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An Investigation of Weight Suppression in a Population-Based Sample of Female Twins

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Abstract

Objective—Weight suppression (WS), maintaining a body weight below one's maximum adult weight, is associated with bingeing, purging and weight gain in clinical samples.

Method—We investigated associations between eating disorder-related variables and WS and additive genetic (A), common (C), and unique (E) environmental contributions to WS in a population-based sample of 1503 female adult twins.

Results—Modeling results were similar for participants reporting no binge eating (NBE) and those reporting binge eating plus loss of control (BE+LOC): 20– 25% percent of the variance in WS was due to A and 70–75% to E. Among NBE participants, Restraint, drive for thinness, body dissatisfaction, and dieting during child/adulthood were related to WS. Restraint, Disinhibition, and dieting during childhood were significantly associated with WS in the BE+LOC subsample.

Discussion—Although maintaining lower body weight could be advantageous, interventionists should take care when addressing weight suppression in individuals vulnerable to eating disorder symptomatology.

Weight suppression (WS), defined as the discrepancy between one's highest adult and current weight, is a relatively understudied concept in the area of eating disorders. It was first highlighted in Lowe's [1] three-factor model of dieting behavior, which included frequency of dieting and overeating, current dieting, and WS. To date, this construct has been investigated primarily among clinical samples, and evidence suggests that WS negatively impacts the course and treatment of eating disorders [e.g., 2]. However, given the rapid increases in rates of overweight and obesity in the U.S. [3–4], it is possible that the

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ability to maintain weight loss could be advantageous among subsamples of the general population. The following sections briefly review studies of WS and eating-and weight-related constructs among eating disordered and non-eating disordered samples.

Restraint theory posits that dietary restraint leads to subsequent overeating [5–6]. However, some have argued that restraint theory does not distinguish between chronic and acute dieting [1]. That is, while acute dieting may increase risk for overeating (including binge eating), chronic dieting may be advantageous for sustaining healthy weight loss. Data suggest that individuals who successfully maintain weight loss in the long term are able to consciously control their dietary intake. For example, participants in the National Weight Control Registry, who have lost an average of 33 kg and maintained this weight loss for more than five years, report fairly high levels of cognitive restraint [7]. Notably, these levels are lower than those of eating disorder patients [7]. Experimental studies suggest that individuals who have lost weight successfully and maintained that loss (i.e., weight suppressors) have adapted their appetitive reactions in a way that facilitates their ability to control weight (e.g., reduced preference for sweets [8]). Thus, the construct of WS may behave differently among individuals with and without eating disorders.

Most investigations have demonstrated the negative effects of WS on eating disorders and related constructs. In addition, it has been hypothesized that WS might be particularly relevant to bulimia nervosa (BN), as individuals with this disorder often enter treatment at weights substantially below their highest adult weights. Specifically, in two samples of BN patients in a low-normal weight range, Garner and Fairburn [9] found that one-third and one-half, respectively, had previously been at least 15% overweight. Further, WS was found to be a significant predictor of weight gain among individuals receiving inpatient treatment for BN [10]. WS was also positively associated with binge eating frequency in a sample of women with BN [11]. Evidence further suggests that WS can impact the course of this disorder [2,11]. For example, Butryn et al. [2] found that WS predicted attrition from BN treatment as well as poor outcomes (i.e., inability to achieve abstinence from bingeing and purging) among treatment completers. Taken together, these results suggest that WS may contribute to the development and maintenance of eating disorders, particularly BN.

In addition to these investigations of WS in samples of patients with eating disorders [2,10,11], one recent study included non-treatment seeking college freshmen [12]. Lowe, Annunziato et al. [12] found that WS and a history of dieting for weight loss predicted weight gain. However, restraint, disinhibition, and emotional eating were not associated with weight gain in this sample. The authors noted that these results provided further support for WS and restraint as distinct constructs; further, they argued that WS appears to be a robust predictor of future weight gain.

