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Evaluation of a healthy-weight treatment program for bulimia nervosa: A preliminary randomized trial

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Abstract

Objective—Conduct a randomized treatment trial to test whether healthy dieting maintains bulimic symptoms or effectively reduces this eating disturbance.

Methods—Female participants ($n = 85$) with full- and sub-threshold bulimia nervosa were randomly assigned to a 6-session healthy dieting intervention or waitlist condition and assessed through 3-month follow-up.

Results—Relative to control participants, intervention participants showed modest weight loss during treatment and demonstrated significant improvements in bulimic symptoms that persisted through follow-up.

Discussion—These preliminary results suggest that this intervention shows potential for the treatment for bulimia nervosa and may be worthy of future refinement and evaluation. Results also provide experimental evidence that dieting behaviors do not maintain bulimia nervosa, suggesting the need to reconsider maintenance models for this eating disorder.

Keywords

Bulimia; Dieting; Maintenance; Treatment

Introduction

Bulimia nervosa is characterized by a chronic course, medical complications, and functional impairment, and increases risk for future onset of obesity, depression, suicide attempts, anxiety disorders, substance abuse, and health problems (Johnson, Cohen, Kasen, & Brook, 2002; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Wilson, Becker, & Heffernan, 2003). Thus, much effort has been devoted to identifying risk and maintenance factors for this pernicious disorder.

Theorists have suggested that dieting increases risk for the onset and maintenance of bulimia nervosa (Fairburn, 1997; Hawkins & Clement, 1984; Polivy & Herman, 1985; Stice & Agras, 1998). Dieting has been defined as intentional and sustained restriction of food intake for the purposes of weight loss or weight maintenance (Herman & Polivy, 1975; Laessle, Tuschl, Kotthaus, & Pirke, 1989; Wadden, Brownell, & Foster, 2002). According to Polivy and Herman (1985), “Successful dieting produces weight loss, which in turn might create a state of chronic hunger, especially if such weight loss leaves the dieter at a weight below the set-point weight that is defended physiologically” (p. 196). The chronic hunger experienced by dieters

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putatively increases the likelihood of binge eating. Polivy and Herman (1985) also argue that a reliance on cognitive controls over eating leaves dieters vulnerable to uncontrolled eating when these cognitive processes are disrupted. Binge eating putatively precipitates redoubled dietary efforts and use of radical weight-control techniques, such as vomiting, which develop into the self-maintaining binge–purge cycle (Fairburn, 1997). In support, longitudinal studies have found that self-reported dieting is a potent predictor of future onset of bulimic symptoms and full- and sub-threshold bulimia nervosa (Field, Camargo, Taylor, Berkey, & Colditz, 1999; Killen et al., 1994, 1996; Stice, 2001; Stice & Agras, 1998; Stice, Presnell, & Spangler, 2002). Elevated self-reported dieting also predicted maintenance of bulimic symptoms in one study (Stice & Agras, 1998), but this effect did not replicate in a second study (Fairburn et al., 2003).

Based on this theory, the treatment of choice for bulimia nervosa, cognitive-behavioral therapy (CBT), aims primarily to reduce dieting (Fairburn, Marcus, & Wilson, 1993). However, the evidence that interventions that do not focus on reducing dieting also decrease bulimic symptoms relative to assessment-only control conditions, including interpersonal therapy, dialectical-behavior therapy, and exercise-based interventions (Fairburn, Jones, Peveler, Hope, & O'Connor, 1993; Safer, Telch, & Agras, 2001; Sundgot-Borgen, Rosenvinge, Bahr, & Schneider, 2002), seems incompatible with the assertion that dieting is the primary factor that maintains bulimia nervosa. In addition, randomized trials have found that assignment to low-calorie weight loss diets (e.g., 1200 calories a day) results in significantly greater decreases in binge eating and bulimic symptoms for normal weight, overweight, and obese adolescent and adult women relative to waitlist controls (Goodrick, Poston, Kimball, Reeves, & Foreyt, 1998; Klem, Wing, Simkin-Silverman, & Kuller, 1997; Presnell & Stice, 2003; Reeves et al., 2001). Participants in the weight loss diet conditions of these trials lost significant weight, thereby confirming that dietary restriction was manipulated. Assignment to a more moderate weight-maintenance diet likewise resulted in greater decreases in bulimic symptoms in normal weight adolescent females over a 1-year period, relative to assessment-only controls (Stice, Presnell, Groesz, & Shaw, 2005).

