



COGNITIVE THERAPY AND EXPOSURE *IN VIVO* IN THE TREATMENT OF OBSESSIVE COMPULSIVE DISORDER

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Summary—The present study is the first controlled study that evaluates the effects of cognitive therapy along the lines of Beck (1976) [*Cognitive therapy and the emotional disorder*. New York: International University Press] and Salkovskis (1985) [*Behaviour Research and Therapy*, 23, 571–583] in obsessive compulsive disorder (OCD) and compares these effects with those of self-controlled exposure *in vivo* with response prevention. Seventy-one patients were randomly assigned to either cognitive therapy or exposure *in vivo*. In each treatment condition seven patients dropped out. Both treatments consisted of 16 sessions. Cognitive therapy as well as exposure *in vivo* led to statistically significant improvement. Multivariate significant differences suggesting a superior efficacy of cognitive therapy in comparison to exposure *in vivo* on the obsessive compulsive measures and on the measures for associated psychopathology. However, no univariate differences were found. Further, in both treatment conditions a considerable percentage of the patients was rated as “recovered”. Significantly more patients were rated as “recovered” in the cognitive therapy. The results show that this form of cognitive therapy is an effective treatment for OCD and suggest that cognitive therapy may be even more effective than exposure *in vivo*.

INTRODUCTION

Until the seventies obsessive compulsive disorder (OCD) was seen as a condition resistant to psychotherapy. During the last two decades several studies have shown that exposure *in vivo* plus response prevention is an effective treatment of OCD (Emmelkamp, 1982; Marks, 1987; Rachman & Hodgson, 1980; Steketee, 1993). In recent years, a substantial number of studies have demonstrated the effectiveness of cognitive therapy for anxiety disorders (e.g. Hollon & Beck, 1994; Margraf, Barlow, Clark & Telch, 1993). A relatively small number of studies have addressed the issue of cognitive treatment with obsessive compulsives. Until now only three controlled studies have investigated the effectiveness of cognitive therapy with OCD.

The first controlled study on the effect of cognitive therapy was conducted by Emmelkamp, van der Helm, van Zanten and Plochg (1980). This study investigated whether self-instructional-training (Meichenbaum, 1975) could enhance the effectiveness of exposure *in vivo* plus response prevention. In the cognitive restructuring group patients were trained to become conscious of the negative self-statements and then to substitute positive coping self-statements for the anxiety-engendering self-statements. The results of this study indicate that self-instructional training did not increase therapeutic efficacy (Emmelkamp *et al.*, 1980). However, Kendall (1983) doubted if self-instructional training is the most appropriate cognitive technique for patients who are already engaging in self-verbalizations, ruminations and doubting. Cognitive treatment that addresses deeper cognitive structures might be more effective in dealing with obsessive compulsive symptoms (McFall & Wollersheim, 1979).

In a following study (Emmelkamp, Visser & Hoekstra, 1988) the effects of rational emotive therapy (RET) along the lines of Ellis (1962) were compared with those of self-controlled exposure *in vivo* plus response prevention. In the RET group patients were trained to record their (irrational)

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thoughts and to challenge these irrational beliefs; behavioural experiments were not included. On the obsessive compulsive targets, and on social anxiety, RET was equally effective as self-controlled exposure *in vivo* plus response prevention. On depression, however, RET was more effective than self-controlled exposure *in vivo* plus response prevention.

Finally, a third study that evaluated the effects of cognitive therapy was reported by Emmelkamp and Beens (1991). They investigated two issues:

- (1) whether RET was as effective as self-controlled exposure *in vivo* plus response prevention; and
- (2) whether a combined package would be superior to exposure *in vivo* with response prevention.

No difference was found between RET and self-controlled exposure *in vivo* plus response prevention and the combined treatment was no more effective than self-controlled exposure alone.

To date, only one single case study (Salkovskis & Warwick, 1985) has illustrated the use of cognitive therapy according to the model of Beck and Salkovskis. The primary aim of the present study was to evaluate the effects of a version of cognitive therapy along the lines of Beck (1976) and Salkovskis (1985), specifically developed for OCD (van Oppen & Arntz, 1994) and to compare the effects of cognitive therapy with those of self-controlled exposure *in vivo* plus response prevention on obsessive compulsive targets and on more generalized measures for associated psychopathology. The secondary aim of this study was to investigate the efficacy of six sessions of cognitive therapy without behavioural experiments vs six sessions of exposure *in vivo* with response prevention without discussing the expectations of consequences in OCD. This version of cognitive therapy aimed at modifying: (i) overestimation of danger; and (ii) inflated responsibility. Previous studies found self-controlled exposure as effective as therapist-controlled exposure (Emmelkamp & Kraanen, 1977; Emmelkamp, van Linden van den Heuvell, Ruphan & Sanderman, 1989). Therefore we used self-controlled exposure *in vivo* instead of therapist-controlled exposure *in vivo* in this study.

