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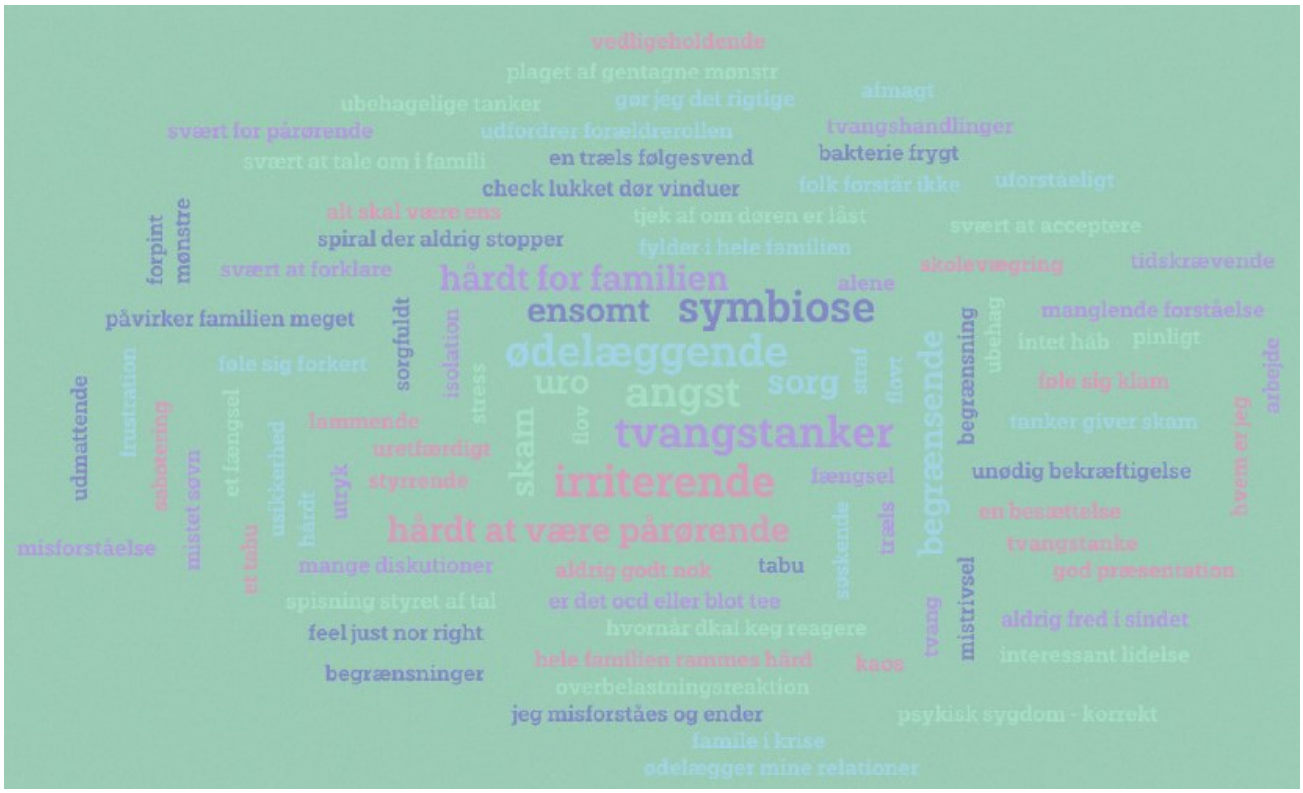

Neurocognitive Correlates of Psychotherapeutic Treatment Response in Children and Adolescents with Obsessive-Compulsive Disorder

PhD Thesis

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Thesis summary

Obsessive-compulsive disorder (OCD) is a common and debilitating mental illness, affecting up to 3 % of children and adolescents. Executive functions (EF) are cognitive processes essential for goal-directed, flexible behavior and are proposed as core mechanisms in OCD symptomatology. However, evidence for EF impairments in pediatric OCD is inconsistent, and neurocognitive correlates of psychotherapeutic treatment response have not been examined across pediatric psychotherapy groups in a randomized design. This thesis aimed to determine, whether deficits in EF and related cognitive processes are present in childhood OCD, whether they improve following psychotherapy, and whether such deficits or changes track symptom improvement.

We included 119 children with OCD who were randomized to 14 sessions of psychotherapy with either cognitive-behavioral therapy (CBT) or an active control condition comprising psychoeducation and relaxation training (PRT). EF was assessed before randomization and after treatment using a comprehensive neurocognitive test battery (Studies 1 and 2) and the BRIEF-2 parent- and self-ratings of daily life EF (Study 3). To our knowledge, this is the first study to assess self-reported EF in children with OCD. Ninety non-psychiatric matched controls were assessed over a comparable time interval.

Neurocognitive testing revealed small, selective difficulties in pediatric OCD that were unrelated to symptom severity. Of these, decision-making (hypothesized finding) and processing speed (explorative finding) improved during psychotherapy. Parent- and child-ratings at baseline indicated moderate-to-large difficulties across EF domains, with a subgroup of patients showing clinically elevated EF impairments. Self-rated difficulties with cognitive flexibility were positively associated with OCD symptoms, and three domains emerged as moderators of treatment response. Most daily life EF domains improved following psychotherapy. However, across neurocognitive and rating-based outcomes, only parent-rated self-monitoring improved differently (i.e., more) for CBT versus PRT, and EF improvements were not associated with symptom reduction.

Taken together, our results indicate widespread daily life EF difficulties in children with OCD, whereas only minor impairments were detected on neurocognitive EF tests. Psychotherapeutic interventions may lead to improvements in EF – particularly in daily life. However, improvement neither appeared related to symptom reduction nor was specific to CBT treatment. Clinicians may pay attention to whether a child with OCD exhibits clinically elevated EF difficulties in daily life before treatment, not least within domains identified as related to treatment response. Future studies are warranted to replicate our self-report results and develop neurocognitive tests that are sensitive to the daily life EF difficulties that children with OCD and their parents experience.

Danish summary (Dansk resumé)

Obsessiv-kompulsiv tilstand (OCD) er en udbredt og indgribende psykisk lidelse, som rammer op til 3 % af børn og unge. Eksekutive funktioner (EF) er de neurokognitive processer, som ligger til grund for mål-orienteret og fleksibel adfærd. EF-vanskeligheder er foreslået som en del af forklaringen på OCD, men studier af EF hos børn og unge med OCD viser blandede resultater, og ingen studier har undersøgt, hvordan EF udvikler sig på tværs af psykoterapiformer i et lodtrækningsforsøg. Formålet med denne afhandling er at undersøge, om børn med OCD udviser EF-vanskeligheder, om de forbedres med psykoterapibehandling, og om vanskeligheder og forbedringer over terapi hænger sammen med sværhedsgraden af OCD-symptomer.

Studiet undersøgte 119 børn med OCD, som blev randomiseret til 14 sessioners psykoterapi bestående af enten kognitiv adfærdsterapi (CBT) eller afspændingsterapi (PRT). EF blev målt før randomisering og efter endt behandling ved hjælp af et omfattende neurokognitivt testbatteri (Studie 1 og 2) samt forældre- og selv-rapport (BRIEF-2; Studie 3). Så vidt vides er dette det første studie, der måler selvrapporteret EF hos børn med OCD. 90 matchede kontrolpersoner uden psykiatrisk diagnose blev undersøgt over et tilsvarende tidsinterval.

De neurokognitive testresultater viste små vanskeligheder inden for få EF-domæner hos børn med OCD, men vanskelighederne var ikke associeret med sværhedsgraden af OCD-symptomer. Beslutningstagning og forarbejdningshastighed forbedredes under psykoterapien. Børn med OCD og deres forældre rapporterede betydelige vanskeligheder på de fleste områder. Selvrapporterede fleksibilitetsvanskeligheder før terapi var korreleret med sværere OCD-symptomer, og tre BRIEF-2-domæner blev identificeret som moderatorer af terapieffekten. De fleste domæner forbedredes under psykoterapi. Af samtlige EF-mål var forældre-rapporteret selvmonitorering det eneste, som bedredes mere for CBT end for PRT, og bedring var ikke associeret med symptomreduktion.

Samlet set indikerer vores resultater udbredte EF-vanskeligheder i dagligdagen, især hos en subgruppe af børn med OCD, mens der kun blev påvist mindre vanskeligheder ud fra de neurokognitive tests. Resultaterne tyder på, at psykoterapeutiske interventioner kan føre til forbedringer i EF – navnlig selv-og forældrerapporteret EF. Forbedringerne synes dog hverken at være relateret til reduktionen i OCD-symptomer eller at skyldes den ene terapitype frem for den anden. Klinikere bør være opmærksomme på, om et barn med OCD eller dets forældre rapporterer klinisk forhøjede EF-vanskeligheder, før familien starter i behandling, især inden for de områder af EF, der fremstår relateret til psykoterapieffekten. Fremtidige studier kan med fordel søge at replicere vores selvrapporterede fund samt udvikle neurokognitive tests, der er sensitive over for de EF-vanskeligheder i dagligdagen, som børn med OCD og deres forældre oplever.

Studies in the thesis

Study 1: The neurocognitive baseline study

Long title: *Atypical neurocognitive functioning in children and adolescents with obsessive–compulsive disorder*. Published in the Journal of European Child & Adolescent Psychiatry (ECAP). DOI: https://doi.org/10.1007/s00787-023-02301-w

Study 2: The neurocognitive follow-up study

Long title: *Neurocognitive correlates of psychotherapeutic treatment response in pediatric obsessive-compulsive disorder: Results from the TECTO trial*. Manuscript ready for submission

Study 3: The BRIEF-2 study

Long title: *Understanding treatment response in pediatric obsessive-compulsive disorder: Executive function ratings from the TECTO trial*. Manuscript under review in the Journal of European Child & Adolescent Psychiatry (ECAP)

Please note that short titles will be used throughout the thesis to improve readability.

Abbreviations

ACC	Anterior Cingulate Cortex
ACT	Acceptance and Commitment Therapy
ADHD	Attention-deficit/hyperactivity disorder
ANOVA	Analysis of Variance
APA	American Psychiatric Association
ASD	Autism spectrum disorder(s)
AST	Attention Switching Task (from the CANTAB test battery)
AST-PCT	Percent correct trials (outcome from the CANTAB AST)
BH	Benjamini-Hochberg
BRIEF	Behavior Rating Inventory of Executive Function
BRIEF-A	Behavior Rating Inventory of Executive Function, Adult Version
BRIEF-2	Behavior Rating Inventory of Executive Function, Second Edition
CANTAB	Cambridge Neuropsychological Test Automated Battery
CBT	Cognitive-Behavioral Therapy
CGAS	The Children's Global Assessment Scale
CGI-I	Clinical Global Impression of Improvement
CGI-S	Clinical Global Impression, Severity
CGT	Cambridge Gambling Task (from the CANTAB test battery)
CGT-QDM	Quality of decision making (outcome from the CANTAB CGT)
CY-BOCS	Children's Yale-Brown Obsessive-Compulsive Scale
CSTC	Cortico-striato-thalamo-cortical (circuits)
DERS	Difficulties in Emotion Regulation Scale
DF	Design Fluency (task from the D-KEFS test battery)
dlPFC	Dorsolateral Prefrontal Cortex
D-KEFS	Delis-Kaplan Executive Function System
DSM	Diagnostic and Statistical Manual of Mental Disorders
EF	Executive function; executive functions; executive functioning
ERP	Exposure and Response Prevention
fMRI	Functional Magnetic Resonance Imaging
FRI	Fluid Reasoning Index (from the Wechsler scales)
GAD	Generalized anxiety disorder

GAI	General Ability Index (from the Wechsler scales)
ges	Generalized eta squared
ICD	International Classification of Diseases
IQ	Intelligence quotient; General cognitive ability
K-SADS	Kiddie Schedule for Affective Disorders and Schizophrenia, Present and Lifetime Version
LFA	Latent Factor Analysis
MANCOVA	Multivariate Analysis of Covariance
MANOVA	Multivariate Analysis of Variance
MD	Mean difference
NICE	National Institute for Health and Care Excellence
NJR	"Not-just-right" (e.g., in the partly sensory "not-just-right" feeling)
OCD	Obsessive-compulsive disorder
OFC	Orbitofrontal Cortex
PANDAS	Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections
PRT	Psychoeducation and Relaxation Training
PSI	Processing Speed Index (from the Wechsler scales)
RT	Reaction time
SD	Standard deviation
SOC	Stockings Of Cambridge (task from the CANTAB test battery)
SWM	Spatial Working Memory (task from the CANTAB test battery)
SRI	Serotonin reuptake inhibitors
SSRI	Selective serotonin reuptake inhibitors
TECTO	Treatment Effects of Family Based Cognitive Therapy in Children and Adolescents with Obsessive-Compulsive Disorder (clinical trial)
TMT	Trail Making Test (from the D-KEFS test battery)
TMT-SRT	Switch condition, reaction time (outcome from the D-KEFS TMT)
TOF	Test Observation Form
VCI	Verbal Comprehension Index (from the Wechsler scales)
VF	Verbal fluency (task from the D-KEFS test battery)
vmPFC	Ventromedial Prefrontal Cortex
VSI	Visuospatial Ability (from the Wechsler scales)

WAIS-IV	Wechsler Adult Intelligence Scale, 4th Edition
WHO	World Health Organization
WISC-V	Wechsler Intelligence Scale for Children, 5th Edition
WMI	Working Memory Index (from the Wechsler scales)

INTRODUCTION

Obsessive-compulsive disorder in pediatric OCD

Characteristics and impact

Obsessive-compulsive disorder (OCD) is a common and potentially debilitating psychiatric disorder affecting up to 3 % of the population worldwide, typically onsetting during childhood or adolescence (Cervin, 2023). It is characterized by the presence of unwanted, distressing, and impairing obsessions and/or compulsions. The disorder is associated with significant functional impairment across school, family, and peer domains (Abramovitch et al., 2024) and substantial socio-economic costs (Kochar et al., 2023). In adults, women are 1.6 times more likely to have OCD than men (Fawcett et al., 2020). The risk of pediatric OCD is higher in girls (0.96 %) than boys (0.63 %) according to a Danish nationwide cohort study (N = 1.3 million) reporting that incidence rates are similar until age 10, after which they increase more steeply in girls, peaking for both sexes in early adolescence (Dalsgaard et al., 2020). Pediatric OCD is frequently accompanied by additional psychiatric conditions, with comorbidity rates reaching 63.6 % – the most common comorbidities include anxiety disorders (31.2 %), depressive disorders (17.1 %), attention-deficit/hyperactivity disorder (ADHD; 16.1 %), and tic disorders (11.9 %) (Sharma et al., 2021). OCD is likely to turn into chronicity; adult patients with a chronic course report earlier OCD onset retrospectively, and longer illness duration increases the risk of OCD remaining chronic (Oudheusden et al., 2018; Visser et al., 2014). On the other hand, early intensive and long-term treatment is associated with better long-term outcomes and remission (Sharma & Math, 2019).

The first-line psychological treatment, Cognitive-Behavioral Therapy (CBT) with Exposure and Response Prevention (ERP), is effective for up to 60 % of children and adolescents, while the remaining individuals are regarded as partial- or non-responders (Uhre et al., 2020). Given the high number of children not sufficiently responding to first-line treatment, there has been several calls for exploring factors that could affect treatment response (Farrell et al., 2023).

Diagnostic criteria

OCD is diagnosed by using the diagnostic manuals: 1) the International Classification of Diseases (ICD) by the World Health Organization (WHO) or 2) the Diagnostic and Statistical Manual of Mental Disorders (DSM) by the American Psychiatric Association (APA) (American Psychiatric

Association, 2013; World Health Organization, 2019). The current newest versions are ICD-11 and DSM-V.

The essential features of OCD are recurrent intrusive thoughts (obsessions) and/or compulsive acts (compulsions). Most commonly, both are present. Obsessions can be thoughts, ideas, beliefs, images, sounds, or impulses/urges that are intrusive and unwanted. They are often (but not always) associated with autonomic anxiety symptoms, such as increasing heart rate, sweating, trembling and other physical reactions. Compulsions (or rituals) are repetitive behaviors or mental acts, often (but not always) carried out in response to obsessions.

To fulfill the diagnostic criteria of OCD, obsessions and/or compulsions must be present most days for at least two weeks and cause significant distress or functional impairment. The obsessions and compulsions must be recognized as the patient's own, acknowledged as excessive or unreasonable, the patient must try (at least minimally) to resist them, and the symptoms cannot be in themselves pleasurable.

While in ICD-10 and DSM-IV, OCD is placed in the chapter of anxiety disorders, in ICD-11 and DSM-V, OCD is categorized under "Obsessive-compulsive or related disorders" (OCRDs), stressing a shift in the view on OCD as a disorder of compulsivity (rather than anxiety). Also, in ICD-11 and DSM-V, the level of insight into disorder-specific beliefs can be specified, thereby expanding the diagnosis criteria to include patients with minimal insight.

Clinical heterogeneity

While being described by a single diagnostic category, the clinical manifestations of OCD exhibit substantial clinical heterogeneity. Obsessions and compulsions can manifest in countless ways and focus on a wide range of themes, and OCD symptoms are typically classified into distinct, overlapping subtypes. A recent factor and network analysis in children and adults with OCD (N = 1366) identified eight broad symptom dimensions (disturbing thoughts, incompleteness, contamination, hoarding, transformation, body focus, superstition, and loss/separation), and of these, incompleteness was most central in children, possibly representing a core phenotype (Cervin et al., 2022). Incompleteness comprises accuracy (e.g., symmetry and ordering) as well as feeling of something being "not-just-right" (NJR) and behaviors carried out until a "just-right" feeling is obtained (e.g., repeating, counting, and evening out). The NJR symptom dimension and the shift in thinking about OCD as compulsivity (rather than anxiety) has given rise to theories understanding OCD as a sensorimotor disease (van den Heuvel et al., 2016; Jalal et al., 2023).

Etiology and treatment in a vulnerability-stress framework

The etiology of OCD continues to be debated but is generally viewed within a vulnerability-stress framework, in which symptoms emerge from the interaction of multiple predisposing vulnerabilities, environmental stressors, and maintaining mechanisms. Understanding the development and maintenance of symptoms and reversing the maintaining mechanisms of the disease is considered essential in the psychological treatment of OCD (Shafran, 2005).

Part of the vulnerability predisposing for OCD is based on genetic factors; twin studies consistently report substantial heritability (the proportion of variation in a population trait that can be attributed to inherited genetic factors) of OCD (Mahjani et al., 2021; Mataix-Cols et al., 2024), with increased estimates in childhood-onset OCD ranging from approximately 45 to 61 % (Hudziak et al., 2004) compared to that of adult-onset OCD. Vulnerability also reflects other biological as well as psychological and environmental factors. Notably, OCD (and tics) has been proposed to arise due to an autoimmune reaction to streptococcal infection (Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections; PANDAS) (Snider & Swedo, 2004). Thus, by acute onset of OCD when conjunct with features like separation anxiety, irritability, or emotional lability, post-infectious etiology should be considered (Łojek & Rzeszutek, 2025).

The current thesis focuses on neurocognitive and neurobehavioral deficits expressing underlying neurobiological mechanisms as potential vulnerabilities and targets for treatment in pediatric OCD. As psychotherapy aims to reverse the maintaining mechanism of OCD, neurocognitive vulnerability may influence how well the treatment works and may itself improve with treatment, possibly closely related with symptom improvement.

While several other predisposing mechanisms and environmental stressors are considered essential for the development and maintenance of OCD, these are beyond the scope of this thesis and not discussed any further.

Neurobiological disease models

A large body of neuroimaging and neurocognitive research links OCD to dysfunction in the cortico-striato-thalamo-cortical (CSTC) circuits of the brain, which support executive function and habit learning (for a review, see Jalal et al., 2023). Imbalances in these circuits may underlie OCD symptomatology by increasing susceptibility to maladaptive learning processes, such as the acquisition and reinforcement of compulsive behaviors. This and the following section contain an introduction to CSTC and cognitive-behavioral models for OCD.

CSTC circuits were first described by Alexander et al. (1986) as a series of thousands of parallelly organized, functionally segregated neuroanatomic circuits linking the basal ganglia and thalamus to cortex. While originally thought to be segregated (closed), more recent work revealed functional interactions between the circuits, indicating only partial segregation (Milad & Rauch, 2012). The circuits consist of interconnected brain regions, including the cortex, striatum, and thalamus, that communicate through networks of neurons; neurons in one region send electrical signals that release neurotransmitters, which in turn excite or inhibit neurons in other regions. Signals loop between cortical and subcortical regions through these partially segregated direct and indirect pathways with opposing net effects on the cortex. These processes allow for continuous information exchange that subserve different behavioral functions, and they are thought to mediate symptom expression in various psychiatric diseases.

Various disease models have linked specific CSTC pathways to OCD, based on neuroimaging findings of aberrant activity within the circuits, sometimes linked to cognitive dysfunction. Based on structural and functional abnormalities implicating the Orbitofrontal Cortex (OFC) in OCD, an early model suggests hyperactivity in orbitofrontal-subcortical circuits (Saxena & Rauch, 2000; Saxena et al., 2001). A related, more detailed model was proposed by Milad & Rauch (2012), who suggested three CSTC circuits to be implicated in OCD: 1) An affective (or motivational) circuit projecting from the Anterior Cingulate Cortex (ACC) and the Ventromedial Prefrontal Cortex (vmPFC), 2) a cognitive circuit (central to executive function) projecting from the Dorsolateral Prefrontal Cortex (dlPFC), 3) and another cognitive circuit (central to motor- and response inhibition), projecting from the anterolateral OFC. Additionally, a sensorimotor circuit (central to habit-based behavior) that projects from (pre)motor cortical regions has been suggested to be implicated in OCD (van den Heuvel et al., 2016).

Based on the CSTC models, the "habit hypothesis" of OCD suggests that the disease may derive from excessive, maladaptive habit formation and failure in goal-directed behavior leading to inflexible thinking and behavior (Graybiel & Rauch, 2000; Gillan & Robbins, 2014).

Cognitive-behavioral disease models

Cognitive-behavioral models of OCD provide a psychological framework for the interaction of cognitive and behavioral aspects of the disease (Salkovskis et al., 1998; Shafran, 2005; Taylor et al., 2006).

Historically, cognitive-behavioral models of OCD derive from learning-based conditioning models. These were based on the two-factor model of fear, developed by the American

psychologist Orval Mowrer, stating that obsessional fear was acquired by classical conditioning and maintained by operant conditioning (negative reinforcement) (Taylor et al., 2006).

Contemporary cognitive-behavioral models anticipate that faulty beliefs (cognitive errors/biases) induce obsessional anxiety (Jalal et al., 2023). Faulty beliefs could be for example overestimation of threat, perfectionism, and intolerance of uncertainty. In order to cope with the obsessional anxiety, compulsive behavior is performed. For example, seeking to avoid the threatening stimulus or neutralizing the threat so that the distress induced by obsessions is avoided or relieved.

The models are based on a body of empirical psychological research, primarily questionnaire studies and laboratory experiments. For example, the models of Salkovskis (Salkovskis et al., 1998) are partly based on evidence from a questionnaire study showing that people with OCD do not distinguish between actively causing harm (commission) and causing harm by failing to act (omission), leading to the conclusion that OCD patients feel overly responsible. Additionally, laboratory experiments found that participants in a high responsibility condition showed greater anxiety and checking behavior than those with low responsibility, mirroring the inflated sense of responsibility seen in OCD (Salkovskis et al., 2000).

While traditional cognitive-behavioral models of OCD assume that obsessions lead to compulsions, research has shown that compulsive behavior can be triggered without anxiety (Gillan et al., 2014; Gillan & Robbins, 2014). Thus, the causal relationship between obsessions and compulsions is not always clear (Fineberg et al., 2018).

Cognitive-behavioral models for OCD provide a theoretical framework for psychological treatment anticipating that OCD symptoms can be ameliorated by changing interpretations of intrusive thoughts and ceasing to act compulsively (Jalal et al., 2023; Shafran, 2005).

Pediatric versus adult OCD

Although adult diagnostic criteria have traditionally been applied to childhood cases of OCD, accumulating evidence suggests important developmental differences, giving rise to the hypothesis that pediatric OCD represents a distinct developmental subtype of the disorder (i.e., the discontinuity hypothesis of OCD) (Geller et al., 2021; Kalra & Swedo, 2009; Thapar et al., 2025). Younger children are more likely to present with compulsions and few or no clearly articulated obsessions. The compulsive behaviors are often driven by rigid rules, not-just-right sensations, and reduced insight (Shaw & Rapoport, 2025; Swedo et al., 1989). A related diagnostic challenge is that many children are not willing or cognitively able to describe their obsessions, which can make these symptoms appear absent or less prominent. While there is substantial overlap in

symptom profiles across ages, certain symptoms show age-specific patterns; superstition (i.e., "magical thinking") are more commonly reported in children, whereas hoarding is less central (Cervin et al., 2022).

Developmental differences also emerge in epidemiological studies. Incidence rates are similar among boys and girls until the age of 10, after which OCD becomes more common in girls during adolescence and adulthood (Dalsgaard et al., 2020). Patterns of comorbidity likewise diverge: Adult OCD is most often associated with mood and anxiety disorders, whereas early-onset OCD more commonly co-occurs with comorbid developmental disorders such as tic disorders, ADHD, and autism spectrum disorders (ASD) – conditions with overall male predominance (Geller et al., 2021).

Familial risk further contributes to the developmental discontinuity hypothesis. Early-onset OCD appears more strongly familial, with first-degree relatives of affected children showing roughly twice the risk observed in relatives of adults (Geller et al., 2021).

Neurocognitive profiles also differ by age. Children generally exhibit more selective and less severe deficits than adults (Abramovitch et al., 2015; Marzuki et al., 2020), a topic reviewed in detail in a later section.

Taken together – across symptom presentation, comorbidity patterns, sex ratios, heredity, and neurocognitive correlates – these cross-sectional differences support the view that pediatric OCD may constitute a developmental subtype of adult OCD, or potentially even a distinct psychiatric disorder (Geller et al., 2021; Kalra & Swedo, 2009; Thapar et al., 2025).

Psychological treatment

The most effective psychological treatment for pediatric OCD is Cognitive-Behavioral Therapy (CBT) with Exposure and Response Prevention (ERP) (Uhre et al., 2020). A recent network meta-analysis of available RCTs reporting mean differences (MDs) in OCD symptom improvement, concludes that individual, in-person CBT is significantly more efficacious than waitlist (MD = 11.10), pill placebo (MD = 7.66), and relaxation training (MD = 5.49) as well as internet-delivered CBT (MD = 3.95) (Cervin et al., 2024).

Based on the robust empirical support, CBT is recommended as the first-line psychological treatment for children and adolescents with moderate to severe OCD in international as well as Danish clinical guidelines (Geller & March, 2012; National Institute for Health and Care Excellence (NICE), 2005; Sundhedsstyrelsen, 2019). CBT is recommended either alone or combined with serotonin reuptake inhibitors (SRIs) in more severe cases. Guidelines stress the

importance of involving family or carers (e.g., family-based psychotherapy) in the treatment of children and young people.

Although CBT is the most effective treatment, it should be noted that there is also a proportion of children not responding sufficiently. A meta-analysis found an average response rate of 68 % (defined as "much-" or "very improved" on the Clinical Global Impression of Improvement (CGI-I) or more than 25 % reduction on the CY-BOCS total score) and an average remission rate of 57 % (defined as a CY-BOCS total score of 14 or less) (McGuire et al., 2015).

The therapeutic elements of standard manualized CBT for OCD are psychoeducation, cognitive restructuring, and ERP, and the treatment typically consists of 12-14 weekly sessions (March & Mulle, 1998; Barrett et al., 2008; Piacentini et al., 2011; Thomsen & Weidle, 2015).

ERP addresses the functional relationship between obsessions and compulsions that are described in the cognitive-behavioral model of OCD. In ERP, the child and therapist construct a hierarchy of increasingly distressing situations/thoughts/objects (typically inducing obsessions) that the child is gradually exposed to while practicing not to engage in compulsions (March & Mulle, 1998). While progressing through the hierarchy, anxiety and/or urges to act compulsive are reduced in most patients. The mechanism through which distress is reduced is debated. While traditional exposure therapy has stressed habituation and belief disconfirmation (reduction in fear and disconfirmation of threat-laden beliefs), more recent approaches apply inhibitory learning principles (developing new, non-threat associations that inhibit, rather than erase, fear response) (Craske et al., 2014).

The cognitive restructuring components of CBT (such as normalizing obsessive thoughts and challenging or reappraising maladaptive beliefs) have not been systematically evaluated in controlled trials. Family factors targeted in CBT for children with OCD include reducing family accommodation (i.e., the family seeking to relieve the child's distress by facilitating avoidance and rituals) and engaging the parents in the ERP training (Thomsen & Weidle, 2015).

Due to insufficient evidence, alternative interventions to CBT are only weakly recommended and only in conjunction with ERP. For example, the Danish national clinical guideline allows for the use of third-wave CBT (typically including acceptance and commitment, metacognitive, dialectical behavior, and mindfulness-based therapy) when preferred by the patient, provided that ERP remains part of the treatment (Sundhedsstyrelsen, 2019). A review of twelve RCTs (two included youth) concludes that the beneficial effects of third-wave CBT and conjunctional ERP is sometimes comparable to traditional CBT (Trent et al., 2021). However, while some treatments (namely Acceptance and Commitment Therapy; ACT) show promising effects (also in children), these effects are not large and consistent enough to justify replacing traditional CBT with ERP.

Executive functions

There has been a recent call for clarifying whether EF deficits are present in childhood OCD and whether they improve following psychological treatment (Jalal et al., 2023).

Definition and historical perspectives

The executive functions (EF) of the human brain (largely synonymous with cognitive control; Robbins et al. (2024)) comprise higher-order cognitive processes that monitor and control the basic cognitive functions (Lezak, 1982; Miyake et al., 2000; Diamond, 2013; Friedman & Robbins, 2022). EF enables goal-directed thinking and behavior that is appropriate and adaptive to an ever-changing environment and counteracts automaticity. The functions are crucial in new, complex, or ambiguous situations where automated reactions or habits are not sufficient and when behavior needs to be actively regulated.

Historically, the concept of EF has its roots in early neurology and clinical neuropsychology (dating back to the 19th century) where observations of patients with frontal lobe damage led to the inference that frontal cortical areas were involved in the regulation of behavior. During the last half of the 20th century, researchers like the famous neuropsychologists Alexander Luria and Karl Pribram shaped the field studying clinical cases, behavioral tasks, and brain lesions, and the term "executive functions" was popularized with Muriel Lezak's 1982 review paper (Lezak, 1982). These early lesion-based accounts set the foundation for later mechanistic models of EF, and with the advent of neuroimaging techniques in the 1990s and the following decades, many of these clinical observations were refined and validated, for example using paradigms as task-based Functional Magnetic Resonance Imaging (fMRI). Thus, a body of research showed that rather than being exclusively linked to the PFC, EF are subserved by interconnected cortical and subcortical regions (Leh et al., 2010), collectively referred to as the cortico-striato-thalamo-cortical (CSTC) circuits.

Subdomains

Although EF is sometimes treated as a unitary construct, most contemporary models emphasize that EF represents separable but interacting processes rather than a single capacity. Specifically, the division of EF into separate categories (subdomains) is notoriously debated (Friedman & Robbins, 2022). Guided by Latent Factor Analysis (LFA) of EF task performance, three core EF have been identified: 1) cognitive flexibility (shifting between tasks or mental sets), 2) working

memory (monitoring and updating representations), and 3) inhibition (stopping prepotent responses) as well as an overall "common" EF factor (Miyake et al., 2000; Engelhardt et al., 2015; Friedman et al., 2008; Friedman & Robbins, 2022). Additional EF, thought to build on these core functions, are reasoning, fluency, problem solving/decision making, and planning/organizing (although this list is not exhaustive). LFA has also been applied to questionnaire-based assessments of EF, such as the Behavior Rating Inventory of Executive Function (BRIEF), which was developed from expert-derived items describing everyday behavior associated with EF dysfunction. LFA results from questionnaire-based assessments have largely parallelled the structure found in task-based studies (Gioia et al., 2000, 2015). Finally, a body of research examines the mediating role of the PFC in different types of EF behavior and identifies six underlying CSTC circuits (for reviews, see Friedman & Robbins (2022); Menon & D'Esposito (2022)).

Working memory

Working memory is defined as holding information in mind (when no longer perceptually present) and mentally manipulating this information (Diamond, 2020). It can be divided into verbal and visuo-spatial working memory. Examples could be remembering instructions or rules while carrying them out or remembering a strategy while implementing it. Working memory is strongly associated with general cognitive ability (intelligence quotient; IQ), with substantial shared variance, although the constructs remain conceptually distinct (Diamond, 2020; Wechsler, 2014).

Inhibitory control

Inhibitory control is broadly defined as the ability to inhibit thought and behavior that is irrelevant and/or inappropriate according to one's goals or the current context (Bari & Robbins, 2013; Logan & Cowan, 1984). Inhibition comprises both self-control (resisting internal urges) and interference control, and it is recruited for example when changing a habit, resisting impulsions or compulsions, thinking before acting, and controlling behavior in distressing situations.

Cognitive flexibility

Cognitive flexibility (also referred to as set shifting) is defined as the ability to appropriately switch between multiple alternatives. Specifically, this may comprise shifting between different perspectives, tasks, or mindsets as well as adjusting/accommodating quickly to change (Diamond, 2020). Examples could be finding an alternative strategy, if the one you intended to use is not available, changing the way you think about a subject, or changing a habit. Cognitive flexibility

recruits both inhibition (suppressing previous perspective) and working memory (imagining and making use of a new perspective). Abnormal cognitive flexibility is suggested to be involved in cognitive rigidity, characterized by poor ability to change perspective and adapt to change.

Cool and hot executive functions

A distinction has been proposed between "cool" and "hot" EF (Zelazo & Müller, 2002; Zelazo & Carlson, 2012). Cool EF refer to affectively neutral (i.e., non-emotional) processes, whereas hot EF are engaged in emotionally significant contexts that involve motivation and reward processing. Examples of hot EF include inhibiting automatic response to emotionally salient content or making decisions involving potential gains or losses. Although it is debated whether these constructs are truly distinct (Moriguchi & Phillips, 2023) they are widely used to differentiate tasks and processing based on their level of emotional involvement.

Measuring executive functions

EF refer to the behavioral manifestations of underlying neurobiological control processes. Accordingly, the operationalization of EF involves categorizing and quantifying such behavior.

Neurocognitive tests

Neurocognitive tests of EF are structured experimental activities designed to measure specific EF domains. In these tasks, participants perform activities intended to recruit the EF domain of interest, and outcomes typically include time spent (reaction time; RT), accuracy (number of errors), and/or error types. Computer-based assessment has been argued to allow for more precise and automated data collection compared to traditional pen-and-paper methods (Chamberlain et al., 2005).

Extensive test paradigms have been developed to examine EF subdomains (Friedman & Robbins, 2022). Inhibitory control (including interference control) is often assessed with the Anti-saccade, Stop-signal, Stroop, or Flanker paradigms. Working memory is commonly studied using N-back, Letter memory, Digit span or Self-ordered selection tasks. Cognitive flexibility is measured with cued task-switching paradigms or more complex tasks such as the Wisconsin card sorting test and the Intra-extra-dimensional/reversal learning task. Planning is typically investigated using the Tower of London or Stockings of Cambridge tasks, while decision making is examined through paradigms such as Delay discounting or gambling tasks (e.g., the Iowa gambling task or Cambridge gambling task).

Neurocognitive tests are frequently used for assessing EF difficulties in clinical populations with neurological and psychiatric conditions. The norm for identification of clinically significant impairment of neurocognitive performance in an individual is generally considered a difference of 2 standard deviations (SDs) from healthy controls (Lezak, Muriel Deutsch, 2012; Abramovitch et al., 2015).

Questionnaires

There has been a call for applying ecologically valid functional measures of EF in children as part of the evaluation (Abramovitch et al., 2015). Indeed, EF-related functioning in daily life can be assessed using questionnaire-based instruments like the Behavior Rating Inventory of Executive Function (BRIEF) (Gioia et al., 2000, 2015). In these questionnaires, the informant rates how frequently a person engages in behaviors related to EF problems. The BRIEF, designed for assessing EF in children and adolescents, is available in parent-, teacher-, and self-report versions, while an adult self-report version (the BRIEF-A) is also available (Roth et al., 2015).

Importantly, questionnaire-based instruments capture EF as expressed in everyday functioning, which may tap into difficulties that are not always detectable in structured laboratory tasks. This distinction becomes central when interpreting discrepancies between task-based and questionnaire-based EF findings in pediatric OCD.

The task impurity problem

A further complication when measuring EF is the "task impurity problem", meaning that any given EF task typically recruits multiple cognitive processes (Friedman & Miyake, 2017). This poses challenges when attempting to isolate specific subdomains and contributes to variability across studies targeting the same subdomain.

Instruments used for assessing EF in the current thesis are described in more detail in the methods' section.

Development and maturation

EF are subserved by the CSTC circuits and thus EF development is closely related to the maturation of the brain anatomy involved in these circuits. The PFC is the last brain region to reach full maturation and is very plastic (susceptible to change) (Leh et al., 2010; Kolk & Rakic,

2022). During childhood and adolescence, processes such as myelination, synaptic pruning, and strengthening of neural connectivity continue to refine PFC and CSTC functioning.

Accordingly, EF mature relatively late and are vulnerable to environmental stressors but also improvable throughout life. Marked improvements in inhibitory control, working memory, cognitive flexibility, and planning are characterizing the early school years (age 6-11 years) (Diamond, 2020). For example, until 6-7 years of age, children can barely do an antisaccade test of inhibitory control (ability to follow instructions to look in the opposite direction of a peripherally presented visual stimulus), but after this age, performance improves markedly for a couple of years until stabilized at a mature level. A similar pattern is seen for task-switching tests of cognitive flexibility. While performance on EF tasks improves rapidly during early school years, peak performance is typically not seen until the early 20s. Delays or disruptions in the maturation of CSTC circuits and accordingly EF may contribute to vulnerability to mental illness (Diamond, 2020).

Taken together, when assessing EF performance in children and adolescents with and without suspected challenge to maturational processes, it is crucial to adjust for age-related variability.

Executive functions in the context of pediatric OCD

OCD as a disease of executive dysfunction

Several factors have contributed to the view that OCD represents a disease characterized by executive dysfunction. This view does not imply that EF deficits are universally diagnostic for OCD, but rather that EF processes may modulate how obsessions and compulsions are generated, maintained, and modified through treatment.

EF and OCD symptomatology

A key clinical feature of OCD is its ego-dystonic nature – patients typically (unless insight is very low) recognize their obsessions and compulsions as excessive, unreasonable, and inconsistent with their goals and values. This interpretation challenges the cognitive-behavioral models of OCD (Jalal et al., 2023), which assume that compulsions are purposeful responses aimed at reducing obsession-induced stress mediated by faulty value attribution and cognitive bias. The neurocognitive habit hypothesis of OCD, based on the CSTC models, suggests that OCD symptoms are characterized by a failure of EF and overreliance on habitual responses (Fineberg et al., 2018; Gillan & Robbins, 2014). Thus, the ritualized behavior in OCD is viewed as excessive habit formation at the cost of adaptive, goal-directed, flexible thinking and behavior. The CSTC-based habit hypothesis of OCD has been supported by resting-state fMRI studies (Hao et al., 2019) and task-based fMRI (Vaghi et al., 2017; van den Heuvel et al., 2016). Hypoconnectivity between the dlPFC and the Putamen have been associated with deficits in goal-directed planning (Vaghi et al., 2017).

Notably, goal-directed instrumental behavior (i.e., negative reinforcement of obsessions and compulsions in the cognitive models of OCD) often develops habitual characteristics following long time of repetition (Kalanthroff & Wheaton, 2022; Robbins et al., 2024). Thus, it is possible that obsessions and compulsions begin as purposeful but eventually feel involuntary and ego dystonic. This integrates or at least provides an overlap between the cognitive models and the CSTC-based habit hypothesis of OCD.

EF and CBT

EF is involved in generating and monitoring as well as formulating and conducting novel plans (Leh et al., 2010; Diamond, 2020). These are actions that are often required and addressed when treating pediatric OCD with CBT. Indeed, in cognitive restructuring, the child is helped to formulate novel ways of understanding their thoughts and continuously monitor and change their

thought patterns. In ERP, the child is helped to plan gradual exposure and actively change behavior, thus implementing new strategies. Consequently, pre-treatment EF capacity is suggested to influence a child's ability to engage in CBT and benefit from ERP or cognitive restructuring, and CBT itself is suggested to train and strengthen EF processes (Chamberlain et al., 2005; Fitzgerald et al., 2021; Norman L.J. et al., 2021; Chamberlain et al., 2021; Goodkind et al., 2016). Based on these notions, EF may act as a moderator and/or mediator of symptom improvement in CBT.

Understanding whether EF deficits influence treatment, improve with treatment – and whether such changes relate to symptom reduction – remain important unanswered questions.

Empirical support – state of the art

Cross-sectional studies in adult and pediatric OCD

In adults with OCD, meta-analyses have stated mild to moderate impairment of executive functions (Abramovitch et al., 2013; Shin et al., 2014; Snyder et al., 2015; Chamberlain et al., 2021) also in their unaffected relatives (Bora, 2020). Medium to large effect sizes were found for the EF subdomains of planning, inhibition, and cognitive flexibility, and a small effect size was seen for working memory. Additionally, impairments were seen in sustained attention, verbal/non-verbal memory, visuospatial abilities, and processing speed. Nevertheless, deficits in visuo-spatial abilities have been proposed as the only neurocognitive diagnostic marker of OCD (Fullana et al., 2020). Neurocognitive impairment is mild to moderately associated with more severe OCD symptoms in adults (Abramovitch et al., 2019; Martínez-Esparza et al., 2021). EF difficulties have also been self-reported in adults with OCD, using the BRIEF-A (Samuels et al., 2018; Fournet et al., 2019).

In pediatric OCD, findings are less consistent, and effects are smaller. The only meta-analysis of neurocognitive test performance in pediatric OCD (Abramovitch et al., 2015) included 11 studies and reported small, non-significant effects of underperformance in OCD versus controls across all EF subdomains. Effects were largest for planning, visuospatial abilities, and cognitive flexibility. The authors emphasize that the small number of included studies and the small samples across studies (N = 14-35) may have influenced the lack of statistically significant differences between OCD groups and healthy control groups. A later systematic review (without meta-analysis) included a larger number of pediatric studies (N = 43) aimed to examine candidate neurocognitive

endophenotypes found in adult-OCD (Marzuki et al., 2020). Results indicated difficulties in decision-making under uncertainty, planning, and visual working memory, while only little evidence was found for difficulties in cognitive flexibility and inhibitory control in pediatric OCD. More recent studies continue to produce mixed results. Poorer performance in spatial working memory has been identified (Negreiros et al., 2020) as well as problems with decision-making under uncertainty (Marzuki et al., 2021). Deficits in cognitive flexibility and inhibitory control was seen in children with OCD as well as their unaffected siblings and parents (Abramovitch et al., 2021), and another study found inhibitory control difficulties in children with OCD as well as in a community sample with high OCD traits but no diagnosis (Schachar et al., 2022). On the other hand, null findings have also been frequently reported. Taken together, the mixed results highlight that EF impairment in pediatric OCD is neither robust nor uniform, and likely depends on age, symptom profile, task demands, and methodological difference across studies.

Conversely, questionnaire studies in pediatric OCD have produced more consistent findings regarding everyday behavior associated with EF dysfunction. Parent- and/or teacher-rated EF difficulties have been reported on the BRIEF and BRIEF-2 (Zandt et al., 2009; Hybel et al., 2017; Negreiros et al., 2020; Rydqvist et al., 2023; Killion et al., 2024). Most studies report index scores (behavioral regulation- and metacognitive index) and/or GEC (total score), while only two provide effects for each EF subdomain (Negreiros et al., 2020; Rydqvist et al., 2023). The two latter indicate problems in all domains and particularly large effects for the BRIEF subdomains of cognitive flexibility and initiating.

Treatment studies in adult and pediatric OCD

Improvement of neurocognitive test results after CBT has been reported in adults for cognitive flexibility, planning, inhibition, visuospatial ability, memory, and processing speed (Moritz et al., 1999; Kuelz et al., 2006; Voderholzer et al., 2013; Katz et al., 2024; Wheaton et al., 2025), but null findings have been seen even more often (Kim et al., 2002; Bannon et al., 2006; Rao et al., 2008; Vandborg et al., 2015; Braga et al., 2016; Reid et al., 2025; Roh et al., 2005). Notably, one neuroimaging study found that normalization of resting-state connectivity during CBT correlated with symptom improvement (Li et al., 2018). Additionally, better cognitive flexibility has been identified as a predictor of better CBT response (D'Alcante et al., 2012), and better processing speed has been identified as a predictor of response versus non-response to CBT (Kuelz et al., 2006).

Five pediatric studies have examined neurocognitive test results after CBT, compared to healthy control groups assessed within a similar time interval (Andrés et al., 2008; Huyser et al., 2010, 2011; Hybel et al., 2017; van der Straten et al., 2018). One reported improvement of non-verbal memory in children with OCD (N = 29) after six months of naturalistic treatment with CBT and/or medication (90 % Sertraline, 10 % Clopramine, 65.5 % CBT), while healthy controls (N = 22) stayed stable (Andrés et al., 2008). Another study found that subtle pre-treatment planning impairments normalized in non-medicated children with OCD (N = 25) after 16 sessions of protocol-based CBT, while controls improved less (N = 25) (Huyser et al., 2010). Finally, a third study also reported improvement of planning in non-medicated pediatric OCD-patients (N = 15) after 16 sessions of CBT and two years of naturalistic follow-up, while controls (N = 16) showed less improvement (van der Straten et al., 2018). Potential associations between changes in OCD symptom severity and changes in neurocognitive performance were not reported (although notably, in Huyser et al. (2010), improvement of symptom severity was found to correlate with increased dlPFC and parietal activation after treatment). Neurocognitive improvements after CBT were not observed in the two remaining pediatric studies (Huyser et al., 2011; Hybel et al., 2017). Finally, findings from one pediatric sample suggest that impaired visuospatial memory and planning predicts worse CBT prognosis (Flessner et al., 2010).

Parent-rated EF was examined with the BRIEF in relation to treatment in three pediatric studies (Hybel et al., 2017; McNamara et al., 2014; Rydqvist et al., 2023). One study reported EF before and after treatment and concluded that patients ($N = 50$) improved relative to controls on the GEC during CBT. All three studies examined whether pre-treatment EF predicted symptom improvement. One study found that greater difficulties on the emotional control subscale were associated with less symptom improvement (McNamara et al., 2014), whereas the other two reported no predictive value of pre-treatment GEC or EF subscales for treatment outcome (Hybel et al., 2017; Rydqvist et al., 2023).

Altogether, potential challenges with the existing literature should be noted. Although EF has been proposed as a central cognitive mechanism in OCD, evidence in pediatric populations remains limited, inconsistent, and methodologically heterogenous. The lack of parallel treatment groups, the absence of self-rated EF behavior data, and the scarcity of subdomain-level longitudinal analyses leave important gaps in the literature. Addressing these gaps is essential for clarifying whether EF deficits are present in youth with OCD, whether they change with CBT, and whether such deficits and changes track CBT-induced symptom improvement – the central aims of this dissertation.

OBJECTIVES AND HYPOTHESES

The aim of this dissertation was to investigate neurocognitive correlates of pediatric OCD and their relation to psychotherapeutic treatment response. Across three related studies using largely overlapping samples from the TECTO trial, we examined neurocognitive test performance and everyday EF associated behavior before and after treatment, as well as their associations with OCD symptom severity.

Study 1: The neurocognitive baseline study

The aim of Study 1 was to investigate baseline neurocognitive test performance in children and adolescents with OCD compared with non-psychiatric controls. In addition, we explored whether performance was associated with OCD symptom severity.

We hypothesized that:

a) OCD patients would show poorer performance than control participants on tests of EF
b) Poorer performance would be associated with higher OCD symptom severity

Study 2: The neurocognitive follow-up study

The aim of Study 2 was to investigate changes in neurocognitive performance during psychotherapeutic treatment for OCD and the relation of these changes to symptom improvement.

