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A Prospective Study of Pediatric Loss of Control Eating and Psychological Outcomes

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Abstract

Loss of control (LOC) eating in youth is associated cross-sectionally with eating-related and psychosocial distress, and is predictive of excessive weight gain. However, few longitudinal studies have examined the psychological impact and persistence of pediatric LOC eating. We administered the Eating Disorder Examination (EDE) and self-reported measures of depressive and anxiety symptoms to 195 boys and girls ($M = 10.4$, $SD = 1.5$ y) at baseline and again 4.7 ($SD = 1.2$) y later to 118 of these youth. Missing data were imputed. Baseline report of LOC was associated with the development of partial or full-syndrome binge eating disorder ($p=.03$), even after accounting for the contribution of sex, race, baseline characteristics (age, disordered eating attitudes and mood symptoms), body mass index growth between baseline and follow-up, and years in study. Half (52.2%, $CI=1.15-6.22$) of children who endorsed experiencing LOC at baseline reported persistence of LOC at follow-up ($p=.02$). Compared to children who never reported LOC eating or reported LOC only at baseline, those with persistent LOC experienced significantly greater increases in disordered eating attitudes ($ps<.001$) and depressive symptoms ($p=.027$) over time. These data suggest that LOC eating in children is a problematic behavior that frequently persists into adolescence and that persistent LOC eating is associated with worsening of emotional distress.

Keywords

Binge eating disorder; loss of control eating; depression; childhood; adolescence

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Introduction

Binge eating disorder (BED), presently considered a form of “eating disorder not otherwise specified” (APA, 2000), is characterized by recurrent episodes of binge eating without regular compensatory behaviors. Binge episodes are defined as the consumption of a large amount of food during which a sense of loss of control over eating is experienced (APA, 2000). BED is common among obese adults and is associated with dysfunctional eating attitudes, marked psychiatric distress (Wilfley, Wilson, & Agras, 2003), and impairments in physical health (Johnson, Spitzer, & Williams, 2001). With the inclusion of BED under consideration for the next edition of the Diagnostic and Statistical Manual of Mental Disorders, establishing the clinical relevance of early eating patterns that may precede the development of the disorder is warranted.

Very limited longitudinal research has examined the precursors of BED. However, prospective studies in adolescent samples have identified some predictors of eating disorders that include binge eating in their symptomatology (Killen, et al., 1996; Killen, et al., 1994; McKnight Investigators, 2003). Three prospective studies of adolescent girls who did not carry a diagnosis of a subclinical or clinical eating disorder but who scored highly on measures of “weight concern” (Killen, et al., 1996; Killen, et al., 1994) and thin body preoccupation (McKnight Investigators, 2003) were found to be at high risk for developing partial- or full-syndrome eating disorders. A fourth study that examined overweight adolescent boys and girls replicated the finding that weight importance predicted disordered eating (Neumark-Sztainer, Wall, Story, & Sherwood, 2009). In this study, binge eating at baseline was not predictive of increases in disordered eating, but the study did not specifically examine the development of partial or full-syndrome eating disorders. To our knowledge, no study has prospectively examined the development of disordered eating, including the development of BED, in younger children. Studying children prior to adolescence is especially warranted because the emergence of disordered eating behavior may begin as early as middle childhood (Tanofsky-Kraff, 2008).

Although youth often report binge eating, it is typically with less frequency than required to meet criteria for BED (Tanofsky-Kraff, 2008). Traditional interview assessments of binge eating behavior require that a binge episode be diagnosed *only* if the amount of food consumed is deemed “unambiguously large” (Bryant-Waugh, Cooper, Taylor, & Lask, 1996; Fairburn & Cooper, 1993). Given the varying energy needs of physically developing boys and girls, it is often difficult to make this determination for children of different ages. For example, the consumption of an entire large pizza by a child or adolescent of any age would likely be considered *unambiguously* large. By contrast, an amount of five slices of pizza eaten by a 16 year old boy might be less clear and thus deemed an *ambiguously* large amount of food that, even if accompanied by a sense of loss of control (LOC) over eating, might not be classified as an objective binge eating episode. However, the experience of LOC over eating, *regardless* of whether the reported amount of food consumed is considered unambiguously or ambiguously large, is common in youth (Tanofsky-Kraff, 2008). Thus, the term LOC eating, as opposed to binge eating, is often adopted when working with children and adolescents in order to be inclusive of all episodes involving LOC (Shomaker, et al., In press).

Most of the existing pediatric literature describes children who report only one episode of LOC eating in the month prior to assessment or even less frequent episodes (Tanofsky-Kraff, 2008). In spite of its low frequency, LOC eating in children is associated with greater body mass index (BMI, kg/m²) and body fat mass as well as greater psychological distress compared to youth without such behaviors (Tanofsky-Kraff, 2008). Both reported binge (Field, et al., 2003; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Tanofsky-Kraff, et

al., 2006) and LOC (Tanofsky-Kraff, Yanovski, et al., 2009) eating in youth predict excessive body weight gain in longitudinal studies of children and adolescents. Although infrequent LOC episodes in young children might be anticipated to be precursors to exacerbated disordered eating, this supposition has not been well-tested. None of the aforementioned prospective studies of eating disorders has examined LOC eating as a predictor (Killen, et al., 1996; Killen, et al., 1994; McKnight Investigators, 2003; Neumark-Sztainer, et al., 2009).

Few theoretical models exist to describe the development of exacerbated disordered eating in young children. Adult theories suggest that the development and maintenance of binge eating episodes are tied to negative affect (e.g., Heatherton & Baumeister, 1991; Leon, Fulkerson, Perry, & Early-Zald, 1995). Binge eating may temporarily reduce momentary negative affective states by serving as a maladaptive coping strategy (Arnow, Kenardy, & Agras, 1992) or as an “escape” from self-awareness (Heatherton & Baumeister, 1991). Yet, a pernicious cycle is proposed to develop in which binge eating ultimately promotes worsening of mood (Barker, Williams, & Galambos, 2006). Longitudinal data examining the development of depression among adolescent girls indicate that symptoms of bulimia nervosa, including binge eating, predict the onset of major depression (Stice, Hayward, Cameron, Killen, & Taylor, 2000). Additionally, adolescent girls’ depressive symptoms and binge eating may interact cyclically to maintain binge eating behaviors (Presnell, Stice, Seidel, & Madeley, 2009).

