  values between .05 and .1, especially when the direction of the relationship is counter to the expected direction.

Additionally, Gelman and Hill (2007) discuss the importance of assessing practical significance because statistical significance and clinical significance do not always equate. The current study found that there was not a significant difference in the average number of preschool ACEs by groups ( $p = .18$ ). Furthermore, 100% of the *emerging overweight* group and the majority of the *expected growth* group (91.7%) had ACEs scores between 0 and 3. Thus, I cannot state with assurance that the relationship between ACEs and problematic BMI trajectory is indeed present. Replication of research is warranted to draw more confident conclusions.

An alternative explanation can be considered by recognizing the uniqueness of the study sample. Data from National Health and Nutrition Examination Survey (NHANES) of 1999 to 2012 demonstrated that non-Hispanic blacks had higher prevalence rates of overweight and obesity (Skinner & Skelton, 2014). Possibly, black children are prone to belonging to a problematic BMI trajectory group, where they experience rapid weight gain. Black children who remain in the steady and normal BMI growth group may be the

ones following an atypical growth trajectory. It could be that ACEs could impede food procurement for the children. Food insecurity in low-income, urban neighborhoods generally means that the children will feed on cheap, high-caloric, non-nutritious food which often leads to an increased risk for obesity; however, in more dire circumstances, children may not have access to sufficient food, which then lowers their risk of gaining weight (Cook et al., 2004). Under such assumption, a higher number of ACEs can act as a factor against overweight.

The second operationalization of school-aged ACEs (4-14 years) may not have predicted group membership for a variety of reasons. Though the link between ACEs and negative health outcomes has been strongly established (Reilly & Kelly, 2005; Karnik & Kanekar, 2012), there have also been some studies that did not find such a link. There were two studies that examined adult data to assess the relationship between ACEs and obesity. Campbell, Walker, and Egede (2015) examined adult data (18 years and above) from five states in the Behavioral Risk Factor Surveillance System (BRFSS) survey to assess whether ACEs occurring prior to 18 years impact a variety of risky health behaviors and morbidity measures. One of the outcome variables was obesity and the authors found that ACEs scores were not associated with obesity or diabetes. Wade et al. (2016) also found that in a sample of urban adult residents (18 years and above) residing in Philadelphia, PA, the relationship between ACEs scores based on those that occurred from 0 years to 18 years of age and obesity or diabetes was not significant. Perhaps for children from urban, minority environments, as depicted in the current study and the Philadelphia study, ACEs do not predict obesity because the level of exposure to ACEs is similar. For instance, for the current study, even though the range of ACEs 4-14

years was wide, the majority of the children (79.1%) had ACEs scores between 1 and 4. Overall, this finding adds to a select number of studies that did not find a significant relationship between ACEs and obesity.

### **Strengths and Limitations**

The strengths of this study are several. Foremost, the study employed a longitudinal prospective design with a large span of years. Previous studies that investigated the relationship between early adversities and obesity either used retrospective data to capture early adversity (Shin & Miller, 2012) or did not utilize an extensive longitudinal data set (Burke et al., 2011; Heerman, Krishnaswami, Barkin, & McPheeters, 2016). The current study measured ACEs prospectively, meaning that the information was collected during the child's childhood to capture potentially traumatic events in the child's life. Many ACEs studies, including the original CDC study, rely on a retrospective report of ACEs and are thus vulnerable to recall bias. As for the BMI data points, there were a total of seven waves of data that ranged from early childhood to late adolescence. Finkelhor et al. (2013) recommends that more longitudinal studies that monitor children from childhood through adulthood are needed in order to better understand the relationship between ACEs and long-term health outcomes. As a result of the multiple time points, the study offered an excellent opportunity for analyzing longitudinal data (Singer & Willet, 2003).

In addition to data collection at multiple time points, the study had a relatively large sample size, which allowed the author to perform LCGA and multinomial regression analyses. For LCGA analysis, Nagin (2005) urges at least 100 cases, and preferably 300 to 500 cases. For multinomial logistic regression, a minimum of 10 cases

per independent variable (Hosmer, Lemeshow, & Sturdivant, 2013; Peduzzi, Concato, Kemper, Holford, & Feinstein; 1996) or a sample larger than 100 (Pampel, 2000) are recommended. The current study sample size was adequate to meet these criteria.

