

# Outcome Predictors in ERP-treatment of Patients with Obsessive Compulsive Disorder

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# OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

## Abstract

**Introduction:** Concentrated Exposure Treatment (cET) encompasses intensive forms of Exposure and Response Prevention (ERP) treatment for patients with an obsessive-compulsive disorder (OCD). Although predictor studies are available for less-intensive forms of OCD-treatment, such studies are not available for cET. This is the first study to assess predictors for cET-effectiveness.

**Method:** The effect of cET on OCD-scores was measured at post-treatment, 3-month, and 6-month follow-up using a repeated measures ANOVA. Pre-treatment OCD severity, depressive symptomatology, and age of onset were used as predictors of remission and treatment response in multiple binary logistic regression analyses, with treatment programme (4-day/8-day) as covariate.

**Results:** cET is associated with reducing OCD-severity post-treatment at all follow-up measurements ( $p < .0001$  for all measurements). For the predictors, pre-treatment OCD severity is not predictive of remission or treatment response. Higher depressive symptomatology is predictive of lower remission rates at post-treatment ( $p = .036$ ) and at 3-month follow-up ( $p = .036$ ). Early OCD-onset is predictive of higher treatment response at post-treatment ( $p = .012$ ). Predictors are not significant at other follow-up measurements.

**Discussion:** cET is an effective form of treatment for OCD, for which predictors appear to exist to a limited extent. Limitations exist on randomisation, available follow-up data, and recall bias on symptomatologic onset. Strict re-coding in accordance with consensus remission- and treatment response criteria results in data-loss. Replication studies are needed.

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

Obsessive-Compulsive Disorder (OCD) is a disorder encompassing the presence of obsessions and/or compulsions in an individual (American Psychiatric Association [APA], 2013). A national study from the United States showed a 2.3% lifetime prevalence of OCD, and a 1.2% 12-month prevalence of OCD (Ruscio, Stein, Chiu, & Kessler, 2010). Obsessions are characterised by unwanted thoughts, mental images, and/or mental impulses that patients try to suppress by using compulsions and/or safety behaviours (APA, 2013). Compulsions can entail both external behaviours such as extensive cleaning behaviours, and the rearrangement of objects; or internal behaviours such as repeating a particular sentence over and over in one's mind (APA, 2013; Hirschtritt et al., 2017). Examples of safety behaviours are asking others for reassurance and carrying cleaning material with oneself throughout the day. The insight of patients into their beliefs and behaviours associated with OCD could vary from good to non-existent (APA, 2013).

Psychological treatments of choice are Cognitive Behavioural Therapy (CBT) and especially Exposure Response Prevention (ERP; APA, 2007; Hirschtritt et al., 2017). There has not been a direct comparison between more concentrated, high intensity forms of OCD-treatment compared to weekly-administered ERP sessions (Kvale et al., 2018). However, researchers found that daily OCD-treatment with intensive exposure and ritual prevention for three weeks trends towards more improvement post-treatment than twice-a-week treatment for eight weeks (Abramowitz et al., 2003). Nevertheless, the difference in improvement favouring more intensive treatment did not hold on follow-up measurements (Abramowitz et al., 2003). More recently, Havnen, Hansen, Öst, and Kvale (2014) showed that four full consecutive days of their concentrated Exposure Treatment (cET) for OCD, including psycho-education and therapist-assisted exposure, resulted in recovery for 77% of their patients at 6-month follow-up measurement. This result was later replicated as they found that 60% of their patients remained in remission at 6-month follow-up measurement in a follow-up study (Havnen, Hansen, Öst, & Kvale, 2017; see also Kvale et al., 2018).

As cET is a relatively new form of treatment, no studies are available on possible treatment outcome predictors. Knowledge on predictors of treatment outcome could, however, yield various benefits in clinical decision-making. The potential benefits of using treatment predictors include better individual-based treatment assignment, the prevention of implicit assumptions in clinical decision making, and a better cost-benefit implementation of evidence-supported treatment by assigning the most optimal form of treatment to a particular patient (Bremer et al., 2018; Ryder, McDonough, Tosteson, & Lurie, 2009).

Given the aforementioned lack of studies on outcome predictors for cET, our

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

hypotheses on potential predictors are derived from studies on predictors in regular OCD-treatment. Most studies on regular OCD-treatment appear to address higher OCD-severity and comorbidity as predictors. In several studies, more severe OCD was associated with more comorbidity (Kempe et al., 2007), and was found to predict worse OCD-treatment outcomes (Knopp, Knowles, Bee, Lovell, & Bower, 2013; Kyrios, Hordern, & Fassnacht, 2015). Despite a relatively high degree of consensus on more severe OCD predicting less treatment effectiveness, diverging findings exist on various other predictors such as depression and onset (See also Knopp et al., 2013).

