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Responses to Weight Loss Treatment Among Obese Individuals with and without BED: A Matched-study Meta-analysis

Bruce E. Blaine
St. John Fisher College, bblaine@sjfc.edu

J. Rodman

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Abstract

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Keywords

Binge eating, weight loss, obesity, depression

Disciplines

Health Psychology | Statistics and Probability

REVIEW ARTICLE

Responses to weight loss treatment among obese individuals with and without BED: A matched-study meta-analysis

B. Blaine*, and J. Rodman**

*Psychology Department, St. John Fisher College, Rochester, and **Hofstra University, Hempstead, NY, USA

ABSTRACT. *The moderating influence of binge eating status on obese individuals' responses to weight loss treatment was evaluated with a meta-analysis of 36 tests of weight loss treatment (n=792) that were matched to control key background variables. After controlling for pre-treatment weight, treatment produced more weight loss in samples of obese non-BED compared with obese BED participants. Weight loss treatment produced large post-treatment reductions in depression in both obese BED and non-BED samples. The results indicate that BED status moderated post-treatment weight loss among people in weight treatment programs. Obese BED (average weight loss= 1.3 kg) samples lost negligible weight compared to obese non-BED (average weight loss= 10.5 kg) samples. BED status did not moderate psychological responses to treatment: both BED and non-BED samples experienced large post-treatment reductions in depression. The clinical implications of these findings are discussed.*

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INTRODUCTION

The prevalence of obesity – defined as having a body mass index (BMI) greater than 30 kg/m^2 – among adults is about 30% in the US and slightly less in the UK (1, 2). Rapid increases in the prevalence of obesity have also occurred in Latin American and European countries (3, 4). Given the serious health risks that are associated with obesity, effective weight loss treatments and programs are in great demand. Research suggests however that large scale, permanent weight loss is rare (5). Even modest weight losses are difficult to sustain, as over 80% of people who diet for the purposes of weight loss regain most or all of the lost weight within a year (6). A recent meta-analytic review of weight loss treatments among obese participants found that weight loss treatment produced long-term weight loss that averaged about 7% of the average participant's weight (7). Despite this modest weight loss, the review also found that weight loss treatment had robust psychological benefits. Obese participants in weight loss treatments realized significant post-treatment reductions in depression, and this occurred even when participants lost little or no actual weight.

One question left unaddressed by that review concerns the moderating influence of subjects' binge eating status on their responses to weight loss treatment. This is an important clinical question because many obese people who seek weight loss treatment have clinical or sub-clinical levels of binge eating disorder (BED) and they may respond to treatment much differently than obese people without BED. Binge eating – eating a greater volume of food than most other people in a short period of time while experiencing a loss of control and psychological distress over the eating episode – is common among obese people (8). BED, which involves chronic, recurrent, and disruptive binge eating, occurs in about 10% of obese people (9). However, BED is estimated to occur in about 30% of obese people in weight loss programs (10), hence the need to understand how obese BED and non-BED individuals respond to weight treatment.

Much research shows that depressive disorders are more common among obese BEDs than among comparable non-BEDs (11-14). Research finds that obese BED compared with obese non-BED people are

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Correspondence to:
Bruce E. Blaine, Ph.D., Psychology Department, St. John Fisher College, 3960 East Avenue, Rochester, NY 14618
E-mail: bblaine@sjfc.edu

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more likely, perhaps as much as six times more likely, to have a current or past diagnosis of major depression. Similarly, 51% of obese BEDs compared with 14% of obese non-BED subjects met the criteria for major depression (15-17). Obese BED compared to obese non-BED people are also more likely to meet the diagnostic criteria for dysthymia, or minor depression (15). Measures of subclinical levels of depressive symptoms are consistent with these findings, with obese BED compared to obese non-BED subjects consistently reporting higher depression and lower self-esteem (18-21). Even within the obese BED subpopulation, where restricted variability in binge eating should attenuate the correlation between binge eating and depression, increases in binge eating scale scores predict greater depression (22).

The co-morbidity of BED and depression is relevant to the current investigation because of evidence showing that depression is more likely a cause than a consequence of changes in one's weight. In a large cohort study of over 9,000 adolescent subjects researchers found that although depression and weight were unrelated at baseline, depression predicted weight one year later after controlling for a set of demographic and behavioral variables (23). Even among those who were not obese to begin with, depressed subjects were over two times more likely than nondepressed subjects to be obese a year later. Other research shows that adolescent depression is a better predictor of adult obesity than adolescent obesity (24). Still other research suggests that depression predicts diet relapse (25). If depression causes weight gain then obese BEDs should fare worse in obesity treatment and, compared with obese non-BEDs, should lose less weight in weight treatment programs.

