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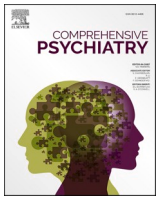

The relationship between cognitive phenotypes of compulsivity and impulsivity and clinical variables in obsessive-compulsive disorder: A systematic review and Meta-analysis

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ABSTRACT

Background: This systematic review and meta-analysis explored the relationship between cognitive phenotypes of compulsivity and impulsivity and clinical variables in obsessive-compulsive disorder (OCD).
Methods: We searched Pubmed, Scopus, Cochrane Library and PsychINFO databases until February 2023 for studies comparing patients with OCD and healthy controls on cognitive tests of compulsivity and impulsivity. The study followed PRISMA guidelines and was pre-registered on PROSPERO (CRD42021299017).
Results: Meta-analyses of 112 studies involving 8313 participants (4289 patients with OCD and 4024 healthy controls) identified significant impairments in compulsivity ($g = -0.58$, [95%CI -0.68, −0.47]; $k = 76$) and impulsivity ($g = -0.48$, [95%CI -0.57, −0.38]; $k = 63$); no significant difference between impairments. Medication use and comorbid psychiatric disorders were not significantly related to impairments. No associations were revealed with OCD severity, depression/anxiety, or illness duration.
Conclusion: Cognitive phenotypes of compulsivity and impulsivity in patients with OCD appear to be orthogonal to clinical variables, including severity of OCD symptomatology. Their clinical impact is poorly understood and may require different clinical assessment tools and interventions.

1. Introduction

Obsessive-Compulsive Disorder (OCD) is a serious neuropsychiatric disorder characterised by a failure to control disabling repetitive, stereotyped behaviours (compulsions) and distressing, intrusive thoughts or feelings (obsessions) [10]. OCD presents as a phenotypically heterogeneous disorder with differing symptomatic presentations, including expression of a broad range of obsessions and compulsions [118]. and clinical courses [113,187].

OCD may therefore be better delineated by identifying stable latent cognitive phenotypes [55]. These cognitive factors represent less visible, but nevertheless measurable manifestations of underlying neurobiology (changes in the structure, function or integrity of the underpinning neural correlates of OCD); and are thought to occupy an intermediate role between the genetic or environmental origins of the disorder and the expressed psychopathology. As they lie closer to the biological determinants of OCD than the expressed symptoms, latent cognitive phenotypes are theoretically likely to be subject to less inter-individual variability and therefore offer greater reliability for investigating the neurobiology of OCD [41,68,72].

Latent cognitive phenotypes of OCD have been subject to considerable study over at least 15 years [44]. Converging evidence implicates in the origins of OCD a broad tendency to persist at repeating stereotyped maladaptive actions, as well as a loss of inhibitory control over the initiation of thoughts or actions. These latent cognitive phenotypes may respectively be termed compulsivity and impulsivity [67,70]. Performance deficits on specific cognitive tasks involving reduced capacity for flexible contingency related attentional set-shifting or behavioural perseveration (which may be considered compulsive: [66,123]) or heightened disinhibition of motor behaviours including disadvantageous decision-making (which may be considered impulsive: [53,136])), have been relatively consistently identified in patients with

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OCD [68,72].

In studies of OCD and related disorders, compulsivity is typically investigated using tests of attentional set-shifting such as the Wisconsin Card Sorting Test [134] and the Intra-Extra Dimensional Set-Shifting Task (IED; [154]). On such tasks, patients with OCD and their unaffected first degree relatives show impaired ability to flexibly adjust their responding to aspects of stimuli and erroneously continue to respond (perseverate) to them after the rule to do so has changed [6,44,163]. In contrast, impulsivity in OCD has been fractionated into two broadly separate subtypes: i) motor impulsivity, representing difficulty inhibiting a pre-programmed (pre-potent) motor action, reliably measured using the Stop-Signal Task [15]; and ii) Decision-making impulsivity, manifested as disadvantageous decision making and delay discounting, reliably measured using the Cambridge Gambling Task [168], Iowa Gambling Task [19] or other similar tasks probing the discounting effect of a temporal delay on the value of a reward [70,72,135]. Whereas patients with OCD and their unaffected first degree relatives show impairment on tests of motor impulsivity [44,126,131], greater uncertainty exists about the degree or consistency of decision-making impulsivity, as some studies reported an association [52,84,148] whilst others have not [8,9].

As these latent phenotypes have been documented in individuals at high genetic or environmental risk for OCD, including unaffected first-degree relatives, they may be viewed as vulnerability traits [43,45,81,201]. Emerging evidence also suggests that similar latent phenotypes can be found in association with other diagnoses in the family of obsessive-compulsive and related disorders (OCRDs) [54,75], though these other disorders have been subjected to less study. Deficits on the Stop Signal Task have however been reported in patients with body dysmorphic disorder [99], trichotillomania [151], binge eating disorder [82], hoarding disorder [139] and skin picking disorder [23]. Similarly, inflexible responding on the IED has been reported in association with obsessive-compulsive personality disorder [71], anorexia nervosa [130], body dysmorphic disorder [99], and schizo-obsessive disorder [157].

Although an implied polarity might be seen in the multifaceted descriptions of impulsivity (e.g., hasty, rash, and risk-taking) and compulsivity (e.g., rigid, controlling, and risk-averse), they commonly co-occur in patients with OCD. The relative contribution of impulsive and compulsive responding however may vary within an individual across time and when these disorders co-occur, they may also be more severe [11,101].

As the response to conventional treatment is so often unsuccessful [69], latent compulsive or impulsive phenotypes could provide a theoretical basis for developing new interventional targets for OCD and by extension OCRDs. As objective biomarkers of increased illness vulnerability, they may also constitute clinically relevant screening aids to enable early preventative intervention, before symptoms become severe, chronic and disabling [46,73,74,170,208]. Further, as key determinants of executive functioning, they may serve as critical markers of functional outcomes. A brain imaging study by members of our group [198], demonstrated that not all patients with an OCD diagnosis exhibited inflexible set-shifting on the IED, but those that did showed significant fronto-striatal connectivity changes. Thus, heterogeneity in the expression of latent phenotypes is likely to emerge among patients with OCD. Identifying who does and does not display these latent phenotypes may have implications for clinical practice, acting as a platform for precision medicine and informing the treatment approach with greater predictive accuracy, resulting in better clinical outcomes. Nonetheless, many studies looking for cognitive latent phenotypes in OCD have produced inconsistent findings, resulting in uncertainty and controversy. Some of this inconsistency may be attributable to the diversity among tests of compulsivity and impulsivity used, some of which may not be sensitive enough to the impairments present in OCD. Some authors have called for improved precision and consistency in the use of tasks, to enable clarification of the specific cognitive latent phenotypes of OCD and OCRDs [47]. Others have questioned this whole area of research [103], or have proposed that the findings in OCD represent non-specific cognitive deficits common to many or all mental disorders and are thus of little or no predictive value [7]. We respond to the controversies in this area by applying meta-analysis to investigate the following research questions:

- Do patients with OCD perform significantly worse than healthy controls on cognitive tests assessing compulsive and impulsive responses?
- Do patients with OCD show a difference in the magnitude of deficit on tests of motor impulsivity compared with decision-making impulsivity?

Comorbid psychiatric disorders such as depression interfere with performance across a wide range of cognitive tasks through non-specific behavioural effects [137,144]. Comorbidity in OCD is prevalent, and up to 60% percent of patients with OCD show some signs of depressive symptomatology [132,133,138,159,160,166], which is commonly believed to be a secondary phenomenon [12,95,140]. Nevertheless, the potential impact of depression and other common comorbidities such as generalised anxiety disorder [14] on neurocognitive performance in OCD is not well understood.

We therefore aim to address an additional research question:

- Are the effect sizes for compulsivity and impulsivity impacted by the presence of clinical variables such as OCD symptomatology, depression, anxiety, and duration of illness?

2. Methods

2.1. Design search strategy

The protocol for this systematic review and meta-analysis was pre-registered at the International Prospective Register of Systematic Reviews: PROSPERO 2021 CRD42021299017 (Available from: https://www.crd.york.ac.uk/prospero/display_record.php?ID=CRD42021299017). The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines were followed in the reporting of this review [156]. Four databases were used in this review: PsychINFO, Cochrane Library, Pubmed, and Scopus. Searches were conducted from the earliest timepoint of each search engine up until December 2022.

The searches employed the following combination of key terms: “Obsessive Compulsive Disorder” OR “OCD” AND Impulsiv* OR Compulsiv* AND Transdiagno* OR phenotyp* AND Neuro* OR Cogniti*.

2.2. Study selection

Following the removal of duplicate publications, the titles of the search results were reviewed with irrelevant studies being removed. The remaining papers were downloaded from their respective databases and uploaded to the Rayyan platform for systematic reviews [153]. Following this, the abstracts of the remaining corpus were screened, and irrelevant abstracts excluded. The full texts for all remaining papers were then scrutinised according to our eligibility criteria (listed below). The reasons for exclusions are outlined in the PRISMA flowchart (see Fig. 1). The studies meeting inclusion criteria were separated into neurocognitive tests and self- and clinician-report measures. In the instance of a discrepancy concerning the potential inclusion of a study, this was discussed among the research team in which the inclusion or exclusion was determined.

In accordance with Fineberg et al.’s [68] subdivision of neurocognitive tasks for impulsivity and compulsivity, performance on the Wisconsin Card Sorting Test and the Intra Extradimensional Set-Shifting Task were prioritised as measures of compulsive responding, as these tasks are validated for the assessment of cognitive inflexibility, a key

factor inherent within compulsive behaviours. While performance on the Stop-Signal Task and the Cambridge Gambling Task were prioritised as measures of motor impulsivity and decision-making impulsivity respectively, inclusion of a broader range of impulsive neurocognitive task was employed because of the greater uncertainty about the role of decision-making impulsivity in obsessive-compulsive phenomena. Additional measures included: the Iowa Gambling Task [19], and the Temporal Discounting Task [173] for decision-making impulsivity and the Go/No-Go Task [79], and Conner's Continuous Performance Task-II [51] for motor impulsivity.

2.3. Eligibility criteria

a. The studies tested participants with a current primary diagnosis of OCD using a structured diagnostic method such as the DSM-5 or the ICD-10 (and earlier versions).

b. Neuropsychological testing of compulsivity (such as the Intra-Extra Dimensional Set-Shifting Task and the Wisconsin Card Sorting Test) or impulsivity (tasks such as the Cambridge Gambling Task and the Stop Signal Task).

c. The studies employed a sample of adults or adolescents (14+ years of age).

d. The studies to be written in the English language.

2.4. Data extraction

The final set of studies to fulfil all inclusion criteria were tabulated within a Microsoft Excel spreadsheet. The primary outcome data comprised task performance on either impulsive or compulsive neurocognitive tasks, or in some cases both should a study employ tasks assessing both latent phenotypes together. The relevant tests of impulsivity and compulsivity often derive multiple outcome metrics. For compulsivity, perseverative errors were the primary metric for the WCST and extra-dimensional errors for the IED. For impulsivity, the quality of decision-making metric was extracted from studies using the CGT, net score from studies using the IGT, and the Stop Signal Reaction Time for the SST. Other impulsive neurocognitive tasks such as the Go/No-Go task or the Conner's continuous task performance-II were too few to substantiate a definitive extractible metric, and thus the metric extracted was dependant on individual study descriptions of primary outcome measures. When uniformity of test metric could not be achieved across studies (e.g., perseverative errors was not available for the WCST) an alternative metric or total score metric was sought and substituted (e.g., % of perseverative errors). For further details on extracted outcome metrics, see Table 1 for study features.

Secondary data was tabulated separately. This consisted of clinician-rated obsessive-compulsive symptoms in addition to any depressive or anxious symptom scores as measured by the Yale-Brown Obsessive-Compulsive scale (Y-BOCs) [80], the Hamilton Anxiety Rating scale (HAR: [88]), and the Hamilton Depression Rating scale (HDR: [89]). Moderator variables were also extracted and included: age of sample, proportions of males and females per sample, years in education, intelligence (IQ), psychiatric or medical/physical comorbidities, and duration of illness.

Once complete, the data was cleaned of all non-numerical information according to the Data Extraction for Complex Meta-Analysis, DECiMAL [158]. This consisted of assigning information with a numerical value which was noted in a glossary; Impulsivity was assigned a value of 1 and Compulsivity was assigned a value of 2. The same followed for the associated neurocognitive tasks e.g., CGT =1, SST = 2 for impulsive measures, and WCST =1, IED =2 for compulsive measures.

2.5. Study quality

The quality of the included studies was assessed using the Appraisal tool for Cross-Sectional Studies, AXIS checklist [61] which is a checklist tool developed to assess quality for cross-sectional studies. The AXIS contains 20 items that assess reporting quality, study design and possible risk of bias. Seven questions assess reporting quality (items: 1, 4, 10, 11, 12, 16, and 18), seven relate to study design quality (items: 2, 3, 5, 8, 17, 19 And 20) and six for possible biases in the study (items: 6, 7, 9, 13, 14, and 15). An assessor is to comment *Yes, No, or Do Not Know*. The checklist also asks whether the interpretation of results may have been influenced by a funding source or a conflict of interest.

2.6. Analysis

Comprehensive Meta-Analysis V3 was used for the analysis of results in this review. Effect sizes were calculated using Hedge's *g* for a random-effects model. Following Cohen's convention, an effect size of 0.2 was considered small, 0.5 as moderate, and 0.8 as large. The mean scores, standard deviation and sample sizes of both the patients with OCD and healthy control groups were used to calculate Hedge's *g*. Some studies did not conform to this way of reporting task performance ([100,128,198]; and [26]). As Kang et al. [100], Vaghi et al. [198], and Bohon, Weinbach, and Lock [26] report between-groups differences, the effect sizes were estimated using group sizes and independent groups *t*-test values. Martoni et al. [128]'s effect size was calculated using group sizes and the *P* value. In cases where a study uses two or more tasks to explore the same phenotype, the mean pooled effect size across multiple tasks was taken to estimate the overall compulsivity [65,180] or impulsivity [44,76]. When a group within a study was employed more than once (e.g. the same control group compared to both an early onset OCD group or a late onset OCD group [109] or compared to a familial or sporadic OCD group [21], the control sample size was divided by the number of comparisons being made to avoid inflating the weighting of effect sizes.

