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Relation of Successful Dietary Restriction to Change in Bulimic Symptoms: A Prospective Study of Adolescent Girls

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Abstract

Recent experimental evidence that dietary restriction results in decreased bulimic and depressive symptoms seems inconsistent with findings from prospective studies and etiologic theory. However, because the dieting manipulated in these experiments may be unrepresentative of real-world weight-loss dieting, we tested whether successful dietary restriction was associated with decreases in these outcomes using longitudinal data from a school-based study of 496 adolescent girls. Moderately overweight participants who evidenced successful dietary restriction showed significantly greater decreases in bulimic symptoms than weight-matched participants who did not show successful dietary restriction, however, there were no effects for depressive symptoms. In conjunction with past experimental findings, results seem to imply that successful dietary restriction curbs bulimic symptoms, suggesting that current etiologic models may need revision.

Theorists have posited that dieting increases risk for onset of bulimic symptoms (Fairburn, Cooper, & Cooper, 1986; Huon, 1996; Polivy & Herman, 1985; Stice, 2001). Dieting is defined as intentional and sustained restriction of caloric intake for the purposes of weight loss (Herman & Polivy, 1975; Laessle, Tuschl, Kotthaus, & Pirke, 1989; Wadden, Brownell, & Foster, 2002; Wilson, 2002) and must result in a negative energy balance for weight loss to occur. Because dietary restraint scales do not appear to be valid measures of acute or chronic dietary restriction (Bathalon et al., 2000; French et al., 1994; Jansen, 1996; Stice, Fisher, & Lowe, 2004), it is useful to define weight-loss dieting in terms of a negative energy balance, as decreases in objective measures of body mass can be used to validate that individuals with elevated dietary restraint scores engaged in weight loss dieting. Another benefit of using a reduction in body mass to confirm dietary restriction is that it adjusts for caloric expenditure; this is important because a given caloric intake can result in weight loss, weight maintenance, or weight gain, depending on caloric expenditure. The rationale for using changes in measured body mass to confirm weight-loss dieting is that it is imperative to use an objective and valid operationalization of this behavior to advance our understanding of its effects. Others have also used documented weight changes to operationalize weight-loss dieting (e.g., Emmons, 1992).

Several mechanisms by which dieting might increase risk for bulimic symptoms have been proposed. 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,

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rather than physiological cues, leaves dieters vulnerable to uncontrolled eating when these cognitive processes are disrupted. Violation of strict dietary rules may also result in the temporary abandonment of dietary restriction because of the abstinence violation effect (Marlatt & Gordon, 1985). In addition, dieting might 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). Binge eating putatively precipitates redoubled dietary efforts and use of radical weight control techniques, such as vomiting, which develop into the self-maintaining binge-purge cycle (Fairburn et al., 1986). Others have theorized that the effects of dieting on bulimic symptoms is moderated by factors such as negative affect and overvaluation of weight and shape (Cools, Schotte, & McNally, 1992; Fairburn et al., 1986), although virtually no prospective or experimental studies have investigated factors that interact with dieting in the prediction of bulimic symptoms.

Consistent with the dieting theory of bulimia nervosa, several prospective studies have found that primarily normal weight school-recruited adolescent girls with elevated scores on dietary restraint scales are at increased risk for future onset of binge eating (Stice, Presnell, & Spangler, 2002; Stice, Killen, Hayward, & Taylor, 1998) and threshold or subthreshold bulimia nervosa (Killen et al., 1994, 1996), and future increases in bulimic symptoms (Stice, 2001). Although prospective studies provide support for the dieting theory of bulimia nervosa, some experimental findings seem incompatible with this theory. Randomized clinical trials have found that assignment to a low-calorie weight loss diet (e.g., 1200 calories a day) results in significantly greater decreases in binge eating for obese (Goodrick, Poston, Kimball, Reeves & Foreyt, 1998; Reeves et al., 2001) and overweight (Klem, Wing, Simkin-Silverman, & Kuller, 1997) adult women relative to waitlist controls. Participants on these weight loss diets lost significant weight, which confirms that dietary restriction was successfully manipulated. These results seem incompatible with the assertion that dietary restriction promotes bulimic symptoms and are troublesome for restraint theory because experiments are more effective than prospective studies in ruling out the possibility that a third-variable explains the dieting-bulimic symptom relation.

