Published in final edited form as:
Obesity (Silver Spring). 2011 May ; 19(5): 982–987. doi:10.1038/oby.2010.249.

Weight Reduction in Obese Adolescents with and without Binge Eating

Chanelle T. Bishop-Gilyard[1], **Robert I. Berkowitz**[1,2], **Thomas A. Wadden**[2], **Christine A. Gehrman**[1], **Joanna L. Cronquist**[1], and **Reneé H. Moore**[2]

[1] The Children's Hospital of Philadelphia, Philadelphia PA

[2] The University of Pennsylvania, Philadelphia, PA

Abstract

Little is known about binge eating (BE) in adolescents. The primary aim of the present study was to examine the relationship between BE and weight loss in adolescents (BMI ≥ 95th percentile) enrolled in a randomized controlled trial of behavioral and pharmacologic treatment of obesity. Participants were 82 treatment-seeking adolescents (BMI = 37.9 ± 3.8 kg/m^2; age = 14.1 ± 1.2 years; 67% females; 42% African American, 55% Caucasian). Participants completed the Children's Depression Inventory, the Piers Harris Self-Esteem Questionnaire, and the Eating Inventory (including cognitive restraint, disinhibition, and hunger scales). BE was assessed by a questionnaire and a confirmatory interview. At baseline, 24% of participants met criteria for BE (N=13 met full BED criteria; N = 7 met sub-threshold BE). There were no significant differences in percentage reduction in initial BMI between participants with or without BE at Month 6 (−7.0 ± 1.6% vs. −6.9 ± 0.9%) or Month 12 (−8.8 ± 2.4% vs. −8.3 ± 1.3%) (omnibus main effect BE p = 0.89, interaction BE*Time p = 0.84, interaction BE*Drug p = 0.61). The rate of BE declined significantly over time from 24% (n=20) at baseline to 8% (n=6) at month 6 and 3% (n=2) at month 12 (p=0.003). There were significant decreases in hunger and disinhibition as well as an increase in cognitive restraint over time (all p ≤ 0.0001). Findings suggest a combination of behavioral and pharmacologic therapy may produce both weight loss and improvement in binge eating.

Keywords

binge eating; adolescents

Binge eating disorder (BED) is characterized by the consumption of an objectively large amount of food, in a brief period (i.e., < 2 hours), during which an individual experiences a subjective sense of loss of control, as well as later distress about the eating episode. Binge episodes must occur on average twice per week for 6 months to meet criteria for BED (1). Unlike bulimia nervosa, BED is not accompanied by inappropriate compensatory behaviors. In the general population, approximately 2% to 3% of adults suffer from BED (2). However, studies have shown that between 20 to 30% of obese treatment-seeking adults suffer from this disorder (3–4). In adults, BED is associated with increased body mass index, medical problems, and psychopathology, including depression, anxiety, and substance abuse (2,5–8).

Correspondences to: Chanelle T. Bishop-Gilyard, Psy.D., The Children's Hospital of Philadelphia, 3440 Market Street, Suite 410, Philadelphia, PA 19104; (T): (215) 590-2295; (F): (215) 590-4251; bishopc@email.chop.edu.

Disclosure
The authors declared no conflict of interest.

Binge eating also occurs in overweight youth (9–11). However, children and adolescents are less likely to meet full criteria for BED (12–13). Instead, they more commonly experience sub-threshold binge eating (BE), in which they do not fulfill criteria of bingeing at least twice a week for the last 6 months or meet other secondary characteristics (9,14–16). Like overweight adults with BED, overweight youth with BE are heavier, report more eating-related psychopathological features, greater weight and shape concerns, and depressive symptoms (9–10,17–18).

It is unclear whether BE negatively affects weight loss efforts in adults or adolescents. Several early studies, with small sample sizes, found that patients with BE lost significantly less weight than did those without this condition (9,19–21). However in the Look AHEAD study, researchers found that adult diabetic participants who reported BE at baseline, but remission of this behavior at one year, were just as successful in losing weight as those who did not endorse BE at either time point, as measured by self-report questionnaire (22). However, those whose BE did not remit or who developed BE over the year did not lose as much weight as those who were free of this problem at 1 year. Other investigators have also reported successful weight loss in obese adults with BE (23–24).

