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Original Article

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Neurobehavioral correlates of changing one's mind in ADHD and OCD

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Cognitive flexibility is an executive function that allows individuals to adjust behavior in response to changing environmental demands. We assessed volitional switching under uncertainty, without rule-based learning, in the "Change Your Mind" task. Nineteen patients with obsessive-compulsive disorder (OCD), 19 patients with attention-deficit hyperactivity disorder (ADHD) and matched control participants (20 per group) completed the task while undergoing a functional MRI scan. The task was a two-alternative forced choice paradigm where each stimulus was presented twice successively, with spurious feedback following the first presentation. This allowed participants the opportunity to repeat or change their response. Participants with ADHD changed their response more frequently than controls following a previously correct response, associated with reduced accuracy on the second trial. This was accompanied with smaller differences between change and repeat trials in the superior frontal gyrus, paracingulate gyrus, and frontal pole compared to controls. Participants with OCD did not differ from healthy controls in their performance but exhibited greater activity on both change and repeat trials in the pre- and postcentral gyri than controls. These results point to distinct neurobehavioral differences in patients with ADHD and OCD underlying what is often termed more broadly inflexible behavior.

Keywords attention-deficit hyperactivity disorder, cognitive flexibility, obsessive compulsive disorder, proactive control

Introduction

Cognitive flexibility refers to the ease with which one can adjust mental processes to adapt behavior to changing environmental demands (Dajani and Uddin 2015). It is a central executive function, alongside other control processes such as inhibition and updating (Miyake et al. 2000). Higher levels of flexibility have been associated with positive life outcomes such as healthy aging (Burke et al. 2019), creativity (Chen et al. 2014), and communication competence (Martin and Anderson 1998). Conversely, impairments in cognitive flexibility may play a role in the pathophysiology of numerous neuropsychiatric disorders. For example, both patients with attention-deficit hyperactivity disorder (ADHD) and with obsessive-compulsive disorder (OCD) exhibit impairments in behavioral flexibility (Boonstra et al. 2005; Chamberlain et al. 2021).

Cognitive flexibility is a multidimensional construct, with behavioral paradigms assessing related but nonetheless differing aspects (Dias et al. 1996; Dajani and Uddin 2015). Existing paradigms that assess flexibility, such as the Wisconsin Card-Sorting Task (WCST) (Grant and Berg 1948), the CANTAB intra-dimensional/extra-dimensional set-shifting (IED) task (Chamberlain et al. 2021), and probabilistic reversal learning (PRL) (Izquierdo et al. 2017), frequently capture reactive flexible responding, in which behavioral adjustments are triggered by external signals indicating a need to change. However, less is known about proactive flexibility, in which these adjustments are initiated by internally generated signals and volitional choices. In a previous study, we presented a novel behavioral paradigm that focuses on proactive flexibility (Zühlsdorff et al. 2023). Forty healthy participants completed a two-alternative forced choice task in which a masked stimulus was presented twice, with feedback orthogonal to their performance being presented after the first response. Following the second stimulus presentation, participants were free to either repeat or change their first response. This enabled us to assess the behavioral and neural correlates of "the road not taken." Given the importance of proactive cognitive flexibility in everyday functioning, we sought to evaluate whether this process is altered in patient groups that have been reported to exhibit impairments in cognitive flexibility.

ADHD is a neurodevelopmental disorder affecting children, adolescents, and adults and is characterized by hyperactivity, impulsivity, and attention difficulties (Faraone et al. 2015). Individuals with ADHD show impairments across several executive functions, including cognitive flexibility, attention, and inhibition (Nigg et al. 2005). Impaired cognitive flexibility is also a key feature of OCD (Robbins et al. 2019), which is a debilitating psychiatric condition characterized

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by intrusive thoughts or behavioral urges that can lead to checking, washing, worrying, and anxiety (American Psychiatric Association 2013). Although both disorders have been associated with difficulties in cognitive flexibility, the mechanisms underlying these difficulties likely differ between them.

Cognitive flexibility is typically investigated using tasks that require participants to respond to an explicit change in task demands. Tasks such as the WCST and IED have identified impairments in flexibility in both ADHD and OCD, including increased perseverative errors and slower or less accurate responding following a shift (Chamberlain et al. 2007; King et al. 2007; Rohlf et al. 2012; Snyder et al. 2015; Alaghband-Rad et al. 2021). However, in everyday behavior, flexibility does not always involve an explicit signal that a change is required. Instead, individuals often need to decide whether to maintain or change a response based on their evaluation of the preceding outcome. This represents a more proactive form of flexibility, in which individuals monitor recent performance and determine whether the available evidence warrants changing their behavior. Our previously published "Change Your Mind" task provides an opportunity to examine flexibility as a volitional decision rather than as a direct response to an externally imposed change in task demands. In healthy participants, changing one's response was associated with increased activation in the anterior insula (AI), anterior cingulate cortex (ACC), inferior frontal junction (IFJ), and dorsolateral prefrontal cortex (dlPFC) (Zühlsdorff et al. 2023). These regions are important for functions beyond flexibility. The AI and ACC are implicated in salience and performance monitoring (Carter et al. 1998; Van Veen et al. 2001), while the IFJ and dlPFC support cognitive control and the maintenance and updating of task-relevant information (Cai et al. 2014; Dajani and Uddin 2015).

Proactive switching may therefore reflect an interaction between flexibility and error monitoring under uncertainty. This distinction may be particularly relevant to ADHD and OCD, as both disorders have been associated with altered activation of frontostriatal regions not only during reactive flexibility tasks, but also on tasks of error monitoring and uncertainty evaluation (Rubia et al. 2011; Hauser et al. 2017). During task switching, adults with ADHD exhibited increased activation in the ACC and AI, whereas activity during PRL demonstrated reduced activation of the left posterior parietal cortex (Dibbets et al. 2010; Aster et al. 2024). Impaired error monitoring has also been proposed to be a central feature of ADHD (Geburek et al. 2013). In OCD, functional MRI studies have found reduced frontostriatal activation in OCD alongside perseverative errors in PRL tasks (Remijnse et al. 2006; Chamberlain et al. 2008). On task-switching paradigms, patients diagnosed with OCD show reduced activation of the OFC and dlPFC and increased activation of the putamen, ACC, and postcentral gyrus (Chamberlain et al. 2008; Gu et al. 2008; Remijnse et al. 2013). Studies have also shown that activation in the ACC and AI does not distinguish between high- and low-uncertainty decision processes in participants with OCD (Zhao et al. 2023), and that there is greater error-related activation in the ACC compared to controls in this group (Fitzgerald et al. 2005). In children with ADHD, the cingulate cortex has also been shown to be underactivated during error processing (Rubia et al. 2011). Therefore, it is important to further investigate whether these neural differences also apply to proactive flexibility tasks is important, as well as their behavioral differences.

