August 2016

Clarifying the Influence of Comorbid Depression on Response Inhibition in Obsessive-Compulsive Disorder and Trichotillomania

Gregory Berlin
University of Wisconsin-Milwaukee

CLARIFYING THE INFLUENCE OF COMORBID DEPRESSION ON RESPONSE INHIBITION IN OBSESSIVE-COMPULSIVE DISORDER AND TRICHOTILLOMANIA

Gregory S. Berlin

A Thesis Submitted in

Partial Fulfillment of the

Requirements for the Degree of

Master of Science

in Psychology

at

The University of Wisconsin-Milwaukee

August 2016

ABSTRACT

CLARIFYING THE INFLUENCE OF COMORBID DEPRESSION ON RESPONSE INHIBITION IN OBSESSIVE-COMPULSIVE DISORDER AND TRICHOTILLOMANIA

by

Gregory S. Berlin

The University of Wisconsin-Milwaukee, 2016
Under the Supervision of Professor Han Joo Lee, Ph.D.

Response inhibition performance in Obsessive-Compulsive and Related Disorders (OCRDs) is emerging as a potential neurocognitive endophenotype of these disorders. A point of needed clarification, however, is how unique such performance is to the OCRD spectrum. Specifically, it is unknown whether comorbid depression, a disorder that frequently occurs with OCRDs (60-80%) (Pallanti et al., 2011) and is also associated with cognitive deficits, can influence response inhibition observed in OCRDs. We sought to clarify whether response inhibition performance could be reliably accounted for OCRD symptomology (in obsessive compulsive disorder and trichotillomania specifically) even when taking into consideration the influence of comorbid depression. Additionally, we investigate the interplay between RI and associated OCRD factors in domains of impulsivity, incompleteness and life disability. We found that response inhibition performance is not accounted for OCRD symptomology in aggregate (i.e., OCD and trichotillomania), but is uniquely related to compulsion severity in the context of OCD. Additionally, response inhibition performance is largely unrelated to associated domains of impulsivity, incompleteness and overall life disability.

TABLE OF CONTENTS

Page

LIST OF FIGURES

LIST OF TABLES

Introduction

What are Obsessive-Compulsive and Related Disorders?

Obsessive Compulsive Disorder (OCD) is a debilitating psychiatric condition characterized by a pattern of intrusive, unwanted mental images or urges and repetitive behaviors or mental acts to reduce stress from such thoughts (DSM-5; American Psychiatric Association, 2013). More specifically, intrusive, unwanted images/urges/thoughts are defined as *obsessions,* while repetitive behaviors/mental acts in response to such obsessions are referred to as *compulsions* (DSM-5; American Psychiatric Association, 2013). OCD is estimated to have a prevalence rate of 1.2-2.3% (Ruscio et al., 2010). OCD symptomology is among the ten leading causes of psychosocial impairment and is associated with detrimental impact on everyday functioning (Lopez & Murray, 1998). OCD symptoms, even at subthreshold levels, are evident in seemingly healthy samples and can lead to functional impairment (de Bruijn et al., 2010).

Trichotillomania (TTM), or "Hair-Pulling Disorder," is characterized by the recurrent pulling out of one's own hair, repeated attempts to stop such pulling, as well as clinically significant distress or impairment from this habit (DSM-5; American Psychiatric Association, 2013). The 12-month prevalence of TTM is estimated at 1-2%, and there has been a reported gender preponderance of those affected (10:1, Females: Males) (Lochner et al., 2005). Though TTM is rarely accompanied by obsessional preoccupation, hair-pulling may be done in response to certain emotional states, or in response to a premonitory urge (Franklin et al., 2011).

OCD and TTM are classified together in the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5) as "Obsessive-Compulsive and Related Disorders (OCRDs)" along with such diagnoses as Body Dysmorphic Disorder (BDD), Skin-Picking Disorder (SPD) and Hoarding Disorder. These seemingly disparate conditions were grouped together based on

existing evidence regarding their symptom profiles, comorbidities, familiality, genetic and neural underpinnings, and treatment response (Phillips et al., 2010). In terms of phenomenology, for instance, TTM is grouped with OCD because hair pulling is thought to be a "body-focused repetitive behavior" (DSM-5; American Psychiatric Association, 2013). These changes were a major departure from DSM-IV where OCD was classified as an anxiety disorder and TTM was classified as an impulse-control disorder (Stein et al., 2010). Taken together, the changes in grouping for OCD and TTM represent a shift towards understanding these disorders dimensionally based on relatedness of symptoms, as well as examining a putative endophenotype that could characterize the dysfunction unique to the OC-Spectrum (Phillips et al., 2010; Chamberlain & Menzies, 2009).

In a similar vein, symptoms of disorders along the OC-Spectrum are roughly grouped together under the National Institutes of Mental Health Research Domain Criteria initiative (NIMH-RDoC). The RDoC initiative is one that hopes to foster the development of cross-cutting symptom domains that are informed by findings in neurobiology and behavioral research (Cuthbert, 2014). RDoC groups OC-symptomology under "Positive Valence Symptoms" and within this category attempts to understand their diverse presentations as habit-based behaviors (NIMH, n.d.). These disorders are then studied jointly via neurobiological, cognitive, behavioral and self-report techniques.

DSM and RDoC nosologic changes converge on the idea that disorders with seemingly different profiles may have common underlying pathological processes. Moreover, they support the notion that disparate disorders can be studied together to uncover common processes that underlie multiple symptomologies without clouding clear differences between diagnoses (Montague et al., 2012; Abramowitz & Jacoby, 2015).

In the spirit of this line of work, this project concurrently studies OCD and TTM to uncover similarities and differences between these disorders on targets of interest. OCD and TTM in particular share the important phenomenological characteristic of "irresistible urges to perform unwanted repetitive behavior" (Chamberlain et al., 2007b). The phenomenological link between TTM and OCD may be particularly important considering that other OC-Spectrum conditions, such as BDD, do not always have as clear repetitive behaviors (Browne et al., 2014; Frare et al., 2004). Additional points of evidence from familiality (Bienvenu et al., 2000), treatment response (Chamberlain et al., 2007b), and genetic risk factors (Monzani et al., 2014) have informed the putative connection between OCD and TTM. Of course, these points are not meant to muddle important differences between the conditions (Chamberlain et al., 2007b). However, these evidence suggest that studying OCD and TTM together may aid in clarifying cross-cutting symptom factors relevant to a broader OC-Spectrum.

What is Response Inhibition? Description and Theories

Response inhibition (RI) is a cognitive process that refers to the ability to inhibit or suppress responses that are inappropriate or no longer required (Verburggen & Logan, 2009). It is a cognitive process that is key to many aspects of executive function, especially working memory, and is paramount to flexible behavior across different environments (Diamond, 2013). RI, as used in this project, refers to a faculty central to the deliberate inhibition of inappropriate *motor* responses. Real-world analogies of RI abound, such as stepping on the brakes in a car when a traffic light suddenly changes.

To understand RI fully, one needs to understand its relationship to other varied forms or cognitive and motor control. RI is functionally different than other forms of inhibitory control such as *reactive inhibition*, where inhibition is a side effect of executing a tangential process

(i.e., response conflict, proactive inhibition in memory tasks). In addition, RI is differentiated from "bottom-up" inhibitory control where a distracting stimulus can grab one's attention and inhibit an ongoing process regardless of conscious control (Diamond, 2013). RI is more appropriately characterized as "top-down" control, or, inhibition driven by active and focused control (Dalley et al., 2011).

It is generally assumed that RI is a *ballistic* cognitive process. In other words, RI theory posits that some motoric actions, after a certain point of initiation, functionally cannot be stopped (Logan, 1994). The point at which a behavior transitions from focused control and into non-inhibitable action is referred to as the *point-of-no-return* (Verbruggen & Logan, 2009). From this theory it follows that the strength of an inhibitory faculty, and any influence a paradigm exerts on manipulating successful inhibition, must come before this ballistic initiation is executed. Additionally, some RI researchers theorize that excitatory and inhibitory processes behind a motoric action are largely independent, and whether a response is inhibited depends on whether a "go" or "stop" process is stronger (Logan, 1994). This idea is what is referred to as a 'horse-race' model, because the success in inhibition is characterized as a 'race' between go and stop processes, and whichever process finishes first is thought to determine the success or failure of the inhibition attempt.

Within RI are multiple inhibitory sub-processes, each of which is probed through a different measurement paradigm. First, one form of RI is *action cancellation*, which refers to the act of cancelling an ongoing response when a signal to stop emerges (Schachar et al., 2007). Action cancellation may also be defined as inhibition of a motor response during the execution of such response (Eagle et al., 2008). The commonly used test of response inhibition is the stop-signal task (SST) (Verbruggen et al., 2008b). A second form of RI is in *action*

restraint/withholding, which captures inhibition of a motor response before a response is initiated (Eagle et al., 2008). This feature of RI is measured through the Go/No-Go Task (GNG) (Bohne et al., 2008). A third form of RI is called *interference control*, which refers to suppression of interference due to stimulus competition and is commonly measured through the Flanker Task (Friedman & Miyake, 2004).

Note that the taxonomy of functions within RI, as well as the relationship between RI and other cognitive and executive functions, are an emerging area in the general literature; while some point to the independence of these functions (Khng & Lee, 2014), others state that these RI processes are intertwined with other cognitive mechanisms and with each other (Roebers et al, 2010; Verbruggen et al., 2004). For instance, more complex models of inhibition have been proposed revision of the standard horse-race model of RI. For instance, while the strength of go and stop processes (and the difference of strength between) may contribute to govern whether a ballistic process is inhibited, internal decisional choices, perceptual processing of go and stop signals and executive monitoring of success/failure on stop-trials have all been shown to account for reaction time on stop trials (Logan et al., 2014).

The SST, used to measure action cancellation, has been one of the most widely used tasks in capturing RI across contexts and disorders (Logan et al., 2014). The SST is a computerized measure that presents a series of "go stimuli" (arrows) which subjects respond to with key presses. Subjects are instructed that periodically a beep sound ("stop-signal") will follow the presentation of an arrow and in these instances they are to withhold a response. Presentation of a stop-signal after go-stimulus comes after a short delay ("stop-signal delay") that is varied dynamically depending on subject performance.

The core variable from the SST is a stop-signal reaction time (SSRT). SSRT is calculated by subtracting the mean stop-signal delay (SSD), varied according to current performance, from subject's reaction time on no-stop trials (goRT), such that the formula is SSRT = goRT - SSD. Figure 1 illustrates the structure of the SST paradigm. Conceptually, SSRT is the finishing time of a competition between go and stop processes; it provides an index of the success and efficiency of a participant attending to and terminating a motor response already in progress. SSRT is reflected in many motor actions that involve competition between go and stop processes; imagine, for instance, a baseball player withholding a swing in progress when he/she sees a bad pitch.

The SST has a number of benefits beyond other measures of RI. For one, the SST may be able to generate the most descriptive and reliable results from any RI measure because each trial can be generated dynamically based on performance (Verburggen & Logan, 2009; Congdon et al., 2012). The SST has also been found to be clinically useful for characterizing dysfunction in a number disorders, especially in attention-deficit/hyperactivity disorder (ADHD) (Alderson et al., 2007; Bari & Robbins, 2013; Aron & Poldrack, 2005). Important models have been developed to explain SST performance (i.e., race model (Logan et al., 2014)), which allow inference into underlying cognitive mechanisms of inhibition. As will be discussed in the following section, action cancellation and the SST may be particularly relevant for OCRDs in terms of clinical phenomenology. Because of its breadth of explanatory data, empirical support and clinical utility, the SST is the central measure of RI in this project.

Response Inhibition and OCRDs

RI performance in conditions along OCRD spectrum have been the focus of recent research in cognitive psychology (Chamberlain et al., 2005). Specifically, RI deficits have been found relatively consistently in adults with OCD when compared to control groups (Chamberlain et al., 2007a; Penades et al., 2007). Some have found that poor inhibitory control can differentiate between those with OCD and other anxiety disorders on a modified GNG task (e.g., panic disorder) (Bannon et al., 2008). In addition, those with OCD have been found to have different patterns neural of activation (fMRI) on GNG tasks compared to controls (Stern & Taylor, 2014). Individuals with OCD and their first degree relatives (FDRs) were compared against healthy controls and it was found that both OCD and FDRs groups showed impaired RI on the Stroop task (Rajender et al., 2011). A meta-analysis conducted by Leopold & Backenstrass (2015) concluded that specific domains within OCD (i.e., checking compulsions) may be particularly associated with poor RI control compared to other OC symptoms.

RI may be useful in conceptualizing disorders across the OCRD spectrum as well. Patterns of poor inhibitory motor control have been observed in TTM as well as OCD (Bohne et al., 2008). Chamberlain et al. (2006) found that an OCD and TTM sample both displayed RI deficits on SST compared to controls, with the TTM showing worse performance. Grant et al. (2011) also found a similar pattern of RI performance in TTM and SPD, with worse performance in the SPD sample. Poor RI performance in SPD has been found in additional studies using the SST (Odlaug et al., 2010). RI performance in hoarding disorder (HD) has not been researched extensively, but Morein-Zamir et al. (2014) found that individuals with HD do not differ in RI task performance compared to individuals with OCD and comorbid HD. No work has been done using RI paradigms in BDD.

On a conceptual level, RI and the notion of impaired inhibitory control is consistent with the nature of dysfunction in these disorders; phenomenologically, OCRDs are characterized by persistent, repetitive, dystonic thoughts or behaviors that patients often wish they could inhibit but cannot (Abramowitz & Jacoby, 2015), and RI is the ability to willfully stop habitual action. A frequent report of those with TTM, for instance, is that once a pulling episode has started it is difficult to stop. Further, this repeated failure to stop pulling is a core feature of TTM (Jones et al., 2010). Where RI is a cognitive function present in everyday life, RI in the OC-Spectrum may be a clinical manifestation of a maladaptive cancellation process (Bannon et al., 2008). Some have found that this maladaptive stopping process is evident in OCD regardless of whether the task involves motor inhibition or thought inhibition (Morein-Zamir et al., 2010). In addition, RI dysfunction may be consistent with the underlying neurocircuitry of OCRDs; for instance, it has been reported that RI performance correlates with understood biological correlates of OCD (i.e., orbitofrontal cortex and right inferior frontal cortex) (Grant & Kim, 2014; Aron et al., 2014).

