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Effects of a Weight Maintenance Diet on Bulimic Symptoms: An Experimental Test of the Dietary Restraint Theory

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Abstract

It is widely accepted that dieting increases the risk for bulimic nervosa, but there have been few experimental tests of this theory. We conducted a randomized experiment with adolescent girls (N=188) to examine the effects of a weight maintenance diet on bulimic symptoms. A manipulation check verified that the diet intervention resulted in weight maintenance and significantly reduced the risk for obesity onset and weight gain observed in assessment-only controls. As hypothesized, the diet intervention resulted in significantly greater decreases in bulimic symptoms and negative affect than observed in controls. These experimental findings, which converge with those from a weight loss diet experiment, appear antithetical to dietary restraint theory and suggest instead that dietary restriction curbs bulimic symptoms.

Bulimic pathology, which includes both threshold and subthreshold bulimia nervosa, is one of the more common psychiatric disturbances to afflict adolescent girls and young women, is associated with functional impairment, and increases the risk for future onset of obesity, depressive disorders, suicide attempts, anxiety disorders, substance abuse, and health problems (Johnson, Cohen, Kasen, & Brook, 2002; Lewinsohn, Streigel-Moore, & Seeley, 2000; Stice, Cameron, Killen, Hayward, & Taylor, 1999).

Numerous theorists have posited that dieting increases the risk for onset of bulimic pathology (Fairburn, 1997; Hawkins & Clement, 1984; Heatherton & Polivy, 1992; Huon, 1996; Polivy & Herman, 1985). 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; Wilson, 2002). Dieting must result in a negative energy balance for weight loss or a balance between caloric intake and expenditure for weight maintenance (Rosenbaum, Leibel, & Hirsch, 1997).¹ The dietary restraint model asserts that a reliance on cognitive control over eating, rather than physiological cues, leaves dieters vulnerable to uncontrolled eating when these cognitive processes are

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¹There are two benefits to defining dieting in terms of energy balance. First, changes in body mass over time can be used as a valid measure of dieting. Weight loss diets require a negative energy balance, which is manifested in a decrease in body mass over the observation period. Weight maintenance diets require a balance between caloric intake and expenditure, which is manifested in a stable body mass over time. The ability to operationalize dieting with objective measures is desirable because self-reports of caloric intake and expenditure have been found to be invalid and the degree of under- and over-reporting (respectively) is positively correlated with the degree of overweight (Bandini, Schoeller, Dyr, & Dietz, 1990; Lichtman et al., 1992) and with higher scores on dietary restraint scales (Bingham et al., 1995). A second benefit to relying on change in body mass to reflect weight loss dieting or weight maintenance dieting is that it automatically adjusts for caloric expenditure – which is important because a given caloric intake level can result in weight loss, weight maintenance, or weight gain, depending on caloric expenditure level. This dependence renders it inappropriate to define dieting solely in terms of caloric intake, much in the same way that it is inappropriate to ignore body mass when examining caloric intake.

disrupted (Polivy & Herman, 1985). Violation of strict dietary rules might also result in the temporary abandonment of dietary restraint because of the abstinence violation effect (Marlatt & Gordon, 1985). Additionally, dieting may result in depletion of tryptophan, a precursor of serotonin, which increases the likelihood of binge eating high-carbohydrate food to restore tryptophan levels (Kaye, Gendall, & Strober, 1998). These binge episodes putatively trigger redoubled dietary efforts and use of radical weight control techniques, such as vomiting and laxative use, which cascade into the self-maintaining binge-purge cycle (Fairburn, 1997).

Consistent with these theoretical assertions, self-reported dieting predicted subsequent onset of binge eating (Stice, Presnell, & Spangler, 2002; Stice, Killen, Hayward, & Taylor, 1998) and subthreshold/threshold bulimia nervosa (Killen et al., 1994, 1996). Because self-reported dieting has emerged as a potent and consistent risk factor for bulimic pathology, numerous researchers and clinicians believe that dieting causes bulimia nervosa (Fairburn, 1997; Hawkins & Clement, 1984; Heatherton & Polivy, 1992; Huon, 1996). Indeed, some investigators have called for a moratorium on dieting because of the belief that it causes eating pathology and have evaluated interventions that decrease dietary restriction (Polivy & Herman, 1992).

Although prospective studies provide support for the dietary restraint model, some experimental findings appear incompatible with this account. Randomized trials have found that assignment to a low-calorie weight loss diet (e.g., 1200 calories per day), versus a waitlist control condition, results in significantly greater decreases in binge eating for obese and overweight individuals (Goodrick, Poston, Kimball, Reeves & Foreyt, 1998; Klem, Wing, Simkin-Silverman, & Kuller, 1997; Reeves et al., 2001). The experimental evidence that weight loss diets result in decreased binge eating seems inconsistent with the claim that dieting promotes bulimic pathology. These findings are particularly worrisome because randomized experiments are more effective than prospective studies in ruling out the possibility that some third-variable explains the dieting-bulimic symptom relation. In addition, participants in the weight loss diet conditions of these trials lost significant weight, thereby confirming that dietary restriction was successfully manipulated. It is vital to clarify the nature of the relation of dieting to bulimic symptoms because of the etiologic and prevention implications. If dieting increases the risk for onset of bulimic symptoms, it would suggest that prevention programs should seek to eradicate dieting. However, if dieting decreases bulimic symptoms, it would imply that prevention programs should help people diet more effectively.

