ORIGINAL ARTICLE

Examining Weight Suppression as a Predictor of Eating Disorder Symptom Trajectories in Anorexia Nervosa

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ABSTRACT

Objective: Research in individuals with bulimia nervosa has highlighted the clinical significance of weight suppression (WS), defined as the difference between one's highest and current weight. More recently, studies have suggested that WS also may play a role in symptom maintenance and weight gain during treatment in anorexia nervosa (AN) and that the influence of WS on AN outcomes may depend on an individual's body mass index (BMI). However, no study has investigated whether WS or the interaction between WS and BMI is associated with the longer-term course of eating pathology following treatment discharge in patients with AN.

Method: The current study examined a sample of females with AN ($N = 180$) who completed interviews and self-report questionnaires at discharge from intensive treatment and at 3, 6, and 12-months after discharge. Latent growth curve models tested whether WS, BMI, or the WS by BMI interaction significantly predicted the trajectory of eating disorder symptoms (i.e., Eating Disorder Examination global score, BMI, frequency of loss of control eating, frequency of purging) over the year following discharge.

Results: WS at discharge predicted change in BMI, and the interaction between WS and BMI predicted growth in eating disorder severity and purging frequency over time. Neither WS nor its interaction with BMI predicted growth in loss of control eating frequency.

Discussion: Results provide further support for the clinical significance of WS in AN symptom maintenance, but suggest that the influence of WS likely depends on an individual's BMI as well as the outcome being measured. © 2016 Wiley Periodicals, Inc.

Keywords: anorexia nervosa; weight suppression; loss of control eating; purging; growth curve models

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Introduction

The construct of weight suppression (WS), defined as the difference between one's highest weight at current height and one's current weight, has gained considerable attention in the eating disorders field. Much of this work has focused on the clinical significance of WS in bulimia nervosa (BN).[1–5] Cross-sectional studies have found associations between higher WS and greater binge eating and purging frequency in individuals with full or subthreshold BN,[2,4] and longitudinal studies have indicated that WS predicts the onset and maintenance of bulimic syndromes up to 10 years later.[5,6] Additionally, higher WS in individuals with BN has predicted greater weight gain during short-term inpatient[7] and outpatient treatment,[8] as well as over a longer follow-up period.[9] With notable exceptions,[8,10,11] associations between WS and BN have been documented even after accounting for covariates and weight-related variables (e.g., body mass index [BMI], duration of illness, dieting frequency), suggesting an important role of WS in bulimic symptomatology.

It has been posited that alterations in physiological processes related to eating and weight regulation (e.g., changes in hormone levels) and psychological consequences of weight loss (e.g., fear of returning to premorbid weight)[1,3,5] may explain associations between WS and maintenance of BN. These mechanisms also may be salient to the maintenance of anorexia nervosa (AN). Although by definition all individuals with AN are

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underweight, variability exists in terms of weight history[12] and WS.[13–15] Similar to individuals with BN, individuals with AN who are higher in WS may have a greater disruption in physiological processes (e.g., reduced anorexigenic [leptin] or increased orexigenic [ghrelin] hormone levels) that could increase vulnerability to bulimic episodes. Moreover, individuals with AN who are high in WS may have lost more weight prior to the onset of AN than individuals low in WS, and consequently, psychological characteristics associated with low weight (e.g., weight gain fears, thinness expectancies) and eating disorder behaviors may be elevated in this group. As such, assessment of WS in individuals with AN may be useful for informing specific weight and psychological targets for treatment.

Four studies have examined associations between WS and AN, with findings similar to those in BN. Specifically, greater WS at admission was associated with increased weight gain and faster rate of weight gain during treatment in two independent samples.[13,15] Wildes and Marcus[15] also found that greater WS at admission predicted the presence of bulimic symptoms within 4 weeks prior to discharge in a sample of 185 patients with AN. Although Carter et al.[13] did not find a statistically significant association between admit WS and bulimic symptoms at discharge potentially due to a smaller sample ($N = 56$) and less variability in WS, the small to moderate effect size was similar to that of Wildes and Marcus.[15] Berner et al.[14] found positive correlations between WS and eating pathology at admission in a residential treatment sample of individuals with AN. Furthermore, WS significantly interacted with BMI to predict eating disorder psychopathology at discharge. Specifically, among patients with a higher BMI at treatment admission, higher WS was associated with greater cognitive and behavioral eating disorder symptoms at discharge whereas among patients with a lower BMI at treatment admission, higher WS was associated with more favorable outcomes. These findings suggest that the influence of WS on AN symptoms may depend on BMI.

Only one study examining WS in AN has included a longer duration of follow-up. Using a series of linear regressions, Witt et al.[16] found significant, positive associations between WS and BMI at 6- and 10-year follow-ups and a trend ($P = 0.06$) at 18-year follow-up. At 6- and 10-year follow-ups, there was also a significant interaction with BMI, such that associations found between WS and later BMI were strongest among those with the lowest BMI at baseline. Nevertheless, this study was limited by a relatively small sample size ($N = 47$), and statistical analyses focused on each time point independently rather than modeling the trajectory of change in BMI over time. Furthermore, the authors did not investigate associations with specific eating disorder behaviors (e.g., binge eating and purging) or overall eating disorder severity, which may be particularly relevant given theoretical models and associations found in BN.

