Title: Abstract task sequence initiation deficit dissociates anxiety disorders from obsessive-compulsive disorder and healthy controls

Authors: Hannah Doyle[1,2], Christina L. Boisseau[3,4], Sarah L. Garnaat[2,3,5], Steven A. Rasmussen[2,3], Theresa M. Desrochers*[1,2,3]

1. Department of Neuroscience, Brown University, RI, USA
2. Robert J. and Nancy D. Carney Institute for Brain Sciences, Brown University, RI, USA
3. Department of Psychiatry and Human Behavior, Alpert Medical School of Brown University, RI, USA
4. Department of Psychiatry and Behavioral Sciences, Feinberg School of Medicine, Northwestern, IL, USA
5. Department of Psychiatry, Dartmouth-Hitchcock Medical Center, NH, USA

*Correspondence should be addressed to: theresa_desrochers@brown.edu

Abstract

In everyday life, humans carry out sequences of tasks. Such sequential tasks may be disrupted in those with obsessive-compulsive disorder (OCD). Compulsions that are part of the disorder often cause repetitions of tasks or sequences that disrupt daily living. Such disruptions have been observed experimentally and may be unique to OCD. Deficits in implicitly learned motor sequences have been observed in participants with OCD compared to those with anxiety disorders (ANX). However, the sequences performed in daily life are most often not implicit motor-only sequences. They require completing a series of tasks that do not depend on the motor actions always being the same, such as cooking, and thus could be considered abstract. We tested the hypothesis that OCD participants exhibit a deficit in abstract sequential task performance compared to healthy controls (HCs) and ANX. A transdiagnostic sample of participants with OCD, anxiety, and related disorders and healthy controls completed

sequences of simple categorization tasks. Surprisingly, participants with OCD did not perform worse than HCs or ANX. However, ANX participants did exhibit significantly higher reaction times throughout the task and specifically at sequence initiation. Further, task switching, a measure of more general cognitive control, was similar in individuals with ANX compared to healthy controls and OCD, suggesting that abstract sequential control was specifically altered in ANX while general cognitive control was preserved. These results implicate abstract sequential control deficits in individuals with anxiety disorders but not in OCD, and further suggest OCD behavior aligns more closely with implicit motor sequence deficits rather than dysfunctional abstract sequential control.

Introduction

Obsessive-compulsive disorder (OCD) is defined by repetitive and intrusive thoughts (obsessions) and actions (compulsions) which feel difficult to control and affects approximately 2-3% of the world population during their lifetime (Kessler et al., 2005; Lack, 2012; Veale & Roberts, 2014). Popular theories for the development and maintenance of OCD emphasize general cognitive control deficits and implicate prefrontal brain circuitry (Rachman, 1997; Stein, 2002; Taylor et al., 2006). However, the etiology of the disorder is still largely unknown, and therefore behavioral manifestations are mostly not understood.

One prominent feature of OCD that remains poorly understudied is the sequential nature of its behavior. Compulsions often manifest as repetitive mental or physical tasks, or even repeated rituals (Lack, 2012). Such behavior can be conceptualized as sequences of subtasks that people with OCD perform too much or have difficulty completing. Indeed, case studies describe OCD symptoms as manifestations of sequence-like behavior, such as repeatedly arranging objects in a specific order (Lin & Gao, 2022) or counting objects in sets of five (Menon, 2013). OCD symptomatology therefore appears sequential in nature, yet this has not been well studied thus far.

A group of theories referred to as general deficit models (Taylor et al., 2006), also referred to as neurobiological models of OCD, may account for sequence behavior associated with OCD symptoms. These models propose OCD is caused by some dysfunction in a range of cognitive tasks, generalized across information processing systems, The general deficit models posit people with OCD have difficulty switching from task to task as well as general deficits in cognitive control, the ability to flexibly choose responses according to task demands (Saxena & Rauch, 2000; Scott, 1962). Task switching, flexibly moving from one set of rules to another in response to a change in environmental contingencies (Manoach, 2009), is necessary for the completion of sequences, and deficits in this domain may contribute to sequential compulsions observed in OCD. The models posit dysfunction in a neurobiological circuit, the cortico-striatal-thalamo-cortical (CSTC) loop, in part results in task-switching and general cognitive control deficits (Saxena & Rauch, 2000; Stein, 2002). Regions in this circuit (e.g., prefrontal cortex and striatum) are thought to play an important role in motor and non-motor sequential control (Albouy et al., 2008; Desrochers et al., 2015, 2019; Doyon et al., 2009; Graybiel & Grafton, 2015; Penhune & Steele, 2012), further implicating OCD behavior as sequential in nature. The general deficit models may therefore capture sequential behavior deficits in OCD as a potential explanation of overall pathology.

Research in implicit sequence learning supports the general deficit models in OCD. Implicit sequences are often composed of motor actions, with the structure of the sequence learned by repeating the same actions over time (e.g., series of button presses in a specific order). The process of learning these sequences is thought to rely on the striatum (Reiss et al., 2005), a part of the CSTC loop implicated by the general deficit models. A common behavioral paradigm used to probe implicit sequence learning is the serial reaction time task (SRT), which requires participants to repeat the same sequence of finger presses across multiple blocks. Studies using this paradigm report increased reaction times (RTs) across blocks in OCD compared to healthy controls (HCs) (Kathmann et al., 2005; Kelmendi et al., 2016) and

compared to anxiety disorders (social anxiety, panic disorder, and agoraphobia) (Goldman et al., 2008). Therefore, the general deficit models of OCD are supported by observed deficits in a specific type of sequence: implicit motor sequences.

The majority of sequences performed on a daily basis, and those clinically disrupted in OCD, may not be fully captured by implicit motor sequences and, therefore, leave a portion of general deficit models untested. Abstract task sequences are series of tasks completed in a particular order, bound by a higher order structure (Desrochers et al., 2022; Lashley, 1951). For example, the sequential structure of “following a recipe” dictates a series of tasks such as measuring flour, cracking eggs, and whisking in sugar, which must be completed in a specific order. However, while the recipe maintains a certain structure, it does not specify each individual motor action (e.g., which shelf to reach for the flour) or the exact identity of each ingredient (e.g., eggs could come from the store or the chickens in your backyard). This non-reliance on exact motor actions or specific ingredients makes the task sequence abstract and defined by the higher-order structure (the recipe). In a lab-based experiment, an abstract task could be a categorization decision (e.g., color) in response to a displayed simple shape. It is not possible to prepare the motor response (e.g., button press) ahead of seeing the shape, but one knows the abstract task (categorize color). In contrast, implicit motor sequences are not dictated by a structure known prior to sequence execution and are defined by their individual motor actions and task identities (e.g., pressing a specific button in response to a displayed stimulus and the series of stimuli and button presses is always the same). Impairment in abstract task sequential control may occur in OCD, as has been previously theorized by one group (Huey et al., 2008), but never empirically tested. For example, an individual with OCD may fear the stove was left on while cooking and repeatedly check the burners. The general deficit models may extend to abstract task sequence deficits in OCD, since it postulates difficulty in task switching and more general cognitive control, both of which are required for more specific abstract sequential control. Further, the CSTC circuit overlaps with the rostrolateral prefrontal cortex

(RLPFC), which has been shown to be necessary for abstract task sequential control (Desrochers et al., 2015, 2019). The models may therefore more completely explain OCD behavior manifestation through the lens of abstract task sequences, ultimately providing a more comprehensive understanding of symptomatology.

Although the general deficit models postulate deficits in general cognitive control underlie OCD behavior, literature in this domain reports mixed results, so this question remains unanswered. Of the variety of cognitive control measures that have been studied, four are particularly relevant to abstract sequential control: task switching, set-shifting, response inhibition and post-error slowing. First, some studies report worse task switching in individuals with OCD compared to healthy controls (HCs) (Gu et al., 2008; Remijnse et al., 2006; van Velzen et al., 2014), while others finding no significant difference (Moritz et al., 2004; Wolff et al., 2018). Second, similar to task switching, set-shifting is unconsciously shifting attention between tasks. Some studies report slowed RTs and decreased accuracy during set-shifting in OCD participants compared to HCs (Chamberlain et al., 2008; Shin et al., 2014), while others report no difference (Kuelz et al., 2004). The third measure of cognitive control, response inhibition, where a response is cancelled due to a change in goals, has been theorized to lead to OCD symptom manifestation. Studies in this domain more consistently show increased RTs in response inhibition tasks (stop-signal, signal reaction time task) in OCD compared to HCs (Boisseau et al., 2012; Chamberlain et al., 2007; McLaughlin et al., 2016; Menzies et al., 2008). Fourth, post-error slowing is a delay in reaction time on a trial following a previous error thought to reflect the capacity to flexibly adjust behavior based on a previous outcome (Dutilh, Vandekerckhove, et al., 2012). One study in OCD showed deficits in post-error slowing during a cognitive control task (Modirrousta et al., 2015), while another did not (Rueppel et al., 2022). Further, studies report mixed results in correlating symptom severity (using the Yale-Brown Obsessive-Compulsive Scale [Y-BOCS] (Goodman et al., 1989a; 1989b)) with these measures of cognitive control in OCD (Kurt et al., 2017; Youssef et al., 2020), although the general deficit

models predict symptom severity correlates with impaired cognitive control (Taylor et al., 2006). In sum, current studies of cognitive control deficits in OCD are inconsistent in their findings, highlighting a potential gap in the explanatory framework provided by the general deficit models in understanding both cognitive and sequential control in OCD symptoms.