It is vital to clarify the nature of the relation between dietary restriction and bulimic symptoms because of the etiologic and prevention implications. If dieting increases the risk for bulimic symptoms, prevention programs should seek to eradicate dieting; however, if dieting decreases the risk for bulimic symptoms, prevention efforts should seek to promote effective dieting. Thus, we conducted two experiments that investigated the effects of manipulations of dietary restriction on bulimic symptoms. Our first study found that assignment to a 6-week low-calorie weight-loss diet resulted in greater decreases in bulimic symptoms in normal weight school-recruited late adolescent females relative to waitlist controls (Presnell & Stice, 2003). Because a prescribed low-calorie diet may be more restrictive than dieting practiced in the real world, our second experiment investigated the effects of a lower intensity weight-maintenance diet. Assignment to a weight-maintenance diet resulted in greater decreases in bulimic symptoms in normal weight school-recruited adolescent females over a 1-year period, relative to assessment-only controls (Stice, Presnell, Groesz, & Shaw, in press). Manipulation checks confirmed that the low-calorie diet resulted in weight loss and the weight-maintenance diet reduced risk for weight gain.

Although results from these five experiments seem incompatible with the dieting theory of bulimia nervosa, it is possible that prescribed weight loss diets are qualitatively or quantitatively different than dietary restriction that occurs in the real world. For instance, weight change is expected and monitored in these trials, which may result in more extreme dietary restriction. Moreover, two of these five trials used structured eating plans (i.e., consumption of regular meals throughout the day), which may be atypical of normal dieting. Indeed, Wadden et al. (2004) recommended that researchers examine large community samples

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to ascertain whether self-directed weight loss influences binge eating. Thus, the primary aim of the current study was to test whether successful weight-loss dieting in the real world is associated with increases or decreases in bulimic symptoms. Based on past experimental findings, we hypothesized that individuals showing successful dietary restriction would show greater decreases in bulimic symptoms over time than those not showing successful dietary restriction.

We focused on successful weight-loss dieting in this study for several reasons. First, Polivy and Herman (1985) argued that "successful dieting" that produces "weight loss" contributes to the emergence of bulimic symptoms. Second, it does not seem logical to conceptualize unsuccessful dietary restriction as a form of dieting. However, we do not mean to imply that dietary restraint scales measure successful dietary restriction. Numerous studies, varying in food types consumed, settings examined, and populations studied have found that dietary restraint scales were not inversely correlated with objective measures of acute (Jansen, 1996; Stice et al., 2004; van Strien, Cleven, & Schippers, 2000) or chronic dietary intake (Bathalon et al., 2000; French et al., 1994; Stice, 2001), as was suggested by the original validity studies that relied on self-reported intake (Laessle, Tuschle, Kotthaus, & Pirke, 1989; van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986) and the item content of the scales. For example, Bathalon et al. (2000) found that dietary restraint scores did not correlate with an objective biological measure of caloric intake (the doubly-labeled water method) over an 18-day period. Stice (2001) found that adolescent girls with elevated dietary restraint scores showed greater increases in BMI over a 2-year period relative to girls with lower dietary restraint scores. Indeed, the absence of a valid self-report measure of dietary restriction led us to use decreases in objectively measured body mass to validate that girls with elevated dietary restraint scores were actually engaging in weight-loss dieting.

Theorists have also suggested that dietary restriction increases the risk for depressive symptoms (Heatherton & Polivy, 1992; Stice et al., 1998). The disappointing results of most weight control efforts putatively increase the risk for depression. Further, tryptophan depletion from dietary restriction may contribute to negative affect by reducing the availability of serotonin (Kaye et al., 1998). Prospective studies have found that adolescent girls with elevated scores on dietary restraint scales are at risk for future increases in depressive symptoms and major depression onset (Stice & Bearman, 2001; Stice, Hayward, Cameron, Killen, & Taylor, 2000). However, randomized obesity treatment trials have found that low-calorie weight loss diets result in greater reductions in depressive symptoms in adults relative to waitlist controls (Foster, Wadden, Kendall, Stunkard, & Vogt, 1996; Rippe et al., 1998). Likewise, we found that a weight maintenance diet resulted in significantly greater decreases in negative affect in adolescent girls than observed in assessment-only controls (Stice et al., in press). Thus, the inconsistent findings from prospective versus experimental studies with regard to the relation of dietary restriction to depressive symptoms mirror the inconsistent findings regarding the relation of dietary restriction to bulimic symptoms. Accordingly, a secondary aim of this study was to test whether successful dietary restriction is associated with increases or decreases in depressive symptoms. Based on the findings from the randomized trials, we hypothesized that successful dietary restriction would be associated with declines in depressive symptoms. We used data from a prospective study of adolescent girls to address the two study aims because this is the developmental period of greatest risk for onset of bulimic and depression symptoms (Hankin et al., 1998; Stice et al., 1998) and because females are at greater risk for these disturbances than males (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993).

