

# Are Girls with ADHD at Risk for Eating Disorders? Results from a Controlled, Five-Year Prospective Study

Joseph Biederman, MD,\*† Sarah W. Ball, SCD,\* Michael C. Monuteaux, SCD,\*†  
Craig B. Surman, MD,\*† Jessica L. Johnson, BS,\* Sarah Zeitlin, BA\*

**ABSTRACT:** *Objective:* To evaluate the association between attention-deficit/hyperactivity disorder (ADHD) and eating disorders in a large adolescent population of girls with and without ADHD. *Method:* We estimated the incidence of lifetime eating disorders (either anorexia or bulimia nervosa) using Cox proportional hazard survival models. Comparisons between ADHD girls with and without eating disorders were then made on measures of comorbidity, course of ADHD, and growth and puberty. *Results:* ADHD girls were 3.6 times more likely to meet criteria for an eating disorder throughout the follow-up period compared to control females. Girls with eating disorders had significantly higher rates of major depression, anxiety disorders, and disruptive behavior disorder compared to ADHD girls without eating disorders. Girls with ADHD and eating disorders had a significantly earlier mean age at menarche than other ADHD girls. No other differences in correlates of ADHD were detected between ADHD girls with and without eating disorders. *Conclusions:* ADHD significantly increases the risk of eating disorders. The presence of an eating disorder in girls with ADHD heightens the risk of additional morbidity and dysfunction.

(*J Dev Behav Pediatr* 28:302-307, 2007) *Index terms:* attention-deficit/hyperactivity disorder, eating disorders, longitudinal.

Recent work by Surman et al.<sup>1</sup> suggested an association between attention-deficit/hyperactivity disorder (ADHD) and eating disorders. Several case reports describe women with bulimia nervosa and ADHD-like symptoms.<sup>2-5</sup> Other researchers report an association between impulsive symptoms and bulimia nervosa, with findings indicating that measures of impulsivity correlate with severity of bulimia nervosa.<sup>6-11</sup> In one of the largest studies of its kind, Surman et al.<sup>1</sup> reported a significant association between ADHD and bulimia nervosa in a large sample of adult females with and without ADHD. Although eating disorders often start in childhood and adolescence, there is almost no information on the association between eating disorders and ADHD in pediatric samples.

Whether an association exists between ADHD and eating disorders has important implications. Considering that ADHD and eating disorders respond to different pharmacological and nonpharmacological treatments, diagnosing ADHD in patients with eating disorders could lead to new therapeutic opportunities.

The first aim of this study, therefore, was to systematically evaluate the association between ADHD and eating

disorders in a large adolescent population of girls with and without ADHD. Our second aim was to describe any clinical and demographic features that may be unique to the co-occurrence of ADHD and eating disorders in girls. We therefore tested the hypothesis that ADHD will be associated with eating disorders and that those girls with both ADHD and eating disorders will exhibit distinct clinical features from those associated with ADHD alone. To the best of our knowledge, this is the first evaluation of the association between ADHD and eating disorders in a pediatric sample followed prospectively into adolescence.

## METHOD

### Participants

Data from a case-control family study of girls with ADHD were examined.<sup>12</sup> Detailed study methodology has been reported elsewhere.<sup>12</sup> Briefly, we identified 63 girls with ADHD from lists of consecutive patients in the pediatric psychiatry clinic at the Massachusetts General Hospital. Another 77 girls with ADHD were identified from lists of children having evidence of ADHD in the computerized medical records of a health maintenance organization (HMO). Within each setting, we selected normal comparison subjects from lists of outpatients at pediatric medical clinics (Massachusetts General Hospital, N = 55; HMO, N = 67). At baseline, all subjects ranged in age from 6 to 18. The mean age of ADHD and control subjects was 11.2 years and 12.2 years, respectively. At follow-up, study participants ranged in age from 10 to 25 years. ADHD subjects had a mean age of 16.4 years and the control subjects had a mean age of 17.1 years.

The study was approved by the institutional review board, and in all cases parents gave written informed

From the \*Clinical and Research Program in Pediatric Psychopharmacology, Massachusetts General Hospital, Boston, MA; †Department of Psychiatry, Harvard Medical School, Boston, MA.

Received July 2006; accepted December 2006.

This work was supported in part by a grant from USPHS (National Institute of Child Health and Human Development), 5R01 HD-36317-07 (J.B.).