Another strength of the study lies on the measurement of the ACEs. The current study looked at the cumulative value of preschool and school-aged ACEs. Both were examined because researchers debate about the timing of ACEs (occurring in early childhood, later childhood, or adolescence) and its potential linkage to negative health and mental health outcomes. For instance, Appleyard et al. (2005) reported that adversity occurring in early childhood was strongly related to problems in adolescence and adulthood rather than those occurring in later years, but Flaherty (2013) asserted that adversities occurring in early adolescence were more strongly related to health problems than those occurring in earlier years.

The study sample was composed of primarily lower income, African American children. This is very different from the sample that was used for the original CDC-ACEs study, which was mainly middle class Caucasians. Examining the relationship between ACEs and BMI in a minority population adds to the current state of the literature.

Finally, the study used an advanced statistical technique (LCGA) that allows various shapes of BMI growth over time to be estimated without forcing a polynomial shape to the data. Through FIML, LCGA makes use of all available data to create trajectories. Researchers have found that when it came to detecting linear growth trajectories, LCGA performed the best out of *K*-means clustering, a two-step approach with mixed modeling and *K*-means clustering, latent class growth analysis, and latent class growth mixture modeling (Twisk, 2014; Twisk & Hoekstra, 2012).

Limitations must also be acknowledged. First, because the study assessed BMI over a lengthy period of time and multiple data collection points, there was attrition in the study. Precautions were taken by setting inclusion criteria (having at least two data points in early childhood and at least one data point in adolescence) and by using a statistical method (LCGA) that accounts for “missing data at random” through its procedure.

LCGA allowed estimation of growth trajectories, but the comparison between actual and estimated growth showed some discrepancy. Twisk and Hoekstra (2012) cautioned that all classification methods, such as LCGA, should be applied and interpreted with caution because of the uncertainty that goes in the class assignment. Hence, additional studies would have to corroborate the findings.

Another statistical limitation was regarding the sample size of the *increasing obesity* group. Because there were only 9 children in the *increasing obesity* group, bivariate analyses were either not performed or were interpreted with caution (McHugh, 2013).

There are limitations that rise from the operationalization of ACEs. Some of the variables in the ACEs could not be measured over the whole time period. For example, maternal depression was assessed over a period of a week instead of over a longer time period. In addition, ACEs does not allow to capture the chronicity of each adversity. Because questions regarding drinking behavior, depression, and violence in the home were based on caregiver report, the caregivers may have been inclined to succumb to social desirability and underreport.

Though the distinctiveness of the study sample can be considered a merit, it can also be considered a weakness due to its potentially limited generalizability. The sample was limited to families within one metropolitan area on the East coast of the U.S. Additionally, the sample composition was unique with three different groups of people; the first had signs of FTT in infancy, the second were at risk for HIV because of the mother's substance use, and the third had similar demographic characteristics, but were without FTT nor HIV risk. The sample was also predominantly African American and from lower socioeconomic background. Although the study findings cannot be generalized to the larger U.S. population, this particularly vulnerable minority group may have divergent trajectories of growth and different factors that influence growth, thus deserving attention to their experience of ACEs and how that may influence obesity outcomes.

## **Implications**

**Practice and policy implications.** Social workers work closely with families whose children have been exposed to traumatic experiences. Most children who are involved with child welfare have experienced at least one adversity and many have multiple, prolonged, and complex trauma histories (Ko, Ford, Kassam, Adams, Berkowitz, Wilson, & Wong, 2008). As professionals interested in promoting the well-being of children (National Association of Social Workers [NASW], 2013), social workers need to be on the forefront of understanding factors, such as ACEs, that impact children's physical and psychosocial outcomes to intervene effectively. Even though the study findings did not show a positive correlation between ACEs and obesity risk, the results do not necessarily advocate against screening for ACEs. Because the state of the

literature is still inconclusive with regards to the relationship between ACEs and obesity, social workers should still continue to be aware of the relationship between ACEs and various health and mental health outcomes, so that they can help caregivers to recognize how the family environment can affect their children's current and future behaviors as well as physical/emotional states.

