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Eating-Disordered Behaviors, Body Fat, and Psychopathology in Overweight and Normal-Weight Children

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Abstract

This study examined eating-disordered pathology in relation to psychopathology and adiposity in 162 non-treatment-seeking overweight (OW) and normal weight (NW) children, ages 6–13 years. Participants experienced objective or subjective binge eating (S/OBE; loss-of-control eating), objective over-eating (OO), or no episodes (NE). OW children experienced significantly higher eating-disordered cognitions and behaviors than NW children and more behavior problems than NW children: 9.3% endorsed S/OBEs, 20.4% reported OOs, and 70.4% reported NEs. OW children reported S/OBEs more frequently than did NW children ($p = .01$), but similar percentages endorsed OOs. S/OBE children experienced greater eating-disordered cognitions (ps from $< .05$ to $< .01$) and had higher body fat ($p < .05$) than OOs or NEs. OOs are common in childhood, but S/OBEs are more prevalent in OW children and associated with increased adiposity and eating-disordered cognitions.

The high prevalence of binge eating behaviors and their associations with adiposity and psychopathology are well-established phenomena in adolescents. Questionnaire surveys of nontreatment 13- to 19-year-olds suggest that as many as 45% of girls and 16% of boys report binge eating (Greenfeld, Quinlan, Harding, Glass, & Bliss, 1987). A school-based study of 7th

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through 12th graders found that overweight participants self-report binge eating more frequently than do their normal weight peers (Neumark-Sztainer et al., 1997). Among non-treatment seeking adolescents, those who report binge eating are mote, likely to manifest eating-related cognitions and depressive symptoms, which are related to the severity of binge eating (Johnson, Grieve, Adams, & Sandy, 1999; Ledoux, Choquet, & Manfredi, 1993; Neumark-Sztainer & Hannan, 2000; Steiger, Puentes-Neuman, & Leung, 1991). Studies of school and community samples that have made use of interview methodology have found that few adolescents meet criteria for full-syndrome eating disorders (between 0.04% and 2.80%; Lewinsohn, Striegel-Moore, & Seeley, 2000; Stice, Presnell, & Bearman, 2001), and only 4% have reported objective binge eating (Stice, Killen, Hayward, & Taylor, 1998). Although obesity appears to be a risk factor for binge eating onset in adulthood (Vogeltanz-Holm et al., 2000), studies have not consistently found that body mass index (BMI) predicts binge eating behaviors in adolescents (Stice, 1998; Stice & Agras, 1998; Stice, Presnell, & Spangler, 2002).

Relatively little is known about when eating pathology first emerges. Several investigators maintain that eating disturbances begin in childhood or preadolescence (e.g., Smolak & Levine, 1994), whereas others suggest that eating pathology emerges in middle adolescence (Lewinsohn et al., 2000; Stice et al., 1998). To date, the prevalence and nature of binge eating in middle childhood (6–12 years) remain relatively unexplored. Only two relevant studies were retrieved by a literature search. Among a nonclinical sample of girls aged 7–13 years assessed by the Children's Eating Attitude Test Questionnaire, 10.4% reported binge eating behaviors (Maloney, McGuirc, Daniels, & Specker, 1989). Surprisingly, 3rd-grade girls were more likely to report binge episodes than 6th-grade girls (16.5% vs. 6.8%). The authors posited that the younger children may not have understood what was meant by binge eating because of the wording of the questionnaire. A second study examining non-treatment seeking overweight children (6–10 years), that used the Questionnaire on Eating and Weight Patterns found that 5.3% of their sample met criteria for binge eating disorder (BED) and that those experiencing loss of control while eating were heavier and had greater body fat than children not experiencing loss of control (Morgan et al., 2002). Furthermore, children reporting loss of control had higher anxiety, more depressive symptoms, and increased body dissatisfaction. However, the criteria used by children to determine the presence of a binge were not fully specified in the questionnaire and were based on the child's subjective experiences. Thus, there appear to be few reliable data describing binge eating and its psychological correlates in preadolescent children.

Despite the limited research on binge eating in middle childhood, child obesity (without specifying binge eating behaviors) has been correlated with many of the same constructs as those identified from studies of adults and adolescents who endorse binge eating. In girls ages 9–10 years, eating-related cognitions including "drive for thinness," "criticism about weight," and "dissatisfaction with physical appearance" were positively associated with adiposity (Striegel-Moore, Schreiber, Pike, Wilfley, & Rodin, 1995). A large-scale questionnaire study of elementary school children revealed that obese children, particularly girls, were significantly more likely to report concerns about their weight and to report restraining their eating than were average-weight children (Vander Wal & Thelen, 2000). In an interview-based study, overweight girls had higher concerns about shape, weight and eating, and restraint than normal weight peers (Burrows & Cooper, 2002). Increased weight has been positively associated with overweight concerns and depressive symptoms in a school sample of third-grade girls but not boys (Erickson, Robinson, Haydel, & Killen, 2000). Behavior problems as measured by the Child Behavior Checklist (Achenbach & Elderbrock, 1991) have also been positively related to weight in nonclinical samples of children (Stradmeijer, Bosch, Koops, & Seidell, 2000; Tershakovec, Weller, & Gallagher. 1994).

