



Review article

An interrogation of cognitive findings in pediatric obsessive–compulsive and related disorders

Elle Brennan*, Christopher Flessner

Department of Psychological Sciences, Kent State University, Kent Hall, 600 Hilltop Drive, Kent, OH 442440, USA



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ABSTRACT

Current findings in the field of psychology have led to increased interest and a new conceptualization of disorders characterized by repetitive behaviors, namely the obsessive compulsive and related disorders (OCRDs). Scant research, however, has sought to collect and categorize the extant research on pediatric OCRDs. Particularly, no adequate review of the pediatric cognitive literature existed until now, despite the clear implication of abnormalities in neuroanatomical structures and cognitive functioning in adult samples. While evidence for cognitive dysfunction in pediatric samples is presented, this paper also suggests that differences in cognitive dysfunction may indeed exist between adults and youth with OCRDs. Specifically, those irregularities present in said youth at varying developmental stages may impact the origination and maintenance of OCRDs across time. Finally, this paper seeks to formulate potential future goals for the research field, particularly through transdiagnostic approaches to processes linked with symptom presentations. This is of particular importance as an improved understanding of the interaction of cognitive function and growth is key to further comprehension of the OCRDs.

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1. Introduction

Recent developments in the understanding of Obsessive Compulsive Disorder (OCD), particularly the neurobiological underpinnings of the disorder, have led to increased interest in other

conceivably related disorders with notable repetitive behaviors. In the Diagnostic and Statistical Manual (DSM) 5 (American Psychiatric Association, 2013) these disorders have been referred to as obsessive–compulsive and related disorders (OCRDs) and primarily consist of trichotillomania (hair pulling disorder, HPD), excoriation or

* Corresponding author. Tel.: +1 330-672-2200.
 E-mail address: ebrenna2@kent.edu (E. Brennan).

pathological skin picking disorder (PSP), hoarding disorder (HD), and body dysmorphic disorder (BDD).

1.1. A brain–behavior approach to understanding OCRDs

While current DSM diagnostic criteria for some OCRDs have been used reliably in clinical applications, many diagnostic categories often fail to match up with other domains of psychological research, such as clinical neuroscience and genetics. Other etiological approaches may offer a better fit to explain these disorders. For example, several researchers have conceptualized OCRDs as falling along a spectrum of compulsivity and impulsivity (Stein et al., 1993; McElroy et al., 1994; Lochner et al., 2005; Ferrão et al., 2006; Stein and Lochner, 2006). These lines of inquiry have generally led to the perception that OCD represents the compulsivity end of the spectrum and disorders such as HPD and PSP fall more towards the impulsivity end (Hollander et al., 1988; Fineberg et al., 2010; Snorrason et al., 2011). This notion is lent further credence by a recent behavioral genetics study demonstrating that two sub-clusters may exist within the broader clustering of OCRDs: (1) OCD, BDD, and HD and (2) HPD and PSP (Monzani et al., 2013). What is more, leading evidence supports OCD as a biological disorder with noted dysfunction in certain neural circuits, as well as with specific cognitive deficits (Rosenberg and Keshavan, 1998; Stein, 2000b; Stein, 2002; Mataix-Cols and van den Heuvel, 2006). Given the above-mentioned prior research and the characteristic obsessive, compulsive, and/or ritualistic behaviors marking these disorders, similar cognitive traits may be common across OCRDs (Stein, 2000a; Chamberlain et al., 2006; American Psychiatric Association, 2013). A neurobiological approach to understanding the cross-cutting behaviors characteristic of multiple OCRDs is well matched with the National Institute of Mental Health (NIMH's) Research Domain Criteria (RDoC) Initiative, an effort to combine the findings of genomics, neuroscience, and clinical observation to inform science's understanding of psychopathology (Insel et al., 2010). The RDoC places an emphasis on brain–behavior relationships across disorders, is agnostic to discrete diagnoses, and has greatly informed our review.