In addition to conducting assessments, evaluating progress, and providing psychological and parenting support, child welfare workers are encouraged to collaborate with professionals from multiple disciplines, such as health, mental health, education, and in the juvenile justice system (Babyak & Koorland, 2001; Emshoff et al., 2007; Hernandez & Hodges, 2006; Lewandowski & GlenMaye, 2002) to better meet the complex needs of children and families (Alkema et al., 2003; Lewandowski & GlenMaye, 2002; Marett et al., 1998; McElheran et al., 2004; Murphy et al., 2005). An increased understanding of BMI trajectories and time periods when children are more prone to weight gain as well as factors that predisposed them to a higher chance of being overweight can enable the social workers to ensure that the children receive adequate healthcare, communicate health risks, and support recommendations by health professionals. At the same time, in spite of the interdisciplinary effort there are silos between health and social workers (Knickman et al., 2016) and bridging that gap for development of more effective prevention and intervention strategies to combat obesity should be advocated.

There are also those otherwise involved with children's services, such as foster parents and mental health clinicians, who often work with parents and caregivers to build parenting skills so that parents and caregivers can provide nurturing and responsible care

to their children. One area of family-based interventions may be related to improving health care for children, for instance, creating a healthy lifestyle by making adjustments in nutrition and activity. Some of the behavior changes include offering nutritious food, setting consistent meal times, rewarding positive behaviors without using food, engaging in physical activities, and limiting sedentary behaviors (Brotman et al., 2012; Perryman, 2011). Altogether, social workers may be able to partake in the prevention and intervention of obesity.

Individual efforts to prevent obesity are important but policy level interventions should not be overlooked. Shapes of growth trajectories in the current study showed that the divergence in an *emerging overweight* or *increasing obesity* group happens in early childhood. Children who have higher BMIs in early or middle childhood could be candidates for a higher level of monitoring, for instance, rather than annual check ups they could have biannual check ups. Tracking BMI in pediatric primary care is encouraged to monitor and intervene with those who are prone to obesity (Flower, Perrin, Viadro, & Ammerman, 2007; Vine, Hargreaves, Briefel, & Orfield, 2013). Education and linkage to resources and support to prevent further weight gain should be provided accordingly.

The findings also call for intervention efforts to focus on young children and more specifically at a community level. Silva-Sanigorski and colleagues (2010) examined the utility of a community-based obesity intervention that focused on capacity building and environmental changes to promote healthy eating and active play in children aged 0-5 years. Though long term impact was not accessed, short term positive changes in eating behaviors and BMI were seen (Silva-Sanigorski et al., 2010). A systematic review of

community-based childhood obesity prevention studies also summarized that community level interventions with a school component and a focus on both diet and physical activity may yield moderately positive outcomes in reducing overweight and obesity (Bleich, Segal, Wu, Wilson, and Wang, 2013). This evidence, together with the current study findings, suggests that obesity interventions should take place when the children are young and possibly at a community level. As Bleich et al. (2013) underlined as well, even if interventions have a modest effect on reducing obesity, community interventions have the potential to yield significant public health benefits because of its cumulative nature.

Another result of this study was that having a higher number of ACEs is not related to being in a more problematic trajectory group and in fact, could lower the risk of belonging to a problematic BMI growth group. Moreover, most children experience few ACEs (based on the current study's operationalization of ACEs) and even in the current study sample almost 85% experienced low ACEs (0-2). This finding then raises the question of focusing on identifying level of ACEs as a part of obesity prevention and intervention. The study findings point to considering other variables, such as the demographics of target population (e.g., gender and race) or family components (e.g., primary maternal caregivers who are obese) to be considered when developing weight management interventions.

Researchers and health care professionals have discussed the necessity to implement interventions that are specifically targeted towards African American females because of the disparity in prevalence of obesity as well as limited effectiveness of weight reduction interventions (Barr-Anderson, Adams-Wynn, DiSantis, & Kumanyika,

2013). When weight reduction interventions are administered to children aged 5 to 18, a systematic review found that African American females are less likely to lose weight compared to White females (Barr-Anderson et al., 2013). Researchers should continue to place efforts on finding interventions for this group of vulnerable African American youth, perhaps especially during the ages of sensitive growth (8-12 years) identified by the trajectories.

