Bupropion for Overweight Women With Binge-Eating Disorder: A Randomized, Double-Blind, Placebo-Controlled Trial

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ABSTRACT

Background: Binge-eating disorder (BED) is defined by recurrent binge eating (eating unusually large quantities of food during which a subjective loss of control is experienced), marked distress about the binge eating, and the absence of inappropriate weight compensatory behaviors. BED is strongly associated with excess weight, and many available psychological and pharmacologic approaches fail to produce much weight loss. The objective of this study was to perform a randomized placebo-controlled trial to evaluate the short-term efficacy of bupropion for the treatment of BED in overweight and obese women.

Method: Sixty-one overweight and obese (mean body mass index [BMI] = 35.8) women who met *DSM-IV-TR* research criteria for BED were randomly assigned to receive bupropion (300 mg/d) or placebo for 8 weeks. Participants were enrolled from November 2006 to December 2010. No dietary or lifestyle intervention was given. Primary outcome measures were binge-eating frequency and percent BMI loss. Secondary outcome measures were dimensional measures of eating disorder psychopathology, food craving, and depression levels.

Results: Eighty-nine percent (n = 54) of randomized participants completed the trial, without differential dropout between the bupropion and placebo groups. Mixed-effects analyses revealed significant time effects for all outcomes but no significant differences between bupropion and placebo on any outcome measure except for weight loss. Participants taking bupropion lost significantly more weight (1.8% vs 0.6% BMI loss; $F = 10.57$, $P = .002$).

Conclusions: Bupropion was well tolerated and produced significantly greater—albeit quite modest—short-term weight loss in overweight and obese women with BED. Bupropion did not improve binge eating, food craving, or associated eating disorder features or depression relative to placebo. Our findings do not support bupropion as a stand-alone treatment for BED. The preliminary findings regarding short-term weight losses suggest the need for larger and longer-term trials to evaluate the potential utility of bupropion for enhancing outcomes of psychological interventions that have demonstrated effectiveness for BED but fail to produce weight loss.

Trial Registration: ClinicalTrials.gov identifier: NCT00414167

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Binge-eating disorder (BED), included in Appendix B of the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision (*DSM-IV-TR*) as a research category,[1] is proposed as a formal diagnosis in *DSM-5*. BED is defined by recurrent binge eating (eating unusually large quantities of food during which a subjective loss of control is experienced), marked distress about the binge eating, and the absence of inappropriate weight compensatory behaviors. BED is a prevalent problem and is associated with obesity and heightened levels of medical and psychiatric comorbidity.[2] BED is distinct from the other eating disorders and obesity[3–5] and is more prevalent than the other eating disorders (anorexia and bulimia nervosa).[2]

Critical review and meta-analysis of the treatment literature for BED concluded that several different medications—through varied mechanisms—have short-term efficacy relative to placebo for reducing binge eating and producing modest weight loss.[6] Randomized clinical trials of antidepressant SSRIs have reported mixed results for the remission of binge eating[7–10] and little or no weight losses. Studies of fluoxetine, fluvoxamine, sertraline, and citalopram have reported weight loss differences of approximately 0–3 kg compared to placebo, depending on the duration of the trial (see Reas and Grilo[6] for summary and effect sizes across studies). Placebo-controlled trials of the currently available antiobesity medication orlistat[11,12] (sibutramine has been recently withdrawn from the market) have reported significant but modest effects for producing weight loss but not for reducing binge eating. Placebo-controlled trials of the anticonvulsants topiramate[13,14] and zonisamide[15] have reported significant effects for reducing both binge eating and weight (with a mean improvement in weight loss of approximately 3–4 kg compared to placebo) but have also reported high rates of dropout and adverse events. Psychological treatments, such as cognitive-behavioral therapy (CBT), are effective for reducing binge eating but do not reduce weight.[8,16]