Overall, initial studies suggest that considering the amount of weight loss, as well as the relationship between weight loss and BMI, may be important in AN. However, given some mixed findings regarding associations with bulimic symptoms and outcome, additional studies are necessary to elucidate the impact of WS in AN. Furthermore, no study has examined whether WS influences behavioral symptoms of eating disorders in AN over a longer follow-up period, including after treatment discharge. Thus, consistent with previous studies,[14,16] we examined WS, BMI, and the interaction between WS and BMI as predictors of eating disorder symptom trajectories over the year following discharge from intensive behavioral treatment for AN. Based on previous research, we hypothesized that higher WS at discharge would be associated with growth in overall eating disorder symptom severity, BMI, and frequency of objective and subjective binge eating and purging following discharge. We also hypothesized that, among those with a higher BMI at discharge, higher WS would be associated with greater severity of eating disorder symptoms in the year following discharge.

Method

Participants

Participants were 194 patients receiving intensive behavioral treatment for AN who were enrolled in a longitudinal study examining personality subtypes in AN; inclusion and exclusion criteria have been described elsewhere.[17,18,a] Nine participants did not provide adequate information to determine WS and were excluded from analyses. Additionally, given the small

[a]Participants in the current study were the same as those included in Wildes and Marcus,[15] which reported on the cross-sectional and longitudinal associations between weight suppression (WS) and clinical variables at treatment admission and associations with outcomes at discharge (e.g., weight gain during treatment, bulimic symptoms). The current study reports on associations between WS at discharge and clinical outcomes in the year following discharge, which have not been reported previously.

TABLE 1. Clinical characteristics of the sample at admission, discharge, 3-, 6-, and 12-month follow-ups

	Admission		Discharge		3-month		6-month		12-month	
	M (range)	SD	M (range)	SD	M (range)	SD	M (range)	SD	M (range)	SD
Weight suppression (kg)[a]	16.81 (0–46.77)	9.89	10.94 (0–45.18)	9.74	11.46 (0–47.63)	9.58	11.92 (0–47.63)	9.61	11.42 (0–47.82)	9.91
Duration of illness (years)	9.04 (0–45)	9.14	–		–		–		–	
AN subtype (% binge-eating/purging)	104	57.8%	–		–		–		–	
Level of care (% inpatient)	153	85%	–		–		–		–	
Days in treatment	44.06 (5–110)	24.82	–		–		–		–	
EDE Global Score	3.09 (0–5.59)	1.46	2.54 (0–5.10)	1.48	2.66 (0–5.67)	1.52	2.57 (0–5.89)	1.56	2.52 (0–5.9)	1.59
Body mass index	15.67 (9.39–18.42)	1.81	17.96 (12.14–22.39)	1.48	17.73 (11.34–23.69)	2.21	17.66 (9.62–24.63)	2.42	17.86 (11.69–24.58)	2.65
LOC eating	6.96 (0–101)	16.41	1.07 (0–52)	4.79	6.07 (0–84)	13.91	6.42 (0–90)	16.40	5.43 (0–108)	15.36
Purging	20.84 (0–812)	67.24	2.70 (0–78)	8.96	12.86 (0–168)	25.01	14.42 (0–140)	28.34	13.98 (0–168)	28.13

Notes: AN, anorexia nervosa; EDE, Eating Disorder Examination; LOC, loss of control; $N = 180$.
[a]The weight suppression variable had one extreme outlier (value [>70 kg] > 5 standard deviations above the mean) due to a patient with a past history of bariatric surgery; thus, we replaced this extreme score with the next highest value and used this "corrected" variable in all analyses. There were no differences between restricting and binge/purge subtype on weight suppression at admission (t (178) = 0.92; $P = 0.36$); LOC eating and purging represent mean number of episodes per month. Level of care indicates treatment status at admission. AN subtype and level of care represent frequency (n) and percentage of participants.

number of males ($n = 5$), the current study included only female participants ($n = 180$), which is consistent with prior research on WS in AN.[14,16] Clinical characteristics of the sample are included in Table 1. One hundred thirteen participants (62.8%) received inpatient treatment only, 19 (10.6%) received day hospital treatment only, and 48 (26.7%) received both inpatient and day hospital treatment. The mean (SD) age of the sample was 26.8 (10.2) years (range = 16–62), and the majority of participants were white/non-Hispanic (95.1%). Participants were well educated with 63.2% ($n = 117$) having completed at least some college.

Procedures

Procedures were approved by the local institutional review board and all participants signed informed consent (or assent for participants under 18 years) prior to participation. Medical charts were reviewed to obtain information on BMI at admission and discharge and treatment type (inpatient and/or day hospital). Participants completed in-person interviews and self-report questionnaires within two weeks of admission and at discharge. Follow-up interviews and questionnaires were completed at 3, 6, and 12 months postdischarge. Follow-ups were conducted either in person or by telephone and mail to increase probability of participation. All assessments were conducted by trained masters or doctoral-level research staff, and interview assessments were audio-recorded. Ten percent of interviews were re-rated by independent interviewers to determine interrater reliability.

