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# A randomised controlled trial comparing EMDR and CBT for Obsessive-Compulsive Disorder

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## **Abstract**

*Background:* This study aimed to evaluate eye-movement desensitisation and reprocessing (EMDR) as a treatment for obsessive-compulsive disorder (OCD), by comparison to cognitive behavioural therapy (CBT) based on exposure and response prevention.

*Method:* This was a pragmatic, feasibility randomised controlled trial in which 55 participants with OCD were randomised to EMDR (n = 29) or CBT (n = 26). The Yale-Brown obsessive compulsive scale (YBOCS) was completed at baseline, after treatment and at 6 months follow-up. Treatment completion and response rates were compared using chi square tests. Effect size was examined using Cohen's *d* and multilevel modelling.

*Results:* Overall, 61.8% completed treatment and 30.2% attained reliable and clinically significant improvement in OCD symptoms, with no significant differences between groups ( $p > .05$ ). There were no significant differences between groups in YBOCS severity post-treatment ( $d = -0.24$ ,  $p = .38$ ) or at 6 months follow-up ( $d = -0.03$ ,  $p = .90$ ).

*Conclusions:* EMDR and CBT had comparable completion rates and clinical outcomes.

## **Key Practitioner Message**

- Fifty five patients with OCD were randomised to receive either EMDR or CBT
- Both treatments had comparable completion rates and clinical outcomes
- No significant differences were found after treatment or at 6 months follow-up

**Key words:** EMDR; CBT; obsessive-compulsive disorder; randomized controlled trial

## **INTRODUCTION**

Obsessive-Compulsive Disorder (OCD) is a condition characterised by intrusive thoughts (obsessions) that are accompanied by intense urges (compulsions) to neutralise the associated distress by performing mental or physical rituals (Abramowitz, Taylor, & McKay, 2009). Cognitive behavioural therapy (CBT) based on exposure and response prevention (ERP) is recommended by clinical guidelines as a first line psychological treatment for this condition (National Institute for Health and Clinical Excellence [NICE], 2005). Meta-analytic reviews of clinical trials indicate that CBT is significantly more effective than waitlist or placebo control conditions and equally as effective as pharmacological treatment (i.e., Abramowitz, 1997, 1998; van Balkom et al., 1994; Olatunji, Davis, Powers, & Smits, 2013). In spite of the evidence favouring CBT, there are also a number of recognised drawbacks. Some studies suggest that patients find it difficult to tolerate exposure exercises and tend to drop out of treatment (Abramowitz, Taylor, & McKay, 2005). For example, Foa et al. (2005) reported that 28% of patients dropped out shortly after commencing exposure and response prevention. Even after completing CBT, more than 30% of patients are reported to access ongoing treatment (Rowa et al., 2007). Furthermore, OCD is considered to be one of the most treatment resistant non-psychotic mental health problems (Ponniah, Magiati, & Hollon, 2013), since relatively few patients (approximately 25%) end treatment completely symptom-free (Fisher & Wells, 2005). These drawbacks raise a question about how to meet the needs of those for whom CBT is less effective.

Recent studies have considered whether eye-movement desensitisation and reprocessing (EMDR) could be a helpful alternative treatment for OCD. EMDR is an empirically supported treatment for post-traumatic stress disorder (Bisson et al., 2007; Davidson, & Parker, 2001; Van Etten, & Taylor, 1998). A number of uncontrolled case series have reported the successful application of

EMDR to alleviate symptoms of OCD (Bekkers, 1999; Bohm & Voderholzer, 2010; Keenan, et al., 2014; Marr, 2012). Furthermore, Nazari et al. (2011) conducted a controlled trial in which 90 OCD patients were randomised to either EMDR or pharmacotherapy (citalopram). This trial reported comparable baseline severity of OCD symptoms using the Yale-Brown Obsessive Compulsive Scale (YBOCS), but significantly lower symptoms in the EMDR group (mean YBOCS = 13.6) by comparison to the pharmacotherapy group (mean YBOCS = 19.06) after 12 weeks of treatment.

Furthermore, there are theoretical reasons why EMDR could be considered as a plausible treatment option for OCD. There is evidence that in some cases OCD may originate in the wake of stressful life events (de Silva & Marks, 1999), and that stressful life events increase the risk of OCD relapse (Steketee, 1993). For example, there is a high incidence of OCD in combat exposed soldiers by comparison to controls (Jordan et al., 1991), and the risk of developing OCD is ten times greater in people with post-traumatic stress disorder by comparison to people without trauma-related problems (Helzer et al., 1987). The *adaptive information processing* (AIP) model of EMDR proposes that psychological symptoms often result from unprocessed traumatic material (Shapiro & Forrest, 2004), or stressful life events. Based on the notion that EMDR works to resolve disturbing memories of traumatic events, it could be that other types of anxiety disorders that develop following a distressing event may also be responsive to EMDR. OCD, whilst different in presentation to PTSD, shares some similarities such as repeated intrusive thoughts and images that evoke intense anxiety and avoidance. Several case studies have also indicated qualitative/metaphorical associations between the content of obsessive intrusions (e.g., 'being or feeling dirty') and the context of traumatic events (e.g., sexual assault) implicated in the onset of obsessional thoughts (de Silva & Marks, 1999). It seems plausible that processing the cognitive, somatic and affective aspects of traumatic

events could afford some alleviation of symptoms that may have arisen from such events.