It appears that the prospective studies generated results that conflicted with the experimental trials because the dietary restraint scales used in the former studies are not valid measures of dietary restriction. Dietary restraint scales do not show substantively meaningful or statistically significant inverse correlations with objective and unobtrusive measures of acute or longer-term caloric intake (Bathalon et al., 2000; Jansen, 1996; Martin et al., in press; Stice, Fisher, & Lowe, 2004; van Strien, Cleven, & Schippers, 2000), as was suggested by the original validity studies that used self-reported caloric intake (Laessle et al., 1989; van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986). The evidence that individuals with elevated scores on these scales gain more weight over both short and long periods of time than their low-scoring counterparts (Klesges, Isbell, & Klesges, 1992; Klesges, Klem, & Bene, 1989; Stice, 2001; Stice et al., 1999) also suggests these measures are not valid measures of longer-term caloric restriction. This analysis suggests that more confidence should be placed in the randomized trials that confirmed that participants were on a weight loss diet, as reflected by significant weight loss relative to controls.

Based on the promising evidence from controlled trials suggesting that weight management interventions result in decreased bulimic symptoms in various populations, the primary objective of this study is to examine the efficacy of a healthy weight control intervention for the treatment of bulimia nervosa. This new intervention, the Healthy Weight Program, has two advantages over extant treatments, such as a shorter duration (thereby reducing time commitment for clients) and a secondary possible benefit of weight loss, which will not only increase the appeal of the program to those dissatisfied with current body weight, but might also reduce health risks associated with elevated weight. If the present study were able to

establish initial evidence for the efficacy of this 6-session intervention, it would provide preliminary empirical support for an alternative, effective treatment that is less costly to implement.

This trial might also be conceptualized as an experimental psychopathology test of the theory that dietary restriction maintenances bulimia nervosa. We believe it is vital to resolve the question of whether dieting maintains bulimic pathology or is an efficacious intervention for this eating disturbance because of the diametrically opposed clinical implications. This is a particularly important question from a health perspective because obesity results in considerably more morbidity and mortality than eating disorders and dietary restriction is the current intervention of choice for obesity prevention and treatment. Although several controlled trials have found that low calorie diets result in decreased bulimic symptoms among individuals without a bulimia nervosa diagnosis, no prior trial has tested whether an intervention that promotes dietary restriction could serve as an efficacious treatment for this disorder. We hypothesize that an intervention that promotes a negative energy balance (i.e., intake fewer calories than are output) would produce significantly greater reductions in bulimic symptoms relative to a waitlist control group among individuals with bulimia nervosa.

Finally, it has been proposed that it is important to demonstrate that treatment interventions impact clinically meaningful outcomes (Kazdin & Weisz, 1998). Therefore, this study tested whether treatment intervention participants, by way of a reduction in bulimic symptoms, evidence greater improvements in psychosocial functioning because these symptoms are thought to contribute to poor social adjustment (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000). This study also tested whether treatment produced greater decreases in health care utilization, because individuals with bulimia seek treatment for a variety of nonspecific health problems at higher rates than the normal population (Johnson, Spitzer, & Williams, 2001).

Methods

Participants and procedures

Study participants ($n = 85$) were recruited from a university (78.8%) and the general community (21.2%) in a metropolitan area of the Southwestern US. Printed advertisements invited bulimic individuals to participate in a program promoting a healthier way to "achieve a healthy weight, increase body satisfaction, and break the binge–purge cycle." Since bulimic pathology is very rare among males (Wilson et al., 2003), only female participants were recruited. Because almost half of the presenting cases at eating disorder treatment clinics are diagnosed with partial syndrome, rather than full syndrome, eating disorders (Fisher, Schneider, Burns, Symons, & Mandel, 2001; Williamson, Gleaves, & Savin, 1992), and because even individuals who meet only sub-threshold levels of bulimic pathology experience subjective distress and functional impairment (Lewinsohn, Striegel-Moore, & Seeley, 2000), sub-threshold participants were included in the sample. Indeed, other recent trials have included sub-threshold patients (e.g., Ghaderi & Scott, 2003; Safer et al., 2001).

The resultant sample ranged in age from 18 to 55 years ($M = 21$ years; SD = 5.3) and was composed of 9% Asians, 2% Blacks, 22% Hispanics, 53% Caucasians, and 13% who specified "other" or mixed racial heritage. Educational attainment of parents, a proxy for socioeconomic status, in our sample (18% high school graduate or less; 20.5% some college; 34% college graduate; 25% graduate degree) was somewhat higher than regional census data (34% high school graduate or less; 25% some college; 26% college graduate; 15% graduate degree; U.S. Bureau of the Census, 2000).

