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Links between Mothers' and Children's Disinhibited Eating and Children's Adiposity

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Abstract

Few studies have examined relationships between parents' and children's specific disinhibited eating behaviors. We investigated links among mothers' and children's binge/loss of control eating, eating in the absence of hunger, and children's adiposity in 305 non-treatment-seeking youth, ages 8–17 years (13.62 ± 2.65 y; 49.8% female) and their mothers. Youths' loss of control eating and eating in the absence of hunger were assessed by interview and self-report questionnaire. Children's adiposity was assessed with BMI-z and air displacement plethysmography. Maternal binge eating, eating in the absence of hunger and highest, non-pregnant BMI were self-reported. In structural equation models controlling for mothers' BMI, mothers' binge eating related to children's loss of control eating, and mothers' eating in the absence of hunger related to children's eating in the absence of hunger. Mothers' binge eating and children's eating in the absence of hunger were unrelated, as were mothers' eating in the absence of hunger and children's loss of control. Further, mothers' binge eating was indirectly related to children's adiposity through children's loss of control eating. Likewise, mothers' eating in the absence of hunger indirectly related to children's adiposity through children's eating in the absence of hunger. Mothers and children share similar, specific disinhibited eating styles.

Keywords

Disinhibited eating; Eating in the absence of hunger; Loss of control eating; Binge Eating; Obesity

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Disinhibited eating refers to a range of eating behaviors characterized by a lack of appropriate restraint over food intake, including binge eating, loss of control eating, and eating in the absence of hunger (Shomaker, Tanofsky-Kraff, & Yanovski, 2009). Several cross-sectional investigations of overall propensity for disinhibited eating have demonstrated a significant association between parents, particularly mothers, and children (Brown & Ogden, 2004; Jahnke & Warschburger, 2008; Provencher, et al., 2005). Twin studies indicate that such correspondence is likely due to the moderate heritability of disinhibited eating, influenced by shared-environmental effects (de Castro & Lilenfeld, 2005; Sung, Lee, Song, Lee, & Lee, 2009; Tholin, Rasmussen, Tynelius, & Karlsson, 2005).

Although prior data have found a relationship between parents' and children's propensity for disinhibited eating, fewer studies have investigated parent-child correspondence of specific disinhibited eating behaviors, such as binge or loss of control eating. Objective binge eating refers to an eating episode during which an individual experiences lack of control while consuming an unambiguously large amount of food (American Psychiatric Association, 2000). Among overweight adults, objective binge eating episodes are prevalent and associated with obesity and elevated psychopathology (Hudson, Hiripi, Pope, & Kessler, 2007). However, in youth, the experience of loss of control over eating, regardless of the amount consumed, often is considered a salient marker of such adverse health characteristics. Loss of control (LOC) eating has been defined as the subjective experience of lack of control over eating, regardless of whether the reported amount of food consumed is deemed objectively or subjectively large (Tanofsky-Kraff, 2008). Compared to youth who report overeating without LOC, those who report LOC (objective or subjective episodes) have greater adiposity and gain more weight and fat over time (Shomaker, Tanofsky-Kraff, Elliott, et al., 2009; Tanofsky-Kraff, Yanovski, et al., 2009; Tanofsky-Kraff, et al., 2004). Support for familial influences on children's LOC eating patterns come from data examining objective binge eating behaviors. Young children (age 5–7 years) with mothers who endorse objective binge eating have a six-fold greater odds of reporting objective binge eating behaviors themselves (Lamerz, et al., 2005). Additionally, twin studies indicate that binge eating is moderately heritable with h^2 coefficients ranging from 0.45 to 0.57 (Bulik, Sullivan, & Kendler, 2003; Javaras, et al., 2008; Mitchell, et al., 2010). To our knowledge, despite the significance in youth of all LOC eating episodes, objective as well as subjective (Tanofsky-Kraff, 2008), familial associations with children's LOC eating (i.e., both objective and subjective binge eating) have not been investigated.

Another specific disinhibited eating behavior that may have familial influences is eating in the absence of hunger (EAH), defined as eating in response to the presence of palatable foods in the absence of physical hunger (Kral & Faith, 2009). Children's EAH assessed in the laboratory (Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Faith, et al., 2006; Fisher & Birch, 2002; Fisher, et al., 2007; Francis, Ventura, Marini, & Birch, 2007; Hill, et al., 2008; Moens & Braet, 2007; Shunk & Birch, 2004) and assessed by questionnaire (Tanofsky-Kraff, Ranzenhofer, et al., 2008) has been associated with children's weight status. Existing research lends support to a potential relationship between parents' and children's EAH. Among predominantly white, non-Hispanic children age 3–6 years, mothers' disinhibition as measured by questionnaire was significantly related to young daughters', but not sons', observed EAH (Cutting, et al., 1999). Similarly, white, non-Hispanic mothers' reported propensity for disinhibited eating was linked to daughters' observed EAH at age 9, 11, and 13 years (Francis, et al., 2007). It has been estimated from a large study of Hispanic siblings that EAH is moderately heritable ($h^2 = 0.51$) (Fisher, et al., 2007). In spite of associations between parents' propensity for disinhibition and children's EAH, to our knowledge, no study has examined how parents' EAH specifically is related to children's EAH.