Overall, these emerging studies and hypotheses suggest that EMDR could be a helpful treatment option for OCD, although there are also several caveats. Small case series are not representative of OCD cases in general healthcare settings and could be prone to selection biases. These case series also lack rigorous designs (i.e., single case experimental design with multiple baselines) and appropriate statistics to account for regression to the mean. The only experimental study to date by Nazari et al. (2011) offers more convincing support for the application of EMDR; however, the lack of post-treatment follow-up raises questions about the sustainability of treatment effects. Furthermore, it is not known if EMDR may be as effective or acceptable as commonly available CBT interventions.

With this backdrop of emerging studies, we conducted a controlled trial that enabled us to compare the application of EMDR with CBT for OCD.

## **METHOD**

### ***Design***

This was a pragmatic randomised controlled trial conducted in a primary care, outpatient, public healthcare system (UK National Health Service). The primary objective was to assess the feasibility (based on attendance and completion rates) of delivering EMDR for OCD in routine care. A secondary objective was to undertake a preliminary comparison of clinical effects between EMDR and CBT, which could inform future trials assessing efficacy and mechanisms of action.

The study was approved by an NHS research ethics committee (Ref: 13/YH/0338) and registered in an international database prior to recruitment (ISRCTN16396325).

## **Setting**

Trial participants were recruited and treated in a primary care mental health service in Leeds, a large and socioeconomically diverse city in the north of England. The service offered access to evidence-based psychological interventions recommended by national guidelines (NICE, 2011) and delivered by qualified practitioners working under clinical supervision. Patients with OCD were referred to the service by general practitioners, or self-referred, and were routinely offered 16 to 20 sessions of cognitive behavioural therapy (NICE, 2005).

## **Interventions**

### *Eye-Movement Desensitisation and Reprocessing (EMDR)*

EMDR is a therapy where a structured approach is used to address the past, present and future aspects of traumatic events. Based on Shapiro's (2001) AIP model, EMDR conceptualises psychiatric disorders as a manifestation of unresolved traumatic events. EMDR therapy uses a eight-phase procedure that begins with history taking and case formulation (phase 1), preparation (phase 2) to ensure the client has the resources to manage the processing of the distressing information to an adaptive resolution. In phase 3 the visual, cognitive (negative and desired positive cognition), affective, and sensory components of the targeted memory are identified, and ratings for levels of distress and level of belief in the positive cognition are taken. In phase 4, the client recalls the targeted memory, while simultaneously engaging in sets of eye movements (or alternating bilateral audio and/or tactile stimulation). This continues until the distress level is rated at 0. A memory is considered to be processed when it no longer elicits any affective or somatic distress. In phase 5 the transition to a convincingly valid positive cognition is strengthened using further bilateral stimulation (for example, going from a negative

cognition of 'I am powerless' to 'I am in control'). The installation and strengthening of the positive cognition is a crucial component of EMDR by focusing on the client's positive self-assessment which is pivotal for positive therapeutic effect (Shapiro 2001). Phase 6 is considered completed when a client can bring the memory and positive cognition to mind without any body tension. Phase 7 involves a careful closure of the session including use of resources, and phase 8 is the re-evaluation which takes place at the start of every subsequent session.

Marr (2012) hypothesised that EMDR could provide a treatment option for OCD whereby processing the fears and ritualised behaviours of OCD would decrease symptoms in the present before turning to work on the underlying events linked to the onset of symptoms. This study applied an EMDR protocol described by Marr (2012), where the treatment plan is in the following order: processing current triggers (OCD obsessions and compulsions which are viewed as separate recent traumatic events); installing a future template (imagining successful future action); and then processing any past related disturbing events.

#### *Cognitive Behavioural Therapy (CBT)*

CBT followed the ERP model (Foa, Yadin, & Lichner, 2012); for simplicity, we apply the acronym CBT to refer to exposure and response prevention in the rest of this manuscript. This treatment requires patients to become exposed to stimuli (i.e., situations, thoughts, sensations) that evoke obsessive thoughts and/or distressing feelings without performing the rituals that aim to reduce that distress (response prevention). Exposure can be in the form of actual (*in-vivo*) contact with anxiety-provoking stimuli or in imagination (imaginal exposure). These procedures are systematically repeated and typically organised along a hierarchical sequence of exposure tasks which escalate in difficulty. With repeated practice, the distress associated with stimuli that trigger obsessions decreases, and

the associated urges to ritualise also decrease (Foa et al., 2012). The treatment proceeded in five phases: (1) assessment and psychoeducation about OCD; (2) development of a case formulation and orientation to the treatment procedures; (3) collaborative development of an exposure hierarchy; (4) repeated ERP in-session and in-between sessions as homework practice; (5) development of a relapse prevention plan to overcome future setbacks.

### ***Standardisation and quality control***

Both treatments were standardised to 16-session protocols and were delivered by therapists (n = 12; 6 EMDR and 6 CBT) that were qualified in each of the treatment modalities (years of experience: EMDR = 2 to 7; CBT = 3 to 15). The majority of therapists had experience of working with OCD cases prior to the trial, except for 3 EMDR therapists. Participating therapists had access to training sessions delivered by expert trainers in each of the treatment models; once prior to starting and once during the trial. Therapists also had access to group supervision and case discussion meetings for their respective treatments, approximately every 6 weeks. Their case notes were audited to ensure fidelity to the treatments and written feedback was provided by the study co-ordinator. No further fidelity checks or procedures were applied.