Interested participants were screened for eligibility and provided with information about the study. Participants were required to report a minimum of 4 binge-eating episodes and 4

compensatory behavior episodes in the past month to be eligible. Participants were excluded if they had received other treatment (including medication) for an eating disorder within the past month. Further exclusion criteria were current low weight (body mass index [BMI]<19), a physical or medical condition hindering ability to make dietary or exercise changes (e.g., thyroid condition, current injury), current suicidal ideation, or a lifetime history of schizophrenia or bipolar disorder.

Participants who met eligibility requirements were randomly assigned to either the Healthy Weight Program ($n = 43$) or to a waitlist comparison group ($n = 42$). At baseline, mid-treatment, posttest, and 3-month follow-up, participants completed a survey and a semi-structured interview. Participants received $10 at each of the first three assessment periods, and $20 for the final assessment. To minimize attrition, participants received reminder telephone calls the day before each scheduled session or assessment. If participants missed a scheduled assessment or group session, a make-up meeting was scheduled. Bulimic symptoms were monitored throughout the study and participants were informed that referrals were available should they desire adjunctive treatment. Waitlist participants were informed that they could participate in the intervention condition at the conclusion of the follow-up assessment.

Healthy weight intervention condition

The Healthy Weight Program was designed to provide alternative tools for achieving a healthy body weight that are more effective and healthier than bulimic compensatory behaviors. Thus, the purpose of the healthy weight intervention is to *only* promote healthy weight control behaviors that would induce a slight negative energy balance by asking participants to make lasting healthy changes in their caloric intake and exercise output. The primary focus of the sessions were to promote a variety of behavior changes, such as cutting out high-fat foods and increasing exercise, that will allow the participant to induce a negative energy balance. The intervention also provides basic nutritional knowledge that may help facilitate these changes (e.g., information about calcium is provided since it aids in maintaining bone strength essential for prolonged exercise, and has also been shown to aid weight loss efforts). Binge eating and compensatory behaviors are never directly addressed or targeted for reduction. What differentiates this program from CBT is that weight management is encouraged, rather than discouraged. In fact, facilitators utilize participant drive for thinness as a motivator to achieve a slim, but healthy figure through healthy means. In other words, this intervention, unlike CBT, promotes caloric restriction and leaves the pursuit of the thin ideal intact.

In the current study, the intervention groups consisted of a small number of participants (6–8) and were led by a Master's level graduate student. The intervention consists of six sessions held weekly for the first four sessions, then every other week for the remaining two sessions. The first four sessions focus on strategies for decreasing intake of high calorie foods (e.g., choosing lower calorie substitutes, substituting another activity for snacking when bored) and increasing exercise (e.g., choosing an activity that is fun or can be done with a friend, providing small rewards for meeting exercise goals). Throughout these sessions, participants are provided with information and handouts about the importance of balanced nutrition (e.g., eating a variety of foods), particular weight-loss benefits of certain types of foods (e.g., fiber, calcium) and exercise safety and tips. At the end of each session, participants are asked to make a public commitment to the group for one moderate dietary goal (e.g., decrease late-night snacking) and exercise goal (e.g., go for 30-min walk 3 times per week) of their own choosing. At the beginning of each subsequent session, participants reviewed their progress with the group and group members assisted each other in troubleshooting problems that interfere with progress towards goals. If participants are already exercising too much or eating a very healthy diet, they are encouraged to enhance what they are already doing by varying exercise activity (e.g., including weight training) or fine-tuning diet (e.g., eating more vegetables, taking a vitamin

supplement). The facilitator monitored progress, helped participants select realistic and manageable goals, and provided support and encouragement. Participants were given food and exercise records at the end of each session for homework to help track their progress throughout the week. The final two sessions focused on maintenance of diet and exercise changes, troubleshooting future barriers to healthy eating and exercise, and longer-term goal setting. In all sessions, participants were encouraged to support one another and assist each other in troubleshooting problem areas.

