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Review

Differential efficacy of cognitive-behavioral therapy and pharmacological treatments for pediatric obsessive–compulsive disorder: A meta-analysis

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ABSTRACT

The aim of this paper is to present a meta-analysis about the differential efficacy of cognitive-behavioral therapy (CBT), pharmacological and combined treatment for pediatric obsessive–compulsive disorder (OCD). The literature research and the application of the inclusion criteria enabled us to locate 18 studies, yielding a total of 24 independent comparisons between a treated (10 pharmacological, 11 CBT, and 3 combined interventions) and a control group. All types of interventions were efficacious in reducing obsessive–compulsive symptoms, with effect sizes adjusted by the type of control group of $d = 1.203$ for CBT, $d = 0.745$ for pharmacological treatments, and $d = 1.704$ for mixed treatments. Depression, anxiety and other secondary responses were also improved, especially with CBT interventions. The analysis of moderator variables showed that the CBT protocol and the total of intervention hours exhibited a significant influence on the effect size. Within pharmacological treatment, clomipramine ($d = 1.305$) was more efficacious than selective serotonin reuptake inhibitors ($d = 0.644$), but its adverse effects were more severe. Finally, the clinical implications of the results are discussed.

Contents

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1. Introduction

Obsessive–compulsive disorder (OCD) is a serious psychological disorder which occurs in approximately 2% of children and adolescents (Apter et al., 1996; Canals, Hernández-Martínez, Cosi, & Voltas, 2012; Rapoport et al., 2000). The main symptoms of pediatric OCD are obsessions and compulsions (American Psychiatric Association, 2013). As in adults, these interfere with the individual's daily life and cause a serious functional impairment (Piacentini, Bergman, Keller, & McCracken, 2003; Valderhaug & Ivarsson, 2005). The OCD is often associated with other psychological disorders, such as tics, attention deficit-hyperactivity disorder, depression, separation anxiety disorder, specific phobias, and multiple anxiety disorders that increase the degree of discomfort and complicate its treatment and prognostic (Storch et al., 2010). The comorbidity has been associated with a lower treatment response and a greater percentage of relapse after treatment both in medication and psychological interventions (Geller, Biederman, Stewart, Mullin, Farrell, et al., 2003; Krebs & Heyman, 2010; March et al., 2007).

The severe consequences of OCD in lives of children and adolescents have encouraged researchers and clinicians to develop new assessment instruments and to improve pharmacological and psychological interventions. Cognitive-behavioral therapy (CBT) has been the most investigated treatment model, with exposure with response prevention (ERP) being the main component. At least in adults, the efficacy of ERP seems to be similar regardless of whether it is applied alone or in combination with other techniques (Rosa-Alcázar, Sánchez-Meca, Gómez-Conesa, & Marín-Martínez, 2008). The treatment components that most frequently accompany ERP are neurobiological psychoeducation (March & Mulle, 1998), family-based treatments with parent training (Barrett, Healy-Farrell, & March, 2004; Freeman et al., 2008), intervention in narrative context (March & Mulle, 1998; Wagner, 2003), and cognitive or anxiety management techniques with the use of age appropriate metaphors to facilitate cognitive restructuring (Barrett et al., 2004; March & Mulle, 1998; Pediatric OCD Treatment Study [POTS] Team, 2004; Piacentini & Langley, 2004).

Empirical studies conducted on pediatric OCD have examined the efficacy of treatments in different modalities. For example, no relevant differences have been found between individual and group CBT, with efficacy figures ranging between 61% and 65% for both modalities (Barrett et al., 2004). Similarly, intensive CBT seems to be as efficacious as CBT applied in a longer format (Franklin et al., 1998; Storch et al., 2007). CBT has been investigated with the inclusion of certain modifications such as telephone format (Turner, Heyman, Futh, & Lovell, 2009), community-based CBT (Farrell, Schlup, & Boschen, 2010), or web-camera delivered CBT (Storch et al., 2011). However, the differential efficacy between the components of CBT has not been sufficiently investigated. Only the study by Simons, Schneider, and Herpertz-Dahlmann (2006) has compared the benefits of ERP and meta-cognitive therapy, finding better results for ERP.

Several studies have examined the efficacy of family-based CBT programs, finding very heterogeneous improvement percentages of around 25–65% (Barrett et al., 2004; Piacentini et al., 2011). On the one hand, some reviews have concluded that the efficacy of family-based CBT presents significant differences between fathers and mothers according to gender, age of children (Bögels & Phares, 2008), and parental style dimensions (McLeod, Wood, & Weisz, 2007). Other studies have concluded that individual exposure-based CBT can be considered as a probably efficacious treatment, whereas family-based CBT must be considered as a possibly efficacious treatment (Barrett, Farrell, Pina, Peris, & Piacentini, 2008; Sukhodolsky, Gorman, Scahill, Findley, & McGuire, 2013).

Pharmacological treatments of pediatric OCD have received wide empirical support from several randomized controlled trials, showing an adequate efficacy to reduce obsessive–compulsive symptoms. Although clomipramine has shown greater efficacy than selective serotonin reuptake inhibitors (SSRIs), its adverse side-effects are more severe than SSRIs (anticholinergic, adrenergic and histaminergic effects). As a consequence, SSRIs are preferred to clomipramine, as they are safer and better tolerated by the patients (Geller, Biederman, Stewart, Mullin, Farrell, et al., 2003; Geller, Biederman, Stewart, Mullin, Martin, et al., 2003; Watson & Rees, 2008).

Only a few studies have assessed the efficacy of combining pharmacological treatment and CBT (Franklin et al., 2011; POTS, 2004). The results have been better when CBT is added to medication than when the treatment is composed of medication alone. However, more controlled studies are needed to confirm and extend these results.

The American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (2012) recommends CBT as the first choice when OCD presents mild to moderate severity, whereas SSRIs combined with CBT are indicated for moderate to severe cases, presence of comorbidity, or absence of clinicians specialized in CBT.

To date, five meta-analytic studies have been published on the efficacy of interventions for pediatric OCD (Abramowitz, Whiteside, & Deacon, 2005; Freeman et al., 2007; Geller, Biederman, Stewart, Mullin, Farrell, et al., 2003; Geller, Biederman, Stewart, Mullin, Martin, et al., 2003; O'Kearney, 2007; Watson & Rees, 2008). The main results of these meta-analyses are summarized in Table 1. Four of the five meta-analyses have investigated the efficacy of CBT. Although all of them exhibited mean effects of large magnitude and statistically significant, these mean effect estimates seem to be somewhat heterogeneous. Thus, Abramowitz, Whiteside, and Deacon (2005) found an average effect of $d_R = 1.98$, whereas Freeman et al. (2007) reported a slightly lower mean effect of $d_R = 1.55$ (see Table 1). O'Kearney (2007) computed the effect sizes for 5 CBT comparison studies and for 14 one-group studies, obtaining d_R indices for obsessive–compulsive symptoms that ranged from 0.78 to 3.49. Given the large variability showed by the effect estimates, he did not report a mean effect. The d_R index used in Abramowitz et al. (2005), Freeman et al. (2007), and O'Kearney (2007) was the standardized mean change between the pretest–posttest measures obtained in each CBT group. Due to the large number of internal validity threats of this effect size index, O'Kearney (2007) considered that the effect estimates obtained from these studies could be overestimations of the true treatment effect. In order to correct the effect estimates, he adjusted their d_R estimates by the mean d_R index obtained with the control groups, concluding that, on average, the mean effect of CBT is close to $d = 1$. In order to avoid the internal validity threats of the d_R index, Watson and Rees (2008) selected only randomized controlled studies that included a psychological or pharmacological treatment and a control group. Thus, they were able to calculate a more adequate effect

Table 1
Summary of the average effect sizes obtained in previous meta-analyses on pediatric OCD treatment.

Meta-analysis	Designs included	Effect size index	*d* indices for psychological treatments				*d* indices for pharmacological treatments					*d* Placebo
			Global	Individual CBT	Group CBT	Family CBT	Global	CLM	FLX	SER	PAR	FLV	
Geller, Biederman, Stewart, Mullin, Farrell, et al. (2003) and Geller, Biederman, Stewart, Mullin, Martin, et al. (2003)	RCTs	*d* and d_C					0.46 *k* = 10						
Abramowitz et al. (2005)	RCTs and Quasi-experimental	d_R	1.98				1.13	1.10					0.48
			k = 10				*k* = 10	*k* = 4					*k* = 7
Freeman et al. (2007)	RCTs and Quasi-experimental	d_R	1.55	1.77	0.76	1.88							
			k = 12	*k* = 7	*k* = 3	*k* = 2							
O'Kearney (2007)	RCTs and Quasi-experimental	d_R	0.78–3.49[a]										
			k = 19										
Watson and Rees, 2008	RCTs	d_C	1.45				0.48	0.85	0.51	0.47	0.44	0.31	
			k = 13				*k* = 10	*k* = 2	*k* = 3	*k* = 2	*k* = 2	*k* = 1	

RCTs: Randomized-controlled trials. *d*: standardized mean difference between the treatment and control groups in the posttest. d_C: standardized mean difference between the change scores of the treatment and control groups. d_R: standardized mean difference between the pretest–posttest measures. *k*: number of studies. CLM: clomipramine. FLX: fluoxetine. SER: sertraline. PAR: paroxetine. FLV: fluvoxamine.
[a] Effect sizes for individual studies.

size, the d_C index, defined as the standardized mean difference between the pretest–posttest change of the treated group and the pretest–posttest change of the control group. As Table 1 shows, CBT reached a large mean effect size of $d_C = 1.45$, slightly lower than those reported by Abramowitz et al. (2005) and Freeman et al. (2007). Freeman et al. (2007) also calculated separate mean effects for individual, group, and family-based CBT (see Table 1), concluding that individual and family-based CBT could be the most promising interventions for OCD in childhood and adolescence. Abramowitz et al. (2005) also calculated a combined effect size of ERP for anxiety and depression symptoms, which was not statistically significant ($d_R = 0.48$).