Familial influences are evident in children's disinhibited eating behaviors (Birch & Davison, 2001). Yet, few studies have investigated the relationships among specific aspects of parental eating behaviors, children's eating behaviors, and children's adiposity. In the current study, we sought to examine the links among mothers' objective binge eating and EAH, children's LOC eating (objective and subjective binge episodes) and EAH, and children's adiposity. Consistent with prior literature (d'Amore, et al., 2001; Tanofsky-Kraff, Ranzenhofer, et al., 2008), we expected that children's LOC eating and EAH would show significant overlap, yet would also be distinct. Youth with LOC eating behaviors report that such LOC episodes often, but not always, take place in the absence of hunger; in contrast, some LOC episodes are reportedly triggered in a state of physiological hunger (Marcus & Kalarchian, 2003; Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). Also, there are many youth who experience EAH without an accompanying subjective experience of lack of control over eating (Shomaker, Tanofsky-Kraff, & Yanovski, 2009). Therefore, because LOC and EAH may be somewhat conceptually differentiated, we hypothesized that mothers' binge eating would be more strongly related to children's LOC eating than to children's EAH, and that mothers' EAH would be more strongly tied to children's EAH, than to children's LOC. Additionally, consistent with both a social modeling and a behavioral genetics framework (de Castro & Lilenfeld, 2005; Patrick & Nicklas, 2005), we hypothesized that mothers' disinhibited eating behaviors would be indirectly related to their children's adiposity through their relations with children's disinhibited eating behaviors. Specifically, we predicted that structural equation models would indicate an indirect association between mothers' binge eating and children's adiposity through children's LOC, and an indirect association between mothers' EAH and children's adiposity through children's EAH.

Method

Participants

Participants were a convenience sample of healthy child volunteers and their mothers participating in non-treatment studies investigating eating behaviors (ClinicalTrials.gov IDs: NCT00631644, NCT00320177). Families were recruited through flyers posted on bulletin boards at the NIH as well as at local area supermarkets, libraries, and listservs in the Washington, DC greater metropolitan area. Flyers and advertisements specified that studies were investigating eating behaviors in healthy pediatric volunteers and that no treatment would be provided. Boys and girls of any race or ethnicity were eligible for participation if they were between the ages of 8 and 17 years, had a body mass index (BMI, kg/m^2) $\geq 5^{\text{th}}$ percentile for age and sex, were in good general health, and had a maternal parent or guardian agree to participate in a medical child and family history interview. Children were excluded if they had a significant medical condition, had abnormal hepatic, renal, or thyroid function, were taking medication known to affect body weight, or had a psychiatric disorder that might impede protocol compliance. Pregnant girls were not eligible for the study, nor were children who had lost >5 lb (2.3 kg) in the 3 months prior to assessment or who were currently undergoing weight-loss treatment. Children provided written assent and parents/guardians gave written consent for study participation. Children were financially compensated for their involvement. Study protocols were approved by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development Internal Review Board.

Procedures and assessments

Participants completed all assessments during an outpatient visit at the NIH Mark O. Hatfield Clinical Research Center (Bethesda, MD). Youth underwent a physical examination by an endocrinologist or a nurse practitioner, and mothers provided a detailed child and family medical history. Each child subsequently participated in an interview and completed

questionnaire measures of eating behavior. Mothers filled out questionnaires describing their own eating behavior and that of their child.

Children's adiposity—Children's anthropometric measurements included height, measured in triplicate, and fasting weight, measured using calibrated electronic instruments. BMI was calculated as weight (in kg) divided by the square of height (in m). BMI scores were converted to BMI standard deviation scores (BMI *z*) using the Centers for Disease Control and Prevention 2000 standards (Kuczmarski, et al., 2002). Youths' total mass (kg), fat mass (kg) and fat-free mass (kg) were measured using air displacement plethysmography (Bod Pod; Life Measurement Inc, Concord, CA) and used to calculate body fat percentage (%). Body composition measurements were obtained in the fasting state with minimal clothing and with shoes removed.