Few studies have examined whether BE is associated with suboptimal weight loss in obese adolescents. Wildes et al. found that children who reported BE had an increase in weight during weight loss treatment, compared to the non bingers who lost weight (25). However, this study was limited by a one item self-report measure of binge eating. Similarly, Braet found that binge symptoms were related to less favorable outcomes after a 2 year follow up of inpatient treatment for obese children (26). Other studies have found no relation between broadly defined BE and weight loss. Jones and colleagues examined overweight teens who reported overeating and some binge eating and who received an Internet-based intervention. These participants achieved a greater reduction, in overeating and binge eating as well as BMI, compared to a wait-list control group. However, these results were limited by self-reported height and weight at post test (27). The authors did not examine the relationship between the severity of overeating and BE at baseline and subsequent weight loss.

The primary goal of the present study was to examine the relationship between BE status and weight loss in 82 obese adolescents who participated in a comprehensive weight-loss program. In addition to examining whether BE would be associated with suboptimal weight loss at the end of both 6 and 12 months of treatment, we also sought to confirm the relationship between BE and baseline psychopathology. Specifically, we predicted that obese adolescents with BE would report significantly greater symptoms of depression and lower self-esteem than would their counterparts without this disorder. We also wished to confirm that BE would be associated with greater levels of dietary disinhibition and hunger, as well as lower levels of cognitive restraint. Finally, we examined whether BE and measures of eating and appetite (i.e., hunger, disinhibition, and cognitive restraint) changed with weight loss treatment.

Methods and Procedures

Participants and Procedures

Participants were 82 treatment-seeking participants who were 13 to 17 years of age with a body mass index (BMI) of 32 to 44 kg/m^2. They were recruited to participate in a randomized, double-blind, placebo-controlled trial that compared the efficacy of a comprehensive program of lifestyle modification to the same program combined with the weight loss medication, sibutramine. The results of this study have been reported previously (28). In brief, participants in both groups received weekly group behavioral counseling for 16 weeks followed by bi-weekly visits for an additional 8 weeks (phase 1 of treatment).

Parents attended separate group sessions in which they were instructed in methods of supporting their children. Following randomization, half of the participants received placebo, while the other half were prescribed up to 15 mg/d of sibutramine (as described previously). After the initial 6 months, an open-label extension of sibutramine was offered to all participants for an additional 6 months (i.e., phase 2). The study was approved by the institutional review boards at The Children's Hospital of Philadelphia and at the University of Pennsylvania.

Prior to treatment, applicants underwent a behavioral and medical screening evaluation and were excluded for high blood pressure and co-morbid psychiatric disorders. Psychiatric exclusions included: major depression; bipolar disorder; psychosis and/or the use of anti-psychotic or antidepressant medication in the past 12 months; history of anorexia or bulimia nervosa; history of alcoholism or other substance abuse; and parental psychopathology. Participants' heights and weights were measured using an electronic scale (Model 5702, Bariatric Stand-On Scale, Scale-Tronix, Carol Steam, IL) and stadiometer (Harpenden Stadiometer, Holtain Limited, Crymch Pembs., UK), respectively. Height, weight, and waist circumference were measured at the major assessments following standard techniques (29).

Measures

Binge Eating—Symptoms of BE were assessed using the Questionnaire on Weight and Eating Patterns (QWEP), which was complemented by an interview to confirm that participants consumed an objectively large amount of food and experienced loss of control (30). Participants were categorized as having: 1) BED (if they met all criteria proposed by Spitzer et al (4)); 2) subthreshold BE (i.e., characterized by overeating with loss of control but not meeting twice weekly frequency of binge episodes or other associated features; or 3) no symptoms of BE.

Eating behavior—Eating behavior was assessed using the Eating Inventory (EI) (31). This self-report measure consists of three subscales - cognitive restraint, disinhibition, and hunger -which measure the ability to control food intake, loss of control over eating, and reported hunger, respectively.