Address for reprints: Joseph Biederman, M.D., Clinical and Research Program in Pediatric Psychopharmacology, Massachusetts General Hospital, Warren 705, 55 Fruit Street, Boston, MA 02114; e-mail: jbiederman@partners.org.

Copyright © 2007 Lippincott Williams & Wilkins

consent for participation; in addition, children signed a special assent form for participation in this study.

## Measures

As described elsewhere,<sup>12</sup> psychiatric assessments were obtained by highly trained and highly supervised raters who were blind to the clinical diagnosis of the child. Psychiatric assessments relied on the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version (K-SADS-E)<sup>13,14</sup> for subjects younger than 18 years of age and the Structured Clinical Interview for DSM-IV (SCID)<sup>15</sup>, (supplemented with modules from the K-SADS-E to assess childhood diagnoses) for subjects 18 years of age and older.<sup>13,14</sup> The SCID was also used for psychiatric assessments of the parents. Because this study had begun prior to the finalization of DSM-IV, our baseline assessment used DSM-III-R-based structured interviews, but we supplemented these with questions that would allow us to make DSM-IV diagnoses. Diagnoses of the subjects were based on independent interviews with the mothers and direct interviews with the children if they were older than 12 years of age. All assessments were made by raters who were blind to the youth's diagnosis (ADHD or non-ADHD control) and ascertainment site. Different interviewers met with mothers and youth in order to maintain blindness to case-control status and prevent information from one informant influencing the assessment of the other.

We considered a disorder positive if diagnostic criteria were unequivocally met in either the baseline or 5-year follow-up interview. Diagnostic uncertainties were resolved by a review committee of at least two board-certified child and adult psychiatrists who were blind to the subject's ascertainment group, the ascertainment site, all data collected from other family members, and all nondiagnostic data (e.g., socioeconomic status [SES]). We computed kappa coefficients of agreement by having experienced, board-certified child and adult psychiatrists and licensed clinical psychologists diagnose subjects from audiotaped interviews. Based on 500 assessments from interviews of children and adults, the median kappa coefficient was .98. Kappa coefficients for individual diagnoses included ADHD (0.88), anorexia nervosa (1.0), bulimia nervosa (.89), conduct disorder (1.0), major depression (1.0), mania (0.95), separation anxiety (1.0), agoraphobia (1.0), panic (.95), substance use disorder (1.0), and tics/Tourette's (0.89).

To increase the sensitivity in identifying cases of eating disorders, a subject was classified as having an eating disorder if full or subthreshold (more than half of the required symptoms for a full diagnosis) DSM criteria were met. Of the 20 subjects classified as having a lifetime eating disorder, 12 met subthreshold criteria for an eating disorder; four of the 20 subjects with a lifetime eating disorder also had a current subthreshold diagnosis of eating disorder.

We adopted this method to increase the sensitivity and capture subclinical cases that have a high likelihood of developing full DSM criteria for eating disorders in the future. We considered the loss of precision that may

result from assuming that subthreshold cases are equivalent to full cases to be less than the loss that would result from taking the overly conservative approach of relying on strict DSM criteria and assuming that subthreshold cases can be grouped together with girls without an eating disorder.

## Growth and Puberty Assessments

To assess pubertal stage, all girls ages 12–18 were asked their age at menarche. All probands were weighed and measured using the same scale. We used a Physician's Beam Scale (Detecto, Webb City, MO), a high-calibration scale with a height rod for precise measurement of height. Growth measurements were plotted on National Center for Health Statistics growth tables.<sup>16</sup> These growth charts are sex specific and standardized. Thus, they permit comparisons of growth deficit findings to normal population data. Based on the information collected on height and weight, the following measures were created.

### Age-corrected height

Height values were converted to a height *z* score, defined as the difference of an individual height from the mean height, for children of the same age and sex, divided by the SD of height for that subgroup, as detailed in prior publications.<sup>17–20</sup>

### Age- and height-corrected weight index

Weight was computed by examining the weight as the percentage of the expected (or standard) weight for height consistent with studies of malnutrition in Third World countries.<sup>21–27</sup> We used this measure to examine weight differences beyond any height effects that may exist. The expected weight for height was derived from standard growth tables.<sup>16</sup> The 50th percentile curve in the height growth chart was compared to the child's height. The height-age is the age at which the child's height is the same as the value on the 50th percentile curve on the height growth chart. The expected weight for height is the 50th percentile weight on the weight growth chart for the height-age just derived.