One of the predictors that has been investigated in relation to the effectiveness of OCD-treatment is comorbid depressive symptomatology. Studies on the comorbidity of depression and OCD have pointed towards one-third or more OCD-patients experiencing a comorbid depression, which makes depression an interesting potential predictor of treatment effectiveness given its prevalence (Pallanti, Grassi, Cantisani, Sarrecchia, & Pellegrini, 2011). The results on depressive symptomatology as predictor of OCD-treatment effectiveness are diverging, as some studies showed a negative impact of depressive symptomatology on OCD-treatment effectiveness (Overbeek, Schruers, Vermetten, & Griez, 2002) while others did not (Pallanti et al., 2011; see also Knopp et al., 2013). For example, Kempe et al.'s (2007) study instead showed more remission in patients with more severe comorbid depressive symptomatology, although the authors have urged caution due to small sample size and relatively low depressive symptomatology in their study. Apart from diverging findings, relatively little appears to be known about the specific role of depressive symptomatology on OCD-treatment outcomes (Pallanti et al., 2011). Arguably however, depressive symptomatology could inhibit learning, or limit the behavioural activation that is necessary for (engaging in) exposure and response prevention (Foa, 1979; Foa et al., 1983; See also Pallanti et al., 2011).

Aside from depressive symptomatology, another interesting potential predictor is the age of OCD-onset in OCD-patients. Onset has been considered an important factor in the conceptualisation of OCD-symptomatology, as aetiology is thought to differ on a stronger contribution of genetics in early-onset OCD (Anholt et al., 2014). It was found that early-onset is associated with a worse OCD-prognosis, as well as more co-morbidity with bipolarity and ADHD (Anholt et al., 2014). Other studies have pointed toward a poorer prognosis for early-onset OCD due to a higher illness persistency, more severe symptoms, and poorer treatment response (Dell'Osso et al., 2013). Studies have furthermore found that an earlier age of onset is related with a longer illness duration, while late-onset OCD-patients generally

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

endure a shorter illness duration prior to treatment itself (Dell'Osso et al., 2013). Similar to the diverging findings on depressive symptomatology as a predictor, diverging findings also exist for onset as a predictor of OCD-treatment effectiveness (Knopp et al., 2013). One study pointed towards a higher chance of remission for early-onset OCD after treatment (Kempe et al., 2007). Complicating research, there is no consensus on what threshold constitutes the onset of OCD itself (onset of symptoms versus onset of distressing symptoms; Anholt et al., 2014). Anholt et al. (2014) have also pointed out the limitation that no agreed-upon age cut-off between early/late-onset exists. Hence, and given the findings in their study, the authors have recommended a cut-off criterion of 20 years to differentiate between early-onset and late-onset OCD (Anholt et al., 2014).

Limitations do not only arise from a lack of studies on outcome predictors for cET and diverging findings on predictors of regular OCD-treatment. Another limitation is that patients with comorbidity are often excluded from studies, thus hindering our understanding of the association of comorbid symptomatology on treatment outcomes (Keeley, Storch, Merlo, & Geffken, 2007). In the present study, however, also patients with (highly) comorbid symptomatology are included in the treatment programme and monitored over time. With our study, we seek to investigate potential outcome predictors of treatment in outpatients with OCD assigned to a 4-day or 8-day cET-programme.

Based on the available literature on the aforementioned OCD-treatment predictors, several hypotheses will be investigated in this study. Our first hypothesis is that cET is significantly associated with Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) rated remission and/or symptom reduction at post-treatment measurement, 3-month, and 6-month follow-up measurement. This hypothesis is based on the effectiveness of Havnen et al.'s 4-day intensive treatment for OCD, incorporating exposure as key element in their treatment for OCD (Havnen et al, 2014; 2017).

Our second hypothesis, regarding our first predictor, is that a higher total level of pre-treatment OCD-severity as measured on the cumulative Dimensional Obsessive-Compulsive Scale (DOCS) scores is associated with lower rates of Y-BOCS-measured remission and/or symptom reduction. This is measured at post-treatment and on follow-up measurement. Higher pre-treatment OCD-severity at treatment onset has been associated with lower remission in the available literature (Knopp et al., 2013).

Our third hypothesis, regarding our second predictor, is that higher levels of depressive symptomatology as measured on Inventory of Depressive Symptomatology self-report (IDS-SR) are associated with lower rates of Y-BOCS-measured remission rates and/or

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

symptom reduction. Both are measured post-treatment and on follow-up measurement. This is based on the assumption that comorbid depressive symptomatology hinders the necessary behavioural activation (Foa, 1979, Foa et al., 1983).

Our fourth hypothesis, regarding our third predictor, is that an earlier age of OCD-onset is associated with lower rates of Y-BOCS-measured remission rates and/or symptom reduction. Similar to the other two predictors, this is measured at post-treatment and on follow-up measurement. This hypothesis is based on the earlier findings that treatment is less effective in patients with early-onset OCD (Dell’Osso et al., 2013), suffering from a worse general prognosis (Anholt et al., 2014).

### Methods

#### Participants

For this study, an existing datasheet of outpatients whom received intensive 4-day or 8-day outpatient treatment for OCD at ‘Pro Persona Overwaal, Center of Expertise for anxiety, OCD, and PTSD’ in the Netherlands was used. For inclusion, patients had to be at least 18 years of age at treatment onset, and meet the DSM-5 criteria for OCD (APA, 2013). A total of  $n = 124$  patients were included in the analysis. Of the patients, 43 (34.7%) identified as males and 81 (65.3%) identified as females. The mean age (years) of the patients at the start of treatment was 33.05 ( $SD = 11.67$ ). Patients gave permission for their data to be used anonymously for scientific research, and were not reimbursed for their participation as the administration of filled-in questionnaires was part of standard treatment procedures and outcome monitoring.