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Method

Participants

Participants were 496 adolescent girls from public and private middle schools in a metropolitan area (M age = 13 at Time 1; range 11–15). The sample was comprised of 2% Asian/Pacific Islanders, 7% African Americans, 68% Caucasians, 18% Hispanics, 1% Native Americans, and 4% who specified other or mixed racial heritage, which was representative of the ethnic composition of the schools from which we sampled (2% Asian/Pacific Islanders; 8% African Americans, 65% Caucasians, 21% Hispanics; 4% other or mixed). Average parental education, a proxy for socioeconomic status, was 29% high school graduate or less, 23% some college, 33% college graduate, and 15% graduate degree, which was representative of the metropolitan area from which we sampled (34% high school graduate or less; 25% some college; 26% college graduate; 15% graduate degree).

Procedures

The study was described to parents and participants as an investigation of adolescent mental and physical health. An active parental consent procedure resulted in an average participation rate of 56%, which was comparable to rates in other school-recruited samples that used active consent procedures and structured interviews (e.g., 61% for Lewinsohn et al., 1993). Participants completed a survey, participated in a structured interview, and had their weight and height measured by female research assistants at Time 1 (T1) and at three annual follow-ups (T2, T3, T4). Participants received a $15 gift certificate to a local book and music store for compensation at each assessment. Female clinical assessors with at least a bachelor's degree in psychology conducted interviews. Assessors attended 24 hours of training, wherein they were taught structured interview skills, reviewed diagnostic criteria for relevant DSM-IV disorders, discussed differential diagnoses, observed simulated interviews, and role-played interviews. Assessors demonstrated acceptable inter-rater agreement (kappa [k] > .80) with experts using tape-recorded interviews before collecting data. Assessors also received ongoing supervision and consultation on diagnostic issues and participated in clinical workshops addressing issues such as suicidality, confidentiality, and rapport-building. Assessments took place at the school during or immediately after school hours or at participants' homes. Assessors were blinded to the successful dietary restriction classification and to the hypotheses of this study.

Measures

Body mass—The body mass index (BMI= Kg/M^2) was used as a proxy measure of body fat. After removal of shoes and coats, height was measured to the nearest millimeter using stadiometers and weight was assessed to the nearest 0.1 kg using digital scales. Two measures of height and weight were obtained and averaged. The BMI correlates with direct measures of total body fat such as dual energy x-ray absorptiometry (r = .80 – .90) and correlates with health measures including blood pressure, adverse lipoprotein profiles, atherosclerotic lesions, serum insulin levels, and diabetes mellitus in adolescent samples (Dietz & Robinson, 1998).

Successful dietary restriction—Because dietary restraint scales do not correlate with objective measures of acute and chronic dietary restriction, we required that participants have both an elevated score on a dietary restraint scale and show a clinically meaningful decrease in body mass in order to be classified as exhibiting successful dietary restriction. Following established procedures (van Strien, 1997), we required that participants score above the median split (1.9 in our sample) on the Dutch Restrained Eating Scale (DRES; van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986). We also required that participants show clinically meaningful reductions in BMI. Based upon evidence that a 10% reduction in BMI results in significant reductions in risk factors for obesity-related diseases (Blackburn, 1995), a 10%

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reduction in BMI has been suggested as a clinical definition of successful weight loss (Jeffery et al., 2000). Our adolescent sample showed an average annual BMI increase of 2%, which has also been observed in nationally representative data (Kuczmarski et al., 2002), so we defined successful dietary restriction as an 8% decrease in BMI over a 1-year period, which corresponds to a 10% decrease after adjusting for age-related changes in BMI. Such successful dietary restriction is likely to be a function of both reductions in caloric intake and increases in exercise, however, this did not seem problematic because nearly 75% of female dieters report both reducing their caloric intake and increasing their physical activity (Levy & Heaton, 1993).