In this study, we report the behavioral and neural correlate underlying the "Change Your Mind" task in patients diagnosed with either OCD or ADHD compared with their sex- and age-matched control groups. In our previous study, we found that negative feedback and an incorrect first response independently resulted in increased changing behavior (Zühlsdorff et al. 2023). Behaviorally, we hypothesized that individuals with ADHD would be more likely to change their response on the second trial following negative feedback due to a heightened sense of uncertainty compared with controls. For patients with OCD, we predicted reduced overall switching regardless of feedback, reflecting the need to alleviate uncertainty through repetitive actions. At a neural level, we anticipated increased activations in the ADHD group and reduced activity in the OCD group in the IFJ, AI, ACC, and dlPFC, which are areas we have previously reported to be associated with changing a response (Zühlsdorff et al. 2023). The primary objective of this study is to unravel performance and the neural correlates of proactive cognitive flexibility in patients with ADHD and OCD.

Materials and methods

Participants

Participants were 78 individuals, which were allocated to one of four groups: ADHD ($N = 19$, N females = 6, mean age = 27.7), ADHD control group ($N = 20$, N females = 6, mean age = 29.11), OCD ($N = 19$, N females = 15, mean age = 35.15), and OCD control group ($N = 20$, N females = 15, mean age = 37.79). Controls for the ADHD and OCD groups were age- and sex-matched (Table 1). All volunteers were aged between 18 and 60 years (Mean = 32.14, SD = 9.98) and were recruited via the University of Cambridge Behavioral and Clinical Neuroscience Institute volunteer panel, using adverts in the Cambridge community or from the Cambridgeshire and Peterborough Foundation NHS Trust and from local support groups.

Patients were diagnosed according to the DSM-IV criteria through an interview with a psychiatrist or clinical psychologist. OCD patients did not meet the DSM-IV criteria for any other Axis I disorders, except two who met the criteria for generalized anxiety disorder. Thirteen OCD patients were on serotonin reuptake inhibitors and one on a tricyclic antidepressant. Exclusion criteria for the OCD group were: substance abuse in the last 3 months, psychotic disorders, schizophrenia, bipolar disorder, or ADHD. Nine of the ADHD patients were unmedicated (of which two were previously medicated), nine were prescribed methylphenidate, and one was taking atomoxetine. On the day of testing, ADHD patients had not taken their medication for the last 24 h. ADHD patients did not meet the DSM-IV criteria for any other disorders. Exclusion criteria for the ADHD group were: substance abuse in the last three months, psychotic disorders, schizophrenia, bipolar disorder or major depressive disorder, or OCD.

Verbal IQ was assessed using the National Adult Reading Test score (Mean = 115.56, SD = 8.68) (Nelson 1982). Additional clinical information was collected from the following questionnaires: Adult ADHD Self-Report Scale (ASRS), Conners' Adult ADHD Rating Scales (CAARS), Obsessive Compulsive Inventory-Revised (OCI-R), and Yale-Brown Obsessive Compulsive Scale (Y-BOCS). All participants provided informed written consent and were reimbursed for their time and travel expenses, with ethical approval granted from the Cambridge Local Research Ethics Committee (08/H0308/65). These data were collected together with other data not reported here from the same participants (Morein-Zamir et al. 2014, 2016) and results from control participants were also previously reported (Zühlsdorff et al. 2023).

Task performance

The "Change Your Mind" task has been described in detail previously (Zühlsdorff et al. 2023) and is also described in the Supplementary Materials, alongside details on neuroimaging acquisition. The primary outcome measure was the mean change in response on the second

Table 1 Participant demographics and clinical characteristics.

Group	Control ADHD (*N* = 20)	ADHD (*N* = 19)	Group comparison statistics	Control OCD (*N* = 20)	OCD (*N* = 19)	Group comparison statistics
Mean age (SD)	27.65 (5.83)	29.11 (7.72)	$t(37) = -0.66$ $P = 0.51$	35.15 (11.6)	37.79 (10.1)	$t(37) = -0.76$ $P = 0.45$
N females	6	6	$\chi^2(1) = 0.02$ $P = 0.90$	15	14	$\chi^2(1) = 0.02$ $P = 0.88$
Mean verbal IQ (SD)	115.9 (9.60)	115.6 (9.47)	$t(37) = 0.10$ $P = 0.92$	117.5 (6.30)	113.3 (9.11)	$t(37) = 0.10$ $P = 0.92$
OCIR (SD)	13.23 (6.88)	22.16 (11.20)	$t(37) = -2.98$ $P = 0.006$ $d = 0.97$	11.55 (6.80)	26.74 (13.65)	$t(37) = -4.36$ $P < 0.001$ $d = 1.42$
Y-BOCS (SD)	0.75 (1.07)	1.42 (2.25)	$t(37) = -1.18$ $P = 0.25$	0.55 (1.15)	18.95 (7.33)	$t(37) = -10.82$ $P < 0.001$ $d = 3.55$
ASRS (SD)	26.10 (7.20)	48.53 (11.80)	$t(37) = -7.12$ $P < 0.001$ $d = 2.31$	24.45 (8.24)	32.00 (9.97)	$t(37) = -2.57$ $P = 0.015$ $d = 0.83$
CAARS (SD)		49.3 (8.90)				

t, *t*-statistic; *P*, *P*-value; *d*, Cohen's *d*; OCIR, obsessive compulsive inventory; Y-BOCS, Yale Brown obsessive compulsive scale; ASRS, adult ADHD self-report scale; CAARS, Conners' adult ADHD rating scales.

presentation of each trial (R2). A secondary outcome measure was R2 accuracy. Data were analyzed using linear mixed-effects models (LMEs) implemented in R version 4.0.4 (R Core Team 2021) using the **lme4** package (Bates et al. 2015). Fixed effects included feedback (positive vs negative), accuracy on the first response (R1: correct vs incorrect), group (control vs ADHD or control vs OCD), and all interactions. Post hoc pairwise comparisons of estimated marginal means were carried out using the **emmeans** package (Lenth et al. 2018; Pinheiro et al. 2021). Multiple comparisons were adjusted using Tukey's method. Three different LMEs were assessed, differing in their random intercepts and interactions. Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), and log-likelihood were used to compare the models. The best-fitting model, which yielded the lowest AIC and BIC values, included random intercepts for subjects: Mean change R2 ~ Feedback × R1 accuracy × Group + (1 + subject).

To examine the relationship between task performance and dimensional symptom severity, correlation analyses were conducted between mean changing behavior and obsessive-compulsive symptoms (OCI-R total score), as well as ADHD symptoms (CAARS total score). Pearson's correlation coefficients were calculated.

Imaging data

First-level and higher-level models

Information on preprocessing can be found in the Supplementary Materials. A first-level fMRI linear model was fit using FEAT (FSL) (Woolrich et al. 2001) for each run and included the following event types: (1) R1 correct, (2) R1 incorrect, (3) positive feedback, (4) negative feedback, (5) R2 change, and (6) R2 repeat. Equivalent event types for easy trials and six movement parameters (*x*, *y*, *z*, pitch, roll, yaw) resulting from the image realignment to control for movement artifacts were also included. Contrasts included only difficult trials and consisted of: (1) R1 incorrect versus correct, (2) negative versus positive feedback, and (3) R2 change versus repeat.