Depression—Symptoms of depression were assessed using the Children's Depression Inventory (CDI). The CDI is a 27-item scale, which is the standard measure for depression in children and adolescents (32). Scores range from 0 to 54, with higher scores indicating greater symptoms of depression. Scores of less than 13 are considered minimal or within the normal range.

Piers Harris Self-Concept Scale—Self-concept was assessed using this 80-item self-report measure for 7–18 year olds (33). This scale measures self-concept as it relates to physical appearance, popularity, behavior, happiness, anxiety, and intellectual status. The Piers Harris provides a total score for overall self-concept, as well as subscale scores. Higher scores are indicative of a healthier self-concept.

Statistical Analysis Plan

All analyses were performed using SAS statistical software, version 9.2 (SAS Institute, Inc., Cary, North Carolina) and the significance level for all analyses was set at alpha equal to 0.05.

Differences in baseline demographic characteristics between participants with and without BE were compared using independent two sample t-tests for continuous variables and using chi-square tests for categorical variables. Baseline characteristics for those with and without

BE were summarized as means (standard deviation) for age, BMI, BMI z-score, weight, and waist circumference and, with frequencies and percentages for gender, race, and treatment group.

Analysis of covariance models (ANCOVA) were fit to determine whether BE was associated with baseline psychosocial variables (Eating Inventory, CDI, and Piers Harris Total scores). For these unadjusted models, we present means (standard error) and effect sizes that were calculated using Cohen's d = Mean (BE) − (non- BE) divided by σ_{pooled}, where $\sigma_{pooled} = \sqrt{[(\sigma_1^2 + \sigma_2^2)/2]}$. In addition, we fitted ANCOVA models that examined the interaction term of BE and baseline characteristics as they related to the psychological outcome variables.

Repeated measures mixed models, adjusting for treatment group, were fit to explore whether BE and non- BE participants lost significantly different amounts of weight at months 6 and 12, as measured by percentage change in BMI and absolute changes in BMI, BMI z-score, weight, and waist circumference (summarized as means (standard error)). Furthermore, the percentages of BE and non- BE patients that achieved three levels of weight loss (≤5% reduction in BMI, 5–10%, and >10% reduction in BMI) were examined. Chi-square tests were used to determine if the percentages of BE and non- BE patients that achieved these weight loss levels were statistically different.

To assess if the prevalence of BE changed significantly from baseline to months 6 and 12, a repeated measures generalized estimating equation (GEE) model was fit. As the number of subjects with BE was too small to obtain stable estimates from the GEE model, a Mantel-Haenszel Chi-Square was conducted to assess change in BE over time. Finally, repeated measures mixed models were fit to model change over time in each of the three Eating Inventory measurements.

Results

BE Status at Baseline

At baseline, 20 of 82 (24%) participants displayed symptoms of BE. Specifically, 13 participants (16%) met full criteria for BED and 7 (8.5%) met sub-threshold criteria (BE). The latter participants engaged in binge eating less than 2 times per week or did not meet other secondary criteria. Analyses indicated that these two groups did not differ significantly on any baseline characteristics or on changes with treatment. Thus, these two groups were collapsed into a single BE sample. The remaining 62 participants (76%) had no symptoms of BE.

Participants' baseline demographic characteristics, by BE status, are presented in Table 1. There were no statistically significant differences in baseline age, weight, race, or treatment group assignment between BE and non-BE participants. A trend toward significance was revealed for gender; the percentage of males with BE was higher than the percentage of females with this disorder (37% and 18%, respectively, $p = .06$).

BE, Baseline Psychosocial Status, and Eating Behavior

There were no significant differences between the combined BE and non- BE groups on psychosocial variables including depression, self-esteem, or cognitive restraint (Table 2). However, participants with BE reported greater disinhibition than those without BE (8.9 ± 0.7 vs. 5.5 ± 0.4, respectively, $p < .0001$). Similarly, hunger scores were significantly higher in BE than non- BE participants (7.6 ± 0.7 vs. 4.1 ± 0.4, respectively, $p = .0001$). Results were similar when adjusting for treatment condition (data not shown).

