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Is there an optimal length of psychological treatment for eating disorder pathology?

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Abstract

Objectives: Treatment guidelines for eating disorders (ED) are unclear about the optimal length of psychological care. We aimed to investigate associations between treatment duration and changes in ED pathology.

Method: Data for 164 outpatients accessing psychological interventions for ED were analyzed using MANOVA. We tested associations between number of therapy sessions and pre-post treatment changes in clinical outcomes (EDE-Q global scores, binge eating, purging); adjusting for baseline measures, diagnoses and treatment type. Secondary analyses included rapid response variables in the above outcomes by treatment session eight. Partial correlations between treatment duration and BMI changes (adjusting for intake BMI) were examined for anorexia nervosa cases.

Results: Treatment duration was not significantly associated with changes in ED outcomes after adjusting for rapid response. BMI change (weight regain) was not correlated with treatment duration in anorexia nervosa cases. Rapid response was associated with better EDE-Q outcomes, but not with changes in binge eating or purging behaviors.

Discussion: ED outcomes are unrelated to treatment duration; rapid response is a useful prognostic indicator for treatment planning.

Key words: eating disorders, anorexia nervosa, bulimia nervosa, psychological therapy

Clinical guidelines for the management of eating disorders recommend that up to 20 sessions of psychological care should be offered over the course of four to twelve months, though it is recognized that conditions such as anorexia nervosa may require longer treatments.^{1,2} These recommendations are informed by clinical experience and data from controlled trials of time-limited interventions.³ To date, there is little evidence on the relationship between treatment duration and changes in eating disorders pathology. However, it has been shown that symptomatic improvements during the earlier stages of therapy (i.e., rapid response by sessions 4 to 6) tend to be associated with better outcomes after treatment for a variety of eating disorders.⁴⁻⁹

Outside of the eating disorders field, there is a growing body of research on treatment duration and clinical improvement. In a citation classic, Howard et al.¹⁰ introduced the notion of dose-response in psychotherapy, showing a curvilinear relationship between the number of therapy sessions and the percentage of improving patients. According to their analysis of 2431 cases with heterogeneous diagnoses accessing various psychological treatment models, they concluded that 26 sessions marks the point at which 75% of patients improve, after which further gains are rare. They also argued that the dose at which 50% of patients improve (6 to 8 sessions) can be used as a criterion for a fair trial of treatment, at which point individual cases should be assessed to determine if further sessions are likely to be beneficial. This dose-response effect has been replicated in several studies and clinical populations.¹¹⁻¹⁷

A consistent observation across these studies is that of diminishing gains after a specific number of sessions, however the 'optimal dose' varies

across samples. For instance, the optimal dose in guided self-help for mild-to-moderate depression and anxiety problems appears to be between 4 to 6 sessions,¹³ whereas Hansen and Lambert¹⁴ estimated an optimal dose of 16 sessions of intensive psychotherapy in a heterogeneous sample of patients in community mental health centers. It is also evident that different problem domains change at different stages of therapy. For example, acute psychiatric symptoms appear to remit earlier compared to social and interpersonal problems.^{12,16,17}

In summary, the dose-response effect has been documented in several psychological therapy studies and clinical populations. However, the association between treatment duration and therapeutic change varies according to specific outcome domains. In particular, it is unclear if and how eating disorder pathology may be associated with treatment duration after adjusting for the influence of rapid response. Therefore, the primary aim of this study was to examine associations between the number of treatment sessions and changes in eating disorder pathology in a clinical sample of outpatients accessing psychological interventions.

METHODS

Setting and interventions

Data for this study were collected as part of outpatient psychological care offered in a specialist eating disorders service in Nottinghamshire, England. The service offered psychological interventions delivered by post-graduate level psychotherapists, counsellors and clinical psychologists. Interventions included cognitive behavioral therapy, cognitive analytic therapy, person centered counselling, gestalt therapy, integrative

psychotherapy, and eye-movement desensitization and reprocessing (EMDR) for trauma related conditions. Treatment duration was decided on an individual case basis and could have been up to 18 months.

