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Latent Profile Analysis to Determine the Typology of Disinhibited Eating Behaviors in Children and Adolescents

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Abstract

Objective—We used latent profile analysis (LPA) to classify children and adolescents into subtypes based on the overlap of disinhibited eating behaviors—eating in the absence of hunger, emotional eating, and subjective and objective binge eating.

Method—Participants were 411 youth (8–18y) from the community who reported on their disinhibited eating patterns. A subset (n=223) ate *ad libitum* from two test meals.

Results—LPA produced five subtypes that were most prominently distinguished by objective binge eating (OBE; n=53), subjective binge eating (SBE; n=59), emotional eating (EE; n=62), a mix of emotional eating and eating in the absence of hunger (EE-EAH; n=172), and no disinhibited eating (No-DE; n=64). Accounting for age, sex, race, BMI-z, the four disinhibited eating groups had more problem behaviors than no disinhibited eating ($p=.001$). OBE and SBE subtypes had greater BMI-z, percent fat mass, disordered eating attitudes, and trait anxiety than EE, EAH-EE, and No-DE subtypes ($p<.01$). However, the OBE subtype reported the highest eating concern ($p<.001$) and the OBE, SBE, and EE subtypes reported higher depressive symptoms than EE-EAH and No-DE subtypes. Across both test meals, OBE and SBE consumed less percent protein and higher percent carbohydrate than the other subtypes ($p<.02$), adjusting for age, sex, race, height, lean mass, percent fat mass, and total intake. EE also consumed greater percent carbohydrate and lower percent fat compared than EE-EAH and No-DE ($p<.03$). The SBE subtype consumed the least total calories ($p=.01$).

Discussion—We conclude that behavioral subtypes of disinhibited eating may be distinguished by psychological characteristics and objective eating behavior. Prospective data are required to determine whether subtypes predict the onset of eating disorders and obesity.

Keywords

loss of control eating; emotional eating; eating in the absence of hunger; obesity; eating disorders

Pediatric obesity (Ogden, Carroll, Kit, & Flegal, 2012) and disordered eating (Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011) are serious public health problems associated with psychiatric and medical comorbidity and reduced quality of life. Disinhibited eating—defined as bouts of overeating prompted by feelings of letting go or a lack of self-regulation—may represent an important set of modifiable behaviors that elevate risk for eating disorders and obesity in youth (Shomaker, Tanofsky-Kraff, & Yanovski, 2010). Research has revealed the presence of several types of disinhibited eating behaviors among youth, including eating in the absence of hunger, emotional eating, and loss of control eating. Laboratory studies have demonstrated that these behaviors can be observable in controlled settings among children and adolescents (Birch, Fisher, & Davison, 2003; Fisher & Birch, 2002; Fisher et al., 2007; Hilbert, Tuschen-Caffer, & Czaja, 2010; Shomaker, Tanofsky-Kraff, Zocca, et al., 2010; Tanofsky-Kraff, McDuffie, et al., 2009; Vannucci et al., 2011). Despite the likely centrality of aberrant eating behavior to the development of obesity and eating disorders, there has been little focus on the characterization of disinhibited eating behaviors and their associated clinical correlates in youth.

Eating in the absence of hunger refers to eating in response to the presence of palatable foods in the absence of physiological hunger (Kral & Faith, 2007), which is exhibited to at least some degree by approximately 60% of youth (Moens & Braet, 2007). Eating in the absence of hunger is associated with overweight status (Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Fisher & Birch, 2002; Fisher, et al., 2007; Hill et al., 2008; Moens & Braet, 2007; Shomaker, Tanofsky-Kraff, Zocca, et al., 2010; Tanofsky-Kraff et al., 2008) and increases with age during early and middle childhood (Birch, et al., 2003; Fisher, et al., 2007). The etiology of eating in the absence of hunger has often been conceptualized in terms of Schachter's (1968) original externality theory of obesity, which suggested that overeating results from a poor responsiveness to physiological, internal cues for hunger and fullness and high responsiveness to environmental, external cues such as smell, taste, and sight. Contemporary iterations of this theory emphasize poor satiety responsiveness—defined as the extent to which an individual's eating behavior tends to correspond to internal, physiological satiety signals—as an appetitive trait occurring on a continuum that promotes susceptibility for eating in the absence of hunger and obesity in today's "obesogenic" environment (Wardle, Guthrie, Sanderson, & Rapoport, 2001).

Emotional eating refers to consuming food in an attempt to cope with transient or enduring negative emotions (Faith, Allison, & Geliebter, 1997) and has been reported by approximately 50% of non-treatment-seeking youth (Shapiro et al., 2007; Tanofsky-Kraff, Theim, et al., 2007). Some (Braet et al., 2008; Braet & van Strien, 1997; Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009), but not all (Caccialanza et al., 2004; Tanofsky-Kraff, Theim, et al., 2007; van Strien & Bazelier, 2007), studies have found a relationship between emotional eating and body weight in youth. Pediatric emotional eating has been linked to excess overall energy intake and consumption of high-fat foods (Braet & van Strien, 1997; Nguyen-Rodriguez, Chou, Unger, & Spruijt-Metz, 2008; Rollins et al., 2011; Vannucci, et al., 2011; Wardle et al., 1992), which may contribute to excessive weight gain. A positive association has been found between emotional eating and disordered eating attitudes (Braet, et al., 2008). Psychosomatic theory proposes that individuals who regularly engage in emotional eating have interoceptive awareness deficits, which make it difficult to distinguish somatic sensations related to the experience of emotions from those associated

with appetite (Bruch, 1973). Similar to the notion of satiety responsiveness, this trait is also thought to occur on a continuum. Further expanding upon psychosomatic theory, recent affective theories of emotional eating suggest underscore that overeating occurs during times when food is sought out to alleviate, escape, or provide comfort from negative affective states (Dubé, LeBel, & Lu, 2005; Heatherton & Baumeister, 1991).

Loss of control eating is characterized by the experience of being unable to control what or how much one is eating regardless of the amount of food consumed (Tanofsky-Kraff, Yanovski, & Yanovski, 2011). Categorical ratings of loss of control eating are warranted in non-treatment seeking samples because the majority of such children report only one episode in the past month, resulting in a limited range of variability. Loss of control eating, by definition, encompasses both objective binge eating—consumption of an unambiguously large amount of food while experiencing loss of control—and subjective binge eating—the experience of loss of control while consuming an ambiguously large amount of food. While the DSM-IV specifies that only objective binge eating episodes constitute a clinically significant threshold for binge eating (APA, 2000), the validity of the binge size criterion has been the subject of much debate in light of mixed data in adult samples (for review, see Wolfe, Baker, Smith, & Kelly-Weeder, 2009). Determining the presence or absence of loss of control eating may be a preferable construct for use in pediatric samples due to the difficulties in determining what constitutes a large amount of food in growing children (Tanofsky-Kraff, Yanovski, et al., 2011). There are also data to suggest that there are few statistically significant differences between psychological correlates of subjectively and objectively large binge episodes in non-treatment seeking youth (Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff et al., 2004). However, further exploration of these distinctions is warranted samples in light of the mixed results in adult samples.