To summarize, the greater levels of clinical and subclinical depression in obese BED compared with obese non-BED people suggest that obese BED people should respond more negatively to weight treatment than obese non-BEDs. Although many tests of weight treatment in the BED subpopulation have been conducted, no quantitative review of these studies exists. In this study we explored the influence of BED status on the effectiveness of weight loss treatment via a matched-study meta-analysis. This method combines the control of a matched-subject design with the statistical power of meta-analysis. Weight treatment studies with obese BED participants were matched with similar studies using obese non-BED participants such that sample size, the percentage of females in the sample, sample age, treatment type, and pre-treatment BMI were controlled. The purpose of this study was to quantify the differences between obese BED and non-BED individuals' responses to weight loss treatment and provide an empirical basis for commenting on the efficacy of treatment for these distinct clinical subpopulations. In addition, we sought to validate the role of depression in obese BEDs' responses to weight treatment by controlling alternative factors that covary with BED status in weight treatment programs.

METHOD

Article identification and matching procedure

Articles were identified from a larger review of weight loss treatment effects in obese people. The search parameters for that search, which yielded 117 weight loss treatment studies, are published and available (7). From those used in the Blaine et al. (7) review, studies were included in this review if they evaluated a weight loss treatment, had a BED grouping variable, and recorded treatment effects on either weight or depression. Studies were excluded if they evaluated a treatment for binge eating rather than weight loss.

This is not a typical meta-analysis; we have not exhaustively searched the literature for all available weight treatment studies with obese BED and obese non-BED subjects. The goal of the present method – a matched-study meta-analysis – was control. We sought to isolate the effect of BED on responses to weight treatment by matching obese BED and obese non-BED samples on key variables that covary with BED status. This matching procedure was done to rule out alternative explanations for why obese BED compared with obese non-BED people might respond differently to weight treatment. By combining effect sizes across these matched study pairs, however, we generated considerable statistical power for this analysis.

The matching procedure started with a BED sample and paired it with a non-BED sample with comparable sample size, percentage of females in the sample, sample mean age, treatment type, and mean sample pre-treatment BMI. When studies included both BED and non-BED samples in their weight treatment program, the non-BED sample was usually the best match for the BED sample. In studies that tested weight treatment in a sample of BED subjects without a non-BED control group, the matching non-BED sample was found from another article. When two levels of BED status were included in a study (e.g., moderate and

severe BED samples), matching non-BED samples were found for each. The pairs of matched studies are presented in Table 4. Of the 16 BED samples, 10 were identified using scores on the Binge Eating Scale (26). The remaining 6 samples identified BED subjects with the Questionnaire on Eating and Weight Patterns – Revised (11), which contains items that assess DSM-IV diagnostic criteria for BED, or an ad hoc DSM-IV diagnostic criteria-based measure.

Article coding

Articles were coded for the following variables: sample size, percentage of females in the sample, sample mean age, treatment duration, treatment type (whether the treatment was generally drug/surgery-based or psychotherapy-based), sample mean pre-treatment BMI, sample mean pre-treatment depression (BDI or Beck Depression Inventory) score, and percentage of attrition from treatment. Effect size statistics were recorded for the effect of treatment on post-treatment weight loss and depression. Effects size statistics reported in the articles (typically pre- and post-treatment means/SDs or a t, F, or p level from a pre- vs post-treatment significance test) were converted to rs for analysis. The effects were analyzed with Meta-analysis 5.3 (27).

RESULTS

The data from 16 obese BED samples (total n=388) and 16 matched obese non-BED samples (total n=465) were analyzed. Of these 16 matched sample pairs 13 reported effect sizes for both post-treatment weight loss and depression; the remaining 3 studies reported effect sizes on one outcome (see Table 4 for descriptive statistics for studies in review). To determine if the BED and non-BED studies were equivalent on the matched variables, significance tests compared sample size, females as a percentage of the sample, sample age, treatment duration, and pretreatment sample BMI scores across the sample type (Table 1). These results show that the matching procedure was effective: BED and non-BED samples were not significantly different on each of the matched variables. Pre-treatment BDI, treatment duration, and attrition rate were not matched variables but were nonetheless compared across sample type. The results (Table 1) show that the treatment duration and attrition rates for obese BED compared to non-BED samples were also not significantly different. However, BED samples were significantly more depressed before treatment than non-BED samples. In addition to the matched variables, then, these analyses control treatment duration and attrition as possible artifacts of group differences in response to weight treatment. The data also contradict findings that BED is a risk factor for attrition in weight treatment programs (31).

