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The Neuropsychological Profile Of Obsessive-Compulsive Disorder In Young Adults

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THE NEUROPSYCHOLOGICAL PROFILE OF OBSESSIVE-COMPULSIVE DISORDER IN
YOUNG ADULTS

by

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University of Toronto, Toronto, Ontario, 2009

A thesis

Presented to Ryerson University

in partial fulfillment of the

requirements for the degree of

Master of Arts

In the Program of

Psychology

Toronto, Ontario, Canada, 2011

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Master of Arts 2011

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Psychology, Ryerson University

Abstract

The purpose of this thesis is to compare the cognitive functioning in young adults with OCD against same-aged healthy controls. Current neuropsychological findings in adults with OCD are mixed, thus by examining cognitive functioning in young adults with OCD aged 18-29 years could clarify whether deficits emerge during this time. A neuropsychological battery was administered to both groups. **Results:** Comparable performance was found in inhibitory control, information processing speed, motor speed and manual dexterity, but deficits appeared in the OCD group on decision-making, set shifting, copy time of a complex figure, and verbal memory. State anxiety showed a large effect that was correlated to performance deficits. **Conclusions:** Young adults with OCD have similar cognitive profiles as their healthy counterparts but they may be more anxious in testing situations. Replication of these findings with a larger sample is necessary to better understand the cognitive profile in young adults with OCD.

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Overview

The purpose of this thesis is to evaluate whether cognitive deficits exist in young-adults with obsessive-compulsive disorder (OCD) when compared to same-age healthy controls. A review of the literature shows that some cognitive impairment is present in adults (18 - 72 years of age) but not in children with OCD. The following overview makes the case that select cognitive impairments in individuals with OCD begin to manifest right after brain maturation is complete, typically in early adulthood. Currently no studies have specifically examined the neuropsychological profile of young adults with OCD (ages 18 – 29 years).

OCD is a chronic neuropsychiatric disorder marked by repetitive intrusive obsessions and/or compulsions, which are highly distressing and disruptive to the lives of the individuals affected. OCD typically develops in childhood or adolescence and up to 85% of these cases appear before 35 years of age (Torres et al., 2006). OCD typically persists into adulthood with minimal remission of symptoms (Rasmussen & Eisen, 1992). OCD occurs equally across genders with prevalence rates ranging from 1.1% to 3% (Rasmussen & Eisen, 1992). The presentation of obsessions and compulsions are heterogeneous, hence subtypes of OCD have been acknowledged and classified according to preoccupations with symmetry/order, hoarding, contamination/cleaning, and obsessions/checking behaviours (Mataix-Cols, do Rosario-Campos, & Leckman, 2005). OCD is complicated by the presentation of co-morbid disorders. A study of individuals with OCD found that at least 62% had at least one other Axis I disorder, such as depression or generalized anxiety disorder (Torres et al., 2006). Patients who developed a major depressive episode after the onset of OCD were noted to have more obsessions than compulsions (Besiroglu, Uguz, Saglam, Agargun, & Cilli, 2007). The co-occurrence of other mental disorders are cited as a source of confounds and limitations in understanding OCD (Grabill et al.,

2008). OCD is a complex disorder, where few individuals ever fully recover (Schruers, Koning, Luermans, Haack, & Griez, 2005). Consequently, OCD sufferers face lifelong impairment in multiple facets of functioning.

Past lesion and neuroimaging literature in OCD provides substantial evidence to support that behavioural symptoms and cognition of OCD patients are mediated by abnormal frontal lobe circuits. For instance, OCD symptoms can emerge following focal brain injury (Berthier, Kulisevsky, Gironell, & Heras, 1996) and following surgery to frontal lobe structures (Lambrecq et al., 2009; Nyman & Mindus, 1995). Moreover, neurological disorders that share similar pathophysiology with OCD, such as Tourette's syndrome, Parkinson's disease, and Huntington's disease manifest stereotypical and repetitive behaviours (Como, Weiner, & Lang, 1995; Tröster & Woods, 2005). The most compelling evidence comes from functional neuroimaging data, which has been shown to differentiate individuals with OCD from healthy people based on the observation of hyper-activation of the orbitofrontal cortex at resting state (Mataix-Cols & van den Heuvel, 2006). Further, provocation of OCD symptoms have been associated with increased basal activation of the orbitofrontal, dorsolateral and anterior cingulate circuits (Adler et al., 2000; Breiter, Rauch, Kwong, & Baker, 1996; Rauch et al., 2002; Rotge et al., 2008). Structural and functional neuroimaging evidence suggests that the frontostriatal pathways, in particular three functionally segregated circuits involving the dorsolateral prefrontal cortex (DLPFC), the orbitofrontal cortex and the anterior cingulate cortex (ACC), play important roles in the pathophysiology of OCD (Fontaine, Mattei, & Robert, 2007).

Current models of OCD based on clinical or behavioural symptoms and meta-cognitive factors (e.g., excessive doubt, confidence in memories), separately or in combination, do not fully account for the etiology of the disorder. Thus, an endophenotypic approach has been

suggested (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). Endophenotypes are intermediate markers that lie between the genetic blueprint and the expression of genetic material, referred to as phenotypes (Gottesman & Gould, 2003). Endophenotypes must meet five criteria set out by Gottesman (2003). The endophenotype must be associated with illness in the affected population and are inheritable. Endophenotypes cosegregate with illness in families and are found at higher rates in unaffected relatives of probands. Lastly, endophenotypes are state-independent and thus manifests whether the illness is present or not. Endophenotypes can be neuropsychological, neuroanatomical, endocrinological, biochemical and cognitive. For example, cognitive processes such as failures in inhibitory control have been thought of as the expression of the underlying genetic code in OCD (Chamberlain et al., 2005). Thus, cognitive endophenotypes, mediated by frontal circuits, may be important in unraveling the multidimensionality of OCD.

Many neuropsychological studies have compared behavioural and cognitive performance in individuals with OCD against healthy controls using a variety of psychometrically validated tests. The findings of cognitive deficits from these studies are based on statistical differences arising from statistical tests that fall below the a priori set p-values (typically set at $p < 0.05$ as being significant). No studies have specifically examined clinically significant differences by comparing the performance of individuals with OCD to normative samples. Thus, this thesis will follow the established convention in the literature and refer to behavioural and cognitive deficits as differences that meet by statistical significance.

A handful of recently published studies in children and adolescents with OCD provide a basis for examining cognitive processes, particularly executive functions in this population. It appears that most executive functions are, for the most part, unaffected in children and

adolescents with OCD. For example, several studies have found that children with OCD performed equally well or slightly worse (but not significantly different) relative to healthy children and adolescents on executive function measures such as inhibitory control, abstract reasoning and planning, and cognitive flexibility (Andres et al., 2007; Beers et al., 1999; Ornstein, Arnold, Manassis, Mendlowitz, & Schachar, 2010; Woolley et al., 2008). In contrast to child studies, recent studies in adults with OCD have shown impairments in select executive tasks, including inhibition (Menzies et al., 2007; Rao, Reddy, Kumar, Kandavel, & Chandrashekar, 2008), cognitive flexibility (Lawrence et al., 2006; Rao et al., 2008), decision-making (Cavedini, Gorini, & Bellodi, 2006; Starcke, Turschen-Caffier, Markowitsch, & Brand, 2010), abstract reasoning and problem solving (Cavedini et al., 2002; Lawrence et al., 2006; Purcell, Maruff, Kyrios, & Pantelis, 1998a). Collectively, there appears to be executive control deficits in adult OCD cases while children with OCD show intact executive functioning. The neurocognitive presentation in children and adults will be discussed in more detail in the neuropsychology section of this paper (Pages 10-19).

Although a large body of literature appears to support some executive function deficits in adults with OCD, there are also some mixed results (see reviews Greisberg & McKay, 2003; Kuelz, Hohagen, & Voderholzer, 2004). The disparity reported across studies may partly be due to the practice of sampling participants from a broad age range (i.e., 18-55 years). Moreover, the role of neuro-anatomical development has not been considered per se, although it is well known that frontal lobe maturation continues well into early adulthood and concomitant cognitive abilities emerge as a result of neural maturation (Stuss, 1992). For example, increasing proficiency on executive type tasks has been observed in a cross-sectional cohort of normal children and adolescents, particularly after 12 years of age, where problem solving and semantic

organization showed the greatest improvement (D. McKay, Taylor, & Abramowitz, 2010; K. McKay, Halperin, Schwartz, & Sharma, 1994; Sowell, Delis, Stiles, & Jernigan, 2001). This implies that developmentally appropriate cognitive functioning cannot be conclusively established until after this maturation period. Thus, the emergence of ‘fully functional’ executive functions likely follows the maturation of the prefrontal cortex in early adulthood (i.e., 18 years and over).

If OCD pathophysiology is postulated to be the result of aberrant brain networks, research should investigate this hypothesis in late adolescence or early adulthood, when functional development is complete (Sowell et al., 2001). Young adults with OCD are an ideal population for investigating cognitive dysfunction arising from frontostriatal abnormalities. If poor executive function performance outcomes are observed in young adults with OCD when compared to healthy controls, these findings would provide support that the neurobiological underpinnings of OCD are based on frontal lobe integrity.

In order to provide background on the cognitive profile across age cohorts, the following section summarizes the neurocognitive performance of children, adolescents and adults with OCD (also refer to Table 1). The small number of child and adolescent studies report relatively intact cognitive abilities (Andres et al., 2007; Beers et al., 1999; Ornstein et al., 2010; Woolley et al., 2008). However, findings from a sizable number of neuropsychological studies in adults with OCD appear to support dysfunction in visuospatial abilities, motor impairments, information processing deficits, memory and select executive function processes (for review, see Chamberlain et al., 2005; Greisberg & McKay, 2003; Kuelz et al., 2004).

Table 1. Summary of cognitive functioning in children and adults with OCD

Domains	Child & Adol.	Authors	Adult	Authors
Executive Functioning				
Inhibitory Processes (BI: Go/no-go, Stop signal; CI: Negative priming, Stroop)	=	Beers et al., 1999, (CI); Ornstein et al., 2010 (BI); Woolley et al., 2008 (BI)	X	Aycicegi et al., 2003, (BI); Bannon et al., 2002, (BI); Hartson et al, 1999, (CI); Chamberlain et al., 2007, (BI); Martinto et al., 1990, (CI); Menzies et al., 2007, (BI); Penadés et al., 2007, (BI); Rao et al., 2008, (CI); Watkins et al, 2004, (BI) Bannon et al., 2002, (CI); Boone et al., 1991, (CI); Ciesielski et al., 2011, (CI); Enright et al., 1995, (CI); Hollander et al., 1993, (CI); Johannes et al., 2001, (BI); Kivircik et al., 2003, (CI); Krikorian et al., 2004, (BI); Mataix-Cols et al., 2002, (CI); Morein-Zamir et al., 2010, (BI); Moritz, et al., 2010, (CI); Page et al., 2004, (BI); Schmidtke et al., 1998, (CI)
Abstract Reasoning & Planning (TOH, TOL, etc.)	X	Behar et al., 1984; Ornstein et al., 2010	X	Cavedini et al., 2001; Mataix-Cols et al., 1999; Purcell et al., 1998a; Purcell et al., 1998b; Rampacher et al., 2010; Schmidtke et al., 1998; Veale et al., 1996; Bédard et al., 2009; Rampacher et al., 2010; Rao et al., 2008; Schmidtke et al., 1998; Simpson et al., 2006
	=	Beers et al., 1999	=	
Cognitive Flexibility/ Set Shifting (WCST, OAT, TMT B, etc.)	X	Shin et al., 2008	X	Abbruzzese et al., 1995; Abbruzzese et al., 1997; Aigner et al., 2007; Aronowitz et al., 1994; Cohen et al., 1996; Fenger et al., 2005; Head et al., 1989; Kim et al., 2009; Lawrence et al., 2006; Lucy et al., 1997; Nelson et al., 1993; Purcell et al., 1998a; Purcell et al., 1998b; Rao et al., 2008; Spitznagel et al., 2002; Veale et al., 1996