## Statistical Analysis

We compared the baseline characteristics of subjects who were and were not assessed at the 5-year follow-up separately in ADHD and control subjects. Among subjects who were followed up at the 5-year reassessment, we compared three groups: ADHD with and without an eating disorder and control subjects without an eating disorder on demographic factors. Analyses of demographic factors relied on logistic and linear regression for binary and dimensional variables, respectively.

To assess our first aim, we estimated the incidence of lifetime eating disorder (either anorexia or bulimia nervosa) using Cox proportional hazard survival models. Rates of eating disorder were defined as present at either assessment (baseline, 5-year follow-up) versus absent at both time points. The models used age one as the start of surveillance, the earliest age at onset as the survival time for cases, and the age at most recent interview as the time of censoring for noncases.

To assess our second aim, we compared girls with and without eating disorders on a range of pubertal and psy-

Eating Disorders in Girls  
with and without ADHD

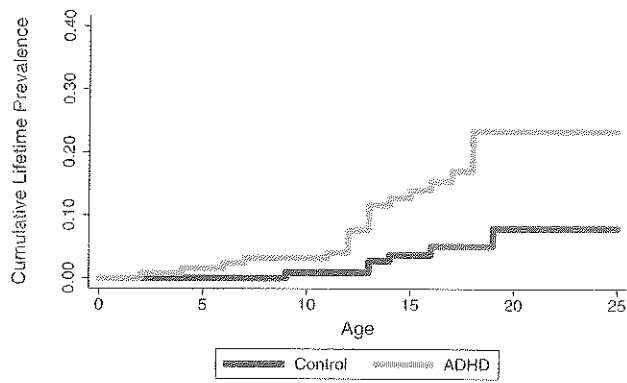


FIGURE 1. Risk of eating disorders by ADHD status.

chiatric outcomes. We used linear regression for continuous variables and logistic regression for binary variables, with the exception of treatment and SES, which were analyzed with the chi-square test and negative binomial regression, respectively. All statistical tests were two tailed, and alpha was set at .05.

## RESULTS

Of the 140 baseline attention-deficit/hyperactivity disorder (ADHD) subjects, 17 subjects did not have follow-up data, resulting in 123 ADHD subjects available for analysis. Of the 122 baseline control subjects, 10 did not have follow-up data. The rate of successful follow-up did not differ between the groups ( $\chi^2_1 = 1.1, p = .30$ ). Sixteen percent ( $n = 20$ ) of the ADHD sample had a history of anorexia or bulimia nervosa. Of those, 30% ( $n = 6$ ) reported anorexia, 50% ( $n = 10$ ) reported bulimia nervosa, and 20% ( $n = 4$ ) reported having experienced both anorexia and bulimia nervosa in different periods of their lifetimes, with a mean age at onset of 12.3 years. In the control group, 5% ( $n = 5$ ) reported a history of an eating disorder, of which 60% ( $n = 3$ ) reported anorexia, 40% ( $n = 2$ ) reported bulimia nervosa, and 20% ( $n = 1$ ) reported both anorexia and bulimia nervosa, with a mean age at onset of 14 years.

Examination of the Cox proportional hazards survival models (Fig. 1) indicated that ADHD females were 3.6 times more likely to meet criteria for an eating disorder (either bulimia nervosa or anorexia) compared to control females over the course of the follow-up period (hazard ratio = 3.6; 95% confidence interval: 1.4-9.9,  $p < .01$ ). Further, ADHD females were 5.6 times more likely to

meet criteria for bulimia nervosa compared to controls (hazard ratio = 5.6; 95% confidence interval: 1.6-19.0,  $p < .01$ ). Survival results for anorexia were not found to be statistically significant, despite a sizable hazard ratio (hazard ratio = 2.7; 95% confidence interval: 0.89-8.7,  $p = .09$ ).

Due to limited sample size, all subsequent analyses included ADHD subjects with either anorexia or bulimia nervosa for increased power. Also, due to the small number of eating disorder cases in the control group ( $n = 6$ ), control subjects with eating disorders were not included in the analyses. Thus, the control group consisted only of those subjects with neither ADHD nor an eating disorder ( $n = 106$ ). As shown in Table 1, there were no differences between ADHD and control girls in sociodemographic characteristics. Controls and subjects with ADHD and eating disorders had a mean age of 17 years at follow-up; ADHD subjects without eating disorders reported a mean age of 16 years. The majority of subjects in all groups (85%, 79%, and 85% of the control, ADHD, and ADHD with eating disorders groups, respectively) were in the high socioeconomic status (SES) category (an SES score of 1 or 2). Forty-three percent of controls were ascertained through the psychiatric referral source, as were 41% of the ADHD without eating disorders group and 60% of the ADHD with eating disorders group. Among the subjects with ADHD and eating disorders, 20% reported receiving counseling for their disorder, 10% received counseling and medication therapy, 50% had never received treatment, and for 20%, the treatment data were not available.