Of the 180 participants included in this study, 173 (96.1%) completed discharge assessments, 152 (84.4%) completed 3-month follow-up assessments, 151 (83.8%) completed 6-month follow-up assessments, and 141 (73.8%) completed 12-month follow-up assessments. More than 90% of the sample ($n = 165$; 91.7%) completed at least one postdischarge assessment. There were no significant differences between participants who completed all follow-up assessments and those with missing data for at least one follow-up on WS, BMI, or any interview variable (P's > 0.10).

Measures

Eating Disorder Diagnosis and Symptoms. The Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-I)[19] and the Eating Disorder Examination Interview 16th edition (EDE)[20] were used to confirm current diagnosis of AN. Diagnoses were based on the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV)[21] criteria with the exception of the amenorrhea criterion.[22] Additionally, individuals who had BMI ≤17.5 and denied fear of fatness were included in the study, consistent with "non-fat phobic" AN.[23] Age of AN

onset was determined by the SCID[19] and used to calculate duration of illness. Interrater reliability for AN diagnosis was excellent ($\kappa = 1.00$).

The EDE[20] assesses symptoms across a 28-day period and yields a global score and four subscales (i.e., restraint, eating concerns, weight concerns, shape concerns). The EDE global score was used to represent overall eating disorder symptom severity. The EDE also establishes the presence and frequency of purging (i.e., vomiting, laxative use, diuretic use) and loss of control (LOC) eating episodes, including objective bulimic episodes (OBE) (i.e., consumption of an unambiguously large amount of food with LOC) and subjective bulimic episodes (SBE) (i.e., LOC during consumption of an amount of food that is not large). Given the relatively low endorsement of OBEs in the current sample (range across time points = 3.3–20.6% [$n = 5$–29] of participants) and the fact that both types of binge episodes had similar growth trajectories, OBEs and SBEs were combined into a frequency of LOC eating variable to increase variability for analyses. Further, recent research suggests that LOC over eating may be the most clinically relevant feature of binge eating.[24] Inter-rater reliabilities for the frequency of OBEs ($r = 1.00$), SBEs ($r = 1.00$), and purging ($r = 1.00$) were excellent.

BMI and WS. Height was measured by a stationary stature board, and weight was measured by a digital scale at each assessment point to calculate BMI (weight in kilograms/height in meters squared). Most participants who completed follow-up assessments via phone and mail provided consent to collect height and weight data from a health professional (e.g., physician, therapist, dietician). If verified height and weight could not be obtained, participants' self-reported height and weight were used (for details see[25]). Weight history was determined using an investigator-administered questionnaire that included self-report of demographic and weight history variables. Identical to prior studies,[13,14] WS was calculated as the difference between one's highest weight at current height (excluding pregnancy) and current weight. Research supports the validity and use of self-reported highest weight in eating disorder samples.[26]

Statistical Analyses

Latent growth curve models were used to test whether WS, BMI, or the interaction between WS and BMI at discharge significantly predicted the trajectory of eating disorder symptoms (i.e., EDE global score, BMI, frequency of LOC eating and purging) over the year following discharge from intensive treatment. Growth curve models for continuous outcomes were used for EDE global score and BMI; growth curve models for count data were used for LOC eating and purging frequency. A negative binomial distribution was specified for these count variables, as negative binomial models are preferred to Poisson models when the mean and variance of the count distribution are not equivalent. Model comparisons confirmed that negative binomial models provided a substantially better fit to the count data than Poisson models.[b] Despite the large number of zero counts for LOC eating and purging (57–83%) across time points, negative binomial models also provided a better fit to the data than zero-inflated negative binomial models,[c] which require estimating double the number of parameters than non-zero inflated models.

Prior to examining predictors of eating disorder symptom stability and change, we fit unconditional models to characterize the trajectory of individual symptoms. Specifically, we tested whether only linear or both linear and quadratic growth processes contributed to change in each symptom following treatment discharge. Time was coded as 0, 1, 2, and 3 for discharge, 3-, 6-, and 12-month follow-ups, respectively. Both fixed and random effects for intercepts and slopes were estimated to model the average change, as well as individual differences in change, over time. Conditional models examined whether WS and BMI at discharge, as well as their interaction, predicted eating disorder symptom severity, LOC eating frequency, and purging frequency at discharge (intercept), as well as change in these variables over the year following discharge (slope(s)). Additionally, we examined whether WS at discharge predicted BMI (intercept) or change in BMI (slope(s)) following discharge. All models controlled for duration of illness, level of care at treatment discharge (inpatient vs. day hospital), and AN subtype (binge-eating/purging vs. restricting). Significant predictive effects were plotted using the online tools specific to latent curve analysis by Preacher, Curran, and Bauer.[27]

Models were fit with Mplus version 7.0.[28] Full information maximum likelihood estimation was used, as this method allows for the use of all available data and accounts for missing data under the assumption that data are missing at random.[29] For continuous outcomes, absolute model fit was evaluated using the χ^2 goodness-of-fit statistic, the comparative fit index (CFI)[30] and the root-mean-square error of approximation (RMSEA).[31] Hu and Bentler[32] suggest that CFI values >0.95 and RMSEA values < 0.06 indicate good model fit, although some more recent papers argue against using strict cut-offs to