We hypothesized that:

a) If EF test performance was atypical in patients at baseline (i.e., poorer than controls), it would improve over the course of treatment relative to controls, with greater improvements following CBT than PRT
b) Test improvements after treatment would correlate with OCD symptom reduction
c) Baseline EF performance would predict treatment response (i.e., better performance would be associated with greater symptom reduction for both treatments)
d) Baseline EF performance would moderate treatment response based on treatment type (i.e., better performance would be associated with greater symptom reduction for CBT versus PRT)

Study 3: The BRIEF-2 study

The aim of Study 3 was to evaluate parent- and self-rated everyday executive function in children with OCD, before and after treatment, and its association with symptom change.

We hypothesized that

a) OCD patients would show more EF difficulties than control participants at baseline
b) More difficulties would be associated with higher OCD symptom severity
c) Difficulties would improve over the course of treatment relative to controls, with greater improvements following CBT than PRT
d) EF improvement after treatment would correlate with OCD symptom reduction
e) Baseline EF would predict treatment response (i.e., better EF would be associated with greater symptom reduction for both treatments)
f) Baseline EF would moderate treatment response based on treatment type (i.e., better EF would be associated with greater symptom reduction for CBT versus PRT)

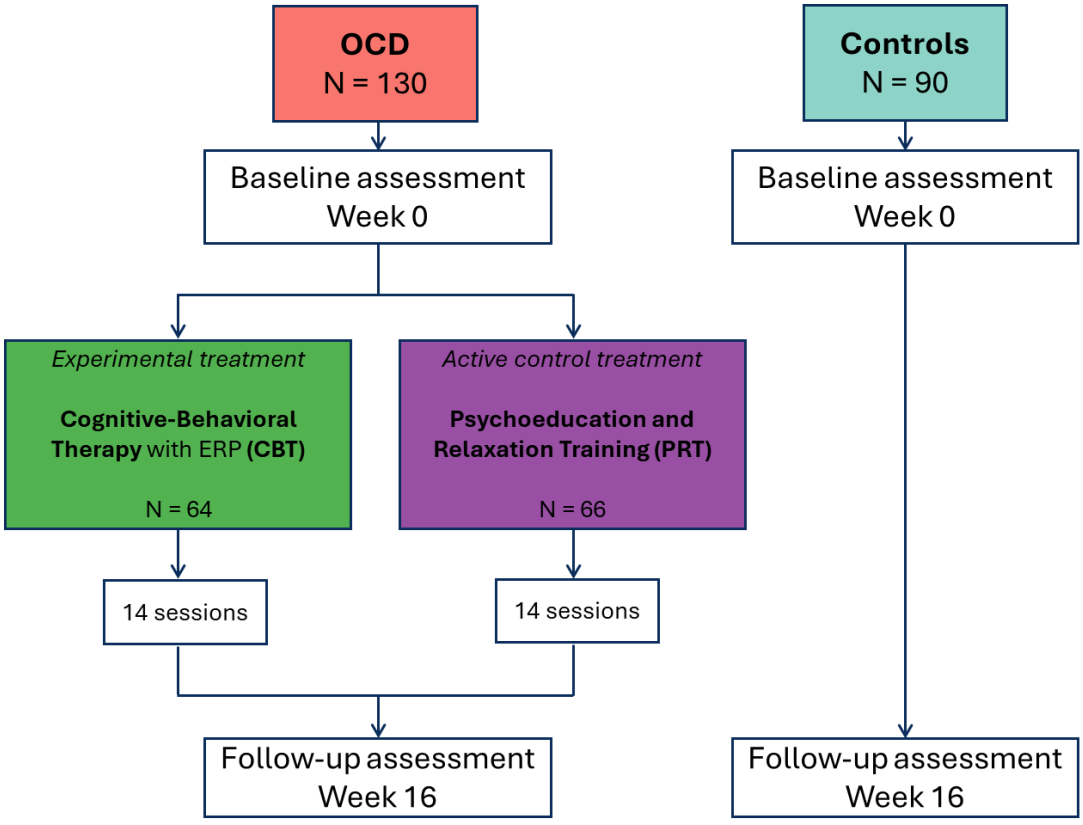
METHODS

The neurocognitive studies included in this thesis were planned as case-control sub-studies of the Danish RCT, TECTO (Treatment Effects of Family Based Cognitive Therapy in Children and Adolescents with Obsessive-Compulsive Disorder). While the main purpose of TECTO was to assess treatment effects of family-based CBT, the trial was also carefully designed to enable identification of neurocognitive correlates of treatment response.

The TECTO trial

This section briefly describes design, methods, and participants of the TECTO study (Pagsberg et al., 2025). For more details, see the protocol (Pagsberg et al., 2022) and statistical analysis plan (Olsen M.H. et al., 2022). TECTO investigates benefits and harms of family-based Cognitive-Behavioral Therapy (CBT) compared to an active control treatment of Psychoeducation and Relaxation Training (PRT) in a superiority RCT design (Figure 1). In addition, TECTO is combined with a longitudinal case-control study investigating neural, neurocognitive, emotional, and endocrine correlates of treatment response, assessed before and after treatment.

Figure 1 TECTO: Design and number of participants



Participants

The RCT included 130 non-medicated pediatric patients (age: 8-17 years) with moderate to severe OCD who were randomized to either CBT (N = 64) or PRT (N = 66) and received 14 psychotherapy sessions over 16 weeks (Figure 1). Patients were recruited from the total pool of children referred with suspected OCD at the Child and Adolescent Mental Health Center, Copenhagen University Hospital – Mental Health Services CPH. For the case-control study, 90 age- and sex-matched non-psychiatric control children were included via the Danish Central Person Registry. Inclusion- and exclusion criteria of patients and controls are provided in Table 1.

Table 1 TECTO: Inclusion and exclusion criteria

Participants	Inclusion criteria	Exclusion criteria
All	Age 8 - 17 years (both inclusive) Signed informed consent	Intelligence quotient (IQ) < 70, assessed with the WISC-V or WAIS-IV
Patients	OCD as primary diagnosis, meeting the criteria for ICD-10 F42.2, verified with the K-SADS interview CY-BOCS entry score ≥ 16 *	Comorbidity that contraindicates trial participation ** OCD treatment within the last 6 months ***
Controls	Sex and age (+/- 3 months) match with an included patient	Any current or previous psychiatric disorder according to ICD-10 criteria, assessed by the K-SADS interview at the time of screening

Note: K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia, Present and Lifetime Version. *) This cut-off score is used in previous studies and chosen for comparability. **) Pervasive developmental disorder not including Asperger's Syndrome (ICD-10 F84.0-84.4 + F84.8-84.9), Schizophrenia/Paranoid psychosis (ICD-10 F20-25 + F28-29), mania or bipolar disorder (ICD-10 F30 and F31), depressive psychotic disorders (ICD-10 F32.3 + F33.3), substance dependence syndrome (ICD-10 F1x.2). ***) CBT, PRT, SSRI, or other antidepressant/antipsychotic medication.

Before inclusion of the first participant, the trial was registered on ClinicalTrials.gov (NCT03595098; July 23, 2018), and participants were assessed between August 2018 and April 2022. Note that since ICD-11 was not yet implemented in Denmark in the assessment period, we used the ICD-10 (World Health Organization, 1992) version for confirming diagnoses.

Clinical assessments

Patients were assessed by trained clinicians (psychologists or medical doctors), while controls were evaluated by the same clinicians or by supervised master's-level psychology students.

Diagnostic status was based on transference to diagnostic criteria in ICD-10 of item criteria from the semi-structured psychopathological interview Kiddie Schedule for Affective Disorders and Schizophrenia, Present and Lifetime Version (K-SADS) (Kaufman et al., 1997) which assesses psychopathology based on the DSM-IV manual. In TECTO, K-SADS was used to confirm a primary diagnosis of OCD and eventual comorbidity in patients and to ensure no prior or present psychopathology in controls. The child and the parent(s) were interviewed separately by the same

assessor. In patients, diagnostic status was conferred with a specialist (psychiatrist or psychologist).

The primary outcome of the TECTO trial was OCD symptom severity, measured with the gold-standard Children's Yale-Brown Obsessive-Compulsive Scale (CY-BOCS) total score (Scahill et al., 1997). CY-BOCS is a semi-structured, clinician-administered interview. In TECTO, it was administered to the child and the parent(s) or in some cases only to the young person. CY-BOCS assesses obsessions and compulsions during the last week with five items each, rating frequency, disturbance, functional level, resistance, and control of symptoms on a Likert-scale of 0-4, adding up to the total score ranging from 0-40.

Clinician-rated Clinical Global Impression was assessed for severity (CGI-S) and improvement (CGI-I) (Busner & Targum, 2007).

Clinician-rated remission was defined in two ways at follow-up: 1) No longer meeting diagnostic criteria for OCD (ICD-10; F42; assessed with K-SADS) and 2) CY-BOCS total score < 11. Clinician-rated treatment response was defined at follow-up as a reduction of ≥ 30 % from baseline in CY-BOCS total score. Child treatment completion was defined as participation in 10 out of 14 sessions within 18 weeks.

Psychotherapeutic interventions

Both TECTO interventions consisted of 14 individual therapy sessions, each lasting 75 minutes and delivered over 16 weeks (Pagsberg et al., 2025). They were both family-based; parents participated together with their child in five full sessions (sessions 1, 2, 7, 11, and 14). In the remaining nine sessions, the child received 45 minutes of individual treatment, followed by a 30-minute parent session without the child present. The treatment was delivered by clinical psychologists or child and adolescent psychiatrists who provided both interventions and received bi-weekly supervision.

Cognitive-Behavioral Therapy with Exposure and Response Prevention (experimental treatment)

The experimental treatment consisted of CBT with ERP, based on the manuals developed by March and Mulle, Barrett and colleagues, and Piacentini and colleagues (March & Mulle, 1998; Barrett et al., 2008; Piacentini et al., 2011). We used the Danish manual adapted for ERP- and

family-based CBT for pediatric OCD, developed for the Nordic OCD treatment study (NordLOTS) (Thomsen et al., 2013; Thomsen & Weidle, 2015). Key components of the treatment included psychoeducation, ERP exercises with associated homework, and active parental involvement, primarily in supporting ERP practice and reducing family accommodation. Additionally, many treatment sessions included activities on cognitive training/restructuring aimed at enhancing control over OCD.

Psychoeducation and Relaxation Training (active control treatment)

The active control treatment consisted of PRT, based on the relaxation manual by Cautela and Groden and adaptions by Piacentini and colleagues (Cautela & Groden, 1978; Piacentini et al., 2011). Key components of the treatment were psychoeducation, relaxation training (including attention training and visualization techniques) and associated homework, and parental involvement, primarily in supporting relaxation practice.

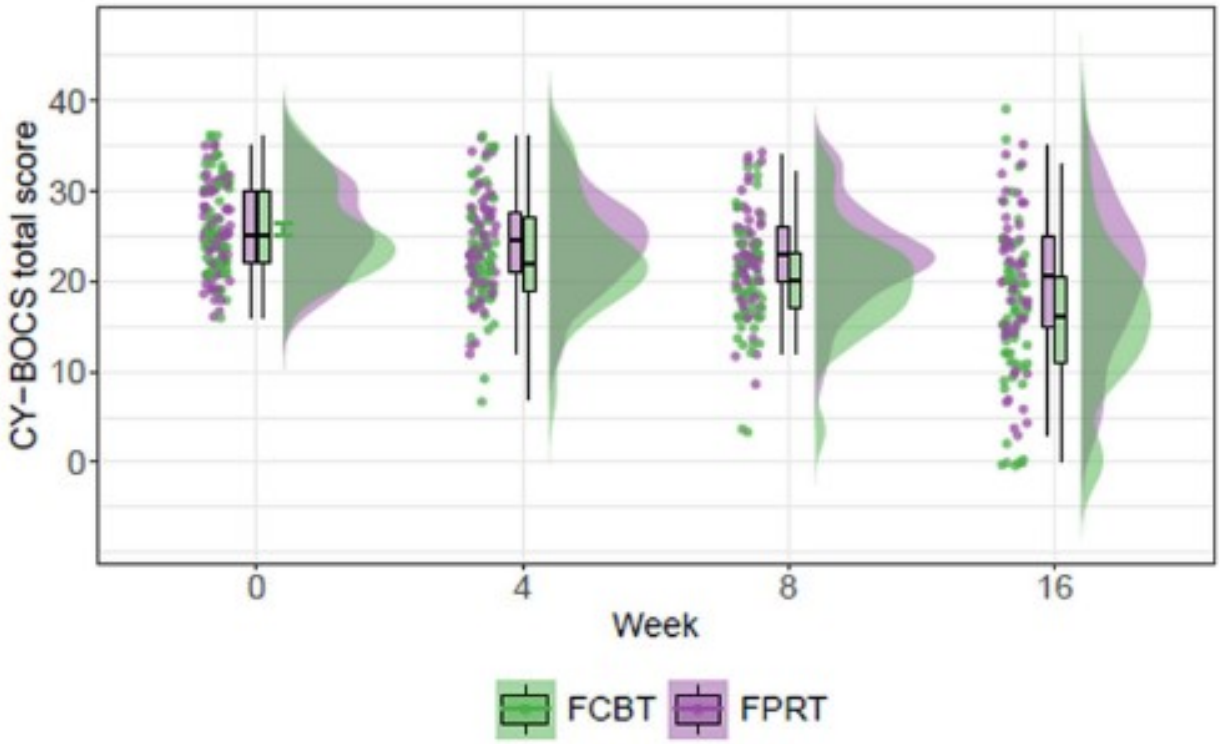
Common factors

The two treatment conditions shared several common elements, including overall structure (number and duration of sessions), level of therapist involvement, amount of homework, extent of family participation, and provision of psychoeducation about OCD. Additional shared components included developing a symptom list and -hierarchy, establishing treatment goals, and conducting cognitive restructuring exercises aimed at externalizing the OCD. Thus, the ingredients primarily disentangling the two interventions were ERP practice and cognitive restructuring aimed at controlling the OCD (CBT) versus relaxation practice.

RCT result

The main result included the intention-to-treat population (CBT = 64, PRT = 66). CY-BOCS total score was significantly lower at end-of-treatment for CBT (*M* = 15.9, *SD* = 8.86) than for PRT (*M* = 19.9, *SD* = 8.09) with a mean difference of -3.89 (*p* = .010) (Pagsberg et al., 2025) (Figure 2). Post-hoc per-protocol analyses restricted to treatment-completers (including participants who completed 10/14 sessions; CBT = 52, PRT = 41) showed a non-significant mean difference of -2.59, *p* = 0.098. Response rate (≥ 30 % reduction on CY-BOCS total score) was higher for CBT (*n* = 30, 46,9 %) versus PRT (n = 17, 25.8 %), *p* = .042.

Figure 2 TECTO: Result of the randomized clinical trial



Note: The figure is reproduced without modification from Pagsberg et al. (2025) under the CC BY-NC-SA license. CY-BOCS = Children's Yale-Brown Obsessive-Compulsive Scale. FCBT = Family-based Cognitive-Behavioral Therapy. FPRT = Family-based Psychoeducation and Relaxation Training.

Neurocognitive tests

Neurocognitive assessment took place at two time points: 1) Baseline (i.e., pre-treatment; week 0) and 2) follow-up (i.e., end-of-treatment; week 16). The test battery was administered and scored by trained psychologists and research assistants (psychology students) under supervision of a trained psychologist. All assessors received supervision by a senior neuropsychologist. Assessors were blinded to treatment allocation but not to diagnostic status (patients versus controls).

Selected tests and outcomes for baseline analyses (Study 1)

Based on the CSTC model and the habit hypothesis of OCD, affected individuals are expected to exhibit EF difficulties centered around cognitive inflexibility (Graybiel & Rauch, 2000; Gillan & Robbins, 2014). Empirical evidence supports small difficulties in planning, visuospatial working memory, decision-making under uncertainty, and cognitive flexibility (Abramovitch et al., 2015; Marzuki et al., 2020; Abramovitch et al., 2021). On this basis, we selected a comprehensive neurocognitive test battery assessing core executive functions (namely cognitive flexibility) and related neurocognitive domains (see Table 2).

The battery comprised 16 outcome measures drawn from widely used standardized, validated instruments: Four from the Cambridge Neuropsychological Test Automated Battery (CANTAB; (Cambridge Cognition, 2019)), three from the Delis-Kaplan Executive Function System (D-KEFS; (Delis et al., 2001a, 2001b)), and one from the Wechsler scales (Wechsler, 2008, 2014). While CANTAB is considered language independent, we used the validated Danish versions of the D-KEFS and the Wechsler scales. General cognitive ability (IQ) was assessed with the General Ability Index (GAI) from the Wechsler scales (described in detail in a separate section).

Notably, although inhibitory control is considered a core executive function, it was assessed in a different sub-study of TECTO (using a stop signal task and fMRI) and thus is not prioritized as an outcome of the current studies. However, it should be noted that inhibitory control is thought to be a crucial part of flexibility tasks.

Table 2 Neurocognitive tests and selected outcomes

Cognitive domains/ tests	Test description	Selected outcomes	Assessed	
			W0	**W16**
Cognitive flexibility				
Attention Switching Task (AST)[a]	Computerized test in which participants respond "right" or "left" based on either the direction or location of an arrow. The response rule switces continually throughout the task.	**Percent correct trials (AST-PCT)**; Switch cost[d]	X	X
Trail Making Test (TMT); Number Letter Switching[b]	Paper-pencil test in which the participant draws a line connecting numbers and letters in ascending order while alternating between them (i.e. 1-A-2-B etc.).	**Completion time in seconds (TMT-SRT)**	X	X
Verbal Fluency Switching[b]	Verbal test in which the participant names as many words as possible in 60 seconds, alternating between two categories (e.g. apple-table-pear-sofa).	Number of correct switches	X	X
Design Fluency, Switching[b]	Paper-pencil test in which requires the participant draws as many different figures as possible in 60 seconds by connecting dots with straight lines, continually switching between filled and empty dots.	Number of correct figures	X	X
Planning and decision-making				
Stockings of Cambridge (SOC)[a]	Computerized test in which the participant copies a model by rearranging items to match the model, trying to use the minimum moves required.	Problems solved in min. moves[e]	X	X
Spatial Working Memory (SWM)[a]	Computerized test in which an increasing number of boxes are presented on the screen, and the participant is instructed to find a blue token behind one of them. When the token is found, it is hidden behind a new box and the participant is asked to avoid returning to a box where a token has already been found.	Strategy[f]	X	X
Cambridge Gambling Task (CGT)[a]	Computerized test in which the participant guesses whether a token is hidden behind red boxes or blue boxes. On each trial, ten boxes are presented and the ratio of blue:red boxes is varied. The participant bets points on their decision, and points are won or subtracted according to outcome and bet-size.	**Quality of decision-making[g] (CGT-QDM)**; Risk adjustment[h]	X	X
Working memory				
Wechsler Working Memory Tests[c]	Visual and verbal subtests of the Wechsler Scales, including Verbal digit span, Picture span task (WISC-V) and arithmetic (WAIS-IV).	Index Score	X	
Wechsler Verbal Digit Span[c]	Verbal task where the participant should recall sequences of digits in forward, backward, and sequencing (ascending numerical order)	Scale score	(X)	X
Spatial Working Memory (SWM)	*See above*	Total errors	X	X
Fluency				
Verbal Fluency, Phonemic[b]	Verbal test with three subtests in which the participant has 60 seconds to name as many words as possible beginning with a specific letter.	Number of correct words	X	X
Verbal Fluency, Semantic[b]	Verbal test with two subtests in which the participant has 60 seconds to name as many words as possible from a specific semantic category.	Number of correct words	X	X
Design Fluency, Empty dots[b]	Paper-pencil test in which the participant draws as many different figures as possible in 60 seconds, connecting empty dots and ignoring filled dots.	Number of correct figures	X	X
Processing speed				
Trail Making Test; Number sequencing[b] and Letter Sequencing	Paper-pencil test in which the participant draws a line, connecting numbers in ascending order or letters in alphabetical order.	Completion time in seconds	X	X

Note: Adapted from Study 1 (Uhre et al., 2023) under the CC BY-NC-SA license. Here, primary outcomes of the neurocognitive follow-up study (Study 2) are marked in bold, Wechsler Verbal Digit Span (subtask of the WMI) is added, and measurement time points are indicated. [a] Tests from the CANTAB battery. [b] Tests from the D-KEFS battery. [c] Tests from the Wechsler Scales: WISC-V and WAIS-IV. W0 = Week 0; baseline. W16 = Week 16; follow-up (end-of-treatment for patients with OCD). [d] Switch cost = The difference in the average reaction time (RT) in milliseconds between switch and repeat trials [e] Problems solved in minimum moves = The number of trials in which the participant matched the model by using as few moves as possible. [f] Strategy = Based on the number of times the participant started each search within a trial with a new box instead of starting with the same box and following a systematic sequence – lower scores represent more efficient strategy use [g] Quality of decision-making = Proportion of trials where participant chose most likely outcome – higher scores represent better decision-making [h] Risk adjustment = Degree to which participant adjusted bet size according to ratio of red:blue boxes – higher scores means better adjustment

Cognitive flexibility

Cognitive flexibility was assessed using four different tasks: The computerized CANTAB Attention Switching Task (AST) and the switching-conditions of the paper-and-pen based D-KEFS Trail Making Task (TMT), Design Fluency Task (DFT), and Verbal Fluency Task (VFT).

The CANTAB AST (Cambridge Cognition, 2019) is a cued switch-task where the participant categorizes stimuli based on two different, switching cues, using the same response keys for both. The participant sits in front of a computer screen and holds a press-pad with the response options "right" or "left". In each trial, an arrow is shown on the screen; the arrow is placed in the right or left side of the screen and pointing either right or left. Also, an instruction (a cue) is shown on each trial; if the instruction says "Side", the participant should respond based on the location of the arrow, and if the instruction says "Direction", the participant should respond based on the direction the arrow points.

Two outcomes from the task were selected: 1) Percent correct trials (percentage of trials, all trials included, where the right answer was chosen) and 2) Switch cost (difference in average RT (milliseconds) on switch and repeat trials).

The D-KEFS TMT, switching condition (Delis et al., 2001a, 2001b) requires the participant draw a line and switch between connecting numbers and letters in numeric and alphabetic order (e.g., 1-A-2-B-3-C etc.). The outcome is completion time in seconds.

The D-KEFS DFT, switching condition, requires the participant to draw as many different figures as possible in 60 seconds by connecting dots with straight lines, switching between black and white dots. The outcome is the number of correct figures completed within time.

Finally, in the D-KEFS VF, switching condition, the participant is instructed to name as many words as possible in 60 seconds while switching between the two categories "fruit" and "furniture" (e.g., apple, chair, pear, table etc.). The outcome is the number of correct switches from one category to the other.

Planning and decision-making

Planning was assessed with two tasks from the computerized CANTAB-battery: The Stockings of Cambridge (SOC) task and the Spatial Working Memory (SWM) task.

The SOC task is based on the Tower of Hanoi. The participant is told to rearrange colored balls to match a model, using as few moves as possible. The minimum number of moves required to match

the model ascends from 1-5. The participant is instructed to plan the moves before initiating them. The outcome is the number of trials where the participant solved the task with as few moves as possible (termed "problems solved in minimum moves"). A higher score reflects better planning.

In the SWM task, the participant is presented with a number of colored boxes on the screen and instructed to find a blue token behind one of them. The number of boxes on the screen increases during the test. When the participant finds the token, a new token is hidden behind a new box. A token will never be hidden in the same place twice, and the participant is instructed not to return to a box where a token has already been found. Planning is assessed in this task from the outcome "strategy" which is calculated based on the number of times the participant started each search (within a trial) with a new box instead of following a systematic sequence. A lower score means that the participant followed a systematic sequence, reflecting better planning.

Decision-making was evaluated using the computerized CANTAB Cambridge Gambling Task (CGT), similar to the Iowa Gambling Task. In this test, the participant views ten colored boxes on the screen – some are red, and some are blue – and are asked to guess, whether a yellow token is hidden behind a red or a blue box. The ratio of red to blue boxes varies across trials from 1:9 to 9:1. In each trial, the participant wagers points on their chosen color. If their choice is correct, their points are doubled, and if wrong, their points are halved. Participants are encouraged to accumulate as many points as possible. We used two outcomes from this task: Quality of decision-making, i.e. the proportion of trials where the most likely outcome was chosen, and Risk Adjustment, i.e. the degree to which the bet size was adjusted according to the ratio of red versus blue boxes. For both outcomes, higher scores reflect better decision-making.

Working memory

Working memory was assessed with the Wechsler working memory index tasks. The index is based on the subtests verbal digit span, picture span task, and (in WAIS-IV) arithmetic. In the digit span test, the participant is asked to recall sequences of digits in three ways: 1) forward, 2) backward, and 3) in ascending numerical order. The picture span task requires the participant to recall and sequence pictures of common objects in the order they were presented. Finally, the arithmetic task consists of mathematical problems increasing in difficulty that the participant is asked to solve (verbally).

Spatial working memory was assessed from the CANTAB SWM task (see description above). The outcome used was the total errors (the number of times the participant chose a box where a token had already been found).

Fluency

Verbal fluency was assessed using the D-KEFS Verbal fluency test. In the phonemic condition, participants were instructed to generate as many words as possible within 60 seconds that begin with the letter "f". In the semantic condition, they were asked to produce as many words as possible belonging to the categories "animals" and "boy's names". The outcome is the number of correct words generated within time.

Design fluency was assessed with the D-KEFS Design fluency task in which the participant is instructed to draw as many different figures as possible in 60 seconds by connecting white dots with straight lines while ignoring black dots. The outcome is the number of correct figures completed within time.

Processing speed

Finally, processing speed was measured with the D-KEFS Trail making task, number- and letter sequencing conditions. In these tasks, participants are asked to draw lines connecting numbers in numerical order and letters in alphabetic order, accordingly, as quickly as possible. The outcome is completion time in seconds.

Selected primary outcomes for follow-up analyses (Study 2)

Before initiating analyses of the neurocognitive follow-up study (Study 2), three primary outcomes were selected for hypothesis testing. The selection was based on the results of the baseline neurocognitive study (Study 1; Uhre et al. (2023)). As primary outcomes, we selected core EF outcomes assessed at both baseline and follow-up for which patients with OCD performed significantly worse than controls ($p < .05$, uncorrected).

Thus, we selected two measures of cognitive flexibility and one of decision-making under uncertainty as our primary outcomes:

- AST percent correct trials (AST-PCT; from the CANTAB Attention Switching Task)
- TMT switching time in seconds (TMT-SRT; from the D-KEFS Trail Making Task, switching condition)

- CGT quality of decision-making (CGT-QDM; from the CANTAB Cambridge Gambling Task)

Note that although patients underperformed compared with controls on the Wechsler Working Memory Index at baseline, this measure was not included as a primary outcome at follow-up because data were not available at both time points (due to the retest rules of the Wechsler scales). Also note that although patients performed worse than controls on the TMT Number Sequencing measure of processing speed, this measure was not designated as a primary outcome because our main focus was on core executive functions. TMT Number Sequencing as well as the remaining outcomes of the neurocognitive test battery (Table **2**) will be analyzed in post-hoc explorative analyses.

General cognitive ability

General cognitive ability (IQ) was included as an exclusion criterion in the clinical trial and as a covariate in baseline analyses of neurocognitive performance.

IQ was assessed at baseline with the Wechsler scales, age appropriate versions; in participants aged 8-16 (both included), the tablet version of the Wechsler Intelligence Scale for Children, 5th Edition (WISC-V) (Wechsler, 2014) was applied, and for participants aged 17, the analogue Wechsler Adult Intelligence Scale, 4th Edition (WAIS-IV) (Wechsler, 2008) was used. The test batteries produce primary index scores reflecting verbal comprehension (VCI), visuospatial ability (VSI), fluid reasoning (FRI), working memory (WMI), and processing speed (PSI), and these add up to the global IQ. Scores are standardized relative to a Scandinavian reference population stratified by age and sex, with normative values set to a mean of 100 and a SD of 15.

As an alternative measure of IQ, the Wechsler Global Ability Index (GAI) can be applied. The GAI is derived from the VCI, VSI, and FRI, and excludes the WMI and PSI. Because the WMI and PSI are often impaired in clinical populations and tend to show lower reliability than the remaining indexes, the GAI helps reduce the influence of working memory and processing speed and provides a purer estimate of core intellectual abilities.

IQ was used in the TECTO trial to exclude participants with intellectual disability (IQ < 70; 2 SDs below the normative mean). The same approach has been used in previous trials (Thomsen et al., 2013), and notably, lower IQ has been associated with poorer response to CBT (D'Alcante et al., 2012). In addition, given the aims of the neurocognitive sub-study, we sought to include participants with normal-range IQ. When general cognitive ability is impaired, specific cognitive

deficits (e.g., in cognitive flexibility) are also likely affected (Diamond, 2020); however, in the present study, our objective was to examine individuals with intact overall abilities to determine whether specific impairments were still present (i.e., we aimed to make sure IQ was not confounding neurocognitive outcomes).

While IQ is affected in mental illnesses like depression, ADHD, and schizophrenia (i.e., slightly impaired general cognitive ability is seen on a group level in some but not all individuals) IQ is generally within typical range in groups with pediatric as well as adult OCD (Abramovitch et al., 2018; Koenen et al., 2009; Kuntsi et al., 2004). However, non-psychiatric comparison groups in clinical studies tend to appear "super healthy" and score above the normative mean on a group level (like in the meta-analysis of IQ in OCD by Abramovitch et al. 2018). As this was also the case in our study, we chose to add sensitivity analyses including IQ, when patients with OCD were compared to controls on neurocognitive measures.

Parent- and self-rated executive functioning

For rating-based assessment of child-EF, we used the Danish versions of the BRIF-2 questionnaires, parent-report and self-report version (Gioia et al., 2015, 2018). The parent-rated version contains 63 items. The self-rated version contains 55 items and is approved for children aged 11-18 years. Parent- and self-report share 29 items, and the remaining items are very similar, differing mainly in wording (synonyms) or in the examples provided. The items describe problematic types of behavior related to EF demanding tasks in daily-life. Examples of items related to flexibility are: "Fixates on the same topic for too long", "Has difficulty shifting from one activity to another", and "Resists or has difficulty accepting alternative ways of solving problems (e.g., homework, social situations, tasks)". The informant rates the frequency on a 3-point Likert-scale (1 = "Never", 2 = "Sometimes", 3 = "Often"). Nine subscales are produced for parent-report and seven for self-report, representing subdomains of EF (for details, see Table 3). The subscales add up to three indexes (behavioral-, emotional, and cognitive regulation) as well as a total score (the global executive composite; GEC) reflecting the overall EF capacity.

For our study, we chose to report results from all subscales. While previous studies (like Hybel et al. (2017)) report only the GEC, we wished to study subdomains rather than global functioning of EF. This also enables comparison with the results of the neurocognitive tests. Using the Hogrefe Test System (HTS 5) (Gioia et al., 2018), we standardized scale results to T-scores (mean = 50, SD = 10) based on Danish normative samples. The BRIEF questionnaires have proven sensitive

as a measure of change in more than 40 treatment studies, and the subscales have demonstrated acceptable to high internal consistency (Cronbach's α = .70 – .95) (Gioia et al., 2018).

Table 3 Description of BRIEF-II subscales

Subscale	Number of Items		Rated Behavior
	Parent-Report	Self-Report	
Inhibition	8	8	Controlling impulses, inhibiting behavior or distractions not relevant to a current activity
Flexibility/Shift	8	8	Adapting to different situations/activities; flexible problem solving
Emotional Control	8	6	Regulation emotional reactions
Self-Monitoring	4	5	Monitoring the effect of one's own behavior
Initiating	4	-	Initiate task/activity; generate new ideas
Task-Completion	-	7	Finishing schoolwork/domestic duties in time; completing tasks within a time frame; working in a sufficient tempo
Working Memory	8	8	Holding information in mind while finishing a task or continuing an activity
Plan/Organize	8	10	Foresee incidents; set goals; planning relevant steps ahead of fulfilling a task; systematic execution of tasks; understanding/communicating a central idea/concept
Task-Monitoring	6	-	Monitoring and evaluating a task during/after completion, eg. checking for mistakes and assure fulfilling the purpose of the task
Organization of Materials	5	-	Organizing and remembering materials necessary for a task, keeping workspace/play area/materials organized

Note: The table is adapted from Table S1 in the Study 3 supplementary material. Source: BRIEF-2 Manual (Gioia et al., 2015, 2018)

Statistical analyses

Overview of primary statistical models

Because the three studies share objectives and statistical methods, an overview of the primary statistical models is provided in Table 4.

Table 4 Overview of primary statistical models

	Study 1 Neurocognitive baseline study	**Study 2** Neurocognitive follow-up study	**Study 3** BRIEF-2 study
Objectives/hypotheses			
Baseline group differences (OCD versus controls) in EF *- H1a; H3a*	MANOVA (one model) - Across all 16 neurocognitive outcomes	-	MANOVA (two models) - One for each informant, across all BRIEF-2 subscales
Baseline association of EF and OCD symptom severity (patients) *- H1b; H3b*	Multiple linear regression (one model) - Including all 16 neurocognitive outcomes	-	Multiple linear regression (two models) - One for each informant, including all BRIEF-2 subscales
EF change during treatment *- H2a; H3c*	-	Repeated measures ANOVA (three models, CBT versus PRT versus controls) - One for each primary outcome	Repeated measures ANOVA (two models, CBT versus PRT) - One for each informant, including all BRIEF-2 subscales
Association of EF change and OCD symptom severity change during treatment (patients) *- H2b; H3d*	-	Multiple linear regression (three models) - One for each primary outcome	Multiple linear regression (two models) - One for each informant, including all BRIEF-2 subscales
EF as a predictor/moderator of OCD symptom improvement (patients) *- H2c; H2d; H3e; H3f*	-	Multiple linear regression (three models) - One for each primary outcome	Multiple linear regression (two models) - One for each informant, including all BRIEF-2 subscales

Note: EF = Executive functioning. H = Hypothesis. MANOVA = Multivariate Analysis of Variance. ANOVA = Analysis of Variance. BRIEF-2 = Behavior Rating Inventory of Executive Function, Second Edition (Gioia et al., 2015, 2018)

Background variables and correction for multiple tests

The same background variables were measured at baseline and considered across all three studies: Sex (male/female), Age, (years) Parental education (years), General cognitive ability (IQ or GAI), and Psychiatric comorbidity (ICD-10 diagnoses stated with the K-SADS). These variables were selected because they are theoretically and empirically known to influence neurocognitive performance, everyday EF, and/or OCD symptom severity, as well as potentially affecting

treatment response. Parental education was reported in years and calculated as the mean of both parents, following previous work (Hybel et al., 2017). Psychiatric comorbidity was assessed only in patients, since it is not present in controls. Controls were recruited to match patients on age and sex to minimize baseline differences. To assess potential confounding, background variables showing significant baseline differences were included as covariates in sensitivity analyses of cross-sectional comparisons. Comorbidity was examined in sensitivity analyses, comparing patients with and without comorbidity to determine whether neurocognitive differences were robust and independent of comorbidity. For treatment analyses comparing CBT versus PRT, randomization was expected to balance background variables across groups.

Because multiple hypotheses were tested in our studies, corrections were applied to control the family-wise Type I error rate (i.e., to reduce the likelihood of false positive results). In the analyses of neurocognitive performance, Bonferroni correction was used. For each primary outcome, p-values were adjusted across the predefined family of tests.
In the analyses of parent- and self-rated EF, correction for multiple comparisons was performed using the Benjamini-Hochberg (BH) procedure with a false discovery rate (FDR) set at 5 %. Each family of tests included five a priori hypotheses, and because parent- and self-reports often differ in clinical studies, the BH procedure was applied for each informant separately.

Data handling of neurocognitive and rating-based outcomes

Neurocognitive assessors were instructed to ensure valid test administration and adherence to standard procedures. Following this, extreme performance outcomes were considered genuine reflections of participants' abilities rather than invalid test completions and thus included in all analyses. Missing values were not imputed, and analyses were conducted on observed data only.
For follow-up analyses, data was standardized to z-scores (mean = 0, SD = 1) based on the baseline scores of the non-psychiatric control group. Scores were also aligned in the same direction so that higher values consistently reflect better neurocognitive functioning. This standardization was applied to enhance comparability and interpretability across neurocognitive measures.

For the BRIEF-2 subscale analyses of everyday EF, we used T-scores rather than raw-scores. T-scores are standardized scores (mean = 50, SD = 10) computed through linear transformations of raw scores often used for psychological data. The T-scores used in our study derived from Danish normative data and was computed using the Hogrefe Test System (HTS 5) (Gioia et al., 2018). As

participants are rating frequency of problematic behavior related to EF, higher scores indicate greater EF difficulties. T-scores were used to enhance interpretability and to enable meaningful comparisons of EF levels with those of the normative background population. While the non-psychiatric control group allows assessment of functioning relative to a typical comparison sample, reference to standardized normative data provides an additional benchmark and strengthens the validity of the conclusions, especially in cases where the control group may not fully represent the broader population.

While the BRIEF-2 parent-rating is validated across our full age range (8-17 years), the self-report version is validated only for ages 11 and above. Accordingly, everyday EF was examined in two partially overlapping samples, and all analyses were conducted separately for parent- and self-ratings. When two completed the BRIEF-2, their mean subscale score was used, as parent interrater-reliability is high in both previous studies and in our own data.

Sample size estimation

Sample size estimation was performed a priori within the framework of the TECTO trial and the primary outcome of the RCT, CY-BOCS (Scahill et al., 1997). The planned patient sample was powered to detect a four-point clinically meaningful difference on the CY-BOCS total score with 80 % power, a two-sided significance level of .05, and an assumed SD of 8 (Olsen M.H. et al., 2022), thus estimating a minimum of 64 patients in each treatment arm as necessary.

Baseline executive functioning

EF in patients versus controls (Hypothesis 1a)

Group differences in EF between OCD patients and controls at baseline (before treatment for patients with OCD) were analyzed using multivariate analyses of variance (MANOVAs), as EF measures were expected to be interrelated. Group (OCD versus controls) was entered as the independent variable, and either neurocognitive test scores (Neurocog) or BRIEF-2 subscale scores (Subscale) were included as dependent variables. Partial eta squared ($\eta_p{}^2$) were reported as the measure of effects size. The robustness of the group comparison was tested in two sensitivity analyses: one including IQ or GAI as a covariate (Multivariate Analysis of Covariance; MANCOVA), and another restricting the patient sample to those with or without psychiatric comorbidity. Significant MANOVA effects were followed up with univariate analyses of variance (ANOVAs) for each dependent variable to identify which specific outcomes contributed to the overall group differences.

Associations with symptom severity (Hypothesis 1b)

Associations between OCD symptom severity and test outcomes were examined using three multiple linear regression analyses: one including all neurocognitive outcomes, one including BRIEF-2 parent-report subscales, and one including BRIEF-2 self-report subscales, each predicting baseline CY-BOCS Total score (CYBOCS). When a main model was significant, individual predictors were subsequently explored. Thus, each model took the general form:

Neurocognitive predictors:

$$CYBOCS_{W0} = \beta_0 + \sum \beta \cdot (Neurocog_{W0}) + \varepsilon$$

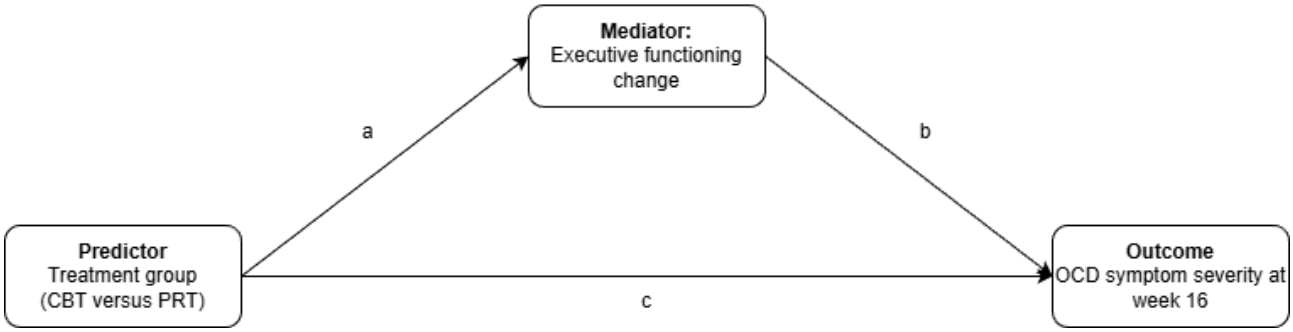
BRIEF-2 Subscale predictors:

$$CYBOCS_{W0} = \beta_0 + \sum \beta \cdot (Subscale_{W0}) + \varepsilon$$

Executive function change after psychotherapy

Our original idea was to examine EF change as a mediator of treatment response (OCD symptom alleviation), using the mediator model of Baron & Kenny (1986) (Figure 3). For the current studies, path *c* was stated significant in Pagsberg et al. (2025). A hypothesis of mediation would be supported, if path *a* and *b* were significant and if the previously significant path *c* were decreased or eliminated when controlling for paths *a* and *b*. However, as paths *a* and *b* turned out non-significant in our results (as we show in the results' section), we did not continue to investigate path *c*.

Figure 3 Executive function change as a mediator of treatment response



Note: The figure is created with draw.io and adapted from Baron & Kenny (1986), Figure 3, under the CC BY-NC-SA license.

EF change in controls versus PRT versus CBT (Hypothesis 2a and Hypothesis 3c)

Change in EF during treatment was analyzed using mixed repeated-measures ANOVAs. For neurocognitive test performance, separate 3 × 2 ANOVAs were conducted for each primary outcome (dependent variable), to allow clear interpretation of test-specific changes, with Group (Controls, PRT, CBT) as a between-subjects factor and Time (baseline, follow-up) as a within-subjects factor:

$$Neurocog = 3\ (Group) \times 2\ (Time)$$

The control group was included in the main analyses because practice-effects were expected on neurocognitive tests and needed to be controlled for. Generalized eta-squared (ges) is reported as the effect size, as it appropriately accounts for within-subject effects.

For BRIEF-2 data, two repeated-measures ANOVAs were conducted; one for parent-rated data, including nine subdomains (2 × 2 × 9) and one for self-rated data, including seven subscales (2 × 2 × 7), with T-scores as the dependent variable, Group (PRT, CBT) as a between-subjects factor, and Time (baseline, follow-up) and Subscale as within-subjects factors. This approach accounts for scale correlations while maintaining interpretability.

Thus, for parent-report and self-report, the models were:

$$Tscore = 2\ (Group: CBT, PRT) \times 2\ (Time) \times 9\ (Subscale)$$

$$Tscore = 2\ (Group: CBT, PRT) \times 2\ (Time) \times 7\ (Subscale)$$

The control group was not included in these BRIEF-2 analyses because no test-retest effects were expected for behavioral ratings, and none were observed in our sample.
Significant main effects of Time were interpreted as overall change across groups, whereas significant Group × Time interactions were interpreted as differential change between groups.

Note that significant change in CBT versus PRT tests path *a* of the mediation model (Figure 3).

Associations with symptom severity change (Hypothesis 2b and Hypothesis 3d)
Associations of change in OCD symptom severity and change (Δ) in neurocognitive outcomes were analyzed with separate simple linear regression models – one for each outcome. The models had CY-BOCS Total score at follow-up as the dependent variable, controlling for CY-BOCS$_{baseline}$, Thus, each model took the general form:

$$CYBOCS_{follow-up} = \beta_0 + \beta_1 \cdot CYBOCS_{baseline} + \beta_2 \cdot \Delta Neurocog + \varepsilon$$

For BRIEF-2, we used multiple linear regression models. Each model (parent-report and self-report) took the form:

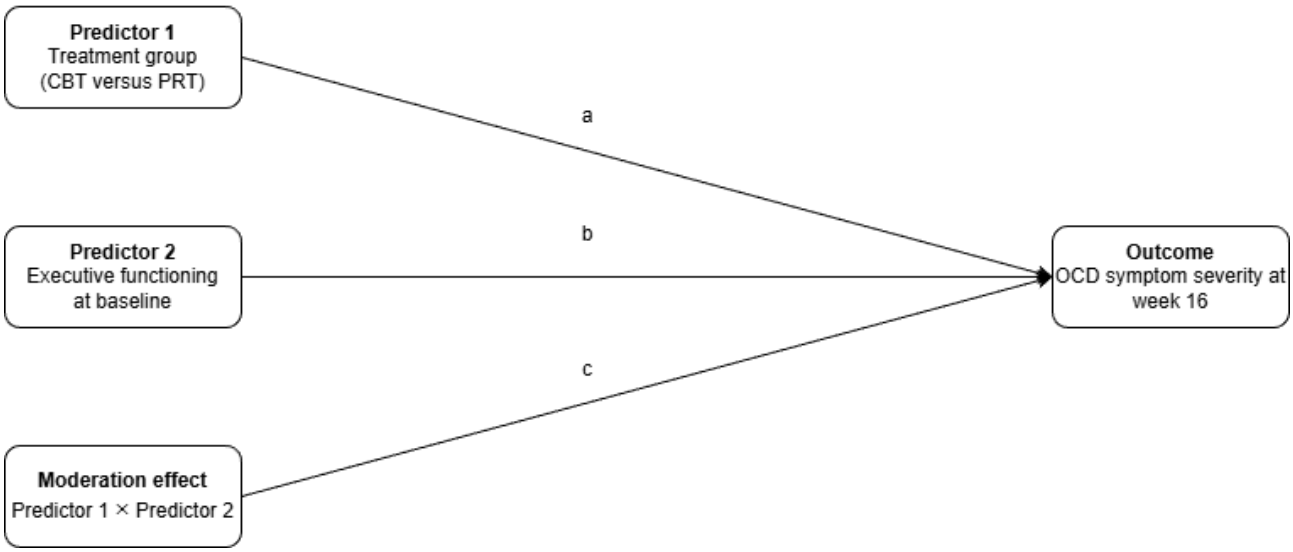
$$CYBOCS_{follow-up} = \beta_0 + \beta_1 \cdot CYBOCS_{baseline} + \sum \beta \cdot (\Delta Subscale) + \varepsilon$$

Note that these models test path *b* of the mediation model (Figure 3).

Executive function as a predictor and moderator of treatment response

We analyzed both neurocognitive and rating-based measures as potential predictors and moderators of treatment response (OCD symptom alleviation), using the moderator model of Baron & Kenny (1986) (Figure 4). For the current studies, path *a* was stated significant in Pagsberg et al. (2025). Our hypothesis of prediction is supported, if path *b* is significant, and our hypothesis of moderation is supported, if path *c* is significant.

Figure 4 Executive functioning as a predictor and moderator of treatment response



Note: The figure is created with draw.io and adapted from Baron & Kenny (1986), Figure 1, under the CC BY-NC-SA license.

Neurocognitive outcomes (Hypothesis 2c and Hypothesis 2d)

Linear regression models were conducted with CY-BOCS$_{\text{follow-up}}$ as the dependent variable, controlling for CY-BOCS$_{\text{baseline}}$, including Neurocog, Group (CBT versus PRT), and Group × Neurocog interaction.

Neurocognitive domains were analyzed in separate regression models because the study focused on three specific outcomes of interest and analyzing them independently allows for clearer interpretation of domain-specific changes. Thus, for each neurocognitive outcome, we used the following model:

$$CYBOCS_{W16} = \beta_0 + \beta_1 \cdot CYBOCS_{W0} + \beta_2 \cdot Group + \beta_3 \cdot Neurocog_{W0} + \beta_4 \cdot (Group \times Neurocog_{W0}) + \varepsilon$$

BRIEF-2 outcomes (Hypothesis 3e and Hypothesis 3f)

In contrast, BRIEF-2 subscales were entered into two single multiple regression models (one for parent-report and one for self-report) because they are conceptually and statistically interrelated; analyzing them together allows identification of their unique contributions while controlling for shared variance.