The relationship between abstract sequential control and more general cognitive control can be thought of as hierarchical, in that sequential control supersedes lower-level cognitive control mechanisms. In cognitive control literature, hierarchy occurs when there are multiple goals that are related to each other and must be managed at once (Badre & Desrochers, 2019). Hierarchy occurs in abstract sequential control since one must keep track of the overall sequential structure while also completing individual tasks within the sequence. Sequential control has therefore been conceptualized as the higher-level structure that encompasses lower-level control mechanisms. Two common underlying control mechanisms are post-error slowing and speed-accuracy trade off (Zimmerman, 2011), the relationship between one's willingness to respond slowly and make fewer errors compared to the willingness to respond quickly and make more errors. We predict, based on this concept of hierarchy, that control over monitoring the sequence structure will supersede and therefore not be accounted for by such lower-lever cognitive control mechanisms.

In the current study, we specifically investigate the role of abstract task sequential control in OCD compared to anxiety disorders (ANX) and healthy controls (HC). We used the same abstract task sequence paradigm that was used to show the necessity of RLPFC in sequential control (Desrochers et al., 2015). Participants completed abstract task sequences composed of a series of easy categorization decisions (e.g., whether a shape is a circle or square) that were instructed at the beginning of each block of trials. The task was administered to a diverse cohort of clinical participants (which included OCD and anxiety disorders) and healthy controls (HCs). Informed by the general deficit models and previous studies, we tested five hypotheses: 1. We will replicate behavioral indicators of sequential and cognitive control

observed previously (Desrochers et al., 2015) in the HCs, OCD, and ANX groups; 2. Informed by previous implicit sequence studies (Goldman et al., 2008; Kathmann et al., 2005; Kelmendi et al., 2016), OCD participants will exhibit deficits in a marker of abstract task sequential control, sequence initiation (Desrochers et al., 2015; Schneider & Logan, 2006), compared to the HC and ANX groups; 3. Informed by the general deficit models and motivated by mixed results in the cognitive control literature in OCD, participants with OCD will exhibit a deficit in task switching compared to the HC and ANX groups; 4. The sequence initiation deficit, if observed, will not be better explained by other lower-level cognitive control mechanisms (i.e., speed-accuracy tradeoff and post-error slowing); 5. Symptom severity will correlate with sequential initiation deficits in OCD, as posited by the general deficit models.

Contrary to our hypotheses, we found that the ANX group exhibits sequence initiation deficits and general cognitive control deficits (increased overall RTs) compared to both OCD and HCs. Further, no task switching deficits were observed in either clinical group, and the initiation deficit in ANX was not better explained by other cognitive control mechanisms. Our results suggest an important role for abstract sequential control in the pathology of non-OCD anxiety disorders. Our findings further point to a dissociation between types of sequential control in OCD, with implicit motor sequential deficits more fully explaining behavioral manifestations.

Methods

Experimental Procedures

Participants

Participants were part of a larger study investigating the neural bases of core constructs (harm avoidance and incompleteness) underlying symptoms of obsessive-compulsive spectrum and anxiety disorders. In the larger study, participants underwent clinical screening and completed several cognitive tasks. Here, we report data from participants who completed the

abstract sequential control paradigm. Two hundred thirty-six (182 female) adults (ages 18-64 years; mean 35.5 years) participated in the experiment. Twenty-six participants were removed from subsequent data analysis due to not completing the behavioral task. From the remaining 210 participants, 31 were excluded from further data analysis for high error rates (ERs) (> 20% overall), creating a total of 179 (140 female) participants (ages 18-64; mean 33.9 years) that were included in data analysis. All participants gave informed, written consent approved by the Butler Hospital International Review Board.

Clinical participant inclusion criteria were as follows: Diagnostic Statistic Manual (DSM-5) obsessive-compulsive spectrum (OCD, obsessive-compulsive personality disorder [OCPD], hoarding) and/or anxiety disorder (panic disorder, agoraphobia, social anxiety disorder), 2) age 18 to 65, 3) English speaking, 4) right-handed, and 5) willing and able to provide written informed consent. Clinical exclusion criteria: 1) Cognitive impairment (organic brain syndrome, dementia) that would interfere with study participation, ability to provide informed consent, or completion of self-report questionnaires, 2) current psychotic disorder, 3) psychiatric medications other than serotonin reuptake inhibitors (SRIs) or medications taken for sleep or occasional anxiety (e.g., hydroxoxyzine, trazodone, etc.), 4) pre-morbid IQ < 85 as measured by the National Adult Reading Test (NART), 5) Implanted metallic substances, metallic tattoos received prior to 1990; and 6) pregnancy and any other conditions not allowed in the scanner that would represent a safety risk for participants. Inclusion and exclusion criteria for the healthy controls were the same as for OCD except for no current (past month) DSM-5 diagnosis of any psychiatric disorder or a lifetime diagnosis of OCD or related target disorder (OCPD, hoarding), any anxiety disorder, psychotic disorder, or bipolar mood disorder.

Measures

Anxiety and Related Disorders Interview Schedule for DSM-5 (ADIS-5) (Brown & Barlow, 2014): The ADIS-5 is a semi-structured interview designed to determine reliable diagnosis of

DSM-5 disorders and to screen for the presence of other conditions. Here, the ADIS-5 was used to assess diagnosis of DSM-5 Axis I disorders (OCD, panic disorder, and social anxiety disorder) and provide dimensional ratings of symptom severity. The ADIS-5 provides dimensional assessment of key features of disorders (0-8 ratings). For each diagnosis, interviewers indicated the degree of distress associated with the disorder with a 0-8 clinical severity rating (0 = *none,* 8 = *very severely disturbing/disabling*). Disorders that met or exceeded a formal DSM-5 diagnosis were assigned ratings of 4 or higher; "subclinical" received ratings below 4.

Yale-Brown Obsessive Compulsive Scale (Y-BOCS) (Goodman et al., 1989a; Goodman et al., 1989b)): The Y-BOCS is a validated clinician-administered interview that assesses the presence and severity of obsessions and compulsions over the past week. Using the Symptom Checklist, participants' obsession and compulsions were recorded and placed into distinct, detailed categories. The Severity scale was used to assess the severity of the obsessions and compulsions, resulting in subscale and total severity scores. Ten questions, with scores ranging from 0-4 (most severe) assess time, interference, distress, resistance and control over obsessions and compulsions (total scores can range from 0-40).

Albany Panic and Phobia Questionnaire (APPQ) (Rapee et al., 1994): The APPQ is a 27-item measure of the dimension of fear activities that produce physical sensations (e.g., exercise) and fear of common agoraphobia and social phobic situations. The measure thus has three subscales, interpreted as reflecting fear of agoraphobic situations ("Agoraphobia", 9 items), feat of activities that produce somatic sensations ("Interoceptive", 8 items), and fear of social situations ("Social Phobia", 10 items).

Procedure

The behavioral task used was the same as in a previous neuroimaging study of HCs (Desrochers et al., 2015) (**Fig. 1**). Participants were presented on each trial with a stimulus of

varying size (small [3.5 x 3.5 cm] or large [7 x 7 cm]), shape (circle or square), and color (blue or red) (**Fig. 1A**). The combination of stimulus size, shape, and color formed 8 possible stimuli, which appeared equally throughout the task and did not repeat on adjacent trials. A white fixation cross was displayed during each intertrial interval (ITI) after each stimulus, which was 0.5 s throughout the task. Each trial was shown with response options for the color and shape of the stimulus, mapped onto 'j' and 'k' keyboard keys. Trials timed out after 4 s if no key response was made. Responses were mapped from two fingers, the index and middle of the dominant right hand, onto the 'j' and 'k' keys. Each key corresponded to one shape and color combination (e.g., 'j' maps to both 'blue' and 'circle' while 'k' maps to both 'red' and 'square'). Participants pressed one button per trial to indicate their choice of color or shape. These stimulus-response mappings were kept the same throughout the task for each participant and were counterbalanced across participants. Stimulus congruency was defined as one key mapping to both the color and shape of an image, such that the identity of the stimulus (its color and shape) corresponded to the same button press. Response congruency occurred when correct answers to consecutive stimuli corresponded to repeat button presses (e.g., index finger button is the correct answer to two stimuli in a row). Both types of congruencies were counterbalanced throughout the task.

