Weight Suppression Is a Robust Predictor of Outcome in the Cognitive–Behavioral Treatment of Bulimia Nervosa

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This study examined weight suppression (difference between highest premorbid weight and pretreatment weight) as a predictor of outcome in 188 outpatients with bulimia nervosa enrolled in a cognitive–behavioral therapy intervention. Participants who dropped out of treatment had significantly higher levels of weight suppression than treatment completers. Of participants who completed treatment, those who continued to engage in binge eating or purging had significantly higher levels of weight suppression than those who were abstinent from bingeing and purging. Results did not change when body mass index, dietary restraint, weight and shape concerns, or other relevant variables were controlled. Relinquishing bulimic behaviors and adopting normal eating patterns may be most feasible for patients who are closest to their highest premorbid weights.

Keywords: weight suppression, bulimia nervosa, dieting, restrained eating, treatment outcome

Weight suppression refers to the discrepancy between an individual’s highest weight ever (since reaching adult height) and his or her present weight (Lowe, 1993). In early writings on bulimia, the discrepancy between patients’ premorbid weight and weight at presentation for treatment was viewed as a major contributor to bulimic psychopathology. In the first systematic description of bulimia, Russell (1979) viewed it as “an ominous variant of anorexia” (p. 429). Garner and Fairburn (1988) also suggested that the unwillingness of patients with bulimia to return to their premorbid weights may contribute to their persistent binge eating and purging.

Evidence indicates that individuals with bulimia typically have substantially higher premorbid weights than do those with anorexia. In two large samples of patients with bulimia who were currently in the low–normal weight range, Garner and Fairburn (1988) found that approximately one third of patients in one sample and more than half of patients in the other sample had previously been more than 15% overweight. Murphy-Eberenz (2000) found that 98 inpatients with anorexia reported a highest weight ever of 119 lb (53.98 kg), whereas 124 inpatients with bulimia reported a highest weight of 157 lb (71.21 kg). The data on previous highest weights in these studies were based on patients’ self-reports. However, the fact that patients with anorexia are much thinner than patients with bulimia supports the validity of these data showing that patients with anorexia also had much lower body mass indexes (BMIs; weight in kilograms/height in meters squared) than patients with bulimia before both groups lost weight.

In the process of developing their disorder, many patients with bulimia lose as much weight as patients with anorexia (Garner & Fairburn, 1988). However, by the time individuals with bulimia present for treatment, they are generally in the normal weight range (Wilson, Fairburn, & Agras, 1997). Nonetheless, they are on average still well below their highest adult weights. For example, Murphy-Eberenz (2000) found the average degree of weight suppression in a sample of patients with bulimia to be approximately 15 kg.

Because the body weights of patients with bulimia are generally in the normal range, there is typically no attempt made to modify body weight during treatment. Among outpatients there is little change in body weight during cognitive–behavioral therapy (CBT) for bulimia (Wilson et al., 1997) despite the substantial reduction in bulimic symptoms associated with this treatment. However, as Russell (1979) noted in his early commentary on bulimia nervosa, simply because weight at presentation for treatment is within the range of normality does not mean that it is clinically unimportant, particularly if the discrepancy between that weight and premorbid weight is large.

There are many reports of abnormally low caloric intake and nutritional insufficiencies in patients with bulimia (e.g., Gendall, Sullivan, Joyce, Carter, & Bulik, 1997; Kirkley, Agras, & Weiss, 1985). The evidence for reduced metabolic rate in individuals with bulimia is mixed (for a review, see de Zwaan, Aslam, & Mitchell, 2002), but several studies have found that the metabolic rate of successfully treated individuals with bulimia is abnormally low (e.g., Gwirtsman et al., 1989; Obarzanek, Lesem, Goldstein, & Jimerson, 1991; Spalter, Gwirtsman, Demitrack, & Gold, 1993) and declines after treatment (e.g., Leonard et al., 1996). Thyroid hormones, which help regulate metabolic rate, also tend to be low in these women (Gendall, Joyce, Carter, McIntosh, & Bulik, 2003; Obarzanek et al., 1991; Spalter et al., 1993).