The initial datasheet contained 148 data-entries of OCD-diagnosed individuals. 10 entries were removed on the basis that they only contained a patient ID, without any other data present. 11 data-entries were removed on the basis that they did not include post-measurement data, or that data on the criterion variable (Y-BOCS scores on pre- or post-treatment-measurement) was missing. 3 entries were removed on the basis that no demographic data was present due to closed dossiers. Hence, 124 patients were included in the final analysis.

#### Materials

The demographic data used in the analysis was self-reported by the patients at treatment onset and used to assess our sample. Several clinical questionnaires and clinical-assessed interview were also administered to obtain the data included in the datasheet and used for the description of the sample. For both the dependent variable, as well as the first two predictors

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

in the second analyses, various questionnaires were used as material. The third predictor variable (onset of OCD) was self-reported by the patient.

*Dependent variable:* OCD-symptoms were assessed with the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman et al., 1989), translated into Dutch (Arrindell & Vlaming, 1999). The Y-BOCS is a clinician-assessed interview which consists of 10 items to measure the severity of OCD-symptomatology in research and clinical practice. Each individual item can be rated from 0 (no symptoms) to 4 (extreme symptoms). The total score of the 10 items ranges between 0 (no symptoms of OCD) and 40 (severe symptoms of OCD). The internal consistency reliability is .97 for the subscale ‘obsessions’ and .95 for the subscale ‘compulsions’, and interrater reliability was high for the subscales (Kendall’s  $W = .897$  for the subscale ‘obsessions’ and Kendall’s  $W = 1$  for the subscale ‘Compulsions’; Arrindell, de Vlaming, Eisenhardt, van Berkum, & Kwee, 2002). Concurrent validity ranged between  $r = .4$  and  $r = .6$  with scores on the items from the self-reported Padua Inventory, (PI; Sanavio, 1988; van Oppen, Hoekstra, & Emmelkamp, 1995; Arrindell et al., 2002). The scores on the Y-BOCS were transformed into a binary remission variable and a treatment response variable for usage as a dependent variable (see data analysis).

*First predictor (Initial OCD-severity):* Initial OCD-severity was assessed with the Dimensional Obsessive-Compulsive Scale (DOCS; Abramowitz et al., 2010). The DOCS is a self-report questionnaire of 20-items concerning four theme-based dimensions (contamination, responsibility for harm and mistakes, incompleteness/symmetry, and taboo thoughts; Abramowitz et al., 2010). The 20 items of the Dutch version are all scored on severity levels 1 – 5. The total of the added scores of the 20 items ranges between 20 (no OCD-symptoms) and 100 (severe OCD-symptoms; Abramowitz et al., 2010). In this study the Dutch version of the DOCS was used. The Dutch version has a high internal consistency for the total instrument (Cronbach’s  $\alpha = .93$ ) and the four subscales (Cronbach’s  $\alpha = .94 - .96$ ; Van der Veld, Duppen, Hendriks, Abramowitz, & Kampman, In press). In the study by Van der Veld et al., the convergent validity with the Y-BOCS (Goodman et al., 1988) as OCD-specific instrument was  $r = .73$ , and the convergent validity with measures of general validity was  $r = .50$  for the EuroQol Visual Analogue Scale (EQ-VAS; The EuroQol Group, 1990),  $r = .65$  for the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988), and  $r = .60$  for the Inventory of Depressive Symptomatology self-report (IDS-SR; Rush et al., 1986; Rush, Gullion, Basco, Jarrett, & Trivedi, 1996).

*Second predictor (depressive symptomatology):* The Dutch translation of the IDS-SR was used to assess depressive symptomatology (Rush et al., 1986; 1996; Akkerhuis, 1997).

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

The self-report version includes 30 items with four severity levels (0 – 3) on depressive symptomatology. The total of the added scores of the 30 symptoms ranges between 0 points (no depressive symptomatology) and 90 points (severe depressive symptomatology; Rush et al., 1986, 1996). Although no validation and reliability studies are available for the Dutch version, the English version has a reportedly high internal consistency (Cronbach's  $a = .94$  for all subjects and Cronbach's  $a = .77$  for symptomatic patients; Rush et al., 1996). Concurrent validity with the Hamilton Rating Scale-Depression (HRS-D; Hamilton, 1960; 1967) was  $r = .68$ , and  $r = .48$  with the Beck Depressive Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Rush et al., 1996).

*Third (age of onset):* The age category of onset of complaints, as reported by the patient, was used as a predictor. Based on the findings of Anholt et al. (2014) the three age categories in the datasheet (younger than 12, between 12 and 19, older than 19) were reduced to two specific qualitative categories in which the 'younger than 12' and 'between 12 and 19' are merged to accommodate to the findings of two differing categories of OCD regarding its age of onset ('early-onset' and 'late-onset'; Anholt et al., 2014).

