NEUROPSYCHOLOGY AND SYMPTOMS OF OBSESSIVE-COMPULSIVE DISORDER

NEUROSYPSYCHOLOGICAL FUNCTIONING, SYMPTOM DIMENSIONS AND COGNITIVE REMEDIATION IN OBSESSIVE-COMPULSIVE DISORDER

By DUNCAN H. CAMERON, B.A.

A Thesis Submitted to the School of Graduate Studies in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy

McMaster University © Copyright Duncan Cameron, August 2018

McMaster University DOCTOR OF PHILOSOPHY (2018) Hamilton, Ontario, (Neuroscience)

TITLE: Neuropsychological Functioning, Symptom Dimensions, and Cognitive Remediation in Obsessive-Compulsive Disorder

AUTHOR: Duncan Cameron, B.A. (McMaster University)

SUPERVISORS: Professor Randi E. McCabe & Associate Professor Margaret C. McKinnon

NUMBER OF PAGES: xiii, 156

ABSTRACT

Objectives: The first objective of this research was to examine whether symptom dimensions in obsessive-compulsive disorder (OCD) are associated with unique neuropsychological performance profiles. The second objective of this research was to further understand the strengths and weaknesses of two models of symptom dimensions in OCD from a quantitative perspective—conventional subtyping by overt symptom, and the core dimensions model. Finally, the third objective of this research was to investigate the efficacy and treatment acceptability of a cognitive remediation program targeting neurocognitive deficits associated with OCD.

Methods: Study 1 reviewed critically studies describing the assessment of differences in neuropsychological functioning between symptom dimensions of OCD, the results of which informed succeeding studies examining: i) the characterization of symptom dimensions in OCD and; ii) the remediation of neuropsychological domains commonly affected in OCD. Accordingly, study 2 compared the suitability of two common statistical approaches, factor analysis and cluster analysis, commonly used in the existing literature to define symptom dimensions based on responses collected from the industry-standard symptom questionnaire, the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), in characterizing symptom dimensions in OCD. Neuropsychological task data were then used to examine the validity of an alternative model of symptom dimensions in OCD (Study 3). Finally, we conducted a feasibility study (Study 4) examining the use of an established cognitive remediation protocol, Goal Management Training (GMT), to target the deficits in neurocognitive function identified in the preceding studies.

Results: Much of the existing literature on neuropsychological task performance differences between symptom dimensions of OCD is limited by methodological issues, primarily those concerning methods for defining symptom dimensions. Here, a comparison of the two most common methods for defining dimensions revealed that neither cluster analysis nor factor analysis produced conceptually meaningful subgroups. By exemplifying differences in neuropsychological task performance between those with harm avoidance and those with incompleteness symptoms, however, concrete evidence was provided to support the core dimensions model of OCD. Pilot data point towards the feasibility and efficacy of GMT as a cognitive remediation program for OCD.

Conclusions: Pursuing the definition of meaningful, distinct symptom dimensions of OCD is not recommended with the combination of current statistical practices and symptom measures. The early evidence presented here shows promise for the validity of the core dimensions model. Preliminary evidence suggests that the neuropsychological impairments observed in this population, although subtle, may be effectively addressed using Goal Management Training.

Keywords: obsessive-compulsive disorder, neuropsychology, symptom dimensions, cognitive remediation, core dimensions

ACKNOWLEDGEMENTS

I would like to thank my parents for instilling in me the intellectual curiosity that has served me so well over the years. I would also like to thank my partner Olivia for her steadfast understanding, love and support over the past four years of my doctoral studies.

I am especially grateful for the supervision, mentorship and many collaborative opportunities provided to me by my supervisors Dr. Randi McCabe and Dr. Margaret McKinnon. I have also appreciated the guidance and suggestions of my supervisory committee members Dr. Karen Rowa and Dr. Suzanna Becker.

I would also like to take this opportunity to thank Dr. David Streiner for his mentorship in biostatistics and for being such an excellent collaborator on several projects. A big thank you also to Dr. Laura Summerfeldt for providing the foundation upon which much of the research contained within this dissertation is based, and for her extensive revisions and comments provided for my writing.

Finally, I have greatly appreciated the financial support of the McMaster Graduate Program in Neuroscience, as well as the Ontario Student Assistance Program for the Ontario Graduate Scholarship provided to support my work.

TABLE OF CONTENTS

PRELIMINARY PAGES **Page**

Abstract iii
Key Words iv
Acknowledgements v
Table of Contents vii
List of Illustrations, Charts & Diagrams viii
List of Tables ix
List of Abbreviations & Symbols x
Declaration of Academic Achievement xii

CHAPTER ONE: BACKGROUND

Psychopathology of Obsessive-Compulsive Disorder 1
Obsessive-Compulsive Disorder: Subtypes/Dimensions 2
Methods for Defining Symptom Dimensions 3
An Alternative Model for Symptom Dimensions 4
The Neuropsychology of Obsessive-Compulsive Disorder 6
Elucidating Symptom Dimensions with Neuropsychology 7
Treatment of Cognitive Deficits in Obsessive-Compulsive Disorder 9
Sandwich Thesis Overview 11
References 13

CHAPTER TWO: STUDY 1 (Neuropsychological Performance in Symptom-Based Subtypes of Obsessive-Compulsive Disorder: Review)

Title, Authorship, Context and Implications 20
Acknowledgements 21
Abstract 22
Introduction 23
Methods 28
Results 30
Discussion 34
References 39
Appendix: Tables and Figures 45

CHAPTER THREE: STUDY 2 (Comparison of Statistical Methods for Defining Symptom Dimensions of OCD)

Title, Authorship, Context and Implications 50
Acknowledgements 51
Abstract 52
Introduction 53
Methods 60

Results 62
Discussion 63
References 68
Appendix: Tables and Figures 75

CHAPTER FOUR: STUDY 3 (Neuropsychological Performance in Core Dimensions of OCD)

Title, Authorship, Context and Implications 85
Acknowledgements 86
Abstract 87
Introduction 88
Methods 91
Results 95
Discussion 96
References 102
Appendix: Tables 107

CHAPTER FIVE: STUDY 4 (Feasibility and Efficacy of Cognitive Remediation in OCD)

Title, Authorship, Context and Implications 109
Acknowledgements 110
Abstract 111
Introduction 112
Methods 118
Results 123
Discussion 127
References 134
Appendix: Tables and Figures 144

CHAPTER SIX: CONCLUSIONS

Summary and Significance of Findings 148
Implications 150
Synthesis of Results for Direction of Future Study 152
Conclusions 155
References 156

LIST OF ILLUSTRATIONS, CHARTS & DIAGRAMS

CHAPTER ONE: BACKGROUND **Page**

None -

CHAPTER TWO: STUDY 1 (Neuropsychological Performance in Symptom-Based Subtypes of Obsessive-Compulsive Disorder: Review)

Figure 1 – Strategy for Selection of Relevant Articles 45

CHAPTER THREE: STUDY 2 (Comparison of Statistical Methods for Defining Symptom Dimensions of OCD)

Figure 1 – Cluster Membership 83
a) Aggressive/Checking 83
b) Contamination/Cleaning 84
c) Symmetry/Ordering 84
d) Mixed Symptoms 84

CHAPTER FOUR: STUDY 3 (Neuropsychological Performance in Core Dimensions of OCD)

None -

CHAPTER FIVE: STUDY 4 (Feasibility and Efficacy of Cognitive Remediation in OCD)

None -

CHAPTER SIX: CONCLUSIONS

None -

LIST OF TABLES

CHAPTER ONE: BACKGROUND **Page**

None -

CHAPTER TWO: STUDY 1 (Neuropsychological Performance in Symptom-Based Subtypes of Obsessive-Compulsive Disorder: Review)

Effect Sizes for Neuropsychological Deficits Organized by Study and Cognitive Domain 46

CHAPTER THREE: STUDY 2 (Comparison of Statistical Methods for Defining Symptom Dimensions of OCD)

Table 1 – Summary of Past Approaches to Defining Symptom Dimensions in OCD 75
Table 2 – Sample Demographics 82
Table 3 – Factor Loadings for Principal Axis Factoring with Promax Rotation 82
Table 4 – Representation of Y-BOCS Symptoms as Primary of Secondary 82
Table 5 – Factor Loadings for Principal Axis Factoring with Varimax Rotation without Removing Variables due to Low Communality or MSA; no MAP test or Parallel Analysis 83

CHAPTER FOUR: STUDY 3 (Neuropsychological Performance in Core Dimensions of OCD)

Table 1 – Sample Demographics 107
Table 2 – Independent Samples t-tests 108

CHAPTER FIVE: STUDY 4 (Feasibility and Efficacy of Cognitive Remediation in OCD)

Table 1 – Sample Demographics 144
Table 2 – Overview of Goal Management Training Protocol 144
Table 3 – Means and Standard Deviations for Statistically Significant 2x2 Repeated Measures ANOVAs 146
Table 4 – Complete List of Assessment Measures 147

CHAPTER SIX: CONCLUSIONS

None -

LIST OF ABBREVIATIONS AND SYMBOLS

ADHD: Attention-deficit/hyperactivity disorder
ANOVA: Analysis of Variance
APA: American Psychiatric Association
BABS: Brown Assessment of Beliefs Scale
BDI-II: Beck Depression Inventory, second edition
BQSS: Boston Qualitative Scoring System
CAC: Compulsive Activity Checklist
CANTAB: Cambridge Neuropsychological Test Automated Battery
CBT: Cognitive Behavioural Therapy
CFA: Confirmatory factor analysis
CFQ: Cognitive Failures Questionnaire
CIHR: Canadian Institutes of Health Research
COWAT: Controlled Oral Word Association Test
CPT: Conners' Continuous Performance Task, second edition
CSTC: Cortico-striato-thalamic-cortical
CVLT-II: California Verbal Learning Test, second edition
DASS-21: Depression Anxiety and Stress Scales, 21-item version
DEX: Dysexecutive Questionnaire
D-KEFS: Delis-Kaplan Executive Function System
DOCS: Dimensional Obsessive Compulsive Scale
DSM-IV: Diagnostic and Statistical Manual of Mental Disorders, 4th Edition
DSM-5: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition
DY-BOCS: Dimensional Yale-Brown Obsessive Compulsive Scale
EFA: Exploratory factor analysis
FA: Factor analysis
GMT: Goal Management Training
HA: Harm avoidance (core dimension)
INC: Incompleteness (core dimension)
IIRS: Illness Intrusiveness Rating Scale
IQ: Intelligence quotient
KMO/MSA: Kaiser-Meyer-Olkin measure of sampling adequacy
LCA: Latent class analysis
MACCS: Memory and Cognitive Confidence Scale
MAP: Velicer's minimum average partials test
ME: Main effect
MOCI: Maudsley Obsessional Compulsive Inventory
OC-CDI: Obsessive-Compulsive Core Dimensions Interview
OC-CDQ: Obsessive-Compulsive Core Dimensions Questionnaire
OCD: Obsessive-compulsive disorder
OCI-R: Obsessive-Compulsive Index-Revised
OFC: Orbitofrontal cortex
PAF: Principal axis factoring
PCA: Principal components analysis
PI: Padua Inventory

RCFT: Rey-Osterrieth Complex Figure Task
SCID-IV: Structured Clinical Interview for Axis I Disorders for DSM-IV
SD: Standard deviation
SDS: Sheehan Disability Scale
SPSS: Statistical Package for the Social Sciences
SS: Standard score
SSRI: Selective Serotonin Reuptake Inhibitor
TMT: Trail Making Task
TOL: Tower of London
WASI: Wechsler Abbreviated Scale of Intelligence
WCST: Wisconsin Card Sorting Task
WHODAS: World Health Organization Disability Assessment Schedule
WMS: Wechsler Memory Scale
WTAR: Wechsler Test of Adult Reading
Y-BOCS: Yale-Brown Obsessive Compulsive Scale

DECLARATION OF ACADEMIC ACHIEVEMENT

This ‘sandwich’ thesis consists of four studies conceived of and written by the student. He developed their premises, objectives and goals, conducted their data analyses, prepared the manuscripts and made revisions in keeping with the suggestions of his co-authors. All of this work was completed between September 1, 2014 and July 1, 2018. As such, the work contained herein meets the requirements for inclusion in the main text of this thesis. In keeping with the requirements of a ‘sandwich’ thesis, below I highlight the contributions made to each study by my co-authors.

Study 1 critically reviewed the existing literature regarding assessment of neuropsychological performance in symptom dimensions of obsessive-compulsive disorder. It was co-authored by Dr. Margaret McKinnon, who provided advice on the neuropsychological variables discussed in the paper, and who critically reviewed the manuscript and made suggestions to improve it. It was also co-authored by Dr. Randi McCabe, Dr. Karen Rowa, and Dr. Neil Rector, all of whom provided advice related to the definition and description of symptom dimensions of obsessive-compulsive disorder, and provided constructive feedback on all drafts of the manuscript.

Study 2 assessed the differences between two statistical methods—cluster analysis and factor analysis—commonly used for defining symptom dimensions of obsessive-compulsive disorder. Dr. David Streiner advised on the correct implementation of these statistical methods and provided feedback on the manuscript. Dr. Karen Rowa, Dr. Margaret McKinnon and Dr. Randi McCabe also co-authored the manuscript and provided revisions at all stages. Dr. Laura Summerfeldt provided revisions regarding manuscript layout and discussion of obsessive-compulsive disorder subtypes.

Study 3 examined differences in neuropsychological task performance between core dimensions of obsessive-compulsive disorder, providing early evidence for the validity of these dimensions as constructs of obsessive-compulsive disorder. Dr. Laura Summerfeldt provided advice regarding the core dimensions and revised all drafts of the manuscript. Dr. Karen Rowa, Dr. Margaret McKinnon and Dr. Randi McCabe all provided revisions at various stages of manuscript preparation. The data from this study was collected as part on an ongoing randomized multi-site clinical trial investigating the effects of exercise as a stand-alone and adjunctive treatment option for obsessive-compulsive disorder. This study was co-led by Dr. Neil Rector and Dr. Peggy Richter from Sunnybrook Health Sciences Centre, as well as Dr. Tisha Ornstein from Ryerson University, all of whom provided constructive feedback on the manuscript.

Study 4 investigated the feasibility and efficacy of a cognitive remediation program, Goal Management Training, in obsessive-compulsive disorder. Dr. Margaret McKinnon co-authored the manuscript and supervised administration of the Goal Management Training protocol. Dr. Randi McCabe, Dr. Karen Rowa, and Ms. Charlene O'Connor provided revisions to the manuscript at all stages of preparation.

CHAPTER ONE

BACKGROUND

Psychopathology of Obsessive-Compulsive Disorder

Obsessive-compulsive disorder (OCD) is recognised as a heterogeneous condition composed of multiple possible symptoms, resulting in a large number of possible permutations. The disorder is defined generally by the existence of recurrent intrusive, unwanted thoughts (obsessions) and repetitive behaviours (compulsions) aimed at reducing anxiety associated with the obsessions. OCD is thought to affect approximately 1%–3% of the population (Kessler et al., 2005), making it about twice as prevalent as bipolar disorder or schizophrenia (Karno, Golding, Sorenson, & Burnam, 1988), and its course is typically expected to be chronic and persist throughout the lifetime. The Diagnostic and Statistical Manual of Mental Disorders (DSM-5, American Psychiatric Association, 2013) offers a broad definition of OCD that includes obsessions and/or compulsions (where either may be present in conjunction with or in the absence of the other). The specific manifestation of these symptoms, however, can vary significantly between individuals with the same diagnosis. For example, one individual might experience troubling thoughts associated with contamination wherein the fear is that germs or dirt will lead to ill health of oneself or loved ones, while another patient may experience intrusive thoughts of damage or harm due to inadvertently leaving doors unlocked or appliances turned on at home. Individuals can also exhibit a variety of compulsive behaviours aimed at reducing or neutralizing the stress associated with an intrusive thought. As in the cases described above, the first patient may perform repetitive, ritualistic hand-washing while the second patient may compulsively check door locks and appliances until a desired level of satisfaction is

achieved. According to Rachman and Hodgson (1980), symptoms of contamination/cleaning, illness, harming, morality, checking, counting, exactness and unwanted disturbing images are all common.

The DSM-5 now describes OCD as an entity distinct from other anxiety disorders (American Psychiatric Association, 2013), although the definition provided therein encompasses a broad array of symptoms. The last two decades have seen a tremendous increase in the amount of research dedicated to investigating whether patients with specific types of symptoms are less responsive to standard treatment options. The heterogeneous presentation of OCD symptoms observed in clinical populations, along with the differential treatment response observed in certain cases, has motivated clinicians and researchers to hypothesize the existence of distinct, symptom-based subtypes of OCD (Abramowitz, McKay, & Taylor, 2005; Mataix-Cols, Rosario-Campos, & Leckman, 2005; McKay et al., 2004; Radomsky & Taylor, 2005). As a result, considerable research has been devoted to investigating various methods for defining these subtypes, with research efforts also focused one valuating any potential differences between them in terms of etiology and treatment response.

Obsessive-Compulsive Disorder: Subtypes and Dimensions

While several methods for subtyping have been proposed (i.e., early vs. late onset, presence vs. absence of tics, presence vs. absence of childhood diseases, or presence vs. absence of psychotic or neurological features; McKay et al., 2004), the large majority of existing research on subtyping in OCD focuses on subtyping based on overt symptoms, such as *washing* versus *checking* as described above. One challenge of this approach is determining whether a subtype model should be considered categorical or dimensional, a debate that surrounds other forms of

neuropsychiatric illness (e.g., Fenton & McGlashan, 1991; Gabbard, 1989; Lanius et al., 2010; Smits et al., 2017). A categorical model, which describes homogeneous, distinct subtypes is an attractive one as it allows for more accurate measurement in both clinical and research applications. A dimensional model would, however, classify clinical features in terms of quantification of attributes (symptoms) rather than the assignment to separate categories. In considering the typical presentation of OCD, it is rare that individuals will present with one and only one category of symptom. For example, patients might list *doubting/checking* symptoms as their most troubling, but in many cases this patient will also exhibit symptoms related to other themes. As such, the discussion of symptom classification in this dissertation will take a dimensional approach rather than categorical, as this more closely reflects the naturalistic presentation observed in the majority of patients with OCD.

Methods for Defining Symptom Dimensions

The categorisation of individuals with OCD based on principal symptom has been a long-standing effort, with early investigations describing comparisons of *washers* versus *checkers* (Lewis, 1936). The first use of a psychometrically validated measure designed to classify patients by subtype was the Maudsley Obsessional Compulsive Inventory (MOCI; Hodgson & Rachman, 1977), with three symptom factors including washing, checking and doubting-conscientiousness. The Compulsive Activity Checklist (CAC; Philpott, 1975) has been used for a similar purpose, with factor analysis identifying a two-factor solution of washing/cleanliness and checking (Freund, Steketee, & Foa, 1987). More recently, the Padua Inventory (PI; Sanavio, 1988) was developed to assess symptoms associated with senseless, repugnant thoughts and unacceptable urges. Again, factor analysis was used here to identify common symptom themes, and an updated version was subsequently published with five factors—washing, checking,

rumination, impulses and precision (van Oppen, Hoekstra, & Emmelkamp, 1995). Evidently, there is considerable overlap present between the dimensions identified in each of these tools. However, the measures described here rely heavily upon the symptoms of OCD that are most commonly seen in clinical samples, and as such several important symptoms, although less frequently observed, are left unrepresented.

The Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989) offers a significantly more substantial representation of the possible symptoms a patient diagnosed with OCD might experience. The Y-BOCS is composed of two parts—a symptom checklist with options to rate a symptom as being present/past/absent, and a symptom severity scale on which the individual's most troubling current symptoms identified from the checklist are rated. Given the greater diversity of symptoms represented on the Y-BOCS, this tool has been the focus of the vast majority of studies attempting to define symptom-based subtypes of OCD using statistical methods. The use of statistical methods to define symptom dimensions based on responses on Y-BOCS is described in great detail in Study 2 of this dissertation. A meta-analysis (Bloch, Landeros-Weisenberger, Rosario, Pittenger, 2008) of studies using factor analysis to achieve this goal identified four common factors—symmetry, forbidden thoughts, cleaning and hoarding. Cluster analysis has also been used to achieve the same end, with generally similar results to those reported in factor analytic studies (Calamari, Wiegartz, & Janeck, 1999; Calamari et al., 2004). Despite being arguably the most comprehensive checklist of OCD symptoms available, the Y-BOCS has still been criticized for underrepresentation of certain symptoms, most notably mental compulsions. The checklist is also subject to the same limitations mentioned above in that the less common OCD symptoms will not be measured with great enough frequency to provide

an accurate sample prior to undergoing cluster or factor analysis, and the categories that end up undergoing analysis are rationally—rather than empirically—derived.

An Alternative Model for Symptom Dimensions in OCD

As mentioned above, the classification of common themes among obsessive-compulsive symptoms is not a neoteric endeavour. French psychologist Pierre Janet also described *les sentiments d'incomplétude* (English translation Pitman, 1987), which provided the early framework upon which Rasmussen and Eisen (1992) described symptoms of *incompleteness*, also described by Rapoport (1991) as *the feeling of knowing* when proposing the basal ganglia as a potential origin of the inability to terminate a repeated behaviour sequence—symptoms considered unique to the diagnosis of OCD. These are defined as those in which an individual must complete a certain behaviour to a desired level of satisfaction and are often, but not exclusively, associated with symptoms of symmetry, counting, repeating and slowness. Shortly after, Summerfeldt (2004) described symptoms of incompleteness as being distinct from other OCD symptoms related to over-exaggeration of threat or *harm avoidance*.

These two core dimensions of OCD have received some research attention, most of which is theoretical in nature (Ecker & Gönner, 2008; 2014; 2017; Summerfeldt, 2004; Summerfeldt, Kloosterman, Antony, & Swinson, 2014). The core dimensions model addresses a potentially significant conceptual issue present in the conventional subtyping method. For example, one patient exhibiting washing symptoms might express the need to wash hands repeatedly for fear that contamination will lead to harm of oneself or one's family, while another patient might need to wash hands to a desired level of satisfaction with no specific harm-related consequence resulting if the ritual is not performed. It has been noted that the latter case may, in

fact, describe up to 40% of individuals with OCD (Tolin et al., 2001). Furthermore, very little evidence has supported any unique neurobiological underpinnings between conventional subtypes on the basis of overt symptomatology (e.g., washing, checking, exactness; Murayama et al., 2013; Nakao et al., 2009). As illustrated in Study 1, research using differential neuropsychological task performance as another potential correlate or endophenotypical marker of overt dimensions has also demonstrated little success in demonstrating distinct differences between subgroups, likely due to significant methodological challenges. Although the core dimensions model has yet to be tested with functional or structural imaging, there is merit to the hypothesis that the inability to terminate a repetitive behavioural sequence may be uniquely associated with a malfunction in the basal ganglia which has been implicated in the pathology of the disorder (Modell, Mountz, Curtis, & Greden, 1989; Rapoport, 1990; Saxena, Brody, Schwartz, & Baxter, 1998; Summerfeldt, 2004). Given the theoretical and methodological limitations inherent in conventional subtyping based on overt symptom presentation, research investigating other viable dimensions of OCD symptomatology remains an important effort.

The Neuropsychology of Obsessive-Compulsive Disorder

The neuropsychology of OCD has been studied extensively with several systematic reviews and meta-analyses published over the last two decades (Abramovitch, Abramowitz, & Mittelman, 2013; Abramovitch et al., 2015; Griesberg & McKay, 2003; Kuelz, Hohagen, & Voderholzer, 2004; Nakao, Okada, & Kanba, 2014; Shin, Lee, Kim, & Kwon, 2014; Tallis, 1997). Indeed, increased interest in the neurobiology of OCD, including the introduction of structural and functional imaging studies (Koch, Reeß, Rus, Zimmer, & Zaudig, 2014; Saxena, Brody, Schwartz, & Baxter, 1998; Whiteside & Ambramowitz, 2004) has resulted in a corresponding increase in neuropsychological studies, which can act to corroborate findings from

imaging studies and can also serve as a low-cost proxy for identifying regions of interest associated with the disorder. Together, these studies have resulted in the neurobiological model of OCD or the cortico-striato-thalamo-cortical (CSTC) model (also often referred to as the frontostriatal model or coticostriatal model; Milad & Rauch, 2012; Pauls, Abramovitch, Rauch & Geller, 2014; Saxena & Rauch, 2000). This model involves a direct pathway between the striatum and the globus pallidus interna and substantia nigra, as well as indirect pathways from the striatum to the globus pallidus externa to the subthalamic nucleus, which is then free to exite the globus pallidus interna and substantia nigra. It is hypothesized that in OCD an imbalance between the direct and indirect pathways results in over-activation of the direct over the indirect. This, in turn, leads to hyperactivation of the orbitofrontal-subcortical pathway, resulting in persistent focus on perceived threat (obsessions) and as a result, compulsions aimed at reducing anxiety associated with that threat. Critically, this model implies that dysfunction of frontostriatal systems should result in a pattern of performance deficits among individuals with OCD centered primarily in the neurocognitive domains typically associated with these systems. Nonetheless, a review of the numerous meta-analyses published on the neuropsychology of OCD to date points towards equivocal findings, rendering it difficult to form a harmonized model of cognitive function in OCD.

Here, the majority of meta-analyses point to small-to-medium effect sizes for dysfunction in subdomains of executive function such as planning, organization and problem-solving, small-to-medium effect sizes in the domains of attention and processing speed and large effects sizes for non-verbal memory—although it should be noted that observed deficits in non-verbal memory may be attributable to poor organisational strategy (Savage et al. 1999). Executive function performance has been the most extensively studied cognitive domain in OCD literature,

and indeed the findings of these studies are in line with the prevailing neurobiological model. The orbitofrontal cortex (OFC)—implicated heavily in the CSTC model—is thought to monitor reinforcement contingencies, and the ability to constrain previously learned behaviour suggests a prominent role of the OFC in inhibition of previously learned behavioural responses (Menzies et al., 2008). It would therefore be expected that individuals with a diagnosis of OCD would display poor performance on tasks measuring planning, decision-making, response inhibition, or set-shifting. Although promising, the vast heterogeneity of findings surrounding neuropsychological performance in OCD is concerning and points to the need to identify additional factors (e.g., heterogeneous symptom dimensions; methodological variability) that may account for observed differences in performance across studies. Notably, many researchers argue that not all individuals with a diagnosis of OCD will exhibit cognitive impairments, and in those who do, small-to-medium effect sizes suggest that impairments are unlikely to be severe in most cases. The neuropsychology of OCD is reviewed in greater detail in Chapter 2 of this thesis.

Elucidating Symptom Dimensions with Neuropsychology

Given the heterogeneous nature of neuropsychological findings in OCD, researchers have suggested that there may be underlying mechanisms that explain these discrepancies—among the most promising of which is the use of symptom dimensions (Abramovitch, Mittelman, Tankersley, Abramowitz, & Schweiger, 2015). One possible explanation for the equivocal nature of these results is that the large majority of studies assessing cognitive performance in OCD tend to view participants as one homogeneous group, when there may be important differences in performance between symptom dimensions of OCD. The examination of these potential differences has benefits both for the elucidation of neuropsychological deficits in OCD and for

the identification of symptom dimensions. The limitations (mentioned above and described in Chapters 1, 2, and 3) of using statistical methods with symptom-based measures for defining dimensions of OCD indicate that additional factors will need to be used in order to identify more homogeneous subgroups.

The differences in neuropsychological performance observed between conventional symptom dimensions of OCD are the topic of Study 1 of this thesis and will be discussed thoroughly therein. However, as mentioned earlier, the conventional subtyping method falls victim to some important limitations and as a result the findings of previously published investigations concerning this topic are limited in their interpretability. The alternative core dimensions model, describing incompleteness and harm avoidance as two separate entities, suggests that characteristics attributed to OCD in general, which are thought to distinguish it from anxiety disorders, may be true only of its incompleteness component. In particular, the generally slowed responding and the exacerbation of decisional slowing with changes to experimental stimuli on a lexical decision task found in OCD sufferers high in incompleteness points towards unique deficits in this group (Summerfeldt & Endler, 1998). Study 3 of this thesis highlights distinct differences observed between incompleteness and harm avoidance dimensions on measures of set-switching and verbal memory, which not only provides support for the core dimensions model, but also provides a new avenue of investigation for the elucidation of neuropsychological results in OCD.

Treatment of Cognitive Deficits in Obsessive-Compulsive Disorder

Despite the large body of literature concerning neuropsychological impairment in OCD, very little research has investigated the potential utility of cognitive remediation strategies in this

population. Cognitive impairments are considered central to the development of obsessive-compulsive symptoms and, in turn, quality of life in patients with OCD (Savage, 1998). For example, affected individuals may exhibit impairments in global organisation leading to aberrant doubt and uncertainty about events (Griesberg & McKay, 2003). Similarly, broad impairments in executive function can cause problems in everyday life, such as directing attention to trivial details rather than the overall context of the problem at hand. Furthermore, once individuals have successfully solved a problem, the memory of whether or not they have done so may in turn be impaired, potentially leading to chronic doubt and repetitive behaviours (Savage, 1998). Thus, not only would addressing any cognitive deficits serve to improve patients' well-being, the benefits may also extend to facilitate better learning in standard treatment approaches (e.g., cognitive behavioural therapy). Only two studies have investigated cognitive retraining in OCD (Buhlmann et al., 2006; Park et al., 2006), both of which utilized specific training strategies using modified neuropsychological tasks—a strategy which limits the external validity of any improvements in performance. There is, therefore, a need for the examination of cognitive remediation protocols that address more generally the needs of individuals with OCD, and which may offer gains more generalizable to daily life.

'Sandwich' Thesis Overview

Each of the four studies contained in this thesis corresponds to a manuscript that is submitted to or under review by an academic, peer-reviewed journal. Study 1 is a critical review of the existing literature on the topic of neuropsychological performance in symptom dimensions of OCD. It highlights the numerous differences between existing studies in terms of subtyping methodology and in the neuropsychological tasks used to assess performance in those subtypes. The extent of these differences precluded a meaningful meta-analysis of the data contained within these studies. Most significant findings were reported as negative correlations between the presence of certain symptoms and lower scores on neuropsychological tasks, with few significant group differences reported. These findings were almost exclusively limited to the doubting/checking, contamination/cleaning, and symmetry/ordering symptom dimensions, and were observed across the cognitive domains of executive function, verbal, non-verbal and working memory, with effect sizes ranging from small to large. The interpretability of these findings is limited by the methodological challenges observed across the included studies.

The manifest heterogeneity of methods for defining symptom dimensions observed in the studies included in the Study 1 review provided the impetus for Study 2 of the thesis, which aimed to compare the two most common statistical methods for determining symptom dimensions in OCD using the Y-BOCS symptom checklist—cluster analysis and factor analysis. I first sought to conduct a factor analysis using the most current and well-accepted methodology for factor analysis of categorical data, and then compared these results against cluster analysis, which is thought by some researchers to be a more appropriate approach (Calamari et al., 1999; 2004). The results of this study revealed limited differences between the final three-factor solution (comprising aggressive/checking, contamination/cleaning, and symmetry/ordering

factors) and the four-cluster solution (comprising aggressive/checking, contamination/cleaning, symmetry/ordering, and mixed symptom clusters). It was observed here that a considerable number of variables from the Y-BOCS were excluded from the analysis due to low communalities or low measures of sampling adequacy—a practice frequently neglected in previous studies on this topic—likely due to low representation of these symptoms in the study population. This, taken together with the fact that the Y-BOCS symptom categories are determined by a priori hypothesis rather than evidence-based methods, led me to conclude that the pursuit of determining symptom dimensions based on factor analysis of responses to the Y-BOCS symptom checklist is unlikely to provide meaningful results. Furthermore, although cluster analysis appears to be an attractive option, a method that groups cases rather than variables does not adequately address the question of defining latent dimensions of symptoms.