These studies provide important information regarding the WS construct; however results from treatment-seeking and college samples may not be applicable to the general population of adults. Although WS has been associated with eating disorder-related traits and weight gain among these groups, eating disordered and college samples might be particularly vulnerable to both more severe eating disorder symptomatology as well as greater weight gain than the general population [12]. Thus, the current study investigated associations among WS and eating disorder-related variables in a population-based sample of female twins, to determine whether these relationships differ for individuals reporting binge eating and loss of control (BE+LOC) vs. those reporting no binge eating (NBE). We chose to examine this form of disordered eating in our population-based sample, as evidence suggests that individuals manifesting subthreshold levels of binge eating report levels of distress comparable to those with binge eating disorder (e.g., [13]).

The current study also estimated genetic and environmental influences on WS. Twin samples are useful for such analyses, as including both monozygotic (MZ) and dizygotic (DZ) twins in a structural equation model allows the variance of a given trait to be partitioned into additive genetic (A), common environmental (i.e., that which is shared among twin pairs; C), and unique environmental (i.e., that which is not shared among twin pairs; E) influences [14]. There are several reasons why twin research could provide insight into WS and related constructs. First, body mass index (BMI) is strongly influenced by genetic factors (e.g., approximately 50–90% of variance in BMI is attributable to additive genetic effects [15]). Second, twin studies of eating disorders have found that AN, BN, and related behaviors (e.g., binge eating and vomiting) are moderately heritable (for a review, see Bulik, et al. [16]). However, twin studies have also indicated that specific disordered eating related behaviors and attitudes are differentially influenced by genetic and environmental factors. For example, Neale et al. [17] found that disinhibited eating was most strongly influenced by additive genetic factors. However, restraint was more strongly influenced by common environmental factors. Similarly, the undue influence of weight on self-evaluation has been found to be more strongly influenced by environmental, rather than genetic, factors [18]. Thus, it is unclear to what degree genetic and environmental factors might influence liability to the specific construct of WS.

In sum, the current study investigated WS among NBE and BE+LOC women in two ways. Regression models investigating relations between WS and eating disorder-related constructs (body dissatisfaction, drive for thinness, Restraint, Disinhibition, Hunger, and time spent dieting before and after age 18) were conducted separately in these two subsamples. We hypothesized that the associations among these variables and WS might differ for women reporting BE+LOC, relative to NBE participants, such that WS would be associated with more pathological behaviors among the BE+LOC group. Second, the genetic and environmental sources of variance were estimated in each subsample. We hypothesized that genetic influences on WS would be stronger in the BE+LOC group, relative to the NBE group, based on results of prior twin studies in the area of eating disorders.

Method

Participants

Twins in this study are from the population-based Virginia Twin Registry (VTR [19]), which now constitutes part of the Mid-Atlantic Twin Registry (MATR). Female-female twin pairs, born between the years of 1934–1974, were eligible if both members had previously responded to a mailed questionnaire, the response rate to which was ~64%. They have been approached for four waves of personal interviews from 1988 to 1997. In late 1999, questionnaires were mailed to all prior participants in these two studies (N=7,230). Only modest resources were available for follow-up, which was largely limited to phone calls to non-responding twins whose co-twin had responded. A total of 2,616 questionnaires were received, representing a 36.2% individual response rate. The present study focuses on 1510 female twins, including female-female MZ and DZ twin pairs as well as female twins with opposite sex twins. Participants' mean age was 40.44 (SD = 8.34) at the time of assessment; all were European American.

Seven women meeting *DSM-IV* criteria for anorexia nervosa (AN) were excluded from analyses. Data from the remaining 1503 individuals were used in the regression models. However, opposite sex DZ twins were not included in biometric models; these analyses included MZ (n=614) and DZ (n=410) same-sex female twins. This research was approved by the Institutional Review Board of Virginia Commonwealth University.

