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Response inhibition and error-monitoring processes in individuals with obsessive-compulsive disorder*

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Abstract

Response inhibition (RI) has been putatively linked to the symptoms of OCD. Despite the enticing link between RI and OCD, there are points in the relationship that require clarification. We examined the RI-OCD relationship taking into account a) the potentially differential pattern of RI-OCD relationship between obsessions and compulsions, and b) the potentially confounding effect of negative affect, particularly in regards to depression. Additionally, we investigated how error-monitoring processes in the inhibitory context account for OCD symptoms. Results showed that the RI-OCD relationship is robust in regards to compulsion symptoms, but not for obsessions, even when controlling for negative affect. Additionally, while individuals with OCD display behavioral slow-down following commission errors on the stop-signal task, slow-down following successful inhibition is significantly related to compulsion symptoms. Findings suggest that future studies investigating RI in OCD should take into account heterogeneous clinical presentations in OCD, as well as incorporate error-monitoring variables to better understand RI processes in OCD.

Keywords

response inhibition; error-monitoring; compulsion; stop-signal task

Introduction

Obsessive-Compulsive Disorder and Response Inhibition

Obsessive-compulsive disorder (OCD) is a debilitating psychiatric condition characterized by intrusive, unwanted mental images or urges (obsessions) and repetitive behaviors to reduce distress from such unwanted thoughts (*compulsions*) (DSM-5; APA, 2013). Epidemiological surveys suggest a lifetime prevalence of OCD of 2.3% (Ruscio, Stein, Chiu, & Kessler, 2010), and rank the condition in the top-ten leading causes of disability

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To examine the potential impact of comorbid ADHD (n=4) on the current findings, we repeated the primary analyses including only participants without ADHD (n=28). SSRT was still a significant predictor of compulsion symptoms ($R^2 = .168$, $F(1, 23) = 4.773$, $p = .039$; $\beta = .029$, $t = -2.185$), as was ERM ($R^2 = .146$, $F(1, 22) = 4.826$, $p = .039$; $\beta = -.414$, $t = -2.197$). Therefore, the overall pattern of findings remained identical.

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worldwide (Lopez & Murray, 1998). The DSM-5 clusters OCD with other related conditions (OCDs) marked by compulsive behaviors such as trichotillomania, skin-picking disorder, body dysmorphic disorder, and hoarding disorder (APA, 2013).

As a relevant cognitive deficit in compulsive behavior, researchers have focused on response inhibition (RI) (Bannon, Gonsalvez, Croft, & Boyce, 2002; Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005; Chamberlain & Menzies, 2009). RI is a cognitive process that refers to the ability to deliberately inhibit inappropriate or irrelevant responses (Verbruggen & Logan, 2009). Characterized as “top-down” inhibitory control (Dalley, Everitt, & Robbins, 2011), RI is often measured through computerized cognitive tasks such as the *stop-signal* (SST) or go/no-go task, which prompt respondents to selectively withhold or cancel pre-potent motor responses (Verbruggen, Logan, & Stevens, 2008).

Deficient RI performance has been observed in a number of studies comparing individuals with OCD with control groups (Bannon et al., 2002; Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Chamberlain et al., 2007; Lei et al., 2015; Morein-Zamir, Fineberg, Robbins, & Sahakian, 2010; Penades et al., 2007; Sohn, Kang, Namkoong, & Kim, 2014), as well as in their first-degree relatives (Chamberlain et al., 2007; Menzies et al., 2007; Rajender et al., 2011). Further, structural and functional abnormalities in inhibitory control circuitry (i.e., fronto-striatal circuit) have been well documented for OCD (de Wit et al., 2012; Kang et al., 2013; Menzies et al., 2007; Page et al., 2009; Roth et al., 2007; Woolley et al., 2008), consistent with current biological models of OCD (Menzies et al., 2008; Vaghi et al., 2017). EEG studies have also shown abnormal event-related potentials in frontal regions (i.e., reduced amplitudes of N2, P3) during RI task performance, which are significantly associated with OCD symptom severity (Herrmann, Jacob, Unterecker, & Fallgatter, 2003; Kim, Kim, Yoo, & Kwon, 2007). It should be noted that a handful of studies failed to demonstrate the behavioral evidence of deficient RI in OCD on the go/no-go task (Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008) or SST (Fan et al., 2017; Kalanthroff et al., 2017). Nonetheless, the overall literature offers extensive neurobehavioral and psychophysiological data pointing to the significance of RI deficits as a pathological underlying feature of OCD (for a review, van Velzen, Vriend, de Wit, & van den Heuvel, 2014). RI deficits have also been found in other OCDs such as trichotillomania (Chamberlain et al., 2006) and skin-picking disorder (Grant, Odlaug, & Chamberlain, 2011; Odlaug, Chamberlain, & Grant, 2010). Phenomenologically, impaired RI faculties are consistent with the overt clinical presentation of OCDs, characterized by inhibitory failures (Abramowitz & Jacoby, 2015). Accumulated data have led numerous researchers to suggest that impaired inhibitory control is a putative “neurocognitive endophenotype” of OCD and its related conditions (Chamberlain et al., 2005; Montague, Dolan, Friston, & Dayan, 2012; Robbins, Gillan, Smith, de Wit, & Ersche, 2012).

