



Published in final edited form as:

J Abnorm Psychol. 2010 May ; 119(2): 268–275. doi:10.1037/a0019190.

Weight Suppression Predicts Maintenance and Onset of Bulimic Syndromes at 10-Year Follow-up

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Abstract

Conflicting results have emerged regarding the prognostic significance of weight suppression for maintenance of bulimic symptoms. This study examined whether the magnitude of weight suppression would predict bulimic syndrome maintenance and onset in college-based samples of men ($n=369$) and women ($n=968$) at 10-year follow-up. Data come from a longitudinal study of body weight and disordered eating with high retention (80%). Among those with a bulimic syndrome at baseline, greater weight suppression significantly predicted maintenance of the syndrome, and, among those without a bulimic syndrome at baseline, greater weight suppression predicted onset of a bulimic syndrome at 10-year follow-up in multivariate models that included baseline body mass index, diet frequency, and weight perception. Future research should address mechanisms that could account for the effects of weight suppression over a long duration of follow-up.

Keywords

Bulimia; weight suppression; longitudinal; maintenance factor; risk factor

Weight suppression (the difference between highest previous adult weight and current adult weight (Lowe, 1993)) has gained increasing attention in models of the development and maintenance of bulimia nervosa (BN). Butryn, Lowe, Safer, and Agras (2006) demonstrated that the magnitude of weight suppression was a robust predictor of treatment response in cognitive behavioral therapy (CBT) for BN. Patients with greater weight suppression demonstrated worse treatment response and a greater likelihood of drop-out. Notably, the effects of weight suppression were observed after controlling for body mass index (BMI), dietary restraint, and weight and shape concerns (Butryn et al., 2006). In contrast, Carter, McIntosh, Joyce, and Bulik (2008) found that weight suppression predicted neither treatment completion nor treatment response in women with BN but did predict weight gain during CBT.

Discrepant results could reflect differences in application of CBT for BN between the two studies (Carter et al., 2008). Specifically, the CBT utilized for patients studied by Butryn et al. (2006) emphasized the early resumption of normal eating which may have elicited greater

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concerns about weight gain among patients who had a history of being significantly heavier. The CBT utilized for patients studied by Carter et al. (2008) did not prescribe regularly scheduled meals and snacks but addressed inadequate food intake as a potential trigger for binge-eating episodes. Discrepant results also could reflect differences between the two samples. Maximum weight suppression and variability of weight suppression was greater in the Butryn et al. (2006) study group compared to the Carter et al. (2008) study group suggesting that a restricted range may have contributed to null results in the latter study. The extent to which the prognostic significance of weight suppression may hinge on the treatments employed and the population studied raises questions about the generalizability of this finding. The ability of weight suppression to prospectively predict changes in bulimic symptoms in a non-clinical sample is central to supporting its role as a risk factor independent of the potential effects of specific treatment interventions.

Weight suppression has been posited as a risk factor for the development and maintenance of BN (Lowe, 1993; Butryn et al., 2006). Women with family histories of overweight and higher personal premorbid weights may have a greater propensity towards excessive food intake and increased risk for binge eating (Fairburn, Welch, Doll, Davies, & O'Connor, 1997). Extreme dieting, in the form of fasting, to counteract genetic predisposition to overweight may facilitate substantial weight loss (and, thus, weight suppression) and further increase risk for developing binge-eating episodes (Stice, Davis, Miller, & Marti, 2008). Although most individuals with BN fall within a normal weight range at diagnosis (American Psychiatric Association, 2000), patients report having lost, on average, 7.1 to 9.4 kg (15.6 – 20.7 lbs.) from their highest previous weight at the beginning of treatment (Butryn et al., 2006; Carter et al., 2008). This substantial weight loss contributed to Russell's (1979) initial characterization of BN as “an ominous variant of anorexia nervosa.” In individuals who have lost the most weight, weight suppression may contribute to maintenance of bulimic symptoms by placing individuals in a “psychobiological bind” as described by Butryn et al. (2006) and discussed below.

In addition to a genetic predisposition to greater weight and greater drive to eat, individuals with high weight suppression may suffer from reduced leptin levels due to the loss of white adipose tissue responsible for the production of leptin (Friedman & Halaas, 1998). Several studies have documented blunted leptin concentrations in individuals with BN compared to weight-matched controls (Favaro, Monteleone, Santonastaso, & Maj, 2008). Circulating leptin levels trigger satiation and reduce food intake (Friedman & Halaas, 1998), suggesting that women with BN with reduced leptin levels may lack an important satiety signal which may further increase vulnerability to experience binge-eating episodes.