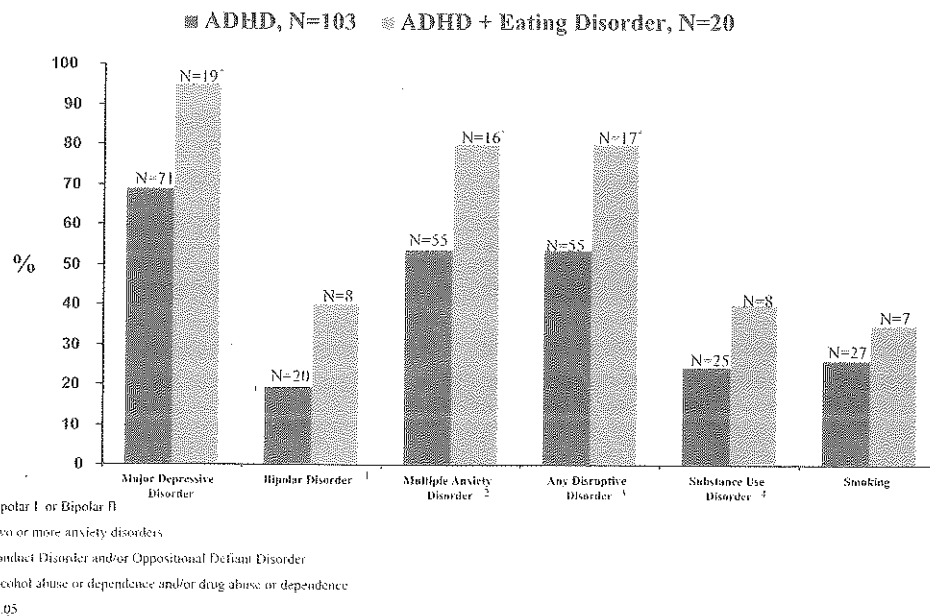
To assess our second hypothesis, we compared ADHD girls with and without eating disorders on rates of psychiatric comorbidity, growth outcomes, and correlates of ADHD. Girls with eating disorders had significantly higher lifetime rates of major depression, anxiety disorder, and disruptive behavior disorder compared to ADHD girls without eating disorders (Figure 2). There were no differences, however, between the groups on rates of bipolar disorder, substance use disorders, or smoking.

The mean age of onset of ADHD in girls with eating disorders was 4.9 years, which although not statistically significant, was somewhat younger than the mean age of onset of 5.3 years reported by girls without eating disorders ( $p = .24$ ). Those girls with eating disorders further reported a longer duration of ADHD than girls without eating disorders (10.8 years vs 9.4 years, respectively;  $p = .31$ ) as well as a higher average number of symptoms than those without eating disorders (13.5 vs 12.3, respectively;

Table 1. Demographic Characteristics of Girls with and without ADHD and Eating Disorders

	Controls (n = 106)	ADHD (n = 103)	ADHD + Eating Disorder (n = 20)	Statistical Significance
Age, yr	17.0 ± 3.0	16.2 ± 3.8	17.1 ± 3.4	$F_{1,227} = .75, p = .38$
SES <sup>a</sup>	1.8 ± 0.9	1.9 ± 1.0	1.7 ± 0.7	$z = .27, p = .79$
Psychiatric referral <sup>b</sup>	46 (43.4)	42 (40.8)	12 (60.0)	$\chi^2_2 = 2.5, p = .28$

Data in table presented as means ± SD or frequency (percentage). ADHD, attention-deficit/hyperactivity disorder; SES, socioeconomic status. <sup>a</sup>Social class defined in categories ranging from 1 through 5; 1 indicates most affluent social class and 5 indicates least affluent. <sup>b</sup>Subjects referred from a pediatric psychiatric clinic versus those subjects referred from a pediatric medical clinic.