Restraint theory proposes that binge eating occurs in response to the application of excessive dietary restraint (Herman & Polivy, 1975). Indeed, prospective research with adolescents has suggested that dieting is a risk factor for binge eating (Stice et al., 2002). Overweight individuals may be more likely to engage in dieting behaviors because they are attempting to attain society's thin ideal (Stice & Agras, 1998; Stice et al., 2002), thus putting themselves at greater risk for binge eating. Retrospective research investigating dieting vulnerability factors has supported the premise that excess adiposity in childhood is a risk for the development of eating-disordered pathology (Fairburm, Cooper, Doll, & Welch, 1999; Fairburn et al., 1998; Fairburn, Welch, Doll, Davies, & O'Connor, 1997). However, there is also prospective evidence that binge eating in adolescence increases the risk for subsequent obesity (Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice et al., 2002), supporting a physiological hypothesis that the excess energy intake that ensues from binge eating may result in weight gain. Although it is unclear whether excess weight or eating-disordered behaviors occur first or whether overweight and eating-related disturbance emerge contemporaneously, examining overweight individuals in middle childhood (6–12 years), prior to the development of partial- or full-syndrome eating disorders, should increase our understanding of the emergence and early risk factors that may serve as points of intervention for prevention efforts.

A limitation of prior studies examining young children is the lack of interview methodology to assess eating-disordered behaviors and cognitions. Therefore, we used an interview method to assess the prevalence of binge eating behaviors and eating-disordered cognitions in a cohort of non-treatment seeking overweight and normal weight girls and boys. In addition, we assessed body composition both by BMI, with height and weight measured by a research team member, and by dual energy X-ray absorptiometry scan (DXA), a highly accurate method for measuring body fat mass that, unlike BMI, can exclude muscle mass and other organ weight. Given research suggesting that overweight may be an important childhood risk factor for eating disorders (Fairburn et al., 1997, 1998, 1999) and that eating pathology in adolescence has been demonstrated to predict subsequent obesity (Stice et al., 1999; Stice et al., 2002), we hypothesized that overweight children, compared with nonoverweight children, would experience greater eating-disordered concerns and behaviors, more depressive symptoms, greater anxiety, and more frequent behavioral problems. Moreover, we posited that children engaging in binge eating behaviors would have higher scores on eating-disordered cognitions and general psychopathology and would have greater body adiposity compared with children who did not engage in eating-disordered behaviors.

Method

Participants

One hundred sixty-two children ages 6.1–13.8 years ($M \pm SD$; 10.0 ± 1.8 years) taking part in ongoing metabolic studies al the National Institutes of Health agreed to participate. Most of the children were African American (38.3%, $n = 62$) or Caucasian (54.9%, $n = 89$), and 1.2% ($n = 2$) were Asian American, 4.3% ($n = 7$) were Hispanic, and a small number of children were identified as other (2.1%, $n = 2$). The sample had a mean BMI (kg/m^3) of 22.1 ± 7.4 (range: 12.9–46.3), and DXA fat mass (kg) ranged from 2.0 to 60.0 (17.47 ± 14.54 kg). The 92 (56.8%) girls and 70 (43.2%) boys were of a similar socioeconomic background, with a mean Hollingshead's Index score of 2.9 ± 1.1 (range: 1–5), and most had either no evidence for puberty or only early signs of puberty (girl's breast Tanner stage: 2.5 ± 1.0, range: 1–5; Boy's testicular volume [cc]: 2.6 ± 2.3, range: 1–15).

Participants were recruited through two waves of notices mailed to 1st-through 5th-grade children in the Montgomery County and Prince George's County, Maryland school districts and by two mailings to local family physicians and pediatricians. Mailings to families requested the participation of children willing to undergo phlebotomy (blood draw) and X-rays for studies

investigating hormones in children. Mailings to physicians requested children willing to participate in similar studies and also specified that no treatment would be offered. Approximately 7% of families responded to each of the school mailings, and participants recruited directly from these mailings constituted 88% of all participants studied. None of the children accepted into the study were undergoing weight loss treatment, and all were aware that they would not receive treatment as part of the study protocol or after participation. Participants were medication free for at least 2 weeks prior to the study, and none had significant medical disease. Each child had normal hepatic, renal, and thyroid function. Children provided written assent and parents gave written consent for participation in the protocol. This study was approved by the National Institute of Child Health and Human Development Institutional Review Board. Some of the psychological and body composition data from 12 (7.4%) of the children in this sample have been included in a previous report (Morgan et al., 2002).

