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ADDING COGNITIVE THERAPY ELEMENTS TO EXPOSURE THERAPY FOR OBSESSIVE COMPULSIVE DISORDER: A CONTROLLED STUDY

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Abstract. Thirty-five outpatients (25 women, 10 men) with a DSM-III-R principal diagnosis of OCD accepted exposure treatment at a psychiatric outpatient clinic. They were randomly assigned to one of two individual treatments for a 6-week exposure therapy treatment based on a treatment manual or to a 6-week waiting list condition. The 12 patients assigned to the waiting list were subsequently randomly assigned to one of the active treatments. Both treatment groups received in vivo or imaginal exposure in each of the 10 twice-weekly treatment sessions held after two assessment sessions. One group ($n = 16$) received cognitive therapy interventions for comorbidity problems or to alter beliefs underlying patients' OCD. The other group ($n = 19$) received relaxation training as an attention placebo control. Both groups received relapse prevention follow-up contacts. Twenty-seven patients completed intensive treatment. Both treatments overall showed satisfactory levels of clinical improvement and large effect sizes. ANCOVAS for treatment completers showed non-significantly lower levels of OCD symptoms, depression and state anxiety in the treatment condition that did *not* include cognitive interventions. The patients receiving additional cognitive therapy showed significantly lesser dropout than those in the other treatment condition, but there were no significant differences in the intention-to-treat analyses.

Keywords: Obsessive Compulsive Disorder, Cognitive Behaviour Therapy, adult, cognitive techniques, between groups design.

Introduction

Consensus-based guidelines (March, Frances, Carpenter, & Kahn, 1997) have recommended exposure with response prevention (ERP) with or without the addition of cognitive therapy techniques (CT) in the treatment of obsessive compulsive disorder (OCD). According to a recent stringently empirically-based review (DeRubeis & Crits-Christoph, 1998), ERP was considered demonstrated as efficacious and with specific effects in the treatment of OCD, while the evidence suggested that CT alone was possibly efficacious.

Enthusiasm for ERP is dampened somewhat by the high rates of refusal (around 25%) of this treatment option among OCD patients (Kozak, 1999). If one adds patients that drop out or show a negative response to treatment, up to 50% of OCD patients seeking exposure treatments do not achieve a satisfying result (Steketee, 1994). Therefore there is still great

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interest in attempting to improve the outcomes of treatment for OCD. One possibility is through the addition of specific cognitive therapy interventions. This may be fruitful since comorbidity is so common in OCD (Steketee, 1993). It is reasonable to assume that the general philosophy of the CT model that has proven effective in the treatment of depressive disorders, anxiety disorders and personality disorders (Beck, Emery, & Greenberg, 1985; Beck, Rush, Shaw, & Emery, 1979) may reduce dropout by addressing patients' other comorbid symptoms and other personality-related beliefs. Recent cognitive models for OCD have emphasized the employment of specific cognitive interventions that may improve treatment outcomes by "loosening-up" beliefs that help to maintain OCD-symptoms (Salkovskis, 1985; Salkovskis & Kirk, 1997). Cognitions such as beliefs have sometimes been informally addressed during the course of standard exposure treatments, but this has not been done in a systematic manner with all patients (Foa & Kozak, 1997; Steketee, 1993). If these beliefs are explicitly dealt with, it seems reasonable that chances of relapse may be reduced. This is also in line with one cognitive behavioural model of the mechanisms thought to be involved in successful exposure treatment (Foa & Kozak, 1986). The addition of a general cognitive orientation and particularly the use of socratic questioning could allow patients more time to process the "meaning" of their exposure experiences.

Several different types of cognitive therapy techniques can be applied. The first study found in the literature (Emmelkamp, Helm, Zanten, & Plochg, 1980) found that training in substitution of positive coping self-statements did not improve outcomes in treating OCD. Use of rational challenges based on techniques from rational-emotive therapy (Ellis, 1962) produced equally good outcomes in two studies as compared to self-controlled exposure (Emmelkamp, Visser, & Hoekstra, 1988; Emmelkamp, 1991). More relevant to the current study, the latter study did not find that adding cognitive techniques improved outcomes over the application of each treatment modality separately.

One study has demonstrated the effectiveness of adding cognitive interventions with OCD (Oppen et al., 1995). Cognitive techniques added to exposure (called "behavioural experiments") proved superior to self-exposure instructions alone. These cognitive techniques were based on Beck's cognitive therapy model (Beck, 1976) in particular as applied to OCD by Salkovskis (1985). Another study added relapse prevention-based interventions after intensive exposure therapy (Hiss, Foa, & Kozak, 1994). These cognitive interventions included interventions directed at the relapse process (Marlatt & Gordon, 1985) as well as interventions directed at irrational thoughts and cognitive distortions related to OCD symptoms. Those receiving cognitive interventions showed less relapse of treatment gains than a control condition.

In another study, OCD patients with obsessive thoughts, but not overt compulsions, were successfully treated (in comparison to a waiting list control condition) with cognitive restructuring techniques added to exposure with response prevention techniques (primarily the use of loop tapes) (Freeston et al., 1997). In addition to a cognitive model of obsessions being presented, four types of dysfunctional appraisal common to OCD were targeted as appropriate to the individual case: 1) over importance of thoughts and magical thinking; 2) exaggerated responsibility for negative consequences; 3) perfectionist demands for certainty and control: and 4) inflated estimates of the probability and severity of consequences of feared events. Relapse prevention strategies were also followed to promote maintenance of gains.

One recent study (McLean et al., 2001) compared ERP in a group format with a group cognitive behavioural therapy (CBT) intervention focusing on the testing of the faulty

appraisals and beliefs common to OCD similar to those of the Freeston et al. study (1997). ERP treated patients were slightly superior to those receiving CBT at posttreatment, and showed superior outcomes at 3-months follow-up. Both treatments were significantly improved compared to a waiting list condition and showed improvements on one (of seven) cognitive belief measures, reflecting reductions in patients' exaggerated responsibility for feared negative consequences.