[b]LOC eating: Poisson Akaike Information Criterion (AIC), Bayesian Information Criteria (BIC) = 3,335.17, 3,351.27 vs. Negative Binomial AIC, BIC = 2,002.58, 2,031.56; Purging: Poisson AIC, BIC = 5,289.11, 5,305.22 vs. Negative Binomial BIC = 2,632.80, 2,661.78

[c]LOC eating: Zero-inflated negative binomial AIC, BIC = 2,010.51, 2,062.33 vs. Negative Binomial AIC, BIC = 2,002.58, 2,031.56; Purging: Zero-inflated negative binomial AIC, BIC = 2,703.81, 2,777.54 vs. Negative Binomial AIC, BIC = 2,632.80, 2,661.78

TABLE 2. Fit indices for unconditional and conditional linear growth curve models for body mass index and Eating Disorder Examination–Global Score

Model	χ^2 (df)	CFI	RMSEA	$\Delta\chi^2$ (Δdf)	AIC	BIC
EDE Global Score						
1. Unconditional quadratic	0.55 (1)	1.00	0.00	–	1,879.00	1,920.73
2. Unconditional linear	10.08 (5)	0.99	0.06	9.53 (4)*	1,880.53	1,909.42
3. Conditional quadratic	9.10 (7)	1.00	0.04	–	1,811.10	1,909.56
Body mass index						
1. Unconditional quadratic	0.003 (1)	1.00	0.00	–	2,390.90	2,432.77
2. Unconditional linear	26.77 (5)	0.94	0.15	26.77 (4)***	2,409.67	2,438.66
3. Conditional quadratic	5.25 (5)	1.00	0.02	–	2,300.81	2,380.49

Notes: EDE, Eating Disorder Examination; CFI, Comparative Fit Index; RMSEA, root-mean-square error of approximation; AIC, Akaike Information Criterion; BIC, Bayesian Information Criterion;
***$P < 0.001$; *$P < 0.05$.

evaluate model fit.[33] Notably, these absolute model fit indices are not available for negative binomial models, so only relative model fit was considered. Both the Akaike Information Criterion (AIC)[34] and the Bayesian Information Criterion (BIC)[35] were used to evaluate relative model fit and compare both nested and non-nested models. AIC and BIC evaluate model fit relative to model parsimony and are lowest in the best-fitting models. Nested model comparisons also were conducted using the χ^2 difference test and the Satorra-Bentler scaled χ^2 difference test[36] for continuous and count outcomes, respectively. Statistically significant χ^2 difference values lead to the rejection of the nested, more restrictive model in favor of the less restrictive model, whereas nonsignificant χ^2 values indicate preference for the more parsimonious model.

Results

Information related to treatment progress and symptom change between admission and discharge has been reported previously for this sample.[15] Notably, of the 180 participants in the current study, 34 (18.9%) reported at least one episode of objective binge eating or purging during the 4 weeks prior to discharge. Of these participants, 12 were receiving inpatient treatment only, 6 were receiving day hospital treatment only, and 16 transitioned between inpatient and day hospital treatment from admission and discharge. Although BMI increased significantly from admission to discharge ($t(179) = -20.49$, $P<0.001$), 110 participants (61.1%) remained below a healthy weight (BMI < 18.5 kg/m^2). Furthermore, of the 129 participants who completed all three follow-up assessments, 28 (21.7%) maintained a healthy weight (BMI > 18.5 kg/m^2) across all follow-up time points. Approximately 81.1% of participants ($n = 146$) received individual outpatient treatment and 48.9% ($n = 88$) were re-hospitalized (i.e., inpatient and/or day hospital) in the year following treatment discharge.

EDE global score and BMI

Unconditional Growth Models. Fit statistics for the linear and quadratic unconditional growth curve models are presented in **Table 2**. The best-fitting model for both EDE global score and BMI was the quadratic growth curve model, as indicated by absolute and relative model fit indices (with the exception of the BIC for EDE global score). Further, χ^2 difference test results indicated that dropping the quadratic slope resulted in a significant decrease in model fit for both EDE global score and BMI (**Table 2**). **Table 3** presents the estimated means and variances from the unconditional quadratic models. EDE global scores did not change significantly across the follow-up period, as indicated by nonsignificant mean estimates for linear and quadratic slopes. However, significant variance estimates indicated that there was variability across participants in EDE global scores at discharge and over the year following discharge. In contrast, the significant mean slope estimates for BMI suggested that, on average, BMI decreased following discharge from treatment, with a slower rate of change in BMI as the year progressed. Significant variance estimates indicated that notable variability in BMI at discharge and over the year following discharge also was present.