With regards to pharmacological treatments, Geller, Biederman, Stewart, Mullin, Farrell, et al. (2003) and Geller, Biederman, Stewart, Mullin, Martin, et al. (2003) conducted a meta-analysis that included 12 randomized controlled trials and used the d_C index as the effect size. The active treatment groups were four SSRIs (paroxetine, fluoxetine, fluvoxamine, and sertraline) and clomipramine. Placebo (10 groups) or desipramine (two groups) was administered in non-active treatment groups. On average, the mean effect of the pharmacological treatments was $d_C = 0.45$, clearly lower than that obtained for CBT in the meta-analyses commented above. This effect estimate was very similar to that obtained by Watson and Rees (2008), $d_C = 0.48$, and clearly lower than that obtained by Abramowitz et al. (2005) with the d_R index, $d_R = 1.13$. Out of the four moderator variables analyzed by Geller, Biederman, Stewart, Mullin, Farrell, et al. (2003) and Geller, Biederman, Stewart, Mullin, Martin, et al. (2003), study design, type of dependent outcome measure, type of outcome score and publication year, only the outcome measure showed a significant influence on the effect size, with the clinician measures (e.g., the *Children Yale-Brown Obsessive–Compulsive Scale*, CY-BOCS; Scahill et al., 1997) reflecting more improvements than the unique self-report measure used in the computation of the effect size (the *Leyton Obsessive–Compulsive Inventory*, LOI-CV; Berg, Rapoport, & Flament, 1986).

Clomipramine exhibited mean effects of large magnitude, both in Abramowitz et al. (2005), $d_R = 1.10$, and in Watson and Rees (2008), $d_C = 0.85$, with the differences being attributable to the different effect size index used. SSRIs obtained lower mean effects than clomipramine in the Watson and Rees' (2008) meta-analysis (see Table 1), although with less adverse effects.

1.1. Objectives

Although several meta-analyses have already been published about the efficacy of psychological and/or pharmacological treatments for pediatric OCD, there are still some issues that remain unsolved. Firstly, the majority of randomized controlled trials (RCTs) on pediatric OCD have been conducted very recently, and therefore six of them were not included in the previous meta-analyses (Bolton et al., 2011; Franklin et al., 2011; Freeman et al., 2008; Piacentini et al., 2011; Storch et al., 2011; Williams et al., 2010). Secondly, despite the fact that the combined treatment of CBT plus SSRI is the first option for moderate to severe cases (American Academy of Child & Adolescent Psychiatry Committee on Quality Issues, 2012), empirical evidence about its effectiveness has not yet been meta-analyzed. In our meta-analysis three comparisons between combined treatment and control groups were included. Thirdly, measures of secondary symptoms such as anxiety, depression, functional impairment, etc., were not analyzed in previous meta-analyses. Only Abramowitz et al. (2005) computed a combined effect size for anxiety and depression symptoms. Fourthly, the influence of treatment, participant, and methodological variables on the effect sizes has not been addressed in depth to date. Hence, the purpose of this meta-analysis was to address these

issues. In addition, we intended to propose a predictive model that adjusts the effect estimates of CBT, pharmacological, and combined treatments according to the methodological quality of the studies.

2. Method

2.1. Study selection criteria

In order to be included in the meta-analysis, the studies had to fulfill the following criteria: (1) to examine the efficacy of CBT, pharmacological or combined treatment for OCD in participants younger than 19 years old, and diagnosed by standardized criteria (e.g. any version of the Diagnostic and Statistical Manual, DSM, or International Classification of Diseases, ICD); (2) inclusion of a control group; (3) the sample size in the posttest had to be of at least 5 participants per group; (4) the statistical data reported in the study had to allow us to compute the effect sizes; (5) due to language limitations, the study had to be written in English, French or Spanish; (6) studies had to be published or carried out between 1980 and 2012, both included.

2.2. Search strategy

In the first place, several electronic databases were consulted: Medline, *PsycINFO*, *PsycARTICLES*, *ERIC*, *Cochrane Library*, and *Google Scholar*, as well as the Spanish databases *CSIC* and *PSICODOC*. The following keywords were combined, in English and Spanish: *obsessive–compulsive, OCD, treatment, cognitive behavioral therapy, CBT, selective serotonin reuptake inhibitors, SSRI, clomipramine, exposure response prevention, ERP, trial, pediatric, child* and adolesce**, which had to be in the title or in the abstract. Second, the references of the five meta-analyses referred to above and seven systematic reviews were consulted (Barrett et al., 2008; Himle, Van Etten, & Fischer, 2003; March, 1995a; March, Franklin, Nelson, & Foa, 2001; Rosa-Alcázar, Iniesta-Sepúlveda, & Rosa-Alcázar, 2012; Turner, 2006; Wolff & Wolff, 1991). Thirdly, we also reviewed the references of the located studies. In order to locate unpublished papers, e-mails were sent to 26 authors selected from those most prolific in the field. Eleven responses were obtained, but none of them allowed us to uncover any unpublished study.

The search strategy produced a total of 1900 references, finding 18 articles that fulfilled the selection criteria, all of them written in English and carried out between 1985 and 2012. Five additional articles included a control group, but they were excluded from the meta-analysis due to different reasons: for not reporting enough statistical data of the groups (Rapoport, Elkins, & Mikkelsen, 1980), for presenting the same data than those reported in Flament, Rapoport, and Kilts (1985; Flament, Rapoport, Murphy, Berg, & Lake, 1987), for presenting data of a subsample of the De Veaugh-Geiss et al.'s (1992) study (March, Johnston, Jefferson, Kobak, & Greist, 1990), for reporting mixed data from tic disorders and OCD (Perlmutter, Leitman, Garvey, Hamburger, Feldman, & Leonard, 1999), and finally the Geller, Biederman, Stewart, Mullin, Farrell, et al.'s (2003) and Geller, Biederman, Stewart, Mullin, Martin, et al.'s (2003) study was also excluded because the placebo data in the pretest pertained to a sample of participants that previously had participated in an open psychopharmacological trial.

The 18 articles finally selected produced 24 comparisons between a treatment group (11 CBTs, 10 pharmacological, and three combined) and a control group (11 pill placebo[1], seven wait-list, and three relaxation training). The total sample included 1223 participants in the posttest (656 in the treatment groups and 567 in the control groups). The studies were carried out in the USA (75%, 18 comparisons), the United Kingdom (16.7%, 4 comparisons), and Australia (8.3%, 2 comparisons). Despite our efforts to locate unpublished studies, only one was found (Himle, 2003).[2]

The degree of overlap between our meta-analysis and the previous ones was 29.2% with Geller, Biederman, Stewart, Mullin, Farrell, et al. (2003) and Geller, Biederman, Stewart, Mullin, Martin, et al. (2003), 29.2% with Abramowitz et al. (2005), 11.1% with Freeman et al. (2007) and 11.1% with O'Kearney (2007). In all cases, the degree of overlap was calculated taking the number of studies in our meta-analysis that were included in each of the previous ones and dividing it by the total number of studies in our meta-analysis. The highest degree of overlap was 66.7% with Watson and Rees (2008). In spite of this fact, the inclusion of recent experimental studies on CBT and combined treatments, together with the methodological improvements in our meta-analysis, justify the originality of our results.

2.3. Coding of moderator variables

In order to examine the potential influence of characteristics of the studies on the effect sizes, treatment, participant and methodological variables were coded in CBT and pharmacological treatments. Moderator variables related to CBT were: (a) the treatment duration (number of weeks), (b) the treatment intensity (number of weekly hours), (c) the treatment magnitude (total number of intervention hours per participant), (d) the degree of parental involvement (low, moderate or high), (e) the focus of the intervention (the OCD child or the whole family), (f) the inclusion of exposure and response prevention (ERP), (g) the mode of CBT (individual versus in group), and (h) manualized treatment protocol. With regards to pharmacological treatments, the following variables were coded: (a) the type of antidepressant (tricyclic or SSRI), (b) the initial dose (mg/day), (c) the maintenance dose (mg/day), and (d) the treatment duration (number of weeks), and (e) the percentage of participants that suffered side effects.

The following variables related to the participants were coded: (a) the mean age of the sample (in years), (b) the gender distribution (percentage of males), (c) the mean duration of the OCD (in years), (d) whether they had received any previous treatment (somebody or nobody), and (e) the comorbidity in the sample.

Several methodological variables were coded: (a) the type of control group (pill placebo, unspecific psychological attention, or nonnactive-waitlist-control), (b) the standardized mean difference in the pretest, (c) the differential attrition between treatment and control groups, and (d) the methodological quality of the study (on a scale of 0–6 points). The items that composed the methodological quality scale were: (1) random versus nonrandom assignment of participants to the groups; (2) the adequacy of the control group (low, moderate, high); (3) the sample size in the posttest; (4) one minus the attrition in the treatment group; (5) the use of intent-to-treat analysis, and (6) the use of blinded assessors. Each one was rated from 0 to 1.

The coding process was carried out in a standardized and systematic way. In order to do this, a codebook and a protocol for registering the variables were produced.* To assess the reliability

[1] As the Franklin et al.'s (2011) study did not include a control group, it was unable to meet all of our selection criteria. Due to the relevance of this study in the field, we decided to include it in the meta-analysis. With this purpose, we paired the control group of Pediatric OCD Treatment Study (POTS) Team (2004) with the treatment groups of Franklin et al. (2011). No statistically significant differences were found in sociodemographic characteristics nor in the pretest for obsessive-compulsive symptoms between the participants of these studies.

[2] This study was located in Watson and Rees's (2008) meta-analysis. As we were not able to obtain a reprint of this paper, the data included in our meta-analysis were those reported in Watson and Rees (2008, p. 492, Table 3).

of the coding process, 20% of the studies were randomly selected and subjected to a double coding process by coders. The coders were trained by one of the authors during three one-hour sessions. The inconsistencies between the coders were solved by consensus. Very satisfactory inter-coder reliability was found, with kappa coefficients ranging between 0.67 and 1 for the qualitative variables, and intra-class correlations between 0.90 and 1 for the continuous ones.

