An experimental test of the effects of dieting on bulimic symptoms: The impact of eating episode frequency

Lisa M. Groesz, Eric Stice*

Department of Psychology, University of Texas at Austin, A 8000, Austin, TX 78712, USA

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Abstract

Prospective studies suggest dieting increases bulimic symptoms, but experiments suggest that dieting decreases bulimic symptoms. One possible explanation for the conflicting findings is that real world dieting involves less healthy dieting techniques, such as meal skipping, than prescribed diets. We tested whether the manipulation of eating episode frequency during dieting impacted bulimic symptoms. We expected that people on a diet involving fewer eating episodes would exhibit greater increases in bulimic symptoms than people on a diet involving more frequent eating episodes or waitlist controls. Participants on both 6-week diets lost more weight than controls, confirming dieting was manipulated, and showed greater reductions in bulimic symptoms than controls; however, the dieting conditions did not differ on either outcome. Results provide further experimental evidence that dieting does not increase bulimic symptoms, but suggests that eating episode frequency has little impact on this outcome.

Keywords: Dieting; Bulimia; Meal frequency

Introduction

Nearly 65% of adults in the US are overweight or obese (Flegal, Carroll, Ogden, & Johnson, 2002), which is alarming because these conditions are credited with nearly 400,000 deaths annually (Mokdad, Marks, Stroup, & Gerberding, 2004). Although low calorie weight loss diets are currently considered the first-line treatment of choice for obesity (Jeffery et al., 2000), theorists have argued that dieting increases the risk for binge eating and bulimic symptoms and have suggested that dieting should be discouraged (Fairburn, 1997; Heatherton, Herman, & Polivy, 1992; Polivy & Herman, 1985). This is an important iatrogenic effect to avoid because bulimic pathology is associated with functional impairment and medical complications, and increases the risk for future depression, suicide attempts, anxiety disorders, substance abuse, obesity, and health problems (Johnson, Cohen, Kotler, Kasen, & Brook, 2002; Newman et al., 1996; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Striegel-Moore, Seeley, & Lewinsohn, 2003). Thus, it is crucial to determine whether dieting, defined as intentional and sustained restriction of caloric intake for the purposes of weight loss (Herman &
Mack, 1975; Laessle, Tuschl, Kotthaus, & Pirke, 1989; Wadden, Brownell, & Foster, 2002; Wilson, 2002) actually increases the risk for bulimic symptoms.

Theorists have proposed several mechanisms by which dieting might increase the risk for bulimic symptoms. 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 putatively increases the likelihood that they will binge eat. Polivy and Herman (1985) also argue that a reliance on cognitive controls over eating, rather than physiological cues, leaves dieters vulnerable to uncontrolled eating when these cognitive controls are disrupted. In addition, violation of strict dietary rules may result in the temporary abandonment of dietary restriction because of the abstinence violation effect (Marlatt & Gordon, 1985). It has also been suggested that dieting induced weight suppression increases susceptibility to food cues (Nisbett, 1972) and decreases perception of satiety cues (Lowe, 1993), which in turn may increase the risk for binge eating. Binge eating putatively precipitates redoubled dietary efforts and use of radical weight control techniques, such as vomiting and laxative use, which develop into the self-maintaining binge–purge cycle (Fairburn, 1997).

Consistent with the dieting model of bulimic symptoms, prospective studies have found that adolescent girls with elevated scores on various dietary restraint scores are at increased risk for future onset of binge eating (Stice, Killen, Hayward, & Taylor, 1998; Stice, Presnell, & Spangler, 2002) and onset of threshold or subthreshold bulimia nervosa (Killen et al., 1994, 1996). Based on these findings, it is widely accepted by researchers and clinicians that dieting causes bulimia nervosa (e.g., Huon, 1996).

In contrast, randomized experiments have produced findings that seem incompatible with the dieting theory of bulimia nervosa. Randomized obesity treatment trials have found that assignment to low-calorie weight loss diets resulted in significantly greater decreases in binge eating for obese (Goodrick, Poston, Kimball, Reeves, & Foreyt, 1998; Reeves et al., 2001) and for overweight adults (Klem, Wing, Simkin-Silverman, & Kuller, 1997) relative to waitlist controls. Experimental trials have also found that assignment to a weight loss diet (Presnell & Stice, 2003) or a weight maintenance diet (Stice, Presnell, Groesz, & Shaw, 2005) resulted in significantly greater reductions in bulimic symptoms for normal weight young women relative to waitlist control conditions. A randomized treatment trial found that assignment to a weight loss intervention resulted in significantly greater decreases in bulimic symptoms and produced significantly higher remission rates for young women with full or subthreshold bulimia nervosa relative to waitlist controls (Burton & Stice, in press). Participants in the dieting conditions of the latter four trials showed significantly greater weight loss than controls, which confirms that dietary restriction was successfully manipulated in those studies. The experimental findings are troublesome for the dieting theory of bulimic symptoms because randomized experiments are more effective than prospective studies in ruling out the possibility that some third-variable explains the dieting–bulimia symptom relation.

It is vital to clarify the nature of the relation between dieting and bulimic symptoms because of the vastly different intervention implications. If dieting increases risk for bulimic symptoms, prevention and treatment programs should seek to eradicate dieting and alternative treatments for obesity should be developed. However, if dieting decreases risk for bulimic symptoms, prevention and treatment interventions should seek to promote effective dieting, as this should reduce risk for both bulimic symptoms and obesity. It is thus crucial to search for explanations for the inconsistent findings from the prospective and experimental studies regarding the relation of dieting to bulimic symptoms.