The contradictory findings led us to conduct an experiment wherein we randomly assigned normal weight women to a 6-week low-calorie weight loss diet or a waitlist control condition (Presnell & Stice, 2003). We wanted to test whether the antithetical findings would emerge with normal weight women and whether a low calorie diet would result in decreases in all of the DSM-IV bulimia nervosa symptoms (versus just binge eating). A manipulation check confirmed that women in the diet condition lost significant weight relative to controls. The weight loss diet resulted in significantly greater decreases in bulimic symptoms than were observed in controls. This appeared to be the first randomized experiment to investigate the relation of dieting to bulimic symptoms.

Although the Presnell and Stice (2003) findings also appear to be incompatible with restraint theory, it is possible that real world dieting is not as effective as the low-calorie diets used in controlled weight loss trials. It may be that most self-initiated dieters do not achieve a lasting negative energy balance. In an effort to examine this alternative explanation for the contradictory findings, the primary aim of this study was to experimentally investigate the effects of a weight maintenance diet, as opposed to a weight loss diet, on bulimic symptoms. A second rationale for studying the effects of a weight maintenance diet is that bulimic pathology may emerge during a period of weight maintenance dieting. Retrospective data

suggest that bulimic pathology often emerges when the patient is significantly below their highest past weight (Garner & Fairburn, 1988), which implies that many of these individuals might have been dieting to maintain their weight when the eating disturbance emerged. A third rationale for examining the effects of a weight maintenance diet is that it is important to explore whether different types of diets show similar relations to change in bulimic symptoms because dieting takes a variety of forms (Levy & Heaton, 1993). This would help establish the circumstances under which dieting shows an inverse relation to bulimic symptoms. Thus, we randomly assigned participants to either a weight maintenance diet or an assessment-only control condition and measured changes in bulimic symptoms over a 1-year period. The experimental evidence that weight loss diets result in decreased bulimic symptoms led us to hypothesize that a weight maintenance diet would result in significantly greater decreases in bulimic symptoms relative to an assessment-only control condition.

We used a randomized prevention trial for this experimental test of the dietary restraint theory because such trials provide powerful tests of theories, given their relative immunity to the possibility that some unmeasured variable explains observed effects (Hinshaw, 2002). A randomized trial is also uniquely suited to our objective because this is one of the few experimental paradigms that could produce long-term dietary restriction.

Another benefit of our randomized trial design is that it provided an opportunity to evaluate a new obesity prevention program. Obesity has reached epidemic proportions and is associated with serious medical problems, including diabetes mellitus, coronary heart disease, and death from all causes (Mokad et al., 2003; Pietrobelli et al., 1998). Unfortunately, most obesity prevention programs have not reduced risk for future obesity onset (Dietz & Gortmaker, 2001).

A secondary aim of this study was to experimentally test the hypothesis that dieting results in increased negative affect (Heatherton & Polivy, 1992). Theoretically, the failures that are often associated with dietary efforts increase the risk for mood disturbances. Dieting-induced deficits in serotonin may also contribute to negative affect (Kaye et al., 1998). In support of these assertions, self-reported dieting predicted future increases in depressive symptoms (Stice & Bearman, 2001) and onset of major depression (Stice, Hayward, Cameron, Killen, & Taylor, 2000). However, randomized obesity treatment trials suggest that low-calorie diets result in greater decreases in depressive symptoms than observed in controls (Foster, Wadden, Kendall, Stunkard, & Vogt, 1996; Rippe et al., 1998). Based on the experimental findings regarding the effects of weight loss diets, we hypothesized that a weight-maintenance diet would likewise result in significantly greater decreases in negative affect than observed in controls.

A tertiary aim was to examine the validity of a widely used dietary restraint scale. One explanation for the contradictory findings is that dietary restraint scales may not be valid measures of dietary restriction. Multiple studies (Jansen, 1996; Stice, Fisher, & Lowe, 2004; van Strien, Cleven, & Schippers, 2000), varying in food types consumed, settings examined, and populations studied, found that dietary restraint scales were not inversely correlated with objective measures of acute caloric intake, in contrast to the original validity studies that found these scales correlated negatively with self-reported intake (Laessle, Tuschle, Kotthaus, & Pirke, 1989; van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986). Similarly, Bathalon and associates (2000) found that one restraint scale did not correlate with an objective biological measure of longer-term caloric intake (over an 18-day period). The evidence that individuals with elevated scores on dietary restraint scales show the steepest rate of weight gain (French et al., 1994; Klesges, Isbell, & Klesges, 1992; Stice et al., 1999) also suggests that these measures may not assess long-term caloric restriction.² However, there is evidence that individuals placed on low-calorie weight loss diets show increases on dietary restraint scales (e.g., Lowe, Foster, Kerzhnerman, Swaim, & Wadden, 2001). Whether these scales are

valid indicators of dietary restriction has a vital implication for how findings from studies using these measures are interpreted, so we decided to conduct further research on the validity of these scales. We tested whether a common dietary restraint scale was sufficiently sensitive to detect changes in intake produced through a weight maintenance diet. Given the validity findings reported above, we hypothesized that the dietary restraint scale would not show concordance with the dietary restriction manipulation.