The prevalence of loss of control eating ranges from 4% to 45%, with higher estimates among overweight youth and adolescents (Tanofsky-Kraff, 2008). Cross-sectional (Morgan et al., 2002; Tanofsky-Kraff, et al., 2005; Tanofsky-Kraff, et al., 2004) and prospective studies (Field et al., 2003; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Tanofsky-Kraff et al., 2006; Tanofsky-Kraff, Yanovski, et al., 2009) have demonstrated a relationship between pediatric loss of control eating and excess body weight and fat. Youth with the presence of loss of control eating report greater eating disordered psychopathology and emotional distress than youth without loss of control eating (Goossens, Braet, & Decaluwe, 2007; Hartmann, Czaja, Rief, & Hilbert, 2010; Tanofsky-Kraff, et al., 2005; Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff, et al., 2004). Among children at-risk for obesity, the presence of loss of control eating also prospectively predicted the emergence of full- and partial-syndrome binge eating disorder and the worsening of disordered eating attitudes and mood symptoms (Tanofsky-Kraff et al., 2011). There is empirical support for several theories of loss of control eating in pediatric samples, including the interpersonal (Elliott et al., 2010), cognitive behavioral (Allen, Byrne, & McLean, 2012; Decaluwe & Braet, 2005), and dual pathway (Goossens, Braet, & Bosmans, 2010; Stice, 2001) models.

Overall, findings suggest that eating in the absence of hunger, emotional eating, and loss of control eating may each play a role in promoting excessive weight gain and disordered eating pathology in youth. As described above, these three disinhibited eating behaviors are conceptually distinct and appear to have differing clinical implications for eating- and weight-related problems. Nonetheless, these three disinhibited eating behaviors are theoretically similar in that they involve a failure to rely solely or primarily on physiological hunger and satiety cues to govern food intake (Shomaker, Tanofsky-Kraff, & Yanovski, 2010). Indeed, data suggest that eating in the absence of hunger, emotional eating, and loss of control eating co-occur in youth (Goossens, et al., 2007; Moens & Braet, 2007;

Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff, et al., 2008; Tanofsky-Kraff, Theim, et al., 2007; Zocca et al., 2011). Taken together, eating in the absence of hunger, emotional eating, and loss of control eating appear to be distinct, yet commonly co-occurring behaviors in children and adolescents. To date, research has focused on characterizing each of these behaviors separately rather than investigating them concurrently. Given the rise in pediatric obesity and eating disorders, there is a need for additional characterization of the co-occurrence of disinhibited eating behaviors in youth in relation to clinical correlates.

We therefore aimed to determine the typology of disinhibited eating behaviors in a community sample of children and adolescents based on the co-occurrence of eating in the absence of hunger, emotional eating, and loss of control eating. We hypothesized that latent profile analysis would yield at least five distinct subtypes based on the theories underlying each disinhibited eating behavior: 1) an asymptomatic subtype defined by the relative absence of all three disinhibited eating behaviors; 2) a subtype with the presence of frequent eating in the absence of hunger only; 3) a subtype characterized by the presence of primarily emotional eating given its conceptual distinctiveness from eating in the absence of hunger; 4) a subtype with the co-occurrence of emotional eating and eating in the absence of hunger since it is feasible that some youth may have both etiological mechanisms of disinhibited eating present; and 5) a subtype distinguished by the co-occurrence of all three disinhibited eating behaviors since many theories of loss of control eating have components of affective and externality theories implicit within them and therefore these youth may have multiple etiological mechanisms that increase their vulnerability for all types of disinhibited eating.

We also sought to investigate the validity of disinhibited eating subtypes with measures of adiposity, psychological functioning, and objective measures of eating behavior. Given data demonstrating adverse psychological correlates of loss of control eating (Field, et al., 2003; Goossens, et al., 2007; Morgan, et al., 2002; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, et al., 2006; Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff, Shomaker, et al., 2011; Tanofsky-Kraff, Yanovski, et al., 2009; Tanofsky-Kraff, et al., 2004), we expected that the “high overlap” subtype would exhibit the most adverse eating- and weight-related characteristics. We also hypothesized that subtypes distinguished by emotional eating, but with the relative absence of loss of control eating, would report greater psychopathology as compared to subtypes with eating in the absence of hunger. However, we expected these subtypes would be similar with regard to adiposity and laboratory eating behavior based on prior data suggesting that both disinhibited eating behaviors are associated with elevated adiposity and aberrant eating behavior in the laboratory.

Methods

Participants

Participants were children and adolescents who were enrolled in non-intervention protocols ([ClinicalTrials.gov](https://clinicaltrials.gov) Identifiers [ID]: NCT00320177, NCT00631644). All participants were recruited through flyers posted on public bulletin boards at the National Institutes of Health (NIH), local libraries, supermarkets, and school parent email listservs in the Washington, DC greater metropolitan area. Healthy boys and girls between 8 and 18 years with any BMI percentile were eligible for participation. Individuals were excluded if they had a significant medical condition; had abnormal hepatic, renal, or thyroid function; were taking medications known to affect body weight; experienced a weight loss of more than five pounds (2.3 kg) in the past three months; were undergoing weight loss treatment; or had a psychiatric disorder that might impede protocol compliance.

Procedures

Potential participants and a parent/guardian were seen at the NIH Hatfield Clinical Research Center. Interested participants completed self-report questionnaires and a semi-structured interview regarding eating disorder psychopathology; fasting anthropometric measures were also taken. A subset of participants ($n = 223$) from the non-intervention study protocol were scheduled for laboratory test meal visits at the NIH as described previously (Tanofsky-Kraff, McDuffie, et al., 2009). Test meal visits took place following an overnight fast. Informed parental consent and child assent were obtained for all studies.

Measures

Eating in the Absence of Hunger—The Eating in the Absence of Hunger Questionnaire for Children (Tanofsky-Kraff, et al., 2008) is a 14-item self-report questionnaire designed for use with 8–18 year olds. The Parent Report version of the Eating in the Absence of Hunger Questionnaire is a parallel version designed to assess the same construct. These questionnaires assess the frequency with which youth eat when they are not hungry or already sated. Respondents rate the frequency that they eat in the absence of hunger on a 5-point scale ranging from “Never” through “Always.” Both versions of the Eating in the Absence of Hunger Questionnaire generate three subscales that reflect distinct aspects of eating in the absence of hunger, including: 1) External eating; 2) Negative affect; and 3) Fatigue/boredom; they also generate a total score created from averaging the individual item scores. These questionnaires have demonstrated good internal consistency, temporal stability, convergent validity, and temporal stability for all scales (Tanofsky-Kraff, et al., 2008; Zocca, et al., 2011). The Parent Report version, but not the child report version, appears to demonstrate construct validity with laboratory meals designed to capture eating in the absence of hunger in response to external cues (Shomaker et al., in revision). Both questionnaires were used in determining the typology of disinhibited eating behaviors in youth. Cronbach’s alpha for the Parent and Child Report versions were 0.93 and 0.87, respectively.

Emotional Eating—The Emotional Eating Scale Adapted for Children and Adolescents, designed for use with 8–18-year-olds (Tanofsky-Kraff, Theim, et al., 2007), was adapted from the Emotional Eating Scale for adults (Arnow, Kenardy, & Agras, 1995). The Emotional Eating Scale Adapted for Children and Adolescents is a 25-item self-report measure used to assess the propensity to cope with negative affect by eating. Respondents rate their desire to eat in response to each emotion on a 5-point scale from “I have no desire to eat” through “I have a very strong desire to eat.” The Emotional Eating Scale Adapted for Children and Adolescents generates three subscales reflecting the urge to eat in response to: 1) Anger, anxiety, and frustration, 2) Depressive symptoms, and 3) Feeling unsettled. In addition, summing of the individual items generates a total score that ranged from 0 to 50; a score of 13 or greater was the point at which individuals consumed more energy in response to negative emotions in the laboratory (Vannucci, et al., 2011). The Emotional Eating Scale Adapted for Children and Adolescents has demonstrated good psychometric properties (Tanofsky-Kraff, Theim, et al., 2007) and construct validity with observational measures of eating behavior (Vannucci, et al., 2011). Cronbach’s alpha for this measure in the current study was 0.94.