Although the temporal relationship between LOC eating and negative affect has not been well studied in youth, it might be expected that pre-adolescent children’s LOC eating would precede and predict the development of exacerbated eating-related and negative affect. Whereas the onset of LOC eating episodes often are reported during childhood (Tanofsky-Kraff, 2008), clinically relevant shape, weight, and eating concerns, dietary restraint, and depressive symptoms typically do not emerge until adolescence (Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000; Stice, Killen, Hayward, & Taylor, 1998). Although self-reports of negative moods states among non-treatment-seeking children with LOC eating patterns are consistently higher than youth without LOC, scores are typically well below clinically significant levels (Tanofsky-Kraff, 2008). Children with LOC often report an experience of “numbing” – a feeling that they are unaware of what is going on in the moment – during LOC episodes (Tanofsky-Kraff, et al., 2007), suggesting both that children with LOC may have difficulty describing emotional states, and that LOC behavior may evolve into serving a similar affective coping function as has been described among adults (Arnow, et al., 1992; Heatherton & Baumeister, 1991). Likewise, the state negative affect that often ensues from children’s LOC eating episodes (Tanofsky-Kraff, et al., 2007), (Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008) could be expected to promote affective distress and worsening of disordered eating attitudes and behaviors as youth enter adolescence.

We therefore tested a number of hypotheses about the prospective relationships among childhood LOC, disordered eating (restraint, shape, weight, and eating concern) and negative affect (measured as depressive and anxiety symptoms). Our primary hypothesis was that childhood LOC eating would predict increased disordered eating and negative affect in adolescence. Furthermore, we expected that adolescents whose childhood LOC persisted (i.e., who reported LOC eating at both baseline and follow-up) would experience the poorest psychosocial functioning at follow-up, compared to those who never had LOC or whose childhood LOC resolved (i.e., who only reported LOC episodes at baseline). Finally, although the prevalence of eating disorders is relatively low during early adolescence (Lewinsohn, Striegel-Moore, & Seeley, 2000; Stice, Presnell, & Bearman, 2001), we investigated LOC eating among middle childhood youth as a precursor of partial and full-syndrome BED during adolescence.

Methods

Participants

A non-treatment seeking sample of overweight and non-overweight children (6–13 y) was studied between July, 1999 and August, 2009. By design, the sample was enriched for overweight children. Participants were recruited through two waves of notices mailed to 1st through 5th grade children in the Montgomery County and Prince Georges County, Maryland school districts, advertisements in local newspapers, and two mailings to local family physicians and pediatricians. Mailings to families and physicians requested participation of children willing to undergo phlebotomy (multiple blood draws) and imaging assessments (i.e., magnetic resonance imaging, pelvic ultrasound for girls, wrist x-ray for determination of bone age) for studies investigating hormones and metabolic functioning in children. Mailings also specified that no treatment would be offered. In addition to the questionnaire and interview assessments, for those families who agreed to participate, children also underwent air displacement plethysmography (Life Measurement Inc., Concord, CA), bioelectrical impedance, skinfold thickness to determine body composition, various measures of insulin sensitivity, repeated urine collections, and a medical history and a physical examination that involved determination of pubertal stage by a pediatric endocrinologist or nurse practitioner. Children's compensation ranged from \$70 to \$170 per visit depending upon their level of participation, with the higher amount for the full assessment panel. Approximately 7% of families responded to each of the school mailings, and subjects recruited directly from these mailings constituted 88% of all subjects studied. All understood that they would not receive treatment as part of the study, but would be financially compensated for their participation. Subjects were healthy and medication-free for at least two weeks prior to baseline evaluation. Children provided written assent and parents gave written consent for participation in the protocol. This study was approved by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development Institutional Review Board.

Procedures

At baseline, the *Eating Disorder Examination version 12OD/C.2* (EDE) (Fairburn & Cooper, 1993) adapted for children (Bryant-Waugh, et al., 1996) was administered to each participant to determine the presence or absence of LOC eating, as described previously (Tanofsky-Kraff, et al., 2007; Tanofsky-Kraff, et al., 2004). Based on their responses to the child EDE, participants were categorized as engaging in objective binge episodes (unambiguous overeating with LOC), subjective binge episodes (LOC with ambiguous overeating or without overeating), objective overeating (overeating without LOC), or no episodes (normal meals involving neither LOC nor overeating) over the 28 days prior to assessment. As described previously (Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005), all children were queried as to whether or not they had *ever* experienced LOC over eating (reporting at least one instance of an objective or a subjective binge episode ever, LOC Ever). The EDE generates four subscales: restraint (cognitive and behavioral dietary restraint), eating concern, shape concern, and weight concern, as well as a global score. These continuous variables were used as measures of children's disordered eating attitudes. Variables generating the subscales are independent of those identifying eating episodes (Tanofsky-Kraff, et al., 2004). Tests of the EDE adapted for children have demonstrated good inter-rater reliability (Spearman rank correlations from .91–1.00) and discriminant validity in eating disordered samples and matched controls ages 8–14 years (Christie, Watkins, & Lask, 2000). Among non-overweight and overweight 6–13 year olds, the child version of the EDE revealed excellent inter-rater reliability with a Cohen's kappa for presence of the different eating episode categories of 1.00 ($p < .001$) (Tanofsky-Kraff, et al., 2004). The child version differs from the adult EDE only in that its script has been edited

to make it more accessible to children ages 8–14 years. Both versions generate the same eating episodes and subscales. In a sample including a broad age spectrum (8–18y), the child and adult EDEs have been successfully combined (Tanofsky-Kraff, et al., 2007). The EDE has good inter-rater reliability for all episode types (Spearman correlation coefficients: $\geq .70$) (Rizvi, Peterson, Crow, & Agras, 2000).

Data collection at follow-up was identical to the baseline assessment other than the adult EDE (Fairburn & Cooper, 1993), as opposed to the child version, was administered to all returning participants. Although children were seen annually for physical assessments, the EDE was only administered at the baseline and follow-up visits.

In addition, the *Standard Pediatric Eating Episode Interview (SPEEI)* (Tanofsky-Kraff, et al., 2007) was administered following the overeating section of the EDE to assess the contextual, behavioral, physical, and emotional aspects of aberrant eating episodes, including the associated features of DSM-IV-TR binge episodes (APA, 2000). The SPEEI is designed to distinguish eating episodes with LOC from those without LOC (Tanofsky-Kraff, et al., 2007).

Partial syndrome BED was defined as reports of fewer than eight episodes per month for 6 months of LOC eating involving unambiguously large amounts of food (full-syndrome BED) but at least four episodes of LOC eating involving equivocally large and/or unambiguously large amounts of food on average per month, for at least three months. For partial or full-syndrome BED, LOC episodes were characterized by at least three DSM-IV-TR associated features of binge eating episodes (e.g., eating more rapidly than normal, eating when not physically hungry, etc.) (APA, 2000). Although consistent with one study of children and adolescents (Tanofsky-Kraff, et al., 2007), this definition is more conservative than that of most adult studies that have typically used a frequency criterion of at least one binge or LOC episode per month (Crow, Agras, Halmi, Mitchell, & Kraemer, 2002; Striegel-Moore, et al., 2000).