One possible explanation for the inconsistent findings is that the prescribed diets examined in the experimental trials promote healthy weight loss behaviors, whereas real world dieting involves unhealthy weight control behaviors that increase risk for bulimic symptoms. To explore this possibility, it will be important to systematically manipulate suspected unhealthy weight control behaviors experimentally to provide the most rigorous test of whether these behaviors are related to bulimic symptoms. The National Task Force on the Prevention and Treatment of Obesity (2000) and other investigators (Neumark-Sztainer, Butler, & Palti, 1995) have categorized meal skipping as an unhealthy dietary technique because it is associated with poor nutritional intake (e.g., less calcium intake) and consumption of higher calorie foods at subsequent feedings (de Castro & Elmore, 1988; Morgan, Zabik, & Stampley, 1986). In addition, a randomized experiment found that increased meal frequency (nine meals a day) was associated with lower fasting total
serum cholesterol and low-density-lipoprotein levels relative to typical meal frequency (three meals a day; 
Arnold, Ball, Duncan, & Mann, 1993). Research suggests that 35% of female and male adult dieters skip 
meals for weight loss purposes (French, Jeffery, & Murray, 1999; Wardle, Griffith, Johnson, & Rapoport, 
2000) and that 50% of female adolescent dieters report meal skipping (Emmons, 1992). Because meal skipping 
is a prevalent weight loss technique that has been categorized as unhealthy, it seems important to 
experimentally investigate the effect of meal distribution on bulimic symptoms.

Skipping meals for the purpose of weight control may increase the risk for binge eating and bulimic 
symptoms through several mechanisms. First, meal skipping results in greater attention to high calorie foods 
(Placanica, Faunce, & Job, 2002) and greater perception of reinforcement from eating (Raynor & Epstein, 
2003), which could lead to elevated intake once food is accessible. Second, approaching a meal hungry predicts 
take of high calorie density foods (de Castro & Elmore, 1987; Morgan et al., 1986), which dovetails with the 
evidence that higher calorie foods are commonly consumed during binge eating (Wilson, Becker, & Heffernan, 
2003). Third, people may justify consuming larger portions because they skipped a meal. In support, acute 
periods of caloric deprivation (e.g., 4–12 h) have generally resulted in greater caloric intake at subsequent 
meals (Agras & Telch, 1998; Telch & Agras, 1996). If the mechanism is the greater temporal distance between 
meals, incorporating more eating episodes such as four to five small meals may decrease the likelihood of 
binge eating and intake of high calorie foods.

To our knowledge, only two studies have examined the effect of experimentally manipulated meal skipping 
on binge eating. The first randomly assigned obese women to a 12-week low-calorie diet that either prescribed 
consumption of three meals a day or to a condition that prescribed consumption of two meals a day (Schlundt, 
Hill, Sbrocco, Pope-Cordle, & Sharp, 1992): the experimental manipulation had no effects on change in binge 
eating or body mass over the 12-week study, although it is possible that the non-significant effects emerged 
because the small sample size \(N = 52\) limited statistical power. The second study examined binge eating 
in the lab among women with binge eating disorder randomly assigned to a condition in which they skipped 
breakfast and lunch (14-h of food deprivation) or a condition in which they consumed breakfast and 
lunch (Agras & Telch, 1998); there were no significant effects of the manipulation on self-reported binge 
eating, but there was significantly higher investigator-coded binge eating in the meal skipping condition 
relative to the non-meal skipping condition. Interestingly, there were no significant differences in overall 
caloric intake over the daylong laboratory session across conditions, implying that participants simply made 
up for missed calories. These studies suggest that chronic meal skipping does may not result in increased 
binge eating but that there was mixed evidence that extended acute caloric deprivation results in greater acute 
binge eating.

Because we were unable to locate a randomized experiment that investigated the effects of meal distribution 
on all of the DSM-IV symptoms of bulimia nervosa, the first aim of the present study was to conduct such a 
trial. To address this aim, we randomly assigned women to a low-calorie diet that prescribed four to five eating 
episodes or to a low-calorie diet that did not prescribe meal frequency, under the assumption that people who 
diet would skip meals (Emmons, 1992; French et al., 1999). This study utilized a manipulation that increased 
meal frequency due to ethical considerations in relation to meal skipping and resistance towards skipping 
meals from participants in a pilot study. The low-calorie diet that promoted more eating episodes distributed 
throughout the day, the Many Small Meals (MSM) condition, encouraged participants to eat numerous small 
meals a day that sum to 1200 kcal intake, including breakfast, a morning snack, lunch, an afternoon snack, 
and dinner, with the rationale that frequent eating decreased the likelihood of overeating high fat and high 
sugar foods. The Normal Diet (ND) condition encouraged participants to restrict their caloric intake to 
approximately 1200 kcals a day, without advice concerning the frequency of eating. Participants in both 
conditions received nutritional education, learned about behavioral modification techniques to alter the food 
environment, and were encouraged to exercise in an effort to keep all other aspects of the low-calorie diets 
constant. Females were recruited because they are much more likely to diet and to experience bulimic 
symptoms than males (Neumark-Sztainer et al., 2000; Wilson et al., 2003). We expected that MSM 
participants would show significantly greater decreases in bulimic symptoms than ND participants because the 
shorter time between eating episodes in the former condition should theoretically cause participants to 
approach meals with less hunger and less preoccupation with food, which should decrease the likelihood of 
binge eating and bulimic symptoms. An added benefit of this design is that it provided an opportunity to test
the hypothesis that fewer eating episodes would be associated with less effective weight loss than a diet characterized by consumption of many small meals.

