Pretreatment and Process Predictors of Outcome in Interpersonal and Cognitive Behavioral Psychotherapy for Binge Eating Disorder

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The present study examined pretreatment and process predictors of individual nonresponse to psychological group treatment of binge eating disorder (BED). In a randomized trial, 162 overweight patients with BED were treated with either group cognitive–behavioral therapy or group interpersonal psychotherapy. Treatment nonresponse, which was defined as nonabstinence from binge eating, was assessed at posttreatment and at 1 year following treatment completion. Using 4 signal detection analyses, greater extent of interpersonal problems prior to treatment or at midtreatment were identified as predictors of nonresponse, both at posttreatment and at 1-year follow-up. Greater pretreatment and midtreatment concerns about shape and weight, among those patients with low interpersonal problems, were predictive of posttreatment nonresponse. Lower group cohesion during the early treatment phase predicted nonresponse at 1-year follow-up. Attention to specific pre- or intreatment predictors could allow for targeted selection into differential or augmented care and could thus improve response to group psychotherapy for BED.

Keywords: predictor, group psychotherapy, interpersonal psychotherapy, cognitive–behavioral therapy, signal detection analysis

Binge eating and associated psychopathology in binge eating disorder (BED) can be substantially reduced through cognitive–behavioral therapy (CBT) and through interpersonal psychotherapy (IPT; National Institute for Clinical Excellence, 2004). Yet, 20%–50% of patients fail to fully respond by treatment's end, and effects tend to wane in the long term (Wilson & Fairburn, 2002; Wonderlich,

de Zwaan, Mitchell, Peterson, & Crow, 2003). Establishing outcome predictors could inform interventions and, thus, could prevent poor response from patients with BED.

Higher initial binge eating (Loeb, Wilson, Gilbert, & Labouvie, 2000; Peterson et al., 2000) and more severe overeating problems (Agras et al., 1995; Agras, Telch, Arnow, Eldredge, & Marnell, 1997) appear to predict poorer posttreatment BED outcome. Evidence is mixed as to whether initial specific eating disorder psychopathology, general psychopathology, and self-esteem are related to outcome (Agras et al., 1997; Carter & Fairburn, 1998; Loeb et al., 2000; Peterson et al., 2000; Safer, Lively, Telch, & Agras, 2002), but BED patients of the high negative affectivity subtype have particularly poor treatment response (Loeb et al., 2000; Stice et al., 2001). Earlier age of binge eating onset, binge eating preceding first dieting attempt (Agras et al., 1995, 1997; Safer et al., 2002), and younger age when receiving treatment (Agras et al., 1997) have been found to be related to poor treatment response, whereas initial weight status is not predictive (Carter & Fairburn, 1998; Loeb et al., 2000).

Prediction of poor treatment outcome based on pretreatment patient characteristics warrants clarification and replication, given the few BED predictor studies and that inconsistent results are likely related to a lack of statistical power and differences in treatment modalities, length of follow-up, and definition and as-

sessment of outcome across studies. Our focus in the present study was, therefore, on examining as outcome predictors those patient characteristics that were previously investigated and that are central to CBT and to IPT treatment models (e.g., dietary restraint or interpersonal problems, respectively) or to both. Within-treatment processes may also prove predictive, as these factors predict treatment outcome for bulimia nervosa (Agras et al., 2000; Fairburn, Agras, Walsh, Wilson, & Stice, 2004; Loeb et al., 2005; Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002; Wilson et al., 1999). Rapid reduction of binge eating predicts better posttreatment outcome for individuals with BED who have undergone CBT (Grilo, Masheb, & Wilson, 2006), but the predictive value of changes in associated psychological symptoms, such as interpersonal problems, during the early treatment phase remains unclear. Nonspecific process-related factors, such as therapeutic alliance or group cohesion, have not been examined as outcome predictors for BED. The present study examines pre- and intreatment factors as predictors of immediate and of long-term nonresponse in an adequately powered, randomized-controlled trial of psychotherapy for individuals with BED (Wilfley et al., 2002).