METHOD

Design

Approximately half of the patients took part in a multicentre trial comparing fluvoxamine, behaviour therapy and cognitive therapy, results of which will be presented elsewhere. The number of patients in the cognitive therapy and exposure *in vivo* conditions in this multicentre trial was increased by patients who did not want to participate in a drug-trial, by patients who were treated with anti-depressants in the preceding year and by patients who had been put on a waiting list (8 weeks) ($n = 25$). Univariate analyses of variance (ANOVAs) or non-parametric tests did not reveal significant differences for the pretreatment dependent variables or for the demographic variables between these two patient-groups. All patients in the multicentre-trial and in the present project were randomly assigned to conditions.

Before the treatment a pretest was held, after 6 sessions an intermediate test (=midtest) took place, and after 16 sessions (16 weeks) a posttest was held. In case all the symptoms of a patient disappeared before the end of the 16 treatment sessions and he/she has had at least nine treatment sessions, a posttest was held and these patients were included in this study ($n = 3$).

Subjects and procedure

The inclusion criteria were:

- (1) a primary DSM-III-R (American Psychiatric Association, 1987) diagnosis of OCD;
- (2) not only obsessions;
- (3) age between 18 and 65;
- (4) a duration of OCD for at least one year;
- (5) absence of an organic mental disorder, mental retardation or a psychotic disorder;
- (6) no cognitive treatment or behavioural treatment in the preceding six months;
- (7) no use of anti-depressants.

Patients were either referred to our departments by general practitioners and by mental health agencies or responded to newspaper articles describing the availability of a treatment program for OCD. Seventy-one patients were included in this study. Fourteen patients dropped out; seven in the cognitive therapy condition (20%) and seven in the self-controlled exposure *in vivo* condition (19%). Patients were screened by a Dutch version of the standardized Anxiety Disorder Interview Schedule [ADIS; Bouman, Scholing, Emmelkamp & Dijkstra (1987); DiNardo, O'Brien, Barlow, Waddell & Blanchard (1983)] by an experienced clinical psychologist or an experienced psychiatrist.

The characteristics of the patients who completed the trial are presented in Table 1. Patients were treated as outpatients at the psychiatric outpatient clinic at the Reinier de Graaf Hospital Delft ($n = 20$) or at the outpatient clinic for anxiety disorders at the Psychiatric Centre Amsterdam ($n = 37$).

Therapists

Therapists were five clinical psychologists and one psychiatrist (3 male and 3 female). All six therapists conducted therapy in both treatment conditions. All six therapists were versed in behaviour therapy and had experience with behavioural treatment of OCD. Prior to the start of the study therapists received training in cognitive therapy for OCD.

Treatment

Both treatments consisted of 16 sessions of approx. 45 min. For both conditions treatment manuals were developed and used to guide the treatment. Weekly group sessions were held with the therapists, where problems that occurred during the treatments were discussed. All therapy sessions were audiotaped and the audiotapes were partly overheard regularly in the weekly group sessions.

Cognitive therapy. The cognitive therapy for OCD used in this study was essentially based on cognitive therapy for depression and anxiety disorders (Beck, 1976; Beck, Emery & Greenberg, 1985; Salkovskis, 1985). The general strategies are: firstly, to consider the intrusions as stimuli; secondly, to identify the distressing thoughts (negative automatic thoughts); thirdly, to challenge these automatic thoughts; and fourthly, to change these thoughts to non-distressing thoughts and finally, the underlying dysfunctional assumptions were looked for, these beliefs were challenged in the therapy sessions, and this also had to be done by the patient as homework assignments. Care was taken that two potentially bias reasoning styles were addressed, i.e. overestimation of danger and inflated personal responsibility. An important general cognitive technique used by the therapist to challenge the automatic thoughts is the Socratic Dialogue (Beck, 1976). Patients were also instructed to monitor and challenge automatic thoughts in diaries as homework assignments. In the therapy sessions problems with the diary were discussed and worked out, and in each session at least one automatic thought was challenged. After session six behavioural experiments were introduced. The behavioural experiments were mainly used to test out the empirical basis of the dysfunctional assumptions [for further details see van Oppen & Arntz (1994)].

Table 1. Patients characteristics

	Cognitive therapy ($n = 28$)	Exposure <i>in vivo</i> ($n = 29$)
<i>Sex</i>		
Men	46%	48%
Women	54%	52%
<i>Mean age*</i>	34.1 (10.8)	35.3 (10.1)
<i>Marital status</i>		
Single	29%	55%
Living together/married	64%	38%
Divorced/widow	7%	7%
<i>Education</i>		
Low	21%	31%
Medium	61%	45%
High	18%	24%
<i>Duration of complaints*</i>	11.7 (9.8)	14.4 (11.7)

*In years; standard deviation in parentheses.