Weight loss

Summary statistics for the effects of weight loss treatment on weight separated by sample type are presented in Table 2. Weight loss treatment produced weight loss in both BED and non-BED samples, but the size of the treatment effect on weight in non-BED samples in standard deviation units (g= -1.48) was nearly four times the size of the effect in BED samples (g= -0.40. These effect sizes are equivalent to average weight losses of 3.4 lbs (1.5 kg) and 23.5 lbs (10.7 kg) in BED and non-BED samples, respectively. Estimating from the average body mass indexes for each group listed in Table 1 this represents an average post-treatment weight loss of about 2% and 11% of body weight in obese BED and obese non-BED samples, respectively. These results are striking given that pretreatment weight, treatment duration, treatment type, and attrition – each a plausible alternative explanation for the group difference – were controlled.

The homogeneity test of sample effect sizes in the non-BED samples indicated the likely influence of a moderating variable or variables. The possible moderating influence of treatment type was dismissed upon inspecting

TABLE 1
Comparison of subject characteristics in BED and matched non-BED samples in weight loss treatment studies.

	Sample type		
	BED	**non-BED**	**t-test of mean diff. (2-tailed)**
Sample size (n)	21.0 (20.5)	30.9 (33.8)	p=0.17
Percentage female	96.8 (7.1)	96.0 (7.1)	p=0.88
Age (years)	41.1 (3.6)	40.8 (3.2)	p=0.61
Pretreatment BMI (kg/m^2)	35.1 (5.7)	36.2 (5.7)	p=0.62
Treatment type:			
DS studies	3	3	
PT studies	13	13	
Treatment duration (months)	3.6 (1.3)	5.3 (4.1)	p=0.12
Attrition rate	20.8% (18.5)	18.0% (17.7)	p=0.66
Pre-treatment BDI	15.1 (3.8)	8.8 (2.3)	p<0.01

DS= drug/surgery-based treatment; PT= psychotherapy-based treatment; BMI= body mass index; BDI= Beck Depression Inventory.

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TABLE 2
Summary meta-analytic statistics for the effects of weight loss treatment on weight loss and depression in obese BED and obese non-BED samples.

	BED samples		non-BED samples	
	Weight loss	Depression	Weight loss	Depression
k	15	14	15	14
n	329	279	463	319
Unweighted mean r	-0.22	-0.40	-0.40	-0.42
Pop. effect size (weighted r)	-0.20	-0.42	-0.59	-0.46
95% CI of weighted r	-0.21 to -0.19	-0.43 to -0.41	-1.1 to -0.10	-0.47 to -0.45
Mean standardized diff. (g)	-0.40	-0.92	-1.48	-1.05
Percent of observed variance due to sampling error	100%	100%	18%	100%
Test of homogeneity (χ^2)	p=0.54	p=0.58	p<0.001	p=0.42

k= number of samples, n= total sample size across k samples, CI= confidence interval, χ^2 df= k-1.

the effect sizes for psychotherapy-based (PT) compared to drug/surgery-based (DS) treatment effects. The heavily overlapping range of effect sizes in PT (0.00 to -0.90) and DS (-0.23 to -0.82) samples did not suggest treatment type moderation. The moderating influence of pre-treatment sample BMI was explored by first separating samples by the median BMI (36 kg/m^2) to create two groups of studies of roughly equal size. In seven out of the eight moderately obese (BMI<36 kg/m^2) samples weight effect sizes ranged -0.02 to -0.30, whereas in five of the seven extremely obese (BMI≥36 kg/m^2) samples the effect sizes ranged from -0.51 to -0.90. Based on these non-overlapping ranges, weight effect sizes in non-BED samples were meta-analyzed separately by BMI sample type. The results (Table 3) confirm the moderating influence of pre-treatment obesity level: the average post-treatment weight loss in extremely obese samples was over three times larger than in moderately obese samples. Was this effect an artifact of attrition? The attrition rates of moderately (12.1%) and extremely (18.2%) obese non-BED samples were not significantly different and in the wrong direction to account for the greater weight loss among the most obese samples.