Children's loss of control (LOC) eating—The *Eating Disorder Examination Version 12.0 (EDE)* (Fairburn & Cooper, 1993) or *EDE Adapted for Children (ChEDE)* (8–14 years) (Bryant-Waugh, Cooper, Taylor, & Lask, 1996) is a semi-structured interview that was administered to assess the presence or absence of ≥ 1 LOC eating episode in the month prior to assessment. LOC was coded as present if the child endorsed either an objective binge eating episode (LOC while consuming an unambiguously large amount of food) or a subjective binge eating episode (LOC perceived by the interviewee as excessive, but judged to be an ambiguous amount by the interviewer). LOC eating, regardless of the episode size, was considered among youth because it is a prospective risk factor for excess weight gain and eating disorder psychopathology (Tanofsky-Kraff, et al., 2010; Tanofsky-Kraff, Yanovski, et al., 2009). Youth who either endorsed no episodes of overeating or overeating without LOC were coded as having no LOC. Interviewers were graduate students in clinical psychology and post-baccalaureate research associates who had attended 15–20 hr of EDE training. The ChEDE differs from the adult version only in that its script has been edited to make it more accessible to children ages 8–14 years and that two items that assess the critical overvaluation of shape and weight have been supplemented with a sort task. As with the EDE, the ChEDE's interview-based, interactive nature allows for questions to be explained so that they are understood by each individual, including children as young as 8 years of age. In addition, special care is taken, and examples are provided, to explain difficult concepts such as “loss of control,” or the sense of being unable to stop eating once started. The EDE and ChEDE have good internal consistency, discriminant, and prospective validity (Christie, Watkins, & Lask, 2000; Fairburn & Cooper, 1993; Rizvi, Peterson, Crow, & Agras, 2000; Williamson, et al., 1995) and have been successfully combined for data analysis in prior studies (Shomaker, et al., 2010; Tanofsky-Kraff, et al., 2007; Tanofsky-Kraff, McDuffie, et al., 2009).

Youth also completed an adapted version of the *Questionnaire on Eating and Weight Patterns-Adolescent Version (QEWP-A)* to assess the reported presence or absence of ≥ 1 LOC eating episode (either objective or subjective binge eating episode) in the six months prior to assessment (Johnson, Grieve, Adams, & Sandy, 1999; Spitzer, et al., 1993). Although the QEWP-A typically has been used to assess objective binge episodes only, it was modified to assess additional experiences of lack of control over any amount of food. The QEWP-A has good concurrent validity and test-retest reliability (Johnson, Kirk, & Reed, 2001).

Children's eating in the absence of hunger (EAH)—Children reported their perceived EAH on the *Eating in the Absence of Hunger Questionnaire for Children and Adolescents (EAH-C)* (Tanofsky-Kraff, Ranzenhofer, et al., 2008). The EAH-C is a 14-item youth self-report measure designed to assess the frequency of eating when one is not hungry among youth ages 6 to 19 years. The EAH-C is comprised of three factor-analysis derived

scales that measure the extent to which youth report eating in the absence of hunger in response to: i) external cues (4 items) (e.g., “How often do you keep eating because the food looks, tastes, or smells so good?”), ii) negative affect (6 items) (e.g., “How often do you keep eating because you are feeling sad or depressed?”) and iii) feelings of fatigue or boredom (4 items) (e.g., “How often do you start eating because you are feeling tired?”). All questions were rated on a 5-point Likert scale from 1 = never to 5 = always. For each scale, items were averaged across questions, such that higher scores reflected greater frequency of EAH. The EAH-C subscales are internally consistent, and the measure has demonstrated good convergent validity and temporal stability (Tanofsky-Kraff, Ranzenhofer, et al., 2008).

Parents’ completed the *Eating in the Absence of Hunger Questionnaire for Parents (EAH-P)*, a parallel version of the EAH-C, to assess their perceptions of their children’s EAH (Shomaker, Tanofsky-Kraff, Elliott, et al., 2009). As with the EAH-C, the EAH-P includes 14 items that load onto three factor-analytically derived scales measuring the frequency of eating when one is not hungry in response to: i) external cues, ii) negative affect, and iii) feelings of fatigue or boredom. In a subset of the current sample ($n = 243$), these scales illustrated acceptable internal reliability ($\alpha s > 0.81$ – 0.94) and temporal stability ($r s = 0.24$ – 0.69) across an average of 21 weeks.

Youths’ EAH-C scores and mothers’ reports of their children’s EAH on the EAH-P were moderately related across all sub-scales (Mean $r = 0.28$). Accordingly, in order to make use of both sources, youths’ and mothers’ reports were averaged to create internally reliable, cross-informant composites for children’s EAH in response to external cues ($\alpha = 0.81$), negative affect ($\alpha = 0.86$), and fatigue or boredom ($\alpha = 0.83$).

Mothers’ BMI—Mothers were queried about their height and highest ever non-pregnant weight by an endocrinologist or nurse practitioner during a family medical history interview. Mothers’ reports on these variables were used to calculate mothers’ BMI. Although some underreporting of weight has been shown to occur (Gorber, Tremblay, Moher, & Gorber, 2007), self-reported height and weight have been found to be highly correlated with objective measurements of BMI in adults (McAdams, Van Dam, & Hu, 2007).

