CHILD EATING IN THE ABSENCE OF HUNGER AND LOSS OF CONTROL EATING

by

Carrie Suzanne Sheets

B.A. in Psychology, Kalamazoo College, 1998

M.S. in Psychology, University of Pittsburgh, 2004

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This dissertation was presented

by

Carrie Suzanne Sheets

It was defended on

May 13, 2011

and approved by

Michele Levine, PhD, Assistant Professor, Psychiatry and Psychology

Anna Marsland, PhD, RN, Associate Professor, Psychology

Robert Noll, PhD, Professor, Psychiatry and Psychology

Michael Pogue-Geile, PhD, Associate Professor, Psychology

Dissertation Advisor: Marsha Marcus, PhD, Professor, Psychiatry and Psychology
Laboratory studies in children have found that, following a self-reported satiating meal, girls who consume relatively large amounts of palatable food in the absence of hunger have a higher BMI and gain more weight across middle childhood than do girls who consume less. Loss of control eating (LOC) in children has been associated with increased BMI, aberrant eating, and general psychopathology in cross-sectional and longitudinal research. Eating in the absence of hunger (EAH) appears to be a behavioral analogue of the dietary disinhibition that is a feature of LOC. However, no studies of which we are aware have examined the relationship between a laboratory measure of EAH and LOC. Additionally, the majority of studies examining EAH have been conducted in homogenous samples comprised primarily of non-Hispanic White girls. Thus, the aims of this study were to replicate the EAH paradigm in a racially diverse sample, evaluate the relationship between BMI and EAH, and examine whether EAH consumption was associated with self-reported LOC in a sample of 51 Black and White girls age 10-13 years. Results indicated that girls consumed an average of 339.7 ($SD = 283.3$) calories in the absence of hunger. EAH intake was not associated with race or BMI. EAH intake predicted LOC, such that for each additional 100 calories consumed, girls were 1.7 times more likely to report LOC. Longitudinal research is needed in younger children to evaluate the nature of EAH as an early behavioral marker of risk for LOC and its sequelae. Additional research is also needed to examine the relationship between mood and EAH in children with LOC. Findings from the current study
suggest that EAH may be used to identify young children at risk for development of aberrant eating and weight problems. Future studies should identify young children who consume large amounts during the EAH procedure and design interventions aimed at reducing eating that is in response to environmental cues.
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PREFACE

Graduate school has been a long journey, and I am very thankful that I had such a wonderful mentor, Dr. Marsha Marcus, to help guide me along the way. She has been such an incredible teacher, and I will be forever grateful for the lessons she taught me about being a better clinician, scientist, and person. I am extremely grateful for the guidance and support of Drs. Melissa Kalarchian and Michele Levine, who provided invaluable feedback on this project and on countless other occasions throughout my graduate career. I would also like to thank my remaining dissertation committee members, Drs. Anna Marsland, Robert Noll, and Michael Pogue-Geile, for sharing their expertise and encouragement over the years.

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1.0 INTRODUCTION

The prevalence and severity of pediatric obesity have increased at alarming rates over the last two decades. Approximately 34.7% of American children ages 6 through 19 years are overweight (≥ 85th percentile of Body Mass Index (BMI) for children of the same age and sex) and 18.7% are obese (≥ 95th percentile of BMI (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010). Pediatric obesity is associated with significant medical (Dietz, 1998; Freedman, Dietz, Srinivasan, & Berenson, 1999; Must, Jacques, Dallal, Bajema, & Dietz, 1992) and psychiatric morbidity (Brown et al., 1998; Eddy et al., 2007; Kimm, Sweeney, Janosky, & MacMillan, 1991; Lumeng, Gannon, Cabral, Frank, & Zuckerman, 2003; Manus & Killeen, 1995; Strauss & Pollack, 2003), including weight-related distress and disordered eating attitudes and behaviors (Eddy et al., 2007; Goldschmidt, Passi Aspen, Sinton, Tanofsky, & Wilfley, 2008; Ranzenhofer et al., 2008; Tanofsky-Kraff et al., 2004; Young-Hyman et al., 2006). The emergence of disordered eating behavior may begin as early as middle childhood (Tanofsky-Kraff, 2008) and is predictive of subsequent obesity (Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice, Presnell, & Spangler, 2002). Similarly, pediatric obesity is a critical childhood risk factor for development of eating disorders (Fairburn et al., 1998; Fairburn, Welch, Doll, Davies, & O'Connor, 1997). Given that pediatric obesity tends to persist into adulthood (Serdula et al., 1993; Whitaker, Wright, & Pepe, 1997), early identification and intervention with problematic eating and overweight in children appears essential.
Efforts to understand pathways implicated in the development and maintenance of pediatric obesity have examined specific eating behaviors that may promote excess energy intake in children and be putative risk factors for the development of overweight and disordered eating. In particular, research efforts have focused on a type of child eating behavior observed in an experimental condition, called “Eating in the Absence of Hunger (EAH).” EAH refers to the amount of palatable snack food a child consumes in a laboratory setting after reporting satiation following a standard meal. Examination of EAH is appealing because it is clearly operationalized, is observable as early as age 3, and can be easily measured in a laboratory setting. EAH has significant relevance to the understanding of pediatric obesity because longitudinal research in demographically homogeneous samples of White girls ages 5-13 years indicates that it is a robust predictor of increases in BMI and is a relatively stable construct within subjects over time (Birch, Fisher, & Davison, 2003; Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Shunk & Birch, 2004).

EAH was first observed as part of a line of research examining parental attitudes and beliefs related to their control over their child’s eating environment, called “parental feeding.” EAH has been associated with mothers’ attitudes and behaviors related to feeding their daughters, such that relatively higher EAH intake is related to greater maternal control and restriction of daughters’ food intake (Birch & Fisher, 2000; Birch, Fisher, & Davison, 2003; Fisher & Birch, 1999b, 2002). Additional evidence indicates that more controlling and restrictive parental feeding practices are positively related to girl’s energy intake (Birch & Fisher, 2000), food preferences (Birch, 1998; Fisher & Birch, 1999b), and body weight (Birch & Fisher, 2000; Francis & Birch, 2005). The majority of research on this topic has been conducted by Birch and colleagues, who hypothesize that parental control over child feeding may
paradoxically lead to the development of children’s overeating and obesity by promoting the child’s use of external cues to regulate his or her energy intake, rather than learning to stop eating on the basis of internal satiety cues (Birch & Fisher, 1998, 2000). Given the ability to identify EAH when children are young and the potential for modifying child eating and parental feeding behavior, research on these constructs has promising implications for the development of pediatric obesity treatments.

The significance of Eating in the Absence of Hunger in relation to disordered eating is unclear. Investigators suggest that intake during the free access procedure may be a behavioral analogue to the construct of dietary disinhibition found in adults (Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Stunkard & Messick, 1985). Disinhibited eating has been characterized as eating that occurs in response to factors other than hunger and satiety, such as the presence and smell of palatable food or emotional states. This potentially problematic eating behavior is associated with weight status and gain (McGuire, Wing, Klem, Lang, & Hill, 1999; Williamson, Lawson, Brooks, & Wozniak, 1995), weight fluctuations (Carmondy, Brunner, & St. Jeor, 1995), and binge eating in adults (Howard & Porzelius, 1999) and is thought to be produced, in part, by self-imposed rigid restrictions placed on eating (Polivy & Herman, 1985; Westenhoefer, Stunkard, & Pudel, 1999). Dietary disinhibition, defined as eating in response to external cues including emotional stressors, and the sight or odor of foods, has been reported by 9- to 12-year old children (Braet & Van Strien, 1997). Furthermore, child self-report data suggests that EAH is often precipitated by negative affect and external food cues (Tanofsky-Kraff et al., 2008). In a review of the literature on binge eating in children and adolescents, Marcus and Kalarchian (2003) suggest that, given the relationship between disinhibited eating and binge eating, EAH may be a useful marker of risk for aberrant eating in children.
Binge eating is defined as the consumption of an unusually large amount of food in a circumscribed period of time that is coupled with a feeling of loss of control over the eating (Spitzer et al., 1993). Eating episodes are classified as either: 1) Objective Binge Episodes (OBEs), defined by the consumption of an objectively large amount of food and a feeling of loss of control (LOC) over eating, or 2) Subjective Binge Episodes (SBEs), defined by the consumption of what the respondent considers to be a large amount of food and a feeling of LOC over eating. Thus, both episodes are characterized by the feeling that one is unable to control how much he or she is eating; however, only OBEs are considered “true” binge episodes for the purpose of diagnosing Binge Eating Disorder (BED). Findings in samples of children and adolescents indicate that binge eating episodes are not uncommon, particularly among girls, are observable in both Black and White children, and can be identified in middle childhood (Croll, Neumark-Sztainer, Story, & Ireland, 2002; Decaluwe & Braet, 2003; Field et al., 1999; Morgan et al., 2002; Shisslak et al., 2006; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2004).

Although children and adolescents often report binge eating, it is typically with less frequency than required to meet criteria for BED (Tanofsky-Kraff, 2008; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). Studies assessing BED in children typically find low rates of the disorder (Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). There are several possible reasons for these low prevalence rates of BED. There is poor concordance between child and parent reports of binge eating (W. G. Johnson, Grieve, Adams, & Sandy, 1999; Tanofsky-Kraff, Yanovski, & Yanovski, 2005) and even between child self-reports compared to child interviews (Field, Taylor, Celio, & Colditz, 2004; Tanofsky-Kraff et al., 2003). Furthermore, determining what constitutes a “large amount of food” in growing children with
changing nutritional needs can be difficult (Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). Researchers have suggested that binge eating may manifest itself differently in children relative to adults (Marcus & Kalarchian, 2003; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). Given that many of the assessment measures of binge eating are developed for adults and subsequently modified for use in children, they may not adequately capture disordered eating in children.

In a study of 367 youth (Shomaker et al., 2010a), children who endorsed OBE or SBE episodes did not differ in their disordered eating attitudes, emotional eating, self-reported eating in the absence of hunger, depressive and anxiety symptoms, or adiposity, and they also reported higher rates of these symptoms relative to those without OBE or SBE episodes. Other research in adults (Niego, Pratt, & Agras, 1997; Pratt, Niego, & Agras, 1998) and children (Morgan et al., 2002; Tanofsky-Kraff et al., 2004) suggests that it is the loss of control (LOC), as opposed to the amount eaten, that is most salient for identifying those with disordered eating. For these reasons, many investigators focus on episodes of loss of control eating (i.e., eating with the associated experience of being unable to control how much food one is eating independent of whether the amount consumed is objectively large) rather than objective binge episodes. Investigators have proposed different criteria to define binge eating and loss of control eating in children that does not include the criterion of objectively large episodes necessary for BED diagnosis in adults (Marcus & Kalarchian, 2003; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). Thus, the term “Loss of Control eating” as opposed to binge eating, is often adopted when working with children and adolescents in order to be inclusive of all episodes involving LOC (Shomaker et al., 2010a), and was the focus of the present study.
Evaluation of binge eating and loss of control eating episodes in children and adolescents is important for several reasons. LOC episodes are associated with greater eating-related psychopathology, negative mood, anxiety, and depressive symptoms (Decaluwe, Braet, & Fairburn, 2003; Glasofer et al., 2007; Goldschmidt et al., 2008a; Goossens, Braet, & Decaluwe, 2007; Morgan et al., 2002; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2004). They are also associated with obesity in school-aged children (Shisslak et al., 1998; Tanofsky-Kraff et al., 2004). Furthermore, LOC is a prevalent behavior among overweight youth that is marked by physical and psychosocial impairment (Tanofsky-Kraff, 2008; Tanofsky-Kraff et al., 2009c) and exacerbates the course and treatment of overweight (Levine, Ringham, Kalarchian, Wisniewski, & Marcus, 2006; Tanofsky-Kraff et al., 2009c). Thus, LOC has considerable implications for various aspects of children’s physical and emotional functioning.

Fisher and colleagues (2007) suggest that although EAH may capture behavioral aspects of disinhibition, the loss of control over eating ascribed to disinhibited eating among adults may involve emotional and cognitive as well as behavioral components (Lowe & Maycock, 1988). Recent research suggests that, relative to youth who deny loss of control, youth who self-report loss of control over eating are more likely to report eating despite a lack of hunger (Tanofsky-Kraff et al., 2007a). A recent study found that children who scored higher on a self-report measure of EAH were more likely to endorse previous episodes of LOC (Tanofsky-Kraff et al., 2008). However, no studies of which we are aware have assessed whether a behavioral measure of eating in the absence of hunger is associated with loss of control over eating. Therefore, one goal of this study was to evaluate whether loss of control episodes are associated with greater EAH among girls.
Findings from several studies examining EAH in boys and girls suggest that the correlates of EAH vary according to child gender (Faith et al., 2006; Fisher & Birch, 1999a, 2000; Hill et al., 2008). Because of the documented sex differences in EAH, the proposed study will include only girls. Furthermore, the relationship between parental weight and behaviors and child disordered eating appears to be particularly pertinent for mothers and their daughters. Mothers’ weight, weight concerns, beliefs, and input related to child weight may differentially affect children’s eating and weight-related attitudes and behaviors relative to that of fathers (Davison & Birch, 2001; Jacobi, Agras, & Hammer, 2001; Smolak, Levine, & Schermer, 1999; Stice, Agras, & Hammer, 1999). Thus, the current study collected data from mothers and female guardians only.

To date, the majority of research has examined EAH in fairly homogeneous samples comprised of predominantly middle-to-upper class, normal weight, non-Hispanic White girls living with both biological parents. Epidemiological evidence indicates that Black girls have significantly higher prevalence of overweight and obesity relative to their White peers (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010; Wang & Beydoun, 2007). This disparity between Black and White girls increases during adolescence and throughout adulthood (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010; Ogden et al., 2006).

Research also suggests that there are racial differences in the demographic, physiological, developmental, and psychosocial correlates of obesity between White and Black girls. For example, lower rates of obesity among White girls are associated with higher levels of income and parental education; however, there is no clear relationship between socioeconomic variables and obesity among Black girls (Kimm et al., 1996). Findings from the NHLBI Growth and Health Study indicate that eating disorder symptoms in Black and White girls vary according to
child race, age, body weight, and parental education (Striegel-Moore et al., 2000). Beginning around the age of 10, Black girls begin to show more advanced pubertal maturation and increases in adiposity than do their White cohorts (Kimm et al., 2001). Furthermore, 9- and 10-year-old Black girls report greater satisfaction with their own body build and physical appearance than do White girls, regardless of their BMI. Their preferred body build and expected adult body size are significantly larger than their White peers (Ghee, 1990).

A study of overweight children found that associations of psychological status, weight, and weight-related distress differed for Black and White children (Young-Hyman et al., 2006). Although overweight is associated with poorer quality of life (QOL) for both White and Black adolescents, Black youth report less overall QOL impairment, as well as impairment in different domains of functioning relative to their White peers (Fallon et al., 2005). In light of the racial differences in the developmental pattern and correlates of obesity and its sequelae between Black and White girls, the existing research on child EAH in Whites may not necessarily generalize to Black girls. Given Black girls’ significant vulnerability to obesity and the need to develop more effective and culturally-appropriate pediatric obesity treatments, examination of EAH in Black girls is essential. Thus, evaluating EAH in a diverse sample comprised of White and Black girls was another goal of the current study.
2.0  BACKGROUND

2.1  EATING IN THE ABSENCE OF HUNGER

The initial experiments examining child EAH, as well as the majority of subsequent research in this area, have been conducted by Birch and colleagues. Initial studies focused on examining the relationships between mothers’ behaviors and attitudes about child feeding and children’s eating behavior. In a series of two experiments with children ages 3 to 6 years, Fisher and Birch (1999b) examined the effects of restricting access to a palatable target food for 5 weeks on children’s subsequent behavioral responses to, selection of, and intake of that target food. Results indicated that restricting access to the target food increased children’s behavioral response to the restricted food. Relative to the highly similar control food, children made more spontaneous comments and behaviors regarding the restricted food, indicating an increased preference post-restriction. Also, restricting access to the palatable food increased children’s selection and intake of that food relative to other times when the food was freely available. Children’s responsiveness to restriction in the experimental setting was positively associated with mothers’ reported restriction of the experimental food at home, and negatively associated with the frequency with which mothers reported purchasing the food. These initial findings suggested that maternal restriction of children’s access to palatable foods may paradoxically increase children’s preferences for and intake of those foods.
Given the potential implications of these results, additional studies were conducted to examine feeding practices and their associations with children’s eating behavior. During these experiments, Birch and colleagues observed children eating in a manner they subsequently referred to as “Eating in the Absence of Hunger (EAH).” EAH is the number of calories from palatable snack foods that children consume following a self-reported satiating meal. An experimental paradigm, called the free-access procedure, has been used to assess individual differences in EAH. The procedure is detailed below, followed by a review of key findings on EAH.

The procedure begins with children consuming a self-selected ad libitum lunch before the free-access session. Immediately after lunch, each girl gives a subjective measure of hunger level by selecting one of three cartoon figures depicting “hungry,” “half-full,” and “full.” Girls who eat very little during lunch or who selected “hungry” or “half-full” after lunch are not included in analyses. Next, the children perform a rank-order preference assessment of each snack food to be used in the free-access procedure. They taste a small portion of each food and then place them in front of cartoon faces depicting ”yummy,” “yucky,” or “just ok.” Ten savory or sweet snack foods are presented, including: popcorn, potato chips, pretzels, nuts, fig bars, chocolate chip cookies, fruit-chew candy, chocolate bars, ice cream, and frozen yogurt. Following the preference assessment, the child is left alone in a room with toys and generous portions of each snack food and told that she can play with the toys and eat any of the foods while the experimenter does some work in the adjacent room. The experimenter leaves the room for 10 minutes and observes the child through a one-way mirror. Following the 10 minute free-access session, the experimenter returns to the room and completes additional assessments with
the child. EAH is calculated as the number of kilocalories consumed during the 10-minute free-access session.

2.1.1 Review of EAH studies by Birch research group

The first studies to use the free access procedure examined the relationship between parent feeding, maternal aberrant eating (e.g., dietary restraint, disinhibition), and child eating and weight in a sample of 3- to 6-year-old children (Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Fisher & Birch, 1999a). Results indicated that maternal restriction was positively correlated with child adiposity, parental dietary restraint, girls’ perceptions of restriction, and greater consumption of these restricted foods during the free access procedure. Greater calorie intake during the free-access procedure (i.e., EAH) was associated with girls’ weight-for-height ratios, as well as maternal disinhibition (Cutting, Fisher, Grimm-Thomas, & Birch, 1999). Both mothers’ disinhibition and daughters’ EAH intake were independent predictors of daughters’ obesity, and together accounted for nearly half of the variance in daughters’ weight-for-height; thus, in addition to daughters’ own EAH intake predicting obesity, maternal disinhibition was a predictor of daughters’ obesity.

Birch and colleagues then conducted a series of experiments examining EAH in a longitudinal community sample comprised of 197 non-Hispanic White girls, assessed at ages 5, 7, 9, 11, and 13 years. At the start of the study, the girls tended to be within the normal weight range (weight-for-height = 62.2 ± 2% for age and sex), from fairly well-educated, middle- to upper-class families with both biological parents living in the home. Throughout the course of the study, parents were administered the following measures: the Child Feeding Questionnaire, Three Factor Eating Inventory (measures the parent’s own dietary restraint, disinhibition, and
hunger), Restricted Access Questionnaire (measures the degree to which the parent restricts child access to foods used in the free-access procedure), and BMI. Girls completed the free-access procedure at each time point, as well as measures of their perceptions of parental restriction, feelings of negative self-evaluation about eating experimental foods, body dissatisfaction, dietary restraint, 24-hour energy intake, and BMI. Additionally, girls completed a laboratory measure called COMPX, which measures the extent to which girls’ short-term energy intake is responsive to the energy density of foods.