The current study findings that high maternal weight is predictive of a child following an overweight trajectory suggest that perhaps there is a genetic and familial disposition to weight gain. Perhaps obesity intervention needs to happen as soon as the time of conception for mothers who are already overweight. This ties into the current literature that posits that obesity interventions should take place during the child's first 1,000 days of life- conception through age 36 months. The first 1,000 days is considered critical in building a healthy weight trajectory; increasing evidence suggests that factors inherent to the pregnant mother as well as behaviors that are perpetuated by caregivers, such as mother's BMI at pregnancy, prenatal tobacco exposure, gestational diabetes, curtailed infant sleep, improper timing of solid food, and overuse of antibiotics in infants may foster obesity among children (Baidal, Locks, Cheng, Blake-Lamb, Perkins, & Taveras, 2016; Stettler, 2007). A recent systematic review also supports the efficacy of obesity intervention for mothers during their pregnancy and in the child's early life stage (Blake-Lamb, Locks, Perkins, Woo Baidal, Cheng, & Taveras, 2016). Blake-Lamb et al. (2016) found that effective interventions focus on individual or family level behavioral changes through home visits, individual or group counseling, a combination of home and group visits in a community setting, and using hydrolyzed protein formula. Efforts to

target and educate African American mothers right from when they learn that they are pregnant may reduce the number of children that follow a high-risk overweight trajectory.

**Research implications.** One area of future research surrounds the operationalization of ACEs. There is contention among researchers regarding what factors should be included as ACEs in studies. The current study replicated ACEs as defined by the original CDC-ACEs study as well as the LONGSCAN operationalization of ACEs. However, researchers, such as Curtis, Fuller-Rowell, Doan, Zgierska, and Ryff (2016) contend that a more thorough list of adversities should be examined. This idea of expanded ACEs may be especially important with a socioeconomically and racially diverse sample. Cronholm et al. (2015) compared rates of conventional ACEs (based on the CDC-ACEs study) and expanded ACEs (included community level ACEs, such as witnessing violence, living in an unsafe neighborhood, experiencing bullying, and a having a history of living in foster care) among adults from different racial backgrounds. They found that minority groups would differ in their rates of ACEs when the expanded ACEs was used compared to the conventional form (Cronholm et al., 2015). The authors emphasized that a broader range of adversities must be used for different demographic groups when doing research with regards to ACEs, especially considering that the original CDC-ACEs study was developed for middle-class Whites.

The operationalization of ACEs is also important, because depending on what factors are included in the ACEs scale, findings may vary. For instance, Curtis et al. (2016) incorporated 21 childhood adversity items that addressed household dysfunction, childhood maltreatment, crime and violence exposure, family environment dynamics, and illness in the family; though they did not find a significant association between ACEs

score and obesity, they found that there was an association between having class III obesity ( $BMI \geq 40 \text{ kg/m}^2$ ) and having four or more ACEs. The relationship was stronger for Whites than Blacks and women more than men (Curtis et al., 2016). Hence, future research should consider having a more comprehensive operationalization of ACEs and examine the relationship between ACEs and obesity.

Because the study sample was limited in generalizability, there is a need for additional studies that examine the impact of ACEs on obesity risk in different populations. In addition, the analytical nature of the current study also calls for more research. There are limitations to classification analyses (Twisk & Hoekstra, 2012) and replication would allow researchers to draw more confident conclusions.

On the other hand, based on the study findings that children with a higher preschool ACEs score were in fact at a lower risk of belonging to the *emerging overweight* growth trajectory group and that school-aged ACEs score did not have predictive value in belonging to the *increasing obesity* growth trajectory group, the author questions the utility of conducting more research to assess ACEs without expanding the ACEs operationalization, as a risk factor for obesity in underserved African American children. Both the inconsistency in the current state of literature regarding the relationship between ACEs and obesity and the findings of the current study, which employed a prospective design as well as rigorous analytical methods, suggests that researchers focus on examining other factors that impact obesity in high risk African American youth. Whether it is the family or lifestyle component, as evidenced by the link between increased primary maternal caregiver's weight and belonging to the *emerging overweight* group compared to the *expected growth* group, or the cultural

component of being female in a predominantly African American community, research and future interventions could benefit more by focusing on factors in addition to ACEs. This recommendation would be even stronger if additional studies that examine the relationship between ACEs and obesity fail to find a definitive linkage.