Depression

Summary statistics for the effects of weight loss treatment on depression separately by sample type are presented in Table 2. Weight loss treatment produced significant and large reductions in depression in both BED and non-BED samples, with effect sizes around one standard deviation in both groups. In clinical terms this effect size is equivalent to an average reduction of depression by 3.3 and 2.6 BDI points for BED and non-BED samples, respectively. Given that pre-treatment BDI was not controlled through the study-matching procedure used in this review, and thus covaries with BED status, perhaps the slightly smaller treatment effect on depression among non-BED samples is due to a floor effect in those studies. The pre-treatment BDI means in Table 1 suggest otherwise: non-BED samples were moderately depressed at the start of treatment (average BDI score= 9.2) with plenty of room for reductions of the magnitude observed in the BED samples. Thus, it does not appear that the modest difference between BED and non-

TABLE 3
Summary meta-analytic statistics for the effects of weight loss treatment on weight loss in moderately and extremely obese non-BED samples.

	Moderately obese (BMI<36)	Extremely obese (BMI≥36)
k	8	7
n	125	338
Unweighted mean r	-0.32	-0.55
Pop. effect size (weighted r)	-0.31	-0.72
95% CI of weighted r	-0.32 to -0.30	-1.08 to -0.35
Mean standardized diff. (g)	-0.65	-2.06
Percent of observed variance due to sampling error	100%	12%
Test of homogeneity (χ^2)	p=0.66	p<0.01

k= number of samples, n= total sample size across k samples, CI= confidence interval, χ^2 df=k-1, BMI= body mass index.

BED samples' post-treatment depression is an artifact of pretreatment differences in depression between BED and non-BED samples.

Did treatment effects on depression depend on having lost weight? Post-treatment weight loss and depression effects were uncorrelated ($r(25)=0.03$, $p>0.05$) indicating that the benefits of treatment for weight and depression were independent. This is consistent with the findings of a larger review of weight treatment studies in which post-treatment reductions in depression occurred even in samples with little

TABLE 4
Descriptive statistics and effect sizes of reviewed studies presented in matched-study pairs.

Study #/Study (ref)	Sample type	n	% female	Age	Pre-BMI	Pre-BDI	Treat. dur.	Attrit. rate	Treat. type	WL	Dep
1A Porzelius et al. (28)	BED	10	100	38	31	7.2	4	10%	PT	-0.04	0.12
1B Porzelius et al. (28)	non-BED	8	100	38	31	10	4	12%	PT	-0.27	-0.53
2A Porzelius et al. (28)	BED	8	100	38	31	10	4	12%	PT	-0.27	-0.15
2B Porzelius et al. (28)	non-BED	8	100	38	31	7.5	4	12%	PT	-0.23	-0.64
3A Jirik-Babb & Geliebter (18)	BED	21	100	44	31	10.1	1	67%	PT		-0.25
3B Jirik-Babb & Geliebter (18)	non-BED	22	100	44	35	4.6	1	64%	PT		-0.19
4A Malone & Alger-Mayer (29)	BED	31	79	44	48	12	6	42%	DS	-0.43	
4B Malone & Alger-Mayer (29)	non-BED	52	79	46	48	9	6	52%	DS	-0.51	
5A Alger et al. (30)	BED	17	82	45	40	12.7	3	0%	DS	-0.18	-0.50
5B Alger et al. (30)	non-BED	16	88	45	37	10	3	6%	DS	-0.23	-0.53
6A Porzelius et al. (28)	BED	9	100	38	31	14.5	4	22%	PT	-0.30	-0.27
6B Marcus et al. (31)	non-BED	17	100	39	31	7.7	2.5	18%	PT	-0.27	-0.55
7A Agras et al. (32)	BED	93	100	46	37	14.9	9	18%	PT	0.00	-0.34
7B Gladis et al. (33)	non-BED	50	100	41	36	11.3	6	14%	PT	-0.81	-0.60
8A Porzelius et al. (28)	BED	11	100	38	31	15	4	18%	PT	-0.15	-0.40
8B Sbrocco et al. (34)	non-BED	12	100	39	33	6.75	12	8%	PT	-0.67	-0.14
9A Fossati et al. (35)	BED	23	100	42	34	16	3	3%	PT	-0.24	-0.51
9B Wadden et al. (22)	non-BED	21	88	43	36	10.1	3	19%	PT	-0.60	-0.38
10A Fossati et al. (35)	BED	25	100	37	36	16	3	3%	PT	-0.45	-0.71
10B Wadden et al. (22)	non-BED	28	100	37	40	13.2	3	18%	PT	-0.90	-0.32
11A Marcus et al. (31)	BED	17	100	39	31	17.5	2.5	23%	PT	-0.27	-0.59
11B Rippe et al. (36)	non-BED	40	100	37	32		3	25%	PT	-0.30	-0.41
12A Marcus et al. (31)	BED	18	100	39	31	17.5	2.5	28%	PT	-0.21	-0.59
12B Marcus et al. (31)	non-BED	17	100	39	31	7.7	2.5	0%	PT	-0.18	-0.55
13A Malone & Alger-Mayer (29)	BED	26	88	45	48	19	6	50%	DS	-0.56	
13B Guisado et al. (37)	non-BED	100	85	41	49		18	0%	DS	-0.82	
14A Nauta et al. (38)	BED	21	100	38	33	19.2	3.75	14%	PT	-0.04	-0.50
14B Nauta et al. (38)	non-BED	21	100	38	33	8.4	3.75	10%	PT	-0.23	-0.13
15A Nauta et al. (38)	BED	16	100	38	33	19.3	3.75	19%	PT	-0.19	-0.40
15B Nauta et al. (38)	non-BED	16	100	38	33	7.3	3.75	12%	PT	-0.02	-0.30
16A Fossati et al. (35)	BED	13	100	46	36	20	3	3%	PT	0.00	-0.45
16B Hayward et al. (39)	non-BED	8	100	45	38	11	6		PT	0.00	-0.58

