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A systematic review of predictors and moderators of response to psychological therapies in OCD: Do we have enough empirical evidence to target treatment?☆,☆☆,★,★★

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HIGHLIGHTS

- Predictors of outcome are commonly reported.
- Potential associations emerged between a small number of predictors and outcome.
- The quality of assessing and reporting of predictors was relatively poor.
- Methodological/reporting guidelines can guide assessment/reporting of predictors.

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ABSTRACT

Obsessive–compulsive disorder (OCD) is a disabling mental health condition. Despite effective psychological treatments for OCD, a significant percentage of patients fail to experience lasting benefit. Factors underlying variable treatment response are poorly understood. Moderators of outcome can help understand "for whom" and "under what circumstances" an intervention works best and thus improve service effectiveness.

This paper synthesizes the evidence on predictors and moderators and assesses the quality of reporting of related analyses in psychological therapies for adults with OCD. Trials were identified through electronic searches (CENTRAL, MEDLINE, PsycINFO, EMBASE), key author, and reference list searches of relevant systematic reviews. Fifty five percent (38/69) of relevant trials reported baseline factors associated with outcome; these encompassed clinical, demographic, interpersonal, OCD symptom-specific, psychological/psychosocial, and treatment-specific variables. Predictors were commonly assessed via a validated pre-randomization measure, though few trials adopted best practice by stating a priori hypotheses or conducting a test of interaction. Potential associations emerged between worse OCD treatment outcome and the following factors: hoarding pathology, increased anxiety and OCD symptom severity, certain OCD symptom subtypes, unemployment, and being single/not married. However, the applied utility of these analyses is currently limited by methodological weaknesses.

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1. Introduction

OCD is a disabling disorder, characterized by a pattern of repetitive obsessive thoughts, images, or impulses and a ritualized pattern of covert mental acts or overt behaviors, aimed at reducing the associated anxiety and fear (APA, 2000). Data from the National Comorbidity Survey Replication suggests a lifetime prevalence of OCD of 1.6% (Kessler, Berglund, Demler, Jin, & Walters, 2005). OCD commonly takes a chronic course (Ayuso-Mateos, 2002) and has been found to affect multiple areas of functioning (Koran, Thienemann, & Davenport, 1996; Schneier, 1997). Although historically conceptualized as a significant treatment challenge (Foa, Franklin, & Kozak, 1998), research efforts during the 1960s and 70s have led to substantial advances in OCD treatments.

Psychological interventions have become an increasingly important part of the management of this condition. On strength of evidence, cognitive behavioral therapy (CBT) has been recommended by the APA (2007) and the National Institute for Health and Care Excellence (NICE, 2006) as the treatment of choice for OCD. The core treatment component consists of exposure and response prevention (ERP—aimed at the gradual habituation to anxiety-provoking stimuli) and additional cognitive strategies, targeting irrational and dysfunctional beliefs about the meaning and significance of obsessive thoughts (NICE, 2006). Systematic reviews have shown such interventions to be highly effective in reducing symptoms (e.g. Eddy, Dutra, Bradley, & Westen, 2004; Rosa-Alcázar, Sánchez-Meca, Gómez-Conesa, & Marín-Matínez, 2008). However, average effects from systematic reviews do not necessarily translate to the individual patient. Despite moderate to large average treatment effects, outcomes vary significantly between trials and participants. In a review by Abramowitz (2006), the author highlights that despite significant progress in the efficacy and effectiveness of psychological interventions in OCD, early drop-out and limited response to the recommended psychological treatment for OCD leavearound 50% of patients clinically unwell. Similarly, Eddy etal. (2004) found that while around two thirds of treatment completers improved, only half of those who failed to complete treatment showed improvements on the Yale–Brown Obsessive-Compulsive Scale (Y–BOCS).

The ability to prospectively distinguish treatment responders from non-responders has interested researchers for many years (e.g. Fritzler, Hecker, & Losee, 1997; Barrett, Farrell, Dadds, & Boulter, 2005; De Araujo, Ito, & Marks, 1996; Marks etal., 1988; McLean etal., 2001). The potential utility of identifying factors which can reliably predict treatment response is substantial. In research, moderators can inform the selection of inclusion and exclusion criteria for stratification in future randomized trials (RCTs) to maximize statistical power, while in clinical practice they can help to identify those atrisk of a poor prognosis and may inform the matching of individual patients with suitable treatments (Kraemer, Wilson, Fairburn, & Agras, 2002). Although a wide range of potential predictors have been identified, there remains limited consensus about what factors are associated with response to psychological treatments in OCD, limiting their applied impact on routine treatment decision-making.

Through a narrative synthesis of 49 open and controlled trials, Keeley and colleagues summarized predictors of response to CBT in OCD (Keeley, Storch, Merlo, & Geffken, 2008). The authors reported a number of variables to be consistently associated with outcome. These comprised: the strength of the therapeutic relationship, the nature of patients' family environment, and several different clinical factors—OCD severity, symptom subtype, severe depression, and concurrent personality disorder (Keeley et al., 2008). However, Keeley et al. (2008) acknowledge the largely conflicting nature of the evidence and limitations in the interpretability of findings due to differences in the measurement of respective predictors. Moreover, these findings ought to be considered in light of a number of limitations of this review. This synthesis included both open and controlled trials, and adult and pediatric samples (Keeley et al., 2008). Importantly, the analyses of predictor effects failed to consider the quality of either the included studies or the predictor analyses conducted. Considering these limitations and the substantial research activity since Keeley et al.'s (2008) review was published, the present systematic review serves to update and strengthen existing evidence on predictors and moderators of response to psychological therapies in OCD. In this synthesis, the following three questions have been addressed:

- What predictors/moderators of outcome have been measured in psychological therapies for OCD?
- What proportion of OCD trials adopts methodological best practice in the assessment of predictors/moderators?
- What is the existing evidence base concerning these predictors/moderators and their relationship with OCD treatment outcome?

2. Methods

2.1. Identification of studies

The PRISMA guidelines for reporting of systematic reviews and meta-analyses were followed (The PRISMA Group, 2009).[1] Articles were primarily identified through an electronic literature search on the Cochrane Collaboration's Clinical Trials Register (CENTRAL), completed in January 2012, using the MESH-, and text term "obsessive–compulsive disorder" and the text terms "OCD OR obsessive–compulsive neurosis". Previous authors have demonstrated 94% sensitivity for a search for randomized controlled trials using only CENTRAL (Royle & Waugh, 2005). In view of the potential delays in uploading trials onto CENTRAL, this primary search was supplemented by additional searches using the MEDLINE, PsycINFO, and EMBASE databases for relevant articles published between 2009 and January 2012, using the text, and MESH terms "obsessive–compulsive disorder" OR "OCD" in combination with "psychological intervention" and "randomized controlled trial". Database searches were supplemented by a key author search. We also identified 25 relevant published systematic reviews on the Cochrane database (Cochrane Database of Systematic Reviews and DARE) and screened these for additional relevant articles.[2] ZETOC automated literature alerts, targeting key journals in the domains of medicine and psychology, were also employed to identify studies published in the time period following the main searches. Study inclusion was determined on the basis of the following criteria: (a) adults with a diagnosis of OCD, consistent with DSM-, ICD-, or equivalent international diagnostic criteria (e.g. CCMD in the Chinese literature), (b) at least one treatment condition involved a psychological intervention, defined as a structured "process designed to bring about modification of feelings, cognitions, attitudes, and behaviour" (Strupp, 1978, p. 3), (c) studies were published in full-text within the search-period and adopted a randomized-controlled, quasi-random, or cross-over design, as defined in Cochrane criteria, (d) papers were not excluded on the basis of language of publication (non-English papers included N = 3), and (e) studies reported on the relationship between baseline variables and treatment outcome.

2.2. Coding of study characteristics

All predictors reported in relevant studies were noted. This report focuses on those predictive factors assessed pre-randomization, which may be conceptualized as potential *moderators* of treatment response (Baron & Kenny, 1986). Table 1 provides a summary of key characteristics of moderators.