Mothers’ binge eating—The reported lifetime presence or absence of mothers’ binge eating was assessed through their responses to a questionnaire designed to assess parental dieting history. In mothers, we assessed presence of objective binge eating rather than all LOC episodes (i.e., objective as well as subjective) because it is the former that has been most consistently related to overweight and eating disorder psychopathology among adults (Hudson, et al., 2007). Binge eating presence ever was assessed through the endorsement of the following item: “Have you often had times when you ate within any two hour period what most people would regard as an unusually large amount of food and had the feeling your eating was out of control?” For descriptive purposes, reported frequency criteria for binge eating disorder was assessed through the item: “Did this out-of-control eating ever occur at least two days a week for at least six months?” (American Psychiatric Association, 2000).

Mothers’ eating in the absence of hunger (EAH)—Mothers’ EAH was assessed via their self-report on the *Eating in the Absence of Hunger Questionnaire for Adults (EAH-A)*. As with the EAH-C and EAH-P, the EAH-A is comprised of 14 items that load onto three factor-analytically derived scales measuring the overall frequency of eating when one is not hungry in response to: i) external cues, ii) negative affect, and iii) feelings of fatigue or boredom. In a subset of the current sample ($n = 243$), the EAH-A displayed good internal validity ($\alpha s = 0.80$ – 0.95) and temporal stability ($r s = 0.35$ – 0.64 , $p s < 0.001$) across an average of 21 weeks.

Statistical analyses

Data were examined to determine that all variables approximated a normal distribution. Scores had acceptable levels of skew and kurtosis, and there were no influential outliers. Correlations were examined to describe the univariate inter-relationships among key variables. All dichotomous variables were dummy-coded (e.g., LOC absence = 0, LOC presence = 1). Structural equation modeling (SEM) based on full information maximum likelihood estimation was conducted using AMOS 16.0, a SPSS 16.0 software (SPSS Inc, 2007), to evaluate the proposed factor structures and to model the hypothesized links among children's LOC eating (presence versus absence of objective and/or subjective binge eating), mothers' objective binge eating (presence versus absence), children's EAH, mothers' EAH and children's adiposity. Compared to other statistical approaches, SEM has a number of advantages, including the ability to model latent variables measured by multiple indicators, to account for measurement error, and to minimize Type I error by reducing the number of analyses run (Kline, 2005; Nelson, Aylward, & Steele, 2008). Following recommended guidelines for SEM (Cole & Maxwell, 2003), a confirmatory factor analysis (CFA) first was conducted to ensure that the proposed factor structures were acceptable for the measurement of each latent construct. Since model chi-square (χ^2_M) is highly sensitive to sample size, two additional fit indices were examined to interpret the acceptability of the measurement model for describing the data: i) comparative fit index (CFI), and ii) root mean square error of approximation (RMSEA). For CFI, a value equal or greater than 0.90 was considered indicative of a reasonably good fitting model (Kline, 2005). A cut-off criterion for RMSEA of equal or less than 0.08 was considered a reasonable error of approximation (Browne & Cudeck, 1993). We next examined structural models of the hypothesized relationships among mothers' disinhibited eating, children's disinhibited eating, and children's adiposity. We examined a separate model for binge/LOC eating behavior and EAH, and then examined a model that incorporated both binge/LOC and EAH behaviors simultaneously. Mothers' BMI was included in all models in order to test the associations of mothers' disinhibited eating with children's eating and adiposity independent of familial similarities in weight. Indirect effects were tested with a product of coefficients approach to testing for intervening variables, which has better statistical power and less likelihood of Type I errors than traditional measures (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). The product of two coefficients was derived: α = the effect of the independent variable (i.e., mothers' binge eating or EAH) on the intervening variable (children's LOC eating or EAH), and β = the effect of the intervening variable (children's LOC eating or EAH) on the dependent variable (children's adiposity). The estimate of the intervening variable effect, $\alpha\beta$, was divided by its SE (Sobel, 1982) and compared to a standard distribution.

Results

Participants were 305 children ranging in age from 8 to 17 years ($M \pm SD$, $13.62 \pm 2.65y$). Half (49.8%) were female and 54.1% were white, non-Hispanic (34.4% African American; 11.5% other). Children represented a spectrum of BMI z scores, ranging from -2.18 to $+3.20$ and averaged $+0.81 \pm 1.10$. In this sample, 16.8% of children were overweight (defined as a BMI $\geq 85^{th}$ percentile to $\leq 94^{th}$ percentile), and 26.0% were obese (defined as a BMI $\geq 95^{th}$ percentile) (Ogden & Flegal, 2010). Mothers' mean BMI was 30.00 ± 7.64 kg/m² (range = 18.42 to 59.19 kg/m²). Among mothers, 26.4% were overweight (defined as a BMI of 25.0–29.9), and 42.4% were obese (defined as a BMI of ≥ 30.0).