Bulimic symptoms—The diagnostic items from the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a structured interview, assessed DSM-IV criteria for bulimia nervosa (the non-diagnostic items were not administered to reduce respondent burden). We formed a symptom composite reflecting the total frequency of objective binge eating episodes and compensatory behaviors, including vomiting, diuretic use, laxative use, excessive exercise, and fasting, in the 3 months previous to each assessment point. As the symptom composite was skewed, a normalizing square root transformation was applied (untransformed means are reported in Table 2). The symptom composite from the adapted version of this interview showed internal consistency (α = .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., in press), as well as internal consistency in the current study (M α = .81). A randomly selected subset of participants (5%) were re-interviewed within a 3-day period by a second assessor who was blinded to the first diagnosis, resulting in high inter-rater agreement (*k* = .88). Another randomly selected subset of participants (5%) completed a second diagnostic interview with the same assessor 1-week later, resulting in high test-retest reliability (*k* = 1.0).

Depressive symptoms—An adapted version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Puig-Antich & Chambers, 1983), a structured diagnostic interview, assessed DSM-IV symptoms of major depression. The 13 severity rating items for each diagnostic symptom, which used ratings that ranged from 1 = *not at all* to 6 = *extreme*, were averaged to form a symptom composite. This composite showed internal consistency (α = .85) and 1-year test-retest reliability (*r* = .62) in prior studies (Stice, Burton, & Shaw, 2004), as well as internal consistency in the current study (M α = .82). A randomly selected subset of participants (5%) were re-interviewed within a 3-day period by a second assessor who was blinded to the first diagnosis, resulting in high inter-rater agreement (*k* = 1.0). Another randomly selected subset of participants (5%) completed a second diagnostic interview with the same assessor 1 week later, resulting in high test-retest reliability (*k* = 1.0).

Results

Preliminary Analyses and Descriptive Statistics

Of the initial 496 participants, 10 (2%) did not provide data at T2, 10 (2%) did not provide data at T3, and 6 (1%) did not provide data at T4, although only 2 did not provide data at any of the follow-ups (1%). Participants missing data at any assessment point did not differ significantly from the remaining participants on demographic factors or any of the study variables, suggesting that attrition should not introduce bias.

From T1 to T2 19 participants (3.8%) showed a 10% age-adjusted decrease in BMI, 33 (6.7%) showed a 10% age-adjusted increase in BMI, and 439 (88.5%) showed less than a 10% age-adjusted change in BMI. Corresponding rates were 5.1%, 6.3%, and 88.6% for T2 to T3 and 5.7%, 5.3%, and 89.0% for T3 to T4. Because an average of only 24 participants showed a

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10% decrease in BMI scores over each of the 1-year intervals, we pooled participants who showed successful dietary restriction across any of the three intervals (T1 to T2, T2 to T3, or T3 to T4) to maximize statistical power. For participants who showed a 10% decrease in BMI over more than one 1-year interval, we used data from the first interval. In total, there were 68 adolescent girls who evidenced a 10% age-adjusted reduction in BMI over any of the three 1-year intervals in this study, though only 50 of these individuals scored above the median on the dietary restraint scale.

Because the 50 participants who showed successful dietary restriction had an average BMI that was higher than the average BMI of participants who did not show change in weight, we selected a subset of the latter group that matched the former group on initial BMI. Up to two participants from the no weight change group were matched to each of the 50 participants from the successful dietary restriction group (on the basis of an initial BMI score that was within 0.5 SD of the initial BMI score of the 50 subjects that showed successful dietary restriction), resulting in a final sample of 89 participants in the no weight change group. Analyses confirmed that these successful dietary restriction and no weight change groups did not differ significantly on initial BMI, dietary restraint, bulimic symptoms, depressive symptoms, or demographic factors (see Table 1). According to sex-and age-specific percentile cut-offs (Cole, Bellizzi, Flegal, & Dietz, 2000), 36% of participants were health weight, 42% were overweight, and 23% were obese.