Method

Participants and Procedure

Participants were 162 overweight individuals with BED, who were recruited for the treatment trial at two sites: New Haven (126 participants; 77.8%) and San Diego (36 participants; 22.2%). Methods and design are detailed in the main outcome report (Wilfley et al., 2002). Participants met diagnostic criteria for BED according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR*;American Psychiatric Association, 2000) and were randomized into either Group CBT or Group IPT after stratification by sex: 134 women (82.7%) and 28 men (17.3%). Both treatments were manual based and consisted of 20 weekly 90-min group sessions and of 3 individual sessions. Groups included 9 patients each and were led by PhD therapists and by cotherapists who were at least advanced doctoral students. All patients signed an informed consent approved by the site-respective institutional review board.

Of the 162 randomized patients, 146 (90.1%) completed treatment and 16 (9.9%) dropped out before the end of treatment. Analyses were based on assessment completers: Of the randomized patients, 158 (97.5%) completed posttreatment assessment and 143 (88.3%) completed assessment at 1-year follow-up.

Assessments

The main outcome criterion of nonresponse to treatment was operationalized as nonabstinence, which was defined as having one or more episodes of binge eating (i.e., eating an unusually large amount of food, accompanied by a sense of loss of control; American Psychiatric Association, 2000) in the past 28 days (Eating Disorder Examination [EDE] 12.0D; Fairburn & Cooper, 1993).

Predictor variables were derived from a structured interview and from self-report questionnaires. Potential pretreatment predictors, which were assessed prior to treatment initiation, included the following: (a) duration since first onset of binge eating; (b) the temporal order of binge eating versus dieting onset; (c) eating disorder psychopathology, specifically, shape/weight concern and restraint (EDE); (d) general psychiatric symptomatology (Global Severity Index [GSI] from the Symptom Checklist–90–Revised [SCL–90–R]; Derogatis, 1977); (e) comorbid psychiatric diagnosis (Structured Clinical Interview for *DSM–III–R* [SCID, SCID II]; Spitzer, Williams, Gibbon, & First, 1990, 1992); (f) self-esteem (Rosenberg Self-Esteem Scale [RSES]; Rosenberg, 1979); (g) negative affectivity subtype (cluster analytically derived from EDE restraint, GSI, and Rosenberg Self-Esteem Scale; Stice et al., 2001); (h) interpersonal problems (Inventory of Interpersonal Problems [IIP]; Horowitz, Rosenberg, Baer, Ureno, & Villasenor, 1988); (i) social functioning (Social Adjustment Scale; Weissman & Bothwell, 1976); (j) site (New Haven versus San Diego); (k) assignment to and perceived suitability of CBT versus IPT; (l) sociodemographic characteristics; and (m) body mass index (kg/m^2), which was calculated from measured height and weight.

Potential intreatment predictors of treatment nonresponse were assessed immediately following Group Sessions 6 and 10 (i.e., at early treatment and at midtreatment); they included group cohesion (Group Attitude Scale [GAS]; Evans & Jarvis, 1986), group climate (Engaged, Avoiding, and Conflict Scales from the Group Climate Questionnaire; MacKenzie, 1981), and psychotherapeutic alliance (California Psychotherapy Alliance Scale; Gaston, 1991). Other potential intreatment predictors were assessed following Session 10 only (i.e., at midtreatment); they included eating disorder psychopathology (Eating Disorder Examination–Questionnaire [EDE–Q]; Fairburn & Beglin, 1994), interpersonal problems (IIP), general psychopathology (GSI), and self-esteem (RSES). For these latter constructs, midtreatment levels and difference scores between pre- and midtreatment were used. In addition, assignment to CBT versus IPT, attrition from treatment (see *Participants and Procedure*), and number of sessions attended were used as predictors in order to capture treatment specificity and dose–response relationship.