Conditional Growth Models. Relative, but not absolute, fit indices suggested that including predictors of growth curve trajectories provided a better fit to the data for both EDE global score and BMI (**Table 2**). As shown in **Table 3**, neither WS nor BMI at discharge predicted EDE global scores at discharge or the trajectory of change in these scores over time. However, the interaction between WS and BMI significantly predicted both the EDE global score intercept and linear slope (**Table 3**). Among those with a high BMI at discharge, higher WS was significantly associated with greater EDE global scores at discharge (**Figure 1a**). In contrast, WS and EDE global scores were unrelated among individuals

TABLE 3. Parameter estimates from the unconditional and conditional continuous models

	Intercept		Linear Slope		Quadratic Slope	
	Estimate (SE)	*P*	Estimate (SE)	*P*	Estimate (SE)	*P*
EDE Global Score						
Unconditional model						
Means	2.61 (0.11)	**<0.001**	0.09 (0.10)	0.33	−0.04 (0.03)	0.14
Variances	2.00 (0.40)	**<0.001**	0.75 (0.40)	0.06	0.06 (0.02)	**0.01**
Conditional model						
Weight suppression	0.16 (0.11)	0.13	0.03 (0.11)	0.80	−0.02 (0.03)	0.57
Body mass index	0.15 (0.12)	0.21	0.02 (0.11)	0.84	−0.005 (0.03)	0.88
Weight suppression × Body mass index	0.24 (0.11)	**0.04**	−0.22 (0.11)	**0.04**	0.06 (0.03)	0.07
Duration of illness	0.10 (0.11)	0.33	0.11 (0.10)	0.27	−0.01 (0.03)	0.69
Level of care	0.20 (0.25)	0.42	−0.24 (0.23)	0.29	0.03 (0.07)	0.63
AN subtype	0.89 (0.22)	**<0.001**	−0.01 (0.20)	0.98	−0.004 (0.06)	0.95
Intercepts	2.03 (0.20)	**<0.001**	0.17 (0.18)	0.35	−0.05 (0.06)	0.40
Residual variances	1.58 (0.38)	**<0.001**	0.59 (0.40)	0.14	0.05 (0.02)	**0.02**
Body mass index						
Unconditional model						
Means	17.99 (0.11)	**<0.001**	−0.33 (0.17)	**0.05**	0.11 (0.05)	**0.04**
Variances	1.68 (0.57)	**0.003**	2.23 (0.78)	**0.004**	0.14 (0.08)	0.07
Conditional model						
Weight suppression	−0.06 (0.11)	0.57	0.39 (0.18)	**0.03**	−0.10 (0.06)	0.11
Duration of illness	−0.07 (0.11)	0.52	−0.20 (0.18)	0.26	0.02 (0.06)	0.72
Level of care	1.28 (0.23)	**<0.001**	0.15 (0.36)	0.68	−0.02 (0.12)	0.87
AN subtype	0.08 (0.22)	0.70	−0.18 (0.34)	0.60	0.06 (0.11)	0.59
Intercepts	17.44 (0.19)	**<0.001**	−0.30 (0.31)	0.32	0.09 (0.10)	0.38
Residual variances	1.46 (0.55)	**0.008**	2.19 (0.76)	**0.004**	0.13 (0.08)	0.11

Notes: EDE, Eating Disorder Examination; Level of care (at discharge), inpatient (0) or day hospital (1). Intercept refers to symptom level at discharge; Slope refers to change in symptoms across the year following discharge. Mean estimates for intercepts and slopes refer to average level of symptoms at discharge and average change in symptoms postdischarge, respectively. Variance estimates for intercepts and slopes refer to degree of individual variability in symptoms at discharge and change in symptoms postdischarge, respectively.

with a low BMI at discharge. With regards to the linear change in EDE global scores postdischarge, higher WS among those with high BMI was associated with the maintenance of high EDE global scores following discharge (**Figure 1b**). Furthermore, higher WS among those with low discharge BMI was associated with a greater increase in EDE global score after discharge than low WS (**Figure 1c**). [d]

Results from analyses examining BMI as the dependent variable indicated that there was a significant effect of discharge WS on the linear change in BMI over time (**Table 3**). Specifically, individuals with lower WS had a significant decrease in BMI after discharge (**Figure 1d**). In contrast, individuals with higher WS did not show this same postdischarge decrease in BMI, suggesting that they maintained weight in the year following discharge.

LOC Eating and Purging

Unconditional Growth Models. Fit statistics for the linear and quadratic unconditional growth curve models are presented in **Table 4**. For both LOC eating and purging, models with only linear sources of change provided a better fit to the data than models with both linear and quadratic sources of change, as evidenced by lower AIC and BIC values for the linear models and nonsignificant scaled χ^2 difference tests (**Table 4**). Slope mean estimates presented in **Table 5** indicate that, on average, there were trend-level significant increases in LOC eating and significant increases in purging following discharge from intensive treatment. Significant intercept and nonsignificant slope variance estimates suggest that individuals varied on their level of LOC eating and purging at discharge, but not on their trajectory of change in these behavioral symptoms over time.

Conditional Growth Models. The inclusion of predictors of LOC eating and purging resulted in better model fit relative to unconditional models (**Table 4**). Data presented in **Table 5** demonstrate that AN subtype was the only variable significantly associated with LOC eating at discharge, and there were no significant predictors of growth in LOC eating frequency over time. Additionally, neither WS nor BMI was significantly associated with purging frequency at discharge, and neither variable significantly predicted purging trajectories over the year following discharge. However, the interaction

[d]We also examined whether WS, BMI, and the WS by BMI interaction predicted growth in each of the EDE subscale scores (i.e., Restraint, Eating Concern, Shape Concern, Weight Concern) over the year following treatment discharge. These analyses were uniformly nonsignificant (data available on request).