	=	Andrés et al, 2007; Beers et al., 1999; Britton, et al., 2010; Ornstein et al., 2010; Woolley et al., 2008	=	Abbruzzese et al.,1995; Basso et al., 2001; Bédard et al., 2009; Boone et al., 1991; Burdick et al., 2008; Christensen, 1992; Fenger et al., 2005; Hwang et al., 2007; Kim et al., 2002; Kivircik et al., 2003; Mataix-Cols et al., 1999; Schmidtke et al., 1998; Simpson et al., 2006; Zielinski et al., 1991
Decision-making (IGT and variants)	?	No known studies conducted in children or adolescents	X	Cavallaro et al., 2003; Cavedini et al., 2000; Cavedini et al., 2010; Foa et al., 2003; Starcke et al., 2010
			=	Lawrence et al., 2006; Nielen et al., 2002
Verbal and non-Verbal Fluency (COWA, DFT)			X	Hwang et al., 2007; Lacerda et al., 2003; Schmidtke et al., 1998
	=	Beers et al., 1999; Ornstein et al., 2010	=	Basso et al., 2001; Boone et al., 1991; Burdick et al., 2008; Christensen, 1992; Kim et al., 2002; Kim et al., 2009; Kivircik et al., 2003; Rao et al., 2008; Zielinski et al., 1991
Motor Speed (Grooved Peg Board, finger tapping)			X	Basso et al., 2001; Bédard et al., 2009; Burdick et al., 2008; Christensen, et al., 1992
	=	Beers et al., 1999; Ornstein et al., 2010	=	--No Findings--
Visuospatial Constructive Ability (Tasks: RCFT, WAIS -BD, Spatial)	X	Andrés et al, 2007	X	Aronowitz et al., 1994; Boone et al., 1991; Hollander et al., 1993; Hwang et al., 2007; Kim et al., 2002; Lacerda et al., 2003; Purcell et al., 1998a; Purcell et al., 1998b; Rao et al., 2008;

Span)				Schmidtke et al., 1998; Segalàs et al., 2008; Shin et al., 2010
	=	Ornstein et al., 2010; Shin et al., 2008	=	Kim et al., 2009; Martin et al., 1993; Rampacher et al., 2010; Simpson et al., 2006; Zielinski et al., 1991
Memory				
Verbal Memory (Tasks: CVLT, LM)			X	Cabrera et al., 2001; Deckersbach et al., 2000; Savage et al., 2000; Sher et al., 1984; Segalàs et al., 2008; Sawamura et al., 2005; Zitterl et al., 2000
	=	Andrés et al., 2008; Beers et al., 1999; Ornstein et al., 2010	=	Bédard et al., 2009; Burdick et al., 2008; Christensen, 1992; Cohen et al., 1996; Dirson et al., 1995; Kim et al., 2009; Martin et al., 1993; Muller et al., 2004; Rampacher et al., 2010; Zielinski et al., 1991
Working Memory (Tasks: WAIS Subtests: DSp, DSy, AR)	X	Andrés et al., 2008	X	--No findings--
	=	Shin et al., 2008 Beers et al., 1999	=	Bédard et al., 2009; Burdick et al., 2008; Hollander et al., 1993; Hwang et al., 2007; Sawamura et al., 2005
Visual Memory (Tasks: VR, BRVT, Spatial Span)	X	Andrés et al., 2008	X	Cohen et al., 1996; Christensen et al., 1992; Dirson et al., 1995; Purcell et al., 1998a; Simpson et al., 2006
	=	Ornstein et al., 2010	=	Bédard et al., 2009; Burdick et al., 2008
Attention (Tasks: CPT, Posner task, d2)			X	Aigner et al., 2007; Schmidtke et al., 1998
	=	Andrés et al., 2008; Beers et al., 1999; Ornstein, et al., 2010; Shin et al., 2008	=	Aronowitz et al., 1994; Clayton et al., 1999; Coetzer et al., 1999; Cohen et al., 1996; Cols et al., 2002; Hollander et al., 1993; Kim et al., 2009; Milliery et al., 2000; Nelson et al., 1993; Rao et

				al., 2008; Schmidtke et al., 1998; Stein et al., 1997; Zielinski et al., 1991
Information Processing (Tasks: WAIS-III LN, WAIS-IV CO, TMT A)	X	Andrés et al., 2008;	X	Bédard et al., 2009; Boone et al., 1991; Burdick et al., 2008; Schmidtke et al., 1998
	=	Ornstein et al., 2010	=	Aronowitz et al., 1994; Berthier et al., 1996; Cohen et al., 1996; Martin et al., 1993; Basso et al., 2001; Moritz et al., 2001a; Moritz et al., 2002

X – OCD impaired relative to controls; **=** OCD performed comparable to controls; **?** Inconclusive or unknown; AR – Arithmetic; BI- Behavioural inhibition; BVRT – Benton Visual Recognition Test; CI – Cognitive inhibition; CVLT – California Verbal Learning Test; CO – Coding; COWA – Controlled Oral Word Association Test; CPT – Continuous Performance Task; DAT – Delayed Alternation Task; DFT – Design Fluency Test; DSp – Digit Span; DSy – Digit Symbol; IGT – Iowa Gambling Task; RCFT – Rey Complex Figure Test; SS – Spatial Span; TOH – Tower of Hanoi; TOL – Tower of London; OAT – Object Alternation Task; TMT A/B – Trails Making Task A or B; WAIS – Wechsler Adult Intelligence Scale; WCST – Wisconsin Card Sorting Task.

Neuropsychology of OCD

Executive Function

Executive function is an umbrella term to describe processes that orchestrate planning, organizing and performing discrete complex actions (Samango-Sprouse, 2007). Executive function refers to higher level cognitive processes comprising abilities such as reasoning and planning that are carried out by the phylogenetically and ontogenetically newer DLPFC while the deeper, and older orbitoventral regions of the prefrontal cortex (PFC) play a role in inhibition and decision-making (Bedard, Joyal, Godbout, & Chantal, 2009).

The emergence of these abilities likely follows the maturation of the prefrontal cortex, which progresses anteriorly from the primary motor cortex to the frontal poles (Dennis, 2006). Increasing proficiency on executive type tasks has been observed in a cross-sectional cohort of healthy children and adolescents, particularly after 12 years of age, where problem solving, attention and semantic organization showed the greatest improvement (Levin, Culhane, Hartmann, & Evankovich, 1991; D. McKay et al., 2010).

Cognition in children and adolescents with OCD has also been investigated. It appears that most executive functions are unaffected in children and adolescents with OCD. For example, several studies have found that children with OCD performed equally well relative to healthy children and adolescents on executive function measures (Andres et al., 2007; Beers et al., 1999; Ornstein et al., 2010; Woolley et al., 2008); however, one study reported trends in impairment on executive function processes, including cognitive flexibility (e.g., Wisconsin Card Sorting Task), verbal fluency, and planning ability (e.g., Tower of London Task; TOL) (Ornstein et al., 2010). Thus, it appears that cognitive changes may be emerging in children with OCD.

In contrast, adults with OCD exhibit impairments in select executive tasks, including inhibition (Bannon, Gonsalvez, Croft, & Boyce, 2002; Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Johannes et al., 2001; Penades et al., 2007), cognitive flexibility (Abbruzzese, Bellodi, Ferri, & Scarone, 1995; Abbruzzese, Ferri, & Scarone, 1997; Lawrence et al., 2006), and abstract reasoning and problem solving (Bannon et al., 2002; Chambers, Garavan, & Bellgrove, 2009). Finally, decision-making may be impaired, but has received little attention to date (Sachdev & Malhi, 2005).

Inhibitory Processes. Adaptive human behaviour depends on the ability to cancel or inhibit responses that are no longer advantageous (Chambers et al., 2009). Inhibition has been conceptualized as two distinct processes (Barkley, 1997; Harnishfeger, 1995; Nigg, 2000).

Behavioural inhibition refers to the suppression of a prepotent motor response or stopping of an ongoing response (Chamberlain et al., 2005). *Cognitive inhibition* speaks to the suppression of internal thoughts and images that are elicited by environmental stimuli (Harnishfeger, 1995), and the resistance of competing interference from varying external sources (Barkley, 1997).

Together, these abnormal inhibitory processes in OCD may account for the core clinical symptoms, which include the failure to inhibit compulsive behaviours and intrusive obsessional thoughts (Chamberlain et al., 2005).

Several experimental tasks devised to measure inhibition appear to have neural correlates in the frontostriatal regions (Menzi et al., 2007; Page et al., 2009). Behavioural inhibition can be objectively measured by the go/no-go task (Rubia et al., 2001) and the stop-signal task (Logan & Cowan, 1984). The go/no-go is a simple choice-reaction task wherein subjects respond when a target letter appears (e.g., M), but must inhibit the response to an infrequent non-target letter (e.g., X). In the stop-signal task, an auditory signal to “stop” is emitted in 25% of randomly

selected trials. Subjects are required to withhold their response when they hear an auditory tone. Hence, the go/no-go task differs from the stop task in that subjects know apriori whether or not to respond to presented stimuli. Both tasks share common activation loci such as the right inferior and middle frontal gyri, anterior cingulate cortex, right inferior parietal lobe and left middle temporal cortex (Chambers et al., 2009; Rubia et al., 2001).

Child OCD studies that have used the go/no-go and stop-signal paradigms have failed to show significant differences between OCD children and healthy controls (Andres et al., 2007; Andres et al., 2008; Beers et al., 1999; Ornstein et al., 2010; Woolley et al., 2008). In contrast, adult patients with OCD struggle with inhibiting recurrent actions and exhibit impairment on tasks that putatively tap behavioural inhibition (Bannon et al., 2002). For instance, adults with OCD had longer stop-signal reaction times than healthy controls on the stop-signal task (Chamberlain et al., 2006). Moreover, using a go/no-go task, participants with OCD exhibited longer reaction times than healthy controls (Aycicegi, Dinn, Harris, & Erkmen, 2003) and made more commission errors compared to patients with panic disorder (Bannon et al., 2002). Further, adults with OCD were significantly less successful at inhibiting their responses.

In regard to the higher number of errors observed in individuals with OCD relative to panic-disorder on the go/no-go task (Bannon et al., 2002), it is not possible to state whether those deficits are specific to OCD given a group of healthy controls was not included for comparison. Nonetheless, these results suggest that individuals with OCD are slower at inhibiting their responses, although it is unclear whether their slowness is the result of obsessional slowness that is often observed in people with OCD (Bilbury & Morley, 1979; Hantouche, 2000; Rachman, 1974; Takeuchi et al., 1997), or whether their information processing speed is impaired, which can affect their behavioural response (e.g., Bedard et al., 2009; Burdick, Robinson, Malhotra, &

Szeszko, 2008); however, most studies have found normal information processing speed (see Table 1).

To assess *cognitive inhibition*, the Stroop Colour Word Test (Golden, 1976) is commonly used. The Stroop task requires the inhibition of automatic reading of a word in favour of naming the ink colour of the printed word. OCD patients had slower reaction times and made more errors in the interference condition of the Stroop task (Hartston & Swerdlow, 1999; Martinot, Allilaire, Mazoyer, & Hantouche, 1990; Penades et al., 2007; Rao et al., 2008) when compared to healthy controls. However, several other studies have also shown that individuals with OCD are not impaired relative to healthy controls on this task (Boone, Ananth, & Philpott, 1991; Hollander, Cohen, Richards, & Mullen, 1993; Schmidtke, Schorb, Winkelmann, & Hohagen, 1998).

To summarize, much interest has been generated in recent years on the possible role of behavioural and cognitive inhibitory processes in the etiology of OCD (Chamberlain et al., 2005). While child studies have found no deficits in inhibitory responses, adults with OCD tend to demonstrate deficits in behavioural inhibition, while the cognitive inhibition findings are somewhat mixed.