2.4. Computation of effect sizes

The effect size index was the standardized mean difference between the change scores of the treatment and the control groups (Morris, 2008). For each study, this index was calculated subtracting the mean pretest–posttest difference of the control group ($\bar{y}^C_{\text{Pre}}$ and $\bar{y}^C_{\text{Post}}$) from the mean pretest–posttest difference of the treatment group ($\bar{y}^T_{Pre}$ and $\bar{y}^T_{Post}$), and dividing this difference by the pooled standard deviation of both groups in the pretest (S_{Pre}):

$$d = c(m)\left[\frac{(\bar{y}^T_{\text{Pre}} - \bar{y}^T_{\text{Post}}) - (\bar{y}^C_{\text{Pre}} - \bar{y}^C_{\text{Post}})}{S_{\text{Pre}}}\right] \quad (1)$$

where $c(m) = 1 - 3/(4N-9)$ is a correction factor of the d index for small sample sizes, with N being the total sample size of the study ($N = n_T + n_C$).

In a single study, a d index was calculated for each of the different types of outcomes: obsessive–compulsive symptoms, anxiety, depression, functional impairment, and other outcome measures. Positive d values indicated a better result of the treatment group in comparison to the control group. When a study applied several measures of the same symptoms (e.g., two different tests on anxiety), a d index was calculated for each one of them, and then they were averaged in order to represent that study with only a d value for that type of outcome (anxiety in the example). We carried out separate meta-analyses for each type of outcome, and the individual studies had not necessarily to include measures of all of them. For example, there were studies that only reported measures on obsessive–compulsive symptoms, depression, and anxiety, but not on the other outcomes. In that case, these studies contributed only to the corresponding meta-analyses.

The assessment instruments most frequently found in the studies were: the Children' Yale-Brown Obsessive–Compulsive Scale (CY-BOCS; Scahill et al., 1997) and the Obsessive–Compulsive Subscale of National Institute of Mental Health Diagnostic Interview Schedule for Children (NIMH-OCS; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) for obsessive–compulsive symptoms; the Multidimensional Anxiety Scale for Children (MASC; March, 1995b) for anxiety; the Children's Depression Inventory (CDI; Kovacs, 1992) and the Hamilton Rating Scale for Depression (HAM-D; Hamilton, 1960) for depression; the Child Obsessive Compulsive Impact Scale-Child/Parent (COIS-C/P; Piacentini & Jaffer, 1999) for functional impairment, and the Clinical Global Impression-Severity/Improvement (CGI-S/I; Guy, 1976; National Institute of Mental Health, 1985) and the Global Assessment Functioning (GAF; Startup, Jackson, & Bendix, 2002) for other outcome measures not included in the previous categories.

In order to assess the reliability of the effect size calculations, the same random sample of studies used to assess the reliability of the coding process was subjected to a double process of effect size calculations. The inter-coder reliability was excellent, with a mean intra-class correlation of 0.87.

2.5. Statistical analysis

Separate meta-analyses were carried out with the effect sizes calculated for each outcome measure. In order to accommodate the variability exhibited by the effect sizes, mixed-effects models were assumed in the meta-analytic calculations. These models involve weighting each effect size by its inverse variance in order to give more weight to the effect sizes obtained from studies with large sample sizes (Borenstein, Hedges, Higgins, & Rothstein, 2009; Sánchez-Meca & Marín-Martínez, 2008). For each outcome measure, a weighted analysis of variance (ANOVA) was calculated in order to compare the mean effects of CBT, pharmacological, and combined treatments. Given that our meta-analysis only included one unpublished paper, several analyses of publication bias were carried out with the effect sizes obtained for the main outcome measure (obsessive–compulsive symptoms). In particular, the safe index and the Egger test were applied separately for CBT and for pharmacological treatments (Rothstein, Sutton, & Borenstein, 2005).

In order to examine the influence of moderator variables on the effect size variability, ANOVAs and meta-regressions were calculated for the qualitative and the continuous variables, respectively (Borenstein et al., 2009). In particular, to test the statistical significance of a moderator variable, Q_B and Q_R statistics were calculated for ANOVAs and meta-regressions, respectively. In addition, Q_W and Q_E statistics, respectively, were computed to assess the model misspecification. In addition, an estimate of the proportion of variance accounted for by the moderator variable was calculated by means of $R^2 = 1 - \hat{\tau}^2_{\text{Res}}/\hat{\tau}^2_{\text{Total}}$, with $\hat{\tau}^2_{\text{Total}}$ and $\hat{\tau}^2_{\text{Res}}$ being the total and residual heterogeneity variance estimates, respectively (López-López, Marín-Martínez, Sánchez-Meca, Van den Noortgate, &Viechtbauer, 2013). The moderator analyses were applied separately for the psychological and the pharmacological treatments, and only for the main outcome measure (obsessive–compulsive symptoms). Finally, an explanatory-predictive model was applied for all the studies in order to find effect size estimates adjusted by the methodological quality of the studies. The statistical analyses were carried out with the program *Comprehensive Meta-analysis 2.2* (Borenstein et al., 2010).

3. Results

3.1. Descriptive characteristics of the studies

Appendix A lists the main characteristics of the 24 studies, as well as the individual effect sizes for obsessive–compulsive symptoms. Out of the 24 comparisons between a treatment and a control group, 21 of them had participants randomly assigned to the groups, whereas one pharmacological study and two combined treatments did not apply a strictly random assignment (Franklin et al., 2011, but see Footnote 1). The mean age of the participants in the 11 CBT, 10 pharmacological and 3 combined treatments were 12.1, 12.7, and 12.6 years old, respectively. The percentage of males was 50.6%, 55.8%, and 46.8%, respectively. Only eight studies (three CBT and five pharmacological studies) reported the mean number of years that the participants in the samples were suffering OCD, with the mean for the pharmacological studies about twice that of the psychological ones (4.1 vs. 2.4 years, respectively). On average, CBT studies presented sample sizes that were clearly lower than those of the pharmacological ones. Thus, the mean sample sizes for the treatment groups in the posttest were 21 and 33, respectively, and for the control groups they were 17 and 32. With the exception of three studies (De Veaugh-Geiss et al., 1992; Flament et al., 1985; Himle et al., 2003), all of them reported data about comorbidity in the samples. Table 2 presents these data for each individual study. In general terms, the percentage of comorbidity present in the samples ranged from 96.7% (Storch et al., 2011) to 10.3% (Riddle et al., 2001). From the inspection of this table, the presence of a large heterogeneity among the studies in the mode of reporting these data

Table 2
Frequency of comorbidity across studies in the pretest.

Study	% of comorbidity in the pretest	% of mood disorders	% of other anxiety disorders (AD)	% of ADHD	% of tics disorder
Barrett et al. (2004) Study 1	2nd diagnosis: 77.1 3rd diagnosis: 57	3rd diagnosis: DD: 4	2nd diagnosis: SAD: 16.6; Specific: 12.5; Social: 8.3; GAD: 33 3rd diagnosis: Specific: 21; GAD: 25		
Barrett et al. (2004) Study 2	2nd diagnosis: 83 3rd diagnosis: 66	2nd diagnosis: Dysthymia: 3.4; MD: 3.4 3rd diagnosis: DD: 3.4; MD: 6.8	2nd diagnosis: SAD: 10.3; Social: 24.1; Specific: 13.8; GAD: 27.5		
Bolton and Perrin (2008)	50 (≥1 comorbid conditions)	MD: 10	AD: 50	10	10
Bolton et al., 2011 Study 1		9.4	AD: 69.4	8.3	2.1
Bolton et al. (2011) Study 2		9.4	AD: 66.7	8.3	2.1
De Veaugh-Geiss et al. (1992)	Not reported	0		0	
Flament et al. (1985)	Not reported				
Franklin et al. (2011) Study 1	Any: 64.3		47.6 (anxiety/mood mixed)	26.2	21.4
Franklin et al. (2011) Study 2	Any: 65		45 (anxiety/mood mixed)	22.5	20
Franklin et al. (2011) Study 3	Any: 50		40.5 (anxiety/mood mixed)	16.7	4.8
Freeman et al. (2008)	Internalizing disorders: 54.8 Externalizing disorders: 35.7			19	9.5
Geller et al. (2001)	Not reported				
Geller et al. (2004)	Any: 30.6		GAD: 6.9	9.4	
Himle (2003)	Not available				
Liebowitz et al. (2002)	46.5	23.8	AD: 52.38	14.3	
March et al. (1998)	2nd diagnosis: 74 3rd diagnosis: 16	2	AD: 2	7	5
Piacentini et al. (2011)	2nd diagnosis: 40.8 3rd: 22.4	6.1	53.1	14.3	12.2
POTS (2004)[a] Study 1	Internalizing diagnosis: 81.5 Externalizing diagnosis: 33.3	0			11.1
POTS (2004) Study 2	Internalizing diagnosis: 63 Externalizing diagnosis: 33.3	0			18.5
POTS (2004) Study 3	Internalizing diagnosis: 53.6 Externalizing diagnosis: 14.3	0			14.3
Riddle et al. (1992)	Any: 71	28	57		14
Riddle et al. (2001)	Any: 10.3				
Storch et al. (2011)	Any: 96.7				
Williams et al. (2010)	Any: 47%	DD: 4.7%	GAD:18; Social: 19 Specific: 19; SAD: 19; Social: 9.5	9.5	

MD: Major depression; DD: Dysthymic disorder; AD = Anxiety disorder; Social: social phobia; Specific: specific phobia; GAD: generalized anxiety disorder; SAD: Separation anxiety disorder; ADHD: attention-deficit/hyperactivity disorder.

Table 3
Results of the weighted ANOVAs to compare the efficacy of CBT, pharmacological and combined treatments in each outcome measure.