FIGURE 1. Effects of weight suppression and the weight suppression by body mass index interaction on eating disorder symptoms in AN. EDE, Eating Disorder Examination; BMI, body mass index; WS, weight suppression.

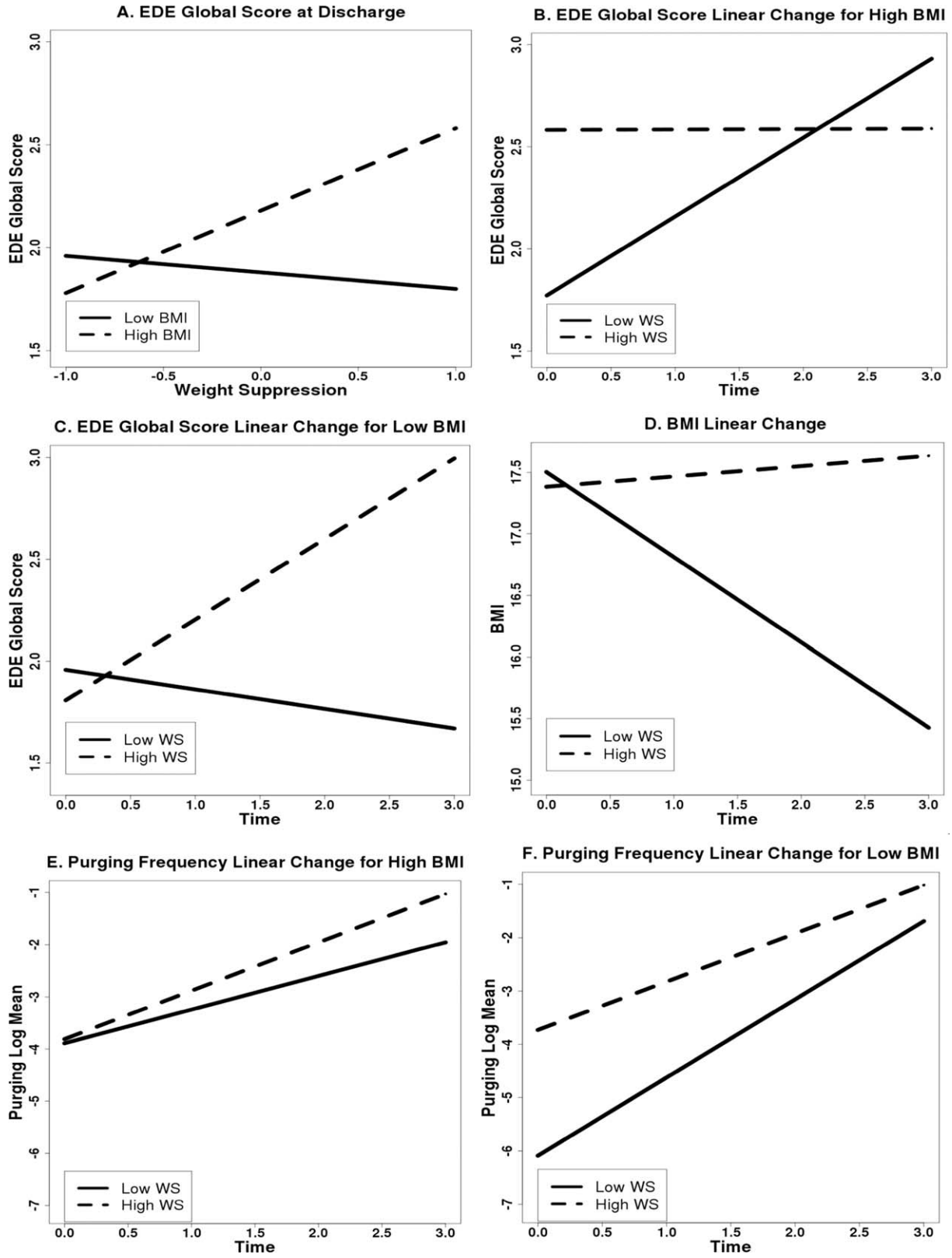

TABLE 4. Fit indices for unconditional and conditional linear growth curve models for loss of control eating and purging frequency

Model	−2lnL (Free Parameters)	$\Delta\chi^2_{S\text{-}B}$ (Δdf)	AIC	BIC
Loss of control eating				
1. Unconditional quadratic	−988.80 (13)	–	2,003.61	2,045.47
2. Unconditional linear	−992.29 (9)	7.73 (4)	2,002.58	2,031.56
3. Conditional linear	−943.65 (21)	–	1,929.30	1,996.23
Purging				
1. Unconditional quadratic	−1,305.71 (13)	–	2,637.42	2,679.42
2. Unconditional linear	−1,307.40 (9)	4.12 (4)	2,632.80	2,661.78
3. Conditional linear	−1,213.94 (21)	–	2,469.87	2,536.81

Notes: −2lnL, minus two times the log-likelihood; $\Delta\chi^2_{S\text{-}B}$, Satorra-Bentler scaled χ^2 difference test; AIC, Akaike Information Criteria; BIC, Bayesian Information Criterion.