The association between BED and obesity[2] and the possible heightened risk for developing future metabolic problems[17] highlight the need to find methods to effectively reduce weight—in addition to eliminating binge eating—in persons with BED.[18] Bupropion is an antidepressant that has been shown to be an efficacious treatment for smoking cessation.[19–22] Bupropion, a selective reuptake inhibitor of dopamine and norepinephrine, reduces cravings for nicotine[21,23] and food[24] potentially by increasing extracellular dopamine.[25] Bupropion has shown some promise for treating obesity in 2 placebo-controlled trials.[24,26] Anderson et al[26] reported that both 300-mg/d and 400-mg/d bupropion dosing produced significantly greater weight loss than placebo when paired with a calorie deficit of 600 kcal/d. Jain and colleagues,[24] in a study with obese patients with subclinical levels of depression, reported that 300 mg/d of bupropion resulted in significantly greater weight losses than placebo when added to

a 500-kcal/d deficit diet. Since food cravings and negative mood are thought to contribute to binge eating,[27–29] and because both the norepinephrine[30] and dopamine[31] systems have been implicated in binge eating, bupropion may be a promising psychopharmacologic agent for treating overweight persons with BED.

The current study was a randomized double-blind trial designed to test the efficacy of bupropion in the treatment of overweight and obese women with BED. Patients were randomly assigned to receive bupropion 300 mg/d or placebo for 8 weeks. No dietary or lifestyle intervention was given. It was hypothesized that, compared to placebo, bupropion would result in significantly greater decreases in the frequency of binge-eating episodes and body weight among women diagnosed with BED. Secondary hypotheses were that, compared to placebo, bupropion would produce greater reductions in negative mood, food cravings, and eating disorder psychopathology.

Clinical Points

- Binge-eating disorder is strongly associated with excess weight, and many available psychological and pharmacologic approaches fail to produce much weight loss.
- In this 8-week randomized controlled trial, bupropion was well tolerated and produced significantly greater—albeit quite modest—short-term weight loss in overweight and obese women with binge-eating disorder.
- Bupropion did not improve binge eating, food craving, or associated eating disorder features or depression relative to placebo.

METHOD

Participants

Participants were 61 overweight or obese (BMI ≥ 25) women with BED recruited via advertisements for overweight women seeking treatment for binge eating and weight loss. Participants were enrolled from November 2006 to December 2010, and the study was registered with ClinicalTrials.gov (identifier: NCT00414167). The study received full institutional review board approval. Participants provided written informed consent prior to completing assessments. Entrance criteria required that participants meet full *DSM-IV-TR* research criteria for BED, have a BMI in the range of 25–50, and be 18–65 years of age. Exclusion criteria were as follows: diabetes; seizure disorders; uncontrolled hypertension; hypothyroidism; current pregnancy or breastfeeding; history of severe renal, hepatic, neurologic, chronic pulmonary disease, or other unstable medical disorder; gallbladder disease; current medications or herbal supplements with psychoactive properties; current treatment for eating/weight; serious psychiatric disorder that warrants a higher level of treatment (eg, bipolar disorder, current substance use disorder); and homicidal or suicidal ideation. A history of anorexia nervosa or bulimia nervosa was an exclusion criterion due to previous research showing elevated risk of grand mal seizure among bulimic patients taking bupropion.[32] The study was powered a priori to detect a difference of 1.7 binge episodes per week between conditions with 80% power using a total sample size of 56 and α level of .05.

The 61 randomized female participants had a mean age of 44.1 (SD = 12.5) years and mean BMI of 35.8 (SD = 6.8). Eighty-eight percent (n = 54) of the participants attended college; 84% (n = 51) were white, 8% (n = 5) were African-American, 5% (n = 3) were Hispanic, and 3% (n = 2) were "other" ethnicity.