### **Procedure**

Before the treatment of the patient commenced, the patient was asked to fill out the questionnaires for the purpose of routine outcome management at the outpatient facility (taking approximately 60-90 minutes). Written ethical permission was obtained for anonymous usage of the filled-in data for academic purposes. Based on clinical judgement, patients meeting the criteria for intensive outpatient treatment for OCD as assessed by a team of clinicians were assigned to either a 4-day or an 8-day cET programme. A description of the treatment is provided in Appendix 1. In total, measurement occurred at 5 points: Pre-treatment, post-treatment, at 3-month follow-up (FU1), at 6-month follow-up (FU2) and at 12-month follow-up). No reimbursement was provided, as routine outcome measurement itself was a part of regular treatment procedures.

### **Data analysis**

The datasheet contained data from September 10, 2015 up to October 31, 2019, and was analysed using IBM SPSS 25<sup>th</sup> edition. The data of 124 patients was used in the analyses. The Y-BOCS values (dependent variable) were coded into two binary variables (1 = remission, 0 = no remission) that were used in the second analyses as dependent variables. Remission was scored '1' in the second analyses if the Y-BOCS score at the moments of measurement were equal or lower than 12. Treatment response (1 = treatment response, 0 =

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

no treatment response) was coded '1' if a response of 35% symptom reduction of Y-BOCS scores is achieved, compared to pre-treatment Y-BOCS levels at the moments of measurement. The aforementioned scores were based on consensus reached by international OCD-researchers who authored peer-reviewed papers between 2007 and 2013 (Mataix-Cols, 2016). An example of the calculation for symptom reduction is '(post-treatment Y-BOCS – pre-treatment Y-BOCS) / - (pre-treatment Y-BOCS)'. The minus after the divider was added for convenience to display positive improvement percentages (see appendix 2). Important for the second analyses, four patients who participated in the 8-day programme did not complete all days of treatment (two patients participated six days and two patients participated seven days). In order to retain as much data as possible, the second analyses conservatively included these cases under the 8-day programme they were initially assigned to.

*First analysis (improvement following OCD-treatment):* For the first analysis, a repeated measures ANOVA was used to assess if participants significantly improved at post treatment, FU1 and FU2, following OCD-treatment. Prior to the analyses, it was determined that the multivariate method would be used if the Huynh-Feldt epsilon would be < .85 (Algina & Keselman, 1997) as it would have greater power (See also Ellis, 2016). If the Huynh-Feldt epsilon would exceed .85, the univariate method would have been used with Huynh-Feldt correction, as it would have yielded the greatest power under those circumstances (Tabachnick & Fidell, 2001; see also Ellis, 2016). In the first analysis, Y-BOCS scores were used as the dependent variable (quantitative), with time (pre-treatment, post-treatment, FU1, and FU2) as the within-subject factor. This analysis was used as a check preceding the second analyses (as assessing predictors without a significant treatment effect would not yield potentially relevant results).

*Second (group of) analyses (outcome predictors of OCD-treatment):* For the second analyses addressing the predictors, two binary logistic regression analyses were conducted for each measurement moment after treatment (post-treatment, FU1, and FU2). For each moment of measurement, the binary remission variable and treatment response variable were calculated based on the Y-BOCS remission/treatment response criteria as formulated by Mataix-Cols et al. (2016). These were the dependent variables in each binary logistic regression analysis (see the earlier description of specific coding procedure according to the criteria by Mataix-Cols et al., 2016). The predictor variables were the Y-BOCS pre-treatment scores, IDS-SR pre-treatment scores (quantitative, 0 - 90), and onset of complaints (predictor variable; qualitative – younger than 19, older than 19). Because patients were not randomly assigned to a treatment programme, we controlled for treatment programme (4-day or 8-day

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

cET). Therefore, treatment programme was used as a covariate. Treatment programme was used as covariate in the first level of the binary logistic regression analyses, while the other predictors were included in the second level of binary logistic regression analyses. The demographics and predictor scores of the treatment groups were compared at pre-treatment levels.

### Results

#### First analysis

*Improvement after treatment:* A repeated measures ANOVA (multivariate method, Huynh-Epsilon correction for sphericity applied  $< .85$  [= .736]) with Y-BOCS scores as dependent variable, and time with four levels (pre-treatment, post-treatment, as well as FU1, and FU2 as within-subject factor, reveals a significant main effect of time (*Wilks' F*(3,37) = 34.182,  $n = 40$ ,  $p < .0001$ ,  $\eta^2 = .735$ , power = 1.0). Univariate follow-up tests reveal a significant effect of time for post-treatment, 3-month follow-up, and at 6-month follow-up Y-BOCS scores in comparison to pre-treatment Y-BOCS scores. The mean scores and standard deviations of the predictors are reported for each measurement moment (table 1). The results of the univariate follow-up tests are displayed in table 2, and available in written form in appendix 3.

