Executive Function in the OCD Spectrum: Phenomenology, Neurocognition, and the Development of a New Assessment Tool

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CHAPTER 1. GENERAL INTRODUCTION

Obsessive-compulsive disorder (OCD) is a psychiatric condition characterized by intrusive, distressing thoughts (obsessions) and repetitive, ritualistic behaviours (compulsions) performed to alleviate anxiety (APA, 2013). These compulsions, often excessive and time-consuming, provide only temporary relief, leaving individuals feeling trapped in a cycle of distress and ritualized behaviour (Eisen et al., 2006; Zisler et al., 2024). Obsessions commonly centre around themes such as aggression, contamination, symmetry, magical thinking, somatic concerns, religion, and sexuality, while compulsions typically involve checking, washing, repeating, ordering, counting, or hoarding (Hunt, 2020). These intrusive thoughts have an overwhelming nature and are ego-dystonic — experienced as alien or inconsistent with the individual's self-perception—further exacerbating distress (Veale & Roberts, 2014). Many individuals with OCD report a diminished sense of agency, feeling unable to resist compulsions or delay their enactment without experiencing intense anxiety (van Oudheusden et al. 2018). The disorder, therefore, is not simply a matter of habit but rather a profound struggle between intrusive thoughts and the perceived necessity of compulsive actions to prevent distress or avert imagined harm. OCD now falls under the diagnostic category of Obsessive-Compulsive and Related Disorders (OCRDs), but this was not always the case. The Diagnostic and Statistical Manual of Mental Disorders III (DSM-III) classified OCD as an anxiety disorder owing to the prominent role of anxiety in driving compulsions (APA, 1980). Within that framework, clinicians viewed compulsions as a byproduct of excessive harm avoidance. As OCD consisted of features existing outside of the diagnostic category for anxiety disorders, but very much within the confines of the OCRDs category, OCD was moved. However, this change did not occur until the 5th edition of the DSM in 2013 after two further editions in 1994 and 2000 of incorrect classification (APA, 1996; 2000). This move was facilitated by numerous papers comparing and contrasting the

phenomenology of OCD with other OCRDs such as Trichotillomania and Dermotillomania (otherwise known as hair pulling disorder and skin picking disorder, respectively) (Storch, Abramowitz, and Goodman, 2008; Hollander, Braun, and Simeon, 2008; Phillips et al, 2010; Stein et al, 2010). The profile of OCD is synonymous with this category of its own namesake. The underlying feature that is shared across this collection of disorders is the function of the compulsion; the compulsion is performed to relieve obsessional or anxious plight whether this be hair pulling, skin picking, hand washing, or object arrangement. This feature is quite interesting as this would supplant the compulsion as the primary issue of the disorder instead of the obsession (Gillan and Robbins, 2014). Indeed, OCD can be viewed as the very archetype, the paradigmatic disorder, for this diagnostic category of OCRDs.

Indeed, many measures of OCD symptomatology exist: the Yale-Brown Obsessive-Compulsive Scale (Goodman et al, 1989), the Obsessive-Compulsive Inventory-Revised (Foa et al, 2002), the Maudsley Obsessive-Compulsive Inventory (Hodgson and Rachman, 1977), and the Padua Inventory (Sanavio, 1988; Burns, 1995) all converge on common obsessions and compulsions, in both sub-clinical and clinical cohorts, and offer a rating of the severity of such symptomatology. As outlined in the Obsessive-Compulsive Inventory-Revised (OCI-R; Foa et al, 2002), compulsions are segregated into cleaning, checking, and ordering. Rowsell and Francis (2015) offered an elaboration on this whereby harm-related obsessions were paired with checking compulsions, symmetry obsessions with ordering compulsions, hoarding behaviours, and obsessions without compulsions. The Padua Inventory (Sanavio, 1988) provides a similar set of subscales, checking compulsions and contamination obsessions and washing compulsions, and also offers an elaboration on obsessional thoughts and impulses to harm to oneself or others. Additionally, the Yale-Brown Obsessive-Compulsive Scale-II (Storch et al, 2010; Rowa et al, 2025) held depictions of obsessions of aggressive, sexual, and religious natures.

In this work, patients with OCD are defined as individuals that have received a formal diagnosis of OCD, with symptomatology meeting the diagnostic criteria outlined in either the DSM-5 or the ICD-11. See Table 1.1 for OCD diagnostic criteria.

Table 1.1. A comparison of diagnostic criteria for OCD between the DSM-5 and ICD-11.

DSM-5 criteria	ICD-11 criteria
A) Presence of obsessions, compulsions, or both	A) Presence of obsessions, compulsions, or both
B) The obsessions or compulsions are time-consuming (e.g., take more than 1 hour per day) or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.	B) The obsessive-compulsive symptoms must be recognized as the individuals own thoughts or feelings
C) Obsessive-compulsive symptoms are not attributable to the physiological effects of a substance or another medical condition.	C) There must be one intrusion that is still resisted unsuccessfully, even though others may be some which the sufferer no longer resists
D) Obsessive-compulsive ssymptoms are not better accounted for by the diagnostic criteria of another mental health disorder	D) The thought of carrying out the compulsion must not in itself be pleasurable (simple relief of tension or anxiety is not regarded as pleasure in this sense)
	E) The intrusions must be unpleasantly repetitive

Although these classification systems provide a reliable diagnostic framework across contexts, there are a number of limitations. Categorising disorders in this format may reflect a set of heterogenous syndromes rather than discrete disorders, which may in turn impede translational research and clinical practice (Kapadia, Desai, and Parikh, 2020). As of the varied ways in which individuals may qualify for a symptom based diagnosis, some may satisfy the criteria for the same diagnosis but with differing symptom presentations. Indeed, a high level of heterogeneity emerges in the presentation of OCD and the distinctness between patients might be likened almost to the uniqueness of their fingerprints (Lochner and Stein,

2003; Hasanpour et al, 2017). Each patient has a unique composition of obsessions and compulsions with differing content and severity. This has implications for an already overstretched mental health care sector and may incur misdiagnosis (Kyrios, Moulding, and Jones, 2010; Drummond et al, 2018; Perez et al, 2022; McLeod et al, 2024), given that patients that meet the criteria for one disorder often meet the criteria for another (known as comorbidity) leading to further diagnostic heterogeneity.

The documented heterogeneity of OCD has resulted in a call for the establishment of homogenous groupings of OCD phenomenology (de Mathis et al, 2006). Numerous heuristic subgroups of OCD phenomena have been presented each with their unique profiles of symptom severities, comorbidities, and treatment responses. The motivations underpinning particular symptom expressions can be used to isolate subtypes of OCD (Summerfeld et al, 2014). The primary motivation underlying washing compulsions is harm avoidance, which is driven by overestimations of potential contagions, contamination, and illness, whereas, ordering and exactness compulsions are motivated by incompleteness; correcting minute flaws in patterns and sequences helps to quell the obsessional anxieties (Pietrefesa and Coles, 2008; Taylor et al, 2013). The age of onset of OCD can also be used to segment phenomenology as an earlier age of onset is associated with greater prevalences in males (Mathes, Morabito, and Schmidt, 2019) and a higher degree of comorbid tics (Nakatani et al, 2011) compared to a later age of onset. Interestingly, the comparison between OCD with and without tics has also provided a device of dividing OCD phenomenology; whilst the two subgroups do not differ in the contents of the obsessional symptoms, those with tics have fewer cleaning rituals than their counterparts (Holzer et al, 1994). These homogenous subgroups have provided some utility in addressing such heterogenous presentations though are still subject to inter-individual variability (Chamberlain et al, 2005; Fineberg et al, 2010).

Using diagnostic categories may also have implications for treatment provisions as they reflect severe conditions that are difficult to treat. As Cognitive Behavioural Therapy with Exposure and Response Prevention (CBT with ERP) is typically offered as the first instance (as reported in the National Institute of Clinical Excellence (NICE) guidelines; for a recent review of efficacy, see Reid et al, 2021), the service provision can only stretch so far to cater to the particular needs of the patient and so there may be aspects (such as particular symptomatic compositions) that reside outside of the care capacity that remains unchallenged, which in turn may impact treatment adherence (Santana, et al, 2013; Schwartz, 2018; Leeuwerik, Cavanagh, and Strauss, 2019; Tjelle et al, 2021).

Crucially, there may be significant overlaps between those that do and do not qualify for a diagnosis of OCD; using a dichotomous 'all or nothing' symptom criteria count may obscure fundamental information concerning the emergence of OCD, its risk factors, and how preventative interventions can be produced and implemented (Hengartner and Lehmann, 2017). As such, scrutinising dimensional and developmental trajectories of psychopathology may identify integral markers for the predisposition and precipitation of OCD.

As patients with OCD occupy the extreme end of a continuum of symptom severity, establishing symptomatic profiles and phenomenological compositions before formal diagnosis is crucial for isolating dimensional trajectories for pathogenesis (Fullana et al, 2010a). This continuum of obsessive-compulsive phenomena ranges from everyday occurrences and experiences such as doubt, perfectionism, and concerns for cleanliness. For some, however, these phenomena increase in frequency, intensity, and interference with daily life, escalating into the severe and disabling symptoms of OCD (Apter et al, 1996; Fullana et al, 2010b). Those whose symptomatic profiles exist before this point of diagnosis are referred to in this work as subclinical participants; those with OC tendencies who score highly on self-rated measures of OC symptoms and severity (such as the OCI-R) but do not qualify for

diagnosis. Subclinical OCD symptomatology, whilst being greater than that of normal/healthy individuals, lacks the significant functional impairment that is so disabling in clinical manifestations of the disorder (Johansen and Dittrich, 2013), though still negatively impacting quality of life. With subclinical OCD compositions comprising symptoms in the absence of functional impairment, it stands to reason that it is the combination of symptoms and functional impairment that contributes to the clinical condition of OCD and delineates the diagnostic partition (Vorstenbosch et al, 2012; Storch et al, 2014). Subclinical samples comprise salient pathogenic markers that may be scrutinised to identify the onset of these functional impairments.

The Research Domain Criteria (RDoC) framework represents a move away from traditional diagnostic categories by integrating diverse phenomena to identify risk markers associated with the onset and perpetuation of psychopathology (Cuthbert, 2022; Morris et al, 2022). These phenomena pertain to: development across the lifespan, environmental and social factors, and functional systems such as regulation, sensorimotor, cognitive control, positive and negative valence systems. Positive valence systems are responsible for responses to positive motivational contexts such as reward seeking, whilst negative valence systems are conversely responsible for responding in contexts of fear, loss, threat and avoidance (Cuthbert and Insel, 2013). As the framework is reliant upon empiricism to facilitate an evidence-based approach to neuropsychiatric and biobehavioural research (Fluyau, 2018), units of analysis are employed to provide standardised metrics on the described domains of functioning; these being imaging paradigms, self-rating tools and neurocognitive performance tasks (Shankman and Gorka, 2015). This framework provides a consolidation of the neuropsychological basis of symptomatic manifestations from across multiple mental health disorders, which is otherwise known as transdiagnosis (Miller and Rockstroh, 2013).

The study of transdiagnostic features is a method of isolating neurobiological markers with a partial exclusivity of phenomena to a collection of psychiatric disorders to establish the commonalities that will ultimately formulate and characterise these transdiagnostic phenotypes (Dalgleish et al, 2020). By applying the transdiagnostic RDoC framework to the phenomenology of OCD, there is of course direct relevance given the idiosyncrasies of the disorder. Emerging empirical evidence recognises OCD as a disorder of deficient neurocognitive faculties (Menzies et al, 2007; Trivedi et al, 2008) with some papers labelling OCD as a neurodevelopmental disorder akin to ASD and ADHD (Factor and Karp, 2024). Indeed, the past 18 years has seen increased attention paid to these functional deficits of OCD as integral aspects of the phenomenology, which has implications for both assessment and for the subsequent development of novel treatments (Chamberlain et al, 2007). The study of executive functioning lends itself to scrutiny in this respect as breakdowns in the structure, integrity, and function of neurological regions responsible for particular cognitive and behavioural outputs have been reported and presented as novel markers for studying a number of neuropsychiatric conditions such as ASD, ADHD, and OCD (Berger et al, 2016; Duijkers et al, 2016; Soraggi-Frez, 2017).

As Rabinovici et al (2015) described, executive function pertains to a collection of latent cognitive abilities that are responsible for successful goal-directed behaviour and adaptation to changing environments. Executive functioning develops early and matures between adolescence to early adulthood, parallel to the development of the prefrontal cortex (Diamond, 2013; Tervo-Clemmens et al, 2023). These abilities include self-monitoring, planning and adjusting behaviour appropriately given the present context, and inhibiting any behaviour that is irrelevant or incongruent with current goals; executive functioning is the governing and regulation of internal mechanisms to manage and meet external demands (Bedard et al, 2009; Suchy, 2009). These abilities can be arranged into lower- and higher-

order functions (Bedard et al, 2009). Attentional control, cognitive flexibility, response inhibition, and working memory constitute lower-order functions (Friedman and Robbins, 2021). Higher-order functions are comprised of problem-solving, fluid intelligence, reasoning, and planning and are formed of the integration and collaboration of the lower-order functions. As such, should there be any dysfunction within the lower-order abilities, this would in turn effect the integrity of the higher-order abilities resulting in functional impairment; deficits in the described goal-directed behaviour (Ustun and Kennedy, 2009). As dysfunction in these latent executive abilities contribute to, and underpin, the phenomenological and symptomatic manifestations of OCD (Kashyap et al, 2013; Snyder et al, 2015) and are linked to psycho-social stressors (Bouchard and Saint-Aubin, 2014; Perez et al, 2016; Ribner et al, 2017), it is necessary to unpick this relationship between executive functioning and psychopathology as it may hold key pathogenic potential.

Latent cognitive phenotypes may represent such putative transdiagnostic markers for executive control as they constitute discrete observable and measurable cognitive profiles that are linked to established neurobiological processes that underpin the phenomenological manifestations of OCD (Gillan, Fineberg, and Robbin, 2017). Latent cognitive phenotypes exist between the genetic and environmental origins of the disorder and so the manifestation of such phenotypes may be less susceptible to inter-individual variability that other markers may otherwise be subject to (Bilder et al, 2009; Bearden et al, 2017). These homogenous markers of OCD may provide an alternative to the pitfall of the previously discussed symptom-based nosology and can be exploited for clinical utility (Suhas and Rao, 2019; Vaghi, 2021).

As defined in work by Gillan and Robbins (2014), OCD is a disorder of dysfunctional goal-directed behaviour. OCD is demarked by its diminished autonomy over cognitive resources

and the allocation of such resources to (1) inhibit the initial responding to negative intrusions no matter the obsessional theme, and (2) to cease the responding once the distress has been neutralised. As such, the previously described cognitive control and positive and negative valence systems of RDoC have crucial relevance to the formulations of the neuropsychological underpinnings of OCD symptomatology and phenomenology. The obsessions, though ego-dystonic in nature and produce distress (Eisen et al, 2006), are overshadowed by the impact that the compulsions present in being disabling as to consume the patients time, energy, and in essence, quality of life (Stengler-Wenzke et al, 2007) demonstrating the functional impairment as of the breakdown in executive control. Converging evidence implicates OCD as the repetitive execution of stereotyped maladaptive behaviours, as well as the loss of inhibitory control of actions and thoughts that can be respectively referred to as compulsivity and impulsivity (Fineberg et al, 2010; Fineberg et al, 2014). Both compulsivity and impulsivity have been linked to deficits in the orbito- and ventromedial prefrontal cortices and are explained as comprising of a loop motif between an overfiring of the caudate nucleus and an underactive prefrontal lobe resulting in the manifestation of disordered goal-directed behaviour (Gisiger and Boukadoum, 2018).

Though there is some inconsistency as to an exact standardised and operationalized definition of compulsivity, as of its various manifestations across clinical disorders of problematic behaviour (Luigjes et al, 2019), there is convergence on this construct representing a drive to perform repetitive habitual actions according to preestablished rigid rules (Albertella et al, 2020). This is not to be confused with the compulsion within OCD symptomatology; compulsivity is the drive underlying the ritualized act, and the compulsion is the very act itself. Moreover, the phenotype of compulsivity is borne of a deficit in the previously mentioned executive function, cognitive flexibility (Benzina et al, 2021; de Souza, Pellegrini, and Fineberg et al, 2024). Cognitive flexibility is the effective modification of thinking and

behaviour to meet contextual demands based on changing environments and information, as well as successfully switching between various tasks and concepts (Dajani and Uddin, 2015; Hohl and Dolcos, 2024). Deficiencies in this executive capacity result in a diminished ability to successfully shift attention between activities, situations, and stimuli (Ionescu, 2012), manifesting in a compulsive disposition demarked by tendencies of perseveration, resistance to change and an inability to adapt; referred to as cognitive inflexibility (Ionescu, 2012).

Although this deficient flexibility is observed across psychiatric disorders with compulsive features, such as Anorexia Nervosa, Body Dysmorphic Disorder, and Trichotillomania, there is a prominent deficit in OCD (Chamberlain et al, 2006; Bohon et al, 2020; Chen et al, 2021). This can be observed within the persistent engagement with ritualised acts despite them being distressing and disabling (Gruner and Pittenger, 2017). The deficient flexibility prevents an individual from disengaging from the behaviour or act despite having completed the task, in this case the task being the neutralising of the obsessions distress (Gu et al, 2008; Zor et al, 2011). For example, those with obsessions of contamination engage in excessive hand-washing resulting in irritated and damaged skin (Monti, Sambvani, and Sacrini, 2006). The perseverance takes the behaviour past a point of completion; they are unable to shift their attention away from this behaviour on to something else.

As the phenotype of compulsivity and its underpinning executive deficit, cognitive inflexibility, constitute a core feature of OCD phenomenology, ample measures have been designed to suit its assessment within research and clinical practice; they are quantified by commentaries on self-rating tools and performance on neurocognitive tasks.

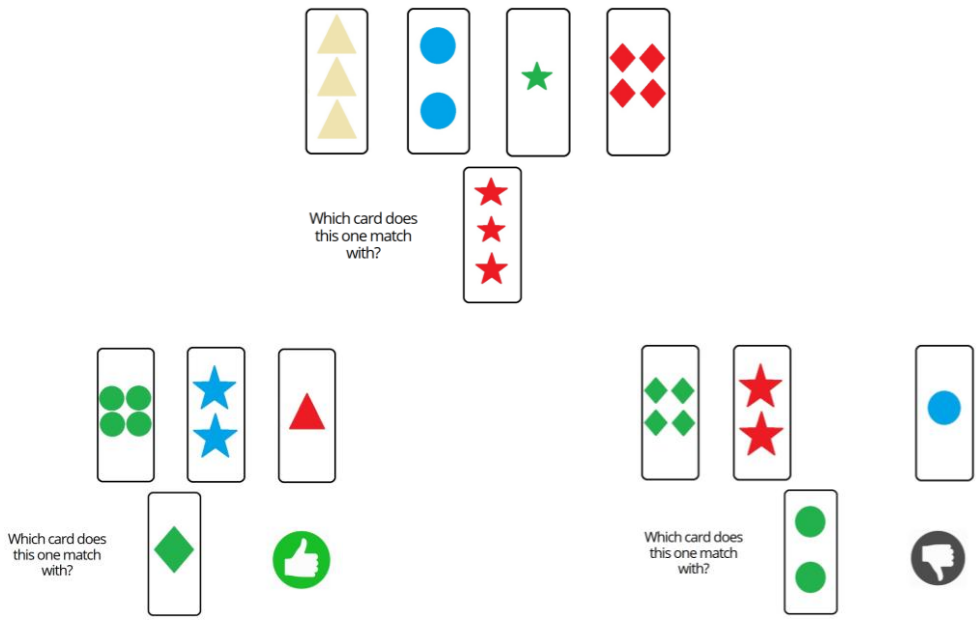
The Compulsive Personality Assessment Scale (CPAS; Fineberg et al, 2007) and the Cambridge-Chicago Compulsivity Trait Scale (CHIT; Chamberlain and Grant, 2018) are concise questionnaires that provide a transdiagnostic assessment of compulsivity. That is to

say that the items constituting these scales harbour limited specificity to any one particular disorder and may in fact be used to quantify compulsive phenomena across a range of psychiatric conditions. These psychometric scales utilise predetermined scenarios with which participants allocate a rating based on the relevance of the questions compared against their own experiences and behaviours; for example, the CPAS asks "*Would you describe yourself as a perfectionist who struggles with completing the task at hand?*" to which a respondent will allocate a rating according to following Likert scale " 0=absent, 1=mild, 2=moderate, 3=severe, and 4=very severe". Moreover, the topic of compulsivity also permeates the subfactors of many other scales such as: compulsive checking and grooming in the Padua Inventory (PI: Sanavio, 1988; Burns, 1995); the rigidity and difficulty with change in the Pathological Obsessive-Compulsive Personality Scale (POPS; Pinto, Ansell, and Wright, 2011); the behavioural and cognitive rigidity domains of the Montefiore-Einstein Rigidity Scale-Revised (MERS-R; Taylor et al, 2022); and the perfectionism, flexibility, and compulsions items of the Cognitive Assessment Instrument of Obsessions and Compulsions (CAIOC-13; Dittrich, Johansen, and Fineberg, 2011). Where self-ratings deliver an assessment by commentary, neurocognitive tasks examine the phenotype of interest under an applied scenario so as to observe the underlying deficit.

The Wisconsin Card Sorting Test (WCST; Milner, 1963) is one such neurocognitive task of cognitive flexibility under a rule matching paradigm. Participants are required to match a given target card to one of four other cards; and to achieve this, participants learn the rule(s) governing matching through trial and error. The matching rule may depend on shape (e.g., matching squares with squares or stars with stars), colour (e.g., blue with blue) or number (e.g., three shapes matched with three shapes). After 8 trials, the matching rule changes (without warning) requiring participants to adapt their answers and learn the new matching rule and reject the previous one. Participants will receive visual feedback on the accuracy of

their responding which will highlight either a correct response or an incorrect response; the latter encourages a change of matching method. The task includes 64 randomised trials: 8 trials involving 8 different rules (see Figure 1.1) . The typical outcome metrics of the WCST are: total errors made, perseverative errors (the participant maintains the previous matching rule despite being informed that this is incorrect), failure to maintain set (an error in the matching rule after a run of correct trials, or a return to a previous matching rule), and non-perseverative errors (errors that do not conform to the description of perseverative errors or a failures to maintain set). The perseverative errors metric is typically preferred to index cognitive (in)flexibility due to its close resemblance to this neurocognitive phenotype of maladaptive rigidity and repetition.

Figure 2.1. Example stimuli of the Wisconsin Card Sorting Test.



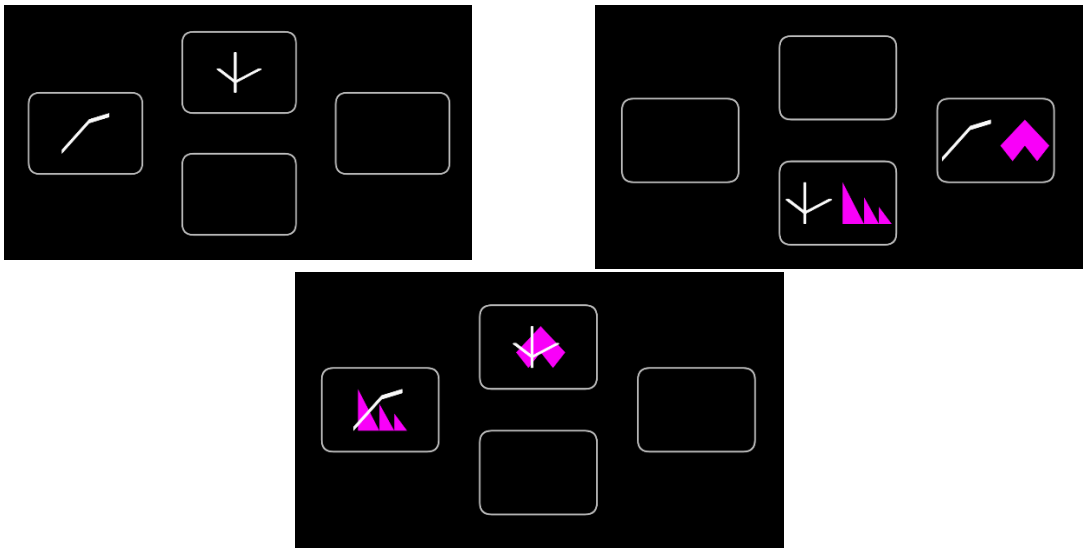
Correct match response

Incorrect match response

Like the WCST, this The Intra-Extradimensional set-shifting task (IDED; Owen et al, 1991) also assesses the ability to flexibly adapt responses in relation to rule acquisition and reversal learning. It comprises nine stages where subjects, through trial and error, must select the

correct paired image, which consists of two different dimensions of stimulus: white lines and pink shapes. After a phase of acclimatization, participants are required to choose the 'correct' shape in the pair. Much like the WCST, the rule changes after several trials forcing the participant to adapt and shift their attention to the acquisition of the new rule. Initial stages of the task reward one of the features of the pair, for instance, a particular shape. Individuals must therefore ignore the lines and attend exclusively to shapes, choosing the correct one. However, the rule (i.e., the correct choice of shape) is reversed once learned. At the intra-dimensional (ID) shift (Stage 6), participants need to adjust their responding to endorse the alternative shape as the new correct selection and let go of the previously correct matching rule. This element of the task approximates to a test of reversal learning. As the task continues, the participant eventually arrives at the Extra-Dimensional shift (EDS; Stage 8), when the rule changes once more. This time, the dimension changes also, so in this example, lines would become the dominant feature, and the participant would need to stop attending to shapes and learn to respond to lines instead. A final stage, the Extra-dimensional shift reversal (EDSR; Stage 9) tests the ability to respond to a further dimensional reversal. Typically used outcome metrics of this task are the number of ED errors at each of the 9 stages, total ED errors, total latency (reaction time), and the number of stages completed. In particular, stages 8 and 9 (EDS and EDSR) measure the ability to effectively switch attentional set and so the ED shift stage is a cardinal measure of attentional flexibility. The relative inflexibility of a participant is thus quantified by the number of errors made at these stages denoting perseverance of incorrect responding despite feedback as to the contrary (see Figure 1.2).

Figure 1.1. Example stimuli of the Intra-Extradimensional set-shifting task.



Patients with OCD exhibit impaired performance on both the WCST and the IDED compared to healthy controls (Lucey et al, 1997; Chamberlain et al, 2021). Though they receive immediate feedback on their responses, patients with OCD are unable to flexibly adapt their actions and behaviours when they know them to be incorrect – this feature exemplifies the compulsive disposition of OCD and underlines the dysfunctional goal-directed behaviour. Research also shows that unaffected first-degree relatives of patients with OCD also demonstrate impaired performances on these tasks compared to healthy controls (Cavedini et al, 2010) thereby exemplifying OCD as a putative neurobiological syndrome of rigid and fixed dispositions.

like compulsivity, impulsivity is observed across psychiatric conditions; it is a transdiagnostic construct that underlies and drives symptomatology (Chamberlain et al, 2019). OCD is considered the archetypal compulsive disorder given its rigid and risk-averse frameworks which conveys a dimensional opposite to a risk-taking impulsive disposition. However, patients with OCD lack the ability to prevent or delay engaging with obsessional and

compulsive symptoms irrespective of consequences (Potenza, Koran, and Pallanti, 2009) which is synonymous with descriptions of impulsivity and speaks towards a deficit in an executive control domain in addition to the described compromised flexibility. As impulsivity presents differently in OCD compared to other conditions, it is vital to unpack the role that impulsivity plays within OCD phenomenology.

Impulsivity is characterised by actions or behaviours performed in the absence of forethought and are often described as risk taking and ill-conceived with minimal consideration for consequences (Hinslie and Shatzky, 1940; Evenden, 1999; Moeller et al, 2001). Impulsivity is not a unitary construct, it is a multifaceted, and as such can be arrived at from various approaches (Knezevic-Budisin et al, 2015; Dalley and Robbins, 2017). Impulsivity may be divided into impulsive action and impulsivity choice, which can be respectively referred to as motor impulsivity and decision-making (decisional) impulsivity. Motor impulsivity concerns the inability to supress an initiated automatic action in which the responding can be described as premature or anticipatory (Chowdhury et al, 2017), whereas decision-making impulsivity relates to decisions that are made without full knowledge of the current situational landscape, with little or no regard for long-term consequences, and with a preference for short-term transient rewards (Zermatten et al, 2005).

These behavioural manifestations of impulsivity are underpinned by deficient executive control, namely response inhibition (Penades et al, 2007). Response inhibition pertains to the executive ability to successful suppress and mitigate unwanted thoughts or automatic behaviours that are incongruent or inappropriate given the situational context or particular goals of focus (Logue and Gould, 2014). Deficient response inhibition can then be defined as the diminished capacity to stop, prevent, or delay an action or thought; and it is in the absence of refined response inhibition that prepotent actions or thoughts cannot be controlled or substituted for more adaptive alternatives (Mar et al, 2021).

Both divisions of deficient response inhibition (motor and decisional) can be observed in OCD phenomenology and in neighbouring OCRDs and impulse-control disorders such as ADHD, pathological gambling, and substance use disorders (Ortal et al, 2015; Ioannidis et al, 2019; Krzyszkowiak, Kuleta- Krzyszkowiak, and Krzanowska, 2019; Fontenelle et al, 2021). However, the manifestation of this deficient inhibition observed in OCD is perceived in only particular aspects of symptomatology (Sohn et al, 2014). There is minimal mitigation or postponement of engaging with the compulsion as there is an overwhelming urgency to dispel the obsessional distress and so the manifestation of impulsivity in OCD may better understood as emanating from dimensions of urgency (Johnson, Elliot, and Carver, 2021); acting out of necessity to alleviate stress (Billieux et al, 2010). Where impulsive-control disorder such as Borderline Personality Disorder comprise reactionary mood states driven by an emotion dimension (Carver and Johnson, 2021; Herman, Critchley, and Duka, 2018), the impulsivity in OCD is observed in the responding to negative valence manifesting in avoidant behaviours.

However, current taxonomies (the DSM-5 and the ICD-11) provide little or no appraisal of the impulsive features of OCD and so, are omitted from what otherwise would be a comprehensive assessment of a patient's presenting issues (Grabill et al, 2008; Singh, Anjankar, and Sapkale, 2023). There is value in considering these features parallel to the symptomatology that the patient presents with as current novel treatment models fail to contact these phenotypes and as such are left unchallenged. This may underpin and contribute to relapse rates as these features remain pervasive in the daily lives of patients resulting in readmissions and referrals back into secondary mental health services (Cavedini et al, 2002).

Much like compulsivity, impulsivity is quantified and operationalised through commentaries on self-rating tools and performances on neurocognitive tasks, which are employed to capture deficits in inhibitory control observed across a range of psychiatric conditions.

The most commonly used psychometric scale to estimate impulsivity is the Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, and Barratt, 1995). This is a 30-item self-report scale rated on a Likert scale ranging "1= Rarely/Never, 2= Occasionally, 3= Often, and 4= Almost always/Always" based on a respondent's self-appraisal of the own tendencies; greater scores are indicative of heightened impulsivity. This psychometric can be fractionated into three subscales, each providing a metric of a distinct impulsive component: attentional impulsivity ("*I often have extraneous thoughts when thinking"*) detailing cognitive instability and issues with maintaining attention; motor impulsivity ("*I act on the spur of the moment*") encompassing an inability to supress immediate and automatic actions, and non-planning impulsivity (*"I say things without thinking"*) which concerns issues in self-control and a lack of fore-planning. An alternative to the BIS-11 is the Urgency-Premeditation-Perseverance-Sensation Seeking-Positive Urgency impulsive behaviour scale (UPPS-P; Lynam et al, 2006; 2013). The UPPS-P is also a self-rated measure of impulsivity with a multidimensional factor structure consisting of: Sensation seeking (reward-orientatied), lack of premeditation (forethought), lack of perseverance (contrast to that of perseveration deficits on the WCST), positive urgency, and negative urgency. The Three Factor Impulsivity Index (Carver et al, 2011) also provides a dissection of impulsive displays by focusing on the emotional dimension that drives and permeates 'impulse'. These are: *Feelings trigger action* (FTA) explores impulses emanating from heightened emotions; *Pervasive influence of feelings* (PIF) explores the impact of emotion on motivation and cognitive processes; and *lack of follow through* (LFT) explores non-emotional impulsivity such as a difficulty maintaining focus and distractibility. Though there are perceived convergences between of the subfactors of these scales and OCD symptomatology, particularly attentional impulsivity, negative urgency, and feelings trigger action, a review of psychometric measures of impulsivity by Hook et al (2021) found that these measures were non-specific to particular psychiatric conditions and

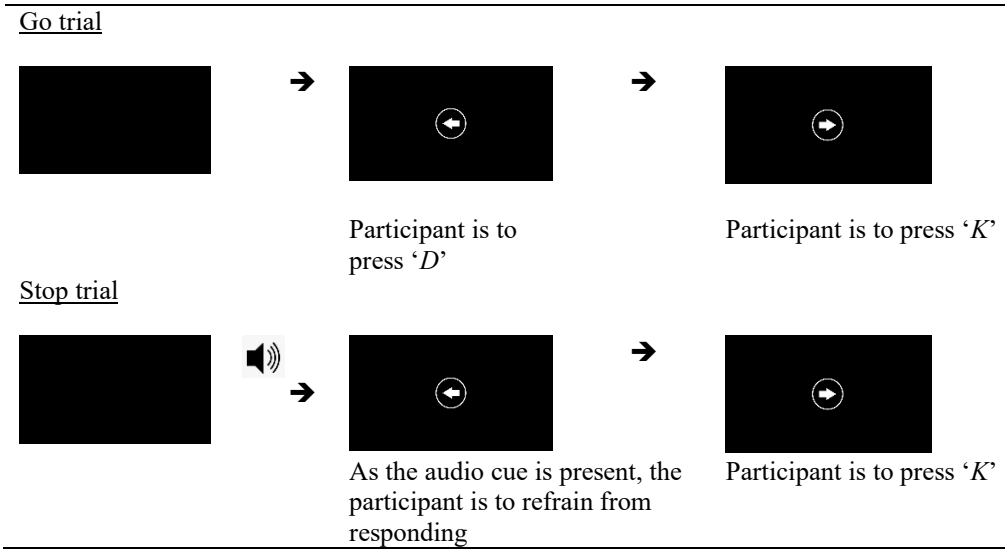
could assess and quantify impulsivity across disorders such as Gambling Disorder, Bipolar 1 disorder, and Attention Deficit Hyperactivity Disorder (Nandagopal et al, 2011; Mestre-Bach et al, 2021).

Interestingly, the Cambridge-Chicago Trait Scale (CHI-T; Chamberlain and Grant et al, 2018), the previously explores transdiagnostic measure of compulsivity beyond the specific obsessive-compulsive symptoms of OCD, taps into behavioural impulsivity ("*I can't resist acting on urges*"), sensation seeking ("*I do things that are immediately rewarding, even if I know it is bad for me in the long run*"), and response inhibition ("*I will keep doing a task over and over until I feel it is done to the highest standards*"). Moreover, the Padua inventory (Sanavio, 1988; Burns, 1995), a self-report measure similar to the OCI-R, provides a representation of impulsive attributes within OCD symptomatology, specifically, within obsessional symptoms ("*I sometimes have an impulse to steal other people's belongings, even if they are of no use to me*") in regard to harming oneself and others that is thematically linked to the motor impulsivity factor of the BIS-11 and the lack of premeditation subscale of the UPPS-P; acting without the consideration of potential consequences.

Alternatively, numerous neurocognitive task paradigms are adapted to operationalise the manifestations of deficient inhibitory control, motor impulsivity and decision-making impulsivity. For motor impulsivity, the most commonly used tasks are the Stop-signal task (Aron, Robbins, and Poldrack, 2004) and the Go/no-Go task (Gomez, Ratcliffe, and Perea, 2007), These tasks measures estimate the ability to successfully inhibit a pre-potent action when given audio-visual cues; participants are required to refrain from responding to stimuli when an audio cue is given despite instructed directives to respond only under certain conditions. Participants are required to press a pre-specified button on the keyboard in response to the presentation of an arrow on the screen, though only of a specified orientation (e.g., press D for arrows pointing left and press K for arrows pointing right); this is the go

trial. Conversely, the participant is to refrain from answering if an audio que is present despite the arrow pointing in the correct direction; this is the stop trial (See Figure 1.3). Participants are first presented with a practice run, to become accustomed to audio cues and the requirements of the task. Performance on the practice block is used to calculate the stop signal delay (SSD) for the three test blocks. The presentation of arrow direction as well as the order of go and stop trials is randomised. There are 10 seconds between each test block, during which, participants are given feedback on their performance (error responses, % of correct responses, Average response time, stop signal errors, and % of correct stop signals). The task lasts 15 minutes in duration. Performance is estimated using the Stop Signal Reaction Time (SSRT) metric; this is the standardised measure of successful response inhibition that eliminates the confounding effects that usually befall other reaction time estimates. Thunberg et al (2024) places normative performance ranges on the SST within 187ms(±)27 and 202ms(±)23, anything above this point indicates deficient inhibitory control.

Figure 1.3. Example Stimuli of the Stop Signal Task.



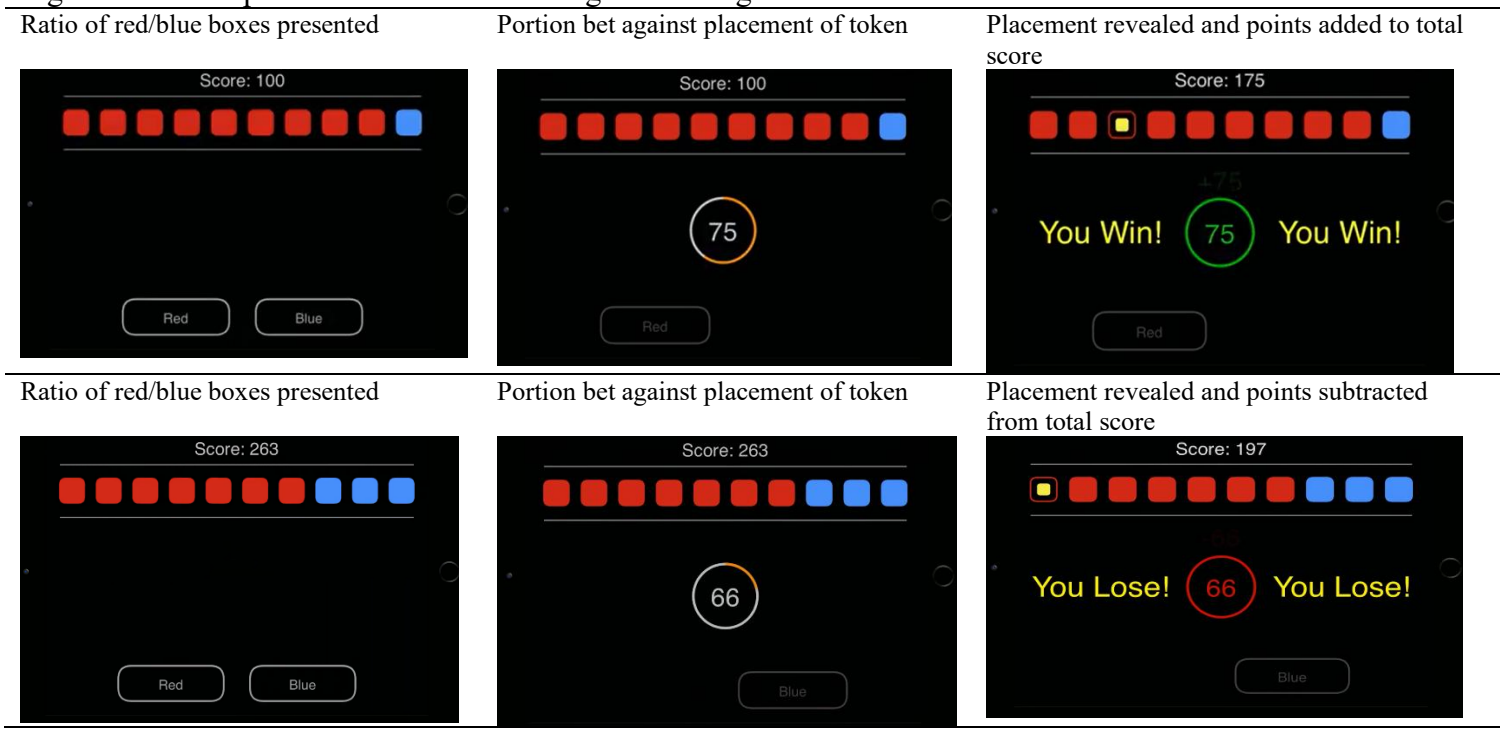
Indeed, patients with OCD have displayed impaired performance on these tasks (Lee, Yost, and Telch, 2009; Berlin and Lee, 2018) implying that they lack the ability to inhibit

responding when actions supersede thought. Some inconsistent evidence however exists as McLaughlin et al (2020) did not find a relation between OCD diagnosis and impaired performance on the SST, while Berlin and Lee (2018) showed a moderate relationship between compulsions, but not obsession, and impaired performance on the SST (r=.356, p<.05). Research indicates that deficient performance on the SST cannot be used as a diagnostic proxy, but when combined with other measures of executive dysfunction such as the IDED, it could support the detection of OCD (Mar et al, 2021).

With regard to decision-making impulsivity, the Cambridge Gambling Task (CGT; Rogers et al, 1999) and the Iowa Gambling Task (IGT; Bechara et al, 1994) are commonly used for an estimation of a diminished decision-making faculty. Participants completing the CGT will be presented with a row of 10 boxes with varying ratios of red to blue boxes (e.g., 2 red to 8 blue boxes) within which a yellow token is concealed. Participants are to choose whether the token is likely to be in a red box or a blue box (e.g., 20% red against 80% blue). For example, if a ratio of 2 red boxes to 8 blue boxes are presented then there is a 20% chance that the yellow token be in a red box and an 80% chance that the token be in a blue box. At the beginning of the task, participants are allocated 100 credits to bet on their choice of token placement being correct; the betting value will be presented onscreen and will increase or decrease incrementally until the participant chooses the portion to bet. Should the token be revealed to be in a box that the participant choose (a correct selection), the betting value will be added to the total credits. However, should the token be revealed to be in a box that the participant did not choose (an incorrect selection), the bettering value will be subtracted from the total credits. Using the previous example, if the participant had predicted that the token is in a blue box and betted 75 points and was correct in this selection, then the previous total amount of credits (100) would increase to 175, though if the token was revealed to be within a red box then the total amount of credits would reduce to 25 (See Figure 1.4). Decisional

manifestations of deficient response inhibition are quantified using the following outcome metrics of the CGT; Quality of decision-making, time taken to decide, risk adjustment, and delay aversion.

Figure 1.4. Example Stimuli of the Cambridge Gambling Task.



These tasks measure the ability to delay an incentive for a greater long-term reward; these tasks measure decision making under risk and ambiguity (Nisitico et al, 2021). However, a stark contrast emerges within the literature on decision-making impairments in patients with OCD. Grassi et al (2018) showed that definitive impairments in decision-making are irrespective of treatment type, and as of this relative independence from treatment, may underpin pervasive implications for functioning in patients with OCD (Cavedini et al, 2002). On the other hand, the conclusions of Summerfeldt et al. (2004) and Abramovitch and McKay (2016) posit OCD as a disorder linked to heightened risk-aversion and premeditative forethought and that impaired performance on decision-making paradigms may be artefacts

of excessive doubting. Indeed, the parameters of impulsivity and underlying deficient inhibitory control within OCD phenomenology are less defined than that of compulsivity.

Self-reports and neurocognitive tasks provide a novel and accessible medium of quantifying these latent cognitive phenotypes across diverse samples, however, they are not without their shortcomings.

The answering patterns and behaviours have a great effect on the representation of the latent trait under investigation, for example the implicit standards with which respondents evaluate their own behaviours may produce differences in responding, and they may not fully comprehend the scale item or may interpret the scale items differently to their intention (Lira et al, 2022). Accuracy of reporting on self-rating scales may also be limited by a lack of self-awareness insofar as respondents not fully comprehending their own motivations or behaviours (Gabriele et al, 2022). As self-reports rely on the memory of respondents being accurate to address the predetermined questions, however, as memory is a constructive and malleable framework, it is susceptible to stress and new information and so recollections of particular traits, emotions, or events may not be accurately represented (Anvari et al, 2002). Respondents also tend to agree with scale items regardless of content and may use extreme responses (Rosenman, Tennekoon, and Hill, 2011). Of course there are the more transient features that may influence accurate recording such as mood and mind set (emotional and cognitive state) at the time of responding to a self-report (Alfonsson, Maathz, and Hursti, 2014). Together, these answering biases may produce spurious and inflated correlations between items that are otherwise distinct and may yield inaccurate representations of the latent trait. Furthermore, further misrepresentation may emanate from the use of self-reports to populate both the predictor and outcome variables which may inflate correlations further, the variance is directly attributed to the use of common methods (common method variance;

Kock, Berbekova, and Assaf, 2021), and so alternative units of analysis are sought after for this source of data, such as the described neurocognitive tasks.

However, neurocognitive tasks are subject to similar interpretative confounds. There may be extraneous variables that influence the performance of a respondent on these neurocognitive indices depending on their environment (e.g., completed at home or in a lab setting). Researchers cannot assume that the reliability of the assessment is constant across contexts, thereby enticing measurement error which subsequently reduces the reliability of neurocognitive tasks (Hedge, Powell, and Summer, 2018). This diminished reliability may arise from a mismatch between the goals of experimental research compared to that examining individual differences. The former aims to demonstrate behavioural effects, whereas, the latter appraise the variability of a latent trait among a testing cohort that is representative of a specified target population. A reliable task measure is achieved where between-participant variance is in excess, however, to achieve substantial power to detect a salient behavioural effect, between-participant variance must be reduced; Hedge, Powell, and Summer (2018) describe this as a '*reliability paradox*'. An example of this us the Stroop effect, a robust effect within the experimental psychological literature in that the vast majority of individuals do in fact show a Stroop effect (Haaf and Rouder, 2017), however, the between-participant variance is low, indicating a concomitant reduced task reliability (Rouder, Kumar, and Haaf, 2019). As such, the most renowned tasks, such as the IDED and the SST, that demonstrate behavioural effects may actually be deficient in appraising individuals differences. The implication here is that although task performance approaches within neuropsychology may produce replicable experimental neurocognitive effects, this may hamper their use as correlational tools. Due to the inconsistent reliability of neurocognitive tasks, they do not make for robust diagnostic tools despite listed as a crucial units of analysis under the RDoC framework for informing diagnostic practices (Dang, King,

and Inzlicht, 2020). Perhaps using these neurocognitive tasks adjacent to their self-report counterparts may address this issue of reliability.

Though these two measures purport to assess the same construct (in this case phenotypic manifestations of deficient executive control), the correlation between self-reports and neurocognitive tasks is often weak and inconsistent (Walvoort et al, 2016; Keen et al, 2020). Weak relationships between modes of assessment are documented across a series of meta-analyses and sizeable investigations from various contexts (Joseph and Newman 2010; Park, Chun, and Lee, 2016; Frey et al, 2017; Murphy and Lilienfeld, 2019), and specifically, measures of executive control and self-regulation show correlations between self-reports and task performances to be no greater than r=0.2 (Saunders et al, 2018; Eisenberg et al, 2019). The lack of correlation between the two modes may emanate from the distinct response profiles that each mode is developed to extract (Dang, King, and Inzlicht, 2020); neurocognitive tasks examine performance whereas self-reports examine perceptions of performance. This is an imperative consideration as some patients with OCD may not perceive themselves to have such compulsive and impulsive dispositions but their performance profiles on objective measures of those very same deficits convey a different effect. Indeed, the inverse may be just as true, insofar as rating themselves as being quite deficient in these faculties that is not objectively reflected owing to under confidences in ability and subsequent overestimations of deficient performance (Janeck et al, 2003; Dar et al, 2022). Certainly, using these two measures in tandem would address potential confounds and effect inflations arising from common method variance, however, numerous papers have used these measures interchangeably, despite their distinction, which may have conflated effects and subsequent interpretations within the literature (Howlett et al, 2023). Alternatively, using these two units of analysis in tandem, with appreciation and

acknowledgement of their respective limitations, may produce a multimodal heuristic for assessing executive dysfunction.

As described, OCD's history is one of inconsistency, with varying diagnostic descriptions and classifications, as well as heterogeneous and idiosyncratic symptomatology. To address this variability, research over the past 18 years has provided a foundation for investigating these latent cognitive phenotypes that underly the manifestation of such variable symptom presentations. Shin, Lee, and Kwon's (2014) meta-analysis of 88 studies examining the executive profile of OCD showed clear deficient performance on tasks probing executive components essential for daily functioning. However, that these broad impairments were of small to moderate sized effects suggesting general impairments. A later meta-analysis of 110 papers by Snyder et al (2015) reported moderate to large effect sizes of executive deficits within the OCD population that were stable across both demographic and clinically salient variables. This inconsistency between syntheses may allude to confounds within the use of neurocognitive tasks as both meta-analyses report the need for a standardised and universal use of these neurocognitive indices and metrics (Gruner and Pittenger, 2017).

Using pooled data from 11 studies (145 healthy controls vs 149 patients with OCD), Chamberlain et al (2021) reported a consistent finding of impaired performance on the IDED in adult patients with OCD (g=0.62[0.28, 0.95]) and made a definitive argument of the destitution of cognitive flexibility as a candidate phenotype for treatment targets. On the other hand, Fradkin et al's (2018) earlier meta-analysis of cognitive flexibility in OCD rendered the inverse insofar as patients with OCD displayed comparable performance to a non-psychiatric control. The notable difference is that the latter synthesis used a collection of tasks probing flexibility whereas Chamberlain et al (2021) focused solely on the IDED. This may speak towards intra-task differences between those of the same executive facet further implying that

they are not wholly interchangeable. This certainly calls for a careful selection of both task and outcome metric (Gruner and Pittenger, 2017).

Regarding the impulsivity phenotype in OCD, Mar et al (2021) conducted a meta-analysis of 21 studies on SST performance, revealing a significant difference between 781 patients with OCD and 837 healthy controls on the SSRT metric of latent response inhibition (raw mean difference =23.4ms [17.42, 29.45], $p<.001$) revealing impaired performance in the clinical group. These findings certainly shows that there is a clear deficit that separates the patients with OCD from a healthy control on a motor dimension of impaired response inhibition. This trend of deficient performance on inhibitory control paradigms is also apparent in meta-analytic comparisons between patients with OCD and a normative comparator on the IGT (Nistico et al, 2021); a task of decisional capacities. Findings revealed that patients with OCD showed deficient decision-making under risk; performance was perhaps hindered by excessive doubt and uncertainty and as a result, decisional inhibitions may form a core underlying feature of the disorder given its manifestation across symptomatic presentations.

The explored meta-analyses clearly show dual impairments of compulsive and impulsive manifestations of executive dysfunction within OCD phenomenology, however, limited work has considered these two phenotypes together. 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 impulsive-compulsive disorders co-occur (i.e., OCD with comorbid substance use disorder), this may contribute to a greater clinical severity (Kashyap et al, 2012; Anger et al, 2015).

In recent years Lopez-Hernandez et al (2023) conducted a review and meta-analysis of 20 studies assessing deficient neurocognitive performance on tasks of flexibility and inhibitory control in children and adolescents with OCD. Their findings revealed impairments in cognitive flexibility (d = - 0.42), and response inhibition across both dimensions of impulsive actions (d = -0.221) and impulsive choices (d = -0.17). Though this goes someway to assessing this phenotypic dichotomy, it is subject to particular shortcomings. The sample comprises children and adolescents with OCD and so, complicates any extrapolation of findings to adult samples with OCD owing to developmental discontinuity of executive control capacities (Geller et al, 2001; Ferguson, Brunsdon, and Bradford, 2021). The authors do not identify the names of the tasks and so it is uncertain if the set used by Lopez-Hernandez et al (2023) are comparable with those reviewed by Chamberlain et al (2021), Mar et al (2021), and Nistico et al (2021). In sum, the different approaches to the assessment of these deficits warrants further exploration into the key relations between putative cognitive phenotypes and OCD symptomatology.

As the response to conventional treatment such as pharmaco- and psychotherapy (i.e., Selective serotonin reuptake inhibitors and CBT with ERP) is so often unsuccessful (Fineberg et al, 2012; Olatunji et al, 2013), latent compulsive or impulsive phenotypes could provide a theoretical basis for developing new interventional targets for OCD and by extension OCRDs given their transdiagnostic properties. 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 (Flessner et al, 2012; Chamberlain et al, 2019; Fineberg et al, 2019; Zartaloudi, Laws, and Bramon, 2019; Romero-Garcia et al, 2021). Further, as key determinants of executive functioning, they may serve as critical markers of functional outcomes. A brain imaging study by Vaghi et al (2017) demonstrated that not all patients with an OCD diagnosis exhibited inflexible set-shifting on

the IDED, 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 latent cognitive 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 (Enkavi et al, 2019), to enable clarification of the specific cognitive latent phenotypes of OCD and OCRDs (Chamberlain et al, 2021). Others have questioned this whole area of research (Kashyap and Abramovitch, 2021) 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 (Abramovitch, Short, and Schweiger, 2021).

As such, this thesis aims to: (1) identify and characterise core domains of executive dysfunction in individuals with Obsessive-Compulsive Disorder (OCD), with a focus on clarifying which specific cognitive impairments are most consistently implicated across studies; (2) examine the methodological variability in the assessment of cognitive phenotypes in OCD, particularly how differences in executive function measures may contribute to inconsistent findings in the literature; (3) evaluate the validity of distinct compulsive and impulsive cognitive phenotypes in OCD as potential biomarkers of illness severity and chronicity, and to assess their relevance for identifying individuals at elevated risk for persistent or treatment-resistant OCD; and (4) explore the potential for these cognitive phenotypes to inform the development of early screening tools and preventative

interventions, with the goal of improving early detection and outcomes in individuals with subclinical or emerging OCD symptoms.

ABSTRACT

This thesis investigated executive impairments in the OCD spectrum. Chapter 1 offers an overview of the general background of this work, focusing on OCD symptomatology, neurocognitive deficits in OCD, and the various forms of measuring both OCD and neurocognitive deficits in OCD. This narrative review highlights key domains of executive dysfunction in OCD that require further empirical investigation to bridge existing knowledge gaps.

The first empirical chapter (Chapter 2) presents a systematic review and meta-analysis of three decades of research (112 studies), examining neurocognitive impairments of cognitive inflexibility, response inhibition, and decisional impulsivity (representing latent compulsivity and impulsivity, respectively) in patients diagnosed with OCD. This comprehensive analysis examines a total of 8,313 participants, comparing 4,289 patients with OCD and 4,024 healthy controls on various neurocognitive tasks. To maintain consistency with previously discussed meta-analytical paradigms, this meta-analysis focused on the WCST, IDED, SST, and IGT, given their relevance in capturing latent neurocognitive phenomena. The review found minimal publication bias and generally adequate methodological quality, although some limitations were noted, concerning sample size justification, inconsistent use of diagnostic screening tools, and discussion of limitations. This chapter revealed moderate effect sizes of deficient neurocognitive performance in patients with OCD that were comparable across neurocognitive compulsivity and impulsivity. These effect sizes were orthogonal to various clinical variables such as: depression and anxiety symptoms, effects of medication, comorbid

conditions, and duration of illness. Importantly, findings revealed distinct relationships between compulsivity and impulsivity and symptoms of OCD. The severity of OCD symptoms did not moderate effect sizes of either neurocognitive compulsivity or impulsivity for between-group contrasts, however, within-group correlations revealed a significant relationship between OCD symptoms and compulsivity but not impulsivity. This work revealed implications for these impairments in OCD that requires substantiation, specifically, the manifestation of these latent cognitive phenotypes and how they are assessed.

The second of the empirical chapters (Chapter 3) explores obsessive-compulsive phenomenology during the easing of lockdown restrictions within the general population of the UK. The pandemic offered an opportunity to look at salient pre-clinical obsessive and compulsive tendencies that may have developed during the lockdown (such as avoidance and excessive cleanliness) and the potential persistence of such tendencies once these restrictions had lifted. This work was a replication and extension of Fineberg et al (2021) and so the methodology largely reflected that of the original paradigm. Indeed, the mediation model was replicated; while the original work demonstrated that OCD symptoms predicted lockdown adjustment difficulties only indirectly via affective symptoms, the current study found both direct and indirect effects of OCD symptoms on post-pandemic adjustment difficulty. This suggested that ongoing nature of the pandemic may have strengthened the impact of obsessive-compulsive symptoms on adjustment over time. Findings also revealed contrasting accounts of compulsivity between assessment modalities. Longitudinal analyses revealed strong correlations between self-rated compulsivity (on the Compulsive Personality Assessment Scale (CPAS; Fineberg et al, 2007) and symptom severity across the three assessment points despite symptom severity declining and compulsivity remaining stable. As such, compulsivity may be a trait vulnerability for the development of OCD, though as this was not reflected on neurocognitive tasks (the Wisconsin Card Sorting Test: Milner et al,

1963) and the Intra-Extradimensional Set-Shifting Task: Owen et al, 1991) further work is required to unpick the manifestation of compulsivity at different junctures of OCD symptom severity.

Chronologically speaking, the COVID-19 longitudinal study was conducted first and influenced the methodological choices of the meta-analysis presented in Chapter 2, particularly the decision to expand the assessment of executive functioning in OCD to include measures of impulsivity alongside compulsivity. It was logical to place the meta-analysis first in this thesis, as it provides a comprehensive overview of neurocognitive impairments in clinical patients diagnosed with OCD, forming the foundation of this work. The longitudinal analysis is then presented to build upon these findings by addressing the clinically relevant questions in a subclinical sample that were raised within the meta-analysis, further advancing the investigation into phenotypic manifestations of OCD.

A synthesis of the findings of the preceding two chapters revealed three core themes to this thesis: (1) the exploration of latent cognitive phenotypes of compulsivity and impulsivity, (2) the two leading methods of assessing these phenotypes, self-reports and neurocognitive tasks, and (3) the distinct relationships that compulsivity and impulsivity each have with OCD symptom severity. This apparent orthogonality between symptom severity and executive impairments raises questions about the adequacy of the current diagnostic scope for OCD.

As such, Chapter 4 reviews current screening tools for OCD, highlighting their respective strengths and limitations. Classical and Modern theoretical frameworks are also explored for their unique provisions for scale development. The review highlighted that most symptom scales have only undergone limited psychometric validation and are very restricted in their respective acknowledgement of the more nuanced neurocognitive aspects of the disorder.

The current shortcomings of OCD assessment, as revealed in the review, highlighted the need for a psychometrically sound measure that integrate the strengths of both classical and modern test frameworks to assess the subtle neurocognitive dimensions of OCD for early identification and novel target interventions. Study 4.1 outlines the initial validation of the Obsessive-Compulsive Phenomenological Assessment Scale (OCPAS). As patients with OCD are one singular point on a spectrum, developing a scale that is too close to the diagnosable illness may have eliminated salient pre-clinical features that may elucidate predisposing and precipitant factors at particular junctures of severity. As such, the OCPAS was initially validated in a non-clinical sample so as to retain features relevant to the development of OCD that might be overlooked at the severe end of the spectrum. An initial corpus of 59 items measuring phenotypic variance in OCD reduced to 45 items under classical test theory (exploratory factor analysis); and further reduced to the final 26-item scale under modern test theory (Rasch analysis) - to provide a novel psychometrically robust questionnaire for the self-assessment of compulsive and impulsive features in OCD phenomenology. The OCPAS consist of three factors. Cognitive Control, Behavioural Control, and Anankastic Goals. Cognitive Control measures attentional inflexibility and deficient executive functioning. The Behavioural Control factor describes a lack of control over behavioural and emotional output and subsequent ceasing of the behaviour once started, Finally, Anankastic Goals pertain to rigid beliefs, a need for control, and a motivational drive to achieve perfectionist standards.

Study 4.2 assesses the construct and convergent validity of the OCPAS in a sample with diagnosable OCD. Scores on the OCPAS are contrasted with self-ratings of OCD symptom severity, compulsivity, and impulsivity (using the OCI-R, CPAS, and BIS-11, respectively) as well as with performance on neurocognitive tasks assessing cognitive flexibility and response inhibition (the IDED and the SST, respectively). These self-rating measures and neurocognitive tasks were selected for consistency with preceding chapters and prior research

that has informed this thesis. This chapter shows that the OCPAS effectively distinguishes between those that do and do not have OCD, and correlates strongly with neurocognitive measures of compulsivity and impulsivity. Interestingly, a relationship was revealed between the OCPAS and the Stop Signal Task (Aron, Robbins, and Poldrack, 2004) suggesting that the OCPAS is sensitive to the nuanced neurocognitive features of the disorder. Again, self-rated compulsivity correlated with symptom severity, which was not reflected on the IDED. However, both self-ratings on the Barratt Impulsiveness Scale-11 (Patton, Stanford, and Barratt, 1995) and task performances (SST) of response inhibition correlated with symptom severity and with each other, which may seem contrary to the orthogonality between assessment modalities revealed in Chapters 2 and 3 in addition to the wider literature (e.g., Evenden, 1999; Frydman et al, 2020), challenging the previously conceived independence of impulsivity from symptom severity. These findings highlight the OCPAS as a robust scale for measuring neurocognitive dimensions of OCD, and offers insights into the relationship between compulsivity, impulsivity, and OCD symptomatology.

Chapter 5 consolidates the findings from the previous empirical chapters by synthesising phenotypic variance across distinct levels of symptom severity, ranging from non- and sub-clinical presentations (in Study 4.1 and Chapter 2) to OCD-likely and formally diagnosed groups (in Study 4.2 and Chapter 1). This synthesis showed that compulsivity shares a similar trajectory to symptom severity across the chapters, given their moderate to strong correlations in Chapters 2, 3, and 4. In contrasting the inconsistencies in the relationship between impulsivity and symptom severity in Chapters 2 and 4, it appears that impulsivity at first correlates and contributes the precipitation of OCD and then remains stable for the duration of the illness given the orthogonality recorded in Chapter 2. An exploratory mediation model is presented, offering a potential mechanism by which these phenotypes influence symptom severity in OCD-likely groups. The model suggests that both compulsivity and impulsivity

contribute to the transition from symptoms to syndrome to illness which may be the difference between managing and not-managing worsening symptomatology.

In conclusion, this thesis reveals the respective manifestations of compulsivity and impulsivity in OCD phenomenology. Both phenotypes display unique associations with symptom severity and contribute to the precipitation of OCD proving to be potential trait markers. However, the current diagnostic assessment of OCD fails to capture such phenotypic variance and so, the OCPAS was developed to provide a psychometrically robust self-report measure of deficient cognitive and behavioural control. It is suggested that future research focuses effort on naturalistic and longitudinal analyses of these phenotypic manifestations to substantiate benchmarks for early identification of at-risk cohorts.

CHAPTER 2: COGNITIVE PHENOTYPES OF COMPULSIVITY AND IMPULSIVITY AND CLINICAL VARIABLES IN OBSESSIVE-COMPULSIVE DISORDER; A SYSTEMATIC REVIEW AND META-ANALYSIS

As explored in Chapter 1, patients with OCD exhibit diminished executive control, specifically cognitive inflexibility and inhibitory control. Indeed, patients with OCD are unable to successfully shift attention between activities, situations, and stimuli (Ionescu, 2012). It is this deficit in cognitive flexibility that results in this compulsive disposition, and much like compulsivity, impulsivity is underpinned by deficient response inhibition – an impaired executive ability to suppress or delay pre-potent action or thought (Penades et al, 2007). These components of phenomenology contribute to key symptomatic manifestations in OCD (Kashyap et al, 2013). Neurocognitive tasks provide a sensitive detection of such executive impairments. Under paradigms of perseveration and reversal learning, compulsivity is typically assessed by the WCST (Milner, 1963) and the IDED (Owen et al, 1991). As described in Chapter 1, deficient inhibitory control can be fractionated into two core components, motor impulsivity and decision-making impulsivity; typically measured by the Stop-signal Task (Aron, Robbins, and Poldrack, 2004) and the Iowa Gambling task (Bechara et al, 1994), respectively. Although both compulsivity and impulsivity contribute to dysfunctional goal-directed behaviour (Gillan and Robbins, 2014), the extent of the impulsive manifestation relative to the compulsive are yet to be captured.

Motor impulsivity in patients with OCD has received inconsistent evidence. Some papers clearly show impaired performance on the SST in patients with OCD (Lee, Yost, and Telch, 2009; Berlin and Lee, 2018) which is suggestive of an inability to inhibit responding when actions supersede thought. By contrast, there are those that report null findings (McLaughlin et al, 2020) whilst others report partial relationships, with compulsions but not with obsessions (Berlin and Lee, 2018); and so, the extent of this phenotype in symptomatic

manifestations warrants attention. Similarly, impaired decision-making in OCD is another point of contention within the literature as some research report such an impairment in OCD (Cavedini et al, 2002; Nistico et al, 2021) whilst others refute this notion owing to OCD traditionally considered a disorder of pronounced forethought and cautious consideration for consequences (Summerfeldt et al, 2004; Abramovitch and McKay, 2016). The permeation of impulsivity within OCD phenomenology certainly warrants further investigation given this incoherent consensus as this phenotype may hold crucial implications for the conceptualisation of OCD.

Current assessments of OCD provide little or no appraisal of the impulsive features of OCD and so, are omitted from what otherwise would be a comprehensive assessment of a patient's presenting issues. Consideration of these features in parallel to presenting symptomatology is required as current novel treatment models fail to address these neurocognitive phenotypes, which otherwise remain unchallenged. The relationships between compulsivity and impulsivity and clinical variables such as symptom severity, duration of illness, and comorbid conditions also require substantiation to isolate these phenotypes for their transdiagnostic viability as assessment markers.

Comorbid psychiatric disorders such as depression interfere with performance across a wide range of cognitive tasks through non-specific behavioural effects (Murphy, Michaels, and Sahakian, 2011; Mondal and Kumar, 2020). Comorbidity in OCD is prevalent, and up to 60% of patients with OCD show some signs of depressive symptomatology (Robins et al, 1984; Pigott et al, 1994; Miguel, Rauch, and Jenike, 1997; Millet et al, 2004; Pinto et al, 2006; Moore and Howell, 2017), which is commonly believed to be a secondary phenomenon (Huppert et al, 2009; Anholt et al, 2011; Moreira-de-Oliveira et al, 2022). Nevertheless, the potential impact of depression and other common comorbidities such as Generalised Anxiety

Disorder (Armstrong, Zald, and Olatunji, 2011) on neurocognitive performance in OCD is not well understood and requires rigorous investigation.

The points explored in Chapter 1 motivated the current collection and synthesis of the empirical evidence pertaining to neurocognitive deficits underlying manifestations of compulsivity and impulsivity in patients with OCD and to assess the relationship between neurocognitive deficits in OCD and a range of clinically salient variables which may have bearing on such manifestations. Systematic review and meta-analysis are applied to investigate the following research questions:

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

METHOD

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 (Page et al, 2021). Systematic reviews provide a consolidation of empirical evidence of a specific research subject and, by adhering to rigorous guidelines such as PRISMA (see Appendix A), can serve a crucial role in clinical decision-making (Muka et al, 2019; Shaheen et al, 2023).

The online databases PsychINFO, Cochrane Library, Pubmed, and Scopus were searched for scholarly peer-reviewed articles 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*.