Self-controlled exposure in vivo with response prevention. In the first treatment session information about the rationale of exposure with response prevention was given. An inventory of the obsessive compulsive avoidance behaviour and of the compulsions was made. After this a hierarchy of obsessive compulsive situations which were mostly avoided or triggered rituals were constructed. All these situations were noted and at each session a number of items of the hierarchy were given as homework tasks. Each session started with a discussion about the performance of the previous homework tasks and new homework tasks were discussed at length; after this patient and therapist agreed on which homework assignment had to be done before the next session. All patients determined the speed at which they worked through the hierarchy by him- or herself. In the first six sessions care was taken that the expectations of disastrous consequences were not discussed. Treatment essentially consisted of two components: self-exposure *in vivo* and self-imposed response prevention [for further details see Emmelkamp (1982)].

Measures

In this study the following measures were completed:

(1) The *Padua Inventory-Revised* [PI-R; Sanavio (1988); Sternberger & Burns (1990); van Oppen (1992); van Oppen, Hoekstra & Emmelkamp (1995b)] consists of 41 self-rated items; range 0–164. All items are rated on a 5-point scale from 0 = not at all to 4 = very much. The PI-R provided 5 subscales: (i) impulses; (ii) washing; (iii) checking; (iv) rumination; and (v) precision. The validity, reliability and the sensitivity to change of the PI-R are satisfactory (van Oppen *et al.*, 1995b; van Oppen, Emmelkamp, van Balkom & van Dyck; 1995a).

(2) The *Yale Brown Obsessive Compulsive Scale* [Y-BOCS; Goodman, Price, Rasmussen, Mazure, Fleischmann, Hill, Heninger & Charney (1989)] is a clinician-rated scale, consisting of ten items to assess OCD symptom severity; range 0–40. The therapist scores the items from 0 = no symptoms to 4 = severe symptomatology, based on a structured interview. There are two subscales: one for severity of obsessions and one for severity of compulsions. In this study only the total score is used. The reliability and convergent validity of the Y-BOCS are satisfactory (Goodman *et al.*, 1989; Woody, Steketee & Chambless, 1994). The Y-BOCS is a sensitive treatment outcome measure (van Oppen *et al.*, 1995a).

(3) The *Anxiety Discomfort Scale* (ADS) is an adapted version of the Watson and Marks (1971) scales. Five main obsessive compulsive targets are rated on a 9 (0 = no uneasiness to 8 = extreme fear/tension) point scale for anxiety/discomfort by the patient. Thus, the situations differ from patient to patient. The total score is the sum of the score on the 5-items. This scale is sensitive to change after treatment in obsessive compulsives (Emmelkamp, 1982; van Oppen *et al.*, 1995a). In this study the ADS was completed by the patient, an assessor and the therapist. Factor-analysis on these three variables at pretest demonstrated that one factor explained 75.2% of the variance. So, in this study we pooled the 3 scores and divided the sum by three.

(4) The revised version of the *Symptom Checklist* [SCL-90-R; Derogatis (1977)] is a multi-dimensional self-report inventory of current psychopathology. Items are rated on a 5 point scale (scores range from 90 to 450).

(5) The *Beck Depression Inventory* [BDI; Beck, Ward, Mendelson, Mock & Erbaugh (1961)] consists of 21-items. Items are rated on a 4 point scale (range 0–63). The total score measures the severity of depressive symptoms. In a Dutch population the validity of this questionnaire is satisfactory (Luteijn & Bouman, 1988).

(6) The *Irrational Belief Inventory* [IBI; Koopmans, Sanderman, Timmerman & Emmelkamp (1994)] is a 50-item questionnaire based on the Irrational Belief Test (Jones, 1968) and the Rational Behavior Inventory (Shorkey & Whithman, 1977). Items are rated on a 5 point scale (1 = strongly disagree to 5 = strongly agree; range 50–250). The total score of the IBI is a index of general irrational beliefs of the patients.

Data analyses

Univariate analyses of variance (ANOVAs) or non-parametric tests were run in order to evaluate the significance of the differences between both conditions on the pretest scores and demographic variables.

Treatment results were analysed with a two (group) by three (time) split-plot design using multivariate and univariate analysis of variance. Two MANOVAs were run: one with the obsessive compulsive measures (Y-BOCS, PI-R and ADS) and the other with the generalized measures (BDI and SCL-90-R) to assess generalization of treatment effects. In case of a multivariate significant time effect ANOVAs for the individual outcome measures were run. One ANOVA with the cognitive measure (IBI) was run to assess cognitive changes. Pre- to posttest paired *t*-tests, with Bonferroni correction, were used to assess changes in time within individual conditions.