TABLE 5. Parameter estimates from the unconditional and conditional negative binomial models

	Loss of Control Eating Frequency				Purging Frequency			
	Intercept		Slope		Intercept		Slope	
	Estimate (SE)	*P*	Estimate (SE)	*P*	Estimate (SE)	*P*	Estimate (SE)	*P*
Unconditional model								
Means	−1.48 (0.55)	**0.008**	0.37 (0.22)	0.08	−1.12 (0.62)	0.07	0.44 (0.21)	**0.03**
Variances	11.74 (2.68)	<**0.001**	0.04 (0.07)	0.60	17.36 (3.53)	<**0.001**	0.07 (0.08)	0.43
Conditional model								
Weight suppression	0.27 (0.38)	0.48	0.03 (0.14)	0.85	0.61 (0.34)	0.07	−0.07 (0.11)	0.51
Body mass index	0.18 (0.42)	0.66	−0.08 (0.15)	0.57	0.52 (0.43)	0.22	−0.20 (0.14)	0.16
Weight suppression × Body mass index	−0.03 (0.38)	0.94	−0.12 (0.14)	0.41	−0.57 (0.31)	0.06	0.21 (0.09)	**0.01**
Duration of illness	−0.01 (0.36)	0.99	−0.07 (0.15)	0.65	−0.17 (0.33)	0.60	0.10 (0.11)	0.36
Level of care	0.22 (0.81)	0.79	−0.21 (0.32)	0.52	−0.84 (0.83)	0.32	−0.38 (0.28)	0.17
AN subtype	3.30 (0.70)	<**0.001**	−0.24 (0.28)	0.40	6.24 (0.80)	<**0.001**	−0.55 (0.29)	0.06
Intercepts	−3.42 (0.93)	<**0.001**	0.64 (0.42)	0.13	−4.38 (0.94)	<**0.001**	0.98 (0.37)	**0.008**
Residual variances	8.43 (2.41)	<**0.001**	0.04 (0.07)	0.59	6.88 (2.32)	**0.003**	0.06 (0.10)	0.55

Notes: Level of care (at discharge), inpatient (0) or day hospital (1). Intercept refers to symptom level at discharge; Slope refers to change in symptoms across the year following discharge. Mean estimates for intercepts and slopes refer to average level of symptoms at discharge and average change in symptoms postdischarge, respectively. Variance estimates for intercepts and slopes refer to degree of individual variability in symptoms at discharge and change in symptoms postdischarge, respectively.

between WS and BMI significantly predicted change in purging frequency over time. Among those with high BMIs at discharge, higher WS predicted greater increases in purging after discharge compared to lower WS (**Figure 1e**). In contrast, at low BMI, lower WS was associated with greater increases in purging after discharge compared to higher WS (**Figure 1f**).

Discussion

Numerous studies have demonstrated the clinical significance of WS in BN, and initial research conducted in individuals with AN suggests that WS impacts eating disorder symptoms and weight gain during treatment. To our knowledge, this is the first study to investigate the influence of WS on eating disorder symptom trajectories after discharge from intensive treatment in patients with AN. Results indicated that the only unique effect of WS was on change in BMI over the year following treatment, with lower WS at discharge from treatment associated with a decrease in BMI over time. In contrast, the interaction between WS and BMI significantly predicted eating disorder symptom severity at discharge as well as growth in global symptom severity and purging frequency over time. These findings extend results from previous studies on WS in AN and suggest that the predictive effects of WS on eating disorder psychopathology after treatment discharge may depend on discharge BMI.

Associations between WS and BMI are relatively consistent with previous research on WS and weight gain in BN and AN. Previous studies have consistently found positive associations between WS and increases in BMI over short (i.e., several weeks) and longer time frames (i.e., 5–10 years). Given the chronicity of the current sample (i.e., mean duration of illness = 9 years), it is unsurprising that BMI significantly decreased in the year following discharge. Although lower WS at discharge

predicted this decrease over time, higher WS at discharge predicted weight maintenance, in that it was not associated with this same decrease in BMI. This finding is similar that of Herzog and colleagues[9] who found that among individuals with BN, lower WS at baseline was associated with modest weight loss over a 5-year follow-up period. In relation to the current study, it is possible that after gaining weight during treatment, individuals low in WS and closest to their lifetime highest weights had greater motivation to lose weight. On the other hand, individuals who remained high in WS at discharge may have been less dissatisfied with their weight, given that they were presumably not as close to their highest weights. The only previous study examining associations between WS and BMI over longer term follow-up in individuals with AN included adolescents followed at 6, 10, and 18 years after baseline.[16] Thus, it is possible that with a longer follow-up in the current study, those with high WS do not just maintain higher BMIs after discharge but might be more likely to gain weight over time.

Among patients with a higher BMI, there were similar patterns in predicting growth in eating disorder severity and purging frequency over time. Individuals with both high BMI and high WS at discharge exhibited maintenance of or an increase in eating disorder severity and purging frequency in the year following discharge, respectively. Although this is the first longitudinal study to examine purging separately from binge eating, Berner et al.[14] found a similar pattern for eating disorder severity. Specifically, among those with higher BMI at admission, higher WS predicted greater severity of eating disorder symptoms at discharge. Greater pathology among individuals with high WS and high BMI may be due to psychological reactions and stress related to weight gain. Theoretically, patients with higher BMIs and high WS may be more likely to have a history of being overweight and may have increased fears of returning to their premorbid weight. This fear may then contribute to increases in cognitive eating disorder symptomatology as well as extreme attempts to maintain weight loss (e.g., increased purging).