Table 1

*Means, standard deviations, and distribution<sup>a</sup>*

Measurement moment	Means, standard deviation, and distribution				
	Y-BOCS <i>M (SD)</i>	DOCS <i>M (SD)</i>	IDS-SR <i>M (SD)</i>	Onset (early/late) <sup>b</sup>	Treatment programme (4-day / 8-day)
Pre-treatment	24.98 (5.66)	29.69 (15.82)	28.58 (14.08)	27 / 12	14 / 26
Post-treatment	13.85 (6.13)	15.08 (10.83)	20.38 (14.56)	27 / 12	14 / 26
FU1	13.35 (7.31)			27 / 12	14 / 26
FU2	14.73 (7.14)			27 / 12	14 / 26

a) Means and standard deviations based on cases included in repeated measures ANOVA

b) Information regarding OCD-onset was not available for one of the 40 participants in the repeated measures ANOVA

Table 2

*Difference in Y-BOCS scores in comparison to pre-treatment Y-BOCS levels*

Measurement moment	Y-BOCS statistics			
	<i>n</i>	<i>F</i>	<i>p</i>	Partial $\eta^2$
Pre-treatment				
Post-treatment	40	102.244	.0001****	.724
FU1	40	72.263	.0001****	.649
FU2	40	52.039	.0001****	.572

\*\*\*\*  $p < .0001$

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

### **Second analyses**

*Predictor analysis remission:* For each of the three measurement moments after treatment (post-treatment, FU1, and FU2) a binary logistic regression analysis was conducted with remission (yes/no, at moment of measurement) as dependent variable and onset, IDS-SR-scores, and DOCS-scores as predictor variables. Early-onset is abbreviated to EO, and late-onset is abbreviated to LO (see appendix 4 for a comparison of the two groups). Treatment programme (abbreviated to ‘treatment’; 4-day or 8-day cET) was incorporated as a covariate (see table 4 for a comparison the two treatment groups). There is a medium but significant effect size (Nagelkerke  $R^2 = .158$ ) of depressive symptomatology on treatment response at post-treatment, and a medium but significant effect size of depressive symptomatology at FU1 (Nagelkerke  $R^2 = .167$ ). More days of treatment is associated with lower remission at post-treatment and at FU2 measurement. The respective effect sizes are both medium (Nagelkerke  $R^2 = .158$  and Nagelkerke  $R^2 = .167$ ). The results are displayed in table 3 (see below).

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

Table 3

*Predictors of remission as assessed by the Y-BOCS*

Measurement moment <sup>a</sup>	Omnibus				Predictors and covariate of remission (binary logistic regression)							
	Chi-squared ( $\chi^2$ (DF))	<i>n</i>	<i>p</i>	Nagelkerke $R^2$	DOCS ( <i>p</i> )	DOCS ( <i>b</i> )	IDS-SR ( <i>p</i> )	IDS-SR ( <i>b</i> )	EO/LO ( <i>p</i> )	EO/LO ( <i>b</i> )	Treatment ( <i>p</i> )	Treatment ( <i>b</i> )
Post-treatment (Level 1)	$\chi^2$ (1) = 4.200	110	.040*	.051							.041*	-.877
Post-treatment (Level 2)	$\chi^2$ (4) = 13.626	110	.009*	.158	.629		.029*	-.048	.655		.078	
FU1 (Level 1)	$\chi^2$ (1) = 0.007	55	.931	.000							.931	
FU1 (Level 2)	$\chi^2$ (4) = 7.378	55	.117	.167	.957		.029*	-.067	.855		.678	
FU2 (Level 1)	$\chi^2$ (1) = 4.939	53	.026*	.123							.029*	-1.386
FU2 (Level 2)	$\chi^2$ (4) = 5.967	53	.202	.147	.915		.419		.780		.033*	-1.420

a) Level refers to the omnibus level of the analyses. For level 1 analyses, only the covariate treatment programme was included while the predictors were also included in the level 2 analysis

\*  $p < .05$

Table 4

*Means, standard deviations, and distributions per treatment program at treatment onset*

Treatment	Means, standard deviation, and distribution					
	Y-BOCS	DOCS	IDS-SR	Age	EO/LO <sup>a</sup>	Gender
	<i>M</i> ( <i>SD</i> ) *	<i>M</i> ( <i>SD</i> ) *	<i>M</i> ( <i>SD</i> )	<i>M</i> ( <i>SD</i> )		(M/F)
4-day	24.86 (6.08)	26.83 (16.34)	29.42 (13.48)	33.72 (11.58)	23/9	12/24
8-day	27.26 (5.63)	33.53 (13.57)	29.55 (12.27)	32.77 (11.76)	48/35	31/57

a) For the 4-day group, 4 values are missing for onset. For the 8-day group, 5 values are missing for onset

\*) Marker for significant difference between 4-day and 8-day treatment group (see textual description below)

*Treatment group comparison (see table 4):* A t-test between the mean starting Y-BOCS severity of the 4-day group and the 8-day group reveals a significant difference between the groups on mean starting Y-BOCS severity ( $t(122) = 2.105, p = .037$ ). A t-test between the mean starting DOCS severity of the 4-day group and the 8-day group reveals a significant difference between the groups on mean starting DOCS severity ( $t(122) = 2.349, p = .020$ ). A t-test between the mean starting IDS-SR severity of the 4-day group and the 8-day group reveals no significant difference between the groups on mean starting IDS-SR severity ( $t(122) = 0.052, p = .096$ ). A t-test between the mean age at treatment onset of the 4-day group and the 8-day group reveals no significant difference between the groups on mean age at treatment onset ( $t(122) = 0.410,$

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

$p = .682$ ). A Chi-square test for independence between the early-onset/late-onset distribution in the 4-day group and the 8-day group reveals no significant difference between the groups on onset ( $\chi^2(1, n = 115) = 1.928, p = .165$ ). A Chi-square test for independence between the gender distribution in the 4-day group and the 8-day group reveals no significant difference between the groups on gender distribution ( $\chi^2(1, n = 124) = 0.041, p = .0841$ ).