Procedure

Treatment assignment was performed independently from the investigators by a research-pharmacist at a separate Yale facility. Participants were randomly assigned with stratification by obesity grade (ie, BMI 25–29.9 = overweight, 30–34.9 = grade 1 obesity, 35–39.9 = grade 2 obesity, ≥ 40 = grade 3 obesity) and by smoking history (current and former versus never) to bupropion or placebo. We stratified by smoking status because it is possible that smoking history/status could moderate treatment outcomes.[33] In accordance with the operational definition of smoking used in Healthy People 2010,[34] participants were classified as smokers if they reported smoking over 100 cigarettes in their lifetime. Never-smokers were those who denied smoking over 100 cigarettes in their lifetime.

This randomized double-blind treatment trial was designed to test the efficacy of bupropion-only treatment with no additional dietary or lifestyle intervention. Participants were instructed to continue eating in their typical pattern for the duration of the 8-week trial. To ensure concealment of the randomization, medication (bupropion, placebo) was prepared in identical-appearing capsules. The sustained-release bupropion (bupropion SR) 300-mg/d schedule consisted of bupropion SR 150-mg tablets taken once daily for the first 3 days, then taken twice daily for study days 4–56. The placebo administration followed the same schedule. Assessment appointments occurred every 2 weeks, during which participants were weighed and evaluated for adverse events and self-monitoring records (described later in the Method) were collected.

Assessments

The assessments allowed for determination of diagnoses and eligibility, for characterization of baseline functioning and measurement of clinical changes, and for ongoing assessment of safety and compliance with treatment. Participants were screened and evaluated using clinical and diagnostic interviews conducted by doctoral-level research-clinicians trained in eating and weight disorders.

The Structured Clinical Interview for *DSM-IV* Axis I Disorders-Patient Version[35] was used to determine BED diagnosis and Axis I psychiatric lifetime and current comorbidity. A questionnaire with interview follow-up assessed smoking status and history. The Eating Disorder Examination (EDE) interview,[36] a semistructured, investigator-based interview, was administered to assess eating disorder psychopathology and to confirm the BED diagnosis. The EDE focuses

on the previous 28 days except for diagnostic items, which are rated for *DSM-IV-TR* duration stipulations. The EDE assesses the frequency of objective bulimic episodes (ie, binge eating defined as unusually large quantities of food with a subjective sense of loss of control), which corresponds to the *DSM-IV-TR* definition of binge eating. The EDE also assesses subjective bulimic episodes, which are marked by a subjective sense of loss of control over eating smaller amounts of food. The EDE comprises 4 subscales (dietary restraint, eating concern, weight concern, and shape concern) and a total global score. Items are rated on 7-point forced-choice scales (range, 0–6), with higher scores reflecting greater severity/frequency. The EDE has well-established interrater and test-retest reliability[37] and validity.[38] In the present study, interrater reliability, determined using 21 clinician pairs, was excellent, with reliability coefficients of 0.84 for binge-episode days and 0.72–0.91 for subscales.

Table 1. Demographic and Psychological Characteristics of Participants[a]

Characteristic	Placebo (n = 30)	Bupropion (n = 31)	Total (N = 61)	Test Statistic	*P* Value
Age, mean (SD), y	43.1 (13.0)	45.2 (12.1)	44.1 (12.5)	$F = 0.412$	.523
Race, nonwhite	3 (10.0)	7 (22.6)	10 (16.4)	$\chi^2 = 1.761$	.185
Education, completed college	19 (63.3)	19 (61.3)	38 (62.3)	$\chi^2 = 0.027$	.869
Axis I comorbidity, lifetime	24 (80.0)	21 (67.7)	45 (73.8)	$\chi^2 = 1.184$	.277
Mood disorder, lifetime	16 (53.3)	16 (51.6)	32 (52.5)	$\chi^2 = 0.018$	.893
Anxiety disorder, lifetime	13 (43.4)	10 (32.3)	23 (37.7)	$\chi^2 = 0.796$	.372
Substance use disorder, lifetime	10 (33.3)	5 (16.1)	15 (24.6)	$\chi^2 = 2.434$	.119
Smoking, lifetime	17 (56.7)	14 (45.2)	31 (50.8)	$\chi^2 = 0.807$	.369

[a]Values are expressed as n (%) unless otherwise noted.