Difficulties in Understanding the RI-OCD relationship

Despite its significance, the RI-OCD relationship is rather poorly understood. Firstly, it is unknown which components of OCD symptomology are associated with RI deficits. OCD shows remarkably heterogeneous clinical features, (Bloch, Landeros-Weisenberger, Rosario, Pittenger, & Leckman, 2008), and it is understudied which specific facets of OCD are linked

to RI deficits. It is possible that overt behavioral failure in inhibition (i.e., compulsions), relative to ruminative/obsessional symptoms, may more directly reflect underlying RI deficits. However, existing studies have primarily relied on overall OCD severity in RI investigations, and there is a lack of fine-grained analyses in the literature (McLaughlin et al., 2016). Particularly, the presence of inconsistent findings in RI-OCD association (Bohne et al., 2008; Fan et al., 2017; Kalanthroff et al., 2017) calls for a more careful and detailed analysis to clarify the nature of inhibitory difficulties in OCD as a function of its symptom dimensions.

Secondly, very little is known how concurrent negative affect (NA), such as depression, color the RI-OCD relationship. Specifically, depression is associated with various cognitive deficits, which are critical for the successful completion of cognitive tasks such as working memory, planning, and global motor slowing (Austin, Mitchell, & Goodwin, 2001; Dillon et al., 2015; McIntyre et al., 2013). Meta-analyses have suggested that in depression, critical deficits in overall cognitive resources and psychomotor slowing can all manifest as poor inhibitory control (Fossati, Ergis, & Allilaire, 2002). Additionally, depression is a markedly common comorbidity (10 times greater than in the general population) of OCD (Denys, Tenney, van Megen, de Geus, & Westenberg, 2004; Pallanti, Grassi, Sarrecchia, Cantisani, & Pellegrini, 2011). There is also some evidence that executive function deficits in OCD are attenuated when concurrent depressive symptoms are controlled for (Aycicegi, Dinn, Harris, & Erkmén, 2003; Basso, Bornstein, Carona, & Morton, 2001), which suggest the possibility that such cognitive deficits in OCD could merely be a byproduct of concurrent negative affect rather than reflecting the core feature of OCD. Considering these findings, continuing research is warranted to better understand the RI-OCD relationship while taking into consideration the influence of NA, including depressive symptoms, as poor RI performance in OCD could merely reflect the influence of concomitant NA.

Lastly, existing literature may have captured only a limited aspect of inhibitory processes in OCD because most studies utilize a single index of RI per task given. For instance, in most stop signal task (SST) investigations, stop-signal reaction time (SSRT) is the core, and only variable of interest that details inhibition (Verbruggen & Logan, 2009). However, RI is a complex cognitive process involving multi-faceted inhibitory sub-processes (Eagle, Bari, & Robbins, 2008; Friedman & Miyake, 2004; Schachar et al., 2007). Other useful avenues for understanding RI exist, which take into account how subjects monitor their own performance while attending to stop-signals. Typically understood as changes in reaction time following 'go' versus 'stop' trials, models have been developed to examine whether subjects engage in "error-monitoring (ERM)", or, a slowing in reaction speed following errors (Schachar et al., 2004). In addition to SSRT, ERM is another executive index that reflects top-down control of behavior (Logan, 1985). In the context of deliberate inhibition, ERM circuits can trigger the internal sensation that 'something is wrong,' leading to changes in internal state (i.e., anxiety) and ultimately in inhibitory task performance (Aouizerate et al., 2004). Taken together, this investigation sought to expand the current literature by examining how concurrent mood symptoms affect the RI-OCD association while taking into account OCD symptom dimensions and utilizing multiple indices of inhibitory processes derived from the well-established SST paradigm.

