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Utilizing Cognitive Assessment to Predict Exposure and Response Prevention Treatment Response

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UTILIZING COGNITIVE ASSESSMENT TO PREDICT EXPOSURE AND RESPONSE PREVENTION TREATMENT RESPONSE

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ABSTRACT

UTILIZING COGNITIVE ASSESSMENT TO PREDICT EXPOSURE AND RESPONSE PREVENTION TREATMENT RESPONSE

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Exposure and response prevention (ERP) is the first-line treatment for the obsessive-compulsive disorder (OCD). However, a substantial portion of individuals in treatment may not see benefit, and efforts to find predictors of treatment outcomes have been challenging. Response inhibition (RI), which has been linked to OCD symptoms, is theoretically promising as a predictor of ERP treatment outcomes. In this study, we utilized inhibitory capabilities, measured at admission to partial hospitalization programs (PHP) at Rogers Memorial Hospital, to predict treatment outcomes in ERP. We hypothesized that worse performance in RI subdomains of action cancellation, action withholding, and interference control, as well as error-monitoring sub-processes within these domains, would be associated with worse treatment outcomes. Though we did not find overall indices of RI (e.g., stop signal reaction time) were associated with response to ERP, we found that excessive slow down following errors in the context of the stop-signal task was associated with less symptom reduction. On a fast-paced measure of action withholding, longer overall reaction time and slow-down following *successful* inhibition were both associated with worse treatment outcomes. Together, our data suggests that individuals who are less likely to see symptom reduction in ERP show an oscillatory responding style involving slow-down and speed-up at inappropriate times. Our findings may help inform ritual prevention procedures, as well as the feasibility of utilizing cognitive assessment in the clinical context.

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Introduction

Obsessive-Compulsive Disorder (OCD) and Exposure and ritual prevention (ERP)

Obsessive-compulsive disorder (OCD) is a debilitating psychiatric condition characterized by intrusive, unwanted, and/or disturbing mental images or urges (i.e., obsessions), and efforts to reduce anxiety and discomfort from such thoughts with repetitive behaviors or mental acts (i.e., compulsions) (APA, 2013). OCD is quite heterogeneous, and symptom concerns can present in diverse dimensions, including contamination, harm avoidance, obsessions surrounding unacceptable thoughts (e.g., blasphemous thoughts, sexual intrusions), and concerns about symmetry, exactness and/or "just-right" feelings (Abramowitz et al., 2010; McKay et al., 2004).

Exposure and ritual prevention (ERP) is the cognitive-behavioral treatment designed to reduce the frequency and severity of obsessional and compulsional symptoms of OCD. It is the most widely used and effective form of psychological intervention for OCD (for a review: McKay et al., 2015). Originally developed from a case report of two individuals (Meyer, 1966), ERP has demonstrated success in the treatment of OCD. Meta-analyses and treatment trials suggest that ERP procedures are highly effective in reducing the symptoms of OCD in randomized controlled trials, general outpatient treatment settings, and in specialized residential treatment settings (Abramowitz, 1996; Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000; Osgood-Hynes, Riemann, & Björgvinsson, 2003; Rosa-Alcazar, Sanchez-Meca, Gomez-Conesa, & Marin-Martinez, 2008).

A critical component of ERP is a procedure called 'response prevention,' where patients must tolerate the fear associated with interacting with distressing stimuli without engaging in compulsive rituals that would only temporarily reduce anxiety. Effective adherence to response

prevention serves a number of important functions (Foa, Steketee, Grayson, Turner, & Latimer, 1984; Himle & Franklin, 2009; Kircanski & Peris, 2015) and has been associated with treatment success in a number of studies (Farris, McLean, Van Meter, Simpson, & Foa, 2013; Wheaton et al., 2016). Conversely, poor adherence to ritual prevention reinforces the compulsive feedback loop and prevents alternative learning from taking place. Considering the importance of response prevention in ERP, it is critical to understand patient features that interfere with successful implementation of ritual prevention.

Though ERP has been shown to produce clinically significant symptom reduction in numerous clinical trials (Abramowitz, 1997; Olatunji, Davis, Powers, & Smits, 2013; Ponniah, Magiati, & Hollon, 2013; Rosa-Alcazar et al., 2008), there are apparent limitations to the treatment. Primarily, not all who receive ERP achieve wellness. Response rates for ERP are estimated at 50-60% (Fisher & Wells, 2005), and a proportion of those who remit symptoms still do not experience any meaningful change in quality of life (Abramowitz, 1998). Thus, not all who receive the treatment are guaranteed to achieve either symptom remission or wellness.

Because of this, efforts have been made to find treatment predictors for those receiving ERP therapy. Broadly, several classes of predictor variables have been utilized in this effort: a) demographic variables (e.g., age, gender, marital status) (Benazon, Ager, & Rosenberg, 2002; Franklin et al., 2000); b) OCD symptom characteristics (e.g., symptom severity and subtypes of OCD) (Olatunji et al., 2013); c) comorbidity characteristics (e.g., comorbid depression or anxiety) (Abramowitz, 2004; Leonard, Jacobi, Riemann, Lake, & Luhn, 2014); d) cognitive factors (e.g., neuropsychological functioning, insight, thought-action fusion, inflated responsibility) (Bolton, Raven, Madronal-Luque, & Marks, 2000; McLean et al., 2001; Steketee & Shapiro, 1995); e) motivational factors (e.g., treatment expectations) (Reid et al., 2017;

Vorstenbosch & Laposa, 2015); f) therapeutic relationship factors (e.g., therapist alliance) (Vogel, Hansen, Stiles, & Gotestam, 2006); g) biological factors (e.g., patterns of neural activation and biological substrates) (Brody et al., 1998); h) other factors such as personality dysfunction (Bjorgvinsson et al., 2013; Keeley, Storch, Merlo, & Geffken, 2008; Kyrios, Hordern, & Fassnacht, 2015).

Despite strong efforts to find predictors of response to ERP, many of the aforementioned predictors have been unsuccessful in consistently predicting response to ERP procedures (Kyrios et al., 2015). For instance, mixed findings have been reported regarding the predictive utility of demographic variables (Knopp, Knowles, Bee, Lovell, & Bower, 2013; Steketee & Shapiro, 1995), symptom severity and comorbid anxiety and depression (Olatunji et al., 2013), and cognitive factors such as thought-action fusion and insight into symptoms (McLean et al., 2001; Rufer et al., 2006). Non-specific factors such as working alliance and motivation for change have shown mixed results as treatment predictors (Reid et al., 2017; Vogel et al., 2006), as have personality features such as perfectionism (Pinto, Liebowitz, Foa, & Simpson, 2011; Sadri et al., 2017). The underlying neurocircuitry of OCD has recently experienced an explosion of research, particularly in implicating dysfunction in cortico-striatal-thalamic circuits in the manifestation of OCD (Menzies et al., 2008; Pauls, Abramovitch, Rauch, & Geller, 2014). However, these gains in knowledge have not been translated into a change in the core ERP treatment protocol (Graybiel & Rauch, 2000).

Response Inhibition (RI)

Response inhibition (RI) refers to the cognitive ability to stop responses that are inappropriate or no longer required (Verbruggen & Logan, 2009). RI is a core cognitive faculty necessary for inhibiting any deliberate motor response. Inhibitory capabilities are critical for a

variety of executive functions related to memory and attention, thus RI is important for many aspects of an individual's functioning in the real-world (Diamond, 2013). RI is also considered to be a deliberate (i.e., responses are stopped intentionally with top-down executive control) (Eagle, Bari, & Robbins, 2008; Logan, 1994), and ballistic cognitive process (i.e., once a response is initiated it can only be stopped with great difficulty) (Logan, 1994).

RI is not a unitary construct and has been theorized to have three distinct sub-components. First, *action cancellation* refers to the ability to stop an action that is in progress in response to a stop signal (Dambacher et al., 2014; Schachar et al., 2007). Action cancellation most closely fits the broad definition of RI, and real-world analogies of this cognitive process are numerous; a driver who quickly stops in response to a pedestrian jumping into the crosswalk may employ action cancellation faculties. Action cancellation is measured using the stop-signal task (SST).

Second, *action withholding* refers to the inhibition of responses before such responses have been initiated (Eagle et al., 2008). Commonly measured via the go/no-go task (GNG), action withholding is conceptualized as a discriminative inhibitory ability where individuals must attend to a string of stimuli and carefully and selectively inhibit (Gomez, Ratcliff, & Perea, 2007). The GNG is designed to build a prepotent motor response by presenting trials rapidly. Inhibitory capabilities probed by the GNG are thought to reflect underlying impulsivity (de Wit, Enggasser, & Richards, 2002).

Lastly, *interference control* refers to the ability to inhibit irrelevant information and attend to task instructions (Friedman & Miyake, 2004). While this ability may seem more related to information processing and attentional control, good performance in interference control requires active suppression of attendance to irrelevant stimuli and has been associated with

frontal cortical areas necessary for inhibition (Bunge, Ochsner, Desmond, Glover, & Gabrieli, 2001). Interference control is frequently measured by the motor flanker task (FT).

Recent work has expanded on the basic conceptualization of RI to detail patterns of behavioral adjustments following success or failure in inhibition, referred to as post-error behavioral adjustments (PEBAs) (Schroder & Moser, 2014). In healthy populations, PEBAs are adaptive adjustments made to improve performance following stop stimuli. They may manifest as post-error slowing (PES) or error-monitoring (ERM), where reaction time (RT) is elongated following errors compared to successful inhibition trials (Rabbitt, 1966), post-error improvement in accuracy, where individuals evidence improvement in accuracy following errors in inhibition (Hester, Barre, Murphy, Silk, & Mattingley, 2008), or post-error reduction of interference, where the interference effect in flanker tasks is reduced (i.e., greater cognitive control) following errors (Ridderinkhof et al., 2002). These PEBAs are thought to be adaptive in that adjustments following inhibition indicate greater deployment of executive control towards execution of task demands or goals. Rather than a separate and distinct phenomenon, ERM and PEBAs occur within the context of RI in response to errors, thus they can be conceptualized as another sub-process of RI. PEBAs are emerging as a feature of interest in affective neuroscience due to their relevancy in exploring anxiety and other forms of pathological processing (Danielmeier & Ullsperger, 2011).