### ***Measures and data sources***

#### *Primary outcome measure*

The Yale-Brown Obsessive Compulsive scale (YBOCS; Goodman et al 1989) is a 10-item measure of OCD symptom severity; each item is rated from 0 (no symptoms) to 4 (extreme symptoms) yielding a total score between 0 – 40 with excellent interrater reliability ( $ICC = .98$ ) and internal consistency ( $\alpha = .89$ ). We applied the self-rated YBOCS developed by Baer et al. (1993), where a cut-off  $\geq 16$  is commonly

applied to identify moderate to severe OCD symptoms (Baer et al., 1993; Steketee, Frost, & Bogart, 1996). Cronbach's alpha for the baseline YBOCS in this sample was  $\alpha = .89$ .

#### *Other measures*

The Mini International Neuropsychiatric Interview (MINI) was developed for use by lay interviewers as a short but accurate psychiatric diagnostic interview based on DSM-IV criteria (Sheehan, 1998). The OCD module of the MINI can be delivered in person or over the telephone, with an average completion time of 15 minutes. The Obsessive Compulsive Inventory (OCI) is a 42-item questionnaire where respondents self-rate their distress levels (0 – 4 Likert scale) on each item across 7 domains: washing, checking, doubting, ordering, obsessing, hoarding and mental neutralising (Foa et al., 1998). The total OCI score ranges between 0 – 168 and has been found to correlate with the YBOCS (Foa et al., 1998). The PHQ-9 is a nine-item measure of depression symptoms (Kroenke, Spitzer, & Williams, 2001); items are rated using a 4-point Likert scale (0 – 3) yielding a total severity score between 0 – 27. The GAD-7 is a seven-item measure of anxiety symptoms (Spitzer, Kroenke, Williams, & Löwe, 2006); it is rated in the same way as the PHQ-9 yielding a total severity score between 0 – 21. The Work and Social Adjustment Scale (WSAS) is a measure of functioning across five domains: work, home management, social leisure activities, private leisure activities, and family and relationships. Each item is rated between 0 (no impairment) and 8 (very severe impairment), with a total severity score between 0 – 40.

De-identified demographic and clinical data were also collected for all consenting participants including age, gender, ethnicity, employment status, number of treatment sessions attended, and completion of agreed number of sessions (versus unilateral dropout).

#### ***Recruitment, randomisation and data collection***

As a feasibility trial, a formal sample size calculation was not estimated, but we aimed to recruit a minimum of 50 participants. All patients presenting to the service had telephone screening contacts with trained mental health practitioners as part of routine care. Those identified as presenting OCD symptoms at screening were referred to a telephone diagnostic interview with a researcher. All clinicians in the service were briefed about the recruitment process to ensure compliance. In addition, a researcher regularly reviewed waitlist records of primary diagnoses to identify any potential OCD cases that may have not been referred by screeners.

Patients who met MINI diagnostic criteria for OCD were invited to take part in the trial via telephone contacts, supplemented by a standard information sheet and consent form. Patients were excluded if (a) they did not meet criteria for OCD; or (b) OCD was not their primary reason for seeking treatment; or (c) they were using benzodiazepines; or (d) they were otherwise unsuitable for treatment in primary care (due to a history of psychotic or bipolar disorders, current suicidal risk, or current substance dependence).

Consenting participants were randomly assigned to either EMDR or CBT, using a computer-generated randomization schedule, by a research facilitator that was independent of the research and clinical teams. After randomization, participants were allocated to a trial therapist who prompted them to self-complete the YBOCS measure once per month (sessions 1, 4, 8, 12, 16). Secondary measures (PHQ-9, GAD-7, WSAS) were completed on a weekly basis, except for OCI which was only completed at the first and last treatment sessions. An independent researcher contacted all participants to gather (self-reported, paper-based) YBOCS measures at 6 months follow-up, regardless of completion (or dropout) status.

### ***Statistical analyses***

The analysis plan proceeded in three steps aiming (1) to assess the integrity of randomization by assessing the balance of characteristics in the randomised groups; (2) to assess the feasibility of delivering EMDR by comparing attendance and completion rates with CBT; (3) to compare clinical outcomes between groups based on *intention-to-treat* analysis. Cases with missing data (n = 9; 16.4%) were dealt with using multiple imputation based on an expectation maximization method (Schafer & Olsden, 1998).

In step 1, we compared baseline characteristics between cases allocated to EMDR and CBT, using categorical (chi-square), parametric (*t*-tests) and non-parametric (Mann-Whitney U) tests according to the distribution of each variable.

In step 2, we compared the percentage of cases completing treatment (versus dropouts) and those that provided 6-month follow-up data between groups using chi-square analysis. We also compared the mean number of treatment sessions between groups using a Mann-Whitney U test, given the skewed distribution of data.

In step 3, we used longitudinal multilevel modelling to examine the change (growth trend) in OCD symptoms over time, using a 2-level model with repeated YBOCS measures (level 1) nested within cases (level 2). Following conventional model building guidelines (Singer & Willett, 2003), we started by examining an unconditional (no predictors) model to determine the level of variance explained at each level. We then added covariates to the model, considered different covariance structures, assessed polynomial functions (i.e, quadratic, cubic) of covariates and assessed impact on model fit. *Goodness of fit* was assessed using  $-2 \log$  likelihood tests. After initial model checking, the primary analysis applied a 2-level linear growth model with unstructured covariance matrix. Covariates included baseline YBOCS severity, a group variable (CBT = reference category coded '0'; EMDR coded '1'), and a group\*time interaction term which was defined as the main hypothesis test (changes in YBOCS over time across groups). Random effects included intercepts and time slopes.