Thus, for each group of informants, we proposed:

$$CYBOCS_{W16} = \beta_0 + \beta_1 \cdot CYBOCS_{W0} + \beta_2 \cdot Group + \sum \beta \cdot Subscale_{T1} + \sum \beta \cdot (Group \times Subscale_{W0}) + \varepsilon$$

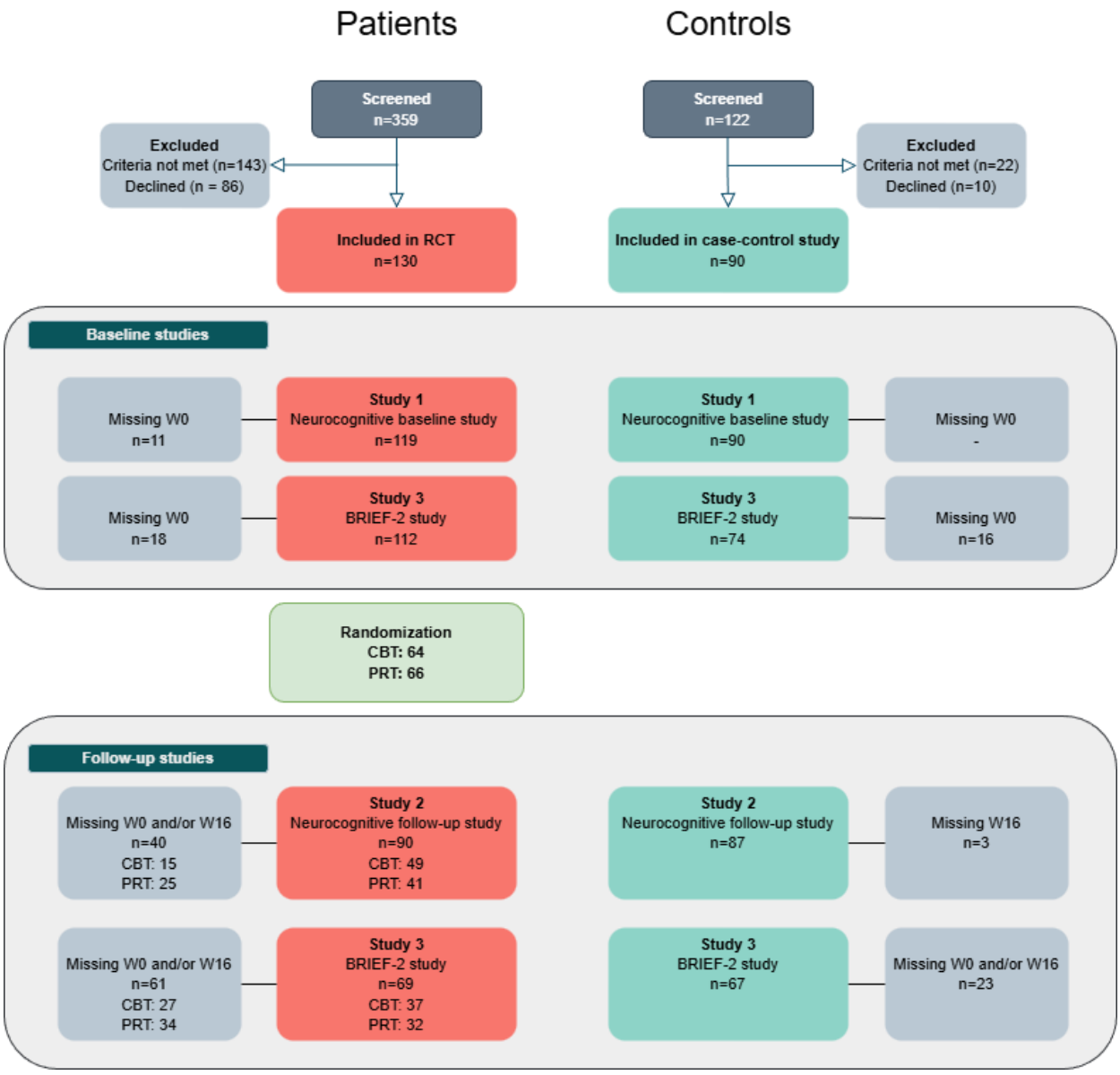
Note that these model tests path *b* and *c* of the moderation model (Figure 4). A significant main effect of Neurocog/Subscale was interpreted as evidence that the measure *predicted* treatment response (regardless of which treatment group). A significant Goup × Neurocog/Subscale interaction was interpreted as *moderation*, indicating that treatment response was predicted differently for CBT versus PRT.

RESULTS

Participants

We aimed to include all participants from the TECTO trial and the longitudinal case-control study. For a simplified flowchart of included and excluded participants, see Figure 5.

Figure 5 Participant flow chart



Note: W0 = Week 0; baseline. W16 = Week 16; follow-up. For BRIEF-2 results, this figure only shows the number of children with parent-report data – for details on self-report, see Study 3. Prediction and moderation allowed missing neurocognitive/BRIEF-2 data at W16 in patients, if CY-BOCS data were complete.; thus, Study 2 included 103 patients (CBT: 54, PRT: 49), and Study 3 included 98 patients (CBT: 50, PRT: 48) for prediction and moderation analyses.

Participants with OCD and controls were similar in terms of mean age and sex distribution across all studies, while mean IQ total score and mean parental education in years differed (see Table 5). Thus, IQ, but not parental education, was included as a covariate in baseline sensitivity analyses to avoid over-adjustment due to their shared variance. A small number of participants with OCD were prescribed medications for comorbid psychiatric or neurological conditions, including melatonin (n = 8; sleep disorders), methylphenidate (n = 1; ADHD), and valproate (n = 1; epilepsy) (Uhre et al., 2023).

Table 5 Participant characteristics

	Study 1 The neurocognitive baseline study		**Study 2** The neurocognitive follow-up study			**Study 3** The BRIEF-2 study		
	CTR Mean (SD) n (%)	**OCD** Mean (SD) n (%)	**CTR** Mean (SD) n (%)	**PRT** Mean (SD) n (%)	**CBT** Mean (SD) n (%)	**CTR** Mean (SD) n (%)	**PRT** Mean (SD) n (%)	**CBT** Mean (SD) n (%)
Number of participants	90	119	87	41	49	67	32	37
Number of females	47 (52.2)	62 (52.1)	44 (50.6)	22 (53.7)	31 (63.3)	34 (50.7)	18 (56.3)	23.0 (62.2)
Age in years	12.93 (2.8)	13.34 (2.8)	13.0 (2.8)	13.3 (2.6)	13.2 (2.8)	13.3 (2.7)	13.3 (2.73)	12.9 (2.8)
Parental education, years	16.8 (1.8)	15.56 (2.1)	16.8 (1.8)	15.7 (1.7)	15.4 (2.5)	16.7 (1.8)	15.9 (1.6)	15.3 (2.6)
IQ (Study 1: GAI)	106.5(13.7)	98.76(12.8)	106.0(13.9)	101.0(13.9)	99.1 (12.7)	105 (14.2)	103.0(13.2)	101.0(12.8)
CY-BOCS total score	-	25.5 (4.9)	-	25.2 (4.4)	24.2 (4.07)	-	24.9 (3.94)	24.1 (3.9)
Any comorbid diagnosis	-	66 (55.5)	-	21 (51.2)	27 (55.3)	-	18 (56.3)	20 (54.1)

Note: For Study 1, this table shows participants with data on at least one neurocognitive outcome at baseline. For Studies 2 and 3, the table shows participants with data on at least one neurocognitive outcome or BRIEF-2 subscale, accordingly, at baseline and follow-up. For Study 3, this table only shows the number of children with BRIEF-2 parent-report data – for details on self-report, see Study 3. Also for Study 3, additional participants were included for baseline analyses; thus, Study 3 included 112 patients and 74 controls. Prediction and moderation allowed missing neurocognitive/BRIEF-2 data at follow-up in patients, if CY-BOCS data were complete.; thus, Study 2 included 103 patients (CBT: 54, PRT: 49), and Study 3 included 98 patients (CBT: 50, PRT: 48) for prediction and moderation analyses. IQ = Intelligence quotient. GAI = General Ability Index.

Study 1: The neurocognitive baseline study

Hypothesis 1a: Children and adolescents with OCD performed significantly poorer than controls on six of 16 neurocognitive outcomes assessed (Table 6): These included two measures of cognitive flexibility (AST-PCT, $p = .033$, $\eta_p^2 = .024$; and TMT-SRT, $p = .024$, $\eta_p^2 = .026$), one measure of decision-making (CGT-QDM, $p < .001$, $\eta_p^2 = .070$), one measure of working memory (WMI, $p < .001$, $\eta_p^2 = .073$), and one measure of processing speed (TMT number sequencing, RT, $p = .005$, $\eta_p^2 = .040$). The effect size was moderate for CGT-QDM and small for the remaining outcomes. Significant group differences remained in sensitivity analyses accounting for comorbidity and IQ (GAI).

Hypothesis 1b: Neurocognitive performance was not associated with OCD symptom severity for any outcome.

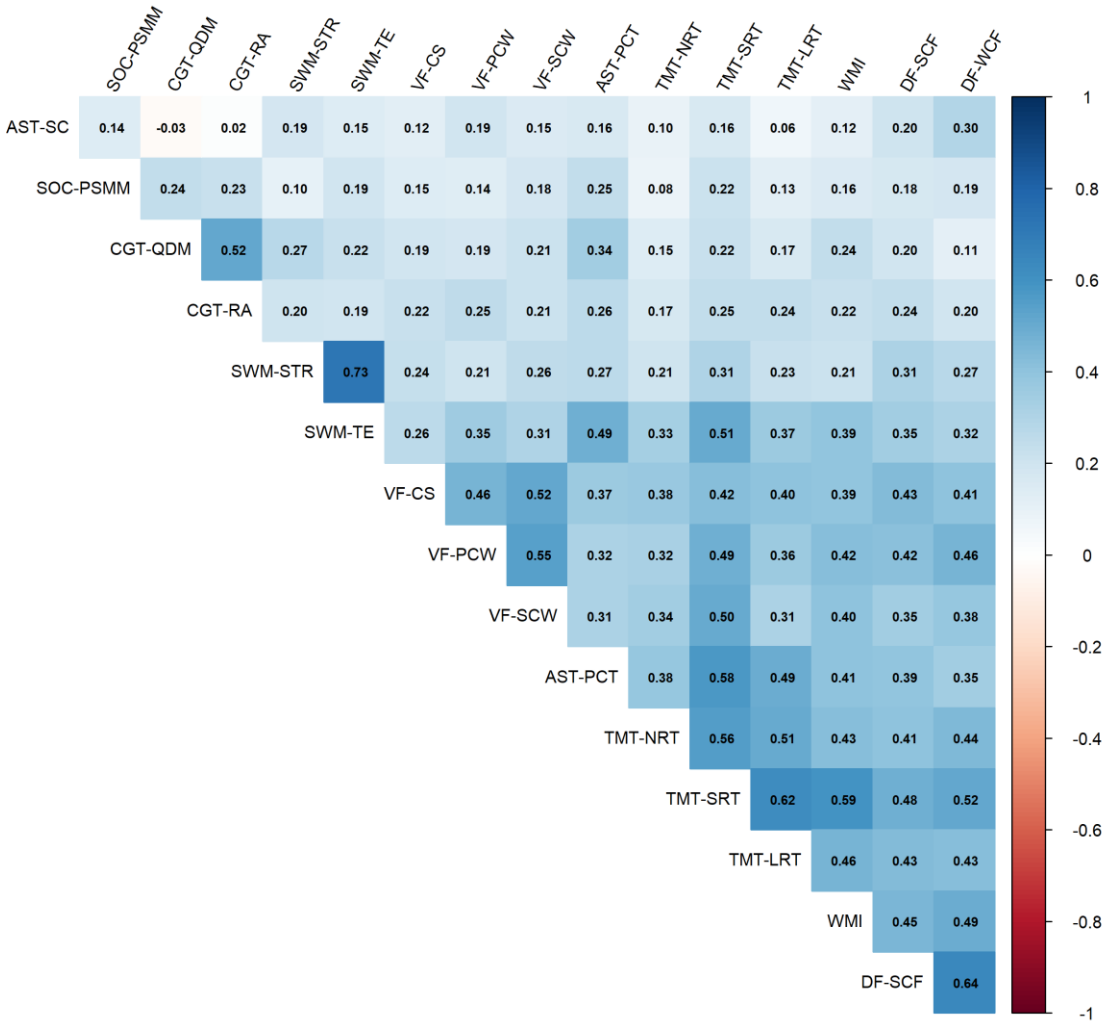
Table 6 Group differences in neurocognitive test performance

	OCD Group $n = 110$		Control Group $n = 83$			
Domain/task/outcome	**Mean**	**SD**	**Mean**	**SD**	p^a	η_p^2
Cognitive Flexibility						
AST[b] Percent Correct Trials	91.62	8.39	93.86	5.13	.033	.024
AST[b] Switch Cost	268.37	145.08	310.17	136.40	.043	.021
TMT[c] Switching, Time in Seconds	96.44	47.07	82.71	32.86	.024	.026
Verbal Fluency Switching[b], Correct Switches	11.63	3.01	11.11	2.86	.227	.008
Design Fluency Switching[b], Correct Figures	6.96	3.10	7.25	2.75	.487	.003
Planning and Decision-making						
SOC[b] Problems Solved in Minimum Moves	8.10	2.16	7.64	2.93	.209	.008
SWM[b] Strategy	31.83	6.23	31.06	5.44	.372	.004
CGT[b] Quality of Decision Making	.90	.11	.95	.06	<.001	.070
CGT[b] Risk Adjustment	1.13	.97	1.23	.75	.465	.003
Working Memory						
Wechsler Working Memory Index[d]	98.96	12.95	106.10	12.16	<.001	.073
SWM[b] Total Errors	24.97	17.41	22.49	16.53	.318	.005
Fluency						
Verbal Fluency Phonemic[c], Correct Words	27.70	10.12	25.81	9.32	.185	.009
Verbal Fluency Semantic[c], Correct Words	38.41	10.62	37.80	8.82	.670	.001
Design Fluency[c], Correct Figures	9.67	3.16	9.10	2.85	.746	.001
Processing Speed						
TMT Number-Sequencing[c], Time in Seconds	37.91	18.44	31.21	13.24	.005	.040
TMT Letter-Sequencing[c], Time in Seconds	39.90	18.40	34.55	19.50	.053	.019

Note: The table is similar to the one presented in Study 1 (Uhre et al., 2023).
[a]*p*-values reported here are from the MANOVA model, only those at $p < .003$ (0.05/16) should be considered significant if examined as individual tests.
[b]Tests from the CANTAB battery (Cambridge Cognition, 2019). SWM, Spatial Working Memory; AST, Attention Switching Task; SOC, Stockings of Cambridge; CGT, Cambridge Gambling Task
[c]Tests from the D-KEFS battery (Delis et al., 2001b, 2001a). TMT, Trail Making Task
[d]Tests from the Wechsler Scales (Wechsler, 2008, 2014)

As outlined in the statistical analysis section, we anticipated neurocognitive outcomes would be interrelated, justifying the use of MANOVA. While these correlations were not reported in the published Study 1 (Uhre et al., 2023), they are presented here post hoc for the dissertation. We used z-scores for interpretability (higher score means better performance). Baseline neurocognitive z-scores were positively correlated across all 16 EF measures, with mean correlations between r = 0.19 and r = 0.46 (Figure 6). These moderate positive associations indicate that performance across domains is related, supporting the use of MANOVA to assess overall group differences while controlling the Type 1 error rate.

Figure 6 Correlations among baseline neurocognitive *z*-scores in all participants

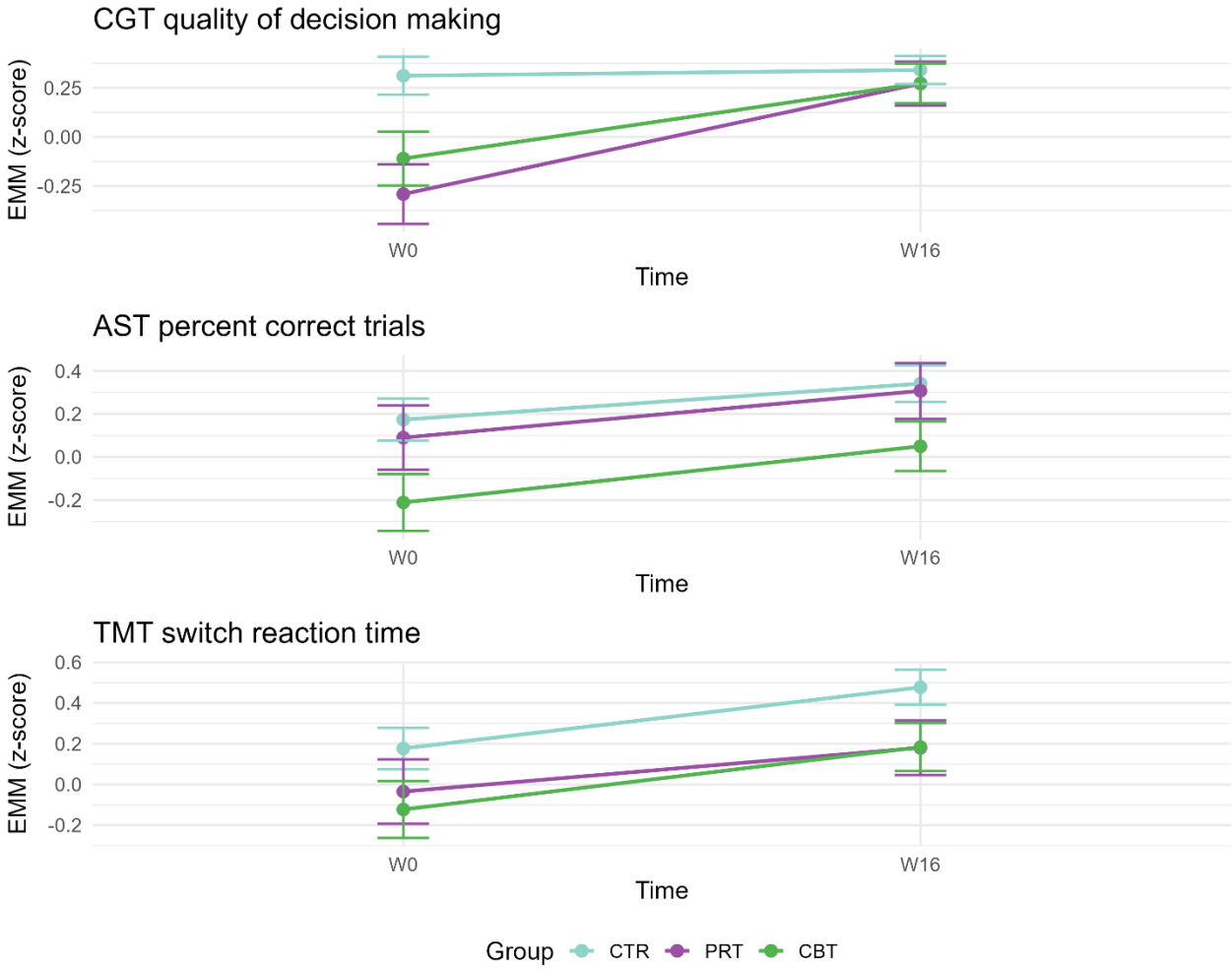


Note: Blue tones represent positive correlations, with darker shades corresponding to values nearer +1. Red tones represent negative correlations, with darker shades corresponding to values nearer -1.

Study 2: The neurocognitive follow-up study

Hypothesis 2a: EF improved significantly across groups for all three primary outcomes (Figure 7) ($p < .001$, ges = .036; $p < .001$, ges = .014; $p < .001$, ges = .022). For CGT-QDM, there was a significant Group × Time ($p = .002$, ges = .020) interaction, reflecting significant improvement in patients (whether CBT or PRT) but not controls. AST-PCT and TMT-SRT improved equally across groups. There was no differential effect of treatment group on EF change, i.e. path *a* in the mediation model (Figure 7) was non-significant. Supplementary analyses restricted to completers (i.e., per protocol) showed similar results for effects of Time ($p <.001$; $p < .001$; $p < .001$) and Group × Time ($p = .003$; $p = .717$; $p = .624$) on the three primary outcomes.

Figure 7 Change in primary neurocognitive outcomes



Note: The figure is similar to the one presented in Study 2. CGT = Cambridge Gambling Task (from the CANTAB battery). AST = Attention switching task (from the CANTAB battery). TMT = Trail making task (from the D-KEFS battery). EMM = Estimated marginal mean. CTR = Control children. CBT = Patients who received cognitive-behavioral therapy (CBT). PRT = Patients who received psychoeducation and relaxation training (PRT). W0 = Week 0; baseline. W16 = Week 16; follow-up (end-of-treatment for patients with OCD).

For EF change in explorative outcomes, only processing speed displayed a significant Group × Time interaction, mirroring more improvement in patients (across treatments) than controls (p = .027). Supplementary analyses showed that overall group differences reported in the original baseline sample (Uhre et al., 2023) persisted in the current smaller follow-up sample at baseline ($F(16,140) = 3.27$, $p < .001$), also when correcting for IQ ($F(16,139) = 3.25$, $p < .001$).

Hypothesis 2b: Neurocognitive improvement did not correlate with OCD symptom improvement, i.e., path *b* in the mediation model (Figure 7) was non-significant.

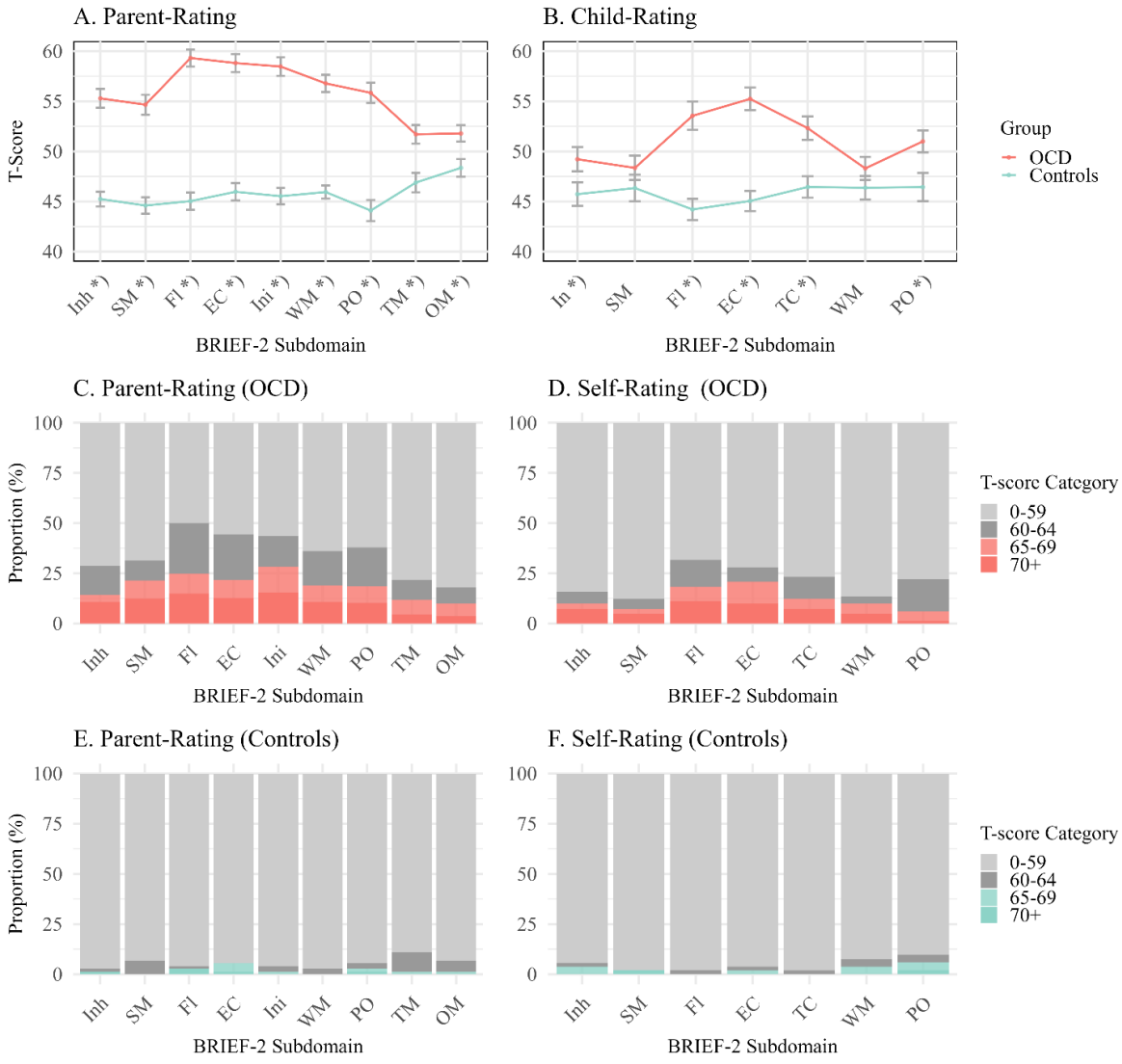
Hypothesis 2c+d: None of the primary outcomes significantly predicted or moderated treatment response in patients. Supplementary analyses restricted to completers (i.e., per protocol) showed similar results.

Study 3: The BRIEF-2 study

Hypothesis 3a: Both parent- and self-reported results showed more EF difficulties in OCD patients versus controls before treatment (p <.001, η_p^2 = 0.50; p <.001, η_p^2 = 0.31). All subdomains differed for parent-report, and all but two for self-report (Figure 8). Both patients and their parents identified most difficulties in flexibility and emotional control. Although group means were within the normal range (T < 60), many patients – but not controls – rated clinically elevated EF difficulties.

Hypothesis 3b: Self-reported flexibility difficulties were associated with higher symptom severity (p = .001, R^2 = .201), while no other domains were.

Figure 8 BRIEF-2 domains at baseline

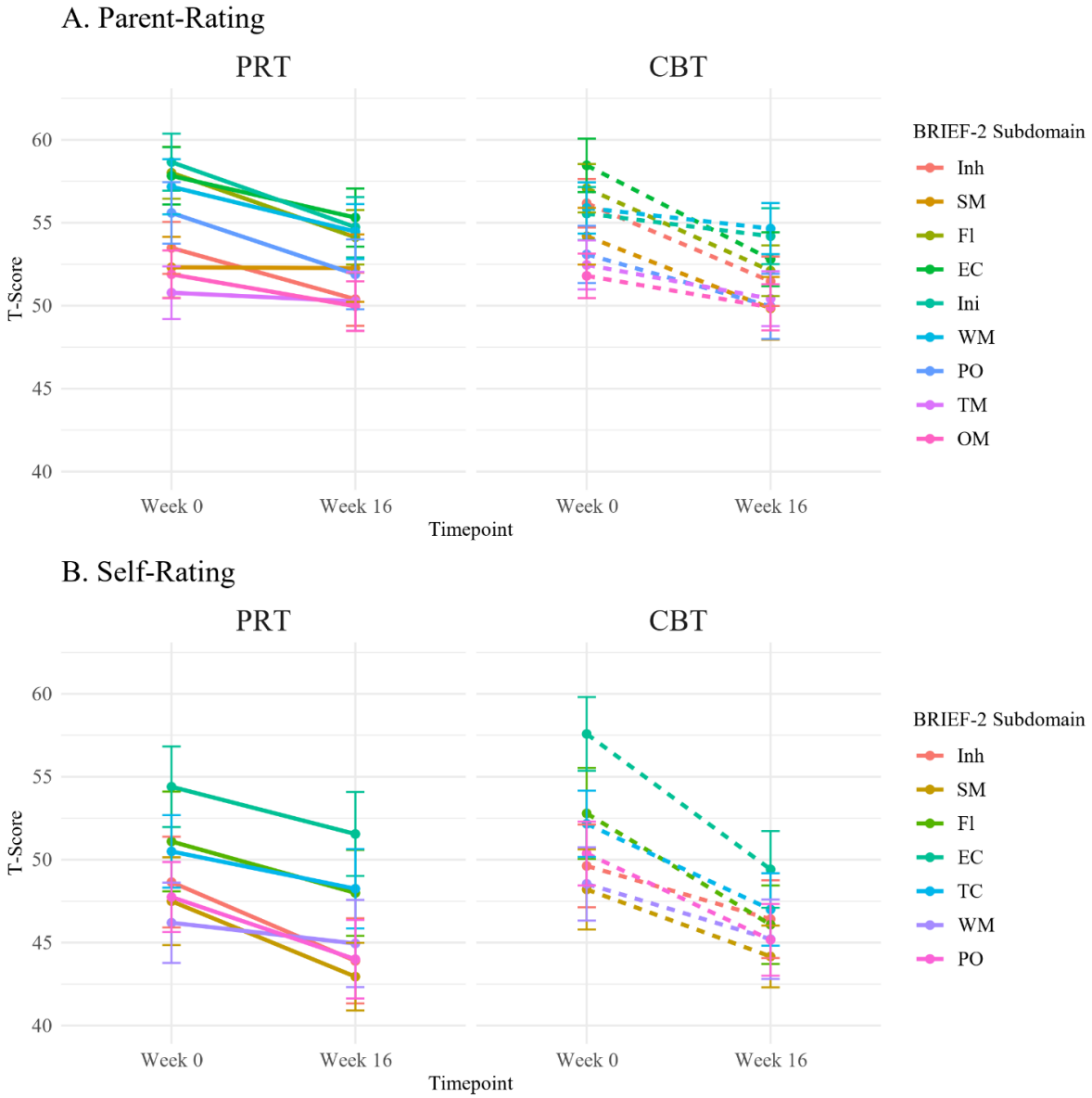


Note: The figure is similar to the one presented in Study 3. BRIEF-2 = Behavior Rating Inventory of Executive Function, Second Edition (Gioia et al., 2015). Inh = Inhibition, SM = Self-Monitoring, Fl = Flexibility/Shift, EC = Emotional Control, Ini = Initiating, WM = Working Memory, PO = Planning/Organizing, TM = Task-Monitoring, OM = Organization of Materials, TC = Task-Completion.

Hypothesis 3c: For OCD patients, parent- and self-reported results improved significantly over Time (p <0.001; $\eta_p^2 = 0.27$; p <0.001; $\eta_p^2 = 0.34$) (Figure 9), while EF remained stable in the control group. For parent-reported self-monitoring in patients, a significant Treatment × Time interaction emerged (p = .010), driven by improvement in CBT (p < .001) but not PRT (p = .971). For all other domains, change in patient performance occurred regardless of treatment.

Hypothesis 3d: Patient improvement was not associated with OCD symptom reduction for any subdomain.

Figure 9 BRIEF-2 domains at baseline and follow-up by Treatment Group in patients with OCD

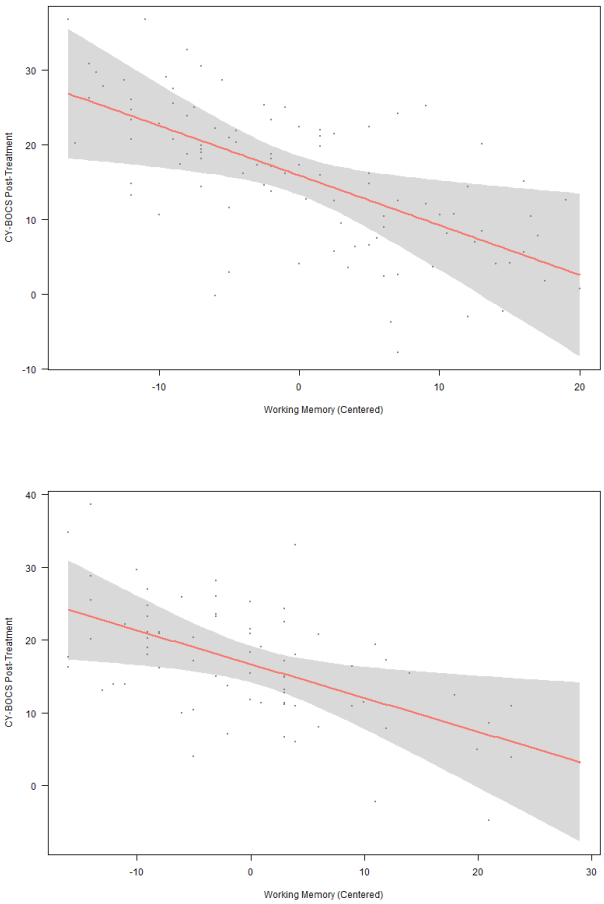


Note: The figure is similar to the one presented in Study 3. PRT = Patients who received psychoeducation and relaxation training (PRT). CBT = Patients who received cognitive-behavioral therapy (CBT). BRIEF-2 = Behavior Rating Inventory of Executive Function, Second Edition (Gioia et al., 2015). Inh = Inhibition, SM = Self-Monitoring, Fl = Flexibility, EC = Emotional Control, Ini = Initiating, WM = Working Memory, PO = Planning/Organizing, TM = Task-Monitoring, OM = Organization of Materials, TC = Task-Completion.

Hypothesis 3e+f: EF ratings predicted and moderated treatment response in several ways. Note that prediction includes the effect of EF across treatments (whether CBT or PRT) and moderation includes the differential association of EF with CBT versus PRT.

In OCD patients, for both parent- and self-ratings, working memory predicted treatment response (p = .002; p = .003) so that more difficulties at baseline was associated with less symptoms after treatment (whether CBT or PRT) (Figure 10). Additionally, more self-rated task-completion difficulties was associated with less benefit from both treatments (p = .003) and less benefit from CBT versus PRT.

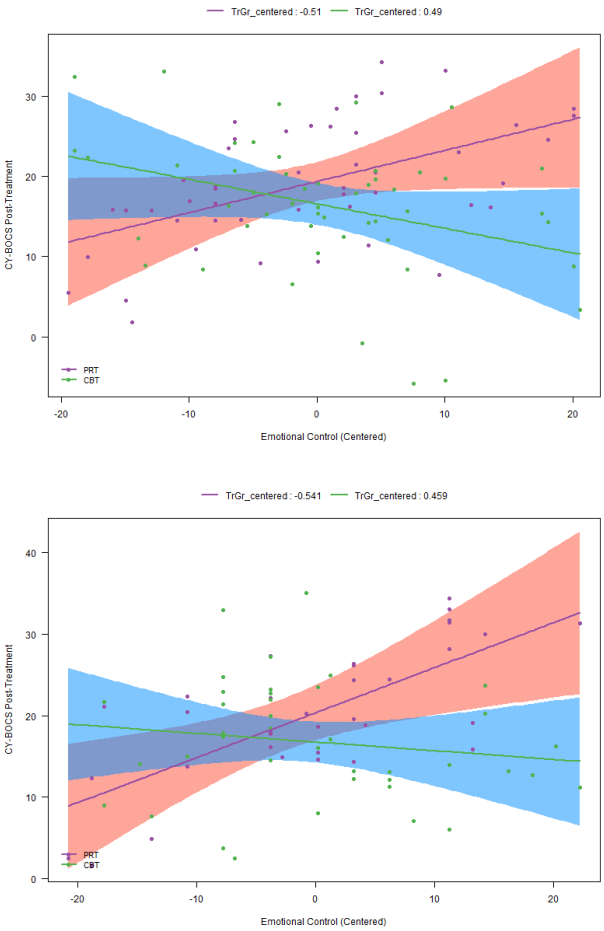
Figure 10 Working memory as a unique predictor in the multiple linear regression model



Note: The figures are similar to the one presented in Study 3, supplementary material. Top: Parent-Report; child age: 8-17, Bottom: Self-Report; child age 11-17.

For both parent- and self-report, emotional control in patients significantly moderated treatment response (p = .014; p = .011) so that more baseline difficulties was associated with less severe symptoms following CBT versus PRT (Figure 11). Additionally, more self-rated task-completion difficulties at baseline was associated with less benefit from CBT versus PRT (p = .044).

Figure 11 Emotional control as a moderator in the multiple linear regression model



Note: The figures are similar to the one presented in Study 3, supplementary material.´ Top: Parent-Report; child age: 8-17. Bottom: Self-Report; child age 11-17.

DISCUSSION

Overview and integration of key findings

The overarching aim of this dissertation was to study the neurocognitive correlates of OCD in children and adolescents and its relevance for psychotherapeutic treatment response. We based our hypotheses upon the CSTC models of OCD which anticipate a direct link between OCD symptomatology and cognitive functioning; according to these models, aberrant CSTC activation induces impairment in cognitive flexibility as well as other related EF and cognitive functions, leading to the inflexible, repetitive thinking and behavior that is seen in OCD (Milad & Rauch, 2012; van den Heuvel et al., 2016; Gillan & Robbins, 2014; Jalal et al., 2023; Robbins et al., 2024). Based on the CSTC models of OCD, EF is suggested a predictor of treatment response (i.e., symptom improvement) and a treatment outcome (Chamberlain et al., 2005; Fitzgerald et al., 2021; Norman L.J. et al., 2021; Chamberlain et al., 2021), and following this, we evaluated EF as a potential moderator and/or mediator of CBT-induced symptom improvement in three studies.

We included a hospital-based clinical sample of children and adolescent aged 8-17 years with OCD of moderate to marked severity. Patients with comorbid mental retardation or severe psychiatric or neurodevelopmental disorders were excluded, while patients with other psychiatric comorbidities were included. Patients had currently and the prior last 6 months not received SSRIs or antipsychotic medication and had not been treated with CBT or PRT. After baseline assessments, patients were randomized to 14 weekly sessions of either CBT or PRT. We included a control group of individuals that were free of prior or current mental disorders and were matched to the patient sample on sex and age. The sample size varied between studies depending on data availability from 69 to 119 patients and from 67 to 90 controls.

Neurocognitive test performance as well as parent- and self-rated EF behavior was assessed before and after psychological treatment and studied in relation to OCD symptom severity in three related studies. As a group, pediatric OCD patients showed small, selective difficulties in neurocognitive test performance when compared to the control group, and only decision-making (as hypothesized) and processing speed (investigated exploratively) improved after psychological treatment. Moreover, children with OCD and their parents reported moderate to large, widespread EF difficulties – some of which were predictive of treatment response – which improved after psychotherapy. On an individual level, more self-rated flexibility difficulties was associated with more severe OCD symptoms, while remaining rating-based and neurocognitive outcomes were not associated with symptom severity.

Altogether, evidence across test performance and rating-based measures produced mixed results. Neurocognitive results did not support the notion of a robust, disorder-wide EF impairment in pediatric OCD that influences or is influenced by psychotherapy. Behavior-ratings, however, indicate more widespread clinically significant daily-life EF difficulties in a substantial subgroup; most improved with treatment, and some were predictive of treatment response.

Is pediatric OCD associated with EF difficulties?

Our initial hypotheses stated that: "OCD patients would show poorer performance than control participants on tests of EF (Hypothesis 1a & Hypothesis 3a)" and "Poorer performance would be associated with higher OCD symptom severity" (Hypothesis 1b & Hypothesis 3b).

Neurocognitive performance on EF- and related tasks was assessed in 119 children with OCD and 90 healthy controls pre-treatment, i.e., at baseline (Study 1). Medium-effect difficulties in patients were identified in decision-making and processing speed, and small-effect difficulties were seen in cognitive flexibility and working memory – none were associated with OCD symptom severity. Everyday EF was parent- and self-rated with the BRIEF-2 in 112 OCD patients and 74 controls (Study 3). In patients, parents identified large-effect difficulties in all BRIEF-2 subdomains with the largest effects seen for emotional control and flexibility. Children with OCD self-reported large-effect difficulties in emotional control and flexibility and small to medium effect difficulties in inhibition, task-completion, and planning. More self-reported flexibility difficulties were associated with worse OCD symptom severity.

Altogether, widespread EF difficulties were reported in everyday life of children with OCD by their parents and – to a smaller extent – the children themselves, while neurocognitive test results only identified more subtle and selective difficulties. We found only very limited evidence for an association between OCD symptom severity and baseline EF.

Clinical significance of our findings

First, it is important to consider whether our findings reflect clinically meaningful difficulties in pediatric OCD. Statistically significant group differences alone do not establish clinical relevance, so additional context is needed. Individual clinical impairment is generally defined as ≥ 2 SD below the normative mean in neurocognitive outcomes (Abramovitch et al., 2015). In our sample, decision-making was the only domain that met this threshold at the OCD group-level, and only a small number of individual participants with OCD met the threshold individually on the remaining tasks. Thus, it can be questioned whether the difficulties found in processing speed and cognitive

flexibility are clinically relevant even in a subgroup of patients. For the BRIEF-2 results, the available T-scores allowed comparison with normative data. The BRIEF-2 defines clinically concerning scores as T-scores > 60 ("potentially elevated"), > 65 ("slightly elevated"), and > 70 ("clinically elevated") (Gioia et al., 2018). Although group-level scores were all below 60, indicating no overall clinical impairment, a substantial subset of children with OCD showed clinically elevated scores, highlighting meaningful individual-level difficulties not captured by group means, e.g., flexibility-scores above 60 were reported by 50.0 % of parents and 31.7 % and children.

Cognitive flexibility

Concerning the association between neurocognitive performance and OCD symptom severity, we hypothesized that: "Poorer performance would be associated with higher OCD symptom severity". Cognitive flexibility deficits are suggested to be markers of OCD symptoms, following the hypothesis of excessive habit-formation subserved by imbalance in CSTC-circuits (Jalal et al., 2023). Poorer performance on flexibility tasks has been consistently identified in adults with OCD (Abramovitch et al., 2013; Bora, 2020; Chamberlain et al., 2021; Shin et al., 2014; Snyder et al., 2015), while only subtle impairment is seen in children (Abramovitch et al., 2013, 2021; Marzuki et al., 2020, 2021). In our OCD sample, we observed mild impairment in cognitive flexibility on the AST-PCT and TMT-SRT. By contrast, no differences were observed on three other measures of cognitive flexibility: AST (switch cost), VF (switching, time), and DF (switching, correct figures). The AST-PCT requires switching to a different rule to respond correctly, but performance may also be influenced by processing speed, working memory, and sustained attention. In comparison, AST switch cost may be considered a purer measure of flexibility because it isolates the additional difficulty specifically associated with switching by calculating the difference between switch and repeat trials. However, patients showed intact (even superior) performance on this measure. As for TMT-SRT, this flexibility measure may also be influenced by the impaired processing speed observed in our OCD sample. Thus, it is questionable whether our results provide strong evidence for impaired cognitive flexibility on neurocognitive tasks. Consistent with previous findings, we suggest that difficulties on cognitive flexibility tasks in pediatric OCD are task-dependent, relatively subtle, and much less pronounced than in adults with OCD.

Conversely, when measuring flexibility with the BRIEF-2, both parents and children reported moderate flexibility difficulties. Parent-rated baseline difficulties replicated previous findings (Negreiros et al., 2020; Rydqvist et al., 2023), whereas the self-reported difficulties represent novel contributions to the literature. Together, these patterns suggest that findings of subjective (child-

rated) and observed (parent-rated) difficulties with flexibility are more robust and clinically salient in pediatric OCD than task-based impairments. This may reflect that rating-based results capture aspects of everyday functioning not adequately reflected in neurocognitive measures, but it is also possible that the neurocognitive tasks we used were not sensitive enough.

Decision-making and emotional control

Decision-making difficulties in pediatric OCD have previously most consistently been observed on tasks involving ambiguous information (i.e., uncertainty), reward-processing (i.e., "hot EF") (Zelazo & Carlson, 2012), and implicit learning – see Marzuki et al. (2020) for a review. Two pediatric studies report poorer performance on the Iowa Gambling Task, which requires implicit learning and involves non-explicit probabilities and reward/punishment (Kodaira et al., 2012; Norman et al., 2018). Additional evidence for intolerance of uncertainty comes from the Information Sampling Task and the Dot Discrimination Task (Erhan et al., 2017), and implicit learning deficits have been identified in two further studies (Vloet et al., 2010; Marzuki et al., 2021). Importantly, the CGT does not rely on learning, and all probabilities are explicit. Thus, our findings demonstrate altered decision-making in pediatric OCD independent of learning demands, reflecting a more specific difficulty in using explicit outcome probabilities to guide behavior (i.e., reducing task impurity). Notably, although the CGT-QDM scores were previously reported as normal in a pediatric OCD sample (Hybel et al., 2017), our results challenge this conclusion and re-open the question of whether decision-making is affected in this population.

Although the BRIEF-2 does not directly measure decision-making and therefore cannot clarify whether our participants struggle with decision-making in everyday contexts, it does assess components of "hot" EF via the emotional control subscale. Both parents and children reported moderate difficulties in emotional control, mirroring previous findings (Rydqvist et al., 2023). Also, another study from the TECTO trial identified more parent- and self-reported emotion regulation difficulties (ED) on the Difficulties in Emotion Regulation Scale (DERS) and more behavioral ED during a frustration task in OCD versus controls (Thoustrup, Blair, et al., 2025). Taken together with the CGT-QDM findings, this suggests that "hot" EF difficulties in pediatric OCD are evident both when measured with behavioral tasks and questionnaires assessing everyday emotional regulation, albeit captured differently across measures.

Associations with OCD symptom severity

Although group-level difference emerged on task-performance measures of cognitive flexibility, decision-making, and processing speed, individual-level analyses revealed no significant

associations between task performance and OCD symptom severity. In relation to CSTC models of OCD, which posit that EF deficits contribute to OCD symptom expression, these findings may suggest that such cognitive difficulties are characteristic of OCD at the group level but do not vary systematically with symptom severity.

The same pattern was observed for the BRIEF-2 subscales, except for self-reported flexibility. Our finding that greater self-reported flexibility difficulties is associated with more severe OCD symptoms is novel and noteworthy, but it requires replication in future studies before it can be considered robust.

Does EF predict and/or moderate treatment response in pediatric OCD?

Among the 16 neurocognitive measures examined, an explorative measure of cognitive flexibility (Design Fluency, switch condition) showed a nominal moderating effect on psychotherapy response, with better baseline performance favoring CBT over PRT; however, this finding did not remain significant after Bonferroni correction. The remaining outcomes did not predict nor moderate treatment outcome. A pediatric study identified better EF performance as a predictor of CBT non-response (Hybel et al., 2017); while this study examined EF as a latent variable, based on multiple performance-based outcomes, we wished to examine the potential effect of specific neurocognitive tasks on psychotherapy response. However, our results only provided very little support for such prediction or moderation.

On the other hand, the BRIEF-2 rating-based measure of EF did predict and moderate psychotherapy response in distinct ways.

BRIEF-2 Task-completion and working memory as significant predictors (whether CBT or PRT)

Our initial hypothesis stated that "Baseline EF would predict treatment response (i.e., more difficulties would be associated with less symptom reduction)" (Hypothesis 2c & Hypothesis 3e). This was confirmed for self-reported task-completion on the BRIEF-2, where more reported problems were associated with poorer treatment response across treatments. These results indicate that children who report problems with finishing tasks are less likely to benefit from CBT and PRT. One possible explanation is that children with such difficulties may struggle to engage fully in in-session exercises and complete homework assignments, which are integral components of these interventions.

Interestingly, for both parent- and self-rated working memory on the BRIEF-2, we identified a prediction in the opposite direction; greater baseline difficulties predicted stronger symptom improvement following CBT and PRT. Notably, working memory was not associated with symptom severity at baseline, suggesting that this effect cannot be explained by regression toward the mean. A similar pattern was reported for neurocognitive outcomes by Hybel et al. (2017), who observed stronger treatment gains in lower-performing children using a latent variable encompassing multiple cognitive measures. One possible explanation is that children with working memory deficits may respond particularly well to the highly structured, scaffolded format of manualized psychotherapy, which provides clear guidance, step-by-step exercises, and support for following through on tasks.

BRIEF-2 Task-completion and emotional control as significant moderators of CBT versus PRT

For analyses of a potential moderation effect (CBT versus PRT), our hypothesis stated: "Baseline EF would moderate treatment response (i.e., less difficulties would be associated with greater symptom reduction for CBT versus PRT)" (Hypothesis 2d & Hypothesis 3f). This pattern was confirmed for self-reported task-completion on the BRIEF-2, where greater difficulties were associated with less symptom reduction in CBT versus PRT. These results suggest that self-rated task-completion difficulties may be an important indicator of a child who requires extra support when entering CBT.