Measures

Weight Suppression—Participants were asked their current weight and highest weight since age 18 (not including pregnancy). Both of these items were included in a biometric model of WS (described below in the Statistical Analyses section). In regression models, a WS variable was created by subtracting current from highest weight, in order to be consistent with previous studies [2,10,11,12].

Body mass index (BMI)—BMI was calculated using participants' self-reported current height and weight.

Eating Disorder Inventory (EDI)—The EDI is a self-report measure consisting of eight subscales, totaling 64 items [20]. The body dissatisfaction subscale (EDI-BD), which consists of nine items, and the drive for thinness subscale (EDI-DT), which consists of seven items, were used in this study. Response options for these items range from 1 = *always* to 6 = *never*. Both subscales yield internally consistent scores (Cronbach's alphas = .91 and .90 for the BD and DT subscales, respectively). Additionally, one-year test-retest reliability estimates are sufficient, with *r*s ranging from .41 to .76 [21]. Criterion-related, convergent, and discriminant validity have been established for these subscales as well [18].

Three- factor Eating Questionnaire (TFEQ)—The current study used a modified form of the TFEQ (as described previously [22]), which utilizes an ordinal response format, compared to the true-false style of the original version [23–24]. This modified version includes 36 of the original items, which comprise the Restraint, Disinhibition, and Hunger subscales. This measure has demonstrated acceptable internal consistency (Cronbach's alpha = .88 [22]).

Dieting—Participants were asked to rate, on a five-point scale, the amount of time they had spent dieting before and since age 18, in order to assess dieting during child- and adulthood.

Binge Eating—Eating disorder symptomatology was assessed in this questionnaire using items based on *DSM-IV* criteria for AN, BN, and binge eating disorder (BED). Diagnostic algorithms used in this sample have been published elsewhere [25]. Participants were categorized as having BE+LOC if they responded affirmatively to each of two questions. The first was "Have you ever had eating binges when you ate what most people would regard as an unusually large amount of food in a short period of time?" The second question, "When you were having eating binges, did you feel that your eating was out of control?" Response options to this question ranged from 0 (*not at all*) to 4 (*extremely*); BE+LOC participants indicated that they experienced feeling a loss of control at least *slightly* while bingeing.

Statistical Analyses

Hierarchical linear regression models were evaluated using Mplus 4.1 [27] to assess the impact of EDI-DT and –BD; TFEQ-Restraint, -Disinhibition, and –Hunger; and dieting before and since age 18 on WS. Age was controlled in the models. The continuous WS variable, representing the difference between participants' highest ever and current weights, was created and served as the dependent variable. Models were conducted separately for participants in the NBE and BE+LOC subsamples. The cluster option was utilized to account for the non-independence of the twin data.

Biometrical modeling, which utilizes twin data to decompose the variance of a given trait or traits into additive genetic (A), common environmental (C), and unique environmental (E) influences, was conducted using Mx [26]. A bivariate model of highest and current weight

was estimated. This bivariate model is superior to a univariate model of WS as a difference score, as difference scores introduce floor effects that could lead to attenuation of parameter estimates. In this bivariate model, the variance of highest and current weights was partitioned into A, C, and E (see Figure 1), and the overall variance components of WS (i.e., the difference between highest and current weight) were calculated using the equation that follows. Three equations were calculated, one each for A, C, and E.

$$a_1^2+a_2^2 - 2a_1a_2r_a$$

These models were estimated separately in the NBE and BE+LOC subsamples.

Results

Descriptives

A total of 275 women were classified as BE+LOC based on their responses to the aforementioned questionnaire items. See Table 1 for correlations among study variables for the NBE and BE+LOC subsamples. The mean of WS scores was 10.75 (*SD* = 14.84) for the NBE subsample and 19.52 (*SD*=24.22) for the BE+LOC subsample. This difference was significant ($T_{130.48} = -3.95$, $p = .00$).