Response Inhibition and OCD

RI is a relevant cognitive variable in conceptualizing OCD symptomology. Recent research has strongly linked deficient RI processes to OCD symptomology (Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Penades et al., 2007; van Velzen, Vriend, de Wit, & van den Heuvel, 2014). Meta-analytic findings suggest medium-to-large sized deficits in

inhibitory control in individuals with OCD compared to other conditions such as major depressive disorder (MDD) or other anxiety disorders, suggesting unique contributions of inhibitory dysfunction towards OC-symptomology (Lipszyc & Schachar, 2010). This poor RI performance has also been found in individuals with OCD compared to healthy controls, as well as in unaffected first-degree relatives of those with OCD (Chamberlain et al., 2007). Individuals with OCD have been shown to have unique patterns of cortical activation on measures of action restraint (GNG) (Stern & Taylor, 2014). Poor RI capabilities have also been observed in other obsessive-compulsive and related disorders (OCRDs) such as trichotillomania (Bohne, Savage, et al., 2005; Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008).

PEBAs have also been experiencing a growth in research due to their utility in explaining symptoms and neural substrates of anxiety and other pathology (Moser, Moran, Schroder, Donnellan, & Yeung, 2013). Moreover, while PEBAs are adaptive adjustments in healthy populations, they may become skewed or deployed ineffectively in pathological populations. For instance, Balogh and Czobor (2016) found that individuals with attention deficit hyperactivity disorder (ADHD) showed the opposite of ERM, and increased response speed following errors. Some have suggested that the physiological markers of performance monitoring indicate a transdiagnostic vulnerability across anxiety disorders including social anxiety disorder and OCD (Endrass, Riesel, Kathmann, & Buhlmann, 2014). Our previous work studied PES/ERM effects in the context of action cancellation in OCD, and found that those with OCD excessively slow down even after successful inhibition, indicative of a failure in inhibiting an excessive error-monitoring process (Berlin & Lee, 2018). Moreover, PES has been strongly linked to activity in the anterior cingulate cortex (ACC) (Shackman et al., 2011), a brain region that has been implicated in error-detection in OCD (Fitzgerald et al., 2005), and OCD symptomology more

broadly (Maia, Cooney, & Peterson, 2008). The strength of these and other findings have led researchers to suggest that deficient inhibitory abilities may be a neurocognitive endophenotype of OCD and its related disorders (Chamberlain & Menzies, 2009; Menzies et al., 2007; Nakao, Okada, & Kanba, 2014; Odlaug, Chamberlain, Derbyshire, Leppink, & Grant, 2014).

Considering the strength of the RI-OCD relationship, RI faculties may be a potent resource in understanding response to ERP procedures. Firstly, OCD symptomology, marked by repetitive, compulsive efforts to relieve distress, may be a reflection of deficiency in inhibitory processing (Chamberlain & Menzies, 2009). Framed in this way, factors involved in inhibitory control may be critically important in both understanding ERP, and adherence to the potent behavioral inhibition component of ERP. Secondly, success in adherence to ritual prevention requires deliberate and conscious effort to inhibit inappropriate, undesirable behaviors that reinforce obsessional fear. Thus, underlying RI capabilities may affect how one responds to the ritual prevention component of ERP. It is possible that underlying cognitive vulnerabilities, particularly related to RI, may be associated with greater difficulty in implementing successful inhibition of compulsive behavior in the context of ritual prevention. In this way, an individual with poor inhibitory performance may not reap the benefits of treatment because of difficulty in inhibiting inappropriate behaviors.

Central Objective and Specific Aims

In this project, we sought to study how inhibitory control processes were associated with treatment outcomes for individuals with OCD receiving ERP. Patients receiving ERP in partial hospitalization programs (PHP) at Rogers Memorial Hospital (RMH) were recruited at admission and administered a battery of cognitive tests to measure the three broad domains of response inhibition. Treatment outcome was defined as a change in OCD symptoms from

admission (= baseline [BL]) to discharge (=post-treatment [PT]). This study had the following aims and hypotheses:

Aim 1: To examine action cancellation (SST) processes as predictors of poor ERP response for OCD.

Hypothesis 1a: Prolonged stop-signal reaction time (SSRT) at admission will be negatively associated with treatment outcomes.

Hypothesis 1b: Prolonged ERM on the SST at admission will be negatively associated with treatment outcomes.

Aim 2: To examine action withholding (GNG) processes as predictors of poor ERP response for OCD.

Hypothesis 2a: Higher levels of commission errors on the GNG task will be negatively associated with treatment outcomes.

Hypothesis 2b: Prolonged ERM on the GNG task will be negatively associated with treatment outcomes.

Aim 3: To examine interference control (Flanker Task) processes as predictors of poor ERP response for OCD.

Hypothesis 3a: Higher discrepancy scores between reaction time on incongruent and congruent trials on the Flanker Task (e.g., Interference RT) will be negatively associated with treatment outcomes.

Hypothesis 3b: Prolonged reaction time on incongruent trials following congruent trials (Inc-pCon) will be negatively associated with treatment outcomes.

Methods

Participants

Study entry criteria for this study were: a) Ages 18-65; b) Diagnosis of OCD (as made by RMH clinical staff); c) Consent to treatment services at RMH. These inclusion criteria were established to collect a sample from a broad age range who were seeking treatment at RMH partial hospitalization programs (PHP). To increase ecological validity and capture the full

breadth of patients seeking treatment at this level of care, individuals were not required to have a primary diagnosis of OCD to participate.

Forty-nine (n=49) individuals consented to participate in the study. Mean age of our sample was 28.06 (SD = 9.59). The sample was largely similar in gender make-up (female = 52.8%, n= 19; male = 47.2%, n=17). Participants were racially homogenous, with a majority being Caucasian (95.2%, n=20). Patients spent a mean of 37.78 days in treatment (SD=19.88), with 3 individuals quickly discharging after completing BL assessment. Nine individuals were stepped down from a higher level of care such as residential or inpatient treatment (18.37%). Most individuals were recruited from the PHP site in Madison (41.7%, n = 20).[1]

Comorbidities in our sample were diverse. Table 1 details diagnostic status entered as primary, secondary, and tertiary diagnoses by RMH clinical staff. While all participants in our study met criteria for OCD as one of their five diagnoses, 19.4% (n = 7) had an OCRD as their primary diagnosis. Most individuals were diagnosed with a depressive disorder as their primary diagnosis (66.6%, n = 24)

The mean overall OCD symptom severity at admission was 22.31 (SD=6.65), which was in the range of moderate symptom severity (Storch et al., 2015). Mean discharge OCD severity was 16.34 (SD=5.31), thus patients in treatment experienced a 26.76% reduction in overall OCD symptoms. A paired samples t-test showed that this reduction was statistically significant ($t(41) = 6.535$, $p < .001$, Cohen's $d = .992$). Similarly, individuals experienced a 37.52% reduction in depression symptoms from admission (M=13.59, SD=5.37) to discharge (M=8.49, SD=5.05).

[1] Please note, demographic and diagnostic data in this report is incomplete as such data is extracted from the RMH assessment system post-discharge and multiple individuals are still in treatment. Future clinical reports will include a complete accounting of such data.

Data Collection Procedures

Participants in this study completed a battery of cognitive measures to assess RI capabilities in domains of action cancellation, action withholding, and interference control at admission (=baseline [BL]) to RMH PHP centers. Cognitive assessment was completed on a 9.7-inch iPad. Participants also completed a battery of questionnaires that measured OCD severity and depression severity as part of their routine clinical assessment at RMH at admission (=baseline [BL]), discharge (=post-treatment [PT]), and on a bi-weekly basis. Initial cognitive assessment was timed to be as close to admission as possible without interfering with patient adjustment to the programs or with routine clinical practice. Individuals were typically recruited by a therapeutic staff member at their respective site. The following four PHP sites were utilized: a) Oconomowoc, WI; b) Madison, WI; c) Appleton, WI; d) Skokie, IL.

RMH PHP programs are centered on maximizing ERP treatment via a standardized, manualized approach. While enrolled in the PHP programs, individuals received approximately 4-8 weeks of ERP and were routinely monitored for symptom change and improvement. Individuals enrolled in these programs received daily ERP treatment, supervised by a behavioral specialist, who monitored fear activation, ritual prevention adherence, and hierarchy adjustment and advancement. PHP programs were utilized in this project for a) their pronounced emphasis on ERP delivery, b) their adherence to standardized treatment across programs, and c) their ecological validity in providing access to a range of OCD cases with moderate or greater symptom severity that were being seen in a bona-fide clinical environment.

Measures: Computerized Cognitive Assessment

Computerized cognitive assessment probed the three broad domains of RI. *Action cancellation* abilities were assessed using the stop-signal task, *action withholding* was measured

using the go/no-go task, and *interference control* was measured through the flanker task. Assessments were administered through Inquisit software following informed consent procedures. The order of assessments was randomized. A detailed description of each task and calculation of key RI and ERM variables is presented below:

Stop-signal task (SST). The SST presented participants with directional arrows and instructions to respond with the corresponding directional button on the screen. Stop-signals (tone) appeared on 25% of trials after a short delay (stop-signal delay = SSD) to signal inhibition. SSD was initially set at 250ms and was adjusted up/down by 50ms depending on performance to ensure the probability of correctly inhibiting stop-signals was about 50%. Subjects had one 32 trial practice block, followed by three main testing blocks with 64 trials each. Stop-signal reaction time (SSRT), the core index of RI, was calculated by subtracting the mean SSD from the mean reaction time (RT) on go trials (SSRT = mean go RT – mean SSD) (Verbruggen, Logan, & Stevens, 2008). To explore the error-monitoring phenomenon, we computed three mean RTs, consistent with previous work (Li et al., 2008): a) mean RT on go trials following other go trials (post-go trials=pG RT); b) go trials following successful inhibition (post-stop success=pSS RT); c) go trials following failed inhibition (post-stop error=pSE RT). The magnitude of excessive error-monitoring, or slow-down following failed inhibition compared to successful inhibition, was computed by subtracting the mean RT of pSS trials from pSE trials (ERM= pSE RT – pSS RT). To explore fine-grained ERM adjustments following inhibition trials, we computed two additional RT variables using pG as a reference: a) pSE-ERM (=pSE RT – pG RT) signified slow-down following inhibition failure, and b) pSS-ERM (=pSS RT – pG RT) signified slow-down following successful inhibition.