Loss of Control Eating and Eating Disorder Psychopathology—The Eating Disorder Examination version 12.0D/C.2 (EDE; Fairburn & Cooper, 1993) or the child version (Bryant-Waugh, Cooper, Taylor, & Lask, 1996) was used to determine the presence or absence of objective binge episodes (OBE; consumption of an unambiguously large amount of food with a sense of loss of control) and subjective binge episodes (SBE; the experience of loss of control while consuming an ambiguously large amount of food).

Consistent with prior research on loss of control eating in pediatric samples (Glasofer et al., 2007; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff, McDuffie, et al., 2009; Tanofsky-Kraff, et al., 2004), the presence or absence of OBEs and SBEs in the past month—rather than the past three months—was determined. The presence or absence of loss of control eating in the past month was coded if youth reported any OBEs, any SBEs, or both. Additionally, this threshold was determined for the current study because it is thought that children can more reliably recall episodes that occurred recently versus several months prior and the proximal timeframe was more consistent with the self-report questionnaires of disinhibited eating. The EDE has had good interrater reliability for all episode types (Spearman correlation coefficients: 0.70) (Rizvi, Peterson, Crow, & Agras, 2000). The EDE adapted for children has shown good interrater reliability (Spearman correlation coefficients: 0.91 to 1.00) and discriminant validity (Cohen's kappa for presence of different eating episode categories of 1.00) in pediatric samples (Glasofer, et al., 2007; Tanofsky-Kraff, et al., 2004; Watkins, Frampton, Lask, & Bryant-Waugh, 2007). Interrater reliability for the presence or absence of loss of control eating among a subset of the current sample (7%) was very good (Cohen's kappa of 0.92). Group consensus was used to determine the episode size when interviewers thought it was ambiguous; this training is reflected in the discriminant validity for the size of different eating episodes in the current study, which was also very good (Cohen's kappa of 1.00).

The Eating Disorder Examination also generates four subscales from items different than those used for the OBE and SBE assessment, including restraint (cognitive and behavioral dietary restraint), and eating, shape, and weight concern, which average to create a global score of eating disorder psychopathology. The EDE has demonstrated good internal consistency, interrater reliability convergent, and discriminant validity for the subscales and global score (Glasofer, et al., 2007; Rizvi, et al., 2000; Tanofsky-Kraff, et al., 2004; Watkins, et al., 2007).

Depressive Symptoms—The Beck Depression Inventory, Second version (Beck, Steer, & Brown, 1996) is a widely used 21-item self-report measure that assesses depressive symptoms. The Beck Depression Inventory was administered to a subset of participants greater than 13 years ($n = 188$). The Beck Depression Inventory has demonstrated excellent psychometric properties (Ambrosini, Metz, Bianchi, Rabinovich, & Undie, 1991). Cronbach's alpha for the Beck Depression Inventory in the current study was 0.86. The Children's Depression Inventory, Second version (Kovacs, 1992) was administered to the other children and adolescents ($n = 223$). The Children's Depression Inventory is a 28-item self-report measure that assesses depressive symptoms and is suitable for use in children between 7–17 years of age. The Children's Depression Inventory has demonstrated good reliability and validity in community and clinical samples (Sitarenios & Kovacs, 1999; Smucker, Craighead, Craighead, & Green, 1986). Cronbach's alpha for the Children's Depression Inventory in the current study was 0.84.

Anxiety Symptoms—The State Trait Anxiety Inventory for Children is a 20-item scale for measuring anxiety proneness as a personality trait in youth greater than 8 years old (Spielberger, Edwards, Lushene, Montuori, & Platzek, 1973). Children rate anxiety symptoms on a three-point scale: 1 = almost never, 2 = sometimes, and 3 = often. The State Trait Anxiety Inventory for Children has good construct validity, internal consistency, and test-retest reliability (Muris, Merckelbach, Ollendick, King, & Bogie, 2002). Cronbach's alpha for the current study was 0.87.

Parent-Reported Behavior Problems—The Child Behavior Checklist (Achenbach & Elderbrock, 1991) was completed by parents to assess their perceptions of their child's internalizing and externalizing symptoms, as well as overall general problem behaviors, in

the previous six months. The Child Behavior Checklist is a 118-item questionnaire designed for use in children between 6–18 years that uses a 3-point scale from “Not true” to “Very true or often true.” T-scores were provided to facilitate the interpretation of results. The Child Behavior Checklist has shown good internal consistency, test-retest reliability, and construct validity (Achenbach & Elderbrock, 1991). Cronbach’s alpha for the current study was 0.82.

Body Composition—Participants’ weight and height were measured in a fasting state using calibrated electronic instruments. BMI was calculated as weight in kilograms divided by the square of height in meters. BMI standard deviation (BMI-z) scores adjusted for age and sex were calculated according to the Centers for Disease Control and Prevention 2000 growth charts (Kuczmarski et al., 2002). Body fat and fat-free mass (kg) were measured by air displacement plethysmography (Bod Pod; Life Measurement Inc., Concord, CA).

Laboratory Test Meals

Observed Intake During Laboratory Test Meals—Participants from one of the study protocols were asked to consume their lunch *ad libitum* from a buffet test meal on two separate days. The proportion of individuals from this laboratory study represents over half of the current sample (54.3%; $n = 223$). In random order, children and adolescents participated in a “normal meal” (at which they were told to “eat as much as you would at a normal meal”) and a “disinhibited” meal (at which they were instructed to “let yourself go and eat as much as you want”). Other than the instruction, all aspects of the conditions were identical. On the day of each test meal, youth were provided with a standard 288-kcal breakfast (7% protein, 19% fat, 74% carbohydrate). Participants remained at the NIH Clinical Center for the next six hours, during which they were observed to ensure that they consumed no calorie-containing foods or beverages and only participated in sedentary activities. Each participant was then presented with a multiple-item, 9,835-kcal food array varied in macronutrient composition (51% carbohydrate, 12% protein, 37% fat, across all foods) and containing a wide assortment of foods (Mirch et al., 2006). All food items presented were weighed to the nearest 0.1 g before and after the test session. Energy content and macronutrient composition were calculated as previously described (Tanofsky-Kraff, McDuffie, et al., 2009).

Assessment of State Negative Affect Prior to the Laboratory Test Meals—

Immediately prior to and following each laboratory test meal, participants completed the well-validated Brunel Mood Scale (Terry, Lane, Lane, & Keohane, 1999), which measures present mood state. As an overall measure of pre-meal state negative affect, a total score was computed by summing the individual items from the anger, confusion, depression, fatigue, and tension subscales and the reverse-coded items from the vigor subscales. Cronbach’s alpha for the current study was 0.82.