The numerous limitations of defining dimensions based on overt symptom presentation, as well as the obstacles inherent in the statistical procedures used for this method informed the research question of Study 3. Here, I sought to investigate an alternative approach to symptom dimensions of OCD using harm avoidance and incompleteness dimensions which are based on symptom *theme* rather than overt symptomatology. Although some research has investigated the validity of this model (Ecker & Gönner, 2017; Summerfeldt et al., 2014), there is a dearth of evidence to demonstrate distinct differences between these groups. I therefore aimed to assess neuropsychological performance in these two groups to determine whether any differences in cognitive function were present. The results of Study 3 showed that although individuals with incompleteness symptoms rated themselves subjectively as having greater cognitive impairment, there was little objective evidence to support this except on a measure of set-shifting and problem-solving. Additionally, the harm avoidance group performed more poorly on a task of

episodic memory compared to the incompleteness group. This supported the hypothesis that whereas incompleteness symptoms may have ties to perfectionism tendencies and are linked to difficulty in set-shifting, harm avoidance symptoms share similarities to other anxiety disorders where deficits observed in episodic memory are also obserevd (Airaksinen, Larsson, & Forsell, 2004). These findings provide early support for the core dimensions model, but remain limited by the lack of an established method for classifying patients into these dimensions.

Having discussed the neuropsychological deficits observed in OCD at length in Studies 1 through 3, I then aimed to determine whether these deficits could be addressed with a cognitive remediation protocol. Noting that there had been very little research on this topic, and that the two existing studies employed cognitive retraining protocols which the limited external validity of any improvement in functioning attained, I decided to assess the feasibility and efficacy of an established cognitive remediation protocol, GMT, which specifically addresses some of the cognitive domains consistently identified as being impaired in OCD. Given the small-to-medium effect sizes for cognitive dysfunction in this population, this study was designed as a pilot investigation to assess the initial utility of this approach. It was observed that the GMT program resulted in significant gains in subjective and objective cognitive functioning, as well as on functional outcome measures, indicating early promise for this approach as a treatment option, particularly for individuals in this population expressing difficulty with cognition.

As can be seen, the studies that comprise this thesis are inter-related. As such, there is some overlap in the content of the introduction and discussion sections, particularly in any discussion of neuropsychological performance in OCD, mentioned in all four studies, and in the discussion of the approaches to and limitations of subtyping methods in OCD. The methods and results of all four studies are unique and serve to inform one another as a whole.

REFERFENCES

Abramowitz, J., McKay, D., & Taylor, S. (2005). Special series subtypes of obsessive-compulsive disorder: introduction. *Behavior Therapy*, *36*(4), 367-369.

Airaksinen, E., Larsson, M., & Forsell, Y. (2005). Neuropsychological functions in anxiety disorders in population-based samples: evidence of episodic memory dysfunction. *Journal of psychiatric research*, *39*(2), 207-214.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.

Calamari, J. E., Wiegartz, P. S., & Janeck, A. S. (1999). Obsessive–compulsive disorder subgroups: a symptom-based clustering approach. *Behaviour Research and Therapy*, *37*(2), 113-125.

Calamari, J. E., Wiegartz, P. S., Riemann, B. C., Cohen, R. J., Greer, A., Jacobi, D. M., ... & Carmin, C. (2004). Obsessive–compulsive disorder subtypes: an attempted replication and extension of a symptom-based taxonomy. *Behaviour Research and Therapy*, *42*(6), 647-670.

Ecker, W., & Gönner, S. (2008). Incompleteness and harm avoidance in OCD symptom dimensions. *Behaviour Research and Therapy*, *46*(8), 895-904.

Ecker, W., & Gönner, S. (2017). Current Status of Research Concerning Incompleteness in Obsessive-Compulsive Disorder. *VERHALTENSTHERAPIE*, *27*(2), 120-128.

Ecker, W., Kupfer, J., & Gönner, S. (2014). Incompleteness and harm avoidance in OCD, anxiety and depressive disorders, and non-clinical controls. *Journal of Obsessive-Compulsive and Related Disorders*, *3*(1), 46-51.

Fenton, W. S., & McGlashan, T. H. (1991). Natural history of schizophrenia subtypes: II. Positive and negative symptoms and long-term course. *Archives of general psychiatry*, *48*(11), 978-986.

Freund, B., Steketee, G.S., & Foa, E.B. (1987). Compulsive activity checklist (CAC): Psychometric analysis with obsessive-compulsive disorder. *Behavioral Assessment, 9*, 67-79.

Gabbard, G. O. (1989). Two subtypes of narcissistic personality disorder. *Bulletin of the Menninger Clinic*.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., ... & Charney, D. S. (1989). Yale-brown obsessive compulsive scale (Y-BOCS). *Arch gen psychiatry*, *46*, 1006-1011.

Greisberg, S., & McKay, D. (2003). Neuropsychology of obsessive-compulsive disorder: a review and treatment implications. *Clinical Psychology Review*, *23*(1), 95-117.

Janet, P. (1903). *Les obsessions et la psychasthénie*. Paris: Alcan.

Karno, M., Golding, J., Sorenson, S., & Burnam, M. A. (1988). The epidemiology of obsessive-compulsive disorder in five U.S. communities. *Archives of General Psychiatry, 45*, 1094-1099.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*, 593-602.

Koch, K., Reeß, T. J., Rus, O. G., Zimmer, C., & Zaudig, M. (2014). Diffusion tensor imaging (DTI) studies in patients with obsessive-compulsive disorder (OCD): a review. *Journal of psychiatric research*, *54*, 26-35.

Kuelz, A. K., Hohagen, F., & Voderholzer, U. (2004). Neuropsychological performance in obsessive-compulsive disorder: a critical review. *Biological psychology*, *65*(3), 185-236.

Lanius, R. A., Vermetten, E., Loewenstein, R. J., Brand, B., Schmahl, C., Bremner, J. D., & Spiegel, D. (2010). Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. *American Journal of Psychiatry*, *167*(6), 640-647.

Lewis, A.J. (1936). Problems of obsessional illness. *Proceedings of the Royal Society of Medicine*, *29*, 325-336.

McKay, D., Abramowitz, J. S., Calamari, J. E., Kyrios, M., Radomsky, A., Sookman, D., ... & Wilhelm, S. (2004). A critical evaluation of obsessive–compulsive disorder subtypes: symptoms versus mechanisms. *Clinical psychology review*, *24*(3), 283-313.

Menzies, L., Chamberlain, S. R., Laird, A. R., Thelen, S. M., Sahakian, B. J., & Bullmore, E. T. (2008). Integrating evidence from neuroimaging and neuropsychological studies of obsessive-compulsive disorder: the orbitofronto-striatal model revisited. *Neuroscience & Biobehavioral Reviews*, *32*(3), 525-549.

Milad, M. R., & Rauch, S. L. (2012). Obsessive-compulsive disorder: beyond segregated cortico-striatal pathways. *Trends in cognitive sciences*, *16*(1), 43-51.

Modell, J. G., Mountz, J. M., Curtis, G. C., & Greden, J. F. (1989). Neurophysiologic dysfunction in basal ganglia/limbic striatal and thalamocortical circuits as a pathogenetic mechanism of obsessive-compulsive disorder. *The Journal of neuropsychiatry and clinical neurosciences*.

Murayama, K., Nakao, T., Sanematsu, H., Okada, K., Yoshiura, T., Tomita, M., ... & Kanba, S. (2013). Differential neural network of checking versus washing symptoms in obsessive-compulsive disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *40*, 160-166.

Nakao, T., Okada, K., & Kanba, S. (2014). Neurobiological model of obsessive–compulsive disorder: evidence from recent neuropsychological and neuroimaging findings. *Psychiatry and clinical neurosciences*, *68*(8), 587-605.

Philpott, R. (1975). Recent advances in the behavioural assessment of obsessional illness: Difficulties common tothese and other measures. *Scottish Medical Journal*, *20*, 33-40 (Suppl.).

Pitman, R. K. (1987). Pierre Janet on obsessive-compulsive disorder (1903): review and commentary. *Archives of General Psychiatry*, *44*(3), 226-232.

Radomsky, A. S., & Taylor, S. (2005). Subtyping OCD: Prospects and problems. *Behavior Therapy*, *36*(4), 371-379.

Rapoport, J. L. (1990). Obsessive compulsive disorder and basal ganglia dysfunction. *Psychological Medicine*, *20*(3), 465-469.

Rapoport, J. L. (1991). Basal ganglia dysfunction as a proposed cause of obsessive-compulsive disorder. *Psychopathology and the brain*, 77-95.

Savage, C.R. (1998). Neuropsychology of OCD: Research findings and treatment implications. In Jenike, M.A., Baer, L., & Minichiello, W.E. (Eds.), *Obsessive-Compulsive Disorders: Practical Management* (254-275). St. Louis, MO: Mosby.

Savage, C. R., Baer, L., Keuthen, N. J., Brown, H. D., Rauch, S. L., & Jenike, M. A. (1999). Organizational strategies mediate nonverbal memory impairment in obsessive–compulsive disorder. *Biological psychiatry*, *45*(7), 905-916.

Saxena, S., Brody, A. L., Schwartz, J. M., & Baxter, L. R. (1998). Neuroimaging and frontal-subcortical circuitry in obsessive-compulsive disorder. *The British Journal of Psychiatry*.

Smits, M. L., Feenstra, D. J., Bales, D. L., Vos, J., Lucas, Z., Verheul, R., & Luyten, P. (2017). Subtypes of borderline personality disorder patients: a cluster-analytic approach. *Borderline personality disorder and emotion dysregulation*, *4*(1), 16.

Summerfeldt, L. J. (2004). Understanding and treating incompleteness in obsessive-compulsive disorder. *Journal of clinical psychology*, *60*(11), 1155-1168.

Summerfeldt, L. J., & Endler, N. S. (1998). Examining the evidence for anxiety-related cognitive biases in obsessive-compulsive disorder. *Journal of anxiety disorders*, *12*(6), 579-598.

Summerfeldt, L. J., Kloosterman, P. H., Antony, M. M., & Swinson, R. P. (2014). Examining an obsessive-compulsive core dimensions model: Structural validity of harm avoidance and incompleteness. *Journal of Obsessive-Compulsive and Related Disorders*, *3*(2), 83-94.

Sanavio, E. (1988). Obsessions and compulsions: The Padua Inventory. *Behavior Research and Therapy*, *26*, 169-177.

Saxena, S., & Rauch, S. L. (2000). Functional neuroimaging and the neuroanatomy of obsessive-compulsive disorder. *Psychiatric Clinics*, *23*(3), 563-586.

Tallis, F. (1997). The neuropsychology of obsessive—compulsive disorder: A review and consideration of clinical implications. *British Journal of Clinical Psychology*, *36*(1), 3-20.

van Oppen, P., Hoekstra, R.J., & Emmelkamp, P.M.G. (1995). The structure of obsessive-compulsive symptoms. *Behavior Research and Therapy*, *33*, 379-390.

CHAPTER 2

STUDY 1

TITLE: Neuropsychological performance across symptom dimensions of obsessive-compulsive disorder: A brief report and critical review of the literature

AUTHORS: Duncan H. Cameron, B.A.; Margaret C. McKinnon, Ph.D., C.Psych.; Karen Rowa, Ph.D., C.Psych.; Neil A. Rector, Ph.D., C.Psych.; Randi E. McCabe, Ph.D., C.Psych.

CONTEXT AND IMPLICATIONS OF THIS STUDY: This first study of the 'sandwich' thesis reviews the literature examining differences in neuropsychological performance between symptom-based subtypes of OCD. As the background section of this thesis suggests, there are both limitations in conventional methods for subtyping in OCD, as well as in previous investigations of neuropsychological performance in OCD, where some of the heterogeneity of findings may be attributable to differences between symptom dimensions. This is the first study attempting to assess differences across all observed symptom dimensions mentioned in the extant research literature. It serves primarily to highlight the substantive shortcomings of previously conducted studies. It also provides direction to the field on how it should proceed so that future study can accurately address the question of whether there are distinct differences in neuropsychological performance between symptom dimensions of OCD.

This review shows that this is a relatively neglected area of study, and in the research that does exist, there are significant methodological challenges and considerable variability in terms of the subtyping methods and neuropsychological tasks employed to assess function. Despite these issues, this review is able to show that there may indeed be important differences between symptom dimensions, particularly in the domain of executive function.

ACKNOWLEDGEMENTS

I would like to thank Dr. David Streiner for his guidance in the calculation of effect sizes based on the data available from each of the papers included in this review.

CONFLICTS OF INTEREST: None.

PUBLISHED IN: Journal of Clinical and Experimental Neuropsychology (under review).

Abstract

Background: It is recognized that obsessive-compulsive disorder (OCD) is a heterogeneous disorder, with multiple symptom profiles. Delineating the neuropsychological characteristics associated with previously identified symptom clusters may therefore be useful in assisting to better define symptom subtypes of OCD. *Method*: A literature search was performed covering dates 1806 to March 2016 for all peer-reviewed articles reporting neuropsychological task performance across symptom dimensions of OCD. *Results*: The search yielded 4,096 total references, 17 of which met inclusion criteria for this review. Two additional references were retrieved from alternative sources, yielding a total of 19 included references. Neuropsychological impairments relative to matched controls were observed primarily in patients with Checking, Washing, and Symmetry symptoms, where deficits clustered generally in the domains of executive function, verbal, non-verbal and working memory, with effect sizes ranging anywhere from small to large. Despite this pattern, there was significant variability within subtypes both with respect to the presence or absence of deficits and the domains of neuropsychological functioning affected. *Conclusions*: The current state of the literature precludes a meaningful meta-analysis of cognitive dysfunction across the breadth of symptom subtypes of OCD. This is due primarily to significant methodological differences observed between studies, both in terms of neuropsychological measures and symptom subtyping methods employed. Future studies addressing these limitations should include more consistent measures with the aim of reproducing the results of previous research to identify more concrete patterns of neuropsychological performance across subtypes. These efforts will be useful in clarifying whether there are unique symptom dimensions of OCD.

Keywords: obsessive-compulsive disorder, neuropsychological deficits, symptom theme

Introduction

Obsessive-compulsive disorder (OCD) is a heterogeneous disorder with clinical presentations differing greatly across individuals. Longstanding efforts have sought to refine further our understanding of this disorder by examining symptom subtypes of OCD that represent more homogeneous clusters. Viewing diagnostic groups as homogeneous entities is useful for both clinical and research purposes. Specifically, heterogeneous groups limit the ability of clinicians to specify treatment options, and to predict treatment outcomes and/or clinical course as accurately as possible. Similarly, identification of distinct groups contributes to accuracy in research where unidentified heterogeneity may leave significant sources of variance overlooked.

Although there appears to be value in identifying meaningful subtypes in OCD, there have been challenges to this work. Part of the difficulty in diagnosing OCD subtypes is that, to date, no subtypes have been well-defined and there is limited consensus surrounding specific subtypes. This is due to a number of issues including, prominently, the paucity of literature that defines concretely homogeneous subtypes. Moreover, in the literature that does exist, methods for identifying subtypes vary between studies, and even those with similar methods reveal diverse results. To date, the most popular method for identifying possible symptom subtypes has been the use of factor analysis of the results of clinical interviews and scales, including the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, & Mazure, 1989) and the Obsessive-Compulsive Inventory-Revised (OCI-R; Foa et al., 2002). Several studies have also used cluster analysis as an alternative approach (Abramowitz, Franklin, Schwartz, & Furr, 2003; Calamari, Wiegartz, & Janeck, 1999; Calamari et al., 2004). However, even if factor analytic approaches converge in their results, the symptom “dimensions” highlighted by this method are not necessarily synonymous with “subtypes.”

Taken together, these findings beg mention of one further, major methodological issue in discussing subtyping OCD, which is the statistical quantification of symptom checklists described in previous literature is only able to identify symptom dimensions, rather than distinct entities. Furthermore, many researchers and clinicians would argue the validity and necessity of homogeneous subtypes, as it cannot be assumed that any individual can be defined by one symptom category given the heterogeneous and often comorbid symptom presentation seen on an individual basis in this disorder. As McKay et al. (2004) note in their critical review of OCD symptom subtypes, the most prudent avenue might be the definition of primary, secondary or tertiary subtypes, should these groups be defined by symptom. Hence, although the broad notion of diagnosing OCD using distinct subtypes is attractive, identification of specific subtypes remains elusive. Reviewing all of the necessary evidence to first delineate *symptom dimensions* is likely the most appropriate method for monitoring whether distinct *subtypes* exist for this disorder. Given this challenge, the subgroups discussed in this paper will be referred to on a dimensional basis, rather than categorical, as even in those studies reporting results of between-groups analyses (whether they performed a factor analysis of their own sample or allocated participants based on results of previous factor analytic studies) it cannot be concluded that these subgroups were truly homogeneous.

Although the extant literature remains equivocal, some trends, however, do appear despite the names for these dimensions often varying. The two symptom patterns present in almost every factor analytic study are “Symmetry obsessions/Ordering compulsions,” and “Contamination obsessions/Cleaning compulsions.” “Checking compulsions” have also been identified in the majority of studies. There has, however, been significant discordance in the associated obsessions, which tend to contain some combination of Aggressive, Sexual, Religious

and Somatic intrusive thoughts. Other frequently identified dimensions include "Hoarding obsessions and compulsions*,"* as well as a "Mixed Symptom profile" dimension. The results of a meta-analysis of factor analytic studies yielded four symptom categories, which included Symmetry, Forbidden Thoughts, Cleaning and Hoarding (Bloch, Landeros-Weisenberger, Rosario, Pittenger, & Leckman, 2008). The findings of this meta-analysis, however, are limited by the fact that all studies are based on factor analysis of the Y-BOCS, which has been criticized for several reasons, one of which being its lack of items assessing avoidance. Abramowitz et al. (2010) aimed to address the issues with previous measures by developing the Dimensional Obsessive Compulsive Scale (DOCS) using items related to those dimensions most consistently identified in existing literature. These dimensions included Contamination, Responsibility, Unacceptable Thoughts and Symmetry (with Hoarding compulsions notably excluded as many researchers viewed these symptoms as a distinct syndrome even prior to its definition in the Diagnostic and Statistical Manual of Mental Disorders, fifth edition [DSM-5]; American Psychiatric Association, 2013).

Given the disparity in results emerging from various efforts to identify OCD subtypes, it would be helpful to identify additional factors that may contribute to heterogeneity in symptom presentation in this disorder and that may explain, in part, these discrepant findings. Neuropsychological functioning has been studied widely in OCD and may represent one key factor that may be associated with differences in symptom presentation (and thus associated subtypes). A recent meta-analysis (Abramovitch, Abramowitz, & Mittelman, 2013) of 115 studies examining neuropsychological performance in OCD revealed performance impairments relative to healthy controls across a wide variety of cognitive domains (including attention, executive function, memory, processing speed, visuospatial abilities, and working memory), with

mean effect sizes ranging from Cohen's *d* = -0.332 (for verbal memory) to *d* = -0.761 (for non-verbal memory) across cognitive subdomains (where Cohen's *d* is interpreted as small = 0.20, moderate = 0.50, and large = 0.80; Cohen, 1988). A meta-analysis by Shin, Lee, Kim, and Kwon (2014) reported similar results, highlighting small-to-medium effect sizes in deficits in executive function, visuospatial memory, verbal memory and verbal fluency; notably, fewer studies were included in this review (see also Nakao, Okada, & Kanba, 2014 for a review of neuropsychological and neuroimaging studies in OCD). Critically, the results of these meta-analyses represent the averaged effect sizes across a wide corpus of individual studies of neuropsychological functioning, converging on the conclusion of widespread alterations in neuropsychological functioning in OCD. By contrast, inspection of individual reports of neuropsychological functioning in OCD reveal mixed findings, and previous attempts to model this heterogeneity have not found significant moderators. In a recent critical review of the challenges associated with assessment of neuropsychological function the authors reported that 47% of papers assessing neuropsychological performance in OCD did not report on symptom severity, and of the studies that did, none attempted to assess a non-linear association, leaving out the potential influence of OCD symptom dimensions (Abramovitch, Mittelman, Tankersley, Abramowitz, & Shweiger, 2015). This suggests that differing presentations of OCD may result in heterogeneous neuropsychological profiles, thus confounding study findings.

Given the difficulty of identifying consistently defined symptom dimensions *and* neuropsychological impairments in OCD, it follows that these might be fundamentally linked. The design of neuropsychological studies in OCD comparing groups across symptom dimensions might in turn assist in establishing more specific parameters of distinct OCD subtypes. Indeed, defining the pattern of neuropsychological impairments associated with

different dimensions of OCD might aid in further characterizing illness severity and in the prediction of treatment response (as well as any number of other disease correlates).

A relatively small number of studies have explored differences in neuropsychological functioning across OCD symptom dimensions, the majority of which have focused on differences between “Checkers” and “Washers” (or, sometimes, “Non-Checkers”). Leopold and Backenstrass (2015) recently conducted a meta-analysis of the results of 13 studies of Washers and Checkers, and reported performance deficits in Checkers compared to Washers with small effect sizes in sustained attention, encoding strategies, verbal memory, nonverbal memory, and processing speed, and large effect sizes in planning/problem solving and response inhibition. Overall, Checkers tended to exhibit significantly worse performance in most cognitive domains than did Washers. Although this meta-analysis provides an extensive examination of the existing literature concerning Washers and Checkers, it did not investigate potential differences in neuropsychological performance across other symptom dimensions.

Accordingly, we sought to conduct a comprehensive review of the existing literature on differences in neuropsychological performance across all symptom dimensions of OCD. Here, symptom dimensions were defined using symptom-based checklists and measures (e.g., Y-BOCS, OCI-R, or Padua Inventory), following the precedent in the literature of this being the most common way to characterize symptom domains. Given the wide range of symptom dimensions, small number of studies examining neuropsychological performance, and discrepant methodology and reporting of results in these studies, it was not possible to conduct a meta-analytic review of these findings as was the case for comparisons between Washers and Checkers (Leopold & Backenstrass, 2015). If neuropsychological results clustered in particular symptom dimensions, this finding would lend incremental support to the hypothesized existence

of underlying, unique symptom subtypes. Our review focused on cortico-striatal-mediated neuropsychological domains most commonly impacted in neuropsychiatric illness (Salloway, Malloy, & Duffy, 2008; Waltz, 2005) and included memory (working, visual and verbal), visuospatial reconstruction, executive functioning, processing speed and attention—domains that have been of significant interest in the literature surrounding neuropsychological functioning in OCD. Based on previous literature, we hypothesized that different symptom dimensions, particularly checking symptoms, may be associated with distinct neuropsychological deficits.

Methods

We conducted searches of MEDLINE (1946-present), PsycINFO (1806-present) and Embase (1974-present), covering dates 1806 to December week 1 2016, using the following terms: “obsessive-compulsive disorder,” “OCD,” “neuropsychology,” “neuropsychological tests,” “cognitive function,” “executive function,” “processing speed,” “memory,” and “attention.” In order to perform a comprehensive search of the literature, a professional Scientific Librarian at McMaster University was consulted to determine the appropriate keywords and databases to search.

Included articles had to be original, peer-reviewed research papers, and had to directly address neuropsychological performance in symptom dimensions of OCD using standardized neuropsychological tests (with the exception of the negative priming protocols described below). Editorials, letters, case studies, conference abstracts and review papers were excluded, although reviews were screened for additional references that might have been missed in the original search. Included articles were also limited to those written in English. After initial abstract review, all papers that met the above criteria were read and those that reported analyses of

neuropsychological performance across any "subtypes" or "dimensions/domains" (symptom-based) of OCD were included in the review. The screening process to include papers was completed by two independent raters, and any discrepancies were resolved by consensus. Given the dearth of literature in this area, all relevant studies were deemed eligible for this review, including those with no control group, and those with a pediatric population. Although some studies also assessed Hoarding symptoms as an OCD dimension, results for this group were excluded given that it now constitutes its own homogeneous diagnosis. Data were extracted onto a data extraction sheet, designed for this review, using the computer program Excel.

To facilitate meaningful comparisons across studies, we calculated effect sizes (Cohen's *d*) in all instances when they were not reported, using methodology outlined by Lipsey & Wilson (2001). Effect sizes already reported in individual papers were not converted to Cohen's *d* (these included non-parametric effect sizes, Pearson's *r*/Spearman's rho/Kendall's tau correlations, and η^2). All effect sizes were interpreted using Cohen's guidelines (**Cohen's *d*:** small = 0.20, medium = 0.50, large = 0.80; **eta-squared:** small = 0.02, medium = 0.13, large = 0.26; **non-parametric:** small = 0.10, medium = 0.30, large = 0.50; **correlation Pearson's *r*/ Spearman's rho/Kendall's tau:** small = 0.10, medium = 0.30, large = 0.50; Cohen, 1988). Significant results were then categorized by effect size (small, medium, large) in Table 1.

The different symptom dimensions assessed across studies should be noted. One group performed their own factor analysis of symptom reports (Jang et al., 2010), some adapted subtypes from previous factor analytic studies (Hashimoto et al., 2011; McGuire et al., 2014; Omori et al., 2007), and others used the symptom clusters defined by the Y-BOCS (Abbruzzese, Bellodi, Ferri, & Scarone, 1993; Abbruzzese, Ferri, & Scarone, 1995; Ceschi, Van der Linden, Dunker, Perroud, & Brédart, 2003; Cha et al., 2008; Dittrich, & Johansen, 2013; Exner, Martin,

& Rief, 2009; Hashimoto et al., 2008; Nakao et al., 2009; Nedeljkovic et al., 2009; Rasmussen, Siev, Abramovitch, & Wilhelm, 2016), dimensional Y-BOCS (DY-BOCS; Martoni, Salgari, Galimberti, Cavallini, & O'Neill, 2015; Pedron, Ferrão, Gurgel, & Reppold, 2015), or Maudsley Obsessional-Compulsive Inventory (MOCI; MacDonald, Antony, MacLeod, & Swinson, 1999; Penadés, Catalán, Andrés, Slamero, & Gastró, 2005) to identify an individual's most prominent symptoms.

Results

We screened a total of 4,098 articles initially by title, 3,660 of which were immediately identified as irrelevant to the question of this paper. A further 424 studies were reviewed by abstract, and 17 were found that pertained to neuropsychological assessment of obsessive-compulsive disorder symptom dimensions. One additional paper was found from the reference list of one of the included papers, and another was found from the reference list of a review retrieved in the literature search, for a total of 19 articles included (see Figure 1 for the article selection process).

Included Articles

After reviewing the results across all 19 studies, it was decided that nature of the results precluded a meaningful meta-analysis or synthesis of common findings in a narrative format. As such the results are reported briefly below in-text, while significant results for each neuropsychological variable are presented in Table 1. In addition, to demonstrate the ratio of significant to insignificant results a full list of variables assessed from each study is available in Table 2.

Executive Function

Symptom Dimension Associations. The Contamination/Cleaning dimension showed the greatest number of negative correlations with performance on tasks of executive functioning. Interestingly, one study (Hashimoto et al., 2011) found better performance associated with this dimension for some tests of executive functioning. Heightened endorsement of Checking and Symmetry/Ordering symptoms was also associated with impaired performance on some tasks, although to a lesser extent than the Contamination/Cleaning dimension.

Group differences. Significant differences were reported largely for Checkers, who demonstrated worse performance relative to the Contamination/Cleaning, Symmetry/Ordering, Repeating/Counting, Obsessionals/Few Compulsions and healthy controls. The Symmetry/Ordering group showed better performance for a smaller number of tasks relative to other dimensions, while the Contamination/Cleaning dimension did not show deficits in performance relative to any symptom dimensions. A number of studies were not associated with any significant group differences or correlations (Abbruzzese et al., 1995; Cha et al., 2008; Hashimoto et al., 2008; Omori et al., 2007; Penadés et al., 2005).

Processing Speed

A total of five studies assessed processing speed with three finding no significant differences or correlations (Hashimoto et al., 2008; Nakao et al., 2009; Omori et al., 2007) while Hashimoto et al. (2011) found a positive correlation between Contamination/Cleaning symptom endorsement and processing speed (i.e., endorsement of symptoms related to greater processing speed) and McGuire et al. (2014) found that the Symmetry/Ordering group performed more poorly on a processing speed measure than did other symptom groups.

Attention

Only one study (Pedron et al., 2015) found a significant negative association between endorsement of Contamination/Cleaning or Symmetry/Ordering symptoms with selective attention as measured by Go/No-Go omission errors, while Nakao et al. (2009) assessed selective attention with an N-back task and did not find any significant differences between symptom subgroups.

Visuospatial Reconstruction

This domain was assessed predominantly using the Rey-Osterreith Complex Figure Task (RCFT) copy trial; no significant differences were found between symptom categories in any studies retrieved. One paper (Pedron et al., 2015) reported a negative correlation between visuospatial reconstruction and Contamination/Cleaning symptoms, indicating poorer performance associated with endorsement of these symptoms.

Verbal Memory

Symptom Dimension Associations. Only one study (Hashimoto et al., 2011) reported significant correlations between symptom dimensions and tasks of verbal memory. Here, the Symmetry/Ordering dimension showed a negative correlation with performance on the immediate and delayed recall trials of the Wechsler Memory Scale Revised (WMS-R) Logical Memory task, while the Contamination/Cleaning dimension showed a positive association, indicating better performance on this task related to endorsement of these symptoms.

Group differences. Few significant group differences were reported for verbal memory performance between dimensions. Whereas checkers showed significantly lower Wechsler Memory Scale (WMS-R) Verbal Memory scores compared to the Contamination/Cleaning dimension in one study, both the Checking and Contamination/Cleaning dimensions

demonstrated worse performance on the CVLT compared to healthy controls in other studies, although they did not differ from each other. Six studies showed no significant group differences or associations with any neuropsychological variables (Ceschi et al., 2003; Cha et al., 2008; Exner et al., 2009; Hashimoto et al., 2008; McGuire et al., 2014; Nakao et al., 2009).

Non-Verbal Memory

Symptom Dimension Associations. In the domain of non-verbal memory, the Symmetry/Ordering dimension was negatively correlated with indicators of non-verbal memory. The Checking dimension was also negatively correlated with time to complete RCFT trials.

Group differences. The Checking dimension showed significant deficits in performance compared to other subgroups in this domain. One study also reported impaired performance on a non-verbal memory task in the Obsessions/Few Compulsions dimension (which includes Symmetry). The Contamination/Cleaning dimension did not demonstrate poorer performance on any tasks in this domain relative to other dimensions. Four studies reported no significant group differences or associations (Hashimoto et al., 2008; McGuire et al., 2014, Omori et al., 2007; Pedron et al., 2015).

Working Memory

There were generally no differences between symptom dimensions on working memory tasks (Exner et al., 2009; Hashimoto et al., 2008; MacDonald et al., 1999; Nakao et al., 2009; Pedron et al., 2015). One study reported that the Checking and Obsessions/Few Compulsions (which includes Symmetry) subgroups had impaired performance compared to other dimensions and healthy controls on the CANTAB Spatial Working Memory task (Nedeljkovic et al., 2009), although the groups did not differ from each other. Checkers also performed more poorly than

the Contamination/Cleaning dimension on a negative priming task in another study (Hoenig et al., 2002).

General Intelligence

There were also several studies assessing measures of global functioning (which also covers aspects of semantic memory) including the Wechsler Abbreviated Scale of Intelligence (WASI) Performance IQ (Exner et al., 2009; Pedron et al., 2015), Verbal IQ (Pedron et al., 2015) and a three-factor adaptation of the WMS-R (Abbruzzese et al., 1995). None of the studies found any significant associations between these measures, save Pedron et al. (2015) who found a negative correlation between endorsement of Contamination/Cleaning symptoms and the WASI Performance IQ index.

Discussion

The purpose of this review was to summarize and comment on the current state of the existing literature investigating neuropsychological task performance across different symptom dimensions of OCD. Theoretical reasoning suggests that specific neuropsychological profiles associated with particular symptom dimensions may offer useful clues about underlying symptom subtypes, an important but still not well understood area of research. It is clear from the above results that the findings of studies examining the neuropsychology of particular symptom dimensions of OCD are equivocal. As such, much of our discussion here focuses on the reasons why these results might be so equivocal, with the aim of informing future research.