Cognitive Flexibility. Attentional set-shifting is the ability to change mental strategies, and is dependent on the degree of cognitive-flexibility, or how readily set-shifting occurs (Lawrence et al., 2006). The Wisconsin Card Sorting Test (WCST) is purportedly a measure of cognitive flexibility, thought to be mediated by the DLPFC (Fuster, 2002). Subjects are required to figure out the sorting rule of a series of cards based on examiner feedback. A “right” response correctly matches target cards based on 1 of 4 key cards, categorized according to colour, shape

or number. After 10 consecutive correct answers, the sorting principle changes. Success on this task requires the ability to adapt to changing rules and subsequently, switch mental sets.

Children and adolescents tested on the WCST do not seem to be impaired (Andres et al., 2007; Beers et al., 1999; Britton et al., 2010; Ornstein et al., 2010; Woolley et al., 2008).

However, the results from adult studies are not as clear. Deficiencies in set-shifting ability have been supported (Boone et al., 1991; De Geus, Denys, Sitskoorn, & Westenberg, 2007; Hymas, Lees, Bolton, Epps, & Head, 1991; Lucey et al., 1997; Okasha et al., 2000), while some studies using the same paradigm found consistent performance between adults with OCD and healthy controls (Abbruzzese, Ferri, & Scarone, 1995; Deckersbach, Otto, Savage, Baer, & Jenike, 2000; Gross-Isseroff, Sasson, Voet, & Hendler, 1996; Moritz et al., 2001; Moritz et al., 2002; Zielinski, Taylor, & Juzwin, 1991). These mixed findings may be explained by the fact that the WCST is not a pure measure of set-shifting; rather, it likely taps many other processes (i.e., inhibition, working memory, and the maintenance of attentional processes) (Anderson, Damasio, Jones, & Tranel, 1991; Barcelo, 2001), and may be more sensitive to the DLPFC than to orbitofrontal cortex dysfunction (Abbruzzese et al., 1997).

Previous work has implicated the orbitofrontal striatal circuitry in the neuropathophysiology of OCD and studies that used different instruments (e.g., Intra-Extra dimensional set shift task on the Cambridge Neuropsychiatric Test Automated Battery, CANTAB ID/ED, Trail Making Test - TMT) that are sensitive to this region, have found significant set-shifting deficits (Abbruzzese et al., 1997; Cavedini, Ferri, Scarone, & Bellodi, 1998; Fenger et al., 2005; Spitznagel & Suhr, 2002). In sum, although a multitude of studies have shown mixed results using the WCST, a small number of studies that used alternating tasks (e.g., TMT, CANTAB ID/ED) found significant impairments.

Abstract Reasoning and Planning. Problem solving and prospective behaviour depends on the ability to reason, think abstractly, strategize and plan sequences, most of which tend to develop fully by 15 - 17 years of age (Luciana & Nelson, 2002). Similar to healthy children (Luciana, Collins, Olson, & Schissel, 2009; Marsh, Gerber, & Peterson, 2008), children and adolescents with OCD do not appear to be impaired on tasks that are purportedly sensitive to these executive functions (e.g., Tower of Hanoi, TOH & Tower of London, TOL) (Behar et al., 1984; Ornstein et al., 2010). On the other hand, conceptual thinking and planning ability may be affected in adults with OCD, as reported in studies that used the same tasks (Rao et al., 2008; Schmidtke et al., 1998; Simpson et al., 2006). The findings show that OCD participants were as accurate as controls in solving the problems, but differences arose in longer response times on the inaccurate trials (Veale, Sahakian, Owen, & Marks, 1996), as well as protracted times required to initiate moves, indicating a possible problem with indecision (Purcell et al., 1998a; Purcell, Maruff, Kyrios, & Pantelis, 1998b).

Decision-Making. There are no published studies on decision-making in children with OCD to date; however, a small literature has examined decision-making in adults with OCD. The Iowa Gambling Task (IGT) (Bechara, Damasio, Damasio, & Anderson, 1994) is considered an analogue task for ambiguous real-life decision-making in which subjects choose cards from four decks that have predetermined advantageous and disadvantageous winnings. Adult patients with ventromedial prefrontal cortical (VMPFC) damage who were administered the IGT continued to choose losing cards over 100 trials (Bechara et al., 1994). Thus, the IGT has been used as an indicator of decision-making ability in various populations (Bechara, Damasio, & Damasio, 2000; Bechara, Damasio, Tranel, & Damasio, 2005; Tranel, 2002).

In a couple of studies, adults with OCD showed risky decision-making based on their poor performance on the IGT. As a group, their net winnings were lower than healthy controls, indicating they chose more cards from the disadvantageous decks than the advantageous decks (Cavedini et al., 2002; Starcke et al., 2010). However, other studies using the same task have not found such differences (Nielen, Veltman, de Jong, Mulder, & den Boer, 2002). Watkins et al. (2005) used a novel decision-making task; however, sensitivity to orbitofrontal cortex damage has not previously been verified (Watkins et al., 2005). Overall, non-significant findings might be related to differences in OCD subtype; people with predominantly hoarding symptoms tended to do worse compared to the other subtypes (Lawrence et al., 2006) albeit, their performance was no different than that seen in healthy controls.

Verbal Fluency. The ability to generate words when given the first letter of the word is an executive function that is established by middle childhood (Gaillard et al., 2000). Overall, in both child (Beers et al., 1999; Ornstein et al., 2010) and the majority of adult OCD studies (Greisberg & McKay, 2003; Kuelz et al., 2004), there is little evidence to support impairment in verbal fluency.

To summarize, most cognitive processes that fall within the executive function umbrella appear intact in children/adolescents with OCD. However, in studies that used similar tasks, adults with OCD showed more consistent deficits on tasks of inhibition, cognitive flexibility, abstract reasoning, and planning. Decision-making deficits may exist in adulthood, but few studies exist, and there are no published studies in children or adolescents to suggest pathological evolution in OCD. Verbal fluency appears to be intact from the early years to adulthood.

Attention

Attention is a widely studied process. In normal children, selective attention measured with tasks such as the Flanker and visual search tasks seem to reach adult performance before seven years of age (K. McKay et al., 1994). In contrast, performance of sustained attention on the Continuous Performance Task improves significantly between 11 years of age and adulthood (K. McKay et al., 1994). Results from the small number of studies in children with OCD that employed selective attention tasks found no significant difference compared to normal children (Andres et al., 2007; Beers et al., 1999; Ornstein et al., 2010; Shin et al., 2008). Sustained attention has not been assessed in children with OCD. The adult literature in OCD provides solid evidence to support non-impairments in sustained attention (Millierey, Bouvard, Aupetit, & Cottraux, 2000) or selective attention (Aronowitz et al., 1994; Clayton, Richards, & Edwards, 1999; R. Coetzer & Stein, 1999; Cols, 2006; Hollander et al., 1993; Schmidtke et al., 1998; Stein, Coetzer, Lee, Davids, & Bouwer, 1997). Taken together, these studies suggest that attention may be relatively preserved in OCD.

Information Processing Speed

The time that is required to absorb, integrate and respond to new information reflects an individual's rate of information processing. The results are mixed in studies on children and adults with OCD. These findings may have been confounded by the influence of psychotropic medication (e.g., selective serotonin reuptake inhibitor, SSRI), which is thought to slow the speed of processing (Basso, Bornstein, Carona, & Morton, 2001; Schmidtke et al., 1998). Medication-free adults with OCD tend to show intact information processing speed (Aronowitz et al., 1994; Cohen, Hollander, DeCaria, & Stein, 1996; Jurado, Junqué, Vallejo, & Salgado, 2001). Processing speed may also affect other executive function tasks that have motoric

components (e.g., TOL, TMT) (Burdick et al., 2008). In light of these findings, it is important to consider how information processing speed contributes to the overall cognitive profile of OCD.

Verbal Memory

To date, verbal memory in children with OCD is impaired based on their inferior performance on list recall compared to healthy controls (Andres et al., 2007; Beers et al., 1999; Ornstein et al., 2010; Shin et al., 2008). Many researchers have attempted to determine whether verbal memory is impaired in adults with OCD. Several studies have suggested that adults with OCD are impaired on verbal memory (Deckersbach et al., 2000; Savage & Rauch, 2000; Sher, Mann, & Frost, 1984; Zitterl et al., 2001) as demonstrated by their performance on the California Verbal Learning Test (CVLT) (Delis, Kramer, Kaplan, & Ober, 1987). On the other hand, other studies have not found such an impairment using this task (Christensen, Kim, Dysken, & Hoover, 1992; Cohen et al., 1996; Zielinski et al., 1991).

Visuospatial Constructive Skills and Visual Memory and Learning

Non-verbal constructional skills are usually measured by having a subject copy a complex geometric figure. According to published studies, visuospatial constructive abilities do not seem impaired in young children nor adolescents with OCD, but these deficits become apparent in adulthood (refer to Table 1). Further, visual memory and learning is typically assessed by the accurate replication of the drawing after a short and long time delay. Neuropsychological findings indicate that adults with OCD manifest impairment on immediate and delayed visual-recall tasks (Cohen et al., 1996; Deckersbach et al., 2000; Martinot et al., 1990; Savage et al., 1999; Savage et al., 2000). It has been suggested that deficits in visuospatial memory may in fact reflect poor organizational strategies and is not a memory deficit per se (Savage et al., 1999). A memory deficit implies abnormalities in the mesio-temporal lobes

(Head, Rodrigue, Kennedy, & Raz, 2008) but adults with OCD do not show impairments with encoding new information because they are able retrieve memories (Savage et al., 2000). In support of this, Cabrera, McNally and Savage (2001) reported that OCD patients compared to controls did not differ in their memory for individual sentences or recognition memory but rather, underused integration of semantic units in complex sentences (Cabrera, McNally, & Savage, 2001).

Motor Speed

Fine motor control and coordination is governed by the premotor and motor areas of the frontal lobes and does not mature until adolescence (Diamond, 2000). Motor speed and coordination can be assessed by the Grooved Peg Board and the Finger Tapping tests (Heaton, Grant, & Matthews, 1992). Studies that used the Grooved Peg Board, a timed measure of manual dexterity and co-ordination, found children with OCD performed equally well as healthy children. On the other hand, adults with OCD were found to be slower than healthy controls on the same task (Basso et al., 2001; Bedard et al., 2009; Burdick et al., 2008). Thus, motor dysfunction in OCD may not become apparent until adulthood.

Rationale and Goals for this Study

Cognitive development in healthy children from age 5 onward reflects a progression of improvement in many areas, including planning, verbal fluency, inhibition and set-shifting abilities (Reynolds & Horton, 2008). Likewise, following a review of the literature in child-onset OCD, most processes appear unaffected; although, there may be trends toward executive dysfunction related to abstract reasoning and planning (Beers et al., 1999; Ornstein et al., 2010). Moreover, certain cognitive impairments are present in adulthood such as abstract reasoning, set-shifting and response inhibition, and arguably, non-verbal memory, and visuospatial impairment.

Hence, it is important to investigate whether these deficits emerge in early adulthood. Such findings would provide an avenue for early treatment intervention.

Impaired functioning in adults with OCD raises questions about the status of cognition in young adults with OCD, who may begin to manifest cognitive impairments that parallel the maturation of the prefrontal cortex. There have, however, been no published attempts to explore the nature of neurocognitive performance in young adults with OCD. Thus, the research goal to be addressed in this thesis is whether young adults with OCD manifest cognitive deficits, particularly in cognitive domains where existing studies suggest differential performance in adults but not children. These processes include: response inhibition, abstract reasoning, cognitive flexibility, decision-making, motor speed, visuospatial constructive ability, verbal and visual memory and learning and finally, information processing speed.

To reconcile some of the observed differences in the literature to date, this study sought to examine whether young adults with OCD between the ages 18 – 29 years exhibit executive function impairment, in addition to memory and visuospatial constructive abilities when compared to healthy, same-aged peers. Two specific objectives were put forth in this study: 1) to investigate whether performance differences in executive function and other cognitive domains exist between the groups; and 2) to evaluate the relationship between clinical symptoms (obsessions/compulsions, and illness duration) and cognitive performance.