The current review aims to provide an overview of neurobiological and, more prominently, cognitive risk factors across pediatric disorders marked by obsessive, compulsive, and repetitive or ritualistic behaviors – namely OCRDs. With that fact in mind, scant research has examined such risk factors in OCRDs – aside from OCD. Said deficiency in the literature is manifested most profoundly and is most concerning with regards to exploration of pediatric samples. For example, while studies on cognitive impairment in childhood OCD exist, they are few and what results have been presented are inconsistent and not reliably replicated (Andres et al., 2007). Moreover, no recent comprehensive review has been published exploring the array of literature regarding cognitive deficits present in youths across the OCRDs. Therefore, the primary aim of this paper is to provide a substantial review of the current findings on the subject of the neurobiological/cognitive underpinnings of pediatric OCRDs with the aim of informing future transdiagnostic research. However, by necessity – due to scant research in this area – evidence from the adult literature will be discussed as well.

2. Neuroanatomical discoveries in pediatric OCRDs

2.1. Overview of adult and pediatric findings in OCD

Habitual and repetitive acts, which are characteristic of many OCRDs, are normative during childhood (Evans et al., 2004; Petrefesa and Evans, 2007), though maturation of the prefrontal cortex may result in a natural “top-down” suppression of these

acts (Beers et al., 1999; Savage and Rauch, 2000). Current literature describes an array of neuroanatomical findings specific to some OCRDs in both adults and children with variations noted in both structural cerebral anatomy (i.e. gray matter density) and functional aspects of the brain (i.e., cerebral blood flow, metabolic functioning). Due to the complexity of the anatomical presentation of these disorders, however, this topic will not be covered in detail in this paper. Therefore, what follows is an overview, albeit brief, in regards to posited neuroanatomical models of OCRDs. When possible, evidence specific to work with pediatric populations is highlighted.

Prominent neuroanatomical research has provided considerable credibility in advocating for the centrality of the cortico-striatal-thalamic-cortical (CSTC) loop in the etiology of OCD (Insel, 1992; Rauch and Baxter, 1998; Mataix-Cols and van den Heuvel, 2006). The CSTC is comprised of two pathways that initiate within the striatum. One pathway ultimately leads to increased inhibition of the thalamus and prefrontal cortex, while the other results in increased activation of the prefrontal cortex. Research on the CSTC has linked dysfunction in said circuits to deficits of implicit processing and intrusive symptoms (Rauch et al., 1997). Furthermore, evidence of abnormal basal ganglia volume (Saxena et al., 1998), though mixed, may expound some of the motoric aspects of OCD, and perhaps of the other related disorders.

Despite a dearth of research compared to their adult counterparts, research in children with OCD has generally demonstrated support for the central role of the CSTC (Rosenberg and Keshavan, 1998; Rosenberg et al., 2007). Abnormalities in white matter and gray matter in children have also been found to be comparable, if not more abnormal, to those found in adults (Macmaster et al., 2010; Zarei et al., 2011). Yet mixed findings are common as well. For example, Rosenberg and Keshavan (1998) and Szeszko et al. (2004) both failed to find differences in certain brain structures in child samples that had previously been indicated in adults (e.g. amygdala, hippocampus). Imaging studies have also found the striatum and thalamus to play a more substantial role in childhood OCD (Rosenberg et al., 1997; Fitzgerald et al., 2000; Gilbert et al., 2000; Rosenberg et al., 2000; Smith et al., 2003).

2.2. Linking findings in other OCRDs in adults

Although based upon fewer studies as compared to the OCD field, available evidence suggests that other OCRDs share a number of the more conspicuous neuroanatomical features found in OCD. It should be noted, however, that complete neurobiological overlap has not been found. This is particularly true with regards to HD where several studies have noted distinctive neuroanatomical qualities (Winsberg et al., 1999), notably in cerebral blood flow and glucose metabolism (Saxena et al., 2004; Tolin et al., 2009), as well as in activation of the left prefrontal gyrus and right orbitofrontal cortex (Mataix-Cols et al., 2004). To this point, neuroimaging studies in youth with symptoms of HD are non-existent.

