University of New Mexico **UNM Digital Repository**

Psychology ETDs

Electronic Theses and Dissertations

Spring 4-17-2017

Defining High Functioning OCD (HFOCD)

Samuel Meyer University of New Mexico - Main Campus

Follow this and additional works at: https://digitalrepository.unm.edu/psy_etds



Part of the <u>Psychology Commons</u>

Recommended Citation

Meyer, Samuel. "Defining High Functioning OCD (HFOCD)." (2017). https://digitalrepository.unm.edu/psy_etds/207

This Thesis is brought to you for free and open access by the Electronic Theses and Dissertations at UNM Digital Repository. It has been accepted for inclusion in Psychology ETDs by an authorized administrator of UNM Digital Repository. For more information, please contact disc@unm.edu.

Samuel Meyer
Candidate
Psychology
This thesis is approved, and it is acceptable in quality and form for publication:
Approved by the Thesis Committee:
Kristina Rewin Ciesielski, Ph.D., Chairperson
Jane Ellen Smith, Ph.D.
Steve Gangestad, Ph.D.

DEFINING HIGH FUNCTIONING OCD (HFOCD)

 \mathbf{BY}

SAMUEL MEYER

B.B.A. INTERNATIONAL FINANCE AND MARKETING, 2005-2009 B.A. CRIMINOLOGY, 2005-2009 UNIVERSITY OF MIAMI (FL)

THESIS

Submitted in Partial Fulfillment of the Requirements for the Degree of

Master of Science Psychology

The University of New Mexico Albuquerque, New Mexico

May 2017

DEDICATION

To my father and mother, Tim and Debra Meyer, this project was made possible by your steadfast, loving, and selfless support in all aspects of my life. I am forever grateful to be your son.

ACKNOWLEDGEMENTS

I would like to thank Dr. Kristina Rewin Ciesielski for her unwavering support in helping me to initiate and complete this study. It would have not been possible without her guidance and vision. I am forever grateful for the time she spent helping see this project through to the end and the patience it took as well. I would also like to thank Dr. Jane Ellen Smith and Dr. Steve Gangestad, my committee members, for their patience, understanding, and support during this process. It is greatly appreciated. I would also like to thank Dr. Paul Lesnik for his consultation with the statistical analysis. His expertise is immensely appreciated. I also want to thank my research assistants, Miranda Self and Kianna Jupiter, and fellow graduate student Isabel Solis. Their help in compiling the data and willingness to sacrifice their own time contributed significantly in the completion of this project.

DEFINING HIGH FUNCTIONING OCD (HFOCD)

 $\mathbf{B}\mathbf{y}$

Samuel Meyer

B.B.A., International Finance and Marketing, University of Miami (FL), 2009
 B.A., Criminology, University of Miami (FL), 2009
 M.S., Psychology, University of New Mexico, 2017

ABSTRACT

Individuals with moderate-to-severe form of OCD symptoms may display exceptional creativity and productivity; the underlying neurobehavioral mechanism of this phenomena is unknown. This study aimed to define the characteristics of individuals who demonstrate high life achievement/creativity and display clinically significant OC symptomatology. The main hypothesis was that OC phenomena is significantly correlated with high levels of creativity, and that individuals displaying this characteristic will demonstrate a neurocognitive profile similar to subjects with clinical OCD.

We investigated cognitive, intellectual, emotional and clinical characteristics of real-world high-achievers (HAs, n=28) as compared to "standard achievers" (SAs, n=29) by measuring OC symptoms (Y-BOCS), depressive moods (BDI), levels of creativity/flexibility (Creativity Scale), Verbal and Visual-Spatial Reasoning proficiency and psychometric IQ. Participants attended two testing sessions for neuropsychological and clinical assessment. Using the clinical cut-off scores for Y-BOCS we identified two

groups with highest scores on OC symptoms (8-19points) in High-Achievers and Standard-Achievers, respectively. Between-groups independent samples t-test analysis and within-groups Kendall's Tau b correlations were performed.

We identified in this study a unique group of individuals, real life High-Achievers, displaying severity of OC symptoms that are clinically significant and demonstrating high scores on the scale of creativity. A valid strong correlative relationship between creativity and OC characteristics emerged readily when OC symptoms reached a higher level of severity and became clinically relevant. The population of the real life high functioning achievers with High OC scores and Hi-Creativity score (we name HFOCD) showed, however, no similarity to the clinical/cognitive profile of subjects with clinical OCD, with no symptoms of depression and no deficits in visual-spatial tasks, but with good verbal proficiency and high psychometric IQ. The results suggest that HFOCD may constitute an entirely unique OC Spectrum population of individuals with etiology and underlying specific brain mechanisms different than in subjects with clinical OCD.

TABLE OF CONTENTS

LIST OF FIGURES	viii
LIST OF TABLES	ix
CHAPTER 1 INTRODUCTION	1
CHAPTER 2 METHODS	12
CHAPTER 3 RESULTS	20
CHAPTER 4 DISCUSSION	28
DEFEDENCES	32

LIST OF FIGURES

Figure 1. Mean Differences Between HA and SA Groups for Creativity and	
YBOCS	21
Figure 2. Mean Differences Between HA and SA Groups for IQ	22
Figure 3. Mean Differences Between HA and SA Groups for VCI and PRI	22
Figure 4. Mean Differences for HA and SA Groups for BDI	23
Figure 5. Mean Differences Between HA and SA Groups for RCFT-IR	23
Figure 6. Mean Differences Between HH and SH Groups for YBOCS Scores	26
Figure 7. Mean Differences Between HH and SH Groups for IQ and VCI Score	es27

LIST OF TABLES

Table 1. Results of Independent Samples t-test Between HA and SA Groups	.45
Table 2. Kendall's Tau B Correlational Results for Entire Data Set	.47
Table 3. Results of Independent Samples t-test for HH and SH Groups	.49
Table 4. Kendall's Tau b Correlational Results for HH Group	.51
Table 5. Kendall's Tau b Correlational Results for SH Group	.53

Chapter1

Introduction

Obsessive-compulsive disorder (OCD) is a chronically debilitating neuropsychiatric syndrome with a population prevalence of 2%-3% (Karno, Golding, Sorenson, & Burnam, 1988). OCD is characterized by obsessions that are often bizarre thoughts, ideas, impulses or images. The obsessions are coupled with a feeling of urgency or catastrophe leading to repetitive, time consuming, and ritualistic behaviors known as compulsions (DSM-V, 2013). In severe cases, obsessions and compulsions can occupy the entire day causing significant distress and impairment in social, occupational, and personal areas of functioning (Rasmussen & Eisen, 1990, 1994; Sasson et al., 1997). The prolonged time spent with overt and covert rituals impedes on social interactions, relationships, learning and working productivity.

Yet, the evidence is available that individuals with a moderate-to-severe form of OCD may become exceptionally creative and productive. Among those reported are some of the most influential minds in our humanity, such as Isaac Newton, Nicola Tesla, Howard Hughes, Charles Darwin, Marie Curie-Sklodowska and many others (Andreasen, 1987; Barlett & Steele, 2004; Freeman, 2013; Mast, 2014). Thus, a question is raised, what are the psychological and brain characteristics that serve the outstanding creativity, despite manifestations of OC symptoms. The current study aims to define these characteristics in individuals who demonstrate high life achievement/creativity and display at least mild OC phenomena (8 points and higher) on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), a standard measure of OCD symptoms.

The Nosology of OCD Phenomena

For decades OCD has been considered to be a homogenous disorder. Only recent clinical and neuroimaging studies have lead to the concept of OCD as a multidimensional disorder (Leckman et al., 2010; Mataix-Cols, Rosario-Campos, & Leckman, 2005; Mataix-Cols, van den Heuvel, & Odile, 2006;). Among the empirically characterized nosological variables are: (i) Etiology; (ii) Time of onset (Mataix-Col et al., 2005); (iii) Behavioral and Neuropsychological Phenotypes (Gross-Iseroff et al., 1996; Mataix-Cols, Alonso, Pifarré, Menchón, & Vallejo, 2002; Savage et al., 1999); (iv) Pattern of underlying brain abnormalities (Friedlander & Desrocher, 2006; Maia, Cooney & Peterson, 2008); (v) Comorbidity with other DSM disorders (Lochner et al., 2014).

Etiology

Considering the mechanisms of etiology three subgroups have been studied. The largest consists of OCD individuals with a clear genetic background of the disease related to high aggregation of the mental disease in family members, and often a heritable pattern of clinical symptoms (Pauls, 2008). The second group includes post-traumatic OCD in which the disease develops after a traumatic event, such as brain injury, particularly involving the anterior cingulate gyrus and striatum (Berthier, Kulisevsky, Gironell, & Lopez, 2001; Grados, 2003; Grados, et al., 2008; Maia, et al., 2008) or psychological trauma (Borges et al., 2011; Boudreaux, Kilpatrick, Resnick, Best, & Saunders, 1998; Fontenelle et al., 2012). The third group includes rare patients who develop OCD symptoms despite a non-significant family background for mental disorders and negative history of trauma (Barlow, 2004). The etiological mechanism of such cases is not understood.

Time of Onset

Two peaks of onset have been described, pediatric (age 5-12) and young adulthood (after age of 20). The prevalence of pediatric OCD affects an estimated 1.5 to 2.2 million children in the United States (Flessner, Berman, Garcia, Freeman, & Leonard, 2009). It presents with a higher severity of symptoms, a preponderance in boys, and is more resistance to treatment than the adulthood form. Pediatric OCD also differs from adult OCD in that it is often co-morbid with disruptive behaviors and developmental disorders (Geller & March, 2012).

Other characteristics of pediatric OCD include a higher propensity of aggression/catastrophe obsessions, hoarding and saving compulsions, poor insight into their disorder compared to adults, and a higher fear of loosing loved ones or hurting themselves. They display typical washing and checking comparable to adult populations. Pediatric OCD patients are also more likely to experience severe cases of separation anxiety disorder. Among co-morbid disorders that were found more frequently in the pediatric OCD population and inversely related to age are Tourette's Syndrome and ADHD (Faro, Geller, Joshi, & Mancuso, 2010; Geller et al., 2001).