Predictor analyses were presented in a number of different ways. Hence, we categorized analyses as follows:

1) Moderator effect—where the effect of the baseline variable on outcome was assessed through a direct test of the interaction between the baseline variable and the intervention(s).
2) Non-specific predictors of outcome, where the main effect of a predictor on outcome was assessed for the sample as a whole.
3) Subgroup analyses involving splitting of trial dataset into different groups:
   (a) Non-specific predictors of outcome, where the main effect of a predictor on outcome was assessed within the treatment or control group only.
   (b) Taking a subgroup of patients from the treatment and control groups and comparing intervention with control patients within that subgroup.
   (c) Splitting the overall sample into two groups and analyzing statistical significance in both groups separately (e.g. splitting the sample into those with and without baseline depression and assessing outcome for both groups separately).

Table 1
Characteristics of moderators of outcome.

Criterion	Moderator characteristic	
Type of question	When/for whom a cause–effect relationship occurs	
Stability of predictor variable	Trait, i.e. a relatively stable characteristic, innate attribute, enduring process, or disposition of an individual/context/environment/situation	
Sequence of operation	Precedes IV and DV	
Relationship with IV	Uncorrelated with IV	
Function in the causal relationship	3rd variable modifying the causal relationship	
Applied function	Clinical practice: –Inform personalized treatment decisions	Research practice: –Inform stratification in RCTs for greater statistical power –Inform inclusion criteria for greater sample homogeneity

Note. DV = dependent variable; IV = independent variable; RCT = randomized controlled trial. Information adapted from MacKinnon and Luecken (2008); Wu and Zumbo (2008).

Initial study selection was conducted by the first author, who excluded studies which were not within the scope of this review.[3] A reliability check of this initial screen was conducted by an independent researcher for a 10% random sample (kappa = 0.95). The remaining studies were assessed as full-texts. Additionally, a reliability check of exclusion at the full-text stage was conducted for 25 studies by one of the study authors (kappa = 1). Two of the review authors independently extracted data on the study characteristics, main outcomes, and predictor effects. Discrepancies were discussed between the two authors; when agreement could not be reached, a third author was consulted.

Predictor variables were categorized post hoc into six groupings:

- Clinical — illness and treatment context characteristics, not part of/linked to the OCD intervention treatment specifically; e.g. referral source, comorbidities.
- Demographic — characteristics relating to the OCD study sample/population; e.g. age, gender, educational status.
- Interpersonal — characteristics of the OCD sample's interpersonal environment/situation; e.g. marital or relationship status; marital satisfaction.
- OCD symptom-specific — variables relating specifically to OCD symptoms; e.g. OCD symptom severity, OCD illness duration, and age of OCD onset.
- Psychological/psychosocial — variables relating to psychological and psychosocial characteristics of the OCD sample or the individual patient; e.g. treatment expectancy and motivation, IQ.
- OCD treatment-specific — treatment context variables, part of/specific to, the intervention treatment(s).

The criteria for the quality assessment of predictor and moderator analyses were based on existing quality criteria outlined in two recent publications by Pincus et al. (2011) and Sun et al. (2012):

- Predictors assessed through a validated assessment tool: The assessment of predictor variables through non-validated tools may call into question the reliability and validity of the constructs tested.
- Predictor measures taken pre-randomization: Predictors ought to be measured pre-randomization as some may change following group allocation. This criterion does not apply to procedural or unmodifiable variables, e.g. age.
- <5 predictors tested: The precision of a predictor model decreases with the number of factors in the model; measuring fewer variables may increase the reliability/credibility of identified predictor effects.

[1] Review protocol available from corresponding author upon request.
[2] List of systematic reviews is available in Appendix A.
[3] Table of excluded studies is available in Appendix B.

- A priori hypothesis of anticipated predictor effect: The selection of predictors ought to be theory-, or evidence-driven with the view to produce confirmatory results. Hence, authors ought to state the anticipated predictor effect. Post hoc testing of predictor effects can at best offer exploratory findings.
- Analysis method-direct test of interaction between predictor and treatment type: A moderator by definition interacts with the independent variable X to predict the outcome variable Y (e.g. age may interact with treatment condition to predict outcome). Thus, to show that a moderation effect has occurred, a test of the interaction between the moderator and X should be conducted.

The above list represents the key quality criteria agreed by the review authors on the basis of which each predictor variable was assessed. The selection of these specific criteria was based on their importance in the relevant methodological publications (Pincus et al., 2011; Sun et al., 2012) and their relevance in the context of the presented literature. For each predictor (e.g. OCD symptom severity), a percentage score of the number of analyses which met these criteria (where applicable) was calculated. This score reflects the quality of the predictor/moderator analyses, not the quality or risk of bias of included studies as a whole.

To determine the risk of bias of included trials we conducted a quality assessment, based on criteria derived from the Cochrane Collaboration's tool for the assessment of risk of bias (Higgins & Altman, 2008). Intervention fidelity, manualization of treatment, generation of random allocation sequence, allocation concealment, blinding of outcome assessors, intention-to-treat analysis, and levels of study attrition at post-treatment were assessed. Rates of attrition were grouped into categories of <5%, 5–20%, and >20%. Participant blinding was not assessed, as this is generally not relevant in the context of psychological interventions. For quantitative analyses of risk of bias, a dichotomised score of allocation concealment (low versus uncertain/high risk of bias) was used, as this is most reliably associated with outcome (Pildal, Hróbjartsson, Jørgensen, Altman, & Gøtzsche, 2007; Schulz, Chalmers, Hayes, & Altman, 1995) and may be of greater relevance in the context of psychological therapies, where other key quality criteria such as adequate blinding of participants and personnel are rarely possible (Bower et al., 2013). Quality scores were assessed independently by two of the review authors. Discrepancies in quality ratings were resolved through discussion amongst the research team.

2.3. Analysis

2.3.1. Publication bias

Assessing study characteristics associated with the reporting of predictor/moderator analyses may aid interpretation and use of findings (Sun et al., 2011), as it may suggest bias in reporting of such analyses. Hence, we identified all trials relating to psychological therapies in OCD, and distinguished those which reported and did not report baseline predictors/moderators of outcome. We assessed whether differences in risk of bias, sample size, year (divided arbitrarily into studies published 2002 onwards and those published prior) and country of publication (coded "USA/Canada" and "other"), and treatment effect were associated with the reporting of predictor/moderator analyses. The selection of these characteristics was determined post hoc. Treatment effect sizes were compared through meta-analysis, using STATA software version 11 (StataCorp., 2009). Only trials comparing active with control conditions (waitlist; attention placebo), and those assessing the independent effect of one or more psychological interventions (e.g. psychological intervention plus drug versus drug) were included in the meta-analysis. Where authors failed to report means and standard deviations (N = 2; Fals-Stewart, Marks, & Schafer, 1993; Jones & Menzies, 1998), effect sizes were estimated using methods previously outlined by Lipsey and Wilson (2000). Missing standard deviations were calculated by taking the median standard deviation from other studies included in this review.

2.3.2. Predictors and moderators of outcome

The optimal analysis would have involved assessment of the relationship between predictors/moderators and treatment outcome through meta-analysis of appropriate interaction statistics (Hunter & Schmidt, 2004). However, many of the included studies failed to report the necessary data from which to calculate an effect size of the interaction. Without the option for meta-analysis, published analyses have adopted a narrative synthesis approach (Keeley et al., 2008). Narrative integrations may be defined as a non-quantitative way "to portray multiple findings in a connected, verbal report" (Smith, Glass, & Miller, 1980, p.36). Here, we adopted a *box-score approach*. This involved tabulating predictors and their reported relationship with outcome, defined in terms of significance and direction (negative, positive, or no relationship; e.g. Green & Hall, 1984); included studies falling into each respective group were tallied and the majority of studies falling into any specific category is considered to indicate the likely relationship between the predictor and outcome (Light & Smith, 1971). The box-score approach enables basic quantification of reported predictor effects and the identification of patterns across the literature. It also facilitates some assessment of the relationship between quality of analyses and reported effects. We consider the advantages and disadvantages of this approach in more detail in the Discussion section.