Measures and data sources

All patients above the age of 18 treated between September 2012 and October 2015 provided consent for the use of their anonymous clinical information for service evaluation and research. Patients attended an initial assessment meeting with the clinical lead or a senior psychotherapist prior to starting therapy. Baseline assessments included a semi-structured interview guided by standard diagnostic criteria,¹⁸ supplemented by self-reported measures of eating disorder pathology (EDE-Q)¹⁹ and functional impairment (CIA).²⁰ The body mass index (BMI) was calculated at baseline and final assessments, based on height / weight measurements taken at the clinic. BMI was only assessed in cases of anorexia nervosa or cases of atypical anorexia nervosa that had rapid weight loss at referral but were still in the healthy BMI range at intake assessments (classified as EDNOS - eating disorder not otherwise specified). After starting therapy, patients were asked to self-complete the above measures at an eight week review session and at the final therapy session.

The Eating Disorder Examination Questionnaire is a 36 item measure of ED psychopathology,¹⁹ with adequate internal consistency, test-retest reliability and criterion validity.^{21,22} The EDE-Q has four subscales: restraint, eating concern, shape concern, and weight concern. An EDE-Q global score can be derived for outcome measurement by estimating the mean of the 22 Likert scale items assessing the core attitudinal features of ED psychopathology. Cronbach's alpha in this study sample for the EDE-Q

global score items was $\alpha = .92$. EDE-Q also captures behavioral aspects of ED pathology, including single items quantifying the frequency of binge eating and purging episodes in the last month.

The Clinical Impairment Assessment (CIA) is a 16 item measure of psychosocial impairment related to eating disorder pathology; with adequate internal consistency, construct and discriminant validity.²⁰ The items can be self-rated using 4-point Likert scales, which are summed to derive a global impairment rating. Cronbach's alpha for the CIA items in this sample was $\alpha = .90$.

Additional data sources included demographics (age, gender, ethnicity), the type of psychological therapy received (coded according to the therapist's theoretical orientation), and the total number of sessions attended.

Sample characteristics

The analysis included 164 cases with available baseline EDE-Q data and who attended at least 1 therapy session. Most patients were females (95.1%), with a mean age of 30.13 years (SD = 9.64; range: 18 to 60) and of a White British background (97.6%). Initial assessments (guided by DSM-IV criteria)²³ indicated that 26.8% presented with bulimia nervosa, 14.0% with anorexia nervosa, 13.4% with binge eating disorder, and 45.7% with EDNOS. Mean baseline EDE-Q global scores, behavioral measures and BMI estimates are described in Table 1. Clinical records indicated that 59.8% completed treatment, 20.1% dropped out (unilateral discontinuation of treatment), and the remaining cases were referred on to other services by mutual agreement.

Clinical interventions recorded in case records were integrative psychotherapy (41.5% of cases), cognitive behavioral therapy (18.3%), person centered counselling (12.2%), cognitive analytic therapy (12.2%), gestalt therapy (11.6%) and EMDR (4.3%). The mean number of attended therapy sessions was 16.23 (SD = 12.97; range = 1 to 76).

Statistical analyses

We formulated an analysis plan in 3 steps aiming to: (1) deal with missing data; (2) to summarize clinical outcomes; and (3) to investigate associations between treatment duration, rapid response and pre-post treatment outcomes.

In the first step we expected that some data were missing systematically since some patients dropped out or were referred to other services prior to completing the final outcome measures. To deal with this, we applied multiple imputation using an expectation maximization method.²⁴ In addition, we applied logistic regression, where all baseline demographic and clinical variables were used to predict cases with missing (vs. complete) outcomes data at the end of treatment. The predicted probabilities from this model were transformed to inverse probability weights (IPW) which enabled us to appropriately weight cases with missing/imputed data.²⁵

In the second step we calculated pre-post treatment effect sizes (Cohen's *d*) weighted by sample size for EDE-Q global scores, EDE-Q behavioral measures (binge eating, purging frequencies) and BMI. The total percentage of cases meeting criteria for reliable and clinically significant improvement (RCSI) in EDE-Q global scores was calculated based on Jacobson and Truax criteria,²⁶ applying a diagnostic cut-off of 2.77 and a

reliable change index of 1.13 which were derived from norms reported by Fairburn and Beglin.¹⁹ We also compared the post-treatment EDE-Q global score for the study sample to published normative data,²⁷ following the methodological recommendations by Bardone-Cone et al.²⁸

