Published in final edited form as:
Behav Res Ther. 2012 April ; 50(4): 266–274. doi:10.1016/j.brat.2012.02.006.

Weight Suppression as a Predictor of Weight Gain and Response to Intensive Behavioral Treatment in Patients with Anorexia Nervosa

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Abstract

Previous studies have documented that weight suppression (a person's highest adult weight minus current weight) predicts weight gain and disordered eating symptoms during treatment of bulimia spectrum disorders, but no research has examined weight suppression in individuals with anorexia nervosa (AN). Thus, this study sought to characterize weight suppression in a large sample of patients with AN (N = 185), and to evaluate whether weight suppression at admission for intensive behavioral treatment predicts weight gain and clinical outcomes at discharge. Weight suppression varied from 0 kg to78 kg (M [SD] = 17.1 [10.8] kg) in AN patients. Higher levels of weight suppression predicted greater total weight gain, a faster rate of weight gain, and bulimic symptoms during intensive treatment even after controlling for body mass index on admission, length and type of intensive treatment received, restricting versus binge-eating/purging AN subtype, and other predictors of study outcomes. These findings converge with previous research documenting the clinical significance of weight suppression in the treatment of eating disorders. Future work is needed to replicate the current findings, and examine whether weight suppression predicts the course of AN following discharge from intensive treatment.

Keywords

eating disorders; anorexia nervosa; weight gain; binge eating; purging; treatment

There is a burgeoning literature documenting the clinical significance of weight suppression (i.e., a person's highest adult weight minus current weight; Lowe, 1993) in individuals with eating disorders. Cross-sectional studies have found positive associations between weight suppression and frequency of binge eating and purging in patients seeking treatment for bulimia spectrum disorders (Butryn, Juarascio, & Lowe, 2011; Lowe, Thomas, Safer, & Butryn, 2007). Moreover, longitudinal research has found that weight suppression predicts

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Disclosure Statement
The authors report no conflicts of interest.

the onset and maintenance of bulimic syndromes (i.e., threshold and subthreshold bulimia nervosa [BN], binge eating spectrum disorders, purging disorder; Keel & Heatherton, 2010; Lowe et al., 2011). There also is evidence that weight suppression predicts weight gain in individuals with BN during inpatient (Lowe, Davis, Lucks, Annunziato, & Butryn, 2006) and outpatient (Carter, McIntosh, Joyce, & Bulik, 2008) treatment and over five-year follow-up (D. B. Herzog, et al., 2010). In contrast, other measures of weight history, such as highest or lowest body mass index (BMI) at current height and the difference between lowest weight and current weight, have failed to demonstrate consistent associations with disordered eating symptoms or weight gain (Butryn et al., 2011; Butryn, Lowe, Safer, & Agras, 2006; Carter et al., 2008), suggesting that weight suppression may be particularly salient to the presentation and course of eating disorders.

Given its relation to bulimic symptoms and weight gain, there has been speculation that weight suppression might impact the treatment of individuals with eating disorders. However, findings in this area have been mixed. In an initial report, Butryn et al. (2006) documented that weight suppression predicted treatment dropout and persistence of binge eating and purging during cognitive behavior therapy (CBT) for BN, even after controlling for baseline BMI and cognitive correlates of disordered eating. However, more recent studies have failed to replicate these results. For example, Carter et al. (2008) found no association between weight suppression and treatment dropout or abstinence from binge eating and purging following CBT for BN, although weight suppression did predict weight gain and severity of purging (at a trend level) at the end of treatment. Furthermore, Zunker and colleagues (2011) found no relation between weight suppression and treatment completion, reduction of or abstinence from binge eating and purging, or weight gain following CBT-based interventions for BN and binge eating disorder (BED). There are several plausible explanations for these discrepant findings including differences among the studies with respect to sample composition and the type of CBT delivered, as well as the possibility that weight suppression is not a reliable predictor of treatment outcome. Additional research is needed to determine whether weight suppression has clinical utility in predicting response to eating disorder treatments.

Another topic that requires investigation concerns the association of weight suppression to the presentation and course of anorexia nervosa (AN). Although several studies have examined weight suppression in individuals with BN, BED, and their variants, no research has focused on weight suppression in AN. This may be due, in part, to the fact that in order to maintain a weight that is significantly below what is “minimally normal for age and height” (American Psychiatric Association, 1994), the majority of individuals with AN are weight suppressed; that is, they weigh less than they once did (the exception are individuals who fall off their growth trajectories and do not recover). Nevertheless, there is variation in weight history among AN patients (Coners, Remschmidt, & Hebebrand, 1999; Miyasaka et al., 2003), which suggests that there may be variability in weight suppression. For example, studies using retrospective review of school records and medical charts to document premorbid characteristics of AN patients have shown that a history of overweight is present in some individuals (Nielsen, 1985; Swenne, 2001) and may be more common in AN patients than in the general population (Swenne, 2001); moreover, one early report found that 28% of a sample of 102 AN patients had a history of obesity (Crisp, Hsu, Harding, & Hartshorn, 1980). Conversely, some individuals with AN have no weight suppression, as their illness develops in the context of a failure to make expected weight gains during a period of growth rather than following a significant weight loss (Rosen & the Committee on Adolescence, 2010).

