

The Relation of Weight Suppression and Body Mass Index to Symptomatology and Treatment Response in Anorexia Nervosa

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Weight suppression, the difference between highest past weight and current weight, is a robust predictor of clinical characteristics of bulimia nervosa; however, the influence of weight suppression in anorexia nervosa (AN) has been little studied, and to our knowledge, no study to date has investigated the ways in which the relevance of weight suppression in AN may depend upon an individual's current body mass index (BMI). The present study investigated weight suppression, BMI, and their interaction as cross-sectional and prospective predictors of psychological symptoms and weight in AN. Women with AN completed depression (Beck Depression Inventory-II) and eating disorder symptomatology measures (Eating Disorder Examination Questionnaire and Eating Disorders Inventory-3) at residential treatment admission ($N = 350$) and discharge ($N = 238$). Weight suppression and BMI were weakly correlated ($r = -.22$). At admission, BMI was positively correlated with all symptom measures except Restraint and Depression scores. Weight suppression was also independently positively correlated with all measures except Weight Concern and Body Dissatisfaction subscale scores. In analyses examining discharge scores (including admission values as covariates), the admission weight suppression \times BMI interaction consistently predicted posttreatment psychopathology. Controlling for weight gain in treatment and age, higher admission weight suppression predicted lower discharge scores (less symptom endorsement) among those with lower BMIs; among those with higher BMIs, higher weight suppression predicted higher discharge scores. These results are the first to our knowledge to demonstrate that absolute and relative weight status are joint indicators of AN severity and prognosis. These findings may have major implications for conceptualization and treatment of AN.

Keywords: anorexia nervosa, weight suppression, treatment, outcome

Research suggests that weight suppression, defined as the difference between highest weight since reaching adult height and current body weight, has high cross-sectional and predictive relevance in bulimia nervosa (BN). The construct has been found to be positively correlated with frequency of objective bulimic episodes among individuals who meet criteria for a diagnosis of BN (Lowe, Thomas, Safer, & Butryn, 2007) and predicted, over a period of 8-year follow-up, longer time to first full remission from BN

(Lowe et al., 2011). Findings among individuals with subthreshold BN were similar: In a large sample of collegiate men and women, individuals with bulimic syndromes had higher weight suppression levels than those without bulimic syndromes, and greater weight suppression predicted increased likelihood of bulimic syndrome maintenance at 10-year follow-up (Keel & Heatherton, 2010). Butryn, Lowe, Safer, and Agras (2006) found that greater weight suppression predicted worse outcomes in the cognitive-behavioral treatment of BN, though two studies did not replicate this finding (Carter, McIntosh, Joyce, & Bulik, 2008; Zunker et al., 2010).

In addition to robust prediction of bulimic symptomatology, weight suppression has also been found to predict weight gain. Among women with BN, results indicate that weight suppression, independent of body mass index (BMI), predicts weight gain both in the short term (Lowe, Davis, Lucks, Annunziato, & Butryn, 2006) and long term (over 5-year follow-up; Herzog et al., 2010). Of note, lower admission BMI was also found to predict short-term weight gain in treatment independently of weight suppression level (Lowe, Davis, et al., 2006).

Among individuals without eating disorders, evidence from experimenter-induced weight loss studies, which do not result in objectively low post-weight loss BMI levels, suggests that an individual's highest past weight may serve as a biological marker to which the body may be metabolically driven to return (e.g., MacLean, Bergouignan, Cornier, & Jackman, 2011; Rosenbaum & Leibel, 2010). In more naturalistic studies of women without

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eating disorders, weight suppression has been found to predict weight gain (Lowe, Annunziato, et al., 2006), even with the inclusion of resting metabolic rate and total energy expenditure as covariates (Stice, Durant, Burger, & Schoeller, 2011).

While much is known about the implications of weight suppression for BN and noneating disordered populations, this construct is only beginning to be investigated among individuals with anorexia nervosa (AN). A large body of evidence supports the clinical importance of objectively low weight in AN, often defined as a BMI under 18.5 mg/kg² (National Institutes of Health, & National Heart, Lung, & Blood Institute, 1998), including a broad array of resulting physiological and metabolic abnormalities (Klein & Walsh, 2004; Leonard & Mehler, 2001; Monteleone, DiLieta, Castaldo, & Maj, 2004) and associated problematic psychological symptoms, including preoccupation with food and eating (e.g., Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950). Patients with AN are all, by definition, low in objective weight; however, variability in highest past weights among individuals with AN (Coners, Remschmidt, & Hebebrand, 1999; Miyasaka et al., 2003) indicates a wide range of weight suppression levels that may also impact the biology and psychology of AN.

Despite a historical focus on the biological and psychological impact of objectively low BMI or percent of ideal body weight in AN, updates to diagnostic criteria reference consideration of both extent of weight loss and the current BMI of an individual with AN. Diagnostic criteria for AN, according to both *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR; American Psychiatric Association, 2000)* and fifth edition (*DSM-5*) criteria (American Psychiatric Association, 2013), require an individual to restrict energy intake and reach a “significantly low body weight” below what is minimally “normal” for adults. In *DSM-5*, BMI also determines an individual’s current AN illness severity rating; however, the rationale for *DSM-5* criteria development stated that the use of BMI alone to define a significantly low body weight “may not adequately reflect an individual’s . . . history,” and that the determination of whether weight is inappropriately low “is best made by the clinician in light of all relevant information” (American Psychiatric Association, 2012). Furthermore, *DSM-5* includes an “Other Specified Feeding or Eating Disorder” diagnosis of “atypical anorexia nervosa,” which includes individuals who have lost a significant amount of weight (i.e., who are high in weight suppression), but have not reached a weight that is below the objectively “normal range.” Thus, the field seems to be moving toward consideration of both objectively low weight and the degree of weight loss, including medical and psychological consequences of the degree of weight loss in AN (e.g., Watson & Andersen, 2003). Further research regarding the independent and joint influences of a patient’s weight loss and current weight status is needed to better determine how researchers and clinicians may best understand and incorporate this information in diagnosis, prevention, and treatment.

Preliminary evidence supports the biological and psychological significance of the construct of weight suppression for individuals with AN. For example, weight-restored (to at least 90% of ideal body weight, based on height) female patients with AN who were higher in weight suppression at the time of weight restoration had lower serum leptin levels and were less likely to have resumed menstruation before the time of hospital discharge (Klein et al., 2011). Thus, weight suppression, beyond absolute weight status,

may serve as an additional indicator of illness severity. Studies have also found that patients with the binge-eating/purging subtype of AN (AN-BP) have histories of higher maximum lifetime weight compared with patients with the restricting subtype (AN-R) and are more likely than patients with AN-R to report pre-morbid obesity (Garfinkel, Moldofsky, & Garner, 1980). Although individuals with AN-BP and those with AN-R are at a low body weight, the distance between highest lifetime weight and current weight (i.e., weight suppression) in individuals with AN-BP may be significantly greater. These findings suggest that weight suppression may be especially relevant to understanding bulimic behaviors in the context of AN. Furthermore, as distance from highest adult weight has been found to predict weight gain in both noneating disorder populations and in BN, it may also serve as a relevant predictor of symptomatology and weight gain in treatment in patients with AN. Most recently, in a large sample of patients with AN ($N = 185$), Wildes and Marcus (2012) found that while weight suppression was not related to achievement of a minimally adequate body weight, weight suppression, controlling for BMI, predicted total weight gain, a faster rate of weight gain, and likelihood of endorsing bulimic symptoms during intensive treatment.

Although research has begun to investigate the predictive power of weight suppression above and beyond BMI in AN, potential interactive effects of weight suppression and BMI have not been considered. Specifically, the extent to which the low BMI levels of patients with AN may moderate the relation between weight suppression and outcome has not yet been studied. Initial results in individuals with BN spectrum disorders support consideration of a joint influence of weight suppression and low BMI on eating disorder symptomatology in AN. BMI was found to moderate the relation between weight suppression and binge eating frequency in full and subthreshold BN, with individuals highest in weight suppression and lowest in objective weight status reporting the highest binge eating frequencies (Butryn, Juarascio, & Lowe, 2011). Similarly, in addition to previously reported main effects of weight suppression in AN, the same degree of weight suppression may have a more dramatic impact on an individual depending on the severity of the resulting low BMI level, particularly because such weight loss may activate varying degrees of physiological starvation responses.