In the third step, we undertook a series of tests to examine if length of treatment is associated with attitudinal (EDE-Q global score), behavioral (EDE-Q binge eating and purging measures) and physical (BMI) outcome indicators. We started by examining the distribution of target variables and checking model assumptions. Preliminary analyses were based on univariate regression models predicting change scores (baseline – final measure, where a positive score denotes improvement) for each of the three EDE-Q outcomes to assess if treatment sessions are better modelled as a continuous, polynomial (quadratic or cubic) or log-transformed variable. *Goodness-of-fit* was based on assessing the AIC, BIC statistics and -2 log likelihood tests. Preliminary tests confirmed that a log-transformed sessions variable offered the best fit to the data (i.e., statistically significant reduction in -2 log likelihood from linear model to log-transformed model), so all subsequent analyses included the transformed variable. We also found that change scores for the two EDE-Q behavioral variables were not normally distributed, so subsequent analyses would need to be robust to potential violations of regression model assumptions.

The primary analysis applied MANOVA, which allows the values of multiple dependent scale variables to be modelled in a single analysis based on their relationships to predictors, reducing the chance for spurious findings that are possible when performing multiple tests of significance in the same data. Change scores in the three EDE-Q outcome measures (global score, binge eating, purging) were entered as dependent variables. Predictors

included diagnosis, type of therapy, baseline EDE-Q measures (described above), baseline CIA, and a log-transformed sessions variable. The model was run with inverse probability weights (explained above) and repeated 1,000 times using bootstrap resampling to address the uncertainty around the sample distributions for EDE-Q behavioral measures. We performed two additional sensitivity analyses to check the robustness of findings. The MANOVA with bootstrap resampling was repeated using winsorized data for the EDE-Q behavioral measures to check if extreme outliers may have influenced results. The analysis was also repeated in a subsample ($n = 142$) that excluded anorexia nervosa cases to assess if this particular group may have biased the results in some way, since prior studies have suggested that EDE-Q may not be sensitive enough to detect meaningful changes in AN cases.²⁹

Next, we undertook an analysis of ‘rapid response’, using data from the subsample of cases ($n = 111$) that had an assessment at session eight and who attended at least nine therapy sessions (so that the final session measure was not confounded with the eight-week assessment). We repeated the above MANOVA strategy and sensitivity analyses entering a binary variable for ‘rapid response’ in each of the EDE-Q measures. To determine rapid response in global EDE-Q scores, cases coded ‘1’ had EDE-Q change scores ≥ 1.13 (reliable change index) by session eight, and cases coded ‘0’ did not show reliable improvement. Rapid responders in behavioral measures had reduced their monthly binge eating or purging episodes by at least 70% by session eight, based on definitions of rapid response applied in previous community sample studies.⁶

Given the restricted number of cases with available BMI data, only partial correlations were applied to investigate relationships between length

of treatment (log-transformed sessions) and BMI change scores (adjusting for baseline BMI). The BMI change score (final – initial BMI) was coded such that a positive value indicated an increase and a negative value indicated a reduction in BMI after treatment. Partial correlations were carried out in the sample of cases with available BMI data ($n = 32$) and repeated in the subsample of AN cases ($n = 22$) excluding atypical cases that were classified under the EDNOS diagnosis.

[Table 1]

RESULTS

Clinical outcomes

Table 1 summarizes pre-post treatment clinical outcome measures. Effect sizes reflected small ($d = 0.32$ for changes in purging) to large ($d = 1.42$ for changes in EDE-Q global scores) estimates of improvement across outcome domains. The large effect size for BMI gain in AN cases ($d = 0.89$) contrasts with the moderate effect ($d = 0.49$) observed in the sample including EDNOS cases, since the therapeutic goal for the latter cases was to maintain a healthy BMI. Between 21.7% and 34.8% of cases met criteria for rapid response by treatment session eight. Overall, 54.5% of all cases met RCSI criteria by the end of treatment. The mean post-treatment EDE-Q global score (2.54) was within 1 standard deviation of the mean score for an age-matched normative sample (EDE-Q global score = 1.58, SD = 1.23).²⁸