Stimuli were presented in blocks (**Fig. 1B**) (24-27 trials each, so that blocks ended on different and unpredictable sequence positions, counterbalanced across blocks), with participants completing 4 blocks per run for a total of 5 runs. At each block start, a 4-item sequence was displayed (5 s), followed by a fixation screen (1 s). The items in the sequence (e.g., color, color, shape, shape) indicated the choice a participant should make for each stimulus. In this example, the choice for the first trial corresponds to the image color, for the second trial the image color, for the third the image shape, and for the fourth the image shape. Participants had to remember the sequence throughout the block and re-initiate every 4 stimuli

until the end of the block. No cues were given to participants to indicate sequence position throughout the block.

At the end of each block, a question was displayed which asked participants to choose which sequence item would occur next if another stimulus appeared. Participants responded with one of four keys ('j', 'k', 'l', and ';') to indicate which sequence position (1, 2, 3, or 4) would have come next. These trials timed out after 10 s if no response was recorded. After this screen, a fixation cross was displayed, followed by instructions for the next block.

Each block consisted of one sequence type, of which there were two total (**Fig. 1D**). Simple sequences followed the pattern AABB (with 'A' corresponding to one choice type and 'B' corresponding to the other, i.e., AABB corresponds to the sequences color, color, shape, shape and shape, shape, color, color) and are termed 'simple' for having one task-switch (from A to B) within the sequence. Complex sequences followed the pattern ABBA (i.e., shape, color, color, shape and color, shape, shape, color) and are termed 'complex' for containing two task-switches (from A to B and B to A) within the sequence (Schneider & Logan, 2006). Although the number of switches differed within each sequence, the number of task switches was equivalent across sequences throughout the block as participants repeated sequences, making the probability of occurring switch or repeat trials equal between blocks of complex and simple sequences (i.e., the first position of simple sequences is a task switch). Therefore, inclusion of two different sequence types controls for task switching effects, particularly at the first sequence position.

Each run consisted of each sequence possibility (i.e., the two possible simple and two possible complex sequences), making a total of 4 blocks per run (**Fig. 1C**). The order of sequence blocks was counterbalanced across the runs.

Participants were trained on the task with 4 practice sequences prior to completing the experiment. Training first included button press practice for color and shape choices individually. Participants were then directed by the experimenter through one sequence practice, and finally

practiced three more sequences without experimenter guidance. Once performance competency was established through training, participants began the experiment.

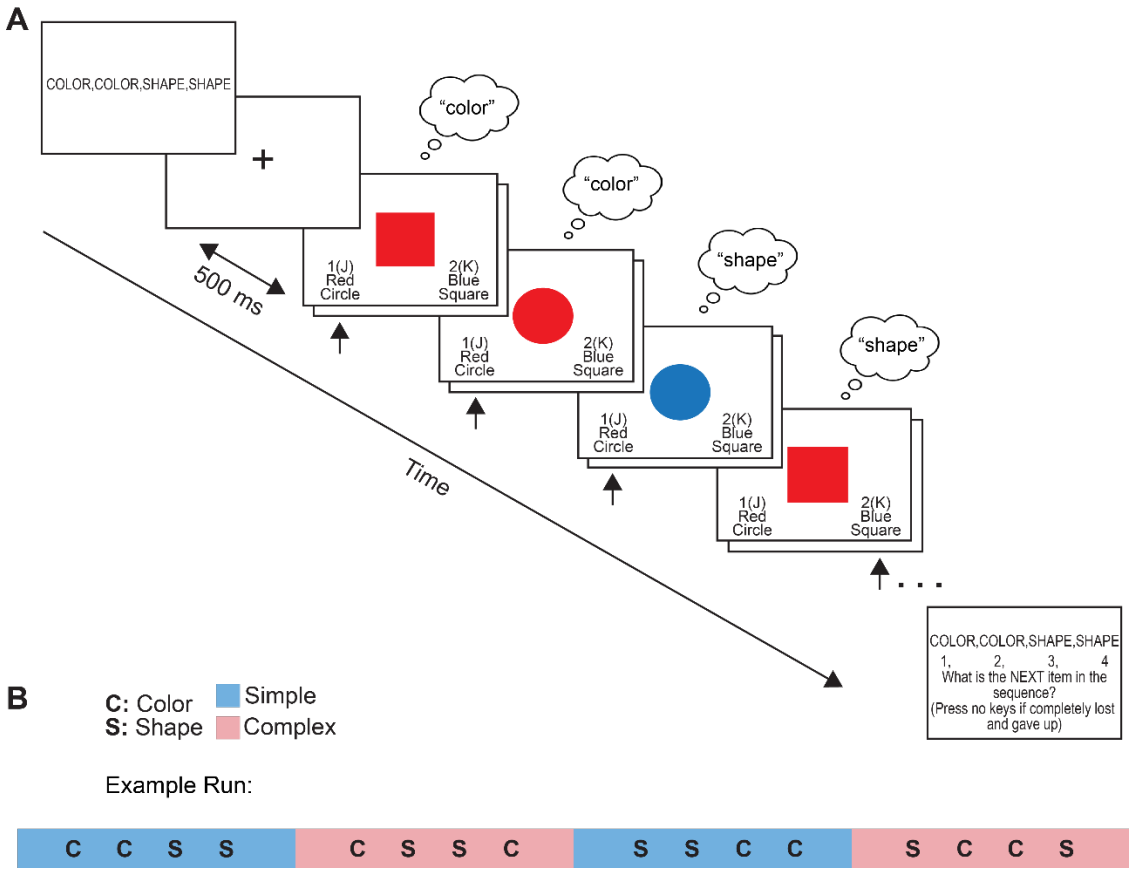

Figure 1. Behavioral task schematic. A) Example trials in a block for the simple sequence. Each block begins with a screen that instructs the sequence, e.g., “COLOR, COLOR, SHAPE, SHAPE”. Each trial consists of one stimulus presentation where the participant must make the correct categorization decision based on the identity of the stimulus and the position in the sequence. The remembered categorization decision for each item is indicated in a thought bubble and the correct choices for each trial are indicated by black arrows. The stimulus remains on screen until a response is made (max 4 sec). After the response (or response time-out), a fixation cross is displayed for the duration of the intertrial interval (ITI, 500 ms). Distance between images is for illustration purposes only and does not represent actual timing. There are approximately 24 trials per block, it can end on any position in the sequence, and the block ends with a sequence position question asking, “What is the NEXT item in the sequence?”. B) Example run containing four blocks, with each block being a simple (CCSS [color, color, shape, shape]; SSCC

[shape, shape, color, color]) or complex (CSSC [color, shape, shape, color]; SCCS [shape, color, color, shape]) sequence. The order of the blocks is counterbalanced across the five runs that each participant performs.

Behavioral Analysis

The first sequence of each block was excluded from analysis (approximately 4% of trials per participant). Additionally, trials with RTs < 100 ms were excluded. ERs were calculated for the remaining trials. Trials were also excluded on which participants "lost track" of the sequence position. To determine these trials, periods of 2 or more error trials were monitored and marked as "lost" periods until the next 4 correct adjacent trials occurred (approximately 3% of trials per participant). Performance was assessed using repeated measures analysis of variance (RM-ANOVA). Age was included as a covariate in all ANOVAs. Sequence initiation was measured by initiation cost, calculated by subtracting RTs at position 3 from position 1 in the sequence (Desrochers et al., 2015; Schneider & Logan, 2006). Positions 1 and 3 were chosen for the initiation cost measure since they were always matched as either task switches or repeats in the sequence (i.e., in each sequence type, positions 1 and 3 are always either both repeats of or switches from the previous trial). Initiation costs were correlated with symptom severity measure composite scores and subscale scores, when applicable. Given our sample size total, variance, number of groups and number of measurements, a post-hoc power analysis (Erdfelder et al., 1996) indicated a required effect size of 0.1 (η_p^2).

Results

Participants completed 5 runs of a sequential task for this experiment (**Fig. 1**). At the start of each block, participants were shown a four-item sequence, which they used to inform decisions about the stimulus color and shape on each trial (**Fig. 1B**). Sequences followed the pattern 'AABB' (simple, containing one task switch, e.g.: color, color, shape, shape) or 'ABBA' (complex, containing two task switches, e.g.: color, shape, shape, color) (**Fig. 1C, D**). Only one

sequence was performed during each of the four blocks during a run (order counterbalanced across runs). Importantly, participants were not provided external sequence cues. Therefore, they had to remember and keep track of the sequence throughout each block, requiring them to maintain an "internal sequence boundary" to respond correctly to each trial. The total number of participants (n = 179) consisted of healthy controls (HCs, n = 47) and a clinical group (n = 132). Clinical diagnoses that were assessed as a part of the study included the following: OCD, obsessive-compulsive personality disorder (OCPD), hoarding disorder (HD), panic disorder (PD), social phobia (SP), agoraphobia (AGP), body dysmorphic disorder (BDD), and post-traumatic stress disorder (PTSD).