BE and Weight Reduction

As originally reported by Berkowitz *et al.* (28), at the end of the first 6 months, participants who received lifestyle modification plus placebo lost 4.0 ± 5.4% of their initial BMI, compared with a significantly greater loss of 8.5 ± 6.8% for those assigned to lifestyle modification with sibutramine. At 12 months, after the 6-month open label extension, weight losses for these two treatment groups were 6.4 ± 8.3% and 8.6 ± 9.9%, respectively (28).

Based on the mixed model, which controlled for treatment condition, there were no significant differences in percentage reduction in initial BMI between participants with or without BE at Months 6 (−7.0 ± 1.6% vs. −6.9 ± 0.9%) or 12 (−8.8 ± 2.4% vs. −8.3% ± 1.3%) (omnibus main effect BE $p = 0.89$; interaction BE *Time $p = 0.84$; interaction BE*Drug $p = 0.61$) (see Table 3). Similarly, in secondary analyses, there were no significant differences in change in absolute BMI, BMI z-score, weight, and waist circumference for BE and non- BE groups at months 6 and 12 (Table 3) (all models $p > 0.10$). Participants who met full criteria for BED did not significantly differ from those with sub-threshold BE on BMI or other weight loss variables (all models $p > .22$, data not shown). Table 4 shows the percentage of BE and non- BE participants who lost < 5%, 5%–10%, and > 10% of initial BMI at months 6 and 12.

Changes Overtime in BE Status and Eating Behavior

The prevalence of BE significantly decreased from 24% (n=20) at baseline to 8% (n=6) at month 6 and 3% (n=2) at month 12 (Mantel-Haenszel Chi-Square p=0.003). Two participants, who were free of BE at baseline, met criteria for sub-threshold BE at month 6, and one of these participants resolved at month 12, but data are unknown for the other participant at month 12. One participant who met criteria for sub-threshold BE at baseline, met full criteria at month 6 but data are unknown at month 12. At month 12, there was one new case of sub-threshold BE, but all other cases of BE decreased from full criteria to sub-threshold or resolved completely.

Hunger, disinhibition, and cognitive restraint scores significantly changed over time (reported as absolute values). When adjusted for the interaction between treatment group and time, hunger significantly decreased from 4.7 ± 0.36 at baseline, to 3.5 ± 0.33 at Month 6, and to 3.2 ± 0.35 at Month 12 ($p = 0.0001$). Similarly, disinhibition decreased from 6.4 ± 0.37 at baseline, to 4.6 ± 0.39 at Month 6, and to 4.1 ± 0.030 at Month 12 ($p < 0.0001$). Cognitive restraint increased from 9.5 ± 0.40 at baseline, to 14.2 ± 0.52 at Month 6, and to 14.9 ± 0.54 at Month 12 ($p < 0.0001$).

Discussion

The principal finding of this study was that obese adolescents with BE lost comparable amounts of weight as those without this condition during 1 year of treatment with behavioral and pharmacologic therapies. Those with BE achieved an 8.9% reduction in BMI, as compared with 8.4% for non- BE participants. Similar findings were obtained for the two groups at 6 months. These findings are consistent with prior studies of adults, most notably the Look AHEAD study, which found no differences in weight loss at the end of 1 year of treatment in obese participants without BE before treatment and those who reported BE at baseline but stopped this behavior by one year (22). A second finding was that the proportion of participants with BE decreased significantly from 24% at baseline to 8% at 6 months and ultimately to 3% at 12 months. The remission of BE was offset by the development over the course of the year of 4 new cases, 3 of which were subthreshold and resolved at 12 months. The present findings are consistent with prior studies of adults and adolescents that have reported reductions in binge eating in response to weight loss

treatment (23–24,27,34–36). In addition, there was minimal evidence that dieting and weight loss precipitated binge eating in participants who were free of this condition prior to treatment. A review of the literature on the behavioral treatment of obesity in children and adolescents similarly found little evidence that dieting or weight loss precipitated binge eating (10,37).