This model was initially implemented up to the time when the treatment ended, and then extended to 6-months follow-up data. As a sensitivity analysis, the same approach was applied using a 3-level model with therapists as the third-level random effects.

Raw means and standard deviations were used to calculate effect sizes (Cohen's  $d$ ) at post-treatment and 6 months follow-up, adjusting for unequal sample sizes.

Finally, we undertook some secondary and exploratory analyses. The numbers of cases attaining reliable and clinically significant improvement (RCSI) were compared between groups using chi-square analyses. A pre-post treatment reduction of 5 or more points plus a final score of YBOCS  $\leq 13$  has been taken to indicate RCSI in prior outcome studies (Diefenbach et al., 2015). Post-treatment scores in secondary outcome measures (OCI, PHQ-9, GAD-7, WSAS) were compared using Mann-Whitney U tests, given their skewed distribution.

We also assessed the rate of change in self-reported anxiety levels (GAD-7) up to session 16, by fitting non-linear growth trends in weekly time-series data for each treatment group. A cubic polynomial term was chosen based on the theoretical assumption that in-vivo exposure could increase anxiety before eventually leading to symptomatic improvements, thus potentially following an s-shaped (cubic) trend.

## **RESULTS**

### ***Random allocation and sample characteristics***

The CONSORT diagram in Figure 1 summarises the flow of participants through different stages of the trial. A total of 154 patients were contacted as part of the recruitment process, of whom 55 eligible and consenting participants were randomized and treated (EMDR = 29; CBT = 26). Table 1 presents sample characteristics for

trial participants; 61.8% were females, 41.8% were unemployed, 90.4% were of white British background, with a mean age of 32.04 (SD = 12.67) and mean YBOCS of 25.82 (SD = 6.40). Baseline severity estimates for secondary measures are also listed in Table 1. Statistical comparisons between the EMDR and CBT groups indicated no significant differences in any demographic or clinical characteristics (all  $p > .05$ ). Therefore, randomization was adequate and yielded comparable samples.

[Table 1]

### ***Feasibility analysis***

As shown in Table 2, the mean number of treatment sessions was 10.49 (SD = 6.18), with no significant differences between groups;  $U(55) = 366.50$ ,  $p = .86$ . Overall, 61.8% of participants completed their agreed number of treatment sessions (30.9% dropped out), with no significant differences in completion status between groups;  $\chi^2(1) = 0.35$ ,  $p = .55$ . Similarly, 83.6% of cases provided 6 months follow-up data, with no significant differences in loss to follow-up between groups;  $\chi^2(1) = 2.71$ ,  $p = .10$ .

[Table 2]

### ***Comparison of clinical outcomes***

The primary multilevel modelling indicated no significant main effects for the group\*time interaction term at post-treatment ( $B = -1.28$ ,  $SE = 0.88$ ,  $p = .16$ ) or at 6 months follow-up ( $B = -0.11$ ,  $SE = 0.42$ ,  $p = .80$ ). Main effects for time (post-treatment  $B = -2.66$ ,  $SE = 0.63$ ; 6 months  $B = -0.89$ ,  $SE = 0.30$ ) and baseline YBCOS (post-treatment  $B = 0.63$ ,  $SE = 0.09$ ; 6 months  $B = 0.57$ ,  $SE = 0.10$ ) were statistically significant in

all models (all  $p < .001$ ). Figure 2 shows the gradual change in YBOCS at each of the measurement points; confidence intervals (dashed curves surrounding linear growth trends) clearly overlap for both treatment groups. These results were unchanged in sensitivity analyses controlling for therapist effects; main effects for group\*time at post-treatment:  $B = -1.28$ ,  $SE = 0.88$ ,  $p = .16$ ; 6 months:  $B = -0.11$ ,  $SE = 0.44$ ,  $p = .80$ ; level-3 random effects:  $Z = 1.31$ ,  $p = .19$ .

[Figure 2]

The YBOCS effect sizes were  $d = -0.24$  ( $p = .38$ ) post-treatment and  $d = -0.03$  at 6 months follow-up ( $p = .90$ ); where the negative sign favours the control group (CBT). Raw means used in effect size calculations are presented in Table 2, along with post-treatment estimates for secondary outcome measures, none of which were significantly different between groups (all  $p > .05$ ). The proportions of cases attaining RCSI criteria were higher in the CBT group, though not statistically significant post-treatment ( $p = .14$ ) or at 6 months follow-up ( $p = .57$ ). Figure 3 shows non-linear growth curves for weekly changes in anxiety symptoms across groups; there was no evidence of differential trends in the rate of change at early or later phases of treatment.

[Figure 3]

## **DISCUSSION**

### ***Main findings***

This pragmatic trial is the first experimental demonstration that EMDR is feasible and safe to apply as a treatment for obsessive-compulsive disorder in routine clinical care, by comparison to CBT. Attendance and completion rates were similar across groups,

indicating that EMDR was as well tolerated as CBT. Our analyses indicated that there were no significant differences between treatments in any of the outcome measures post-treatment or at 6 months follow-up. We note, however, that this trial was not powered to detect small outcome differences between treatments (if these exist). Our preliminary effect size calculations yielded a small effect favouring CBT ( $d = -0.24$ ), though this was not statistically significant and virtually disappeared at 6 months follow-up ( $d = -0.03$ ). Furthermore, CBT cases were not more prone to dropout and we found no evidence that CBT cases experienced more intense anxiety at the early sessions of treatment by comparison to EMDR cases. These findings challenge the popular clinical notion that exposure can lead people to feel worse before they get better (Richard & Lauterbach, 2006).