For this study of abstract sequential control in OCD and anxiety disorders, we focused on two clinical groups: OCD and anxiety disorders (ANX). The OCD group (n = 46) was defined as any participant with a primary OCD diagnosis of those disorders assessed and could include other non-assessed comorbidities. OCD was defined as the primary diagnosis if the clinical severity rating (CSR) was greater for OCD compared to other assessed anxiety disorders: PD, SP, and AGP. The ANX group (n = 22) was defined as anyone who was diagnosed with PD, AGP, and/or SP and not diagnosed with OCD. The groups did not have any overlapping membership. **Table 1** summarizes the demographics for the HC, OCD, and ANX groups. Overall, the participants across these groups performed the task as instructed and well (average ER = 7.78, +/- 8.49 [1 SD]), with no difference in overall error rate (ER) across groups (one-way ANOVA: $F(2,114) = 0.02$, $p = 0.98$). Since ERs were similar across groups, we focused on RTs.

Group	Number of Participants	Age (years; mean +/- 1 STD)	Sex	Y-BOCS total (mean +/- 1 STD)
OCD	46	31.1 (+/- 12.3)	38 f (9 m)	21.1 (+/- 5.0)
ANX	22	36.3 (+/- 12.8)	17 f (5 m)	N/A
HC	47	31.2 (+/- 11.8)	34 f (13 m)	N/A

Table 1. Group descriptive statistics. Descriptive statistics for the three groups of interest (OCD, ANX, HC). Information provided includes number of group members, average age (+/- 1 standard deviation), number of males and females and average Y-BOCS (range 0-40).

To test our first hypothesis, that we will replicate previously observed behavioral indicators of sequential and cognitive control in HCs (Desrochers et al., 2015, 2019; Schneider & Logan, 2006), we will examine the HC, OCD, and ANX groups separately. First, we examined initiation cost as an indicator of abstract sequential control. Initiation costs are observed over and above any potential effects of switching or repeating tasks. Initiation cost in this task was calculated as the difference in reaction times (RTs) between positions one and three in the sequence. This comparison was performed because task switching and repeating is matched at those positions across sequences (e.g., in the complex sequence, ABBA, positions one and three are both task repeats). We found that all groups exhibited significant initiation costs (**Fig. 2A; Table 2**, rows 1-3). Thus, all participant groups perform the abstract tasks as sequences and show evidence of sequential control.

To test more general cognitive control that is not necessarily dependent on abstract sequential properties within our first hypothesis, we examined switch costs. Switch cost is the difference between switching and repeating tasks (e.g., in AABB, the difference between positions two and three). For this analysis, we examined both ERs and RTs, as switch costs have been observed in both measures in HCs (Desrochers et al., 2015, 2019; Schneider & Logan, 2006). We replicated those previous results and observed switch costs in all groups independently (**Table 3**, rows 1-3**; Fig.2 B,D**). Thus, general indicators of cognitive control are present in the current sample in support of the first hypothesis.

Group	Factor	*dfs*	*F*	*p*	η_p^2
OCD	Position	1,45	185.1	<0.001	0.8
ANX	Position	1,21	126.7	<0.001	0.86
HC	Position	1,46	161.6	<0.001	0.78

OCD vs. HC	Group	1,90	0.57	0.45	0.0063
	Position	1,90	53.33	<0.001	0.37
	Group x Position	2,90	2.03	0.16	0.022
OCD vs. ANX	Group	1,65	5.17	0.026	0.074
	Position	1,65	60.99	<0.001	0.48
	Group x Position	2,65	3.31	0.074	0.048
ANX vs. HC	Group	1,66	6.68	0.012	0.092
	Position	1,66	37.56	<0.001	0.36
	Group x Position	2,66	7.6	0.0075	0.1

Table 2. Within and between groups rmANOVA for initiation cost. Repeated measures ANOVAs were conducted on each individual group (rows 1-3) and between groups (rows 4-12) for position 1 vs. 3 RTs. Degrees of freedom (dfs), F value (F), p value (p), and effect size (η_p^2) are reported for each factor.

Having now provided evidence that markers of sequential and general cognitive control were present across the participant groups independently, we next tested hypotheses between OCD and the other groups. Specifically, we next tested the second hypothesis that participants with OCD will exhibit abstract sequence performance deficits compared to HC and ANX groups, similar to findings in previous studies of implicit motor sequences (Goldman et al., 2008; Kathmann et al., 2005; Kelmendi et al., 2016). To test this hypothesis, we performed planned comparisons of initiation costs in OCD to the HC and ANX groups. We did not find a difference in RTs between the groups or a significant increase in initiation cost (i.e., sequential control deficits) in OCD compared to HC (**Table 2**, row 6**; Fig. 2A**). We found a marginal difference between the OCD and ANX groups; however, it was not in the hypothesized direction. We found significantly greater RTs and marginally greater initiation costs in the ANX group compared to the OCD group (**Table 2**, row 9; **Fig. 2A**). Follow up testing also revealed that participants with ANX exhibited significantly higher RTs and initiation costs compared to the HC group (**Table 2**, row 12). In summary, we do not provide evidence to support the second hypothesis that OCD

has greater initiation costs than HC and ANX and instead provide evidence that ANX participants may have initiation cost deficits compared to OCD and HC.

Based on the general deficit models, the third hypothesis is that participants with OCD will exhibit a deficit in task switching compared to the HC and ANX groups. To test this hypothesis, we compared RT and ER switch costs in OCD to HC and ANX groups. Our observations did not support the hypothesis. There were no significant differences for these group comparisons in switch costs in either RTs (**Table 3,** rows 4-9; **Fig. 2B**) or ER (**Table 3**, rows 4-9**; Fig. 2D**). For completeness and given the observed differences in initiation cost between the ANX and HC groups, we compared switch costs in these two groups as well and also found no differences in switch costs in RT (**Table 3**, rows 10-12**; Fig. 2B**) or ER (**Table 3**, rows 10-12**; Fig. 2D**). Therefore, these results do not support the general deficit models or our third hypothesis and highlight the importance of examining cognitive control in more complex tasks. We will expand on this point in the discussion.

		Reaction time				Error rate			
Group	Factor	*dfs*	*F*	*p*	η_p^2	*dfs*	*F*	*p*	η_p^2
OCD	Trial Type	1,45	142.61	<0.001	0.76	1,45	20.35	<0.001	0.31
ANX	Trial Type	1,21	33.1	<0.001	0.61	1,21	11.89	0.0025	0.37
HC	Trial Type	1,46	78	<0.001	0.63	1,46	31	<0.001	0.41
OCD vs. HC	Group	1,90	0.036	0.85	<0.001	1,90	<0.001	0.98	<0.001
	Trial Type	1,90	19.56	<0.001	0.18	1,90	2.19	0.14	0.024
	Group x Trial Type	2,90	0.69	0.41	0.0076	1,90	0.17	0.68	0.0019
OCD vs. ANX	Group	1,65	3.78	0.056	0.055	1,65	0.19	0.67	0.0029
	Trial Type	1,65	18.57	<0.001	0.22	1,65	7.31	0.0087	0.1
	Group x Trial Type	2,65	0.38	0.54	0.006	1,65	0.13	0.72	0.002
ANX vs. HC	Group	1,66	3.58	0.063	0.051	1,66	0.14	0.71	0.072

	Trial Type	1,66	11.22	0.0013	0.15	1,66	5.12	0.027	0.0021
	Group x Trial Type	2,66	0.0086	0.93	<0.001	1,66	0.81	0.37	0.012

Table 3. **Within and between groups rmANOVA for switch cost.** Repeated measures ANOVAs were conducted on each individual group (rows 1-3) and between groups (rows 4-12) for trial type (switch v.s. repeat). Degrees of freedom (dfs), F value (F), p value (p), and effect size (η_p^2) are reported for each factor.