The majority of the results from the studies described in this review found few significant neuropsychological deficits between symptom dimensions. However, from the significant findings that were present, it can be seen that impairments in several cognitive domains generally

stand out across three primary symptom dimensions: Checking, Washing and Symmetry. Individuals with Checking symptoms displayed the broadest impairments in performance relative to other groups. These impairments spanned across several domains, including executive function, verbal memory, non-verbal memory and working memory. Indicators of Washing symptoms showed strong correlations with difficulties in executive function, processing speed, visuospatial reconstruction, verbal and non-verbal memory. Interestingly several positive correlations were also reported indicating relations between washing symptoms and stronger performance on some neuropsychological variables, although it should be noted that these results were seen largely in one study (Hashimoto et al., 2011). Those with Symmetry symptoms had somewhat widespread impairments, with significant results—shown by both negative associations between symptom endorsement and task performance, and in group differences—reported for the domains of executive function, processing speed, visuospatial reconstruction, verbal, non-verbal and working memory. The effect sizes for these findings tended to range evenly from small to large. These results suggest that there are some distinct neuropsychological features related to particular symptom dimensions. However, it is quite possible that the differences in results observed are due, in large part, to methodological differences between studies and substantially more research linking neuropsychological profiles with symptom dimensions is needed before we can extrapolate the findings to better understand whether there are distinct and meaningful subtypes in OCD.

Simply because no concrete conclusions can be drawn from the existing studies, however, does not mean that this is not an important endeavor. As stated earlier, the majority of neuropsychological studies in OCD do not distinguish between symptom presentations, meaning that if any dimensions are represented with varying frequencies in a given study, then the results

yielded could be significantly biased. Even research designs comparing Checkers and Non-Checkers may conceal differences by clustering all other symptom presentations in to one "non-checking" category. This consideration should be noted when interpreting the results of previous findings or designing future studies of neuropsychological performance in OCD.

Deficits in executive functioning were the most frequently reported in the studies reviewed here. Executive functioning is a highly-referenced domain in the study of OCD, and, as can be seen in Table 1, the studies in this review implemented considerably more tests of executive function compared to other domains. Although several domains of memory were also assessed in most studies, it should be noted that the over-emphasis on executive function can lead to a potential bias in results. Although deficits in this domain have been relatively well-documented in OCD, given the increasing acceptance of this disorder as a heterogeneous condition it would be wise to include more comprehensive assessments of neuropsychological function in future study.

One form of bias that should be noted is the representation of subtypes across studies, as several studies chose to focus solely on Checkers and Washers (Ceschi et al., 2003; Cha et al., 2008; Dittrich & Johansen, 2013; Nakao et al., 2009) or, more broadly, Checkers and Non-Checkers (Exner et al., 2009; Hoenig et al., 2002; MacDonald et al., 1999). In fact, even several studies that used more comprehensive strategies only chose to focus on a select few of these dimensions. For example, Omori et al. (2007) employed a 5-dimension model from Mataix-Cols et al. (1999) but only chose to examine Washers and Checkers, while Nakao et al. (2009) used the Y-BOCS to define several groups but only investigated Washers and Checkers (out of the 8 possible obsession symptom clusters, and 7 compulsion clusters). While the results from these studies have shed light on the possible neuropsychological dysfunction associated with these

subtypes, based on the results reported in this review it is likely that there are more than two distinct symptom subtypes associated with this disorder. Even in studies employing 4-5 symptom dimensions, the more common symptom presentations (i.e., Checking ,Washing and Symmetry) tend to result in disproportionate group sizes. Future research should aim to investigate symptom dimensions more broadly, with particular emphasis on greater representation of the less common symptom dimensions (such as Scrupulous or Repeating/Counting symptoms).

The wide variation in methods across these studies should also be acknowledged. While there were several tasks used frequently across studies, such as the TMT, the WCST, the RCFT and the WMS-R, many other tasks were only used once. The fact that most studies used separate measures for similar cognitive domains underscores the discretion that must be taken in interpreting the results. Additionally, although most papers did involve a control group, only five (Cha et al., 2008; Dittrich & Johansen, 2013; MacDonald et al., 1999; Nedeljkovic et al., 2009; Rasmussen et al., 2016) compared the control groups against OCD subgroups; most others compared OCD as a whole to controls, and compared subtypes only to each other. Although it is informative to know how subtype groups perform relative to one another, the utility of this information is limited without a control comparison.

Several recommendations for future study can be taken from the findings reported in this review. As mentioned, examination of subtypes of OCD will need to involve substantially large samples in order to accommodate multiple possible subtypes and maintain sufficient power. Additionally, a more standardized neuropsychological battery should be applied in order to increase the generalizability of results. This issue can be addressed—while simultaneously reducing type I error rate within individual studies—by adopting a strategy similar to that of MacDonald et al. (1999) by reporting neuropsychological variables grouped into their respective

cognitive domains rather than reporting results for the copious variables provided from each measure. Finally, although there is no standardized method for subtyping individuals with OCD based on symptoms, it would serve researchers well to employ the few evidence-based symptom measures that allow for dimensions to be identified, such as the DOCS (Abramowitz et al., 2010) or the Dimensional Y-BOCS (Rosario-Campos et al., 2006).

In summary, the aim of this article was to comment on the state of the existing literature examining differences in neuropsychological performance between symptom dimensions of obsessive-compulsive disorder. Generally, the research summarized in this review found that individuals in the Washing, Checking and Symmetry categories may well have impairments across several cognitive domains relative to other symptom categories, but this cannot be concluded concretely given the differences in methodology observed between these studies. The research that exists on this topic is not yet extensive enough to generalize to a larger population. However, by highlighting any issues with existing research, we hope that identifying potential differences in neuropsychological profiles between symptom dimensions could provide the foundation for the establishment of better-defined symptom subtypes for obsessive-compulsive disorder. We would encourage future study of neuropsychological performance to incorporate analysis of symptom dimensions whenever possible, attempting to apply methodology consistent with previous study.

References

Abbruzzese, M., Bellodi, L., Ferri, S., & Scarone, S. (1993). Memory functioning in obsessive-compulsive disorder. *Behavioural neurology*, *6*(3), 119-122.

Abbruzzese, M., Ferri, S., & Scarone, S. (1995). Wisconsin Card Sorting Test performance in obsessive-compulsive disorder: no evidence for involvement of dorsolateral prefrontal cortex. *Psychiatry research*, *58*(1), 37-43.

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: A meta-analysis. *Clinical psychology review*, *33*(8), 1163-1171.

Abramovitch, A., Mittelman, A., Tankersley, A.P., Abramowitz, J.S., Shweiger, A. (2015). Neuropsychological investigations in obsessive-compulsive disorder: A systematic review of methodological challenges. *Psychiatry Research*, 228, 112-120.

Abramowitz, J. S., Deacon, B. J., Olatunji, B. O., Wheaton, M. G., Berman, N. C., Losardo, D., ... & Björgvinsson, T. (2010). Assessment of obsessive-compulsive symptom dimensions: Development and evaluation of the Dimensional Obsessive-Compulsive Scale. *Psychological assessment*, *22*(1), 180.

Abramowitz, J. S., Franklin, M. E., Schwartz, S. A., & Furr, J. M. (2003). Symptom presentation and outcome of cognitive-behavioral therapy for obsessive-compulsive disorder. *Journal of consulting and clinical psychology*, *71*(6), 1049-1056.

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

Bloch, M. H., Landeros-Weisenberger, A., Rosario, M. C., Pittenger, C., & Leckman, J. F. (2008). Meta-analysis of the symptom structure of obsessive-compulsive disorder. *American Journal of Psychiatry*, *165*(12), 1532-1542.

Calamari, J. E., Wiegartz, P. S., & Janeck, A. S. (1999). Obsessive–compulsive disorder subgroups: a symptom-based clustering approach. *Behaviour Research and Therapy*, *37*(2), 113-125.

Calamari, J. E., Wiegartz, P. S., Riemann, B. C., Cohen, R. J., Greer, A., Jacobi, D. M., ... & Carmin, C. (2004). Obsessive–compulsive disorder subtypes: an attempted replication and extension of a symptom-based taxonomy. *Behaviour research and therapy*, *42*(6), 647-670.

Ceschi, G., Van der Linden, M., Dunker, D., Perroud, A., & Brédart, S. (2003). Further exploration memory bias in compulsive washers. *Behaviour Research and Therapy*, *41*(6), 737-747.

Cha, K. R., Koo, M. S., Kim, C. H., Kim, J. W., Oh, W. J., Suh, H. S., & Lee, H. S. (2008). Nonverbal memory dysfunction in obsessive-compulsive disorder patients with checking compulsions. *Depression and anxiety*, *25*(11), E115-E120.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. Academic press.

Dittrich, W. H., & Johansen, T. (2013). Cognitive deficits of executive functions and decision-making in obsessive-compulsive disorder. *Scandinavian journal of psychology*, *54*(5), 393-400.

Exner, C., Martin, V., & Rief, W. (2009). Self-focused ruminations and memory deficits in obsessive–compulsive disorder. *Cognitive Therapy and Research*,*33*(2), 163-174.

Foa, E.B., Huppert, J.D., Leiberg, S., Hajcak, G., Langner, R., et al. (2002). The Obsessive-Compulsive Inventory: Development and validation of a short version. *Psychological Assessment*, 14, 485-496.

Goodman, W.K., Price, L.H., Rasmussen, S.A., & Mazure, C. (1989). The Yale-Brown obsessive-compulsive scale: Development, use and reliability. *Archives of General Psychiatry*, 46, 1006-1011.

Hashimoto, N., Nakaaki, S., Omori, I. M., Fujioi, J., Noguchi, Y., Murata, Y., ... & Furukawa, T. A. (2011). Distinct neuropsychological profiles of three major symptom dimensions in obsessive–compulsive disorder. *Psychiatry Research*,*187*(1), 166-173.

Hashimoto, T., Shimizu, E., Koike, K., Orita, Y., Suzuki, T., Kanahara, N., ... & Iyo, M. (2008). Deficits in auditory P50 inhibition in obsessive–compulsive disorder. *Progress in Neuro-psychopharmacology and Biological Psychiatry*,*32*(1), 288-296.

Hoenig, K., Hochrein, A., Müller, D. J., & Wagner, M. (2002). Different negative priming impairments in schizophrenia and subgroups of obsessive–compulsive disorder. *Psychological Medicine*, *32*(3), 459-468.

Jang, J. H., Kim, H. S., Ha, T. H., Shin, N. Y., Kang, D. H., Choi, J. S., ... & Kwon, J. S. (2010). Nonverbal memory and organizational dysfunctions are related with distinct symptom dimensions in obsessive-compulsive disorder. *Psychiatry research*, *180*(2), 93-98.

Lipsey, M.W., & Wilson, D.B. (2001). *Practical meta-analysis*. Sage Publications, Inc.

Leopold, R., & Backenstrass, M. (2015). Neuropsychological differences between obsessive-compulsive washers and checkers: A systematic review and meta-analysis. *Journal of anxiety disorders*, *30*, 48-58.

MacDonald, P. A., Antony, M. M., MacLeod, C. M., & Swinson, R. P. (1999). Negative priming for obsessive-compulsive checkers and noncheckers. *Journal of Abnormal Psychology*, *108*(4), 679.

Martoni, R.M., Salgari, G., Galimberti, E., Cavallini, M.C., & O'Neill, J. (2015). Effects of gender and executive function on visuospatial working memory in adult obsessive-compulsive disorder. *European archives of psychiatry and clinical neuroscience*, *265*(8), 707-718.

Mataix-Cols, D., Rauch, S. L., Manzo, P. A., Jenike, M. A., & Baer, L. (1999). Use of factor-analyzed symptom dimensions to predict outcome with serotonin reuptake inhibitors and placebo in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, 156(9), 1409-1416.

McGuire, J. F., Crawford, E. A., Park, J. M., Storch, E. A., Murphy, T. K., Larson, M. J., & Lewin, A. B. (2014). Neuropsychological performance across symptom dimensions in pediatric obsessive compulsive disorder. *Depression and anxiety*, *31*(12), 988-996.

McKay, D., Abramowitz, J. S., Calamari, J. E., Kyrios, M., Radomsky, A., Sookman, D., Taylor, S., & Wilhelm, S. (2004). A critical evaluation of obsessive–compulsive disorder subtypes: symptoms versus mechanisms. *Clinical psychology review*, *24*(3), 283-313.

Nakao, T., Nakagawa, A., Nakatani, E., Nabeyama, M., Sanematsu, H., Yoshiura, T., ... & Kanba, S. (2009). Working memory dysfunction in obsessive–compulsive disorder: a

neuropsychological and functional MRI study. *Journal of psychiatric research*, *43*(8), 784-791.

Nakao, T., Okada, K., & Kanba, S. (2014). Neurobiological model of obsessive–compulsive disorder: evidence from recent neuropsychological and neuroimaging findings. *Psychiatry and clinical neurosciences*, *68*(8), 587-605.

Nedeljkovic, M., Kyrios, M., Moulding, R., Doron, G., Wainwright, K., Pantelis, C., ... & Maruff, P. (2009). Differences in neuropsychological performance between subtypes of obsessive–compulsive disorder. *Australian and New Zealand Journal of Psychiatry*, *43*(3), 216-226.

Omori, I. M., Murata, Y., Yamanishi, T., Nakaaki, S., Akechi, T., Mikuni, M., & Furukawa, T. A. (2007). The differential impact of executive attention dysfunction on episodic memory in obsessive-compulsive disorder patients with checking symptoms vs. those with washing symptoms. *Journal of psychiatric research*, *41*(9), 776-784.

Pedron, A. C., Ferrão, Y. A., Gurgel, L. G., & Reppold, C. T. (2015). Relations Between Executive Functions and Different Symptomatic Dimensions in Obsessive Compulsive Disorder. *Paidéia (Ribeirão Preto)*,*25*(61), 229-239.

Penadés, R., Catalán, R., Andrés, S., Salamero, M., & Gastó, C. (2005). Executive function and nonverbal memory in obsessive-compulsive disorder. *Psychiatry research*, *133*(1), 81-90

Rasmussen, J., Siev, J., Abramovitch, A., & Wilhelm, S. (2016). Scrupulosity and contamination OCD are not associated with deficits in response inhibition. *Journal of behavior therapy and experimental psychiatry*,*50*, 120-126.

Rosario-Campos, M. C., Miguel, E. C., Quatrano, S., Chacon, P., Ferrao, Y., Findley, D., ... & Tolin, D. (2006). The Dimensional Yale–Brown Obsessive–Compulsive Scale (DY-BOCS): an instrument for assessing obsessive–compulsive symptom dimensions. *Molecular psychiatry*, *11*(5), 495.

Salloway, S. P., Malloy, P. F., & Duffy, J. D. (Eds.). (2008). *The frontal lobes and neuropsychiatric illness*. Washington, DC: American Psychiatric Pub.

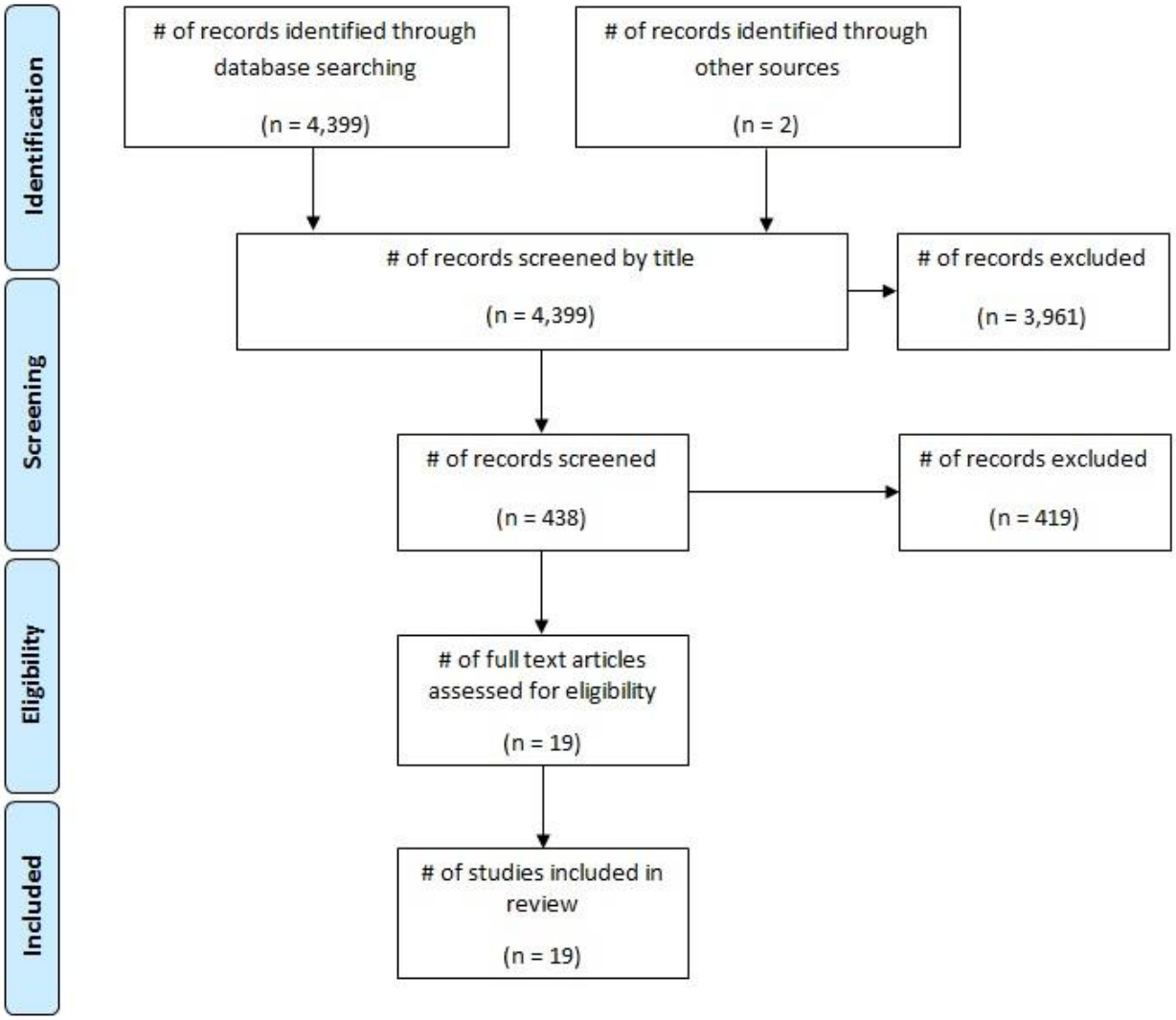
Shin, N. Y., Lee, T. Y., Kim, E., & Kwon, J. S. (2014). Cognitive functioning in obsessive-compulsive disorder: a meta-analysis. *Psychological medicine*, *44*(6), 1121-1130.

Waltz, A. (2005). Impairments in memory and reasoning in patients with neuropsychiatric illness: Disruptions of dynamic cognitive binding? In R.W. Engle, G. Sedek, U. Von Hecker, & D.N. McIntosh (Eds.), *Cognitive limitations in aging and psychopathology*. Cambridge: Cambridge University Press.

Appendix I

Tables & Figures

Figure 1
Strategy for Selection of Relevant Articles



From: Moher, D., Liberati, A., Tetziaff, J., & Altman, D.G., The PRISMA Group. (2009). *P*referred *R*eporting *I*tems for *S*ystematic Reviews and *M*eta-*A*nalyses: The PRISMA Statement. *PloS Med* 6(6): e1000097. doi: 10.1371/journal.pmed100009

Table 1 – Effect Sizes for Neuropsychological Deficits Organized by Study and Cognitive Domain

Cognitive Domain	Cognitive Test	No association	Effect Size		
			Small	Medium	Large
	Pedron et al., 2015				
	Go/No-Go: Commission Errors	2, 3, 4	-	1	-
	WASI: Matrix Reasoning	2, 3, 4	-	-	1
	WCST: # of Correct Responses	2, 3, 4	-	1	-
	WCST: "Learning to Learn"	2, 3, 4	-	-	1
	Martoni et al., 2015				
	CANTAB: Spatial Working Memory (strategy)	1, 2, 4, 11	-	-	3
	McGuire et al., 2014				
	D-KEFS: Design Fluency	1, 2, 4	-	3	-
	D-KEFS: Colour-Word Interference	1, 2, 4	3	-	-
	D-KEFS: Letter-Number Switching	1, 2, 4	3	-	-
	Dittrich et al., 2013				
	D-KEFS: Tower of Hanoi Movement Accuracy Ratio	1	-	-	2
	Hashimoto et al., 2011				
Executive Function	TMT: B-A Time	1 (P), 3 (P)	1* (Y), 2 (P)	2 (Y), 3 (Y)	-
	Stroop Task: Interference Score	1 (P), 1 (Y), 2 (P), 2 (Y), 3 (Y)	-	3 (P)	-
	Jang et al., 2010				
	RCFT: BQSS Organization Score	1, 3, 5	2	-	-
	Nakao et al., 2009				
	WCST: Milner-Type Errors	1	-	-	2
	Nedeljkovic et al., 2009				
	CANTAB: SOC (initial move latency)	1, 6, 13	-	2, 12	-
	CANTAB: SOC (subsequent move latency)	1, 2, 6, 13	12	-	-
	Omori et al., 2007				
	Stroop Task: Number of Errors	1	-	-	2
	Stroop Task: Completion Time	1	-	2	-
	TMT: B-A time	1	-	-	2
	GO/NO GO: Commission	1	-	2	-

	Errors				
	COWAT: Category Fluency	1	-	-	2
Processing Speed	*Pedron et al., 2015*				
	Stroop Task: Trial 2	2, 3, 4	-	-	1
	McGuire et al., 2014				
	D-KEFS: Number Sequencing Trials	1,2,4	-	3	-
	D-KEFS: Letter Sequencing Trials	1,2,4	-	3	-
	D-KEFS: Motor Speed Trials	1,2,4	-	3	-
	Hashimoto et al., 2011				
	WAIS-R: Digit Symbol	1 (Y), 2 (P), 2 (Y), 3 (P), 3 (Y)	-	1 (P)*	-
Selective Attention	Pedron et al., 2015				
	Go/No-Go: Omission Errors	2, 4	-	3	1
Visual Spatial Reconstruction	*Pedron et al., 2015*				
	RCFT: Direct Copy Trial Time	1, 2, 3, 4	-	-	2
	WASI: Block Design	2, 3, 4	-	-	1
Verbal Memory	*Hashimoto et al., 2011*				
	WMS-R: Logical Memory (immediate recall)	2 (P), 2 (Y)	-	1 (P)*, 3 (P)	1 (Y)*, 3 (Y)
	WMS-R: Logical Memory (30-min delay)	2 (P), 2 (Y)	-	1 (P)*, 3 (P)	1 (Y)*, 3 (Y)
	Nakao et al., 2009				
	WMS-R: Verbal Memory Score	1	-	-	2
	Ceschi et al., 2003				
	CVLT: Trial 1-5 Total	13	-	-	1, 2
	CVLT: Delayed Free Recall	13	-	-	1, 2
Non-Verbal Memory	*Pedron et al., 2015*				
	RCFT: Recall Time	1, 3, 4	-	-	2
	Martoni et al., 2015				
	CANTAB: Spatial Recognition Memory (% correct)	1, 2, 4, 11	-	-	3
	Jang et al., 2010				
	RCFT: BQSS Immediate Presence Score	1, 2, 5	-	3	-
	RCFT: BQSS Delayed Presence Score	1, 2, 5	-	3	-

	Nakao et al., 2009				
	WMS-R: Visual Memory Score	1	-	-	2
	Nedeljkovic et al., 2009				
	CANTAB: Pattern Recognition	1, 6, 12, 13	2	-	-
	CANTAB: Spatial Recognition	1, 2, 12, 13	6	-	-
	Cha et al., 2008				
	RCFT: Immediate Recall	1, 13	-	2	-
	RCFT: Delayed Recall	1, 13	-	-	2
Working Memory	*Nedeljkovic et al., 2009*				
	CANTAB: Spatial Working Memory (between search errors)	1, 6, 13	-	2, 12	-
	Hoenig et al., 2002				
	Negative Priming Task	10	-	-	2

Legend: BQSS = Boston Qualitative Scoring System; COWAT = Controlled Oral Word Association Test; CVLT = California Verbal Learning Test; D-KEFS = Delis-Kaplan Executive Function System; HVLT = Hopkins Verbal Learning Test; MARS = Maudsley Attention and Response Suppression battery; P = Padua Inventory; RCFT = Rey-Osterrieth Complex Figure Test; TMT = Trail-Making Test; WAIS-R = Wechsler Adult Intelligence Scale-Revised; WCST = Wisconsin Card Sorting Test; WASI = Wechsler Abbreviated Scale of Intelligence; WMS = Wechsler Memory Scale; WRAML = Wide Range Assessment of Memory and Learning; Y = Yale-Brown Obsessive Compulsive Scale

*****Denotes a result from a correlational study where correlation indicates positive performance on any neuropsychological measure. All other correlational results assumed to indicate poor performance.

Dimensions: 1 = Contamination/Cleaning; Washers; Washing; Cleanliness/Washing; Contamination/Washing; Cleaning

2 = Aggressive/Checking; Checkers; Checking; Obsessions/Checking; Doubting/Checking

3 = Symmetry/Ordering; Precision; Harm Avoidance/Mixed Symptoms (including Symmetry)

4 = Forbidden Thoughts; Scrupulous; Sexual/Religious

5 = Repeating/Counting

6 = Obsessionals/Few Compulsions

7 = Mental Checkers

8 = Slowness

9 = Doubting

10 = Non-Checkers

11 = Somatic

12 = Mixed Symptom Profile

13 = Healthy Controls

CHAPTER THREE

STUDY 2

TITLE: A comparison of cluster and factor analytic techniques for identifying symptom-based dimensions of obsessive-compulsive disorder

AUTHORS: Duncan H. Cameron, B.A.; David L. Streiner, Ph.D., C.Psych.; Laura J. Summerfeldt, Ph.D., C.Psych.; Karen Rowa, Ph.D., C.Psych.; Margaret C. McKinnon, Ph.D., C.Psych.; Randi E. McCabe, Ph.D., C.Psych.

CONTEXT AND IMPLICATIONS OF THIS STUDY: Study 2 of this thesis builds on the previous study by attempting to address the limitations observed in the methods employed for defining symptom subtypes of OCD across the studies included in the review. This study includes a brief review of all previously published literature describing statistical approaches to defining symptom subtypes, including factor analysis, cluster analysis and latent class analysis. This brief review serves to highlight common themes and procedural oversights present in a majority of previous research.

Having identified areas for improvement, I then performed a factor analysis following the best-accepted practices for this method, and also completed a cluster analysis in the same large, clinical sample, using the clinician-administered version of the Y-BOCS. Following the appropriate guidelines for factor analysis with the current data, it was observed that these data do not lend themselves well to this type of analysis. Furthermore, though cluster analysis has been identified by some researchers as a more appropriate method, the analysis does not adequately address the question at hand. This study acts as a recommendation to future research by

cautioning that this endeavour will likely not provide meaningful results for clinical or research applications.

ACKNOWLEDGEMENTS

None

CONFLICTS OF INTEREST: None.

PUBLISHED IN: Behaviour Research Methods, (under review).

Abstract

Background: A growing body of literature suggests that obsessive-compulsive disorder (OCD) is a heterogeneous condition. Given the wide range of possible symptom presentations, many attempts have been made to categorize distinct subtypes or dimensions using statistical methods of symptom/item reduction or classification. The studies addressing this topic have been limited by numerous methodological differences and sample characteristics, and there is some uncertainty in the literature as to which statistical approach is the most appropriate. The purpose of this study was to compare the two most commonly applied statistical techniques used in addressing this question in the same large cohort of individuals with OCD. *Methods:* Both cluster analysis and factor analysis were used to examine OCD symptom data as measured by the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) Symptom Checklist for 355 individuals with a primary diagnosis of OCD. *Results:* The factor analysis revealed a three-factor model best described as Symmetry obsessions/Ordering compulsions, Contamination obsessions/Cleaning compulsions, and Aggressive obsessions/Checking compulsions. In contrast, the cluster analysis yielded a stable four-cluster solution best described as Symmetry obsessions/Ordering compulsions, Contamination obsessions/Cleaning compulsions, Aggressive-Somatic-Religious obsessions/Checking compulsions and a Mixed symptom profile. *Discussion:* Although there was overlap in the models resulting from these two statistical approaches, cluster analysis better captured the dimensional nature of OCD by demonstrating the prevalence of symptom categories in each subgroup. Though both analyses are capable of providing similar outputs, the validity of these results is limited given the input of a priori symptom categories from the Y-BOCS.

Keywords: obsessive-compulsive disorder, subtypes, dimensions, factor analysis, cluster analysis

Introduction

A significant body of literature has developed over the past two decades investigating whether obsessive-compulsive disorder (OCD) can—or should—be classified into subgroups based on symptom presentation (McKay et al., 2004; Radomsky & Taylor, 2005; Starcevic & Brakoulias, 2008). The fact that several individuals could be provided the same diagnosis while presenting with vastly different symptoms has led many clinicians and researchers to accept that OCD is a heterogeneous disorder, but whether, and how, this heterogeneity can be defined in any meaningful way remains a debatable topic. Further, given that no concrete dimensions have been universally accepted, their utility for both clinical and research applications remains to be seen.

Despite these lingering issues, the increasingly large pool of research addressing the topic continues to provide some insight into possible dimensions of OCD. Existing studies that have attempted to define symptom dimensions are listed in Table 1, which shows the method of analysis and the dimensions found. As can be seen here, the large majority of studies used principal components analysis (PCA), exploratory factor analysis (EFA), or confirmatory factor analysis (CFA), while some also use cluster analysis. Latent class analysis (LCA) is becoming an increasingly popular method for defining latent subgroups within a larger population, and one study has attempted to do this in OCD. Table 1 also shows that these statistical analyses are applied to the most frequently used symptom measure for this population, the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989), which consists of a symptom checklist (with options for present, absent, or lifetime) and a symptom severity rating scale.

Researchers have attempted several methods of coding responses on the Y-BOCS symptom checklist (Y-BOCS-SC) to yield meaningful category scores which lend themselves to

classification or dimension reduction statistical analyses. The most ubiquitous method—originally proposed in Baer's (1994) seminal work in this area, modified slightly—has been to provide a score of 0 if any symptom within a category is rated as absent, 1 if a symptom is present, and 2 if that symptom is rated as present *and* also listed as one of the respondent's most troubling obsessions or compulsions in the Y-BOCS severity rating scale. These scores are provided for each of the 13 rationally derived categories of the Y-BOCS, which include aggressive, contamination, sexual, hoarding, religious, symmetry, and somatic obsessions, as well as cleaning, checking, repeating, counting, ordering, and hoarding compulsions. There are also two miscellaneous categories to capture any additional symptoms, although these are almost always left out of analyses given their heterogeneous nature (see Summerfeldt et al., 2004 for item-level confirmatory factor analysis including miscellaneous symptoms).

Two other scoring methods are commonly seen. One is to simply rate the symptom as present versus absent, as 1 or 0, respectively, though this method is criticized for not capturing the important symptoms which may be most dominant in a patient's presentation or for artificially inflating the importance of less dominant symptoms (e.g., Pinto et al., 2008). Another strategy is to generate a weighted score for each symptom category by dividing the number of total symptoms endorsed as present by the total number of symptoms in that category. This method is likely the most appropriate for confirmatory factor analysis techniques which might require summing these weighted scores into hypothesized factors (as in Summerfeldt et al., 2004). However, for exploratory factor analysis this method provides scores that are mostly a reflection of the number of items in each symptom category. For example, an individual might endorse 5/9 possible symptoms in the cleaning category, yielding a weighted score of .556, and might also list that symptom as their most troubling. But if that person also endorses the one

possible item in the counting category, this would yield a score of 1.0 (the highest possible) even if this symptom is only very rarely experienced. The 0-1-2 scoring system is not without its own pitfalls. In contrast to the weighted scores, this method reflects the severity of an individual's symptoms but only for those listed as currently most troubling, and the total number of symptoms is disregarded. Furthermore, the small number of studies examining temporal stability of obsessive-compulsive symptoms throughout the lifetime describe equivocal results (Mataix-Cols et al., 2002; Rufer, Grothusen, Maß, Peter, & Hand, 2005), and this, among other factors, would significantly contribute to the determination of the current most troubling symptoms. Doubtless there is no ideal method for reducing multi-item dichotomous data into a single statistic, but the 0, 1, 2 scoring method likely contains the fewest methodological issues.