Research that attempts to test the comparative benefits of cognitive interventions versus exposure-based interventions for OCD faces a difficult task. Exposure therapy interventions are among the most powerful means for changing cognitions as well as behaviours in the anxiety disorders (Wells, 1997). A recent review (Abramovitz, Franklin, & Foa, unpublished manuscript) found that studies where ERP was utilized alone produced effect sizes of 1.50, while studies utilizing exclusively cognitive procedures produced effect sizes of only 1.19. The latter result was characterized as similar to that of a "substandard" exposure. However, as yet there have been few studies employing cognitive procedures directed towards comorbidity and specific OCD-related beliefs. Thus future refinements in cognitive techniques may improve on these rates.

In the current study, it is hypothesized that adding elements of cognitive therapy (versus adding elements of progressive muscle relaxation (REL) training) to standard manualized exposure treatment for OCD will improve treatment outcomes for patients in two ways: 1) Minimizing the disturbing effects of comorbidity should help maintain patient motivation and thus increase the acceptability of ERP procedures and reduce dropout. 2) Identification and reality-testing of dysfunctional cognitions underlying OCD should increase treatment efficiency both during treatment and during follow-up and should produce fewer signs of relapse (Salkovskis & Kirk, 1997).

In the current study the cognitive interventions would be flexibly tied to individualized case formulations of each patient's disorder and thus less controlled. Randomization still allows an empirical test of the usefulness of such a "principle-driven" application of cognitive techniques, in line with suggestions made by some critics of traditional highly controlled clinical research (Persons & Silberschatz, 1998).

Progressive muscle relaxation has been thought to be of no benefit in the treatment of OCD, and has been employed as attention placebo control in research on OCD treatment (Fals-Stewart, Marks, & Schafer, 1993).

Method

Participants

Fifty-four patients were referred consecutively to a psychiatric outpatient clinic in a major city of Norway from 1993 to 1998. Patients were included in the study if they met the DSM-III-R criteria for OCD according to the Anxiety Disorders Interview Schedule-Revised (DiNardo & Barlow, 1988), and if OCD was considered the principal diagnosis. Patients were excluded if they met criteria for a lifetime history of psychotic disorder, alcohol or drug addiction. Patients were excluded from the study for the following reasons: obsessions without compulsions ($n = 4$), another axis I diagnosis was considered primary ($n = 5$), unstable acting-out or suicidal behaviour ($n = 2$), psychosis ($n = 1$), chronic (over 30 years) ego-syntonic OCD ($n = 1$), and subclinical OCD ($n = 2$).

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Thirty-nine patients met inclusion criteria and were offered treatment. Four patients (three females, one male) refused the offer of treatment. Thus the study consisted of 35 patients (25 females and 10 males) with a principal diagnosis of OCD. They had a mean age of 35.7 years ($SD = 12.1$).

Patients on stable doses of anti-obsessional medications were allowed in the study if they agreed to maintain dosage levels unchanged during the study. Twelve patients (34%) were taking such medications, anti-obsessional SSRIs ($n = 6$). clomipramine ($n = 4$), and other tricyclic anti-depressive medications ($n = 2$). Nine of them were being maintained on clinically adequate dosages.

All patients completed a written informed consent form describing the research conditions. A detailed research protocol for the study had been approved by the Regional Ethics Committee for research with human subjects. The ADIS-R interviews were performed by the first author. The inter-rater reliability of axis I diagnoses was assessed by using a paired-rater design. All intake interviews and 12-month follow-up interviews were videotaped. Twelve randomly selected videotaped ADIS-R interviews were independently scored by another clinical psychologist. The agreement rate for an OCD diagnosis was 100%. Kappa values for the various other axis I diagnoses varied from 0.89 to 1.0. The kappa value for a principal diagnosis of OCD was 0.80.

Since personality disorder comorbidity might influence treatment outcome, assessment of personality disorders was performed using the Structured Clinical Interview for DSM-III-R personality disorders (SCID-II; Spitzer, Williams, Gibbon, & First, 1990). The SCID-II interviews were performed by the first author. The prevalence of at least one Axis-II disorder was 66%. The inter-rater reliabilities of the various clusters of the Axis-II diagnoses were assessed by means of a paired-rater design. Five randomly selected videotaped SCID-II interviews were rated by another clinical psychologist. The kappa value for the presence of at least one current personality disorder was 1.00. The kappa values for the presence of any Cluster A personality disorder were .40, for Cluster B was 1.00, and for Cluster C was 1.00.

Treatments

Those accepting treatment in the study were first either randomly assigned to a waiting list (WL) condition or directly to one of two individual ERP-based treatments, both based on adaptations of a treatment manual (Foa, 1991). The second author, who was blind to the treatment assignments, prepared slips of paper with equal numbers of the three initial treatment conditions (WL, ERP+REL, and ERP+CT). He then placed one of them inside each of the envelopes, sealed the envelopes and then shuffled their order. When patients were accepted for treatment, the next available envelope was drawn and opened. If the slip inside specified assignment to waiting list, the patient was given an appointment after the 6-week waiting period, otherwise the patients were assigned directly to start in one of the two active treatments. The envelope and enclosed slip was then discarded. This method for randomization thus utilized sampling without replacement to help ensure that approximately equal proportions of patients were assigned to the different conditions.

After 6 weeks, waiting list patients were assigned directly to one of the two active treatments by use of another set of envelopes similarly prepared by the second author. These envelopes contained slips of paper with only one of the two active treatments listed on them. These procedures produced similar numbers of patients assigned to each treatment at each stage of

treatment and thus the randomization technique employed appears adequate. Treatment was without charge. Those terminating treatment were offered alternative treatment-as-usual that did not include exposure therapy, which was unavailable.