Adult onset OCD has different co-morbid disorders than pediatric OCD. While Pediatric OCD patients have shown higher Tourette's, Separation Anxiety Disorder, and ADHD rates than adult OCD patients, Mancebo et al. (2008) showed that adults had higher rates of substance abuse and eating disorders than their pediatric counter parts.

Adult OCD patients also showed lower rates of hoarding compared to pediatric patients (Mataix-Cols, Nakatani, Micali, Heyman, 2008). Adult onset OCD also has been shown to have less of a biological or genetic component as compared to pediatric OCD (Walitza

et al, 2010). Wang et al. (2012) found differences in obsessions and compulsions between pediatric and adult OCD patients. Adult patients were found to have less symmetry/exactness obsessions. Other studies have also shown that adult onset OCD endorsed less aggressive/catastrophic obsessions, checking compulsions, and that superstition and magical thoughts, parasite obsessions and repeating, counting, tapping/rubbing and collecting compulsions were significantly less frequent in adult OCD (Garcia et al., 2009; Millet et al., 2004).

Cognitive & Behavioral Phenotype

Subjects with OCD have been separated into clusters considering the diversity of clinical and cognitive neuropsychological phenotypes. Those with a more severe display of obsessions than compulsions are reported to be more resistant to treatment and more susceptible to suicidal attempts than persons with OCD who predominantly display compulsions (Barlow, 2004). Moreover, a significant portion of the population with OCD present with significant deficits in the visual-spatial domain but with very well preserved verbal functions, in contrast to those who do not demonstrate significant cognitive deficits (Dirson, Bouvard, Cottraux, & Martin, 1995; Krishna et al., 2011; Kuelz, Hohagen, & Voderholzer, 2004). Abnormalities in executive organization have been also shown consistently in the neuropsychological profiles of OCD patients. The abnormality in the organizational process leads to apprehension, doubt, anxiety and ritualized behaviors are utilized to offset these deficits. Poor inhibition of interference has been considered to underlie deficits mostly in visual–spatial recall, cognitive set shifting, and organization during visual-spatial working memory tasks (Christensen et al., 1992; Gross-Iseroff et al., 1996; Malloy, 1987; Rosenberg et al., 1997; Savage et al., 1999).

Long reaction times (RTs) (Galderisi et al., 1995) and shorter latency of cortical responses in more severe OCD cases (Ciesielski et al., 1981; Purcell et al., 1998; Towey et al., 1990) have also been related to inhibitory deficits. On tasks of executive functions OCD patients often exhibit increased response latencies, set over-shifting, and problems incorporating feedback to adjust their responses (Galderisi et al., 1995; Olley, Malhi, & Sachdev, 2007). These deficits in executive organization are thought to be the major underlying cause of the abnormalities seen in visual-spatial memory in OCD patients (Penadés, Catalán, Andrés, Salamero, & Gastó, 2005; Savage et al., 1999; Shin et al., 2004).

Brain Abnormalities

Inherent persistence of obsessive thoughts and compulsive behaviors have been taken as a primary indicator of inhibitory failures and originally linked to frontal cortical over-activation (Flor-Henry, 1983; Flor-Henry et al., 1979). The common conclusion from earlier studies support a model of frontal-striatal deficits and its relatedness to failures in inhibitory control of motor and cognitive functions (Rauch et al., 2001; Rosenberg and Keshavan, 1998). Currently, the evidence about abnormalities in the striatum, the anterior cingulate cortex (ACC), the dorsolateral prefrontal cortex (DLPC), the head of the caudate nucleus, and the thalamus comprise the circuitry underlying the frontal-striatal hypothesis of OCD (Aouizerate et al., 2004; Gu et al., 2008; Pauls, Abramovitch, Rauch, & Geller, 2014). Recent fMRI studies also showed a reduction in the orbital-frontal cortex (OFC) in OCD patients during repeated tasks, and this abnormality may be due to an abnormal volume of gray matter in the OFC in OCD

patients (Aouizerate et al., 2004; Gu et al., 2008; Maia et al., 2008; Remijnse et al., 2006).

Findings of more recent neuroimaging studies elucidated patterns of brain abnormalities involving the Frontal-Parietal network. For example, fMRI studies demonstrated that in the frontal-parietal networks of OCD patients, both the cortical mantle and white matter show significantly abnormal density in volume and oscillatory activation (Ciesielski et al., 2007), suggesting a white matter endophenotype for OCD (Menzies et al., 2008; Shin et al., 2006). In one study gray-matter in the left cuneus and the cerebellum were reduced in OCD patients contrary to the abnormalities they expected in the frontal-subcortical circuits (Kim et al., 2001). The authors suggested that these abnormalities contribute to the abnormal visual-spatial processing and visual memory in subjects with OCD. Consistently, recent MEG studies in non-depressed subjects with OCD displayed abnormally reduced activation in the parietal regions and abnormally elevated activation in the prefrontal medial and dorsal lateral cortex during visual-spatial working memory tasks (Ciesielski, Hämäläinen, Lesnik, Geller, & Ahlfors, 2005; Ciesielski et al., 2012). Since functional coupling between the parietal-occipital and frontal alpha provides insight into the neural mechanisms that are failing during abnormal top-down inhibitory control (Neupper & Pfurtscheller, 2001; Ward, 2003), our focus on integrity of the frontal-parietal network, as the main substrate of top-down-inhibitory control mechanism, is well justified.

Comorbidity

Using the criterion of co-morbidity many of the OCD cases demonstrate cooccurrence with other DSM-V disorders, such as major depressive disorder (MDD) in ~20% of cases. Comorbid OCD with MDD responds to a different treatment than regular OCD as only a small number of selective serotonin reuptake inhibitors (SSRI's) alleviate symptoms. This has led to speculation that OCD comorbid with MDD may be qualitatively different than OCD by itself and the comorbid MDD may be an integral part of OCD and not a separate entity (Fineberg, Fourie, Gale, & Sivakumaran, 2005).

ADHD is also a highly comorbid disorder with OCD (~30% of cases). This is unusual as OCD and ADHD are distinctly separate conditions and the explanations behind the high comorbidity rates are undetermined (Walitza et al., 2008). Comorbid cases present with an earlier onset, more severe symptoms, and higher persistence (Borges, et al., 2011; Flessner et al., 2009). A pure OCD with no confound of depressive symptoms constitute only ~25% of OCD cases. The comorbidity rate and severity of a recurring disorder may fluctuate during the lifetime of a patient (Masclitis, Rector, & Richter, 2003).

The Severity of Symptoms: Subclinical OCD

HFOCD needs to be differentiated from a construct of subclinical OCD, in which OC level symptoms may intermittently interfere or not with daily functioning. On the Y-BOCS, a tool for assessing the severity of OC symptoms, the clinical cutoff score is 16 and of a possible 40. Scores below 7 are conventionally considered as representing a subclinical form of OCD (Goodman et al., 1989). Subclinical OCD is defined as the presence of obsessive thoughts or compulsive rituals that do not cause impairment or distress, that are not time consuming (i.e. <1 hour per day), and thus, do not fulfill the clinical criteria for DSM-V (Lesnik, Ciesielski, Hart, Benzel, & Sanders, 1998). However, Rachman and de Silva (1978), who were the first to report thought intrusions in about 70% of an average normal population, stated that occurrence of intrusions was

related to the severity of depressive and anxiety symptoms of an individual, and the attempts to resist the thoughts. The content of thought intrusions in the general population resembled obsessions in subjects with OCD and included: impulses of harm or attack towards someone, thoughts about sexual assault, family health problems etc. Subclinical OCD is an important but underappreciated member of the OCD spectrum. It is commonly found among first-degree relatives of persons with OCD, either through a genetic link, and/or behavioral modeling by the parent. Recent clinical and neuroimaging studies revealed the presence of endophenotype in OCD, the internal intermediate phenotype that constitutes continuity between genes and eventual disease. Abnormalities in the parietal white matter cortex, cortical orbital regions and frontal subcortical regions have been identified as endophenotypes of OCD (for example Menzies et al., 2007; Szeszko et al., 2004). Subclinical OCD may proceed to the clinical form in ~ 20% of cases (Black & Gaffney, 2008).

The Level of Functioning: High Functioning OCD (HFOCD)

The present study predicts the presence of a new cluster of individuals with OCD, from the perspective of the level of their functioning. While the lower end of this spectrum will aggregate individuals who are devastated by the disease and fail in all aspects of life (low-functioning patients with OCD), the far end of the spectrum will concentrate on those individuals with OCD who, despite the highly distressing and clinical (as per DSM-V criteria) symptoms of OCD, could be highly productive and successful. We will call this population *High-Functioning OCD* (HFOCD). Cases with HFOCD are rare and, therefore, gaining a testing access to this HFOCD population in a sufficiently large sample is unattainable. However, we may examine this relationship

between high achievement/creativity and OC symptoms, by accepting a model of a non-clinical population of "real-world" high achievers" (HAs) and assessing them using measures of creativity and the manifestation of OC symptoms. If this link between the HAs, creativity and OC symptoms exists, we will be able to characterize the pattern of neuropsychological and clinical measures and, therefore, the kind of brain mechanism that characterizes these individuals that are able to function well while experiencing OC symptoms, or who may even employ OC symptoms as an ally towards success.

Currently, there is a paucity of research in this area. Furnham, Hughes, and Marshal (2013) attempted to show a negative correlation between OCD and creativity, as deriving from over-inclusive thinking. Thus, the rigid thinking styles that are commonly associated with OCD (Moritz et al., 2002), such as orderliness, neatness, and perfectionism, would come at the cost of flexibility and creativity (Furnham & Crump, 2013). In contrast, it was found that sub-clinical OCD was positively correlated with creative behaviors even when not reported on self-evaluations of creativity (Furnham et al. 2013).