Method

Participants

Participants were 188 adolescent girls (M age = 16.7, SD = 1.5). We focused solely on girls because bulimic pathology is very rare in boys (Lewinsohn et al., 2000). The sample was composed of 6% Asians/Pacific Islanders, 8% African Americans, 23% Hispanics, 1% Native American, 57% Caucasians, and 5% who specified other or mixed racial heritage. Parental education (a proxy for socioeconomic status) ranged from less than high school graduate (2% of mothers and 7% of fathers) to advanced degree (22% of mothers and 26% of fathers).

Procedures

Participants were recruited from high schools and a university using direct mailings, flyers, and leaflets that invited females between the ages of 14 and 19 with body image concerns to participate in a study evaluating an intervention designed to improve body acceptance. We relied entirely on self-selection for this targeted prevention trial and did not require a specific level of body image disturbances for inclusion. Because no mention was made of the expectation that the intervention might produce reductions in negative affect and bulimic symptoms, participants were blinded to the study hypotheses. Participants were randomly assigned to either the healthy weight management intervention or an assessment-only control condition. The healthy weight intervention consisted of three weekly 1-hour sessions. Intervention groups were composed of 6–10 participants. Sessions were facilitated by a Ph.D. level clinical psychologist or doctoral student in clinical psychology and co-facilitated by a B.A. level research assistant.

Participants in the intervention group completed an interview and a survey immediately before the first session, immediately following the final session, and at 6-month and 12-month follow-ups. Participants in the assessment-only control group completed parallel assessments. Assessors were blind to the experimental condition of participants. Participants were compensated \$100 for completing the assessments.

Healthy Weight Control Intervention

Session 1.—Participants were first presented with the rationale that body image concerns often arise because people do not have a full grasp of healthy weight control skills. They were told that this intervention would help them make small permanent lifestyle changes that would allow them to bring their energy intake into equilibrium with their energy needs and thus achieve and maintain a healthier body weight and enhanced body satisfaction. They were not asked to count calories or reduce their caloric intake to a certain level. Next, the thin-ideal was defined and contrasted to the healthy-ideal, and it was made clear that this intervention focused on achieving the latter. Motivational interviewing was used to allow the group to explore advantages of the healthy ideal, such as less illness and social acceptance. They were instructed

²It has been suggested that certain dietary restraint scales reflect successful dieting and others reflect unsuccessful dieting (Heatherton, Herman, Polivy, King, & McGree, 1988). However, because both types of dietary restraint scales did not show significant inverse correlations with unobtrusive measures of acute caloric intake and identify participants with elevated weight gain trajectories, it appears that this conceptual distinction is not valid.

in how to develop a balanced diet and were encouraged to begin these dietary changes as part of a gradual healthy lifestyle adaptation. Strategic self-presentation (verbal public commitments to making changes in intake and activity) was used to reinforce lifestyle changes and to foster a feeling of control over one's body. To ensure an individualized plan, participants applied these behavior modification tips to their own eating and exercise habits (e.g., list intake of fattening foods and decide how they will reduce intake of such foods). The few participants who were thinner than the healthy ideal were encouraged to increase their intake and reduce their exercise so that they could achieve the healthy ideal and the few participants who were overweight were encouraged to reduce their intake and increase their exercise so that they could achieve this ideal. Finally, participants were asked to complete a three-day food diary and an exercise diary as homework to help them identify ways to fine-tune their caloric intake and expenditure.

Session 2.—For motivational enhancement purposes, participants were asked to brainstorm benefits of maintaining a healthy ideal. It was reiterated that food deprivation is not the goal and they should not go long periods without eating. Participants then reviewed their eating and exercise diary and group members provided support for each other if problems were encountered regarding their behavior modification goals. Participants then discussed the importance of exercise and listed the benefits of regular activity to provide further motivational enhancement. The homework assignment was to list ten personally meaningful reasons for pursuing the healthy ideal in addition to completing the eating and exercise diaries.

Session 3.—After reviewing the past session, participants discussed problems they encountered when trying to improve dietary intake and group members suggested ways to overcome these barriers. Then participants shared changes in their exercise level during the previous week and additional activity was encouraged on an individual basis. The importance of balancing input and output was again emphasized. Participants then shared their reasons for signing up for the group and what they hoped to accomplish. Finally, participants were asked to e-mail the facilitators their progress in making lifestyle alterations after one week.

Measures

Obesity.—The body mass index ($BMI = \text{kg/m}^2$) was used to reflect adiposity (Pietrobelli et al., 1998). After removal of shoes and coats, height was measured to the nearest millimeter using portable stadiometers and weight was assessed to the nearest 0.1 kg using digital scales. Two measures of height and weight were obtained and averaged. BMI shows convergent validity with direct measures of body fat such as dual energy x-ray absorptiometry ($r = .80 - .90$) and health measures such as blood pressure, adverse lipoprotein profiles, and diabetes mellitus (Dietz & Robinson, 1998; Pietrobelli et al., 1998). BMI was temporally reliable in this study (1-month test-retest reliability for the assessment-only controls = .99). As recommended by Barlow and Dietz (1998), adolescent obesity was defined using the 95th centiles of body mass index for age and sex based on nationally representative survey data.

Healthy eating.—As a further manipulation check, we generated four items assessing healthy eating behaviors (sample items: *I have eaten more fruits and vegetables* and *I have reduced the amount of fat in my diet*). These four items were averaged to form an overall healthy eating composite at each assessment point. This scale was internally consistent ($\alpha = .82$) at pretest and temporally reliable in this study (1-month test-retest reliability for controls = .63).