Despite the scant and somewhat inconsistent findings within the HD literature, neurobiological overlap amongst the other OCRDs is more commonly found. For example, in the only PSP neuroimaging study to date, Grant et al. (2013) found abnormalities in the anterior cingulate cortices (ACC), regions implicated in the CSTC. What is more, the ACC has also been strongly implicated in HPD (Chamberlain et al., 2010). Each disorder is also characterized by unique structural and functional aspects which likely contribute to the distinguishing phenotypic presentations of the disorders. Generally speaking, neuroimaging studies in adults with HPD have been mixed, with some indicating the presence of structural abnormalities (Swedo et al., 1991; Keuthen et al., 2007; Chamberlain et al., 2008, 2010) and others indicating no abnormalities (Stein et al., 1997;

Table 1
Cognitive functioning studies in obsessive–compulsive related disorders.

Cognitive functioning in pediatric OCRDs compared to healthy controls					
Study	Sample	Age	Comorbidity	Tasks Administered	Findings
Behar et al., 1984	17 OCD 16 HC	14 years (means)	<u>OC</u> D: 3 MDD <u>HC</u> : none	MRM, StyM, RAVLT, CFT, dihapctic testing, SRT, two-flash threshold	OC D < planning and motor function
Cox et al., 1989	42 OCD early onset 35 HC	8–18 years	Not reported	StyM, WCST, MRM, visual recognition (words, patterns), CFT, RAVLT, auditory task, dihapctic encoding task,	OC D < verbal performance, set shifting, visual copy and recall, planning
Beers et al., 1999	21 OCD 21 HC	OC D: 12.2 years HC: 12.3 years (means)	None	Stroop, WCST, COWA, TOH, GNG, CVLT, Pegboard, WISC (digit span, block design), TMT	OC D > motor inhibition, verbal fluency and cognitive inhibition
Andres et al., 2007	35 OCD 35 HC	7–18 years	None	WISC (block design, coding, digit span), WMS, CFT, RAVLT, TMT, WCST, COWA, Stroop	Controlled: depression; OC D < verbal and visual memory, organization, attention, information processing speed, cognitive inhibition, cognitive flexibility
Petrefesa and Evans, 2007	22 Group1 20 Group2 (OCD)	Group1: 4–5 years Group2: 5–8 years	Not reported	Group1: DCCS, Livesey, K-CPT Group2: WCST, Stroop, CPT Both: GLST, Simon Says	Group1 < executive functioning
Chang et al., 2007	16 OCD 15 TS 15 HC	7–17 years	Not reported	OAT, CFT, Stroop, CVLT, Digit Span, ACT, Finger Windows	OC D = TS; OC D < cognitive inhibition, attention, and memory than HC
Shin et al., 2008	17 OCD 25 ADHD 21 Tic 20 MD 23 HC	6–16 years	None	WISC-R (all), CPT, WCST, TMT-B, CFT	OC D < cognitive flexibility than HC; OC D < visual and auditory attention than ADHD
Andres et al., 2008	29 OCD 22 HC	7–18 years	None	WMS, WISC, CFT, RAVLT, TMT, WCST, Stroop, COWA	OC D < verbal and visual memory, information processing speed, and executive functioning
Schwartz et al., 2009	1 OCD (case study)	13 years	<u>OC</u> D: ADHD, Tic	WAIS, behavioral assessment	< motor functioning, executive functioning (planning, impulse inhibition, focus/attention, self-regulation/frustration tolerance)