Measures

Bulimic symptoms—The diagnostic items from the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a semi-structured investigator-based interview, assessed DSM-IV bulimia nervosa symptoms. The primary outcomes for this trial were total binge episodes in the past month and total compensatory behaviors in the past month, as well as bulimia nervosa diagnoses. A normalizing square root transformation was applied to the two frequency variables. Participants who reported at least 8 binge-eating episodes and 8 compensatory behavior episodes in the past month as well as weight and shape overvaluation received a full-threshold bulimia nervosa diagnosis; those reporting fewer binge eating and/or compensatory behavior episodes (at least 4 of each in the past month) and weight and shape overvaluation received a sub-threshold diagnosis. The EDE exhibits good internal consistency (α = .76–.90), inter-rater reliability (κ = .70–.99), and test–retest reliability (*M r* = .80), and discriminates between eating disordered individuals and controls (Fairburn & Cooper, 1993; Rizvi, Peterson, Crow, & Agras, 2000; Williamson, Anderson, Jackman, & Jackson, 1995). EDE assessors were blinded to experimental condition and were required to demonstrate a minimum agreement with expert raters (κ>.80) with audiotaped interviews prior to data collection.

Body mass—The BMI (Kg/M^2) was used as a proxy measure of adiposity. After removal of shoes and coats, height was measured to the nearest millimeter using stadiometers and weight was assessed to the nearest 0.1 kg using digital scales that were regularly calibrated for accuracy. The scale had a hand-held readout that was only visible to the assessor. The BMI correlates with direct measures of total body fat such as dual energy X-ray absorptiometry (*r* = .80–.90) and with health measures including blood pressure, adverse lipoprotein profiles, atherosclerotic lesions, serum insulin levels, and diabetes mellitus in adolescent samples (Dietz & Robinson, 1998; Pietrobelli et al., 1998).

Psychosocial functioning—Items adapted from the Social Adjustment Scale (Weissman & Bothwell, 1976) assessed psychosocial functioning in the family, peer group, school, and work spheres. The SAS has shown convergent validity with clinician and collateral ratings (*M r* = .72), discriminates between controls and psychiatric patients, and is sensitive to treatment effects (Weissman & Bothwell, 1976). This adapted scale has shown internal consistency (α = .77) and 1-week test–retest reliability (*r* = .83; Stice, Fisher, & Martinez, 2004).

Health care utilization—Health care utilization was assessed with four items adapted from the Health Survey Utilization Scale (HSUS; Ryan, Millstein, Greene, & Irwin, 1996) which assess frequency of utilization of health and mental health services (sample item: *In the past six months, did you get health care for a medical problem or an illness when you were feeling sick?*). If participants endorse health care service utilization, they were asked to indicate the primary reason for treatment. With this sample, the scale demonstrated acceptable reliability (α = .77) and 20-week test–retest reliability (*r* = .62).

Results

Preliminary analyses

At baseline, 52% (n = 44) of participants were classified as sub-threshold (i.e., either binge eating or engaging in compensatory behaviors only once a week on average). Those participants who were considered sub-threshold experienced an average of 4.6 binge episodes per month, and were using compensatory behaviors an average of 16 times per month. The remaining 48% (n = 41) of the sample experienced at least two each of binge and compensatory episodes per week and were classified as full-threshold. The full-threshold participants binge ate on average 17.4 times per month, and engaged in compensatory behaviors on average 30.3 times per month.

Preliminary analyses indicated that participants assigned to the two conditions did not differ significantly on ethnicity, age, parental education, BMI, or binge and purge frequency at baseline, suggesting that randomization succeeded in creating initially equivalent groups. Analyses also confirmed that diagnostic status (sub- or full-threshold) did not moderate any of the primary outcome variables.

A total of 16 (19%) participants dropped from the study before providing follow-up data. Those who dropped did not differ from those who provided complete data on baseline variables (all p-values>.36–.90), suggesting that attrition did not systematically bias the results. However, because attrition was significantly greater in the intervention (n = 12; 28%) versus control condition (n = 4; 9.5%; χ^2 [1] = 4.70, p = .030), we used maximum likelihood estimation, based on expectation-maximization algorithm, to impute missing data points, as this approach produces more accurate and efficient parameter estimates than listwise deletion or alternative imputation approaches such as last-observation-carried-forward or mean imputation (Shafer & Graham, 2002). It is also the recommended approach for studies in which attrition is significantly related with a study variable, i.e., treatment condition (Shafer & Graham, 2002). It should be noted that the pattern of main effects for the primary outcome variables was similar when the more common approach of listwise deletion was used, which is also referred to as completer analyses; all significant effects remained significant and all non-significant effects remained non-significant. All continuous variables showing marked skew (2.0 or greater) were normalized so that they satisfied the assumptions of the parametric tests. Participants who provided complete data attended all sessions and completed all homework assignments.