Exercise intensity.—As another manipulation check, we generated two items assessing weekly frequency of exercise (*How many days did you exercise during the past week*) and duration of exercise (*On average, how long did you exercise during the past week*). These two

items were multiplied to form a quantity x frequency measure of exercise behavior at each assessment. This scale was temporally reliable in this study (1-month test-retest reliability for controls = .61).

Bulimic symptoms.—The seven diagnostic items from the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a structured interview, were used to assess DSM-IV symptoms of bulimia nervosa. The diagnostic items were averaged to form a past month bulimic symptom composite and subscales assessing binge frequency, compensatory behavior frequency (vomiting, diuretic use, laxative use, fasting), and overvaluation of weight and shape were created. Because the symptom composite, binge frequency measure, and purge frequency measure were skewed, a normalizing $\log_{10}$ transformation was applied. The EDE has been found to have good internal consistency ($\alpha = .76 - .90$) and inter-rater reliability ($k = .70 - .99$), and to discriminate between eating disordered individuals and controls (Fairburn & Cooper, 1993; Williamson, Anderson, Jackman, & Jackson, 1995). The bulimic symptom composite was internally consistent ($\alpha = .96$ at pretest) and temporally reliable in this study (1-month test-retest reliability for controls = .95).

Negative affect.—Negative affect was assessed with the sadness, guilt, and fear/anxiety subscales from the Positive Affect and Negative Affect Scale-Revised (PANAS-X; Watson & Clark, 1992). Participants reported the extent to which they had felt various negative emotional states (e.g., sad) over the past month on 5-point scales ranging from *very slightly or not at all* to *extremely*. Items were averaged. This scale has adequate internal consistency ($\alpha = .95$) and convergent validity (Watson & Clark, 1992). This scale was internally consistent ($\alpha = .95$ at pretest) and temporally reliable in this study (1-month test-retest reliability for controls = .72).

Self-reported dieting.—The Dutch Restrained Eating Scale (DRES; van Strien et al., 1986) was included to assess whether this dieting scale was sensitive enough to detect the effects of our dietary manipulation. We used the DRES rather than the Restraint Scale (Polivy, Herman, & Warsh, 1978) because the latter suffers from criterion confounding (contains items assessing binge eating) and does not even correlate with self-reported caloric intake (Laessle et al., 1989). We used the DRES rather than the Three Factor Eating Questionnaire Restraint subscale (Stunkard & Messick, 1985) because the latter has not been shown to predict future onset of bulimic pathology. On the DRES, participants indicate the frequency of dieting behaviors using a 5-point scale ranging from *never* to *always*. Items were averaged. The DRES has adequate internal consistency ($\alpha = .95$) and test-retest reliability ($r = .82$; Stice et al., 2002; van Strien et al., 1986). This scale was internally consistent ($\alpha = .93$ at pretest) and temporally reliable in this study (1-month test-retest reliability for controls = .79).

Results

Preliminary Analyses

One-way analysis of variance (ANOVA) and chi-square tests indicated that the participants in the healthy weight and control conditions did not differ significantly on age, ethnicity, parental education, body mass, healthy eating, exercise intensity, bulimic symptoms, negative affect, or dieting behaviors, suggesting that random assignment generated groups that were equivalent at baseline. Nonetheless, analyses testing for intervention effects controlled for baseline levels of the outcomes to ensure that even non-significant initial differences between groups could account for intervention effects.

We verified that our recruitment approach attracted high-risk participants with elevated body dissatisfaction. The mean body dissatisfaction (3.5) score on the Satisfaction and Dissatisfaction with Body Parts Scale (Berscheid, Walster, & Bohmstedt, 1973) for participants was significantly higher than the mean score on this variable from a community-recruited sample of adolescent girls (3.0; Stice et al., 2002).³

Of the initial 188 participants, 4% dropped out of the study before providing complete data. Participants who dropped from the study did not differ significantly from those who remained in the study on demographic factors or any of the study variables and attrition was not significantly different across conditions. Nonetheless, we used an intent-to-treat analytic approach, wherein the last available score was carried forward for participants who dropped, to decrease the likelihood that even non-significant differential attrition biased the findings.

Manipulation Check

To provide a manipulation check of our weight maintenance diet, we tested whether participants in the healthy weight intervention were at lower risk for onset of obesity over the 1-year follow-up than controls. Twenty-six participants (13.8%) met the criteria for obesity at pretest and were excluded from these analyses to ensure a prospective test. A logistic regression model that controlled for initial body mass indicated that the risk for onset of obesity at any point during the 1-year follow-up was significantly lower (risk ratio = 0.12, $p = .032$) in the healthy weight condition (1.2%) than in the control condition (11.4%) among the initially non-obese individuals. Figure 1 provides a graphic depiction of the cumulative risk for obesity onset over the 1-year follow-up. A repeated measure ANOVA model also tested for differential change in BMI over the 1-year follow-up across the two conditions to ensure that participants in the healthy weight condition maintained their weight, rather than lost or gained weight (condition was a 2-level between-subjects factor and time was a 4-level within-subjects factor). The time-by-condition interaction indicated that there were no significant differences in change in BMI across the two conditions over the full follow-up period ($F [3/549] = 0.75$, n.s., 0.6% variance explained). Paired t-tests (Bonferroni corrected alpha = .017) verified that there were no significant decreases or increases in BMI in the healthy weight condition (see Table 1 BMI scores, as well as weight and height data). It was noteworthy that these paired t-tests revealed that participants in the control condition showed a significant increase in BMI from pretest to posttest. A repeated measures ANOVA model that used just pretest and posttest data confirmed that the increases in BMI in the control group was significantly greater than the non-significant change in the healthy weight condition ($F [1/183] = 4.06$, $p = .045$, 2.9% variance explained). Results suggest that the healthy weight intervention successfully produced a weight maintenance diet that reduced the risk for obesity onset and increases in weight observed in controls over the same period.