Search strategy and study selection

The papers were downloaded from their respective databases and uploaded to the Rayyan platform for systematic reviews (Ouzzani et al, 2016). Once duplicate studies were removed from the resultant collection, results were screened based on title and abstracts. The full texts for all remaining papers were then scrutinised according to the eligibility criteria (listed below). Reference lists of papers were screened for any relevant candidates for inclusion. In the instance of a discrepancy concerning the potential inclusion of a study, this was discussed amongst the research team in which the inclusion or exclusion was determined. An illustration of the screening process can be found in the PRISMA flowchart (Figure 2.1).

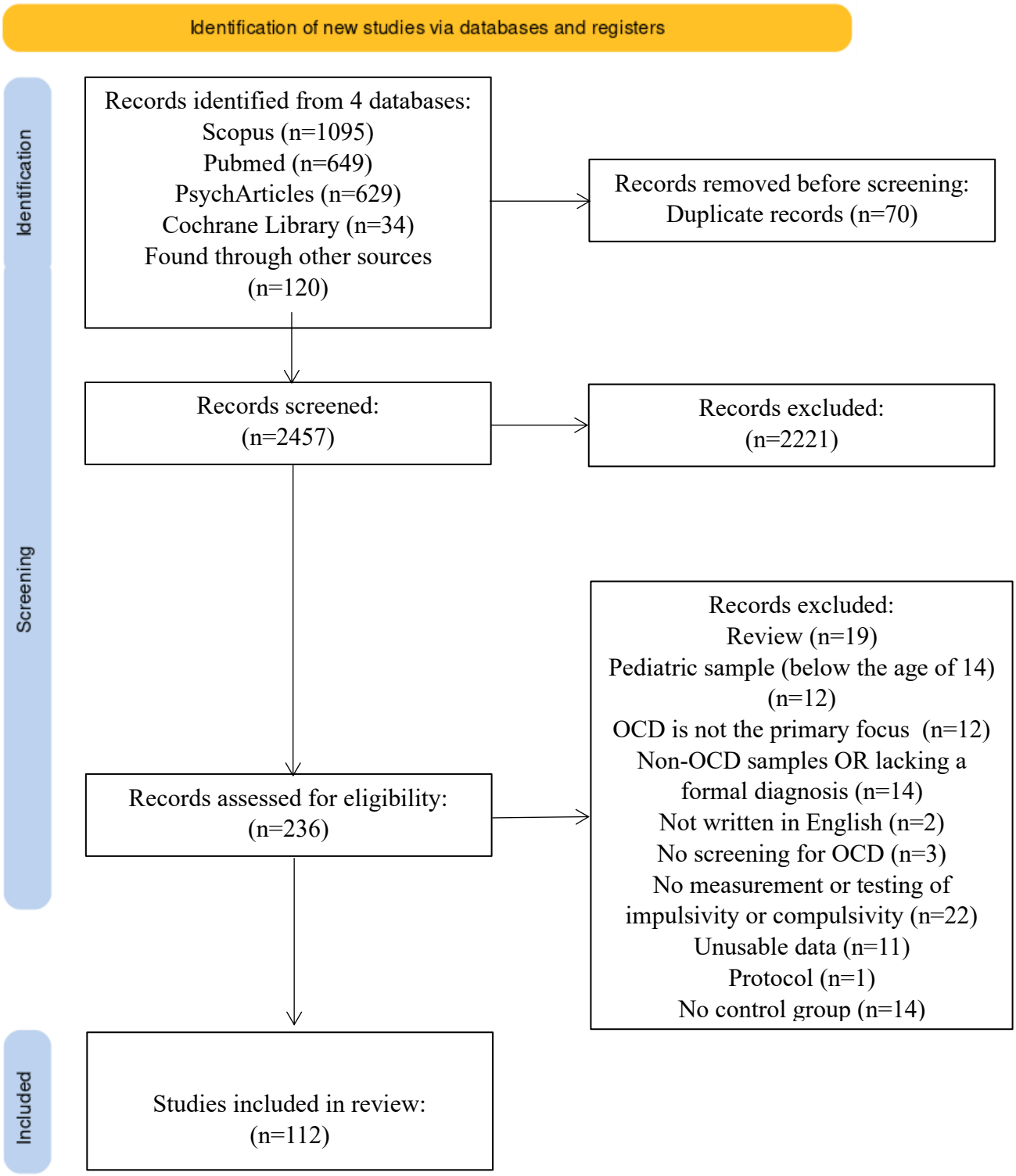
Inclusion and exclusion criteria

Studies were included if they tested participants with a primary diagnosis of OCD using a structured diagnostic method such as the DSM-5 or the ICD-10 (and earlier versions), and if there was a 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). Behavioural responses (performance metrics) to standardised objective cognitive tests were chosen to measure these latent cognitive phenotypes and not clinical scales measuring compulsive (e.g., CHIT; Chamberlain and Grant, 2018) or impulsive responding (e.g., UPPS; Lynam et al, 2006), because objective tasks in this area of cognitive-behavioural science may be less subject to bias than clinical scales, which are more subject to deficiencies in metacognitive appraisal, such as those described in Chapter 1 (Friedman and Gustavson, 2022). Moreover, studies were included if they employed a sample of adults or adolescents above 14 years of age. As previous research has shown that executive functioning matures around 14-15 years of age (Best and Miller, 2010; Tervo-Clemmens et al, 2023), those samples below 14 years of age were excluded due to the differing neurocognitive profiles. Studies were to be published in English.

Studies were excluded if they possessed any of the following features: pediatric sample (below the age of 14), OCD was not the primary focus, Non-OCD samples OR lacking a formal diagnosis (subclinical samples), not written in English, no screening for OCD, no measurement or testing of impulsivity or compulsivity, unusable data, protocol or review, no control group, or only included psychometric data.

Figure 2.1. PRISMA flowchart of 112 studies meeting inclusion criteria.



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 compulsive or impulsive neurocognitive tasks, or in some cases both should a study employ tasks assessing both latent phenotypes together.

In accordance with Fineberg et al's (2010) subdivision of robust neurocognitive tasks for compulsivity and impulsivity, performance on the Wisconsin Card Sorting Test and the Intra Extradimensional Set-Shifting Task were prioritised as measures of compulsivity. These tasks assess attentional set-shifting and are the most abundant and are extensively validated within a variety of cohorts for the assessment of cognitive flexibility, a key factor inherent within this latent cognitive phenotype (Henry, 2006; Chamberlain et al, 2021). In addition, performance on the Stop-Signal Task and the Cambridge Gambling Task were prioritised as measures of inhibitory control; motor impulsivity and decision-making impulsivity, respectively.

The primary outcome data comprised task performance on either compulsive or impulsive 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, the perseverative errors mean score and the extra-dimensional errors mean score on the WCST and the IED respectively were selected as the primary metrics as they closely represent attentional set-shifting.

For response inhibition, measures of decision-making impulsivity and motor impulsivity were included. For decision-making impulsivity, the quality of decision-making metric was extracted from studies using the CGT; and in studies using the Iowa Gambling Task (Bechara et al, 1994), the net score was used (calculated by subtracting disadvantageous selections

from advantageous selections in 100 trials). For motor impulsivity the Stop Signal Reaction Time from the SST was selected. This metric is a calculated standardised reaction time that eliminates potential confounds of background variables; Band, van der Molen, and Logan, 2003; Verbruggen et al, 2019).

A broader range of impulsive neurocognitive tasks was employed because of the greater uncertainty about the role of impulsivity in obsessive-compulsive phenomena as well as the aforementioned inconsistency in established effects (Abramovitch and McKay, 2016; Croft et al, 2022). The additional measures included were the IGT (Bechara et al, 1994), the Temporal Discounting task (Rubia et al, 2009), the Go/No-Go task (Gomez, Ratcliffe, and Perea, 2007), and Conner's Continuous Performance Task-II (Conner and Staff, 2000). Of the tasks measuring response inhibition, the SST, the Go-No/Go task, and the CCPT-11 were selected to assess motor impulsivity, and the Cambridge Gambling Task, the Iowa Gambling Task, and the Temporal Discounting task to assess decision-making impulsivity.

When uniformity of the test metric could not be achieved across studies (e.g., if 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 were 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) (Goodman et al, 1989), the Hamilton Anxiety Rating Scale (HARS: Hamilton, 1959), and the Hamilton Depression Rating Scale (HDRS: Hamilton, 1960). Moderator variables were also extracted and included: age of sample, proportions of females per sample, years in education, intelligence (IQ), psychiatric or medical/physical comorbidities, and duration of illness.

Study Quality

The quality of the included studies was assessed using the Appraisal tool for Cross-Sectional Studies, AXIS checklist (Downes et al, 2016) 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.

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 participants with OCD and the healthy control groups were used to calculate Hedge's *g*. Some studies did not conform to this way of reporting task performance (Kang et al, 2013; Martoni et al 2015; Vaghi et al, 2017; and Bohon, Weinbach, and Lock, 2020). As Kang et al (2013), Vaghi et al (2017), and Bohon, Weinbach, and Lock (2020) report between-groups differences, the effect sizes were estimated using group sizes and independent groups t-test values. Martoni et al's (2015) 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 (Fenger et al, 2005; Simpson et al, 2006) or impulsivity (Chamberlain et al 2007b; Frydman et al, 2020). 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 (age of onset <17) or a late onset OCD group (age of onset ≥ 17) (Kim et al, 2020) or compared to a familial (those with an affected first degree relative) or sporadic OCD group (those without an affected first degree relative: e.g. Bhattacharya et al, 2021), the control sample size was divided by the number of comparisons being made to avoid inflating the weighting of effect sizes.

The following coding methods were used to classify grouping for moderator categorical variables for subgroups analyses. *Clinical status:* participants with OCD recruited from outpatient clinics = 1, recruited from inpatient clinics =2; *Effects of medication:* participants with OCD receiving medication =1, participants with OCD that have not yet received any medication =2, and participants with OCD withdrawn from medication=3. Studies including participants with OCD with comorbidities showed minimal uniformity in the reporting of comorbid disorders and so were also coded as follows; studies excluding OCD with comorbid disorders and only including OCD as the primary and only presenting diagnosis were assigned a value of 1, OCD with comorbidities =2. All declared comorbid disorders can be found in appendix B.

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 comorbid symptoms 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, recommendations of at least six to ten studies for a continuous variable and at least four studies per group for a categorical subgrouping variable were followed (Fu et al, 2010; Higgins et al, 2019).

The I^2 statistic was used to assess heterogeneity and for interpretation. Following Cochrane guidance (Higgins et al, 2019), I^2 values between 0-40% 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, the assessment of small study effects and publication bias. If asymmetry appeared to be present, it was further examined using Duval and Tweedie's Trim and Fill method.

RESULTS

Narrative Review Of The Literature

Our searches identified a total of 2,527 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 (exclusions are detailed in Figure 2.1). 112 studies met the inclusion criteria published between 1989 and 2022. A total of 8,313 participants were tested (4,289 participants with OCD and 4,024 healthy controls) with a combined mean age of 31.41 (SD=5.90). The main characteristics of the 112 included studies are presented in Table 2.1.

In the final set of 112 studies derived from the described search strategies, the most commonly used measure to ascertain OCD symptom severity was the Y-BOCS (Goodman et al, 1989), appearing in 99 papers. Six papers use the Obsessive-Compulsive Inventory-revised (OCI-R; Foa et al, 2002) in conjunction with the Y-BOCS (Lawrence et al, 2006; Menzies et al, 2007; Page et al, 2009; Yang et al, 2015; Vaghi et al, 2017; and Frydman et al, 2020) to provide analyses on symptom severity and symptom dimensions. Similarly, Martinez-Esparza et al (2021) employs the Y-BOCS adjunct to the Obsessional Beliefs

Spanish Inventory-Revised (Belloch et al, 2010). A collection of papers used alternative tools to assess symptom severity. Blom et al (2011) and Tolin et al (2014) used the OCI-R, Luo et al (2020) used the Padua Inventory-Washing State University Revision (PI-WSUR; Burns, 1995), and both Head et al (1989) and Hymas et al (1991) use the Maudsley Obsessive-Compulsive Inventory (MOCI; Hodgsen and Rachman, 1977). 11 papers exclusively use these described scales as screening tools and did not use them in any of their respective analyses.

This narrative review shows that only 42 of 112 studies (37.50%) provide a correlational analysis between OCD symptom severity and task performance impairments (see Table 2.1).

Table 2.1. Characteristics of Included studies and correlations between Y-BOCS scores and task performance metrics.

Study	Diagnosis	Mean age	Y-BOCS score	Percentage female	OCD (n)	Control (n)	Neurocog Test	Neurocog Measure	Correlations	AXIS study quality rating
Head 1989	DSM-III	33.30	No measure	46.67	15	15	WCST	Perseveration	-	13
Boone 1991	DSM-III	F-OCD 40.60 S-OCD 32.70	Not given	Not stated	7 13	16	WCST	Perseverative responses	-	14
Hymas 1991	DSM-III	36.90	No measure	37.50	16	16	WCST	Perseverative responses	-	14
Gambini 1993	DSM-III-R	30.30	24.30	43.48	23	27	WCST	Perseverative errors	-	13
Abbruzzesse 1995a	DSM-III-R	30.90	27.21	42.42	33	33	WCST	Perseverative errors	-	14
Abbruzzesse 1995b	DSM-III-R	29.50	18.40	44.00	25	25	WCST	Perseverative errors	-	15
Gross-Isseroff 1996	DSM-III-R	34.80	26.50	100.00	15	15	WCST	% of perseverative errors	-	14
Veale 1996	DSM-III	36.10	Not stated	57.50	40	22	IED	ED errors	-	16
Abbruzzesse 1997	DSM-III-R	30.00	No measure	31.67	60	30	WCST	Perseverative errors	-	14
Lucey 1997	DSM-III-R	38.00	23.00	47.37	19	19	WCST	Perseverative errors	***r*=.46, *p*<.05**	16
Purcell 1998	DSM-IV	40.60	24.10	66.70	30	30	IED	EDS trial score	-	16
Okasha 2000	ICD-10	24.06	27.10	30.00	19	19	WCST	Perseverative errors	*Non-sig (No data)*	14
Basso 2001	DSM-IV	30.06	No measure	40.00	20	31	WCST	% of perseverative errors	***r*=0.32, *p*<.01**	14
Moritz 2001	DSM-IV	33.22	24.40	55.56	36	36	WCST	Perseveration	*p*>.20	14
Sanz 2001	SCID-DSM-III	25.80	Not stated	54.20	18	24	WCST	Perseverative errors	***r*=0.54, *p*<.01**	14

Cavedini 2002	DSM-IV	33.70	Not given	47.00	34	34	IGT	Ad vs disad deck	-	13
Kim 2002	DSM-IV	29.82	25.43	30.77	39	31	WCST	Perseverative errors	-	15
Moritz 2002	DSM-IV	31.6	24.10	56.00	25	70	WCST	Perseveration	r=0.03, p>.30	14
Cavallaro 2003	DSM-IV	30.50	No measure	50.80	67	56	IGT WCST	No. of disad deck Perseverative errors	*Not assessed*	14
Harris & Dinn 2003	DSM-IV	39.40	Not stated	52.00	25	11	Go/no-go	Block performance	-	15
Herrmann 2003	DSM-IV	41.20	21.20	33.30	12	12	Go/no-go	Commission errors	-	14
Kim 2003	SCID-IV	26.74	24.16	26.32	19	21	WCST	Perseverative errors	*Non-sig (No data)*	16
Kwon 2003	SCID-IV	28.50	25.50	28.57	14	14	WCST	Perseverative errors	-	15
Lacerda 2003	DSM-IV	33.07	23.71	28.57	14	14	WCST	Perseverative errors	-	16
Nielen 2003	DSM-IV	37.20	24.10	57.89	19	24	IED	EDS trial score	-	18
Choi 2004	SCID-IV	26.50	24.24	17.64	34	34	WCST	Perseverative errors	-	16
Roth 2004	DSM-IV	36.30	26.80	55.60	27	27	WCST	% of Perseverative errors	-	15
Bohne 2005	SCID-IV	31.90	Not given	57.10	20	26	WCST	Perseverative errors	-	16
Boldrini 2005	DSM-IV	32.70	22.70	40.00	25	15	WCST	Perseverative errors	-	15
Fenger 2005	DSM-IV & ICD-10	39.00	27.20	53.33	15	17	IED WCST	EDS trial score Perseveration	-	15
Nakao 2005	SCID-DSM-III	32.40	29.00	60.00	10	13	WCST	Total errors	-	16

Roh 2005	DSM-IV	26.26	26.94	19.00	21	20	WCST	Perseverative errors	-	15
Ruchsow 2005	DSM-IV & Y-BOCS	Age matched sample	19.50	45.45	11	11	Go/no-go	Mean number of errors	-	14
Bucci 2006	DSM-IV	25.73	25.52	53.34	30	30	WCST	% of Perseverative errors	*Non-sig (No data)*	16
Chamberlain 2006	DSM-IV	35.30	20.40	Not stated	20	20	CGT	% of rational decisions	$p>.05$	16
Lawrence 2006	SCID-DSM-IV	36.10	25.13	48.72	38	39	WCST IGT	Perseverative errors Total score	β =-0.24, $p>.05$ β =0.11, $p>.05$	16
Simpson 2006	DSM-IV	Cur 41.47 Com 40.47	19.50 26.00	50.00 46.67	30 15	35	IED WCST	EDS trial score Perseverative errors	all $r<.18$ and all p values $p>.20$	18
Chamberlain 2007a	MINI	35.30	20.40	50.00	20	20	CGT	Percent rational decisions	-	16
Chamberlain 2007b	MINI-DSM-IV	32.10	22.70	80.00	20	20	CGT SST IED	Percent rational decisions SSRT Trials to criterion Extra-dimensional	$p>.05$ $p>.05$ $p>.05$	16
De Geus 2007	MINI	36.1	27.30	74.36	39	26	WCST	% of Perseverative errors	**r=.57 ,p<.001**	14
Kim 2007	DSM-IV	25.73	21.45	46.67	15	15	Go/no-go	Commission errors	**r=-0.64, p<.05**	15
Kitis 2007	DSM-IV	32.87	22.70	73.90	23	22	WCST	Perseverative errors	**r=0.51, p<.01**	15
Menzies 2007	MINI-DSM-IV	32.50	21.90	70.97	31	31	SST	SSRT (log transformed)	-	15
Roth 2007	SCID-DSM-IV	37.80	22.30	58.30	12	14	Go/no-go	Commission errors	-	16

Bohne 2008	SCID	33.00	17.90	52.40	21	26	Go/no-go	False positives	-	16
Cha 2008	SCID	Clean 30.90 Check 34.00	24.70 25.40	30.43 41.67	23 24	20	WCST	Perseverative errors	-	15
Rao 2008	MINI-DSM-IV	27.77	22.84	26.67	30	30	WCST	% of Perseverative errors	*Non-sig (No data)*	17
Trivedi 2008	ICD-10	34.98	23.87	82.00	30	30	WCST	Perseverative errors	r=-0.19, p=0.31	13
Nedeljkovic 2009	ADIS-DSM-IV	37.80	23.55	61.02	59	59	IED	ED trial level	-	16
Page 2009	SCID-DSM-IV	39.1	23.50	0.00	10	11	Go/no-go	Probability of inhibition	-	16
Starcke 2009	DSM-IV	36.36	No measure	50.00	14	15	IGT	Total net score	-	15
Cavedini 2010	DSM-IV	35.60	28.30	42.90	35	31	IGT WCST	Number of advantageous deck selections % of Perseverative errors	- -	15
Starcke 2010	SCID-DSM-IV	35.25	No measure	43.48	23	22	mWCST IGT	Perseverations Net score	-	14
Blom 2011	DSM-IV	43.00	No measure	Not stated	17	19	SST	SSRT	-	15
Borges 2011	DSM-IV	33.00	27.20	58.20	67	17	WCST IGT	Perseverative errors Net score	**r=0.50, p<.001**	15
da Rocha 2011	MINI-DSM-IV	28.40	26.61	45.80	107	107	IGT CCPT-II	Net score Commission errors	**r=0.43, p<.05**	15
Krishna 2011	DSM-IV	26.00	23.00	22.58	31	31	WCST IGT	% perseverative errors decks A+B-(C+D)	r=0.08, p>.05 r=0.18, p>.05	15

Rajender 2011	DSM-IV-TR	25.60	23.62	50.00	30	30	WCST	Perseverative errors	*p*>.20	15
Boisseau 2012	DSM-IV	22.32	22.53	100.00	19	21	SST	SSRT	-	15
Cavedini 2012	DSM-IV-TR	36.05	28.30	33.00	20	18	IGT	Net score	-	15
de Wit 2012	DSM-IV-TR	38.60	21.90	49.00	41	37	SST	SSRT	*Non-sig (No data)*	16
Hur 2012	SCID	26.90	23.40	25.81	31	52	WCST	Perseverative errors	-	15
Bersani 2013	MINI	36.44	22.20	76.00	25	21	SST IED	SSRT EDS errors	*Non-sig (No data)* *Non-sig (No data)*	15
Boisseau 2013	MINI-DSM-IV	22.32	22.53	100.00	19	21	IGT	Mean taken from block scores	-	16
Dittrich 2013	DSM-IV	43.20	16.20	69.23	10	10	CGT	Percent rational decisions	-	17
Endrass 2013	SCID-DSM-IV	33.40	18.00	64.00	25	25	Task similar to the WCST	Reversal errors	*p*>.10	15
Kang 2013	SCID-DSM-IV	24.90	22.10	33.33	18	18	SST	SSRT	-	16
Kashyap 2013	MINI-DSM-IV	27.56	27.46	37.30	150	105 75	WCST IGT	% of perseverative errors decks A+B-(C+D)	-	18
Guo 2014	ICD-10	26.4	29.32	38.00	139	139	WCST	Perseverative errors	-	14
Sharma 2014	DSM-IV	65% aged 18-26	Not given	40.00	20	20	WCST	% of Perseverative errors	-	15

Sohn 2014	SCID-IV	27.80	21.70	26.20	80	76	SST	SSRT	*p*>.05	16
Tolin 2014	DSM-IV	33.54	No measure	25.00	24	24	Go/no-go	Commission errors	-	16
Wen 2014	DSM-IV	27.13	24.47	38.46	26	20	WCST	Perseverative errors	-	15
Grassi 2015	SCID-DSM-IV	36.29	21.79	39.47	38	39	IGT	IGT final net score	*Non-sig (No data)*	15
Kim 2015	SCID-DSM-IV	26.62	22.07	21.54	65	58	IGT	Total net score	***r*=-0.26, *p*<.05**	16
Kohli 2015	ICD-10	32.75	Not given	40.00	20	20	WCST	Perseverative errors	-	16
							WCST	Perseverative errors	-	
Lei 2015	SCID	21.67	30.33	45.24	42	42	SST	SSRT	*Non-sig (No data)*	17
Martoni 2015	DSM-IV	34.43	26.98	52.04	269	120	IGT	Mean IGT score	-	15
Singh 2015	MINI	W/O D 30.43 W Dep 34.05	21.33 27.85	46.67 40.00	30 20	25	WCST	Perseverative errors	***r*=0.72, *p*<.01** Not stated	15
Toobaei 2015	DSM-IV-TR	23.91	Not given	55.56	27	23	WCST	Perseverative errors	-	15
Yang 2015	DSM-IV	26.62	23.71	29.31	58	58	WCST	Perseverative errors	-	16
Zhang 2015a	SCID-CV	23.00	24.20	42.50	40	40	WCST	Perseverative errors	*Non-sig (No data)*	15
Zhang 2015b	SCID-DSM-IV	NM 28.07 M 27.92	28.14 26.25	52.63 54.55	57 77	115	WCST IGT	Perseverative errors Net score	-	16
Zhang 2015c	DSM-IV-TR	26.51	22.67	60.00	55	55	WCST IGT	Perseverative errors Net scores	*p*>.11 *p*>.11	15
Fan 2016	SCID	Auto O 20.36 Reac O 22.37	30.76 30.57	42.90 38.90	40 47	58	SST	SSRT	*p*>.10	16

Lei 2017	SCID	EO 20.68 LO 24.64	27.83 25.06	36.51 30.30	63 33	51	SST	SSRT	*Non-sig (No data)*	18
Posner 2017	DSM-IV	29.10	25.60	46.66	30	32	CCPT II	Commission errors	-	16
Norman 2017	SMD	15.75	22.32	0.00	20	20	TDT	TD – delayed	-	16
Saremi 2017	DSM-IV	32.45	25.03	74.00	35	35	WCST Go/no-go	Perseverative errors Commission errors	r=-0.26, p>.05 r=-0.12, p<.05	16
Vaghi 2017	MINI	36.14	22.20	52.27	44	43	IED	Errors at ED stage	-	14
Yun 2017	DSM-IV	24.9	19.30	29.17	24	34	WCST	Perseverative errors	-	16
Zhang 2017	MINI-DSM-IV	30.71	25.84	29.41	51	31	IGT	Mean score from block data	*Non-sig (No data)*	15
Grassi 2018	SCID-I/P	44.75	Median given.	45.00	40	40	IGT	Final net score	*Non-sig (No data)*	15
Kertzman 2018	DSM-IV	32.46	No measure	Not stated	61	131	Go/no-go	False alarms	-	14
Martoni 2018	DSM-5	30.68	26.21	27.78	36	36	SST	SSRT (last half)	-	16
Norman 2018	ICD-10	15.76	22.32	0.00	20	20	IGT IED	Net score EDS errors	p>.05 p>.05	16
Cillo 2019	MINI-Plus	33.50	Not given	39.51	81	124	CGT	Quality of decision making	-	14
Grassi 2019	DSM-IV	33*	Median given	31.80	44	40	IGT	Net score	-	14
Tomiyama 2019	SCID	33.49	24.57	56.76	37	40	WCST	% of Perseverative errors	p>.09	18
Yu 2019	ICD-10	24.70	17.97	44.00	25	27	SST	SSRT	*Non-sig (No data)*	18
Bohon 2020	SCID	15.64	18.70	100.00	11	24	WCST	Perseverative errors	-	15

Frydman 2020	SCID	35.88	24.05	17.65	17	17	CGT SST	Quality of decision making RT on go trials	- -	16
Kim 2020	DSM-IV	EO 23.27 LO 25.00	27.47 26.04	28.57 34.62	EO 49 LO 52	103	IED CGT	IED errors Quality of decision making	-	16
Luo 2020	DSM-5	19.71	20.50	33.30	24	26	IGT	Net scores	***r*=-0.31, *p*<.01**	16
Thorsen 2020	SCID-DSM-IV	30.19	26.83	61.00	24	19	SST	SSRT	-	16
Bhattacharya 2021	MINI	F-OCD 37.15 S-OCD 32.75	13.91 17.23	50.00 47.27	f-OCD 54 s-OCD 55	60	SST	Mean stop RT	-	15
Chen 2021	DSM-5	31.20	24.90	68.00	29	28	IED SST	ED errors SSRT	*r*=0.26, *p*=.177 *r*=-0.22, *p*=.247	16
Martinez-Esparza 2021	SCID-I, SCID-II	32.81	Not given	53.00	32	30	WCST Go/no-go	Perseverative errors Commission errors	-	15
Moritz 2021	ICD-10, DSM-5	34.77	Not given	69.00	13	30	WCST	Perseveration	-	15
Tomiyama 2021	SCID-I/P	33.34	24.17	60.98	41	49	SST	SSRT	-	16
Hasuzawa 2022	SCID-DSM-IV	32.80	24.50	60.00	50	55	IGT	Total net score	*Non-sig (No data)*	16
Sternheim 2022	SCID-I	33.92	23.91	Not stated	72	67	IED	No. of trials to reach stage 9 (ED switch-cost)	-	16

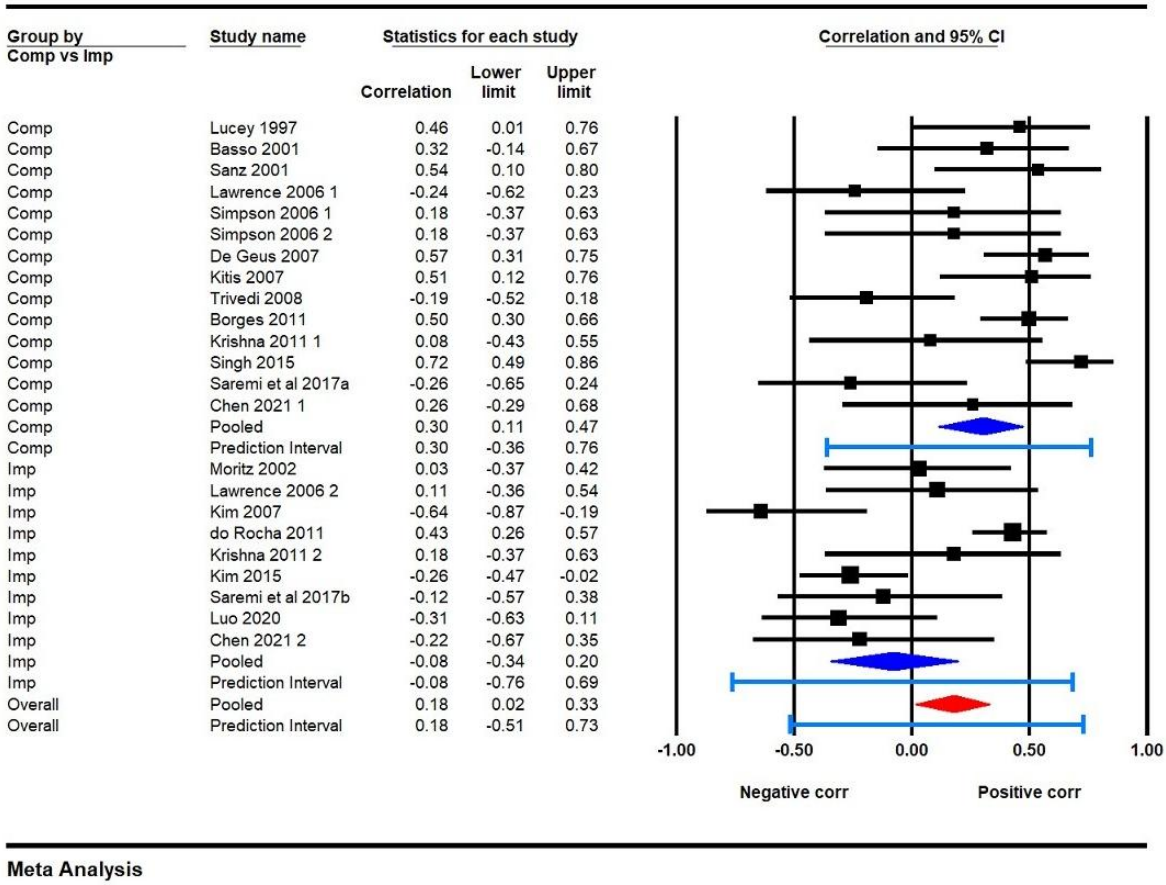
Note: Y-BOCS=Yale-Brown Obsessive-Compulsive Scale, AO=Autogenous obsessional group, RO=Reactive obsessions group, WCST=Wisconsin Card Sorting Test, PE=Perseverative errors, %PE= % of Perseverative errors, FTMS=Failure to maintain set, CGT=Cambridge Gambling Task, RD=rational decisions, %RD = % of rational decisions, G/N-G=Go/No-Go task, com errors=commission errors, SST=Stop Signal Task, SSRT=Stop Signal Reaction Time, CPT-II=Continuous Performance Task-II, om errors=omission errors, IGT=Iowa Gambling Task, total net def=total net difference.

Of this collection of 42 studies, 11 studies revealed significant associations between Y-BOCS scores and neurocognitive task performances whilst 31 studies reported null findings. 28 papers assessed compulsivity (WCST k=23, IDED k=5) of which only 7 studies report correlations between Y-BOCS scores and WCST outcome metrics; the IDED did not significantly correlate with the Y-BOCS. Interestingly, in De Geus et al (2007), the failure to maintain set metric correlated with Y-BOCS scores rather than perseverative errors, as is reflected in Lucey et al (1997), Kitis et al (2007), and Borges et al (2011).

Conversely, 23 papers used a neurocognitive task that assess dimensions of impulsivity; the CGT (k=2), the IGT (k=10), the SST (k=9), the Go/no-go task (k=2), and the Continuous Performance Task-II (k=1). Chamberlain et al (2007b) used for the CGT and SST. Of these papers, only 4 significant correlations were revealed. Both Kim et al (2015) and Luo et al (2020) report a negative correlation between Y-BOCS scores and the IGT, Kim et al (2007) shows that the Go/no-go task also negatively correlates with Y-BOCS scores, and finally, do Rocha et al (2011) reports a positive correlation between impaired performance on the Continuous Performance Task-II and Y-BOCS scores.

Meta-analyses were performed on correlational data extracted from included studies (see Figure 1.2). Findings revealed a significant pooled effect size correlation between Y-BOCS scores and neurocognitive measures of compulsivity ($r=0.30$,[95%CI 0.11, 0.47], $k=14$, $p<.05$). However, no significant effect size correlation was revealed between Y-BOCS scores and neurocognitive measures of impulsivity ($r=-0.08$,[95%CI -0.34, 0.20], $k=9$, $p>.05$).

Figure 2.2. Correlation effect sizes of Y-BOCS scores and compulsivity and impulsivity neurocognitive performance.



Only a small proportion of papers (8 of 112) included a self-assessment of neurocognitive deficits adjacent to the neurocognitive task; six papers included a self-rating of impulsivity and two papers included a self-rating of compulsivity (see Table 2.2). The BIS-11 (Patton, Stanford, and Barratt, 1995) and its three subfactors (attentional, motor, and non-planning impulsivity) were consistently used as measures of impulsivity. However, the relationship between BIS-11 and OCD symptom severity scores was inconsistent as only two papers report an association. Both Boisseau et al (2012) and Frydman et al (2020) show that greater OCD symptom severity is positively correlated with greater impulsivity.

Self-assessments for compulsivity were more seldom in the included papers. Sternheim et al (2022) used the Detail and flexibility questionnaire (Roberts et al, 2011) and found that the attention subscale (but not the rigidity subscale as might be expected) positively correlated with Y-BOCS scores. Boisseau et al (2013) used the Frost multidimensional perfectionism scale (Frost et al, 1990) to self-assess compulsivity, however the relations with OCD symptom severity were not explored.

Meta-analyses could not be performed on this data between self-rated scores of compulsivity and impulsivity and OCD symptom severity scores as there were too few studies that report on and provide correlational data.

Table 2.2. Correlations between OCD symptom severity and self-report measures of compulsivity and impulsivity

	Sample size (n)	**Y-BOCS scores**	**Self-rating scale**	**Correlations between measures**
Chamberlain et al (2006)	20	20.40	BIS-11 – total score	-
Boisseau et al (2012)	**19**	**22.53**	**BIS-11 – attentional imp**	***r*=0.44, *p*<0.05**
Boisseau et al (2013)	19	22.53	FMPS – total score	-
Sohn et al (2014)	80	21.70	BIS-11 – total score	-
Grassi et al (2015)	38	21.79	BIS-11 – total score	*Non-sig (No data)*
Grassi et al (2019)	44	24 median	BIS-11 – total score	-
Frydman et al (2020)	**17**	**24.23***	**BIS-11 – total score**	***β*= 0.54, *p*= .01**
Sternheim et al (2022)	**72**	**23.91**	**DFlex – attentional score**	***r*=0.43, *p*<.01,**

*Note: *=OCI-R score, BIS-11 = Barratt Impulsiveness Scale-11, attentional imp = attentional impulsivity, FMPS = Frost Multidimensional Perfectionism Scale, DFlex = Detail and Flexibility Questionnaire.*

The same 8 papers including a self-report measure and neurocognitive task that assess compulsivity and/or impulsivity were scrutinised for correlational analyses. As shown in Table 2.3, only 5 of these papers report a correlational analysis and all record null-findings between the BIS-11 and the SST (Boisseau et al, 2012; Sohn et al, 2014; Frydman et al, 2020), the BIS-11 and the IGT (Grassi et al, 2015), and the FMPS and the IGT (Boisseau et

al, 2013). Chamberlain et al (2006), Grassi et al (2019), and Sternheim et al (2022) do not provide any correlational assessment.

As before, there were too few studies to substantiate meta-analyses on correlations between self-rated scores and neurocognitive task measures of impulsivity and compulsivity (Fu et al, 2010; Higgins et al, 2019).

Table 2.3. Correlations between self-report measures and neurocognitive task performance assessing compulsivity and impulsivity

	Sample size (n)	**Self-rating scale**	**Task and metric**	**Correlations between measures**
Chamberlain et al (2006)	20	BIS-11 – total score	CGT-%RD	-
Boisseau et al (2012)	19	BIS-11 – total score	SST-SSRT	$p>.28$
Boisseau et al (2013)	19	FMPS – total score	IGT-net performance	$p>.25$
Sohn et al (2014)	80	BIS-11 – total score	SST-SSRT	$p>.05$
Grassi et al (2015)	38	BIS-11 – total score	IGT-final net score	*Non-sig (No data)*
Grassi et al (2019)	44	BIS-11 – total score	IGT-net score	-
Frydman et al (2020)	17	BIS-11 – total score	SST-SSRT	*Non-sig (No data)*
Sternheim et al (2022)	72	DFlex – total score	IDED-no. of trials to reach S9	-

Note: BIS-11 = Barratt Impulsiveness Scale-11, attentional imp = attentional impulsivity, FMPS = Frost Multidimensional Perfectionism Scale, DFlex = Detail and Flexibility Questionnaire, CGT-%RD= Cambridge Gambling Task - % of rational decisions, SST-SSRT = Stop Signal Task-Stop Signal Reaction Time, IGT = Iowa Gambling Task, IDED-no. of trials to reach S9 = Intra Extradimensional set-shifting task- number of trials to reach stage 9.

Of the 112 eligible studies identified in the review, 139 independent comparisons were extracted. They were all cross-sectional. For compulsivity, 59 studies used the Wisconsin Card Sorting Test (Milner, 1963), and 13 used the Intra-Extradimensional Set Shifting Task (Owen et al, 1991); for impulsivity, 18 used the Stop Signal Task (Aron, Robbins, and Poldrack, 2004), and seven used the Cambridge Gambling Task (Rogers et al, 1999). Other impulsive-related neurocognitive tasks also included: 22 using the Iowa Gambling Task (Bechara et al, 1994), 11 using the Go/No-Go task (Gomez, Ratcliffe, and Perea, 2007), one used the temporal discounting task (Rubia et al, 2009), and three used the Conner's continuous performance task-II (Conner and Staff, 2000).

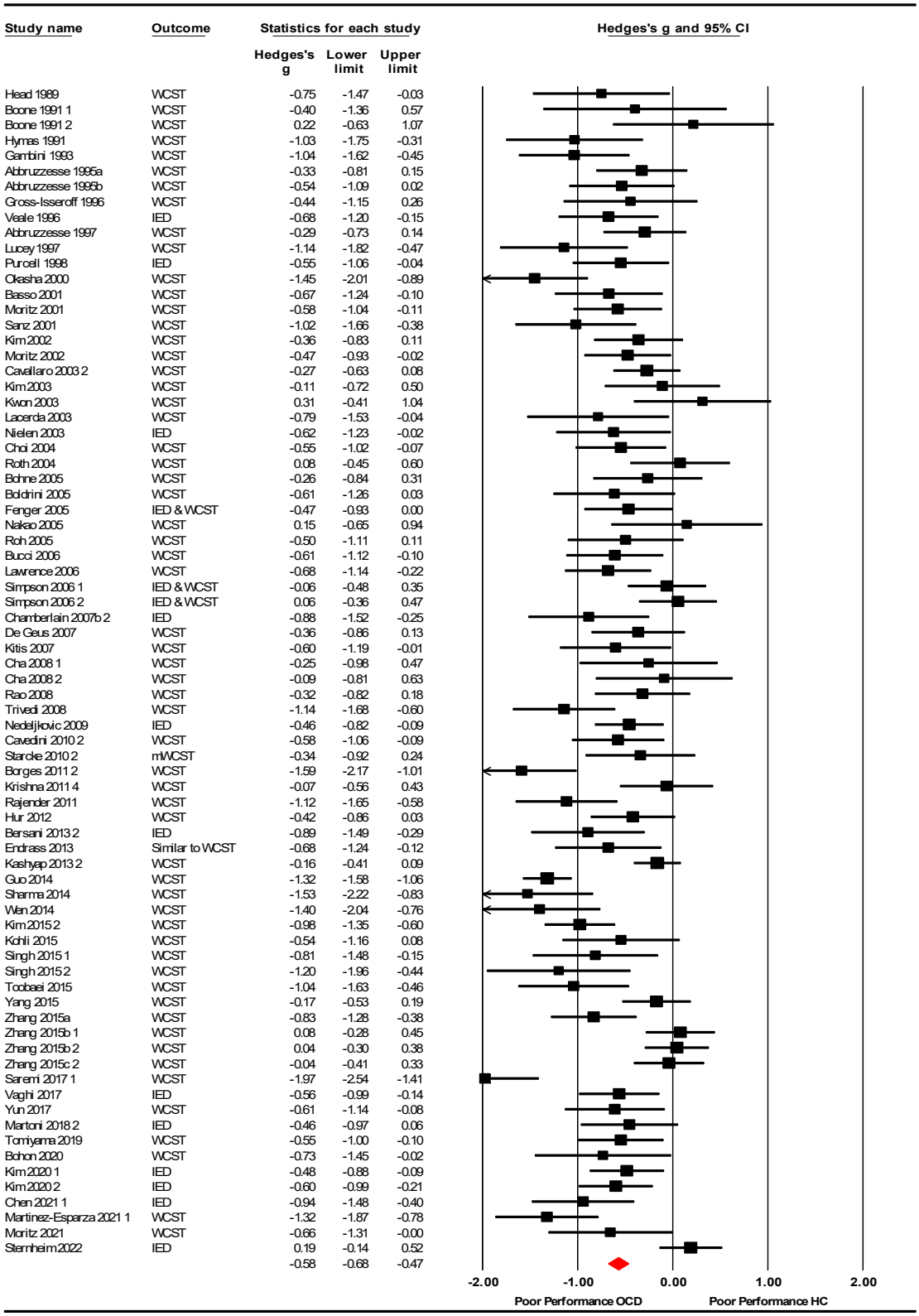
Four studies used two neurocognitive tasks to assess the same compulsivity or impulsivity phenotype; Fenger et al (2005) and Simpson et al (2006) used both the WCST and the IED to explore compulsivity whilst Chamberlain et al (2007b) and Frydman et al (2020) used both the CGT and the SST to explore impulsivity. Similarly, Krishna et al (2011) and da Rocha et al (2011) used the IGT and the CCPT-II for impulsivity. The scores of participants 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 cognitive domains e.g. the IED and the CGT (Cavallaro et al, 2003; Chamberlain et al, 2007b; Cavedini et al, 2010; Starcke et al, 2010; Borges et al, 2011; Krishna et al, 2011; Bersani et al, 2013; Kashyap et al, 2013; Kim et al, 2015; Zhang et al, 2015b; Zhang et al, 2015c; Saremi et al, 20017; Martoni et al, 2018; Kim et al, 2020; Chen et al, 2021; Martinez-Esparza et al, 2021). Although Lawrence et al (2006) and Blom et al (2011) use the IGT, effect sizes from the data presented in these papers could not be calculated and they were excluded from the meta-analysis.

Neurocognitive Impulsivity vs compulsivity

Random effects meta-analyses were performed for: 76 comparisons of participants with OCD and healthy controls on neurocognitive compulsivity measures and 63 comparisons on neurocognitive impulsivity measures. Participants 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); see Figure 2.3. Similarly, impulsive neurocognitive tasks revealed worse performance in participants with OCD than healthy controls (g = -0.48, [95%CI -0.57, -0.38]; k = 63, p <.001); substantial heterogeneity

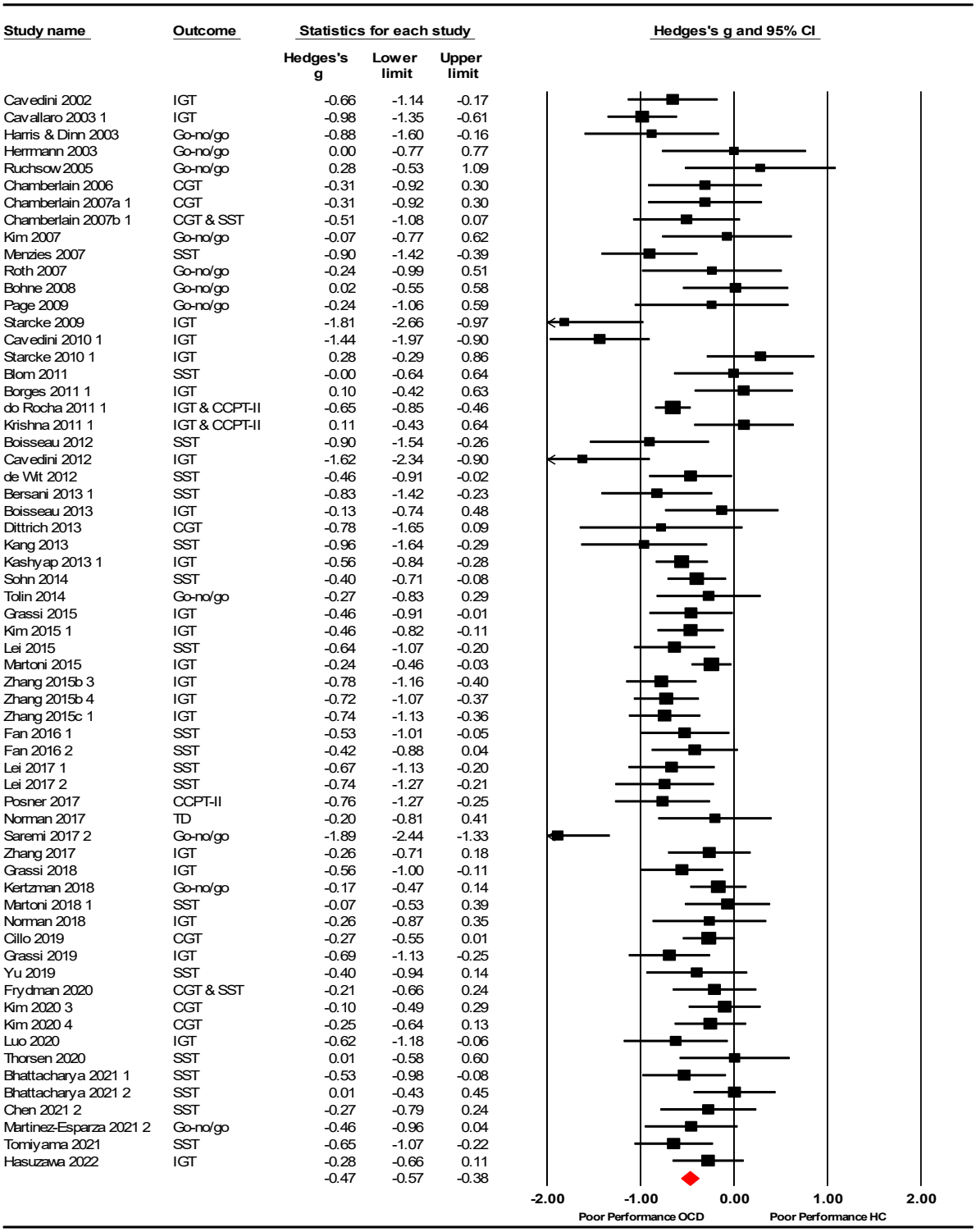
was observed across these effect sizes ($I^2 = 58.48$, $p<.001$); see Figure 2.4. The effect sizes for compulsivity and impulsivity did not significantly differ ($Q=1.99$, df =1, $p=.16$).

Figure 2.3. Compulsivity measures effect sizes.



Note: WCST = Wisconsin Card Sorting Test, IED = Intra-Extradimensional Set-Shifting Task

Figure 2.4. Impulsivity measures effect sizes.



Note: IGT = Iowa Gambling Task, CGT = Cambridge Gambling Task, SST = Stop Signal Task, TD = Temporal Discounting Task, CCPT-II = Conner's Continuous Performance Task-II.

Small study effects and potential publication bias was considered by examining funnel plots. The funnel plot for compulsive-related studies (Figure 2.5) 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 (Figure 2.6).

A subgroup analysis of compulsivity tasks 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.

Figure 2.5. Funnel plot for studies assessing compulsivity.

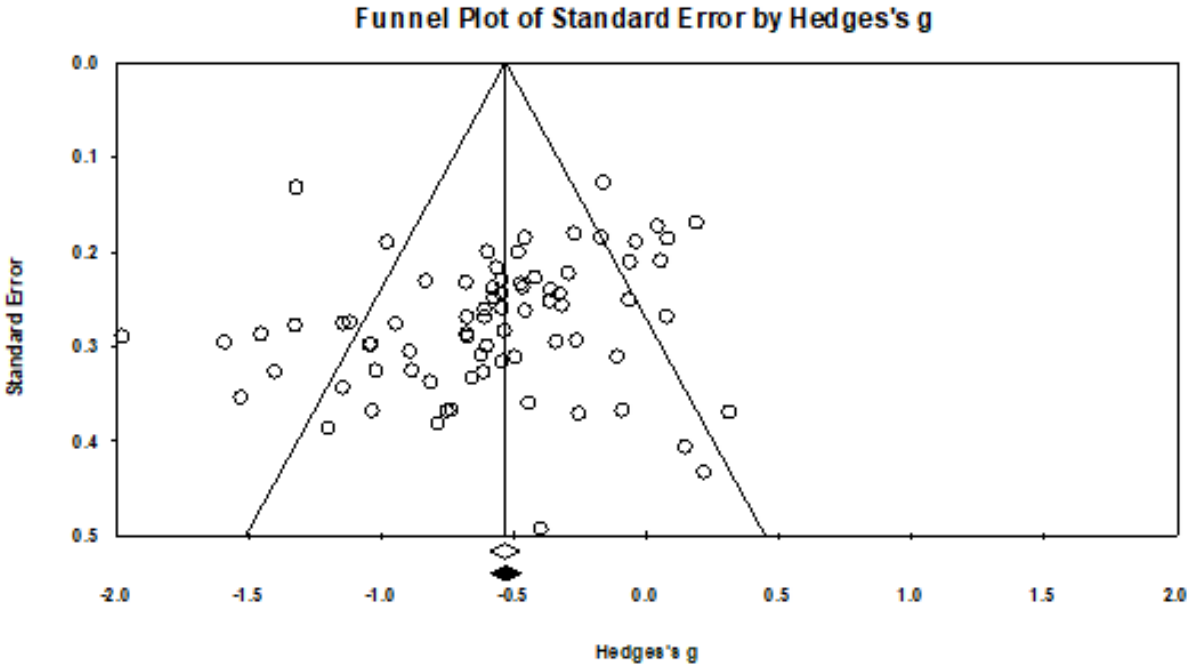
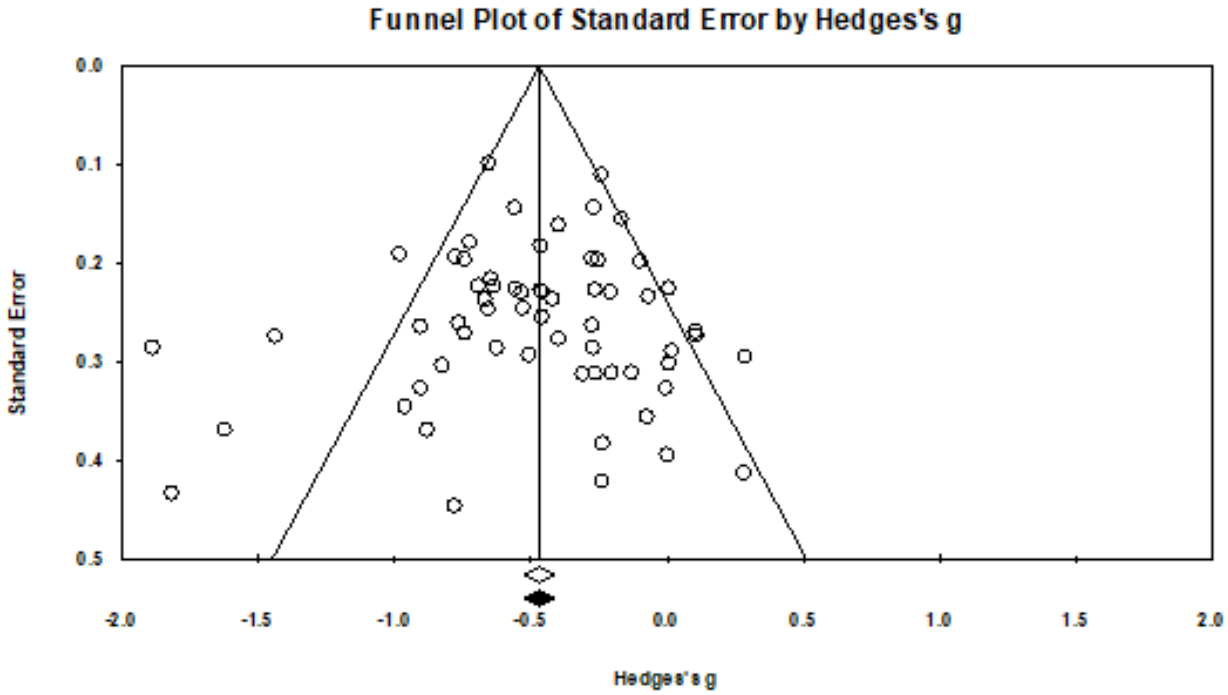


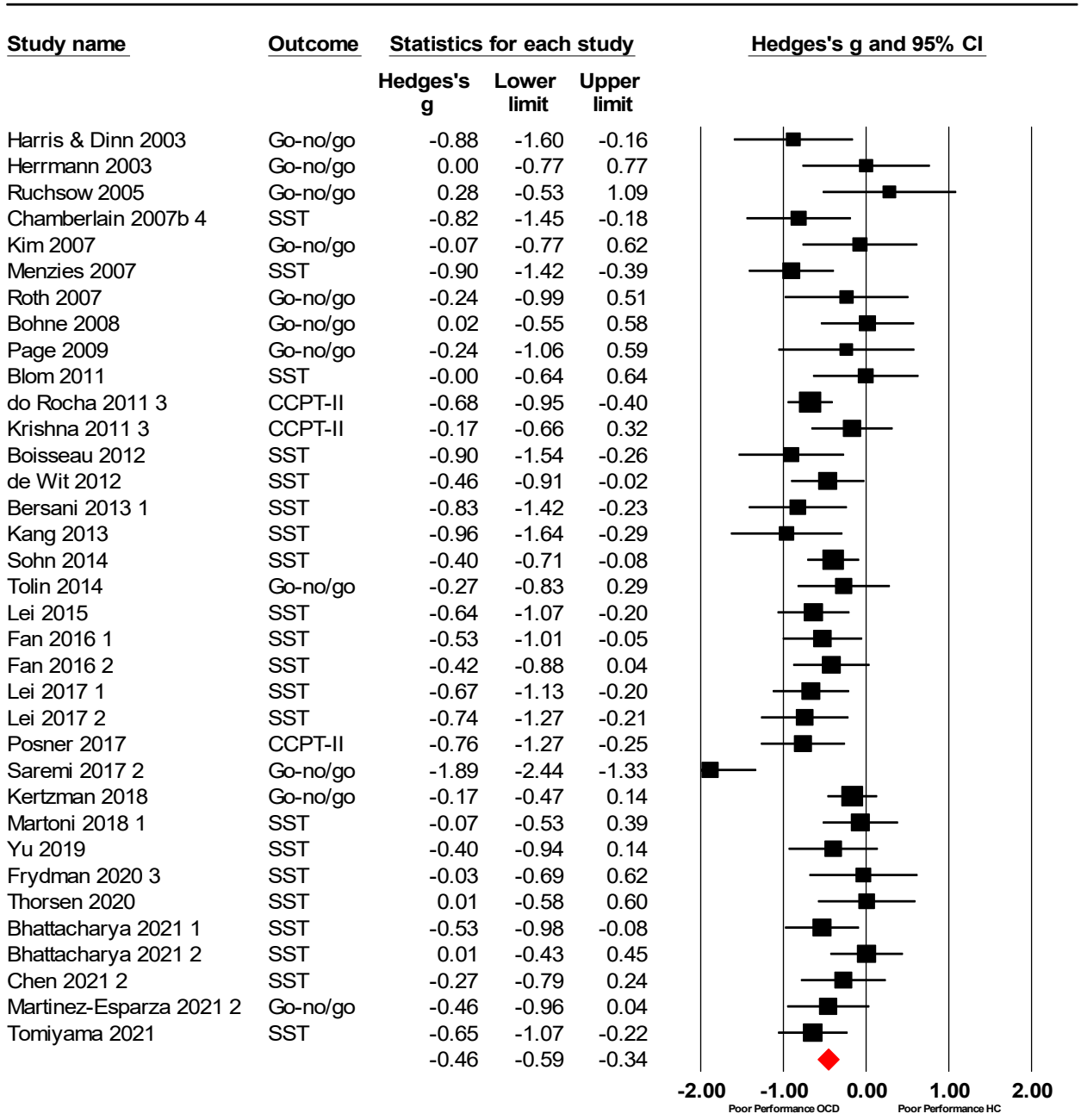
Figure 2.6. Funnel plot for studies assessing impulsivity.



Motor Impulsivity vs Decision-Making Impulsivity

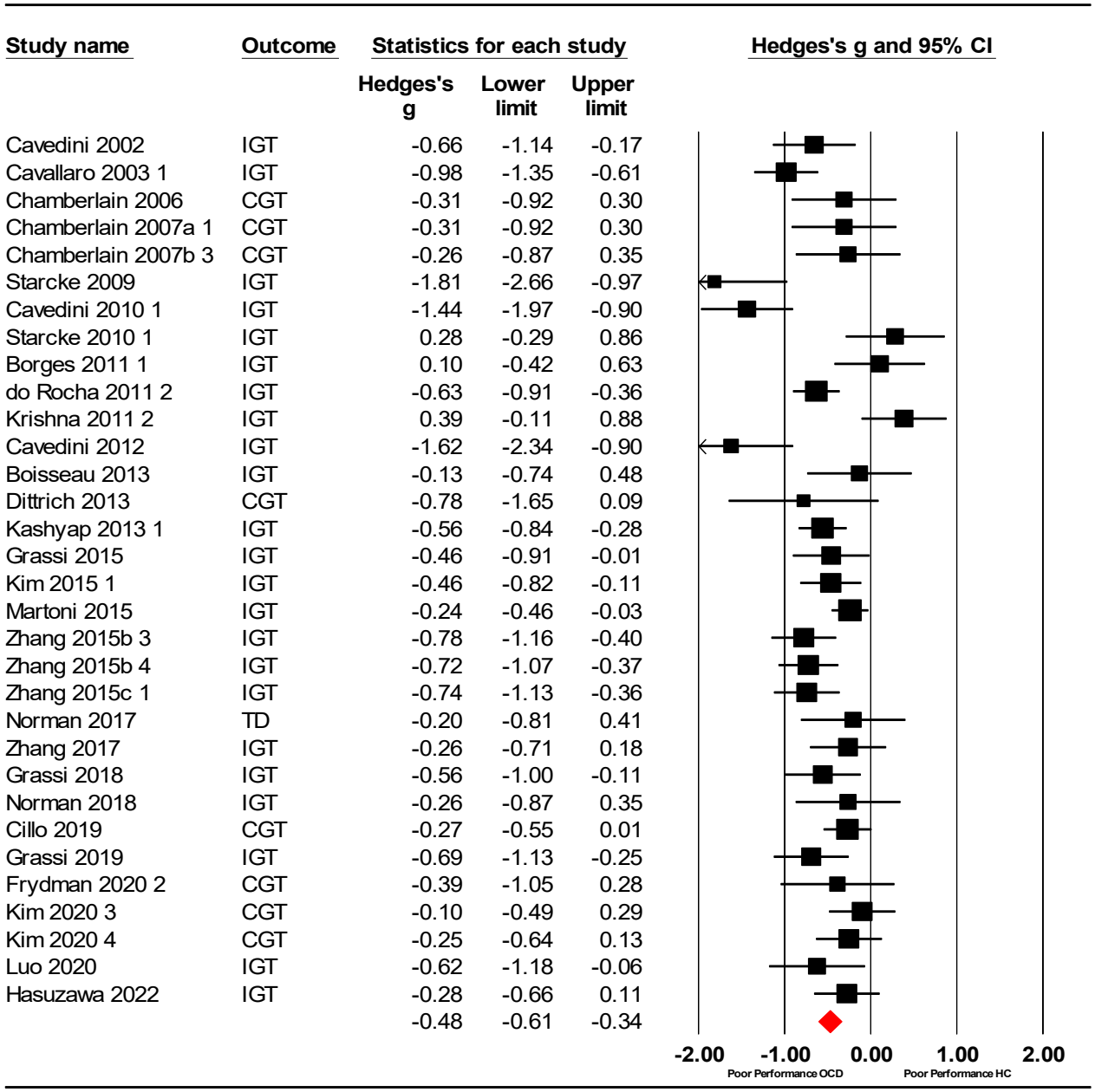
A subgroup analysis contrasted the two facets of impulsivity: motor and decision making (Figure 2.7 and 2.8 respectively). A small to moderate effect size was observed for motor impulsivity (g = -0.46, [95%CI -0.59, -0.34]; k = 35, p <.001; I^2 = 52.23, p<.001). The funnel plot showed no evidence of 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.

Figure 2.7. Motor impulsivity.



Note. SST = Stop Signal Task, CCPT-II = Conners Continuous Performance Task-II

Figure 2.8. Decision-making impulsivity.



Note. IGT = Iowa Gambling task, CGT = Cambridge Gambling Task, TD =Temporal Discounting Task

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. 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).

Subgroup and moderator analyses

Comorbidities

In many studies, comorbidities were an exclusion criterion; those with diagnoses adjunctive to a primary diagnosis of OCD were removed from trials. Subgroup analyses were conducted to examine the influence of comorbidity on the task performance of participants with OCD (compared to healthy controls) with and without comorbidities (See Appendix C and D 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).

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. 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.

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 2.4, two significant moderators emerged: greater impulsivity impairment occurred in studies with a greater proportion of female participants 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. Exploratory meta-regressions using years in education and intelligence (IQ) did not show any moderation of compulsivity or impulsivity effects sizes.

Table 2.4. Meta-regressions using clinical and demographic variables to predict compulsivity and impulsivity.

		Mean (SD)	**Range**	**Z-test**
Age				
	Comp (k=75)	31.29 (4.94)	15.64-41.47	Z=0.83, *p*=.41
	Imp (k=60)	30.81 (6.60)	15.75-44.75	Z=-0.16, *p*=.88
Proportion of Females				
	Comp (k=73)		17.64-100.00	Z=-1.24, *p*=.22
	Imp (k=59)		0.00-100.00	**Z=-2.10, *p*=.04***

Duration of illness				
	Comp (k=50)	9.62 (4.40)	2.80-21.60	Z=-0.14, *p*=.89
	Imp (k=28)	9.62 (4.01)	3.25-18.24	Z=1.00, *p*=.32
OCD symptoms (Y-BOCS)				
	Comp (k=60)	24.45 (2.52)	18.00-33.64	Z=0.21 , *p*=.83
	Imp (k=54)	23.73 (3.71)	13.91-30.76	Z=-1.51, *p*=.13
Anxiety scores (HAM-A)				
	Comp (k=35)	11.29 (4.01)	1.93-16.90	Z=0.77, *p*=.44
	Imp (k=27)	9.07 (3.33)	4.05-15.16	Z=-0.62, *p*=.53
Depression scores (HDRS)				
	Comp (k=49)	10.57 (5.49)	2.47-24.40	Z=-1.08, *p*=.28
	Imp (k=41)	7.47 (3.40)	3.98-14.37	Z=-0.45, *p*=.65
Years in education				
	Comp (k=55)	13.13 (1.46)	10.34-17.20	*Z*=0.75, *p*=.45
	Imp (k=38)	13.40 (1.27)	10.87-17.20	*Z*=1.75, *p*=.08
Intelligence (IQ)				
	Comp (k=31)	107.16 (5.79)	93.50-114.70	*Z*=0.89, *p*=.37
	Imp (k=19)	111.50 (6.08)	93.50-117.70	*Z*=-0.92, *p*=.36
AXIS				
	Comp (k=76)	15.18 (1.10)	13.00-18.00	**Z=2.36, *p*=.02***
	Imp (k=62)	15.57 (1.07)	13.00-18.00	Z=-0.53, *p*=.60
Year of Publish				
	Comp (k=76)	2007.88 (8.36)	1989-2022	Z=-0.81, *p*=.42
	Imp (k=62)	2013.84 (5.36)	2002-2022	Z=1.10, *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

Exploratory analyses

Outpatient vs Inpatient recruitment

An exploratory subgroup analysis was performed to explore patient care (inpatient versus outpatient) on task performance (See Appendix E and F for Forest plot). Compulsivity effect sizes were significant for both participants with OCD in 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, inpatient participants with OCD (*g* =-0.50, [95%CI -0.74, -0.26];k = 13, *p* <.001; I^2 = 76.52, *p*<.001), outpatient participants with OCD (*g* = -0.47, [95%CI -0.60, -0.34];k = 36, *p* <.001; I^2= 56.41, *p*<.001) and mixed outpatient and inpatient participants with OCD 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 between participant samples emerged (*Q* =0.10, df =2, *p* =.95).

Effects of medication

The potential moderating effect of medication on effect size 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 G and H for Forest plots. 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* =-.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 medication-withdrawn samples (k=37; 25.30, SD=2.52) (t (98) =-3.93, p<.001). No difference was found in either compulsivity or impulsivity across these subgroups, further evidencing the independence of the latent cognitive phenotypes from symptom severity.

Reporting Quality, Study Design and Risk Of Bias for included studies

The quality of the included studies was assessed using the AXIS checklist (Appraisal tool for Cross-Sectional Studies: Downes et al, 2016), which was 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 within each study (items: 6, 7, 9, 13, 14, and 15). Following recent research (Antczak et al, 2020), AXIS quality scores were classified 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%); see Table 2.1 for total AXIS scores for each of the 112 studies. Reporting quality among all 112 studies was excellent (mean score 6.70 (SD=0.46)), study quality was satisfactory (mean score 5.45 (SD=0.62)), as was publication bias (mean score 3.16 (SD=0.58)). The mean AXIS score across all 112 studies was 15.36 (SD=1.10). Information pertaining to the number of papers meeting each AXIS criterion can be found in Table 2.5.

Table 2.5. Total number of studies meeting AXIS criteria items

AXIS Item	Yes	No	Unsure
Introduction			
1. Were the aims/objectives of the study clear?	112	0	0
Methods			
2. Was the study design appropriate for the stated aim(s)?	112	0	0
3. Was the sample size justified?	0	112	0
4. Was the target/reference population clearly defined? (Is it clear who the research was about?)	112	0	0
5. Was the sample frame taken from an appropriate population base so that it closely represented the target/reference population under investigation?	112	0	0
6. Was the selection process likely to select subjects/participants that were representative of the target/reference population under investigation?	112	0	0
7. Were measures undertaken to address and categorise non-responders?	12	99	1
8. Were the risk factor and outcome variables measured appropriate to the aims of the study?	112	0	0
9. Were the risk factor and outcome variables measured correctly using instruments/measurements that had been trialled, piloted or published previously?	112	0	0
10. Is it clear what was used to determine statistical significance and/or precision estimates? (e.g. p-values, confidence intervals)	112	0	0
11. Were the methods (including statistical methods) sufficiently described to enable them to be repeated?	112	0	0
Results			
12. Were the basic data adequately described?	112	0	0
13. Does the response rate raise concerns about non-response bias?	2	109	0
14. If appropriate, was information about non-responders described?	10	102	0
15. Were the results internally consistent?	110	2	0
16. Were the results presented for all the analyses described in the methods?	112	0	0
Discussion			
17. Were the authors' discussions and conclusions justified by the results?	112	0	0
18. Were the limitations of the study discussed?	79	33	0
Other			
19. Were there any funding sources or conflicts of interest that may affect the authors' interpretation of the results?	48	61	3
20. Was ethical approval or consent of participants attained?	103	9	0

Note: reporting quality – items 1, 4, 10, 11, 12, 16, and 18; Study design – items 2, 3, 5, 8, 17, 19 and 20; and Possible biases – items 6, 7, 9, 13, 14, and 15.