The second-level model was constructed by averaging the first-level results for each participant across the three runs. The second-level contrasts were then entered into group-level (third-level) mixed-effects analyses using two-sample *t*-tests to compare the groups. Whole-brain results were thresholded with a cluster-forming threshold of $Z = 3.1$ and a cluster-corrected significance level of $P < 0.05$ to control for multiple comparisons (Woolrich et al. 2004). FSLeyes was used to create the figures (Smith et al. 2004). The main event types of interest were R2 change, R2 repeat, positive feedback, negative feedback, R2 change versus repeat, and negative versus positive feedback.

Connectivity analysis

Additionally, whole-brain psychophysiological interaction (PPI) analyses were conducted in FSL. Three regions-of-interest ("seeds") were selected based on regions reported to mediate proactive cognitive flexibility (Dajani and Uddin 2015; Uddin 2021). The regions selected were the AI, ACC, and inferior frontal gyrus (IFG). A 5-mm sphere was placed in the center of the respective region based on the Harvard-Oxford Atlas. Mean time series for each participant and each ROI were extracted.

This analysis assesses if there is an interaction between the ROI time series and a cognitive process which accounts for the neural responses observed in other brain regions (Neufang et al. 2008). This requires the addition of extracted time series, the response to the cognitive process of interest (change vs repeat) and an interaction term between them in the first-level model. Contrasts tested differences in connectivity in the change and the repeat condition in patients versus controls (ADHD vs controls or OCD vs controls). Two-sample *t*-tests for each seed region were conducted and clusters above a *z*-threshold of 2.5 and $P < 0.05$ were considered significant.

Results

R2 change and accuracy in ADHD versus controls

A comparison of mean R2 change between ADHD and the matched control group revealed a significant effect of group ($F(1,37) = 7.85$, $P = 0.008$, $\eta^2 P = 0.17$), with overall higher changing behavior in the ADHD group (control: mean = 0.28, ADHD: mean = 0.38) (Fig. 1A).

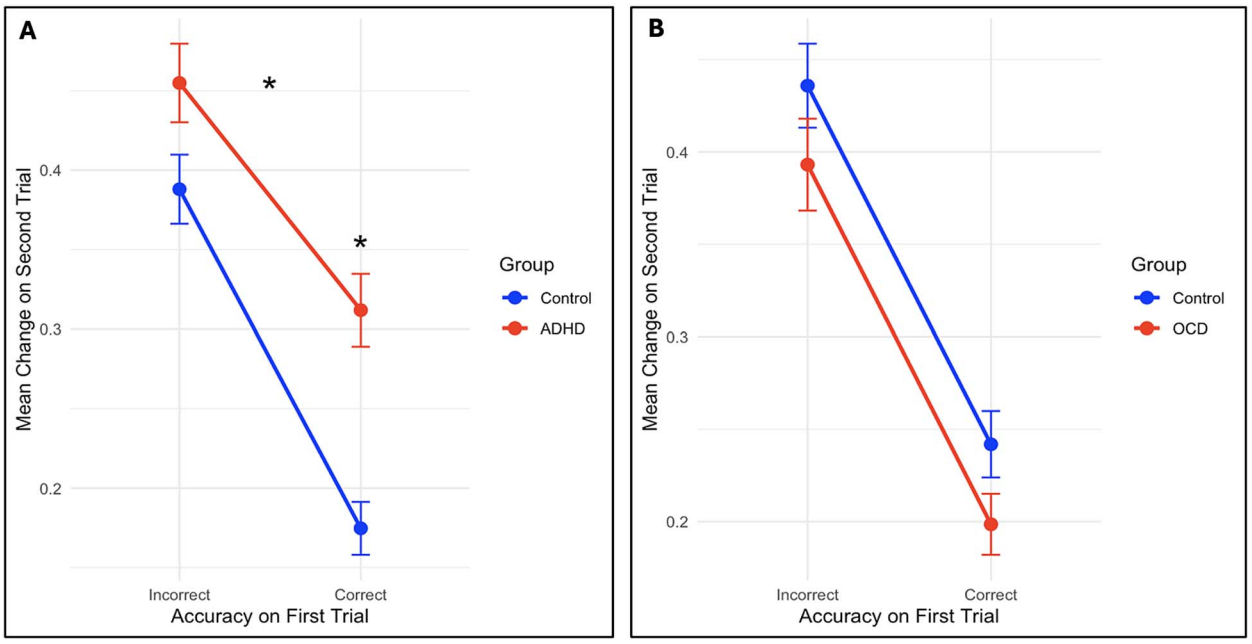


Figure 1 Effect of accuracy and group on R2 change. (A) ADHD group: The linear mixed-effects model for R2 change indicated significant Group and Group × Accuracy on the first trial (Acc1) effects. Participants with ADHD changed on the second trial more overall, with an especially increased R2 change following a correct response on the first trial. (B) OCD group: The linear mixed-effects model for mean R2 change showed no differences between the OCD and control groups. Error bars represent standard error of the mean. $^{*}P < 0.05$.

There was also a significant effect of feedback on R2 change across all participants ($F(1,423) = 129$, $P < 0.001$, $\eta^2P = 0.23$), with higher R2 change following negative feedback (negative feedback: mean = 0.43, positive feedback: mean = 0.23); however, there was no significant group × feedback interaction. R1 Accuracy also affected mean R2 change ($F(1,423) = 105$, $P < 0.001$, $\eta^2P = 0.20$). Mean change was higher when the first trial was incorrect (correct: mean = 0.24, incorrect: mean = 0.42). Furthermore, there was a significant Group × R1 Accuracy interaction ($F(1,423) = 4.08$, $P = 0.044$, $\eta^2P = 9.55 \times 10^{-3}$). Post hoc analyses found that there was higher switching in the ADHD group after a correct response compared to the control group ($t(56) = 3.40$, $P = 0.007$). There were no significant differences in mean changing after an incorrect R1.

As a secondary measure, we also assessed the mean accuracy on the second trial (mAcc2). There was a significant effect of group on mAcc2 ($F(1,37) = 6.22$, $P = 0.017$, $\eta^2P = 0.14$), with lower mAcc2 observed in the ADHD group (control: mean = 0.60, ADHD: mean = 0.55). R1 accuracy influenced the accuracy on the second trial ($F(423) = 271$, $P < 0.0001$, $\eta^2P = 0.39$), with reduced accuracy following an incorrect first trial (correct: mean = 0.74, incorrect: mean = 0.41). We also found a Group × Acc1 interaction ($F(1,423) = 26.76$, $P < 0.0001$, $\eta^2P = 0.06$) (Fig. 2a). Post hoc comparisons showed that the accuracy on the second trial was lower in the ADHD group when participants were correct on the first trial ($t(129) = 5.39$, $P < 0.0001$) but there was no group difference when participants were incorrect.

R2 change and accuracy in OCD versus controls

Analyses of mean R2 change showed that feedback significantly affected mean R2 change ($F(1,411) = 42.86$, $P < 0.001$, $\eta^2P = 0.09$), with higher R2 change after negative feedback (positive: mean = 0.27, negative: mean = 0.37), however, there was no significant group × feedback interaction. R1 accuracy also affected mean R2 change ($F(1,411) = 152.41$, $P < 0.001$, $\eta^2P = 0.27$), with lower values following a correct response (correct: mean = 0.22, incorrect: mean = 0.42). However, no significant effects of group or interactive effects were found (Fig. 1B).

