Journal of Abnormal Psychology
2008, Vol. 117, No. 1, 225–235

0021-843X/08/$12.00 DOI: 10.1037/0021-843X.117.1.225

Eating Pathology Among Adolescent Girls With Attention-Deficit/Hyperactivity Disorder

Amori Yee Mikami
University of Virginia

Stephen P. Hinshaw
University of California, Berkeley

Katherine A. Patterson
University of California, San Diego and
San Diego State University

Joyce Chang Lee
University of California, Los Angeles

The authors investigated prospectively assessed eating pathology (body image dissatisfaction and bulimia nervosa symptoms) among an ethnically and socioeconomically diverse sample of adolescent girls with attention-deficit/hyperactivity disorder–combined type (ADHD-C; $n = 93$), ADHD–inattentive type (ADHD-I; $n = 47$), and a comparison group ($n = 88$). The sample, initially ages 6–12 years, participated in a 5-year longitudinal study (92% retention rate). After statistical control of relevant covariates, girls with ADHD-C at baseline showed more eating pathology at follow-up than did comparison girls; girls with ADHD–I were intermediate between these two groups. Baseline impulsivity symptoms, as opposed to hyperactivity and inattention, best predicted adolescent eating pathology. With statistical control of ADHD, baseline peer rejection and parent–child relationship problems also predicted adolescent eating pathology. The association between punitive parenting in childhood and pathological eating behaviors in adolescence was stronger for girls with ADHD than for comparison girls. Results are discussed in terms of the expansion of longitudinal research on ADHD to include female-relevant domains of impairment, such as eating pathology.

Keywords: attention-deficit/hyperactivity disorder, girls, eating pathology, bulimia nervosa, body image dissatisfaction

It is well-known that children with attention-deficit/hyperactivity disorder (ADHD) are at risk for delinquency, academic failure, substance abuse, and depression and anxiety in adolescence (Barkley, 2002; Mannuzza & Klein, 2000). Yet research has not examined relationships between ADHD and adolescent eating pathology, considered in this study to be body dissatisfaction and maladaptive bingeing–purging behaviors symptomatic of bulimia nervosa (BN). In this introduction, we review (a) reasons why such investigation has been limited, (b) the theoretical rationale as to why ADHD and eating pathology may be linked, (c) existing research on this question, and (d) the potential for differential predictions regarding the ADHD–inattentive (ADHD-I) and ADHD–combined (ADHD-C) types.

Lack of Research on ADHD and Eating Pathology

Two factors that have limited the investigation of links between ADHD and eating pathology are the strong preponderance of males in the ADHD literature, far exceeding the community male: female ratio of 3:1 (Hinshaw & Blachman, 2005), and the focus on children as opposed to adolescents with ADHD. Eating pathology overwhelmingly affects females—who outnumber males 9:1 in diagnoses of BN and 3:1 in subclinical BN symptoms and body image dissatisfaction (Sweeting & West, 2002)—and postpubertal adolescents (Stice, Presnell, & Bearman, 2001). A prospective longitudinal sample of girls with ADHD followed into adolescence is required to conduct a sensitive test of risk for eating pathology, yet this type of sample is rare (see Hinshaw, Owens, Sami, & Fargeon, 2006).

Theoretical Rationale

There are several theoretical reasons to believe that girls with ADHD may be at risk for BN symptoms and body image dissatisfaction in adolescence. First, although boys with ADHD often display adolescent conduct and substance abuse disorders, girls may show distress through eating and internalizing disorders, problem areas with female predominance. Supporting this idea are findings that compared with boys with conduct disorder, girls with

Amori Yee Mikami, Department of Psychology, University of Virginia; Stephen P. Hinshaw, Department of Psychology, University of California, Berkeley; Katherine A. Patterson, Joint Doctoral Program in Clinical Psychology, University of California, San Diego and San Diego State University; Joyce Chang Lee, Division of Child and Adolescent Psychiatry, Semel Institute for Neuroscience and Human Behavior, University of California, Los Angeles.

Work on this research was supported by National Institute of Mental Health Grant R01 MH45064. We express our great appreciation to the girls and their families who participated in this project. We also thank Elizabeth Owens, Nilofar Sami, and Samantha Fargeon, as well as the many graduate students and research assistants who helped with data collection.

Correspondence concerning this article should be addressed to Amori Yee Mikami, Department of Psychology, University of Virginia, 102 Gilmer Hall, P.O. Box 400400, Charlottesville, VA 22904-4400. E-mail: mikami@virginia.edu

conduct disorder are at higher risk for developing depression, suicide, and somatization disorders (Pajer, 1998). If there is a parallel with conduct problems, girls with ADHD may display a greater degree of multifinality with respect to long-term outcomes than do boys with this condition (Hinshaw et al., 2006)—such that one risk factor (in this case, childhood ADHD) predicts diverse problems.

Second, impulsivity is part of the diagnostic criteria for youths with ADHD-C, and ADHD has been conceptualized as a disorder of "behavioral inhibition" (Barkley, 2003). Dysregulated and impulsive personality traits are similarly central to BN (Fahy & Eisler, 1993). Impulsive behaviors in life domains beyond eating, such as delinquency and substance abuse, have been shown to predict onset of BN symptoms 9 months later in a sample of adolescent girls (Wonderlich, Connolly, & Stice, 2004). Furthermore, individuals with BN who display other impulsive behaviors (e.g., truancy, stealing, drug abuse, multiple sexual partners, or self-injury) may have a worse treatment prognosis than those without such multiple indicators of impulsivity (Wiederman & Pryor, 1996). Individuals with BN have even been found to show inhibitory control deficits on a stop-signal task (Nederkoorn, Van Eijs, & Jansen, 2004), a computerized neuropsychological measure on which youths with ADHD-C have been established to show deficient performance (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005).

Third, children with ADHD are at high risk for concurrent problems in parent–child and peer relationships (Asarnow, 1988), two domains that may be related to BN symptoms. It is well documented that children with ADHD have more conflictual relationships with their parents than do children without ADHD (Johnston & Mash, 2001). Parents of girls with ADHD also show higher levels of expressed emotion (EE)—composed of critical and overinvolved attitudes parents hold toward offspring—than do parents of comparison girls (Peris & Hinshaw, 2003). Negative parent–child relationships have been theorized to influence the development of BN (Strober & Humphrey, 1987). Conflict and criticism (Polivy & Herman, 2002; Strober & Humphrey, 1987), as well as EE per se (Butzlaff & Hooley, 1998; Hodes & le Grange, 1993; van Furth, van Strien, & Martina, 1996), have been shown to (a) be prevalent in families of youths with concurrent BN, (b) be reported retrospectively by individuals with BN to have existed in childhood, and (c) predict poor treatment prognosis among already symptomatic individuals with BN. Two studies have found that parent-reported instances of arguments in childhood predicted BN in adolescence (Johnson, Cohen, Kasen, & Brook, 2002; Marchi & Cohen, 1990). Yet prospective longitudinal studies linking observed parent–child relationship problems to future BN symptoms are lacking (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004).

High rates of peer rejection, clearly seen in children with ADHD (Hoza et al., 2005), may also contribute to BN. Individuals with eating pathology retrospectively self-report more childhood teasing by peers—particularly about weight—than do nonclinical individuals (Fairburn et al., 1998; Striegel-Moore, Dohm, Pike, Wilfley, & Fairburn, 2002). Provocative data from three longitudinal studies have revealed that girls with low self-reported social support (Bearman, Presnell, Martinez, & Stice, 2006; Stice, Spangler, & Agras, 2001) and high self-reported unpopularity (Killen et al., 1994) were vulnerable to developing BN symptoms. However, to our knowledge the predictive relationship between peer-reported, sociometrically assessed rejection (considered the gold standard in the field) and BN eating pathology has not been tested.

Parent–child and peer relationship problems may be more predictive of BN symptoms or body image dissatisfaction among girls with ADHD than among comparison girls because other research has demonstrated that these relationship factors influence boys with ADHD to a greater extent that they do boys without such a disorder. Boys with ADHD reap benefits from authoritative parenting practices in terms of peer acceptance (Hinshaw, Zupan, Simmel, Nigg, & Melnick, 1997), but experiencing such parenting appears less important for comparable social outcomes of comparison boys. Boys with ADHD also display greater susceptibility to the negative effects of a deviant peer group (Marshal, Molina, & Pelham, 2003) than do comparison boys, who are not as influenced by such peer processes. Yet models of parent–child and peer influences on outcomes have not to date been well studied in girls with ADHD, so it remains unknown whether processes will be similar among girls.