Finally, for both parent- and self-rated emotional control, we observed a differential treatment effect, but in the opposite direction to our hypothesis: children with greater difficulties in emotional control were likely to benefit more from CBT than from PRT. This suggests that CBT may be particularly effective for children who struggle with emotional control, at least as measured by parent- and self-report. Interestingly, a similar pattern has been observed in another TECTO study, which assessed emotion regulation using the self-reported DERS total score (Thoustrup, Olsen, et al., 2025). One possible explanation is that CBT is well suited for addressing challenges in "hot" EF – that is, EF in emotionally significant situations involving motivation and reward processing (Zelazo & Carlson, 2012) – by providing structured strategies to regulate emotions, manage frustration, and respond adaptively in challenging contexts. These strategies may be particularly beneficial for children with greater difficulties in emotional control, helping to account for the larger treatment gains observed in CBT.

Does EF change during psychological treatment for pediatric OCD?

We studied whether EF improved during psychotherapy and anticipated that: "EF would improve over the course of treatment relative to controls, with greater improvements following CBT than PRT" (Hypothesis 2a & Hypothesis 3c) and "Improvements after treatment would correlate with OCD symptom reduction" (Hypothesis 2b & Hypothesis 3d).

Improvement in decision-making (hypothesized) and processing speed (explorative)

For neurocognitive outcomes, patients demonstrated significant improvement in decision-making (CGT-QDM), relative to controls assessed in a similar time interval. Notably, CGT-QDM was the only pre-selected outcome on which patients showed clinically meaningful poorer baseline performance relative to controls. For the remaining two primary outcomes measuring flexibility, patients did not improve relative to controls. While previous studies have reported improvement in other EF domains such as visual memory and planning (Andrés et al., 2008; Huyser et al., 2010; van der Straten et al., 2018), our study is the first to demonstrate decision-making improvement in pediatric OCD. The CGT-QDM evaluates the percentage of trials in which participants selects the option with the highest probability of success, reflecting their capacity to make decisions informed by outcome probabilities. In the context of OCD treatment, improvements on this measure may indicate that patients have trained their decision-making skills, which could contribute to greater control over obsessions and compulsions.

Among the 13 explorative neurocognitive outcomes, only processing speed (TMT letter sequencing RT) improved relative to controls. This result may suggest that treatment (whether CBT or PRT) helped participants process information more quickly, possibly because their overall mental and physical slowing (psychomotor retardation) improved. This result is novel in children, but has been observed in adults with OCD (Voderholzer et al., 2013).

Improvement in BRIEF-2 subscales

For parent- and self-reported EF, we identified widespread EF improvement across most BRIEF-2 subscales. The control group, as we expected, did not change during the 16 weeks assessment period.

Our results align with previous reports of parent-reported improvements on the BRIEF GEC (Hybel et al., 2017) and additionally adds several new insights. First, by examining the individual BRIEF-2 subdomains, we showed that most EF components captured by this instrument improved with psychotherapy. Second, these gains were evident in both parent- and self-ratings. Third, parents reported uneven improvements across domains, with the most pronounced changes in

flexibility, emotional control, inhibition, and planning – patterns that were also reflected in the self-report data, although with smaller effects.

Improvements in planning and emotional control align with prior neurocognitive findings (Huyser et al., 2010; van der Straten et al., 2018) and rating-based findings (Krebs et al., 2013; Thoustrup, Olsen, et al., 2025) in pediatric OCD. Also, improvements in inhibition and flexibility has been seen in adults with OCD (Katz et al., 2024; Kuelz et al., 2006; Voderholzer et al., 2013; Wheaton et al., 2025). Because deficits in flexibility and inhibition are considered central features of OCD and potential treatment targets (Chamberlain et al., 2021; Jalal et al., 2023), we expected CBT – particularly its emphasis on inhibiting compulsions and practicing alternative responses – to drive such and related EF improvements. However, comparable effects were observed for PRT and CBT, suggesting that shared therapeutic mechanisms account for these changes. Moreover, while prior work showed CBT-specific gains in child-rated emotion regulation on the DERS, our results indicate no differential effect of CBT versus PRT. This discrepancy may relate to measurement differences: the BRIEF-2 emphasizes observable behavioral reactions, whereas the DERS captures broader psychological processes (e.g., emotional awareness and acceptance), potentially making it more sensitive to CBT-specific effects.

Finally, we found that parent-reported self-monitoring improved significantly after CBT but not PRT. The self-monitoring domain captures how well the child evaluates its own behavior and its impact on others (Gioia et al., 2018). It is possible that CBT influences this domain more because ERP emphasizes awareness and adjustment of thoughts and actions. However, given the explorative nature of these analyses, this interpretation should be viewed cautiously.

Clinical significance of the improvement

It should be noted that improvement of decision-making and processing speed relative to controls as well as improvement of BRIEF-2 subdomains only showed a small effect. Improvement was smaller than 1 SD for all outcomes. Thus, it is debatable whether the improvement would be considered clinically meaningful. However, while this conclusion in based on group means, when inspecting subgroups of patients, it becomes clear that the proportion of patients with T-scores in clinically elevated categories had decreased after treatment.

No association between EF improvement and OCD symptom improvement

Interestingly, none of the neurocognitive or rating-based EF outcomes improving over the course of treatment showed a significant correlation with changes in OCD symptom severity. Thus, while some of these deficits appear to be modifiable and state-like manifestations of OCD, they may

reflect treatment targets distinct from clinical symptom reduction. This challenges the CSTC models of OCD where EF impairment is thought to underlie symptoms and mediate treatment effect.

No differential EF change for CBT versus PRT

We hypothesized that building skills for active thought- and behavior modification in CBT would result in more reduced EF difficulties for CBT versus PRT. However, since EF improvement was not differential between CBT and PRT (with parent-rated self-monitoring as the exception), we anticipate the effect derives from non-specific therapy processes shared between these two treatments. Such processes may include externalizing OCD (i.e., cognitive restructuring), setting treatment goals, and planning how to fulfill these (i.e., active skill building). Also, it is possible that cognitive control is involved in suppressing obsessions and compulsions in relaxation exercises as well as ERP exercises.

Methodological considerations

This section discusses several strengths and limitations of the methods employed in the studies. By reflecting critically on our methodological choices, it is acknowledged that certain aspects of the design and implementation may have influenced the results and interpretations.

Several strengths should be stressed. Across studies, sample characteristics were carefully considered. The patient groups were non-medicated and had not received psychotherapy for at least six months prior to participation, thus reducing potential confounding treatment effects. In particular, patients did not receive medication for OCD; as SSRI treatment has been associated with structural brain changes and neurocognitive performance in OCD (Bruin et al., 2020; Sip et al., 2018), this is a major strength. The patient samples also included a broad range of comorbidities, reflecting real-world clinical population. Importantly, the presence of comorbidity did not account solely for observed neurocognitive difficulties, supporting the view that at least some degree of these difficulties is linked to pediatric OCD rather than secondary pathology. In addition, the inclusion of an age- and sex-matched control group accommodated the potential issues of examining a broad age range (8-17) where EF are known to change. Furthermore, the controls were thoroughly and systematically screened with K-SADS to ensure absence of prior and current psychiatric disorders.

The study designs further strengthened the conclusions. Longitudinal assessments, including repeated measures in both patients and matched non-psychiatric controls, allowed for the isolation of OCD-specific and therapy-related effects. In particular, the use of two parallel treatment groups in a randomized design enabled the separation of specific and non-specific treatment factors. Moreover, the longitudinal assessment of non-psychiatric controls enabled the reduction or elimination of test-retest effects.

The studies also benefitted from a comprehensive approach to measuring EF. We included a large battery of well-established neurocognitive tasks, providing detailed information about specific EF components under controlled conditions, carried out by trained clinicians under supervision. To complement these task-based measures, we incorporated the BRIEF-2 and a multiple-informant approach, enabling assessment of EF as it manifests in daily life rather than only in structured testing environments. Including rating-based subscales further allowed comparison with existing neuropsychological literature, which typically focuses on specific EF domains (e.g., flexibility) rather than global function. Together, these features offered a comprehensive picture of EF in pediatric OCD, integrating insights from both structured neurocognitive tasks and daily-life assessments.

At the same time, the studies are subject to several limitations that may affect the interpretation of the findings. First, despite employing a broad and well-established neurocognitive battery, the inherent constraints of task-based EF assessment must be acknowledged. The task-impurity problem may have confounded our aim to measure specific EF components, as performance on one task typically engages multiple EF. Moreover, ceiling effects in the control group also pose challenges for interpreting the neurocognitive findings on CGT-QDM. Additionally, ecological validity may have been limited in the highly structured test situations which may not be very demanding on EF compared to unstructured daily-life activities. Indeed, children with significant EF difficulties in daily life may perform unexpectedly well in quiet, supportive test settings (Diamond, 2020). This discrepancy raises the possibility that the neurocognitive measures lacked sensitivity to real-world EF challenges, and it is possible that especially subtle impairments may have gone undetected.

While we aimed to improve ecological validity of our results by including the BRIEF-2, rating-based EF measures also come with limitations. Although multiple informants were included to reduce rater bias, subjective reports can be influenced by expectations, insight, or general impressions of functioning rather than specific EF processes. It remains debated to what extent rating scales reflect neurocognitive constructs rooted in CSTC circuits versus broader behavioral

or emotional difficulties. However, it strengthens our conclusions that both parents and children identified difficulties that were most pronounced in flexibility and emotional control.

Notably, the developmental range of participants introduces complexity. Executive functions undergo substantial maturation from childhood into late adolescence, and even with age- and sex-matched controls, developmental heterogeneity may have influenced both neurocognitive and rating-based outcomes. Additionally, the control group was exceptionally healthy and well-functioning, which may have amplified group differences.

Although the CY-BOCS total score is the gold standard for assessing OCD severity, relying solely on this measure may have limited our ability to capture aspects of clinical change that are potentially more closely related to EF. Including subscale scores (obsessions and compulsions) of the CY-BOCS or broader indicators such as the CGI-S, CGI-I, or CGAS might have provided additional nuance.

Another limitation relates to the validity of the OCD diagnosis and its boundaries with closely related disorders. Notably, OCD shares features with neurodevelopmental disorders associated with EF difficulties (Sadozai et al., 2024) and OC-related disorders (Jalal et al., 2023). Also, CSTC and EF impairment may be specific to compulsive behavior which is also seen in several other psychiatric diseases (Robbins et al., 2024). While structured diagnostic procedures were employed, it remains possible that subtle or emerging comorbidities influenced EF performance. Relatedly, the high rate of psychiatric comorbidity in the patient samples – though clinically typical – complicates efforts to isolate EF alterations that are specific to OCD. Sensitivity analyses suggested robustness, but the possibility that comorbid symptoms contributed to observed patterns cannot be fully excluded.

Finally, attrition in TECTO – and consequently in the present studies – is likely to affect both statistical power and the generalizability of our findings. First, the reduction in sample size limits our ability to detect subtle effects. Second, participant drop-out introduces potential bias. Although TECTO was conducted as an RCT following the intention-to-treat principle, the treatment sub studies rely on complete-case analyses, which may result in selection bias. In Study 2, treatment completion rates are higher in our sample (91.8 % in CBT and 85.4 % in PRT) than in the full TECTO sample (81 % in CBT and 64 % in PRT). Also, remission rates are also slightly higher (24.5 % CBT; 17.1 % PRT) compared with the overall TECTO sample (21.9 % CBT; 12.1 % PRT). This pattern emphasizes that participants who complete treatment tend to benefit more and indicates that our samples may overrepresent patients with better treatment outcomes while underrepresenting those with limited or no treatment response. Moreover, attrition is greater in PRT than in CBT, and the primary reason for non-completion of PRT is lack of perceived treatment

effect (Pagsberg et al., 2025). Consequently, PRT-completers may constitute a subgroup experiencing the largest treatment effects, potentially attenuating the observed differences between CBT and PRT. Taken together, these considerations suggest that our findings may be most applicable to children and adolescents who complete treatment and follow-up assessments and may not fully generalize to all children with OCD.

Implications and future directions

Despite the methodological limitations, the present findings may have relevance for clinical practice and future research on psychotherapy for childhood OCD. A key novel contribution of this work is the inclusion of child self-reported EF and a longitudinal controlled design.

Overall, the findings tentatively suggest that EF difficulties experienced in daily life may be more widespread and more responsive to psychotherapy than is typically captured by neurocognitive assessments. The limited correspondence between subjective reports and performance-based measures may reflect a measurement challenge and underscore the need for future studies to develop or refine neurocognitive tasks that better reflect everyday EF demands. Further research is also needed to clarify whether EF difficulties and their improvement are related to OCD symptomatology and are particularly sensitive to CBT-specific treatment processes. Including more participants and examining EF in relation to more fine-grained outcomes – such as CY-BOCS subscales – or functional outcomes may help address this question.

From a clinical perspective, it may be useful to attend to EF difficulties when planning and delivering treatment. In particular, children with self-reported task-completion difficulties may require additional support during psychotherapy, as they may struggle with core components such as homework assignments and exercises. Allowing flexibility in time demands, breaking tasks into smaller steps, and providing structured support may facilitate engagement. The findings further suggest that structured, manualized psychotherapy is be especially well suited for children with working memory difficulties and that CBT is especially effective in addressing emotional control difficulties, possibly reflecting a superior effect of ERP versus relaxation training in managing emotions.

Finally, future studies should seek to replicate the findings, including the novel self-rating results. In clinical use and future studies, practice effects and ceiling effects should be carefully considered when using EF tests as measures of change.

These future directions may help refine how EF is assessed in psychotherapy for childhood OCD, ultimately contributing to more informed and responsive treatment approaches alongside the many other factors shaping the lives of affected children, adolescents, and their families.

CONCLUSIONS

This PhD project was initiated with the long-term goal of identifying neurocognitive targets enabling optimization of psychotherapy for children and adolescents with OCD and extending its effectiveness to a broader range of patients. To pursue this, we investigated, whether EF deficits are present in children and adolescents with OCD, whether they change with CBT, and whether such deficits and changes track CBT-induced symptom improvement.

These questions were addressed across three studies comprising children and adolescents with OCD and non-psychiatric controls. Patients were drawn from the Danish RCT TECTO and were assessed before randomization to CBT or PRT and again after 14 sessions of psychotherapy, while controls were assessed across a comparable time interval. We assessed EF with neurocognitive tests and the parent- and child-reported BRIEF-2 measure of daily-life EF behavior.

Neurocognitive testing revealed small and selective difficulties in pediatric OCD that were unrelated to symptom severity (Study 1). Among these, decision-making (hypothesized) and processing speed (explorative) showed improvements during psychotherapy, regardless of treatment modality and unrelated to symptom improvement (Study 2). Parent- and child-report measures indicated moderate-to-large difficulties in OCD across EF domains, with a subgroup of patients exhibiting clinically elevated impairments and with self-reported difficulties in cognitive flexibility being positively associated with OCD symptom severity (Study 3). Most BRIEF-2 domains improved over therapy, however largely unrelated to treatment modality and symptom improvement. Child-reported task-monitoring difficulties predicted poorer treatment outcome, and children with emotional control difficulties responded better to CBT than to PRT.

Taken together, these findings suggest that while children with OCD experience widespread EF difficulties in daily life that improve with psychotherapy, only minor effects are detectable on neurocognitive tests. Psychotherapeutic interventions may contribute to improvements in EF, particularly in everyday functioning, but it remains unclear whether such improvements are related to OCD symptom reduction or whether they are particularly sensitive to CBT-specific treatment factors. Children with task-completion difficulties may need extra support during CBT, and one potential advantage specific to CBT may be that the treatment appears to succeed in addressing emotional control difficulties. Future studies should aim at replicating the novel self-report findings, developing neurocognitive assessments sensitive of daily life EF, and investigating EF in relation to other treatment outcomes than overall OCD symptom improvement. Alongside other influencing factors, such ongoing research would bring the field closer to informing more effective and individualized treatment for the many children and adolescents suffering from OCD.

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ORIGINAL CONTRIBUTION

Atypical neurocognitive functioning in children and adolescents with obsessive–compulsive disorder (OCD)

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Abstract

Atypical neurocognitive functioning has been found in adult patients with obsessive–compulsive disorder (OCD). However, little work has been done in children and adolescents with OCD. In this study, we investigated neurocognitive functioning in a large and representative sample of newly diagnosed children and adolescents with OCD compared to non-psychiatric controls. Children and adolescents with OCD ($n = 119$) and non-psychiatric controls ($n = 90$) underwent psychopathological assessment, intelligence testing, and a neurocognitive test battery spanning cognitive flexibility, planning and decision-making, working memory, fluency, and processing speed. The MANOVA main effect revealed that children and adolescents with OCD performed significantly worse than the control group ($p < .001$, $\eta_p^2 = 0.256$). Atypical patient performance was particularly found for indices of cognitive flexibility, decision-making, working memory, and processing speed. We found no evidence of differences in planning or fluency. Moreover, we found no significant associations between neurocognitive performance and OCD symptom severity or comorbidity status. Our results indicate that children and adolescents with OCD show selective atypical neurocognitive functioning. These difficulties do not appear to drive their OCD symptoms. However, they may contribute to lifespan difficulties and interfere with treatment efficacy, an objective of our research currently.

Keywords Cognition · Case–control studies · Child · Adolescent · Obsessive–compulsive disorder

Introduction

Obsessive–compulsive disorder (OCD) is a common psychiatric disorder, affecting 1–3% of children and adolescents worldwide [1–3]. Core symptoms include intrusive and distressing thoughts, urges, or mental images (i.e., obsessions) and repetitive behavioral or mental rituals (i.e., compulsions) [4]. However, determining specific neurocognitive deficits in individuals with OCD, particularly in children and adolescents, has proven difficult.

An extensive body of data indicates that adult patients with OCD present with neurocognitive deficits in response

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inhibition, flexibility, planning, working memory (WM), and error monitoring [5]. However, meta-analyses of these data report significant deficits with small-to-medium effect sizes in the domains of processing speed, non-verbal memory, and executive functions [6–8]. Moreover, considerable heterogeneity in results and inconsistency across studies makes it difficult to draw firm conclusions [9]. Indeed, a recent umbrella review of potential biological and neurocognitive biomarkers in adults with OCD reported that the only marker showing 'convincing' (Class I) differential power between OCD and non-psychiatric controls were deficits in visuospatial abilities [10].

Data on children and adolescents with OCD are considerably sparser. There are indications of neurocognitive difficulties [11], but systematic reviews and meta-analyses show smaller effect sizes in children and adolescents with OCD compared to those reported in adults with OCD [12, 13]. Moreover, children and adolescents seem to show a smaller range of deficits. To our knowledge, only one meta-analysis of behavioral neurocognitive outcomes in children and adolescents with OCD has been published [12]. The meta-analysis included 11 studies covering different neurocognitive domains and found no significant group differences for children and adolescents with OCD vs. non-psychiatric controls. The direction of effects was consistent with underperformance in the OCD groups relative to non-psychiatric controls, but no differences reached statistical significance and effect sizes were small to medium, ranging from 0.04 to 0.40 across domains. A recent systematic review (without meta-analysis) of a larger number of neurocognitive studies indicated that children and adolescents with OCD show difficulties in visual WM, planning, and decision-making under uncertainty (e.g., during implicit learning of rewards) [13]—though little evidence for deficits in domains such as response inhibition and reversal learning. Given potential differences in the neurocognitive difficulties between child and adult OCD, it is critical to determine effects in pediatric samples. Some even argue that child and adult OCD should be classified as distinct psychiatric disorders given the little developmental continuity from one subtype to the other [14, 15] and the differences in symptom presentation and pattern of comorbidity [16, 17].

Several concerns emerge from the current OCD literature in children and adolescents relating to small sample sizes (though this is less of a concern recently; e.g., [11, 18–21]), comorbidities, and use of medications for OCD while participating. Medication may impact neurocognitive performance, at least in adults with OCD (see, e.g., [22]) and has been associated with changes in brain anatomy in large-scale analyses of structural neuroimaging data from the ENIGMA-OCD consortium [23].

In this study, we investigated neurocognitive functioning in a large clinical sample of children and adolescents with moderate to severe OCD compared with non-psychiatric controls. Participants had not been treated for OCD before, and their comorbidities were well characterized. Based on the prior literature, we hypothesized that OCD patients would perform worse than non-psychiatric controls on the neurocognitive tests.

Methods

Participants

Participants with OCD and non-psychiatric controls were recruited between September 2018 and June 2022. All participants were part of a case–control study nested in a randomized controlled trial, which included neurocognitive assessment at baseline (pre-treatment) and follow-up (post-treatment; The TECTO trial [24], clinicaltrials.gov NCT03595098). This paper presents baseline results.

All participants with OCD were recruited from psychiatric outpatient units in the Capital Region of Denmark. Inclusion criteria were age 8–17 years, primary OCD diagnosis (International Classification of Diseases, 10th revision; ICD-10) [25], and symptom severity score ≥ 16 on the Children's Yale-Brown Obsessive–Compulsive Scale (CY-BOCS) [26]. We excluded children and adolescents with OCD if they had a full scale IQs < 70 [27, 28], had received within the past 6 months cognitive behavioral therapy (CBT), psychoeducation and relaxation therapy (PRT), antidepressants or antipsychotics, or had a diagnosis of schizophrenia, other psychotic disorder, mania, bipolar disorder, substance dependence disorder, or pervasive developmental disorder (except for Asperger's syndrome) [24]. The TECTO trial included 130 participants with OCD. Of these, baseline neurocognitive data was collected on 119. Ninety non-psychiatric controls were recruited from the regional population via a random age-matched sample of children and adolescents, drawn from the Danish Central Person Registry [29] (for details, see Supplemental Material). Non-psychiatric controls were excluded if they had ever met the diagnostic criteria for a psychiatric disorder as per study screening or fulfilled any of the exclusion criteria also used for participants with OCD (i.e., IQ < 70).

Procedures

Study procedures were approved by The Ethics Committee of Capital Region of Denmark (approval number: H-18010607) and The Knowledge Centre on Data Protection Compliance in The Capital Region of Denmark (VD-2018-263, I-Suite no.: 6502). Participants and their families gave informed consent prior to participation, adhering to the Declaration of Helsinki [30]. Diagnostic assessments and

neurocognitive tests were administered by trained investigators (for details, see Supplemental Material). All participants received a gift card for their research activities (value ~ 38 USD per session).

Clinical assessment

All participants were assessed for any present and/or prior psychopathology with the Kiddie Schedule for Affective Disorders and Schizophrenia—Present and Lifetime version (K-SADS-PL) [31]. OCD patients and non-psychiatric controls were screened by trained clinicians and/or master-level psychology students under supervision. OCD symptom severity in patients was assessed with CY-BOCS [26]. To evaluate presence and severity of co-occurring disorders, children and adolescents with OCD were assessed with additional tests when relevant (e.g., Autism Diagnostic Observation Schedule [32] for patients with suspected autism spectrum disorder, according to the K-SADS interview). Diagnoses were determined with a specialist in psychiatry in the outpatient clinic (for details, see Supplemental Material). Behavioral or emotional problems during testing were assessed with the Test Observation Form (TOF) [33].

Neurocognitive tests

The test battery consisted of tests from the Cambridge Neuropsychological Test Automated Battery (CANTAB) [34], the Delis–Kaplan Executive Function System (D-KEFS) [35, 36], and the age-appropriate version of the Wechsler Scales [27, 28]. Note that we used the validated Danish versions of D-KEFS and the Wechsler Scales [36–39], while the CANTAB tasks are considered to be language independent [40]. Intelligence was assessed with the General Ability Index (GAI) from the Wechsler Scales. The remaining test battery assessed cognitive flexibility, planning and decision-making, WM, verbal and non-verbal fluency, and processing speed (Table 1). Primary outcomes from each test were chosen prior to study initiation, based on comparability with previous studies and consensus in the author group on which outcomes best reflected each function. All neurocognitive tests were administered and scored by trained psychologists and psychology master's students under expert supervision. Standard administration and scoring procedures were followed.

Statistical analysis

Analyses were done in IBM SPSS Statistics version 25 [41].

Demographic and clinical characteristics of the groups were compared using independent-samples *t*-tests for numerical data (e.g., age) and Chi-square test for categorical data (sex). Following previous work [21], parental education in years was calculated as either the mean of both parents, or, if only one parent participated, that parent's value was used.

Neurocognitive test performance was compared across participants with OCD and non-psychiatric controls initially, using a multivariate analysis of variance (MANOVA). This analysis was then repeated as a multivariate analysis of covariance (MANCOVA) controlling for potential effects of intelligence (as indexed via the General Ability Index; GAI). Considerable data reveal intelligence as an important determinant of neurocognitive performance [42]. Note that GAI did not include any of our neurocognitive outcome measures.

A series of exploratory analyses were conducted. First, a MANCOVA was conducted to determine an association of symptom severity (CY-BOCS total score) with test performance. Second, a group-based MANCOVA was conducted with the two processing speed measures as covariates. Third, three additional MANOVAs were conducted where the group contrasts were: (i) OCD patients with comorbidities vs. non-psychiatric controls, (ii) OCD patients without comorbidities vs. non-psychiatric controls; and (iii) OCD patients with comorbidities vs. OCD patients without comorbidities.

Effect sizes were calculated within SPSS as partial eta squared (η_p^2).

Results

Demographic and clinical characteristics

Nine patients with OCD and seven non-psychiatric controls had incomplete neurocognitive data and were thus not included in our main MANOVA analyses. The participants with OCD and the non-psychiatric control group were similar in terms of age and sex but differed significantly in GAI (intelligence), years of parental education and behavioral and emotional problems during testing as assessed with the TOF Total Score (see Table 2). Some participants with OCD were taking medications for other psychiatric/neurological conditions than OCD (Melatonin, $n = 8$ (sleep disorders); Methylphenidate, $n = 1$ (attention-deficit hyperactivity disorder); Valproate, $n = 1$ (epilepsy)).

Group differences in neurocognitive function

The MANOVA on group differences in neurocognitive function was highly significant for Group ($F(16,176) = 3.78$, $p < 0.001$, $\eta_p^2 = 0.256$). With respect to individual neurocognitive measures, compared to non-psychiatric controls, the main effect was driven by patients with OCD showing reduced accuracy on the AST, slower RTs during TMT switch trials, poorer quality of CGT decision-making,

Table 1 Neurocognitive tests and selected outcomes

Functions and tests	Test description	Outcomes
Cognitive flexibility		
Attention switching task (AST)[a]	Computerized test in which participants respond "right" or "left" based on either the direction or location of an arrow. The response rule switches continually throughout the task	Percent correct trials; switch cost[d]
Trail making test (TMT); number letter switching[b]	Paper–pencil test in which the participant draws a line connecting numbers and letters in ascending order while alternating between them (i.e., 1-A-2-B)	Completion time in seconds
Trail making test; verbal fluency switching[b]	Verbal test in which the participant names as many words as possible in 60 s, alternating between two categories (e.g., apple–table–pear–sofa)	Number of correct switches
Trail making test; design fluency, switching[b]	Paper–pencil test in which the participant draws as many different figures as possible in 60 s by connecting dots with straight lines, continually switching between filled and empty dots	Number of correct figures
Planning and decision-making		
Stockings of Cambridge (SOC)[a]	Computerized test in which the participant copies a model by rearranging items to match the model, trying to use the minimum moves required	Problems solved in min. moves[e]
Spatial working memory (SWM)[a]	Computerized test in which an increasing number of boxes are presented on the screen, and the participant is instructed to find a blue token behind one of them. When the token is found, it is hidden behind a new box and the participant is asked to avoid returning to a box where a token has already been found	Strategy[f]
Cambridge gambling task (CGT)[a]	Computerized test in which the participant guesses whether a token is hidden behind red boxes or blue boxes. On each trial, ten boxes are presented and the ratio of blue:red boxes is varied. The participant bets points on their decision, and points are won or subtracted according to outcome and bet-size	Quality of decision-making[g]; risk adjustment[h]
Working memory		
Wechsler working memory tests[c]	Visual and verbal subtests of the Wechsler Scales, including Verbal digit span, Picture span task (WISC-V) and arithmetic (WAIS-IV)	Working Memory Index (WMI) Score
Spatial working memory	*See above*	Total errors
Fluency		
Verbal fluency, phonemic[b]	Verbal test with three subtests in which the participant has 60 s to name as many words as possible beginning with a specific letter	Number of correct words
Verbal fluency, semantic[b]	Verbal test with two subtests in which the participant has 60 s to name as many words as possible from a specific semantic category	Number of correct words
Design fluency, empty dots[b]	Paper–pencil test in which the participant draws as many different figures as possible in 60 s, connecting empty dots and ignoring filled dots	Number of correct figures

Table 1 (continued)

Functions and tests	Test description	Outcomes
Processing speed		
Trail making test; number sequencing[b]	Paper–pencil test in which the participant draws a line, connecting numbers in ascending order	Completion time in seconds
Trail making test; letter sequencing[b]	Paper–pencil test in which the participant draws a line that connects letters in alphabetical order	Completion time in seconds

[a]Tests from the CANTAB battery [33, 34].

[b]Tests from the D-KEFS battery [34–36]

[c]Tests from the Wechsler Scales: WISC-V and WAIS-IV [26–28]

[d]The difference in the average reaction time (RT) in milliseconds between switch and repeat trials

[e]The number of trials in which the participant matched the model using as few moves as possible

[f]Based on the number of times the participant started each search within a trial with a new box instead of starting with the same box and following a systematic sequence—lower scores represent more efficient strategy use

[g]The proportion of trials where the participant chose the most likely outcome—higher scores represent better decision-making

[h]The degree to which the participant adjusted the bet-size according to the ratio of red:blue boxes—higher scores means higher degree of adjustment

Table 2 Demographic and clinical characteristics

Characteristic	OCD group Mean (*SD*), *n* (%)	Control group Mean (*SD*), *n* (%)	*p*	η^2
Number of participants	110	83		
Number of females	57 (51.8)	42 (50.6)	0.885	
Age in years	13.44 (2.70)	13.04 (2.81)	0.321	0.005
Parental education in years	15.55 (2.09)	16.71 (1.77)	< 0.001	0.77
General Ability Index (GAI; intelligence)[a]	99.03 (12.88)	106.57 (13.53)	< 0.001	0.076
TOF[b] Total Score	17.19 (20.20)	7.00 (7.77)	< 0.001	0.090
OCD severity				
CY-BOCS[c] total score	25.32 (4.75)			
CY-BOCS obsessions score	12.58 (2.70)			
CY-BOCS compulsions score	12.74 (2.43)			
Co-occurring psychiatric disorders				
ADHD (F90.0)	13 (11.82)			
Asperger's syndrome (F84.5)	13 (11.82)			
Generalized anxiety disorder (F41.1 and F93.8)	9 (8.18)			
Tourette's disorder (F95.2)	6 (5.45)			
AD[d] with mixed anxiety and depressed mood (F43.23)	5 (4.55)			
Other[e]	33 (30.00)			
Total with at least one co-occurring disorder	61 (55.45)			

[a]Wechsler Scales

[b]Test Observation Form

[c]Children's Yale-Brown Obsessive–Compulsive Scale

[d]Adjustment disorder

[e]Other: F30–F39 ($n = 1$), F40–F49 ($n = 12$), F50–59 ($n = 5$), F60-69 ($n = 1$), F80-89 ($n = 4$), F90–98.9 ($n = 13$)

reduced WMI, and slower RT in TMT Number-Sequencing (see Table 3). When we included the participants who were excluded from the MANOVA due to at least one missing value ($n = 9$; $n = 7$), results were comparable, and RT during TMT Letter-Sequencing turned out significant ($p = 0.031$, $\eta_p^2 = 0.023$; see Supplementary Material for more information).

Table 3 Group differences in neurocognitive test performance

Domain/task/outcome	OCD group $n=110$		Control group $n=83$		p^a	η_p^2
	Mean	SD	Mean	SD		
Cognitive flexibility						
AST[b] percent correct trials	91.62	8.39	93.86	5.13	0.033	0.024
AST[b] switch cost	268.37	145.08	310.17	136.40	0.043	0.021
TMT[c] switching, time in seconds	96.44	47.07	82.71	32.86	0.024	0.026
Verbal fluency switching[b], correct switches	11.63	3.01	11.11	2.86	0.227	0.008
	6.96	3.10	7.25	2.75	0.487	0.003
Planning and decision-making						
SOC[b] problems solved in minimum moves	8.10	2.16	7.64	2.93	0.209	0.008
SWM[b] strategy	31.83	6.23	31.06	5.44	0.372	0.004
CGT[b] quality of decision-making	0.90	0.11	0.95	0.06	< 0.001	0.070
CGT[b] risk adjustment	1.13	0.97	1.23	0.75	0.465	0.003
Working memory						
Wechsler working memory Index[d]	98.96	12.95	106.10	12.16	< 0.001	0.073
SWM[b] total errors	24.97	17.41	22.49	16.53	0.318	0.005
Fluency						
Verbal fluency phonemic[c], correct words	27.70	10.12	25.81	9.32	0.185	0.009
Verbal fluency semantic[c], correct words	38.41	10.62	37.80	8.82	0.670	0.001
	9.67	3.16	9.10	2.85	0.746	0.001
Processing speed						
TMT number-sequencing[c], time in seconds	37.91	18.44	31.21	13.24	0.005	0.040
TMT letter-sequencing[c], time in seconds	39.90	18.40	34.55	19.50	0.053	0.019

[a]Note p values reported here are from the MANOVA model, only those at $p<0.003$ (0.05/16) should be considered significant if examined as individual tests.

[b]Tests from the CANTAB battery [33, 34]. SWM, Spatial Working Memory; AST, Attention Switching Task; SOC, Stockings of Cambridge; CGT, Cambridge Gambling Task.

[c]Tests from the D-KEFS battery [34–36]. TMT, Trail Making Task.

[d]Tests from the Wechsler Scales [26–28]

Covariate analyses

The MANCOVA including IQ (General Ability Index; GAI) as a covariate also revealed highly significant group differences in neurocognitive function ($F(16,169)=3.168$, $p<0.001$, $\eta_p^2=0.231$) as well as highly significant effects on performance of intelligence ($F(16,169)=6.633$, $p<0.001$, $\eta_p^2=0.386$). With respect to individual neurocognitive measures, compared to non-psychiatric controls, patients with OCD showed reduced accuracy on the AST, slower RTs during TMT switch trials, poorer quality of CGT decision-making, and slower TMT number-sequencing RT (see Supplemental Table 4 for details)—note though that only the quality of CGT decision-making and working memory results would be considered significant if the tasks were examined via individual statistics.

Exploratory analyses

The first exploratory analysis using MANCOVA with CY-BOCS total score as a covariate revealed no significant association between OCD severity and neurocognitive test performance among the patients irrespective of whether IQ were used as additional covariates or not ($F(16,93)=1.587$, $p=0.088$, $\eta_p^2=0.214$ and $F(16,86)=1.372$, $p=0.175$, $\eta_p^2=0.203$, respectively).

The second exploratory analysis examining group differences involved a group-based MANCOVA with the two processing speed measures as covariates. This again revealed highly significant group differences ($F(14,176)=3.615$, $p<0.001$, $\eta_p^2=0.223$) as well as highly significant associations of performance with TMT number and letter sequencing RTs ($F(14,176)=2.690$, $p<0.001$, $\eta_p^2=0.176$ and $F(14,176)=5.656$, $p<0.001$, $\eta_p^2=0.310$, respectively).

The third exploratory analysis focused on determining whether group differences were present as a function of comorbidity within the patients with OCD. The first MANOVA revealed a significant group difference for patients without comorbidities ($n = 47$) relative to typically developing children/adolescents ($F(16,113) = 3.673$, $p < 0.001$, $\eta_p^2 = 0.34$). The second MANOVA revealed a significant group difference for patients *with* comorbidities ($n = 63$) relative to typically developing children ($F(16,129) = 2.784$, $p = 0.001$, $\eta_p^2 = 0.257$). The third MANOVA revealed no significant group difference for patients with comorbidities relative to patients without comorbidities ($F(16,93) = 0.574$, $p = 0.896$, $\eta_p^2 = 0.090$).

Discussion

We compared neurocognitive functioning in children and adolescents with moderate to severe OCD and non-psychiatric controls aged 8–17 years on neurocognitive measures assessing cognitive flexibility, planning and decision-making, WM, fluency, and processing speed. Analyses revealed that the patients with OCD showed markedly reduced neurocognitive performance relative to non-psychiatric controls, even after controlling for IQ as a potential covariate. These group differences were particularly driven by group performances on the AST, TMT, CGT, and WMI.

The extent to which children and adolescents with OCD show reduced neurocognitive performance relative to non-psychiatric controls has been debated with one meta-analysis reporting no significant group differences [12] while a recent review argued that they could be seen for visual WM, planning, decision-making under uncertainty and abnormal action monitoring [13]. Much of the older evidence was hampered by relatively small sample sizes. However, this has been less of a concern in recent work [11, 18–20] which, like the current study, has found evidence of group differences in neurocognitive performance.

Several important points are worth noting regarding the current results. First, the main effect of group, even for the MANCOVA including IQ and age as covariates, was highly significant. This indicates that children and adolescents with OCD do struggle with neurocognitive difficulties. Second, while groups differed in IQ—and IQ is a major determinant of neurocognitive performance (e.g., [42]), as was also seen in our data—the group difference in neurocognitive performance remained stable even after the influence of this variable was covaried out. Third, some have suggested that reduced processing speed underlies the neurocognitive underperformance seen in adults with OCD [42–44]. However, exploratory analyses with the current data indicated that the group differences in performance existed over and above group differences in processing speed. Fourth, despite the marked group differences in neurocognitive performance, our MANCOVA in the patients examining task performance and OCD symptom severity, as indexed by the CY-BOCS, revealed no significant association. In short, and consistent with previous work with adults and children/adolescents with OCD [6, 11, 45, 46], the neurocognitive difficulties indexed in the current study were not major contributors to patient symptom severity. Fifth, patients with OCD showed group differences with the non-psychiatric control participants whether these the patients presented with comorbidities or not while patients with versus without comorbidities did not differ in neurocognitive performance. These results are consistent with prior work in children/adolescents [11, 45, 46] and adults with OCD [6] and suggest that the neurocognitive difficulties identified cannot be attributed to comorbid conditions. Sixth, the neurocognitive difficulties shown by the children and adolescents with OCD were potentially particularly marked for decision-making and WM.

Difficulties in decision-making in the participants with OCD could be seen on the CGT. In line with this finding, recent studies have reported that children/adolescents with OCD, relative to comparison participants, were slower to learn response–outcome relationships and worse at adapting response strategy when previously rewarded actions were devalued compared to non-psychiatric control children [47, 48] and show poorer decision-making ability [49, 50]. The Wechsler WMI revealed group differences while the SWM index did not. Reduced working verbal and visual memory has been relatively consistently seen in adults with OCD [5] and also reported in child/adolescent samples (for a review, see [13]). Recently, reduced verbal WM was reported in one larger *N* study [11], whereas spatial WM was not in another [19].

Our study has several strengths: (i) the N was relatively large, (ii) the patients were not receiving pharmacological interventions or psychotherapy for OCD (nor had for the last 6 months) thereby avoiding potential confounding treatment effects [22, 23]; and (iii) the patient sample included a broad range of comorbidities (i.e., it was representative of real-world clinical populations). Moreover, and importantly, we could show groups of patients with and without comorbidities differed in neurocognitive performance relative to typically developing children/adolescents but did not differ from each other; i.e., the neurocognitive performance difficulties seen in the patients cannot be attributed to pathology associated with the comorbidities.

Our study also has several limitations. First, since the data presented here are cross-sectional, we were unable to evaluate if the observed neurocognitive underperformance is moderated by e.g., illness duration, earlier pharmacological or therapeutic intervention, or age of onset. Second, we cannot ensure that the patients were *naïve* to pharmacological and/or psychotherapeutic interventions for OCD as we did

not assess if patients received antidepressants or antipsychotics before 6 months prior to testing. Third, we included patients with a variety of comorbidities that are known to be associated with neurocognitive underperformance. As such, it could be argued that some of our findings might relate to pathology associated with these comorbidities. Importantly, though, follow-up MANOVAs indicated that the results were not likely to be attributed to these comorbidities. Fourth, the groups differed in IQ, which is in itself a determinant of neurocognitive performance [42]. However, controlling for intelligence in secondary analyses led to results that largely mirrored those of our main analysis.

The results have clinical implications. While the atypical neurocognitive functioning observed in patients was not associated with patient symptom severity, it was highly significant and as such likely conferring some detrimental impact on patients' lives. Moreover, this atypical neurocognitive functioning may interfere with particularly psychosocial interventions. Knowing that forms of neurocognitive dysfunction which are seen in adult patients, are already present in childhood OCD, stresses the need for studying their developmental trajectory in patients with OCD.

In conclusion, the current study indicates that many pediatric patients with OCD show a degree of atypical neurocognitive function even if the forms of atypical functioning identified here, and in previous work, do not appear to be associated with the specific symptoms associated with OCD. As such, a more precise neurocognitive model of OCD is clearly required such that it can be tested in the future with specifically designed target tests. Moreover, it will be important to determine the extent to which the neurocognitive difficulties observed here are ameliorated by treatment for OCD or, indeed, moderate treatment efficacy.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00787-023-02301-w.

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Author contributions CFU: Conceptualization, data collection, data analysis, writing the main manuscript text, review and editing. MR: Data collection, data analysis, review and editing JRMJ: Conceptualization, data analysis plan, supervision, review and editing. VFU: Data collection, data analysis, review and editing. NNL: Data collection, data analysis plan, review and editing ADM: Conceptualization, review and editing KJP: Conceptualization, data analysis plan, supervision, review and editing SV: Conceptualization, data analysis plan, supervision, review and editing RJB: Data analysis, review and editing AKP: Conceptualization, data analysis plan, supervision, review and editing All authors reviewed the final manuscript.

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Data Availability The data presented in this article is from a longitudinal case-control study embedded in a randomized controlled trial. The trial was recently completed and main findings are not yet published. The data from the the trial and substudies are not available in a public repository at the moment.

Declarations

Conflict of interests None.

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Supplementary material

The Danish Central Person Register (CPR) is managed by the Danish Health Data Authority and contains personal identification numbers of all Danes, making it possible to link data across the national health data registers. It can be accessed for research by researchers. For the recruitment of non-psychiatric control children, we acquired basic information on 10 random children/adolescents for each included patient, matched on age and sex and living in the Capital Region of Denmark. We contacted parents and included children/adolescents who met inclusion criteria and consented to participate.

Assessments were carried out by trained investigators (including authors CFU, MR, VFU, and NNL). K-SADS and CY-BOCS were administered by medical doctors, clinical psychologists, and psychology master's students. They trained for administering K-SADS by completing and passing an official course for clinicians at the CAMHC and for CY-BOCS by observing and getting observed by an experienced psychologist. Neurocognitive tests were administered and scored by trained psychologists and psychology master's students under expert supervision by authors, JRMR and SV.

The process by which diagnosis is determined involves a clinical conference, in which a child psychiatrist or a psychologist (specialized in child psychiatry with a four-year education) evaluate all results of the psychiatric screening.