The rightmost column of Table 1 displays the symptom dimensions found in each study. As can be seen, there is relatively consistent overlap in at least three groups. These most often consist of aggressive and/or uncertainty (sometimes including sexual and religious) obsessions and checking compulsions, contamination obsessions and cleaning compulsions, and symmetry obsessions with ordering/arranging compulsions. A fourth dimension, hoarding, is also commonly reported. However, given that the majority of these studies were published prior to the release of the Diagnostic and Statistical Manual, 5th Edition (DSM-5; American Psychological Association, 2013) it cannot be determined whether these symptoms described hoarding within the context of OCD or whether they constituted hoarding disorder criteria. Hoarding is also the only symptom on the Y-BOCS to have one item only in each of the obsessions and compulsions sections with the same label ("hoarding") for both, making it highly likely that these two items would be endorsed together, and consequently load together as a factor of their own in factor analysis. Other than these four domains, those remaining—if more

than three to four are reported—tend to have great variability in their labels and in the symptoms included. A decade has passed since the publication of a meta-analysis on the existing studies of the factor structure of OCD (Bloch, Landeros-Weisenberger, Pittenger, & Leckman, 2008). Here, a four factor structure was suggested with symmetry (including symmetry obsessions with counting, repeating and ordering compulsions), forbidden thoughts (including aggressive, religious, sexual and somatic obsessions with checking compulsions), contamination (with contamination obsessions and cleaning compulsions), and hoarding (with hoarding obsessions and compulsions only).

As evidenced by the number of studies listed in Table 1 (as well as the existence of a meta-analysis on the topic), many researchers have chosen to attempt to define symptom dimensions of OCD using factor analysis. Though factor analysis is by far the most commonly employed method for defining these dimensions, there is great variability in the type of factor analysis that can be carried out. PCA—which is not, technically speaking, factor analysis—is a common factor extraction method and provides the expression of each component (factor) as a linear combination of the input variables, and requires that the components be orthogonal. This technique attempts to create components that maximize inter-individual variance. This is, in a sense, contrary to the goal of attempting to define symptom dimensions of OCD. Principal axis factoring (PAF), conversely, is an exploratory factor analysis technique—and often referred to as "common factor analysis" or "exploratory factor analysis"—that attempts to define latent constructs, or underlying similarities that we cannot overtly observe, and it is assumed that the variables are linear combinations of these latent factors. In the context of OCD symptom dimensions, employing this method would imply that there is some hypothesized latent structure

in obsessive-compulsive symptom presentation that can be viewed by similarities in response patterns of individuals within each of these latent constructs.

In addition to factor extraction method, there are several options for rotation methods, which can generally be classified as being either oblique or orthogonal. Varimax is a common orthogonal rotation where the factors are kept at right angles (i.e., uncorrelated) to one another. On the other hand, promax is an oblique rotation that begins as an orthogonal varimax solution but relaxes the orthogonality such that the factors are not required to be uncorrelated. This method has the ability of providing a middle ground between methods because if there truly is no correlation between the factors, the rotation will still be relatively orthogonal.

The overview of the various methodological approaches above serves to highlight the incongruity between the type of factor analytic approach frequently used and the question being answered by this approach. Table 1 shows that almost all existing studies have used PCA with varimax rotation. It stands to reason, however, that obsessive-compulsive symptom dimensions—at least when expressed as items on the Y-BOCS—will exhibit a latent structure. Additionally, obsessive-compulsive symptoms might be expected to have some degree of correlation amongst their latent factors. Therefore, although many datasets will yield similar results following any combination of factor analytic techniques, principal axis factoring with promax rotation is likely the best reflection of reality for these data.

In addition to choice of extraction and rotation method, there are certain diagnostic audits which should be carried out prior to performing factor analysis of a dataset. Failure to follow these steps may have significant implications for the viability of the analysis and the validity of findings. Factor analysis begins with the production of a correlation matrix and, particularly in

SPSS, the values represented are Pearson's *r* correlations. However, Pearson's *r* is not an appropriate correlation method for the use of dichotomous data (Babakus, 1985), such as those in the Y-BOCS symptom checklist and frequently used statistical software (e.g., SPSS) is not capable of producing tetrachoric or polychoric correlations (although there is an available plugin to use the open source program R with SPSS; see Basto & Pereira, 2012). Though not always the case, there can be a vast difference between Pearson's *r* and polychoric correlations (Olsson, 1979).

After performing the appropriate correlations, there are several important values that should be inspected prior to moving forward with factor extraction: the Kaiser-Meyer-Olkin measures of sampling adequacy (MSA), both for the whole model and for each variable, and the communality values for each variable. The MSA is a measure of the proportion of variance among input variables that might be common variance, so a value close to zero means that there are large partial correlations compared to the sum of correlations which can be a problem for factor analysis. Communalities, however, are squared multiple correlations and provide a measure of how an item correlates with all other items. A stringent cut-off for individual variable MSA values is ≥ .70 while ≥ .50 is appropriate for communality values (Norman & Streiner, 2008). Kaiser (1970) suggests that any value >.60 for the overall model is "miserable" or "unacceptable" and any dataset providing this value, after removing individual variables with values below the cut-offs mentioned above, is likely not suitable for factor analysis. Only two studies of all those included in Table 1 report an MSA (Denys, de Geus, van Megen, & Westenberg, 2004a; KMO = .74; and Asadi et al., 2016; KMO = .808), and most do not report communalities. Finally, although statisticians debate the exact number, factor analysis is generally most valid with a participant-to-variable ratio of at least five-to-one, if not ten-to-one,

so studies with smaller samples should be interpreted with caution. In some cases, a large enough sample can help to address lower communalities although values ideally should still not fall below .50 (MacCallum, Widaman, Zhang & Hong, 1999). While these issues may not be problematic in all cases, they raise some concern about the interpretability of previous results from factor analytic investigations of the Y-BOCS.

Some of the methodological limitations of factor analysis when attempting to examine latent constructs of the Y-BOCS have led researchers to consider other approaches. Calamari, Wiegartz, & Janeck (1999) were the first to suggest that cluster analysis might be a more appropriate method because, although factor analysis is ideal for determining underlying structure in a dataset of *variables*, cluster analysis can be used to group *cases* to find smaller groups that are representative of data as a whole. Furthermore, factor analysis has no way of ensuring that one individual's responses are not partitioned across several factors, whereas cluster analysis can provide homogeneous groups. Cluster analysis, too, has many possible clustering techniques. The most prevalent of these are hierarchical cluster analysis, which organizes observations in a hierarchical manner based on cluster similarity (or dissimilarity); and *k*-means clustering which requires the input of a pre-specified number of clusters and attempts to fit observations to those clusters. Hasanpour and colleagues (2017) conducted a study comparing a multitude of clustering strategies with Y-BOCS data, and found that no one strategy stood out as significantly better than another for defining symptom clusters.

Given the consistent interest in the structure of OC symptoms for the past two decades and the aforementioned potential methodological challenges, the objectives of the present study were twofold. With data from a large clinical sample of individuals with OCD, this study aimed to perform 1) a factor analysis using best-practice strategies, that is, principal axis factoring with

promax rotation based on tetrachoric correlations, and compare results against previous findings, as well as 2) a cluster analysis, and investigate differences in the outcomes of each method, both with the aim of illustrating the differences inherent in the methods and thus to help to elucidate the most accurate choices for characterizing OCD symptom dimensions.

Methods

Participants

Data were collected from *N* = 355 participants aged 18-65 with a primary DSM-IV diagnosis of OCD who were referred for assessment and treatment to a large outpatient anxiety disorders clinic located in an academic community hospital. All participants received diagnostic assessment using the Structured Clinical Interview for Axis I Disorders for DSM-IV (SCID-IV; First, Spitzer, Gibbons & Williams, 1995). Those with a confirmed principal diagnosis of OCD then received the clinician-administered Y-BOCS and also completed a package of self-report questionnaires. The clinic received institutional ethics approval for an ongoing database for individuals assessed at the clinic. The data reported on in this study were collected between 2003 and 2010. Demographics for the sample are presented in Table 2. The mean age at the time of assessment was 33.5 (SD = 12.0) and the mean Y-BOCS severity score was 24.1 (SD = 5.2).

Measures

Yale-Brown Obsessive Compulsive Scale (Y-BOCS) (Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989). The Y-BOCS is a standardized scale with two parts. The first part is a 74-item checklist of symptoms covering seven rationally derived categories of obsessions and six categories of compulsions, with each also having a respective additional miscellaneous category. The 74-item

version also provides one open-ended question ("other" symptom) per category for both obsessions and compulsions. Most previous studies excluded the miscellaneous categories and the 10 open-ended questions to increase validity of results and reduce heterogeneity due to the large variability in potential answers. We also chose to exclude hoarding items from this analysis as these data were collected prior to the release of the DSM-5. The Y-BOCS part two is a standardized severity scale with 10 items pertaining to obsessions and compulsions on a 5-point adjectival scale ranging from 0 (no symptoms) to 4 (severe symptoms), and was only used for baseline characteristics in this sample. The clinician-administered version was used for this study in an attempt to increase validity of the data as the two versions may have only moderate convergence in some samples (Federici et al., 2010).

Procedure

Y-BOCS-SC responses were coded according to Mataix-Cols, Rauch, Manzo, Jenike, & Baer's (1999) adapted method from Baer (1994). As described above, a score of 0, 1 or 2 is assigned to each symptom category. A score of 2 was assigned to a category containing at least one item listed as present *and* as one of an individual's most upsetting obsessions or compulsions, 1 if the symptoms in that category were present only, and 0 if the category contained only absent symptoms. Lifetime symptoms were excluded from the present study to eliminate any potential recall bias.

Data Analyses

Factor analysis. Factor analysis was completed with the computer program RStudio, version 3.3.2. First, correlations were computed using the "polycor" package. The factor analysis was accompanied by a modified version of Glorfeld's parallel analysis and Velicer's minimum

average partials (MAP) test to determine the number of factors to extract (Glorfeld, 1995; O'Connor, 2000; Velicer 1976). Principal axis factoring followed by promax rotation was then performed on the polychoric correlation matrix; both analyses were performed using the "paramap" package.

Cluster analysis. Given the finding from Hansapour et al. (2017) that clustering methods are essentially comparable when attempting to cluster Y-BOCS data we chose to apply the method used in Calamari et al. (1999; 2004) of hierarchical cluster analysis using Ward's agglomerative procedure with squared Euclidean distance as the similarity measure. Calamari et al. (2004) followed their hierarchical cluster analysis with *k*-means clustering which, they noted, is sometimes used to address the limitations of hierarchical clustering alone (Borgen & Barnett, 1987; Milligan & Sokal, 1980) but reported that this did not result in any significant improvement in interpretability of the clusters. We, therefore, decided to perform hierarchical clustering only. Cluster analysis was completed using the "fpc" package for RStudio version 3.3.2.

Results

Factor analysis of the full eleven Y-BOCS categories revealed many communality and MSA values below the recommended thresholds. The sexual, religious and somatic obsessions, and repeating and counting compulsions categories were removed from the analysis, and the factor analysis was then performed with the remaining six variables. A stable three-factor structure was found explaining 83.2% of the variance, with an overall KMO of .707 and a significant Bartlett's test of Sphericity ($\chi^2 = 743.1$, $p < .001$). Results of the parallel analysis indicated that three factors should be retained, with eigenvalues of 1.29, 1.21, and 1.15 for

factors one through three, respectively. Similarly the MAP test indicated three factors for sample size of N = 364 and k = 11 variables, where eigenvalues must be 2.23, 1.27 and 1.13 for factors one through three, respectively, using the 95th percentile and 1000 replications. The factor loadings, with communalities and MSAs for each variable are found in Table 3. The first factor, symmetry obsessions and ordering compulsions, yielded an eigenvalue of 2.56 and accounted for 42.7% of the variance. Contamination obsessions and cleaning compulsions (eigenvalue = 1.23) accounted for 20.6% of the variance, while aggressive obsessions and checking compulsions (eigenvalue 1.20) accounted for a further 19.9%.

Cluster solutions for n = 2 to n = 11 clusters were evaluated and solutions beyond four did not improve the average silhouette or cluster interpretability. An average silhouette value of 0.30 ("fair") for the four cluster model was the highest achieved for any solution. Figure 1 shows the mean values for each Y-BOCS symptom category in each of the four clusters. Cluster one, aggressive obsessions and checking compulsions, accounted for 34.2% of the sample. Clusters two and three, contamination/cleaning, and symmetry/ordering, accounted for 23.2% of the sample each. A fourth cluster representing mixed symptoms across almost all categories accounted for the final 19.5% of individuals.

Discussion

The purpose of this study was to employ the current best practices in factor analysis with ordinal data on responses to the Y-BOCS-SC, and compare the results of this analysis with the output from cluster analysis. Factor analysis using principal axis factoring and promax rotation resulted in three factors including symmetry/ordering, contamination/cleaning and aggressive/checking symptoms. These three factors are consistent with the majority of previous

factor analytic studies attempting to define symptom dimensions of OCD using the Y-BOCS symptom checklist. Where this study differs from previous approaches is in the inclusion of the remaining Y-BOCS symptom categories, which all exhibited very low communality values. Because communalities represent the squared multiple correlations, the low values for these variables reflects the fact that these symptom categories are not strongly related to each other and are poorly represented in the final factor solution. As can be seen in Table 3, the communality and MSA values for the retained variables are barely within the recommended range for factor analysis. This finding is arguably more important than the resulting factor solution. What we have learned from a rigorous investigation of factor analysis of the Y-BOCS symptom checklist is that the data, at least, when coded in such a way that multi-item data are reduced to a single, ordinal metric, likely do not lend themselves well to this type of analysis, casting doubt on the interpretability of previous findings.

Table 4 shows the frequency of symptoms endorsed in our sample, which provides some insight into the nature of these results. It can easily be seen that there were considerably more individuals ranking symptoms of aggressive/checking, contamination/cleaning, and symmetry/ordering categories as present and/or most troubling compared to all other symptom categories, which reflects the nature of the presentation of OCD. These limitations, however, do not necessarily stop computerized statistical software from continuing with the analysis and defining what seem to be meaningful factors.

To demonstrate this, results of a factor analysis performed without excluding variables with low communalities or MSA values, and without Velicer's MAP test and parallel analysis indicating the recommended number of factors is presented in Table 5. The resulting four factor model presented here shows all symptom checklist categories, except counting compulsions,

loading significantly onto at least one factor with a value of > .30. The factors representing dimensions with symmetry/ordering/repeating, contamination/cleaning, aggressive/checking, and sexual/religious/somatic/repeating symptoms appears similar to those presented in previous studies, when excluding hoarding symptoms (e.g., Cavallini, Di Bella, Siliprandi, Malchiodi, & Bellodi, 2002; Kim, Lee, & Kim, 2005; Mataix-Cols et al., 1999; Pinto et al., 2007).

Cluster analysis, on the other hand, may offer a slightly different insight into symptom structure, at least when using Y-BOCS-SC responses as input variables. The low representations of less frequently endorsed Y-BOCS items can still be observed by the dominance of the aggressive/checking, contamination/cleaning and symmetry/ordering categories in the largest three clusters, as well as by the relatively low mean scores for all other categories within each cluster. Not only is this affected by certain symptoms being less frequently endorsed overall, but further by the fact that even when these symptoms are endorsed, they are very rarely listed as a respondent's most troubling symptom (see Table 4) . However, the results yielded by this method differ from factor analysis in the fourth cluster, which appears to represent individuals who tend to express symptoms from a variety of different obsessive-compulsive categories with no single type of symptom being more prevalent than the next. This is a similar finding to previous cluster analytic studies, particularly those of Calamari et al. (1999; 2004) who defined "obsessional" and "certainty" subgroups which displayed similar trends to the fourth, mixed cluster described here.

These results should inform future researchers on several levels. First, it is apparent that factor analysis might not be capable of defining symptom dimensions of OCD based on responses recorded on the Y-BOCS symptom checklist. It appears superficially as though cluster analysis, then, might be the more appropriate method for this research question. However, given

that the goal is to define taxonomy, a method which seeks to group cases rather than symptoms does not adequately address the question. If researchers choose to follow this route, an alternative to cluster analysis, Gaussian mixture modelling, holds the benefit of providing a maximum likelihood model of the data. If factor analysis is chosen, researchers should take care to report communality and MSA values for individual variables and assure adequate sample size to maximize validity and interpretability of results. Furthermore, this approach is predicated upon the assumption that factor or cluster solutions of the Y-BOCS are equal to structure of OCD. It is probable that the Y-BOCS itself, despite being the most comprehensive symptom checklist available for OCD, cannot provide the data necessary for this task.

Many additional considerations plague the attempts to define symptom dimensions in OCD, such as whether they should be defined as dimensions or categories. Powerful statistical analyses that might be able to help to provide meaningful dimensions are becoming increasingly popular, some of which include latent class analysis, factor mixture modeling, multidimensional scaling, and—possibly the most promising for this endeavour—machine learning. However, the meaningfulness of performing these analyses on the Y-BOCS symptom checklist will almost certainly be limited, as it similarly has been with factor and cluster analysis, due to the nature of the measure itself, and the typical presentation of less-commonly endorsed obsessive-compulsive symptoms in naturalistic populations.

For those wishing to assess symptom severity within dimensions of OCD in clinical or research settings, the Dimensional Obsessive-Compulsive Scale (DOCS; Abramowitz et al., 2010) was developed with empirically supported, rather than rationally derived symptom categories. Even if the statistical techniques described in this paper were without flaw, any results of an attempt to define symptom dimensions using the Y-BOCS could be rebutted given

that this method relies on a priori symptom categories. This is exemplified by the item-level investigations which show that, while close to the results of the categorical approaches, several items do not match their a priori category designation. For example, previous investigations find that when analyzed at the item-level, fear of aggressive impulse items are placed in a dimension comprising "taboo thoughts" while items of excessive responsibility for harm load onto a "doubting/checking" dimension (Denys, de Geus, van Megen, & Westenberg, 2004b; Pinto et al., 2008; Summerfeldt, Kloosterman, Antony, Richter, & Swinson, 2004), whereas these items are lumped into a combined aggressive/sexual/religious/somatic/checking dimension when using a categorical approach, as described in Table 5. It is recommended that researchers also collect symptom level data wherever possible as this will help inform future investigations of this nature. However, future attempts to draw meaningful symptom dimensions of OCD using the Y-BOCS symptom checklist should seek to use item-level analysis only, or they risk losing potentially critical detail to rationally derived but not empirically driven categories.

References

Abramowitz, J. S., Deacon, B. J., Olatunji, B. O., Wheaton, M. G., Berman, N. C., Losardo, D., ... & Björgvinsson, T. (2010). Assessment of obsessive-compulsive symptom dimensions: Development and evaluation of the Dimensional Obsessive-Compulsive Scale. *Psychological assessment*, *22*(1), 180.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.

Asadi, S., Daraeian, A., Rahmani, B., Kargari, A., Ahmadiani, A., & Shams, J. (2016). Exploring yale-brown obsessive-compulsive scale symptom structure in Iranian OCD patients using item-based factor analysis. *Psychiatry research*, *245*, 416-422.

Babakus, E. (1985). *The sensitivity of maximum likelihood factor analysis given violations of interval scale and multivariate normality*, unpublished PhD dissertation, The University of Alabama.

Baer, L. (1994). Factor analysis of symptom subtypes of obsessive compulsive disorder and their relation to personality and tic disorders. *The Journal of clinical psychiatry*.

Basto, M., & Pereira, J. M. (2012). An SPSS R-menu for ordinal factor analysis. *Journal of statistical software*, *46*(4), 1-29.

Bernstein, G. A., Victor, A. M., Nelson, P. M., & Lee, S. S. (2013). Pediatric obsessive–compulsive disorder: Symptom patterns and confirmatory factor analysis. *Journal of Obsessive-Compulsive and Related Disorders*, *2*(3), 299-305.

Bloch, M. H., Landeros-Weisenberger, A., Rosario, M. C., Pittenger, C., & Leckman, J. F. (2008). Meta-analysis of the symptom structure of obsessive-compulsive disorder. *American Journal of Psychiatry*, *165*(12), 1532-1542.

Borgen, F. H., & Barnett, D. C. (1987). Applying cluster analysis in counseling psychology research. *Journal of Counseling Psychology*, *34*(4), 456.

Calamari, J. E., Wiegartz, P. S., & Janeck, A. S. (1999). Obsessive–compulsive disorder subgroups: a symptom-based clustering approach. *Behaviour Research and Therapy*, *37*(2), 113-125.

Calamari, J. E., Wiegartz, P. S., Riemann, B. C., Cohen, R. J., Greer, A., Jacobi, D. M., ... & Carmin, C. (2004). Obsessive–compulsive disorder subtypes: an attempted replication and extension of a symptom-based taxonomy. *Behaviour Research and Therapy*, *42*(6), 647-670.

Cavallini, M. C., Di Bella, D., Siliprandi, F., Malchiodi, F., & Bellodi, L. (2002). Exploratory factor analysis of obsessive-compulsive patients and association with 5-HTTLPR polymorphism. *American Journal of Medical Genetics Part A*, *114*(3), 347-353.

Cullen, B., Brown, C. H., Riddle, M. A., Grados, M., Bienvenu, O. J., Hoehn-Saric, R., ... & Nestadt, G. (2007). Factor analysis of the Yale–Brown Obsessive Compulsive Scale in a family study of obsessive–compulsive disorder. *Depression and Anxiety*, *24*(2), 130-138.

Delorme, R., Bille, A., Betancur, C., Mathieu, F., Chabane, N., Mouren-Simeoni, M. C., & Leboyer, M. (2006). Exploratory analysis of obsessive compulsive symptom dimensions in children and adolescents: a prospective follow-up study. *BMC psychiatry*, *6*(1), 1.

Denys, D., de Geus, F., van Megen, H. J., & Westenberg, H. G. (2004a). Use of factor analysis to detect potential phenotypes in obsessive-compulsive disorder. *Psychiatry research*, *128*(3), 273-280.

Denys, D., de Geus, F., van Megen, H. J., & Westenberg, H. G. (2004). Symptom dimensions in obsessive-compulsive disorder: factor analysis on a clinician-rated scale and a self-report measure. *Psychopathology*, *37*(4), 181-189.

Federici, A., Summerfeldt, L. J., Harrington, J. L., McCabe, R. E., Purdon, C. L., Rowa, K., & Antony, M. M. (2010). Consistency between self-report and clinician-administered versions of the Yale-Brown Obsessive–Compulsive Scale. *Journal of Anxiety Disorders*, *24*(7), 729-733.

Feinstein, S. B., Fallon, B. A., Petkova, E., & Liebowitz, M. R. (2003). Item-by-item factor analysis of the Yale-Brown obsessive compulsive scale symptom checklist. *The Journal of neuropsychiatry and clinical neurosciences*, *15*(2), 187-193.

First, M. B., Spitzer, R. L., Gibbon, M. W. J. B., & Williams, J. B. (1995). Structured clinical interview for DSM-IV axis I disorders. *New York: New York State Psychiatric Institute*.

Glorfeld, L. W. (1995). An improvement on Horn's parallel analysis methodology for selecting the correct number of factors to retain. *Educational and psychological measurement*, *55*(3), 377-393.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Delgado, P., Heninger, G. R., & Charney, D. S. (1989). The Y-BOCS, II: validity. *Archives of General Psychiatry*, *46*, 1012-1019.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., ... & Charney, D. S. (1989). Yale-brown obsessive compulsive scale (Y-BOCS). *Arch gen psychiatry*, *46*, 1006-1011.

Hasanpour, H., Asadi, S., Meibodi, R. G., Daraeian, A., Ahmadiani, A., Shams, J., & Navi, K. (2017). A critical appraisal of heterogeneity in Obsessive-Compulsive Disorder using symptom-based clustering analysis. *Asian journal of psychiatry*, *28*, 89-96.

Hasler, G., Pinto, A., Greenberg, B. D., Samuels, J., Fyer, A. J., Pauls, D., ... & Rauch, S. L. (2007). Familiality of factor analysis-derived YBOCS dimensions in OCD-affected sibling pairs from the OCD Collaborative Genetics Study. *Biological psychiatry*, *61*(5), 617-625.

Katerberg, H., Delucchi, K. L., Stewart, S. E., Lochner, C., Denys, D. A., Stack, D. E., ... & den Boer, J. A. (2010). Symptom dimensions in OCD: item-level factor analysis and heritability estimates. *Behavior genetics*, *40*(4), 505-517.

Kim, S. J., Lee, H. S., & Kim, C. H. (2005). Obsessive-compulsive disorder, factor-analyzed symptom dimensions and serotonin transporter polymorphism. *Neuropsychobiology*, *52*(4), 176-182.

Leckman, J. F., Grice, D. E., Boardman, J., Zhang, H., Vitale, A., Bondi, C., ... & Goodman, W. K. (1997). Symptoms of obsessive-compulsive disorder. *American Journal of Psychiatry*, *154*(7), 911-917.

Lochner, C., Hemmings, S. M., Kinnear, C. J., Nel, D., Seedat, S., Moolman-Smook, J. C., & Stein, D. J. (2008). Cluster analysis of obsessive-compulsive symptomatology: identifying obsessive-compulsive disorder subtypes. *Israel Journal of Psychiatry and Related Sciences*, *45*(3), 164.

MacCallum, R. C., Widaman, K. F., Zhang, S., & Hong, S. (1999). Sample size in factor analysis. *Psychological methods*, *4*(1), 84.

Mataix-Cols, D., Marks, I. M., Greist, J. H., Kobak, K. A., & Baer, L. (2002). Obsessive-compulsive symptom dimensions as predictors of compliance with and response to behaviour therapy: results from a controlled trial. *Psychotherapy and psychosomatics*, *71*(5), 255-262.

Mataix-Cols, D., Nakatani, E., Micali, N., & Heyman, I. (2008). Structure of obsessive-compulsive symptoms in pediatric OCD. *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(7), 773-778.

Mataix-Cols, D., Rauch, S. L., Manzo, P. A., Jenike, M. A., & Baer, L. (1999). Use of factor-analyzed symptom dimensions to predict outcome with serotonin reuptake inhibitors and placebo in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *156*(9), 1409-1416.

Matsunaga, H., Maebayashi, K., Hayashida, K., Okino, K., Matsui, T., Iketani, T., ... & Stein, D. J. (2008). Symptom structure in Japanese patients with obsessive-compulsive disorder. *American Journal of Psychiatry*, *165*(2), 251-253.

McKay, D., Abramowitz, J. S., Calamari, J. E., Kyrios, M., Radomsky, A., Sookman, D., ... & Wilhelm, S. (2004). A critical evaluation of obsessive–compulsive disorder subtypes: symptoms versus mechanisms. *Clinical psychology review*, *24*(3), 283-313.

McKay, D., Piacentini, J., Greisberg, S., Graae, F., Jaffer, M., & Miller, J. (2006). The structure of childhood obsessions and compulsions: dimensions in an outpatient sample. *Behaviour Research and Therapy*, *44*(1), 137-146.

Milligan, G.W., & Sokal, L.M. (1980). A two-stage clustering algorithm with robust recovery characteristics. *Educational and Psychological Measurement, 40*, 755-759.

Norman, G., & Streiner, D. (2008). Principal components and Factor Analysis. In *Biostatistics: The bare essentials* (208-225). Hamilton, ON: BC Decker Inc.

O'connor, B. P. (2000). SPSS and SAS programs for determining the number of components using parallel analysis and Velicer's MAP test. *Behavior research methods, instruments, & computers*, *32*(3), 396-402.

Olsson, U. (1979). Maximum likelihood estimation of the polychoric correlation coefficient. *Psychometrika*, *44*(4), 443-460.

Pinto, A., Eisen, J. L., Mancebo, M. C., Greenberg, B. D., Stout, R. L., & Rasmussen, S. A. (2007). Taboo thoughts and doubt/checking: a refinement of the factor structure for obsessive–compulsive disorder symptoms. *Psychiatry research*, *151*(3), 255-258.

Pinto, A., Greenberg, B. D., Grados, M. A., Bienvenu, O. J., Samuels, J. F., Murphy, D. L., ... & Pauls, D. L. (2008). Further development of YBOCS dimensions in the OCD Collaborative Genetics study: symptoms vs. categories. *Psychiatry research*, *160*(1), 83-93.

Radomsky, A. S., & Taylor, S. (2005). Subtyping OCD: Prospects and problems. *Behavior Therapy*, *36*(4), 371-379.

Rufer, M., Grothusen, A., Maß, R., Peter, H., & Hand, I. (2005). Temporal stability of symptom dimensions in adult patients with obsessive–compulsive disorder. *Journal of affective disorders*, *88*(1), 99-102.

Starcevic, V., & Brakoulias, V. (2008). Symptom subtypes of obsessive–compulsive disorder: are they relevant for treatment?. *Australian & New Zealand Journal of Psychiatry*, *42*(8), 651-661.

Stewart, S. E., Rosario, M. C., Baer, L., Carter, A. S., Brown, T. A., Scharf, J. M., ... & Rasmussen, S. (2008). Four-factor structure of obsessive-compulsive disorder symptoms in children, adolescents, and adults. *Journal of the American Academy of Child & Adolescent Psychiatry*, *47*(7), 763-772.

Stewart, S. E., Rosario, M. C., Brown, T. A., Carter, A. S., Leckman, J. F., Sukhodolsky, D., ... & Pauls, D. L. (2007). Principal components analysis of obsessive–compulsive disorder symptoms in children and adolescents. *Biological psychiatry*, *61*(3), 285-291.

Summerfeldt, L. J., Kloosterman, P. H., Antony, M. M., Richter, M. A., & Swinson, R. P. (2004). The relationship between miscellaneous symptoms and major symptom factors in obsessive-compulsive disorder. *Behaviour Research and Therapy*, *42*(12), 1453-1467.

Summerfeldt, L. J., Richter, M. A., Antony, M. M., & Swinson, R. P. (1999). Symptom structure in obsessive-compulsive disorder: a confirmatory factor-analytic study. *Behaviour Research and Therapy*, *37*(4), 297-311.

Tek, C., & Ulug, B. (2001). Religiosity and religious obsessions in obsessive–compulsive disorder. *Psychiatry Research*, *104*(2), 99-108.

Velicer, W.F. (1976). Determining the number of components from the matrix of partial correlations, *Psychometrika*, *41*, 321-327.