*Predictor analysis treatment response:* For each of the three measurement moments (post-treatment, FU1, and FU2) a binary logistic regression analysis was conducted with treatment response (yes/no, at moment of measurement) as dependent variable and onset (EO/LO), IDS-SR-scores, and DOCS-scores as predictor variables. Treatment programme (abbreviated to ‘treatment’; 4-day or 8-day cET) was incorporated as a covariate. Early-onset predicted a better response to treatment at post-treatment measurement. The size of effect is small (Nagelkerke’s  $R^2 = .099$ ). The results are displayed in table 5 (see below).

Table 5

*Predictors of treatment response as assessed by the Y-BOCS*

Measurement moment <sup>a</sup>	Omnibus				Predictors and covariate of treatment response (binary logistic regression)							
	Chi-squared ( $\chi^2$ (DF))	<i>n</i>	<i>p</i>	Nagelkerke $R^2$	DOCS ( <i>p</i> )	DOCS ( <i>b</i> )	IDS-SR ( <i>p</i> )	IDS-SR ( <i>b</i> )	EO/LO ( <i>p</i> )	EO/LO ( <i>b</i> )	Treatment ( <i>p</i> )	Treatment ( <i>b</i> )
Post-treatment (Level 1)	$\chi^2(1) = 0.009$	110	.925	.000							.925	
Post-treatment (Level 2)	$\chi^2(4) = 8.332$	110	.080	.099	.890		.486		.012*	-1.103	.804	
FU1 (Level 1)	$\chi^2(1) = 0.186$	55	.666	.005							.669	
FU1 (Level 2)	$\chi^2(4) = 7.378$	55	.117	.167	.747		.169		.622		.515	
FU2 (Level 1)	$\chi^2(1) = 1.670$	53	.196	.041							.202	
FU2 (Level 2)	$\chi^2(4) = 2.049$	53	.727	.051	.603		.775		.821		.192	

a) Level refers to the omnibus level of the analyses. For level 1 analyses, only the covariate treatment programme was included while the predictors were also included in the level 2 analysis

\*  $p < .05$

### Discussion

We investigated potential predictors in outpatients with OCD, naturalistically assigned to a 4-day or 8-day cET programme. As we hypothesized, cET is an effective form of treatment as lower Y-BOCS scores on post-treatment, FU1, and FU2 were identified with the first analysis. Controlled for in the subsequent predictor analyses, the scores of the covariate 4-day cET treatment group were associated with more remission at post-treatment and FU2 measurement than the scores of the 8-day cET treatment group. For FU1, there were no significant differences between the treatment groups on remission, nor on treatment response. For the first predictors, higher pre-treatment DOCS scores indicating more severe OCD did not predict remission or treatment response at any point of measurement. For the second predictor, higher pre-treatment IDS-SR scores indicating higher depressive symptomatology significantly predicted lower remission at post-treatment measurement and FU1 measurement (with medium effect sizes; although the omnibus test was not significant for the model itself for FU1 measurement). However, higher IDS-SR scores did not predict remission at FU2, nor did IDS-SR scores predict treatment response. Contrary to our hypothesis on the third predictor, late OCD-onset predicted worse treatment response as measured post-treatment (small effect size). OCD-onset did not predict treatment response at follow-up measurements, nor did OCD-onset predict remission.

Our study confirms the high effectiveness of cET, as was earlier reported and replicated by Havnen et al. (2014, 2017). This study is, however, the first to show that pre-treatment depressive symptomatology hinders achieving remission defined as scores equal or lower than 12 on the Y-BOCS post cET and on 3-month follow-up measurement. Formulated otherwise, individuals with less depressive symptomatology pre-treatment have a better chance of achieving remission after cET. Our findings are partly in line with earlier findings (Marcks, Weisberg, Dyck, & Keller, 2011) indicating that a Major Depressive Disorder (MDD) limits remission after treatment (although higher scores on depressive symptoms are not predictive of remission at 6-month follow-up in our study). However, we found that depressive symptomatology did not predict whether someone will improve by more than 35% on OCD-severity as measured on the Y-BOCS.

There are various explanations for how comorbid depressive symptomatology may hinder remission. For example, a patient's compliance with instructions provided by the therapist may be more complex with co-morbid depressive symptomatology, hindering the effect of treatment (Abramowitz & Foa, 2000). Early compliance in the first week of treatment, which may arguably be hindered though comorbid depressive symptomatology,

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

appears especially predictive of treatment outcomes for OCD-treatment (De Araujo, Ito, & Marks, 1996). Furthermore, inhibited learning due to depressive symptomatology has been suggested as a factor that complicates learning throughout ERP-treatment (Abramowitz & Foa, 2000).

A result contrary to our expectations is that individuals suffering from late-onset OCD respond less to treatment than patients with early-onset OCD at post-treatment. This result did not hold on follow-up measurements. Despite being predictive of more than 35% OCD-symptom reduction at post-treatment measurement, onset did not nearly approach significance as a predictor of remission. In order to get a clearer picture of this result, we reviewed the characteristics of the early-onset group and the late-onset group (appendix 4). The late-onset group consisted of slightly but not significantly more males (relatively speaking). Furthermore, the mean age of the late-onset group (37.93) was significantly higher at treatment onset than the mean age of the early-onset group (29.75). These demographical differences between early- and late-onset samples are in line with Anholt et al.'s (2014) findings on the difference between patients with early-onset and late-onset OCD.