Two-hour sessions were held twice weekly for 6 weeks in both treatment groups (24 hours total). Two assessment sessions resulted in the determination of an exposure hierarchy. In session one, a habituation-based rationale from the Foa manual was presented to both treatment groups. The importance of the completion of the hierarchy items, adherence to rules for response prevention, and home exposure exercises were all rooted in the habituation rationale for both treatment groups. The cognitive interventions were presented in one rationale (ERP+CT) as additional aids to increase motivation for the completion of exposure exercises and to prevent dropout and relapse by addressing comorbid problems that may co-exist with the OCD. Relaxation exercises were added to the habituation rationale of the other group (ERP+REL) and presented as aids to completing the stressful exposure exercises and also as potentially beneficial in dealing with other comorbid symptoms of anxiety and depression. A measure of treatment expectancy, based on a commonly used measure of treatment expectancy (Borkovec & Nau, 1972), was administered immediately after presentation of the rationale for treatment. This was before any dropouts had occurred and revealed that the two treatment groups both had highly positive expectations to treatment outcome for themselves in both conditions (rated from 0 – 100% confidence). The mean for ERP+REL was 81.8% ($s = 19.9$) and for ERP+CT was 74.7% ($s = 19.4$); a non-significant difference between groups.

This was followed by 10 sessions during which in vivo and/or imaginal exposure were applied. Modifications of response prevention guidelines to minimize dropout were followed (Steketee, 1993). OCD ritualistic behaviours that had been the subject of in-session exposure were prohibited. Situations or objects high up on the patient's hierarchy could be avoided until they had been addressed in-session. Guidelines for "normal" standards of washing and checking behaviour were followed throughout treatment. Brief handwashing was permitted before meals and after the use of toilets, as well as daily 10-minute showers, and normal checking (once only) of locks and electrical appliances. However, these situations were to be avoided for at least 2 hours after treatment sessions or after home exposure exercises. In line with the Foa-manual, violations of the response prevention guidelines were discussed at the start of each treatment session. Most patients attempted to meet the response prevention guidelines.

Some patients experienced significant increases in other comorbid symptoms (panic, depression, substance abuse) during their treatment. These symptoms were given brief attention within the treatment sessions of both treatment conditions. Many of these patients dropped out of treatment. Of those completing treatment, one patient had a relapse of alcohol abuse problems, but was able to complete treatment (ERP+CT). One patient who became increasingly suicidal was removed from therapy (ERP+REL condition).

Adherence and treatment integrity

Written records were available from most treatment protocols. Lowered N due to missing data is listed under each analysis. This allowed an unambiguous estimate of how many minutes of the actual sessions were devoted to exposure exercises. Significantly less total time for exposure exercises (minutes per session) was utilized in the ERP+CT condition ($N = 13$,

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$M = 31$, $SD = 5.6$) than in the ERP+REL condition ($N = 10$, $M = 52$, $SD = 12.2$; F (1, 22) = 29.8, $p < .001$, two-tailed).

A minimum of two home exposure exercises were assigned on a written form after each treatment session (sessions 3–11) and the results of these exercises were then discussed at the start of the next treatment session. There were no significant differences between the two treatment groups as to how many home exposure exercises were completed. by the ERP+CT group members ($N = 15$, $M = 17.3$, $SD = 10$) than by the ERP+REL participants ($N = 10$, $M = 20.6$, $SD = 7.2$, F (1, 24) = .8, n.s.).

One group (ERP+CT, $N = 16$) had a minimum of 30 minutes per session available for addressing case-specific comorbidity or specific OCD-related beliefs, through application of general cognitive therapy techniques or through the application of cognitive therapy (CT) techniques specific for the treatment of OCD. The introduction of these elements of cognitive therapy was not manualized, but flexibly applied according to the specifics of the case in hand, in accordance with an individual case conceptualization approach (Persons, 1991). Inspection of written therapy protocols, supplemented in some cases with videotapes of treatment sessions, allowed an estimate of what percent of the cognitive interventions were specific to OCD, and what percent were aimed at non-OCD comorbid symptoms. The distribution of this measure was highly bimodal. For 10 of the 15 patients completing treatment, 80% or more of the cognitive therapy interventions they received were directed at OCD-related underlying beliefs and appraisals (Salkovskis & Warwick, 1985; Salkovskis & Kirk, 1989; Oppen & Arntz, 1994) or directed at aspects of patients' "core beliefs" (Beck, Freeman, & Associates, 1990; Young, 1990). For the other five patients, this percent was 43% or less due to the presence of disturbing comorbidity.

Patients in the other treatment condition (ERP+REL, $N = 19$) received 30 minutes of relaxation training each session. The training was based on elements of a treatment manual (Bernstein & Borkovec, 1973). This training was within-session progressive muscle relaxation and release-only relaxation exercises, which have been shown to have a neutral effect on OCD symptoms in previous research (Fals-Stewart et al., 1993). No home practice of relaxation was prescribed as this would mean that one treatment had two different types of homework assignments to follow and would produce a potential confound.

To minimize dropout in the follow-up period, all patients were offered on a volunteer basis monthly (15-minute) telephone follow-ups for relapse prevention and support. This intervention followed guidelines derived from the Stages of Change model (Prochaska & DiClemente, 1984). Briefly this model emphasizes patient self-attribution of responsibility for treatment gains and includes motivational techniques designed to increase patients' self-efficacy when faced with temptations to relapse. The two treatment conditions did not differ significantly in the degree to which they utilized this option (ERP+CT, $N = 11$, $M = 1.6$, $SD = 2.5$, ERP+REL, $N = 9$, $M = 3.3$, $SD = 2.8$, F (1, 19) = 2.2, n.s.) Almost all patients met for 60-minute supportive follow-up sessions (without further exposure therapy) at 3, 6, 9, and 12 months posttreatment. Some of these follow-ups were performed over telephone for the four patients who had moved from town, but all attended the final 12-month follow-up.

Therapists

Three very experienced therapists (11, 15, and 17 years of experience) administered both types of therapy. All three were experienced in cognitive and behavioural (ERP) interventions. All

therapists had formal training in cognitive therapy (2, 5, and 6 years of training); two were certified supervisors in cognitive therapy and the third was in supervisor training. Sessions were taped and weekly supervision given.

Measures

The following instruments were used to measure treatment outcome at all assessment points (prior to treatment condition assignment, mid-therapy, post-treatment, 3-, 6-, and 12 month follow-ups): the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) (Goodman et al., 1989), The Beck Depression Inventory (BDI) (Beck & Steer, 1987), and Spielberger State-Trait Anxiety Inventory-S Anxiety scale (STAI-S) (Spielberger, Gorsuch, Lushene, Vagg, & A, 1983).