Appendix I

Tables & Figures

Table 1

Summary of past approaches to defining symptom dimensions in OCD

Author, Year	*Population (child/ adult)*	*Symptoms*	*Scoring Method*	*Analysis Type (item level or category)*	*Analysis Method*	*Factors/Clusters*
Baer, 1994	Adult	Current	0,1,2	Category	PCA Varimax	Factor 1: Hoarding obsessions and compulsions, repeating, ordering, counting (20.7%) Factor 2: Contamination, cleaning, somatic, checking (16.0%) Factor 3: Aggression, sexual, religious (11.3%)
Hantouche & Lancrenon 1996	Adult	Current	0,1	Both	PCA Varimax	Factor 1: Hoarding obsessions and compulsions, symmetry, repeating, ordering, and checking Factor 2: Aggressive, sexual, religious, miscellaneous obsessions and compulsions Factor 3: Cleaning, contamination, and somatic obsessions
Leckman et al., 1997	Adult	Lifetime	Sum total symptoms per category	Category	PCA Varimax	Factor 1: Aggression, sexual, religious, somatic, checking (30.1%) Factor 2: Symmetry, ordering, repeating, counting (13.8%) Factor 3: Contamination, cleaning (10.2%) Factor 4: Hoarding obsessions and compulsions (8.5%)
Mataix-Cols et al., 1999	Adult	Current	0,1,2	Category	PCA Varimax	Factor 1: Symmetry, ordering, repeating, counting (19.0%) Factor 2: Hoarding obsessions and compulsions (13.8%)

						Factor 3: Contamination, cleaning (12.7%) Factor 4: Aggressive, checking (10.4%) Factor 5: Sexual, religious (9.7%)
Tek & Ulug, 2001	Adult	Current	0,1	Category	PCA Varimax	Factor 1: Contamination, cleaning, repeating (17.8%) Factor 2: Symmetry, somatic, ordering (15.2%) Factor 3: Aggressive, counting (13.7%) Factor 4: Sexual, religious (9.7%) Factor 5: Hoarding compulsions and checking (9.2%)
Mataix-Cols et al., 2002	Adult	Current	0,1,2	Category	PCA Varimax	Factor 1: Aggressive, religious, checking, repeating, and counting (23.3%) Factor 2: Contamination, cleaning (13.3%) Factor 3: Symmetry, ordering, counting (10.9%) Factor 4: Hoarding obsessions and compulsions (8.3%) Factor 5: Sexual, somatic (7.9%)
Cavallini et al., 2002	Adult	Lifetime	0,1	Category	PCA Varimax	Factor 1: Contamination, cleaning (17.0%) Factor 2: Hoarding obsessions and compulsions (13.0%) Factor 3: Aggressive, sexual, somatic, religious, checking, repeating (11.5%) Factor 4: Symmetry, ordering, religious (9.5%) Factor 5: Repeating, counting, symmetry (8.8%)
Feinstein et al., 2003	Adult	Current	0,1	Both	PCA Varimax	Factor 1: Symmetry, ordering, repeating, counting (14.2%) Factor 2: Contamination, cleaning, aggressive, checking (14.2%) Factor 3: Hoarding obsessions and compulsions (13.9%) Factor 4: Sexual, religious (11.8%)
Denys et al., 2004a	Adult	Current	0,1,2	Item	PCA Varimax	Factor 1: Contamination, cleaning (16.4%) Factor 2: Aggressive, sexual, religious (9.8%)

						Factor 3: Somatic and checking (5.9%) Factor 4: Symmetry, repeating, ordering, counting, hoarding obsessions and compulsions (5.6%) Factor 5: High-risk and checking (4.0%)
Denys et al., 2004b	Adult	Current	0,1,2	Item	PCA Varimax	Factor 1: Aggressive, sexual, religious (14.5%) Factor 2: Contamination, cleaning, washing (11%) Factor 3: Somatic, checking (6.3%) Factor 4: Symmetry, exactness, arranging, ordering (5.8%) Factor 5: High risk assessment and checking (4.8%)
Kim et al., 2005	Adult	Current	0,1,2	Category	PCA Varimax	Factor 1: Hoarding obsessions and compulsions, repeating, ordering, counting (34.0%) Factor 2: Contamination, cleaning (11.1%) Factor 3: Aggression, sexual (10.0%) Factor 4: Religious, somatic (7.7%)
Delorme et al., 2006	Child	Current	Total symptoms per category	Category	PCA Varimax	Factor 1: Symmetry, checking, repeating, ordering (35.2%) Factor 2: Aggressive, sexual, somatic, counting (12.7%) Factor 3: Contamination, religious, cleaning (11%) Factor 4: Hoarding obsessions and compulsions (8.1%)
McKay et al., 2006	Child	Current	Symptom number	Category	PCA Oblimin	Factor 1: Cleaning, checking, repeating, counting, ordering, superstitious behaviours, rituals involving others (17.3%) Factor 2: Aggressive, sexual, magical thinking (15.9%) Factor 3: Contamination, aggressive, sexual, magical thoughts, somatic, religious, repeating, counting, symmetry, rituals involving others (12.7%)

						Factor 4: Hoarding somatic, counting, ordering (11.9%)
Pinto et al., 2007	Adult	Current	Number endorsed divided by total number in category	Category	PCA Varimax	Factor 1: Symmetry, ordering, repeating, counting (22.5%) Factor 2: Hoarding obsessions and compulsions (13.3%) Factor 3: Pathological doubt, somatic, checking (12.2%) Factor 4: Contamination and cleaning (10.3%) Factor 5: Aggressive, sexual, religious (7.4%)
Cullen et al., 2007	Adult	Lifetime	0,1	Category	Dichotomous EFA Oblique	Factor 1: Aggressive, sexual, religious, somatic Factor 2: Cleaning, contamination Factor 3: Symmetry obsessions, repeating, ordering, counting, sensory/ motor compulsions Factor 4: Hoarding obsessions and compulsions
Hasler et al., 2007	Adult	Lifetime	0,1	Category	PCA Promax	Factor 1: Aggressive, sexual, religious, somatic, and checking (17.7%) Factor 2: Symmetry, repeating, counting, ordering (15.4%) Factor 3: Cleaning, contamination (15.4%) Factor 4: Hoarding obsessions and compulsions (14.2%)
Stewart et al., 2007	Child	Lifetime	Total symptoms per category	Category	PCA Promax	Factor 1: Symmetry, ordering, repeating, checking (27.0%) Factor 2: Contamination, cleaning, aggressive, somatic (14.4%) Factor 3: Hoarding obsessions and compulsions (11.1%) Factor 4: Religious, sexual (9.1%)
Matsunaga et al., 2008	Adult	Lifetime	0,1,2	Category	PCA Varimax	Factor 1: Cleaning, contamination (21.2%) Factor 2: Hoarding obsessions and compulsions (14.3%) Factor 3: Symmetry, repeating, ordering (11.9%)

						Factor 4: Aggression, checking (10.3%)
Mataix-Cols et al., 2008	Child	Lifetime	Total symptoms per category	Category	PCA Varimax	Factor 1: Hoarding obsessions and compulsions, checking (14.1%) Factor 2: Aggressive, sexual, religious (13.7%) Factor 3: Contamination, cleaning, somatic (13.6%) Factor 4: Symmetry, ordering, repeating, checking (13.1%)
Pinto et al., 2008	Adult	Lifetime	0,1	Item	Dichotomous Factor Analysis Varimax	Factor 1: Taboo Thoughts (22.4%) Factor 2: Symmetry, ordering (11.6%) Factor 3: Hoarding (6.9%) Factor 4: Contamination, cleaning (6.6%) Factor 5: Doubt, checking (4.9%)
Katerberg et al., 2010	Adult	Lifetime	Number endorsed divided by total number in category	Both	PCA Promax	Factor 1: Symmetry, ordering, counting, repeating Factor 2: Aggressive, sexual, religious, checking Factor 3: Contamination, cleaning Factor 4: Hoarding
Asadi et al., 2016	Adult	Current	0,1 and severity scores	Item	Maximum Likelihood Varimax	Factor 1: Aggression, checking (19.5%) Factor 2: Contamination, cleaning (5.8%) Factor 3: Symmetry, ordering, counting, repeating, hoarding (3.5%) Factor 4: Sexual (2.7%) Factor 5: Somatic (2.7%)
				Confirmatory Factor Analysis		
Summerfeldt et al., 1999	Adult	Current	0,1	Category	CFA Maximum Likelihood	Factor 1: Aggressive, sexual, religious, somatic, checking Factor 2: Symmetry, repeating, counting, ordering Factor 3: Contamination, cleaning, washing Factor 4: Hoarding, saving, collecting
Summerfeldt et al., 2004	Adult	Current	Number endorsed divided by	Both	Logistic Regression/CFA	Factor 1: Obsessions and checking Factor 2: Symmetry Factor 3: Contamination and cleaning Factor 4: Hoarding

			number in category; weighted scores summed into CFA categories			
Stewart et al., 2008	Adult/child	Current	Total symptoms per category	Category	CFA Maximum Likelihood	Factor 1: Aggressive, sexual, religious, somatic, checking Factor 2: Symmetry, ordering, counting, repeating Factor 3: Contamination, cleaning Factor 4: Hoarding
Katerberg et al., 2010	Adult	Lifetime	Number endorsed divided by total number in category	Item	CFA Weighted Least Squares	Factor 1: Taboo (sexual, aggressive, religious) Factor 2: Contamination, cleaning Factor 3: doubts (obsessions related to fear, compulsions related to these fears) Factor 4: rituals/superstition (superstitions obsessions, eating and mental rituals) Factor 5: hoarding/symmetry (hoarding, symmetry, ordering, arranging, fear of losing things)
Bernstein et al., 2013	Child	Current	Total symptoms per category	Item	CFA Maximum Likelihood	Factor 1: Contamination, somatic, cleaning Factor 2: Magical, checking, repeating, counting, ordering Factor 3: Hoarding Factor 4: Aggressive, sexual, religious
				Cluster Analysis		
Calamari et al., 1999	Adult	Current	0,1,2	Items	Ward's Method	Cluster 1: Harming Cluster 2: Hoarding Cluster 3: Contamination Cluster 4: Certainty Cluster 5: Obsessionals
Calamari et al., 2004	Adult	Current	0,1,2	Items	Ward's Method and K-means	Cluster 1: Contamination Cluster 2: Harming Cluster 3: Obsessionals

						Cluster 4: Certainty Cluster 5: Contamination/Harming Cluster 6: Symmetry Cluster 7: Low symptoms
Lochner et al., 2008	Adult	Current	0,1	Items	CA Ward's Method	Cluster 1: Contamination, washing Cluster 2: Hoarding, collecting Cluster 3: symmetry, order, arranging, repetitive rituals, counting, checking Cluster 4: Sexual Cluster 5: Somatic, religious, diverse Cluster 6: Aggressive, harm-related
Hasanpour et al., 2017	Adult	Current	Symptom Checklist and Severity	Both	Comparison of 5 CA techniques	Cluster 1: Higher symptom severity Cluster 2: Lower symptom severity Significant clusters did not differ as a result of symptom presentation, but rather by symptom severity only
				Latent Class Analysis		
Delucchi et al., 2011	Adult	Lifetime	0,1 and Severity	Items	LCA	Classes did not differ in terms of symptom presentation but by level of symptom endorsement.

Legend: CA = cluster analysis; CFA = confirmatory factor analysis; EFA = exploratory factor analysis; LCA = latent class analysis; PCA = principal components analysis

Table 2

Sample demographics

Variable	*Frequency*
Age, mean (SD)	33.5 (12.0)
Sex (% female)	55.7 %
Age of OCD Onset, mean (SD)	17.8 (9.7)
Comorbidity (≥ 1 secondary Axis I diagnosis)	77.9%
Y-BOCS Severity Score	24.1 (5.2)
Mean number of current symptoms endorsed	12.5 (6.6)

Note: Sample size *N* = 355

Table 3

Factor loadings for principal axis factoring with promax rotation

Variable	*Factor 1*	*Factor 2*	*Factor 3*	*Communality*	*MSA*
Aggressive Obsessions	-	-	**.734**	.547	.664
Contamination Obsessions	-	**.814**	-	.672	.697
Symmetry Obsessions	**.826**	-	-	.684	.693
Cleaning Compulsions	-	**.869**	-	.764	.708
Checking Compulsions	-	-	**.709**	.522	.773
Ordering Compulsions	**.896**	-	-	.803	.692

Legend: MSA = Measures of sampling adequacy for individual variables.

Table 4

Representation of Y-BOCS symptoms as primary or secondary

	Frequency (%)		
Symptom Domain	*0*	*1*	*2*
Aggressive	14.6	45.4	40.0
Checking	11.0	47.3	41.7
Contamination	23.7	42.8	33.5
Cleaning	21.7	43.9	34.4
Symmetry	34.6	48.2	17.2
Ordering	37.5	46.8	15.8
Sexual	69.6	28.2	2.3
Religious	64.2	31.8	3.9
Somatic	76.6	18.9	4.5
Repeating	38.3	54.1	7.6
Counting	65.6	33.2	1.1

Note – 0 = symptom absent; 1 = symptom present; 2 = symptom present and listed as one of currently most troubling.

Table 5

Factor loadings for principal axis factoring with varimax rotation without removing variables due to low communality or MSA, no MAP test or parallel analysis

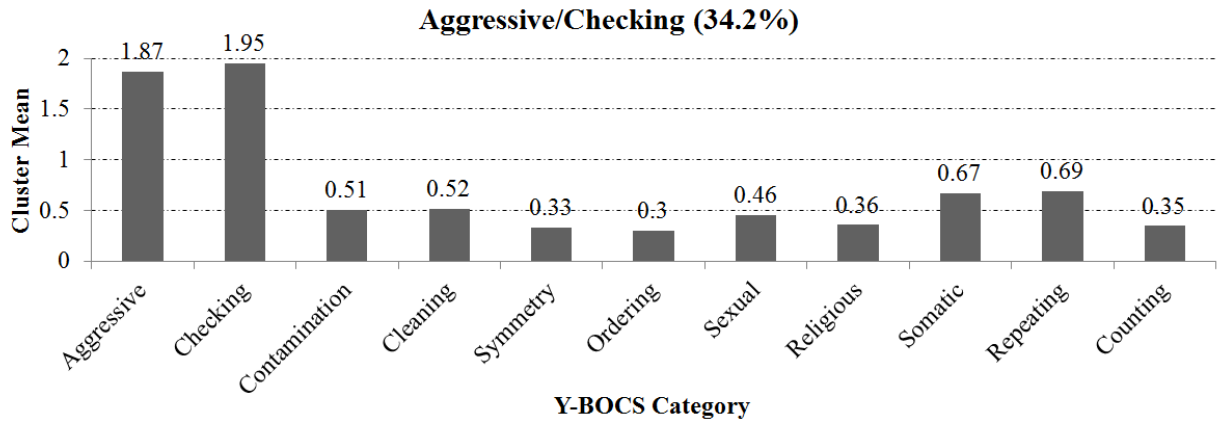
Variable	*Factor 1*	*Factor 2*	*Factor 3*	*Factor 4*	*Communality*	*MSA*
Aggressive Obsessions	.193	.189	**.870**	**.456**	.758	.694
Contamination Obsessions	.234	**.885**	.198	**.309**	.795	.617
Symmetry Obsessions	**.886**	.282	.209	.259	.800	.655
Cleaning Compulsions	**.359**	**.802**	.100	**.353**	.660	.642
Checking Compulsions	**.309**	**.373**	**.580**	**.606**	.472	.794
Ordering Compulsions	**.834**	**.333**	.158	.250	.700	.664
Sexual Obsessions	.115	.026	.267	**.448**	.209	.696
Religious Obsessions	.103	.167	**.363**	**.520**	.291	.776
Somatic Obsessions	.192	.183	.218	**.444**	.199	.818
Repeating Compulsions	**.467**	.273	.253	**.508**	.343	.878
Counting Compulsions	.281	.073	.174	.244	.108	.803

Legend: MAP = Velicer's Minimum Average Partials test; MSA = Measure of sampling adequacy for individual variables.

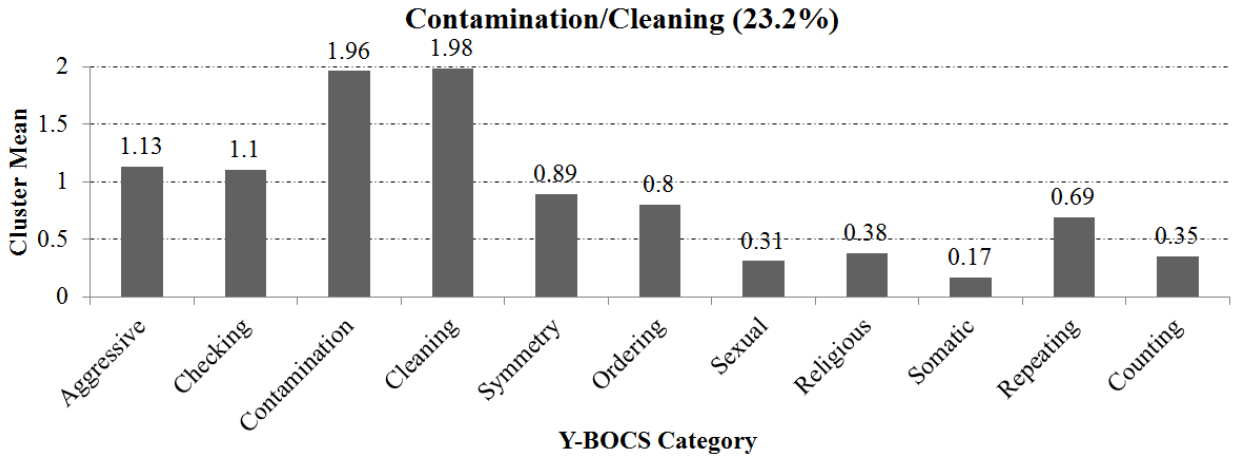
Figure 1

Cluster Membership

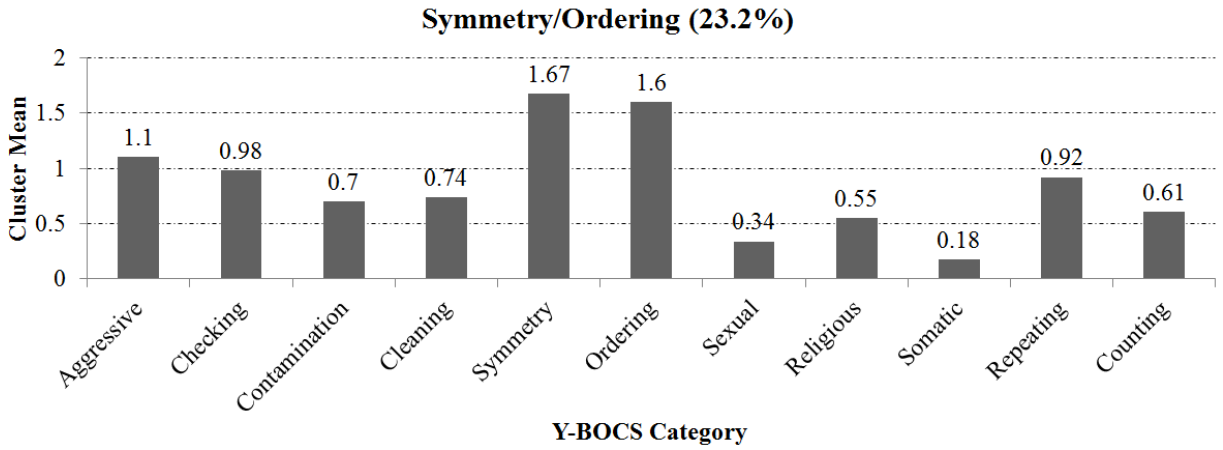
1a)



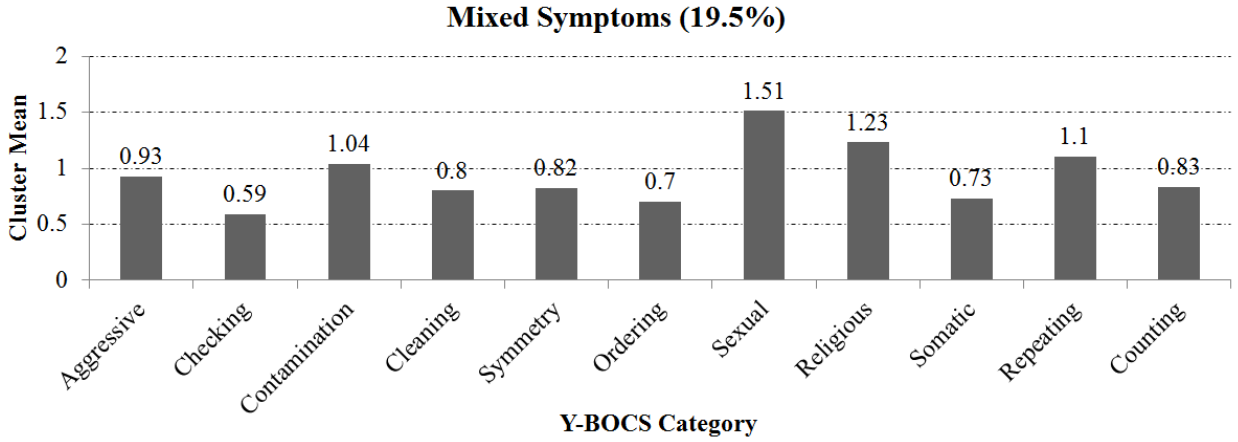
1b)


Contamination/Cleaning (23.2%)
Cluster Mean
2
1.5
1
0.5
0
1.13
1.1
1.96
1.98
0.89
0.8
0.31
0.38
0.17
0.69
0.35
Aggressive
Checking
Contamination
Cleaning
Symmetry
Ordering
Sexual
Religious
Somatic
Repeating
Counting
Y-BOCS Category

1c)


Symmetry/Ordering (23.2%)
Cluster Mean
2
1.5
1
0.5
0
1.1
0.98
0.7
0.74
1.67
1.6
0.34
0.55
0.18
0.92
0.61
Aggressive
Checking
Contamination
Cleaning
Symmetry
Ordering
Sexual
Religious
Somatic
Repeating
Counting
Y-BOCS Category

1d)


Mixed Symptoms (19.5%)
Cluster Mean
2
1.5
1
0.5
0
0.93
0.59
1.04
0.8
0.82
0.7
1.51
1.23
0.73
1.1
0.83
Aggressive
Checking
Contamination
Cleaning
Symmetry
Ordering
Sexual
Religious
Somatic
Repeating
Counting
Y-BOCS Category

CHAPTER 4

STUDY 3

TITLE: Differences in Neuropsychological Performance between Incompleteness and Harm Avoidance Core Dimensions in Obsessive-Compulsive Disorder

AUTHORS: Duncan H. Cameron, B.A.; Laura J. Summerfeldt, Ph.D., C.Psych.; Karen Rowa, Ph.D., C.Psych.; Margaret C. McKinnon, Ph.D., C.Psych.; Neil A. Rector, Ph.D., C.Psych.; Peggy Richter, M.D., F.R.C.P.(C); Tisha Ornstein, Ph.D., C.Psych.; Randi E. McCabe, Ph.D., C.Psych.

CONTEXT AND IMPLICATIONS OF THIS STUDY: Study 3 of this thesis aims to address the limitations of the conventional subtyping methods detailed at length in the previous two studies. Having concluded with the results of the factor and cluster analyses that the typical method of using dimension reduction procedures on responses to the Y-BOCS may not be the most suitable approach to defining symptom dimensions, I then sought to investigate the validity of the core dimensions model by assessing neuropsychological performance in harm avoidance and incompleteness subgroups.

Based on previous theoretical publications, I hypothesized that whereas incompleteness symptoms might be linked to deficits in executive function typically seen in OCD, harm avoidance symptoms might be more closely related to other traits of anxiety disorders and, as such, would be linked to deficits in episodic memory. Indeed it was found that whereas individuals with incompleteness symptoms performed worse on a task measuring set-shifting and problem-solving, the harm avoidance group displayed worse performance on a task of verbal

memory. Furthermore, the incompleteness group showed high ratings of subjective cognitive impairment, although this was not evident on the majority of objective measures.

ACKNOWLEDGEMETNS

This study was funded in part by a grant from the Canadian Institutes of Health Research as part of a randomized clinical trial investigating the effects of exercise as a stand-alone and adjunctive treatment option for obsessive-compulsive disorder.

CONFLICTS OF INTEREST: None

PUBLISHED IN: Journal of Obsessive-Compulsive and Related Disorders, submitted

Abstract

Background: Considerable attention has been given to research investigating potential symptom-based subtypes of obsessive-compulsive disorder (OCD). The identification of several common symptom subtypes from dimension reduction techniques applied to OCD symptom questionnaires, however, has not translated well into further clinical or research applications. The division of symptom themes into those related to incompleteness (INC) and those related to harm avoidance (HA) has been identified as an alternative, though research in this area remains embryonic. *Objective:* The aim of this study was to elucidate any potential differences between these two symptom themes based on neuropsychological task performance. *Method:* Participants (N = 124) with a primary DSM-5 diagnosis of OCD were recruited and grouped into INC or HA based on direct report of their current most troubling symptoms. All participants completed a set of neuropsychological tasks covering verbal memory and various executive function subdomains. *Results:* Those in the INC group performed better on several variables related to verbal memory ($p < .05$) while those in the HA group showed better performance on the Tower of London task ($p < .05$). The INC group also rated themselves as significantly more impaired across domains of subjective cognition. *Discussion:* The differences observed in this study on neuropsychological task performance are in line with the hypothesis that HA symptoms of OCD may be more closely related to generalized anxiety—with poorer performance observed on verbal memory—while INC symptoms appear to be related to focused executive function and problem-solving deficits. Despite similar performance on most other neuropsychological tasks, those in the INC group rated themselves as having significantly greater impairment on a measure of subjective cognition, indicating greater perfectionism tendencies and negative self-evaluation.

Keywords: *OCD, subtypes, neuropsychology, harm avoidance, incompleteness*

Introduction

Obsessive-compulsive disorder (OCD) is characterized by recurring, unwanted and intrusive thoughts (obsessions), and by attempts to ignore or suppress these thoughts or neutralize them with actions such as performing compulsions (American Psychological Association, 2013). OCD is typically recognized as a heterogeneous condition wherein several patients with the same diagnosis could present with completely different overt symptoms. This observation led to the notion that OCD may be usefully defined by characteristic subtypes. While several subtyping methods have been explored, symptom-based subtypes in which specific obsessions could be paired with matching compulsions in presentations frequently observed have dominated the literature on this topic. The most common method for identifying these subtypes has been to perform factor analysis on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989) symptom checklist (e.g., Baer, 1994, Mataix-Cols, Rauch, Manzo, Jenike, & Baer, 1999; Summerfeldt, Richter, Antony, & Swinson, 1999)—a comprehensive yet still incomplete list of symptoms commonly observed in OCD.

A 2008 meta-analysis of factor analysis results showed four common factors including symmetry, forbidden thoughts, cleaning and hoarding (Bloch et al., 2008). Cluster analysis has also been used to a similar end in several cases (Abramowitz et al., 2003; Calamari et al., 1999; Calamari et al., 2004; and Hasanpour et al., 2017). To-date, approximately thirty studies have investigated dimension reduction techniques in the context of the Y-BOCS symptom checklist.

An alternate approach to understanding heterogeneity in OCD emphasizes the motivational and affective themes which underlie OCD symptoms. Building upon a framework originally proposed by Rasmussed & Eisen (1992), Summerfeldt (2004) operationalized the

concept of "core dimensions" of OCD. Rather than grouping by specific overt symptoms, as described above, the core dimensions model relies posits two broader categories—Harm Avoidance (HA) and Incompleteness (INC). Individuals with INC may present symptom themes more commonly associated with the perfectionism tendencies unique to OCD, such as the need to complete an action to a desired level of satisfaction, or over-evaluation of need to correct feelings of dissatisfaction (sometimes referred to as "not just right experiences"). HA symptoms, meanwhile, are more similar to themes commonly seen in generalized anxiety, such as excessive exaggeration of potential harm.

Since Summerfeldt's original presentation of these dimensions (2004) a number of studies have investigated their validity and have attempted to elucidate their theoretical utility (e.g., Ecker & Gönner, 2008, 2017; Summerfeldt, Kloosterman, Antony, & Swinson, 2014). While this research is still embryonic in its development, the core dimensions model holds promise both theoretically and methodologically over some other subtyping methods in that it allows symptoms to be classified based on motivation rather than specific behaviour. For example, while any two individuals with doubting/checking symptoms might be classified into the same dimension or subtype using the common symptom subtyping approach, the core dimensions model allows a researcher or clinician to distinguish between whether this behaviour stems from a need to prevent harm, or a desire to extinguish feelings of incompleteness. Though the compulsive behaviour remains the same between these two cases, the source obsession underlying the compulsion is different. Indeed, the two core dimensions have been found to cross over distinct overt symptom presentations, while being correlated with some more than others (e.g., Belloch et al., 2016; Ecker & Gönner, 2008; Sibrava, Boisseau, Eisen, Mancebo, & Rasmussen, 2016).

The core dimensions model may have utility for clarifying the neurobiological features of OCD. Neuropsychology has been used to aid in the elucidation of these neurobiological underpinnings, but results across individual studies have been consistently heterogeneous. Indeed it may be the case that the different symptoms of OCD are a possible explanation for this heterogeneity, and harm avoidance and incompleteness might inform this research. Several groups have attempted to elucidate the common symptom subtypes of OCD by evaluating group differences or correlations between symptom groups and performance on various neuropsychological tasks, with the majority providing mixed results (Leopold & Backenstrass, 2015). One potential reason why the majority of results reported in this large body of literature is that the groups being tested—if the participants aren't tested homogeneously—aren't etiologically valid. Regardless, neuropsychological performance can offer significant insights into the neurobiological correlates involved of a condition, and, by extension, can be used to empirically support differences between symptom presentation patterns.

Although the existing research on the core dimensions model is mostly theoretical in nature, some trends emerge that inform the framework for the present study. Meta-analyses of neuropsychology in OCD tend to show that executive function subdomains are those most frequently identified to be impaired, albeit with small-to-medium effects (Abramovitch, Abramowitz, & Mittelman, 2013; Shin, Lee, Kim, & Kwon, 2014). However, some new hypotheses arise when taken in context with the clinical observations that harm avoidance aligns with themes of anxiety and anxiety disorders (e.g., Ecker & Gönner, 2008) and incompleteness with the selection of behavioural responses, the production of coherent subsequences of goal-oriented actions, and the switching of task priorities in response to feedback. It has also been suggested that a possible neurobiological correlate of incompleteness might lie in dysfunction of

the basal ganglia resulting in an inability to proactively terminate a repetitive behaviour (Rapoport, 1991; Summerfeldt, 2004).

As such, the aim of the present study was to investigate differences in neuropsychological performance between individuals with harm avoidance symptoms, and those with incompleteness symptoms. There is a paucity of peer-reviewed research on neuropsychological performance in traditional anxiety disorders, with many negative results reported; however, deficits in episodic memory (e.g., as measured by the California Verbal Learning Test; CVLT) have been reported (Airaksinen, Larsson, & Forsell, 2004; Castadena, Tuulio-Henriksson, Marttunen, Suyisaari, & Lönngvist, 2008). We therefore hypothesized that individuals in our sample with harm avoidance-related symptoms would perform more poorly on measures of episodic memory, whereas those with incompleteness-related symptoms would perform more poorly on measures of executive function, particularly those related to set-shifting.

Method

Participants

Participants (N = 124) with a primary DSM-5 diagnosis of OCD were recruited at two Canadian Anxiety Disorders Clinics: the Frederick W. Thompson Anxiety Disorders Centre, Sunnybrook Health Sciences Centre in Toronto, Ontario, and the Anxiety Treatment and Research Clinic, St. Joseph's Healthcare Hamilton in Hamilton, Ontario. The sample described in this study was part of an ongoing randomized clinical trial investigating the effects of exercise as a stand-alone and adjunctive (with cognitive behavioural therapy; CBT) treatment option for OCD. Inclusion criteria were 1) between 18 and 55 years of age, 2) stable medication status for a minimum of eight weeks, and 3) a Y-BOCS score ≥ 17. Exclusion criteria included 1) previous

course (≥ 8 sessions) of CBT for OCD in the past two years, 2) concurrent diagnosis of a severe mood disorder, schizophrenia or other psychotic disorders, or substance abuse/dependence, 4) suspected organic pathology, and 5) incapable of providing informed consent. The sample was 57% female, and the mean age was 33.3 (SD = 10.3; see table 1 for sample demographics).

Procedure

Upon confirmation of initial eligibility all participants completed a Structured Clinical Interview for Axis I Disorders (SCID-I/P; First, Spitzer, Karg, & Spitzer, 2015) to assess the presence of DSM-5 OCD. Participants then completed a battery of neuropsychological tasks assessing a variety of cognitive domains (described below) as well as several symptom severity questionnaires and a measure of subjective cognition. Participants were then assigned to either the HA group or the INC group based collectively on their responses to three separate symptom measures—the currently most troubling obsessions and compulsions as ascertained by the Y-BOCS symptom checklist, the Brown Assessment of Beliefs Scale (BABS) and the Subtype-Q (see below). Group assignment was based primarily on verbatim responses of each participant's current most troubling symptom on the Subtype-Q, but in cases where this was unclear, responses from the Y-BOCS and BABS (identification of most troubling belief) were used to ensure correct group assignment. Group assignment was performed after completion of the randomized-controlled trial mentioned earlier. Blind assignment was completed by one of the authors of the study (DHC) with an inter-rater reliability check completed by the study site coordinator, and any disagreements were resolved by consensus. In the case of the present study, secondary symptoms were not considered in group assignment. This study protocol was approved by the ethics review boards of both institutions.

Measures

Symptom Measures. The *Yale-Brown Obsessive-Compulsive Scale (Y-BOCS)* was used to assess obsessive-compulsive symptom severity (Goodman, Price, Rasmussen, & Mazure, 1989). The Y-BOCS is a standardized rating scale measuring 10 items pertaining to obsessions and compulsions on a five-point adjectival scale ranging from 0 (no symptoms) to 4 (severe symptoms).