Out of 12 earlier studies on OCD-onset identified by Taylor (2011), none found that an earlier age of onset was correlated with a better OCD-treatment response. Although one study by Kempe et al. (2007) showed higher remission rates for the early-onset group, this would make our study an exception relative to the vast majority of other OCD-treatment studies. Worthy of noting, though, none of these other studies specifically included cET as a form of treatment. Potentially, the intensive form of ERP-treatment disconfirms a patient's belief already held since childhood more strongly than less intensive forms of treatment for OCD do. It may arguably become clear to the patients that through intensive treatment, even symptoms held since childhood do not have to remain for the rest of one's life, creating hope and anticipation. Another potential explanation is that unexpected events (such as successful interventions) generate more neural activity which are therefore more easily recalled (Ranganath & Rainer, 2003; Foster & Keane, 2019). If symptoms were already present since childhood, successful treatment results may have been less expected. Nevertheless, these explanations are strongly preliminary and therefore warrant caution. As this is the first study to evaluate onset as a predictor in cET, replication studies are called for.

For the covariate treatment programme, 4-day treatment is associated with more remission than 8-day treatment at post-treatment measurement and 6-month follow-up measurement. A potential explanation for this outcome is the naturalistic nature of our study. Patients were not randomly assigned to a treatment-condition. Instead, clinicians judged the

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

severity of the complaints and assigned patients to either 4-day treatment or 8-day treatment on that basis. Despite assignment to the 8-day treatment group would increase the amount of ERP-treatment received, the chances of these patients of achieving remission were possibly lower due to higher initial clinical severity as assessed by the clinicians' judgement. Measurable differences between the groups indicate that both Y-BOCS scores and DOCS scores were significantly higher at pre-treatment measurement for the 8-day group.

Based on various earlier findings, we expected that higher OCD-severity at pre-treatment would be more predictive of lower treatment response across measurement moments (Kempe et al., 2007, Knopp et al., 2013). Although these predictions were not confirmed, this is a promising result for patients suffering from OCD. In our study, achieving treatment response and remission following cET were not hindered by more severe OCD-symptomatology at pre-treatment measurement. It is possible that cET, through its treatment intensity, counters potential treatment resilience more effectively than less intensive forms of treatment. For example, Hansen, Kvale, Hagen, Havnen, & Öst (2018) have found that cET yields relatively similar remission rates for patients rated with moderate OCD (82%) and severe/extreme OCD (69%) after treatment. The small percentual difference disappeared at follow-up measurement (68% and 69% remission respectively; Hansen et al., 2018).

Our study has various limitations. First of all, patients were not randomly assigned to treatment conditions. As previously mentioned, our study depended on clinician's judgement to assign a patient to either 4-day treatment or 8-day treatment, thus hindering adequate comparison of the effect of length of treatment itself and its inclusion as a predictor on its own. Second, only 32% of the 124 patients completed the post-treatment assessment and the two follow-up assessments, which can be attributed to ongoing data collection. While the Chi-square analyses for independence are not dependent on the availability of data for a preceding moment of measurement, the lack of data for one of the points of measurement makes it harder to analyse the full effects of our treatment on one patient over time. Third, the statistical power of our analyses was not optimal. Opinions diverge on the optimal sample size calculations for logistic regression analyses. A recently suggested rule of thumb would recommend approximately 300 participants for a logistic regression analysis with 4 independent variables according to a strict Events Per Variable (EPV) criterion, and 180 participants with a less strict EPV criterion (Bujang, Sa'at, & Bakar, 2018; Austin & Steyerberg, 2017). Fourth, our data was recoded according to stringent remission- and treatment response criteria (Mataix-Cols et al., 2016). While adhering to strict agreed-upon

## OUTCOME PREDICTORS IN TREATMENT OF PATIENTS WITH OCD

threshold criteria is arguably good clinical practice and not a limitation in itself, the recoding procedure for the second analysis inherently resulted in a loss of data (Altman & Royston, 2006). The Y-BOCS scores were recoded into a binary variable, and age of onset was reduced from three categories in our sample to two categories in accordance with findings distinguishing early- and late-onset OCD into two specific age categories (Anholt et al., 2014). One example of potential data loss is that in the second analyses a symptom reduction of 37% counted just as heavy as a symptom reduction of 90% (since both are binary-coded into '1' as they both exceed 35% symptom reduction). Fifth, a limitation that was also pointed out by Anholt et al. (2014), is that onset as a predictor is vulnerable to recall bias. Patients may not be able to accurately recall when symptoms began; especially when patients were younger when symptoms began (Masia et al., 2003).

Suggestions for future research would include a study with random assignment to a 4-day and 8-day cET treatment, as the naturalistic approach incorporating clinician's judgement would hinder the effective analysis of cET-length on OCD-symptom reduction. Furthermore, replication studies of the current predictors are called for, as well as studies on other potential predictors of cET-effectiveness. Due to practical restrictions, predictors such as treatment history, anxiety, and interpersonal problems were not included in this study. Replicating current predictors and investigating new predictors would arguably increase our insight in OCD, improve patient-based treatment assignment, and aid in differentiating intensive cET treatment from less intensive treatment forms for OCD.