Go/No-Go Task (GNG): The GNG presented participants simple letters that comprised *go* and *no-go* trials. Subjects were instructed to respond to all target letters (*go)* with the response button but inhibit responses when an 'X' appeared on the screen (*no-go*). Subjects completed a practice block (8 trials) followed by a single test block of 140 trials. Two-thirds (66%) of trials were go trials, while the remaining third (33%) were no-go trials. RI in this task was primarily conceptualized via commission errors (CE), where a response key was pressed on a no-go trial. We also computed omission errors (OE), where no key was pressed on a go trial, and overall mean RT on correct go trials (=CrGoRT) (Meule, 2017). Total accuracy percentage (= [total correct trials/total trials] x 100) was computed to check data for overall validity. To explore ERM in the action withholding context, we calculated the following: a) post-stop success (=pSS RT) was calculated by averaging the reaction time on go trials following successful inhibition on no-go trials; b) post-stop error (=pSE RT) was calculated by averaging RT on go trials following failed no-go trials; c) error-monitoring was computed from the difference between the pSE and pSS (ERM = pSE RT – pSS RT). Fine-grained ERM analyses were computed using go-trials following other go-trials (=pG) as a reference: a) pSS-ERM was used to signify excessive slow-down following successful inhibition (=pSS RT – pG RT), and b) pSE-ERM was used to signify excessive slow-down following failure in inhibition (=pSE RT – pG RT).

Flanker Task (FT). The flanker task presented participants with a line of arrows pointing left and right with instructions to locate the central arrow and specify its direction with a response key. The central arrow was flanked by arrows that provided congruent (in the same direction as target) or incongruent (mixed flankers) information. Subjects completed one practice block (12 trials with feedback) and one test block (80 trials, no feedback). Twenty-five percent (25%) of trials used incongruent flankers. RI in this task was conceptualized via two variables: a)

Interference RT (=mean RT on correct incongruent trials – mean RT on correct congruent trials) showed the magnitude of activation in interference control; b) interference error (=number of incongruent errors – number of congruent errors) showed a proportion of interference error. Because this task did not present a stop-stimulus, previous calculations used for ERM were unsuitable. However, we computed four novel indices that differentiated RT depending on the type of stimulus that immediately preceded it. We theorized that these variables would measure relative recruitment of inhibitory capabilities depending on task demands, thereby illustrating additional facets of the interference control process: 1) mean RT on congruent trials following other congruent trials (=Con-pCon RT) indexed a reference condition where inhibitory demands were thought to be lowest; b) RT on congruent trials following incongruent trials (=Con-pInc RT); c) RT on incongruent trials following other incongruent trials (=Inc-pInc RT), and; d) RT on incongruent trials following congruent trials (=Inc-pCon RT). We theorized that the RT reflected in each of these variables would provide insight into the relative recruitment of interference control processes. Further, we theorized that longer RT on Inc-pCon trials would be negatively associated with treatment outcomes because it would capture the immediate activation of such control processes, thereby signifying inhibitory interference effects.

Measures: Questionnaires and Treatment Outcomes

Participants completed questionnaires at admission, discharge, and on a bi-weekly basis as part of their routine clinical care. The Self-Report Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) provided scores for total OCD symptom severity, as well as sub-scores for compulsional and obsessional symptom severity (Federici et al., 2010). The Y-BOCS was the primary outcome measure in this study, and treatment outcome was defined as a change in symptoms from admission to discharge. This was computed as a simple difference score such

that Y-BOCS Total Δ = Y-BOCS BL Total – Y-BOCS PT Total. Change scores were computed for obsessional and compulsional sub-scores in the same way. Treatment outcomes were defined in this way because participants were expected to remain symptomatic after treatment due to their high baseline OCD symptoms. Thus, a cross-sectional snapshot of end-state functioning would not provide an ideal index of treatment outcome and we opted to understand treatment outcomes as a difference score in symptom severity. While some have suggested that change scores can be problematic due to regression to the mean, others have argued that change scores are suitable in non-experimental data if a predictor (X) is temporally subsequent to first measurement of a symptom score (Y_1), as is reflected in our study design (Allison, 1990).

The Obsessive-Compulsive Inventory-Revised (OCI-R) was also used to measure OCD symptoms as a secondary outcome measure (Huppert et al., 2007). The OCI-R directly probes specific domains of obsessions and compulsions, thus we believed it could offer additional utility in detecting change in symptoms. OCI-R outcomes were defined in a similar way such that OCI-R Total Δ = OCI-R BL Total – OCI-R PT Total. Depression was measured via the Quick Inventory of Depressive Symptomology (QIDS) (Rush et al., 2003) and was included in our analyses as a covariate in regression analyses because of the frequent incidence of depression in our sample.

Analysis Strategy

Analyses for this study were conducted in the same way for each specific aim. First, RI and ERM variables in each task were entered in a bivariate correlation with treatment outcome measures to explore the overall relationship between such variables. Then, we utilized hierarchical linear regression to explore how facets of RI could explain change in OCD symptoms. Baseline depression severity (BL QIDS) and time in treatment (TTX) were entered

together in Step 1 of each analysis to control for characteristics that may also explain treatment outcomes, and RI or ERM variables were added in Step 2. Regression models were calculated separately using change scores (Y-BOCS total, obsession, and compulsion, and OCI-R Δ) as dependent variables in the analysis.

Results

Aim 1: To examine action cancellation (SST) processes as predictors of poor ERP response for OCD.

Data selection filters were applied to select only valid cognitive test data in exploring treatment outcomes. We selected data based on the following criteria for the SST: a) More than 80% correct responses on go trials (e.g. "hits"), b) less than 10% omission omissions on go trials (e.g., "misses"), and c) less than 25% deviation from the 50% chance of correctly responding to stop-signals set by the internal tracking algorithm built into the task. In applying this filter, 13 cases (26% of sample) were removed from analysis for Aim 1.

Mean values and correlation amongst action cancellation variables and indices of OCD symptom change are found in Table 2. Mean SSRT latency was 156.1ms (SD=62.34), which is somewhat shorter than SSRT published in previous reports (Berlin & Lee, 2018: M=221, SD=43; Chamberlain et al., 2006: M=211.6, SD=57.9). SSRT was not significantly associated with changes in total Y-BOCS scores ($r(32) = -.045$, $p > .05$), obsessional severity change ($r(32) = -.131$, $p > .05$), compulsional severity change ($r(32) = .032$, $p > .05$), or OCI-R change scores ($r(31) = .035$, $p > .05$). Similarly, mean go RT and SSD were not significantly associated with changes in OCD symptoms.

However, error-monitoring variables showed significant association with changes in OCD symptoms in a few areas. Firstly, pSE-ERM, the amount of excessive slow-down following failed inhibition, was negatively related to Y-BOCS Total Δ ($r(32) = -.389$, $p < .05$), and Y-

BOCS Obsessional Δ ($r(32) = -.483$, $p < .01$), but not to Y-BOCS Compulsion Δ ($r(32) = -.271$, $p > .05$) or OCI-R Total Δ ($r(32) = .112$, $p > .05$). Additionally, ERM, which indicates slow-down following failed inhibition when taking into account pSS RT, was significantly associated with change in Y-BOCS Obsessional Δ ($r(32) = -.370$, $p < .05$), but not with Y-BOCS Total Δ ($r(32) = -.188$, $p > .05$), Y-BOCS Compulsion Δ ($r(32) = -.017$, $p > .05$), or OCI-R Δ ($r(32) = .171$, $p > .05$). pSS-ERM was not significantly related to treatment outcomes.

To evaluate Hypotheses 1a & 1b, we utilized hierarchical linear regression with the following analytic strategy: covariates (baseline depression severity and time in treatment) were added together in Step 1, SSRT was added in Step 2 to evaluate the variance explained in symptom change by RI capabilities (i.e., R^2 Δ), and ERM was added in Step 3 to further evaluate variance explained in treatment outcomes when controlling for other RI capabilities. Our earlier exploratory correlation suggested that ERM was significantly related to some treatment outcomes, thus this regression analysis was constructed in a way to arrange for a stringent test of ERM's predictive utility even after controlling for covariates and other potent RI variables. Results of these regression computations are found in Table 3.

Hypotheses 1a & 1b: Prolonged SSRT and ERM will be negatively associated with treatment outcomes.

Baseline depression severity ($\beta = .124$, $t = .666$, $p = .511$) and time in treatment ($\beta = .052$, $t = .278$, $p = .783$) only explained 2% of the variance in Y-BOCS Total Δ. SSRT ($\beta = -.025$, $t = -.131$, $p = .897$) explained an additional 1.7% variance in total symptoms change, and ERM ($\beta = -.182$, $t = -.952$, $p = .349$) accounted for another 3.2%. However, in predicting change in Y-BOCS Obsessional Δ, while SSRT ($\beta = -.102$, $t = -.557$, $p = .582$) accounted for 1% of the variance, ERM emerged as a predictor with marginal significance ($\beta = -.342$, $t = -1.943$, $p = .062$) and accounted for 11.3% of variance in obsessional symptom change. SSRT explained less

than 1% of variance in Y-BOCS Compulsional Δ (β = .043, t = .225, p = .823) and OCI-R Δ (β = .029, t = .150, p = .882). ERM did not emerge as a significant predictor of Y-BOCS Compulsion Δ (β = -.029, t = -.147, p = .884), but did explain 3.8% of the variance in OCI-R Δ (β = .200, t = 1.020, p = .317).

Regression analyses were repeated using pSS-ERM and pSE-ERM added together in Step 2 to evaluate the variance explained in treatment outcomes via error-monitoring performance. Results of these analyses are found in Table 4. In exploring Y-BOCS Total Δ, pSS-ERM was not a significant predictor (β = -.094, t = -.514, p = .612), but pSE-ERM demonstrated marginal significance as a predictor of overall OCD symptom change (β = -.355, t = -1.910, p = .067). The two variables explained 15% of the variance in these change scores. pSE-ERM emerged as a significant predictor for Y-BOCS Obsessional Δ (β = -.475, t = -2.732, p = .011), but pSS-ERM did not (β = .071, t = .413, p = .683). These two variables accounted for approximately 20% of the variance in change in obsessional symptoms. Neither pSS-ERM nor pSE-ERM were significant predictors of Y-BOCS Compulsion Δ or OCI-R Δ, but they did explain about 12% of the variance in compulsion change.

Overall results from Aim 1 analyses suggests a few things. Firstly, in a moderately severe, treatment-engaged sample, basic RI capabilities as captured by SSRT may not be nuanced enough to capture change in symptoms evoked from ERP procedures. However, reaction time on trials immediately following inhibition reveals that excessive monitoring of one's own performance is a poor prognostic factor for the amount symptom reduction one may experience as part of ERP procedures. Moreover, excessive engagement with performance monitoring following failed inhibition was negatively associated with treatment gains for both overall OCD symptoms and obsessional symptoms.

Aim 2: To examine action withholding (GNG) processes as predictors of poor ERP response for OCD.

