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Weight suppression predicts weight change over 5 years in bulimia nervosa

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ABSTRACT

Recent studies suggest that weight suppression (WS), defined as the discrepancy between current and highest past weight, predicts short-term weight gain in bulimia nervosa (BN) during treatment. The current study was designed to build on this preliminary work by examining the relation between WS and long-term weight change in BN. Treatment-seeking women ($N=97$) with DSM-IV BN participated in a naturalistic longitudinal follow-up study of eating disorders. At intake, height and weight were measured and highest past weight was assessed. Self-reported weights were collected every 6 months for 5 years. Hierarchical Linear Modeling (HLM) estimated growth curves for weight change over time. Significant inter-person variability was detected for intercepts and slopes ($P<0.001$) so both were treated as random effects. Participants' weights increased over the study course, moderated by baseline WS ($P<0.001$), such that higher WS predicted more rapid weight gain. Weight change was not associated with entry weight, height, or highest-ever weight, suggesting that WS per se predicted weight change. These findings complement previous short-term studies in BN by demonstrating that WS predicts weight gain over 5 years. Because weight gain could spur radical dieting that maintains BN, these results have important treatment implications.

1. Introduction

Most individuals with bulimia nervosa (BN) are in the normal weight range, yet their apparently unremarkable body weight conceals a history of once weighing considerably more than they currently do. This discrepancy, which Lowe (1993) has labeled "weight suppression" (WS), was emphasized by Russell (1979) in his classic paper that first identified BN as an eating disorder, but has been little studied since. Weight suppression is defined as the difference between highest past weight (since reaching adult height) and current measured body weight. Individuals with BN are high in WS; Butryn et al. (2006) reported a mean WS of 9.6 kg in outpatients and Lowe et al. (2006) reported a mean WS of 12.0 kg in residential patients with BN. Because individuals in these studies had body mass indices (BMI; kg/m^2) in the middle of the normal weight range, this means that many of those with BN were once overweight, an observation that coincides with the weight history findings from a community-based study of BN (Fairburn et al., 1997).

Interestingly, though much has been written about how weight loss in obese individuals may set the stage for weight regain (Rosenbaum et al., 2008), few such concerns have been raised about the major weight losses most individuals with BN have undergone. The weight losses experienced by individuals with BN may be of even greater concern because, given that most women with BN have never been obese, a weight loss of a given size represents a larger percentage reduction in most individuals with BN than in obese individuals. Furthermore, if reducing weight well below its highest previous level makes weight gain more likely, such effects could exacerbate bulimic behavior because the dieting that may be required to avoid or reverse weight gain could drive the maintenance of binge eating and purging (Fairburn et al., 1993b). Studies have in fact found that WS predicts weight gain in nonclinical women (Lowe et al., 2006), in outpatients with BN (Carter et al., 2008), and in residential patients with BN (Lowe et al., 2006). Lowe et al. (2007) found that WS was positively related to the frequency of objective binge eating in individuals with BN and Butryn et al. (2006) found that greater WS predicted poorer outcome (study dropout and continuation of eating disorder symptoms at the end of treatment) in the cognitive–behavioral treatment of BN (though Carter et al. (2008) did not replicate this effect). These studies suggest that individuals with BN who are high in WS may be caught in a "biobehavioral bind": Suppressing their weight may reduce their feelings of fatness and unattractiveness but may fuel

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binge eating, purging and eventual weight gain, leading to renewed intensive dieting and further binge eating and compensatory behavior.

Although WS has been consistently predictive of weight gain from periods ranging from several weeks to several months, no study has examined its longer-term relation to weight change. If WS as a predictor of weight gain is restricted to small amounts of weight over relatively brief periods of time, then its role in the perpetuation of BN may be limited. However, if WS predicts large weight gains over long periods of time, then it may play a more significant role in the maintenance of BN because such weight gain would presumably be anathema to these individuals, leading to intensified weight loss efforts that help perpetuate the disorder.

The Massachusetts General Hospital (MGH) Longitudinal Study of Anorexia and Bulimia Nervosa is a prospective, naturalistic examination of 246 treatment-seeking women with anorexia nervosa (AN) or BN. The detailed longitudinal weekly data available from study participants offers a unique opportunity for investigating the relationship between WS among women with BN and prospective weight change. The availability of comprehensive assessment data at entry also allowed us to examine whether relevant covariates (i.e. age and entry BMI) might account for any prospective prediction of weight change by WS at entry. This study extends the design of previous studies by investigating weight change over a longer duration of follow-up and by assessing weight at frequent intervals. Based on the prediction of short-term weight gain in previous studies of WS, we hypothesized that weight suppression at study entry would predict greater weight gain over 5 years.

2. Method

2.1. Participants

Participants in the MGH Longitudinal Study of Anorexia and Bulimia Nervosa were recruited from MGH and other Boston-area treatment centers between October 1987 and June 1990. Five hundred and fifty-four women were screened for study inclusion. Of those, 229 women agreed to participate in the study after meeting the criteria for AN or BN as detailed in the Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (APA, 1987). Participants had to be English-speaking, live within 200 miles of the study site, be at least 12 years of age, and have no evidence of an organic brain syndrome or terminal illness. Prior to the first follow-up interview, four women discontinued participation, leaving a sample of 225 women. Twenty one additional participants with AN were recruited in 1991, increasing the sample to 246. When the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders was published (APA, 1994), participants' eating disorder entry diagnoses were reclassified according to the new criteria; the sample comprised 51 women with AN restricting subtype, 85 women with AN binge/purge subtype, and 110 women with BN. Characteristics of the full sample at intake have been described elsewhere (Herzog et al., 1992, 1999).