### ***Strengths and limitations***

By comparison to earlier case-series, we recruited patients accessing a routine primary care setting and took steps to mitigate selection bias (such as screening waitlist records). We note that a considerable number of patients approached for screening did not consent to this (89 of 154; 58%). Nevertheless, the pre-treatment YBOCS mean (25.8) for the sample included in the trial was within the range of symptom severity reported in prior trials (21.8 to 28.7; reviewed by Fisher & Wells, 2005), so our sample was comparable to previous studies.

An important limitation concerns the sample size, which does not rule out the possibility that there could be small differences between treatments. The post-treatment effect size reported in this study could be used to calculate a sample size for future non-inferiority trial designs. The funding and time constraints of this study did not enable us to undertake more stringent fidelity checks, such as ratings of video recorded sessions to assess adherence and competence of treatment delivery. This is also an important limitation, as we cannot be sure about the extent to which the interventions were

delivered with competence and fidelity to the respective treatment manuals. We note, however, that meta-analytic evidence indicates that there are no significant differences in effect sizes between OCD trials with and without treatment integrity checks (Olatunji et al., 2013). Like most prior OCD trials (Eddy, Dutra, Bradley, & Western, 2004; Rosa-Alcázar, Sánchez-Meca, Gómez-Conesa, & Marín-Martínez, 2008), this study was also limited by a relatively brief follow-up period which did not enable us to assess the maintenance of improvements beyond 6 months after treatment.

### ***Implications for theory and research***

Acknowledging the need for further replication in larger samples, the current evidence indicates that EMDR and CBT attain similar outcomes in the treatment of OCD. It is likely that some of the clinical effect in both interventions could be explained by common factors. For example, meta-analyses of several studies demonstrate that psychotherapy outcomes are influenced by the degree to which therapists are empathic (Elliott, Bohart, Watson, & Greenberg, 2011), foster a positive therapeutic alliance (Horvath, Del Re, Flückiger, & Symonds, 2011), enhance patients' expectations (Constantino et al., 2011) and motivation to change (Norcross, Krebs, & Prochaska, 2011). Indeed, practice guidelines for OCD discuss the importance of these common factors to enhance collaboration and therapeutic change (Koran et al., 2007).

Although common factors are important, previous studies comparing CBT with other active treatments or psychological placebo controls (relaxation training, stress and anxiety management) have reported small but statistically significant advantages favouring CBT (Olatunji et al., 2013). This leads us to think that common factors (presumed to be present in active control conditions) may offer a facilitative and therapeutic context within which specific change processes can be employed to enhance OCD symptom improvements.

The technical differences between treatments in this trial are not enlightening in this regard, since EMDR patients improved without being directed towards in-vivo exposure and CBT patients improved without bilateral stimulation or the processing of past memories. On the other hand, it is plausible that EMDR and CBT apply similar change processes, which are more specific than the common facilitative factors described above.

The most apparent shared change mechanism is that of exposure to anxiety-provoking stimuli, which EMDR applies *imaginally* (i.e., in imagination) and CBT applies both imaginally and in-vivo (Foa et al., 2012). Previous studies have shown that treatments that combine in-vivo plus imaginal exposure attain better outcomes than exposure in-vivo alone (Rosa-Alcázar et al., 2008). Hence it is possible that imaginal exposure is a key shared component that enhances therapeutic change. Wolpe described the successful treatment of anxiety through imaginal exposure as early as the 1950's (Wolpe, 1958), leading to the eventual development of *emotional processing theory* which informs –to some extent– the hypothesis of systematic desensitization that is common to EMDR and ERP. From this perspective, one possible explanation for our results is that patients in both groups attained some degree of desensitization to obsessional thoughts, and (repeated and prolonged) imaginal exposure is sufficient to achieve this. In fact, both treatments aim to observe within-session-habituation, as reported by the patient in subjective units of distress.

Even though emotional processing theory is still widely accepted by clinicians, there are some contradictory findings that challenge the basic tenets of habituation. For example, studies have shown that within-session-habituation does not necessarily correlate with longer term anxiety reduction (Baker et al., 2010), fears can often be spontaneously reinstated after extinction (Craske & Mystkowski, 2006), and successful fear reduction can occur in the absence of exposure (Rachman, Craske, Tallman, & Solyom, 1986). An alternative

perspective could be offered by contemporary theories on the mechanisms of fear acquisition and inhibitory learning. Supported by numerous laboratory and clinical studies (see Craske et al., 2014), *inhibitory learning theory* posits that a learned association between conditioned and unconditioned stimuli (CS-US) is not entirely eradicated during extinction (i.e., during exposure procedures). The original CS-US pairing is left intact as a memory, while a new and secondary learning set is formed (CS no-US) that serves to inhibit and effectively compete against the original set. Concerning OCD treatment, it is possible that CBT (ERP results in disconfirmation of feared expectations) and EMDR (installation of a positive cognition competes against feared expectations) facilitate inhibitory learning in slightly different ways, which accounts for distress reduction over time with secondary gains in self-efficacy and functioning. Overall, aside from common facilitative factors, it is plausible that both EMDR and CBT rely extensively on shared change mechanisms such as exposure and reappraisal strategies. While our theoretical interpretations are largely speculative, future studies could aim to investigate possible mechanisms of action which may be common to treatments that involve imaginal exposure and inhibitory learning processes for the treatment of OCD.