As expected given the strategy used to create the comparison groups, the successful dietary restriction group showed significantly greater decreases in BMI over the 1-year period than in the no weight change group, as indicated by a significant time-by-condition interaction in a repeated measures analysis of variance (ANOVA) model ($F\ [1/127] = 33.19$, $p < .001$, 21.2% variance explained). The means and standard deviations for the outcomes in the two groups at each assessment are presented in Table 2, along with the results of the pairwise contrasts that tested whether there were significant changes in the means across time within each group. There was a highly significant decrease in BMI in the successful dietary restriction group (an average decrease of 7.1 kg), but no significant change in BMI in the no weight change group.

With regard to the bulimic symptoms exhibited by this school-recruited sample of adolescent females, the total frequency of binge eating and compensatory behaviors ranged from 0 to 44 at T1 and from 0 to 70 at T2. The bulimic symptoms were largely confined to a subgroup of participants: only 22.6% and 21.2%, at T1 and T2 respectively, reported one or more binge eating or compensatory episodes. None of the participants in this subsample met full diagnostic criteria for bulimia nervosa, but 13 reported at least monthly binge eating and compensatory episodes over the past three months.

Relation of Successful Dietary Restriction to Change in Bulimic and Depressive Symptoms

Regarding the primary aim of this study, the successful dietary restriction group showed significantly greater decreases in the frequency of binge eating and compensatory behaviors over time relative to the no weight change group, as indexed by a significant time-by-condition interaction ($F\ [1/130] = 6.44$, $p = .012$, 4.7% variance explained). Paired t-tests indicated that there was a significant decrease in bulimic symptoms in the successful dietary restriction group, but a non-significant change in the comparison group (see Table 2).

The secondary aim was to test whether successful dietary restriction was associated with decreases in depressive symptoms. There were no significant differences in change in depressive symptoms across the 1-year period for participants showing successful dietary restriction versus the comparison group, as indexed by a non-significant time-by-condition interaction ($F\ [1/129] = 0.23$, $p =$ n.s., 0.2% variance explained; see Table 2).

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To ensure that these effects were not due to weight loss resulting from aberrant circumstances, we excluded 16 participants that might have experienced weight loss because they (a) were taking psychotropic medications that can cause weight loss (e.g., stimulant medications or anti-depressants), (b) met criteria for major depression during the period of weight loss, (c) were pregnant, or (d) showed excessive increases in height (above the 90th percentile in the full sample). The pattern of findings remained the same when these participants were excluded (i.e., that all significant effects remained significant and all non-significant effects remained non-significant).

Discussion

Research on the relation of dieting to bulimic symptoms has generated contradictory results. The primary aim of this study was to test whether successful dietary restriction in the real world was accompanied by decreases in bulimic symptoms, as would be suggested by past experimental findings. As expected, moderately overweight school-recruited adolescent girls who showed successful dietary restriction evidenced significantly greater decreases in bulimic symptoms than those who did not show successful dietary restriction. These findings might be considered more ecologically valid than the experimental trials because participants did not undergo a prescribed weight control intervention. Moreover, the fact that we documented significant decreases in direct measures of body mass confirms that the successful dieters achieved a state of negative energy balance. It is unlikely that expectancies or experimenter bias contributed to these effects because both participants and assessors were blinded to the hypotheses and the successful dieting classification used in this study. Further, this effect remained when we excluded participants with medical conditions, psychiatric conditions, or excessive growth that might have caused relative weight loss.

These results dovetail with findings from two randomized experiments indicating that a weight loss diet and a weight maintenance diet resulted in significant decreases in bulimic symptoms in normal weight adolescent and late adolescent females (Presnell & Stice, 2003; Stice, Presnell et al., 2004), as well as with three randomized clinical trials that found that a weight loss diet resulted in significant decreases in binge eating in overweight and obese adult females (Goodrick, et al., 1998; Klem et al., 1997; Reeves et al., 2001). The results from these six studies collectively suggest that weight loss diets, weight maintenance diets, and successful dietary restriction evidenced in the real world all result in decreases, rather than increases, in bulimic symptoms. However, because three of these studies involved adult overweight or obese women, the findings from these studies may not generalize to normal-weight adolescent girls who are at highest risk for bulimic pathology onset. More confidence could be placed in the tentative conclusion that dietary restraint curbs bulimic symptoms if additional studies examined the relation of dieting to bulimic pathology in adolescent girls.