At baseline and follow-up, participants completed the Children's Depression Inventory, a reliable and well-validated 27-item self-rated measure of depressive symptoms (Kovacs, 1982). Internal consistency reliability in this widely used measure is good, with coefficients ranging from .71 to .89 across healthy and depressed samples of children age seven to 17 years (Kovacs, 1982). The total score was used. They also completed the State-Trait Anxiety Inventory for Children A—Trait Scale, a 20-item self-report measure of trait anxiety that is widely used and psychometrically sound (Spielberger, 1983). Internal consistency for this well-validated questionnaire is very good, with correlations ranging from .78 to .87 in samples of elementary school children (Spielberger, 1983). Height and weight were measured, and body mass index (BMI, kg/m^2) was calculated, as previously described (Tanofsky-Kraff, et al., 2004).

Statistical Analysis

Analyses were conducted with SPSS 16.0 or SAS 8.0. Skew and kurtosis were satisfactory on all variables, and outliers were adjusted to fall 1.5 times the interquartile range below or above the 25th or 75th percentile (Behrens, 1997). This 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. Demographic characteristics for those with missing data were examined with independent samples t-tests or chi-square analyses to test differences between children who did and did not complete a follow-up assessment. Missing data were imputed using the multiple imputation (MI) procedure in SAS. The missing data model included demographic variables of age (y), sex (male versus female), race/ethnicity (non-Hispanic Caucasian

versus other), socioeconomic status (Hollingshead, 1975), and puberty according to the stages of Tanner (Marshall & Tanner, 1969, 1970), years in study, and both baseline and follow-up values of BMI (kg/m²), disordered eating attitudes (restraint, eating concern, shape concern, and weight concern subscales and global score), LOC eating (presence versus absence) and symptoms of depression and anxiety. Due to the large missing data fraction, ten imputed data sets were produced (Allison, 2009; Schafer & Graham, 2002). Following standard procedures for MI, each data set was analyzed separately, and the results for the ten data sets were combined using the MIANALYZE procedure in SAS.

Independent samples t-tests were used to compare youth with and without baseline LOC Ever (presence versus absence) on demographic characteristics, baseline and follow-up disordered eating, depressive, and anxiety symptoms. A series of analyses of covariance (ANCOVAs) were conducted to examine whether the independent variable of baseline LOC Ever (presence versus absence) predicted the dependent variables of follow-up disordered eating attitudes (global, restraint, eating concern, shape concern, and weight concern), symptoms of depression, and symptoms of anxiety. We examined both “simple” adjusted models that accounted for years between baseline and follow-up and the respective baseline symptom (disordered eating, depressive or anxiety symptoms), and models that adjusted for these covariates as well as baseline age (y), sex (male versus female), race/ethnicity (non-Hispanic Caucasian versus other), and BMI change between baseline and follow-up.

Logistic regression was used to describe the unadjusted odds of the dependent variable of LOC at follow-up (presence versus absence) based upon the independent variable of baseline LOC Ever status (presence versus absence). Odds of LOC at follow-up based upon baseline LOC Ever also was examined after adjusting for years between baseline and follow-up. Change in number of LOC eating episodes in the past month (defined continuously) between baseline and follow-up were described with paired samples t-tests among the whole sample, as well as just among children who reported persistent LOC eating (present at both baseline and follow-up). Repeated measures analysis of variance was used to adjust these comparisons for time between baseline and follow-up. Another series of ANCOVAs were conducted to investigate whether the independent variable of LOC persistence predicted the dependent variables of follow-up disordered eating attitudes (global, restraint, eating concern, shape concern, and weight concern), depressive symptoms, and anxiety symptoms. LOC persistence was defined categorically as: i) no LOC at baseline or follow-up (“never LOC”), ii) LOC Ever at baseline, no LOC at follow-up (“resolved”), iii) no LOC Ever at baseline, LOC at follow-up (“emergent”), and iv) LOC at both baseline and follow-up (“persistent”). Simple models were examined adjusting only for years between baseline and follow-up and the respective baseline symptom (disordered eating, depressive or anxiety symptoms), and we also examined models that adjusted for these covariates as well as baseline age (y), sex (male versus female), race/ethnicity (non-Hispanic Caucasian versus other), and BMI change between baseline and follow-up. As recommended (Cohen, 1990; Saville, 1990), two-tailed, least squares difference tests were used to follow up on all pairwise comparisons between groups.

A logistic regression was conducted to assess predictors of the dependent variable of follow-up partial or full-syndrome BED onset. The independent variables included LOC Ever (presence versus absence), disordered eating attitudes, depressive and anxiety symptoms as predictors. The sets of covariates considered were years between baseline and follow-up alone, plus years between baseline and follow-up, baseline age (y), sex (male versus female), race/ethnicity (non-Hispanic Caucasian versus other), and BMI change between baseline and follow-up.

Results

One-hundred-ninety-five children (6–13y) were seen for a baseline visit. Baseline data for 162 participants have been reported elsewhere (Tanofsky-Kraff, et al., 2005; Tanofsky-Kraff, et al., 2004). Of the 195 study participants, 118 (60.5%) completed a follow-up assessment an average of 4.7 (SD, 1.2, range: 2.6–7.1) years later. Compared to those who did not return for a follow-up assessment, youth who completed a follow-up visit were older at baseline ($M=10.38$, $SD=1.49$ versus $M=9.63$, $SD=1.63y$, $p=.01$), but did not significantly differ in sex, race or baseline BMI, prevalence of LOC eating, disordered eating, depressive, or anxiety symptoms. As described above, data were imputed for the 77 children who did not return for a follow-up assessment, and combined results from the imputed data are presented.

At baseline, no child met full or partial-syndrome criteria for a DSM-IV-TR eating disorder. Forty-six children (23.6%) reported having experienced LOC eating at least once in their lifetime. Of these participants, approximately half ($n=22$, 47.8%) experienced at least 1 recent LOC episode in the month prior to assessment. In terms of the types and number of episodes in the past month, nine children reported only objective binge episodes (eight reported 1 episode and one reported 4 episodes); eleven reported only subjective binge episodes (four children reported 1 episode, five reported 2 episodes, one reported 3 and one child reported 4 episodes); two youth reported 1 objective binge episode and 1 subjective binge episode in the prior month. Consistent with previously published data (Tanofsky-Kraff, et al., 2005), youth with baseline LOC Ever had significantly higher baseline BMI, BMI-Z scores (Kuczmarski, et al., 2002), disordered eating attitudes, and depressive symptoms ($ps<.01$; Table 1) compared to those who never experienced LOC. Compared to those without baseline LOC, youth with baseline LOC Ever also were significantly younger at follow-up ($p=.04$) and continued to have significantly higher follow-up BMI, BMI-Z scores (Kuczmarski, et al., 2002), and EDE global scores and restraint and shape and weight concern subscale scores ($ps<.01$; Table 2).