Significant multivariate interaction effects were further analysed with ANCOVAs, with the pretest as covariate.

Further, the effect sizes (ES) for improvement on the three obsessive compulsive measures were calculated with the following formula (Cohen, 1988; Shavelson, 1981):

$$ES = \frac{x_2 - x_1}{\sqrt{s_1^2 + s_2^2 - 2r_{12}s_1s_2}}$$

where x_1 = pre-scores; x_2 = post-scores; s_1 = standard deviation of pre-scores; s_2 = standard deviation of post-scores; r_{12} = Pearson correlation between pre- and post-scores.

Jacobson and colleagues (Jacobson, Follette & Revensdorf, 1984; Jacobson & Truax, 1991) suggested a statistical approach to assess the clinical significance of results achieved. A subject according to Jacobson and colleagues is "recovered" when they met the two-fold criterion for clinically significant change; (i) the reliable change index; and (ii) the determination of recovery (Jacobson & Truax, 1991). Since calculation of change and determination of recovery are complementary procedures (Steketee & Chambless, 1992), we investigated both the reliable change index and the determination of recovery.

To determine statistically reliable change Jacobson and his colleagues (1984) proposed a formula for a Reliable Change index (RC), later modified by Christensen and Mendoza (1986). Recently Hageman and Arrindell (1993) proposed a further adjustment of the RC index by improving the pre-post difference scores (RC_{ID}) taking into account regression to the mean. To investigate the change which has occurred after treatment we used this latter RC_{ID} index.

The RC_{ID} should be > 1.96. This RC_{ID} classifies subjects according to whether or not they had improved reliably, independently of the clinical significance of this improvement. So, in addition to the RC_{ID} we determined whether the change was clinically significant (determination of recovery). Given the nature of the Y-BOCS and the ADS, norms for non-clinical samples cannot be determined. Therefore we determined the cutoff point for clinically significant change at two standard deviations from the mean of the OCD patients in this study (Jacobson & Truax, 1991). For the PI-R data from a normal sample are available (van Oppen, 1992). When the functional and dysfunctional distributions are only slightly overlapping as in our case, Jacobson and Truax (1991) recommend a cutoff point at two standard deviations from the mean of the functional population. Finally, non-parametric tests were run to compare the percentage of recovery of patients between the conditions.

RESULTS

Pretreatment characteristics

Results at the pretest are presented in Table 2. The univariate analyses of variance (ANOVAs) did not reveal significant differences for the pretreatment dependent variables except for the ADS. The pretest score of the ADS was significantly higher in the exposure condition [$F(1,55) = 3.9$, $P = 0.05$]. No significant differences were found between the cognitive and exposure condition on the demographic variables, duration of complaints [$F(1,55) = 0.9$, $P = 0.34$], marital status ($\chi^2 = 4.3$, $df = 2$, $P = 0.11$), age [$F(1,55) = 0.2$, $P = 0.67$], gender ($\chi^2 = 0.0$, $df = 1$, $P = 0.89$) and education ($\chi^2 = 1.4$, $df = 2$, $P = 0.48$). In both conditions the average symptom duration was > 11 years and the gender ratio was nearly 1:1 (see Table 1). This is comparable to other studies [see for reviews Marks (1987); Steketee (1993)].

Table 2. Descriptive results (means and standard deviations) for cognitive therapy and exposure

		Pretest	Midtest	Posttest
Obsessive compulsive measures				
<i>ADS*</i>				
Cognitive	<i>n</i> = 28	28.7 (5.3)	18.9 (8.8)	13.4 (9.4)
Exposure	<i>n</i> = 29	31.4 (5.0)	23.8 (7.1)	17.9 (9.0)
<i>PI-R</i>				
Cognitive	<i>n</i> = 28	72.6 (25.6)	62.7 (27.9)	51.5 (27.8)
Exposure	<i>n</i> = 29	69.9 (19.6)	63.3 (22.7)	58.1 (23.5)
<i>Y-BOCS</i>				
Cognitive	<i>n</i> = 28	24.1 (5.5)	19.3 (9.0)	13.3 (8.5)
Exposure	<i>n</i> = 29	25.4 (7.0)	19.2 (7.5)	17.3 (8.3)
Generalized measures				
<i>SCL-90-R</i>				
Cognitive	<i>n</i> = 28	197.1 (70.8)	184.7 (59.2)	166.3 (58.8)
Exposure	<i>n</i> = 29	192.8 (47.3)	169.3 (38.2)	178.3 (48.7)
<i>BDI</i>				
Cognitive	<i>n</i> = 27	16.3 (10.1)	14.3 (10.1)	11.3 (8.7)
Exposure	<i>n</i> = 29	16.6 (7.9)	13.3 (6.3)	12.9 (8.5)
Cognitions measure				
<i>IBI</i>				
Cognitive	<i>n</i> = 27	158.3 (18.0)	152.4 (14.2)	149.6 (21.1)
Exposure	<i>n</i> = 28	163.2 (19.1)	156.5 (13.8)	159.2 (17.4)

Standard deviation in parentheses.