The mean score was 15.33 (SD=1.09) across all 112 studies: 15.21 (SD=1.12) and 15.51 (SD=1.00) for studies assessing compulsivity and impulsivity respectively. Compulsivity and impulsivity studies did not differ in research quality, potential bias, or total AXIS score (all p>05: see Table 2.6) but did differ in study design with impulsivity showing greater study design than compulsivity studies. The year of publishing showed a positive relationship with both sample size (r=0.34, p<.001), research quality (r=0.47, p<.001) and with study design (r=0.36, p<.001) but no significant relation with the potential bias (r=-0.03, p>.05). Hence, recent studies have employed larger samples and show better powering and better study quality.

Table 2.6. Axis quality scores.

	Impulsivity (k=57) Mean (SD)	Compulsivity (k=70) Mean (SD)	t-test
Research quality	6.79 (0.41)	6.68 (0.48)	t (125) = 1.65, p =.10
Study design	5.60 (0.53)	5.37 (0.64)	t (125) = 2.13, p<.05
Potential of bias	3.12 (0.57)	3.19 (0.60)	t (125) = 0.60, p=.55
Total AXIS score	15.51 (1.00)	15.21 (1.12)	t (125) = 1.55, p=.12

Representativeness

As shown in Table 2.5, studies were consistent in their description of the target population, their respective samples, and the selection process. The status of the clinic (outpatient and inpatient) was clear in all 112 papers. Participant exclusions were also detailed, typically if participants had a history of neurological injury of some description as well as sustained administration of psychotropic medication as some forms were described as having effects on brain morphology (Lieberman et al, 2005). Moreover, all studies declared the diagnostic tool that was employed in the screening for OCD.

Sample size justification

Though the 112 studies were rated moderate to high quality, some notable shortcomings of the included articles do emerge. As shown in Table 2.1, sample sizes were not justified in any of the 112 studies. This does bring the statistical power of these papers under scrutiny. The mean sample size was 71 (median = 56) indicating that between subjects designs are sufficiently powered to detect moderate and large effect sizes (both .99) but not small effect sizes (.51); effect size estimates were taken as .20, .50, and .70 for small, moderate, and large effects, respectively (Cohen, 1988).

Of the 50 studies published after the innovation of the Open Science Framework repository in 2013, only four studies (8%) have pre-registered the protocol of their intended aims, hypotheses, and analyses (Zhang et al, 2017; Frydman et al, 2020; Thorsen et al, 2020; and Sternheim et al, 2022).

Interestingly, the year of publishing showed a positive relationship with sample size (r=0.34, p<.001), research quality (r=0.45, p<.001) and 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.

Non-respondence rates and bias

As discussed, information regarding participant exclusions prior to experimentation (such as neurocognitive lesions and particular psychotropic medications) was reported, however, less detail was given as to withdrawals, drop-outs, and incomplete datasets. As shown in Table 2.1, only 12 papers acknowledged non-responses within their cohorts. Of these 12, only two papers were indicative of non-response bias as the remaining 10 provided information about their respective non-response rates.

Discussion of limitations

Of the 112 studies identified in the literature review one in four (29.46%) failed to provide any discussion of their respective limitations. The earliest publication to omit an appraisal of limitations is Head et al (1989) and the latest is Cillo et al (2019).

Ethical approval

Another oversight in a small number of papers (k=9) is an omission of detailing pertaining to the procurement of (a) informed consent from participants, and (b) ethical approval from a recognised institutional committee.

DISCUSSION

Do participants with OCD differ significantly from healthy controls on tasks of compulsivity and impulsivity?

This systematic literature review and meta-analysis was conducted to provide a narrative synthesis of empirical evidence pertaining to executive functioning in patients with OCD. The current meta-analysis provides the most comprehensive meta-analysis to date (112 studies with 8,313 participants) and is the first to document significant deficits on specific cognitive tests representing both cognitive phenotypes of compulsivity and impulsivity in participants 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 participants 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.

As previously discussed, Fradkin et al (2018) contended with an argument of functioning flexibility in patients within OCD (i.e., performance within normal parameters), which stands in contrast to these findings presented here where there is a moderate effect of deficient flexibility in patients with OCD. However, Fradkin et al (2018) utilised a composite of multiple testing paradigms and so may have diluted effects of specific compulsive substrates. Similar to Chamberlain et al (2021), this current meta-analysis focuses on attentional set-shifting to detail compulsive manifestations owing to deficient cognitive flexibility, and whilst the effects reported by Fradkin et al (2018) may reflect attentional set-shifting, it is difficult to discern this from other compulsive dimensions such as reversal learning, alternation, and other task-switching paradigms found among the trail making and stroop tasks. Indeed, Gruner and Pittenger (2017) stress the need to examine these substrates independently of each so as not to convolute interpretations.

With regard to inhibitory control, the findings reported here are reflected in previous meta-analyses that show impaired response inhibition in patients with OCD. Norman et al (2019) showed that, compared to a healthy control, patients with OCD demonstrated impaired performance on tasks of response inhibition (0.22, $p = .02$; 95%CI [0.039, 0.399]), one of these being the Stop-Signal Task. Interestingly, the underlying neural correlates of this impaired inhibitory control in OCD appears to be distinct from that of ADHD (Norman et al, 2016) and ASD (Carlisi et al, 2017); larger insular-striatal portions with concomitant deficiencies in prefrontal cortices. It follows that this deficit in OCD has a unique neurobiological profile and may serve as a candidate biobehavioural marker that can be used

in conjunction with other units of analysis to render comprehensive diagnoses. However, it is important to note that the described meta-analyses fall short of sufficient statistical controlling of type 1 errors and so this may not be wholly reflective of true effects (Eickhoff et al, 2016). This, coupled with more recent analyses showing no such deficient inhibitory control in patients with OCD (Suñol et al, 2020; Hollestein et al, 2021) may raise concerns of replicability for meta-analytical findings (Uhre et al, 2022).

As such, it is paramount that research undertaking assessments of neurocognitive performance select specific dimensions relevant to experimental hypotheses and maintain ample statistical controlling in line with published standardised guidelines and recommendations. This will go some way in addressing the inconsistencies in executive performance in patients with OCD, as is addressed in both Chapter 1 and this current chapter.

Compulsivity, impulsivity, and OCD symptom severity

Researchers have repeatedly commented upon the potential influence of symptomatology on cognitive function and particularly regarding executive function. Abramovitch et al (2012) 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, Abramovitch and colleagues have argued that executive deficits are a secondary causal consequence of OCD symptomatology and they further infer 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 (2019) reported significant inverse correlations for OCD symptomatology with performance on both response inhibition (r=-0.280) and set-shifting (r=-0.277) tasks. The findings of this meta-analysis are only partially consistent as a significant aggregated correlation was revealed between Y-BOCS

scores and neurocognitive task measures of compulsivity, but not between Y-BOCS scores and neurocognitive task measures of impulsivity (r=0.30 vs r=-0.08). By contrast, the meta-regressions in this analysis provide no support for a significant relationship between OCD symptomatology (as measured by Y-BOCS scores) and either compulsivity or impulsivity effect sizes, suggesting a relative orthogonality between symptom severity scores and impaired neurocognitive task performance in between-group contrasts. The mean Y-BOCS scores across the compulsivity and impulsivity samples was 24.45 and 23.73 respectively, indicating OCD symptomatology to be at the upper-end of moderate severity (Storch et al, 2015) - ranging from 13.91 (Bhattacharya et al, 2021) to 30.76 (Fan et al, 2016). Hence, the failure to find a relationship does not reflect a lack of variability or low levels of OCD symptomatology.

The main difference between the current meta-analytic study and that of Abramovitch et al (2019), is that moderator analyses in this analysis are predicting variability in executive effect size *differences* between participants 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 participants with OCD only. These findings are not necessarily inconsistent as the degree of cognitive impairment for participants with OCD 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). Finally, while the Y-BOCS remains one of the most well validated scales for measuring symptom severity in OCD, it may not capture the full extent of OCD including commonly associated cognitive deficits.

Consistent with the independence of cognitive deficits and symptomatology, studies of participants with OCD in remission, show that they continue to exhibit executive impairment

in the domains of compulsivity and impulsivity (Rao et al, 2008; Sharma et al, 2014). Sharma et al (2014) assessed 15 remitted participants with OCD with Y-BOCS scores of 0-7, while Rao et al (2008) assessed 30, who were asymptomatic having mean Y-BOCS =2.57 and no clinically significant concurrent depression (mean Hamilton Depression Rating Scale score=1.97) or anxiety (mean Hamilton Anxiety Rating Scale score=1.93). Both studies found that compared to matched controls, those who had recovered from OCD continued to show impaired performance on tests of compulsivity and impulsivity. Some authors have further speculated that such cognitive deficits may be associated with or even underpin the occurrence of symptomatic relapse (Bannon et al, 2006). Bannon et al reported impaired functioning on tests of compulsivity (WCST) and impulsivity (Go/No-Go; Stroop) in 60 participants with OCD compared to an anxiety (panic disorder) control group. Crucially, they followed up 20 participants with OCD (on average 1.4 years later) who had remitted, and these individuals continued to show impaired performance on measures of compulsivity and impulsivity. 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.

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

Our analyses go further in showing that variance in compulsivity and impulsivity deficits across studies is not associated with severity of OCD symptoms or other common conditions and symptomatology that often occur comorbidly with OCD (such as depression and anxiety symptoms). No differences in effect size emerged for compulsivity or impulsivity when studies that included participants with comorbid psychiatric problems were compared to

those that did not. Comorbidities were varied (see Appendix A and B), but data were assessed concerning the common comorbid symptoms of depression and anxiety. Major depressive disorder is the most common comorbidity in OCD (Sharma et al, 2021), with high rates of current (32%) and lifetime (77%) comorbidity existing between OCD and MDD (Brown et al, 2001). Individuals diagnosed with MDD alone are known to be impaired on tests of compulsivity and impulsivity and such deficits are related to depression severity (Snyder, 2013). Indeed, concurrent depressive symptomatology has been advanced as a possible explanation for the impaired executive function performance in participants with OCD (see Basso et al 2001; Moritz et al, 2001). Nonetheless, the analyses of large numbers of compulsivity (k=49) and impulsivity (k=41) studies found no significant association with depressive symptomatology (as assessed using the Hamilton Depression Rating Scale). Similarly, anxiety symptoms as measured using the Hamilton Anxiety Rating Scale (Hamilton, 1959) also failed to predict effect sizes for either compulsivity (k=34) or impulsivity (k=27). The mean HDRS and HARS 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.

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

Compulsivity and impulsivity are related to a categorical diagnosis of OCD, but according to meta-regression the variance in effect sizes is not explained by clinical variables under test including severity of OCD symptomatology. The dissociation between symptom severity and cognitive phenotypes may act as a proxy for the distinction between *state* and *trait* components of OCD respectively (Snyder et al, 2015). 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. This stability is also reported in previous meta-analyses by Shin et al (2014) and Snyder et al (2015) who both demonstrated that deficits in cognitive flexibility and response inhibition in patients with OCD were unrelated to demographic and clinical variables such as age, gender, medication use, comorbid depression, and OCD symptomatology; though similar to this meta-analysis, they were also unable to unpick specific relationships between executive deficits and symptom subtypes.

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, unaffected first-degree relatives of those with OCD also show impairments on compulsivity and impulsivity tasks (e.g., Bora, 2020; Zartaloudi, Laws, and Bramon, 2019). For example, Bora (2020) reports that relatives of those with OCD perform poorer than healthy controls on tasks of both impulsivity (Stroop and Stop-signal tasks: d=0.58, 95%CI 0.29 to 0,86) and compulsivity (WCST, IDED and Trial Making Task: 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. Due to minimal inclusions of first-degree relatives within included studies, subgroup analyses comparing unaffected first-degree relatives with participants with OCD and healthy controls could not be performed and so the neurocognitive performance of a subclinical profile could not be ascertained.

The current findings partly accord with a recent meta-analysis of paediatric OCD (Lopez-Hernandez et al, 2023) in child and adolescent samples which shows significant performance deficits on tests for compulsivity (*d*=-0.42; 95%CI -0.61 to -0.14; k=12), inhibition (referred to in this current meta-analysis as motor impulsivity) (*d*=-0.22; 95%CI -0.34 to -0.11; k=15), and decision-making (*d*=-0.17; 95%CI -0.41 to 0.08; k=3). 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.

The mean period of time since diagnosis reported by Lopez-Hernandez et al (2023) was 3.7 years, while the illness duration across the adult samples in this meta-analysis was of course much longer, exceeding 10 and 15 years for impulsivity and compulsivity studies respectively. The series of planned meta-regression analyses showed that duration of illness failed to predict either compulsivity or impulsivity effect sizes in adults with OCD. Although significant, the mean effect size for impulsivity in the younger samples reported by Lopez-Hernandez et al (2023) falls below the lower end of the 95% confidence intervals reported here for adult samples. The current findings in conjunction with those of Lopez-Hernandez et al (2023) suggest that the compulsivity and impulsivity performance deficits may be relatively stable from the early development of OCD that stabilise from late adolescents and throughout adulthood.

Heterogeneity across tasks

Although the included studies were of relatively good design and quality – with minimal evidence of small study biases, moderate to considerable heterogeneity was observed in all subgroup analyses. Though the study samples were restricted to only include those with a formal diagnosis of OCD (made by structured diagnostic tools), some variability in the clinical presentation of the disorder itself may partly underpin this high degree of heterogeneity. As can be seen in Table 2.1 (study characteristics), several OCD-derivatives might account for this; familial vs sporadic (Boone et al, 1991; Bhattacharya et al, 2021), reactive vs autogenous obsessions – obsessions resulting from an overt external stimulus compared to obsessions that are not (Fan et al, 2016), cleaning vs checking compulsions (Cha et al, 2008), early onset vs late onset (Lei et al, 2017; Kim et al, 2020), and finally, OCD with depression vs OCD without depression (Singh et al, 2015). These derivates are too few to subject them to a subgroup analysis.

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 analyses focused on data relating to WCST perseverative errors and the extra-dimensional shift stage of the IED (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 IED metrics are viewed as the classical outcomes associated with cognitive flexibility/rigidity of thinking and although the sensitivity analysis revealed no difference in their effect sizes, there were instances where uniformity among metrics was not possible and so an alternative metric was chosen as the best fit e.g., % of perseveration or total errors in the absence of perseverative errors for the WCST. In cases such as these, the primary outcome measure as described by the authors was extracted.

The latent phenotypes of compulsivity and impulsivity investigated here are not mutually exclusive and their corresponding tasks are complex, multifaceted, and partially overlapping (see Miyake et al, 2000). For example, cognitive tasks assessing both compulsivity and impulsivity may contain aspects of response inhibition to navigate and meet the task demands, which may contribute to their comparable effect sizes. Nonetheless, performance on these tasks are potentially dissociable. For example, participants with OCPD show impairments on the IED but not the SST (Grant and Chamberlain, 2019), and conversely, participants with trichotillomania show deficits on the SST but not the IED (Chamberlain et al, 2006). Furthermore, some evidence suggests that the task metrics examined in the current meta-analysis may be relatively separable. Steinmetz and Houssemand (2011) assessed the relationship between behavioural inhibition and various metrics from the WCST. They found that WCST non-perseverative errors were much stronger predictors of inhibition functions (Go/No-Go and Stop Signal) than WCST perseverative errors (which were the focus of this meta-analysis).

The differences within- as well as between-latent phenotypes were also explored as impulsivity is not a unitary construct (Robbins et al, 2012). Inhibitory control 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). This meta-analysis identified moderate effect sizes indicating impairment of both (g= -0.46 and g= -0.48 respectively).

Our findings largely accord with a recent meta-analysis by Mar et al (2021) documenting impaired inhibitory control in participants with OCD compared to controls (measured as raw mean differences in RTs on the SST) with greater impairment in older aged samples. An exploratory analysis of 14 studies in this 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 (Mancini et al, 2018; Mar et al, 2021), but has presented an indistinct consensus regarding decision-making impulsivity, with some research reporting the relationship (Grassi et al, 2018; Nistico et al, 2021; Croft et al, 2022) and others not (Abramovitch and McKay, 2016).

While the 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 Chamberlain et al 2005) 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 under 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). In consideration of decisional impulsivity in OCD, as well as for the wider examination of impaired decision-making capacities, this distinction between the IGT and the CGT suggests that these tasks are not wholly interchangeable and should not be considered together. This certainly aligns with Enkavi et al (2019)'s call for improved systematic use of executive tasks. However, this may also partly reflect the somewhat greater cognitive demands of the IGT than the CGT.

Treatment implications

While the medicated samples included here showed lower symptom severities than unmedicated and medication-withdrawn samples, these samples did not differ significantly in compulsivity and impulsivity measures. It is worth noting that a proportion of these medicated samples were also receiving benzodiazepines and antipsychotics such as risperidone or olanzapine (see Appendix I). These adjunctive medications may have mitigated the observable effects. Similarly, results from individual studies suggest executive function deficits in patients with OCD have been found unresponsive to psychological interventions such as CBT (Vandborg et al, 2015) and other medical interventions e.g., deep brain stimulation of the nucleus accumbens and the anterior limb of the internal capsule (Huys et al, 2019) despite symptomatology responding. In contrast, Tyagi et al (2019) found that DBS targeting the subthalamic nucleus and the cognitive corticostriatal loop (but not the orbitofrontal loop) improved OCD symptoms and IDED performance, strongly implicating the specific neural circuit in the origins of compulsive features such as cognitive inflexibility and a specific interventional target for those patients with IDED 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.

OCD Symptom Severity against Self-Rated assessments

Self-rated assessments of compulsivity and impulsivity were extracted from the included papers. A collection of 8 papers utilised a self-rating measures adjunct to the neurocognitive task assessment (see Table 2.2), of which, 6 papers use the Barratt Impulsiveness Scale-11 (Patton, Stanford, and Barratt, 1995); analyses could not be conducted on such a small

collection of papers. Three papers reported correlational analyses though only two report significant findings. Boisseau et al (2012) and Frydman et al (2020) concur with greater impulsiveness with greater symptom severities. Boisseau et al (2012) showed that the attentional impulsivity subscale of the BIS-11 in particular was related to obsessional Y-BOCS symptoms and that this impulsivity is a manifestation of obsessional anxiety; no relationships were observed for compulsive symptoms or total score. Alternatively, Frydman et al (2020) used the OCI-R to estimate their sample's symptom severity and so the correlation in this analysis may speak towards a relationship between attentional impulsivity and particular symptom dimensions. As the Y-BOCS is a severity scale for OCD symptoms, it does not measure different "dimensions" of symptoms but rather focuses on the severity and frequency of the most apparent symptom components. In contrast, the OCI-R (Obsessive-Compulsive Inventory-Revised) is a dimensional assessment of a broader range of OCD-related symptoms subtypes (e.g., cleaning, washing, ordering). It may be that the impulsivity in OCD may correspond more to obsessional symptomatology. Indeed, Onur et al (2016) does show that the attentional impulsivity subscale of the BIS-11 was greater in patients with sexual and aggressive obsessions compared to patients with alternative obsessional themes. However, further work is needed to confirm this.

Only two papers (Boisseau et al, 2013; Sternheim et al, 2022) included a self-rating of compulsivity. Boisseau et al (2013) used the Frost multidimensional perfectionism scale (Frost et al, 1990) but did not assess any potential relationships with Y-BOCS scores. Sternheim et al (2022) however, used the Detail and Flexibility Questionnaire (Roberts et al, 2011) and reported a moderate positive correlation between the attentional to detail subscale and Y-BOCS scores. As such the relationship between self-rated scales of compulsivity and OCD symptom severity in studies revealed in this literature review could not sufficiently explored.

Contrasting Self-Rated Assessments against Neurocognitive Task Performance

The previously discussed 8 studies that included a self-rating tool adjacent to their neurocognitive task measure were revisited to observe any analyses that had been performed to assess concordance between the two differing measures of latent cognitive phenotypes (see Table 2.3). No significant relationships were revealed in the five papers that do provide a correlational analysis between self-rated tools and neurocognitive task performances. Indeed, research exploring the qualitative and dimensional differences between self-ratings and task performances that assess the same phenotype is scarce. Reynolds et al (2006) showed a distinct orthogonality between the differing methods of assessing impulsivity. These discrepancies may imply that the differing methods of assessment are in fact assessing different aspects of the same phenotype (for example state vs trait features; Enticott and Ogloff, 2006) though more research is needed to confirm this. On the other hand, this may be a reflection of meta-cognitive processes as one application is an assessment of the ability itself and the other is a self-appraisal and commentary of ability and so this disparity between the assessment methods may be a reflection of the different applications of the assessment (Dar et al, 2022).

Correlations between OCD symptom severity and assessments of neurocognitive impairments, both subjectively and objectively, is scarcely reported within the same paradigm. Very little can be derived from the small quantities of analyses described here and so no clear conclusions can be made though there is a recurrent aspect of attentionally impulsive phenomena implicated within the OCD samples in a small collection of papers (De Geus et al, 2007; do Rocha et al, 2011; Boisseau et al, 2012). Moreover, with the discrepancies between the tasks appropriately explored, can the multifaceted natures of these phenotypes be fully acknowledged and understood.

Limitations

Certainly, the included papers are not without shortcomings. With the reclassification of OCD as an Obsessive-Compulsive and Related Disorders in the DSM-5 in 2013 (previously listed under anxiety disorders in the DSM-III, IV, and IV-TR), the representativeness of some samples is questioned as some cohorts that meet and fulfil the diagnostic criteria for a positive diagnosis of OCD under a previous taxonomy may no longer meet the diagnostic criteria of current models. Twenty studies of a total 44 (45.45%) published after 2013 (the publication of the DSM-5) use the DSM-IV as their screening tool, and two studies use the DSM-III for screening after the publication of the DSM-IV-TR in 2000. As such, one in five papers (19.64%) screen their participants under outdated and redundant diagnostic tools.

As shown in Table 2.4, not a single paper justified their sample size, though given the mean sample size of 71, analyses show that this is sufficient to detect medium to large effect sizes. Moreover, only 12 papers sufficiently acknowledge non-responses in their sample. The federal committee for statistical methodology necessitate the provision of such information and outline the best practice for declaring non-response (Madans et al, 2023). In the absence of this information, particular areas of interest may be prone to underestimation as a true representation of effects cannot be rendered (Cheung et al, 2017); the higher the non-response rate, the greater the likelihood of non-response bias (Davern, 2013). Without sample size justifications, it is unclear whether there is any potential for undeclared non-response, and if this non-response rate within the included papers was detrimental to the powering of certain statistical analyses.

Moreover, 33 studies failed to provide a delineation of their respective limitations. This is quite an oversight as it is a standard convention for reporting psychological research (APA

publications and communications board working group on journal article reporting standards, 2008). Indeed, self-disclosing the shortcomings of a paper emphasises gaps in research and highlights recommendations for improved practice; it hinders the replicability and enrichment of the theoretical field (Brutus, Aguinis, and Wassmer, 2012). The limitations of these papers may have had crucial bearing on the interpretations of the results, and as such, the representativeness and generalisability of these findings are thereby restricted.

Studies of neurocognitive function in participants with OCD are notable for the failure to assess quality of life and functional outcomes. Indeed, just one of 112 studies included here assessed the functional impairment of their sample (Frydman et al, 2020). 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 (2006), psychosocial functioning is indeed impaired in OCD, with other authors propounding the relation between functional impairment and poor quality of life (Sahoo, Sethy, and Ram, 2017). With the aim of using quality of life as an outcome metric for therapeutic interventions (Subramaniam et al, 2013), conceptualising the variation in functional impairment between participants with OCD (Vorstenbosch et al, 2012) will aid person centred care.

The vast majority of studies meeting the inclusion criteria were cross-sectional and thus inhibit an appraisal of temporal variance of effects, which longitudinal research might elicit. Indeed, only 3/112 (<3%) of the included studies described follow up assessments (Kim et al, 2002; Roh et al, 2005; Thorsen et al, 2020) across 3 to 4 months. None of these studies report any consistent association between OCD symptoms and the performance on the neuropsychological tests; and so seem to be consistent with the findings of stable impaired neurocognitive performance in the included cross-sectional studies. Further naturalistic

studies across longer time periods, with larger sample sizes, are required to further examine the nature of any relationship between OCD symptomatology and cognitive performance.

Compulsivity has been defined as a result of an imbalance between the brain's goal-directed and habit-learning processes (Gillan et al, 2016). However, few studies have investigated habitual behaviours in OCD using cognitive tasks (e.g., investigating model-based versus model-free decision making) and systematic evidence on this domain is lacking (Daw et al, 2011; Gillan et al., 2015; Voon et al, 2014). For this reason, as reported in the pre-registered protocol, the evaluation of compulsivity focused on the investigation of attentional inflexibility as frequently reported in latent phenotype studies of individuals with OCRDs and in some cases their unaffected family members (Chamberlain et al., 2021). The fact that habit-related learning tasks were not included and analyses instead focused on the attentional component of cognitive inflexibility could, however, be considered a limitation of this analysis. Further studies on the role of habit in compulsivity are warranted to fill this key gap in knowledge.

While no significant relationship between compulsivity or impulsivity effect sizes and Y-BOCS total symptom scores were revealed, the possibility that relationships might exist between cognition and specific symptom clusters cannot be excluded (see Bragdon, Gibb & Coles, 2018). Although the 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 Y-BOCS scores may however be misleading as the same total scores can be generated by quite different symptom-profiles and thus mask substantial heterogeneity amongst participants with OCD (Diniz et al 2015). Future studies should focus on increasing the specificity of both neurocognitive and symptomatic measures as far as possible.

The poorer performance on tests of compulsivity and impulsivity documented here are, however, not unique to participants with OCD and have been documented in other disorders such as major depression (Snyder, 2013), eating disorders (Wu et al, 2014), schizophrenia (Laws, 1999), and bipolar disorder (Robinson et al, 2006). The fact that these deficits are found as common cognitive deficits in many mental disorders has led researchers to contend an argument of non-exclusivity to the diagnostic class of OCD (Abramovitch, Short, and Schweiger, 2021). This may also allude to these neurocognitive impairments being transdiagnostic in nature (Beauchaine and Constantino, 2017).

Conclusions

This comprehensive meta-analysis, involving over 8,000 participants, is the first to highlight the dual presence of neurocognitive compulsivity and impulsivity in individuals with OCD. Seldomly considered together, the current findings reveal that compulsivity and impulsivity are in fact comparable in their respective impairment within OCD phenomenology which may be attributable to shared underlying neurological regions. The findings reveal that these latent cognitive phenotypes are not related to or predicted by clinical status, OCD symptom severity, comorbid psychiatric conditions such as depression and anxiety, and levels of depression and anxiety symptomatology. This orthogonality with typical clinical variables emphasises that the neurocognitive deficits associated with impulsivity and compulsivity appear to be latent cognitive traits of OCD, rather than being directly tied to OCD symptomatology. These findings have implications for current treatment models of OCD. These interventions appear to be adept at targeting state-phenomena such as OCD symptoms but seem ineffective in addressing specific cognitive traits, implying that these impairments in executive control remain even after symptoms have remitted. This calls for precise

identification of these trait markers in OCD presentations. However, this review of 112 studies on impaired executive functioning in OCD highlights the inconsistency in how compulsivity and impulsivity are assessed, both in self-reports and neurocognitive tasks and so work is required to address this. Methodological flaws have hindered the accurate representation of these cognitive traits and as such, future research should focus on isolating and exploring these phenotypes independently while considering their interconnections. Investigating various models of assessment and their relationship with OCD symptom severity will be essential to refine the understanding and treatment of these cognitive components in OCD.

The next chapter assesses OCD symptomatology and executive functioning in a sub-clinical cohort, using both self-rated and neurocognitive tasks to detail latent cognitive traits and their respective relationship to symptom severity. Specifically, the role of OCD symptoms and traits in adjustment to the COVID-19 lockdown is investigated.

CHAPTER 3: FROM LOCKDOWN TO LIBERATION: HOW INFLEXIBLE THINKING, OBSESSIVE-COMPULSIVE AND AFFECTIVE SYMPTOMS SHAPE PANDEMIC ADJUSTMENT

INTRODUCTION

This PhD began during the lockdown period of the COVID-19 pandemic (in April 2021), and as described within the chapter synopses of Chapter 1, the contents of this present chapter were conducted before the preceding chapter. Indeed, the findings and interpretations described below were formative and instrumental in motivating this comprehensive investigation into executive functioning in the OCD spectrum.

The COVID-19 pandemic offered a *unique* opportunity to examine sub-clinical OCD phenomena in at-risk cohorts as well as the potential for developing OCD symptoms and traits as of the stringent lockdown restrictions. The pandemic was utilised as a unique experimental space to observe the predisposing quality of rigid dispositions, not unlike the inflexibility observed in patients with OCD as explored in Chapter 2.

The relationship between OCD symptom severity and neurocognitive task performance is inconsistent; meta-analysis of both continuous and categorical data revealed conflicting findings and the difference in performance on measures of executive dysfunction between patients with OCD and healthy controls cannot be explained by Y-BOCS scores alone (see Chapter 2). The relationship between OCD symptom severity and executive control certainly requires substantiation.

The COVID-19 pandemic introduced a new way of living imposing sanctions on various aspects of everyday life (Haleem and Javaid, 2020). Social distancing and mask mandates were implemented, those considered high-risk went into shielding, as a protective retreat from social engagement, and non-vital vocations were ‘furloughed’, as a form of paid leave.

A gradual easing of lockdown restrictions took place from 23rd March 2020, transitioning to a "living with COVID" phase, which ended in the UK on 19th July 2021 when all pandemic-related restrictions were lifted (Brown et al, 2021; Sherrington, 2022; gov.uk Home Office "*The impact of COVID-19 lockdown on crime demand and charge volumes in England and Wales,* 2024).

Adjustment to the "new normal" post-pandemic lifestyle imposed additional pressures beyond the initial disruption caused by the pandemic with societal implications. Some of these adjustment difficulties may have stemmed from societal factors predating the lifting of restrictions, including controversial differences in the stringency of regulations introduced across different countries, with some jurisdictions imposing more extreme measures than others (Hans et al, 2020; Song and Choi, 2023), as well as changes made to the regulations relating to social distancing and shielding over the course of the pandemic. It is recognized that those with pre-existing mental health conditions may have been particularly affected by the pandemic (Breslau et al, 2021), not least as mental health care and support was heavily impacted by the pandemic (Pereira-Sanchez et al, 2020; Foye et al, 2021; Duden, Gersdorf, and Stengler, 2022). Similarly, those at risk of developing mental health conditions may have had new disorders triggered by the stress of the pandemic and its consequences (World Health Organisation, 2022a).

Fineberg et al (2021) found that individuals with a personal or family history of mental health disorders or symptoms of OCD, Obsessive-Compulsive Personality Disorder (OCPD), depression, anxiety, and stress struggled to adjust to post-pandemic life. Specifically, in a population-based survey of post-pandemic adjustment in over 500 adults, OCD symptoms predicted adjustment difficulty and crucially, this was mediated by depression, anxiety and stress and COVID-19-related anxiety (Fineberg et al 2021). The same study also

demonstrated that OCPD traits, in particular perfectionism and over-conscientiousness, correlated with poorer adjustment to the lifting of lockdown restrictions. The findings of Fineberg et al (2021, 2022) are however limited. A follow up was incorporated a short duration after the main procedural screening, though was conducted on a relatively small sample (n=40).

Cognitive inflexibility, as previous described, is seen across various mental health disorders associated with compulsive behaviour (Patel et al, 2009; McAnarney et al, 2011; Jefferies-Sewell et al, 2016; Grant and Chamberlain, 2023) and provides particularly prominent implications for the lockdown restrictions. Certainly, those that were considered 'poor adjusters' in Fineberg et al's (2021) paper showed impaired performance profiles on the intra-extradimensional set-shifting task (Owen et al, 1991) compared to 'good adjusters'. Though correlations with OCD symptom severity were not assessed, this does suggest that these deficits in executive functioning exist in sub-clinical cohorts and may serve to predispose and precipitate the onset of mental health disorders such as OCD under such adversities. Recent systematic reviews have documented a general exacerbation of obsessive-compulsive symptoms in both OCD and non-OCD cohorts during the COVID-19 pandemic period (Linde, Varga, and Clotworthy 2022; Guzick et al, 2021). Similarly, a study of the Swiss general population (Otte, Schicktanz, and Bentz, 2023) identified a significant increase in those scoring above the clinical threshold of the OCI-R, almost doubling from 13% to 24%. Those with a washing/contamination subtype of OCD were considered particularly vulnerable to exacerbations as the strict hygiene measures may have served to confirm notions of threat avoidance thereby worsening and reinforcing OCD symptomatology (Baldi and Schruers, 2021). Similarly, checking subtypes of OCD were also considered vulnerable owing to an intolerance of uncertainty in relation to restrictions and guidelines (Jassi et al, 2020).

Hence, the findings of Fineberg et al (2021) merit substantiation in a longitudinal study as the temporal variance of these deficits in executive functioning is not well defined as the current evidence base mostly comprises cross-sectional analyses. As outlined in the previous chapter, these neurocognitive features are disparate from other psychiatric phenomena (symptom severity, medication, comorbid disorders etc) and so assessing these deficits in a subclinical sample may yield novel insights into their pre-clinical compositions. If the direction of causality is confirmed, these symptoms and cognitive factors may be used as predictive markers to identify at-risk individuals who may benefit from preventative interventions in future pandemics.

This study therefore employed a longitudinal design to assess the following research questions:

- Is poor adjustment to the easing of (COVID-19) restrictions predicted by a personal or familial history of mental disorders, or obsessive-compulsive symptoms or traits?
- Does cognitive flexibility, as measured by an online set-shifting task, predict post-pandemic adjustment?
- Do self-ratings of mental health measures change over six months post-pandemic?

METHOD

Institutional review board approval

This research was approved by the University of Hertfordshire Health, Science, Engineering and Technology Ethics Committee with Delegated Authority (ECDA) (protocol number: LMS/PGR/UH/04554). It was pre-registered on the Open Science Framework

(https://osf.io/v4c28) and reported according to the Checklist for Reporting Results of Internet E-Surveys (CHERRIES, Eysenbach, 2004; 2012).

Design

As this study was a replication of Fineberg et al (2021), it was imperative that the testing conditions (described below) remained consistent with the original methods; procedure (adjustment group classification), materials (self-reports of clinical variables and a neurocognitive task of cognitive flexibility), and design (mediation paradigm). The study also employed a longitudinal design to assess temporal variances of these clinical constructs, with participants who completed baseline assessments being offered the opportunity to continue in the study with follow-up assessments at 3-months and 6-months after baseline. The survey was hosted on the experiment building software *Gorilla* (Anwyl-Irvine et al, 2020).

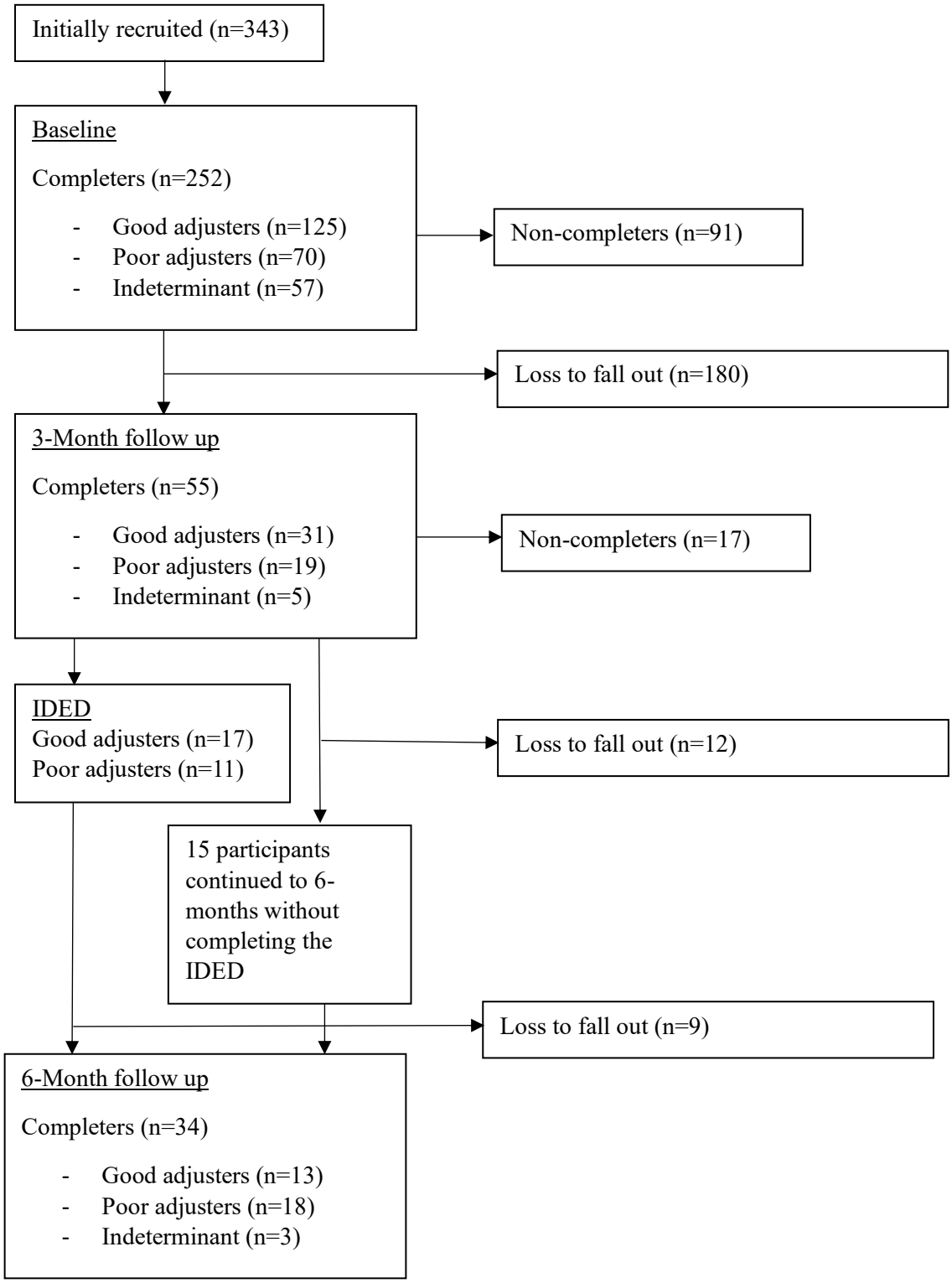
Sample

Three hundred and Forty three members of the UK public were recruited over a 15-month period between May 2021 to April 2022 (pandemic related restrictions in the UK officially ended July 2021). Participants included within this chapter's analyses are referred to as subclinical participants. They are taken to represent a point before symptoms become disabling and chronic that would warrant a formal diagnosis of OCD but which symptoms are still of some disturbance and impairment to daily functioning. Importantly, subclinical participants comprise salient pathogenic markers that may be scrutinised to identify the onset of these functional impairments.

Participants were recruited from social media platforms (Twitter, Facebook, and LinkedIn); survey-sharing sites such as SurveyMonkey and Survey Circle and from the University of

Hertfordshire, at which students were allocated study course credits for completing the survey. This was the only incentive used in the recruitment process. Owing to differing COVID-19 regulations across different countries, the current study focused solely on UK residents aged 18 years and above. Measures were taken to ensure that only those within the UK completed this survey. The exclusivity to UK-citizens was stressed on the survey advertisement with an addition to the demographic screening questions asking if the respondent was a UK-citizen; answering 'no' would end the survey prematurely. Moreover, Gorilla output details the time zone that the survey was completed in, recording the host time zone as '1' and would allocate increments to this value according to the deviation from this point; only those with a time zone value of 1 were included within this analysis. Although other countries reside within the same time zone as the UK, the survey was published only in English. These methods together were employed as quality control features. Two follow-up assessments took place 3 and 6 months after recruitment (October 2021 to July 2022 and March 2022 to October 2022, respectively). See Figure 3.0.1 for participation flow across the study timelines.

Figure 3.0.1. Sample sizes across Baseline, 3-month follow up, and 6-month follow up.



Consent

Participants were directed to an information sheet detailing all aspects of the research via an embedded link within the study advertisement. Informed consent was obtained from participants on an online consent form.

Participants were asked at baseline and at each follow-up stage (i.e. at 3 and 6-months) to provide consent to participate in the subsequent follow-up assessments. Participants were asked to provide their email address so that they could be contacted with the survey information at 3-months and 6-months. Participant email addresses were stored in accordance with BPS Code of Human Research Ethics (2014); this information was kept on a password encrypted OneDrive that was only accessible to the lead researcher (AC).

Measures, variables, and outcomes

Baseline Assessment

At baseline testing, the following tests were administered:

Post-pandemic Adjustment Scale (PPAS: Fineberg et al, 2021).

The PPAS is a self-rated scale comprising seven items on a Likert scale (each item rated from 1-5) assessing post-pandemic adjustment difficulties: ‘Completely disagree’, ‘Disagree’, ‘Neither disagree nor agree’, ‘Agree’, and ‘Completely agree’. The first item (*I am having great difficulty adjusting to the easing of the Covid-19 pandemic restrictions*) measures global adjustment problems (see Appendix J). Consistent with Fineberg et al (2021), an a-priori criterion was used to determine good adjusters if participants scored 1 or 2 on this item

and poor adjusters as those who scored 4 or 5; those scoring a 3 were considered indeterminant. The PPAS shows excellent internal consistency (α = 0.87; ω =.87).

Obsessive-Compulsive Inventory-Revised (OCI-R: Foa et al, 2002).

The OCI-R is a self-rated scale consisting of 18 items, that is commonly used to assess obsessive-compulsive symptom severity and symptom subtypes; Obsessing, Ordering, Checking, Washing, Hoarding, and Neutralizing. The OCI-R has also been validated in both clinical and population-based samples (Huppert et al, 2007). It shows good internal consistency (α=0.84; ω =.93). A total score of 21 or above indicates a strong likelihood of the presence of OCD (Foa et al, 2002).

Compulsive Personality Assessment Scale (CPAS: Fineberg et al, 2015).

The CPAS is an 8-item self-rated (or observer-rated) instrument measuring the severity of the individual traits of DSM-5 obsessive-compulsive personality disorder (APA, 2013); Preoccupation with details, Perfectionism, Workaholism, Over-conscientiousness, Hoarding, Need for control, Miserliness, and Rigidity. The CPAS has been found to differentiate individuals with OCPD both in a university student sample (Fineberg et al, 2015) where it was validated against an objective measure of cognitive inflexibility (IDED task), and among various clinical groups of patients. The CPAS has excellent internal consistency (α = 0.86; ω =.86).

Depression, Anxiety, and Stress Scale (DASS-21: Lovibond & Lovibond 1995).

The DASS-21 consists of 21-items divided across three subscales, each comprising 7 items, for the self-assessment of depression, anxiety, and stress symptoms. It has excellent internal consistency (α = 0.92; ω =.95) and has been validated in both clinical samples and as a self-

assessment tool in population-based research (Henry and Crawford, 2005; Hellberg, Buchholz, and Abramowitz, 2019).

COVID-19 Safety Behaviour Scale (Timpson et al, 2020)

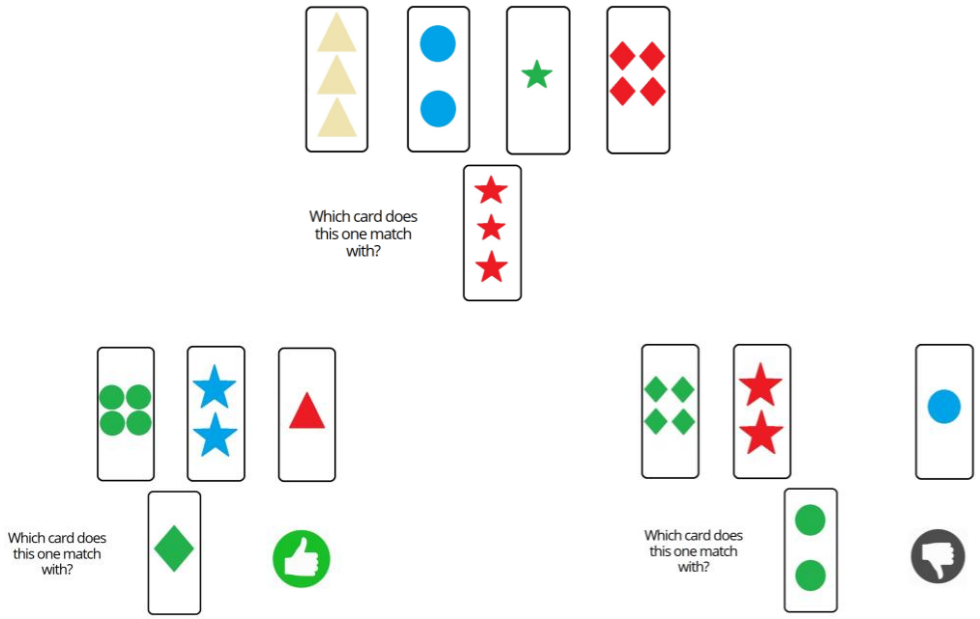
The Covid-19 Safety Behaviour Scale is a 15-item self-rating scale describing the range of safety behaviours used during the COVID-19 lockdown period (see Appendix K), which was created for the purposes of this study, as no similar measure existed. The scale was developed from the first of 10 sections of the UK COVID-19 questionnaire (Timpson et al, 2020), which includes questions relating to safety behaviours that members of the public adopted during the pandemic. Questions were re-worded to explore the current use of each of these safety behaviours and added a 5-point Likert scale (0-4): 'Never', 'Hardly ever', 'Often', 'Most of the time', and 'All of the time' to each item, to quantify each item. Two items were removed from the questionnaire relating to being a student or a parent as not all participants would be able to rate these items. The internal consistency of this scale in this study was good (α = 0.83; ω =.83).

The Wisconsin Card Sorting Test (WCST: Milner, 1963).

This study used a digital adaptation of the WCST – an established cognitive test that measures flexibility in shifting and maintaining attention. As the original study had employed the IDED on a small subset of participants (n=40) from their original sample (n=514), it was imperative to reflect this measurement of cognitive flexibility in this current analysis. However, as both between-group contrasts and within-group correlations were required for this analysis, the IDED was not financially feasible for a predicted sample size of 500 participants. As the WCST was readily available within the Gorilla survey experiment builder, this was a cost-effective alternative. Participants are required to match a given target card to one of four other cards; and to achieve this, participants learn the rule(s) governing

matching through trial and error. The matching rule may depend on shape (matching squares with squares or stars with stars), colour (blue with blue) or number (three shapes matched with three shapes). After 8 trials, the matching rule changes (without warning) requiring participants to adapt their answers and learn the new matching rule and reject the previous one. As shown in Figure 3.0.2, participants will receive visual feedback on the accuracy of their response, which will highlight either a correct response or an incorrect response; the latter requires a change of matching method. The task includes 64 randomised trials: 8 trials involving 8 different rules. The typical outcome metrics of the WCST are: total errors made, perseverative errors (the participant maintains the previous matching rule despite being informed that this is incorrect), failure to maintain set (an error in the matching rule after a run of correct trials, or a return to a previous matching rule), and non-perseverative error (errors that do not conform to the description of perseverative errors or a failure to maintain set). For the purposes of this analysis, the perseverative errors metric was chosen to represent cognitive (in)flexibility as it is the preferred metric in studies employing the WCST due to its close resemblance to this neurocognitive phenotype (see Chapter 2, Table 2.1). Moreover, as the WCST was chosen in place of the IDED as the primary measure of cognitive inflexibility in the baseline condition, it was imperative to utilize perseverative errors is this best reflected Extra-dimensional errors of the IDED for consistency with Fineberg et al (2021).

Figure 3.0.2. Example stimuli of the Wisconsin Card Sorting Test



Correct match response

Incorrect match response

Assessment 3 months after baseline

The first follow-up assessment comprised the same questionnaires as at baseline (Post-Pandemic Adjustment Scale (PPAS), OCI-R, CPAS, DASS-21, and the Covid-19 Safety Behaviour scale) apart from the WCST, which was replaced with the Intradimensional-Extradimensional set-shifting task (IDED) (see below), a subtest of the Cambridge Neuropsychological Test Automated Battery (CANTAB) (https://cambridgecognition.com/intra-extra-dimensional-set-shift-ied/; Sandberg, 2011). The IDED task was used in place of the WCST to avoid learning and practice issues inherent within repeated neurocognitive testing paradigms (Kopp, Lange, and Steinke, 2019); this would render a measurement of procedural memory instead of varying flexibility between assessment points thereby confounding interpretation. Importantly, the IDED was not

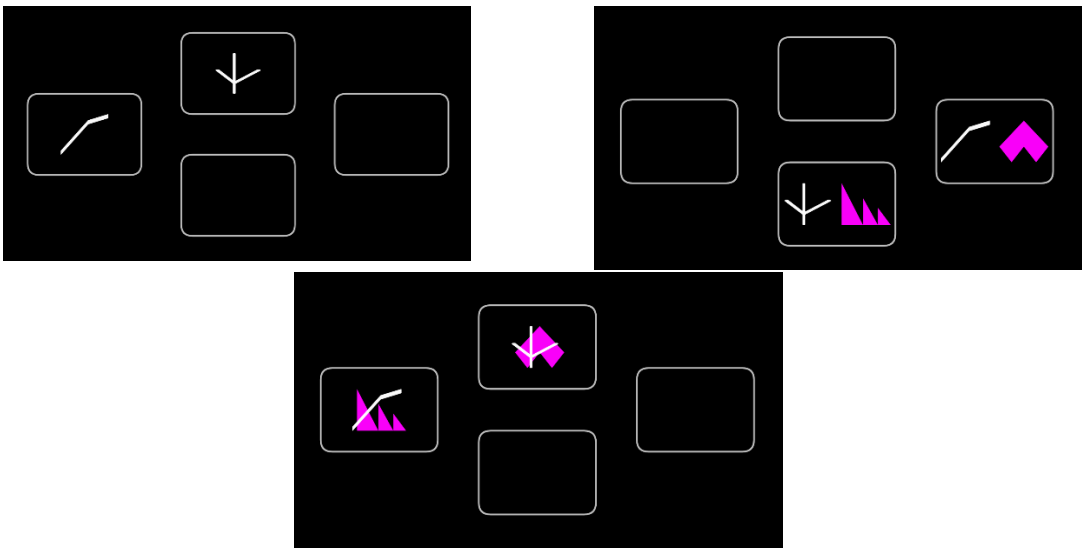
submitted within the pre-registration of the study and was added post-registration; all analyses pertaining to this task are therefore exploratory.

Intra-Extradimensional set-shifting task (IDED; Owen et al, 1991).

Like the WCST, this cognitive task also assesses the ability to flexibly adapt responses in relation to rule acquisition and reversal learning. The IDED may be a more sensitive measure of different forms of attentional inflexibility than WCST as it can identify in which stage of the learning process a deficit lies (Chamberlain et al, 2021). It comprises nine stages where subjects, through trial and error, must select the correct paired image, which consists of two different dimensions of stimulus: white lines and pink shapes (see Figure 3.0.3). After a phase of acclimatization, participants are required to choose the 'correct' shape in the pair. Much like the WCST, the rule changes after several trials forcing the participant to adapt and shift their attention to the acquisition of the new rule. Initial stages of the task reward one of the features of the pair, for instance, a particular shape. Individuals must therefore ignore the lines and attend exclusively to shapes, choosing the correct one. However, the rule (i.e., the correct choice of shape) is reversed once learned. At the intra-dimensional (ID) shift (Stage 6), participants need to adjust their responding to endorse the alternative shape as the correct one and let go of the previously correct response. This element of the task approximates to a test of reversal learning. As the task continues, the participant eventually arrives at the Extra-Dimensional shift (EDS; Stage 8), when the rule changes once more. This time, the dimension changes, so in this example, lines would become the dominant feature, and the participant would need to stop attending to shapes and learn to respond to lines instead, i.e. switching attentional set. The ED shift stage is thus a cardinal measure of attentional flexibility. A final stage, the Extra-dimensional shift reversal (EDSR; Stage 9) tests the ability

to respond to a further reversal, this time of the ED shift. The key outcome metrics of this task are the number of ED errors at each of the 9 stages, total ED errors, total latency (reaction time), and the number of stages completed. Adjusted total scores are also calculated to compensate for missing cases. Extra-dimensional errors at stages 8 and 9 (EDS and EDSR) were the chosen outcome metrics of the IDED in this analysis to express the cognitive (in)flexibility of this subclinical sample. This maintained consistency with Fineberg et al (2021) and the wider literature (as documented in Chapter 2, Table 2.1).

Figure 3.0.3. Example stimuli of the Intra-Extradimensional set-shifting task.



Assessment 6 months after baseline

This assessment phase comprised the same questionnaires used at baseline and at 3-months (PPAS, OCI-R, CPAS, DASS-21, and the Covid-19 Safety Behaviour scale). No neurocognitive task was used in this final assessment.

Statistical analysis

A statistical power analysis was used to estimate the sample size needed to address the main hypotheses. G-power software estimated a sample size of 376 participants (type 1 error rate of .01, a desired moderate effect size (Cohen's *d*) of .40, and desired statistical power at .90). Based on a 30% participant drop-rate from Fineberg et al (2021) between initial testing and follow-up, recruitment of a larger sample (n=500) was planned to accommodate attrition and incomplete datasets.

All analyses were conducted using SPSS v 28 (IBM Corp, 2021) and JASP 0.16.3 (JASP team, 2022) after all three data collections had been completed. Participant email addresses were collected and stored for the purpose of matching participant data from across baseline, 3-month, and 6-month testing conditions as well as from CANTAB. A number of JASP modules were used in this analysis; Descriptives, Frequencies, T-tests, ANOVA, and PROCESS. Differences in sociodemographic categorical variables (age, gender, ethnic group, educational level, occupation, contraction of COVID-19 and resultant bereavements, and personal and familial history of mental disorders) were assessed using Chi square; Phi (φ) was used to detail effect sizes.

Confirmatory mediation analysis was used to assess indirect effects (Baron and Kenny, 1986; Hayes, 2017). As per Fineberg et al (2021), predictor variables were OCI-R, CPAS, and mental health history; mediators were DASS-21 (M^1) and COVID-19 Safety Behaviour Scale (M^2); and the outcome variable was post-pandemic adjustment difficulty. A correlational matrix was used to test collinearity and Durbin-Watson values were consulted to detail independence of errors (1.88). Homoscedasticity was also assessed by observing standardised residuals on a Q-Q plot (see Appendix L) ; no patterns were revealed in the spread of residuals suggesting constant variance (Yang, Tu, and Chen, 2019). Minimal multicollinearity

was revealed as variance inflation factors for the predictor variables in this model resides within acceptable limits (Tabachnick and Fidell, 2001).

As reported in Fineberg et al (2021), the first item of the bespoke Post-Pandemic Adjustment Scale '*I am having great difficulty adjusting to the easing of the COVID-19 pandemic restrictions'* was used to separate participants into subgroups based on adjustment difficulty. Student's t test was used to compare good adjusters to poor adjusters on measures of post-pandemic adjustment difficulty (post-pandemic adjustment scale) obsessive-compulsive symptoms (OCI-R), compulsive traits (CPAS), depression anxiety, and stress symptoms (DASS-21), and safety behaviours (Covid-19 Safety Behaviour Scale).

As the neurocognitive data for both the WCST and IDED was non-normally distributed, Mann-Whitney U was used to assess differing profiles of cognitive flexibility between the two groups: mean scores of good and poor adjusters were compared on the outcome measures of the WCST at baseline and IDED at three months. Rank-Biserial Correlations (*rrb)* were used to detail effect sizes for non-parametric analyses.

Repeated measures ANOVAs were used to assess longitudinal trends in clinical variables. Continuous variables (adjustment difficulties, obsessive-compulsive symptoms and traits, symptoms of depression, anxiety, and stress and COVID-19 safety behaviours) were examined over time by comparing scores from baseline, three-month, and six-month (within-subject factor). Between-group contrasts (good vs poor adjusters) were also assessed at each of the three levels of the within-subject factor. Data was normally distributed, and Mauchley's test of Sphericity was also met (Armstrong, 2017). Pairwise comparisons were used to identify discrepancies between the time points and Partial eta squared (η_p^2) was used to detail effect sizes of significant main and interaction effects.

RESULTS

Baseline Assessment

Of the 343 participants recruited, 252 completed the baseline assessment (see Table 3.0.1 for demographic details). Age, gender identity, and ethnicity did not differ between baseline completers and non-completers. At 3-month follow-up, 55 participants (22%) completed the questionnaire assessment, of whom 28 (51%) also completed the IDED and 34 (62% of the 3-month sample) went on to complete the 6-month follow-up assessment.

Table 3.0.1. Participant demographic information (n=252).

Gender	**%**				
Male	28.6				
Female	69.8				
Non-binary	1.6				
Age range	**%**	**Ethnicity**	**%**	**Level of Education**	**%**
18-27	60.3	White	59.5	GCSE's	0.8
28-37	14.7	Black	8.7	A-Levels	18.7
38-47	10.7	Mixed	6.7	Undergraduate	57.9
48-57	9.1	Asian	24.2	Postgraduate/ MSc	14.3
58-67	4.4	Other	0.8	Doctorate/ PhD	8.3
68+	0.8				
Living status	**%**	**Occupation**	**%**		
Own property	10.3	Employed	31.3		
Mortgage	13.5	Unemployed	4.8		
Renting	26.2	Health workers	12.7		
Living with parents	33.3	Students	50.8		
University dorms	15.1				
Other	1.6				

At baseline, most respondents were female (69.8%) aged 18-27 (60.3%), white (59.5%) and educated to at least undergraduate level (57.9%), which is comparable with the sample demographics of Fineberg et al (2021). Over two thirds (68.7%) of the sample were unemployed during the recruitment procedure. Of the proportion that were employed, 12.7%

were frontline workers (such as health workers and carers whose vocations required close working with those potentially exposed to the virus).

One hundred and fifty-six participants (62%) said that they or a close family member had contracted COVID-19. Forty-three participants (17%) reported a COVID-19 related death of someone close to them (see Appendix M); one hundred and eight participants (42.8%) reported a personal history of mental disorder and ninety-four (37.3%) reported some family history of mental disorder. Table 3.0.2 details self-report scores of clinical variables as well as neurocognitive performances.

Table 3.0.2. Baseline descriptives of clinical measures and WCST outcome metrics

	Mean	SD
Post-Pandemic Adjustment Scale (PPAS)	20.38	8.35
Obsessive-Compulsive Inventory-Revised (OCI-R)	25.69	15.67
Compulsive Personality Assessment Scale (CPAS)	11.12	6.42
Depression, Anxiety, and Stress Scale-21 (DASS-21)	22.00	14.23
COVID-19 Safety Behaviour Scale (C-19 SBS)	30.49	12.02
WCST perseverative errors	13.40	6.60
WCST failure to maintain set	2.98	2.58
WCST non-perseverative errors	9.19	4.88
WCST total errors	25.26	9.48

Note: WCST=Wisconsin Card Sorting Task.

The OCI-R screening threshold for possible OCD (a score of ≥ 21) was exceeded by 151 (59.9%) participants, of whom 78 (51.66%) reported a personal history of mental health disorders, suggesting that half of those screening positive for OCD had not been diagnosed as being mentally unwell. Nevertheless, there was a significant association between a personal history of mental health disorders and scoring above the clinical threshold for OCD (χ^2=11.91, φ =-.22, p<.001), suggesting that OCD contributed significantly to mental health problems in this sample. Importantly, those with a personal history of mental health disorder scored significantly higher on the OCI-R (31.82; SD=15.61) compared to those without

(21.10; SD=14.11); $t(250) = 5.71$, $p<.001$, $d=0.72$. Moreover, the mean OCI-R scores reported here (25.69; SD=15.67) was also considerably lower than that reported in Fineberg et al (35.50; SD=11.70). The same can be observed in DASS-21 scores recorded here (22.20; SD =14.23) compared to the previous analysis (32.20; SD =15.70), suggesting the current sample experienced less obsessive-compulsive, depression, anxiety and stress symptoms (see Table 3.0.1).

Correlation analyses

Table 3.0.3 shows that all clinical ratings were significantly intercorrelated. As can be seen, OCI-R scores strongly correlated with CPAS and DASS-21 scores. Crucially, PPAS scores positively and moderately correlated with all clinical measures i.e., OCI-R, CPAS, DASS-21, and the COVID-19 safety behaviour scale (all $p<.01$).

Table 3.0.3. Bivariate correlational data

	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*
(1) PPAS	--							
(2) OCI-R	.45**	--						
(3) CPAS	.39**	.70**	--					
(4) DASS-21	.40**	.63**	.66**	--				
(5) C-19 SBS	.46**	.31**	.19**	.14*	--			
(6) WCST perseverative errors	.08	.19**	.16*	.12	.05	---		
(7) WCST failure to maintain set	.07	.10	.16*	.15*	-.07	-.54**	.--	
(8) WCST non-perseverative errors	.00	-.02	.07	.04	-.04	-.63**	.05	.--
(9) WCST total errors	.08	.17**	.19**	.15*	-.01	-.99**	.74**	.55**

*Note: * Significant at the p<.05 level, **significant at the p<.01 level.*

The WCST outcome measures showed a varying degree of correlation with clinical measures, but no significant correlation with the PPAS. As expected, WCST perseverative errors were highly significantly positively correlated with OCI-R and CPAS scores (both $p<.01$) indicating a relationship with the presence of compulsive symptoms and traits and significantly correlated with DASS-21 ($p<.05$) scores indicating an additional relationship with depression anxiety and stress (see Table 3.0.3).

Mediation Analysis

Replicating the model described by Fineberg et al (2021), the OCI-R, CPAS, and personal history of mental health disorders were designated as predictor variables; and DASS-21 and Covid-19 safety behaviour scale scores were designated as mediator variables, with PPAS as the outcome variable.

This mediation model (Figure 3.0.4) accounted for 35% of the variance observed in PPAS (adjustment) scores. Where Fineberg et al (2021) showed that no predictor variables had a direct effect on adjustment difficulty, this model showed that OCI-R scores did have a direct effect on adjustment difficulties (See Table 3.0.4).

Table 3.0.4. Mediation analysis of PPAS scores (n=252).

				95%CI				
Type	Effect	Estimate	SE	Lower	Upper	β	Z	p
Indirect	**OCI-R ⇒ DASS-21 ⇒ PPAS**	**0.02**	**0.01**	**0.00**	**0.04**	**.05**	**2.08**	**.04**
	OCI-R ⇒ COVID-19SBM ⇒ PPAS	**0.04**	**0.01**	**0.03**	**0.06**	**.09**	**2.80**	**.01**
	CPAS ⇒ DASS-21 ⇒ PPAS	**0.07**	**0.03**	**0.01**	**0.13**	**.07**	**2.18**	**.03**
	CPAS ⇒ COVID-19SBM ⇒ PPAS	0.01	0.03	-0.05	0.07	.01	0.32	.75
	PH-MHD⇒ DASS-21 ⇒ PPAS	**-0.53**	**0.25**	**-1.02**	**-0.03**	**-.08**	**-2.10**	**.02**
	PH-MHD⇒COVID-19SBM ⇒PPAS	0.52	0.30	-0.07	1.12	.08	1.73	.08
Component	**OCI-R ⇒ DASS-21**	**0.26**	**0.06**	**0.15**	**0.37**	**.29**	**4.73**	**<.001**
	DASS-21 ⇒ PPAS	**0.08**	**0.04**	**0.01**	**0.15**	**.18**	**2.31**	**.02**
	OCI-R ⇒ COVID-19SBM	**0.21**	**0.07**	**0.08**	**0.33**	**.27**	**3.13**	**.01**
	COVID-19SBM ⇒ PPAS	**0.18**	**0.03**	**0.13**	**0.24**	**.34**	**6.30**	**<.001**
	CPAS ⇒ DASS-21	**0.86**	**0.13**	**0.60**	**1.12**	**.39**	**6.48**	**<.001**
	CPAS ⇒ COVID-19SBM	0.05	0.16	-0.26	0.37	.03	0.32	.75
	PH-MHD⇒ DASS-21	**-6.51**	**1.32**	**-9.09**	**-3.94**	**-.46**	**-4.95**	**<.001**
	PH-MHD⇒ COVID-19SBM	2.85	1.58	-0.25	5.94	.24	1.80	.07

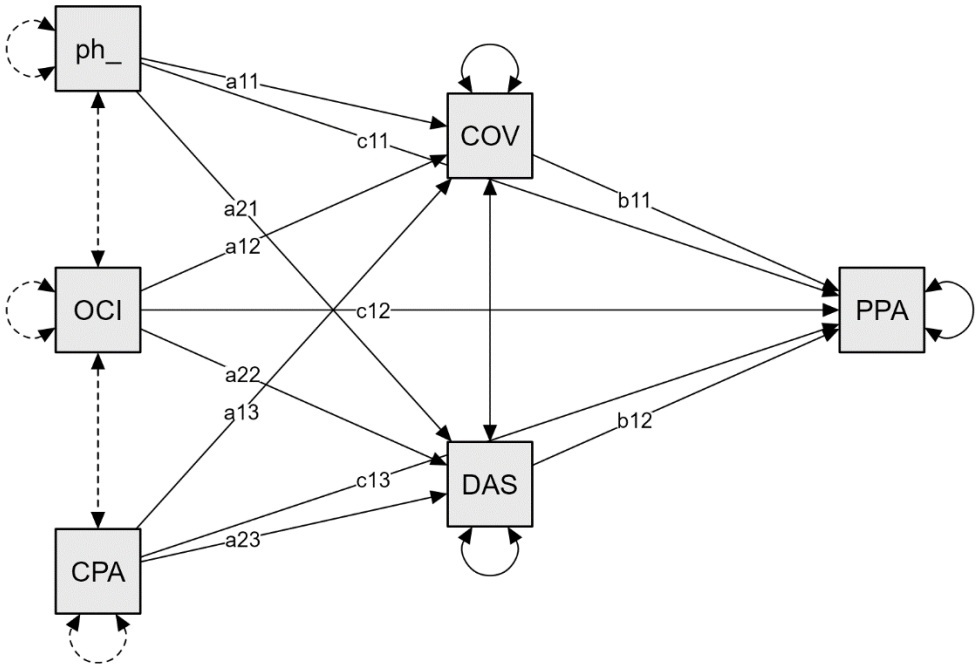
Direct	**OCI-R ⇒ PPAS**	**0.08**	**0.03**	**0.02**	**0.15**	**.20**	**2.63**	**.01**
	CPAS ⇒ PPAS	0.06	0.08	-0.10	0.22	.06	0.78	.45
	PH-MHD ⇒ PPAS	-0.39	0.77	-1.89	1.12	-.06	-0.50	.61
Total	**OCI-R ⇒ PPAS**	**0.14**	**0.03**	**0.08**	**0.21**	**.34**	**4.36**	**<.001**
	CPAS ⇒ PPAS	0.14	0.08	-0.02	0.30	.14	1.74	.08
	PH-MHD ⇒ PPAS	-0.39	0.79	-1.94	1.16	-.06	-0.50	.62

Note: Confidence intervals computed with method: Standard (Delta method)
Betas are completely standardised effect sizes. Significant findings are in bold

Moreover, CPAS scores had an indirect effect on adjustment difficulty via DASS-21 scores (Z=2.18, p<.05), as was also the case for a personal history of mental health disorders (Z=-2.10, p<.05), whereas OCI-R predicted changes in adjustment via DASS-21 (Z=2.08, p<.05) and COVID-19 safety behaviour scale scores (Z=2.80, p<.001), the latter being the stronger mediator of the relationship between OCI-R scores and PPAS scores.