Variations in weight suppression may have important implications for the treatment of AN. Because individuals with AN are, by definition, underweight, current treatment guidelines

emphasize weight restoration (American Psychiatric Association, 2006; National Institute for Clinical Excellence [NICE], 2004). For severely underweight patients, individuals who have lost weight rapidly, and those with serious medical or psychiatric complications, a structured behavioral intervention including the prescription of an adequate diet and supervised meals may be required to initiate weight gain and normalize eating behaviors (Attia & Walsh, 2009). Structured behavioral interventions typically are provided in intensive settings (e.g., inpatient, day hospital), and approximately 50% of individuals with AN require this level of care at some point during their illness (Agras et al., 2004). In light of evidence that weight suppression predicts weight gain during the treatment of BN (Carter et al., 2008; Lowe et al., 2006; but, see Zunker et al., 2011), it seems plausible that weight suppression might be associated with larger or more rapid weight gains during intensive treatment for AN. The mechanisms responsible for this effect probably would be similar to those hypothesized for other groups; e.g., increased metabolic efficiency (but, see Stice, Durant, Burger, & Schoeller, 2011), genetic predisposition, or hormonal abnormalities that disrupt satiety cues and increase vulnerability to overeating or binge eating (Butryn et al., 2006; Keel & Heatherton, 2010; Lowe et al., 2006). However, the clinical implications of an association between weight suppression and weight gain in AN patients are unclear. Larger or more rapid weight gains might increase the likelihood that patients achieve a healthy weight during intensive treatment, which has been associated with a decreased risk of re-hospitalization (Baran, Weltzin, & Kaye, 1995). Alternatively, several reports have documented that more rapid weight gain is associated with poor outcomes following AN treatment, including a decreased likelihood of achieving target weight, faster weight loss post-treatment, and higher rates of re-hospitalization (T. Herzog, Zeeck, Hartmann, & Nickel, 2004; Lay, Jennen-Steinmetz, Reinhard, & Schmidt, 2002; Willer, Thuras, & Crow, 2005; but, see Lund et al., 2009). Thus, weight suppression might predict a poor response to AN treatment by virtue of its relation to rate or degree of weight gain.

Finally, binge eating and purging are common in individuals with AN. Prospective studies have documented that up to 62% of individuals with the restricting subtype of AN (AN-R) eventually meet criteria for AN binge-eating/purging type (AN-B/P; Eddy et al., 2008), and crossover to BN occurs in up to 50% of patients (Anderluh, Tchanturia, Rabe-Hesketh, Collier, & Treasure, 2009). Although rates of binge eating and purging often decrease during AN treatment, particularly when it is provided in an intensive setting, these behaviors do persist or emerge in some individuals during weight restoration (Treat, McCabe, Gaskill, & Marcus, 2008). Given that weight suppression has been associated with higher rates of binge eating and purging following CBT for BN in some reports (Butryn et al., 2006; Carter et al., 2008), it is possible that weight suppression might predict the onset or persistence of bulimic symptoms during AN treatment. In particular, AN patients who are high in weight suppression might have an increased risk of binge eating relative to their non-weight suppressed counterparts, or they might be more likely to engage in compensatory behaviors out of fear that increased food intake will lead to uncontrolled weight gain (Butryn et al., 2006; Keel & Heatherton, 2010). Given that binge eating and purging have been associated with indices of severity (e.g., suicide attempts, self-injurious behaviors) and negative outcome in several cross-sectional and longitudinal studies of AN patients (for review, see Peat, Mitchell, Hoek, & Wonderlich, 2009), identifying potential predictors of these behaviors during intensive treatment could have important implications for future research and clinical practice.

In summary, there is growing evidence that weight suppression impacts the presentation and course of bulimia spectrum disorders, but no research has examined the clinical significance of weight suppression in patients with AN. Thus, this study sought to characterize weight suppression in a large sample of patients receiving intensive treatment for AN, and to evaluate whether weight suppression predicts weight gain and treatment response in this

group. We hypothesized that higher levels of weight suppression at admission for intensive treatment would predict greater and more rapid weight gains, even after controlling for relevant covariates. We also hypothesized that higher levels of weight suppression would be associated with a poor response to intensive treatment. Specifically, based on previous studies that have documented that more rapid weight gain predicts a decreased likelihood of achieving target weight in AN patients (T. Herzog et al., 2004) and higher levels of weight suppression predict the persistence of bulimic symptoms (Butryn et al., 2006; Carter et al., 2008) and higher rates of treatment dropout (Butryn et al., 2006) in individuals with BN, we hypothesized that higher weight suppression would predict a decreased likelihood of achieving a minimally adequate body weight (defined as BMI ≥ 18.5 for patients aged ≥ 20 years or BMI percentile ≥ 10 for patients aged 16–19 years; Hebebrand, Wehmeier, & Remschmidt, 2000), an increased likelihood of binge eating or purging (i.e., self-induced vomiting, laxative misuse, diuretic misuse) during the last four weeks of treatment, and a greater risk of treatment dropout in patients with AN.