Finally, children with ADHD are likely to maintain or develop internalizing and disruptive disorders in adolescence (Hinshaw et al., 2006; Mannuzza & Klein, 2000), which may also increase risk for BN symptoms and body image dissatisfaction (Herzog, Nussbaum, & Marmor, 1996). Internalizing problems, particularly negative affect, have been shown to be predictive of BN and body image dissatisfaction (Bearman et al., 2006; Leon, Fulkerson, Perry, Keel, & Klump, 1998). Disruptive problems such as impulsive substance use (Leon et al., 1998) and self-reported aggressive behavior (Killen et al., 1994) have been linked with BN.

Existing Research

Despite these provocative leads, extant research has largely not investigated eating pathology as an outcome for children with ADHD because most prospective studies of this condition have few female participants (e.g., Barkley, Fischer, Smallish, & Fletcher, 2002; Biederman, Newcorn, & Sprich, 1991; Latimer et al., 2003; Mannuzza & Klein, 2000; Weiss & Hechtman, 1993). One notable exception is the Biederman et al. (2006) investigation including an assessment of eating disorders in a 5-year longitudinal study of girls with ADHD. Results showed a marginally significant pattern ($p = .06$) such that more girls with ADHD displayed eating disorders (both BN and anorexia nervosa combined) than girls without ADHD. However, the wide age range and the ethnic homogeneity of the sample (6–18 years old at baseline; 94% of girls with ADHD were White), the restricted outcome variable of diagnosable eating disorders, the lack of differentiation between eating disorder diagnoses and between the combined and inattentive types of ADHD, and the lack of inclusion of mediators or moderators all constitute limits of this investigation.

Two studies using cross-sectional methodology have recently reported exclusively descriptive findings of a concurrent, higher BN prevalence rate among females with ADHD relative to comparison females (Mattos et al., 2004, in a Brazilian sample; Surman, Randall, & Biederman, 2006, whose sample included the girls from the Biederman et al. (2006) study cited earlier); neither study differentiated between ADHD-C and ADHD-I types, incorporated covariates, or probed this link. To our knowledge, there are no other published, empirical, group comparison investigations of

the relationship between eating pathology and ADHD. However, case studies have reported that stimulant medication for ADHD had the effect of improving patients' comorbid BN (Schweickert, Strober, & Moskowitz, 1997; Sokol, Gray, Goldstein, & Kaye, 1999).

ADHD Types

We aim to differentiate BN symptoms and body image dissatisfaction between girls with ADHD-I (characterized by primary deficits in attention) versus those with ADHD-C (characterized by deficits in both attention and hyperactivity–impulsivity). Because ADHD-hyperactive–impulsive type is primarily found in preschoolers and these children largely become classified as ADHD-C by school age (Lahey et al., 1994), this group was not included in the present sample. Girls with ADHD-C may be at higher risk than girls with ADHD-I for BN symptoms, given that impulsivity is central to the diagnostic criteria for ADHD-C but not for ADHD-I. However, in addition to displaying attention problems, at least six of nine total symptoms of hyperactivity (six symptoms) and/or impulsivity (three symptoms) are required for a diagnosis of ADHD-C, but fewer than six of nine symptoms are required for a diagnosis of ADHD-I (American Psychiatric Association, 2000). Thus, it is possible for a girl with ADHD-C to have no impulsive symptoms (and six hyperactive symptoms) and for a girl with ADHD-I to have three impulsive symptoms (and zero to two hyperactive symptoms). To the extent that impulsive symptoms exist within the ADHD-I type, this group may also be at risk for BN.

Hypotheses

We hypothesize that girls with ADHD will be at higher risk than comparison girls for adolescent eating pathology—defined in this study as BN symptoms and body image dissatisfaction—with control of age, childhood comorbid disorders and satisfaction with physical appearance, IQ, body mass index (BMI), medication use, and early puberty. We further hypothesize that girls with ADHD-C will be at greater risk than girls with ADHD-I. Next, we hypothesize that relative to childhood inattention and hyperactivity symptoms, childhood impulsivity will best predict adolescent eating pathology; we also hypothesize that childhood parent–child and peer relationship problems will predict eating pathology and that ADHD diagnosis will serve as a moderator, such that predictive relationships will be stronger for girls with ADHD than for comparison girls. Finally, we examine associations between concurrent adolescent diagnoses of ADHD, internalizing and externalizing problems, and eating pathology.

We included the covariates described above because higher BMI (Stice, Agras, & Hammer, 1999), early puberty (Stice, Presnell, & Bearman, 2001), and older adolescent age (Shore & Porter, 1990) are risk factors for BN and body image dissatisfaction. Stimulant medication is considered because it is the most widely used treatment for ADHD, and it has the side effect of appetite suppression (Barkley, 2004). Consistent use may lead to slight reductions in BMI (Swanson et al., 2007). Finally, to better examine the independent effects of childhood ADHD on eating pathology, we include baseline comorbidities, IQ, and satisfaction with physical appearance in predictive models.

Method

Overview of Procedures

Girls with ADHD and comparison girls participated together in research summer day camps, grouped into classes of same-age but mixed-diagnosis peers. Approximately half of girls with ADHD had been receiving stimulant medication prior to the programs, but nearly all agreed to participate while unmedicated; because of the short half-life of stimulant medication, they discontinued use 24 hr before the program. For girls whose families requested a medication trial, data herein reflect behavior during unmedicated periods. Participants returned for follow-up assessments between 4 and 5 years following the summer program; during these assessments, parent, teacher, and adolescent reports of functioning in a variety of domains were collected. For further details about methodology, including participants, measures, and an overview of findings (including descriptive statistics on a subset of eating pathology measures), see Hinshaw (2002) and Hinshaw et al. (2006).

Participants

The baseline sample included 228 participants (ages 6–12, mean age = 9.5): 93 with ADHD-C, 47 with ADHD-I, and 88 comparison girls. At follow-up, 92% of the sample returned (ages 11–18, mean age = 14.2): 86 with ADHD-C, 41 with ADHD-I, and 82 comparison girls. Both girls with ADHD and comparison girls were initially recruited through pediatricians, schools, and advertisements; girls with ADHD were also recruited through mental health centers. The sample was diverse in socioeconomic status and ethnicity (53% Caucasian, 27% African American, 11% Latina, and 9% Asian American). Girls with ADHD met clinical cutoffs on parent and teacher rating scales (Child Behavior Checklist [CBCL], Achenbach, 1991a; Teacher's Report Form [TRF], Achenbach, 1991b; and the Swanson, Nolan, and Pelham Rating Scale—4th ed. [SNAP-IV], Swanson, 1992), and *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) diagnosis of ADHD was validated in parent clinical interview (Diagnostic Interview Schedule for Children—4th ed. [DISC–IV], Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). All girls had at least six inattentive symptoms: Those with six or more hyperactive–impulsive symptoms were designated ADHD-C; those with fewer than 6 hyperactive–impulsive symptoms were designated as ADHD-I. Comparison girls were below ADHD cutoffs on rating scales, and there could be no diagnosis of ADHD on the DISC–IV, but a few comparison girls had other disorders. Comparison girls were at national norms on parent and teacher rating scales of ADHD symptoms, disruptive behaviors, and internalizing behaviors (Hinshaw, 2002)

The retained sample did not differ from those lost to attrition on 29 of 31 baseline symptom and demographic variables. Those lost to attrition (a) were more likely to be from single-parent homes and (b) had higher teacher-reported internalizing scores (see Hinshaw et al., 2006).

We also found no differences on demographic measures among the ADHD-C, ADHD-I, and comparison girls: age, $F(2, 225) = 0.88$, $p > .10$; family income, $F(2, 216) = 1.22$, $p > .10$; proportion White versus non-White, $\chi^2(2, N = 228) = 2.13$, $p > .10$; parental marital status, $\chi^2(4, N = 228) = 4.33$, $p > .10$; or

number of adults in household, $F(2, 225) = 0.12$, $p > .10$. (See Hinshaw, 2002; Hinshaw et al., 2006.)

Baseline Measures

Independent variable: Baseline ADHD symptoms and diagnoses. Parents reported on the DISC–IV, a well-validated, structured interview yielding categorical diagnoses and symptom counts for the major disorders in the *DSM–IV*. Parents and teachers reported on the SNAP–IV, a checklist of the nine *DSM–IV* items for inattention, the six items for hyperactivity, and the three items for impulsivity, with each scored on a 0 (*not at all*) to 3 (*very much*) metric. The SNAP–IV is extensively used for the screening of ADHD—for example, it was a primary scale used in the Multimodal Treatment of Children With ADHD study (Hinshaw, March, et al., 1997), with acceptable to excellent internal consistency, test–retest reliability, and validity statistics (Swanson, 1992).