Bivariate correlations among mothers' disinhibited eating, children's disinhibited eating, and children's adiposity

As displayed in Table 1, 28.5% of children endorsed ≥ 1 recent episode of LOC on the EDE or ChEDE, and 16.5% endorsed LOC presence on the QEWP-A. Consistent with the non-

treatment seeking nature of this sample (Shomaker, Tanofsky-Kraff, Elliott, et al., 2009), on the EDE or ChEDE, most youth (42.8%, $n = 36$) reporting LOC endorsed only 1 episode in the past month; 22.6% ($n = 19$) endorsed 2 episodes; 10.7% ($n = 10$) reported 3 episodes; and the remaining 23.9% reported between 4 and 28 episodes. A history of binge eating ever was endorsed by 29.3% of mothers. Frequent binge eating (i.e., ≥ 2 episodes per week for ≥ 6 months) was reported by 7.8% of mothers.

Correlations indicated that measures of children's LOC eating presence were significantly inter-related ($p < 0.001$), as were composite scales assessing children's EAH ($ps < 0.001$), and scales of mothers' reported EAH ($ps < 0.001$). There was significant overlap between specific types of disinhibited eating within individuals. Mothers' binge eating presence and mothers' EAH were significantly related ($ps < 0.001$), as were children's LOC eating presence and children's EAH ($ps < 0.01$). Mothers' binge eating presence was related to children's LOC eating presence and children's EAH ($ps < 0.01$), and mothers' EAH was related to children's EAH ($ps < 0.001$).

Confirmatory factor analysis (CFA)

A CFA was conducted to examine the acceptability of the proposed measurements of latent constructs. The measurement model fit the data adequately, $\chi^2 (35, N = 305) = 68.93$, $p < 0.001$, RMSEA = 0.06, CFI = 0.97, and all manifest variables significantly loaded on their hypothesized latent constructs ($ps < 0.001$; Table 2). Children's LOC eating presence was a latent variable composed of two manifest constructs generated by the EDE and the QEWP-A. Children's EAH was a latent construct comprised of the three indicators of negative affect, external cues and fatigue/boredom mother-child composite scales. Children's adiposity was a latent variable represented by BMI-z, total mass (kg), and fat mass (%). All assessments of adiposity were associated with one another ($ps < 0.001$). Mothers' EAH was a latent construct composed of the negative affect, external cues and fatigue/boredom scales of the EAH-A. Mothers' binge eating presence and mothers' BMI were modeled as single manifest variables.

Structural equation modeling (SEM) examining relationships among mothers' disinhibited eating, children's disinhibited eating, and children's adiposity

The model examining binge/LOC eating behavior was an acceptable fit to the data, $\chi^2 (df = 10, N = 305) = 21.65$, $p = 0.02$, RMSEA = 0.06, CFI = 0.98 (Figure 1A). There was a significant relationship between mothers' binge eating and children's LOC eating ($path = 0.35$, $p < 0.001$), even after adjusting for mothers' BMI. Additionally, there was a significant association between children's LOC eating and children's adiposity ($path = 0.31$, $p < 0.001$). The direct association between mothers' binge eating and children's adiposity was not significant ($p = 0.25$). However, there was a significant indirect relationship of mothers' binge eating with children's adiposity through children's LOC eating ($z = 2.34$, $p = 0.02$).

The model examining EAH was also an acceptable fit to the data, $\chi^2 (df = 27, N = 305) = 41.30$, $p = 0.04$, RMSEA = 0.04, CFI = 0.99 (Figure 1B). Mothers' EAH and children's EAH were significantly related ($path = 0.48$, $p < 0.001$), even after accounting for maternal BMI. The link between children's EAH and children's adiposity also was significant ($path = 0.29$, $p < 0.001$). There was no significant direct link between mothers' EAH and children's adiposity ($p = 0.15$); yet, mothers' EAH had a significant indirect association with children's adiposity through children's EAH ($z = 3.22$, $p = 0.001$).

A combined model examining binge/LOC and EAH together was an acceptable fit to the data, $\chi^2 (df = 49, N = 305) = 84.93$, $p = 0.001$, RMSEA = 0.05, CFI = 0.97 (Figure 2). Mothers' binge eating and mothers' EAH were significantly correlated ($r = 0.40$, $p < 0.001$),

as were children's LOC eating and children's EAH ($r = 0.47, p < 0.001$). The direct link between mothers' binge eating and children's LOC eating remained significant ($path = 0.30, p < 0.001$), as did the link between mothers' EAH and children's EAH ($path = 0.46, p < 0.001$). Mothers' binge eating was not related to children's EAH ($p = 0.44$) nor was mothers' EAH related to children's LOC ($p = 0.52$). In this combined model, only children's EAH remained significantly, directly related to children's adiposity ($path = 0.23, p < 0.05$). Likewise, the only indirect effect was that mothers' EAH remained significantly, indirectly associated with children's adiposity through children's EAH ($z = 2.15, p = 0.03$).