When the girls were assessed at age five (Birch & Fisher, 2000), structural equation models indicated that mothers’ own dietary restraint and perceptions of their daughters’ risk of obesity predicted more controlling maternal feeding practices, which in turn predicted their daughters’ relative weight and less adequate short-term control of energy intake (a combination of COMPX and EAH scores). Additionally, 50% of the girls reported “eating too much” and 44% reported a negative emotion related to eating at least one or more of the snack foods (Fisher & Birch, 2000). Approximately one-third of the girls indicated that they would feel bad if their mother (30%) or father (37%) found out what they had eaten. Parental restriction predicted both child EAH intake, as well as girls’ negative self-evaluation of eating. Interestingly, girls’ perceptions of eating “too much” snack food and negative self-evaluation were not related to the amount of snack foods consumed, but rather to their perceptions of whether their parents allowed them to have the snack foods. Thus, results from this study suggest that girls’ perceptions of overeating and negative feelings about their snack food intake were more closely related to parents’ reports of restriction and girls’ perceptions of restriction than to the amount of restricted foods they actually consumed.
Longitudinal findings with this cohort at ages 5 and 7 years indicated that EAH was moderately stable during that two-year period, and greater EAH intake was associated with the increased likelihood of girls’ obesity at both timepoints (Birch, Fisher, & Davison, 2003). Parental restriction when the child was 5-years-old predicted EAH at age 7, independent of both BMI and EAH at age 5 years. Thus, findings suggest that EAH is stable across time, associated with increased risk of obesity, and predicted by parents’ report of restrictive feeding practices.

Several studies reported outcomes when sample participants were between the ages of 5 and 9 years. When grouping participants into either obese or not obese at age 5 years (obese defined as ≥75th percentile BMI), maternal restriction was associated with increases in girls’ EAH in the entire sample (Fisher & Birch, 2002). However, the 5-year-old girls who were already obese and experienced higher levels of restriction showed the greatest EAH at 9 years and the largest increases in EAH from 5 to 9 years. Similar to findings regarding the association between maternal feeding and child BMI (Faith et al., 2004; Powers, Chamberlin, van Schaick, Sherman, & Whitaker, 2006), this study suggests that the effects of early restriction appear to be greatest among girls who are already obese at age 5. Girls who ate relatively large amounts of food in the absence of hunger at age 5 years were almost four times as likely to eat large amounts of food at age 7 years compared to girls who ate relatively little in the absence of hunger at age 5 years. The girls who ate relatively large amounts of food in the absence of hunger at both ages were at increased risk of being overweight at 5 and 7 years of age.

Another study grouped children according to whether their mothers were obese or not when the children were 5 years old (Francis & Birch, 2005). Obese and non-obese mothers did not differ in the level of restriction they used. However, obese mothers’ high restriction when their daughters were 5 years old predicted daughters’ EAH over the following four years, and
higher EAH intake was associated with greater BMI change from age 5 to 9 years. These relationships did not exist for daughters of non-obese mothers. Thus, findings suggest that both child and maternal weight status may affect the relationship between feeding practices and child weight outcomes, within a sample of primarily normal weight girls.

The most recent study with this cohort examined whether parental obesity status and disinhibited overeating were predictive of greater increases in girls’ BMI and EAH intake between the ages of 5 to 13 years (Francis, Ventura, Marini, & Birch, 2007). Girls were divided into three groups: those having two obese parents, one obese parent, and no obese parents. Results indicated that girls with two obese parents had the most rapid increases in BMI from 5 to 13 years and were eight times more likely to be obese at age 13, controlling for BMI at age 5. Additionally, girls with two obese parents had the highest levels of EAH across all ages relative to the other groups. Although EAH intake increased over time in the entire sample, girls with two obese parents had the largest increases relative to all other groups. With respect to the relationship between parent and child disinhibited eating, mothers’ disinhibited eating was not significantly related to EAH when daughters were age 5 or 7 years; however, it was correlated with daughters’ EAH at ages 9, 11, and 13 years. Fathers’ disinhibited eating was not related to daughters’ EAH at any time point.

2.1.2 Review of other EAH literature

Given the potential implications of Birch and colleagues’ research to the understanding of the development, maintenance, and treatment of pediatric obesity, other researchers began studying Eating in the Absence of Hunger. A study by Fisher and colleagues (2007) used a family design to examine the heritability of EAH and its association with obesity among Hispanic boys and
girls ages 5 to 18 years. After consuming an ad libitum dinner, children completed the free-access procedure, and EAH was computed as a percentage of total energy expenditure. There were no effects of sex or age on EAH, but obese children consumed 14% more energy in the absence of hunger than did non-obese children. Both ad libitum dinner intake and EAH showed heritabilities of 50%, suggesting considerable additive genetic and/or shared familial environmental influences. When expressed as a percentage of total energy expenditure, EAH did not vary with age, suggesting that age-related increases in energy intake were proportional to the children’s capacity.

Another study with this cohort (Butte et al., 2007) found that EAH predicted weight gain over one year; however, the effect became non-significant once baseline BMI and child age, sex, and Tanner stage were taken into account. During the free-access portion, children consumed an average of 389 kcal ($SD = 224$). These studies offer cross-cultural evidence that EAH is observable from early childhood through adolescence and associated with obesity in a sample of Hispanic children. However, little is known about whether previously documented findings regarding child EAH will replicate in Black children.

A number of other studies have examined EAH with equivocal findings. Several studies evaluated the relationships between EAH and risk for obesity based on parental body weight. Faith and colleagues (2006) examined EAH in 5-year-old children at low or high risk for obesity based on maternal prepregnancy body weight. Child EAH was measured using the Birch laboratory paradigm, albeit with three modifications: all children were encouraged to eat until they felt full, an evening meal, instead of lunch, was provided, and the study was conducted in a hospital-based laboratory, rather than a university-based preschool setting. In order to measure parental restriction of each snack food used in the free-access session, parents were asked six
questions regarding the degree to which they purchase and limit child access and consumption of the food. Findings indicated that there was no difference in EAH scores between high- and low-risk girls. Parents of high-risk children were more likely than were low-risk parents to limit how often they purchased the snack foods for their home. There was a trend for parental limitations on the frequency of child consumption of snack foods at home to be associated with greater EAH. High-risk boys consumed significantly more calories in the absence of hunger, but EAH did not differ between low-risk and high-risk girls. This sex difference suggests that variability in EAH may be more prone to environmental influences for girls relative to boys.

When this cohort was reassessed at age 13 (N = 31), the mean energy intake during the EAH portion for the entire sample was 293 ± 43 kcal, which amounted to an intake of 137 ± 20% of participants’ daily discretionary caloric allowance (Kral et al., 2010). Low-risk girls consumed 2.5 times more EAH calories than did high-risk girls and twice as many calories as high-and low-risk boys when intake was expressed as a percentage of daily allowance for discretionary calories. A similar pattern was identified when dividing participants by current weight status, as normal-weight females consumed 2.5 times more EAH calories than did obese girls and normal-weight males. The authors suggested that the changes in this cohort may be attributable to developmental differences in eating behavior before and after puberty, or they may be a function of sex differences in social desirability or dieting behavior.

Other studies have conducted the free-access session in group settings. Hill and colleagues (2008) found similar results in two additional studies of EAH in 7-12 year old girls and boys, one of which was conducted in a group setting at school and the other in the homes of twin pairs. Boys consumed more EAH calories than did girls during the free-access session, thus analyses were run separately for boys and girls. Among boys, EAH intake was positively
associated with adiposity after adjusting for age, ethnicity, neighborhood SES level of deprivation or maternal education, and self-reported appetite, hunger, and mood, with a linear increase in EAH intake across underweight, “healthy” weight, and overweight groups. There was no association between adiposity and EAH for girls in either study; however, in the school-based study there was a quadratic trend with EAH increasing through the underweight and “healthy” weight ranges and decreasing in the overweight and obese groups. Notably, children participated in the free-access portion in a group with their classmates (in the school-based study) or together with their siblings (in the twin study); thus, their intake was potentially observable by at least one other person. The absence of an association between weight and EAH among girls in these studies may be due to overweight girls limiting their intake in an effort to meet perceived social expectations regarding eating behavior.

Moens and Braet (2007) examined EAH in a Belgian sample of 26 overweight treatment-seeking youth and 26 normal weight age-and gender-matched controls. The free-access procedure was conducted in the participating child’s home, and was initiated after the study observer arrived and the participating parent (usually the mother) prepared a typical dinner. Following dinner, a camera was installed in a separate room, the child was given time to habituate to the camera, and then rated his or her satiety on a 10-point scale. Small amounts of ten different sweet or salty snacks were placed in the room in separate packages of approximately 100 calories each, and children were allowed to play with toys and eat the snacks while the observer left the room for 20 minutes. EAH was examined as a binary variable, with children grouped according to whether or not they consumed any snacks during the free-access session. In the normal weight group, 89% of the girls ate the presented snacks, which was significantly higher than the 56% of overweight girls who consumed snacks. In both weight
groups, the same percentage of boys (63%) consumed snacks. The children who did not consume any snacks reported a higher level of satiety than did those who consumed snacks following dinner. On average, normal weight and overweight girls consumed a similar number and amount of snacks. In contrast, obese boys consumed twice as many snack foods relative to their normal weight peers. Lower weight status, higher composite scores on a measure of emotional and external eating, and lower level of satiety predicted a greater likelihood of eating snacks during the free-access session; however, maternal BMI, SES, child gender, restrained eating, and parental modeling and feeding did not significantly contribute to the model’s prediction.

Similar to other studies in which children were ostensibly aware that their EAH intake was being monitored, overweight girls were less likely to consume calories during the free-access session. It is possible that the presence of a camera affected the child’s intake due to social desirability biases (Gardner, 2000) that likely disproportionately affect girls relative to boys (Hill et al., 2008). Additionally, the overweight children were seeking weight loss treatment, which may have affected their intake of snack foods during free-access. Finally, data suggest that providing relatively larger food portions can lead to increases in energy intake (Ello-Martin, Ledikwe, & Rolls, 2005); thus, presenting smaller portions of snack foods in premeasured portions may have limited intake.

Overweight youth have the capacity to consume more at meals than their nonoverweight peers (Ebbeling et al., 2004; Jansen et al., 2003) and may therefore require greater energy intakes, relative to energy requirements, to achieve satiety. Shomaker and colleagues (2010b) suggested that since the Birch EAH paradigm appeared to be designed to reduce, rather than eliminate, hunger, it may be that overweight children simply eat more overall as opposed to eating more in the absence of hunger relative to their nonoverweight peers. Because the energy
intake required to reach satiety differs among individuals, they posited that a standard meal may not ensure the absence of hunger among participants of varying weights. Thus, they tested EAH consumption in 13-17 year-old adolescents following 1) a lunch meal standardized to provide 50% of the child’s daily estimated energy requirements based on age, sex, and BMI, and 2) a lunch meal comprised of a very large array (>10,000 kcal) of lunch-type foods. On average, participants consumed 861 ± 30 kcal at the standardized meal and 1309 ± 55 kcal at the large-array meal. Adolescents reported significantly less hunger following the large-array meal compared to the standardized meal. EAH intake following the standardized lunch was associated with intake following the large-array lunch; however, participants ate an average of 70 kcal less during the free-access session following the large-array (295 ± 18 calories) compared to the standardized meal (365 ± 20 calories). After controlling for age, sex, race, pubertal stage, and meal intake, BMI z score and overweight status were positively associated with EAH consumption and intake during both meals. Thus, a considerable body of research suggests that eating when not physiologically hungry may contribute to excess body weight (Shunk & Birch, 2004).

Although no studies have examined the effects of emotional state on EAH in laboratory studies of children, several studies have examined the effect of acute stress on EAH consumption in adult samples. Rutters and colleagues (2009) found that following a mathematical task designed to induce an acute stress state, participants consumed more calories from sweet foods and more total energy than they did in the control condition. Differences in energy intake between stress and control conditions were a function of increases in state anxiety scores during the stress task, an effect that was more pronounced in those with high disinhibition scores. The differences in state anxiety scores were a function of trait anxiety, suggesting that acute
psychological stress is associated with EAH, especially in vulnerable individuals characterized by disinhibited eating and sensitivity to chronic stress. A related study in nine normal weight adult females found that intake of carbohydrates and proteins in the absence of hunger was greater and satiety was rated lower in a stress condition relative to a rest condition (Born et al., 2010). Previous findings of increased intake of fats during stress may not have been replicated because this sample was comprised of primarily nonoverweight women. There appears to be a genetic basis for some of these differences, as polymorphisms in the BcII genotype, which have been previously associated with insulin resistance and increased BMI, waist-hip ratio, and leptin levels, were correlated with increased sensitivity to psychological stress and increased EAH after stress (Rutters et al., 2010).

Given the results from studies on mood inductions described above and other research documenting the role of emotional and external cues as potential triggers for overeating (Van Strien, Schippers, & Cox, 1995), Tanofsky-Kraff and colleagues (2008) developed a questionnaire to assess EAH in children called the EAH-C. Specifically, the 14-item measure assessed the frequency of eating either past satiation or initiating eating in the absence of hunger in response to each of the precipitants captured by the following subscales: 1) Negative Affect (sad or depressed; anxious or nervous; angry or frustrated); 2) External precipitants (sensory cues such as appearance, taste, or smell of food; and social cues such as being around others who are eating); and 3) Boredom or Fatigue. To evaluate its psychometric properties, 226 obese and non-obese youth completed the EAH-C, along with measures of LOC eating, general psychopathology (depression and anxiety), and emotional eating in response to: 1) depression, 2) anger, anxiety, and frustration, and 3) feeling unsettled.
Notably, LOC and scores on each subscale of the EAH-C and the emotional eating questionnaire were all positively correlated with each other. All correlations remained significant when examining boys and girls separately, as well as non-obese children and obese children, except the relationship between EAH-C Boredom/Fatigue and the emotional eating measure of Anger, Frustration, and Anxiety became a trend for obese children. These relationships between EAH-C and the emotional eating measure varied according to race, as EAH-C Boredom/Fatigue was related to a measure of emotional eating in response to anger, frustration, and anxiety for White youth but not for Black youth. The relationship between anxiety and EAH-C Negative Affect and EAH-C External Eating also differed according to race. Results indicated that obesity was associated with higher scores on the negative affect subscale of the EAH-C, and higher EAH-C scores were associated with more symptoms of anxiety and depression among girls. Finally, after controlling for sex, children 12 years and under with LOC had significantly higher EAH-C Boredom/Fatigue scores relative to those without LOC. Thus, this study provided preliminary evidence of a relationship between a self-report measure of EAH and child-reported LOC episodes (Tanofsky-Kraff et al., 2008). Furthermore, results suggested that eating in the absence of hunger in response to emotional, as opposed to external, cues may show a greater association with excess weight. Finally, findings also indicated that correlates of EAH may vary according to race.

In summary, a considerable body of research has examined Eating in the Absence of Hunger, particularly in samples of primarily normal weight, non-Hispanic White girls from middle-to-upper class families. Research on EAH indicates that this behavior can be observed as early as 3 years of age, correlates with child BMI and adiposity in most studies, is relatively stable over time, and is associated with more restrictive maternal feeding practices and greater
maternal dietary restraint and disinhibition. Additionally, findings suggest that self-reported EAH is associated with emotional eating and loss of control eating. EAH is often conceptualized as a behavioral indicator of disinhibited eating and is one of the behavioral pathways implicated in the etiology of pediatric obesity. Available evidence suggests that eating large amounts of food in the absence of hunger can be distinguished from a more normative tendency to show some responsiveness to environmental cues. Given that EAH is associated with obesity development, is identifiable when girls are young, and child eating behavior is potentially modifiable, it presents a potentially promising target for pediatric obesity prevention and treatment programs.

Several questions regarding EAH remain unanswered. The first is whether the same relationships between child weight and EAH found in homogeneous samples of White girls will exist in a more socioeconomically diverse sample of Black and White girls. Additionally, the degree to which EAH may be an early behavioral marker of susceptibility to eating-related psychopathology and disinhibition is unknown. Dietary disinhibition, or eating in response to external cues, including emotional stressors, or the sight or odor of foods, has been reported by 9- to 12-year-old children (Braet & Van Strien, 1997). Children who report episodes of binge eating also endorse high scores on measures of disinhibited eating (i.e., eating in response to cognitive, environmental, and affective stimuli), suggesting that they are disproportionately affected by external food cues (Berkowitz, Stunkard, & Stallings, 1993; Mirch et al., 2006). In a review of the literature on binge eating in children and adolescents, Marcus and Kalarchian (2003) suggest that, given the relationship between disinhibited eating and binge and LOC eating, EAH may be a useful marker of risk for aberrant eating in children. However, it is unknown whether EAH is associated with LOC.
2.2 LOSS OF CONTROL EATING

Binge eating is defined as the consumption of an unusually large amount of food in a circumscribed period of time that is coupled with a feeling of loss of control over the eating (Fairburn & Wilson, 1993; Spitzer et al., 1992; Stunkard, 1959). Recurrent binge eating without the regular compensatory behaviors seen in bulimia nervosa is a cardinal feature of binge eating disorder (BED), a research diagnostic category in the 4th edition of Diagnostic and Statistical Manual of Mental Disorders (APA, 2000). As indicated earlier, LOC eating denotes episodes characterized by the feeling that one is unable to control what or how much one is eating, regardless of the actual amount of food consumed. The Eating Disorder Examination (EDE; the child version is termed the ChEDE) clinical interview is considered the gold standard measure of binge eating and entails classification of various types of eating episodes, including Objective Binge Episodes (OBEs) and Subjective Binge Episodes (SBEs). Previous research has shown no differences between children with OBEs and SBEs (Shomaker et al., 2010a), or between children with no eating-related psychopathology and those who endorse eating objectively large amounts without LOC (Morgan et al., 2002; Tanofsky-Kraff et al., 2004). Thus, LOC appears to be more significant in the evaluation of problem eating than is the amount of food consumed (Marcus & Kalarchian, 2003).

2.2.1 Epidemiology of Loss of Control eating

Binge eating and episodes of loss of control over eating have been documented in children beginning in early to middle childhood. Among a sample of children at high risk for adult obesity, 2.9% of children aged 6-8 years reported an episode of LOC in the previous month,
7.1% of children aged 9-10 years endorsed LOC, and 3.6% of 11-12 year-olds endorsed LOC (Tanofsky-Kraff et al., 2009c). In samples of obese children, the average reported age of first episode of loss of control over eating was 8.4 years ($SD = 1.9$; range 5 - 13 years) and 10.8 years ($SD = 2.6$) for first OBE, suggesting that binge eating behaviors are observable in middle childhood (Decaluwe & Braet, 2003; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005).

Rates of binge eating in children are variable, depending on the child’s age, sex, weight, and clinical status. Lifetime prevalence rates were 1.0% for threshold BED and 4.6% for subthreshold BED (Stice, Marti, Shaw, & Jaconis, 2009) in a community sample of females assessed annually until age 20. A cross-sectional study in a sample of adolescent boys and girls found that approximately 1% met criteria for Binge Eating Disorder (BED) and 11% reported nonclinical binge eating, which includes symptoms such as episodic overeating, eating with distress, or binge eating syndrome not meeting full BED criteria (W. G. Johnson, Grieve, Adams, & Sandy, 1999). Results from studies employing survey and questionnaire reports to assess binge eating in non-treatment seeking children ages 6 – 14 have documented prevalence rates between approximately 2% to 10% (Field et al., 1999; Lamerz et al., 2005; Maloney, McGuire, Daniels, & Specker, 1989). In an examination of nontreatment-seeking obese and non-obese children’s eating behaviors within the previous month (Tanofsky-Kraff et al., 2004), approximately 9.3% of children endorsed loss of control over eating, and 20.4% of children reported episodes of overeating without loss of control. A school sample of 808 adolescent girls aged 12-14 years assessed with the EDE-Q found that 21% reported at least one OBE within the past two weeks (Carter, Stewart, & Fairburn, 2001), with an average number of episodes of 3.8
Although BED is rare, LOC episodes are not uncommon in nonclinical samples of children and adolescents across the weight spectrum.