Baseline LOC Eating as a Predictor of Follow-up Disordered Eating and Negative Affect

In simple models adjusting only for years between baseline and follow-up and the respective baseline subscale score, baseline LOC Ever was associated with greater follow-up EDE global, restraint, shape and weight concern ($ps<.02$). Even after adjusting for additional covariates (baseline age, sex, race/ethnicity, and BMI change) baseline LOC Ever predicted greater follow-up EDE global, restraint, shape and weight concern scores ($ps\leq.04$, Figure 1). Baseline LOC Ever significantly predicted follow-up anxiety in both the simple model adjusting for only baseline anxiety and years between baseline and follow-up ($p=.03$) and in a model adjusting for all covariates ($p=.05$). Baseline LOC Ever did not predict follow-up eating concern or depressive symptoms in either model ($ps>.48$).

Descriptive Information on Persistence of LOC Eating

The presence of LOC Ever at baseline was associated with more than a two-fold greater likelihood of reported LOC at follow-up ($OR=2.35$, 95% $CI=1.10-5.01$, $p=.03$). Among those reporting LOC Ever at baseline ($n=46$), 52.2% ($n=24$) reported persistent LOC eating in the month prior to follow-up assessment. In contrast, among those who did not report LOC Ever at baseline ($n=149$), only 30.9% ($n=46$) reported emergent LOC at follow-up; the majority of this group (69.1%; $n=103$) never reported LOC at either time. Of those endorsing LOC Ever at baseline, 47.8% ($n=22$) were resolved at follow-up. After adjusting for years between baseline and follow-up, baseline LOC Ever remained a significant predictor of follow-up LOC ($OR=2.67$, 95% $CI=1.15-6.22$, $p=.02$). When persistence was characterized in terms of number of LOC episodes in the past month, LOC episodes for the

whole sample increased from baseline ($M, SE=0.19, 0.04$ episodes per month) to follow-up ($M, SE=0.53, 0.16$ episodes per month, $p=.04$). Similarly, among the 24 youth who endorsed baseline LOC Ever and follow-up LOC, the average number of LOC episodes significantly increased by 1.33 ($SE=0.53$) episodes per month between baseline ($M, SE=0.79, 0.24$) and follow-up ($M, SE=2.12, 0.40, p=.01$). Significant increases in number of LOC episodes continued to be observed after accounting for years between baseline and follow-up, both in the whole sample and just among those with persistent LOC ($ps<.04$).

Persistence of LOC Eating as a Predictor of Disordered Eating and Negative Affect

In simple models (only covariates of years between baseline and follow-up and respective baseline subscale) and in models accounting for all covariates, there were significant overall effects for LOC persistence (never LOC, resolved, emergent, persistent) for all four follow-up EDE subscales (all main effects $ps<.004$; global score in model adjusting for all covariates, $F(3,143)=12.12, p<.001$, Figure 2A). In simple adjusted models, pairwise comparisons indicated that youth with persistent LOC had significantly higher scores than the never LOC group for EDE global score and all four EDE subscales ($ps<.006$), higher scores than the resolved group on EDE global score, eating concern and shape concern ($ps<.02$), and higher scores than the emergent LOC group on EDE global score ($p=.04$). Youth with emergent LOC had higher scores on EDE global and all four subscales compared to the never LOC group ($ps<.02$), and emergent LOC youth also had higher EDE eating concern than youth with resolved LOC ($p=.02$). These significant differences remained after accounting for all covariates ($ps<.05$).

In a simple adjusted model and a model accounting for all covariates, there was an overall main effect of LOC persistence on symptoms of depression (in model adjusting for all covariates, $F(3,262)=3.11, p=.03$, Figure 2B). In a simple adjusted model, pairwise comparisons indicated that persistent LOC youth had higher follow-up depressive symptoms than the never LOC group ($p=.03$) and the resolved LOC group ($p=.01$). The same significant differences were observed when accounting for all covariates ($ps<.05$). LOC persistence showed non-significant trends for association with follow-up symptoms of anxiety in both the simple adjusted model ($F(3,311)=2.44, p=.06$) and after adjusting for all covariates ($F(3, 300)=2.28, p=.08$).

Development of Partial or Full-Syndrome BED

At follow-up, 9 (4.5%) participants met partial or full-syndrome criteria for BED ($M, SE=5.7, 1.3$ LOC episodes in the month prior to assessment, range: 4–15). A subset (60%) of EDE/SPEEI interviews for these participants were taped and co-rated to examine inter-rater reliability for presence of the partial and full BED diagnosis. Cohen's kappa for the identification of partial or full syndrome BED was 1.00 ($p<.001$). In the model examining the development of partial or full-syndrome BED at follow-up, only baseline reports of LOC Ever served as a significant contributor ($OR=10.8, 95\% CI 1.3–88.1, p=.03$), after accounting for all other variables in the model including BMI change, baseline symptoms of depression and anxiety, and all four EDE subscales at baseline. Since the sample of participants who developed BED was small, yielding a very wide confidence interval for the predictive value of LOC Ever, we re-analyzed the model removing all non-significant variables except years in the study (sex, race, baseline age, depressive, anxiety, and disordered eating symptoms, as BMI growth). Baseline reports of LOC Ever continued to serve as a significant predictor of BED ($OR= 5.07, 95\% CI 1.1–24.5, p=.04$). This analysis suggested that youth who reported ever having experienced LOC at baseline were greater than five times more likely to develop partial or full-syndrome BED at follow-up.

Follow-up Exploratory Analyses

Secondary analyses were conducted to examine predictors of LOC onset at follow-up. To examine predictors of the dependent variable of follow-up LOC eating onset (presence versus absence), we conducted binary logistic regression analyses with only the subset of children who did not endorse baseline LOC Ever ($n=149$). The independent variables were baseline disordered eating attitudes (global, restraint, shape concern, or weight concern), depressive symptoms, or anxiety symptoms. The sets of covariates considered were years between baseline and follow-up alone, and then years between visits and baseline age (y), sex (male versus female), race/ethnicity (non-Hispanic Caucasian versus other), and BMI change between baseline and follow-up. In the subset of children who reported no LOC Ever at baseline, neither disordered eating attitudes, nor symptoms of depression or anxiety predicted follow-up emergent LOC eating in any model ($ps>.28$).