Taken together, these findings indicate that it is not necessary to treat binge eating in obese individuals prior to inducing weight loss. Persons with BE appear to lose weight satisfactorily. Contrary to concerns of some investigators, a structured weight reduction program appears to ameliorate rather than exacerbate binge eating. Therefore, the current findings contribute to the growing literature that suggests that interventions that target weight-loss may also reduce BE in both adults and adolescents.

The present study confirmed previous findings that BE was associated with greater dietary disinhibition, as assessed by the Eating Inventory. This was expected, given that loss of control over eating (i.e., dishinibition) is a defining feature of BE. Participants with BE, compared to those without this disorder, also reported greater hunger, confirming the results of prior studies of adolescents (10,38). We note that two studies of adults found that hunger scores were similar between BE and non-BE participants (39,40). Disinhibition and hunger decreased with treatment. Disinhibition (loss of control over eating) is a component of BE and its reduction is consistent with the decreased prevalence of BE. Cognitive restraint also improved with treatment, as has been reported in other studies of weight loss (40). The present study did not confirm findings that BE was associated with greater symptoms of depression or lower self-esteem. One limitation and potential explanation for the lack of findings is that severely depressed participants (as determined by a CDI raw score above 17) were not included in the present study. Thus, we were unable to capture the full range of depressive symptomatology. If more depressed adolescents had been included in our study, we may have found an association between BE, depression, and self-esteem. With the exclusion of severely depressed participants, we also cannot comment on weight loss or remission in binge eating in patients who have both BE and depression. We also did not examine anxiety in our study and, thus, cannot address the association between anxiety and BE reported in other studies. Given these limitations, our failure to confirm greater psychopathology (e.g., higher negative mood and anxiety, lower self-esteem) observed among adolescents with BE (9–10,14–16), must be interpreted with caution.

Strengths of this study include an ethnically diverse sample and the examination of BE in adolescents who received pharmacologic and behavioral treatment for their obesity. However, limitations include a relatively small sample size and a restricted range of psychopathology and BE. Such limitations could pose a threat to generalizability. Further potential limitations include the use of a diagnostic interview that is not as extensive as other established methods for determining BE and failure to assess inter-rater reliability.

In summary, this study found that 24% of a sample of obese adolescents had BE prior to treatment. Participants with BE lost comparable amounts of weight as those who were free of this disorder at baseline, and the proportion of participants with BE declined from 24% to 3% at 12 months. We believe that these data suggest that a comprehensive behavioral weight loss program may be used as the first step of treatment for obese adolescents who suffer from BE. This recommendation ultimately must be supported by larger investigations that potentially include adolescents who suffer from more severe depression and BE.

Acknowledgments

This study was supported by grants 5R01DK054713-07 (Dr. Berkowitz), K24DK065018 (Dr. Wadden) from the National Institutes of Diabetes, Digestive, and Kidney Disease and grant UL1RR024134 from the National Center

for Research Resources. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Center for Research Resources or the National Institutes of Health. Abbott provided sibutramine for the study.