The second aim of this study was to attempt an independent replication of a prior trial that found that a low-calorie diet resulted in greater decreases in bulimic symptoms relative to a waitlist condition (Presnell & Stice, 2004). This aim was accomplished by including a waitlist (WL) condition in the present trial. Based on the previous findings, we hypothesized that participants in the two dieting conditions would show significantly greater decreases in bulimic symptoms than WL participants.

The third aim was to investigate the impact of the dieting manipulations on factors that may account for any effects of these interventions on bulimic symptoms. We included measures of perceived hunger and disinhibited eating because researchers have posited that dieting may increase the risk for binge eating because it results in chronic hunger and disinhibited eating (Polivy & Herman, 1985). We also included a measure of dietary restraint because it provided an opportunity to investigate the validity of this scale by testing whether it detected the dieting manipulation. This is important because recent studies have suggested that dietary restraint scales are not valid measures of acute or chronic dietary restriction (Bathalon et al., 2000; Jansen, 1996; Stice, Fisher, & Lowe, 2004). Finally, we included a measure of body satisfaction because we wanted to test the hypothesis that a reduction in body mass—assuming dieting would be successfully manipulated—would result in improved body satisfaction. This prediction emerged from the finding that body mass is one of the most consistent predictors of future increases in body dissatisfaction (Stice, 2002).

**Method**

**Participants**

A total of 160 females (mean age = 21.1, ±2.1; mean body mass index [BMI] = 26.0, ±4.9), were recruited from a large southwestern city through newspaper ads and flyers that invited women to participate in a dieting study. On average, participants were overweight, indicating that they represented typical dieters. Although open to the community, the sample consisted primarily of college students (92%). The sample included 31% Asians/Pacific Islanders, 8% Blacks, 39% Caucasians, 21% Hispanics, and 1% who specified other or mixed racial heritage. We excluded individuals who denied dieting in the past year because it did not seem important to generalize our findings to non-dieters. We also excluded individuals with an initial BMI of 20 or less because we did not want to encourage weight loss that might be medically hazardous. Participants were also excluded if they suffered from a heart or medical condition or were pregnant. Out of the 212 females who expressed interest, 42 were excluded for these reasons and 10 refused to participate.

**Procedure**

Participants were randomly assigned to one of three conditions: MSM (n = 59), ND (n = 51), and WL (n = 50). Participants completed a 15-min survey and a 30-min structured interview and had their weight and height measured by female research assistants at pretest and posttest 6 weeks later. Female clinical assessors received instruction in structured interview skills, reviewed diagnostic criteria for DSM-IV eating disorders (American Psychiatric Association, 1994), and observed interviews. They demonstrated acceptable inter-rater agreement (kappa > .80) with experts using tape-recorded interviews before collecting data.

The two treatment conditions, MSM and ND, consisted of four 1-h sessions every other week over 6 weeks and the completion of weekly food diaries. We selected a 6-week duration for the weight loss interventions because the average diet lasts approximately 4–6 weeks (Williamson, Serdula, Anda, Levy, & Byers, 1992). Groups were composed of 6–10 participants and were facilitated by a doctoral student in clinical psychology and co-facilitated by an undergraduate. Participants were asked to maintain a diet of 1200 calories per day and the sessions were adapted from Brownell’s LEARN program (Brownell, 1997). They were told that weight and food intake would be monitored. WL participants completed pretest and posttest assessments, and had the option to participate in the diet at study completion. Participants received $20 for completing each assessment.
Many small meals intervention

Session 1
To enhance motivation for weight loss, the group initially discussed the costs of obesity, including social, physical, and emotional consequences, such as increased likelihood of cardiovascular disease and reduced self-esteem. Facilitators offered strategies for making lasting lifestyle changes to better balance energy intake and output. Participants were asked to limit accessibility of high-fat, high-calorie foods in their homes. Facilitators recommended meals that included fibrous foods such as apples, raisins, and bran. Foods high in carbohydrates, protein, and vitamins were promoted over those high in fat, as high fiber carbohydrate foods result in lower hunger and lower subsequent energy intake. Participants were also told that limiting the variety of foods would help maintain weight loss. To ensure an individualized plan, participants determined replacement strategies that would be personally effective, such as eating low fat low sugar frozen yogurt instead of ice cream in order to maintain 1200 calories while still enjoying dessert. Daily intake of three meals and healthy snacks was recommended with the rationale that regular meals make people less likely to eat highly dense foods, with the proviso that snacks should consist of food such as carrots, bananas, rice cakes, and smoothies with fresh fruit and non-fat plain yogurt rather than highly processed foods such as chips. As homework, participants completed food diaries representing 3 weekdays and 2 weekend days between each session because a necessary component to successful dieting is to become aware of caloric intake. Documenting food choices putatively increases awareness of what is being ingested.

Session 2
For motivational enhancement purposes, participants brainstormed benefits of healthy weight. Participants also reviewed material from the previous session, such as the utility of replacement strategies. The need to take ownership over the diet was emphasized as a method for greater success and maintenance of loss. Participants were reminded that one is less likely to make poor eating choices if high fat foods are not in the home. Participants verbalized the foods that they would not purchase or select in the cafeteria or stores over the next 2 weeks.