Data selection filters were implemented to remove those who made a significant amount omission and commission errors (i.e., 2.5 standard deviations greater than the mean). Doing so removed 4 cases (8.1% of sample). Subject performance on the GNG was overall highly attentive and accurate, with mean total accuracy of 96%, mean commission errors (CE) of 4.09 (SD=3.56), and mean omission errors of 1.24 (SD = 1.35). Because of the high level of accuracy in our sample, post-error RT variables (e.g., pSE-ERM and ERM) should be interpreted with caution as their calculation was drawn from a very small subset of trials. Means, standard deviations, and inter-correlations between action withholding variables and treatment outcomes are found in Table 5.

Correct go RT (CrGoRT) showed a strong negative relationship with OCI-R Δ ($r(38) = -.505$, $p < .01$). This relationship is illustrated in Figure 1. However, CrGoRT was not significantly associated with change in Y-BOCS Total Δ ($r(39) = -.193$, $p > .05$), Y-BOCS Obsessional Δ ($r(39) = -.167$, $p > .05$), or Y-BOCS Compulsional Δ ($r(39) = -.198$, $p > .05$). Larger values in CrGoRT indicate elongated reaction time, and that participants were taking longer to consider the go stimulus before responding. The negative relationship between CrGoRT and OCI-R Δ suggests that a less impulsive responding style (or, an overly inhibited response) is related to smaller gains in treatment. Neither CE's nor OE's were not significantly related to treatment outcomes.

The mean of pSS-ERM was -44.65 ms (SD=38.92). Because this variable indicates slow-down following successful inhibition compared to simple pG RT, a negative value indicates that participants were speeding up following successful inhibition on no-go trials. pSS-ERM was significantly positively related to YBOCS Total Δ ($r(39) = .367$, $p < .05$), Y-BOCS Obsessional

Δ (r(39) = .348, p < .05), and Y-BOCS Compulsional Δ (r(39) = .349, p < .05). It was not significantly related to change in OCI-R Δ (r(38) = .229, p > .05). These findings suggest that an expedited responding style immediately following successful inhibition is a marker for reduced gains in treatment. This relationship is visualized in Figure 2. OE's, total accuracy, pSE-ERM, and ERM were not related to any treatment outcomes, but again these results should be interpreted with caution due to the low rate of errors in our sample.

Hypothesis 2a: Higher levels of commission errors on the GNG task will be negatively associated with treatment outcomes.

To explore how inhibitory capabilities were related to treatment outcomes, we entered relevant covariates (BL QIDS and TTX) in Step 1 of the regression equation and entered CrGoRT and CE's together in Step 2. Results of these analyses are shown in Table 6. Neither CrGoRT (β = -.242, t =-1.206, p = .236) nor CE's (β = -.104, t = -.532, p = .598) were significant predictors of Y-BOCS Total Δ and explained about 3.9% of the variance in this change score. Similarly, CrGoRT and CE explained ~2% of the variance in Y-BOCS Obsessional Δ, and 6.5% of the variance in Y-BOCS Compulsional Δ. Neither CrGoRT nor CE's were significant predictors of obsessional or compulsional symptom change. However, CrGoRT emerged as a significant predictor of OCI-R Δ (β = -.441, t = -2.361, p = .024). While CE's did not predict OCI-R Δ (β = .049, t = .276, p = .784), the two variables explained 19.5% of the variance in change in OCI-R scores.

Hypothesis 2b: Prolonged ERM on the GNG task will be negatively associated with treatment outcomes.

We also computed a regression equation to explore the predictive power of pSS-ERM on treatment outcomes by entering this variable alone in Step 2 of the regression after controlling for covariates. Results of this regression are found in Table 7. We found that pSS-ERM was a

significant predictor of both Y-BOCS Total Δ (β = -.337, t = 2.175, p = .036) and Y-BOCS Compulsion Δ (β = .335, t = 2.101, p = .043), and accounted for about 11% of the variance in each outcome. It also showed marginal significance as a predictor of obsessional change (β = .303, t = 2.005, p = .053).

Findings from the GNG paint a curious picture of inhibitory responding styles as they relate to treatment outcomes. First, an elongated reaction time on simple go trials was negatively associated with overall OCD symptom change (OCI-R), suggesting that an overly-inhibited responding style is associated with poor treatment outcomes. Second, speed-up following successful inhibition was also associated with poor treatment outcomes. Taken together, these findings suggest that a responding style characterized by an oscillatory, unstable inhibitory profile is associated with poor outcomes in ERP treatment.

Aim 3: To examine interference control (Flanker Task) processes as predictors of poor ERP response for OCD.

Flanker task data was cleaned to remove cases that made 20 or more total errors. This removed only three cases (6% of sample). Similar to GNG performance, participants evidenced conscientious and accurate performance on the flanker task. Mean error rate was 1.39 (SD= 2.01). Means, standard deviations, and correlation coefficients of interference control variables with treatment outcomes can be found in Table 8.

Interference RT was not significantly associated with YBOCS Total Δ (r(40) = .003, p > .05), YBOCS Obsessional Δ (r(40) = -.061, p > .05), YBOCS Compulsion Δ (r(40) = .062, p > .05), or OCI-R Δ (r(39) = -.051, p > .05). Similarly, interference error was not related to treatment outcomes, including YBOCS Total Δ (r(40) = -.247, p > .05), YBOCS Obsessional Δ (r(40) = -.220, p > .05), YBOCS Compulsion Δ (r(40) = -.250, p > .05), or OCI-R Δ (r(39) = .101, p > .05).

Hypothesis 3a: Higher discrepancy scores between reaction time on incongruent and congruent trials on the Flanker Task will be negatively associated with treatment outcomes.

Interference RT and interference error were entered together in Step 2 of a regression equation to predict treatment outcomes. Results of these analyses are presented in Table 9. Neither index of interference control emerged as a significant predictor of treatment outcomes. Entered together, these facets of cognitive control explained between 2-6.8% of the variance in symptom change across treatment outcomes. Results suggest that core measures of interference control show limited utility in predicting change in OCD symptoms.

Hypothesis 3b: Prolonged reaction time on incongruent trials following congruent trials (Inc-pCon) will be negatively associated with treatment outcomes

We also explored how behavioral adjustments following congruent and incongruent trials were related to treatment outcomes. We did not find that any of the indices generated to conceptualize increasingly difficult cognitive load were significantly associated with treatment outcomes (Table 8). We entered these variables together in Step 2 of a regression equation after controlling for relevant covariates; none of the behavioral adjustment variables emerged as significant predictors of treatment outcomes (Table 10). These RT indices explained between 1-7% of the variance in treatment outcomes.

Power Analysis

Post-hoc power analysis was calculated with G*Power to compute achieved power given computed effect size, alpha, sample size (Faul, Erdfelder, Buchner, & Lang, 2009). For Aim 1, with a computed effect size for our main finding (f^2=.17), an alpha error probability of 0.05, sample size of n=31, and 2 tested predictors while controlling for two covariates, our maximum power was estimated at 0.47. For Aim 2, our main finding had a computed effect size of f^2=.14, a sample size of n=37, and 2 tested predictors while controlling for two covariates, thus total

power for our main Aim 2 finding was 0.40. Thus, total power for the primary findings in our analyses were estimated between 0.40-0.47.

Discussion

ERP, the first-line treatment for OCD, is effective in reducing symptom severity for many individuals with OCD, but either fails to help reduce symptoms or promote good quality of life in up to 40-50% of those who undergo treatment. This project examined how inhibitory capabilities were related to treatment outcomes for individuals receiving ERP at partial hospitalization programs (PHP) at Rogers Memorial Hospital (RMH). We recruited individuals with OCD receiving ERP at four PHP centers in the Wisconsin and Illinois area and administered a battery of computerized cognitive tests to probe inhibitory capabilities. We theorized that worse RI performance at admission would be associated with poor treatment outcomes due to difficulty inhibiting rituals in the context of treatment. Treatment outcomes were defined as a change in symptoms from admission to discharge.

Aim 1: To examine action cancellation processes as predictors of poor ERP response for OCD.

We hypothesized that elongated SSRT at admission would predict treatment outcomes in the ERP context. We did not find that SSRT was a predictor of change in overall OCD symptoms, or in compulsional or obsessional change as part of ERP treatment. We also found a notably shorter SSRT latency in our sample compared to what has been presented in previous published reports. Thus, we did not find support for this hypothesis.

There are numerous reasons for this discrepant finding. Particularly, the sample studied in this project is different in key ways from RI studies that use controlled samples free of comorbidities (Penades et al., 2007), seek medication stabilization prior to assessment (Bohne, Keuthen, Tuschen-Caffier, & Wilhelm, 2005), or utilize undergraduate students with elevated

symptoms (Abramovitch, Shaham, Levin, Bar-Hen, & Schweiger, 2015). In contrast, our sample was comprised of individuals with elevated symptomology, multiple diverse comorbidities including personality dysfunction and substance use, varied baseline experience with ERP, and a medication regimen that was being adjusted as part of treatment. While the bona-fide nature of this clinical sample increases the generalizability of results, the complex nature of their clinical presentation can muddy what would otherwise be a clean connection between RI performance and treatment outcomes. Additionally, the observed shorter SSRT may be an artifact of our measurement device; the SST is typically administered with an analogue keyboard and monitor, and we are not aware of other projects that have implemented the task with a touchscreen iPad in a real-world clinical setting. Thus, while we did not specifically find evidence to support *Hypothesis 1a*, we believe that multiple contextual factors may account for this null finding. Further systematic investigation is needed to better understand when inhibitory deficits are likely to be detected in OCD.

We also hypothesized that excessive error-monitoring would be negatively associated with treatment outcomes. We found marginal significance for the predictive utility of ERM in explaining change in obsessional symptoms. This overall excessive error-monitoring explained about 11.3% of the variance in change in obsessional symptoms as part of ERP treatment. To expand on this finding, we bisected the ERM variable to conceptualize excessive monitoring following successful and unsuccessful inhibition and found that performance monitoring following errors was significantly negatively related to obsessional change. In other words, the more one slows down following failed inhibition, the less one is expected to make gains in obsessive symptomology during treatment. This post-error slow-down accounted for approximately 20% of the variance in obsessional change in treatment.