A small number of included papers reported overlapping samples. Where this was the case, predictor effects tested for the whole sample were reported while effects tested for patient subgroups were excluded from the analyses to avoid the double-reporting of data relating to the same sample. When study authors assessed the impact of the same predictor via multiple statistical tests, these were ordered by rigor and results of the most adequate form of analysis (ordered as: test of the interaction between the predictor and treatment type(s), multivariate analyses of main predictor effects, univariate analyses of predictor effects, and correlation analyses). This grading of analysis quality was based on relevant methodological publications, highlighting the superiority of interaction tests in producing reliable results in the assessment of baseline predictors of outcome (Brookes et al., 2004; Schulz & Grimes, 2005). Where the impact of the same predictor was assessed for multiple time-points and outcome measures within studies, the results closest to post-treatment for a measure of overall OCD symptom severity were reported. Where there was a conflict between the quality of the statistical analysis and the dependent variable of interest, the appropriate outcome measure was prioritized.

In light of the substantial number of predictors reported, the box-score assessment of the predictor/moderator-outcome relationship was limited to those predictors reported in ≥5 studies (see Fig. 2).

3. Results

In the subsequent paragraphs we present the following data:

1) Description of trial characteristics.
2) Description of the predictors/moderators measured and outcomes for which predictor effects were assessed.
3) Analyses of publication bias.
4) Box-score analysis of predictors/moderators of outcome.
5) Risk of bias of included studies.

3.1. Characteristics of included studies

A PRISMA diagram details the study identification and selection process (Fig. 1). Thirty eight trials met the study inclusion criteria. A further 31 trials of psychotherapy for OCD, which did not attempt the analysis of predictors of outcome, were used solely in the assessment of publication bias. The following study characteristics relate to the 38 studies included in this review of predictor effects.

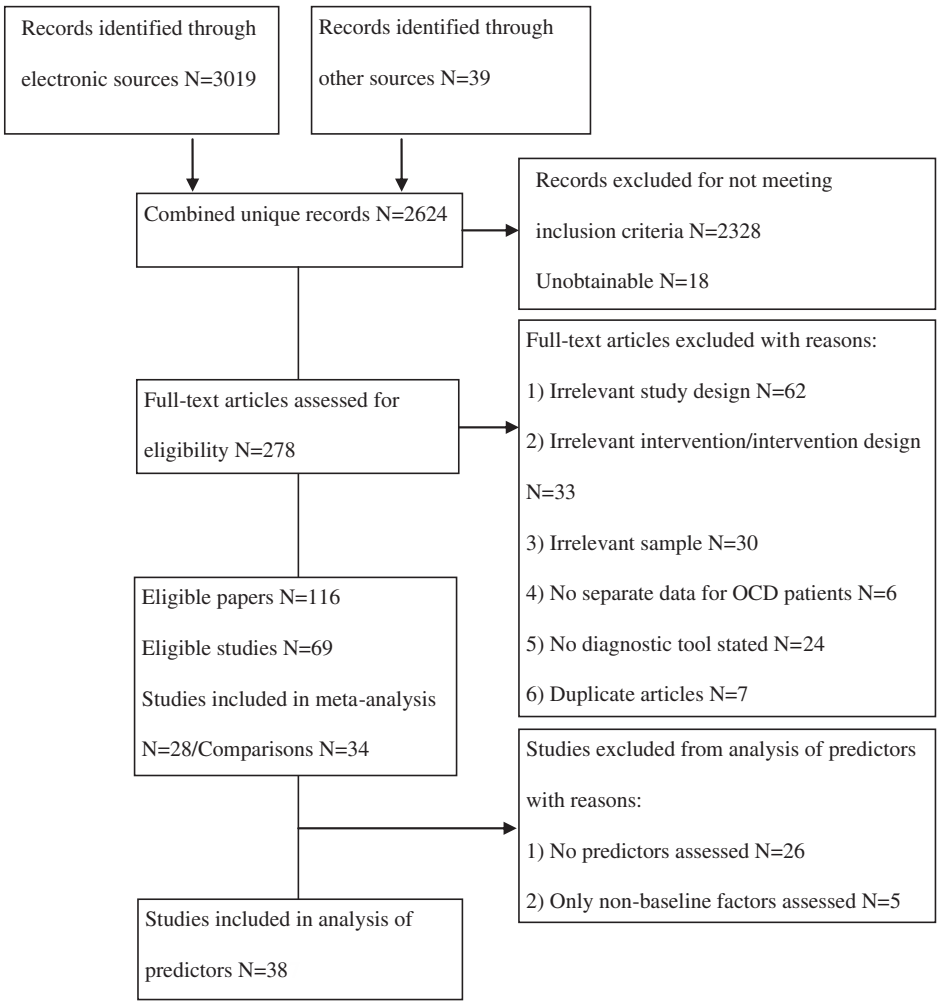

Fig. 1. PRISMA diagram of study identification and selection.

Relevant trials included a total of 2274 participants (mean sample size = 65, SD = 42.7). Participants had a mean age of 35.3 years and on average study samples consisted of a slightly greater number of women than men. All studies included OCD patients only. Two studies recruited patients on the basis of primary obsessions with no apparent compulsive symptoms, or compulsions unrelated to-, or less severe than obsessive symptoms (Freeston et al., 1997; Whittal, Woody, McLean, Rachman, & Robichaud, 2010). Fifty percent of included trials offered information on the symptom breakdown of the OCD sample.

Study interventions varied significantly in terms of content, duration, intensity, and delivery. Eight trials included minimal-contact treatments, involving less than 10 h of patient–therapist contact (NICE, 2006); in four of these, treatment was delivered remotely, substituting patient–therapist interaction with computerized or manualized therapy tools. Intervention content was largely based on CBT principles. Ninety three percent of psychological interventions were categorized as CBT, comprised of ERP and cognitive techniques. The remaining interventions consisted primarily of (a) cognitive therapy without ERP and (b) other treatment approaches (Stress Management Training). On average participants received 17.9 treatment sessions (SD = 13.3). In those studies offering face-to-face treatment, session duration ranged between 30 and 150 min and the average intervention intensity was 22.7 h (SD = 12.2). Study interventions were delivered by a wide range of professionals with varying levels of experience and training, including clinical psychologists, psychiatrists, counselors, social workers, and students in related fields.

3.2. Predictors/moderators and outcomes

Characteristics of the 38 included studies, baseline predictors/moderators, and the outcomes for which they were assessed are specified in Table 2. Three studies specifically assessed mediation effects (Simpson et al., 2010; Van Balkom et al., 1998; Van Oppen et al., 1995) and one study assessed a moderated mediation model (Whittal et al., 2010). These meditational analyses are not reported further here.

While study authors reported a wide range of different predictors (Fig. 2), certain variables and categories of predictors were far more commonly assessed. Most frequently, OCD symptom-specific variables were reported, including OCD symptom severity, illness duration, symptom subtypes, obsessive–compulsive beliefs, and age of illness onset. Similarly, clinical variables, not directly associated with OCD were commonly reported; within this category, depression severity, medication use, past treatment, and anxiety severity were assessed. These were followed by demographic variables (age, gender, employment, and educational status), the interpersonal factor (marital/relationship status) and a psychological predictor (treatment expectancy).

3.3. Comparison of trials with and without predictor analyses

Comparative analyses indicated that two study characteristics were significantly associated with the reporting of predictor or moderator analyses. Trials reporting predictor analyses were larger (respective mean sample sizes: 65 versus 42.7) and at lower risk of bias [$\chi 2\ (1, 69) = 4.3$, $p = .039$; Table 3] than those which did not.

Table 2
Characteristics of included studies.