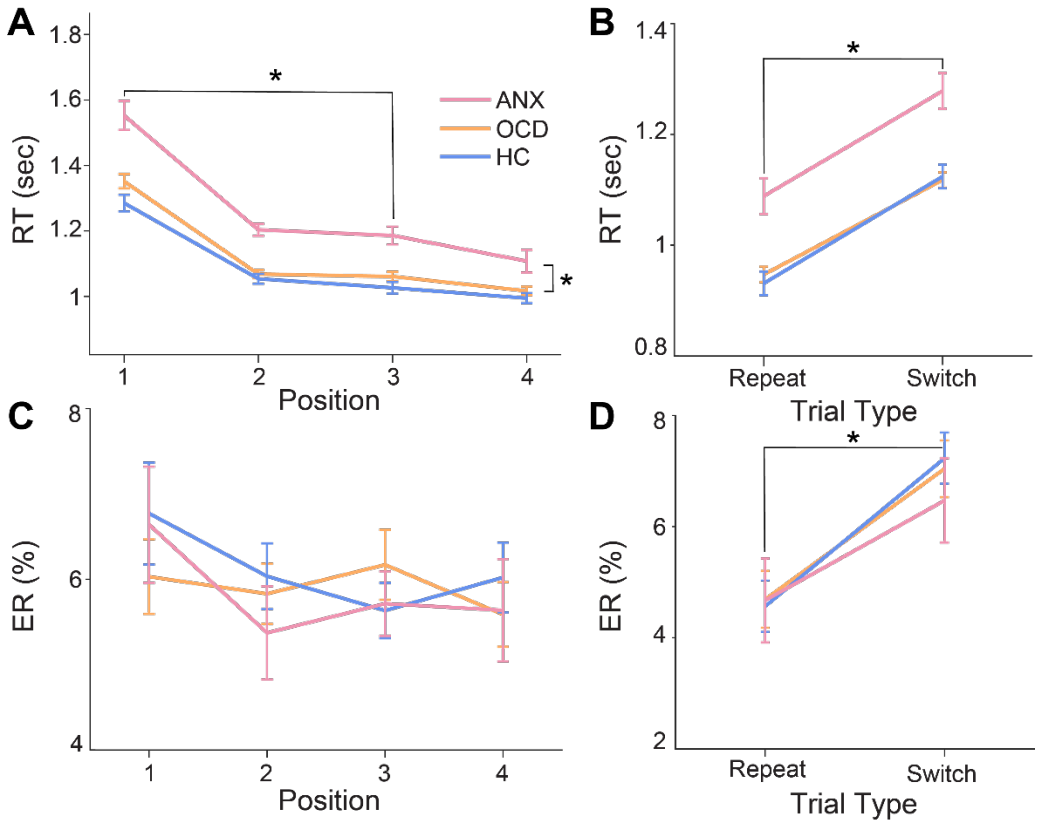

Figure 2. General behavior results for OCD, ANX, and HC groups. We report RT results in the first row and ER results in the second row. A) ANX exhibit significantly increased RT at position 1 compared to positions 2,3,4 compared to the HC group and OCD groups, and significantly higher initiation costs compared to the HC group. ANX exhibits significantly higher RTs across all positions compared to the HC

and OCD groups. B) RT switch costs are not significantly different between any groups. (C,D) ERs across position and ER switch costs are not significantly different between any groups.

Originally, our fourth hypothesis had been that a sequence initiation deficit in OCD would not be better explained by lower-level cognitive control mechanisms such as a speed-accuracy tradeoff or post-error slowing. However, we did not observe a sequence initiation deficit in OCD (compared to HC or ANX), and we did observe a sequence initiation deficit in ANX. Therefore, as a follow-up to these results, we examined whether these other cognitive control mechanisms could better explain the observed initiation deficit in ANX. First, it was possible that, for participants with ANX, slower responses enabled decreased ER at the first position in the sequence (i.e., a speed-accuracy tradeoff). If this were the case, there would be a negative correlation between RTs and ER (such that increases in RT would lead to decreases in ER). We did not find evidence of this negative correlation in the ANX group, but rather a marginally positive correlation ($p = 0.079$, $r = 0.38$, slope = 5.97) at the first position (**Fig. 3A**). A positive correlation is commonly observed during cognitive tasks (C. C. Wood & Jennings, 1976). Further, ERs at position 1 between groups were not significantly different (independent samples t-test, ANX v.s. HC: $t(67) = -0.089$, $p = 0.93$; ANX v.s. OCD: $t(66) = 0.39$, $p = 0.69$). These results indicate that a speed-accuracy trade-off did not underlie the observed abstract sequential control difference in ANX, supporting hypothesis four.

Another possible behavior that could account for significantly higher initiation costs in the ANX group is post error slowing. Post error slowing is an increase in RT on the response following an incorrect response and can be used as a marker of cognitive control, as this process is thought to reflect the capacity to flexibly adjust behavior after an outcome (Dutilh, Vandekerckhove, et al., 2012; Dutilh, van Ravenzwaaij, et al., 2012). In non-sequential tasks, increased post error slowing (greater RTs following error trials) has been observed in ANX compared to both OCD and HCs (Rueppel et al., 2022). In the current experiment, increased

post error slowing in response to errors at position 4 could give rise to increased RTs at position 1 and the appearance of an increased initiation cost in participants with ANX compared to HCs. This post error slowing effect could therefore account, at least in part, for the increased initiation costs observed in ANX (**Fig. 2**). To test this possibility, we first compared ER at position 4 between groups and then RTs at position 1 following position 4 errors between groups. We found that there was significant overall post error slowing in each group separately (within-subjects rmANOVA; HC: F(1,44) = 110, p = 1.46e-13, η_p^2 = 0.73; ANX: F(1,21) = 92.6, p = 3.78e-09, η_p^2 = 0.82; OCD: F(1,45) = 88.9, p = 3.20e-12, η_p^2 = 0.66). We did not find that the ANX group made significantly more errors at position 4 compared to the HC group (independent samples t-test, t(67) = -0.29, p = 0.78; **Fig. 2B**). We also did not find that the ANX group was significantly slower at position 1 following a position 4 error compared to the HC group (between-groups rmANOVA; F(1,58) = 0.36, p = 0.55; η_p^2 = 0.0061 **Fig. 3B**). Therefore, we did not observe differences in the post error slowing effect between groups, which supports hypothesis 4 for the ANX group compared to HC. The initiation cost result may therefore be related to a difference in abstract sequential control rather than in general cognitive control, since post error slowing does not account for the slowed initiation in ANX compared to HCs.

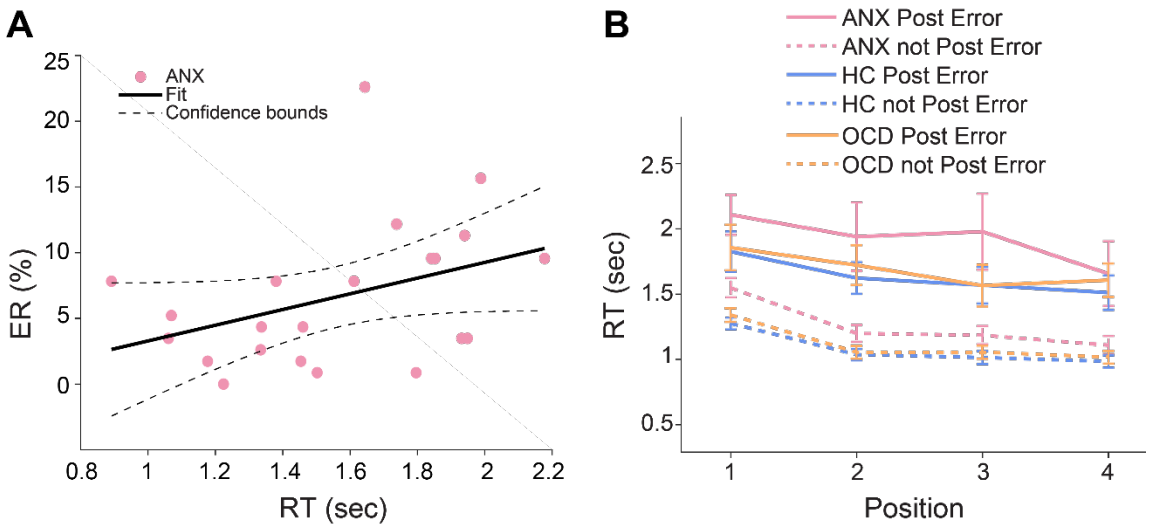

Figure 3. Cognitive control measures do not lead to a sequence initiation difference in ANX. A) Correlations between RT and ER do not indicate a speed/accuracy tradeoff at position 1 in the ANX group. B) Post-error RTs at position 1 in the ANX group are not significantly different than those in the HC group.

The fifth hypothesis was that symptom severity will correlate with sequential initiation deficits in OCD. To test this hypothesis, we correlated initiation cost and total Y-BOCS scores as a measure of symptom severity in OCD. We did not observe a reliable correlation (Y-BOCS total: $p = 0.40$, $r = -0.13$). Further, in light of the finding that ANX has impaired initiation costs, we performed an exploratory analysis to correlate a measure of symptom severity in ANX, the APPQ measure, and initiation cost in that group. We did not observe a reliable correlation (APPQ: $p = 0.49$, $r = 0.-15$); however, the measures included in this study were not optimized for assessing participants with ANX.