Given the number of analyses conducted, a Bonferroni correction was employed to control for familywise error (Armstrong, 2014); the corrected alpha is now considered at the 0.0125 level. With regard to this mediation model, the indirect mediation pathways via the DASS-21 are to be considered with caution.

Figure 3.0.4. Mediation model used to predict post-pandemic adjustment (after Fineberg et al, 2021).



Note. Ph=Personal history of mental health disorders; OCI=Obsessive-Compulsive Inventory-Revised; CPA=Compulsive Personality Assessment Scale; COV=COVID-19 Safety Behaviour Scale; DAS=Depression Anxiety Stress Scale-21; PPA = Post Pandemic Adjustment Scores.

Comparison of extreme groups

Participants were categorised as good or poor adjusters based on their Post-Pandemic Adjustment Scale (PPAS) item 1 score ('I am having great difficulty adjusting to the easing of the COVID-19 pandemic restrictions'). Consistent with Fineberg et al (2021), an a-priori criterion was used to determine good adjusters if participants scored 1 or 2 on this item and poor adjusters as those who scored 4 or 5. According to the *a priori* criterion established by response to Question 1 of the PPAS, 70/252 participants (27.78%) were identified as 'poor adjusters' using, 125/252 (49.60%) were identified as 'good adjusters'; 57/252 (22.10%)

were considered as indeterminate responders and the latter were excluded from subsequent analyses.

Good and poor adjusters were contrasted on demographic variables. No significant differences emerged for: age (χ^2=4.37, p=.49), sex (χ^2=5.22, p=.07), ethnicity (χ^2=1.93, p=.75), education (χ^2=5.78, p=.22), or occupation (χ^2=1.92, p=.59), contraction of COVID-19 (χ^2=4.95, p=.08), or COVID-19 related bereavements (χ^2=.04, p=.84). Compared to good adjusters, however, poor adjusters reported a higher level of personal (χ^2=8.37, φ=-.21, p<.05) and familial (χ^2=4.55, φ=-.15, p<.05) histories of mental health disorders.

Table 3.0.5 shows that poor adjusters showed significantly higher levels of OCD symptomatology (OCI-R), compulsive traits (CPAS), depression, anxiety and stress (DASS-21), and Covid safety behaviours (COVID-19 SBS) (all p<.001).

Table 3.0.5. Self-rating questionnaire responses for good (n=125) and poor adjusters (n=70).

	Good adjusters Mean (SD)	Poor adjusters Mean (SD)	*t*-value and effect size (*d*)
OCI-R	22.56 (15.17)	30.37 (15.74)	$t_{(193)}$ = -3.40*, d =0.51
CPAS	9.35 (5.92)	12.77 (6.74)	$t_{(193)}$ = -3.68*, d =0.55
DASS-21	18.07 (13.23)	28.87 (14.62)	$t_{(193)}$ = -5.26*, d =0.79
COVID-19 SBS	26.15 (11.82)	36.00 (10.68)	$t_{(193)}$ = -3.16*, d =0.47

*Note. * p<.001*

WCST Performance – Good Adjusters Versus Poor Adjusters

On the WCST, the good and poor adjuster groups showed no statistically significant difference in perseverative errors (U=4187.00, p=.62, rrb=-0.04), failures to maintain set (U=4373.00, p=1.00, rrb=-0.00), non-perseverative errors (U=4589.00, p=.57, rrb=0.05), or

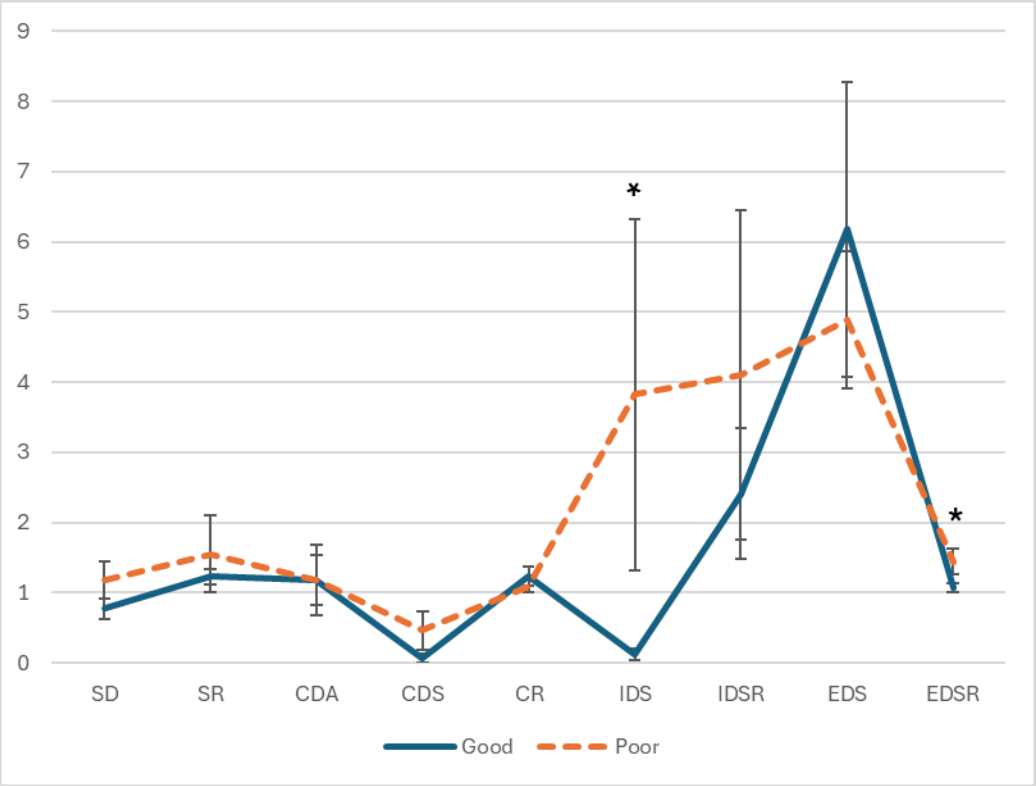
total errors (*U*=4486.50, *p*=.77, *rrb*=0.03); WCST metrics were additionally not correlated with PPAS scores (*p*>.05).

Exploratory analysis of the IDED at the 3-month follow-up

Of the 31 participants that completed the IDED at 3-month testing, 17 were identified as *good adjusters* and 11 as *poor adjusters*; the remaining 3 were identified as *indeterminant* on the PPAS. As shown in Figure 3.0.5, poor adjusters made significantly more errors than good adjusters at stage 6 (IDS) (*U*=60.00, *p*<.05, *rrb*=-0.36) and stage 9 (EDSR) (*U*=42.00, *p*<.05, *rrb*=-0.38) of the IDED. Given the reduced adjustment group sizes between baseline and 3-month follow up, these analyses are not sufficiently powered to detect true effects (.55), so caution is warranted with interpretation.

Moreover, for the whole sample, IDS errors significantly correlated with PPAS scores (r=.38, p=.035). No other differences on IDED test metrics between adjustment groups reached significance, nor correlated with PPAS scores.

Figure 3.0.5. Mean errors for good (n=17) and poor (n=11) adjusters on each stage of the IDED task.



Note. Error bars represent standard error
SD=Simple discrimination; SR = Simple reversal; CDA = Compound Discrimination Adjacent; CDS = Compound Discrimination Superimposed; CR = Compound Reversal; IDS = Intradimensional Shift; IDSR = Intradimensional Shift Reversal; EDS = Extradimensional Shift; EDSR = Extradimensional Shift Reversal

Exploratory correlational analyses of OCD symptoms and neurocognitive performance

The OCI-R was separated into its subscales of distinct symptom dimensions; hoarding, checking, ordering, neutralising, washing, and obsessing (Foa et al, 2002; Abramovitch et al, 2020. Both hoarding and checking symptoms shared weak correlations with non-perseverative and total errors on the WCST at baseline (See Appendix N). No other symptom types correlated with outcome metrics of the WCST (all >.05). At three-months, a weak-moderate correlation was revealed between washing symptoms and the first stage (Simple discrimination of the IDED (r=-.39, p<.05). No other significant correlations were revealed

between OCI-R symptom dimensions and IDED outcome metrics at three months (see Appendix O).

Longitudinal data

At the 6-month end point, a total of 34 participants had completed all three assessment time-points (13 good adjusters and 18 poor adjusters; the remaining 3 participants were identified as indeterminant). Table 3.0.6 compares the mean scores on all clinical questionnaires for good and poor adjusters at each of the three assessment time-points.

Table 3.0.6. Mean questionnaire scores for good (n=13) and poor (n=18) adjusters across baseline, 3-months and 6 months.

Measure	Time	Good Adjusters (n=13) Mean (SD)	Poor Adjusters (n=18) Mean (SD)	Main effect (Time)	Main effect (Group)	Interaction effect
PPAS						
	Baseline	13.23 (5.67)	24.89 (6.82)	**p < .001**	**p < .001**	n.s.
	3-month	13.46 (5.35)	22.28 (7.05)			
	6-month	11.85 (7.34)	19.11 (7.46)			
OCI-R						
	Baseline	26.08 (15.65)	26.11 (15.39)	**p < .001**	n.s.	n.s.
	3-month	16.46 (13.41)	17.67 (12.49)			
	6-month	17.15 (15.17)	17.50 (13.02)			
CPAS						
	Baseline	11.00 (7.59)	11.06 (6.78)	n.s.	n.s.	n.s.
	3-month	11.54 (6.16)	11.00 (6.44)			
	6-month	10.46 (8.16)	10.33 (6.28)			
DASS-21						
	Baseline	15.38 (8.58)	29.44 (16.77)	n.s.	**p < .05**	n.s.
	3-month	15.53 (8.58)	25.89 (13.93)			
	6-month	13.69 (10.53)	25.17 (13.81)			
C-19 SBS						
	Baseline	26.15 (11.82)	36.00 (10.68)	**p < .001**	**p < .05**	n.s.
	3-month	22.46 (11.16)	32.71 (14.77)			
	6-month	11.77 (7.43)	21.76 (9.65)			

Repeated measures ANOVAs were conducted to assess differences between the two adjustment groups (between groups variance; a main effect of group) across the three time

points (within-group variances; a main effect of time). Analyses revealed a main effect of time for COVID-19 safety behaviour scores (η_p^2 = .56) and a main effect of group, with poor adjusters scoring significantly higher than good adjusters on the range of safety behaviours used during the pandemic (η_p^2 = .18). A main effect of time was also revealed for OCI-R scores which highlighted a depreciation in scores over time (η_p^2 = .43). Further, a main effect of group was revealed for DASS-21 scores (η_p^2 = .20); good adjusters scored significantly lower than poor adjusters. No significant main effects of group or time were revealed for CPAS scores which indicated that there were no marked differences between good and poor adjusters and also there were no differences in scores between assessment points. No significant group-by-time interactions emerged on any variables, indicating no particular trend of good or poor adjusters showing a marked depreciation in scores over time.

DISCUSSION

Is poor adjustment to the easing of (COVID-19) restrictions predicted by obsessive-compulsive symptoms, traits, or a personal or familial history of mental disorders?

The current study offers a longitudinal assessment of psychological and neurocognitive factors influencing post-pandemic adjustment, extending previous work by Fineberg et al (2021). Key findings here replicated the mediation model from the initial lockdown period, revealing that depression, anxiety, and stress mediated adjustment difficulties. Furthermore, the current analyses of this later Covid-19 period now shows that OCI-R scores also predicted adjustment directly as well as indirectly through affective symptoms. This suggests that OC symptoms, which may act as vulnerability factors, and the psychological distress they provoke drive adjustment problems. The finding of a direct relationship between obsessive-compulsive symptoms and post-pandemic adjustment differs from the model arising from the

original paper. It suggests that the ongoing nature of the pandemic may have strengthened the impact of obsessive-compulsive symptoms on adjustment over time within the general population.

Following the initial national lockdown, studies have reported increased levels of OC symptoms in the general population (Grant et al, 2022). Indeed, the previous analysis (Fineberg et al, 2021) in a large general population sample (n=514), during the easing of the first UK lockdown (June 2020) showed that 21% of that sample with no previous history of mental health disorders scored above the OCI-R clinical threshold score of 21 (Foa et al., 2002), while 31% of those reporting a history of mental health disorders also scored above this point. In the current study, 60% of this sample showed OCI-R scores above the clinical threshold for OCD symptoms at baseline, even though scores were obtained sometime after the initial lockdown (May 2021). Interestingly, 51% of those with no history of mental health disorders (n=144) scored above the clinical threshold for OCD which is greater than that recorded in Fineberg et al's (2021) analysis. Nonetheless, the current study shows that mean OCI-R scores decreased across time both in good and in poor adjusters, which might be consistent with a recent meta-analysis of longitudinal data (Prati and Mancini, 2021) which suggested that mental health impacts of the lockdown may be transient.

By contrast, CPAS scores did not differ between good and poor adjusters and did not change across time implying stability on this measure of trait compulsivity. As noted above, this contrasts with the decrease in OCI-R scores, which is, of course, a measure of *state* OC symptoms. Taken together, these findings are consistent with an interpretation suggesting that the pandemic increased state (symptoms) rather than trait mental health factors. Additionally, however, Pereira et al (2022) showed that under the pandemic those with perfectionist traits

were significantly affected and experienced a heightened degree of psychological distress, possibly related to ambiguity pervading all aspects of daily life.

The one area where symptom scores remained consistently higher for poor compared to good adjusters throughout the full 6-month testing period, concerns the DASS-21. At final testing, DASS-21 scores remained high in poor adjusters, being almost double those for good adjusters. While it is unclear if high DASS-21 scores in poor adjusters predated the start of the study, they did remain consistently high throughout the testing period. Meta-analytical estimates of prevalence rates of population samples during COVID-19 indicate inflation for anxiety, depression and stress - at 31.9%, 33.7% and 29.6% respectively (see Salari et al 2020; see also Necho et al, 2021). Both Necho et al (2021) and Salari et al (2020) acknowledged that few studies have followed-up on assessments of these variables. The current study provides longitudinal evidence that concurs with persistently high rates of depression, anxiety and stress in a general population sample during and after the COVID-19 pandemic. This persistence of psychological distress suggests that, for some, the end of lockdown did not bring immediate relief, but rather prolonged emotional strain. While OCI-R scores did decrease across the duration of the study, the consistently elevated DASS-21 scores in poor adjusters appears to highlight that emotional distress may present the greatest barrier to post-pandemic recovery.

A critical finding was that depression, anxiety, and stress, as measured by the DASS-21, consistently emerged as consistent mediators of adjustment difficulties. OCI-R scores were both directly and indirectly associated with poorer adjustment through both DASS-21 and COVID-19 safety behaviours, while CPAS scores and personal histories of mental health disorders influenced adjustment via DASS-21 scores only. These results underscore the central role of obsessive-compulsive and affective symptoms in adjustment, with obsessive-

compulsive symptoms additionally contributing indirectly through psychological distress. Importantly, there was no direct relationship between personal mental health histories and adjustment difficulties, reinforcing the idea that these factors set the stage for distress rather than drive adjustment problems independently (D'Urso et al, 2023).

Does cognitive inflexibility, as measured by cognitive tasks, predict ability to adjust?

Cognitive inflexibility was examined using both the WCST at baseline and the IDED at the 3-month follow-up. WCST results showed no significant differences between good and poor adjusters, nor did WCST scores correlate with PPAS adjustment outcomes. However, exploratory IDED analyses revealed that poor adjusters made significantly more errors at the intra-dimensional shift (IDS) and extra-dimensional shift reversal (EDSR) stages, suggesting deficits in both set-shifting and reversal learning. This suggests that the participants that had a greater difficulty in adjusting to the release of the COVID-19 restrictions showed a definite deficit in both set-shifting and reversal learning capacities. This indicator of an inflexible thinking style may account for some of the difficulties shown by individuals during this period. Indeed, research has recently shown that an inflexible thinking style is also associated with COVID-19 vaccine hesitancy (Pellegrini et al, 2024).

The current findings contrast somewhat with the previous finding that poor adjusters showed significantly poorer performance on the final 9th stage of the IDED (see Fineberg et al, 2021) in that poor adjusters also showed errors on stage 6 (intradimensional shift) in addition to errors on stage 9. This may speak towards some difference between the two epochs of assessment. The earlier analysis was conducted after the initial lockdown release, whereas this current analysis was conducted after repeated lockdowns and releases had occurred. Differences across time and studies in the specific forms of inflexibility shown by poor

adjusters may reflect the impact of fluctuations in restriction rules on adjustment. For example, by having to repeatedly learn and unlearn socially avoidant behaviours (Brown et al, 2021), resulting in greater uncertainty and ambiguity about the correct course of action, those in the current study with a broader profile of cognitive inflexibility may have experienced adjustment problems.

The IDED and WCST tasks are generally thought to be comparable tasks (see Clarke et al, 2024). Nevertheless, although the WCST revealed no differences between good and poor adjusters in a large sample, poor adjusters made significantly more IDS and EDSR stage errors than good adjusters in a smaller sample and performance deficits at both stages showed large effect sizes. One possible explanation is that unlike the IDED, the WCST may be less sensitive to inflexibility that reflects reversal learning. Indeed, Monni et al (2022) showed that when compared to a reversal learning task paradigm, the WCST detects a different aspect of cognitive inflexibility. It seems then that the post-pandemic adjustment problems witnessed in the current study may be more linked to reversal learning than simple attentional set-shifting which is consistent with previous research denoting a multidimensional composition within cognitive flexibility (Guner and Pittenger, 2017). Taken as a whole, these findings suggest that aspects of cognitive inflexibility represent objective population-based markers (biomarkers) indicating vulnerability to adjustment difficulties. As trait markers, they may be of value for identifying at-risk individuals before they experience adjustment problems, as a basis for preventative intervention.

Deficient reversal learning performance is implicated in OCD as well as depression (Remijnse et al, 2006; Remijnse et al, 2009, Apergis-Schoute, and Ip, 2020). Indeed, Tezcan, Tumkaya, and Bora (2017) showed that, whilst deficient reversal learning may be an inherent trait in OCD, state variables can still influence and contribute to this deficit which may have

contributed to the variance seen in this analysis. Variables such as state obsessive-compulsive symptoms appear to represent consequences of the restrictions, whereas variables such as OC traits and affective symptoms (CPAS and DASS-21 scores) are more fixed and stable. These interactions may have resulted in a reduced capacity for behavioural adjustment under uncertain and ambiguous circumstances (Apergis-Schoute, and Ip, 2020, Apergis-Schoute et al 2024).

The identification of obsessive-compulsive symptoms as predictors of adjustment suggest measures should be taken during a pandemic and in its immediate aftermath to proactively identify those struggling with these symptoms to lessen their impact. Identification of depression, anxiety and stress as mediators of adjustment suggests that interventions targeting these symptoms could help alleviate broader adjustment difficulties. It is well known that people with obsessive-compulsive disorders tend to present late for treatment, and the duration of untreated illness adversely affects clinical outcomes (Fineberg et al 2019: Pellegrini et al, 2025). In this study, many individuals endorsed significant obsessive-compulsive symptomatology, but around 50% of them did not consider themselves to have a mental disorder. Psychoeducation and even the use of brief, easy-to-use screeners (Fineberg and Roberts, 2001; Kühne, Paunov, and Weck, 2021, 2022), may be helpful at a population-level to support timely recognition and intervention with treatments tailored to symptom-severity and functional disability (NICE, 2005).

Limitations

One limitation of this research is the level of attrition. As per the pre-registered power analysis, the recruitment goal was 500 participants but 343 were recruited. Of the original 343 participants, 252 (73.47%) completed the baseline assessment, 59 (17.20%) completed

the 3-month follow-up and 34 completed all three time-points. High attrition rates reduced the sample sizes for follow-ups, limiting the generalisability of these longitudinal findings (Gustavson et al, 2012). Notably, poorer cognitive flexibility (as measured by WCST errors) was associated with baseline dropouts, suggesting that the follow-up sample may underestimate the full extent of cognitive inflexibility in poor adjusters (see Appendix P). There was however a comparative depreciation of good and poor adjusters: 75.20% reduction in good adjusters between baseline and 3-months, compared to a reduction of 72.86% in poor adjusters. Nonetheless, between 3-months to 6-months, a disproportionate loss of poor adjusters emerged: 73.68% reduction in poor adjusters compared to only 22.58% in good adjusters.

As described, 34 participants who completed the baseline condition were followed up at 3-months and at 6-months based on their answer to item 1 of the PPAS; of the original sample of 343 participants, 20.41% (n=70) self-identified as being poor adjusters (see method section for details). Additional analyses were performed to observe any potential shift from the poor to the good adjustment group and vice versa. In the final corpus of 34 participants, three poor adjusters moved into the indeterminant category (scoring 3 on item 1 of the PPAS); however, the remaining 10 shifted into the good adjuster category. Another 3 participants who were originally situated in the indeterminant group moved into the good adjuster category. This relative improvement in the ability to successfully adjust may account for the disproportionate loss of poor adjusters; i.e., this may not reflect attrition but may instead reflect a possible metric of adjustment. Regardless, PPAS scores remained stable across the three assessment time-points (see Table 3.5). It may be that these participants are slowly acclimatising to their poor adjustment style, in that they have habituated to the difficulty they experience, and as such appraise their adjustment ability as good; and so, such participants may overestimate how well they adjust (Norman, 2020). Additionally, while IDED results

highlighted key cognitive differences, these analyses were exploratory and warrant cautious interpretation.

As outlined in chapter 1, there are various limitations to using self-reports (self-report bias, social desirability, and similar response styles) which may have influenced the interpretations in this analysis, namely the mediation model. As participants rated multiple clinical constructs, the observed covariance between these constructs and their predictive quality may have been subject to common method variance; as the constructs are measured by the same response method then this may have inflated the observed relationships between these variables with the mediation model as opposed to being a true reflection of those relationships (Kock, Berbekova, and Assaf, 2021). Statistical inflation (probability of a type 1 error) may have also resulted from repeated statistical testing. A Bonferroni correction was applied; a stricter lower alpha level of .0125 (original alpha/ no. of statistical analyses), revealed that any *p* value greater than this was at risk of family-wise error (Armstrong, 2014). Notably, the indirect mediation pathways via DASS-21 scores are to be considered with caution.

As is widely documented, self-report and online surveys are often effected by situational mood states by biasing current experiences and memory retrieval (Burke, Brief, and George, 1993; Costa et al, 2005). To account for this, DASS-21 scores (symptoms of depression, anxiety, and stress) were partialled out of correlational analyses. Controlling for depression, anxiety, and stress (DASS-21) scores revealed that OCI-R and CPAS scores remained correlated with PPAS suggesting that situational mood sates at the time of examination did not affect scores thereby strengthening this relationship between obsessive-compulsive symptoms (r=.28, p<.001) and traits (r=.18, p<.05) with pandemic adjustment difficulties.

Future directions for public health policy

Recent papers show that certain susceptibilities and predispositions (pre-pandemic stress, disgust proneness, and insecure attachments) have predicted adversities during the pandemic such as anxiety responses, emotional difficulties, and suicidality (Cox et al, 2020; Arikan, 2021; Miller et al, 2021; Lewis et al, 2022). The following diathesis-stress model is described to accentuate the relationships between the discussed variables. Trait compulsivity (as measured by the CPAS and the IDED) may represent a vulnerability marker for those with a personal history of mental health disorders, seeing as there is little change in CPAS scores (over time or between groups) and a marked deficit in cognitive flexibility in poor adjusters. This vulnerability is then perpetuated by risk factors of depression, anxiety, and stress symptoms given that DASS-21 scores mediate all three relations between predictors and PPAS scores (see Figure 3.3). The interaction of such variables appears to result in the development of a putative syndrome of post-pandemic adjustment difficulties.

Indeed, there is a great need for public health reformations regarding mental health (Moreno et al, 2020; Hunter, Littlejohns, and Weale, 2022; Lloyd et al, 2023) as this domain of public health was deprived of the fortifications afforded to other domains such as the elderly and those considered high-risk due to pre-existing physical health concerns (Future Care Capital, 2024). An estimated 20% of the UK population now require new or additional support with their mental health (Care quality commission, 2022). As such, the continued assessment of risk markers such as rigid personalities in relation to adjustment problems may aid the development of a suited intervention in the anticipation of subsequent pandemics. Future research will also need to consider how to address and account for this relationship between cognitive inflexibility and attrition rates in longitudinal analyses, as those that are dropping

out may resemble marginalised and under-represented cohorts that are not receiving adequate support.

Conclusion

In conclusion, this study extends prior research by demonstrating that obsessive-compulsive symptoms, depression, anxiety, and stress — with compulsive traits acting as a vulnerability factor — drive post-pandemic adjustment difficulties. Cognitive inflexibility, particularly deficits in reversal learning, further distinguishes poor adjusters. These findings echo the state and trait interactions detailed in Chapter 2; it is suggestive that such interactions between state/trait phenomena precipitate impaired functioning. Findings reveal key intra- and inter-measure differences. The IDED was sensitive to an inflexibility that the WCST was not, reversal learning, suggesting some partial, but not complete, overlap between executive tasks. Moreover, self-reports and neurocognitive task performances of compulsivity remain largely unrelated to each other. Finally, the two assessment modes show unique associations with symptom severity i.e., self-reports of compulsivity (on the CPAS) correlate with symptom severity, which is not reflected on neurocognitive tasks (on the IDED). These findings offer critical insights for public health strategies, advocating for interventions that address obsessive-compulsive symptoms, emotional distress and cognitive rigidity to foster better long-term adaptation.

The following section synthesizes findings from the meta-analyses of Chapter 2 and this current chapter to reveal emerging commonalities in assessing executive impairments in OCD symptomatology.

CHAPTER 3.1: GENERAL DISCUSSION

Latent Cognitive Phenotypes of Impulsivity and Compulsivity in OCD

The synthesis of findings from Chapter 2 and this current chapter illustrates the expressions of impulsivity and compulsivity in obsessive-compulsive symptomatology in both clinical and sub-clinical cohorts.

Sub-clinical compulsive traits appear to be relatively stable across time and have indirect effects on contingent behavioural adaptation via affective symptomatology (symptoms of depression, anxiety, and stress). The profile of compulsivity depicted in Chapter 3 (by the CPAS, WCST, and the IDED), is suggestive of having a predisposing quality to the development of a syndrome defined by inflexibilities and difficulties in adjusting. By extension, the phenotypic manifestation of compulsivity owing to deficient cognitive flexibility can be observed as having key etiological potential for the precipitation of psychopathologies, such as OCD and other compulsive-related disorders (Liu et al, 2023). This emphasises the role of this inflexibility in avoidant-habitual behaviours in sub-clinical severity levels of OCD (Gillan et al, 2016; Gillan, 2021) that underscores the repetitive and ritualistic nature of compulsive symptomatology that is observed at higher symptom severities (Simmler and Ozawa, 2019). Phenotypic manifestations of impulsivity and underlying deficits in inhibitory control was not assessed in this longitudinal assessment and so the pre-clinical composition cannot be commented upon.

Moreover, meta-analytical findings of Chapter 2 substantiate clear deficient performances in patients with OCD compared to healthy controls on neurocognitive tasks of compulsivity (g=-.58, p<.001) and impulsivity (g=-.48, p<.001); and that these phenotypes are of comparable impairment in their relative manifestations in OCD phenomenology (Q=1.99, df =1, p=.16). These latent phenotypes showed distinct independences from typical clinical

markers such as duration of illness, age, medication, comorbid conditions, clinical status (outpatient vs inpatient), and OCD symptom severity. In comparisons with similar meta-analytical paradigms, the analyses conducted revealed that these latent phenotypes are relatively stable from late adolescence and remain fixed irrespective of fluctuating symptom severity (Lopez-Hernandez et al, 2023).

Taken together, these findings reveal crucial clinical (and certainly pre-morbid) implications posed by these latent cognitive phenotypes of compulsivity and impulsivity in OCD. Although the testing paradigm is different between chapters, one aspect remains clear. These latent phenotypes (whether subclinical or clinical) share an interesting relationship (or lack thereof) with OCD symptom severity and other clinically salient features.

Certainly, the pandemic induced something that bore the hallmarks of OCD but was not severe enough to warrant diagnosis, and interestingly, these symptoms remitted over time (as seen in the longitudinal analysis, see Table 3.1.6) despite there being no targeted clinical intervention. The key differences between chapters 2 and 3, between clinical and subclinical OCD, highlight that people with diagnosed OCD have underlying cognitive deficits (trait-like phenomena) as well as symptoms (state-like) while the sub-clinical participants show the symptoms but the not the underlying cognitive deficits. As there was minimal deficient flexibility in the subclinical cohort, this would suggest that both state symptomatology and neurocognitive trait phenomena contribute to the diagnostic class of OCD; the presence of traits appears to sustain and exacerbate the symptomatology. Contrasting these findings with that of Chapter 2 would imply a categorical nature to these traits (see Table 3.1.1) and a dimensionality of OCD symptomatology; a continuum ranging from symptoms to syndrome to an illness severe enough to meet the diagnostic criteria of OCD (Abramowitz et al, 2014).

Table 3.1.1. State and trait configurations between clinical and subclinical samples.

	State symptomatology	Trait neurocognitive deficits
Sub-clinical participants	Yes	No
Patients with OCD	Yes	Yes

As shown in Table 3.2.1, the diagnostic class of OCD represents states in conjunction with traits and subclinical representations comprise states in the absence of traits. The remaining configuration of states and traits would be the presence of neurocognitive deficits in the absence of state symptomatology, a composition found in first-degree relatives of patients with OCD. Previous research has detailed impaired performance on neurocognitive markers despite their level of state symptomatology being indifferent from that of normal comparators (Rajender et al, 2011). This cohort possess a genetic predisposition to developing OCD owing to familial probands and so hold the propensity for psychopathological precipitation (do Rosario-Campos et al, 2005; Chacon et al, 2018). As this work has not focused on this particular group, very little can be argued though this group of familial probands may hold phenomenological compositions that have integral implications for research and clinical practice.

Differing Methods Of Assessing Latent Cognitive Phenotypes

The analyses described in Chapters 2 and 3 relied upon self-reports and neurocognitive tasks to estimate the size of executive impairments in cognitive flexibility and response inhibition, phenotypically manifesting as compulsivity and motor and decision-making impulsivity, respectively. Whilst self-reports provide an accessible assessment medium for research and clinical purposes, they have several shortcomings which may ultimately affect their utility.

Both chapters use widely validated symptomatic scales to detail the severity of OCD symptomatology (the Y-BOCS and the OCI-R) to give accurate representations of current

symptomatic states emphasising their utility within the practical setting. Moreover, chapter 3 utilised self-reports to provide a longitudinal tracking of behavioural indices over a 6-month period detailing excellent test-retest properties of those self-reports used, namely, the OCI-R, the CPAS, and the DASS-21. Although situational mood states were eliminated for their potential influence on reliable assessments in Chapter 3, showing that the relationships observed were not attributable to the presence of depression, anxiety, or stress symptoms at the time of testing, the self-reports used may have been alternatively effected by other sources of interference. Self-appraisal is subject to various manipulations, emanating from a fallible recall bias, agreeability with statements, and the implicit standards by which respondents evaluate their behaviour may differ from that of another (Rosenman, Tennekoon, and Hill, 2011; Alfonsson, Maathz, and Hursti, 2014; Lira et al, 2022). As described in Chapter 1, with this degree of malleability, self-appraisals can render spurious correlations and non-representative data thereby brining into question the reliability and validity of this assessment method within research endeavours to truly capture latent traits such as deficient executive control.

Indeed, there is also a great difficulty in truly isolating and examining specific executive functions of interest as the neurocognitive tasks devised to assess them often rely on various underlying simultaneous cognitive processes (Burgess, 1997); termed the task impurity problem. For example, the Intra-Extradimensional set-shifting task (IDED; Owen et al, 1991), renowned for its robust assessment of cognitive inflexibility, can be drawn on to illustrate this point. Participants are to discriminate between white lines and pink shapes in various compositions to decipher the matching rule, and to then retain this matching rule. This will require efficient working memory, visuospatial capacities, as well as syntactic processing (Sauteraud et al, 2009; Martoni et al, 2015b). Moreover, each successive stage

requires greater cognitive demand and so those with lower IQs may render performance profiles that are comparable to those that are truly cognitively rigid.

Similarly, the Stop-Signal Task (SST; Aron, Robbins, and Poldrack, 2004) requires successful hand-eye coordination to press the correct key that corresponds with the direction of the presented arrow at the appointed and appropriate time. As such, a portion of the variance observed in analyses employing these measures may not be wholly reflective of the impairment (Phillips, 1997); peripheral cognitive deficits, and a potential ceiling effect of IQ, convolute the correct interpretation of the deficit of interest (Miyake, Emerson, and Friedman, 2000; Diaz-Asper, Schretlen, and Pearlson, 2004). Outcome metrics of these tasks may instead provide an estimate of a composite impairment and not a 'pure' assessment of the intended impairment.

Another issue of neurocognitive tasks is the degree of heterogeneity, specifically in task instructions and outcome metrics. As shown in Chapter 2, there is substantial heterogeneity among tasks assessing compulsivity (I^2 =70.09, p<.001). Although, sensitivity analyses in Chapter 2 revealed comparable effect sizes between the IDED and the WCST, analyses in Chapter 3 revealed that the IDED may have been more sensitive to the detection of an inflexibility that the WCST was not; reversal learning (Wildes, Forbes, and Marcus, 2014; Monni et al, 2022). This suggests some partial, but incomplete overlap between tasks measuring the same executive component. The WCST and IDED partly measure the same but also different features of cognitive flexibility and so they may not be completely interchangeable (Gruner and Pittenger, 2017). Of course, these analyses were conducted on separate samples and so this may implicate the IDED as being the more sensitive to cognitive inflexibilities in subclinical samples.

Similarly, the heterogeneity across studies assessing impulsivity was sizeable (I^2 =58.48, p<.001). This was to be expected as a wider scope of impulsive task was included as of the incongruent consensus on inhibitory control (motor and decision-making) impairments in patients with OCD. As shown in the series of conducted sensitivity analyses, the SST did not differ from the Go/no-Go task in assessing response inhibition in patients with OCD. Research has shown that under Electroencephalography (EEG), these two tasks operate on distinct neurocognitive mechanisms (Raud et al, 2020) and therefore illustrate that the deficit in response inhibition is not unitary and that these two tasks are fundamentally distinct in their assessment of this deficit. As each of the two tasks (the SST and Go/no-Go) have distinct neurocorrelates, suggesting that these tasks are complementary rather than hierarchical in detecting group differences in response inhibition.

With regard to a decision-making impairment, subgroup analysis did reveal superiority of the IGT over the CGT in the estimation of this deficit in patients with OCD. Again, caution is warranted here as the IGT measures decision-making under ambiguity and the CGT measures decision-making under risk. This follows as patients with OCD are not typically risk-taking, but risk-averse (Admon et al, 2012) and furthermore, there is a considerable element of indecision that leaves these individuals essentially immobile in the presence of ambiguity and extreme doubt (Rassin and Muris, 2005; Chiang and Purdon, 2023). Importantly, the observed heterogeneity across studies assessing impulsive task performance in patients with OCD can be attributed to the multifaceted nature of impulsivity itself. The variations in task design highlight the challenge of fully capturing this multifactorial deficit; some tasks may not be as equally relevant as others. The consideration of task-specific factors is imperative when interpreting studies assessing impulsivity in the broader context of OCD phenomenology.

The outcome metric selected to estimate the impairment is also a source of this heterogeneity within the use of neurocognitive tasks. As shown in Chapter 2 (see Table 2.1), there were a variety of different outcome metrics used across studies using the same task; Perseverative responses, perseverative errors, and % of perseverative errors were each used to assess cognitive inflexibility on the WCST. However, a key difference emerges between these metrics that has a bearing on their respective interpretation. Miles et al (2021) explained that the 'sandwich' method (looking at the response made before and after the one in question) can be used to decipher the erroneous response. A perseverative response is scored when the preceding response was correct and the next is incorrect and that the matching corresponded with the previous rule. On the other hand, perseverative errors are continuous incorrect responses made using the previous matching rule despite feedback on incorrect responding. Similarly, the Extradimensional shift (EDS) and ED shift reversal (EDSR), stages 8 and 9, are the preferred metrics of choice for the IDED. However, some studies used the total ED errors metric, a collective of ED errors across all 9 stages (Owen et al, 1991), which contain miscellaneous estimates that are not of crucial relevance and may convolute the pooled estimation of set-shifting deficits when comparing with other papers.

As of this impurity and heterogeneity, some have argued for the use of such neurocognitive instruments to be reconsidered or discontinued (Enkavi et al, 2019a) and that the use of these tasks be carefully considered against the research question that is being pursued; the use may be context dependant (Enkavi et al, 2019b). On the other hand, others have argued that these task-based measures are relevant and invaluable in estimating key cognitive-behavioural deficits and can be used in tandem with questionnaires (Friedman and Banich, 2019).

As described previously, one limitation of using psychometric scales within experimental paradigms is common method variance (Kock, Berbekova, and Assaf, 2021); the use of scale

data to populate both the dependent and independent variables thereby contributing to an inflation of the observed correlations that is not representative of the true relationship between variables. By employing both units of analysis (as listed under the RDoC frame work; Cuthbert, 2022), neurocognitive and scale indices provide a comprehensive examination of, in this case, deficient executive control within OCD phenomenology.

However, papers utilising both assessment types are scarce but a consistent discrepancy emerges between self-ratings and task performances of phenotypic manifestations. Indeed, Frydman et al's (2020) use of both assessment types of impulsivity (BIS-11 and SST against the Y-BOCS) showed that the manifestation of impulsivity in OCD was more subjectively reflected and that the two measurement types may be assessing different dimensions of the same phenotype. As described in Chapter 2, in the five papers (of a total of 112) that do assess within-group correlations (between self-ratings and neurocognitive task performances), no significant relationships were revealed. Interestingly, the post-pandemic analyses lend themselves to this narrative of orthogonality between self-ratings and task performances as only a slight correlation was revealed between the CPAS and the WCST (r=.16, p<.05) and no relationship was revealed between the CPAS and the IDED (p>.05).

Although both methods purport to measure the same construct, the approach that each takes may have a bearing on the depiction of the phenotype (Dang, King, and Inzlicht, 2020). This is to say that the very construction and fundamental nature of the method affects the assessment of the phenotypes. For example, self-ratings ask participants to rate and deliver a commentary about the phenotype and how it manifests in their daily lives, whereas the neurocognitive tasks are a direct application of a method that can look at the behaviours under a microscope. Although effected by task impurity, the neurocognitive tasks are said to be more objective and less subject to biases as self-ratings are. Self-ratings are said to be

prone to deficiencies in meta-cognitive appraisals (Friedman and Gustavson, 2022), and so this may account for the relative discrepancy between the differing methods of phenotypic assessment parallel to OCD symptomatology. However, as these tasks render replicable behavioural effects, this is at a loss of between-participant variance (that is typically needed for a robust measurement of individual differences) which may account for their poor correlational capacity with self-reports of the same construct (Dang, King, and Inzlicht, 2020). The psychiatric ill health of the participants that have been examined in the preceding chapters may have also hindered accurate responding under testing paradigms (Kurtz, 2006; McTeague, Goodskin, and Etkin, 2016; Sha et al, 2019) which typically affect the overall reliability of these 'objective' tasks. However, as shown in Chapter 2 comorbidities had no effect on performance and similarly in chapter 3, personal nor familial histories of mental health disorders had any bearing on neurocognitive performance.

The Relationship Between Latent Cognitive Phenotypes And OCD Symptom Severity

OCD symptom severity was measured by the Y-BOCS in Chapter 2. As explained, the differences between healthy controls and patients with OCD on neurocognitive task performances were not moderated by Y-BOCS scores which highlighted an orthogonality between symptom severity and impaired neurocognition, which is consistent with previous meta-analytical findings (Shin et al, 2014; Snyder et al, 2015). However, within-group correlations revealed a significant correlation between symptom severity and impaired neurocognition, though only on assessments of compulsivity. As this the Y-BOCS focuses on the relative severity and disruptive and impairing qualities that these obsessions and compulsions have on daily functioning (Goodman et al, 1989) as opposed to symptom

content or dimensions, the possibility that relationships might exist between cognition and specific symptom clusters, like those examined by the OCI-R, could not be excluded.

Alternatively, the OCI-R was used to assess OCD symptomatology in Chapter 3 by providing an assessment of both symptom severity and symptom clusters (Foa et al, 2002). A significant weak correlation was revealed between OCI-R total scores and perseverative errors on the WCST (r=.19, p<.05) which is consistent with meta-analytical findings of greater symptom severities corelating with greater cognitive inflexibilities in Chapter 2 which did suggest some parallel development of neurocognitive compulsive traits akin to symptom severity albeit cross-sectional discontinuity between clinical and subclinical samples as well as between different symptom severity scales.

Moreover, correlational analyses of OCI-R symptom dimensions detailed a number of weak positive correlations between the hoarding and checking subscales and the non-perseverative and total errors on the WCST. This was not reflected in the three month follow up assessment; no significant relationships were revealed between OCI-R total or subscale scores and the EDS and EDSR stages of the IDED. This may be due to a reduction in OCI-R scores between baseline and three-months (25.69[SD=15.67] vs 17.86[SD=13.22]), though equally, analyses of attrition rates did reveal that the more cognitively inflexible participants did not continue past the baseline condition; and so this lack of relationship may be a reflection of this despite a significant difference between good and poor adjusters on the EDSR stage at three months.

This latter finding is interesting as a difference between groups could be detected on the IDED at three months when OCI-R scores had declined but could not be detected on the WCST at baseline when OCI-R scores were higher, though this may be a reflection on the aforementioned distinction between the WCST and the IDED insofar as not being wholly

interchangeable. This may also suggest that symptom severity and neurocognitive deficits are not mutually exclusive and that impaired executive control can exist independent of clinical significance. This highlights that these impairments can exist pre-morbidly and may constitute a predisposing factor. This is certainly seen in Chapter 3 as rigid dispositions appear to contribute to a diathesis stress model of a syndrome defined by post-pandemic adjustment difficulties.

Interestingly, first-degree relatives show similar neurocognitive patterns to their OCD probands (relatives with OCD) though dissimilar symptom severity (Rajender et al, 2011) which is parallel to findings reported in Chapter 3; impaired performance on the IDED in the absence of diagnosable OCD symptom severity. However, some patients with OCD will show no cognitive impairments. For example, Vaghi et al (2017) show that a portion of their sample is free of this impaired flexibility while other participants do have this deficit and so the identification and assessment of this deficient executive profile is key as they may provide novel implications for clinical practice, such as effects on treatment response (Keeley et al, 2008; Waters et al, 2020).

Research by Stilley et al (2010) does show that cognitive inflexibility was a predictor of treatment responsiveness in that those with greater inflexibilities struggled with adhering to medicinal stratagems. However, van Passel et al (2025) showed that remediating (correcting and reducing) cognitive inflexibility does not necessarily (and indeed directly) influence treatment outcome and so mechanisms of change may be context-dependant and effected by other mediating factors within the clinical setting. This is reminiscent of the collection of subgroup analyses recorded in Chapter 2 that depicted an independence of cognitive inflexibility from clinically salient variables such as medication effects, comorbid conditions,

and clinic status. As such, conceptualising cognitive inflexibility, adjacent to symptom severity, may hold prognostic significance.

Importantly, impulsivity was not assessed in the longitudinal analysis of Chapter 3 and so inferences made here on its composition within OCD phenomenology reflect and extend discussions from Chapter 2. The relationship between OCD symptom severity and impulsivity appears to be distinct to the relationship between OCD symptom severity and compulsivity and certainly warrants unpicking.

The impaired performance of patients with OCD compared to that of healthy controls cannot be attributed to fluctuating Y-BOCS scores (given the non-significant moderation of between-group comparisons). Furthermore, this is also the case for within-group correlations – impulsive task performance was not moderated by Y-BOCS scores. Taken together these findings are indicative of a stable latent cognitive trait of impaired inhibitory processes in patients with OCD that appears to be independent of symptom severity. It may be that impulsive features of the disorder only manifest at particular junctures of symptom severity; impulsive components may represent a non-linear demarcation for worsening obsessive-compulsive symptomatology.

Prior research does document an absence of such impulsive phenomena at lower symptomatic levels in OCD (Ettelt et al, 2006) which is consistent with this conjecture of impulsive prominence once a clinically significant threshold has been surpassed. However, Rajender et al (2011) showed that first-degree relatives did not exhibit dissimilar response inhibition to their familial probands' performance suggesting that this deficit can exist pre-morbidly, though this may be a reflection of a familial predisposition and may not be true for sporadic cases. Indeed, Bhattacharya et al (2021) showed that patients with familial OCD performed

worse on the SST compared to patients with sporadic OCD though this failed to reach significance.

Taken together, the qualitative differences in the relationships between compulsivity and impulsivity with OCD symptom severity resonate with the proposal of Chamberlain et al (2017) "…to use a multi-tiered approach examining not just categorical mental disorders but also dimensional psychopathology" (p.814).

The synthesis of findings from the preceding two chapters have delineated clear deficiencies in executive functioning in patients with OCD; latent cognitive phenotypes of impulsivity and compulsivity and their respective manifestations and unique clinical implications. Findings have further revealed that the assessment of these latent cognitive phenotypes exclusively relies on self-ratings and neurocognitive tasks, and how each method of assessment casts a different perspective on the relationship between these phenotypes and OCD symptom severity. The relative orthogonality between these phenotypes and OCD symptomatology indicates insufficient probing of such deficits within the clinical assessment of OCD leaving these impairments unchallenged by current treatment interventions. Subjecting the current assessments of OCD symptomatology and phenomenology to scrutiny will reveal novel insights into how diagnostic and therapeutic frameworks may be refined in the conceptualisation of OCD. As such, this could highlight the need for a more nuanced multidimensional assessment tool which could ultimately enhance the specificity of symptom profiling by accounting for the cognitive underpinnings of the disorder.

The following chapter reviews current assessments of OCD symptomatology and their respective scope for measuring executive impairments. The development and validation of a new self-report tool assessing these functional impairments in two independent samples is detailed.

CHAPTER 4: DEVELOPMENT OF THE OBSESSIVE-COMPULSIVE PHENOMENOLOGY ASSESSMENT SCALE (OCPAS) FOR THE SELF-ASSESSMENT OF OCD SYMPTOMS AND TRAITS

Measuring OCD Symptom Severity

The universal features of OCD, obsessions and compulsions, are captured in clinical classifications such as the DSM-5 (American Psychiatric Association, 2013) and the ICD-11 (World Health Organisation, 2022b), which are used to converge on a diagnosis of OCD. While the definition of OCD highlights universal symptoms, the diverse range of phenomenology makes presentation heterogeneous (Lochner and Stein, 2003). Researchers are urging for the inclusion of additional phenomena such as impulsive tendencies and neurocognitive traits in the conceptualisation and assessment of OCD to better understand their interaction and influence on clinical presentation (Bragdon and Coles, 2017; Tiego et al, 2018; Suhas and Rao, 2019). As OCD is a disorder of dysfunctional goal-directed behaviour (Gillan and Robbins, 2014), then the '*goal*' is the elimination of obsessional anxiety - the suppression of ego-dystonic intrusions. Patients with OCD have limited autonomy in the attendance to this distress (Meynen, 2012; de Haan, Reitveld, and Denys, 2015). As explored in Chapter 2, this lack of autonomy, from a neurocognitive perspective, can be attributed to

irregular executive functioning (most notably cognitive inflexibility and impaired inhibitory control) and so accurate self-assessment tools of such neuropsychiatric nuances would be crucial for diagnosing and managing OCD. A summary of key measures of Obsessive-compulsive symptomatology can be found in Table 4.0.1.

Table 4.0.1. Summary of Obsessive-compulsive measures

Measure	Number of items	Key factors assessed	Psychometric properties	Example items
Yale-Brown Obsessive-Compulsive Scale (Goodman et al, 1989) Clinician-rated	10	Obsessions: Time occupied; Interference; Distress; Resistance; and Degree of control. Compulsions: Time occupied; Interference; Distress; Resistance; and Degree of control.	Excellent internal consistency and inter-rate reliability. (Goodman et al, 1989); Good temporal validity (Federici et al, 2010); and excellent measurement invariance across groups (Schulze et al, 2023).	How much distress do your obsessive thoughts cause you? How much time do you spend performing compulsive behaviours? How much longer than most people does it take to complete routine activities because of your rituals? How frequently do you do rituals?
Yale-Brown Obsessive-Compulsive Scale - II (Storch et al, 2010) Clinician-rated Patient-rated	19	Obsessions: Time occupied; Obsessional-free interval; Interference; Distress; Resistance; and Degree of control. Compulsions: Time occupied; Compulsion-free interval; Interference; Distress; Resistance; and Degree of control. Insight into obsessions and compulsions; Avoidance; Degree of Indecisiveness; Overvalued sense of responsibility; Pervasive slowness/Disturbance of inertia; Pathological doubting; Global severity; Global improvement; and Reliability.	Can discriminate OCD from other disorders with anxiety and depression symptoms. Sufficient convergent validity with other clinician-rated measures of OCD. Sufficient discriminant validity with measures of worry and impulsivity. (Storch et al, 2010; Rowa et al, 2025).	Do you think your concerns or behaviours are reasonable? What do you think would happen if you did not perform the compulsion(s)? Are you convinced something would really happen?
Obsessive-Compulsive Inventory-Revised (Foa et al, 2002)	18	Hoarding; Obsessing; Ordering; Washing; Checking; and Neutralising	Excellent internal consistency (Foa et al, 2002); can identify diagnosable OCD severities (Hupper et	I check things more often than necessary.

Patient-rated			al, 2007; Veale et al, 2016a);	I sometimes have to wash or clean myself simply because I feel contaminated.
Padua-Inventory (Sanavio, 1988) Patient-rated	39	Contamination obsessions and washing compulsions; Dressing/grooming compulsions; Checking compulsions; Obsessional thoughts of harm to self/others; and Obsessional impulses to harm self/others	Good convergent and discriminant validities (Sternberger and Burns, 1990; van Oppen, 1992); and reliable factor structure (Burns, 1995).	I feel obliged to follow a particular order in dressing, undressing, and washing myself. While driving, I sometimes feel an impulse to drive the car into someone or something.
Obsessive Beliefs Questionnaire (Obsessive Compulsive Cognitions Working Group, 2003) Patient-rated	44	Responsibility/threat estimation; perfectionism/certainty; importance/control of thoughts	Adequate internal consistency (Moulding et al, 2011); Construct validity with obsessive-compulsive symptom measures (Fergus, Latendresse, and Wu, 2017).	Having violent thoughts means I will lose control and become violent. Having a blasphemous thought is as sinful as committing a sacrilegious act.
Obsessive-Compulsive Trait Core Dimension Questionnaire (Summerfeldt et al, 2001) Patient-rated	20	Harm avoidance; Incompleteness	Strong internal consistency and construct validity with obsessive-compulsive personality traits and symptom severity (Ecker, Gonner, and Wilm, 2011; Cervin and Perrin, 2019)	I get scared as if something bad could happen or has already happened. I feel like I have to do things in a very specific way, although it would be difficult to put this particular way into words.
Dimensional Obsessive-Compulsive Scale (Abramowitz et al, 2010) Patient-rated	20	Concerns about germs and contamination; Concerns about being responsible for harm, injury, or bad luck; Unacceptable thoughts; Concerns about symmetry, completeness, and the need for things to be 'just right'	Proven reliability convergent validity with other OCD measurements (Kuhne et al, 2021) and is sensitive to treatment-realted change in symptom severity (Farrell et al, 2024).	To what extent have you avoided situations so that you did not have to check for danger or worry about possible harm or disasters? When unwanted or unpleasant thoughts come to mind against your will how distressed or anxious did you become?
Maudsley Obsessive-Compulsive Inventory (Hodgson and Rachman, 1977)	30	Checking compulsions; Washing/cleaning compulsions; Slowness; Doubting	This measure can reliably differentiate individuals with OCD from those with other psychiatric conditions such as Anxiety and Anorexia Nervosa (Karpov et al, 2022).	I frequently have to check things (e.g. gas or water taps, doors, etc.) several times. I have a very strict conscience.

The Yale-Brown Obsessive-Compulsive Scale (Goodman et al, 1989) is the preferred clinical tool of assessing OCD symptom severity. A five-point Likert scale (0-4) is used to rate the severity of obsessions and compulsions on the following five domains; time occupied by symptoms, interference due to symptoms, distress associated with symptoms, resistance against symptoms, and degree of control over symptoms. The latter two domains, resistance and degree of control, loosely allude to the previously described deficits in executive functioning, diminished inhibitory control processes in both thoughts and behaviours as well as a weakened capacity to stop goal-directed behaviour once the goal has been attained – resulting in perpetual (repetitive) action. Indeed, this may account for the effect observed in Chapter 2's meta-analysis of within-group correlations between Y-BOCS scores and performances on the WCST and the IDED. The Y-BOCS has sufficient internal consistency and inter-rater reliability (Goodman et al, 1989; Storch et al, 2010) and has proven to be sensitive to changes in symptom severity; Ramakrishnan et al (2024) showed that benchmarks on the Y-BOCS can successfully delineate treatment response and remission rates. However, with regard to symptom content, the Y-BOCS leaves much to be desired. As suggested in Chapter 2, relationships may exist between specific symptom clusters and cognition; Bragdon, Gibb, and Coles (2018) show that patients with symmetry and ordering symptoms were more functionally impaired than those patients with obsession and checking symptoms. The Y-BOCS does include a checklist of some common obsessions and compulsions but this measure does not lend itself to dimensional symptom clustering that is common among other psychometric assessments of obsessive-compulsive symptomatology such as the Obsessive-compulsive Inventory-Revised (Foa et al, 2002) and the Padua Inventory (Sanavio, 1988; Burns, 1995).

The updated Y-BOCS (2nd edition; Storch et al, 2010) enlists items 1b and 6b, detailing on durations of symptom-free intervals upon clinical improvement. Other salient additions are:

insight into symptoms, avoidance, degree of indecisiveness, overvalued sense of responsibility, pervasive slowness/disturbance of inertia, pathological doubting, and global severity. Similar to its predecessor, this updated rendition has sufficient psychometric properties (Wu et al, 2016; Rowa et al, 2025). The pervasive slowness/disturbance of inertia is of particular interest here as there is an overlap between the item's description (*Do you have difficulty starting or finishing tasks? Do many routine activities take longer than they should?*) and cognitive inflexibility. Certainly, this item is consistent with measures of transdiagnostic compulsivity, namely the Compulsive Personality Assessment Scale (CPAS; Fineberg et al, 2007) and the Cambridge Chicago Trait Scale (CHIT; Chamberlain and Grant, 2018). However, both this item and global severity are not included within the calculation of symptom severity and so there is no estimation of these functional deficits within the clinical assessment of OCD under the Y-BOCS-II. Moreover, there remains no elicitation of impulsive features within OCD phenomenology between revisions of the Y-BOCS. Though there is an element of impaired decision-making in the second edition (*Do you have trouble making decisions about little things that other people might not think twice about?*), this is less of an impulsive choice and more of the premeditative forethought as argued by Abramovitch and McKay (2016). Further, as the Y-BOCS is a clinician-rated outcome measure, this does subject the scale to observer bias – assessment and scoring is based on interpretations and commentaries and so this may not be a true reflection of the patients' reality.

Clinician-rated outcome measures (CROMs) are typically designed for clinicians to render diagnostic assessments of symptomatology and functional impairments such as the Y-BOCS (Goodman et al, 1989) and the Montefiore-Einstein Rigidity Scale-Revised (Mendlovic et al, 2022; Taylor et al, 2022), though as described an inherent bias exists within this mode of assessment. Patients are considered experts of their own lived experience and can contend

with scale items concerning current experiences and symptoms and so patient-rated outcome measures (PROMs) are developed to capture such insights (Kendrick et al, 2016). This also reduces the observer bias that is inherent within CROMs as interpretation is not required from clinicians (Food and Drug Administration, 2009; Baandrup et al, 2021). This is not to say that PROMs are without fault. In fact, given the idiosyncratic nature of OC symptomatology, patients may endorse multiple subtypes on a scale (e.g., washing, checking, ordering) which may be misconstrued as a greater overall severity, when it simply reflects diversity in symptoms rather than intensity (Overduin and Furnham, 2021). Nonetheless, Clements et al, (2006) showed that psychiatric outcome was better predicted by self-ratings than by clinician ratings. Though there may be differences between PROMs and CROMs (such as the Y-BOCS; Goodman et al, 1989), previous research contends with sufficient concordance between self-and other-ratings (Nuzum, Ready, and Clark, 2019). Indeed, there is some convergence between clinician and self-ratings on the Y-BOCS-II-SR (Rowa et al, 2025). PROMs may certainly have the advantage in the assessment of psychopathology as these measures have been shown to facilitate the relationship between patient and clinician, thereby promoting patient involvement and treatment adherence (Kristensen, 2020; Baandrup et al, 2021).

The Obsessive-Compulsive Inventory-Revised (OCI-R; Foa et al, 2002) is one such PROM that provides a dimensional structure of OCD symptom content. The scale consists of 18 items that are measured on a 5-point Likert scale (0-4; not at all, a little, moderately, a lot, and extremely) that captures symptom dimensions pertaining to Washing, Obsessing, Ordering, Checking, hoarding, and Neutralising phenomenology. The OCI-R can be used to distinguish OCD symptomatology from Hoarding disorder symptomatology (Wootton et al, 2015) and has a threshold score of ≥ 21 for potentially diagnosable OCD symptoms (Huppert et al, 2007; Veale et al, 2016a). As shown in Chapter 3 (post-pandemic adjustment difficulties), the

OCI-R was able to detect gradual decreases in OCD symptomatology within the sample of non- to sub-clinical participants over the three assessment points highlighting a sensitivity to temporal variances in OCD symptom severity. The OCI-R is certainly a robust patient-rated measure of OCD symptomatology.

However, there is no overt measurement of the more functionally impaired aspects of OCD phenomenology, such as cognitive flexibility and inhibitory control, within the OCI-R and there appears to be a limited convergence between higher scores on the OCI-R and impaired performance on the WCST (r=0.19, p<.01; as seen in Chapter 3, Table 3.2) implying that the OCI-R has little sensitivity in detecting these functional aspects of the condition. However, this was a longitudinal assessment of an undiagnosed cohort and so this may be indicative of these functional deficits existing at the higher end of OCD symptomatology, which may also account for why within-group correlations were observed between Y-BOCS scores and cognitive task performances in patients with OCD but not observed in between-group contrasts of patients with OCD against healthy controls (see meta-analytical findings in Chapter 2). This difference in finding may also speak towards differences between the two metrics of OCD symptomatology, the Y-BOCS being of a clinician interpretation of illness severity and the OCI-R being of a meta-critical commentary on symptomatology, though the OCI-R does show comparable symptom severity benchmarks with the Y-BOCS (Abramovitch et al, 2020).

An alternative patient-rated outcome measure to the OCI-R is the Padua Inventory (PI; Sanavio, 1988). Similarly, this scale is rated on a 5-point Likert scale (0-4; Not at all, A little, Moderately, A lot, and Extremely) and consists of 39 items that comprise 5 distinct subscales. These being: contamination obsessions and washing compulsions, dressing/grooming compulsions, checking compulsions, obsessional thoughts of harm to self or others, and

obsessional impulses to harm self or others. The Padua Inventory has convergent and divergent validities with other scales (Sternberger and Burns, 1990; van Oppen, 1992), excellent internal consistency and test-retest reliability (.93 and .84, respectively; Sánchez-Meca et al, 2017) and has been verified across culturally diverse samples (Mataix-Cols, Sanchez-Turet, and Vallejo, 2002; Zhong et al, 2011; Seyedsalehi et al, 2016). Parallels exist between the PI and other scales (the OCI-R and the Y-BOCS) with regards to washing, checking, ordering, and symmetry symptom dimensions; the feature that is of key import in this consideration of the Padua Inventory is the focus on obsessional symptoms. Certainly, the OCI-R consists of an obsessing subscale, consisting of three items that converge on the negativity of the intrusions and the lack of control that the patient can exert over these thoughts and feelings, and the Y-BOCS delivers a measure of the severity of obsessional symptoms (Goodman et al, 1989; Fao et al, 2002; Storch et al, 2010), however, the PI explores obsessional symptomatology with a particular focus on intrusions of thoughts pertaining to the harm of oneself or others. This subscale captures examples of these anxiety provoking thoughts and beliefs (*I think or worry at length about having hurt someone without knowing it*) and an acknowledgement of impulsive features; urges to act on these obsessional intrusions (*When I look down from a bridge or a very high window, I feel an impulse to throw myself into space*) that is not captured in other scales of OCD phenomenology. There are thematic parallels between these two subscales of the Padua Inventory and response inhibition deficits, however, limited data exists on the associations between scores on the PI and neurocognitive data of impaired inhibition.

In sum, contrasting the measures of OCD symptomatology reveal two prominent themes. The first being the method of application, clinician-rated vs patient-rated outcome measures. It appears that the rater, the one who completes the assessment, has a significant bearing on the scoring of phenomenology. Anholt et al (2009) showed that this very difference could underly

the relative divergences observed between the Y-BOCS and Padua Inventory scores of OCD symptom severity. The second theme is that these measures fall short in the acknowledgment and subsequent assessment of functional impairments. Though the Y-BOCS now includes an item that captures cognitive inflexibility, labelled as pervasive slowness/disturbance of inertia (Storch et al, 2010), this is not included in the total symptom severity score, and there remains no distinct assessment of impaired inhibitory control in OCD symptomatology aside from the two subscales of the Padua Inventory. The phenotypic variance of OCD symptomatology may underscore overlapping neurocognitive circuits (Hashimoto et al, 2011) and so developing a scale that isolates the inherent compulsive and impulsive features across the various symptom profiles, dimensions, and manifestations would be novel to identifying the more subtle functional impairments within OCD symptomatology and phenomenology.

Capturing Compulsivity and Impulsivity in the Assessment of OCD

Disinhibition is a crucial component to the understanding of OCD symptomatology and phenomenology (Ettelt et al, 2007; Benatti et al, 2013) and is of an equivocally sized deficit compared to cognitive inflexibility (see Chapter 2). However, aside from the aforementioned lack of acknowledgement of impulsivity within current assessments of OCD (Kashyap et al, 2012), transdiagnostic measures of impulsivity that capture impulsive dispositions in a general non-specific capacity across psychiatric conditions have been used to capture this phenotype in OCD. The most common assessments of impulsivity are the Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, and Barratt, 1995) and the UPPS-P impulsive behaviour scale (UPPS-P; Lynam et al, 2006; 2013); both have received ample psychometric validation in clinical and non-clinical samples (Vasconcelos, Malloy-Diniz, and Correa, 2012; Dugre et al, 2019; McCredie et al, 2020). The BIS-11 consists of three subscales: attentional impulsivity, motor impulsivity, and non-planning impulsivity and the UPPS-P impulsive

behaviour scale consists of five subscales, negative urgency, lack of premeditation, lack of perseverance, sensation-seeking, and positive urgency. A number of these subscales resonate with aspects of OCD phenomenology. The negative urgency subscale of the UPPS-P is described as acting impulsively to alleviate negative affect (Billieux et al, 2021) which is consistent with patients with OCD feeling that they must attend to their ritualistic compulsions to find relief from obsessional distress (Morillo, Belloch, and Garcia-Soriano, 2007). This certainly requires unpacking as this would implicate impulsivity within the mechanism of obsessional symptoms. Gay, Schmidt, and Van der Linden (2010) contend with associations between the UPPS-P urgency subscale and control over thoughts (r=0.53) and could significantly predict scores on the obsessing symptoms subfactor of the OCI-R (β =0.45, t=4.78, p<.001).

Further research substantiates this link between obsessional symptoms in OCD and impulsivity as obsessive thoughts were shown to correlate with cognitive instability (r=0.60, p<.05), a first order factor of the BIS-11 that comprises attentional impulsivity (Koenn et al, 2023). As discussed in the narrative synthesis of Chapter 2, there were a number of papers that commented on an association between attentional phenomena and Y-BOCS scores, more specifically, obsessional scores (De Geus et al, 2007; da Rocha et al, 2011; Boisseau et al, 2012). Interestingly, Onur et al (2016) showed that higher attentional impulsivity scores are associated with key obsessional types (sexual, aggressive, and religious) in patients with OCD that overlap with other impulsive control disorders. As impulsivity appears to underscore a crucial component in OCD symptomatology, this does bring into the question the orthogonality observed between performances on neurocognitive tasks of impulsivity and Y-BOCS scores in Chapter 2. As explored in Frydman et al (2020), the impulsivity in OCD phenomenology is of a subjective (self-rated) derivative and as the impulsive phenotype in Chapter 2 was exclusively measured using objective neurocognitive tasks, this may account

for the observable orthogonality between these tasks and the Y-BOCS. Despite this, Hudiburgh et al (2021) Showed that domains of impulsivity underly the relationship between obsessions and compulsions. Obsessions appear to be motivated and predicted by *pervasive influence of feelings*, and compulsions are subsequently motivated by *feelings trigger action* and so this harkens back to the earlier descriptions of patients with OCD initiating and performing compulsive acts out of an impulse (an urgency) to neutralise the obsessional affect. This theme of urgency may resemble a manifestation of the impaired response inhibition and decision-making (as detailed in Chapter 2) as patients with OCD are not able to delay or inhibit, or choose to delay or inhibit, acting in immediate gratification (this alleviation of intrusions) for long term benefit (Pushkarskaya et al, 2016). The phenotype of impulsivity is an important construct to consider in the assessment of OCD as it underscores obsessional symptoms and its acknowledgement will help to illustrate and highlight the relative mechanisms behind these symptoms and how they interlace with compulsions.

Similarly, compulsivity may underlie various behavioural aspects of OCD such as a need for control, a need for certainty, and habitual behaviours (Moulding et al, 2008; Gillan et al, 2011; Moore et al, 2023). As compulsivity constitutes a core feature of OCD phenomenology, ample measures have been designed to suit its assessment within research and clinical practice, such as the Compulsive Personality Assessment Scale (CPAS; Fineberg et al, 2007). The CPAS includes 8 items rated on 5-point Likert scale (0-4), each with a distinct focus on compulsive manifestations: Preoccupation with details, Perfectionism, Workaholism, Over-conscientiousness, Hoarding, Need for control, Miserliness, and Rigidity. The scale was developed on the template of Obsessive-compulsive personality disorder (APA, 2013), otherwise known as Anankastic personality disorder (WHO, 2022b) and resembles the archetype of pervasive, rigid, and perfectionist dispositions. As detailed in Chapter 3, the CPAS provides a robust measure of trait compulsivity with excellent internal consistency in a

general sample (α = 0.86) (r=0.16, p<.05) and exhibits some limited overlap with the WCST (r=0.16, p<.05). Moreover, the topic of compulsivity permeates subfactors of many other scales such as: compulsive checking and grooming in the Padua Inventory (PI: Sanavio, 1988; Burns, 1995; 1996); the rigidity and difficulty with change in the Pathological Obsessive-Compulsive Personality Scale (POPS; Pinto, Ansell, and Wright, 2011); the behavioural and cognitive rigidity domains of the Montefiore-Einstein Rigidity Scale-Revised (MERS-R; Taylor et al, 2022); and the perfectionism, flexibility, and compulsions items of the Cognitive Assessment Instrument of Obsessions and Compulsions (CAIOC-13; Dittrich, Johansen, and Fineberg, 2011). Whilst descriptions given by the aforementioned scales and subscales for compulsive dispositions are of an attentional inflexibility, habitual behaviours may describe the more inherent and covert aspects of compulsivity.