The prevalence of binge eating episodes in samples of overweight children tends to be relatively higher than samples that include normal weight children. At least one episode of binge eating was reported by 34% of children who were currently obese or at risk for obesity based on parental obesity. A study of exclusively overweight nontreatment-seeking children aged 6 – 10 years found that 5.3% met BED criteria and 33.1% endorsed at least one lifetime LOC episode (Morgan et al., 2002). Among a sample of 105 children aged 6 – 13 years, no children met full BED criteria; however, 30% reported at least one lifetime episode of LOC (Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005). In a study of 249 youth aged 6-18 years that combined overweight treatment-seeking youth and non-treatment-seeking youth oversampled for obesity (Theim et al., 2007), ChEDE results indicated that 19.7% endorsed OBEs, 12.9% reported SBEs, 33.3% reported OOs (objective overeating without LOC), and 34.1% denied any episodes during the past month. Those who endorsed LOC (32.6% of the sample) were heavier and more likely to be seeking weight loss treatment than were those without LOC. In a comparison of 188 overweight youth ages 8-18 years who were either seeking treatment or not (Goossens, Braet, Van Vlierberghe, & Mels, 2009a), 40% (n = 46) of those seeking weight-loss treatment reported LOC in the previous 3 months compared to 21% (n = 15) of those not seeking treatment. Overall, youths endorsed a mean of 7.4 LOC episodes over the prior three months (range: 1 - 37 episodes).

As suggested above, higher prevalence rates of LOC tend to be found in treatment-seeking samples of overweight youth (Eddy et al., 2007; Glasofer et al., 2007; Goossens, Braet, & Decaluwe, 2007; Levine, Ringham, Kalarchian, Wisniewski, & Marcus, 2006) compared to
nontreatment-seeking samples of youth who are overweight (Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2004). Rates of subthreshold binge eating in clinical samples of overweight adolescents are substantial (W. G. Johnson, Rohan, & Kirk, 2002), ranging from approximately 20% (Isnard et al., 2003) to 30% (Decaluwe, Braet, & Fairburn, 2003). Among treatment-seeking overweight children, studies have documented prevalence rates of 11.5% (Wildes et al., 2010) and 15% for binge eating (Levine, Ringham, Kalarchian, Wisniewski, & Marcus, 2006) and 37% for LOC (Tzischinsky & Latzer, 2006). Using a clinical interview (EDE and ChEDE) in an adolescent treatment-seeking sample, Glasofer and colleagues (2007) found that 6.3% met BED criteria, 23.8% endorsed binge episodes in the previous 3 months, 15% endorsed LOC episodes greater than 3 months prior to assessment, and 55% denied any history of LOC or binge episodes. Youth who met BED criteria reported an average number of 35.0 ± 18.5 days with a binge episode in the prior 6 months, and those who endorsed recent LOC episodes reported an average of 3.7 ± 3.3 days with an LOC episode over 3 months prior to assessment. Thus, LOC eating in children is not uncommon and appears to be more prevalent in samples of exclusively overweight children, especially those seeking weight loss treatment.

Similar to findings in adults (Smith, Marcus, Lewis, Fitzgibbon, & Schreiner, 1998), children who report binge eating or LOC tend to be heavier and have greater adiposity than do children who deny these behaviors, in samples comprised of both overweight and nonoverweight children (Shisslak et al., 1998; Tanofsky-Kraff et al., 2004), as well as exclusively overweight children (Decaluwe & Braet, 2003; Morgan et al., 2002). Several studies of nontreatment-seeking youth across the weight spectrum have documented an association between greater body weight or adiposity and episodes of LOC (Tanofsky-Kraff et al., 2004) and binge eating (Field et
al., 1999; Lamerz et al., 2005; Shisslak et al., 1998). The association between greater obesity and binge eating in children also exists among samples of exclusively overweight children seeking treatment (Braet & Van Strien, 1997; Decaluwe & Braet, 2003; Decaluwe, Braet, & Fairburn, 2003; Epstein, Paluch, Saelens, Ernst, & Wilfley, 2001).

Loss of control eating is also associated with relatively greater weight gain over time. A prospective study (Tanofsky-Kraff et al., 2009c) of 143 nontreatment-seeking children ages 6-12 years who were either overweight or at-risk for overweight based on parental BMI found that LOC eating was predictive of an increased rate of BMI growth over the follow-up period of approximately 4.5 ± 1.9 years. Compared to children who denied LOC, those who endorsed LOC gained an additional 2.4 kg per year. Interestingly, LOC was predictive of increased BMI growth regardless of whether or not the amount of food consumed during the episode was objectively large.

Among children at high risk for adult obesity based on their own or parental overweight, binge eating predicted an average gain of 15% more fat mass across middle childhood compared to those without binge eating (Tanofsky-Kraff et al., 2006). Dieting was also a significant predictor of increases in body fat, whereas depressive symptoms and disturbed eating attitudes were not significant predictors. Among 9-14 year old children (Field et al., 2003), binge eating, measured by survey responses, independently predicted weight gain over the subsequent three years. Similar results have been found in prospective studies of adolescents (Stice, Cameron, Killen, Hayward, & Taylor, 1999) and adults (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000). In summary, children and adolescents who endorse binge eating tend to gain more weight over time than do youth who deny binge eating (Field et al., 2003; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice, Presnell, & Spangler, 2002; Tanofsky-Kraff et al., 2006).
Research indicates that binge eating is significantly more common in girls relative to boys (Croll, Neumark-Sztainer, Story, & Ireland, 2002; Decaluwe & Braet, 2003; Field et al., 1999). Survey reports from 9-14 year-olds indicate that the prevalence of binge eating at least monthly increased with age among girls, but remained stable among boys (Field et al., 1999). Research also suggests that girls typically experience greater distress than do boys following binge episodes (LaPorte, 1997; Sierra-Baigrie, Lemos-Giraldez, & Fonseca-Pedrero, 2009). Additionally, a recent laboratory study found that LOC status differentially affected overweight girls compared to overweight boys with regard to energy consumption (Tanofsky-Kraff et al., 2009b). Furthermore, findings indicate that binge eating is at least as common among Black girls as it is among White girls (Morgan et al., 2002; Shisslak et al., 2006; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2004). Thus, findings in samples of children and adolescents indicate that LOC episodes are not uncommon, particularly among girls and overweight children seeking weight loss treatment, are observable in both Black and White children, and are predictive of weight gain over time.

2.2.2 Etiological theories of LOC

Research has examined biological, genetic, environmental, and personality factors that may contribute to the development of LOC eating in children and adolescents. Twin studies indicate that genetic influences on disordered eating symptoms, including weight preoccupation, body dissatisfaction, binge eating, and compensatory behaviors, account for a minimal proportion (6%) of the variance in disordered eating at age 11, but account for 46% of the variance at ages 14 and 18 years. During this period of time, shared environmental influences decrease substantially (Klump, Burt, McGue, & Iacono, 2007). Additional research suggests that these
changes in genetic effects tend to occur during early- to mid-puberty (Culbert, Burt, McGue, Iacono, & Klump, 2009). By adolescence and early adulthood, an adoption study found considerable genetic influences (59% - 82%) on disordered eating symptoms of weight preoccupation, body dissatisfaction, and binge eating cognitions and behaviors, with nonshared environmental factors accounting for the remaining variance. Shared environmental factors did not meaningfully contribute to the variance in any form of disordered eating (Klump, Suisman, Burt, McGue, & Iacono, 2009).

Researchers have examined specific genes that may influence LOC eating. Specifically, studies have focused on the variant rs9939609 of the fat mass and obesity associated gene (FTO), which is highly expressed in the hypothalamic regions important for appetite and therefore may affect weight by influencing eating behavior. Children with rs9939609 FTO variant alleles are predisposed to greater adiposity than are those with two wild-type alleles. A study of 6-19 year-old youth found that the 67.7% of the sample with one or two variant alleles had significantly greater BMI, BMI-z scores, and fat mass than did those with two wild-type alleles. Furthermore, they endorsed more frequent LOC eating episodes (34.7% vs. 18.2%) and consumed a greater percentage of energy from fat during a buffet test meal (Tanofsky-Kraff et al., 2009a).

Research has examined parallels between etiological factors hypothesized to contribute to BED in adults and findings from research in children with LOC. Research indicates that obese adults with BED have larger gastric capacities (Hellstrom, Geliebter, Naslund, & al., 2004) and less nociperception (Raymond et al., 1995) than do obese adults without BED, suggesting that they may have impaired ability to sense fullness. A laboratory study in 6-12 year-old overweight children is consistent with these findings, as children who reported prior episodes of binge eating consumed larger quantities of palatable foods during two buffet meals, along with decreased
subsequent satiety, compared to children who denied binge eating (Mirch et al., 2006). Although there is no imaging data in children, the relationship between emotional eating and LOC in children is consistent with neuroimaging studies of emotional eaters (Bohon, Stice, & Spoor, 2009) and individuals with BED (Schienle, Schafer, Hermann, & Vaitl, 2009), suggesting that processing of food-related stimuli may differ between those with and without disordered eating behaviors.

Theories developed to understand adult eating disturbances postulate that the development and maintenance of binge eating episodes are associated with negative affect (Heatherton & Baumeister, 1991; Leon, Fulkerson, Perry, & Early-Zald, 1995). The Affect Regulation model proposes that individuals who lose control over their eating and binge eat view eating as a means of distraction and comfort from painful negative emotions (Burton, Stice, Bearman, & Rohde, 2007). Binge eating may temporarily reduce momentary negative affective states by functioning as a maladaptive coping strategy (Arnow, Kenardy, & Agras, 1992) or by providing a distraction or escape from self-awareness (Heatherton & Baumeister, 1991). Unfortunately, binge eating may promote subsequent worsening of mood and lead to development of a vicious cycle between negative affect and binge eating (Barker, Williams, & Galambios, 2006).

Researchers suggest that given a high correlation between a sense of “numbing” and LOC episodes, LOC eating may be an early behavioral marker preceding or masking negative affect and disordered eating attitudes (Tanofsky-Kraff et al., 2007a; Tanofsky-Kraff et al., 2011). It is conceivable that children with LOC may have difficulty describing and experiencing emotional states and then LOC develops to serve an affective coping function comparable to that which is reported in adult samples (Arnow, Kenardy, & Agras, 1992; Heatherton & Baumeister,
Similarly, the state negative affect that follows children’s LOC episodes (Tanofsky-Kraff et al., 2007a; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008) could theoretically promote affective distress and worsening of disordered eating attitudes and behaviors as children enter adolescence. Both theories are consistent with the “escape theory” of binge eating in adults, which postulates that binge eating functions to provide an escape from unpleasant self-awareness or emotional distress (Heatherton & Baumeister, 1991). In summary, evidence indicates that multiple genetic, biological, environmental, and psychological factors likely affect the onset and course of LOC eating.

2.2.3 Correlates of LOC

In addition to associations with increased weight, binge eating and LOC in children have been associated with a number of detrimental psychological outcomes. BED and LOC episodes in children and adolescents are associated with greater eating-related psychopathology, negative mood, anxiety, and depressive symptoms (Decaluwe, Braet, & Fairburn, 2003; Glasofer et al., 2007; Morgan et al., 2002; Tanofsky-Kraff et al., 2004). Children who endorse LOC report significantly more disordered attitudes and cognitions related to eating, shape, and weight (Levine, Ringham, Kalarchian, Wisniewski, & Marcus, 2006; Morgan et al., 2002; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2004) compared to children who deny LOC. Additionally, overweight children reporting LOC endorse more general psychopathology than do children without LOC (Morgan et al., 2002; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005). Specifically, LOC is associated with greater anxiety and depressive symptoms (Morgan et al., 2002), as well as lower self-esteem, ineffectiveness, and parent-reported externalizing problems (Tanofsky-Kraff, Faden, Yanovski,
Negative affect has been identified as a robust predictor and correlate of binge eating in children (Stice, Killen, Hayward, & Taylor, 1998; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005) and is one of the strongest longitudinal predictors of binge eating (Stice, 1998).

Studies in adolescents seeking weight-loss treatment have found that those who report episodes of binge eating have greater eating-related distress and depressive symptomatology than do those who deny prior episodes (Berkowitz, Stunkard, & Stallings, 1993; Decaluwe, Braet, & Fairburn, 2003; Isnard et al., 2003; W. G. Johnson, Grieve, Adams, & Sandy, 1999). Among seriously overweight children aged 8-12 years seeking weight-loss treatment (Wildes et al., 2010), self-reported binge eating was associated with younger age, lower self-esteem, and increased depressive, anxiety, and eating disorder symptoms relative to those without binge eating. In a study of 64 overweight adolescents matched on treatment referral status, age, gender, and degree of overweight, those who endorsed LOC within the previous 3 months displayed a greater severity of dysfunctional schemas than did those denying prior LOC (Van Vlierberghe, Braet, & Goossens, 2009). Specifically, youth with LOC acknowledged maladaptive schema related to eating-disordered cognitions, dietary restraint attitudes, and depressive symptoms.

A comparison of youth with and without LOC on a measure of emotional eating found that recent LOC eating was associated with higher scores on all subtypes of emotional eating: eating in response to anxiety, anger, and frustration; depressive symptoms; and feeling unsettled (Tanofsky-Kraff et al., 2007b). A community sample of youth aged 8-13 with at least one LOC episode in the previous 3 months and a matched control group with no LOC found that those with LOC made higher use of dysfunctional emotion regulation strategies, particularly for anxiety, but there were no group differences in use of adaptive strategies (Czaja, Rief, & Hilbert,
Similar results were found in a laboratory study comparing children with or without LOC, as children with LOC reported relatively greater increases in negative affect following two ad libitum meals where they were instructed to “eat normally” and to “binge eat” (Tanofsky-Kraff et al., 2009b). Recent research documented positive associations between binge eating, negative affect, and weight-based teasing in a sample of 265 adolescent female twins, with negative affect partially mediating the association between weight-based teasing and binge eating (Suisman, Slane, Burt, & Klump, 2008).

In a community sample of 120 girls aged 8 to 13 years, investigators compared girls with and without LOC episodes on measures of personality and psychopathology (Hartmann, Czaja, Rief, & Hilbert, 2010). Girls who endorsed LOC showed lower self-directedness, less cooperativeness, higher impulsivity, and greater novelty-seeking but not harm avoidance relative to those who denied prior LOC. In a school sample of Italian adolescents, those who endorsed at least one episode of “bingeing on food” in the previous 6 months reported more symptoms of depression, thought problems, somatic complaints, social withdrawal, attention-seeking, and phobic-anxious behavior than did their peers who denied bingeing (Sierra-Baigrie, Lemos-Giraldez, & Fonseca-Pedrero, 2009). Among a sample of treatment-seeking overweight children, those who endorsed an episode of binge eating in the previous 28 days scored higher on a computerized gambling task measuring impulsivity than did overweight children without a recent binge episode (Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006).

Frequency of LOC episodes has been associated with greater pathology in some samples. One study found greater disturbed eating-related cognitions and depressive symptomatology in 10-18 year-olds with questionnaire-assessed BED relative to those with subthreshold symptoms (W. G. Johnson, Grieve, Adams, & Sandy, 1999). Among a sample of overweight adolescents
seeking weight loss treatment, youths were placed into one of four categories based on responses to the EDE interview: full-syndrome binge-eating disorder (BED), recent but infrequent binge episodes within previous 3 months (Recent Binge), remote and infrequent LOC episodes occurring more than 3 months before assessment (Past LOC), and no history of LOC episodes (NE) (Glasofer et al., 2007). Results indicated that the BED group endorsed higher global EDE scores (mean of restraint, eating concern, shape concern, and weight concern subscales) and more negative mood and anxiety than the other three groups. Recent Binge, but not Past LOC, was associated with significantly higher anxiety and eating-related psychopathology.

Although greater frequency of LOC episodes may correlate with increased pathology, research suggests that endorsement of as few as one LOC episode is associated with weight and psychological problems. One study comparing children and adolescents with BED and subthreshold BED did not find significant differences in depressed mood, self-esteem, or body dissatisfaction (Ackard, Neumark-Sztainer, Story, & Perry, 2003). In a study using structured clinical interviews to assess LOC in overweight children ages 6-13, those who endorsed one or more lifetime episodes of LOC also reported greater general and eating-disordered psychopathology compared with those who never experienced an episode of LOC (Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005). Similar results were found in a prospective study that followed a community sample of youth across middle childhood (Tanofsky-Kraff et al., 2011). Youth who endorsed at least one lifetime LOC episode had significantly higher baseline BMI, BMI $z$ scores, disordered eating attitudes, and depressive symptoms relative to youth who denied prior LOC. Those endorsing LOC at baseline continued to have higher follow-up BMI, BMI $z$ scores, and EDE global eating pathology scores, along with higher restraint, weight concern, and shape concern subscale scores. In summary, LOC is
associated with a number of indices of psychological distress and eating pathology, even among children who endorse a limited history of previous episodes.

2.2.4 Characteristics of LOC episodes

Research has examined the emotional, environmental, and food-related contingencies that occur before, during, and after LOC episodes in order to better characterize loss of control eating behavior. In a multisite sample of 445 youths, LOC was associated with eating forbidden food before the episode, eating when not hungry, eating alone, and experiencing secrecy, negative emotions, and a feeling of “numbing” while eating (Tanofsky-Kraff et al., 2007a). Hierarchical cluster analyses revealed that adolescents reporting LOC had a similar presentation as adults with binge eating disorder. However, children ages 6-12 years who endorsed LOC had a presentation that differed from adolescents, in that they reported the LOC episode took place in the afternoon, at a home not their own, and they were eating more than others. They also reported experiencing a negative emotion and trigger prior to the episode, eating in secret, and feeling numb. Negative affect both preceded and followed the episodes; however, positive mood was a precursor to eating episodes in general. Importantly, these children reported that before the LOC episode, they began eating despite a lack of hunger (Tanofsky-Kraff et al., 2007a). Onset of related behaviors of secretive and inhibited eating have been associated with maternal BMI and paternal history of overweight (Stice, Agras, & Hammer, 1999). Retrospective studies that utilized questionnaire assessments of LOC similarly found that emotional eating in response to sadness, anxiety, or discontent, and depressive symptoms was associated with increased LOC in youth (Eddy et al., 2007; Goldschmidt et al., 2008b; Tanofsky-Kraff et al., 2007b).
In a study of youth age 6-18 years old who either reported or denied episodes of LOC over the previous month (Theim et al., 2007), participants were asked to describe the type and quantity of food eaten during a LOC episode (for those endorsing LOC), an episode of overeating without LOC, or a normal eating episode. There were no differences in total calories consumed between LOC and overeating or normal episodes; however, LOC episodes were characterized by consumption of a significantly lower percentage of calories from protein and a higher percentage from carbohydrates. Specifically, LOC episodes consisted of a higher percentage of calories from snacks and desserts. Analyses of food diaries completed by overweight youth enrolled in weight-loss treatment indicated that those scoring high on the Binge Eating Scale tended to record greater intake of food and calories, along with greater carbohydrate content, relative to those who scored low on binge eating (Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005). Following a multi-item, buffet-style test meal, youth who endorsed LOC episodes were less accurate at reporting the percentage of energy they consumed from carbohydrates and their intake of desserts relative to those who denied LOC (Wolkoff et al., 2011).

Because children with LOC may have difficulties with accurately recalling food consumed during eating episodes, researchers have employed ecological momentary assessment (EMA) and laboratory studies to examine energy and nutrient intake. A recent study used EMA to compare eating behavior, mood, and eating disorder-specific cognitions during a four day period between children aged 8-13 years who reported LOC and those who denied prior LOC (Hilbert, Rief, Tuschen-Caffier, de Zwaan, & Czaja, 2009). Results indicated that LOC episodes led to a significantly greater intake of energy, particularly from carbohydrates, than regular meals with and without LOC eating. LOC episodes were preceded and followed by cognitions
about food, eating, and body image; however, there was minimal evidence that negative mood states were antecedents of LOC episodes.