Discussion

This study investigated abstract sequential control and more general cognitive control in participants with OCD and anxiety disorders using abstract task sequences. Surprisingly, and in contrast to our hypotheses based on general deficit models and previous studies of implicit motor sequences, we found that an indicator of sequential control (sequence initiation) was

disrupted in the ANX but not the OCD group. Participants with ANX exhibited greater RTs and initiation costs compared to the OCD and HC groups. Further, we did not observe any differences across the groups in a more general measure of cognitive control, task switching. Initiation costs did not correlate significantly with symptom severity in either clinical group. Together, these results are the first to show specific abstract sequential control differences in individuals with anxiety disorders and reveal a new behavioral axis that may dissociate OCD from ANX symptomatology. These results also encourage a reevaluation of general deficit models in the context of more complex cognitive tasks such as abstract task sequences.

The unexpected sequence initiation results in the OCD group inform our current understanding of sequential behavior in this disorder and call for further studies investigating this symptom dimension. We expected OCD participants to exhibit an abstract task sequence deficit based on the general deficit models (Taylor et al., 2006) and previous studies showing impaired implicit sequence learning in this group (Goldman et al., 2008; Kathmann et al., 2005; Kelmendi et al., 2016). However, since we did not observe abstract task sequence deficits in OCD compared to HCs or the ANX group, sequential behavior in OCD may be better explained by dysfunction in implicit motor sequence learning. The observed lack of sequential deficits may further be explained by mixed cognitive control literature in OCD, with some studies finding no deficits in task switching (Moritz et al., 2004; Wolff et al., 2018), set-shifting (Kuelz et al., 2004), and post error slowing (Rueppel et al., 2022) in OCD. Since abstract task sequential control is closely tied to these aspects of cognitive control (Desrochers et al., 2022), these studies may support our similar result showing OCD participants do not exhibit sequence initiation deficits compared to ANX and HCs. Given this study was the first of its kind and had a limited sample size, further studies are needed to probe abstract task sequential control in OCD and the possible dissociation that exists between implicit motor and abstract task sequential control in this group.

The unexpected sequence initiation deficit observed in ANX suggests a possible dissociation between OCD and anxiety disorders along the axis of sequential control. Our finding was unexpected since implicit sequence learning has been shown to be intact in people with SP, AGP, and PD (Goldman et al., 2008) compared to in those with OCD. However, people with anxiety disorders exhibit deficits in attentional control (Rueppel et al., 2022; Yu et al., 2018), and set-shifting (Kertz et al., 2016). Abstract task sequential control deficits therefore contribute to the literature on the role of cognitive control in ANX symptom manifestation and motivate future studies. Additionally, these results suggest a tool to dissociate OCD from other non-OCD anxiety disorders may be abstract sequential task performance, which may inform clinical theories and future treatments for these disorders.

This study was limited primarily because of small group sizes when isolating particular diagnoses due to the co-occurrence of many of the assessed diagnoses within participants. Further, recruitment of participants was optimized for a larger study with different primary research questions and not for the questions we posed in this current study. Both these factors may contribute to the marginally significant effect in initiation costs observed between the OCD and ANX groups, and the medium effect sizes we observed in some results. Though these conditions were not ideal, we found it notable that we provided evidence for an initiation cost deficit to possibly isolate anxiety disorders from OCD. This research highlights the utility of studies using large sample sizes across an array of measures and calls for future studies investigating abstract task sequential control in both groups.

General deficit models may also need to be expanded with respect to the lack of task switching differences that we observed. We predicted impaired task switching in OCD based on predictions from general deficit models and other studies that observed task switching deficits in OCD (Gu et al., 2008; Remijnse et al., 2006; van Velzen et al., 2014). However, a key difference between our behavioral task and typical task switching paradigms is that task switching in our study is nested within a higher-order sequential structure. This hierarchical structure may

increase task demands and lead to different effects at different hierarchical levels (i.e., the "higher" sequence level and the "lower" task level). In support of this idea, a previous study showed that when participants with OCD performed a secondary task along with task switching, RTs in the secondary task significantly increased but task switching in the primary task was preserved (Demeter et al., 2017). Therefore, an increase in task demands can impair performance on the more demanding (or higher-order) part of the task, while task switching performance remains unchanged. Similarly, the addition of sequential structure in our paradigm may lead to a shift in the control of higher-level sequential structure while preserving switching between subtasks within a sequence. Our results suggest that general deficit models (specifically their prediction about impaired task switching in OCD) may not sufficiently explain OCD behavior and that more demanding, hierarchically structured tasks should be considered to inform these models.

Although no reliable behavioral differences were observed in OCD in the present sequential paradigm, the underlying neural circuitry may differ between OCD and HCs and abstract task sequence performance may be accomplished differently in these two groups. It has been recognized that similar behavior between groups, particularly in clinical groups, may be caused by different neural mechanisms (Huys et al., 2016). Further, neural circuits implicated in OCD, especially in prefrontal and striatal regions, overlap with brain regions involved in abstract sequence processing (Alexander et al., 1986; Desrochers et al., 2015, 2019, 2022; Greenberg et al., 1997; Harrison et al., 2009; Page et al., 2009; Roth et al., 2007). Therefore, although no behavioral differences between OCD and HC were observed in our task, an open question remains as to whether or not this group exhibits a distinct neural mechanism to achieve abstract sequential control compared to HCs.

Previous work shows neural circuits associated with ANX overlap with those involved in abstract sequential control, which will inform future studies investigating the modulation of these circuits for treatment of these disorders. In anxiety disorders, neuroimaging studies point to

circuitry involving prefrontal regions and subcortical areas that underlie pathology (Berkowitz et al., 2007). Further, prefrontal cortex dysfunction has been observed in participants with generalized anxiety disorder during an emotion dysregulation task (Mochcovitch et al., 2014) and participants with high anxiety exhibit impaired cognitive control (J. Wood et al., 2001), which involves frontal cortex. Prefrontal circuitry is therefore implicated in ANX and overlaps with the RLPFC, shown to be necessary for abstract sequential control (Desrochers et al., 2015, 2019). Cognitive control literature in ANX and our present results call for future studies investigating the role of RLPFC and other sequence-related brain regions in abstract sequential control in ANX. Future studies may also attempt to dissociate OCD and ANX further through the lens of neural circuitry that underlies sequential control. Understanding the neural mechanisms underlying sequential behavior in OCD and ANX will provide further insight to possible differences in how these groups perform sequences compared to HCs.

Using abstract task sequences, we showed that ANX participants exhibit an initiation deficit in abstract task sequential control compared to HCs (with a marginal effect compared to OCD). The findings from this study provide a novel framework under which to interpret both OCD and ANX symptomatology, suggesting OCD sequential behavior may be explained more clearly by implicit motor sequence deficits and showing a dissociation between OCD and ANX during abstract task sequences. Results from this study will provide the foundation for future studies specifically investigating abstract sequential control in clinical groups and may aid in the development of new and more efficacious treatments for OCD and anxiety disorders.

References

Albouy, G., Sterpenich, V., Balteau, E., Vandewalle, G., Desseilles, M., Dang-Vu, T., Darsaud, A., Ruby, P., Luppi, P.-H., Degueldre, C., Peigneux, P., Luxen, A., & Maquet, P. (2008). Both the hippocampus and striatum are involved in consolidation of motor sequence memory. *Neuron*, *58*(2), 261–272. https://doi.org/10.1016/j.neuron.2008.02.008

Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, *9*, 357–381. https://doi.org/10.1146/annurev.ne.09.030186.002041

Badre, D., & Desrochers, T. M. (2019). Hierarchical cognitive control and the frontal lobes. *Handbook of Clinical Neurology*, *163*, 165–177. https://doi.org/10.1016/B978-0-12-804281-6.00009-4

Berkowitz, R. L., Coplan, J. D., Reddy, D. P., & Gorman, J. M. (2007). The human dimension: How the prefrontal cortex modulates the subcortical fear response. *Reviews in the Neurosciences*, *18*(3–4), 191–207. https://doi.org/10.1515/revneuro.2007.18.3-4.191

Boisseau, C. L., Sibrava, N. J., Garnaat, S. L., Mancebo, M. C., Eisen, J. L., & Rasmussen, S. A. (2018). The Brown Incompleteness Scale (BINCS): Measure development and initial evaluation. *Journal of Obsessive-Compulsive and Related Disorders*, *16*, 66. https://doi.org/10.1016/j.jocrd.2017.12.008

Boisseau, C. L., Thompson-Brenner, H., Caldwell-Harris, C., Pratt, E., Farchione, T., & Barlow, D. H. (2012). Behavioral and cognitive impulsivity in obsessive-compulsive disorder and eating disorders. *Psychiatry Research*, *200*(2–3), 1062–1066. https://doi.org/10.1016/j.psychres.2012.06.010

Brown, T. A., & Barlow, D. H. (2014). *Anxiety and Related Disorders Interview Schedule for DSM-5 (ADIS-5) - Adult and Lifetime Version: Clinician Manual*. Oxford University Press.