Hypotheses

It was predicted that young adults with OCD would do worse than healthy controls on executive functions. Specifically, impaired performance relative to healthy controls is expected to be observed on executive indices reflecting inhibitory processes, abstract reasoning, mental flexibility and decision-making. Poorer performance was also hypothesized in the domains of

motor speed, visuospatial constructive ability and learning, and verbal memory. Information processing speed is hypothesized to be intact. Attention and verbal fluency appear intact in child and adults with OCD, and thus will not be evaluated in this study. A second goal was to examine the relation between clinical variables (i.e., obsessions, compulsions) and neuropsychological performance in young adults with OCD. Previous studies suggest that clinical variables are not associated with cognitive function (Bedard et al., 2009; Deckersbach et al., 2000; Dirson, Bouvard, Cottraux, & Martin, 1995; Lucey et al., 1997) therefore, we do not anticipate finding any correlations between performance on neuropsychological tests and clinical symptoms.

Methods and Procedure

Participants

There were 14 participants with OCD (8 females and 6 males) and 13 healthy controls (8 females and 5 males) ranging in age from 18-28 years and with similar age and IQ (i.e., intellectual functioning). Demographic and clinical data on the participants can be found in Table 2. Ethics approval for this project was obtained from the Research Ethics Board at Ryerson University. Participants with OCD were recruited from three Ryerson services, the medical centre, access centre, and counseling centre), and community postings. Advertisements for healthy controls were posted on Ryerson campus grounds. For the OCD participants, a clinical interview (e.g., Mini International Neuropsychiatric Interview, MINI; Sheehan et al., 1998) and self-report measure of OCD severity (e.g., Yale-Brown Obsessive-Compulsive Scale, Y-BOCS; Goodman et al., 1989) was administered to establish OCD diagnosis and severity. The State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) and

the Beck Depression Inventory, second edition (BDI-II; Beck, Steer, & Brown, 1996) were administered to assess for state anxiety and depression, respectively.

Three young adults with OCD were taking SSRIs (e.g., sertraline) and one was taking medication for Crohn's disease. OCD age of onset spanned a range of 8-20 years, averaging $M = 12.71$, $SD = 3.75$. The young adults with OCD had the disorder for 2-20 years ($M = 10.07$, $SD = 5.09$). Eleven of the 14 participants presented with symptoms for mental disorders based solely on the MINI (e.g., depression, generalized anxiety disorder, substance dependence). All participants were right-handed.

Suitability for inclusion in the study was established over the phone prior to the face-to-face meeting. Individuals were excluded from the study if they met criteria for one or more of the following: history of head injury, psychosis, medical or neurological condition, or colour blindness. The OCD participants were not excluded based on co-morbid symptomology or medication usage given the small sample size obtained and the difficulty of recruiting medication-free participants with a sole diagnosis. One test session lasting approximately four hours took place in a quiet lab room at Ryerson's Psychology Research and Training Centre. The following was explained to all participants: the purpose and nature of the study, participation is voluntary and the individual can withdraw from the study at anytime without penalty. Consent to participate in the study was obtained from participants before testing commenced. All participants were presented with a battery of neuropsychological measures that tapped the domains of interest. The tests were ordered in such a sequence that they were unlikely to interfere with each other on performance (i.e., verbal tests did not follow one another). Periodic breaks were offered to the participants to minimize fatigue. Participants received financial compensation of \$40 for 4 hours of their time.

Measures

Clinical Measures

The MINI (Sheehan et al., 1998, Cronbach's alpha = .66) is a brief structured diagnostic interview that assesses the presence of psychiatric disorders, including the primary diagnosis of OCD. The MINI has good convergent validity with the Structured Clinical Interview for DSM Disorders. Following screening questions, if the presence of particular symptoms were positive, participants were further queried on the specific criteria for the mental disorder. The diagnosis of a particular mental disorder was based on meeting all the criteria set out in the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) (American Psychiatric Association, 2000).

The Y-BOCS (Goodman et al., 1989, Cronbach's alpha = .83) is a valid and reliable measure that was used to assess the nature and severity of OCD symptoms. This self-administered questionnaire asks the participant to rate the type and degree of obsessions and compulsions on a scale ranging from 0-4. A total score out of 40 comprised the sum of “obsessive” and “compulsive” subtotals, each with a possible maximum of 20. Scores on either the compulsion or obsessions inventory that fell between 0-10 indicate subclinical status; 11-20 as mild; 21-30 as moderate, and; 31-40 as severe symptoms.

Mood Measures

The BDI-II (Beck et al., 1996, Cronbach's alpha = .85) is a widely used 21-item self-report questionnaire to evaluate the existence and severity of depressive symptoms in adults and adolescents over the age of 13 years. The BDI-II possesses content, concurrent and construct validity. All of the items are based on components of depression, including mood, pessimism, self-dislike, indecisiveness, insomnia, and guilt, amongst others. Each item can be scored from 0-

3, and the total score is derived from a sum of the individual scores. Scores between 0-9 are indicative of minimal depression, 10-18 are indicative of mild depression, 19-29 are indicative of moderate depression, and 30-63 are considered severe.

The level of anxiety was assessed using the STAI (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), which is a widely used, valid and reliable measure of anxiety in adults.

Temporary anxiety caused by the test environment was assessed using the state component from this self-report questionnaire, which consists of 20-items, rated on a likert scale from 1 – 4. A score from 1-20 indicates minimal anxiety; 21-40, suggests slight anxiety; 41-60, reflects moderate anxiety and 61-80, index severe anxiety.

Neuropsychological Measures

Intelligence. The Wechsler Test of Adult Reading - 3rd edition (WTAR-III Wechsler, 2001) provides a reliable estimate of intelligence in clinical and research settings and has convergent and divergent validity. The WTAR-III has been demonstrated to be valid and reliable. Subjects are scored on their correct pronunciation of 50 words. The total score is the sum of correctly pronounced words.

Executive Function

The Stop-Signal Task (Logan & Cowan, 1984, Cronbach's alpha = .80) assesses inhibitory control. The stop-signal task is conceptualized as two processes. The “go” task requires subjects to respond to a target stimulus whereas the “stop” task requires subjects to withhold their response if they hear an auditory tone. The tone randomly occurs in 25% of the trials. This “horse-race” model defines successful inhibition as a function of the competition between the go-task and stop-task processes. If the go task process is completed before the stop-task, the subject responds to the target stimulus. If the stop task finishes before the go task, the

go task is inhibited. Typically, it is more difficult to inhibit a response when the auditory stop-signal is presented with a long latency after the presentation of the go stimulus. If the subject inhibits successfully, the stop signal delay is increased by 50 msec but if the subjects fails to inhibit, the stop signal delay is decreased by 50 msec. This tracking procedure ensures inhibition occurs 50% of the time. Response times to the go-signal (go reaction time, goRT) and stop-signal (stop-signal reaction time, SSRT) serves as dependent measures. The task is presented in 5 blocks of 32 trials. There are 24 go trials and 8 stop trials in each block. The mean latency of the goRT is obtained from 75% of trials in which no stops signal is presented. The SSRT is indirectly measured by calculating the difference between the mean reaction times of the stop signal delay and goRT (e.g., $SSRT = \text{delay} - \text{goRT}$). The dependent variables are the goRT and the SSRT.

The Stroop Colour and Word Test (Golden, 1976, Cronbach's $\alpha = .80$) is a valid measure used to assess cognitive inhibition. There are three tasks, all of which require reading printed words on a single sheet (within 45 seconds) that is organized in 5 columns. The first task requires reading colour words (i.e., 'red') that are printed in black ink. The second task requires naming the ink colours of 'XXXX's that are printed in red, green or blue. On the third task, called the 'interference task', subjects must name the ink colour of the printed word (e.g., respond "green" to the word "red" that is incongruently printed in green ink). This last task is a measure of a person's ability to suppress and withhold automatic reading of words in favour of processing a competing and more cognitively demanding task of naming the ink colour of the incongruous word. Subjects must name as many colours correctly within the specified time limit. Subjects typically take longer to name incongruent ink colour words (Golden, 1976).

The Tower of London Task (TOL) (Owen, Downes, Sahakian, Polkey, & Robbins, 1990, Cronbach's $\alpha = .77$) is a valid and reliable measure used to assess planning ability and problem solving. Using the least number of moves, subjects are to move several disks of varying diameters that are placed on three pegs of a board to match a target pattern without breaking two rules. The first rule requires participants to move one disc at a time and the second, forbids participants to place a larger disc on top of a smaller disc. The trials increase in difficulty by increasing the number of disks (ranging from 2 to 5) required to create the pattern. The measure of interest is the achievement score, which is defined as the total number of moves for a correct match.

The Wisconsin Card Sorting Task (WCST) (Berg, 1948) is a valid test of set-shifting with high internal consistency (Cronbach's $\alpha = .94$). The WCST consists of two decks of 64 cards with symbols on them that vary according to colour, form, and number. This task requires the subject to sort cards according to rules that change unpredictably. The test assesses the ability of abstract problem solving, shifting and maintaining, and feedback utilization. This is a 20 minute task. Dependent measures include total trials for completion, errors, perseverative errors, and non-perseverative errors.

The Iowa Gambling Test (IGT) (Bechara et al., 1994, Cronbach's $\alpha = .88$) captures decision-making skills. Subjects select 100 cards from four decks of cards with the goal of maximizing their profit starting from a \$2000 loan. Decks A and B are disadvantageous in that winnings and penalties are higher so in the long run, participants lose more money. Decks C and D are advantageous such that participants win less money, but their penalties are also lower, resulting in an overall gain over the long-term. Risk taking is defined as the total number of cards taken from the advantageous decks (i.e., C and D) minus those taken from the

disadvantageous decks (i.e., A and B). A negative value indicates riskier decision-making. Good decision-makers over time will choose more cards from the advantageous decks (i.e., after accounting for losses, the net winnings are positive) while impaired decision-makers will choose more cards from the disadvantageous decks (i.e., net winnings over time are negative).

Information Processing Speed

The Digit Symbol subtest of the Wechsler Adult Intelligence Test (WAIS) (Wechsler, 1999, Cronbach's $\alpha = .77$) requires the pairing of numbers and symbols based on a legend. The Symbol Search subtest of the WMS requires correct identification of a target stimulus from an array of 5 different visual stimuli. Both tasks are timed for 2 minutes. Participants are encouraged to work as quickly and as accurately as possible until they are told to stop. The number of correct identifications from both measures is combined to give an overall index of processing speed. Both tests are valid and reliable subtests of the WAIS.

Visuospatial Constructive Skills, Visual Memory and Learning

The Rey Osterrieth Complex Figural Test (RCFT) (Meyers & Meyers, 1942) is a widely used neuropsychological test for the evaluation of visuospatial constructional ability, visual memory and learning and has construct validity and adequate internal reliability (Cronbach's $\alpha = .80$). The RCFT consists of four test conditions: copy, immediate recall, delayed recall, and recognition. Subjects are given the RCFT stimulus card, and then asked to copy the figure. At both three minutes and 30 minutes after the copy trial, participants are instructed to draw the figure from memory. For the recognition trial, which follows the 30-minute recall trial, participants are presented with 24 elements from the complex figure and are asked to circle the items that correspond to the figure. The dependent variables include copy, immediate and delayed recall, and recognition scores.

Verbal Memory

The California Verbal Learning Test (CVLT) (Delis et al., 1987) is a neuropsychological test used to assess an individual's verbal memory and learning abilities with high internal reliability (Cronbach's $\alpha = .94$) and construct validity. There are several components in this test. The examiner reads aloud a list of 16 common words (List A) and asks the participant to recall as many words as possible from that list. This is repeated for five trials. Next, a short-delay is instigated by having the examiner recite a second and separate list of 16 words (List B) for the participant to recount. For the next trial, called the short-delay free recall (SDFR), the participant must produce from memory as many words as possible that were on List A. Next, four short-delayed cued recall trials asks participants to name words that fall in each of these categories: furniture, vegetables, ways of traveling and animals. There is then a delay of 20 minutes, after which time, long-term recall is measured by free recall and by semantic categories. A forced-choice (Yes or No) recognition trial precedes a 10-minute delay. Finally, participants are read two words at a time and are asked to choose which of the two words was from the first list.