A conceptual representation of the relation between weight suppression and current and highest adult BMI is shown in Figure 1. For example, a 30-lb weight loss in an individual whose highest adult BMI was 24 kg/m² (Figure 1, line C) may not have the same psychological and biological implications as a 30-lb weight loss in an individual whose highest adult BMI was 22 kg/m² (Figure 1, line A). Furthermore, weight loss to the same objectively low weight may have a more dramatic impact on individuals depending on degree of weight suppression. For example, weight loss to a BMI of 18 kg/m² may have different implications for an individual whose highest adult BMI was 24 kg/m² (Figure 1, line C) compared with an individual whose highest adult BMI was 19 kg/m² (Figure 1, line D). As with BN, weight suppression among individuals with AN may have implications independent of or in interaction with current BMI for symptomatology, diagnosis, and outcome.

The results reported by Wildes and Marcus (2012) mark an important first step in the investigation of weight suppression in

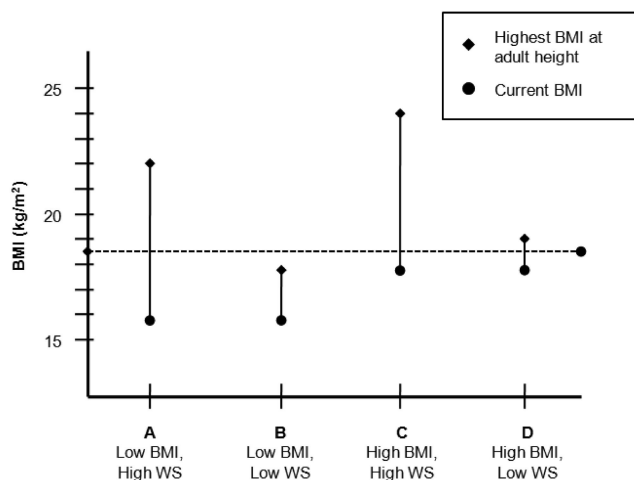


Figure 1. Conceptual representation of the relation between varying levels of weight suppression and current and highest adult body mass index (BMI; kg/m^2) among patients with anorexia nervosa. The vertical bars represent weight suppression (WS), the difference between highest BMI at adult height (diamonds) and current BMI (circles).

AN, and replication of these initial findings is needed. Furthermore, weight suppression has not yet been investigated as a baseline correlate of psychological symptoms in AN or as a predictor of changes in psychological symptoms during treatment in this population.

The present study investigates, in a sample of women with AN admitted to residential treatment, whether level of weight suppression and BMI at admission, both independently and in interaction, (a) relate to admission symptomatology and (b) predict weight or symptom levels at discharge, controlling for baseline values. This examination includes individuals who were all receiving a fairly standardized residential inpatient treatment and were all required to gain weight. As a cross-sectional relation and a prognostic relation between variables may be different, the present study maintains consistency with previous literature examining the relevance of weight suppression in individuals with BN by using a combination of cross-sectional and longitudinal analyses. Previous studies indicating that weight suppression is a correlate of eating disorder symptoms and predictor of treatment outcome in eating disorder populations have included BMI as a covariate; the current investigation represents an extension of these findings by examining the possibility that the cross-sectional and predictive effects of weight suppression may depend upon BMI level. We therefore conceptualize our analyses as tests of BMI as a moderator of the effects of weight suppression.

Potential relations among weight suppression, BMI, and AN etiology are beyond the scope of our investigation; nonetheless, an improved understanding of illness course, potential maintenance factors, and predictors of treatment response seems a critical addition to the literature on this pernicious and treatment-resistant disorder. Cross-sectional analyses examining weight suppression and BMI in relation to psychological symptoms at admission of course prevent determination of causality. Body dissatisfaction or eating concern, for example, may drive behaviors that result in weight suppression, and/or brain changes associated with high

degrees of weight suppression to objectively low body weights may exacerbate preoccupation with weight and eating. Regardless of the direction of this relation, examination of cross-sectional associations may support consideration of both absolute and relative weight status in determining illness severity ratings.

Method

Participants

Study participants were patients admitted to one of two residential treatment facilities for eating disorders (one in Pennsylvania and one in Florida) between July 1, 2007, and December 31, 2008. Both treatment facilities are owned and operated by a large, national center for eating disorders. All individuals were eligible if they were admitted to one of these sites during the period of data collection and met *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994)* criteria for AN, except for amenorrhea, as assessed by one of four psychiatrists. Individuals who met all criteria for standard AN except that, despite significant weight loss, the individual's weight was within the normal range (above $18.5 \text{ kg}/\text{m}^2$) were excluded from the present sample to maintain diagnostic consistency ($n = 12$, 3.3% of sample). Of the 337 women who responded, 77.2% denied taking oral contraceptives at the time of residential inpatient admission. Menstrual status data were available for 237 of the 260 women with AN who denied taking oral contraceptives, and 79.3% of these women had missed at least three periods in the 4 months before admission.

Because reliability of the treatment facilities' psychiatrist diagnoses has not been established, self-report items from the Eating Disorder Examination Questionnaire (EDE-Q) served as a proxy measure of diagnostic reliability. Similar to procedures described in previous research (Wolk, Loeb, & Walsh, 2005), individuals were considered to meet *DSM-IV-TR* AN diagnostic criterion B if they endorsed EDE-Q item 10 (fear of gaining weight) and were considered to meet criterion C if they endorsed item 11 (felt fat), item 22 (overvaluation of weight), or item 23 (overvaluation of shape) with a rating of 4 or higher (indicating clinically significant presence of that symptom on 16 days or more of the past 28 days). Information regarding menstrual status (criterion D) and birth control usage were also obtained from the EDE-Q, and amenorrhea was considered present if individuals endorsed missing three or more periods in the past four months. Approximately 40.3% of the sample ($n = 141$) met all diagnostic criteria for AN, including amenorrhea, with an additional 21.7% ($n = 76$) who otherwise met full criteria but were taking a form of birth control, preventing assessment of amenorrhea. Of those not taking birth control, 10% met all diagnostic criteria except for amenorrhea ($n = 36$). Because research suggests that there is little difference between individuals who do and do not experience amenorrhea (e.g., Attia & Roberto, 2009; Dalle Grave, Calugi, & Marchesini, 2008; Roberto, Steinglass, Mayer, Attia, & Walsh, 2008), and this criterion was eliminated in *DSM-5*, individuals who met all criteria except amenorrhea were considered to have an EDE-Q-confirmed diagnosis of AN.

A total of 4.6% did not meet criterion B (fear of fat, $n = 16$), and 22.9% did not meet criterion C (disturbance in or undue influence of weight and shape, $n = 80$). Although these individuals did not

endorse significant weight and shape concerns on the self-report questionnaire, because all patients included in analyses had been admitted to a residential treatment center for eating disorders and were admitted with a BMI under 18.5 kg/m², it is likely that this 27.5% of the sample who did not meet these two criteria per EDE-Q responses were a clinically relevant group. Analyses were run both with and without the 27.5% of the sample who did not meet either criterion B or C, and both results have been reported below (“all patients” vs. “confirmed-diagnosis patients”).

Approximately 35% of patients admitted to these facilities in Pennsylvania and Florida receive a diagnosis of AN. The 350 patients who completed the admission assessment represented approximately 80% of all patients with AN admitted during the study period. Study participants were all female, because only females are admitted to these treatment facilities. Approximately two-thirds of the sample ($n = 238$) also completed a discharge assessment. Mean length of stay in the program was 37.2 days ($SD = 20.0$).

Procedure

The study was approved by the research committee that oversees both treatment facilities, and access to and analysis of the data was approved by the institutional review board at Drexel University. All participants provided informed consent prior to completing assessments. The measures administered were completed as part of the standard admission battery, and completion rates reflect the typical rates of completion at each of the two facilities. Admission questionnaires were administered via computer on the second day of treatment, and discharge questionnaires were completed in the same manner within the final 2 days of treatment.

The comprehensive treatment program at both facilities focused on normalization of eating patterns, weight gain, and elimination of compensatory behaviors. Patients received an intensive program of individual, group, and family therapy, provided by a multidisciplinary team consisting of psychiatrists, psychologists, nutritionists, and nurses. Each patient received a nutritionist-developed, individualized meal plan designed to promote weight gain of 2 to 3 pounds per week. The theoretical orientation at both facilities is eclectic and is largely based on psychodynamic and feminist-relational theories.