**FIGURE 2.** Rates of comorbid disorders by ADHD status.

$p = .15$ ), although these differences were not statistically significant. Girls with ADHD and eating disorders had a significantly earlier mean age at onset of menarche than other ADHD girls (11.4 years vs 12.8 years, respectively;  $p < .05$ ). No differences were found, however, in terms of age-corrected height or weight index (Table 2). The average age-corrected height  $z$  score was 0.189 in controls, 0.160 in girls with ADHD but without eating disorders, and 0.343 in girls with ADHD and eating disorders. The average weight index of the controls was 1.14. The ADHD groups with and without eating disorders had average weight indexes of 1.24 and 1.10, respectively. For those girls with a current diagnosis of bulimia, the average weight index was 1.35.

## DISCUSSION

This study examined the association between attention-deficit/hyperactivity disorder (ADHD) and eating disorders in a large group of ADHD girls followed prospectively into adolescence. We found that adolescent females with ADHD were at an elevated risk of developing an eating disorder, with a particular risk for developing bulimia nervosa. Girls with ADHD and an eating disorder had increased rates of mood, anxiety, and disruptive behavior disorders and an earlier age at onset of menarche compared to their ADHD peers without eating disorders.

The finding that adolescent girls with ADHD are at

increased risk of bulimia nervosa is consistent with a previous report documenting a similar association in women with ADHD. Surman et al.<sup>1</sup> documented that adult females had a significant overrepresentation of bulimia nervosa compared to women without ADHD.

The finding that adolescent girls with ADHD and an eating disorder had an increased risk of major depression and anxiety disorders is consistent with research on eating disorder samples. For example, a recent study by Blinder et al.<sup>28</sup> found that 94% of patients with eating disorders had comorbid mood disorders and 56% had comorbid anxiety disorders. Although there is some discrepancy in the rates of the comorbid disorders, with some studies reporting smaller, yet significant rates, a converging literature has documented a strong association between mood and anxiety disorders in subjects with eating disorders.<sup>29-32</sup> Considering the morbidity associated with mood and anxiety disorders and their unique therapeutic requirements, girls with ADHD and eating disorders may benefit from interventions aimed at addressing these disorders.

The finding that girls with ADHD and eating disorders are at increased risk of comorbid disruptive behavior disorders is novel. Our findings that the two ADHD groups did not differ on ADHD correlates suggests that the comorbidity is not a function of ADHD severity, but

**Table 2.** Puberty/Growth Characteristics of Youths with and without ADHD and Eating Disorders

	Controls (n = 105)	ADHD (n = 103)	ADHD + Eating Disorder (n = 20)	Statistical Significance
Puberty characteristics				
Age at menarche, yr	12.1 ± 1.1	12.8 ± 1.2	11.4 ± 0.9 <sup>b</sup>	$F_{2,88} = 3.3, p = .04$
Growth characteristics				
Age-corrected height $z$ score	0.189 ± 1.4	0.160 ± 1.4	0.343 ± 1.0	$F_{2,213} = 0.15, p = .86$
Age and height adjusted weight $z$ score	1.135 ± 0.30	1.095 ± 0.22	1.240 ± 0.44	$F_{2,208} = 2.1, p = .12$

ADHD, attention-deficit/hyperactivity disorder. <sup>b</sup>Versus ADHD,  $p < .05$ .

rather represents a true comorbidity. Although the reasons for this association remain unclear, considering the morbidity and dysfunction associated with disruptive disorders,<sup>35</sup> more work is needed to confirm this association.

Also novel is the finding that eating disorders were associated with earlier menarche in girls with ADHD. The reasons for this association are intriguing when considering that eating disorders have been associated with menstrual irregularities and amenorrhea.<sup>34</sup> More work is needed to confirm and understand this finding.

Our results must be interpreted in the context of some methodological limitations. Although we did not exclude any ethnic group, nearly all our subjects were white. Thus, our results may not generalize to other racial and ethnic groups. Also since our subjects have been referred, our findings may not generalize to community samples. Although our results are based on a large sample of girls with ADHD, the number of girls with eating disorders was relatively small and we did not have the power to fully elucidate all differences between girls with and without an eating disorder or to examine anorexia and bulimia nervosa as separate disorders. Our sample of ADHD girls with anorexia and bulimia was composed of girls with either a full clinical or subclinical diagnosis. We adopted this method to increase the sensitivity and capture subclinical cases that have a high likelihood of developing full DSM criteria for eating disorders in the future since our sample was not entirely through the period of risk of developing an eating disorder. However, it is possible that our results may have been different if the entire sample had full clinical diagnoses. In future follow-up assessments of this sample, we plan to revisit these questions.