Statistical Analyses

Data were screened for normality. Extreme, but plausible, outliers were recoded to fall 1.5 times the interquartile range below or above the 25th or 75th percentile so they were no longer outliers (Behrens, 1997). This procedure places the outliers in the lower or upper 1–2% of the distribution. No more than 5% of the sample for a given variable was considered an outlier. This a priori strategy was used because it minimizes outliers’ influence on the characteristics of the distribution, minimally changes the distribution overall, and avoids potential bias associated with eliminating outliers altogether (Behrens, 1997). After adjusting the outliers, it was confirmed that skew and kurtosis were satisfactory on all variables. To evaluate whether this approach potentially influenced results, a follow-up

sensitivity analysis was conducted by comparing all validation analyses with dependent variables with and without the outliers recoded; none of the results changed.

Latent profile analysis (LPA) was used to characterize subtypes of disinhibited eating behaviors based on patterns of overlap among children and adolescents. LPA is an empirically-driven approach that uses categorical and continuous indicators in cross-sectional samples to identify latent classes, or subgroups, of individuals (Lazerfeld & Henry, 1968). These subgroups are considered latent because membership is not directly observed, but rather it is inferred by examining the patterns of interrelationships among indicator variables (Lazerfeld & Henry, 1968). LPA identifies subgroups by maximizing homogeneity within each class and maximizing heterogeneity between classes. The LPA was performed with Latent Gold version 4.5 (Vermunt & Magidson, 2005).

Participants were included in the LPA if they completed at least three out of five possible indicators of disinhibited eating, which included: 1) presence or absence of loss of control eating in the previous month; 2) presence or absence of OBEs in the previous month; 3) presence or absence of SBEs in the previous month; 4) Emotional Eating Scale for Children and Adolescents total score; and 5) Eating in the Absence of Hunger total score. There is substantial support for the use of the term loss of control eating in children (Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, et al., 2005; Tanofsky-Kraff, et al., 2004), and adult data indicate that the core feature of binge eating is the presence of loss of control over eating (Wolfe, et al., 2009). Yet, the lack of differences between objective and subjective binge episodes is not yet definitive (Wolfe, et al., 2009); therefore, both objective and subjective binge episodes were also included as indicators of this secondary binge feature. Binge eating features were considered categorically rather than dimensionally because these variables are highly skewed since a small percentage of youth report the presence of loss of control and the vast majority of youth with loss of control report very few episodes in the past month. Additionally, this classification method is consistent with prior studies of loss of control eating and provides an opportunity to generalize to other studies (Goossens, et al., 2007; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff, McDuffie, et al., 2009; Tanofsky-Kraff, Yanovski, et al., 2009; Tanofsky-Kraff, et al., 2004). Although the psychometric properties of the Parent Report version of the Eating in the Absence of Hunger Questionnaire appear to be more robust based on recent data (Shomaker, et al., in revision), the LPA was conducted separately using the Child and Parent Reports.

The most parsimonious number of latent classes was determined by examining four fit indices: 1) the Bayesian information criterion (BIC; Schwarz, 1978); 2) the sample size adjusted BIC (aBIC; Sclove, 1987); and 3) the consistent Akaike information criterion (cAIC; Bozdogman, 1987). The lowest value of these fit indices is indicative of the best fitting model. Data from simulation studies examining general trends in the performance of each indicator suggest that the BIC and cAIC are conservative indices—meaning they tend to underestimate the true number of classes—and that the aBIC is liberal index—exhibiting a tendency to overestimate the true number of classes (Dayton & Macready, 1988; Lin & Dayton, 1997; Yang, 2006). Bivariate residuals, which are indices of the remaining correlations between indicators of disinhibited eating within latent classes (i.e., conditional dependence), were examined to ensure that none had a value greater than 3.0. This value was selected based on the significance test for the residual based upon a chi-square with 1 degree of freedom. The .05 cutoff for the chi-square distribution is 3.84, so we used a residual cutoff of 3.00 to be conservative.

After identifying the best fitting model and examining bivariate residuals, participants were assigned to a latent class on the basis of posterior probabilities. Consistent with the

objectives of the study, this approach was selected to facilitate the clinical interpretation of the classes in the validation analyses. One limitation of this approach is that it does not capture the probabilistic nature of the latent class model and the reality that latent class membership is not fixed. Therefore, it should be recognized that the uncertainty of individuals that comprise each latent class is not captured with the case assignment approach and that potentially relevant measurement error may be ignored in validation analyses. To evaluate the impact of this measurement error, latent classes were compared on body composition and psychological validators entered as covariates within the context of the latent profile analyses after controlling for other relevant covariates.

Analyses of covariance (ANCOVA) were used to validate the subtypes identified in the LPA with regard to body composition, eating disorder psychopathology, and general psychopathology. The independent variable was the latent class group assignment. Dependent variables included BMI-z scores, total fat mass, the EDE global score and subscales (restraint, and eating, shape and weight concern), BDI/CDI scores, trait anxiety scores, and CBCL internalizing, externalizing and total scores. Covariates for all models included age (years), race (coded as non-Hispanic Caucasian or Other), and sex (coded as male or female). Height was included as a covariate for the model examining total fat mass and BMI-z was included as a covariate in models examining psychological variables.

For the subset of youth who participated in the laboratory test meals, linear mixed models with repeated measures were used to examine the relationship between disinhibited eating behavior subtypes and observed energy intake. The repeated measure was meal instruction (normal versus disinhibited). The dependent variables were total energy intake (kcal; logarithm transformed), percent macronutrient content consumed (carbohydrate, fat, and protein; arcsine transformed), and pre- and post-meal ratings of state negative affect. The independent variable was the latent class group assignment. In a second analysis, the two-way interaction of meal instruction and latent class group assignment was tested to determine whether the association between disinhibited eating subtypes and eating behavior varied as a function of the type of meal (i.e., normal versus disinhibited instruction). Covariates included age (years), race (coded as non-Hispanic Caucasian or other), sex (coded as male or female), height (cm), lean mass (kg), percent fat mass (%), and meal instruction. Total energy consumed (kcal; logarithm transformed) was included as a covariate in the models examining percent macronutrient content intake (arcsine transformed) and post-meal state negative affect. Pre-meal negative affect was included as a covariate in the model examining post-meal negative affect.

For all tests examining the validity of the subtypes, SPSS version 19.0 was used. Omnibus tests were considered significant when p values were ≤ 0.05 . Bonferroni-Hochberg post hoc tests were used to examine pair-wise differences when omnibus tests were significant and therefore the p value for determining significant post-hoc differences was often between .01 and .03. The Bonferroni-Hochberg post hoc test was selected to control for the family-wise error rate when conducting multiple comparisons. All tests were two-tailed.

Results

Data from 411 children and adolescents (8 to 18 years, $M = 14.02$ years, $SD = 2.54$; 51.2% female) were included in the LPA. The racial/ethnic breakdown of the sample was 55.4% non-Hispanic White, 32.0% non-Hispanic Black or African American, 4.9% Asian Origin, 2.8% Hispanic/Latino, 1.5% Multiple Races, and 3.4% Other/Unknown. Youth represented a wide range of weight strata (BMI-z score: Range = -2.24 to 3.20 , $M = 0.80$, $SD = 1.11$).