Individuals with high weight suppression may fear resumption of normal eating due to increased likelihood of significant weight gain following discontinuation of bulimic symptoms (Carter et al., 2008; Lowe, Davis, Lucks, Annunziato, & Butryn, 2006). Weight gain following resumption of normal eating in those with high weight suppression may result from slowed metabolic rate (Butryn et al., 2006) as well as a tendency to return to a weight influenced by genetic make-up, as reflected in family history (Fairburn et al., 1997) and highest previous adult weight. Of interest, previous studies have found no significant association between weight fluctuation (measured as either the difference between highest and lowest adult body weight or between lowest and current body weight) and maintenance of BN (Butryn et al., 2006; Carter et al., 2008). These results suggest that it is the state of weighing less than one's previous highest weight that may contribute to maintenance of bulimic symptoms rather than that bulimic symptoms are simply associated with greater weight fluctuations. Importantly, this psychobiological model for the association between weight suppression and development and maintenance of bulimic symptoms should be observed in community settings and in both women and men.

The current study extends the literature on weight suppression in three ways. First, it examines the predictive significance of weight suppression for bulimic symptoms and syndromes in a non-clinical sample. Second, it examines its predictive significance over a long duration of follow-up, 10 years following baseline assessments compared to follow-up durations of 18 weeks (Butryn et al., 2006) or less (Carter et al., 2008) in previous studies of BN. Third, the current study included both women and men allowing for the first examination, to our knowledge, of the predictive significance of weight suppression in men.

Methods

Participants

Participants came from two cohorts recruited in 1982 and 1992 and completed prospective 10-year follow-up assessments in 1992 and 2002, respectively. In the springs of 1982 and 1992, a total of 1,188 women and 511 men participated in a survey of health and eating patterns (Zuckerman, Colby, Ware, & Lazerson, 1986; Heatherton, Nichols, Mahamedi, & Keel, 1995). As reported previously, racial/ethnic breakdown of the 1982 and 1992 cohorts was 78% Caucasian, 6% Black, 11% Asian, and 5% Hispanic (Zuckerman et al., 1986; Heatherton et al., 1995). In addition to recruiting a new cohort of participants in 1992, the 1982 cohort was contacted to participate in 10-year follow-up assessments in 1992 (Heatherton, Mahamedi, Striepe, Field, & Keel, 1997), and the 1992 cohort was contacted to participate in 10-year follow-up assessments in 2002. Among those contacted, three women and three men had died, leaving a potential sample of 1,185 women and 508 men, among whom 968 (82%) women and 369 (73%) men participated. No significant differences were found between the 1982 and 1992 cohorts in 10-year follow-up participation rates for women ($\chi^2(1)=0.04$, $p=.83$) or men ($\chi^2(1)=1.57$, $p=.21$). This is the first report to include 10-year follow-up data for the 1992 cohort ($N=622$), and the first examination of weight suppression as a variable of interest.

Procedure

Written consent forms and surveys were mailed to participants up to three times to obtain as high a participation rate as possible. In addition, in 2002, a web-based version of the survey was offered for convenience. Confidentiality of on-line data was ensured by the use of encryption software and randomly assigned login/passwords. A small proportion of respondents ($n=29$; 5%) opted to complete the on-line version of the survey, and no significant differences were found for mean item scores or internal reliability of scales based on measurement type (all p values $> .05$), consistent with data indicating that computer versus paper-and-pencil administration has little effect on a questionnaire's psychometric properties (Vispoel, Boo, & Bleiler, 2001). Items covered demographic background, height and weight, dieting, eating disorder symptoms, and other health-related behaviors. Survey items were held constant across assessments to ensure comparability of data between cohorts and over time.

Utilizing a two-stage design, we contacted individuals whose survey responses indicated the presence of an eating disorder during baseline or follow-up surveys (cases) and demographically-matched controls to complete structured clinical interviews in a case-control design initiated in 2002. Among 232 participants who had survey-based eating disorder diagnoses at baseline or follow-up, 160 (69%) volunteered for interview assessments.