Measures

Eating disorder symptomatology. The EDE-Q (Fairburn & Beglin, 2008) is a 36-item self-report questionnaire that focuses on eating disorder symptoms over the past 28 days. It was adapted from the Eating Disorder Examination (Fairburn & Cooper, 1993), an investigator-based, semistructured interview. The EDE-Q contains Shape Concern, Weight Concern, Eating Concern, and Restraint subscales in addition to a Global scale score computed from the average of the four subscales. Number of missed menstrual periods in the past four months was also obtained from this questionnaire. Data from community and clinical populations indicate good concurrent validity of the EDE-Q for all features except binge eating (Fairburn & Beglin, 1994). Acceptable levels of internal consistency have been observed for the EDE-Q Global and subscale scores (Cronbach's α coefficients above .70; Peterson et al., 2007), and the level of internal consistency was found to be acceptable in our sample (Cronbach's $\alpha = .88$ for Shape Concern,

.82 for Weight Concern, .72 for Eating concern, .84 for Restraint, and .94 for Global subscale scores).

The Eating Disorders Inventory-3 (EDI-3; Garner, 2004) is a 96-item self-report inventory that measures eating disorder symptom severity and psychological dimensions associated with eating disorders. The EDI-3 is organized into 12 primary scales; however the current study included only the Drive for Thinness, Body Dissatisfaction, and Bulimia subscales. The inventory has adequate psychometric properties, and the test-retest reliability of these subscales among women diagnosed with eating disorders has been shown to be excellent (Clausen, Rosenvinge, Friborg, & Rokkedal, 2011). The level of internal consistency was found to be acceptable in our sample (Cronbach's $\alpha = .88$ for Drive for Thinness, .89 for Bulimia, and .91 for Body Dissatisfaction).

Depressive symptomatology. The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996), a 21-item, multiple choice self-report measure, was used to assess depressive symptomatology. At both treatment facilities, the item assessing suicidality was excluded. The BDI-II has adequate test-retest reliability, internal consistency (Cronbach's $\alpha = .93$ in our sample), and convergent validity (Sprinkle et al., 2002; Steer, Ball, Ranieri, & Beck, 1997).

Weight status. Weights were measured using a digital scale and height was measured by stadiometer. BMI at admission and discharge was calculated as weight in kilograms divided by height in meters squared. Highest lifetime nonpregnancy weight at adult height was obtained via self-report from the cover page of the EDI-3. Weight suppression was calculated by subtracting weight at admission in kg from this reported highest weight in kilograms. The validity of recalled past weights has been supported by previous research (Tamakoshi et al., 2003), including a study of adolescents with AN and BN reporting a correlation of .92 between highest measured premenstrual weight from school records and highest recalled premenstrual weight (there were no differences between diagnostic groups; Swenne, Belfrage, Thurffjell, & Engström, 2005). Use of recalled highest past weight in the calculation of weight suppression is standard (Butryn et al., 2006; Carter et al., 2008; Herzog et al., 2010; Keel & Heatherton, 2010; Lowe, Annunziato, et al., 2011; Lowe, Davis, et al., 2006; Lowe et al., 2007; Zunker et al., 2010). Rate of weight change from admission to discharge was calculated as (discharge weight in kilograms – admission weight in kg)/days in residential treatment.

Statistical Analysis

Hierarchical multiple regression analyses were used to test whether weight suppression, admission weight status (BMI), and their interaction would significantly predict symptomatology (BDI-II scores and EDE-Q and EDI-3 subscales) at admission and discharge. All variables included in computing interactions were centered. Cross-sectional analyses at baseline were examined by entering covariates in the first block followed by weight suppression and BMI in the second block and the interaction term in the final block. Prospective analyses at discharge were examined by entering covariates and admission scores for the dependent variable in the first block, followed by weight suppression and BMI in the second block, and the interaction term in the final block. This approach adjusts all discharge outcome variables for their baseline values, permitting examination of posttreatment symptom levels

regardless of admission illness severity. All variables within each block were entered simultaneously, and were not interpreted unless the block itself was significant (Cohen & Cohen, 1983).

Several variables (admission: weight suppression, EDE-Q Restraint, EDI-3 Drive for Thinness, EDI-3 Bulimia, age, duration of illness; discharge: EDI-3 Bulimia) did not satisfy criteria for a normal distribution (skewness and kurtosis not exceeding an absolute value of 1), therefore square root, log, and inverse transformations were computed for each variable and compared to identify which brought the distribution closest to normality. With the exception of age, for which a log transformation was used, all of these variables were transformed using a square root transformation. After transformation, all variables satisfied criteria for a normal distribution, and the transformed versions of these variables were used in all analyses reported below. All regression assumptions of linearity, normality, homoscedasticity of residuals, and nonmulticollinearity were met. Outliers were identified using studentized residuals, leverage, and Cook's distances, and between zero and four cases were removed from each analysis in the results reported below. The pattern of results was similar when all cases were included in the analyses.

Cross-sectional analyses examining the prediction of menstrual status excluded participants who reported taking oral contraceptives, 22.9% of the full sample. In addition, initial analyses indicated that BMI, weight suppression, and their interaction were predictive of weight change from admission to discharge; therefore, discharge analyses for other outcome variables were conducted including weight change as a covariate. In addition, preliminary analyses indicated that baseline weight suppression had a small to moderate correlation with age, $r(360) = .33, p < .001$, and duration of illness, $r(357) = .25, p < .001$. These variables were considered for inclusion as covariates. Age and duration of illness were themselves highly correlated, $r(347) = .81, p < .001$, and therefore, to avoid violation of the multicollinearity assumption,

could not be simultaneously entered into the regression. Results were rerun separately including each covariate. Results reported below include age as a covariate; however, results including duration of illness as a covariate were equivalent.

Results

Sample Characteristics

Demographic and clinical characteristics of the full sample and confirmed-diagnosis sample at admission and discharge are presented in Table 1. The majority of the full sample (91.4%) self-identified as Caucasian (3.1% Multiracial or Other, 2.0% Hispanic, 1.7% African American, and 1.4% Asian). The majority of patients (85.7%, $n = 300$) received at least one comorbid diagnosis from program psychiatrists, most commonly major depressive disorder (49.4%, $n = 173$), generalized anxiety disorder (15.7%, $n = 55$), depressive disorder not otherwise specified (14.9%, $n = 52$), and substance use disorders (13.4%, $n = 47$). Approximately half (55.7%) of study participants in the full sample reported at least one previous psychiatric hospitalization.

Two hundred thirty-eight participants (68.0%) completed measures at discharge. Completers and noncompleters were compared on all baseline demographic and outcome measures listed in Table 1. The only statistically significant difference between these groups was in length of stay, with completers staying an average of 40.7 days ($SD = 21.4$) compared with noncompleters' average stay of 29.6 days ($SD = 14.1$). Because of frequent, unanticipated cessation of insurance coverage and resulting early discharge from the treatment centers, the difference in length of stay was expected. As those who did not complete discharge assessments did not differ at admission in weight suppression, BMI, or other admission symptomatology levels from those who did, the completer group was considered to be representative of the full sample. Discharge

Table 1
Sample Characteristics at Admission and Discharge

	Admission: Full sample			Admission: Confirmed-diagnosis sample			Discharge: Full sample			Discharge: Confirmed-diagnosis sample		
	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>	<i>N</i>	<i>M</i>	<i>SD</i>
Weight suppression (lbs)	350	34.8	20.2	253	35.5	20.4						
BMI (kg/m ²)	350	15.7	1.5	253	15.8	1.5	350	18.1	1.3	253	18.2	1.3
Highest adult BMI (kg/m ²)	350	21.5	3.4	253	21.7	3.4						
Age (years)	350	23.9	10.0	253	23.8	9.6						
Duration of illness (years)	347	7.6	8.2	251	8.0	8.4						
EDE-Q subscale scores												
Restraint	349	4.3	1.6	253	4.9	1.1	238	1.4	1.2	171	1.6	1.3
Weight concern	349	4.0	1.6	253	4.0	1.1	238	3.0	1.6	171	3.5	1.5
Shape concern	349	4.5	1.5	253	5.2	0.8	238	3.8	1.6	171	4.3	1.4
Eating concern	349	3.5	1.4	253	4.8	1.0	238	1.9	1.2	171	2.2	1.1
Global	349	4.1	1.3	253	4.7	0.7	238	2.5	1.3	171	2.9	1.2
Periods missed in the last 4 months	285	3.0	1.5	136	3.2	1.3						
EDI-3 subscale scores												
Drive for thinness	348	20.3	7.4	251	23.5	4.1	237	14.8	7.6	171	16.6	7.1
Bulimia	348	6.6	7.2	251	7.4	7.3	237	2.5	3.4	171	2.7	3.3
Body dissatisfaction	348	26.3	10.4	251	30.3	8.4	236	23.5	9.9	170	26.2	9.0
Beck Depression Inventory-II score	343	31.9	13.3	247	35.7	11.6	237	16.2	12.0	171	18.2	12.3

Note. WS = weight suppression (kg); BMI = body mass index (kg/m²); EDE-Q = Eating Disorder Examination-Questionnaire; EDI-3 = Eating Disorders Inventory-3. Means and sample sizes presented are not transformed.

analyses include completers only, with the exception of analyses of BMI and rate of weight gain, which were available for all participants ($n = 350$) because weight was recorded in medical charts and thus did not depend upon patients completing discharge self-report measures.