BED= Binge eating disorder; Pre-BMI= Mean sample BMI before treatment; Pre-BDI= Mean sample Beck Depression Inventory score before treatment; Treat Dur= Length of treatment in months; Attrit Rate= Percentage of sample lost before post-treatment measurement; Treat Type= Treatment type: Psychotherapy-based (PT) or Drug/surgery-based (DS); WL= Treatment effect size (r) on weight (negative rs indicate lower post-treatment compared to pre-treatment weight); Dep= Treatment effect size (r) on depression (negative rs indicate lower post-treatment compared to pre-treatment depression).

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or no weight loss (see Blaine et al., in press). It also is in keeping with an understanding of depression as causally prior to weight change – that is, causing weight gain but not responsive to weight loss.

CONCLUSION

Binge eating status moderated weight loss treatment effects on weight but not depression. In a quantitative analysis of obese BED and non-BED samples in weight treatment, post-treatment weight loss was much smaller in obese BED than in obese non-BED samples. Indeed, obese BED individuals' average post-treatment weight loss (3.3 lbs/1.5 kg or 2% of their body weight over about $3^1/_2$ months of treatment) hardly justifies the expense of weight treatment. Subjects in non-BED samples responded positively to treatment (losing, on average, 11% of their body weight) and the benefits were greater among samples of extremely compared with moderately obese people. Thus, the chief clinical implication of this review is that BED is a risk factor for poor responses to weight treatment, at least where weight loss is concerned. Whether the poor post-treatment weight loss in obese BED samples is due to initial weight loss followed by regain, or simply failure to lose weight at all, cannot be determined from this analysis. However, research that does find an association between BED status and weight regain after loss (40) suggests that weight cycling, even within the duration of a weight treatment program, is more likely in BEDs than non-BEDs. Research documents greater weight cycling in general among obese BED than obese non-BED people (19, 41). The proclivity to weight cycle complicates obese BEDs' response to weight treatment and represents a real health risk associated with weight treatment.

Because this analysis controlled several factors that were plausibly related to responses to weight treatment (i.e., pre-treatment BMI, treatment type, treatment duration, attrition rate), their influence on the treatment effect is controlled. Of these variables that routinely covary with BED status, controlling the effects of treatment type presented the greatest challenge. Treatments that are designed for obese BEDs compared with obese people with no binge eating symptoms are often much different, and only the most basic categorization scheme (drug/surgery-based vs. psychotherapy-based could be used to match studies on treatment type. Caution is appropriate, therefore, in concluding that treatment type is completely ruled out as a possible cause of the treatment effects. Although this study did not test an explanatory hypothesis, these controls nevertheless make it more likely that the explanation for obese BEDs' poor weight loss lies in their greater levels of depression.

Despite negligible weight loss among obese BED samples, they did realize psychological benefits from treatment. The effects of weight treatment for post-treatment reductions in depression were equally large for obese BED and obese non-BED samples. Most of the studies in this review used weight treatments that combined diet, exercise, and nutritional components with cognitive-behavioral therapy. Given that weight loss was not a prerequisite for reductions in depression, it is reasonable to conclude that the psychotherapeutic elements of weight treatment are effective and worthwhile in the obese non-BED population. Among obese BEDs, however, the clinician must balance the psychological benefit of weight treatment in the form of greatly reduced depression with the poor prospects for reducing weight.

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