Goldschmidt and colleagues (2011) conducted a laboratory study of overweight girls aged 6-12 years who either endorsed or denied prior LOC. Girls underwent both sad and neutral mood inductions, followed by multi-item buffet meals. There were no group differences in overall energy intake; however, girls with LOC consumed a greater percent of energy from fat following the sad induction as compared to the neutral condition. Following the sad mood induction, pre-induction negative affect in girls with LOC was associated with increased likelihood of experiencing loss of control during the test meal.

In a laboratory study of overweight treatment-seeking children aged 6 – 12 years, children who reported prior episodes of binge eating consumed larger quantities of palatable foods during two self-selected buffet meals (following an overnight fast and a standardized breakfast), along with decreased duration of subsequent satiety, compared to children who denied binge eating (Mirch et al., 2006). However, the macronutrient content of the meals did not differ between groups. Additionally, those who reported prior binge episodes became hungry more quickly following a fixed-calorie preload and reported a greater desire to eat relative to children who denied prior binge episodes.

Tanofsky-Kraff and colleagues (2009b) conducted a laboratory study of 177 nonoverweight and overweight boys and girls aged 8-17 years in which they instructed participants to either “binge eat” or “eat normally” ad libitum from multi-item food arrays during two lunchtime meals. All participants consumed more energy at the binge meal compared to the normal meal. There were no overall differences in energy intake at either meal between youth reporting LOC and those denying LOC. However, at both meals, children with LOC consumed a
greater percentage of calories from carbohydrates and a smaller percentage from protein, as well as more snack and dessert foods and less meats and dairy, relative to those without LOC. Secondary analyses restricted to overweight and obese girls found that those with LOC consumed more energy at the binge meal relative to girls without LOC. These findings suggest that LOC may contribute to weight gain through intake of excessive calories and certain types of foods, coupled with relatively less satiety after eating. Furthermore, this research suggests that these episodes are often characterized by negative affect and commonly occur in specific types of settings.

2.2.5 Course and significance

Currently, there are minimal data regarding the stability and course of LOC in children over time. One recent study by Tanofsky-Kraff and colleagues (2011) administered self-report measures of depression and anxiety along with the ChEDE to assess LOC in 195 boys and girls (mean age = 10.4 years, SD = 1.5) in a sample designed to be “enriched” for obesity at baseline and again 4.7 years (SD = 1.2) later to 118 participants. Forty-six children (23.6%) reported at least one lifetime episode of LOC at baseline; among those participants, 47.8% experienced at least one LOC episode during the month prior to assessment. Report of at least one lifetime LOC episode at baseline was associated with the subsequent development of partial- or full-syndrome binge eating disorder (approximately 5%), even after accounting for the contribution of sex, race, BMI change over follow-up, years in study, and baseline characteristics including age, disordered eating attitudes, and mood symptoms. Approximately half of children who endorsed LOC at baseline reported persistence of LOC at follow-up. Compared with children who denied any history of LOC or who endorsed LOC at baseline only, those with LOC at both baseline and
follow-up experienced the greatest increases in disordered eating attitudes and depressive symptoms over time. Thus, even infrequent episodes of LOC may be an important marker for subsequent development of increased disordered eating and development of partial-or full-syndrome BED. Notably, only LOC, as opposed to weight, shape, and eating concerns, or negative affect, was a clinically relevant behavior for the development of BED.

An 8-year longitudinal study of the course of eating disorders in a community sample of adolescents beginning in 7th or 8th grade (Stice, Marti, Shaw, & Jaconis, 2009) found that the peak age of onset was 17-18 years for BED and relapse rate was 33%. Subthreshold BED progressed to threshold BED in 13% of cases during the assessment period, and crossover from BED to BN occurred in 42% and from BED to purging disorder occurred in 4% of cases. Additionally, there was crossover from BN to BED in 19% and from purging to BED in 9% of cases. There were no differences between subthreshold BED and threshold BED participants in rates of mental health treatment, functional impairment, or emotional distress; however, those with threshold and subthreshold BED had greater rates of mental health treatment, impairment, and distress than did controls. Notably, subthreshold cases were more prevalent than were threshold eating disorders and were associated with marked impairment (Stice, Marti, Shaw, & Jaconis, 2009). Thus, binge eating and LOC appear to persist or worsen over time for a considerable number of individuals.

A limited number of studies have examined the relationship between LOC and weight-loss treatment outcomes in youth. Children with LOC appear to be at greater risk for early attrition from weight loss treatment. During the course of a family-based treatment for seriously overweight children ages 8-13, only 50% of children who endorsed LOC at baseline completed treatment compared to 70% of children who denied LOC (Levine, Ringham, Kalarchian,
Wisniewski, & Marcus, 2006). Similar findings of relatively higher dropout rates from behavioral weight loss treatment have been documented for obese adults who binge eat when compared with those who do not binge eat (Marcus, Wing, & Hopkins, 1988). However, one recent study found that LOC eating was associated with a relatively lower drop-out rate (8.3%) in a residential weight-loss program for obese youth, possibly due to the inpatient nature of the treatment (Goossens, Braet, Van Vlierberghe, & Mels, 2009b).

Several studies have examined the relationship between LOC and related behaviors on weight loss in intervention studies. One study of 7-17 year old participants in an inpatient healthy lifestyle change program found that youth reported fewer episodes of binge eating both at post-treatment and 14-month follow-up (Braet, Tanghe, Decaluwe, Moens, & Rosseel, 2004). Pre-treatment eating disorder symptoms were associated with less weight loss and less improvements in global self-worth following completion of an inpatient pediatric obesity treatment program (Braet, 2006). Additionally, following completion of a 6-month family-based behavioral weight loss intervention, children aged 8-12 years who reported binge eating showed a 2.6% increase in percent overweight compared to an 8.5% decrease among children without binge eating (Wildes et al., 2010). Although these effects were not maintained throughout the follow-up period, the short-term disparity in weight change is noteworthy given that short-term weight loss appears predictive of longer-term success (Goossens, Braet, Van Vlierberghe, & Mels, 2009b; Reinehr, Temmesfeld, Kersting, de Sousa, & Toschke, 2007). The majority of pediatric obesity interventions are characterized by small changes in relative weight and considerable relapse (Goldfried, Raynor, & Epstein, 2004; Jelalian & Saelens, 1999). Given the association between LOC and increased weight gain over time, findings of poor treatment
response among overweight children with LOC highlights the need for early identification and intervention.

In summary, LOC eating is not uncommon, particularly in samples of overweight children seeking treatment, and is associated with increased weight gain over time (Field et al., 2003; Schienle, Schafer, Hermann, & Vaitl, 2009; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice, Presnell, & Spangler, 2002; Tanofsky-Kraff et al., 2006). Furthermore, LOC is associated with increased levels of eating-disordered and general psychopathology. Given the persistence of LOC over time (Tanofsky-Kraff et al., 2011), additional research to identify early markers of LOC appears warranted.

2.3 STATEMENT OF PROBLEM

The evaluation of problem eating among children ages 10-13 is important for several reasons. Given that adolescence is a well-documented risk factor for the onset of eating disorders (Stice, Agras, & Hammer, 1999), the identification of aberrant eating patterns in early to middle childhood has potential implications for prevention or treatment of eating disorders. Given its similarity to disinhibition, Eating in the Absence of Hunger is one eating behavior that may be associated with loss of control. Because it can be identified in children as young as 3 years of age (Cutting, Fisher, Grimm-Thomas, & Birch, 1999), determining whether EAH is associated with aberrant eating in middle childhood could help direct development of early intervention programs. Fisher and colleagues (2007) suggest that although EAH may capture behavioral aspects of disinhibition, the loss of control over eating ascribed to disinhibited eating among adults may involve emotional and cognitive as well as behavioral components (Lowe &
Maycock, 1988). A recent study found a relationship between a questionnaire measure of EAH and LOC (Tanofsky-Kraff et al., 2008); however, research suggests that children with LOC may have difficulty accurately describing their eating episodes (Wolkoff et al., 2011). Therefore, it is important to examine the relationship between EAH and LOC using a behavioral measure of EAH. Thus, one goal of the proposed study is to directly assess whether EAH is associated with children’s reports of loss of control over eating.

In summary, a considerable body of evidence indicates that Eating in the Absence of Hunger is associated with the development of obesity in White girls. However, it is not known whether similar relationships between EAH and weight exist in Black girls. Thus, the proposed study aims to examine the relationships between child weight, EAH, and loss of control in a racially diverse sample of girls in middle childhood. Given the ability to identify EAH in children at a relatively young age and the capacity to quantify differences in EAH in a laboratory setting, examination of EAH in a sample of girls who are particularly vulnerable to obesity is crucial. Finally, given the potential to modify child eating behaviors, evaluating the relationship between LOC and EAH could have implications for the development of pediatric obesity treatments.

The study addressed the following objectives:

Aim 1: The first aim of this study was to examine EAH in a racially and socioeconomically diverse sample of girls age 10-13 years.

Aim 2: The second aim was to evaluate the relationship between EAH and self-reported episodes of loss of control over eating.
3.0 METHODS

3.1 PARTICIPANTS

Fifty-one girls and their mothers were enrolled in this study. For inclusion in the study, mothers were required to live with their child at least half-time. Eligible children were 1) female, and 2) between the age 10-13 years at the time of study participation. Exclusion criteria for children included 1) developmental delays that could compromise the ability to complete study assessments, 2) allergy to any of the foods used in the experimental procedure, 3) regular use of a medication that affects body weight such as oral steroids or antipsychotic medication, 4) current enrollment in a structured weight loss program, and 5) recent initiation (less than 4 months) of stimulant or antidepressant medications. All procedures were approved by the University of Pittsburgh Institutional Review Board.

A total of 54 mothers contacted the study to inquire about participation. Three were not eligible for the following reasons: child was too old to participate (n = 2) and child was currently enrolled in a weight loss program (n = 1). All 51 participants who were scheduled for a study appointment attended the appointment, signed an informed consent form (mothers) and provided assent (children), and completed the study procedure.
3.1.1 Child characteristics

Girls (N = 51) who completed the study had a mean age of 12.02 years (SD = 0.95; range = 10.16 - 13.84). Participants self-reported race as Caucasian (n = 36; 70.6%), Black or Biracial (n = 11; 21.6%), Hispanic (n = 3; 5.9%), and Asian (n = 1; 2%). Mean BMI was 21.86 (SD = 5.61; range = 15.70 - 44.38). Additional child characteristics are reported in Tables 1 and 2.

Table 1: Child, maternal, and family characteristics: Means.

<table>
<thead>
<tr>
<th></th>
<th>Range</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Child Characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>10.16 – 13.84</td>
<td>12.02</td>
<td>0.95</td>
</tr>
<tr>
<td>BMI</td>
<td>15.70 – 44.38</td>
<td>21.86</td>
<td>5.61</td>
</tr>
<tr>
<td>BMI percentile</td>
<td>18.4 – 99.8</td>
<td>70.54</td>
<td>23.22</td>
</tr>
<tr>
<td>Tanner Stage</td>
<td>2 – 5</td>
<td>3.33</td>
<td>0.77</td>
</tr>
<tr>
<td><strong>Maternal and Family Characteristics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>28.84 – 58.21</td>
<td>43.58</td>
<td>7.22</td>
</tr>
<tr>
<td>BMI</td>
<td>18.20 – 49.91</td>
<td>29.58</td>
<td>7.31</td>
</tr>
<tr>
<td>% Above Poverty Line</td>
<td>-75 – 300</td>
<td>143.37</td>
<td>81.35</td>
</tr>
<tr>
<td># Children in Household</td>
<td>1 - 3</td>
<td>1.96</td>
<td>0.69</td>
</tr>
<tr>
<td>Total # in Household</td>
<td>2 - 6</td>
<td>3.76</td>
<td>1.07</td>
</tr>
</tbody>
</table>

(N = 51 mother-daughter dyads)
**Table 2:** Child, maternal, and family characteristics: Proportions.

<table>
<thead>
<tr>
<th>Child Characteristics</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White, non-Hispanic</td>
<td>36</td>
<td>70.6%</td>
</tr>
<tr>
<td>Black or Biracial</td>
<td>11</td>
<td>21.6%</td>
</tr>
<tr>
<td>Hispanic</td>
<td>3</td>
<td>5.9%</td>
</tr>
<tr>
<td>Asian</td>
<td>1</td>
<td>2%</td>
</tr>
<tr>
<td><strong>Weight status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight or Obese</td>
<td>19</td>
<td>37.3%</td>
</tr>
<tr>
<td>Not overweight</td>
<td>32</td>
<td>62.7%</td>
</tr>
<tr>
<td><strong>Tanner Stage</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early Pubertal</td>
<td>6</td>
<td>11.8%</td>
</tr>
<tr>
<td>Midpubertal</td>
<td>25</td>
<td>49.0%</td>
</tr>
<tr>
<td>Late Pubertal</td>
<td>17</td>
<td>33.3%</td>
</tr>
<tr>
<td>Postpubertal</td>
<td>3</td>
<td>5.9%</td>
</tr>
<tr>
<td><strong>Maternal and Family Characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Income</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than 10,000</td>
<td>2</td>
<td>3.9%</td>
</tr>
<tr>
<td>10,001 - 30,000</td>
<td>10</td>
<td>19.6%</td>
</tr>
<tr>
<td>30,001 - 50,000</td>
<td>6</td>
<td>11.8%</td>
</tr>
<tr>
<td>50,001 – 70,000</td>
<td>8</td>
<td>15.6%</td>
</tr>
<tr>
<td>70,001 – 90,000</td>
<td>12</td>
<td>23.6%</td>
</tr>
<tr>
<td>More than 90,000</td>
<td>13</td>
<td>25.4%</td>
</tr>
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</table>
### Table 2 (continued)

**Education**

<table>
<thead>
<tr>
<th>Education</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>High School Grad</td>
<td>3</td>
<td>5.9%</td>
</tr>
<tr>
<td>Some College</td>
<td>18</td>
<td>35.3%</td>
</tr>
<tr>
<td>College Graduate</td>
<td>14</td>
<td>27.5%</td>
</tr>
<tr>
<td>Postgraduate Degree</td>
<td>16</td>
<td>31.4%</td>
</tr>
</tbody>
</table>

**Marital Status**

<table>
<thead>
<tr>
<th>Marital Status</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Married or Cohabitating</td>
<td>30</td>
<td>58.8%</td>
</tr>
<tr>
<td>Never Married</td>
<td>10</td>
<td>19.6%</td>
</tr>
<tr>
<td>Separated</td>
<td>4</td>
<td>7.8%</td>
</tr>
<tr>
<td>Divorced</td>
<td>6</td>
<td>11.8%</td>
</tr>
<tr>
<td>Deceased</td>
<td>1</td>
<td>2.0%</td>
</tr>
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</table>

**Poverty Status***

<table>
<thead>
<tr>
<th>Poverty Status</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 85%</td>
<td>12</td>
<td>23.5%</td>
</tr>
<tr>
<td>86-199%</td>
<td>8</td>
<td>15.7%</td>
</tr>
<tr>
<td>200% +</td>
<td>31</td>
<td>60.8%</td>
</tr>
</tbody>
</table>

**Housing Status**

<table>
<thead>
<tr>
<th>Housing Status</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Own home</td>
<td>38</td>
<td>74.5%</td>
</tr>
<tr>
<td>Rent</td>
<td>10</td>
<td>19.6%</td>
</tr>
<tr>
<td>Not paying for housing/Other</td>
<td>3</td>
<td>5.9%</td>
</tr>
</tbody>
</table>

**Relationship of Mother to Daughter**

<table>
<thead>
<tr>
<th>Relationship of Mother to Daughter</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biological Mother</td>
<td>45</td>
<td>88.2%</td>
</tr>
<tr>
<td>Adoptive Mother</td>
<td>6</td>
<td>11.8%</td>
</tr>
</tbody>
</table>

*N = 51; *Poverty Status: families at ≤ 85% qualify for free or reduced school lunch program*
Independent-samples t-tests and chi-square analyses were done to examine potential racial differences in background variables between Black and non-Hispanic White girls. White girls came from households that were a higher percentage above poverty level relative to their Black peers, \( t (1, 45) = 3.23, p < .01 \) \( (M = 161.56\%; SD = 69.96\% \) vs. \( M = 69.96\%; SD = 89.17\%) \). Mothers of Black girls had a higher BMI than did mothers of White girls \( t (1, 45) = -2.27, p < .05 \) \( (M = 34.07; SD = 9.56 \) vs. \( M = 28.44; SD = 6.37) \). White girls were also significantly more likely to be living with both of their biological parents than were Black girls, \( \chi^2 (df = 1, N = 47) = 16.82; p < .001 \) \( (77.8\% \) vs. \( 9.1\%) \). There were no racial differences in child age, Tanner Stage, or maternal education, age, or percent of children participating with their biological mothers. Tables 3 and 4 summarize the relationships between race and other study variables.
Table 3: Relationships between race and study variables.

<table>
<thead>
<tr>
<th></th>
<th>White (n = 36)</th>
<th>Black or Biracial (n=11)</th>
<th>Combined (N = 47)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child Age</td>
<td>12.06 (0.91)</td>
<td>11.77 (0.98)</td>
<td>11.99 (0.92)</td>
<td>0.91</td>
<td>.367</td>
</tr>
<tr>
<td>BMI</td>
<td>67.84 (24.16)</td>
<td>77.31 (23.44)</td>
<td>70.06 (24.08)</td>
<td>-1.15</td>
<td>.258</td>
</tr>
<tr>
<td>Tanner Stage</td>
<td>3.25 (0.84)</td>
<td>3.45 (0.52)</td>
<td>3.30 (0.78)</td>
<td>-0.76</td>
<td>.451</td>
</tr>
<tr>
<td>Poverty Level</td>
<td>161.56 (69.96)</td>
<td>69.96 (89.17)</td>
<td>142.11 (81.96)</td>
<td>3.23</td>
<td>.002</td>
</tr>
<tr>
<td>Maternal BMI</td>
<td>28.44 (6.37)</td>
<td>34.07 (9.56)</td>
<td>29.76 (7.52)</td>
<td>-2.27</td>
<td>.028</td>
</tr>
<tr>
<td>Education</td>
<td>4.83 (0.94)</td>
<td>4.55 (0.93)</td>
<td>4.77 (0.94)</td>
<td>0.89</td>
<td>.379</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>43.67 (5.96)</td>
<td>41.57 (10.42)</td>
<td>43.18 (7.17)</td>
<td>0.85</td>
<td>.401</td>
</tr>
<tr>
<td>EAH Consumption</td>
<td>312.08 (255.94)</td>
<td>414.23 (321.39)</td>
<td>335.99 (272.41)</td>
<td>-1.09</td>
<td>.281</td>
</tr>
<tr>
<td>Dinner Consumption</td>
<td>723.99 (225.08)</td>
<td>1002.37 (390.07)</td>
<td>789.14 (292.94)</td>
<td>-2.99</td>
<td>.005</td>
</tr>
<tr>
<td>Total Calorie</td>
<td>1036.08 (371.45)</td>
<td>1416.60 (395.75)</td>
<td>1125.14 (406.87)</td>
<td>-2.93</td>
<td>.005</td>
</tr>
<tr>
<td>%DRI- EAH</td>
<td>13.43 (10.73)</td>
<td>17.09 (14.31)</td>
<td>14.28 (11.60)</td>
<td>-0.92</td>
<td>.365</td>
</tr>
<tr>
<td>%DRI- Dinner</td>
<td>31.14 (8.55)</td>
<td>38.67 (12.50)</td>
<td>32.90 (10.00)</td>
<td>-2.29</td>
<td>.027</td>
</tr>
<tr>
<td>%DRI- Total</td>
<td>44.56 (14.57)</td>
<td>55.76 (15.68)</td>
<td>47.19 (15.43)</td>
<td>-2.19</td>
<td>.034</td>
</tr>
</tbody>
</table>

*df (1, 45) for all variables*
Table 4: Categorical proportions of child characteristics by race.