Author/year	Country	Study design	Sample size	Intervention(s)	Psychological intervention intensity	Control group(s)	Predictor/moderator variables	Outcome variables
Althaus et al. (2000)	Germany	RCT	30	OCD-specific group CBT; generic group CBT	U/C	Na	Age; OCD symptom duration	OCD symptom reduction; depressive symptom reduction; therapist-rated overall state/symptoms; subjective improvement
Belotto-Silva et al. (2012)	Brazil	RCT	158	Group CBT; fluoxetine	30 h	Na	Age; gender; MDD; comorbidity (social phobia; dysthymia; PTSD; BD; compulsive buying)	OCD symptom reduction
Valpato Cordioli et al. (2003)	Brazil	RCT	47	Group CBT	24 h	WL	OCD severity; intensity of overvalued ideas; age at illness onset; comorbid anxiety/depression; anti-obsessional medication use	Relapse; OCD severity
Cottraux et al. (1990)	France	RCT	60	BT + fluvoxamine	U/C	Anti-exposure + fluvoxamine/BT + placebo	Pre-tx depression severity; OCD symptom duration; tx expectancy; behavioral avoidance; target compulsions; compulsive activity checklist	Compulsive symptoms (duration/day); tx success
Cottraux et al. (2001)	France	RCT	65	CT; BT	20 h	Na	Tx expectancy; pre-tx severity of OCD/depression/responsibility/interpretations of intrusive thoughts/obsessive thoughts	OCD symptom reduction
De Araujo et al. (1995)	UK	RCT	56	BT (in vivo & imaginal exposure); BT (in vivo exposure only)	13.5 h	Na	OCD symptom subtype (primary washing/cleaning compulsions); age; gender; OCD symptom duration; age at illness onset; referral source; non-OCD psychiatric symptoms; pre-tx severity of OCD/target obsessions/compulsions/depression/avoidance/free-floating anxiety/overall well-being/work & social adjustment/beliefs; medication use	Change in target compulsions/obsessions/overall well-being (CGI); compliance at week 1
Dreessen et al. (1997)	Netherlands	RCT	52	CT; BT; CBT	12 h	Na	PD trait/sub-threshold (avoidant; dependent; OC; paranoid; schizotypal; self-defeating; passive-aggressive); N of PDs; N of PD traits/sub-clinical PDs; ≥1 PD/sub-threshold PD	OCD symptom reduction/change/recovery; composite score (MOCI/BAT); composite score (MOCI/BAT/LOI/Rational Belief Inventory/Depressive Symptoms Inventory); OCD recovery; behavioral avoidance reduction/change
Emmelkamp et al. (1990)	Netherlands	RCT	54	Partner-assisted BT; patient-based BT	Min. 6.75 h	Na	Marital distress	OCD severity; anxiety-related discomfort; marital adjustment; problem-solving capacity; depression; state/trait anxiety; anger; curiosity
Fineberg et al. (2005)	UK	Quasi-random study	48	Group CBT	24 h	Group relaxation	OCD symptom duration; anti-obsessional medication use	OCD symptom change/reduction
Foa et al. (1984)	USA	Quasi-random study	32	Exposure/response prevention/combined ERP	38 h	Na	Hospitalization	Compulsion checklist; urge to ritualize; main fear; anxiety during exposure; avoidance of daily activities; severity of obsessions/compulsions; anxiety/depression; general functioning; time spent on compulsions
Foa et al. (2005)	USA	RCT	122	BT; BT + clomipramine	40/46 h	Clomipramine; placebo	Tx site; OCD severity	OCD severity; time to relapse; drop-out status
Freeston et al. (1997)	Canada	RCT	38	CBT	60 h	WL	Pre-tx credibility; pre-tx expectancy	Tx outcome (U/C)
Greist et al. (2002)	USA/Canada	RCT	218	Computerized CBT; therapist-led CBT	HW only/10 h	Progressive muscle relaxation	OCD symptom subtype (ritual type; hoarding symptoms; sexual & religious obsessions); prior SSRI tx; tx site; tx expectations; pre-tx severity of OCD/depression	% tx goals considered completed; OCD symptom reduction; early tx discontinuation
Jaurietta et al. (2008)	Spain	RCT	57	Individual/group CBT	15/30 h	WL	OCD symptom subtype (contamination/cleaning obsessions; ordering/symmetry obsessions; hoarding obsessions/compulsions; checking obsessions/compulsions; slowness); employment status	Depression-, anxiety-, OCD symptom reduction

Jónsson et al. (2011)	Denmark	RCT	110	Individual/group CBT	19/37 h	Na	Responsibility; thought-action fusion; pre-tx severity of OCD/depression; age; gender; age at illness onset; OCD symptom duration; OCD symptom subtype; educational level; employment status; relationship status; comorbid Axis I/II comorbidities; medication use	OCD symptom reduction/change
Keijsers et al. (1995)	Netherlands	Cross-over study	U/C	Phase 1: Exposure; Phase2: Response prevention (vice versa)	8 h (per treatment phase)	Na	AD medication use; pre-tx compulsive behavior/obsessive fear; depression; OCD symptom duration; tx motivation; marital satisfaction	Compulsive behavior; obsessive fear; tx success
Kenwright et al. (2005)	UK	RCT	44	Computerized CBT + scheduled/requested help-line support	U/C	Na	Tx preference; age; gender; OCD symptom duration; past BT; SRI medication use	OCD severity
Lakatos (1997)	Germany	RCT	28	CBT; BT	U/C	Na	Pre-tx depression severity	OCD severity; drop-out status
Marks et al. (1988)	UK	RCT	49	Self-controlled BT + clomipramine; self- & therapist-controlled BT + clomipramine	U/C	Anti-exposure + clomipramine	Age; gender; marital status; OCD symptom duration; age at illness onset; OCD severity; tx expectancy; factor analyzed OCD symptom clusters	Drop-out status; target compulsions; time spent on compulsions; severity of obsessions/compulsions; social disability/adjustment; free-floating anxiety; depression; habituation to brief neutral/aversive stimuli
Marks et al. (2000)	UK	RCT	15	Computerized CBT; therapist-led CBT	HW only/10 h	Progressive muscle relaxation	Pre-tx discomfort during/after OCD imagery	Work-social adjustment; severity of OCD/depression
McLean et al. (2001)	Canada	RCT	93	Group BT/; group CBT	30 h	WL	OCD symptom subtype; age; pre-tx OCD severity; medication use; gender; belief measures (U/C); depression; disability status; educational level; age at illness onset; OCD symptom duration; pre-tx thought-action fusion/responsibility/obsessive beliefs/thematic similarity (of OCD symptom content across tx group members)	Drop-out status; OCD severity/improvement; recovery status; tx refusal
Meyer et al. (2010)	Brazil	RCT	93	MI + thought mapping + group CBT; Information + group CBT	26 h	Na	Age; gender; age at illness onset; OCD symptom duration; age at symptom interference; depression; pre-tx overall well-being (CGI); OCD severity	Drop-out status
Nakao et al. (2005)	Japan	RCT	32	BT + placebo	9 h	Autogenic training + placebo; autogenic training + fluvoxamine	OCD symptom duration	OCD symptom reduction
Nakatani et al. (2005)	Japan	RCT	28	BT + placebo	9 h	Autogenic training	Age at illness onset; OCD symptom duration; IQ; history of MDD; pre-tx severity of obsessive/compulsive symptoms; OCD symptom subtype; pre-tx OCD severity; pre-tx overall well-being (CGI)	OCD symptom reduction
Rector et al. (2009)	Canada	RCT	29	CBT for OCD & MDD; CBT for OCD	20 h	Na	Age	Drop-out status
Rowa et al. (2007)	Canada	RCT	37	Home-/office-based BT	21 h	Na	OCD related impairment	U/C
Simpson et al. (2008)	USA	RCT	111	BT (authors class as CBT) + SSRI	41 h	CBT (SMT) + SSRI	Tx site; OCPD diagnosis/severity; pre-tx OCD severity, QoL; N of comorbid Axis I/II disorders; N of past SRI trials; depression; anxiety; satisfaction with social situation; gender; OCPD symptoms (preoccupation with details; perfectionism; excessive devotion to work; hyper-morality; inability to discard; reluctance to delegate tasks; miserliness; rigidity/stubbornness); age; marital status; employment status; OCD symptom insight; OCD symptom subtype (hoarding); OCD symptom duration	OCD severity; satisfaction with social situation; depression; anxiety; QoL

(continued on next page)

Table 2 (*continued*)