Predictor: Peer rejection. Standard sociometric procedures (Coie, Dodge, & Coppotelli, 1982) were performed during the summer camp. All children in a classroom nominated three peers with whom they would most like and least like to be friends. Proportion scores were calculated by dividing the number of "most liked" and "least liked" nominations received by the number of peers providing nominations. The stability of nominations over 5 weeks was high for positive nominations, $r(226) = .51$, $p < .001$, and extremely high for negative nominations, $r(226) = .85$, $p < .001$; see Blachman and Hinshaw (2002) for further details. Peer rejection was operationalized by subtracting the proportion of "most liked" nominations from the proportion of "least liked" nominations.

Predictor: Parenting practices. Primary caregivers (usually mothers) reported on the Alabama Parenting Questionnaire (Shelton, Frick, & Wootton, 1996) Corporal Punishment subscale, assessing spanking and hitting of the child (3 items; $\alpha = .62$). Caregivers reported on the Ideas about Parenting (Heming, Cowan, & Cowan, 1990) Authoritarian subscale, tapping power-assertive discipline (17 items; $\alpha = .76$). In vivo parent–child interactions, held prior to the summer programs, were coded by raters unaware of the child's diagnostic status. Caregivers asked their daughters to clean up items. Coders judged the extent to which the parent made negative comments about the child, intruded in the task, and used physical control. Coding for each item was on a 4-point Likert scale (intraclass correlations = .62–.80, αs = .83–.92). A score of parental negativity was composed of the average of the three behaviors. For two of the three summer programs, caregivers engaged in the Five Minute Speech Sample in which a parent speaks for 5 uninterrupted minutes about his or her child. This procedure has been widely used (see Hodes & le Grange, 1993) to capture EE, a construct measuring parental criticism of and emotional overinvolvement with the child. In the standardized coding system, both criticism ("She gets on everyone's nerves") and overinvolvement ("She is not allowed to spend the night at a friend's house because she might get hurt") are rated as low, borderline, or high. For EE to be coded as present, the parent must have high criticism, high overinvolvement, or both; the presence of both borderline criticism and overinvolvement would not result in a classification of EE. Interrater reliability was acceptable (agreement = 86%–92%, κs = .61–.80). See Peris and Hinshaw (2003) for further details.

Covariate: Comorbid disorders. Disruptive and internalizing comorbid disorders at baseline were assessed by means of parent report on the DISC–IV. Disruptive disorders were coded as 1 or 0 for the presence or absence of oppositional defiant disorder or conduct disorder. Internalizing disorders were coded as 1 or 0 for the presence or absence of anxiety or mood disorders (note that anxiety disorders had to include conditions beyond specific phobias). The use of categorical codes for comorbidities reflects practice in psychopathology research (e.g., Biederman et al., 1999; Hinshaw, 2002).

Covariate: Cognitive ability. We assessed Full Scale IQ using the Wechsler Intelligence Scale for Children, third edition (Wechsler, 1991), a widely used, psychometrically sound test. Test–retest and split-half reliabilities are high for the Full Scale IQ (.94–.96; Kaufman, 1993).

Covariate: Satisfaction with general physical appearance. This was assessed using the Harter Self-Perception Profile for Children (Harter, 1985) Physical Appearance (ages 8 and above) and Physical Competence (ages 6–7) subscales. The Harter assesses self-perceptions of competence in a number of domains (e.g., peer competence, academic skills, and physical appearance) and is a well-established and widely used measure in the child clinical literature. Each subscale has six items, with alphas of .71–.86. This subscale describes different children ("Some kids feel that they look okay, but other kids worry that they do not look as good as other children") and asks the participant to first indicate which description is most like herself and then whether that description is "very much" like herself or only "somewhat" like herself, yielding a score on a 4-point metric. The sum of all items yields a measure of satisfaction with one's global physical appearance.

Covariate: Body mass index (BMI). This standardized measure of adiposity (collected with two thirds of the sample at baseline; missing data were equally distributed across ADHD-C, ADHD-I, and comparison groups) was calculated as follows: weight in kilograms divided by the square of height in meters (Keys, Fidanza, Karvonen, Kimura, & Taylor, 1972).

Follow-Up Measures

Dependent variables: Eating pathology. Adolescents reported on the Eating Disorders Inventory—II (EDI–II: Garner, 1991), a well-validated measure of eating pathology symptoms. Subscales include Body Dissatisfaction (nine items); Bulimia (seven items); and Drive for Thinness (seven items). Internal consistencies range from .69 to .93, with a mean of .87; test–retest reliabilities range from .77 to .97; and validity is extensively documented (Garner, 1991). Each item was scored on a 1–6 metric (1 = *never*, 2 = *rarely*, 3 = *sometimes*, 4 = *often*, 5 = *usually*, and 6 = *always*). As recommended by Garner (1991), scores were compressed such that the less pathological scores of 1–3 were recoded as 0, and the more pathological scores of 4, 5, and 6 were recoded as 1, 2, and 3, respectively. Scores on each item were summed to form the corresponding scale.

Adolescents reported on the Eating Attitudes Test (EAT: Garner, Olmstead, Bohr, & Garfinkel, 1982), a well-validated measure of dysfunctional eating behaviors. Subscales are Dieting (13 items;

e.g., "I think about burning up calories when I exercise"), Oral Control (7 items; e.g., "I avoid eating when I am hungry"), and Bulimia/Food Preoccupation (6 items; e.g., "I feel that food controls my life"). Subscales yield alphas between .8 and .9 and discriminate adolescents with eating disorders from comparison youths (Garner & Garfinkel, 1979). The EAT was scored and recoded on the same metric as the EDI–II, as recommended by Garner et al. (1982). Scores on each item were summed to form the corresponding scale.

Adolescents reported on the Body Image Survey, containing nine sketches of women of varying weights, associated with the numbers 10, 20, and so forth through 90 (Fallon & Rozin, 1985). Girls indicated the number of the sketch corresponding to their current weight and to their ideal weight; girls could also choose numbers corresponding to weights between two figures (e.g., 25 as weight between Sketchs 2 and 3). The current minus ideal weight represented a desire to be thinner.

Parents reported on the DISC–IV (Shaffer et al., 2000) to yield symptom counts and diagnosis of BN.

Concurrent symptomatology: Follow-up ADHD diagnoses. Parallel to the procedure at baseline, ADHD diagnostic status was assessed through the parent-reported DISC–IV (Shaffer et al., 2000) and parent and teacher symptom counts on the SNAP–IV (Swanson, 1992).

Concurrent symptomatology: Externalizing and internalizing problems. As described in Mikami and Hinshaw (2006), a composite score for externalizing symptoms was created from parent and teacher report on the Aggressive Behavior and Delinquency subscales on the CBCL and parallel TRF and from participant self-report on the Self-Reported Delinquency scale (Elliott, Huizinga, & Ageton, 1985). A composite score for internalizing symptoms was created from parent and teacher report on the Anxiety/Depression subscale on the CBCL and TRF and from participant self-report on the Child Depression Inventory (Kovacs, 1992). Factor analyses with varimax rotation supported the combination of the measures into these two composites; see Mikami and Hinshaw (2006) for further details. Both composites were formed by taking the unweighted mean of the *z* scores of all items in the composite.

Covariate: BMI. This was calculated by the same means as at baseline, collected with the full sample at follow-up.

Covariate: Stimulant medication. Parents reported whether the girl had been taking stimulant medication for ADHD during any time between baseline and follow-up.

Covariate: Early puberty. This variable was assessed through adolescent retrospective self-report and defined as onset of menarche before age 12 (Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003; Keski-Rahkonen et al., 2005; Stice, Presnell, & Bearman, 2001).