*Therapist, assessor and patient ratings are pooled and divided by three.

Overall and differential treatment effects

Descriptive results for both treatments on the outcome variables are shown in Table 2 and Fig. 1. Results of evaluation MANOVA's assumptions (Tabachnick & Fidell, 1989) of normality, linearity and multicollinearity were satisfactory. MANOVAs revealed a significant multivariate time effect on the obsessive compulsive measures (Y-BOCS, PI-R and ADS) [$F(6,50) = 32.9, P < 0.000$] and

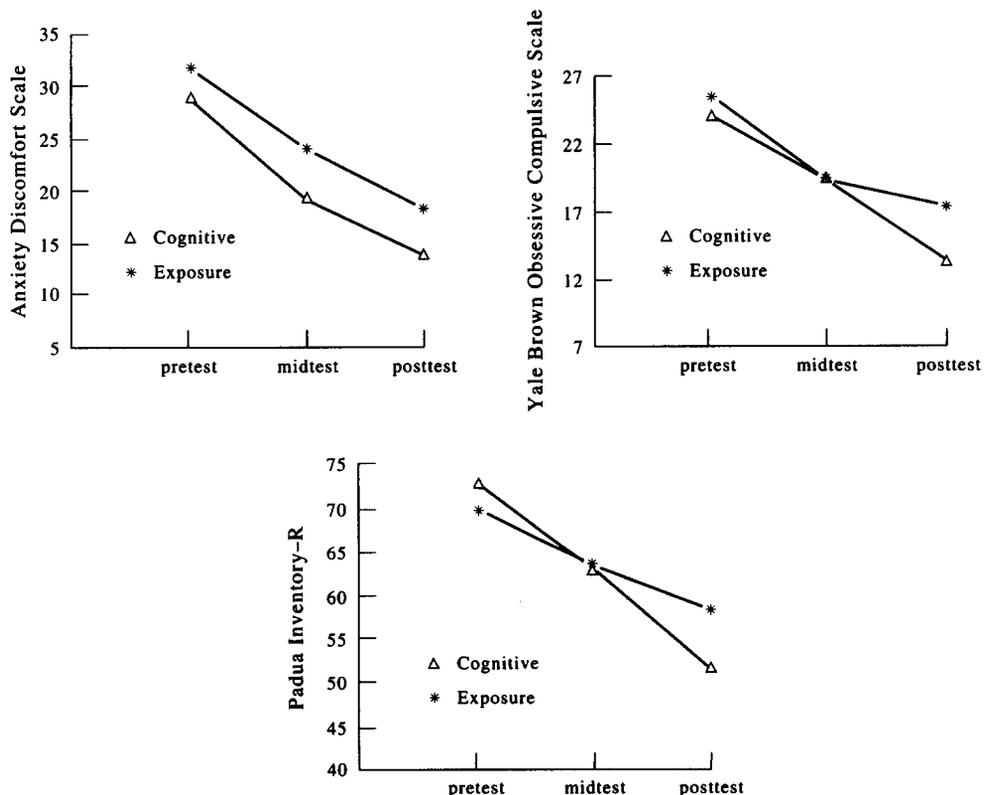


Fig. 1. Improvements on the obsessive compulsive measures for both conditions.

Table 3. ANCOVAs for differential treatment effects with the pretest as covariate

	Pretest-midtest	Pretest-posttest
ADS	$F(1,54) = 2.26, P = 0.14$	$F(1,54) = 0.97, P = 0.33$
Y-BOCS	$F(1,54) = 0.22, P = 0.64$	$F(1,54) = 2.56, P = 0.12$
PI-R	$F(1,54) = 0.32, P = 0.58$	$F(1,54) = 2.57, P = 0.12$
SCL-90-R	$F(1,54) = 1.57, P = 0.22$	$F(1,54) = 1.54, P = 0.22$
BDI	$F(1,54) = 0.51, P = 0.48$	$F(1,53) = 0.61, P = 0.44$