Post-hoc exploratory multiple-group SEM analyses

Multiple-group SEM modeling was conducted to explore whether sex (male versus female), race/ethnicity (white, non-Hispanic versus other), or age (pre-adolescent <13 years versus adolescents ≥ 13 years) moderated the relationships in the combined model. The fit of a model in which the associations among mothers' binge eating, mothers' EAH, children's LOC eating, children's EAH, and children's adiposity were constrained to be equal for each group was compared with a model in which the associations were unconstrained. For sex and race/ethnicity, the comparative fits of the constrained and unconstrained models were not significantly different ($ps = 0.88$ and 0.52 , sex and race/ethnicity, respectively), suggesting that the relationships were not different based upon sex or race/ethnicity. In the multiple-group model examining age, the unconstrained model was a better fit than a constrained model, $\chi^2_{\text{difference}} (df_{\text{difference}} = 18, N = 305) = 39.87, p = 0.002$. The relationships between mother's binge eating and children's EAH, between children's EAH and children's LOC eating, and between children's EAH and children's adiposity were relatively stronger among preadolescents compared to adolescents ($ps < 0.001$). The paths between mothers' EAH and mothers' EAH, mothers' EAH and children's adiposity, and mothers' BMI and children's adiposity were relatively stronger among adolescents compared to pre-adolescents ($ps < 0.05$).

Discussion

Findings from the current study contribute to our understanding of familial links between mothers' and children's disinhibited eating. Building upon prior data illustrating a relationship between mothers' and children's general propensity for disinhibition (Brown & Ogden, 2004; Jahnke & Warschburger, 2008; Provencher, et al., 2005), we investigated two specific types of children's disinhibited eating behaviors: loss of control over eating (including both objective as well as subjective binge eating episodes) and eating in the absence of hunger. Consistent with prior findings utilizing portions of the current data set (Tanofsky-Kraff, Ranzenhofer, et al., 2008), children's loss of control eating and children's eating in the absence of hunger were significantly, moderately related. Yet, in spite of this overlap, the associations between mothers' and children's disinhibited eating behaviors showed discriminant, construct-specific relationships: in the structural equation model, mothers' binge eating was related only to children's loss of control eating, and mothers' eating in the absence of hunger was related only to children's eating in the absence of hunger (Shomaker, Tanofsky-Kraff, & Yanovski, 2009; Tanofsky-Kraff, Ranzenhofer, et al., 2008). Although there is a very limited body of research investigating the overlap and differentiation among specific types of pediatric disinhibited eating behaviors, prior data suggest that youth with loss of control (LOC) frequently, but not always, report lack of control while eating in the absence of hunger (EAH), and conversely, that EAH is sometimes, but far from always, accompanied by LOC (Marcus & Kalarchian, 2003; Shomaker, Tanofsky-Kraff, & Yanovski, 2009; Tanofsky-Kraff, Marcus, et al., 2008). Taken together with findings from the present study, these data begin to formulate the

hypothesis that pediatric LOC and EAH may be overlapping, but also represent distinct endophenotypes for obesity risk.

The observed relationship between mothers' objective binge eating to their children's LOC eating (objective and subjective episodes) is consistent with prior literature demonstrating an association between maternal binge eating and young children's objective binge eating episodes (Lamerz, et al., 2005). Furthermore, before accounting for EAH, mothers' binge eating was indirectly associated with children's adiposity through its direct relationship with children's LOC eating. Although these data are cross-sectional and correlational in nature, a number of potential theoretical mechanisms might explain these patterns. From a behavioral genetics framework, the relationships between mothers' binge eating and children's LOC eating partially may be due to the moderate heritability of binge eating behaviors within families (Bulik, et al., 2003; Javaras, et al., 2008; Mitchell, et al., 2010). Indeed, recent data have begun to identify particular genes (e.g., *FTO*) that are associated with LOC eating in youth (Tanofsky-Kraff, Han, et al., 2009). The direct associations of mothers' binge eating with children's LOC and adiposity remained after accounting for maternal BMI. This bolsters the likelihood of there being other influential mechanisms on children's eating and weight, beyond that of familial similarities in weight. Social modeling, for instance, highlights that children learn about their own eating preferences and behaviors not only through self-discovery, but also by watching others (Patrick & Nicklas, 2005). The family may be one major source from which children and adolescents learn various styles of eating. Prior literature shows that 43% of children and adolescents age 9–14 years eat dinner at home with their family (Gillman, et al., 2000), and youth consume 68% of meals and 78% of snacks in the home (Lin, Guthrie, & Frazao, 1999). Thus, it is possible that children's disinhibited eating episodes, behaviors that have been linked to obesity (Shomaker, Tanofsky-Kraff, & Yanovski, 2009), may be both genetically and environmentally influenced.