Treatment duration and treatment outcomes

Bootstrap parameter estimates for the primary MANOVA analysis suggested that treatment duration was not significantly associated with

changes in EDE-Q global scores ($B = -0.06$, $SE = 0.35$, $p = 0.88$) or binge eating behaviors ($B = 1.22$, $SE = 1.02$, $p = 0.23$), but duration was positively associated with improvement in purging behaviors ($B = 3.47$, $SE = 1.54$, $p = 0.03$). As expected, higher baseline measures were significantly associated with changes in the corresponding dependent variables (main effects for baseline measures had positive coefficients and $p < 0.01$). Higher baseline binge eating behaviors were negatively associated with changes in purging behaviors ($B = -0.30$, $SE = 0.09$, $p < 0.01$). The contrasts between treatment modalities suggested that CBT was positively associated with improved EDE-Q global scores ($B = 0.70$, $SE = 0.33$, $p = 0.03$), but no differences were found between treatments in behavioral change measures (all $p > 0.05$). No other variables were significantly associated with treatment outcomes. Sensitivity analyses yielded the same results and association patterns, suggesting the results were not unduly influenced by extreme outliers or AN cases.

Analysis of rapid response

After adjusting for rapid response in secondary MANOVA analyses, the main effect for treatment duration was not significantly associated with changes in EDE-Q global scores ($B = -0.15$, $SE = 0.73$, $p = 0.84$), binge eating ($B = -0.76$, $SE = 2.68$, $p = 0.79$) or purging behaviors ($B = 3.35$, $SE = 3.01$, $p < 0.27$). Rapid response in EDE-Q global scores predicted improvement in the same outcome measure ($B = 1.69$, $SE = 0.28$, $p < 0.01$), but not in behavioral measures ($p > 0.05$). None of the rapid response variables were associated with changes in behavioral measures ($p > 0.05$). Contrasts between diagnostic groups or treatment modalities were not

statistically significant ($p > 0.05$) after adjusting for rapid response. Sensitivity analyses yielded the same pattern of results described above.

Treatment duration and BMI changes

After adjusting for baseline BMI, partial correlations between log-transformed sessions and BMI change scores were not statistically significant in the full sample of cases with available BMI data ($n = 32$, $r = 0.19$, $p = 0.30$), nor in the subsample of AN cases ($n = 22$, $r = 0.17$, $p = 0.47$).

DISCUSSION

The present study set out to examine the relationship between treatment duration and improvement in eating disorders pathology in community based psychological treatment. Approximately 1 in 2 patients in this sample met criteria for reliable and clinically significant improvement in EDE-Q global scores. Pre-post treatment effect sizes were moderate for behavioral changes ($d = 0.32$ to 0.41) and large for EDE-Q global scores ($d = 1.42$) and BMI gain in anorexia nervosa cases ($d = 0.89$). Overall, we found little evidence of associations between treatment duration and changes in attitudinal (EDE-Q global scores), behavioral (binge eating, purging) and physical (BMI) outcome indicators. Initial results suggested that longer duration of therapy may be associated with improvements in purging behaviors; however this was no longer significant after adjusting for rapid response variables.

There are a number of limitations that warrant attention. Sample size constraints precluded more detailed contrasts between each of the diagnostic categories. A considerable issue related to data which were not

missing at random, since approximately 20% of cases dropped out before completing final outcome measures. To overcome this potential problem, we took a rigorous approach to deal with missing data,^{25,30} applying a combination of multiple imputation and inverse probability weighting. We were also unable to determine the exact time-point (i.e., session) at which symptomatic changes occurred, given the infrequent assessment points.

To some extent, these findings reflect the narrative of clinical guidelines for the management of eating disorders,² which are not very specific or prescriptive about the expected duration of psychological care. It is difficult to predict how long treatment should last for individual patients and there is little evidence that longer treatments lead to better outcomes in eating disorder pathology. In spite of this, we note that in clinical practice the duration of treatment is often considerably lengthy. For example, a recent survey of 157 patients with eating disorders accessing cognitive behavioral interventions revealed that they received a mean of 43.82 sessions, ranging from 1 to 400.³¹ In particular, clinicians may be more inclined to offer lengthy treatment to AN cases.³¹ The current clinical wisdom suggests that AN cases require longer treatments to regain weight,^{1,2} and therefore attending to BMI change is of critical importance in addition to EDE-Q measures. However, we found no significant associations between treatment duration and BMI gain, although this small sample of AN cases may not be representative of more severe cases typically seen in inpatient settings.

