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Research report

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, aged 8–17 years (13.62 ± 2.65 years; 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.

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Introduction

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

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(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 et al., 2004; Tanofsky-Kraff, Yanovski, et al., 2009). Support for familial influences on children's LOC eating patterns comes from data examining objective binge eating behaviors. Young children (aged 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 the best of our knowledge, despite the significance in youth of all LOC eating episodes, objective and 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 aged 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 the best of 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$5th 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)