<table>
<thead>
<tr>
<th></th>
<th>White, non-Hispanic n = 36</th>
<th>Black or Biracial n = 11</th>
<th>Total N = 47</th>
<th>$\chi^2$ (df = 1, N = 47)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lives with biological parents</td>
<td>77.8%</td>
<td>9.1%</td>
<td>61.7%</td>
<td>16.82</td>
<td>.000</td>
</tr>
<tr>
<td>Endorsed LOC episode</td>
<td>8.3%</td>
<td>63.6%</td>
<td>21.3%</td>
<td>15.39</td>
<td>.000</td>
</tr>
<tr>
<td>Lives with biological mother</td>
<td>91.7%</td>
<td>81.8%</td>
<td>89.4%</td>
<td>0.86</td>
<td>.354</td>
</tr>
<tr>
<td>Overweight or Obese</td>
<td>36.1%</td>
<td>54.5%</td>
<td>40.4%</td>
<td>1.19</td>
<td>.276</td>
</tr>
</tbody>
</table>

Independent-samples t-tests and chi-square analyses were also conducted to determine whether there were any significant differences between normal-weight and overweight (BMI percentile ≥ 85th) children on any of the background variables. Overweight girls reported more advanced pubertal development as measured by Tanner Stage than did their non-overweight peers, $t (1, 49) = -2.23$, $p < .05$ ($M = 3.63$; $SD = 0.76$ vs. $M = 3.16$; $SD = 0.72$). Additionally, mothers of overweight girls had a significantly higher mean BMI than did mothers of normal weight children, $t (1, 49) = -3.13$, $p < .01$ ($M = 33.41$; $SD = 8.20$ vs. $M = 27.30$; $SD = 5.73$). There were no differences between normal-weight and overweight children in age, race, poverty level, or percent living with two biological parents, or maternal education, age, or percent of biological mothers participating in the study. Tables 5 and 6 summarize the relationships between weight status and other study variables.
Table 5: Relationships between weight status and study variables.

<table>
<thead>
<tr>
<th></th>
<th>Not Overweight (n = 32)</th>
<th>Overweight/Obese (n= 19)</th>
<th>Entire Sample (N = 51)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child Age</td>
<td>11.87 (0.98)</td>
<td>12.26 (0.87)</td>
<td>12.02 (0.95)</td>
<td>-1.45</td>
<td>.154</td>
</tr>
<tr>
<td>BMI Percentile</td>
<td>57.12 (18.85)</td>
<td>93.13 (5.20)</td>
<td>70.54 (23.22)</td>
<td>-8.11</td>
<td>.000</td>
</tr>
<tr>
<td>Tanner Stage</td>
<td>3.16 (0.72)</td>
<td>3.63 (0.76)</td>
<td>3.33 (0.77)</td>
<td>-2.23</td>
<td>.031</td>
</tr>
<tr>
<td>Poverty Level</td>
<td>137.97 (87.08)</td>
<td>152.47 (72.00)</td>
<td>143.37 (81.35)</td>
<td>-0.61</td>
<td>.544</td>
</tr>
<tr>
<td>Maternal BMI</td>
<td>27.30 (5.73)</td>
<td>33.41 (8.20)</td>
<td>29.58 (7.31)</td>
<td>-3.13</td>
<td>.003</td>
</tr>
<tr>
<td>Education</td>
<td>4.78 (0.98)</td>
<td>4.95 (0.91)</td>
<td>4.84 (0.95)</td>
<td>-0.60</td>
<td>.550</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>34.31 (6.55)</td>
<td>44.05 (8.39)</td>
<td>43.58 (7.22)</td>
<td>-0.35</td>
<td>.728</td>
</tr>
<tr>
<td>EAH Consumption</td>
<td>360.86 (296.57)</td>
<td>304.17 (263.30)</td>
<td>339.74 (283.29)</td>
<td>0.69</td>
<td>.495</td>
</tr>
<tr>
<td>Dinner Consumption</td>
<td>666.80 (206.91)</td>
<td>933.78 (349.66)</td>
<td>766.27 (295.89)</td>
<td>-3.44</td>
<td>.001</td>
</tr>
<tr>
<td>Total Calorie Consumption</td>
<td>1027.67 (386.56)</td>
<td>1237.96 (442.37)</td>
<td>1106.01 (416.70)</td>
<td>-1.78</td>
<td>.081</td>
</tr>
<tr>
<td>%DRI- EAH</td>
<td>16.07 (13.07)</td>
<td>11.93 (10.50)</td>
<td>14.53 (12.23)</td>
<td>1.17</td>
<td>.246</td>
</tr>
<tr>
<td>%DRI- Dinner</td>
<td>29.78 (8.78)</td>
<td>35.93 (11.63)</td>
<td>32.07 (10.27)</td>
<td>-2.14</td>
<td>.037</td>
</tr>
<tr>
<td>%DRI- Total</td>
<td>45.85 (16.36)</td>
<td>47.86 (16.00)</td>
<td>46.60 (16.09)</td>
<td>-0.43</td>
<td>.670</td>
</tr>
</tbody>
</table>

*df (1, 49) for all variables*
Table 6: Categorical proportions of child characteristics by weight status.

<table>
<thead>
<tr>
<th></th>
<th>Not Overweight n = 32</th>
<th>Overweight n = 19</th>
<th>Total N = 51</th>
<th>$\chi^2$ (df = 1, N = 51)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lives with biological parents</td>
<td>35.3%</td>
<td>23.5%</td>
<td>58.8%</td>
<td></td>
<td>.24</td>
</tr>
<tr>
<td>Endorsed LOC episode</td>
<td>7.8%</td>
<td>11.8%</td>
<td>19.6%</td>
<td>2.75</td>
<td>.097</td>
</tr>
<tr>
<td>Lives with biological mother</td>
<td>56.9%</td>
<td>31.4%</td>
<td>88.2%</td>
<td>0.47</td>
<td>.492</td>
</tr>
<tr>
<td>Caucasian, non-Hispanic</td>
<td>45.1%</td>
<td>25.5%</td>
<td>70.6%</td>
<td>0.07</td>
<td>.794</td>
</tr>
</tbody>
</table>

$df$ (1, 49) for all variables

3.1.2 Maternal and family characteristics

Mothers (N = 51) who completed the study had an average BMI of 29.58 (SD = 7.31; range = 18.20 - 49.91). The majority of the maternal participants were the child’s biological mother (88.2%), and 58.8% were married. Annual family income was less than $30,000 for 19.6% of the sample, and 23.5% qualified for the free or reduced cost school lunch program according to federal poverty guidelines. The majority of the mothers (58.9%) had completed college or postgraduate study. Additional maternal and family characteristics are described in Tables 1 and 2.
3.2 PROCEDURE

Participants were recruited from Craigslist postings, print ads and voicemail announcements to employees and affiliates of the university’s medical center, and flyers posted throughout Pittsburgh in community settings such as churches, university buildings, restaurants, community centers, and bus stops. Efforts were made to post the flyers in a variety of neighborhoods in order to target a socioeconomically and racially diverse sample. All participants enrolled in this study between May of 2008 to May of 2009. Recruitment materials described the investigation as a study of girls’ food preferences following dinner consumption. Interested individuals were invited to contact the program regarding the study. When potential participants contacted the study by phone, a brief screening call was completed with mothers to determine study eligibility. The study was explained briefly, and after providing verbal consent for the investigator to ask questions, participants were asked to provide preliminary demographic and health information, including child height, weight, food allergies or restrictions, and use of medications known to affect weight, in order to determine study eligibility.

If it appeared that the family met the study criteria, participant questions were addressed, children’s dinner preferences were obtained, and they were scheduled to begin the assessment between 4:30 and 6:30 pm. All study appointments took place in the Behavioral Medicine Lab at the University of Pittsburgh Medical Center. Children were instructed to eat their usual breakfast and lunch on the day of the assessment, and to refrain from eating between lunch and the beginning of the procedure.

Prior to the participants’ arrival, the food used for dinner and the free-access procedure was weighed to the nearest gram and recorded. Upon arrival, the study investigator reviewed the consent form with the mother and daughter and answered any questions. Mothers provided
consent and children provided assent for enrollment before the experimental procedure commenced. After consenting, mothers were moved to a different room to complete questionnaires. Children were offered a standard test meal comprised of carrots, apples, oranges, yogurt, a 12-inch Subway submarine sandwich pre-made according to the child’s preferences, and a beverage (grape or apple juice, fruit punch, or water). Portion sizes offered were roughly equivalent across participants, except children identified as significantly overweight during the screening call were offered an additional 6-inch sub. All children were informed they could request additional portions, if desired. They were encouraged to eat until they were comfortably full and not hungry anymore, but not to the extent that they were extremely full.

Children were left alone in a conference room to eat and after finishing the meal, they were interviewed to assess their degree of satiety. Girls were shown a 5-point Likert scale with descriptions of differing degrees of satiety: “1) extremely hungry,” “2) hungry,” “3) half full,” “4) comfortably full,” and “5) extremely full.” Two participants selected a “3” on the Likert scale (“half full”) and were encouraged to continue eating until they reported feeling comfortably full. Those selecting a “4” on the scale (“comfortably full”) proceeded to the next stage of the experiment.

Next, girls were moved to a different room with serving containers filled with large quantities of 10 different sweet or savory snack foods (e.g., white cheddar popcorn, potato chips, pretzels, tortilla chips, nuts, fig bars, chocolate chip cookies, Starburst fruit chew candy, M & M chocolate pieces, and red licorice), as well as art supplies and word search books. They were informed that the snacks were left over from a meeting held earlier in the afternoon and were told that they could eat as much of the snack foods as they would like. The questionnaire on pubertal development was explained to them and they were encouraged to ask questions if any
item was unclear. Girls were instructed to first complete the questionnaire and then they could engage in the leisure activities and/or eat any of the food available while the experimenter reviewed some information with their mother in a separate room for 15-20 minutes.

While the children were involved in the free-access procedure, mothers completed a demographic questionnaire, and then their heights and weights were measured. After 20 minutes of the free-access procedure, the experimenter returned to the room and administered children a battery of self-report questionnaires assessing food preferences, and eating and weight-related attitudes and behaviors. Questionnaires were read to the girls to assure they comprehended the questions. Children were also asked what they ate between lunch and the beginning of the assessment, if anything. After completing the questionnaires, daughters had their height and weight measured.

Once the protocol was completed, mothers and daughters were debriefed together about the actual purpose of the experiment, and they were given time to ask questions. Girls were given a $20 payment card and mothers given a $15 payment card for their participation. After the family left the laboratory, every food offered for dinner and the free-access procedure was weighed and recorded to determine the total number of grams consumed. Each study appointment typically lasted 60-90 minutes.

3.2.1 Modifications to EAH laboratory paradigm

The laboratory procedure in the current study differed from the original paradigm used by Birch and colleagues, as well as other researchers, in several aspects. First, the length of time in the free-access procedure was extended from 10 to 20 minutes. In order not to influence children’s consumption, their preference for snack foods used in the procedure was assessed in conjunction
with their preference for other foods, rather than asking them to sample and rate each food immediately prior to being exposed to them in the free-access procedure. Furthermore, child fullness ratings were queried using a 5-point Likert scale, rather than a 3-point pictorial scale. In order to maximize use of all consented participants, the two children who reported hunger after finishing dinner were encouraged to eat until they were comfortably full so that they could participate in the free-access session. Additionally, the procedure measured EAH consumption following dinner, rather than lunch. Finally, several of the previous studies were structured such that children consumed either the test meal or the snacks during the free-access portion in a group with other participants (Hill et al., 2008; Moens & Braet, 2007). Given experimental evidence suggesting that the presence of a peer differentially affects overweight and non-overweight children’s food selection and consumption (Salvy, Kieffer, & Epstein, 2008), children completed all aspects of the present study individually.

3.3 MEASURES

3.3.1 Maternal measures

Demographic Questionnaire. This investigator-designed measure documented maternal and child age, race, ethnicity, education, income, child’s grade in school, family composition, marital status, mother’s relationship to participating child, and degree of financial hardship. Annual income and household size were used to calculate the family’s percentage above or below poverty levels according to 2008 or 2009 Federal guidelines (Leavitt).
**Height and weight.** Height and weight was measured using a calibrated digital scale and stadiometer after questionnaires were completed. Weight was measured to the nearest 0.1 kilogram, and height was measured to the nearest millimeter. From these measurements, body mass index (BMI; kg/m$^2$) was calculated. All participants were weighed in street clothes without shoes.

### 3.3.2 Child measures

**Height and Weight.** Height and weight were measured using a calibrated digital scale and stadiometer after questionnaires were completed. Weight was measured to the nearest 0.1 kilogram, and height was measured to the nearest millimeter. From these measurements, body mass index (BMI; kg/m$^2$) was calculated and converted to a BMI percentile (CDC, 2000). Based on current guidelines, participants were classified as overweight if their BMI was $\geq 85^{th}$ percentile. All participants were weighed in street clothes without shoes.

**Calorie intake during dinner.** Each food offered for dinner was weighed to the nearest gram using a digital scale before and after the child ate. The number of calories per gram of each food was calculated using manufacturer’s information. The difference between the pre- and post-dinner weights in grams was multiplied by the number of calories per gram and summed across foods to determine the total number of calories consumed during dinner.

In order to examine the proportion of calories consumed relative to each child’s energy needs, we calculated two types of estimated daily caloric requirements: Recommended Dietary Allowance (RDA) and Dietary Reference Intakes (DRI). The RDA is the average daily dietary nutrient intake level sufficient to meet the nutrient requirement of nearly all (97- 98%) healthy individuals within a particular age and gender group and was calculated based on the child’s age
and sex. The child’s DRI is the average dietary energy intake that is likely to maintain her energy balance calculated with the child’s age, sex, weight, height, and level of physical activity. In addition to absolute calories consumed during dinner, each child’s dinner calories were expressed as a proportion of her RDA and DRI.

*Calorie intake during free-access period.* Each food offered during the free-access session was weighed to the nearest gram using a digital scale before and after the child ate. The number of calories per gram of each food was calculated using manufacturer’s information. The difference between the pre- and post-session weights in grams was multiplied by the number of calories per gram and summed across foods to determine the total number of calories consumed during the free-access session. Similar to measures of calorie intake during dinner, free-access calories were also expressed as a proportion of RDA and DRI.

*Pubertal Development Scale (PDS;*Petersen, Crockett, Richards, & Boxer, 1988)). The Pubertal Development Scale is a five item self-report questionnaire constructed to measure pubertal status in children and adolescents. The measure is comprised of four items for which the child rates breast and pubic hair development, along with changes in skin and height, on a 4-point Likert scale. The final item asked girls to report whether they have begun menstruating and, if yes, the date of their first period. Menstrual status was dichotomized, such that premenarcheal girls were coded as “1” and postmenarcheal girls as “4” on the Likert scale (Petersen, Crockett, Richards, & Boxer, 1988). The mean of the five items was calculated to create an overall pubertal development score. For descriptive purposes, scores were converted to Tanner stages using an algorithm described by Brooks-Gunn and colleagues (1987) such that girls were classified into one of five stages: 1) prepubertal, 2) early pubertal, 3) midpubertal, 4) late pubertal, and 5) postpubertal. Among girls age 11-13 years, internal consistency and validity
of the PDS is good, with alpha coefficients ranging from .62 to .83 and correlations of PDS scores and physician ratings of pubertal status ranging from .64 to .67 (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Petersen, Crockett, Richards, & Boxer, 1988).

**Questionnaire on Eating and Weight Patterns-Adolescent version (QEWP-A; (W. G. Johnson, Grieve, Adams, & Sandy, 1999).** The QEWP-A is a self-report measure designed to identify children with eating disorders as described by the DSM-IV. It is derived from the adult version, the QEWP (Spitzer, Yanovski, & Marcus, 1993), and differs from the QEWP in that some of the language has been simplified to make it more appropriate for children and adolescents. Specifically, this measure is designed to assess criteria for Binge Eating Disorder. The QEWP-A appears to have adequate concurrent validity when correlated to measures of abnormal eating attitudes and depression (W. G. Johnson, Grieve, Adams, & Sandy, 1999), and has significant stability for males and females over a 3-week timeframe, although males’ reports appear to be more consistent over time compared with those of females (W. G. Johnson, Kirk, & Reed, 2001).

In order to ensure that all episodes of loss of control were documented, a modified protocol for the administration of the QEWP-A, used by Morgan and colleagues (2002), was followed. All children were asked about episodes of loss of control, even if they denied any episodes of overeating. This differed from the original instructions for QEWP-A administration, which stated that questions regarding loss of control should be skipped when the child denied any episodes of overeating. Several examples of loss of control were offered to the child if initially she did not appear to understand the concept. Furthermore, the child was asked about episodes of loss of control within the previous 1 month, 6 months, and within her lifetime. Finally, questions regarding inappropriate compensatory methods for weight control were not
restricted to the previous 3 months; rather, the child was asked if she ever engaged in those behaviors. As discussed earlier, loss of control appears to be of relatively greater importance to identifying episodes of disordered eating in children than does the amount of food consumed. Therefore, any reported eating episodes involving loss of control were classified as LOC episodes, regardless of whether the amount eaten was objectively “large.” A copy of this measure can be found in Appendix A.

*Food Preferences Questionnaire* (developed by the investigator for the present study) Participants completed this investigator-designed measure after the free-access period. Children were asked to rate on a 5-point Likert scale how much they prefer each of 20 listed foods. Included on the list were the 10 foods used in the free-access procedure and 10 other kinds of snack foods.

### 3.4 PRIMARY HYPOTHESES

The primary goals of this study were to document EAH in a racially and socioeconomically diverse sample of girls and to examine the relationships between child BMI, Eating in the Absence of Hunger, and loss of control over eating. Additionally, we aimed to explore the associations between certain child and family characteristics (e.g., race, BMI, SES) and eating in the absence of hunger and loss of control over eating.

We examined the following hypotheses:

**Hypothesis 1a.** Similar to previous findings in socioeconomically homogeneous samples of non-Hispanic White girls, we predicted that girls in the proposed study would consume calories in the absence of hunger. Given that this is the first study of which we are aware that
examines EAH in this population, we also examined the associations between EAH calories consumed and other potential covariates, including child race, SES, BMI, pubertal status, age, and maternal BMI.

**Hypothesis 1b.** We predicted that greater child BMI percentile would be associated with higher EAH intake, controlling for all identified covariates.

**Hypothesis 1c.** After controlling for BMI and relevant covariates, we hypothesized that there would be no racial differences in EAH intake between Black and White girls.

**Hypothesis 2a.** We predicted that higher child BMI percentile would be associated with increased likelihood of a previous loss of control (LOC) episode.

**Hypothesis 2b.** After controlling for BMI, we hypothesized that greater child EAH would be associated with increased likelihood of girls’ report of at least one episode of loss of control over eating.

### 3.4.1 Sample size estimation

Given that previous research on EAH has documented medium to large effect sizes, a conservative estimate of an effect size in the current study is .20. Power calculations were based on a multiple regression model with $\alpha = .05$ and power of .80. For Aim One, which has one predictor variable, a sample size of 50 is sufficient to detect an effect size of .16. For Aim Two, which includes two predictor variables, a sample size of 50 is sufficient to detect an effect size of .20. Therefore, we recruited and ran 51 girls through the study protocol.
3.4.2 Data analytic plan

All analyses were performed with SPSS 13.0. Data from predictor and outcome variables were examined for both univariate and multivariate outliers. One participant consumed an inordinately large amount of calories during the free-access session (2313 calories); examination of z-scores indicated that this data point was greater than 3 standard deviations above the mean intake. EAH intake from this participant was adjusted to fall 1.5 times the interquartile range above the 75\textsuperscript{th} percentile, a procedure advocated by Behrens (1997) and used in previous EAH research (Shomaker et al., 2010b). This adjustment was used because it minimizes the influence of the outlier on the characteristics of the distribution, minimally modifies the distribution overall, and avoids potential biases associated with the elimination of the outlier altogether. All analyses were conducted with the unadjusted data from this participant, without data from this participant, and with the participant’s data adjusted as described above. For all three methods, the outcomes of all analyses were the same and all post-hoc analyses indicated that this data point was not unduly influencing the outcomes. Thus, adjusted data from this participant were used for all analyses unless noted.