Data Reduction

Self-report continuous subscales of eating pathology were converted into *z* scores to increase ease of interpretation and combination as recommended by Aiken and West (1991) and entered into a principal components analysis with varimax rotation. With seven subscales entered and more than 200 participants, the requirement of 10–15 participants per item was exceeded (Thompson, 2000). Two factors emerged with eigenvalues greater than 1.0, and examination of the scree plot suggested that this solution was best. Factor 1, Desire to Lose Weight, was composed of the Body Dissatisfaction (loading = .86) and Drive for Thinness (loading = .87) subscales on the EDI–II and desire to be thinner as reported on the Body Image Survey (loading = .70). Factor 2, Pathological Eating Behaviors, was composed of the Bulimia subscale on the EDI–II (loading = .53) and the Dieting (loading = .70), Bulimia/Food Preoccupation (loading = .90), and Oral Control (loading = .86) subscales on the EAT. We created two composites of eating pathology symptoms corresponding to the factors by calculating the unweighted mean of the *z* scores of pertinent measures for that factor. The correlation between the two factors in our sample was .39 ($p < .01$).

The measures of parenting practices were entered into a principal components analysis, and results yielded one composite of Punitive Parenting with an eigenvalue greater than 1 and loadings ranging from .58 to .79. Measures were *z* scored and combined by taking the unweighted mean.

Data Analytic Plan

To ascertain whether eating pathology—specified as adolescent BN symptoms and body image dissatisfaction—is related to childhood ADHD diagnosis, we performed analysis of variance procedures controlling for key covariates: age, IQ, baseline satisfaction with general physical appearance, and comorbid disorders, history of stimulant medication, baseline and concurrent BMI (and the change in BMI from childhood to adolescence), and early puberty. IQ and baseline comorbid disorders were included because they distinguished ADHD and comparison samples, and we sought to determine whether eating pathology was related specifically to ADHD diagnosis (independent of problems commonly associated with ADHD). We included medication use, BMI, early puberty, and age because of the theoretical rationale of why these variables would be associated with eating pathology. We included baseline measures of satisfaction with physical appearance and BMI as covariates in an attempt to account for some potential preexisting differences in eating pathology. Although we tested Age × ADHD interactions and effects of ethnicity, neither predicted eating pathology, so we have omitted them herein.

To test our first hypothesis, that girls with ADHD in childhood would be at higher risk than comparison girls for eating pathology in adolescence, we conducted a multivariate analysis of covariance (MANCOVA) with the self-report eating pathology composites as dependent variables, with inclusion of covariates. The independent variable was baseline ADHD-C and ADHD-I versus comparison status. Following a significant MANCOVA, we tested our second hypothesis that girls with ADHD-C would display higher levels of eating pathology than girls with ADHD-I by examining separate eating pathology outcomes via ANCOVAs plus Bonferroni post hoc comparisons of each group contrast. We performed parallel analyses with the dependent variable of parent report of total symptoms of BN. We next examined our hypothesis that childhood impulsivity would uniquely predict eating pathology by conducting partial correlations between impulsivity and eating pathology, controlling for inattention and hyperactivity. We tested our third hypothesis, that baseline parent–child and peer relationship problems would predict adolescent eating pathology, by conducting regressions predicting eating pathology in which we controlled for

ADHD status in Step 1, added the pertinent predictor in Step 2, and placed the interaction between ADHD and the predictor in Step 3. Finally, we tested associations of eating pathology with concurrent adolescent symptomatology by repeating MANCOVAs using adolescent ADHD diagnosis as the independent variable. We also ascertained correlations between concurrent (adolescent) internalizing or externalizing symptoms and eating pathology.

Results

Descriptive Statistics

Table 1 displays group mean scores on study variables. Girls with ADHD-C showed the highest levels of BN symptoms and body image dissatisfaction, followed by girls with ADHD-I and then comparison girls (see initial presentation in Hinshaw et al., 2006). Girls with ADHD-C similarly showed the highest levels of disruptive and internalizing problems, parent–child and peer relationship problems, and medication use, followed by girls with ADHD-I and then comparison girls. Effect sizes were small to medium between the ADHD-C and ADHD-I groups, medium to large between the ADHD-I and comparison groups, and large between the ADHD-C and comparison groups (see Table 1). Girls with both types of ADHD appeared to have equally higher adolescent BMIs and lower IQs relative to comparison girls. Groups did not differ in early puberty, baseline satisfaction with general physical appearance, or baseline BMI.

Childhood ADHD Status and Adolescent Eating Pathology

Childhood diagnostic status (ADHD-C, ADHD-I, or comparison) was significantly related to the two self-reported eating pathology composites in a multivariate analysis of variance, $F(4, 404) = 7.40$, $p < .001$. After adding the covariates of age, adolescent BMI, IQ, baseline satisfaction with physical appearance, baseline disruptive and internalizing comorbidity, early puberty, and stimulant medication use, childhood ADHD diagnosis continued to predict the eating pathology composites, $F(4, 372) = 2.78$, $p < .05$. The only significant covariate was concurrent adolescent BMI, $F(2, 186) = 38.75$, $p < .001$, which was positively associated with eating pathology.

Because childhood BMI was collected with two thirds of the sample (whereas all other covariates were collected with the full sample), we conducted a separate MANCOVA in which we inserted childhood BMI and adolescent BMI as covariates predicting the eating pathology composites. Childhood BMI was not a significant predictor of adolescent eating pathology, $F(2, 131) = 0.83$, $p > .10$, although adolescent BMI continued to be significant, $F(2, 131) = 17.89$, $p < .001$, as did the independent variable

Table 1
Comparison of Groups on Study Variables

Variable	ADHD-C		ADHD-I		Comparison		Effect sizes[a]			Omnibus tests[b]
	M	*SD*	*M*	*SD*	*M*	*SD*	1–3	2–3	1–2	
Baseline measures										
Peer rejection[c]	0.12_x	0.29	0.00_y	0.16	-0.13_y	0.12	1.13	0.92	0.51	$F(2, 225) = 32.57^{**}$
Punitive parenting[d]	0.26_x	0.72	-0.05_y	0.75	-0.27_y	0.63	0.78	0.32	0.42	$F(2, 200) = 12.03^{**}$
EE[e]	0.57_x	0.50	0.50_x	0.51	0.10_y	0.30	1.14	0.96	0.14	$F(2, 128) = 16.06^{**}$
Disruptive disorders[f]	$74\%_x$		$49\%_y$		$7\%_z$		37.80	12.76	2.97	$\chi^2(2, N = 227) = 83.67^{**}$
Internalizing disorders[f]	$29\%_x$		$21\%_x$		$3\%_y$		13.22	8.60	1.54	$\chi^2(2, N = 228) = 21.09^{**}$
Satisfaction with appearance[d]	−0.07	1.10	−0.17	1.18	0.17	0.75	−0.25	−0.34	0.09	$F(2, 225) = 2.15$
Body mass index[g]	19.01	6.21	19.27	7.63	17.71	3.70	0.25	0.26	−0.04	$F(2, 148) = 1.11$
Full Scale IQ	99.57_x	13.24	99.81_x	14.21	111.95_y	12.71	−0.95	−0.90	−0.02	$F(2, 223) = 23.01^{**}$
Follow-up measures										
Desire to lose weight[d]	0.27_x	1.08	0.04_{xy}	0.50	-0.27_y	0.54	0.63	0.60	0.27	$F(2, 203) = 9.09^{**}$
Pathological eating[d]	0.29_x	1.09	-0.16_y	0.32	-0.21_y	0.22	0.64	0.18	0.56	$F(2, 203) = 11.07^{**}$
BN symptom count[d, f]	0.25_x	1.25	0.08_{xy}	0.75	-0.22_y	0.73	0.46	0.21	0.31	$F(2, 204) = 4.92^{**}$
Externalizing symptoms[d]	0.26_x	0.70	0.06_x	0.42	-0.30_y	0.33	1.02	0.95	0.35	$F(2, 206) = 23.69^{**}$
Internalizing symptoms[d]	0.20_x	0.61	0.06_x	0.49	-0.23_y	0.47	0.79	0.60	0.25	$F(2, 206) = 13.95^{**}$
Body mass index[g]	22.66_x	5.57	23.26_x	7.83	20.81_x	3.90	0.38	0.40	−0.09	$F(2, 201) = 3.49^{*}$
Stimulant medication	$65\%_x$		$62\%_x$		$0\%_y$				1.14	$\chi^2(1, N = 199) = 82.72^{**}$
Early puberty	31%		41%		26%		1.28	1.98	0.65	$\chi^2(2, N = 209) = 3.21$

Note. ADHD-C = attention-deficit/hyperactivity disorder-combined type; ADHD-I = ADHD–inattentive type; EE = expressed emotion; BN = bulimia nervosa.