References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4. American Psychiatric Association; Washington, DC: 2000. Text Revision
2. Hudson JI, Hiripi E, Pope HG, Kellser RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol Psychiatry. 2007; 61:348–358. [PubMed: 16815322]
3. Hsu LK, Mulliken B, McDonagh B, Krupa Das S, Rand W, Fairburn CG, Rolls B, McCrory MA, Saltzman E, Shikora S, Dwyer J, Roberts S. Binge eating disorder in extreme obesity. Int J Obes Relat Metab Disord. 2002; 26:1398–1403. [PubMed: 12355338]
4. Spitzer RL, Yanovski S, Wadden T, et al. Binge eating disorder: its further validation in a multisite study. Int J Eat Disord. 1993; 13:137–53. [PubMed: 8477283]
5. Yanovski SZ. Binge eating disorder: current knowledge and future directions. Obes Res. 1993; 1:306–324. [PubMed: 16350580]
6. Wilfley DE, Friedman MA, Dounchis JZ, Stein RI, Welch RR, Ball SA. Comorbid psychopathology in binge eating disorder: relation to eating disorder severity at baseline and following treatment. J Consult Clin Psychol. 2000; 68:641–649. [PubMed: 10965639]
7. Javaras KN, Pope HG, Lalonde JK, Roberts JL, Nillni YI, Laird NM, Bulik CM, Crow SJ, McElroy SL, Walsh BT, Tsuang MT, Rosenthal NR, Hudson JI. Co-occurrence of binge eating disorder with psychiatric and medical disorders. J Clin Psychiatry. 2008; 69:266–273. [PubMed: 18348600]
8. Grilo CM, White MA, Masheb RM. DSM-IV psychiatric disorder comorbidity and its correlates in binge eating disorder. Int J Eat Disord. 2009; 42:228–234. [PubMed: 18951458]
9. Decaluwé V, Braet C, Fairburn CG. Binge eating in obese children and adolescents. Int J Eat Disord. 2003; 33:78–84. [PubMed: 12474202]
10. Berkowitz RI, Stunkard AJ, Stallings VA. Binge-eating disorder in obese adolescent girls. Annals of the New York Academy of Science. 1993; 699:200–206.
11. Britz B, Siegfried W, Ziegler A, et al. Rates of psychiatric disorders in a clinical study group of adolescents with extreme obesity and in obese adolescents ascertained via population based study. Int J Obes. 2000; 24:1707–1714.
12. Stice E, Killen JD, Hayward C, Taylor CB. Age of onset for binge eating and purging during late adolescence: a 4-year survival analysis. J Abnorm Psychol. 1998; 107:671–675. [PubMed: 9830254]
13. Stice E, Presnell K, Bearman SK. Relation of early menarche to depression, eating disorders, substance abuse, and comorbid psychopathology among adolescent girls. Dev Psychol. 2001; 37:608–619. [PubMed: 11552757]
14. Isnard P, Michel G, Frelut ML, Vila G, Falissard B, Naja W, Navarro J, Mouren-Simeoni MC. Binge eating and psychopathology in severely obese adolescents. Int J Eat Disord. 2003; 34:235–243. [PubMed: 12898560]
15. Tanofsky-Kraff M, Yanovski SZ, Wilfley DE, Marmarosh C, Morgan CM, Yanovski JA. Eating-disordered behaviors, body fat, and psychopathology in overweight and normal-weight children. J Consult Clin Psychol. 2004; 72:53–61. [PubMed: 14756614]
16. Glasofer D, Tanofsky-Kraff M, Eddy K, Yanovski S, Theim K, Mirch M, Ghorbani S, Ranzenhofer LM, Haaga D, Yanovski JA. Binge eating overweight treatment-seeking adolescents. Journal of Pediatric Psychology. 2007; 32(1):95–105. [PubMed: 16801323]
17. Morgan CM, Yanovski SZ, Nguyen TT, et al. Loss of control over eating, adiposity, and psychopathology in overweight children. Int J Eat Disord. 2002; 31:430–441. [PubMed: 11948648]
18. Tanofsky-Kraff M, Faden D, Yanovski SZ, Wilfley DE, Yanovski JA. The perceived onset of dieting and loss of control eating beahviors in overweight children. Int J Eat Disord. 2005; 38:112–122. [PubMed: 16134103]