What are the cognitive and neurobiological mechanisms underlying the successful battle with OCD? Understanding these mechanisms is important as this knowledge will help us to unravel the processes in which some individuals are able to control their OC symptoms and concentrate that energy on a particular target of achievement, while others succumb to the disease. Unraveling the basis of such resilience to OCD may instigate future studies on prevention. It may also reflect on the evolutionary persistence of this highly debilitating disorder that is markedly resistant to treatment. Based on empirical observations we predict that the link between OC symptoms and high real-world

creativity may have an evolutionary and, therefore, ubiquitous character. The understanding of a neurobehavioral basis of HFOCD may help us to unravel the unexpected developmental alliance between OCD and exceptional achievement and creativity.

Hypotheses

The overarching hypothesis of this research is that the presence of HFOCD can be documented using an approximate model in real-world "high achievers" (HAs) as compared to "standard achievers" (SAs). The co-display of obsessive-compulsive (OC) symptoms with creativity and exceptional life achievement raises some intriguing questions: Are high creativity and high life achievements innately linked to OC characteristics in the population at large, and, if this is the case, what could be the neural/behavioral mechanisms underlying such a link? Understanding this mechanism is critically important for gaining insight into the mechanism of resilience against obsessions and compulsions and, therefore, for designing future prevention approaches for OCD. Our specific hypotheses are:

Hypothesis 1: The <u>HAs</u> will demonstrate a significantly higher scores on creativity measures, and higher prevalence to OC characteristics than <u>SAs</u>, with high correlations between these two measures.

Hypothesis 2: The neurobehavioral profile demonstrated in HAs, as compared to SAs, will resemble the profile reported in clinical OCD, with depressive moods, abnormal inhibitory control of distracters, a deficient performance on visual-spatial organization and memory tasks but high verbal proficiency.

Hypothesis 3: The neural mechanism underlying the high scores on creativity tasks in HAs in parallel with prominent OC characteristics, will be associated with a high psychometric IQ and normal scores on Brief Ataxia Rating Scale (BARS), reflecting a high level of integrity within the frontal-parietal network.

Chapter 2

Methods

Rationale

We aimed to characterize 'real-world' <u>high achievers (HAs)</u> in comparison to "real-world" <u>standard achievers (SAs)</u> using four groups of characteristics: **Group 1** for severity of Obsessive-Compulsive symptoms (OC) and Depressive Moods; **Group 2** for Measures of Creativity/Flexibility; **Group 3** for the psychometric IQ; and **Group 4** the Verbal vs Visual-Spatial performance. IQ helped us to determine the compatibility of subjects for both groups, HAs and SAs, and with the final interpretation of acquired data. Our working hypothesis was that HAs are similar to patients with OCD and will, in contrast to SAs, display a significantly higher level of O-C characteristics and higher scores on the creativity measure than SA's. We also predicted a significantly lower performance on visual-spatial tasks than SAs. Such defined HAs provided a valid model to initiate studies on the entanglement of OC characteristics and creativity with a well-justified extension of the examination of brain networks in HFOCD.

Participants

Fifty-Seven participants were recruited to participate in this study. The exclusionary criteria was identical for all subjects and included: a history or current clinical diagnosis of a DSM-V disorder, CNS medication, TBI, current or past severe substance/alcohol abuse and medical conditions, including seizures. Two participants were excluded. One due to depression and one due to current substance abuse. Participants in both groups who expressed interest in participating were e-mailed a preliminary screening questionnaire to asses for the exclusionary criteria. The

questionnaire also assessed whether or not they were currently a graduate student or planned on applying for/attending graduate school. All participants were compensated monetarily in a uniform amount, except for some participants in the SA group that were given class credit.

From these 57 participants, 28 'real-world' **HAs**, (17 female and 11 male, age range = 20-39, *M* age=27.8, *SD*=5.17), were recruited from the population of graduate and doctoral students, post-doctoral researchers, and medical students from multiple graduate departments of The University of New Mexico College of Arts & Sciences, The University of New Mexico School of Medicine, The University of New Mexico School of Engineering, The University of New Mexico School of Architecture and Planning, and The Mind Research Network. These participants were recruited by putting up flyers in each respective school/department and sending email messages to all the graduate and doctoral students, as well as post-doctoral researchers, in each. Once they responded expressing interest in participating they subsequently completed the Screening Questionnaire Form, and if eligible, were invited to participate in person.

The remaining 29 participants made up the <u>SA</u> group (19 female and 10 male, age range=18-36, *M* age=22.9, *SD*=5.08). These participants were recruited from undergraduate psychology courses at the University of New Mexico and from associate degree and apprenticeship programs from Central New Mexico Community College. The SA group was recruited by flyers displayed around the University of New Mexico campus, the SONA recruiting system online, and mass emails to undergraduate psychology classes. Like the HA group, once the SA participants responded expressing interest in participating they subsequently completed the Screening Questionnaire Form,

and if eligible, were invited to participate in person. These participants indicated on their screening form that they were not planning on attending graduate school.

Measurements

Four groups of measurements comprised the following testing tools:

Group 1. Severity of Obsessive-Compulsive Symptoms and Depressive Moods

Yale-Brown Obsessive Compulsive Scale (Y-BOCS) and Structured Clinical Interview

For assessment of severity of Obsessive-Compulsive symptoms (OC); Severity of OC symptoms was assessed using Y-BOCS (Goodman et al., 1989): Quantitative severity for obsessions includes items 1 – 5 and compulsions 6 – 10 by time consumed, life interference, internal distress, control over symptoms, also insight, avoidance, indecisiveness, pervasive slowness. The scale has earned high validity and reliability. The sub-clinical cutoff score is 8 and below, out of a possible 40. Scores of people in the community-at-large may fall between 0 and 8.

BDI-II – The Beck Depression Inventory II is a 21 question multiple choice self-report questionnaire designed to assess the severity of depression. Each question has an option of selecting 0-3 and the scores from the questions are totaled for the first set of ten questions and the second set of ten questions. Ratings of two or three on questions, two (hopelessness) and nine (suicidal ideation) should be closely attended to as these are red flags. The cut off scores for the total scores are as follows: 0-13 minimal depression, 14-19 mild depression 20-28 moderate depression, 29-63 severe depression (Beck, Steer, Ball, & Ranieri, 1996).

Group 2. Achievement/Creativity and Cognitive Flexibility

The Creativity Achievement Questionnaire (CAQ) (Carson, Peterson, & Higgins, 2003) is a self-report measure used to assess creativity of an individual. The measure asks about 10 different domains of creativity (Visual Arts, Music, Dance, Architectural Design, Creative Writing, Humor, Inventions, Scientific Discovery, Theater and Film, Culinary Arts) and requires the participant to put a check mark by the sentence that best describes their relation to that domain such as music or visual arts. Within each domain there are seven sentences describing their level of expertise with zero being no training or experience and seven usually being national recognition for that domain. If the participant selects seven they are also asked to indicate the number of times that sentence applies to them. For example, the music in domain seven states, "My compositions have been critiqued in a national publication" and if selected the participant would write how many times that has happened. At the end, the numbers next to the sentences selected are added up and a total score is formed. If the number seven sentence is selected it is multiplied by the number of times the participant indicated it has happened and then added to the total score.

Wisconsin Card Sorting Test (WCST) – This test was used to assess executive function, abstract reasoning, and set shifting flexibility. The 64-card deck of the WCST will be administered, which required participants to match each of the cards to 1 of 4 template cards on the basis of three principles (color, form or number). Very little feedback was given to the participant regarding their responses, therefore, requiring the use of abstract reasoning skills in order to carry out the task (The same format as Heaton, Chelune, Talley, Kay & Curtiss, 1993). Participants were required to correctly sort to a

given principle (e.g., color) ten consecutive times, before the sorting principle is changed (e.g., to form). The task was continued until the participant has sorted all 64 cards in the deck. The following WCST outcome variables were used in the analyses: (1) categories completed (every 10 correct consecutive sort to a correct principle is counted as one completed category) (2) total errors (every time a card is incorrectly sorted) (3) perseverative responses (repetitive sorting) (Kohli & Kaur, 2006).

Group 3. Psychometric IQ

WASI-II – The Wechsler Abbreviated Scale of Intelligence is an abbreviated measure of cognitive intelligence that uses subtests from the WAIS-IV and is meant for people ages 6-90. These subtests assess for Verbal and Perceptual reasoning. The Vocabulary and Similarities subtests make up the Verbal Comprehension Index (VCI) and Block Design and Matrix Reasoning form the Perceptual Comprehension Index. The scores from all four subtests are combined to form the Full-Scale IQ-4 (FSIQ-4). The classifications for FSIQ-4 scores are as follows (Wechsler, 2011): 70 extremely low; 70-79 borderline; 80-89 low average; 90-109 average; 110-119 high average; 120-129 superior; 130 very superior. This test was used to estimate Verbal and Visual IQ.

Block Design – This subtest assesses the ability to analyze and synthesize visual stimuli, visual perception and organization, nonverbal concept formation, and visual motor coordination. The examinee used red and white blocks to recreate two dimensional designs from the stimulus booklet. There were 13 total trials and each trial had a time limit in which the examinee must complete the design to receive points. Depending on how the fast the examinee correctly puts together the blocks, more points were awarded for questions 5-13 (McCrimmon & Smith, 2013).

Vocabulary – This subtest assesses word knowledge, verbal concept formation, crystallized intelligence, and degree of language development. The examinee was required to verbally define or describe a word or concept that is presented orally to them. They were also able to see the word written out in the stimulus book. Scoring is dependent on the examinees responses and can be either zero points, one point, or two points with some responses necessitating follow up responses (McCrimmon & Smith, 2013).

Matrix Reasoning – This subtest assesses fluid and visual intelligence, spatial ability, and perceptual organization. Within the stimulus booklet are 30 visually-depicted matrices that are not completed. The examinee was then required to view the matrix and pick one of the five images in a row below that matrix that would logically complete it. Each correct trial was worth one point (McCrimmon & Smith, 2013).