[a] Effect sizes: Cohen's *d* for continuous variables, odds ratios for categorical variables (those indicated by percentages in the first three columns of the table). 1 = ADHD-C; 2 = ADHD-I; 3 = comparison. [b] Omnibus group contrasts in this table are tested using one-way analyses of variance without covariates. Means are raw scores unadjusted for covariates. Means with different subscripts differ significantly, based on Bonferroni post hoc comparisons (alpha for each set = .05/3 or .0167) or 2 × 2 chi-square tests. [c] Proportion of negative nominations minus proportion of positive nominations in the classroom. [d] These are *z*-scored composites, where positive numbers correspond to greater amounts of the construct. [e] EE was assessed using parental criticism and overcontrol variables from the Five-Minute Speech Sample, collected with two thirds of the sample. [f] Reported by parent on the Diagnostic Interview Schedule for Children—4th ed. Disruptive behavior disorders = oppositional defiant disorder/conduct disorder. Internalizing disorders = depressive or anxiety disorders, beyond specific phobias. [g] Weight (kg) divided by square of height (m); body mass index at baseline (not follow up) was collected with two thirds of the sample.

$^{*}p < .05$. $^{**}p < .01$.

of childhood ADHD diagnosis, $F(4, 262) = 5.41$, $p < .001$, in this delimited sample. We also considered the change in BMI from childhood to adolescence by calculating the standardized residual of childhood BMI predicting adolescent BMI (which again could only be computed for two thirds of the sample). When we inserted this BMI change score in the MANCOVA, it did not predict the eating pathology composites, and the variable of childhood ADHD diagnosis again remained significant. Similarly, when we restricted the Harter measure assessing satisfaction with physical appearance to the version received by girls age 8 and above at baseline—because this version more closely assesses dislike of one's body shape and size than does the version for younger girls—the variable of childhood ADHD diagnosis again remained significant in the MANCOVA.

We followed the significant MANCOVA by conducting analyses of covariance (ANCOVAs) with each eating pathology composite as the dependent variable. We included the covariate of concurrent BMI because it was a significant predictor in the MANCOVA. Even though baseline satisfaction with physical appearance had not been a significant variable, we included it as a covariate as an attempt to control for some preexisting differences in eating pathology. The effect of childhood ADHD diagnosis was significant in these analyses: desire to lose weight, $F(2, 195) = 7.20$, $p < .001$, and pathological eating behaviors, $F(2, 195) = 10.43$, $p < .001$. We then reconducted analyses adding baseline BMI to the covariates of concurrent BMI and baseline satisfaction with physical appearance, even though baseline BMI had not been significant. Despite a reduction in sample size, the effect of childhood ADHD remained significant in predicting both desire to lose weight, $F(2, 131) = 6.75$, $p < .01$, and pathological eating behaviors, $F(2, 131) = 7.06$, $p < .001$. Similarly, we reconducted analyses adding the covariate of BMI change score (in place of baseline and follow-up BMI scores) to baseline satisfaction with physical appearance, and again the effect of childhood ADHD remained significant in predicting both desire to lose weight and pathological eating behaviors.

Because of the significant omnibus ANCOVAs, we next computed subgroup contrasts between ADHD-C, ADHD-I, and comparison groups using Bonferroni corrections. Table 1 displays the results of these contrasts, which are raw scores unadjusted for covariates. Girls with ADHD-C showed higher levels of desire to lose weight than did comparison girls, with a medium effect size ($d = .63$). The ADHD-I group was intermediate but not significantly different from either the ADHD-C or the comparison group. Girls with ADHD-C showed higher levels of pathological eating behaviors than did both girls with ADHD-I and comparison girls; the latter two groups did not differ. The effect size was medium for the ADHD-C contrast with the ADHD-I group ($d = .56$), and slightly larger in relation to the comparison group ($d = .64$).

Analyses were repeated using the BN symptom count from the parent-reported DISC–IV. No girl met diagnostic criteria for BN. Parent-reported symptoms correlated significantly with adolescent-reported desire to lose weight ($r = .42$, $p < .001$) and with pathological eating behaviors ($r = .38$, $p < .001$); these relationships are comparable to findings that the average correlation between multiple informants on behavior problems is approximately .30 (Achenbach, McConaughy, & Howell, 1987). In an ANCOVA with covariates of concurrent BMI and childhood satisfaction with general physical appearance, childhood ADHD diagnosis was related to the parent-reported BN symptom count, $F(2, 196) = 4.45$, $p < .05$. When including the covariate of childhood BMI with the covariates of concurrent BMI and childhood satisfaction with physical appearance, childhood ADHD diagnosis remained significant, despite reduced sample size, $F(2, 134) = 5.03$, $p < .01$. Similarly, adding the covariate of BMI change score from childhood to adolescence did not affect the statistical significance of childhood ADHD diagnosis in predicting parent-reported BN symptoms. Pairwise contrasts with a Bonferroni correction showed that the ADHD-C group was higher than the comparison group, with an effect size approaching medium ($d = .46$). The ADHD-I group was intermediate but not significantly different from either the ADHD-C or the comparison group.

Symptom Counts and Comparison to National Norms

As suggested by Aiken and West (1991), we had converted eating pathology measures to *z* scores before analyses. A downside of this approach is that it does not allow interpretation of clinical severity. A minority of girls, about 5%–10% of the ADHD-C group relative to 0%–1% of the ADHD-I and comparison groups, showed clinically concerning eating pathology. Parents reported on the DISC–IV that 8% of girls with ADHD-C had engaged in at least one *DSM–IV*–defined binge-eating episode in the past year and 1% had engaged in inappropriate purging to prevent weight gain, relative to 0% of girls with ADHD-I and comparison girls for either symptom. Parents also reported that 6% of girls with ADHD-C, 5% of girls with ADHD-I, and 0% of comparison girls had their self-evaluation unduly influenced by weight and shape.

Regarding the self-report measures, 10% of girls in the ADHD-C group surpassed the clinical cutoff total score of 20 on the EAT (Garner et al., 1982), as opposed to 0% in the ADHD-I and comparison groups. A similar pattern was found on the EDI–II, on which 9% of girls with ADHD-C fell over the 90th percentile relative to national age norms on the Drive for Thinness, Bulimia, and/or Body Dissatisfaction subscales, as opposed to 2% with ADHD-I and 0% of comparison girls.

Other Childhood Predictors

We examined relative contributions of childhood symptoms of inattention, hyperactivity, and impulsivity by taking means of each symptom cluster on the parent-reported SNAP–IV, allowing investigation of continuous symptom variability across the entire sample. As expected, inattention, hyperactivity, and impulsivity symptoms were highly correlated with one another (rs = .71–.86, all ps $< .001$). Still, impulsivity symptoms predicted desire to lose weight ($r = .17$, $p < .05$) and pathological eating behaviors ($r = .13$, $p < .05$) in partial correlations controlling for both inattention and hyperactivity symptoms. It was never the case that either inattention or hyperactivity predicted eating pathology with control of impulsivity.

We next tested childhood parenting practices and peer rejection as predictors of adolescent eating pathology, as shown in Table 2. In total, we conducted six regressions: For each of the two adolescent self-report eating pathology composites, we included models in which the main predictor was (a) punitive parenting, (b) parental EE, and (c) peer rejection. After controlling for childhood ADHD status, punitive parenting predicted higher levels of patho-

Table 2
Effects of Peer and Family Factors on Eating Pathology

Variable	Total R^2	ΔR^2	*B*	*SE B*	β
Dependent variable: Follow-up desire to lose weight					
1. Childhood ADHD diagnosis	.08	.08***	.26	.06	.29
2. Childhood peer rejection	.12	.04**	.84	.27	.23
3. Interaction between 1 and 2	.12	.00	.08	.36	.04
Dependent variable: Follow-up pathological eating behaviors					
1. Childhood ADHD diagnosis	.08	.08***	.26	.06	.29
2. Childhood punitive parenting	.11	.03*	.19	.08	.18
3. Interaction between 1 and 2	.14	.03**	.24	.09	.31
Dependent variable: Follow-up pathological eating behaviors					
1. Childhood ADHD diagnosis	.13	.13***	.39	.10	.35
2. Childhood EE[a]	.19	.06**	.56	.20	.27
3. Interaction between 1 and 2	.19	.01	.59	.58	.28

Note. ADHD = attention-deficit/hyperactivity disorder; EE = expressed emotion. ΔR^2 effect size conventions: .01 = small, .06 = medium, and .14 = large (Cohen, Cohen, West, & Aiken, 2003).
[a] As measured from the Five-Minute Speech Sample, assessing parental criticism and overcontrol.
* $p < .05$. ** $p < .01$. *** $p < .001$.

logical eating behaviors. Furthermore, there was a significant interaction between punitive parenting and ADHD diagnosis. Probing revealed that whereas punitive parenting did not predict pathological eating behaviors for the girls in the comparison ($B = -0.11$, $p > .10$) sample, there were positive associations between the two constructs in the ADHD-I ($B = 0.20$, $p > .10$) and ADHD-C ($B = 0.26$, $p < .05$) samples.