This finding is notable in demonstrating that post-error behavioral adjustments have predictive power in explaining obsessional symptom change as part of ERP treatment. Obsessions in OCD have been conceptualized as a covert and difficult to manage component of the disorder (Belloch, Carrio, Cabedo, & Garcia-Soriano, 2015), and some obsessional subtypes (e.g., violence obsessions) have been related to higher rates of suicide (Ching, Williams, & Siev, 2017). Additionally, research has suggested that obsessions reflect failure in cognitive inhibition and thought suppression, while compulsions reflect breakdown in behavioral or motoric inhibition (Harsanyi et al., 2014). Theoretically, our finding implies that post-error behavioral adjustments tap into a cognitively-based OCD process that has been identified as notably difficult to manage.

Practically, these findings point towards a potentially useful therapeutic adjustment that can address obsessional change. If an OCD patient who tends to slow down following errors is on track to make poor gains in obsessional symptom reduction, then it follows that a therapist needs to implement techniques immediately following exposure to disengage any invisible ruminative processes on perceived errors. This is obviously easier said than done, but some research suggests that mindfulness skills can be temporarily effective in disengaging ruminative processes (Hilt & Pollak, 2012). Disengagement from the exposure and fear process runs the risk of becoming distraction or avoidance (Gillihan, Williams, Malcoun, Yadin, & Foa, 2012), thus excellent clinical judgement is required to determine when mindfulness or distraction are needed in the case of excessive error-monitoring. This area is fruitful for future work exploring how ERP outcomes can be augmented for individuals who are not expected to make gains in obsessional severity.

Aim 2: To examine action withholding processes as predictors of poor ERP response for OCD.

We theorized that higher levels of commission errors in the context of a fast-paced test of action withholding would be negatively associated with treatment outcomes. Additionally, we hypothesized that reaction time on go trials following errors in the context of action withholding would predict poor treatment outcomes. The overall performance accuracy of our sample was quite high, thus we observed a floor effect in total commission errors (participants only made about four commission errors in 140 trials). As such, we did not have variance broad enough to detect support for *Hypotheses 2a & 2b*. Considering the low error rate in our sample, accuracy-based analyses were limited but RT-based analyses were promising in exploring treatment outcomes.

The likely scenario is that our task was too easy, thus even participants with significant psychopathology were able to perform quite well. Danielmeier and Ullsperger (2011) suggest that exploration of PES/ERM effects requires a well-distributed spread of errors evoked in tasks that do not systematically adjust difficulty based on subject performance, thus a dataset with a low error rate may not be an ideal context to study PEBAs. Certainly, compared to the SST task, the GNG does not adapt difficulty based on performance thus a lower proportion of errors was somewhat expected. To detect RI and ERM effects in the context of action withholding, task parameters should be made more difficult. This can be accomplished by increasing the rate of stimuli presentation to aggravate the pre-potent motor response process, reducing the time allotted to respond to stimuli, or presenting a more complex set of rules to trigger inhibition.

We did discover that the overall reaction time on go trials in the action withholding context was significantly negatively related to overall OCD symptom change. This slow and methodical responding style accounted for approximately 20% of the variance in change OCD

symptoms from admission to discharge. These results suggest that the more time an individual takes to consider a stimulus before responding, the less likely they are to see gains in treatment. When reaction times are short, they are typically considered to represent an "approach tendency" (Meule, 2017; Meule et al., 2014) or indicate an underlying impulsivity (Andreou et al., 2007). As impulsivity and compulsivity have been theorized to lie on a continuum (Chamberlain, Leppink, Redden, & Grant, 2016), an elongated go RT in this context could illustrate an over-inhibited approach tendency where stimuli are evaluated carefully with caution for risk of erroneous responding. This finding is congruent with models of OCD that emphasize harm-avoidance as a risk factor for OCD symptoms in both patients and their first-degree relatives (Ettelt et al., 2008). One may reasonably ask why an elongated RT was found in the GNG task but not SST, and why that RT was related to treatment outcomes. The GNG task, in comparison to the SST, is a fast-paced measure of inhibitory control where the lack of a stop-signal delay alters the way in which a participant executes the task, and the generally slower RT on go trials are likely to reflect an overly inhibited response style (especially among individuals suffering from psychopathology of OCD). Thus, while they are both grouped under an umbrella RI domain (Bender, Filmer, Garner, Naughtin, & Dux, 2016), they may operate from discernable inhibitory subdomains (Littman & Takacs, 2017).

In accounting for reaction time following successful inhibition trials on the GNG task, we found two interesting facets of RI performance. First, we found that compared to normal go-trials, participants showed RT speed-up after successfully inhibiting responses on no-go trials (e.g., negative RT value). This RT speedup reflects disinhibition. Second, we found that that this speedup (negative value) was positively associated with symptom change indices for overall OCD symptoms, as well as compulsion and obsessional severity. Essentially, this means that

speed-up following successful inhibition (i.e., a shift towards disinhibition) was associated with *less* gains in OCD symptoms as part of ERP treatment.

Synthesizing our two main findings that a) slow-down following failed inhibition (e.g., error-monitoring) in the action cancellation context was associated with poor gains in symptoms, and the converse that b) speed-up following successful inhibition in the action withholding context is also associated with poor gains in symptoms, we interpret these findings to suggest that individuals with OCD who are likely to see poor treatment outcomes in ERP may evidence an unstable inhibitory responding style. To illustrate, such an individual may show the following pattern of reaction times in relation to deliberate inhibition: after making an error on a ballistic motor response, they slow-down excessively to prevent future error, but after successfully inhibiting a prepotent and simple response, they quickly try and transition towards the next objective. In this way, the cognitive profile of a poor treatment responder is one of an oscillatory and unstable inhibitory mode where both over-inhibition and disinhibition collide and interfere with treatment outcomes. Our data do not allow us to draw conclusions as to whether these phenomena co-occur within the same individual (i.e., RT in flux within a subject), or whether there are independent types of slow-down and speed-up. Future studies should employ RT variability paradigms and multi-level modeling techniques to examine how RT changes within a task in reaction to stop signals.

These findings have significant implications for detection of treatment non-responders. Particularly, because predictors of poor treatment outcomes have been difficult to locate (Kyrios et al., 2015), these findings are notable in identifying an OCD-relevant, and easily measurable, cognitive domain that is related to poor outcomes in ERP. Moreover, our findings suggest that an

objective measure of inhibition, taken at baseline, can significantly account for the lack of gains made in a validated treatment.

Findings from the GNG may also lead to practical considerations to augment ERP procedures. Similar to what we found in the domain of action cancellation, a slow and methodical rate of response (e.g., overall go RT), or a response style in which error-monitoring systems are activated, was associated with poor treatment outcomes. However, considering that those with worse treatment outcomes also evidenced the opposite responding style (of disinhibition), critical therapeutic intervention is needed to stabilize this oscillatory inhibition network. Practically, a therapist would need keen attention towards a patient's engagement with exposure exercises to a) teach them disengagement skills when excessive-error monitoring networks are activated, and b) have them slow down and engage with exposure more thoroughly when rushing past successfully completed activities. We would liken this to a cognitive metronomic adjustment where "dragging" and "rushing" after exposures are carefully attenuated depending on what behavior a patient is demonstrating. This theoretical conjecture can greatly be augmented with further research in discrete timing of brain events to directly measure activation and oscillation of brain areas linked to error-monitoring processes (Menon, 2012), and drawing on theory from accounts of mental chronometry to explore RT variability to probe if this rushing/dragging phenomenon can manifest within a single subject (Medina, Wong, Diaz, & Colonius, 2015).

Aim 3: To examine interference control processes as predictors of poor ERP response for OCD.

We also sought to explore how interference control capabilities were related to treatment outcomes. Unfortunately, RI capabilities in the domain of interference control, and behavioral adjustments therein, were not significantly related to change in OCD symptoms. In the domain of

RI, interference control is comparatively less studied in relation to OCD symptoms. Typically, action cancellation and withholding abilities are utilized to explore OC-phenomenology (Menzies et al., 2007), thus it could be that measures of cognitive interference are less relevant for these types of concerns than capabilities of motoric inhibition. We did also utilize an abbreviated version of the task (a single 80 trial block) as to not occupy additional treatment time, thus there may have not been enough trials to sufficiently evoke a reliable estimate of interference RT.

There are notable strengths to highlight in this study. Primarily, this is the first investigation to utilize measures of RI to predict treatment outcomes in a partial hospitalization setting. Moreover, we are not aware of other studies employing computerized cognitive assessment via touch-screen tablet computers in bona-fide specialty anxiety clinics. The novelty and promise of useful data from this endeavor show that such a mode of assessment is a promising avenue for expansion in these settings, and that data from experimental computerized cognitive paradigms could be useful for clinical purposes. Data from future studies employing tablet-based assessment may be valuable in uncovering additional treatment-relevant cognitive abilities, and in further elucidating the nature of RI as it relates to clinical pathology.

Additionally, this project expanded conceptualizations of basic RI variables (i.e., SSRT, CE's, and OE's) to incorporate indices of error-monitoring in a translational clinical setting. Previous work exploring RI capabilities in OCD largely use SSRT as the main variable of interest (Chamberlain et al., 2006; Lipszyc & Schachar, 2010), but our work showed that such indices can be expanded to account for additional phenomenological aspects of OCD. Moreover, broadening RI conceptualization to incorporate PEBA phenomenon may allow future studies to conduct in-depth investigation of continuous performance within cognitive tasks.

There are also important shortcomings in our study to address. Notably, our overall sample size was small (n=49), thus our statistical tests are limited in power. Additionally, without a healthy control sample to reference, it is difficult to ultimately state whether error-monitoring observed in our sample is truly excessive or pathological. However, this study will continue to accrue subjects and ultimately compare cognitive performance to age and gender-matched healthy control subjects.

While all participants in the study were diagnosed with OCD, very few had a primary diagnosis of OCD and many had comorbid depression which can contribute to reaction times via psychomotor slowing (Schlosser et al., 2013). While recruitment from clinical centers bolsters the ecological validity of our data, it comes at the risk of adding complexity to parsing out etiologies of cognitive deficits. OCD symptoms were measured via self-report in this study; while the self-report version of the Y-BOCS has shown good convergent validity with the clinical administered version, the self-administered version may underrate symptoms (Storch et al., 2017). While we utilized a difference score to capture symptom change, this static difference score may not have adequately considered baseline symptom scores in our sample. Lastly, our study was focused on predicting acute outcomes in intensive treatment programs. While long-term treatment outcome prediction was out of the scope of this project, future studies should utilize RI variables in the context of long-term prediction to examine how inhibitory deficits carry the effects of treatment forward.