Table 4. Effect sizes for improvement

	Cognitive therapy ($n = 28$)	Exposure <i>in vivo</i> ($n = 29$)
ADS	1.90	1.76
Y-BOCS	1.42	1.03
PI-R	0.92	0.63

on the generalized measures (SCL-90-R and BDI) [$F(4,51) = 5.6, P < 0.002$]. Subsequent ANOVAs yielded significant time effects on all measures. The ANOVA on the cognitive measure (IBI) also revealed a significant time effect [$F(2,53), P < 0.008$]. *Post hoc t*-tests were done to investigate changes between pre- and posttest in each treatment group separately, with alpha set at 0.025 (Bonferroni correction). Both conditions improved significantly on almost all variables. Only on the SCL-90-R ($t = 1.45, df = 28, P = 0.16$) and on the IBI ($t = 1.60, df = 28, P = 0.12$) no time effects were demonstrated in the exposure condition. Moreover, both MANOVAs also revealed a significant multivariate interaction effect on the obsessive compulsive measures [$F(6,50) = 2.7, P < 0.03$] and on the generalized measures [$F(4,51) = 3.1, P < 0.03$]. These results suggested a greater efficacy of cognitive therapy in comparison to exposure *in vivo*.

Results of evaluation ANCOVAs assumptions (Tabachnick & Fidell, 1989) of normality of sampling distributions, linearity, homogeneity of variance, homogeneity of regression and reliability of covariates were satisfactory. Significant interaction effects were further analysed with ANCOVAs, with the pretest score as covariate. These results can be seen from Table 3. Neither the ANCOVAs after the first treatment period nor the ANCOVAs after the second treatment period yielded significant differential treatment effects. Finally, no significant multivariate between group differences were found.

The effect sizes for improvement on the obsessive compulsive measures are presented in Table 4: these effect sizes were slightly higher in the cognitive therapy condition as compared to the exposure condition.

Reliable change and clinical significance

The RC_{ID} on the ADS was ≥ 3 , on the Y-BOCS ≥ 6 and on the PI-R > 9 . The cutoff point for clinically significant change (Jacobson & Truax, 1991) was for the ADS 19, for the Y-BOCS 12 and for the PI-R 53. Patients were rated as recovered when the score fulfilled the criteria for RC_{ID} as well as for a clinically significant change. For an overview of the percentages of the reliable change index and the recovered index on the obsessive compulsive measures see Table 5. As can be seen, the percentage of patients who fulfilled the reliable change index and the recovered index were for all measures higher in the cognitive therapy condition than in the exposure condition. Statistically significant differences between the treatment conditions were found between the percentage of patients who recovered on the PI-R ($\chi^2 = 5.2, df = 1, P < 0.03$), on the Y-BOCS ($\chi^2 = 4.1, df = 1, P < 0.05$) and on a minimum of two measures ($\chi^2 = 6.5, df = 1, P < 0.02$). A trend was found on percentage patients, who recovered on all measures ($\chi^2 = 3.4, df = 1, P < 0.07$), while no statistically significant difference was found on percentage patients who recovered on the ADS ($\chi^2 = 0.6, df = 1, P < 0.64$).

Table 5. Percentage reliable change index and the recovered index on the obsessive compulsive measures

	Reliable change		Recovered	
	Cognitive	Exposure	Cognitive	Exposure
PI-R	75	62	57	31
Y-BOCS	75	66	50	28
ADS	96	90	68	62
On a minimum of two measures	75	72	61	48
On all three measures	68	48	39	17

Subgroups of OCD

Post-hoc a number of explorative analyses were done. Firstly, we investigated whether there are differences in efficacy between cognitive therapy and exposure *in vivo* for the subgroup of “checkers”. Several experiments (Röper, Rachman & Hodgson, 1973; Röper & Rachman, 1976; Rachman & Hodgson, 1980) demonstrated the role of responsibility in compulsive checking, but the sense of responsibility seemed less relevant in the case of cleaners (Rachman, 1993a). The issue of responsibility was explicitly dealt with in our cognitive therapy condition (van Oppen & Arntz, 1994). So, we expected that the subgroup checkers would profit more from cognitive therapy than from exposure *in vivo*. Patients were rated by the first three authors to which category they belonged. These categories were: (a) washers; (b) checkers; (c) a combination of washing and checking; and (d) other obsessive compulsive complaints. Seven patients were rated as “checkers” in the cognitive therapy condition vs ten patients in the exposure *in vivo* condition. *Post-hoc* ANCOVAs, with the pretest as covariate, did not reveal any significant between group differences on the three obsessive compulsive measures for the subcategory “checkers”. These analyses revealed a trend on the Y-BOCS [$F(1,14) = 3.4, P < 0.10$], in favour of cognitive therapy.