Planned meta-regressions using a method of moments approach were used to assess various continuous moderator variables, including: age, proportion of females per sample, duration of illness, years in education, intelligence (IQ), study quality, symptom severity as measured by the Y-BOCS and levels of depressive and anxious phenomena as clinician-rated on the Hamilton Depression Rating Scale and the Hamilton Anxiety Rating Scale, respectively. For meta-regression and sub-group analyses, we followed recommendations of at least six to ten studies for a continuous variable and at least four studies per group for a categorical subgrouping variable [77,94].

The I^2 statistic was used to assess heterogeneity and for interpretation, we followed Cochrane guidance [94]: I^2 values between 0 and 40% were interpreted as might not be important, 30–60% as some moderate heterogeneity, 50–90% substantial heterogeneity, and 75–100% may present considerable heterogeneity. Funnel plots were observed for potential asymmetry in the assessment of small study effects and publication bias and if present, examined using the Duval and Tweedie's Trim and Fill method.

3. Results

3.1. Description of studies

Our searches identified a total of 2527 studies. Seventy duplicates were removed and following inspection of titles and abstracts, a further 2221 were excluded. Of the 236 papers subject to a full-text review, 124 failed to meet eligibility criteria. For details, see the PRISMA flowchart (Fig. 1). This left a total of 112 studies using neurocognitive tasks to assess compulsivity and impulsivity ($N = 8313$; 4289 patients with OCD and 4024 healthy controls). The main characteristics of the 112 included studies are presented in Table 1.

3.2. Narrative review

We identified 112 eligible studies, with 139 independent

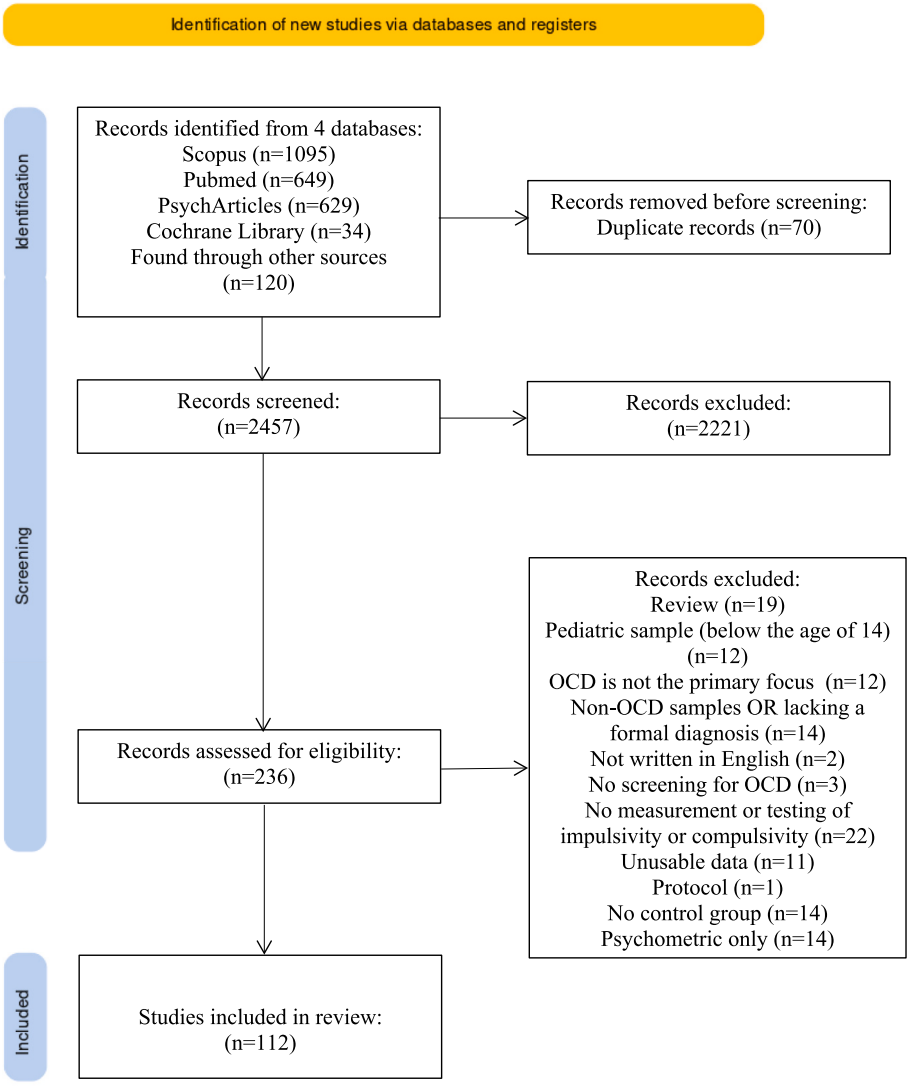

Fig. 1. PRISMA flowchart of 112 studies meeting inclusion criteria.

comparisons. For compulsivity, 59 studies used the Wisconsin Card Sorting Test [134], and 13 used the Intra-Extradimensional Set Shifting Task [154]; for impulsivity, 18 used the Stop Signal Task [15], and seven used the Cambridge Gambling Task [168]. Other impulsive-related neurocognitive tasks also included: 22 using the Iowa Gambling Task [19], 11 using the Go/No-Go task [79], one used the temporal discounting task [173], and three used the Conner's continuous performance task-II [51].

Four studies used two neurocognitive tasks to assess the same compulsivity or impulsivity phenotype; Fenger et al. [65] and Simpson et al. [180] used both the WCST and the IED to explore compulsivity whilst Chamberlain et al. [44] and Frydman et al. [76] used both the CGT and the SST to explore impulsivity. Similarly, Krishna et al. [112] and da Rocha et al. [58] used the IGT and the CCPT-II for impulsivity. The scores of patients with OCD and healthy controls were taken from both tasks and compiled into a single effect size to reflect a pooled value of compulsivity or impulsivity across tasks. 15 studies used neurocognitive tasks to assess both phenotypes e.g. the IED and the CGT ([20,32,36,38,44,102,105,112,186,210,211]; Saremi et al., 20,017; [48,109,127,129]). Although Lawrence et al. [116] and Blom et al. [22] use the IGT, we could not derive effect sizes from the data presented in these papers and they were excluded from the meta-analysis.

3.3. Impulsivity vs compulsivity

Random effects meta-analyses were performed for: 76 comparisons of patients with OCD and healthy controls on compulsive measures and 63 comparisons on impulsivity measures. Patients with OCD performed poorer on compulsive neurocognitive tasks than healthy controls ($g = -0.58$, [95%CI -0.68, −0.47]; k = 76, $p < .001$) with substantial heterogeneity ($I^2 = 70.09$, $p < .001$) (Fig. 2). Similarly, impulsive neurocognitive task revealed worse performance in patients with OCD than healthy controls ($g = -0.48$, [95%CI -0.57, −0.38]; k = 63, $p < .001$) (Fig. 3); substantial heterogeneity was observed across these effect sizes ($I^2 = 58.48$, $p < .001$). The effect sizes for compulsivity and impulsivity did not significantly differ ($Q = 1.99$, df = 1, $p = .16$).

Small study effect and potential publication bias was considered by examining funnel plots. The funnel plot for compulsive-related studies (Fig. 4) showed little or no asymmetry and the trim and fill method did not identify any potentially missing studies. For studies assessing impulsivity, the funnel plot similarly showed little evidence of asymmetry, and this was confirmed by a trim and fill analysis (Fig. 5).

We conducted an exploratory comparison of compulsivity tasks, which revealed no difference in the pooled effect sizes for the WCST (g = −0.61 [95%CI –0.74, −0.48; k = 59]) and IED tasks (g = −0.53 [95% CI –0.73, −0.34; k = 12) Q = 0.40, df = 1, $p = .53$.

Table 1
Characteristics of Included studies.

Study	Diagnosis	Mean age	Proportion female	OCD (n)	Control (n)	Test	Measure
[92]	DSM-III	33.30	46.67	15	15	WCST	Perseveration
[30]	DSM-III	f-OCD 40.60 s-OCD 32.70	Not stated	7 13	16	WCST	Perseverative responses
[98]	DSM-III	36.90	37.50	16	16	WCST	Perseverative responses
[78]	DSM-III-R	30.30	43.48	23	27	WCST	Perseverative errors
[1]	DSM-III-R	30.90	42.42	33	33	WCST	Perseverative errors
[2]	DSM-III-R	29.50	44.00	25	25	WCST	Perseverative errors
[86]	DSM-III-R	34.80	100.00	15	15	WCST	% of perseverative errors
[200]	DSM-III	36.10	57.50	40	22	IED	ED errors
[3]	DSM-III-R	30.00	31.67	60	30	WCST	Perseverative errors
[122]	DSM-III-R	38.00	47.37	19	19	WCST	Perseverative errors
[162]	DSM-IV	40.60	66.70	30	30	IED	EDS trial score
[152]	ICD-10	24.06	30.00	19	19	WCST	Perseverative errors
[17]	DSM-IV	30.06	40.00	20	31	WCST	% of perseverative errors
[141]	DSM-IV	33.22	55.56	36	36	WCST	Perseveration
[176]	SCID-DSM-III	25.80	54.20	18	24	WCST	Perseverative errors
[37]	DSM-IV	33.70	47.00	34	34	IGT	Ad vs disad deck
[106]	DSM-IV	29.82	30.77	39	31	WCST	Perseverative errors
[142]	DSM-IV	31.6	56.00	25	70	WCST	Perseveration
[36]	DSM-IV	30.50	50.80	67	56	IGT WCST	No. of disad deck Perseverative errors
[90]	DSM-IV	39.40	52.00	25	11	Go/no-go	Block performance
[93]	DSM-IV	41.20	33.30	12	12	Go/no-go	Commission errors
[107]	SCID-IV	26.74	26.32	19	21	WCST	Perseverative errors
[114]	SCID-IV	28.50	28.57	14	14	WCST	Perseverative errors
[115]	DSM-IV	33.07	28.57	14	14	WCST	Perseverative errors
[147]	DSM-IV	37.20	57.89	19	24	IED	EDS trial score
[49]	SCID-IV	26.50	17.64	34	34	WCST	Perseverative errors
[171]	DSM-IV	36.30	55.60	27	27	WCST	% of Perseverative errors
[25]	SCID-IV	31.90	57.10	20	26	WCST	Perseverative errors
[29]	DSM-IV	32.70	40.00	25	15	WCST	Perseverative errors
[65]	DSM-IV & ICD-10	39.00	53.33	15	17	IED WCST	EDS trial score Perseveration
[145]	SCID-DSM-III	32.40	60.00	10	13	WCST	Total errors
[169]	DSM-IV	26.26	19.00	21	20	WCST	Perseverative errors
[174]	DSM-IV & Y-BOCS	Age matched sample	45.45	11	11	Go/no-go	Mean number of errors
[35]	DSM-IV	25.73	53.34	30	30	WCST	% of Perseverative errors
[42]	DSM-IV	35.30	Not stated	20	20	CGT	% of rational decisions
[116]	SCID-DSM-IV	36.10	48.72	38	39	WCST	Perseverative errors
[180]	DSM-IV	Cur 41.47 Com 40.47	50.00 46.67	30 15	35	IED WCST	EDS trial score Perseverative errors
[42]	MINI	35.30	50.00	20	20	CGT	Percent rational decisions
[43]	MINI-DSM-IV	32.10	80.00	20	20	CGT SST IED	Percent rational decisions SSRT Trials to criterion Extra-dimensional
[59]	MINI	36.1	74.36	39	26	WCST	% of Perseverative errors
[108]	DSM-IV	25.73	46.67	15	15	Go/no-go	Commission errors
[110]	DSM-IV	32.87	73.90	23	22	WCST	Perseverative errors
[131]	MINI-DSM-IV	32.50	70.97	31	31	SST	SSRT (log transformed)
[172]	SCID-DSM-IV	37.80	58.30	12	14	Go/no-go	Commission errors
[25]	SCID	33.00	52.40	21	26	Go/no-go	False positives
[40]	SCID	Clean 30.90 Check 34.00	30.43 41.67	23 24	20	WCST	Perseverative errors
[164]	MINI-DSM-IV	27.77	26.67	30	30	WCST	% of Perseverative errors
[196]	ICD-10	34.98	82.00	30	30	WCST	Perseverative errors
[146]	ADIS-DSM-IV	37.80	61.02	59	59	IED	ED trial level
[155]	SCID-DSM-IV	39.1	0.00	10	11	Go/no-go	Probability of inhibition
[185]	DSM-IV	36.36	50.00	14	15	IGT	Total net score
[38]	DSM-IV	35.60	42.90	35	31	IGT WCST	Number of advantageous deck selections % of Perseverative errors
[186]	SCID-DSM-IV	35.25	43.48	23	22	mWCST IGT	Perseverations Net score
[22]	DSM-IV	43.00	Not stated	17	19	SST	SSRT
[32]	DSM-IV	33.00	58.20	67	17	WCST IGT	Perseverative errors Net score
[58]	MINI-DSM-IV	28.40	45.80	107	107	IGT CCPT-II	Net score Commission errors
[112]	DSM-IV	26.00	22.58	31	31	WCST IGT	% of perseverative errors decks A + B-(C + D)
[163]	DSM-IV-TR	25.60	50.00	30	30	WCST	Perseverative errors
[27]	DSM-IV	22.32	100.00	19	21	SST	SSRT
[39]	DSM-IV-TR	36.05	33.00	20	18	IGT	Net score
[60]	DSM-IV-TR	38.60	49.00	41	37	SST	SSRT
[96]	SCID	26.90	25.81	31	52	WCST	Perseverative errors
[20]	MINI	36.44	76.00	25	21	SST	SSRT

(*continued on next page*)

Table 1 (*continued*)