Arguably, our study raises several new questions. While cET is effective at reducing OCD-symptoms at post-treatment and all follow-up measurements contrasted to pre-treatment levels, the results of the specific predictors are less consistent over time. More depressive symptomatology appears predictive of less remission at post-treatment and 3-month follow-up, and an earlier age of onset appears predictive of more treatment response post-treatment. However, these associations did not hold on further follow-up measurements. Nevertheless, our study was the first study to assess treatment predictors for cET in patients with OCD. While many questions remain unanswered, our study provides a first step in assessing potential predictors of cET. Its limitations may become other studies' strength in further assessing cET as a highly effective form of treatment for OCD.

Appendices

**Appendix 1: remission per treatment group**

Every treatment day began with a treatment interview of 1.5 hours, followed by two to three treatment blocks of 1.5 hours consisting of exposure-therapy (4.5 – 6 total hours of treatment per day). Furthermore, a module focussing on positive psychology was a standard part of the treatment programme and consisted of four session of 1.5 hours in total. The 4-day or 8-day programme was followed by four 1.5-hour booster sessions once-a-week after treatment. For follow-up purposes, the Y-BOCS scores were also obtained 3- and 6-months after treatment was completed (taking approximately 30-60 minutes; either at the outpatient facility or through telephone/e-mail contact).

**Appendix 2: Table of predictor analyses**

Appendix 2 table

*Table of analyses with dependent- and predictor variables.*

Analyses	Variables	
	Dependent	Predictors*
Post-treatment analysis	Remission = 1 IF Post-treatment Y-BOCS = $\leq 12$ Treatment response = 1 IF (post-treatment Y-BOCS – pre-treatment Y-BOCS)/ - (pre-treatment Y-BOCS) = $\geq 0.35$	<ul style="list-style-type: none"> <li>• OCD severity at onset (DOCS)</li> <li>• IDS-SR (pre-treatment)</li> <li>• Age category at onset of complaints</li> </ul>
3-month follow-up analysis	Remission = 1 IF 3-month FU Y-BOCS = $\leq 12$ Treatment response = 1 IF (3-month FU Y-BOCS – pre-treatment Y-BOCS)/ - (pre-treatment Y-BOCS) = $\geq 0.35$	<ul style="list-style-type: none"> <li>• OCD severity at onset (DOCS)</li> <li>• IDS-SR (pre-treatment)</li> <li>• Age category at onset of complaints</li> </ul>
6-month follow-up analysis	Remission = 1 IF 6-month FU Y-BOCS = $\leq 12$ Treatment response = 1 IF (6-month FU Y-BOCS – pre-treatment Y-BOCS)/ - (pre-treatment Y-BOCS) = $\geq 0.35$	<ul style="list-style-type: none"> <li>• OCD severity at onset (DOCS)</li> <li>• IDS-SR (pre-treatment)</li> <li>• Age category at onset of complaints</li> </ul>

\* Treatment programme (4-day or 8-day) is incorporated as covariate in every analyses

**Appendix 3: Written description Y-BOCS scores improvement**

Y-BOCS scores between pre-treatment measurement and post-treatment measurement differed significantly ( $F(1,39) = 102.244, p < .0001, \eta^2_{\text{Partial}} = .724$ ) with means 24.98 ( $SD = 5.66$ ) and 13.85 ( $SD = 6.13$ ) respectively. Y-BOCS scores between pre-treatment measurement and 3-month follow-up measurement differed significantly ( $F(1,39) = 72.263, p < .0001, \eta^2_{\text{Partial}} = .649$ ) with means 24.98 ( $SD = 5.66$ ) and 13.35 ( $SD = 7.31$ ) respectively. Y-BOCS scores between pre-treatment measurement and 6-month follow-up measurement differed significantly ( $F(1,39) = 52.039, p < .0001, \eta^2_{\text{Partial}} = .572$ ) with means 24.98 ( $SD = 5.66$ ) and 14.73 ( $SD = 7.14$ ) respectively.

**Appendix 4: Analyses of the early-onset vs late-onset group**

A t-test was conducted between the mean starting Y-BOCS severity of the early-onset group ( $m = 26.70$ ,  $SD = 5.33$ ,  $n = 71$ ) and the late-onset group ( $m = 25.73$ ,  $SD = 6.58$ ,  $n = 44$ ). There was no significant difference between the groups on mean starting Y-BOCS severity ( $t(113) = 0.867$ ,  $p = .388$ ).

A t-test was conducted between the mean starting age at treatment of the early-onset group ( $m = 29.75$ ,  $SD = 10.86$ ,  $n = 71$ ) and the late-onset group ( $m = 37.93$ ,  $SD = 11.271$ ,  $n = 44$ ). There was a significant difference between the groups on mean starting age ( $t(113) = 3.8692$ ,  $p = .0002$ ).

The early-onset sample ( $n = 71$ ) consisted of 22 males (31.0%) and 49 females (69.0%). The late-onset sample ( $n = 44$ ) consisted of 19 males (43.2%) and 25 females (56.8%). Relatively speaking, the late-onset sample consisted of more males than the early-onset sample.

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