Latent Profile Analysis: Characterization of Disinhibited Eating Subtypes

The extent of missing data for each indicator variable in the LPA models were as follows: 0.5% ($n = 2$) for OBE presence/absence, 0.2% ($n = 1$) for SBE presence/absence, 18.9% ($n = 78$) for emotional eating, and 13.6% ($n = 56$) for eating in the absence of hunger. The extent of this missing data is below the threshold for determining accurate models based on a study using monte carlo simulations (Swanson, Lindenberg, Bauer, & Crosby, 2012). LPA models were evaluated with the number of clusters ranging from one to eight. Bivariate residuals were less than 3.0 for models with four to eight clusters (Table 1), indicating that the conditional independence assumption was met for these models. The LPA models did not converge when including the Child Report version of the Eating in the Absence of Hunger Questionnaire as an indicator, supporting evidence that this measure may not be as psychometrically sound as the Parent Report version.

Overall, fit indices revealed the presence of a four to six cluster solution (Table 1). The cAIC, which is also considered a conservative index, was lowest for a four-cluster model with 31 parameters. The BIC—a conservative index—was lowest for a five-cluster model with 39 parameters. The aBIC, which is considered a liberal index and may overestimate the number of clusters (Crow et al., 2011), was lowest for an six-cluster solution with 46 parameters. Therefore, the five-cluster solution was selected because it used a conservative index and was in between the range of potential solutions. The relative frequency of loss of control eating (OBE and SBE), objective binge episodes, and subjective binge episodes and the relative severity of parent-reported eating in the absence of hunger and self-reported emotional eating for each of the five clusters is depicted in Figure 1 and the associated means are summarize in Table 2.

Cluster 1 comprised 15.6% ($n = 64$) of the sample and resembled an asymptomatic cluster with little to no disinhibited eating behaviors. Among youth in Cluster 1, none reported any episodes of loss of control eating, and nearly non-existent levels of emotional eating were reported; however, there were low levels of reported eating in absence of hunger. Cluster 2 comprised 42.0% ($n = 173$) of the sample and appeared to consist of a group reporting moderate levels of emotional eating and eating in the absence of hunger. None of the youth in Cluster 2 reported the presence of loss of control eating (OBE or SBE). Cluster 3 comprised 15.1% ($n = 62$) of the sample and was characterized by high levels of emotional eating, little to no eating in the absence of hunger, and the absence of loss of control eating (OBE or SBE). Cluster 4 comprised 14.4% ($n = 59$) of the sample and was characterized by the presence of SBEs, high levels of emotional eating, and moderate levels of eating in the absence of hunger. All youth in Cluster 4 reported SBEs, while none reported OBEs. Cluster 5 comprised 12.9% ($n = 53$) of the sample and appeared to consist of a group characterized by high severity with regard to all three disinhibited eating behaviors. All youth in Cluster 5 reported the presence of OBEs, and 52% also reported the presence of SBEs. Cluster 5 youth also reported highest levels of emotional eating and moderate levels of eating in the absence of hunger. For ease of discussion, the clusters will be referred to based on their most distinguishing characteristic: 1) Cluster 1 as the “No Disinhibited Eating (No-DE)” subtype; 2) Cluster 2 as the “Mixed Emotional Eating and Eating in the Absence of Hunger (EE-EAH)” subtype; 3) Cluster 3 as the “Emotional Eating (EE)” subtype; 4) Cluster 4 as the “Subjective Binge Episode (SBE)” subtype; and 5) Cluster 5 as the “Objective Binge Episode (OBE)” subtype.

Disinhibited Eating Subtypes and Clinical Characteristics

Demographics, body composition, and psychological variables based upon disinhibited eating subtypes are summarized in Table 2. There were no differences among the subtypes with racial/ethnic background ($p = .43$). Those in the EE subtype were significant older than

youth in the other subtypes ($p = .003$). Those in the OBE and SBE subtypes were more likely to be female as compared to those in the EE, EE-EAH, and No-DE Subtypes ($p = .002$). Youth in the OBE and SBE subtypes had significantly greater BMI- z scores ($p = .001$) and higher overall fat mass ($p = .01$) than youth in the EE and No-DE subtypes. Youth in the EE-EAH subtypes did not significantly differ from any other subtypes in BMI- z scores and overall fat mass.

Youth in the OBE and SBE subtypes reported higher global eating disorder psychopathology than those in the EE, EE-EAH, and No-DE subtypes ($p < .001$). This pattern of results was the same for restraint, $F(4, 396) = 6.68, p < .001, \eta^2 = .06$, shape concern, $F(4, 394) = 15.74, p < .001, \eta^2 = .14$, and weight concern, $F(4, 394) = 7.70, p < .001, \eta^2 = .07$. Youth in the OBE subtype reported the greatest eating concern ($M = 0.58, SD = 0.81$) as compared to other subtypes, $F(4, 395) = 17.99, p < .001, \eta^2 = .15$. Those in the SBE subtype reported higher eating concern ($M = 0.43, SD = 0.49$) than those in the EE subtype ($M = 0.13, SD = 0.34$), EE-EAH subtype ($M = 0.08, SD = 0.21$), and No-DE subtype ($M = 0.03, SD = 0.08$).

Differences among the disinhibited eating subtypes also emerged with regard to measures of general psychopathology. Among the subset of adolescents that completed Beck Depression Inventory ($n = 188$), those in the OBE, SBE, and EE subtypes reported greater depressive symptoms than those in the EE-EAH and No-DE subtypes ($p = .001$). Among youth who completed the Children's Depression Inventory ($n = 223$), the OBE and SBE subtypes reported higher depressive symptoms than the EE, EE-EAH, and No-DE subtypes ($p < .001$). Similarly, trait anxiety was higher among those in the OBE and SBE subtypes as compared to those in the EE, EE-EAH, and No-DE subtypes ($p < .001$).

As compared to those in the No-DE subtype, youth in the OBE, SBE, EE, and EE-EAH subtypes had higher overall total problems as reported by their parents ($p = .01$). Youth in the OBE ($M = 49.22, SD = 10.59$) and EE ($M = 49.21, SD = 10.83$) subtypes had significantly higher parent-reported internalizing symptoms than those in the No-DE subtype ($M = 44.13, SD = 10.83$); youth in the SBE ($M = 47.57, SD = 12.22$) and EE-EAH ($M = 45.45, SD = 10.08$) subtypes did not significantly differ from any other subtypes in parent-reported internalizing symptoms, $F(4, 380) = 2.71, p = .03, \eta^2 = .03$. Youth in the EE-EAH subtype ($M = 47.86, SD = 10.37$) had significantly higher parent-reported externalizing symptoms as compared to those in all other subtypes ($M = 42.30 - 44.06, SD = 8.62 - 10.45$), $F(3, 380) = 2.39, p = .05, \eta^2 = .02$.

Disinhibited Eating Subtypes and Eating Behavior in the Laboratory

A subset ($n = 223$) of youth participated in a study protocol that involved two test meals. There were no significant differences between youth who did and did not participate in the test meals with regard to subtype group assignment ($p = .72$), sex ($p = .37$), racial/ethnic background ($p = .64$), or BMI- z ($p = .08$). Youth participating in the test meals were significantly younger than those who did not participate in the test meals ($M = 12.98, SD = 2.80$ vs. $15.29, SD = 1.35$ years; $p < .001$).