In conclusion, both treatments studied in this trial had similar effects in the treatment of OCD, although it is important to remark that some patients dropped out and did not attain symptom improvements. It is possible that some may find one or the other treatment more tolerable, credible or acceptable. Future qualitative studies focusing on acceptability and investigations of mechanisms of change may help us to better understand how to maximise the effectiveness of psychological treatments for OCD.

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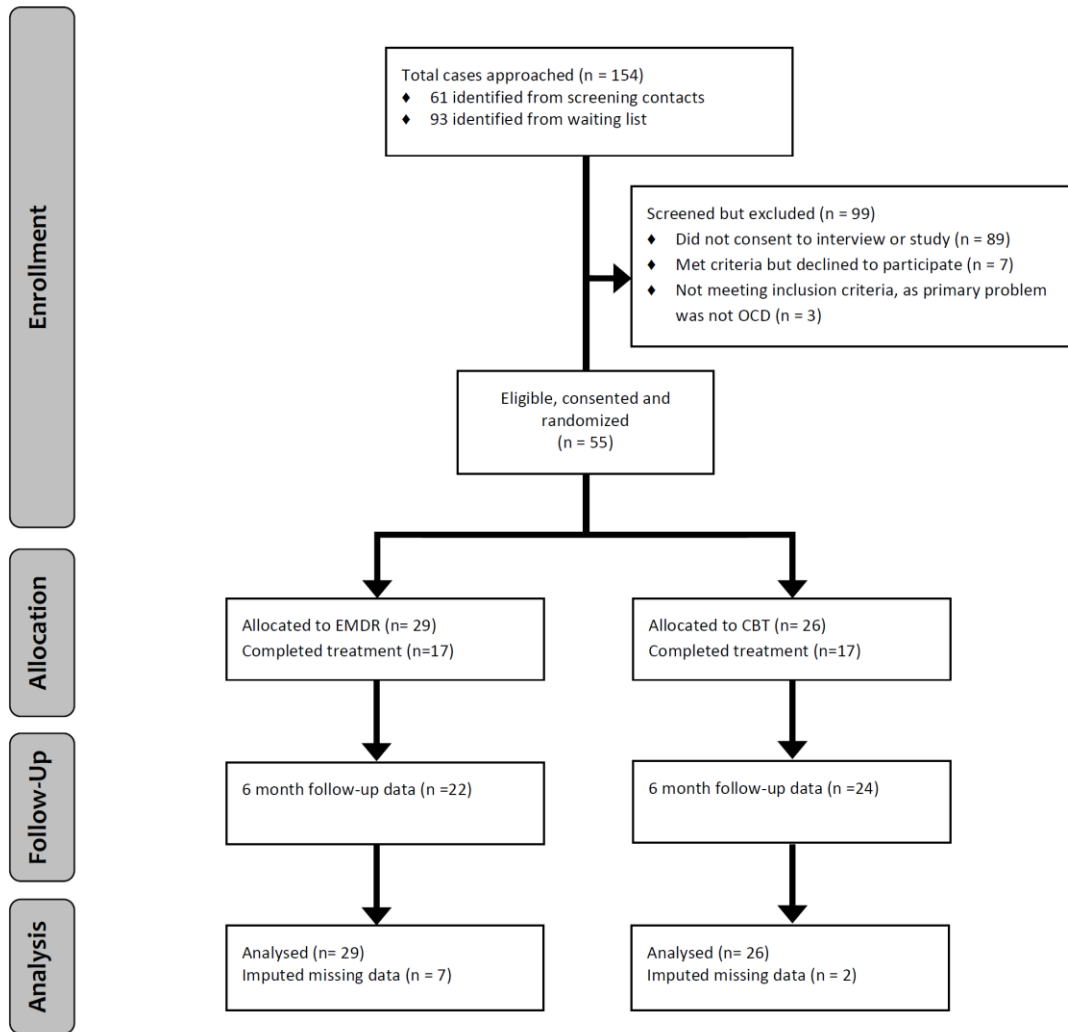
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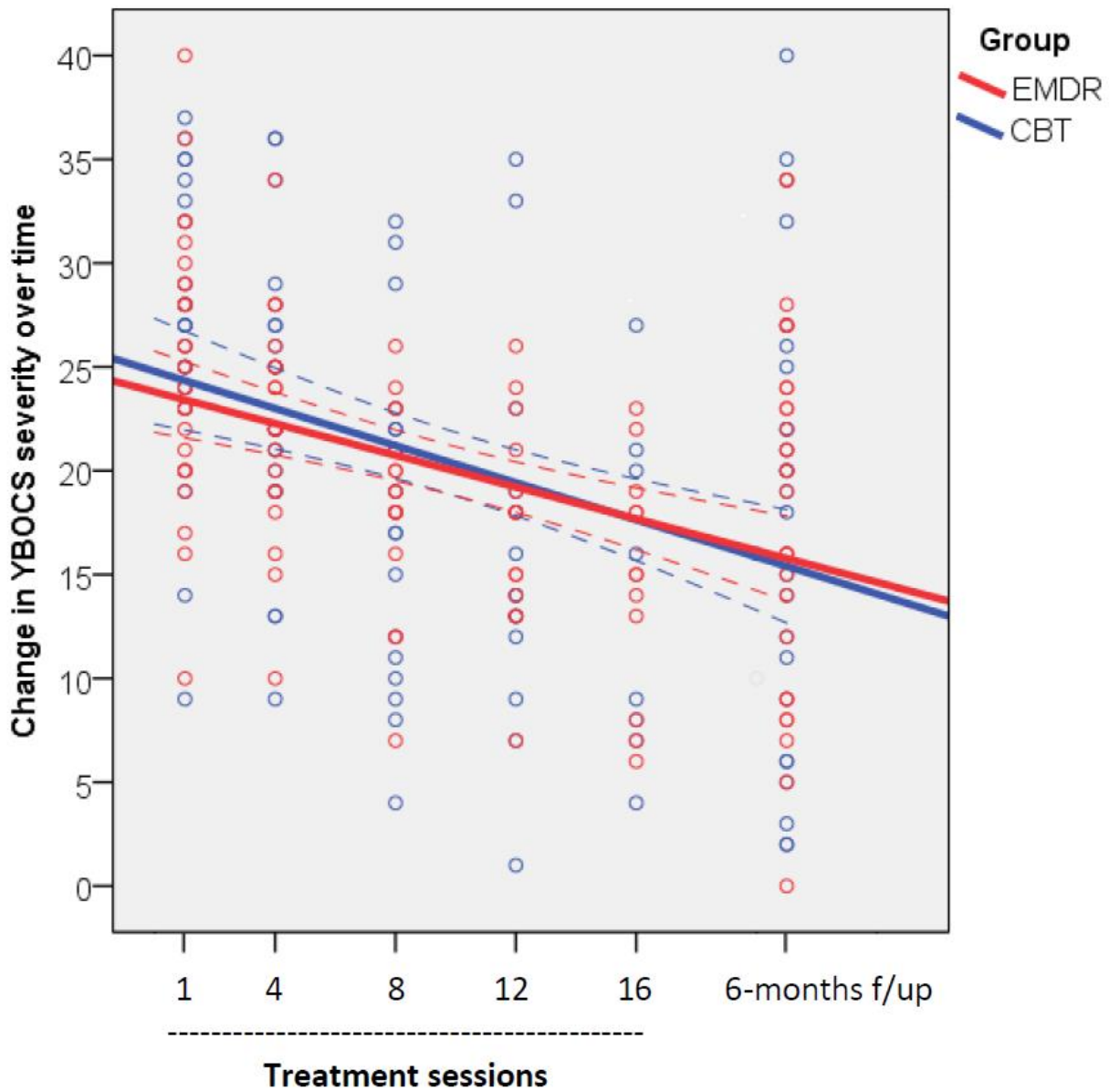
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**Figure 1. CONSORT diagram**