Data Analytic Plan

Signal detection analysis was used to identify distinct patient subgroups likely to show treatment nonresponse based on pretreatment and intreatment characteristics (Kraemer, 1992). Signal detection analysis is a well-established procedure that is hypothesis generating rather than hypothesis testing, nonparametric and distribution-free, and allows for consideration of large sets of predictors, while being robust to multicollinearity, outliers, and missing data problems (Kiernan, Kraemer, Winkleby, King, & Taylor, 2001). Sensitive to detecting interactions between predictors, it is well-suited to clinical decision making, as algorithms are derived for identification of patients at risk of treatment nonresponse (Agras et al., 2000). The signal detection analytic method of calculating receiver operating characteristics (ROC) and recursive partitioning was applied; all predictor variables were included, regardless of their zero-order associations. First, for each variable, cutoff points were determined to split the sample into likely treatment nonresponse versus response by computing sensitivity and specificity. Next, equally weighting those cutoff points for all variables, the optimal predictor and cutoff point was identified, creating sample subsets with a predicted negative versus positive outcome; this process was repeated on the identified sample subsets, and so on, using chi-square tests at $p < .01$ as a stopping rule

or proceeding until there were $n < 10$ individuals by subset. Four ROC analyses were conducted, including two outcomes (nonresponse at posttreatment and at 1-year follow-up) and two sets of predictors (pre- and intreatment variables). Pre- and intreatment predictors were analyzed separately in order to facilitate clinical decision-making prior to treatment or during the early treatment phase. The ROC-derived subgroups were further characterized on all pretreatment and all intreatment variables (analyses of variance and Tukey honestly significant difference tests or chi-square tests, respectively; $p < .01$). Effect size of ROC-derived classification was evaluated with the area under the curve (AUC) statistic (Kraemer & Kupfer, 2006).[1]

Results

ROC Analyses: Nonresponse at Posttreatment

Results from ROC analyses are depicted in Figure 1. Of 158 patients, 35 (22.1%) showed posttreatment nonresponse. ROC analysis for pretreatment predictors revealed that posttreatment nonresponse was best predicted by an initial IIP score ≥ 1.7 (indicative of greater interpersonal problems) or, in the case of a lower IIP score, a combined shape/weight concern score on the EDE ≥ 4.5 (indicative of more severe shape/weight concern; both $ps < .01$), overall test, $\chi^2(1, N = 156) = 15.29$, $p < .001$. For prediction of posttreatment nonresponse by intreatment variables, ROC analysis found nonresponse best predicted by a midtreatment score on the IIP ≥ 1.9, among individuals with lower IIP scores, a midtreatment EDE shape/weight concern score ≥ 3.1 (both $ps < .01$), overall test, $\chi^2(1, N = 149) = 18.80$, $p < .001$.

ROC Analyses: Nonresponse at 1-Year Follow-Up

As depicted in Figure 1, 43 out of 143 patients (30.1%) showed nonresponse at 1-year follow-up. ROC analysis of pretreatment characteristics identified a pretreatment IIP score ≥ 1.3 as a significant predictor of follow-up nonresponse ($p < .01$). ROC analysis of intreatment characteristics revealed that follow-up nonresponse was best predicted by a GAS score < 148 at Session 6 (indicative of lower perceived group cohesion; $p < .01$).

Clinical Utility of Algorithms

The algorithms derived from ROC analyses had adequate sensitivity and specificity (see Figure 1). If used to identify patients at risk of nonresponse to the standard treatments of CBT or IPT, and to thus select them for differential or augmented care, 64.8%–73.2% would correctly be assigned to standard, to differential, or to augmented care (true positives, true negatives), 18.1%–23.2% would unnecessarily be assigned to differential or to augmented care (false positives), and 7.4%–15.2% would be assigned to standard treatment but not respond (false negatives). Effect size of algorithm-based classification was mostly medium (61.8% $\leq$ AUC $\leq$ 65.5%).