Outcome measure/treatment	k	d_+	95% I.C.		ANOVA Results
			d_l	d_u	
Obsessive–compulsive sympt.:					$Q_B(2) = 13.81$, $p = .0010$;
CBT	11	1.742	1.335	2.149	$R^2 = 0.340$
Pharmacological treatment	10	0.746	0.362	1.130	$Q_W(21) = 97.76$, $p < .0001$
Combined treatment	3	1.710	1.000	2.421	
Anxiety:					$Q_B(1) = 2.17$, $p = .141$;
CBT	6	0.592	0.314	0.869	$R^2 = 0.656$
Pharmacological treatment	3	0.228	−0.167	0.624	$Q_W(7) = 7.77$, $p = .376$
Depression:					$Q_B(1) = 0.50$, $p = .480$;
CBT	6	0.395	0.070	0.720	$R^2 = 0.045$
Pharmacological treatment	5	0.226	−0.111	0.546	$Q_W(9) = 14.05$, $p = .121$
Functional Impairment:					$Q_B(1) = 2.21$, $p = .137$;
CBT	4	0.835	0.488	1.183	$R^2 = 0.379$
Pharmacological treatment	2	0.370	−0.135	0.876	$Q_W(4) = 5.52$, $p = .238$
Other Result Measures:					$Q_B(1) = 4.13$, $p = .042$;
CBT	5	1.025	0.588	1.462	$R^2 = 0.418$
Pharmacological treatment	6	0.449	0.106	0.792	$Q_W(9) = 27.87$, $p = .001$
d index in the pretest:[a]					$Q_B(1) = 2.31$, $p = .314$;
CBT	11	−0.124	−0.329	0.081	$R^2 = 0.009$
Pharmacological treatment	10	0.077	−0.091	0.244	$Q_W(21) = 26.92$, $p = .173$
Combined treatment	3	−0.063	−0.390	0.265	

k: number of studies. d_+: mean effect size. d_l and d_u: lower and upper confidence limits for d_+. Q_B: between-categories Q statistic. Q_W: within-categories Q statistic. R^2: proportion of variance accounted for.

[a] The dependent variable was the d index in the pretest for obsessive–compulsive symptoms.

was evident. As a consequence, it was not possible to examine the relationship between comorbidity and effect size.

CBT and pharmacological studies exhibited several differences in methodological characteristics. Thus, the attrition rates in the posttest of the pharmacological studies were clearly higher than those for CBT, with mean values of 18.4% and 8.6%, respectively, for the treatment groups, and 21.3% and 9.8% for the control groups. The differential attrition between the treatment and control groups were very similar, however, in both groups of studies (3% and 1.2%, respectively). In addition, the mean score obtained in the methodological quality scale was better for the pharmacological studies (mean = 5.5) than for CBT studies (mean = 4.9).

3.2. Comparing CBT, pharmacological and combined treatment

In order to compare the differential efficacy of CBT, pharmacological, and combined treatments for pediatric OCD, weighted ANOVAs were applied for each outcome measure. Table 3 presents the results.

For the main outcome measure, obsessive–compulsive symptoms, all types of treatment obtained a statistically significant mean effect in favor of the treatment groups. Fig. 1 presents a forest plot of these effect sizes grouped by the type of treatment. According to Cohen's (1988) criteria, these were very large for CBT ($d_+ = 1.742$) and combined interventions ($d_+ = 1.710$), and moderate-to-large for pharmacological treatments ($d_+ = 0.746$). The differences among the three mean effects in reducing obsessive–compulsive responses were statistically significant ($p = .001$), with a large proportion of variance accounted for of 34%.

With regards to secondary OCD-related measures, these were analyzed only for CBT and pharmacological treatments, because no studies reported data for these outcome measures with the combined treatment. As Table 2 shows, both CBT and pharmacological interventions improved other secondary responses related to OCD, although these results reflected lower mean effect sizes than for obsessive–compulsive measures. For anxiety, depression, and functional impairment, the mean effect for CBT was statistically significant, whereas for pharmacological treatments it did not reach statistical significance. The difference between the two mean effects was not statistically significant for any of these outcome measures, although for anxiety and functional impairment measures the proportion of variance accounted for was very large (0.656 and 0.379, respectively). The small number of studies probably led to a nonstatistically significant difference between CBT and pharmacological treatment. The largest effects were found in the category '*other result measures*', in CBT ($d_+ = 1.025$) and in pharmacological treatments ($d_+ = 0.449$), and both of these reached statistical significance. In addition, these two mean effects also exhibited a statistically significant difference in favor of CBT.

3.3. Analysis of publication bias

Since all the studies included in the meta-analysis were published papers, we carried out a study of publication bias. In order to do this, we focused on the effect sizes calculated for the obsessive–compulsive measures. To address this issue, we computed the safe number for null results, which involves calculating the number of unpublished studies averaging a null effect that must exist in order for the mean effect obtained in a meta-analysis to become zero (Becker, 2005). If this number is larger than five times the number of studies, k, plus 10, then publication bias can be discarded as a threat for the validity of the meta-analytic results. In our meta-analysis, the safe number for CBT studies was $5k + 10 = 5(11) + 10 = 65$ and the safe number for null results was equal to 181. This means that there would have to exist 181 unpublished papers with null effects to reduce the mean effect of CBT to zero. The safe number for pharmacological studies was 65, also larger than the safe number ($N_{fs} = 60$). As the number of null results was clearly larger than the safe number, we could conclude that, on a reasonable basis, publication bias does not seem to be a threat for the mean effect of CBT nor for pharmacological treatments.

To complement these results, Egger tests were also computed. The Egger test consists of constructing an unweighted simple regression, with the effect size as the dependent variable and the standard error of each effect size as the independent one. A nonstatistically significant result of the t-test for the hypothesis of an intercept equal to zero would enable us to discard publication bias as a threat to the validity of the results. Both the effect sizes obtained for CBT and those obtained with the pharmacological treatments exhibited a non-statistically significant intercept (CBT: $p = .134$;

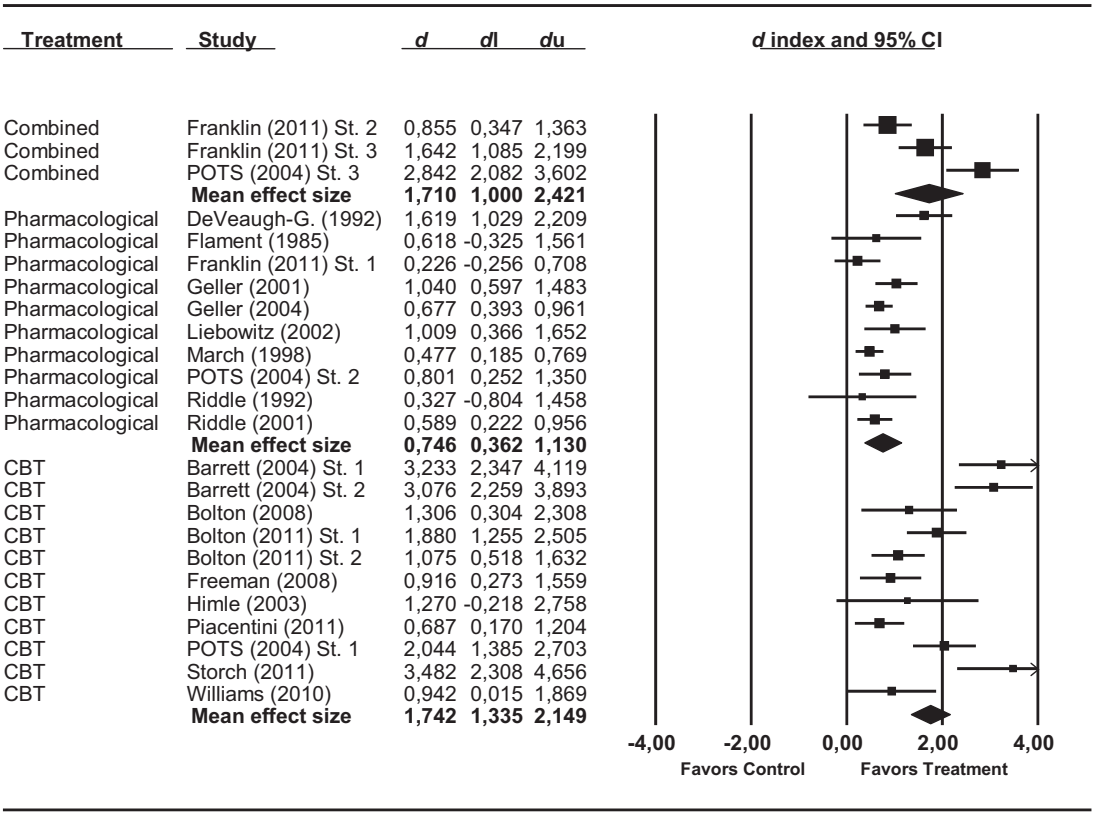

Fig. 1. Forest plot of the effect sizes for obsessive–compulsive symptoms classified as a function of the type of treatment. *d*: standardized mean change index. *d*l and *d*u: 95% lower and upper confidence limits around *d*.

pharmacological treatments: $p = .447$). Therefore, the results of the different analyses for examining publication bias led us to discard this threat for our meta-analytic results.

3.4. Analysis of moderator variables

The results presented in Table 3 on the efficacy of CBT and pharmacological treatments in reducing obsessive–compulsive symptoms evidenced the existence of a great heterogeneity, according to the Q_w test ($p < .0001$). As a consequence, we examined the influence of several characteristics related to the interventions, the methodology and the participants in the studies. These analyses were conducted separately for CBT and pharmacological studies, and in both cases the dependent variable was the *d* index for obsessive–compulsive symptoms.

3.4.1. Cognitive-behavioral therapy

Tables 4 and 5 present weighted ANOVAs and simple meta-regressions for the analysis of qualitative and continuous moderator variables, respectively. Out of the *treatment characteristics* analyzed, the only qualitative variable that showed a significant association with the effect size was the protocol treatment used in the study ($p < .001$), with a 74% of variance accounted for. In particular, five different standardized CBT programs were administered in the nine studies included in this ANOVA. The most effective programs were those developed by Barrett (2009; $d_+ = 3.151$), March and Mulle (1998; $d_+ = 2.553$), and Salkovskis (1999; $d_+ = 1.330$). All of them exhibited large and significant effects. The protocols developed by Choate-Summers et al. (2006; $d_+ = 0.916$) and Piacentini, Langley, and Roblek (2007; $d_+ = 0.687$) reached smaller effects than the previous ones, and none of them reached statistical significance. No relationship with effect size was found for the inclusion of ERP, parental involvement, the focus of the treatment, or the mode of training. However, as can be seen in Table 4, large differences among the mean effects were found in some of them.

With regard to the continuous variables (Table 5), the magnitude of the intervention presented a statistical association with the effect size ($p < .05$) and with 44.7% of variance accounted for. In particular, interventions with a greater number of hours were associated with the largest effect sizes. No significant relationships were found between the effect size and the number of weeks of intervention or the hours per week administered.