Measures

Bulimic symptoms were measured using items from the *Eating Disorders Inventory* (EDI; Garner, Olmstead, & Polivy, 1983), forming a version of the Bulimia subscale available when the study was initiated in 1982. The same items were used in each assessment. Notably, the EDI Bulimia scale contains several items that assess propensity for binge eating and only a single item assessing thoughts of trying to vomit. Thus, the scale focuses heavily on binge-

eating behavior. Analyses support factor invariance between the original EDI subscales published in 1983 and those empirically derived from the items included in the current study (Keel, Baxter, Heatherton, & Joiner, 2007). Analyses further support factor invariance of the EDI in men and women and from baseline to 10- and 20-year follow-up (Keel et al., 2007). Supporting the representativeness of our sample, mean scores did not differ significantly between our female participants and the female comparison sample in Garner et al. (1983) ($p=.20$). However, men in our sample reported lower mean Bulimia scores (mean(SD)=0.7 (1.6)) compared to their male comparison sample (mean(SD)=1.0 (1.8)) ($t(671)=2.31, p=.02$). Research supports the validity of the EDI in discriminating between eating disorder patients and non-eating disorder controls (Garner et al., 1983; Cooper, Cooper, & Fairburn, 1985). Within our own sample, EDI Bulimia scores differed significantly between individuals with and without BN ($t(1665)=13.45, p<.001; d=2.33$) and between individuals with and without a bulimic syndrome diagnosis ($t(1674)=22.16, p<.001; d=1.59$). Cronbach's alpha for the Bulimia scale was high in both cohorts and over time (α values $>.80$).

In addition to the EDI Bulimia scale, a separate set of questions probed for the presence and frequency of DSM-III-R BN symptoms. Responses to these items were used to create bulimic syndrome diagnoses based on computer algorithm (Keel, Heatherton, Dorer, Joiner, & Zalta, 2006). DSM-III-R diagnoses of BN required: a) Current binge eating occurring twice or more per week; b) lack of control over eating during binge episodes; c) use of self-induced vomiting, fasting, diet pills, laxatives, and/or diuretics to control weight once or more per week; and d) over concern with weight or shape. Algorithms for EDNOS diagnoses included: 1) a subthreshold BN-type EDNOS requiring a combination of over concern with weight or shape, recurrent binge-eating episodes and compensatory behavior at a frequency or duration that fell short of that required to diagnose full BN, 2) a binge eating disorder type EDNOS requiring binge eating episodes at least once per week, lack of control over episodes, and worry over episodes; and 3) purging disorder that required self-induced vomiting, laxatives, and/or diuretic use to control weight once or more per week and over concern with weight or shape. Using structured clinical interviews as the gold-standard, surveys demonstrated good sensitivity (.87 and .91, for the 1982 and 1992 cohorts, respectively) and good specificity (.96 and .99, for the 1982 and 1992 cohorts, respectively) despite the fact that interviews were conducted up to 10 or 20 years after the time frame on which survey-based diagnoses were based.

The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) (First, Spitzer, Gibbon, & Williams, 1998) was used to diagnose DSM-IV BN and EDNOS using the case-control design described above. For all interview-based EDNOS diagnoses, participants were required to endorse recurrent disordered eating behaviors that were associated with present distress, functional impairment, or increased risk of suffering from death, pain, or disability. Interviews were audiotaped with participant consent, and 15% were randomly selected to evaluate interrater reliability. Interrater reliability for eating disorder diagnosis was high ($\kappa=.79$ and .81, for the 1982 and 1992 cohorts, respectively). Interviewers documented the presence or absence of an eating disorder for the following time frames: lifetime, past month, and time of survey completion. Importantly, interviewers remained blind to whether participants had received a survey-based diagnosis while conducting interviews to allow for independent assessment of eating disorder status.

Body Mass Index (BMI; kg/m^2) was calculated from self-reported weight and height. Studies have demonstrated high correspondence between self-reported and objectively measured weight in both men and women of different age groups in the US (Stunkard & Albaum, 1981) as well as height and weight in both college-age men and women (Imrhan, Imrhan, & Hart, 1996) and middle-aged men and women (Spencer, Appleby, Davey, & Key, 2002). Small biases have been reported with men overestimating height by approximately 1.23 cm (0.48 inches) and women underestimating weight by 1.4 kg (3.1 pounds) (Spencer et al., 2002) with

absolute errors ranging from 1.00-3.54% (Imrhan et al., 1996). However, studies have uniformly concluded that self-reported height and weight are reliable and valid for large-scale studies in which direct assessments of height and weight are not feasible (Imrhan et al., 1996; Spencer et al., 2002; Stunkard & Albaum, 1981). Within our sample, stability in self-reports of height from baseline to follow-up was high, $r(1323)=.98, p<.001$ as was rank ordering for body weight, Spearman's $r(1318)=.85, p<.001$.