Our findings converge with studies that have shown that early improvements in eating disorder pathology are predictive of post-treatment outcomes.⁴⁻⁹ However, our results indicate that specifically rapid response in EDE-Q global scores is a helpful prognostic indicator, whereas early changes

in binge eating and purging were not predictive of final treatment outcomes. Monitoring rapid response seems to be a clinically sensible strategy to inform the management of eating disorder pathology, consistent with evidence-based interventions for eating disorders⁸ and a stepped care approach.³²

These findings underline the importance of routine outcome measurement and the early review of treatment response. For example, patients who have shown rapid response in EDE-Q global scores during the first month or so are more likely to be responders, and thus an extension of treatment may be justifiable. On the other hand, cases without rapid response are much less likely to benefit from therapy, and considering obstacles to improvement (or alternative support options) at that early review point may be important. In summary, we recommend regular outcome monitoring and review of progress within the early stages of treatment to inform future treatment planning and to determine the duration of an outpatient episode of psychological therapy. Future research could assess the acceptability and clinical effectiveness of such a decision-making strategy in psychological care for eating disorders.

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References

1. American Psychiatric Association. Practice Guideline for the treatment of patients with eating disorders, third edition. APA, 2006.
2. National Institute for Health and Clinical Excellence. Eating disorders. Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders. CG9. London, UK: National Institute for Health and Clinical Excellence; 2004.
3. [Hay PP, Bacaltchuk J, Stefano S, Kashyap P. Psychological treatments for bulimia nervosa and bingeing. *Cochrane Database Syst Rev* 2009;7\(4\):CD000562. doi: 10.1002/14651858.CD000562.pub3.](#)
4. [Vall E, & Wade TD. Predictors of Treatment Outcome in Individuals with Eating Disorders: A Systematic Review and Meta-Analysis. *Int J Eat Disord* 2015;48\(7\):946–971.](#)
5. Doyle PM, Le Grange D, Loeb K, Doyle AC, Crosby RD. Early Response to Family-Based Treatment for Adolescent Anorexia Nervosa. *Int J Eat Disord* 2010;43:659–662.
6. Grilo CM, White MA, Wilson GT, Gueorguieva R, & Masheb RM. Rapid response predicts 12-month post-treatment outcomes in binge-eating disorder: theoretical and clinical implications. *Psychol Med* 2012;42:807–817.

7. MacDonald DE, Trottier K, McFarlane T, & Olmsted MP. Empirically defining rapid response to intensive treatment to maximize prognostic utility for bulimia nervosa and purging disorder. *Behav Res Ther* 2015;68:48–53.
8. Raykos BC, Watson HJ, Fursland A, Byrne SM, & Nathan P. Prognostic Value of Rapid Response to Enhanced Cognitive Behavioral Therapy in a Routine Clinic Sample of Eating Disorder Outpatients. *Int J Eat Disord* 2013;46:764–770.
9. Turner H, Bryant-Waugh R, & Marshall E. The impact of early symptom change and therapeutic alliance on treatment outcome in cognitive-behavioural therapy for eating disorders. *Behav Res Ther* 2015;73:165–169.
10. Howard KI, Kopta SM, Krause MS, & Orlinsky DE. The dose–effect relationship in psychotherapy. *Am Psychol* 1986;41:159–164.
11. Anderson EM, & Lambert MJ. A survival analysis of clinically significant change in outpatient psychotherapy. *J Clin Psychol* 2001;57:875–888.
12. Barkham M, Rees A, Stiles WB, Shapiro DA, Hardy GE, & Reynolds S. Dose–effect relations in time-limited psychotherapy for depression. *J Consult Clin Psychol* 1996;64:927–935.

13. Delgadillo J, McMillan D, Lucock M, Leach C, Ali S, & Gilbody S. Early changes, attrition, and dose–response in low intensity psychological interventions. *Br J Clin Psychol* 2014;53:114–130.
14. Hansen NB, & Lambert MJ. An Evaluation of the Dose–Response Relationship in Naturalistic Treatment Settings Using Survival Analysis. *Ment Health Serv Res* 2003;5(1):1-12.
15. Kadera SC, Lambert MJ, & Andrews AA. How much therapy is really enough? A session-by-session analysis of the psychotherapy dose–effect relationship. *J Psychother Pract Res* 1996;5:132–151.
16. Kopta SM, Howard KI, Lowry JL, & Beutler LE. Patterns of symptomatic recovery in psychotherapy. *J Consult Clin Psychol* 1994;62:1009–1016.
17. Maling MS, Gurtman MB, & Howard KI. The response of interpersonal problems to varying doses of psychotherapy. *Psychother Res* 1995;5:63–75.
18. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Press, 1994.
19. Fairburn CG, & Beglin SJ. Assessment of eating disorders: Interview or self-report questionnaire? *Int J Eat Disord* 1994;16:363–370.