Cognitive assessment of OCRDs, especially through RI, may be an important step forward scientifically and clinically. OCRDs are heterogeneous in their presentation, so finding a common cognitive presentation shared by and unique to OCRDs may aid in clinical conceptualization and diagnostic utility. For instance, individuals may have diagnoses that both fall in an OCRD class, and they may share genetic influence in how their symptoms present, but their overt symptoms may share few similarities. Moreover, common cognitive presentations shared between these conditions can help build upon a putative "neurocognitive endophenotype" for OCRD conditions (Robbins et al., 2012). In other words, these shared cognitive profiles between disorders are thought to provide a bridge between the behaviors we can observe overtly, and basic neurobiological and genetic findings (Montague et al., 2012). Some have posited that

this specific cognitive ability may be paramount to how we conceptualize dysfunction the OCRD spectrum as a failure of top-down inhibitory control (Chamberlain et al., 2005).

Unknown Factors Regarding Response Inhibition in OCRDs

Despite the work that has been done with RI in the OCRD spectrum there are apparent discrepancies and problems that need to be addressed. First, it is unclear how reliably and consistently RI performance can be measured in OCRDs. Systematic review done by Wright et al. (2014) on specific measures of RI (GNG, Connors Continuous Performance Task (CPT), Sustained Attention to Response (SART)) illustrate that deficits in inhibitory control are most profound in Bipolar Disorder, ADHD and Autism, while OCD shows medium effect sizes for RI which are most pronounced in the cognitive capability of cancellation. Conversely, a meta-analysis of SST performance found a larger effect size for RI in OCD than ADHD (Lipszyc & Schachar, 2010). Additionally, Abramovitch et al. (2015) found that RI performance in a GNG were impaired in a subclinical analogue sample when compared to controls, suggesting that these difficulties with RI are present and related to OC-symptoms regardless of clinical diagnosis; this finding is consistent with the idea of RI as a potential endophenotype of OCRDs. In contrast, multiple studies have not found any difference in RI performance between OCD and controls (Tolin et al., 2014; Blom et al., 2011). Thomas et al. (2014) found no difference between panic disorder and OCD in GNG performance and also found similar patterns of electroencephalogram (EEG) activation in these two disorders when compared against healthy controls. Stern & Taylor (2014) suggest that RI tasks are themselves confounded by mechanisms that are impaired in OCD, such as performance monitoring, which may result in falsely attributing RI deficits as core to OC-symptomology.

Second, it has been posited that RI performance in OCRDs can be better accounted for by comorbid depression. Depression is a multifaceted condition that is also accompanied by a host of cognitive deficits (McIntyre et al., 2013; Austin et al., 2001; Harvey, 2007). These deficits in depression may also be associated with tangential features necessary for effective SST performance, namely executive functioning processes such as working memory and planning ability. Some have suggested that OCD is accompanied by a set of unique cognitive functioning deficits that are independent of depression (Aycicegi et al., 2003), and that the difficulties in depression do not necessarily overlap with those in OCD (Vergara-Lopez et al., 2013). Others, however, posit that both OCD and depression share deficits in selective attention, and suggest that this process is key to RI performance in both conditions (Koch & Exner, 2015).

The potential influence of comorbid depression on RI performance in OCRDS may be particularly troubling for at least two reasons. First, depression has also been found to have a moderate degree of difficulty in inhibitory control (Lau et al., 2007; Schulz et al., 2007). There is a growing body of literature suggesting that poor inhibitory control in depression may be particularly accentuated when stimuli in RI measures are emotionally relevant (Albert et al., 2010). It may be the case that the emotional quality of stimuli themselves, specifically in their degree of eliciting arousal, may affect RI performance (De Houwer & Tibboel, 2010; Verbruggen & De Houwer, 2007). Some have found that individuals with Major Depressive Disorder (MDD) have slower reaction time generally than controls, which may in turn affect RI performance (Schlosser et al., 2013). With specific regards to the SST, slow reaction time may lead to a distorted SSRT; because go-reaction time is a critical component of calculating SSRT, a systematic slowing of response time can muddle the value of this RI index. Dillon et al. (2015) suggest that mood related aspects of depression, such as anhedonia, may also affect RI

performance. Interestingly, the presence of comorbid depression seems to affect RI performance in other disorders besides OCD, such as alcohol dependence (Jakubczyk et al., 2012).

Second, depression is a common comorbidity of OCD. A breadth of data suggests that depression is the most common comorbidity in OCD (Pallanti et al., 2011), and is ten times more prevalent in OCD patients than in the general population (Denys et al., 2004). Pallanti et al. (2011) suggest that between 60-80% of individuals with OCD will experience at least one major depressive episode in their lifetime. Further, these comorbid depressive features can have important implications for treatment response (Torp et al., 2015). While comorbid depression does not necessarily produce more severe OC-symptoms, such OC-symptoms are a significant predictor of depression (Yap et al., 2012). Thus, if depression may account for a modicum of poor RI performance, and depression is highly comorbid with OCD, then our current understanding of RI in OCRDs may be inaccurate in not having factored out the influence of cognitive and executive functioning deficits associated with depression. The problem then becomes: how can one be sure that RI performance in OCRD are truly related to their core symptomologies and not their comorbidities?

There are additional dimensions of psychopathology that may be important in interpreting RI performance in OCRDs. First, the relationship between trait impulsivity and RI may be an important one. Many disorders characterized by impulsivity, such as attention-deficit hyperactivity disorder (ADHD), show marked impairments on the same measures of RI and interference control (van Velzen et al., 2014; Sjoerds et al., 2014). Disorders characterized by dysfunctional impulse control, such as pathological gambling (PG), may also show impaired RI (Grant & Kim, 2014). Additionally, individuals affected by ADHD and OCRDs demonstrate a similar pattern of neural activation during SST, namely reduced inhibition-related activation of

the caudate nucleus, inferior frontal gyrus (IFG) and supplementary motor area (SMA) (van Velzen et al., 2014). The fact that RI tasks show similar performance in disorders of compulsivity (i.e. OCRDs) and impulsivity (i.e., ADHD, PG) can imply a number of things, such as a putative relationship between these symptom dimensions (Robbins et al., 2012; Berlin & Hollander, 2013) or something else entirely. The current state of the literature, however, does not address how RI functions within broad domains of compulsivity and impulsivity and more work is needed to clarify its standing.

Second, "not just rightness," or incompleteness, may be and important variable related to OCRD symptomology and RI. Not just right experiences (NJREs) refer to a mismatch between a perceived current affective state compared to a desired state of experiences (Fergus, 2014), or, an "irremediable sense that one's actions or experiences are not 'just right'" (Summerfeldt, 2004). These experiences have recently emerged in the literature as an important symptom domain within OCRDs (Coles et al., 2005), and challenge the conception that OC-symptomology is driven solely by harm avoidance (Taylor et al., 2014). One study analyzed incompleteness in OCRDs and compared them against other conditions (PG and eating disorders) and concluded that this domain was specific to OCRD symptomology (Sica et al., 2015). Incompleteness may be related to RI as a motivational factor; incompleteness may drive someone to complete a task to fullness, and this difficulty stopping a behavior prior to the point of "just-rightness" could be either a cause or reflection of RI (Ecker & Gönner, 2008). This area of research is quite new and there is a paucity of data regarding how NJREs fit together with cognitive functioning in OCRDs and RI. However, given the potential importance of both RI and incompleteness in OCRD symptoms, it is critical to see how these variables are related.

Lastly, it is unknown whether RI performance is a clinically useful indicator in conceptualizing patient disability. No research exists that studies the relationship between RI and quality of life (QoL) or overall disability. OCD itself has been characterized by both functional impairment and reductions in QoL when compared with healthy controls (Huppert et al., 2009). Some have posited that two factors within OCD are main contributors to life distress: obsessional severity and comorbid depressive symptomology (Masellis et al., 2003). Some suggest that different levels of impairment and relapse rates depend on different obsessional content (Eisen et al., 2013; Matsunaga, 2013). However, the active ingredient within OCD that leads to general life impairment is still unclear. Work needs to be done to analyze how specific symptom domains and relevant cognitive deficits may contribute to QoL and disability among OCRDs.

Study Aims and Hypotheses

The aim of this project was to parse out the influence of comorbid psychopathology on RI performance in OCRDs. To this end, we utilized data from an ongoing clinical trial to examine RI performance in OCD and TTM along with symptoms of depression. Primarily, we hypothesized that RI performance would be better explained by symptoms of OCRDs than by symptoms of comorbid depression. Specifically, we predicted that variance in the core measure of RI in the SST would be better accounted for by OCRD symptomology than by symptoms of comorbid depression.

We were also interested in probing how underlying, OC-related traits were related to RI performance. Specifically, we sought to investigate how domains of impulsivity and incompleteness related to SST performance in OCD and TTM. As literature in these area are not developed well, we explored whether the magnitude of impulsivity and incompleteness would be statistically related to the SST indices that reflect core RI. Additionally, we aimed to study how

overall life disability is associated with RI in OCD and TTM. Again, though no data are available to suggest a directional relationship between these two factors, we expected that the magnitude of SSRT would increase in tandem with difficulties in QoL.

Methods

Participants

For the current study, participants were considered for eligibility assessment if they were ages 18-60, were actively symptomatic for OCD or TTM, did not report or evidence any severe psychopathology on a phone screening (i.e., bipolar disorder, substance use disorders, schizophrenia, suicidality, etc.), and were not in current cognitive-behavioral therapy for OCRDs (i.e., exposure and response prevention, habit reversal therapy).

Sixty-one individuals were assessed at the main study visit for this project, but 49 individuals were included in the final analysis. Seven individuals were excluded because they were assessed prior to the inception of this masters project, thus they did not receive the full assessment battery with measures of depression. Additionally, 5 individuals were excluded for reasons of either not having OCD or TTM, or presenting with a cognitive complaint that made assessment with SST invalid.

The mean age of participants was 29.59 (SD = 10.80). There was a gender preponderance for females (female = 67.3%, n = 33; male = 32.7 %; n = 16). The sample was largely Caucasian but showed some variance in race: Caucasian = 81.6% (n = 40), African-American = 10.2% (n = 5), Two or more races = 6.1% (n = 3). For ethnicity, 2% (n = 1) of our sample identified as Hispanic/Latino. The mean WASI FSIQ-2 score for our sample was 105.04 (SD = 10.45). These and additional demographic characteristics are presented in Table 1.

Fifty-five percent of our sample had only OCD as an OCRD diagnosis (n = 27), 26.5% of our sample had only TTM as an OCRD diagnosis (n = 13), 6.1% of our sample had primary OCD with secondary TTM (i.e., OCD+TTM, n = 3) and 12.2% had primarily TTM with secondary OCD (i.e., TTM+OCD, n = 6). Those with dual diagnoses were grouped together for analyses due to a small individual sample. Our final analytic groups were as follows: OCD only (n = 27), TTM only (n = 13) and Combined diagnoses (n = 9).

In terms of comorbidities, 42.9% (n = 21) individuals met criteria for current major depressive disorder (i.e., MDD-current depressive episode), and 8.2% (n = 4) for persistent depressive disorder (PDD). For other comorbidities, from most to least frequently diagnosed, 57.1% (n = 28) met criteria for generalized anxiety disorder (GAD), 44.9% (n = 22) for social anxiety disorder, 18.4% (n = 9) for post-traumatic stress disorder (PTSD), 16.3% (n = 8) for a specific phobia, 14.3% (n = 7) for ADHD, 8.2% (n = 4) for agoraphobia, 6.1% (n = 3) individuals for panic disorder, 6.1% (n = 3) for separation anxiety disorder, 6.1% (n = 3) for body dysmorphic disorder (BDD), 6.1% (n = 3) for substance use disorder (SUD), 6.1% (n = 3) who reported diagnoses on domains not assessed (i.e., dermatillomania), 4.1% (n = 2) for bulimia nervosa, 2% (n = 1) for illness anxiety disorder, and 2% (n = 1) for alcohol use disorder. Chi-square analyses showed significant group differences in rates of diagnosis for separation anxiety, PTSD and SUD. No group differences were found on other demographic variables or diagnostic status.

Data collection procedures

Data were collected as part of a clinical trial testing the effectiveness of RI training in OCD and TTM. Specifically, data for this project were collected as part of an on-site "full

eligibility assessment," which took place following online and phone-based pre-screen procedures and prior to baseline and randomization.

Prior to eligibility assessment, individuals interested in the project contacted study staff and completed self-report measures of illness severity (Obsessive-Compulsive Inventory Revised (OCI-R) (Wootton et al., 2015); Massachusetts General Hospital Hairpulling Scale (MGH) (Keuthen et al., 2007). Subjects who scored above a certain threshold (OCI-R $\geq$ 21 or MGH $\geq$ 12) were contacted for assessment of exclusion criteria by phone to ensure that we were identifying an actively symptomatic sample (Monzani et al., 2014; Stein et al., 2010). Individuals were screened to ensure that they were ages 18-60, were not currently experiencing mania, psychosis, alcohol use or substance use disorder. Though not specifically assessed, participants were excluded if they reported suicidality. Individuals were also assessed to ensure the presence of OCD or TTM on the Mini International Neuropsychiatric Interview 6.0 (MINI-6.0) OCD module and TDI. Participants who met these criteria were invited to the University of Wisconsin-Milwaukee for a single-session eligibility assessment for the larger clinical trial to which they were interested in. Note, however, that information endorsed/not endorsed on phone screening was not truly confirmed until on-site screening; subjects over/underreported symptoms on the phone which led to a heterogeneous clinical sample in this project. Actual subject characteristics included those who would not meet criteria for the main clinical trial.

Measures

All measures given for this project were administered in a single session at the University of Wisconsin-Milwaukee. At the study visit where data was collected, participants were assessed for general diagnostic status (Anxiety Disorders Interview Schedule for DSM-5 [ADIS-5]), OCRD diagnostic status and symptom severity (Trichotillomania Diagnostic Inventory [TDI];

self-report versions of the Yale-Brown Obsessive-Compulsive Scale [Y-BOCS]; MGH), and RI (SST). Additional self-report measures for our exploratory aims were given at this eligibility session as well. A full list of measures used for this project are provided in Table 2. All self-report measures excluding BDI-II were given via Qualtrics on-site in the laboratory. The BDI-II was given on paper. A detailed description of measures follows:

Anxiety Disorders Interview Schedule for DSM-5 Disorders (ADIS-5) (Brown & Barlow, 2013). The ADIS-5 is a structured clinical interview designed to assess and diagnose DSM-5 disorders (i.e., depression, anxiety, trauma, OCRDs and SUDs).

Self-Report Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) (Federici et al., 2010). The YBOCS is the gold-standard assessment tool for OC-symptoms. The instrument uses a checklist of current OCD symptoms, and uses these symptoms as a reference for a 10-item severity score. The self-report version has been shown to have good convergence with the clinician-rated version, though clients may underreport symptoms on the self-report version.

Massachusetts General Hairpulling Scale (MGH) (Keuthen et al., 2007). The MGH is a 7-item brief self-report measure of hair pulling that measures urges, active pulling time, perceived control and associated distress.