The observational measure of EE predicted higher levels of pathological eating behaviors, controlling for childhood ADHD diagnostic status. Childhood peer rejection predicted higher levels of desire to lose weight, with control of childhood ADHD status. Neither interactions between EE and ADHD nor interactions between peer rejection and ADHD were significant.

Of note, we reconducted regressions reversing the order of predictors: We placed the parent–child or peer relationship problems as predictors on Step 1 and ADHD on Step 2. In all cases, ADHD was significant in predicting eating pathology outcomes in these analyses, suggesting that the correlation between ADHD and eating pathology is not driven by the shared relationship problems in both disorders.

Concurrent Symptomatology

We investigated the association between concurrent adolescent ADHD status and eating pathology. Most comparison girls at baseline had maintained comparison status at follow-up (95%). In contrast, 63% of the ADHD-I sample remained so at follow-up, with most of the remaining 37% changed to comparison status. Only 39% of the ADHD-C sample remained so at follow-up, with the majority changed to comparison or ADHD-I status (see Hinshaw et al. 2006). We removed from the analysis girls who had been diagnosed with ADHD-C or ADHD-I at baseline and had converted to the comparison group at follow-up, leaving a purer contrast between adolescent ADHD-C and ADHD-I groups with a comparison group of girls who had never met criteria for ADHD. Results replicated the pattern from the baseline diagnostic groups: Adolescent ADHD status predicted eating pathology variables in a MANCOVA with covariates of adolescent BMI and childhood body image dissatisfaction, $F(4, 314) = 4.43$, $p < .01$, with the ADHD-C group at highest risk, followed by the ADHD-I group, and then the comparison group. Effect sizes were similar to those using baseline diagnosis.

Finally, we examined correlations between eating pathology and concurrent internalizing and externalizing symptomatology. Externalizing problems were correlated at low and nonsignificant levels with desire to lose weight ($r = .12$, $p = .10$) and pathological eating behaviors ($r = .10$, $p > .10$). Internalizing problems, however, were significantly correlated with desire to lose weight ($r = .31$, $p < .001$) and pathological eating behaviors ($r = .22$, $p < .01$). Tests for the significance of difference between dependent correlations revealed that the correlation between desire to lose weight and internalizing problems was greater than the correlation between desire to lose weight and externalizing problems, $t(203) = 2.88$, $p < .001$. Similarly, pathological eating behaviors were more strongly correlated with internalizing problems than with externalizing problems, $t(203) = 1.79$, $p < .05$.

Discussion

We found evidence that girls with ADHD-C in childhood are at risk for self-reported, dimensional BN behaviors and body image dissatisfaction in adolescence, as well as parent-reported BN symptoms, relative to comparison girls. This finding survived stringent statistical control of age, baseline comorbid disruptive and internalizing disorders, a baseline measure of satisfaction with general physical appearance, Full Scale IQ, early puberty, baseline and concurrent BMI, BMI change from childhood to adolescence, and history of stimulant medication use (as well as Bonferroni corrections for multiple comparisons). Effect sizes for ADHD-C versus comparison contrasts were in the medium to large range. Girls with ADHD-I consistently fell between the ADHD-C and comparison samples in eating pathology; their scores were typically closer to those of the comparison girls than to those of the girls with ADHD-C. Effect sizes between the ADHD-C and ADHD-I groups were medium.

We also found that childhood impulsivity symptoms best predicted adolescent eating pathology, above effects of inattention and hyperactivity. We found some evidence for unique contributions of childhood peer and parent–child relationship problems in predicting adolescent eating pathology. Peer rejection predicted increased desire to lose weight after controlling for ADHD. Punitive parenting (punishment-oriented and harsh discipline practices) as well as observed EE (parental criticism and overcontrol) predicted adolescent pathological eating behaviors, particularly for girls with ADHD-C ($B = 0.26$, $p < .05$); a similar although nonsignificant relationship was found for girls with ADHD-I ($B = 0.20$, $p > .10$). The relationship between parenting and eating pathology was nonsignificant for comparison girls ($B = -0.11$, $p > .10$).

Concurrent associations between adolescent ADHD status (particularly ADHD-C) and eating pathology were found in contrast to a restricted comparison group of girls who had never met criteria for ADHD. In addition, we found moderate correlations between

adolescent internalizing symptoms and eating pathology but only modest (and significantly weaker) relationships between adolescent externalizing symptoms and eating pathology.

Overall, girls with ADHD-C appeared different from, and more impaired than, girls with ADHD-I in eating pathology. Yet both girls with ADHD-C and girls with ADHD-I tended to have equally higher adolescent BMIs than did comparison girls. At follow-up, comparison girls were near the 50th percentile and girls with ADHD were near the 80th percentile in age-normed BMI (Hammer, Kraemer, Wilson, Ritter, & Dornbusch, 1991). Our finding that girls with ADHD had inflated BMIs is consistent with preliminary evidence from the Multimodal Treatment of Children With ADHD study (Swanson et al., 2007) in which youths with ADHD (80% male) had greater weight and height relative to age norms. Nonetheless, in our study girls with ADHD-I did not engage in eating pathology behaviors in response to their weight to the same extent as did girls with ADHD-C. It may be that inattention and disorganization around healthy eating, common in both ADHD-I and ADHD-C, leads to increased BMI in adolescence; notably, ADHD-C, ADHD-I, and comparison groups did not differ in childhood BMI. However, the impulsivity characteristic of ADHD-C may be the key contributor to disordered and pathological BN behaviors. Results from the symptom analyses confirm this hypothesis, in that high childhood impulsivity (but not inattention or hyperactivity) uniquely contributed to adolescent eating pathology. This finding supports the theoretical argument that the lack of self-regulation in ADHD-C and BN may link these two types of psychopathology.

Another potential pathway between childhood ADHD and adolescent BN eating pathology may involve a shared dysregulated temperament present at birth before symptoms of either ADHD or eating pathology develop. Evidence suggests that children with ADHD-C in particular have trouble with accurately perceiving and regulating their own emotional states (Maedgen & Carlson, 2000). Individuals with BN display similar emotion-processing and emotion-regulation problems (Bydlowski, Corcos, & Jeammet, 2005). It may be that when girls with a dysregulated temperamental style enter adolescence and experience pressures for thinness, they are unable to cope effectively and are prone to turn to bingeing and purging behaviors. Eating pathology may be most likely to result for dysregulated girls if they lack supportive parent–child or peer relationships to buffer against stressors. This pattern is supported by our finding that negative parenting in childhood more strongly predicted pathological eating behaviors for girls with ADHD than for comparison girls.

A strength of our study is the use of observational measures of parent–child and peer relationship problems in childhood to prospectively predict risk for eating pathology in adolescence. The majority of existing research has relied on retrospective reports and/or self-report (not observer-recorded) measures of relationship problems.

One limitation to this research is that the Harter measure of satisfaction with general physical appearance at baseline is not fully consistent with the adolescent measures of desire to lose weight from the EDI–II. The baseline measure asks about satisfaction with appearance in a global manner, whereas the follow-up measures ask specifically about dissatisfaction with the large size of particular body parts such as thighs or stomach. In addition, we did not have baseline measures of disordered eating, as captured at follow-up, or baseline measures of other constructs found to be linked to BN, such as overconcern with body weight. Thus, there may be preexisting differences in eating pathology at baseline that are not fully controlled for in our analyses. Although (a) the young age and prepubertal status of the girls at baseline and (b) the lack of ADHD–comparison group differences on the baseline Harter measure and childhood BMI both increase confidence in our assertion that ADHD status may prospectively predict increases in adolescent eating pathology, future research should include better measures to assess BN and dissatisfaction with specific body parts in childhood. Furthermore, our dichotomous early puberty and stimulant medication usage measures are limited in sensitivity, and future studies might include more comprehensive assessment of these variables.

Another limitation is that the mean age at follow-up (14.2 years; range = 11–18) of the sample is below the peak incidence of BN diagnoses. Perhaps this factor is why no girl in this study met criteria for clinical diagnoses of BN. Yet the "right-censored" nature of our follow-up data also place a conservative bias with respect to predictions to eating pathology.