Secondly, we investigated whether there were differences in efficacy between cognitive therapy and exposure *in vivo* on the subscales obsessions and compulsions of the Y-BOCS. It has been noted in the literature that cognitive therapy may be effective in dealing with obsessions (Emmelkamp, 1987; Rachman, 1993b; Salkovskis & Warwick, 1985; Salkovskis & Westbrook, 1989). However, ANCOVAs revealed no significant differences in efficacy between the treatment conditions on the Y-BOCS-obsessions [$F(1,52) = 0.0, P = 0.44$], nor on the Y-BOCS-compulsions [$F(1,51) = 0.5, P = 0.49$].

Drop-outs

In each treatment conditions 7 patients dropped out: 20% in the cognitive therapy and 19% in the exposure *in vivo* group. The reasons for drop-outs were: a lack of improvement ($n = 5$), the depressive symptoms became more important than the obsessive compulsive symptoms ($n = 2$), the symptoms disappeared before sessions nine ($n = 2$), practical reasons unrelated to the treatment ($n = 3$), or remained unclear ($n = 2$). The ANOVAs did not reveal significant differences on the outcome variables between completers and drop-outs at pretest. Further, no significant differences on demographic variables were found between completers and drop-outs. The analyses revealed only a trend on the variable duration of complaints [$F(1,69) = 3.0, P < 0.09$]. The completers had a mean duration of complaints of 12.7 year ($SD = 10.8$) and the drop-outs of 7.6 year ($SD = 4.7$).

Further, all drop-outs were assessed on the Y-BOCS at the last treatment session. Because 2 patients dropped out after the first session, data (minimal 2 Y-BOCS scores) was available for 12 drop-outs. A *post-hoc* paired *t*-test revealed significant improvement for the drop-outs on the Y-BOCS ($t = 2.2, df = 11, P < 0.05$). No difference was found on the Y-BOCS of the drop-outs between cognitive therapy and exposure *in vivo*.

DISCUSSION

This is the first controlled study that demonstrates cognitive therapy along the lines of Beck and Salkovskis specifically developed for OCD (van Oppen & Arntz, 1994) to be an effective treatment for obsessive compulsive disorder. Both cognitive therapy and exposure *in vivo* led to statistically as well as clinically significant improvement. Patients treated with cognitive therapy improved significantly on all variables, and patients treated with exposure *in vivo* on almost all variables, except on the SCL-90-R and on the IBI. The demonstrated efficacy of exposure *in vivo* on the obsessive compulsive measures are in line with other recent outcome studies of exposure *in vivo* in OCD (Emmelkamp & Beens, 1991; Emmelkamp *et al.*, 1988).

Multivariate significant interaction effects on the obsessive compulsive measures and on the generalized measures suggested a greater efficacy of cognitive therapy in comparison to exposure *in vivo*. However, these differences did not stand up in separate ANCOVAs in which initial differences between conditions were taken into account. From Table 3 it can be seen that the ANCOVAs (with the pretest as covariate) revealed a trend on the Y-BOCS and on the PI-R in

favour of cognitive therapy. Possibly, the MANOVAs which consider dependent variables in combination were more powerful in determining significant differences than subsequent separate ANCOVAs. The differences in effect size and in the percentage of recovered patients suggest that cognitive therapy might be superior to exposure *in vivo*.

In recent years, composite variables are frequently used in treatment outcome studies to increase the statistical power. But, given the low correlations between the PI-R, the Y-BOCS and the ADS (van Oppen *et al.*, 1995a) we did not use a composite-measure because these measures seem to assess relatively different features of OCD.

In both conditions the effect sizes for improvement on the obsessive compulsive measures were large, except for the improvement on the PI-R in the exposure condition. The effect sizes on the obsessive compulsive measures were all larger in the cognitive therapy than in the exposure condition.

To address the issue of clinical relevance, both reliable change indexes and recovered indexes were determined. In both treatment conditions a considerable percentage of the patients demonstrated a reliable change and was rated as "recovered" on the PI-R, the Y-BOCS and on the ADS. Significantly more patients were rated as reliable changed or as "recovered" in the cognitive therapy on the PI-R, on the Y-BOCS and on a minimum of two measures. No significant differences were found on the ADS and the percentage of patients who recovered on all three measures. These findings are in line with the multivariate significant differences, suggesting a possible superiority of cognitive therapy in comparison to exposure *in vivo*.