Participants for this report were drawn from the women with BN at study entry; thirteen participants were removed from the analyses because they became pregnant during the course of follow-up (i.e., between study entry and 5 years), leaving a final sample of 97. At entry, the mean duration of eating disorder illness was 6.2 years (S.D. = 6.6) and the mean age of the participants was 25.7 years (S.D. = 6.7). Women with BN were followed for a median of 9.5 years and 93.6% of these women were followed for 5 years (attrition rate = 7/110, 6.4%). On the basis of a Mann–Whitney *U* test, there were no significant differences between those who remained in the study and those who discontinued participation with regard to age, weight, or WS. A 5-year follow-up interval was used in the current report to maximize the available data and to avoid the widening confidence intervals that exist beyond 5 years due to attrition.

2.2. Procedure

Participants meeting inclusion criteria for the study were interviewed in person by a trained research assistant. Current and lifetime Axis I disorders were assessed, and data were gathered on current and past eating disorder symptomatology. Additionally, objective measurements of participants' height and weight were taken during this interview. During follow-up, participants were interviewed every 6 months. Interviews were conducted in person whenever possible or by telephone. Approximately 90% of follow-up body weights were collected via self-report. However, because we found near-perfect agreement between self-reported and measured body weights at baseline ($r = 0.96$, $P < 0.001$), this procedure presumably introduced little bias. Furthermore, any bias would be consistent over time within participants. Compensation was provided for all interview assessments.

This study was approved by the MGH Institutional Review Board and written informed consent was obtained from all participants.

2.3. Measures

At entry, current and lifetime diagnoses were assessed with a modified version of the Schedule for Affective Disorders and Schizophrenia—Lifetime Version (Spitzer and Endicott, 1979), which included a section from the Diagnostic Interview Schedule (Robins et al., 1981) with criteria for AN and BN. Women completed the Eating Disorders Inventory questionnaire (EDI) (Garner et al., 1983), which assessed self-reported current weight, height, highest past weight (phrased to inform participants to exclude pregnancy), and lowest past weight. A calibrated stadiometer and balance beam scale were used to obtain current height and weight measurements. See Herzog et al. (1992) for a complete description of all measures completed during the entry interview.

Measurement of WS at study entry was calculated by subtracting participants' current weight from their highest past weight (since reaching their adult or current height), as self-reported in the EDI. The validity of recalled past weights has been supported by a study that found a correlation of 0.85 between measured body weight at age 25 and recalled weights for age 25 that were collected an average of 20 years later (Tamakoshi et al., 2003); the mean error of recalled weights was just 1.28 kg, suggesting that the absolute size of the error in recalled weights was small. If on entry to the study a participant reported that she currently weighed her highest weight, WS equaled zero.

During follow-up, participants were interviewed with the Longitudinal Interval Follow-up Evaluation—Eating Disorders Version (LIFE-EAT II), a modified version of the LIFE II interview (Keller et al., 1987). The LIFE-EAT II was used to assess symptoms of eating disorders, comorbid diagnoses, pharmacological and psychological treatment participation, and psychosocial functioning. Self-reported weights were recorded at each follow-up interview and were analyzed in this report at 6-month intervals.

Entry weight and follow-up weights were calculated into BMI measurements (kilograms divided by (meters)^2).

2.4. Data analytic procedure

The broad analytic plan used followed Hedeker and Gibbons (2006) and Peugh and Enders (2005). SPSS version 14 was used to complete all analyses. As a first step, descriptive statistics were generated to identify the distribution of scores and the need for transformation. Unless noted otherwise, the distribution of all variables was sufficiently normal.

Hierarchical linear modeling (HLM) was used to investigate changes in BMI over time. HLM is a form of multi-level modeling that expands upon traditional regression techniques by (a) more accurately modeling changes in individuals, (b) treating time as continuous rather than a fixed set of points, (c) allowing inclusion of individuals with missing data, and (d) allowing greater flexibility in the specification of covariance structures among repeated measures.

Our HLM analyses included two levels. Changes in the outcome over time (weight) were modeled at level-1 for each individual. Specifically, participants' BMI was measured every 6 months for 60 months. BMI at intake was treated as time 0, thereby setting baseline BMI as the model intercept. Participant characteristics (i.e., WS and age) were modeled at level-2. HLM assumes that, for each individual, the dependent variable (i.e., BMI) is a function of intraindividual variables (i.e., time), person-level variables (i.e., WS), and error.

Our HLM analysis included two major steps. In the first step, unconditional models were specified to determine whether patterns could be identified in the mean change in BMI over time (fixed effects), and whether these patterns varied by individual (random effects). Covariates representing linear and non-linear (quadratic, cubic, and quartic) patterns of change were included in this step. This was done to determine whether significant interindividual variability exists in BMI slopes (i.e., change in BMI over time), which may be accounted for by level-2 covariates. Models with good fit are generally identified by parsimony, the presence of significant variability in slopes, and reliable coefficients (i.e., reliability > 0.1).

In the second step, level-2 covariates (WS and age) were added to the model to predict variability in BMI slopes. Level-2 predictors were mean centered before entry into the analysis. These models tested the hypotheses that baseline WS and age predict the direction and rate of change in BMI after a diagnosis of BN. Full maximum likelihood estimation was used in all HLM models to facilitate model comparison.

3. Results

3.1. Descriptives

Ninety-seven participants with a mean age of 25.47 years (S.D. = 6.66) were assessed at baseline. Mean BMI and the number of participants who contributed data at each assessment, are reported in Table 1. Mean baseline weight was 62.78 kg (S.D. = 9.63). Mean baseline WS was 3.68 (S.D. = 4.33) BMI units, or 10.11 kg (S.D. = 12.12). Baseline WS and BMI were uncorrelated ($r = -0.01$, $P > 0.1$,