Method

Participants

Participants were 185 patients aged ≥ 16 years enrolled in an ongoing study designed to evaluate the clinical utility of comorbidity subtypes in patients with AN. The study inclusion and exclusion criteria have been described previously (Wildes et al., 2011). Briefly, participants were recruited from 256 consecutive, unique admissions to the inpatient and day hospital eating disorder programs at an academic medical center between April 2008 and April 2011. After the study was described, 194 patients (75.78%) signed consent forms approved by the local Institutional Review Board (assent for individuals under 18 years) and completed baseline study assessments. Nine participants (4.63%) were excluded from the current report because they did not provide information about weight history. There were no differences between included (n = 185) and excluded (n = 9) participants with respect to AN-R versus AN-B/P diagnosis, BMI at admission for intensive treatment, type of intensive treatment (inpatient, day hospital, or inpatient + day hospital), weight gain, rate of weight gain, likelihood of achieving a minimally adequate body weight, the presence of bulimic symptoms at discharge, or treatment dropout (p's > .08). Excluded participants were younger, M (SD) = 17.11 (1.90) versus M (SD) = 26.96 (10.16) years old, $t(192) = 2.90$, $p < .01$, and more likely to be male, 44.44% versus 2.70%, X^2 (1) = 33.80, $p < .001$, than included participants. Demographic and clinical characteristics of the final sample (N = 185) are presented in Table 1.

Treatment

The inpatient and day hospital programs at our center have been have been described previously (Treat et al., 2008; Wildes et al., 2011). The milieus are informed by cognitive behavioral (CBT) and dialectical behavioral therapy (DBT) principles, and the primary treatment modalities are group therapy and meal support. However, the inpatient setting is more structured, providing 24-hour care, while the day hospital program operates 32 hours per week. Although many patients step-down to the day hospital program following inpatient treatment, there are instances in which low-weight patients are managed exclusively with day hospital care, or when step-up from day hospital to inpatient treatment is required (Attia & Walsh, 2009). Conversely, some individuals are unable or unwilling to attend a day hospital program and thus are treated only on the inpatient unit. Information about the types of intensive treatment received by participants in the current study is reported in Table 1.

Because the primary goals of intensive treatment for AN are to normalize eating behaviors and facilitate weight gain (Attia & Walsh, 2009), nutrition rehabilitation is a central component of the inpatient and day hospital programs. Consistent with established practice guidelines (American Psychiatric Association, 2006; NICE, 2004), patients are expected to gain approximately 0.5–1.0 kg (roughly 1.0–3.0 lb) per week. Caloric intake typically begins at 30–40 kcal/kg per day (approximately 1,000–1,600 kcal/day), and is increased by 200–300 kcal every 2–4 days, to a maximum of 70–100 kcal/kg. Patients are encouraged to gain weight by eating regular foods during supervised meals, and are expected to consume 100% of their prescribed calories. If a patient is unable or unwilling to consume her/his prescribed calories, a liquid nutrition supplement is provided, and cognitive-behavioral techniques are employed to help the patient identify triggers for caloric restriction. In rare instances, nasogastric feeding is offered on the inpatient unit to initiate weight gain; however, this is a temporary intervention, as the goal of nutrition rehabilitation is for patients to meet caloric requirements through oral ingestion of fluids, solid foods, and liquid supplements. Finally, physical activity is monitored closely; patients engage in sedentary activities (e.g., therapy groups) after meals, and energy expenditure is limited to light stretching or walking.

Assessments

The data included in this manuscript were collected from research assessments completed within approximately two weeks of admission for intensive treatment (*M* [*SD*] = 12.87 [10.02] days after admission) and at discharge, and medical chart reviews. As reported previously (Wildes et al., 2011), baseline assessments were delayed for two weeks after admission to mitigate the effects of acute starvation on cognitive functioning. Assessments were conducted by research staff independent of the treatment team. Interviews were audiotaped, and 10% were rerated by independent clinicians to establish reliability.

Weight and course of treatment—Participants' medical records were reviewed to obtain information about: 1) height and weight on the day of admission and at discharge, 2) days in intensive treatment, 3) type of intensive treatment, 4) number of prescribed calories, and 5) discharge type (planned or against medical advice [equivalent to treatment dropout]). As part of the standard treatment protocol, participants were weighed on a digital scale in a hospital gown without shoes. Height was measured using a stationary stature board. BMI at admission and discharge was calculated as weight in kg divided by height in meters squared. Weight gain was calculated as *discharge weight – admission weight*, and rate of weight gain (kg/wk) was calculated as *(discharge weight – admission weight)/(days in intensive treatment/7)*.

Weight suppression—Participants completed an investigator-designed questionnaire at baseline to document demographics, previous treatment, and weight history. Participants were queried about their highest and lowest non-pregnancy weights between the ages of 12 and 18 years and over 18 years. Self-reported heights and ages corresponding to each weight also were recorded. The validity of recalled past weights has been supported in previous research (Tamakoshi et al., 2003), and this approach is standard in the weight suppression literature (see, e.g., Carter, et al., 2008; Keel & Heatherton, 2010; Lowe, et al., 2006). Consistent with previous work (D. B. Herzog et al., 2010), weight suppression was defined as *highest weight at current height – weight at admission to intensive treatment.*

Weight history—Because studies of individuals with BN have indicated that weight suppression is superior to other measures of weight history in predicting the course of eating disorders (Butryn et al., 2006; Carter et al., 2008), we calculated several additional weight variables to determine whether this association would hold for individuals with AN, as well. First, we calculated indices of weight fluctuation that have been used in previous reports

including participants' highest and lowest self-reported BMIs at their current height (Butryn et al., 2011), and the difference between lowest weight (at current height) and current weight (Butryn et al., 2011; Butryn et al., 2006; Carter et al., 2008). We did not consider the difference between highest and lowest weight at current height, although this has been used in previous studies (Butryn et al., 2011; Carter et al., 2008), because highest weight at current height minus lowest weight at current height was virtually equivalent to weight suppression in the present sample, $r(183) = 0.94$, $p < .001$. Finally, we determined whether participants had a history of overweight or obesity based on BMI (for weights that occurred when participants were ≥ 20 years old) or BMI ≥ 85th percentile for age and sex (weights that occurred between the ages of 12 and 19).