Study	Diagnosis	Mean age	Proportion female	OCD (n)	Control (n)	Test	Measure
						IED	EDS errors
[27]	MINI-DSM-IV	22.32	100.00	19	21	IGT	Mean taken from block scores
[57]	DSM-IV	43.20	69.23	10	10	CGT	Percent rational decisions
[63]	SCID-DSM-IV	33.40	64.00	25	25	Task similar to the WCST	Reversal errors
[100]	SCID-DSM-IV	24.90	33.33	18	18	SST	SSRT
[101]	MINI-DSM-IV	27.56	37.30	150	105	WCST	% of perseverative errors decks A + B-(C + D)
					75	IGT	
[87]	ICD-10	26.4	38.00	139	139	WCST	Perseverative errors
[179]	DSM-IV	65% aged 18–26	40.00	20	20	WCST	% of Perseverative errors
[184]	SCID-IV	27.80	26.20	80	76	SST	SSRT
[192]	DSM-IV	33.54	25.00	24	24	Go/no-go	Commission errors
[203]	DSM-IV	27.13	38.46	26	20	WCST	Perseverative errors
[83]	SCID-DSM-IV	36.29	39.47	38	39	IGT	IGT final net score
[105]	SCID – DSM-IV	26.62	21.54	65	58	IGT	Total net score
[111]	ICD-10	32.75	40.00	20	20	WCST	Perseverative errors
						WCST	Perseverative errors
[119]	SCID	21.67	45.24	42	42	SST	SSRT
[128]	DSM-IV	34.43	52.04	269	120	IGT	Mean IGT score
[181]	MINI	W/O D 30.43 W Dep 34.05	46.67 40.00	30 20	25	WCST	Perseverative errors
[195]	DSM-IV-TR	23.91	55.56	27	23	WCST	Perseverative errors
[205]	DSM-IV	26.62	29.31	58	58	WCST	Perseverative errors
[209]	SCID-CV	23.00	42.50	40	40	WCST	Perseverative errors
[210]	SCID-DSM-IV	NM 28.07	52.63	57	115	WCST IGT	Perseverative errors Net score
		M 27.92	54.55	77			
[211]	DSM-IV-TR	26.51	60.00	55	55	WCST IGT	Perseverative errors Net scores
[64]	SCID	Auto O 20.36 Reac O 22.37	42.90 38.90	40 47	58	SST	SSRT
[120]	SCID	EO 20.68 LO 24.64	36.51 30.30	63 33	51	SST	SSRT
[161]	DSM-IV	29.10	46.66	30	32	CCPT II	Commission errors
[149]	SMD	15.75	0.00	20	20	TDT	TD – delayed
[177]	DSM-IV	32.45	74.00	35	35	WCST Go/no-go	Perseverative errors Commission errors
[198]	MINI	36.14	52.27	44	43	IED	Errors at ED stage
[207]	DSM-IV	24.9	29.17	24	34	WCST	Perseverative errors
[212]	MINI-DSM-IV	30.71	29.41	51	31	IGT	Mean score from block data
[84]	SCID-I/P	44.75	45.00	40	40	IGT	Final net score
[104]	DSM-IV	32.46	Not stated	61	131	Go/no-go	False alarms
[129]	DSM-5	30.68	27.78	36	36	SST	SSRT (last half)
[150]	ICD-10	15.76	0.00	20	20	IGT	Net score
						IED	EDS errors
[50]	MINI-Plus	33.50	39.51	81	124	CGT	Quality of decision making
[85]	DSM-IV	33*	31.80	44	40	IGT	Net score
[193]	SCID	33.49	56.76	37	40	WCST	% of Perseverative errors
[206]	ICD-10	24.70	44.00	25	27	SST	SSRT
[26]	SCID	15.64	100.00	11	24	WCST	Perseverative errors
[76]	SCID	35.88	17.65	17	17	CGT	Quality of decision making
						SST	RT on go trials
[109]	DSM-IV	EO 23.27 LO 25.00	28.57 34.62	EO 49 LO 52	103	IED	IED errors
						CGT	Quality of decision making
[124]	DSM-5	19.71	33.30	24	26	IGT	Net scores
[191]	SCID-DSM-IV	30.19	61.00	24	19	SST	SSRT
[21]	MINI	f-OCD 37.15 s-OCD 32.75	50.00 47.27	f-OCD 54 s-OCD 55	60	SST	Mean stop RT
[48]	DSM-5	31.20	68.00	29	28	IED SST	ED errors SSRT
[127]	SCID-I, SCID-II	32.81	53.00	32	30	WCST Go/no-go	Perseverative errors Commission errors
[143]	ICD-10, DSM-5	34.77	69.00	13	30	WCST	Perseveration
[194]	SCID-I/P	33.34	60.98	41	49	SST	SSRT
[91]	SCID-DMS-IV	32.80	60.00	50	55	IGT	Total net score
[188]	SCID-I	33.92	Not stated	72	67	IED	No. of trials to reach stage 9 (ED switch-cost)

Foot note: DSM-III = Diagnostic and Statistical Manual of Mental health disorders-3rd edition, DSM-III-R = DSM-III-revised, DSM-IV = DSM-4th edition, DSM-IV-TR = DSM-IV-text revision, DSM-5 = DSM-5th edition, ICD-10 = International Classification of diseases-10th edition, SCID-DSM-III = Structured clinical interview for DSM-III, SCID – DSM-IV = Structured clinical interview for DSM-IV, SCID-CV = Structured clinical interview – Clinician version, SCID-I/P = Structured Clinical interview for DSM-IV Axis I Disorder-Patient Edition, MDI = Maudsley diagnostic interview, SMDI = Standardised Maudsley diagnostic interview, MINI = Mini international Neuropsychiatric interview, MINI-plus = Mini international Neuropsychiatric interview-plus, ADIS-DSM-IV = Anxiety Disorder interview schedule-DSM-IV, WCST = Wisconsin Card Sorting Test, mWCST = modified WCST, CGT = Cambridge Gambling Task, SST = Stop Signal Task, SSRT = stop signal reaction time, IED = Intra/Extradimensional Set-Shifting Task, IGT = Iowa Gambling Task, CCPT-II = Conner's Continuous Performance Test-II, TDT = Temporal Discounting Task, Ad vs disad = advantageous vs disadvantageous decks, NM = non-medicated, M = medicated, * = median, Clean = cleaning compulsions, Check = checking compulsions, W/O D = OCD without depression, W Dep = OCD with depression, Auto O = Autogenous obsessions, Reac O = Reactive obsessions.

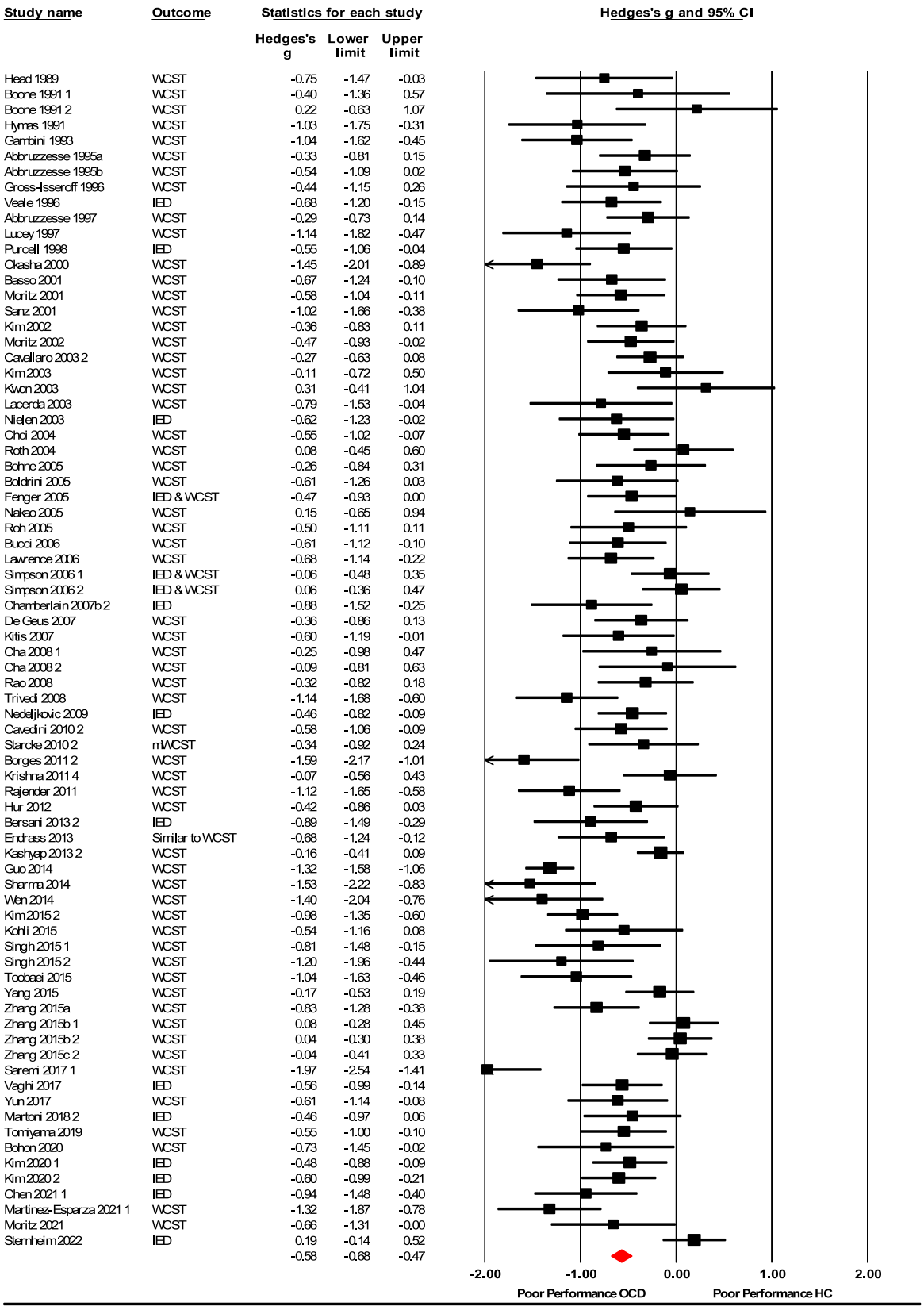

Fig. 2. Compulsivity measures effect sizes.

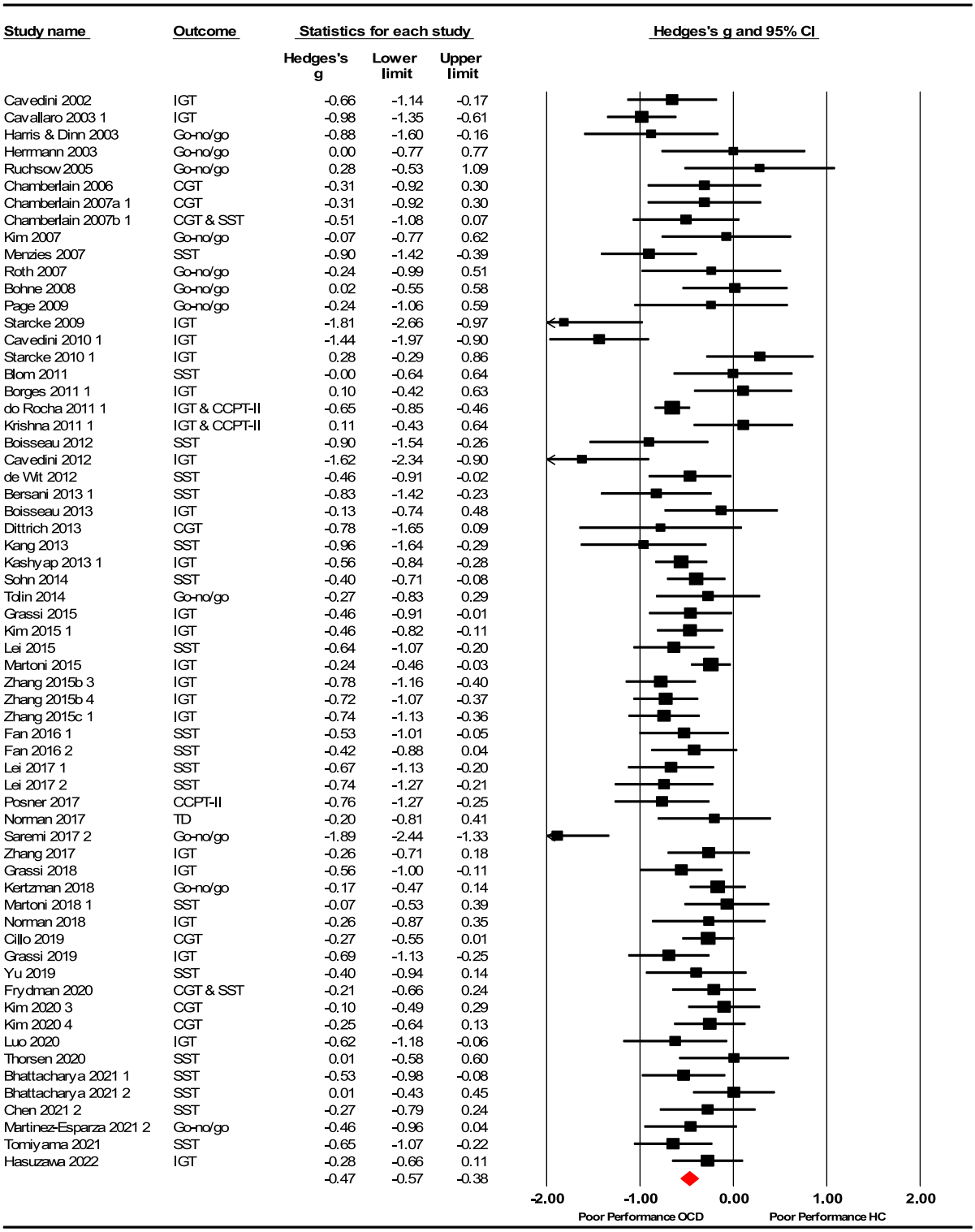

Fig. 3. Impulsivity measures effect sizes.