19. Marcus MD, Wing RR, Hopkins J. Obese binge eaters: Affect, cognitions, and behavioral weight control. J Consult Clin Psychol. 1988; 56:433–439. [PubMed: 3397436]
20. Yanovski SZ, Gormally JF, Leser MS, Gwirtsman HE, Yanovski JA. Binge eating disorder affects outcome of comprehensive very-low-calorie diet treatment. Obes Res. 1994; 2:205–212. [PubMed: 16353422]
21. Ferguson KJ, Spitzer RL. Binge eating disorder in a community-based sample of successful and unsuccessful dieters. Int J Eat Disord. 1995; 18:167–172. [PubMed: 7581419]
22. Gorin AA, Niemeier HM, Hogan P, Coday M, Davis C, DiLillo VG, Gluck ME, Wadden TA, West DS, Williamson D, Yanovski SZ. Look AHEAD Research Group. Binge eating and weight loss outcomes in overweight and obese individuals with type 2 diabetes: results from the Look AHEAD trial. Arc h Gen Psychiatry. 2008; 65:1447–1455.
23. Sherwood NE, Jeffery RW, Wing RR. Binge status as a predictor of weight loss treatment outcome. Int J Obes Relat Metab Disord. 1999; 23:485–493. [PubMed: 10375051]
24. Gladis MM, Wadden TA, Vogt R, Foster G, Kuehnel RH, Bartlett SJ. Behavioral treatment of obese binge eaters: do they need different care? J Psychosom Res. 1998; 44:375–384. [PubMed: 9587881]
25. Wildes JE, Marcus MD, Kalarchian MA, Levine MD, Houck PR, Cheng Y. Self-reported binge eating in severe pediatric obesity: impact on weight change in a randomized controlled trial of family-based treatment. Int J Obes. 201010.1038/ijo.2010.35
26. Braet C. Patient characteristics as predictors of weight loss after an obesity treatment for children. Obesity. 2006; 14:148–155. [PubMed: 16493133]
27. Jones M, Luce KH, Osborne MI, Taylor K, Cunning D, Doyle AC, Wilfley DE, Taylor CB. Randomized, controlled trial of an internet-facilitated intervention for reducing binge eating and overweight in adolescents. Pediatrics. 2008; 121:453–462. [PubMed: 18310192]
28. Berkowitz RI, Wadden TA, Tershakovec AM, Cronquist JL. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. JAMA. 2003; 289:1805–1812. [PubMed: 12684359]
29. Calloway, CW.; Chumlea, WC.; Bouchard, C. Circumferences. In: Lohman, TC.; Roche, AR.; Martorell, R., editors. Anthropometric Standardization Manual. Human Kinetics Publishers; Champaign, Ill: 1988. p. 39-64.
30. Spitzer RL, Devlin M, Walsh BT, Hasin D, Wing R, Marcus M, Stunkard M, Wadden T, Yanovski S, Agras S, Mitchell J, Nonas C. Binge eating disorder: a multisite field trial of the diagnostic criteria. Int J Eating Disord. 1992; 11:191–203.
31. Stunkard, AJ.; Messick, S. Eating Inventory Manual. Psychological Corporation; San Antonio: 1988.
32. Kovacs, M. Children's Depression Inventory (CDI) Manual. Multi-Health Systems; New York: 1992.
33. Piers, EV. Revised Manual. Western Psychological Services; Los Angeles, CA: 1986. The Piers-Harris Children's Self-Concept Scale.
34. Alger SA, Malone M, Cerulli J, Fein S, Howard L. Beneficial effects of pharmacotherapy on weight loss, depressive symptoms, and eating patterns in obese binge eaters and non-binge eaters. Obes Res. 1999; 7:469–476. [PubMed: 10509604]
35. Wilfley DE, Crow SJ, Hudson JI, Mitchell JE, Berkowitz RI, Blakesley V, Walsh BT. Sibutramine Binge Eating Disorder Research Group. Efficacy of sibutramine for the treatment of binge eating disorder: a randomized multicenter placebo-controlled double-blind study. Am J Psychiatry. 2008; 165:51–58. [PubMed: 18056225]
36. Appolinario JC, Bacaltchuk J, Sichieri R, Claudino AM, Godoy-Matos A, Morgan C, Zanella MT, Coutinho W. A randomized, double-blind, placebo-controlled study of sibutramine in the treatment of binge-eating disorder. Arch Gen Psychiatry. 2003; 60:1109–1116. [PubMed: 14609886]
37. Butryn ML, Wadden TA. Treatment of overweight in children and adolescents: does dieting increase the risk of eating disorders? Int J Eat Disord. 2005; 37:285–293. [PubMed: 15856498]
38. Bas M, Bozan N, Cigerim N. Dieting, dietary restraint, and binge eating disorder among overweight adolescents in Turkey. Adolescence. 2008; 43:635–648. [PubMed: 19086675]

39. Samuels F, Zimmerli EJ, Devlin MJ, Kissileff HR, Walsh BT. The development of hunger and fullness during a laboratory meal in patients with binge eating disorder. Int J Eat Disord. 2009; 42:125–129. [PubMed: 18803172]