The secondary measure of mAcc2 also did not show any effects of group or any significant interactions (Fig. 2b). There was a significant effect of Acc1 ($F(1,447) = 294$, $P < 0.0001$, $\eta^2P = 0.40$). There was also a significant Feedback × Acc1 interaction ($F(1,447) = 21.19$, $P < 0.0001$). Both positive feedback and correct Acc1 resulted in the highest mAcc2 compared to negative feedback and incorrect Acc1 ($t(411) = 12.3$, $P < 0.0001$).

Brain activations when changing versus repeating a response in ADHD versus controls

The primary imaging outcome was group differences in the activations underlying changing or repeating a response. There were no significant differences between the two groups in the change or repeat conditions separately. Comparing the change versus repeat contrast between the two groups revealed a larger difference between these two conditions in the control group than the ADHD group. This was observed in the superior frontal gyrus (SFG), paracingulate gyrus, right frontal pole, and the right lateral occipital cortex (Fig. 3).

Brain activations when changing and repeating a response in OCD versus controls

When comparing the change and repeat contrasts in individuals with OCD versus controls, greater activity in the left pre- and postcentral gyri (including Brodmann area 4) in the OCD group were observed when both changing and repeating a response compared to control participants (Fig. 4). No differences in the repeat versus change contrast were observed between the two groups.

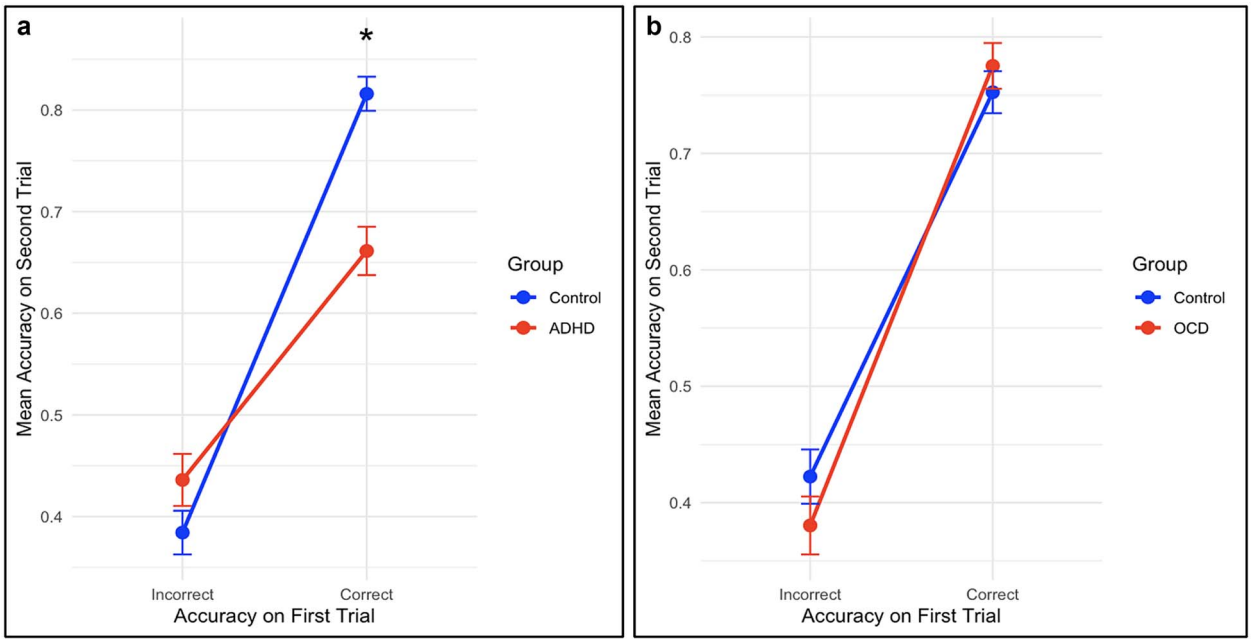


Figure 2 Effect of accuracy on the first trial and group on mean accuracy on the second trial. (a) ADHD group: The linear mixed-effects model for mean accuracy on the second trial (mAcc2) had significant Group and Group × Accuracy on the first trial (Acc1) effects. Participants with ADHD showed lower accuracy on the second trial when the first trial was correct. (b) OCD group: The linear mixed-effects model for mAcc2 showed no differences between the OCD and control groups. Error bars represent standard error of the mean. $*P < 0.05$.

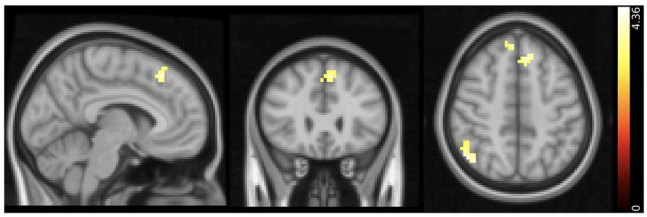


Figure 3 Results summary of the differences in change vs repeat contrast in control vs ADHD groups. Cross-section (MNI $x = -6$, $y = 27$, $z = 48$) of the contrast highlighting key regions activated more when participants changed compared to when they repeated their responses. This showed significantly greater differences between the two event types in controls than participants with ADHD, ie in controls, the difference in BOLD activity between the change and the repeat trials was greater than in individuals with ADHD. Activations detected with a whole-brain analysis involving one-sample *t*-tests with cluster thresholding with a *Z*-threshold of 3.1 and $P < 0.05$. The color bar represents the *t*-statistic.

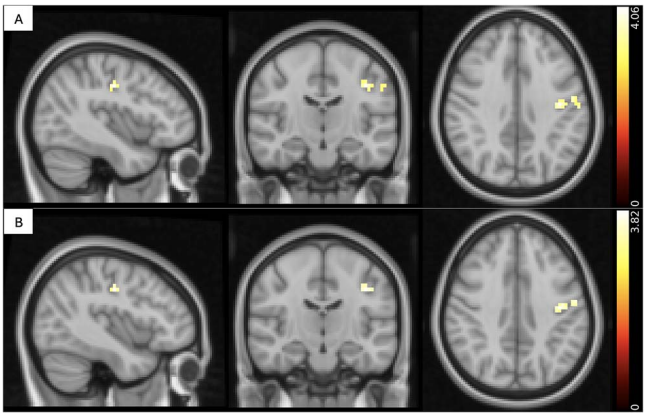


Figure 4 Results summary of the differences in the change and repeat contrast in control vs OCD groups. Cross-section of the contrast highlighting key regions activated more in the OCD group than in controls when participants (a) changed (MNI $x = -40$, $y = -16$, $z = 38$) or (b) repeated (MNI $x = -6$, $y = 27$, $z = 48$) their responses (pre- and postcentral gyrus) . Activations detected with a whole-brain analysis involving one-sample *t*-tests with cluster thresholding with a *Z*-threshold of 3.1 and $P < 0.05$. The color bar represents the *t*-statistic.