3.4. Motor impulsivity vs decision-making impulsivity

A subgroup analysis contrasted the two facets of impulsivity: motor and decision making (Figs. 6 and 7 respectively). A small to moderate effect size was observed for motor impulsivity ($g = -0.46$, [95%CI -0.59, −0.34]; k = 35, $p < .001$; $I^2 = 52.23$, $p < .001$). The funnel plot showed no evidence of asymmetry. A small to moderate effect size was also established for decision-making impulsivity ($g = -0.48$, [95%CI -0.61, −0.34]; k = 32, $p < .001$; $I^2 = 64.98$, $p = .001$). There was no significant difference between the two facets of impulsivity ($Q = 0.03$, df = 1, $p = .87$). The funnel plot showed no evidence of asymmetry.

Exploratory subgroup analyses were used to assess the two most used tasks to assess both motor and decision-making impulsivity. For motor impulsivity, the effect sizes for the Go/no-Go task ($g = -0.37$, [95%CI –0.72, −0.02]; k = 11) and the Stop Signal Task ($g = -0.48$, [95%CI –0.60, −0.35]; k = 21) were both significant but small-moderate in size.

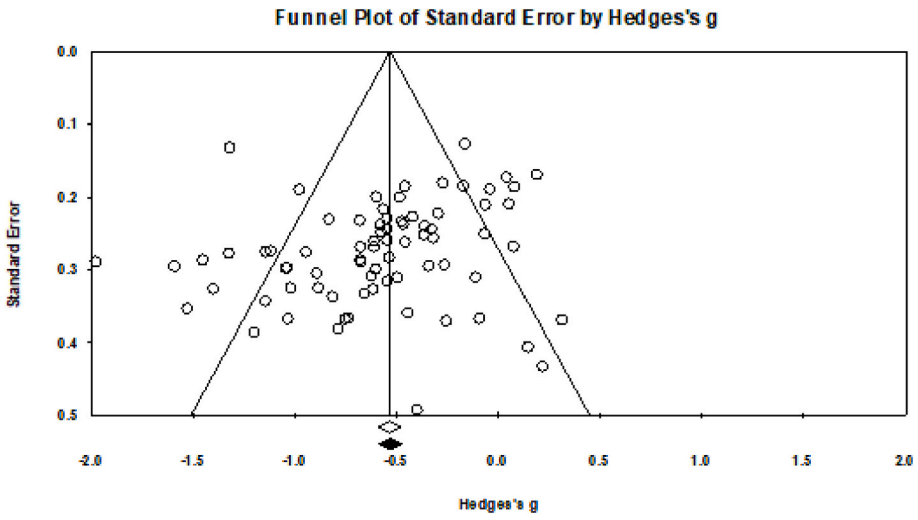

Fig. 4. Funnel plot for studies assessing compulsivity.

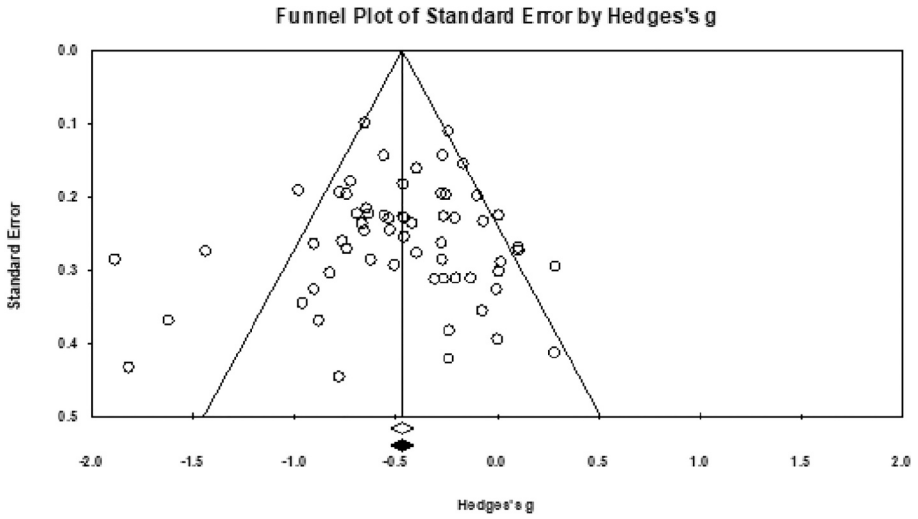

Fig. 5. Funnel plot for studies assessing impulsivity.

These effect sizes did not differ significantly ($Q = 0.29$, df = 1, $p = .59$).

A second analysis of decision-making impulsivity tasks revealed a moderate effect size for the Iowa Gambling Task ($g = -0.55$, [95%CI –0.52, −0.29]; k = 23) and a small effect size for the Cambridge Gambling Task ($g = -0.27$, [95%CI –0.43, −0.10]; k = 8). While both analyses revealed a significant decision-making impairment, the effect size was significantly larger for the IGT than the CGT ($Q = 5.47$, df = 1, $p = .02$).

3.5. Subgroup and moderator analyses

3.5.1. Comorbidities

In many studies, comorbidities were an exclusion criterion. For inpatient studies, 10 included comorbid disorders and 15 reported them as an exclusion criterion. In the case of outpatient-based studies, 39 reported exclusions and 21 included comorbid disorders. Subgroup analyses examining the influence of comorbidity on the task performance compared studies with and without comorbidities (See Appendix for Forest plot). Moderate-large effect sizes emerged for compulsivity in studies excluding ($g = -0.70$, [95%CI -0.85, −0.55]; k = 39, $p < .001$; $I^2 = 66.41$, $p < .001$) and including comorbidities ($g = -0.53$, [95%CI -0.71, −0. 35]; k = 18, $p < .001$; $I^2 = 60.91$, $p < .001$). No significant difference in effect size emerged for studies including and excluding comorbid disorders ($Q = 2.11$, df = 1, $p = .15$).

Task performance on impulsive measures showed a moderate effect size for studies excluding comorbidities ($g = -0.51$, [95%CI -0.67, −0.36]; k = 26, $p < .001$; $I^2 = 59.57$, $p < .001$) and small-moderate for studies including comorbidities ($g = -0.35$, [95%CI -0.47, −0.22]; k = 23, $p < .001$; $I^2 = 38.68$, $p < .05$). Again, no statistically significant difference emerged between the inclusion or exclusion subgroups ($Q = 2.72$, df = 1, $p = .10$).

Of the studies including comorbidities, the disorders were too varied and too few to perform further subgroup analyses to detail the influence more precisely on task performance.

3.5.2. Meta-regressions

Our pre-registered and planned meta-regression analyses assessed a series of variables as potential predictors of effect sizes. As shown in Table 2, two significant moderators emerged: greater impulsivity impairment occurred in studies with a greater proportion of female patients with OCD and larger compulsivity effect sizes in studies with lower study quality. Crucially, neither impulsivity nor compulsivity were moderated by any of the following planned predictor variables: OCD, anxiety or depression symptomatology; age, or illness duration; or

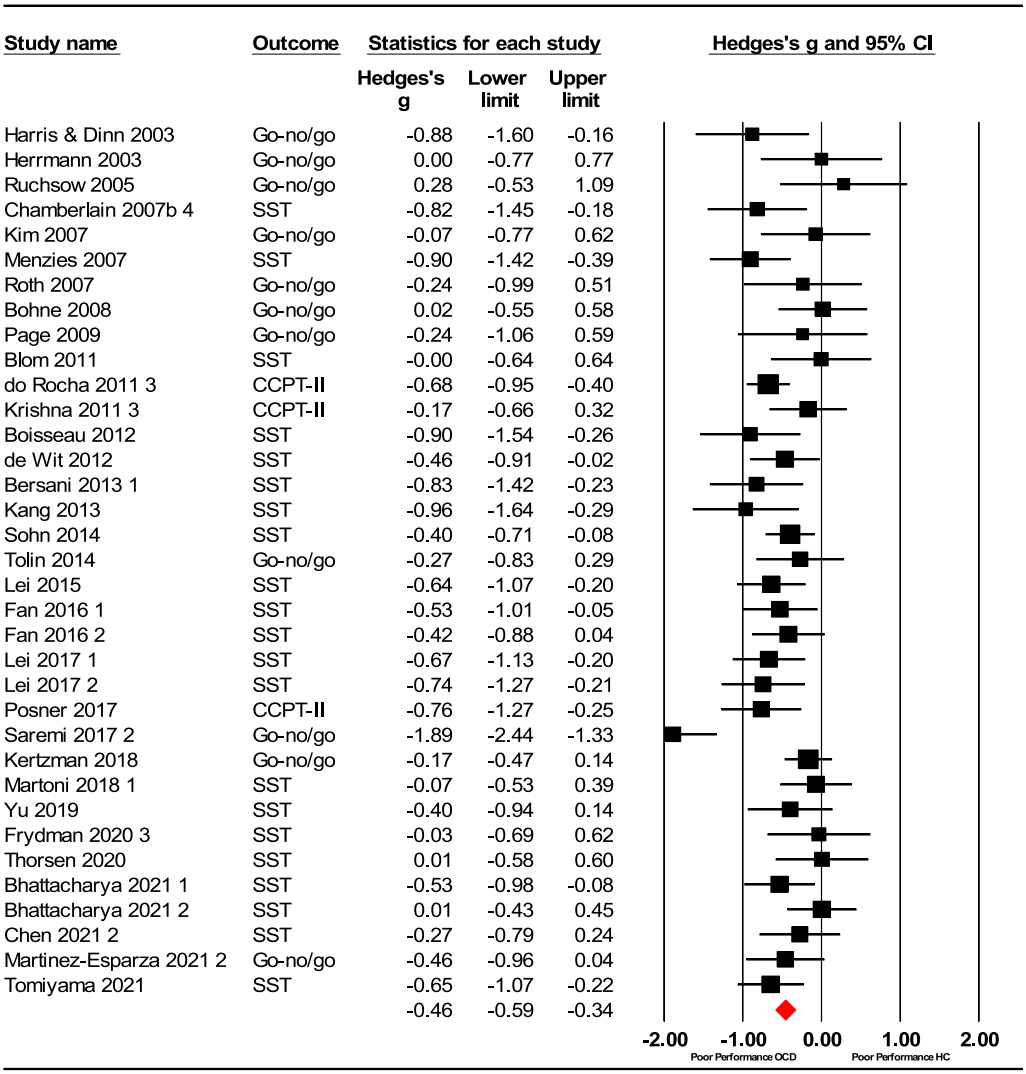

Fig. 6. Motor impulsivity.

exploratory analyses using years in education and intelligence (IQ).

3.5.3. Study quality

Study quality was assessed using the Appraisal tool for Cross-Sectional Studies (AXIS) checklist [61]. Following recent research [13], we classified AXIS quality scores according to the number of "YES" responses for the 20 items for each study – so, studies achieving 80% "yes" responses indicated high quality, 60–80% indicated moderate quality, and < 60% indicated low quality. All 112 studies were rated as moderate (64/112: 57.14%) to high quality (48/112: 42.86%). The mean score was 15.36 (1.10) across all 55 studies: 15.18 (1.10) and 15.57 (1.07) for studies assessing compulsivity and impulsivity respectively. As can be seen in Table 3, no significant differences were observed between compulsivity and impulsivity studies in research quality, study design, potential bias, or total AXIS scores. Whilst the items relating specifically to reporting quality scored highly, the detail relating to study design and possible biases are lower and more variable.

Compulsivity and impulsivity studies did not differ in research quality or potential bias (both $p > 05$: see Table 3) but did differ in study design and total AXIS scores with impulsivity showing greater study design and overall study quality than compulsivity studies. The year of publishing showed a positive relationship with both sample size ($r = 0.34$, $p < .001$), research quality ($r = 0.45$, $p < .001$) and with study design ($r = 0.45$, $p < .001$) but no significant relation with the potential bias ($r = -0.01$, $p > .05$). Hence, recent studies have employed larger samples and show better powering and better study quality.

3.6. Exploratory analyses

3.6.1. Outpatient vs Inpatient recruitment

An exploratory subgroup analysis was performed to explore patient care (inpatient versus outpatient) on task performance (See Appendix for Forest plot). Compulsivity effect sizes were significant for both inpatient clinics ($g = -0.56$, [95%CI -0.70, −0.43];k = 17, $p < .001$; $I^2 = 29.57$, $p = .12$) outpatient departments ($g = -0.59$, [95%CI -0.74, −0.43];k = 45, $p < .001$; $I^2 = 75.99$, $p < .001$) and mixed outpatient and inpatient samples ($g = -0.56$, [95%CI -0.90, −0.22]; k = 5, $p < .05$; $I^2 = 60.20$, $p = .04$); no difference emerged across effect sizes ($Q = 0.05$, df = 2, $p = .98$). Similarly, for impulsivity effect sizes, impatient ($g = -0.50$, [95%CI -0.74, −0.26];k = 13, $p < .001$; $I^2 = 76.52$, $p < .001$), outpatient samples ($g = -0.47$, [95%CI -0.60, −0.34];k = 36, $p < .001$; $I^2 = 56.41$, $p < .001$) and mixed outpatient and inpatient samples ($g = -0.45$, [95% CI -0.66, −0.23]; k = 6, $p < .001$; $I^2 = 46.41$, $p = .10$) were impaired; no significant difference emerged ($Q = 0.10$, df = 2, $p = .95$).