Motor Speed

The Grooved Peg Board is a valid and reliable indicator of manual dexterity and motor speed (Heaton et al., 1992). Subjects are timed while they insert 25 grooved pegs into holes on a board, one at a time as quickly as possible. Subjects must first use their dominant hand (i.e., the hand they use to write), then switch to their non-dominant hand. Time to complete the task for each hand served as the dependent measure.

The Finger Tapping Test measures motor speed and has acceptable internal consistency with Cronbach's α ranging from .69 - .89. Participants are to tap their index finger as

quickly as possible for 10 seconds on a lever device with a counter (Heaton et al., 1992). Five to 10 trials are repeated for each hand until 5 consecutive trials are within 5 counts of each other. A practice trial and brief rests are given alternating every 3 trials. The average number of taps is the dependent variable.

Results

Statistical analyses were performed with IBM Statistical Package for the Social Sciences (SPSS) Version 19 for Windows (IBM, 2010). Demographic and clinical measures were analyzed using χ^2 and independent sample *t*-tests. To compare for differences between young adults with OCD and healthy controls, *t*-tests were conducted separately for each measure. For neuropsychological tasks such as the CVLT and RCFT, which have two or more repeated measures, a mixed ANOVA was performed with group as the between-subject factor and trials as the within-subject factor. Simple contrasts were used to explore significant differences between levels (or between the groups for levels). Pearson-moment correlations were computed in the young adults with OCD group to evaluate the relation between clinical variables and neuropsychological tests. In spite of the multiple group comparisons, statistical significance was assumed at $p < .05$, given the novelty and exploratory nature of this study.

We did not covary for anxiety because it is closely correlated with OCD (diagnostically OCD is classified an anxiety disorder) and individuals with OCD tend to be anxious. To conduct an ANCOVA, the covariate (i.e., anxiety) must be independent from the experimental effect (i.e., across groups) (Field, 2009); however, we found moderate to strong correlations between anxiety and performance, thus the shared variance between anxiety and OCD could not be statistically controlled for. However, to better understand the relationship of anxiety and

symptom severity on neuropsychological performance, we conducted multiple regression analyses using the enter method for set-shifting, decision-making, verbal and visuospatial data.

Missing data points were excluded from statistical analyses for some measures (i.e., excluded listwise). For example, one participant could not complete the Stroop Colour-Word Test because they declared colour blindness during the test. Another participant was not given the Iowa Gambling Test due to administrator error.

Demographic and Clinical Findings

The OCD group did not differ as compared to the healthy controls for age, length of education, and full scale IQ. OCD severity on the Y-BOCS indicated moderate severity of symptoms (range: 21-30; $M = 21.87$, $SD = 6.38$) among the OCD group that significantly differed from the non-clinical score observed by the healthy controls ($M = 4.33$, $SD = 6.46$, $p < .001$). The OCD group showed mild depressive symptoms ($M = 18.86$, $SD = 13.23$) relative to healthy controls ($M = 1.78$, $SD = 3.03$, $p < .01$) and were found to be extremely anxious ($M = 64.71$, $SD = 27.95$ vs. $M = 34.17$, $SD = 18.05$, $p < .01$). Demographic and clinical information is shown in Table 2.

Table 2. Demographic information of participants.

	OCD Mean (SD)	Controls Mean (SD)	<i>p</i> -value
Sample Size	N = 14	N = 13	
Age (years)	22.80 (2.70) (Range: 19-28)	23.6 (2.60) (Range: 19-28)	ns
Females: Male	8:6	8:5	ns
Education (years)	15.4 (2.5)	15.8 years (1.48)	ns
Full Scale IQ	107 (6.0)	104 (7.0)	ns
Y-BOCS (Total)	21.89 (6.38)	4.33 (6.46)	< .001
Y-BOCS (Obsessions)	10.89 (3.40)	2.56 (3.78)	< .001
Y-BOCS (Compulsions)	11.00 (3.23)	1.78 (3.03)	< .001
BDI-II	18.86 (13.23)	6.8 (6.48)	< .01
STAI-S	64.71 (27.95)	34.17 (18.05)	< .01

BDI-II, Beck Depression Inventory Second Edition, STAI-S, State-Trait Anxiety Inventory- State; FSIQ, Full scale IQ; Y-BOCS, Yale-Brown Obsessive Compulsive Disorder.

ns, nonsignificant; SD = standard deviation

Neuropsychological Findings

Individual performance on neuropsychological tests was compared to published norms where manuals were available (see Table 3). For the majority of tests, participant's performance in both groups fell within normal limits with the exception of the finger tapping test (non-dominant hand) and grooved peg board (dominant hand). However, there were no group differences on both the finger tapping test and the grooved peg board (see Table 4).

Neuropsychological findings in young adults with OCD and healthy control groups are presented in Table 4. For executive functions, independent sample *t*-tests yielded significant differences between groups on the set-shifting and decision-making tasks. Specifically, the young adults with OCD made more preservative responses than their same-age peers on the WCST, $t(24) = -2.11, p = .02$. On the IGT, young adults with OCD had a mean net loss of \$407.14 (419.41) compared to healthy young adults who had a net loss of \$41.67 (621.43),

showing a trend towards significance, $F(1, 24) = 3.17, p = .09$. Risky decision-making as defined by the net number of cards selected from decks $(C + D) - (A + B)$ was significant, $F(1, 24) = 6.35, p = .02$, indicating that young adults with OCD selected on average, more cards from disadvantages decks C and D. In regard to deck preference across all five blocks, the OCD group chose more cards from the disadvantageous decks A and B than healthy controls, $F(1,24) = 5.21, p = .03$, and similarly chose fewer advantageous cards from decks C and D, $F(1,24) = 7.6, p = .01$, as illustrated in Figure 1.

Using a mixed ANOVA, we further examined the pattern of net risky choices that were selected block-by-block (20 cards in each of 5 blocks) as the within-subjects factor and groups as the between-subject factor. The result was significant for the main block-by-block effect, $F(4,21) = 7.58, p < .001$. Contrasts for both groups revealed that the first 20 cards chosen were riskier cards (i.e., Block 1) than the last 20 cards (i.e., Block 5), $F(1, 24) = 9.24, p = .006$, suggesting that in both groups, performance improved over time. No other significant differences were observed from block to block. A main effect for groups was also found, indicating that overall, young adults with OCD chose fewer net advantageous cards than healthy controls $F(1,24) = 6.10, p = .02$. An interaction effect for block and group approached significance $F(4,21) = 2.12, p = .09$. Significant post-hoc contrasts revealed that only in Block 3, the OCD group ($M = -0.71, SD = 6.25$) chose more disadvantageous cards than the control group, ($M = 8.17, SD = 6.25$), $t(24) = 2.88, p = .008$.

Table 3. The number of participants who scored below average on various neuropsychological tests

Neuropsychological Test	OCD Worse than Normal	Healthy Controls Worse than Normal
CVLT Total Learning Slope 1-5	0	0
WCST Perseverative Responses	0	0
WCST Perseverative Errors	0	0
WCST Non-perseverative errors	0	0
Finger tapping, Non-dominant Hand	1	4
Tower of London Achievement Score	0	0
Groove Peg Board, Dominant Hand	3	1
Groove Peg Board, Non-dominant Hand	1	0
Stroop Test, Interference Condition	0	1
Information Processing Speed	0	0

Table 4. Neuropsychological performance of young adults with OCD and healthy controls

	OCD, Mean (SD)	Control, Mean (SD)	Statistic	p-values	Effect Size, r
<i>Set Shifting (Wisconsin Card Sorting Test)</i>					
Number of Trials	88.71 (21.38)	88.00 (19.43)	t(24) = -0.89	.93	0.18
Total Errors	10.14 (16.09)	15.75 (8.69)	t(24) = -0.85	.41	0.17
Perseverative Responses	2.93 (3.79)	0.33 (0.49)	t(24) = -2.11	.02*	0.40
Perseverative Errors	10.86 (8.25)	8.33 (5.73)	t(24) = -0.89	.38	0.18
Non-Perseverative Errors	9.21 (8.85)	7.42 (3.96)	t(24) = -0.65	.52	0.13
<i>Decision-Making (Iowa Gambling Task)</i>					
Disadvantageous Decks (A + B)	47.57 (8.86)	38.83 (10.68)	F(1, 24) = 5.21	.03*	0.18
Advantageous Decks (C + D)	52.36 (7.24)	61.7 (10.06)	F(1, 24) = 7.62	.01*	0.24
Net (CD – AB)	4.79 (16.00)	22.92 (20.67)	F(1, 24) = 6.35	.02*	0.21
Net Winnings	-407.14 (419.41)	-41.67 (621.43)	F(1, 24) = 3.17	.09	0.12
<i>Response Inhibition (Stop-signal Task)</i>					
SSRT	261.74 (43.36)	271.03 (79.57)	t(24) = 0.38	.71	0.08
Go RT	789.41 (150.32)	741.71 (170.84)	t(24) = -0.76	.46	0.15
<i>Cognitive Inhibition (Stroop Test)</i>					
Interference	57.07 (10.13)	50.05 (10.48)	t(22) = 0.10	.92	0.02
<i>Abstract Reasoning & Planning (Tower of London)</i>					
Achievement Score	10.57 (3.11)	10.42 (3.32)	t(24) = -0.12	.90	0.02
<i>Memory – Verbal (California Verbal Learning Test)</i>					
Total Words Trails 1-5	55.79 (10.48)	63.3 (5.51)	t(25) = 2.31	.03*	0.42

Across Trial Consistency	84.2 (10.19)	91.46 (5.44)	t(25) = 2.26	.03*	0.41
<i>Visuospatial constructive Ability and Memory (Rey Osterrieth Complex Figure Test)</i>					
Copy Raw Score	30.86 (4.64)	33.46 (2.85)	t(25) = 1.74	.09	0.11
Immediate Raw Score	18.18 (6.36)	21.69 (3.95)	t(25) = 1.51	.10	0.11
Delayed Raw Score	17.43 (7.15)	21.81 (3.99)	t(25) = 0.87	.06	0.13
Copy Time	182.00 (107.70)	110.42 (39.48)	t(25) = -2.26	.03*	0.42
<i>Motor Speed & Manual Dexterity (Finger Tapping & Grooved Peg Board)</i>					
Time (s) Dominate Hand (Finger Tapping)	44.79 (10.75)	43.00 (10.75)	t(25) = -0.43	.67	0.09
Time (s) Non-Dominant Hand (Finger Tapping)	46.43 (11.71)	41.61 (8.79)	t(25) = -1.20	.24	0.23
Time (s) GPB – Dominant Hand	39.36 (16.46)	44.92 (9.91)	t(25) = 1.05	.30	0.21
Time (s) GPB – Non-Dominant Hand	43.21 (13.69)	46.08 (9.29)	t(25) = 0.60	.53	0.12
<i>Information Processing Speed (Wechsler Adult Intelligence Scale)</i>					
PSI	112.86 (16.32)	123.46 (17.69)	t(25) = 1.62	.12	0.31

* p < 0.05; ** p < 0.01; SD – standard deviation
SSRT, Stop-signal reaction time.