Similarities – This subtest assesses verbal concept formation, crystallized intelligence, abstract reasoning, associative and categorical thinking, and verbal expression. The test includes 24 items and examinees were required to respond verbally describing two concepts or objects presented orally by the examiner. Scoring for each item ranged from 0-2 depending on the examinees response and how well these responses adhered to the guidelines presented in the manual (McCrimmon & Smith, 2013).

Group 4. Neurological Screening Test

Brief Ataxia Rating Scale (BARS); Schmahmann, Gardner, MacMore & Vangel, 2009). BARS is a well established tool with separate assessment of posture and gait, kinetic functions, speech disorders and oculomotor disorders. The quantification of

the specific core of the cortical-cerebellar syndrome is possible and useful for both developmental and pathological application.

Rey-Ostereith Complex Figure Test – (Lezak, 2003). This neuropsychological tool is used to assess visual-spatial abilities, nonverbal memory skills, organizational skills, visual-motor coordination and top-down inhibitory control, all functions closely relying on the frontal-parietal- network. The participants viewed a picture of the complex figure and drew a copy of it. Next, in the Immediate Recall (RCFT-IR), the picture of the figure was taken away and the subject was asked to draw the image from memory. Finally, in the delayed condition, the participant was asked to draw the complex figure as best they could one more time (Shin, Park, Park, Seol, and Kwon, 2006).

Procedure

The testing procedure was split into two sessions. The first session took approximately 1.5 hours and the second session took approximately 1 hour and 20 minutes.

First Session

In the first session, the subject was invited by the Student Investigator to the Pediatric Neuroscience Laboratory (PNL) to participate. After the subject was read their rights as a participant and gave their informed consent testing began. This test was followed by a vision test on a standard eye chart. The participant was directed to stand approximately 10 feet from the chart and read the line as indicated by the researcher. This was repeated for both eyes. The participant then underwent an auditory test. The researcher would rub their fingers together either to the left, right, or directly behind the participant. The participant would then indicate if they heard the sound by raising their

respective hand (both hands for directly behind). These tests were administered to make sure the participant's sensory functions were intact.

After these tests were complete, the participant was led to another testing room consisting of a table (with testing material already set up) and two chairs. The first test administered was Annette's Test of Handedness to determine if the participant was left or right handed. The test battery was administered in the following order: WASI-II (entire test), Rey-Ostereith Complex Figure Test (copy), Rey-Ostereith Complex Figure Test (immediate recall), WCST, CAQ, and the Rey-Ostereith Complex Figure Test (delayed recall). After all tests were completed the participant was led back to the PNL and received their payment or class credit. All of the above assessments were administered by the Student Investigator who was trained in the administration of each measure.

Second Session

After the first session, the participant was invited back to the PNL by the Principle Investigator. This session consisted of the following assessments in order: Structured Clinical Interview, BARS, BDI-II, and Y-BOCS. All of these assessments were administered by the licensed Clinical Neuropsychologist. After the participant completed all the assessments, they were given the second half of their reimbursement.

Chapter 3

Results

Between Group Analysis for Entire Data Set

A series of between-group t-tests revealed that our groups, HAs and SAs, did not differ significantly in terms of age and gender (p>.05).

A summary of the independent samples t-test results, related to the complete set of data for all HAs and SAs participating in this study, is shown in **TABLE 1**. Four out of ten variables were assumed to have unequal variances based off results from Levene's Test for Equality of Means and the unequal p-values are reported for those four variables. There was a statistically significant difference between HA Creativity scores (M = 16.75, SD = 8.64) and SA Creativity scores (M = 10.86, SD = 9.11), t(55) = 2.503, p = .015 with HAs scoring higher. However, there was no statistically significant difference between the HA Y-BOCS scores (M = 7.23, SD = 5.93) and the SA Y-BOCS scores (M = 5.44, SD = 4.35), t(45.85) = 1.23, p = .224. **FIGURE** 1 below shows that HAs obtained a significantly higher score on creativity than SAs, but did not differ from SAs in their OC scores on Y-BOCS scale (see **TABLE** 1).

FIGURE 1. Mean Differences Between HA and SA Groups for Creativity and YBOCS

Scores Error bars represent 95% confidence interval. *p<.05

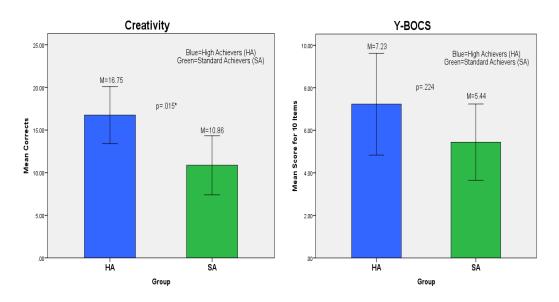


TABLE 1 shows also statistically significant difference between the following variables: HA's IQ scores (M = 109.79, SD = 9.06) and the SA's IQ scores (M = 96.34, SD = 10.88), t(55) = 5.06, p < .001, HA VCI scores (M = 106.18, SD = 8.89) and SA VCI scores (M = 93.84, SD = 10.08), t(55) = 4.90, p < .001, HA PRI scores (M = 111.32, SD = 13.08) and SA PRI scores (M = 101.45, SD = 11.29), t(55) = 3.06, p = .003, and HA BDI scores (M = 5.62, SD = 5.12) and SA BDI scores (M = 9.36, SD = 5.83), t(49) = -2.44, p = .018, and HA's RCFT Immediate scores (M = 25.46, SD = 5.94) and SA's RCFT-IR (M = 19.70, SD = 8.24), t(50.95) = 3.04, p = .004. For all of these comparisons, HA scores were significantly higher, except for BDI scores on which SA scores were higher. FIGURE 2 shows mean IQ differences between HA and SA groups. FIGURE 4 shows mean BDI differences between HA and SA groups. FIGURE 5 shows mean RCFT Immediate differences between HA and SA groups.

FIGURE 2. Mean Differences Between HA and SA Groups for IQ

Error bar represents 95% confidence interval. ***p<.001

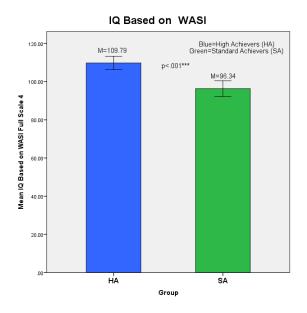


FIGURE 3. Mean Differences Between HA and SA Groups for VCI and PRI Error bar represents 95% confidence interval. **p<.01 ***p<.0001

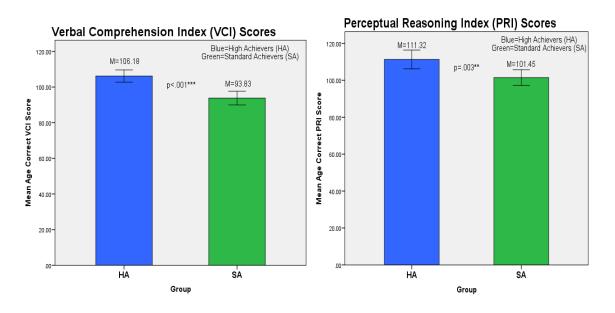


FIGURE 4. Mean Differences Between HA and SA groups for BDI Error bar represents 95% confidence interval. *p<.05

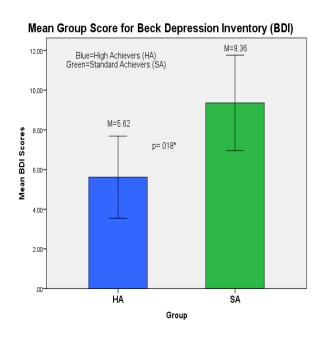
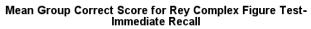
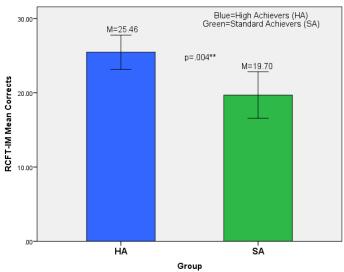


FIGURE 5. Mean Differences Between HA and SA Groups for RCFT-IR

Error bars represent 95% confidence interval. ** p<.01





The results suggest that HAs had higher IQ scores, significantly higher verbal and perceptual reasoning scores, and significantly lower scores (less depressive answers) on BDI. These results are consistent with expected higher psychometric IQ in HAs. These results are not consistent with our hypothesis that HAs will have elevated scores on BDI. The results are also not consistent with our prediction that HAs will perform significantly more poorly than SAs on tests of visual-spatial ability.

Within-Group Analysis: Correlational Analysis for Entire Data Set

Next, a nonparametric correlation was conducted using Kendall's Tau-b to examine the relationship of IQ, Creativity, Y-BOCS, BDI, BARS, VCI, PRI, RCFT-ID, WCST Perseverative Errors, and WCST Categories Completed within the entire group of 57 participants. The Kendall's Tau-b correlation was used instead of the Pearson Correlation to adjust for the non-normal distributions due to four of our variables having unequal variances. Also, given the number of correlations being computed, Kendall's Tau-b gave us an exact *p*-value so no Bonferroni correction was necessary. The results are summarized in **TABLE** 2.

The results showed a statistically significantly correlation of IQ with Creativity ($\tau b = .274$, p = .004), BDI ($\tau b = .224$, p = .025), RCFT Immediate ($\tau b = .432$ p < .001), and WCST Perseverative Errors ($\tau b = .221$, p = .025). These results suggest that as IQ increases, so does creativity, and visual-spatial ability and memory as well as cognitive flexibility. It also suggests that as IQ rises depression decreases. The results also showed a statistically significant correlation between Creativity and Y-BOCS ($\tau b = .260$, p = .01), VCI ($\tau b = .373$, p < .001), PRI ($\tau b = .19$, p = .042), and RCFT Immediate ($\tau b = .233$, p =

.012). This suggests that as OC symptoms rise, so does creativity. Consistent also is an increase in verbal and visual-spatial reasoning and visual-spatial memory.