3.7. Effects of medication

The potential moderating effect of medication on task performance

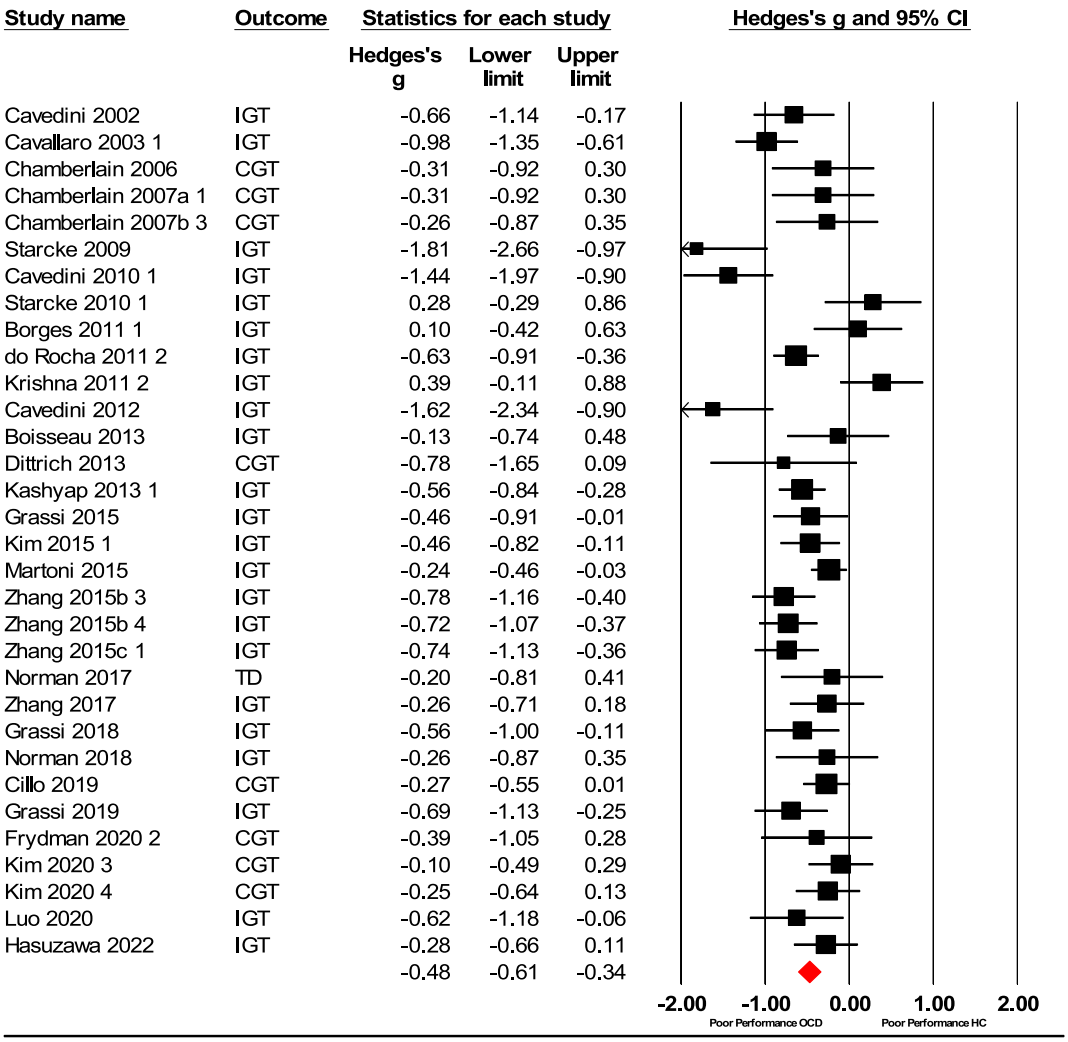

Fig. 7. Decision-making impulsivity.

was examined by contrasting medicated samples with those withdrawn from medication (who underwent a 'wash out period' typically 4 weeks before the study) – see Appendix for Forest plot. Medicated samples demonstrated a moderate effect size for compulsivity ($g = -0.50$, [95% CI -0.62, −0.38]; k = 40, $p < .05$; $I^2 = 67.91$, $p < .001$), as did unmedicated samples ($g = -0.58$, [95%CI -0.83,-0.34]; k = 19, $I^2 = 78.08$, $p < .001$) and those withdrawn from medication ($g = -0.57$, [95%CI -0.77, −0.37]; k = 14, $p < .001$; $I^2 = 47.71$, $p < .05$); and these effect sizes did not differ significantly ($Q = 0.57$, df = 2, $p = .75$). Similarly, on tasks measuring impulsivity, significantly impaired performance emerged in medicated ($g = -0.39$, [95%CI -0.49, −0.29]; k = 41, $p < .001$; $I^2 = 39.08$, $p < .05$) and unmedicated samples ($g = -0.56$, [95%CI -0.73, −0.40]; k = 16, $p < .05$; $I^2 = 53.01$, $p < .05$); and again, they did not significantly differ ($Q = 3.12$, df = 1, $p = .08$).

Lower symptom severities, as measured by the Y-BOCS, were found for the medicated samples (k = 63; 23.04, SD = 3.10) compared to unmedicated and withdrawn samples (k = 37; 25.30, SD = 2.52) (t (98) = −3.93, $p < .001$). No difference was found in either compulsivity or impulsivity further evidencing the independence of phenotype from symptom severity.

4. Discussion

4.1. Do patients with OCD differ significantly from healthy controls on tasks of cognitive flexibility and response inhibition?

The current meta-analysis provides the most comprehensive meta-analysis to date (112 studies with 8313 participants) documenting significant impairments of cognitive compulsivity and impulsivity in patients with OCD when compared to healthy controls (Hedge's $g = -0.58$ and $g = -0.48$ respectively). The included studies showed substantial heterogeneity, but were all of moderate to high-quality and showed no evidence of small study effects or publication bias. Planned moderator analyses showed that neither impulsivity nor compulsivity impairments varied according to various clinical variables, including whether patients with OCD were: medicated vs unmedicated; inpatients vs outpatients; or with and without comorbid psychiatric disorders. Furthermore, meta-regression analyses showed that neither compulsivity nor impulsivity were associated with severity of OCD, depression or anxiety symptomatology, illness duration, years in education or IQ.

4.2. Cognitive inflexibility, response inhibition, and OCD symptom severity

Researchers have commented upon the potential influence of

Table 2
Meta-regressions compulsivity, impulsivity and clinical variables.

	Mean (SD)	Range	Z-test
Age			
Comp (k=75)	31.29 (4.94)	15.64–41.47	Z = 0.83, df = 1,74, p = .41
Imp (k = 60)	30.81 (6.60)	15.75–44.75	Z = -0.16, df = 1,59, p = .88
Proportion of Females			
Comp (k = 73)		17.64–100.00	Z = -1.24, df = 1,72, p = .22
Imp (k = 59)		0.00–100.00	Z = -2.10, df = 1,58, p = .04*
Duration of illness			
Comp (k = 50)	9.62 (4.40)	2.80–21.60	Z = -0.14, df = 1,49, p = .89
Imp (k = 28)	9.62 (4.01)	3.25–18.24	Z = 1.00, df = 1,27, p = .32
OCD symptoms (Y-BOCS)			
Comp (k = 60)	24.45 (2.52)	18.00–33.64	Z = 0.18, df = 1,58, p = .86
Imp (k = 54)	23.73 (3.71)	13.91–30.76	Z = -1.51, df = 1,48, p = .13
Anxiety scores (HAM-A)			
Comp (k = 35)	11.29 (4.01)	1.93–16.90	Z = 0.77, df = 1,34, p = .44
Imp (k = 27)	9.07 (3.33)	4.05–15.16	Z = -0.62, df = 1,26, p = .53
Depression scores (HDRS)			
Comp (k = 49)	10.57 (5.49)	2.47–24.40	Z = -1.08, df = 1,48, p = .28
Imp (k = 41)	7.47 (3.40)	3.98–14.37	Z = -0.45, df = 1,40, p = .65
Years in education			
Comp (k = 55)	13.13 (1.46)	10.34–17.20	Z = 0.75, df = 1,54, p = .45
Imp (k = 38)	13.40 (1.27)	10.87–17.20	Z = 1.75, df = 1,37, p = .08
Intelligence (IQ)			
Comp (k = 31)	107.16 (5.79)	93.50–114.70	Z = 0.89, df = 1,30 p = .37
Imp (k = 19)	111.50 (6.08)	93.50–117.70	Z = -0.92, df = 1,18, p = .36
AXIS			
Comp (k = 76)	15.18 (1.10)	13.00–18.00	Z = 2.36, df = 1,75, p = .02*
Imp (k = 62)	15.57 (1.07)	13.00–18.00	Z = -0.53, df = 1,61, p = .60
Year of Publish			
Comp (k = 76)	2007.88 (8.36)	1989–2022	Z = -0.81, df = 1,74, p = .42
Imp (k = 62)	2013.84 (5.36)	2002–2022	Z = 1.10, df = 1,61, p = .27

Foot note: Comp = Compulsivity, Imp = Impulsivity, Y-BOCS = Yale-Brown Obsessive Compulsive Scale, HAM-A = Hamilton Anxiety Rating Scale, HDRS = Hamilton Depression Rating Scale, AXIS = Appraisal tool for Cross-sectional studies.

Table 3
Axis quality scores.

	Impulsivity (k = 63)	Compulsivity (k = 76)	
	Mean (SD)	Mean (SD)	t-test
Research quality	6.81 (0.40)	6.68 (0.47)	t (136.94*) =1.71, p = .09
Study design	5.59 (0.53)	5.26 (0.64)	t (137) =3.21, p < .05
Potential of bias	3.17 (0.58)	3.21 (0.62)	t (137) = −0.35, p = .73
Total score	15.57 (1.07)	15.18 (1.10)	t (137) =2.08, p < .05

Foot note: * = equal variances not assumed as of a significant Levene's test (p < .05).

symptomatology on cognitive function and especially regarding executive function. Abramovitch et al. [4] proposed that "the overflow of OC symptoms in OCD causes an overload on the executive system that result in neuropsychological impairments" (p.166). In other words, executive deficits are assumed to be a secondary consequence of OCD symptomatology and Abramovitch et al. further argued that "…successful treatment reducing OC symptoms in OCD will be complemented by reduction in neuropsychological impairments" (ibid p. 184). Consistent with this notion, Abramovitch et al. [5] reported significant inverse correlations for OCD symptomatology with performance on both response inhibition ($r = -0.280$) and set-shifting ($r = -0.277$) tasks. By contrast, the current meta-analysis provides no support for a relationship between OCD symptomatology (as measured by Y-BOCS scores) and either compulsivity or impulsivity effect sizes. The mean Y-BOCS scores across the compulsivity and impulsivity samples was 24.45 and 23.73 respectively, indicating that OCD symptomatology was at the upper-end of moderate severity [189]; ranging from 13.91 [21] to 30.76 [64]. Hence, the failure to find a relationship does not reflect a lack of variability or low levels of OCD symptomatology. The main difference between Abramovitch et al. [5] and the current meta-analytic study, is that our moderator analyses are looking at predicting variability in executive effect size *differences* between patients and controls from patient Y-BOCS scores (i.e. case-controlled deficits in cognitive performance), while Abramovitch et al. assessed variability in within-group correlations for patients only. These findings are not necessarily inconsistent as the degree of cognitive impairment for patients compared to controls may be unrelated to patient symptom levels even if symptoms and cognition display a within-patient correlation (as, for example, a similar relationship may exist for controls).

Consistent with the independence of cognitive deficits and symptomatology, we also note that patients with OCD in remission continue to exhibit executive impairment in the domains of set-shifting and inhibition [164,179]. Sharma et al. [179] assessed 15 remitted patients with OCD with Y-BOCS scores of 0–7, while Rao et al. [164] assessed 30, who were asymptomatic having mean Y-BOCS =2.57 and no clinically significant concurrent depression (mean HDRS = 1.97) or anxiety (mean HARS = 1.93). Both studies found that compared to matched controls, those who had recovered from OCD continued to show impaired performance on tests of set-shifting and inhibition. Some authors have further speculated that such cognitive deficits may be associated with or even underpin the occurrence of symptomatic relapse [16]. Bannon et al.[16] reported impaired functioning on tests of set-shifting (WCST) and inhibition (Go/No-Go; Stroop) in 60 patients with OCD compared to an anxiety (panic disorder) control group. Crucially, they followed-up 20 patients with OCD (on average 1.4 years later) who had remitted, and these individuals continued to show impaired performance on set-shifting and inhibition. These findings not only point to the independence of OCD symptomatology from cognitive compulsivity and impulsivity, but also the fact that the latter cognitive deficits remain despite treatments leading to remittance of OCD symptoms.

4.3. Are effect sizes for compulsivity and impulsivity impacted by the presence of comorbid symptoms or disorders?

Our analyses go further in showing that the compulsivity and impulsivity deficits are not only independent of OCD symptoms, but also unrelated to other common conditions and symptomatology that are often comorbid with OCD. No differences in effect size emerged for compulsivity or impulsivity when we compared studies that did versus those that did not include participants with comorbid psychiatric problems. Comorbidities were quite varied (see Appendix 1 and 2), but we further assessed data concerning the common comorbid symptoms of depression and anxiety. Major depressive disorder is the most common comorbidity in OCD [178], with high rates of current (32%) and lifetime (77%) comorbidity existing between OCD and MDD [34]. Individuals diagnosed with MDD alone are known to be impaired on tests of set-shifting and inhibition and such deficits are related to depression severity [182]. Indeed, concurrent depressive symptomatology has been advanced as a possible explanation for the impaired executive function performance in patients with OCD (see [17,141]). Nonetheless, our analyses of large numbers of compulsivity (k = 49) and impulsivity (k = 41) studies found no significant association with depressive symptomatology (as assessed using the HDRS). Similarly, anxiety symptoms as measured using the Hamilton Anxiety Rating Scale [88] also failed to predict effect sizes for either compulsivity (k = 34) or impulsivity (k = 27). We note that the mean HDRS and HAM-A scores of included samples indicate low-to-mild levels of depression and anxiety and so, it remains possible that higher levels might impact executive task performance.

4.4. Are effect sizes for compulsivity and impulsivity impacted by other clinical variables?

The dissociation between symptom severity and cognitive phenotype may act as a proxy for the distinction between *state* and *trait* components of OCD respectively [183]. Indeed, a latent phenotype approach to neurocognitive deficits assumes that neurocognitive deficits are trait features. In this context, compulsivity and impulsivity are akin to trait phenomena since no differences were observed in either phenotype across: OCD symptomatology, presence or absence of comorbidities, level of depression or anxiety symptoms, age, years in education, IQ, duration of illness, inpatient versus outpatient status, medicated versus unmedicated or medication withdrawal.