Weight Suppression and BMI as Correlates of Symptomatology at Admission

Intercorrelations among all variables at admission are presented in Table 2. The correlation between baseline BMI and weight suppression was statistically significant but small, $r(350) = -.22$, $p < .001$, such that those who had lower BMIs were more highly weight suppressed. Statistical information for individual predictors presented in Tables 3 and 4 represent results from the final model after all predictors have been included. We compared these with coefficient results for predictor variables when including only Steps 1 and 2 of the model (prior to the addition of the interaction term). Differences between the models in the size of standardized beta coefficients and semipartial correlations were small in size, and the statistical significance of individual predictors did not change between the two models for any outcome variable.

In analyses including the full sample, weight suppression and BMI were independently and positively associated with severity of symptomatology on all baseline measures (Table 3). Controlling for age and weight suppression, BMI independently predicted all symptom measures except EDE-Q Restraint subscale and BDI-II scores. Controlling for age and BMI, weight suppression independently predicted all symptom measures except EDE-Q Weight Concern subscale and EDI-3 Body Dissatisfaction scores. After controlling for age and the independent effects of BMI and weight suppression, the weight suppression \times BMI interaction was not a statistically significant predictor of any scores at admission.

When baseline analyses were conducted including only participants with an EDE-Q-confirmed AN diagnosis ($N = 253$), BMI was found to be a statistically significant independent correlate only of EDI-3 Bulimia subscale scores, and weight suppression was found to be a significant correlate only of EDI-3 Bulimia subscale scores and number of missed menstrual periods (Table 4). Of note, as demonstrated in Table 1, removal of participants based on subclinical scores (below 4 out of 6) on certain EDE-Q items resulted in restricted ranges in the confirmed-diagnosis sample in EDE-Q subscale scores and scores on other outcome measures that were highly correlated with those subscales in the full sample (see Table 1 for differences in means and SDs across the two samples). This likely reduced correlations among those outcome variables and all other measures, including the proposed predictors (e.g., coefficients for bivariate correlations among outcome measures were reduced on average by $r = .2$ (range = .06–.33) in comparison to the full sample).

Weight Suppression and BMI as Predictors of Treatment Outcome

Means and standard deviations for outcome variables at discharge as well as intercorrelations among all discharge outcome measures are presented in Tables 1 and 5, respectively. Similar to admission results, statistical information for individual predictors presented in Tables 6 and 7 represent results from the final model

Table 2
Intercorrelations at Admission

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Weight suppression (lbs)	—	-.22***	.86***	.31***	.23***	.09	.03	.06	.09	.07	.19**	.03	.15**	.04	.12*
2. BMI (kg/m ²)	-.28***	—	.25***	-.03	-.05	.05	.10	.09	.10	.09	-.22***	.08	.19***	.12*	.07
3. Highest adult BMI (kg/m ²)	.86***	.19**	—	.29***	.21***	.11*	.07	.08	.12*	.10	.07	.06	.22***	.09	.15**
4. Age (years)	.37***	-.001	.37***	—	.81***	.08	.08	.06	.06	.08	-.19***	-.05	.11*	.002	.18***
5. Duration of illness (years)	.29***	-.01	.30***	.84***	—	.19***	.20***	.19***	.17***	.21***	-.19***	.10	.18***	.14*	.26***
EDE-Q subscale scores															
6. Restraint subscale	.11	-.05	.07	.12	.14*	—	.67***	.68***	.61***	.84***	.01	.66***	.26***	.56***	.42***
7. Weight concern	.02	.12*	.08	.15*	.16*	.37***	—	.89***	.67***	.91***	.06	.76***	.20***	.78***	.57***
8. Shape concern	.06	.15*	.11	.07	.11	.35***	.72***	—	.70***	.93***	.12*	.77***	.25***	.82***	.56***
9. Eating concern	.12	.08	.15*	.05	.08	.36***	.41***	.43***	—	.84***	.13*	.61***	.49***	.57***	.53***
10. Global	.10	.09	.12*	.12	.15*	.70***	.81***	.79***	.73***	—	.09	.80***	.34***	.77***	.59***
11. Periods missed in the last 4 months	.21**	-.21**	.08	-.21**	-.24**	-.14*	-.05	.02	.05	-.04	—	.09	-.01	.10	-.01
EDI-3 subscale scores															
12. Drive for thinness	-.002	.02	-.002	-.13*	-.07	.36***	.54***	.53***	.33***	.54***	.05	—	.26***	.71***	.52***
13. Bulimia	.11	.17***	.18***	.05	.07	.06	.04	.06	.43***	.20***	-.04	.09	—	.12*	.18***
14. Body dissatisfaction	.02	.14*	.08	.01	.07	.37***	.64***	.72***	.35***	.66***	.02	.52***	-.04	—	.53***
15. Beck Depression Inventory-II score	.14*	.08	.17***	.15**	.20**	.19***	.39***	.33***	.35***	.41***	-.04	.26***	.05	.35***	—

Note. WS = weight suppression (kg); BMI = body mass index (kg/m²); EDE-Q = Eating Disorder Examination-Questionnaire; EDI-3 = Eating Disorders Inventory-3. Correlations above the diagonal = results including all participants; correlations below the diagonal = results including only participants with EDE-Q confirmed anorexia nervosa diagnosis. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 3

Relations Among Weight Suppression, Body Mass Index (BMI), and Symptomatology at Admission: Results Including All Participants (N = 350)

Outcome variable	Block 1		Block 2			Block 3	
	ΔR^2	Age β <i>b</i> (<i>SE</i>)	ΔR^2	WS β <i>b</i> (<i>SE</i>)	BMI β <i>b</i> (<i>SE</i>)	ΔR^2	WS \times BMI β <i>b</i> (<i>SE</i>)
EDE-Q subscale scores							
Restraint subscale score	.01*	.06 .08 (.07)	.03*	.18** .07 (.02)	.11 .03 (.02)	.01	-.10 -.03 (.02)
Weight concern score	.01*	.09 .38 (.25)	.02*	.10 .14 (.08)	.14* .14 (.06)	.004	-.06 -.06 (.06)
Shape concern score	.01	.06 .23 (.22)	.03*	.15* .19 (.08)	.14** .14 (.05)	.01	-.08 -.07 (.05)
Eating concern score	.01	.04 .14 (.22)	.04**	.19*** .24 (.07)	.15** .14 (.05)	.01	-.10 -.09 (.05)
Global score	.01*	.07 .25 (.20)	.03**	.17** .20 (.07)	.15** .13 (.05)	.01	-.10 -.08 (.05)
EDI-3 subscale scores							
Drive for thinness score	.001	-.07 -.23 (.19)	.02*	.14* .16 (.06)	.12* .09 (.04)	.01	-.08 -.06 (.04)
Bulimia score	.01*	.05 .20 (.22)	.07***	.21*** .29 (.08)	.26*** .25 (.05)	.01	-.10 -.09 (.05)
Body dissatisfaction score	<.001	-.01 -.18 (1.62)	.03*	.10 .98 (.54)	.17** 1.14 (.38)	.01	-.07 -.46 (.35)
BDI-II score	.03***	.13* 4.87 (2.04)	.02*	.14* 1.68 (.69)	.09 .82 (.49)	.01	-.07 -.58 (.45)
Number of missed menstrual periods (N = 231)	.03**	-.26*** -.96 (.23)	.16***	.31*** .37 (.08)	-.19** -.15 (.05)	.001	.03 .02 (.05)

Note. WS = weight suppression (kg); BMI = body mass index (kg/m²); EDE-Q = Eating Disorder Examination-Questionnaire; EDI-3 = Eating Disorders Inventory-3.