All of the children who reported a lifetime episode of loss of control indicated that at least one episode occurred within the previous month; thus, all children reporting at least one lifetime episode of loss of control over eating were collapsed into one group for analyses. Analyses that included EAH were conducted with EAH expressed both in absolute calories and as a proportion of the child’s estimated daily caloric needs derived from Dietary Reference Intake calculations to adjust for age and BMI-related differences in daily calorie needs. Four participants self-identified their race as something other than non-Hispanic White or Black (3 were Hispanic and 1 was Asian). Because several of our hypotheses were designed to examine
racial differences between White and Black girls, we excluded data from these four participants in all analyses that included race as a variable.

Data were examined to assure that the criteria needed to use parametric tests were fulfilled. Normality was assessed by examining skewness and kurtosis statistics, and conducting the Shapiro-Wilk and Kolmogorov-Smirnov tests. Given that the number of calories consumed during the free-access session was not normally distributed, square root transformations were computed for EAH data and the DRI-EAH percentages. The transformed data were used in all multiple regression analyses. Because logistic regression does not require normal distribution of independent variables, untransformed values of EAH were used for all logistic regression analyses. EAH data used for logistic regression analyses were expressed in 100-calorie increments in order to make interpretation of outcomes more clinically meaningful. The Variance Inflation Factor (VIF) was examined for all variables used in regression analyses and results indicated that there were not any multicollinear independent variables.

Each hypothesis was evaluated in the following manner. First, we reported descriptive statistics when applicable. Next, we examined correlations between putative predictor variables and the outcome variable of interest. Pearson correlations were used for continuous variables, and point-biserial correlations were examined for dichotomous variables. Predictor variables significantly associated with the outcome in univariate analyses were selected for inclusion as covariates in the respective regression model. Hierarchical multiple regression analyses were used to predict EAH intake, and logistic regression analyses were used to predict LOC episodes. For all regression equations with more than two predictor variables, covariates identified in univariate analyses were entered into the first block of the model, and the independent variables of interest for the respective hypotheses were entered into the next block. When regression
models yielded statistically significant predictor variables, analyses were repeated including the respective interaction terms. Following each regression analysis, statistics including Cook’s Distance, Mahalanobis distances, standardized DFBeta values, and studentized residuals were examined to identify influential cases and problems with the model fit.

In all analyses, the alpha level was set at .05, and two-tailed tests of significance were used.
4.0 RESULTS

4.1 PRIMARY HYPOTHESES

4.1.1 Hypothesis 1a. Description of EAH

First, calories consumed during dinner were examined. Girls consumed an average of 766.3 calories during dinner ($SD = 295.9$), which constituted a mean of 40.9% of their RDA ($SD = 16.2\%$) and 32.1% of their DRI ($SD = 10.3\%$). Additional information on calories consumed is presented in Table 7.
**Table 7:** Summary of primary outcome variables.

<table>
<thead>
<tr>
<th></th>
<th>Range</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>EAH Calories (unadjusted)</td>
<td>0 – 1159.40</td>
<td>339.74</td>
<td>283.29</td>
</tr>
<tr>
<td>Dinner Calories</td>
<td>227.75 – 1815.49</td>
<td>766.27</td>
<td>295.89</td>
</tr>
<tr>
<td>Total Calories</td>
<td>227.75 – 2164.75</td>
<td>1106.01</td>
<td>416.70</td>
</tr>
<tr>
<td>%RDA- EAH</td>
<td>0 – 66.63</td>
<td>18.17</td>
<td>15.13</td>
</tr>
<tr>
<td>%RDA- Dinner</td>
<td>12.65 – 100.86</td>
<td>40.86</td>
<td>16.23</td>
</tr>
<tr>
<td>%RDA- Total Calories</td>
<td>12.65 – 120.26</td>
<td>59.03</td>
<td>22.48</td>
</tr>
<tr>
<td>%DRI- EAH</td>
<td>0 – 49.23</td>
<td>14.53</td>
<td>12.23</td>
</tr>
<tr>
<td>(unadjusted)</td>
<td>0 – 98.20</td>
<td>15.49</td>
<td>16.27</td>
</tr>
<tr>
<td>%DRI- Dinner</td>
<td>9.15 – 62.88</td>
<td>32.07</td>
<td>10.27</td>
</tr>
<tr>
<td>%DRI- Total Calories</td>
<td>9.15 – 84.72</td>
<td>46.60</td>
<td>16.09</td>
</tr>
</tbody>
</table>

**EAH Intake by Percentile**

<table>
<thead>
<tr>
<th>Percentile</th>
<th>EAH Calories (adjusted)</th>
<th>EAH Calories Excluding Outlier</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>102.60</td>
<td>99.38</td>
</tr>
<tr>
<td>50</td>
<td>297.30</td>
<td>295.76</td>
</tr>
<tr>
<td>75</td>
<td>525.30</td>
<td>505.73</td>
</tr>
<tr>
<td>90</td>
<td>783.44</td>
<td>768.15</td>
</tr>
</tbody>
</table>

**Loss of Control**

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>10</td>
<td>19.6</td>
</tr>
<tr>
<td>No</td>
<td>41</td>
<td>80.4</td>
</tr>
</tbody>
</table>

*Note.* Unadjusted values of EAH data include the original value of the outlier.
On average, children consumed most of their dinner calories from the sub sandwich \((M = 555.7, SD = 247.3\) calories), beverage \((M = 98.0, SD = 66.3)\), and yogurt \((M = 68.9, SD = 74.4)\). Additional descriptive information regarding the types of foods consumed is summarized in Table 8.

**Table 8:** Mean dinner calories consumed by type.

<table>
<thead>
<tr>
<th>Food Type</th>
<th>Range</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sub Sandwich</td>
<td>191.92 – 1303.52</td>
<td>555.67</td>
<td>247.26</td>
</tr>
<tr>
<td>Carrots</td>
<td>0 – 51.88</td>
<td>11.57</td>
<td>10.80</td>
</tr>
<tr>
<td>Apples</td>
<td>0 – 150.28</td>
<td>20.15</td>
<td>36.39</td>
</tr>
<tr>
<td>Oranges</td>
<td>0 – 105.47</td>
<td>11.96</td>
<td>28.46</td>
</tr>
<tr>
<td>Yogurt</td>
<td>0 – 198.24</td>
<td>68.91</td>
<td>74.41</td>
</tr>
<tr>
<td>Beverage</td>
<td>0 – 270.67</td>
<td>98.01</td>
<td>66.25</td>
</tr>
</tbody>
</table>

As shown in Table 7, results indicated that girls’ free-access consumption varied widely \((M = 339.7 and SD = 283.3\) calories; range = 0 – 1159.4 calories). Excluding data from the outlier yielded an average consumption of 323.3 calories \((SD = 260.6;\) range = 0 – 893.8). All but three participants (6%) consumed calories during the free access session. On average, girls consumed 18.2% of their Recommended Daily Allowance of calories \((SD = 15.1\%;\) range = 0 – 66.6%) and 14.5% of their Dietary Reference Intake \((SD = 12.2\%;\) range = 0 – 49.2%) in the absence of hunger. Girls tended to consume more calories from the sweet snack foods relative to
the salty foods, as the mean caloric intake was highest for Starburst fruit chews \((M = 88.6, SD = 145.1\) calories), M & M chocolates \((M = 75.9, SD = 129.9)\), and licorice \((M = 68.2, SD = 88.1)\).

Additional data on snack food consumption are presented in Table 9.

**Table 9**: Mean EAH calories consumed by food type.

<table>
<thead>
<tr>
<th>Food Type</th>
<th>Range</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Popcorn</td>
<td>0 – 217.14</td>
<td>13.78</td>
<td>39.31</td>
</tr>
<tr>
<td>Pretzels</td>
<td>0 – 117.86</td>
<td>12.17</td>
<td>27.87</td>
</tr>
<tr>
<td>Potato Chips</td>
<td>0 – 294.64</td>
<td>16.81</td>
<td>49.65</td>
</tr>
<tr>
<td>Tortilla Chips</td>
<td>0 – 91.07</td>
<td>3.05</td>
<td>14.30</td>
</tr>
<tr>
<td>Nuts</td>
<td>0 – 631.43</td>
<td>18.21</td>
<td>89.59</td>
</tr>
<tr>
<td>Licorice</td>
<td>0 – 311.35</td>
<td>68.24</td>
<td>88.06</td>
</tr>
<tr>
<td>Starburst</td>
<td>0 – 640.00</td>
<td>88.63</td>
<td>145.14</td>
</tr>
<tr>
<td>M &amp; M’s</td>
<td>0 – 525.00</td>
<td>75.88</td>
<td>129.85</td>
</tr>
<tr>
<td>Fig Newtons</td>
<td>0 – 110.00</td>
<td>8.84</td>
<td>25.77</td>
</tr>
<tr>
<td>Cookies</td>
<td>0 – 504.33</td>
<td>56.76</td>
<td>111.25</td>
</tr>
</tbody>
</table>

To examine total caloric intake, we combined the number of calories consumed during the free-access session and dinner. On average, girls consumed a total of 1106.0 calories \((SD = 416.7;\) range = 227.8 – 2164.8). Their overall consumption accounted for an average of 59.1%
of their Recommended Daily Allowance ($SD = 22.5\%; \text{ range } = 12.7 - 120.3\%)$ and $46.6\%$ of their Dietary Reference Intake ($SD = 16.1\%; \text{ range } = 9.2 - 84.7\%)$.

Next, we evaluated bivariate correlations between EAH calories consumed (expressed in both the number of calories and as a percentage of DRI) and background variables, including child age, Tanner Stage, race, and level of poverty, and maternal BMI, age, and education. Results indicated that there were not any significant bivariate correlations between EAH calories consumed or EAH-DRI percentage and any of the demographic variables. A complete correlation matrix is depicted in Table 10.
**Table 10**: Pearson and point-biserial correlations between all variables.

<table>
<thead>
<tr>
<th></th>
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<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. BMI Percentile</td>
<td>.13</td>
<td>-</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Tanner Stage</td>
<td>.54**</td>
<td>.32*</td>
<td>.25</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Race</td>
<td>-.14</td>
<td>.17</td>
<td>.11</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. % Above Poverty Level</td>
<td>.25</td>
<td>-.01</td>
<td>.06</td>
<td>-.43**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Maternal BMI</td>
<td>-.08</td>
<td>.36*</td>
<td>-.07</td>
<td>.32*</td>
<td>-.30*</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>7. Maternal Age</td>
<td>.10</td>
<td>-.07</td>
<td>-.13</td>
<td>.33*</td>
<td>-.17</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Education</td>
<td>.01</td>
<td>.12</td>
<td>.10</td>
<td>-.13</td>
<td>.63**</td>
<td>-.20</td>
<td>.38**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. EAH</td>
<td>.02</td>
<td>.02</td>
<td>.04</td>
<td>.16</td>
<td>-.09</td>
<td>-.09</td>
<td>-.10</td>
<td>-.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. DRI- EAH</td>
<td>-.03</td>
<td>-.04</td>
<td>.00</td>
<td>.14</td>
<td>-.08</td>
<td>-.14</td>
<td>-.09</td>
<td>.00</td>
<td>.99**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Dinner</td>
<td>.09</td>
<td>.44**</td>
<td>.16</td>
<td>.41**</td>
<td>-.07</td>
<td>.42**</td>
<td>-.07</td>
<td>-.12</td>
<td>.04</td>
<td>-.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. DRI- Dinner</td>
<td>.02</td>
<td>.31*</td>
<td>.08</td>
<td>.32*</td>
<td>-.08</td>
<td>.29*</td>
<td>-.07</td>
<td>-.15</td>
<td>.05</td>
<td>.02</td>
<td>.95**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. LOC</td>
<td>.08</td>
<td>.30*</td>
<td>.17</td>
<td>.57**</td>
<td>-.22</td>
<td>.07</td>
<td>-.03</td>
<td>-.02</td>
<td>.34*</td>
<td>.27</td>
<td>.37**</td>
<td>.27</td>
<td></td>
</tr>
</tbody>
</table>

Note. * = p < .05,  ** = p < .01. Race: “0” = White, non-Hispanic, “1” = Black or Biracial (n = 47)
4.1.2 Hypothesis 1b. Child BMI and EAH intake

As shown in Table 10, results of correlation analyses between putative predictor variables and EAH were not significant. Child BMI percentile was not associated with EAH intake ($r = .02; p = .90$) or EAH-DRI ($r = -.04; p = .76$) in univariate analyses.

A regression analysis was conducted with child BMI percentile as the only predictor of EAH intake. Results indicated that the model did not significantly predict EAH intake, $F(1, 49) = 0.00, p = .88, R^2 = .01$. The regression model using BMI percentile to predict EAH-DRI was also not significant, $F(1, 49) = 0.16, p = .69, R^2 = .00$.

4.1.3 Hypothesis 1c. Race and EAH

As noted earlier, there were not any significant associations between EAH and potential covariates. Race was not significantly correlated with EAH ($r = .16; p = .28$) or with EAH-DRI ($r = .14; p = .37$).

We conducted a simultaneous multiple regression analysis including race as the only predictor of EAH. The model was not significant in predicting EAH intake, $F(1, 45) = 1.31, p = .26, R^2 = .03$. We conducted another multiple regression analysis using race to predict EAH-DRI. Results indicated that the model was not significant in predicting EAH-DRI, $F(1, 45) = 1.55, p = .22, R^2 = .03$. 
4.1.4 Hypothesis 2a. BMI and Loss of Control

In the present study, 10 girls (19.6%) reported at least one lifetime episode of loss of control (LOC). All of the girls endorsing lifetime LOC had at least one episode in the 30 days immediately prior to the day of study participation.

We examined correlations between predictor variables and LOC to identify covariates to include in the analysis. As shown in Table 10, report of at least one lifetime episode of loss of control over eating was associated with higher child BMI percentile ($r = .30; p = .034$) and child race ($r = .57; p < .001$), with Black girls more likely to report an episode of LOC relative to their White peers.

Next, we conducted a hierarchical logistic regression analysis to predict LOC using child BMI percentile. Race was entered as a covariate into the first block of the model, and child BMI percentile was entered into the second block to predict LOC. As shown in Table 11, the first block of the model was significant in predicting LOC, $\chi^2(df = 1, N = 47) = 13.58, p = .00$. The addition of child BMI percentile to the model approached clinical significance in improving the model’s predictive ability ($p = .051$), and the overall model was significant in predicting loss of control, $\chi^2(df = 2, N = 47) = 17.40, p = .000$. Race ($Exp(B) = 20.23, p = .002$) was a significant predictor of LOC, such that Black girls were more likely to report a previous LOC episode. Child BMI percentile approached significance in the model, indicating that there was a trend towards higher BMI percentile and increased likelihood of prior LOC ($Exp(B) = 1.04, p = .08$).
Table 11: Hypothesis 2a: Logistic regression using BMI percentile to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Exp(B)</th>
<th>Wald</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>2.96</td>
<td>0.87</td>
<td>19.25</td>
<td>11.56</td>
<td>.001</td>
</tr>
<tr>
<td>2</td>
<td>Race</td>
<td>3.01</td>
<td>0.96</td>
<td>20.23</td>
<td>9.89</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>Child BMI Percentile</td>
<td>.04</td>
<td>0.02</td>
<td>1.04</td>
<td>3.06</td>
<td>.080</td>
</tr>
</tbody>
</table>

Model, Block 1: $\chi^2(df = 1, N = 47) = 13.58, p = .000$, Cox and Snell $R^2 = .25$; Nagelkerke $R^2 = .39$

Block 2: $\chi^2(df = 1, N = 47) = 3.81, p = .051$

Overall Model: $\chi^2(df = 2, N = 47) = 17.40, p = .000$, Cox and Snell $R^2 = .31$; Nagelkerke $R^2 = .48$; Hosmer-Lemeshow $\chi^2(df = 7) = 2.29; p = .94$

Given that child BMI percentile approached significance in the model with race in predicting LOC, we conducted an exploratory logistic regression analysis to test for an interaction between race and BMI percentile. Race and BMI percentile were entered into the first block of the model, and a race x BMI percentile term was entered into the second block to predict LOC. As shown in Table 12, the omnibus model was significant in predicting LOC, $\chi^2(df = 3, N = 47) = 18.38, p = .000$. However, none of the variables were significant predictors of LOC in the model.
Table 12: Hypothesis 2a: Logistic regression using race, BMI, and race x BMI interaction to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Exp(B)</th>
<th>Wald</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>3.01</td>
<td>0.96</td>
<td>20.23</td>
<td>9.89</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>BMI Percentile</td>
<td>0.04</td>
<td>0.02</td>
<td>1.04</td>
<td>3.06</td>
<td>.080</td>
</tr>
<tr>
<td></td>
<td>Race x BMI</td>
<td>-0.06</td>
<td>0.07</td>
<td>0.94</td>
<td>0.73</td>
<td>.392</td>
</tr>
</tbody>
</table>

Block 1: $\chi^2(df = 2, N = 47) = 17.40, p = .000$, Cox and Snell $R^2 = .31$; Nagelkerke $R^2 = .48$

Block 2: $\chi^2(df = 1, N = 47) = 0.98, p = .322$

Overall Model: $\chi^2(df = 3, N = 47) = 18.38, p = .000$, Cox and Snell $R^2 = .32$; Nagelkerke $R^2 = .50$; Hosmer-Lemeshow $\chi^2(df = 7) = 1.03; p = .994$

4.1.5 Hypothesis 2b. EAH and LOC

First, bivariate correlation analyses were examined to identify potential covariates for the regression analysis. As noted in the previous hypothesis, race, child BMI percentile, and EAH intake ($r = .34, p = .016$) were the only background and outcome variables correlated with LOC. The correlation for EAH-DRI and LOC approached significance ($r = .27; p = .06$).

Next, a hierarchical logistic regression analysis was conducted to examine whether EAH predicted LOC. Race and BMI percentile were entered into the first block of the model. In the second block, EAH intake was entered to examine whether it significantly contributed to the prediction of LOC. The addition of EAH to the model significantly improved its predictive ability ($p = .003$), and the overall model was significant in predicting LOC, $\chi^2(df = 3, N = 47)$ =
26.08, \( p = .000 \). As shown in Table 13, results indicated that EAH consumption (\( Exp(B) = 1.72;\ p = .016 \)) was associated with loss of control, such that with each 100-calorie increase in EAH consumption during the free access procedure, children were 1.72 times more likely to report loss of control. Additionally, Black girls (\( Exp(B) = 38.27;\ p = .004 \)) and higher BMI percentile (\( Exp(B) = 1.08;\ p = .043 \)) were associated with increased likelihood of LOC. On average, girls who endorsed LOC consumed 531.22 (SD = 362.68) calories in the absence of hunger, and girls who denied LOC consumed 283.22 (SD = 220.20) calories.

**Table 13:** Hypothesis 2b: Logistic regression using EAH to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>( B )</th>
<th>( SE ) ( B )</th>
<th>( Exp(B) )</th>
<th>Wald</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>3.01</td>
<td>0.96</td>
<td>20.23</td>
<td>9.89</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>BMI Percentile</td>
<td>0.04</td>
<td>0.02</td>
<td>1.04</td>
<td>3.06</td>
<td>.080</td>
</tr>
<tr>
<td>2</td>
<td>Race</td>
<td>3.65</td>
<td>1.26</td>
<td>38.27</td>
<td>8.35</td>
<td>.004</td>
</tr>
<tr>
<td></td>
<td>BMI Percentile</td>
<td>0.08</td>
<td>0.04</td>
<td>1.08</td>
<td>4.08</td>
<td>.043</td>
</tr>
<tr>
<td></td>
<td>EAH Intake</td>
<td>0.54</td>
<td>0.23</td>
<td>1.72</td>
<td>5.79</td>
<td>.016</td>
</tr>
</tbody>
</table>

Model, Block 1: \( \chi^2(df = 2, \ N = 47) = 17.40, \ p = .000 \), Cox and Snell \( R^2 = .31 \); Nagelkerke \( R^2 = .48 \)

Block 2: \( \chi^2(df = 1, \ N = 47) = 8.68, \ p = .003 \)

Overall Model: \( \chi^2(df = 3, \ N = 47) = 26.08, \ p = .000 \), Cox and Snell \( R^2 = .43 \); Nagelkerke \( R^2 = .66 \); Hosmer-Lemeshow \( \chi^2(df = 7) = 2.70; \ p = .911 \)

We conducted the same regression analysis using EAH-DRI, instead of EAH, to predict LOC. The omnibus model was significant in predicting LOC, \( \chi^2(df = 3, \ N = 47) = 24.68, \ p = .000 \). EAH-DRI (\( Exp(B) = 1.13;\ p = .023 \)) was associated with LOC, such that for every one
percent increase in EAH-DRI, girls were 1.13 times more likely to report LOC. As shown in Table 14, race and BMI percentile were also significant in the regression equation.