Session 3
After reviewing the past session, participants discussed problems they encountered while trying to limit caloric intake and maintain 4–5 smaller meals per day. Group members and facilitators suggested ways to overcome these barriers, such as carrying portable snacks to accommodate busy schedules. Participants were asked to list the benefits of exercise. Moderate exercise was encouraged because physical activity can increase caloric output and weight maintenance requires the balance of input and output.

Session 4
After reviewing the previous session, participants troubleshooted problem areas and discussed what had and had not worked over the previous 6 weeks. They were encouraged to make a commitment to a lifestyle change and techniques such as eating slowly, not finishing everything on the plate, and creating a meal schedule were discussed. Finally, participants discussed the healthy behavior changes they hoped to maintain after the group ended.

Normal diet intervention

Sessions 1–4
Similar to the MSM condition, participants randomized to the ND condition were asked to maintain a diet of 1200 calories per day. The sessions were adapted from Brownell’s LEARN program (Brownell, 1997), and participants were told that weight data would be collected by facilitators and food intake would be self-monitored. The key difference between the two dieting interventions was that participants in this condition were not instructed to consume many small meals per day, but were instead encouraged to diet normally, meaning that participants learned strategies similar to people in the MSM condition except for the meal distribution component. Because eligible participants had dieted in the past year, and significant portions of
dieters skip meals, it was expected that being told to diet as normal would implicitly prescribe meal skipping as a method to reduce caloric intake. In all other respects, the four sessions included the same information as the MSM condition.

Measures

Obesity

The body mass index (BMI = kg/m²) was used as a proxy measure of body fat. Height was measured to the nearest millimeter using stadiometers, and weight was assessed to the nearest .1 kg using digital scales. Two measures of height and weight were obtained and averaged. The BMI shows correlations between .80 and .90 with direct measures of total body fat such as dual energy X-ray absorptiometry (Dietz & Robinson, 1998).

Meal structure

Food diaries were used to assess the meal distribution of participants. Participants also documented daily caloric intake in their food diaries and these data were used to determine whether experimental manipulation was successful. We interpreted the food diaries with caution because people typically under-report the amount of calories ingested (Wadden et al., 2002). The use of objectively garnered BMI in addition to self-report was another way to determine whether or not dietary restraint occurred.

Bulimic pathology

The Eating Disorder Examination (EDE), a semi-structured psychiatric interview, was used to assess DSM-IV criteria for eating disorders (Fairburn & Cooper, 1993). To streamline the interview, the version used included only diagnostic items assessing criteria for anorexia nervosa, bulimia nervosa, and binge eating. Items assessing frequency of binge eating, frequency of compensatory behaviors, and weight and shape overvaluation were summed to form a continuous measure of bulimic symptoms (z = .74). The symptom composite from the streamlined version of this interview used in the present study showed internal consistency (z = .96), 1-month test–retest reliability (r = .95), convergent validity with alternative measures of eating pathology, and sensitivity to detecting intervention effects in previous studies (Stice, Fisher, & Martinez, 2004; Stice et al., 2005).

Perceived hunger, disinhibition, and dietary restraint

The Three-Factor Eating Questionnaire (TFEQ) was used to assess perceived hunger, disinhibition, and dietary restraint, also termed control (Stunkard & Messick, 1985). An abbreviated 36-item version of this scale was used, which included only the true–false items, because it reduced respondent burden. The internal consistency coefficient for the abbreviated hunger, disinhibition, and dietary restraint scales (z = .73, z = .63, z = .71, respectively) were lower than observed in full scales studies (z = .85, z = .91, z = .92, respectively; Stunkard & Messick, 1985).

Body satisfaction

Body dissatisfaction was assessed with an adapted form of the Satisfaction and Dissatisfaction with Body Parts Scale (Berscheid, Walster, & Bohrnstedt, 1973). This scale asked participants to indicate their level of satisfaction with eight body parts on six-point scales ranging from 1 = extremely dissatisfied to 5 = extremely satisfied. Items were averaged for analyses, and the internal consistency in the current study was z = .88. This scale has shown acceptable internal consistency (z = .94), temporal reliability (3-week test–retest coefficient r = .90), and predictive validity for future bulimic symptoms onset (Stice et al., 2002).

Results

Preliminary analyses

A multivariate ANOVA model verified that participants assigned to the MSM, ND, and WL conditions did not differ significantly on age, ethnicity, parental education, BMI, bulimic symptoms, body satisfaction, or the
TFEQ hunger, disinhibition, or dietary restraint subscales \((F[16/294] = 1.51, \text{n.s.})\). In addition, a multivariate ANOVA model found that there were no differences between groups at baseline in terms of binging symptoms \((F[2, 157] = .47, p = .63, \text{\(\nu^2 = .00\)})\). These results suggest that randomization created equivalent groups at baseline.

As observed in other dieting studies (e.g., Schlundt et al., 1992), 17% of participants did not complete the study and provide posttest data. Participants were significantly \((\chi^2 [N = 160] = 9.46, p = .009)\) more likely to drop out of the MSM condition (25%) or ND condition (22%) than from the WL (4%) condition, however, dropout status was not significantly related to any other variable examined in this report. Nonetheless, we used full information maximum likelihood (ML) estimation, based on expectation-maximization (EM) algorithm, to impute missing data because this approach produces more accurate and efficient parameter estimates than list-wise deletion (a.k.a., completer analysis) or alternative imputation approaches such as last-observation-carried-forward (Schafer & Graham, 2002). It is also the recommended strategy for data in which missingness is significantly associated with one or more of the variables in the study. It should be noted, however, that all significant effects remained significant and all non-significant effects remained non-significant when the more common approach of list-wise deletion was used.