It may be that with additional follow-up assessments, the association between ADHD and eating disorders could be clarified further. Due to our lack of a control group with eating disorders, we are not able to fully elucidate the relationships between ADHD, eating disorders, and comorbid internalizing and disruptive behavior disorders. It is possible that the well-known association between ADHD and disruptive behavior disorders drives the relationship between eating disorders and disruptive behavior disorders found in our data. Similarly, it is possible that the relationship we found between ADHD and internalizing disorders is an artifact of the well-established association of eating disorders and internalizing disorders. Further research with controls with eating disorders is needed to clarify these associations.

Finally, since our sample was originally ascertained using DSM-III-R criteria, it is possible that our results may not generalize to samples ascertained by DSM-IV criteria. However, considering the very high degree of overlap between the two definitions (93% of DSM-III-R cases received a DSM-IV diagnosis)<sup>35</sup> and our use of DSM-IV criteria in our follow-up assessment, any effect on these results should be minimal.

Despite these concerns, our findings document a significant association between ADHD and eating disorders in adolescent girls. Eating disorders and ADHD require different pharmacological and nonpharmacological treat-

ment approaches and therefore, clinical evaluations of females with eating disorders may benefit from systematic identification of ADHD and vice versa. Among individuals with comorbid ADHD and bulimia nervosa, the impulsivity of ADHD might contribute to the severity of eating disordered behavior. Patients with bulimia nervosa and ADHD may benefit from treatments commonly used to treat ADHD.<sup>36,37</sup>

Furthermore, eating disorders in girls with ADHD significantly increased the risk of depression, anxiety, and disruptive behavior disorders. Although more research is needed to confirm these findings, our results suggest that ADHD significantly increases the risk of eating disorders and that the presence of an eating disorder in girls with ADHD heightens the risk of additional morbidity and dysfunction.

## REFERENCES

1. Surman C, Randell E, Biederman J. Association between attention-deficit/hyperactivity disorder and bulimia nervosa: analysis of 4 case-control studies. *J Clin Psychiatry*. 2006;67:351-354.
2. Schweickert L, Strober M, Moskowitz A. Efficacy of methylphenidate in bulimia nervosa comorbid with attention-deficit hyperactivity disorder: a case report. *Int J Eat Disord*. 1997;21:299-301.
3. Sokol MS, Gray NS, Goldstein A, et al. Methylphenidate treatment for bulimia nervosa associated with a cluster B personality disorder. *Int J Eat Disord*. 1999;25:233-237.
4. Drimmer EJ. Stimulant treatment of bulimia nervosa with and without attention-deficit disorder: three case reports. *Nutrition*. 2003;19:76-77.
5. Mattos P, Saboya E, Ayrao V, et al. [Comorbid eating disorders in a Brazilian attention-deficit/hyperactivity disorder adult clinical sample]. *Rev Bras Psiquiatr*. 2004;26:248-250.
6. Sohlberg S, Norring C, Holmgren S, et al. Impulsivity and long-term prognosis of psychiatric patients with anorexia nervosa/bulimia nervosa. *J Nerv Ment Disord*. 1989;177:249-258.
7. Keel PK, Mitchell JE. Outcome in bulimia nervosa. *Am J Psychiatry*. 1997;154:313-321.
8. Keel PK, Mayer SA, Hamden-Fischer JH. Importance of size in defining binge eating episodes in bulimia nervosa. *Int J Eat Disord*. 2001;29:249-301.
9. Carroll JM, Touyz SW, Beaumont PJ. Specific comorbidity in anorexia and bulimia nervosa and personality disorders. *Int J Eat Disord*. 1996;19:159-170.
10. O'Brien KM, Vincent NK. Psychiatric comorbidity in anorexia and bulimia nervosa: nature, prevalence, and causal relationships. *Clin Psychol Rev*. 2003;23:57-74.
11. Rossiter EM, Agras WS, Telch CF, et al. Cluster B personality disorder characteristics predict outcome in the treatment of bulimia nervosa. *Int J Eat Disord*. 1993;13:349-357.
12. Biederman J, Monuteaux M, Mick E, et al. Psychopathology in females with attention-deficit/hyperactivity disorder: a controlled, five-year prospective study. *Biol Psychiatry*. 2006;60:1098-1105.
13. Orvaschel H. Psychiatric interviews suitable for use in research with children and adolescents. *Psychopharmacol Bull*. 1985;21:737-745.
14. Orvaschel H, Puig-Antich J. *Schedule for Affective Disorders and Schizophrenia for School-Age Children: Epidemiologic Version*. Fort Lauderdale, FL: Nova University; 1987.
15. Spitzer RL, Williams JB, Gibbon M, et al. *Structured Clinical Interview for DSM-III-R: Non-Patient Edition (SCID-NP, Version*