Some other areas that future studies should take into account include eating behaviors and physical activity levels to examine changes in weight status (Carlson, Crespo, Sallis, Patterson & Elder, 2012) that are modifiable. Environmental factors such as food insecurity (Kaur & Ogden, 2016), food desserts (Cummins, 2016), school endorsement of positive eating and exercising behavior (Williams, Mesidor, Winters, Dubbert, & Wyatt, 2015) should also be included in research to guide the development of forthcoming obesity intervention policies for youth.

## **Conclusion**

The current study examined BMI trajectories in a sample of high-risk children from an urban, lower-income, mainly African American community in Baltimore, Maryland. The children's growth was followed from age 4 to 18 (7 data collection points). About one in five followed a trend towards obesity and 1 in 20 followed a consistently obese trajectory from 4 years all the way up to 18 years. The majority of the children followed a relatively normal growth trajectory, though even in this group, a third of the individuals belonging to this group were overweight/obese at 18 years. The current study found that certain developmental periods are more prone to significant weight gain. Additionally, being female, having a primary maternal caregiver who is overweight, and having a lower preschool ACEs score increased the risk of belonging in

the *emerging overweight* group. These findings should be taken into account when devising interventions and policies to combat obesity.

## Appendix

**Table A.1. Literature review of growth trajectories**

<b>Literature Review on Growth Trajectories</b>				
<b>Name, year</b>	<b>Population, setting, sample, age range</b>	<b>Design and statistical analysis</b>	<b>Measures</b>	<b>Trajectories identified</b>
Balistreri and Van Hook (2011)	<p>Early Childhood Longitudinal Study-Kindergarten Cohort (ECLS-K)</p> <p>N=14000</p> <p>Males: 59% White, 20% Hispanic, 13% Black, 3% Asian, 5% Other</p> <p>Females: 58% White, 20% Hispanic, 14% Black, 3% Asian, 5% Other</p> <p>7 data points: twice in Kindergarten and 1<sup>st</sup> grade, once in 3<sup>rd</sup>, 5<sup>th</sup>, 8<sup>th</sup></p>	Semiparametric mixture models (SPMM)	BMI percentile	<p>Normal (59% boys, 55% girls) (Consistently normal weight whereby BMI percentile remained below the 85<sup>th</sup> percentile)</p> <p>Gradual onset (15% boys, 20% girls) (Gradual onset of overweight/at risk or overweight)</p> <p>Always at risk (27% boys, 25% girls) (high probability of sustained overweight or at risk of overweight from Kindergarten through 8th grade)</p>
Chen et al. (2016)	<p>Southeast Texas elementary school system (45 elementary schools) 2005 kindergarten class</p> <p>n=1651</p> <p>11 data points, biannually from kindergarten to the beginning of 5<sup>th</sup> grade</p> <p>50% male, 32% White, 23% Black, and 23% Hispanic</p>	Group-based trajectory model	BMI percentile	<p>Persistently non-overweight/obese weight (51.1 %) (low probability of being in the overweight/obese category)</p> <p>Chronically overweight/obese (21.8%) (high risk of being overweight/obese across)</p> <p>Early-onset overweight/obese (9.2 %) (low obesity risk in Kindergarten, but increased risk summer after Kindergarten)</p> <p>Late-onset overweight/obese (9.7 %) (healthy weight range from Kindergarten through 2nd Grade Spring, but increased risk in the summer after 2<sup>nd</sup> grade through 4<sup>th</sup> grade)</p> <p>Becoming healthy weight (8.2 %) (low obesity risk in Kindergarten, but tended to increase their probability of overweight/obesity in the summer after Kindergarten)</p>