As habits are fundamental in behavioural development, they form an essential subject for understanding ritualised action and behaviour in OCD. The repetitive behaviours and actions become ingrained and automatic under particular circumstances which Ersche et al (2017) refers to as *automaticity* (automatic behavioural patterns) which may account for the overwhelming engagements with compulsions despite insight into the ego-dystonic themes (APA, 2013). This certainly implicates the habitual system as having salient features in the development and maintenance of OCD symptomatology (Kalanthroff et al, 2016). As described by Gillan et al (2016), the mismatch between goal-directed behaviour and stimulus-driven habitual learning systems reflects the essence of compulsivity. With regard to the assessment of habits in OCD phenomena, the Cambridge-Chicago Compulsivity Trait Scale (CHIT; Chamberlain and Grant, 2018) fosters a single item with an explicit mention of habits within trait compulsivity (e.g., 'I am a creature of habit'). Additionally, the Padua Inventory and OCI-R consist of items with particular phrasing (*I feel obliged* or *I feel compelled*) that allude to the routine and automaticity of habitual behaviours within compulsive

symptomatology. However, as described in Chapter 2, there is an underrepresentation of the estimations of habit under performance task paradigms that can be cross-examined with current models of cognitive inflexibility, and so the extent of executive function associated with habitual systems is lacking. To conject, the early stages of the WCST (and the IDED) may provide feedback to the participant on correct responses which may encourage continued responding regardless of the rule change and as Grundler et al (2009) showed, patients with OCD are unable to adapt responding despite negative feedback on performance, and so this culminates in perseveration; inflexible responding owing to early positive reinforcement.

As described, patients with OCD, upon the instance of an intrusion feel an urgency to quench this obsessional negative affect. It is here where a developed routine or ritual that has previously abated this anxiety or distress (the compulsion) will be activated to respond to the intrusion – this automacity. The compulsion is performed with limited thought or regard for consequences (Julien, O'Connor, and Aardema, 2007); which is consistent with previous discussions on deficient inhibitory processes in patients with OCD. This is an example of a negative feedback loop. This implicates both impulsivity and compulsivity as core features in the perpetuation of dysfunctional goal-directed behaviour in OCD (Hogarth, Chase, and Baess et al, 2012; Gillan et al, 2020). There appears to be a dichotomy between these two phenotypes; and with the implications of this negative feedback loop, may share a symbiotic relationship across psychiatric conditions. However, these phenotypes are seldom considered and assessed in unison.

Work by Chamberlain et al (2017) does contend with a core underlying feature of disinhibition in OCD and that compulsivity and impulsivity represent partitions of the same central deficit. However, psychometric scales that assess compulsivity adjunct to impulsivity are scarce. The CHIT is primarily a scale of trait compulsivity of 15 items rated on a 4-point

Likert scale (1-4) with verified psychometric properties (Tiego et al, 2023), though there a number of items that do cohere with themes of impulsivity (*I can't resist acting on urges* and *I do things that are immediately rewarding, even if I know it is bad for me in the long run*). However, this is not enough to substantiate a subscale of impulsivity akin to the compulsivity. Alternatively, the Impulsive-Compulsive Behaviours Checklist (Guo et al, 2017) consists of 33 items that are rated on a 4-point Likert scale. Items were sampled from the DSM-IV-TR descriptions of disorders with pathological disinhibition. Some relationships were observed between this scale and symptom dimensions of the OCI-R, the strongest being between ordering and checking subscales and impulsive-compulsions. These impulsive-compulsions are described as compulsive actions or behaviours with inherent impulsive undertones which may characterise OCD as a disorder of deficient impulse control. However, as the study authors note, the scale is not effective at identifying the core motivations of the included items despite the sizeable length of the scale and so further questioning and adjunctive measures would be needed to supplement the use of this Impulsive-Compulsive Behaviours Checklist. There is certainly a need for an updated and more concise assessment of the subtleties of impaired executive functioning that captures this connectivity between compulsivity and impulsivity.

Theoretical Frameworks For Scale Development

As discussed, the assessment of OCD symptomatology and phenomenology mostly relies on assessments by clinician-rated outcome measures (such as the Y-BOCS; Goodman et al, 1989) or patient-rated outcome measures (such as the OCI-R (Foa et al, 2002) and Padua Inventory (Sanavio, 1988; Burns, 1995)). This is because these psychiatric constructs resemble unobservable traits and so the exploration of such dispositions must rely on behavioural observation or self-detailed commentaries (Kaplan and Saccuzzo, 2001).

Measures such as these are developed under two competing theoretical frameworks; Classical Test Theory (CTT) and Modern Test Theory (MTT). Both methods assess and outline ways in which the reliability and validity of psychometric instruments can be improved (Progar, Sočan, and Peč, 2008). However, one key difference exists between these two approaches to scale development; CTT assumes equal capabilities among all respondents while MTT assumes that answering is dependent upon the level of the trait in the respondent (Cappalleri, Lundy, and Hays, 2014). This certainly has important implications for clinical practice as some variables of import that are unique to this discipline may go untreated in the development of such instruments, thereby impacting an otherwise precise diagnostic assessment.

Classical test theory is a theoretical framework that is based on a linear method of developing psychometric scales (Hambleton and Jones, 1993). CTT posits that the observable test score (X) can be measured by the sum of two unobservable statistics, error score (E) and true score (T); which can be represented as $X=E+T$. As such, the observable score, or test score as referred to in some texts (DeVellis, 2006), is a reflection of the information pertaining to the variable of interest in addition to the error associated with each of the scale items. Principal Component Analysis (PCA), an applied method of CTT, aims to reduce data dimensions by identifying core features that capture the most variance at the lowest possible number of items whilst maintaining internal consistency (Jolliffe and Cadima, 2016). The Y-BOCS, the OCI-R, and the Padua Inventory have been subject to PCA in their respective psychometric validations (Sternberger and Burns, 1990; Stewart et al, 2007; Wootton et al, 2015). The assumptions of this method are easy to meet and is therefore more common place in the academic literature as well as in applied disciplines due to this high accessibility and replicability (Kaplan and Saccuzzo, 2001). This method also allows for sensitivity and specificity of the scale to be assessed (De Champlain, 2010). Additionally, this method does

not require a sizeable sample size to satisfy the statistical model; 200-500 participants depending on the rule of thumb. Everitt (1975) suggests a recruitment rule of 10 participants for each scale item, whereas, Hair et al (1998) suggests that 5 participants would be sufficient. However, Guadagnoli and Velicer (1988) argue that minimum sample sizes for scale development under CTT is more effective than item ratios for devising and justifying sample sizes. Both methods have merit though favouring one method over another may have some effect on the inferences made about the scale as the sample may not be representative or be sufficiently powered to detect effects (Cliff, 1970; Magno, 2009; Jolliffe and Cadima, 2016). Scales that have been developed under Classical Test Theory are not scrutinised under the strict guidelines and parameters akin to Modern Test Theory and so scales such as the OCI-R may harbour inherent biases.

Conversely, Modern Test Theory is a non-linear approach to scale development; it works by mapping item response on to the levels of latent traits. This method is invariant across samples and populations and is less susceptible to biases from sample properties that CTT may be prone to (Reise, Ainsworth, and Haviland, 2005). MTT provides a unidimensional evaluation of scale properties of an instrument and is inclusive of differing rating regimes; ye/no categorical answering systems (dichotomous) as well as scores rated on a Likert scale (polytomous). Three utilities to emerge from this theoretical framework: (1) items are developed to capture the most information possible about the degree of the latent trait under investigation, (2) to tailor the items to a particular difficulty for interpretative purposes, and (3) to reduce the total number of scale items without impacting reliability or validity (Harvey and Hammer, 1999; Urbina, 2014).

As described, both CTT and MTT are adept at pruning inadequacies for the improved performance of the scale with regard to internal consistency and validity, though of course

there is deviation within the methods applied. An application of Modern Test Theory is the Rasch model which assesses items independent of the sample (Hambleton and Jones, 1993; Yang and Kao, 2014) and is a widely used MTT method for the development of PROMs. As is documented in a scoping review of 55 papers recording scale development under MTT frameworks, 32.7% utilised the Rasch model (Liu, Mueller, and Sedaghat, 2024). Certainly, this model can assess whether the participants addressed the items as intended (which helps to identify feigned results) as well as highlighting any biases that incurred by demographic variance, called Differential Item Functioning (DIF). This can help with eliminating any deviation and variation in responses to each item that may have incurred through selection biases (Penfield and Lam, 2005). This certainly improves internal consistency and external construct validities (Penfield and Camilli, 2006). Moreover, the item difficulty can be mapped along a continuum using Wright maps to provide a visual representation of the relative difficulty of the items (Boone, 2016). This can be used to compare and contrast items of the same latent traits (Stout et al, 2012). In essence, this method assesses the fit of the items to the overall model (Kline, 2005; Boone, Staver, and Yale, 2014). The Likert scale can also be assessed, if the parameters are too wide then this can be identified and addressed (0-7 reduced to 0-4 for example) to improve the overall functionality of the scale (Kline, 2005). These are features that CTT cannot provide.

An alternative MTT framework to the Rasch model is Item Response Theory (IRT). IRT provides greater flexibility in the information entered into parameter estimates and introduces item discrimination to expand upon person ability and characteristics. This method finds the model that best fits the variance observed within the data and so in each case of the model being assessed, there may some degree of variation depending on the data and person ability (Stemler and Adam, 2021). However, this flexibility may come at cost of the scale's invariance across different populations, which may prove to be a detriment to the

development of a PROM. As this model is heavily sample-dependent, there would be variability (heterogeneity) both between test-users (e.g., at-risk sub-clinical cohorts, patients diagnosed with OCD) and also within the ability estimates of the same individual (measurement of the latent trait). The ability of IRT to provide a measurement of the latent trait at each level of person ability is compromised and may be considered a statistical correction to the of CTT with regard to weighted scores but does not advance meaningful scale development and so the invariant propositions of the Rasch model would be most advantageous for the aims of this work (Stemler and Adam, 2021).

The merit of the Rasch model is found within its fit statistics. It allows for the evaluation and development of a linear scale that functions irrespective of the respondent sample; aiding meaningful interpretation of the latent trait as test scores have the same meaning across participants (which would otherwise be lost under IRT). As the purpose of this work is to produce a scale that can assess latent traits and discriminate respondents based on the level of the latent trait, the Rasch model is the most appropriate MTT model to use.

The Maudsley Obsessive-Compulsive Inventory (MOCI; Hodgson and Rachman, 1977) is the sole measure of OCD symptomatology to have undergone psychometric validation under the Rasch model (Inchausti and Delgado, 2012). Though there are conflicting accounts on the superiority of MTT over CTT (Fan, 1998; Jabrayilov, Emons, and Sijtsma, 2016), the sophistication of MTT methods restricts its use in both research and applied fields. Further, the sample sizes required to satisfy such methods are far in advance of CTT, ranging from 200 to 1000 depending on the number of identified dimensions (Downing, 2003; Kose and Demirtasli, 2012).

Interestingly, Jabrayilov, Emons, and Sijtsma (2016) show that MTT is superior to CTT in detecting significant clinical change, though the authors do note that this was exclusive to

scales in excess of 20 items and that for scales under this threshold, CTT was more adept at detecting significant change in clinical cohorts. However, some research contends with these methods being used in tandem to (1) detect latent dimensions and (2) to improve the functionality of the test (Awopeju and Afolabi, 2016). Indeed, Petrillo et al (2015) demonstrates that CTT can be used to identify descriptive features such as the identification of underlying dimensions (as well as pruning redundant items) which can then be consolidated by MTT to provide a more applied consideration for the development of a patient-rated outcome measure using item difficulty and DIF techniques. Indeed, both frameworks can be used to improve the content validity of the PROM by determining whether the scale and items both align with the patient population (Cappalleri, Lundy, and Hays, 2014).

Models of recovery now focus on the improvement of functional impairments as opposed to a singular improvement of symptomology (Leamy et al, 2011). This resonates with meta-analytical findings of Chapter 2, as functional impairments and symptomatology appear to be orthogonal, and other clinically relevant variables such as age, duration of illness, comorbid disorders, and medication-use also failed to predict changes in task performance effect sizes suggesting that these functional impairments reside outside of the current diagnostic scope of OCD and may not be challenged by current treatment methods (Eisen et al, 2013). This implicates functional impairment as preventing successful symptom remission and thereby potentially underlying relapse rates (Cavedini et al, 2002). This evidences the importance of their recognition within PROMs as they can be used to devise novel treatment paths (Edlund et al, 2018; East-Richard et al, 2020). Nonetheless, PROMs suited to the assessment of impaired functioning are certainly lacking (Baandrup et al, 2021) and may also reflect the scarce acknowledgement of this issue within the OCD literature. Stilley et al (2010) showed that cognitive inflexibility (and other impaired domains of executive functioning) was a

predictor of treatment adherence as those with greater executive impairments were associated with higher rates of treatment drop out.

As reported by Inchausti and Delgado (2012), most scales assessing OCD phenomenology have only received limited psychometric validation (developed under CTT) with only a single measure having undergone rigorous validation and quality control analyses under MTT, the Maudsley Obsessive-Compulsive Inventory (MOCI; Hodgson and Rachman, 1977). A measurement attuned to the more functionally impaired portions of OCD phenomenology, that has undergone rigorous psychometric testing, is required as it is not currently assessed or sufficiently acknowledged by pre-existing measurements or tools.

STUDY 4.1: INITIAL VALIDATION OF THE OCPAS

Using both classical and modern test theoretical frameworks (Exploratory factor analysis and the Rasch model), the aim of this study was to develop a patient-rated outcome measure of OCD phenotypic variance that is inclusive of neurocognition and latent cognitive phenotypes. As patients with OCD are one singular point on a spectrum ranging from symptoms to syndrome to illness, developing a scale that is too close to the diagnosable illness may have eliminated salient pre-clinical features that may elucidate predisposing and precipitant factors at particular junctures of severity; as such a ‘bottom-up’ approach was employed to construct this new scale. We, therefore, hypothesise that the multifactorial nature of OCD phenomenology will be confirmed by the factor structure of this prototype scale.

METHOD

This investigation was preregistered (Open Science Framework; https://osf.io/jwv8x/). This research is reported according to the Checklist for Reporting Results of Internet E-Surveys (CHERRIES, Eysenbach, 2004; 2012).

Materials and survey construction

To construct a comprehensive self-report measure of OCD phenomenology, discussions were held among a multidisciplinary group of experienced OCD clinicians (LD, NF, LP), researchers (KL, AC), and an expert through the lived experience of OCD (MZ), resulting in the following phenotypes being considered: obsessive symptoms, compulsive symptoms, motor impulsivity, decision making impulsivity, indecisiveness, executive functioning, set-shifting/perseveration, habit, perfectionism, need for control, rigidity, need for certainty, conscientiousness/responsibility, just-right perceptions, miserliness, hoarding, tics, mood/fatigue, disgust/sensations, central coherence, theory of mind, and moral judgement.

Next, nine validated instruments were reviewed for their estimation and assessment of the above list of obsessive-compulsive and related phenomena. These include: the Yale-Bown Obsessive-Compulsive Scale (Y-BOCS; Goodman et al, 1989), the Obsessive-Compulsive Inventory-Revised (OCI-R; Foa et al, 2002), the Padua Inventory (PI; Sanavio, 1988; Burns, 1995), the Compulsive Personality Assessment Scale (CPAS; Fineberg et al, 2007), the Cognitive Assessment Instrument of Obsessions and Compulsions (CAIOC-13; Dittrich, Johansen, and Fineberg, 2011), the Cambridge-Chicago Trait Scale (CHI-T; Chamberlain and Grant, 2018), the Autism Spectrum Quotient (AQ-10; Allison, Auyeung, and Baron-Cohen, 2012), the Creature Of Habit Scale (COHS; Ersche et al, 2017), and the Obsessive-Compulsive Trait Core Dimension Questionnaire (OCTCDQ; Summerfeldt et al, 2001). This consolidation of current assessment tools provided a standardised method of highlighting

areas that have been somewhat neglected and avoided the inclusion of arbitrary items or those that have been covered elsewhere in preexisting scales. From a total of 169 items in these pre-existing scales, 24 items noted to assess the relevant OC phenomena were taken and adapted for use in this new scale (see Table 4.1.1).

Table 4.1.1. Sources of initial 59-item corpus.

Symptom	Scale	Item
Obsessive symptoms		
	CHIT	I get stuck thinking about one thing
	Bespoke	I am troubled by unwanted thoughts that repeat in my mind
	Bespoke	I am effective in stopping or diverting unwanted repetitive thoughts (Reversed scored)
Compulsive symptoms		
	Bespoke	I am troubled by having to repeat behaviours that seem excessive or pointless
	Bespoke	I am effective in stopping or diverting unwanted behaviours (Reversed scored)
	Bespoke	I am troubled by having to repeat mental acts that seem excessive or pointless
	Bespoke	I am effective in stopping or diverting unwanted urges (Reversed scored)
Motor impulsivity		
	Bespoke	I frequently act on impulses
	Modified from the CHIT	I resist acting on urges (Reversed scored)
	Bespoke	Once I have started an action I find it hard to stop
Decision-making impulsivity		
	CHIT	I do things that seem rewarding at the time even if I know they are bad for me in the long run
Indecisiveness		
	CAIOC-13	I have difficulty making decisions about everyday matters
Executive functioning		
	Y-BOCS	I have difficulty getting started on new tasks
	Y-BOCS	I have difficulty finishing tasks that I have started
	Y-BOCS	Routine activities take longer than they should
	CAIOC-13	I have difficulty organising and carrying out a planned task e.g., a holiday, shopping trip or a birthday celebration
	Bespoke	I get stuck when performing routine tasks
Set-shifting/Perseveration		
	AQ-10	If there is an interruption, I can switch back to what I was doing very quickly (Reversed scored)
	CHIT	I find it hard moving from task to task
	Bespoke	I find it hard to manage unexpected changes to my routine
Habit		
	CHIT	I am a creature of habit
	Modified from the COHS	I tend to do things according to a routine (e.g., eating meals at a specific time, using the same utensils, sitting in a specific seat etc).
Perfectionism		

	CAIOC-13	I feel the need to have things perfect
	CAIOC-13	I doubt having done things properly
	Bespoke	I am troubled when things are not complete e.g., missing items in a set
	Bespoke	I am disturbed by small errors or imperfections
Need for control		
	Bespoke	Others describe me as someone who needs to feel in control
	Bespoke	I like to be in control or else I feel upset
	Bespoke	I find it easy to trust others to carry out tasks the way they ought to be done (Reversed scored)
Rigidity		
	CHIT	People have told me I can be stubborn or rigid
	Bespoke	I can tolerate breaking a rule (Reversed scored)
	Bespoke	I can be rigid
	Bespoke	I can be flexible or easy going (Reversed scored)
	Modified from the OCTCDQ	I tend to have rigid views about how things look or have to be
	Bespoke	I prefer all or nothing decisions
	Bespoke	I feel uncomfortable if I do not get my own way
	Bespoke	I am happy to compromise (Reversed scored)
Need for certainty		
	Bespoke	I find uncertainty troubling
	Modified from the PI	When I read, I sense I may have missed something and must go back and reread the passage
Conscientiousness/ Responsibility		
	Modified from the Y-BOCS	I tend to feel guilty when things go wrong even when it is not my fault
	Bespoke	I shoulder responsibility beyond what could be reasonably expected
Just-right perceptions		
	Modified from the OCTCDQ	I try to do things (i.e., activities, tasks, or movements) until they feel "just right"
	Bespoke	When something I have done does not feel quite right, I need to repeat it until it does
Miserliness		
	Bespoke	I have difficulty spending money especially when it is on myself or a loved one
	Modified from the CPAS	I see money as something I should save for future possible needs
Hoarding		
	Bespoke	I have strong urges to acquire things when I don't really need them.
	Bespoke	I hoard things
	OCI-R	I avoid throwing things away because I am afraid I might need them later.
Tics		
	Bespoke	I perform unnecessary physical or vocal actions e.g., muscle twitching, coughing, sniffing, throat clearing, saying certain words
	Bespoke	Others have commented that I make noises or movements e.g., sniffing, humming, grunting
Mood/Fatigue		
	CAIOC-13	I lack energy, motivation or drive
	Bespoke	I feel mentally exhausted

Disgust/Sensation		
	Bespoke	I feel disgust in the presence of bodily secretions e.g., blood, sweat, urine, faeces
	Bespoke	Certain foods, textures, and smells easily disgust me
Central Coherence		
	CPAS	I get preoccupied with details
	AQ-10	I usually concentrate more on the whole picture, rather than the small details (Reversed scored)
Theory of Mind		
	Bespoke	I am good at working out how people are thinking and feeling even when they do not tell me (Reversed scored)
Judgemental		
	Bespoke	I take strong positions on things when I believe they are right or wrong
	Bespoke	Others see me as being hyper-critical

For those remaining OCD phenotypes that were not sufficiently covered by the pre-existing scales, a further 35 items were created 'de novo' and reviewed for interpretability and accessibility by expert members of the research team (NF, LD, KL, MZ, and LP) following a series of iterative discussions.

The resulting self-report measure had a total of 59 items, 12 of which were reversed scored to encourage attentive responses and reduce response bias. The order of the 59 items was randomised to reduce order effects (see appendix Q for original 59 items). The scale was presented to participants using Gorilla, a behavioural experiment building platform (Anwyl-Irvine et al, 2020).

Participants were instructed that "The below statements refer to impulsive and compulsive tendencies and behaviours. Please rate these statements on how relevant they are to you. For example, if a statement is not applicable to yourself, please select "Not at all", whereas, if something has great applicability to you, then select "very much". Ratings were made on a 5 point Likert style scale: "Not at all", "A little", "Quite a lot", "A lot", and "Very much".

Sample

Our sample consisted of healthy participants from the UK and USA and were recruited through Prolific (Palan and Schitter, 2018). A ratio of 10 participants to 1 item was followed (Everitt, 1975) and so, for 59 items, a total of 590 participants aged 18 and above (mean age 40.42; SD = 13.28) completed the study. This also surpassed the recommendations of Cappalleri, Lundy, and Hays (2014) for a minimum of 500 participants to satisfy psychometric validation of a patient-rated outcome measure.

A priori criteria ensured a balanced sample based on gender (295 males and 295 females). Sociodemographic information is presented in Appendix R.

Procedure

Participants were informed of all aims and intentions of the research, their role as a participant and their right to withdraw. Participants were required to provide written digital consent should they wish to participate. They were initially asked questions about demographic factors, including: age, sex, ethnicity, level of education, faith group, and personal and familial histories of mental health disorders. They were then asked to complete all 59 scale items, responding to each item by choosing the response that best fitted their experience of themselves. Once they had completed the scale, respondents were thanked for their participation and reminded of their right to withdraw. A completion code was given to participants to receive payment for their participation (via the Prolific platform).

Statistical analysis

Given the limitations of self-report data and incentivised participation (Singer and Couper, 2008; Chan, 2009), quality control checks were employed to ensure valid and reliable responses. Reverse scored items were included within the prototype scale and so consistent irregularities on these items resulted in exclusions. Moreover, as the prototype takes approximately 3-4 minutes to complete, responses that completed the scale significantly quicker or slower were also excluded from analyses. All participants satisfied these checks.

An exploratory factor analysis of responses was conducted using maximum likelihood method of extraction; the rotation method was ProMax as the items are conceptually related and are thus likely to correlate. As outlined in the Cattell method (1966), the scree plot was examined for Eigenvalues above a value of 1.00 to identify the number of underlying factors. A parallel analysis was conducted to determine the number of factors to retain (Horn, 1965). Factor loadings above .40 were suitable for inclusion. This analysis was performed in JASP (version 18; JASP team, 2023) using the descriptives, frequencies, factor, and reliability modules.

Following factor analysis, Winsteps (version 5.4.3.0; Linacre, 2023) was used to perform a further series of analyses under the Rasch model of scale assessment. Response bias was assessed via person and item reliability checks. The benchmark for acceptable reliability was considered when values are equal to or exceed .70 (Bland and Altman, 1997). Following recommendations for Mean Square Scores (MNSQ), logits between 0.5 and 1.5 were considered appropriate for testing (Linacre, 1994; Smith, Schumacker, and Bush, 1998). A threshold analysis was performed to assess the functioning of the rating scale (Bond and Fox, 2001a). Items were assessed for their fit within the factors revealed by the exploratory factor analysis via an item fit analysis. Similarly, Wright maps were consulted to assess the relative difficulty of interpretating the scale items correctly and answering accordingly. The relative

difficulty of items were considered along a scale of -3.00 to 3.00 logits; items of particular difficulty (greater than 1.00 standard deviation) were removed from the scale to maintain consistency (Bond and Fox, 2001b; Boone, 2016). A series of Differential Item Functioning (DIF) analyses were performed to examine any potential bearing of demographic variables on scale responses to identify potential selection biases and were removed accordingly (Holland and Wainer, 1993; Penfield and Lam, 2005). Items were similarly removed for underfitting or overfitting on infit and outfit statistics revealing the presence of anomalous data (Linacre, 2002).

RESULTS

Exploratory factor analysis

The minimum factor loading considered acceptable for interpretation was .40 (Stevens, 2002). Eigenvalues that exceeded the parallel analysis were used to interpret the number of factors to retain (Horn, 1965). The 59 items in the questionnaire showed a Kaiser-Meyer-Olkin value of .938 indicating that the variance in the data may be attributable to underlying factors thereby necessitating a factor analysis (Kaiser, 1974). Bartlett's sphericity was assessed ($X^2(1711) = 17721.63$, $p<.001$) indicating a relationship between extractible factors (Bartlett, 1951).

As shown in Table 4.1.2, the simulated data eigenvalues exceeded the actual data eigenvalues after Factor 7. As Factor 7 comprised only 2 items, this factor was removed as a minimum of 3 items is required to substantiate a factor (Child, 2006; Izquierdo, Olea, and Abad, 2014) and so Factors 1 through 6 were retained in this model. As depicted in Table 4.1.3 and appendix Q (Scree plot), the factor analysis revealed six factors, accounting for 44.10% of the total

variance. Fourteen items (items 1, 6, 16, 24, 25, 28, 29, 34, 35, 45, 47, 48, 50, and 57) did not load on any factors and were excluded (see appendix S for these items). The factor correlations are reported in Table 4.1.4.

Table 4.1.2. Actual and parallel analysis for factor retention.

	Real data eigenvalues	**Simulated data eigenvalues**
Factor 1	16.04	1.67
Factor 2	4.02	1.62
Factor 3	3.38	1.58
Factor 4	2.21	1.53
Factor 5	1.86	1.51
Factor 6	1.71	1.47
Factor 7	1.47	1.44
Factor 8	1.33	1.41

Footnote: 8th factor included in table 3 to illustrate retention threshold.

Factor loading patterns

Table 4.1.3. Factor loadings

Items	**Factor 1**	**Factor 2**	**Factor 3**	**Factor 4**	**Factor 5**	**Factor 6**	**Factor 7**
Item 12: I have difficulty finishing tasks that I have started.	0.96						
Item 5: I have difficulty getting started on new tasks.	0.91						
Item 32: I have difficulty organising and carrying out a planned task e.g., a holiday, shopping trip or a birthday celebration.	0.85						
Item 11: I have difficulty making decisions about everyday matters.	0.84						
Item 21: Routine activities take me longer than they should.	0.82						
Item 44: I find it hard moving from task to task.	0.82						
Item 31: I lack energy, motivation or drive.	0.82						
Item 41: I get stuck when performing routine tasks.	0.69						

Item 52: I feel exhausted.	0.61				
Item 22: I doubt having done things properly.	0.59				
Item 51: I avoid getting rid of things because I am afraid that I might need them later.	0.43				0.54
Item 39: When something I have done does not feel quite right, I need to repeat it until it does.		0.86			
Item 26: I try to do things (i.e., activities, tasks, or movements) until they feel "just right".		0.75			
Item 20: Once I have started an action I find it hard to stop.		0.73			
Item 46: I am troubled when things are not complete e.g., missing items in a set.		0.60			
Item 2: I am troubled by having to repeat behaviours that seem excessive or pointless .		0.58			
Item 30: I perform unnecessary physical or vocal actions e.g., muscle twitching, coughing, sniffing, throat clearing, saying certain words.		0.51			
Item 19: I am troubled by having to repeat mental acts that seem excessive or pointless.		0.50			
Item 54: Others have commented that I make noises or movements e.g., sniffing, humming, grunting.		0.49			
Item 55: I am disturbed by small errors or imperfections.		0.46			
Item 53: I tend to do things according to a routine (e.g., eating meals at a specific time, using the same utensils, sitting in a specific seat etc).		0.42			
Item 7: Others describe me as someone who needs to feel in control .			0.72		
Item 14: People have told me I can be stubborn or rigid.			0.64		
Item 23: I like to be in control or else I feel upset.			0.58		
Item 18: I take strong positions on things when I believe they are right or wrong.			0.56		
Item 36: Others see me as being hyper-critical.			0.50		
Item 33: I can be rigid.			0.49		

Item 42: I tend to have rigid views about how things look or have to be.			0.43				
Item 15: I find uncertainty troubling.			0.43				
Item 49: I get preoccupied with details.			0.41				
Item 13: I feel the need for things to be perfect.			0.40				
Item 58: I am happy to compromise (reverse scored).				0.66			
Item 38: I can be flexible or easy going (reverse scored)				0.65			
Item 59: I usually concentrate more on the whole picture, rather than the small details (reverse scored)				0.52			
Item 56: I find it easy to trust others to carry out tasks the way they ought to be done (reverse scored				0.50			
Item 17: I am effective in stopping or diverting unwanted repetitive thoughts (reverse scored)				0.42		0.57	
Item 37: If I am interrupted, I can quickly switch back to what I was doing (reverse scored).				0.40			
Item 3: I frequently act on impulse.					0.65		
Item 4: I do things impulsively that seem rewarding at the time even if I know they are bad for me in the long run.					0.64		
Item 10: I resist acting on urges (reverse scored).					0.63		
Item 9: I am effective in stopping or diverting unwanted behaviours or urges (reverse scored).					0.45		
Item 40: I see money as something I should save for future possible needs.					-0.43		
Item 27: I am effective in stopping or diverting unwanted thoughts (reverse scored).						0.62	
Item 8: I am troubled by unwanted thoughts that repeat in my mind.						0.44	
Item 43: I can easily get rid of things that I do not need (reverse scored).							0.61
Eigenvalues	16.04	4.02	3.38	2.21	1.86	1.71	1.47
% of variance	26.30	5.90	4.80	2.90	2.20	2.00	1.60

Factor 1 – Cognitive control.

Factor 1 consisted of 11 items (items 12, 5, 32, 11, 21, 44, 31, 41, 52, 22, and 51). These items measure attentional inflexibility and deficient executive functioning (e.g., '*I find it hard moving from task to task'*) and was thus labelled Cognitive control. Reliability analysis showed excellent internal consistency for Factor 1 ($\alpha = 0.92$; $\omega = .92$).

Factor 2– Behavioural control

Factor 2 is populated with 10 items (items 39, 26, 20, 46, 2, 30, 19, 54, 55, and 53) describing a lack of control over behavioural and emotional output and subsequent ceasing of the behaviour once started (e.g., '*once I have started an action, I find it hard to stop*' and '*I am troubled by having to repeat behaviours that seem excessive or pointless*') and was named Behavioural control. Reliability analysis revealed good internal consistency for Factor 2 ($\alpha = 0.86$; $\omega = .86$).

Factor 3 – Anankastic goals.

Factor 3 contains 10 items (items 7, 14, 23, 18, 36, 33, 42, 15, 49, and 13) which are consistent with the ICD-11 classification of the anankastic trait domain (WHO, 2022b). This factor was punctuated with items depicting rigid beliefs, a need for control, and a motivational drive to achieve perfectionist standards (e.g., '*I tend to have rigid views about how things look or have to be';'I feel the need for things to be perfect*'). This factor was labelled Anankastic goals. Internal consistency for Factor 3 was revealed to be good ($\alpha = 0.86$; $\omega = .86$).

Factor 4 – Proportionality.

Factor 4 is comprised of 5 items (items 58, 38, 59, 56, and 37). Each item in this factor is reversed scored and thus the description of these items reflect normative functioning and central coherence (e.g., '*If I am interrupted, I can quickly switch back to what I was doing'* and *'I usually concentrate more on the whole picture, rather than the small details'*) and was named proportionality. Reliability analysis revealed acceptable internal consistency ($\alpha = 0.71$; $\omega = .71$).

Factor 5 – Impulsivity.

Factor 5 is comprised of 5 items (items 3, 4, 10, 9, and 40). These items center upon themes of urgent action without consideration for consequence, motivated by situational influences (e.g., '*I frequently act on impulse*' and '*I do things impulsively that seem rewarding at the time even if I know they are bad for me in the long run*') and was labelled Impulsivity. Internal consistency was revealed to be poor ($\alpha = 0.51$; $\omega = .61$). Although thematic similarities exist between impulsivity and behavioural control, they are in fact different constructs. Behavioural control is a general trait defined by deficient control over the stopping and starting of behavioural actions, whilst impulsivity is more reactionary to mood states (Joyner et al, 2021).

Factor 6 – Unwanted thoughts.

Factor 6 consists of 3 items (items 17, 27, and 8). The items describe the distress caused by intrusive thoughts and the inability to control or suppress these thoughts, notions, and beliefs (e.g., 'I *am effective in stopping or diverting unwanted repetitive thoughts*' and '*I am troubled by unwanted thoughts that repeat in my mind*') and was named Unwvanted thoughts. Internal consistency was acceptable ($\alpha = 0.79$; $\omega = .80$).

Table 4.1.4. Factor correlations.

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5
Factor 1	-				
Factor 2	0.66	-			
Factor 3	0.49	0.63	-		
Factor 4	0.30	0.24	0.19	-	
Factor 5	0.28	0.06	-0.13	0.03	-
Factor 6	0.19	0.18	0.31	0.21	-0.05

Rasch analysis

Following factor analysis, a multidimensional Rasch analysis was conducted using Winsteps software (version 5.4.3.0; Linacre, 2023) to further validate the scale.

To assess if the rating scale was working in the intended way, a threshold analysis was computed. The category measure values increased monotonically, indicating that the 5-point rating scale is functioning in the intended way (-2.51, -0.96, -0.01, 0.96, 2.54).

Person Fit

Individual person fit was analysed using Infit and Outfit statistics to indicate how well data conformed to the Rasch model.

The overall person reliability score was revealed to be 0.92 suggesting a sufficient degree of reliability; Infit Mean Square (MNSQ) = 1.03, Outfit MNSQ = 1.03 was also within the expected threshold, (Linacre, 1994; Bond and Fow, 2001c; Smith, Schumacker, and Bush, 1998). Item reliability was 0.99 (Infit MNSQ = 1.02, Outfit MNSQ = 1.03). Table 5 shows a breakdown of person fit analysis for the separate latent factors.

As can be seen in Table 4.1.5, all latent factors of the rating scale satisfy the expected values for infit and outfit MNSQ. The item separation values for Factors 4, 5, and 6 are below threshold where separation values should be 2.00 or higher (Linacre, 2003). Similarly, the

reliability of Factors 4 and 5 are also low; a value of 0.80 or greater denotes sufficient reliability (Bond and Fox, 2001b).

Table 4.1.5. Person fit analysis.

Factor	Person Reliability	Separation	INFIT MNSQ	OUTFIT MNSQ
1 Cognitive control	0.88	2.67	1.03	1.01
2 Behavioural control	0.81	2.04	1.00	1.01
3 Anankastic goals	0.83	2.18	1.00	1.00
4 Proportionality	**0.71**	**1.56**	0.99	1.00
5 Impulsivity	**0.49**	**0.97**	1.03	1.02
6 Unwanted thoughts	0.79	**1.92**	0.97	0.99

*Note: Details in **Bold** indicate values below threshold. All latent factors of the rating scale satisfy the expected values for infit and outfit MNSQ.*

Differential Item Functioning

Table 4.1.6 depicts the differential item functioning (DIF). A difference larger than or equal to 0.5 logits (Wang et al, 2006) was taken as a sign of substantial DIF. Item 11 was shown to be influenced by gender in that females scored higher than males. No other DIF effect emerged for items on any other examined demographic variables. Moreover, as values for MNSQ are to be between 0.5 and 1.5 (Linacre, 1994), item 40 overfitted on infit and outfit MNSQ's, item 51 overfitted on infit and outfit MNSQ's, and item 54 overfitted on infit MNSQ.

Table 4.1.6. Maximum differences in the estimates for differential item function (DIF) over demographic variables, infit and outfit MNSQ statistics

Factor	Item Number	Total score	Gender	Faith Group	Education	PHofMHD	FHofMHD	INFIT MNSQ	OUTFIT MNSQ
1 Cognitive control	41	517	0.09	0.36	0.30	0.03	0.36	0.90	0.83
	32	598	0.07	0.12	0.18	0.00	0.25	1.03	0.91
	44	602	0.33	0.26	0.17	0.11	0.14	0.84	0.84
	21	630	0.38	0.36	0.22	0.05	0.05	0.95	0.88
	11	665	0.51*	0.07	0.21	0.03	0.03	0.93	0.89
	12	788	0.26	0.01	0.25	0.15	0.04	0.78	0.79
	22	892	0.04	0.07	0.01	0.01	0.00	1.05	1.07
	31	932	0.19	0.01	0.00	0.02	0.18	0.95	0.91
	5	951	.010	0.36	0.36	0.25	0.16	0.83	0.86
	51	983	0.16	0.01	0.07	0.01	0.02	**1.75**	**1.97**
	52	1106	0.01	0.16	0.32	0.16	0.08	1.15	1.18
2 Behavioural control	54	348	0.08	0.11	0.50	0.22	0.04	**1.59**	1.30
	30	455	0.03	0.15	0.27	0.39	0.08	1.30	1.07
	2	693	0.02	0.07	0.06	0.07	0.08	1.10	1.13
	20	714	0.22	0.34	0.02	0.16	0.26	0.80	0.82
	55	768	0.13	0.34	0.31	0.23	0.24	0.68	0.69
	19	777	0.20	0.05	0.27	0.02	0.09	1.10	1.11
	39	889	1.40	0.06	0.07	0.03	0.01	0.77	0.77
	53	933	0.22	0.16	0.05	0.06	0.06	1.25	1.25
	46	980	0.14	0.08	0.15	0.39	0.19	0.91	0.92
	26	1056	0.4	0.10	0.10	0.41	0.00	0.84	0.82

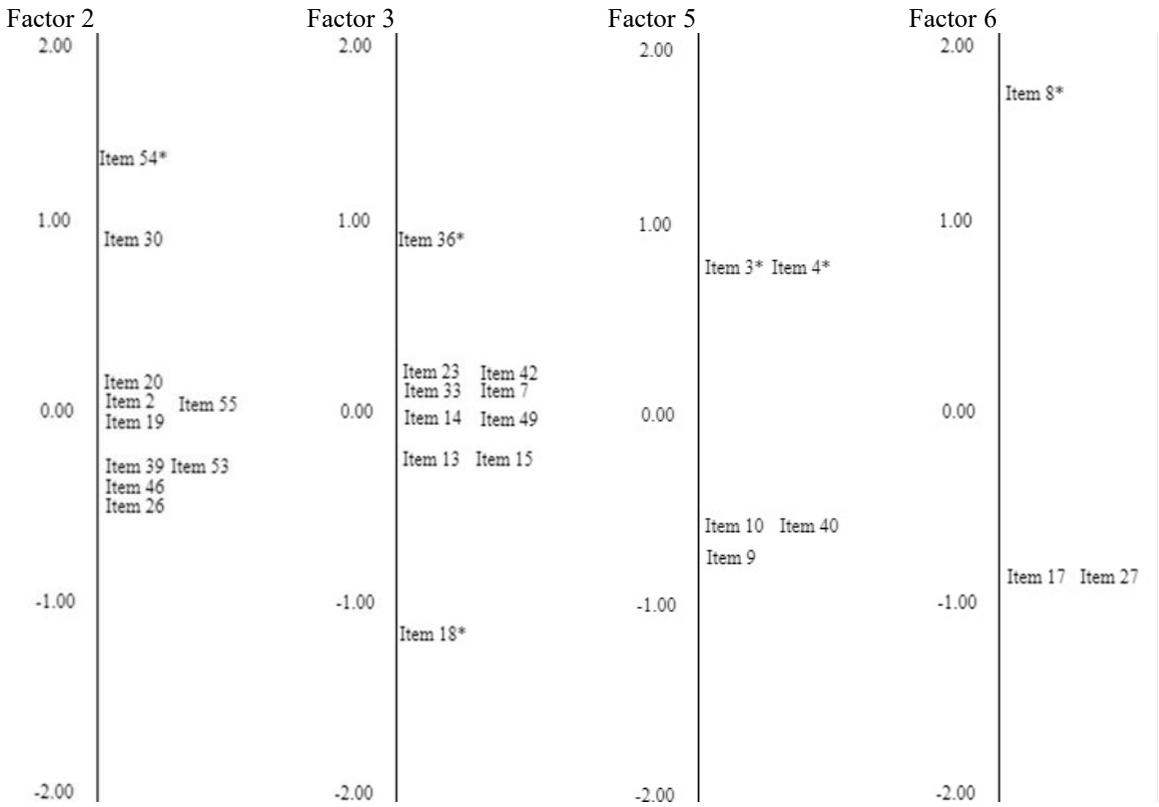
3 Anankastic goals	36	693	0.31	0.01	0.12	0.10	0.20	1.14	1.08
	42	831	0.19	0.08	0.08	0.00	0.07	0.81	0.78
	23	838	0.04	0.23	0.15	0.16	0.19	0.92	0.89
	33	855	0.07	0.12	0.11	0.12	0.08	0.74	0.74
	7	873	0.11	0.00	0.06	0.16	0.17	1.15	1.13
	14	1000	0.08	0.19	0.27	0.34	0.14	1.02	1.02
	49	1007	0.19	0.22	0.23	0.02	0.24	0.84	0.85
	13	1180	0.07	0.29	0.11	0.13	0.05	1.18	1.15
	15	1183	0.41	0.08	0.12	0.25	0.01	1.05	1.05
	18	1468	0.15	0.21	0.27	0.11	0.05	1.19	1.30
4 Proportionality	38	1111	0.01	0.11	0.12	0.29	0.27	0.89	0.88
	58	1169	0.00	0.14	0.13	0.14	0.16	0.79	0.80
	59	1302	0.04	0.01	0.07	0.12	0.07	1.01	1.00
	37	1333	0.05	0.07	0.06	0.05	0.21	1.10	1.08
	17	1385	0.16	0.10	0.17	0.22	0.26	1.11	1.12
	56	1500	0.14	0.02	0.00	0.24	0.16	1.14	1.12
5 Impulsivity	3	741	0.08	0.09	0.07	0.12	0.10	0.71	0.71
	4	764	0.15	0.23	0.10	0.08	0.22	0.76	0.76
	10	1256	0.02	0.15	0.17	0.10	0.15	0.66	0.66
	40	1276	0.10	0.02	0.02	0.02	0.03	**2.01***	**2.21***
	9	1299	0.05	0.37	0.08	0.06	0.18	0.77	0.77
6 Unwanted thoughts	8	850	0.10	0.03	0.18	0.04	0.03	1.40	1.50
	17	1395	0.25	0.08	0.05	0.33	0.21	0.78	0.83
	27	1391	0.12	0.12	0.22	0.42	0.27	0.67	0.71

Note: PHofMHD = Personal history of mental health disorder; FHofMHD = Familial history of mental health disorder. Demographic variables with multiple levels were collapsed into dichotomous variables for DIF analysis: Gender: 1 = Males, 2 = Females; Faith groups: 1 = present, 2 = absent; PHofMHD: 1 = present, 2 = absent; FHofMHD: 1 = present, 2 = absent. Education: 1 = school education; 2 = higher education. Those in **Bold** over-fitted on infit and outfit MNSQs. Those with '*' showed substantial differential item functioning.

Item difficulty

Item difficulty was assessed to determine the relative difficulty of interpretating and answering of the scale items. Scale items were distributed along a logit scale to observe the difficulty of the scale items relative to each other (Stout et al, 2012; Boone, 2016); item difficulty analyses were performed and assessed separately for each factor (see Figure 4.1.1). In factor 2 (*Behavioural control*), Item 54 showed to be greater than nearly 1.5 standard deviations compared to other items in this factor. In factor 3 (*Anankastic goals*), item 18 sits below –1.00 logits and is 1 standard deviation lower than most items in this factor and item 36 sits at 1.00 logits, whilst all other items in this factor cluster at 0.00 logits. In factor 5 (*Impulsivity*), items 3 and 4 sit below 1 logit and are 1.5 standard deviations away from other items in this factor. In factor 6 (*Unwanted thoughts*), item 8 sits below 2 logits and is 2 standard deviations away from items 17 and 27.

Figure 4.1.1. Wright maps for Factors 2, 3, 5 and 6.



*Note: *=removed items.*

Item removals

Table 4.1.7 shows the items removed from each factor and the statistical rationale.

Table 4.1.7. Items removed.

Factor	Item	Reasons
1	11	Substantial DIF (see table 6, row 5).
	51	Overfitted in INFIT and OUTFIT (see table 6, row 10).
2	54	Item difficulty (see Figure 1), overfitted in INFIT (see table 6, row 12).
3	18	Item difficulty (see Figure 1).
	36	Item difficulty (see Figure 1).
4	**all**	The whole factor was removed – Person fit and separation scores were below threshold (see Table 2, row 4).

5	**all**	The whole factor was removed – Person fit and separation scores were below threshold (see Table 2 row 5); items 3&4 item difficulty (see Figure 1); Item 40 overfitted in INFIT and OUTFIT (see table 6 row 41). This left item 9 and 10; a factor with just two items is insubstantial
6	**all**	The whole factor was removed – separation scores were below. threshold (see Table 2, row 6); item 8, item difficulty (see Figure 1). This left item 17 and 27; a factor with just two items is insubstantial.

Corrected scale and final analyses

Table 4.1.8. Revised scale and factors.

Factor 1 – Cognitive control (α=0.92;ω=.92)	
Item 12	I have difficulty finishing tasks that I have started.
Item 5	I have difficulty getting started on new tasks.
Item 32	I have difficulty organising and carrying out a planned task e.g., a holiday, shopping trip or a birthday celebration.
Item 21	Routine activities take me longer than they should.
Item 44	I find it hard moving from task to task
Item 31	I lack energy, motivation or drive.
Item 41	I get stuck when performing routine tasks.
Item 52	I feel exhausted.
Item 22	I doubt having done things properly.
Factor 2 – Behavioural control (α=0.85;ω=.85)	
Item 39	When something I have done does not feel q0075ite right, I need to repeat it until it does.
Item 26	I try to do things (i.e., activities, tasks, or movements) until they feel "just right".
Item 20	Once I have started an action I find it hard to stop.
Item 46	I am troubled when things are not complete e.g., missing items in a set.
Item 2	I am troubled by having to repeat behaviours that seem excessive or pointless.
Item 30	I perform unnecessary physical or vocal actions e.g., muscle twitching, coughing, sniffing, throat clearing, saying certain words.
Item 19	I am troubled by having to repeat mental acts that seem excessive or pointless.
Item 55	I am disturbed by small errors or imperfections.
Item 53	I tend to do things according to a routine (e.g., eating meals at a specific time, using the same utensils, sitting in a specific seat etc).
Factor 3 – Anankastic goals (α=0.84; ω=.84)	
Item 7	Others describe me as someone who needs to feel in control.
Item 14	People have told me I can be stubborn or rigid.
Item 23	I like to be in control or else I feel upset.
Item 33	I can be rigid.
Item 42	I tend to have rigid views about how things look or have to be.
Item 15	I find uncertainty troubling.
Item 49	I get preoccupied with details.
Item 13	I feel the need for things to be perfect.

Table 4.1.8 shows the refined scale following the described analyses and removals with excellent internal consistency (ω=.93). Following the item exclusions, a second threshold analysis was performed. Category measure values continue to increase monotonically indicating that the 5-point rating scale is functioning in the intended way (-2.64, -1.01, 0.00, 1.02, 2.63). Factor correlations are presented in Table 4.1.9.

Table 4.1.9. Factor correlations of final scale model.

	Factor 1 Cognitive control	Factor 2 Behavioural control
Factor 1 Cognitive control	--	
Factor 2 Behavioural control	0.57	--
Factor 3 Anankastic goals	0.52	0.67

Footnote: all $p<.001$

Exploratory analyses

No significant differences were observed between those with and without a personal history of mental health disorders (see Table 4.1.10). Similarly, those with a familial history of mental health disorders did not significantly differ from those reporting no familial history. Of the 261 participants reporting a personal history of mental health disorders, 166 (63.60%) also reported a familial history of mental health disorders. A significant moderate association was revealed between personal and familial histories ($\chi^2 = 126.74$, $\varphi = .46$, $p<.001$).

Table 4.1.10. Subscale and total scores by histories of mental health disorders.

	Factor 1 Cognitive control	Factor 2 Behavioural control	Factor 3 Anankastic goals	Total score
Personal Hx of MHD				
Personal Hx (n=261)	12.06 (8.39)	12.50 (6.44)	13.03 (6.00)	50.83 (19.21)
No personal Hx (n=329)	11.76 (8.09)	12.31 (7.19)	13.27 (6.62)	50.54 (20.57)
Familial Hx of MHD				
Familial Hx (n=226)	12.17 (8.25)	12.51 (6.38)	13.30 (5.87)	51.39 (18.64)
No familial Hx (n=364)	11.72 (8.21)	12.32 (7.15)	13.08 (6.62)	50.23 (20.93)

Footnote: Data presented as Mean (Standard deviation). Hx = History, MHD = mental health disorders.

DISCUSSION

The factor structure of OCD phenomenology

The aim of this research was to develop a comprehensive self-report measure – the Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS) – to appraise the phenotypic variance observed in obsessive-compulsive phenomenology. The initial approach, using exploratory factor analysis reduced the initial pool of 59 items to a 44-item scale loading on 6 factors. Following this, Rasch analysis was employed for polytomous data and the analyses resulted in a final corpus of 26 items across three factors. The factors of OCD phenomenology were identified as: *Cognitive Control*, *Behavioural Control*, and *Anankastic Goals*. These three latent factors were moderately intercorrelated.

Cognitive control, which accounted for the largest amount of variance in responding (26%), typically refers to the diminished ability to effectively use cognition and behaviours to achieve goals and objectives; and the items in this factor encapsulate key aspects of task management and executive functioning. A wealth of literature reports deficient control and flexibility in task performance in patients with OCD (for a recent meta-analysis, see Clarke et al, 2024). Research does show that patients diagnosed with OCD score worse than healthy controls on self-rating of flexibility and control over thoughts and behaviours (the Cognitive Assessment Instrument of Obsessions and Compulsions (CAIOC-13: Dittrich, Johansen, and Fineberg, 2013) and the Compulsive Personality Assessment Scale (CPAS; Fineberg et al, 2007) indicating greater inflexibilities and deficits in goal-directed behaviour in OCD.

Impaired task management impacts people with OCD, with daily activities often taking much longer to complete and being related to greater OCD symptom severity (see Zisler et al 2024). Zisler and colleagues show that obsessions and compulsions interfere with efficient task performance and that the striving for control results in tasks being prolonged and perpetuated. Moritz, Hubner, and Kluwe (2010) show that this issue with cognitive control also links to difficulties in switching between tasks. To date, measures assessing compulsivity

have neglected the affective consequences of such cognitive inflexibility and impaired task management. As described in items 31 and 52 ("*I lack energy, motivation or drive*" and "*I feel exhausted*"), an element of mental fatigue and exhaustion also appears to be associated with the striving for control over repetitive thoughts and actions and is reflected in a recent review of mental fatigue in psychiatric disorders, including OCD (Mozuraityte et al 2022).

Behavioural control emerged as the second factor on the OCPAS. This factor largely identifies the failure to inhibit pointless actions and behaviours, which clearly occurs in people with OCD (Rosenberg et al, 1997; Krikorian, Zimmerman, and Fleck, 2004; Joyner et al, 2021). Like inflexibility, behavioural control is transdiagnostic in nature but when considered together, have been shown to account for OC-related problems (Tiego et al, 2018). Previous research has denoted an association between self-rated scores of OCD symptom severity, behavioural control and inflexibility in as much as influencing manifestations of the disorder (Chamberlain et al, 2016; Prochazkova et al, 2017). The behavioural control factor also includes items that assess the 'not just-right experiences' (NJREs). So-called NJREs are the subjective sense that something is not how it ought to be or is incomplete and may contribute generally to behavioural control and may constitute a unique motivation of OCD symptomatology, separate from harm avoidance and threat overestimations (Coles and Ravid, 2016).

The current paper underscores the distinction between impulsivity and behavioural control, which are not interchangeable (see Joyner et al 2021). While the initial exploratory factor analysis identified an impulsivity factor, this was later removed for a lack of fit and poor reliability. Impulsivity is defined by contextually bound, reactionary thoughts and behaviours that are influenced by situational mood states and may be broad non-specific risk markers across various disorders. Behavioural control, on the other hand, may be described as a

generic, inherent trait that predisposes one to diminished emotional and behavioural output that is coherent across contexts and scenarios. Unlike impulsivity, behavioural control is related to psychopathology, especially externalising disorders and presents as a robust phenotypic marker (Brislin et al, 2019). Though both impulsivity and behavioural control derive from the dysregulation of inhibition, Joyner et al (2021) cautions for careful consideration and distinctiveness of the two concepts. Findings for the OCPAS suggest that behavioural control may be more implicated in OCD, at least for self-assessment.

The third and final factor identified was Anankastic goals. This factor depicts the motivational striving for perfectionist standards through rigid belief systems, preoccupations with order and organization, and an encompassing need for control. Some have proposed that derivatives of anankastia such as mental rigidity and compulsive habitual tendencies may account for as much as 49.4% of the variance in subclinical OCD symptomatology (Ramakrishnan, Robbins, and Zmigrod 2022); here this factor represents the driving force that these phenomena have on the cyclical nature of OCD symptomatology. Features relating to inflexible, rigid thinking appear to be crucial for more severe OCD symptoms, potentially highlighting their importance in assessing OCD phenomenology.

Perfectionist ideals, the need for control, and anankastic domains may be commonplace in disorders permeated by compulsive undertones such as those classified under the OCRDs diagnostic category (Uzgan et al, 2021; Vanzhula, Kinkel-Ram, and Levinson, 2021) and Obsessive-Compulsive Personality Disorder, otherwise known as Anankastic Personality Disorder (World Health Organisation, 2022b). OCPD is defined in the DSM-5 as a disorder marked by rigid fixations and a need for control and order (American Psychiatric Association, 2013). Some degree of concordance emerges between the factors revealed in this analysis and the phenomenology of OCPD as well as OCD. Interestingly, Grant and Chamberlain (2019)

showed that whilst people with OCD and those with OCPD show impairment on tasks assessing cognitive flexibility, only patients with OCD show deficient performance on tasks of response inhibition. The dimension of Behavioural control, as discussed above, is integral to understanding OCD as a psychiatric condition. The absence of this dysfunctional behavioural control in OCPD may suggest that this factor might constitute a differential component that separates the diagnostic class of OCD from that of OCPD (Pinto et al, 2014) though this does require further testing.

The three factors of the OCPAS were moderately inter-correlated – sharing 25-45% of the variance depending on the pair examined – and so may share some core overlapping features of OCD phenomenology (see Appendix T). Indeed all three factors appear to converge on the theme of maintaining control of thoughts and behaviours and so, may speak to OCD being a syndrome relating to the experience of diminished control and the resulting need to regain control. Patients with OCD exhibit a diminished perceived control over their thoughts, and this negatively correlates with symptom severity (Grisham and Williams, 2009). Moulding and Kyrios (2006) depicted a discrepancy between the perceived and desired levels of control in OCD, which may contribute to an impoverished sense of free-will (van Oudheusden et al, 2018), and that the consideration of control and the underlying cognitions can aid comprehension of the disorder. Indeed, Reuven-Magril, Dar, and Liberman (2008) contend that compulsivity in OCD requires revisiting to better reflect compulsions as attempts to regain control.

Overall, the OCPAS provides a comprehensive deliberation of novel compulsive facets inherent within OCD phenomena and delivers a focus on aspects not otherwise tapped by other measures in this field; cognitive and behavioural control. The factor of Cognitive control consists of 9 items describing elements of daily life, such as task management, that

may be effected by dysfunctional cognitive control; an aspect of executive functioning. Considering these items together, a richer representation of deficient executive function is consolidated compared to that of the CPAS (2 items; Fineberg et al, 2007), the CAIOC-13 (2-items; Dittrich, Johansen, and Fineberg, 2011), and the Y-BOCS – to an extent (disturbance of inertia; Storch et al, 2010). Moreover, the delivery of the content explored in this factor may mitigate the failure to appraise such deficiencies in self-ratings that may be present in the item-framing within other measures tapping this phenomenology. There currently exists no measure, whether transdiagnostically or with specificity to a particular disorder, that acknowledges the presence of deficient behavioural control features within compulsive dispositions; the OCPAS is the first in this respect. Though there is documented divergence between the CHIT and response inhibition task performance (Chamberlain and Grant, 2018), Behavioural Control is described here as a diminished ability to inhibit, control, or cease pointless actions or behaviours which is concordant with definitions of compulsivity and of the ego-dystonic repetitions of compulsions as documented within the nosological definitions of OCD (American Psychiatric Association, 2013; World Health Organisation, 2022b). With regard to Anankastic goals, the items of this subfactor converge with the CPAS and CHIT (Fineberg et al, 2007; Chamberlain and Grant, 2018) in assessing overly rigid fixations and preoccupations with perfectionist ideals within those with obsessive-compulsive symptoms. The OCPAS reveals a tripartite structure of OCD phenomenology that is consistent with cognitive-behavioural frameworks of OCD (Leckman, Rauch, and Mataix-Cols, 2007; Finberg et al, 2010).

A factor defined by unwanted thoughts was identified by the exploratory factor analysis but accounted for a small amount of variance and was ultimately removed owing to low reliability. This suggests perhaps that intrusive and unwanted thoughts may not form part of the overall aspect of control outlined above. Indeed, the OCPAS shows a marked absence of

intrusive thoughts in this corpus of items given that research has substantiated significant obsessional thinking in people who develop OCD (Mataix-Cols, Vallejo, and Sanchez-Turet, 2000) and does appear to be a distinguishing factor for OCD from other OCRDs (Abramowitz et al, 2006). One possibility is that intrusive and unwanted thoughts are not a key component of sub-clinical OCD related phenomenology. It may be that obsessions (at least at sub-clinical levels) are a secondary phenomenon to compulsions. In this context, OCD can be viewed as a disorder of deficient behavioural control (Gillan et al, 2011) and obsessions serve to reinforce the compulsions. Indeed, some have proposed a reconceptualization of OCD with obsessions and anxieties arising from compulsions rather than vice-versa – potentially relabelling OCD as compulsive-obsessive disorder (COD: Gillan and Robbins, 2014) thus "acknowledging the difficulty of accepting rigid causal relationships between obsessive and compulsive symptoms" (Robbins, Vaghi, and Banca, 2019, p.30).

Turning to demographic details, total OCPAS scores (and the three subscale scores) were not moderated by whether respondents had a personal history of mental health disorders or a familial history of mental health disorders. Differences may be expected for those reporting personal or familial histories of mental health disorders such as anxiety, depression, and eating disorders as these disorders also exhibit compulsivity to varying degrees (Tchanturia et al, 2011; Stange et al, 2016; Citkowska-Kisielewska et al, 2020). Owing to the transdiagnostic nature of compulsivity and impulsivity (Jacob, Landeros-Weisenberger, and Leckman, 2009; Fletcher, van Kirk, and Hundt, 2020; Tibi et al, 2020; Freis et al, 2022; Schaub et al, 2023), this does suggest that the OCPAS is measuring phenomena specific to OCD and may not be tapping transdiagnostic features. As this sample was non-clinical, this is certainly a point that requires future testing in people with OCD (at greater symptom

severities), as to whether the OCPAS assesses dimensions relating specifically to OCD rather than transdiagnostically.

Limitations and future directions

A limitation of this work concerns the online format of the survey. Although a quick and convenient method, a number of elements may have impeded accurate responding. Notably, participants completed the survey appropriately and within an acceptable timeframe, perhaps discouraged owing to the incentive awarded for participation. Though these scores may still have been influenced by state-dependent reporting (emotional state at the time of administration) and a lack of self-awareness, insofar as respondents not fully comprehending their motivations or behaviours, rendering inaccurate responding (Rosenman, Tennekoon, and Hill, 2011; Anvari et al, 2022).

Moreover, the sample described in this current analysis are referred to as non-clinical as the focus was on the assessment of pre-clinical phenomena that may contribute to the precipitation of OCD. However, other than declaring a personal and/or familial history of mental health disorders, there was no screening of psychiatric phenomena and so there may have been some symptomatic variance within this non-clinical sample. Although these histories had no effect on OCPAS total or subfactor scores, acknowledging the average OCD symptom severity of this sample relative to clinical thresholds (such as a score of ≥ 21 on the OCI-R; Foa et al, 2002) for diagnosable conditions would have aided the representability of these findings to the wider context of pre-clinical compositions of OCD.

Further research is required to validate the OCPAS in clinical samples diagnosed with OCD (or other disorders). The validity of the OCPAS could also be assessed in conjunction with existing self-report and clinician rating measures assessing OCD symptomatology (such as the OCI-R or Y-BOCS). Longitudinal analyses would also help determine the temporal variance and reliability of the OCPAS in both clinical and sub-clinical samples.

Conclusion

The 26-item Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS) delivers a simple, quick and highly robust tool for the self-assessment of cognitive and behavioural control. The OCPAS assesses three key dimensions of OCD phenomenology: Cognitive control, Behavioural control, and Anankastic goals. Further psychometric validation of the OCPAS will be explored in the next section in people with diagnosable OCD. The construct validity of the OCPAS will be assessed against both self-ratings and neurocognitive executive tasks of compulsivity and impulsivity.

STUDY 4.2: VALIDATION OF THE OCPAS IN PEOPLE WITH AN OCD DIAGNOSIS.

INTRODUCTION

As discussed in previous chapters, self-reports and neurocognitive tasks are listed under Research Domain Criteria as units of analysis that can facilitate diagnosis (Cuthbert, 2022). However, there remains the concern, documented in this work and in the wider literature, of the lack of concordance between the two measurements methods of phenotypic manifestations (Howlett et al, 2023; Cyders and Coskunpinar, 2011). For self-reports and task performances for compulsivity, Chapter 3 does show a correlation between the CPAS and the

WCST, albeit weak (r=0.16, p<.05). This was not, however, corroborated with the IDED and the CPAS, which as discussed may be attributed to these tasks not being wholly interchangeable. On the other hand, Chamberlain and Grant (2018) showed that the CHIT correlated with the risk adjustment metric of the CGT (*rho* = 0.26, p = 0.006) but not the quality of decision-making (*rho* = -0.18, p = 0.067). Though an argument can be made as to measuring decisional rigidity, the CGT is not a traditional measurement of cognitive flexibility; this would only give an estimate of a specific expression of an inflexible disposition, whereas, the IDED and WCST would render a more robust estimate given that the outcome metrics of these tasks reside quite closely to the phenotype of impaired flexibility. Only with supplementing the CGT with the CHIT could the compulsive features be derived in this study. This lack of concordance between measures of the same phenotype is also present in the assessment of impulsive manifestations. Jauregi, Kessler, and Hassel (2018) showed that the Stop Signal Task, a tool adapted to the assessment of response inhibition (Aron, Robbins, and Poldrack, 2004), did not correlate with the self-rated Barratt Impulsiveness Scale-11 (Patton, Stanford, and Barratt, 1995) or any of its subfactors (attentional impulsiveness, motor impulsiveness, or non-planning impulsiveness) and so, seem to be orthogonal to each other (all r<.01 and all p>.05). This is suggestive of the two assessment methods mapping on to distinct and independent dimensions of impaired inhibitory control; a dissociation also reflected in findings reported by Boisseau et al (2012) and Frydman et al (2020). Whilst tasks and questionnaires appear to assess the same latent phenotype albeit through differing test parameters, they may in fact assess different dimensions of the same multidimensional construct (Snyder, Friedman, and Hankin, 2020).

This lack of correlation between the two units of analysis could also emanate from their respective limitations such as those described in Chapter 1 *introduction* and Chapter 3.1 *general discussion*, and also on the very approach that each method takes; assessing

performance vs assessing the perception of performance (Dang, King, and Inzlicht, 2020). Nevertheless, a measure that can tap into the neurocognitive trait phenomena is certainly required to provide a reliable and robust estimate of functional impairments in OCD to supplement diagnosis for better long term outcome.

Certainly, this work does document a correlation between measures of impaired cognitive flexibility and the YBOCS (r=0.30,[95%CI 0.11, 0.47], k=14, p<.05) in Chapter 2 which is reflected in Abramovitch et al (2019), and also a correlation between the WCST and the OCI-R (r=.19, p<.05) in Chapter 3 which may hint that these traits develop parallel to symptomatic states albeit cross-sectional discontinuity. No such correlations were established between symptom severity scores and performance on inhibitory control tasks. However, when between-group differences were considered (i.e., estimating the size of the impairment in respect of a healthy comparator) there is no such moderation of symptom severity on task performance of both cognitive flexibility (Z=0.18, df=1,58, p=.86) and inhibitory control (Z=-1,51, df=1,48, p=.13) which is consistent with previous meta-analyses by Shin et al (2014) and Snyder et al (2015) who also demonstrated an orthogonality between the OCD symptom severity and Neurocognitive deficits. This may of course be due to the fact these measures are designed to assess symptomatic states, and although they are adept at assessing changes over time in relation to therapeutic intervention (Storch et al, 2010), there is of course the absence of specificity to the traits underlying such manifestations of impaired behavioural output. Interestingly, these latent cognitive phenotypes were also shown to be independent from illness duration, age, comorbid disorder, and effects of medication which is suggestive of a relative stability of these phenotypes from clinical variables.

As such the OCPAS was designed with these shortcomings in mind; to provide a self-report measure that is of no interpretative difficulty that can tap into these neurocognitive tasks

within OCD phenomenology. As noted in the previous section, the Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS) provides a psychometrically robust 26-item self-rated assessment of functional deficits in OCD phenomenology that is comprised of three subfactors; Cognitive control, Behavioural control, and Anankastic goals. The OCPAS underwent rigorous psychometric validation in a non-clinical sample of 590 participants. Developing the OCPAS at the lower bounds of OCD symptom severity was chosen so that a broad spectrum of obsessive-compulsive phenomena could be detected and explored allowing for vital insights into the transitions between symptoms, syndrome, and illness to be captured. Initially developing the scale in a diagnosed cohort (from the upper bounds of symptom severity) may have only captured the more extreme aspects of the obsessive-compulsive spectrum. Although the OCPAS appears to tap into the more subtle executively impaired features of OCD phenomenology, further work is needed to verify this. Cross-examining the OCPAS with both subjective self-ratings and neurocognitive executive task performances of latent cognitive phenotypes will be most valuable.