To further explore the nature of the significant relationships observed between persistence of LOC and increases in disordered eating and negative affect, we conducted a series of follow-up linear multiple regressions regressing the dependent variables of change scores (follow-up minus baseline) in disordered eating (EDE global score, restraint, eating, shape concern, and weight concern subscales), depressive symptoms, or anxiety symptoms on the independent variables of change score in LOC eating episodes in the past month. In both simple adjusted models accounting only for years between baseline and follow-up, and in models accounting for all covariates, there was no significant association between change in number of LOC episodes and any of the dependent variables (all $ps>.50$).

Discussion

Using the Eating Disorder Examination, a well-accepted interview assessment method for identification of eating disorders, we found, among a sample of non-treatment seeking children age 6 to 13 years who were reassessed approximately 5 years later, that those who reported having ever experienced loss of control over their eating at baseline were significantly more likely to develop partial or full-syndrome binge eating disorder than children who had never experienced loss of control. The presence of reported loss of control eating at baseline also predicted increases in disordered eating attitudes and symptoms of anxiety at follow-up, but not follow-up depressive symptoms. However, children with reported loss of control eating at both baseline and follow-up experienced the greatest disordered eating within all domains and the greatest increases in symptoms of depression.

The finding that childhood LOC is a significant indicator for the development of persistent or worsened disordered eating attitudes and anxiety symptoms almost five years later is novel and may have important clinical implications. LOC eating has been consistently associated in cross-sectional studies with increased psychological distress (Tanofsky-Kraff, 2008) and found to be predictive of excess body weight gain in young children (Tanofsky-Kraff, Yanovski, et al., 2009). Our data suggest that infrequent reports of LOC may also be a precursor for increased disordered eating and the development of partial or full syndrome BED. Some preliminary studies indicate that reducing binge and LOC eating may be effective for both weight loss (Jones, et al., 2008) and obesity prevention (Tanofsky-Kraff, Wilfley, et al., 2009) in youth. It remains to be determined if such interventions may simultaneously prevent the development of eating disorders.

Almost 5% of the sample developed partial or full-syndrome BED at follow-up. Although there are limited data on the prevalence of BED in early adolescence, rates for LOC eating, typically defined as one episode in the month prior to assessment or even less frequent LOC episodes, range from approximately 2% to 40%, with higher estimates among weight loss treatment-seeking (versus community) samples and higher prevalence among adolescents

(versus children) and in studies using questionnaires rather than diagnostic interviews (Tanofsky-Kraff, 2008). In adult samples, the rate of DSM-IV-TR BED is approximately 3% in the general population (Hudson, Hiripi, Pope, & Kessler, 2007), with higher estimates among individuals seeking weight-loss treatment (de Zwaan, 2001). Although our sample was not seeking weight loss treatment, it differed from other pediatric community samples because it was enriched for overweight youth, which likely contributed to the relatively higher rate of partial and full-syndrome BED found at follow-up.

It is notable that only LOC, as opposed to weight, shape and eating concerns, or negative affect, was a clinically relevant behavior for the development of BED. This finding is in contrast to studies that examined the development of any eating disorder (i.e., not exclusively BED) during adolescence, where body shape and weight concerns were important predictors for later eating disorders (Killen, et al., 1996; Killen, et al., 1994; McKnight Investigators, 2003). Although the aforementioned studies included different covariates than those included in the present analyses, our findings generally did not differ whether we conducted simple analyses adjusting only for years to follow-up and the respective baseline variable or analyses including all covariates. It should be noted that these investigations did not examine baseline LOC eating as a predictor. Our results may be related to the developmental differences in emotional and cognitive constructs that are found during middle childhood versus adolescence. With regard to symptoms of depression and anxiety, it is possible that at baseline the children in our sample had yet to develop symptoms of mood problems. Given that our sample consisted of non-treatment-seeking children without a current eating disorder, depressive symptoms and trait anxiety may not have been a common component of their baseline psychological status. Indeed, the baseline scores reported by the sample were well below clinically significant cut-offs (Kazdin, Colbus, & Rodgers, 1986; Lobovits & Handal, 1985; Spielberger, 1983). LOC eating may conceivably be an early behavioral marker preceding, or even possibly masking, disordered eating attitudes and negative affect. In a multi-site study of children and adolescents, the experience of “numbing” was highly correlated with LOC eating episodes (Tanofsky-Kraff, et al., 2007). These data point to another possibility; namely, that children reporting LOC may be less aware of their emotional experiences than of their actual behaviors. Both suppositions potentially may support “escape theory,” which views binge eating as a motivated attempt to escape aversive self-awareness or emotional distress (Heatherton & Baumeister, 1991). Regardless, during middle childhood, reported eating patterns may thus be more salient in predicting later exacerbated disordered eating in adolescence than reports of either body weight or mood-related distress.

Children who reported having engaged in LOC eating even one time at baseline were at greater risk for the development of increased shape and weight concerns at follow-up than those who did not report such episodes. Notably, over-concern with body shape and weight is a key feature across the eating disorder diagnoses (Wilfley, Schwartz, Spurrell, & Fairburn, 2000) and is associated with greater impairment when present in individuals with BED (Grilo, Masheb, & White, 2009). However, those youth who reported LOC at both baseline and follow-up were most at risk for exacerbated disordered eating and increases in depressive symptoms, with the average unadjusted Children’s Depression Inventory total score nearing the clinical concern cutoff of 12 (Kazdin, et al., 1986; Lobovits & Handal, 1985). These findings persisted even after accounting for the contribution of BMI growth, suggesting that the increased distress predicted by LOC cannot be attributed solely to excess weight gain. Coupled with data indicating that binge and LOC eating have been shown to predict excess weight and fat gain in youth (Field, et al., 2003; Stice, et al., 1999; Tanofsky-Kraff, et al., 2006; Tanofsky-Kraff, Yanovski, et al., 2009), our findings support proposals that obesity and eating disorder interventions should be coordinated (Neumark-Sztainer, et al., 2009; Yanovski, 2003).

Our findings from the present and prior (Tanofsky-Kraff, Yanovski, et al., 2009) studies suggest that in young children, the report of the infrequent objective or subjective binge eating episodes that together comprise LOC eating is predictive of untoward results. Even quite rare LOC eating episodes appear to be associated with later adverse psychological outcomes. Although this may be the result of an overly conservative estimate of what constitutes a “large” amount of food in growing children, the experience of LOC eating may identify youth who are consuming more than they want to eat. It is possible that individuals with reported LOC have some disturbance in satiety signaling or reward activation pathways (Adam & Epel, 2007; Davis, et al., 2008). The relationship between LOC and eating in the absence of hunger has been documented (Tanofsky-Kraff, Ranzenhofer, et al., 2008). Further, data indicate that youth with LOC tend to consume highly palatable dessert and snack-type foods (Hilbert, Tuschen-Caffier, & Czaja, 2010; Tanofsky-Kraff, McDuffie, et al., 2009; Theim, et al., 2007); thus, LOC behaviors may be related to variations in opioid (reward) pathways, stemming from genetic polymorphisms in opioid/dopamine receptor genes, as suggested in studies of adults with and without BED (Davis, et al., 2008). Though little is known about the neural circuitry of LOC eating in children, there are neuroimaging data in emotional eaters (Bohon, Stice, & Spoor, 2009) and individuals with BED (Schienle, Schafer, Hermann, & Vaitl, 2009) to suggest that the processing of food-related stimuli may be fundamentally different in persons with and without aberrant eating patterns. Longitudinal data are needed to explore these potential neural pathways underlying LOC eating and the development of BED.