Self-Report Measures

The Food Craving Inventory (FCI)[39,40] was administered at baseline and at all assessment visits. The FCI is a brief measure of specific food cravings and has been validated for use with obese patients with BED.[40] The FCI assesses the frequency of cravings over the past month and generates 4 subscales (high fats, sweets, carbohydrates/starches, and fast food fats) and a total score. The Beck Depression Inventory (BDI)[41] 21-item version is a well-established self-report[42] measure of symptoms of depression. The BDI was administered at baseline and at all assessment visits.

Self-Monitoring

Participants were instructed to monitor and record binge-eating episodes in written food diaries (Self-Monitoring[38,43]). Self-monitoring instruction was based on EDE definitions of different types of overeating episodes involving the loss of control (ie, objective bulimic episodes and subjective bulimic episodes). Each daily record specifically asked whether participants had any objective bulimic episodes and subjective bulimic episodes and, if so, how many. Patients self-monitored on a daily basis throughout treatment, and records were reviewed at each assessment visit for completeness. This prospective method of obtaining binge-eating data overcomes a number of pitfalls of retrospective methods.[38,43] At the first clinic appointment, prior to administration of the medication, all participants retrospectively self-reported the frequency of binge episodes occurring over the previous 7 days using EDE definitions. This frequency was used as the baseline measure of binge episodes.

Assessment visits occurred at weeks 2, 4, 6, and 8 (posttreatment). At each assessment visit, participants were weighed and self-monitoring forms were collected. At weeks 0, 2, 4, 6, and 8, participants completed the FCI and BDI and were evaluated for side effects/adverse events. Participants were paid $100 following completion of the 8-week assessment.

Analytic Plan

Data were analyzed using SPSS Version 19 (IBM Corp; Armonk, New York). Baseline analyses (*t* and χ^2 tests) tested for group differences on demographic variables. Rate of change in binge frequency and weight change (percent BMI loss) between the bupropion and placebo groups (the primary study outcomes) were tested using mixed-effects regression for continuous outcomes.[44,45] Mixed-effects models allow for different numbers of observations per subject, use all available data on each subject, and are unaffected by randomly missing data. Mixed-effects models also have the capacity to test and account for individual difference contributions to the treatment outcome.[46–48] Mixed-effects regression also provides flexibility in modeling the correlation structure of the data. For these analyses, time, medication group, and their interaction were tested, as well as random subject effects. Several different error structures were considered (eg, AR1, independence), and the best-fitting structure was selected based on information criteria. Data on subjects who discontinued the study protocol but had subsequent measurements were used in the primary analyses as randomized. A series of complementary binary logistic regression analyses tested the effects of bupropion versus placebo on the categorical outcome of remission from binge eating. Remission was defined as 28 days of continuous abstinence from binge eating.

RESULTS

Overall, 93 participants were evaluated for eligibility, and 61 were randomized to bupropion (n = 31) or placebo (n = 30). The 32 participants who were not randomized to treatment were excluded due to non-interest or for failure to meet eligibility criteria. Fifty-four of the 61 randomized participants (89%) completed the trial, and all participants who remained in the study at 8 weeks completed the posttreatment assessment. The 2 treatment conditions (bupropion vs placebo) did not differ significantly in completion rates (87% [27/31] bupropion vs 90% [27/30] placebo; $\chi^2 = 0.13$, $P = .72$). Three participants voluntarily withdrew from the placebo condition (no reason given), 3 withdrew from the bupropion condition (no reason given), and 1 was withdrawn from the bupropion condition due to a medical event. No medication-related adverse events were reported. No seizures were reported. The 2 treatment groups did not differ significantly on any demographic feature (Table 1).