Research into ERM processes in OCD is still in a nascent stage, and has mostly utilized the GNG task in the context of event-related potential (ERP). Endrass, Klawohn, Schuster, and Kathmann (2008) found enhanced correct-related negativity (CRN) amplitudes that were correlated with symptom severity in an OCD sample, and suggested that individuals with OCD may possess a hyperactive performance monitoring mechanism and cannot effectively disengage from observing their own mistakes (Endrass et al., 2010). While Riesel, Kathmann, and Endrass (2014) did not find a significant difference between OCD patients and controls on behavioral indices of ERM, they found a similar amplified error-related negativity (ERN), indicative of hyperactive performance monitoring. In sum, examining how subjects monitor their own performance, especially on SST trials in which inhibitory processes occur, can add valuable data for more nuanced understanding of inhibitory processes in the context of OCD (Bissett & Logan, 2011).

Present Study

This study sought to examine the RI-OCD association taking into account a) the potentially confounding influence of NA, and b) the potential difference between obsessions and compulsions in their association with RI performance. We hypothesized that when controlling for NA, RI performance would still remain a significant predictor of OCD symptoms. We predicted that this would be observed particularly for the severity of compulsions (rather than obsessions), as the motor inhibition failure captured by SSRT appears to closely match the pathological behavioral features of compulsions. Additionally, due to the theorized hyperactive monitoring mechanism proposed from existing ERP data, we hypothesized that a) subjects would exhibit greater slow down after failed inhibition trials relative to successful inhibition trials, and b) the amount of slow-down would be associated with symptoms of OCD, particularly to compulsive symptoms. We sought to examine how SSRT and ERM variables would contribute to predicting OCD symptoms relative to each other.

Method

Participants

Participants were recruited for a clinical trial testing the effectiveness of a computer-based treatment for OCD and TTM. They were assessed in the context of a full eligibility assessment following an online pre-screening survey and brief phone screening. Inclusion criteria were: a) OCD as the primary OCD diagnosis on the Anxiety and Related Disorders Interview Schedule for DSM-5 (ADIS-5); b) ages between 18-60; c) overall intellectual functioning (as assessed by the Wechsler Abbreviated Scale of Intelligence-II) ≥ 80 . Exclusion criteria were: a) bipolar or other psychotic disorders; and b) invalid responding on the SST (e.g., failed to follow instructions; a final stop accuracy significantly different from 50%; a number of non-responded go trials due to strategic waiting or inattention [30 or more omission errors]).

Among 83 individuals screened, 32 met the study entry criteria. Their demographic characteristics are provided in Table 1. Their mean age was 27.50 (SD=10.10). There were more females (female = 62.5%, $n=20$; male = 37.5%, $n = 12$), and the sample was largely

Caucasian (81.3%, $n = 26$). The three most common comorbidities, in order, were: generalized anxiety disorder (GAD) (59.4%, $n = 19$), social anxiety disorder (53.1%, $n = 17$), and major depressive disorder-current episode (37.5%, $n = 12$).

Measures

Diagnostic and clinical measures—All measures given were administered in a single session following the informed consent procedure. All procedures were approved by the Institutional Review Board (IRB). At the study session, participants were assessed for a) diagnostic status, b) symptom severity, and c) response inhibition performance. Diagnostic status was confirmed through the ADIS-5 (ADIS-5) (Brown & Barlow, 2013) and TTM was assessed with the Trichotillomania Diagnostic Interview (TDI) (Rothbaum & Ninan, 1994). Overall OCD symptoms were assessed with the self-report version of the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS-SR) (Federici et al., 2010) and NA variables (e.g., depression, anxiety, stress) were assessed with the Depression Anxiety Stress Scale (DASS-21) (Lovibond & Lovibond, 1995).