**Manipulation check**

To provide a check of our eating episode distribution manipulation, we reviewed food diaries; 96% of participants who completed the dieting groups filled out food diaries. Only 7% of participants in the MSM condition averaged at 3 or fewer eating episodes per day, whereas 95% of participants in the ND condition averaged 3 or fewer eating episodes per day. Participants in the MSM condition reported significantly \((t[76] = 11.95, p < .001, \text{\(\nu^2 = .42\)})\) more eating episodes per day \((M = 3.8, \text{SD} = .66)\) during the study than did participants in the ND condition \((M = 2.8, \text{SD} = .46; \text{Table 1})\). Thus, our experiment successfully manipulated eating episode frequency.

We also distinguished between snacks versus meals to determine how the difference in eating episode intake was generated in a randomly selected percentage of the dieters (71%, \(N = 78\)). Prior research has defined snack versus meal intake based on time of day rather than on caloric intake (e.g., Cullen, Watson, Baranowski, Baranowski, & Zakeri, 2005; Green, Wales, Lawton, & Blundell, 2000). However, because we did not ask participants to record the time of day for each eating episode, we defined snacks as an eating episode under 300 calories and defined a meal as an eating episode at 300 or above in caloric content. Participants in the MSM condition ate significantly more snacks per day on average \((M = .83, \text{SD} = .23)\) than participants in the ND condition \((M = .31, \text{SD} = .15; \text{\(t[76] = 11.95, p < .001\)})\). There was no significant difference \(t[76] = 1.48, p = \text{n.s.}\) in terms of the frequency of meals per day in the MSM \((M = 1.66, \text{SD} = .45)\) and ND conditions \((M = 1.81, \text{SD} = .48)\). Thus, results suggest that participants in the MSM engaged in more eating episodes relative to the ND condition because the former consumed more snacks, which was consistent with the request that they consume a greater amount of smaller meals and spread out their intake over the day.

To provide a check for our dietary restriction manipulation we conducted a repeated measure ANOVA model that tested for differential change in BMI across treatment (condition was a 3-level between-subjects factor and time was a 2-level within-subjects factor). The time-by-condition interaction indicated that there were significant differences in change in BMI across the three conditions \((F[2, 157] = 2.61, p = .029, \text{\(\nu^2 = .04\)})\). Follow-up models indicated that MSM participants showed significantly greater reductions in BMI than WL controls \((F[1, 107] = 6.49, p = .006, \text{\(\nu^2 = .06\)})\) and that ND participants showed significantly greater reductions in BMI than WL controls \((F[1, 99] = 3.91, p = .025, \text{\(\nu^2 = .04\)})\). There were no significant differences in change in BMI between the MSM and ND conditions \((F[1, 108] = .00, \text{n.s.}, \text{\(\nu^2 = .00\)})\). Follow-up paired \(t\)-tests confirmed that participants in the MSM and ND conditions showed significant decreases in BMI over time, but that WL participants did not show significant changes in BMI (Table 1). These results confirm that both dietary interventions resulted in significant weight loss relative to controls (i.e., participants in these conditions were in a state of negative energy balance). Although food diaries were not completed at baseline so change in caloric intake could not be investigated, the food diaries indicated an equivalent average intake of 1190 \((\text{SD} = 216)\) kcal for the MSM condition and 1168 \((\text{SD} = 283)\) kcal for ND participants over the 6 week.
intervention period, which did not represent a significant difference between conditions (\(F[1,108] = .21, \text{n.s.}, \ \chi^2 = .00\)).

**Intervention effects on bulimic symptoms**

A repeated measures ANOVA model indicated that there were significant differences in the changes in the continuous bulimic symptom measure over time across the three conditions (\(F[2,157] = 5.24, \ p = .003, \)

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Table 1

Means and standard deviations for MSM, ND, and WL conditions at pretest and posttest, as well as results of tests of within cell change over time