Strengths of this investigation include the prospective design, the use of a structured interview, and measured heights and weights. Limitations include the fact that children who reported LOC eating Ever (i.e., before the last month) did not supply information on size of the meal consumed, the use of questionnaires to assess depressive and anxiety symptoms and the relatively small sample at follow-up. Moreover, there was only one EDE follow-up assessment, which precluded an examination of the average duration of LOC eating during the course of the follow-up period. Due to this limitation, we are also unable to explore why approximately 48% of those with baseline LOC eating no longer reported such behaviors at follow-up. In addition, despite the fact that children were not seeking treatment, they were not recruited in a population-based fashion, were enriched for overweight, and had to travel to the National Institutes of Health (NIH) Clinical Research Center to participate. These factors likely limit the generalizability of our findings to young children willing to participate in research that involves both physical and psychological measurements. Although there are no available data in similar samples involving the same assessments, our response rate is lower than that of prospective studies of eating disorder development in adolescents that made use of interviews in school samples (Lewinsohn, Striegel-Moore, et al., 2000; Stice, Marti, Shaw, & Jaconis, 2009). Indeed, our response rate of approximately 7% is likely reflective of the challenges involved in studying young children using various physical assessments, especially those that are often perceived as invasive (e.g., phlebotomy). Nevertheless, families were recruited for metabolic studies, understood that they would not receive treatment, and neither parents nor children had prior knowledge that they would be asked about disordered eating behaviors and attitudes. However, because of their willingness to participate in metabolic studies at the NIH, study children may have differed from the general population.

In conclusion, among a non-treatment sample of overweight and non-overweight middle-childhood youth, those who reported having engaged in LOC eating are more likely to develop worsening disordered eating. Moreover, those whose LOC eating persists over time appear to be at higher risk for increases in symptoms of depression. Future investigation is necessary to determine if interventions aimed at reducing LOC eating during middle-

childhood are efficacious in the prevention of both eating disorders and excessive weight gain.

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References

- Adam TC, Epel ES. Stress, eating and the reward system. *Physiol Behav* 2007;91(4):449–458. [PubMed: 17543357]
- Allison, PD. Missing data. In: Millsap, RE.; Maydeu-Olivares, A., editors. *The SAGE Handbook of Quantitative Methods in Psychology*. Thousand Oaks, CA: Sage Publications; 2009. p. 72-89.
- APA. *Diagnostic and Statistical Manual of Mental Disorders DSM-IV-TR*. Washington, DC: Authors; 2000.
- Arnow B, Kenardy J, Agras WS. Binge eating among the obese: a descriptive study. *J Behav Med* 1992;15(2):155–170. [PubMed: 1583679]
- Barker ET, Williams RL, Galambos NL. Daily spillover to and from binge eating in first-year university females. *Eating Disorders* 2006;14:229–242. [PubMed: 16807216]
- Behrens JT. Principles and procedures of exploratory data analysis. *Psychol Methods* 1997;2:131–160.
- Bohon C, Stice E, Spoor S. Female emotional eaters show abnormalities in consummatory and anticipatory food reward: a functional magnetic resonance imaging study. *Int J Eat Disord* 2009;42(3):210–221. [PubMed: 19040270]
- Bryant-Waugh RJ, Cooper PJ, Taylor CL, Lask BD. The use of the eating disorder examination with children: a pilot study. *Int J Eat Disord* 1996;19(4):391–397. [PubMed: 8859397]
- Christie, D.; Watkins, B.; Lask, B. Assessment. In: Lask, B.; Bryant-Waugh, RJ., editors. *Anorexia nervosa and related eating disorders in childhood and adolescence*. 2. East Essex, UK: Psychology Press; 2000. p. 105-125.
- Cohen J. Things I have learned (so far). *Am Psychol* 1990;45:1304–1312.
- Crow SJ, Agras WS, Halmi K, Mitchell JE, Kraemer HC. Full syndromal versus subthreshold anorexia nervosa, bulimia nervosa, and binge eating disorder: a multicenter study. *Int J Eat Disord* 2002;32(3):309–318. [PubMed: 12210645]
- Davis C, Levitan RD, Kaplan AS, Carter J, Reid C, Curtis C, et al. Reward sensitivity and the D2 dopamine receptor gene: A case-control study of binge eating disorder. *Prog Neuropsychopharmacol Biol Psychiatry* 2008;32(3):620–628. [PubMed: 18262320]
- de Zwaan M. Binge eating disorder and obesity. *Int J Obes Relat Metab Disord* 2001;25(Suppl 1):S51–55. [PubMed: 11466589]
- Fairburn, CG.; Cooper, Z. *The Eating Disorder Examination*. In: Fairburn, CG.; Wilson, GT., editors. *Binge eating, nature, assessment and treatment*. 12. New York: Guilford; 1993. p. 317-360.
- Field AE, Austin SB, Taylor CB, Malspeis S, Rosner B, Rockett HR, et al. Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics* 2003;112(4):900–906. [PubMed: 14523184]
- Grilo CM, Masheb RM, White MA. Significance of Overvaluation of Shape/Weight in Binge-eating Disorder: Comparative Study With Overweight and Bulimia Nervosa. *Obesity* (Silver Spring). 2009
- Heatherton TF, Baumeister RF. Binge eating as escape from self-awareness. *Psychological Bulletin* 1991;110:86–108. [PubMed: 1891520]
- Hilbert A, Tuschen-Caffier B, Czaja J. Eating behavior and familial interactions of children with loss of control eating: a laboratory test meal study. *American Journal of Clinical Nutrition* 2010;91(3): 510–518. [PubMed: 20089727]