Beck Depression Inventory-II (BDI-II) (Beck et al., 1996). The BDI-II is a 21-item self-report questionnaire that measures major domains of depression symptomology (i.e., cognitive, mood, and somatic symptoms as well as suicidality). Items on the BDI-II range from 0-3 and a total score is calculated by summing all 21 items. Scores from the BDI-II can produce both continuous measures of depression (i.e., scores from 0-63) and

categorical cut-off scores (i.e., "moderate depression" as scores from 20-28) (Yap et al., 2012). The BDI-II, as used in this project, was mainly for assessment of suicidality.

Depression Anxiety Stress Scale (DASS) (Lovibond & Lovibond, 1995). The DASS is a 42-item self-report scale that probes three domains: Depression (subscales assess mood components), anxiety (somatic symptoms and subjective experience), and stress (levels of non-chronic arousal). Questions are in a four-point likert format ranging from 0 ("did not apply to me at all") to 4 ("applied to me very much, or most of the time").

Barratt Impulsiveness Scale-11 (BIS-11) (Patton et al., 1995)**.** The BIS-11 is a 30-item self-report questionnaire that probes domains of attentional, motoric, cognitive, inhibitory and perseverance impulsivity and is one of the most widely used measures of impulsivity. The BIS-11 produces a total score as well as scores for six first-order factors and three second-order factors.

Obsessive-Compulsive Trait Core Dimensions Questionnaire (OC-TCDQ) (Summerfeldt et al., 2001). The OC-TCDQ is a 20-item self-report measure that assesses core dimensions of OCD in regards to harm avoidance and incompleteness. Items are in a likert format (0= "Never applies to me, 4= "Always applies to me"). The questionnaire produces a two-factor structure (harm avoidance and incompleteness). Both subscales in this measure have shown strong internal consistency in nonclinical samples (Coles et al., 2005).

Sheehan Disability Scale (SDS) (Sheehan et al, 1996). The SDS is a widely used questionnaire that utilizes three self-reported items to measure impairment in occupational, social, familial/home function. Each item is rated on a 0 ("not at all") to 10 ("very severely") scale. Items on this scale are accompanied by visual anchors (i.e.,

mildly, moderately, markedly). The SDS has been widely used to assess disability as a result of symptoms in primary care and treatment outcome research.

Wechsler Abbreviated Scale of Intelligence (WASI-II) (Wechsler & Hsiao-pin, 2011). The WASI-II is an abbreviated measure of overall cognitive and intellectual functioning appropriate for individuals ages 6 to 90. Individual subtests of vocabulary and matrix reasoning were administered to get a short index of overall cognitive functioning.

Cognitive Assessment

Response inhibition performance was measured via the SST (Logan, 1994). The task is computerized and administered through Inquisit software (Verbruggen et al., 2008b). Subjects underwent one practice block with 32 trials and three testing blocks with 64 trials each. Stop-signals occurred randomly on 33% percent of trials such that there was a 3:1 ratio of no-stop-signals to stop-signal trials within each block. The stop-signal delay (SSD) was varied depending on subject performance (lengthened or shortened) such that the probability of a participant responding correctly to any given stop-signal trial was approximately 50%. This SSD was initially set at 250ms and adjusted up or down by 50ms depending on performance (max = 1150ms, min = 50ms). Data was recorded for response time, omission and commission errors, mean stop-signal delayed and probability of response for stop-signals. Stop-signal reaction time (SSRT) is the core measure of RI in this paradigm and was calculated by the software by averaging mean reaction times on ‘go’ trials without a stop-signal and subtracting the mean SSD (i.e., SSRT = goRT – SSD) (Chamberlain et al., 2007a; Verbruggen & Logan, 2009).

Results

Symptom Profile

A detailed description of symptom scores is provided in Table 3. Mean YBOCS scores for those who had only OCD as an OCRD diagnosis was 22.81 (SD = 5.27), and mean MGH scores for those with TTM as an only OCRD diagnosis were 17.07 (SD = 4.42). For those with both OCD and TTM, mean total YBOCS scores were 22.00 (SD = 4.66) and mean MGH scores were 18.11 (SD = 5.13). Mean SSRT for the whole sample was 223.71 (SD = 43.91) and the mean DASS-Depression (DASS-D) subscale was 12.57 (SD = 10.94; Score Descriptor = Mild Depression).

A one-way ANOVA was conducted to detect differences between diagnostic groups on the measures administered. No significant differences were found between groups on critical measures of depression, SSRT, SSD or goRT. We found significant differences between groups on measures of anxiety ($F(2, 46) = 3.24$), $p < 0.05$), attentional impulsivity ($F(2, 46) = 5.01$, $p < 0.05$), harm avoidance ($F(2, 46) = 11.70$, $p < 0.001$) and incompleteness ($F(2, 46) = 12.62$, $p < 0.001$). Post-hoc analysis with a Scheffé test of significance showed that the OCD and mixed group were elevated compared to the TTM ground on incompleteness, and the OCD group was elevated on anxiety, harm avoidance, and attentional impulsivity compared to the TTM group. This suggests that even though core variables of interest are comparable across groups, certain dimensional measures (i.e., anxiety) may need to be added as a covariate in statistical analysis in explaining SSRT.

Preliminary Analyses

Prior to testing our main hypothesis, we implemented two preliminary procedures. First, z-transformation was applied to OCRD symptom indices (i.e., YBOCS and MGH scores) in both

OCD and TTM groups. Thus, among those with OCD as a primary diagnosis, z-transformation took total YBOCS Scores for each participant, subtracted the mean score for the OCD group and divided that value by the standard deviation of the group ((YBOCS Score – M)/SD [m=0, SD=1]). In those with TTM as a primary diagnosis, z-score transformation followed the same procedure and instead used MGH scores ((MGH Score-M)/SD [m=0, SD=1]). These z-scores were consolidated together into a uniform 'OCRD z-score' to be entered as one variable to signify overall OCRD severity. This was to ensure that symptom scores could be analyzed together across the two conditions regardless of actual diagnostic status.

Second, a series of bivariate correlations were conducted to get a broad understanding of how critical variables of depression, OCD and TTM symptoms, and SST variables related to each other both within and between diagnostic groups. SST variables (i.e., SSRT, SSD, etc.) and symptom severity indices (i.e., OCRD z-scores, YBOCS, MGH) were entered into this correlation. Analysis was performed for the whole sample (i.e., RDoC approach) and across diagnostic groups to catch potentially important group differences. Results are presented in Tables 4 and 5.

Across the whole sample, SSRT was not significantly related to depression. However, SSRT was also not significantly related to the z-centered OCRD symptom scores. In fact, none of the seven indices from the SST were significantly related to OCRD severity or either measure of depression. There was a trending pattern of significance to suggest that depression severity was negatively associated with SSRT (i.e., less depression, longer SSRT). Moreover, depression showed a trending positive pattern with goRT and SSD.

The same variables were entered into a correlation, but data were split so the analyses were conducted for those with only OCD or TTM as an OCRD diagnosis. Amongst those with

OCD, neither YBOCS total scores nor obsessional severity were significantly related to SSRT. However, compulsion severity was significantly associated with SSRT ($r(26) = .433$, $p = 0.027$) whereas depression scores were not. The relationship between YBOCS compulsion scores and SSRT are reflected in a scatterplot in Figure 2. Though it did not reach significance level, YBOCS total scores were trending towards a pattern that suggest that worse OC-severity, especially amongst obsessional severity, is associated with lower percentage of correct responses on go trials ($r(26) = -.340$, $p = 0.09$).

Amongst those with TTM, hair pulling severity was not significantly related to SSRT, nor was SSRT significantly related to measures of depression. This comes in contrast to the OCD group, where compulsion severity was correlated with SSRT. Hair pulling severity was approaching significance levels with commission reaction time, overall go-trial reaction time and percentage of omissions. Due to small sample size ($n = 9$), the mixed diagnosis group analysis is not included here.

Results of these correlations suggest that SSRT may have a more specific relationship with symptom severity when considering an OCRD diagnosis. More specifically, these correlations suggest that certain symptoms may be more relevant to SSRT (i.e., compulsions vs. obsessions). Thus, we utilized dummy coding with hierarchical regression to quantitatively examine any differential patterns of OC-Symptom—RI performance association depending on primary diagnosis (OCD, TTM, or mixed diagnosis). Our two dummy coded variables represented three groups: OCD (reference group), TTM, and OCD with TTM (mixed diagnosis); D1 = OCD vs. TTM and D2 = OCD vs. Mixed Diagnosis. SSRT was the DV being predicted in this regression. In Step 1, we added the following variables to get a broad overview of how covariates would be accounting for RI performance: gender, DASS depression, stress and

anxiety scores. Depression, stress and anxiety scores were centered around their respective grand means to reduce potential multicollinearity issues. In Step 2, we added the two dummy coded variables (i.e., D1 = OCD vs. TTM, D2 = OCD vs. Mixed) and the z-centered OCRD scores. Lastly in Step 3, we added interaction scores for OCD vs. TTM and OCD vs. Mixed Diagnosis. Results are presented in Table 6.

There was a marginally significant interaction between the OCD vs. TTM status and the severity of OCRD symptoms in predicting SSRT (β = -.328, t = -1.996, p = 0.53). Additionally, there was a significant interaction between the OCD vs. mixed diagnosis status and the severity of OCRD symptoms in predicting SSRT (β = -.515, t = -3.025, p = 0.004). This interaction is illustrated in Figure 2. These findings suggest that there is some merit to looking beyond our sample in aggregate. Thus, for our subsequent analyses we use both our whole sample and specific diagnostic subgroups to explore important differences.

Primary Analyses

The central question of this project is whether RI performance, as defined by SSRT, is better accounted for by OCRD symptomology or by comorbid depressive symptoms. Our primary hypothesis was that among individuals diagnosed with OCRDs, the severity of OCRD symptoms would significantly predict RI even when comorbid depression was accounted for.

To test our main hypothesis, we utilized hierarchical linear regression. In this analysis, SSRT scores were entered as a dependent variable. In Step 1, DASS depression, anxiety and stress and gender were entered to produce a model to estimate the variance in SSRT accounted for by Step 1 variables (i.e., R^2). Anxiety and stress were added because they are common covariates of depression. Gender was added as a covariate because (a) there was a gender preponderance of females in our sample, and (b) depression has been linked generally to a higher

incidence in women. In Step 2, OCRD z-scores were entered to examine the R^2 change, or, to see the additional variance in SSRT that was significantly explained by OCRD symptoms even after for controlling for additional factors of depression, anxiety, stress and gender. Results of this and the following regression analyses are presented in Table 7.

Step 1 variables accounted for approximately 11.7% of the variance in SSRT, however this was not statistically significant ($R^2 = .117$, $F(4, 43) = 1.425$, $p > 0.05$). Adding OCRD z-scores in Step 2 only explained approximately 0.3% of the additional variance in SSRT ($R^2 \Delta = .003$, $F(5, 42) = 1.51$, $p > 0.05$; $\beta = .063$, $t = .406$, $p = .687$).

Based on the results of earlier correlational analyses, it was thought that SSRT values may be affected by the diagnostic status. Thus, we utilized regression and divided our sample based on whether participants had OCD, TTM or combined OCD+TTM. So, for our first sub-group analysis cases were selected if participants had only OCD (i.e., no TTM or OCD + TTM cases). We entered depression, anxiety and stress scores and gender in Step 1 of the regression and used non-z-centered YBOCS total scores as a predictor variable in Step 2. Here, Step 1 variables accounted for about 18.5% of the variance in SSRT ($R^2 =.185$, $F(4, 21) = 1.191$, $p = .344$), but when adding YBOCS total scores approximately 11.2% additional variance was explained in SSRT ($R^2 \Delta =.112$, $F(5, 20) = 1.686$, $p = .184$). Specifically within this model, YBOCS total scores were the best predictor of SSRT values and were approaching statistical significance ($\beta = .365$, $t = 1.782$, $p = .090$). This analysis was repeated but instead of YBOCS total scores being entered in Step 2, YBOCS subscales of obsession and compulsion severity were entered together. Here, OCD symptoms accounted for an additional 20% of variance in SSRT scores ($R^2 \Delta =.207$, $F(6, 19) = 2.043$, $p = .109$) and though obsessional severity was not a

predictor of SSRT (β = -.027, t = -.175, p = .863), compulsion severity was the only significant predictor of RI (β = .473, t = 2.473, p = .023).

These analyses were conducted in the same fashion with the TTM subsample. Gender, depression, anxiety and stress scores were entered in Step 1 of the regression and MGH total scores in Step 2. MGH scores here explained about 12.7% of the additional variance in SSRT which was non-significant ($R^2 \Delta$ =.127, F(5, 7) = 1.230, p = .387; β = -.403, t = -1.291, p = .238). Within this model, the only variable approaching significance as a predictor was general stress (β = -.882, t = -2.039, p = .081).

Lastly, we computed regression using cases with mixed diagnoses using the z-centered OCRD symptom scores as a Step 2 variable. OCRD severity in this mixed group only accounted for approximately 1.3% in the additional variance in SSRT and this was not statistically significant ($R^2 \Delta$ =.013, F(5, 3) = .622, p = .701; β = -.189, t = -.283, p = .796).

Taken together, our primary hypothesis that SSRT symptoms would be better accounted for by OCRD symptoms than by depression was only found in the OCD group, and not for those presenting with TTM or dual-diagnoses of OCD+TTM. Further, within those with OCD, compulsion severity was the best (and only) significant predictor of SSRT. Depression was not a significant predictor of SSRT in any model, but amongst those with TTM general stress was a variable that was approaching significance as a predictor of SSRT.

Secondary Analyses

An exploratory aim of this project was to evaluate the relationship between SSRT and associated symptom factors of OCRDs such as impulsivity [BIS-11], incompleteness [OC-TCDQ], and life disability [SDS]. Additionally, we were interested to see how these associated factors were related to core OC-symptomology. We expected to see that SSRT would increase

commensurate with levels of life impairment, impulsivity (particularly in terms of motor impulsivity) and harm avoidance/incompleteness. Additionally, we expected that those with higher OC-symptoms may have higher levels of life impairment.

To explore these relationships we utilized bivariate correlation. Analyses were conducted with the aggregate sample and by diagnostic subgroups of OCD and TTM (note: the mixed diagnostic group was not included due to small sample size (n = 9)). To generate a correlational matrix we entered z-transformed indices of symptom severity, SST variables, BIS sub-scale scores of attentional, motor and non-planning impulsivity, OC-TCDQ harm avoidance and incompleteness scores, SDS scores for global functioning impairment, and DASS anxiety, depression and stress subscale scores. For subgroup analyses, OCRD scores were substituted with either YBOCS (total, obsession and compulsion) or MGH total scores depending on the diagnostic group. We were interested primarily in how associated features of OCRDs related to (a) core OCRD symptoms via YBOCS, MGH and z-centered scores and (b) SSRT (Tables 8 & 9).