Conceptually, although it is standard in the ADHD literature to control for disruptive and internalizing comorbidities, so as to assess the independent contribution of ADHD apart from these other problems, we also note that this practice may constitute statistical overcontrol (Miller & Chapman, 2001). Future investigations should consider this point in analyses.

In conclusion, we contend that BN symptoms and body image dissatisfaction should be incorporated into conceptions of risk and impairment among youths with ADHD. The strong predominance of males in the study of ADHD to date has restricted understanding and investigation of female-relevant domains of impairment such as eating pathology. A fuller understanding of the sequalae associated with ADHD in girls is essential for both theoretical elucidation and treatment applications.

References

Achenbach, T. M. (1991a). *Manual for Child Behavior Checklist and Revised Child Behavior Profile.* Burlington, VT: University Associates in Psychiatry.

Achenbach, T. M. (1991b). *Manual for Teacher's Report Form and 1991 Profile.* Burlington, VT: University Associates in Psychiatry.

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin, 101,* 213–232.

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions.* Thousand Oaks, CA: Sage.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Asarnow, J. R. (1988). Peer status and social competence in child psychiatric inpatients: A comparison of children with depressive, externalizing, and concurrent depressive and externalizing disorders. *Journal of Abnormal Child Psychology, 16,* 151–162.

Barkley, R. A. (2002). ADHD: Long-term course, adult outcome, and comorbid disorders. In P. S. Jensen & J. R. Cooper (Eds.), *Attention-deficit/hyperactivity disorder: State of the science and best practices* (pp. 4-1–4-12). Kingston, NJ: Civic Research Institute.

Barkley, R. A. (2003). Attention-deficit/hyperactivity disorder. In E. J.

Mash & R. A. Barkley (Eds.), *Child psychopathology* (2nd ed., pp. 75–143). New York: Guilford Press.

Barkley, R. A. (2004). Adolescents with attention-deficit/hyperactivity disorder: An overview of empirically based treatments. *Journal of Psychiatric Practice, 10,* 39–56.

Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2002). The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *Journal of Abnormal Psychology, 111,* 279–289.

Bearman, S. K., Presnell, K., Martinez, E., & Stice, E. (2006). The skinny on body dissatisfaction: A longitudinal study of adolescent girls and boys. *Journal of Youth and Adolescence, 35,* 229–241.

Biederman, J., Faraone, S. V., Mick, E., Williamson, S., Wilens, T. E., Spencer, T. J., et al. (1999). Clinical correlates of ADHD in females: Findings from a large group of girls ascertained from pediatric and psychiatric referral sources. *Journal of the American Academy of Child & Adolescent Psychiatry, 38,* 966–975.

Biederman, J., Monuteaux, M., Mick, E., Spencer, T. J., Wilens, T. E., Klein, K. L., et al. (2006). Psychopathology in females with attention-deficit/hyperactivity disorder: A controlled, five-year prospective study. *Biological Psychiatry, 60,* 1098–1105.

Biederman, J., Newcorn, J., & Sprich, S. (1991). Comorbidity of attention deficit hyperactivity disorder with conduct, depressive, anxiety, and other disorders. *American Journal of Psychiatry, 148,* 564–577.

Blachman, D. R., & Hinshaw, S. P. (2002). Patterns of friendship among girls with and without attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology, 30,* 625–640.

Butzlaff, R. L., & Hooley, J. M. (1998). Expressed emotion and psychiatric relapse. *Archives of General Psychiatry, 55,* 547–552.

Bydlowski, S., Corcos, M., & Jeammet, P. (2005). Emotion-processing deficits in eating disorders. *International Journal of Eating Disorders, 37,* 321–329.

Cohen, J., Cohen, P., West, S. G., & Aiken, L. (2003). *Applied multiple regression/correlation analyses for the behavioral sciences* (3rd ed.). Mahwah, NJ: Erlbaum.

Coie, J. D., Dodge, K. A., & Coppotelli, H. (1982). Dimensions and types of social status: A cross-age perspective. *Developmental Psychology, 18,* 557–570.

Elliott, D. S., Huizinga, D., & Ageton, S. (1985). *Explaining delinquency and drug use.* Thousand Oaks, CA: Sage.

Fahy, T., & Eisler, I. (1993). Impulsivity and eating disorders. *British Journal of Psychiatry, 162,* 193–197.

Fairburn, C. G., Doll, H. A., Welch, S. L., Hay, P. J., Davies, B. A., & O'Connor, M. E. (1998). Risk factors for binge eating disorder. *Archives of General Psychiatry, 55,* 425–432.

Fallon, A. E., & Rozin, P. (1985). Sex differences in perceptions of desirable body shape. *Journal of Abnormal Psychology, 94,* 102–105.

Garner, D. M. (1991). *Eating Disorder Inventory* (2nd ed.). Odessa, FL: Psychological Assessment Resources.

Garner, D. M., & Garfinkel, P. E. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. *Psychological Medicine, 9,* 273–279.

Garner, D. M., Olmstead, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The Eating Attitudes Test: Psychometric features and clinical correlates. *Psychological Medicine, 12,* 871–878.

Hammer, L. D., Kraemer, H. C., Wilson, D. M., Ritter, P. L., & Dornbusch, S. L. (1991). Standardized percentile curves of body mass index for children and adolescents. *American Journal of Disease of Child, 145,* 259–263.

Harter, S. (1985). *Manual for the Self-Perception Profile for Children.* Unpublished manuscript, University of Denver.

Heming, G., Cowan, P. C., & Cowan, C. P. (1990). Ideas about parenting. In J. Touliatos, P. F. Perlmutter, & M. A. Straus (Eds.), *Handbook of family measurement techniques* (pp. 382–383). Thousand Oaks, CA: Sage.

Herzog, D. B., Nussbaum, K. M., & Marmor, A. K. (1996). Comorbidity and outcome in eating disorders. *Psychiatric Clinics of North America, 19,* 844–859.

Hinshaw, S. P. (2002). Preadolescent girls with attention-deficit/hyperactivity disorder: I. Background characteristics, comorbidity, cognitive and social functioning, and parenting practices. *Journal of Consulting and Clinical Psychology, 70,* 1086–1098.

Hinshaw, S. P., & Blachman, D. R. (2005). Attention-deficit/hyperactivity disorder in girls. In D. Bell, S. Foster, & E. J. Mash (Eds.), *Handbook of behavioral and emotional problems in girls* (pp. 117–148). New York: Kluwer Academic/Plenum.

Hinshaw, S. P., March, J. S., Abikoff, H., Arnold, L. E., Cantwell, D. P., Conners, C. K., et al. (1997). Comprehensive assessment of childhood attention-deficit hyperactivity disorder in the context of a multisite, multimodal clinical trial. *Journal of Attention Disorders, 1,* 217–234.

Hinshaw, S. P., Owens, E. B., Sami, N., & Fargeon, S. (2006). Prospective follow-up of girls with attention-deficit/hyperactivity disorder into adolescence: Evidence for continuing cross-domain impairment. *Journal of Consulting and Clinical Psychology, 74,* 489–499.

Hinshaw, S. P., Zupan, B. A., Simmel, C., Nigg, J. T., & Melnick, S. M. (1997). Peer status in boys with and without attention-deficit hyperactivity disorder: Predictions from overt and covert antisocial behavior, social isolation, and authoritative parenting beliefs. *Child Development, 64,* 880–896.

Hodes, M., & le Grange, D. (1993). Expressed emotion in the investigation of eating disorders: A review. *International Journal of Eating Disorders, 13,* 279–288.

Hoza, B., Mrug, S., Gerdes, A. C., Bukowski, W. M., Kraemer, H. C., Wigal, T., et al. (2005). What aspects of peer relationships are impaired in children with attention-deficit/hyperactivity disorder? *Journal of Consulting and Clinical Psychology, 73,* 411–423.

Jacobi, C., Hayward, C., de Zwaan, M., Kraemer, H. C., & Agras, W. S. (2004). Coming to terms with risk factors for eating disorders: Application of risk terminology and suggestions for a general taxonomy. *Psychological Bulletin, 130,* 19–65.

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2002). Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *American Journal of Psychiatry, 159,* 394–400.

Johnston, C., & Mash, E. J. (2001). Families of children with attention-deficit/hyperactivity disorder: Review and recommendations for future research. *Clinical Child and Family Psychology Review, 4,* 183–207.