Author/year	Country	Study design	Sample size	Intervention(s)	Psychological intervention intensity	Control group(s)	Predictor/moderator variables	Outcome variables
Simpson et al. (2010)	USA	RCT	30	MI + BT; BT	Min. 27 h	Na	Pre-tx OCD severity; depression severity; OCD symptom insight; QoL; Axis I comorbidity; N of past SRI trials; gender; employment status; OCD symptom subtype (hoarding); readiness to change; work impairment	OCD severity
Sousa et al. (2006)	Brazil	RCT	56	Group CBT; Sertraline	24 h	Na	Pre-tx AD medication use	OCD severity
Tenneij et al. (2005)	Netherlands	RCT	96	BT + medication	17.5 h	Medication	OCD severity; depression/anxiety symptoms; OCD symptom duration; age	Drop-out status
Tolin, Frost, and Steketee (2007)	USA	RCT	41	Patient-/therapist-led BT	U/C	Na	Pre-tx expectancy	OCD symptom change (post-tx; 1, 3, 6 months follow-up)
Tolin, Diefenbach, and Gilliam (2011)	USA	RCT	34	Stepped-care BT; BT	36 h	Na	Depression	OCD symptom change; treatment cost
1) Van Oppen et al. (1995)/2) Van Balkom et al. (1998)[a]	Netherlands	RCT(s)	1) 71 2) 117[b]	CT; BT; CT + fluvoxamine; BT + fluvoxamine	12 /11 h	WL	Pre-tx OCD severity; tx motivation; comorbid PD; age at illness onset; marital/relationship status; prior tx; employment status; OCD symptom duration; depression; psychiatric symptoms (SCL-90); referral source; gender; age; anxiety symptoms; OCD symptom subtype; N of Axis I comorbidities; rumination; impulses; precision; irrational beliefs; tx site; MDD	OCD severity/remission; response status; drop-out status; outcome (U/C)
Van Oppen et al. (2010)	Netherlands	RCT	118	Self-/therapist-led BT (experienced vs. inexperienced therapist)	6.5/16.5 h	Na	Gender; education level; OCD symptom duration; comorbid Axis I disorders; prior tx; employment status; marital status; severity of OCD/ depression; anxiety-related discomfort	Drop-out status
Vogel, Stiles, and Götestam (2004)	Norway	RCT	37	ERP + CT; ERP + relaxation	24 h	WL	Age; gender; marital status; pre-tx OCD severity; tx motivation; tx expectancy; comorbid GAD/panic disorder; Cluster A/B/C PD	OCD severity; drop-out status
Whittal et al. (2005)	Canada	RCT	83	BT; CBT	12 h	Na	Pre-tx severity of OCD/depression; tx credibility; education level; employment status; disability status; marital status; age; ethnicity; medication use; comorbid Axis I disorders; age at illness onset; referral source; gender; OCD symptom duration	Drop-out status; tx refusal; OCD severity
Whittal et al. (2010)	Canada	RCT	73	CBT; stress management training	13 h	WL	Medication use; depression; referral source; psychosocial stressors (U/C); educational level	Tx effect (U/C); drop-out status

Note. AD = antidepressant; BD = Bipolar Disorder; BAT = Behavioral Avoidance Test; BT = behavior therapy; CGI = Clinical Global Impression; CT = cognitive therapy; CBT = cognitive behavior therapy; ERP = exposure and response prevention; GAD = Generalized Anxiety Disorder; h = hours; HW = homework; IQ = intelligence quotient; LOI = Leyton Obsessional Inventory; MI = motivational interviewing; MDD = Major Depressive Disorder; Min. = minimum; MOCI = Maudsley Obsessional Compulsive Inventory; N = number; Na = not applicable; OC = obsessive compulsive; OCD = Obsessive–Compulsive Disorder; OCPD = Obsessive Compulsive Personality Disorder; PD = Personality Disorder; pre-tx = pre-treatment; PTSD = Post-Traumatic Stress Disorder; QoL = quality of life; RCT = randomized controlled trial; relax. = relaxation; SCL-90 = Symptom Checklist-90; (S)SRI = (selective) serotonin reuptake inhibitor; tx = treatment; U/C = unclear; WL = waitlist.

[a] Predictors assessed for combined dataset from both trials.

[b] Approximately 50% of participants took part in both trials.

3.4. Box-score review and quality assessment

The following paragraphs synthesize the existing evidence of predictor effects, the quality of predictor analyses, and overall risk of bias.

3.4.1. Predictors/moderators included in the box-score analysis

Seventeen trials reported the effect of overall baseline OCD symptom severity on outcome. Two studies assessed the effect of obsessive or compulsive symptoms only (Cottraux et al., 1990; Keijsers et al., 1995) and one study assessed the effect of a measure of general illness severity, derived through a factor analysis (Marks et al., 1988). A further study reported conflicting data within respective trial reports (Simpson et al., 2008); these studies were excluded from the box-score analysis. Nineteen trials reported the effect of baseline depression on treatment outcome; of these, one study failed to report data to judge the direction of the relationship and was therefore omitted from the box-score analysis (Greist et al., 2002). The relationship between age and treatment outcome was assessed in 14 of the included trials; one study reported conflicting findings in respective trial reports and was therefore excluded (Vogel et al., 2004). Gender was assessed in 13 studies; one trial with conflicting reports of predictor effects was excluded (Vogel et al., 2004). The effect of OCD symptom content was assessed in ten trials; one of these was excluded from the box-score analysis as the symptom clusters assessed were derived from a factor analysis and did not correspond with validated OCD symptom subtypes (Marks et al., 1988).

Table 3
Comparisons of study characteristics in trials with and without predictors of outcome.

Study characteristic	All trials N/Mean (SD)	Trials with predictor analyses N/Mean (SD)	Trials without predictor analyses N/Mean (SD)	Comparison
Year of publication	31	14	16	$\chi 2$ (1,69) = .974,
1984–2001	38	24	15	p = .324
2002–2012				
Country	21	13	8	$\chi 2$ (1,69) = .111,
USA/Canada	48	25	23	p = .739
Other				
Risk of bias	13	11	2	$\chi 2$ (1,69) = 4.275,
Low	56	27	29	p = .039
Uncertain/high				
Sample size	54.5 (36.7)	65 (42.7)	42.7 (24.1)	U = 372.0, p = .028

Consistent significant associations between predictors and treatment outcome were rare among the commonly assessed variables (Table 4). For many predictors, including OCD symptom severity, OCD symptom subtype, OCD illness duration, age, gender, marital/relationship status,