Procedure and Assessment

Participants were seen at the National Institutes of Health Warren Grant Magnuson Clinical Center (Bethesda, MD). All children underwent a medical history and a physical examination conducted by a pediatric endocrinologist or a trained pediatric nurse practitioner. Each child subsequently participated in measures of eating-disorder pathology, symptoms of depression, trait anxiety, and body composition. A parent also completed a measure of behavior problems for each child.

Assessment of Eating Disorder Pathology

The Eating Disorder Examination version 12OD/C.2 (EDE; Fairburn & Cooper, 1993) adapted for children (ChEDE; Bryant-Waugh, Cooper, Taylor, & Lask, 1996) is an investigator-based interview that differs from the adult version only in that its script has been edited to make it more accessible to children ages 8–14 years and that two items that assess the critical overvaluation of shape and weight have been supplemented with a sort task. As with the EDE, the ChEDE's interview-based, interactive nature allows for questions to be explained so that they are understood by each individual. In addition, special care is taken, and examples are provided, to explain difficult concepts such as "loss of control," or the sense of being unable to stop eating once started. For example, when a child does not readily understand the concept of loss of control, one of the standardized descriptions used is that the experience is "like a ball rolling down a hill, going faster and faster."

As with the adult version, the ChEDE contains 21 items that assess disordered attitudes and behaviors related to eating, body-shape, and weight and 13 items designed to diagnose specific *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV;* American Psychiatric Association, 1994) eating disorders. Responses are coded with four subscales: Restraint (dietary restraint, as measured by behavioral and/or cognitive restraint), Eating Concern, Shape Concern, and Weight Concern. An overall global score consisting of the average of the four subscales can also be generated. The ChEDE also identifies three types of eating episodes: objective binge eating (OBE; overeating with loss of control), subjective binge eating (SBE; loss of control without objective overeating as assessed by the interviewer, but viewed as excessive by the interviewee), and objective overeating (OO; overeating without loss of control). The continuous global score and subscales (measures of cognitions and related behaviors) are generated from variables different from those used for the categorical OBEs, SBEs, and OOs (measures of behavior). Tests of the Adult EDE's discriminant validity, internal consistency, and concurrent validity (Fairburn & Cooper, 1993; Williamson, Anderson, Jackman, & Jackson, 1995) and test–retest reliability (Rizvi, Peterson, Crow, & Agras, 2000) support its use. There have been a limited number of published studies that used the ChEDE (Burrows & Cooper, 2002). In a sample of young girls (8–14 years) diagnosed with anorexia nervosa, selective eating (Bryant-Waugh & Kaminski, 1933), or food avoidance emotional

disorder (Higgs, Goodyer, & Birch. 1989) compared with healthy controls, interrater reliability (Spearman's rank correlations for the individual questions ranged from .91 to 1.00), internal consistency, and discriminant validity supported the use of the ChEDE (Frampton, 1996). Indeed, girls with a clinical diagnosis of anorexia nervosa had very similar subscale scores to adults with the same disorder, whereas those with selective eating or food avoidance emotional disorder scored similarly to normal controls. Further, most of the individual responses were found to be consistent with clinical observation, supporting the validity of the instrument (Christie, Watkins, & Lask, 2000).

Training, which is required to administer the ChEDE, was conducted by Marian Tanofsky-Kraff and Denise E. Wilfley. Interviewers were graduate students in clinical psychology and postundergraduate research associates who attended 15–20 hr of training wherein the criteria for eating disorders were reviewed and each EDE and ChEDE question was described. Prior to administering interviews, each interviewer was trained on both the EDE and the ChEDE by listening to audiotapes of sample adult and child interviews, conducting a practice EDE and ChEDE, and observing the trainer conducting a ChEDE. Each interviewer was also viewed administering a ChEDE, during which the trainer coded along with the trainee. Training was continued until at least 95% agreement between trainee and trainer ratings was demonstrated. To ensure administration quality, we audiotaped each interview and held ongoing weekly meetings to review every interview throughout data collection. Several precautions were taken to decrease the impact of social desirability pressures on the responses of certain participants. The nature of the ChEDE includes sensitive yet detailed probing designed to help the interviewer uncover information that the child might otherwise feel uncomfortable revealing. Interviewers were trained to set a positive rapport with the aim to set each child at ease so that participants would feel comfortable speaking honestly. Participants were told that interviews were confidential unless the child disclosed information involving imminent danger to self or others.

A book of photographed food portions (Hess, 1997) in various sizes and types was used during every interview to determine the amount and variety of food eaten. At the weekly meetings, any responses that were difficult to code and the coding of eating episode sizes were determined by means of consensus. To examine what constitutes a large amount of food for children, we asked all participants, whether or not they described disordered eating, to describe the *largest* amount eaten within the past 28 days. With this information, and the use of the portion book, team members discussed the amount of food eaten and came to a unanimous consensus regarding whether the amount was unambiguously large (e.g., eight slices of pizza) or subjectively large (e.g., two slices of pizza) given the circumstances for the child's age. If one or more team members disagreed with the rest of the group, an outside team proficient at administering both the EDE and ChEDE was contacted for further input. If, in the final analysis, an episode was still questionable, it was coded conservatively and rated down as a normal amount of food.