Intervention effects for BMI

To confirm that the intervention created a negative energy balance, repeated measures ANOVA models tested whether intervention participants showed significantly greater BMI decreases than waitlist controls. There were significant differences in change in BMI across the four waves of data (F [3/249] = 2.98, p<.05, r = .19). Intervention participants showed greater reductions in BMI than controls, which showed slight increases in BMI, suggesting that our manipulation was successful. This r value translates into a d = .39, which falls almost exactly in between a small and medium effect size according to the Cohen (1988) criteria. More focused time-by-condition interactions indicated that there were not significant changes in BMI from pretest to mid-treatment (F [1/83] = 1.48, p = .227, r = .13), but that intervention participants showed significantly greater decreases in BMI than controls from pre- to posttest (F [1/83] = 5.80, p<.05, r = .26) and marginally greater decreases in BMI at 3-month follow-up relative to controls (F [1/83] = 3.06, p = .084, r = .19). Follow-up paired t-tests (Bonferroni corrected α = .016) of change in BMI within conditions were not significant though (Table 1). The means indicated that intervention participants tended to lose weight across assessments from pre- to posttest, then gained a partial amount of that weight back by 3-month follow-up. By comparison, waitlist participants tended to gain weight across all assessment points. The pattern

of means, combined with the significant pre to post ANOVA model, suggest that, while actively participating in the intervention, participants showed significantly greater weight loss than the weight gain observed in controls and were therefore on the proper trajectory for weight loss. However, after treatment termination, participants in both condition showed weight gain.

Intervention effects for bulimic symptoms

A repeated measure ANOVA model indicated that there were significantly greater decreases in binge eating frequency among intervention participants than controls across the four waves of data (F [3/249] = 10.21, p<.001, r = .33). Follow-up models indicated that there were significantly greater decreases in binge eating from pretest to mid-treatment (F [1/83] = 4.47, p<.05, r = .12), pretest to posttest (F [1/83] = 28.33, p<.001, r = .50), and pretest to 3-month follow-up in the intervention versus control condition (F [1/83] = 25.11, p<.001, r = .48). There were also significantly greater decreases in compensatory behavior frequency across all four waves of data (F [3/249] = 5.21, p<.01, r = .24). Follow-up models indicated significant reductions in compensatory behaviors from pretest to mid-treatment (F [1/83] = 6.86, p = .01, r = .28), pretest to posttest (F [1/83] = 10.60, p<.01, r = .34), and pretest to 3-month follow-up in the intervention versus control condition (F [1/83] = 8.44, p<.01, r = .30). Follow-up paired t-tests (Bonferroni corrected α = .016), verified that the reductions in binge eating and compensatory behaviors in the intervention condition were statistically significant from pretest to mid-treatment, pretest to posttest, and pretest to 3-month follow-up (Table 1 presents the untransformed means). Although waitlist participants also showed significant decreases in these symptoms across time (which is likely a product of regression to the mean), the time-by-condition interactions indicate that the reductions were significantly larger among intervention participants versus controls.

Reliable change of main effects

Reliable change score analyses were conducted to provide an indication of the clinical significance of the change in frequency of bulimic symptoms from pretest to posttest and follow-up. Participants were classified on the basis of pretest to posttest and pretest to follow-up change scores on binge eating and compensatory behavior frequency using Jacobson & Truax's (1991) reliable change index (RCI) into improved (RCI<−1.96) and not improved (RCI ⩾−1.95). From pretest to posttest, intervention participants showed significantly higher rates of reliable change than controls on binge-eating frequency (90% versus 49%, respectively, (χ^2 [1] = 12.85, p<.001) and compensatory behaviors (90% versus 62%, respectively. χ^2 [1] = 6.78, p<.01). From pretest to 3-month follow up, intervention participants demonstrated higher rates of reliable change than controls on binge eating frequency (94% versus 55%, respectively, χ^2 [1] = 12.54, p<.001) and on compensatory behavior frequency (81% versus 50%, respectively, χ^2 [1] = 6.94, p<.01).

Remission rates

Remission is typically operationalized as the absence of binge episodes and compensatory behaviors in the past month (e.g., Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000). By posttest, 16% of intervention participants were in remission, versus 2% of controls, which represented a significant difference (χ^2 [1] = 4.81, p<.05). By 3-month follow-up, 35% of intervention participants were in remission, versus 10% of the controls, which was significantly different (χ^2 [1] = 6.75, p<.01). Of those considered in remission at T3, all of the controls relapsed at T4, while 29% of the intervention participants relapsed.