Table 2. Clinical Measures at Baseline and Posttreatment[a]

	Placebo (n = 30)				Bupropion (n = 31)			
	Week 0		Week 8		Week 0		Week 8	
Measure	Mean	SD	Mean	SD	Mean	SD	Mean	SD
BMI[b]	35.4	7.1	35.2	7.4	36.2	6.6	35.7	6.6
EDE subscales[c]								
Dietary restraint	1.8	1.2	1.6	0.8	1.6	1.2	1.4	1.0
Eating concern	2.0	1.4	1.1	1.3	1.8	1.2	1.0	1.2
Shape concern	3.7	1.1	2.9	1.5	3.5	1.4	2.4	1.3
Weight concern	3.3	1.0	2.6	1.0	3.2	1.2	2.6	1.0
EDE global[c]	2.7	0.8	2.0	0.9	2.5	1.1	1.8	0.9
Objective binge episodes								
EDE, monthly[b]	13.6	6.5	6.3	8.0	17.8	11.9	5.0	9.4
Self-report, per week[b,d]	3.0	2.6	1.0	1.5	3.3	3.3	0.8	1.2
Subjective binge episodes								
EDE, monthly[b]	10.3	14.2	7.5	8.4	13.5	11.2	9.3	21.4
Self-report, per week[b,d]	2.7	3.4	2.3	2.4	3.6	2.7	2.2	4.2
Beck Depression Inventory[b]	10.8	6.1	8.7	7.2	13.4	9.8	8.0	8.3
Food Craving Inventory total[b]	2.4	0.7	2.0	0.6	2.6	0.6	2.0	0.6

[a]Missing values at week 8 (n = 7) replaced with baseline values.
[b]Administered at all assessment visits (every 2 weeks).
[c]Administered at baseline and posttreatment.
[d]Via daily monitoring.
Abbreviations: BMI = body mass index, EDE = Eating Disorder Examination.

Table 3. Mixed-Effects Models

	Posttreatment Value Different From Zero		Time		Medication	
Measure	*F*	*P*	*F*	*P*	*F*	*P*
Percent BMI[a] loss	4.14	.046	5.65	.021	10.57	.002
EDE subscales[b]						
Dietary restraint	116.47	.000	4.08	.045	1.39	.242
Eating concern	101.81	.000	85.13	.000	0.84	.361
Shape concern	303.49	.000	65.82	.000	1.71	.195
Weight concern	315.71	.000	28.34	.000	0.05	.816
EDE global[b]	335.94	.000	122.08	.000	2.06	.154
Objective binge episodes						
EDE[b]	121.44	.000	55.35	.000	0.08	.775
Self-report[a,c]	17.95	.000	7.78	.007	2.01	.162
Subjective binge episodes						
EDE[b]	22.54	.000	2.89	.095	0.96	.331
Self-report[a,c]	10.53	.002	6.32	.015	0.47	.498
Beck Depression Inventory[a]	28.27	.000	22.65	.000	0.04	.843
Food Craving Inventory total[a]	319.41	.000	63.07	.000	0.10	.755

[a]Administered at all assessment visits (every 2 weeks).
[b]Administered at baseline and posttreatment.
[c]Via daily monitoring.
Abbreviations: BMI = body mass index, EDE = Eating Disorder Examination.

Table 2 summarizes BMI and the clinical measures at baseline and posttreatment (week 8). At baseline, the bupropion and placebo groups did not differ significantly on BMI or on any of the clinical measures (analyses of variance not reported; range of *P* values, .09–.94).