On the basis of their assessed eating behavior from the ChEDE, participants were categorized into four groups: OBE, SBE, OO, and those children who had engaged in no episodes (NE). Following usual practice, the 5 children engaging in more than one type of eating episode were categorized according to the most pathological eating behavior described. As the *DSM–IV* diagnostic criteria for binge eating episodes require large amounts of food for the diagnosis to be made (American Psychiatric Association, 1994), OBEs were considered the most pathological, SBEs intermediate, and OOs the least pathological behavior for the purposes of this analysis. Although the ChEDE was originally designed for children ranging in age from 8 to 14 years and 14 of the participants were below this cutoff, the majority of these children (n = 10) were over the age of 7 years. Exclusion of children less than 8 years old did not alter the direction or significance of any analysis. Interrater reliability, assessed with 40 (25%)

randomly selected ChEDE interviews, revealed excellent interrater reliability (intraclass correlation) for subscales from .95 to .99 (all *p*s < .01). Cohen's kappa for presence of the different eating episode categories was 1.00 ($p < .01$).

General Psychopathology

Children completed the Children's Depression Inventory (Kovacs, 1992), a 27-item self-rated measure of depressive feelings that generates a total score and five subscale scores (Negative Mood, Interpersonal Problems, Ineffectiveness, Anhedonia, and Negative Self Esteem) and the State-Trait Anxiety Inventory for Children (STAIC) A—Trait Scale (Spielberger. Edwards, Lushene, Montuori, & Platzek, 1973), a 20 item self-report measure of trait anxiety developed for use with elementary school children, Parents were asked to complete the Child Behavior Checklist for ages 4–18, an empirically derived measure with excellent norms that assesses a range of internalizing and externalizing symptoms.

Body Composition

Measurement of height (measured three times to the nearest 1 mm) was performed using a stadiometer (Holtain, Crymmych, Wales) calibrated before each child's height measurement to the nearest 1 mm. Weight to the nearest 0.1 kg was obtained using a calibrated digital scale (Scale-Tronix, Wheaton, IL). From these measurements, BMI (kg/m^2) was calculated.

Each participant also underwent a DXA scan (Hologic QDR-2000, Waltham, MA) in the pencil-beam mode for determination of body fat mass and lean body mass. Findings from DXA fat mass measurements have demonstrated excellent reproducibility in children ($r > .96$, interassay CV < 6%; Figueroa-Colon, Mayo, Treuth, Aldridge, & Weinsier, 1998), and a growing body of evidence indicates that DXA is an accurate method of quantifying fatness in children (Brunton, Bayley, & Atkinson, 1993; Chan, 1992; Ellis, Shypailo, Pratt, & Pond, 1994) and obese adults (Hicks et al, 1993).

Pubertal breast and pubic hair stage were assigned through physical examination by a pediatric endocrinologist or trained pediatric nurse practitioner to one of the five standards of Tanner (Marshall & Tanner, 1969, 1970). Testicular volume (in cc) for boys was also assessed using an orchidometer. Pubertal stage and testicular volume can be considered surrogate measurements of neurocognitive maturity as well as objective measures of physical maturity (Marshall & Tanner, 1969, 1970).

Analysis

Comparisons between groups were performed using one-way analysis of variance (ANOVA) with Bonferroni-Hochberg's correction, a conservative test accounting for multiple comparisons applied to each family of post hoc tests. Means ± standard deviations are reported, and nominal probability values are shown, unless otherwise indicated. DXA body fat mass was log-transformed so that these data approximated a normal distribution. A *z* score (Frisancho, 1990) that standardized for age, sex, and race was used for BMI (BMI-SD). To determine relevant covariates, we entered age, race, socioeconomic status (SES), gender, and pubertal stage into each full model and removed the covariates that were not significant. The finding that age remained an important covariate for several variables was expected given the natural developmental increases in frame size and ability to understand questions on psychological measures. Age was used as a covariate in analyses of the ChEDE Restraint and Global scales, the Children's Depression Inventory total and Anhedonia scores, the STAIC, BMI-SD, and DXA body fat mass. Race was used as a covariate for analyses of the anxiety measure, as a significant relationship between race and the STAIC was detected. For two-group comparisons, effect size is expressed as an effect size correlation, and for comparisons greater than two groups, effect size is expressed as an eta squared. Means adjusted for covariates are reported.

Pearson's chi-square tests were used to analyze differences between weight status and eating behavior. Differences and associations between groups were considered significant when *p* probability values after correction for multiple comparisons were less than or equal to .05, and all tests were two-tailed.