In the aggregate sample no features of impulsivity were significantly related to SSRT or OCRD severity. This was also the case for overall life impairment, in that neither SSRT nor OCRD severity showed a significant relationship. Neither harm avoidance nor incompleteness were significantly related to SSRT, and they were not significantly associated with overall OCRD severity. Taken together, none of our hypotheses that SSRT would be related to OC-associated factors were confirmed. Additionally, none of these associated factors were related to OCRD severity.

In the OCD group, again SSRT was not significantly related with any of the OC-associated of impulsivity, harm avoidance/incompleteness or overall life disability. Additionally,

these factors were not significantly related to core OCD symptomology of obsessions, compulsions or total OCD severity. The only factors that were related to core OCD symptoms were harm avoidance and incompleteness, but these factors are themselves an emerging component of OCD.

In the TTM group, a similar null pattern emerged in that SSRT was not significantly related to the exploratory associated features. However, non-planning impulsivity was negatively related to hair pulling severity ($r(13) = -.649$, $p = .016$), suggesting that those who are more impulsive in this domain actually have less severe TTM.

In sum, the variables chosen as exploratory measures due to their potential relatedness to OCRDs were largely unrelated to SSRT. Additionally, these factors were unrelated to core OCRD symptomology of OCD and TTM except in one domain which was non-planning impulsivity, and only for those with TTM. These finding suggest that SST and the indices from the BIS-11, OC-TCDQ and SDS are measurements of unrelated symptom factors, and their relationships do not differ as a product of specific OCRD diagnosis.

Results from regression demonstrated that SSRT was most related to compulsional aspects of OCD. Thus, we were interested to see which specific features of compulsions and compulsive behavior in OCD and TTM were contributing to SSRT. Thus, we entered specific items from the YBOCS and MGH (depending on diagnostic group) into a bivariate correlation with SST variables to see how SSRT was related to specific features of each disorder. Results are presented in Tables 10 and 11.

In the OCD group, interference of compulsions (YBOCS item #7) and distress from compulsion prevention (YBOCS item #8) were both significantly related to SSRT. In the TTM group, however, no individual items were associated with SSRT. Rather, the significant relations

among individual items in those with TTM were from MGH items and extraneous SST variables such as reaction times. The lack of significant relation between SSRT and individual hair pulling symptoms may help to explain null findings earlier in our regression analysis.

Discussion

Cognitive faculties among OCRDs have become the focus of recent research. Particularly, RI has emerged as a potentially useful marker in capturing symptomology unique to the OCRD spectrum. A growing line of research goes so far as to suggest that RI and the notion of impaired inhibitory control may be useful as a putative endophenotype of OCRDs (Chamberlain & Menzies, 2009; Robbins et al., 2012).

However, despite the growing interest in RI among OCRDs there a number of points that require critical clarification. Most importantly, the influence of comorbid psychopathology, notably of depression, needs to be accounted for in explaining RI performance. Additionally, similarities and differences in RI performance across different OCRDs need to be established and replicated. Lastly, the current state of the literature does not suggest how RI is related to other symptom domains that may be relevant for OCRDs such as impulsivity, incompleteness and life disability.

To explore these questions, we utilized a community sample of individuals with OCD and TTM. Individuals were assessed as part of an eligibility assessment for a clinical trial testing RI training. Participants were assessed with a battery for diagnostic status, symptom severity, RI performance, as well as depression, anxiety, impulsivity, incompleteness and life disability. Data were subjected to a series of hierarchical multiple regression analyses and bivariate correlations.

Our primary hypothesis that OCRD severity, understood as symptom severity across multiple diagnoses on the obsessive-compulsive spectrum, would predict RI performance above

and beyond depression was not supported for the overall OCRD symptoms included in this study; even before controlling for the influence of depression, combined symptoms of OCD and TTM were not predictive of RI.

However, we found a significant group-by-symptom severity interaction in predicting the SSRT index. Particularly, in the OCD group, compulsion severity was a significant predictor of inhibitory control whereas obsessions and total OCD severity were not. This finding was robust even when controlling for depression and depression related covariates of stress and anxiety. Meanwhile in the trichotillomania group, there was no relationship between RI and pulling symptoms. In mixed sample of those with both OCD and hair pulling, overall symptom scores were not predictive of inhibitory control. Though the SSRT values we found were comparable to other findings in previous studies (Chamberlain et al., 2006: OCD SSRT = 211.6, TTM SSRT = 264.9), we did not find that those with TTM had longer SSRTs than those with OCD. Additionally, we were unable to replicate the findings that those with worse hair-pulling severity had more difficulty in RI, and we found that SSRT was related to compulsion severity, which it was not in this previous study (Chamberlain et al., 2006). The SSRT values we found, compared to those of the healthy controls reported in Chamberlain et al. (2006) [SSRT = 167.8], were substantially elevated, however; comparison of these data reinforce the notion that SSRT in OCRDS (both in OCD and TTM) are elevated compared to healthy populations.

Thus, our primary hypothesis was supported only for a subgroup of individuals with OCD, and not for those with symptoms of TTM. The relationship we found suggests that those with greater compulsional severity have worse RI performance, and meanwhile worse obsessional severity has little influence in RI performance. These data relate to earlier meta-analytic suggest that deficiencies in RI may themselves pose as a vulnerability for compulsional

severity, but not for obsessional pathology in the context of OCD symptoms (Leopold & Backenstrass, 2015). Moreover, our data corroborate these meta-analytic findings that RI may have particular utility in exploring the hallmark compulsions of OCD. Phenomenologically, the linkage between compulsions and RI as defined by SST performance are meaningful; the SST is a measurement of motor inhibition, or, stopping actions that are no longer necessary (Verbruggen & Logan, 2009).

The relationship between RI capabilities and compulsion severity was found when factoring out the influence of depression and its covariates, suggesting that depression actually has very little influence in generating RI deficits. There are theoretical reasons to suspect that depression may be an insidious comorbidity in accounting for cognitive performance across disorders; facets of impaired inhibitory control (Lau et al., 2007), slow reaction time on cognitive tasks generally (Schlosser et al., 2013) and strong underlying anhedonia (Dillon et al., 2015) can all theoretically contribute to what looks like difficulty in stopping an inappropriate response. Despite these suggestions, however, the relationship between depressive symptoms and RI were not robust especially when compared to the influence of compulsions on explaining RI.

Our data suggest that depression was negatively correlated with the SSRT (-.30 for the OCD and -.67 for the Trich group). Depressive symptoms showed non-significant but numerically positive correlation coefficients with goRT and SSD. This suggests that the level of depression may be linked to slowed reaction time, and increased success in stopping, which may have resulted in the negative association between depression and SSRT. Thus, in the current sample, the comorbid depressive symptoms may have attenuated the manifestation of RI deficits. From this consideration, the finding that compulsional severity is a significant predictor of SSRT even after controlling for depressive symptoms is quite robust.

We also explored the relationship between OCRD symptoms, RI and a number of symptom clusters that are secondarily related to OCRDs such as impulsivity, harm avoidance, incompleteness, and overall life disability as a result of psychiatric illness. Across the whole sample, and by diagnostic group, SSRT was not related to any of these associative measures. In other words, factors of impulsivity, harm avoidance, incompleteness or life distress are not reflected in SSRT in either OCD or TTM. Additionally, these variables were largely unassociated with core OC-symptoms of obsessions, compulsions and hair pulling. An exception was found in a significant relationship between non-planning impulsivity and hair pulling severity.

Findings from these secondary analyses suggest that RI is a cognitive process that is distinguished from what is measured on other scales of impulsivity, and that RI capabilities do not necessarily proscribe information about life distress or associated OC-factors of incompleteness or harm avoidance. More specifically, wheras impulsivity is typically understood a broad, higher order and affectively laden construct, RI, as defined and measured by the SST paradigm, may be conceptualized more precisely as a cognitive process, removed from more affective contexts, involved in initiation and execution of motor responses. One additional consideration is that a lack of association between RI and additional measures may have come from a difference in mode of assessment (i.e., cognitive vs. self-report measures); considering the potential influence of measurement type, the link between SSRT and compulsion severity may be quite meaningful.

So, the broad pattern of results suggest that SSRT is differentially important in capturing OC-symptoms in that disorder-specific compulsive behavior may be more important in

explaining RI. In other words, the pattern of OCRD symptom-RI performance association was observed only for OCD but not for TTM, and also not for combined OCD-TTM.

There are a number of possibilities that may explain the discrepant association between conditions. First, the difference may stem from the nature of inhibition varying between OCD and TTM. Differential clinical phenomenology between hair pulling and compulsions may be reflected in their respective patterns of RI performance. The full picture of OCD seems to involve impaired inhibition at two levels: first, the intrusion of unwanted or irrelevant cognitions (i.e., impaired cognitive inhibition) and second the repetition of dysfunctional rituals (i.e., impaired motor inhibition) which come in direct response to intrusive thoughts (Bohne et al., 2008). Conversely, cognitive inhibition is largely secondary to the hallmark motor inhibition failure of TTM (Bohne et al., 2005). Thus, functionally the two disorders have different styles of compulsions: while those with OCD may attempt to reduce distress from intrusive thoughts, leading to repetitive and largely non-functional stereotypy, such distressing emotional contexts are not as prominent in those with TTM. Though cognitive inhibition deficit was not measured in our study, it is conceivable that motor inhibition in OCD (vs. TTM) may encompass differential factors (e.g., presence of distressing emotion, and accompanying difficulty in cognitive inhibition), which warrants systematic investigation in the future.

Second, it may be the case that various types of RI are differentially important in explaining the symptoms of OCD and TTM. More precisely, inhibitory control and RI, as discussed in the introduction, is a broad term and has distinctive subcomponents both behaviorally and neurally. The SST is thought to primarily tap into one subset of RI called action cancellation, which is the stopping of a motor response already in progress (Eagle et al., 2008). The other two forms of RI, action withholding and interference control, may be more or less

useful in capturing the diverse nature of OCRD symptoms (Friedman & Miyake, 2004). This possibility can and most likely will be addressed empirically through systematic investigation of RI patterns across different OCRD conditions utilizing different measures. As our data stand, however, we cannot resolve the inconsistency between the diagnostic groups but merely point to suggestions.

Third, it may be that within the SSRT, there are subcomponents that are in fact useful in measuring symptoms across OCD and TTM. The horse-race model of understanding RI and the quantitative measurement of this model, SSRT, will be refined and revised to enable new ways of extracting SST data. Thus, while the concept behind the SST may be valid for OCD and TTM in that both disorders need to inhibit prepotent motor responses, the SSRT may not be the only metric within the task that can capture dysfunction in both disorders. Particularly, new methods of analysis that take into account error monitoring and post-response slowing may be fruitful in uncovering similarities and differences in OCRDs (Li et al., 2008). For instance, models of performance monitoring and decisional making can help to inform why compulsive behavior is continued despite significant interference and distress (Montague et al., 2012).

Lastly, in terms of measurement, perhaps our main methods of measuring overt OC-symptoms (i.e., self-report YBOCS and MGH) did not capture a range of symptoms wide enough with which SSRT could correlate. Although the self-report YBOCS is considered to be quite reliable, participants do tend to under-report symptoms (Federici et al., 2010) and this may also be the case for the MGH. For instance, there are important features of trichotillomania that may not be thoroughly assessed on the MGH such as success in resisting pulling urges, or ability to stop pulling when having notices a pulling episode.

Despite the lack of uniformity of findings for conditions along the OCRD spectrum, these findings may in fact have important clinical implications. If there truly is a differential pattern of OC-symptom to RI performance relationship, then the depending on the specific type of OCRD a client presents with, there may be more/less important aspects of inhibitory control that are driving symptoms. For instance, we failed to find robust relationships between the obsessional content of OCD and any features of RI; thus, clients with OCD whose distress comes mostly from obsessional intrusions may see less benefit from treatment programs centered around inhibitory control. Likewise, it may be that action withholding is more important than action cancellation in TTM; Bohne et al (2008), for instance, found motor inhibition deficits in TTM on the GNG task, which measures action withholding, but not for OCD. Clinically, this proscribes treatment for TTM in line with habit reversal therapy, which is targeted to establish incompatible behaviors that can prohibit the initiation of maladaptive motor responses (Woods et al., 2006). Further investigation, however, is needed to uncover the specificity of different RI mechanisms to specific OCRDs.

This study is not without its limitations. Most notably, our sample size for those with TTM, and for those with both OCD and TTM are quite low. While some relationships showed a trending pattern of significance, more broad statements about the relationships between variables should be made with a sample with higher power. Additionally, our predominantly white sample may preclude the generalizability of our results. Despite the low sample used here, however, our participants were from the community and were thoroughly assessed with DSM-5 criteria from the ADIS-5; thus, results may be more relevant than having used an analogue sample.

Second, this study may suffer from a small measurement battery which lacks any clinician-rated measurement. The core battery used to assess our primary hypothesis had one

cognitive measure, one measure of OCD and one measure of hair pulling severity. Thus, any features of RI or OCRD symptoms not touched upon in this brief battery would simply be absent in analysis. Future research will undoubtedly explore the cognitive profile of OCRDs using larger and more fully built assessment batteries.

Despite the limitations, these findings are important in several ways. First, our RI data were in a comparable range to other studies that have used the SST in OCD and TTM (Chamberlain et al, 2006). This goes to show that SSRT can indeed be measured reliably across multiple clinical samples. Second, these data do inform the specificity and value of SSRT on describing OCRDs. Namely, the data show that amongst those with OCD, RI is indeed more attributable to OCD symptoms than to comorbid depression, and these findings may help to inform future evidence-based treatments. Additionally, the lack of correlation between SSRT and three subscales of impulsivity help refine our understanding of a distinction between inhibitory control as measured by the SST and broader impulsive behavior. These data can inform future avenues of research on what symptoms may lend themselves to cognitive assessment, and what cognitive batteries may be beneficial in exploring OCRDs.

References

Abramovitch, A., Shaham, N., Levin, L., Bar-Hen, M., & Schweiger, A. (2015). Response inhibition in a subclinical obsessive-compulsive sample. *Journal of Behavior Therapy and Experimental Psychiatry, 46*, 66-71.

Abramowitz, J. S., & Jacoby, R. J. (2015). Obsessive-compulsive and related disorders: A critical review of the new diagnostic class. *Annual Review of Clinical Psychology*, *11*, 165-186.

Albert, J., Lopez-Martin, S., & Carretie, L. (2010). Emotional context modulates response inhibition: Neural and behavioral data. *Neuroimage, 49*(1), 914-921.

Alderson, R. M., Rapport, M. D., & Kofler, M. J. (2007). Attention-deficit/hyperactivity disorder and behavioral inhibition: A meta-analytic review of the stop-signal paradigm. *Journal of Abnormal Child Psychology, 35*(5), 745-758.