Table 3 summarizes results of the mixed-effects models testing change in clinical outcomes. Significant effects for time were found for all variables, indicating all clinical outcomes improved significantly over the course of the study. Mixed-effects models revealed that bupropion and placebo differed significantly for percent BMI loss but not for any other outcome variable. Bupropion and placebo groups did not differ on binge-eating frequency, any measures of eating psychopathology, food craving, or depressed mood at posttreatment. Figure 1 shows the pattern of weight loss at each time point throughout the study. Participants in the bupropion condition lost a mean of 1.8% (SD = 2.6%) of their BMI, and participants in the placebo condition lost a mean of 0.6% (SD = 2.1%) of their BMI. Expressed in terms of absolute weight loss, participants in the bupropion condition lost 1.68 kg (SD = 2.59) after 8 weeks on the study medication, compared to 0.43 kg (SD = 2.12) for participants taking placebo. Figure 2 shows the weekly binge-eating frequency and remission rates by treatment group. The test for medication versus placebo on rates of change in binge eating (Table 3) was not statistically significant when the change was measured continuously ($P = .16$) nor when it was evaluated categorically in terms of remission from binge episodes, defined as no binge episodes during the past 4 weeks (42% [n = 13] for bupropion vs 27% [n = 8] for placebo; $\chi^2_1 = 1.58$ [n = 61], ϕ coefficient = 0.16, $P = .21$).

DISCUSSION

This randomized double-blind controlled trial tested the short-term efficacy of bupropion in the treatment of overweight and obese women with BED. Overall, the medication was well tolerated, and no medication-related adverse events were experienced. The bupropion and placebo groups did not differ in rate of dropout from the study, which was low overall, with 89% of the participants completing treatment. After 8 weeks of treatment, significant improvements were observed for eating-specific and general psychopathology, although these improvements did not differ significantly between bupropion and placebo. Significant overall improvements were observed in terms of reduced frequency of binge eating and binge-eating remission (defined as zero objective bulimic episodes for the last 28 days of treatment), although the bupropion and placebo groups did not differ significantly in remission rates (42% vs 27%, respectively).

However, 300 mg/d of bupropion resulted in significantly greater weight loss (1.8% or 1.68 kg) compared to placebo (0.6% or 0.43 kg). Collectively, our findings do not support bupropion as a stand-alone treatment for BED. The findings regarding short-term weight losses—significant albeit very modest during this brief 8-week trial—suggest the need for larger and longer-term trials to evaluate the potential utility of bupropion for enhancing outcomes of psychological interventions that have demonstrated efficacy for BED but fail to produce weight loss.

The remission rates and percent reductions in binge-eating frequency observed in this pharmacotherapy-only

Figure 1. Body Mass Index (BMI) Loss for (A) Completer Data and (B) Intent-to-Treat (ITT; missing values replaced with 0% BMI loss)