These findings support our hypothesis that the neural mechanism underlying the high scores on creativity tasks in HAs in parallel with prominent OC characteristics, will be associated with high psychometric IQ and subclinical scores for depression. It also supports our hypothesis that there is a correlation between higher creativity and high OC characteristics. Further, these findings support our hypothesis that verbal scores will be correlated with higher OC characteristics but does not support our hypothesis that the reduced visual-spatial ability will be negatively correlated with higher OC characteristics.

A partial correlation was also conducted to make sure IQ was not influencing the relationship between Creativity and Y-BOCS. Controlling for IQ, there was still a significant correlation between Creativity and Y-BOCS (r = .334, p = .018). These findings propose that despite differences in IQ, creativity and OC characteristics are still related and IQ is not driving this relationship.

Between Groups Analysis of High High Achievers (HH) and Standard High Achievers (SH)

In an effort to accurately capture OC characteristics from the HA group, we used the 8 points cutoff for subclinical OCD scores on the Y-BOCS. Participants who scored 8 or above on the Y-BOCS were put into a new group called High-High Achievers or HH. These 12 participants constituted the top 46% of the HA group in terms of Y-BOCS scores. To accurately compare the HH group with the SA group, we took the same percentage, 46%, of top Y-BOCS scores from the SA group to create a group called Standard High Achievers or SH. This not only gave us a more valid look at participants

with OC characteristics, it also reduced the variability of Y-BOCS scores in the populations as seen by the decrease in each SD. The HH Y-BOCS score is M = 12.92 SD = 2.75; The SH Y-BOCS score became M = 9.25 and SD = 2.56.

To compare the two groups and determine whether the HH group represented a profile of HFOCD (higher Y-BOCS and Creativity scores) compared to the SH group, we used a two sample independent t-test. The HH group was statistically significantly higher in the following areas: Y-BOCS t(22) = 3.82, p = .003 HH (M = 12.92, SD = 2.75) SH (M = 9.25, SD = 2.56), Creativity t(22) = 1.30 p = .003 HH (M = 17.75, SD = 8.90) SH (M = 12.92, SD = 9.37), IQ t(17.64) = 3.05 p = .007 HH (M = 108.83, SD = 7.36) SH (M = 95.92, SD = 12.70), and VCI t(22) = 1.04 p = 0.01 HH (M = 107.50, SD = 8.59) SH (M = 95.95, SD = 11.46). Figure 6 represents the YBOCS mean group differences. Figure 7 below shows the mean HH vs. SH group differences for IQ and VCI.

Figure 6. Mean Differences Between HH and SH Groups for YBOCS Scores Error bar represents 95% confidence interval. **p<.01

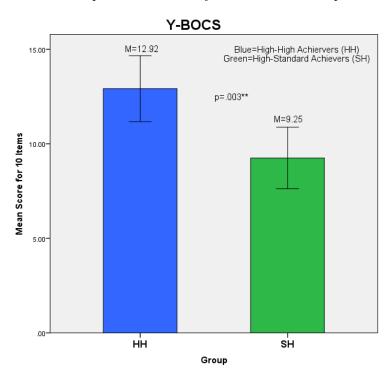
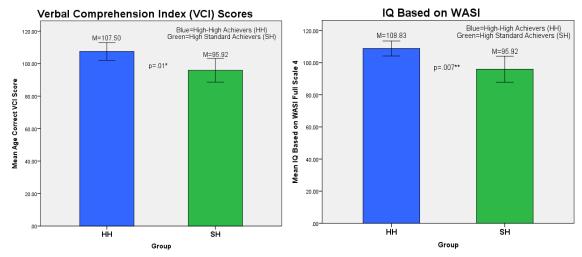


Figure 7. Mean Differences Between HH and SH Groups for IQ and VCI Scores Error bar represents 95% confidence interval. * p<.05 **p<.01



Within-Group Analysis: Correlational Analysis for HH and SH Groups

To further test our hypothesis that HH will show a high correlation between creativity and OC characteristics a Kendall's Tau B correlation was performed (**TABLE 4**). Within the HH group Creativity and Y-BOCS were significantly positively correlated ($\tau b = .552$, p = .015). Their Y-BOCS and BDI scores were also correlated ($\tau b = .569$, p = .014). The only significant correlation for the SH group was a negative correlation between IQ and BDI ($\tau b = .457$, p = .014), Y-BOCS and BARS ($\tau b = .-.610$, p = .01), and RCFT-IR and IQ ($\tau b = .554$, p = .013) (**TABLE 5**).

Chapter 4

DISCUSSION

In this study we identified a unique group of individuals, real life High-Achievers displaying a severity of OC symptoms that are clinically significant (between 8-19 points on Y-BOCS) and demonstrating high scores on the scale of creativity. It is important to note that the Y-BOCS scores we report in our population of college students ranging between 5 and 9 points (out of 40) on the Y-BOCS scale, are highly consistent with earlier reported average scores in college students across campuses (Steketee, Frost & Bogart, 1996), and therefore appear to be a valid representation of the average distribution of OC traits across student populations at large. However, a valid strong correlative relationship between creativity and OC characteristics seems to emerge only when OC symptoms reach a higher level of severity, and become clinically relevant.

It is important to note, that our HH achievers (that we named HFOCD), displaying a moderate-to-severe clinical score on Y-BOCS, did not display a pattern of clinical and cognitive characteristics resembling patients with OCD. They scored low on the Beck Depression Inventory and did not have significant difficulties with performance on visual-spatial tasks, although poor visual-spatial proficiency is a cognitive marker of OCD (Bloch et al., 2011). Additionally, most of HFOCD subjects achieved a significantly higher level of IQ than the rest of the tested population. Thus, the population of the real life high functioning achievers with High OC scores and Hi-Creativity scores (HFOCD) also showed a high IQ, and good Verbal score. In contrast to patients with OCD they also show high visual-spatial skills, and low scores on the Beck Depression Inventory. Thus, the results permit speculation that HFOCD may constitute an entirely

unique population of individuals with different etiology and underlying brain mechanisms.

So how are HHs able to achieve with significantly higher OC characteristics than the normal population? The answer may lie in their cognitive flexibility. Researchers have long thought that the cognitive processes in individuals who are achieving at the top of their field are operating on a different level of thinking quantitatively and qualitatively (Simonton, 1988, 1999). These differences were proposed to be explained by a lower level of latent inhibition (LI- inability to cognitively suppress past stimuli and events). It has been suggested that that low latent inhibition is associated with increased creative achievement in high-functioning individuals (Carson et al., 2003; Takeu et al., 2011). Specifically, their studies show a significant relationship between reduced LI and high creativity compared to controls with high LI and an average conceptual creativity. This greater exposure to stimuli variability would lead to more cognitive flexibility and the ability to combine stimuli in more original and more creative ways, than by someone limited by high LI. The research also showed that high IQ is a modulating factor for high creativity in individuals with low LI. Those with high IQ have demonstrated the ability to avoid the psychopathology usually associated with low LI. This high IQ (quantitative) combined with other yet unknown cognitive strengths may hold the key in deciding whether low LI will lead to high creative achievement or low functioning psychopathology (Carson et al., 2003; Chirila & Feldman, 2012).

This pattern of low LI driving creativity and being modulated by high IQ may explain the findings of our study. The IQ differences between the HH and SH groups was significant and in the HH group there was a strong significant correlation between

creativity and YBOCS scores that was not seen in the SH group. Across the HA and SA groups HA's were significantly more creative and again had higher IQs. While IQ may not be driving the relationship between creativity and YBOCS scores in our sample per our partial correlation a trend has emerged with the HA and HH groups having higher IQs. Applying the low LI theory to these results we would suggest that low LI has led to higher creativity in HAs than SAs. Those with the lowest LI would presumably have the most severe OC characteristics as seen in the clinical YBOCS scores of the HH group. This group had the highest IQ's and YBOCS scores while also showing the strongest correlation between creativity and OC characteristics. Thus, their high IQ acts as a protective factor or cognitive strength in the way Carson et al. (2003) suggested allowing their low LI to manifest into high creativity and high achievement while protecting them from the more severe psychopathology of OCD.

Behaviorally the characteristics of OCD such as obsessions and compulsivity, when modulated with high IQ or other cognitive resilience, may become adaptive for an individual and allow them to achieve higher creativity than their standard counterparts.

OCDs tend to have more personality types and behaviors that are associated with orderliness and perfectionism at the expense of efficiency and flexibility. While this preoccupation with neatness, orderliness, and perfectionism is not often thought of in the same vein as traditional creativity, OCD has been shown to be associated with creative behaviors in sciences and technology OCDs, where they may achieve the most (Furnham et al., 2013).

Current neuroimaging studies associate a specific integrative functional connectivity between the Default Mode Network (DMN), the Fronto-Parietal Networks

(FPN) and Inferior Medial Frontal regions, with high creativity in healthy individuals (Beaty et al., 2014; Zhu et al., 2017). These networks also play an important role in the neurobiological models of OCD (Ciesielski et al., 2012; Menzies et al., 2008). The currently identified unique population of high functioning real-life high achievers with high OC characteristics (HFOCD), with high markers of originality and no depressive symptoms invites a continuation of these studies using neuroimaging technology. Understanding the underlying network connectivity in HFOCD subjects may inspire innovative ideas about the preventive interventions for OCD.

References

- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Andreasen, N. C. (1987). Creativity and mental illness: prevalence rates in writers and their first-degree relatives. *The American Journal of Psychiatry*, *144*(10), 1288–1292.
- Aouizerate, B., Guehl, D., Cuny, E., Rougier, A., Bioulac, B., Tignol, J., & Burbaud, P. (2004). Pathophysiology of obsessive–compulsive disorder: A necessary link between phenomenology, neuropsychology, imagery and physiology. *Progress in Neurobiology*, 72(3), 195–221.
- Barlett, D. L., & Steele, J. B. (2004). Howard Hughes: His life and madness (reissue edition).