The only qualitative variable related to participant characteristics was whether the participants had received any previous treatment (see Table 4), and it did not reach a statistical association with the effect size ($p = .659$). Similarly, the simple meta-regressions conducted on the quantitative variables mean age and gender distribution of the sample also evidenced the absence of a significant association with the effect sizes (see Table 5).

The third cluster of moderator variables was that of the methodological characteristics. As Table 4 shows, the type of control group used to compare the results obtained with the treatment group exhibited a non-statistically significant relationship with the effect sizes ($p = .125$), but the mean effect size for the inactive controls was clearly larger ($d_+ = 2.099$) than that of the active controls ($d_+ = 1.216$). The scarce number of studies led to a non-statistically significant result. Three continuous moderator variables related to the methodological characteristics were analyzed: the differential attrition between the treatment and control groups, the methodological quality of the study, and the *d* index in the pretest. As Table 5 shows, none of them reached statistical significance.

3.4.2. Pharmacological treatments

Tables 4 and 5 present the weighted ANOVAs and meta-regressions of the qualitative and continuous moderator variables,

Table 4
Results of the weighted ANOVAs for the influence of qualitative moderator variables on the effect sizes for obsessive–compulsive symptoms in CBT and pharmacological studies.

Moderator variable	*k*	d_+	95% C.I.		ANOVA results
			d_l	d_u	
(A) CBT studies:					
ERP:					
Yes	7	2.046	1.289	2.803	$Q_B(1) = 1.122$, $p = .289$; $R^2 = 0.0$
No	3	1.311	0.180	2.441	$Q_W(9) = 57.679$, $p < .001$
Parental involvement:					
Low	4	1.308	0.249	2.368	$Q_B(2) = 1.531$, $p = .465$; $R^2 = 0.0$
Moderate	1	2.044	−0.030	4.119	$Q_W(7) = 55.989$, $p < .001$
High	5	2.195	1.241	3.148	
Focus of treatment:					
OCD Child	5	1.462	0.567	2.357	$Q_B(1) = 1.241$, $p = .265$; $R^2 = 0.0$
Family	5	2.185	1.280	3.090	$Q_W(8) = 59.370$, $p < .001$
Treatment protocol:					
March and Mulle (1998)	2	2.553	1.691	3.416	$Q_B(4) = 22.201$, $p < .001$ $R^2 = 0.74$
Barrett (2009)	2	3.151	2.301	4.001	$Q_W(4) = 8.95$, $p < .001$
Salkovskis (1999)	3	1.330	0.698	1.962	
Piacentini et al. (2007)	1	0.687	−0.307	1.682	
Choate-Summers et al. (2006)	1	0.916	−0.149	1.981	
Mode of training:					
Group	2	2.350	0.975	3.726	$Q_B(1) = 0.797$, $p = .372$; $R^2 = 0.10$
Individual	9	1.669	1.082	2.256	$Q_W(9) = 50.313$, $p < .001$
Previous treatments					
Yes	7	2.041	0.870	3.211	$Q_B(1) = 0.194$, $p = .659$; $R^2 = 0.0$
No	3	1.726	0.962	2.490	$Q_W(8) = 59.636$, $p < .001$
Type of control group:					
Inactive	7	2.099	1.415	2.783	$Q_B(1) = 2.350$, $p < .125$; $R^2 = 0.09$
Active	4	1.216	0.318	2.114	$Q_W(9) = 48.214$, $p < .001$
(B) Pharmacological studies:					
Type of antidepressant:					
SSRI	8	0.644	0.449	0.840	$Q_B(1) = 4.775$, $p < .05$; $R^2 = 0.554$
Clomipramine	2	1.305	0.746	1.863	$Q_W(8) = 12.300$, $p < .138$
Previous treatments:					
Yes	4	0.655	0.451	0.860	$Q_B(1) = 2.065$, $p = .151$; $R^2 = 0.99$
No	2	0.307	−0.121	0.736	$Q_W(4) = 1.267$, $p < .866$

k: number of studies. d_+: mean effect size. d_l and d_u: lower and upper confidence limits around d_+. Q_B: between-categories *Q* statistic. Q_W: within-categories *Q* statistic. R^2: proportion of variance accounted for.

respectively, for the effect sizes obtained from pharmacological treatments. One of the most important variables analyzed was the type of antidepressant used in the study (all used antidepressants). As Table 4 shows, clomipramine ($d_+ = 1.305$) obtained a higher mean effect size than SSRIs ($d_+ = 0.644$), and this difference was statistically significant ($p < .05$), with a 55.4% of variance accounted for. However, it is worth noting that the two studies that applied clomipramine (De Veaugh-Geiss et al., 1992; Flament et al., 1985) reported side effects in more than 30% of the participants (dry mouth, fatigue, dizziness, sweating, sleepiness), with a mean number of side effects of 5.5 (SD = 2.1), whereas the SSRI studies clearly exhibited a smaller number of side effects (mean = 2.3; SD = 3.0).

Table 5
Results of the simple meta-regressions on the effect sizes for obsessive–compulsive symptoms in CBT and pharmacological treatments.

Moderator variable	*k*	b_j	Q_R	Q_E	R^2
(A) CBT studies:					
Duration (no. of weeks)	11	0.101	0.635	59.508***	0.062
Intensity (no. hours per week)	10	0.859	1.753	50.317***	0.160
Magnitude (total no. of hours)	10	0.147	5.293*	45.676***	0.447
Mean age (in years)	10	−0.022	0.019	59.722***	0.002
Gender (% of males)	10	0.027	0.868	49.145***	0.076
Differential attrition	11	0.223	0.004	59.983***	< .001
Methodological quality scale (0–6)	10	−0.074	0.025	56.249***	0.003
d index in the pretest	11	−0.882	1.218	57.279***	0.114
(B) Pharmacological studies:					
Initial dose (mg/day)	8	−0.009	0.474	13.489*	0.070
Maintenance dose (mg/day)	9	−0.002	1.207	12.703	0.143
Duration (no. of weeks)	10	0.004	0.008	19.311*	0.001
Mean age (in years)	10	0.037	0.093	19.320*	0.010
Gender (% of males)	9	0.023	1.728	14.046*	0.190
Differential attrition	10	0.327	0.069	18.924*	0.008
Methodological quality scale (0–6)	10	0.035	0.014	19.321*	0.001
d index in the pretest	10	1.048	5.125*	11.559	0.590

k: number of studies. b_j: unstandardized regression coefficient. Q_R: *Q* statistic for testing the statistical significance of the moderator variable. Q_E: *Q* statistics for testing the model misspecification. R^2: proportion of variance accounted for by the moderator variable.
* $p < .05$.
*** $p < .001$.

Other characteristics analyzed in relation with the pharmacological interventions were the initial dose, the maintenance dose, and the duration of the drug administration, none of them reaching statistical significance (Table 5). None of the moderator variables related to the participants in the samples showed a statistical relationship with the effect sizes: whether the subject received previous treatment (Table 4), the mean age, or the gender distribution (Table 5). Finally, the only methodological variable that showed a statistically significant relationship with the effect size was the *d* index in the pretest. The positive slope found in this relationship indicated that the largest effect sizes were associated to the largest *d* indices in the pretest; that is, the better the participants in the treatment group with respect to the control group in the pretest, the better the benefits of the treatment group were in comparison with those of the control group (Table 5). It was not possible to analyze the influence of the type of control group because all pharmacological studies applied a placebo pill.

3.5. Predictive model

As shown in Section 3.1, differences were found in the control of methodological variables between CBT and pharmacological studies. Out of the different method characteristics assessed, CBT and pharmacological studies showed clear differences in the type of control group applied (active vs. inactive). In particular, the 10 pharmacological studies used an active control group (placebo pill), whereas seven out of the 11 CBT studies used inactive control groups. Consequently, the mean effect sizes reported in Table 1 for the two groups of studies may have been affected by differences in the methodological quality of the studies. With the purpose of obtaining a more precise estimate of the differential efficacy between CBT and pharmacological treatments, a mixed-effects multiple meta-regression was applied that included three predictor variables: the type of control group (0, inactive; 1, active), whether the study included CBT (0, no; 1, yes), and whether the study included pharmacological treatment (0, no; 1, yes). The dependent variable was the effect size for obsessive–compulsive symptoms. The results are presented in Table 6. The full model reached a statistically significant association with the effect sizes ($p = .0002$), with 41.1% of variance accounted for. Once controlled for the type of treatment applied, the control group type exhibited a statistically significant relationship with the effect sizes ($p = .037$). The inclusion of CBT also showed a statistically significant association with the effect sizes ($p = .015$), but the inclusion of pharmacological treatment did not reach statistical significance ($p = .292$). The meta-regression model allowed us to make predictions of the expected effect for CBT, pharmacological, and combined treatment once controlled for the type of control group. Thus, assuming an active control group, the expected effects for CBT, pharmacological, and combined treatments were 1.203, 0.745, and 1.704, respectively. These predictions were more precise than those presented in Table 3 in assessing the differential efficacy of the treatments. Finally, as the Q_E statistic reached statistical significance, the model was mis-specified. This means that there were other relevant moderator variables not included in the model.

4. Discussion

The main purpose of this meta-analysis was to examine the differential efficacy of CBT, pharmacological and combined treatments for OCD in children and adolescents. The results supported the efficacy of the three types of intervention in reducing obsessive–compulsive symptoms in this population, since the three mean effect sizes reached statistical significance. Nevertheless, CBT ($d = 1.742$) and combined treatments ($d = 1.710$) exhibited a significantly higher effect size than pharmacological treatments ($d = 0.746$). These results were, at first, similar to those shown in previous meta-analyses. Abramowitz et al. (2005) also showed this superiority of CBT ($d = 1.98$) on medication ($d = 1.13$), with their estimations being slightly higher than ours. The effect size index used in Abramowitz et al. (2005) was the standardized mean change between the pretest and posttest means and, in consequence, it had a lower internal validity than that applied in our meta-analysis. A more accurate estimate of the treatment effects in Abramowitz et al. (2005) could be obtained by subtracting the mean effect size for the placebo groups ($d = 0.48$) from the CBT mean effect ($d_{adj} = 1.98–0.48 = 1.50$) and medication mean effect ($d_{adj} = 1.13–0.48 = 0.65$). A better result for CBT than for pharmacological treatments was also found by Watson and Rees (2008), in this case with lower effect estimates than ours (CBT: $d = 1.45$; Medication: $d = 0.48$). For pharmacological treatments, Geller, Biederman, Stewart, Mullin, Farrell, et al. (2003) and Geller, Biederman, Stewart, Mullin, Martin, et al. (2003) reported a mean effect ($d = 0.45$) very similar to that of Watson and Rees (2008) and slightly lower than the adjusted mean effect obtained by Abramowitz et al. (2005) and the one obtained in our study.