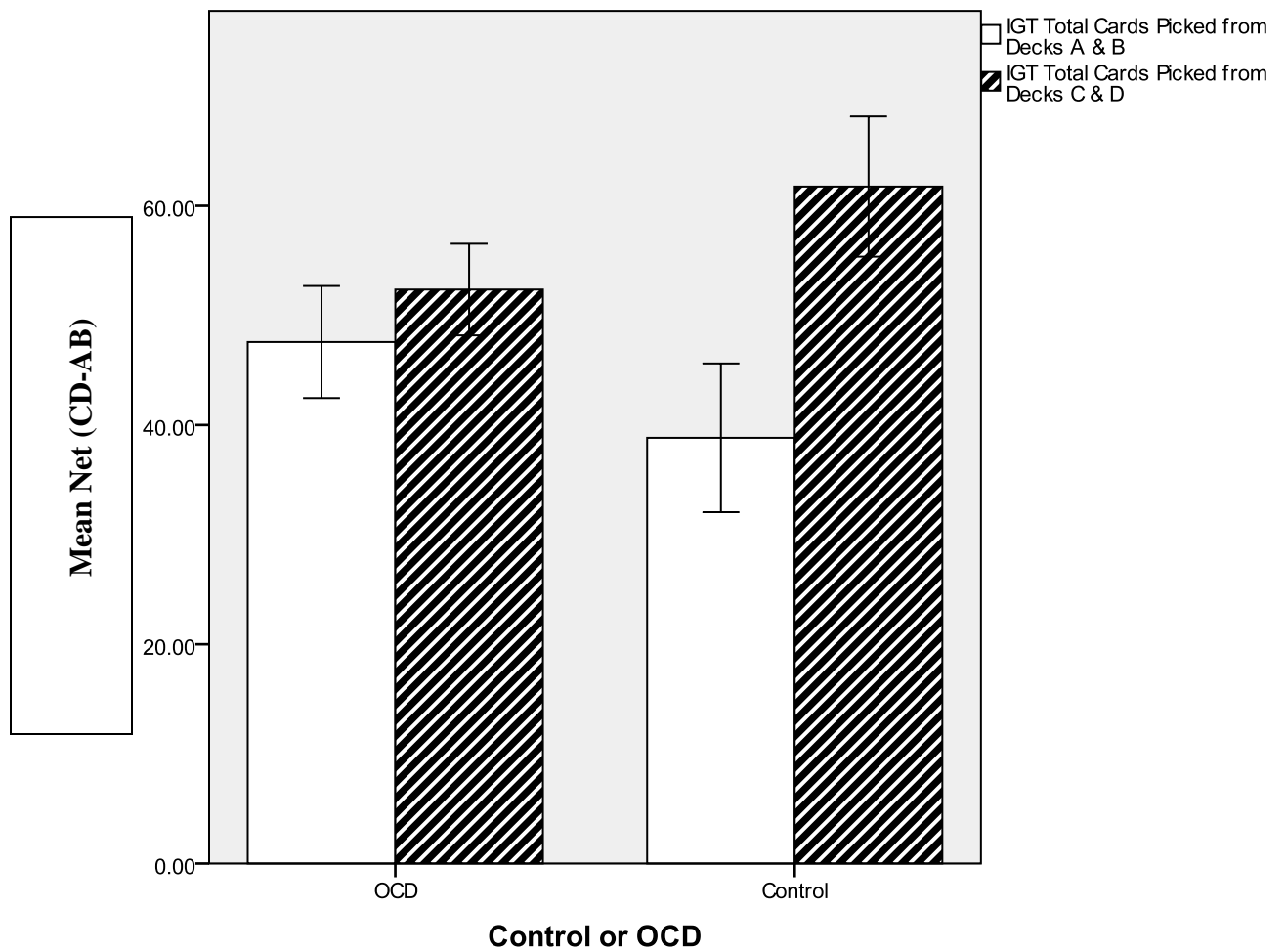


Figure 1. Young adults with OCD chose more cards from riskier decks (A + B) and fewer cards from advantageous decks (C + D) than healthy controls, resulting in a net loss of loaned money compared to healthy controls. Error bars denote standard errors.

No significant differences emerged between the young adults with OCD and healthy controls (see Table 4) on executive function tasks of behavioural inhibition (stop-signal task), cognitive inhibition, and abstract reasoning and planning. In summary, executive dysfunction in young adults with OCD was observed for perseverative responding on the set-shifting task and poorer decision-making and learning on the IGT as compared to healthy controls.

On the memory measure (i.e., CVLT), young adults with OCD remembered fewer total number of words than healthy controls, $t(25) = 2.31, p = .03$ and their performance was not consistent across trials, $t(25) = 2.26, p = .03$, indicating possible encoding or retrieval problems. With regard to visuospatial-constructive ability (i.e., RCFT), a 2 (group) x 3 (trials) mixed ANOVA yielded trends of significance between group for copy raw score, $F(1, 25) = 3.03, p = .09$ and 30-minute delay score, $F(1, 25) = 3.78, p = .06$, indicating lower visuospatial constructive ability and recall in young adults with OCD. No within level effects across trials (i.e., copy, immediate recall and delayed recall) were evident. However, young adults with OCD took significantly longer to copy the complex figure, $t(25) = -2.26, p = .03$. To summarize, verbal and visuospatial memory measures indicated deficits in young-adults with OCD.

No performance differences were observed on other neuropsychological tests such as motor speed (i.e., finger tapping test: dominant hand, , non-dominant hand,) and manual dexterity (grooved peg board) between groups. Additionally, no significant differences between groups were observed for information processing speed on the WAIS.

Clinical and Psychological Symptoms and Neuropsychological Performance in Young Adults with OCD

Y-BOCS obsessions, compulsions and total scores were associated with performance on some neuropsychological tasks. For obsessions, only trends were observed between obsession severity and the selection of disadvantageous cards on the IGT (i.e., Deck A), $r = -.52, p = .06$ (i.e., more obsessive tendencies was associated with fewer risky card selections), number of incorrect answers on the RCFT, $r = .53, p = .06$ (i.e., higher severity of obsessions was related to more incorrect answers on the RCFT), and the SSRT on the stop-signal task, $r = .47, p = .09$ (i.e., higher obsessions was related to longer SSRT) and digit-symbol subtest of the WAIS, $r = -.51, p = .06$ (i.e., more obsessive thoughts were associated with lower scores on a component of the information processing speed index). Compulsions were related to more errors on the RCFT, $r = .61, p = .03$, and a trend in longer copy time on the RCFT, $r = .51, p = .07$. Similarly, total Y-BOCS score was associated with a higher number of mistakes on the RCFT, $r = .59, p = .03$.

Pearson product-moment correlations assessing the relation among depression and anxiety on neuropsychological performance were summarized in Table 4. Level of depression was positively correlated with the Y-BOCS total OCD severity score ($r = .57, p = .035$) and obsessions ($r = .57, p = .033$), but showed trends for compulsions ($r = .52, p = .059$).

State anxiety seemed to affect performance in young adults with OCD. Positive correlations were noted between anxiety and the number of cards that were drawn from risky decks A & B on the IGT, suggesting that higher anxiety was associated with riskier card selection. Additionally, increasing anxiety correlated with a higher number of trials needed to complete all six categories on the WCST, total errors, preservative responses, perseverative errors, and non-perseverative errors. These indices reflect the negative impact of anxiety on set-

shifting ability. Moreover, a negative correlation was observed between state anxiety and CVLT total learned words, (i.e., higher anxiety was related to fewer remembered words); average time on the finger tapping test in the non-dominant hand (i.e., higher anxiety led to shorter time to complete the task); IGT decks D, net decks (CD-AB) (i.e., higher anxiety was associated with fewer advantageous cards picked from deck D and from net total decks), and the TOL achievement score (i.e., higher anxiety was related to lower scores on the planning ability and abstract reasoning task).

Depression levels in young adults with OCD correlated significantly with scores on the total words recalled across five trials on the CVLT, $r = -.63, p < .05$, reflecting lower verbal memory recall with higher depression scores. In addition, visual recognition recall was negatively impacted by higher depression levels, $r = -.63, p < .05$ and similarly TOL achievement score was adversely affected by higher reported depressive symptoms, $r = -.62, p < .05$.

Correlations were also computed between neuropsychological measures and duration of illness in young adults with OCD. Duration of illness was related to the net selection of decks (CD – AB), $r = -.55, p < .05$, indicating good decision making is less likely the longer one has OCD. Longer duration of illness was associated with a higher number of cards selected from disadvantageous decks (A & B), $r = .57, p < .05$. By the same token, a negatively correlated trend was observed between illness duration and the number of cards selected from advantageous decks, (C & D), $r = -.51, p = .06$, suggesting that the longer the illness, the fewer number of “good cards” selected.

Table 5. Correlation coefficients between severity, illness duration, depression, anxiety and neuropsychological tests for young adults with OCD.

Measures	OCD				Controls	
	Y-BOCS Total	Illness Duration	BDI-II	STAI-State Anxiety	BDI-II	STAI- State Anxiety
Y-BOCS Obsessions	.964**	-.429	.572*	-.080	-.097	-.360
Y-BOCS Compulsions	.960**	-.220	.516	.009	-.465	-.036
Y-BOCS Total	1.00	-.340	.566*	-.038	-.275	-.227
BDI-II	.566*	.344	1.00	.462	1.00	-.196
CVLT Total Learning Slope 1-5	.020	-.258	-.625*	-.631**	-.038	-.108
Finger Tapping Dominant Hand	-.318	-.196	-.457	-.091	.001	.141
Finger Tapping Non- Dominant Hand	-.137	-.298	-.419	-.588*	.012	-.072
Groove Peg Board Dominant Hand	-.101	.195	.105	-.093	-.186	.057
Groove Peg Board Non- dominant Hand	-.178	.174	.037	-.220	-.297	.534
IGT Cards Drawn from Deck B	.062	.285	-.059	.629*	.353	-.244
IGT Cards Drawn from Deck D	-.201	-.315	-.317	-.654**	.210	.103
IGT Cards Drawn from Deck A & B	-.295	-.569*	-.189	.549*	.291	-.246
IGT Cards Net Winnings (CD-AB)	.262	.547*	.162	-.544*	-.278	.228
RCFT Copy Score	-.028	-.042	.033	-.258	-.212	-.138
RCFT Immediate Score	.057	.123	-.097	-.351	-.222	.299
RCFT Delayed Raw Score	.028	-.168	-.059	-.413	.042	.066
RCFT Copy Time	.461	-.241	-.043	-.188	.148	-.181
RCFT Total Recognition Recall	-.258	-.168	-.625*	-.341	-.009	.376

Stop-signal SSRT	.412	-.210	.118	.065	-.203	-.097
Stop-signal GoRT	.270	-.078	.352	.299	.576*	.104
Stroop Interference	-.317	-.303	-.292	-.076	.150	-.257
Tower of London Achievement Score	-.395	-.110	-.623*	-.708**	-.040	.167
WCST Total Trials	.042	.260	.320	.662*	.447	-.363
WCST Total Errors	.061	.295	.285	.658**	.466	-.316
WCST Perseverative Responses	-.151	.303	.009	.643*	.376	-.324
WCST Perseverative Errors	-.024	.339	.255	.604*	.360	-.329
WCST Non- perseverative errors	.126	.213	.287	.639*	.110	-.128
CVLT, California Verbal Learning Test; IGT, Iowa Gambling Task; RCFT, Rey-Complex Figure Test; WCST, Wisconsin Card Sorting Test; * $p < .05$; ** $p < .01$						

Multiple regression analyses were conducted with anxiety entered in the first block, YBOCS entered in the second block and group entered in the third block for dependent measures on the decision-making, set-shifting, verbal and visuospatial memory tasks. State anxiety was a significant predictor in the decision-making, set-shifting, verbal memory and visuospatial tasks. Table 6 summarizes the results.

Table 6. Standardized coefficients from the multiple regression analysis with STAI-state, Y-BOCS and Group as predictors.