* $p < .05$. ** $p < .01$. *** $p < .001$.

after all predictors have been included. We again compared these with coefficient results for predictor variables when including only Steps 1 and 2 of the model (prior to the addition of the interaction term) and found that differences between the models in the size of standardized beta coefficients and semipartial correlations were small in size, and that the statistical significance of individual predictors did not change between the two models for any outcome variable, with the exception of two minor differences in predictors crossing the threshold for statistical significance in analyses restricted to the confirmed-diagnosis sample.¹

In prospective analyses, for almost all measures, the interaction between weight suppression and BMI at intake was the most consistent predictor of discharge outcome, both in the full sample and in the confirmed-diagnosis sample (Tables 6 and 7). Prediction of EDI-3 Bulimia subscale scores and BDI-II scores was only statistically significant in the confirmed-diagnosis sample (Table 7). Examination of the interaction effects indicated that for most variables, with the exception of discharge weight, admission weight suppression was inversely associated with severity of symptoms at discharge among those with lower BMIs at admission (i.e., higher admission weight suppression was associated with better outcome among those who entered treatment at especially low BMIs). In contrast, among those with higher BMIs at admission, admission weight suppression was positively associated with severity of symptoms at discharge (i.e., higher weight suppression at admission was associated with poorer outcome among those who entered treatment at higher BMIs; Figure 2).

Results indicated that weight suppression consistently predicted higher BMIs at discharge, but this relation was strongest among those with lower BMIs at admission (Figure 3). Rate of weight change was statistically significantly predicted independently by both BMI and weight suppression, but the weight suppression \times BMI interaction was not a statistically significant predictor of rate of weight change (Tables 6 and 7).

To address the possibility that the observed relations between weight suppression, BMI and our outcome measures might be better accounted for by AN subtype, we conducted a series of exploratory analyses. Although individuals with higher weight suppression and individuals with higher BMIs at admission were more likely to be classified as having the AN-BP subtype, the addition of subtype as a covariate in analyses did not change the

¹ In the confirmed-diagnosis sample, the statistical significance of BMI in predicting EDI Drive for Thinness, Full model: $\beta = .18$, Part = .13, $t(162) = 1.89$, $p = .06$; Partial model: $\beta = .21$, Part = .16, $t(163) = 2.14$, $p = .03$; and that of weight suppression in predicting discharge BMI, Full model: $\beta = .18$, Part = .13, $t(247) = 2.43$, $p = .02$; Partial model: $\beta = .12$, Part = .10, $t(248) = 1.89$, $p = .06$, differ slightly between full and partial models.

Table 4

Relations Among Weight Suppression, Body Mass Index (BMI), and Symptomatology at Admission: Results Including Only Participants With Eating Disorder Examination-Questionnaire (EDE-Q) Confirmed Diagnosis of Anorexia Nervosa (AN; N = 253)

Outcome variable	Block 1		Block 2		Block 3	
	ΔR^2	Age β <i>b</i> (<i>SE</i>)	ΔR^2	WS β <i>b</i> (<i>SE</i>)	BMI β <i>b</i> (<i>SE</i>)	WS x BMI β <i>b</i> (<i>SE</i>)
EDE-Q subscale scores						
Restraint subscale score	.01	.10 .10 (.07)	.005	.06 .02 (.02)	-.03 -.01 (.02)	<.001 .01 .003 (.01)
Weight concern score	.02*	.16* .46 (.20)	.02	-.02 -.02 (.07)	.10 .07 (.05)	.01 .05 (.04)
Shape concern score	.01	.05 .11 (.16)	.03*	.07 .05 (.05)	.15* .09 (.04)	.01 .08 .04 (.03)
Eating concern score	.003	-.003 -.01 (.21)	.02*	.15* .15 (.07)	.12 .09 (.05)	<.001 .002 .001 (.04)
Global score	.02	.09 .20 (.15)	.02	.08 .06 (.05)	.10 .05 (.03)	.003 .06 .03 (.03)
EDI-3 subscale scores						
Drive for thinness score	.02*	-.14* -.35 (.17)	.004	.05 .04 (.05)	.02 .01 (.04)	.004 .06 .03 (.03)
Bulimia score	.002	-.04 -.16 (.28)	.07***	.23** .29 (.09)	.26*** .25 (.06)	.01 -.07 -.06 (.06)
Body Dissatisfaction score	<.001	-.02 -.37 (1.67)	.02	.06 .46 (.54)	.15* .83 (.38)	.001 .03 .16 (.33)
BDI-II score	.02*	.11 3.49 (2.28)	.02	.12 1.27 (.75)	.11 .86 (.54)	.002 .05 .33 (.45)
Number of missed menstrual periods (N = 171)	.05**	-.34*** -1.26 (.28)	.16***	.36*** .41 (.09)	-.12 -.10 (.06)	<.001 .01 .01 (.05)

Note. WS = weight suppression (kg); BMI = body mass index (kg/m²); EDE-Q = Eating Disorder Examination-Questionnaire; EDI-3 = Eating Disorders Inventory-3.

* $p < .05$. ** $p < .01$. *** $p < .001$.

pattern of our results, nor did it alter the statistical significance of any of the previously reported prospective findings.²

Discussion

The body weights of individuals with AN are, by definition, objectively low, but previous research indicates varying weight histories among these individuals (Coners et al., 1999; Miyasaka et al., 2003). The field has historically focused on the significance of objectively low current weight status in AN, and the relevance of patients' weight histories to AN symptomatology is only beginning to be considered. Only two previous studies (Klein et al., 2011; Wildes & Marcus, 2012) have examined weight suppression in AN. To our knowledge, no study to date has investigated the possibility that, as has been found in BN, BMI may moderate the effects of degree of weight suppression on psychological and physiological symptoms. The low correlation between BMI and weight suppression in the present sample permitted extension of the initial findings of Wildes and Marcus (2012) and examination of the possibility that a patient's level of weight suppression below her previous highest adult weight and her current, objectively low weight status may interact to predict weight gain achieved in treatment, the speed with which she gains weight in treatment, and the ways in which psychological symptoms change in response to treatment.

At admission to residential treatment, both BMI and weight suppression were independently and positively associated with several measures of psychopathology, including Shape Concern,

Eating Concern, and Global EDE-Q scores as well as EDI-3 Bulimia and Drive for Thinness scores. In addition, higher weight suppression, controlling for BMI, was associated with higher EDE-Q Restraint and depression scores. Controlling for weight suppression, patients with higher BMIs had higher Weight Concern subscale scores and EDI-3 Body Dissatisfaction scores. Patients with lower BMIs, as would be expected, reported more missed menstrual periods. Interestingly, weight suppression was also associated with number of missed menstrual periods, even when controlling for BMI. These results suggest that higher weight suppression, above and beyond absolute weight status, and higher BMI, independently of weight suppression, are both associated with increased eating pathology, general distress, and physiological abnormalities. No statistically significant interaction between BMI and weight suppression was found for any admission variables.

² A total of 46.3% of the sample were classified as having the AN-BP subtype. Results of hierarchical logistic regressions indicated that patients with higher weight suppression and with higher BMIs were more likely to be categorized as AN-BP ($b = .31$, $SE = .11$, $Wald = 8.57$, $p = .003$, $Exp(B) = 1.36$; and $b = .30$, $SE = .08$, $Wald = 14.17$, $p < .001$, $Exp(B) = 1.34$, respectively), but the weight suppression \times BMI interaction was not related to subtype ($p = .782$). All cross-sectional and prospective analyses were repeated adding AN subtype to step 1 of hierarchical regressions. AN subtype was a statistically significant covariate in almost all cross-sectional models (with exception of BDI-II analysis, $p = .07$); however, subtype was not a statistically significant covariate in any models predicting discharge variables.