**Table 14:** Hypothesis 2b: Logistic regression using EAH-DRI to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Exp(B)</th>
<th>Wald</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>3.01</td>
<td>0.96</td>
<td>20.23</td>
<td>9.89</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>BMI Percentile</td>
<td>0.04</td>
<td>0.02</td>
<td>1.04</td>
<td>3.06</td>
<td>.080</td>
</tr>
<tr>
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<td>1.20</td>
<td>33.84</td>
<td>8.66</td>
<td>.003</td>
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<td></td>
<td>BMI Percentile</td>
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<td>0.04</td>
<td>1.09</td>
<td>4.35</td>
<td>.037</td>
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<tr>
<td></td>
<td>EAH-DRI</td>
<td>0.12</td>
<td>0.05</td>
<td>1.13</td>
<td>5.18</td>
<td>.023</td>
</tr>
</tbody>
</table>

For the purposes of model building, we were interested in further examining the relationship between EAH, race, and child BMI percentile in predicting LOC. Given that the main effects of child BMI percentile, race, and EAH were all significant in predicting LOC, we conducted two additional regression analyses to examine whether there was an interaction between race and EAH or an interaction between BMI percentile and EAH in the prediction of LOC. First, we tested the interaction between race and EAH by conducting a hierarchical logistic regression analysis. Race, BMI percentile, and EAH intake were entered into the first block of
the model, and the EAH x race interaction term was entered into the second block. As shown in Table 15, the overall model was significant in predicting LOC, $\chi^2(df = 4, N = 47) = 35.14, p = .000$. The addition of the EAH x race interaction term significantly improved the model’s prediction of LOC ($p = .003$). However, none of the variables were significant in predicting LOC.

**Table 15:** Hypothesis 2b: Logistic regression using EAH, race, BMI, and race x EAH interaction to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Exp(B)</th>
<th>Wald</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>3.65</td>
<td>1.26</td>
<td>38.27</td>
<td>8.35</td>
<td>.004</td>
</tr>
<tr>
<td></td>
<td>BMI Percentile</td>
<td>0.08</td>
<td>0.04</td>
<td>1.08</td>
<td>4.08</td>
<td>.043</td>
</tr>
<tr>
<td></td>
<td>EAH Intake</td>
<td>0.54</td>
<td>0.23</td>
<td>1.72</td>
<td>5.79</td>
<td>.016</td>
</tr>
<tr>
<td>2</td>
<td>Race</td>
<td>1235.85</td>
<td>30787.29</td>
<td>.00</td>
<td>.00</td>
<td>.968</td>
</tr>
<tr>
<td></td>
<td>BMI Percentile</td>
<td>0.03</td>
<td>0.04</td>
<td>1.03</td>
<td>.80</td>
<td>.373</td>
</tr>
<tr>
<td></td>
<td>EAH Intake</td>
<td>162.11</td>
<td>4040.85</td>
<td>.00</td>
<td>.00</td>
<td>.968</td>
</tr>
<tr>
<td></td>
<td>EAH x Race Interaction</td>
<td>-162.03</td>
<td>4040.85</td>
<td>.00</td>
<td>.00</td>
<td>.968</td>
</tr>
</tbody>
</table>

*Model, Block 1: $\chi^2(df = 3, N = 47) = 26.08, p = .000$, Cox and Snell $R^2 = .43$; Nagelkerke $R^2 = .66$*

*Block 2: $\chi^2(df = 1, N = 47) = 9.07, p = .003$*

*Overall Model: $\chi^2(df = 4, N = 47) = 35.14, p = .000$, Cox and Snell $R^2 = .53$; Nagelkerke $R^2 = .82$; Hosmer-Lemeshow $\chi^2 (df = 4) = 0.79; p = .939$*

Next, we tested the interaction between BMI percentile and EAH by conducting a hierarchical logistic regression analysis. Race, child BMI percentile, and EAH intake were
entered into the first block of the model, and the interaction between BMI percentile and EAH was entered into the second block of the model. Results indicated that the overall model was significant in predicting LOC, \((df = 4, N = 47) = 27.65, p = .000\). However, the addition of the BMI x EAH interaction term did not significantly contribute to the model \((p = .210)\). As shown in Table 16, race \((Exp(B) = 86.12; p = .005)\) was the only variable in the model that was significant in predicting LOC.

**Table 16**: Hypothesis 2b: Logistic regression using EAH, race, BMI, and BMI x EAH interaction to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Exp(B)</th>
<th>Wald</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>3.65</td>
<td>1.26</td>
<td>38.27</td>
<td>8.35</td>
<td>.004</td>
</tr>
<tr>
<td></td>
<td>BMI Percentile</td>
<td>0.08</td>
<td>0.04</td>
<td>1.08</td>
<td>4.08</td>
<td>.043</td>
</tr>
<tr>
<td></td>
<td>EAH Intake</td>
<td>0.54</td>
<td>0.23</td>
<td>1.72</td>
<td>5.79</td>
<td>.016</td>
</tr>
<tr>
<td>2</td>
<td>Race</td>
<td>4.46</td>
<td>1.59</td>
<td>86.12</td>
<td>7.89</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td>BMI Percentile</td>
<td>0.02</td>
<td>0.06</td>
<td>1.02</td>
<td>0.07</td>
<td>.792</td>
</tr>
<tr>
<td></td>
<td>EAH Intake</td>
<td>-0.51</td>
<td>0.80</td>
<td>0.60</td>
<td>0.40</td>
<td>.527</td>
</tr>
<tr>
<td></td>
<td>EAH x BMI</td>
<td>0.01</td>
<td>0.01</td>
<td>1.01</td>
<td>1.56</td>
<td>.211</td>
</tr>
</tbody>
</table>

*Model, Block 1*: \(\chi^2(df = 3, N = 47) = 26.08, p = .000\), Cox and Snell \(R^2 = .43\); Nagelkerke \(R^2 = .66\)

*Block 2*: \(\chi^2(df = 1, N = 47) = 1.57, p = .210\)

*Overall Model*: \(\chi^2(df = 4, N = 47) = 27.65, p = .000\), Cox and Snell \(R^2 = .45\); Nagelkerke \(R^2 = .69\); Hosmer-Lemeshow \(\chi^2(df = 7) = 0.58; p = .999\)
4.2 EXPLORATORY ANALYSES

4.2.1 Maternal BMI

Previous studies have used maternal BMI as an index of child obesity risk (Faith et al., 2006; Kral et al., 2010). For exploratory purposes, we conducted analyses to examine the role of maternal BMI, along with race and child BMI percentile, in the prediction of LOC. Race and maternal BMI percentile were entered into the first block of the model, and child BMI percentile was entered into the second block to predict LOC. As shown in Table 17, the overall model was significant in predicting loss of control, $\chi^2(df = 3, N = 47) = 23.43, p = .000$. Maternal BMI ($Exp(B) = 0.81, p = .043$) was significant in predicting LOC. Additionally, race ($Exp(B) = 149.96, p = .003$) and child BMI percentile ($Exp(B) = 1.10, p = .025$) were significant predictors in the model, such that Black girls and those with higher BMI were more likely to report a previous LOC episode.

Table 17: Exploratory Hypothesis 2a: Logistic regression using BMI percentile, maternal BMI, and race to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>$B$</th>
<th>$SE, B$</th>
<th>$Exp(B)$</th>
<th>Wald</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>3.36</td>
<td>1.03</td>
<td>28.90</td>
<td>10.76</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td>Maternal BMI</td>
<td>-0.06</td>
<td>0.06</td>
<td>0.95</td>
<td>0.89</td>
<td>.347</td>
</tr>
<tr>
<td>2</td>
<td>Race</td>
<td>5.01</td>
<td>1.71</td>
<td>149.96</td>
<td>8.59</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>Maternal BMI</td>
<td>-0.21</td>
<td>0.10</td>
<td>0.81</td>
<td>4.10</td>
<td>.043</td>
</tr>
<tr>
<td></td>
<td>Child BMI Percentile</td>
<td>0.10</td>
<td>0.04</td>
<td>1.10</td>
<td>5.06</td>
<td>.025</td>
</tr>
</tbody>
</table>

Model, Block 1: $\chi^2(df = 2, N = 47) = 14.53, p = .001$, Cox and Snell $R^2 = .27$; Nagelkerke $R^2 = .41$
Block 2: $\chi^2(df = 1, N = 47) = 8.90, p = .003$

*Overall Model:* $\chi^2(df = 3, N = 47) = 23.43, p = .000$, Cox and Snell $R^2 = .39$; Nagelkerke $R^2 = .61$; Hosmer-Lemeshow $\chi^2 (df = 7) = 11.47; p = .119$

We were interested in further examining the relationship between race, child BMI percentile, and maternal BMI in predicting LOC. Given that the main effects of maternal BMI, child BMI percentile, and race were all significant in predicting LOC, we conducted an additional regression analysis to examine whether there was an interaction between race and child BMI percentile in a model that included maternal BMI. Race, child BMI percentile, and maternal BMI were entered into the first block of the model, and the interaction between race and child BMI was entered into the second block to predict LOC. As depicted in Table 18, the model was significant in predicting LOC, $\chi^2(df = 4, N = 47) = 23.91, p = .000$. The contribution of maternal BMI approached significance ($Exp(B) = 0.82, p = .052$); however, none of the other variables in the model were significant predictors of LOC.
Table 18: Exploratory Hypothesis 2a: Logistic regression using BMI percentile, maternal BMI, race, and race x BMI interaction to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Exp(B)</th>
<th>Wald</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>5.01</td>
<td>1.71</td>
<td>149.96</td>
<td>8.59</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>Child BMI Percentile</td>
<td>0.10</td>
<td>0.04</td>
<td>1.10</td>
<td>5.06</td>
<td>.025</td>
</tr>
<tr>
<td></td>
<td>Maternal BMI</td>
<td>-0.21</td>
<td>0.10</td>
<td>0.81</td>
<td>4.10</td>
<td>.043</td>
</tr>
<tr>
<td>2</td>
<td>Race</td>
<td>9.87</td>
<td>8.22</td>
<td>19400.73</td>
<td>1.44</td>
<td>.230</td>
</tr>
<tr>
<td></td>
<td>Child BMI Percentile</td>
<td>0.14</td>
<td>0.09</td>
<td>1.15</td>
<td>2.37</td>
<td>.124</td>
</tr>
<tr>
<td></td>
<td>Maternal BMI</td>
<td>-0.19</td>
<td>0.10</td>
<td>0.82</td>
<td>3.78</td>
<td>.052</td>
</tr>
<tr>
<td></td>
<td>Race x BMI Percentile</td>
<td>-0.06</td>
<td>0.09</td>
<td>0.94</td>
<td>0.40</td>
<td>.526</td>
</tr>
</tbody>
</table>

Model, Block 1: $\chi^2(df = 3, N = 47) = 23.43, p = .000$, Cox and Snell $R^2 = .39$; Nagelkerke $R^2 = .61$

Block 2: $\chi^2(df = 1, N = 47) = 0.49, p = .485$

Overall Model: $\chi^2(df = 4, N = 47) = 23.91, p = .000$, Cox and Snell $R^2 = .40$; Nagelkerke $R^2 = .62$; Hosmer-Lemeshow $\chi^2(df = 7) = 3.67; p = .816$

We conducted additional analyses to examine the role of maternal BMI and EAH, along with race and child BMI percentile, in the prediction of LOC. Race, child BMI percentile, and maternal BMI percentile were entered into the first block of the model, and EAH intake was entered into the second block to predict LOC. As shown in Table 19, the overall model was significant in predicting loss of control, $\chi^2(df = 4, N = 47) = 35.78, p = .000$. There were trends approaching statistical significance for race ($Exp(B) = 221984.10, p = .059$), child BMI percentile ($Exp(B) = 1.30, p = .059$), and EAH intake ($Exp(B) = 4.20, p = .079$) in the model. Maternal BMI was not significant ($Exp(B) = 0.52, p = .111$).
Table 19: Exploratory Hypothesis 2b: Logistic regression using race, child BMI, maternal BMI, and EAH to predict LOC.

<table>
<thead>
<tr>
<th>Block</th>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>Exp(B)</th>
<th>Wald</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Race</td>
<td>5.01</td>
<td>1.71</td>
<td>149.96</td>
<td>8.59</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>Child BMI Percentile</td>
<td>0.10</td>
<td>0.04</td>
<td>1.10</td>
<td>5.06</td>
<td>.025</td>
</tr>
<tr>
<td></td>
<td>Maternal BMI</td>
<td>-0.21</td>
<td>0.10</td>
<td>0.81</td>
<td>4.10</td>
<td>.043</td>
</tr>
<tr>
<td>2</td>
<td>Race</td>
<td>12.31</td>
<td>6.51</td>
<td>221984.10</td>
<td>3.58</td>
<td>.059</td>
</tr>
<tr>
<td></td>
<td>Child BMI Percentile</td>
<td>0.26</td>
<td>0.14</td>
<td>1.30</td>
<td>3.56</td>
<td>.059</td>
</tr>
<tr>
<td></td>
<td>Maternal BMI</td>
<td>-0.66</td>
<td>0.41</td>
<td>0.52</td>
<td>2.54</td>
<td>.111</td>
</tr>
<tr>
<td></td>
<td>EAH Intake</td>
<td>1.44</td>
<td>0.82</td>
<td>4.20</td>
<td>3.09</td>
<td>.079</td>
</tr>
</tbody>
</table>

Model, Block 1: $\chi^2 (df = 3, N = 47) = 23.43, p = .000$, Cox and Snell $R^2 = .39$; Nagelkerke $R^2 = .61$

Block 2: $\chi^2 (df = 1, N = 47) = 12.36, p = .000$

Overall Model: $\chi^2 (df = 4, N = 47) = 35.78, p = .000$, Cox and Snell $R^2 = .53$; Nagelkerke $R^2 = .83$; Hosmer-Lemeshow $\chi^2 (df = 7) = 3.12; p = .874$

The analyses presented in Tables 15 and 16 that included race, child BMI percentile, EAH, and the interaction terms (race x EAH and BMI x EAH, respectively) were repeated including maternal BMI in the first block of the model. Results indicated that the omnibus models were significant in predicting LOC; however, none of the individual predictors were significant for any of the models.
5.0 DISCUSSION

The present investigation examined Eating in the Absence of Hunger and Loss of Control eating in a sample of 10-13 year-old Black and White girls. The major finding was that the number of EAH calories consumed was associated with increased likelihood of a previous LOC episode. In addition, Black girls and those with higher BMI were more likely to report LOC. Previous associations between BMI and EAH were not supported in this sample. All of the major findings will be discussed in turn below, followed by a review of study limitations and suggestions for future research.

5.1 REVIEW OF FINDINGS

5.1.1 Eating in the Absence of Hunger

As expected, girls in this sample consumed calories in the absence of hunger. Caloric intake during the free-access session was slightly higher or considerably higher than amounts documented in studies comprised of children in similar age groups (Butte et al., 2007; Fisher et al., 2007; Shomaker et al., 2010b). Almost all of the children (94%) in the study consumed calories during the free-access session, a proportion that is higher than at least one other study (Moens & Braet, 2007) but comparable to another (Hill et al., 2008). Average dinner intake was
comparable to amounts documented in previous studies (Fisher et al., 2007; Shomaker et al., 2010b).

Although most children consumed calories in the absence of hunger, the amount consumed varied widely. Variability of EAH calorie consumption was considerably greater in this study relative to that which has been documented in previous research (Birch & Fisher, 2000; Birch, Fisher, & Davison, 2003; Fisher & Birch, 1999a; Shomaker et al., 2010b). The only study of which we are aware that reported a somewhat comparably high number of calories and variability in EAH intake was conducted in a sample of 879 Hispanic American boys and girls aged 4-19 (Butte et al., 2007). Children consumed 389 ± 224 calories in the absence of hunger, and their consumption did not predict weight gain over time.

There are several possible reasons why girls in the present study consumed a relatively large amount of calories. We provided considerably larger portions, amounting to approximately 2-4 times more available calories, than were offered in previous free-access procedures. Research has shown that children of all weight strata eat more when offered greater portion sizes and greater variety (Ello-Martin, Ledikwe, & Rolls, 2005; Fisher, Arreola, Birch, & Rolls, 2007; Fisher, Liu, Birch, & Rolls, 2007). Thus, it is possible that, relative to participants in other studies, girls in this study ate more calories because they were offered significantly more food. Additionally, findings from several EAH studies suggested that girls may limit their EAH intake because they are concerned about being perceived as overeating (Hill et al., 2008; Moens & Braet, 2007). Theoretically this concern could make girls less likely to consume a large amount of food taken from smaller portions of snack foods, as this excess consumption would be apparent to others. Similarly, children in several previous studies completed the free-access procedure in the presence of peers or family members. Thus, the relatively smaller portions of
snack foods offered and the presence of others during the free-access session in previous EAH research may have constrained intake.

The considerable degree of variability in EAH intake in the present study may have been due, at least in part, to sample heterogeneity in race, age, and pubertal development. Many of the studies examining EAH have been conducted in fairly homogenous samples with respect to race, age, and SES. Thus, it is possible that studies comprised of more heterogeneous samples may find greater variability in EAH intake. Future research should attempt to elucidate potential influences of demographic variables on levels of EAH.

Notably, children consumed a considerable percentage, approximately 15%, of their individual daily caloric needs during the free-access session. Given that participants were reportedly not hungry at the time, intake of that magnitude is significant. Furthermore, girls consumed almost half of their daily calorie needs during dinner and the free-access session combined. If this pattern of eating generalizes to girls’ environments, it could potentially result in excess energy intake and development of weight problems over time. The present study did not assess whether children adjusted their intake at subsequent meals to compensate for the excess calories consumed during the free-access session. Future studies should measure children’s intake over a longer period of time (e.g., 24 hours) to determine the degree to which children adjust their intake, if at all.

5.1.2 Body Mass Index and Eating in the Absence of Hunger

In the present study, there were not any demographic variables associated with EAH in univariate analyses. In contrast to previous research, child BMI was not associated with EAH intake in this sample. More consistent findings of an association between BMI and EAH have
tended to be with younger children (Birch, Fisher, & Davison, 2003; Fisher & Birch, 1999a, 2002), males or mixed-sex participants (Faith et al., 2006; Fisher et al., 2007; Hill et al., 2008; Shomaker et al., 2010b), demographically homogenous samples (Birch, Fisher, & Davison, 2003; Fisher et al., 2007), and involved a free-access session conducted individually rather than in a dyad or group setting (Hill et al., 2008; Moens & Braet, 2007).

Findings from the current study add to a growing body of literature that has not documented an association between BMI and EAH in girls. Many of the previous studies that documented a relationship between weight and EAH made comparisons between normal weight and overweight children, or between children grouped into high-or low-risk for obesity based on parental weight (Birch, Fisher, & Davison, 2003; Fisher & Birch, 2002; Francis & Birch, 2005; Moens & Braet, 2007; Shunk & Birch, 2004). Very few studies have found a relationship between EAH and a continuous measure of weight or adiposity (Butte et al., 2007; Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Hill et al., 2008), and it is unclear whether findings from the Cutting and Butte studies were driven by differences between overweight and normal weight children. Furthermore, children in the present study had a relatively high BMI percentile on average. Although similarly high BMIs have been documented in racially diverse samples (Vander Wal, 2004), the constrained range of BMI may have limited our ability to detect an association. Future studies should examine whether there is an association between EAH and a continuous measure of BMI that spans the entire weight spectrum.