Our findings suggest that the inconsistent results from the prospective and experimental studies did not emerge because weight loss that occurs in the real world has different effects on bulimic symptoms than weight loss that occurs in prescribed weight loss interventions. It is therefore important to consider other explanations for the discrepant findings. One possible explanation for the contradictory findings is that the prospective studies used invalid measures of dietary restraint. Dietary restraint scales do not correlate with objective measures of acute (Jansen, 1996; Stice et al., 2004; van Strien et al., 2000) or chronic intake (Bathalon et al., 2000; French et al., 1994; Stice, 2001), which implies these measures are not valid measures of dietary restriction, as was suggested by the original validity studies that relied on self-reported intake (Laessle et al., 1989; van Strien et al., 1986).

Another interpretation of the findings from the validity studies that used objective measures of dietary intake is that most weight loss dieters do not enter a state of negative energy balance

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necessary for weight loss, but instead practice relative caloric restriction (i.e., are eating less than they want to eat rather than what is necessary to induce a negative energy balance; Timmerman & Gregg, 2003). If most people who attempt to lose weight through dieting do not reduce intake sufficiently for weight loss, it is possible that the dietary restraint scales are largely identifying unsuccessful weight loss dieters. This may explain why the results from the prospective studies that use these scales are inconsistent from the findings from studies that experimentally manipulate successful dietary restriction. Although dieters who successfully lose weight and those who do not both engage in cognitive restraint, there may be important differences between individuals who effectively restrict their caloric intake and those who do not. These two groups may differ on the behavioral or cognitive approaches used for weight control (e.g., successful dieters may replace high fat foods with healthy foods, such as fruit and vegetables, whereas unsuccessful dieters may omit healthy foods to reduce their caloric intake) or on individual difference factors (e.g., subjective reinforcement from eating).

A third possible explanation for the contradictory findings between the longitudinal and experimental studies is that some unmeasured variable explains the prospective relations between dieting and future development of bulimic symptoms (Stice, Cameron, Killen, Hayward, & Taylor, 1999). This explanation is suggested by the fact that randomized experiments are more effective in ruling out third variable explanations than prospective studies. We posited that a chronic tendency towards caloric overconsumption may lead to both self-reported dieting and eventual onset of bulimic symptoms (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. Research should test whether a propensity towards overconsumption, assessed via objective and unobtrusive procedures, is a risk factor for future onset of dieting and bulimic symptoms.

A fourth possible explanation for the conflicting findings is that prescribed diets promote healthy dietary behaviors, but that it is unhealthy dietary behaviors that lead to bulimic symptom onset. The evidence that 20–50% of dieters report skipping meals (Levy & Heaton, 1993) suggests that such potentially unhealthy dieting behaviors are prevalent. It will be necessary 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 prescribing daily meal skipping or a diet proscribing meal skipping. However, one experiment that took this approach did not find any effects of meal skipping on binge eating (Schlundt, Hill, Sbrocco, Pope-Cordle, & Sharp, 1992).

A fifth possible explanation for the inconsistent effects is that the relation of dieting to bulimic symptoms is moderated by body mass or age, as all of the prospective studies finding that dietary restraint predicted future onset of bulimic symptoms involved largely normal weight adolescent girls, whereas three of the five experimental studies that manipulated dieting involved overweight or obese adult women. Dieting in a normal weight adolescent girl may produce different effects on eating behavior than dieting in overweight or obese individuals who are probably in a state of positive energy balance (Wadden et al., 2004). In line with this suggestion, post hoc analyses revealed that the effect of successful dietary restriction on decreases in bulimic symptoms was significantly stronger for participants with higher initial BMI scores ($b = 0.83$, $p < .001$, 9.0% variance explained). However, age did not moderate the relation between successful dietary restriction and change in bulimic behaviors, though the narrow age range of the sample might explain this null finding. These findings suggest that it will be important to test whether body mass moderates the relation of dieting to bulimic symptoms in future studies.[1] It will also be important for additional experimental studies to

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examine the effects of dieting in normal weight adolescents, because there is a dearth of studies in this area.