Table 5
Intercorrelations at Discharge

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Weight suppression (lbs)	—	-.22***	.86***	.31***	.23***	.05	.06	.02	.03	.04	.03	.09	.05	-.02
2. BMI (kg/m ²) ^a	-.28***	—	.25***	-.03	-.05	.02	-.003	-.01	.05	.02	.16*	.12	.11	.16*
3. Highest adult BMI (kg/m ²)	.86***	.19**	—	.29***	.21***	.07	.06	.003	.05	.05	.11	.13*	.09	.07
4. Age (years)	.37***	-.001	.37***	—	.81***	-.17**	-.13	-.16*	-.15*	-.17**	-.12	-.04	-.03	-.02
5. Duration of illness (years)	.29***	-.01	.30***	.84***	—	-.07	-.08	-.11	-.08	-.10	-.05	.004	.03	-.04
EDE-Q subscale scores														
6. Restraint subscale	.06	.05	.10	-.22**	-.12	—	.63***	.59***	.64***	.78***	.52***	.18**	.50***	.45***
7. Weight concern	.08	-.02	.09	-.12	-.09	.63***	—	.77***	.82***	.89***	.71***	.32***	.64***	.55***
8. Shape concern	.01	-.01	.004	-.19*	-.18*	.57***	.75***	—	.91***	.93***	.75***	.23***	.79***	.53***
9. Eating concern	.01	.07	.05	-.17*	-.14	.63***	.81***	.88***	—	.95***	.77***	.25***	.76***	.61***
10. Global	.04	.03	.06	-.20**	-.15	.79***	.89***	.91***	.94***	—	.77***	.27***	.76***	.60***
EDI-3 subscale scores														
11. Drive for thinness	.002	.14	.08	-.14	-.10	.51***	.69***	.72***	.76***	.76***	—	.35***	.74***	.59***
12. Bulimia	.03	.12	.08	-.02	.04	.14	.23**	.19*	.23**	.23**	.34***	—	.19*	.30***
13. Body dissatisfaction	.07	.09	.11	-.01	.01	.51***	.60***	.74***	.72***	.73***	.69***	.16*	—	.53***
14. Beck Depression Inventory-II Score	-.05	.18*	.05	-.05	-.05	.47***	.51***	.50***	.60***	.59***	.59***	.26***	.50***	—

Note. WS = weight suppression (kg); BMI = body mass index (kg/m²); EDE-Q = Eating Disorder Examination-Questionnaire; EDI-3 = Eating Disorders Inventory-3. Correlations above the diagonal = results including all participants; Correlations below the diagonal = results including only patients with EDE-Q confirmed anorexia nervosa diagnosis.

^aCorrelations are with BMI at admission.

* $p < .05$. ** $p < .01$. *** $p < .001$.

While the positive relation between BMI and symptomatology is surprising in light of prior research on the psychological effects of starvation (Keys et al., 1950), consistently higher levels of body dissatisfaction and concern with shape and weight at admission to residential treatment among patients with higher BMIs may be related to a desire to become objectively thinner, anticipated exposure to patients with lower BMIs in the treatment milieu, or anticipation of weight gain in treatment. The observed positive association between BMI and the EDI-3 Bulimia subscale at admission replicates prior findings that bulimic behaviors among patients with AN are associated with higher weight (e.g., Garner, Olmsted, Bohr, & Garfinkel, 1982; Eddy et al., 2002). In addition, the observed association between weight suppression and higher EDI-3 Bulimia subscale scores, independently of BMI, replicates results among patients with BN (Lowe et al., 2007). Both findings are also consistent with the results of our exploratory examinations of AN subtype: individuals higher in weight suppression, independent of BMI, and individuals with higher BMI, independent of weight suppression, were more likely to be diagnosed with AN-BP.

Higher weight suppression may be independently associated with greater levels of psychopathology for both psychological (e.g., increased food obsessions) and biological (e.g., abnormal serotonin levels; Bailer & Kaye, 2011) reasons; however, the cross-sectional nature of admission results precludes determination of the direction of causality. For instance, as previously noted, high body dissatisfaction might be associated with weight suppression because of greater motivation to lose weight, and the association between EDE-Q Restraint and weight suppression may reflect the fact that individuals high in weight suppression previously exerted greater restraint over eating to reach their more highly weight-suppressed state. Whether weight suppression exacerbates AN symptoms, or increased severity of eating disorder psychopathology motivates individuals to suppress their weight to a greater degree cannot be determined in the current investigation. Nevertheless, the observed association between weight suppression and symptomatology, even when BMI was controlled, suggests that weight suppression is an indicator of illness severity above and beyond absolute weight status.

Of note, a number of the aforementioned associations at treatment admission were nonsignificant in analyses including only women whose AN diagnosis was confirmed by EDE-Q responses. In this confirmed-diagnosis sample, weight suppression was statistically significantly associated only with EDI-3 Bulimia score and number of missed menstrual periods, while BMI was associated only with Shape Concern scores and EDI-3 Bulimia and Body Dissatisfaction scores. A likely explanation for the reduction in the number of statistically significant associations in cross-sectional results between the full and confirmed-diagnosis samples is the previously noted restricted ranges resulting from the use of cut-off scores on several EDE-Q items to define the confirmed-diagnosis sample. Reduced power in the confirmed-diagnosis sample also may have impacted our ability to detect effects. Given the limitations of determining diagnoses based on self-report items from the EDE-Q and given the broadening of criteria for AN in DSM-5 (i.e., addition to criterion B of “persistent behavior that interferes with weight gain, even though at a significantly low weight” and addition to criterion C of “persistent lack of recognition of the seriousness of the current low body weight”), the results based on

Table 6
Prediction of Symptomatology at Discharge: Results Including All Participants (N = 238)

Outcome variable	Block 1			Block 2			Block 3	
	ΔR^2	Admission score β <i>b</i> (<i>SE</i>)	Age β <i>b</i> (<i>SE</i>)	Weight change β <i>b</i> (<i>SE</i>)	WS β <i>b</i> (<i>SE</i>)	BMI β <i>b</i> (<i>SE</i>)	WS \times BMI ΔR^2	β <i>b</i> (<i>SE</i>)
BMI (<i>N</i> = 350)	.27***	.58*** .51 (.04)	-.09 -.33 (.17)		.03*** .20*** .23 (.06)		.02**	-.15** -.12 (.04)
Rate of weight change (<i>N</i> = 350)	.001		-.08 -.02 (.01)		.12*** .16*** .01 (.004)	-.28*** -.02 (.003)	.003	-.06 -.003 (.003)
EDE-Q subscale scores								
Restraint subscale score	.16***	.31*** .778 (.16)	-.24*** -.78 (.21)	.20** .17 (.07)	.01 -.06 -.07 (.08)	.09 .07 (.06)	.03**	.18** .14 (.05)
Weight concern score	.35***	.53*** .56 (.06)	-.25*** -1.10 (.25)	.29*** .33 (.08)	.02* .02 .03 (.09)	.17** .19 (.08)	.02**	.14** .13 (.05)
Shape concern score	.39***	.68*** .68 (.06)	-.24*** -1.06 (.24)	.26*** .29 (.08)	.003 -.02 -.03 (.08)	.07 .07 (.07)	.01*	.12* .12 (.05)
Eating concern score	.25***	.44*** .36 (.05)	-.17** -.54 (.19)	.27*** .22 (.06)	.01 -.02 -.02 (.07)	.12 .09 (.06)	.03**	.17** .12 (.04)
Global score	.41***	.58*** .56 (.05)	-.25*** -.88 (.19)	.28*** .24 (.06)	.01 -.02 -.02 (.07)	.10 .09 (.06)	.02**	.14** .11 (.04)
EDI-3 subscale scores								
Drive for thinness score	.28***	.50*** 3.23 (.36)	-.13* -2.78 (1.23)	.07 .34 (.40)	.01 .01 .04 (.43)	.13 .65 (.36)	.03**	.17** .75 (.25)
Bulimia score	.34***	.59*** .44 (.04)	-.10 -.31 (.17)	.02 .01 (.05)	.001 -.03 -.03 (.06)	.03 .02 (.05)	.01	.09 .06 (.04)
Body dissatisfaction score	.45***	.66*** .65 (.05)	-.03 -.70 (1.45)	.17** 1.15 (.46)	.01 -.06 -.57 (.49)	.09 .58 (.43)	.02**	.13** .75 (.29)
BDI-II score	.21***	.45*** .40 (.05)	-.11 -3.71 (2.06)	.14 1.14 (.66)	.02 -.11 -1.23 (.72)	.14 .14 1.07 (.61)	.01	.11 .78 (.44)

Note. WS = weight suppression (kg); BMI = body mass index (kg/m²); EDE-Q = Eating Disorder Examination-Questionnaire; EDI-3 = Eating Disorders Inventory-3.

* $p < .05$. ** $p < .01$. *** $p < .001$.

this restricted sample should be interpreted with caution. In addition, those who did not meet EDE-Q diagnostic criteria were at an objectively low weight, would likely meet criteria for a diagnosis of AN per *DSM-5*, and were admitted to residential treatment for an eating disorder; therefore, the full sample is conceivably clinically representative.