Clinical Profiles of Subgroups Identified by ROC Analyses

As presented in the left side of Table 1, patients with high interpersonal problems, who were likely to show posttreatment nonresponse, had greater general psychopathology and lower self-esteem at pre- and midtreatment, higher pretreatment rates of any personality

disorder, of any Cluster B personality disorder, and of a high negative affectivity subtype, and they also displayed lower initial social adjustment, compared with patients who had low interpersonal problems and low shape/weight concerns (all $ps < .01$). Those patients who had low interpersonal problems but high shape/weight concerns had an intermediate position between the other two groups; their level of shape/weight concerns persisted through midtreatment, whereas it decreased from pre- to midtreatment in both other groups ($p < .01$). Patients with high interpersonal problems, who were likely to show nonresponse at 1-year follow-up, had a similar constellation of mood- and personality-related symptoms that coincided with low perceived cohesion of the therapeutic group and with low engagement in the therapeutic group (see Table 1, right side; all $ps < .01$).
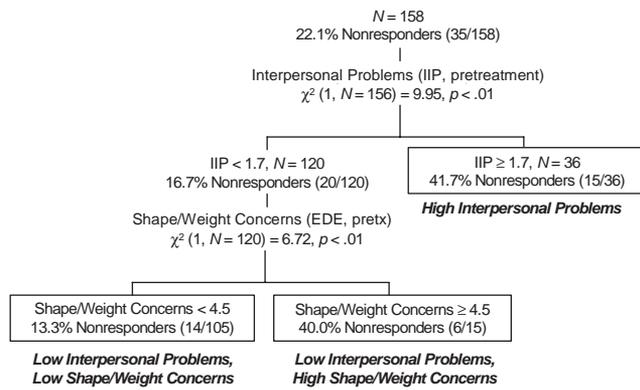
Discussion

The current study examined pre- and intreatment characteristics as predictors of poor treatment outcome in a large, randomized trial of group CBT and group IPT for individuals with BED. Using ROC analyses, greater extent of interpersonal problems prior to treatment initiation or at midtreatment emerged as a major negative prognostic indicator, predicting posttreatment and long-term nonresponse. Greater shape and weight concerns, among those with low interpersonal problems, were also predictive of posttreatment nonresponse. In addition, lower group cohesion in the early treatment phase emerged as a process-related predictor of long-term nonresponse.

A higher level of interpersonal problems and less perceived group cohesion were thus both central in determining group treatment outcome and were likely intertwined. Patients with a higher level of interpersonal dysfunction and with a greater level of related general psychopathology and personality disturbance also perceived less group cohesion. The latter finding parallels the predictive value of early therapeutic alliance in individual treatment of bulimia nervosa (Loeb et al., 2005; Wilson et al., 1999). The present results further indicate that interpersonal problems and shape and weight concerns may be differentially important for the maintenance of binge eating in subgroups of patients with BED (Fairburn, Cooper, & Shafran, 2003), which points to the necessity of further understanding and delineating the heterogeneity associated with this disorder. Although patients with higher interpersonal problems or higher shape and weight concerns had a greater likelihood of poor treatment outcome, it should be noted that overall, CBT and IPT significantly improved these and other psychological symptoms associated with binge eating abstinence, the most rigorous outcome criterion (Wilfley et al., 2002).

The predictors of treatment nonresponse are valid for both treatments; treatment-specific moderators or mediators were not identi-

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[1] The AUC statistic estimates the probability that a randomly selected patient with a positive test (e.g., a GAS score < 148 at Session 6 for the prediction of treatment nonresponse at 1-year follow-up) will more likely show nonresponse to treatment than will a patient with a negative test (i.e., GAS $\geq$ 148). The AUC statistic can similarly be interpreted for continuous and for categorical variables, and effect-size classification reflects that of Cohen's d (low, AUC $< 63.8\%$, Cohen's $d < 0.5$; medium, $63.8\% \leq$ AUC $< 71.4\%$, $0.5 \leq$ Cohen's $d < 0.8$; large, AUC $\geq 71.4\%$, Cohen's $d \geq 0.8$). An AUC $=$ 50.0% indicates that a patient with a positive test is just as likely to show treatment nonresponse as is a patient with a negative test, whereas an AUC $=$ 100.0% indicates that every patient with a positive test shows treatment nonresponse and that every patient with a negative test shows treatment response (for further details, see Kraemer & Kupfer, 2006).