The second goal was to analyze the effect of the interventions on OCD-related secondary responses (anxiety, depression, functional impairment and other result measures). Although only the differences between CBT and pharmacological treatment were significant for *other outcome measures*, the mean effect sizes in all secondary measures were higher for CBT, ranging from moderate to large and all statistically significant. On the contrary, the mean effect sizes for pharmacological treatments in these measures ranged from poor to moderate and were not statistically significant for anxiety, depression, and functional impairment. It is possible to affirm that our results were more promising for OCD-related problems than those reported by Abramowitz et al. (2005), who did not find significant effects for either ERP or SSRI in reducing anxious-depressive symptoms.

The third goal was focused on exploring the potential influence of several moderator variables on the effect size obtained for obsessive–compulsive symptoms. Regarding CBT, the treatment protocol significantly influenced the effect size reached by the studies. The most efficacious protocol was that implemented by Barrett et al. (2004) which is a family-focused CBT whose main components are ERP and high parental involvement. Although these characteristics did not show a significant influence on the effect sizes separately, larger mean effect sizes were found for studies with moderate and high parental involvement ($d = 2.195$ and 2.044, respectively) than for those with low parental involvement ($d = 1.308$). Similarly, studies that included ERP reached greater effect sizes ($d = 2.046$) than studies that did not include this component ($d = 1.311$). These results must be interpreted with caution due to the reduced number of studies examining this protocol. Probably, other factors could be affecting the protocol efficacy, but our meta-analysis was not able to identify them. With respect to the previous findings in this sense, Freeman et al. (2007) reported a superior efficacy for family-based treatment but they did not statistically analyze this difference. Barrett et al. (2008) also emphasized the role that family plays in the treatment of children and adolescents. Another variable related to the efficacy of CBT was the total intervention hours. Interventions with a greater magnitude were more efficacious. This finding has not been reported previously. On the other hand, neither participant nor methodological variables were associated with the efficacy of CBT.

The analysis of moderator variables for pharmacological studies evidenced a significant higher efficacy for clomipramine in comparison with SSRIs in reducing obsessive–compulsive symptoms. This result confirmed the findings of Geller, Biederman, Stewart, Mullin, Farrell, et al. (2003) and Geller, Biederman, Stewart, Mullin,

Table 6
Results of the mixed-effects multiple meta-regression model on effect sizes for obsessive–compulsive symptoms.

Predictor variable	b_j	*SE*	*Z*	*p*
Intercept	1.103	0.469	2.352	.019
CBT	0.959	0.395	2.429	.015
Pharmacological treatment	0.501	0.476	1.054	.292
Control group	−0.859	0.312	−2.084	.037
Analysis of full model:	$Q_R(3) = 19.196$, $p = .0002$; $R^2 = 0.411$ $Q_E(20) = 85.942$, $p < .0005$			

b_j: unstandardized regression coefficient. *SE*: standard error of b_j. *Z*: statistic for testing the significance of each predictor variable. *p*: probability level. Q_R: statistic for testing the significance of the model. Q_E: statistic for testing the model misspecification. R^2: proportion of variance accounted for.

Martin, et al. (2003) and Watson and Rees (2008). On the contrary, Abramowitz et al. (2005) did not find significant differences between the two types of drugs. Due to the large side effects of clomipramine, SSRIs are the first choice in clinical practice since they are better tolerated (AACAP Committee on Quality Issues, 2012). The *d* index in the pretest showed a positive, statistically significant relationship with the effect sizes for obsessive–compulsive symptoms, meaning that larger benefits were found in the pharmacological treatments when the participants in the treatment groups were better than those in the control groups in the pretest.

The majority of the CBT studies used an inactive control group in their comparisons, whereas all of the pharmacological studies included placebo control groups. As a consequence, the better mean effect size obtained by CBT in comparison to that of pharmacological treatment could be confounded by the type of control group. Thus, a meta-regression model that included the type of treatment (CBT vs. pharmacological treatment) and the type of control group as predictors, allowed us to predict the expected effect for CBT, pharmacological, and combined treatment once the type of control group was controlled. Thus, assuming an active control group, the expected effects for CBT experienced a reduction from 1.742 to1.203, whereas the expected effect sizes for pharmacological (from 0.746 to 0.745) and combined treatment (from 1.710 to1.704) were very similar to the corresponding unadjusted mean effect sizes. When the type of control group was controlled, CBT still exhibited a larger effect size than pharmacological treatments. This result coincided with those found in individual empirical studies on combined treatment (Franklin et al., 2011; POTS, 2004) and in some qualitative reviews (AACAP Committee on Quality Issues, 2012; Barrett et al., 2008; Rosa-Alcázar et al., 2012).

4.1. Implications for clinical practice

One of the clearest implications of our results for the clinical practice is the efficacy found for the three types of intervention (CBT, pharmacological treatments and combined treatment) in reducing obsessive–compulsive symptoms of pediatric OCD. Despite the scarce number of studies conducted, we can conclude that combined treatments exhibited the best results. This is just in agreement with the guidelines recommended by the American Academy of Children and Adolescent Psychiatry (AACAP) for treating severe cases of pediatric OCD AACAP Committee on Quality Issues (2012). In particular, cases with high comorbidity and a score over 23 in the CY-BOCS, should be treated by combining SSRI and CBT. It is also important to note that CBT produced significant reductions in other symptoms such as anxiety, depression, or functional impairment, among others.

Concerning the specific techniques used in CBT, our findings suggest that the most promising treatments are those based on ERP and a high parental involvement in the treatment. With this last component, the therapist will be able of providing the child and family with coping techniques for managing future problems. Parents can actively participate, on the one hand, in the assessment and reporting of more objective data and information to the therapist and, on the other hand, in the treatment implementation, reducing accommodation to symptoms, applying exposure at home, and modifying beliefs and attitudes toward OCD (AACAP Committee on Quality Issues, 2012; Barrett et al., 2008).

Regarding pharmacological treatments, it is important to note that despite two studies that evidenced higher efficacy for clomipramine in comparison with SSRIs in reducing obsessive–compulsive symptoms, our conclusions concur with the guidelines proposed by the AACAP Committee on Quality Issues (2012) of recommending SSRIs instead of clomipramine due to their minor adverse events.

4.2. Limitations and implications for future research

One of the main limitations in the current meta-analysis was the scarce number of studies that complied with our selection criteria due to the low number of experimental designs in the field of pediatric OCD that included a control group. Nevertheless, we decided to maintain our restrictive selection criteria in favor of a larger internal validity of the effect size index. Another limitation derived from the selection criteria was that we could not analyze the long-term efficacy, since the participants in control groups were treated after the posttest assessment. Consequently, the conclusions reached in the current research are limited to short-term efficacy. The lack of information in the studies on variables related to participants or application context prevented us from being able to conduct some relevant analyses. One of the greatest difficulties was the analysis of comorbidity, since the studies did not use the same criteria to report it. Other limitations in the current meta-analysis were the lack of information about the specific components of CBT, the absence of psychological placebo control groups and the heterogeneity of the samples, aspects already pointed by other authors (Barrett et al., 2008; Futh, Simonds, & Micali, 2012).

On the other hand, there are several aspects related to the treatments that are worth investigating in greater depth. Firstly, the combined treatment should receive more experimental research for this range of age, if we want to definitively establish its good performance. Secondly, the majority of SSRIs have been investigated in only one or two experimental studies, which is not enough to conclude their efficacy or safety in children and adolescents. In addition, to date some SSRIs, such as citalopram, have not been tested in RCTs. Thirdly, the study of the differential efficacy of psychological treatment techniques through the use of dismantling strategies is an important unsolved issue in this field (Sukhodolsky et al., 2013). Finally, it is also relevant to develop strategies for treating nonresponders, taking into account such factors as comorbidity, family functioning, gender, and age of participants (Barrett et al., 2008; Futh et al., 2012; Storch et al., 2011).

Appendix A. Some of the characteristics of the studies included in the meta-analysis

Study	*N*	Mean age	% of males	Type of treatment	Intensity	Duration	Parent involvement	Control group	*d*
Barrett et al. (2004) Study 1	48	11.25	52.10	FCBT	1.50	14.00	High	WL	3.233
Barrett et al. (2004) Study 2	53	12.38	47.20	Group-FCBT	1.50	14.00	High	WL	3.076
Bolton and Perrin (2008)	20	13.20	70.00	ERP	2.19	5.03	Low	WL	1.306
Bolton et al. (2011) Study 1	60	14.60	44.00	Full-CT	1.17	12.00	Low	WL	1.880
Bolton et al. (2011) Study 2	60	14.20	40.00	Brief-CT	0.42	12.00	Low	WL	1.075
De Veaugh-Geiss et al. (1992)	60	14.25	65.00	Clomipramine	N/A	8.00	N/A	P	1.619
Flament et al. (1985)	19	14.50	73.68	Clomipramine	N/A	5.00	N/A	P	0.618
Franklin et al. (2011) Study 1	70	13.52	48.60	Several SSRIs	N/A	12.00	N/A	P	0.226
Franklin et al. (2011) Study 2	68	13.16	48.60	Several SSRIs + CBT	0.38	12.00	Low	P	0.855
Franklin et al. (2011) Study 3	70	12.54	47.20	Several SSRIs + Brief-CBT	1.04	12.00	Low	P	1.642
Freeman et al. (2008)	42	7.11	43.00	FCBT	0.84	12.67	High	RT	0.916
Geller et al. (2001)	120	11.40	47.57	SSRI (Fluoxetine)	N/A	13.00	N/A	P	1.040
Geller et al. (2004)	203	11.30	57.60	SSRI (Fluoxetine)	N/A	8.57	N/A	P	0.677
Himle (2003)	10	N/A	N/A	Group-CBT	N/A	10.00	N/A	RT	1.270
Liebowitz et al. (2002)	42	12.65	58.14	SSRI (Fluoxetine)	N/A	16.00	N/A	P	1.009
March et al. (1998)	187	12.60	N/A	SSRI (Sertraline)	N/A	10.68	N/A	P	0.477
Piacentini et al. (2011)	71	12.20	36.60	FCBT	1.28	14.00	High	RT	0.687
POTS (2004)[a] Study 1	56	11.85	50.00	CBT	1.00	12.00	Moderate	P	2.044
POTS (2004) Study 2	56	12.00	55.30	SSRI (Sertraline)	N/A	12.00	N/A	P	0.801
POTS (2004) Study 3	56	12.00	44.60	CBT + SSRI (Sertraline)	1.17	12.00	Moderate	P	2.842
Riddle et al. (1992)	15	11.80	42.86	SSRI (Fluoxetine)	N/A	8.00	N/A	P	0.327
Riddle et al. (2001)	120	13.03	53.40	SSRI (Fluvoxamine)	N/A	10.00	N/A	P	0.589
Storch et al. (2011)	31	11.10	61.30	Web-CBT	1.45	12.00	High	WL	3.482
Williams et al. (2010)	21	13.60	61.90	CT	0.83	12.00	Moderate	WL	0.942