Despite data suggesting that weight suppression may be an important variable for understanding the symptoms and risks associated with bulimia, this variable has been subjected to little...
empirical investigation. Previously obese participants in the National Weight Control Registry lost substantial weight and kept it off (Wing & Hill, 2001), thereby fulfilling the definition of weight suppression. However, a prospective study found that these weight suppressors were highly susceptible to weight regain (Wing & Hill, 2001). In two recent studies, it was found that weight suppression prospectively predicted weight gain during the freshman year of college (Lowe, Annunziato, et al., 2006) and prospectively predicted amount of weight gained by patients with bulimia during a psychiatric hospitalization (Lowe, Davis, Lucks, Annunziato, & Butryn, in press). These findings may be due in part to the fact that weight reduction increases metabolic efficiency (Leibel, Rosenbaum, & Hirsch, 1995), thereby increasing risk for future weight gain.

In sum, there are several reasons to hypothesize that weight suppression among individuals with bulimia may represent an obstacle to the successful treatment of this disorder. Weight suppression might increase the risk of disinhibited or binge eating (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950), increase metabolic efficiency and the storage of ingested energy as body fat (Leibel et al., 1995), or both, thereby rendering those highest in weight suppression most susceptible to future weight gain.

In the present study, we examined weight suppression as a prospective predictor of treatment outcome in a previously published study of CBT for bulimia (Agras et al., 2000). We predicted that individuals with bulimia nervosa who were higher in weight suppression before beginning treatment would show poorer treatment outcome. Because the results supported this hypothesis, we used a number of additional eating-disorder-related variables as covariates to help determine whether weight suppression per se was responsible for the outcomes observed.

Method

Participants

The description of participants was adopted from the original article reporting on predictors of outcome in this sample (Agras et al., 2000). Participants were 188 women enrolled in a multisite study of predictors of outcome in CBT for bulimia nervosa. Six potential participants were dropped from the original studies for a variety of reasons (see Agras et al., 2000). Participants met Diagnostic and Statistical Manual of Mental Disorders (3rd ed., rev.; American Psychiatric Association, 1987) diagnostic criteria for bulimia nervosa. Data on exclusion criteria and descriptive information on the sample are available in the original report (Agras et al., 2000).

Treatment

Participants received 18 sessions of CBT conducted by doctoral-level psychologists using a common treatment manual (Fairburn, Marcus, & Wilson, 1993). Details of the treatment, staff training, and monitoring are in the original report (Agras et al., 2000).

Assessment

Participants completed several assessments relevant to the current study. Weight and height were measured. Information on participants’ previous highest and lowest weights at their current height, not due to pregnancy or illness, was gathered by self-report. The validity of recalled past weights has been supported by a study that found a correlation of .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 in these participants was just 1.28 kg, suggesting that the absolute size of the error in recalled weights was small. Also, Swenne (2001) retrieved historical measured weights in girls before they developed an eating disorder and found, in line with the data on highest previous weights reported here, that these girls’ premorbid relative weights were higher than those of age-matched girls in the general population.

In the current study, weight suppression was defined as the difference between participants’ previous highest self-reported weight and pretreatment measured weight.

Participants completed the Three-Factor Eating Questionnaire (Stunkard & Messick, 1985), a self-report measure of cognitive restraint, disinhibition, and hunger. The Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a semistructured interview, was also conducted to assess eating-related pathology. These measures have demonstrated acceptable reliability and validity (for details, see the original report of this study by Agras et al., 2000).

Statistical Analysis Plan

To examine the relationship between weight suppression and treatment outcome, we examined two outcome variables. First, treatment completion was examined as a binary outcome variable (completing treatment vs. discontinuing treatment). Second, abstinence from bingeing and purging in the final 4 weeks of treatment was examined as a binary outcome variable (abstinent vs. nonabstinent) in those participants who did complete treatment. (Number of episodes of bingeing and purging was measured with the EDE.) Logistic regressions were conducted for each outcome variable to determine whether weight suppression predicted outcome and, if it did, to examine whether this outcome changed when several other variables were controlled.

Results

Descriptive Statistics

At pretreatment, participants had a mean BMI of 23.5 (SD = 4.5) and a mean weight of 63.8 kg (SD = 12.8). On average, participants’ highest lifetime BMI was 27.0 (SD = 5.1), and their highest weight was, on average, 72.9 kg (SD = 14.3). Thus, the mean pretreatment weight suppression was 3.5 BMI units (SD = 3.6) or 9.4 kg (SD = 9.5).