Regression models

Three regression models were fit for the three sets of predictors: 1) EDI-DT and –BD scores, 2) TFEQ-Restraint, -Disinhibition, and –Hunger scores, and 3) the amount of time spent dieting before and after age 18. Results in the NBE subsample indicated that age, Restraint, DT, and time spent dieting before and after age 18 were positively associated with WS, although the standardized regression coefficients were modest (see Table 2). BD had a significant, but small, negative association with WS in this subsample. Only Disinhibition and Hunger were not significantly associated with WS.

Within the BE+LOC subsample, Restraint, Disinhibition, and time spent dieting during childhood were significantly (positively) related to WS. Age, dieting during adulthood, DT, BD, and Hunger were not significantly associated with WS in this subsample.

Biometrical modeling

Within the subsample of NBE women, biometrical modeling results (−2LL=14371.122, df=1626, AIC=11119.122) indicated that 20% (95% CI: .00, .34) of the variance in WS was due to A and 80% (95% CI: .66, .96) due to E; the impact of C was nil (95% CI: .00, .27). Within the subsample of women meeting criteria for BE+LOC, results (−2LL=3779.313, df=369, AIC=3041.313) indicated that 25% of the variance in WS was due to A (95% CI: . 00, .56), 75% (95% CI: .44, 1.0) due to E; C was again estimated at .00 (95% CI: .00, .39).

Discussion

Twin modeling of WS suggested that this construct is moderately influenced by genetic factors and strongly influenced by unique environmental factors; shared environmental factors do not appear to play a significant role in liability to this behavior. The current findings that unique environmental factors contributed significantly to the variance in WS are noteworthy. Although the estimate of unique environment includes error, these results suggest that, while genetic factors do play a role in WS, environmental factors have the strongest impact on this variable. This finding is consistent with data from the National Weight Control Registry, which suggest that long-term weight loss maintenance is

associated with environmental variables or specific behaviors such as regular exercise, frequent self-weighing, and breakfast eating [7,28].

These twin modeling results differ from those obtained in a previous study using the same sample examining genetic and environmental influences on the related construct of Restraint (as measured by a version of the TFEQ [22]). Specifically, Neale et al. [17] found that Restraint was significantly influenced by the shared environment, suggesting that dieting might be a learned behavior. Nonetheless, insofar as our WS variable captures the construct of weight suppression and the Restraint variable reflects the behavior of dieting, the current results combined with those of Neale et al. provide additional support for Lowe's [1] multidimensional model of dieting. Similarly, regression results provided evidence for a modest but significant relation between Restraint and WS. This finding is consistent with results from the National Weight Control Registry, which found that previously obese individuals who lost and kept off a large amount of weight scored high on a measure of Cognitive Restraint [28].

Of note, current findings also suggest that WS might be experienced differently, or reflect different processes, in individuals with and without histories of binge eating. Specifically, although greater cognitive control of eating was associated with WS in the NBE and the BE +LOC subsamples, the absolute value of the relation between Restraint and WS was much greater in the group with disordered eating behaviors. Further, dieting in childhood was most strongly associated with WS within the subsample of women reporting binge eating. The current study was cross-sectional, and cannot address causal relations among variables. However, future studies should extend this research and evaluate whether these childhood dieting experiences might serve as a trigger for problematic WS, as well as disordered eating behaviors, in adulthood.

This study has several strengths, including its use of a large population-based sample. However, limitations of this investigation should also be noted. Specifically, due to the relatively small number of individuals meeting criteria for EDs, we examined differences among non-eating disordered participants as well as those meeting criteria for BE+LOC, rather than threshold eating disorders. All measures were self-report, a common limitation of ED research, particularly when conducted in a large, population-based sample. Finally, the response rate was somewhat low, although similar to that obtained in other twin research (e.g., [29]). It is possible that unmeasured variables influenced participation and, consequently, biased the sample. The current study could not fully explain differences in healthy and unhealthy WS. Importantly, however, results of the current study do suggest that care must be taken in developing health promotion or obesity prevention programs that address the issue of WS. Future studies of successful dieters should investigate behaviors associated with WS and healthy weight loss maintenance. Specifically, research is needed (including individuals with and without ED histories) to clarify ways in which interventions could promote the idea of maintaining a healthy, lower weight, while not (albeit inadvertently) encouraging participants to strive for an unrealistically small body size that might trigger disordered eating in vulnerable individuals.