Results

Eighty-two (50.6%) of the children were overweight (BMI ≥ 85th percentile for age, race, and sex; Must, Dallal, & Dietz, 1991), with a mean BMI of 27.6 ± 7.1 kg/m^2; 80 (49.4%) of the children were of normal weight (BMI 15th–84th percentile), with a mean BMI of 17.3 ± 2.0 kg/m^2. Only 2 children (1.2%) had BMI ≤ 5th percentile and were included in the normal weight group. The data of these 2 children did not differ from the mean scores of the sample and thus their exclusion did not affect results. Overweight and normal weight children did not differ significantly in age, race, gender, pubertal stage, or SES. As expected, DXA fat mass significantly correlated with BMI (r = 0.91), BMI-SD (r = 0.83), and BMI centile (r = 0.76; all ps < .01).

Eating Behavior and Related Cognitions

According to their ChEDE interviews, no child met *DSM–IV* (American Psychiatric Association, 1994) criteria for anorexia nervosa, bulimia nervosa, or BED. However, overweight children scored significantly higher on the global ChEDE Scale, Restraint, and Shape and Weight Concern subscales (all ps < .01), and the Eating Concern subscale (p < .05; see Table 1). In analyses separating results of children with BMI ≥ 95th centile (n = 59) from those with BMI from the 85th up to the 95th centile (n = 23), participants with BMI in or above the 95th percentile had significantly higher Global, Weight Concern (both ps < .01), and Shape Concern scores (p < .01) and higher total Child Behavior Checklist scores (p < .05) than children with BMIs from the 85th up to the 95th percentile. These two subgroups of overweight children did not differ on any other variable. In addition, there was no interaction between gender and any of the ChEDE scales.

On the basis of their described eating behaviors over the past 28 days, 6.2% (n = 10) of children engaged in OBEs, 3.1% (n = 5) endorsed SBEs, 20.4% (n = 33) described OOs, and 70.4% (n = 114) described NEs. The frequencies with which participants engaged in eating episodes were low relative to the frequencies observed in those with eating disorders (e.g., Wilfley, Schwartz, Spurrell, & Fairburn, 2000). For those children engaging in OBEs, the frequency of episodes ranged from one to four in the past 28 days, with 82% (n = 7) of these children experiencing only one OBE within the timeframe. Similarly, SBE frequency ranged from one to four episodes per 28 days. Of those children engaging in OOs, episode frequency ranged between one and four episodes per 28 days, with 80% (n = 32) of these children reporting one OO episode.

Preliminary analyses revealed no significant quantitative or qualitative differences between the OBE and SBE groups on any of the dependent variables. This was not surprising given the small number of children engaging in these types of episodes. Given that studies in adults (Niego, Pratt, & Agras, 1997; Pratt, Niego, & Agras. 1998) and children (Morgan et al., 2002) have suggested that it is the loss of control, as opposed to the amount eaten, that is most salient for identifying those with disordered eating, we grouped the two types of loss-of-control episodes into one variable called subjective and objective binge eating (S/OBE). Pearson's chi-square tests found significant differences in the distribution of weight status by type of eating episode, $\chi^2(2, N = 106) = 6.2$, p < .05. S/OBEs were significantly more prevalent in overweight (14.6%, n = 12) than normal weight (3.8%, n = 3) children (p = .01). The BMI percentiles (39th, 60th, and 64th) of the 3 normal weight children engaging in loss-of-control episodes

were not near the 85th percentile cutpoint for diagnosis of overweight. Of the S/OBE group, 2 were boys and 13 were girls; 8 were African American and 7 were Caucasian.

When subscale scores from the ChEDE were examined among children endorsing S/OBE, OO, and NE (see Table 2), S/OBE scores were significantly greater for Restraint, $F(2, 157) = 3.5$, $p < .05$, Eating Concern, $F(2, 159) = 9.4$, $p < .01$, Shape Concern, $F(2, 159) = 5.8$, $p \leq .01$, and Weight Concern, $F(2, 159) = 7.3$, $p \leq .01$, compared with the OO and NE groups. ChEDE Global score also differed among groups, $F(2, 158) = 7.8$, $p \leq .01$. When these analyses were conducted with the overweight children only, findings remained constant with the exception of the Restraint and Shape Concern subscales, which no longer differed significantly among groups.

General Psychopathology

No significant group differences were found on any of the Children's Depression Inventory scales or on the STAIC when participants were analyzed according to body weight. Overweight children had significantly higher internalizing and externalizing scores compared with normal weight children (see Table 1). No differences were found on the Children's Depression Inventory, STAIC, or Child Behavior Checklist according to the endorsement of abnormal eating episodes (see Table 2). These findings remained constant when only the overweight children were examined.

Body Composition

BMI-SD, $F(2, 158) = 3.6$, $p < .05$, and DXA body fat mass, $F(2, 153) = 3.1$, $p < .05$, were significantly greater in children endorsing S/OBE than OO or NE (see Table 2). These differences were maintained in analyses of covariance correcting for age and/or pubertal stage as appropriate.[1]

Discussion

In this exploratory study of normal weight and overweight non-treatment seeking children, we found that overweight children experienced more eating-disordered behavior, greater levels of eating-disordered cognitions, and more parent-reported behavior problems than normal weight children. Moreover, eating with loss of control, compared with no overeating or overeating without loss of control, was significantly related to greater eating-disordered pathology, higher BMI, and greater body fat mass. No differences were found among children endorsing S/OBEs, OOs, or NE in depressive symptoms, trait anxiety, or parent-reported behavior problems.