Eating disorder symptoms and diagnoses—Participants were interviewed at baseline using the Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2007) and the Eating Disorder Examination, 16th edition (EDE; Fairburn, Cooper, & O'Connor, 2008). Diagnoses of AN were made using *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition* (*DSM-IV*) criteria with two exceptions: 1) amenorrhea was not required because this criterion has been shown to have limited validity (Attia & Roberto, 2009); and 2) 16 patients who entered intensive treatment with a BMI < 17.5, but denied fear of fatness were included, consistent with descriptions of "non-fat-phobic AN" (Becker, Thomas, & Pike, 2009). Restricting (AN-R) versus binge-eating/purging (AN-B/P) subtypes were assigned using data collected from the EDE. Consistent with recommendations of the Eating Disorders Work Group for DSM-5 (Peat et al., 2009), participants were diagnosed with AN-B/P if they reported ≥ 1 episode of binge eating or purging (i.e., self-induced vomiting, laxative misuse, diuretic misuse) per month during the three months prior to treatment. The psychometric properties of the SCID-I and EDE are well-documented (Berg, Peterson, Frazier, & Crow, 2011; First, Gibbon, Spitzer, & Williams, 2002). Inter-rater reliabilities for number of binge eating episodes, number of purging episodes, and AN-R versus AN-B/P diagnosis were $r = 0.97$, $r = 0.99$, and $\kappa = 1.00$, respectively.

To evaluate changes in eating disorder symptoms during intensive treatment, participants were re-interviewed at discharge using a modified version of the EDE designed to cover the period of time since the baseline assessment.1 Psychometric properties of the discharge EDE were consistent with those of the original measure. Internal consistencies of the subscales ranged from $\alpha = 0.61$ to $\alpha = 0.92$, and inter-rater reliabilities for number of binge eating episodes and number of purging episodes were $r = 1.00$ and $r = 1.00$, respectively.

Statistical Analysis

Descriptive statistics were performed to characterize weight suppression and weight history in patients with AN, and data were screened for skewness and outliers. The distribution for weight suppression was positively skewed and had one extreme outlier (value > 5 standard deviations above the mean) due to a patient with a past history of bariatric surgery; thus, it was square-root transformed prior to use in parametric analyses (Howell, 2010).2 Two extreme positive outliers also were identified in the distribution for rate of weight gain due to patients having dropped out of treatment after five days and eight days, respectively. Square-root transformation was unsuccessful in correcting the distribution, and scatterplots indicated that the outliers obscured associations between rate of weight gain and other study variables. We considered running the analyses without these participants, but elected instead

[1]Participants who discharged less than one week after the baseline assessment ($n = 28$) were not re-interviewed.
[2]Results were the same when the participant with a history of bariatric surgery was excluded from the analyses (data available upon request).

to replace the extreme scores with the next highest value in the distribution to maximize data integrity and interpretation (Howell, 2010).3

Associations between weight suppression and baseline demographic and treatment characteristics were examined using Pearson correlations and independent samples t-tests. Bivariate relations of weight suppression and weight history to weight gain and rate of weight gain also were examined using correlations. To evaluate the clinical significance of the correlations, we divided the sample into *high weight suppressors* and *low weight suppressors* using median split (*Mdn* = 14.65 kg), and calculated descriptive statistics for weight gain and rate of weight gain in each group. Finally, hierarchical multiple regression analyses were performed to test whether weight suppression at admission for intensive treatment predicted weight gain and rate of weight gain, controlling for significant covariates.

To evaluate the relation of weight suppression to indices of treatment response in patients with AN, we conducted logistic regression analyses predicting minimally adequate body weight at discharge and the presence of at least one episode of binge eating or purging during the last four weeks of treatment.4 Cox proportional hazards regression analysis was used to predict risk of discharge against medical advice (data were censored at the point at which participants terminated intensive treatment). If weight suppression significantly predicted an outcome in the univariate analyses, hierarchical regression was performed to evaluate the contribution of weight suppression relative to other significant predictors. Analyses were conducted using IBM SPSS Statistics 19, and two-tailed alpha was set at .05.

Results

Weight Suppression in Patients with AN

Descriptive statistics for weight suppression and weight history in the sample are presented in Table 2. There were no significant associations between weight suppression at admission for intensive treatment and sex, race, admission BMI, AN-R versus AN-B/P subtype, severity of binge eating or purging prior to treatment (AN-B/P only), duration of AN symptoms, or repeated (≥ 2) hospitalizations for eating disorder (*p*'s > .05). Not surprisingly, weight suppression was higher in individuals with a history of overweight, $t(183) = 10.42$, $p < .001$, or obesity, $t(183) = 7.48$, $p < .001$, than in the rest of the sample. Weight suppression also was positively correlated with age, $r(183) = 0.30$, $p < .001$, and age at onset of eating disorder symptoms, $r(183) = 0.25$, $p = .001$, in AN patients.