Y-BOCS interviews with 5 obsession and 5 compulsion items (0–4 point ratings) were conducted by the first author for all patients pretreatment and at 12-month follow-ups. Only total scores from Y-BOCS were utilized. Therapists conducted Y-BOCS interviews at all other points of assessment. Therapist-ratings were audio- or videotaped. Difficult YBOC ratings were discussed with the first author. This occurred with very few cases and involved only minor, if any, adjustments in the Y-BOC ratings. Another clinical psychologist, blind to treatment condition and treatment outcome, re-rated a random selection of 16 Y-BOCS pretreatment ratings and seven Y-BOCS 12-month follow-up ratings. The inter-rater agreement with Kendall's tau-b statistic for pretreatment Y-BOCS total scores was .66 ($p < .001$, two-tailed) and for Y-BOCS 12-month follow-up ratings was .95 ($p < .001$, two-tailed). This indicates a satisfactory level of inter-rater agreement for both sets of ratings.

BDI is a self-report inventory with 21 items (0–4 point ratings) rating depressive symptoms for the past week. STAI-S is a self-report inventory with 20 items (1–4 point ratings) rating current symptoms of state anxiety. Both measures have been shown to be highly reliable and valid and are often used as measures of change in treatment outcome studies. Patients completed both measures immediately after being rated on the Y-BOCS.

Statistics

Pretreatment group differences were tested by independent *t*-tests or chi-square statistics as appropriate. Two-tailed tests and standard significance levels were employed. On all tests of the results of the treatment interventions, a Bonferroni-correction was applied because three measures of treatment effect were employed. This was usually the case for almost all of the analyses reported in the Results section. Thus a minimum alpha level of $p < .017$ was required for significance.

Patients completing a WL condition were compared to patients directly entering active treatment at posttreatment on Y-BOCS, BDI, and STAI-S using one-way ANCOVAS with pretest values as covariates. This was followed up with appropriate post hoc tests.

A two-way (treatment group x time) repeated measures ANCOVA was performed using pretest values as covariates both for intention to treat data and for completers. Estimates of effect size were also obtained for these measures. An indication of the clinical significance of treatment effects was obtained through application of the methods outlined by (Jacobsen & Truax, 1991).

Results

Active treatments versus waiting list

ANCOVAS, with pretreatment measures as covariates, indicated significant main effects for completers at posttreatment on Y-BOCS: F (2, 29) = 25.43, $p < .017$, and on STAI-S: F (2, 25) = 5.14, $p < .017$, but not on BDI: F (2, 24) = 3.97, $p < .05$, n.s. Post hoc comparisons showed that patients in both active treatments were significantly lower than patients in WL on Y-BOCS ($p < .017$), but that only patients in the ERP+REL treatment showed significantly less elevation on STAI-S than those in WL ($p < .017$). Y-BOCS posttreatment means were as follows: ERP+REL = 10.1 ($n = 7$), ERP+CT = 13.6 ($n = 10$), and WL = 25.2 ($n = 12$). BDI posttreatment means were: ERP+REL = 5.8 ($n = 6$), ERP+CT = 7.8 ($n = 9$), and WL = 19.3 ($n = 9$). STAI-S posttreatment means were: ERP+REL = 30.3 ($n = 6$), ERP+CT = 41.5 ($n = 10$), and WL = 49.1 ($n = 9$). Similar results were obtained using intention to treat criteria, except that there was no longer a significant main effect at posttreatment on STAI-S.

The WL patients, who were virtually unchanged on Y-BOCS, BDI, and STAI-S, were subsequently randomly assigned to either the ERP+REL or the ERP+CT treatment conditions, and their data added to the patients who had entered active treatment directly for the purposes of all further analyses reported here.

Initial pretreatment characteristics

There were no significant differences between the two treatment groups at pretreatment. Table 1 lists the demographic, diagnostic, and dependent variables that were tested.

Intention to treat analyses

Since one of the main hypotheses of this study was based on the prevention of dropouts and relapse, the intention to treat data analysis should be of primary interest. The results for both active treatments were analysed according to intention to treat criteria. Patients with missing data at any point during treatment or follow-up were assigned the scores from the last measurement obtained from them (the last observation carried forward procedure).

Seven ERP+REL patients and one patient from ERP+CT discontinued treatment. There were no dropouts from the WL-condition. This differential dropout rate was significant (Fisher's Exact Probability Test, $p < .05$, two-tailed). After dropout, chi-square tests showed that significantly more patients with Cluster B personality disorder were now present in the cognitive therapy condition, and that patients in that treatment group were significantly younger ($p < .05$, two-tailed). There was also a trend suggesting that younger females dropped out more often from the ERP+REL condition ($p = .10$, two-tailed).

Table 2 displays the means, and standard deviations for all patients on Y-BOCS, BDI, and STAI-S at all assessment points. The effects sizes following treatment are also displayed utilizing a formula derived from (Cohen, 1988).

Within group effect sizes on the Y-BOCS were large and in general quite similar for both treatment groups both at posttreatment and all follow-up assessments. On BDI means, both groups produced medium sized effects at posttreatment as well as at all follow-ups. For the STAI-S group means, both groups displayed small to medium effects at posttreatment and at follow-ups.