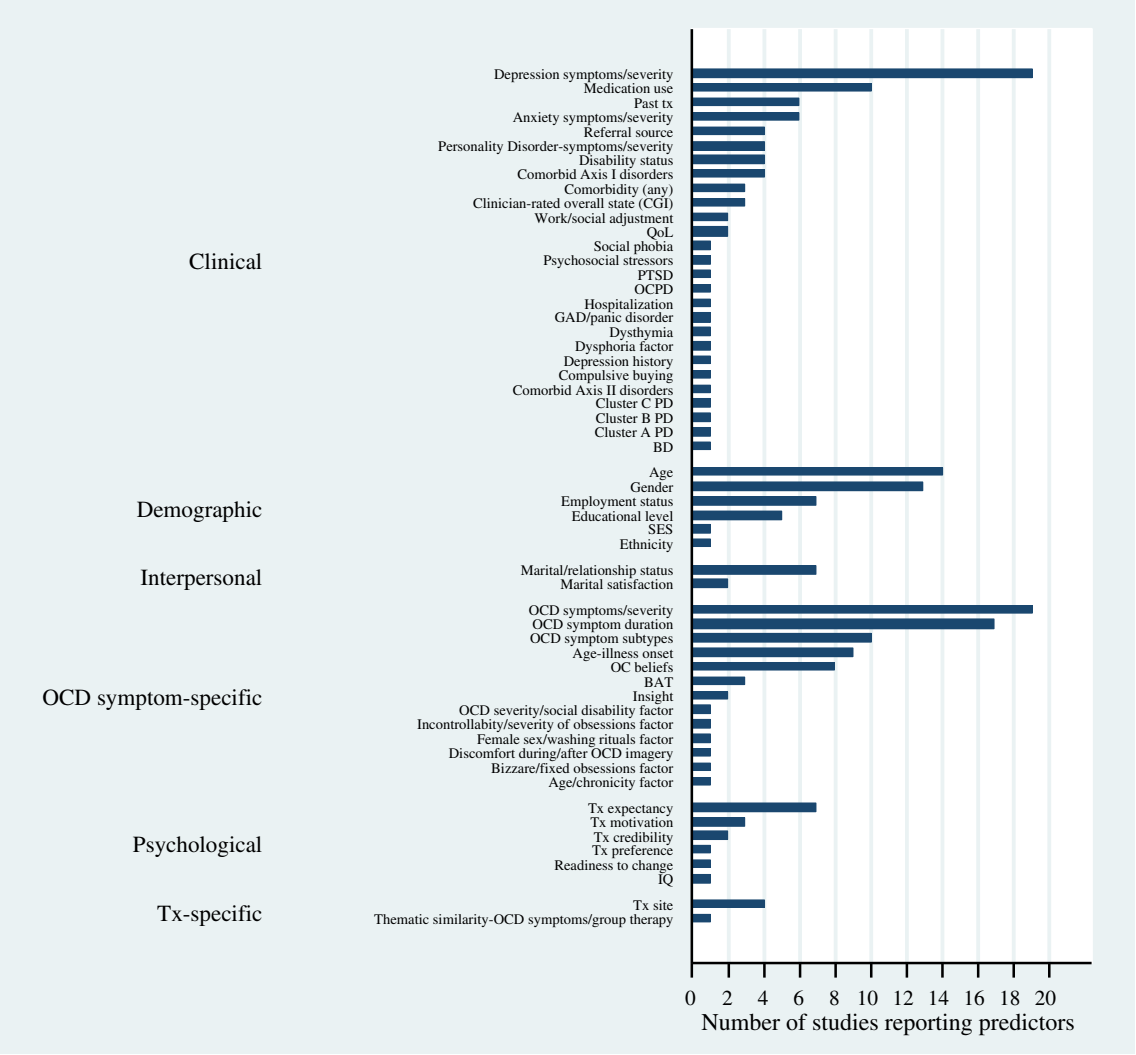

Fig. 2. Horizontal bar graph of predictors assessed across trials by predictor category. Note. BAT = behavioral avoidance test; BD = Bipolar Disorder; CGI = Clinical Global Impression; GAD = Generalized Anxiety Disorder; IQ = intelligence quotient; OC = obsessive compulsive; OCD = Obsessive–Compulsive Disorder; OCPD = Obsessive Compulsive Personality Disorder; PD = Personality Disorder; PTSD = Post-Traumatic Stress Disorder; QoL = quality of life; SES = socio-economic status; Tx = treatment.

Table 4
Box-score review of predictor effects on OCD treatment outcome and quality assessment.

Predictor effects on outcome				Quality assessment for combined analyses of specific predictors				
Predictor/moderator	Test of interaction	Predictor: All	Predictor: Subgroup	Valid measure of predictor	Predictor tested prerandomization	<5 predictors assessed	A priori hypothesis	Direct test of interaction
				Y	Y	Y	Y	Y
OCD symptom severity	+(SMT) 0(ERP)	+ + + + 0 0 0 0 0 0 0	0 0 0	100%	87%	7%	7%	7%
Comorbid depression severity	0 0 0	+ 0 0 0 0 0 0 0 0 0 0 ?[a]	+ 0 0	100%	84%	11%	0%	16%
OCD illness duration		+ 0 0 0 0 0 0 0 0 0 0 0 0 0 ?[a]	+ +(IG) 0(CG) 0	Na	Na	18%	0%	6%
Patient age	0 0	+ + + 0 0 0 0 0 0 0 0		Na	Na	14%	0%	14%
Patient gender		0 0 0 0 0 0 0 0		Na	Na	0%	0%	15%
Male	0	+						
Female	+	0						
Patient medication use	0 0	− 0 0 0 0 0	0 0	Na	Na	20%	0%	20%
OCD symptom subtypes		+ 0 0	+(cCBT)	100%	93%	0%	27%	13%
Washing vs. checking	0							
Aggressive/contamination obsessions			0					
Cleaning/contamination		+ 0	+(FLV) 0(ERP)					
Checking		+ 0						
Hoarding[c]	0	+ + +						
Sexual/religious		+						
Ordering/symmetry		+						
Slowness		+						
Age at OCD onset		0 0 0 0 0 0 0	0 0	Na	Na	0%	0%	0%
Patient employment status	0	− − 0 0 0 0		Na	Na	0%	0%	14%
Patient marital/relationship status	0	− − 0 0 0 0		Na	Na	0%	0%	14%
OCD tx expectancy		− 0 0 0 0 ?[b]	0	0%	29%	29%	0%	0%
Past OCD tx	+	0 0 0 0 0		Na	Na	0%	17%	17%
OC beliefs		0 0		94%	100%	0%	25%	6%
Thought–Action Fusion		+(Moral subscale only) 0						
Obsession-related beliefs/ thoughts (IBRO/OTC)		0	+(CT) 0(ERP)					
Responsibility		0 0	0					
Intrusive thought interpretation (ITIQ)			0					
Intensity, rigidity, insight (OVIS)			0					
Insight	0	0						
Irrational beliefs (IBI)		0						
Patient educational level	0	0 0 0 0		Na	Na	0%	0%	20%
Comorbid anxiety severity		+ 0 0 0 0	+	100%	83%	0%	0%	17%
Total quality score				82.3%	79.3%	6.6%	5.1%	11.9%

Note. + = positive relationship with outcome ($p < 0.05$); − = negative relationship with outcome ($p < 0.05$); 0 = no relationship with outcome; cCBT = computerized cognitive behavior therapy; CG = control group; CT = cognitive therapy; ERP = exposure and response prevention; FLV = Fluvoxamine; IBRO = Inventory of Beliefs Related to Obsessions; IG = intervention group; ITIQ = Intrusive Thoughts and Their Interpretations Questionnaire; OC = obsessive compulsive; OCD = Obsessive–Compulsive Disorder; OTC = Obsessive Thoughts Checklist; OVIS = Overvalued Ideas Scale; SMT = stress management training; Y = criterion met; Na = not applicable. ?[a], different predictor effects reported for measures of Compulsive Behavior and Obsessive Fear, respectively. ?[b], inconsistent predictor effects reported - no relationship: Total Targets Improvement, Social Adjustment, BDI, HAM-D; negative relationship: Total Rituals, Total Obsessions. [c]As noted by a referee, hoarding is now considered a separate disorder in the DSM-5; herein it has been categorized as an OCD symptom subtype, as it was classified as such when this review was completed.

treatment expectancy, employment status, past treatment, and severity of depression and anxiety symptoms, a number of studies reported significant associations while others showed no relationship with outcome.

In relation to the broad category of OCD symptom subtypes, hoarding symptoms were significantly related to worse treatment outcome in three of four relevant trials (Greist et al., 2002; Jaurietta et al., 2008; Simpson et al., 2010). Outcomes including depressive and anxiety symptoms, OCD severity, and treatment continuation were negatively affected by hoarding symptoms, although a test of interaction showed no association (Maher et al., 2010). One third of relevant trials supported an association between greater OCD and anxiety severity and worse treatment outcome, defined in terms of post-treatment Y–BOCS scores and drop-out status.

In relation to symptom subtypes other than hoarding, there was some evidence to suggest that the category of symptom subtype may play a role in response to treatment; although findings of specific symptom subtypes were commonly based on single studies and their reliability therefore difficult to judge. Non-clinical variables, employment status and marital/relationship status, showed an association with outcome in approximately one third of included studies, with unemployed and single individuals experiencing a worse prognosis. For the predictors medication use, age of OCD onset, OCD related beliefs, and educational level, no significant associations were found in the overwhelming majority of relevant trials. Importantly, these findings need to be considered in light of the quality of predictor/moderator analyses as well as overall risk of bias of included studies.