<table>
<thead>
<tr>
<th>Experimental condition</th>
<th>(n)</th>
<th>Baseline mean (SD)</th>
<th>(n)</th>
<th>Posttreatment (6 weeks) mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Calories/day</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Many small meals</td>
<td>59</td>
<td>25.53 (4.53)</td>
<td>44</td>
<td>25.20 (4.41)*</td>
</tr>
<tr>
<td>Normal diet</td>
<td>51</td>
<td>26.08 (4.07)</td>
<td>40</td>
<td>25.76 (4.08)*</td>
</tr>
<tr>
<td>Waitlist</td>
<td>50</td>
<td>26.54 (6.11)</td>
<td>48</td>
<td>26.50 (6.21)</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Many small meals</td>
<td>59</td>
<td>3.11 (1.22)</td>
<td>44</td>
<td>2.43 (0.84)*</td>
</tr>
<tr>
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<td>3.47 (1.16)</td>
<td>40</td>
<td>2.65 (0.85)*</td>
</tr>
<tr>
<td>Waitlist</td>
<td>50</td>
<td>2.93 (1.26)</td>
<td>48</td>
<td>2.65 (0.99)</td>
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<td><strong>Bulimic symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Many small meals</td>
<td>59</td>
<td>6.33 (2.54)</td>
<td>44</td>
<td>5.34 (2.49)*</td>
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<tr>
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<td>6.22 (2.61)</td>
<td>40</td>
<td>5.32 (2.51)*</td>
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<td>5.90 (2.58)</td>
<td>48</td>
<td>6.13 (2.83)</td>
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<td><strong>TFEQ hunger</strong></td>
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<td></td>
</tr>
<tr>
<td>Many small meals</td>
<td>59</td>
<td>8.25 (2.60)</td>
<td>44</td>
<td>6.32 (2.58)*</td>
</tr>
<tr>
<td>Normal diet</td>
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<td>8.76 (2.41)</td>
<td>40</td>
<td>6.83 (2.28)*</td>
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<td>6.75 (2.62)</td>
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<td><strong>TFEQ disinhibition</strong></td>
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<tr>
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<td>7.05 (2.28)</td>
<td>44</td>
<td>8.43 (1.91)*</td>
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<td>48</td>
<td>6.03 (3.06)</td>
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<tr>
<td><strong>Body satisfaction</strong></td>
<td></td>
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<tr>
<td>Many small meals</td>
<td>59</td>
<td>2.26 (0.69)</td>
<td>44</td>
<td>2.71 (0.82)*</td>
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<tr>
<td>Normal diet</td>
<td>51</td>
<td>2.08 (0.70)</td>
<td>40</td>
<td>2.50 (0.79)*</td>
</tr>
<tr>
<td>Waitlist</td>
<td>50</td>
<td>2.44 (0.96)</td>
<td>48</td>
<td>2.46 (0.99)</td>
</tr>
</tbody>
</table>

*Note: * denotes means that were significantly different across time at \(p < .01\) level.
$\chi^2 = .60$. Follow-up models indicated that MSM participants showed significantly greater reductions in bulimic symptoms than WL controls ($F[1, 107] = 7.00, p = .005, \chi^2 = .60$) and that ND participants showed significantly greater reductions in bulimic symptoms than WL controls ($F[1, 99] = 8.75, p = .002, \chi^2 = .80$). However, there were no significant differences in change in bulimic symptoms between the MSM and ND conditions ($F[1, 108] = .60, n.s., \chi^2 = .00$). Follow-up paired $t$-tests confirmed that participants in the MSM and ND conditions showed significant decreases in bulimic symptoms over time, but that WL controls did not show significant changes (Table 1).

To determine whether the dieting interventions produced changes in the behavioral symptoms of bulimia nervosa, we conducted follow-up tests focusing on change in binge eating and compensatory behaviors. Because the binge eating and compensatory behaviors frequency variables were skewed and normalizing transformations did not correct the violation of the assumption of normality we created a dichotomous variable reflecting the proportion of participants who reported any binge eating or compensatory behaviors at pretest and at posttest and used non-parametric models to test for change across conditions. Wilcoxin Signed Rank tests indicated that there were significant reductions in the proportion of participants who reported any binge eating or compensatory behaviors from pretest to posttest in the MSM condition (from 32% to 7%; $z = -3.44, p = .001$) and in the ND condition (from 47% to 16%; $z = -3.77, p < .01$), but not in the WL control condition (from 24% to 22%; $z = -3.0, p = .76$).

**Intervention effects on hunger, disinhibition, dietary restraint, and body satisfaction**

Repeated measures ANOVA models indicated significant time-by-condition interactions for hunger ($F[2, 157] = 4.86, p = .005, \chi^2 = .06$), disinhibition ($F[2, 157] = 8.18, p < .001, \chi^2 = .09$), and dietary restraint ($F[2, 157] = 8.25, p < .001, \chi^2 = .09$) across all three conditions. Follow-up models indicated that both MSM and ND participants showed significantly greater reductions in hunger than WL controls ($F[1, 107] = 9.16, p = .002, \chi^2 = .08$; $F[1, 99] = 6.75, p = .006, \chi^2 = .06$, respectively), but that there were no significant difference in this outcome across the MSM and ND conditions ($F[1, 108] = .27, n.s., \chi^2 = .00$). MSM and ND participants also showed significantly greater reductions in disinhibition than WL controls ($F[1, 107] = 12.87, p < .001, \chi^2 = .11$; $F[1, 99] = 13.94, p < .001, \chi^2 = .12$, respectively), but there were no significant difference across the MSM and ND conditions ($F[1, 108] = .01, n.s., \chi^2 = .00$). Similarly, MSM and ND participants showed significantly greater increases in dietary restraint than WL controls ($F[1, 107] = 13.96, p < .001, \chi^2 = .11$; $F[1, 99] = 15.03, p < .001, \chi^2 = .13$, respectively), but there were no significant difference across the MSM and ND conditions ($F[1, 108] = .02, n.s., \chi^2 = .00$). Paired $t$-tests indicated that participants in the dieting conditions showed significant decreases in hunger and disinhibition, and increases in dietary restraint, but that controls did not show significant changes in these outcomes (Table 1).

Similarly, a repeated measure ANOVA model indicated significant time-by-condition interaction for body satisfaction ($F[2, 157] = 12.21, p < .001, \chi^2 = .13$) across all three conditions. Follow-up models indicated that both MSM and ND participants showed significantly greater increases in body satisfaction than WL controls ($F[1, 107] = 19.76, p < .001, \chi^2 = .16$; $F[1, 99] = 18.97, p < .001, \chi^2 = .16$, respectively), but that there were no significant differences in this outcome across the MSM and ND conditions ($F[1, 108] = .15, p = .695, \chi^2 = .00$). Paired $t$-tests indicated that participants in the dieting conditions showed significant increases in body satisfaction, but that controls did not show significant changes in body satisfaction (Table 1).