Repeated measure ANOVA models also tested whether participants in the healthy weight condition showed increases in healthy eating and exercise intensity over the follow-up. There were significantly greater increases in healthy eating ($F [3/540] = 7.87$, $p < .001$, 4.2 variance explained) and exercise intensity ($F [3/534] = 8.02$, $p < .001$, 4.3% variance explained) in the healthy weight condition compared to the control condition. Follow-up paired t-tests (Bonferroni corrected alpha = .017) indicated that the increases in healthy eating in the healthy weight intervention condition were significant from pretest to posttest, pretest to 6-month follow-up, and pretest to 1-year follow-up (Table 1). Similarly, the increases in exercise intensity from pretest to posttest and pretest to 6-month follow-up were statistically significant

³It might be noted that the healthy weight intervention resulted in significantly greater reductions in body dissatisfaction than the control condition from pretest through 1-year follow-up ($F [3/558] = 2.70$, $p = .045$, 1.4% variance explained).

in the healthy weight group, but this effect was no longer significant by 1-year follow-up. In contrast, changes in healthy eating and exercise intensity over time were not significantly different in the control group (although the decreases in exercise from pretest to posttest and from pretest to 6-month follow-up would have been significant if an alpha of .05 had been used). Findings suggested that the weight maintenance intervention decreased the risk for onset of obesity and weight gain because of both reduced intake and increased exercise.

Intervention Effects on Bulimic Symptoms

A repeated measure ANOVA model tested whether participants in the healthy weight condition showed greater increases or decreases in bulimic symptoms over the 1-year follow-up period than participants in the control condition. As hypothesized, there were significantly greater decreases in bulimic symptoms in the healthy weight condition relative to the control condition, as indexed by a significant time-by-condition interaction ($F [3/528] = 4.53, p = .004, 3\%$ variance explained). Paired t-tests (Bonferroni corrected alpha = .017) indicated that the decrease in bulimic symptoms in the healthy weight intervention condition was significant from pretest to posttest, pretest to 6-month follow-up, and pretest to 1-year follow-up (Table 2). The changes in bulimic symptoms over the 1-year follow-up were not significant in the control group.⁴

To further probe the nature of these findings, we conducted separate repeated measures ANOVA models for the three symptom domains of bulimic nervosa: binge eating frequency, compensatory behavior frequency, and overvaluation of weight and shape. Time-by-condition interactions indicated that there were significantly greater decreases in binge frequency ($F [3/522] = 2.61, p = .05, 1.5\%$ variance explained), purge frequency ($F [3/528] = 2.94, p = .033, 1.6\%$ variance explained), and weight and shape overvaluation ($F [3/528] = 2.87, p = .036, 1.6\%$ variance explained) in the healthy weight condition relative to the control condition. Bonferroni adjusted paired t-tests (Table 2) confirmed that participants in the healthy weight condition showed significant decreases in these symptom domains throughout the follow-up period, whereas control participants did not. Results suggest that the weight maintenance diet produced decreases in each of the core symptoms of bulimia nervosa.

Intervention Effects on Negative Affect

A repeated measure ANOVA model tested whether participants in the healthy weight condition showed greater increases or decreases in negative affect over the 1-year follow-up period than controls. There were significantly greater decreases in negative affect in the healthy weight condition relative to the control condition ($F [3/555] = 3.42, p = .017, 2\%$ variance explained). Paired t-tests (Bonferroni corrected alpha = .017) indicated that the decrease in negative affect in both conditions was significant from pretest to posttest, pretest to 6-month follow-up, and pretest to 1-year follow-up (Table 2). Because the paired t-tests indicated that there were significant decreases over time in both conditions, more focused repeated measures ANOVA models were used to determine the period of time when the two groups showed significantly different change in this outcome. The decreases in negative affect were significantly greater in the healthy weight condition than in the control condition from pretest to posttest ($F [1/185]$

⁴It is possible that the significantly greater reductions in bulimic symptoms observed in the weight maintenance condition relative to the control condition resulted because more participants in the former condition lost weight (i.e., were actually on a weight loss diet) than in the latter condition. However, the proportion of individuals who showed a 5% decrease in BMI over the 1-year follow-up was not significantly different ($\chi^2 [df = 1, N = 188] = 0.98, p = .321$) for the healthy weight condition (12%) than for the control condition (7%). Moreover, the a repeated measures ANOVA model verified that there were still significantly greater decreases in bulimic symptoms over the 1-year follow-up in the weight maintenance condition than in the control condition ($F [3/504] = 3.62, p = .014, 4.6\%$ variance explained).

= 4.87, $p = .029$, 3% variance explained), but not from pretest to 6-month follow-up ($F [1/185] = 0.07$, n.s.) or from pretest to 1-year follow-up ($F [1/185] = 0.18$, n.s.).