Another possible reason we did not find an association is the relatively older age of participants in our study. It is conceivable that the association between EAH and BMI diminishes or disappears as children grow older. Research suggests that variability in EAH may be more prone to environmental influences for girls relative to boys, particularly during the early
stages of pubertal development (Faith et al., 2006). As children approach adolescence, the nature of environmental influences on eating behavior likely changes and expands as their independent access to food increases. In younger samples of girls, relatively stronger relationships between EAH and BMI have been documented for children whose mothers employ more restrictive feeding practices (Birch, Fisher, & Davison, 2003; Fisher & Birch, 2002), especially among those who were overweight or had overweight mothers at age 5 years. Thus it is possible that parental feeding might mediate the relationship between EAH and weight. Given that parents’ role in controlling and modifying their child’s food environment tends to decrease as children proceed through middle childhood to adolescence, it is conceivable that other factors may affect the relationship between EAH and weight.

Interpreting associations between weight and EAH has also been complicated by findings of gender differences in studies including girls and boys. As discussed earlier, some studies have found associations between weight and EAH in girls but not boys (Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Fisher & Birch, 1999a), others have found relationships in boys but not girls (Faith et al., 2006; Moens & Braet, 2007), and a recent study in Hispanic American children aged 4-19 found no gender differences (Fisher et al., 2007). In studies that found an effect only for girls, the suggested mechanism for the association was girls’ increased responsiveness to parental restriction of snack foods relative to boys (Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Fisher & Birch, 1999a), which theoretically resulted in greater disinhibition in the presence of similar foods. Studies that found an effect only for boys cited girls’ susceptibility to social desirability as a factor possibly limiting overweight girls’ intake (Hill et al., 2008; Moens & Braet, 2007). Our study did not evaluate whether these, or other, variables affected intake during the free-access procedure. Given a growing body of equivocal findings related to weight
and EAH, future research should focus on identifying possible mediators and moderators in diverse samples.

5.1.3 Race and Eating in the Absence of Hunger

As expected, the number of calories consumed during the free-access session did not differ significantly between Black and White girls. A recent study that examined EAH in a sample of older male and female adolescents also did not find any racial differences in EAH consumption (Shomaker et al., 2010b). This is the first study of which we are aware to examine racial differences between Black and White children in EAH intake in a sample comprised exclusively of girls during middle childhood. Our study lends additional support for EAH as a cross-cultural phenomenon (Fisher et al., 2007). Additional research is needed to evaluate determine whether these findings extend to racially diverse samples of younger children and males.

5.1.4 Loss of Control eating

Rates of LOC in the current study (19.6%) were comparable to those found in similar samples of children across the weight spectrum (Carter, Stewart, & Fairburn, 2001; Tanofsky-Kraff et al., 2004; Theim et al., 2007). This is noteworthy because it provides additional evidence that LOC is not uncommon, even in non-clinical samples. Black girls were significantly more likely to endorse an episode of LOC than were their White peers. Previous research has generally found comparable rates of binge and LOC eating in Black and White girls (Morgan et al., 2002; Shisslak et al., 2006; Shomaker et al., 2010a; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2004). The prevalence of LOC in this diverse sample
further highlights the importance of evaluating children from all racial backgrounds for disordered eating behaviors.

All of the girls who endorsed an episode of LOC within the one month prior to assessment also endorsed another episode in the previous six months. However, we did not examine whether the frequency of LOC episodes was associated with any of the demographic or outcome variables. Future studies on LOC, particularly those that include EAH as a variable of interest, should examine whether higher frequency of LOC episodes is associated with greater intake during the free-access session.

5.1.5 BMI and Loss of Control

BMI was correlated with LOC in univariate analyses and predictive of LOC in multivariate models when controlling for race and maternal BMI. This association is consistent with numerous other studies in children (Decaluwe & Braet, 2003; Morgan et al., 2002; Shisslak et al., 1998; Tanofsky-Kraff et al., 2004). Given that this is a non-clinical sample with children of varying weight strata, the fairly high prevalence of LOC eating episodes and their relation to increased BMI is noteworthy. Loss of control has been associated with increases in shape and weight concerns, emotional eating, questionnaire measurements of eating in the absence of hunger, and general psychopathology (Morgan et al., 2002; Shomaker et al., 2010a; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2008; Tanofsky-Kraff et al., 2007b). Evidence also suggests that LOC tends to persist over time and is associated with increases in adiposity across development (Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice, Presnell, & Spangler, 2002; Tanofsky-Kraff et al., 2006; Tanofsky-Kraff et al., 2011).
Although children in this sample were not seeking treatment for their weight, the fairly high prevalence of LOC in this sample of girls in middle childhood highlights an important consideration. During this stage of development, children have limited ability to obtain food independently, which may constrain their intake during LOC episodes (Tanofsky-Kraff, 2008). In fact, children report that LOC episodes often occur at someone else’s house (Tanofsky-Kraff et al., 2007a). During adolescence, many youths’ access to transportation, money, and food outside of their home and school environments increases dramatically. Children who already have LOC eating when they enter adolescence may be at risk for even greater difficulties with eating and weight as highly palatable food is increasingly available. Furthermore, research suggests that negative affect is often an antecedent and a consequence of LOC (Tanofsky-Kraff et al., 2007a). The psychological distress that follows LOC could potentially exacerbate disordered eating behaviors (Tanofsky-Kraff et al., 2007a; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008) and contribute to the development of a vicious cycle between negative affect and LOC (Barker, Williams, & Galambos, 2006). Thus, early identification and intervention with these children before aberrant eating behavior becomes more entrenched and weight problems worsen appears to be essential.

5.1.6 Loss of Control and Eating in the Absence of Hunger

The major finding in this study is the association between a laboratory measure of EAH and increased likelihood of LOC. With each 100-calorie increase in EAH intake, girls were 1.72 times more likely to report a previous LOC episode. This is the first study of which we are aware to identify a relationship between these two variables and extends findings from recent research identifying an association between a questionnaire measure of EAH and LOC episodes
(Tanofsky-Kraff et al., 2008). These results suggest that although it may be normative to eat at least some calories in the absence of hunger, consuming significant amounts of food may be reflective of aberrant eating behavior.

There are several potential implications of this finding. LOC in children is associated with increased adiposity, weight gain over time, and poor response to weight loss treatment (Braet, 2006; Tanofsky-Kraff et al., 2011; Tanofsky-Kraff et al., 2009c; Tanofsky-Kraff et al., 2004; Wildes et al., 2010). Children with previous LOC episodes endorse greater eating-related psychopathology, including emotional eating, dietary restraint, and disordered attitudes and cognitions related to eating, shape, and weight (Decaluwe, Braet, & Fairburn, 2003; Levine, Ringham, Kalarchian, Wisniewski, & Marcus, 2006; Morgan et al., 2002; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2007b; Tanofsky-Kraff et al., 2004). Furthermore, LOC is associated with increases in general psychopathology; specifically, anxiety, depression, lower self-esteem, ineffectiveness, and negative affect (Morgan et al., 2002; Stice, Killen, Hayward, & Taylor, 1998; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005). Report of as few as one LOC episode is associated with these increases in BMI, disordered eating, and general psychopathology both cross-sectionally and prospectively (Ackard, Neumark-Sztainer, Story, & Perry, 2003; Tanofsky-Kraff et al., 2011).

Thus, LOC is a temporally stable marker of risk for obesity and multiple mental health concerns. However, it is difficult to reliably measure LOC when children are young because they may lack the capacity to reliably and accurately describe their own eating behavior. Moreover, LOC often does not emerge until middle childhood or adolescence. In contrast, EAH can be measured as early as age 3 years, and evidence indicates that it is a relatively stable phenomenon between ages 5-13. Thus, EAH has considerable potential to serve as an early
behavioral marker of risk for the development of aberrant eating, obesity, and psychopathology. Longitudinal research is needed to evaluate whether the relationship between EAH and LOC exists in younger children and to examine its course over time. If findings from our study are replicated in prospective studies, the EAH paradigm can potentially be used to identify very young children at-risk for the development of overweight and LOC and its sequelae.

5.2 LIMITATIONS

Several characteristics of our sample may limit generalizability of findings. Children in our study had fairly high BMI percentiles. The average BMI percentile was comparable to those found in other racially diverse samples (Vander Wal, 2004), but may have constrained analyses in which BMI was used as a predictor. The project was advertised as a study examining children’s food preferences after dinner, and participating families were offered the chance to attend a one-hour seminar on healthy eating and activities for children. It is possible that families who contacted the study had greater concern or were more invested in their child’s weight. Mothers participating in the study were fairly well-educated and a considerable number of children came from middle- to upper-SES families. Although maternal education levels and income were not associated with any of the outcome variables, findings from this study may not generalize to lower-SES families with mothers who have fewer years of formal education. Finally, the study was conducted with girls during middle childhood. Given the extensive body of literature that has documented gender- and age-related differences in eating behavior, findings may not generalize to boys or children in other age groups.
There are several other study limitations due to the cross-sectional nature of the design. First, we are not able to determine whether there are any causal relationships between variables. Furthermore, it is unclear whether the relationships between EAH and LOC persist over time or are predictive of weight gain or development of aberrant eating. We did not collect data on participants’ caloric intake preceding or following the study session. Therefore, we do not know whether children who overate in the free-access paradigm adjusted their intake in anticipation of dinner or to compensate for excess consumption during the procedure. Finally, eating behavior is likely affected by numerous factors, including social context, mood, levels of hunger and satiety, individual food preferences, to name just a few. One observation of eating behavior in a laboratory setting is likely inadequate for capturing the various contingencies that may trigger or maintain eating in the absence of hunger. Studies that are longitudinal and capture multiple measurements of eating behavior are needed to clarify the nature of the relationship between EAH, LOC, race, and BMI.

Finally, we assessed LOC using the QEWP-A, a well-validated measure of binge eating behavior selected for its ease of administration and time efficiency. The ChEDE is widely considered as the gold-standard for measuring LOC, but it was not used in the present study in an effort to minimize the time commitment required of participants. The ChEDE would likely provide a more comprehensive assessment of LOC and its sequelae and may be beneficial to use in future studies examining EAH and LOC.
5.3 SUMMARY AND FUTURE DIRECTIONS

In summary, this study is the first of which we are aware to examine EAH in Black and White girls in middle childhood. It helps advance the EAH literature by confirming the paradigm in a diverse sample and provides additional evidence that EAH is a cross-cultural phenomenon that is not limited by SES, race, age, parent education, or BMI.

Nearly all children consumed some calories in the absence of hunger, and on average, intake was considerable. This observation suggests that it is easy for children to overeat in the presence of palatable food. In a culture described as “obesigenic,” in part due to the abundance of readily accessible, energy-dense foods, this tendency to overeat has implications for the development of eating and weight problems.

This study is the first of which we are aware to identify an association between a behavioral measure of EAH and self-reported LOC. Although the finding is cross-sectional, it appears that EAH may be an antecedent as well as a correlate of LOC. Findings from this study provide preliminary evidence that EAH may be an early behavioral marker of risk for the development of aberrant eating, obesity, and psychopathology. Furthermore, we have identified EAH as a laboratory marker of risk for LOC that can be used in future studies to strengthen self-report of LOC. Laboratory studies of LOC often instruct children to binge eat or explicitly monitor children’s food intake. The EAH procedure provides a measure that may reduce some confounds in those studies.

The present study had several strengths. First, we used a racially diverse sample to examine EAH and LOC. Additionally, the study was designed to reduce several of the methodological confounds found in previous research. Children ate dinner and completed the free-access session individually, rather than in pairs or groups, in order to minimize social
desirability concerns that could influence eating behavior. Furthermore, efforts were made to limit participants’ awareness that their food intake was being monitored, and to obscure the overall purpose of the study until the procedure was completed. Additionally, we used a laboratory measure of EAH, rather than a questionnaire. The use of a laboratory design is beneficial because it provides direct observation of eating behaviors, more precise measurements of energy intake, and is fairly consistent with eating behavior outside of the laboratory (Hadigan, Kissileff, & Walsh, 1989; Tanofoşky-Kraff, Haynos, Kotler, Yanovski, & Yanovski, 2007; Walsh, Kissileff, Cassidy, & Dantzic, 1989). Children and adolescents tend to underestimate their caloric consumption (Fisher, Johnson, Lindquist, Birch, & Goran, 2000), so laboratory assessment has considerable advantages. Finally, we used a measure of LOC that was administered by a trained interviewer, rather than asking girls to complete a questionnaire on their own. This provided the opportunity for participants to ask for clarification and examples of LOC, which helped ensure that they understood the concept.

Results from the present study have potential implications for intervention and highlight several important areas for future research. The finding of a relationship between EAH and LOC in this study is noteworthy as both behaviors tend to persist over time and are associated cross-sectionally and prospectively with overweight and psychological distress. One of the most promising aspects of research in this area is that EAH behavior can be reliably measured in very young children, who may have difficulty responding accurately to self-report measures of eating behavior. The free-access procedure may be a helpful way of identifying young children at risk of developing problems with eating and weight, especially LOC. Another advantage of laboratory assessment is that it can be used to provide feedback for children regarding their eating behavior. Children with LOC often endorse a sense of “numbing” or lack of awareness
when they overeat (Tanofsky-Kraff et al., 2007a). The free-access session could be videotaped or
children could be provided with feedback regarding the number of calories they consumed as a
way of teaching them to self-monitor their eating.

Results from this study highlight other potentially promising targets for intervention.
Children with LOC fare poorly in behavioral weight loss interventions and may be at higher risk
of dropout (Braet, 2006; Levine, Ringham, Kalarchian, Wisniewski, & Marcus, 2006; Wildes et
al., 2010). Identification and intervention with children at younger ages may lead to better
prevention and treatment outcomes. In fact, an intervention study was conducted with preschool
children across the weight spectrum that employed a pre-and post-test design to measure
children’s ability to self-regulate their intake (S. L. Johnson, 2000). On two occasions, children
were administered drinks that differed in energy density and carbohydrate content, but not in
taste or appearance, prior to consuming an ad libitum lunch. Self-regulation was measured by the
degree to which children adjusted their caloric intake during lunch following a high energy
density drink. Children varied significantly in their ability to modify their intake. Following the
assessment, children participated in a 6-week intervention designed to help them recognize
internal cues of hunger and satiety. Following the intervention, both undereaters and overeaters
showed an improved ability to self-regulate their intake. Thus, children as young as preschool-
age appear to be capable of learning how to attend to internal hunger and satiety cues and adjust
their intake accordingly. The free-access procedure could be used to identify children who have
the tendency to overeat in the absence of hunger, and then they could be provided with treatment
designed to help them learn skills to regulate their eating based on internal cues and reduce
eating behavior triggered by external cues.
The role of mood in EAH and LOC behavior has not been examined in a laboratory study. Questionnaire evidence suggests that EAH can be precipitated by both external food-related cues and negative affect (Tanofsky-Kraff et al., 2008). In this study, EAH in response to emotional, rather than external food-related, cues showed a greater association with excess weight. Given that the free-access theoretically relies on food-related cues to trigger eating, the eating behavior it captures may be qualitatively different from that triggered by affective cues. There is no data of which we are aware in children that measures EAH intake in different mood states. Because LOC is associated with increases in negative affect, and questionnaire data suggest that EAH behavior tends to be triggered by negative affect, the next step in this line of research could be to examine the effects of different mood inductions on EAH behavior, particularly in children with LOC.
APPENDIX A

QUESTIONNAIRE OF EATING AND WEIGHT PATTERNS: ADOLESCENT VERSION

1. During the past 6 months, did you ever eat what most people, like your friends, would think was a really big amount of food?
   1  YES
   2  NO

Did you ever eat a really big amount of food within a short time (2 hours or less)?
   1  YES
   2  NO

If YES to either of above questions, document how much child consumed. Specifically, document type and amount of food and period of time to consume food.

2. When you ate a really big amount of food, did you ever feel that you could not stop eating? Did you feel that you could not control what or how much you were eating?
   NOTE: If participant does not endorse eating a really big amount of food, ask “Have you ever felt that you could not stop eating or that you could not control what or how much you were eating?”
   1  YES
   2  NO

(If NO, skip to question #5)

3. During the past 6 months, how often did you eat a really big amount of food with the feeling that your eating was out of control?  NOTE: If participant only endorsed question 1 or 2, ask about the frequency of that behavior.

There may have been some weeks when you did not eat this way at all. And some weeks you may have eaten like this a lot. But, in general, how often did this happen?
4. When you ate a really big amount of food and you could not control your eating, did you:
   a) Eat very fast?                      YES  NO
   b) Eat until your stomach hurt or you felt sick in your stomach?  YES  NO
   c) Eat really big amounts of food even when you were not hungry?  YES  NO
   d) Eat really big amounts of food during the day without regular meals like breakfast, lunch, and dinner?  YES  NO
   e) Eat by yourself because you did you want anyone to see how much you ate?  YES  NO
   f) Feel really bad about yourself after eating a lot of food?  YES  NO

5. During the past 6 months, how bad did you feel when you ate too much or more food than you think is best for you?
   1  Not bad at all
   2  Just a little bad
   3  Pretty bad
   4  Very bad
   5  Very, very bad
   0  I did not eat too much.

6. How bad did you feel that you could not stop eating or could not control what or how much you were eating?
   1  Not bad at all
   2  Just a little bad
   3  Pretty bad
   4  Very bad
   5  Very, very bad
   0  I did not lose control over my eating.

7. During the past 6 months, has your weight or the shape of your body mattered to how you feel about yourself? Compare this feeling to how you feel about other parts of your life - like how you get along with your parents, how you get along with your friends, and how you do at school.
   1  Weight and shape were not important at all to how I felt about myself.
   2  Weight and shape were somewhat important to how I felt about myself.
   3  Weight and shape were pretty important to how I felt about myself.
   4  Weight and shape were very important to how I felt about myself.
8. Did you ever make yourself vomit, throw up, or get sick to keep from gaining weight after eating a really big amount of food?
   1  YES
   2  NO

(IF NO: go to question #9)

How often - on the average - did you do that?
   1  Less than once a week
   2  Once a week
   3  Two or three times a week
   4  Four or five times a week
   5  More than five times a week

9. Have you ever taken medicine (pills, liquid, gum, powder) that would make you go to the bathroom in order to not gain weight after eating a really big amount of food?
   1  YES
   2  NO

(IF NO: go to question #10)

Were these laxatives (makes you have a bowel movement or B.M.) or diuretics (makes you urinate or pee)?

Circle which one(s): Laxatives Diuretics

Did you ever take more than twice the amount you were told to take on the box or bottle?
   1  YES
   2  NO

How often - on the average - did you do that?
   1  Less than once a week
   2  Once a week
   3  Two or three times a week
   4  Four or five times a week
   5  More than five times a week

10. Did you ever not eat anything all for at least 24 hours (a full day) to keep from gaining weight after eating a really big amount of food?
    1  YES
    2  NO

(IF NO: go to question #11)
How often - on the average - did you do that?
   1  Less than once a week
   2  Once a week
   3  Two or three times a week
   4  Four or five times a week
   5  More than five times a week

11. Did you ever exercise *for more than one hour* at a time *only* to keep from gaining weight after eating a really big amount of food?
   1  YES
   2  NO

(IF NO: go to question #12)

How often - on the average - did you do that?
   1  Less than once a week
   2  Once a week
   3  Two or three times a week
   4  Four or five times a week
   5  More than five times a week

12. During the past 3 months, did you ever take diet pills to keep from gaining weight after eating a *really big* amount of food?
   1  YES
   2  NO

(IF NO: stop here)

Did you ever take *more than twice* the amount you were told to take on the box or bottle?
   1  YES
   2  NO

How often - on the average - did you do that?
   1  Less than once a week
   2  Once a week
   3  Two or three times a week
   4  Four or five times a week
   5  More than five times a week

Source: (W. G. Johnson, Grieve, Adams, & Sandy, 1999)
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