The stability of compulsivity and impulsivity deficits and, as noted above, their relative independence from symptomatology also concurs with such deficits remaining largely untouched by current OCD treatments. Conversely, we note also that unaffected first-degree relatives of those with OCD also show impairments on compulsivity and impulsivity tasks (e.g., [31,208]). For example, Bora [31] reports that relatives of those with OCD perform poorer than healthy controls on tasks of both inhibition (Stroop and Stop-signal tasks: $d = 0.58$, 95%CI 0.29 to 0,86) and set-shifting (WCST, IED and trials: $d = 0.37$, 95%CI 0.04 to 0.69). Such findings accord both with the idea that the compulsivity and impulsivity deficits are not secondary to symptomatology and the idea that such deficits are strong candidates for potential trait markers for OCD.

The current findings partly accord with a recent meta-analysis of Pediatric OCD samples [121] insofar as in child and adolescent samples evidence suggests significant deficits for compulsivity ($d = -0.42$; 95% CI -0.61 to −0.14; k = 12) and inhibition ($d = -0.22$; 95%CI -0.34 to −0.11; k = 15), although not for decision-making ($d = -0.17$; 95%CI -0.41 to 0.08; k = 3). Although significant, the mean effect size for impulsivity in younger samples falls below the lower end of the 95% confidence intervals reported here for adult samples. Unfortunately, Lopez-Hernandez did not identify which tests and outcome measures were assessed and the number of decision-making samples is too few to make any definitive conclusions. Another possibility is that smaller effect sizes in children reflects the fact that the tasks used are not well-validated in children and therefore they may fail to sensitively discriminate poor task performance in the younger age group. Our series of planned meta-regression analyses also showed that duration of illness failed to predict either compulsivity or impulsivity effect sizes in adults with OCD. The current findings in conjunction with those of Lopez-Hernandez et al. [121] suggest that the compulsivity and impulsivity deficits may be relatively stable from the early development of OCD. The mean period of diagnosis reported by Lopez-Hernandez et al. [121] was 3.7 years, while the illness duration across our adult samples was of course much longer, exceeding 10 and 15 years for impulsivity and compulsivity studies respectively. The evidence accords with early mild to moderate cognitive impairment that stabilises from late adolescence and throughout adulthood.

4.5. Heterogeneity across tasks

A further potential source of heterogeneity concerns the variation in tasks assessing compulsivity and impulsivity and the respective outcome measures employed for these tasks across studies. Compulsivity was assessed on two key tasks – the WCST and the IDED; and we focused on data relating to WCST perseverative responses and the extra-dimensional shift stage of the IDED (where a previously irrelevant visual dimension e.g., lines become relevant, and a previously relevant visual dimension e.g., shapes become irrelevant). These WCST and IDED metrics are viewed as the classical outcomes associated with cognitive flexibility/rigidity of thinking and our exploratory analysis revealed no difference in their effect sizes.

We explored the differences within- as well as between-latent phenotypes as impulsivity is not a unitary construct [165]. Impulsivity has been assessed on a range of measures tapping into both motor impulsivity (e.g., Stop Signal Task, the Go/No-Go task) and decision-making impulsivity (the Cambridge Gambling Task and the Iowa Gambling Task). Our meta-analysis identified moderate effect sizes indicating impairment of both ($g = -0.46$ and $g = -0.48$ respectively). This accords with OCD characteristics such that patients exhibit a diminished capacity and choice to inhibit or delay their compulsive behaviours.

Our findings largely accord with a recent meta-analysis by Mar et al. [126] documenting impaired inhibitory control in patients with OCD compared to controls (measured as raw mean differences in RTs on the SST) with greater impairment in older samples. An exploratory analysis of 14 studies in our meta-analysis using the SST identified an overall significant impairment (g = 0.48 [95%CI –0.61, −0.35; k = 14) but no relationship with age (z = 1.32, $p = .19$) - and indeed pointed to a greater deficit in younger participants.

Previous literature had attested to the association between OCD and motor impulsivity [125,126], but has presented an indistinct consensus regarding decision-making impulsivity, with some research reporting the relationship [52,84,148] and others not [9].

While our exploratory analyses found no motor impulsivity differences when comparing effect sizes for SST and the Go/No-go Task, analysis of decision-making impulsivity revealed a significantly larger deficit for the Iowa Gambling task ($g = -0.55$) than the Cambridge Gambling Task ($g = -0.27$). Compulsive behaviours in people with OCD may broadly be conceptualised as failures in decision-making (see [41]) and the disparity of performance across decision-making tasks may offer important information about the character of this deficit. While the IGT probes decision-making in ambiguous conditions (with participants unaware of reward probabilities), the CGT probes decision-making under conditions of risk (with participants aware of reward probabilities). With this distinction in mind, people with OCD may be more impaired at decision-making under ambiguity (IGT) than in situations with defined risk (CGT); however, this may also partly reflect the somewhat greater cognitive demands of the IGT than the CGT.

4.6. Treatment implications

While the medicated samples included here showed lower symptom severities than unmedicated and withdrawn samples, these samples did not differ significantly in compulsivity and impulsivity measures. Similarly, executive function deficits in patients with OCD have been found unresponsive to psychological interventions such as CBT [199] and other medical interventions e.g., deep brain stimulation of the nucleus accumbens and the anterior limb of the internal capsule [97] despite symptomatology responding. In contrast, Tyagi et al. [197] found that DBS targeting the subthalamic nucleus and the cognitive corticostriatal loop (but not the orbitofrontal loop) improved OCD symptoms and IED performance, strongly implicating that specific neural circuit in the origins of cognitive inflexibility and a specific interventional target for those patients with IED deficits. One possibility is that typical psychological and medical treatments for OCD are more efficacious at alleviating the *state* aspects of OCD as opposed to the trait-like components. These findings point to the necessity of complementing existing treatment with interventions aimed at ameliorating these underlying core cognitive deficits, which may involve precisely targeted neurostimulation approaches.

4.7. Limitations

Studies of neurocognitive function in patients with OCD are notable for their failure to assess quality of life and functional outcomes. Indeed, just one of 112 studies included here assessed the functional impairment of their sample [76]. This substantial oversight prohibits an estimation of the relative impact that neurocognitive deficits may have on these key clinical variables. As detailed by Eisen et al. [62], psychosocial functioning is indeed impaired in OCD, with other authors propounding the relation between functional impairment and poor quality of life [175]. With the aim of using quality of life as an outcome metric for therapeutic interventions [190], conceptualising the variation in functional impairment between patients with OCD [202] will aid person centred care.

While we found no significant relationship between compulsivity or impulsivity effect sizes and YBOCS total symptom scores, we cannot exclude the possibility that relationships might exist with more specific symptom clusters (see [33]). Although our analyses have focussed on trying to be as specific as possible with the neurocognitive assessment outcome measures, most studies have looked at executive functions in relation to an overall symptomatic score rather than ratings for specific symptoms. Total YBOCS scores may be misleading as the same total scores can be generated by quite different profiles and mask substantial heterogeneity among patients with OCD [56]. Future studies should focus upon increasing the specificity of both neurocognitive and symptomatic measures as far as possible.

The poorer performance on tests of cognitive flexibility and impulsivity documented here are, of course, not unique to patients with OCD and have been documented in other disorders such as major depression [182], eating disorders [204], schizophrenia [117], and bipolar disorder [167]. The fact that these deficits are found among various disorders has led researchers to contend an argument of non-exclusivity to the diagnostic class of OCD, but as common cognitive deficits in most mental disorders [7]. This may also allude to these neurocognitive impairments being transdiagnostic in nature [18].

4.8. Conclusion

The current comprehensive meta-analysis involving >8000 participants evidences a dual deficit of cognitive inflexibility and response inhibition in patients with OCD. Crucially, this work clearly shows that such deficits in OCD are independent of a range of variables related to clinical status. We have also shown that the neurocognitive impairments of impulsivity and compulsivity appear to be independent of OCD symptomatology (as well as comorbid symptoms of depression and anxiety). These deficits appear to continue in patients whose symptoms have remitted as well as in first degree relatives – these findings collectively converge on the notion that neurocognitive impulsivity and compulsivity are latent trait components of OCD. A key implication of this observation concerns the importance of exploring potential interventions for these specific cognitive difficulties that appear unresponsive to existing treatments that show efficacy with OCD symptomatology.

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CRediT authorship contribution statement

Aaron T. Clarke: Data curation, Formal analysis, Methodology, Writing – original draft. **Naomi A. Fineberg:** Conceptualization, Supervision, Writing – review & editing. **Luca Pellegrini:** Conceptualization, Writing – review & editing. **Keith R. Laws:** Conceptualization, Supervision, Writing – review & editing.

Declaration of competing interest

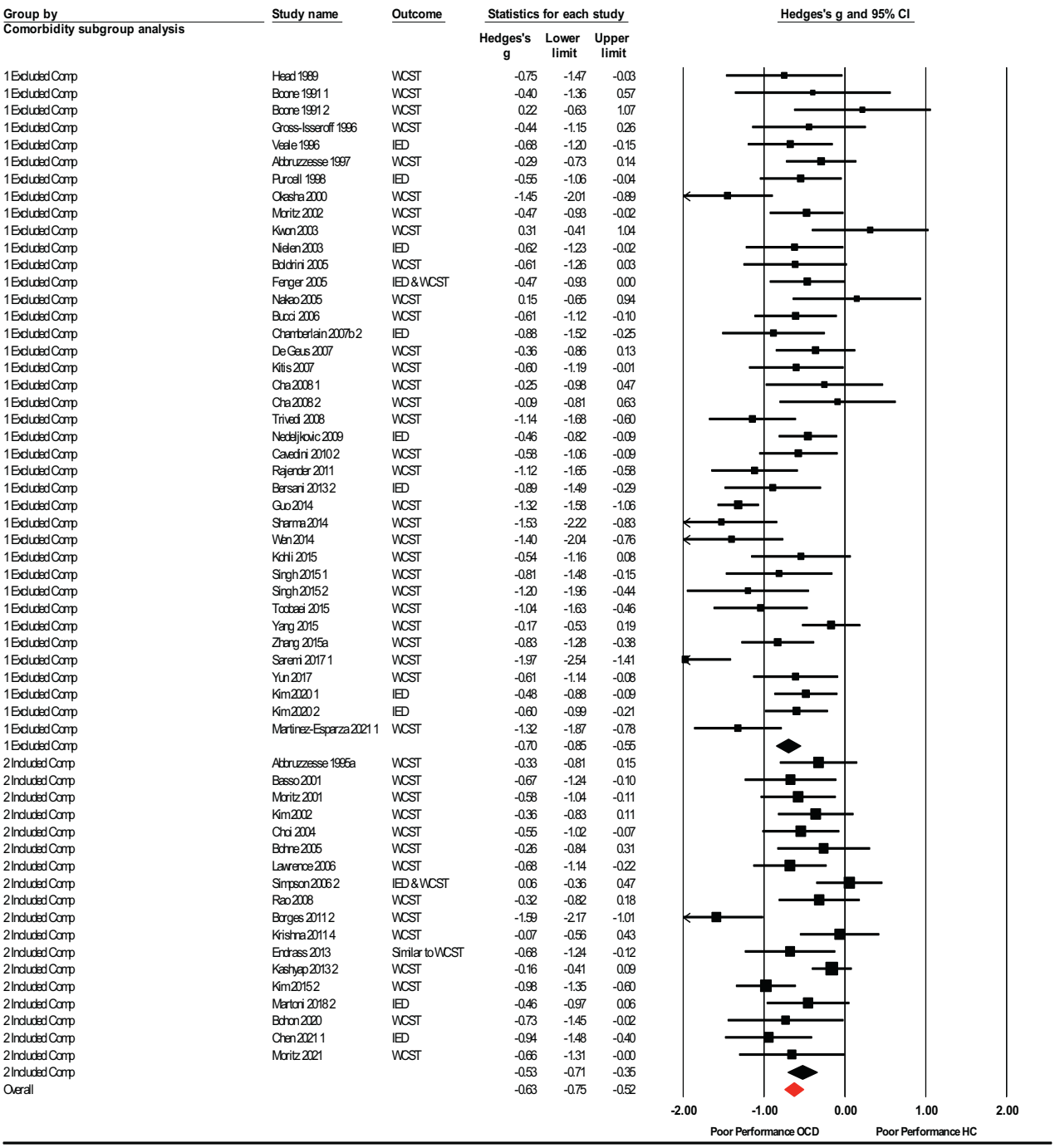
Prof. Naomi Fineberg reports in the past 3 years she has held research or networking grants from the UK NIHR, COST Action, Orchard, Horizon Europe, UKRI; accepted travel and/or hospitality expenses from the BAP, ECNP, RCPsych, CINP, World Psychiatric Association; received payment from Elsevier for editorial duties and the Mental Health Academy and Children and Screens for lecturing. Previously, she has accepted paid speaking engagements in various industry supported symposia and recruited patients for various industry-sponsored studies in the field of OCD treatment. She leads an NHS treatment service for OCD. She holds Board membership for various registered charities linked to OCD. She gives expert advice on psychopharmacology to the UK MHRA. She has participated in a WHO working group focusing on diagnosis and classification of obsessive compulsive or related disorders for the ICD-11.

Acknowledgements

None.