(a) Pretreatment Predictors of Posttreatment Non-Response

$N = 158$
22.1% Nonresponders (35/158)

Interpersonal Problems (IIP, pretreatment)
$\chi^2 (1, N = 156) = 9.95$, $p < .01$

IIP < 1.7, $N = 120$
16.7% Nonresponders (20/120)

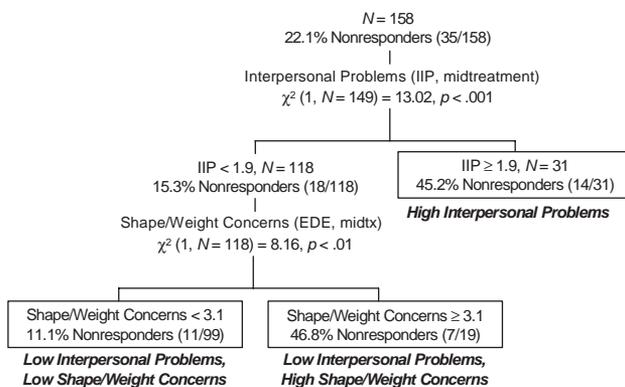IIP ≥ 1.7, $N = 36$
41.7% Nonresponders (15/36)
High Interpersonal Problems

Shape/Weight Concerns (EDE, pretx)
$\chi^2 (1, N = 120) = 6.72$, $p < .01$

Shape/Weight Concerns < 4.5
13.3% Nonresponders (14/105)
**Low Interpersonal Problems,
Low Shape/Weight Concerns**

Shape/Weight Concerns ≥ 4.5
40.0% Nonresponders (6/15)
**Low Interpersonal Problems,
High Shape/Weight Concerns**

(b) Pretreatment Predictors of Non-Response at One-Year Follow-Up

$N = 143$
30.1% Nonresponders (43/143)

Interpersonal Problems (IIP, pretreatment)
$\chi^2 (1, N = 142) = 9.09$, $p < .01$

IIP < 1.3, $N = 83$
20.5% Nonresponders (17/83)
Low Interpersonal Problems

IIP ≥ 1.3, $N = 59$
44.1% Nonresponders (26/59)
High Interpersonal Problems

(c) Intreatment Predictors of Posttreatment Non-Response

$N = 158$
22.1% Nonresponders (35/158)

Interpersonal Problems (IIP, midtreatment)
$\chi^2 (1, N = 149) = 13.02$, $p < .001$

IIP < 1.9, $N = 118$
15.3% Nonresponders (18/118)

IIP ≥ 1.9, $N = 31$
45.2% Nonresponders (14/31)
High Interpersonal Problems

Shape/Weight Concerns (EDE, midtx)
$\chi^2 (1, N = 118) = 8.16$, $p < .01$

Shape/Weight Concerns < 3.1
11.1% Nonresponders (11/99)
**Low Interpersonal Problems,
Low Shape/Weight Concerns**

Shape/Weight Concerns ≥ 3.1
46.8% Nonresponders (7/19)
**Low Interpersonal Problems,
High Shape/Weight Concerns**

(d) Intreatment Predictors of Non-Response at One-Year Follow-Up

$N = 143$
30.1% Nonresponders (43/143)

Group Coherence (GAS, session 6)
$\chi^2 (1, N = 138) = 6.94$, $p < .01$

GAS ≥ 148, $N = 93$
22.6% Nonresponders (21/93)
High Group Coherence

GAS < 148, $N = 45$
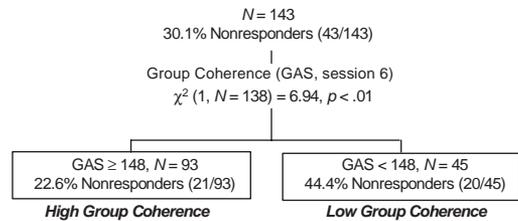44.4% Nonresponders (20/45)
Low Group Coherence