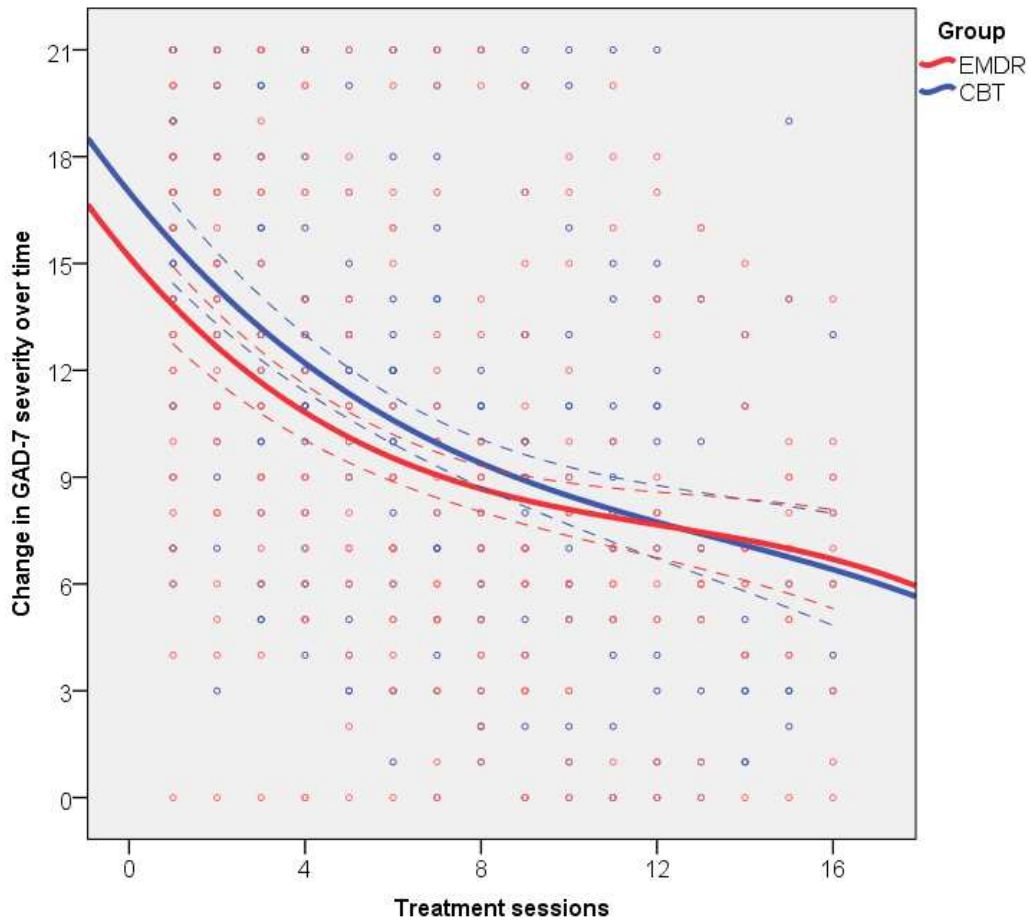
**Figure 2. Linear growth trends and confidence intervals for YBOCS measures**