40. Downe KA, Goldfein JA, Devlin MJ. Restraint, hunger, and disinhibition following treatment for binge eating disorder. Int J Eat Disord. 2009; 42:498–504. [PubMed: 19130489]

Table 1

Baseline characteristics of obese adolescents with and without binge eating (BE)

Characteristics	BE* (N = 20)	Non-BE (N = 62)	*P*
Age, years	14.1 (1.2)	14.1 (1.2)	0.97
Weight, kg	103 (16)	103 (15)	0.99
BMI, kg/m^2	38.4 (3.8)	37.5 (3.8)	0.37
BMI z-score	2.5 (0.2)	2.4 (0.2)	0.12
Waist circumference, cm	110 (12)	109 (11)	0.74
Gender, N (%)			
Male	10 (37%)	17 (63%)	0.06
Female	10 (18%)	45 (82%)	
Race, N (%)			
Non-Caucasian	7 (19%)	30 (81%)	0.30
Caucasian	13 (29%)	32 (71%)	
Treatment Group, N (%)			
Placebo	13 (33%)	26 (67%)	0.07
Drug	7 (16%)	36 (84%)	

Values shown are mean (SD) or number (N) of participants.

*BE represents a collapsed variable including participants meeting full criteria for BED, as well as those who met sub-threshold criteria. These two conditions were collapsed into a single BE sample, due to a lack of difference between groups.

Table 2

Relationship of binge eating status to psychosocial variables at baseline

	BE* (N = 20)	Non-BE (N = 62)	Effect Size Cohen's D	*P*
Eating Behavior				
Three Factor Eating Inventory				
Hunger	7.6 (0.7)	4.1 (0.4)	1.11	< 0.0001
Disinhibition	8.9 (0.7)	5.5 (0.4)	1.08	< 0.0001
Cognitive Restraint	8.3 (0.8)	9.9 (0.5)	0.42	0.07
Depression				
Child Depression Inventory	7.3 (1.2)	6.3 (0.7)	0.18	0.49
Self-concept				
Piers Harris Total Score	56.1 (2.0)	59.6 (1.1)	0.41	0.12

Values shown are mean (SE).

* BE represents a collapsed variable including participants meeting full criteria for BED, as well as those who met sub-threshold criteria. These two conditions were collapsed into a single BE sample, due to a lack of difference between groups.

Table 3

Percentage change in initial BMI and absolute changes in BMI, BMI z-scores, weight, and waist circumference in BE and non-BE participants, adjusted by treatment group.

	BE (N = 20)		Non-BE (N = 62)		BE *P*, BE*Time *P* BE*Drug *P*
	Mo., 6	Mo., 12	Mo., 6	Mo., 12	
% change BMI	−7.0 (1.6)	−8.8 (2.4)	−6.9 (0.9)	−8.3 (1.3)	0.89, 0.84, 0.61
BMI, kg/m^2	−2.8 (0.6)	−3.3 (0.9)	−2.6 (0.3)	−3.1 (0.5)	0.83, 0.97, 0.73
BMI-Z score	−0.18 (0.05)	−0.29 (0.08)	−0.19 (0.03)	−0.30 (0.05)	0.87, 0.97, 0.63
Weight, kg	−5.3 (1.5)	−5.3 (2.5)	−6.1 (0.9)	−6.4 (1.4)	0.67, 0.85, 0.36
Waist Circumference, cm	−3.5 (2.1)	−5.5 (2.8)	−3.8 (1.1)	−4.7 (1.5)	0.93, 0.62, 0.10

Values shown are mean (SE).

Table 4

Number (percentage) of BE and non-BE participants who met different weight loss criteria at months 6 and 12.

	BE N (%)	Non-BE N (%)	*P*
Mo. 6, % change in initial BMI			0.36
<5%	13 (65%)	29 (47%)	
5–10%	3 (15%)	15 (24%)	
≥10%	4 (20%)	18 (29%)	
Mo. 12, % change in initial BMI			0.77
<5%	11 (55%)	29 (47%)	
5–10%	3 (15%)	9 (14%)	
≥10%	6 (30%)	24 (39%)	