Figure 1. ROC analyses: Prediction of posttreatment and long-term nonresponse from pretreatment and intreatment characteristics in group cognitive–behavioral therapy and in group interpersonal psychotherapy of binge eating disorder. ROC = receiver operating characteristics; IIP = Inventory of Interpersonal Problems (range: 0–4*; scores indicating less favorable conditions are asterisked); EDE = Eating Disorder Examination (range; 0–6*); GAS = Group Attitude Scale (sum score range: 20*–180); AUC = area under the curve as a measure of effect size (low: AUC% < 63.8%, medium: 63.8% ≤ AUC% < 71.4%, large: AUC% ≥ 71.4%). (a) Sensitivity: 0.60; specificity: 0.75; false positives: 19.2%; false negatives: 9.0%; AUC: 64.0%; 2 missing values. (b) Sensitivity: 0.66; specificity: 0.75; false positives: 19.5%; false negatives: 7.4%; AUC: 65.5%; 1 missing value. (c) Sensitivity: 0.60; specificity: 0.67; false positives: 23.2%; false negatives: 12.0%; AUC: 61.8%; 9 missing values. (d) Sensitivity: 0.49; specificity: 0.74; false positives: 18.1%; false negatives: 15.2%; AUC: 65.5%; 5 missing values.

fied.[2] As both treatments were equally intense, were delivered in group format, were adapted to BED, and were equally potent, subgroups of patients who responded differently to CBT versus IPT may not have emerged. Treatment-specific mechanisms of action may not have been identified, because change in binge eating or in the associated psychopathology earlier than at midtreatment was not assessed. In fact, as suggested by examinations of mediators and of time course in comparative treatment trials of bulimia nervosa and of BED, published after the current study was designed (Grilo et al., 2006; Wilson et al., 1999, 2002), treatment specificity may more likely be found in the course of binge eating and of associated psychological symptoms during the initial treatment sessions, for example, through the 1st month of treatment.

Specific algorithms for identifying patients who require additional clinical attention were derived. These algorithms could be used in potential targeted selection of patients into differential or augmented care, which would improve patient response to psychological group therapy for BED. Utility of algorithms was substantial, leading to correct selections of more than two thirds of

[2] In a randomized clinical trial, a moderator of treatment is a pretreatment variable that is uncorrelated with treatment and that has an interactive effect with treatment condition for predicting intervention response; thus, a moderator of treatment indicates for whom or under what conditions a treatment works. A mediator of treatment is a process variable that is correlated with treatment and that has a main or interactive effect with it on outcome; thus, a mediator of treatment indicates why and how a treatment works. A predictor is defined here as a variable that precedes and has a main effect on outcome but that has no interactive effect with treatment (see Kraemer, Wilson, Fairburn, & Agras, 2002).

Table 1

Outcome, Predictor, and Profile Variables for ROC Subgroups in the Prediction of Posttreatment and Long-Term Nonresponse to Group Cognitive Behavioral Therapy (CBT) and Group Interpersonal Psychotherapy (IPT) for Binge Eating Disorder

	(a) Pretreatment prediction of posttreatment nonresponse			(b) Pretreatment prediction of nonresponse at 1-year follow-up	
	Interpersonal problems			Interpersonal problems	
	Low		High	Low	High
	Shape concern				
	Low	High			
	$N = 105$, 67.3%	$N = 15$, 9.6%	$N = 36$, 23.1%	$N = 83$, 58.5%	$N = 59$, 41.5%
Outcome variable					
Nonabstinence from binge eating, n (%)	14[a] (13.3)	6[a,b] (40.0)	15[b] (41.7)	17[a] (20.5)	26[b] (44.1)
Pretreatment predictor variables, M (SD)					
IIP	**0.9[a] (0.4)**	**1.0[a] (0.3)**	**2.1[b] (0.3)**	**0.8[a] (0.3)**	**1.8[b] (0.4)**
Shape/weight concern composite EDE	3.2[a] (0.9)	4.9[b] (0.4)	4.0[c] (0.8)	3.5 (1.0)	3.6 (0.9)
Pretreatment profile variables					
Treatment preference, CBT, n (%)	51 (49.5)	9 (60.0)	11 (31.4)	48[a] (58.5)	19[b] (32.8)
Current major depression (SCID I), n (%)	14 (13.3)	2 (13.3)	9 (25.0)	7[a] (8.4)	15[b] (25.4)
Any personality disorder (SCID II), n (%)	22[a] (21.0)	6[a] (40.0)	30[b] (83.3)	20[a] (24.1)	33[b] (55.9)
Any Cluster B personality disorder (SCID II),					
n (%)	9[a] (8.6)	1[a,b] (6.7)	10[b] (27.8)	6 (7.2)	11 (18.6)
High negative affect subtype[d], n (%)	11[a] (10.5)	5[a,b] (33.3)	29[b] (80.6)	6[a] (7.2)	34[b] (57.6)
Global Severity Index T score (SCL-90-R),					
M (SD)	39.4[a] (6.3)	43.1[a] (5.4)	52.2[b] (7.5)	38.4[a] (6.3)	48.3[b] (7.3)
RSES, M (SD)	28.9[a] (5.2)	26.5[a,b] (5.0)	22.2[b] (4.3)	29.4[a] (5.0)	23.8[b] (4.8)
SAS, M (SD)	2.0[a] (0.5)	2.3[a,b] (0.6)	2.4[b] (0.5)	2.0[a] (0.5)	2.3[b] (0.5)