[Figure legend]

Solid lines = YBOCS linear growth trend; dashed curves = 95% confidence intervals; EMDR = eye-movement desensitization and reprocessing; CBT = cognitive behavioural therapy; f/up = follow-up

**Figure 3. Non-linear growth trends and confidence intervals for weekly anxiety (GAD-7) measures**



[Figure legend]

Solid curves = GAD-7 cubic growth trend; dashed curves = 95% confidence intervals; EMDR = eye-movement desensitization and reprocessing; CBT = cognitive behavioural therapy

**Table 1. Sample characteristics and comparisons between groups**

	<b>Full sample</b> N = 55 (100%)	<b>EMDR group</b> N = 29 (52.7%)	<b>CBT group</b> N = 26 (47.3%)	<b>test statistic</b>	<b>p</b>
<b>Demographics</b>					
Females	34 (61.8)	17 (58.6)	17 (65.4)	$\chi^2(1)=0.26$	.61
Mean age (SD)	32.04 (12.67)	30.90 (9.79)	33.31 (15.37)	U(55)=376.00	.99
Unemployed	23 (41.8)	14 (48.3)	17 (65.4)	$\chi^2(1)=1.05$	.31
Ethnicity*					
White British	47 (90.4)	23 (88.5)	24 (92.3)	$\chi^2(1)=0.22$	.64
Other	5 (9.6)	3 (11.5)	2 (7.7)		
<b>Baseline severity measures</b>					
YBOCS mean (SD)	25.82 (6.40)	25.07 (6.23)	26.65 (6.61)	$t(53)=0.92$	.36
OCI mean (SD)	71.13 (30.19)	73.93 (28.94)	68.12 (31.80)	$t(50)=-0.69$	.49
PHQ-9 mean (SD)	12.40 (6.58)	11.86 (6.30)	13.04 (6.98)	$t(50)= 0.64$	.52
GAD-7 mean (SD)	14.38 (5.06)	13.64 (5.45)	15.25 (4.53)	U(52)=283.50	.33
WSAS mean (SD)	18.51 (10.21)	17.81 (10.19)	19.29 (10.39)	$t(49)= 0.51$	.61

CBT = cognitive behavioural therapy; EMDR = eye movement desensitization and reprocessing; YBOCS = Yale-Brown Obsessive Compulsive Scale; OCI = Obsessive Compulsive Inventory; PHQ-9 = measure of depression symptoms; GAD-7 = measure of anxiety symptoms; WSAS = work and social adjustment scale;  $t$  = Student's t-test;  $U$  = Mann-Whitney U test;  $\chi^2$  = Chi-square test; \* percentages exclude 3 cases with missing data

**Table 2. Comparison of treatment outcomes between groups**

	<b>Full sample</b> N = 55 (100%)	<b>EMDR group</b> N = 29 (52.7%)	<b>CBT group</b> N = 26 (47.3%)	<b>test statistic</b>	<b>p</b>
<b>Attendance and follow-up data</b>					
Mean treatment sessions (SD)	10.49 (6.18)	10.17 (6.63)	10.85 (5.73)	U(55)=366.50	.86
Completion status*					
Completed	34 (61.8)	17 (58.6)	17 (65.4)	$\chi^2(1)= 0.35$	.55
Dropped out	17 (30.9)	10 (34.5)	7 (26.9)		
Referred onwards	4 (7.3)	2 (6.9)	2 (7.7)		
Assessed @6 months follow-up	46 (83.6)	22 (75.9)	24 (92.3)	$\chi^2(1)= 2.71$	.10
<b>Outcomes data</b>					
Post-treatment outcomes					
YBOCS mean (SD)	17.75 (8.69)	18.72 (8.01)	16.65 (9.43)	$t(53)= -0.88$	.38
YBOCS RCSI**	16/53 (30.2)	6/28 (21.4)	10/25 (40.0)	$\chi^2(1)= 2.16$	.14
OCI mean (SD)	46.78 (35.22)	47.90 (33.24)	45.54 (37.93)	U(55)=403.50	.66
PHQ-9 mean (SD)	7.64 (7.03)	7.55 (6.99)	7.73 (7.20)	U(55)=370.00	.91
GAD-7 mean (SD)	8.96 (6.09)	9.14 (6.18)	8.77 (6.10)	U(55)=390.00	.83
WSAS mean (SD)	10.91 (9.76)	11.17 (9.33)	10.62 (10.40)	U(55)=402.00	.67
6 months follow-up outcomes ♦					
Mean YBOCS score (SD)	18.09 (9.55)	18.24 (8.59)	17.92 (10.69)	$t(53)= -0.12$	.90
YBOCS RCSI**	15/53 (28.3)	7/28 (25.0)	8/25 (32.0)	$\chi^2(1)= 0.32$	.57

CBT = cognitive behavioural therapy; EMDR = eye movement desensitization and reprocessing; YBOCS = Yale-Brown Obsessive Compulsive Scale; OCI = Obsessive Compulsive Inventory; PHQ-9 = measure of depression symptoms; GAD-7 = measure of anxiety symptoms; WSAS = work and social adjustment scale; RCSI = reliable and clinically significant improvement;  $t$  = Student's t-test;  $U$  = Mann-Whitney U test;  $\chi^2$  = Chi-square test; \* statistical comparisons made between completers vs. dropouts; \*\* comparisons made between cases that scored above YBOCS cut-off  $\geq 16$ ; ♦ calculated using imputed 6 months outcomes data