Brain activations when receiving negative and positive feedback in patients

Furthermore, we investigated differences in brain activations following the presentation of positive or negative feedback. The contrast between the two feedback conditions was also compared between the two groups. When comparing the ADHD and control groups, no significant differences were found when positive or negative feedback were presented. However, the contrast between negative and positive feedback showed a greater difference in BOLD-dependent activity between these two events (negative vs positive feedback) in the control than in the ADHD group (Supplementary Fig. S2). This was observed in the right IFG, right precentral gyrus, and right lateral occipital cortex.

When receiving negative feedback, we observed that patients with OCD showed lower activity in the right and left SFG, cingulate gyrus, and paracingulate gyrus compared to the control group (Supplementary Fig. S3). No significant differences during the presentation of positive feedback or in the negative versus positive feedback contrast were found.

Neural connectivity when changing or repeating a response

The results from the PPI analyses in ADHD and their control group demonstrated differential interactions between brain regions when changing or repeating a response in the task. Specifically, we observed

that during change trials there was lower connectivity between the IFG seed and the right and left precuneus cortex in the ADHD group (Supplementary Fig. S4). Additionally, during repeat trials, there was greater connectivity in the same area and the right and left lingual gyri in the ADHD group compared to controls. No differences in the connectivity between the IFG and other brain regions were found in the change versus repeat contrast between the two groups. There were also no significant effects observed with the AI and ACC seeds.

A comparison of the OCD group with controls during change or repeat trials highlighted differences in functional connectivity between the AI and frontal pole in both change and repeat trials (Supplementary Fig. S5). Specifically, there was greater connectivity in the OCD group between the AI and left frontal pole during change trials, but reduced connectivity compared with the control group during repeat trials (Supplementary Fig. S6). No other significant effects where observed.

Discussion

We report distinct behavioral and neuroimaging findings from ADHD and OCD patients performing the "Change Your Mind" task. Our findings provide new information on an alternative form of flexibility and how it differs in these two patient groups. We replicated the findings from our previous study across all groups, specifically, that higher levels of changing are seen after negative compared to positive feedback (Zühlsdorff et al. 2023). Higher levels of changing were also seen when the response on the first trial was incorrect, which was also seen in our previous study. This form of flexibility has been poorly investigated to date: behavioral changes representing volitional control rather than task-set switching, rule-based learning, or set shifting. Our results showed that patients with ADHD changed their responses on the second stimulus more frequently than controls, and even more so when their first response was correct, possibly indicating a lack of confidence in their first response. This tendency was associated with lower accuracy on the second trial following a correct response, ie they changed their mind more after their first response, despite their first response being correct. This behavior was accompanied by a smaller difference in the hemodynamic BOLD response when contrasting change versus repeat trials in the SFG, paracingulate gyrus, and frontal pole than in controls. On the other hand, patients with OCD, exhibited no behavioral differences on the task compared with the control group. There was increased activity in the left pre- and postcentral gyri in this group when both changing and repeating a response compared to the control group. They also showed reduced activity following presentation of negative feedback in the SFG, cingulate gyrus, and paracingulate gyrus. Moreover, functional connectivity between the AI and frontal pole increased during change trials but decreased during repeat trials.

A prominent observation in this study was that individuals with ADHD changed their mind more often than controls and even when their first response was correct. This finding is consistent with previous behavioral evidence showing that ADHD is associated with increased switching on a PRL task and heightened trial-to-trial variability reflected in increased exploration rather than exploitation (Luman et al. 2010; Hauser et al. 2014). Studies investigating reinforcement learning mechanisms in ADHD have demonstrated that individuals show reduced sensitivity to positive feedback and have difficulties implementing successful response strategies (Frank et al. 2006; Groen et al. 2013). The increased switching we report in this study therefore likely reflects instability in employing goal-directed actions and an overreliance on reactive, feedback-driven responding. This may be due to an increased reliance on sensory feedback, rather than priors.

The ADHD group also showed a smaller difference between change and repeat trials in prefrontal regions in the ADHD group compared to control participants, possibly suggesting blunted recruitment of the frontoparietal network. One of the functions of this network is to support sustained attention (Prado et al. 2010; Kam et al. 2019). This points to impaired proactive control and potentially reduced confidence in internally generated decisions. The impaired proactive control in ADHD is reflected in lower thresholds to switch responses, resulting in less stable decision-making and is accompanied by a failure to recruit the appropriate neural networks required for switching, which include prefrontal cortical areas such as the SFG and paracingulate gyrus. There was also a smaller difference in the BOLD-response in the IFG during presentation of negative versus positive feedback in ADHD compared with controls. Previously, this same ADHD group showed more commission errors and reduced activation in the IFG during no-go trials on a combined shifting go/no-go task, suggestive of impaired response inhibitory control (Morein-Zamir et al. 2014). Other studies have also reported hypoactivation of the IFG and ventrolateral PFC, as well as overactivation of the visual cortex in patients with ADHD on flexibility tasks (Rubia et al. 2005; Cortese et al. 2012). The impaired control signal in the IFG previously noted in this sample as well as the differential response during feedback reported in this study may both contribute to the abnormal shifting behavior exhibited in ADHD.

Consistent with the differences in clinical presentation between ADHD and OCD, the behavioral and neural findings in the OCD group were markedly different. This aligns with findings from the go/no-go task in the same samples (Morein-Zamir et al. 2014, 2016). Unexpectedly, task performance revealed no behavioral differences between the OCD and control groups, suggesting that flexible choice driven by volitional rather than rule-based control is unimpaired in OCD. The lack of behavioral differences contrasts with previous studies reporting impairments in OCD in several paradigms assessing flexibility, such as the WCST (Bohon et al. 2020), the IED set-shifting task (Chamberlain et al. 2021), deterministic reversal learning (Gu et al. 2008; Apergis-Schoute et al. 2024), and probabilistic reversal learning tasks (Remijnse et al. 2005; Apergis-Schoute et al. 2024). Nevertheless, there is some occasional heterogeneity in performance of OCD patients on cognitive flexibility tasks (see Chamberlain et al. 2021). Moreover, WCST performance was shown not to be impaired in adolescents with OCD (Marzuki et al. 2021) and performance on the probabilistic reversal task in OCD is often characterized by reduced "stickiness" in response repetition (Kanen et al. 2019; Apergis-Schoute et al. 2024). Additionally, patients with OCD often do not show impaired flexibility in task-switching paradigms (switch costs), instead showing overall slowing (Gu et al. 2008) or more errors (Remijnse et al. 2013; Liu et al. 2023). This pattern of findings is consistent with cognitive flexibility reflecting multiple component processes and OCD only being impaired in some (Dajani and Uddin 2015; Gruner and Pittenger 2017). In our previously published study on this OCD sample, which was free from comorbid depression, in a test combining go/no-go performance with set-shifting performance in the latter also did not differ from controls (Morein-Zamir et al. 2016). Moreover, two thirds of the patients were receiving selective serotonin reuptake inhibitor medication which might have aided cognitive flexibility based on recent findings (Apergis-Schoute et al. 2017; Conceição et al. 2026).