The 18-week treatment was completed by 140 participants (i.e., 74%), whereas 48 participants (i.e., 26%) prematurely discontinued treatment. Of those who completed treatment, 58 participants (i.e., 41% of completers) were abstinent from bingeing and purging at treatment completion, whereas 82 participants (i.e., 59% of completers) continued to binge or purge at treatment completion. As shown in Table 1, participants who completed treatment had a mean pretreatment weight suppression of 6.6 kg (SD = 7.3), whereas participants who discontinued treatment had a mean weight suppression of 17.7 kg (SD = 1.4). Of participants who completed treatment, those who were abstinent at posttreatment had a mean weight suppression of 4.2 kg (SD = 4.5), whereas those who continued to binge or purge at posttreatment had a mean weight suppression of 8.3 kg (SD = 8.4). Level of weight suppression in each outcome group is illustrated in Figure 1.

Weight Suppression as a Predictor of Treatment Completion

Logistic regression indicated that weight suppression at pretreatment was a significant predictor of treatment completion, B =
Weight, Weight Suppression (WS), and Body Mass Index (BMI) by Treatment Outcome Group

Table 1

<table>
<thead>
<tr>
<th>Outcome group</th>
<th>WS (kg)</th>
<th>Highest weight (kg)</th>
<th>Pretreatment weight (kg)</th>
<th>Highest BMI (kg/m²)</th>
<th>Pretreatment BMI (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Completer: abstinent (n = 58)</td>
<td>4.2</td>
<td>71.5</td>
<td>67.5</td>
<td>26.4</td>
<td>24.9</td>
</tr>
<tr>
<td>Completer: nonabstinent (n = 82)</td>
<td>8.3</td>
<td>69.8</td>
<td>61.5</td>
<td>26.0</td>
<td>22.9</td>
</tr>
<tr>
<td>Dropout (n = 48)</td>
<td>17.7</td>
<td>79.9</td>
<td>62.2</td>
<td>29.6</td>
<td>23.0</td>
</tr>
</tbody>
</table>

-0.37, SE = 0.07, p < .01, Exp(B) = 0.69. On average, for each increase of 1 kg of weight suppression, the odds of not completing treatment are multiplied by a factor of 0.69. Thus, if a patient with bulimia is weight suppressed by 11 kg (the mean difference between treatment completers and noncompleters), we would estimate that that patient would be 7.59 times (11 × 0.69) less likely to complete treatment than a patient who was not weight suppressed.

To determine whether other variables might account for the relationship found between weight suppression and treatment outcome, we repeated the logistic regression while controlling for several variables: baseline BMI, duration of binge eating, Three-Factor Eating Questionnaire—Restraint, EDE—Restraint, EDE—Weight Concern, EDE—Shape Concern, EDE—Eating Concern, EDE—Objective Binge Eating Episoses, and EDE—Purging Episodes. We conducted individual logistic regressions while controlling for each variable. In each case, weight suppression remained a significant (i.e., p < .05) predictor of treatment completion. None of the individual covariates accounted for significant variance in the prediction of treatment completion (all ps > .05). One larger logistic regression also was conducted in which each of the covariates was entered simultaneously. In this multivariate regression equation, treatment completion was significantly predicted by weight suppression, B = −0.17, SE = 0.03, p < .01, Exp(B) = 0.85. The only other significant predictor in this multivariate model was EDE—Shape Concern scores, B = −1.01, SE = 0.34, p < .01, Exp(B) = 0.36; those with greater shape concerns had lower odds of completing treatment.

Through the use of the Cox and Snell R², the variance accounted for in treatment completion status could be calculated for each of these logistic regression models. The variance accounted for by weight suppression when entered alone was 22%, by all of the variables except for weight suppression was 11%, and for weight suppression and the other variables together was 31%.