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

Aron, A. R., & Poldrack, R. A. (2005). The cognitive neuroscience of response inhibition: Relevance for genetic research in attention-deficit/hyperactivity disorder. *Biological Psychiatry, 57*(11), 1285-1292.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014). Inhibition and the right inferior frontal cortex: One decade on. *Trends in Cognitive Sciences, 18*(4), 177-185.

Austin, M. P., Mitchell, P., & Goodwin, G. M. (2001). Cognitive deficits in depression: Possible implications for functional neuropathology. *The British Journal of Psychiatry : The Journal of Mental Science, 178*, 200-206.

Aycicegi, A., Dinn, W. M., Harris, C. L., & Erkmen, H. (2003). Neuropsychological function in obsessive-compulsive disorder: Effects of comorbid conditions on task performance. *European Psychiatry : The Journal of the Association of European Psychiatrists, 18*(5), 241-248.

Bannon, S., Gonsalvez, C. J., & Croft, R. J. (2008). Processing impairments in OCD: It is more than inhibition! *Behaviour Research and Therapy, 46*(6), 689-700.

Bari, A., & Robbins, T. W. (2013). Inhibition and impulsivity: Behavioral and neural basis of response control. *Progress in Neurobiology, 108*, 44-79.

Beck AT, Steer RA and Brown GK (1996) "Manual for the Beck Depression Inventory-II". San Antonio, TX: Psychological Corporation

Berlin, G. S., & Hollander, E. (2014). Compulsivity, impulsivity, and the DSM-5 process. *CNS Spectrums, 19*(1), 62-68.

Bienvenu, O. J., Samuels, J. F., Riddle, M. A., Hoehn-Saric, R., Liang, K. Y., Cullen, B. A., et al. (2000). The relationship of obsessive-compulsive disorder to possible spectrum disorders: Results from a family study. *Biological Psychiatry, 48*(4), 287-293.

Blom, R. M., Samuels, J. F., Grados, M. A., Chen, Y., Bienvenu, O. J., Riddle, M. A., et al. (2011). Cognitive functioning in compulsive hoarding. *Journal of Anxiety Disorders, 25*(8), 1139-1144.

Bohne, A., Keuthen, N. J., Tuschen-Caffier, B., & Wilhelm, S. (2005). Cognitive inhibition in trichotillomania and obsessive–compulsive disorder. *Behaviour Research and Therapy*, 43(7), 923-942.

Bohne, A., Savage, C. R., Deckersbach, T., Keuthen, N. J., & Wilhelm, S. (2008). Motor inhibition in trichotillomania and obsessive-compulsive disorder. *Journal of Psychiatric Research, 42*(2), 141-150.

Brown, T. A., & Barlow, D. H. (2013). Anxiety and Related Disorders Interview Schedule for DSM-5, Adult and Lifetime Version: Clinician Manual. Oxford University Press.

Browne, H. A., Gair, S. L., Scharf, J. M., & Grice, D. E. (2014). Genetics of obsessive-compulsive disorder and related disorders. *The Psychiatric Clinics of North America, 37*(3), 319-335.

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive compulsive disorder: The importance of failures in

cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience and Biobehavioral Reviews, 29*(3), 399-419.

Chamberlain. S. R., Finerberg, N.A., Blackwell, A.D., Robbins, T.W., Sahakian, B.J. (2006). Motor inhibition and cognitive flexibility in obsessive-compulsive disorder and trichotillomania. *Am J Psychiatry, 163*, 1282-1284.

Chamberlain, S. R., Fineberg, N. A., Menzies, L. A., Blackwell, A. D., Bullmore, E. T., Robbins, T. W., et al. (2007a). Impaired cognitive flexibility and motor inhibition in unaffected first-degree relatives of patients with obsessive-compulsive disorder. *The American Journal of Psychiatry*, *164*(2), 335-338.

Chamberlain SR, Menzies L. (2009). Endophenotypes of obsessive-compulsive disorder: rationale, evidence and future potential. *Expert Rev Neurother*. 9:1133–1146.

Chamberlain, S. R., Menzies, L., Sahakian, B. J., & Fineberg, N. A. (2007b). Lifting the veil on trichotillomania. *The American Journal of Psychiatry, 164*(4), 568-574.

Coles, M. E., Heimberg, R. G., Frost, R. O., & Steketee, G. (2005). Not just right experiences and obsessive-compulsive features: Experimental and self-monitoring perspectives. *Behaviour Research and Therapy, 43*(2), 153-167.

Congdon, E., Mumford, J. A., Cohen, J. R., Galvan, A., Canli, T., & Poldrack, R. A. (2012). Measurement and reliability of response inhibition. *Frontiers in Psychology, 3*, 37.

Cuthbert, B. N. (2014). The RDoC framework: Facilitating transition from ICD/DSM to dimensional approaches that integrate neuroscience and psychopathology. *World Psychiatry : Official Journal of the World Psychiatric Association (WPA), 13*(1), 28-35.

Dalley, J. W., Everitt, B. J., & Robbins, T. W. (2011). Impulsivity, compulsivity, and top-down cognitive control. *Neuron, 69*(4), 680-694.

de Bruijn, C., Beun, S., de Graaf, R., ten Have, M., & Denys, D. (2010). Subthreshold symptoms and obsessive-compulsive disorder: Evaluating the diagnostic threshold. *Psychological Medicine, 40*(6), 989-997.

De Houwer, J., & Tibboel, H. (2010). Stop what you are not doing! emotional pictures interfere with the task not to respond. *Psychonomic Bulletin & Review, 17*(5), 699-703.

Denys, D., Tenney, N., van Megen, H. J., de Geus, F., & Westenberg, H. G. (2004). Axis I and II comorbidity in a large sample of patients with obsessive-compulsive disorder. *Journal of Affective Disorders, 80*(2-3), 155-162.

Diamond, A. (2013). Executive functions. *Annual Review of Psychology, 64*, 135-168.

Dillon, D. G., Wiecki, T., Pechtel, P., Webb, C., Goer, F., Murray, L., et al. (2015). A computational analysis of flanker interference in depression. *Psychological Medicine,* , 1-12.

Eagle, D. M., Bari, A., & Robbins, T. W. (2008). The neuropsychopharmacology of action inhibition: Cross-species translation of the stop-signal and go/no-go tasks. *Psychopharmacology, 199*(3), 439-456.

Eisen, J. L., Sibrava, N. J., Boisseau, C. L., Mancebo, M. C., Stout, R. L., Pinto, A., et al. (2013). Five-year course of obsessive-compulsive disorder: Predictors of remission and relapse. *The Journal of Clinical Psychiatry, 74*(3), 233-239.

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

Fergus, T. A. (2014). Are "not just right experiences" (NJREs) specific to obsessive-compulsive symptoms?: Evidence that NJREs span across symptoms of emotional disorders. *Journal of Clinical Psychology, 70*(4), 353-363.

Franklin, M. E., Zagrabbe, K., & Benavides, K. L. (2011). Trichotillomania and its treatment: A review and recommendations. *Expert Review of Neurotherapeutics, 11*(8), 1165-1174.

Frare, F., Perugi, G., Ruffolo, G., & Toni, C. (2004). Obsessive-compulsive disorder and body dysmorphic disorder: A comparison of clinical features. *European Psychiatry : The Journal of the Association of European Psychiatrists, 19*(5), 292-298.

Friedman, N. P., & Miyake, A. (2004). The relations among inhibition and interference control functions: A latent-variable analysis. *Journal of Experimental Psychology.General, 133*(1), 101-135.

Grant, J. E., & Kim, S. W. (2014). Brain circuitry of compulsivity and impulsivity. *CNS Spectrums, 19*(1), 21-27.

Grant, J. E., Odlaug, B. L., & Chamberlain, S. R. (2011). A cognitive comparison of pathological skin picking and trichotillomania. *Journal of Psychiatric Research, 45*(12), 1634-1638.

Harvey, P. D. (2007). Cognitive impairments in major depression and bipolar disorders. *Psychiatry (Edgmont (Pa.: Township)), 4*(1), 12-14.

Huppert, J. D., Simpson, H. B., Nissenson, K. J., Liebowitz, M. R., & Foa, E. B. (2009). Quality of life and functional impairment in obsessive-compulsive disorder: A comparison of patients with and without comorbidity, patients in remission, and healthy controls. *Depression and Anxiety, 26*(1), 39-45.

Jakubczyk, A., Klimkiewicz, A., Topolewska-Wochowska, A., Serafin, P., Sadowska-Mazuryk, J., Pupek-Pyziol, J., et al. (2012). Relationships of impulsiveness and depressive symptoms in alcohol dependence. *Journal of Affective Disorders, 136*(3), 841-847.

Jones, G. C., Coutinho, K., Anjaria, D., Hussain, N., & Dholakia, R. (2010). Treatment of recurrent Rapunzel syndrome and trichotillomania: case report and literature review. *Psychosomatics*, *51*(5), 443-446.

Khng, K. H., & Lee, K. (2014). The relationship between stroop and stop-signal measures of inhibition in adolescents: Influences from variations in context and measure estimation. *PloS One, 9*(7), e101356.

Keuthen,N.J., Flessner,C.A., Woods,D.W., Franklin,M.E., Stein,D.J., Cashin,S.E., Trichotillomania Learning Center Scientific Advisory Board (2007). Factor analysis of the Massachusetts General Hospital Hairpulling Scale. *J Psychosom Res*, 62(6)707-709.

Koch, J., & Exner, C. (2015). Selective attention deficits in obsessive-compulsive disorder: The role of metacognitive processes. *Psychiatry Research, 225*(3), 550-555.

Lau, M. A., Christensen, B. K., Hawley, L. L., Gemar, M. S., & Segal, Z. V. (2007). Inhibitory deficits for negative information in persons with major depressive disorder. *Psychological Medicine, 37*(9), 1249-1259.

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

Li, C. S. R., Huang, C., Yan, P., Paliwal, P., Constable, R. T., & Sinha, R. (2008). Neural correlates of post-error slowing during a stop signal task: a functional magnetic resonance imaging study. *Journal of Cognitive Neuroscience, 20*(6), 1021-1029.

Lipszyc, J., & Schachar, R. (2010). Inhibitory control and psychopathology: A meta-analysis of studies using the stop signal task. *Journal of the International Neuropsychological Society : JINS, 16*(6), 1064-1076.

Lochner, C., Seedat, S., du Toit, P. L., Nel, D. G., Niehaus, D. J., Sandler, R., et al. (2005). Obsessive-compulsive disorder and trichotillomania: A phenomenological comparison. *BMC Psychiatry, 5*, 2.

Logan, G. D. (1994). On the ability to inhibit thought and action: A user's guide to the stop signal paradigm. In D. Dagenbach & T. H. Carr, Inhibitory processes in attention, memory, and language (pp. 189-239). San Diego: Academic Press

Logan, G. D., Van Zandt, T., Verbruggen, F., & Wagenmakers, E. J. (2014). On the ability to inhibit thought and action: General and special theories of an act of control. *Psychological Review, 121*(1), 66-95.

Lopez, A. D., & Murray, C. C. (1998). The global burden of disease, 1990-2020. *Nature Medicine, 4*(11), 1241-1243.

Lovibond, S.H.; Lovibond, P.F. (1995). "Manual for the Depression Anxiety Stress Scales" (2nd ed.). Sydney: Psychology Foundation.

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

Matsunaga, H. (2013). Clinical features, treatments and outcome of obsessive-compulsive disorder (OCD) focusing on the assessment and characteristics of patients with treatment-refractory OCD. *Seishin Shinkeigaku Zasshi = Psychiatria Et Neurologia Japonica, 115*(9), 967-974.

McIntyre, R. S., Cha, D. S., Soczynska, J. K., Woldeyohannes, H. O., Gallaugher, L. A., Kudlow, P., et al. (2013). Cognitive deficits and functional outcomes in major depressive disorder: Determinants, substrates, and treatment interventions. *Depression and Anxiety, 30*(6), 515-527.

Montague, P. R., Dolan, R. J., Friston, K. J., & Dayan, P. (2012). Computational psychiatry. *Trends in Cognitive Sciences, 16*(1), 72-80.

Monzani, B., Rijsdijk, F., Harris, J., & Mataix-Cols, D. (2014). The structure of genetic and environmental risk factors for dimensional representations of DSM-5 obsessive-compulsive spectrum disorders. *JAMA Psychiatry, 71*(2), 182-189.

Morein-Zamir, S., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2010). Inhibition of thoughts and actions in obsessive-compulsive disorder: Extending the endophenotype? *Psychological Medicine, 40*(2), 263-272.

Morein-Zamir, S., Papmeyer, M., Pertusa, A., Chamberlain, S. R., Fineberg, N. A., Sahakian, B. J., et al. (2014). The profile of executive function in OCD hoarders and hoarding disorder. *Psychiatry Research, 215*(3), 659-667.

National Institutes of Mental Health. (n.d.). NIMH Research Domain Criteria (RDoC). Retrieved April 2nd, 2015 from http://www.nimh.nih.gov/research-priorities/rdoc/nimh-research-domain-criteria-rdoc.shtml

Odlaug, B. L., Chamberlain, S. R., & Grant, J. E. (2010). Motor inhibition and cognitive flexibility in pathologic skin picking. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34*(1), 208-211.

Pallanti, S., Grassi, G., Sarrecchia, E. D., Cantisani, A., & Pellegrini, M. (2011). Obsessive-compulsive disorder comorbidity: Clinical assessment and therapeutic implications. *Frontiers in Psychiatry, 2*, 70.

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the barratt impulsiveness scale. *Journal of Clinical Psychology, 51*(6), 768-774.

Penades, R., Catalan, R., Rubia, K., Andres, S., Salamero, M., & Gasto, C. (2007). Impaired response inhibition in obsessive compulsive disorder. *European Psychiatry : The Journal of the Association of European Psychiatrists, 22*(6), 404-410.

Phillips, K. A., Stein, D. J., Rauch, S. L., Hollander, E., Fallon, B. A., Barsky, A., et al. (2010). Should an obsessive-compulsive spectrum grouping of disorders be included in DSM-V? *Depression and Anxiety, 27*(6), 528-555.

Rajender, G., Bhatia, M. S., Kanwal, K., Malhotra, S., Singh, T. B., & Chaudhary, D. (2011). Study of neurocognitive endophenotypes in drug-naive obsessive-compulsive disorder patients, their first-degree relatives and healthy controls. *Acta Psychiatrica Scandinavica, 124*(2), 152-161.

Robbins, T. W., Gillan, C. M., Smith, D. G., de Wit, S., & Ersche, K. D. (2012). Neurocognitive endophenotypes of impulsivity and compulsivity: Towards dimensional psychiatry. *Trends in Cognitive Sciences, 16*(1), 81-91.