Weight Suppression as a Predictor of Weight Gain during Intensive Treatment for AN

Prior to examining whether weight suppression predicted weight gain in patients with AN, we conducted a series of preliminary analyses to determine whether weight suppression was related to the degree or quality of intensive treatment that participants received. There were no associations between weight suppression and length of intensive treatment episode, type of intensive treatment received, maximum number of calories prescribed, or number of calories prescribed at admission or discharge (*p*'s > .45). Finally, we conducted analyses to ensure that associations between weight suppression and the study outcomes did not differ as a function of AN-R versus AN-B/P subtype or the type of intensive treatment patients received. The pattern of results was the same across each group (data available upon request).

[3]Results were the same when the participants were excluded from the analyses (data available upon request).
[4]For patients who completed fewer than four weeks of intensive treatment (*n* = 59), persistence of binge eating or purging at discharge was defined as any binge eating or purging during the treatment episode.

Next, we conducted univariate analyses to determine whether weight suppression and other weight history variables predicted weight gain and rate of weight gain during intensive treatment for AN. As expected, higher weight suppression at admission to intensive treatment was associated with greater overall weight gain, $r(183) = 0.19$, $p = .009$, and a faster rate of weight gain, $r(183) = 0.20$, $p = .007$, in AN patients. High weight suppressors gained an average of 7.49 (*SD* = 4.47) kg compared to 4.97 (*SD* = 3.31) kg in low weight suppressors. Rate of weight gain in the high weight suppressors averaged 1.26 (*SD* = 0.63) kg/wk compared to 0.99 (*SD* = 0.89) kg/wk in the low weight suppressors.5

There were no significant associations between weight gain and highest BMI at current height ($p = .11$) or history of overweight or obesity (p's > .50). Similarly, there were no significant associations between rate of weight gain and highest BMI at current height ($p = .30$), history of overweight or obesity (p's > .10), or the difference between lowest weight and current weight ($p > .50$). Lowest BMI at current height and the difference between lowest weight and current weight were inversely related to weight gain, $r(183) = -0.40$, $p < .001$ and $r(183) = -0.16$, $p = .03$, respectively. Lowest BMI at current height also was inversely related to rate of weight gain, $r(183) = -0.22$, $p = .003$. There were no significant associations between weight suppression and lowest BMI at current height ($p > .50$) or the difference between lowest weight and current weight ($p > .10$), indicating that these variables measured distinct constructs in the present sample.

Finally, we conducted hierarchical regression analyses to evaluate whether higher levels of weight suppression at admission to intensive treatment predicted a greater degree of weight gain and faster rate of weight gain in AN patients, controlling for significant covariates.6 As documented in Table 3, higher levels of weight suppression continued to explain a small, but significant, amount of variance in weight gain and rate of weight gain during intensive treatment for AN in the multivariate models.

Weight Suppression as a Predictor of Response to Intensive Treatment for AN

One hundred fifteen patients (62.16%) completed the intensive treatment program, and 70 (37.83%) discharged against medical advice (dropped out). At discharge, 44.32% ($n = 82$) of participants had achieved a minimally adequate body weight (i.e., BMI ≥ 18.5 or BMI percentile ≥ 10), and 55.68% (n = 103) remained underweight; the average BMI was 17.98 (*SD* = 1.56). The majority of patients were abstinent from bulimic symptoms at discharge, but 34 (18.38%) reported at least one episode of binge eating ($n = 1$), purging ($n = 29$), or both ($n = 4$) during the last four weeks of treatment.7 Notably, three of the participants who endorsed binge eating or purging at discharge met criteria for AN-R at admission (two reported no history of binge eating or purging, and one had a lifetime history of purging, but denied engaging in compensatory behaviors during the past three months).

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[5]Because recommendations for rate of weight gain differ for inpatient and outpatient settings [e.g., the NICE guidelines (2004) recommend an average of 0.5–1.0 kg/wk during inpatient treatment and 0.5 kg/wk in outpatient care], we calculated descriptive statistics for rate of weight gain in patients that received inpatient treatment only or day hospital treatment only. Among individuals who received inpatient treatment only, the average rate of weight gain was 1.47 (*SD* = 0.59) kg/wk in the high weight suppressors ($n = 61$) and 1.20 (*SD* = 0.76) kg/wk in the low weight suppressors ($n = 55$). Similarly, among individuals who received day hospital treatment only, the average rate of weight gain was 0.61 (*SD* = 0.28) kg/wk in the high weight suppressors ($n = 7$) versus 0.38 (*SD* = 0.27) kg/wk in the low weight suppressors ($n = 13$).

[6]Non-significant univariate predictors of weight gain included age, race, duration of illness, age at onset of eating disorder symptoms, and history of repeated hospitalizations for eating disorder (p's > .10). Non-significant univariate predictors of rate of weight gain included age ($p = .07$), sex ($p > .10$), race ($p > .10$), AN subtype ($p > .10$), duration of illness ($p = .06$), and age at onset of eating disorder symptoms ($p > .10$).

[7]Of participants who reported ≥ 1 episode of binge eating or purging during the last four weeks of treatment, 12 (35.29%) were treated on the inpatient unit only, 6 (17.65%) were treated in the day hospital program only, and 16 (47.06%) were treated in both the inpatient and day hospital programs.