- Hollingshead, A. Four factor index of social status. Yale University; New Haven: 1975.
- Hudson JI, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry* 2007;61(3):348–358. [PubMed: 16815322]
- Johnson JG, Spitzer RL, Williams JB. Health problems, impairment and illnesses associated with bulimia nervosa and binge eating disorder among primary care and obstetric gynaecology patients. *Psychological Medicine* 2001;31(8):1455–1466. [PubMed: 11722160]
- Jones M, Luce KH, Osborne MI, Taylor K, Cuning D, Doyle AC, et al. Randomized, controlled trial of an internet-facilitated intervention for reducing binge eating and overweight in adolescents. *Pediatrics* 2008;121(3):453–462. [PubMed: 18310192]
- Kazdin AE, Colbus D, Rodgers A. Assessment of depression and diagnosis of depressive disorder among psychiatrically disturbed children. *J Abnorm Child Psychol* 1986;14(4):499–515. [PubMed: 3782622]
- Killen JD, Taylor CB, Hayward C, Haydel KF, Wilson DM, Hammer L, et al. Weight concerns influence the development of eating disorders: a 4-year prospective study. *J Consult Clin Psychol* 1996;64(5):936–940. [PubMed: 8916622]
- Killen JD, Taylor CB, Hayward C, Wilson DM, Haydel KF, Hammer LD, et al. Pursuit of thinness and onset of eating disorder symptoms in a community sample of adolescent girls: a three-year prospective analysis. *Int J Eat Disord* 1994;16(3):227–238. [PubMed: 7833956]
- Kovacs M. The children's depression inventory. Unpublished manuscript. 1982
- Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, et al. 2000 CDC Growth Charts for the United States: methods and development. *Vital Health Stat* 2002;11(246):1–190.
- Leon GR, Fulkerson JA, Perry CL, Early-Zald MB. Prospective analysis of personality and behavioral vulnerabilities and gender influences in the later development of disordered eating. *J Abnorm Psychol* 1995;104(1):140–149. [PubMed: 7897036]
- Lewinsohn PM, Rohde P, Seeley JR, Klein DN, Gotlib IH. Natural course of adolescent major depressive disorder in a community sample: predictors of recurrence in young adults. *American Journal of Psychiatry* 2000;157(10):1584–1591. [PubMed: 11007711]
- Lewinsohn PM, Striegel-Moore RH, Seeley JR. Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry* 2000;39(10):1284–1292. [PubMed: 11026183]
- Lobovits DA, Handal PJ. Childhood depression: prevalence using DSM-III criteria and validity of parent and child depression scales. *J Pediatr Psychol* 1985;10(1):45–54. [PubMed: 3989635]
- Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. *Arch Dis Child* 1969;44:291–303. [PubMed: 5785179]
- Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. *Arch Dis Child* 1970;45:13–23. [PubMed: 5440182]
- McKnight Investigators, T. Risk factors for the onset of eating disorders in adolescent girls: results of the McKnight longitudinal risk factor study. *Am J Psychiatry* 2003;160(2):248–254. [PubMed: 12562570]
- Neumark-Sztainer D, Wall M, Story M, Sherwood NE. Five-year longitudinal predictive factors for disordered eating in a population-based sample of overweight adolescents: implications for prevention and treatment. *Int J Eat Disord* 2009;42(7):664–672. [PubMed: 19642214]
- Presnell K, Stice E, Seidel A, Madeley MC. Depression and eating pathology: prospective reciprocal relations in adolescents. *Clin Psychol Psychother* 2009;16(4):357–365. [PubMed: 19569042]
- Rizvi SL, Peterson CB, Crow SJ, Agras WS. Test-retest reliability of the eating disorder examination. *Int J Eat Disord* 2000;28(3):311–316. [PubMed: 10942917]
- Saville DJ. Multiple comparison procedures: The practical solution. *Am Stat* 1990;44:174–180.
- Schafer JL, Graham JW. Missing data: our view of the state of the art. *Psychol Methods* 2002;7(2):147–177. [PubMed: 12090408]
- Schienze A, Schafer A, Hermann A, Vaitl D. Binge-eating disorder: reward sensitivity and brain activation to images of food. *Biol Psychiatry* 2009;65(8):654–661. [PubMed: 18996508]

- Shomaker LS, Tanofsky-Kraff M, Elliott C, Wolkoff LE, Columbo KM, Ranzenhofer LM, et al. Salience of loss of control for pediatric binge episodes: Does size really matter? *Int J Eat Disord*. In press.
- Spielberger, G.; Lushene, Vagg; Jacobs. *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologist Press; 1983.
- Stice E, Cameron RP, Killen JD, Hayward C, Taylor CB. Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *Journal of Consulting and Clinical Psychology* 1999;67:967–974. [PubMed: 10596518]
- Stice E, Hayward C, Cameron RP, Killen JD, Taylor CB. Body-image and eating disturbances predict onset of depression among female adolescents: a longitudinal study. *Journal of Abnormal Psychology* 2000;109(3):438–444. [PubMed: 11016113]
- Stice E, Killen JD, Hayward C, Taylor CB. Age of onset for binge eating and purging during late adolescence: A 4-year survival analysis. *Journal of Abnormal Psychology* 1998;107:671–675. [PubMed: 9830254]
- Stice E, Marti CN, Shaw H, Jaconis M. An 8-year longitudinal study of the natural history of threshold, subthreshold, and partial eating disorders from a community sample of adolescents. *J Abnorm Psychol* 2009;118(3):587–597. [PubMed: 19685955]
- Stice E, Presnell K, Bearman SK. Relation of early menarche to depression, eating disorders, substance abuse, and comorbid psychopathology among adolescent girls. *Developmental Psychology* 2001;37(5):608–619. [PubMed: 11552757]
- Striegel-Moore RH, Dohm FA, Solomon EE, Fairburn CG, Pike KM, Wilfley DE. Subthreshold binge eating disorder. *Int J Eat Disord* 2000;27(3):270–278. [PubMed: 10694712]
- Tanofsky-Kraff, M. Binge eating among children and adolescents. In: Jelalian, E.; Steele, R., editors. *Handbook of Child and Adolescent Obesity*. Springer; 2008. p. 41–57.
- Tanofsky-Kraff M, Cohen ML, Yanovski SZ, Cox C, Theim KR, Keil M, et al. A prospective study of psychological predictors of body fat gain among children at high risk for adult obesity. *Pediatrics* 2006;117(4):1203–1209. [PubMed: 16585316]
- Tanofsky-Kraff M, Faden D, Yanovski SZ, Wilfley DE, Yanovski JA. The perceived onset of dieting and loss of control eating behaviors in overweight children. *International Journal of Eating Disorders* 2005;38(2):112–122. [PubMed: 16134103]
- Tanofsky-Kraff M, Goossens L, Eddy KT, Ringham R, Goldschmidt A, Yanovski SZ, et al. A multisite investigation of binge eating behaviors in children and adolescents. *Journal of Consulting and Clinical Psychology* 2007;75(6):901–913. [PubMed: 18085907]
- Tanofsky-Kraff M, Marcus MD, Yanovski SZ, Yanovski JA. Loss of control eating disorder in children age 12 years and younger: proposed research criteria. *Eat Behav* 2008;9(3):360–365. [PubMed: 18549996]
- Tanofsky-Kraff M, McDuffie JR, Yanovski SZ, Kozlosky M, Schvey NA, Shomaker LB, et al. Laboratory assessment of the food intake of children and adolescents with loss of control eating. *American Journal of Clinical Nutrition* 2009;89(3):738–745. [PubMed: 19144730]
- Tanofsky-Kraff M, Ranzenhofer LM, Yanovski SZ, Schvey NA, Faith M, Gustafson J, et al. Psychometric properties of a new questionnaire to assess eating in the absence of hunger in children and adolescents. *Appetite* 2008;51(1):148–155. [PubMed: 18342988]
- Tanofsky-Kraff M, Wilfley DE, Young JF, Mufson L, Yanovski SZ, Glasofer DR, et al. A pilot study of interpersonal psychotherapy for preventing excess weight gain in adolescent girls at-risk for obesity. *International Journal of Eating Disorders*. 2009
- Tanofsky-Kraff M, Yanovski SZ, Schvey NA, Olsen CH, Gustafson J, Yanovski JA. A prospective study of loss of control eating for body weight gain in children at high risk for adult obesity. *International Journal of Eating Disorders* 2009;42(1):26–30. [PubMed: 18720473]
- Tanofsky-Kraff M, Yanovski SZ, Wilfley DE, Marmarosh C, Morgan CM, Yanovski JA. Eating disordered behaviors, body fat, and psychopathology in overweight and normal weight children. *Journal of Consulting and Clinical Psychology* 2004;72:53–61. [PubMed: 14756614]
- Theim KR, Tanofsky-Kraff M, Salaita CG, Haynos AF, Mirch MC, Ranzenhofer LM, et al. Children's descriptions of the foods consumed during loss of control eating episodes. *Eating Behaviors* 2007;8(2):258–265. [PubMed: 17336796]