The *Beck Depression Inventory-II (BDI)* was used to measure depressive symptom severity (Beck, Steer, & Brown, 1996). The BDI is a 21-item scale with answer options ranging from 0 (not present) to 3 (severe).

The *Depression Anxiety and Stress Scales 21 (DASS-21)* is a self-report questionnaires with 21 items covering three factors—depression, anxiety and stress (Lovibond & Lovibond, 1995). Each item is scored from 0 (did not apply to me at all) to 3 (applied to me very much or most of the time).

The *Brown Assessment of Beliefs Scale (BABS)* is a seven-item clinician-administered measure assessing delusionality of beliefs in (Eisen et al., 1998). The first step in the scale's use required identification of the respondent's current dominant belief, in this case an obsession and the matching compulsion. This was the only item of this scale used, and the severity scale portion was not used in the present analysis.

The *Subtype-Q* is a measure developed specifically for use in current study. It is a clinician-administered measure and requires the participant to report their current most troubling obsession and compulsion.

Subjective Cognition. The *Memory and Cognitive Confidence Scale (MACCS)* was used to assess subjective cognitive performance (Nedeljkovic & Kyrios, 2007). The MACCS is a 28-item scale with items scored from 1 (strongly disagree) to 5 (strongly agree), and the items cover four subscales measuring general memory, decision-making, attention/concentration, and high standards about one's cognitive performance.

Neuropsychology measures. The *California Verbal Learning Test—Second Edition (CVLT-II)* was used to assess verbal memory (Delis, Kramer, Kaplan, & Ober, 2000). The task requires the participant to learn a list of 16 words covering four different semantic categories over the course of five trials. The list is then tested during a) an immediate free and cued recall trials, followed by b) delayed free and cued recall trials (20 minutes following immediate recall). Finally, a recognition trial is administered following the delayed recall trial. The number of correct responses, as well as repetitions and intrusions are measured. The Trial 1-5 total score, Interference Trial (List B) score, Short and Long Delay Cued and Free Recall trial scores, and Total Repetitions and Intrusions were analyzed in the present study.

The *Wechsler Test of Adult Reading (WTAR)* was used to estimate verbal IQ (Wechsler, 1999). This task requires the participant to read a list of 50 words of increasing difficulty and was only used to assess pre-morbid function but not as a primary outcome measure.

The *Tower of London (TOL)* was used to assess decision-making/planning performance (Culbertson & Zillmer, 2001). This task requires participants to arrange three coloured beads on a peg board to match a given pattern in as few moves as possible. More problems solved correctly and shorter execution time indicate greater problem-solving skills and mental flexibility, while greater initiation time indicates greater planning and inhibition of impulsivity.

The *Golden Stroop Task* was used to assess processing speed, attention and executive function (Golden & Freshwater, 1978). The task requires reading a word list as quickly as possible without making mistakes in the first trial, reading colours in the second trial, and reading the colour of the ink in which a word is printed in the third, interference trial.

Data Analysis

A series of independent samples *t*-tests were used to test for differences between the HA and INC group on all primary outcome measures. Pearson's chi-square test or independent samples *t*-tests were used to test for differences between groups on demographic variables. Cases with missing data were excluded from the analysis. Hedges' *g* was used as a measure of effect size given the unequal sample size between groups (interpretation: small = .20, medium = .50, large = .80; Cohen, 1997).

Results

A total of $N = 87$ participants were classified as having symptoms related to harm avoidance while $N = 37$ participants were assigned to the incompleteness group. As shown in Table 1, there were no significant differences between the two groups on any demographic or symptom severity measures. The groups were also equal in their performance on the WTAR, indicating equivalent verbal IQ.

Subjective Cognition

The INC group had significantly higher self-ratings of subjective cognitive impairment compared to the HA group, as measured by the MACCS total score ($t(116) = -2.11$, $p = .037$) as well as on the subscales of decision-making ($t(118) = -2.26$, $p = .025$), attention/concentration ($t(121) = -2.84$, $p = .005$) and high standards ($t(122) = -2.40$, $p = .024$).

Verbal Memory

Participants in the HA group were seen to perform worse on several variables of the CVLT-II, including trial 1-5 total ($t(122) = -1.99$, $p = .048$), short delay free recall ($t(122) = -2.01$, $p = .047$), short delay cued recall ($t(122) = -2.08$, $p = .040$) and long delay cued recall ($t(121) = -2.40$, $p = .034$), indicating generally worse verbal memory performance in the HA group relative to the INC group.

Executive Function

The participants in the INC group solved significantly fewer problems correctly on the TOL than did those in the HA group ($t(121) = 2.15$, $p = .034$), indicating better problem solving ability and mental flexibility in the HA group. Those in the HA group also took significantly less time to execute the problems than did participants in the INC group ($t(120) = 2.13$, $p = .035$). No other significant differences were seen for any other TOL variables or on the Stroop task.

Discussion

The purpose of this study was to investigate differences in neuropsychological performance between individuals with primary obsessive-compulsive symptoms related to harm avoidance and those with symptoms related to incompleteness. In line with the notion that incompleteness is more related to unique features of OCD, we hypothesized that participants in the INC group would perform more poorly on measures of executive function which tend to dominate reviews of neuropsychological performance in this disorder. Those in the HA group—with symptoms more related to features of anxiety—were expected to perform more poorly on a verbal memory task. We found that, while those in the INC group rated themselves as being subjectively more impaired than the HA group across a variety of cognitive domains,

neuropsychological task performance did not differ significantly between groups. There were two exceptions. On the TOL, participants in the INC group solved significantly fewer problems correctly and took more time to execute the problems, indicating greater impairment in problem-solving, planning and set-shifting ability. The HA group scored significantly lower than the INC group on the CVLT, indicating poorer verbal memory performance in the HA group. No significant differences were found for the Stroop Task, indicating specific executive subdomain impairments rather than broad executive dysfunction for those in the INC group.

Impairment on the TOL task often appears among the highest of effects in meta-analyses of neuropsychological function in OCD (Shin et al., 2014) and it has been suggested that frontostriatal dysfunction as indicated by impairment in planning may act as an endophenotype of OCD (Vaghi, Hampshire, Fineberg, & Kaser, 2017). This is the first study, however, to assess performance in putative HA and INC subgroups. The focused and highly specific deficits observed for the TOL in the INC group relative to those in the HA group provide some potentially interesting insights into the differences behind the defining features of these core dimensions. The presence of a significant difference in the number of problems solved correctly and in the execution time, but not on any other variables indicates that the INC group is spending more time on response checking or is having greater difficulty in generating alternative strategies once an error is made, though they are not making significantly more errors, as measured by total move score. This corroborates the hypothesis of potential set-shifting difficulties associated with incompleteness. Furthermore, differences in processing speed and attention were not observed on the Stroop Task, eliminating these as possible explanations for the longer execution time seen on the TOL. Finally, given that the INC group was significantly more impaired on this task, and showed mean standard scores falling in the low average range (total correct = 91.51, execution

time = 91.19) compared to average for the HA group, the notion of a planning difficulty as an endophenotype of OCD may need to be re-evaluated in the context of the well-accepted heterogeneity of this disorder.

In contrast to the highly circumscribed differences between the INC and HA groups in executive function impairments, the HA group showed significantly greater impairments in verbal memory performance on several measures of the CVLT. Although very little research is available comparing anxiety disorders in terms of neuropsychological function, episodic memory is one of few domains observed to be impaired relative to healthy controls (Airaksinen et al., 2004; Castadena et al., 2008). Interestingly, verbal memory is rarely seen to have more than a small effect in meta-analyses of neurocognitive function in OCD (Abramovitch et al., 2013; Shin et al., 2014), and in the present study it was indeed the case that both groups demonstrated adequate performance with trial 1-5 total T-scores falling in the average range. The finding that the HA group was significantly deficient on this task relative to the INC group highlights some further differences between these two symptom themes and draws a fundamental link between harm avoidance tendencies and other anxiety disorders, though the mechanisms underlying these differences will require elucidation in future study.

Perhaps the most intriguing finding in the present study is the fact that the INC group rated themselves as significantly more impaired on a measure of subjective cognition, relative to the HA group, when in fact there were relatively few objective differences to support this and the INC group exhibited better memory performance. Interestingly, the general memory subscale of the MACCS was the only domain for which there was not a significant difference between groups. While several past studies have investigated the role of memory and cognitive confidence—particularly as they pertain to checking rituals—in OCD (for example, Boschen &

Vuksanovic, 2007; Radomsky & Alcolado, 2010; Tolin et al., 2001), further study might examine the relationship between decreased memory confidence and increased checking between core dimensions. In addition, metacognition may also play a key role here as it has been implicated in the aetiology of OCD symptoms, and treatments involving metacognitive strategies have shown promise, but this topic has not been assessed in conjunction with neuropsychology or core dimensions. This finding underscores exaggerated negative self-evaluation and perfectionism tendencies potentially associated with incompleteness symptoms in OCD, and not observed in those with harm avoidance symptoms in this sample.

This study had several limitations, which also suggest directions for future research. First, as might be expected from the naturalistic presentation of OCD, the INC group was considerably smaller than the HA group, and although the results of this study were interpreted in that context, future study should seek to corroborate these results with greater representation of INC symptom profiles. We also did not correct for multiple comparisons in our analysis as each measure reflected distinct processes at different levels of ecological validity and given this is the first study to examine these questions. However, a Bonferroni correction for multiple comparisons (alpha = .05/26 = .002) would render all results observed here insignificant.

We also did not include a healthy control group, which would aid with comparison in order to determine magnitude of impairment in cognitive function. Nevertheless, the tasks we included in the present study all have norms, and as can be seen in Table 2, the T-scores and standard scores fall exclusively in the range of low-average to average range, which is typical of the modest impairments generally observed in this population. In addition, recent literature suggests a mediating effect of obsessive-compulsive symptoms and motivational factors (Moritz, Hauschildt, Saathoff, & Jelinek, 2017) as well as stereotype threat (Moritz, Spirandelli, Happach,

& Lion, 2018) on neuropsychological task outcomes in OCD. None of these were assessed in this study and future investigations of neuropsychology and OCD should seek to include them.

Finally, we identified HA and INC participants inferentially, on the basis of current overt symptoms. When seeking to define core dimension subgroups, simply stratifying by current most troubling symptom may not be the most accurate approach as OCD can frequently present with multiple symptoms and what patients deem to be their most troubling symptom at one time may be subject to change. To-date, there are no published measures specifically designed for this purpose. In addition, measuring HA and INC directly may yield different results. Measures developed to operationalize these constructs include the Obsessive-Compulsive Core Dimensions Questionnaire (OC-CDQ) or Obsessive-Compulsive Core Dimensions Interview (OC-CDI; Summerfeldt et al., 2001, 2014). Grouping of participants with the aid of such a tool would greatly increase the validity of results.

The aim of this study was to investigate differences in neuropsychological performance between individuals with primary obsessive-compulsive symptoms related to harm avoidance and those with symptoms related to incompleteness. Our results reflect distinct, focused differences between these two symptom dimensions and provide some early objective evidence supporting some of the hypotheses behind these constructs. It was observed that some of the neuropsychological features thought to be a trademark OCD (including executive function subdomains such as planning, problem-solving and set-shifting ability) appear to be true only of those with symptoms related to incompleteness, whereas those which characterize other anxiety disorders (e.g., episodic memory) are true only of participants with symptoms related to harm avoidance. These findings hold implications not only for our understanding of the neuropsychological substrates of OCD, but also for the heterogeneity observed in this population

and possibly its taxometric placement. Future study should aim to reinforce these findings with additional neuropsychological measures and the use of clinical questionnaires such as the OC-CDQ or OC-CDI.

References

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: A meta-analysis. *Clinical psychology review*, *33*(8), 1163-1171.

Abramowitz, J. S., Franklin, M. E., Schwartz, S. A., & Furr, J. M. (2003). Symptom presentation and outcome of cognitive-behavioral therapy for obsessive-compulsive disorder. *Journal of Consulting and clinical psychology*, *71*(6), 1049.

Airaksinen, E., Larsson, M., & Forsell, Y. (2005). Neuropsychological functions in anxiety disorders in population-based samples: evidence of episodic memory dysfunction. *Journal of psychiatric research*, *39*(2), 207-214.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.

Baer, L. (1994). Factor analysis of symptom subtypes of obsessive compulsive disorder and their relation to personality and tic disorders. *The Journal of clinical psychiatry*.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Beck depression inventory-II. *San Antonio*, *78*(2), 490-8.

Belloch, A., Fornés, G., Carrasco, A., López-Solá, C., Alonso, P., & Menchón, J. M. (2016). Incompleteness and not just right experiences in the explanation of obsessive–compulsive disorder. *Psychiatry research*, *236*, 1-8.

Belloch, A., Morillo, C., Luciano, J. V., García-Soriano, G., Cabedo, E., & Carrió, C. (2010). Dysfunctional belief domains related to obsessive-compulsive disorder: A further examination of their dimensionality and specificity. *The Spanish Journal of Psychology*, *13*(1), 376-388.

Bloch, M. H., Landeros-Weisenberger, A., Rosario, M. C., Pittenger, C., & Leckman, J. F. (2008). Meta-analysis of the symptom structure of obsessive-compulsive disorder. *American Journal of Psychiatry*, *165*(12), 1532-1542.

Boschen, M. J., & Vuksanovic, D. (2007). Deteriorating memory confidence, responsibility perceptions and repeated checking: Comparisons in OCD and control samples. *Behaviour research and therapy*, *45*(9), 2098-2109.

Calamari, J. E., Wiegartz, P. S., & Janeck, A. S. (1999). Obsessive–compulsive disorder subgroups: a symptom-based clustering approach. *Behaviour Research and Therapy*, *37*(2), 113-125.

Calamari, J. E., Wiegartz, P. S., Riemann, B. C., Cohen, R. J., Greer, A., Jacobi, D. M., ... & Carmin, C. (2004). Obsessive–compulsive disorder subtypes: an attempted replication and extension of a symptom-based taxonomy. *Behaviour Research and Therapy*, *42*(6), 647-670.

Castaneda, A. E., Tuulio-Henriksson, A., Marttunen, M., Suvisaari, J., & Lönnqvist, J. (2008). A review on cognitive impairments in depressive and anxiety disorders with a focus on young adults. *Journal of affective disorders*, *106*(1-2), 1-27.

Culbertson, W. C., & Zillmer, E. (2001). *Tower of London-Drexel University (TOLDX)*. Multi-Health Systems.

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *CVLT-II: California verbal learning test: adult version*. Psychological Corporation.

Ecker, W., & Gönner, S. (2008). Incompleteness and harm avoidance in OCD symptom dimensions. *Behaviour Research and Therapy*, *46*(8), 895-904.

Ecker, W., & Gönner, S. (2017). Current Status of Research Concerning Incompleteness in Obsessive-Compulsive Disorder. *VERHALTENSTHERAPIE*, *27*(2), 120-128.

Eisen, J. L., Phillips, K. A., Baer, L., Beer, D. A., Atala, K. D., & Rasmussen, S. A. (1998). The brown assessment of beliefs scale: reliability and validity. *American Journal of Psychiatry*, *155*(1), 102-108.

First, M. B., Spitzer, R. L., Gibbon, M. W. J. B., & Williams, J. B. (1995). Structured clinical interview for DSM-IV axis I disorders. *New York: New York State Psychiatric Institute*.

Cohen, J. (1988). Statistical power analysis for the behavioral sciences. 2nd.

Culbertson, W. C., & Zillmer, E. (2001). *Tower of London-Drexel University (TOLDX)*. Multi-Health Systems.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., ... & Charney, D. S. (1989). Yale-brown obsessive compulsive scale (Y-BOCS). *Arch gen psychiatry*, *46*, 1006-1011.

Hasanpour, H., Asadi, S., Meibodi, R. G., Daraeian, A., Ahmadiani, A., Shams, J., & Navi, K. (2017). A critical appraisal of heterogeneity in Obsessive-Compulsive Disorder using symptom-based clustering analysis. *Asian journal of psychiatry*, *28*, 89-96.

Leopold, R., & Backenstrass, M. (2015). Neuropsychological differences between obsessive-compulsive washers and checkers: A systematic review and meta-analysis. *Journal of anxiety disorders*, *30*, 48-58

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour research and therapy*, *33*(3), 335-343.

Mataix-Cols, D., Rauch, S. L., Manzo, P. A., Jenike, M. A., & Baer, L. (1999). Use of factor-analyzed symptom dimensions to predict outcome with serotonin reuptake inhibitors and placebo in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *156*(9), 1409-1416.

Moritz, S., Hauschildt, M., Saathoff, K., & Jelinek, L. (2017). Does impairment in neuropsychological tests equal neuropsychological impairment in obsessive-compulsive disorder (OCD)? Momentary influences, testing attitude, and motivation are related to neuropsychological performance in OCD. *Journal of obsessive-compulsive and related disorders*, *14*, 99-105.

Moritz, S., Spirandelli, K., Happach, I., Lion, D., & Berna, F. (2018). Dysfunction by Disclosure? Stereotype Threat as a Source of Secondary Neurocognitive Malperformance

in Obsessive-Compulsive Disorder. *Journal of the International Neuropsychological Society*, 1-9.

Nedeljkovic, M., & Kyrios, M. (2007). Confidence in memory and other cognitive processes in obsessive–compulsive disorder. *Behaviour Research and Therapy*, *45*(12), 2899-2914.

Radomsky, A. S., & Alcolado, G. M. (2010). Don't even think about checking: Mental checking causes memory distrust. *Journal of Behavior Therapy and Experimental Psychiatry*, *41*(4), 345-351.

Rapoport, J. L. (1991). Basal ganglia dysfunction as a proposed cause of obsessive-compulsive disorder. *Psychopathology and the brain*, 77-95.

Rasmussen, S. A., & Eisen, J. L. (1992). The epidemiology and clinical features of obsessive compulsive disorder. *Psychiatric Clinics*, *15*(4), 743-758.

Rees, C. S., & Anderson, R. A. (2013). A review of metacognition in psychological models of obsessive–compulsive disorder. *Clinical Psychologist*, *17*(1), 1-8.

Shin, N. Y., Lee, T. Y., Kim, E., & Kwon, J. S. (2014). Cognitive functioning in obsessive-compulsive disorder: a meta-analysis. *Psychological medicine*, *44*(6), 1121-1130.

Sibrava, N. J., Boisseau, C. L., Eisen, J. L., Mancebo, M. C., & Rasmussen, S. A. (2016). An empirical investigation of incompleteness in a large clinical sample of obsessive compulsive disorder. *Journal of anxiety disorders*, *42*, 45-51.

Summerfeldt, L. J., Kloosterman, P. H., Antony, M. M., Richter, M. A., & Swinson, R. P. (2004). The relationship between miscellaneous symptoms and major symptom factors in obsessive-compulsive disorder. *Behaviour Research and Therapy*, *42*(12), 1453-1467.

Summerfeldt, L. J., Kloosterman, P. H., Antony, M. M., & Swinson, R. P. (2014). Examining an obsessive-compulsive core dimensions model: Structural validity of harm avoidance and incompleteness. *Journal of Obsessive-Compulsive and Related Disorders*, *3*(2), 83-94.

Summerfeldt, L. J., Richter, M. A., Antony, M. M., & Swinson, R. P. (1999). Symptom structure in obsessive-compulsive disorder: a confirmatory factor-analytic study. *Behaviour Research and Therapy*, *37*(4), 297-311.

Tolin, D. F., Abramowitz, J. S., Brigidi, B. D., Amir, N., Street, G. P., & Foa, E. B. (2001). Memory and memory confidence in obsessive–compulsive disorder. *Behaviour Research and Therapy*, *39*(8), 913-927.

Vaghi, M. M., Hampshire, A., Fineberg, N. A., Kaser, M., Brühl, A. B., Sahakian, B. J., ... & Robbins, T. W. (2017). Hypoactivation and dysconnectivity of a frontostriatal circuit during goal-directed planning as an endophenotype for obsessive-compulsive disorder. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *2*(8), 655-663.

Wechsler, D. (2001). *Wechsler Test of Adult Reading: WTAR*. Psychological Corporation.

Appendix I
Tables

Table 1
Sample Demographic Characteristics

	Harm Avoidance (n = 87)	Incompleteness (n = 37)	
Variable	Mean (SD)	Mean (SD)	*t*
Age (years)	32.30 (9.84)	35.30 (11.31)	-1.48
Sex (% female)	55%	62%	.518[a]
Education (years)	15.21 (2.52)	14.94 (2.19)	.547
Age of Onset	17.68 (10.22)	18.46 (12.52)	-.341
Duration of Illness (years)	14.31 (10.38)	17.10 (13.13)	-1.22
WTAR Standard Score	112.59 (10.84)	111.97 (8.82)	.301
Y-BOCS			
Obsessions Score	11.78 (3.20)	12.00 (2.84)	-.337
Compulsions Score	11.77 (3.48)	13.03 (2.40)	-1.94
Total Score	23.55 (5.97)	25.03 (4.84)	-1.28
DASS-21			
Depression Scale	8.47 (6.52)	9.36 (5.79)	-.709
Anxiety Scale	7.01 (5.44)	6.83 (4.78)	.171
Stress Scale	10.71 (5.21)	11.67 (4.80)	-.949
Total Score	26.19 (15.29)	27.86 (13.24)	-.572
BDI	20.54 (5.97)	25.03 (4.84)	-.641

Legend: BDI = Beck Depression Inventory; DASS = Depression Anxiety and Stress Scales; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; WTAR = Wechsler Test of Adult Reading.

a: Pearson's χ^2

Table 2
Independent Samples t-test

Variable	Harm Avoidance *N*	Harm Avoidance Mean (SD)	Incompleteness *N*	Incompleteness Mean (SD)	*t*	*p*	*g*[b]
MACCS							
Total Score	81	77.98 (19.94)	37	86.30 (19.59)	-2.11	**.037**	.420
General Memory	81	37.19 (13.36)	37	38.95 (13.25)	-.672	.503	.132
Decision-Making	83	14.23 (4.67)	37	16.32 (4.67)	-2.26	**.025**	.448
Attention/ Concentration	86	11.16 (3.90)	37	13.37 (4.11)	-2.84	**.005**	.558
High Standards	87	.53 (.98)	37	.95 (.87)	-2.40	**.024**	.443
CVLT							
Trial 1-5 Total	87	53.97 (11.21)	37	58.23 (10.22)	-1.99	**.048**	.390
List B	87	7.09 (6.79)	37	6.32 (2.12)	.673	.502	.132
SD Free Recall	87	11.40 (3.15)	37	12.59 (2.69)	-2.01	**.047**	.394
SD Cued Recall	87	12.23 (2.88)	37	13.35 (2.38)	-2.08	**.040**	.409
LD Free-Recall	86	11.99 (3.19)	37	12.81 (2.79)	-1.36	.176	.267
LD Cued-Recall	86	12.37 (2.91)	37	13.51 (2.18)	-2.40	**.034**	.420
Total Intrusions	86	3.65 (4.52)	37	2.54 (3.25)	1.55	.179	.265
Total Repetitions	86	5.16 (5.08)	37	5.89 (4.93)	-.736	.463	.145
LD Recognition	86	14.91 (1.52)	37	15.24 (1.03)	-1.22	.223	.237
TOL[a]							
Total Correct	86	98.40 (15.10)	37	91.51 (18.79)	2.15	**.034**	.423
Total Move Score	86	94.72 (17.98)	37	92.87 (15.84)	.544	.588	.121
Initiation Time	86	103.42 (16.43)	37	102.73 (16.65)	.216	.829	.042
Execution Time	85	96.66 (13.13)	37	91.19 (12.79)	2.13	**.035**	.420
Total Time	86	95.12 (13.98)	37	95.08 (14.76)	.012	.990	.003
Time Violations	86	95.79 (18.73)	37	96.08 (18.18)	-.353	.724	.016
Rule Violations	86	100.42 (12.10)	37	99.16 (23.49)	.391	.696	.077
Stroop Test							
Trial 1 T-Score	87	47.90 (8.05)	37	46.41 (7.25)	.971	.333	.190
Trial 2 T-Score	87	46.87 (9.44)	37	45.30 (7.40)	.903	.368	.177
Trial 3 T-Score	87	51.77 (12.36)	37	51.81 (9.59)	-.018	.986	.003
Interference T-Score	87	53.40 (8.67)	37	54.70 (7.22)	-.801	.425	.157

Legend: CVLT = California Verbal Learning Test – Second Edition; LD = Long Delay; MACCS = Memory and Cognitive Confidence Scale; SD = Short Delay; TOL = Tower of London.

a: Values are standard scores.

b: Hedge's *g* interpretation: 0.20 = Small Effect; 0.50 = Medium Effect; 0.80 = Large Effect (Cohen, 1997).

CHAPTER 5

STUDY 4

TITLE: A Pilot Study Examining the Use of Goal Management Training in Individuals with Obsessive-Compulsive Disorder

AUTHORS: Duncan H. Cameron, B.A..; Randi E. McCabe, Ph.D., C.Psych.; Karen Rowa, Ph.D., C.Psych.; Charlene O'Connor, MSc.; Margaret C. McKinnon, Ph.D., C.Psych.

CONTEXT AND IMPLICATIONS OF THIS STUDY: This fourth and final study of the thesis collectively addresses the neuropsychological themes discussed in the previous three studies. It is well accepted that neuropsychological impairment is likely present in individuals with a diagnosis of OCD. A small body of literature exists describing cognitive changes before and after treatment for OCD (medication or cognitive behavioural therapy) but very little research has been dedicated to treatment options that directly address these deficits.

Having discussed at length the cognitive deficits present in OCD in previous chapters, Study 4 builds upon these by describing the implementation of an established cognitive remediation program designed address deficits in fronto-temporally-mediated processes such as planning and decision-making—some of the domains seen to be consistently implicated in OCD. Its findings show that this treatment can confer significant benefits for objective and subjective cognitive performance, as well as for levels of daily functioning. Although these results are preliminary, the finding that cognitive remediation can benefit affected individuals offers a significant contribution to the existing standard treatment options for this chronically affected population. This study offers a foundation upon which future research may investigate its utility as an adjunctive treatment option when paired with other cognitive behavioural approaches.

ACKNOWLEDGEMETNS

None

CONFLICTS OF INTEREST: None

PUBLISHED IN: Psychiatry Research, submitted

Abstract

Background: Results from recent meta-analyses point toward cognitive impairments in obsessive-compulsive disorder (OCD), particularly in such executive function subdomains as planning and organization. Scant attention has focused on cognitive remediation strategies that may reduce cognitive dysfunction, with the potential for a corresponding decrease in core symptoms of OCD. *Objective:* The aim of this study was to assess the feasibility and efficacy of a standardized cognitive remediation program, Goal Management Training (GMT), in a pilot sample of individuals with OCD. *Method:* Nineteen individuals with a primary DSM-5 diagnosis of OCD were randomized to receive either the nine-week GMT program (active group) or to complete a nine-week waiting period (waitlist control). Groups were assessed at baseline, post-treatment, and three-month follow-up. The assessment consisted of neuropsychological tasks assessing a variety of cognitive domains as well as subjective measures of functioning and of symptom severity. *Results:* The active condition showed significant improvements from baseline to post-treatment on measures of inattention, impulsivity, problem-solving and organization compared to controls. Moreover, whereas the active group reported a significant improvement in subjective cognition over the course of treatment, no such improvement emerged in the waitlist group over this same period. Neither group showed improvement on indices of depressive, anxiety or OCD-related symptom severity. *Discussion:* The results of this small pilot investigation of GMT in OCD point towards the potential efficacy of this treatment approach in this population. Replication of these findings is awaited, with current results potentially limited by sample characteristics including motivation to seek and complete treatment.

Introduction

Obsessive-compulsive disorder (OCD) is characterized by the presence of recurrent obsessions and/or compulsions that cause marked anxiety and interfere with daily functioning (American Psychiatric Association, 2013). OCD affects between 2%–3% of adults, and about 1%–2% of adolescents and children (American Psychiatric Association, 2013; Kessler et al., 2005; Zohar, 1999), making it approximately twice as prevalent as schizophrenia or bipolar disorder (Karno, Golding, Sorenson, & Burnam, 1988). The neuropsychology of OCD has received considerable attention, with more than 250 peer-reviewed articles published in the last quarter century exploring cognitive performance in this disorder (Abramovitch & Cooperman, 2015). Individuals with OCD show poor performance across multiple cognitive domains, including on measures of memory and of executive function, with these impairments likely involved in the etiology and maintenance of symptoms (Anderson & Savage, 2004; Fontenelle, Mendlowicz, Mattos, & Versiani, 2006; Greisberg & McKay, 2003; Kathmann, 2008; Kuelz et al., 2006; Muller & Roberts, 2005; Olley, Malhi, & Sachdev, 2007).

In most cases, untreated OCD runs a chronic and deteriorating course. Of individuals diagnosed with OCD, 84% have a chronic course and 14% experience a deteriorating illness (Attiullah, Eisen, & Rasmussen, 2000). Given this chronicity, the psychosocial morbidity of OCD is high (Koran, Thienemann, & Davenport, 1996) exerting a significant adverse impact of persistent symptoms on the person's quality of life (Cassin, Richter, Zhang, & Rector, 2009; Masellis, Rector, & Richter, 2003). According to the World Health Organization, OCD is among the top 10 leading causes of disability worldwide (Brundtland, 2000). In light of its prevalence and associated personal and societal costs, OCD is a significant public health concern in Canada, rendering identification and development of effective, evidence-based treatment approaches

critically important. The National Institute of Clinical Excellence guidelines (National Institute for Health and Clinical Excellence, 2014) on the treatment of OCD recommend cognitive-behavioural therapy (CBT) and selective serotonin reuptake inhibitors (SSRIs) as first-line psychological and medical interventions for OCD. Here, numerous quantitative reviews provide support for the clinical efficacy of CBT and pharmacological interventions for OCD (Eddy, Dutra, Bradley, & Westen, 2004; Olatunji, Davis, Powers, & Smits, 2013). Nonetheless, a successful outcome in OCD is defined typically as a reduction in symptom severity of 25%–50% (Tolin, Abramowitz, & Diefenbach, 2005); the majority of "responders" to first-line treatments are left with residual symptoms that are clinically relevant and disabling.

OCD has been linked neurobiologically to altered functioning in cortico-striato-thalamic-cortical (CSTC) circuits, with the fronto-striatal network thought strongly implicated in the poor performance observed, relative to healthy controls, on measures of executive functioning in this disorder (Chudasama & Robbins, 2006). Consistent with alterations in these circuits, recent meta-analyses reveal primarily medium effect sizes on measures of response inhibition (Cohen's d = -.499), planning (d = -.44), response inhibition (d = -.24 to -.49), set shifting (d = -.32 to -.52), and processing speed (d = -.34 to -.52) (Abramovitch, Abramowitz & Mittelman, 2013; Snyder, Kaiser, Warren & Heller, 2015) in OCD. Interestingly, largest effects are observed on measures of nonverbal memory where effects range from d = -.43 to -.76 (Abramovitch et al., 2013; Rajender et al., 2011; Shin et al., 2014; Snyder et al., 2015). Here, a number of authors suggest that the poor performance observed on some memory tasks among individuals with OCD may be mediated, in part, by organizational deficits (Olley et al., 2007; Savage et al., 1999), underscoring further the central role executive functions play in the development and maintenance of cognitive symptoms in OCD. By contrast, only small effects (d = -.21 to -.33)

have been observed for verbal memory performance. Finally, several studies point towards poor performance on measures of cognitive flexibility/planning and motor inhibition relative to matched controls among unaffected first-degree relatives (Cavedini, Zorzi, Piccinni, Cavallini, & Bellodi, 2010; Lennertz et al., 2012; Viswanath, Reddy, Kumar, Kandavel, & Chandrashekar, 2009), suggesting that alterations in executive functioning might represent a cognitive endophenotype of this illness that is consistent with its proposed neurobiological basis.