Given their age, and that none of the children in our study were seeking treatment for obesity or eating disturbances, it was not surprising that none met criteria for an eating disorder. Although this finding is in contrast to Morgan et al.'s (2002) questionnaire study with overweight children, it is supported by prior research in adolescents indicating that despite high prevalence rates of binge eating, only a very small percentage report meeting criteria for BED (Johnson et al., 1999). In community-based samples of adolescents in which researchers used interview methods of eating disorder assessment, no individuals met criteria for BED (Stice et al., 2001), and very low rates of anorexia nervosa and bulimia nervosa were detected (Stice et al., 1998; Stice et al, 2001). Indeed, studies in adults that have used more stringent interview-based assessments have tended to find lower prevalence rates of BED (Grilo, 2002).

[1]When study children were grouped by age (6.0–8.0 years [$n = 17$, 10.5%), 8.1–10.0 years [$n = 60$, 37%], and 10.1 and older [$n = 85$, 52.5%]), none of the children in the youngest (6.0–8.0 years) group engaged in any S/OBEs. The youngest child reporting any episodes of loss of control was 8.3 years. The oldest ($\geq$ 10.1 years) children had significantly lower trait anxiety scores compared with those of children in the two younger ($\leq$ 10 years) groups ($p < .05$). The children did not differ by age on any other psychological variables. Omitting the youngest (6.0–8.0 years) group did not alter the observed relationships between adiposity and other variables.

Approximately 9% (n = 15) of the children in our sample engaged in at least one OBE or SBE over the past 28 days. This finding is comparable with Maloney et al.'s (1989) assessment, which found that between 8.8% and 11.5% of non-treatment seeking children report engaging in disordered eating behaviors, but differed from Maloney et al.'s report in that younger children were not more likely to report disordered eating. In the present study, we also found that significantly more overweight, compared with normal weight, children engaged in loss-of-control episodes. These results support prior findings in adolescents (e.g., Fairburn & Wilson, 1993; Greenfeld et al., 1987; Neumark-Sztainer et al., 1997) and suggest that overweight children may be at greater risk for future full-syndrome eating disorder diagnoses compared with their normal-weight peers. However, other studies in adolescent samples have supported the finding that binge eating may predict weight gain (Stice et al., 1999) and obesity onset (Stice et al., 2002). Thus, it is likely that multiple pathways exist to the development of eating disorders: Obesity or eating pathology may initiate the process or they may occur synergistically. The finding of greater restraint scores in children endorsing episodes of loss of control may be evidence consistent with restraint theory. However, because S/OBE children were already heavier than those without loss of control, our data are also consistent with the possibility that weight gain itself leads to (often futile) attempts to restrain further weight gain.

We also found that 1 out of every 5 normal-weight and overweight children engaged in OOs without a sense of loss of control or distress. As children with OOs did not differ from those endorsing NEs in adiposity, mood, or eating-related cognitions, we hypothesize that children engaging in OOs will not be more likely to develop eating-disordered pathology in the future than the general population.

The finding that overweight children experience significantly greater eating-disordered cognitions is consistent with the results of previous studies that used questionnaires to assess the presence of eating-disordered pathology (Striegel-Moore et al., 1995; Vander Wal & Thelen, 2000). Moreover, the finding that overweight participants with BMIs at or above the 95th percentile, in comparison to those from 85th up to the 95th percentile, had higher ChEDE scores supports literature in adolescents (e.g., Buddeberg-Fischer, Klaghofer, & Reed, 1999) and children (e.g., Erickson et al., 2000; Striegel-Moore et al., 1995) indicating that eating-disordered pathology often increases as BMI rises. Although the mean ChEDE subscale scores of our sample were not at the high levels observed in eating disorder clinic patients, the fact that overweight children's scores were significantly higher than the normal weight participants' scores is notable. If social desirability pressures when being asked face-to-face questions caused the overweight children to underreport their experiences, the comparatively elevated scores should be considered that much more salient. Given that prospective research has suggested that adolescent girls who do not carry a diagnosis of a subclinical or clinical eating disorder but who score highly on measures of weight concern (Killen et al., 1994, 1996) or thin body preoccupation (McKnight Investigators, 2003) are at high risk for developing partial- or complete-syndrome eating disorders when examined 3 years later, the overweight children in our sample who scored relatively higher on the ChEDE subscales may be at high risk for developing eating-disordered pathology in the future. Despite concerns of increasing eating pathology in the future, some literature suggests that eating-disordered cognitions, in particular body dissatisfaction and weight concerns, may be adaptive by serving as motivators for healthy lifestyle changes (Heinberg, Thompson, & Matzon, 2001). Longitudinal studies examining the stability and consequences of eating-disordered cognitions and behaviors in children are needed.