Intervention effects for other clinically significant outcomes

We also hypothesized intervention effects for health care utilization and social functioning. These outcomes were assessed only at baseline and 3-month follow-up. Repeated measures

ANOVA models tested whether there were significantly greater decreases in these outcomes for intervention participants relative to controls. As hypothesized, there were significantly greater decreases in health care utilization from pretest to 3-month follow-up (F [1/83] = 5.92, p<.05, r = .26) in the intervention condition relative to the control condition. A follow-up paired t-test (Bonferroni corrected α = .016), verified that the reduction in health care utilization was statistically significant from pretest to 3-month follow-up in the intervention, but not control, condition (Table 1). There was a trend for significant improvements in social functioning from pretest to 3-month follow-up (F [1/83] = 3.63, p = .06, r = .21). Paired t-tests indicated a significant reduction in this outcome for intervention participants, but not controls (Table 1).

Discussion

Individuals randomized to the Healthy Weight Program lost more weight over the intervention period than comparison controls (who gained weight over time), indicating that the intervention successfully manipulated weight-loss dieting. This weight loss effect highlights the primary difference between this intervention and CBT treatment, in that participants in the latter trials typically show a small increase in BMI by posttest (e.g., 0.9 BMI increase in Fairburn et al., 1991, Fairburn, Jones et al., 1993 and Fairburn, Marcus et al., 1993). However, in this trial, pretest to posttest decreased in BMI was relatively small (eta^2 = .065 or r = .26) and this effect faded by 3-month follow-up. The pattern of weight loss and regain observed in this trial is typical of most weight control interventions, wherein significant weight loss is not observed during the first few weeks, and maximum effects occur at posttest and fade over follow-up (Bennett, 1986; Jeffery et al., 2000). In fact, typical short-term low-calorie weight loss diets show similar small decreases in weight during a similar duration to this study (e.g., 0.21 decrease in BMI over 6-months of active treatment; Goodrick et al., 1998). Furthermore, virtually all obesity treatment programs show an erosion of effects after termination of treatment (Jeffery et al., 2000; Kramer, Jeffery, Forster, & Snell, 1989). These findings collectively suggest that it is extremely difficult to promote lasting healthy lifestyle changes, even in normal weight individuals, and casts doubt on the ability of this intervention to produce lasting weight-loss effects.

Nonetheless, as hypothesized, intervention participants demonstrated significantly greater reductions in bulimic symptoms, including both binge and purge frequencies, than controls throughout the study. The fact that such large decreases in bulimic symptoms were observed, and that the effects persisted to 3-month follow-up, suggests that the Healthy Weight Program may show promise as a treatment for bulimia nervosa. The pretest to posttest effects for binge eating and compensatory behaviors were large and medium in magnitude (r = .50 and .34, respectively), whereas a CBT meta-analysis found that the average pre to post effect sizes for binge eating and compensatory behaviors were both large (r = .55 and .61, respectively; Ghaderi & Andersson, 1999). One key difference between the interventions, however, was that the Healthy Weight Program lasts only 6-sessions over 8-weeks in duration, compared to the standard 20-week, 20-session CBT protocol. The average per-session effect size for change in binge eating and compensatory behaviors is r = .03 for both outcomes for CBT, whereas the comparable per session effects sizes are r = .08 and .06 for these outcomes for the Healthy Weight intervention. The fact that the clinical yield per hour of intervention is higher for the Healthy Weight intervention suggests that an expansion of the intervention may produce larger overall effects. The 3-month follow-up remission rate of 35% for the Healthy Weight intervention also compares favorably to the 1-year remission rates observed for CBT of 29% (Agras et al., 2000). Similarly, 29% of intervention participants in remission at T3 relapsed at T4, which is also comparable to relapse rates observed in CBT trials (e.g., 34% in Agras et al., 2000). These considerations suggest that it might be worthwhile to continue to refine and evaluate this treatment in larger-scale efficacy trials.

It was also hypothesized that the Healthy Weight Program would produce decreases in health care utilization and increases in social functioning. Significant intervention effects were observed for frequency of health care use, suggesting that an added benefit of this brief intervention is that it may reduce health care utilization and the associated costs. Given the high cost of health care, it is possible that the savings in health care costs may offset the expense of delivering this brief treatment. One possible explanation for the fact that the social function effects were only marginally significant is that improvements in social functioning may require a longer amount of time to develop, perhaps in part due to the nature of complex social relationships. Indeed, it has been hypothesized that improvements in social functioning are a secondary effect of treatment, and one that is not observed in some clinical trials until closer to 1-year follow-up (Fairburn, Jones et al., 1993).