Differences between disinhibited eating subtypes and mood and eating behavior in the laboratory are depicted in Table 3. After controlling for sex, age, race, height (cm), lean mass (kg), and percent fat mass (kg), and meal instruction, there was a main effect of disinhibited eating subtype on total energy intake ($p = .02$). Specifically, post hoc tests revealed that youth in the SBE subtype consumed less overall energy than those in all other subtypes. Additionally, youth in the OBE and EE subtypes consumed more overall energy than those in the EE-EAH and No-DE subtypes. Youth in the OBE and SBE subtypes consumed a smaller percentage of energy from protein than those in the EE, EE-EAH, and

No-DE subtypes ($p = .01$), after controlling for sex, age, race, height (cm), lean mass (kg), and percent fat mass (kg), meal instruction, and total energy intake (kcal). Regardless of meal type and relevant covariates, the EE subtype consumed a larger percentage of energy from fat than the SBE, EE-EAH, and No-DE subtypes ($p = .01$); the OBE subtype did not significantly differ from any of the disinhibited eating subtypes. Additionally, youth in the OBE, SBE, and EE subtypes consumed a larger percentage of energy from carbohydrate as compared to those in the EE-EAH and No-DE subtypes ($p = .02$). Youth in the OBE, SBE, and EE subtypes reported greater state negative affect before the test meals than the EE-EAH and No-DE subtypes, after accounting for relevant covariates ($p < .001$). Youth in the SBE subtype reported greater state negative affect after the test meals as compared to the OBE, EE, EE-EAH, and No-DE subtypes ($p = .05$), after controlling for demographics, adiposity, pre-meal state negative affect, and total intake during the test meals. There was no interaction of meal type with disinhibited eating subtype for any of the models ($p = .13-.36$).

Secondary Validation Analysis

To evaluate whether using a case assignment approach ignored pertinent measurement error, latent classes were compared on body composition and psychological validators entered as covariates within the context of the latent profile analyses after controlling for other relevant covariates. Overall, the pattern of results remained the same when using this approach, and in most cases the results were even more robust. However, there were a few noteworthy differences. The EE-EAH subtype had significantly lower BMI-z scores and overall fat mass than the OBE and SBE subtypes, but had significantly higher BMI-z scores and overall fat mass than the EE and No-DE subtypes ($p < .01$). Youth in the OBE subtype also had significantly higher overall fat mass than those in the SBE subtype ($p = .01$). The EE and EE-EAH subtypes reported significantly lower shape concern than the OBE and SBE subtypes and significantly higher shape concern than the No-DE subtype ($p < .01$). There were no longer any significant subtype differences in depressive symptoms as reported by the Children's Depression Inventory ($p > .05$). Trait anxiety was higher among those in the OBE, SBE, and EE subtypes as compared to those in the EE-EAH and No-DE subtypes ($p < .03$). The same overall pattern of results emerged for parent-reported total problems, except that those in the SBE subtype had the highest parent-reported total problems ($p < .04$). Youth in the SBE and EE subtypes had higher parent-reported internalizing symptoms than those in the OBE, EE-EAH, and No-DE subtypes ($p < .03$). The SBE and EE-EAH subtypes had higher parent-reported externalizing symptoms as compared to those in the OBE, SBE, and No-DE subtypes ($p < .05$).

Discussion

This study made use of latent profile analysis (LPA) to identify five subtypes based on the co-occurrence of eating in the absence of hunger, emotional eating, and loss of control eating. Results indicated substantial overlap among reports of these disinhibited eating behaviors. However, the subtypes were characterized based on distinguishing features of "objective binge episode (OBE)," "subjective binge episode (SBE)," "emotional eating (EE)," "a mix of emotional eating and eating in the absence of hunger (EE-EAH)," and "no disinhibited eating (No-DE)." Our findings suggest that these subtypes differ on measures of adiposity, psychological functioning, and laboratory eating behavior, such that the loss of control (OBE and SBE) subtypes were the most pathological in terms of reported and objective measures. The EE subtype also exhibited elevated psychopathology and aberrant eating behavior in the laboratory. Our findings highlight the importance of considering the entire range of disinhibited eating behaviors in youth and provide novel information about the characterization of pediatric disinhibited eating behaviors.

The No-DE subtype was characterized by youth who reportedly ate in the absence of hunger occasionally and had no emotional eating or loss of control eating. Notably, despite a wealth of research on eating in the absence of hunger (Cutting, et al., 1999; Fisher & Birch, 2002; Fisher, et al., 2007; Hill, et al., 2008; Moens & Braet, 2007; Shomaker, Tanofsky-Kraff, Zocca, et al., 2010; Zocca, et al., 2011), a distinct subtype distinguished primarily by high levels of eating in the absence of hunger did not emerge. Indeed, it is possible that the contemporary obesogenic environment creates conditions in which the vast majority of children and adolescents—at least occasionally—rely on external food cues rather than internal hunger and satiety signals (Wardle, 2005). Therefore, eating in the absence of hunger—when simultaneously considering other affectively-influenced disinhibited eating dimensions—may not be particularly clinically meaningful. In support of this notion, a prospective link between eating in the absence of hunger and obesity has yet to be demonstrated (Butte et al., 2007). However, it is possible that eating in the absence of hunger in vulnerable youth may elevate risk for the development of more pathological disinhibited eating behaviors, chronic overeating, and obesity.

Children and adolescents comprising the EE-EAH subtype reported moderate levels of emotional eating and were described by parents as sometimes eating in the absence of hunger, but they had no loss of control eating. Findings indicate that the co-occurrence of occasional emotional eating and eating in the absence of hunger in pediatric samples may be an indicator of some emerging mood and behavior problems. Specifically, youth with the EE-EAH subtype had higher parent-reported behavior problems and had greater state negative affect prior to the test meals as compared to those from the No-DE subtype. Intriguingly, the EE-EAH subtype was the only disinhibited eating subtype to have higher parent-reported externalizing behavior problems than the No-DE subtype. This preliminary finding suggests that these youth may potentially be more reactive towards their surrounding environment, which is consistent with Schachter's (1968) notion that "external" individuals are overly responsive to all environmental cues in addition to those related to food. Youth in this subtype did not significantly differ in their adiposity from youth in other subtypes, with mean BMI-z and percent fat mass scores falling between the LOC (OBE and SBE) and non-LOC (EE and No-DE) subtypes. Yet, our data also suggest that these youth were not yet engaging in laboratory disordered eating behavior or experiencing disordered eating attitudes. Results represent the first known data on a subtype of youth distinguished by the co-occurrence of emotional eating and eating in the absence of hunger, and require replication to further characterize this subset in relation to clinical correlates.

Children and adolescents comprising the EE subtype reported very high levels of emotional eating, but they had no loss of control eating and only occasional parental reports of eating in the absence of hunger. As compared to the EE-EAH and No-DE subtypes, the EE subtype had similar adiposity and disordered eating attitudes but exhibited greater psychopathology and aberrant eating behavior in the laboratory. In fact, youth with the EE subtype had comparable depressive symptoms (BDI only), parent-reported internalizing symptoms, and pre-meal state negative affect to those with OBE and SBE subtypes. These findings are consistent with prior studies that demonstrated a positive association between emotional eating and psychological dysfunction (Braet & van Strien, 1997; Nguyen-Rodriguez, et al., 2008; Tanofsky-Kraff, Theim, et al., 2007; van Strien, Engels, Leeuwe, & Snoek, 2005) and with affective theories positing that frequent reports of emotional eating should be accompanied by a frequent experience of negative affective states (Dubé, et al., 2005; Heatherton & Baumeister, 1991). Similar to those in the OBE subtype, youth in the EE subtype consumed greater overall energy (~100 kcal) and a higher percentage of calories from carbohydrate in the laboratory as compared to those in the EE-EAH and No-DE subtypes. However, those in the EE subtype consumed a lower percentage of their total

intake from fat relative to youth in other subtypes. The impact of this eating pattern on weight outcomes should be investigated over time.