Unlike the cross-sectional findings, in which weight suppression and low BMI could represent mere consequences of eating disorder psychopathology, results of the longitudinal analyses indicate that baseline weight suppression interacts with BMI in the prediction of degree of symptom change over treatment, even when controlling for potential explanatory variables, including age and weight change during treatment. In prospective analyses including baseline values as covariates, the weight suppression \times BMI interaction was the most consistent predictor of discharge symptomatology levels, including all of the EDE-Q subscales, as well as EDI-3 Drive for Thinness and Body Dissatisfaction. The nature of the significant interaction was the same for all of the above variables: at lower admission BMIs, greater weight suppression predicted better scores at discharge, while among those with the highest BMIs at admission, greater weight suppression predicted higher scores at discharge. Notably, the prediction of psychological symptom outcome was independent of the amount of weight gained in treatment and patient age, and results of exploratory analyses indicated that these findings cannot be better accounted for by diagnostic subtype.

When analyses were restricted to the sample of women whose diagnosis was confirmed by EDE-Q, the weight suppression \times

BMI interaction predicted depression and EDI-3 Bulimia scores at discharge in addition to the variables mentioned above. Again, at lower admission BMIs, greater weight suppression predicted lower scores on these measures at discharge while among those with the highest BMIs at admission, greater weight suppression predicted higher discharge scores. Although results in the EDE-Q confirmed-diagnosis sample must be interpreted with caution given previously noted limitations, the interactive effect of weight suppression and BMI in predicting Bulimia subscale scores is particularly interesting in light of recent findings that patients with AN high in weight suppression at admission, regardless of admission BMI, are more likely to endorse bulimic behaviors while in treatment (Wildes & Marcus, 2012). The present results, while preliminary and only statistically significant in the EDE-Q-confirmed-diagnosis sample, suggest that high weight suppression in combination with higher BMI at admission may best predict increased bulimic symptoms over the course of treatment. As patients with AN with relatively higher BMIs and high levels of weight suppression at admission may be most comparable in absolute and relative weight status to previously studied individuals with BN who are high in weight suppression, the current findings appear consistent with prior BN research (Butryn et al., 2011).

Of note, effect sizes for the weight suppression \times BMI interaction in both the full sample and confirmed-diagnosis sample are small and only explain an additional 2%–6% of the variance in outcome measures. Nonetheless, the pattern of results is markedly

Table 7

Prediction of Symptomatology at Discharge: Results Including Only Participants With EDE-Q Confirmed Diagnosis of AN (N = 171)

Outcome variable	Block 1			Block 2			Block 3	
	ΔR^2	Admission score β b(SE)	Age β b(SE)	Weight change β b(SE)	ΔR^2	WS β b(SE)	BMI β b(SE)	WS x BMI β b(SE)
BMI (N = 253)	.27***	.59*** .51 (.05)	-.03 -.10 (.21)		.01	.15* .17 (.07)		.02** -.16** -.12 (.04)
Rate of weight change (N = 253)	.001		-.10 -.02 (.01)		.12***	.19** .01 (.005)	-.25*** -.01 (.003)	.01 -.12 -.005 (.003)
EDE-Q subscale scores								
Restraint subscale score	.15***	.23** .79 (.26)	-.31*** -1.12 (.28)	.27** .24 (.08)	.03	-.02 -.02 (.10)	.20* .17 (.08)	.04** .17 (.06)
Weight concern score	.15***	.26*** .36 (.10)	-.24** -1.01 (.33)	.40*** .41 (.10)	.04*	.02 .03 (.11)	.24** .24 (.09)	.04** .19 (.06)
Shape concern score	.18***	.26*** .43 (.12)	-.24** -.96 (.31)	.38*** .37 (.09)	.02	-.01 -.01 (.10)	.15 .14 (.09)	.03** .16 (.06)
Eating concern score	.13***	.22** .23 (.07)	-.15 -.49 (.25)	.39*** .31 (.08)	.03	-.01 -.01 (.09)	.19* .14 (.07)	.05** .16 (.05)
Global score	.24***	.34*** .50 (.10)	-.28*** -.94 (.35)	.39*** 4.32 (.08)	.03	.01 .01 (.08)	.21* .16 (.07)	.03** .13 (.05)
EDI-3 subscale scores								
Drive for thinness score	.10***	.27*** 2.27 (.61)	-.10 -2.10 (1.59)	.16 .76 (.48)	.03	-.01 -.07 (.53)	.18 .84 (.44)	.06*** 1.04 (.31)
Bulimia score	.35***	.61*** .45 (.05)	-.06 -.16 (.20)	.10 .07 (.06)	.01	-.08 -.07 (.07)	.05 .04 (.06)	.02* .09 (.04)
Body dissatisfaction score	.38***	.56*** .59 (.07)	.03 .69 (1.71)	.26** 1.61 (.51)	.01	-.06 -.44 (.56)	.13 .74 (.48)	.03** 1.00 (.33)
BDI-II score	.15***	.36*** .37 (.08)	-.09 -2.92 (2.69)	.14 1.19 (.81)	.02	-.14 -1.53 (.92)	.12 .96 (.79)	.02* .16* 1.16 (.55)

Note. WS = weight suppression (kg); BMI = body mass index (kg/m²); EDE-Q = Eating Disorder Examination-Questionnaire; EDI-3 = Eating Disorders Inventory-3.

* $p < .05$. ** $p < .01$. *** $p < .001$.

consistent across outcome measures, and effects were slightly larger in the confirmed-diagnosis sample.

One possible mechanism of the consistently observed weight suppression \times BMI interaction may be differential psychological reactions to a given degree of weight gain as a function of individual weight history. Patients with AN with relatively higher BMIs and high weight suppression are more likely to have a history of being closer to overweight than those with higher BMIs and low weight suppression (e.g., Figure 1, lines C and D). The prospect of weight regain may be more distressing for patients with a history of being near-overweight or overweight, as they may fear that weight regain will culminate in a return to their previous weight status. Thus, increased distress about gaining weight, even when amount of weight gained is held constant, may explain the higher discharge scores on measures of body dissatisfaction and concerns about weight and shape among individuals with high admission BMIs and high weight suppression. Further, those who are more distressed about weight gain may be more likely to resist increases in food intake in treatment, which could account for higher EDE-Q Restraint subscale scores at discharge in this group.

The inverse relation between weight suppression and discharge symptomatology found among patients with the lowest BMIs is somewhat surprising. Because amount of weight gained in treatment was included as a covariate, differential weight gain cannot explain this finding. The outcome may again be a result of differences in psychological reactions to weight gain. Patients with low BMIs and high weight suppression levels, while potentially less

likely than patients with higher BMIs and high weight suppression levels to have a history of being overweight, have previously reached weights substantially higher than their low weights at admission to treatment (e.g., Figure 1, line A). Conversely, patients with low BMIs and low weight suppression levels are more likely to have been chronically underweight and have never reached adult weights significantly higher than their admission weights (Figure 1, line B). As a result, weight gain during treatment may be more distressing for these patients, who may reach or surpass their highest historical adult weights while in treatment. As previously noted, such differences in distress associated with weight gain, even when amount of weight gain is held constant, may account for differences in EDE-Q and EDI-3 scores at discharge.