It was noteworthy that the attrition rate for intervention participants in this trial (28%) was similar to rates observed in previous clinical trials of CBT (27%; Agras et al., 2000). Despite the shorter treatment duration, nearly one in three intervention participants withdrew from the study. This robust pattern of attrition may suggest that one-third of patients presenting for eating disorder treatment are actually ambivalent about treatment, regardless of form or content. In general, though many bulimic individuals may be distressed by their binge and purge routines and choose to seek treatment, they may also derive pleasure to their ability to binge-eat high fat and sugar foods without marked weight gain. The positive reinforcement attained from binge eating, for some individuals, may then outweigh the perceived benefits of treatment, thus resulting in a subset of treatment participants who are not willing to relinquish their disordered behaviors.

This trial also provided a unique opportunity to examine the role of dieting in the maintenance of bulimic symptoms. Intervention participants successfully lost weight during the intervention and evidenced significant decreases in binge eating and compensatory behaviors compared to controls. Remarkably, this current study represents the seventh experimental study of the effects of confirmed weight loss or weight maintenance diets on bulimic symptoms. Five other randomized clinical trials have found that assignment to low-calorie weight loss diets results in significantly greater decreases in binge eating and bulimic symptoms for normal weight, overweight, and obese adolescent and adult women relative to waitlist controls (Goodrick et al., 1998; Groesz & Stice, 2005; Klem et al., 1997; Presnell & Stice, 2003; Reeves et al., 2001). Assignment to a more moderate weight-maintenance diet similarly decreased bulimic symptoms over the course of a year in normal weight adolescents compared to waitlist controls (Stice, Presnell et al., 2005). To the best of our knowledge, no experimental study that has manipulated dieting has provided support for the dietary restraint model of bulimic pathology. Furthermore, the current study is a unique addition, which illustrates that even a clinical population of individuals with bulimia nervosa demonstrates reductions in bulimic behaviors when encouraged to engage in a moderate weight-loss diet.

Collectively, these findings raise serious questions about the role of dieting in the maintenance of bulimic pathology. Though it is possible that dieting is a proxy risk factor, not a direct contributor to the development of bulimia nervosa, it appears unlikely that attempts to achieve a negative energy balance, per se, are maintaining bulimic symptoms. Furthermore, a recent study also suggests that participants in a weight maintenance diet show lower rates of binge eating and compensatory behavior onset than assessment-only controls (Stice, Shaw, Burton, & Wade, in press), which raises the possibility that the role of dieting in the onset of bulimic symptoms may be suspect as well.

The most likely reason for the discrepancy between longitudinal studies indicating that dieting predicts onset of bulimic symptoms and experimental treatment trials indicating that assignment to weight loss diets decease bulimic symptoms is that the self-report measures of

dieting used in the former studies are invalid. Research has found that dietary restraint scales do not show a significant or substantively meaningful inverse correlation with objective measures of acute or chronic caloric intake (Bathalon et al., 2000; Stice et al., 2004), as was suggested by the original validity studies that relied on self-reported caloric intake. In addition, individuals with elevated scores on dietary restraint scales tend to gain significantly more weight over both short and long periods of time relative to their lower-scoring counterparts (French, Jeffery, & Wing, 1994; Klesges et al., 1989; Klesges et al., 1992; Stice, 2001; Stice et al., 1999), providing further evidence that they are not entering a lasting state of negative energy balance.

If this assertion is true, it is possible that dieting has been falsely implicated as a risk and maintenance factor for bulimic pathology. Alternative treatments that do not target dieting (e.g., IPT and DBT) also show promise for treating this disorder, and results from the present trial demonstrate that promoting a negative energy balance can successfully reduce bulimic symptoms in a clinical population. These collective findings suggest that factors other than dietary restraint might be maintaining bulimia nervosa. Perhaps the effects from CBT may come primarily from its focus on weight and shape overvaluation, or perhaps both the CBT and Healthy Weight Program effects stem from the fact that both interventions promote healthy dieting and healthy eating strategies, albeit in different ways. It is also possible that participant expectancies, demand characteristics, or attention account for effects across all treatments, which suggests that future placebo-controlled trials are warranted to rule out this possibility. Future replication is also certainly indicated, but initial findings from this study appear incompatible with the notion that dieting is a principal factor in the maintenance of bulimia nervosa.