Nonetheless, we feel that these data will inform the RI literature about the clinical utility of this cognitive construct. Error-monitoring and performance on these tests of inhibition are promising targets to uncover the heterogeneous response to ERP procedures. Future studies can expand on these results in a number of ways. For instance, baseline RI capabilities should be

measured in structured clinical trials where primary diagnoses are homogenous and treatment dose is fixed. Additionally, future studies can compare these results against age and gender matched healthy controls to examine the divergence of ERM performance compared to clinical populations. Lastly, this type of assessment should also be employed at other levels of treatment (i.e., residential and intensive outpatient) to see if RI capabilities can still account for treatment outcomes in populations with varied severity.

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Appendix – Tables

Table 1. Demographic characteristics of study sample.

Age [M(SD)]	28.06 (9.59)		
Sex			
Male	47.2% (n = 17)		
Female	52.8% (n = 19)		
Race			
Caucasian	95.2% (n = 20)		
African American	4.8% (n = 1)		
Hispanic/Latino	2.7% (n = 1)		
Time in treatment [M(SD)]	37.78 days (19.88)		
Previous Level of Care		Study Site	
No treatment	6.1% (n = 3)	Oconomowoc	27.1% (n = 13)
Outpatient treatment	60.4% (n = 29)	Madison	41.7% (n = 20)
IOP	4.2% (n = 2)	Skokie	27.1% (n = 13)
PHP	10.4% (n = 5)	Appleton	4.2% (n = 2)
Residential Treatment	10.4% (n = 5)		
Inpatient Hospitalization	8.3% (n = 4)		
Comorbidity	Primary Diagnosis	Secondary Diagnosis	Tertiary Diagnosis
OCD	19.4% (n = 7)	41.4% (n = 12)	60.0% (n = 12)
MDD	66.6% (n = 24)	10.3% (n = 3)	5.0% (n = 1)
SOP	2.8% (n = 1)	17.2% (n = 5)	5.0% (n = 1)
GAD		17.2% (n = 5)	15.0% (n = 3)
PD		6.9% (n = 2)	
SUD	11.1% (n = 4)	3.4% (n = 1)	
PTSD			10.0% (n = 2)
ED			5.0% (n = 1)
Other		3.4% (n = 1)	

Note. Abbreviations: IOP = Intensive Outpatient Program, PHP = Partial Hospitalization Program, OCD = Obsessive Compulsive Disorder, MDD = Major Depressive Disorder, SOP = Social Phobia, GAD = Generalized Anxiety Disorder, PD = Panic Disorder, SUD = Substance Use Disorder, PTSD = Post-Traumatic Stress Disorder, ED = Eating Disorder.

Table 2. Means and standard deviations of action cancellation variables, and their zero-order correlations with OCD symptom change indices.

Measure	M(SD)	1	2	3	4	5	6	7	8	9	10
1. SSRT (ms)	156.1 (62.34)	-	-	-	-	-	-	-	-	-	-
2. goRT (ms)	721.77 (126.21)	-.465**	-	-	-	-	-	-	-	-	-
3. SSD (ms)	564.96 (165.47)	-.733***	.943***	-	-	-	-	-	-	-	-
4. pG RT (ms)	711.29 (129.0)	-.445**	.995***	.932***	-	-	-	-	-	-	-
5. pSS-ERM (ms)	38.41 (59.57)	-.178	-.076	.008	-.157	-	-	-	-	-	-
6. pSE-ERM (ms)	58.61 (69.08)	.012	-.204	-.159	-.275	.300	-	-	-	-	-
7. ERM (ms)	20.21 (76.51)	.150	-.125	-.150	-.126	-.508**	.670***	-	-	-	-
8. YBOCS Total Δ	7.03 (6.57)	-.045	.204	.176	.233	-.201	-.389*	-.188	-	-	-
9. YBOCS Obs Δ	3.88 (3.17)	-.131	.159	.172	.186	-.075	-.483**	-.370*	.936***	-	-
10. YBOCS Comp Δ	3.16 (3.77)	.032	.221	.162	.250	-.288	-.271	-.017	.955***	.790***	-
11. OCI-R Δ	7.29 (8.08)	.035	-.174	-.149	-.166	-.093	.112	.171	.326	.210	.393*

Note. *p<.05; **p<.01, ***p<.001; ms = millisecond.
Abbreviations: SSRT = Stop-signal reaction time, goRT = Mean go-trial reaction time, SSD = Stop-signal delay, pG RT = Mean reaction time on go trials following go trials, pSS-ERM = Successful inhibition [stop-success] reaction monitoring, pSE-ERM = Unsuccessful inhibition [stop-error] reaction monitoring, ERM = Overall error-monitoring index (= pSE – pSS), Y-BOCS = Yale-brown obsessive compulsive scale (primary outcome measure), OCI-R = Obsessive-compulsive inventory revised (secondary outcome measure), Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Table 3. Utilizing linear regression to predict change in OCD symptom scores using action cancellation indices.

		Step 1					Step 2					Step 3				
DV	**IV**	***B***	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
		R^2 = .020, F(2, 29) = .295, p = .747					$R^2\Delta$ = .001, FΔ (1, 28) = .017, p = .897					$R^2\Delta$ = .032, FΔ (1, 27) = .907, p = .349				
	BL QIDS	.156	.234	.124	.666	.511	.153	.240	.121	.639	.528	.127	.242	.101	.525	.604
Y-BOCS Total Δ	TTX	.021	.074	.052	.278	.783	.020	.076	.049	.257	.799	.027	.076	.068	.355	.725
	SSRT						-.003	.020	-.025	-.131	.897	.000	.020	-.003	-.017	.986
	ERM											-.016	.016	-.182	-.952	.349
		R^2 = .068, F(2, 29) = 1.062, p = .359					$R^2\Delta$ = .010, FΔ (1, 28) = .311, p = .582					$R^2\Delta$ = .113, FΔ (1, 27) = 3.776, p =.062				
	BL QIDS	.157	.110	.258	1.425	.165	.151	.112	.249	1.348	.188	.127	.108	.209	1.183	.247
Y-BOCS Obs Δ	TTX	.003	.035	.015	.084	.934	.001	.035	.004	.020	.984	.008	.034	.040	.225	.824
	SSRT						-.005	.009	-.102	-.557	.582	-.003	.009	-.062	-.351	.729
	ERM											-.014	.007	-.342	-1.943	.062†
		R^2 = .006, F(2, 29) = .087, p = .917					$R^2\Delta$ = .002, FΔ (1, 28) = .051, p = .823					$R^2\Delta$ = .001, FΔ (1, 27) = .022, p = .884				
	BL QIDS	-.001	.135	-.002	-.009	.993	.002	.138	.002	.013	.990	-.001	.142	-.001	-.004	.997
Y-BOCS Comp Δ	TTX	.018	.043	.077	.414	.682	.019	.044	.082	.429	.671	.019	.045	.085	.435	.667
	SSRT						.003	.012	.043	.225	.823	.003	.012	.046	.237	.814
	ERM											-.001	.010	-.029	-.147	.884
		R^2 =.019, F(2, 28) = .274, p = .762					$R^2\Delta$ = .001, FΔ (1, 27) = .023, p = .882					$R^2\Delta$ = .038, FΔ (1, 26) = 1.04, p = .317				
	BL QIDS	.170	.307	.104	.554	.584	.169	.313	.103	.542	.593	.226	.318	.138	.713	.482
OCI-R Δ	TTX	-.049	.093	-.100	-.531	.600	-.049	.095	-.098	-.513	.612	-.056	.095	-.112	-.585	.564
	SSRT						.004	.026	.029	.150	.882	-.001	.027	-.006	-.033	.974
	ERM											.021	.021	.200	1.020	.317

Note. †p<.10, *p<.05; **p<.01, ***p<.001.

Abbreviations: BL QIDS = Admission depression severity, TTX = Time in treatment, SSRT = Stop-signal reaction time, ERM = Overall error-monitoring index (= pSE – pSS), Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Table 4. Utilizing linear regression to predict change in OCD symptom scores with action cancellation error-monitoring sub-indices

		Step 1					Step 2				
DV	**IV**	***B***	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
Y-BOCS Total Δ		R^2 = .020, F(2, 29) = .295, p = .747					R^2 Δ = .150, FΔ (2, 27) = 2.432, p = .107				
	BL QIDS	.156	.234	.124	.666	.511	.072	.227	.057	.316	.755
	TTX	.021	.074	.052	.278	.783	.028	.071	.071	.399	.693
	pSS-ERM						-.010	.020	-.094	-.514	.612
	pSE-ERM						-.034	.018	-.355	-1.910	.067†
Y-BOCS Obsession Δ		R^2 = .068, F(2, 29) = 1.062, p =.359					R^2 Δ = .205, FΔ (2, 27) = 3.805, p = .035*				
	BL QIDS	.157	.110	.258	1.425	.165	.108	.103	.177	1.049	.303
	TTX	.003	.035	.015	.084	.934	.010	.032	.050	.298	.768
	pSS-ERM						.004	.009	.071	.413	.683
	pSE-ERM						-.022	.008	-0.475	-2.732	.011*
Y-BOCS Compulsion Δ		R^2 = .006, F(2, 29) = .087, p =.917					R^2 Δ = .124, FΔ (2, 27) = 1.917, p = .166				
	BL QIDS	-.001	.135	-.002	-.009	.993	-.036	.133	-.050	-.269	.790
	TTX	.018	.043	.077	.414	.682	.019	.042	.082	.451	.656
	pSS-ERM						-.014	.012	-.224	-1.192	.244
	pSE-ERM						-.012	.011	-.219	-1.151	.260
OCI-R Δ		R^2 = .019, F(2, 28) = .274, p = .762					R^2 Δ = .039, FΔ (2, 26) = .536, p = .591				
	BL QIDS	.170	.307	.104	.554	.584	.232	.323	.142	.719	.479
	TTX	-.049	.093	-.100	-.531	.600	-.055	.095	-.111	-.581	.566
	pSS-ERM						-.019	.027	-.143	-.718	.479
	pSE-ERM						.022	.024	.187	.916	.368

Note. †p<.10, *p<.05; **p<.01, ***p<.001.

Abbreviations: BL QIDS = Admission depression severity, TTX = Time in treatment, SSRT = Stop-signal reaction time, pSS-ERM = Successful inhibition [stop-success] reaction monitoring, pSE-ERM = Unsuccessful inhibition [stop-error] reaction monitoring, Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Table 5. Means, standard deviations, and zero-order correlation of action withholding variables and indices of OCD symptom change.