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Table 1. Pretreatment status on demographic, diagnostic, and dependent measure variables for the two treatment conditions in means (standard deviations) or in percents (N)

Variable	ERP+CT	ERP+REL	Significance test
Age in years	31.4 (10.4)	39.3 (12.6)	$t = 1.98$, $p < .10$
Female gender	56% (9)	84% (16)	n.s.
Marital status			n.s.
Single	31% (5)	26% (5)	
Married/Cohab.	56% (9)	68% (13)	
Divorced/Wid.	13% (2)	13% (2)	
OCD-subtypes			n.s.
Washing-cleaning	38% (6)	47% (9)	
Checking	31% (5)	32% (6)	
Div. covert rituals	31% (5)	16% (3)	
Hoarding	0% (0)	5% (1)	
Depressive disorder	38% (6)	53% (10)	n.s.
Dysthymi	31% (5)	47% (9)	n.s.
Any anxiety disorder	56% (9)	74% (14)	n.s.
Specific phobia	44% (7)	42% (8)	n.s.
General Anxiety Disorder	25% (4)	32% (6)	n.s.
Social phobia	19% (3)	32% (6)	n.s.
No other Axis I dis.	38% (6)	21% (4)	n.s.
Axis II: Cluster A	13% (2)	21% (4)	n.s.
Axis II: Cluster B	31% (5)	11% (2)	n.s.
Axis II: Cluster C	63% (10)	53% (10)	n.s.
Y-BOCS	24.9 (2.9)	23.7 (3.2)	n.s.
BDI	15.7 (6.7)	16.1 (10.6)[a]	n.s.
STAI-S	48.9 (8.7)	47.6 (12.5)[a]	n.s.

[a] $N = 18$ due to missing data.

During treatment. ANCOVAS on Y-BOCS, BDI, and STAI-S, with pretest values as covariates, were performed for all patients in the two treatment groups (ERP+REL, for Y-BOCS, $n = 19$, and for BDI and STAI-S, $n = 18$, ERP+CT, $n = 16$). There were no significant ($p < .017$, two-tailed) differences between treatments on Y-BOCS, BDI or STAI-S during intensive treatment.

During follow-up. Time by Group (3×2) repeated measures ANCOVAS using pretreatment values as covariates, were run for all three measures of treatment effect for all the patients. No significant effects for Group, Time, or Group $\times$ Time interaction were found on any of the measures.

Treatment completers analyses

Table 3 displays the means, and standard deviations for treatment completers at all assessment points for Y-BOCS, BDI, and STAI-S. The effects sizes following treatment are also displayed utilizing a formula derived from (Cohen, 1988).

The identical analyses performed with the intention to treat data were performed with treatment completers of the two treatment groups (ERP + REL, $n = 12$ for Y-BOCS, $n = 11$

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Table 2. Number, means, standard deviations, and effect sizes for ERP+CT ($N = 16$) and ERP+REL ($N = 19$ for Y-BOCS, $N = 18$ for BDI and STAI-S): Intention to treat

	ERP+CT			ERP+REL		
Variable	*M*	(*SD*)	ES	*M*	(*SD*)	ES
Y-BOCS						
Pretreatment	24.9	(2.9)		23.7	(3.2)	
Midtreatment	18.4	(5.2)		19.3	(5.2)	
Posttreatment	16.7	(7.2)	1.49	16.1	(7.3)	1.35
3-month F.U.	14.4	(7.0)	1.96	17.3	(7.1)	1.16
6-month F.U.	14.3	(7.1)	1.96	17.8	(5.7)	1.28
12-month F.U.	14.8	(8.3)	1.62	15.4	(8.1)	1.35
BDI						
Pretreatment	15.7	(6.7)		16.1	(10.6)	
Midtreatment	13.3	(7.9)		12.8	(8.4)	
Posttreatment	10.6	(9.2)	.63	9.7	(8.3)	.67
3-month F.U.	9.4	(9.3)	.78	10.8	(6.0)	.55
6-month F.U.	10.4	(10.4)	.61	10.7	(8.1)	.57
12-month F.U.	11.2	(9.7)	.54	9.2	(8.8)	.71
STAI-S						
Pretreatment	48.9	(8.7)		47.6	(12.5)	
Midtreatment	51.2	(11.5)		44.4	(9.7)	
Posttreatment	45.8	(12.8)	.28	38.8	(11.9)	.72
3-month F.U.	41.6	(13.6)	.64	43.2	(14.0)	.33
6-month F.U.	43.9	(15.2)	.40	42.3	(13.3)	.41
12-month F.U.	46.1	(13.9)	.24	38.7	(14.0)	.67

for BDI and STAI-S, ERP+CT, $n = 15$). The pattern of the results of the ANCOVA analyses both during treatment and during follow-up were similar to that found in the intention to treat analyses so these statistics are not reported in detail.

Treatment completers showed trends to lower levels of OCD symptoms, depression and state anxiety in the treatment condition that did not include cognitive interventions at mid- and posttreatment assessments, but none of these differences were significant due to the Bonferroni correction employed.

Clinical improvement analyses

To evaluate the number of patients in the total sample that showed clinically significant improvement at posttreatment and at follow-up, the methods of Jacobson and Truax (1991) were followed to determine three criteria of clinical improvement. Based on the nonpatient Y-BOCS data obtained in a previous study (Steketee, Frost, & Bogert, 1996), and as described in a recent study (Abramowitz, Foa, & Franklin, 2003), it was possible to estimate the number of patients who achieved end-state functioning within the nonpatient distribution of Y-BOCS total scores (cutoff score $c < 16$). A second criteria of reliable change was obtained based on the test-retest reliability of Y-BOCS ($r = .79$). Patients achieving both criteria of improvement were considered to have met criteria for clinically significant improvement. Table 4 depicts

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Table 3. Number, means, standard deviations, and effect sizes: treatment completers

	ERP+CT				ERP+REL			
Variable	*N*	*M*	(*SD*)	ES	*N*	*M*	(*SD*)	ES
Y-BOCS								
Pretreatment	15	25.1	(2.8)		12	23.4	(3.5)	
Midtreatment	15	18.3	(5.3)		12	16.3	(3.9)	
Posttreatment	15	16.4	(7.3)	1.57	12	11.3	(4.0)	3.20
3-month F.U.	15	14.0	(7.0)	2.09	12	13.3	(5.4)	2.24
6-month F.U.	14	12.7	(5.9)	2.66	11	13.7	(2.7)	3.06
12-month F.U.	14	13.2	(7.7)	2.06	12	10.2	(4.7)	3.18
BDI								
Pretreatment	15	16.2	(6.6)		11	16.0	(11.8)	
Midtreatment	15	13.7	(8.1)		11	9.9	(7.8)	
Posttreatment	14	9.1	(7.3)	.97	11	5.6	(4.3)	1.17
3-month F.U.	14	7.8	(7.1)	1.17	11	7.4	(6.0)	.92
6-month F.U.	14	8.9	(8.9)	.93	10	6.8	(5.2)	1.02
12-month F.U.	14	9.3	(8.4)	.86	11	4.7	(4.9)	1.25
STAI-S								
Pretreatment	15	48.3	(7.9)		11	48.7	(12.9)	
Midtreatment	15	51.3	(11.9)		11	42.7	(9.7)	
Posttreatment	14	43.3	(10.3)	.46	11	34.4	(9.4)	1.27
3-month F.U.	14	38.6	(10.2)	1.00	11	41.5	(15.1)	.51
6-month F.U.	14	41.2	(13.1)	.66	10	39.4	(14.3)	.70
12-month F.U.	14	44.6	(11.7)	.34	11	34.3	(13.3)	1.35