3.4.2. Quality of predictor/moderator analyses

The quality of predictor analyses varied widely (Table 4). While most studies assessed predictors through validated measurement tools prior to randomization, the majority of trials reported >5 outcome predictors (mean = 8.2; SD = 8.27), few stated a priori hypotheses, specifying the anticipated direction of the predictor effect, or used a test of interaction to evaluate the predictor–outcome relationship. The one exception to this overall trend was treatment expectancy which was not assessed via a validated tool in any of the included trials and only in 29% of assessments was this variable measured prerandomization.

Table 5
Risk of bias of included studies.

Author/year	Sequence generation	Allocation concealment	Blinding-outcome assessors	Attrition <20%	Intention-to-treat analysis	Tx fidelity assessed	Manualized tx	High/uncertain/low risk of bias
Althaus et al. (2000)	?	?	?	+	?	−	−	Uncertain
Belotto-Silva et al. (2012)	−	+	+	−	+	−	−	Low
Valpato Cordioli et al. (2003)	+	+	+	+	+	−	+	Low
Cottraux et al. (1990)	?	?	+	−	−	−	−	Uncertain
Cottraux et al. (2001)	?	?	+	+	+	−	+	Uncertain
De Araujo et al. (1995)	?	?	+	+	−	−	−	Uncertain
Dreessen et al. (1997)	?	?	?	+	+	−	−	Uncertain
Emmelkamp et al. (1990)	?	?	?	+	−	−	−	Uncertain
Fineberg et al. (2005)	−	−	+	−	−	−	+	High
Foa et al. (1984)	?	?	+	+	−	−	−	Uncertain
Foa et al. (2005)	?	?	+	−	?	−	+	Uncertain
Freeston et al. (1997)	?	−	−	−	−	+	+	High
Greist et al. (2002)	?	?	−	+	+	+	−	Uncertain
Jaurietta et al. (2008)	+	+	−	+	+	−	+	Low
Jónsson et al. (2011)	?	+	−	−	?	−	−	Low
Keijsers et al. (1995)	?	?	?	?	−	−	−	Uncertain
Kenwright et al. (2005)	+	+	?	−	+	−	−	Low
Lakatos (1997)	?	?	?	−	−	+	+	Uncertain
Marks et al. (1988)	?	?	+	−	−	−	−	Uncertain
Marks et al. (2000)	+	+	?	+	−	−	−	Low
McLean et al. (2001)	?	+	+	−	+	+	+	Low
Meyer et al. (2010)	+	+	+	+	+	−	+	Low
Nakao et al. (2005)	?	?	+	+	−	−	?	Uncertain
Nakatani et al. (2005)	?	+	+	+	−	−	+	Uncertain
Rector et al. (2009)	?	?	+	−	+	+	+	Uncertain
Rowa et al. (2007)	?	?	+	−	−	−	+	Uncertain
Simpson et al. (2008)	+	+	+	+	−	+	+	Low
Simpson et al. (2010)	?	?	+	+	+	+	+	Uncertain
Sousa et al. (2006)	?	?	?	+	−	−	+	Uncertain
Tenneij et al. (2005)	?	?	+	+	+	+	+	Uncertain
Tolin et al. (2007)	+	+	+	+	+	−	+	Low
Tolin et al. (2011)	?	?	+	?	+	+	−	Uncertain
Van Balkom et al. (1998)	?	?	?	−	+	−	+	Uncertain
Van Oppen et al. (1995)	?	?	?	+	−	+	+	Uncertain
Van Oppen et al. (2010)	?	?	+	+	+	+	+	Uncertain
Vogel et al. (2004)	+	−	−	−	+	+	+	High
Whittal et al. (2005)	+	?	+	−	+	+	+	Uncertain
Whittal et al. (2010)	?	?	+	+	+	+	+	Uncertain

Note. Tx = treatment; + = criterion met; ? = unclear whether criterion met; − = criterion not met.

3.4.3. Risk of bias

A meta-analysis indicated no association between risk of bias (dichotomized score of allocation concealment) and study effect size (SMD = −0.93; 95% CI = −1.08 to −0.77 versus SMD = −1.12; 95% CI = −1.46 to −0.77 in trials with uncertain/high and low risk of bias respectively). The assessment of risk of bias of included studies is presented in Table 5. While over one half (61%) of included studies reported the manualization of treatment, a formal assessment of intervention fidelity through the review and rating of recorded sessions was conducted in only 37% of trials. Twenty four percent of included studies reported an adequate method of sequence generation as outlined in Cochrane criteria (Higgins & Altman, 2008), while 68% of studies lacked the necessary information to judge the adequacy of the method of randomization. Similarly, an appropriate method of allocation concealment was reported in 29% of trials and 63% of studies failed to specify whether an appropriate method was employed to conceal group allocation. Outcome assessors were blinded in 61% of trials and around one half of included studies (51%) reported conducting intention-to-treat analyses. Rates of attrition below 20% at post-treatment were reported in 55% of trials.

4. Discussion

4.1. Main findings

This systematic review synthesized the existing evidence concerning baseline predictors/moderators of response to psychological therapies in OCD. There was considerable overlap in the most common predictors across trials; trial authors regularly reported on the relationship between OCD symptom-specific, clinical, and demographic variables and treatment outcome. Interestingly, psychological variables received relatively little attention as potential moderators of outcome (although many may have been assessed as mediators of treatment effect and would have been excluded from the present review). While the relevant literature generated relatively varied and ambiguous findings, a number of variables were relatively consistently related to outcome. The box-score analysis provided some support that hoarding pathology, increased anxiety and OCD symptom severity, certain OCD symptom subtypes, unemployment, and being single/not married are associated with worse treatment outcome. Variables which failed to show an association with outcome in the great majority of relevant trials include medication use, age of OCD onset, OCD related beliefs, and educational level.

Reporting of predictors was not associated with age of the study, country of publication, or treatment effect. The lack of association with treatment effect is important; it suggests that authors are not only conducting subgroup analyses when the primary analyses fail to show significant effects. The associations found between reporting and study size and risk of bias would suggest that the existing findings are based on the stronger studies in the literature, which would support the reliability of the analyses. However, the overwhelming majority of trials were judged as at significant risk of bias calling for caution in the interpretation of these findings generally. Moreover, sample sizes were generally far from satisfactory to ensure statistical power in the context of predictor/moderator analyses (Brookes et al., 2004).

Considering the lack of adequate statistical power and the suboptimal quality of predictor analyses in much of the included literature, we

adopted a conservative approach to interpreting the presented research evidence. Given the need for rigorous findings in applying predictor evidence in clinical practice, we consider a test of interaction to be the gold standard; this approach is more sensitive to the comparative benefit of treatment in different groups in a trial and is thus vital for informing evidence based decisions about tailoring treatment. However, we acknowledge that interaction analyses require larger sample sizes than overall regression/correlation models, which are more likely to show significant effects, in particular in the context of relatively small studies (e.g. Brookes et al., 2004). In light of the small sample sizes in the current literature, views may differ as to the utility of interaction tests in this context. Hence, while we would call for caution in the interpretation of associations derived from analyses of overall and subgroup-specific effects, the evidence on some factors (for example on hoarding pathology) may be interpreted as consistent when adopting a less conservative stance.

4.2. Comparison with the wider literature

The box-score approach, adopted in the current study, was used to overcome caveats in the reporting of relevant statistical data. It differs from the narrative review of Keeley et al. (2008), although both methods have advantages and disadvantages. The box-score provides basic quantification of patterns in the literature, and facilitates consideration of study quality and other factors as confounders of those patterns. The narrative review may in some ways be less reliable, as the methods of analysis and synthesis are less transparent and standardized and potentially more open to subjective judgment. However, the box-score model takes a necessarily coarse approach to the literature. This form of analysis may therefore be less able to take account of more sophisticated aspects of the studies, such as the context in which they were conducted and subtle clinical and therapeutic issues which are better captured in a narrative synthesis. Given those differences, there is a natural interest in the comparability of the results.