Supplemental Table 1 Co-occurring disorders (*n* = 110; all co-occurring disorders included)

Characteristic	**OCD group** *n* (%)
Number of participants	110
Co-occurring disorders	
Major Depressive Disorder, single episode, mild (F32.0)	1 (0.91)
Agoraphobia (F40.0)	1 (0.91)
Social Anxiety Disorder (F40.1)	1 (0.91)
Specific (isolated) Phobias (F40.2)	1 (0.91)
Generalized Anxiety Disorder (F41.1)	4 (3.64)
Other Specified Anxiety Disorders (F41.8)	3 (2.73)
Adjustment Disorder, Unspecified (F43.20)	1 (0.91)
Adjustment Disorder with Depressed Mood (F43.21)	3 (3.64)
Adjustment Disorder with Anxiety (F43.22)	2 (1.82)
Adjustment Disorder with Mixed Anxiety and Depressed Mood (F43.23)	5 (4.55)
Adjustment Disorder with Disturbance of Conduct (F43.24)	1 (0.91)
Adjustment Disorder with mixed Disturbance of Emotions and Conduct (F43.25)	1 (0.91)
Other Reactions to Severe Stress (F43.8)	1 (0.91)
Anorexia Nervosa (F50.0)	1 (0.91)
Atypical Anorexia Nervosa (F50.1)	1 (0.91)
Other Eating Disorders (F50.8)	1 (0.91)
Eating Disorder, Unspecified (F50.9)	1 (0.91)
Other sleep disorders not due to a substance or known physiological condition (F51.8)	1 (0.91)
Anxious [Avoidant] Personality Disorder (F60.6)	1 (0.91)
Asperger's Syndrome (F84.5)	13 (11.82)
Other Disorders of Psychological Development (F88.0)	2 (1.82)
Other Disorders of Psychological Development NEC (F88.9)	2 (1.82)
Attention-Deficit Hyperactivity Disorder, Predominantly Inattentive Type (F90.0)	13 (11.82)
Attention-Deficit Hyperactivity Disorder, Predominantly Hyperactive Type (F90.1)	1 (0.91)
Oppositional Defiant Disorder (F91.3)	1 (0.91)
Other Childhood Emotional Disorders (F93.8)	5 (4.55)
Transient Tic Disorder (F95.0)	2 (1.82)
Chronic Motor or Vocal Tic Disorder (F95.1)	1 (0.91)
Combined Motor and Vocal Tic Disorder [de la Tourette] (F95.2)	6 (5.45)
Other Tic Disorders (F95.8)	1 (0.91)
Tic Disorder, Unspecified (F95.9)	2 (1.82)
Enuresis Not Due To A Substance or Known Psychological Condition (F98.0)	3 (2.73)
Other specified behavioural and emotional disorders with onset usually occurring in childhood and adolescence (F98.8)	2 (1.82)
Total with at least one co-occurring disorder	61 (55.45)

Supplemental Table 2 Demographic and Clinical Characteristics ($N = 209$; all subjects included)

Characteristic	**OCD group** Mean (*SD*), *n* (%)	**Control group** Mean (*SD*), *n* (%)	***p***	η^2
Number of participants	119	90		
Number of females	62 (52.10)	47 (52.22)	.549	
Age in years	13.34 (2.79)	12.93 (2.79)	.300	.005
Parental education in years	15.56 (2.08)	16.79 (1.75)	<.001	.087
General Ability Index (WISC-V/WAIS-IV[a])	98.76 (12.76)	106.51 (13.73)	<.001	.079
TOF[b] total score	16.86 (19.80)	6.99 (7.85)	<.001	.088
OCD severity				
CY-BOCS[c] total score	25.49 (4.94)			
CY-BOCS obsessions score	12.70 (2.75)			
CY-BOCS compulsions score	12.79 (2.54)			
Co-occurring disorders				
Attention Deficit Hyperactivity Disorder (F90.0)	14 (11.76)			
Asperger's Syndrome (F84.5)	14 (11.76)			
Generalized Anxiety Disorder (F41.1 and F93.8)	9 (7.56)			
Tourette's Disorder (F95.2)	6 (5.04)			
AD[d] with Mixed Anxiety and Depressed Mood (F43.23)	5 (4.20)			
AD with Depressed Mood (43.21)	4 (3.36)			
Other	30 (25.21)			
Total with at least one co-occurring disorder	66 (55.46)			

[a]Wechsler Intelligence Scale for Children Version V; Wechsler Adult Intelligence Scale Version IV
[b]Test Observation Form
[c]Children's Yale-Brown Obsessive Compulsive Scale
[d]Adjustment Disorder
[e]Other: F30-F39 ($n = 1$), F40-F49 ($n = 14$), F50-59 ($n = 5$), F60-69 ($n = 1$), F80-89 ($n = 5$), F90-98.9 ($n = 14$)

Supplemental Table 3 Co-occurring disorders (*n* = 119; all patients and co-occurring disorders included)

Characteristic	**OCD group** *n* (%)
Number of participants	119
Co-occurring disorders	
Major Depressive Disorder, single episode, mild (F32.0)	1 (0.84)
Agoraphobia (F40.0)	1 (0.84)
Social Anxiety Disorder (F40.1)	2 (1.68)
Specific (isolated) Phobias (F40.2)	1 (0.84)
Generalized Anxiety Disorder (F41.1)	4 (3.36)
Other Specified Anxiety Disorders (F41.8)	3 (2.52)
Acute Stress Reaction (F43.0)	1 (0.84)
Adjustment Disorder, Unspecified (F43.20)	1 (0.84)
Adjustment Disorder with Depressed Mood (F43.21)	4 (3.36)
Adjustment Disorder with Anxiety (F43.22)	2 (1.68)
Adjustment Disorder with Mixed Anxiety and Depressed Mood (F43.23)	5 (4.20)
Adjustment Disorder with Disturbance of Conduct (F43.24)	1 (0.84)
Adjustment Disorder with mixed Disturbance of Emotions and Conduct (F43.25)	1 (0.84)
Other Reactions to Severe Stress (F43.8)	1 (0.84)
Anorexia Nervosa (F50.0)	1 (0.84)
Atypical Anorexia Nervosa (F50.1)	1 (0.84)
Other Eating Disorders (F50.8)	1 (0.84)
Eating Disorder, Unspecified (F50.9)	1 (0.84)
Other sleep disorders not due to a substance or known physiological condition (F51.8)	1 (0.84)
Anxious [Avoidant] Personality Disorder (F60.6)	1 (0.84)
Other Developmental Disorders of Scholastic Skills (F81.8)	1 (0.84)
Asperger's Syndrome (F84.5)	14 (11.76)
Other Disorders of Psychological Development (F88.0)	2 (1.68)
Other Disorders of Psychological Development NEC (F88.9)	2 (1.68)
Attention-Deficit Hyperactivity Disorder, Predominantly Inattentive Type (F90.0)	14 (11.76)
Attention-Deficit Hyperactivity Disorder, Predominantly Hyperactive Type (F90.1)	1 (0.84)
Oppositional Defiant Disorder (F91.3)	1 (0.84)
Phobic Anxiety Disorder of Childhood (F93.1)	1 (0.84)
Other Childhood Emotional Disorders (F93.8)	5 (4.20)
Transient Tic Disorder (F95.0)	2 (1.68)
Chronic Motor or Vocal Tic Disorder (F95.1)	1 (0.84)
Combined Motor and Vocal Tic Disorder [de la Tourette] (F95.2)	6 (5.04)
Other Tic Disorders (F95.8)	1 (0.84)
Tic Disorder, Unspecified (F95.9)	2 (1.68)
Enuresis Not Due To A Substance or Known Psychological Condition (F98.0)	3 (2.52)
Other specified behavioural and emotional disorders with onset usually occurring in childhood and adolescence (F98.8)	2 (1.68)
Total with at least one co-occurring disorder	66 (55.46)

Supplemental Table 4 Group Differences in Neurocognitive Test Performance (N = 209; all subjects included)

	OCD Group n = 119		Control Group n = 90			
Domain/task/outcome	**Mean**	**SD**	**Mean**	**SD**	p^a	η_p^2
Cognitive Flexibility						
AST[b] Percent Correct Trials	91.58	8.28	93.80	5.10	.027	.024
AST[b] Switch Cost	267.83	142.46	309.77	134.66	.034	.022
TMT[c] Switching, Time in Seconds	96.34	46.70	82.83	32.08	.020	.026
Verbal Fluency Switching[c], Correct Switches	11.47	3.03	11.24	2.88	.569	.002
Design Fluency Switching[c], Correct Figures	6.85	3.13	7.24	2.77	.350	.004
Planning and Decision-making						
SOC[b] Problems Solved in Minimum Moves	8.04	2.14	7.59	2.99	.205	.008
SWM[b] Strategy	31.84	6.06	31.08	5.31	.347	.004
CGT[b] Quality of Decision Making	.89	.11	.95	.06	<.001	.080
CGT[b] Risk Adjustment	1.12	.96	1.13	.79	.251	.007
Working Memory						
Wechsler Working Memory Index[d]	98.45	13.07	105.93	12.22	<.001	.079
SWM[b] Total Errors	25.15	17.06	22.39	16.85	.246	.007
Fluency						
Verbal Fluency Phonemic[c], Correct Words	27.31	10.21	25.52	9.43	.202	.008
Verbal Fluency Semantic[c], Correct Words	37.94	10.57	37.51	8.85	.755	.000
Design Fluency[c], Correct Figures	9.59	3.15	9.59	2.90	.995	.000
Processing Speed						
TMT Number-Sequencing[c], Time in Seconds	38.25	18.29	31.08	12.91	.002	.047
TMT Letter-Sequencing[c], Time in Seconds	40.09	18.28	34.42	18.85	.031	.023

[a]Note only p-values at $p < .003$ (0.05/16) survive multiple comparison correction
[b]Tests from the CANTAB battery [33]. SWM, Spatial Working Memory; AST, Attention Switching Task; SOC, Stockings of Cambridge; CGT, Cambridge Gambling Task
[c]Tests from the D-KEFS battery [34, 35]. TMT, Trail Making Task
[d]Tests from the Wechsler Scales [26, 27]

Supplemental Table 3 Group Differences in Neurocognitive Test Performance (Covariate analyses; IQ)

	OCD Group *n* = 104		Control Group *n* = 83			
Domain/task/outcome	**Mean**	**SEM**	**Mean**	**SEM**	p^a	η_p^2
Cognitive Flexibility						
AST[b] Percent Correct Trials	91.85	.61	93.54	.68	.129	.012
AST[b] Switch Cost	279.85	13.47	304.71	15.14	.248	.007
TMT[c] Switching, Time in Seconds	94.41	3.18	85.10	3.58	.137	.012
Verbal Fluency Switching[c], Correct Switches	11.74	.24	10.93	.27	.073	.017
Design Fluency Switching[c], Correct Figures	6.95	.23	7.14	0.26	.669	.001
Planning and Decision-making						
SOC[b] Problems Solved in Minimum Moves	8.18	.24	7.48	.27	.069	.018
SWM[b] Strategy	31.76	.55	31.24	.62	.563	.002
CGT[b] Quality of Decision Making	.90	.01	.94	.01	<.002	.050
CGT[b] Risk Adjustment	1.20	.08	1.18	.09	.888	.000
Working Memory						
Wechsler Working Memory Index[d]	101.09	1.06	103.95	1.19	.079	.017
SWM[b] Total Errors	24.43	1.50	23.49	1.69	.704	.001
Fluency						
Verbal Fluency Phonemic[c], Correct Words	28.27	.77	25.05	.87	.029	.026
Verbal Fluency Semantic[c], Correct Words	38.74	.81	37.09	.91	.260	.007
Design Fluency[c], Correct Figures	9.76	.26	9.37	.29	.393	.004
Processing Speed						
TMT Number-Sequencing[c], Time in Seconds	38.45	1.44	31.51	1.62	.017	.031
TMT Letter-Sequencing[c], Time in Seconds	39.90	1.56	35.90	1.76	.362	.005

[a]Note *p*-values reported here are from the MANOVA model, only those at $p < .003$ (0.05/16) should be considered significant if examined as individual tests.

[b]Tests from the CANTAB battery [33]. SWM, Spatial Working Memory; AST, Attention Switching Task; SOC, Stockings of Cambridge; CGT, Cambridge Gambling Task

[c]Tests from the D-KEFS battery [34, 35]. TMT, Trail Making Task

[d]Tests from the Wechsler Scales [26, 27]

Title: Neurocognitive correlates of psychotherapeutic treatment response in pediatric obsessive-compulsive disorder: Results from the TECTO trial

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Abstract

Background:

Atypical neurocognitive functioning is consistently reported in adults with obsessive-compulsive disorder (OCD), while studies in childhood/early-onset OCD show smaller and more selective effects. Neurobiological models of OCD implicate aberrant activation within cortico-striato-thalamo-cortical (CSTC) circuits and related executive function (EF) impairment as core mechanisms underlying OCD symptomatology. Neurocognitive correlates of psychotherapeutic treatment response have not been examined across different pediatric psychotherapy groups in a randomized design.

Methods: Ninety children (aged 8-17 years) with OCD were randomized to 14 sessions of either cognitive-behavioral therapy (CBT) or psychoeducation and relaxation training (PRT) and assessed with a comprehensive neurocognitive test-battery before randomization (baseline) and after treatment (week 16). An age- and sex-matched control group of 87 non-psychiatric children were assessed within a comparable assessment interval.

Results: Children with OCD showed subtle improvements during treatment, relative to controls, on neurocognitive domains identified as atypical at baseline, specifically decision-making (pre-selected) and processing speed (explorative). However, there was no differential effect of treatment type (CBT versus PRT) and no association between neurocognitive change and OCD symptom improvement. Symptom improvement was neither predicted nor moderated by baseline neurocognitive performance.

Conclusion: These findings indicate that psychotherapeutic interventions may yield neurocognitive benefits in pediatric OCD. However, the small and selective effects, together with the lack of differential treatment effects and the absence of association with symptom improvement, raise questions about the clinical relevance of these changes and whether CSTC-related processes play a central role in pediatric OCD.

Keywords: Obsessive-compulsive disorder, Cognition, Executive function, Children, Cognitive-behavioral therapy, Exposure and response prevention, Randomized clinical trial

1 Introduction

Obsessive-compulsive disorder (OCD) is a mental illness characterized by obsessions and compulsions that impair the everyday function and affects up to 3 % of children and adolescents worldwide (Farrell et al., 2023). Cognitive-behavioral therapy (CBT) with exposure and response prevention (ERP) is an effective treatment for many children and adolescents with OCD (Cervin et al., 2024; National Institute for Health and Care Excellence (NICE), 2005). Nonetheless, a considerable number of children with OCD (around 40 %) are partial or non-responders, while around 60 % of children experience remission of OCD symptoms (McGuire et al., 2015; Stewart et al., 2004). Understanding the neurocognitive underpinnings of the disorder and the impact of psychotherapeutic treatment response in pediatric OCD on those could inform our understanding of therapy mechanisms and support the development of more effective and personalized interventions.

The classical neurobiological model of the illness attributes OCD symptoms to imbalances in the antagonistic pathways within the cortico-striato-thalamo-cortical (CSTC) loops in the brain (Jalal et al., 2023; Robbins et al., 2024). Increased activity in direct excitatory pathways is thought to underlie the inflexible pattern of obsessions and compulsions seen in OCD, and following this, a deficit in cognitive flexibility and related executive functions (EF) is suggested to underlie the disease. Aberrant CSTC activity has been associated with neurocognitive dysfunction, which is consistently found in adults with OCD (Snyder et al., 2015) and their unaffected relatives (Bora, 2020). However, pediatric studies show smaller and more selective effects (Abramovitch et al., 2015; Marzuki et al., 2020). The EF domains most often associated with OCD symptom severity are flexibility, planning, and response inhibition (Abramovitch et al., 2015, 2019). Our own research group documented selective atypical performance in patients with OCD relative to controls in the EF domains of flexibility, decision-making, and working memory, and also in processing-speed (Uhre et al., 2023). It should also be noted that, within the OCD group, we found no associations between neurocognitive performance and OCD symptom severity at baseline. This suggests that the neurocognitive functions classically indexed may not directly underpin OCD symptoms – even if they likely contribute to the child's level of well-being and functioning. However, they may influence the individual's response to treatment and perhaps be ameliorated by treatment. The current study investigates the extent to which two forms of psychotherapy impact neurocognitive test performance and the extent to which pre-treatment performance predicts and/or moderates treatment response.

Following the CSTC model of OCD, treatment with CBT has been suggested to work by ameliorating EF deficits as well as OCD symptoms, and pre-treatment EF difficulties have been suggested a predictor of poorer CBT response. Although some studies have indicated improvement in neurocognitive test performance after OCD treatment in adult patients (Moritz et al., 1999; Kuelz et al., 2006; Voderholzer et al., 2013; Katz et al., 2024; Wheaton et al., 2025), other studies did not observe similar effects (Kim et al., 2002; Roh et al., 2005;

Bannon et al., 2006; Rao et al., 2008; Braga et al., 2016; Reid et al., 2025). Five studies with pediatric samples have also been conducted (Andrés et al., 2008; Huyser et al., 2010, 2011; Hybel et al., 2017; van der Straten et al., 2018). The pediatric literature, like the adult literature, reports mixed findings with three studies reporting improvements in neurocognitive function after CBT (and also SSRIs) (Andrés et al., 2008; Huyser et al., 2010; van der Straten et al., 2018) while two studies did not (Huyser et al., 2011; Hybel et al., 2017). In particular, planning- and visual memory related functions (overall mean completion time on the Tower of London Task (Huyser et al., 2010; van der Straten et al., 2018) and accuracy on an immediate visual reproduction test as well as copying time on the Complex Figure Test (Andrés et al., 2008)) were reported to improve following treatment.

Pre-treatment neurocognitive functioning may influence psychotherapy response, acting as a potential predictor and/or moderator of treatment outcome. Consistent with this, one study with adult OCD patients identified processing speed as a predictor of treatment response versus non-response (Kuelz et al., 2006). However, two studies observed no association between baseline neurocognition and treatment outcome (Braga et al., 2016; Voderholzer et al., 2013). Two pediatric studies have investigated this issue. One reported that poorer recall accuracy on the Complex Figure Test predicted poorer outcome of CBT (Flessner et al., 2010), while the other reported that poorer executive function, based on a latent variable, predicted *greater* symptom improvement (Hybel et al., 2017).

Taken together, these studies first suggest that CBT may lead to selective cognitive improvements of pediatric OCD – namely in visual memory and planning – but findings remain inconsistent and are generally limited by small sample sizes, heterogeneous treatments, no control intervention, and reliance on either narrow test batteries or an aggregated latent variable. Moreover, only one pediatric study has examined baseline executive function (based on the latent variable) as a potential predictor of treatment response, highlighting the need for further research to clarify which cognitive domains might influence therapy outcomes and how they change following treatment.

To address the gaps in the literature, the present study compares a group of children and adolescents with OCD randomized to two active treatment conditions: CBT with ERP or an active control treatment of psychoeducation and relaxation training (PRT). Additionally, we include a non-psychiatric control group. We use a broader neurocognitive test battery that allows for analyses of specific EF and related domains. Clinical outcomes of the trial (including the intention-to-treat population) indicated that both treatments reduced OCD symptoms, with CBT showing greater improvement than PRT (*Mean difference* = -3.89, $p = .010$) (Pagsberg et al., 2025). For the present study, primary EF outcomes were selected based on domains showing baseline atypical functioning (Uhre et al., 2023): i.e., tests of cognitive flexibility (completion time and accuracy on set-shifting tasks) and decision-making. We hypothesized that a) CBT and, to a lesser extent, PRT would improve patients' atypical flexibility and decision-making performance; b) neurocognitive improvement was

associated with symptom alleviation; c) baseline flexibility and decision-making difficulties would be significantly associated with less symptom improvement seen for both of these interventions, and d) more so for CBT versus PRT, i.e., they act as predictors and moderators of symptom improvement.

2 Methods

2.1 Trial design

This study is part of the TECTO randomized clinical trial (RCT), which compares family-based CBT with ERP to an active control intervention, family-based PRT, in 130 non-medicated children aged 8-17 years with moderate to severe OCD (Pagsberg et al., 2025). Patients received 14 sessions of therapy over 16 weeks. The TECTO study also included 90 age- and sex-matched non-psychiatric control participants. Primary OCD diagnosis and potential comorbidities were assessed in patients, and the absence of current or past diagnoses was confirmed in controls, using The Kiddie Schedule for Affective Disorders and Schizophrenia Present and Lifetime Version (K-SADS-PL) (Kaufman et al., 1997), a semi-structured diagnostic interview. Diagnoses were established according to the ICD-10 criteria (ICD 10; World Health Organization, 2019). General cognitive ability was assessed with the age-appropriate version of the Wechsler Intelligence Scales (WISC-V; WIAS-IV) (Wechsler, 2014, 2008). Children with a full-scale intelligence quotient (IQ) of < 70 were excluded. For further methodological details of the RCT trial (e.g., inclusion- and exclusion criteria and treatment details), see (Pagsberg et al., 2022; Olsen et al., 2022; Pagsberg et al., 2025).

2.2 Study outcomes

2.2.1 Neurocognitive performance

Neurocognitive functioning was assessed in patients at before randomization (baseline) and after treatment (week 16) and in controls with a similar time interval. Based on which EF domains appeared atypical at baseline (Uhre et al., 2023), three outcomes were chosen as primary for the current study, prior to the initiation of analyses: 1) Cambridge Gambling Task, quality of decision-making (CGT-QDM), 2) Attention Switching Task, percent correct trials (AST-PCT), and 3) Trail Making Task, switch condition, reaction time (TMT-SRT). The first two are computer-based tasks from the Cambridge Neuropsychological Test Automated Battery (CANTAB) (Cambridge Cognition, 2019) and the third is paper-and-pencil based and from the Delis-Kaplan Executive Function System (D-KEFS) (Delis et al., 2001). Note than working memory was only assessed at baseline and thus could not be studied at follow-up. In addition to the primary outcomes, the neurocognitive battery included 13 other outcomes from CANTAB, D-KEFS and the Wechsler scales (Wechsler, 2008, 2014), which were examined in exploratory analyses. All outcomes are described in detail in Uhre et al. (2023).

2.2.2 OCD symptom severity

The primary outcome of the TECTO trial was OCD symptom severity, measured using the total score from the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS) semi structured interview (Scahill et al., 1997). The scale rates severity of obsessions and compulsions with regard to frequency, functional impairment, psychological distress, efforts to resist, and degree of control. The items are summed to yield a total score ranging 0-40. A CY-BOCS total score of ≥ 16 was an inclusion criterion.

In addition, we reported the percentage of treatment completers (i.e., completing ≥ 10 sessions out of 14) and the percentage of patients in remission (i.e., no longer meeting ICD-10 criteria for OCD) for each treatment group.

2.3 Participants

We included all TECTO participants and non-psychiatric controls with neurocognitive data available at both baseline and 16-week follow-up for at least one neurocognitive outcome. Thus, we included 90 children and adolescents with OCD (41 received PRT, and 49 received CBT) and 87 controls. For prediction and moderation analyses, we included an additional 13 patients who had missing neurocognitive data but not missing CY-BOCS at follow-up (CBT: 5, PRT: 8).

Data analysis

We carried out statistical analyses with R (version 4.5.0). To control for family-wise error rate, p-values for each primary outcome were Bonferroni-adjusted across a family of five *a priori* tests (main effect of Time, Time × Group interaction, correlation between neurocognitive improvement and OCD symptom improvement, prediction, and moderation). The adjusted significance threshold was set to $\alpha = .05 / 5 = .010$. For exploratory analyses, an alpha level of .05 was applied. For primary analyses, model assumptions were considered and evaluated. Although our hypotheses specified expected direction of the effect, we employed two-sided tests to provide a more conservative assessment of the effects.

2.3.1 Demographic and clinical characteristics

For demographic and clinical characteristics, continuous variables are reported as means and standard deviations (SD), and we tested potential group differences using analysis of variance (ANOVA). In case of a significant main effect, follow-up independent samples *t*-tests were used to examine pairwise comparisons. For categorical variables, we report counts (*n*) and percentages, and potential group differences were assessed using chi-square tests. In case of a significant main effect, pairwise comparisons were performed using Fisher's exact test. In line with previous work (Uhre et al., 2023), parental education in years was calculated as the

mean of both parents. If only one parent participated, we used that parent's value. Psychiatric comorbid diagnoses were reported if present in more than 3 % of the patients in our sample. Analyses of CY-BOCS scores and comorbidity were restricted to patients only (PRT versus CBT).

2.3.2 Baseline neurocognitive performance

Baseline neurocognitive performance was compared between patients and controls with follow-up data. This was done to determine, whether previously reported group differences (Uhre et al., 2023) observed in the original sample (patients: $N = 110$; controls: $N = 83$) could also be seen in the current subset of participants with complete follow-up data (patients: $N = 77$; controls: $N = 80$). An overall Multivariate Analysis of Variance (MANOVA) was conducted to examine global group differences across neurocognitive domains, followed by individual ANOVAs for each neurocognitive outcome. To assess whether group differences persisted when controlling for general cognitive ability, a supplementary MANCOVA was conducted with IQ included as a covariate was conducted.

2.3.3 Change in neurocognitive performance

All neurocognitive outcomes were standardized to z-scores using the mean and SD of the non-psychiatric control group (CTR) at baseline. Change in neurocognitive performance over 16 weeks (hypothesis a) was analyzed using three 3 (Group: CTR, PRT, CBT) x 2 (Time: week 0, 16 weeks) mixed repeated measures analyses of variance (ANOVA). We used the *afex* package in R (Singmann et al., 2025). Each neurocognitive outcome was entered separately as the dependent variable, and each ANOVA included Group as a between-subjects factor and Time as a within-subjects factor, and effect sizes were reported as generalized eta squared (ges). Follow-up analyses reported estimated marginal means, using the *emmeans* package in R (Lenth et al., 2025). Assumptions of normality of residuals and homogeneity of variance were considered and inspected using residual plots and QQ plots; because sphericity was not an issue with only two time points, no formal correction was required. To ensure robustness against potential violations, sensitivity analyses were conducted for the three primary outcomes using aligned rank transform ANOVA. In case of a significant main effect of Time, Group, and/or a significant Group by Time interaction, post-hoc pairwise comparisons of estimated marginal means were conducted. Sensitivity analyses restricted to treatment-completers were added.

We conducted three linear regression analyses to test, whether improvement in neurocognitive performance predicted OCD symptom improvement in patients (hypothesis b). Follow-up CY-BOCS total score was the dependent variable, while baseline CY-BOCS total score and change in EF domains score were predictors.

In addition to the primary outcomes, exploratory analyses were carried out for 13 other neurocognitive outcomes, using the same repeated measures ANOVA and linear regression approaches.

2.3.4 Prediction and moderation

Three linear regression analyses were conducted to examine whether baseline performance on the three primary neurocognitive measures predicted symptom improvement (in both treatment groups; hypothesis c) and/or moderated the effect of treatment group (CBT versus PRT; hypothesis d) in patients with OCD. Regression models were performed using the base R function *lm()* for linear modelling. For each model, follow-up CY-BOCS score served as the dependent variable, with baseline CY-BOCS score, baseline neurocognitive performance, and treatment group as predictors. Each model also included the interaction between baseline neurocognitive performance and treatment group to test whether neurocognition moderated treatment-related symptom improvement.

In addition to the primary outcomes, exploratory analyses were carried out for 13 other neurocognitive outcomes, using the same regression analysis approach.

3 Results

The mean time between neurocognitive assessments was 16.1 weeks (11.9-22.6 range) for controls, 17.2 weeks (13.1-39.3 range) for PRT, and 16.6 weeks (13.0-22.6 range) for CBT. Of included participants, data were available at both baseline and follow-up for 97.7-98.9 % controls, 85.4-90.2 % receiving PRT, and 87.8-95.9 % receiving CBT across the three primary outcomes. For a more detailed overview of available data by outcome, group, and time point, see Supplemental Table S1.

3.1 Demographic and clinical characteristics

Baseline characteristics are presented in Table 1. The groups differed significantly in parental education and IQ (F (2,154) = 7.693, $p < .001$, $\eta^2 = .091$; F(2,172) = 4.452, $p < .001$, $\eta^2 = .049$), and pairwise comparisons showed that for both variables, the PRT group and the CBT group differed significantly from the control (CTR) group while not from each other. No other group differences were identified.

Moreover, 91.8 % of CBT participants and 85.4 % of PRT participants were completers (i.e., completing at least 10 out of 14 sessions), and finally, 24.5 % of CBT and 17.1 % of PRT participants were in remission (i.e., did no longer meet ICD-10 criteria for OCD).

Table 1 Demographic and clinical characteristics

	CTR	**PRT**	**CBT**			
	Mean (SD), n (%)	Mean (SD), n (%)	Mean (SD), *n* (%)	*p*	Effect	Pairwise comparisons
Number of participants	87	41	49			
Number of females	44 (50.6)	22 (53.7)	31 (63.3)	.356	.108	
Age in years	13.0 (2.81)	13.3 (2.59)	13.2 (2.84)	.816	.002	
Parental education, years	16.8 (1.76)	15.7 (1.71)	15.4 (2.45)	<.001	.091	CTR > PRT, CBT; CBT = PRT
IQ	106.0 (13.9)	101.0 (13.9)	99.1 (12.7)	<.001	.049	CTR > PRT, CBT; CBT = PRT
CY-BOCS total score	-	25.2 (4.44)	24.2 (4.07)	.245	.250	-
Comorbid diagnoses						
Asperger's (F84.5)	-	6 (14.6)	3 (6.1)	.323	.141	-
ADHD (F90.0)	-	4 (9.8)	5 (10.2)	1	.337	-
Adj. disorder (F43.23)	-	3 (7.3)	2 (4.1)	.837	.070	-
GAD (F41.1 & F93.8)	-	4 (9.8)	1 (2.0)	.259	.168	-
Adj. disorder (F43.21)	-	3 (7.3)	1 (2.0)	.486	.128	-
Tourette's (F95.2)	-	2 (4.9)	2 (4.1)	.307	.170	-
Any comorbid diagnosis	-	21 (51.2)	27 (55.3)	.876	.039	-

Note: CTR = Control children. CBT = Patients who received cognitive-behavioral therapy (CBT). PRT = Patients who received psychoeducation and relaxation training (PRT). Effect size: As effect sizes we used Eta-squared or Cohens *d* for numerical data and Cramer's *V* for categorical data. IQ = Intelligence Quotient. CY-BOCS = Children's Yale-Brown Obsessive-Compulsive Scale. Comorbid diagnosis is reported if present in > 3 % of patients. ADHD = Attention-Deficit/Hyperactivity Disorder. GAD = Generalized Anxiety Disorder. Adj. disorder = Adjustment disorder (F43.23 = Adjustment disorder, mixed disturbance; F 43:21 = Adjustment disorder, depressive reaction).

3.2 Baseline neurocognitive performance

Baseline neurocognitive performance was compared between patients and controls to assess whether previously reported differences (Uhre et al., 2023) persisted in the current follow-up sample.

The MANOVA remained significant for group differences ($F(16,140) = 3.27$, $p < .001$). For individual primary neurocognitive measures, patients still showed significantly poorer CGT-QDM ($F(1,168) = 13.25$, $p < .001$) and slower reaction times on the TMT-SRT ($F(1,171) = 5.25$, $p = .020$), while accuracy on the AST-PCT approached significance ($F(1,170) = 3.66$, $p = .057$). For the explorative outcomes, patients remained significantly slower on the TMT number sequencing ($F(1,171) = 10.20$, $p < .001$) and approached significance for the TMT letter sequencing ($F(1,171) = 3.62$, $p = .060$), while there were no group differences for the remaining outcomes. Full details of baseline performance are provided in Supplemental Table S2.

The supplementary MANCOVA controlling for IQ continued to show a significant group effect ($F(16,139) = 3.25$, $p < .001$).

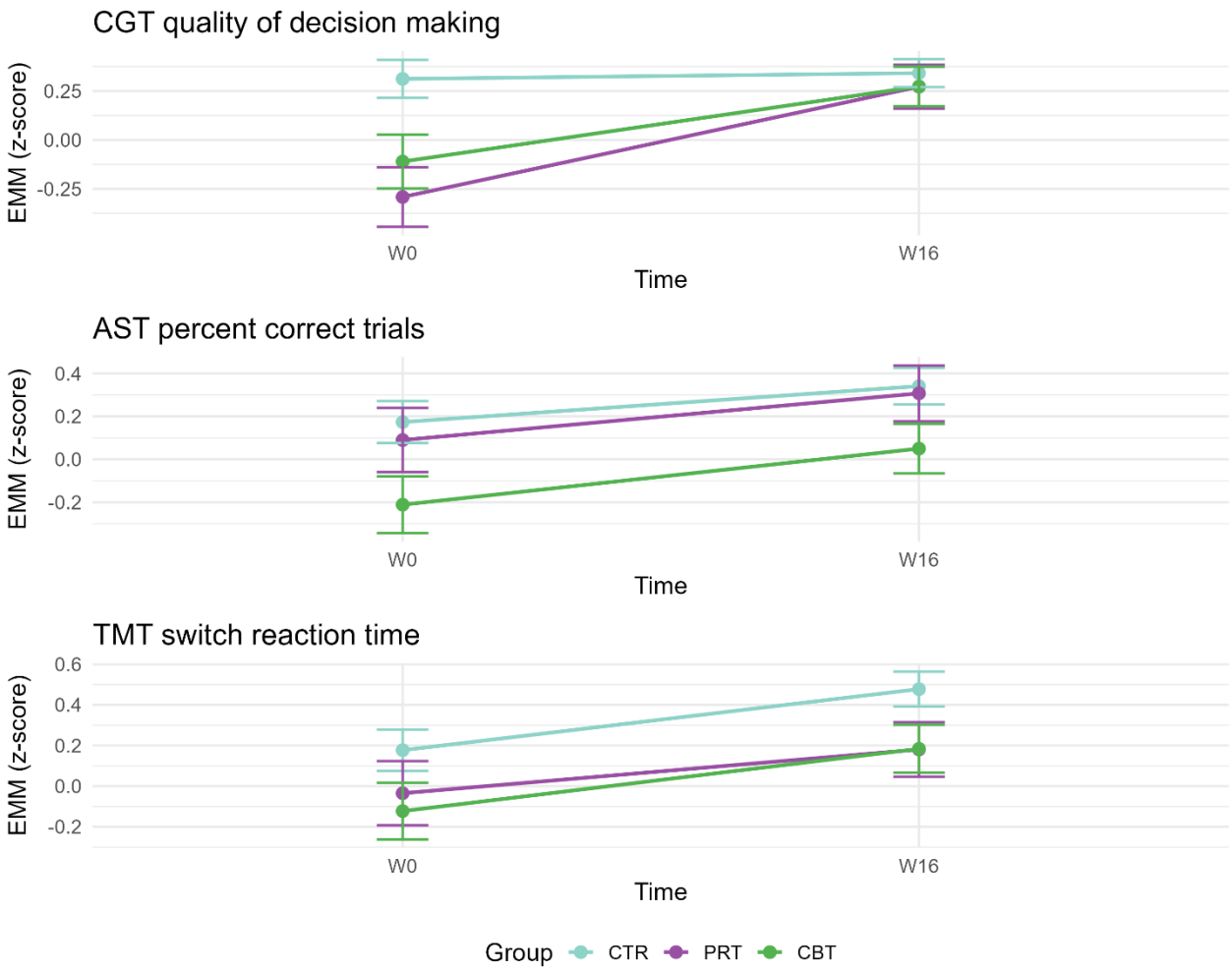
3.3 Change in neurocognitive performance

The repeated measures ANOVAs (Figure 1; Figure S1) showed a significant effect of Time on all primary outcomes (CGT-QDM: $F(1,161) = 23.61$, $p < .001$, ges = .036; AST-PCT: $F(1,167) = 16.86$, $p < .001$, ges = .014; TMT switch cost: $F(1,162) = 29.09$, $p < .001$, ges = .022). For CGT-QDM, there was also a significant Group by Time interaction ($F(2,161) = 6.56$, $p = .002$, ges = .020), reflecting significant improvement in patients (PRT: $p < .001$; CBT: $p = .002$) but not controls ($p = .73$). For the other two, the interactions were not significant ($p = .717$; $p = 753$). There was a significant effect of Group for CGT-QDM ($p = .025$) but not the remaining two ($p = .059$; $p = .089$). For details on assumption checks and sensitivity analyses using non-parametric testing, see supplementary material (including Figures S2-4).

Per-protocol analyses, restricted to participants who completed the full intervention (i.e., 10 out of 14 sessions), produced similar results for the main effects of Time ($p <.001$; $p < .001$; $p < .001$) and the Group × Time interaction ($p = .003$; $p = .717$; $p = .624$).

Post hoc analyses examined Pearson correlations among the primary outcomes across all participants. CGT-QDM showed weak to moderate correlations with the two flexibility measures ($r = 0.34$; $r = 0.22$), whereas two flexibility measures were strongly correlated ($r = 0.58$).

Figure 1 Change in primary neurocognitive outcomes



Note: CGT = Cambridge Gambling Task (from the CANTAB battery). AST = Attention switching task (from the CANTAB battery). TMT = Trail making task (from the D-KEFS battery). EMM = Estimated marginal mean. CTR = Control children. CBT = Patients who received cognitive-behavioral therapy (CBT). PRT = Patients who received psychoeducation and relaxation training (PRT). W0 = Week 0; baseline. W16 = W 16 (follow-up; end-of-treatment for patients with OCD).

It should be noted that a substantial proportion of individuals in the control group reached the maximum score of 100 % on the CGT-QDM at baseline (17.33 %). This may have limited the ability to detect further improvements or between-group differences (i.e., ceiling effects). However, the increase to 33.41 % at follow-up shows that an even larger proportion of the control group achieved the maximum score over time, indicating further improvement. For visual inspection, see Supplemental Figure S1.

Exploratory analyses of the remaining 13 neurocognitive outcomes showed that most improved significantly over time. Group by Time interactions were generally non-significant, except for TMT number sequencing RT, where patients (PRT and CBT) improved more than controls (p = .027; Figure S5).

Finally, no significant correlations were found between neurocognitive improvement and OCD symptom improvement for any neurocognitive outcome.

Post hoc analyses stated the percentage of children with OCD falling <1 SD below the control group mean at baseline. For CGT-QDM, the proportion of patients with scores below -1 went from: 20.93-25.71 % at baseline to 4.65-5.71 % at follow-up. For AST-PCT, the proportions changed from 8.10-17.02 % to 2.70-10.64 %, and for TMT-SRT, they changed from 14.29-20.00 % to 8.88-11.43. For full details, see Supplemental Table S4.

Supplementary analyses compared patients and controls at follow-up to determine whether differences reported at baseline were still present. The MANOVA across all 16 neurocognitive outcomes was still significant for group at follow-up ($F(16,132) = 1.746$, $p < .046$). On individual neurocognitive measures, patients still had slower reaction times on the TMT-SRT ($F(1,164) = 5.726$, $p = .018$). On the other hand, patients no longer displayed poorer CGT-QDM ($F(1,167) = 0.434$, $p = .511$), reduced accuracy on the AST-PCT ($F(1,170) = 2.313$, $p = .130$), and slower reaction time on the TMT number sequencing ($F(1,164) = 0.721$, $p = .397$) than controls.

3.4 Prediction and moderation

Three separate linear regression analyses examined whether baseline neurocognitive performance predicted follow-up CY-BOCS score (controlling for baseline CY-BOCS score) and moderated the effect of treatment group (PRT versus CBT) in patients with OCD. While the overall models were significant ($F(4,95) = 5.473$, $p < .001$, $R^2 = 0.19$; $F(4,98) = 6.141$, $p < .001$, $R^2 = 0.20$; $F(4,97) = 5.454$, $p < .001$, $R^2 = 0.18$), neither baseline neurocognitive performance or its interaction with treatment group (PRT versus CBT) significantly predicted OCD symptom improvement for any of the models. Instead, only CY-BOCS baseline and treatment group were significant predictors in all models (see Supplemental Tables S5-S7). Supplementary analyses restricted to completers (i.e., per protocol) showed similar results. Exploratory analyses of the remaining 13 neurocognitive outcomes showed similar non-significant results. Notably, for the Design Fluency switch condition (an accuracy-based measure of cognitive flexibility), the interaction between treatment group and baseline performance did not survive Bonferroni correction ($p = .037$). However, the pattern suggested a trend toward moderation, such that participants with higher baseline performance showed a greater benefit from CBT compared with PRT. For illustration, see Supplemental Figure S6.

4 Discussion

This study examined the extent to which two types of psychotherapy influenced neurocognitive functioning in pediatric patients with OCD, and whether pre-treatment neurocognitive performance predicted or moderated symptom improvement during treatment. Children with OCD showed improvements on one out of the three executive function (EF) measures that were atypical pre-treatment (Uhre et al., 2023) – decision-making. For this outcome, patients demonstrated improvement and reached the level of the control group, while controls stayed stable, suggesting that therapy may have benefits for this aspect of neurocognition. In contrast, for the two cognitive flexibility outcomes, patients and controls demonstrated similar gains, suggesting that therapy did not influence these domains. However, there were no differential effects between the CBT and PRT groups. Pre-treatment neurocognitive performance did not predict or moderate the degree of symptom improvement following therapy.

4.1 Change in neurocognitive performance after treatment

We first examined whether the two psychological interventions improved neurocognitive functions that were atypical at baseline (Uhre et al., 2023) – that is, decision-making and cognitive flexibility. We hypothesized that these functions would improve following both treatments, with greater gains for CBT relative to PRT, and greater improvement in patients compared with controls (i.e., controlling practice effects).

Initial analyses showed that the current follow-up subsample was largely comparable to the baseline sample described in Uhre et al. (2023) with respect to demographic and clinical characteristics as well as EF group differences before treatment.

One primary outcome – decision-making – improved more in patients than in controls (ges = .020) over the 16-week period, during which OCD patients received psychotherapy and controls were observed without intervention. Supplementary categorical analyses also indicated a substantial reduction in the proportion of patients scoring more than 1 SD below the control baseline mean following treatment. Controls showed no change on this measure. We considered whether this might reflect a ceiling effect in the control group. Although baseline scores indicated that controls still had some room for improvement, the measure may be less sensitive to detecting change in high-performing individuals, which limits our ability to draw firm conclusions about stability versus improvement in this group. Consequently, this result should be interpreted with appropriate caution. This is the first study to demonstrate improvement in decision-making performance following treatment in pediatric OCD. These findings extend previous pediatric reports of improvements in other EF domains such as planning and visual memory (Andrés et al., 2008; Huyser et al., 2010; van der Straten et al., 2018). The CGT-QDM assesses the proportion of trials where the participant chose the most likely outcome, thereby indexing the ability to make appropriate decisions based on outcome probabilities. In the

context of OCD treatment, patients may have strengthened their capacity to make informed decisions, contributing to better control over obsessions and compulsions. However, since improvement did not differ between CBT and PRT, the effect likely reflects non-specific therapeutic processes shared by both treatments. These may include externalizing OCD through cognitive restructuring, setting treatment goals, and engaging in active skill building. Cognitive control may also play a role in managing obsessions and compulsions during both relaxation exercises and ERP.

It should also be noted that some statistical assumptions were violated for this outcome, but however, non-parametric sensitivity analyses yielded consistent results, suggest that these findings are robust.

In contrast to CGT-QDM results, performance on the two remaining primary outcomes, measuring cognitive flexibility – the CANTAB Attention Switching Task, percent correct trials (AST-PCT) and the D-KEFS Trail Making Task, Switch reaction time (TMT-SRT) – improved similarly across patients and controls, contrary to our hypothesis. The comparable improvement suggests that these changes likely reflect time- or practice-related effects rather than treatment-specific effects. A similar pattern was observed for interference control in one previous pediatric OCD study (Huyser et al., 2011), whereas another found no neurocognitive improvement in either patients or controls (Hybel et al., 2017).

Across the 13 explorative outcomes, a significant effect of time was observed for all but three. Only processing speed, as measured by TMT number sequencing reaction time, showed significantly larger improvement in patients than in controls, while TMT letter sequencing reaction time showed a trend towards statistical significance. These results suggest that the treatment may have helped participants process information more quickly, likely because their overall mental and physical slowing (psychomotor retardation) improved. A similar result was found in adults with OCD after CBT, also using the TMT number sequencing (Voderholzer et al., 2013).

None of the primary outcomes improved differentially between CBT and PRT. Although prior studies have attributed neurocognitive improvements to CBT (Huyser et al., 2010; Katz et al., 2024; Kuelz et al., 2006; Moritz et al., 1999; van der Straten et al., 2018; Voderholzer et al., 2013; Wheaton et al., 2025), the present study is the first study (in adult *and* pediatric literature) to assess neurocognition during CBT compared with a structurally matched active control intervention. Contrary to our hypotheses, no differential treatment effects were found, suggesting that patient improvements in decision-making (and the exploratory processing speed) may reflect non-specific effects of participating in a highly structured therapeutic program rather than CBT-specific mechanisms. Indeed, the interventions do share some therapeutic factors – e.g., both comprise goal-setting, self-reflection, and therapist support. Notably, we expected neurocognitive improvement to be correlated with reductions in OCD symptoms and anticipated that CBT would enhance cognitive outcomes more strongly, parallelling its effects on symptoms, in line with the CSTC model of OCD, hypothesizing direct links between symptomatology and cognitive functioning. However, supplementary analyses indicated that

changes in neurocognitive performance were not correlated with changes in symptom severity, as measured by the CY-BOCS total score, on any neurocognitive measure. Altogether, these findings suggest that neurocognitive gains observed during psychotherapy for OCD may not be specific to the ERP and cognitive restructuring components of CBT.

4.2 Prediction and moderation

Finally, we examined whether baseline neurocognitive functioning was associated with the extent of symptom improvement in either intervention, testing for potential predictive and/or moderating effects. None of the primary outcomes in decision-making and cognitive flexibility that appeared atypical at baseline (CGT-QDM, AST-PCT, and TMT-SRT) predicted or moderated treatment outcome. This finding is contrary to our hypothesis and to two prior pediatric studies that have linked baseline cognitive function to CBT response in OCD; Flessner et al. (2010) reported that poorer visuo-perceptual memory and organizational strategy predicted poorer CBT outcomes, whereas Hybel et al. (2017) found that poorer baseline EF predicted *greater* symptom improvement. In contrast, the present study found no such evidence. The same pattern was seen for all but one of the 13 exploratory measures. For one explorative measure of cognitive flexibility, there was a non-significant trend suggesting that poorer baseline performance was associated with smaller treatment gains in CBT, but not in PRT. A possible interpretation could be that cognitive flexibility is recruited in CBT and thus a certain level is an advantage. However, this outcome was explorative, and we did not see similar effects on the remaining measures of cognitive flexibility. Altogether, although we had hoped to identify neurocognitive profiles that might inform individualized treatment adaption, our findings did not support such applications at this stage.

4.3 Strengths and limitations

The study has several limitations that may have influenced our results and interpretations. First, assessing neurocognitive functioning, especially EF, in highly structured test situations may not reflect neurocognitive difficulties experienced in real-life (i.e., ecological validity may be questioned). We investigate this in a separate study, assessing daily-life EF with the Behavior Rating Inventory of Executive Function, Second Edition (BRIEF-2) questionnaire (Ritter et al., in review); interestingly, children and their parents identify difficulties across most BRIEF-2 domains. Second, practice effects on repeated neurocognitive testing influenced observed improvements in test performance. We used the same tests twice which may have increased practice effects. The inclusion of a healthy control group allowed us to account for such nonspecific gains, but future studies may consider using parallel instead of similar tests at follow-up to minimize the influence of practice effects. Also, ceiling effects in the control group on one primary outcome (decision-making) may have limited our ability to detect further improvement during the assessment period and explain why patients appear to improve more than controls. Supplementary descriptive analyses showed that individual

improvement took place in the control group although it was not visible at a group level. Future studies assessing decision-making should use tasks more suited for preventing ceiling effects and allow room for measurable change. Third, it should be noted that the group differences identified between patients and controls at baseline (in the follow-up sample) were only small to medium (η^2 = .03-.07). Notably, clinically relevant impairment of neurocognitive outcomes is generally defined as 2 SD below the normative mean (Abramovitch et al., 2015), but however, only a few patients in our sample scored that low, and additionally, comparing patients to a very well-functioning healthy control group could have further inflated the effects. Following this, only little improvement relative to controls could be expected, and the clinical relevance of such effects could be questioned. Indeed, the interactions observed were of small effects (ges = .01-.02). Also, it is possible that our tests were not sensitive enough to detect potential interactions among remaining outcomes this subtle. Finally, we acknowledge that excluding all TECTO-participants with incomplete neurocognitive data may have introduced selection bias, if dropout from assessments is related to treatment or outcome, e.g., drop-out from treatment, potentially due to limited treatment effect. Thus, our findings may best apply to participants who completed most treatment sessions and research assessments but not necessarily to all children with OCD.

Despite limitations and small group-level effects, there is still a call to improve understanding of how neurocognitive functioning is implicated in pediatric OCD, considering the more robust findings in adults with OCD and individual-level findings of difficulties in a subset of children. A design like the present study is much suited for such examination; including two randomized treatment groups and a matched non-psychiatric control group assessed longitudinally is helpful for isolating the effects of OCD and psychotherapy. Future studies may consider following patients over a longer time span additionally, and finally, the exploratory finding of improved processing speed among patients relative to controls may be worth testing in future studies.

5 Conclusion

Our findings suggest that psychotherapeutic interventions confer modest, selective neurocognitive benefits in pediatric OCD. As the first pediatric study, we demonstrate improvement of decision-making (as hypothesized) and in processing speed (explorative) following psychotherapy for OCD. However, the absence of differential effects between CBT and the active control intervention indicates that these neurocognitive gains may reflect non-specific psychotherapy factors. Moreover, the limited scope of the observed effects and their lack of association with symptom improvement raise important questions about their clinical relevance and, more broadly, about the extent to which CSTC-related processes play a central role in pediatric OCD. Future studies may benefit from replicating these findings with refined methodological approaches.

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Supplementary material: Neurocognitive correlates of psychotherapeutic treatment response in pediatric obsessive-compulsive disorder: Results from the TECTO trial

Index

1 Aims

1.1 Mediation effects

We initially planned to examine, whether changes in neurocognition mediated the effect of treatment (PRT and/or CBT) on OCD symptom severity. Given the absence of a significant association between change in neurocognitive performance and change in symptoms (see Supplemental section 2.4), we instead report simple correlations between these change scores and did not continue with any formal mediation analysis.

2 Results

2.1 Available data/Missing data

Table S1 Overview of *N* (%) available data per outcome, group, and time point

Outcome	Group	Total n	Week 0	Week 16	Both week 0 and 16
			N (%)	N (%)	N (%)
Primary outcomes					
CGT quality of decision making	CTR	87	86 (98.9)	87 (100.0)	86 (98.9)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	46 (93.9)	45 (91.8)	43 (87.8)
AST percent correct trials	CTR	87	86 (98.9)	87 (100.0)	86 (98.9)
	PRT	41	38 (92.7)	38 (92.7)	37 (90.2)
	CBT	49	48 (98.0)	47 (95.9)	47 (95.9)
TMT switch RT	CTR	87	87 (100.0)	85 (97.7)	85 (97.7)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	48 (98.0)	45 (91.8)	45 (91.8)
Exploratory outcomes					
AST switch cost	CTR	87	86 (98.9)	87 (100.0)	86 (98.9)
	PRT	41	38 (92.7)	38 (92.7)	37 (90.2)
	CBT	49	48 (98.0)	47 (95.9)	47 (95.9)
VF correct switches	CTR	87	86 (98.9)	82 (94.3)	81 (93.1)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	48 (98.0)	45 (91.8)	45 (91.8)
Design fluency correct switches	CTR	87	87 (100.0)	85 (97.7)	85 (97.7)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	46 (93.9)	46 (93.9)	44 (89.8)
SOC prob. solved in min. moves	CTR	87	87 (100.0)	87 (100.0)	87 (100.0)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	47 (95.9)	46 (93.9)	45 (91.8)
SWM strategy	CTR	87	87 (100.0)	87 (100.0)	87 (100.0)
	PRT	41	38 (92.7)	37 (90.2)	36 (87.8)
	CBT	49	48 (98.0)	46 (93.9)	46 (93.9)
CGT risk adjustment	CTR	87	86 (98.9)	87 (100.0)	86 (98.9)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	46 (93.9)	45 (91.8)	43 (87.8)
Digit span raw score	CTR	87	84 (96.6)	81 (96.6)	81 (93.1)
	PRT	41	40 (97.6)	34 (82.9)	34 (82.9)
	CBT	49	44 (89.8)	42 (85.7)	37 (75.5)
SWM total errors	CTR	87	87 (100.0)	87 (100.0)	87 (100.0)
	PRT	41	38 (92.7)	37 (90.2)	36 (87.8)
	CBT	49	48 (98.0)	46 (93.9)	46 (93.9)
VF, correct words, phonemic	CTR	87	85 (97.7)	83 (95.4)	81 (93.1)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	48 (98.0)	45 (91.8)	45 (91.8)
VF, correct words, semantic	CTR	87	86 (98.9)	83 (95.4)	82 (94.3)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	48 (98.0)	45 (91.8)	45 (91.8)
DF correct figures, white	CTR	87	87 (100.0)	86 (98.9)	86 (98.9)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	47 (95.9)	46 (93.9)	45 (91.8)
TMT number sequencing RT	CTR	87	87 (100.0)	85 (97.7)	85 (97.7)
	PRT	41	38 (92.7)	36 (87.8)	35 (85.4)
	CBT	49	48 (98.0)	45 (91.8)	45 (91.8)
TMT letter sequencing RT	CTR	87	87 (100.0)	85 (97.7)	85 (97.7)
	PRT	41	38 (92.7)	35 (85.4)	34 (82.9)
	CBT	49	48 (98.0)	45 (91.8)	45 (91.8)

Note: CGT = CANTAB Cambridge Gambling Task. AST = CANTAB Attention Switching Task. TMT = D-KEFS Trail Making Task. VF = D-KEFS Verbal Fluency. SOC = CANTAB Stockings Of Cambridge. SWM = CANAB Spatial Working Memory. DF = D-KEFS design fluency.