Table 4. Number of patients (and %) for each treatment condition achieving nonpatient endstate functioning, reliable change, and clinical significant improvement (both types of improvement): on Y-BOCS at posttreatment, 3- and 12 month follow-ups

Treatment group assessment	Nonpatient distribution *n* (%)	Reliable change *n* (%)	Clinical significance *n* (%)
ERP+CT			
Posttreatment	6 (38)	10 (63)	6 (38)
3-month F.U.	9 (56)	11 (69)	9 (56)
12-month F.U.	9 (56)	10 (63)	9 (56)
ERP+REL			
Posttreatment	10 (53)	12 (63)	10 (53)
3-month F.U.	7 (37)	10 (53)	7 (37)
12-month F.U.	10 (53)	12 (63)	10 (53)

the number of patients in both treatment groups achieving these three types of improvement at post-treatment, 3-month and 12-month follow-up (intention to treat criteria).

No significant differences on chi-square analyses between the two treatment conditions were found at any of the assessment periods.

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Treatment integrity analyses

To test for the potentially confounding effect of the differing amounts of in-session exposure administered by the two treatment conditions, a hierarchical multiple regression analysis was performed using posttreatment Y-BOCS as the dependent measure. In step 1, age and sex variables were entered, in step 2, Y-BOCS pretreatment scores were entered, treatment group was entered in step 3, in step 4, average minutes of within session exposure were entered, and finally in step 5, the interaction between treatment group and exposure session time was entered. None of the variables entered in the various steps lead to significant increases in the variance explained in posttreatment Y-BOCS scores of treatment completers except for the variables of age and sex that were entered in the first step (F change $(2, 20) = 3.52$, $p < .05$, two-tailed).

Discussion

Both active treatment conditions in this study produced significant reductions in OCD symptoms as compared to a waiting list condition. Overall, this study essentially found no significant differences in treatment outcome between the two active treatment groups. There was some support for hypothesis 1: patients receiving additional cognitive therapy were significantly less likely to drop out. However, adding CT interventions to ERP procedures did not show any significant advantage in terms of treatment efficiency over the ERP interventions to which relaxation exercises were added, as had been predicted by hypothesis 2. The intention to treat analyses showed essentially the same results as the completer-analyses.

Although there were significantly less dropouts in the treatment condition receiving additional cognitive therapy, the lack of significant differences in the intention to treat analyses seems to suggest that the patients remaining in treatment did not benefit additionally from receiving the cognitive interventions. Even though no overall differences were obtained between the two treatment conditions, one cannot rule out the possibility that subgroups of patients with certain comorbidity may have profited more from one of the treatment conditions in the present study than did patients with other characteristics. More Cluster B personality disorder patients dropped out of the standard ERP treatment condition. This tentatively suggests that the cognitive interventions may have aided in maintaining or enhancing the motivation to comply with the ERP procedures of a subgroup of patients that otherwise would have dropped out of treatment. This needs further investigation.

On the other hand, among treatment completers, the patients receiving additional cognitive therapy showed non-significant trends to higher levels of comorbid symptoms of anxiety and depression than for the patients receiving the standard ERP treatment, which seems to go against the first hypothesis of this study. The latter finding may have been accounted for by the unfortunately confounding effects of greater amounts of time being devoted to within-session exposure by the standard treatment. A previous study (Foa, Kozak, Steketee, & McCarthy, 1992) has shown that ERP administered with a pill placebo produced significant reductions in depression as measured by the BDI, and in anxiety as measured by STAI-S. It seems likely that in patient populations with primary diagnoses of OCD, ERP interventions that reduce OCD symptoms are indirectly helpful in reducing the symptoms of depression and anxiety that are secondary to the OCD.

It is important to note that the type of cognitive therapy interventions utilized in this study were primarily based on the general cognitive model of Beck (1976). Patient beliefs relating to

their OCD were derived socratically from the patients' exposure experiences during sessions and during home exposure exercises. Some critics have suggested that cognitive techniques may be misused by patients to "neutralize" exposure produced anxiety. In the same manner, the use of relaxation exercises after the exposure exercises could be subject to the same criticism. The large effect sizes produced in the current study by both treatments would seem to argue against that pessimistic interpretation.

The cognitive therapy interventions of the current study should be distinguished from more recent cognitive therapy interventions for OCD that are based on "stronger" cognitive models that provide their own cognitively-based explanations of the obsessive-compulsive symptoms as alternatives to habituation-based rationales. This appears intuitively to be a more powerful method to use when applying cognitive interventions to OCD. In the present study, the fact that the cognitive interventions were "tacked-on" to the habituation-based exercises could have weakened the importance patients attributed to the opportunities they had to test beliefs that may be crucial to maintaining their obsessive-compulsive symptoms. Some of these cognitively-based models of OCD (Rachman, 1997; Salkovskis, 1985; Salkovskis, Forrester, & Richards, 1998; Wells, 1997, 2002) have already received some empirical support (Cottraux et al., 2001; van Oppen et al., 1995). Future studies of cognitive interventions for OCD should probably be based on these models.

One of these new models was employed by Mark Freeston and his colleagues in a recent study of the treatment of pure obsessives (Freeston et al., 1997). In that study specific beliefs thought to underlie the obsessive symptoms received a primary focus and were tested with exposure-based exercises. In addition to having a primarily cognitive focus, that study differed from the current study in that the cognitive therapy was administered over a longer period of time (5 weeks in the current study versus and average of 19 weeks in Freeston et al.) Perhaps cognitive interventions require application over a long period to produce effects on OCD.