Interestingly, cognitive functioning has emerged as a provocative predictor of treatment response in OCD. For example, one preliminary study reported that whereas better performance in some cognitive domains (such as verbal IQ and performance on the Stroop task) predicted increased response to CBT, better verbal memory was predictive of increased response to fluoxetine (D'Alcante et al., 2012). Studies examining changes in neurocognitive function after CBT, or CBT and medication, however, have yielded equivocal results (Abramovitch & Cooperman, 2015). For example, one group found that OCD patients improved significantly on measures of visuospatial reconstruction and nonverbal memory following SSRI treatment (Kang et al., 2003). No such cognitive gains emerged, however, after treatment with fluoxetine (Nielen & Den Boer, 2003). Two studies (Kuelz et al., 2006; Voderholzer et al., 2013) have reported improvements in nonverbal memory and set-shifting ability following CBT; one of these studies also reported gains in visuospatial reconstruction following treatment (Voderholzer et al., 2013). Finally, several studies investigating the effects of combination treatment (i.e., both medication and CBT) report that OCD patients fail to show improvements on any neuropsychological tasks post-treatment (Bannon, Gonsalvez, Croft, & Boyce, 2006; Kim, Park, Shin, & Kwon, 2002; Roh et al., 2005).

Given the low response rate to current front-line treatments for OCD, and equivocal findings surrounding the impact of these interventions on cognitive functioning, there is a pressing need for alternative and/or supplementary treatment options that address not only core symptoms of OCD but also associated neuropsychological changes associated with this disorder. Accordingly, the aim of the present study was to conduct an initial feasibility study examining the efficacy of a well-established cognitive intervention, Goal Management Training (GMT; Levine et al., 2000), in the treatment of cognitive deficits among individuals with OCD. A secondary aim was to determine the impact of this approach on functional outcomes and measures of OCD symptom severity.

Notably, despite knowledge of reduced cognitive performance in OCD and its potential to exert deleterious effects on treatment and on functional outcomes, to date, cognitive remediation has received scant attention in the OCD literature. Here, two studies reported positive effects of cognitive retraining strategies for organizational impairment. In one, OCD participants were trained briefly on organizational strategies, and when assessed using the Rey-Osterrieth Complex Figure Task (RCFT), the training group showed significantly greater organization and accuracy scores compared to healthy controls, but this improvement could not be attributed to the treatment itself (Buhlmann et al., 2006). In a second study, Park and colleagues (2006) used a revised version of the Wechsler Adult Intelligence Scale (WASI) block design task as a training tool, and aided participants in applying problem-solving and organizational strategies to everyday life over the course of nine 60-minute sessions. The authors found that memory function in the treatment group improved and that clinical symptoms were reduced after training when compared to a matched control group. Although neither of these studies employed an established protocol, they point to the potential of cognitive remediation,

focusing on improvement of organizational and planning strategies, as a treatment strategy for patients with OCD. Notably, despite an emphasis on applying organizational and problem-solving strategies, both interventions described here targeted focused skills with the primary aim of improving performance on specific neuropsychological tasks (organizational strategy on either RCFT or WASI block design). Although effective for improving task-specific performance, these approaches often lack the ability to show generalizable improvements.

Goal Management Training (GMT) is a staged cognitive remediation program aimed at recovery of executive function and goal-directed behaviour (Levine et al., 2000). This program is unique in applying a "top-down" approach, focusing primarily on higher-order executive function domains and teaching skills (such as goal-setting and monitoring progress) aimed at regulating these systems Here, participants are expected to leave treatment with strategies that can be applied to a variety of daily tasks, ideally improving not only performance on neuropsychological tasks but also leading to improvements in daily functioning, which can offer significant benefits over the task-specific approaches mentioned above. The efficacy of this approach, both as a stand-alone treatment and a supplementary treatment to psychotherapy, has been demonstrated in clinical and non-clinical populations that experience deficits in executive functioning, attention and memory. These primarily include older adults (Levine et al., 2007; van Hooren et al., 2007) and individuals who have suffered a traumatic brain injury (Krasny-Pacini et al., 2014; Levine et al., 2000; 2011). This program has also been implemented in a variety of populations including ADHD (de Braek, Dijkstra, Ponds, & Jolles, 2012), polysubstance abuse disorder (Alfonso, Caracuel, Delgado-Pastor, & Verdejo-García, 2011), and spina bifida (Stubberud, Langenbahn, Levine, Stanghelle, & Schanke, 2013). In these studies, participants demonstrated improvements in functional outcomes (such as completing everyday tasks) as well

improvements in executive functions including decision-making, working memory and selective attention. A recent meta-analysis of GMT covering 21 studies with 19 separate treatment populations found significant small-to-moderate effects across a wide range of executive function, working memory and long-term memory tasks, further suggesting that GMT offers an effective cognitive remediation intervention (Stamenova & Levine, 2018). Critically, when assessed at follow-up, the effects of GMT on executive function task performance were maintained (Hedges' g = .549). Subjective reports of executive function, however, were not, a finding that requires further investigation.

GMT targets primarily the brain's sustained attention system, regulated by several regions including dorsolateral prefrontal cortex, posterior parietal and thalamic regions (O'Connor, Robertson, & Levine, 2011, Levine et al., 2011; Posner & Peterson, 1990) collectively implicated in executive functioning and higher-order attentional processing. The cognitive strategies trained in GMT are designed to facilitate the resumption of executive control and a reinstatement of self-regulatory goals. Critically, the current CSTC model of the neurobiology of OCD points towards poor performance and related slowness on measures of executive function assessing response inhibition, decision making, task switching and planning in association with dysregulation in related regions including dorsolateral prefrontal cortex and orbitofrontal cortex with these subdomains serving in concert to regulate complex behaviour. Accordingly, we predicted that GMT, targeting selectively neural regions and associated cognitive functions implicated in the CTSC model of OCD, and with demonstrated success in remediating poor performance in clinical populations with executive dysfunction, would be effective in reducing cognitive performance deficits in OCD and in improving daily functioning. Hence, the primary objective of the present study was to conduct a novel test of the clinical

efficacy of a cognitive remediation program aimed at improving goal-directed behaviours that are dependent executive functioning in individuals with DSM-5-diagnosed OCD. Specifically, we aimed to examine whether a well-established, 9-week group cognitive training program, Goal Management Training, results in a significant improvement in performance in the cognitive domains of executive function, attention and memory. We hypothesized that GMT would lead to significant improvements in performance on tasks related to planning, organization and attention from pre- to post-treatment relative to no such improvements in waitlist controls over the same time period. Further, we expected that the GMT group will show significant improvement on ratings of subjective cognition and of functional outcomes, relative to waitlist controls.

Method

Participants

Nineteen (N = 19) participants with a principal diagnosis of DSM-5 OCD were recruited from the Anxiety Treatment and Research Clinic at St. Joseph's Healthcare Hamilton. Inclusion criteria included: 1) between the ages of 18 and 60 years; 2) experiencing clinically significant obsessive-compulsive symptoms based on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), score > 17; 3) if on medications for OCD, on stable dose for a minimum of 8 weeks prior to initiation of the study; 4) must not have completed > 8 sessions of CBT for OCD in the last 6 months, and must refrain from participation in CBT throughout the duration of the study; and 5) are able to provide written informed consent. Exclusion criteria include: 1) a concurrent diagnosis of a severe mood disorder, schizophrenia or other psychotic disorders, or substance abuse/dependence; 2) suspected organic pathology; 3) active comorbid medical condition that might require urgent intervention during the course of treatment; and 4) a history of traumatic

brain injury or concussion/loss of consciousness. Clinical and demographic characteristics of the study sample are described in Table 1. There were no significant differences between the waitlist control and active group participants at baseline on any demographic variables. The mean WTAR raw score was 41.6 (SD = 6.7) and the mean WTAR standard score was 113.2 (SD = 10.4) indicating slightly above average estimates of pre-morbid intelligence at baseline for the combined active treatment group and waitlist group. The mean Y-BOCS score was 21.2 (SD = 5.6) in the entire sample.

Experimental Design and Procedure

Participants were assigned randomly to receive: 1) a 9-week structured cognitive remediation program, GMT; or 2) a 9-week waitlist condition. Participants were assessed at baseline, post-treatment, and 3-month follow-up. The experimental design is a 2 (treatment condition) by 3 (assessment phase) repeated-measures factorial design. Participants randomized to the waitlist condition were informed that they would have the opportunity for therapist-led GMT group treatment at the end of the study.

Participants were introduced to the study by research staff at the point of referral to our clinic and those interested in hearing a detailed explanation were invited to speak with a research assistant to review the study in detail and obtain informed consent. Baseline assessments were completed within 14 days of initial contact. At study entry, participants completed a battery of symptom and subjective cognition measures. Participants also completed neuropsychological testing to assess executive functioning, attention, and memory (see below), as well as several functional outcome measures. Trained researchers at the graduate level or higher administered

the neuropsychological testing. All measures were completed/administered at baseline, post-treatment and 3-month follow-up.

Study Conditions

Goal Management Training (GMT). GMT is a structured, short-term, present-oriented cognitive remediation program with an emphasis on mindfulness and practice in planning and goal-oriented behaviours. The primary objective of GMT is to train patients to stop ongoing behaviour in favour of executive control in order to define goal hierarchies and monitor performance. This is achieved through nine weekly two-hour sessions, including instructional material, interactive tasks, discussion of patients' real-life deficits, and homework assignments. Each of the nine GMT sessions is detailed further in Table 2. Mindfulness meditation is also incorporated for the purpose of developing the skill of bringing one's mind to the present to monitor ongoing behaviour, goal states, and the correspondence between them. The program also incorporates real-life examples provided by the group facilitator and the participants to illustrate goal attainment failures and successes, as well as in-session practice on complex tasks that mimic real-life tasks that are problematic for individuals with executive function deficits (such as planning and set-shifting tasks).

In the present study, participants who terminated treatment before completing 55% (or 5/9) of the GMT sessions were considered "drop-outs."

Waitlist Control Group. Individuals randomized to this group were required to wait nine weeks without participating in traditional CBT (or other psychotherapy) for OCD, and, if on medication, must remain on stable dosage for the duration of the study. Upon completion of the follow-up assessment, participants were invited to commence GMT at our clinic.

Measures and Materials

Symptom Measures. *Yale-Brown Obsessive Compulsive Scale* (Y-BOCS;Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989).The Y-BOCS is a standardized rating scale measuring 10 items pertaining to obsessions and compulsions on a 5-point Likert scale ranging from 0 (no symptoms) to 4 (severe symptoms). Both the self-report and clinician interview versions of the Y-BOCS have been shown to possess high internal consistency and validity.

Depression, Anxiety and Stress Scales (DASS-21; Lovibond & Lovibond, 1995). The DASS-21 is a set of three self-report scales designed to measure depression, anxiety and stress. Each subscale consists of seven items rated on a scale from 0 (did not apply at all) to 3 (applied to me very much).

Subjective Cognition. Participants completed three, brief self-report measures addressing cognitive performance. The *Cognitive Failures Questionnaire* (CFQ;Broadbent, Cooper, FitzGerald, & Parkes, 1982) captures daily errors in distractibility, blunders, names, and memory. The *Dysexecutive Questionnaire* (DEX; Burgess, Alderman, Evans, Wilson, & Emslie, 1996) involves self- and informant-ratings of inhibition, positive and negative affect, memory and intention. The *Memory and Cognitive Confidence Scale* (MACCS;Nedeljkovic & Kyrios, 2007) involves several questions about confidence in one's own memory and was developed for use in OCD.

Functional Outcome Measures. Participants completed several self-report measures assessing functional outcomes. The *WHO Disability Assessment Scale 2.0* (WHODAS) is a 36-item questionnaire which assesses an individual's ratings of their own performance across

domains of cognition, mobility, self-care, getting along, life activities and participation in social activities (Ustun et al., 2010). The *Illness Intrusiveness Rating Scale* (IIRS; Devins, 2010) is a 13-item instrument providing ratings of quality of life over three domains including relationships and personal development, intimacy and instrumentals. Finally, the *Sheehan Disability Scale* (SDS) is a brief measure of disability in work, social relationships and family life (Sheehan, 1983).

Neuropsychological Assessment. Here, we assessed several, separable cognitive domains that are sensitive to OCD. Attention and response inhibition were measured using *Conners' Continuous Performance Task* (CPT; Conners, 2000). The *Stroop Colour and Word Test* (Golden, 1976) assesses processing speed (colour and word reading) and sensitivity to suppress habitual responses (interference trial). The *Tower of London* (TOL; Culbertson & Zillmer, 2000) task requires participants to match a pattern on a board with three pegs of different sizes, and involves aspects of planning, organization and problem-solving. Verbal memory was assessed using the *California Verbal Learning Test – Second Edition* (CVLT-II; Delis et al., 2000), which provides indices of immediate and delayed memory performance, interference learning and recognition. The *Wechsler Test of Adult Reading* (WTAR; Wechsler, 2001) was used to estimate pre-morbid intellectual functioning.

Qualitative Interview. A qualitative exit interview designed for this study was implemented to gather participant opinions of the GMT program and protocol. This interview was conducted at post-treatment and consisted of a mixture of 11 open- and closed-ended questions concerning opinions about various aspects of the program and how helpful participants found it.

Data Analysis

Only six of nineteen (32%) of participants completed the three-month follow-up assessment, with the primary reason for low rate of completion being refusal to return for the final assessment visit. Accordingly, the primary outcome analyses for this study were the analyses measuring change from pre- to post-treatment. Repeated measures ANOVAs were used for all outcome variables with estimates of partial eta-squared for effect size (interpreted conservatively as small = .01, medium = .10, and large = .25). Simple main effects are reported for those results where an interaction was significant. To avoid further risk of type I error due to multiple comparisons, 2x3 ANOVAs for follow-up data were only completed for significant pre/post interactions or main effects of group. Results from the qualitative exit interview were summarized using percentages. All analyses were conducted using IBM SPSS version 23.

Results

Neuropsychological Assessment

Means and standard deviations for significant results are presented in Table 3; a full list of all variables tested is presented in Table 4.

Planning and Problem-Solving. There was a significant Group X Time interaction for the total number of problems solved correctly on the Tower of London ($F(1,17) = 4.6$, $p = .047$) as well as a main effect of Time ($F(1,17) = 20.7$, $p < .001$). Simple main effect analysis revealed that whereas the GMT group showed a significant improvement from baseline to post-treatment ($F(1,17) = 22.6$, $p < .001$, $\eta^2_p = .571$), the waitlist group did not ($F(1,17) = 2.8$, $p = .108$, $\eta^2_p = .145$). There was also a Group X Time interaction for TOL initiation time ($F(1,17) = 18.4$, $p < .001$) with simple main effects revealing an increase in initiation time from baseline to post-

treatment in the treatment group (*F(1,17)* = 8.1, *p* = .011, η^2_p = .324) but not in the waitlist controls (*F(1,17)* = .48, *p* = .83, η^2_p = ,003).

Attention and Processing Speed. The number of commission errors on the CPT was lower at post-treatment compared to baseline for both groups, represented by a marginally significant main effect of Time (*F(1,16)* = 3.9, *p* = .06, η^2_p = .199). There was also a significant Group X Time interaction for Hit Reaction Time (*F(1,16)* = 47.1, *p* = .017). Simple main effects revealed that there was a significant reduction in reaction time to correct responses for the GMT group (*F(1,16)* = 4.8, *p* = .043, η^2_p = .232) but not for those in the waitlist control group (*F(1,16)* = .003, *p* = .96, η^2_p = .000). No additional differences emerged on the CPT.

Verbal Memory. There was a main effect of time for number of correct responses on CVLT Trial 1 (*F(1,17)* = 9.2, *p* = .007, η^2_p = .351) and Long Delay Free Recall (*F(1,17)* = 10.2, *p* = .005, η^2_p = .375), indicating improved performance for both groups from baseline to post-treatment. No other significant differences emerged on the CVLT.

Cognitive Interference. The Stroop Task did not yield any significant differences at a critical level of α = .05. There were, however, trends towards a main effect of Group (*F(1,17)* = 3.3, *p* = .085, η^2_p = .165) and of Time (*F(1,17)* = 3.3, *p* = .084, η^2_p = .165) for the Colour-Word trial, with both groups improving over time and the waitlist group having slightly higher scores at both time points (see Table 3).

Functional Outcomes

There was a trend towards Group X Time interactions on the Work (*F(1,17)* = 3.6, *p* = .076, η^2_p = .173), Social (*F(1,17)* = 3.8, *p* = .066, η^2_p = .185), and Family (*F(1,17)* = 3.6, *p* = .073, η^2_p = .176) subscales of the Sheehan Disability Scale. Simple main effects were not

calculated for these marginally significant results, however, means and SDs displayed in Table 3 illustrate that whereas participants' scores on these disability subscales generally trended downward from baseline to post-treatment in the GMT group they tended to increase over time in the waitlist control group.

There were significant Group X Time interactions for the Instrumental subscale ($F(1,17)$ = 3.6, p = .076, η^2_p = .173) and Total Score ($F(1,17)$ = 3.6, p = .076, η^2_p = .173) of the IIRS. Here, simple main effects revealed that whereas the scores of individuals in the GMT group improved significantly from baseline to post-treatment on the IIRS Instrumental subscale ($F(1,17)$ = 3.9, p = .046, η^2_p = .188) no such improvement was observed in the waitlist control group ($F(1,17)$ = 1.5, p = .236, η^2_p = .082). This pattern was also observed for the IIRS Total Score (GMT: $F(1,17)$ = 6.8, p = .018, η^2_p = .286; waitlist: $F(1,17)$ = .727, p = .406), η^2_p = .041.

The WHODAS 2.0 total score measuring subjective ratings of overall functioning in daily activities decreased significantly for both groups (main effect of Time ($F(1,17)$ = 5.2, p = .036, η^2_p = .234). There was also a significant main effect of Time ($F(1,17)$ = 4.8, p = .042, η^2_p = .222) and a significant Group X Time interaction ($F(1,17)$ = 4.8, p = .042, η^2_p = .222) for the Understanding subscale, which contains items related to cognition, and understanding while communicating with others. Whereas simple main effects revealed a significant decrease in scores over time for the GMT group ($F(1,17)$ = 10.2, p = .005, η^2_p = .376) no such improvement emerged in the waitlist group ($F(1,17)$ = 0, p = 1.0, η^2_p = .000).

Subjective Cognition Measures

There were significant main effects of Group and Time for the MACCS Total Score (Group: $F(1,17)$ = 8.2, p = .011, η^2_p = .222; Time: ($F(1,17)$ = 7.9, p = .012, η^2_p = .222) and the

General Memory subscale (Group: $F(1,17) = 8.9$, $p = .008$, $\eta^2_p = .222$; Time: $F(1,17) = 11.7$, $p = .003$, $\eta^2_p = .222$), revealing higher scores in the GMT group, and an improved scores in both groups over time.

There was also a significant Group X Time interaction ($F(1,17) = 9.2$, $p = .007$) and main effect of Time ($F(1,17) = 6.4$, $p = .022$) for the CFQ total score. Simple main effects revealed that overall ratings of subjective cognition improved significantly over the course of treatment for the GMT group ($F(1,17) = 16.3$, $p = .001$, $\eta^2_p = .490$) but not the waitlist control group ($F(1,17) = .121$, $p = .732$, $\eta^2_p = .007$). There were no significant differences on the DEX.

Symptom Severity

There were no differences observed between time points or groups on either the Y-BOCS or the DASS-21 (total and subscale scores).

Three-Month Follow-Up Data

Follow-up data were only available for $N = 6$ participants (three waitlist and three GMT). No additional differences were seen when a set of 2x3 ANOVAs were run for these participants. When carrying forward the significant results from the 2x2 model to the 2x3, all significant main effects became insignificant.

Qualitative Exit Interview

All of the participants who completed the GMT program ($N = 9$) completed the qualitative interview. Of these participants, two (22%) stated that the program was helpful for reducing OCD symptoms, while five (56%) responded that the program helped them feel better in day-to-day activities. When asked which aspect of the program they found most helpful, seven

(78%) reported the STOP! Technique (learning to take time to think before acting) and all (100%) participants reported that the mindfulness techniques were the least helpful. Providing feedback on the program, eight individuals (89%) stated that the content was too simple or slow-moving. Participants also reported that the homework assignments were helpful for applying the techniques to real-life situations (78%) but 67% responded that they did not fully commit to practicing the assignments at home. When asked what could be done, if anything, to make the program more effective, 78% stated that the material could be condensed into fewer sessions and 67% requested that there be more OCD-specific content. All of the participants reported that they felt they would be able to apply the skills from this program to their everyday lives. Seven of nine participants stated that they planned to continue using the GMT skills in future (the other two said "probably") while only 33% reported plans to continue mindfulness practice. Three participants stated that they would recommend this program to others, while five of the remaining participants said they would recommend the program if someone was experiencing difficulty with memory or concentration.

Discussion

The results from this study indicate that GMT has the potential to serve as an effective cognitive remediation program for individuals with OCD. Analyses showed focused, significant improvements in cognitive functioning for individuals in the GMT program relative to those in the waitlist control group. Specifically, improvements in performance on neuropsychological tasks assessing planning and impulsivity (TOL initiation time), problem-solving (TOL total problems solved correctly), and inattention and processing speed (CPT hit reaction time) were observed for the GMT group from pre- to post-treatment but not for those in the waitlist control group. These results are in keeping with the emphasis placed by GMT on planning, problem-

solving and attention, executive functioning subdomains putatively affected in this population. Subjective report indicated that participants in the GMT group reported a decrease in how severely their OCD symptoms affected their lives both overall (IIRS total score) and on items related to daily functioning (IIRS Instrumental subscale), as well as improved outcomes related to daily cognitive functioning (WHODAS Understanding subscale) and general day-to-day tasks (WHODAS Total Score).

Critically, given that no significant differences were observed for any symptom measures in the active treatment and waitlist group, it seems likely that the functional improvements seen here are attributable primarily to the cognitive remediation program itself. Notably, there were marginally significant improvements from pre- to post-treatment for GMT relative to waitlist for each of the Work, Social and Family subscales of the Sheehan Disability Scale, suggesting reductions in subjective disability across these three areas. Finally, the GMT group only showed significant improvement in subjective cognition as rated by the Cognitive Failures Questionnaire. Although we failed to find that positive effects of GMT were maintained at follow-up, it is probable that this negative finding stemmed from the small number of participants (N = 6) available for follow-up testing.

Taken together, the findings reported here support the “top-down” nature of the Goal Management Training protocol in that those variables showing significant effects due to treatment were related largely to executive control systems. Moreover, the performance gains observed are clinically meaningful given the typical presentation of cognitive deficits in the areas of response inhibition, decision-making, task switching and planning observed in individuals with OCD, as proposed by the CSTC model (Pauls, Abramovitch, Rauch, & Geller, 2014). Here, given the potential association between cognitive dysfunction and reduced treatment response to

pharmacological and non-pharmacological interventions in OCD (D'Alcante et al., 2012; Flessner et al., 2010; Fontenelle et al., 2001; Muscatello et al., 2011) it is possible that cognitive interventions such as GMT may be best positioned prior to the onset of standard behavioural approaches such as CBT, thus enhancing the potential to benefit from these standard approaches.

Results of the qualitative exit interview revealed that although participants seemed generally to enjoy the program and noticed subjective benefits, they also felt that the program was too long and the material was slow moving. Here, cognitive deficit observed in individuals with OCD—despite small-to-medium effects reported for some domains in meta-analyses (Abramovitch et al., 2013; Snyder et al., 2015)—tend to be subtle. Thus, gains attributable to GMT are likely to be perceived as equally subtle by participants, thus increasing the likelihood that patients take more notice of, for example, the repetition of concepts. Given that GMT remains one of few established cognitive remediation protocols available, addressing the pacing and content layout of the program to be more specific to this particular population would make it an excellent candidate for future therapeutic and research applications in OCD.

Here, results of pre- and post-test measurements of neuropsychological functioning are reported using normative values. Inspection of the mean standard scores or T-scores in Table 3 reveals that for those neuropsychological domains showing improvement, participant scores tended to improve from the average to high-average range of performance. Here, the GMT group showed an improvement from the average to high average performance for the total number of problems solved correctly on the Tower of London that was accompanied by a corresponding increase in problem initiation time (high average to superior performance) reflecting increased inhibitory control). Scores on the Stroop task colour-word trial were also seen to increase from low average to average in the GMT group. Similarly, for sustained attention, for the CPT mean

hit reaction time, the GMT group's scores changed from slow to average compared to no change for the waitlist group.

Interestingly, a recent study by Moritz et al. (2017) suggests that poor neuropsychological performance in OCD may be mediated, in part, by obsessive-compulsive symptoms—for example, an individual with symmetry obsessions might take longer to perform a task not because of neuropsychological impairment but because of the need to achieve symmetry with the items used in that task and a decrease in motivation, two domains not measured in this study. Notably however, performance time *increased* on a measure of inhibitory control (Tower of London initiation time) in the GMT group only, suggesting that a simple increase in reaction time on neuropsychological measures cannot account solely for poor pre-intervention performance in this OCD sample.

Further inspection of Table 3 reveals that although the GMT and wait list groups did not differ statistically on any objective measures at baseline, nor were there any significant main effects of group only (though attention should be drawn to the main effects of group and time for the MACCS), participants in the GMT group appeared to endorse higher subjective ratings of cognitive and functional impairment on most measures. Here, individuals who agreed to participate in the study after initial recruitment and randomization (i.e., the GMT group) may have exhibited greater treatment-seeking behaviour, potentially enhancing treatment gains. By contrast, only three of the waitlist participants sought placement in a GMT group following completion of their final assessment, pointing towards decreased treatment motivation.

On balance, participants in the treatment group showed improvements in planning problem solving, impulsivity/inhibitory control, sustained attention and verbal memory that were

not observed in a matched wait group. Critically, these treatment gains were accompanied by subjective reports of improved cognitive functioning on measures with items primarily pertaining to executive function and general memory. The observed translation of gains in neuropsychological performance to improvements in functioning is in keeping with the proposed generalizability of GMT, suggesting that cognitive improvements stemming from participation in the group are linked to improved performance on everyday tasks required for successful functioning (e.g., at home and at work). Participation in the GMT group was also associated with broadly positive responses at the exit interview, pointing again to its potential utility in an OCD sample. Future work, however, is urgently required to identify the mechanisms by which this protocol leads to the cognitive, functional and subjective improvements observed in the present study. It is notable here that corresponding improvements were not observed in core symptoms of OCD, suggesting that improvements in disease severity cannot account for the neuropsychological, functional and subjective gains observed here.

It is unlikely that cognitive remediation will be necessary for all individuals diagnosed with OCD, but given the promising results seen in this early investigation, it appears appropriate that it be offered to those who report subjectively impairment in daily cognitive functioning. Future approaches may also involve fewer, condensed sessions and might serve most effectively as a primer to, or in conjunction with current standard cognitive behavioural therapies. Given that Goal Management Training is already an established protocol and results of the exit interview from this feasibility study showed that participants tended to enjoy the program content, the current program would likely provide the foundations for an adapted cognitive remediation protocol with a brief structure and more OCD-specific material.

There are several limitations to this study that may serve to inform future investigation. Despite lacking power, this small pilot sample served to adequately demonstrate the potential positive effects of cognitive remediation in this population while also pointing towards changes that may be necessary to the program structure and content. One major limitation is the potential for type I error due to the large number of multiple comparisons (refer to Table 4 for a list of all measures included in the assessment), a problem often inherent with use of neuropsychological tasks involving multiple subscales—although it can be argued that many of these variables represent distinct processes at different levels of functioning. In addition, the majority of our sample had recently completed a course of cognitive behavioural therapy for OCD and 74% were taking medication for OCD symptoms. The findings presented here, therefore, might not be generalizable to a wider or treatment-naïve population. However, we would expect that, if anything, a sample not treated previously would be more likely to show greater improvement in functioning over the course of treatment. Finally, due to resource and personnel limitations, full blinding was not always feasible. Where possible, the research team completed assessments by individuals blind to participant condition but when this was not the case there was potential for bias to have affected final results.

The results of this pilot study are in line with our predictions surrounding the potential benefits of GMT in individuals with OCD and align closely with previous studies of GMT in various populations in that we observed focused, specific improvements in neuropsychological performance on variables of planning, problem-solving, impulsivity and attention, and general improvements in ratings of subjective cognition and daily functioning, particularly on domains related to cognition/understanding and instrumental daily functioning. The Goal Management Training protocol in its current form is likely delivered over more sessions than necessary for

most individuals with OCD, but will serve as an excellent base for the adaptation or development of a new protocol with material specifically tailored toward this population. These encouraging findings can inform future research, which should investigate a more condensed cognitive remediation format with larger sample and rigorous study design. Future study should also consider the addition of an active control group in order to determine specific effects of the cognitive remediation program relative to other treatment options. Though likely not necessary for all individuals with OCD, the findings of this preliminary investigation suggest that the utility of cognitive remediation strategies should be investigated as they may confer significant gains for interested individuals particularly those who are experiencing difficulty with cognitive function.

References

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (DSM-5). Arlington, VA: American Psychiatric Publishing.

Abramovitch, A., & Cooperman, A. (2015). The cognitive neuropsychology of obsessive-compulsive disorder: A critical review. *Journal of Obsessive-Compulsive and Related Disorders, 5*, 24-36.

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: a meta-analysis. *Clinical psychology review, 33*(8), 1163-1171.

Abramovitch, A., Dar, R., Schweiger, A., & Hermesh, H. (2011). Neuropsychological impairments and their association with obsessive-compulsive symptom severity in obsessive-compulsive disorder. *Archives of Clinical Neuropsychology, 26*(4), 364-376.

Alfonso, J. P., Caracuel, A., Delgado-Pastor, L. C., & Verdejo-García, A. (2011). Combined goal management training and mindfulness meditation improve executive functions and decision-making performance in abstinent polysubstance abusers. *Drug and alcohol dependence, 117*(1), 78-81.

Anderson, K. E., & Savage, C. R. (2004). Cognitive and neurobiological findings in obsessive–compulsive disorder. *Psychiatric Clinics of North America, 27*(1), 37-47.

Attiullah, N., Eisen, J. L., & Rasmussen, S. A. (2000). Clinical features of obsessive-compulsive disorder. *Psychiatric Clinics of North America, 23*(3), 469-491.

Bannon, S., Gonsalvez, C. J., Croft, R. J., & Boyce, P. M. (2006). Executive functions in obsessive–compulsive disorder: state or trait deficits? *Australian and New Zealand Journal of Psychiatry, 40*(11-12), 1031-1038.

Broadbent, D. E., Cooper, P. F., FitzGerald, P., & Parkes, K. R. (1982). The cognitive failures questionnaire (CFQ) and its correlates. *British Journal of Clinical Psychology, 21*(1), 1-16.

Brundtland, G. H. (2000). Mental health in the 21st century. *Bulletin of the World Health Organization, 78*(4), 411-411.

Buhlmann, U., Deckersbach, T., Engelhard, I., Cook, L. M., Rauch, S. L., Kathmann, N., . . . Savage, C. R. (2006). Cognitive retraining for organizational impairment in obsessive–compulsive disorder. *Psychiatry research, 144*(2), 109-116.

Burgess, P. W., Alderman, N., Evans, J. J., Wilson, B. A., & Emslie, H. (1996). The dysexecutive questionnaire. In B. A. Wilson, N. Alderman, P. W. Burgess, H. Emslie & J. J. Evans (Eds.), *Behavioural Assessment of the Dysexecutive Syndrome*. Bury St. Edmunds: Thames Valley Test Company.

Cassin, S. E., Richter, M. A., Zhang, K. A., & Rector, N. A. (2009). Quality of life in treatment-seeking patients with obsessive-compulsive disorder with and without major depressive disorder. *Canadian Journal of Psychiatry, 54*(7), 460.

Cavedini, P., Zorzi, C., Piccinni, M., Cavallini, M. C., & Bellodi, L. (2010). Executive dysfunctions in obsessive-compulsive patients and unaffected relatives: searching for a new intermediate phenotype. *Biological psychiatry, 67*(12), 1178-1184.

Chudasama, Y., & Robbins, T. (2006). Functions of frontostriatal systems in cognition: comparative neuropsychopharmacological studies in rats, monkeys and humans. *Biological psychology, 73*(1), 19-38.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. New York, NY: Academic Press.

Culbertson, W. C., & Zillmer, E. (2001). *Tower of London-Drexel University (TOLDX)*. Multi-Health Systems.