Two subtypes emerged from the LPA that were differentiated from other subtypes by the presence of loss of control eating. The OBE subtype consisted of youth who all reported consuming unambiguously large amounts of food while experiencing a sense of loss of control. In contrast, the SBE subtype was comprised of children and adolescents who reported the presence of only ambiguously large binge episodes. High emotional eating and occasional eating in the absence of hunger co-occurred with loss of control eating in these subtypes. These findings are comparable to previous studies indicating an overlap among the disinhibited eating behaviors of loss of control, eating in the absence of hunger, and emotional eating (Goossens, et al., 2007; Moens & Braet, 2007; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, et al., 2008; Tanofsky-Kraff, Theim, et al., 2007; Zocca, et al., 2011). Also in line with previous findings (Shomaker, Tanofsky-Kraff, Elliott, et al., 2010), the loss of control subtypes had higher adiposity, eating disorder psychopathology, and trait anxiety than the EE, EE-EAH, and No-DE subtypes, but did not differ from each other on these measures. In comparison to the EE-EAH and No-DE subtypes, the loss of control eating subtypes—consistent with prior research (Hilbert, et al., 2010; Tanofsky-Kraff, McDuffie, et al., 2009)—consumed test meals that were lower in protein and higher in carbohydrate composition. However, the OBE, SBE, and EE subtypes were similar with regard to measures of depression and internalizing behaviors; this novel finding highlights the potential clinical value of assessing the full range of disinhibited eating in youth.

Despite the similarities between OBE and SBE subtypes, there were marked differences that provide new information about the utility of subtyping youth above and beyond the presence of loss of control eating. Youth comprising the OBE subtype reported higher eating concerns than those in the SBE subtype, suggesting that the co-occurrence of all the disinhibited eating behaviors in youth may be associated with elevated eating disorder psychopathology. Despite the similarity of the macronutrient composition of the test meals between the OBE and SBE subtypes, the overall amount of food consumed by youth in these subtypes differed substantially. As compared to the EE-EAH and No-DE subtype youths, the OBE subtype youths consumed approximately 100 kcal more whereas the SBE subtype youths consumed approximately 200 kcal less. If these results generalize to the natural environment, those with the SBE subtype may be less prone to the excessive weight gain associated with loss of control eating. However, it is also possible that youth with the SBE subtype overeat following periods of restriction, and may explain why loss of control episodes, regardless of episode size, promote excess weight gain (Tanofsky-Kraff, Yanovski, et al., 2009). Furthermore, SBE subtype youths reported greater state negative affect following the test meals as compared to those in other subtypes, indicating that there may be some increased distress following eating in this subset of youths. It could also be argued that SBEs are more pathological; distress may be more expected after someone eats a large amount of food but is more surprising after an individual consumes a small or normative amount of food. These findings indicate that there may be subtle, yet clinically important, differences between youths in the OBE and SBE subtypes. Longitudinal studies are needed to examine the long-term eating and weight outcomes of these disinhibited subtypes distinguished by loss of control eating.

Overall, findings from the current study provide compelling support for distinct disinhibited eating subtypes. Therefore, it seems prudent for clinicians to assess for the presence of all disinhibited eating behaviors in order to obtain a more comprehensive clinical picture of behaviors associated with eating, weight, and psychological problems. Future research should explore these behaviors from a developmental perspective to determine the time

course of the emergence of disinhibited eating behaviors. That is, it is possible that some disinhibited eating behaviors emerge earlier than others. For instance, studies have shown that eating in the absence of hunger can be observed in children as young as four years old (Fisher & Birch, 2002; Fisher, et al., 2007), whereas emotional eating is rarely reported in early childhood (Carper, Fisher, & Birch, 2000; Wardle, et al., 2001) and starts to be seen with greater frequency during middle childhood (Shapiro, et al., 2007; Tanofsky-Kraff, Theim, et al., 2007; van Strien & Oosterveld, 2008). Retrospective reports in adults with binge eating disorder (Abbott et al., 1998; Grilo & Masheb, 2000; Spurrell, Wilfley, Tanofsky-Kraff, & Brownell, 1997) and overweight children (Tanofsky-Kraff, et al., 2005) suggest that loss of control eating often may begin between middle childhood and early adolescence. It is conceivable that eating in the absence of hunger may increase risk for the development of emotional eating and subsequently binge eating in youth. Prospective studies that assess all of these disinhibited eating behaviors are needed to clarify the temporal relationships among the development of these behaviors.

A strength of this study is the application of LPA to investigate the typology of pediatric disinhibited eating behaviors. There are no studies that have used this empirical classification method to characterize the pattern of co-occurrence among subthreshold disordered eating behaviors and the associated clinical significance. Further, the validation analyses were similar when using a case assignment approach and a latent variable approach; this consistency across methods increases confidence in the study findings. Additional study strengths include the large and diverse community sample of children and adolescents spanning a broad weight range and the use of interview assessment of eating disorder psychopathology. While the objective assessment of energy intake at a test meal designed to capture disinhibited eating represents an important strength, there also may be limited generalizability of these findings to the natural environment. Future studies of disinhibited eating behaviors would benefit from using ecological momentary assessment to capture affect and eating patterns. Another limitation is the cross-sectional design that limits our capacity to make causal inferences; longitudinal studies of disinhibited eating behaviors are required. Finally, the nature of the recruitment methods that targeted children interested in eating behavior studies may limit generalizability to the general population. Replication of these findings in treatment-seeking samples is needed.

In conclusion, this study suggests that LPA can be used to identify subtypes based on the co-occurrence of eating in the absence of hunger, emotional eating, and loss of control eating in children and adolescents. These findings highlight the heterogeneity of youth with disinhibited eating, which may confer differential risk for obesity and eating disorders. Although the presence of loss of control eating may be the most salient overall indicator of eating- and weight-related problems in youth, findings suggest that there may be clinical utility for subtyping youth based on the presence of OBEs and SBEs when they co-occur with emotional eating and eating in the absence of hunger. Additionally, high levels of emotional eating alone may signal depressive symptomatology and a unique eating pattern. Within the context of other disinhibited eating behaviors, eating in the absence of hunger in and of itself does not appear to be clinically meaningful in children and adolescents. Prospective research is required to determine whether disinhibited eating subtypes are predictive of distinct eating and weight outcomes over time. Since findings support the notion that OBE, SBE, and EE subtypes are associated with notable distress and aberrant eating behavior, the development and evaluation of intervention programs for these subtypes is warranted.

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Figure 1.

Relative Frequency of Latent Profile Analysis Indicators Among Disinhibited Eating Subtypes.

Note: The figure depicts the relative frequency of the five indicator variables for the four disinhibited eating subtypes that emerged from the latent profile analysis. The presence or absence of loss of control eating (LOC) in the past month, which included both objective binge episodes (OBEs) and subjective binge episodes (SBEs), was assessed using the Eating Disorder Examination. Emotional eating (EE) was assessed via self-report, while youths' eating in the absence of hunger (EAH) were assessed via parent report.

*LOC, OBE, and SBE reflect the percentage of youth in each subtype reporting the presence of each indicator. EES-C and EAH-P scores represent the standardized means for each subtype.