The very large effect size reductions produced by both treatments in obsessive-compulsive symptoms on the Y-BOCS, and the substantial reductions in symptoms of depression and anxiety produced on the BDI and STAI-S, were directly comparable in size to those produced by a twice-weekly intervention in another recent study (Abramowitz et al., 2003). This latter study had been performed at Edna B. Foa's clinic where staff receive excellent training and supervision in a ERP-manual for OCD similar to the one employed in the present study. The fact that comparable results were obtained from the present study suggests that the Foa manual, with or without the addition of loosely structured CT interventions, also has proven highly generalizable even when administered outside of an expert site.

The rates of clinically significant improvement obtained in the current study were also comparable to that found in the Abramowitz et al. study at follow-up. That study also found at posttreatment significantly higher rates of clinically significant improvement for a matched group of patients that had received a more intensive (5-days per week) intervention, but this difference was no longer significant at 3-month follow-up. It is also worth noting that the present study only had a 10% refusal rate as compared to the 25% rate of refusal common for intensive treatment at Foa's clinic (Kozak, 1999).

However, the present study has several methodological limitations that should be taken into consideration. First, the study only has informal data, from the written treatment protocols, to assess the integrity of both the behavioural and the cognitive interventions. The treatment condition employing the cognitive interventions did not follow specified clinical decision rules determining when specific cognitive treatment procedures should have been employed.

Secondly, the amount of time available for within session exposure was not equivalent in the two active treatments. Thirdly, BDI and STAI-S were employed as measures of depression and state anxiety, respectively. Other measures of depression and anxiety may have produced different results. Fourthly, the study did not employ an independent rater to assess the benefits of treatment. Fifthly, the study included a relatively small sample size, which may have increased the risk of Type II errors. However, the sample size was sufficient to detect a very *large* clinically significant effect reduction on the Y-BOCS (8-point) with a power of .83. This was considered satisfactory in order to test the effect of the innovative use of cognitive techniques versus the waiting list condition. Also the sample size proved sufficient to detect a significant dropout effect between the active treatments.

Despite the above limitations, the current study serves as a replication and extension of the Abramowitz et al. study and supports the use of a less intensive exposure-based treatment schedule that clearly would be more easily transferable to non-speciality treatment settings. Apparently the addition of cognitive interventions to exposure-based behavioural interventions does not increase its treatment efficacy, but this study tentatively suggests that cognitive interventions may be useful as an adjunct to exposure treatment for patients with certain types of comorbid psychopathology who would otherwise drop out of treatment.

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References

ABRAMOWITZ, J. S., FOA, E. B., & FRANKLIN, M. E. (2003). Exposure and ritual prevention for obsessive-compulsive disorder: Effectiveness of intensive versus twice-weekly treatment sessions. *Journal of Consulting and Clinical Psychology*, *71*, 394–398.

ABRAMOWITZ, J. S., FRANKLIN, M. E., & FOA, E. B. (1999). Empirical status of cognitive behavioral therapy for obsessive compulsive disorder: A meta-analytic review. Unpublished manuscript.

BECK, A. T. (1976). *Cognitive therapy of the emotional disorders*. New York: New American Library.

BECK, A. T., EMERY, G., & GREENBERG, R. L. (1985). *Anxiety disorders and phobias: A cognitive perspective*. New York: Basic Books.

BECK, A. T., FREEMAN, A., & ASSOCIATES. (1990). *Cognitive therapy of personality disorders*. New York: The Guilford Press.

BECK, A. T., RUSH, A. J., SHAW, B. F., & EMERY, G. (1979). *Cognitive therapy of depression*. New York: The Guilford Press.

BECK, A. T., & STEER, R. A. (1987). *Beck Depression Inventory: Manual*. San Antonio, TX: The Psychological Corporation, Harcourt Brace Jovanovich.

BERNSTEIN, D. A., & BORKOVEC, T. D. (1973). *Progressive relaxation training*. Champaign, IL: Research Press.

BORKOVEC, T. D., & NAU, S. D. (1972). Credibility of analogue therapy rationales. *Journal of Behavior Therapy & Experimental Psychiatry*, *3*, 257–260.

COHEN, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: LEA.

COTTRAUX, J., NOTE, I., YAO, S. N., LAFONT, S., NOTE, B., MOLLARD, E., et al. (2001). A randomized controlled trial of cognitive therapy versus intensive behavior therapy in obsessive compulsive disorder. *Psychotherapy and Psychosomatics*, *70*, 288–297.

DERUBEIS, R. J., & CRITS-CHRISTOPH, P. (1998). Empirically supported individual and group psychological treatments for adult mental disorders. *Journal of Consulting and Clinical Psychology*, *66*, 37–52.

DI NARDO, P. A., & BARLOW, D. H. (1988). *Anxiety Disorders Interview Schedule-Revised(ADIS-R)* (P. A. V. (Norwegian), Trans.). Albany, NY: Phobia and Anxiety Disorders Clinic, State University of New York.

ELLIS, A. (1962). *Reason and emotion in psychotherapy*. New York: Lyle-Stuart.

EMMELKAMP, P. M. G. (1991). Cognitive therapy with obsessive-compulsive patients: A comparative evaluation. *Behaviour Research and Therapy*, *29*, 293–300.

EMMELKAMP, P. M. G., HELM, V. D. M., ZANTEN, V. B., & PLOCHG, I. (1980). Contributions of self-instructional training to the effectiveness of exposure in vivo: A comparison with obsessive-compulsive patients. *Behaviour Research and Therapy*, *18*, 61–66.

EMMELKAMP, P. M. G., VISSER, S., & HOEKSTRA, R. (1988). Cognitive therapy vs. exposure in the treatment of obsessive-compulsives. *Cognitive Therapy and Research*, *12*, 103–114.

FALS-STEWART, W., MARKS, A. P., & SCHAFER, B. A. (1993). A comparison of behavioral group therapy and individual behavior therapy in treating obsessive-compulsive disorder. *The Journal of Nervous and Mental Disease*, *181*, 189–193.