Validity of Self-Reported Dieting

The tertiary aim was to test whether a commonly used dietary restraint scale was sufficiently sensitive to detect changes in intake produced through a weight maintenance diet. There were no significant differences in change in dietary restraint scores across time in the healthy weight and control conditions ($F [3/555] = 1.54$, $p = .206$, 2% variance explained). Both groups showed significant decreases on this dieting scale over time (see Table 2). Thus, the dieting scale did not detect the effect of the weight maintenance diet, despite the fact that control participants were at significantly higher risk for onset of obesity and weight gain than participants in the healthy weight intervention.

Discussion

The primary aim was to test the hypothesis that a weight maintenance diet would produce significant decreases in bulimic symptoms. A manipulation check, which relied on direct measures of body mass, confirmed that our intervention successfully produced a weight maintenance diet that reduced the risk for obesity onset and increases in weight observed in the control group. As hypothesized, results suggested that our weight maintenance diet resulted in significant reductions in bulimic symptoms relative to an assessment-only control condition over the 1-year follow-up. The intervention effects cannot be easily attributed to expectancies or demand characteristics because participants were not told that the intervention was expected produce decreases in bulimic symptoms. It is also unlikely that experimenter bias produced these effects because assessors responsible for conducting diagnostic interviews and collecting body mass data were kept blind to the condition of participants.

The evidence that a weight maintenance diet resulted in decreased bulimic symptoms converges with past experimental findings indicating that a weight loss diet resulted in decreased bulimic symptoms (Presnell & Stice, 2003). To our knowledge, this is the second experimental test of whether dietary restraint produces change in bulimic symptoms, although findings converge with those from three randomized trials that indicated that a weight loss diet resulted in decreases in binge eating (Goodrick et al., 1998; Klem et al., 1997; Reeves et al., 2001). The results from these five experiments consistently suggest that dieting curbs subdiagnostic levels of bulimic symptoms in these high-risk samples. These experimental findings appear to be at odds with the assertion that dietary restraint promotes bulimic pathology (Polivy & Herman, 1985). Thus, our results add to the mounting findings that are incompatible with the dietary restraint model (e.g., Lowe et al., 1996; Lowe, Gleaves, & Murphy-Eberenz, 1998).

There are at least three possible explanations for the contradictory findings between the prospective studies reporting that elevated dietary restraint scores predict future onset of bulimic symptoms and the experimental finding that assignment to a diet results in decreased bulimic symptoms. First, the incongruous findings may have occurred because the prospective studies used invalid measures of dietary restraint. Numerous studies have found that dietary restraint scales do not show the expected inverse correlations with unobtrusive measures of acute and long-term caloric intake (Bathalon et al., 2000; French et al., 1994; Klesges et al., 1992; Stice et al., 2004; van Strein et al., 2000). These results imply that these scales do not assess dietary restriction as suggested by validation studies that relied on self-reported caloric intake (Laessle et al., 1989; van Strien et al., 1986) and the content of the items. It will therefore be important for future studies to further assess the validity of dietary restraint scales with

objective and unobtrusive measures and to develop more valid techniques of measuring dietary restraint.

A second possible explanation for the contradictory findings between the prospective and experimental studies is suggested by the inferential limitations of certain designs. Because randomized experiments rule out third variable explanations and prospective studies do not, the positive relation of self-reported dieting to increases in bulimic pathology may have emerged because some third variable increases the risk for both variables. It has been suggested that a tendency towards caloric overconsumption may lead to both self-reported dieting and eventual onset of bulimic pathology (Stice et al., 1999). If this were the case, self-reported dieting would be a proxy risk factor for bulimic symptoms solely because it is a marker for chronic overconsumption. It will be important for future studies to directly test whether a propensity towards overconsumption, assessed via objective and unobtrusive procedures, is a risk factor for onset of dieting and binge eating.

A third possible explanation for the conflicting findings is that the experimental interventions promote healthy dietary behaviors, but that it is unhealthy dietary behaviors, such as meal skipping, that leads to onset of bulimic pathology. The evidence that 20% of dieters report skipping meals (Levy & Heaton, 1993) suggests that such unhealthy dieting behaviors are prevalent. One way to address this possibility would be to manipulate “dieting as usual” in a randomized experiment to provide an ecologically valid test of whether dieting, in what ever form it usually takes in the real world, results in increased or decreased bulimic symptoms. It would also be useful to experimentally manipulate meal skipping, by assigning participants to a diet involving daily meal skipping or a diet requiring regular meals. However, one experimental trial that took this approach did not find any experimental effects on binge eating frequency (Schlundt, Hill, Sbrocco, Pope-Cordle, & Sharp, 1992).

The secondary aim of this experiment was to test whether dietary restraint results in increased negative affect, as has been hypothesized previously (Heatherton & Polivy, 1992), or significant decreases in negative affect, as has been observed in controlled weight loss trials (e.g., Foster et al., 1996). Results suggested that assignment to a weight maintenance diet resulted in decreases in negative affect, though this effect was of limited duration. The fact that the weight maintenance diet was less intense than weight loss diets may explain the transient effect. Moreover, a reduction of clinically significant obesity is more likely to produce improvements in mood than a less drastic lifestyle change like that encouraged in the current intervention. Regardless, our results suggest that assigning participants to a weight maintenance diet did not produce any adverse effects on mood, as has been previously theorized.