	Model 1			Model 2			Model 3		
	<i>B</i>	<i>SE B</i>	β	<i>B</i>	<i>SE B</i>	β	<i>B</i>	<i>SE B</i>	<i>B</i>
<i>Set shifting - Perseverative responses (Wisconsin Card Sorting Test)</i>									
STAI- State	.08	.02	.69**	.08	.02	.75**	.08	.02	.74*
Y-BOCS				-.03	.05	-.11	-.04	.08	-.13
Group							.18	2.04	.03
R^2		.48			.49			.49	
<i>F</i> for change in R^2		19.08**			0.37			0.01	
<i>Decision-making – Cards selected from Decks A & B (Iowa Gambling Task)</i>									
STAI- State	.16	.06	.50*	.20	.07	.60*	.17	.08	.52*
Y-BOCS				-.18	.19	-.22	-.33	.28	-.39
Group							4.94	6.84	.27
R^2		.25			.28			.30	
<i>F</i> for change in R^2		6.89*			0.98*			0.20	
<i>Decision-making – Net Cards selected from Decks (CD-AB) (Iowa Gambling Task)</i>									
STAI- State	-.31	.12	-.51*	-.36	.13	-.59	-.31	.15	-.50
Y-BOCS				.27	.35	.17	.60	.53	.37
Group							-10.74	12.89	-.31
R^2		.27			.28			.31	
<i>F</i> for change in R^2		7.69*			0.26			0.89	
<i>Verbal memory –Total words recalled Trials 1-5 (California Verbal learning Test)</i>									
STAI- State	-.15	.07	-.43*	-.12	.08	-.34	-.09	.09	-.24
Y-BOCS				-.179	.21	-.19	.01	.32	.01
Group							-6.04	7.68	-.31
R^2		.19			.22			.24	
<i>F</i> for change in R^2		4.88*			0.73			0.62	

Visuospatial constructive ability (Rey-Osterrieth Complex Figure Test)

STAI- State	.43	.71	.13**	-.55	.71	-.17	-.65	.82	-.20
Y-BOCS				5.23	1.86	.61*	4.71	2.86	.55
Group							17.09	69.48	.09
R^2		.12			.30			.30	
F for change in R^2		0.36			7.90*			0.61	

* $p < 0.05$; ** $p < 0.01$;

Discussion

This thesis sought to characterize the cognitive profile of young adults with OCD. The first hypothesis that young adults with OCD would exhibit EF deficits when compared to healthy controls was partially supported by the significant performance deficits observed for decision-making and perseverative responding; however, no differences emerged for inhibitory processes, set-shifting, abstract reasoning and planning. Similarly, and consistent with the second hypothesis, there were group differences in verbal memory, across-trial consistency for verbal recall, copy speed for visuospatial construction and trends toward significance for delayed visuospatial memory, while information-processing speed, motor speed, and manual dexterity were comparable between groups. The second objective was to examine the relationship between clinical and neuropsychological variables. We expected there would be no associations between obsessions/compulsions and neuropsychological performance. On most tests, groups did not differ; however, YBOCS symptom severity was related to decision-making, visuospatial recall and visuospatial copy time. There were also significant associations among state anxiety, depression, illness duration and some neuropsychological measures. Specifically, there was a large observed effect between state anxiety and some measures. Furthermore, in a regression analysis, state anxiety was observed to be a significant predictor for performance on measures where group differences were observed. Overall, the results suggest that performance deficits in young adults with OCD could have been accounted for by state anxiety.

Decision-Making

Our results concur with some extant studies that the OCD group showed more risky decision-making on the IGT when compared to healthy controls. Similar to Cavedini et al.'s (2006) study, we report that risky decision-making was not related to depression in the OCD

group; however, it was associated with state anxiety suggesting that poor decision-making may be accounted for by anxiety. This finding suggests that under high arousal, and subject to novel and ambiguous situations, individuals with OCD do not appear to be influenced by delay incentives and are more influenced by immediate rewards that are nevertheless intrinsically less advantageous to them.

Decision-making based on reward and punishment depends on intact frontostriatal circuits encompassing the orbitofrontal cortex, caudate and basal ganglia (Aouizerate et al., 2004; Blanchette & Richards, 2010; Huey et al., 2008). Aberrant activation in the orbitofrontal cortex, particularly the ventral striatum during gain and loss anticipation on a monetary incentive delay task was recently reported in adults with OCD (Jung et al., 2011). Psychophysiological evidence (i.e., somatic markers) of disadvantageous decision-making on the IGT involves markedly reduced galvanic skin response in patients with ventromedial prefrontal cortex lesions (Bechara et al., 1994). Psychophysiological evidence has also been replicated in individuals with OCD; specifically, reduced galvanic skin conductance was observed in tandem with poor performance on the IGT when compared to controls (Starcke, Tuschen-Caffier, Markowitsch, & Brand, 2009; Starcke et al., 2010).

The neurobiology of anxiety is also associated with orbitofrontal cortex functioning. In healthy individuals, high state anxiety was related to a 32% increase in neurochemicals in the orbitofrontal cortex (Grachev et al., 1998). In OCD, state anxiety may exacerbate the disruption of somatic marker production in the orbitofrontal cortex that guides sound decision-making. However, due to the shared variance between OCD and anxiety we cannot be certain whether performance differences were a result of OCD diagnosis or the effects of state anxiety. Our correlation and regression analyses nevertheless supports to the mediating role of anxiety in

decision-making. For instance, anxiety accounted between 30-43% of the variance on IGT measures and was a significant predictor of IGT performance. Thus, it is important to parse out the effect of state anxiety in future designs by comparing varying levels of anxiety (e.g., high vs. low) in young adults with OCD.

In terms of learning, the data (refer to Figure 2) indicate that healthy controls chose riskier cards early in the game (i.e., Block 1: cards 1-20), presumably because they were learning the values of the decks. Later on however, the controls chose more advantageous cards (from Decks C & D) between blocks 2-5 (cards 21-100). On the other hand, young adults with OCD took longer to learn to stay away from the riskier decks, requiring up to 60 card choices (i.e., Block 3: cards 41-60) compared to healthy controls who only made 20 net risky card selections before selecting net advantageous cards. The observation that young adults with OCD took longer to learn the riskiness of decks A and B may be the result of their insensitivity to loss (Jung et al., 2011). Other physiological studies have also shown that reduced galvanic skin response and heart rate alterations are only observed prior to selecting disadvantageous cards and not prior to selecting advantageous cards (Starcke et al., 2009). In light of these recent findings, young adults with OCD may be less sensitive to negative incentive processing. Another interpretation of the findings is that the presence of high state anxiety in this sample of OCD participants could have played a role in the protracted learning time required by the OCD group. That is, individuals with OCD may need more trials to learn the implicit rules of the IGT because their mental effort was consumed by having to deal with anxious thoughts and feelings.

The finding of poor decision-making in young adults with OCD is somewhat inconsistent with prior evidence suggesting that individuals with other anxiety disorders (i.e., social anxiety disorder) are risk-averse and tend to make decisions that facilitate the avoidance of threat (Lorian

& Grisham, 2010; Lorian & Grisham, 2011; Maner & Schmidt, 2006; Maner et al., 2007). However, these studies looked only at trait and not situational anxiety and therefore, at this point, it is unclear the nature of the association between state anxiety and decision-making. It has been reported that anxious individuals are more sensitive to negative interpretations and tend to inflate a higher probability of negative events and personal involvement (Blanchette & Richards, 2010). Hence anxiety has been postulated to be responsible for risk-avoidance behaviours in anxious individuals (Lorian & Grisham, 2010). Avoiding real-life anxiety provoking stimuli such as germs in individuals with OCD with contamination preoccupations might be construed as risk-avoidance; however, these risks are conscious decisions with known consequences to the individual (i.e., reduction in anxiety). However, in the IGT, the outcomes of decisions are not directly known but must be experienced by the subject (i.e., must be learned) in order to understand the costs of their decisions (Upton, Bishara, Ahn, & Stout, 2011). Therefore the outcomes are not explicitly known to the subject and this lack of volitional control at least in the first part of the IGT is different and far removed from intentional decisions of avoidance. In studies that used risky decision-making paradigms with explicit reward and punishment probabilities at the outset of the game (e.g., Game of dice, GDT), adults with OCD performed similar to controls (Starcke et al., 2010) but performed worse than controls on the IGT. This implies that prior knowledge of probabilities prevented risky decision-making in individuals with OCD. A paucity of studies currently exists that examine reward/punishment contingencies and the mediating effect of anxiety, thus new studies in this area is a fruitful area to explore.

Our findings also suggest that the duration of illness was related to decision-making in young adults with OCD. A longer duration of illness was associated with riskier deck selection and hence, more likelihood of loss. This finding has treatment implications especially if risky

decision-making on the IGT translates into similar poor financial decisions outside the laboratory. As a percentage of initial investment, young adults with OCD showed a 20% net loss at the end of the IGT while healthy controls had a 2% net loss. This large difference can have negative financial consequences, especially if such poor decision-making pervades a person's choices over time. This study provides preliminary evidence that young adults with OCD under stress and ambiguous situations are less sensitive to punishment and reward cues.

Set-Shifting Abilities

Our findings of comparable performance in set-shifting between young adults with OCD and healthy controls are similar to the findings in child studies (Behar et al., 1984; Britton et al., 2010; Ornstein et al., 2010; Shin et al., 2008). On several WCST indices in this study, there were no observable differences in the number of trials to completion, the number of perseverative errors, total errors and non-perservative errors. However, young adults with OCD committed more perseverative responses compared to controls; however state anxiety accounted for 41% of the variability in performance. Perseverative responding may closely relate to the repetitive clinical obsessions and compulsions endorsed in OCD. A proposition that was put forth links compulsive perseveration to the failure of frontostriatal circuits in inhibiting motor and cognitive programs in the basal ganglia (Fineberg et al., 2010). In support of this, perseveration has been observed in healthy adults who sustained damage to the head of the caudate, and areas surrounding the basal ganglia (B. R. Coetzer, 2004). In this sample however, perseverative responding in young adults with OCD fell within the normal range of performance (99th percentile), thus, the observed group differences may have had to do with the interaction of high state anxiety and clinical symptoms. In summary, set-shifting likely falls within the normal

range in young adults in OCD, a finding that concurs with many adult neuropsychological studies (refer to Table 1).

Inhibitory Processes

Recently, inhibitory processes were put forth as possible endophenotypes for the underlying pathophysiology of OCD (Chamberlain et al., 2005); however our results do not support this hypothesis. Rather, our finding of intact inhibitory control was consistent with the extant child studies (e.g., Beers et al., 1999; Ornstein et al., 2010; Woolley et al., 2008). Our sample of young adults with OCD had lower SSRT than did the healthy controls (i.e., they were faster), albeit this finding was not statistically different between the groups. This finding may be due to the adequate functioning (i.e., based on self-report, socially engaged, working or attending university) of the young adults with OCD included in this study. A similar finding was reported in untreated, non-clinical adults with OCD with high average IQ and reportedly moderate OCD symptoms (Krikorian, Zimmerman, & Fleck, 2004). Similarly, in our study, the young adults with OCD had moderate obsessive-compulsive severity, shorter illness duration and were recruited from the community, and therefore may have had better executive function control than more severe cases from clinical populations in other studies (Abramovitch, Dar, Schweiger, & Hermesh, 2011; Lacerda et al., 2003). Future studies should recruit young adults with OCD from clinical settings with a wider range of severity to assess whether intact inhibitory control is generalizable.

Only a few studies have found the Stroop test to be sensitive to cognitive inhibition in individuals with OCD (Martinot et al., 1990; Rao et al., 2008) but our findings concur with many others studies that have found comparable performance relative compared to healthy controls, some of which criticized the Stroop test as being an insensitive measure of orbitofrontal cortex

functioning (MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003; Moritz et al., 2008; Moritz, Kloss, & Jelinek, 2010). Our findings may be explained by a recent imaging study that also found no performance deficits in individuals with OCD on the Stroop task as compared to controls (Ciesielski et al., 2011). Ciesielski et al. (2011) reported increased activation in the lateral prefrontal and anterior cingulate areas in OCD cases, suggesting the recruitment of other brain regions which could serve as a means of compensatory adaptations. Thus, hyper-activation of other neural structures may have helped young adults with OCD to maintain top-down attentional control in the face of interfering stimuli.