Measure	M(SD)	1	2	3	4	5	6	7	8	9	10
1. CrGoRT	495.82 (57.18)	-	-	-	-	-	-	-	-	-	-
2. CE	4.09 (3.56)	-.548***	-	-	-	-	-	-	-	-	-
3. OE	1.24 (1.35)	.301*	-.090	-	-	-	-	-	-	-	-
4. Total Accuracy	0.96 (0.03)	.396**	-.923**	-.284	-	-	-	-	-	-	-
5. pSS-ERM	-44.65 (38.92)	-.331*	.175	-.261	-.069	-	-	-	-	-	-
6. pSE-ERM	49.92 (104.86)	.138	-.348*	-.141	.354*	.187	-	-	-	-	-
7. ERM	93.38 (105.12)	.255	-.424*	-.006	.369*	-.200	.925***	-	-	-	-
8. YBOCS Total Δ	7.03 (6.70)	-.193	-.024	-.120	.126	.367*	.098	-.035	-	-	-
9. YBOCS Obs Δ	3.92 (3.37)	-.167	.021	-.120	.079	.348*	.106	-.031	.944***	-	-
10. YBOCS Comp Δ	3.10 (3.68)	-.198	-.063	-.108	.157	.349*	.081	-.035	.953***	.801***	-
11. OCI-R Δ	6.61 (8.28)	-.505**	.284	-.254	-.151	.229	.199	.163	.364*	.261	.423**

Note. *p<.05; **p<.01, ***p<.001.
Abbreviations: CrGoRT = Mean RT on correct go trials, CE = Commission errors, OE = Omission Errors, pSS-ERM = Successful inhibition [stop-success] reaction monitoring, pSE-ERM = Unsuccessful inhibition [stop-error] reaction monitoring, ERM = Overall error-monitoring index (= pSE – pSS), Y-BOCS = Yale-brown obsessive compulsive scale (primary outcome measure), OCI-R = Obsessive-compulsive inventory revised (secondary outcome measure), Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Table 6. Predicting change in OCD symptoms with indices of action withholding.

		Step 1					Step 2				
DV	**IV**	***B***	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
Y-BOCS Total Δ		R^2 = .067, F(2, 36) = 1.285, p = .289					R^2 Δ = .039, FΔ (2, 34) = .732, p = .488				
	BL QIDS	.306	.198	.250	1.548	.130	.274	.206	.224	1.330	.192
	TTX	-.032	.060	-.085	-.528	.601	-.007	.064	-.019	-.107	.916
	CrGoRT						-.028	.023	-.242	-1.206	.236
	CE						-.204	.383	-.104	-.532	.598
Y-BOCS Obsession Δ		R^2 = .126, F(2, 36) = 2.590, p =.089					R^2 Δ = .019, FΔ (2, 34) = .379, p = .687				
	BL QIDS	.212	.096	.344	2.200	.034	.212	.101	.343	2.088	.044*
	TTX	-.022	.029	-.116	-.743	.462	-.012	.032	-.066	-.389	.700
	CrGoRT						-.008	.011	-.141	-.717	.478
	CE						.011	.189	.011	.057	.955
Y-BOCS Compulsion Δ		R^2 = .021, F(2, 36) = .387, p =.684					R^2 Δ = .065, FΔ (2, 34) = 1.209, p = .311				
	BL QIDS	.094	.111	.140	.845	.404	.062	.114	.092	.544	.590
	TTX	-.010	.034	-.049	-.293	.771	.005	.036	.027	.153	.880
	CrGoRT						-.020	.013	-.312	-1.534	.134
	CE						-.214	.213	-.200	-1.007	.321
OCI-R Δ		R^2 = .085, F(2, 35) = 1.623, p = .212					R^2 Δ = .195, FΔ (2, 33) = 4.470, p = .019*				
	BL QIDS	.188	.255	.119	.736	.466	.198	.236	.126	.839	.407
	TTX	-.123	.074	-.268	-1.654	.107	-.050	.073	-.108	-.682	.500
	CrGoRT						-.062	.026	-.441	-2.361	.024*
	CE						.123	.444	.049	.276	.784

Note. †p<.10, *p<.05; **p<.01, ***p<.001.
Abbreviations: CrGoRT = Mean RT on correct go trials, CE = Commission errors, BL QIDS = Admission depression severity, TTX = Time in treatment, Y-BOCS = Yale-brown obsessive compulsive scale (primary outcome measure), OCI-R = Obsessive-compulsive inventory revised (secondary outcome measure), Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Table 7. Predicting change in OCD symptoms with behavioral adjustments in the context of action withholding.

		Step 1					Step 2				
DV	**IV**	***B***	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
Y-BOCS Total Δ		R^2 = .178, F(2, 36) = 1.285, p = .289					R^2 Δ = .111, FΔ (1, 35) = 4.730, p = .036*				
	BL QIDS	.306	.198	.250	1.548	.130	.250	.190	.204	1.318	.196
	TTX	-.032	.060	-.085	-.528	.601	-.024	.057	-.064	-.416	.680
	pSS-ERM						.059	.027	.337	2.175	.036*
Y-BOCS Obsession Δ		R^2 = .126, F(2, 36) = 2.590, p =.089					R^2 Δ = .090, FΔ (1, 35) = 4.021, p = .053†				
	BL QIDS	.212	.096	.344	2.200	.034	.187	.093	.303	1.998	.054
	TTX	-.022	.029	-.116	-.743	.462	-.018	.028	-.097	-.646	.523
	pSS-ERM						.027	.013	.303	2.005	.053†
Y-BOCS Compulsion Δ		R^2 = .021, F(2, 36) = .384, p =.684					R^2 Δ = .110, FΔ (1, 35) = 4.416, p = .043*				
	BL QIDS	.094	.111	.140	.845	.404	.064	.107	.094	.592	.558
	TTX	-.010	.034	-.049	-.293	.771	-.006	.032	-.028	-.174	.863
	pSS-ERM						.032	.015	.335	2.101	.043*
OCI-R Δ		R^2 = .085, F(2, 35) = 1.623, p = .212					R^2 Δ = .041, FΔ (1, 34) = 1.602, p = .214				
	BL QIDS	.188	.255	.119	.736	.466	.127	.258	.081	.495	.624
	TTX	-.123	.074	-.268	-1.654	.107	-.120	.074	-.261	-1.628	.113
	pSS-ERM						.044	.035	.207	1.266	.214

Note. †p<.10, *p<.05; **p<.01, ***p<.001.
Abbreviations: BL QIDS = Admission depression severity, TTX = Time in treatment, pSS-ERM = Successful inhibition [stop-success] reaction monitoring, Y-BOCS = Yale-brown obsessive compulsive scale (primary outcome measure), OCI-R = Obsessive-compulsive inventory revised (secondary outcome measure), Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Table 8. Means, standard deviations, and zero-order correlations of interference control variables with indices of OCD symptom change.

Measure	M(SD)	1	2	3	4	5	6	7	8	9
1. Interference RT	91.96 (85.58)	-	-	-	-	-	-	-	-	-
2. Interference Error	.70 (1.76)	0.255	-	-	-	-	-	-	-	-
3. Con-pCon RT	635.11 (122.73)	0.066	-.506***	-	-	-	-	-	-	-
4. Con-pInc RT	652.32 (128.45)	0.118	-.481***	.963***	-	-	-	-	-	-
5. Inc-pInc RT	722.26 (151.61)	.509***	-0.213	.736***	.762***	-	-	-	-	-
6. Inc-pCon RT	731.78 (159.70)	.597***	-0.267	.829***	.842***	.822***	-	-	-	-
7. YBOCS Total Δ	7.53 (7.07)	0.003	-0.247	-0.042	0.002	-0.005	-0.048	-	-	-
8. YBOCS Obs Δ	4.20 (3.55)	-0.061	-0.220	-0.083	-0.013	-0.075	-0.101	.952***	-	-
9. YBOCS Comp Δ	3.33 (3.85)	0.062	-0.250	-0.001	0.016	0.060	0.006	.959***	.826***	-
10. OCI-R Δ	7.00 (8.39)	-0.051	0.101	-0.190	-0.235	-0.148	-0.205	.396*	0.305	.448**

Note. †p<.10, *p<.05; **p<.01, ***p<.001.

Abbreviations: Interference RT = Interference control reaction time index (=mean reaction time on incongruent trials – mean reaction time on congruent trials), Con-pCon RT = Mean reaction time on congruent trials following congruent trials, Con-pInc RT = Mean reaction time on congruent trials following incongruent trials, Inc-pInc RT = Mean reaction time on incongruent trials following incongruent trials, Inc-pCon RT = Mean reaction time on incongruent trials following congruent trials, Y-BOCS = Yale-brown obsessive compulsive scale (primary outcome measure), OCI-R = Obsessive-compulsive inventory revised (secondary outcome measure), Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Table 9. Predicting change in OCD symptoms utilizing indices of interference control.

		Step 1					Step 2				
DV	**IV**	***B***	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
Y-BOCS Total Δ		R^2 = .067, F(2, 37) = 1.337, p = .275					R^2 Δ = .047, FΔ (2, 35) = .923, p = .407				
	BL QIDS	.306	.207	.235	1.477	.148	.258	.211	.198	1.221	.230
	TTX	-.051	.064	-.127	-.797	.430	-.047	.064	-.118	-.739	.465
	Interference RT						.004	.013	.053	.319	.751
	Interference Error						-.924	.680	-.227	-1.358	.183
Y-BOCS		R^2 = .119, F(2, 37) = 2.50, p =.096					R^2 Δ = .030, FΔ (2, 35) = .612, p = .548				
Obsession Δ	BL QIDS	.211	.101	.322	2.083	.044*	.196	.104	.300	1.885	.068†
	TTX	-.029	.031	-.146	-.946	.350	-.028	.031	-.137	-.878	.386
	Interference RT						-.001	.006	-.032	-.199	.843
	Interference Error						-.334	.335	-.163	-.997	.325
Y-BOCS		R^2 = .026, F(2, 37) = .493, p =.615					R^2 Δ = .068, FΔ (2, 35) = 1.306, p = .284				
Compulsion Δ	BL QIDS	.095	.115	.134	.826	.414	.062	.116	.087	.530	.599
	TTX	-.021	.035	-.098	-.603	.550	-.020	.035	-.090	-.558	.581
	Interference RT						.005	.007	.127	.759	.453
	Interference Error						-.590	.374	-.266	-1.576	.124
OCI-R Δ		R^2 = .093, F(2, 36) = 1.851, p = .172					R^2 Δ = .023, FΔ (2, 34) = .437, p = .650				
	BL QIDS	.153	.255	.095	.600	.552	.200	.265	.124	.753	.456
	TTX	-.138	.076	-.291	-1.833	.075	-.140	.077	-.294	-1.811	.079†
	Interference RT						-.007	.016	-.079	-.465	.645
	Interference Error						.727	.807	.151	.900	.374

Note. †p<.10, *p<.05; **p<.01, ***p<.001.
Abbreviations: BL QIDS = Admission depression severity, TTX = Time in treatment, Interference RT = Interference control reaction time index (=mean reaction time on incongruent trials – mean reaction time on congruent trials), Y-BOCS = Yale-brown obsessive compulsive scale (primary outcome measure), OCI-R = Obsessive-compulsive inventory revised (secondary outcome measure), Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Table 10. Predicting change in OCD symptoms utilizing behavioral adjustments in the context of interference control.