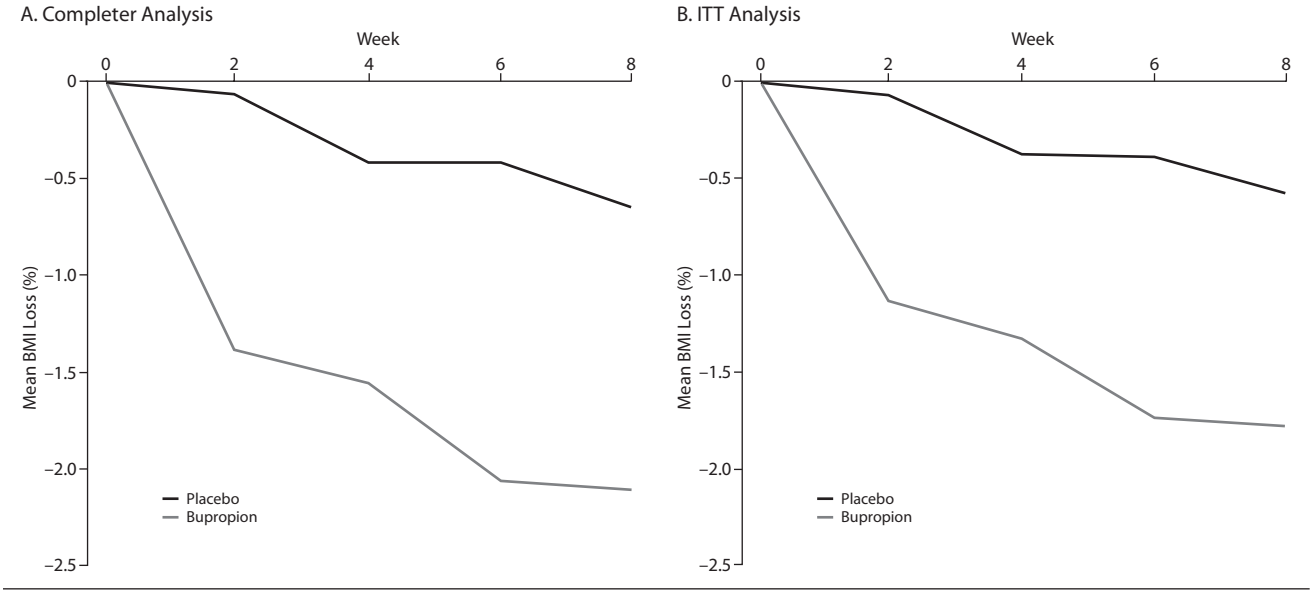
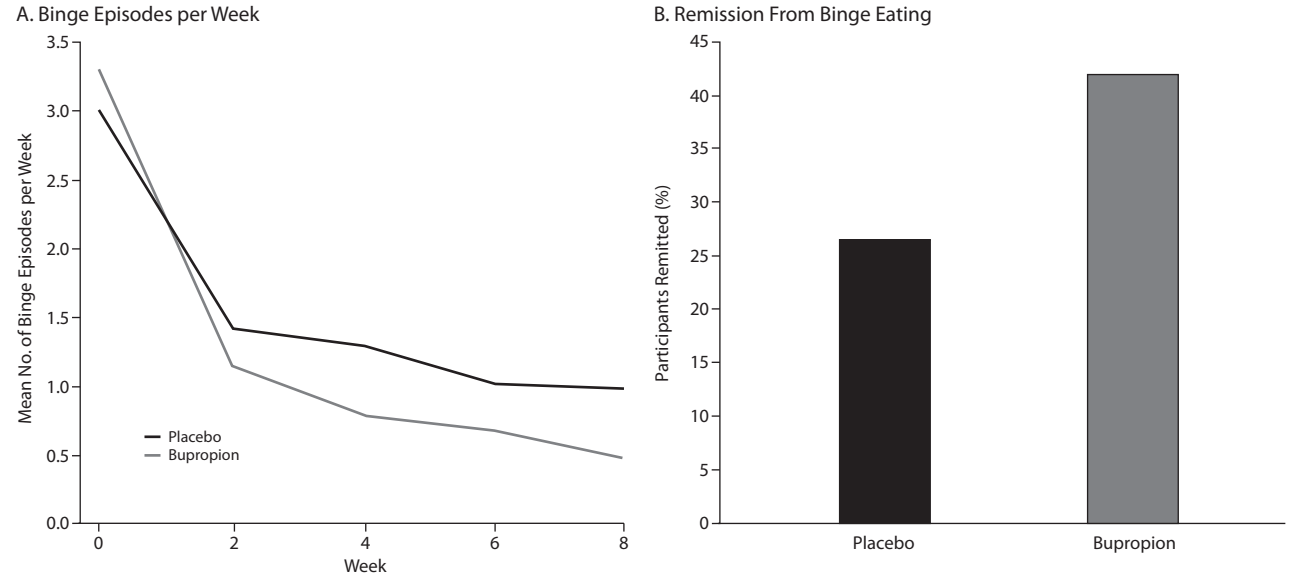