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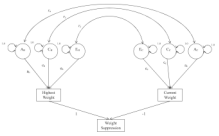

Figure 1.

A Biometric Model of Weight Suppression.

Note: A=variance due to additive genetic influences, C=variance due to common environment, E= variance due to unique environment. A_H, C_H, E_H = variance components for highest weight; A_C, C_C, E_C = variance components for current weight. a_h, c_h, e_h = parameters for highest weight; a_c, c_c, e_c = parameters for current weight. r_a, r_c, r_e=genetic, common environmental, and unique environmental correlations between highest and current weight.

Table 1

Correlations among study variables for the NBE and BE+LOC subsamples.

Measure	1. Age	2. Restraint	3. Disinhibition	4. Hunger	5. EDI-DT	6. EDI-DT	7. WS	8. Diet-child	9. Diet-adult
1	--	.06	.00	.01	−.14*	−.01	.04	−.10*	−.04
2	−.04	--	−.05	−.07	.38*	.01	.14*	.17*	.33*
3	.14	−.26*	--	.71*	.42*	.49*	−.06	.09*	.30*
4	.14	−.23*	.78*	--	.37*	.37*	−.09*	.06	.17*
5	−.10	.38*	.37*	.33*	--	.51*	−.01	.22*	.48*
6	.11	−.06	.46*	.30*	.44*	--	−.05	.15*	.35*
7	.05	.21*	.11	.04	.12	.07	--	.15*	.16*
8	−.02	.16	.20*	.12	.33*	.18*	.21*	--	.46*
9	.03	.29*	.37*	.18*	.48*	.33*	.25*	.38*	--

Note: Correlations for the NBE subsample appear above the diagonal; correlations for the BE+LOC subsample appear below. EDI-DT =Eating Disorder Inventory-Drive for Thinness subscale; EDI-BD = Eating Disorder Inventory-Body Dissatisfaction subscale; WS = weight suppression; Diet-child = time spent dieting before age 18; Diet-adult = time spent dieting after age 18; BMI = body mass index.

* denotes significance at $p < .05$. Correlations were obtained in Mplus, accounting for non-independence of the twin data.

Table 2

Regression coefficients and T-values for independent variables, with WS as the dependent variable.

Measure	NBE women	BE+LOC women
Age	**.08** (*T*=2.89)	.08 (*T*=1.40)
EDI-DT	**.09** (*T*=2.13)	.05 (*T*=.63)
EDI-BD	**−.08** (*T*= −2.02)	.02 (*T*=.33)
R^2	.01	.01
Restraint	**.14** (*T*=4.11)	**.25** (*T*=3.45)
Disinhibition	.02 (*T*=.40)	**.25** (*T*=2.32)
Hunger	−.09 (*T*= −1.89)	−.11 (*T*= −1.05)
R^2	.03	.08
Diet-child	**.10** (*T*=2.20)	**.22** (*T*=2.27)
Diet-adult	**.15** (*T*=3.96)	.12 (*T*=1.90)
R^2	.05	.09

Note: EDI-DT =Eating Disorder Inventory-Drive for Thinness subscale; EDI-BD = Eating Disorder Inventory-Body Dissatisfaction subscale; Diet-child = time spent dieting before age 18; Diet-adult = time spent dieting after age 18; BMI = body mass index. NED=non-eating disordered; ED=eating disordered; Significant coefficients are in bold-face type.