Kaltiala-Heino, R., Marttunen, M., Rantanen, P., & Rimpela, M. (2003). Early puberty is associated with mental health problems in middle adolescence. *Social Science and Medicine, 57,* 1055–1074.

Kaufman, A. S. (1993). King WISC the third assumes the throne. *Journal of School Psychology, 31,* 345–354.

Keski-Rahkonen, A., Bulik, C. M., Neale, B. M., Rose, R. J., Rissanen, A., & Kaprio, J. (2005). Body dissatisfaction and drive for thinness in young adult twins. *International Journal of Eating Disorders, 37,* 188–199.

Keys, A., Fidanza, F., Karvonen, M. J., Kimura, N., & Taylor, H. L. (1972). Indices of relative weight and obesity. *Journal of Chronic Disease, 25,* 329–343.

Killen, J. D., Taylor, C. B., Hayward, C., Haydel, K. F., Hamer, L. D., Robinson, T. N., et al. (1994). Pursuit of thinness and onset of eating disorder symptoms in a community sample of adolescent girls. *International Journal of Eating Disorders, 16,* 227–238.

Kovacs, M. (1992). *Children's Depression Inventory (CDI) manual.* Toronto: Multi-Health Systems.

Lahey, B. B., Applegate, B., McBurnett, K., Biederman, J., Greenhill, L. L., Hynd, G. W., et al. (1994). DSM-IV field trials for attention

deficit/hyperactivity disorder in children and adolescents. *American Journal of Psychiatry, 151,* 1673–1685.

Latimer, W. W., August, G. J., Newcomb, M. D., Realmuto, G. M., Hektner, J. M., & Mahty, R. M. (2003). Child and familial pathways to academic achievement and behavioral adjustment: A prospective six-year study of children with and without ADHD. *Journal of Attention Disorders, 7,* 101–116.

Leon, G. R., Fulkerson, J. A., Perry, C. L., Keel, P. K., & Klump, K. L. (1998). Three to four year prospective evaluation of personality and behavioral risk factors for later disordered eating in adolescent girls and boys.

Lijffijt, M., Kenemans, J., Verbaten, M., & van Engeland, H. (2005). A meta-analytic review of stopping performance in attention-deficit/hyperactivity disorder: Deficient inhibitory motor control? *Journal of Abnormal Psychology, 114,* 216–222.

Maedgen, J. W., & Carlson, C. L. (2000). Social functioning and emotional regulation in the attention deficit hyperactivity disorder subtypes. *Journal of Clinical Child Psychology, 29,* 30–42.

Mannuzza, S., & Klein, R. G. (2000). Long term prognosis in attention-deficit/hyperactivity disorder. *Child and Adolescent Psychiatric Clinics of North America, 9,* 711–726.

Marchi, M., & Cohen, P. (1990). Early childhood eating behaviors and adolescent eating disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 29,* 112–117.

Marshal, M. P., Molina, B. S. G., & Pelham, W. E. (2003). Childhood ADHD and adolescent substance use: An examination of deviant peer group affiliation as a risk factor. *Psychology of Addictive Behaviors, 17,* 293–302.

Mattos, P., Saboya, E., Ayrao, V., Segrenreich, D., Duchesne, M., & Coutinho, G. (2004). Comorbid eating disorders in a Brazilian attention-deficit/hyperactivity disorder adult clinical sample. *Revista Brasileira de Psiquiatria, 26,* 248–250.

Mikami, A. Y., & Hinshaw, S. P. (2006). Resilient adolescent adjustment among girls: Buffers of childhood peer rejection and attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology, 26,* 823–837.

Miller, G. A., & Chapman, J. P. (2001). Misunderstanding analysis of covariance. *Journal of Abnormal Psychology, 110*, 40–48.

Nederkoorn, C., Van Eijs, Y., & Jansen, A. (2004). Restrained eaters act on impulse. *Personality and Individual Differences, 37,* 1651–1658.

Pajer, K. A. (1998). What happens to "bad" girls? A review of the adult outcomes of antisocial adolescent girls. *American Journal of Psychiatry, 155,* 862–870.

Peris, T. S., & Hinshaw, S. P. (2003). Family dynamics and preadolescent girls with ADHD: The relationship between expressed emotion, ADHD symptomatology, and comorbid disruptive behavior. *Journal of Child Psychology and Psychiatry, 44,* 1177–1190.

Polivy, J., & Herman, C. P. (2002). Causes of eating disorders. *Annual Review of Psychology, 53,* 187–213.

Schweickert, L. A., Strober, M., & Moskowitz, A. (1997). Efficacy of methylphenidate in bulimia nervosa comorbid with attention-deficit/hyperactivity disorder: A case report. *International Journal of Eating Disorders, 21,* 299–301.

Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children, Version IV: Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child & Adolescent Psychiatry, 39,* 28–38.

Shelton, K. K., Frick, P. J., & Wootton, S. (1996). Assessing the parenting practices in families of elementary school-age children. *Journal of Clinical Child Psychology, 25,* 317–329.

Shore, R. A., & Porter, J. E. (1990). Normative and reliability data for 11 to 18 year olds on the Eating Disorder Inventory. *International Journal of Eating Disorders, 9,* 201–207.

Sokol, M. S., Gray, N. S., Goldstein, A., & Kaye, W. H. (1999). Methylphenidate treatment for bulimia nervosa associated with a cluster B personality disorder. *International Journal of Eating Disorders, 25,* 233–237.

Stice, E., Agras, W. S., & Hammer, L. D. (1999). Risk factors for the emergence of childhood eating disturbances: A five-year prospective study. *International Journal of Eating Disorders, 25,* 375–387.

Stice, E., Presnell, K., & Bearman, S. K. (2001). Relation of early menarche to depression, eating disorders, substance abuse, and comorbid psychopathology among adolescent girls. *Developmental Psychology, 37,* 608–619.

Stice, E., Spangler, D., & Agras, W. S. (2001). Exposure to media-portrayed thin-ideal images adversely affects vulnerable girls: A longitudinal experiment. *Journal of Social and Clinical Psychology, 20,* 270–288.

Striegel-Moore, R. H., Dohm, F., Pike, K. M., Wilfley, D. E., & Fairburn, C. G. (2002). Abuse, bullying, and discrimination as risk factors for binge eating disorder. *American Journal of Psychiatry, 159,* 1902–1907.

Strober, M., & Humphrey, L. L. (1987). Familial contributions to the etiology and course of anorexia nervosa and bulimia. *Journal of Consulting and Clinical Psychology, 55,* 654–659.

Surman, C. B. H., Randall, E. T., & Biederman, J. (2006). Association between attention-deficit/hyperactivity disorder and bulimia nervosa: Analysis of 4 case-control studies. *Journal of Clinical Psychiatry, 67,* 351–354.

Swanson, J. M. (1992). *Assessment and treatment of ADD students.* Irvine, CA: K. C. Press.

Swanson, J. M., Elliot, G. R., Greenhill, L. L., Wigal, T., Arnold, L. E., Vitiello, B., et al. (2007). Effects of stimulant medication on growth rates across 3 years in the MTA follow-up. *Journal of the American Academy of Child & Adolescent Psychiatry, 46,* 1015–1027.

Sweeting, H., & West, P. (2002). Gender differences in weight related concerns in early to late adolescence. *Journal of Epidemiology and Community Health, 56,* 701–702.

Thompson, B. (2000). Ten commandments of structural equation modeling. In L. G. Grimm & P. R. Yarnold (Eds.), *Reading and understanding more multivariate statistics* (pp. 261–284). Washington, DC: American Psychological Association.

van Furth, E. F., van Strien, D. C., & Martina, L. M. L. (1996). Expressed emotion and the prediction of outcome in adolescent eating disorders. *International Journal of Eating Disorders, 20,* 19–31.

Wechsler, D. (1991). *Manual for the Wechsler Intelligence Scale for Children* (3rd ed.). New York: Psychological Corporation/Harcourt Brace.

Weiss, G., & Hechtman, L. T. (1993). *Hyperactive children grown up* (2nd ed.). New York: Guilford Press.

Wiederman, M. W., & Pryor, T. (1996). Multi-impulsivity among women with bulimia nervosa. *International Journal of Eating Disorders, 20,* 359–365.

Wonderlich, S. A., Connolly, K. M., & Stice, E. (2004). Impulsivity as a risk factor for eating disorder behavior: Assessment implications with adolescents. *International Journal of Eating Disorders, 36,* 172–182.

Received June 26, 2006
Revision received July 5, 2007
Accepted July 17, 2007 ■