Limitations

First, the present study compared an active intervention to a waitlist control, but did not utilize a placebo control group. Although comparison to waitlist allowed for the ability to rule out regression to the mean, passage of time, and measurement-artifact alternative explanations for the effects of the intervention, a placebo comparison group has the added advantage to rule out the possibility that intervention effects resulted from expectancy effects, demand characteristics, or attention. In this first study we thought it best to utilize a standard control group to facilitate comparison of results from this study to those of past trials. Indeed, past psychosocial treatment trials for eating disorders have almost exclusively compared active interventions to waitlist control groups instead of placebo or alternative intervention control groups. Nevertheless, the development of an equally credible placebo control that controls for time, dosage, and attention effects would significantly benefit all clinical trials in this field. Second, the present study only used a 3-month follow-up. It would have been preferable to follow participants much longer, and track treatment effects in regular intervals throughout. Third, with the exception of direct measures of height and weight, we relied solely on self-report data in this trial. In the future, it would be preferable to, whenever possible, collect multiple-informant data and objective behavioral data. Fourth, the moderate sample size limited our power to detect intervention effects. Though we had adequate power (>.82) to detect medium effect sizes (r = .30; Cohen, 1988), effects that were smaller in magnitude may have been missed.

Conclusions

In sum, this study sought to examine the efficacy of a healthy weight-management program as a treatment for bulimia nervosa and to experimentally test the role of dieting in the maintenance of this disorder. This trial established some preliminary support for the efficacy of the Healthy Weight Program. Although attrition and remission rates were no better in this trial than CBT

trials, the fact that the intervention effects per hour of intervention were larger for the present intervention relative to the current treatment of choice suggests it is worthy of future development and evaluation. Furthermore, the fact that this intervention promoted modest weight loss may suggest it could be used to target this outcome among overweight individuals with bulimia nervosa. The evidence that the Healthy Weight Program resulted in weight loss while simultaneously reducing bulimic symptoms seems incompatible with the assertion that dieting maintains bulimic symptoms. As this is the seventh experimental study to generate data that are inconsistent with this theory, it seems that researchers should consider reevaluating the notion that dieting plays a role in the etiology or maintenance of bulimic symptoms. It will also be important to experimentally examine other potential risk and maintenance factors for this pernicious eating disorder.

Uncited references

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Table 1

Means and standard deviations for the healthy weight intervention (HW) condition and waitlist control condition on primary outcome and other clinically significant outcomes and results from the paired comparisons

Dependent variable	Baseline (Week 1) *M* (SD)	Mid-treatment (Week 4) *M* (SD)	Termination (Week 8) *M* (SD)	3-month follow-up (Week 20) *M* (SD)
Body mass index				
HW (*n* = 43)	24.19 (3.60)	24.07 (3.41)	23.96 (3.49)	23.96 (3.42)
WL (*n* = 42)	23.50 (2.84)	23.54 (2.82)	23.67 (2.90)	23.64 (2.81)
Binge-eating frequency				
HW (*n* = 43)	11.00 (10.70)$_a$	9.81 (27.44)$_b$	2.27 (3.58)$_b$	1.19 (2.44)$_b$
WL (*n* = 42)	10.52 (9.89)$_a$	8.22 (8.35)	6.65 (5.39)$_b$	7.82 (10.64)$_b$
Compensatory behavior frequency				
HW (*n* = 43)	25.33 (22.07)$_a$	16.25 (38.15)$_b$	7.50 (13.31)$_b$	6.94 (13.14)$_b$
WL (*n* = 42)	20.45 (21.58)$_a$	13.70 (13.77)$_b$	11.43 (12.76)$_b$	12.05 (13.21)$_b$
Social functioning *				
HW (*n* = 43)	34.95 (9.18)$_a$			30.40 (6.07)$_b$
WL (*n* = 42)	34.15 (7.71)			32.69 (8.32)
Health care utilization				
HW (*n* = 43)	0.93 (0.96)$_a$			0.49 (0.76)$_b$
WL (*n* = 42)	0.71 (0.74)			0.61 (0.75)

Notes: HW = Healthy Weight intervention condition. WL = waitlist condition. Means in the same row with different subscripts were statistically significantly different (p<.016).

*Higher scores on this scale score represent greater impairments in social functioning.