Contrary to the study hypotheses, weight suppression at admission for intensive treatment was unrelated to achievement of a minimally adequate body weight both in univariate analyses and after controlling for potential confounders including age, BMI at admission, days in intensive treatment, and type of intensive treatment received (*p*'s > .25). Weight suppression also was unrelated to treatment dropout ($p > .10$). However, higher levels of weight suppression did predict the presence of bulimic symptoms at discharge, $\beta = 0.32$, $SE = 0.15$, Wald $X^2(1) = 4.50$, $p = .03$, OR = 1.38, 95% CI [1.03, 1.86]. AN patients who endorsed binge eating or purging during the last four weeks of intensive treatment had a mean weight suppression of 20.12 (*SD* = 10.85) kg compared to 16.17 (*SD* = 9.69) kg in those who were abstinent from binge eating or purging. Highest BMI at current height also was positively associated with the persistence of bulimic symptoms in AN patients, $\beta = 0.11$, $SE = 0.04$, Wald $X^2(1) = 7.04$, $p = .008$, OR = 1.12, 95% CI [1.03, 1.21]. There were no significant associations between the other weight history variables and binge eating or purging (*p*'s > .06 for lowest BMI and history of overweight; *p*'s > .20 for the difference between lowest weight and admission weight and history of obesity).

Because univariate analyses indicated that weight suppression was positively associated with binge eating or purging at discharge from intensive treatment, we conducted hierarchical logistic regression to determine whether weight suppression predicted bulimic symptoms after controlling for significant covariates.8 As shown in Table 4, higher levels of weight suppression continued to predict binge eating or purging during the last four weeks of treatment in the multivariate model. The change in Cox and Snell R^2 from step 1 to step 2 was 0.025, indicating that weight suppression explained 2.5% of the variance in bulimic symptoms after controlling for BMI at admission, type of intensive treatment, and AN-R versus AN-B/P subtype.

Discussion

This study provides novel evidence that the predictive validity of weight suppression extends across the eating disorders spectrum, and is not limited to normal weight or overweight individuals with bulimic syndromes. In a large sample of AN patients, we found that higher levels of weight suppression at admission for intensive behavioral treatment predicted a faster rate and larger degree of weight gain, and the presence of binge eating or purging episodes during the four weeks prior to discharge. Although these effects were modest, they remained after controlling for powerful covariates including BMI at admission, AN-R versus AN-B/P subtype, length of intensive treatment episode, and type of intensive treatment received. Moreover, given the high costs of AN in terms of treatment utilization (Striegel-Moore, Leslie, Petrill, Garvin, & Rosenheck, 2000), medical and psychiatric morbidity (Kaplan & Garfinkel, 1993; O'Brien & Vincent, 2003), and mortality (Birmingham, Su, Hlynsky, Goldner, & Gao, 2005), identifying novel factors that may moderate the course or outcome of AN treatment is important even if the effects are small.

There were no associations between weight suppression and length or type of intensive treatment received or number of calories prescribed, suggesting that the current findings were not an artifact of treatment differences between high and low weight suppressors. Furthermore, weight suppression was unrelated to indices of AN severity including duration of AN symptoms, repeated hospitalizations for eating disorder, AN-B/P subtype, and BMI at admission, indicating that it was not a proxy for acuity of illness. Finally, other measures of weight history, such as highest and lowest BMI at current height, the difference between

[8]Non-significant univariate predictors of binge eating or purging at discharge included age, sex, race, duration of illness, age at onset of eating disorder symptoms, history of repeated hospitalizations for eating disorder, length of intensive treatment episode, discharge against medical advice, and number of days covered by the discharge assessment (*p*'s > .10).

current weight and lowest weight, and history of overweight or obesity failed to demonstrate consistent associations with the study outcomes, providing further evidence for the superiority of weight suppression relative to other indices of weight fluctuation in predicting the course of eating disorders.

The finding that higher levels of weight suppression were associated with a faster rate of weight gain during intensive behavioral treatment may have important implications for clinical practice. Although the magnitude of this effect was small after controlling for covariates, the average rate of weight gain in the high weight suppressors (1.26 kg/wk) exceeded the maximum rate of weekly weight gain recommended by NICE (2004) guidelines by 0.26 kg/wk (approximately 0.6 lb/wk). In contrast, the average rate of weight gain in the low weight suppressors (0.99 kg/wk) was within recommended limits. These patterns remained when we examined rates of weight gain in individuals that received inpatient or day hospital treatment only, indicating that higher levels of weight suppression at admission to AN treatment predict faster rates of weight gain in both inpatient and outpatient settings.

The association between rate of weight gain and treatment outcome in AN is complex, and there is evidence that faster rates of weight gain predict a decreased likelihood of achieving target weight, faster weight loss post-treatment, and an increased risk of re-hospitalization (T. Herzog et al., 2004; Lay et al., 2002; Willer, et al., 2005; but, see Lund, et al., 2009). Although the specific mechanisms responsible for these effects are unknown, a plausible explanation might be that rapid weight gain exacerbates or triggers the extreme fear of fatness that characterizes AN, leading to decreased levels of treatment engagement and an increased likelihood of engaging in disordered eating behaviors as a means of slowing or reversing the effects of weight restoration. This speculation is timely in light of the renewed interest in aggressive feeding strategies for patients with AN (see, e.g., Garber, Michihata, Hetnal, Shafer, & Moscicki, 2012). Although challenges to the so-called “start low, advance slow” approach to restoring weight in AN patients recommended by current treatment guidelines (see, e.g., Katzman, 2012) may be applicable to individuals low in weight suppression, the results of the present study suggest that current approaches are more than adequate for achieving weight gain in high weight suppressors. Furthermore, given the potential sequelae of rapid weight gain noted above, evaluation of weight suppression among individuals with AN could provide important information to guide decisions about calorie prescriptions and the aggressiveness of refeeding to prevent possible iatrogenic effects of treatment.