2.2 Baseline neurocognitive performance in follow-up sample (patients versus controls)

Table S2 Baseline neurocognitive performance in follow-up sample (raw-scores)

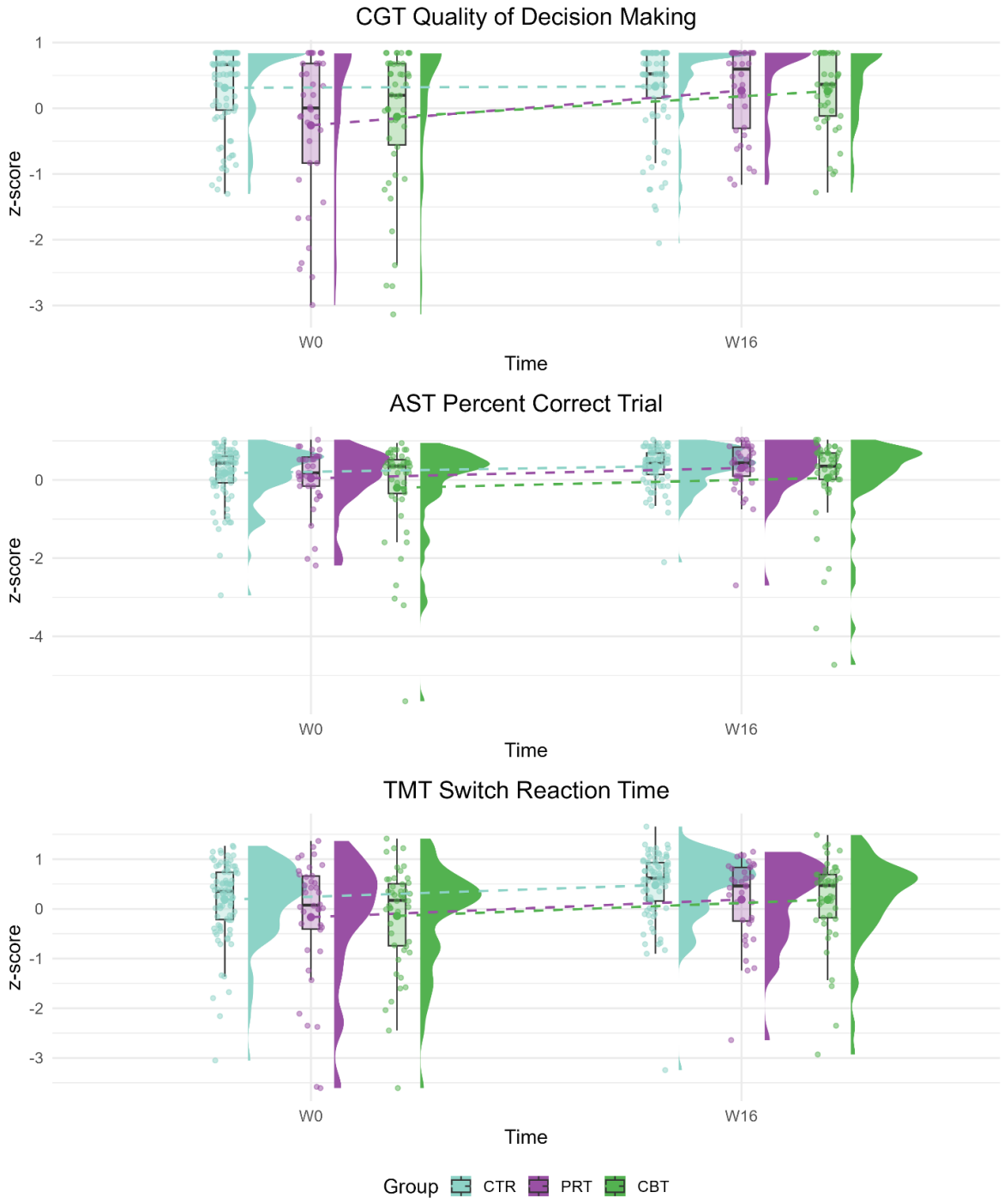
	CTR		*OCD*		*F*	*p*	η^2
	n	*Mean (SD)*	*n*	*Mean (SD)*			
Primary outcomes							
CGT quality of decision making	86	0.95 (0.06)	84	0.90 (0.11)	13.25	**< .001**	.070
AST percent correct trials	86	93.69 (5.11)	86	91.72 (8.06)	3.66	.057	.020
TMT switch RT	87	82.94 (32.48)	86	96.93 (46.66)	5.25	**.020**	.030
Exploratory outcomes							
AST switch cost	86	312.02 (135.39)	86	279.29 (141.63)	2.40	.120	.010
VF correct switches	86	11.20 (2.91)	86	11.63 (3.06)	0.89	.350	.010
Design fluency correct switches	87	7.29 (2.75)	84	6.90 (2.95)	0.77	.380	.000
SOC prob. solved in min. moves	87	7.53 (2.99)	85	7.98 (2.21)	1.24	.270	.010
SWM strategy	87	31.13 (5.24)	86	32.23 (5.96)	1.68	.200	.010
CGT risk adjustment	86	1.25 (0.79)	84	1.09 (0.97)	1.45	.230	.010
Digit span raw score	84	25.90 (4.11)	84	25.07 (4.93)	1.41	.240	.010
SWM total errors	87	22.61 (16.96)	86	26.13 (17.29)	1.82	.180	.010
VF, correct words, phonemic	85	25.58 (9.58)	86	27.99 (10.40)	2.49	.120	.010
VF, correct words, semantic	86	37.47 (8.98)	86	39.23 (11.14)	1.31	.250	.010
DF correct figures, white	87	9.61 (2.94)	85	9.98 (3.12)	0.63	.430	.000
TMT number sequencing RT	87	31.18 (13.06)	86	39.22 (19.45)	10.20	**< .001**	.060
TMT letter sequencing RT	87	34.47 (19.11)	86	40.05 (19.42)	3.62	.060	.020

Note: CTR = Control children. CBT = Patients who received cognitive-behavioral therapy (CBT). CGT = CANTAB Cambridge Gambling Task. AST = CANTAB Attention Switching Task. TMT = D-KEFS Trail Making Task. VF = D-KEFS Verbal Fluency. SOC = CANTAB Stockings Of Cambridge. SWM = CANAB Spatial Working Memory. DF = D-KEFS design fluency.

2.3 Change in neurocognitive performance

2.3.1 Detailed plots of change (primary outcomes)

Figure S1 Detailed plots (primary outcomes)



Note: CTR = Control children. CBT = Patients who received cognitive-behavioral therapy (CBT). CGT = CANTAB Cambridge Gambling Task. AST = CANTAB Attention Switching Task. TMT = D-KEFS Trail Making Task.

2.3.2 QQ plots (primary outcomes; residuals)

Figure S2 QQ Plot of CGT QDM residuals

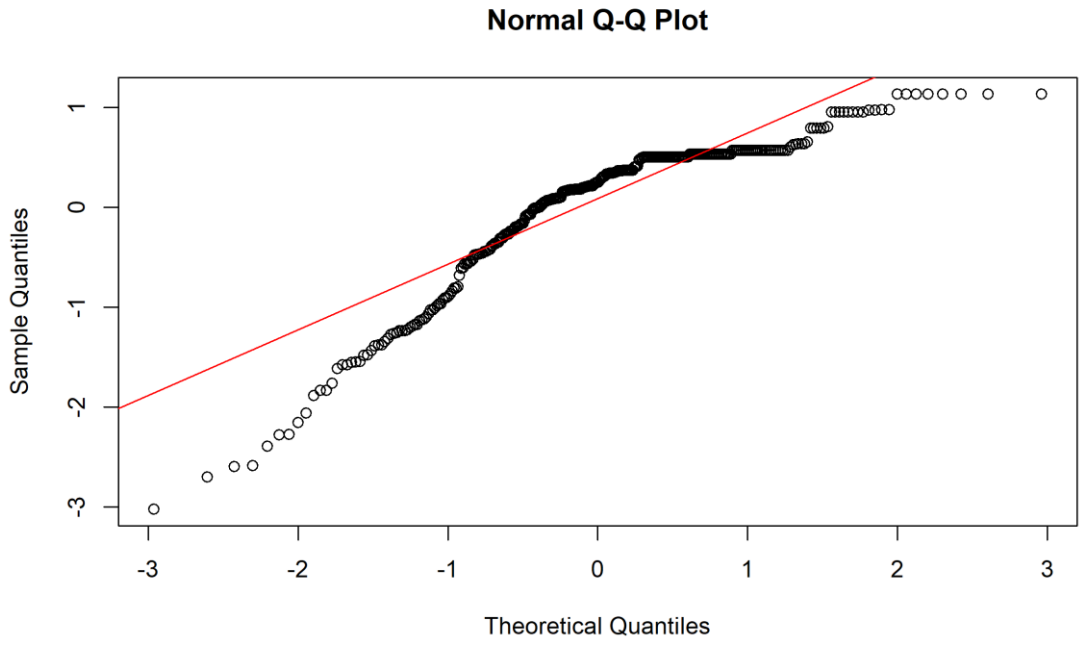


Figure S3 QQ Plot of AST-PCT residuals

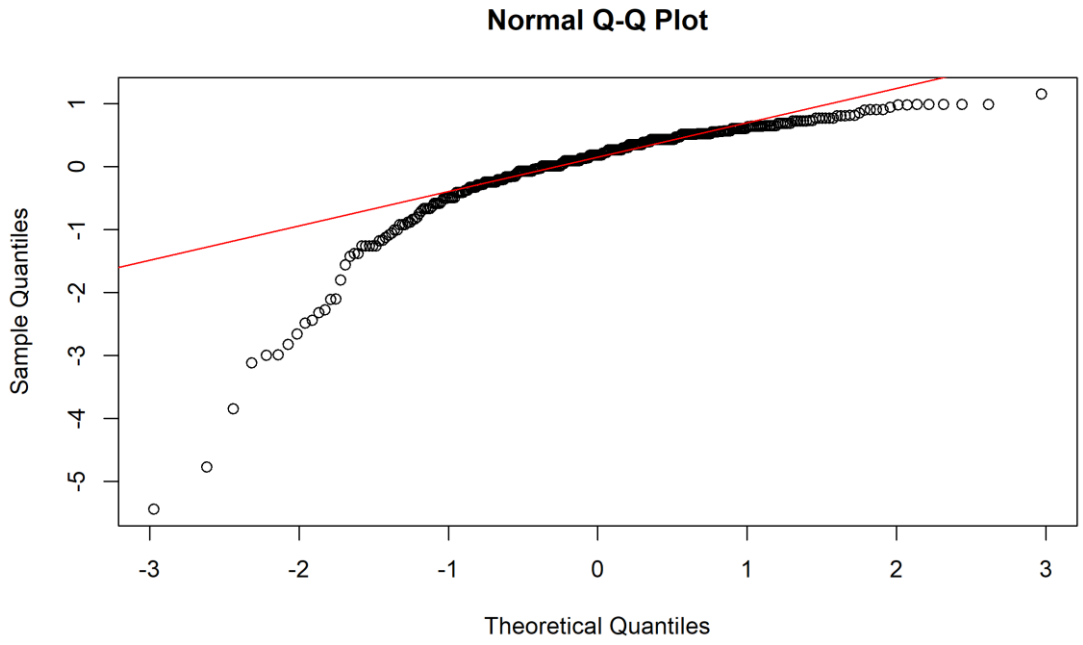
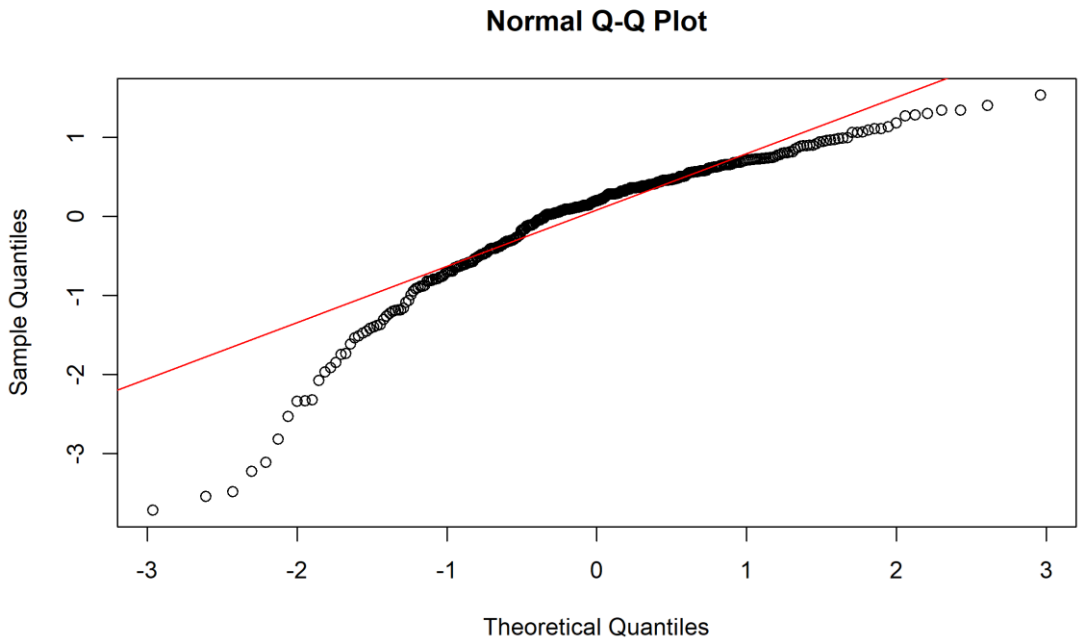


Figure S4 QQ Plot of TMT-SRT residuals



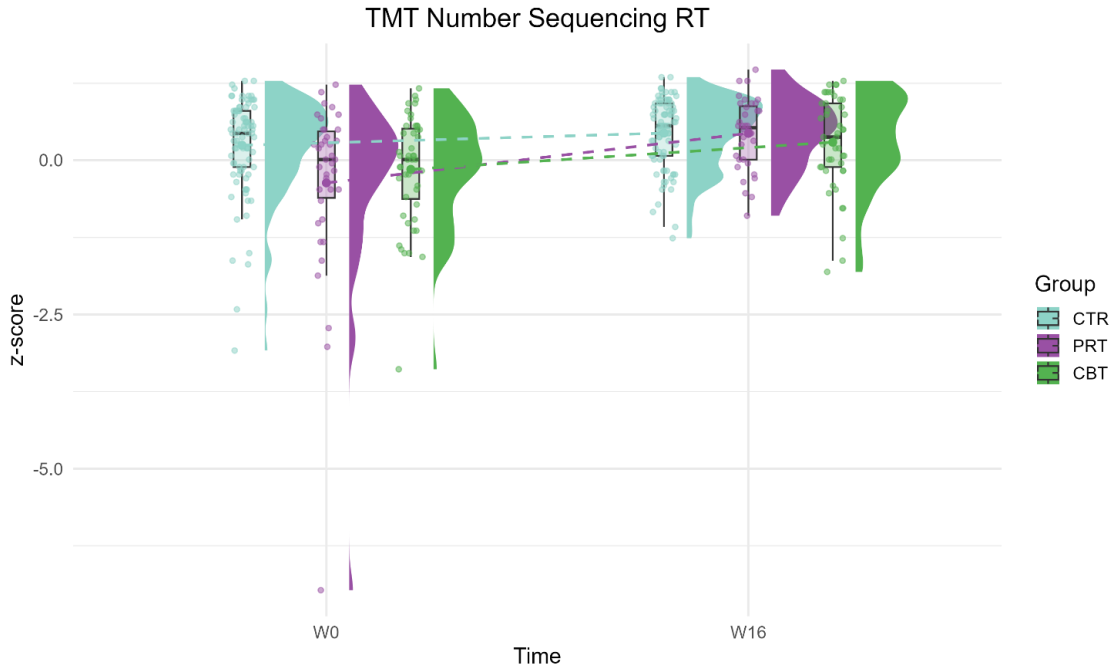
2.3.3 Non-parametric sensitivity analyses

Inspection of QQ plots and Shapiro-Wilk tests indicated that the residuals for the primary outcomes deviated from normal distribution. Visual inspection of the QQ plots (Figures S2-4) suggested deviations from normality (negative residuals were more extreme, and positive residuals were less extreme than expected), which was confirmed by the Shapiro-Wilk tests ($W = 0.868$, $p < .001$; $W = 0.769$, $p < .001$; $W = 0.888$, $p < .001$). Levene's tests indicated unequal variances across groups only for CGT QDM baseline ($F(2,167) = 5.69$, $p = .004$), while homogeneity of variance was satisfied for all other primary outcomes/timepoints.

To ensure robustness against violations of these assumptions, we conducted sensitivity analyses using the non-parametric aligned rank transform ANOVA for the three primary outcomes, which produced results consistent with the parametric analyses. The significant effect of Time on all three outcomes remained significant (CGT QDM: $F(1,161) = 3.560$, $p = .001$; AST-PCT: $F(1,167) = 16.564$, $p < .001$; TMT switch cost: $F(1,162) = 31.769$, $p < .001$) as well as the significant Group by Time interaction for CGT QDM ($F(2,161) = 6.96$, $p = .001$).

2.3.4 Detailed plot of change (Processing speed)

Figure S5 Detailed plot of change (Trail Making, Number Sequencing, RT)



Note: CTR = Control children. CBT = Patients who received cognitive-behavioral therapy (CBT). CGT = CANTAB Cambridge Gambling Task. AST = CANTAB Attention Switching Task. TMT = D-KEFS Trail Making Task.

2.3.5 2×3 Repeated measures ANOVA table

Table S3 Repeated measures ANOVA results for neurocognitive outcomes

Outcome	Effect	df	F	p	ges	Notes
Primary outcomes						
CGT quality of decision making	Group	2,161	3.76	**.025**	.034	
	Time	1,161	23.61	**<.001**	.036	
	Group × time	2,161	6.56	**.002**	.020	ΔCTR < ΔCBT & ΔPRT
AST percent correct trials	Group	2,167	2.88	.059	.029	
	Time	1,167	16.86	**<.001**	.014	
	Group × time	2,167	0.33	.717	<.001	
TMT switch RT	Group	2,162	2.46	.089	.026	
	Time	1,162	29.09	**<.001**	.022	
	Group × time	2,162	0.19	.753	<.001	
Exploratory outcomes						
AST switch cost	Group	2,167	1.01	.365	.009	
	Time	1,167	58.88	**<.001**	.080	
	Group × time	2,167	0.76	.468	.002	
VF correct switches	Group	2,158	2.54	.082	.018	
	Time	1,158	0.54	.464	.001	
	Group × time	2,158	2.02	.136	.011	
Design fluency correct switches	Group	2,161	0.45	.636	.005	
	Time	1,161	24.10	**<.001**	.072	
	Group × time	2,161	2.07	.130	.005	
SOC prob. solved in min. moves	Group	2,162	1.15	.320	.010	
	Time	1,164	6.58	**.011**	.012	
	Group × time	2,164	0.86	.423	.003	
SWM strategy	Group	2,166	0.98	.376	.010	
	Time	1,166	14.19	**<.001**	.014	
	Group × time	2,166	0.02	.978	<.001	
CGT risk adjustment	Group	2,161	0.56	.570	.006	
	Time	1,161	15.24	**<.001**	.015	
	Group × time	2,161	0.83	.440	.002	
Digit span raw score	Group	2,149	1.41	.247	.015	
	Time	1,149	1.41	.237	.002	
	Group × time	2,149	2.29	.105	.006	
SWM total errors	Group	2,166	0.67	.513	.007	
	Time	1,166	12.54	**<.001**	.012	
	Group × time	2,166	0.69	.504	.504	
VF, correct words, phonemic	Group	2,158	3.21	**.043**	.035	
	Time	1,158	16.10	**<.001**	.011	
	Group × time	2,158	0.73	.484	.001	
VF, correct words, semantic	Group	2,159	2.58	.079	.028	
	Time	1,159	0.11	.735	<.001	
	Group × time	2,159	0.70	.498	.001	
DF correct figures, white	Group	2,163	0.84	.432	.009	
	Time	1,163	14.80	**<.001**	.015	
	Group × time	2,161	0.48	.619	<.001	
TMT number sequencing RT	Group	2.162	2.71	.070	.025	
	Time	1,162	47.17	**<.001**	.063	
	Group × time	2,162	3.68	**.027**	.010	ΔCTR < ΔCBT & ΔPRT
TMT letter sequencing RT	Group	2,161	1.36	.259	.013	
	Time	1,161	29.80	**<.001**	.038	
	Group × time	2,161	0.52	.596	.001	

Note: CGT = CANTAB Cambridge Gambling Task. AST = CANTAB Attention Switching Task. TMT = D-KEFS Trail Making Task. VF = D-KEFS Verbal Fluency. SOC = CANTAB Stockings Of Cambridge. SWM = CANAB Spatial Working Memory. DF = D-KEFS design fluency. Ges = Generalized eta squared.

2.3.6 Proportion below 1 SD of control baseline mean

Table S4 Proportion below 1 SD of control baseline mean

Outcome	Group	Total n	< -1		% < -1	
			W0	W16	W0	W16
Primary outcomes						
CGT quality of decision making	CTR	86	4	7	4.65	8.13
	PRT	35	9	2	25.71	5.71
	CBT	43	9	2	20.93	4.65
AST percent correct trials	CTR	86	8	1	9.30	1.06
	PRT	37	3	1	8.10	2.70
	CBT	47	8	5	17.02	10.64
TMT switch RT	CTR	85	6	1	7.06	1.18
	PRT	35	5	4	14.29	11.43
	CBT	45	9	4	20.00	8.88
Exploratory outcomes						
AST switch cost	CTR	86	14	5	16.28	5.81
	PRT	37	5	3	13.51	8.11
	CBT	47	7	3	14.89	6.38
VF correct switches	CTR	81	16	24	19.75	29.63
	PRT	35	3	8	8.57	22.86
	CBT	45	9	11	20.00	24.44
Design fluency correct switches	CTR	85	13	13	15.29	15.29
	PRT	35	4	4	11.43	11.43
	CBT	44	9	3	20.45	6.82
SOC prob. solved in min. moves	CTR	87	15	12	17.24	13.79
	PRT	35	4	1	11.43	2.86
	CBT	45	8	3	17.78	6.67
SWM strategy	CTR	87	7	5	8.05	5.75
	PRT	36	4	4	11.11	11.11
	CBT	46	11	7	23.91	15.22
CGT risk adjustment	CTR	86	12	4	13.95	4.65
	PRT	35	7	3	20.00	8.57
	CBT	43	9	7	20.93	16.28
Digit span raw score	CTR	81	11	8	13.58	9.88
	PRT	34	5	4	14.71	11.76
	CBT	37	7	5	18.92	13.51
SWM total errors	CTR	87	12	5	13.79	5.75
	PRT	36	7	6	19.44	16.67
	CBT	46	12	4	20.09	8.70
VF, correct words, phonemic	CTR	81	13	15	16.05	18.52
	PRT	35	4	2	11.43	5.71
	CBT	45	4	3	8.89	6.67
VF, correct words, semantic	CTR	82	9	12	10.98	14.63
	PRT	35	2	2	5.71	5.71
	CBT	45	5	3	11.11	6.67
DF correct figures, white	CTR	86	10	8	11.63	9.30
	PRT	35	1	1	2.86	2.86
	CBT	45	6	6	13.33	13.33
TMT number sequencing RT	CTR	85	5	2	5.88	2.35
	PRT	35	6	0	17.14	0.00
	CBT	45	9	3	20.00	6.67
TMT letter sequencing RT	CTR	85	5	5	11.11	11.11
	PRT	34	3	0	8.82	0.00
	CBT	45	5	5	11.11	11.11

Note: CGT = CANTAB Cambridge Gambling Task. AST = CANTAB Attention Switching Task. TMT = D-KEFS Trail Making Task. VF = D-KEFS Verbal Fluency. SOC = CANTAB Stockings Of Cambridge. SWM = CANAB Spatial Working Memory. DF = D-KEFS design fluency. Ges = Generalized eta squared.

2.4 Mediation

There was no significant correlation between change in OCD symptoms during treatment and change in CGT quality of decision making (r = 0.10, 95% CI = [-0.23, 0.21], p = 0.934). Thus, cognitive improvement was not associated with symptom reduction. Given the lack of association, no formal mediation analyses were conducted.

2.5 Moderation

2.5.1 Linear regression tables

Supplemental Table S5 Linear Regression Results for CGT Quality of Decision Making

Predictor variable*	*β*	*p*
Intercept	4.34	.336
CY-BOCS pre-treatment	0.62	**<.001**
Treatment Group	-4.64	**.005**
CGT QDM*	0.96	.375
Treatment group × CGT QDM	-0.66	.648

Note: *) CANTAB CGT QDM = Cambridge Gambling Task, Quality of Decision Making, pre-treatment

Supplemental Table S6 Linear Regression Results for AST Percent Correct Trials

Predictor variable*	*β*	*p*
Intercept	3.74	.405
CY-BOCS pre-treatment	0.64	**<.001**
Treatment Group	-4.08	**.011**
AST-PCT*	1.26	.450
Treatment group × AST-PCT	-1.17	.228

Note: *) CANTAB Attention Switching Task, Percent Correct Trials, pre-treatment

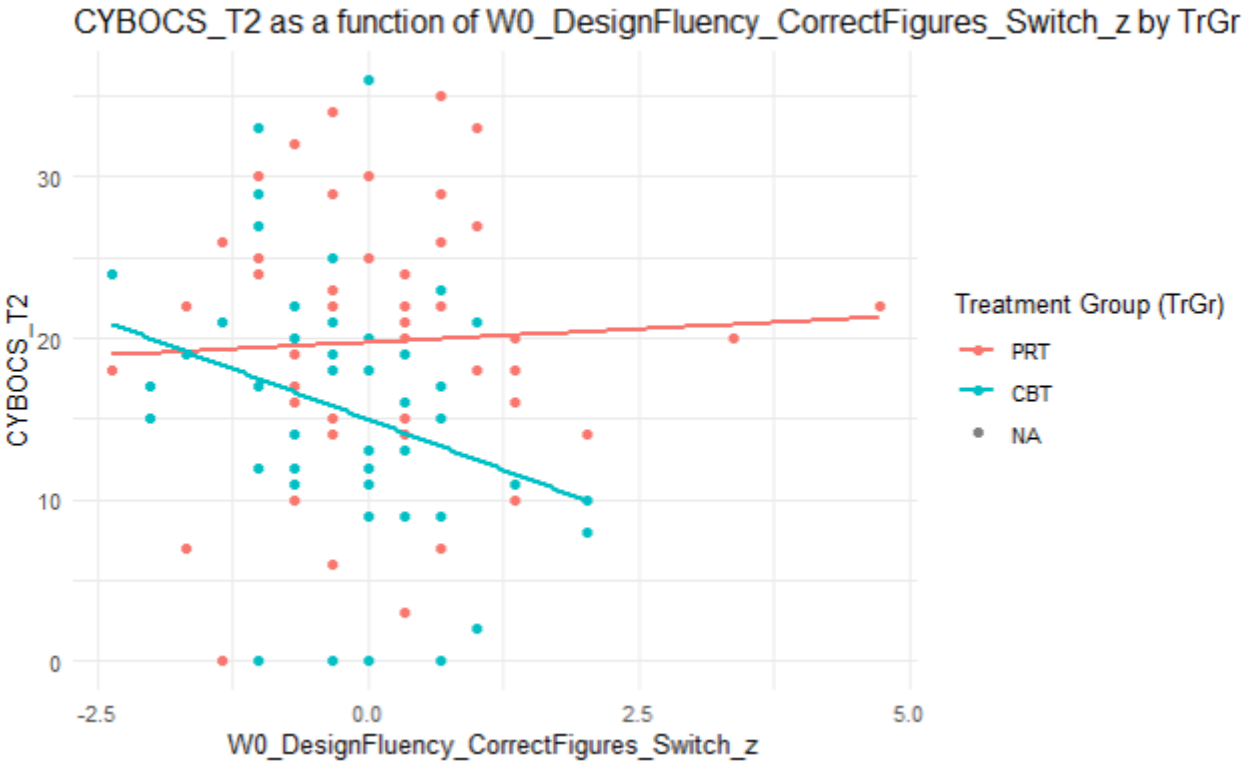
Supplemental Table S7 Linear Regression Results for TMT Switch Reaction Time

Predictor variable*	*β*	*p*
Intercept	4.32	.341
CY-BOCS pre-treatment	0.61	**<.001**
Treatment Group	-4.37	**.008**
TMT-SRT*	0.02	.986
Treatment group × TMT-SRT	0.25	.853

Note: *) TMT-SRT = D-KEFS Trail Making Task, Switch condition, reaction time, pre-treatment

2.5.2 Linear regression plot

Figure S6 Design Fluency, Switch, Correct Figures as a moderator of treatment outcome



Title: Understanding treatment response in pediatric obsessive-compulsive disorder: Executive function ratings from the TECTO trial

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Abstract

Objective: Executive function (EF) difficulties in pediatric obsessive-compulsive disorder (OCD) is suggested to interact with psychotherapy response. We investigated the role of parent- and self-rated EF in a randomized trial comparing cognitive-behavioral therapy (CBT) and psychoeducation with relaxation training (PRT). We investigated the extent to which: 1) EF difficulties are evident in non-medicated pediatric OCD patients; 2) difficulties improve during psychotherapy; and 3) EF moderates symptom alleviation.

Methods: We included 114 patients with OCD (aged 8-17 years), who received 14 sessions of CBT or PRT. EF was assessed before and after treatment with both the parent-rated (full age span) and self-rated (age 11-17 years) Behavior Rating Inventory of Executive Function, 2nd version (BRIEF-2). All BRIEF-2 subdomains were reported. OCD symptom severity was assessed with the Children's Yale-Brown Obsessive-Compulsive Scale (CY-BOCS). We assessed seventy-four matched non-psychiatric control children with a similar time interval.

Results: Parent- and self-ratings indicated EF difficulties across most BRIEF-2 domains in a subgroup of OCD patients. Self-rated flexibility difficulties were associated with more severe OCD symptoms pre-treatment. Parents and children reported improvement within most domains after psychotherapy. Parent-rated self-monitoring improved after CBT but not PRT. EF improvement was not associated with symptom alleviation in patients. Finally, emotional control, working memory, and task-completion ability moderated OCD-symptom improvement.

Conclusion: A sub-group of pediatric OCD patients show EF difficulties pre-treatment, which improve following psychotherapy with minimal differences between CBT and PRT. Although EF improvement is not associated with symptom alleviation, several domains of EF pre-treatment are moderators of treatment effect.

Keywords: OCD, Executive Function, Children, Psychotherapy, Cognitive Behavioral Therapy

1 Introduction

1.1 Obsessive-compulsive disorder in youth

Obsessive-compulsive disorder (OCD) is a debilitating psychiatric disorder that affects up to 3 % of children and adolescents worldwide (Abramowitz et al., 2009; Heyman et al., 2006). OCD is characterized by intrusive, distressing, recurring thoughts/images (obsessions) and repetitive behavioral or mental rituals (compulsions) (Cervin, 2023). Psychiatric comorbidity is common in pediatric OCD (63.6 %), with attention-deficit/hyperactivity disorder (ADHD), and tic disorders as some of the most common (Sharma et al., 2021).

1.2 Executive function and the corticocortico-striato-thalamo-cortical model of OCD

The cortico-striato-thalamo-cortical (CSTC) model of OCD (Brem et al., 2012; Milad & Rauch, 2012; Robbins et al., 2024), implicates executive function (EF) deficits and imbalances within CSTC brain circuits in OCD pathophysiology (Jalal et al., 2023). EF describes a set of interrelated, separable cognitive processes responsible for goal-directed behavior (Miyake et al., 2000). The exact hierarchy of functions continues to be debated but typically comprises specific subdomains, e.g., flexibility, working memory, and response inhibition (Friedman & Robbins, 2022). The processes manifest behaviorally and are typically measured with neuropsychological tasks (targeting EF capacity in specific tasks) and questionnaires, such as the Behavior-Rating Inventory of Executive Function (BRIEF) (Gioia et al., 2000, 2015, 2018), targeting everyday EF. Questionnaires and task results are generally weakly correlated, indicating an issue related to validity or that they represent distinct, complementary EF expressions (Snyder et al., 2021).

1.3 Executive function pre-treatment and association with symptom severity

Atypical EF is well documented in adults with OCD (Abreu et al., 2024; Snyder et al., 2015) and to some extent in children (Abramovitch et al., 2015; López-Hernández et al., 2022; Marzuki et al., 2020; Uhre et al., 2023), but associations with OCD symptom severity are inconsistent (Abramovitch et al., 2019; Geller et al., 2018; Negreiros et al., 2020; Rydqvist et al., 2023; Uhre et al., 2023). Questionnaire studies report more EF difficulties in children with OCD compared to controls, though not linked to symptom severity (Killion et al., 2024; Negreiros et al., 2020; Rydqvist et al., 2023; Zandt et al., 2009). To our knowledge, self-ratings in pediatric OCD have not been studied.

1.4 Psychotherapy for pediatric OCD

Family-based cognitive-behavioral therapy (CBT) with exposure and response prevention (ERP) is an effective psychological intervention and first-line treatment for OCD (Cervin et al., 2024; National Institute for Health

and Care Excellence (NICE), 2005; Reid J.E. et al., 2021; Uhre et al., 2020). The child practices setting goals and achieving them through cognitive restructuring and behavior modification (Piacentini et al., 2011). While effective for many, up to 40 % of children are non- or partial responders to CBT (Freeman et al., 2008, 2014; Pagsberg et al., 2025; Piacentini et al., 2011). The mechanisms underlying therapeutic effects need to be further identified and targeted to improve outcomes. Relaxation training (RT), often used as a control intervention and sometimes combined with psychoeducation (Freeman et al., 2008, 2014; Piacentini et al., 2011; Russman Block S. et al., 2023), is less effective but still reduces symptoms (Cervin et al., 2024; Pagsberg et al., 2025). In contrast to CBT, RT does not involve ERP, which could be viewed as EF-training due to the active skill building training cognitive and behavioral modification.

1.5 Executive function change after treatment and association with symptom change

Successful treatment of OCD is suggested to reduce EF difficulties, indicating EF as state-dependent (Hybel et al., 2017). Preliminary evidence suggests that test results of response inhibition, planning, and flexibility as well as BRIEF total score (General Executive Composite; GEC) improve after OCD treatment (Andrés et al., 2008; Huyser et al., 2010; Hybel et al., 2017) but associations with symptom alleviation are unclear.

1.6 Executive function as a moderator

EF deficits may also predict or moderate treatment response so that successful response to psychotherapy may depend on the individual's pre-treatment EF (Fitzgerald et al., 2021; Hybel et al., 2017; López-Hernández et al., 2022). Note that while a conceptual distinction can be made between predictors (main effects) or moderators (interaction with treatment type), we use the term "moderator" throughout for simplicity. In two studies, children with better parent-rated emotional control had less severe symptoms following CBT than children with poorer emotional control (McKenzie et al., 2020; McNamara et al., 2014) while others did not find an effect (Hybel et al., 2017; Killion et al., 2024; Rydqvist et al., 2023). Most prior work was open-label and included medicated patients, potentially masking a moderation effect on psychotherapy.

In summary, as recently noted (Jalal et al., 2023), longitudinal investigations of EF in pediatric OCD that include well-defined treatment- and control groups are needed.

1.7 Aims

The current study is the first to examine EF in pediatric OCD, via the BRIEF-2 (Gioia et al., 2018), through self- and parent-ratings. We use data from the Danish TECTO (Treatment Effects of Family-Based Cognitive Therapy in Children and Adolescents with Obsessive Compulsive Disorder) randomized clinical trial (RCT), comparing effects of family-based CBT versus family-based psychoeducation and relaxation training (PRT),

and data from a group of age- and sex-matched non-psychiatric control children (hereafter: controls) (Pagsberg et al., 2022, 2025).

Our aims were to examine the extent to which 1) EF difficulties are reported pre-treatment in patients relative to controls and whether EF difficulties in patients are associated with higher OCD symptom severity before treatment; 2) Psychological treatment is associated with alleviating EF difficulties in patients and whether this is associated with OCD symptom severity reduction; and 3) Pre-treatment EF in patients moderates treatment-related OCD symptom reduction. For Aims 2 and 3, analyses also examined differential effects for the two treatments (CBT versus PRT).

Our hypotheses were: Aim 1: (1.1) Greater EF difficulties will be reported in children with OCD pre-treatment compared with controls and (1.2) in patients greater EF difficulties will be associated with more severe OCD symptoms. Aim 2: (2.1) EF difficulties in children with OCD improve with psychotherapy, which is moderated by psychotherapy type with CBT outperforming PRT, and (2.2) Greater EF improvement will be associated with larger reductions in OCD symptoms. Aim 3: Pre-treatment EF difficulties moderate OCD symptom reduction after treatment. While our hypotheses were directional, we used two-sided tests to provide a more conservative evaluation of the effects.

2 Methods

2.1 Trial design

The study included participants from the Danish TECTO trial, which assesses benefits and harms of family-based CBT compared with an active control treatment (Family-Based Psychoeducation and Relaxation Training; PRT) in a superiority RCT design (ClinicalTrials.gov: NCT03595098). For a thorough description of study design and procedures, please see (Olsen M.H. et al., 2022; Pagsberg et al., 2022, 2025).

TECTO included 130 children and adolescents with OCD (age 8-17 years) and 90 control children, matched on age and sex, between July 2018 and July 2022. Diagnostic status (for patients and controls) according to the International Classification of Diseases 10th Revision (ICD-10) (World Health Organization, 1992) was based on transference of item criteria from assessments by the Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime version (K-SADS-PL) (Kaufman et al., 1997). Inclusion in the OCD group required a Children's Yale-Brown Obsessive-Compulsive Scale (CY-BOCS) (Scahill et al., 1997) total score of ≥ 16. Participants were excluded if they had an Intelligence Quotient (IQ) < 70 measured with age-appropriate Wechsler Scales (Wechsler, 2008, 2014). Patients were recruited from a single center in the Capital Region of Denmark and randomized to either family-based CBT or family-based PRT, both including up to 14 therapy sessions of 75 minutes each during a period of 16 weeks. Randomization was stratified by age (8-

12 and 13-17 years) and CY-BOCS total score before treatment (moderate: 16-23 and severe: 24-40). Controls were recruited via the Danish national register. Patients were assessed before the first therapy session (baseline; week 0) and after the last therapy session (end-of-treatment; week 16), and the matched control children were assessed with the same time interval.

2.2 Measures

2.2.1 OCD symptom severity

OCD symptom severity was assessed pre- and post-treatment with the clinician-rated semi-structured interview CY-BOCS (Scahill et al., 1997) total score. This score reflects frequency, impact, distress, resistance, and control of obsessions and compulsions on a scale from 0-40. The CY-BOCS was administered to the child and (in most cases) their parent by a trained clinician (medical doctor, psychologist, or psychology student under supervision).

2.2.2 Parent- and self-rated executive function

EF of the children was assessed with the BRIEF-2 questionnaires (Gioia et al., 2015, 2018) – both the parent-rated version (63 items) and the self-rated version (55 items – only children aged 11-18 years). The BRIEF-2 items describe problematic behavior related to EF demanding tasks in everyday life and are rated according to frequency ("Never", "Sometimes", or "Often"). The questionnaire produces nine subscale scores for parent-ratings and seven for self-ratings. The scales add up to three index scores as well as a total score (GEC), reflecting the total amount of EF-related problematic behavior (for details, see Supplementary Table S1).

In the current study, we were interested in examining EF subdomains. Thus, BRIEF-2 subscale (hereafter: subdomains) scores were included in our analyses but not BRIEF index scores or GEC. If an informant was missing two or more items on a given subdomain, that scale score was excluded for that informant, while remaining scale scores were retained, as recommended by Gioia et al. (2018). Scale results were standardized to T-scores with a linear transformation, based on normative samples with a mean of 50 and a standard deviation of 10, using the Hogrefe Test System (HTS 5) (Gioia et al., 2018). Higher scores indicate higher levels of EF difficulties. The BRIEF-2 scales have demonstrated good psychometric properties. Internal consistency is acceptable to high (Cronbach's α = .70 – .95) for all but two scales (initiating and self-monitoring) in Danish normative pediatric samples (age: 5-18 years) (Gioia et al., 2018).

2.3 Participants

We aimed to include all participants from the TECTO trial in the present study. We also included a control group to compare the OCD patients to a non-psychiatric matched group and to control for potential effects of repeated testing. Participants were included if at least one informant (child or parent) had reported BRIEF-2 at baseline. Thus, 114 children and adolescents (hereafter: children) with OCD and 74 control children were included in this study (for a full participant flow chart, see Supplementary Figures S1 and S2).

2.4 Data analysis

Primary and secondary analyses are described in this section. Additional details of the statistical analysis plan are provided in the supplementary material. Following the Benjamini-Hochberg correction procedure, we used critical values of 0.01281 for parent-ratings and 0.00567 for self-ratings (details are provided in the supplementary material).

2.4.1 Executive function pre-treatment and associations with symptom severity (Aim 1)

Aim 1.1: Group differences (OCD versus controls) in EF domains (BRIEF subdomains) before treatment were analyzed by two multivariate analyses of variance (MANOVAs) reporting Pillai's Trace statistics – one for parent-report and one for self-report BRIEF data. In case of significant results, follow-up univariate analyses of variance (ANOVAs) were performed on each individual subdomain. To test the robustness of the group comparisons, sensitivity analyses were conducted by 1) including IQ and parental education as covariates, 2) restricting the OCD sample to patients without any comorbidity, and 3) restricting the sample to patients without specific comorbidity (Asperger's, ADHD, generalized anxiety (GAD), and Tourette's syndrome).

Aim 1.2: Associations between executive function and symptom severity was examined via two multiple linear regression analyses calculated to predict symptom severity (CY-BOCS Total Score) in OCD patients, based on the BRIEF subdomains as potential predictors.

Post-hoc descriptive analyses reported, for each subdomain, the proportion of participants falling within clinically elevated categories: T scores ≥ 60 = Slightly elevated, ≥ 65 = Potentially elevated, and ≥ 70 = Clinically elevated (Gioia et al., 2018).

2.4.2 Executive function change after treatment and association with symptom change (Aim 2)

Aim 2.1: Change in EF during therapy was analyzed using two 2 (treatment group) × 2 (time) × 7 (BRIEF-2 domains) repeated measures analyses of variance (ANOVA) with BRIEF-2 T-Scores as the dependent variable. One ANOVA examined parent-rated data (note the parent-rated data will be analyzed via 2x2x9 ANOVA as two more domains are determined from the parent data). The second ANOVA examined child

self-rated data. Follow-up analyses report estimated marginal means (using the *emmeans* package in R) (Lenth et al., 2025) pre- and post-treatment for BRIEF-2 domains in the two treatment groups and the control group.

Aim 2.2: We examined whether change in OCD symptom severity was associated with change in EF scores via two multiple linear regression analyses – one for parent-ratings and one for child-ratings. The models predicted post-treatment symptom severity (CY-BOCS Total Score) in OCD patients, based on the change in BRIEF-2 domains and pre-treatment CY-BOCS as potential predictors.

Additional ANOVAs were conducted to determine whether any time related reductions in symptom severity reflected practice and/or developmental effects (i.e., whether improvement was particularly marked in patients relative to controls). We conducted a 2 (group) × 2 (time) × 7 (BRIEF-2 domain) repeated measures ANOVA for self-ratings and a similar 2x2x9 ANOVA for parent-ratings.

2.4.3 Executive function as a moderator (Aim 3)

Aim 3: We used a Baron and Kenny moderation analysis (Baron & Kenny, 1986) to examine if any BRIEF-2 domain pre-treatment moderated the relationship between treatment group and post-treatment symptom severity. We conducted two multiple linear regression analyses, calculated to predict post-treatment symptom severity (CY-BOCS). Fixed effects included pre-treatment CY-BOCS scores, treatment group (CBT versus PRT), all pre-treatment BRIEF-2 subdomains, and interaction terms between BRIEF-2 subdomain and treatment group. If the interaction term between the putative moderator and treatment group is significant there is a moderation effect (Jaccard & Turrisi, 2003).

BRIEF-2 subdomains, treatment group, and CY-BOCS pre-treatment were mean-centered to reduce potential multicollinearity between the predictors and their interaction terms (Jaccard & Turrisi, 2003). Thus, treatment groups (with almost the same N) were coded approximately as follows: PRT = -1 and CBT = 1.

3 Results

Mean time between BRIEF-2 assessments for parent-ratings was 17.6 weeks (13.0-31.4 range) in the OCD group and 13.3 weeks (12.0-22.6) in the control group. For child self-ratings, mean time between assessments was 17.4 weeks (range: 13.0-31.4) in the OCD group and 16.0 weeks (range: 12.0-19.6) in the control group.

3.1 Demographic and clinical characteristics

Demographic and clinical characteristics pre-treatment are presented in Table 1 for participants with complete data (similar numbers for patients with complete data at both time points; see Supplementary Table S2). Results are reported separately for parent- and self-rating samples, as these differ. Patients differed significantly from

controls in parental education and IQ (see Table 1). The treatment groups did not differ on any measured characteristic.

3.2 Executive function pre-treatment and associations with symptom severity (Aim 1)

Aim 1.1: The MANOVA analyses indicated a significant multivariate effect of group (OCD versus controls) on the combined BRIEF-2 subdomains for both parent-ratings (Pillai's Trace = 0.516, $F(9,173) = 20.477$, $p < .001$, $\eta_p{}^2 = 0.50$) and self-ratings (Pillai's Trace = 0.314, $F(7,126) = 8.235$, $p < .001$, $\eta_p{}^2 = 0.31$). Given the significant main effects of group, follow-up ANOVAs were performed to investigate the effect on each BRIEF-2 subdomain individually. Parent-rated data indicated a significant effect of group on all domains, and self-rated data revealed group effects for all but self-monitoring and working memory (Figure 1A and 1B; for more details, see Supplementary Tables S3 and S4). This pattern was robust across all sensitivity analyses (for more details, see Supplementary Tables S5-S7). Notably, while all patient mean scores fall within the normal range (up to T = 60) (Gioia et al., 2018) and control mean scores fall a bit below the normative mean of T = 50, a considerable proportion of patients, but not controls, fall above the normal range within each subdomain (Gioia et al., 2018) (Figure 1C-1F; for more details, see Supplementary Tables S8 and S9). Additionally, patients are more likely than controls to fall above the normal range on one or more domains at a time (Supplementary Table S10; Figures S7-9).

Aim 1.2: The multiple regression analyses (predicting symptom severity (CY-BOCS total score) in patients, based on BRIEF-2 subdomain) revealed a significant regression model for the self-ratings ($F(7,74) = 3.904$, $p = .001$, $R^2 = .201$) but not for parent-ratings ($F(9,100) = 2.026$, $p = .044$, $R^2 = .078$) data.

The self-rating model accounted for 20.1 % of the variance, and examination of the individual predictors indicated that pre-treatment flexibility was a significant predictor of pre-treatment CY-BOCS score ($\beta = 0.21$, $t(74) = 4.272$, $p < .001$) whereas the rest of the subdomains were not (see Supplementary Material, Table S11). Thus, patients who had a higher flexibility score on BRIEF-2 (i.e., more flexibility problems) showed a higher CY-BOCS total score at baseline (i.e., more severe OCD symptoms) than patients with lower flexibility scores.