Despite the lack of behavioral differences, at the neural level the OCD group exhibited some, albeit subtle, differences from controls.

Greater activity in the left precentral and post central gyri was observed during both staying and changing in the OCD group compared to controls. This could be interpreted as evidence of greater cognitive effort to aid decisional performance. Altered connectivity between the frontal pole and AI specifically during change trials is consistent with prior findings (Stern et al. 2011; Grützmann et al. 2021) and potentially reflects heightened monitoring of decision outcomes supporting this compensatory interpretation. This change in connectivity may possibly be reflective of greater cognitive effort when changing behavior, a process reliant on the interaction between the AI and the frontoparietal control network (Jiang et al. 2015). It is notable, however, that this same group of OCD patients, however, exhibited significant *under*-activation of the frontoparietal network during set-shifting performance when behavioral performance was similarly preserved (Morein-Zamir et al. 2016). The SFG and cingulate cortex, which are involved in error monitoring (Carter et al. 1998), showed reduced activity following negative feedback in the OCD group. Although this contradicts previous studies showing increased activity in those areas (Fitzgerald et al. 2005; Stern et al. 2011), it could reflect generally altered feedback processing (Agam et al. 2014) and is consistent with a recent meta-analysis of task-based fMRI (Dzinalija et al. 2025).

Assessing ADHD and OCD using the change your mind task has allowed us to highlight different patterns of proactive flexibility despite the prominence of executive dysfunction and cognitive rigidity in the conceptualization of both (Snyder et al. 2015). In the ADHD group, behavioral and neural abnormalities point to diminished top-down processing and impairments in proactive flexibility. In contrast, only subtle differences in the OCD were noted with intact performance and largely effective, sometimes apparently compensatory, recruitment of the relevant brain regions. When abnormalities were observed, they were associated with diminished neural responses to negative feedback and increased neural recruitment during the second response, regardless of whether participants repeated or changed their choice. As this task does not reliably distinguish between internal and external sources of uncertainty in changing one's mind, it remains unclear how each source contributes to performance in the two patient groups. Furthermore, the behavioral and neuroimaging findings should be considered complementary, rather than directly corresponding, as they reflect different aspects of proactive flexibility. Disparate findings during current task performance are in line with the disparate clinical presentations of the two conditions, whereby executive dysfunction more broadly can contribute to impulsivity versus compulsivity (Fineberg et al. 2014; Faraone et al. 2015). A further limitation of the present study concerns differences in medication status across participants. Both the ADHD and OCD groups included individuals taking a range of medications, resulting in considerable heterogeneity within each clinical group. However, as the ADHD group did not take their medication prior to being tested, this group may be considered unmedicated. This was not the case for the OCD group. This variability may have influenced behavioral and neural measures and therefore makes it difficult to disentangle disorder-specific effects from those potentially attributable to pharmacological treatment.

In conclusion, this study highlights differences in volitional changing of behavior in two patient groups characterized by cognitive inflexibility. Our findings suggest that this type of flexibility is selectively altered in patients with ADHD. In patients with OCD, we observed no behavioral differences on this particular type of flexibility but found subtle changes in neural processing that could be indicative of effective compensatory mechanisms. Our results do not highlight any decisional impairment or a lack of confidence in this group. These findings potentially inform future research on the neural and behavioral mechanisms of cognitive flexibility and guide the development of targeted interventions to improve adaptive behavior in individuals with ADHD and OCD.

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Author contributions

Katharina Zühlsdorff (Formal analysis, Investigation, Methodology, Validation, Visualization, Writing—original draft, Writing—review & editing), Jeffrey W. Dalley (Supervision, Writing—review & editing), Trevor W Robbins (Conceptualization, Funding acquisition, Resources, Supervision, Writing—review & editing), and Sharon Morein-Zamir (Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Writing—original draft, Writing—review & editing)

Supplementary material

Supplementary material is available at *Cerebral Cortex* online.

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Conflicts of interest

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References

Agam Y et al. 2014. Aberrant error processing in relation to symptom severity in obsessive–compulsive disorder: a multimodal neuroimaging study. *NeuroImage Clin.* 5:141–151. https://doi.org/10.1016/J.NICL.2014.06.002.

Alaghband-Rad J, Dashti B, Tehranidoost M, Zargarinejad G, Farhadbeigi P. 2021. A preliminary investigation of deficits in executive functions of adults with attention deficit hyperactivity disorder. *J Nerv Ment Dis.* 209:35–39. https://doi.org/10.1097/NMD.0000000000001247.

American Psychiatric Association. 2013. Diagnostic and statistical manual of mental disorders. *American Psychiatric Association.* https://doi.org/10.1176/appi.books.9780890425596.

Apergis-Schoute AM et al. 2017. Neural basis of impaired safety signaling in Obsessive Compulsive Disorder. *Proc Natl Acad Sci USA*. 114: 3216–3221. https://doi.org/10.1073/pnas.1609194114.

Apergis-Schoute AM et al. 2024. Perseveration and shifting in obsessive-compulsive disorder as a function of uncertainty, punishment, and serotonergic medication. *Biol Psychiatry Glob Open Sci*. 4:326–335. https://doi.org/10.1016/j.bpsgos.2023.06.004.

Aster HC et al. 2024. Impaired flexible reward learning in ADHD patients is associated with blunted reinforcement sensitivity and neural signals in ventral striatum and parietal cortex. *NeuroImage Clin*. 42:103588. https://doi.org/10.1016/J.NICL.2024.103588.

Bates D, Mächler M, Bolker B, Walker S. 2015. Fitting linear mixed-effects models using lme4. *J Stat Softw*. 67:1–48. https://doi.org/10.18637/JSS.V067.I01.

Bohon C, Weinbach N, Lock J. 2020. Performance and brain activity during the Wisconsin Card Sorting Test in adolescents with obsessive–compulsive disorder and adolescents with weight-restored anorexia nervosa. *Eur Child Adolesc Psychiatry*. 29:217–226. https://doi.org/10.1007/S00787-019-01350-4/FIGURES/2.

Burke SN et al. 2019. What are the later life contributions to reserve, resilience, and compensation? *Neurobiol Aging*. 83:140–144. https://doi.org/10.1016/J.NEUROBIOLAGING.2019.03.023.

Boonstra AM, Oosterlaan J, Sergeant JA, Buitelaar JK. 2005. Executive functioning in adult ADHD: A meta-analytic review. *Psychol Med*. 35(8):1097–1108. https://doi.org/10.1017/S003329170500499X

Cai W, Ryali S, Chen T, Li CSR, Menon V. 2014. Dissociable roles of right inferior frontal cortex and anterior insula in inhibitory control: evidence from intrinsic and task-related functional parcellation, connectivity, and response profile analyses across multiple datasets. *J Neurosci*. 34(44):14652–67. https://doi.org/10.1523/JNEUROSCI.3048-14.2014.

Carter CS et al. 1998. Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*. 280:747–749. https://doi.org/10.1126/science.280.5364.747.

Chamberlain SR et al. 2007. Impaired cognitive flexibility and motor inhibition in unaffected first-degree relatives of patients with obsessive-compulsive disorder. *Am J Psychiatry*. 164:335–338. https://doi.org/10.1176/AJP.2007.164.2.335.