- 1.0). Washington, DC: American Psychiatric Press; 1990.
16. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. CDC growth charts: United States. *Adv Data*. 2000;314:1-27.
  17. Chinchilli VM, McEnery PT, Chan JCM. Statistical methods and determination of sample size in the growth failure in children with renal diseases study. *J Pediatr*. 1990;116:S32-S36.
  18. Abitbol C, Foreman JW, Strife CF, et al. Quantitation of growth deficits in children with renal diseases. *Semin Nephrol*. 1989;9:31-36.
  19. Spencer T, Biederman J, Harding M, et al. Growth deficits in ADHD children revisited: evidence for disorder-associated growth delays? *J Am Acad Child Adolesc Psychiatry*. 1996;35:1460-1469.
  20. Biederman J, Faraone SV, Monuteaux M, et al. Growth deficits and ADHD revisited: impact of gender, development and treatment. *Pediatrics*. 2003;111:1010-1016.
  21. Polito C, Oporto MR, Totino SF, et al. Normal growth of nephrotic children during long-term alternate-day prednisone therapy. *Acta Paediatr Scand*. 1986;75:245-250.
  22. Spencer T, Biederman J, Wright V, et al. Growth deficits in children treated with desipramine: a controlled study. *J Am Acad Child Adolesc Psychiatry*. 1992;31:235-243.
  23. Waterlow JC. Classification and definition of protein-calorie malnutrition. *BMJ*. 1972;3:566-569.
  24. Waterlow JC. Note on the assessment and classification of protein-energy malnutrition in children. *Lancet*. 1973;2:87-89.
  25. Waterlow JC. Some aspects of childhood malnutrition as a public health problem. *BMJ*. 1974;4:88-90.
  26. McLaren DS, Read WWC. Weight/length classification of nutritional status. *Lancet*. 1975;2:219-221.
  27. Anderson MA. Comparison of anthropometric measures of nutritional status in preschool children in five developing countries. *Am J Clin Nutr*. 1979;32:2339-2345.
  28. Blinder BJ, Cumella EJ, Sanathara VA. Psychiatric comorbidities of female inpatients with eating disorders. *Psychosom Med*. 2006;68:454-462.
  29. Kaye WH, Bulik CM, Thornton LM, et al. Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *Am J Psychiatry*. 2004;161:2215-2221.
  30. Godart NT, Flament MF, Curt F, et al. Anxiety disorders in subjects seeking treatment for eating disorders: a DSM-IV controlled study. *Psychiatry Res*. 2003;117:245-258.
  31. Herzog DB, Nussbaum KM, Marmor AK. Comorbidity and outcome in eating disorders. *Psychiatr Clin North Am*. 1996;19:843-859.
  32. Brewerton TD, Lydiard RB, Herzog DB, et al. Comorbidity of axis I psychiatric disorders in bulimia nervosa. *J Clin Psychiatry*. 1995;56:77-80.
  33. Greene RW, Biederman J, Zerwas S, et al. Psychiatric comorbidity, family dysfunction, and social impairment in referred youth with oppositional defiant disorder. *Am J Psychiatry*. 2002;159:1214-1224.
  34. Mitan LAP. Menstrual dysfunction in anorexia nervosa. *J Pediatr Adolesc Gynecol*. 2004;17:81-85.
  35. Biederman J, Faraone SV, Weber W, et al. Correspondence between DSM-III-R and DSM-IV attention deficit hyperactivity disorder (ADHD). *J Am Acad Child Adolesc Psychiatry*. 1997;36:1682-1687.
  36. Messner E. Methylphenidate treatment of bulimia nervosa after surgery. *Can J Psychiatry*. 1989;34:824-826.
  37. Ong YL, Checkley SA, Russell GF. Suppression of bulimic symptoms with methylamphetamine. *Br J Psychiatry*. 1983;143:288-293.