Weight suppression was assessed using the difference between highest previous adult body weight and current body weight, consistent with its conception by Lowe (1993) and its definition in previous studies (Carter et al., 2008; Butryn et al., 2006; Lowe et al. 2006; Lowe, Thomas, Safer, & Butryn, 2007). As with BMI, weights were based on self-report.

Participants' weight perception was assessed by a single item with response options of Very Underweight, Underweight, Average, Overweight, and Very Overweight. This item has demonstrated predictive validity in examining prospective changes in EDI Bulimia scale scores in women and men at 10- (Heatherton et al., 1997; Vohs, Bardone, Joiner, Abramson, & Heatherton, 1999) and 20-year follow-up (Keel et al., 2007).

Dieting frequency was assessed using a single item with response options of Never, Rarely, Sometimes, and Often. A single-item assessment of current dieting demonstrated concurrent validity with a validated self-report measure of caloric intake in both women and men, and these associations were similar to those found for a multi-item measure of dietary restraint (Neumark-Sztainer, Jeffery, & French, 1997). Like other measures of dietary restraint, self-reported dieting frequency is unlikely to be associated with actual caloric intake (Stice, Fisher, & Lowe, 2004). However, previous research supports significant associations between self-report measures of dieting and bulimic symptom levels (Lowe et al., 2007), suggesting the importance of including dieting frequency as a covariate in analyses.

Analyses

EDI Bulimia scores were positively skewed at baseline and 10-year follow-up and were transformed by taking their square root before being used in parametric analyses. Bivariate associations between baseline variables and bulimia scores at 10-year follow-up were examined using correlations. Multivariate associations were examined using hierarchical regression analyses. To explore the nature of prospective associations between weight suppression and changes in EDI Bulimia scores, logistic regression analyses were conducted to predict the presence of a bulimic syndrome at 10-year follow-up separately in those with a bulimic syndrome at baseline (reflecting bulimic syndrome maintenance) and those who did not have a bulimic syndrome at baseline (reflecting risk for developing a bulimic syndrome). Given the small number of independent variables examined and use of multivariate analyses, alpha was set at .05.

Results

Table 1 presents descriptive statistics for variables included in multivariate analyses, separately for men and women at baseline and 10-year follow-up. Bivariate analyses (see Table 2) supported significant correlations between all baseline variables and EDI Bulimia scores at both baseline and 10-year follow-up in women and men, with the exception of a non-significant correlation between weight suppression and BMI at baseline in women. A large effect size was observed for the association between EDI Bulimia scores at baseline and 10-year follow-up in both women and men, suggesting reasonable stability in rank ordering of symptom levels over time. However, EDI Bulimia scores decreased significantly in both women ($t(955)=18.76, p<.001$) and men ($t(362)=3.29, p<.001$) from baseline to 10-year follow-up.

In order to control for the association between weight suppression and EDI Bulimia scores at baseline, bulimia scores were entered in the first block and accounted for approximately 28% of variance in follow-up bulimia scores in both women and men. Cohort was entered in the second block. A significant effect of cohort was observed in women indicating greater decreases in bulimia scores in the 1982 compared to the 1992 cohort. No significant effect of cohort was observed in men. Remaining predictors (BMI, weight perception, dieting frequency, and weight suppression) were entered in the third block, and results from the final model are presented in Table 3.

In women, BMI, weight perception, dieting frequency, and weight suppression were all significant predictors of changes in EDI Bulimia scores. Higher BMI and weight perception were associated with higher bulimia scores at follow-up. Greater dieting frequency in college was associated with lower bulimia scores at follow-up, and higher weight suppression at baseline was associated with higher bulimia scores at 10-year follow-up.

In men, neither BMI nor weight perception at baseline significantly predicted EDI Bulimia scores at 10-year follow-up. Examination of standardized beta for analyses in women and men, suggest that non-significant results for BMI may reflect limited statistical power due to the smaller sample of men compared to women. Both dieting frequency and weight suppression were significant predictors of changes in bulimia scores. Unlike women, men who reported greater dieting frequency in college reported higher bulimia scores at follow-up. Consistent with results in women, greater weight suppression in college predicted higher bulimia scores at 10-year follow-up in men.