Children's EAH, measured by a composite of mother and child reports, was significantly associated with mothers' perceived EAH in themselves. These findings are congruent with prior data illustrating an association between mothers' general propensity for disinhibited eating and children's observed EAH (Cutting, et al., 1999; Fisher & Birch, 2002). The significant correspondence between mothers' EAH and their children's EAH is also in line with existing studies supporting a role for familial influences on EAH (Faith, et al., 2006) and the moderate heritability of EAH (Fisher & Birch, 2002). Further, novel results from the current data point to an indirect relationship of mothers' EAH with children's adiposity through children's EAH. Although speculative, it is plausible that mothers' EAH indirectly influences children's adiposity through its effect on children's eating behavior.

Strengths of this study include the examination of specific disinhibited eating behaviors (i.e., LOC eating and EAH) in a relatively large and diverse sample of boys and girls varying in age (8–17 years) and race. The use of statistical equation modeling (SEM) in the current project had a number of advantages such as accounting for missing data, measurement error, and the ability to model multiple assessments from different sources (i.e., children as well as their mothers) and methods (i.e., interviews, questionnaires, and objective physical measurements) (Kline, 2005; Nelson, et al., 2008). Furthermore, post-hoc exploratory SEM analyses examining potential moderators of the modeled relationships suggested similarities across sex and various races/ethnicities, and alternatively, pointed to possible age differences that warrant further testing. Perhaps most notably, the link between mothers' BMI and children's adiposity was stronger in adolescents than in preadolescents, which is in keeping with prior research showing that the genetic component to adiposity becomes more strongly related in family members over time (Goode, Cherny, Christian, Jarvik, & de Andrade, 2007).

Limitations of the current study include the cross-sectional nature of the data, which hindered the ability to determine directionality in the relationships among the behaviors investigated. Another limitation was the reliance on questionnaire measures of EAH that, although related to adiposity, may be less valid than observed measures of this behavior. Moreover, because the focus of the larger studies from which the current data were drawn was on children's rather than their parent's eating, maternal measurements were limited. For example, although children's LOC eating was assessed by both interview and questionnaire, binge eating in mothers was measured by a single questionnaire item. Similarly, although objective measurements were obtained of children's adiposity, we relied on self-reported maternal BMI. Self-reported BMI and objective BMI have been found to be highly related in adult samples (Gorber, et al., 2007; McAdams, et al., 2007); yet, underreporting tends to occur, especially among overweight women (Gorber, et al., 2007). Although a continuous (rather than categorical) measurement of BMI based upon maternal highest ever non-pregnant weight may have minimized the influence of this bias (Stewart, Jackson, Ford, & Beaglehole, 1987), it is still possible that reliance on self-reported maternal weight may have produced underestimates of its effects on maternal and child disinhibited eating and child adiposity. Another drawback is the inclusion of only mothers. Although fathers have historically been understudied, some research suggests that fathers influence the development of disordered eating patterns in their children (Rodgers & Chabrol, 2009); future research efforts that examine paternal influences on children's LOC and EAH are warranted. Moreover, the influence of non-familial, environmental effects (e.g., peers) on children's disinhibited eating behaviors should be considered, as data suggest that non-shared environmental factors contribute to the development of eating patterns (Reichborn-Kjennerud, Bulik, Tambs, & Harris, 2004).

In conclusion, the current study findings support the likelihood that children and mothers exhibit similar specific disinhibited eating behaviors, which likely reflect both genetic and environmental influences (de Castro & Lilenfeld, 2005; Sung, et al., 2009; Tholin, et al., 2005). Of particular interest was the fact that specific aspects of disinhibited eating – LOC eating and EAH – showed overlap within children, yet appeared to be uniquely related to qualities of mothers' reported binge eating and EAH, respectively. Further research is required to elucidate the pathways through which parents and children develop similar eating behaviors, which may in turn influence children's adiposity.

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Figure 1.

Figure 1A. Model of links among mothers' objective binge eating, children's loss of control (LOC) eating (objective and subjective binge episodes), and children's adiposity, adjusted for mothers' body mass index (BMI), χ^2 (df = 10, N = 305) = 21.65, $p = 0.02$, RMSEA = 0.06, CFI = 0.98. ** $p < 0.01$, *** $p < 0.001$.