Chamberlain SR et al. 2008. Orbitofrontal dysfunction in patients with obsessive-compulsive disorder and their unaffected relatives. *Science (New York, NY)*. 321:421–422. https://doi.org/10.1126/SCIENCE.1154433.

Chamberlain SR, Solly JE, Hook RW, Vaghi MM, Robbins TW. 2021. Cognitive inflexibility in OCD and related disorders. *Curr Top Behav Neurosci*. 49:125–145. https://doi.org/10.1007/7854_2020_198.

Chen Q et al. 2014. Association of creative achievement with cognitive flexibility by a combined voxel-based morphometry and resting-state functional connectivity study. *NeuroImage*. 102:474–483. https://doi.org/10.1016/j.neuroimage.2014.08.008.

Conceição VA et al. 2026. Serotonin reduces belief stickiness. *Nat Mental Health*. 4:775–791. https://doi.org/10.1038/s44220-026-00621-9.

Cortese S et al. 2012. Toward systems neuroscience of ADHD: a meta-analysis of 55 fMRI studies. *Am J Psychiatry*. 169:1038–1055. https://doi.org/10.1176/APPI.AJP.2012.11101521;REQUESTEDJOURNAL:JOURNAL:AJP;WGROUP:STRING:PUBLICATION.

Dajani DR, Uddin LQ. 2015. Demystifying cognitive flexibility: implications for clinical and developmental neuroscience. *Trends Neurosci*. 38:571–578. https://doi.org/10.1016/J.TINS.2015.07.003/ASSET/5696C0F5-702D-411B-888B-19F9D0EC976F/MAIN.ASSETS/GR3.SML.

Dias R, Robbins TW, Roberts AC. 1996. Dissociation in prefrontal cortex of affective and attentional shifts. *Nature*. 380(6569):69–72. https://doi.org/10.1038/380069a0.

Dibbets P, Evers EAT, Hurks PPM, Bakker K, Jolles J. 2010. Differential brain activation patterns in adult attention-deficit hyperactivity disorder (ADHD) associated with task switching. *Neuropsychology*. 24:413–423. https://doi.org/10.1037/A0018997.

Dzinalija N et al. 2025. Inhibitory control and error processing in obsessive-compulsive disorder: a mega-analysis of task-based fMRI data by the ENIGMA-OCD consortium. *bioRxiv*. https://doi.org/10.1101/2025.10.22.683868.

Faraone SV et al. 2015. Attention-deficit/hyperactivity disorder. *Nat Rev Dis Primers*. 1:1–23. https://doi.org/10.1038/nrdp.2015.20.

Fineberg NA et al. 2014. New developments in human neurocognition: clinical, genetic, and brain imaging correlates of impulsivity and compulsivity. *CNS Spectr*. 19:69–89. https://doi.org/10.1017/S1092852913000801.

Fitzgerald KD et al. 2005. Error-related hyperactivity of the anterior cingulate cortex in obsessive-compulsive disorder. *Biol Psychiatry*. 57:287–294. https://doi.org/10.1016/j.biopsych.2004.10.038.

Frank MJ, Santamaria A, O'Reilly RC, Willcutt E. 2006. Testing computational models of dopamine and noradrenaline dysfunction in attention deficit/hyperactivity disorder. *Neuropsychopharmacology*. 32(7):1583–1599. https://doi.org/10.1038/sj.npp.1301278.

Geburek AJ, Rist F, Gediga G, Stroux D, Pedersen A. 2013. Electrophysiological indices of error monitoring in juvenile and adult attention deficit hyperactivity disorder (ADHD)—a meta-analytic appraisal. *Int J Psychophysiol*. 87:349–362. https://doi.org/10.1016/j.ijpsycho.2012.08.006.

Grant DA, Berg E. 1948. A behavioral analysis of degree of reinforcement and ease of shifting to new responses in a Weigl-type card-sorting problem. *J Exp Psychol*. 38:404–411. https://doi.org/10.1037/H0059831.

Groen Y, Tucha O, Wijers AA, Althaus M. 2013. Processing of continuously provided punishment and reward in children with ADHD and the modulating effects of stimulant medication: an ERP study. *PLoS ONE*. 8:e59240. https://doi.org/10.1371/JOURNAL.PONE.0059240.

Gruner P, Pittenger C. 2017. Cognitive inflexibility in obsessive-compulsive disorder. *Neuroscience*. 345:243–255. https://doi.org/10.1016/J.NEUROSCIENCE.2016.07.030.

Grützmann R et al. 2021. Error-related brain activity in patients with obsessive-compulsive disorder and unaffected first-degree relatives: evidence for protective patterns. *Biol Psychiatry Glob Open Sci*. 2:79. https://doi.org/10.1016/J.BPSGOS.2021.07.001.

Gu BM et al. 2008. Neural correlates of cognitive inflexibility during task-switching in obsessive-compulsive disorder. *Brain*. 131:155–164. https://doi.org/10.1093/BRAIN/AWM277.

Hauser TU et al. 2014. Role of the medial prefrontal cortex in impaired decision making in juvenile attention-deficit/hyperactivity disorder. *JAMA Psychiatry*. 71:1165–1173. https://doi.org/10.1001/JAMAPSYCHIATRY.2014.1093.

Hauser TU et al. 2017. Increased fronto-striatal reward prediction errors moderate decision making in obsessive–compulsive disorder. *Psychol Med*. 47:1246–1258. https://doi.org/10.1017/S0033291716003305.

Izquierdo A, Brigman JL, Radke AK, Rudebeck PH, Holmes A. 2017. The neural basis of reversal learning: an updated perspective. *Neuroscience*. 345:12–26. https://doi.org/10.1016/j.neuroscience.2016.03.021.

Jiang J, Beck J, Heller K, Egner T. 2015. An insula-frontostriatal network mediates flexible cognitive control by adaptively predicting changing control demands. *Nat Commun*. 6:8165. https://doi.org/10.1038/ncomms9165.

Kam JWY et al. 2019. Default network and frontoparietal control network theta connectivity supports internal attention. *Nat Hum Behav*. 3:1263–1270. https://doi.org/10.1038/S41562-019-0717-0;SUBJMETA.

Kanen JW, Ersche KD, Fineberg NA, Robbins TW, Cardinal RN. 2019. Computational modelling reveals contrasting effects on reinforcement learning and cognitive flexibility in stimulant use disorder and obsessive-compulsive disorder: remediating effects of dopaminergic D2/3 receptor agents. *Psychopharmacology*. 236:2337–2358. https://doi.org/10.1007/s00213-019-05325-w.

King JA, Colla M, Brass M, Heuser I, von Cramon DY. 2007. Inefficient cognitive control in adult ADHD: evidence from trial-by-trial Stroop test and cued task switching performance. *Behav Brain Funct*. 3:42. https://doi.org/10.1186/1744-9081-3-42.

Lenth R, Singmann H, Love J, Buerkner P, Herve M. 2018. *Package 'emmeans'. Estimated marginal measn, aka least-squares means*. R package version 1.15-15, Vol. 34.