**Discussion**

We experimentally tested whether a diet that promoted more frequent eating episodes would result in greater decreases in bulimic symptoms than a diet with fewer eating episodes because one possible explanation for the inconsistent findings regarding the relation of dieting to bulimic symptoms is that the prescribed diets promote healthy weight loss behaviors, whereas real world dieting involved unhealthy weight loss behaviors, such as meal skipping, which increase risk for bulimic symptoms. We conceptualize this as an experimental psychopathology test of the causal relation of dieting to bulimic symptoms, rather than a test of etiology or maintenance processes. It would be necessary to manipulate dieting among initially asymptomatic individuals
and test whether there is a differential rate of symptom onset to experimentally test whether dieting plays an etiologic role in the onset of bulimic symptoms. To experimentally test whether dieting maintains bulimic symptoms, it would be necessary to manipulate dieting among a sample of individuals with bulimia nervosa and test for differential rates of persistence. Our manipulation check suggested we succeeded in manipulating meal distribution, in that ND participants reported significantly fewer eating episodes than MSM participants; indeed this was the largest effect observed in the trial, accounting for 42% of the variance. Contrary to our first hypothesis, however, our results indicated that our experimental manipulation of eating episode frequency had no effects on bulimic symptoms. This finding converges with the null effects on change in binge eating observed in the one other study that manipulated long-term meal skipping (Schlundt et al., 1992). It was also noteworthy that there were no significant differences in weight loss across the many small meal and normal diets in our study or the Schlundt et al. (1992) study. A third study similarly found that weight loss was comparable at 24 months between participants in a snack dieting condition and participants in a no-snack dieting condition (Poston et al., 2005). Collectively, these findings do not support the suggestion that dieting characterized by meal skipping would result in less efficient weight loss (Morgan et al., 1986). It appears that dieting characterized by fewer eating episodes, and potential meal skipping, is not more likely to result in increased bulimic symptoms or weight gain than is dieting characterized by increased eating episode frequency. These findings imply that people tend to consume about the same amount of calories regardless of meal frequency (i.e., they titrate their overall caloric intake), which dovetails with lab-based experiments that found that acute caloric deprivation in the morning, versus no deprivation, had no effect on overall daily caloric intake (Agras & Telch, 1998).

The second aim of the present study was to provide an independent replication of a prior trial that found that a low-calorie diet resulted in decreases in bulimic symptoms relative to waitlist controls (Presnell & Stice, 2004). It is vital to attempt to replicate this effect because this was the first study to experimentally test whether dietary restriction resulted in increased or decreased bulimic symptoms. Our results confirmed that we successfully manipulated dietary restriction, in that participants in both dieting conditions lost significantly more weight than control participants. Results from the present study replicated the earlier findings: participants assigned to weight loss diets showed significantly greater reductions in DSM-IV bulimic symptoms and binge eating and compensatory behaviors, as assessed by blinded diagnostic interviews, than did waitlist controls. It is important to note that participants were overweight on average at baseline, which is typical of self-identified dieters (Lowe, 1993). Collectively, these results imply that dieting results in decreased bulimic symptoms. These finding converge with the results of six other randomized trials regarding the impact of experimentally manipulated dieting on changes in binge eating and bulimic symptoms (Burton & Stice, in press; Goodrick et al., 1998; Klem et al., 1997; Presnell & Stice, 2004; Reeves et al., 2001; Stice et al., 2005). Although participants in the current study were only followed for 6 weeks, participants in the Klem et al. (1997) study showed both weight loss effects and decreases in binge eating that persisted over the full 6-month intervention period, suggesting that this effect is not limited to brief periods of dietary restriction. The fact that seven randomized trials have found that dietary restriction results in decreased bulimic symptoms seems incompatible with the assertion that dieting is causally related to bulimia nervosa (Fairburn, 1997; Polivy & Herman, 1985). In fact, the absence of any experimental evidence that provides support for the dieting theory of bulimia nervosa suggests it might be useful to consider replacing this theory with one that accords with experimental findings.

There appear to be two possible explanations for the consistently contradictory findings between the prospective studies reporting that elevated dieting scores predict bulimic symptom onset and the experimental finding that dieting results in decreased bulimic symptoms. First, the inconsistent findings may have occurred because the prospective studies used invalid measures of dieting. 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, Jeffery, & Wing, 1994; Klesges, Isbell, & Klesges, 1992; Stice et al., 2004) that was found in the original validity studies that used self-reported caloric intake as the criterion (Laessle et al., 1989; van Strien Frijters, van Staveren, Defares, & Deurenberg, 1986). The fact that the prospective studies are not identifying participants that are actually engaging in dietary restriction probably explains why the results from these studies differ from experimental trials that directly confirm that participants are on weight loss diets by confirming that they show weight loss.
A second possible explanation for the contradictory findings is suggested by the inferential power of various research designs. Because randomized experiments rule out third variable explanations and prospective studies do not, the positive relation of self-reported dieting to future increases in bulimic pathology may have emerged because some third variable (i.e., a confound) 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 simply be a proxy risk factor for bulimic symptoms solely because it is a marker for chronic overconsumption. Stated differently, elevated scores on self-report dieting scales appear to be a risk marker rather than a causal risk factor.