Figure 2. Improvement in Binge Eating During the 8-Week Study Phase



trial are comparable in magnitude to those observed generally in pharmacotherapy-only trials for BED.[6] For example, Wilfley et al[49] reported remission rates (defined as zero objective bulimic episodes in the last 2 weeks of treatment) of 44% for sibutramine and 30% for placebo. The magnitude of differences between bupropion and placebo for binge-eating outcomes, however, was not statistically significant, and this finding adds to the mixed literature for pharmacotherapy-only treatment[8,50] (see Reas and Grilo[6] for review). Such results may perhaps reflect partly the relatively limited sample size and insufficient power to detect small effect sizes. It is unlikely, though, that the brief length of the present trial obscured potential effects on binge eating given that response to antidepressant treatments for BED is generally quite rapid and observed by 4 weeks.[51] However, given its unique mechanism of action, bupropion cannot be directly compared to other classes of pharmacotherapy (eg, anticonvulsants, weight loss medications) that have shown some efficacy in reducing binge eating.[6]

Weight losses at 8 weeks were comparable to previous reports with several different medications tested for obese patients with BED, except for the reports of greater weight losses with topiramate,[6] but were less than those reported in 2 previous randomized controlled trials of bupropion

for the treatment of obesity without comorbid BED.[24,26] Importantly, the present study tested medication only, whereas the previous obesity trials tested bupropion and placebo administered concurrently with reduced calorie diets and for longer durations.[24,26] Additionally, the dosing in the current study (300 mg/d) was less than in previous studies that administered in doses of 400 mg/d[26] and 300 mg/d, increasing to 400 mg/d in the event of modest initial weight loss.[24] Future studies should test bupropion in longer trials, at higher dosing, and administered concurrently with either CBT or behavioral weight loss treatment to determine whether there is an additive effect. Previous research employing these designs with other specific pharmacologic agents has found limited benefit of medication over and above that provided by CBT or behavioral weight loss treatments in terms of binge eating, although it has found an advantage for certain medication in terms of weight loss. For example, Grilo et al[12] found no differences in binge-eating remission or percent reduction in binge eating between orlistat and placebo administered alone or in combination with CBT. However, the CBT + orlistat group lost significantly more weight than the group taking placebo. Similarly, Claudino et al[14] reported that reductions in binge-eating frequency did not differ for topiramate and placebo administered concurrently with CBT, although the topiramate group lost significantly more weight after 21 weeks. A slightly longer trial[11] comparing orlistat to placebo, with concurrent administration of a calorie-reduced diet, found greater weight loss at 24 weeks for orlistat, but no differences in terms of binge-eating frequency at posttreatment.

This study has several strengths and limitations that should be noted as context for interpreting its findings. The study completion rate was quite high, with 89% of participants completing the trial and providing posttreatment data. It should be noted, however, that subject payment ($100 for completing the posttreatment evaluation) may have contributed partly to our high retention rates. The study also enrolled overweight (ie, BMI ≥ 25 and < 30) as well as obese (BMI ≥ 30) women into the study, which increases its generalizability. Finally, because of the possibility that smoking history/status could moderate treatment outcomes,[33] participants were stratified by smoking history/status to ensure equivalence across cells. Limitations include the short study duration and sample size; it is possible, for example, that longer treatment might have resulted in greater weight losses, or that a larger sample size with greater power might have allowed statistical detection of smaller differences between active and placebo conditions. The lack of follow-up after medication discontinuation is also a limitation; unfortunately, this is the case for all published randomized controlled trials of medication-only treatments for BED to date,[6] except for 1 recent report.[52] With this context, we cautiously conclude that our findings do not support bupropion as a stand-alone treatment for BED, although the findings regarding short-term weight losses suggest the need for longer-term trials to evaluate further the potential utility of bupropion for enhancing outcomes of other interventions that have demonstrated efficacy for BED but fail to produce weight loss.

Drug names: bupropion (Wellbutrin, Aplenzin, and others), citalopram (Celexa and others), fluoxetine (Prozac and others), orlistat (Xenical), sertraline (Zoloft and others), topiramate (Topamax and others), zonisamide (Zonegran and others).

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Editor's Note: We encourage authors to submit papers for consideration as a part of our Focus on Women's Mental Health section. Please contact Marlene P. Freeman, MD, at mfreeman@psychiatrist.com.