				Chronically overweight/obese (21.8 %)
Garden, Marks, Simpson, & Webb (2012)	<p>Childhood Asthma Prevention Study (CAPS), Sydney, Australia (children at high risk for asthma)</p> <p>Born between 1997 and 2000, n=370</p> <p>51% male, 49% female,</p> <p>Data collection at 1, 3, 6, 9, 12, 18 months, and every 6 months thereafter until 5 years, and at 8 and 11.5 years.</p>	Latent basis growth mixture model	BMI percentile	<p>Boys: Normal BMI (61%) (tracked along the 50th percentile)</p> <p>Early and persistent increase in BMI (12%) (75th percentile at 2 years and continued to increase to be above the 95th percentile at 11.5 years)</p> <p>Late increase in BMI (27%) (50th percentile from birth to 5 years increased to the 85th percentile at 8 years and the 90th percentile at 11.5 years)</p> <p>Girls: Normal BMI (62%)</p> <p>Early and persistently high BMI (12%) (increased to the 95th percentile at 3 years and reduced to be at the 85th percentile at 11.5 years)</p> <p>Late increase in BMI (26%) (50th percentile from birth to 2 years at which time the BMI increased to the 85th percentile at 8 years and the 95th percentile at 11.5 years)</p>
Lane, Bluestone, Burke (2012)	<p>National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD)</p> <p>10 communities in the United States</p> <p>Data collection at 2, 3, 4.5, ~6, ~8, ~10, ~11 years</p> <p>1,238 children (51% male, 49% female), 76% White, 13% African American, 6% Hispanic, 5% Other</p>	Latent growth mixture model	BMI percentile	<p>Stable (38.8%) (average BMI percentile of 51% at 24 months and steady)</p> <p>Steady increase (36.7%) (increased in BMI percentile at 36 months and leveled off near the 70th percentile by age 8)</p> <p>Elevated (24.5%) (average BMI percentile at 24 months was 57%, increased through the preschool years, leveling off just above the 70th percentile by kindergarten)</p>
Li, Goran, Kaur, Nollen, &	National Longitudinal Survey of Youth 1979 (NLSY79)	Latent growth mixture	BMI	Normal/never-overweight (83.9%) (Low probability of overweight)

Ahluwalla (2007)	<p>Born between 1984 and 1990 and followed from 2 to 12 years old</p> <p>n=1739</p> <p>53.9% male, 46.2% female, 77.8% white, 15.3% black, 6.9% Hispanic</p> <p>Data collection every 2 years</p>	model		<p>throughout childhood and were never overweight)</p> <p>Early onset overweight (10.9%) (Early onset of overweight that persisted throughout childhood)</p> <p>Late onset overweight (5.2%) (Moderately high probability of overweight at age 2 years, low probability of overweight at age 4 and 6 years, but growing probability of overweight after age 8 years)</p>
Nonnemaker, Morgan-Lopez, Pais, & Finkelstein (2009)	<p>National Longitudinal Survey of Youth 1979 (NLSY79)</p> <p>Born between 1984 and 1990</p> <p>Age at first data collection between 12 and 17, data collected annually for 6 years</p> <p>n=1739</p> <p>53.9% male, 46.2% female, 77.8% white, 15.3% black, 6.9% Hispanic</p>	General growth mixture model (GMM)	BMI	<p>High risk (at high risk for becoming obese by young adulthood, 67% obese at 12 and 90% obese at 23 years)</p> <p>Moderate-to-high risk (55% obese at 12 and 68% obese at 23 years)</p> <p>Low-to-moderate risk (8% obese at 12 and 27% obese at 23 years)</p> <p>Low risk (few obese at any age during this developmental period)</p> <p>*Note: percentages of youth belonging to each class unavailable.</p>
Pryor et al (2011)	<p>Quebec Longitudinal Study of Child Development</p> <p>Born between Oct, 1997 and July, 1998</p> <p>n = 1957</p> <p>9 data points: 5 months, yearly till 8 years</p> <p>49.7% female, 93% White</p>	Group-based developmental trajectories	BMI	<p>Low-stable (54.5%)</p> <p>Moderate (41.0%) (slightly higher average BMI at each data point than low-stable)</p> <p>High-rising (4.5%) (similar pattern from 5 months to 2.5 years to the other groups, but between 3.5 and 8 years, this group increased in BMI, reaching a mean BMI of 24 at 8 years)</p>
Stuart & Panico (2016)	<p>The Millennium Cohort Study</p> <p>United Kingdom</p> <p>Born between 2000-2002</p> <p>n=9699</p>	Group-based trajectory modeling	BMI	<p>Low normal (44.8%) (remained in a normal range with an average BMI of 16)</p> <p>Mid normal (37.8%) (higher average BMI at age 3 than 'low normal' but remained below overweight)</p>