**Weight Suppression as a Predictor of Abstinence From Bingeing and Purging**

Logistic regression indicated that weight suppression at baseline also was a significant predictor of abstinence from bingeing and purging. B = −0.14, SE = 0.04, p < .01, Exp(B) = 0.87. On average, for each increase of 1 kg of weight suppression, the odds of not abstaining from bingeing and purging at treatment completion are multiplied by a factor of 0.87. Each of the variables that was controlled for in the treatment completion logistic regressions was entered in the same manner into logistic regression equations for the prediction of abstinence. In each case, weight suppression remained a significant (i.e., p < .05) predictor of abstinence from bingeing and purging. In the multivariate regression equation in which all of the above variables were entered as covariates, abstinence from bingeing and purging was not significantly predicted by any other variables, and weight suppression remained a significant predictor.

Through the use of the Cox and Snell R², the variance accounted for in abstinence status could be calculated for each of these logistic regression models. The variance accounted for by weight suppression when entered alone was 12%, by all of the variables except for weight suppression was 5%, and for weight suppression and the other variables together was 16%.

**Weight Suppression Versus Weight Fluctuation**

One additional alternative explanation for these results is that weight suppression is simply a proxy of weight fluctuation (i.e., that the discrepancy between highest historical and current weights is predictive only because it is a marker of individuals who have frequently gained and lost large amounts of weight). To address this possibility, the difference between participants’ lowest weights (at current height) and current weights was calculated (discrepancy between lowest and current weights: M = 11.5 kg, SD = 9.6 kg). When entered in logistic regressions, this value alone was not predictive of treatment completion or abstinence from bingeing and purging (both ps > .05), nor was it predictive...
when entered simultaneously with weight suppression. In the latter regressions, weight suppression remained a significant predictor of both treatment completion and abstinence from bingeing and purging. Finally, when highest past weight was entered as a covariate to determine whether obesity proneness (as indexed by highest past weight) could account for the predictive effects of weight suppression, weight suppression remained a significant predictor of both treatment completion and abstinence from bingeing and purging.

Discussion

The purpose of this study was to determine whether weight suppression predicted treatment outcome during outpatient CBT for bulimia nervosa. The results indicated that higher levels of weight suppression were associated with lower likelihood of completing treatment. Among those who completed treatment, weight suppression was associated with a lower likelihood of abstaining from binging and purging when these variables were controlled. Each covariate selected could theoretically account for the relationship between weight suppression and outcome. For instance, it could be hypothesized that weight suppression is associated with the dimension of weight and shape concern, which has been hypothesized to be the core psychopathology in bulimia nervosa (Wilson et al., 1997). Because it is logical to expect that participants with greater weight or shape concerns might be suppressing their weight furthest below its greatest previous level, we used the Weight Concern and Shape Concern subscales from the EDE as covariates. We also used the difference between lowest previous weight and current weight as a covariate to determine whether this proxy of weight fluctuation was predictive of outcome. However, weight suppression remained a significant predictor of treatment outcome when each of these potentially confounding variables was accounted for. Although it is possible that another variable could be responsible for the predictive value of weight suppression, this study accounted for many of the constructs that could be suggested as alternative explanations for the results.

Given the conclusion that elevated levels of weight suppression increase the risk of poor outcome during outpatient CBT for bulimia nervosa, it is important to determine why weight suppression might have this effect. It is possible that weight suppression itself could interfere with the patient’s ability to comply with CBT. For instance, the most powerful component of the CBT package is the rapid reduction in restrained eating that occurs early in treatment during which reductions in dieting and normalization of food intake are targeted (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002). These changes in eating patterns could have actual effects on energy balance that would result in weight gain, or they could be perceived by patients to be likely to produce that effect. Weight-suppressed individuals with bulimia nervosa may resemble previously obese individuals in that their metabolic state could be more inclined toward energy storage relative to energy utilization (Leibel et al., 1995). For example, a formerly obese person who lost 10 kg and is concerned about regaining weight and a patient with bulimia whose current weight is 10 kg below her highest historical weight and who fears gaining weight may have physiologically similar bases for their worries, although clinicians may perceive their presentations as quite dissimilar. However, weight suppression has been associated with accelerated weight gain among the successful dieters in the National Weight Control Registry (Wing & Hill, 2001) and in inpatients entering treatment for bulimia (Lowe et al., in press). Because their weight loss may have created a more efficient metabolic state and enhanced their tendency to store ingested energy as body fat (Nicklas, Rous, & Goldberg, 1997), individuals who are high in weight suppression who try to normalize their eating may have fears of weight gain that are at least partially grounded in reality.