D'Alcante, C. C., Diniz, J. B., Fossaluza, V., Batistuzzo, M. C., Lopes, A. C., Shavitt, R. G., . . . Hoexter, M. Q. (2012). Neuropsychological predictors of response to randomized treatment in obsessive–compulsive disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 39*(2), 310-317.

de Braek, D. M. I., Dijkstra, J. B., Ponds, R. W., & Jolles, J. (2012). Goal management training in adults with ADHD: An intervention study. *Journal of attention disorders*, 1087054712468052.

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *CVLT-II: California verbal learning test: adult version.* Psychological Corporation.

Devins, G. M. (2010). Using the illness intrusiveness ratings scale to understand health-related quality of life in chronic disease. *Journal of psychosomatic research, 68*(6), 591-602.

Eddy, K. T., Dutra, L., Bradley, R., & Westen, D. (2004). A multidimensional meta-analysis of psychotherapy and pharmacotherapy for obsessive-compulsive disorder. *Clinical psychology review, 24*(8), 1011-1030.

Flessner, C. A., Allgair, A., Garcia, A., Freeman, J., Sapyta, J., Franklin, M. E., ... & March, J. (2010). The impact of neuropsychological functioning on treatment outcome in pediatric obsessive–compulsive disorder. *Depression and anxiety, 27*(4), 365-371.

Fontenelle, L. F., Mendlowicz, M. V., Mattos, P., & Versiani, M. (2006). Neuropsychological findings in obsessive-compulsive disorder and its potential implications for treatment. *Current Psychiatry Reviews, 2*(1), 11-26.

Fontenelle, L., Marques, C., Engelhardt, E., & Versiani, M. (2001). Impaired set-shifting ability and therapeutic response in obsessive-compulsive disorder. *The Journal of neuropsychiatry and clinical neurosciences, 13*(4), 508-510.

Golden, C. J. (1976). Identification of brain disorders by the Stroop Color and Word Test. *Journal of clinical psychology, 32*(3), 654-658.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Delgado, P., Heninger, G. R., & Charney, D. S. (1989). The yale-brown obsessive compulsive scale: II. *Validity. Archives of general Psychiatry, 46*(11), 1012-1016.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., . . . Charney, D. S. (1989). The Yale-Brown obsessive compulsive scale: I. Development, use, and reliability. *Archives of general Psychiatry, 46*(11), 1006-1011.

Greisberg, S., & McKay, D. (2003). Neuropsychology of obsessive-compulsive disorder: a review and treatment implications. *Clinical psychology review, 23*(1), 95-117.

Kang, D. H., Kwon, J., Kim, J. J., Youn, T., Park, H. J., Kim, M., . . . Lee, M. (2003). Brain glucose metabolic changes associated with neuropsychological improvements after 4

months of treatment in patients with obsessive–compulsive disorder. *Acta Psychiatrica Scandinavica, 107*(4), 291-297.

Karno, M., Golding, J. M., Sorenson, S. B., & Burnam, M. A. (1988). The epidemiology of obsessive-compulsive disorder in five US communities. *Archives of general Psychiatry, 45*(12), 1094.

Kathmann, N. (2008). *Neuropsychologie der Zwangsstörungen (Neuropsychology of Obsessive-Compulsive Disorder)*. Göttingen: Hogrefe Verlag.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of general Psychiatry, 62*(6), 593-602.

Kim, M.-S., Park, S.-J., Shin, M. S., & Kwon, J. S. (2002). Neuropsychological profile in patients with obsessive-compulsive disorder over a period of 4-month treatment. *Journal of psychiatric research, 36*(4), 257-265.

Koran, L. M., Thienemann, M. L., & Davenport, R. (1996). Quality of life for patients with obsessive-compulsive disorder. *The American journal of psychiatry, 153*(6), 783.

Krasny-Pacini, A., Limond, J., Evans, J., Hiebel, J., Bendjelida, K., & Chevignard, M. (2014). Context-sensitive goal management training for everyday executive dysfunction in children after severe traumatic brain injury. *The Journal of head trauma rehabilitation, 29*(5), E49-E64.

Kuelz, A. K., Riemann, D., Halsband, U., Vielhaber, K., Unterrainer, J., Kordon, A., & Voderholzer, U. (2006). Neuropsychological impairment in obsessive-compulsive

disorder—Improvement over the course of cognitive behavioral treatment. *Journal of clinical and experimental Neuropsychology, 28*(8), 1273-1287.

Lennertz, L., Rampacher, F., Vogeley, A., Schulze-Rauschenbach, S., Pukrop, R., Ruhrmann, S., . . . Wagner, M. (2012). Antisaccade performance in patients with obsessive–compulsive disorder and unaffected relatives: further evidence for impaired response inhibition as a candidate endophenotype. *European archives of psychiatry and clinical neuroscience, 262*(7), 625-634.

Levine, B., Robertson, I. H., Clare, L., Carter, G., Hong, J., Wilson, B. A., . . . Stuss, D. T. (2000). Rehabilitation of executive functioning: An experimental–clinical validation of goal management training. *Journal of the International Neuropsychological Society, 6*(03), 299-312.

Levine, B., Schweizer, T. A., O'Connor, C., Turner, G., Gillingham, S., Stuss, D. T., . . . & Robertson, I. H. (2011). Rehabilitation of executive functioning in patients with frontal lobe brain damage with goal management training. *Frontiers in Human Neuroscience, 17*(5), 9.

Levine, B., Stuss, D. T., Winocur, G., Binns, M. A., Fahy, L., Mandic, M., . . . Robertson, I. H. (2007). Cognitive rehabilitation in the elderly: effects on strategic behavior in relation to goal management. *Journal of the International Neuropsychological Society, 13*(01), 143-152.

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour research and therapy, 33*(3), 335-343.

Masellis, M., Rector, N. A., & Richter, M. A. (2003). Quality of life in OCD: differential impact of obsessions, compulsions, and depression comorbidity. *Canadian Journal of Psychiatry, 48*(2), 72-77.

Muller, J., & Roberts, J. E. (2005). Memory and attention in obsessive–compulsive disorder: a review. *Journal of anxiety disorders, 19*(1), 1-28.

Muscatello, M. R. A., Bruno, A., Pandolfo, G., Micò, U., Scimeca, G., Romeo, V. M., ... & Zoccali, R. A. (2011). Effect of aripiprazole augmentation of serotonin reuptake inhibitors or clomipramine in treatment-resistant obsessive-compulsive disorder: a double-blind, placebo-controlled study. *Journal of clinical psychopharmacology, 31*(2), 174-179.

National Institute for Health and Clinical Excellence. (2014). *Obsessive-compulsive disorder: Core interventions in the treatment of obsessive-compulsive disorder and body dysmorphic disorder*. London, UK: Author.

Nedeljkovic, M., & Kyrios, M. (2007). Confidence in memory and other cognitive processes in obsessive–compulsive disorder. *Behaviour Research and Therapy, 45*(12), 2899-2914.

Nielen, M., & Den Boer, J. (2003). Neuropsychological performance of OCD patients before and after treatment with fluoxetine: evidence for persistent cognitive deficits. *Psychological medicine, 33*(05), 917-925.

O'connor, C., Robertson, I. H., & Levine, B. (2011). The prosthetics of vigilant attention: Random cuing cuts processing demands. *Neuropsychology, 25*(4), 535.

Olatunji, B. O., Davis, M. L., Powers, M. B., & Smits, J. A. (2013). Cognitive-behavioral therapy for obsessive-compulsive disorder: a meta-analysis of treatment outcome and moderators. *Journal of psychiatric research, 47*(1), 33-41.

Olley, A., Malhi, G., & Sachdev, P. (2007). Memory and executive functioning in obsessive–compulsive disorder: a selective review. *Journal of affective disorders, 104*(1), 15-23.

Park, H. S., Shin, Y. W., Ha, T. H., Shin, M. S., Kim, Y. Y., Lee, Y. H., & Kwon, J. S. (2006). Effect of cognitive training focusing on organizational strategies in patients with obsessive-compulsive disorder. *Psychiatry Clin Neurosci, 60*(6), 718-726. doi: 10.1111/j.1440-1819.2006.01587.x

Pauls, D. L., Abramovitch, A., Rauch, S. L., & Geller, D. A. (2014). Obsessive-compulsive disorder: an integrative genetic and neurobiological perspective. *Nature Reviews Neuroscience, 15*(6), 410-424.

Roh, K. S., Shin, M. S., KIM, M. S., HA, T. H., SHIN, Y. W., Lee, K. J., & Kwon, J. S. (2005). Persistent cognitive dysfunction in patients with obsessive-compulsive disorder: A naturalistic study. *Psychiatry and Clinical Neurosciences, 59*(5), 539-545.

Savage, C. R., Baer, L., Keuthen, N. J., Brown, H. D., Rauch, S. L., & Jenike, M. A. (1999). Organizational strategies mediate nonverbal memory impairment in obsessive–compulsive disorder. *Biological psychiatry, 45*(7), 905-916.

Sheehan, D. V. (1983). Sheehan disability scale. *Handbook of psychiatric measures, 2*, 100-2.

Snyder, H. R., Kaiser, R. H., Warren, S. L., & Heller, W. (2015). Obsessive-compulsive disorder is associated with broad impairments in executive function: A meta-analysis. *Clinical Psychological Science, 3*(2), 301-330.

Stamenova, V., & Levine, B. (2018). Effectiveness of goal management training® in improving executive functions: A meta-analysis. *Neuropsychological rehabilitation*, 1-31.

Stubberud, J., Langenbahn, D., Levine, B., Stanghelle, J., & Schanke, A.-K. (2013). Goal management training of executive functions in patients with spina bifida: A randomized controlled trial. *Journal of the International Neuropsychological Society, 19*(06), 672-685.

Tolin, D. F., Abramowitz, J. S., & Diefenbach, G. J. (2005). Defining response in clinical trials for obsessive-compulsive disorder: a signal detection analysis of the Yale-Brown obsessive compulsive scale. *The Journal of clinical psychiatry, 66*(12), 1,478-1557.

Üstün, T. B., Chatterji, S., Kostanjsek, N., Rehm, J., Kennedy, C., Epping-Jordan, J., ... & Pull, C. (2010). Bulletin of the World Health Organization. *Bulletin of the World Health Organization, 88*, 815-823.

van Hooren, S. A., Valentijn, S. A., Bosma, H., Ponds, R. W., van Boxtel, M. P., Levine, B., . . . Jolles, J. (2007). Effect of a structured course involving goal management training in older adults: A randomised controlled trial. *Patient education and counseling, 65*(2), 205-213.

Viswanath, B., Reddy, Y. J., Kumar, K. J., Kandavel, T., & Chandrashekar, C. (2009). Cognitive endophenotypes in OCD: a study of unaffected siblings of probands with familial OCD. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 33*(4), 610-615.

Voderholzer, U., Schwartz, C., Freyer, T., Zurowski, B., Thiel, N., Herbst, N., . . . Kuelz, A. K. (2013). Cognitive functioning in medication-free obsessive-compulsive patients treated with cognitive-behavioural therapy. *Journal of Obsessive-Compulsive and Related Disorders*, *2*(3), 241-248.

Wechsler, D. (2001). *Wechsler Test of Adult Reading: WTAR*. Psychological Corporation.

Zohar, A. H. (1999). The epidemiology of obsessive-compulsive disorder in children and adolescents. *Child and Adolescent Psychiatric Clinics of North America*, *8*(3), 445-60.

Appendix I

Tables

Table 1

Sample Demographics Information

Variable	*Frequency*
Age, Mean (SD)	45.5 (12.6)
Sex (female)	55%
Education, Mean years (SD)	14.6 (2.3)
OCD Duration, Mean years (SD)	23.7 (12.4)
Medication Status, % on SSRI	73.7%
Y-BOCS Total Score, Mean (SD)	21.2 (5.6)

Legend: Y-BOCS = Yale-Brown Obsessive Compulsive Scale

Table 2

Overview of Goal Management Training Protocol

GMT Session	*Description*
Session 1: The Absent Mind, the Present Mind	Introduce the concept of absentmindedness and normalize the experience. Explain present-mindedness using mindfulness techniques.
Session 2: Absentminded Slip-Ups	Introduce construct of absentminded slips with examples, and discuss emotional and practical consequences. Introduce the “Body Scan” mindfulness exercise.
Session 3: The Automatic Pilot	Describe “automatic pilot” as being a habitual mechanism which can lead to inappropriate responses or actions if not monitored. Introduce the “Breathing Exercise” mindfulness technique.
Session 4: Stop the Automatic Pilot	Participants are introduced to the “STOP!” technique as a method of bringing one’s attention to the present to monitor current behaviour. The short “Breath Focus” mindfulness exercise is described.

Session 5: The Mental Blackboard	The construct of working memory as a "mental blackboard," which can be erased or over saturated with information, is explained. Participants are taught to check "the mental blackboard" to keep current goals at the forefront of memory. Introduce how to incorporate present-mindedness (specifically the "Breath Focus") into behaviour monitoring and executing difficult tasks as a method for increasing accuracy and memory.
Session 6: State Your Goal	Describe how goals can become entangled when attempting to multi-task. Introduce the concept of stating one's goal as a way to aid encoding and recall of that goal.
Session 7: Making Decisions	Introduce the concept of conflicting goals and detail strategies for how to make decisions. Review methods for keeping track of complex goals using to-do lists.
Session 8: Splitting Tasks into Subtasks	Practice completing tasks that are too complex to rely on working memory only, and detail strategies for how to divide large goals into a series of smaller, more manageable subgoals.
Session 9: STOP!	Review the material covered across previous sessions and underscore the importance of goal monitoring (the "STOP!" technique).

Table 3

Means and Standard Deviations (SDs) for Statistically Significant 2x2 Repeated Measures ANOVAs

		Pre-Treatment		*Post-Treatment*	
Variable	*Group*	*Mean*	*SD*	*Mean*	*SD*
Neuropsychological Assessment					
Stroop Colour-Word T-Score	GMT	42.9	6.1	48.3	7.1
(MEs of Time and Group)	WLC	50.0	8.3	51.8	8.1
TOL Total Correct SS	GMT	100.0	14.1	114.2	16.8
(Group X Time and ME of Time)	WLC	93.1	15.8	98.4	15.8
TOL Initiation Time SS	GMT	108.8	18.4	121.2	18.6
(Group X Time)	WLC	105.1	12.5	104.1	8.8
CPT Commission Errors T-Score	GMT	54.1	9.2	50.2	8.5
(ME of Time)	WLC	52.2	7.8	48.6	8.1
CPT Hit Reaction Time T-Score	GMT	60.2	11.2	53.2	7.2
(Group X Time)	WLC	52.3	7.4	52.5	8.1
Functional Outcomes					
SDS Work	GMT	3.8	2.9	3.1	2.9
(Group X Time)	WLC	3.0	1.9	3.3	2.1
SDS Social	GMT	5.1	3.4	4.5	3.5
(Group X Time)	WLC	2.9	1.7	4.0	2.1
SDS Family	GMT	4.8	2.5	4.2	2.4
(Group X Time)	WLC	3.2	2.4	4.0	2.2
IIRS Instrumental Subscale	GMT	15.3	5.3	13.6	4.8
(Group X Time)	WLC	11.4	2.9	12.6	2.9
IIRS Total Score	GMT	49.1	15.1	43.3	11.6
(Group X Time)	WLC	35.2	10.3	37.4	9.4
WHODAS 2.0 Understanding	GMT	6.7	4.0	5.4	4.0
(Group X Time and ME of Time)	WLC	6.5	2.9	6.6	3.2
Subjective Cognition					
MACCS Total Score	GMT	92.9	9.6	84.1	13.7
(MEs of Time and Group)	WLC	76.8	7.7	74.6	10.3
MACCS General Memory	GMT	44.2	8.7	38.9	8.0
(MEs of Time and Group)	WLC	33.4	6.8	29.8	7.6
CFQ	GMT	45.1	10.4	35.3	9.2
(Group X Time and ME of Time)	WLC	43.4	8.1	44.3	7.9

Note: The value in parentheses below each variable is the result provided by 2x2 repeated measures ANOVA. **Legend:** CFQ = Cognitive Failures Questionnaire; CPT = Conners' Continuous Performance Task; GMT = Goal Management Training Group; IIRS = Illness Intrusiveness Rating Scale; MACCS = Memory and Cognitive Confidence Scale; ME = Main Effect; TOL = Tower of London; WHODAS = World Health Organization Disability Assessment Schedule; WLC = Waitlist Control Group.

Table 4

Complete List of Assessment Measures

Measure
Neuropsychological Assessment
California Verbal Learning Test – Second Edition
Stroop Task
Tower of London
Conners' Continuous Performance Task – Second Edition
Wechsler Test of Adult Reading (baseline only)
Functional Outcome Measures
Sheehan Disability Scale
Illness Intrusiveness Rating Scale
WHO Disability Assessment Schedule 2.0 (36-item self-report)
Subjective Cognition Measures
Memory and Cognitive Confidence Scale
Cognitive Failures Questionnaire
Dysexecutive Questionnaire
Symptom Measures
Yale-Brown Obsessive-Compulsive Scale
Depression Anxiety and Stress Scales 21-item

CHAPTER SIX

CONCLUSIONS

Obsessive-compulsive disorder is a burdensome illness that typically runs a chronic course and is expected to persist throughout the lifetime (American Psychiatric Association; 2013). Numerous quantitative reviews provide support for the clinical efficacy of cognitive behavioural therapy (CBT) and pharmacological interventions for OCD (Eddy, Dutra, Bradley, & Westen, 2004; Olatunji, Davis, Powers, & Smits, 2013) but an important caveat to this is that a successful treatment outcome is generally defined by a 25%-50% reduction in symptom severity (Tolin, Abramowitz, & Diefenbach, 2005). Thus, there is an ongoing effort to find alternative treatment options, or methods by which standard treatments may be augmented. Two avenues through which this might be accomplished are by targeting neuropsychological impairment (if present) and by tailoring treatment dependent on symptom profile. The neuropsychology of OCD has been a topic of significant interest in past literature and despite the focus on the documented impairments, almost no research has been dedicated to treatment options for this impairment. Similarly, although there is good evidence to support the notion treating obsessive-compulsive symptom profiles individually (e.g., Frost & Steketee, 2002), the significant limitations present in much of the existing research concerning symptom dimensions in OCD have resulted in little clinical utility yielded from this work. This thesis has reviewed critically conventional subtyping methods while also providing support in the form of differential neuropsychological task performance for an understudied—and perhaps more valid—model of symptom dimensions, and has also described early evidence to show that neuropsychological deficits can be effectively ameliorated through cognitive remediation.

Study 1 of this thesis critically reviewed research on the associations between neuropsychological task performance and OCD symptom dimensions and drew attention to the prominent methodological challenges and limitations inherent in this work. The findings of this review show that there may be deficits in neuropsychological functioning associated with symptom dimensions such as aggressive/checking, contamination/cleaning, and symmetry/ordering across a number of cognitive domains, but the heterogeneity in methodology—both for subtyping strategies and in the tasks used to assess cognitive performance—necessitate caution in the interpretation of these results.

Considering the difficulty in interpreting the findings of Study 1, due primarily to the lack of consensus surrounding an ideal method for defining symptom dimensions, Study 2 aimed to compare the two most commonly used statistical methods for this venture to assess whether one technique led to more meaningful results than the other. Employing the most widely accepted processes for each of these analyses, it was observed that factor analysis likely does not lend itself well to the data captured by the Y-BOCS. Cluster analysis, although appearing superficially to be a more effective technique, does not truly identify latent categories of symptoms as it groups cases rather than variables, and thus is not directly addressing the question. It was also noted that the statistical techniques are less to blame than the nature of the data gathered from the Y-BOCS itself, in addition to the naturalistic presentation of OCD symptoms observed in most samples (i.e., less frequently observed symptom profiles leads to poor representation in the sample).

Having identified numerous conceptual and methodological issues with subtyping based on overt symptomatology alone, Study 3 sought to examine neuropsychological performance differences between core dimensions (harm avoidance versus incompleteness) of OCD, as a

means to further investigate the validity of this alternative model. It was observed that those with incompleteness symptoms had higher ratings of subjective cognitive impairment, performed worse on a task of problem-solving and planning, but demonstrated better performance on a task of verbal memory than did those with harm avoidance symptoms. This study described some of the first concrete evidence showing distinct differences between these dimensions, offering support for the core dimensions model.

Finally, the common theme throughout Studies 1 and 3 of neuropsychological performance in OCD was addressed through an assessment of the feasibility and efficacy of a cognitive remediation program. This study showed that the Goal Management Training program can lead to significant gains in performance on neuropsychological tasks of executive function, as well as improvements in ratings of subjective cognition and daily functioning. This pilot investigation also highlighted some areas of improvement for the use of this protocol in OCD, such as consolidating treatment content to reduce program length, and adding more OCD-specific content.

Implications

The first two studies of this thesis serve to inform researchers and clinicians on the difficulty—and perhaps futility—of defining distinct symptom subtypes based on overt symptom presentation given current symptom measures and statistical methods. The findings presented in this thesis have significant implications both for assessment of treatments and assessment of neuropsychological performance in OCD. It has been shown that certain types of symptoms may respond better to different treatment options (Foa et al., 1983; Frost & Steketee, 2000) but our understanding of the mechanisms underlying these differences—and indeed the differences

themselves—remains in stasis until a concrete model for symptom dimensions is reached. This issue is exemplified by the case of hoarding, which was seen to respond more effectively to treatment when it was targeted separately (Frost & Hartl, 1996; Rachman, 1997; 1998; 2002) and indeed it now stands as a distinct entity in the DSM-5—though it is noted that hoarding symptoms can exist in the context of OCD (American Psychiatric Association, 2013). Although it is likely not the case that other symptoms of OCD would constitute respective diagnoses should concrete dimensions be defined, it would be of substantial value to clinicians to know which symptoms respond best to which forms of treatment, or whether symptom profiles have varying trajectories of change. This would also greatly increase the validity of research in OCD, as it may hold true that a considerable portion of the variance observed in OCD literature, particularly that which concerns neuropsychological assessment, is attributable to differences in symptomatology—hence the call for reporting of symptoms whenever possible (Abramovitch, Mittelman, Tankersley, Abramowitz, & Schweiger, 2015). By highlighting the potential flaws in conventional subtyping methods and providing support for the validity of the core dimensions model, the findings presented in this thesis important direction for future investigations to proceed in the elucidation of OCD symptom dimensions.

Another prominent theme throughout this thesis has been the neuropsychology of OCD. The mutual relationship between OCD symptoms and neuropsychological performance has been discussed at length, but perhaps equally notable have been the findings describing the positive effects of cognitive remediation in this population. Very little research has been dedicated to examining this topic, and the few studies that do exist report the results of “bottom-up” protocols. Study 4 of this thesis details the results of the first investigation of a “top-down” approach, which emphasizes the implementation of skills and behaviours relying on executive

functions which can then be adapted to a number of situations in everyday life. As mentioned in Study 4, the efficacy of Goal Management Training has been exemplified in numerous populations (e.g., In de Braek, Dijkstra, Ponds, & Jolles, 2017; Stubberud, Langebahn, Levine, Stanghelle, & Schanke, 2015). Perhaps the most important aspect of this trial was the qualitative exit interview, which provided critical feedback for future implementations of cognitive remediation in OCD. By showing that this protocol can confer significant benefits for this population, and by providing key areas of improvement for future implementation of the treatment, this pilot work has laid the foundation for a new area of research and treatment in this chronically affected population.

Synthesis of Results for Direction of Future Research

There is considerable work yet to be done in the investigation of symptom dimensions and neuropsychology of OCD. The primary objective of this thesis has been to critically evaluate previous research in this area in order to inform future study. The finding that exploratory factor analysis was unable to provide a meaningful output inclusive of all OCD symptoms should caution researchers against using this practice in future. Furthermore, the limitations inherent with the Y-BOCS such as the use of a priori categories which are not entirely supported by item-level factor analysis (Denys, de Geus, van Megen, & Westenberg, 2004; Pinto et al., 2008; Summerfeldt, Richter, Antony, & Swinson, 1999), and the flawed practice of reducing multi-item categorical data to a single ordinal metric (Nunnally & Bernstein, 1994) should discourage future use of these strategies. Although new statistical methods such as machine learning may hold promise for the elucidation of symptom dimensions based on overt symptom presentation, factor analysis remains a strong option for answering this question. However, any future investigations seeking to do this should employ item-level analysis only, which requires a

substantially larger sample size. Perhaps with a more comprehensive obsessive-compulsive symptom checklist that addresses the few symptoms (such as mental compulsions) missing from the Y-BOCS, and with better understanding of the heterogeneous miscellaneous obsessions and compulsions items, there may be definitive answer to this issue.

As many clinicians are aware, patients with OCD rarely present with one symptom or category of symptoms alone. It may be more clinically meaningful, therefore, to assess based on symptom *theme* rather than on most troubling symptom. This is one of the potential strengths that the core dimensions model holds over conventional subtyping. Research on this topic is still preliminary, but there is support for the structural validity of the harm avoidance and incompleteness dimensions (Summerfeldt, Kloosterman, Antony, & Swinson, 2014), and the results described in Study 3 of this thesis indicate that there appear to be distinct neuropsychological differences between these groups as well. Future research should aim to corroborate these findings, and address the group assignment limitation of Study 3. Here, participants were still grouped based on current most troubling symptom, and in order to ensure validity of symptom theme, the entirety of participants' symptomatology must be considered. This necessitates the validation of symptom measures which can accurately capture this information. Although tools such as the OC-CDI and OC-CDQ exist, they remain understudied and are not yet commercially available. In the meantime, the more labour intensive process of reviewing a complete picture of each participant's symptoms and the motivations underlying them will likely be the only accurate way to group participants for core dimensions.

Future study of these dimensions should employ larger and more evenly distributed samples than in Study 3. The implementation of a more comprehensive neuropsychological battery would in turn allow for a broader examination of the cognitive differences between these

two groups. As seen in the results of the review in Study 1, and based on the heterogeneous findings of meta-analyses of neuropsychology in OCD (e.g., Abramovitch, Ambramowitz, & Mittelman, 2013), future study of cognitive performance in this population—both in dimensions and in general—should aim to replicate previous findings and should attempt to employ the same neuropsychological tasks as previous studies to improve consistency. Finally, recent studies by Moritz et al. (Moritz, Hauschildt, Saathoff, & Jelinek, 2017; Moritz, Spirandelli, Happach, Lion, & Berna, 2018) indicate that several factors such as motivation, OCD symptoms, and stereotype threat might mediate neurocognitive impairment in affected individuals. Consequently, these factors should be assessed for in any future study of neuropsychology in OCD—particularly in the context of studies offering treatment or substantial reimbursement to participants as these factors might affect an individual's willingness to give a valid effort on all tasks.

Despite the enduring uncertainty surrounding the neuropsychology of OCD, the consistently identified subdomains of executive function reported across meta-analyses (Abramovitch et al., 2015; Shin, Lee, Kim, & Kwon, 2014) taken together with the notion that certain cognitive skills may predict response to cognitive behavioural therapy (D'Alcante et al., 2012) provide a strong argument for the use of cognitive remediation—at least for patients who are experiencing trouble with cognitive performance. Future study of this topic should adopt the recommendations from Study 4 of this thesis either to revise Goal Management Training specifically for use in OCD, or should draft a new cognitive remediation protocol based on a similar curriculum. Once a revised protocol has been validated, its implementation should be assessed both as a stand-alone treatment and as a primer to cognitive behavioural therapies to assess whether its use can augment treatment outcomes from standard approaches. Finally, due to the pilot structure of Study 4, it was not possible to assess outcomes by symptom dimension

with adequate power. Future investigations of this topic with larger samples should assess for symptom dimensions to determine whether dimensions are associated with different outcome trajectories.

Conclusions

The investigation of conventional symptom-based subtypes of OCD is fraught with conceptual and methodological challenges and limitations. Although there may indeed be validity to this model, the definition of concrete subtypes or dimensions remains elusive. The early research of the core dimensions model shows promise for this approach as a valid and meaningful method of defining dimensions. Finally, whether assessed within the context of symptom profiles or in the general OCD population, the use of cognitive remediation to target executive functioning processes appears both feasible and effective.

References

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive–compulsive disorder: a meta-analysis. *Clinical psychology review*, *33*(8), 1163-1171.

Abramovitch, A., Mittelman, A., Tankersley, A. P., Abramowitz, J. S., & Schweiger, A. (2015). Neuropsychological investigations in obsessive–compulsive disorder: A systematic review of methodological challenges. *Psychiatry research*, *228*(1), 112-120.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.

Eddy, K. T., Dutra, L., Bradley, R., & Westen, D. (2004). A multidimensional meta-analysis of psychotherapy and pharmacotherapy for obsessive-compulsive disorder. *Clinical Psychology Review*, *24*(8), 1011-1030

Foa, E. B., Grayson, J. B., Steketee, G. S., Doppelt, H. G., Turner, R. M., & Latimer, P. R. (1983). Success and failure in the behavioral treatment of obsessive–compulsives. *Journal of Consulting and Clinical Psychology*, *51*, 287–297.

Frost, R.O., & Hartl, T.L. (1996). A cognitive behavioral model of hoarding. *Behavior Research and Therapy*, *31*, 367-381.

Frost, R.O., & Steketee, S. (Eds.)(2000). *Cognitive approaches to obsessions and compulsions: Theory, assessment and treatment*. Elsevier: Oxford.

In de Braek, D. M., Dijkstra, J. B., Ponds, R. W., & Jolles, J. (2017). Goal management training in adults with ADHD: An intervention study. *Journal of Attention Disorders*, *21*(13), 1130-1137.

Moritz, S., Hauschildt, M., Saathoff, K., & Jelinek, L. (2017). Does impairment in neuropsychological tests equal neuropsychological impairment in obsessive-compulsive disorder (OCD)? Momentary influences, testing attitude, and motivation are related to neuropsychological performance in OCD. *Journal of obsessive-compulsive and related disorders*, *14*, 99-105.

Moritz, S., Spirandelli, K., Happach, I., Lion, D., & Berna, F. (2018). Dysfunction by Disclosure? Stereotype Threat as a Source of Secondary Neurocognitive Malperformance in Obsessive-Compulsive Disorder. *Journal of the International Neuropsychological Society*, 1-9.

Nunnally, J.C., Bernstein, I.H. (1994). *Psychometric theory* (3rd ed.). Toronto: McGraw-Hill.

Olatunji, B. O., Davis, M. L., Powers, M. B., & Smits, J. A. (2013). Cognitive-behavioral therapy for obsessive-compulsive disorder: a meta-analysis of treatment outcome and moderators. *Journal of Psychiatric Research*, *47*(1), 33-41.

Pinto, A., Greenberg, B. D., Grados, M. A., Bienvenu III, O. J., Samuels, J. F., Murphy, D. L., ... & Pauls, D. L. (2008). Further development of YBOCS dimensions in the OCD Collaborative Genetics study: symptoms vs. categories. *Psychiatry research*, *160*(1), 83-93.

Rachman, S. (1997). A cognitive theory of obsessions. *Behaviour Research and Therapy*, *35*, 793-802.

Rachman, S. (1998). A cognitive theory of obsessions: Elaborations. *Behaviour Research and Therapy, 36*, 385-401.

Rachman, S. (2002). A cognitive theory of compulsive checking. *Behaviour Research and Therapy, 40*, 624-639.

Shin, N. Y., Lee, T. Y., Kim, E., & Kwon, J. S. (2014). Cognitive functioning in obsessive-compulsive disorder: a meta-analysis. *Psychological medicine*, *44*(6), 1121-1130.

Stubberud, J., Langenbahn, D., Levine, B., Stanghelle, J., & Schanke, A. K. (2015). Emotional health and coping in spina bifida after goal management training: A randomized controlled trial. *Rehabilitation psychology*, *60*(1), 1.

Summerfeldt, L. J., Richter, M. A., Antony, M. M., & Swinson, R. P. (1999). Symptom structure in obsessive-compulsive disorder: a confirmatory factor-analytic study. *Behaviour Research and Therapy*, *37*(4), 297-311.

Tolin, D. F., Abramowitz, J. S., & Diefenbach, G. J. (2005). Defining response in clinical trials for obsessive-compulsive disorder: a signal detection analysis of the Yale-Brown obsessive compulsive scale. *Journal of Clinical Psychiatry*, *66*(12), 1549-1557.