Stop Signal Reaction Time (SSRT)—Response inhibition performance was measured via the stop-signal task (SST) (Logan, 1994; Verbruggen et al., 2008), administered through Inquisit software. Subjects underwent a practice block with 32 trials and three main blocks with 64 trials each. Stop-signals occurred randomly on 33% percent of trials. During the practice block, the stop-signal delay (SSD), which was initially set at 250ms, was adjusted (i.e., increased by 50ms after a successful stop trial; decreased by 50ms after a failed inhibition) to establish a personally-relevant starting SSD value for the main blocks. SSD was adjusted continuously throughout the main task to render the probability of correctly inhibiting stop-signals approximately 50%. SSRT is calculated by subtracting the mean SSD from mean go RT (Chamberlain et al., 2007; Verbruggen & Logan, 2009), and reflects the latency of an individual's stop process (Verbruggen & Logan, 2008).

Error-monitoring (ERM) Indices—To index error-monitoring, we computed mean RTs from three different types of go trials on the SST, following C. S. Li et al. (2008): (a) go trials following other go trials (post-go trials: pG), (b) go trials following successful stop trials (post-stop-success trials = pSS), and (c) go trials following failed stop trials (post-stop-error trials = pSE) (C.-s. R. Li et al., 2008; Li, Milivojevic, Kemp, Hong, & Sinha, 2006). First, we computed the hyper-error-monitoring index using mean RTs from pSE and pSS trials: $ERM = pSE\ RT - pSS\ RT$. This indicates the magnitude of slow-down after inhibitory failure as compared with successful inhibition. In line with the hyper-error-monitoring account of OCD, we expected pSE RT to be longer than pSS RT. Significant differences between pSE and pSS may suggest inefficiency in inhibitory control associated with excessive error-monitoring. Second, to further specify the nature of ERM, we produced two sub ERM indices, using pG trials as a reference condition: (1) $pSE-ERM (=pSE-pG)$ and (2) $pSS-ERM (=pSS-pG)$. These indices reflect the magnitude of slow-down following stop trials (failed vs. successful, respectively) in reference to the regular go RT. Existing data suggest that healthy controls slow-down following inhibition trials regardless of their success or failure, compared to pG trials (i.e., pSS and pSE vs. pG) (C.-s. R. Li et al., 2008). In contrast, OCD symptoms may be differentially associated with the two sub-ERM

processes in the context of inhibitory control processes. Taken together, ERM, pSE-ERM, and pSS-ERM were used in this study to examine the error-monitoring processes in the SST context.

Data Analysis

We utilized hierarchical linear regression to parse out the relative influence of three domains in explaining symptoms of OCD: negative affect [NA] (depression, anxiety and stress) in Step 1, SSRT in Step 2, and ERM indices in Step 3. Three regression models were tested separately, with overall OCD severity, obsessional severity, or compulsion severity as dependent variables.

Results

Mean values and correlation amongst measures are presented in Table 2. Y-BOCS total scores, ($r = .372$, $p < .05$) and Y-BOCS obsession scores ($r = .393$, $p < .05$) were significantly associated with anxiety, but not with depression, stress, SSRT, or ERM indices. In contrast, Y-BOCS compulsion scores were significantly associated with SSRT ($r = .356$, $p < .05$), and showed marginal significance with ERM ($r = -.319$, $p = .075$). SSRT was not significantly associated with any ERM indices. Depression ($r = .427$, $p < .05$), anxiety ($r = .393$, $p < .05$), and stress ($r = .420$, $p < .05$) were all significantly associated with pSE-ERM. Ranges for clinical measures are as follows: Y-BOCS obsession severity (= 0-20); Y-BOCS compulsion severity (= 6-20); Y-BOCS total scores (= 6-40); DASS depression severity (= 0-42); DASS anxiety severity (= 2-38); DASS stress severity (= 4-42).

To explore relative magnitudes of ERM indices, we calculated a paired-samples t-test for pG RT, pSS RT, and pSE RT. These results are illustrated in Figure 1. pSE RT was significantly greater than pSS RT ($t(31)=4.114$, $p < .001$), and was significantly greater than pG RT ($t(31)=5.237$, $p < .001$). pSS RT did not differ significantly from pG RT ($t(31)=1.293$, $p > .05$).

Results from the main regression analysis are presented in Table 3. First, in predicting total OCD severity, Step 1 variables (depression, anxiety, stress) explained approximately 15.3% of the variance ($R^2 = .153$, $F(3, 28) = 1.684$, $p = .193$). In Step 2, SSRT explained an additional 6.9% of the variance in total OCD symptoms but was not a significant predictor ($R^2 = .069$, $F(1, 27) = 2.403$, $p = .133$; $\beta = .287$, $t = 1.550$, $p = .133$). In Step 3, ERM accounted for an additional 1.6% of the variance and was not a significant predictor ($R^2 = .016$, $F(1, 26) = .560$, $p = .461$; $\beta = -.133$, $t = -.749$, $p = .461$).