Liu Q et al. 2023. Cognitive inflexibility is linked to abnormal frontoparietal-related activation and connectivity in obsessive-compulsive disorder. *Hum Brain Mapp*. 44:5460–5470. https://doi.org/10.1002/HBM.26457.

Luman M, Tripp G, Scheres A. 2010. Identifying the neurobiology of altered reinforcement sensitivity in ADHD: a review and research agenda. *Neurosci Biobehav Rev*. 34:744–754. https://doi.org/10.1016/j.neubiorev.2009.11.021.

Martin MM, Anderson CM. 1998. The cognitive flexibility scale: three validity studies. *Commun Rep*. 21:1–9. https://doi.org/10.1080/08934219809367680.

Marzuki AA et al. 2021. Association of environmental uncertainty with altered decision-making and learning mechanisms in youths with obsessive-compulsive disorder. *JAMA Network Open*. 4(11): e2136195. https://doi:10.1001/jamanetworkopen.2021.36195

Miyake A et al. 2000. The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: a latent variable analysis. *Cogn Psychol*. 41:49–100. https://doi.org/10.1006/cogp.1999.0734.

Morein-Zamir S et al. 2014. Hypoactivation in right inferior frontal cortex is specifically associated with motor response inhibition in adult ADHD. *Hum Brain Mapp*. 35:5141–5152. https://doi.org/10.1002/HBM.22539.

Morein-Zamir S et al. 2016. Divergent subcortical activity for distinct executive functions: stopping and shifting in obsessive compulsive disorder. *Psychol Med*. 46:829–840. https://doi.org/10.1017/S0033291715002330.

Nelson HE. 1982. *The National Adult Reading Test (NART): test manual*, Vol. 124. NFER-Nelson, Windsor, UK.

Neufang S, Fink GR, Herpertz-Dahlmann B, Willmes K, Konrad K. 2008. Developmental changes in neural activation and psychophysiological interaction patterns of brain regions associated with interference control and time perception. *NeuroImage*. 43:399–409. https://doi.org/10.1016/J.NEUROIMAGE.2008.07.039.

Nigg JT et al. 2005. Executive functions and ADHD in adults: evidence for selective effects on ADHD symptom domains. *J Abnorm Psychol*. 114:706–717. https://doi.org/10.1037/0021-843X.114.3.706.

Pinheiro J et al. 2021. Linear and nonlinear mixed effects models contact. In: *Linear and nonlinear mixed effects models, Version 3*.

Prado J, Carp J, Weissman DH. 2010. Variations of response time in a selective attention task are linked to variations of functional connectivity in the attentional network. *NeuroImage*. 54:541. https://doi.org/10.1016/J.NEUROIMAGE.2010.08.022.

R Core Team. 2021. R: A language and environment for statistical computing. In: *R foundation for statistical computing*.

Remijnse PL, Nielen MMA, Uylings HBM, Veltman DJ. 2005. Neural correlates of a reversal learning task with an affectively neutral baseline: an event-related fMRI study. *NeuroImage*. 26:609–618. https://doi.org/10.1016/J.NEUROIMAGE.2005.02.009.

Remijnse PL et al. 2006. Reduced orbitofrontal-striatal activity on a reversal learning task in obsessive-compulsive disorder. *Arch Gen Psychiatry*. 63:1225–1236. https://doi.org/10.1001/ARCHPSYC.63.11.1225.

Remijnse PL et al. 2013. Cognitive inflexibility in obsessive-compulsive disorder and major depression is associated with distinct neural correlates. *PLoS ONE*. 8(4): e59600. https://doi.org/10.1371/JOURNAL.PONE.0059600.

Robbins TW, Vaghi MM, Banca P. 2019. Obsessive-compulsive disorder: puzzles and prospects. *Neuron*. 102:27–47. https://doi.org/10.1016/J.NEURON.2019.01.046/ASSET/5FA2221F-95E9-4638-89DC-1266FF678447/MAIN.ASSETS/GR2.JPG.

Rohlf H et al. 2012. Set shifting and working memory in adults with attention-deficit/ hyperactivity disorder. *J Neural Transm*. 119:95–106. https://doi.org/10.1007/S00702-011-0660-3/METRICS.

Rubia K, Smith AB, Brammer MJ, Toone B, Taylor E. 2005. Abnormal brain activation during inhibition and error detection in medication-naive adolescents with ADHD. *Am J Psychiatry*. 162:1067–1075. https://doi.org/10.1176/APPI.AJP.162.6.1067.

Rubia K, Halari R, Mohammad AM, Taylor E, Brammer M. 2011. Methylphenidate normalizes frontocingulate underactivation during error processing in attention-deficit/hyperactivity disorder. *Biol Psychiatry*. 70:255–262. https://doi.org/10.1016/J.BIOPSYCH.2011.04.018.

Smith SM et al. 2004. Advances in functional and structural MR image analysis and implementation as FSL. *NeuroImage*. 23:S208–S219. https://doi.org/10.1016/j.neuroimage.2004.07.051.

Snyder HR, Kaiser RH, Warren SL, Heller W. 2015. Obsessive-compulsive disorder is associated with broad impairments in executive function. *Clin Psychol Sci*. 3:301–330. https://doi.org/10.1177/2167702614534210.

Stern ER et al. 2011. Hyperactive error responses and altered connectivity in ventromedial and frontoinsular cortices in obsessive-compulsive disorder. *Biol Psychiatry*. 69:583–591. https://doi.org/10.1016/j.biopsych.2010.09.048.

Uddin, L.Q. 2021. Cognitive and behavioural flexibility: neural mechanisms and clinical considerations. *Nat Rev Neurosci*. 22:167–179. https://doi.org/10.1038/s41583-021-00428-w

Van Veen V, Cohen JD, Botvinick MM, Stenger VA, Carter CS. 2001. Anterior cingulate cortex, conflict monitoring, and levels of processing. *NeuroImage*. 14:1302–1308. https://doi.org/10.1006/nimg.2001.0923.

Woolrich MW, Ripley BD, Brady M, Smith SM. 2001. Temporal autocorrelation in univariate linear modeling of FMRI data. *NeuroImage*. 14:1370–1386. https://doi.org/10.1006/NIMG.2001.0931.

Woolrich MW, Behrens TEJ, Beckmann CF, Jenkinson M, Smith SM. 2004. Multilevel linear modelling for FMRI group analysis using Bayesian inference. *NeuroImage*. 21:1732–1747. https://doi.org/10.1016/J.NEUROIMAGE.2003.12.023.

Zhao YJ et al. 2023. Evidence accumulation and neural correlates of uncertainty in obsessive-compulsive disorder. *Biol Psychiatry Cognit Neurosci Neuroimaging*. 8:1058–1065. https://doi.org/10.1016/j.bpsc.2023.05.011.

Zühlsdorff K, Dalley JW, Robbins TW, Morein-Zamir S. 2023. Cognitive flexibility: neurobehavioral correlates of changing one's mind. *Cereb Cortex*. 33:5436–5446. https://doi.org/10.1093/CERCOR/BHAC431.

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