Supporting prior research (e.g., Achenbach & Elderbrock, 1991; Stradmeijer et al., 2000; Tershakovec et al., 1994), overweight children, compared with normal-weight children in our sample, experienced more internalizing and externalizing problems, as reported by their parents, but not more problems on self-reported depressive symptoms or trait anxiety. This

was unexpected given that overweight has been related to depressive symptoms in non-treatment seeking children (Erickson et al., 2000; Striegel-Moore et al., 1995; Vander Wal & Thelen, 2000), and our measures of behavior problems, depressive symptoms, and trait anxiety were predicted to assess a similar broad construct. Our findings are also in contrast to research in adolescents supporting the relationship between binge eating behaviors and other psychological problems (e.g., Johnson et al., 1999; Ledoux et al., 1993; Steiger et al., 1991). It may be that individuals in middle childhood are not yet fully aware of their emotional experiences, which might explain why significant differences were detected on the ChEDE and the Child Behavior Checklist, measures that do not depend on self-interpretations. Alternatively, it is possible that overweight young children and those engaging in binge eating behaviors may have yet to develop the early signs of other psychological problems. Given that our sample consisted of non-treatment seeking children without a current eating disorder, depressive symptoms and trait anxiety may not be a common component of their current psychological makeup. In contrast, behavioral problems as measured by the Child Behavior Checklist may be reflecting parent observations of the poor social skills and lack of social acceptance that stigmatized overweight children often endure (Dietz, 1995). Another contributing factor may be that in completing the Child Behavior Checklist, parents infer that their overweight children are more emotionally distressed given the negative social attitudes toward overweight individuals. Our findings do, however, support research in adults, which suggests that the relationship between obesity and psychopathology is highly inconsistent (Friedman & Brownell, 2002).

The relationship between increased BMI and binge eating in adolescents has been demonstrated in some cases (e.g., Moore, 1988, 1990; Stice et al, 2002), although two studies did not find body mass to predict binge eating (Stice, 1998; Stice & Agras, 1998). Our findings lend support to the hypothesis that loss-of-control eating and adiposity may share an important developmental relationship in childhood (Morgan et al., 2002). Not only BMI but also DXA fat mass was greater in those endorsing OBEs or SBEs. Thus, our findings suggest that there may be a distinct relationship between binge eating and body fat, although the mediating variables (e.g., emotional eating, self-esteem, home environment, and social support, among many others) of this association merit further exploration.

Limitations of this study include the fact that children were not recruited in a truly population-based fashion. However, children and their families were recruited for studies measuring plasma hormones, understood that they would not receive any treatment as a result of participation, and did not have descriptions of the contents of the psychological interviews and questionnaires in advance. Thus, we believe this sample is likely to be reasonably representative of the general population of normal-weight and overweight children, and it is unlikely to be enriched for individuals who were, or were not, concerned with eating behavior. Nonetheless, because the families in our sample chose to respond to our notices, they may be unique in other ways, limiting the external validity of the study. In addition, the possibility of interviewer bias against body size and social desirability pressures may still have been present, even though precautions were taken to avoid bias during the interview. Finally, merging OBEs and SBEs may be a limitation. In contrast to some studies (Niego et al, 1997; Pratt et al., 1998), using the adult EDE, Rizvi et al. (2000) found poor test-retest reliability for SBEs but not for OBES, suggesting that size of a loss-of-control eating episode may be a distinguishing feature of disordered eating episodes.

Strengths of this study include not only the methodology used to measure body adiposity and eating pathology but also the representation of both Caucasian and African American children. Indeed, the finding that similar numbers of African American and Caucasian children engage in binge eating episodes and that race did not appear to influence the degree to which children struggle with eating-disordered cognitions and general psychopathology is similar to findings

in community samples of adults (Striegel-Moore, Wilfley, Pike, Dohm, & Fairburn, 2000; Yanovski, 2000).

In summary, our findings suggest that the percentage of children engaging in eating-disordered episodes is sizable and that there is evidence of eating disturbance in preadolescence but that full-syndrome eating pathology is not yet present. Intervention with prepubescent overweight children or those demonstrating early signs of eating disturbance may potentially decrease the number of children who later develop more extensive eating problems or obesity. Longitudinal studies are needed to determine whether overweight children who report binge eating and experience increased levels of eating-disordered cognitions are at greater risk to develop clinical eating disorders in the future.