In predicting obsessional severity, NA indices in Step 1 explained 18.6% of the variance in obsessions ($R^2 = .186$, $F(3, 28) = 2.138$, $p = .188$) with anxiety as a significant predictor ($\beta = .594$, $t = 2.161$, $p = .039$). In Step 2, SSRT explained less than 1% in additional variance ($R^2 = .006$, $F(1, 27) = .191$, $p = .666$; $\beta = .082$, $t = .437$, $p = .666$). In Step 3, ERM explained less than 1% in additional variance in obsessional severity ($R^2 = .003$, $F(1, 26) = .095$, $p = .760$; $\beta = .056$, $t = .308$, $p = .760$).

In predicting compulsion severity, Step 1 variables explained 3.8% of the variance in compulsions ($R^2 = .038$, $F(3, 28) = .370$, $p = .775$), with none of the three NA indices being significant predictors. In Step 2, SSRT was a significant predictor, explaining an additional 17.8% of variance in compulsion severity ($R^2 = .178$, $F(1, 27) = 6.121$, $p = .020$; $\beta = .460$, $t = 2.474$, $p = .020$). In Step 3, ERM was also a significant predictor, explaining an additional 11.1% of the variance in compulsion severity ($R^2 = .111$, $F(1, 26) = 4.278$, $p = .049$; $\beta = -.345$, $t = -2.068$, $p = .049$). In the final step, only the SSRT and ERM remained as significant predictors of the severity of compulsion. These findings suggest that even when factoring out the influence of NA, both SSRT and ERM significantly contributed to predicting compulsive symptoms.

To further elaborate on the contribution of ERM in predicting compulsion severity, we conducted an additional hierarchical regression analysis where ERM in Step 3 was substituted by the error-monitoring sub-processes of pSS-ERM and pSE-ERM. In this analysis, SSRT remained a significant predictor ($\beta = .456$, $t = 2.540$, $p = .018$), and pSS-ERM also emerged as a significant predictor ($\beta = .403$, $t = 2.153$, $p = .041$), but not pSE-ERM ($\beta = -.264$, $t = -1.295$, $p = .207$). These findings suggest that slow-down following *successful* inhibition was significantly associated with the psychopathology of compulsions, whereas slow-down after errors was not.

Taken together, in predicting compulsive symptoms of OCD, SSRT was a strong predictor even when factoring out the influence of depression and negative affect. Likewise, ERM variables also significantly predicted severity of compulsions, particularly indices of slow-down following successful inhibition.¹

Discussion

RI and inhibitory capabilities have become a point of focus in conceptualizing OCD and its related conditions. Using a community sample of individuals diagnosed with OCD, we sought to further examine the nature of RI-OCD relationship, taking into account fine-grained analyses of OC symptoms, factoring out potentially confounding effects of negative affect, and exploring error-monitoring as additional indices of inhibitory process. We hypothesized that RI processes would significantly predict symptoms of OCD, even when controlling for the potentially confounding influence of NA (including depressive symptoms), particularly in regards to severity of compulsions. We found that SSRT was significantly related to OCD symptoms *only* for their compulsional severity (approximately 18% of the variance in symptoms). Previous RI studies in OCD rarely took into consideration distinctive symptom presentations (obsessions vs. compulsions) (McLaughlin et al., 2016), which may have contributed to inconsistent RI findings in OCD. Our data suggest the possibility that the RI deficits observed in OCD may be largely due to compulsions rather than obsessions. Phenomenologically, compulsions may be a behavioral demonstration of underlying RI deficits (Verbruggen & Logan, 2009).

It should also be noted that the relationship between RI deficits and compulsion severity remained significant when factoring out the influence of negative affect, including depressive symptoms, suggesting that the RI-compulsion relationship is robust to the

influence of NA. This is an important point as some evidence exists that executive function deficits in OCD could be attributable to comorbid depressive symptoms (Aycicegi et al., 2003; Basso et al., 2001). However, our data suggest that the RI deficits manifested by individuals with OCD are unlikely to be merely a byproduct of concurrent mood symptoms, but rather a pathological cognitive feature that is meaningfully associated with the severity of compulsions.