- Wilfley DE, Schwartz MB, Spurrell EB, Fairburn CG. Using the eating disorder examination to identify the specific psychopathology of binge eating disorder. *Int J Eat Disord* 2000;27(3):259–269. [PubMed: 10694711]
- Wilfley DE, Wilson GT, Agras WS. The clinical significance of binge eating disorder. *International Journal of Eating Disorders* 2003;34(Suppl):S96–106. [PubMed: 12900990]
- Yanovski SZ. Binge eating disorder and obesity in 2003: could treating an eating disorder have a positive effect on the obesity epidemic? *Int J Eat Disord* 2003;34(Suppl):S117–120. [PubMed: 12900992]

Figure 1.

Baseline loss of control eating and follow-up disordered eating attitudes. Experiencing loss of control over eating ever at baseline predicted increases on the Eating Disorder Examination Restraint, Shape Concern and Weight Concern subscales at follow-up. Analysis adjusted for baseline age, sex, race, years in study, body mass index growth, respective baseline disordered eating subscale. $N=195$, $p \leq .04$.

Figure 2.

A: Persistent loss of control eating and follow-up disordered eating. Eating Disorder Examination Global score at follow-up is shown. Children who reported ever experiencing loss of control at baseline and loss of control at follow-up (persistent) had significantly greater disordered eating at follow-up compared to youth who never reported loss of control (never LOC), those who reported loss of control at baseline only (resolved), and those who reported loss of control at follow-up (emergent). Analysis adjusted for baseline age, sex, race, years in study, body mass index growth, baseline global disordered eating. $N=195$. Main effect $p<.001$.

B: Persistent loss of control eating and follow-up depressive symptoms. Children who reported ever experiencing loss of control at baseline and loss of control at follow-up (persistent) had significantly greater total scores on the Children's Depression Inventory at follow-up compared to youth who never reported loss of control (never LOC) and those who reported loss of control at baseline only (resolved). Analysis adjusted for baseline age, sex, race, years in study, body mass index growth, baseline depressive symptoms. $N=195$. Main effect $p=.03$.

Table 1

Participant Characteristics at Baseline

	No LOC at Baseline <i>n</i> = 149	LOC at Baseline <i>n</i> = 46	<i>p</i> -value
	M (SEM)	M (SEM)	
Age (y)	10.2 (.02)	10.3 (.06)	.61
Female sex (percent)	52.3%	73.9%	.001
Race (percent)	62.4% Non-Hispanic White 37.6% Other	56.5% Non-Hispanic White 43.5% Other	.48
BMI (kg/m ²)	21.8 (.31)	25.4 (1.1)	.003
BMI z score [‡]	.95 (.12)	1.6 (.22)	.008
Child EDE [*] Subscales			
Restraint	.27 (.002)	.55 (.008)	.002
Eating Concern	.05 (.001)	.27 (.003)	<.001
Shape Concern	.35 (.002)	.74 (.01)	<.001
Weight Concern	.56 (.004)	1.17 (.03)	<.001
Symptoms of Depression	6.29 (.46)	8.57 (.99)	<.001
Symptoms of Anxiety	34.5 (.68)	36.3 (1.34)	.205

Note. Values are means (SEM) unless otherwise indicated; LOC at baseline, reports of ever experiencing a loss of control over eating; BMI, Body Mass Index (kg/m²) by measured height and weight;

[‡] BMI z-score, BMI accounting for age and sex according to Centers for Disease Control and Prevention (Kuczmarski, et al., 2002);

^{*} EDE, Eating Disorder Examination at follow-up; subscale scores range from 0–6.

Table 2

Participant Characteristics at Follow-up.

	No LOC at Baseline <i>n</i> = 149	LOC at Baseline <i>n</i> = 46	<i>p</i> -value
	M (SEM)	M (SEM)	
Age (y)	15.2 (.17)	14.4 (.33)	.03
BMI (kg/m ²)	26.8 (.65)	29.9 (1.5)	.004
BMI z-score [‡]	1.4 (.97)	2.0 (.74)	.02
BMI change per year	1.1 (.08)	1.3 (.31)	.36
EDE* Subscales			
Restraint	.20 (.04)	.48 (.08)	<.001
Eating Concern	.09 (.03)	.18 (.05)	.06
Shape Concern	.56 (.08)	1.1 (.18)	.01
Weight Concern	.57 (.07)	1.2 (.17)	<.001
Symptoms of Depression	4.2 (.41)	5.1 (.84)	.29
Symptoms of Anxiety	29.9 (.57)	31.6 (1.27)	.22

Note. Values are means (SEM) unless otherwise indicated; LOC at baseline, reports of ever experiencing a loss of control over eating; BMI, Body Mass Index (kg/m²) by measured height and weight;

[‡] BMI z-score, BMI accounting for age and sex according to Centers for Disease Control and Prevention (Kuczmarski, et al., 2002);

* EDE, Eating Disorder Examination at follow-up; subscale scores range from 0–6.