		Step 1					Step 2				
DV	**IV**	***B***	***SE B***	***β***	***t***	***p***	***B***	***SE B***	***β***	***t***	***p***
Y-BOCS Total Δ		R^2 = .067, F(2, 37) = 1.337, p = .275					R^2 Δ = .016, FΔ (4, 33) = .146, p = .964				
	BL QIDS	.306	.207	.235	1.477	.148	-.018	.035	-.328	-.521	.606
	TTX	-.051	.064	-.127	-.797	.430	.025	.035	.465	.716	.479
	Con-pCon RT						-.000	.015	-.001	-.004	.997
	Con-pInc RT						-.005	.016	-.115	-.311	.758
	Inc-pInc RT						-.018	.035	-.328	-.521	.606
	Inc-pCon RT						.025	.035	.465	.716	.479
Y-BOCS Obsession Δ		R^2 = .119, F(2, 37) = 2.50, p =.096					R^2 Δ = .054, FΔ (4, 33) = .543, p = .705				
	BL QIDS	.211	.101	.322	2.083	.044*	.199	.112	.304	1.769	.086†
	TTX	-.029	.031	-.146	-.946	.350	-.026	.032	-.132	-.818	.419
	Con-pCon RT						-.016	.017	-.579	-.967	.340
	Con-pInc RT						.023	.017	.842	1.365	.182
	Inc-pInc RT						-.004	.007	-.169	-.541	.592
	Inc-pCon RT						-.003	.008	-.121	-.346	.732
Y-BOCS Compulsion Δ		R^2 = .026, F(2, 37) = .493, p =.615					R^2 Δ = .009, FΔ (4, 33) = .077, p = .989				
	BL QIDS	.095	.115	.134	.826	.414	.084	.131	.119	.639	.527
	TTX	-.021	.035	-.098	-.603	.550	-.024	.038	-.110	-.631	.533
	Con-pCon RT						-.002	.020	-.069	-.106	.916
	Con-pInc RT						.002	.019	.077	.116	.908
	Inc-pInc RT						.004	.008	.154	.455	.652
	Inc-pCon RT						-.002	.009	-.099	-.261	.796
OCI-R Δ		R^2 = .093, F(2, 36) = 1.851, p = .172					R^2 Δ = .069, FΔ (4, 32) = .663, p = .622				
	BL QIDS	.153	.255	.095	.600	.552	.174	.276	.108	.630	.533
	TTX	-.138	.076	-.291	-1.833	.075†	-.128	.078	-.270	-1.642	.110
	Con-pCon RT						.042	.041	.641	1.044	.304
	Con-pInc RT						-.051	.040	-.810	-1.287	.207
	Inc-pInc RT						.011	.018	.202	.585	.563
	Inc-pCon RT						-.009	.020	-.175	-.452	.655

Note. †p<.10, *p<.05; **p<.01, ***p<.001.

Abbreviations: BL QIDS = Admission depression severity, TTX = Time in treatment, Con-pCon RT = Mean reaction time on congruent trials following congruent trials, Con-pInc RT = Mean reaction time on congruent trials following incongruent trials, Inc-pInc RT = Mean reaction time on incongruent trials following incongruent trials, Inc-pCon RT = Mean reaction time on incongruent trials following congruent trials, Y-BOCS = Yale-brown obsessive compulsive scale (primary outcome measure), OCI-R = Obsessive-compulsive inventory revised (secondary outcome measure), Y-BOCS Total Δ = Change in overall OCD symptoms from baseline to discharge, Y-BOCS Obs Δ = Change in obsessional symptoms from baseline to discharge, Y-BOCS Comp Δ = Change in compulsion symptoms from baseline to discharge, OCI-R Δ = Change in overall OCD symptoms from baseline to discharge.

Appendix – Figures

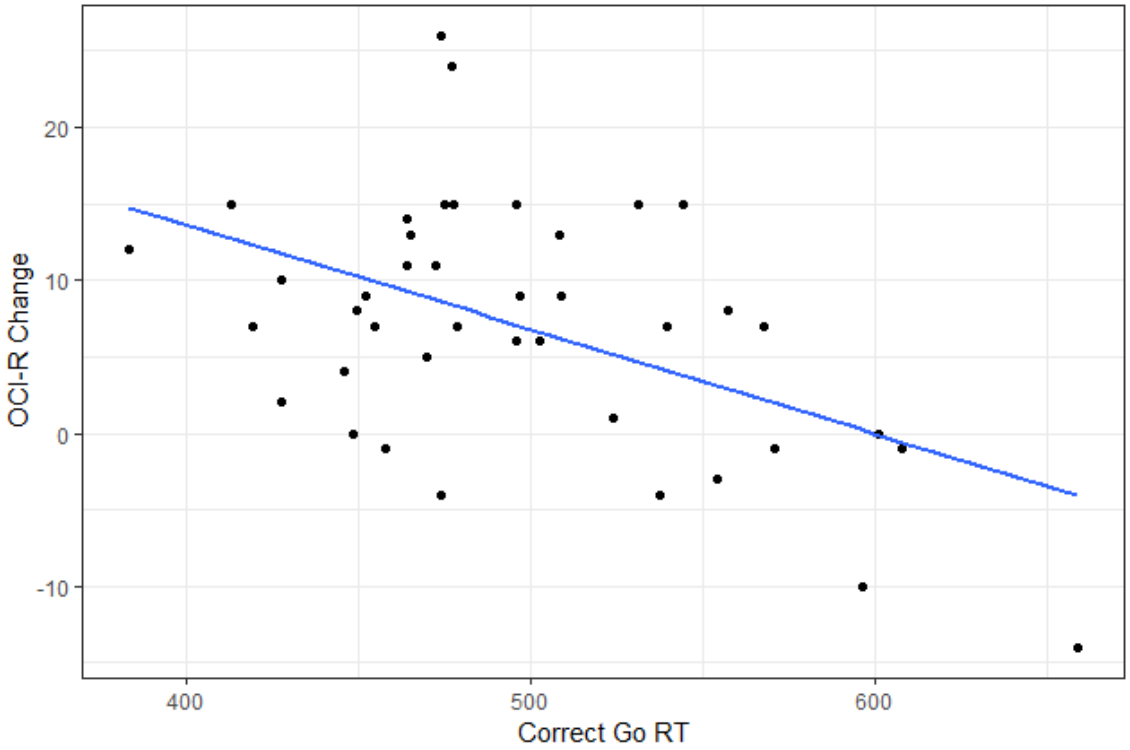

Figure 1. Illustration of the relationship between overall reaction time on correct go trials and change in OCI-R scores from admission to discharge ($r(38) = -.505$, $p < .01$). Findings drawn from the action cancellation domain (go/no-go task) of RI. This negative relationship shows that an elongated reaction time on go trials is associated with smaller gains made in treatment.

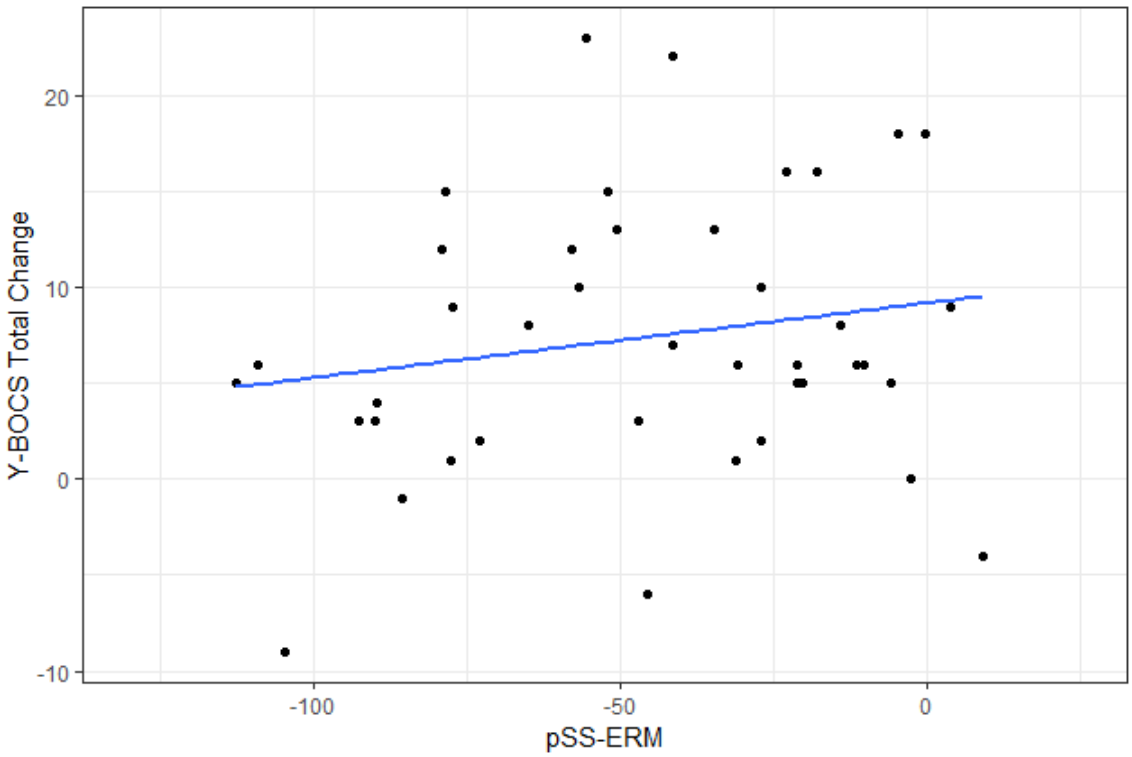

Figure 2. Illustration of the relationship between reaction time following successful inhibition and total change in OCD symptoms from baseline to discharge. Findings drawn from the action cancellation domain (go/no-go task) of RI. Because these pSS-ERM values are negative, they signify RT speed-up. Thus, the positive relationship shows that such speed-up following stop-success is associated with smaller gains made in treatment.

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