ERM indices explained an additional 11% of the variance in compulsive symptoms even after taking into account the influence of NA and the SSRT index. Contrary to our prediction based on the hyper-error-monitoring account, compulsion severity was significantly associated with behavioral slow-down following *successful* inhibition, but not for slow-down after inhibitory errors. Existing ERM studies suggest that healthy individuals display no difference in RT between pSE and pSS trials (C.-s. R. Li et al., 2008; C. S. Li et al., 2008). Taken together, these data suggest that slow-down following inhibitory error (i.e., failed inhibition) is a normal cognitive process whereas slow-down that occurs even after successful inhibition is a dysfunctional inhibitory process that may characterize OCD in the form of excessive error monitoring. Thus, pSS-related slow-down on the SST task warrants further investigations as a potentially useful cognitive index for OCD.

Several limitations should be mentioned. First, a lack of a control group limits the inference we can draw about how deviant the RI processes of the current OCD sample are. However, it should be noted that the overall SST value of our OCD sample (mean = 221ms, SD = 42) is comparable to those in existing studies: mean = 211ms, SD = 57 (Chamberlain et al., 2006); mean = 212ms, SD = 51 (Lei et al., 2015); and mean = 253ms, SD = 55 (Kalanthoff et al., 2017). Second, a relatively small sample size of the current study limits our statistical power. However, it is notable that this study revealed significant associations of the RI indices with the severity of compulsions, using a clinical sample recruited from a sizable community. Third, we utilized a self-report version of Y-BOCS as our only index of OCD severity (Y-BOCS-SR). Although the self-report Y-BOCS is considered to be quite reliable, participants do tend to under-report symptoms (Federici et al., 2010). Thus, future research should examine the RI indices and their association with OCD symptoms using clinician-administered ratings of symptoms. Lastly, this study focused on individuals with a primary OCD diagnosis to clarify how OCD symptom presentations are associated with various RI processes on SST. Future studies should expand the scope of these analyses to better understand the nature of RI processes in a broader OCRD context. Despite their grouping under the same OCRD cluster, OCD and other more body-focused repeated behavior problems (e.g., hair pulling or skin-picking) present important phenomenological and neurobehavioral differences (Grant & Kim, 2014; Roos, Grant, Fouché, Stein, & Lochner, 2015; van Velzen et al., 2014), which warrants sufficient research efforts for each OCRD condition to better understand how anomalous RI processes are implicated in these conditions in both unique and common ways.

Limitations notwithstanding, there are important aspects of our data that are expected to contribute to the literature. First, our data demonstrate the robust association between elevated SSRT and OCD symptoms (particularly, severity of compulsions) regardless of concurrent depressive and anxiety symptoms. This indicates the importance of considering

OCD symptom dimensions in RI research, as overlooking its clinical heterogeneity might have contributed to observed inconsistencies in OCD-RI findings. Second, even after controlling for the contribution of SSRT, the anomalous ERM index (i.e., increased monitoring/slow-down following successful inhibition) emerged as another meaningful index of inhibitory control that significantly explained the severity of compulsions. These findings indicate the need for the expanded scope of RI research in OCD by incorporating multiple relevant parameters of inhibitory processes to understand a more complete picture of RI deficits and their association with heterogeneous clinical presentations of OCD.

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Highlights

- Response inhibition (RI) has been linked to obsessive-compulsive disorder (OCD).
- Using the stop-signal task (SST), we examined aspects of the RI-OCD relationship.
- Poor RI on the SST predicted the severity of compulsions (but not obsessions).
- This relationship was not affected by the level of negative affect.
- Slow-down after successful inhibition also significantly predicted compulsions.

Figure 1.

Illustration of the mean RT values of trials following go trials (=pG RT), trials following stop trials where inhibition was successful (=pSS RT), and following stop trials where inhibition was unsuccessful (=pSE RT). A paired-samples t-test demonstrated significant differences between pSE RT and pG RT ($t(31)=5.237, p < .001$), and between pSE RT and pSS RT ($t(31)=4.114, p < .001$). pSE RT did not significantly differ from pG RT ($t(31)=1.293, p > .05$). n.s. = non-significant; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Table 1
Demographic Characteristics of Study Participants