Zandt et al., 2009	17 OCD 19 ASD 18 HC	7–16 years	None	WISC-III (block design, similarities, vocabulary, coding), NEPSY (verbal fluency), CGT, CFT, TEACH (Walk, Don't Walk) SRT; delayed aversion	OC D and HC > verbal fluency than ASD
Vloet et al., 2010	20 OCD 20 ADHD 25 HC	10–18 years	<u>OC</u> D: 2 Tic, 4 Anx, 2 ODD <u>ADHD</u> : 2 Tic, 3 Anx, 3 ODD <u>HC</u> : 2 Anx, 1 ODD	SRT; delayed aversion	OC D < simple reaction time than ADHD & HC; ADHD less likely to choose larger, delayed reward than both OC D and HC
Britton et al., 2010	15 OCD 20 HC	10–17 years	<u>OC</u> D: 2 GAD, 2 SP, 1 AG; 3 MD, 1 Tic, 1 ADHD <u>HC</u> : none	Set shifting	OC D < performance on mixed vs. repeated trials
Ornstein et al., 2010	14 OCD 24 HC	8–16 years	<u>OC</u> D: 2 TS, 2 GAD <u>HC</u> : not assessed	SSP, WCST, spatial span, N-back test, DKEFS, TMT, Tower, SDMT, CVLT, CFT	OC D trends < psychomotor speed and attention, cognitive flexibility, planning, and verbal working memory
Rettew et al., 1991	21 HPD 12 OCD 17 CC 16 HC	HPD: 16–44 years OC D: 20–46 years CC: 21–51 years HC: 16–45 years	<u>HPD</u> : 2 Panic, 9 GAD, 9 MDD, 2 BP, 7 SUD <u>OC</u> D: 10 MDD, 1 SUD, 3 Panic, 4 BP <u>CC</u> : 10 SA, 5 Panic+AG, 1 Panic, 1 GAD <u>HC</u> : none	MRM, StyM	Controlled: med status HPD < spatial memory than HC; OC D trend < spatial memory than HC; CC > rule violations than HPD and HC; HPD and OC D > rule violations than HC
Longitudinal predictors of adult-diagnosed OCD					
Study	Sample	Age	Comorbidity	Tasks Administered	Findings
Grisham et al., 2009	13 OCD (as adults) 687 other	32 years at diagnosis	<u>OC</u> D: 5 MDD, 4 panic, 4 GAD, 3 SP, 2 SA, 2 SUD	WISC-R (selected subtests including Mazes), CFT, RAVLT, TMT, Pegboard, COWAT	OC D < perceptual organization, visual memory, mazes, and motor control
Cognitive outcomes following treatment of pediatric OCRDs					
Study	Sample	Age	Comorbidity	Tasks Administered	Findings
Flessner et al., 2010	63 OCD	7–17 years	25 GAD, 18 SP, 11 SAD, 11 Tic, 9 ADHD, 7 ODD	ROCF, WISC (info, coding, block, vocab, object assembly)	Nonresponders < percent recall, particularly those who received CBT alone
Andres et al., 2008	29 OCD 22 HC	7–18 years	None	WISC (vocab, block, digit, coding), WMS-III (LM,VR), CFT, RAVLT, TMT, WCST, COWA, Stroop	Pre-Treatment: OC D < verbal and visual memory, speed of info processing, some executive functions Post-Treatment: OC D < verbal memory

OC D=Obsessive Compulsive Disorder; HC=Healthy Controls; CC=Clinical Controls; ADHD=Attention-Deficit Hyperactivity Disorder; TS=Tourette Syndrome; Tic=Any Tic Disorder; ODD/CD=Oppositional Defiant Disorder/Conduct Disorder; Anx=Anxiety Disorder; GAD=Generalized Anxiety Disorder; SP=Simple Phobia; SA=Social Anxiety, AG=Agoraphobia; MD=Major Depression or Depression NOS; DD=Dysthymic Disorder; SUD=Substance Use Disorder; BP=Bipolar Disorder.