 New York: W. W. Norton & Company.
- Barlow, D.H. (2004). Anxiety and its disorders. Chapter 8. Guilford, London, New York.
- Beaty, R. E., Benedek, M., Wilkins, R. W., Jauk, E., Fink, A., Silvia, P. J., ... Neubauer, A. C. (2014). Creativity and the default network: A functional connectivity analysis of the creative brain at rest. *Neuropsychologia*, *64*, 92–98.
- Beck, A. T., Steer, R. A., Ball, R., & Ranieri, W. (1996). Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. *Journal of Personality Assessment*, 67(3), 588–597.
- Berthier, M. L. M. D., Kulisevsky, J. M. D., Gironell, A. M. D., & Lopez, O. L. M. D. (2001). Obsessive-Compulsive Disorder and Traumatic Brain Injury: Behavioral, Cognitive, and Neuroimaging Findings. *Neuropsychiatry, Neuropsychology, & Behavioral Neurology*, 14(1), 23–31.

- Black, D. W., & Gaffney, G. R. (2008). Subclinical Obsessive-Compulsive Disorder in Children and Adolescents: Additional Results From a "High-Risk" Study. *CNS Spectrums*, *13*(S14), 54–61.
- Bloch, M. H., Sukhodolsky, D. G., Dombrowski, P. A., Panza, K. E., Craiglow, B. G., Landeros-Weisenberger, A., ... Schultz, R. T. (2011). Poor fine-motor and visuospatial skills predict persistence of pediatric-onset obsessive-compulsive disorder into adulthood. *Journal of Child Psychology and Psychiatry*, 52(9), 974–983.
- Borges, M. C., Braga, D. T., Iêgo, S., D'Alcante, C. C., Sidrim, I., Machado, M. C., ...

 Fontenelle, L. F. (2011). Cognitive Dysfunction in Post-Traumatic Obsessive—

 Compulsive Disorder. *Australian and New Zealand Journal of Psychiatry*, 45(1), 76–85.
- Boudreaux, E., Kilpatrick, G., Resnick, H. S., Best, C. L., & Saunders, B. E. (1998). Criminal victimization, posttraumatic stress disorder, and comorbid psychopathology among a community sample of women. *Journal of Traumatic Stress*, *11*(4), 665–678.
- Carson, S. H., Peterson, J. B., & Higgins, D. M. (2003). Decreased latent inhibition is associated with increased creative achievement in high-functioning individuals. *Journal of Personality and Social Psychology*, 85(3), 499–506.
- Chirila, C., & Feldman, A. (2012). Study of latent inhibition at high-level creative personality

 The link between creativity and psychopathology. *Procedia Social and Behavioral*Sciences, 33, 353–357.
- Christensen, K. J., Kim, S. W., Dysken, M. W., & Maxwell Hoover, K. (1992).

 Neuropyschological performance in obsessive-compulsive disorder. *Biological Psychiatry*, *31*(1), 4–18.

- Ciesielski, K. T., Beech, H. R., & Gordon, P. K. (1981). Some electrophysiological observations in obsessional states. *The British Journal of Psychiatry*, *138*(6), 479–484.
- Ciesielski, K. T., Hämäläinen, M. S., Geller, D. A., Wilhelm, S., Goldsmith, T. E., & Ahlfors, S. P. (2007). Dissociation between MEG alpha modulation and performance accuracy on visual working memory task in obsessive compulsive disorder. *Human Brain Mapping*, 28(12), 1401–1414.
- Ciesielski, K. T., Hämäläinen, M. S., Lesnik, P. G., Geller, D. A., & Ahlfors, S. P. (2005).

 Increased MEG activation in OCD reflects a compensatory mechanism specific to the phase of a visual working memory task. *NeuroImage*, 24(4), 1180–1191.
- Ciesielski, K. T., Rauch, S. L., Ahlfors, S. P., Vangel, M. E., Wilhelm, S., Rosen, B. R., & Hämäläinen, M. S. (2012). Role of medial cortical networks for anticipatory processing in obsessive-compulsive disorder. *Human Brain Mapping*, *33*(9), 2125–2134.
- Dirson, S., Bouvard, M., Cottraux, J., & Martin, R. (1995). Visual memory impairment in patients with obsessive-compulsive disorder: A controlled study. *Psychotherapy and Psychosomatics*, *63*(1), 22–31.
- Faro, A., Geller, D. A., Joshi, G., & Mancuso, E. (2010). Treatment of pediatric obsessive-compulsive disorder: a review. *Journal of Child and Adolescent Psychopharmacology*, 20(4), 299+.
- Fineberg, N. A., Fourie, H., Gale, T. M., & Sivakumaran, T. (2005). Comorbid depression in obsessive compulsive disorder (OCD): Symptomatic differences to major depressive disorder. *Journal of Affective Disorders*, 87(2–3), 327–330.

- Flessner, C. A., Berman, N., Garcia, A., Freeman, J. B., & Leonard, H. L. (2009). Symptom profiles in pediatric obsessive-compulsive disorder (OCD): The effects of comorbid grooming conditions. *Journal of Anxiety Disorders*, 23(6), 753–759.
- Flor-Henry, P., T, L., J, Z., & G, B. (1979). Neuropsychological and power spectral EEG investigations of the obsessive-compulsive syndrome. *Biological Psychiatry*, *14*(1), 119–130.
- Flor-Henry, P. (1983). *The OCD syndrome. Cerebral basis of psychopathology*. Boston (MA): John Coright, 301-11.
- Fontenelle, L. F., Cocchi, L., Harrison, B. J., Shavitt, R. G., do Rosário, M. C., Ferrão, Y. A., ... Torres, A. R. (2012). Towards a post-traumatic subtype of obsessive—compulsive disorder. *Journal of Anxiety Disorders*, 26(2), 377–383.
- Freeman, D. (2013, December 3). Nikola Tesla fell in love with a pigeon--and six more freaky facts about the iconic inventor. Retrieved September 23, 2014, from http://www.huffingtonpost.com/2013/12/03/nicola-tesla-love-pigeon-facts-inventor_n_4320773.html
- Friedlander, L., & Desrocher, M. (2006). Neuroimaging studies of obsessive-compulsive disorder in adults and children. *Clinical Psychology Review*, 26(1), 32–49.
- Furnham, A., & Crump, J. (2013). The sensitive, imaginative, articulate art student and conservative, cool, numerate science student: Individual differences in art and science students. *Learning and Individual Differences*, 25, 150–155.
- Furnham, A., Hughes, D. J., & Marshall, E. (2013). Creativity, OCD, Narcissism and the Big Five. *Thinking Skills and Creativity*, *10*, 91–98.

- Galderisi, S., Mucci, A., Catapano, F., D'Amato, A. C., & Maj, M. (1995).

 Neuropsychological slowness in obsessive-compulsive patients. Is it confined to tests involving the fronto-subcortical systems? *The British Journal of Psychiatry*, *167*(3), 394–398.
- Garcia, A. M., Freeman, J. B., Himle, M. B., Berman, N. C., Ogata, A. K., Ng, J., ... Leonard,
 H. (2009). Phenomenology of Early Childhood Onset Obsessive Compulsive Disorder.
 Journal of Psychopathology and Behavioral Assessment, 31(2), 104–111.
- Geller, D. A., & March, J. (2012). Practice Parameter for the Assessment and Treatment of Children and Adolescents With Obsessive-Compulsive Disorder. *FOCUS*, *10*(3), 360–373.
- Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., ... Charney, D. S. (1989). The Yale-Brown Obsessive Compulsive Scale. I.

 Development, use, and reliability. *Archives of General Psychiatry*, 46(11), 1006–1011.
- Grados, M. A. (2003). Obsessive-compulsive disorder after traumatic brain injury. International Review of Psychiatry, 15(4), 350–358.
- Grados, M. A., Vasa, R. A., Riddle, M. A., Slomine, B. S., Salorio, C., Christensen, J., & Gerring, J. (2008). New onset obsessive-compulsive symptoms in children and adolescents with severe traumatic brain injury. *Depression & Anxiety* (1091-4269), 25(5), 398–407.
- Gross-Isseroff, R., Sasson, Y., Voet, H., Hendler, T., Luca-Haimovici, K., Kandel-Sussman, H., & Zohar, J. (1996). Alternation learning in obsessive-compulsive disorder. *Biological Psychiatry Biological Psychiatry*, *39*(8), 733–738.

- Gu, B.-M., Park, J.-Y., Kang, D.-H., Lee, S. J., Yoo, S. Y., Jo, H. J., ... Kwon, J. S. (2008).