The secondary aim of this study was to test whether successful dietary restriction results in increased negative affect, as hypothesized previously (Heatherton & Polivy, 1992), or decreases in negative affect, as observed in randomized trials of weight loss treatments (e.g., Foster et al., 1996). Contrary to expectations and the findings from weight loss treatment trials, successful dietary restriction was not associated with decreases in depressive symptoms. Although it is tempting to conclude that the null findings occurred because participants in this study lost less weight than those in obesity treatment trials, the adolescent girls in our study lost an average of 13% of their initial body mass, which is similar to the average degree of weight loss observed in treatment trials for women (Jeffery et al., 2000). This pattern of findings may suggest that such a reduction in relative body mass has a greater impact on mood for obese women relative to moderately overweight adolescent girls. Alternatively, the effects of successful dieting on mood may be more acute and not easily captured by our 1-year measurement interval. Most importantly, our results imply that participants who achieved weight loss did not experience any adverse effects on mood, as has been previously theorized.

There were several limitations to the current study. First, although we required that successful dieters show both elevated scores on a dietary restraint measure and clinically significant weight loss, it is possible that some of these individuals did not engage in volitional dieting to lose weight. The results were the same when we excluded participants who might have shown weight loss because of aberrant circumstances (e.g., used medications or showed anomalous increase in height), but it is impossible to more definitively confirm that each participant showed weight loss because of intentional dieting.[2] Second, because there is evidence that typical weight loss dieting efforts in the real world last from 4 weeks to 6 months (Levy & Heaton, 1993; Williamson, Serdula, Anda, Levy, & Byers, 1992), it is possible that different effects might have emerged if we had used a shorter time interval between assessments. However, this limitation would be more likely to attenuate the magnitude of the findings, rather then reverse their valence. Third, because the individuals who evidenced successful dietary restraint were primarily overweight, findings may not generalize to normal weight adolescent girls who are at higher risk for bulimic pathology.

The current findings have several implications for future research. First, further experimental studies should examine the impact of dieting on bulimic symptoms, particularly in normal-weight adolescent girls, as bulimic pathology most commonly emerges in this population. As only one experiment examined the effects of a weight-loss diet and only one experiment has examined the effects of a weight-maintenance diet on normal-weight adolescent girls, we know little about the effects of longer-term or more severe dietary restriction in this group. Because

[1]To follow-up this interactive effect, we tested whether T1 DRES scores predicted onset of recurrent binge eating and compensatory behaviors among participants who were asymptomatic T1 and whether T1 BMI moderated this effect with the longitudinal data from the full sample of 496 adolescent girls followed over a 4-year period in this study. T1 DRES scores predicted future onset of bulimic symptoms (OR = 1.75, $p < .001$), but this effect was not moderated by T1 BMI (OR = 1.0, $p = .98$). We also tested whether initial BMI moderated the relation between assignment to a weight loss diet, versus assignment to a waitlist control condition, on change in bulimic symptoms over time with the data from the Presnell and Stice (2003) experiment. Results indicated that initial BMI did not moderate the significant main effect of the weight loss diet on change in bulimic symptoms ($r = .07$, $p = .394$). Finally, we tested whether initial BMI moderated the relation between assignment to a weight maintenance diet, versus assignment to an assessment-only control condition, on change in bulimic symptoms over time with the data from the Stice et al. (in press) experiment. Results indicated that initial BMI did not moderate the significant main effect of the weight maintenance diet on change in bulimic symptoms ($r = .03$, $p = .981$). Collectively, these findings provide limited support for the suggestion that the effects of dieting on bulimic symptoms is moderated by BMI, but it will be important to test for this relation in future experimental and prospective studies.

[2]Because individuals who lost weight volitionally might be expected to report at least as much dietary restraint at the end of the 1-year study period as at the beginning, we re-estimated the models excluded the 12 participants successful weight loss participants who showed a decrease in their DRES scores over the 1-year period. Results indicated that all significant effects remained significant and all non-significant effects remained non-significant.

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bulimic symptoms only emerge in a subset of those who report dieting, it is possible that an atypical and unhealthy form of dieting, rather than typical dieting, increases risk for bulimic symptoms. Second, it will be crucial to attempt to manipulate "dieting as usual" to provide a more rigorous experimental test of whether dietary restriction, in the form that it takes in the real world, results in decreased bulimic symptoms. Third, future research should examine the specific behaviors that lead to successful weight loss among real world dieters. Fourth, it will be important to develop valid measures of dietary restriction, because without such measures it will be impossible to make valid inferences about the effects of dieting. It is also essential to identify the construct that is assessed by extant dietary restraint scales. The fact that these scales identify individuals at risk for bulimic symptom onset suggests that they reliably measure some construct that plays a critical role in the etiology of this eating disorder (e.g., an inability to successfully modulate dietary intake). Finally, future prospective and experimental studies should examine factors that have been theorized to amplify the relation of dieting to bulimic symptoms, such as negative affect and overvaluation of weight and shape.