WMS=Wechsler Memory Scale-Logical Memory (LM)/Visual Reproduction (VR); OAT=Object Alteration Task; CFT=Complex Figure Task; RAVLT=Rey's Auditory Verbal Learning Test; TMT=Trail Making Test; WCST=Wisconsin Card Sorting Test; Stroop=Stroop Test; COWA=Controlled Oral Word Association; CVLT=California Verbal Learning Test; Digit Span=subscale of WISC; Finger Windows=subtest of Wide Range Assessment of Memory and Learning; ACT=Auditory Consonant Trigrams; SSP=Stop Signal Paradigm; Spatial Span=subscale of WISC-III; D-KEFS=The Delis-Kaplan Executive Function System; Tower=D-KEFS Tower Test; SDMT=The Symbol Digits Modalities Test; CPT=Continuous Performance Test; GNG=Go-No-Go Task; Pegboard=Grooved Pegboard; TOH=Tower of Hanoi; NEPSY=A Developmental Neuropsychological Assessment; CGT=Concept Generation Test; TEACH=Test of Everyday Attention for Children; CPT-II=Conner's Continuous Performance Test II; ADT=Auditory Discrimination Test; Token=Token Test; BNT=Boston Naming Test; FRT=Facial Recognition Test; JLO=Judgment of Line Orientation Test; TPT=Tactical Performance Test; FTT=Finger Tapping Test; LNNB=Luria Nebraska Neuropsychological Battery; BVRT=Benton Visual Recognition Test; Booklet=Booklet Category Test; VSAT=Visual Search and Attention Test; PASAT=Paced Auditory Serial Addition Test; MFFT=Matching Familiar Figures Test; FDWT=Fused Dichotic Words Test; Info=Information Sampling Task; MRM=Money's Road Map; StyM=Stylus Maze; OAT=Object Alteration Task; SRT=Simple Reaction Time; DCCS=Dimensional Change Card Sort; GLST=Global-Local Stroop Task; K-CPT=Kiddie-Continuous Performance Test; CPT=Conner's Continuous Performance Test; Livesey=task of response inhibition.

Rauch et al., 2007). Specifically, decreased volume of the left putamen may lead to sensorimotor symptoms (O'Sullivan et al., 1997; Rauch and Baxter, 1998) such as those evidenced by patients with HPD engaging in "automatic" (i.e., pulling outside of one's awareness) pulling. Additionally, increased metabolic rates in the cerebellum and right superior parietal lobe may be present in adults with HPD (Swedo et al., 1991). Chamberlain et al. (2008) have suggested brain regions implicated in HPD to be associated with habit learning, affective regulation, and cognition, the dysregulation of which may directly correlate with symptoms of HPD.

Unfortunately, no imaging studies have been conducted focusing on children with HPD and, aside for the previously cited study, investigations of the neurobiology of PSP in general are regrettably lacking. What research there is relating to brain abnormalities in BDD has suggested increased total white matter and left-biased asymmetry in the caudate (Rauch et al., 2003), increased activation in the left hemisphere, specifically in the inferior frontal gyrus (IFG), and hyperactivity in the amygdala (Feusner et al., 2008), some of which may be particular to facial processing. Conversely, a successive study found no differences in brain density, or in activation of the IFG, caudate, or amygdala (Feusner et al., 2009). While research on these topics is growing, more work is needed to explicate the overall variation and inconclusiveness seen throughout neuroanatomical research in the OCRDs.

Although reports across disorders and researchers have been, at times, inconsistent, neuroimaging studies have assisted in unveiling the mystery of which neuroanatomical structures are implicated in a number of the OCRDs. The obvious lack of research looking at neuroanatomy in children with OCRDs is disconcerting, however. What the available research has led to is the proposal of a seemingly pivotal relationship between abnormalities in cerebral structure and functioning and a number of cognitive deficits. For instance, executive functioning (including impulse control and inhibition of behaviors), and thus dysfunction, has been linked with the dorsolateral prefrontal cortex, thalamus, and striatum (Robbins et al., 1996; Purcell et al., 1998), regions implicated in the pathophysiology of several OCRDs. Similarly, modulatory control (ability to regulate socially appropriate behaviors), which may be involved in the blind focus connected with obsessions in OCD, implicates the orbitofrontal cortex, medial prefrontal cortex, and cingulate gyrus in adults (Saxena et al., 1998; Friedlander and Desrocher, 2006), regions also implicated in other OCRDs. As such, we now turn our attention to available evidence linking deficits in performance on cognitive tasks and domains to OCRDs.