Importantly, patterns observed with EDI Bulimia scores could indicate that greater weight suppression predicted the maintenance of bulimic symptoms, less dramatic decreases in bulimic symptoms over time, or actual increases in bulimic symptoms from baseline to follow-up. In order to examine the nature of associations further, participants were divided on the basis of whether or not they had a bulimic syndrome diagnosis at baseline, and multivariate logistic regression analyses were used to determine whether weight suppression predicted the presence of a bulimic syndrome diagnosis at follow-up. Results of prospective logistic regression analyses are presented in Table 4.

Among individuals with bulimic syndromes at baseline, greater weight perception (but not BMI) and greater weight suppression (but not dieting frequency) predicted increased likelihood of disorder maintenance at 10-year follow-up (see Table 4). Sex was also a significant predictor; men demonstrated greater likelihood of syndrome maintenance than women. Among individuals without bulimic syndromes at baseline, greater weight suppression emerged as the only significant predictor of bulimic syndrome onset at 10-year follow-up.

Restricting analyses to those individuals for whom bulimic syndrome diagnoses were confirmed by structured clinical interview ($n=118$) produced the same pattern of results in which both greater weight perception and greater weight suppression at baseline predicted the maintenance of a bulimic syndrome at 10-year follow-up (weight perception: $\beta(\text{SE}(\beta))=1.09 (.43)$; Wald $\chi^2=6.40$, $p=.01$, OR (95% CI)=2.98 (1.28-6.94); weight suppression: $\beta(\text{SE}(\beta))=.08 (.03)$; Wald $\chi^2=7.72$, $p=.005$, OR (95% CI)=1.08 (1.02-1.14)). It was not possible to restrict analyses of onset to those interviewed because only a small subset of those without eating disorders at baseline were recruited for interviews in the case-control design.

Of note, individuals with bulimic syndromes at baseline ($n=183$) reported significantly greater weight suppression ($M(\text{SD})=9.21 (8.39)$ pounds) compared to individuals without bulimic syndromes ($n=1467$; $M(\text{SD})=6.74 (6.97)$ pounds); $t(1648)=4.41$, $p<.001$). Similarly, individuals with bulimic syndromes at follow-up ($n=59$) reported significantly greater weight suppression at follow-up ($M(\text{SD})=14.90 (13.56)$ pounds) compared to individuals without

bulimic syndromes at follow-up ($n=1261$; $M(SD)=9.21 (11.39)$; $t(1318)=3.71$, $p<.001$). Thus, we examined whether prospective associations between weight suppression at baseline and changes in EDI Bulimia scores at follow-up might reflect autocorrelation via a significant association between weight suppression at baseline and weight suppression at follow-up. At 10-year follow-up, participants reported a mean (SD) weight suppression of 9.47 (11.66) pounds which was significantly greater than weight suppression at baseline (7.11 [7.10] pounds) ($t(1309)=7.48$, $p<.001$). In addition, weight suppression at follow-up was significantly correlated with both weight suppression at baseline ($r=.34$, $p<.001$, $N=1310$) and EDI Bulimia scores at follow-up ($r=.20$, $p<.001$, $N=1328$). However, the partial correlation between weight suppression at baseline and EDI Bulimia score at follow-up remained significant when controlling for the effects of both EDI Bulimia score at baseline and weight suppression at follow-up ($r=.08$, $p=.007$, $N=1295$), suggesting that baseline level of weight suppression predicts trajectory of bulimic symptoms beyond the influence of autocorrelation.

Discussion

Consistent with results first reported by Butryn et al. (2006), magnitude of weight suppression was a significant predictor of maintenance of bulimic symptoms and syndromes. This effect was demonstrated in multivariate analyses that controlled for other factors implicated in the development and maintenance of BN, including BMI, weight perception, and dieting frequency. The effect was demonstrated over a long duration of follow-up and was found for both women and men. These results suggest that weight suppression is important for uncovering factors that may make it difficult for individuals to recover from BN, such as those proposed within a psychobiological model of symptom maintenance (Butryn et al., 2006). In addition, greater weight suppression in those without a bulimic syndrome at baseline predicted the onset of a bulimic syndrome at follow-up. To our knowledge, this is the first study to demonstrate a prospective association between weight suppression and the later development of a bulimic disorder. These findings are consistent with Russell's (1979) early conceptualization of BN as reflecting a variant of AN in which significant weight loss frequently preceded the onset of bulimia.