Just as admission weight suppression's prediction of discharge symptomatology depended upon admission BMI, so, too, did its prediction of weight gain over the course of treatment. As was found by Wildes and Marcus (2012), those higher in weight suppression gained more weight in treatment; however, in the present sample, weight suppression had the strongest relation with amount of weight gained in treatment among those with the lowest BMIs at admission, and at higher BMIs, the relation between weight suppression and weight gain was weak. Though the admission BMI of all patients was under 18.5 kg/m², and all patients included in the sample were required to gain weight, the observed weaker relation between weight suppression and weight gain at higher admission BMIs may be related to these patients needing to

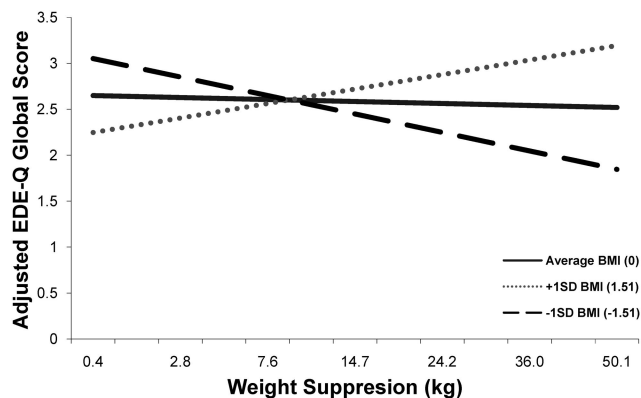


Figure 2. Relations among admission weight suppression, admission body mass index (BMI), and Eating Disorder Examination Questionnaire (EDE-Q) Global scores at discharge. EDE-Q Global subscale score values graphed represent discharge values controlling for baseline values, age, and weight change over treatment, with all participants included. The relation between weight suppression and EDE-Q Global score was significantly moderated by BMI ($p = .007$). The pattern of results was similar using only the confirmed-diagnosis sample, and for all statistically significant predictions of EDE-Q, Eating Disorders Inventory-3 (EDI-3), and Beck Depression Inventory-II (BDI-II) outcome measures.

gain comparatively less weight to reach their goal weight in treatment. The current study also replicated previous findings that those higher in weight suppression gain weight more quickly in treatment (Wildes & Marcus, 2012); however, unlike the results of Wildes and Marcus (2012), BMI was also an independent predictor of rate of weight gain such that those at lower BMIs gained weight more quickly.

Our results indicate that weight restoration may be most difficult for those who do not have a history of ever weighing significantly more than their current weight. Those who do have this history may find it easier to regain weight for both psychological and metabolic reasons. Prior results in BN as well as in nonclinical populations indicate that weight suppression is associated with proneness to later weight gain (e.g., Herzog et al., 2010; Lowe, Davis, et al., 2006; Stice et al., 2011), and evidence from studies of weight loss among both obese and normal-weight populations suggests that weight loss is associated with biological changes that promote weight regain (Leibel, Rosenbaum, & Hirsch, 1995; Rosenbaum & Leibel, 2010). Because the residential treatment program in the present study prescribed meals designed to promote a standardized rate of weight gain, the differences in amount and rate of weight gain as a function of admission weight suppression and BMI suggest differences in either compliance with treatment, biological mechanisms related to weight gain, such as level of energy expenditure, or both.

In terms of rate of weight gain, our findings indicate that high levels of weight suppression at admission, regardless of BMI, and lower BMIs at admission, regardless of weight suppression, are associated with the most rapid weight gain in treatment. Wildes and Marcus (2012) note that previous findings regarding the effects of rapid weight gain in treatment have been mixed: There is evidence to suggest that a faster rate of weight gain in treatment may be iatrogenic (Herzog, Zeeck, Hartmann, & Nickel, 2004;

Lay, Jennen-Steinmetz, Reinhard, & Schmidt, 2002; Willer, Thuras, & Crow, 2005) and contrasting evidence that a faster rate of weight restoration predicts improved clinical outcome at 1-year follow-up among patients with AN (Lund et al., 2009). In light of these previous, mixed findings, further research is needed to determine how our results concerning rate of weight gain may inform treatment recommendations.

In summary, results of the present study confirm recently reported findings that among patients with AN, weight suppression predicts faster rate of weight gain during intensive treatment independently of admission BMI (Wildes & Marcus, 2012). As previously reported (Wildes & Marcus, 2012), higher weight suppression levels were associated with greater weight gain in treatment, but in the current sample the strength of this relation varied with BMI. When amount of weight gained in treatment was held constant, an individual's weight history and current weight status interacted to predict psychological outcome at discharge.

Taken together, our results suggest that weight suppression is an important clinical indicator in AN and that it should be assessed and considered in conjunction with BMI or percent of ideal body weight (e.g., based on the Metropolitan Life Insurance Tables; Pai & Paloucek, 2000) in evaluating the illness severity and prognosis of AN patients. High weight suppression was associated with greater psychopathology at baseline, and, among patients with higher BMIs, predicted high levels of symptomatology at discharge. The combination of low weight suppression with low BMI may also represent a negative prognostic indicator.

These findings could carry important implications for the treatment of AN. Target weights in treatment are often set based on absolute weight status, but the statistically significant main effects of weight suppression and the weight suppression \times BMI interactions found in the present study, if confirmed by further research, suggest that weight goals may need to be more idiographically determined. Standard guidelines of 90% of ideal body weight as a minimum "weight restoration" goal (American Psychiatric Association, 2006; Golden et al., 1997), for example, do not

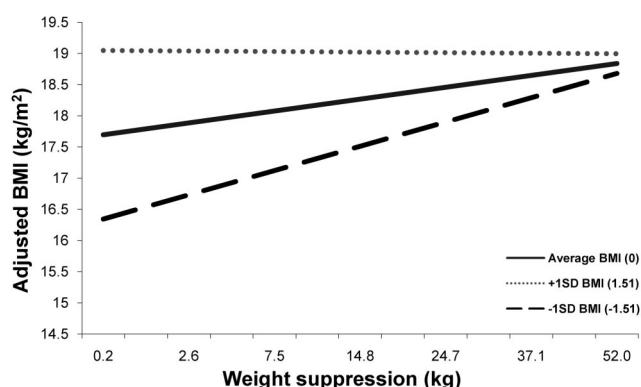


Figure 3. Relations among admission weight suppression, admission body mass index (BMI), and BMI at discharge. BMI values graphed represent discharge values controlling for baseline values and age, with all participants included. Weight suppression at admission was consistently positively associated with absolute weight status at discharge, but admission BMI moderated this relation ($p = .006$), which was strongest among those with lower BMIs at admission. The pattern of results was similar including only the confirmed-diagnosis sample.

account for the influence of individual weight history found to be important in this sample and in others (Klein et al., 2011; Wildes & Marcus, 2012). Target weights based only on absolute weight status could contribute to higher rates of relapse and more difficult long-term weight stabilization and “normalization.”

Strengths of this study include the investigation of a novel construct in this population and the inclusion of a variety of psychological outcome measures. The large sample size is particularly important given the low prevalence of AN and the resulting difficulty of conducting large-scale research on the disorder. In addition, the study was conducted at large, community-based eating disorder treatment facilities, there were no exclusion criteria beyond an admission BMI threshold, and the length of stay was limited to approximately six weeks on average, supporting the “real-world” clinical significance of the results.

Limitations of this study must also be noted. First, our symptom measures were limited to self-report questionnaires which, particularly for the assessment of bulimic symptoms, may be prone to bias. This may be especially true among women with AN, to whom any eating in a treatment setting could be accompanied by a sense of “loss of control.” In addition, although there is evidence supporting the validity of self-reported highest past weights (Swenne et al., 2005; Tamakoshi et al., 2003), validity of self-reported weight history has not been studied among adults with AN. The lack of a rigorously standardized treatment approach, despite the advantage of generalizability, is also a limitation: while weight gain during treatment was controlled in prospective analyses, we are unable to rule out the possibility that differences in the treatment approach across patients may have been a confounding variable.

Though the predominance of Caucasian women in the sample reflects the higher rates of Caucasians who seek treatment for eating disorders (American Psychiatric Association, 1993; Becker, Franko, Speck, & Herzog, 2003), the applicability of these findings to other populations cannot be assumed. Further, menstrual status data provided one marker of physiological abnormality, but we were unable to directly assess any other physiological variables (e.g., hormones, electrolyte levels) in the study sample. An additional limitation is the high level of attrition in prospective analyses (32% of the initial sample did not complete discharge self-report measures). It is possible that those who did and did not complete discharge self-report measures differ in some meaningful way; however, analyses comparing these subsamples on all variables of interest yielded no statistically significant differences except in length of stay, which was expected.

In addition to replication of the current findings, future research should attempt to identify factors that may explain the relatively better symptom levels at discharge of women with higher levels of weight suppression and lower BMIs at admission. Further investigations should also extend the present work by examining weight suppression, BMI, and their interaction as predictors of the course of illness after discharge including time to remission, as has been investigated in BN (Lowe et al., 2011). Both weight suppression and BMI were related to menstrual status at admission, and additional studies should investigate, over a longer time period, whether admission absolute and relative weight statuses predict resumption of menses. As the results of Klein and colleagues (2011) would suggest, individuals high in weight suppression may need to reach a higher BMI in order to resume regular menstrua-

tion and other physiological functions compared to patients with comparable admission BMIs and lower levels of weight suppression. The identification of biological markers associated with weight suppression in AN also represents an important direction for future research.

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