The tertiary aim was to test whether a commonly used dietary restraint scale would be sufficiently sensitive to detect changes in dietary restraint produced by our weight maintenance diet. As expected, the DRES did not detect the effects of our weight maintenance diet, despite ample statistical power. These null findings are noteworthy because they occurred despite the fact that objective measures of body mass implied that our intervention produced a weight maintenance diet. Although it is tempting to interpret these null findings as additional evidence that dietary restraint scales lack validity, it is important to note that these scales are sensitive enough to detect the effects of low-calorie weight loss diets (Lowe et al., 2001). This pattern of findings seems to suggest that dietary restraint scales may only be sensitive to dramatic changes in dietary restraint, but not to more subtle changes involved in a weight maintenance diet.

Limitations of the Current Study

The limitations of this study should be considered when interpreting the findings. First, participants in the healthy weight group were not self-initiating a diet, and therefore may be unrepresentative of those who diet in the real world. They were also recruited to participate in a trial of a body acceptance intervention. Thus, the findings should be generalized with caution. Second, because our sample included only females, results should be generalized to males with caution. However, this may not be a significant limitation given the low rates of bulimic pathology among males (Lewinsohn et al., 2000). Third, because we focused solely on adolescents, our results should also be generalized with caution to younger or older populations. Again, however, we focused on adolescence because this is the peak risk period for onset of bulimic pathology (Stice et al., 1998). Fourth, we used body mass as our indicator of adiposity. The validity of the body mass index is supported by the fact that it correlates highly with direct measures of total body fat and with obesity-related health indicators (Dietz & Robinson, 1998; Pietrobelli et al., 1998). Nonetheless, it would have been preferable to had used direct measures of adipose tissue, such as dual energy x-ray absorptiometry.

Research and Clinical Implications

The current findings have several implications for future research. First, further studies on the impact of experimentally manipulated dietary restraint on bulimic symptoms should be conducted. The fact that most dietary restraint studies have not manipulated this variable appears to have produced faulty inferences regarding the effects of dieting on bulimic pathology. As noted above, these experiments should attempt to manipulate the dieting practices that are typically practiced in the real world, versus prescribed weight loss or weight maintenance diets, to enhance the ecological validity of the inferences drawn from these studies. It would also be useful if future studies investigated the effects of successful weight loss on bulimic symptoms. Large epidemiological studies that follow participants over time may lend themselves well to this application because it is assumed that at least some proportion of individuals succeed in losing weight. Such a study would be particularly useful because it would address the criticism that prescribed dietary interventions are not representative of dieting that occurs in the real world. Second, it would be useful if future randomized prevention trials evaluated whether prescribed weight loss and weight maintenance diets are effective in preventing and potentially even treating bulimic pathology. Third, additional studies should continue to examine the validity of dietary restraint scales using objective measures of caloric intake and weight change. Fourth, it will be important for future research to develop more valid measures of dietary restriction. Without such instruments, it will be difficult to make accurate inferences about the consequences of dieting. Fifth, it is also vital that we determine what construct is assessed by extant dietary restraint scales. That these scales identify individuals at high risk for onset of bulimic symptoms suggests that they reliably measure some construct that may play a critical role in the etiology of this eating disorder (e.g., an inability to successfully modulate dietary intake).

In terms of clinical implications, the current findings suggest that a weight maintenance diet is an effective prevention program for bulimic symptoms. The healthy weight intervention has emerged as one of only six programs to produce significant intervention effects on eating disorder symptoms that persist over follow-up, out of 38 programs that have been evaluated (Stice & Shaw, 2004). The magnitude of the effect for the healthy weight intervention on bulimic symptoms comparables favorably to those observed for the other programs that produced intervention effects for this outcome. This suggests that both weight loss diets and weight maintenance diets help individuals curtail bulimic symptoms. In addition, the healthy weight intervention possess the added benefit of decreasing future risk for onset of obesity – which is an outcome that prior eating disorder prevention programs have not achieved. Indeed,

prior obesity prevention programs have rarely achieved this outcome (Dietz & Gortmaker, 2001). This intervention might have been particularly effective for this outcome because we worked with a group of individuals who were highly motivated to alter lifestyle behaviors because they wanted to decrease their current body image concerns (versus the more distal outcome of decreasing their risk for future obesity related health problems). Given that obesity has reached epidemic proportions and is associated with marked morbidity and mortality, an intervention that addresses both bulimic symptoms and obesity holds much public health appeal.

Conclusions

These experimental findings, which converge with those from four other randomized trials, challenge the widely held belief that dieting increases the risk for bulimic symptoms. Paradoxically, they suggest instead that effective dietary restriction may represent a potent tool for curbing non-diagnostic levels of bulimic symptoms and risk for obesity onset. Future randomized experiments on the effects of dieting are needed to extend our understanding of the relation between dieting and bulimic pathology. More generally, these findings underscore the hazards of relying on prospective studies for making etiologic inferences and suggest that it is vital to attempt confirm the relations suggested by such studies with randomized experiments that manipulate the putative risk factor.