Indeed, this implied that the current diagnostic scope of OCD is not able to sufficiently identify the more functional impairments (traits) that accompany these symptoms (states), highlighting the need for an assessment tool designed to capture the underlying latent cognitive phenomena; such as the OCPAS. The OCPAS requires validation in a clinical cohort of patients diagnosed with OCD to verify the detection of these functionally impaired aspects of OCD phenomenology.

Aims and Hypothesis

The aim of this study was concerned with the validation of the newly developed Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS) in a clinical cohort of people with

OCD. The convergent and discriminant validities between the OCPAS and measures of compulsivity and impulsivity i.e., both subjective and objective, are investigated. As discussed, previous research indicates little overlap between self-assessment scales and neurocognitive tasks in the assessment of these phenotypes which may hint at the assessment methods measuring disparate unidimensional constructs. As such, this research was concerned with the validation of the OCPAS in a clinical cohort of participants with OCD and this dissociation between phenotype assessment methods. A comprehensive multidimensional assessment of impulsivity and compulsivity within people with OCD is provided.

It is hypothesised that participants diagnosed with OCD would show different assessment profiles of compulsivity and impulsivity between self-rated scores compared to neurocognitive task performance scores.

METHODS

This research was pre-registration on the Open Science Framework (https://osf.io/j7n3m/) and was approved by the University of Hertfordshire Health, Science, Engineering and Technology Ethics Committee with Delegated Authority (ECDA) (protocol number: LMS/PGR/UH/05648). The reporting of this study adhered to the Checklist for Reporting Results of Internet E-Surveys (CHERRIES, Eysenbach, 2004; 2012).

Design

This research adhered to an observational design. This study was produced and hosted in the experiment building software *Gorilla* (Anwyl-Irvine et al, 2020).

Sample

A power analysis indicated a minimum sample size of 98 would be sufficient to detect significant effects. Participants were recruited from two sources. Participants were recruited from the Orchard OCD registry (https://orchardocdregistry.org/), a service that connects researchers and individuals diagnosed with OCD who wish to contribute to research. The participant recruitment service Prolific (Palan and Schitter, 2018), was also employed. A priori criteria ensured that the survey was only shared with those diagnosed with OCD.

Consent

Participants were presented with an information sheet outlining the particulars of the research. Informed consent to participate was obtained on an online consent form. A number of platforms were used in the creation of this survey, namely Gorilla, Millisecond, and CANTAB. Participants first completed questionnaires on Gorilla and were then directed to Millisecond to complete the Stop Signal Task and CANTAB to complete IDED set-shifting task. As the survey directed participants between platforms, participants were asked to supply their email addressed so that their questionnaire data could be matched with their task performance data.

Participant information and email addresses were stored in accordance with BPS Code of Human Research Ethics (2014), on a password encrypted OneDrive that was only accessible to the lead researcher (AC).

Materials

Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS).

The OCPAS is a self-rated assessment consisting of 26 items that comprise three subfactors converging on themes of compromised control and efforts to regain and reassert control; Cognitive control, Behavioural control, and Anankastic goals, each representing a core facet

of obsessive-compulsive phenomenology. Items are rated on a 5-point Likert scale 0-4 “Not at all”, “A little”, “Quite a lot”, “A lot”, and “Very much”, respectively, and shows excellent internal consistency (α=.92; ω=.92).

Obsessive-Compulsive Inventory-Revised (OCI-R: Foa et al, 2002).

The OCI-R is an 18-item self-rated assessment tool OCD that assesses symptom severity and has been validated in population based research (Abramowitz and Deacon, 2006; Huppert et al, 2007); a score of ≥ 21 is indicative of a strong likelihood of diagnosable OCD. This scale is rated on a 5-point Likert scale 0-4 “Not at all”, “A little”, “Moderately”, “A lot”, and “Extremely”. This scale comprises five symptom subscales (washing, obsessive, ordering, checking, and neutralizing) and has good internal consistency (α=.89; ω=.88).

Compulsive Personality Assessment Scale (CPAS: Fineberg et al, 2007).

The CPAS is an 8-item scale that can be self-rated or observer rated. This scale assesses the severity of compulsive traits. Each item is rated 0-4 (0 = absent; 1 = mild; 2 = moderate; 3 = severe; 4 = very severe) and correspond to individual traits of obsessive compulsive personality disorder as outlined in the DSM-5 (APA, 2013) and has good internal consistency (α=.80; ω=.79). The CPAS was chosen as the self-report measure for compulsivity in this analysis as several items of the CHIT were adapted in the initial development of the OCPAS (see Table 4.1.1.). As a result, any correlation between the CHIT and the OCPAS may have been attributable to overlapping content. Using the CPAS avoided this issue and ensured consistency with Chapter 3 in the measurement of self-reported compulsivity.

The Barratt Impulsiveness Scale-11 (BIS-11: Patton, Stanford, and Barratt, 1995).

The BIS-11 is a 30-item measure suited to the measurement of impulsive tendencies. This measure can be partitioned into three separate facets; motor impulsiveness, non-planning

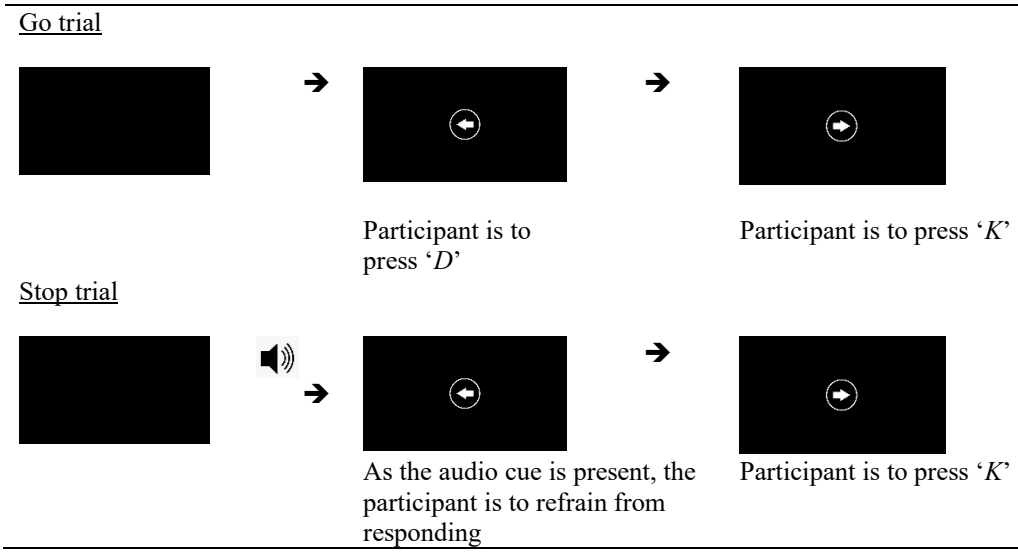
impulsiveness, and attentional impulsiveness. This scale is rated on a 4-point Likert scale 1-4 (Rarely/ Never, Occasionally, Often, and Almost always/Always) and has sufficient internal consistency (α=.77; ω=.77). The BIS-11 is proficient in the assessment of impulsivity in clinical and sub-clinical cohorts (Li and Chen, 2007; Kang et al, 2014). As the BIS-11 was used across a number of studies examining neurocognitive impulsivity in Chapter 2, the BIS-11 was selected for this study to ensure consistency. The BIS-11 is also shorter than its counterparts, the UPPS-P and the Three Factor Impulsivity Index (both exceeding 50 items), a convenient attribute given the duration of the survey across three platforms.

Stop Signal Task (SST: Aron, Robbins, and Poldrack 2004).

The SST assessed the successful inhibition of responding to a given stimuli. Participants are required to press a pre-specified button (D and K) on the keyboard in response to the presentation of an arrow on the screen, though only of a specified orientation (press D for arrows pointing left and press K for arrows pointing right); this is the go trial. Moreover, the participant is to refrain from answering if an audio que is present despite the arrow pointing in the correct direction; this is the stop trial. Participants are first presented with a practice run, to become accustomed to audio cues and the requirements of the task, consisting of 32 trials (8 stop signal trials and 24 go trials). Performance on the practice block is used to calculate the stop signal delay (SSD) for the three test blocks, each consisting of 72 trials (18 stop signal trials and 54 go trials). The presentation of arrow direction as well as the order of go and stop trials is randomised. Each trials lasts 2000ms with the stop signal cue presented between 50ms-1150ms. There are 10 seconds between each test block, during which participants are given feedback on their performance (error responses, % of correct responses, Average response time, stop signal errors, and % of correct stop signals). The task lasts 15 minutes in duration. Performance is estimated using the Stop Signal Reaction Time (SSRT)

metric which is the standardised measure that eliminates the confounding effects that usually befall other reaction time estimates. Participants are instructed not to wait for the stop signal as this can affect the stop signal delay thereby effect correct calculation of the SSRT. Task instructions and example stimuli for the Stop Signal Task can be found in Figure 4.2.1. This task can be found on the Millisecond platform (https://www.millisecond.com/download/library/stopsignaltask; Millisecond software, 2015; Verbruggen et al, 2019).

Figure 4.2.1. Example Stimuli of the Stop Signal Task.

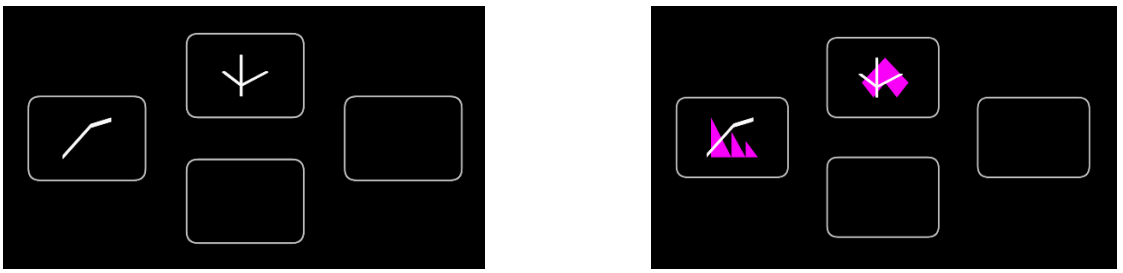


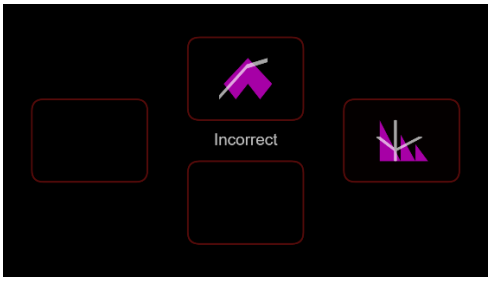
Intra-Extradimensional set-shifting task (IDED: Owen et al, 1991).

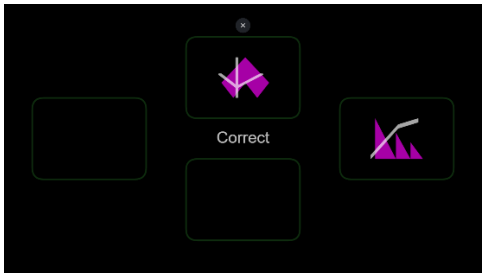
The IDED is a neurocognitive task that assesses the flexibility of responding to changing stimuli. The task focuses on rule acquisition, retention, and reversal. The task is constituted of nine stages, each targeting a different facet of cognitive inflexibility; Simple discrimination, Simple reversal, Compound discrimination adjacent, Compound discrimination superimposed, Compound reversal, Intradimensional shift, Intradimesional shift reversal, Extradimensional shift, and Extradimensional shift reversal. Through trial and error,

participants must match paired images (consisting of either white lines or pink shapes of differing orientations). After 6 trials, the matching rule changes forcing the participant to adapt their responding. Stages 1 and 2 assess the ability to distinguish between two pictures of white lines. Stages 3, 4, and 5 introduces distractions (pink shapes) to this task of discrimination. Next, stages 6 and 7 assess the ability to relinquish the previous rule and acquire the new rule; matching now with pink shapes as opposed to white lines. This is the Intradimensional shift and encompasses a measure of reversal learning. As the task progresses, participants will reach stages 8 and 9, the Extradimensional shift. The matching rule will now change from pink shapes back to white lines; participants will need to adjust their responding to the previously salient stimuli. This stage measures set-shifting. Stage 9 is an integral measure of attentional flexibility. The outcome metrics of this task are formed of the errors made per trial and so the lower the error rate, the better the performance. Echoing the outcome metric rationale from Chapter 3, Extra-dimensional errors at stages 8 and 9 (EDS and EDSR) were the chosen outcome metrics of the IDED in this analysis to express the cognitive (in)flexibility of this clinical sample. Example stimuli presentations can be found in Figure 4.2.2. This task is part of the Cambridge Neuropsychological Test Automated Battery (CANTAB) (https://cambridgecognition.com/intra-extra-dimensional-set-shift-ied/; Sandberg, 2011).

Figure 4.2.2. Intra-Extradimensional set-shifting task.







Procedure

After giving consent to participate, participants were asked questions pertaining to demographic information such as age, sex, ethnicity, level of education, faith group, as well as personal and familial histories of mental health disorders. Participants also declared their OCD status according to 4 pre-determined labels; formally diagnosed with OCD, receiving treatment for OCD (e.g., medication, CBT), self-identify as having OCD, or having been informed by a medical professional that they have OCD. Following this, participants progressed through a battery of scales (the OCPAS, OCI-R, CPAS, and BIS-11) and neurocognitive tasks (the SST and the IDED). The online survey took approximately 20-25 minutes to complete. Once the host of instruments had been completed, participants were thanked for their participation and were reminded of their right to withdraw. For those participants recruited from Prolific, a completion code could be found in the debrief firm so that they could receive their payment for participation.

Statistical analysis

Analyses were conducted in SPSS v29.0.1 (IBM Corp, 2023) and JASP 0.19.2 (JASP team, 2024); descriptives, frequencies, t-test, and regression JASP modules were employed for the described analyses. Sociodemographic variables such as age, sex, ethnicity, education, faith group, personal and familial histories of mental health, and the OCD status of the

participants were extracted. This produced a categorical variable with 4 levels for between group contrasts; (1) formally diagnosed, (2) receiving treatment for OCD (CBT or medication), (3) self-identify as having OCD, and (4) had been informed by a medical professional that they have OCD.

The data from the non-clinical participants analysed in Study 4.1: *Development of the Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS) for the self-assessment of OCD symptoms and traits* was used in comparisons with this current dataset of clinical participants with OCD to assess the ability of the OCPAS to distinguish between those that do and do not have OCD. Comparisons between the two datasets were conducted using Student's *t* for between group variances. Levene's test was used to assess the equality of error variance between groups. There were a total of 32 of 59 violations; equal variances could not be assumed and so the degrees of freedom were adjusted (Zhou, Zhu, and Wong, 2023).

To explore the underlying relations between variables further, the OCPAS, the OCI-R, and the BIS-11 were separated into their constituent subfactors. The OCPAS was scored into its three subscales (Cognitive control, Behavioural control, and Anankastic goals), the OCI-R was score into five symptom subtypes (washing, obsessing, ordering, checking, and neutralising), and the BIS-11 was scored into three subfactors (attentional impulsiveness, non-planning impulsiveness, and motor impulsiveness). Stage 8 and 9 (Extra-dimensional shift and Extra-dimensional shift reversal) of the IDED were used as they closely resemble the phenotype of compulsivity.

Similarly, the Stop Signal Reaction Time of the SST was the chosen to estimate neurocognitive impulsivity; this metric is a calculation of the overall latency of a series of processes implicated in stopping a response. The integration method was chosen to calculate the SSRT as it not impacted by the probability of reacting in stop signal trials and go

omissions that would otherwise impact the previously preferred mean method (Verbruggen et al, 2019). Both the race model and probability of responding to the stop signal were used to highlight violations in performance scores. Typical probability of responding to the stop signal is considered to be .50, with acceptable limits set between .25 and .75, performance scores below or above these limits are considered a violation. A race model violation occurs when the mean response time in a stop-signal trials is greater than the mean response time in a go trial. In these instances, the SSRT was not calculated.

Correlational matrices were conducted to establish convergent and discriminant validities between OC phenomenology (OCPAS), OCD symptom severity (OCI-R), compulsive traits (CPAS), impulsive tendencies (BIS-11), cognitive inflexibility (IDED), and response inhibition (SST). Convergent and discriminant validities were also assessed between the different modes of assessment (self-report assessment and neurocognitive tasks). This also gave an indication of collinearity for the ensuing regression analysis.

The predictive quality of self-rated psychometric scores (OCPAS, OCI-R, CPAS, and BIS-11) on neurocognitive performance (IDED and SST) was assessed using multiple linear regression analyses. The first regression model assessed the prediction of the Stop Signal Reaction Time by OCPAS, OCI-R and BIS-11 scores. The prediction of ED errors at Stage 8 and 9 (EDS and EDSR) of the IDED were both predicted by OCPAS, OCI-R, and CPAS scores. Homoscedasticity was assessed by observing standardised residuals on a Q-Q plot, and Durbin-Watson values were consulted for estimates on autocorrelation (Bartels and Goodhew, 1981). Minimal multicollinearity was revealed as variance inflation factors for the predictor variables in these regression models fall within acceptable limits (Tabachnick and Fidell, 2001).

RESULTS

Descriptive statistics and sample Characteristics

One hundred and eleven participants were recruited from the Orchard OCD registry (n=35) and Prolific (n=76). Thirty participants declared a personal histories of mental health disorders or disorders other than OCD and were thus removed from analyses to maintain a homogenous dataset of only those diagnosed with OCD. Repeating email addresses in the data revealed duplicate entries. Three further participants were removed for duplicate entries; all data associated with the duplicates were removed.

The final sample consisted of 78 participants; 44 participants declared a formal diagnosis of OCD, 18 reported receiving treatment for OCD (CBT, medication, etc), 10 had been informed by a health care professional that they have OCD, and 6 participants self-identified has having OCD. As of the disproportionate group sizes, between group contrasts pertaining to OCD status were not performed. Using comparable benchmarks for OCD symptom severity between the Y-BOCS and the OCI-R (as outlined in Abramovitch et al, (2020); mild = 0-15, moderate = 16-27, and severe = 28-72), the mean OCI-R score reported here (34.15[SD=14.83]) is of a severe nature, indicating that the participants in this study experience high levels of OCD symptoms, comparable with those participants described within the meta-analysis of Chapter 2. Sixty-four participants (82.10%) scored over the OCI-R threshold for diagnosable OCD (≥21) whilst 14 participants scored below. Sociodemographic information can be found in Table 4.2.1.

Table 4.2.1. Participant demographic information (n=78).

Age	35.49 (SD=12.48)	**OCD status**	**%**
Sex	49 F (62.82%)	Primary diagnosis of OCD	56.40
Ethnicity	**%**	Receiving treatment for OCD	23.10
White	76.92	Self-identify as having OCD	7.70
Black	2.56	Informed by HCP as having OCD	12.80

Asian	7.69	**Comorbidities**	**%**
Mixed	8.97	Generalised Anxiety Disorder	6.41
Other	3.85	Social Anxiety Disorder	2.56
Education	**%**	Depression	3.85
Primary ed	3.85	Bipolar	1.28
Secondary ed	35.90	Borderline Personality Disorder	1.28
A-level	1.28	Eating disorder	1.28
Undergrad	37.18	PTSD	1.28
Postgrad	17.95	Panic disorder	1.28
Other	3.85	**Familial history of mental health disorder**	**%**
Faith group	**%**	OCD	16.67
Agnostic	12.82	Generalised Anxiety Disorder	17.95
Atheist	37.18	Autism Spectrum Disorder	1.28
Christian	33.33	Depression	30.77
Muslim	8.97	Bipolar	5.13
Other	7.69	Eating disorder	1.28
		Personality disorder	2.56
		Schizophrenia	1.28
		No history of mental health disorder	28.21

Some participants (7.69%) declared a comorbid disorder in their personal histories of mental health disorders (see Table 4.2.1), however, these disorders were too few and too varied to substantiate between group analyses. Participants were identified as sporadic OCD (n=65) or familial OCD (n=13) based on their personal and familial mental health profiles. Again, due to insufficient group sizes between-group analyses could not be performed.

Interestingly, females scored significantly higher (20.37, SD=7.84, n=46) than males (16.59, SD=8.68, n=29) on the OCPAS Cognitive Control subfactor ($t(76)=1.98$, $p=.05$, $d=0.46$). No other sex differences were revealed on any of the scale variables or neurocognitive metrics. Age did not correlate with OCPAS total or subscale scores, OCI-R scores, CPAS scores, or BIS-11 scores (all $p>.05$) which is suggestive of relative stabilities of these variables across the lifespan. Mean scores for self-rated assessments (OCPAS, OCI-R, CPAS, and BIS-11) and task performances (IDED and SST) can be found in Table 4.2.2.

Table 4.2.2. Descriptive statistics of scale scores and neurocognitive task performance.

	Mean	SD
OCPAS total score	59.74	19.66
Cognitive control	18.96	8.31
Behavioural control	20.96	7.96

Anankastic goals	19.82	7.54
OCI-R total score	34.15	14.83
CPAS total score	14.91	6.30
BIS-11 total score	69.71	7.24
SSRT	221.99	82.28
IDED stage 1 - SD	1.03	0.74
IDED stage 2 - SR	1.26	0.72
IDED stage 3 - CDA	1.41	2.01
IDED stage 4 - CDS	0.26	0.60
IDED stage 5 - CR	1.31	0.73
IDED stage 6 - IDS	1.39	2.49
IDED stage 7 - IDSR	2.28	4.21
IDED stage 8 - EDS	4.66	6.14
IDED stage 9 - EDSR	1.23	0.60
IDED total errors	12.66	4.50

Note: OCPAS = Obsessive-Compulsive Phenomenology Assessment scale; OCI-R = Obsessive-Compulsive Inventory-revised; CPAS = Compulsive Personality Assessment Scale' BIS-11 = Barratt Impulsiveness Scale-11; SSRT = Stop Signal Reaction Time; IDED – Intra-Extradimensional Set-Shifting Task.
SD=Simple discrimination; SR = Simple reversal; CDA = Compound Discrimination Adjacent; CDS = Compound Discrimination Superimposed; CR = Compound Reversal; IDS = Intradimensional Shift; IDSR = Intradimensional Shift Reversal; EDS = Extradimensional Shift; EDSR = Extradimensional Shift Reversal

OCPAS scores in clinical and non-clinical groups

The previous non-clinical cohort examined in Chapter 4, Study 4.1 were compared to this current clinical cohort on all 59 original items from the original full corpus of OCPAS items to ensure that no salient phenomena were discarded. Interestingly, only 5 items showed no difference between the two cohorts, these being items 16, 18, 24, 28, and 40 (see Table 4.2.3), none of which were in the final OCPAS, and many of the excluded items only produced small effect sizes. This clinical cohort scored significantly higher than the previous

non-clinical cohort on all 26 items of the revised OCPAS (*d*=0.42-1.05) which gives more credence for their retention in this refined scale.

Table 4.2.3. Mean OCPAS item score comparisons between non-clinical and clinical cohorts.

	Non-clinical (n=590) Mean (SD)	Clinical (n=78) Mean (SD)	Between group comparisons *t & d values*
2 I am troubled by having to repeat behaviours that seem excessive or pointless.	1.25(1.20)	2.53(1.29)	**8.72, 1.05****
5 I have difficulty getting started on new tasks.	1.61(1.22)	2.36(1.33)	**5.03, 0.61****
7 Others describe me as someone who needs to feel in control.	1.48(1.19)	2.30(1.33)	**5.60, 0.68****
12 I have difficulty finishing tasks that I have started.	1.34(1.20)	2.06(1.42)	**4.91, 0.59** v.e.v.**
13 I feel the need for things to be perfect.	2.00(1.20)	2.71(1.23)	**4.72, 0.57****
14 People have told me I can be stubborn or rigid.	1.70(1.20)	2.58(1.42)	**5.96, 0.72** v.e.v.**
15 I find uncertainty troubling.	2.01(1.22)	3.00(1.16)	**6.82, 0.82****
19 I am troubled by having to repeat mental acts that seem excessive or pointless.	1.32(1.18)	2.45(1.23)	**7.95, 0.96****
20 Once I have started an action I find it hard to stop.	1.21(1.03)	2.27(1.19)	**8.38, 1.01** v.e.v.**
21 Routine activities take me longer than they should.	1.07(1.16)	2.09(1.36)	**7.15, 0.86** v.e.v.**
22 I doubt having done things properly.	1.51(1.19)	2.32(1.30)	**5.56, 0.67****
23 I like to be in control or else I feel upset.	1.42(1.14)	2.19(1.23)	**5.56, 0.67****
26 I try to do things (i.e., activities, tasks, or movements) until they feel "just right".	1.79(1.17)	2.71(1.25)	**6.46, 0.78****
30 I perform unnecessary physical or vocal actions e.g., muscle twitching, coughing, sniffing, throat clearing, saying certain words.	0.77(1.10)	1.71(1.46)	**6.74, 0.81** v.e.v.**
31 I lack energy, motivation or drive.	1.58(1.24)	2.28(1.27)	**4.68, 0.56****
32 I have difficulty organising and carrying out a planned task e.g., a holiday, shopping trip or a birthday celebration.	1.01(1.17)	1.67(1.34)	**4.57, 0.55** v.e.v.**
33 I can be rigid.	1.45(1.00)	2.18(1.32)	**5.82, 0.70** v.e.v.**
41 I get stuck when performing routine tasks.	0.88(1.02)	1.89(1.35)	**7.87, 0.95** v.e.v.**
42 I tend to have rigid views about how things look or have to be.	1.41(1.02)	2.28(1.19)	**6.95, 0.84** v.e.v.**
44 I find it hard moving from task to task.	1.02(1.06)	1.71(1.26)	**5.26, 0.63** v.e.v.**

46 I am troubled when things are not complete e.g., missing items in a set.	1.66(1.15)	2.41(1.30)	**5.32, 0.64** v.e.v.**
49 I get preoccupied with details.	1.71(1.14)	2.59(1.30)	**6.33, 0.76** v.e.v.**
52 I feel exhausted.	1.88(1.28)	2.59(1.19)	**4.69, 0.57****
53 I tend to do things according to a routine (e.g., eating meals at a specific time, using the same utensils, sitting in a specific seat etc).	1.58(1.20)	2.10(1.47)	**3.51, 0.42** v.e.v.**
55 I am disturbed by small errors or imperfections.	1.30(1.04)	2.30(1.21)	**7.76, 0.93** v.e.v.**
3 I frequently act on impulse.	1.26(1.01)	1.92(0.96)	**5.52, 0.67****
4 I do things impulsively that seem rewarding at the time even if I know they are bad for me in the long run.	1.30(1.08)	2.33(1.23)	**7.82, 0.94** v.e.v.**
6 I am a creature of habit	2.33(1.07)	2.98(1.01)	**5.06, 0.61** v.e.v.**
8 I am troubled by unwanted thoughts that repeat in my mind.	1.44(1.26)	2.97(1.14)	**10.22, 1.23****
9 I am effective in stopping or diverting unwanted behaviours or urges (reverse scored).	2.20(1.05)	2.74(0.96)	**4.31, 0.52** v.e.v.**
10 I resist acting on urges (reverse scored).	2.13(1.01)	2.54(0.95)	**3.40, 0.41****
11 I have difficulty making decisions about everyday matters.	1.13(1.15)	2.18(1.23)	**7.55, 0.91** v.e.v.**
17 I am effective in stopping or diverting unwanted repetitive thoughts (reverse scored).	2.35(1.05)	2.87(0.97)	**4.18, 0.50** v.e.v.**
18 I take strong positions on things when I believe they are right or wrong.	2.49(1.01)	2.67(1.07)	1.46, 0.18
27 I am effective in stopping or diverting unwanted thoughts (reverse scored).	2.36(1.08)	2.96(0.95)	**4.70, 0.57** v.e.v.**
36 Others see me as being hyper-critical.	1.18(1.13)	2.04(1.33)	**6.22, 0.75** v.e.v.**
37 If I am interrupted, I can quickly switch back to what I was doing (reverse scored).	2.26(1.07)	2.71(1.02)	**3.47, 0.42****
38 I can be flexible or easy going (reverse scored).	1.88(1.06)	2.51(0.86)	**5.05, 0.61** v.e.v.**
39 When something I have done does not feel quite right, I need to repeat it until it does.	1.51(1.11)	2.50(1.38)	**7.19, 0.87** v.e.v.**
40 I see money as something I should save for future possible needs.	2.16(1.18)	1.91(1.32)	1.75, 0.21
43 I can easily get rid of things that I do not need (reverse scored).	2.20(1.20)	2.69(1.20)	**3.42, 0.41****
51 I avoid getting rid of things because I am afraid that I might need them later.	1.67(1.29)	2.19(1.51)	**3.32, 0.40****
54 Others have commented that I make noises or movements e.g., sniffing, humming, grunting.	0.59(1.04)	1.31(1.44)	**5.46, 0.66** v.e.v.**
56 I find it easy to trust others to carry out tasks the way they ought to be done (reverse scored).	2.54(1.06)	2.95(1.12)	**3.16, 0.38***

58 I am happy to compromise (reverse scored).	1.98(0.95)	2.51(0.86)	**4.71, 0.57****	
59 I usually concentrate more on the whole picture, rather than the small details (reverse scored).	2.21(1.01)	2.76(1.12)	**4.45, 0.54****	
1 I get stuck thinking about one thing.	1.83(1.16)	2.89(0.95)	**7.63, 0.92****	**v.e.v.**
16 I am good at working out how people are thinking and feeling even when they do not tell me (reverse scored).	1.87(1.15)	1.63(1.33)	1.72, 0.21	**v.e.v.**
24 I can tolerate breaking a rule (reverse scored).	2.35(1.00)	2.41(1.06)	0.48, 0.06	
25 I tend to feel guilty when things go wrong even when it is not my fault	1.81(1.27)	2.74(1.19)	**6.13, 0.74****	
28 I have difficulty spending money especially when it is on myself or a loved one	1.12(1.20)	1.24(1.44)	0.83, 0.10	**v.e.v.**
29 I have strong urges to acquire things when I don't really need them.	1.67(1.18)	2.09(1.31)	**6.43, 0.77****	**v.e.v.**
34 When I read, I sense I may have missed something and must go back and reread the passage	1.67(1.24)	2.18(1.32)	**6.56, 0.79****	
35 I shoulder responsibility beyond what could be reasonably expected	1.62(1.23)	2.42(1.32)	**5.40, 0.65****	
45 I prefer all or nothing decisions	1.36(1.25)	1.91(1.42)	**3.60, 0.43****	
47 I feel disgust in the presence of bodily secretions e.g., blood, sweat, urine, faeces	1.30(1.19)	1.97(1.47)	**4.61, 0.56****	**v.e.v.**
48 I feel uncomfortable if I do not get my own way	1.12(0.94)	1.81(1.25)	**5.79, 0.70****	**v.e.v.**
50 I find it hard to manage unexpected changes to my routine	1.54(1.16)	2.58(1.23)	**7.33, 0.88****	**v.e.v.**
57 Certain foods, textures, and smells easily disgust me	1.16(1.19)	1.89(1.40)	**4.96, 0.60****	**v.e.v.**

Note: R=reversed scored item

*Items in **bold** are those retained in the final 26-item OCPAS*

df=666 for all between-group comparisons.

***=p<.001*

**=p<.05*

v.e.v. = Violation of equal variance assumption (Levene's test)

The mean OCPAS score reported here (59.74; SD=19.66, n=78) for people with OCD is significantly higher than the mean score recorded in non-clinical participants in Study 4.1 (37.45; SD=18.21, n=590); $t(666)=10.07$, $p<.001$, $d=1.21$. Subfactor scores are also higher for OCD patients compared to the non-clinical cohort; Cognitive control (18.96(SD=8.31) vs 11.89(SD=8.22); $t(666)=7.13$, $p<.001$, $d=0.86$), Behavioural control (20.96(SD=7.96) vs 12.39(SD=6.86); $t(666)=10.16$, $p<.001$, $d=1.23$), and Anankastic goals (19.82(SD=7.54) vs 13.16(SD=6.34); $t(666)=8.51$, $p<.001$, $d=1.03$). There is a stark contrast between the two samples as evidenced by the large effect sizes which suggests that deficits in cognitive and behavioural control are quite pronounced at greater symptom severity. All analyses that follow were calculated using the revised 26-item scale.

Self-assessment questionnaires

A significant difference emerged on OCPAS total scores for those scoring above (64.55; SD=16.27, n=64) and below (37.79; SD=19.31, n=14) the OCI-R threshold ($t(76)=5.39$, $p<.001$, $d=1.59$). Similarly, CPAS scores significantly differed for those scoring above (15.97; SD=5.94, n=64) and below (10.07; SD=5.81, n=14) the OCI-R threshold ($t(76)=3.378$, $p<.001$, $d=1.00$).

Normative limits for impulsiveness on the BIS-11 are scores of between 52 and 71; with scores of 72+ being indicative of high impulsiveness (Patton, Stanford, and Barratt, 1995). The BIS-11 score recorded in this sample (69.71; SD=7.24) falls within this range at the upper limits of normative performance and on the cusp of high impulsiveness.

Neurocognitive task performance

There were a total 11 performances that violated the assumptions in calculating the SSRT; 3 race model violations and a further 9 violations in the probability of responding to the stop

signal (one participant violated both assumptions). In light of Thunberg et al's (2024) analysis of the SST, normative performance ranges reside between 187ms(±)27 and 202ms(±)23 indicating that the SSRT recorded here resides at the uppermost limits of normative performance (221.99;SD=82.28). Table 4.2.4 displays the outcome data for the SST.

Table 4.2.4. Stop Signal Task outcome metrics.

	Mean	(SD)
Probability of go omission (no response)	4.12 (%)	(8.97)
Probability of choice errors on go trial	95.11 (%)	(8.76)
RT on go trials	551.94 (Ms)	(141.06)
Probability of responding on a stop trial (for each SSD)	48.63 (%)	(7.53)
Average SSD	303.96 (Ms)	(153.16)
SSRT	221.99	(82.28)
RT of go responses on unsuccessful stop trials	473.02 (Ms)	(113.66)

Note: RT = reaction time; SSD = Stop Signal Delay; SSRT = Stop Signal Reaction Time; Ms = milliseconds.

The extradimensional shift (EDS; stage 8) and the extradimensional shift reversal (EDSR; stage 9) metrics are typically used to quantify cognitive inflexibility. Here, the mean EDS score was 4.66 (SD=6.14), mean EDSR score was 1.23 (SD=0.60), and a total errors score was 12.66 (SD=4.50) – see Appendix U for IDED mean scores. Previous work has shown ED shift error scores (10.11; SD=2.23) and total error scores (21.13; SD=2.84) to be greater in participants with OCD (Chen et al, 2021) suggesting that the current sample exhibits a normal extradimensional shift performance profile on the IDED. No between group difference on EDS or total errors emerged for those scoring above or below the threshold for diagnosable OCD (both p>.05).

Correlations

A correlational matrix was conducted to assess the association between neurocognitive task performance and self-rating scales (see Table 4.2.5). All of the self-rating scales (OCPAS,

OCI-R, CPAS, and the BIS-11) share moderate to strong relations (all $p<.001$); the strongest correlation revealed between OCPAS scores and OCI-R scores ($r=.73$, $p<.001$); see Figure 4.2.3 for the scatterplot for the correlation between OCPAS and OCI-R scores.

Interestingly, the SST shared weak to moderate relationships with OCI-R and BIS-11 scores (both $r=.37$, $p<.05$). No relationships were established between the IDED outcome metrics and the OCPAS, OCI-R, CPAS, or the BIS-11. The covariation between OCPAS subscales and neurocognitive metrics of the Stop Signal Task and the Intra-Extradimensional set-shifting task is assessed in Table 4.2.4. Cognitive control was significantly negatively correlated with Compound Discrimination Adjusted, Compound Discrimination Superimposed ($r=-0.37$ and $r=-0.36$, respectively). Behavioural control was moderately positively correlated with the Stop Signal Reaction Time ($r=0.40$), and Anankastic goals were not correlated with any of the neurocognitive outcome metrics.

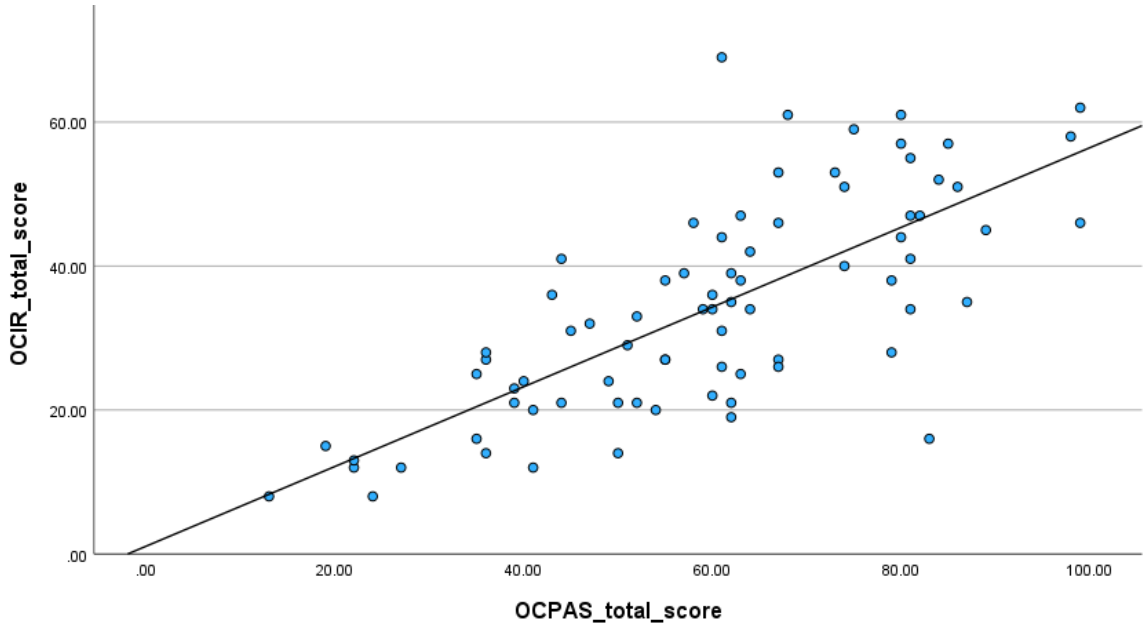
However, it is important to note that only a portion of the sample completed the neurocognitive tasks; 31 participants completed the SST and 35 participants completed the IDED. As such, the sample size may be too small and analyses underpowered to detect true effects and so caution is warranted with interpretating these correlations between neurocognitive tasks and self-reports. As a number of participants started but did not complete the IDED (stages 1-7 n= 39; stage 8 n=38; and stage 9 n=35), the IDED total score (adjusted) outcome metric was used to account for missing cases.

Table 4.2.5. Correlational matrix of total self-rating scores and neurocognitive test metrics.

	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*	*(10)*	*(11)*	*(12)*	*(13)*	*(14)*	*(15)*	*(16)*	*(17)*
(1) OCPAS	-																
(2) Cognitive control	0.81**	-															
(3) Behavioural control	0.84**	0.49**	-														
(5) Anankastic goals	0.83**	0.49**	0.58**	-													
(5) OCI-R	0.73**	0.50**	0.78**	0.54**	-												
(6) CPAS	0.67**	0.40**	0.62**	0.65**	0.67**	-											
(7) BIS-11	0.36**	0.19	0.43**	0.29*	0.50**	0.56**	-										
(8) SSRT	0.24	0.17	0.40*	0.03	0.37*	0.33	0.37*	-									
(9) IDED stage 1 – SD	0.04	0.04	0.00	0.05	0.09	-0.08	-0.15	-0.07	-								
(10) IDED stage 2 – SR	0.07	-0.02	0.20	-0.02	0.11	-0.07	0.29	0.41*	-0.26	-							
(11) IDED stage 3 – CDA	-0.31	-0.37*	-0.12	-0.26	-0.09	-0.23	0.15	-0.04	-0.33*	0.24	-						
(12) IDED stage 4 – CDS	-0.16	-0.36*	0.05	-0.05	0.13	0.21	0.10	0.47*	0.34*	0.03	0.02	-					
(13) IDED stage 5 – CR	-0.01	-0.11	0.05	0.05	0.22	0.07	-0.20	-0.14	-0.02	0.05	-0.09	0.24	-				
(14) IDED stage 6 – IDS	0.00	-0.22	0.14	0.12	0.07	-0.07	-0.06	-0.04	-0.01	0.17	0.04	0.23	0.38*	-			
(15) IDED stage 7 – IDSR	-0.15	-0.13	-0.05	-0.20	-0.08	0.11	0.01	0.02	0.01	-0.11	-0.09	-0.03	0.01	-0.10	-		
(16) IDED stage 8 – EDS	-0.25	-0.20	-0.30	-0.11	-0.20	-0.19	0.13	-0.42*	-0.10	0.05	0.18	-0.08	-0.16	-0.19	-0.23	-	
(17) IDED stage 9 – EDSR	-0.29	-0.17	-0.29	-0.26	-0.15	-0.22	0.04	-0.18	-0.22	-0.07	0.59**	-0.18	0.08	0.09	-0.15	0.29	-
(18) IDED total errors (adj)	-0.31	-0.30	-0.24	-0.22	-0.18	-0.11	0.11	-0.25	-0.10	0.06	0.24	-0.02	-0.03	0.08	0.59**	0.93**	0.47*

SD=Simple discrimination; SR = Simple reversal; CDA = Compound Discrimination Adjacent; CDS = Compound Discrimination Superimposed; CR = Compound Reversal; IDS = Intradimensional Shift; IDSR = Intradimensional Shift Reversal; EDS = Extradimensional Shift; EDSR = Extradimensional Shift Reversal; (adj) = adjusted.

Figure 4.2.3. Scatterplot of OCPAS total scores against OCI-R total scores.



To explore subscale inter-relations, each measure was separated into its respective subfactors: the OCI-R was separated into Ordering, Obsessing, Washing, Checking, and Neutralising; and the BIS-11 was separated into Attentional impulsiveness, Non-planning impulsiveness, and Motor impulsiveness. As each item of the CPAS corresponds with a unique facet of compulsive dispositions, each item was assessed for correlations with the described subscale scores (see Table 4.2.6).

Table 4.2.6. Correlational matrix of subscale scores for the OCPAS, OCI-R, BIS-11, and CPAS.

	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*	*(10)*	*(11)*	*(12)*	*(13)*	*(14)*	*(15)*	*(16)*	*(17)*	*(18)*	*(19)*	*(20)*
(1) OCPAS – Cognitive Control	--																			
(2) OCPAS – Behavioural control	0.49**	--																		
(3) OCPAS – Anankastic goals	0.49**	0.58**	--																	
(4) OCI-R – OCD component	0.46**	0.80**	0.55**	--																
(5) OCI-R – Hoarding component	0.40**	0.38**	0.30*	0.48**	--															
(6) OCI-R – Washing	0.25*	0.49**	0.21	0.56**	0.14	--														
(7) OCI-R – Obsessing	0.29*	0.35**	0.13	0.61**	0.26*	0.24*	--													
(8) OCI-R – Ordering	0.37**	0.67**	0.65**	0.76**	0.49**	0.23*	0.24*	--												
(9) OCI-R – Checking	0.40**	0.59**	0.46**	0.78**	0.45**	0.10	0.46**	0.62**	--											
(10) OCI-R – Neutralising	0.31*	0.63**	0.38**	0.75**	0.32*	0.29*	0.29*	0.49**	0.55**	--										
(11) BIS-11 – Attentional Imp	0.46**	0.36**	0.34*	0.46**	0.40**	0.20	0.43**	0.37**	0.27*	0.34*	--									
(12) BIS-11 – Motor Imp	0.22*	0.09	0.03	0.17	0.38**	-0.06	0.10	0.30*	0.05	0.21	0.32*	--								
(13) BIS-11 – Non-planning Imp	0.16	-0.17	-0.11	-0.11	0.18	-0.15	0.01	-0.09	-0.13	-0.01	0.30*	0.43**	--							
(14) CPAS – Preoccupation with detail	0.24*	0.48**	0.46**	0.44**	0.44**	0.17	0.15	0.49**	0.36**	0.33*	0.36**	0.31*	-0.02	--						
(15) CPAS – Perfectionism	0.36**	0.57**	0.68**	0.45**	0.37**	0.20	0.16	0.54**	0.31*	0.33*	0.29*	0.18	-0.01	0.66**	--					
(16) CPAS – Workaholism	0.20	0.46**	0.27*	0.39**	0.37**	0.13	0.20	0.44**	0.30*	0.29*	0.20	0.22*	-0.14	0.44**	0.39**	--				
(17) CPAS – Over-conscientiousness	0.26*	0.20	0.29*	0.23*	0.40**	0.17	0.12	0.21	0.14	0.15	0.26*	0.26*	0.03	0.50**	0.37**	0.5*	--			
(18) CPAS – Hoarding	0.26*	0.27*	0.20	0.38**	0.73**	0.00	0.110	0.43**	0.41**	0.36**	0.36**	0.35*	0.20	0.37**	0.31*	0.29*	0.32*	--		
(19) CPAS – Need for control	0.22*	0.38**	0.26**	0.44**	0.36**	0.06	0.23*	0.50**	0.37**	0.36**	0.17	0.13	-0.23*	0.32*	0.37**	0.47**	0.27*	0.30*	--	
(20) CPAS – Miserliness	0.17	0.35*	0.25*	0.28*	0.08	0.16	0.05	0.27*	0.28*	0.19	-0.08	-0.11	-0.56**	0.15	0.18	0.38**	0.18	0.00	0.22	--
(21) CPAS – Rigidity	0.39**	0.47**	0.78**	0.51**	0.37**	0.18	0.26*	0.63**	0.42**	0.26*	0.37**	0.14	0.01	0.44**	0.58**	0.28*	0.44**	0.27*	0.39**	0.19

*NOTE: OCPAS = Obsessive-Compulsive Phenomenology Scale; OCI-R = Obsessive-Compulsive Inventory-Revised; BIS-11 = Barratt Impulsiveness Scale-11; Imp = Impulsivity; CPAS= Compulsive Personality Assessment Scale; *=p<.05, **=p<.001*

Cognitive Control, Behavioural Control, and Anankastic Goals shared moderate to strong relations with the Ordering, Checking, and Neutralising subfactors of the OCI-R. Though Cognitive Control shared a weak correlation with Motor Impulsivity (r=0.22), all three of the OCPAS subscales correlated with Attentional Impulsiveness. With regard to CPAS items, OCPAS components consistently shared weak to strong relations with Preoccupation with Details, Perfectionism, Need for control, and Rigidity (r=0.22-0.78).

Correlational matrices were conducted to assess the relative convergence between self-ratings and neurocognitive task performances of compulsivity and impulsivity in OCD phenomenology. For compulsivity, a collection of moderate correlations were observed between the CPAS items and the stages of the IDED: preoccupation with detail and perfectionism were negatively correlated with EDS and both perfectionism and rigidity were negatively correlated with EDSR (see Appendix V). For impulsivity, the SST positively correlated with total BIS-11 score (r=0.37, p<.05) but not with any subscale (see Appendix W).

Planned regression analyses

Three multiple linear regressions were performed to assess the predictive quality of self-ratings on neurocognitive task performance.

To test the prediction of response inhibition, OCPAS, OCI-R, and BIS-11 scores were set as predictors, and SSRT was set as the dependent variable. A QQ-plot was used to assess homoscedasticity (see Appendix X); no patterns or outliers were revealed and so constant variance was assured (Yang, Tu, and Chen, 2019). However, this regression model showed a Durbin-Watson value of 2.47 suggesting a slight negative autocorrelation. The utility of the

predictive model was non-significant ($F(3,30)=1.96$, $R^2=.18$, $p=0.14$) and so caution is warranted with interpretation. No significant prediction was revealed between self-report scores and SSRT (all $p<.01$).

To test the prediction of cognitive flexibility, OCPAS, OCI-R, and CPAS scores were predictor variables and ED errors at stage 8 (Extradimensional shift) and stage 9 (Extradimensional shift reversal) of the IDED were the dependent variable. Whilst a Q-Q plot revealed a number of outlies for the predictive model of stage 8 and 9 (see Appendices Y and Z), the Durbin-Watson values of 2.21 and 2.18, respectively, revealed minimal autocorrelation. The predictive model of stage 8 ($F(3,37)=0.76$, $R^2=.06$, $p=.52$) and stage 9 ($F(3,34)=1.07$, $R^2=.09$, $p=.38$) were not significant. No significant prediction was revealed between self-report scores and ED errors at stage 8 or stage 9 (all $p<.01$).

DISCUSSION

The aim of this study was to ascertain the relative associations between subjective and objective indices of compulsivity and impulsivity in participants with OCD and to assess the convergent and discriminant validities of the Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS). As such, only partial support was found for the hypothesis: "*participants diagnosed with OCD would show different assessment profiles between self-rated scores compared to neurocognitive performance scores*". As described, phenotypes of compulsivity and impulsivity within OCD phenomenology were explored using both subjective self-assessments and objective task performances. Participants with OCD showed conflicting profiles of compulsivity in that their self-ratings (on the CPAS) depicted overly rigid dispositions that were not reflected in their task performances (on the IDED). On the

other hand, their profiles of impulsivity are more consistent across both self-assessment and neurocognitive performance (the BIS-11 and SST, respectively) given the moderate correlation between the two modalities (r=.37, p<.05). This suggested that impulsivity may be more reliably represented in both subjective and objective measures within this clinical sample.

Comparison of OCPAS total and subfactor scores between this current clinical sample and the non-clinical sample used in *STUDY 4.1: INITIAL VALIDATION OF THE OCPAS* showed that scores were higher in this clinical group for all 26 retained items. This supports the initial psychometric validation of the OCPAS and also exemplifies its ability in distinguishing those that do have OCD from those that do not. 28 of the 33 discarded items also showed significant differences with worse performance in this current sample compared to the non-clinical sample in Study 4.1. Moreover, OCPAS scores correlated strongly with symptom severity scores as rated by the OCI-R and its subscales (see Figure 4.2.4). Given that this sample experienced high levels of OCD symptom severity (according to benchmarks described in Abramovitch et al (2020) for the OCI-R), this may hint at a linear progression of these phenomena (Cognitive control, Behavioural control, and Anankastic control) parallel to that of OCD symptom severity.

Cross examination of OCPAS subfactors; Compulsivity

Self-ratings

As shown in Table 4.2.4, OCPAS and CPAS scores share a strong relationship (r=0.67, p<.001) and by looking at the correlational matrix of the various subfactors (Table 4.2.5), the items of the CPAS are consistently related to the subfactors of the OCPAS, hinting at compulsive undertones in all three, though it is the Anankastic goals subfactor that is the most strongly correlated with the CPAS. There may be some concordance between the Anankastic

goals subfactor and the phenomenology of Obsessive-Compulsive Personality Disorder (OCPD; APA, 2013), otherwise known as Anankastic Personality Disorder (WHO, 2022b); characterised by its preoccupation with perfectionist ideals that permeate all facets of daily life, having implications for both occupational and interpersonal relationships (Fineberg et al, 2015; Hellriegel et al, 2017). Indeed, there is a strong convergence between Anankastic goals and both perfectionism and rigidity items of the CPAS (items 2 and 8 of the CPAS; r=0.68 and 0.78, respectively). Previous research does not contend with relationships between OCD symptom severity and self-ratings of compulsivity (Gadelkarim et al, 2017; 2019) which is in contrast to the current findings using the CPAS which shows significant correlations with several symptom dimensions of the OCI-R. Though notably, there is no correlation between the obsessing factor and the CPAS and so the lack of relationship previously reported by Gadelkarim et al (2017) may be attributed to the presence of obsessional symptoms on the Y-BOCS, as obsessions are the defining aspect in the differential diagnosis between OCD and OCPD, in that obsessions are absent in the latter (APA, 2013; WHO, 2022b). This is also the case for the washing factor of the OCI-R and items of the CPAS (all>.05). Where the excessive cleaning in OCD is motivated by threat overestimations of contagion, the maladaptive cleaning habits in OCPD may be differently motivated by a need for order and control (Gibbs and Oltmanns, 1995). Interestingly, the obsessing and washing subfactors of the OCI-R did not correlate with the OCPAS Anankastic goals subfactor, reinforcing the parallel previously drawn between OCPAS Anankastic goals and OCPD symptomatology. The subfactor of Anankastic goals certainly represents the more compulsive features of OCD phenomenology compared to the other two OCPAS factors and may also be transdiagnostic in scoring and assessing compulsive features of other psychiatric phenomena, though this requires further investigation.

Neurocognitive task performance

The Intra-Extradimensional set-shifting task (IDED; Owens et al, 1991) provides an objective measure of latent compulsivity through the assessment of cognitive inflexibility, a deficit that is well evidenced in OCD (Nedeljkovic et al, 2009; Isobe et al, 2022). However, the relationships between OCPAS scores and the IDED is contrary to the expected. This sample of participants with OCD did not show the expected patterns of deficits at the EDS and EDSR stages of the IDED. Table 4.2.5 revealed correlations between Cognitive control and ED errors on stages 3 and 4 (CDA and CDS, the introduction of irrelevant distractors) which is suggestive of better performance on the IDED at higher OCPAS scores. This sample of participants with OCD did not however show the expected patterns of deficits at the EDS and EDSR stages of the IDED. As the key index of the IDED in the assessment of cognitive inflexibility is ED errors at stages 8 and 9 and the preceding stages are an estimate of basic learning (Owen et al, 1991; Chamberlain et al, 2021), this suggests that the correlations observed between the OCPAS and performances on the CDA and CDS stages are spurious. Though these relationships may be spurious, the fact that the OCPAS is detecting something that that OCI-R is not suggests that the OCPAS is assessing aspects that are closer to the subtle neurocognitive deficits of the disorder that standard symptom scales fail to.

Performance on the IDED may have been influenced by IQ in producing a ceiling effect as previous research has shown neurocognitive testing to be predictive of average and below average IQs, but less so of above average IQ (Diaz-Asper, Schretlen, and Pearlson, 2004). However, IQ was not tested in this analysis and so this cannot be ascertained, though 55.13% of the participants in this sample had engaged with higher education to graduate status. Errors on the IDED could not differentiate between those with a graduate status compared to those without (all $p>.05$).

Alternatively, these findings may speak towards a self-appraisal of a deficit that is not objectively reflected on task performances. Research does implicate metacognition as an integral aspect in the study of neurocognitive deficits in patients with OCD as there appears to be an attentional bias associated with metacognitive beliefs in the ability to attend to stimuli in tasks such as those used here (Hermans et al, 2003; Hezel and McNally, 2016). There is a tendency to reflect upon cognitive processes in patients with OCD, and with an under confidence in their abilities, this may lead to overestimations of deficient performances (Janeck et al, 2003; Dar et al, 2022). This supposition is supported in the negative correlations revealed between preoccupation with detail, perfectionism, and rigidity of the CPAS and the final two stages of the IDED (see Appendix T). Interestingly, research does show that high perfectionism can exist parallel to intact cognitive functioning within the same sample (Robinson and Abramovitch, 2020). This may be true of the current sample as there are conflicting accounts of compulsivity between assessment methods; the task performances recorded here fall within normative ranges, whereas, self-ratings on the CPAS depict heightened rigidity. These findings may also reflect the dissociation between objective and subjective modes of assessment (as discussed in Chapter 3) and so the conflicting account of compulsivity may derive from a commentary of under confidences in ability and subsequent overestimations of a rigid disposition that is not reflected on executive tasks of the same phenotype (Janeck et al, 2003; Dang, King, and Inzlicht, 2020; Dar et al, 2022).

Moreover, OCI-R scores did not correlate with any of the IDED stages (all $p>.05$) suggesting that this sample with severe OCD symptoms has functioning flexibility. This is consistent with Wolff et al (2018) who demonstrated that greater flexibility was found in patients with OCD (n=20) compared to healthy controls (n=22). However, this sample comprised exclusively of adolescent patients which, and as previously discussed, have a distinct executive profile to adults under the same diagnostic label (Geller et al, 2001; Farrell and

Barrett, 2010) and so these findings may not be wholly reflective of the performance of this current sample. Moreover, Fradkin et al's (2018) meta-analysis of cognitive flexibility showed that adult patients with OCD exhibited indifferent performance to that of a normative comparator, though as discussed, Fradkin et al used a composite of compulsive dimensions to estimate the relative profile of (impaired) flexibility including reversal learning, alternation, and attentional set-shifting which may have ultimately concealed specifically impaired domains resulting in a potentially false negative interpretation. Previous research concurs with a relationship between OCD symptom severity and attentional set-shifting in formally diagnosed cohorts as reported in within-group correlations in Chapter 2 and by Abramovitch et al (2019). However, between-group contrasts reported in of Chapter 2 and Snyder et al (2015) showed that deficient performance scores on tasks of set-shifting do not correlate with symptom severity scores, suggesting that there is no association between the size of this executive impairment and the severity of OCD symptomatology, though it is important to note that this contrast in findings may be attributable to the statistical paradigm used (between-group differences vs within-group correlations).

Since the participants of this analysis were largely self-diagnosed, this may speak towards a continuum of OCD phenomenology, from symptoms to syndrome to illness, in that these deficits only manifest at a particular junction of symptom severity. These neurocognitive deficits appear to be additive and parallel to symptom severity and may be the switch between managing and not managing these symptoms resulting in admissions to public health services (Brakoulias and Sara, 2011; Veale et al, 2016b), thereby underscoring the need for appropriate acknowledgement within the diagnostic assessment of OCD.

Cross examination of OCPAS subfactors; Impulsivity

Self-ratings

A moderate correlation was revealed between OCPAS and BIS-11 scores, though interestingly total BIS-11 scores shared a stronger relation with the Behavioural control subfactor (r=0.36 vs r=0.43). This subfactor of the OCPAS consists of items that converge on deficient inhibitory control over thoughts and actions which may account for this convergence with BIS-11 scores. The Behavioural control subfactor may resemble a factor upon which impulsive and compulsive undertones co-exist given its correlations with both the BIS-11 and the CPAS. Prochazkova et al (2017) showed that a cluster of both compulsive and impulsive features contributed towards greater symptom severity in patients with OCD and indeed Behavioural control was strongly correlated with symptom severity as scored by the OCI-R (r=0.78, p<.001). The evidence base of trait impulsivity in OCD symptomatology is however inconsistent. With previous research employing the Y-BOCS for the measurement of symptom severity (Grassi, Makris, and Pallanti, 2019; Frydman et al, 2020) not contending with this association with impulsiveness, and the literature propounding the relationship between BIS-11 and OCI-R scores being scarce, it is altogether interesting that total OCI-R scores in this analysis do in fact share a moderate to strong correlation with BIS-11 scores (r=0.50, p<.001) suggesting heightened impulsiveness at great symptom severities within this sample.

Interestingly, all three OCPAS subfactors moderately correlated with attentional impulsivity (r=0.34-0.46). Research conducted by Benatti et al (2014) does contend with the impulsivity in OCD as being attentional in nature. Patton, Stanford, and Barratt (1995) describe the attentional impulsivity subscale of the BIS-11 as inattention. This may hint at a permeation of attentionally impulsive features throughout the phenomenology of OCD. A moderate correlation was revealed between the obsessing subfactor of the OCI-R and attentional

impulsivity of the BIS-11 (r=0.43, p<.001) which aligns with previous research exploring impulsivity in obsessional symptoms in OCD (Boisseau et al, 2012). The manifestation and behaviour of the impulsive phenotype within OCD may contribute to the understanding of subtypes of OCD that are comorbid with impulsive control disorders (Moulding, Aardema, and O'Connor, 2014; Onur et al, 2016).

However, it is important to note that the attentional impulsivity subscale loads onto its counterparts (motor and non-planning impulsivity); it is not a stable construct and so may underly impulsivity as a whole (Patton, Stanford, and Barratt, 1995). Indeed, McDonald's Omega (ω) showed that items 8, 17, 23, and 25 negatively correlated with the overall scale demonstrating weak coherence within the underlying construct. As such, attentional impulsiveness may not reflect a definitive cognitive substrate of impulsivity but may provide a non-specific index for psychopathology (Stanford et al, 2009). As of this ill-fitting substrate of attentional impulsiveness, exploring the relationships it shares with other scales in this analysis may be flawed. Both Swann et al (2002) and Reise et al (2013) show that the BIS-11 is better presented as a two-dimensional measure, converging on themes of the inability to wait for a reward and rapid response styles which suggests some redundancy of the attentional impulsivity subfactor in the assessment of impulsivity. The two dimensional structure may map onto neurocognitive task metrics far better than the three-dimensional structure given the resemblance between these two factors and the inhibitory control paradigms of the CGT and the SST. These two dimensions put forward by Swann et al (2002) and Reise et al (2013) are also consistent with earlier descriptions of the lack of mitigation and agency over pre-potent action being ultimately impaired in patients with OCD. Given earlier descriptions of the *negative urgency* subfactor of the UPPS-P (Lynam et al, 2006; 2013), as well as the *feelings trigger action* subfactor of the Three Factor Impulsivity Index (Carver et al, 2011), in having thematic similarities with the attendance to obsessional stress,

these scales may offer a better and more psychometrically verified alternative to the BIS-11 in estimating the impulsivity phenotype within OCD phenomenology.

Neurocognitive task performance

The Stop-signal task (SST; Aron, Robbins, and Poldrack, 2004), an objective measurement of successful prepotent action inhibition that can be used to detail impulsive tendencies, correlated with OCPAS Behavioural control (r=0.40, p<.05) suggesting deficient inhibitory processes in those that struggle with both delaying and preventing (inhibiting) their compulsive actions (Enright and Beech, 1993; Morein-Zamir et al, 2010). It is this lack of inhibition and control over their thoughts and actions that contributes to a greater symptom severity (van Velzen et al, 2014). This also demonstrates that the OCPAS is able to tap into dimensions closer to the subtle neurocognitive nuances than previously developed scales. Crucially, total OCI-R scores were also revealed to correlate with SST performance (r=0.37, p<.05), as was the neutralising subscale (r=0.43, p<.05). This subscale captures the extent to which patients with OCD engage with the compulsions in order to dispel the distress produced by obsessional intrusions (Foa et al, 2002); a negative valence system, as listed under the RDoC framework (Cuthbert et al, 2022). This neutralising subscale also strongly correlates with the Behavioural control subfactor (r=0.63, p<.001).

Certainly, previous research has shown that when compared to healthy controls, patients with OCD do exhibit impaired performance on the SST (Penades et al, 2007; Morein-Zamir et al, 2010; Boisseau et al, 2012). However, findings by Snorrason, Smari, and Olafsson (2009) do not concur with this relationship between OCD symptom severity and impaired SST performance that is shown here, although the authors do note the use of a college sample and so this difference between findings may be a reflection of an emergence of impulsive traits only at diagnosable symptom severities such as in this current sample.

This is also reflected in Chapter 2 in that moderate effect sizes of response inhibition deficits were revealed in clinically diagnosed OCD, which was also comparable to effect sizes of cognitive inflexibility; an established deficit in OCD phenomenology. Interestingly, the SST and stage 8 of the IDED (Extradimensional Shift) did show a degree of correlation in this analysis which may allude to a shared underlying neurobiological pathway (Fineberg et al, 2014). The mean SST performances recorded here (221.99;SD=82.28) reside at the upper limits of normative performance as decreed by Thunberg et al (2024). Indeed, trait impulsivity in OCD is considered to be at the lower end of impairment when compared to other psychiatric conditions (Banwell et al, 2024); as no comparator group were tested, the exact magnitude of deficient performance cannot be ascertained.

Moreover, a moderate positive correlation was revealed between both indices of impulsivity, total BIS-11 scores and performance scores on the SST (r=0.37, p<.05), and when non-planning impulsivity was controlled for, the strength of this correlation increased (r=0.40, p<.05); Motor impulsivity also appeared to approach significance (see Appendix U). This stands in contrast to previous research as null-findings for this relationship have been consistently reported between the SST and the BIS-11 (Boisseau et al, 2012; Sohn et al, 2014; Grassi et al, 2015; Frydman et al, 2020; Banwell et al, 2024) as well as for alternatives such as the UPPS-P and the Iowa Gambling task (Cyders, 2020). The lack of relationship observed between the various methods of assessing these phenotypes may speak to differences in state and trait measures of impulsivity (Enticott and Ogloff, 2006), and this difference may conflate interpretations of impulsivity within OCD phenomenology (Summerfeldt et al, 2004). As such, there is an emphasis on clarifying definitions of impulsivity owing to its multifaceted composition and the various approaches to operationalise impulsiveness (Boisseau et al, 2012). In the context of these current findings in participants with OCD, it may be that these impulsive tendencies are less subject to the over-estimations of impairment

that appears to be apparent in compulsive tendencies but may in fact receive relatively accurate self-ratings. This would account for this moderate concordance between the two different assessment methods of impulsivity. Further work on meta-cognitive aspects and insight in relation to compulsive and impulsive phenotypes in OCD is needed to investigate this vitality of this argument for the difference in effects observed here.

Limitations

The Orchard OCD registry does not have a rigorous screening procedure and though 44 participants declared a primary diagnosis of OCD (see Table 4.2.1.), the sample may largely be composed of individuals who are self-diagnosed as having OCD. As those in direct attendance of a clinic were not tested, it is uncertain that these findings are a true reflection of that which is typically observed in patients with OCD. However, this sample's symptom severity is comparable to that of a clinical sample (Fulop et al, 2024) and 82% of the sample do score above the threshold for diagnosable OCD on the OCI-R (Foa et al, 2002). Further work is warranted to replicate these findings in formally diagnosed samples of patients with OCD.

Although between-group comparisons on the OCPAS with the non-clinical sample of study 4.1 revealed greater control deficiencies at higher symptom severity in this current group (despite cross-sectional discontinuity), the lack of a comparison group restricts the representativeness of the neurocognitive data. As only within-group correlations were performed on the IDED and the SST, the magnitude of any deficient performance in this clinical group relative to a healthy comparator cannot be ascertained.

Another limitation of this study is that only half of the participants completed the neurocognitive tasks, and so the powering of these within-group correlations is questionable. As series of post-hoc power revealed that the relationship between OCPAS behaviour control

and the Stop signal task meets threshold (r=.40, n=31, p<.001; powering =.77), as do the relationships between OCPAS cognitive control and IDED stage 3 (r=.37, n=39, p<.05; powering =.79) and IDED stage 4 (r=.36, n=39, p<.05; powering =.76) (Dorey, 2010; Serdar et al, 2020). However, caution is still warranted with interpretation as the p values of these correlations with the IDED exceed the Bonferroni corrected alpha limit of p=0.125.

The accuracy of responding may have also been hindered by psychiatric ill health. The influence of situational moods states could not be assessed in this analysis, as was the case in Chapter 3, as no measure of mood (depressive or anxious states) was included in this study. As symptoms of depression are often concurrent with OCD phenomena (Altintas and Taskintuna, 2015; Buchholz et al, 2019), it is uncertain whether the effects observed are influenced in any way by concurrent depressive phenomena. Although the meta-regressions of Chapter 2 showed no moderation of depression or anxiety symptomatology on the neurocognitive performance of patients with OCD, these scores were of moderate proportions and so greater severities may have had such an effect on executive performance.