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Table 1

ChEDE, Children's Depression Inventory, STAIC-Trait, and Child Behavior Checklist Scores

Measure	Normal weight (n = 80)		Overweight (n = 82)				
	M	*SD*	*M*	*SD*	*F*	*df*	Effect size *r*
ChEDE							
Restraint subscale	0.13	0.31	0.76	1.00	27.0**	1, 161	.39
Eating Concern subscale	0.05	0.17	0.13	0.30	4.4*	1, 161	.16
Shape Concern subscale	0.21	0.36	0.75	1.00	20.8**	1. 162	.34
Weight Concern subscale	0.31	0.60	1.1	12.00	30.2**	1,162	.78
Global score	0.17	0.27	0.70	0.74	35.9**	1, 162	.43
Children's Depression Inventory							
Total	5.8	5.9	6.5	4.9	1.2	1, 157	−.06
Negative Mood	1.3	1.6	1.2	1.1	0.36	1, 157	−.04
Interpersonal Problems	0.51	0.85	0.58	0.98	0.28	1, 157	.04
Ineffectiveness	0.92	1.4	1.1	1.4	0.93	1, 157	.06
Anhedonia	2.1	2.3	3.0	2.6	5.1	1, 157	.18
Negative Self-Esteem	0.82	1.2	0.62	0.92	1.3	1, 157	−.09
STAIC-Trait Anxiety	32.7	6.4	34.3	8.0	2.1	1, 152	.11
Child Behavior Checklist							
Internalizing *T* score	46.9	9.4	50.4	10.9	4.5*	1, 155	.17
Externalizing *T* score	46.7	9.6	49.8	9.9	4.0*	1,155	.16
Total *T* score	47.4	11.7	50.6	12.4	2.5	1, 155	.13

Note. Analyses for the Children's Eating Disorder Examination (ChEDE) Restraint subscale and global score and the Children's Depression Inventory total and Anhedonia scores include age as a covariate; analyses for State-Trait Anxiety Inventory for Children (STAIC-Trait Anxiety) include age and race as covariates.

* $p < .05$.

** $p < .01$.

Table 2

ChEDE, Children's Depression Inventory, STAIC, and Child Behavior Checklist Results Based on Eating Behavior

Measure	NE (*n* = 114)		OO (*n* = 33)		S/OBE (*n* = 15)				
	M	*SD*	*M*	*SD*	*M*	*SD*	*F*	*df*	η^2
ChEDE									
Restraint subscale	0.43_a	0.85	0.27_a	0.80	0.93_b	0.81	3.5^*	2,157	.04
Eating Concern subscale	0.06_a	0.21	0.08_a	0.17	0.33_b	0.39	9.4^{**}	2,159	.11
Shape Concern subscale	0.44_a	0.75	0.37_a	0.63	1.1_b	1.2	7.3^{**}	2,159	.07
Weight Concern subscale	0.64_a	0.96	0.58_a	1.1	1.7_b	1.2	5.8^{**}	2,159	.10
Global score	0.37_a	0.58	0.37_a	0.54	1.1_b	0.86	7.8^{**}	2,158	.10
Children's Depression Inventory									
Total	6.0	5.4	6.7	6.5	6.1	3.2	0.19	2,150	.02
Negative Mood	1.2	1.3	1.4	1.6	1.4	1.0	0.12	2,151	.01
Interpersonal Problems	0.57	0.98	0.52	0.81	0.43	0.65	0.16	2,151	.01
Ineffectiveness	1.0	1.4	0.96	1.6	1.2	1.4	0.15	2,151	.01
Anhedonia	2.5	2.5	2.8	2.6	2.2	1.5	0.39	2,150	.02
Negative Self-Esteem	0.68	1.0	0.84	1.4	0.79	0.98	0.29	2,151	.01
STAIC-Trait Anxiety	33.2	7.4	34.2	6.4	33.7	8.0	0.24	2,149	.004
Child Behavior Checklist									
Internalizing *T* score	48.6	10.3	47.3	10.4	51.9	9.4	0.92	2,148	.01
Externalizing *T* score	47.2	9.6	49.9	10.3	52.2	10.1	2.1	2,148	.03
Total *T* score	48.0	12.4	50.6	11.9	52.7	10.2	1.2	2,148	.02
Body composition									
BMI (kg/m^2)	21.9_a	8.0	23.8_a	8.6	28.2_b	8.1	3.6^*	2,158	.02
BMI-SD	1.5_a	2.3	1.4_a	2.4	3.2_b	2.4	3.6^*	2,158	.04
DXA fat mass	12.59_a	11.4	12.59_a	6.6	19.95_b	4.6	3.1^*	2,153	.03

Note. Analyses for the Children's Eating Disorder Examination (ChEDE) Restraint subscale and global score, the Children's Depression Inventory Total and Anhedonia scores, and body mass index (BMI), BMI standardized for age, sex, and race (BMI-SD), and dual energy X-ray absorptiometry (DXA) fat mass include age as a covariate; analyses for State-Trait Anxiety Inventory for Children (STAIC-Trait Anxiety) include age and race as covariates. Different subscript letters indicate significant differences between groups. NE = no episodes of overeating; OO = objective overeating; S/OBE - subjective and objective binge eating.

$^*p < .05$.

$^{**}p < .01$.