 Neural correlates of cognitive inflexibility during task-switching in obsessive-compulsive disorder. *Brain: A Journal of Neurology*, *131*(Pt 1), 155–164.
- Heaton, R.K., Chelune, G.J., Talley, J.L., Kay, G.G. and Curtiss, G. 1993. Wisconsin Card Sorting Test manual, revised and expanded, Odessa, FL: Psychological Assessment Resources, Inc.
- Karno, M., Golding, J. M., Sorenson, S. B., & Burnam, M. A. (1988). The epidemiology of obsessive-compulsive disorder in five US communities. *Archives Of General Psychiatry*, 45(12), 1094–1099.
- Kim, J.-J., Lee, M. C., Kim, J., Kim, I. Y., Kim, S. I., Han, M. H., ... Kwon, J. S. (2001). Grey matter abnormalities in obsessive—compulsive disorder. *The British Journal of Psychiatry*, *179*(4), 330–334.
- Kohli, A., & Kaur, M. (2006). Wisconsin Card Sorting Test: Normative data and experience. *Indian Journal of Psychiatry*, 48(3), 181–184.
- Krishna, R., Udupa, S., George, C. M., Kumar, K. J., Viswanath, B., Kandavel, T., ... Reddy, Y. C. J. (2011). Neuropsychological performance in OCD: a study in medication-naïve patients. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *35*(8), 1969–1976.
- Kuelz, A. K., Hohagen, F., & Voderholzer, U. (2004). Neuropsychological performance in obsessive-compulsive disorder: a critical review. *Biological Psychology*, 65(3), 185–236.
- Leckman, J. F., Denys, D., Simpson, H. B., Mataix-Cols, D., Hollander, E., Saxena, S., ... Stein, D. J. (2010). Obsessive–compulsive disorder: a review of the diagnostic criteria

- and possible subtypes and dimensional specifiers for DSM-V. *Depression & Anxiety* (1091-4269), 27(6), 507–527.
- Lesnik PG, Ciesielski KT, Hart BL, Benzel EC, Sanders JA. (1998). Evidence for cerebeller–frontal subsystem changes in children treated with intrathecal chemotherapy for leukemia: Enhanced data analysis using an effect sized model. *Archives of Neurology*, 55: 1561-1568.
- Lezak, M. D. (2003). Principles of neuropsychological assessment. Behavioral neurology and neuropsychology, 2.
- Lochner, C., Fineberg, N. A., Zohar, J., van Ameringen, M., Juven-Wetzler, A., Altamura, A. C., ... Stein, D. J. (2014). Comorbidity in obsessive–compulsive disorder (OCD): A report from the International College of Obsessive–Compulsive Spectrum Disorders (ICOCS). *Comprehensive Psychiatry*, 55(7), 1513–1519.
- Malloy, P. (1987). Frontal lobe dysfunction in obsessive-compulsive disorder. In *The frontal lobes revisited* (pp. 207–223). New York, NY, US: The IRBN Press.
- Mancebo, M. C., Garcia, A. M., Pinto, A., Freeman, J. B., Przeworski, A., Stout, R., ...

 Rasmussen, S. A. (2008). Juvenile-onset OCD: clinical features in children, adolescents and adults. *Acta Psychiatrica Scandinavica*, 118(2), 149–159.
- Mascltis, M., Rector, N. A., & Richter, M. A. (2003). Quality of Life in OCD: Differential Impact of Obsessions, Compulsions, and Depression Comorbidity. *Canadian Journal of Psychiatry*, 48(2), 72.
- Mast, A. (2014, February 4). America's Forgotten Innovator, Nikola Tesla. Retrieved Sept. 23, 2014,

- http://www.magnet.fsu.edu/mediacenter/publications/flux/vol1issue1/magnetmilestones.h tml.
- Maia, T. V., Cooney, R. E., & Peterson, B. S. (2008). The Neural Bases of Obsessive-Compulsive Disorder in Children and Adults. *Development and Psychopathology*, 20(4), 1251–1283.
- Mataix-Cols, D., Alonso, P., Pifarré, J., Menchón, J. M., & Vallejo, J. (2002).

 Neuropsychological performance in medicated vs. unmedicated patients with obsessive—
 compulsive disorder. *Psychiatry Research*, 109(3), 255–264.
- Mataix-Cols, D., Nakatani, E., Micali, N., & Heyman, I., (2008). Structure of obsessive-compulsive symptoms in pediatric OCD. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47(7), 773–78.
- Mataix-Cols, D., Rosario-Campos, M. C. do, & Leckman, J. F. (2005). A multidimensional model of obsessive-compulsive disorder. *The American Journal of Psychiatry*, 162(2), 228–238.
- Mataix-Cols, D., & van den Heuvel, O. A. (2006). Common and distinct neural correlates of obsessive-compulsive and related disorders. *The Psychiatric Clinics of North America*, 29(2), 391–410, viii.
- McCrimmon, A. W., & Smith, A. D. (2013). Review of the Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-II). *Journal of Psychoeducational Assessment*, 31(3), 337–341.
- Menzies, L., Achard, S., Chamberlain, S. R., Fineberg, N., Chen, C.-H., del Campo, N., ...

 Bullmore, E. (2007). Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain*, 130(12), 3223–3236.

- Menzies, L., Chamberlain, S. R., Laird, A. R., Thelen, S. M., Sahakian, B. J., & Bullmore, E.
 T. (2008). Integrating evidence from neuroimaging and neuropsychological studies of obsessive-compulsive disorder: The orbitofronto-striatal model revisited. *Neuroscience & Biobehavioral Reviews*, 32(3), 525–549.
- Millet, B., Kochman, F., Gallarda, T., Krebs, M. O., Demonfaucon, F., Barrot, I., ...

 Hantouche, E. G. (2004). Phenomenological and comorbid features associated in obsessive–compulsive disorder: influence of age of onset. *Journal of Affective Disorders*, 79(1–3), 241–246.
- Moritz, S., Birkner, C., Kloss, M., Jahn, H., Hand, I., Haasen, C., & Krausz, M. (2002). Executive functioning in obsessive—compulsive disorder, unipolar depression, and schizophrenia. *Archives of Clinical Neuropsychology*, *17*(5), 477–483.
- Neuper, C., & Pfurtscheller, G. (2001). Event-related dynamics of cortical rhythms: frequency-specific features and functional correlates. *International Journal of Psychophysiology*, 43(1), 41–58.
- Olley, A., Malhi, G., & Sachdev, P. (2007). Memory and executive functioning in obsessive-compulsive disorder: a selective review. *Journal of Affective Disorders*, 104(1–3), 15–23.
- Pauls, D. L. (2008). The genetics of obsessive compulsive disorder: A review of the evidence.

 *American Journal of Medical Genetics Part C: Seminars in Medical Genetics, 148C(2), 133–139.
- Pauls, D. L., Abramovitch, A., Rauch, S. L., & Geller, D. A. (2014). Obsessive-compulsive disorder: an integrative genetic and neurobiological perspective. *Nature Reviews Neuroscience*, 15(6), 410–424.

- Penadés, R., Catalán, R., Andrés, S., Salamero, M., & Gastó, C. (2005). Executive function and nonverbal memory in obsessive-compulsive disorder. *Psychiatry Research*, *133*(1), 81–90.
- Purcell, R., Maruff, P., Kyrios, M., & Pantelis, C. (1998). Cognitive Deficits in Obsessive— Compulsive Disorder on Tests of Frontal–Striatal Function. *Biological Psychiatry*, *43*(5), 348–357.
- Rachman, S., & de Silva, P. (1978). Abnormal and normal obsessions. *Behaviour Research* and *Therapy*, 16(4), 233–248.
- Rasmussen, S. A., & Eisen, J. L. (1990). Epidemiology of obsessive compulsive disorder. *Journal of Clinical Psychiatry*, *51*(2, Suppl), 10–13.
- Rasmussen, S. A., & Eisen, J. L. (1994). The epidemiology and differential diagnosis of obsessive compulsive disorder. *The Journal of Clinical Psychiatry*, *55 Suppl*, 5-10; discussion 11-14.
- Rauch, S. L., Dougherty, D. D., Cosgrove, G. R., Cassem, E. H., Alpert, N. M., Price, B. H.,
 ... Fischman, A. J. (2001). Cerebral metabolic correlates as potential predictors of
 response to anterior cingulotomy for obsessive compulsive disorder. *Biological Psychiatry*, 50(9), 659–667.
- Remijnse, P. L., Nielen, M. M. A., van Balkom, A. J. L. M., Cath, D. C., van Oppen, P., Uylings, H. B. M., & Veltman, D. J. (2006). Reduced orbitofrontal-striatal activity on a reversal learning task in obsessive-compulsive disorder. *Archives of General Psychiatry*, 63(11), 1225–1236.
- Rosenberg, D. R., & Keshavan, M. S. (1998). Toward a Neurodevelopmental Model of Obsessive–Compulsive Disorder. *Biological Psychiatry*, 43(9), 623–640.

- Rosenberg, D. R., Keshavan, M. S., Dick, E. L., Bagwell, W. W., Master, F. P. M., & Birmaher, B. (1997). Corpus callosal morphology in treatment-naive pediatric obsessive compulsive disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 21(8), 1269–1283.
- Sasson, Y., Zohar, J., Chopra, M., Lustig, M., Iancu, I., & Hendler, T. (1997). Epidemiology of obsessive-compulsive disorder: a world view. *The Journal of Clinical Psychiatry*, 58 (Suppl 12), 7–10.
- Savage, C. R., Baer, L., Keuthen, N. J., Brown, H. D., Rauch, S. L., & Jenike, M. A. (1999).

 Organizational strategies mediate nonverbal memory impairment in obsessive—

 compulsive disorder. *Biological Psychiatry*, 45(7), 905–916.
- Schmahmann, J. D., Gardner, R., MacMore, J., & Vangel, M. G. (2009). Development of a brief ataxia rating scale (BARS) based on a modified form of the ICARS. *Movement Disorders: Official Journal of the Movement Disorder Society*, 24(12), 1820–1828.
- Shin, Y.-W., Kwon, J. S., Kim, J.-J., Kang, D.-H., Youn, T., Kang, K. W., ... Lee, M. C.(2006). Altered neural circuit for working memory before and after symptom provocation in patients with obsessive—compulsive disorder. *Acta Psychiatrica Scandinavica*, 113(5), 420–429.
- Shin, M. S., Park, S. J., Kim, M. S., Lee, Y. H., Ha, T. H., & Kwon, J. S. (2004). Deficits of organizational strategy and visual memory in obsessive-compulsive disorder.

 Neuropsychology, 18(4), 665–672.
- Shin, M.-S., Park, S.-Y., Park, S.-R., Seol, S.-H., & Kwon, J. S. (2006). Clinical and empirical applications of the Rey–Osterrieth Complex Figure Test. *Nature Protocols*, *1*(2), 892–899.

- Simonton, D. K. (1988). *Scientific Genius: A Psychology of Science*. Cambridge University Press.
- Simonton, D. K. (1999). *Origins of Genius: Darwinian Perspectives on Creativity*. Oxford University Press.
- Steketee, G., Frost, R., & Bogart, K. (1996). The Yale-Brown Obsessive Compulsive Scale: Interview versus self-report. *Behaviour Research and Therapy*, *34*(8), 675–684.
- Szeszko, P. R., MacMillan, S., McMeniman, M., Chen, S., Baribault, K., Lim, K. O., ...