	data points: 3, 5, 7 and 11 years			<p>Overweight (14.4%) (their BMI continues to increase and BMI maintains above the overweight cutoff but below the obese cutoff.)</p> <p>Obese (3.1%) (were obese at 3 years and continued to increase in BMI)</p>
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**Table B.1.** Multinomial logistic regression identifying demographic factors and ACEs (4 years) that predict membership in BMI trajectory groups without imputed data

Variable	B	SE of B	<i>p-value</i>	Exp(B)	95% CI for OR (Lower bound)	95% CI for OR (Upper Bound)
<b>Emerging Overweight (<i>n</i>=35) (Ref: Expected Growth, <i>n</i>=126)</b>						
Child characteristics						
Gender	-1.10	0.43	0.01*	0.33	0.14	0.76
FTT	0.23	0.47	0.63	1.26	0.50	3.19
HIV	-0.15	0.55	0.79	0.86	0.30	2.53
Preschool ACEs	-0.26	0.19	0.17	0.77	0.54	1.12
Parent Characteristics						
Maternal Weight (kg)	0.03	0.01	0.01*	1.03	1.01	1.05
Years of education	-0.06	0.15	0.72	0.95	0.71	1.27
Household income (AFDC Receipt)	0.05	0.49	0.92	1.05	0.40	2.74
<b>Increasing Obesity (<i>n</i>=8) (Ref: Expected Growth, <i>n</i>=126)</b>						
Child characteristics						
Gender	-0.64	0.78	0.41	0.53	0.11	2.43
FTT	0.02	1.05	0.98	1.02	0.13	8.06
HIV	-0.97	0.97	0.32	0.38	0.06	2.55
Preschool ACEs	0.04	0.31	0.89	1.04	0.57	1.91
Parent Characteristics						
Maternal Weight (kg)	-0.03	0.03	0.23	0.97	0.92	1.02
Years of education	0.14	0.27	0.60	1.15	0.68	1.93
Household income (AFDC Receipt)	0.31	0.94	0.74	1.36	0.22	8.55

**Table B.2.** Multinomial logistic regression identifying demographic factors and ACEs (4-14 years) that predict membership in BMI trajectory groups without imputed data

Variable	B	SE of B	<i>p-value</i>	Exp(B)	95% CI for OR (Lower bound)	95% CI for OR (Upper Bound)
<b>Emerging Overweight (<i>n</i> =35) (Ref: Expected Growth, <i>n</i>=126)</b>						
Child characteristics						
Gender	-1.08	0.42	0.01*	0.34	0.15	0.78
FTT	0.28	0.48	0.55	1.33	0.52	3.37
HIV	-0.03	0.53	0.96	0.98	0.34	2.78
School-aged ACEs	-0.10	0.11	0.35	0.90	0.73	1.12
Parent Characteristics						
Maternal Weight (kg)	0.03	0.01	0.01*	1.03	1.01	1.05
Years of education	-0.04	0.15	0.77	0.96	0.71	1.28
Household income (AFDC Receipt)	0.14	0.48	0.77	1.15	0.45	2.96
<b>Increasing Obesity (<i>n</i>=8) (Ref: Expected Growth, <i>n</i>=126)</b>						
Child characteristics						
Gender	-0.64	0.78	0.41	0.53	0.11	2.45
FTT	0.00	1.06	1.00	1.00	0.13	7.96
HIV	-0.97	0.96	0.31	0.38	0.06	2.51
School-aged ACEs	0.04	0.20	0.83	1.04	0.71	1.53
Parent Characteristics						
Maternal Weight (kg)	-0.03	0.03	0.24	0.97	0.92	1.02
Years of education	0.14	0.26	0.59	1.15	0.69	1.93
Household income (AFDC Receipt)	0.32	0.94	0.73	1.38	0.22	8.71

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