As for energy intake, it is possible that the requirement that CBT imposes of normalizing eating (e.g., by eating a wider variety of foods) and consuming meals and snacks several times a day (even when the meals and snacks are small) intensifies bulimic patients’ fears of overeating and weight gain. Again, given the role of dramatic weight loss and weight suppression in the genesis of binge eating (Garner & Fairburn, 1988; Keys et al., 1950; Russell, 1979), this fear may be well-founded, especially in those patients who are most weight suppressed. The foregoing observations suggest that future research with individuals with bulimia should measure not only concerns about weight and shape but also fears that substantial weight will be gained if eating is normalized and purging is eliminated. The current results suggest that such fears would be positively related to degree of weight suppression.

The CBT model conceptualizes individuals with bulimia nervosa as caught in a psychological bind: An overemphasis on weight and shape as determinants of self-worth produces extreme weight control behaviors and binge eating, which in turn undermine body satisfaction and self-esteem. This bulimic cycle causes feelings of helplessness and shame and threatens self-esteem, a feedback loop that further reinforces dieting behaviors (Wilson et al., 1997). There is a substantial amount of support for this model (Wilson et al., 1997, 2002).

However, it may be more accurate to suggest that individuals with bulimia nervosa are caught in a psychobiological bind. Individuals with bulimia nervosa have premorbid relative weights that are higher than those of their nonbulimic peers, and they are also more likely to have one or both parents who are overweight (Fairburn, Welch, Doll, Davies, & O’Connor, 1997; Garner & Fairburn, 1988). This tendency toward overweight may be part of what spurs a radical weight loss diet in many individuals with incipient bulimia. This attempted “solution” is very problematic because whether a predisposition to weight gain is behavioral or metabolic in nature, the major diet-induced weight loss that most individuals with incipient bulimia undergo would be expected to worsen, rather than improve, whatever behavioral or metabolic predisposition toward weight gain existed in the first place. Thus, returning to normal eating without gaining weight may be particularly difficult for patients highest in weight suppression. In sum, large weight losses may not only initiate binge eating but also help maintain binge eating even when the full bulimic syndrome has developed.

The hormonal abnormalities often seen in bulimia are consistent with the hypothesis that patients with bulimia are suppressing their weight below a biologically appropriate level. Bulimic patients
with the lowest caloric intakes and the lowest body weights tend to show the greatest disturbances in neuroendocrine function (Fichter & Pirke, 1995). When these patients stop binging and purging, most neuroendocrine indices normalize, but some do not (Fichter & Pirke, 1995). In addition, a study by Frank, Kaye, Ladenheim, and McConaha (2001) found that levels of the neuropeptide gastrin releasing peptide remained significantly lower in long-term recovered (i.e., regular menstrual cycles, normal weight, and no binging or purging for longer than 1 year) patients with bulimia compared with healthy control women. Frank et al. concluded that these persistent gastric releasing peptide abnormalities after recovery from bulimia may contribute to episodic binge eating in these women. Taken together, the aforementioned findings suggest that weight suppression could represent an obstacle to successful treatment, could heighten risk for relapse, and could help sustain unhealthy levels of restrained eating even among successfully treated patients with bulimia (Safer, Agras, Lowe, & Bryson, 2004).

Further research with bulimic individuals should be conducted to determine the extent to which the hormonal and metabolic abnormalities that are often observed in these patients may be a function of weight suppression. Studies of abnormalities in eating regulation in patients differing in weight suppression are also needed. Studies of the duration of past highest weight and recentness of weight loss in weight-suppressed patients would be valuable as well.

It would be useful to determine how weight-suppressed patients who drop out of treatment or continue to binge or purge explain their behavior. One hypothesis is that they have formed the belief, given their high premorbid weights, that if they eat normally (i.e., without binging and/or purging), they will gain weight. They may believe that purging is necessary to maintain a lower body weight. A belief in the effectiveness of the binge/purge cycle is likely illusory, given evidence that, on average, over 1,000 kcal are retained after binging and purging (Kaye, Weltzin, Hsu, McConaha, & Bolton, 1993). The belief that such behaviors will prevent the weight gain that weight-suppressed patients have previously experienced may be an important target for treatment. More generally, it is important to determine how CBT may need to be modified in light of evidence that significant weight suppression may be a major impediment to successful treatment outcome.

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