In contrast to the present study, Emmelkamp and his colleagues (Emmelkamp *et al.*, 1988; Emmelkamp & Beens, 1991) did not find differences between cognitive therapy and exposure *in vivo* on the obsessive compulsive measures. Since the effects obtained for exposure *in vivo* are comparable to those found in other studies, these findings cannot be explained by a lower effect of exposure *in vivo*. In a meta-analysis of van Balkom, van Oppen, Vermeulen, Nauta, Vorst and van Dyck (1994) the mean effect size for exposure was 1.47, mostly determined by the ADS, which is roughly comparable to the effect sizes in the present study. Another explanation might be the large *ns* in both conditions in the present study. Large *ns* solve the power problem which has frequently plagued outcome studies in OCD. Finally, the differences in outcome between the present study and those by Emmelkamp and his colleagues might be related to the particular form of cognitive therapy used in this study. We used cognitive therapy along the lines of Beck and Salkovskis which also included behavioural experiments after session six, whereas Emmelkamp and his colleagues used RET (Ellis, 1962) and excluded all behavioural exercises. Further, in the cognitive therapy along the lines of Beck the Socratic dialogue is an important therapist's technique and attention is paid to idiosyncratic assumptions instead of the 11 basic beliefs of RET. And finally, the cognitive therapy used in this study, which was specifically developed for OCD, mainly focused on: (i) estimation of catastrophes and on (ii) the estimation of personal responsibility (van Oppen & Arntz, 1994).

The ANCOVAs after the first treatment period (six sessions), did not reveal any significant differential treatment effects. Thus, cognitive therapy without behavioural experiments was as effective as self-controlled exposure *in vivo* with response prevention without cognitive elements. These findings are consistent with the findings of Emmelkamp and his colleagues (Emmelkamp *et al.*, 1988; Emmelkamp & Beens, 1991). It has to be noticed that Emmelkamp *et al.* (1988) found that the efficacy of cognitive therapy was greater than the efficacy of exposure *in vivo* on a measure of depression. Although we did not find similar results on the BDI, the multivariate differences on the generalized measures are in line with these previous findings.

Does cognitive therapy change cognitions, whereas exposure *in vivo* does not? Only in the cognitive condition a significant time effect was found on the index of irrational cognitions, which supports the construct validity of cognitive therapy. However, this difference on the IBI was not statistically significant between the conditions. We found significant correlations between the improvement on the IBI on the one hand and improvement on the Y-BOCS ($r = 0.47$, $P < 0.001$); ADS ($r = 0.45$, $P < 0.001$) and on the PI-R ($r = 0.55$, $P < 0.001$) on the other. The same results were found by Emmelkamp and his colleagues (Emmelkamp & Beens, 1991; Emmelkamp *et al.*, 1988). Data from a normal sample demonstrated that this normal sample has less irrational cognitions (\bar{X} mean = 139.2, SD = 18.5 and \bar{Y} mean = 143.3, SD = 16.4) than the OCD sample in

this study (see Table 2) (Koopmans *et al.*, 1994). It has to be noticed that the score of the IBI an index is of general irrational beliefs. Additional research is needed to develop and to investigate measures which assess the more specific cognitions related to OCD.

Post-hoc comparisons did not reveal any significant difference between cognitive therapy and exposure *in vivo* on the obsessive compulsive measures for the subgroup "checkers". This may be due to the small *ns* for each subgroup per condition. Further, even though the overestimation of responsibility was an important focus of the cognitive therapy, other issues were also addressed such as the estimation of chance of catastrophe (van Oppen & Arntz, 1994). The responsibility issue deserves further empirical study. A challenging task for further research is to investigate which subgroup of OCD patients profit from which form of therapy. We further investigated whether there were differences between the efficacy of cognitive therapy and exposure *in vivo* on obsessions on the one hand and compulsions on the other. Explorative analyses did not reveal significant differences between the treatment conditions on the subscales obsessions and compulsions of the Y-BOCS. As patients with obsessions only were excluded from this study, an explanation for these findings could be that the obsessions of patients who have also compulsions are different from the obsessions of patients with only obsessions. Additional research is needed to investigate whether cognitive therapy is more effective than exposure *in vivo* with pure obsessional patients.

No significant differences were found between completers and drop-outs on demographic variables or pretest scores. Even though no significant differences were found on the duration of obsessive compulsive complaints, drop-outs appeared to have a shorter duration of obsessive compulsive complaints than completers ($P < 0.09$). Although Hansen, Hoogduin, Schaap and de Haan (1992) found at pretest a significantly lower score on a OCD measure we could not replicate those findings. The results of the improvement of the drop-outs are consistent with the recent findings of improvement of social phobic drop-outs (Scholing & Emmelkamp, 1993a, 1993b).

Taking these results together, this study demonstrates the efficacy of cognitive therapy along the lines of Beck (1976) and Salkovskis (1985). The present results suggest that this form of cognitive therapy, which also includes behavioural experiments, may be even more effective than exposure *in vivo*. However, the long term effects of exposure are well established (Steketee, 1993) whereas further studies are needed to investigate the long term effects of cognitive therapy with OCD.

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