Roebers, C. M., Schmid, C., & Roderer, T. (2010). Encoding strategies in primary school children: Insights from an eye-tracking approach and the role of individual differences in attentional control. *The Journal of Genetic Psychology, 171*(1), 1-21.

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the national comorbidity survey replication. *Molecular Psychiatry, 15*(1), 53-63.

Schachar, R., Logan, G. D., Robaey, P., Chen, S., Ickowicz, A., & Barr, C. (2007). Restraint and cancellation: Multiple inhibition deficits in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 35*(2), 229-238.

Schlosser, N., Wolf, O. T., Fernando, S. C., Terfehr, K., Otte, C., Spitzer, C., et al. (2013). Effects of acute cortisol administration on response inhibition in patients with major depression and healthy controls. *Psychiatry Research, 209*(3), 439-446.

Schulz, K. P., Fan, J., Magidina, O., Marks, D. J., Hahn, B., & Halperin, J. M. (2007). Does the emotional go/no-go task really measure behavioral inhibition? convergence with measures on a non-emotional analog. *Archives of Clinical Neuropsychology : The Official Journal of the National Academy of Neuropsychologists, 22*(2), 151-160.

Sica, C., Bottesi, G., Orsucci, A., Pieraccioli, C., Sighinolfi, C., & Ghisi, M. (2015). "Not just right experiences" are specific to obsessive-compulsive disorder: Further evidence from italian clinical samples. *Journal of Anxiety Disorders, 31*, 73-83.

Sheehan DV, Harnett-Sheehan K, Raj BA: The measurement of disability. Int Clin Psychopharmacol 11(suppl 3):89-95, 1996.

Sjoerds, Z., van den Brink, W., Beekman, A. T., Penninx, B. W., & Veltman, D. J. (2014). Response inhibition in alcohol-dependent patients and patients with depression/anxiety: A functional magnetic resonance imaging study. *Psychological Medicine, 44*(8), 1713-1725.

Stein, D. J., Grant, J. E., Franklin, M. E., Keuthen, N., Lochner, C., Singer, H. S., et al. (2010). Trichotillomania (hair pulling disorder), skin picking disorder, and stereotypic movement disorder: Toward DSM-V. *Depression and Anxiety, 27*(6), 611-626.

Stengler, K., Olbrich, S., Heider, D., Dietrich, S., Riedel-Heller, S., & Jahn, I. (2013). Mental health treatment seeking among patients with OCD: Impact of age of onset. *Social Psychiatry and Psychiatric Epidemiology, 48*(5), 813-819.

Stern, E. R., & Taylor, S. F. (2014). Cognitive neuroscience of obsessive-compulsive disorder. *The Psychiatric Clinics of North America, 37*(3), 337-352.

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

Summerfeldt, L. J., Kloosterman, P., Parker, J. D. A., Antony, M. M., & Swinson, R. P. (2001). Assessing and validating the obsessive-compulsive-related construct of incompleteness. Poster presented at the 62nd annual convention of the Canadian Psychological Association, Ste-Foy, Quebec.

Taylor, S., McKay, D., Crowe, K. B., Abramowitz, J. S., Conelea, C. A., Calamari, J. E., et al. (2014). The sense of incompleteness as a motivator of obsessive-compulsive symptoms: An empirical analysis of concepts and correlates. *Behavior Therapy*, *45*(2), 254-262.

Thomas, S. J., Gonsalvez, C. J., & Johnstone, S. J. (2014). How specific are inhibitory deficits to obsessive-compulsive disorder? A neurophysiological comparison with panic disorder. *Clinical Neurophysiology : Official Journal of the International Federation of Clinical Neurophysiology*, *125*(3), 463-475.

Tolin, D. F., Witt, S. T., & Stevens, M. C. (2014). Hoarding disorder and obsessive-compulsive disorder show different patterns of neural activity during response inhibition. *Psychiatry Research, 221*(2), 142-148.

Torp, N. C., Dahl, K., Skarphedinsson, G., Compton, S., Thomsen, P. H., Weidle, B., et al. (2015). Predictors associated with improved cognitive-behavioral therapy outcome in pediatric obsessive-compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 54*(3), 200-207.e1.

van Velzen, L. S., Vriend, C., de Wit, S. J., & van den Heuvel, O. A. (2014). Response inhibition and interference control in obsessive-compulsive spectrum disorders. *Frontiers in Human Neuroscience, 8*, 419.

Verbruggen, F., & De Houwer, J. (2007). Do emotional stimuli interfere with response inhibition? Evidence from the stop signal paradigm. Cognition and Emotion, 21(2), 391-403.

Verbruggen, F., Liefooghe, B., & Vandierendonck, A. (2004). The interaction between stop signal inhibition and distractor interference in the flanker and stroop task. *Acta Psychologica, 116*(1), 21-37.

Verbruggen, F., & Logan, G. D. (2009). Models of response inhibition in the stop-signal and stop-change paradigms. *Neuroscience and Behavioral Reviews, 33*, 647-661.

Verbruggen, F., Logan, G. D., & Stevens, M. A. (2008b). STOP-IT: Windows executable software for the stop-signal paradigm. Behavior Research Methods, 40(2), 479-483.

Vergara-Lopez, C., Lopez-Vergara, H. I., & Colder, C. R. (2013). Executive functioning moderates the relationship between motivation and adolescent depressive symptoms. *Personality and Individual Differences, 54*(1), 18-22.

Wechsler, D., & Hsiao-pin, C. (2011). WASI-II: Wechsler abbreviated scale of intelligence. Pearson.

Woods, D. W., Wetterneck, C. T., & Flessner, C. A. (2006). A controlled evaluation of acceptance and commitment therapy plus habit reversal for trichotillomania. *Behaviour research and therapy*, 44(5), 639-656.

Wootton, B.M., Diefenbach, G.J., Bragdon, L.B., Steketee, G., Frost, R.O., Tolin, D.F. (2015). A contemporary psychometric evaluation of the obsessive compulsive inventory-revised (OCI-R). *Psychol Assess*, Epub ahead of print.

Wright, L., Lipszyc, J., Dupuis, A., Thayapararajah, S. W., & Schachar, R. (2014). Response inhibition and psychopathology: A meta-analysis of go/no-go task performance. *Journal of Abnormal Psychology, 123*(2), 429-439.

Yap, K., Mogan, C., & Kyrios, M. (2012). Obsessive-compulsive disorder and comorbid depression: The role of OCD-related and non-specific factors. *Journal of Anxiety Disorders, 26*(5), 565-573.

Appendix A – Tables

Table 1.

Demographic Characteristics

	Whole Sample (n = 49)	OCD group (n = 27)	TTM group (n = 13)	Mixed Diagnosis (n = 9)	*F or* χ^2
Age [M(SD)]	29.59(10.8)	28.04 (10.74)	31.77 (12.262)	31.11 (9.02)	ns
Sex					
Male	32.7% (n = 16)	33.3% (n = 9)	15.4% (n = 2)	55.6% (n = 5)	ns
Female	67.3% (n = 33)	66.7% (n = 18)	84.6% (n = 11)	44.4% (n = 4)	ns
Race					
Caucasian	81.6% (n = 40)	84.6% (n =22)	84.6% (n = 11)	77.8% (n = 7)	ns
African American	10.2% (n = 5)	7.7% (n = 2)	15.4% (n = 2)	11.1% (n = 1)	ns
Two or more races	6.1% (n = 3)	7.7% (n =2)	0% (n = 0)	11.1% (n = 1)	ns
Hispanic/Latino	2.0% (n = 1)	3.7% (n = 1)	n/a	n/a	ns
WASI-2	105.04(10.45)	103.52(10.66)	107.66(9.25)	105.87(11.90)	ns
Comorbidity					
PD	6.1% (n = 3)	7.4% (n = 2)	0% (n = 0)	11.1% (n = 1)	ns
Agoraphobia	8.2% (n = 4)	7.4% (n = 2)	15.4% (n = 2)	0% (n = 100)	ns
SoP	44.9% (n = 22)	51.9% (n = 14)	30.8% (n = 4)	44.4% (n = 4)	ns
SA	6.1% (n = 3)	0% (n = 0)	23.1% (n = 3)	0% (n = 0)	8.849*
GAD	57.1% (n = 28)	66.7% (n = 18)	38.5% (n = 5)	55.6% (n = 5)	ns
BDD	6.1% (n = 3)	3.7% (n = 1)	0% (n = 0)	22.2% (n = 2)	ns
Phobia	16.3% (n = 8)	7.4% (n = 2)	30.8% (n = 4)	22.2% (n = 2)	ns
PTSD	18.4% (n = 9)	3.7% (n = 1)	30.8% (n = 4)	44.4% (n = 4)	9.287**
MDD	42.9% (n = 21)	40.7% (n = 11)	38.5% (n = 5)	55.6% (n = 5)	ns
PDD	8.2% (n = 4)	11.1% (n = 3)	7.7% (n = 1)	0% (n = 0)	ns
IAD	2% (n = 1)	0% (n = 0)	0% (n = 0)	11.1% (n = 1)	ns
AUD	2% (n = 1)	0% (n = 0)	7.7% (n =1)	0% (n = 0)	ns
SUD	6.1% (n = 3)	0% (n = 0)	0% (n = 0)	33.3% (n = 3)	14.203***
ADHD	14.3% (n = 7)	11.1% (n = 3)	7.7% (n = 1)	33.3% (n = 3)	ns
BN	4.1% (n = 2)	3.7% (n = 1)	7.7% (n =1)	0% (n = 0)	ns
Other	6.1% (n =3)	7.4% (n = 2)	0% (n = 0)	11.1% (n = 1)	ns

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: FSIQ-2=Full-Scale IQ, PD=Panic Disorder, SoP=Social Phobia, SA=Separation Anxiety, GAD=Generalized Anxiety Disorder, BDD=Body Dysmorphic Disorder, PTSD=Post-Traumatic Stress Disorder, IAD=Illness Anxiety Disorder, MDD/PDD=Major/Persistent Depressive Disorder, AUD= Alcohol Use Disorder, SUD=Substance Use Disorder, ADHD=Attention Deficit Hyperactivity Disorder, BN=Bulimia Nervosa.

Note: WASI-2 Full-Scale IQ Scores are derived from Matrix Reasoning and Vocabulary subtests.

Table 2.

Measures Used in This Project

Construct	Measure	Type of Measure
Diagnostic Status	Anxiety Disorders Interview Schedule for DSM-5 Disorders (ADIS-5)	Structured interview
Illness Severity (OCD)	Yale-Brown Obsessive Compulsive Scale (Y-BOCS) Self-Report Version	Self-report (Qualtrics)
Illness Severity (TTM)	Massachusetts General Hospital Hairpulling Scale (MGH)	Self-report (Qualtrics)
Response Inhibition	Stop-Signal Task (SST)	Computerized paradigm
Depression	Beck Depression Inventory II (BDI-II)	Self-report (paper)
Depression	Depression Anxiety Stress Scale (DASS)	Self-report (Qualtrics)
Anxiety & Stress	Depression Anxiety Stress Scale (DASS)	Self-report (Qualtrics)
Impulsivity	Barratt Impulsiveness Scale (BIS-11)	Self-report (Qualtrics)
Incompleteness	Obsessive-Compulsive Trait Core Dimensions Questionnaire (OC-TCDQ)	Self-report (Qualtrics)
Life Impairment	Sheehan Disability Scale (SDS)	Self-report (Qualtrics)
Overall Intellectual Functioning	Wechsler Abbreviated Scale of Intelligence II (WASI-II)	Intelligence assessment

Table 3.

Symptom Characteristics

	OCD group (n = 27)	TTM group (n = 13)	Mixed Diagnosis (n = 9)	F	p	Post -hoc
	M(SD)	M(SD)	M(SD)			
OCRD Symptoms						
YBOCS-T	22.82(5.27)	n/a	22.00(4.66)	.170	.683	
YBOCS-O	11.12(3.58)	n/a	10.77(2.33)	.084	.774	
YBOCS-C	11.66(2.84)	n/a	11.22(2.68)	.169	.683	
MGH-T	n/a	17.07(4.42)	18.11(5.13)	.255	.619	
SST Variables						
SSRT	224.65(43.18)	221.14(40.67)	224.71(54.82)	.029	.971	
SSD	432.29(145.57)	372.28(164.13)	356.47(177.86)	1.098	.342	
goRT	658.07(130.32)	594.52(134.37)	582.57(146.43)	1.566	.220	
Symptom Variables						
DASS-D	13.18(11.47)	11.07(9.36)	12.88(12.41)	.162	.851	
DASS-A	13.92(8.06)	6.92(5.86)	11.55(10.85)	3.249	.048*	1>2[a]
DASS-S	19.92(9.99)	12.46(8.45)	20.44(10.38)	2.945	.063	
Additional Symptoms						
BIS-11 AI	25.81(6.62)	12.92(8.47)	18.33(11.29)	11.709	<.001***	1>2
BIS-11 MI	27.88(8.16)	12.61(8.75)	24.77(8.91)	12.621	<.001***	1,3>2
BIS-11 NPI	18.40(6.52)	14.46(8.23)	14.33(6.02)	2.016	.145	
OCTDCQ-HA	21.07(4.13)	16.69(3.17)	19.66(5.09)	5.015	.011*	1>2
OCTDCQ-I	18.85(4.99)	18.92(4.03)	21.77(4.63)	1.405	.256	
SDS-GI	25.29(5.63)	22.61(5.47)	25.33(5.14)	1.138	.329	

*p < 0.05; **p < 0.01; ***p < 0.001.

a 1 = OCD group, 2 = TTM group, 3 = Mixed group.

Abbreviations: YBOCS-T=Yale-Brown Obsessive-Compulsive Scale Total Scores, YBOCS-O= Yale-Brown Obsessive-Compulsive Scale Obsessional Severity, YBOCS-C=Yale-Brown Obsessive-Compulsive Scale Compulsion Severity, MGH-T=Massachusetts General Hairpulling Scale Severity, SSRT= Stop-Signal Reaction Time, SSD=Stop-Signal Delay, goRT=Mean reaction time on Go trials, DASS-D=Depression, DASS-A=Anxiety, DASS-S=Stress, BIS-11 AI= Attentional Impulsivity, BIS-11 MI= Motor Impulsivity, BIS-11 NPI=Non-Planning Impulsivity, HA=Harm Avoidance, OCTDCQ-HA=Harm Avoidance, OCTDCQ-I=Incompleteness, SDS-GI=Global Impairment.

Table 4.