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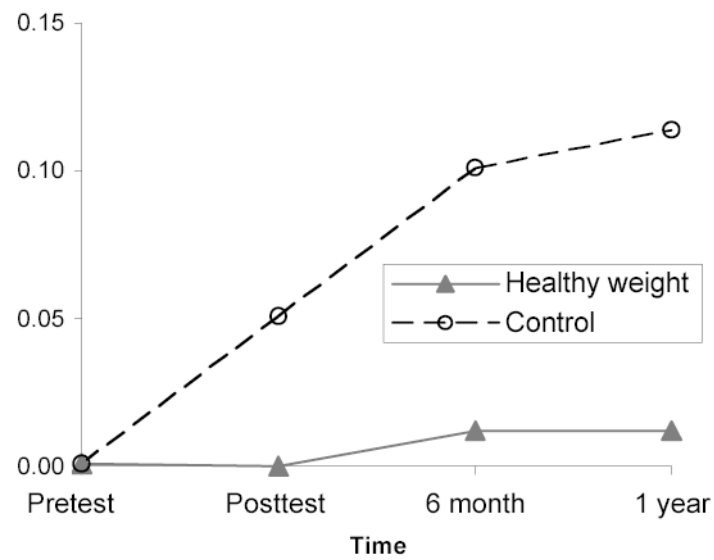


Figure 1. Cumulative risk for onset of obesity in the healthy weight and control conditions over the 1-year follow-up among the initially non-obese participants.

Table 1
Means and Standard Deviations for the Healthy Weight and Assessment-Only Control Condition on BMI, Weight, Height, Healthy Eating, and Exercise Intensity.

Dependent variable	Pretest M (SD)	Posttest M (SD)	6-month follow-up M (SD)	1-year follow-up M (SD)
<u>BMI</u>				
Healthy Weight	22.82 (4.16)	22.88 (4.17)	23.01 (4.30)	23.00 (4.40)
Control	23.79 (5.44) _a	23.99 (5.43) _b	24.01 (5.55)	23.89 (5.44)
<u>Weight (cm)</u>				
Healthy Weight	61.21 (13.04) _a	61.34 (13.07)	62.06 (13.13) _b	62.18 (13.31) _b
Control	63.16 (13.53) _a	63.48 (13.41)	63.89 (13.79) _b	63.50 (13.22)
<u>Height (kg)</u>				
Healthy Weight	163.5 (7.0) _a	163.5 (7.1)	164.0 (6.9) _b	164.3 (7.3) _b
Control	163.0 (7.0) _a	163.1 (7.1)	163.4 (7.1) _b	163.4 (7.3) _b
<u>Healthy eating</u>				
Healthy Weight	2.83 (0.86) _a	3.35 (0.81) _b	3.19 (0.87) _b	3.17 (0.87) _b
Control	2.78 (1.01)	2.73 (0.92)	2.80 (1.03)	2.74 (0.98)
<u>Exercise intensity</u>				
Healthy Weight	21.44 (18.76) _a	30.01 (17.44) _b	26.11 (19.92) _b	24.81 (19.85)
Control	20.72 (16.57)	17.60 (16.60)	17.05 (15.86)	17.55 (17.19)

Note: Means within the same row with different subscripts were significantly different ($p < .0167$). Cell sizes were $n = 94$ for the healthy weight group and $n = 94$ for the assessment-only control group.

Table 2
Means and Standard Deviations for the Healthy Weight and Assessment-Only Control Condition on Bulimic Symptoms, Negative Affect, and Self-Reported Dieting.

Dependent variable	Pretest M (SD)	Posttest M (SD)	6-month follow-up M (SD)	1-year follow-up M (SD)
<u>Bulimic symptoms</u>				
Healthy Weight	1.50 (1.28) _a	0.96 (0.84) _b	0.92 (0.98) _b	0.95 (1.09) _b
Control	1.49 (1.31)	1.36 (1.12)	1.45 (1.70)	1.31 (1.55)
<u>Binge frequency</u>				
Healthy Weight	0.79 (2.33) _a	0.33 (1.72) _b	0.39 (2.15) _b	0.20 (0.73) _b
Control	1.42 (4.48)	1.05 (3.72)	1.76 (7.73)	1.98 (7.42)
<u>Compensatory behavior frequency</u>				
Healthy Weight	0.64 (1.82) _a	0.20 (0.77) _b	0.23 (0.92) _b	0.31 (1.66) _b
Control	0.47 (1.37)	0.29 (0.95)	0.41 (1.29)	0.25 (0.70)
<u>Weight and shape overvaluation</u>				
Healthy Weight	3.18 (1.46) _a	2.87 (1.56)	2.57 (1.63) _b	2.56 (1.62) _b
Control	3.12 (1.52)	2.73 (1.76)	2.94 (1.75)	2.81 (1.72)
<u>Negative affect</u>				
Healthy Weight	2.11 (0.80) _a	1.59 (0.77) _b	1.85 (0.87) _b	1.84 (0.89) _b
Control	2.21 (0.93) _a	1.93 (0.88) _b	1.92 (0.84) _b	1.89 (0.78) _b
<u>Self-reported dieting</u>				
Healthy Weight	2.67 (0.98) _a	2.29 (0.94) _b	2.22 (0.94) _b	2.25 (0.91) _b
Control	2.71 (0.92) _a	2.43 (0.99) _b	2.47 (1.01) _b	2.42 (0.93) _b

Note: Means within the same row with different subscripts were significantly different ($p < .0167$). Cell sizes were $n = 94$ for the healthy weight group and $n = 94$ for the assessment-only control group. Untransformed means and standard deviation are reported for bulimic symptoms, but the transformed versions were used in the analyses.