Chamberlain, S. R., Fineberg, N. A., Menzies, L. A., Blackwell, A. D., Bullmore, E. T., Robbins, T. W., & Sahakian, B. J. (2007). Impaired cognitive flexibility and motor inhibition in unaffected first-degree relatives of patients with obsessive-compulsive disorder. *The American Journal of Psychiatry*, *164*(2), 335–338. https://doi.org/10.1176/ajp.2007.164.2.335

Chamberlain, S. R., Menzies, L., Hampshire, A., Suckling, J., Fineberg, N. A., del Campo, N., Aitken, M., Craig, K., Owen, A. M., Bullmore, E. T., Robbins, T. W., & Sahakian, B. J.

(2008). Orbitofrontal dysfunction in patients with obsessive-compulsive disorder and their unaffected relatives. *Science (New York, N.Y.)*, *321*(5887), 421–422. https://doi.org/10.1126/science.1154433

Demeter, Gy., Harsányi, A., Csigó, K., & Racsmány, M. (2017). STOPPING OF TASK-SWITCHING IN PATIENTS WITH OBSESSIVE-COMPULSIVE DISORDER (OCD). *European Neuropsychopharmacology*, *27*(6), 620. https://doi.org/10.1016/j.euroneuro.2016.07.026

Desrochers, T. M., Ahuja, A., Maechler, M. R., Shires, J., Yusif Rodriguez, N., & Berryhill, M. E. (2022). Caught in the ACTS: Defining Abstract Cognitive Task Sequences as an Independent Process. *Journal of Cognitive Neuroscience*, *34*(7), 1103–1113. https://doi.org/10.1162/jocn_a_01850

Desrochers, T. M., Chatham, C. H., & Badre, D. (2015). The necessity of rostrolateral prefrontal cortex for higher-level sequential behavior. *Neuron*, *87*(6), 1357–1368. https://doi.org/10.1016/j.neuron.2015.08.026

Desrochers, T. M., Collins, A. G. E., & Badre, D. (2019). Sequential Control Underlies Robust Ramping Dynamics in the Rostrolateral Prefrontal Cortex. *The Journal of Neuroscience*, *39*(8), 1471–1483. https://doi.org/10.1523/JNEUROSCI.1060-18.2018

Doyon, J., Bellec, P., Amsel, R., Penhune, V., Monchi, O., Carrier, J., Lehéricy, S., & Benali, H. (2009). Contributions of the basal ganglia and functionally related brain structures to motor learning. *Behavioural Brain Research*, *199*(1), 61–75. https://doi.org/10.1016/j.bbr.2008.11.012

Dutilh, G., van Ravenzwaaij, D., Nieuwenhuis, S., van der Maas, H. L. J., Forstmann, B. U., & Wagenmakers, E.-J. (2012). How to measure post-error slowing: A confound and a simple solution. *Journal of Mathematical Psychology*, *56*(3), 208–216. https://doi.org/10.1016/j.jmp.2012.04.001

Dutilh, G., Vandekerckhove, J., Forstmann, B. U., Keuleers, E., Brysbaert, M., & Wagenmakers, E.-J. (2012). Testing theories of post-error slowing. *Attention, Perception & Psychophysics*, *74*(2), 454–465. https://doi.org/10.3758/s13414-011-0243-2

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER: A general power analysis program. *Behavior Research Methods, Instruments, & Computers*, *28*(1), 1–11. https://doi.org/10.3758/BF03203630

Goldman, B. L., Martin, E. D., Calamari, J. E., Woodard, J. L., Chik, H. M., Messina, M. G., Pontarelli, N. K., Marker, C. D., Riemann, B. C., & Wiegartz, P. S. (2008). Implicit learning, thought-focused attention and obsessive-compulsive disorder: A replication and extension. *Behaviour Research and Therapy*, *46*(1), 48–61. https://doi.org/10.1016/j.brat.2007.10.004

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., Heninger, G. R., & Charney, D. S. (1989). The Yale-Brown Obsessive Compulsive Scale: I. Development, Use, and Reliability. *Archives of General Psychiatry*, *46*(11), 1006–1011. https://doi.org/10.1001/archpsyc.1989.01810110048007

Graybiel, A. M., & Grafton, S. T. (2015). The Striatum: Where Skills and Habits Meet. *Cold Spring Harbor Perspectives in Biology*, *7*(8), a021691. https://doi.org/10.1101/cshperspect.a021691

Greenberg, B. D., George, M. S., Martin, J. D., Benjamin, J., Schlaepfer, T. E., Altemus, M., Wassermann, E. M., Post, R. M., & Murphy, D. L. (1997). Effect of prefrontal repetitive transcranial magnetic stimulation in obsessive-compulsive disorder: A preliminary study. *The American Journal of Psychiatry*, *154*(6), 867–869. https://doi.org/10.1176/ajp.154.6.867

Gu, B.-M., Park, J.-Y., Kang, D.-H., Lee, S. J., Yoo, S. Y., Jo, H. J., Choi, C.-H., Lee, J.-M., & Kwon, J. S. (2008). Neural correlates of cognitive inflexibility during task-switching in

obsessive-compulsive disorder. *Brain*, *131*(1), 155–164. https://doi.org/10.1093/brain/awm277

Harrison, B. J., Soriano-Mas, C., Pujol, J., Ortiz, H., López-Solà, M., Hernández-Ribas, R., Deus, J., Alonso, P., Yücel, M., Pantelis, C., Menchon, J. M., & Cardoner, N. (2009). Altered corticostriatal functional connectivity in obsessive-compulsive disorder. *Archives of General Psychiatry*, *66*(11), 1189–1200. https://doi.org/10.1001/archgenpsychiatry.2009.152

Huey, E. D., Zahn, R., Krueger, F., Moll, J., Kapogiannis, D., Wassermann, E. M., & Grafman, J. (2008). A Psychological and Neuroanatomical Model of Obsessive-Compulsive Disorder. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *20*(4), 390–408. https://doi.org/10.1176/appi.neuropsych.20.4.390

Huys, Q. J. M., Maia, T. V., & Frank, M. J. (2016). Computational psychiatry as a bridge from neuroscience to clinical applications. *Nature Neuroscience*, *19*(3), 404–413. https://doi.org/10.1038/nn.4238

Kathmann, N., Rupertseder, C., Hauke, W., & Zaudig, M. (2005). Implicit sequence learning in obsessive-compulsive disorder: Further support for the fronto-striatal dysfunction model. *Biological Psychiatry*, *58*(3), 239–244. https://doi.org/10.1016/j.biopsych.2005.03.045

Kelmendi, B., Adams, T., Jakubovski, E., Hawkins, K. A., Coric, V., & Pittenger, C. (2016). Probing Implicit Learning in Obsessive-Compulsive Disorder: Moderating Role of Medication on the Weather Prediction Task. *Journal of Obsessive-Compulsive and Related Disorders*, *9*, 90–95. https://doi.org/10.1016/j.jocrd.2016.03.003

Kertz, S. J., Belden, A. C., Tillman, R., & Luby, J. (2016). Cognitive Control Deficits in Shifting and Inhibition in Preschool Age Children are Associated with Increased Depression and Anxiety Over 7.5 Years of Development. *Journal of Abnormal Child Psychology*, *44*(6), 1185–1196. https://doi.org/10.1007/s10802-015-0101-0

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602. https://doi.org/10.1001/archpsyc.62.6.593

Kuelz, A. K., Hohagen, F., & Voderholzer, U. (2004). Neuropsychological performance in obsessive-compulsive disorder: A critical review. *Biological Psychology*, *65*(3), 185–236. https://doi.org/10.1016/j.biopsycho.2003.07.007

KURT, E., YILDIRIM, E., & TOPÇUOĞLU, V. (2017). Executive Functions of Obsessive Compulsive Disorder and Panic Disorder Patients in Comparison to Healty Controls. *Archives of Neuropsychiatry*, *54*(4), 312–317. https://doi.org/10.5152/npa.2016.14872

Lack, C. W. (2012). Obsessive-compulsive disorder: Evidence-based treatments and future directions for research. *World Journal of Psychiatry*, *2*(6), 86. https://doi.org/10.5498/wjp.v2.i6.86

Lashley, K. S. (1951). The problem of serial order in behavior. In *Cerebral mechanisms in behavior; the Hixon Symposium* (pp. 112–146). Wiley.