Correlations among OCRD Symptoms, Depression and SST Indices in an Overall Sample

Measure	1	2	3	4	5	6	7	8
1. p.RS	–	–	–	–	–	–	–	–
2. SSD	-.625***	–	–	–	–	–	–	–
3. SSRT	.232	-.594***	–	–	–	–	–	–
4. Commission RT	-.547***	.917***	-.332*	–	–	–	–	–
5. goRT	-.644***	.965***	-.363*	.954***	–	–	–	–
6. Go-%	-.102	-.216	-.085	-.252	-.287*	–	–	–
7. Omis-%	-.018	.319*	.050	.362*	.392**	-.974**	–	–
8. OCRD Severity	-.059	.231	-.005	.250	.262	-.024	.073	–
9. DASS-D	.058	.260	-.245	.193	.221	-.223	.275	.066

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: p.RS=Probability of Commission, SSD=Stop Signal Delay, goRT=Mean Reaction Time on go Trials, Go-%=Correct go-trial percentage, Omis-%=Omission Percentage, DASS-D=Depression, OCRD Severity=Z-centered YBOCS and MGH scores, DASS-D=Depression.

Table 5.

Bivariate correlations among OCRD Symptoms, Depression and SST indices in OCD and TTM

		TTM											
	Measure	1	2	3	4	5	6	7	8	9	10	11	12
OCD	1. p.RS	–	-.749**	.525	-.618*	-.753**	.038	-.159	n/a	n/a	n/a	-.112	-.340
	2. SSD	-.479*	–	-.791**	.916***	.983***	-.394	.511	n/a	n/a	n/a	.368	.291
	3. SSRT	-.052	-.494**	–	-.569*	-.666*	.358	-.460	n/a	n/a	n/a	-.280	-.255
	4. Commission RT	-.459*	.915***	-.248	–	.945***	-.272	.384	n/a	n/a	n/a	.487	.152
	5. goRT	-.550**	.957***	-.222	.944***	–	-.385	.495	n/a	n/a	n/a	.363	.285
	6. Go-%	.193	-.574**	-.058	-.587**	-.628***	–	-.975***	n/a	n/a	n/a	-.014	-.271
	7. Omis-%	-.326	.628***	-.048	.664***	.686***	-.946***	–	n/a	n/a	n/a	.066	.260
	8. YBOCS-T	.178	-.002	.326	.041	.103	-.340	.323	–	n/a	n/a	n/a	n/a
	9. YBOCS-O	-.038	.155	.136	.057	.216	-.366	.334	.862***	–	n/a	n/a	n/a
	10. YBOCS-C	.378	-.197	.433*	.005	-.081	-.170	.179	.770***	.341	–	n/a	n/a
	11. MGH-T	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	–	.247
	12. DASS-D	.118	.251	-.299	.181	.176	-.046	.171	-.018	-.055	.036	n/a	–

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: p.RS=Probability of Commission, SSRT= Stop-Signal Reaction Time, SSD=Stop-Signal Delay, goRT=Mean reaction time on Go trials, Go-%=Correct go-trial percentage, Omis-%=Omission Percentage, YBOCS-T=Yale-Brown Obsessive-Compulsive Scale Total Scores, YBOCS-O= Yale-Brown Obsessive-Compulsive Scale Obsessional Severity, YBOCS-C=Yale-Brown Obsessive-Compulsive Scale Compulsion Severity, DASS-D=Depression, MGH-T=Massachusetts General Hairpulling Scale Severity.

Table 6.

Interaction of Diagnostic Group on the Relationship between OCRD Symptoms and SSRT

DV: SSRT	Model 1 R^2 = .117, F(4, 43) = 1.425, p = .242					Model 2 $R^2 \Delta$ = .008, F(7, 40) = .815, p = .580					Model 3 $R^2 \Delta$ = .192, F(9, 38) = 1.959, p = .072				
Variable	*B*	*SE B*	*β*	*t*	*p*	*B*	*SE B*	*β*	*t*	*p*	*B*	*SE B*	*β*	*t*	*p*
Gender	16.84	13.33	.183	1,26	.213	17.02	14.95	.185	1.13	.262	10.34	13.79	.112	.750	.458
DASS-D	-.555	.785	-.139	-.707	.483	-.461	.835	-.116	-.552	.584	.060	.780	.015	.077	.939
DASS-A	.780	1.142	.153	.683	.498	.559	1.24	.109	.448	.657	1.29	1.16	.254	1.10	.274
DASS-S	-1.29	1.10	-.300	-1.17	.247	-1.36	1.17	-.317	-1.16	.252	**-2.44**	**1.11**	**-.566**	**-2.188**	**.035***
OCD vs. TTM						-7.46	6.92	-.076	-.441	.661	-11.56	15.48	-.118	-.747	.460
OCD vs. Mixed Diagnosis						-2.84	18.16	-.026	-.157	.876	15.08	17.76	.136	.849	.401
OCRD Severity						2.816	7.08	.065	.398	.693	**17.10**	**8.06**	**.393**	**2.12**	**.041***
Interaction: OCD vs. TTM											-29.72	14.88	-.328	-1.99	.053
Interaction: OCD vs. Mixed											**-52.47**	**17.34**	**-.515**	**-3.025**	**.004****

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: DASS-D=Depression, DASS-A=Anxiety, DASS-S=Stress, OCD=Obsessive Compulsive Disorder, TTM=Trichotillomania, Mixed Diagnosis=OCD + TTM Diagnoses, OCRD Severity=z-centered YBOCS and MGH scores.

Table 7.

Hierarchical Regression used to predict SSRT scores using OCRD Severity

	DV: SSRT	Model 1					Model 2				
	Variable	*B*	*SE B*	*β*	*t*	*p*	*B*	*SE B*	*β*	*t*	*p*
		R^2 =.117, F(4, 43) = 1.425, p = .242					$R^2 \Delta$ =.003, F(5, 42) = 1.151, p = .349				
Whole Sample	Gender	16.848	13.334	.183	1.264	.213	18.133	13.832	.197	1.311	.197
	DASS-D	-.555	.785	-.139	-.707	.483	-.537	.794	-.135	-.676	.503
	DASS-A	.780	1.142	.153	.683	.498	.678	1.180	.133	.575	.569
	DASS-S	-1.295	1.103	-.300	-1.175	.247	-1.291	1.114	-.299	-1.159	.253
	OCRD Severity						2.739	6.750	.063	.406	.687
		R^2 =.185, F(4, 21) = 1.191, p = .344					$R^2 \Delta$ =.112, F(5, 20) = 1.686, p = .184				
OCD	Gender	3.312	17.991	.037	.184	.856	9.040	17.426	.102	.519	.610
	DASS-D	-.860	1.062	-.231	-.810	.427	-.378	1.046	-.102	-.361	.722
	DASS-A	2.663	1.714	.507	1.554	.135	2.355	1.640	.448	1.436	.166
	DASS-S	-2.009	1.654	-.473	-1.215	.238	-2.527	1.601	-.594	-1.578	.130
	YBOCS-T						2.938	1.649	.365	1.782	.090
		R^2 =.185, F(4, 21) = 1.191, p = .344					$R^2 \Delta$ =.207, F(6, 19) = 2.043, p = .109				
OCD	Gender	3.312	17.991	.037	.184	.856	7.874	16.633	.088	.473	.641
	DASS-D	-.860	1.062	-.231	-.810	.427	-.740	1.020	-.199	-.726	.477
	DASS-A	2.663	1.714	.507	1.554	.135	2.668	1.575	.508	1.694	.107
	DASS-S	-2.009	1.654	-.473	-1.215	.238	-2.288	1.533	-.538	-1.493	.152
	YBOCS-O						-.440	2.508	-.037	-.175	.863
	YBOCS-C						**7.042**	**2.848**	**.473**	**2.473**	**.023***
		R^2 =.341, F(4, 8) = 1.034, p = .446					$R^2 \Delta$ =.127, F(5, 7) = 1.230, p = .387				
TTM	Gender	10.183	31.647	.094	.322	.756	-2.337	31.913	-.022	-.073	.944
	DASS-D	.382	1.534	.088	.249	.809	1.185	1.599	.273	.741	.483
	DASS-A	1.082	2.506	.156	.432	.677	1.948	2.499	.281	.779	.461
	DASS-S	-3.343	2.041	-.695	-1.637	.140	-4.246	2.082	-.882	-2.039	.081
	MGH						-3.708	2.871	-.403	-1.291	.238
		R^2 =.496, F(4, 4) = .984, p = .506					$R^2 \Delta$ =.013, F(5, 3) = .622, p = .701				
Mixed Diagnosis	Gender	48.065	52.068	.462	.923	.408	34.532	76.201	.332	.453	.681
	DASS-D	.331	3.312	.075	.100	.925	.603	3.895	.137	.155	.887
	DASS-A	-3.285	3.690	-.650	-.890	.424	-2.814	4.522	-.557	-.622	.578
	DASS-S	.318	3.248	.060	.098	.927	.001	3.868	.000	.000	1.000
	OCRD Severity						-10.472	36.995	-.189	-.283	.796

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: SSRT=Stop-signal reaction time, YBOCS-T=Yale-Brown Obsessive-Compulsive Scale Total Scores, YBOCS-O= Yale-Brown Obsessive-Compulsive Scale Obsessional Severity, YBOCS-C=Yale-Brown Obsessive-Compulsive Scale Compulsion Severity, DASS-D=Depression, DASS-A=Anxiety, DASS-S=Stress, MGH=Massachusetts General Hairpulling Scale Total Severity, OCD=Obsessive Compulsive Disorder, TTM=Trichotillomania, OCRD Severity=z-centered YBOCS and MGH scores.

Table 8.

Whole Sample Correlations of OCRD Severity and SST Indices with Associated Features of OCRDs

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13
1. p.RS	–	–	–	–	–	–	–	–	–	–	–	–	–
2. SSD	-.625***	–	–	–	–	–	–	–	–	–	–	–	–
3. SSRT	.232	-.594***	–	–	–	–	–	–	–	–	–	–	–
4. Commission RT	-.547***	.917***	-.332*	–	–	–	–	–	–	–	–	–	–
5. goRT	-.644***	.965***	-.363*	.954***	–	–	–	–	–	–	–	–	–
6. Go-%	-.102	-.216	-.085	-.252	-.287*	–	–	–	–	–	–	–	–
7. Omis-%	-.018	.319*	.050	.362*	.392**	-.974**	–	–	–	–	–	–	–
8. OCRD Severity	-.059	.231	-.005	.250	.262	-.024	.073	–	–	–	–	–	–
9. BIS-11 AI	-.013	.132	-.045	.080	.138	-.142	.147	.061	–	–	–	–	–
10. BIS-11 MI	.106	-.132	.093	-.205	-.123	-.195	.196	.077	.372**	–	–	–	–
11. BIS-11 NPI	-.026	-.037	.048	-.087	-.027	-.027	.027	-.074	.465**	.616***	–	–	–
12. OCTDCQ-HA	-.083	.404**	-.239	.312*	.390**	-.237	.289*	.261	.605***	.225	.260	–	–
13. OCTDCQ-I	-.224	.384**	-.123	.340*	.406**	-.320*	.354*	.206	.443**	.229	.122	.717***	–
14. SDS-GI	-.112	.169	-.047	.134	.179	-.015	.045	.137	.489***	.357*	.327*	.478***	.428**

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: p.RS=Probability of Commission, SSRT= Stop-Signal Reaction Time, SSD=Stop-Signal Delay, goRT=Mean reaction time on Go trials, Go-%=Correct go-trial percentage, Omis-%=Omission Percentage, BIS-11 AI= Attentional Impulsivity, BIS-11 MI= Motor Impulsivity, BIS-11 NPI=Non-Planning Impulsivity, HA=Harm Avoidance, OCTDCQ-HA=Harm Avoidance, OCTDCQ-I=Incompleteness, SDS-GI=Global Impairment.

Table 9.

Correlations of OCRD Severity and SST Indices with Associated Features of OCRDs in Both OCD and TTM

		TTM															
	Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
OCD	1. p.RS	–	-.749**	.525	-.618*	-.753**	.038	-.159	n/a	n/a	n/a	-.112	-.172	-.014	-.235	-.144	-.133	-.461
	2. SSD	-.479*	–	-.791**	.916***	.983***	-.394	.511	n/a	n/a	n/a	.368	.375	.088	.095	.516	.236	.564*
	3. SSRT	-.052	-.494**	–	-.569*	-.666*	.358	-.460	n/a	n/a	n/a	-.280	.010	-.015	-.183	-.479	-.213	-.221
	4. Commission RT	-.459*	.915***	-.248	–	.945***	-.272	.384	n/a	n/a	n/a	.487	.379	-.061	-.173	.378	.016	.509
	5. goRT	-.550**	.957***	-.222	.944***	–	-.385	.495	n/a	n/a	n/a	.363	.468	.110	.068	.489	.230	.623*
	6. Go-%	.193	-.574**	-.058	-.587**	-.628***	–	-.975***	n/a	n/a	n/a	-.014	-.569*	-.590*	-.236	-.526	-.576*	-.327
	7. Omis-%	-.326	.628***	-.048	.664***	.686***	-.946***	–	n/a	n/a	n/a	.066	.551	.574*	.205	.610	.611*	.413
	8. YBOCS-T	.178	-.002	.326	.041	.103	-.340	.323	–	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
	9. YBOCS-O	-.038	.155	.136	.057	.216	-.366	.334	.862***	–	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
	10. YBOCS-C	.378	-.197	.433*	.005	-.081	-.170	.179	.770***	.341	–	n/a	n/a	n/a	n/a	n/a	n/a	n/a
	11. MGH-T	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	–	.073	-.355	-.649*	.278	.040	.290
	12. BIS-11 AI	.108	-.067	-.152	-.249	-.132	.146	-.077	.121	.228	-.063	n/a	–	.572*	.247	.433	.674*	.797***
	13. BIS-11 MI	-.034	-.213	.247	-.243	-.161	.086	-.029	.211	.201	.137	n/a	.452*	–	.569*	.373	.580*	.418
	14. BIS-11 NPI	.130	-.211	.178	-.202	-.178	.060	-.031	.085	.026	.124	n/a	.392*	.739***	–	.221	.319	.208
	15. OCTDCQ-HA	.145	.270	-.318	.049	.194	-.225	.215	.367	.478*	.078	n/a	.495**	.176	.019	–	.666*	.567*
	16. OCTDCQ-I	-.342	.516**	-.203	.407*	.510**	-.409*	.388*	.305	.453*	-.005	n/a	.047	.089	-.217	.397*	–	.706**
	17. SDS-GI	.301	-.280	.171	-.239	-.257	.195	-.190	.044	-.070	.169	n/a	.234	.420*	.296	.141	.090	–

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: p.RS=Probability of Commission, SSRT= Stop-Signal Reaction Time, SSD=Stop-Signal Delay, goRT=Mean reaction time on Go trials, Go-%=Correct go-trial percentage, Omis-%=Omission Percentage, BIS-11 AI= Attentional Impulsivity, BIS-11 MI= Motor Impulsivity, BIS-11 NPI=Non-Planning Impulsivity, HA=Harm Avoidance, OCTDCQ-HA=Harm Avoidance, OCTDCQ-I=Incompleteness, SDS-GI=Global Impairment, YBOCS-T=Yale-Brown Obsessive-Compulsive Scale Total Scores, YBOCS-O= Yale-Brown Obsessive-Compulsive Scale Obsessional Severity, YBOCS-C=Yale-Brown Obsessive-Compulsive Scale Compulsion Severity, MGH-T=Massachusetts General Hairpulling Scale Severity, YBOCS-T=Yale-Brown Obsessive-Compulsive Scale Total Scores, YBOCS-O= Yale-Brown Obsessive-Compulsive Scale Obsessional Severity, YBOCS-C=Yale-Brown Obsessive-Compulsive Scale Compulsion Severity.