	(c) Intreatment prediction of posttreatment nonresponse			(d) Intreatment prediction of nonresponse at 1-year follow-up	
	Interpersonal problems			Group cohesion	
	Low		High	High	Low
	Shape concern				
	Low	High			
	$N = 99$, 66.4%	$N = 19$, 12.8%	$N = 31$, 20.8%	$N = 93$, 67.4%	$N = 45$, 32.6%
Outcome variable, n (%)					
Nonabstinence from binge eating (EDE)	11[a] (11.1)	7[b] (36.8)	14[b] (45.2)	21[a] (22.6)	20[b] (44.4)
Intreatment predictor variables, M (SD)					
IIP	**1.1[a] (0.4)**	**1.2[a] (0.4)**	**2.2[b] (0.3)**	1.2[a] (0.6)	1.6[b] (0.6)
Change shape/weight concern composite (EDE–Q)[f]	**1.7[a] (0.7)**	**3.5[b] (0.5)**	**2.1[a] (0.8)**	2.0 (1.0)	2.0 (0.8)
GAS	152.2 (21.2)	156.9 (16.2)	142.3 (28.6)	**164.3[a] (8.5)**	**124.4[b] (15.5)**
Intreatment profile variables, M (SD)					
Early treatment, Session 6					
Group Climate Engaged (GCQ)	5.5 (0.8)	5.8 (0.7)	5.4 (0.8)	5.8[a] (0.6)	5.2[b] (0.8)
Midtreatment, Session 10					
GAS	151.4 (25.2)	149.9 (24.9)	146.7 (26.4)	158.3[a] (21.4)	137.7[b] (25.2)
Global Severity Index t score (SCL-90-R)	35.7[a] (8.5)	33.3[a] (8.4)	50.1[b] (7.2)	36.4[a] (10.3)	41.2[b] (8.6)
RSES	29.9[a] (5.3)	27.7[a] (5.4)	22.7[b] (4.6)	28.9 (5.6)	27.2 (5.1)
Pre- to midtreatment change[e]					
Change shape/weight concern composite (EDE)	1.3[a] (1.0)	−0.1[b] (0.8)	1.0[a] (1.1)	1.1 (1.1)	1.1 (1.2)

Note. The outcome variable nonabstinence from binge eating over the past 28 days was assessed for (a) and (c) at posttreatment and for (b) and (d) at 1-year follow-up. Results for predictors identified by ROC analysis are in bold type. Only profile variables indicating group differences significant at $p < .01$ are presented (analyses of variance and Tukey honestly significant difference tests or chi-square tests, respectively). Missing values: (a), 2; (b), 1; (c), 9; (d), 5. IIP = Inventory of Interpersonal Problems (range: $0–4^*$; scores indicating less favorable conditions are asterisked); EDE = Eating Disorder Examination ($0–6^*$); SCID I, II = Structured Clinical Interview for *DSM-III-R*; SCL-90-R = Symptom Checklist-90-Revised (Global Severity Index T score ≥ 63 as cutoff score for clinically significant psychopathology); RSES = Rosenberg Self-Esteem Scale (sum score range: $10^*–40$); SAS = Social Adjustment Scale ($1–5^*$); GAS = Group Attitude Scale (sum score range: $20^*–180$); GCQ = Group Climate Questionnaire ($1^*–7$); EDE–Q = Eating Disorder Examination–Questionnaire ($0–6^*$).
[a,b,c] Different superscripts indicate significant group differences ($p < .01$). [d] Negative affectivity subtyping was cluster analytically derived from EDE restraint, SCL-90-R Global Severity Index, and RSES. [e] Change scores between pre- and midtreatment were calculated as pretreatment score minus midtreatment score. [f] The EDE was administered at pre- and at midtreatment.