Although results for weight suppression were consistent between men and women, gender differences were observed for the predictive significance of dieting frequency. For women, higher dieting frequency at baseline predicted greater decreases in EDI Bulimia scores at follow-up in a multivariate model that included BMI, weight suppression, and weight perception. These findings are consistent with results from cross-sectional analyses of female patients with BN (Lowe et al., 2007). Lowe et al. (2007) suggested that individuals more intent on controlling their food intake through dieting may be more successful in decreasing the frequency of binge-eating episodes (a central feature measured by the EDI Bulimia scale). For men, higher dieting frequency at baseline was a significant predictor of greater EDI Bulimia scores at follow-up. Results in men are consistent with the restraint model which posits that dieting increases the risk for binge eating (Polivy & Herman, 1985). Previous comparisons of men and women in this sample (Heatherton et al., 1995; Keel et al., 2007) and in other samples (Rosen, Gross, & Vara, 1987; Tiggemann, 1994) suggest that men are more likely to perceive themselves as overweight and initiate dieting when they are actually overweight compared to women who perceive themselves as overweight and diet even when they are within a normal weight range. This may translate into different roles for dieting in men and women. In men, dieting frequency may reflect, in part, attempts to counteract tendencies to overeat that are themselves significant risk factors for developing and maintaining binge-eating episodes.

Finally, weight perception in college was a significant and robust predictor of maintenance of a bulimic syndrome at 10-year follow-up in multivariate logistic regressions including both sex and BMI. This finding is consistent with results supporting the predictive significance of

body image disturbance for course of eating disorders in clinical samples (Fairburn, Peveler, Jones, Hope, & Doll, 1993; Keel, Dorer, Franko, Jackson, & Herzog, 2005) and the potential value of interventions focused on body image (Cash & Lavalley, 1997; Delinsky & Wilson, 2006).

The current study had several noteworthy strengths. First, we had large samples of men and women with high retention rates over 10-year follow-up. This enabled us to detect even modest effect sizes as well as examine the prognostic significance of weight suppression in men for the first time. Second, we used a psychometrically sound measure of bulimic symptoms that allowed us to measure changes in symptom levels in a non-clinical sample. This feature is important for understanding whether associations between weight suppression and bulimic symptoms are restricted to patients receiving specific forms of intervention or reflect processes that impact illness course in a wider population of those who suffer from disordered eating. Third, we extended symptom-level findings by showing that weight suppression predicted the development and maintenance of bulimic syndromes in multivariate logistic regression analyses and confirmed that findings remained significant when restricting analyses to those for whom diagnostic status was confirmed by structured clinical interview. Fourth, our duration of follow-up speaks to the potential for weight suppression to have a long-term effect on bulimic syndrome maintenance and shifts attention from proximal factors to underlying mechanisms that may account for this association. Such factors may include the effects of weight suppression on biological processes that influence eating and weight maintenance and psychological processes related to the fear that recovery will cause weight gain.

Despite study strengths, certain weaknesses should be noted. Most analyses are based upon self-report data which may introduce error in assessment (Meyer, McPartlan, Sines, & Waller, 2009), and single-item assessments were used for several constructs. Importantly, all studies of weight suppression base highest previous adult weight on self-report given the lack of feasibility of directly measuring individuals at the given point in their lives when weight peaked. In addition, assessment error would decrease our ability to detect significant associations or might produce spurious results that would be inconsistent with previous findings. Neither problem was observed in our results. Another limitation is that findings come from a college-based sample which may not be representative of those who do not attend college in terms of weight or disordered eating patterns. Thus, care should be taken in generalizing findings to groups with different demographic backgrounds. Finally, results reflect findings for broadly defined bulimic syndromes and may or may not extend to maintenance or onset of BN or specific forms of EDNOS due to differences in symptom configuration, frequency, or duration.

Results add to a growing literature regarding the clinical significance of weight suppression for understanding the development and maintenance of bulimic syndromes. Future research should address the mechanisms that account for the effects of weight suppression. Specifically, studies could examine whether beliefs about the effects of disordered eating practices on weight or alterations in physiological processes that regulate eating and weight mediate associations between weight suppression and maintenance of bulimic symptoms.