FOA, E. B. (1991). *Therapist manual for exposure treatment of obsessive-compulsives* (P. A. V. & E. V., Norwegian Trans.). Philadelphia, PA: Medical College of Pennsylvania.

FOA, E. B., & KOZAK, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*, 20–35.

FOA, E. B., & KOZAK, M. J. (1997). *Mastery of Obsessive-Compulsive Disorder: Client Workbook*. New York: Graywind Publications.

FOA, E. B., KOZAK, M. J., STEKETEE, G. S., & MCCARTHY, P. R. (1992). Treatment of depressive and obsessive-compulsive symptoms in OCD by imipramine and behaviour therapy. *British Journal of Clinical Psychology*, *31*, 279–292.

FREESTON, M. H., LADOUCEUR, R., GAGNON, F., THIBODEAU, N., RHÉAUME, J., LETARTE, H., & BUJOLD, A. (1997). Cognitive-behavioral treatment of obsessive thoughts: A controlled study. *Journal of Consulting and Clinical Psychology*, *65*, 405–413.

GOODMAN, W. K., RASMUSSEN, S. A., PRICE, L. H., MAZURE, C., HENINGER, G. R., & CHARNEY, D. S. (1989). The Yale-Brown Obsessive-Compulsive Scale: Development, use, and reliability. *Archives of General Psychiatry*, *46*, 1006–1016.

HISS, H., FOA, E. F., & KOZAK, M. J. (1994). A relapse prevention program for treatment of obsessive-compulsive disorder. *Journal of Consulting and Clinical Psychology*, *62*, 801–808.

JACOBSEN, N. S., & TRUAX, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology*, *59*, 12–19.

KOZAK, M. J. (1999). Evaluating treatment efficacy for obsessive-compulsive disorder: Caveat practitioner. *Cognitive and Behavioral Practice*, *6*, 422–426.

MARCH, J. S., FRANCES, A., CARPENTER, D., & KAHN, D. A. (1997). The expert consensus guidelines series: Treatment of obsessive-compulsive disorder. *Journal of Clinical Psychiatry*, *58* (Suppl. 4).

MARLATT, G. A., & GORDON, J. R. (Eds.). (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York: Guilford Press.

MCLEAN, P. D., WHITTAL, M. L., THORDARSON, D. S., TAYLOR, S., SÖCHTING, I., KOCH, W. J., et al. (2001). Cognitive versus behavior therapy in the group treatment of obsessive-compulsive disorder. *Journal of Consulting and Clinical Psychology*, *69*, 205–214.

OPPEN, P. V., & ARNTZ, A. (1994). Cognitive therapy for obsessive-compulsive disorder. *Behaviour Research and Therapy*, *32*, 79–87.

OPPEN, P. V., HAAN, E. D., BALKOM, A. J. L. M. V., SPINHOVEN, P., HOOGDUIN, K., & DYCK, R. V. (1995). Cognitive therapy and exposure in vivo in the treatment of obsessive-compulsive disorder. *Behaviour Research and Therapy*, *33*, 379–390.

PERSONS, J. B. (1991). Psychotherapy outcome studies do not accurately represent current models of psychotherapy: A proposed remedy. *American Psychologist*, *46*, 99–106.

PERSONS, J. B., & SILBERSCHATZ, G. (1998). Are results of randomized controlled trials useful to psychotherapists. *Journal of Consulting and Clinical Psychology*, *66*, 126–135.

PROCHASKA, J. O., & DICLEMENTE, C. C. (1984). *The transtheoretical approach: Crossing traditional boundaries of therapy*. Homewood, IL: Dow Jones/Irwin.

RACHMAN, S. (1997). A cognitive theory of obsessions. *Behaviour Research and Therapy, 35*, 793–802.

SALKOVSKIS, P. M. (1985). Obsessional-compulsive problems: A cognitive-behavioural analysis. *Behaviour Research and Therapy*, *23*, 571–583.

SALKOVSKIS, P. M., FORRESTER, E., & RICHARDS, C. (1998). Cognitive-behavioural approach to understanding obsessional thinking. *British Journal of Psychiatry*, *173* (suppl 35), 53–63.

SALKOVSKIS, P. M., & KIRK, J. (1997). Obsessive-compulsive disorder. In D. M. Clark & C. G. Fairburn (Eds.), *Science and practice of cognitive behaviour therapy* (pp. 179–208). Oxford: Oxford University Press.

SALKOVSKIS, P. M., & WARWICK, H. M. C. (1985). Cognitive therapy of obsessive-compulsive disorder: Treating treatment failures. *Behavioural Psychotherapy*, *13*, 243–255.

SPIELBERGER, C. D., GORSUCH, R. L., LUSHENE, R., VAGG, P. R., & JACOBS, J. A. (1983). *Manual for the State-Trait Anxiety Inventory (form Y)*. Palo Alto, CA: Consulting Psychologists Press.

SPITZER, R. L., WILLIAMS, J. B. W., GIBBON, M., & FIRST, M. (1990). *Structured clinical interview for DSM-III-R – personality disorders*. Washington, DC: American Psychiatric Press.

STEKETEE, G., FROST, R., & BOGERT, K. (1996). The Yale-Brown obsessive-compulsive scale: Interview versus self-report. *Behaviour Research and Therapy*, *34*, 675–684.

STEKETEE, G. S. (1993). *Treatment of obsessive compulsive disorder*. New York: The Guilford Press.

STEKETEE, G. S. (1994). *When standard treatments for OCD don't work: From research to practice*. Paper presented at the 28th Annual AABT Convention, San Diego.

WELLS, A. (1997). *Cognitive therapy of anxiety disorders: A practice manual and conceptual guide*. Chichester, England: John Wiley & Sons.

WELLS, A. (2002). *Emotional disorders and metacognition: Innovative cognitive therapy*. Chichester, England: John Wiley & Sons Ltd.

YOUNG, J. E. (1990). *Cognitive therapy for personality disorders: A schema-focused approach*. Sarasota, FL: Professional Resource Exchange.