N: total sample size in the posttest (treated + control groups). FCBT: Family-Based Cognitive-Behavioral Treatment. ERP: Exposure with Response Prevention. CT: Cognitive Therapy. SSRI: Selective Serotonin Reuptake Inhibitor. Intensity: hours per week of treatment. Duration: number of weeks of treatment. WL: Waiting-List. P: Placebo. RT: Relaxation Training. *d*: standardized mean difference between the change scores of the treatment and the control groups for obsessive–compulsive symptoms. N/A: Not Available.

[a] POTS (2004) is referenced in Reference section as Pediatric OCD Treatment Study (POTS) Team (2004).

References[3]

American Academy of Child and Adolescent Psychiatry Committee on Quality Issues. (2012). Practice parameter for the assessment and treatment of children and adolescents with obsessive–compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*, 98–113.

Abramowitz, J. S., Whiteside, S. P., & Deacon, B. J. (2005). The effectiveness of treatment for pediatric obsessive–compulsive disorder: a meta-analysis. *Behavior Therapy*, *36*, 55–63.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: APA.

Apter, A., Fallon, T. J., King, R. A., Ratzoni, G., Zohar, A. H., Binder, M., et al. (1996). Obsessive–compulsive characteristics: from symptoms to syndrome. *Journal of American Academic of Child and Adolescent Psychiatry*, *35*, 907–912.

Barrett, P. M. (2009). FOCUS for life: freedom from obsessions and compulsions using skills. In *Therapist manual and workbooks*. Brisbane, QLD: Pathways Health and Research Centre.

Barrett, P. M., Farrell, L., Pina, A. A., Peris, T. S., & Piacentini, J. (2008). Evidence-based psychosocial treatments for child and adolescent obsessive–compulsive disorder. *Journal of Clinical Child and Adolescent Psychology*, *37*, 131–155.

*Barrett, P. M., Healy-Farrell, L., & March, J. S. (2004). Cognitive-behavioral family treatment of childhood obsessive compulsive disorder: a controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*, 46–62.

Becker, B. J. (2005). Failsafe *N* or file-drawer number. In: H. R. Rothstein, A. J. Sutton, & M. Borenstein (Eds.), *Publication bias in meta-analysis: prevention, assessment and adjustments* (pp. 111–125). Chichester, UK: Wiley.

Berg, C. J., Rapoport, J. L., & Flament, M. (1986). The Leyton Obsessional Inventory-Child Version. *Journal of American Academy of Child and Adolescent Psychiatry*, *25*, 84–91.

Bögels, S., & Phares, V. (2008). Fathers' role in the etiology, prevention and treatment of child anxiety: a review and new model. *Clinical Psychology Review*, *28*, 539–558.

*Bolton, D., & Perrin, S. (2008). Evaluation of exposure with response-prevention for obsessive compulsive disorder in childhood and adolescence. *Journal of Behavior Therapy and Experimental Psychiatry*, *39*, 11–22.

*Bolton, D., Williams, T., Perrin, S., Atkinson, L., Gallop, C., Waite, P., et al. (2011). Randomized controlled trial of full and brief cognitive-behaviour therapy and wait-list for pediatric obsessive–compulsive disorder. *Journal of Child Psychology and Psychiatry*, *52*, 1269–1278.

Borenstein, M. J., Hedges, L. V., Higgins, J., & Rothstein, H. (2009). *Introduction to meta-analysis*. Chichester, UK: Wiley.

Borenstein, M. J., Hedges, L. V., Higgins, J., & Rothstein, H. (2010). *Comprehensive meta-analysis (Vers. 2.2)*. Englewood, NJ: Biostat.

Canals, J., Hernández-Martínez, C., Cosi, S., & Voltas, N. (2012). The epidemiology of obsessive–compulsive disorder in Spanish school children. *Journal of Anxiety Disorders*, *26*, 746–752.

Choate-Summers, M. L., Freeman, J. B., Garcia, A. M., Coyne, L., Przeworski, A., & Leonard, H. L. (2006). Clinical considerations when tailoring cognitive behavioral treatment for young children with obsessive compulsive disorder. *Education and Treatment of Children*, *31*, 395–416.

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

*De Veaugh-Geiss, J., Moroz, G., Biederman, J., Cantwell, D., Fontaine, R., Greist, J. H., et al. (1992). Clomipramine hydrochloride in childhood obsessive–compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 45–49.

Farrell, L. J., Schlup, B., & Boschen, M. J. (2010). Cognitive–behavioral treatment of childhood obsessive–compulsive disorder in community-based clinical practice: clinical significance and benchmarking against efficacy. *Behavior Research and Therapy*, *48*, 409–417.

*Flament, M. F., Rapoport, J. L., & Kilts, C. (1985). A controlled trial of clomipramine in childhood obsessive compulsive disorder. *Psychopharmacology Bulletin*, *21*, 150–152.

Flament, M. F., Rapoport, J. L., Murphy, D. L., Berg, C. J., & Lake, C. R. (1987). Biochemical changes during clomipramine treatment of childhood obsessive compulsive disorder. *Archives of General Psychiatry*, *44*, 219–225.

Franklin, M. E., Kozak, M. J., Cashman, L., Coles, M., Rheingold, A., & Foa, E. B. (1998). Cognitive-behavioral treatment of pediatric obsessive–compulsive disorder: an open clinical trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*, 412–419.

*Franklin, M. E., Sapyta, J., Freeman, J. B., Khanna, M., Compton, S., Almirall, D., et al. (2011). Cognitive behavior therapy augmentation of pharmacotherapy in pediatric obsessive–compulsive disorder: the pediatric OCD treatment study II (POTSII) randomized controlled trial. *Journal of the American Medical Association*, *306*, 1224–1232.

*Freeman, J. B., Garcia, A., Coyne, M., Ale, L., Przeworski, A. Ch., Himle, M., et al. (2008). Early childhood OCD: preliminary findings from a family-based cognitive-behavioral approach. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*, 593–602.

Freeman, J. B., Choate-Summers, M. L., Moore, P. S., Garcia, A. M., Sapyta, J. J., Leonard, H. L., et al. (2007). Cognitive behavioral treatment for young children with obsessive–compulsive disorder. *Biological Psychiatry*, *61*, 337–343.

Futh, A., Simonds, L. M., & Micali, N. (2012). Obsessive–compulsive disorder in children and adolescents: parental understanding, accommodation, coping and distress. *Journal of Anxiety Disorders*, *26*, 624–632.

Geller, D. A., Biederman, J., Stewart, S. E., Mullin, B., Farrell, C., Wagner, K. D., et al. (2003). Impact of comorbidity on treatment response to paroxetine in pediatric obsessive–compulsive disorder: is the use of exclusion criteria empirically supported in randomized clinical trials? *Journal of Child and Adolescent Psychopharmacology*, *13*, S19–S29.

[3] References marked with an asterisk were included in the meta-analysis.

Geller, D. A., Biederman, J., Stewart, S. E., Mullin, B., Martin, A., Spencer, T., et al. (2003). Which SSRI? A meta-analysis of pharmacotherapy trials in pediatric obsessive–compulsive disorder. *American Journal of Psychiatry*, *160*, 1919–1928.

*Geller, D. A., Hoog, S., Heiligenstein, J., Ricardi, R., Tamura, R., Kluszynski, S., et al., & Fluoxetine Pediatric OCD Study Team. (2001). Fluoxetine treatment for obsessive–compulsive disorder in children and adolescents: a placebo control trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 773–779.

*Geller, D. A., Wagner, K., Emslie, G., Murphy, T., Carpenter, D. J., Wetherhold, E., et al. (2004). Paroxetine treatment y children and adolescents with obsessive–compulsive disorder: a randomized, multicenter, double-blind, placebo-controlled trial. *Journal of American Academy of Child and Adolescent Psychiatry*, *43*, 1387–1396.

Guy, W. (1976). *Assessment manual for psychopharmacology*. Washington, DC: US Government Printing Office (DHEW Publication No. ABM 76-366; revised).

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, *23*, 56–61.

*Himle, J. A. (2003). Group cognitive behavioral therapy versus group anxiety management for adolescent OCD: a randomized, controlled pilot study. In *Poster presented at the National Conference of the Anxiety Disorders Association of America* Toronto, Canada.

Himle, J. A., Van Etten, M., & Fisher, D. J. (2003). Group cognitive behavioral therapy for obsessive compulsive disorder: a review. *Brief Treatment and Crisis Intervention*, *3*, 217–229.

Kovacs, M. K. (1992). *Child depression inventory*. New York: Multi-Health Systems.

Krebs, G., & Heyman, I. (2010). Treatment-resistant obsessive–compulsive disorder in young people: assessment and treatment strategies. *Child and Adolescent Mental Health*, *15*, 2–11.