Table 10.

Relationship between Individual Items on the YBOCS with SST Variables in an OCD Group

Item	p.RS	SSD	SSRT	Com.RT	goRT	Go-%	Omis-%
Obsession Items							
Time occupied by obsessive thoughts	.053	.146	.007	.032	.163	-.196	.160
Interference from obsessions	.042	-.123	.283	-.166	-.047	-.099	.089
Distress from obsessions	-.038	.081	.108	-.053	.124	-.359	.338
Effort to resist obsessions	-.093	.291	.066	.303	.349	**-.593*****	**.543****
Control over obsessions	-.128	.228	.090	.119	.285	-.241	.231
Compulsion Items							
Time spent compulsions	.095	-.061	.262	.060	.013	-.296	.338
Interference from compulsions	.294	-.147	**.469***	-.032	-.012	-.112	.079
Distress from prevented compulsions	**.482***	-.345	**.402***	-.160	-.254	.216	-.226
Effort to resist compulsions	.280	-.227	.277	-.047	-.162	-.163	.143
Control over compulsions	.177	.119	.129	.217	.170	-.240	.300
Insight	.102	.005	.175	.015	.067	-.272	.208
Avoidance	.311	-.156	.288	-.194	-.081	-.241	.213

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: p.RS=Probability of Commission, SSRT= Stop-Signal Reaction Time, SSD=Stop-Signal Delay, Com.RT=Commission Reaction Time, goRT=Mean reaction time on Go trials, Go-%=Correct go-trial percentage, Omis-%=Omission Percentage.

Table 11.

Relationship between Individual Items on the MGH with SST Variables in a TTM Group

Item	p.RS	SSD	SSRT	Com.RT	goRT	Go-%	Omis-%
Frequency of urges	-.257	.397	-.121	**.591***	.443	.114	-.025
Intensity of urges	-.508	.530	-.272	**.661***	**.559***	.048	.071
Ability to control urges	-.152	.322	-.226	.325	.324	.027	.037
Frequency of hairpulling	-.074	.317	-.052	.551	.368	-.173	.240
Attempts to resist pulling	.401	-.022	-.246	-.025	-.096	-.009	-.044
Control over hairpulling	.021	.252	-.343	.218	.208	-.278	.299
Associated distress	.010	.020	-.091	.092	-.001	.111	-.160

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: p.RS=Probability of Commission, SSRT= Stop-Signal Reaction Time, SSD=Stop-Signal Delay, Com.RT=Commission Reaction Time, goRT=Mean reaction time on Go trials, Go-%=Correct go-trial percentage, Omis-%=Omission Percentage.

APPENDIX B – FIGURES

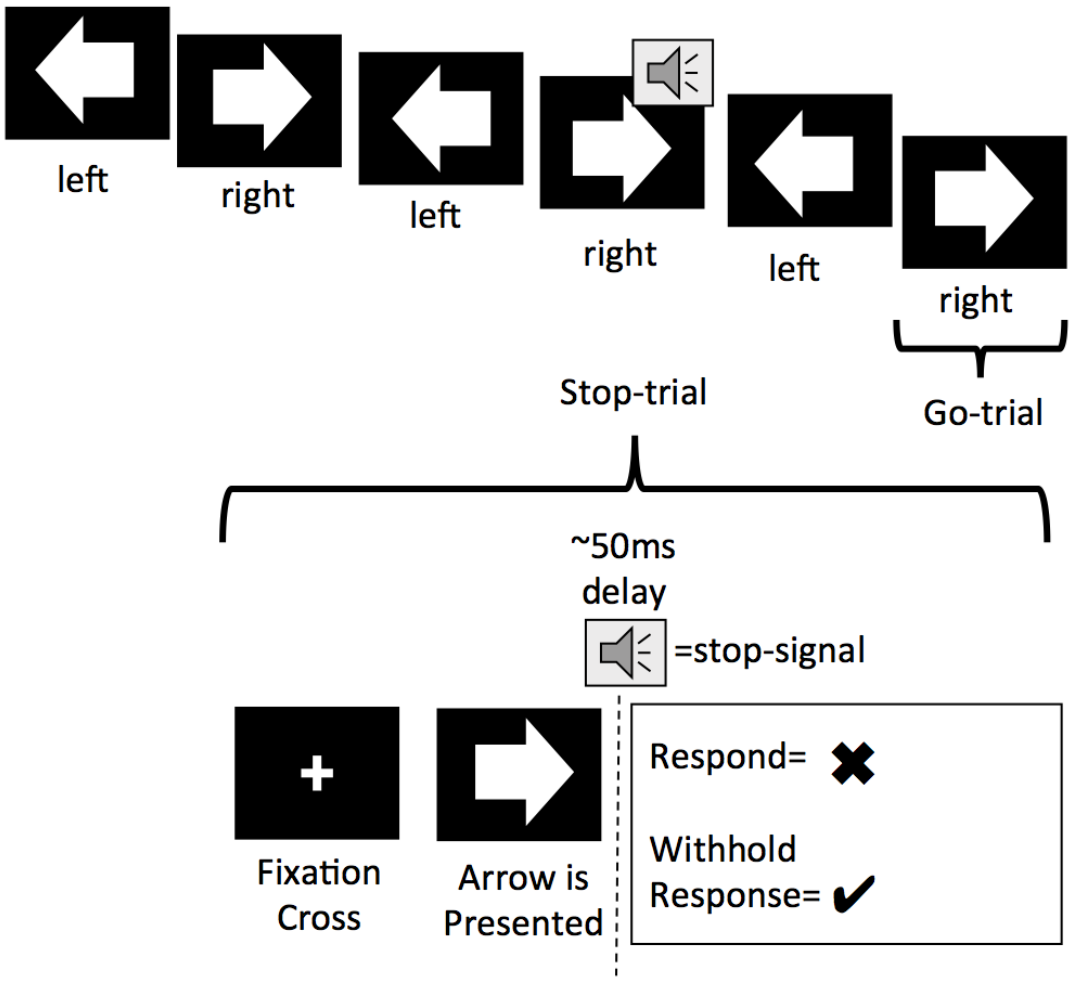


Figure 1. Illustration of the stop-signal task (SST) (Verbruggen & Logan, 2009). In this task, a subject is presented with a series of stimuli to respond to ("go-stimuli") with arrow keys. On 33% percent of the trials subjects are presented with a "stop-stimuli" (i.e., a beep sound) after a very short delay ("stop-signal delay"). Subjects need to withhold a response that has already been initiated, and data from these responses are extrapolated into a "Stop-Signal Reaction Time," an index of response inhibition capabilities.

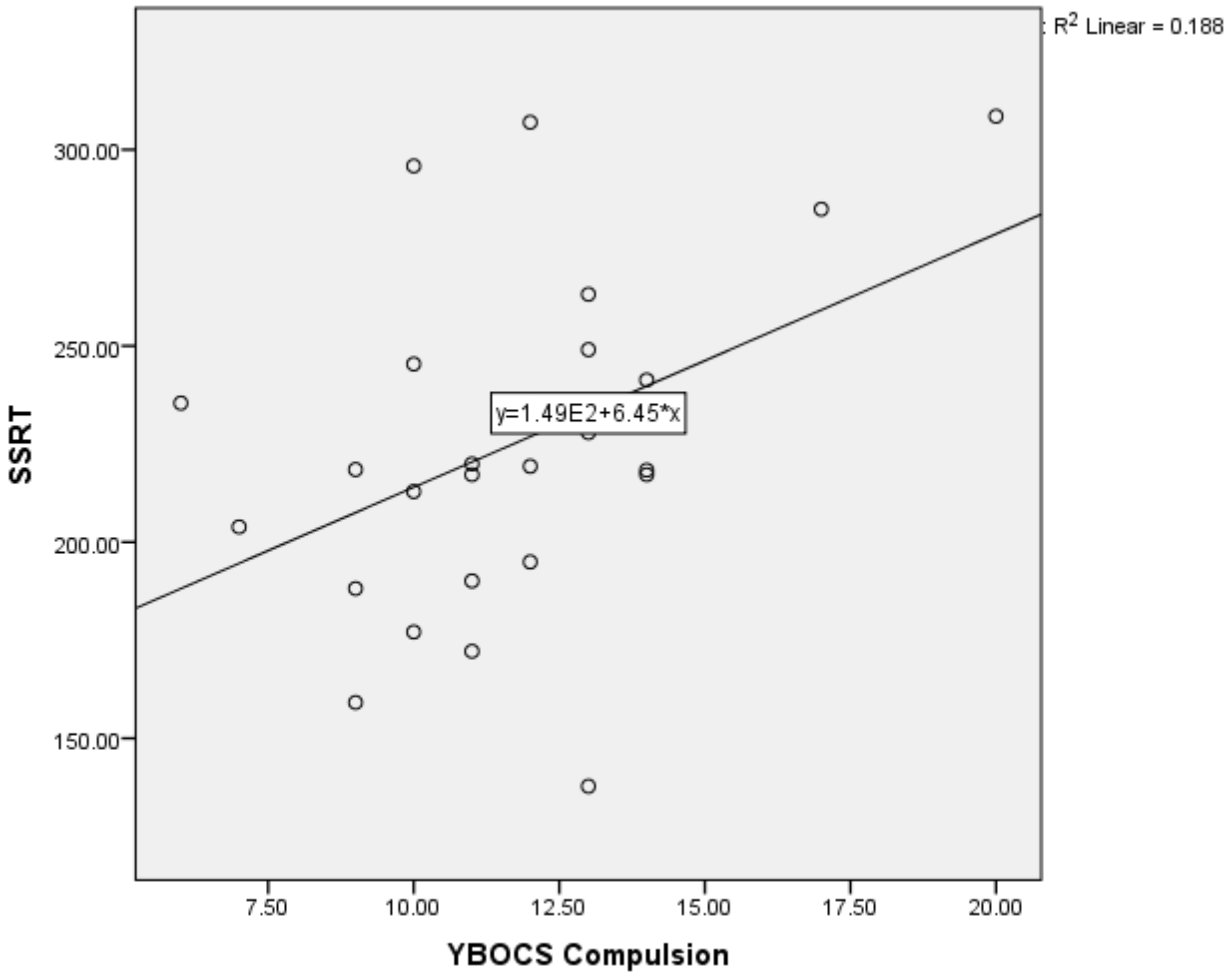
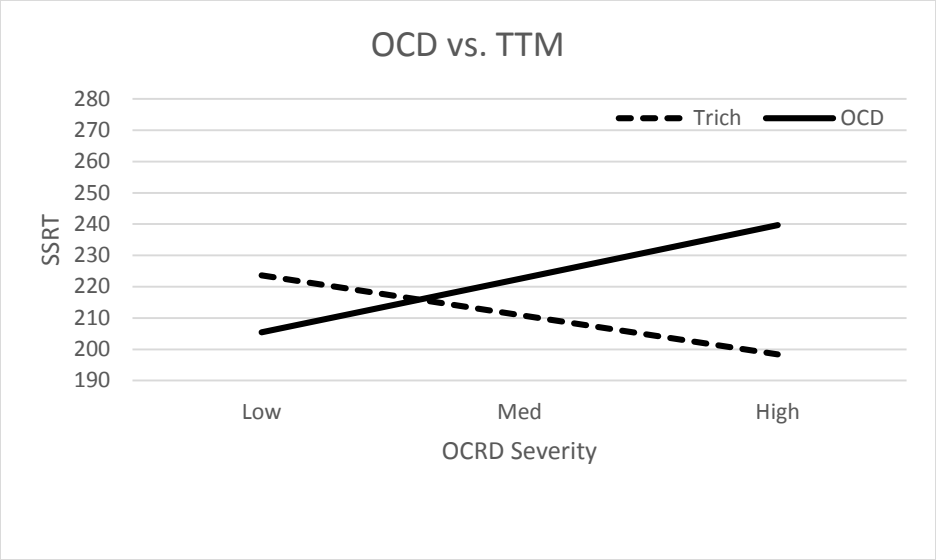


Figure 2. Scatterplot illustration of the relationship between SSRT and YBOCS compulsion scores among those with OCD.



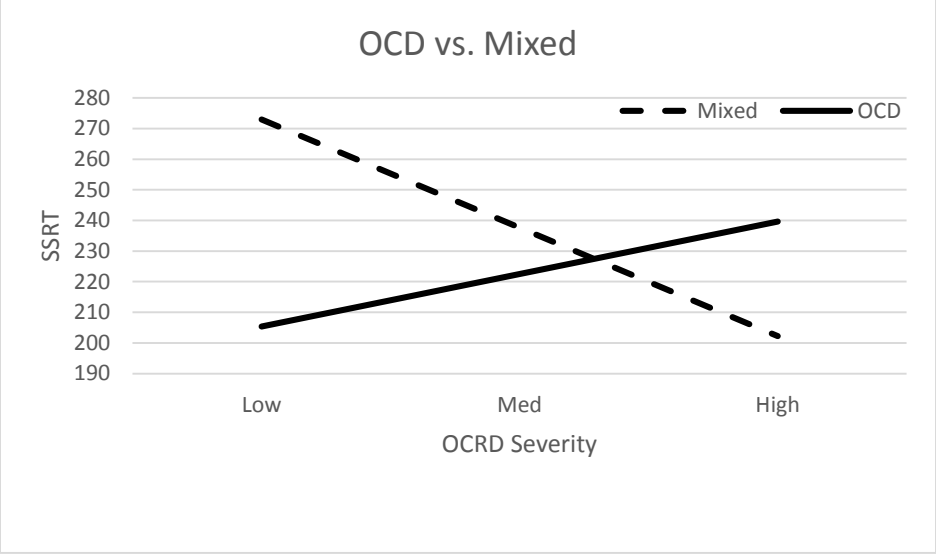


Figure 2. Interaction of diagnostic group (i.e., OCD vs TTM and OCD vs. Mixed Diagnosis) in predicting OCRD severity and SSRT.