In addition to finding that WS influenced symptom trajectories among those with high BMIs, the current study also found that, at low BMIs, higher WS predicted greater increases in eating disorder severity in the year following discharge. This finding is in contrast to that of Berner et al.[14] in which low BMI and high WS at treatment admission was associated consistently with lower eating disorder severity at discharge. It is possible that the combined influence of WS and BMI on symptoms at discharge may be distinct from their combined influence on longer-term outcome, which may, in part, reflect effects of treatment setting on cognitions and behaviors. Furthermore, the relationship between WS and BMI on outcome also may depend on the specific outcome being measured. Indeed, although higher WS among those with lower BMIs predicted increased eating disorder severity over time in the current study, lower WS among those with lower BMIs predicted greater increases in purging compared to individuals with low BMI and high WS. However, this latter finding may reflect regression to the mean as those with lower WS started off with relatively lower rates of purging at discharge.

Taken together, these findings highlight potential problems with focusing solely on BMI as a measure of improvement and the potential importance of considering one's weight history during treatment. Despite being at potentially healthy BMIs, individuals high in WS may need to gain to target weights higher than individuals with lower WS to prevent maintenance of eating disorder psychopathology post-treatment. Given that patients with high WS and higher BMIs may be most fearful of gaining weight, these individuals may need additional support and education reflecting the role of WS on the continuation of symptoms.

Contrary to hypotheses, neither WS nor the interaction between WS and BMI had an effect on LOC eating frequency at discharge or growth in LOC eating over time. It is possible that patients were particularly committed to dietary restriction and weight loss in reaction to increased stress from gaining weight during treatment; thus, any relations between WS, BMI, and LOC eating may be unlikely during this short follow-up period. With that said, a previous study using this same sample found that WS predicted the presence of bulimic symptoms (i.e., objective binge eating or purging) at discharge from treatment[15] and a separate study[14] found that the interaction between WS and BMI predicted self-reported bulimic symptom scores at discharge. However, two recent studies did not detect statistically significant associations between WS and composite measures of bulimic symptoms in AN[13] or in a mixed eating disorder sample.[37] Mixed results may be due to differences in methodology across studies including outcome measures, dichotomous versus continuous variables, and examination of moderators.

Given that this was the first study to examine WS as a predictor of LOC eating postdischarge, it also may be that WS is less relevant for longer term

outcomes related to LOC eating in individuals with AN. This finding contrasts with longitudinal findings in BN and suggests that WS may play a distinct role in the maintenance of LOC eating among those who are a healthy weight versus underweight. Indeed, although participants gained a significant amount of weight from treatment intake to treatment discharge, the average weight was still below a healthy weight (BMI <18.5). It is possible that the impact of WS on LOC eating is obscured while BMIs are low, but would be more evident in a fully weight-restored sample. Future studies examining the influence of WS on longer-term outcome in a more completely weight-restored sample of individuals with AN may help to elucidate whether WS is a meaningful predictor of eating behavior in AN.

Strengths of this study include use of a large clinical sample of individuals with AN followed over multiple time points for one year. Moreover, retention was very high with over 90% of the sample completing at least one follow-up assessment. The use of latent growth curve models enabled us to test whether WS, BMI, and the WS by BMI interaction predicted eating disorder symptom trajectories, rather than level of eating disorder symptoms, over a one year period. Finally, we used a semi-structured interview assessment with good psychometric properties to assess eating disorder symptoms and controlled for important covariates (i.e., duration of illness, AN subtype, level of care) in analyses.

Limitations include the fact that all participants were followed after receiving intensive behavioral treatment, which likely resulted in a restricted range of eating disorder symptoms at discharge. The majority of patients remained significantly underweight at discharge, thus we were unable to examine specifically how WS may be associated with maintenance of weight restoration. Moreover, it is difficult to determine whether findings would generalize to a period that did not immediately follow treatment discharge or to a less severe, non-treatment seeking sample. Similarly, participants were female, so results may not generalize to males with AN. In addition, despite having a large sample for a longitudinal study of patients with AN, our sample size was relatively modest for conducting latent growth curve models, especially with count outcomes, and we did not have a sufficient number of individuals endorsing objective binge eating episodes to examine objective and subjective binge eating episodes separately. Finally, like all previous studies on WS, we relied on self-report to assess highest previous weight, which may include biases inherent in any self-report measure.

In sum, our findings confirm that WS has clinical relevance for responsiveness to intensive behavioral treatment and support assessment of WS among individuals seeking treatment for AN. Indeed, this information might be used to help identify patients for whom additional psychoeducation, support, and coping techniques may be warranted. In light of previous and current findings, additional studies examining the influence of WS and its interaction with BMI on the longer-term course of AN in larger, more diverse samples appear warranted. Future studies using a fully weight-restored sample may help elucidate the importance of WS in symptom maintenance in AN and the potential role of intensive treatment in these associations.

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