Moreover, Hedge et al (2020) showed that self-reports can be influenced by 'impulsive information processing' insofar as Likert selections being made on the immediate impression of the scale item. As this sample showed deficient inhibitory control on the SST that was correlated with higher scores on the BIS-11, analyses were conducted to assess the accuracy of self-appraisals. Having controlled for impulsive dispositions by partialling out SSRT scores, all correlations between self-reports were sustained apart from the relationship between the OCPAS and the BIS-11 implying that there was some impulsive responding that contributed to the relationship between these two scales.

Conclusion

The OCPAS demonstrated convergent validity with a measure of OCD symptom severity and was able to distinguish between those with and without OCD. Moreover, the convergent and divergent relationships between the OCPAS and its subfactors with differing measurements of cognitive and behavioural control (self-rated scales and neurocognitive task performances) exemplify the OCPAS as a robust assessment of impaired cognitive and behavioural control.

Moreover, the CPAS did not correlate with the IDED cementing the divergence between these two modes of assessment which is furthered by their respective relationships with symptom severity; correlations were established between the OCI-R and the CPAS but not between the OCI-R and the IDED. This is reflective of Chapter 3, though the symptom severity here is greater. Conversely, this was not the case for impulsivity as both the BIS-11 and the SST each correlated with the OCI-R and with each other suggesting a convergence between the two modes of assessment. As discussed in Chapter 3, there is the implication of meta-cognitive commentary in self-reports leading to both under- and overrepresentations in ability and so this concordance between the BIS-11 and the SST may suggest that impulsivity is more reliably rated compared to compulsivity within this sample.

Crucially, the relationship between the SST and OCI-R is singular in advancing earlier conjectures of impulsivity in OCD symptomatology insofar as having no direct relationship to symptom severity. As shown in Chapter 2, there was no relationship between inhibitory deficits and Y-BOCS in either between- and within-group analyses which detailed a stability across the lifespan given other orthogonalities with age and duration of illness. It is interesting to note that the sample of Chapter 4, Study 4.2 is not strictly diagnosed with OCD and so this cohort occupies a space on a continuum just before formal diagnosis and so it is interesting that neurocognitive impulsivity does in fact correlate with symptom severity at this juncture. This may then represent a window where inhibitory issues, coupled with

aspects of compulsivity contribute to worsening symptomology that will ultimately exacerbate the illness enough to then meet criteria for a diagnosis of OCD.

The following chapter provides a consolidation of all empirical evidence presented across the chapters of this thesis and presents interpretations of the manifestation of neurocognitive compulsivity and impulsivity within OCD symptomatology and phenomenology.

CHAPTER 5: CONCLUSIONS AND INTERPRETATIONS

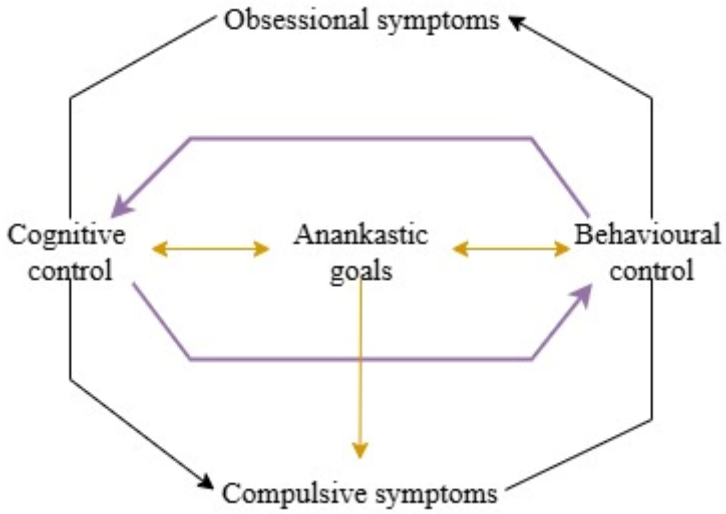
An elaboration on the OCPAS

As discussed in Chapter 4, there have been previous attempts to quantify compulsivity adjacent to impulsivity; the CHIT and the Impulsive-Compulsive Behaviours Checklist (Chamberlain and Grant, 2018; Guo et al, 2017). The CHIT is primarily a transdiagnostic assessment of compulsivity, though a number of items do appear to tap impulsivity (e.g., "*I can't resist acting on urges*" and "*I do things that are immediately rewarding, even if I know it is bad for me in the long run*"), though not enough to substantiate a separate subscale of impulsiveness. The Impulsive-Compulsive Behaviours Checklist on the other hand does provide a broad exploration of both compulsive and impulsive behaviours. However, this scale is unable to probe motivational drivers of these phenotypes and so its application is restricted to superficial assessments. The development and validation of the Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS) has addressed these limitations by providing a robust assessment of compulsive and impulsive phenomenology with strong construct and convergent validity that can distinguish between those with and without OCD.

The OCPAS captures a tripartite structure of this diminished capacity of control which is consistent with previous conceptions of OCD phenomenology insofar as having distinctive cognitive and behavioural components (Salkovskis, Forrester, and Richards, 1998). Due to deficiencies in executive functioning, patients with OCD exhibit a compromised ability to consciously and wilfully exert control over thoughts and actions. It then follows that these cognitive and behavioural components assessed by the OCPAS may influence one another; As behavioural contingencies (the compulsions; i.e., washing, checking etc) are enacted to dispel obsessional distress (pertaining to ego-dystonic themes), deficiencies in goal-directed behaviour (control of cognitive and behavioural faculties) may contribute to the inception of

a maladaptive system of perpetuating symptomatology that is underscored by deficient cognitive flexibility and inhibitory control over these thoughts and actions. This dynamic exemplifies a maladaptive autopoiesis (a negative feedback loop); a system that reproduces and sustains itself (Juarrero, 2009) – an ouroboros of OCD symptomatology (see Figure 5.1 for a descriptive representation).

Figure 5.1. Negative feedback loop of cognitive and behavioural control in OCD phenomenology.



Where the Reciprocal Interaction model (RIM), proposed by Kalanthroff and Wheaton (2022), based on clinical neuroscience and traditional cognitive-behavioural theories of OCD, is consistent with this negative feedback loop (Figure 5.1), the presence of Anankastic goals in the factor structure of the OCPAS provides a novel insight into how this model is sustained and reinforced. As described in Chapter 4, this factor represents perfectionist standards, rigid beliefs and fixed objectives of order and symmetry that may serve as the core motivational drive that sustains the cycle of dysfunctional goal-directed behaviour in patients with OCD (Gillan and Robbins, 2014). Where there is a lack of cognitive and behavioural control, there

is an equal drive to achieve these high standards to rectify this, to fill this hole, this breach in autonomy. These strivings for perfectionism and order provide a structure to compensate for this loss of control (Zor et al, 2006; Moretz and McKay, 2009; Pinto et al, 2017).

Alternatively, the dysfunctional goal-directed behaviour in OCD may be viewed as a diminished feeling of *'knowing'* that the goal has been achieved. Szechtman and Woody (2004; 2006) label this self-generated signal *yedasentience*, a failure to cease action and behaviours due to the absence of this signal of knowing and comprehending that the goal has been achieved resulting in a persistence with maladaptive behaviours and rituals. Owing to the aforementioned executive deficits, upon the insurgence of obsessional distress, there is an urgency to reassert and regain control, however, due to the loss of these markers for goal attainment, there is a perseverance and inability to inhibit these compulsions. Anankastic goals may represent a collection of methods aimed to emulate this yedasentience signal, pseudo-markers for goal attainment; once a sense of just-right, or indeed perfection, has been achieved, the compulsion subsides.

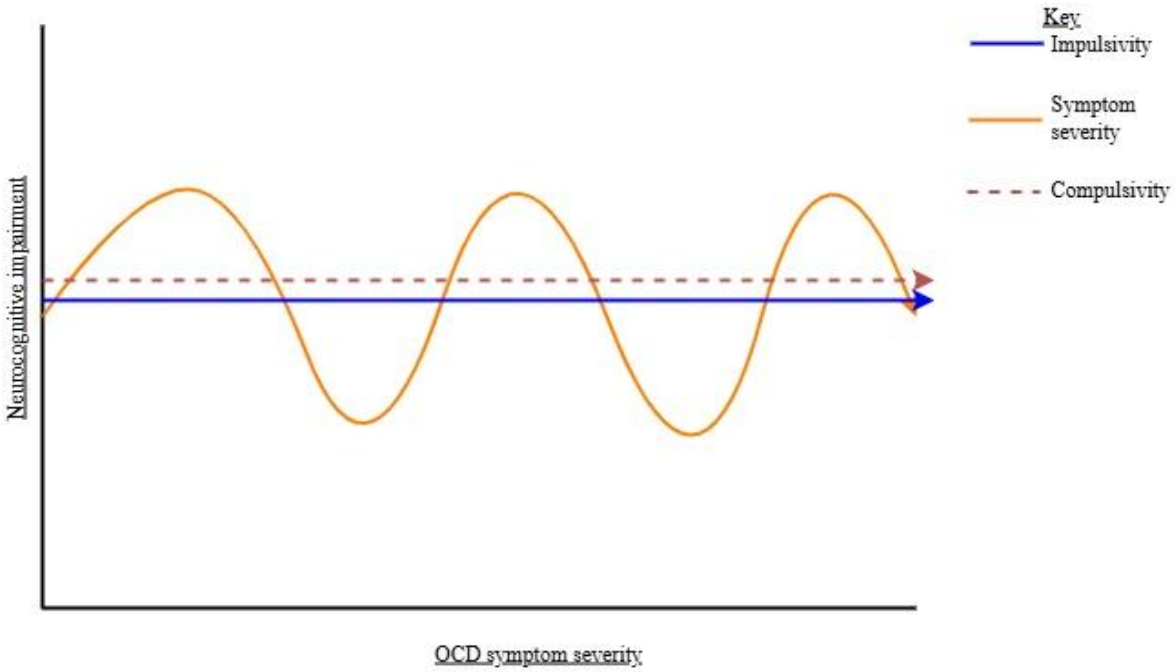
Indeed, the Behavioural control and Anankastic goals may also have relevance for the development of habitual behaviours. The Behavioural control subfactor of the OCPAS provides a collection of items that largely reflect a need to act, there is an overwhelming insurgence of anxiety and distress brought on by the obsessional (ego-dystonic) intrusion and so the compulsion is engaged with in order to bring about a reprieve from such distress. These item descriptions are consistent with avoidant behaviours which are of course reminiscent of the negative urgency subscale of the UPPS-P (Lymen et al, 2006; 2013) and the feelings trigger action of the Three-Factor Impulsivity Index (Carver et al, 2011). Once the obsessional distress has been neutralised, these anankastic behaviours and standards would then be favoured as devices to employ upon future resurgences of this distress, owing

to previous emotional valence (Voon et al, 2015). The items comprising Anankastic goals are consistent with ego-syntonic themes in that these behaviours align with the individual's sense of self, values and desires (Stairs et al, 2012) emphasising their relevance in the abating of (ego-dystonic) obsessional distress (Audet, Aardema, and Moulding, 2016). Whilst the Anankastic goals subfactor represents the reassertion of control, the Behavioural control subfactor is the loss of control. As these valence systems, both positive and negative appear to be inherent within these habitual learning processes (Cuthbert, 2022), the OCPAS and its subfactors may have practical implications for assessing the precipitation and perpetuation of OCD.

Tracking phenotypic variance at different junctures of OCD symptom severity

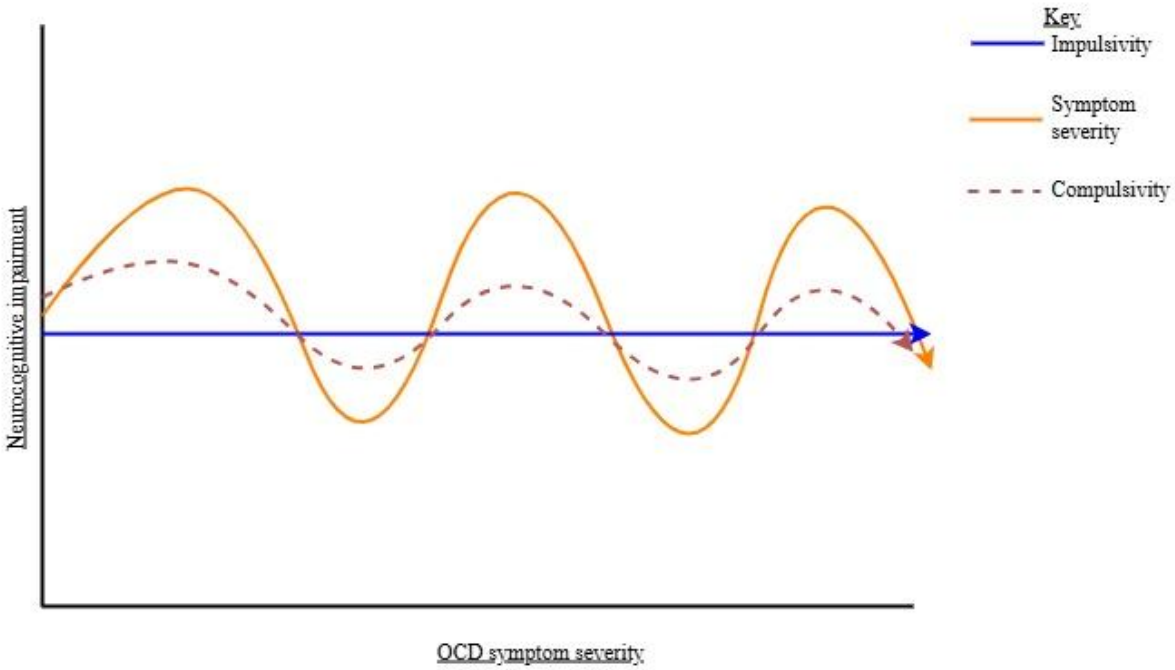
Meta-regressions of Chapter 2 revealed an orthogonal relationship between symptom severity and between-group contrasts of patients with OCD against healthy controls on neurocognitive tasks assessing latent cognitive phenotypes of compulsivity and impulsivity. This is to say that, compared to healthy controls, compulsivity and impulsivity are stable traits irrespective of fluctuating severities in patients with OCD (see Figure 5.2).

Figure 5.2. Between-group differences of symptom severity and neurocognitive task performance of compulsivity and impulsivity



However, when within-group correlations are considered, neurocognitive performance on compulsivity tasks is revealed to correlate with symptom severity (r=0.30,[95%CI 0.11, 0.47], k=14, p<.05); this is shown in Figure 5.3 – the lines of compulsivity and symptom severity show overlapping oscillations. Neurocognitive performance on impulsivity tasks remains largely unrelated to symptom severity (p>0.5).

Figure 5.3. Within-group correlations of symptom severity and neurocognitive task performance of compulsivity and impulsivity



Moreover, this is not to disregard the previous findings of orthogonality between compulsivity and symptom severity; this illustrated that the differences between healthy controls and patients with OCD cannot be fully explained by symptom severity alone, as when patients are considered independently from controls, there is such a relationship between neurocognitive compulsivity and symptom severity. This contrast in outcome between the two difference approaches in Chapter 2 (between vs within) is an exemplification of the divergence in consensus within the literature concerning the relationship between OCD symptom severity and neurocognitive impairments.

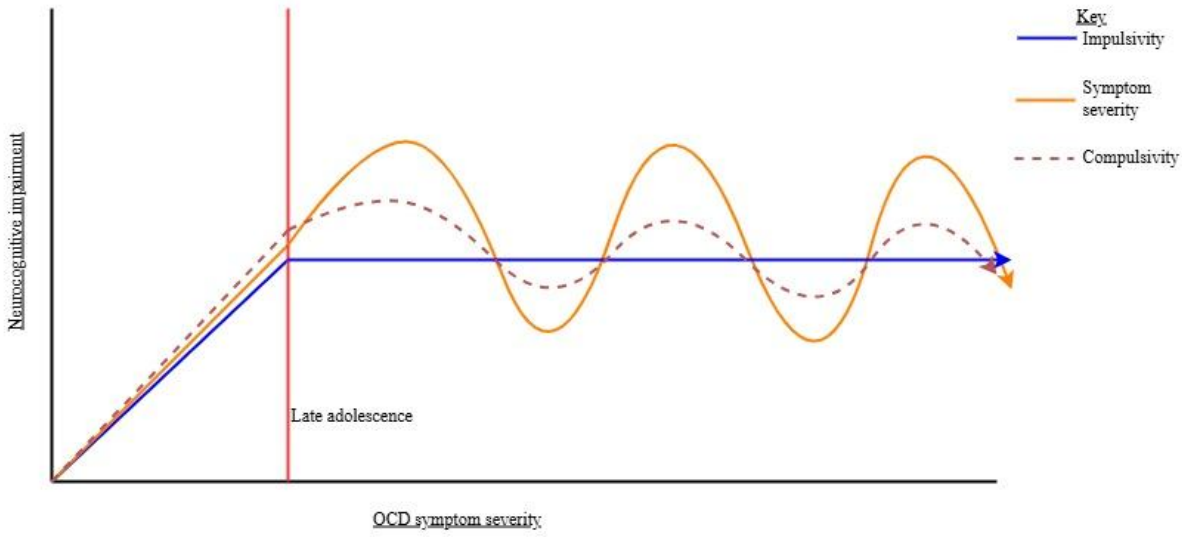
Abramovitch et al's (2019) meta-analysis of within-group correlations showed that deficient set-shifting correlated with severe Y-BOCS scores (r=-.28[95%CI -.35, -.20], k=38, p<.001). However, the findings of Chapter 2 also conflict with Abramovitch et al (2019) as a within-group correlation was detected for response inhibition and Y-BOCS scores in their analysis

(*r*=-.28[95%CI -.41, -.14], k=19, *p*<.001) which was not reflected in this work. However, if the single outcome metrics are then considered separately in Abramovitch et al (2019), the SSRT does not correlate with Y-BOCS scores (*r*=-.15[95%CI -.41, .13], k=4, *p*=.28) indicating that the aggregated effect size in their paper is mostly reflective of the Stroop task (Stroop, 1935). Though both the Stroop task and the SST are both measures of response inhibition, the former may be pulling from other cognitive components that the SST does not, and so this variation between analyses may be a product of unique inter-task differences; such is the task impurity and heterogeneity issue explored in Chapter 3. Although Abramovitch et al (2019) contends with such a relationship between symptom severity and executive deficits, these findings are restricted as the very size of the impairments examined within their clinical sample cannot be estimated as they did not employ a comparator group.

On the other hand, Abramovitch, Abramowitz, and Mittelman (2013), Shin et al's (2014) and Snyder et al's (2015) meta-analyses did utilise a control group and showed that patients with OCD have impaired performance on tasks of cognitive flexibility (*d*=.30-.52) and response inhibition (*d*=.49-.62) which largely accord with the effect sizes revealed in this work. Crucially, these meta-analyses are consistent with the orthogonality between the neurocognitive performance and OCD symptom severity: Both Abramovitch, Abramowitz, and Mittelman (2013) and Shin et al (2014) report null findings, and Snyder et al (2015) reported a definitive non-relation ($z = 1.30$, $p = .194$), all of which stand in stark contrast to that of Abramovitch et al (2019). The statistical paradigm used to explore these relationships here proves to show vital implications for interpretation. This does harken back to the arguments made for (1) greater between-study consistency using neurocognitive tasks (Gruner and Pittenger, 2017), and (2) the crucial consideration of the research question when using neurocognitive tasks (Enkavi et al, 2019).

As previously discussed, the meta-analysis conducted by Lopez-Hernandez et al (2023) depicted correlations between OC symptom severity and deficiencies in both cognitive flexibility and response inhibition in early childhood to late adolescence. As detailed in Chapter 2, comparisons were made between the two meta-analyses (Lopez-Hernandez et al, 2023; Clarke et al, 2024) that surmised a linear progression of these latent phenotypes parallel to OC symptom severity in early development that appears to stabilise in late adolescence and throughout adulthood. This is visually represented in Figure 5.4 to depict the linear progression of these latent cognitive phenotypes relative to symptom severity from childhood, through adolescence, and into adulthood.

Figure 5.4. Visual representation of symptom severity and latent cognitive phenotypes across the lifespan.



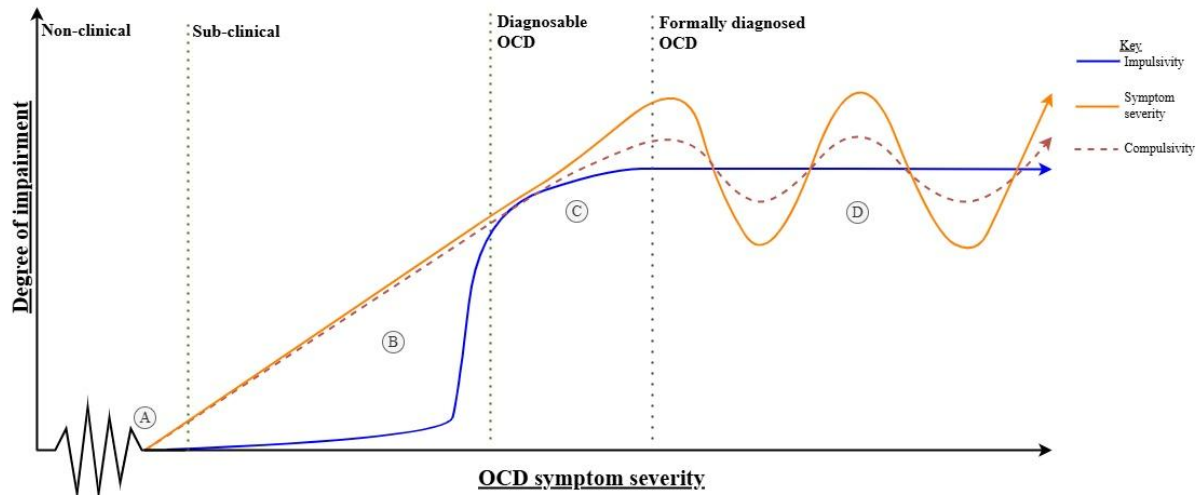
Though these is cross-sectional discontinuity limiting the generalisability of this current developmental pathway depicted in Figure 5.4, there is support for such distinctions in the (neuro)pathogenic development of OCD. Both Rosenberg and Keshavan (1998) and Maia, Cooney, and Peterson (2008) showed volumes in the orbito-and ventral prefrontal cortices as

well as the striatum and caudate nucleus to be of import in the neurological underpinnings of OCD and symptom severity is relates to such irregular volumes. Certainly, these portions have been shown to be implicated within cognitive flexibility and response inhibition (Blasi et al, 2006; Kehagia, Murray, and Robbins, 2010). These studies showed that there is a relationship between these atypical volumes and symptom severity but not with duration of the illness and so it may seem that this relationship between symptom severity is only present at the initial presentation of OCD and may diverge at later developmental stages; this is reflected in the meta-regression of Chapter 2 as illness duration did not moderate neurocognitive performance. However, Rosenberg and Keshavan (1998) only included a small sample of Pediatric patients (n=21) between the ages of 7 and 17, and as the mean duration of illness was not reported, it is unclear how far these findings may extend. Research documenting clear developmental trajectories between Pediatric, adolescent, and adult forms of OCD is scarce, though this may allude to this trajectory being categorial than continuous as of developmental discontinuities with executive maturation also being a consideration (Boelema et al, 2014; Tervo-Clemmens et al, 2023).

However, this model is based on the assumption that obsessive-compulsive symptomatology begins in early childhood and as such is only reflective of those with early-onset OCD. Those with late-onset OCD (manifestation in early adulthood) would perhaps show a distinct profile to the model proposed in Figure 5.4 as there are reported clinical markers that distinguish the two subgroups of OCD (Sobin, Blundell, and Karayiorgou, 2000). It is to be noted that the exact distinctions between early and late onsets were never directly assessed in the presented and described analyses within the preceding chapters, nonetheless, it is important to note the findings of Lopez-Hernandez et al (2023) are exclusive to patients with early-onset OCD.

The findings of Lopez-Hernandez et al (2023) aside, the meta-analytical findings of Chapter 2 depicted the manifestations of these deficits relative to symptom severity in those with a formal diagnosis of OCD; those at the upper end of a continuum from symptoms to syndrome, to illness. Indeed, as sub-clinical groups (those without a diagnosis) exhibit OCD symptomatology that is comparable with patients formally diagnosed with OCD, research calls for clear delineations in the transition from symptoms to syndrome (Apter et al, 1996); where these changes occur along a continuum of symptom severity and potential markers for this transition. When considered in tandem, the different chapters offer sequential observations of the phenotypic variance of compulsivity and impulsivity at different junctures of OCD symptom severity: Study 4.1 – non-clinical; Chapter 3 – sub-clinical; Study 4.2 – diagnosable OCD; and Chapter 2 – formally diagnosed OCD (see Figure 5.5).

Figure 5.5. Visual representation of latent cognitive phenotypes at junctures of OCD symptom severity.



As described in Study 4.1, relatively healthy participants were recruited in the initial development of the self-rated OCPAS to reveal pre-clinical features of OCD phenomenology. As no rigorous screening was used to identify those with higher symptom severities, it was uncertain if there was some symptomatic variation within this sample. However, when compared to the sample of Study 4.2 on the OCPAS, it was clear that the relative severity of the sample of Study 4.1 was certainly below clinical significance given the effect sizes of between-groups differences on total and subfactor scores of the OCPAS between the two samples (see Appendix S). As described, the factor structure of the OCPAS does reveal compulsive and impulsive undertones within Cognitive control, Behavioural control, and Anankastic goals. However, the exact composition of these phenotypes relative to symptom severity cannot be ascertained in this sample, albeit the findings do hint at these components being fundamental OCD phenomena even at lower severities (see Figure 5.5, Point A).

Where Study 4.1 represents phenotypic variance in a non-clinical sample, Chapter 3 serves as the sub-clinical segmentation of OCD symptomatology and phenomenology. This sample represents at point on the presented continuum (Figure 5.5) before symptoms become disabling and chronic but are still impairing to everyday life. The adverse circumstances captured within this chapter saw pronounced OC symptomatology in undiagnosed members of the general public. Both self-rated compulsivity (rates on the CPAS) and OC symptoms (rated on the OCI-R) were observed as having predisposing qualities in the development of difficulties in adjusting to contingent behavioural strategies to reduce the spread of the Coronavirus. Indeed, over a series of three assessment points, CPAS and OCI-R scores positively correlated (r=.70-.88, all p<.001) demonstrating a strong relationship between compulsivity and symptom severity that is temporally consistent within this sample. This is certainly consistent with previous formulations of worsening inflexibilities at greater OC symptom severity in sub-clinical samples (Kim, Jang, and Kim, 2009). This is depicted in the

overlapping plots of symptom severity and compulsivity in Figure 5.5 (point B), and as impulsivity was not measured in this sample, very little can be commented on as to its sub-clinical composition from these findings. Ettelt et al (2006) do contend with an absence of impulsivity compared to levels of compulsivity and OC symptoms in undiagnosed sub-clinical samples, albeit later research does contend with some impulsive phenomena within subclinical OCD compositions, though when population norms were controlled for, performance on relevant tasks and indices were within normative ranges (Zermatten and Van der Linden, 2008; Abramovitch et al, 2015). Subclinical impulsivity is represented by the orthogonal display at Point B, relative to compulsivity and symptom severity.

According to comparable severity benchmarks between the Y-BOCS and the OCI-R (as provided by Abramovitch et al, 2020), the sample in Study 4.2 can be described as having severe OCD (34.15 [SD=14.83]) which was greater than the symptom severity recorded in the sub-clinical group of Chapter 3 (25.69 [SD=15.67]), which can be considered moderate. The sample in Study 4.2 largely consists of those self-diagnosed with OCD, with only 35% of this sample declaring having been informed by a health care professional as having OCD, or currently receiving some form of treatment (medication, CBT, etc); formal diagnoses could not be assured. As such, the sample of Study 4.2 is taken to represent a window between symptoms and illness; a syndrome of diagnosable OCD symptomatology (see Figure 5.5, Point C).

As reflected in Figure 5.5 (point C), this window of syndromal OCD symptomatology displays parallel trajectories for all three variables; symptom severity, compulsivity and impulsivity as rated by the OCI-R, CPAS, and BIS-11, respectively. While impulsivity has previously been described as absent from OCD phenomenology – both in non- and sub-clinal

samples (at Points A and B; see Figure 5.5) – there may be inhibitory deficits that emerge only at greater levels of severity that contribute to the diagnostic profile of OCD. The analyses from Study 4.2 reveal that impulsivity begins to correlate with symptom severity only at higher levels. It is at this juncture – where symptomatology intensifies – that impulsive traits become noticeable. This suggests some contribution of both compulsivity and impulsivity that exacerbates symptomatology, past a threshold of functional impairment (Benzina et al, 2016). This threshold might represent the point at which neurocognitive trait features of OCD emerge more distinctly, marking the transition from syndrome to illness; from managing to not managing. Crucially, this implies that impulsivity, and specifically deficits in behavioural control, may represent a non-linear marker for diagnosable OCD. Unlike compulsivity, which shows a more continuous relationship with symptom severity, impulsivity appears categorical. It is largely absent in non- and sub-clinical groups but present in OCD-likely groups and those diagnosed with OCD (points C and D; see Figure 5.5). Impulsivity may reflect a dichotomous, categorical shift in the disorder's presentation.

As stated, compulsive and impulsive traits may contribute to the diagnostic class of OCD and that the emergence of neurocognitive features may serve as a catalyst for worsening symptomatology. The following exploratory mediation analysis was conducted to investigate the predictive capacities of compulsive and impulsive traits on OCD symptom severity via levels of dysfunctional cognitive and behavioural control. As the attentional impulsivity subscale of the BIS-11 correlated highly with the CPAS, OCPAS and the OCI-R (see Table 4.2.5), and with previous research implicating this facet of impulsivity as underscoring obsessional symptoms, this subscale was favoured over BIS-11 total scores as a predictor variable in the following mediation model (see Table 5.1) as this is a composite variable also consisting of motor and non-planning impulsiveness, both of which showed non-significant correlations with the tested variables.

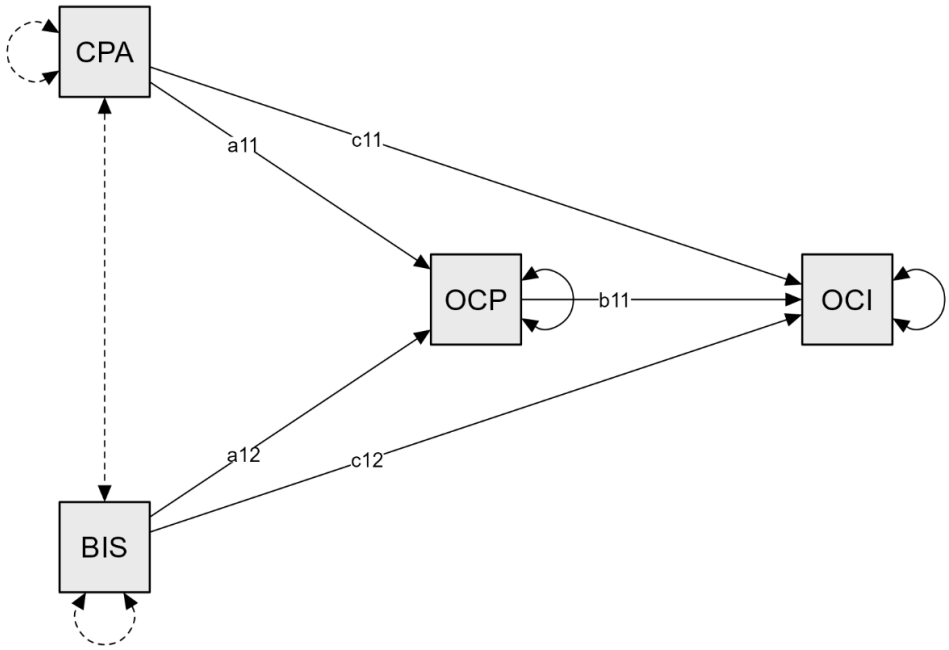
Table 5.1. Exploratory mediation analysis of OCI-R scores (n=78).

				95%CI				
Type	Effect	Estimate	SE	Lower	Upper	β	Z	p
Indirect								
	CPAS ⇒ OCPAS ⇒ OCI-R	0.60	0.16	0.29	0.92	.26	3.73	<.001
	BIS-11 Att ⇒ OCPAS ⇒ OCI-R	0.45	0.18	0.10	0.80	.12	2.54	<.05
Component								
	CPAS ⇒ OCPAS	1.79	0.27	1.27	2.31	.57	6.72	<.001
	BIS-11 Att ⇒ OCPAS	1.33	0.43	0.49	2.18	.26	3.08	<.05
	OCPAS ⇒ OCI-R	0.34	0.08	0.19	0.49	.45	4.49	<.001
Direct								
	CPAS ⇒ OCI-R	0.72	0.22	0.29	1.16	.31	3.25	<.001
	BIS-11 Att ⇒ OCI-R	0.66	0.30	0.07	1.26	.17	2.18	<.05
Total	CPAS ⇒ OCI-R	1.32	0.20	0.94	1.71	.56	6.68	<.001
	BIS-11 Att ⇒ OCI-R	1.11	0.32	0.48	1.74	.29	3.46	<.001

Note: Confidence intervals computed with method: Standard (Delta method)
Betas are completely standardised effect sizes. Significant findings are in bold

This mediation model underscores the vital influence that compulsive (CPAS) and impulsive (BIS-11 attentional impulsiveness) traits have in the manifestation and exacerbation of OCD symptomatology. Both compulsivity and impulsivity were found to directly predict symptom severity, as measured by the OCI-R, highlighting them as key risk factors in OCD. Importantly, OCPAS scores were shown to mediate these pathways predicting fluctuations in symptom severity. This suggests that executive impairments, cognitive and behavioural control deficits, serve as drivers of worsening symptomatology. This model accounts for 62% of the variance in OCI-R scores, offering a robust account of how these phenotypes contribute to this transitional period of syndromal OCD (see Figure 5.6). These findings highlight executive control deficits as central catalysts of symptom severity and novel targets for intervention.

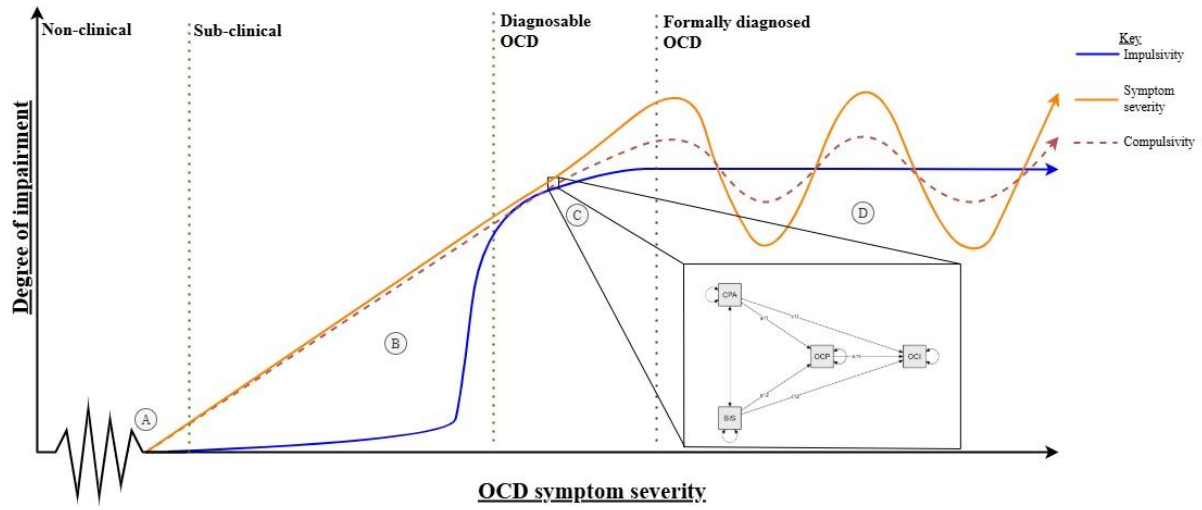
Figure 5.6. Mediation model predicting OCD symptom severity.



Note. CPA=Compulsive Personality Assessment Scale; BIS=Barratt Impulsiveness Scale-11 Attentional impulsivity; OCP=Obsessive-Compulsive Phenomenological Assessment Scale; OCI=Obsessive-Compulsive Inventory-Revised

Figure 5.7 offers a revised perspective of symptom severity and the phenotypic variance of compulsivity and impulsivity at point C given the findings of the exploratory mediation model that illustrates the mechanism driving the relationships between variables. Note that the waxing and waning of symptom severity may not be as severe as that depicted in Figure 5.7; this is an illustration of synthesised inferences from the different chapters and so the oscillations may be more subtle than this.

Figure 5.7. Updated visual representation of phenotypic variance parallel to diagnosable OCD severity.



The transition from a diagnosable syndrome to receiving a formal diagnosis of OCD may be marked by a delay in treatment seeking, which can lead to an initial exacerbation of symptomatology prior to treatment engagement (Fineberg et al, 2019; Pellegrini et al, 2025).

The absence of a relationship between impulsivity and symptom severity in Chapter 2 insinuates that impulsivity only correlates with symptom severity in this transitional interval (at Point C); as shown in the findings of Study 4.2. This suggests that impulsive traits become orthogonal to symptom severity after this point and remain relatively fixed in the degree of impairment for the remainder of the clinical course, given additional findings of orthogonality between impulsive traits and both age and duration of illness (see Chapter 2; Table 2.4). The same relationships (or lack thereof) were observed between compulsivity and age and duration of illness, however, as shown in Figure 5.3, compulsivity shares some degree of relationship with symptom severity and so there is some overlap in their respective fluctuations in clinically diagnosed OCD (see Figure 5.7; Point D). As between-group comparisons reveal equivocal impairments of cognitive flexibility and response inhibition

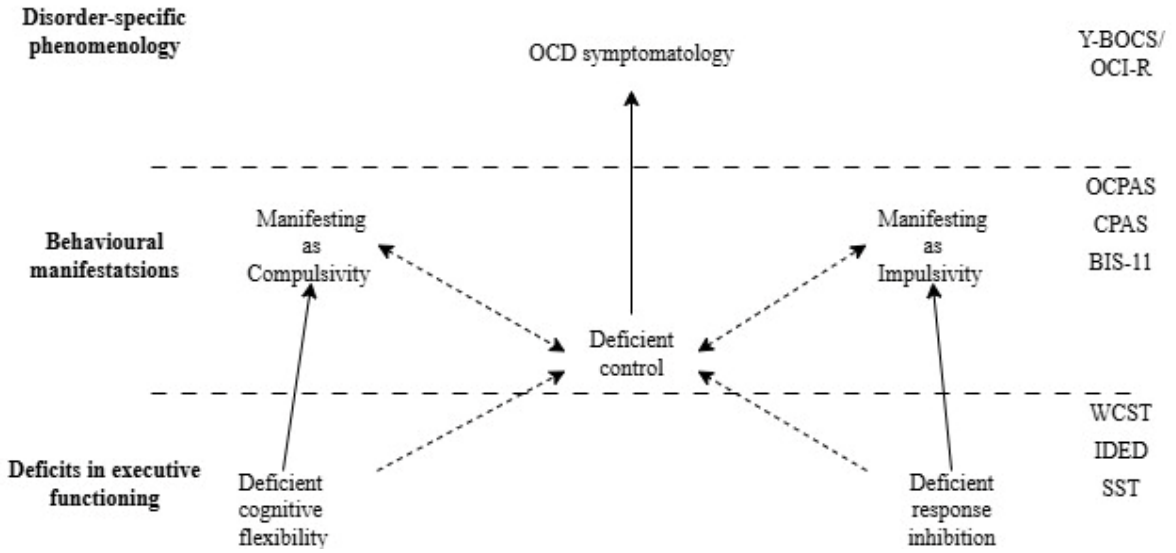
manifesting respectively as compulsivity and impulsivity in patients with OCD compared to healthy controls, this warrants equal exposure in the diagnostic assessment of OCD.

As such, the respective findings of Study 4.1, Chapter 3, Study 4.2, and Chapter 2 plot a course of phenotypic variance of compulsivity and impulsivity at salient junctures of symptom severity (non-clinical, sub-clinical, diagnosable OCD, and formally diagnosed OCD) which is consistent with the dimensional approach to OCD symptomatology (see Abramowitz et al, 2014). Further, the neurocognitive deficits explored in Chapter 2 (meta-analysis) appear to be almost categorical in that their manifestations are exclusive to illnesses severe enough to meet criteria for OCD - this cohort represents a blend of both state-like symptoms and trait-like executive deficits. The model presented in Figure 5.7 provides an eclectic approach to assessing psychopathology, considering both the dimensionality of OCD symptomatology and the categorical nature of neurocognitive deficits (Abramowitz et al, 2014; Chamberlain et al, 2017). Naturalistic and longitudinal analyses of these phenotypic manifestations are needed to substantiate categorical benchmarks for early identification of at-risk cohorts.

The development and validation of the OCPAS takes the evidence further by showing that deficits in the domain of self-control appear to be a driver of worsening OCD symptomatology. Themes of control do punctuate the definitions of cognitive inflexibility and deficient inhibitory control (Schel, Scheres, and Crone, 2014; Bukowski et al, 2024) which implies that the control in this respect is multidimensional. Work by Chamberlain et al (2017) does contend with a core underlying feature of deficient response control in OCD and that compulsivity and impulsivity may represent manifestations of this same central deficit. This could also be attributable to a shared underlying dysfunctional neural pathway between the striatum and the prefrontal cortices (Fineberg et al, 2014) which is reflected in the dual

impairments of cognitive flexibility and response inhibition observed in Chapter 2. Figure 5.8 provides a visual representation delineating the orientation and configuration of phenomenology and symptomatology relative to one another, as well as the levels and units of analysis. As shown, behavioural manifestations of compulsivity and impulsivity and underlying deficits of cognitive inflexibility and impaired response inhibition (as assessed by self-reports (the CPAS and the BIS-11) and neurocognitive task performances (the WCST, the IDED, and the SST)) both converge on themes of deficient control, as measured by the OCPAS, which ultimately underscores the exacerbation of OCD symptomatology (as measured by the OCI-R and the Y-BOCS). These findings provide a comprehensive multidimensional assessment of the construct of control within OCD phenomenology. Interestingly, the OCPAS was first developed in a non-clinical sample of little symptom severity suggesting that these domains of control are salient even at the lower end of the severity spectrum.

Figure 5.8. Layers of phenomenology, relationships, and associated measurement modalities.



It then follows that these categorical junctures of OCD symptom severity can be used to determine the threshold from symptoms to syndrome to illness, and from managing to not managing worsening OCD symptomatology, that can be utilised for targeted interventions. For example, Pellegrini et al (2023) showed that under transcranial direct current stimulation, patients with OCD performed better on the IDED (improved cognitive flexibility) when the Orbital-frontal cortex was stimulated, and likewise, performance on the SST (improved response inhibition) improved when the supplementary motor area was stimulated, thereby highlighting these facets of OCD phenomenology as novel personalised treatment targets with key neurocorrelates. However, assessing the sustained effect of improved executive faculties would prove difficult given the novelty of the tasks design to assess these phenomena insofar as repeated use being a reflection of practice and order effects as opposed to being a true reflection of treatment efficacy (Benedict and Zgaljardic, 1998; Bartels et al, 2010). The OCPAS may be of some utility in this respect by providing a self-rated mechanism of change, tracking the improvement of such neurocognitive traits given that the OCPAS appears to tap into the more subtle neurocognitive dimensions of OCD phenomena, and its strong relationship with a measure of OCD symptom severity, the OCI-R (Foa et al, 2002).

Limitations and methodological issues

Although the model presented in Figure 5.7 has implications for clinical courses in both early and late onset OCD, one shortcoming to consider in this theoretical model of symptomatology and neurocognitive phenomenology is that it is based on adjoining cross-sectional data; the model assumes equivocal levels at each stage for all participants considered under this model. Although Chapter 3 is fundamentally a longitudinal analysis, the

sample occupies only a single segment of the continuum explored in Figure 5.7 and so the external validity of this model is thereby restricted by inter-sample variances.

One methodological issue of the present work is the use of various neurocognitive measures utilised to estimate executive impairments across samples. Cognitive inflexibility was measured by the WCST and IDED in Chapter 2, the WCST and IDED in Chapter 3, and solely, the IDED in Chapter 4, Study 4.2. Likewise, response inhibition was measured by the SST, Go/no-go task, and the Continuous performance Task-II in Chapter 2, and solely, by the SST in Chapter 4, Study 4.2. Despite efforts in the described work to maintain consistency in the use of tasks and the associated outcome metrics, each use delivered a conflicting account of the respective deficit and also the corresponding relationship with OCD symptom severity.

The SST, measuring response inhibition, correlated with symptom severity scores in Study 4.2 which is different to the findings of Chapter 2 in that deficient response inhibition was shown to be orthogonal to symptom severity under both within-group correlations and meta-regression of between-group contrasts. The effect size of response inhibition in Chapter 2 consisted of both decisional and motor impulsivity and though the two fractions of impaired inhibitory control in subgroup analyses showed no difference, this is a composite representation of underlying deficient inhibitory control as opposed to a singular assessment in Study 4.2. With regard to cognitive inflexibility, Chapter 2 established within-group correlations between performance on the WCST and the IDED with symptom severity in formally diagnosed patients with OCD which was not completely reflected in undiagnosed subclinical participants in Chapter 3. Whilst a small correlation was revealed between the WCST and OCI-R scores in Chapter 3, this was not reflected on the IDED at a later testing stage. This, as discussed, could be attributed to (1) the more inflexible participants dropping out before IDED testing at three-months follow up and (2) differing symptom severities

between the two samples. However, further use of the IDED at greater symptom severities in the syndromal sample of Study 4.2 also revealed no significant relationship with OCI-R scores. This does suggest that impaired performance, on tasks of cognitive inflexibility and response inhibition, is exclusive to formally diagnosed cohorts. This is, however, contrary to previous research documenting these impairments in undiagnosed samples and first-degree relatives (Rajender et al, 2011), variability of impairments between clinical presentations of OCD (Vaghi et al, 2017), and ultimately, research refuting such impaired performance in those diagnosed with OCD (Fradkin et al, 2018). The issues described here may be a product of task impurity as well as inter- and intra-task heterogeneity, as explored in Chapter 3.1, General Discussion.

Further complication to the interpretation of neurocognitive deficits is the orthogonality between assessment modalities of the same construct; self-reports and neurocognitive tasks. Regarding the assessment of impaired cognitive flexibility in OCD phenomenology, the findings of Chapter 3 revealed a weak correlation between the WCST and the CPAS (r=0.16, p<.05) suggesting some convergence, albeit minimal, on the measurement of compulsive phenomena between the two modalities. However, this correlation was not replicated between the CPAS and the IDED which may allude to an underpowered sample (n= 28) as well as intra-task differences between the IDED and WCST as not being wholly interchangeable in measuring facets of flexibility. Despite the more inflexible participants dropping out after baseline testing (i.e., before the administration of the IDED), longitudinal analyses showed that self-ratings of compulsive traits remained stable over the 6-month testing period, highlighting a disparity between actual rigidity and self-perceived rigidity.

Exploration of this distinction between modalities in the assessment of impulsivity and underlying impairments in response inhibition is only discernible from Chapter 2 and Study

4.2. Only 4 papers (of a total 112) provided analyses assessing the convergence of assessment mode (see Table 2.3), all of which report non-significant correlations between the BIS-11 and both the SST (Boisseau et al, 2012; Sohn et al, 2014; Frydman et al, 2020) and the IGT (Grassi et al, 2015) suggesting a common discordance between the two assessment types insofar as assessing different dimensions of the same multi-dimensional phenotype. Conversely, the findings of Study 4.2 revealed a moderate relationship between the BIS-11 and the SST (r=0.37, p<.05; see Table 4.2.4) suggesting an impulsive disposition in this cohort that is accurately appraised and acknowledged, given the strength of the correlation. As the OC phenomena in the sample employed in Study 4.2 was of a syndromal composition (i.e., mostly representing a high symptomatic state with neurocognitive traits), this may implicate the insight of the diagnosed patients (those with a greater clinical severity) into their own dispositions given the orthogonality recorded in Chapter 2. Albeit, not directly assessed in the described work, this may suggest that insight diminishes in greater severities of OCD symptomatology (Cherian et al, 2012).

Alternatively, the very approach of the assessment may in fact influence the estimation of the latent trait. This point is one touched upon in Chapter 4 Study 4.2, in which it was argued that self-ratings are a commentary on the described latent traits, whereas, neurocognitive tasks offer a paradigm under which the latent trait can be directly observed under task demands; this becomes a difference of performance against the perception of performance (Dang, King, and Inzlicht, M. (2020). Where this may suggest that the neurocognitive tasks may be a more objective and accurate assessment, limitations such as the described task impurity hinder this interpretation. The rationale of the chosen task is often lacking and arbitrary and may depend on factors of convenience (availability, affordability, and accessibility) and so the task used may not be the most appropriate choice for the investigation. To address this issue, research by Gruner and Pittenger (2017) as well as Enkavi et al (2019a; 2019b) calls for a

homogenous and standardised use of these neurocognitive tasks and for this use to be based on the research question that is being investigated. It is the recommendation of this work that a formulated rationale be presented alongside the description of the measurement tool.

With these neurocognitive tasks being popular in experimental psychiatry for their novel assessment of latent traits, the participants employed (specifically those in Study 4.2) may have engaged with these tasks before and so the assessments may not be as novel as previously assumed (Benedict and Zgaljardic, 1998; Bartels et al, 2010) and so the reliability of these trait measures is questioned. The number of practice trials and number of trial in total does lessen the measurement 'noise' which may increase the reliability of the assessment. Indeed, Skirrow et al (2022) contend that CANTAB materials, such as the IDED, have sufficient test-retest properties, albeit there may be some difficulty with discerning true effects from practice effects. As discussed in Chapter 3.1, General Discussion, these tasks are merited for their ability to reproduce behavioural effects though at a cost for between-participant variance, which is ultimately needed for a reliable measurement of the latent trait (Haaf and Rouder 2017; Rouder, Kumar, and Haaf, 2019). A method of accounting for this would be to increase the sample size so as to increase the outcome variability; the lack of relationships observed for the IDED may be indicative of low outcome variability owing to a small sample size. As there are multiple confounds between uses, the reliability of these tasks cannot be assumed to be absolute (Zorowitz and Kiv, 2024) and so the ability to tap trait phenomena may be somewhat compromised. Although Condon et al (2012) support the use of the Stop-Signal Task as a trait measure of inhibitory control, Thunberg et al's (2024) comprehensive paper dissecting the use of the SST ruled this as a measurement of state rather than trait phenomena; as response inhibition is governed by situational determinants, tasks assessing this neurocognitive feature may not reflect stable traits. As trait phenomena is defined as being cross-contextual in that they predispose behavioural output across scenario

(Carr and Kingsbury, 1938), using a cross-sectional design may limit a true reflection of the trait. With standardised procedures implemented and with repeated-measures testing, this contention on the true nature of these neurocognitive tasks may addressed.

On the other hand, research does such suggest that patients are the experts of their own experience (Kendrick et al, 2016) and so their ratings may lie closer to a true representation of the latent trait. However, self-ratings are companion to meta-cognitive implications in that a participant may under- or over-rate their latent trait which may not be reflected in their task performances (Janeck et al, 2003; Dar et al, 2022). As described in Chapter 4, most self-rating scales have only been submitted to a principle components analysis (PCA; Abdi and Williams, 2010) which may in fact contribute to the observed variability as selection biases and other such demographic factors (Karpen, 2018) may have had some influence over the ratings that would have otherwise been eliminated had these scales been subjected to the rigorous psychometric testing that the OCPAS and the MOCI have been subject to under the Rasch model (Hodgson and Rachman, 1977; Inchausti and Delgado, 2012).

Another limitation of the present work pertains to the demographic variance across the presented chapters. Of the 920 participants across the three cross-sectional analyses (Chapter 3, n=252; Study 4.1, n=590; and Study 4.2, n=78), there was a bias towards Caucasian (78.48%) female participants (56.52%) that were educated to graduate status (66.85%). Overall, these samples may not be wholly reflective of the target population. Clinically relevant differences - such as symptom content and access to treatment - have indeed been reported between minority and non-minority patients with OCD (Jordan et al, 2020; Wang et al, 2021). However, no significant differences in symptom severity or functional impairments have been observed across ethnic backgrounds, suggesting that neurocognitive dysfunction may be a cross-cultural feature of the disorder. While this does suggest that there would be

little variation between ethnic groups on the observed effects, participants from minority ethnic backgrounds were underrepresented in the present analyses. As such, these present chapters are more reflective of western populations. Analyses tailored to examining ethnic variance on neurocognitive performance could not be performed due to insufficient subgroup sizes.

Previous research does contend with sex as playing a significant role in the presentation of OCD as females are 1.6 times more likely to develop OCD than males, develop OCD later in life compared to males, and commonly exhibit contamination/cleaning subtypes (Labad et al, 2007; Fontenelle and Hasler, 2008; Fawcett, Power, and Fawcett, 2020). However, little difference emerges between the sexes on neurocognitive performance (Mataix-Cols et al, 2006; Miranda et al, 2019; Monni et al, 2022) which is the case in these analyses as no between-group differences of sex were revealed on the WCST, IDED, or the SST. Moreover, the proportion of females per sample in the meta-analyses of Chapter 2 could not moderate effect sizes of both compulsivity and impulsivity task performance under meta-regression and so there is confidence that the over-representation of females in the described chapters is unlikely to have impacted the interpretation of findings.

Regarding education, previous research suggests that higher education is associated with improved neurocognitive performance on the WCST (Boone et al, 1993). However, the present chapters show that this relationship is not uniform across populations or tasks as there was no relationship observed between education and performance on the IDED in Study 4.2. Subgroup analyses could not be performed on the subclinical group of Chapter 3 due to non-proportionate group sizes. Conversely, higher education was associated with worse performance on the SST ($t(29)=3.63$, $p<.001$, $d=1.34$) in Study 4.2, though this may be a product of proactive slowing in the anticipation of the stop signal (Verbruggen and Logan,

2009). Previous research contends with a potential ceiling effect of IQ in that neurocognitive performance is predictive of below average and average IQ but less so of above average IQs (Diaz-Asper, Schretlen, and Pearlson, 2004), and though education may be regarded as a proxy for IQ, this is not always the case. This highlights the importance of considering both task demands and cognitive strategy when interpreting the role of education in neurocognitive performance.

This work is also limited by oversights on key domains of neurocognitive functioning; global impairments, and previous neurocognitive events – such as injuries, surgeries, or neurodevelopmental disorders. As noted in Chapter 2, the only paper (of 112) to have provided an assessment of global impairment was Frydman et al (2020). However, this paper only performed a between-group contrast between patients and healthy controls on this metric which showed greater impairment in the former (t=-17.75; df=32.00; p<.0001); correlational analyses were not conducted and so relationships between global impairment and symptom severity or neurocognitive performance could not be ascertained. As performance on these neurocognitive tasks assesses not only on the faculty in question (cognitive flexibility and response inhibition) but a number of cognitive domains as well (i.e., syntactic processing, working memory etc), assessing global functioning would outline the relative impact that this is having on overall task performance (Wang et al, 2002). As orbito- and dorsolateral prefrontal cortices are implicated as neurocorrelates of cognitive inflexibility and response inhibition, lesions may produce facsimiles of the performances observed in psychiatric groups (Eslinger and Grattan, 1993). For example, those diagnosed with ASD exhibit similar rigidity profiles to those observed in OCD (Lage, Smith, and Lawson, 2024). Factors such as these represent potential confounds to the analyses conducted on neurocognitive indices, though considering the relative performances across the tested samples, it seems that these scores were not influenced comorbidities. Findings from Chapter 2, 3, and Study 4.2

consistently show an orthogonality between comorbid conditions and neurocognitive performance and so the presentation of these deficits in this work are arguable reflective of OCD phenomenology irrespective of comorbidities. Though it is to be noted that the degree of additional (i.e., secondary to the OCD) psychiatric phenomena showed great variability within samples and so specific comorbid profiles could not be substantiated and examined.

Future directions

Manifestations of compulsivity and impulsivity, underpinned by deficiencies in cognitive flexibility and response inhibition, are trait level vulnerabilities and risk markers for the precipitation and maintenance of psychopathology, and as identified in Chapter 3, there is certainly a call for public health services to improve person-centred care accordingly, particularly for early identification and intervention for at-risk cohorts. Tailoring recognition and interventions may improve long-term outcomes (Brakoulias, Perkes, and Tsalamanios, 2017). For those identified as having higher levels of compulsivity and impulsivity, Acceptance and Commitment therapy as well as Cognitive behavioural therapy have some recorded utility in treating problematic behaviours emanating from compulsive and impulsive dispositions by manipulating cognitive distortions (Aguilar-Yamuza et al, 2024), as have pharmacological alternatives such as lithium, anticonvulsants and SSRIs (Conacher, 1997; Moeller et al, 2001; Rinne et al, 2002). Dialectical behavioural therapy has also been shown to have some merit in the treatment of emotional regulation, specifically for impulsive dispositions (Verhuel et al, 2003; Sigel et al, 2010). Though arguably, this assumes that patients with such executive deficits have the calmness and readiness required to tap into their cognitive functioning (Anda et al, 2006).

Alternatively, attempts have been made to target and remediate these executive deficits directly. Collectively referred to as Cognitive Rehabilitation by Bakizadeh et al (2024), these methods consist of organisational training, cognitive remediation, attention splitting, and goal management (all having implications and relevance for OCD). However, this review of randomised controlled trials was inconclusive concerning the efficacy of these methods in treating OCD, and as this review consisted of only 6 studies, the statistical powering is certainly questioned. van Passel et al' (2025) paper on the remediation of cognitive inflexibility showed no relationship between the treatment outcome for OCD and measures of cognitive inflexibility. The authors did question mechanisms of change in OCD, which as discussed prior is difficult with such tasks prone to practice effects upon repeated use. However, it is important to note an attrition rate of 50% between testing points and a lack of control for additional psychiatric phenomena which may have influenced influences interpretations. Further work by van Passel et al (2016; 2020) also report null-findings for the successful remediation of such cognitive deficits in OCD. Despite this, randomised controlled trials are too few in number and are not well controlled for discernible conclusions. Going forward, Trapp et al (2022) suggests that for a better utility of remediating practices, the interventions should be motivating, mirror everyday life, and for processing deficits to screened. Indeed, Cheng and Ebrahimi (2023) showed that the gamification of these therapeutic tools for mental health symptoms do have improved efficacy.

One of the critical areas for development is an improvement in assessment practices, particularly in relation to the differences and similarities between self-ratings and neurocognitive task performances. As described previously, there is a discordance both between and within these measurement types. A greater transparency is required to prevent a conflation of effects which would in turn enhance the overall interpretability of these latent cognitive phenotypes. A promising line of enquiry involves the estimation of meta-cognitive

abilities parallel to self-rating tools; as these scales are a commentary of the latent trait and are considered subjective, measuring the ability to acknowledge ones interoceptive and introspective abilities would equip a greater reliability of the self-rated trait (Goffin and Olsen, 2011; Zhang, 2010; Miller et al, 2013). Similarly, an assessment of global functioning (as described above) would provide an estimate of deficient cognitive processes that these neurocognitive task rely upon to assess the executive impairment in question. This would outline potential confounds. Moreover, a greater clarity of rationale as per the use of the task according to the research question would provide greater transparency (Enkavi et al, 2019a; 2019b). Finally, uniformity of task outcome metric would improve consistency across studies thereby eliminating inter-study the heterogeneity documented in Chapter 2.

Moreover, future research should aim to fill the gap in assessment of decision-making impairments according to the depth afforded to cognitive inflexibility and response inhibition in this work. As outlined in Chapter 2, patients with OCD are equivocally impaired in decisions-making processes as they are in motor impulsivity, and as the response inhibition effect size that was subjected to meta-regression was a composite of both fractions of deficient inhibition control, decision-making impairments may also be orthogonal to symptom severity and other salient clinical factors (Cavedini et al, 2002). As decision-making was not assessed in chapters 3 and 4, its composition in both clinical and sub-clinical cohorts requires substantiation to observe any similar (or indeed dissimilar) trajectories adjacent to compulsivity and symptom severity (such as that captured in Figure 5.7).

As a formally diagnosed cohort could not be assured in Study 4.2, further validation of the OCPAS in a cohort that is actively engaging with primary and/or secondary mental health services is warranted. Even at lower severities, the subfactors of the OCPAS appear to be fundamental OC phenomena that may prove to have etiological salience; markers for early

identification for at-risk cohorts. Moreover, as the OCPAS is conceptually consistent with neurological findings of functional connectivity, it would be interestingly and valuable to examine associations between the factor structure of the OCPAS and fMRI under inflexibility and inhibitory paradigms.

As discussed, unique phenotypical and symptomatologic compositions differentiate symptoms, syndrome, and illness. These distinctions provide important benchmarks for the transitions between non-clinical and clinical states of symptoms and impairments. Importantly, there is a critical need to develop the demarcations of severity junctures (Apter et al, 1996). The research reported here is cross-sectional and as such, the proposed benchmarks are empirically-driven but remain conjectural. To bridge this gap, naturalistic and longitudinal analyses are required to validate and extend these findings.

CHAPTER 6: CONCLUSIONS

This thesis explores executive deficits, specifically cognitive inflexibility and impaired response inhibition manifesting as cognitive phenotypes of compulsivity and impulsivity, in individuals diagnosed with OCD. This research was guided by three core aims: (1) to examine how these latent cognitive phenotypes relate to OCD symptomatology, (2) to evaluate the accuracy of existing screening and measurement tools, and (3) to assess the relationships of these phenotypes with clinically significant variables.

Chapter 2 examined neurocognitive compulsivity and impulsivity in clinically diagnosed patients with OCD. Analyses revealed dual and comparable impairments of compulsivity and impulsivity. Furthermore, meta-regression of between group-contrasts revealed an independence of these impairments from symptom severity. On the other hand, within group correlations revealed that compulsivity fluctuated in parallel with symptom severity, while impulsivity appeared to be categorical and only manifested at greater levels of symptom severity. This highlighted distinct underlying cognitive mechanisms. However, the accurate assessment of these impairments is complicated by methodological inconsistencies across both self-report scales and neurocognitive tasks. Moreover, meta-regressions revealed orthogonality with other typical clinical variables such as duration of illness, comorbidities, and depression and anxiety symptomatology. This indicated that current assessment models of OCD are suited to the detection of symptoms (states) but not deficits (traits).

Chapter 3 then examined OCD symptoms and traits in a subclinical cohort of the general population of the UK during the lifting of pandemic restrictions and adapting to the ‘new normal’. This Chapter showed that whilst self-ratings of compulsivity correlated with symptom severity, neurocognitive compulsivity did not. This of course contrasts with the preceding chapter, though this may be a product of differing severities between chapters 2

and 3. These analyses also showed that a contribution of both state (symptom severity) and trait variables (cognitive inflexibility and impaired response inhibition), whilst not exclusively correlating, both contributed to exacerbations in adjustment difficulty. Contrasting and comparing chapters 2 and 3 revealed that the modes of assessment (self-ratings vs neurocognitive tasks) have a bearing on the estimation of executive deficits and that this may be due to meta-cognitive commentaries of self-rating tools and also of issues with task impurity and heterogeneity among neurocognitive tasks. This may ultimately have a knock-on effect with their respective relationship with symptom severity. The differences between self-ratings and neurocognitive tasks, and their respective bearing on OCD symptom severity, identified a need for a scale that was able to detect subtle key neurocognitive components of the disorder that is accessible for both clinical and subclinical cohorts.

Chapter 4 reviewed the current clinician- and patient-rated outcome measures of OCD and their respective sensitivity to executive deficits. It was revealed that there is a limited capacity of these scales to capture such functional impairments and that most scales are not developed under rigorous psychometric testing, only under exploratory factor analysis, and so there may be inherent biases within current scales, namely the OCI-R and the Y-BOCS. Study 4.1 described the development of the OCPAS. Subjecting the initial corpus of 59 items to both classical and modern test theoretical frameworks proved efficacious in reducing the scale to an acceptable size whilst maintaining optimum key psychometric properties. The final 26-item scale was labelled the Obsessive-Compulsive Phenomenology Assessment Scale (OCPAS) and consists of three subfactors; Cognitive control, Behavioural control, and Anankastic goals. The scale uses a five-point Likert scale to assess participants' deficient flexibility and inhibitory control.

Study 4.2 described the validation of the OCPAS in a diagnosable OCD cohort. The OCPAS demonstrated construct validity with measures of compulsivity and impulsivity and is able to distinguish between those that do and do not have diagnosable OCD. The OCPAS is also consistent with previous cognitive-behavioural frameworks of OCD phenomenology. Within this chapter, the same relationships between the IDED, CPAS and the OCI-R from chapter 3 are observed in that self-rated compulsivity correlates with symptom severity, but not neurocognitive performance on tasks of compulsivity and so this may suggest that neurocognitive deficits of compulsivity may emerge at greater severities as shown in Chapter 2. Differently, the BIS-11, SST and the OCI-R all interrelate and so this may suggest that impulsivity is more accurately rated than compulsivity. This may also hint at a window where impulsivity does correlate and interact with compulsive features to exacerbate symptomatology; an interaction between state and trait variables that then becomes orthogonal but stable in greater severities, as observed in Chapter 2.

Chapter 5 offers a consolidation of the empirical evidence by presenting a combination of categorical and dimensional symptomatology whereby each chapter represents a key window of phenotypic variance that is plotted against a continuum of OCD symptom severity: Study 4.1, non-clinical; Chapter 3, sub-clinical; Study 4.2 diagnosable OCD; and Chapter 2, formally diagnosed OCD. Compulsivity appears to share similar trajectories to symptom severity across the continuum and shares similar oscillations to symptom severity in clinical cohorts, given the within-group correlations in Chapter 2 (as shown in Figure 5.7), whilst impulsivity appears to be a dichotomous categorical variable, only becoming salient during a diagnosable window after which it becomes a stable fixed trait that is relatively unchallenged by current interventions. A model is presented whereby the interaction of both impulsivity and compulsivity interact (a blend of both state and trait variables) to catalyse exacerbations in OCD symptomatology enough to meet diagnostic criteria for OCD.

In concert, the work provides novel insights into the nuanced relationship between these executive impairments of compulsivity and impulsivity and OCD symptomatology and that the identification of trait-like impulsivity and compulsivity supports their use as biobehavioural risk markers, which may help detect individuals at risk before clinically significant OCD emerges. Moreover, the differences between self-report and neurocognitive results suggest the importance of integrating multiple assessment modalities, especially in diagnostic and research settings (Snyder, Friedman, and Hankin, 2020). Considering targeted interventions, if impulsivity emerges at greater symptom severities, then this may require distinct intervention strategies compared to compulsivity (which appears more fluid given its described correlations with symptom severity). Finally, this thesis challenges the traditional view of OCD as a uniform disorder of compulsivity by introducing a two-dimensional executive model involving both compulsivity and impulsivity and their dynamic interplay; and that symptoms and deficits are orthogonal and represent different aspects of OCD phenomenology. The symptoms represent key state phenomena and may exist without the trait impairments in subclinical OCD but co-occur in clinical OCD.

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*: study included in the meta-analysis.