3.3 Executive function change after treatment and association with symptom change (Aim 2)

Aim 2.1: The 2 (treatment group) × 2 (time) × 9 (BRIEF-2 domain) repeated measures ANOVA for parent-rated BRIEF-2 T-scores revealed significant main effects of time ($F(1, 67) = 25.336$; $p < 0.001$; $\eta_p^2 = 0.27$) and BRIEF-2 domain ($F(5, 334.79) = 8.313$; $p < 0.001$; $\eta_p^2 = 0.11$). The same was seen for the 2 × 2 × 7 repeated measures ANOVA for self-rated BRIEF-2 T-scores ($F(1, 43) = 21.762$; $p < 0.001$; $\eta_p^2 = 0.34$); $F(4.56, 195.96)$

= 7.39; p <0.001; η_p^2 = 0.15). Thus, both parents and children reported that EF improved after treatment and that there were significant differences in the T-scores of different BRIEF-2 domains (see Figure 2).

Follow-up analyses stated that, for parent-ratings, the time × BRIEF-2 domain interaction and the treatment group × time × BRIEF-2 domain interaction were significant (F(6.57, 439.95) = 2.596; p = 0.014; η_p^2 = 0.04, and (F(6.57, 439.95) = 2.452; p = .020; η_p^2 = 0.04, respectively). Thus, the treatment groups differed in improvement levels in EF across BRIEF-2 domains (for more details, see Supplementary Figure S10).

No other main effects or interactions were significant (for full details, see Supplementary Tables S12 and S13).

Follow-up analyses showed a significant treatment × time interaction for self-monitoring (F(1,67) = 6.08, p = .010), where CBT demonstrated a notable improvement in self-monitoring (t(67) = 3.661, p < .001) relative to PRT (t(67) = 0.037, p = .971).

Follow-up analyses estimating pre- and post-treatment scores for all BRIEF-2 domains in the two treatment groups as well as in the control group are found in Supplementary Tables S14-S17. EF remained stable in the control group while it improved in the OCD group (in both treatments) during the assessment period.

Post-hoc descriptive analyses reported the proportion of patients that fall within the clinically elevated categories (see Supplementary Figure S11).

Aim 2.2: The multiple linear regression analyses predicting change in symptom severity (CY-BOCS Total Score) from change in BRIEF-2 subdomains were not significant for neither parent- nor child rated data (F(9,59) = 0.978, p = .467; F(7,40) = 0.751, p = 0.631). Thus, symptom improvement was not predicted by change in executive function.

3.4 Executive function as a moderator (Aim 3)

Aim 3: The multiple regression analyses of CY-BOCS post-treatment (higher means more severe symptoms) revealed significant regression models for both parent-ratings (F(20,77) = 2.191, p = .008, R^2 = .363) and self-ratings (F(16,57) = 2.578, p = .0045, R^2 = .420). Thus, pre-treatment EF difficulties and treatment type explain a significant amount of variance in post-treatment symptom severity. Full regression results, including all predictors and interactions, are available in Supplementary Tables S18 and S19.

The parent-rating model accounted for 36.3 % of the variance. Follow-up analyses showed that pre-treatment working memory was a significant predictor (β = -0.60, t(85) = -3.189, p = .002), while there was no interaction with treatment group; children with more working memory pre-treatment difficulties showed greater benefits from psychotherapy (whether CBT or PRT). There was a significant interaction between treatment group and emotional control (β = -0.70, t(85) = -2.509, p = .014); emotional control moderates the effect of treatment on OCD so that more pre-treatment emotional control difficulties were associated with less severe OCD

symptoms following treatment with CBT but not PRT (due to the coding of treatment allocation; CBT ≈ 1 and PRT ≈ -1, a negative coefficient reflects a steeper negative slope for CBT).

The self-rating model accounted for 42.0 % of the variance in CY-BOCS post-treatment. Follow-up analyses showed that pre-treatment task-completion and working memory were significant predictors (β = 0.41, $t(57)$ = 3.126, p = .003; β = -0.35, $t(57)$ = -2.296, p = .025). Children reporting working memory pre-treatment difficulties showed greater benefits from psychotherapy (CBT or PRT), while children reporting greater pre-treatment task completion difficulties showed less benefit from treatment. Like for parent-ratings, there was a significant interaction between treatment group and emotional control (β = -0.66, $t(57)$ = -2.644, p = .011) as well as between treatment group and task completion (β = 0.53, $t(57)$ = 2.056, p = .044). Pre-treatment emotional control difficulties were associated with less severe OCD symptoms following CBT rather than PRT.

See Supplementary Figures S12-S17 for plots of the unique predictor effects, controlled for other variables in the models, including interaction plots.

4 Discussion

This study examined EF in pediatric OCD, focusing on pre-treatment difficulties, treatment-related change, and moderation of psychotherapy outcomes. The study included 8–17-year-old children, 114 with moderate to severe OCD and 74 healthy matched controls. Compared to controls, children with OCD showed EF difficulties, particularly in flexibility and emotional control, which improved following both CBT and PRT. EF improvements in patients after 14 weeks of psychotherapy were not associated with symptom reduction, but pre-treatment EF moderated treatment outcomes in distinct ways, highlighting potential value in tailoring interventions.

4.1 Executive function pre-treatment and associations with symptom severity (Aim 1)

EF skills are crucial for adaptive behavior in everyday life. In OCD, EF impairment is suggested to underlie difficulties in controlling obsessive thoughts and compulsive behavior (Jalal et al., 2023). Consistent with prior studies (Hybel et al., 2017; Negreiros et al., 2020; Rydqvist et al., 2023; Zandt et al., 2009), children with OCD in our study displayed EF difficulties relative to controls, particularly in flexibility, emotional control, and task completion. Self-rated flexibility was modestly associated with OCD symptom severity, aligning partially with adult data and suggesting a potential role for flexibility in OCD pathology (Abramovitch et al., 2019). However, it should be noted that most previous work with pediatric and adult samples with OCD has found little evidence for such an association (Geller et al., 2018; Negreiros et al., 2020; Rydqvist et al., 2023). Indeed,

in our previous work with this sample using neuropsychological tests, underperformance in EF was also observed in OCD-patients but not associated with severity of OCD symptoms (Uhre et al., 2023).

4.2 Executive function change after treatment and association with symptom change (Aim 2)

Prior, albeit limited, studies have examined the impact of psychotherapy for pediatric OCD on EF. Neuropsychological work has indicated that measures of inhibition, flexibility, and planning improved from pre- to post treatment (Andrés et al., 2008; Huyser et al., 2010). Additionally, parent-rated BRIEF Global Executive Composite was reported to improve after CBT (Hybel et al., 2017). Our results are broadly consistent with this previous literature; parents of patients reported improvement in all BRIEF-2 domains following psychotherapy (whether CBT or PRT), and the patients themselves reported improvement in several domains (whether CBT or PRT). Importantly, we extend this literature in several ways. First, by incorporating individual BRIEF-2 subdomains into our statistical model, we could determine that most domains improve as a function of psychotherapy (whether CBT or PRT). This is seen whether EF is indexed via parent- or self-ratings. Notably, EF in the matched control group did not significantly change over the treatment interval whether indexed by parent- or self-ratings (which was also seen in the American norm material (Gioia et al., 2015); i.e., the results cannot be attributed to repeat testing (see Supplementary Tables S13 and S14). Second, parents reported differing improvement levels across subdomains, with the largest improvement seen for flexibility and emotional control as well as inhibition and planning/organizing. It should be noted that the largest effect sizes for self-rated data were also seen within these domains. As such, our results are consistent with previous work in both children (Andrés et al., 2008; Huyser et al., 2010) and adults (Kuelz et al., 2006; Moritz et al., 1999; Voderholzer et al., 2013) reporting improvement. Third, we observed a significant treatment group × time × BRIEF-2 domain interaction for the parent rated data, i.e., there were significantly different improvement levels of BRIEF-2 subdomains as a function of treatment group. Follow-up results revealed that this particularly reflected a notable improvement in self-monitoring following CBT, while no improvement was seen for PRT on this subdomain.

The self-monitoring domain measures the ability to evaluate own behaviors/skills and how these affect other people (Gioia et al., 2018). It is conceivable that CBT has a greater impact on self-monitoring than PRT due to the ERP component, which trains awareness of thinking and behavior, and how to adjust them. However, given the exploratory nature of our follow up tests, this result should be treated with caution.

Several features should be noted with respect to the large improvement in flexibility, emotional control, inhibition, and planning/organizing domains. Both flexibility and inhibition difficulties are putative key features of OCD that might improve with treatment (Jalal et al., 2023). It is suggested that if patients train their ability to inhibit unwanted thoughts/behavior and flexibly update this with appropriate alternate solutions during CBT, this might mediate reduction in OCD symptoms. Two pediatric studies in OCD have identified

improvement of neuropsychological test-performance on flexibility, inhibition, and planning tasks after CBT (Andrés et al., 2008; Huyser et al., 2010), and some adult studies found the same for flexibility (Kuelz et al., 2006; Moritz et al., 1999; Voderholzer et al., 2013). Our study supports inhibition, flexibility, and planning/organizing improving after psychotherapy for OCD, but opposite to our expectations, this effect was not specific to CBT but could also be obtained through PRT. Moreover, both parents and children recognized significant improvement of the emotional control domain in our study. One aspect of emotional control, temper outburst, is observed to improve with CBT for pediatric OCD (Krebs et al., 2013). Additionally, a study from the TECTO trial, currently under review (Thoustrup et al., submitted), observed that child-rated emotion regulation improved after CBT, but not after PRT, when measured by the Difficulties in Emotion Regulation (DERS) scale. The current study supports improvement of emotional control during psychotherapy but did not see a specific effect for CBT. Notably, whereas the 8-item BRIEF-2 scale focuses on strong behavioral reactions (Gioia et al., 2018), the 36-item DERS might better capture psychological aspects of emotional control (such as acceptance and awareness of one's own emotions), which could explain the difference.

Change in EF following psychotherapy was not associated with the magnitude of symptom alleviation in OCD symptomatology. While inconsistent with our predictions, this result is in line with a previous study using parent-rated BRIEF scores (Hybel et al., 2017). In short, while psychotherapy improves EF in patients with OCD as well as alleviating their symptoms (especially CBT; Pagsberg et al., 2025), these impacts appear relatively independent of each other. These data suggest that while many patients with OCD experience EF-related difficulties that improve with psychotherapy, these problems may be secondary to the core pathophysiology driving OCD symptoms. Improvement in EF, however, may still enhance daily functioning and well-being, reflecting benefits beyond the trajectory of OCD symptom reduction.

4.3 Executive function as a moderator (Aim 3)

Our study extends prior literature (McNamara et al., 2014) by adding self-ratings and comparing non-medicated children in a parallel psychotherapy design, as suggested by Jalal et al. (2023). Our results suggest that several EF domains predict symptom severity post-treatment and differentiate CBT and PRT in distinct ways. Children with emotional control difficulties pre-treatment benefitted relatively more from CBT than PRT. This may suggest that children with emotional control difficulties pre-treatment may benefit relatively more from CBT compared to PRT, possibly because CBT with ERP helps children build skills for identifying and managing strong emotions, while PRT provides less support for handling such distress. Pre-treatment working memory difficulties predict less severe OCD symptoms following psychotherapy when looking at all patients (whether CBT or PRT). Thus, pre-treatment working memory difficulties may allow patients to benefit relatively more from treatment, and children with such difficulties seem to respond well to the intensive support and structure provided in psychotherapy. Finally, more difficulties with task-completion were associated with

more severe symptoms after treatment for all patients (regardless CBT or PRT). Children with task-completion problems struggle to complete tasks within a time frame or at a sufficient pace, and it is possible they also struggle to do the tasks (both in session and as homework) required for psychotherapy. Altogether, children with task-completion difficulties pre-treatment may require additional support to complete therapeutic tasks.

4.4 Strengths and limitations

Our study has numerous strengths. We provide one of the most complex descriptions of daily-life EF in pediatric OCD to date by using a multiple-informant approach, assessing multiple EF domains before and after two types of treatment, and compare a non-medicated sample to a thoroughly screened control group. Reporting specific EF domains allows our findings to be compared with the neuropsychological literature, which often focuses on individual EFs rather than overall capacity. The repeated measures in the control group demonstrate stability of EF over time, supporting the view that observed changes in patients are related to therapy effects. Finally, by using the BRIEF-2, we assessed EF in a real-life context, in contrast to task-based measures that capture momentary performance in a specific setting and may not reflect overall daily functioning.

Several limitations should be considered. First, the sample size for self-rated data was smaller than for parent-rated, mainly due to the age restrictions (> 11 years), and some attrition which occurred before follow-up. While the study was powered a priori for the primary outcome of the TECTO trial (CY-BOCS) rather than BRIEF-2, several moderate effects were detected for BRIEF-2 outcomes indicating that the available sample size still allowed us to detect effects of at least this magnitude while smaller effects may have gone undetected. Second, our study could have potential selection-biases deriving from which parents/children completed the BRIEF-2. If completion were dependent on, for example, symptom severity, comorbidity, or another variable, this might impact the generalizability of our results. Notably, the considerable patient drop-out rate (which was higher for PRT) could introduce bias. Third, as with all patient-reported outcomes, the validity of the BRIEF-2 relies on subjective reporting. To reduce potential rater bias, included multiple informants and instructed them to rate items broadly rather than focusing on specific (e.g., OCD) contexts. Fourth, the high rate of psychiatric comorbidity in our sample, reflecting the typical pediatric OCD population, made it challenging to isolate effects specific to OCD. Nonetheless, our findings remained robust in sensitivity analyses. Finally, our control group was highly functioning, as expected for children without mental illness. To provide a meaningful context, we reported T-scores and proportion of children above clinical thresholds (≥ 60, ≥ 65, ≥ 70) highlighting a substantial subgroup of children with OCD who have difficulties well beyond both controls and population norms.

5 Conclusion

This study is the first to assess patient-reported EF in pediatric OCD and the first to report longitudinal assessment of parent- and self-rated EF across two psychotherapies in non-medicated youth with OCD compared with a non-psychiatric control group. While the BRIEF-2 scores were reported to be within normal limits for most children with OCD, a considerable subgroup showed pronounced EF difficulties, particularly in flexibility and emotional control, which improved after treatment with either CBT or PRT. EF change was not associated with symptom improvement, but these EF gains may nonetheless enhance daily functioning and quality of life. Pre-treatment EF moderated treatment outcomes in distinct ways, highlighting EF ratings as a potential tool in tailoring interventions. Specifically, children with emotional control difficulties may benefit relatively more from CBT, those with working memory difficulties appear to respond well to structured interventions, and children with task-completion problems may require additional therapeutic support to complete their tasks during psychotherapy effectively. This study establishes that child-reported EF difficulties exist in pediatric OCD and, together with parent-report, reveal that pre-treatment EF profiles can provide valuable guidance for tailoring personalized interventions.

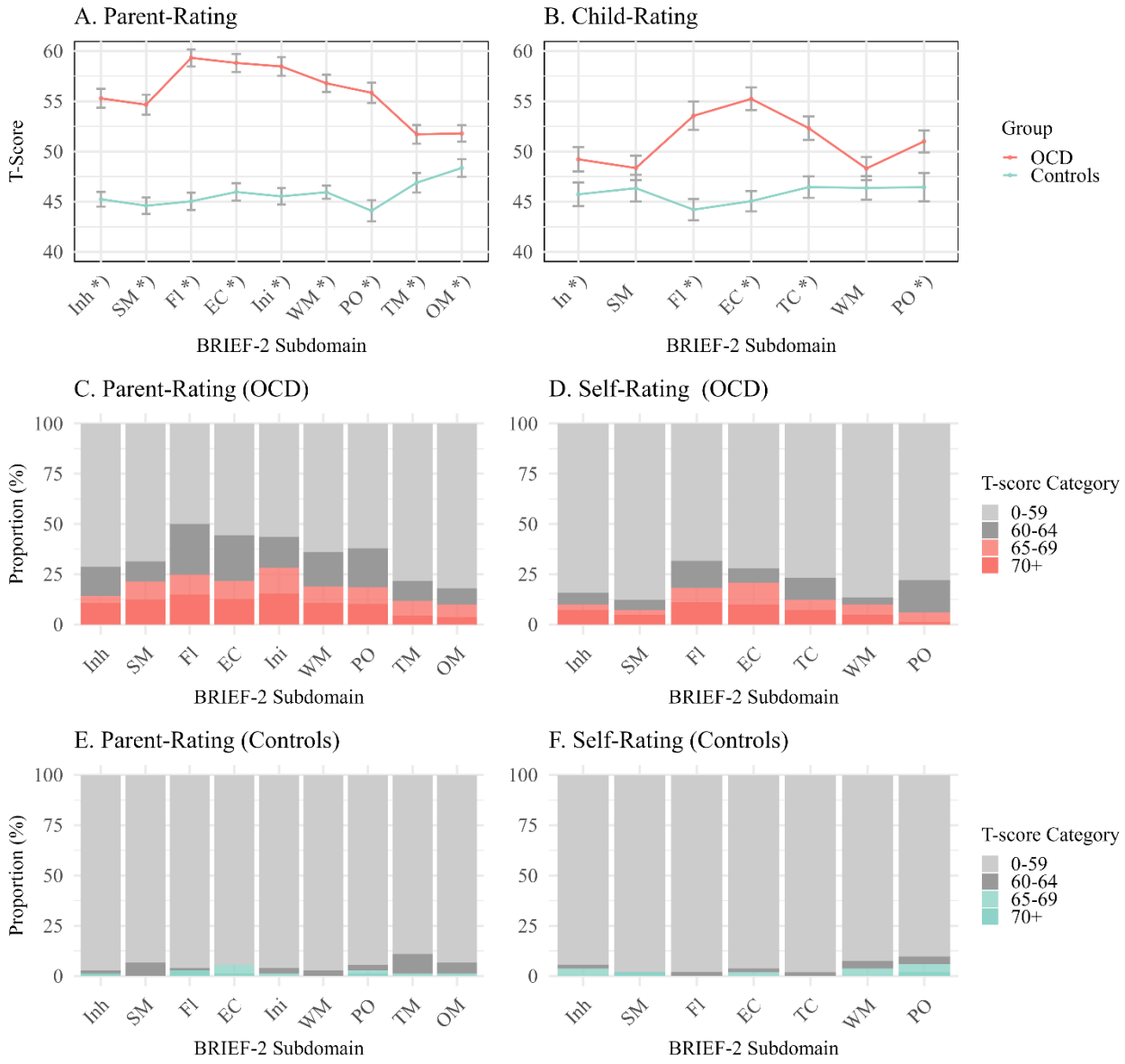
Tables and figures

Table 1. Demographic and Clinical Characteristics pre-treatment (Parent- and Self-Rating Samples)

	OCD	**CTR**	**OCD vs CTR**		**CBT**	**PRT**	**CBT vs PRT**	
	Mean(*SD*), *n*(%)	Mean(*SD*), *n*(%)	*p*; Effect size		Mean(*SD*), *n*(%)	Mean(*SD*), *n*(%)	*p*; Effect size	
Number of participants								
Parent-rating	112	74			54	58		
Self-ratings	82	52			43	39		
Number of females								
Parent-ratings	59 (52.7)	37 (50.0)	.835	.020	33.0 (61.1)	26 (44.8)	.125	.163
Self-ratings	49 (59.8)	31 (59.6)	1	.001	27.0 (62.8)	22 (56.4)	.716	.065
Age, years								
Parent-ratings	13.3 (2.88)	13.3 (2.74)	.985	.003	13.1 (2.94)	13.5 (2.82)	.481	.134
Self-ratings	14.7 (1.99)	14.8 (1.92)	.775	.050	14.4 (2.06)	15.0 (1.90)	.201	.284
Parental education, years								
Parent-ratings	15.6 (2.11)	16.7 (1.83)	**<.001**	.569	15.5 (2.2)	15.7 (1.77)	.607	.134
Self-ratings	15.5 (2.19)	16.8 (1.57)	**<.001**	.675	15.3 (2.5)	15.7 (1.80)	.443	174
Intelligence Quotient (IQ)								
Parent-ratings	99.2 (12.7)	105 (14.2)	**<.001**	.454	98.9 (12.0)	99.4 (13.5)	.850	.036
Self-ratings	98.9 (12.2)	104 (14.0)	**<.039**	.385	98.2 (11.8)	99.5 (12.8)	.648	.102
CY-BOCS Total Score								
Parent-ratings	25.2 (6.67)	-	-	-	25.1 (4.6)	25.8 (4.73)	.385	.165
Self-ratings	25.2 (4.53)	-	-	-	25.1 (4.6)	25.4 (4.48)	736	.075
Comorbid disorders								
Asperger's (F84.5)								
Parent-ratings	15 (13.4)	0 (0)	-	-	3 (8.11)	6 (18.75)	.129	.170
Self-ratings	9 (11.0)	0 (0)	-	-	2 (7.14)	4 (19.05)	.116	.212
ADHD (F90.0)								
Parent-ratings	12 (10.7)	0 (0)	-	-	5 (13.51)	4 (12.50)	.861	.045
Self-ratings	5 (6.1)	0 (0)	-	-	2 (7.14)	1 (4.67)	1	.039
GAD (F41.1 & F93.8)								
Parent-ratings	9 (8.0)	0 (0)	-	-	1 (2.70)	4 (12.50)	.201	.154
Self-ratings	7 (8.5)	0 (0)	-	-	0 (0)	3 (14.29)	.086	.233
Tourette's (F95.2)								
Parent-ratings	7 (6.3)	0 (0)	-	-	2 (5.41)	2 (6.25)	.494	.102
Self-ratings	4 (4.9)	0 (0)	-	-	1 (3.57)	2 (9.52)	1	.011
Any comorbid disorder								
Parent-ratings	64 (57.1)	0 (0)	-	-	20 (54.05)	18 (56.25)	.201	.154
Self-ratings	41 (50.0)	0 (0)	-	-	13 (46.43)	11 (52.38)	.086	.233

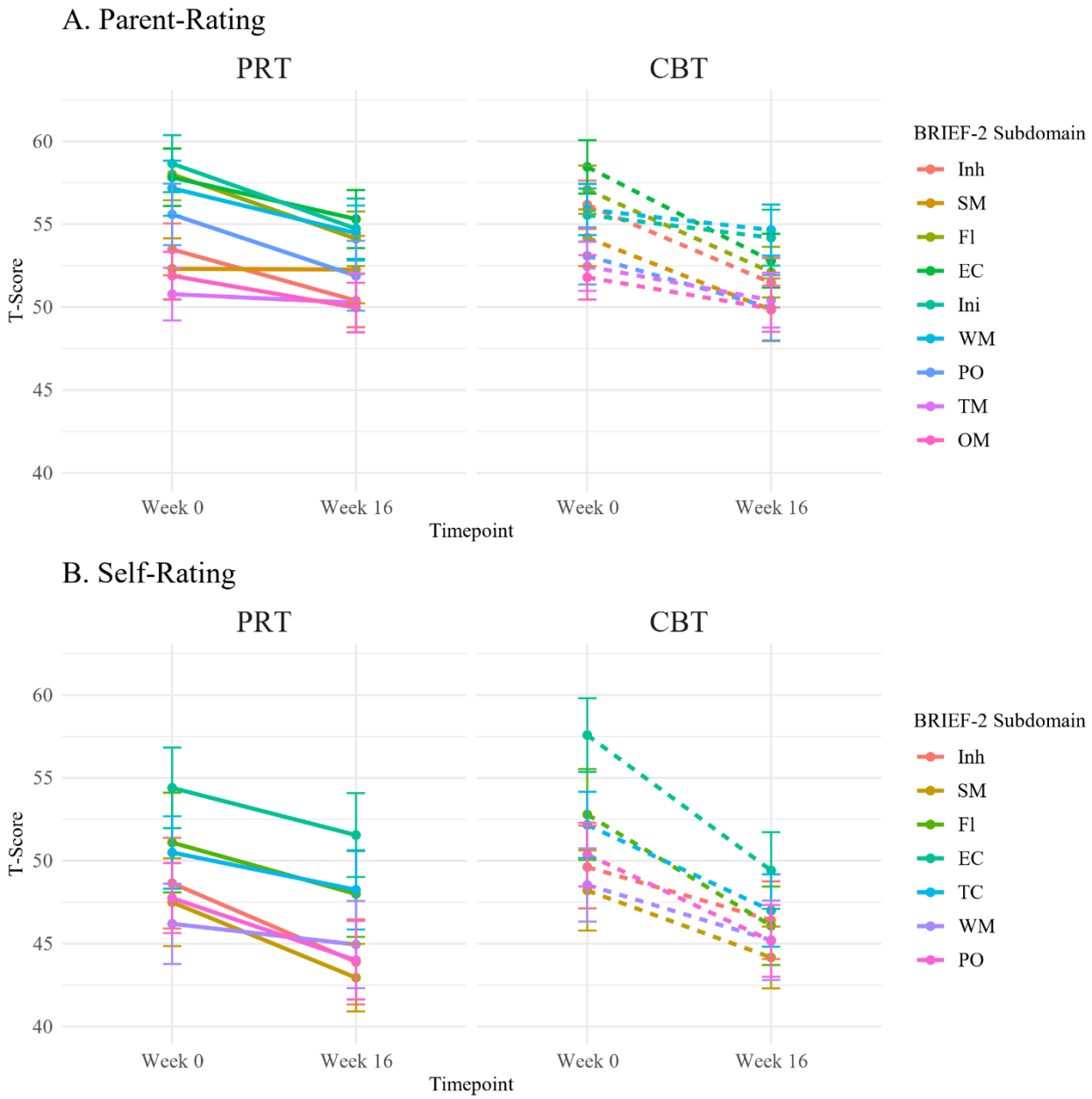
Note: OCD = Patients with OCD. CTR = Control children. CBT = Patients who received cognitive-behavioral therapy (CBT). PRT = Patients who received psychoeducation and relaxation training (PRT). Effect size: As effect sizes we used Cohens *d* for numerical data and Cramer's *V* for categorical data. CY-BOCS = Children's Yale-Brown Obsessive-Compulsive Scale. Comorbid diagnosis is reported if present in > 3 % of patients (self-ratings/parent-ratings). ADHD = Attention-Deficit/Hyperactivity Disorder. GAD = Generalized Anxiety Disorder.

Figure 1 BRIEF-2 Domains Pre-Treatment



Note: BRIEF-2 = Behavior Rating Inventory of Executive Function, Second Edition (Gioia et al., 2015). Inh = Inhibition, SM = Self-Monitoring, Fl = Flexibility, EC = Emotional Control, Ini = Initiating, WM = Working Memory, PO = Planning/Organizing, TM = Task Monitoring, OM = Organization of Materials, TC = Task Completion.

Figure 2 BRIEF-2 Domains Pre- and Post-Treatment by Treatment Group



Note: PRT = Patients who received psychoeducation and relaxation training (PRT). CBT = Patients who received cognitive-behavioral therapy (CBT). BRIEF-2 = Behavior Rating Inventory of Executive Function, Second Edition (Gioia et al., 2015). Inh = Inhibition, SM = Self-Monitoring, Fl = Flexibility, EC = Emotional Control, Ini = Initiating, WM = Working Memory, PO = Planning/Organizing, TM = Task Monitoring, OM = Organization of Materials, TC = Task Completion.

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Supplementary Material:

Understanding treatment response in pediatric obsessive-compulsive disorder: Executive function ratings from the TECTO trial

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Content

1 Methods

1.1 Measures

1.1.1 Parent- and Self-Reported Executive Function

Table S1 Description of BRIEF-II Clinical Scales

Clinical Scale	Number of Items		Rated Behavior
	Parent-Report	Self-Report	
Inhibit	8	8	Controlling impulses, stop behavior at the right moment
Shift	8	8	Shifting between different situations/activities or from one part of a problem to another, adapting to circumstances; shifting; flexible problem solving
Emotional Control	8	6	Moderate emotional control in a suiting manner
Self-Monitor	4	5	Monitoring effect of one's own behavior
Initiate	4	-	Initiate task/activity; generate new ideas
Task-Completion	-	7	Finishing schoolwork/domestic duties in time; completing tasks within a time frame; working in a sufficient tempo
Working Memory	8	8	Keeping information during finishing a task; continuing an activity
Plan/Organize	8	10	Foresee incidents; set goals; planning relevant steps ahead of fulfilling a task; systematical execution of tasks; understanding/communicating a central idea/concept
Task-Monitor	6	-	Controlling ones work; evaluating work during/after a task to assure fulfilling the purpose of the task
Organization of Materials	5	-	Organizing working space, playing area, and materials in a good way

Note: Source: BRIEF-2 Manual (Gioia et al., 2015)

1.2 Participants

For parent-ratings (covering age 8-17 years), 112 patients and 74 controls were included, and of these, 69 patients and 67 controls also participated with follow-up data. For self-ratings (covering age 11-17 years), 82 patients and 52 controls were included, and of these, 49 patients and 49 controls additionally participated with follow-up data.

Figure S1 Participant Flow (Parent-report)

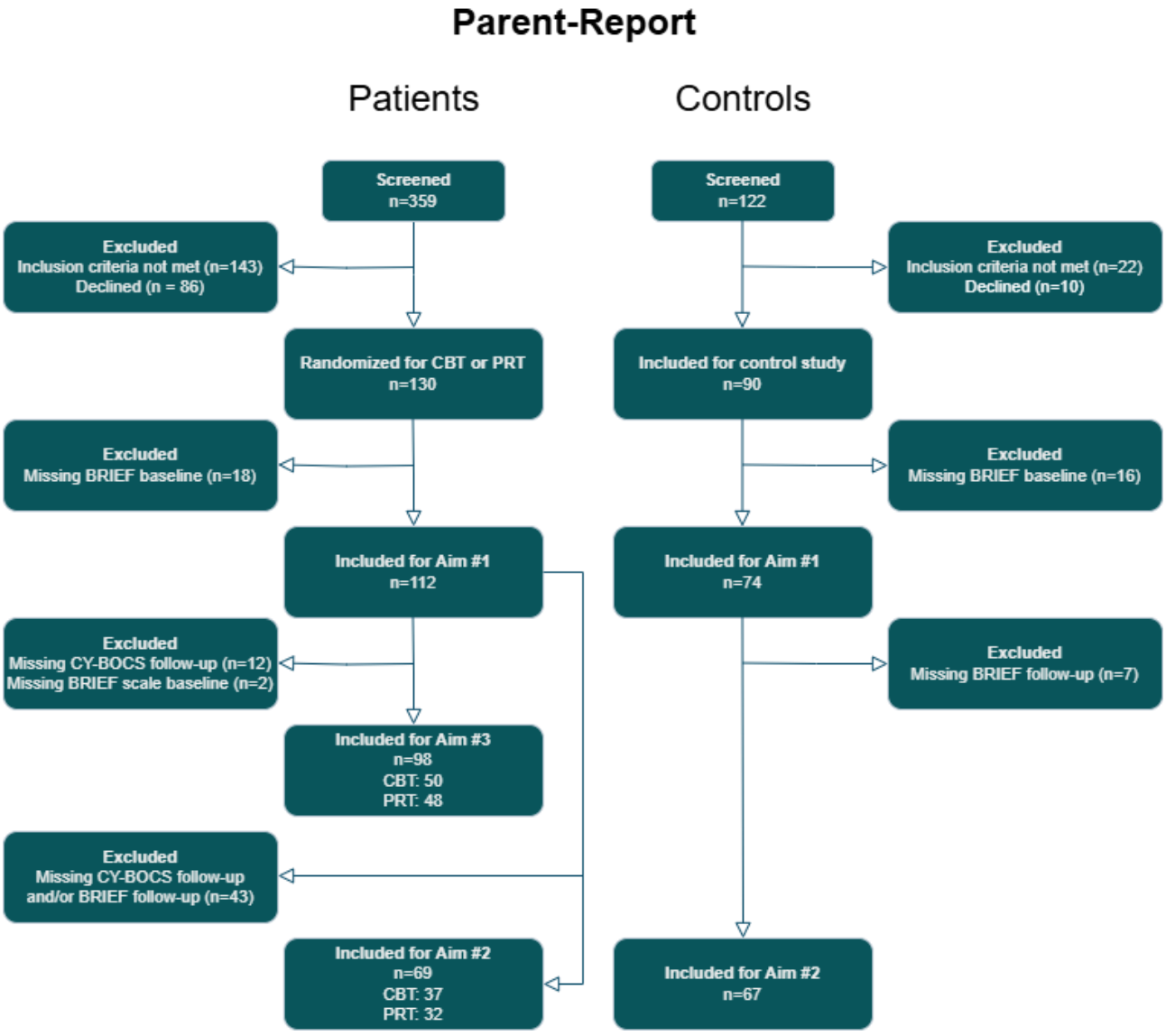
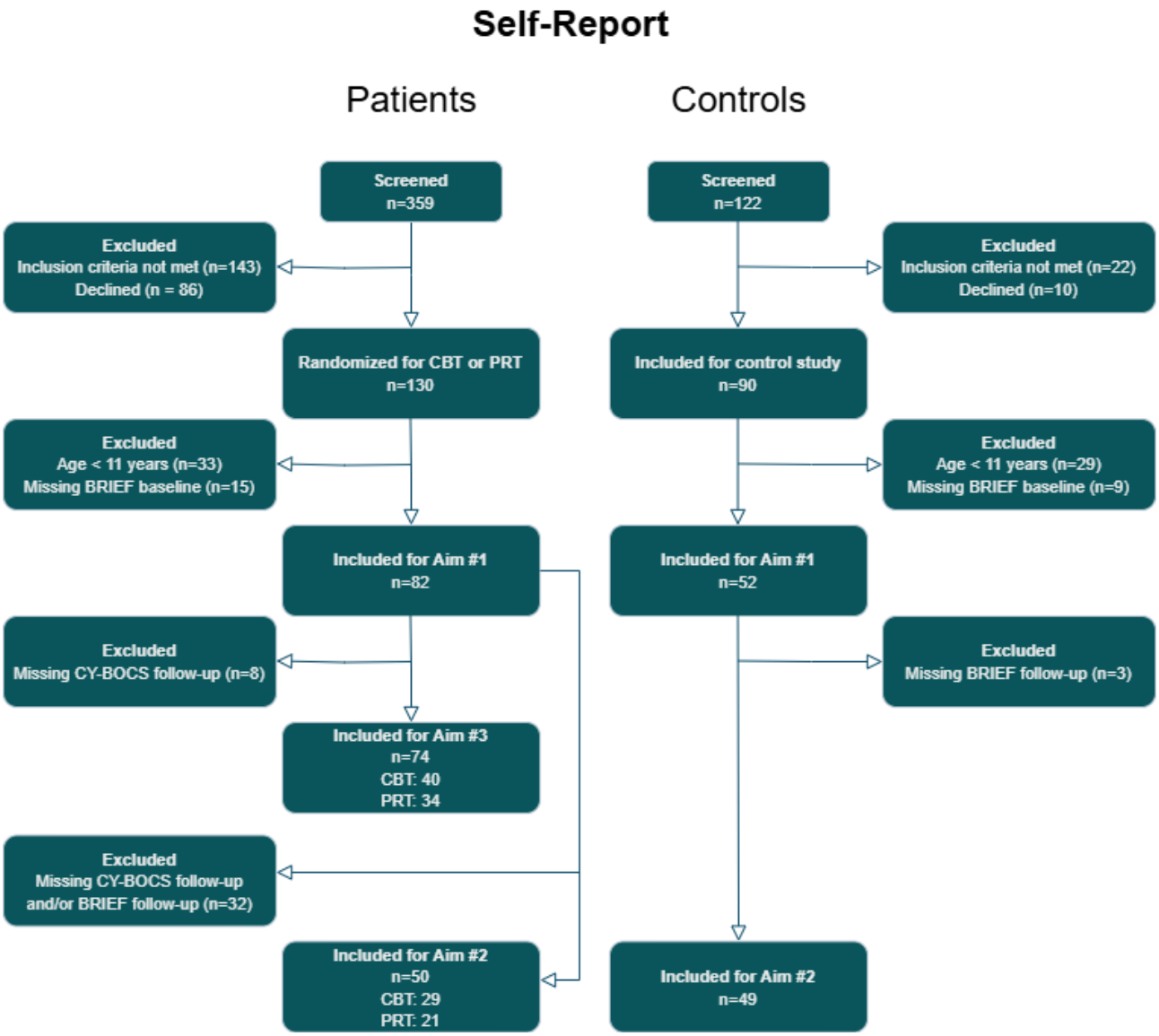


Figure S2 Participant Flow (Self-report)



2 Data Analysis

Statistical analyses were performed with the statistical software R (version 4.5.0). We conducted complete case analyses for each statistical test (for details about amount of missing data, see Figure S1 and S2). Treatment allocation was represented by a binary variable (PRT = 0, CBT = 1. Analyses were conducted separately for parent-rated (N=112 patients and 74 controls) and self-rated data (N=82 patients and 52 controls). For cases with two parent ratings available, parent-mean T-scores were used in analyses – interrater-correlation between parents has been reported as moderate to high in both normative and clinical samples (Gioia et al., 2018), and for the current sample (patients and controls at baseline), parent agreement was high across domains (r = 0.63-0.77, all p < .001). An alpha level of .05 was used for all statistical tests. Multiple linear regression models were fitted using the base lm() function in R. All BRIEF-2 domains were inspected for normality via QQ-plots (See Supplementary Figures S3-S6).

To correct for multiple comparisons, the Benjamini-Hochberg (BH) procedure with a cut-off for false discovery rate (FDR) of 5 % was used within each main analysis. Critical values were 0.01281 for parent-ratings and 0.00567 for self-ratings. Since parent- and child reports often differ in clinical studies, we applied the BH procedure for the two informants separately.

For the main analyses, we test five hypotheses (aims 1.1, 1.2, 2.1, 2.2, and 3), and thus the BH correction is based on the five corresponding p values. For main analyses of parent-report data, we get the following p values:

$2.2 \times e^{-16}$, 0.04396, $2.375 \times e^{-06}$, 0.467, 0.007686

and a critical value of 0.01281.

For main analyses of self-report data, we get the following p values:

$3.009 \times e^{-08}$, 0.001131, $5.252 \times e^{-05}$, 0.631, 0.004536

and a critical value of 0.00567.

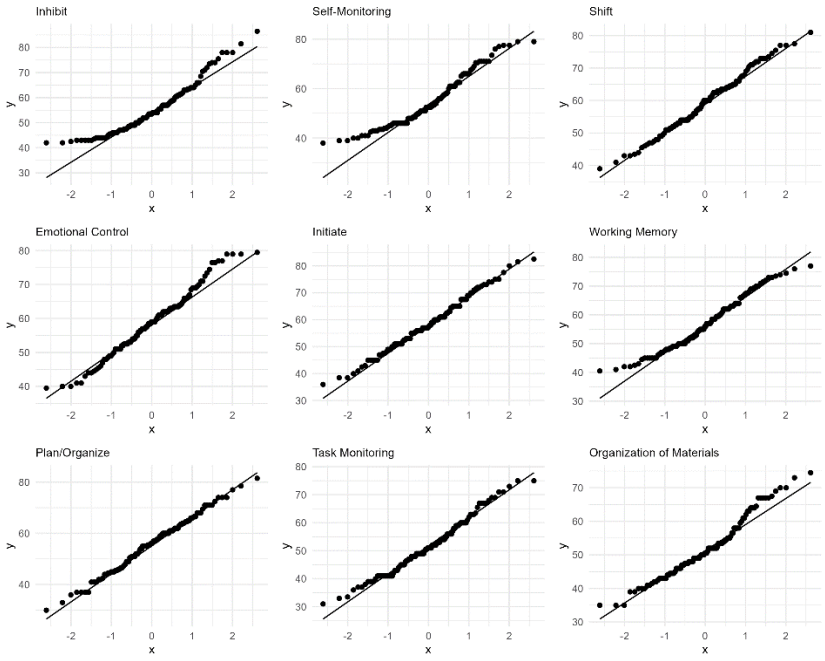
2.1 Demographic and clinical characteristics

Potential differences in baseline characteristics of patients versus controls were tested using independent-samples *t*-tests for continuous variables and chi-square tests for binary variables. Following previous work (Hybel et al., 2017; Uhre et al., 2023), years of parental education was calculated as either the mean of both parents, or, if only one parent participated, that parent's value was used. We reported the comorbid psychiatric diagnoses that were most frequently present in our sample (> 3 %).

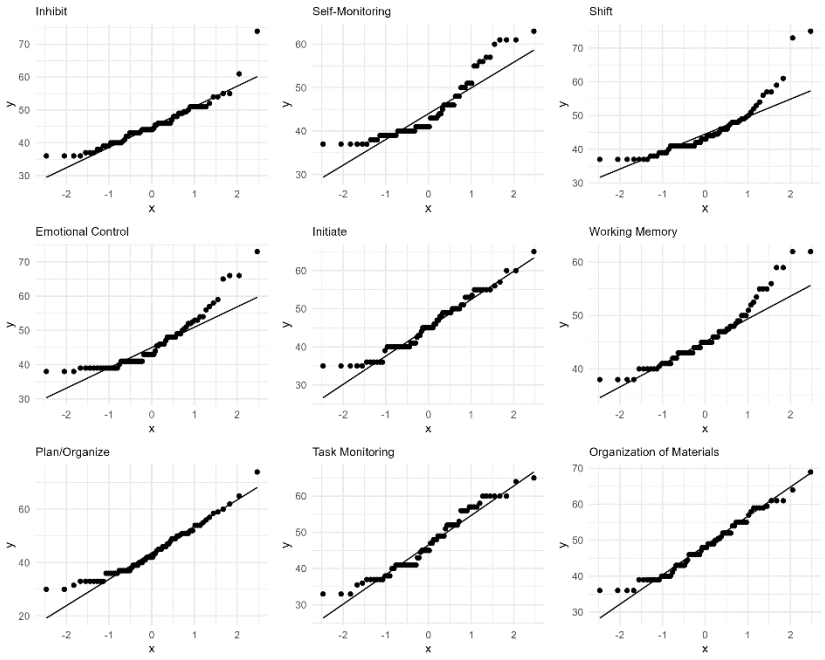
2.2 Checking Assumptions

2.2.1 Normality (QQ Plots)

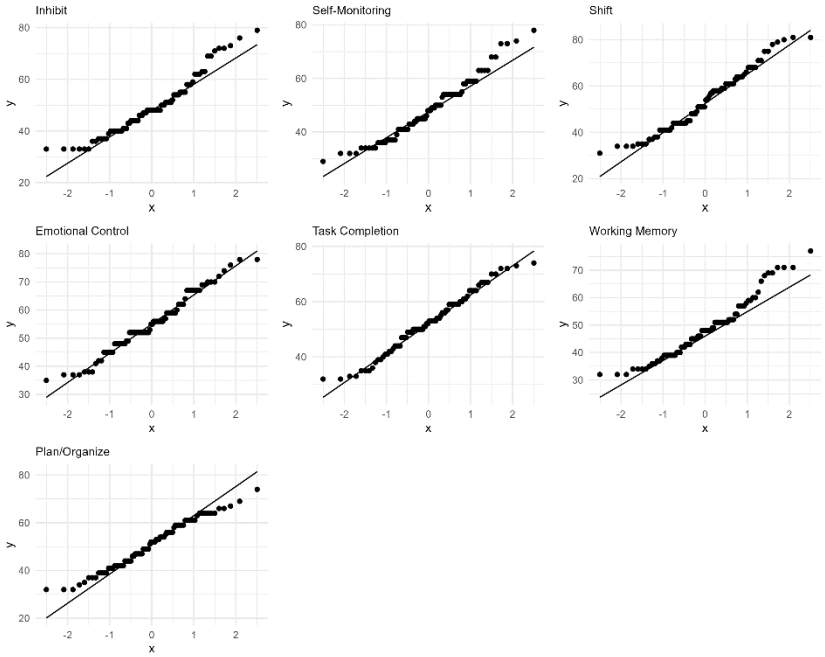
Supplementary Figure S3 QQ Plot of BRIEF sub-scales for Patients (Parent-Report; child age: 8-17)



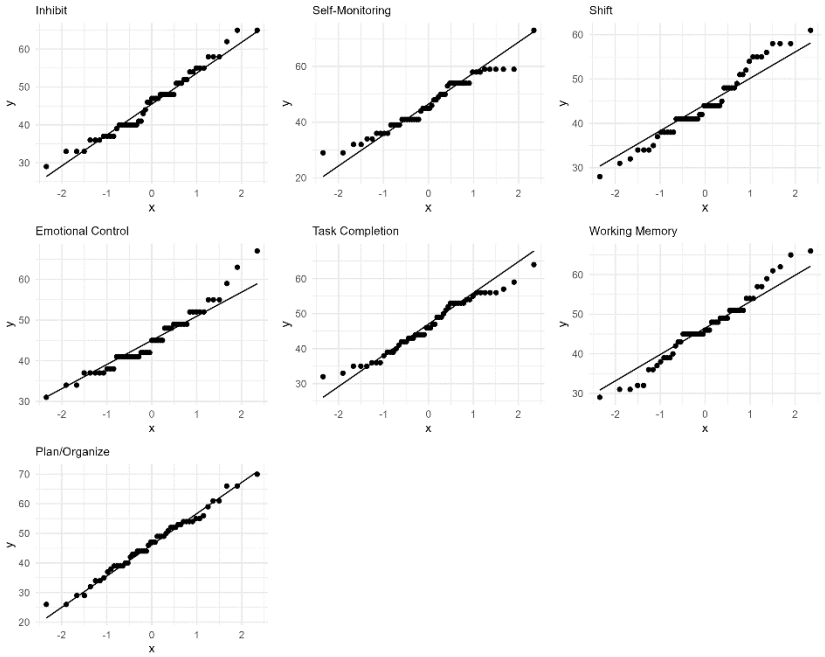
Supplementary Figure S4 QQ Plot of BRIEF sub-scales for Controls (Parent-Report; child age: 8-17)



Supplementary Figure S5 QQ Plot of BRIEF sub-scales for Patients (Self-Report; child age: 11-17)



Supplementary Figure S6 QQ Plot of BRIEF sub-scales for Controls (Self-Report; child age: 11-17)



3 Results

3.1 Demographic and clinical characteristics

Supplementary Table S2 Demographic and Clinical Characteristics pre-treatment for patients with complete BRIEF data at pre- and post-treatment (Parent-Report and Self-Report)

	OCD Mean(*SD*), *n*(%)	**CBT** Mean(*SD*), *n*(%)	**PRT** Mean(*SD*), *n*(%)	**CBT vs PRT** *p*; Effect	
Number of participants					
Parent-report	69	37	32		
Self-report	50	29	21		
Number of females					
Parent-report	41 (59.4)	23 (62.2)	18 (56.3)	.800	.060
Self-report	34 (68.0)	10 (34.5)	15 (71.4)	.893	.063
Age in years					
Parent-report	13.1 (2.74)	12.9 (2.77)	13.3 (2.73)	.631	.117
Self-report	14.5 (1.94)	14.3 (2.00)	14.8 (1.86)	.386	.248
Parental education, years					
Parent-report	15.6 (2.20)	15.3 (2.56)	15.9 (1.60)	.292	.250
Self-report	15.4 (2.22)	15.3 (2.49)	15.6 (1.75)	.682	.114
Intelligence Quotient (IQ)					
Parent-report	102 (12.9)	101 (12.8)	103 (13.2)	.565	.140
Self-report	101 (11.4)	101 (11.4)	101 (11.7)	.804	.072
CY-BOCS Pre-Treatment Total Score					
Parent-report	24.5 (3.92)	24.1 (3.93)	24.9 (3.94)	.439	.188
Self-report	24.6 (4.00)	24.1 (3.78)	25.1 (4.30)	.804	.251
Co-morbid disorders					
Asperger's (F84.5)					
Parent-report	9 (13.0)	3 (8.1)	6 (18.7)	-	-
Self-report	6 (12.77)	2 (7.7)	4 (19.0)	-	-
ADHD (F90.0)					
Parent-report	9 (13.0)	5 (13.5)	4 (12.5)	-	-
Self-report	4 (8.51)	3 (11.5)	1 (4.8)	-	-
GAD (F41.1 & F93.8)					
Parent-report	5 (7.25)	1 (2.7)	4 (12.5)	-	-
Self-report	3 (6.38)	0 (0)	3 (14.3)	-	-
Tourette's (F95.2)					
Parent-report	4 (5.80)	2 (5.4)	2 (6.25)	-	-
Self-report	3 (6.38)	1 (3.8)	2 (9.5)	-	-
Any co-morbid disorder					
Parent-report	38 (55.07)	20 (54.05)	18 (56.25)	-	-
Self-report	25 (50.0)	14 (48.3)	11 (52.4)	-	-

Note: OCD = Patients with OCD. CTR = Control children. CBT = Patients who received cognitive-behavioral therapy (CBT). PRT = Patients who received psychoeducation and relaxation training (PRT). Effect: As effect sizes we used Cohens *d* for numerical data and Cramer's *V* for categorical data. Comorbid diagnosis is reported if present in > 3 % of patients (self-report/parent-report). ADHD = Attention-Deficit/Hyperactivity Disorder. GAD = Generalized Anxiety Disorder.