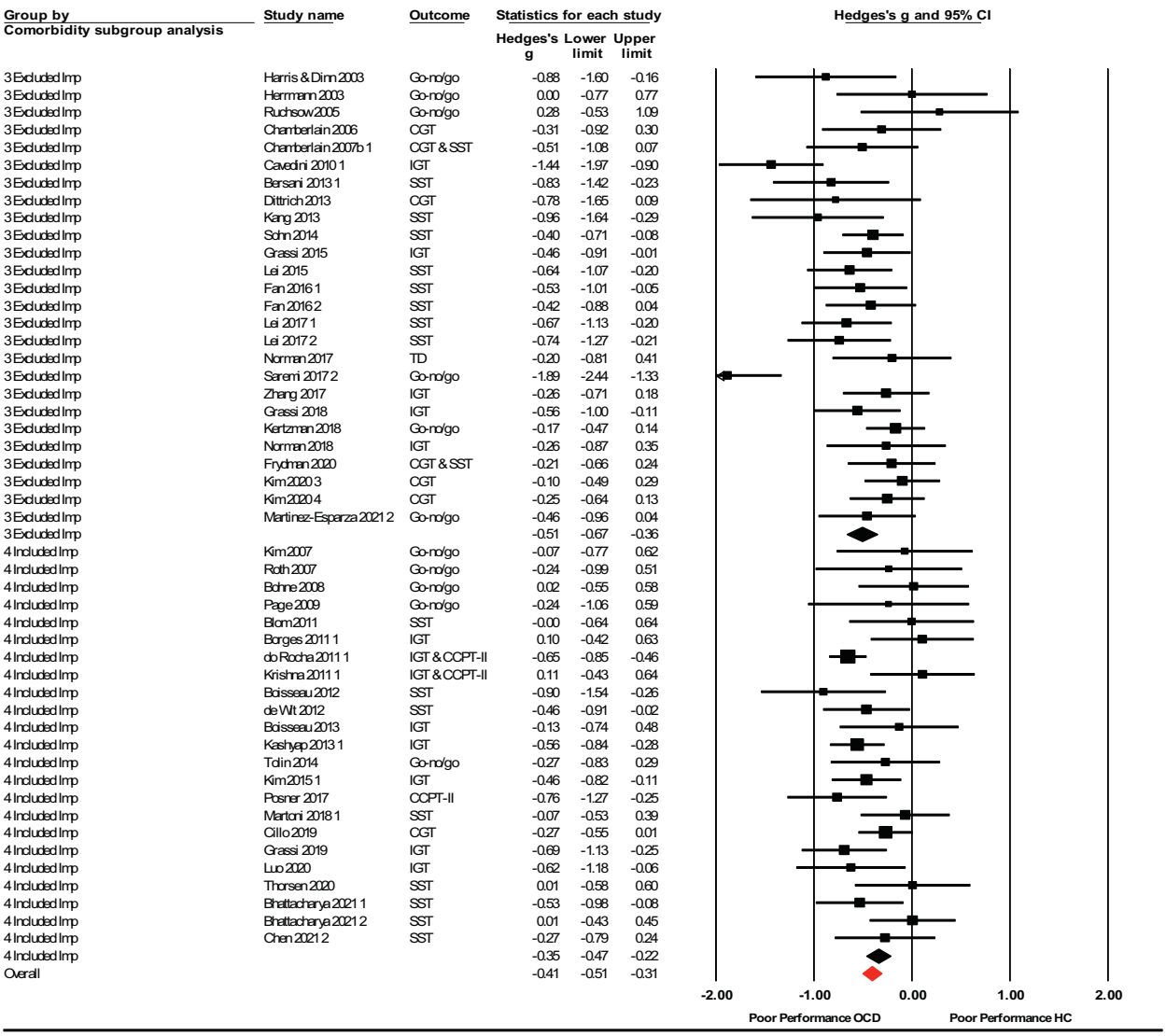
Appendix

Appendix 1
Included and excluded comorbidities for compulsivity.



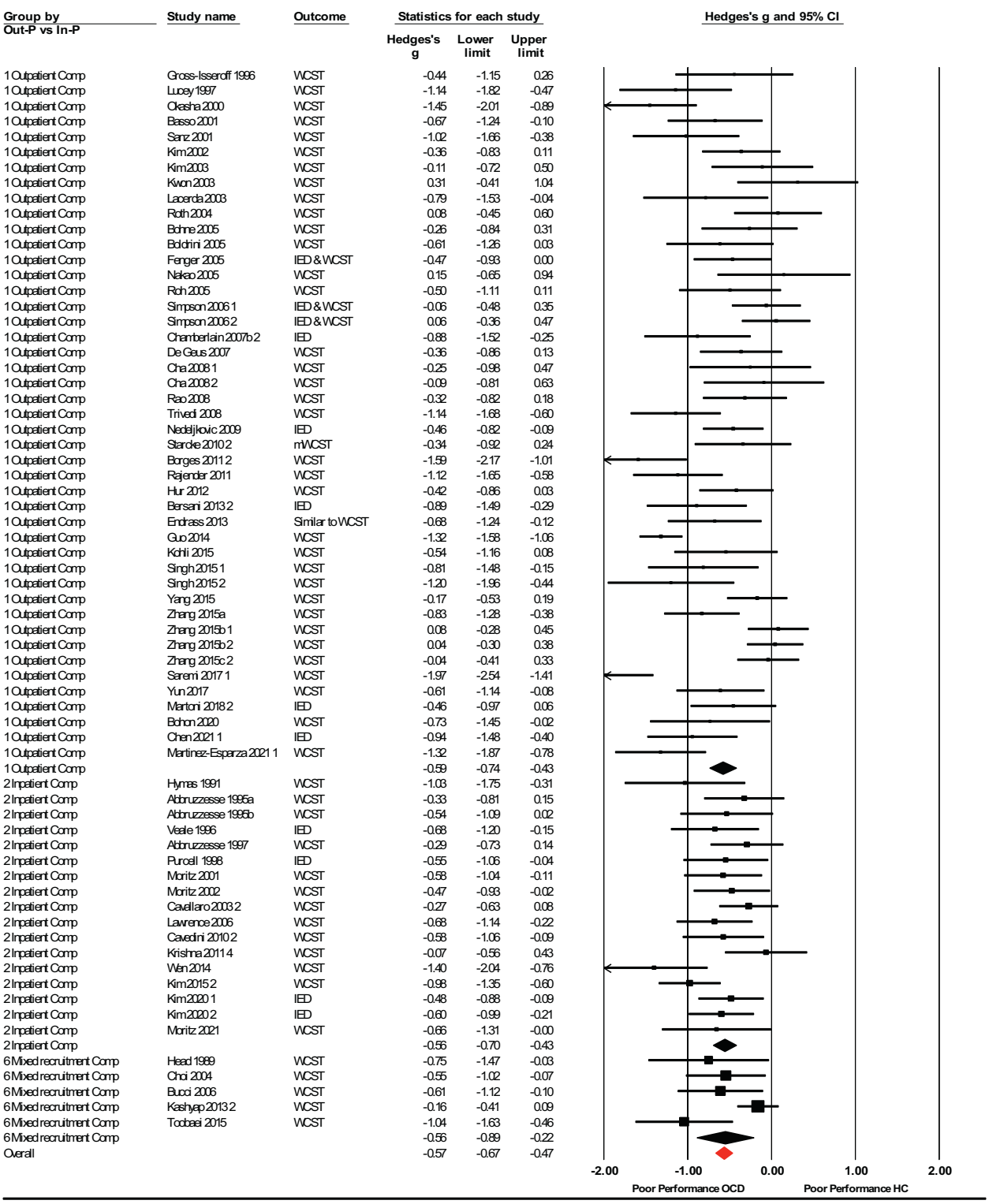
Appendix 2

Included and excluded comorbidities for impulsivity



Appendix 3

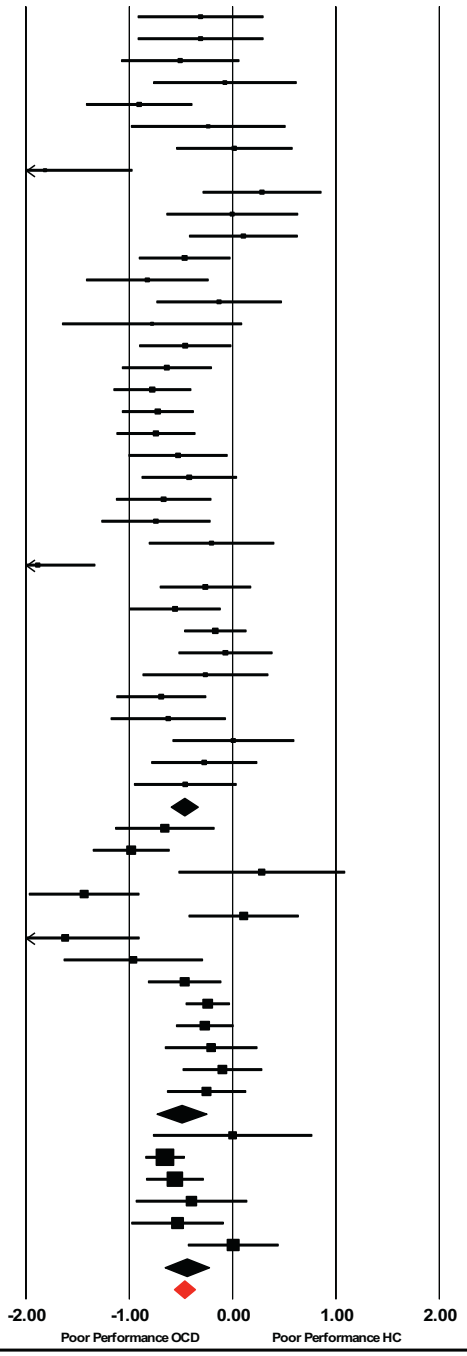
Subgroup analysis of clinical status and compulsivity



Appendix 4

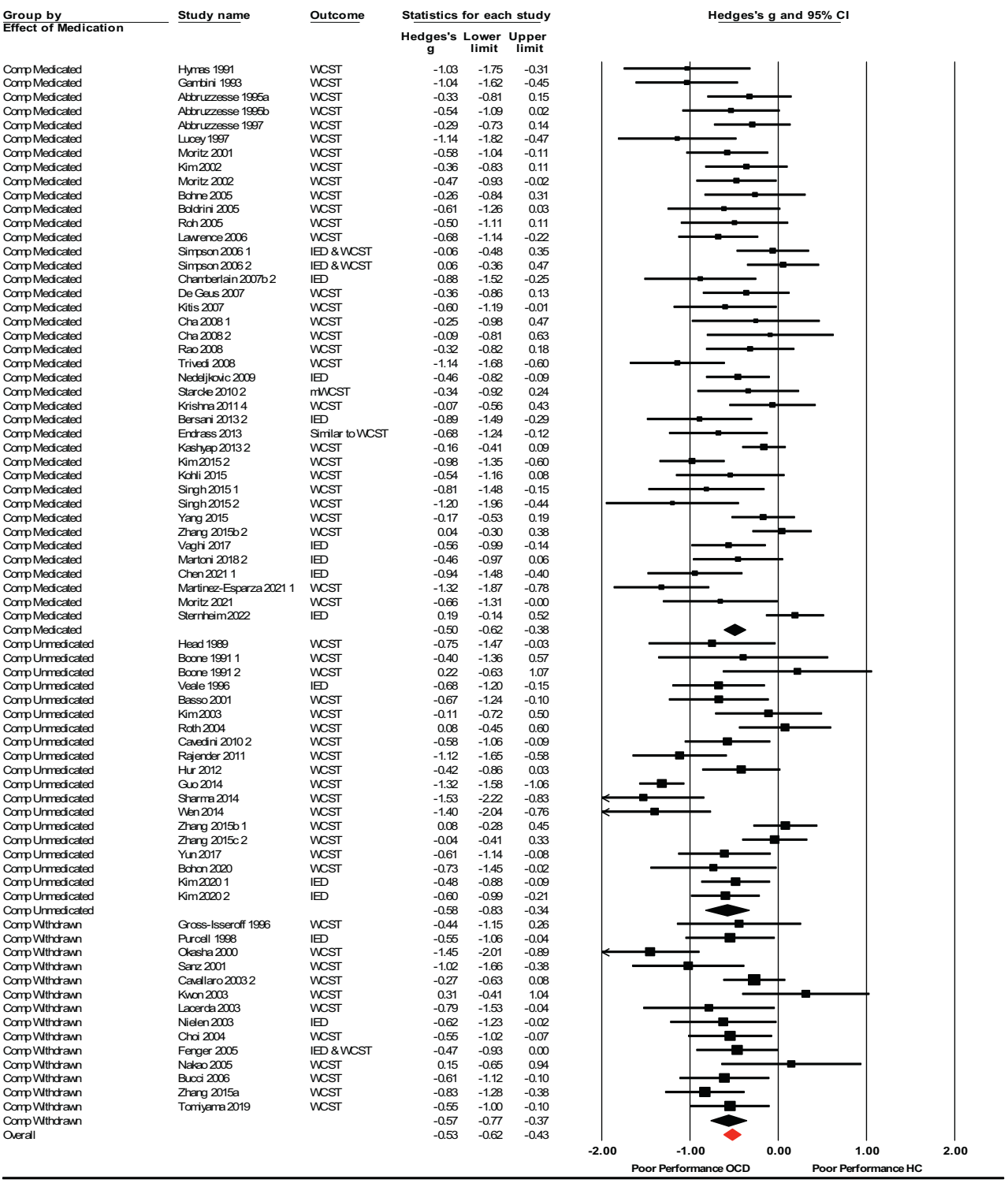
Subgroup analysis of clinical status and impulsivity