GLOSSARY

Patients with OCD	Those individuals whose symptoms are so severe that they meet criteria for a formal diagnosis of OCD; Each patient has a unique composition of obsessions and compulsions with differing content and severity.
Subclinical OCD	Those whose symptomatic profiles exist before this point of diagnosis and lacks the significant functional impairment that is so disabling in clinical manifestations of the disorder though still negatively impacting quality of life.
Syndromal OCD	Those whose symptomatic compositions are diagnosable and show a great deal of impairment with daily life. A point where symptomatic presentation have become disabling and qualify for a diagnosable condition.
Executive function	This term pertains to a collection of latent cognitive abilities that are responsible for successful goal-directed behaviour and adaptation to changing environments. Executive functioning develops early and matures between adolescence to early adulthood, parallel to the development of the prefrontal cortex.
Cognitive flexibility	The effective modification of thinking and behaviour to meet contextual demands based on changing environments and information, as well as successfully switching between various tasks and concepts.
Cognitive inflexibility	A diminished ability to successfully shift attention between activities, situations, and stimuli manifesting in a compulsive disposition demarked by tendencies of perseveration, resistance to change and an inability to adapt.
Response inhibition	The executive ability to successful suppress and mitigate unwanted thoughts or automatic behaviours that are incongruent or inappropriate given the situational context or particular goals of focus.
Impaired response inhibition	The diminished capacity to stop, prevent, or delay an action or thought; and it is in the absence of refined response inhibition that prepotent actions or thoughts cannot be controlled or substituted for more adaptive alternatives.
Phenotype	Cognitive and behavioural presentations/manifestations of underlying impaired executive function. Latent cognitive phenotypes represent putative transdiagnostic markers for

	executive control as they constitute discrete observable and measurable cognitive profiles that are linked to established neurobiological processes that underpin the phenomenological manifestations.
Compulsivity	This represents a drive to perform repetitive habitual actions according to preestablished rigid rules. Compulsivity is borne of a deficit in the previously mentioned executive function, cognitive flexibility.
Motor impulsivity	The inability to supress an initiated automatic action in which the responding can be described as premature or anticipatory.
Decision-making impulsivity	Decisions that are made without full knowledge of the current situational landscape, with little or no regard for long-term consequences, and with a preference for short-term transient rewards.
States	A temporary and transient condition, expressed for a short duration, influenced by external factors and is dependent on context.
Traits	Inherent and stable characteristics that consistently influence behaviour across situations and scenarios. They are internally driven.
Self-rating tools	Standardised questionnaires developed to capture latent states and traits of interest. They are easy to administer and are a quick and convenient method of data collection for both research and practical (i.e., clinical) purposes. Participants and/or patients are asked to score their own experiences along a continuum according to predetermined statements.
Neurocognitive tasks	A non-invasive interactive devices developed to examine the phenotype of interest under an applied scenario so as to observe underlying deficits in cognition such as attention, memory, reaction time, and language.

APPENDICES

Appendix A. PRISMA checklist

Section and Topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	Page 1
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	Page 2
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Page 3-6
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Page 6-7
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Page 10-11
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Page 8
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Page 8
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Page 8
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	Page 10-11
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Page 10-13
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Page 11-13
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Page 12

Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Page 12-13
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Page 10-13
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	Page 15-16
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	N/A
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	Page 12-14
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Page 14
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	Page 13-14
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Page 12
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Page 14
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Fig. 1 and page 14-15
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Page 15-16
Study characteristics	17	Cite each included study and present its characteristics.	Table 1
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Table 3
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Page 16-22 & fig. 2, 3, 4, 5, 6, & 7
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	Table 1 &3, page 19-20

	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Page 16-22
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Page 28-29
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	Page 17-18
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	Page 19-20
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	Page 16-17
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Page 22-33
	23b	Discuss any limitations of the evidence included in the review.	Page 31-33
	23c	Discuss any limitations of the review processes used.	Page 31-32
	23d	Discuss implications of the results for practice, policy, and future research.	Page 30-31
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	Page 8
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	Page 8
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	N/A
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Page 34
Competing interests	26	Declare any competing interests of review authors.	Page 34-35
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	N/A

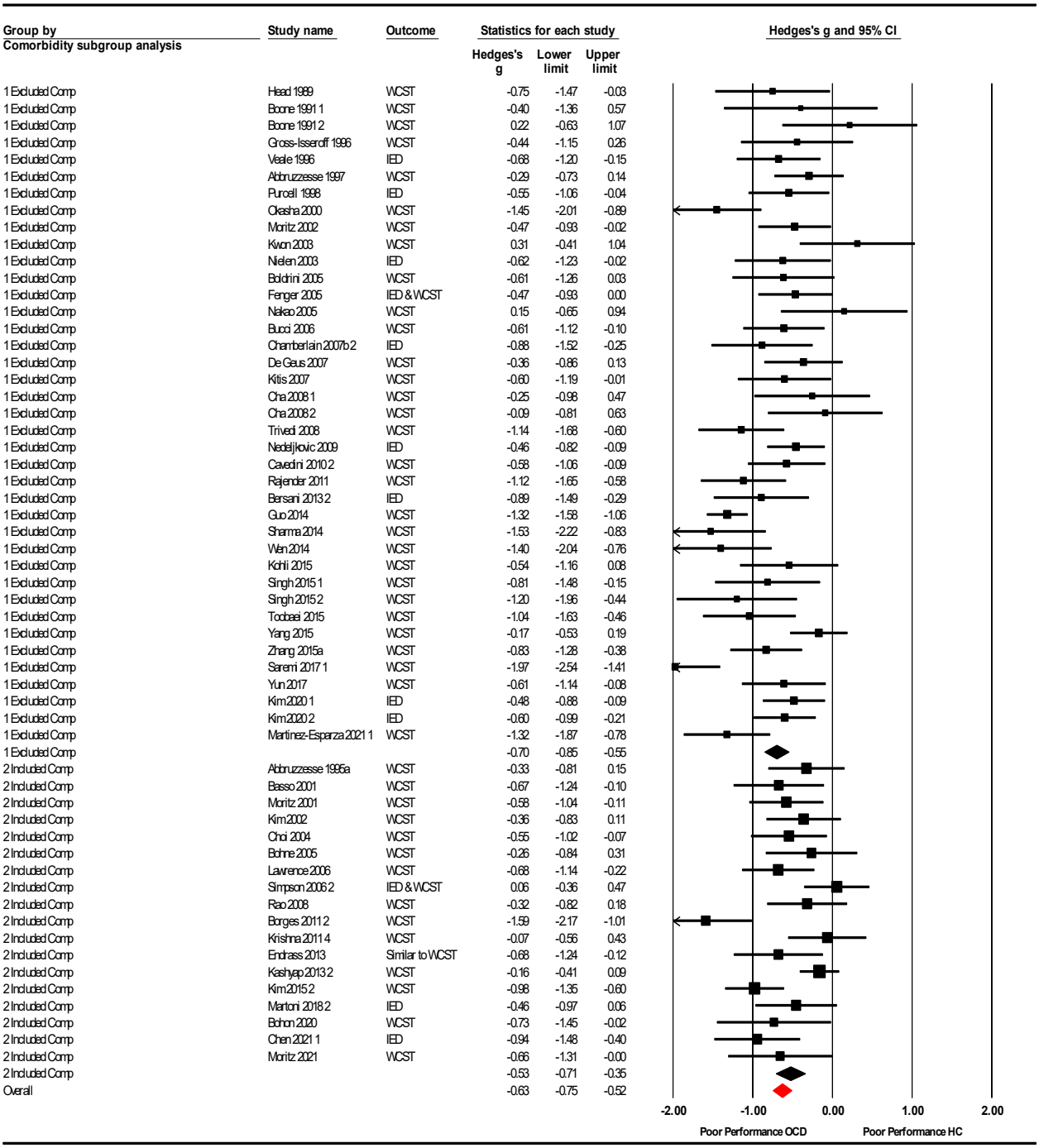
Appendix B: Listed comorbidities in included studies.

Study names	Comorbidities reported
Abbruzzesse 1995a	17 of 33 had a comorbid disorder; exact disorders not specified.
Basso 2001	MDD (n=4), Chronic tic (n=1), Schizoaffective disorder (n=1).
Moritz 2001	MDD (N=10), Anxiety disorder (n=4).
Kim 2002	MDD (n=5), Phobia (n=1), Bulimia Nervosa (n=1)
Choi 2004	MDD (n=1)
Bohne 2005	10 reports of comorbid disorder; exact disorders not specified.
Lawrence 2006	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)
Simpson 2006	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)
Kim 2007	Depression (n=2)
Roth 2007	MDD (n=2)
Bohne 2008	11 cases of comorbidity; not specified.
Rao 2008	MDD (n=15), Suicide risk (n=5), Panic disorder (n=4), Agoraphobia (n=2), GAD (n=2), Social phobia (n=1)
Page 2009	Dysthymic disorder (n=2), past history of MDD (n=3), Alcohol dependence (n=1).
Blom 2011	Depression (n=10), Eating disorder (n=1), ADHD (n=1)
Borges 2011	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)
da Rocha 2011	Social phobia (n=22), GAD (n=22), Agoraphobia (n=19), Depressive disorder (n=16), Panic disorder (n=15), Bipolar II disorder (n=2).
Krishna 2011	Dysthymia (n=4), Specific phobia (n=3), Social phobia (n=2), GAD (n=2).
Boisseau 2012	MDD (n=1), MDD & Social Phobia (n=1), Panic disorder (n=1), GAD (n=1).
de Wit 2012	22 reports of comorbid disorder; exact disorders not specified.
Boisseau 2013	40% of the sample had a comorbid disorder; exact diagnoses and counts not given.
Endrass 2013	Panic disorder (n=5), MDD (n=4), GAD (n=4), Agoraphobia (n=3), Specific phobia (n=2), Social phobia (n=1), Hypochondriasis (n=1)
Kashyap 2013	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)
Tolin 2014	Anxiety disorders (n=8), Depressive disorders (n=8).
Kim 2015	MDD (n=8), Panic disorder (n=3), Bipolar II Disorder (n=2), Tic disorder (n=2), Social phobia (n=1), BDD (n=1)

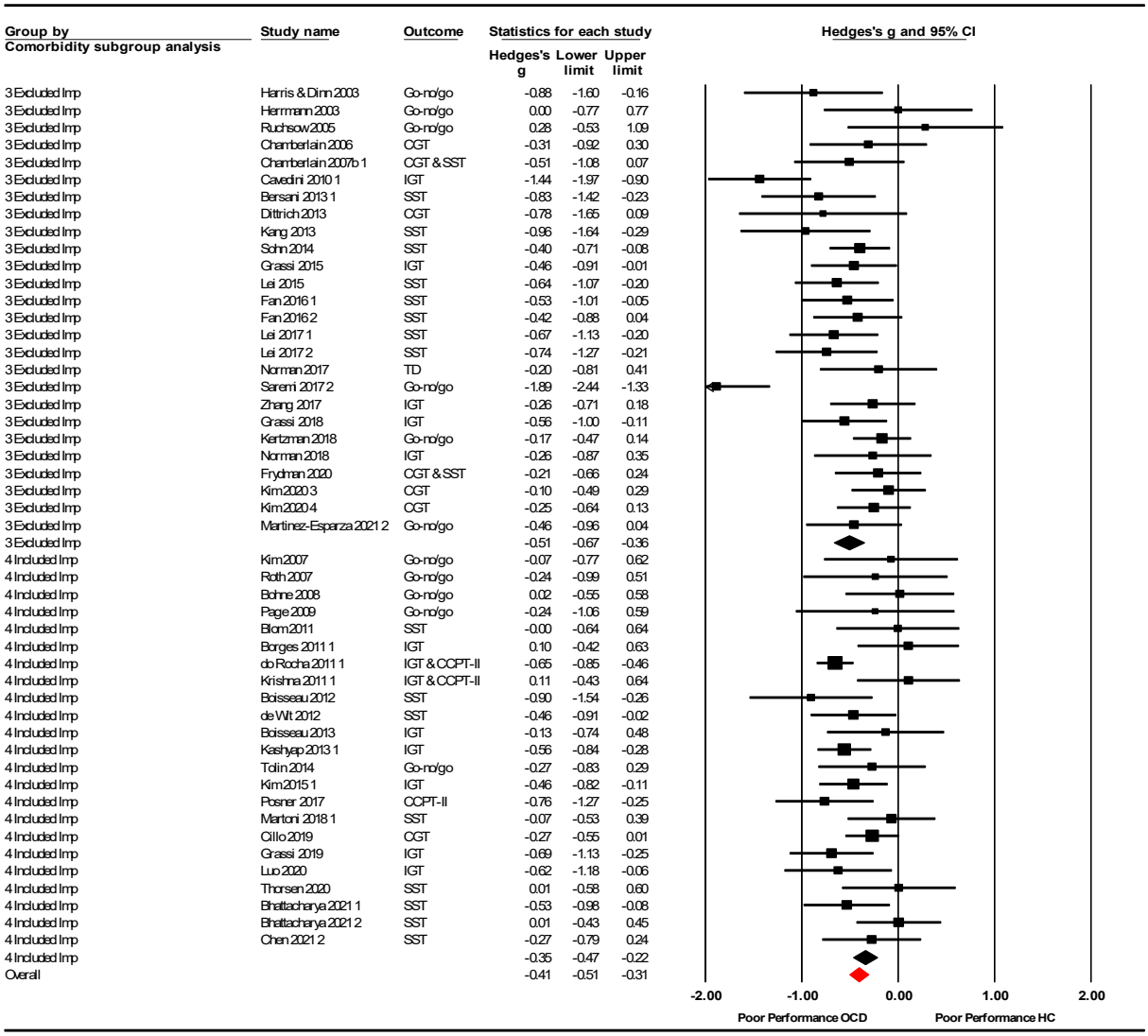
Posner 2017	History of MDD (n=4), Social phobia and history of MDD (n=2), Specific phobia (n=1), Social phobia (n=1), Special phobia, Social phobia, and history of MDD (n=1), Binge eating disorder, Social phobia, and history of MDD (n=1)
Martoni 2018	Anorexia Nervosa (n=1), Tourette syndrome (n=1), Skin picking disorder (n=1), Gambling disorder (n=1), Panic Disorder (n=1), MDD (n=1), Trichotillomania (n=1), Hoarding disorder (n=1)
Cillo 2019	Social Phobia = 1; Mood disorders = 4; Dysmorphophobia = 1.
Grassi 2019	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).
Bohon 2020	GAD (n=3), Specific phobia of crowds (n=1), Social phobia (n=1)
Luo 2020	OCPD; exact count not specified.
Thorsen 2020	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).
Bhattacharya 2021	**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)
Chen 2021	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).
Moritz 2021	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

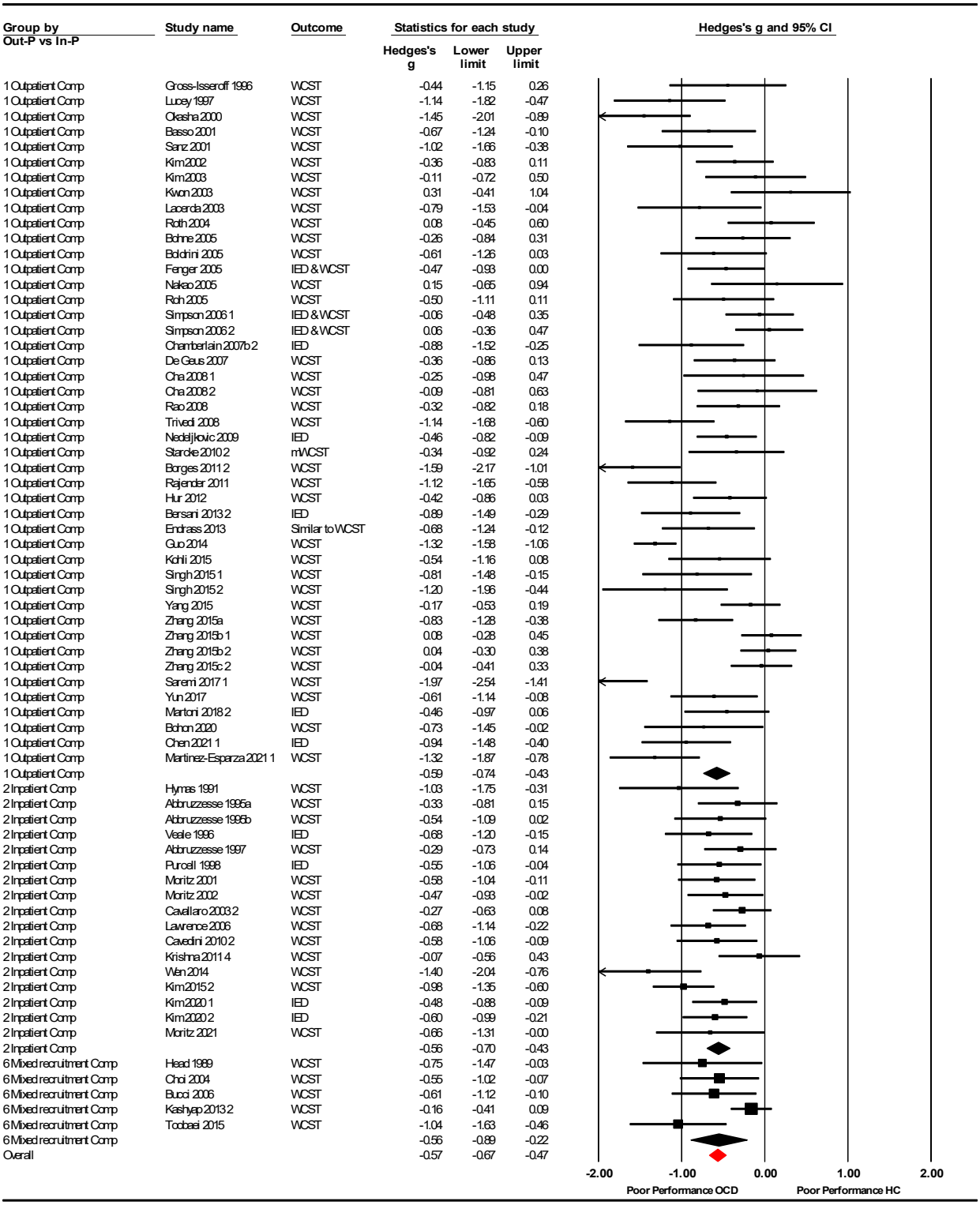
Appendix C. Subgroup analysis of compulsive task performance between OCD and healthy controls moderated by the inclusion and exclusion of comorbidities.



Appendix D: Subgroup analysis of impulsive task performance between OCD and healthy controls moderated by the inclusion and exclusion of comorbidities.

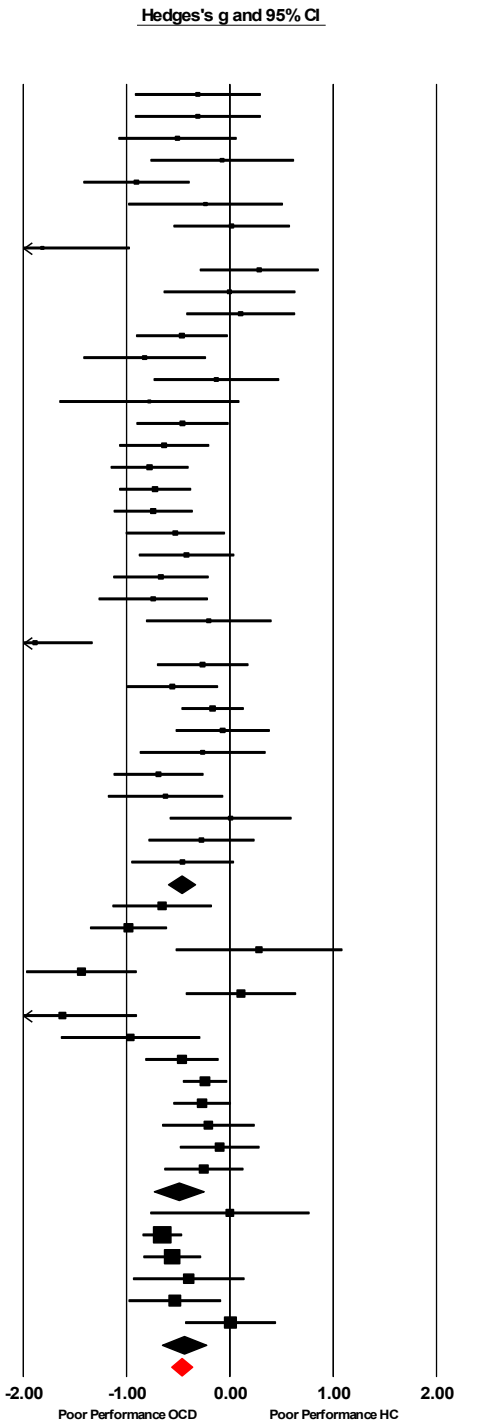


Appendix E: Subgroup analysis of compulsive task performance between OCD and healthy controls moderated by clinical status.

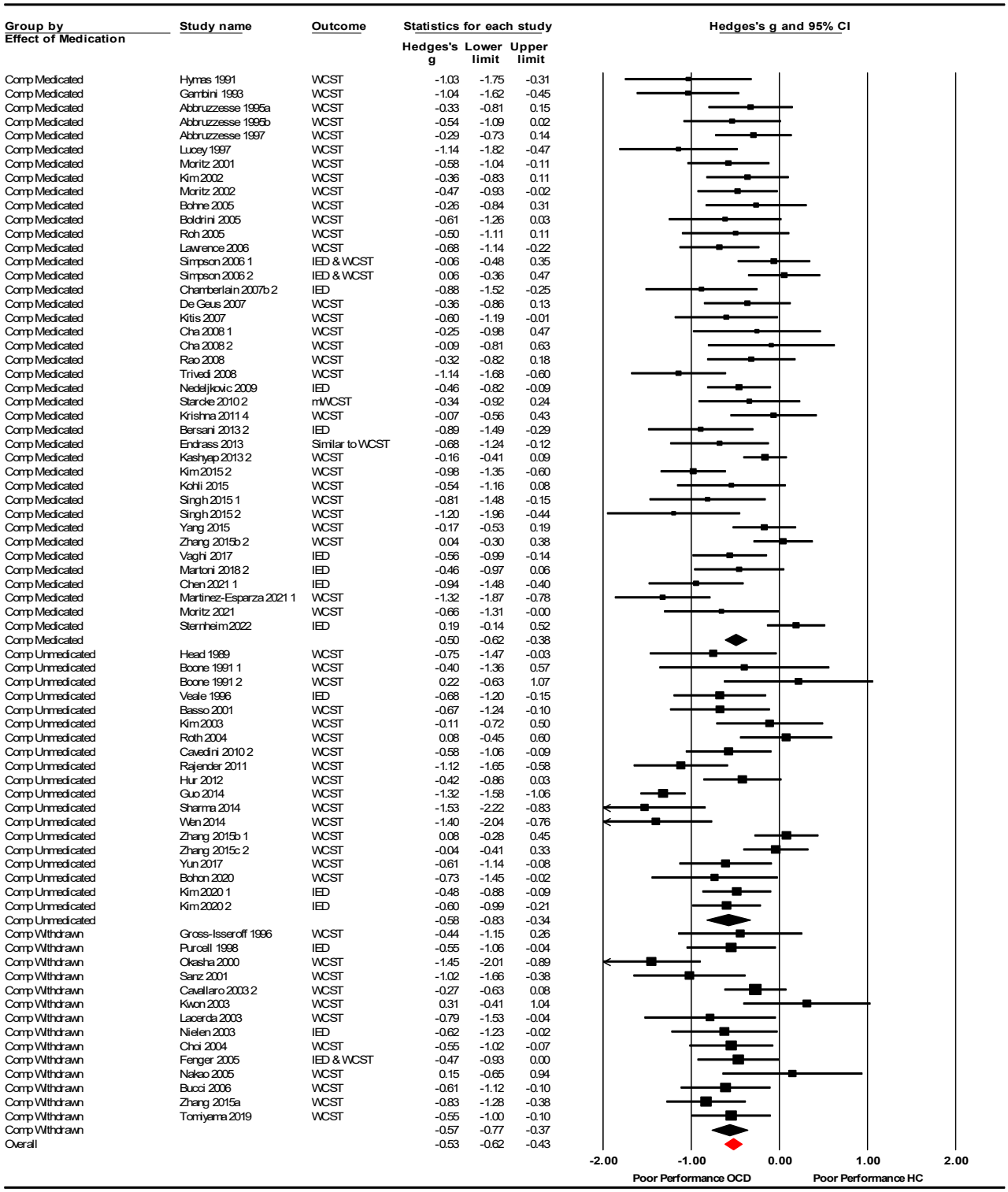


Appendix F: Subgroup analysis of impulsive task performance between OCD and healthy controls moderated by clinical status.

Group by Out-P vs In-P	Study name	Outcome	Statistics for each study		
			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

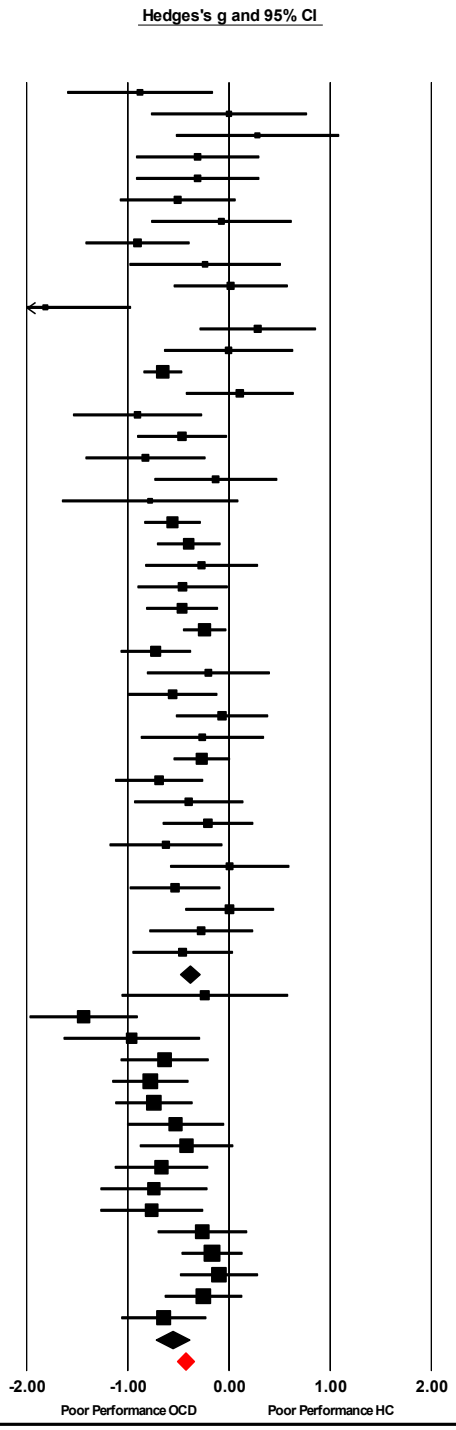


Appendix G: Subgroup analysis of compulsive task performance between OCD and healthy controls moderated by effects of medication.



Appendix H: Subgroup analysis of impulsive task performance between OCD and healthy controls moderated by effects of medication.

Group by Effect of Medication	Study name	Outcome	Hedges's g	Lower limit	Upper limit
Imp Medicated	Harris & Dinn 2003	Go-no/go	-0.88	-1.60	-0.16
Imp Medicated	Herrmann 2003	Go-no/go	0.00	-0.77	0.77
Imp Medicated	Ruchsow 2005	Go-no/go	0.28	-0.53	1.09
Imp Medicated	Chamberlain 2006	CGT	-0.31	-0.92	0.30
Imp Medicated	Chamberlain 2007a 1	CGT	-0.31	-0.92	0.30
Imp Medicated	Chamberlain 2007b 1	CGT & SST	-0.51	-1.08	0.07
Imp Medicated	Kim 2007	Go-no/go	-0.07	-0.77	0.62
Imp Medicated	Menzies 2007	SST	-0.90	-1.42	-0.39
Imp Medicated	Roth 2007	Go-no/go	-0.24	-0.99	0.51
Imp Medicated	Bohne 2008	Go-no/go	0.02	-0.55	0.58
Imp Medicated	Starcke 2009	IGT	-1.81	-2.66	-0.97
Imp Medicated	Starcke 2010 1	IGT	0.28	-0.29	0.86
Imp Medicated	Blom 2011	SST	-0.00	-0.64	0.64
Imp Medicated	do Rocha 2011 1	IGT & CCPT-II	-0.65	-0.85	-0.46
Imp Medicated	Krishna 2011 1	IGT & CCPT-II	0.11	-0.43	0.64
Imp Medicated	Boisseau 2012	SST	-0.90	-1.54	-0.26
Imp Medicated	de Wit 2012	SST	-0.46	-0.91	-0.02
Imp Medicated	Bersani 2013 1	SST	-0.83	-1.42	-0.23
Imp Medicated	Boisseau 2013	IGT	-0.13	-0.74	0.48
Imp Medicated	Dittrich 2013	CGT	-0.78	-1.65	0.09
Imp Medicated	Kashyap 2013 1	IGT	-0.56	-0.84	-0.28
Imp Medicated	Sohn 2014	SST	-0.40	-0.71	-0.08
Imp Medicated	Tolin 2014	Go-no/go	-0.27	-0.83	0.29
Imp Medicated	Grassi 2015	IGT	-0.46	-0.91	-0.01
Imp Medicated	Kim 2015 1	IGT	-0.46	-0.82	-0.11
Imp Medicated	Martoni 2015	IGT	-0.24	-0.46	-0.03
Imp Medicated	Zhang 2015b 4	IGT	-0.72	-1.07	-0.37
Imp Medicated	Norman 2017	TD	-0.20	-0.81	0.41
Imp Medicated	Grassi 2018	IGT	-0.56	-1.00	-0.11
Imp Medicated	Martoni 2018 1	SST	-0.07	-0.53	0.39
Imp Medicated	Norman 2018	IGT	-0.26	-0.87	0.35
Imp Medicated	Cillo 2019	CGT	-0.27	-0.55	0.01
Imp Medicated	Grassi 2019	IGT	-0.69	-1.13	-0.25
Imp Medicated	Yu 2019	SST	-0.40	-0.94	0.14
Imp Medicated	Frydman 2020	CGT & SST	-0.21	-0.66	0.24
Imp Medicated	Luo 2020	IGT	-0.62	-1.18	-0.06
Imp Medicated	Thorsen 2020	SST	0.01	-0.58	0.60
Imp Medicated	Bhattacharya 2021 1	SST	-0.53	-0.98	-0.08
Imp Medicated	Bhattacharya 2021 2	SST	0.01	-0.43	0.45
Imp Medicated	Chen 2021 2	SST	-0.27	-0.79	0.24
Imp Medicated	Martinez-Esparza 2021 2	Go-no/go	-0.46	-0.96	0.04
Imp Medicated			-0.39	-0.49	-0.29
Imp Unmedicated	Page 2009	Go-no/go	-0.24	-1.06	0.59
Imp Unmedicated	Cavedini 2010 1	IGT	-1.44	-1.97	-0.90
Imp Unmedicated	Kang 2013	SST	-0.96	-1.64	-0.29
Imp Unmedicated	Lei 2015	SST	-0.64	-1.07	-0.20
Imp Unmedicated	Zhang 2015b 3	IGT	-0.78	-1.16	-0.40
Imp Unmedicated	Zhang 2015c 1	IGT	-0.74	-1.13	-0.36
Imp Unmedicated	Fan 2016 1	SST	-0.53	-1.01	-0.05
Imp Unmedicated	Fan 2016 2	SST	-0.42	-0.88	0.04
Imp Unmedicated	Lei 2017 1	SST	-0.67	-1.13	-0.20
Imp Unmedicated	Lei 2017 2	SST	-0.74	-1.27	-0.21
Imp Unmedicated	Posner 2017	CCPT-II	-0.76	-1.27	-0.25
Imp Unmedicated	Zhang 2017	IGT	-0.26	-0.71	0.18
Imp Unmedicated	Kertzman 2018	Go-no/go	-0.17	-0.47	0.14
Imp Unmedicated	Kim 2020 3	CGT	-0.10	-0.49	0.29
Imp Unmedicated	Kim 2020 4	CGT	-0.25	-0.64	0.13
Imp Unmedicated	Tomiyama 2021	SST	-0.65	-1.07	-0.22
Imp Unmedicated			-0.56	-0.73	-0.40
Overall			-0.43	-0.52	-0.35



Appendix I. Reported medication in medicated samples.

Study	Type of medication (n)
Hymas 1991	Imipramine (n=1), diazepam with dothiepin (n=1), zuclopentixol (n=1), flupenthixol (n=1)
Gambini 1993	Antidepressants (n=13); Clomipramine and fluvoxamine
Abbruzzese 1995a	Fluvoxamine
Abbruzzese 1995b	Fluvoxamine
Abbruzzese 1997	Fluvoxamine
Lucey 1997	7 were medicated, drug not specified
Moritz 2001	15 were medicated; sertraline, fluoxetine, paroxetine. Risperidone (n=5), Olanzapine (n=2), clonazepam (n=3), trazodone (n=2)
Moritz 2002	Antidepressants (n=14), benzodiazepine (n=2)
Harris and Dinn 2003	SSRI or clomipramine (n=12)
Hermann 2003	Fluoxetine (n=2), temazepam and lorazepam (n=1), clomipramine, mianserine perizine, and clopenthixol (n=1), clomipramine (n=3), doxepin and lorazepam (n=1)
Kim 2003	Paroxetine (n=2)
Bohne 2005	Psychtropic medication (n=13) – not specified
Boldrini 2005	Tricyclic antidepressants (clomipramine, imipramine) or SSRI (fluvoxamine, paroxetine, sertraline)
Roh 2005	SSRI (n=8); Sertraline, fluoxetine, fluvoxamine, paroxetine, and clomipramine. SSRI with risperidone (n=11). Olanzapine (n=2)
Ruchsow 2005	Medication names and quantities not specified
Chamberlain 2006	SSRI (n=16)
Lawrence 2006	Antidepressants: Citalopram (n=4), Clomipramine (n=2), Fluoxetine (n=8), Fluvoxamine (n=3), Paroxetine (n=8), Venlafaxine (2). Additional medications: Diazepam (n=2), Zopiclone (n=2), Buspirone (n=1), and amitriptyline (n=1)
Simpson 2006	SSRI (n=17), SRI + another medication ((n=5); Benzodiazepine (n=3), Bupropion (n=1, Buspirone (n=1)), and Dextroamphetamine (n=1)
Chamberlain 2007a	SSRIs
Chamberlain 2007b	SSRIs
de Geus 2007	Paroxetine (n=14), Citalopram (n=11), venlafaxine (n=5), fluoxetine (n=4), fluvoxamine (n=3), imipramine (n=1), clomipramine (n=1)
Kim 2007	Sertraline, fluoxetine, fluvoxamine, risperidone (n=2), and clonazepam (n=4)
Kitis 2007	Antidepressants (n=11), antidepressants with antipsychotics (n=2)
Menzies 2007	SSRI (n=21), Clomipramine (n=1), Quetiapine (n=1)
Roth 2007	SSRI (n=6)
Bohne 2008	Psychotropic medication (n=13) – not specified
Cha 2008	Sertraline, Paroxetine, and fluoxetine
Rao 2008	SSRI (n=29); Fluoxetine (n=10), Sertraline (n=7), Citalopram (4), Escitalopram (n=3), Paroxetine (n=3), and Clomipramine (n=1). 8 were also taking Clonazepam, and 1 was taking Ziprasidone
Trivedi 2008	Medication not specified
Nedeljkovic 2009	Serotonergic medication
Starcke 2009	SSRI (9), of which 3 received anxiolytic medication
Starcke 2010	SSRI (14), of which 6 received anxiolytic medication
Blom 2011	SSRI (n=8), SSRI and antipsychotic (n=4), SSRI with Bupropion (n=3), lithium with antipsychotic (n=1)
do Rocha 2011	SSRI (n=52), Clomipramine (n=15), Clomipramine with risperidone (n=6), SSRI with risperidone (n=6)

Boisseau 2012	Psychiatric medication (n=5); antidepressants, anxiolytics, and atypical neuroleptics.
Bersani 2013	SSRI (n=23) and or clomipramine with benzodiazepine
Boisseau 2013	Antidepressant (n=6), anxiolytic (n=2), neuroleptics (n=1)
Dittrich 2013	Citalopram, Fluvoxamine, Fluoxetine, and Sertraline
Endrass 2013	Escitalopram (n=1), Citalopram (n=2), Fluoxetine (n=1), Paroxetine (n=3), and Clomipramine (n=1)
Kashyap 2013	SSRIs (n=120), of which 36 were receiving fluoxetine
Sohn 2014	SSRI (n=78), SSRI with a non-SSRI (n=31); benzodiazepine (n=24), benzodiazepine and antipsychotic (n=7)
Tolin 2014	Psychiatric medication – not specified
Grassi 2015	Serotonergic and/or dopaminergic agents (n=32), SSRIs (n=5), SSRIs with antipsychotics (n=27)
Kim 2015	SSRI (n=63), with benzodiazepine (n=43), with antipsychotic (n=8)
Kohli 2015	Medication not specified
Martoni 2015	SSRI (n=78), Clomipramine (n=5), SSRI and low dose antipsychotic (n=9)
Singh 2015	Medication not specified
Yang 2015	Paroxetine, fluoxetine, sertraline, fluvoxamine, and escitalopram
Zhang 2015b	Fluoxetine (n=22), Sertraline (n=18), Paroxetine (n=16), Citalopram (n=6), Fluvoxamine (n=6), Clomipramine (n=5), Escitalopram (n=4); 14 were receiving benzodiazepines (clonazepam (n=9), estazolam (n=5)) and 21 were receiving antipsychotic medication (risperidone (n=11), olanzapine (n=4), Quetiapine (n=4), and aripiprazole (n=2)
Norman 2017	SSRI (n=4). One patient was also receiving Risperidone
Vaghi 2017	SSRI (n=27); of this, 4 were also receiving tricyclic antidepressant, and 2 were receiving and an atypical antipsychotic
Grassi 2018	SSRI (n=6), SSRI with CBT (n=26)
Martoni 2018	24 receiving SSRI, 9 of which were also treated with another drug (not specified)
Norman 2018	SSRI (n=4), Risperidone (n=1)
Cillo 2019	Fluoxetine (n=3), Fluvoxamine (n=4), Paroxetine (n=1), Citalopram (n=2), Fluvoxamine & Fluoxetine (n=2), Citalopram & Clomipramine (n=3), Paroxetine & topiramate (n=1), Fluoxetine & gabapentin (n=2), Paroxetine (n=2), Paroxetine & risperidone (n=1)
Grassi 2019	SSRI (31), SSRI with antipsychotic medication (n=10)
Yu 2019	SSRI (n=24), SSRI with Olanzapine (n=1)
Luo 2020	SSRI (n=10), SSRI with antipsychotics (n=2), just antipsychotics (n=1)
Frydman 2020	Fluoxetine (n=5), Sertraline (n=5), Paroxetine (n=2), Escitalopram (n=2), Clomipramine (n=2), Venlafaxine (n=1)
Thorsen 2020	SSRI (n=6), Methlyphenidate (n=1)
Bhattacharya 2021	SSRI, antipsychotics, and benzodiazepine (names and quantities not given)
Chen 2021	SSRI (n=23)
Martinez-Esparza 2021	Sertraline, Fluoxetine, Fluvoxamine, Citalopram, Haloperidol. Of the 18 medicated, 16 were only receiving SSRIs, 1 was receiving an antipsychotic, and 1 was receiving both antidepressant and antipsychotic
Moritz 2021	Antidepressant (n=10), antipsychotic (n=4), tranquiliser (n=4)
Sternheim 2022	Antidepressants (n=35), antipsychotics (n=7), benzodiazepine (n=11)

Appendix J. Post-Pandemic Adjustment Scale (PPAS: Fineberg et al, 2021).

1. I am having great difficulty adjusting to the easing of the COVID-19 pandemic restrictions.

2. I am finding it harder to manage my fears about COVID now that the COVID-19 pandemic restrictions are easing than I did when the restrictions were fully in force.

3. I am finding it very stressful going out of the house now that the COVID-19 pandemic restrictions are easing.

4. I am thinking too much about contracting or spreading coronavirus now that the COVID-19 pandemic restrictions are easing.

5. I am thinking too much about other risks to my or others' physical health now that the COVID-19 pandemic restrictions are easing.

6. I am finding it hard to stop physical distancing or avoiding contact with people now that the COVID-19 pandemic restrictions are easing.

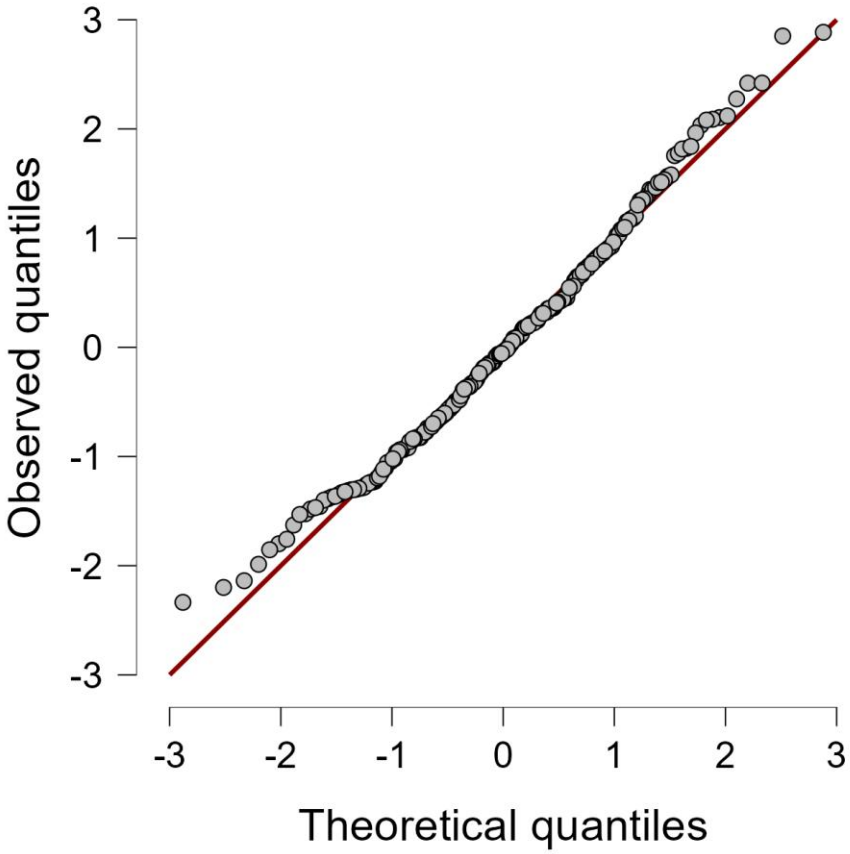
7. I am finding it hard to stop disinfecting behaviours (e.g. handwashing, use of sterile wipes, use of gloves, masks, etc.) that are no longer officially recommended now that the COVID-19 pandemic restrictions are easing.

These items were assessed on a 5-point Likert scale; 1= completely disagree, 2= disagree, 3=neither agree nor disagree, 4=agree, and 5= completely agree.

Appendix K. Covid-19 Safety Behaviour Scale**.**

	Never 0	Hardly ever 1	Often 2	Most of the time 3	All of the time 4
I cancel my usual social activities					
I do not go to work					
I do not go shopping for non-essential things					
I do not go to grocery stores or pharmacies					
I do not leave the house					
I comply with wearing face mask recommendations when outside my home					
I try to avoid physical contact with people					
I follow handwashing recommendations					
I use hand sanitizer more than usual					
I follow coughing and sneezing recommendations					
I use tissues more than usual					
I wear gloves while going out of my house					
I avoid public transport					
I avoid going to restaurants/ bars/ pubs					
I avoid going for walks or exercise outside					

Appendix L: Homoscedasticity assessment for post-pandemic adjustment difficulty mediation model: Q-Q Plot Standardized Residuals.



Appendix M. Covid-19 and mental health related information.

Have you or a close family member contracted COVID-19?	
Yes	156
No	94
Unsure	2
Have you experienced a COVID-19 related death of someone close to you?	
Yes	43
No	208
Unsure	1
Do you have a personal history of mental disorders?	
Yes	108
No	144
Is there a history of mental health disorders in your family?	
Yes	94
No	158

Appendix N. Correlational matric of OCI-R symptom dimensions against WCST outcome metrics (n=252).

	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*
(1) Hoarding	-								
(2) Checking	.64**	-							
(3) Ordering	.55**	.64**	-						
(4) Neutralising	.51**	.64**	.58**	-					
(5) Washing	.45**	.59**	.54**	.57**	-				
(6) Obsessing	.52**	.63**	.57**	.58**	.48**	-			
(7) WCST – P errors	.06	.07	.02	.06	.01	-.02	-		
(8) WCST -F errors	.12	.06	.08	.05	.08	.04	.08	-	
(9) WCAT -NP errors	.15*	.13*	.10	.09	.08	.09	.05	.43**	-
(10) WCST – T errors	.16*	.14*	.10	.12	.08	.05	.74**	.54**	.64**

Note: WCST = Wisconsin Card Sorting Test, P errors = Perseverative errors, F errors = Failure to maintain set errors, NP errors = Non-perseverative errors, T errors = Total errors

***=p<.001*

**=p<.05*

Appendix O. Correlations matrix of OCI-R symptom dimensions against IDED outcome metrics(n=31).

	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*	*(10)*	*(11)*	*(12)*	*(13)*	*(14)*	*(15)*	*(16)*
(1) OCI-R total	-															
(2) Hoarding	**.66****	-														
(3) Checking	**.79****	**.43****	-													
(4) Ordering	**.73****	**.36****	**.57****	-												
(5) Neutralising	**.75****	**.39****	**.62****	**.51****	-											
(6) Washing	**.75****	**.25***	**.54****	**.52****	**.45****	-										
(7) Obsessing	**.78****	**.48****	**.51****	**.32***	**.54****	**.50****	-									
(8) IDED – SD	-.23	-.22	-.20	-.24	-.14	**-.39***	.05	-								
(9) IDED – SR	-.02	.06	-.19	.10	-.05	-.11	.04	-.20	-							
(10) IDED – CDA	-.16	.00	-.17	-.19	-.18	-.30	-.01	.24	.07	-						
(11) IDED – CDS	-.14	-.15	-.03	-.14	-.10	-0.7	-.14	**.36***	-.11	**.47****	-					
(12) IDED – CR	-.07	-.08	-.06	-.03	-.05	-0.6	-.03	.06	.10	-.13	-.14	-				
(13) IDED – IDS	-.12	-.13	-.02	-.01	-.12	-.13	-.14	.02	-.06	-.2-	-.08	.06	-			
(14) IDED – IDSR	-.05	-.16	-.01	.15	-.01	-.21	.03	.08	-.10	.13	-.04	.22	**.81****	-		
(15) IDED – EDS	-.18	.06	-.23	-.25	-.18	-.26	-.05	.32	.02	.40*	.10	**-.44***	-.13	.01	-	
(16) IDED - EDSR	-.07	-.12	.05	-.13	-.10	-.12	.04	.10	.32	-.04	-.02	-.26	**.51****	-.02	-.18	-
(17) IDED – total	-.15	-.10	-.25	-0.1	-.14	-.37	.10	**.39***	.20	**.80****	**.46***	-.05	.04	**.69****	**.70****	.04

Note: SD=Simple discrimination; SR = Simple reversal; CDA = Compound Discrimination Adjacent; CDS = Compound Discrimination Superimposed; CR = Compound Reversal; IDS = Intradimensional Shift; IDSR = Intradimensional Shift Reversal; EDS = Extradimensional Shift; EDSR = Extradimensional Shift Reversal

*** = p<.001*

** = p<.05*

Appendix P. ANOVA of completers against non-completers.

	Dropped out after baseline assessment N=194 Mean(SD)	Dropped out after 3-month assessment N=24 Mean(SD)	Completed all assessments N=34 Mean(SD)		
PPAS	18.54(6.22)	19.71(7.78)	20.38(8.35)	$F(2,249)=1.37$, $\eta p^2=.01$	
OCI-R	25.69(15.87)	23.63(13.46)	27.12(16.28)	$F(2,249)=0.35$, $\eta p^2=.00$	
CPAS	11.02(6.25)	10.75(5.83)	11.88(7.80)	$F(2,249)=0.30$, $\eta p^2=.00$	
DASS-21	21.73(13.72)	20.17(15.27)	24.82(16.28)	$F(2,249)=0.90$, $\eta p^2=.01$	
C-19 SBS	29.87(11.87)	32.00(12.99)	32.97(12.10)	$F(2,249)=1.18$, $\eta p^2=.01$	
WCST					
P-Errors	14.02(6.90)	11.08(3.22)	11.53(5.97)	**$F(2,249)=3.79$, $\eta p^2=.03$***	*No group differences revealed*
FTMS-Errors	3.25(2.72)	2.13(1.90)	2.06(1.79)	**$F(2,249)=4.70$, $\eta p^2=.04$***	1x3 **$t=2.52$, $d=0.47$***
R-Errors	9.51(5.00)	8.46(4.01)	7.88(4.58)	$F(2,249)=1.91$, $\eta p^2=.02$	
T-Errors	26.47(9.65)	21.25(7.01)	21.15(8.13)	**$F(2,249)=7.29$, $\eta p^2=.06$****	1x2 **$t=2.61$, $d=0.57$*** 1x3 **$t=3.10$, $d=0.58$***

Note: PPAS=Post-Pandemic Adjustment Scale, OCI-R=Obsessive=Compulsive Inventory-Revised, CPAS=Compulsive Personality Assessment Scale, DASS-21=Depression, Anxiety, and Stress Scale-21, C-19 SBS=COVID-19 Safety Behaviour Scale, WCST=Wisconsin Card Sorting Test, P-Errors=Perseveration errors, FTMS-Errors=Failure To Maintain Set errors, R-Errors = Random errors, and T-Errors=Total errors.

**p*<.05

***p*<.001

Appendix Q. Original 59 items with rating scale.

The below statements refer to impulsive and compulsive tendencies and behaviours. Please rate these statements on how relevant they are to you. For example, if a statement is not applicable to yourself, please select **"Not at all"**, whereas, if something has great applicability to you, then select **"very much"**	Not at all	A little	Quite a lot	A lot	Very much
Item 1: I get stuck thinking about one thing					
Item 2: I am troubled by having to repeat behaviours that seem excessive or pointless					
Item 3: I frequently act on impulse					
Item 4: I do things impulsively that seem rewarding at the time even if I know they are bad for me in the long run.					
Item 5: I have difficulty getting started on new tasks					
Item 6: I am a creature of habit					
Item 7: Others describe me as someone who needs to feel in control					
Item 8: I am troubled by unwanted thoughts that repeat in my mind					
Item 9: I am effective in stopping or diverting unwanted behaviours or urges ®					
Item 10: I resist acting on urges ®					
Item 11: I have difficulty making decisions about everyday matters					
Item 12: I have difficulty finishing tasks that I have started					
Item 13: I feel the need for things to be perfect					
Item 14: People have told me I can be stubborn or rigid					
Item 15: I find uncertainty troubling					
Item 16: I am good at working out how people are thinking and feeling even when they do not tell me ®					
Item 17: I am effective in stopping or diverting unwanted repetitive thoughts ®					
Item 18: I take strong positions on things when I believe they are right or wrong					
Item 19: I am troubled by having to repeat mental acts that seem excessive or pointless					
Item 20: Once I have started an action I find it hard to stop					
Item 21: Routine activities take me longer than they should					
Item 22: I doubt having done things properly					
Item 23: I like to be in control or else I feel upset					
Item 24: I can tolerate breaking a rule ®					
Item 25: I tend to feel guilty when things go wrong even when it is not my fault					
Item 26: I try to do things (i.e., activities, tasks, or movements) until they feel "just right"					
Item 27: I am effective in stopping or diverting unwanted thoughts ®					
Item 28: I have difficulty spending money especially when it is on myself or a loved one					
Item 29: I have strong urges to acquire things when I don't really need them.					
Item 30: I perform unnecessary physical or vocal actions e.g., muscle twitching, coughing, sniffing, throat clearing, saying certain words					
Item 31: I lack energy, motivation or drive					
Item 32: I have difficulty organising and carrying out a planned task e.g., a holiday, shopping trip or a birthday celebration					
Item 33: I can be rigid					
Item 34: When I read, I sense I may have missed something and must go back and reread the passage					
Item 35: I shoulder responsibility beyond what could be reasonably expected					
Item 36: Others see me as being hyper-critical					
Item 37: If I am interrupted, I can quickly switch back to what I was doing ®					
Item 38: I can be flexible or easy going ®					
Item 39: When something I have done does not feel quite right, I need to repeat it until it does					
Item 40: I see money as something I should save for future possible needs					
Item 41: I get stuck when performing routine tasks					
Item 42: I tend to have rigid views about how things look or have to be					

Item 43: I can easily get rid of things that I do not need ®
Item 44: I find it hard moving from task to task
Item 45: I prefer all or nothing decisions
Item 46: I am troubled when things are not complete e.g., missing items in a set
Item 47: I feel disgust in the presence of bodily secretions e.g., blood, sweat, urine, faeces
Item 48: I feel uncomfortable if I do not get my own way
Item 49: I get preoccupied with details
Item 50: I find it hard to manage unexpected changes to my routine
Item 51: I avoid getting rid of things because I am afraid that I might need them later.
Item 52: I feel exhausted
Item 53: I tend to do things according to a routine (e.g., eating meals at a specific time, using the same utensils, sitting in a specific seat etc).
Item 54: Others have commented that I make noises or movements e.g., sniffing, humming, grunting
Item 55: I am disturbed by small errors or imperfections
Item 56: I find it easy to trust others to carry out tasks the way they ought to be done ®
Item 57: Certain foods, textures, and smells easily disgust me
Item 58: I am happy to compromise ®
Item 59: I usually concentrate more on the whole picture, rather than the small details ®

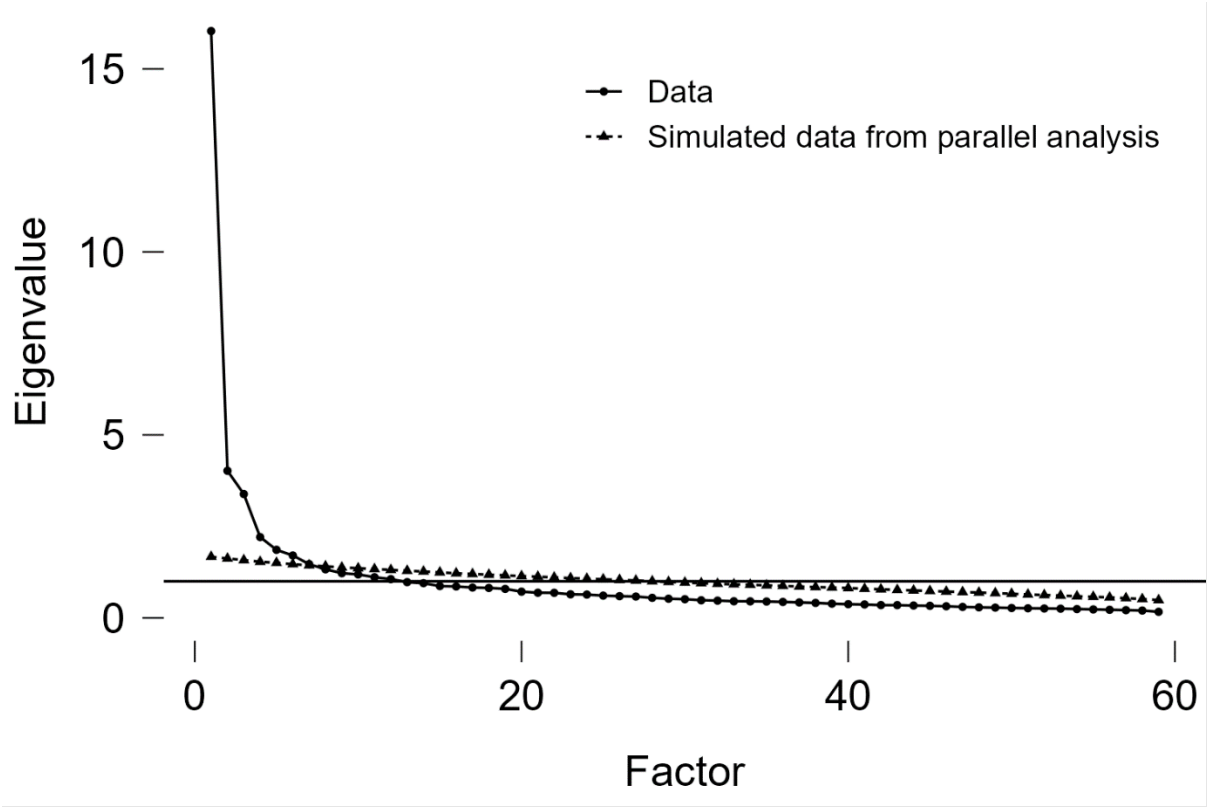
Footnote: ® = reverse scored item.

Appendix R. Sociodemographic characteristic of non-clinical sample (n=590).

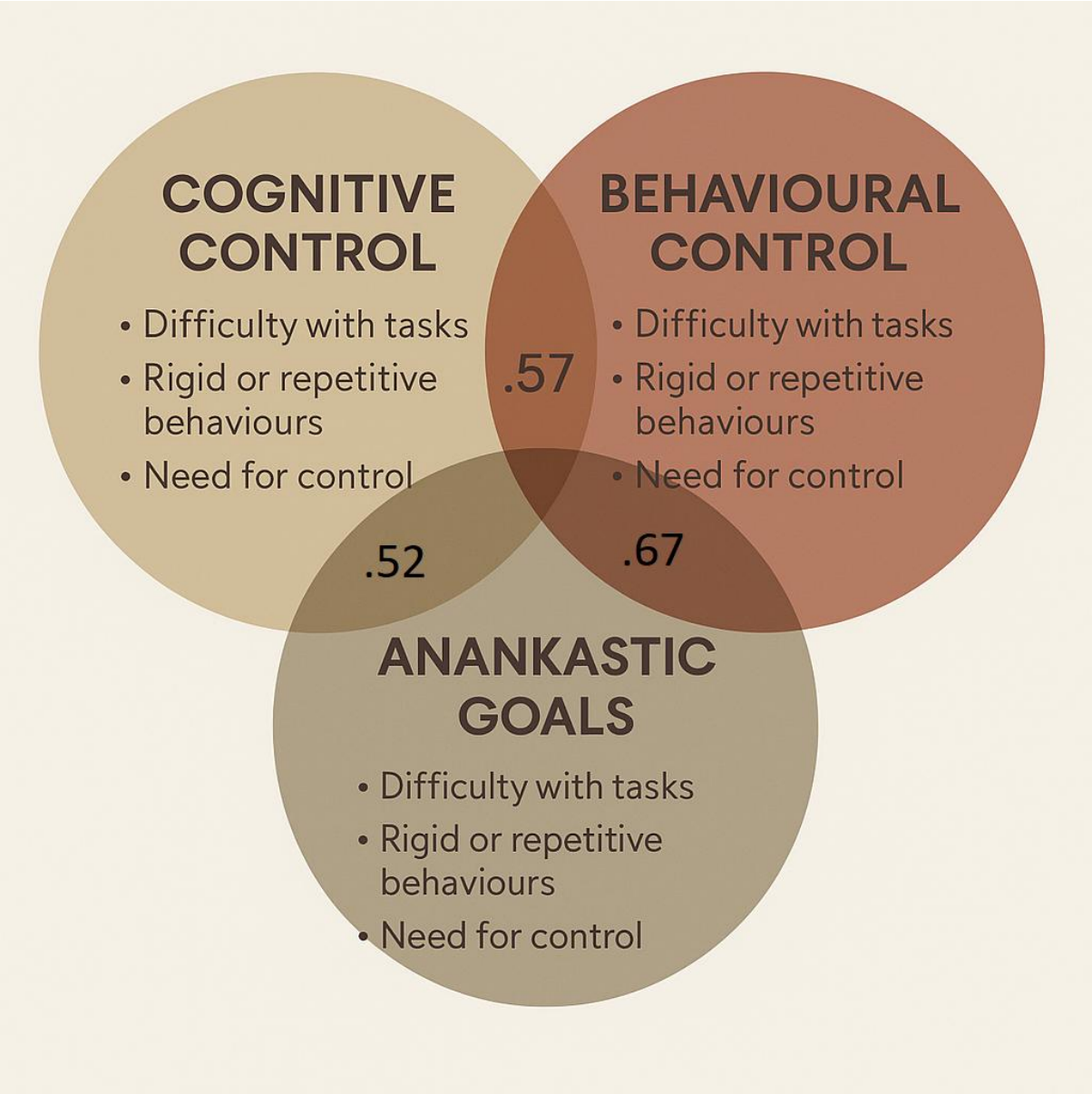
	N (%)		N (%)
Faith group		Education	
Catholic/Christian	173 (29.30)	Primary ed	3 (0.50)
Muslim	13 (2.20)	Secondary ed	190 (32.20)
Hindu	5 (0.80)	A-Levels	8 (1.40)
Jewish	1 (0.20)	Uni undergraduate	255 (43.20)
Buddhist	4 (0.70)	Uni Postgraduate	115 (19.50)
Sikh	2 (0.30)	Other	19 (3.20)
Agnostic	44 (7.50)		
Atheist	335 (56.80)		
Other	13 (2.20)		
Ethnicity		Personal Hx of MHD	
White	512 (86.80)	Self-reported Hx	261 (44.20)
Black	24 (4.10)	No personal Hx	329 (55.80)
Asian	29 (4.90)	Familial Hx of MHD	
Mixed	24 (4.10)	Self-reported Hx	226 (38.30)
Other	1 (0.20)	No familial Hx	364 (61.70)

Footnote: Hx = History, MHD = mental health disorder

Appendix S. Scree plot for the extraction of factors.



Appendix T. Venn diagram illustrating the overlapping phenomenology of the OCPAS subscales.



Appendix U. Mean error scores on each stage of the IDED.

	Mean (SD)	Ranges
Stage 1 – SD	1.03(0.74)	0.00-3.00
Stage 2 – SR	1.26(0.72)	1.00-5.00
Stage 3 – CDA	1.41(2.01)	0.00-10.00
Stage 4 – CDS	0.26(0.60)	0.00-2.00
Stage 5 – CR	1.31(0.73)	1.00-4.00
Stage 6 – IDS	1.39(2.49)	0.00-13.00
Stage 7- IDSR	2.82(4.21)	1.00-27.00
Stage 8 – EDS	4.66(6.14)	0.00-26.00
Stage 9 – EDSR	1.23(0.60)	1.00-4.00
IDED Total score	12.66(4.50)	5.00-24.00

Appendix V. Correlational matrix of IDED stages and CPAS items.

	Item 1 Preocc	Item 2 Perf	Item 3 Work	Item 4 OverC	Item 5 Hoard	Item 6 NfC	Item 7 Mis	Item 8 Rigid	CPAS Total
Stage 1 – SD	-0.06	0.15	-0.08	0.06	0.03	-0.18	-0.30	0.02	-0.08
Stage 2 – SR	0.12	-0.06	-0.25	-0.12	-0.26	-0.07	0.11	0.12	-0.07
Stage 3 – CDA	-0.04	**-0.34***	-0.16	**-0.32***	-0.18	-0.22	0.16	-0.21	-0.23
Stage 4 – CDS	**0.31***	0.19	**0.32***	0.26	-0.08	0.14	-0.17	-0.10	0.21
Stage 5 – CR	0.02	0.09	0.12	0.07	**0.35***	0.02	-0.28	-0.07	0.07
Stage 6 – IDS	0.08	0.03	0.03	-0.18	-0.17	0.10	-0.19	-0.04	-0.07
Stage 7 – IDSR	0.06	0.05	0.22	0.14	0.22	-0.09	0.14	-0.16	0.11
Stage 8 – EDS	**-0.34***	**-0.41***	-0.11	-0.03	-0.10	-0.02	0.05	-0.14	-0.19
Stage 9 – EDSR	-0.17	**-0.39***	-0.11	-0.18	0.00	-0.10	0.03	**-0.36***	-0.22
Total errors	-0.07	-0.26	-0.02	-0.22	-0.10	-0.06	-0.23	-0.24	-0.22

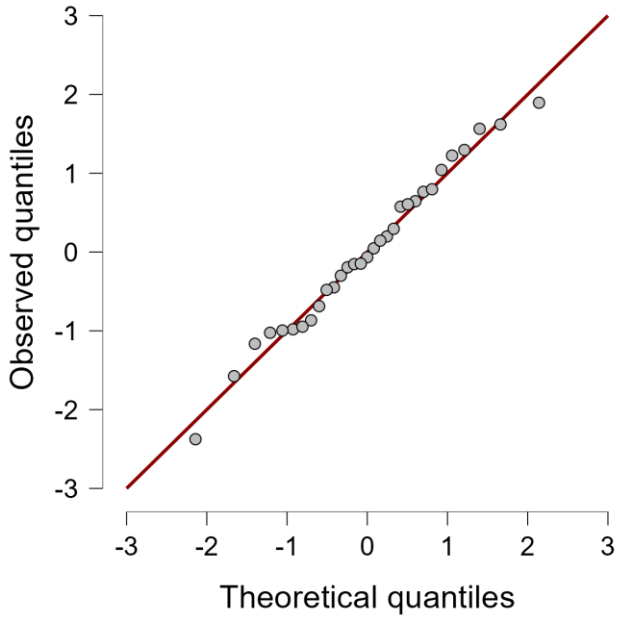
Note: Preocc = Preoccupation, Perf = Perfectionism, Work = Workaholism, OverC = Over conscientiousness, Hoard = Hoarding, NfC = Need for control, Mis = Miserliness, Rigid = Rigidity
** = p<.05, ** = p<.001*
*Those in **Bold** are significant correlations*

Appendix W. Correlations between SST and BIS-11 total and subscale scores.

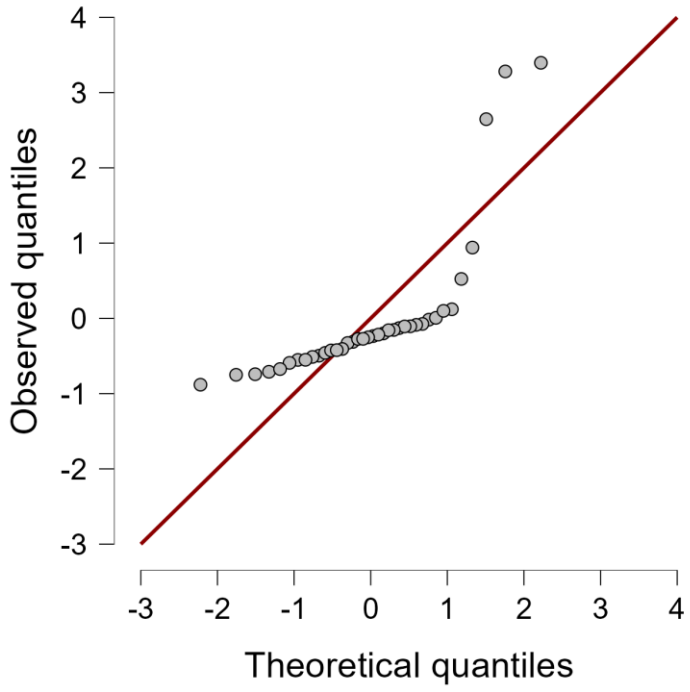
	Attentional Impulsivity	Motor Impulsivity	Non-planning Impulsivity	BIS-11 Total score
SSRT	0.20	0.33	0.07	**0.37***

*Note: * = p<.05*
*Those in **Bold** are significant correlations*

Appendix X: Homoscedasticity assessment for SSRT regression: Q-Q Plot Standardized Residuals.



Appendix Y: Homoscedasticity assessment for IDED stage 8 regression: Q-Q Plot Standardized Residuals.



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Appendix Z: Homoscedasticity assessment for IDED stage 9 regression: Q-Q Plot Standardized Residuals.