Acknowledgments

This work was supported by a fellowship from the Radcliffe Institute and grants from the Milton Fund and National Institute of Mental Health (R01 MH63758). We thank the Radcliffe Institute's Henry Murray Center for providing access to data from Dr. Colby's *Prevalence of Bulimia Among College Students* and Dr. Heatherton's Follow-up and Replication of Prevalence Among College Students and the Alumni Office of Harvard University. Portions of this paper were presented at the 2008 Eating Disorders Research Society Meeting in Montreal, Canada.

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Table 1

Descriptive Statistics for Men and Women at Baseline and 10-year Follow-up.

Variable	Men N=369		Women N=968	
	Mean	SD	Mean	SD
Baseline				
BMI	22.74	2.59	21.52	2.72
Weight Perception	2.94	.56	3.40	.62
Diet Frequency	1.41	.75	2.48	1.11
Weight Suppression	5.34	5.58	7.80	7.50
EDI Bulimia	9.92	3.18	13.59	5.17
Follow-up				
BMI	24.44	2.88	22.45	3.61
Weight Perception	3.18	.57	3.26	.62
Diet Frequency	1.68	.95	2.18	1.04
Weight Suppression	6.85	9.90	10.48	12.12
EDI Bulimia	9.43	3.10	11.03	4.06
Bulimic Syndrome				
Baseline ^a	14	3.8	140	14.5
Follow-up	11	3.0	48	5.0

^aThe point prevalence of DSM-III-R BN was 2.9% in women and 0.8% in men at baseline, as reported previously (Keel et al., 2006), and 0.5% in women and 0% in men at 10-year follow-up.

Table 2

Correlations between Baseline Predictors and Follow-up Bulimia Scores in Men (above the diagonal) and Women (below the diagonal).

Variable	BMI	Weight Perception	Diet Frequency	Weight Suppression	Baseline Bulimia	Follow-up Bulimia
BMI	--					
Weight Perception	.56***	--				
Diet Frequency	.23***	.39***	--			
Weight Suppression	.00	.06*	.18***	--		
Baseline Bulimia	.30***	.47***	.51***	.13***	--	
Follow-up Bulimia	.33***	.37***	.27***	.11***	.57***	--

Note: BMI=Body Mass Index.

* p<.05

** p<.01

*** p<.001

Table 3
 Predictors of EDI Bulimia Scores at 10-year Follow-up in Men and Women from Hierarchical Multiple Regression.

Predictor	Men		Women	
	ΔR^2	Effect Size (f^2)	ΔR^2	Effect Size (f^2)
Step 1	.282***	.39	.326***	.48
Baseline EDI Bulimia		.46***		.53***
Step 2	.002	<.01	.007**	.01
Cohort		.06		.09**
Step 3	.074***	.08	.031***	.03
BMI		.10		.11**
Weight Perception		.02		.10**
Diet Frequency		.14**		-.07*
Weight Suppression		.15**		.08**
Total R^2	.358***	.56	.364***	.57
n	353		931	

According to Cohen (1992), $f^2 = .02$ is small, $f^2 = .15$ is medium, and $f^2 = .35$ is large.

* $p < .05$

** $p < .01$

*** $p < .001$

Table 4
 Prediction of Bulimic Syndromes at 10-year Follow-up in those with Bulimic Syndromes at Baseline (left panel) and those without Bulimic Syndromes at Baseline (right panel).

Predictor	Bulimic Syndrome at 10-Year Follow-up											
	Bulimic Syndrome Present at Baseline (n=145) Prediction of Maintenance						Bulimic Syndrome Absent at Baseline (n=1,129) Prediction of Onset					
	β	SE (β)	Wald χ^2	OR	95% CI	β	SE (β)	Wald χ^2	OR	95% CI		
Sex	-2.29	.92	6.17*	.10	.02-.62	.24	.56	.19	1.28	.43-3.79		
Cohort	.60	.58	1.06	1.06	.58-5.67	-.41	.43	.94	.66	.29-1.53		
BMI	-.03	.11	.08	.97	.78-1.21	.07	.07	.89	1.07	.93-1.23		
Weight Perception	2.48	.64	14.93***	11.96	3.40-42.12	.60	.41	2.16	1.81	.82-4.02		
Diet Frequency	.06	.32	.03	1.06	.56-1.99	.01	.20	<.01	1.00	.68-1.49		
Weight Suppression	.11	.03	12.05***	1.12	1.05-1.19	.05	.02	6.79**	1.05	1.01-1.08		

OR=Odds Ratio; CI=Confidence Interval