Finally, if AN patients with high levels of weight suppression have a greater propensity for weight gain, this raises the intriguing possibility that these individuals could achieve weight restoration in an outpatient setting. There is considerable interest in the application of a stepped-care model to the treatment of eating disorders (Wilson, Vitousek, & Loeb, 2000), and current guidelines emphasize the importance of referring patients to the least restrictive setting necessary for achieving treatment goals (American Psychiatric Association, 2006; NICE, 2004). However, for severely underweight individuals, it often is unclear when referral to an outpatient intervention is appropriate. With the exception of a specific form of family therapy for adolescents (Lock et al., 2010), support for the efficacy of outpatient psychotherapeutic or pharmacologic treatments for AN has been equivocal (for review, see Bulik, Berkman, Brownley, Sedway, & Lohr, 2007). Importantly, because outpatient interventions require individuals to normalize eating behaviors in an unstructured environment, it often is difficult to achieve weight restoration during outpatient care (Guarda, 2008). Nevertheless, there is preliminary evidence that certain forms of psychotherapy, including CBT and an AN-specific treatment termed Specialist Supportive Clinical Management (SSCM), may hold promise for helping some individuals to gain

weight and decrease eating disorder symptoms on an outpatient basis (Channon, de Silva, Hemsley, & Perkins, 1989; McIntosh et al., 2005). Given that weight suppression was positively correlated with rate and degree of weight gain during intensive treatment for AN in the current study, it is possible that AN patients with high levels of weight suppression would be more likely than low weight suppressors to gain weight and achieve weight restoration in an outpatient setting. The preliminary comparison of rates of weight gain during day hospital treatment in the high and low weight suppressors supports this contention. However, future research is needed to replicate these findings, and to examine whether weight suppression predicts weight gain during less structured outpatient treatments for AN (e.g., individual therapy), before definitive recommendations can be made about the utility of weight suppression for guiding the selection of treatment.

The current data were mixed with respect to the utility of weight suppression as a predictor of initial response to intensive behavioral treatment for AN. Contrary to the study hypotheses, weight suppression was unrelated to achievement of a minimally adequate body weight or treatment dropout. On first glance, the failure of weight suppression to predict achievement of a minimally adequate body weight despite its positive association with rate and degree of weight gain is difficult to reconcile. However, at least one study has shown that faster rates of weight gain are associated with a decreased likelihood of achieving target weight (T. Herzog et al., 2004), suggesting that the relation between rate of weight gain and discharge BMI in AN may not be linear. Moreover, external constraints on length of intensive treatment might have obscured associations among weight suppression, rate of weight gain, and discharge BMI in the current sample (e.g., some health care coverage providers require referral to individual treatment for all patients at or above 80% of ideal body weight). Finally, although one report found that weight suppression predicted dropout from CBT for BN (Butryn et al., 2006), subsequent studies have been unable to replicate this finding (Carter et al., 2008; Zunker et al., 2011).

Higher levels of weight suppression did predict the presence of binge eating or purging during the four weeks prior to discharge, however. Binge eating and purging are important clinical outcomes in AN research because these behaviors have been shown to predict severity and duration of illness (Peat et al., 2009; Steinhausen 2002), and are present in the majority of individuals with AN (Eddy et al., 2008). Moreover, it is noteworthy that three of the 34 patients who endorsed bulimic symptoms at discharge met criteria for AN-R at admission, suggesting the possibility that weight suppression might be associated with diagnostic crossover in individuals with AN. Future studies are needed to determine whether weight suppression and persistence of bulimic symptoms predict higher rates of binge eating and purging after discharge from intensive behavioral treatment for AN. In addition, the current findings suggest that AN patients with high levels of weight suppression may require closer monitoring during weight restoration treatment to prevent the onset or resumption of bulimic behaviors.

The present study has several strengths including a focus on testing novel hypotheses about the relation of weight suppression to weight gain and treatment response in individuals with AN using a large sample and a prospective design. Nevertheless, certain limitations must be considered when interpreting the findings. First, participants were patients receiving intensive treatment for AN at an eating disorders specialty clinic in an academic medical center. Thus, findings may not generalize to other treatment settings. Second, the nine individuals who were excluded from the current report because they did not provide information about weight history were younger and more likely to be male than included participants. Consequently, results should be generalized with caution to adolescents and males with AN. Third, consistent with all previous studies of weight suppression, we relied on participant report of weight history rather than an objective assessment because it was not

feasible to collect objective weight information (either directly or by medical chart review) at the time when participants reached their highest and lowest weights. Although research has supported the validity of recalled past weights in healthy populations (Tamakoshi et al., 2003), this procedure may be vulnerable to assessment error, especially in individuals with eating disorders (Meyer, McPartlan, Sines, & Waller, 2009). However, as noted by Keel and Heatherton (2010), assessment error likely would have decreased our ability to detect significant associations between variables or produced spurious findings, neither of which was a problem in the current report. Fourth, although data regarding weight gain, rate of weight gain, and treatment dropout were collected from medical charts, persistence of bulimic symptoms was based on participant self-report using a standardized interview and thus may be vulnerable to recall bias. However, there is no theoretical reason that AN patients high in weight suppression would be more likely than AN patients low in weight suppression to endorse binge eating or purging during intensive treatment, particularly because there was no relation between weight suppression and illness severity at baseline. Finally, the reported effects generally were small, and the possibility of Type I error resulting from multiple comparisons cannot be ruled out definitively. Although we think this risk is mitigated by the fact that the data analyses were hypothesis-driven and the results were consistent with previous studies examining the association of weight suppression to weight gain and bulimic symptoms during the treatment of eating disorders (Butryn et al., 2006; Carter et al., 2008; Lowe et al., 2006), future research is needed to replicate the present findings and examine whether effects observed during intensive treatment influence the longer-term course of AN.