Age [M(SD)]	27.50 (10.10)
Sex	
Male	37.5% (n = 12)
Female	62.5% (n = 20)
Race	
Caucasian	81.3% (n =26)
African American	9.4% (n = 3)
Two or more races	6.3% (n =2)
Hispanic/Latino	3.1% (n = 1)
WASI-2 FSIQ	102.40 (9.75)
Comorbidity	
PD	6.3% (n = 2)
Agoraphobia	6.3% (n = 2)
SAD	53.1% (n = 17)
GAD	59.4% (n = 19)
BDD	9.4% (n = 3)
Phobia	12.5% (n = 4)
PTSD	3.1% (n = 1)
MDD	37.5% (n = 12)
PDD	9.4% (n = 3)
IAD	3.1% (n = 1)
SUD	3.1% (n = 1)
TTM	9.4% (n = 3)
TD	3.1% (n = 1)
ADHD	12.5% (n = 4)
BN	3.1% (n = 1)
Other	3.1% (n = 1)

Note. Abbreviations: FSIQ-2=Full-Scale IQ, PD=Panic Disorder, SAD=Social Anxiety Disorder, GAD=Generalized Anxiety Disorder, BDD=Body Dysmorphic Disorder, PTSD=Post-Traumatic Stress Disorder,

MDD/PDD=Major/Persistent Depressive Disorder, IAD=Illness Anxiety Disorder, SUD=Substance Use Disorder, TTM=Trichotillomania, TD=Tic Disorder, ADHD=Attention Deficit Hyperactivity Disorder, BN=Bulimia Nervosa.

Table 2
Means and Standard Deviations of Study Measures, and their Zero-Order Correlations

Measure	Mean (SD)	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Y-BOCS Obs	10.78 (3.94)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2. Y-BOCS Comp	11.53 (2.81)	.376*	-	-	-	-	-	-	-	-	-	-	-	-	-
3. Y-BOCS Total	22.31 (5.64)	.887**	.762**	-	-	-	-	-	-	-	-	-	-	-	-
4. SSRT (ms)	221 (43)	.080	.356*	.233	-	-	-	-	-	-	-	-	-	-	-
5. SSD (ms)	414 (157)	.200	-.166	.057	-.432*	-	-	-	-	-	-	-	-	-	-
6. goRT (ms)	636 (143)	.242	-.078	.130	-.177	.964**	-	-	-	-	-	-	-	-	-
7. DASS-D	13.31 (11.88)	.134	.135	.161	-.328	.269	.193	-	-	-	-	-	-	-	-
8. DASS-A	14.00 (8.93)	.393*	.195	.372*	-.137	.248	.222	.662**	-	-	-	-	-	-	-
9. DASS-S	20.44 (10.48)	.221	.158	.233	-.316	.190	.107	.745**	.773**	-	-	-	-	-	-
10. pG RT (ms)	628 (143)	.222	-.093	.109	-.150	.952**	.995**	.153	.186	.072	-	-	-	-	-
11. pSS RT (ms)	643 (145)	.293	.040	.224	-.210	.909**	.930**	.265	.303	.163	.895**	-	-	-	-
12. pSE RT (ms)	694 (177)	.269	-.094	.142	-.233	.935**	.951**	.295	.308	.227	.923**	.924**	-	-	-
13. ERM (ms)	51 (70)	.076	-.319	-.106	-.156	.485**	.481**	.199	.151	.239	.483**	.270	.616**	-	-
14. pSS-ERM (ms)	15 (66)	.163	.288	.258	-.138	-.058	-.106	.251	.265	.201	-.193	.265	.038	-.451**	-
15. pSE-ERM (ms)	66 (71)	.225	-.048	.134	-.281	.423*	.374*	.427*	.393*	.420*	.296	.509**	.641**	.567**	.480**

Note.

* $p < 0.05$;

** $p < 0.01$; ms = millisecond

Y-BOCS Obs=Obsessional severity, Y-BOCS Comp=Compulsion severity, Y-BOCS Total=Total OCD severity, SSRT=Stop-signal reaction time, SSD=Stop-signal delay, goRT=Mean go-trial reaction Time, DASS-D=Depression severity, DASS-A=Anxiety severity, DASS-S=Stress severity, pG RT=Mean reaction time on go trials following go trials, pSS RT=Mean reaction time on go trials following successful stop-trials, pSE RT=Mean reaction time on go trials following unsuccessful stop trials, ERM=Error-monitoring index (= pSE -pSS), pSS-ERM=Successful inhibition [stop-success] reaction monitoring (= pSS - pG), pSE-ERM=Unsuccessful inhibition [stop-error] reaction monitoring (= pSE - pG).