Within this context, it is important to consider the evidence for the hypothesis that dieting serves as a maintenance factor for bulimic symptoms once they have emerged. Two prospective studies have tested whether dietary restraint predicts persistence of bulimic symptoms in community-recruited samples. Stice and Agras (1998) found that elevated dietary restraint predicted persistence of compensatory symptoms, but not binge eating, among adolescent girls over a 9-month follow-up. Fairburn et al. (2003) found that elevated dietary restraint did not predict persistence of binge eating or compensatory behaviors over a 5-year follow-up period among women who initially met criteria for full threshold bulimia nervosa. It has also been argued that CBT is an effective treatment for bulimia nervosa because it reduces dietary restraint, on the basis that the latter variable emerged as a significant mediator of the effects of this treatment (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002). However, we think this latter finding should be interpreted with caution for several reasons. First, because the EDE-restraint scale has not been found to show inverse correlations with objectively observed (Sysko, Walsh, Schebendach, & Wilson, 2005) caloric intake, it is not clear that it is a valid measure of dietary restriction. Second, because CBT for bulimia nervosa does not solely focus on reducing dietary restraint, but also targets several other factors (e.g., weight and shape overvaluation), treatment studies of this intervention do not permit focused inferences regarding the effects of dietary restriction. Third, because this study did not include a placebo control condition, it is not possible to rule out the possibility that the intervention effects for CBT are driven by expectancy effects, demand characteristics, or non-specific factors (e.g., clinical attention). Although we think it would be premature to conclude that dieting does not maintain bulimic symptoms, we feel that more experimental tests of this hypothesis are needed.

The third aim of the present study was to investigate the impact of the dieting manipulations on factors that may account for any effects of these interventions on bulimic symptoms. Contrary to what would be expected based on previous theory (Polivy & Herman, 1985), assignment to both dieting conditions resulted in greater decreases in disinhibited eating than were observed in the control group. Assignment to the dieting conditions also appeared to result in decreased hunger relative to the waitlist control condition. On the one hand, this latter finding may suggest that this scale has questionable validity. However, it may also suggest that people who reduce an overeating tendency experience a reduction in perceived hunger. One mechanism for such an effect might involve gastric capacity. Research has found that overeating results in an enlarged gastric capacity (Geliebter & Hashim, 2001); conversely, a reduction in overeating should result in a reduction in gastric capacity, which might lead to an overall reduction in perceived hunger. There were greater increases on the dietary restraint scale in the dieting conditions than in the waitlist control condition, which provides evidence that the TFEQ scale may be a valid measure of within-person change in dietary restriction. This finding converges with prior evidence that this dietary restraint measure decreased in response to low-calorie weight loss treatment for obesity (Lowe, Foster, Kerzhnerman, Swain, & Wadden, 2001).

There was also support for the hypothesis that weight loss diets would result in increased body satisfaction—a finding that also converges with effects in other randomized trials of dietary interventions (Stice et al., 2005). Presumably this effect emerges because people who are able to exercise control of their caloric intake and show reductions in weight are more satisfied with their bodies. This interpretation accords with the evidence that elevated body mass is a powerful risk factor for future increases in body dissatisfaction (Cattarin & Thompson, 1994).

Certain limitations of this study should be considered. First, because the meal frequency manipulation was rather subtle, care should be used when interpreting our findings. We initially attempted to directly encourage meal skipping in a pilot test by stating that many people use meal skipping to control weight, but most participants dropped from that group. Thus, both ethical and pragmatic considerations lead us to use a more
subtle manipulation. Although fewer ND participants skipped meals than anticipated, there was a large effect for differences in eating episode frequency across condition, which provides evidence that we manipulated this variable. In addition, the fact that our results converge with a study that directly instructed participants to skip a meal (Schnundt et al., 1992), suggests that the non-significant differences between the ND and MSM conditions cannot be easily ascribed to the subtle manipulation. The fact that we observed many significant effects in this trial also suggests that we had sufficient power to detect moderate-sized effects. Second, the dieting intervention used in this study may not be representative of real-world dieting, which places a limit on the ecological validity of the findings. For instance, the LEARN manual promotes stimulus control techniques, which may not be used in the real world as a dieting tool. Third, we observed relatively high attrition. Although we used a data imputation approach to correct for the potential impact of this on parameter estimates, findings should be interpreted with care.

In conclusion, our results suggest that dieting characterized by less frequent eating episodes does not appear to result in increased bulimic symptoms, or weight gain, relative to dieting characterized by more frequent eating episodes. Nonetheless, our results indicated that dietary restriction, regardless of eating episode frequency, results in decreases in bulimic symptoms. These findings, which converge with the experimental findings from six other randomized trials are incompatible with the dieting model of bulimia nervosa and instead suggest that dietary restriction is an efficacious method of reducing bulimic symptoms, as well as overweight and body dissatisfaction. Accordingly, future studies should provide more rigorous experimental tests of the effects of dieting, as well as other putative risk factors for bulimic pathology, as it appears that a sole reliance on prospective studies may result in faulty inferences about the causes of this pernicious eating disorder. Nonetheless, it will be important to investigate whether particularly rare types of unhealthy dieting, such as fasting, increase risk for bulimic pathology. It will also be important to search for moderators that may lead a subset of individuals engaging in dietary restriction to show onset of binge eating and bulimic symptoms, such as impulsivity, greater sensitivity to food reward, or very low weight.

References