Fit Indices for the Latent Profile Analysis Determining the Typology of Pediatric Disinhibited Eating Behaviors.

Table 1

Clusters	Parameters	BIC	aBIC	cAIC	LL	Entropy	Largest Residual
1	7	4581.81	4559.60	4588.81	-2269.86	1.00	263.49
2	15	4161.55	4113.95	4176.55	-2035.65	0.99	33.26
3	23	4097.75	4024.76	4120.75	-1979.69	0.83	33.30
4	31	4076.05	3977.69	4107.05	-1944.78	0.85	0.83
5	39	4073.17	3949.41	4112.17	-1919.27	0.79	1.80
6	47	4085.51	3936.37	4132.51	-1901.38	0.77	0.50
7	55	4118.58	3944.05	4173.58	-1893.84	0.78	0.41
8	63	4147.32	3947.41	4210.32	-1884.15	0.75	0.22

Note: BIC = Bayesian Information Criterion; cAIC = Consistent Akaike Information Criterion; aBIC = sample size adjusted BIC; LL = Log-likelihood; Lower BIC, aBIC, and cAIC values indicate better model fit. Entropy is a measure of classification accuracy, with high values indicating better accuracy. The high residual tests the conditional independence assumption (should be less than 3.0).

Table 2

Disinhibited Eating Subtype Differences with Regard to Demographic Variables, Disinhibited Eating Behaviors, Body Composition, and Eating Disorder and General Psychopathology.

Variable	OBE Subtype (n = 53)		SBE Subtype (n = 59)		EE Subtype (n = 62)		EE-EAH Subtype (n = 172)		No-DE Subtype (n = 64)		Test statistic	p value	Effect Size ^d
	M	SD	M	SD	M	SD	M	SD	M	SD			
Age (years)	14.02 _a	2.53	13.83 _a	2.63	15.15 _b	2.01	13.99 _a	2.51	13.49 _a	2.55	F(4, 401) = 3.99	.003	.04
BMI-z	1.16 _a	1.11	1.12 _a	0.99	0.47 _b	0.98	0.79 _{ab}	1.11	0.53 _b	1.17	F(4, 397) = 5.85	.001	.05
Total fat mass (%)	30.19 _a	12.93	29.91 _a	10.61	22.27 _b	10.82	25.15 _{ab}	12.50	23.17 _b	11.12	F(4, 393) = 3.54	.01	.04
EAH	2.03 _a	0.67	2.15 _a	0.68	1.86 _a	0.46	1.97 _a	0.52	1.36 _b	0.25	F(4, 344) = 21.61	<.001	.16
Emotional Eating	20.26 _a	16.08	20.00 _a	14.35	34.90 _b	9.29	10.63 _c	5.67	1.58 _d	1.41	F(4, 322) = 103.23	<.001	.56
LOC episodes	3.71 _a	5.07	2.53 _b	2.77	0.00 _c	0.00	0.00 _c	0.00	0.00 _c	0.00	F(4, 398) = 42.52	<.001	.30
OBE episodes	2.58 _a	4.13	0.00 _b	0.00	0.00 _b	0.00	0.00 _b	0.00	0.00 _b	0.00	F(4, 396) = 31.93	<.001	.25
SBE episodes	1.13 _a	3.09	2.53 _b	2.77	0.00 _c	0.00	0.00 _b	0.00	0.00 _c	0.00	F(4, 397) = 17.31	<.001	.26
EDE Global Score	1.06 _a	1.00	0.98 _a	0.85	0.37 _b	0.46	0.38 _b	0.47	0.27 _b	0.25	F(4, 396) = 20.14	<.001	.17
BDI Scores	8.31 _a	6.79	6.65 _a	5.46	6.13 _a	5.14	3.83 _b	3.62	3.00 _b	3.53	F(4, 175) = 5.26	.001	.11
CDI Scores	8.55 _a	8.12	7.69 _a	5.45	4.67 _b	4.05	4.73 _b	4.32	3.39 _b	3.37	F(4, 214) = 6.76	<.001	.11
Trait Anxiety	35.65 _a	5.16	33.18 _a	8.49	29.47 _b	7.08	28.70 _b	5.70	28.31 _b	6.40	F(4, 211) = 11.20	<.001	.15
CBCL Total Problems	48.71 _a	11.69	47.30 _a	12.85	47.24 _a	10.76	45.47 _a	11.40	40.87 _b	10.25	F(4, 380) = 3.67	.01	.04
	n	%	n	%	n	%	n	%	n	%			
Female	34	64.2 _a	40	67.8 _a	34	54.8 _b	79	45.9 _b	23	35.9 _b	χ^2 (4, n = 411) = 24.28	.002	.24
Non-White Ethnicity	27	50.9	27	45.8	22	35.5	75	43.6	32	50.0	χ^2 (4, n = 410) = 3.80	.43	.10

Note: Subscripts that differ represent differences between the subtypes using a Bonferroni-Hochberg post-hoc test. All LOC, OBE, and SBE episode means reflect the average number of episodes in the month prior to assessment. LOC = Loss of control eating; OBE = Objective binge episode; SBE = subjective binge episode; EE = Emotional eating; EAH = Eating in the absence of hunger; No-DE = No disinhibited eating; EDE = Eating Disorder Examination; BDI = Beck Depression Inventory; CDI = Children's Depression Inventory; CBCL = Child Behavior Checklist (parent-reported psychopathology).

^aEffect sizes for analyses of variance are partial η^2 ; effect sizes for chi-square tests are ϕ .

Table 3
Disinhibited Eating Subtype Differences with Regard to State Negative Affect and Eating Behavior in the Laboratory.

Variable	OBE Subtype (n = 31)		SBE Subtype (n = 35)		EE Subtype (n = 30)		EE-EAH Subtype (n = 91)		No-DE Subtype (n = 36)		Test statistic	P value
	M	SE	M	SE	M	SE	M	SE	M	SE		
Pre-meal negative affect	6.88 _a	0.54	6.86 _a	0.56	6.31 _a	0.51	4.81 _b	0.31	3.07 _c	0.49	F(4, 540) = 10.52	<.001
Post-meal neg. affect	2.79 _a	0.35	3.76 _b	0.35	2.81 _a	0.34	2.56 _a	0.20	2.53 _a	0.32	F(4, 371) = 2.40	.05
Total intake	1538.50 _a	64.02	1268.23 _b	63.60	1532.65 _a	63.07	1461.52 _c	36.42	1419.78 _c	58.32	F(4, 378) = 2.97	.02
% Protein	13.76 _a	0.48	13.78 _a	0.47	15.82 _b	0.47	15.05 _b	0.27	15.88 _b	0.43	F(4, 377) = 3.51	.01
% Fat	36.60 _{ab}	0.83	37.63 _a	0.82	35.86 _b	0.82	38.78 _a	0.47	38.61 _a	0.74	F(4, 377) = 3.34	.01
% Carbohydrate	51.16 _a	1.09	49.92 _a	1.08	49.77 _a	1.07	47.56 _b	0.62	46.92 _b	0.98	F(4, 377) = 3.09	.02

Note: N = 223. Means and standard errors are presented as a representation from both test meals. Subscripts that differ represent differences between the subtypes using a Bonferroni-Hochberg post-hoc test. OBE = Objective binge episode; SBE = subjective binge episode; EE = Emotional eating; EE-EAH = Mixed emotional eating and eating in the absence of hunger; No-DE = No disinhibited eating