3.2 Executive Function Pre-Treatment and Associations with Symptom Severity

3.2.1 Follow-up ANOVA tables

Supplementary Table S3 Follow-Up ANOVAs of BRIEF domains pre-treatment (OCD vs. controls; parent-report; child age: 8-17)

	OCD Estimated Mean	**CTR** Estimated Mean	F(9,174)	*p*	Cohen's *d*
BRIEF-II sub-domain					
Inhibition	55.28	45.25	71.30	**<.001**	1.16
Self-Monitoring	54.66	44.60	61.85	**<.001**	1.09
Flexibility	59.31	45.04	139.38	**<.001**	1.70
Emotional Control	58.81	45.97	104.28	**<.001**	1.46
Initiating	58.46	45.53	108.59	**<.001**	1.46
Working Memory	56.78	45.95	101.90	**<.001**	1.38
Planning/Organizing	55.84	44.10	65.24	**<.001**	1.18
Task Monitoring	51.71	46.89	12.97	**<.001**	0.52
Organization of Materials	51.79	48.36	8.19	**.005**	0.42

Note: OCD = Patients with OCD. CTR = Non-psychiatric control children.

Supplementary Table S4 Follow-Up ANOVAs of BRIEF domains pre-treatment (OCD vs. controls; self-report; child age: 11-17)

	OCD Estimated Mean	**CTR** Estimated Mean	F (7,126)	*p*	Cohen's *d*
BRIEF-II sub-domain					
Inhibition	49.22	45.74	4.30	**.040**	0.35
Self-Monitoring	48.35	46.35	1.24	.267	0.19
Flexibility	53.55	44.21	27.84	**<.001**	0.84
Emotional Control	55.25	45.06	45.23	**<.001**	1.11
Task Completion	52.32	46.45	13.90	**<.001**	0.62
Working Memory	48.30	46.38	1.39	.241	0.20
Planning/Organizing	51.00	46.44	6.55	**.012**	0.46

Note: OCD = Patients with OCD. CTR = Non-psychiatric control children.

3.2.2 Sensitivity analyses (including Parental Education and IQ)

The MANCOVA analyses with IQ and parental education as covariates continued to indicate a significant multivariate effect of group (OCD versus controls) on the combined BRIEF clinical scales for both self-report and parent-report (Pillai's Trace = 0.350, F(7,75) = 5.781, p <.001, $\eta_p{}^2$ = 0.33; Pillai's Trace = 0.543, F(9,109) = 14.372, p <.001, $\eta_p{}^2$ = 0.52). The multiple linear regression remained significant for the self-report (F(9,65) = 3.662, p <.001, R^2 = .336) and insignificant for the parent-report data (F(11,89) = 1.789, p = .068, R^2 = .181).

Supplementary Table S5 Follow-Up ANOVA's with IQ and Parental Education (OCD vs. controls; parent-report; child age: 8-17)

BRIEF-II sub-domain	*F*(9,109)	*p*
Inhibition	57.559	**<.001**
Self-Monitoring	24.793	**<.001**
Flexibility	71.697	**<.001**
Emotional Control	54.412	**<.001**
Initiating	52.338	**<.001**
Working Memory	66.267	**<.001**
Planning/Organizing	32.726	**<.001**
Task Monitoring	13.500	**<.001**
Organization of Materials	6.126	**.015**

Supplementary Table S6 Follow-Up ANOVA's with IQ and Parental Education (OCD vs. controls; self-report; child age: 11-17)

BRIEF-II sub-domain	*F*(7,75)	*p*
Inhibition	4.930	**.021**
Self-Monitoring	1.931	.169
Flexibility	10.220	**.002**
Emotional Control	35.544	**<.001**
Task Completion	10.900	**.001**
Working Memory	1.015	.317
Planning/Organizing	2.630	.109

Supplementary Table S7 Multiple Regression with IQ and Parental Education predicting CY-BOCS baseline (self-report; child age: 11-17)

Independent variable	Regression coefficient	*t*	*p*
Intercept	12.660	2.135	.037
BRIEF-II sub-domain			
Inhibition	-0.097	-1.480	.144
Self-Monitoring	-0.143	-2.450	.017
Flexibility	0.233	4.457	**<.001**
Emotional Control	0.028	0.446	.673
Task Completion	-0.013	-0.182	.856
Working Memory	0.011	0.143	.887
Planning/Organizing	0.053	0.665	.211

3.2.3 Proportion of Participants with Clinically Elevated Domain Score

Supplementary Table S8 Proportion of Participants with Clinically Elevated Scores (Parent-Rating)

Scale	0-59	60-64	65-69	70+
Inhibit	79 (71.2%)	16 (14.4%)	4 (3.6%)	12 (10.8%)
Self-Monitor	77 (68.8%)	11 (9.8%)	10 (8.9%)	14 (12.5%)
Shift	54 (50%)	27 (25%)	11 (10.2%)	16 (14.8%)
Emotional Control	61 (55.5%)	25 (22.7%)	10 (9.1%)	14 (12.7%)
Initiate	62 (56.4%)	17 (15.5%)	14 (12.7%)	17 (15.5%)
Working Memory	71 (64%)	19 (17.1%)	9 (8.1%)	12 (10.8%)
Planning/Organizing	67 (62%)	21 (19.4%)	9 (8.3%)	11 (10.2%)
Task-Monitor	87 (78.4%)	11 (9.9%)	8 (7.2%)	5 (4.5%)
Organization of Materials	91 (82%)	9 (8.1%)	7 (6.3%)	4 (3.6%)

Supplementary Table S9 Proportion of Participants with Clinically Elevated Scores (Self-Rating)

Scale	0-59	60-64	65-69	70+
Inhibit	69 (84.1%)	5 (6.1%)	2 (2.4%)	6 (7.3%)
Self-Monitor	72 (87.8%)	4 (4.9%)	2 (2.4%)	4 (4.9%)
Shift	56 (68.3%)	11 (13.4%)	6 (7.3%)	9 (11%)
Emotional Control	59 (72%)	6 (7.3%)	9 (11%)	8 (9.8%)
Task-Completion	63 (76.8%)	9 (11%)	4 (4.9%)	6 (7.3%)
Working Memory	71 (86.6%)	3 (3.7%)	4 (4.9%)	4 (4.9%)
Planning/Organizing	64 (78%)	13 (15.9%)	4 (4.9%)	1 (1.2%)

3.2.4 Number of clinically elevated BRIEF-2 domains

Table S10 Number of clinically elevated BRIEF-2 domains

ClinGr	n_elevated	n	Percent	Cutoff	Report
1	0	19	17.3	≥60	Parent
1	1	19	17.3	≥60	Parent
1	2	16	14.5	≥60	Parent
1	3	14	12.7	≥60	Parent
1	4	10	9.1	≥60	Parent
1	5	8	7.3	≥60	Parent
1	6	7	6.4	≥60	Parent
1	7	5	4.5	≥60	Parent
1	8	8	7.3	≥60	Parent
1	9	4	3.6	≥60	Parent
2	0	56	75.7	≥60	Parent
2	1	12	16.2	≥60	Parent
2	2	1	1.4	≥60	Parent
2	3	2	2.7	≥60	Parent
2	4	2	2.7	≥60	Parent
2	8	1	1.4	≥60	Parent
2	5	0	0.0	≥60	Parent
2	6	0	0.0	≥60	Parent
2	7	0	0.0	≥60	Parent
2	9	0	0.0	≥60	Parent
1	0	52	47.3	≥65	Parent
1	1	18	16.4	≥65	Parent
1	2	8	7.3	≥65	Parent
1	3	9	8.2	≥65	Parent
1	4	6	5.5	≥65	Parent
1	5	7	6.4	≥65	Parent
1	6	5	4.5	≥65	Parent
1	7	2	1.8	≥65	Parent

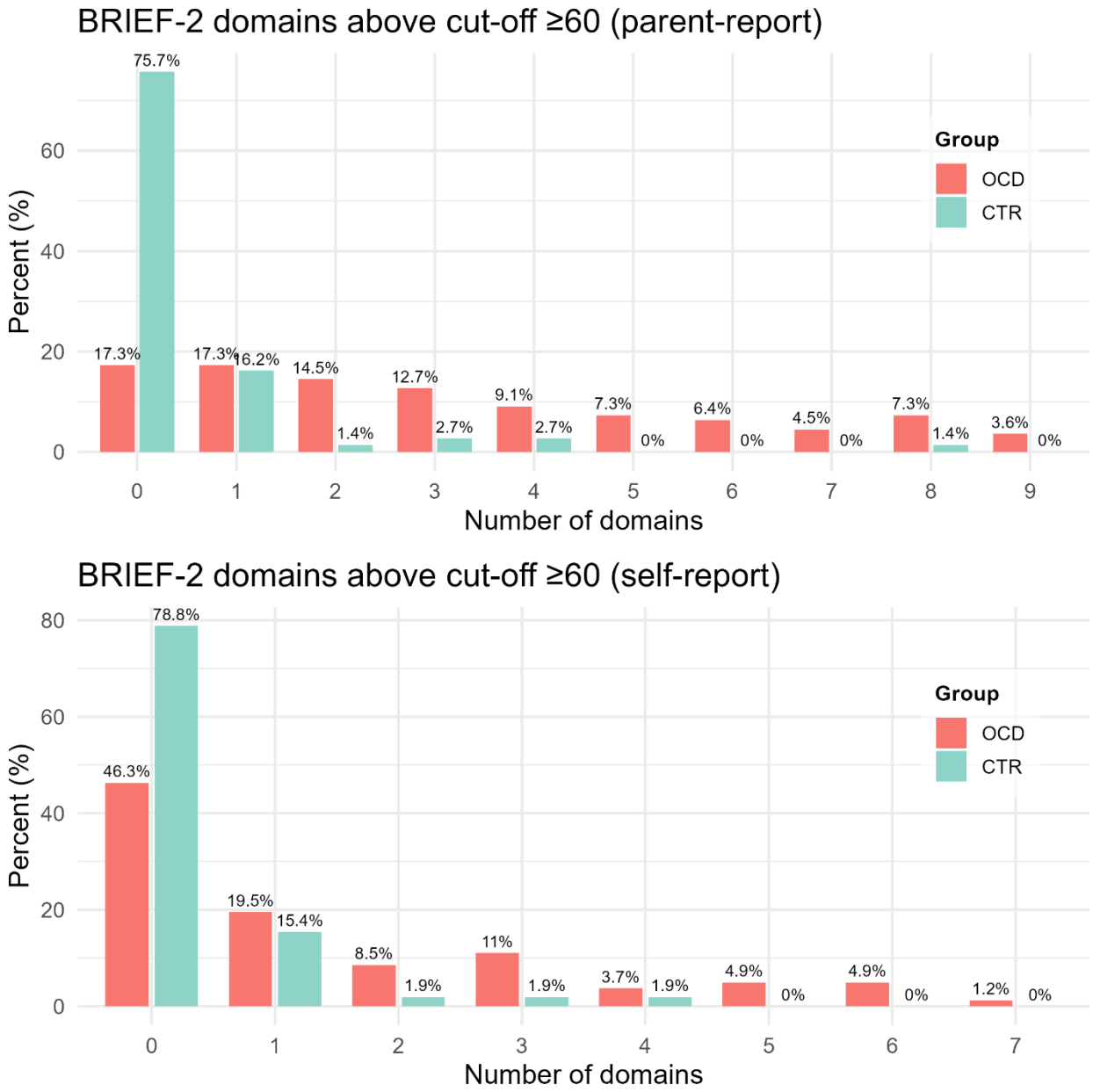
ClinGr	n_elevated	n	Percent	Cutoff	Report
1	8	3	2.7	≥65	Parent
2	0	68	91.9	≥65	Parent
2	1	3	4.1	≥65	Parent
2	2	1	1.4	≥65	Parent
2	3	1	1.4	≥65	Parent
2	4	1	1.4	≥65	Parent
2	5	0	0.0	≥65	Parent
2	6	0	0.0	≥65	Parent
2	7	0	0.0	≥65	Parent
2	8	0	0.0	≥65	Parent
1	9	0	0.0	≥65	Parent
2	9	0	0.0	≥65	Parent
1	0	66	60.0	≥70	Parent
1	1	19	17.3	≥70	Parent
1	2	10	9.1	≥70	Parent
1	3	6	5.5	≥70	Parent
1	4	4	3.6	≥70	Parent
1	5	1	0.9	≥70	Parent
1	6	2	1.8	≥70	Parent
1	7	1	0.9	≥70	Parent
1	8	1	0.9	≥70	Parent
2	0	70	94.6	≥70	Parent
2	1	3	4.1	≥70	Parent
2	2	1	1.4	≥70	Parent
2	3	0	0.0	≥70	Parent
2	4	0	0.0	≥70	Parent
2	5	0	0.0	≥70	Parent
2	6	0	0.0	≥70	Parent
2	7	0	0.0	≥70	Parent
2	8	0	0.0	≥70	Parent
1	9	0	0.0	≥70	Parent

ClinGr	n_elevated	n	Percent	Cutoff	Report
2	9	0	0.0	≥70	Parent
1	0	38	46.3	≥60	Child
1	1	16	19.5	≥60	Child
1	2	7	8.5	≥60	Child
1	3	9	11.0	≥60	Child
1	4	3	3.7	≥60	Child
1	5	4	4.9	≥60	Child
1	6	4	4.9	≥60	Child
1	7	1	1.2	≥60	Child
2	0	41	78.8	≥60	Child
2	1	8	15.4	≥60	Child
2	2	1	1.9	≥60	Child
2	3	1	1.9	≥60	Child
2	4	1	1.9	≥60	Child
2	5	0	0.0	≥60	Child
2	6	0	0.0	≥60	Child
2	7	0	0.0	≥60	Child
1	0	50	61.0	≥65	Child
1	1	14	17.1	≥65	Child
1	2	8	9.8	≥65	Child
1	3	5	6.1	≥65	Child
1	4	2	2.4	≥65	Child
1	5	2	2.4	≥65	Child
1	6	1	1.2	≥65	Child
2	0	44	84.6	≥65	Child
2	1	7	13.5	≥65	Child
2	2	1	1.9	≥65	Child
2	3	0	0.0	≥65	Child
2	4	0	0.0	≥65	Child
2	5	0	0.0	≥65	Child
2	6	0	0.0	≥65	Child

ClinGr	n_elevated	n	Percent	Cutoff	Report
1	7	0	0.0	≥65	Child
2	7	0	0.0	≥65	Child
1	0	62	75.6	≥70	Child
1	1	9	11.0	≥70	Child
1	2	6	7.3	≥70	Child
1	3	3	3.7	≥70	Child
1	4	2	2.4	≥70	Child
2	0	50	96.2	≥70	Child
2	1	2	3.8	≥70	Child
2	2	0	0.0	≥70	Child
2	3	0	0.0	≥70	Child
2	4	0	0.0	≥70	Child
1	5	0	0.0	≥70	Child
2	5	0	0.0	≥70	Child
1	6	0	0.0	≥70	Child
2	6	0	0.0	≥70	Child
1	7	0	0.0	≥70	Child
2	7	0	0.0	≥70	Child

3.2.5 Number of clinically elevated BRIEF-2 domains (T score ≥ 60)

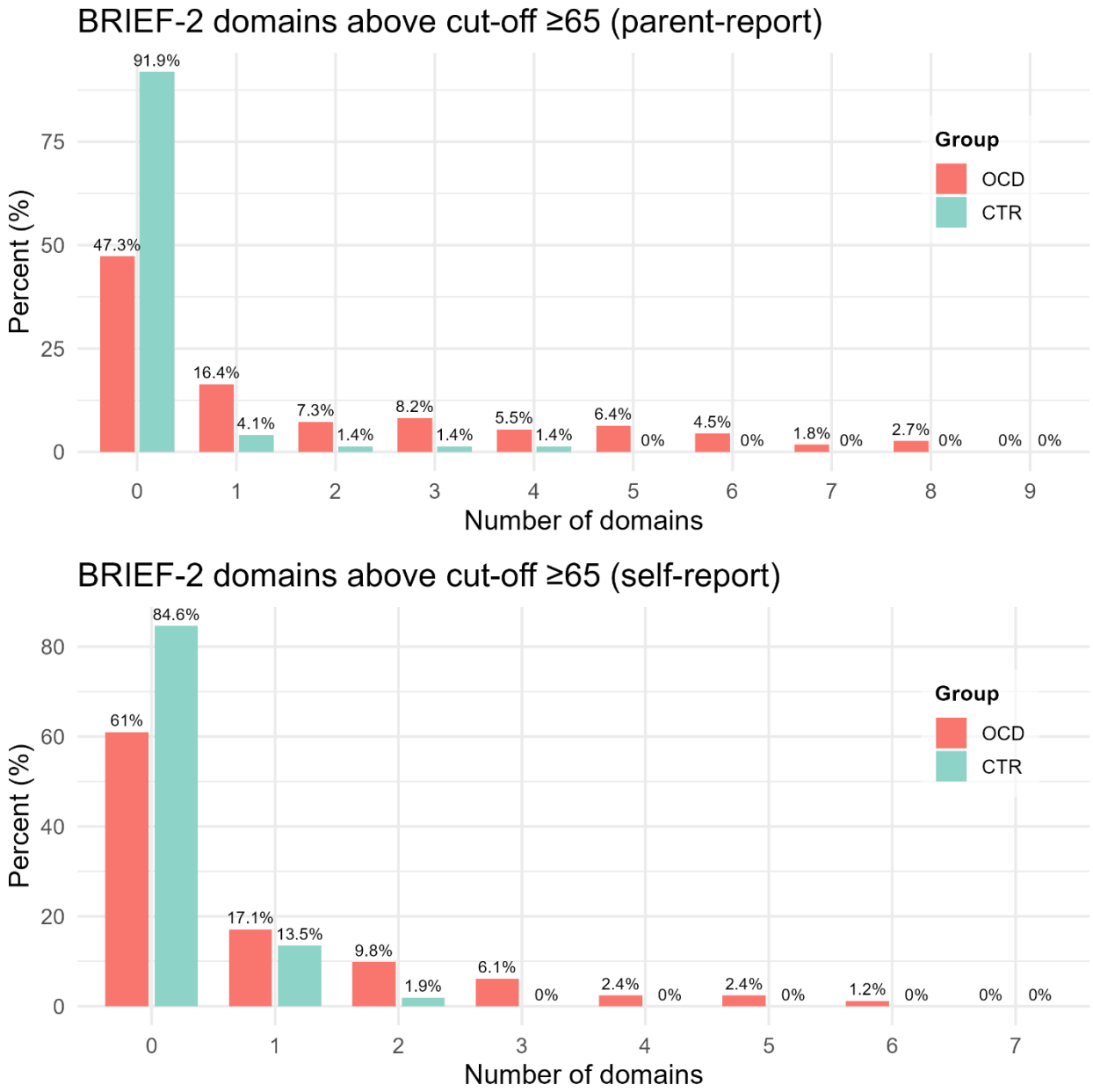
Figure S7 Number of clinically elevated BRIEF-2 domains (T score ≥ 60)



Note: OCD = Patients with OCD. CTR = Control children.

3.2.6 Number of clinically elevated BRIEF-2 domains (T score ≥ 65)

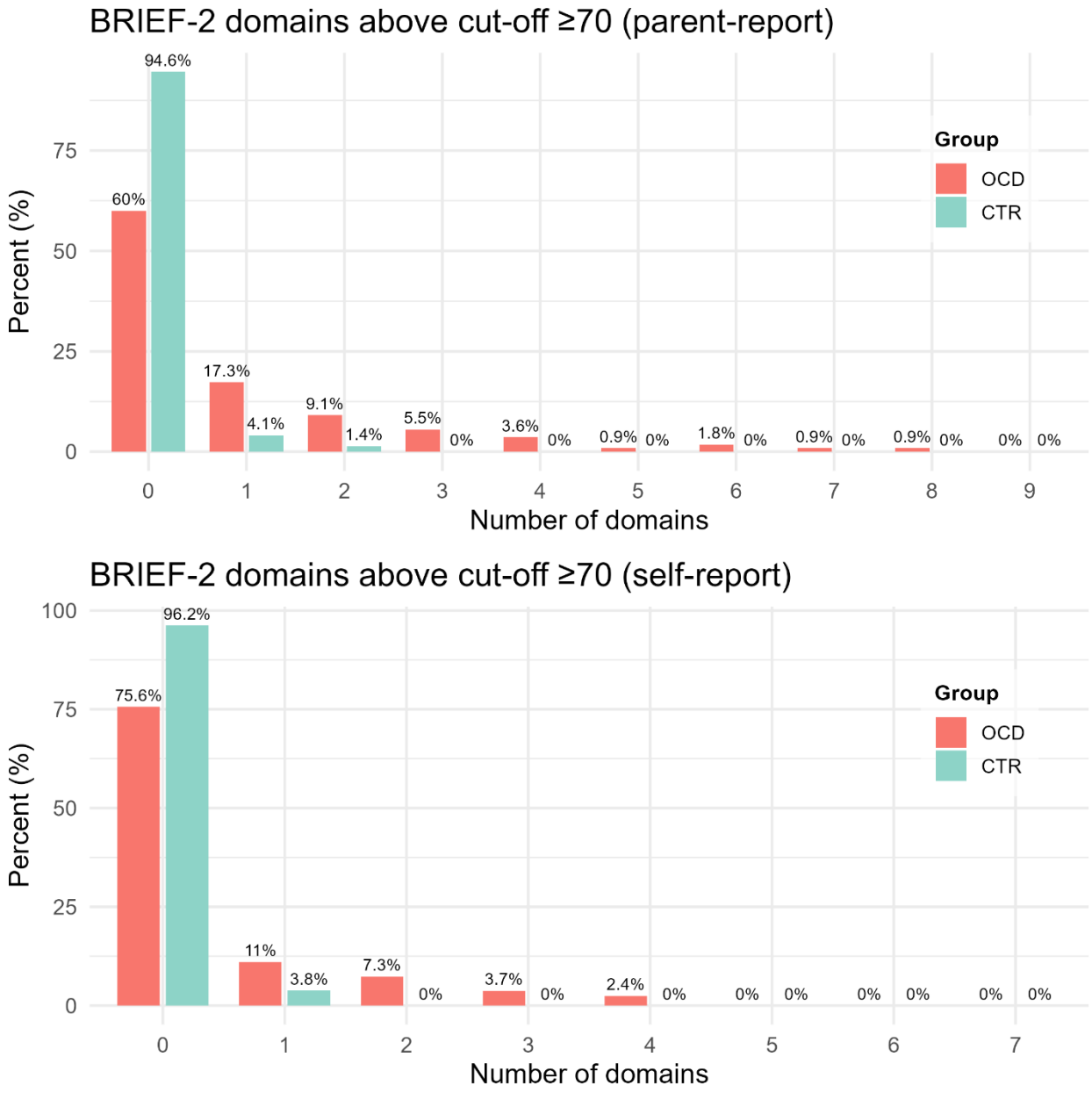
Figure S8 Number of clinically elevated BRIEF-2 domains (T score ≥ 65)



Note: OCD = Patients with OCD. CTR = Control children.

3.2.7 Number of clinically elevated BRIEF-2 domains (T score ≥ 70)

Figure S9 Number of clinically elevated BRIEF-2 domains (T score ≥ 70)



Note: OCD = Patients with OCD. CTR = Control children.

3.2.8 Individual predictors from multiple linear regression

Supplementary Table S11 Multiple Linear Regression Results (self-report; child age: 11-17)

Independent variable	Regression coefficient	*t*	*p*
Intercept	20.975	6.971	**<.001**
BRIEF-II sub-domain			
Inhibition	-0.115	-1.850	.068
Self-Monitoring	-0.114	-2.018	.047
Flexibility	0.207	4.272	**<.001**
Emotional Control	0.037	0.648	.518
Task Completion	-0.032	-0.511	.611
Working Memory	0.046	0.638	.526
Planning/Organizing	0.034	0.441	.661

3.3 Executive Function Change during Treatment and Association with Symptom Change

3.3.1 Repeated Measures ANOVA, Complete Results

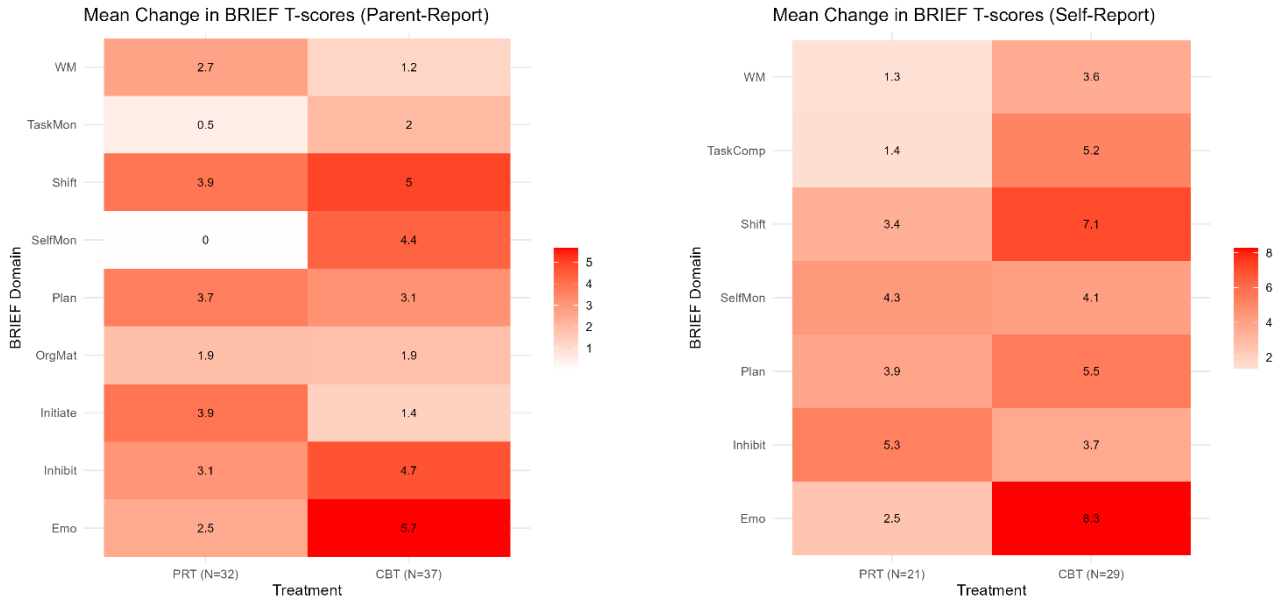
Table S12 2×2×9 Repeated Measures ANOVA (parent-report; child age: 8-17)

Effect	*df*	*F*	*p*	η_p^2
Treatment Group	1.00, 67.00	0.891	.766	0.00
Time	1.00, 67.00	25.336	**<.001**	0.27
Treatment Group × Time	1.00, 67.00	0.472	.495	0.01
BRIEF Domain	5.00, 334.79	8.313	**<.001**	0.11
Treatment Group × Brief Domain	5.00, 334.79	0.805	.546	0.01
Time × BRIEF Domain	6.57, 439.95	2.596	**.014**	0.04
Treatment Group × Time × BRIEF Domain	6.57, 439.95	2.452	**.020**	0.04

Table S13 2×2×7 Repeated Measures ANOVA (parent-report; child age: 8-17)

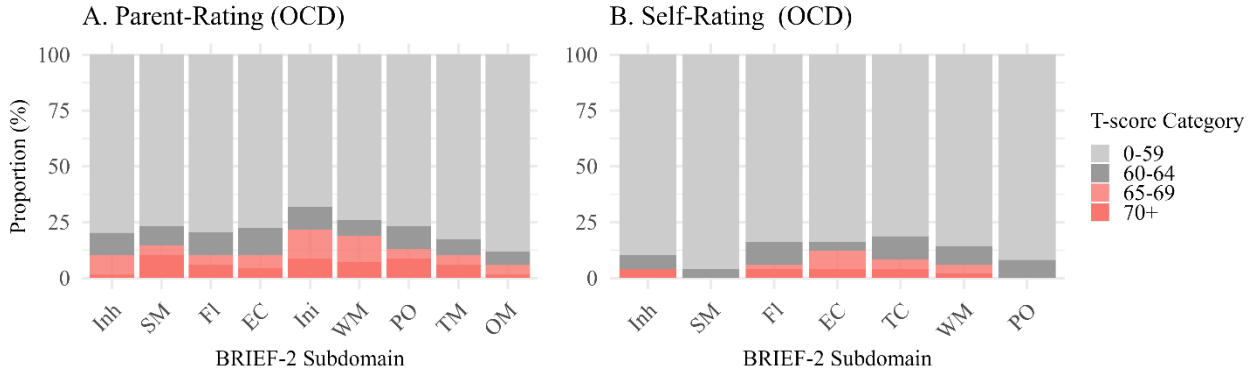
Effect	*df*	*F*	*p*	η_p^2
Treatment Group	1, 43	0.065	.800	0.00
Time	1, 43	21.762	**<.001**	0.34
Treatment Group × Time	1, 43	1.203	.279	0.03
BRIEF Domain	4.56, 195.96	7.391	**<.001**	0.15
Treatment Group × Brief Domain	4.56, 195.96	0.152	.937	0.00
Time × BRIEF Domain	5.16, 221.75	1.089	.368	0.02
Treatment Group × Time × BRIEF Domain	5.16, 221.75	1.696	.134	0.04

Figure S10 Mean change in BRIEF Domain T-scores by Treatment Group (Heat Plot)



3.3.2 Proportion of Participants with Clinically Elevated Scores Post-treatment

Figure S11 Clinically Elevated BRIEF Scores (Patients, post-treatment)



Note: BRIEF-2 = Behavior Rating Inventory of Executive Function, Second Edition (Gioia et al., 2015). Inh = Inhibition, SM = Self-Monitoring, Fl = Flexibility, EC = Emotional Control, Ini = Initiating, WM = Working Memory, PO = Planning/Organizing, TC = Task Completion.

3.3.3 Follow-up Estimated Marginal Means (Patients)

The *emmeans* package in R was used (Lenth et al., 2025).

Table S14 2×2×9 Follow-up Estimated Marginal Means (parent-report; child age: 8-17)

	PRT Pre-treatment M(SE) [95 % CI]	**PRT** Post-treatment M(SE) [95 % CI]	**CBT** Pre-treatment M(SE) [95 % CI]	**CBT** Post-treatment M(SE) [95 % CI]
BRIEF-II Sub Domain				
Inhibition	53.5 (1.6) [50.4, 56.6]	50.4 (1.6) [47.2, 53.6]	56.2 (1.5) [53.3, 59.1]	51.5 (1.5) [48.5, 56.3]
Self-Monitoring	52.3 (1.8) [48.6, 56.0]	52.3 (2.0) [48.2, 56.3]	54.2 (1.7) [50.8, 57.6]	49.8 (1.9) [46.1, 53.6]
Flexibility	58.1 (1.6) [54.9, 61.1]	54.1 (1.6) [50.8, 57.4]	57.1 (1.5) [54.2, 60.0]	52.1 (1.5) [49.1, 55.2]
Emotional Control	57.8 (1.7) [54.4, 61.3]	55.3 (1.5) [51.8, 58.8]	58.8 (1.6) [55.2, 61.7]	52.8 (1.6) [49.6, 56.0]
Initiating	58.7 (1.6) [55.2, 62.1]	54.7 (1.8) [51.1, 58.4]	55.6 (1.6) [52.4, 58.7]	54.2 (1.7) [50.8, 57.6]
Working Memory	57.2 (1.7) [53.8, 60.5]	54.5 (1.7) [53.8, 51.2]	55.9 (1.5) [52.8, 59.0]	54.6 (1.5) [51.6, 57.7]
Planning/Organizing	55.6 (1.9) [51.9, 59.3]	51.9 (2.1) [47.7, 56.1]	53.1 (1.7) [49.6, 56.5]	50.0 (2.0) [46.0, 53.9]
Task Monitoring	50.8 (1.6) [47.6, 54.0]	50.3 (1.8) [46.7, 53.8]	52.5 (1.5) [49.5, 55.4]	50.4 (1.7) [47.1, 53.7]
Organization of Materials	51.9 (1.4) [49.0, 54.8]	50.0 (1.5) [47.0, 53.0]	51.8 (1.3) [49.1, 54.5]	49.9 (1.4) [47.1, 52.7]

Table S15 2×2×7 Follow-up Estimated Marginal Means (self-report; child age: 11-17)

	PRT Pre-treatment M(SE) [95 % CI]	**PRT** Post-treatment M(SE) [95 % CI]	**CBT** Pre-treatment M(SE) [95 % CI]	**CBT** Post-treatment M(SE) [95 % CI]
BRIEF-II Sub Domain				
Inhibition	49.3 (2.6) [44.1, 54.5]	44.0 (2.5) [39.0, 49.0]	49.2 (2.3) [44.6, 53.8]	45.6 (2.2) [41.2, 50.0]
Self-Monitoring	47.3 (2.5) [42.2, 52.4]	43.0 (2.0) [39.0, 46.9]	47.5 (2.2) [43.0, 52.0]	43.2 (1.7) [39.7, 46.7]
Flexibility	51.6 (2.9) [45.7, 57.4]	48.1 (2.5) [43.1, 53.1]	51.3 (2.8) [46.1, 56.5]	45.0 (2.2) [40.6, 49.4]
Emotional Control	54.3 (2.4) [49.5, 59.1]	51.8 (2.5) [46.8, 56.7]	56.2 (2.1) [52.0, 60.4]	48.0 (2.2) [43.6, 52.4]
Task Completion	50.7 (2.1) [46.4, 54.9]	49.2 (2.4) [44.5, 54.0]	51.8 (1.8) [48.1, 55.5]	46.1 (2.1) [42.0, 50.4]
Working Memory	46.4 (2.3) [41.8, 51.1]	45.1 (2.5) [40.1, 50.1]	48.4 (2.1) [44.3, 52.5]	44.5 (2.2) [40.1, 48.9]
Planning/Organizing	48.5 (2.1) [44.4, 52.7]	44.7 (2.3) [40.0, 49.3]	50.1 (1.8) [46.5, 53.8]	44.4 (2.0) [40.3, 48.5]

3.3.4 Follow-up Estimated Marginal Means (Controls)

Table S16 2×2×9 Follow-up Estimated Marginal Means (controls; parent-report; child age: 8-17)

	CTR Week 0 M(SE) [95 % CI]	**CTR** Week 16 M(SE) [95 % CI]
BRIEF-II Sub Domain		
Inhibition	45.6 (0.8) [44.0, 47.1]	46.5 (0.9) [44.7, 48.2]
Self-Monitoring	44.7 (0.9) [43.0, 46.4]	44.6 (0.8) [43.0, 46.3]
Flexibility	45.4 (0.9) [43.6, 47.3]	45.6 (0.8) [44.0, 47.2]
Emotional Control	46.3 (1.0) [44.4, 48.2]	45.8 (0.9) [44.0, 47.5]
Initiating	45.8 (0.9) [44.0, 47.6]	46.5 (0.9) [44.7, 48.3]
Working Memory	46.1 (0.7) [44.7, 47.5]	46.7 (0.8) [45.0, 48.3]
Planning/Organizing	44.6 (1.1) [42.3, 46.8]	45.4 (1.0) [43.4, 47.5]
Task Monitoring	47.3 (1.0) [45.2, 49.3]	47.6 (1.1) [45.4, 49.7]
Organization of Materials	48.5 (1.0) [46.6, 50.5]	48.3 (0.8) [46.7, 50.0]

Table S17 2×2×7 Follow-up Estimated Marginal Means (controls; self-report; child age: 8-17)

	CTR Week 0 M(SE) [95 % CI]	**CTR** Week 16 M(SE) [95 % CI]
BRIEF-II Sub Domain		
Inhibition	45.8 (1.2) [43.3, 48.2]	43.7 (1.2) [41.2, 46.1]
Self-Monitoring	46.3 (1.4) [43.5, 49.1]	43.9 (1.1) [41.6, 46.2]
Flexibility	44.2 (1.1) [41.9, 46.5]	42.7 (1.1) [40.5, 44.9]
Emotional Control	45.2 (1.1) [43.1, 47.3]	44.6 (0.9) [42.7, 46.4]
Task Completion	46.4 (1.1) [44.2, 48.7]	45.3 (1.2) [43.0, 47.7]
Working Memory	46.4 (1.2) [43.9, 49.0]	45.1 (1.3) [42.4, 47.8]
Planning/Organizing	46.4 (1.5) [43.4, 49.4]	45.3 (1.4) [42.5, 48.1]

3.4 Pre-Treatment Executive Function as a Moderator

3.4.1 Multiple linear regression tables

These tables include the coefficients, standard errors, t-values, and p-values for all predictors and interactions in the regression models predicting CY-BOCS post-treatment based on CY-BOCS pre-treatment, EF pre-treamtent, and interactions with treatment group.

Supplementary Table S18 Multiple Linear Regression Results for Parent-Report (child age: 8-17)

Predictor variable*	*β*	*95 % CI [lower, upper]*	*p*
Intercept	17.69	[16.02, 19.35]	**<.001**
CY-BOCS pre-treatment	0.65	[0.24, 1.06]	**.002**
Treatment Group	-2.65	[-5.98, 0.68]	.117
BRIEF-II sub-domain			
Inhibition	0.02	[-0.26, 0.29]	.911
Self-Monitoring	-0.02	[-0.25, 0.21]	.873
Flexibility	0.01	[-0.29, 0.31]	.943
Emotional Control	0.03	[-0.24, 0.31]	.809
Initiating	0.27	[-0.01, .055]	.054
Working Memory	-0.60	[-0.97, -0.23]	**.002**
Planning/Organizing	0.33	[-0.01, 0.66]	.054
Task Monitoring	0.11	[-0.16, 0.39]	.405
Organization of Materials	-0.13	[-0.43, 0.18]	.410
Interaction terms			
Inhibition*Treatment Group	0.19	[-0.37, 0.75]	.511
Self-Monitoring*Treatment Group	0.20	[-0.28, 0.68]	.411
Flexibility*Treatment Group	0.39	[-0.22, 0.99]	.205
Emotional Control*Treatment Group	-0.70	[-1.25, -0.14]	**.014**
Initiating*Treatment Group	0.18	[-0.38, 0.74]	.523
Working Memory*Treatment Group	-0.13	[-0.88, 0.62]	.726
Planning/Organizing*Treatment Group	0.08	[-0.58, 0.74]	.807
Task Monitoring*Treatment Group	-0.27	[-0.82, 0.28]	.329
Organization of Materials*Treatment Group	-0.04	[-0.64, 0.56]	.895

Note: *) All predictor variables are centered to have a mean of 0.

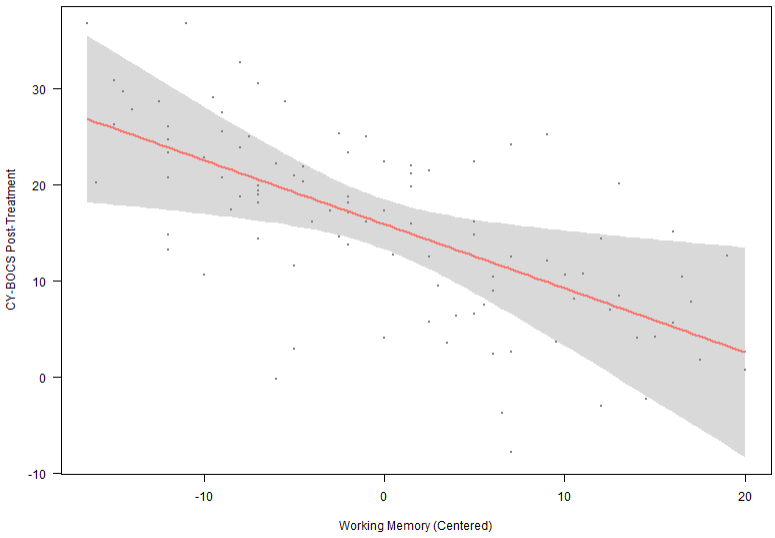
Supplementary Table S19 Multiple Linear Regression Results for Self-Report (child age: 11-17)

Predictor variable*	*β*	*95 % CI [lower, upper]*	*p*
Intercept	17.48	[15.67, 19.29]	**<.001**
CY-BOCS pre-treatment	0.49	[0.01-0.97]	**0.044**
Treatment Group	-2.88	[-6.53, 0.76]	.119
BRIEF-II sub-domain			
Inhibition	-0.13	[-0.41, 0.15]	.366
Self-Monitoring	-0.17	[-0.41, 0.07]	.157
Flexibility	0.01	[-0.22, 0.25]	.911
Emotional Control	0.20	[-0.05, 0.44]	.118
Task Completion	0.40	[0.15, 0.66]	**.003**
Working Memory	-0.35	[-0.65, -0.04]	**.025**
Planning/Organizing	0.24	[-0.08, 0.57]	.142
Interaction terms			
Inhibition*Treatment Group	0.42	[-0.15, 0.99]	.145
Self-Monitoring*Treatment Group	-0.12	[-0.57, 0.34]	.616
Flexibility*Treatment Group	0.06	[-0.36, 0.48]	.791
Emotional Control*Treatment Group	-0.66	[-1.16, -0.16]	**.011**
Task Completion*Treatment Group	0.53	[0.01, 1.05]	**.044**
Working Memory*Treatment Group	-0.26	[-0.87, 0.36]	.409
Planning/Organizing*Treatment Group	-0.17	[-0.82, 0.48]	.605

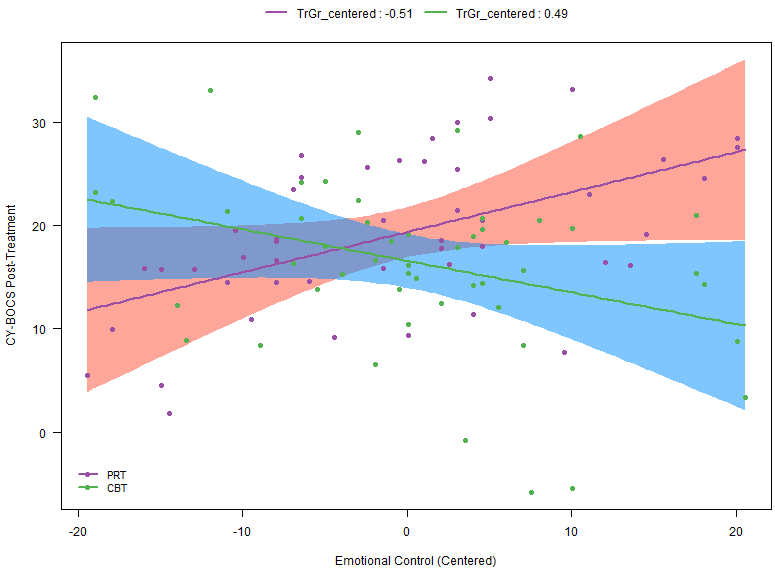
Note: *) All predictor variables are centered to have a mean of 0.

3.4.2 Plots of predictors and interactions

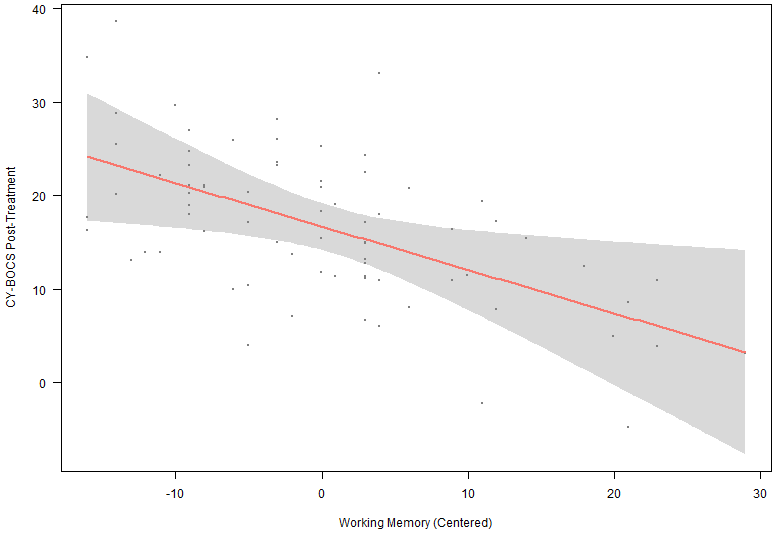
Supplementary Figure S12 Plot: Working Memory as a Unique Predictor in the Multiple Linear Regression Model (Parent-Report; child age: 8-17)



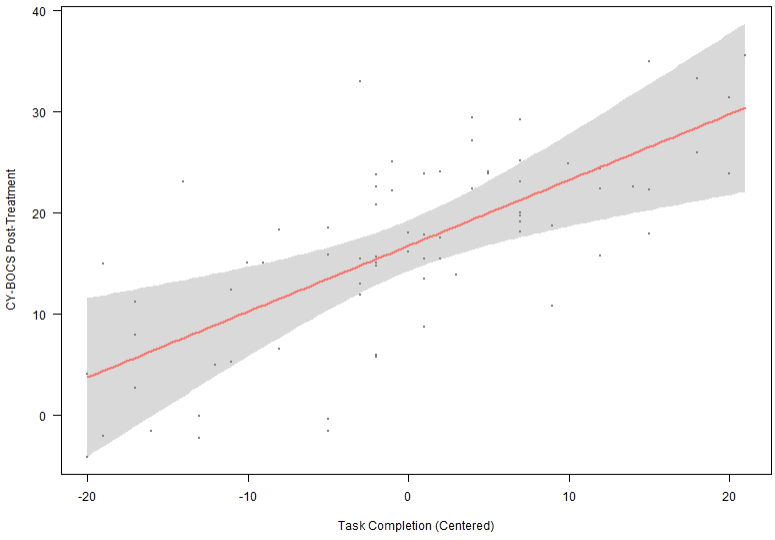
Supplementary Figure S13 Plot: Emotional Control as a Moderator in the Multiple Linear Regression Model (Parent-Report; child age: 8-17)



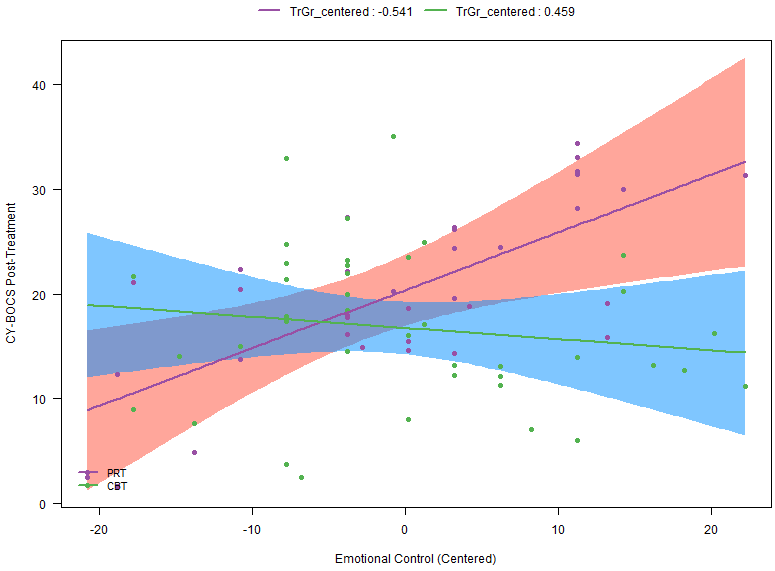
Supplementary Figure S14 Plot: Working Memory as a Unique Predictor in the Multiple Linear Regression Model (Self-Report; child age: 11-17)



Supplementary Figure S15 Plot: Task Completion as a Unique Predictor in the Multiple Linear Regression Model (Self-Report; child age: 11-17)



Supplementary Figure S16 Interaction Plot: Emotional Control as a Moderator (Self-Report; child age: 11-17)



Supplementary Figure S17 Plot: Task Completion as a Unique Predictor in the Multiple Linear Regression Model (Self-Report; child age: 11-17)