There was overlap between those variables identified as potential predictors in the current review and that conducted by Keeley et al. (2008). Common variables included symptom subtype and OCD symptom severity. Keeley et al. (2008) found the nature of the familial environment to be predictive of treatment response; however, most relevant studies were excluded from the current review as they did not meet our inclusion criteria in terms of study design or sample characteristics. Studies included in both reviews showed inconsistent findings regarding the role of family factors in treatment outcome for OCD (Emmelkamp et al., 1990; Keijsers, Hoogduin, & Schaap, 1994; Mehta, 1990). However, in the current review marital/relationship status predicted outcome in 29% of trials where it was assessed, further suggesting that interpersonal and familial factors may be important. Also in line with Keeley et al. (2008) the present review shows that a number of commonly assessed outcome predictors, including OCD illness duration and comorbid depression, are inconsistent in terms of their relationship with outcome. Similarly, the reviews agree that findings relating to gender, education level, and treatment expectations are largely non-significant.

However, there are a number of discrepancies between Keeley et al.'s findings (2008) and those of the present review. The previous review reported relative consistency in the relationship between certain symptom subtypes and major depressive disorder (MDD) and worse treatment outcome. Although, the same predictors emerged in the present review, we arrive at more tentative conclusions. Few studies assessed the predictive role of specific symptom subtypes. While sexual/religious, ordering/symmetry, cleaning/contamination symptom content, and obsessive–compulsive slowness were found to be significantly associated with worse outcome, based on single study reports, these findings cannot be considered reliable. Further research in the context of adequately powered randomized trials, using gold standard methods in the assessment of predictors is needed to give further support to existing findings.

The evidence for hoarding symptoms is stronger. However, acknowledging the phenomenological differences between OCD and hoarding, in the classification of disorders in the DSM-5, hoarding is no longer considered an OCD symptom dimension (Steketee, 2010). Its relevance as a predictor of outcome in the context of psychological treatment for OCD is nonetheless interesting. Differences between the two disorders have resulted in the development of treatments specifically adapted for hoarding pathology (Muroff, Bariotis, & Steketee, 2010; Tolin et al., 2007). Even so, the implementation of adequate interventions appears to lag behind and hoarding disorder continues to be commonly treated through standard ERP interventions, which fail to meet the needs of individuals with this condition. Furthermore, hoarding may occur comorbidly with OCD and its effect on treatment response in this context may be worth investigating. Lastly, the two reviews disagreed with regard to the role of MDD. Citing Abramowitz(2004), Keeley et al. (2008) state that patients with comorbid MDD may benefit from additional cognitive therapy components aimed at ameliorating related thinking-patterns. Differences in inclusion criteria (study randomization) of the respective reviews meant that we failed to find empirical evidence in support of such recommendations. Those trials reporting on the relationship between MDD and outcome in the present review (Belotto-Silva et al., 2012; Valpato Cordioli et al., 2003) reported no significant associations.

The finding that those trials which assessed predictors of outcome reported greater sample sizes than trials which failed to conduct such analyses is in line with evidence reported by Sun et al. (2011). Discrepancies between the findings of the current review and that by Sun et al. (2011) may represent significant differences related to the clinical areas.

4.3. Implications for research and practice

While outcome predictors are commonly reported in the field of mental health research, the quality of analyses is less than optimal (e.g. Assmann, Pocock, Enos, & Kasten, 2000; Sun et al., 2012). The exploratory analysis of predictor effects can open early avenues for future research investigation (e.g. Pincus et al., 2011); however, the misrepresentation of exploratory findings as confirmatory may be highly problematic (e.g. Kraemer et al., 2002). The quality of analyses reviewed here was not optimal. The integration of existing quality criteria into routine research practice could assist in the rapid development of an evidence base to inform the personalization of interventions for OCD. Improvements in methodology may prove particularly useful in the assessment of those factors for which existing findings already show promising results.

In concordance with Keeley et al. (2008), we found significant differences in the assessment of predictors, outcome, and the definition of treatment outcome, as well as variation in intervention types, which make the interpretation of study findings very difficult. Nonetheless, Keeley et al. (2008) report some implications of their findings for the psychological care of OCD patients. The authors suggest that efforts ought to be devoted to developing targeted treatments for respective OCD symptom dimensions. While our findings point to the significance of symptom subtype in treatment outcome, inconsistencies in the categorization of OCD symptom dimensions and the fact that the current findings are commonly based on single studies call their reliability into question. Using the opportunity of well-conducted psychotherapy trials in OCD to assess the predictive role of specific symptom dimensions and the nature and quality of the familial environment may prove fruitful in optimizing the provision of mental health services by allowing for treatment to be tailored on the basis of empirical evidence. However, this said, clinical experience in support of such findings may also be used to justify appropriate personalization of treatment based on patient characteristics. In light of the distinction between hoarding pathology and OCD (Steketee, 2010), recent research efforts have been well placed to develop and assess suitable treatment techniques for hoarding symptoms (e.g. Muroff et al., 2010; Tolin et al., 2007); this review highlights the need to routinely implement these approaches.

Additionally, it may be of interest to investigate potential differences in the predictive role of hoarding disorder when comorbid with OCD versus in isolation. Moreover, the finding that marital/relationship status and employment status showed a likely association with outcome may indicate the significance of addressing circumstantial factors in therapy, which may otherwise detract from treatment and lessen its effectiveness. However, the successful implementation of such changes requires the careful consideration and planning of the analyses of third variables at all stages of the research process—including early planning of predictor analyses in the initial research stages, reporting of a priori hypotheses of anticipated effects, the adoption of adequate statistical analyses, and full reporting of analytic methods and resultant findings.

4.4. Strengths and limitations of the findings

This review used a rigorous approach to the identification of relevant trials. Moreover, all data was extracted independently by two reviewers, and reliability checks were conducted. By including foreign language studies and dissertations, the authors aimed to reduce the effect of publication bias, although unpublished research is likely underrepresented in the review. Assessing the impact of predictors on outcome through a meta-analytic approach was not possible for reasons outlined. The box-score approach, adopted in the current review, has known limitations, as the classification of effects in terms of statistical significance and direction fails to consider the *magnitude* of reported effects (Green & Hall, 1984) and may therefore result in taking an overly conservative stance to interpreting the evidence and wrongly accepting the null hypothesis. An additional issue is the lack of consensus on the interpretation of findings, in terms of the number and consistency of findings required to make clinical or policy recommendations. Green and Hall (1984) highlighted that box-score analyses can be misinterpreted if analysts fail to take into account how many significant results might be expected under the null hypothesis. They suggested that significant support could be assumed for predictors where around 30% of studies reported significant findings in the hypothesized direction (Green & Hall, 1984, p. 41). To reduce the risk of underestimating potential predictor effects, we assessed findings in line with the suggestions made by the authors (Green & Hall, 1984) and performed a balanced discussion of the presented evidence. Counting the proportion of studies reporting significant effects ignores the *quality* of the methodology underlying those analyses. A key strength of the current review was the comprehensive assessment of overall risk of bias and the quality of predictor analyses, thus allowing the reader to evaluate the credibility and reliability of reported predictor effects. There was significant variation in reporting of multiple and varying measurement time-points, forms of analyses, and measurement tools within and across trials, but we sought to achieve the greatest possible consistency in our extraction analyses.

5. Conclusion

While there is clearly a strong interest in predictors and moderators of outcome, and an increasing body of literature to guide the conduct of high quality research in this context (Brookes et al., 2001; Emsley, Dunn, & White, 2010; Pincus et al., 2011; Rothwell, 2005; Sun et al., 2012; Wang, Lagakos, Ware, Hunter, & Drazen, 2007), only few consistent findings can be drawn from the existing literature, highlighting the gap between best practice and its implementation in applied mental health research. It is promising that authors commonly report analyses of predictor effects; however, the potential of such analyses has not been maximized, as analyses only rarely meet published quality criteria (Pincus et al., 2011; Sun et al., 2012). Considering the significant applied role of these analyses in personalized patient care and the optimal use of limited healthcare resources, it appears surprising that this domain has not received more mention in research guidelines. In line with Emsley et al. (2010) we conclude that the theoretical and methodological foundation has been laid for conducting empirically sound research into prediction effects, and the principal focus ought to be on applying this knowledge.

Conflict of interest

The authors declare no conflict of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.cpr.2013.08.008.

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