20. Bohn K, Fairburn CG. The Clinical Impairment Assessment Questionnaire (CIA 3.0). In Fairburn C.G. (ed). Cognitive Behavior Therapy and Eating Disorders. New York: Guilford Press, 2008.
21. Luce KH, & Crowther JH. The reliability of the Eating Disorder Examination-Self-Report Questionnaire version (EDEQ). *Int J Eat Disord* 1999;25:349–351.
22. Mond JM, Hay PJ, Rodgers B, Owen C, & Beumont PJV. Validity of the Eating Disorders Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behav Res Ther* 2004;42:551–567.
23. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), 4th edition. American Psychiatric Press, Washington, 1994.
24. Schafer JL, & Olsden MK. Multiple imputation for multivariate missing-data problems: A data analyst's perspective. *Multivariate Behav Res* 1998;33:545-571.
25. Curtis LH, Hammill BG, Eisenstein EL, Kramer JM, & Anstrom KJ. Using Inverse Probability-Weighted Estimators in Comparative Effectiveness Analyses With Observational Databases. *Med Care* 2007;45:S103–S107.

26. Jacobson NS, & Truax P. Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *J Consult Clin Psychol* 1991;59(1):12–19.
27. Mond JM, Hay PJ, Rodgers B, Owen C. Eating disorder examination questionnaire (EDE-Q): norms for young adult women. *Behav Res Ther* 2006; 44:53–62.
28. Bardone-Cone AM, Harney MB, Maldonado CR, Lawson MA, Robinson DP, Smith R, Tosh A. Defining recovery from an eating disorder: Conceptualisation, validation, and examination of psychosocial functioning and psychiatric comorbidity. *Behav Res Ther* 2010; 48:194–202.
29. Couturier J, Lock J. What is recovery in adolescent anorexia nervosa? *Int J Eat Disord* 2006; 39:550–555.
30. Seaman S, & White I. Re-analysis using Inverse Probability Weighting and Multiple Imputation of Data from The Southampton Women's Survey. Cambridge: Institute of Public Health; 2008.
31. Cowdrey ND, & Waller G. Are we really delivering evidence-based treatments for eating disorders? How eating-disordered patients describe their experience of cognitive behavioral therapy. *Behav Res Ther* 2015;75:72–77.

32. Bower P, & Gilbody S. Stepped care in psychological therapies: access, effectiveness and efficiency: narrative literature review. *Br J Psychiatry* 2005;186:11–17.

Table 1. Pre- and post-treatment outcome measures

Outcome measure	Pre-treatment measure	Post-treatment measure	Pre-post effect size		Response rates	
	Mean (SD)	Mean (SD)	Cohen's <i>d</i>	95% CI	%RR	%RCSI
EDE-Q global score	4.31 (1.24)	2.54 (1.45)	1.42	1.19, 1.65	34.8	54.5
EDE-Q binge eating	7.04 (10.29)	2.82 (4.72)	0.41	0.25, 0.57	31.3	
EDE-Q purging	7.28 (11.54)	3.54 (7.21)	0.32	0.17, 0.48	21.7	
BMI (AN + EDNOS)	17.96 (1.82)	18.87 (2.15)	0.49	0.22, 0.75		
BMI (AN)	17.02 (1.09)	18.03 (1.79)	0.89	0.45, 1.33		

Notes: EDE-Q = eating disorder examination questionnaire; EDE-Q binge eating and purging = number of episodes in the last month; BMI = body mass index; AN = anorexia nervosa sample; EDNOS = sample of atypical anorexia nervosa cases with healthy range BMI; SD = standard deviation; CI = confidence intervals; RR = rapid response in the relevant measure by treatment session 8, for cases with at least 9 treatment sessions; RCSI = reliable and clinically significant improvement in EDE-Q global scores