Figure 1B. Model of links among mothers' eating in the absence of hunger (EAH), children's EAH, and children's adiposity, adjusted for mothers' body mass index (BMI), χ^2 (df = 27, N = 305) = 41.30, $p = 0.04$, RMSEA = 0.04, CFI = 0.99. *** $p < 0.001$.

Figure 2.

Model of links among mother's disinhibited eating (binge eating and eating in the absence of hunger [EAH]), children's disinhibited eating (LOC eating and EAH), and children's adiposity, adjusted for mothers' body mass index (BMI), χ^2 (df = 49, N = 305) = 84.93, $p = 0.001$, RMSEA = 0.05, CFI = 0.97. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 1
Descriptive information and interrelationships among mothers' and children's disinhibited eating variables

	1.	2.	3.	4.	5	6.	7.	8.	M±SD	N
1. Child LOC-EDE ^a	--								28.5% (Present)	295
2. Child LOC-QEWP-A ^b	0.44***	--							16.5% (Present)	200
3. Mother Binge ^c	0.22***	0.30***	--						29.3% (Present)	266
4. Child-EAH-C/P-Neg Aff ^d	0.37***	0.26***	0.21**	--					1.47±0.48	263
5. Child-EAH-C/P-External ^d	0.17**	0.30***	0.18**	0.41***	--				2.59±0.65	263
6. Child-EAH-C/P-Fatigue ^d	0.28***	0.21**	0.18**	0.65***	0.58***	--			1.83±0.65	263
7. Mother-EAH-A-Neg Aff ^e	0.12	0.16*	0.34***	0.34***	0.22***	0.32***	--		1.69±0.82	252
8. Mother-EAH-A-External ^e	0.02	0.13	0.32***	0.22***	0.46***	0.30***	0.47***	--	2.55±0.80	252
9. Mother-EAH-A-Fatigue ^e	0.12	0.09	0.33***	0.31***	0.30***	0.49***	0.70***	0.56***	1.81±0.82	252

^a Loss of control (LOC) presence assessed with the Eating Disorder Examination (Fairburn & Cooper, 1993) or version adapted for children (Bryant-Waugh, et al., 1996), dummy-coded "0" = no LOC in past month, "1" ≥ 1 LOC episode in past month;

^b LOC assessed with the Questionnaire on Eating and Weight Patterns-Adolescent Version (Johnson, et al., 1999), dummy-coded "0" = no LOC in past six months, "1" ≥ 1 LOC episode in past six months;

^c Dummy-coded "0" = no reported history of binge eating, "1" = positive history of binge eating.

^d Child eating in the absence of hunger assessed using a composite of child reports on the Eating in the Absence of Hunger Questionnaire for Children and Adolescents (Tanofsky-Kraff, Ranzenhofer, et al., 2008) and mother reports on the Eating in the Absence of Hunger Questionnaire for Parents;

^e Mother eating in the absence of hunger assessed using the Eating in the Absence of Hunger Questionnaire for Adults.

Table 2

Standardized confirmatory factor loadings on latent variables

Latent Variable	Manifest Variable	Standardized Loading
Child LOC	LOC-EDE ^a	0.75***
	LOC-QEWP-A ^b	0.56***
Child EAH	EAH-C/P Composite Negative Affect ^c	0.74***
	EAH-C/P Composite External ^c	0.64***
	EAH-C/P Composite Fatigue ^c	0.89***
Mother EAH	EAH-A Negative Affect ^d	0.77***
	EAH-A External ^d	0.63***
	EAH-A Fatigue ^d	0.92***
Child Adiposity	BMI z ^e	0.96***
	Total Mass (kg) ^f	0.74***
	Fat Mass (%) ^f	0.79***

^a Child loss of control (LOC) presence assessed with the Eating Disorder Examination (Fairburn & Cooper, 1993) or version adapted for children (Bryant-Waugh, et al., 1996), dummy-coded “0” = no LOC in past month, “1” ≥ 1 LOC episode in past month;

^b Child LOC presence assessed with the Questionnaire on Eating and Weight Patterns-Adolescent Version (Johnson, et al., 1999), dummy-coded “0” = no LOC in past six months, “1” ≥ 1 LOC episode in past six months;

^c Child eating in the absence of hunger assessed with a composite of child reports on the Eating in the Absence of Hunger Questionnaire for Children and Adolescents (Tanofsky-Kraff, Ranzenhofer, et al., 2008) and mother reports on the Eating in the Absence of Hunger Questionnaire for Parents;

^d Mother eating in the absence of hunger assessed with the Eating in the Absence of Hunger Questionnaire for Adults;

^e BMI standard deviation scores based upon the Centers for Disease Control and Prevention 2000 growth charts (Kuczmarski, et al., 2002);

^f Total mass (kg) and fat mass (%) assessed using air displacement plethysmography (Bod Pod; Life Measurement Inc, Concord, CA).