When we consider that both inhibitory tasks given in this study showed comparable performance in the OCD and control groups, it suggests that inhibitory control is intact in this young cohort with OCD. It is possible that inhibitory deficits found in other studies may be due to the sampling of older participants. This implies that inhibitory control deficits may manifest at a later stage in life. Volumetric brain studies in healthy individuals have confirmed regional brain changes along an inverted U-shaped trajectory, with volumetric increase in young adulthood that plateaus with precipitous shrinkage beginning in middle adulthood (Raz, Ghisletta, Rodrigue, Kennedy, & Lindenberger, 2010). Volumetric shrinkage due to normal aging have been associated with decrements in information processing speed and inhibition (Drag & Bieliauskas, 2010); hence it is important to instigate longitudinal experimental designs in individuals with OCD to shed light on the trajectory of inhibitory control.

Verbal Memory and Learning

Unlike children and adolescents with OCD who showed no deficits in verbal memory (Andres et al., 2007; Beers et al., 1999; Ornstein et al., 2010), performance on the CVLT indicates emerging deficits in young adults with OCD. We found significantly lower total words

recalled and across trial consistency in the OCD group. This corresponds to a number of previously published studies in adults with OCD (Hwang et al., 2007; Lacerda et al., 2003; Schmidtke et al., 1998), but contradicts others (see Table 1).

Our correlational analyses support the hypothesis that poor verbal recall in young adults with OCD may have been related to the anxiety and depression symptoms reported. We found a negative correlation between the total number of words recalled and state anxiety which indicates the adverse association between anxiety and verbal recall performance. Results from a multiple regression suggests that verbal memory performance may be a function of state anxiety in young adults with OCD. It may be that anxiety exerts a “cognitive price” by disrupting and diverting attention to threat-related thoughts and pre-occupations (Abramovitch et al., 2011; Eysenck & Byrne, 1994), and as a result, reduces the individual’s memory encoding capacity.

Similarly, our findings showed that depressive symptomatology also significantly correlated negatively with CVLT recall. Depression has been associated with volume reduction in the frontal lobe (7%) and medial orbitofrontal cortex (32%) (Lesser & Chung, 2007) and atrophy in these areas was associated with disturbances in memory. Further, neuroimaging evidence found lower cerebral glucose metabolism in the caudate, thalamus and hippocampus in participants with both OCD and major depression than in subjects with a primary diagnosis of OCD (Saxena et al., 2001) indicating the negative impact of the presence of co-morbidity. Other studies that have looked at concurrent depression in OCD reported that depression accounted for impaired neuropsychological performance (Basso et al., 2001; Segalàs et al., 2008); however other research did not differentiate between individuals with co-morbid OCD and major depressive disorder as compared to healthy controls (Cavedini et al., 1998; Christensen et al., 1992; Dirson et al., 1995). Future studies that evaluate individuals with OCD with and without

depression will help clarify the nature and extent of verbal memory dysfunction in these disorders.

Information Processing Speed

Although information processing speed was found to be slower in individuals with OCD than in healthy controls, the results were statistically insignificant. It may be that information processing is considered a non-specific executive function that is orchestrated by multiple, parallel distributed networks in the prefrontal cortex (B. L. Miller, 2007; E. K. Miller & Cohen, 2000) and is not heavily affected by aberrant orbitofrontal pathophysiology characteristic of OCD. Further, the mediating role of age on prefrontal processes should be considered. The fastest rate of volumetric decline has been observed in the prefrontal cortex relative to slower declines that were observed in the temporal, parietal and occipital structures (Haug & Eggers, 1991; Raz et al., 2010). Therefore, declines in processing speed which is dependent on prefrontal cortex integrity likely affects other cognitive processes cognitive (Salthouse, 1996). Several studies have reported that up to 71% of the shared variance was observed between information processing speed and performance on cognitive tasks in various domains (Rozas, Juncos-Rabadan, & Gonzalez, 2008; Verhaeghen & Salthouse, 1997). Two mechanisms have been proposed to explain the findings (Salthouse, 1996). Firstly, cognitive function is dependent on time-limited processes that cannot be executed when processing speed becomes inefficient with age. Secondly, cognitive abilities that require simultaneous processes become redundant when earlier cognitive processes are lost by the time they are required for later processes. In light of the above findings, young age may be a protective factor with regards to intact information processing speed, and this theory may account for some of the findings of impairment observed in studies that sampled an older cohort of adults with OCD. Hence, we

advocate for a longitudinal design in future experiments where the mediating role of age on processing speed and cognitive function can be shown.

Motor Speed and Manual Dexterity

Contrary to some recent findings of slow motor processing in adults with OCD (Bedard et al., 2009; Burdick et al., 2008), our findings showed that young adults with OCD were not impaired, a finding that mirrors the performance of children and adolescents with OCD (Beers et al., 1999; Ornstein et al., 2010). It is possible that impaired motor speed in the studies above are the result of sampling older participants (i.e., the average age in each study was 35 and 41, years respectively). Motor slowing has been demonstrated in healthy aging adults (Raz et al., 2005) but slowing generally does not appear until late adulthood. In previous studies, depression was postulated to explain slow motor and processing speed observed in adults with OCD (Basso et al., 2001; Bedard et al., 2009; Burdick et al., 2008). In our correlational analyses, depressive symptomology was not related to either motor speed or processing speed.

Visuospatial Constructive Abilities and Memory

Trends towards significance were seen in visuospatial constructive ability in young adults with OCD, based on their lower scores on the copy trial of the RCFT. This finding corresponds to one child study (Andres et al., 2007) and several adult studies (Boone et al., 1991; Martinot et al., 1990). Visuospatial memory in young adults with OCD may also be compromised following a 30-minute delay which was consistent with all child studies conducted and some adult studies (Deckersbach et al., 2000; Savage & Rauch, 2000). Further, notable differences in time to copy a complex figure were observed; young adults with OCD were prominently slower than healthy controls. Findings from the regression analysis suggests that the speed of copying is possibly a

function of both state anxiety and clinical symptoms where state anxiety acts to improve performance while the severity of obsessions/compulsions hinders copying speed.

The relationship between clinical and neuropsychological measures

To address the second objective of this study, correlational analyses were conducted and some associations between clinical variables on select cognitive functions emerged. For example, obsessions negatively correlated with the number of disadvantageous cards picked, suggesting that internal preoccupations might be beneficial to some degree in OCD. Incessant mental thoughts may have distracted the tendency to respond to immediate prospects, and consequently reduced risky decision-making.

In this work, slow copy speed and poor accuracy on the RCFT were related to obsessive-compulsive symptoms. The presence of obsessions and compulsions may interfere with cognitive processing because individuals are subject to continual mental checking (Hymas et al., 1991; Sawle, Hymas, Less, & Frackowiak, 1991); hence additional time is required to process the task at hand. The preoccupation with internal thoughts is also presumed to divert the focus required to pay attention to details of the complex figure and thus affected performance accuracy.

Some evidence for the relationship between clinical and neuropsychological performance has been previously reported. For example, negative associations between YBOCS scores and executive function composite scores (Abramovitch et al., 2011), verbal memory (Segalàs et al., 2008) and visuospatial memory (Lacerda et al., 2003) have been published. Further, neural correlates in the orbitofrontal and ventral striatum have also been associated with thoughts related to individual obsessions and compulsions (Chamberlain et al., 2008; Lacerda et al., 2003) and in symptom provocation paradigms (Adler et al., 2000; Breiter et al., 1996; Rauch et al.,

2002; Rotge et al., 2008). Finally, the reduction in obsessive-compulsive symptoms following behavioural therapy correlated with improved neuropsychological performance in adults with OCD (Kuelz et al., 2006). In children and adolescents with OCD, cognitive impairments in visuospatial recall, and copy time resolved following six months of treatment thus, supporting the benefits of targeting symptoms early (Andres et al., 2008). Hence, studies examining cognitive performance in young adults with OCD pre- and post-treatment might be an avenue of research that could elucidate how clinical symptoms affect cognition.

Study Strengths and Limitations

Several strengths are present in this study design. This is the first study to examine cognition in young adults with OCD and helps to bridge the gap in research between child/adolescent and adult OCD research. In this sample of young adults with OCD, decision-making deficits emerged and this is consistent with several published accounts in adults with OCD (Cavallaro et al., 2003; Starcke et al., 2010) and with the orbitofrontal cortex model of neuropathophysiology of OCD. This study also found that young adults with OCD however are prone to high state anxiety, which was associated with poor decision-making, thus highlighting the role of anxiety in OCD. Another strength of this study is the use of a wide range of psychometrically-sound instruments with well known neural correlates and sensitivity in individuals with OCD. Finally, we believe that sampling a young cohort, presumably when cognitive functions are fully developed has the advantage of ruling out deficits based on immature cognitive systems and/or compensatory related learning on cognition. Even healthy young adults exhibit compensatory ‘scaffolding’ whereby other neurocircuitry is recruited in response to challenge or inefficiency in particular neural structures, especially in the frontal cortex (Park & Reuter-Lorenz, 2009). By sampling young adults and then following their

cognitive trajectory over time will help elucidate whether compensatory scaffolding occurs, and hence may explain why some of the findings in the adult OCD literature remain unimpaired in certain domains.

Some limitations of the study need mention. First, generalizing conclusions from this thesis is limited because of the small sample size; a larger study replicating these findings is necessary to substantiate our hypotheses. A large sample size will also improve the power of statistical tests to detect differences and improve effect sizes. Moreover, a larger sample size might afford the exclusion of medicated individuals and those who presented with other clinical symptoms. It could be argued that the presentation of other clinical symptoms could have accounted for some of the findings in this study; however, the range of symptoms (e.g., depression, substance use) was heterogeneous with theoretically different mechanisms of action. Thus, it is unlikely that these different disorders interacted to affect performance in a specific way.

Causal inferences are limited based on the design of this study. It is not possible to determine whether performance deficits observed in this study are trait markers of OCD or whether group differences were attributable to anxiety. We noted that we could not statistically separate the shared variance between OCD status and state anxiety using ANCOVA. ANCOVA assumes that a covariate (e.g., state anxiety) can not be statistically significantly different between the treatment (e.g., OCD) and control (Field, 2009; G. A. Miller & Chapman, 2001). One way to account for this confounding factor is for future studies to match state anxiety in individuals with OCD and controls by using anxiety provoking paradigms (e.g., public speaking manipulation) in healthy controls. In the above suggested design, state anxiety can then be statistically separated to reveal whether OCD pathophysiology affects cognition. Future studies

should also obtain pre- and post-anxiety measures in future studies would help reveal the temporal effect of anxiety on performance. A final limitation is that the significant statistical deficits that were observed in this sample do not necessarily imply clinical significance. It appears that the decrements in performance in the OCD group fell within the normal range of performance, thus the findings in this study has limited clinical utility.

Future Directions

The implications of these results support future longitudinal studies that can track cognitive function in young children as they mature. This developmental trajectory will help clarify whether specific cognitive impairments manifest at a certain stage in life in individuals with OCD. Only one study (Grisham, Anderson, Poulton, Moffitt, & Andrews, 2009) thus far has examined the developmental trend in adolescents to adulthood and found that adults who still met a diagnosis of OCD were impaired in visuospatial constructive ability, some forms of executive function, memory, and exhibited motor slowness. Furthermore, a longitudinal design would help address whether cognition can be considered a reliable measure of trait vulnerability. Over time, some individuals will recover from their OCD symptoms and their neuropsychological performance in the inactive phase of the disorder can be assessed. If cognitive deficits persist in symptom-free individuals, this would lend additional support to candidate cognitive endophenotypes. Another design to address the trait vulnerability issue is to assess individuals who are at risk of developing OCD (i.e., child or siblings of affected OCD patients). In light of the significant decision-making findings from this study and the few extant studies in this area, various aspects of decision-making (i.e., emotional, strategic, contingency-based) warrant investigation. Further, elucidating the mediating role of state anxiety in young individuals with OCD is a promising line of research.

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