Lin, S.-S., & Gao, J.-F. (2022). Woman diagnosed with obsessive-compulsive disorder became delusional after childbirth: A case report. *World Journal of Clinical Cases*, *10*(10), 3261–3267. https://doi.org/10.12998/wjcc.v10.i10.3261

Manoach, D. S. (2009). Cognitive Deficits in Schizophrenia. In L. R. Squire (Ed.), *Encyclopedia of Neuroscience* (pp. 1101–1109). Academic Press. https://doi.org/10.1016/B978-008045046-9.00425-3

McLaughlin, N. C. R., Kirschner, J., Foster, H., O'Connell, C., Rasmussen, S. A., & Greenberg, B. D. (2016). Stop Signal Reaction Time Deficits in a Lifetime Obsessive-Compulsive Disorder Sample. *Journal of the International Neuropsychological Society: JINS*, *22*(7), 785–789. https://doi.org/10.1017/S1355617716000540

Menon, V. (2013). Juvenile obsessive-compulsive disorder: A case report. *Industrial Psychiatry Journal*, *22*(2), 155–156. https://doi.org/10.4103/0972-6748.132932

Menzies, L., Chamberlain, S. R., Laird, A. R., Thelen, S. M., Sahakian, B. J., & Bullmore, E. T. (2008). Integrating evidence from neuroimaging and neuropsychological studies of obsessive-compulsive disorder: The orbitofronto-striatal model revisited. *Neuroscience and Biobehavioral Reviews*, *32*(3), 525–549. https://doi.org/10.1016/j.neubiorev.2007.09.005

Mochcovitch, M. D., da Rocha Freire, R. C., Garcia, R. F., & Nardi, A. E. (2014). A systematic review of fMRI studies in generalized anxiety disorder: Evaluating its neural and cognitive basis. *Journal of Affective Disorders*, *167*, 336–342. https://doi.org/10.1016/j.jad.2014.06.041

Modirrousta, M., Meek, B. P., Sareen, J., & Enns, M. W. (2015). Impaired trial-by-trial adjustment of cognitive control in obsessive compulsive disorder improves after deep repetitive transcranial magnetic stimulation. *BMC Neuroscience*, *16*(1), 63. https://doi.org/10.1186/s12868-015-0205-z

Moritz, S., Hübner, M., & Kluwe, R. (2004). Task switching and backward inhibition in obsessive-compulsive disorder. *Journal of Clinical and Experimental Neuropsychology*, *26*(5), 677–683. https://doi.org/10.1080/13803390409609791

Page, L. A., Rubia, K., Deeley, Q., Daly, E., Toal, F., Mataix-Cols, D., Giampietro, V., Schmitz, N., & Murphy, D. G. M. (2009). A functional magnetic resonance imaging study of inhibitory control in obsessive-compulsive disorder. *Psychiatry Research*, *174*(3), 202–209. https://doi.org/10.1016/j.pscychresns.2009.05.002

Penhune, V. B., & Steele, C. J. (2012). Parallel contributions of cerebellar, striatal and M1 mechanisms to motor sequence learning. *Behavioural Brain Research*, *226*(2), 579–591. https://doi.org/10.1016/j.bbr.2011.09.044

Rachman, S. (1997). A cognitive theory of obsessions. *Behaviour Research and Therapy*, *35*(9), 793–802. https://doi.org/10.1016/S0005-7967(97)00040-5

Rapee, R. M., Craske, M. G., & Barlow, D. H. (1994). Assessment instrument for panic disorder that includes fear of sensation-producing activities: The Albany Panic and Phobia Questionnaire. *Anxiety*, *1*(3), 114–122. https://doi.org/10.1002/anxi.3070010303

Reiss, J. P., Campbell, D. W., Leslie, W. D., Paulus, M. P., Stroman, P. W., Polimeni, J. O., Malcolmson, K. A., & Sareen, J. (2005). The role of the striatum in implicit learning: A functional magnetic resonance imaging study. *Neuroreport*, *16*(12), 1291–1295. https://doi.org/10.1097/01.wnr.0000175615.93312.1a

Remijnse, P. L., Nielen, M. M. A., van Balkom, A. J. L. M., Cath, D. C., van Oppen, P., Uylings, H. B. M., & Veltman, D. J. (2006). Reduced orbitofrontal-striatal activity on a reversal learning task in obsessive-compulsive disorder. *Archives of General Psychiatry*, *63*(11), 1225–1236. https://doi.org/10.1001/archpsyc.63.11.1225

Roth, R. M., Saykin, A. J., Flashman, L. A., Pixley, H. S., West, J. D., & Mamourian, A. C. (2007). Event-related functional magnetic resonance imaging of response inhibition in obsessive-compulsive disorder. *Biological Psychiatry*, *62*(8), 901–909. https://doi.org/10.1016/j.biopsych.2006.12.007

Rueppel, M., Mannella, K. A., Fitzgerald, K. D., & Schroder, H. S. (2022). Post-error slowing in anxiety and obsessive-compulsive disorders. *Cognitive, Affective & Behavioral Neuroscience*, *22*(3), 610–624. https://doi.org/10.3758/s13415-021-00976-9

Saxena, S., & Rauch, S. L. (2000). FUNCTIONAL NEUROIMAGING AND THE NEUROANATOMY OF OBSESSIVE-COMPULSIVE DISORDER. *Psychiatric Clinics of North America*, *23*(3), 563–586. https://doi.org/10.1016/S0193-953X(05)70181-7

Schneider, D. W., & Logan, G. D. (2006). Hierarchical control of cognitive processes: Switching tasks in sequences. *Journal of Experimental Psychology. General*, *135*(4), 623–640. https://doi.org/10.1037/0096-3445.135.4.623

Scott, W. A. (1962). Cognitive Complexity and Cognitive Flexibility. *Sociometry*, *25*(4), 405–414. https://doi.org/10.2307/2785779

Shin, N. Y., Lee, T. Y., Kim, E., & Kwon, J. S. (2014). Cognitive functioning in obsessive-compulsive disorder: A meta-analysis. *Psychological Medicine*, *44*(6), 1121–1130. https://doi.org/10.1017/S0033291713001803

Stein, D. J. (2002). *Cognitive-Affective Neuroscience of Depression and Anxiety Disorders*. CRC Press. https://doi.org/10.3109/9780203639740

Taylor, S., Abramowitz, J., & Mckay, D. (2006). Cognitive-behavioral models of obsessive-compulsive disorder. In *Psychological treatment of OCD: Fundamentals and beyond* (pp. 9–29). https://doi.org/10.1037/11543-001

van Velzen, L. S., Vriend, C., de Wit, S. J., & van den Heuvel, O. A. (2014). Response inhibition and interference control in obsessive-compulsive spectrum disorders. *Frontiers in Human Neuroscience*, *8*, 419. https://doi.org/10.3389/fnhum.2014.00419

Veale, D., & Roberts, A. (2014). Obsessive-compulsive disorder. *BMJ*, *348*, g2183. https://doi.org/10.1136/bmj.g2183

Wolff, N., Giller, F., Buse, J., Roessner, V., & Beste, C. (2018). When repetitive mental sets increase cognitive flexibility in adolescent obsessive–compulsive disorder. *Journal of Child Psychology and Psychiatry*, *59*(9), 1024–1032. https://doi.org/10.1111/jcpp.12901

Wood, C. C., & Jennings, J. R. (1976). Speed-accuracy tradeoff functions in choice reaction time: Experimental designs and computational procedures. *Perception & Psychophysics*, *19*(1), 92–102. https://doi.org/10.3758/BF03199392

Wood, J., Mathews, A., & Dalgleish, T. (2001). Anxiety and Cognitive Inhibition. *Emotion*, *1*, 166–181. https://doi.org/10.1037/1528-3542.1.2.166

Youssef, A. M., AbouHendy, W., Elshabrawy, A., & Amin, S. I. (2020). Executive function in obsessive compulsive disorder at Zagazig University Hospitals: A case-control study. *Middle East Current Psychiatry*, *27*(1), 27. https://doi.org/10.1186/s43045-020-00033-0

Yu, Y., Jiang, C., Xu, H., Yang, Q., Li, J., Xu, Y., Xiang, W., Peng, L., Liu, B., Lv, F., & Li, M. (2018). Impaired Cognitive Control of Emotional Conflict in Trait Anxiety: A Preliminary Study Based on Clinical and Non-Clinical Individuals. *Frontiers in Psychiatry*, *9*. https://www.frontiersin.org/articles/10.3389/fpsyt.2018.00120

Zimmerman, M. E. (2011). Speed–Accuracy Tradeoff. In J. S. Kreutzer, J. DeLuca, & B. Caplan (Eds.), *Encyclopedia of Clinical Neuropsychology* (pp. 2344–2344). Springer. https://doi.org/10.1007/978-0-387-79948-3_1247