*Liebowitz, M., Turner, S., Piacentini, J., Beidel, D., Clarvit, S., Davies, S. O., et al. (2002). Fluoxetine in children and adolescents with OCD: a placebo controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 1431–1438.

López-López, J. A., Marín-Martínez, F., Sánchez-Meca, J., Van den Noortgate, W., & Viechtbauer, W. (2013). Estimation of the predictive power of the model in mixed-effects meta-regression: a simulation study. *British Journal of Mathematical and Statistical Psychology* (in press)

McLeod, B. D., Wood, J. J., & Weisz, J. R. (2007). Examining the association between parenting and childhood anxiety: a meta-analysis. *Clinical Psychology Review*, *27*, 155–172.

March, J. S. (1995a). Cognitive-behavioral psychotherapy for children and adolescents with obsessive–compulsive disorder: a review and recommendations for treatment. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 17–18.

March, J. S. (1995b). *Manual for the Multidimensional Anxiety Scale for Children (MASC)*. Toronto: Multihealth Systems.

*March, J. S., Biederman, J., Wolkow, R., Safferman, A., Mardekian, J., Cook, E. H., et al. (1998). Sertraline in children and adolescents with obsessive–compulsive disorder: a multicenter randomized controlled trial. *Journal of the American Medical Association*, *280*, 1752–1756.

March, J. S., Franklin, M. E., Leonard, H., Garcia, A., Moore, P., Freeman, J., et al. (2007). Tics moderate treatment outcome with sertraline but not cognitive-behavior therapy in pediatric obsessive–compulsive disorder. *Biological Psychiatry*, *61*, 344–347.

March, J. S., Franklin, M., Nelson, A., & Foa, E. (2001). Cognitive-behavioral psychotherapy for pediatric obsessive compulsive disorder. *Journal of Clinical Child Psychology*, *30*, 8–18.

March, J. S., Johnston, H., Jefferson, J. W., Kobak, K. A., & Greist, J. H. (1990). Do subtle neurological impairments predict treatment resistance to clomipramine in children and adolescents with obsessive–compulsive disorder? *Journal of Child and Adolescent Psychopharmacology*, *1*, 133–140.

March, J. S., & Mulle, K. (1998). *OCD in children and adolescents: a cognitive-behavioral treatment manual*. New York: Guilford.

Morris, S. B. (2008). Estimating effect sizes from pretest–posttest control group designs. *Organizational Research Methods*, *11*, 364–386.

National Institute of Mental Health. (1985). Rating scales and assessment instruments for use in pediatric psychopharmacology research. *Psychopharmacological Bulletin*, *21*, 839–843.

O'Kearney, R. (2007). Benefits of cognitive-behavioural therapy for children and youth with obsessive–compulsive disorder: re-examination of the evidence. *Australian and New Zealand Journal of Psychiatry*, *41*, 199–212.

*Pediatric and Treatment Study (POTS) Team. (2004). Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive–compulsive disorder: the pediatric OCD treatment study (POTS) randomised controlled trial. *Journal of the American Medical Association*, *292*, 1969–1976.

Perlmutter, S. J., Leitman, S. F., Garvey, M. A., Hamburger, S., Feldman, E., & Leonard, H. L. (1999). Therapeutic plasma exchange and intravenous immunoglobulin for obsessive–compulsive disorder and tic disorders in childhood. *Lancet*, *354*, 1153–1158.

*Piacentini, J., Bergman, R. L., Chang, S., Langley, A., Peris, T., Wood, J. J., et al. (2011). Controlled comparison of family cognitive behavioral therapy and psychoeducation/relaxation training for child obsessive–compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*, 1149–1161.

Piacentini, J., Bergman, R. L., Keller, M., & McCracken, J. (2003). Functional impairment in children and adolescents with obsessive–compulsive disorder. *Journal of Child and Adolescent Psychopharmacology*, *13*, 61–69.

Piacentini, J., & Jaffer, M. (1999). *Measuring functional impairment in youngsters with OCD: manual for the child OCD impact scale (COIS)*. Los Angeles: UCLA Department of Psychiatry.

Piacentini, J., & Langley, A. K. (2004). Cognitive-behavioral therapy for children who have obsessive–compulsive disorder. *Journal of Clinical Psychology*, *60*, 1181–1194.

Piacentini, J., Langley, A., & Roblek, T. (2007). *Cognitive-behavioral treatment of childhood OCD: therapist guide*. New York: Oxford University Press.

Rapoport, J. L., Elkins, R., & Mikkelsen, E. (1980). Clinical controlled trial of clomipramine in adolescents with obsessive–compulsive disorder. *Psychopharmacology Bulletin*, *16*, 61–63.

Rapoport, J. L., Inoff-Germain, G., Weissman, M. M., Greenwald, S., Narrow, W. E., Jensen, P. S., et al. (2000). Childhood obsessive–compulsive disorder in the NIMH MECA study: parent versus child identification of cases. *Journal of Anxiety Disorders*, *14*, 535–548.

*Riddle, M. A., Reeve, E. A., Yaryura-Tobias, J. A., Yang, H. M., Claghorn, J. L., Gaffney, G., et al. (2001). Fluvoxamine for children and adolescents with obsessive–compulsive disorder: a randomized, controlled, multicenter trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 222–229.

*Riddle, M. A., Scahill, L., King, R. A., Hardin, M. T., Anderson, G. M., Ort, S. I., et al. (1992). Double-blind, crossover trial of fluoxetine and placebo in children and adolescents with obsessive–compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*, 1062–1069.

Rosa-Alcázar, A. I., Iniesta-Sepúlveda, M., & Rosa-Alcázar, A. (2012). Effectiveness of cognitive-behavioral treatments of obsessive–compulsive disorder in children and adolescents: a qualitative review. *Anales de Psicología*, *28*, 313–326.

Rosa-Alcázar, A., Sánchez-Meca, J., Gómez-Conesa, A., & Marín-Martínez, F. (2008). Psychological treatment of obsessive–compulsive disorder: a meta-analysis. *Clinical Psychology Review*, *28*, 1310–1325.

Rothstein, H. R., Sutton, A. J., & Borenstein, M. (Eds.). (2005). *Publication bias in meta-analysis: prevention, assessment, and adjustments*. Chichester, UK: Wiley.

Sánchez-Meca, J., & Marín-Martínez, F. (2008). Confidence intervals for the overall effect size in random-effects meta-analysis. *Psychological Methods*, *13*, 31–48.

Salkovskis, P. M. (1999). Understanding and treating obsessive compulsive disorder. *Behaviour Research and Therapy*, *37*, S29–S52.

Scahill, L., Riddle, M. A., McSwiggin-Hardin, M., Ort, S. I., King, R. A., Goodman, W. K., et al. (1997). Children's Yale-Brown Obsessive–Compulsive Scale: reliability and validity. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 844–852.

Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DICS-IV): description, differences from previous versions, and reliability of some common diagnoses. *Journal American Academy of Child and Adolescent Psychiatry*, *39*, 28–38.

Simons, M., Schneider, S., & Herpertz-Dahlmann, B. (2006). Metacognitive therapy versus exposure and response prevention for pediatric obsessive–compulsive disorder. *Psychotherapy and Psychosomatics*, *75*, 257–264.

Startup, M., Jackson, M. C., & Bendix, S. (2002). The concurrent validity of the global assessment of functioning (GAF). *British Journal of Clinical Psychology*, *41*, 417–422.

Storch, E. A., Geffken, G. R., Merlo, L. J., Mann, G., Duke, D., Munson, M., et al. (2007). Family-based cognitive-behavioral therapy for pediatric obsessive–compulsive disorder: comparison of intensive and weekly approaches. *Journal of the American Academy of Child and Adolescent Psychiatry*, *46*, 469–478.

*Storch, E. A., Caporino, N. E., Morgan, J. R., Lewin, A. B., Rojas, A., Brauer, L., et al. (2011). Preliminary investigation of web-camera delivered cognitive-behavioral therapy for youth with obsessive–compulsive disorder. *Psychiatry Research*, *189*, 407–412.

Storch, E. A., Larson, M. J., Muroff, J., Caporino, N., Geller, D., Reid, J. M., et al. (2010). Predictors of functional impairment in pediatric obsessive–compulsive disorder. *Journal of Anxiety Disorders*, *24*, 275–283.

Sukhodolsky, D. G., Gorman, B. S., Scahill, L., Findley, D., & McGuire, J. (2013). Exposure and response prevention with or without parent management training for children with obsessive–compulsive disorder complicated by disruptive behavior: a multiple-baseline across-responses design study. *Journal of Anxiety Disorders*, *27*, 298–305.

Turner, C. (2006). Cognitive-behavioural theory and therapy for obsessive–compulsive disorder in children and adolescents: current status and future directions. *Clinical Psychology Review*, *26*, 912–938.

Turner, C., Heyman, I., Futh, A., & Lovell, K. (2009). A pilot study of telephone cognitive-behavioral therapy for obsessive–compulsive disorder in young people. *Behavioral and Cognitive Psychotherapy*, *37*, 469–474.

Valderhaug, R., & Ivarsson, T. (2005). Functional impairment in clinical samples of Norwegian and Swedish children and adolescents with obsessive–compulsive disorder. *European Child and Adolescent Psychiatry*, *14*, 164–173.

Watson, H. J., & Rees, C. S. (2008). Meta-analysis of randomized, controlled treatment trials for pediatric obsessive–compulsive disorder. *Journal of Child Psychology and Psychiatry*, *49*, 489–498.

Wagner, A. P. (2003). Cognitive-behavioral therapy for children and adolescents with obsessive compulsive disorder. *Brief Treatment and Crisis Intervention*, *3*, 291–306.

*Williams, T. I., Salkovskis, P. M., Forrester, L., Turner, S., White, H., & Allsop, M. A. (2010). A randomised controlled trial of cognitive behavioural treatment for obsessive compulsive disorder in children and adolescents. *European Child and Adolescent Psychiatry*, *19*, 449–456.

Wolff, R. P., & Wolff, L. S. (1991). Assessment and treatment of obsessive–compulsive disorder. *Children Behavior Modification*, *15*, 372–393.