Table 3
Hierarchical Linear Regression to Predict OCD Severity with Negative Affect, SSRT, and ERM

DV	Variable	Step 1					Step 2					Step 3				
		B	SE B	β	t	p	B	SE B	β	t	p	B	SE B	β	t	p
Y-BOCS Total		$R^2 = .153, F(3, 28) = 1.684, p = .193$					$R^2 = .069, F(1, 27) = 2.403, p = .133$	$R^2 = .016, F(1, 26) = .560, p = .461$								
	DASS-D	-.060	.127	-.126	-.475	.639	-.023	.126	-.049	-.184	.855	-.020	.127	-.043	-.160	.874
	DASS-A	.319	.177	.505	1.802	.082 [†]	.260	.177	.412	1.470	.153	.254	.179	.402	1.422	.167
	DASS-S	-.034	.170	-.063	-.201	.842	.022	.169	.042	.133	.896	.039	.172	.073	.229	.821
	SSRT						.038	.024	.287	1.550	.133	.037	.025	.277	1.480	.151
	ERM											-.011	.014	-.133	-.749	.461
Y-BOCS Obs		$R^2 = .186, F(3, 28) = 2.138, p = .188$					$R^2 = .006, F(1, 27) = .191, p = .666$	$R^2 = .003, F(1, 26) = .095, p = .760$								
	DASS-D	-.061	.087	-.183	-.701	.489	-.053	.090	-.161	-.596	.556	-.054	.091	-.163	-.595	.557
	DASS-A	.262	.121	.594	2.161	.039 [*]	.251	.126	.567	1.986	.057 [†]	.252	.129	.571	1.964	.060 [†]
	DASS-S	-.038	.116	-.102	-.331	.743	-.027	.121	-.072	-.224	.824	-.032	.124	-.085	-.259	.797
	SSRT						.008	.017	.082	.437	.666	.008	.018	.087	.451	.656
	ERM										.003	.010	.056	.308	.760	
Y-BOCS Comp		$R^2 = .038, F(3, 28) = .370, p = .775$					$R^2 = .178, F(1, 27) = 6.121, p = .020$	$R^2 = .111, F(1, 26) = 4.278, p = .049$								
	DASS-D	.001	.067	.003	.011	.992	.030	.063	.127	.479	.635	.034	.059	.143	.570	.574
	DASS-A	.057	.094	.180	.604	.551	.010	.089	.031	.110	.913	.002	.084	.006	.021	.983
	DASS-S	.004	.090	.016	.048	.962	.050	.085	.184	.584	.564	.072	.081	.266	.886	.384
	SSRT						.030	.012	.460	2.474	.020 [*]	.029	.012	.434	2.465	.021 [*]
	ERM										-.014	.007	-.345	2.068	.049 [*]	
Y-BOCS Comp		$R^2 = .038, F(3, 28) = .370, p = .775$					$R^2 = .178, F(1, 27) = 6.121, p = .020$	$R^2 = .127, F(2, 25) = 2.416, p = .110$								
	DASS-D	.001	.067	.003	.011	.992	.030	.063	.127	.479	.635	.027	.061	.114	.447	.659
	DASS-A	.057	.094	.180	.604	.551	.010	.089	.031	.110	.913	-.010	.086	-.031	-.112	.911
	DASS-S	.004	.090	.016	.048	.962	.050	.085	.184	.584	.564	.073	.081	.270	.892	.381
	SSRT						.030	.012	.460	2.474	.020 [*]	.030	.012	.456	2.540	.018 [*]
	pSS-ERM										.017	.008	.403	2.153	.041 [*]	
	pSE-ERM										-.010	.008	-.264	-1.295	.207	

Note.

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p < 0.10,
*
**
p < 0.05;
d
/

p < 0.01. Y-BOCS Obs=Obsessional severity, Y-BOCS Comp=Compulsion severity, Y-BOCS Total=Total OCD severity, SSRT=Stop-signal reaction time, DASS-D=Depression severity, DASS-A=Anxiety severity, DASS-S=Stress severity, ERM=Error-monitoring index (= pSE -pSS), pSS-ERM=Successful inhibition [stop-success] reaction monitoring (= pSS - pG), pSE-ERM=Unsuccessful inhibition [stop-error] reaction monitoring (= pSE - pG).