Regarding clinical implications, the current findings suggest that successful real world dietary restriction is associated with decreases in bulimic symptoms in moderately overweight adolescent girls. In conjunction with experimental studies that have manipulated weight-loss and weight-maintenance diets in a variety of populations, our findings seem to imply that effective caloric restriction curbs bulimic symptoms. In addition, these weight management interventions possess the added benefit of decreasing current weight and future risk for onset of obesity (Klem et al., 1997; Stice et al., in press). Thus, effective dieting holds much public health appeal because it may be a means to reduce both bulimic symptoms and obesity, the latter of which causes marked morbidity and mortality (Dietz & Robinson, 1998).

In conclusion, results suggested that successful dietary restriction is associated with decreases in bulimic symptoms, which converges with the findings from five experimental trials conducted by three independent labs. Collectively, these results appear incompatible with the widely held belief that dieting increases risk for bulimic symptoms. Rather, these findings imply that effective dietary restriction reduces bulimic symptoms. These findings add to a literature that has generated results that are incompatible with the dieting theory of bulimic nervosa (e.g., Bachar, Canetti, & Berry, 2005; Lowe, Gleaves, & Murphy-Eberenz, 1998). Although additional experiments are needed to complete our understanding of the effects of the various forms of dietary restriction on bulimic symptoms, particularly studies of normal weight adolescent girls, these findings suggest that it may be time to revise our theories regarding the etiologic role of dieting in the development of bulimic symptoms. These findings also imply that future research should seek to develop eating disorder prevention programs, and potentially treatment interventions, that promote healthy weight management skills. Such interventions would have the added benefit of addressing the current epidemic of obesity that is responsible for far more premature deaths than all of the psychiatric disorders combined.

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Table 1

Descriptive Statistics for the Successful Dietary Restriction and No Weight Change Groups

T1 demographic factors	Successful dietary restriction N = 50 M (SD)	No weight change N = 89 M (SD)
Age	13.84 (0.74)	14.02 (0.71)
Dietary restraint	2.63 (0.85)	2.59 (0.78)
BMI	26.00 (4.99)	25.55 (4.66)
Ethnicity	%	%
Asian (%)	4.0	2.2
Black (%)	12.0	15.7
Hispanic (%)	22.0	22.5
Native American (%)	0.0	1.1
Caucasian (%)	60.0	51.7
Other (%)	2.0	6.7
Maternal education	%	%
Grade school graduate (%)	4.3	0.0
High school graduate (%)	19.1	17.9
College graduate (%)	40.4	42.9
Graduate degree (%)	10.6	9.5
Paternal education	%	%
Grade school graduate (%)	2.1	1.3
High school graduate (%)	17.0	16.3
College graduate (%)	34.0	32.5
Graduate degree (%)	8.5	15.0
School type	%	%
Public (%)	86.0	88.8
Private (%)	14.0	11.2

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Table 2

Means and Standard Deviations for the Comparisons between the Successful Dietary Group and the No-Weight Change Group

Outcomes	Baseline Mean (SD)	1-year follow up Mean (SD)
BMI		
Successful dietary restriction group	26.00 (4.99) $_a$	22.63 (4.17) $_b$
No weight change group	25.55 (4.66)	25.78 (4.77)
Binge eating and compensatory behavior frequency		
Successful dietary restriction group	2.70 (6.32) $_a$	0.42 (1.36) $_b$
No weight change group	2.21 (7.31)	3.43 (9.88)
Depressive symptoms		
Successful dietary restriction group	1.50 (0.43)	1.51 (0.43)
No weight change group	1.34 (0.31)	1.38 (0.35)

Note: N = 50 for the successful dietary restriction group and 89 for the no weight change group. Untransformed means and standard deviation are reported for bulimic symptoms, but the transformed versions were used in the analyses. Means within rows with different subscripts were statistically significantly different ($p < .05$).