patients and to selection for unnecessary augmented care in less than one fourth of patients (only $\leq$ 15.2% false negatives). Using ROC analyses on random splits of the current sample, the same predictors, or correlates of them, were confirmed as negative prognostic indicators.[3] Nevertheless, validation of derived algorithms in an independent sample is needed.

Clinically, our results indicate that patients whose interpersonal dysfunction is similar to or greater than population norms for psychiatric disorders (e.g., those with personality disturbances; Horowitz et al., 1988; Wilfley et al., 2000) and patients with low interpersonal dysfunction who suffer from severe shape and weight concerns may need differential or augmented care. Special interventions on interpersonal disturbance (e.g., Markowitz, Skodol, & Bleiberg, 2006) and on body image disturbance (e.g., Fairburn et al., 2003) should be considered for these patient subgroups; optimal timing and sequencing of such targeted treatment awaits examination. To prevent potential low perceived group cohesion, therapists need to focus on patients' group engagement, for example, by enhancing patients' mutual understanding and by fostering positive group treatment expectations (see Constantino, Arnow, Blasey, & Agras, 2005). Fine-grained examination of the time course of treatment, which would allow identification of moderators and of mediators, could enhance specification of treatment components and evaluation of models of adjunctive, extended, or sequential care (National Institute of Clinical Excellence, 2004). Such clinical research is warranted and would allow researchers to pinpoint the optimal treatment delivery for likely nonresponders, key to improving these patients' response to psychotherapy for BED.

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[3] For determining reproducibility of the identified predictors, we ran post hoc ROC analyses using samples with two random selections of 75% of patients in each of the four ROC analyses; thus, eight post hoc ROC analyses on eight randomly selected samples were conducted. These split-sample analyses identified either the same predictors (IIP, GAS), with cutoff points similar to those of the full-sample analyses, or predictors that were significantly correlated with them ($p < .001$):

(a) Prediction of posttreatment nonresponse from pretreatment predictor variables. Sample 1: SCL–90–R GSI $\geq$ 53; $\chi^2(1, N = 117) = 15.04$, $p < .001$; Pearson correlation coefficient with pretreatment IIP score, $r = .76$, $p < .001$. Sample 2: no solution ($p > .01$). (b) Prediction of nonresponse at 1-year follow-up from pretreatment predictor variables. Sample 3: SCL–90–R GSI $\geq$ 45; $\chi^2(1, N = 107) = 9.93$, $p < .01$; Pearson correlation coefficient with pretreatment IIP score, see above ($p < .001$). Sample 4: no solution ($p > .01$). (c) Prediction of posttreatment nonresponse from intreatment predictor variables. Sample 5: midtreatment IIP $\geq$ 1.8; $\chi^2(1, N = 116) = 16.31$, $p < .001$. Sample 6: GAS at Session 6 < 140; $\chi^2(1, N = 116) = 9.58$, $p < .01$. (d) Prediction of nonresponse at 1-year follow-up from intreatment predictor variables. Sample 7: decrease of IIP from pre- to midtreatment $\geq$ 0.25; $\chi^2(1, N = 94) = 9.58$, $p < .01$. Sample 8: no solution ($p > .01$).

Thus, although these split-sample analyses expectedly did not confirm the exact results from the full-sample analyses, the results were overall consistent with high interpersonal problems, or with correlates of them, as main negative prognostic indicators of posttreatment and of long-term nonresponse to group psychotherapy for BED. The finding of no statistically significant solutions for several analyses is likely attributable to a lack of power related to the sample split.

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