In closing, this study contributes to a growing body of research documenting that weight suppression predicts weight gain and bulimic symptoms during treatment for eating disorders (Butryn et al., 2006; Carter et al., 2008; Lowe et al., 2006). Although available data regarding the clinical significance of weight suppression are mixed (Butryn et al., 2006; Carter et al., 2008; Zunker et al., 2011), the current findings indicate that additional research to elucidate the relation between weight suppression and treatment response in eating disorders is warranted. Furthermore, the results of the present study suggest that future research should focus on individuals with AN, as well as those with bulimia spectrum disorders.

Acknowledgments

This research was supported by K01 MH080020 from the National Institute of Mental Health. We would like to thank Courtney Alesandro, Ashley Bright, Juliana Bandi, Jill Gaskill, Joanna Gould, Katherine Heron, and Rebecca Ringham for their contributions to data collection.

The funding source had no involvement in the study design; the collection, analysis or interpretation of study data; the writing of the report; or, the decision to submit the article for publication.

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Table 1

Demographic and Clinical Characteristics of the Sample (N = 185)

Characteristic	*M*	*SD*	*n*	%
Age (years)	26.96	10.16		
Female			180	97.30
Non-white race			8	4.32
BMI at admission for intensive treatment	15.66	1.83		
Weight at admission for intensive treatment (kg)	42.03	6.16		
Anorexia nervosa subtype				
Restricting			80	43.24
Binge-eating/purging			105	56.76
Duration of illness (years)	8.88	9.08		
≥ 2 hospitalizations for eating disorder			89	48.11
Days in intensive treatment	44.31	24.66		
Type of intensive treatment				
Inpatient			116	62.70
Day hospital			20	10.81
Inpatient + day hospital			49	26.49
Maximum number of calories prescribed	3039.25	604.22		
Weight gain during intensive treatment (kg)	6.24	4.12		
Rate of weight gain during intensive treatment (kg/wk)	1.12	0.78		

Note. BMI = body mass index

Table 2

Weight Suppression and Weight History in Patients with Anorexia Nervosa (N = 185)

Variable	*M*	*SD*	*Min*	*Max*	*n*	%
Highest weight at current height (kg)	59.08	12.44	32.66	122.02		
Lowest weight at current height (kg)	38.88	6.79	20.41	57.15		
Weight suppression (kg)	17.05	10.78	0	78.24		
Current weight minus lowest weight (kg)	3.10	3.49	0	16.78		
Highest BMI at current height	22.00	4.16	14.08	43.41		
Lowest BMI at current height	14.47	2.11	8.52	18.19		
History of overweight					43	23.24
History of obesity					17	9.19

Note. BMI = body mass index

Table 3

Hierarchical Multiple Regression Analyses Predicting Weight Gain and Rate of Weight during Intensive Treatment for Anorexia Nervosa

	Weight Gain		
Predictor	**ΔR^2**	**Effect size (f^2)**	**β**
Step 1	.564***	1.29	
BMI at admission			−.35***
Days in intensive treatment			.53***
Inpatient vs. day hospital			−.23**
Inpatient vs. inpatient + day hospital			−.05
Sex			−.13*
AN subtype			−.03
Step 2	.010*	.02	
Weight suppression			.10*
Total R^2	.574***		
	Rate of Weight Gain		
Predictor	ΔR^2	Effect size (f^2)	β
Step 1	.229***	.30	
BMI at admission			−.04
Inpatient vs. day hospital			.38***
Inpatient vs. inpatient + day hospital			.09
Repeated hospitalizations for ED			.03
Step 2	.025*	.03	
Weight suppression			.16*
Total R^2	.254***		

Note. BMI = body mass index; AN = anorexia nervosa; ED = eating disorder. Per 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

Hierarchical Logistic Regression Predicting Bulimic Symptoms at Discharge from Intensive Treatment for Anorexia Nervosa

	≥ 1 episode of binge eating or purging during the last 4 weeks of intensive treatment				
Predictor	**β**	**SE (β)**	**Wald $X^2(1)$**	**OR**	**95% CI**
Step 1					
BMI at admission	0.06	0.15	0.16	0.69	0.79–1.42
Inpatient vs. day hospital	1.90	0.74	6.48*	6.65	1.55–28.62
Inpatient vs. inpatient + day hospital	2.06	0.53	15.24***	7.88	2.80–22.22
AN subtype	2.98	0.68	19.51***	19.75	5.26–74.21
Step 2					
Weight suppression	0.43	0.18	5.68*	1.54	1.08–2.21

Note. BMI = body mass index; AN = anorexia nervosa

* $p < .05$.

*** $p < .001$.