Group by Out-P vs In-P	Study name	Outcome	Hedges's g	Lower limit	Upper limit
3 Outpatient Imp	Chamberlain 2006	CGT	-0.31	-0.92	0.30
3 Outpatient Imp	Chamberlain 2007a 1	CGT	-0.31	-0.92	0.30
3 Outpatient Imp	Chamberlain 2007b 1	CGT & SST	-0.51	-1.08	0.07
3 Outpatient Imp	Kim 2007	Go-no/go	-0.07	-0.77	0.62
3 Outpatient Imp	Menzies 2007	SST	-0.90	-1.42	-0.39
3 Outpatient Imp	Roth 2007	Go-no/go	-0.24	-0.99	0.51
3 Outpatient Imp	Bohne 2008	Go-no/go	0.02	-0.55	0.58
3 Outpatient Imp	Starcke 2009	IGT	-1.81	-2.66	-0.97
3 Outpatient Imp	Starcke 2010 1	IGT	0.28	-0.29	0.86
3 Outpatient Imp	Blom 2011	SST	-0.00	-0.64	0.64
3 Outpatient Imp	Borges 2011 1	IGT	0.10	-0.42	0.63
3 Outpatient Imp	de Wit 2012	SST	-0.46	-0.91	-0.02
3 Outpatient Imp	Bersani 2013 1	SST	-0.83	-1.42	-0.23
3 Outpatient Imp	Boisseau 2013	IGT	-0.13	-0.74	0.48
3 Outpatient Imp	Dittrich 2013	CGT	-0.78	-1.65	0.09
3 Outpatient Imp	Grassi 2015	IGT	-0.46	-0.91	-0.01
3 Outpatient Imp	Lei 2015	SST	-0.64	-1.07	-0.20
3 Outpatient Imp	Zhang 2015b 3	IGT	-0.78	-1.16	-0.40
3 Outpatient Imp	Zhang 2015b 4	IGT	-0.72	-1.07	-0.37
3 Outpatient Imp	Zhang 2015c 1	IGT	-0.74	-1.13	-0.36
3 Outpatient Imp	Fan 2016 1	SST	-0.53	-1.01	-0.05
3 Outpatient Imp	Fan 2016 2	SST	-0.42	-0.88	0.04
3 Outpatient Imp	Lei 2017 1	SST	-0.67	-1.13	-0.20
3 Outpatient Imp	Lei 2017 2	SST	-0.74	-1.27	-0.21
3 Outpatient Imp	Norman 2017	TD	-0.20	-0.81	0.41
3 Outpatient Imp	Saremi 2017 2	Go-no/go	-1.89	-2.44	-1.33
3 Outpatient Imp	Zhang 2017	IGT	-0.26	-0.71	0.18
3 Outpatient Imp	Grassi 2018	IGT	-0.56	-1.00	-0.11
3 Outpatient Imp	Kertzman 2018	Go-no/go	-0.17	-0.47	0.14
3 Outpatient Imp	Martoni 2018 1	SST	-0.07	-0.53	0.39
3 Outpatient Imp	Norman 2018	IGT	-0.26	-0.87	0.35
3 Outpatient Imp	Grassi 2019	IGT	-0.69	-1.13	-0.25
3 Outpatient Imp	Luo 2020	IGT	-0.62	-1.18	-0.06
3 Outpatient Imp	Thorsen 2020	SST	0.01	-0.58	0.60
3 Outpatient Imp	Chen 2021 2	SST	-0.27	-0.79	0.24
3 Outpatient Imp	Martinez-Esparza 2021 2	Go-no/go	-0.46	-0.96	0.04
3 Outpatient Imp			-0.47	-0.60	-0.34
4 Inpatient Imp	Cavedini 2002	IGT	-0.66	-1.14	-0.17
4 Inpatient Imp	Cavallaro 2003 1	IGT	-0.98	-1.35	-0.61
4 Inpatient Imp	Ruchsow 2005	Go-no/go	0.28	-0.53	1.09
4 Inpatient Imp	Cavedini 2010 1	IGT	-1.44	-1.97	-0.90
4 Inpatient Imp	Krishna 2011 1	IGT & CCPT-II	0.11	-0.43	0.64
4 Inpatient Imp	Cavedini 2012	IGT	-1.62	-2.34	-0.90
4 Inpatient Imp	Kang 2013	SST	-0.96	-1.64	-0.29
4 Inpatient Imp	Kim 2015 1	IGT	-0.46	-0.82	-0.11
4 Inpatient Imp	Martoni 2015	IGT	-0.24	-0.46	-0.03
4 Inpatient Imp	Cillo 2019	CGT	-0.27	-0.55	0.01
4 Inpatient Imp	Frydman 2020	CGT & SST	-0.21	-0.66	0.24
4 Inpatient Imp	Kim 2020 3	CGT	-0.10	-0.49	0.29
4 Inpatient Imp	Kim 2020 4	CGT	-0.25	-0.64	0.13
4 Inpatient Imp			-0.50	-0.74	-0.26
5 Mixed recruitment Imp	Herrmann 2003	Go-no/go	0.00	-0.77	0.77
5 Mixed recruitment Imp	do Rocha 2011 1	IGT & CCPT-II	-0.65	-0.85	-0.46
5 Mixed recruitment Imp	Kashyap 2013 1	IGT	-0.56	-0.84	-0.28
5 Mixed recruitment Imp	Yu 2019	SST	-0.40	-0.94	0.14
5 Mixed recruitment Imp	Bhattacharya 2021 1	SST	-0.53	-0.98	-0.08
5 Mixed recruitment Imp	Bhattacharya 2021 2	SST	0.01	-0.43	0.45
5 Mixed recruitment Imp			-0.45	-0.66	-0.23
Overall			-0.47	-0.57	-0.37



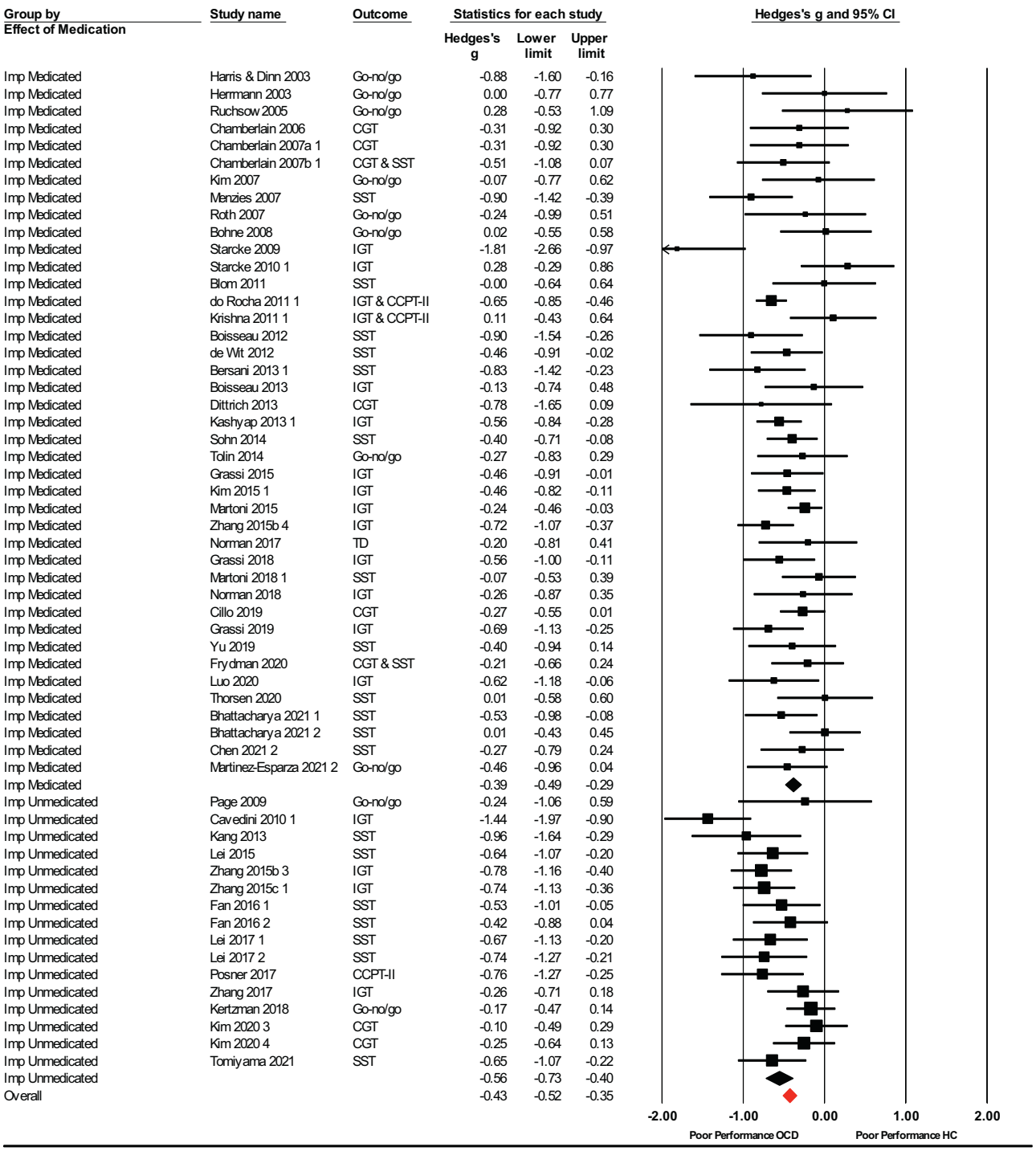
Appendix 5

Subgroup analysis of the effects of medication on compulsivity



Appendix 6

Subgroup analysis of the effects of medication on impulsivity



Appendix 7

Listed comorbidities in included studies.

Study names	Comorbidities reported
[1]	17 of 33 had a comorbid disorder; exact disorders not specified.
[17]	MDD ($n = 4$), Chronic tic ($n = 1$), Schizoaffective disorder ($n = 1$).
[141]	MDD ($N = 10$), Anxiety disorder (n = 4).
[106]	MDD ($n = 5$), Phobia (n = 1), Bulimia Nervosa (n = 1)
[49]	MDD (n = 1)
[24]	10 reports of comorbid disorder; exact disorders not specified.

(*continued on next page*)

Appendix 7 (*continued*)

Study names	Comorbidities reported
[116]	Avoidant PD (n = 10), MDD (n = 8), Dysthymia (n = 7), Obsessive-compulsive PD (n = 6), Depressive PD (n = 5), Paranoid PD (n = 3), Negativistic PD (n = 2), Borderline PD (n = 2) Social phobia (n = 2), Schizoid PD (n = 1) Specific phobia (n = 1), Panic disorder (n = 1), Panic disorder with Agoraphobia (n = 1), PTSD (n = 1), GAD (n = 1), Hypochondriasis (n = 1), BDD (n = 1)
[180]	MDD (n = 5), Anxiety disorder such as social phobia, specific phobia, and panic disorder (n = 3), Both a depressive and anxious disorder (n = 6), Binge eating disorder with a depressive and anxiety disorder (n = 1)
[108]	Depression (n = 2)
[171]	MDD (n = 2)
[25]	11 cases of comorbidity; not specified.
[164]	MDD (n = 15), Suicide risk (n = 5), Panic disorder (n = 4), Agoraphobia (n = 2), GAD (n = 2), Social phobia (n = 1)
[155]	Dysthymic disorder (n = 2), past history of MDD (n = 3), Alcohol dependence (n = 1).
[22]	Depression (n = 10), Eating disorder (n = 1), ADHD (n = 1)
[32]	MDD (n = 35), Dysthymic disorder (n = 4), Panic disorder (n = 2), Social phobia (n = 15), Specific phobia (n = 16), GAD (n = 11), Substance abuse (n = 2)
[58]	Social phobia (n = 22), GAD (n = 22), Agoraphobia (n = 19), Depressive disorder (n = 16), Panic disorder (n = 15), Bipolar II disorder (n = 2).
[112]	Dysthymia (n = 4), Specific phobia (n = 3), Social phobia (n = 2), GAD (n = 2).
[27]	MDD (n = 1), MDD & Social Phobia (n = 1), Panic disorder (n = 1), GAD (n = 1).
de Wit et al., 2012	22 reports of comorbid disorder; exact disorders not specified.
[28]	40% of the sample had a comorbid disorder; exact diagnoses and counts not given.
[63]	Panic disorder (n = 5), MDD (n = 4), GAD (n = 4), Agoraphobia (n = 3), Specific phobia (n = 2), Social phobia (n = 1), Hypochondriasis (n = 1)
[103]	Any co-morbid Axis I disorder (n = 92), Any depressive disorder (n = 75), Any anxiety disorder (n = 41), MDD (n = 44), Dysthymia (n = 39), Recurrent depressive disorder (n = 9), Social phobia (n = 21), GAD (n = 15), Panic disorder (n = 10), Specific phobia (n = 2), Post-traumatic stress disorder (n = 1), Eating disorder (n = 2), Hypochondriasis (n = 2), Somatoform disorder (n = 6), Dissociative disorder (n = 1), Schizotypal disorder (n = 2), Body dysmorphic disorder (n = 1), Other disorders (n = 3)
[203]	Anxiety disorders (n = 8), Depressive disorders (n = 8).
Kim et al., 2015	MDD (n = 8), Panic disorder (n = 3), Bipolar II Disorder (n = 2), Tic disorder (n = 2), Social phobia (n = 1), BDD (n = 1)
[161]	History of MDD (n = 4), Social phobia and history of MDD (n = 2), Specific phobia (n = 1), Social phobia (n = 1), Special phobia, Social phobia, and history of MDD (n = 1), Binge eating disorder, Social phobia, and history of MDD (n = 1)
[129]	Anorexia Nervosa (n = 1), Tourette syndrome (n = 1), Skin picking disorder (n = 1), Gambling disorder (n = 1), Panic Disorder (n = 1), MDD (n = 1), Trichotillomania (n = 1), Hoarding disorder (n = 1)
[50]	Social Phobia = 1; Mood disorders = 4; Dysmorphophobia = 1.
[85]	MDD (n = 6), Anxiety disorders; panic disorders (n = 2), Social anxiety disorder (n = 2), OCD spectrum disorders; Body dysmorphic disorders (n = 1), Hoarding disorders (n = 1), Chronic tic disorder (n = 4).
[26]	GAD (n = 3), Specific phobia of crowds (n = 1), Social phobia (n = 1)
[124]	OCPD; exact count not specified.
[191]	MDD (n = 9), GAD (n = 9), Social anxiety disorder (n = 7), Specific phobia (n = 4), Panic disorder with and without Agoraphobia (n = 3), Hypochondriasis (n = 3), Dysthymia (n = 2), PTSD (n = 1), ADHD (n = 1), Somatisation disorder (n = 1), Pain disorder (n = 1).
[21]	**Familial OCD:** MDD (n = 25), GAD (n = 9), Dysthymia (n = 6), Tic disorder (n = 6), Social anxiety disorder (n = 4), Panic disorder (n = 3) **Sporadic OCD:** MDD (n = 23), Dysthymia (n = 6), GAD (n = 6), Social anxiety disorder (n = 6), Panic disorder (n = 5), Tic disorder (n = 3)
[48]	Depression (n = 10), Social phobia (n = 2), Panic disorder (n = 2), Generalised anxiety disorder (n = 2), Bulimia Nervosa (n = 0), Tourette's syndrome (n = 3), Trichotillomania (n = 1).
[143]	Depression (n = 11), Anxiety (n = 1)

Foot notes: OCD = Obsessive-Compulsive Disorder, PD = Personality Disorder, MDD = Major Depressive Disorder, GAD = Generalised Anxiety Disorder, BDD = Body Dysmorphic Disorder.

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