 Rosenberg, D. R. (2004). Brain Structural Abnormalities in Psychotropic Drug-Naive

 Pediatric Patients With Obsessive-Compulsive Disorder. *American Journal of Psychiatry*,

 161(6), 1049–1056.
- Takeuchi, H., Taki, Y., Hashizume, H., Sassa, Y., Nagase, T., Nouchi, R., & Kawashima, R. (2011). Failing to deactivate: The association between brain activity during a working memory task and creativity. *NeuroImage*, 55(2), 681–687.
- Towey, J., Bruder, G., Hollander, E., Friedman, D., Erhan, H., Liebowitz, M., & Sutton, S. (1990). Endogenous event-related potentials in obsessive-compulsive disorder. *Biological Psychiatry*, 28(2), 92–98.
- Walitza, S., Wendland, J. R., Gruenblatt, E., Warnke, A., Sontag, T. A., Tucha, O., & Lange,
 K. W. (2010). Genetics of early-onset obsessive—compulsive disorder. *European Child & Adolescent Psychiatry*, 19(3), 227–235.
- Walitza, S., Zellmann, H., Irblich, B., Lange, K. W., Tucha, O., Hemminger, U., ... Warnke, A. (2008). Children and adolescents with obsessive-compulsive disorder and comorbid attention-deficit/hyperactivity disorder: preliminary results of a prospective follow-up study. *Journal of Neural Transmission*, 115(2), 187–190.

- Wang, X., Cui, D., Wang, Z., Fan, Q., Xu, H., Qiu, J., ... Xiao, Z. (2012). Cross-sectional comparison of the clinical characteristics of adults with early-onset and late-onset obsessive compulsive disorder. *Journal of Affective Disorders*, *136*(3), 498–504.
- Ward, L. M. (2003). Synchronous neural oscillations and cognitive processes. *Trends in Cognitive Sciences*, 7(12), 553–559.
- Wechsler, D. (2011). Wechsler Abbreviated Scale of Intelligence–Second Edition (WASI-II).

 San Antonio, TX: NCS Pearson.
- Zhu, W., Chen, Q., Xia, L., Beaty, R. E., Yang, W., Tian, F., ... Qiu, J. (2017). Common and distinct brain networks underlying verbal and visual creativity. *Human Brain Mapping*, 38(4), 2094–2111. https://doi.org/10.1002/hbm.23507

TablesTable 1

Results of Independent Samples t-test Between HA and SA Groups

Variables	HA				SA		t-test					
	n	M	SD	n	M	SD	t	df	Sig. (2-tailed)	Cohen's d		
IQ	28	109.76	9.06	29	96.34	10.88	5.06	55	<.001***	1.34		
Creativity	28	16.75	8.64	29	10.86	9.11	2.50	55	.015*	.66		
YBOCS	26	7.23	5.93	25	5.44	4.35	1.23	45.86	.224	.34		
BDI	26	5.62	5.12	25	9.36	5.83	-2.44	49	.016*	.77		
BARS	26	5.08	2.48	25	4.72	2.79	.483	49	.631	.16		
VCI	28	106.18	8.89	29	93.83	10.08	4.90	55	<.001***	1.30		
PRI	28	111.32	13.08	29	111.32	13.08	3.06	55	.003**	.81		
RCFTIMM	28	25.46	5.94	29	25.46	5.94						
WCSTPE	28	5.79	3.87	29	5.79	3.87						
WCSTCC	28	4.43	1.10	29	4.43	1.10						

Note. YBOCS=Yale-Brown Obsessive-Compulsive Scale; BDI=Beck Depression Inventory; BARS= Brief Ataxia Rating Scale; VCI=Verbal Comprehension Index; PRI = Perceptual Reasoning Index; RCFTIMM = Rey Complex Figure Test Immediate Recall; WCSTPE = Wisconsin Card Sorting Test Perseverative Errors; WCSTCC = Wisconsin Card Sorting Test Categories Complete. *p<.05 **p<.01 ***p<.001

Table 2

Kendall's Tau B Correlational Results for Entire Data Set

	M	SD	IQ	CREA	YBOCS	BDI	BARS	VCI	PRI	RCFT	WCST	WCST
										IMM	PE	CC
IQ	102.95	12.03										
Creativity	13.75	9.29	.27**									
YBOCS	6.35	5.24	.03	.26**								
BDI	7.45	5.74	22*	.073	.16							
BARS	4.90	2.61	01	04	.12	04						
VCI	99.89	11.30	.68**	.37***	.18	19	02					
PRI	106.30	13.07	.67**	.19*	03	10	09	.40***				
RCFTIMM	22.53	7.71	.43***	.23**	.17	10	.11	.38***	.42***			
WCSTPE	6.81	5.22	22*	08	.03	.10	.10	16	14	15		
WCSTCC	4.09	1.43	.18	.60	.01	00	02	.15	.20	.21*	55***	

Note. YBOCS=Yale-Brown Obsessive-Compulsive Scale; BDI=Beck Depression Inventory; BARS= Brief Ataxia Rating Scale; VCI=Verbal Comprehension Index; PRI = Perceptual Reasoning Index; RCFTIMM = Rey Complex Figure Test Immediate Recall; WCSTPE = Wisconsin Card Sorting Test Perseverative Errors; WCSTCC = Wisconsin Card Sorting Test Categories Complete.

*p<.05 **p<.01 ***p<.001

Table 3

Results of Independent Samples t-test for HH and SH Groups

Variables	НН		SH			t-test				
	n	M	SD	n	M	SD	t	df	Sig. (2-tailed)	Cohen's d
IQ	12	108.83	7.36	12	95.92	12.70	3.05	17.64	.007**	1.24
Creativity	12	17.75	8.90	12	12.92	9.37	1.30	22	.209	.53
YBOCS	12	12.92	2.75	12	9.25	2.56	3.38	22	.003**	1.36
BDI	12	6.50	5.57	12	10.25	5.55	-1.65	22	.113	.67
BARS	12	5.83	2.25	12	4.92	2.07	1.04	22	.310	.42
VCI	12	107.50	8.59	12	95.92	11.46	2.80	22	.01*	1.14
PRI	12	108.08	10.63	12	100.50	12.33	1.61	22	.121	.66
RCFTIMM	12	25.54	6.35	12	22.08	8.74	1.11	22	.279	.45
WCSTPE	12	6.67	5.58	12	9.17	8.62	84	22	.408	.34
WCSTCC	12	4.42	1.24	12	3.83	1.64	.96	22	.337	.40

Note. YBOCS=Yale-Brown Obsessive-Compulsive Scale; BDI=Beck Depression Inventory; BARS= Brief Ataxia Rating Scale; VCI=Verbal Comprehension Index; PRI = Perceptual Reasoning Index; RCFTIMM = Rey Complex Figure Test Immediate Recall; WCSTPE = Wisconsin Card Sorting Test Perseverative Errors; WCSTCC = Wisconsin Card Sorting Test Categories Complete. *p<.05 **p<.01 ***p<.001

Table 4

Kendall's Tau b Correlational Results for HH Group

	M	SD	IQ	CREA	YBOCS	BDI	BARS	VCI	PRI	RCFT	WCST	WCST
										IMM	PE	CC
IQ	108.83	7.36										
Creativity	17.75	8.90	.00									
YBOCS	12.92	2.75	12	.55*								
BDI	6.50	5.57	13	.34	.57*							
BARS	5.83	2.25	08	02	13	.02						
VCI	107.50	5.59	.52*	.03	28	25	22					
PRI	108.08	10.63	.50*	.17	.31	.02	.00	05				
RCFTIMM	25.54	6.35	.14	.02	03	27	.41	14	.41			
WCSTPE	6.67	5.58	41*	.29	.19	.19	.41	30	.05	.05		
WCSTCC	4.42	1.24	.07	36	12	.07	14	07	.07	11	50	

Note. YBOCS=Yale-Brown Obsessive-Compulsive Scale; BDI=Beck Depression Inventory; BARS= Brief Ataxia Rating Scale; VCI=Verbal Comprehension Index; PRI = Perceptual Reasoning Index; RCFTIMM = Rey Complex Figure Test Immediate Recall; WCSTPE = Wisconsin Card Sorting Test Perseverative Errors; WCSTCC = Wisconsin Card Sorting Test Categories Complete *p<.05 **p<.01 ***p<.001

TABLE 5Kendall's Tau b Correlational Results for SH Group

	M	SD	IQ	CREA	YBOCS	BDI	BARS	VCI	PRI	RCFT	WCST	WCST
										IMM	PE	CC
IQ	95.92	12.70										
Creativity	12.92	9.37	.21									
YBOCS	9.25	2.56	.13	.22								
BDI	10.25	5.55	.46*	.00	02							
BARS	4.92	2.07	.10	23	61*	10						
VCI	95.92	11.46	.71**	.26	.26	26	10					
PRI	100.50	12.33	.61**	.19	.24	16	08	.66**				
RCFTIMM	22.08	8.74	.55*	.17	.16	05	.06	.61**	.53*			
WCSTPE	9.17	8.62	24	07	.03	.10	.17	.03	06	.03		
WCSTCC	3.83	1.64	.26	.11	02	.06	23	.19	.37	.22	74**	

Note. YBOCS=Yale-Brown Obsessive-Compulsive Scale; BDI=Beck Depression Inventory; BARS= Brief Ataxia Rating Scale; VCI=Verbal Comprehension Index; PRI = Perceptual Reasoning Index; RCFTIMM = Rey Complex Figure Test Immediate Recall; WCSTPE = Wisconsin Card Sorting Test Perseverative Errors; WCSTCC = Wisconsin Card Sorting Test Categories Complete**p*<.05 ***p*<.01 ****p*<.001