3. A cognitive approach to pediatric OCRDs

Abnormal cognitive functioning has been postulated to be central to each of the OCRDs with Table 1 providing an overview of available studies within the pediatric realm. Typically, researchers have sought to investigate functioning across a broad array of cognitive domains such as memory (logical recall, visual recall), visual organization, processing speed, cognitive flexibility, impulse control, and set shifting. Measurement of cognitive abilities can be particularly challenging in pediatric samples, however, as cognitive abilities vary greatly across normative age groups. Despite this, there are a number of methods and measures that can be utilized in both children and adults, including paper-and-pencil measures as well as automated computer batteries, such as the Cambridge Neuropsychological Test Automated Battery (CANTABeclipse; (Cambridge Cognition Limited, 2006). Such advances in neuropsychological testing have allowed for increases in ease of administration and interpretability of findings, hopefully leading to an increase in cognitive investigations across OCRDs.

3.1. Cognitive functioning in pediatric OCD

A significant body of research has been conducted linking specific neurological functions and pathways in the symptomatology of OCD, including many of the cognitive functions mentioned above (Chamberlain et al., 2005; Burdick et al., 2008). While there is some evidence for fluidity between symptom presentations in both adults and youth (i.e., Swedo et al., 1989; Rettew et al., 1992), differences in *in vivo* neural correlates may exist (e.g., Cottraux et al., 1996; Rauch et al., 1998; Phillips et al., 2000; Mataix-Cols et al., 2004). Furthermore, while much of the research utilized small sample sizes and findings have not been readily replicated, several studies have suggested differences in cognitive performance between subtypes of OCD in adults (McKay et al., 2004). For example, Nedeljkovic et al. (2009) and Omori et al. (2007) both reported differences in cognitive functioning between OCD subtypes, with greater deficits for "checkers." Kyrios and colleagues (1999) similarly reported differences in performance on a pattern recognition task and planning task between washers, checkers, and obsessional. However, these effects disappeared when symptom severity was accounted for. Penades et al. (2007) also found no differences in reduced functioning between OCD subtypes. Additionally, no such research has yet been published utilizing a pediatric OCD sample. A bimodal distribution has, however, been proposed with early- and late-onset cases of OCD exhibiting differing underlying pathophysiology (Busatto et al., 2001; Roth et al., 2005), though much of the extant research has been focused on adults. Interestingly, Roth et al. (2005) found that adults with later-onset OCD exhibited greater deficits in executive functioning and auditory attention than adults who developed OCD earlier in life. Later-onset OCD was also associated with worse visual memory relative to controls. A unique longitudinal study conducted by Grisham et al. (2009) examined neuropsychological performance at age 13 and then followed individuals until age 32. The authors found that participants receiving a diagnosis of OCD at age 32 had shown impaired performance on visuospatial, visuo-constructive, and visuomotor skills, though findings regarding executive functioning were mixed with deficits noted in planning and organization but not in other areas such as set-shifting. Adding to the confusion, Mushtaq and Helal (2009) commented that, in the same sample, those given diagnoses of OCD at age 18 did not show cognitive differences compared to performance at 13. This research, unfortunately, mirrors much of the extant literature interrogating cognitive domains in relation to pediatric OCD and related disorders.

Unsurprisingly, findings from research specifically examining pediatric OCD are abundantly mixed, expressly within the cognitive literature. For example, an early study of cognitive deficits in pediatric OCD evidenced frontal dysfunction in 16 adolescents when compared to normal controls (Behar et al., 1984). This study was replicated by Cox et al. (1989) in a sample with an average age of 14 years indicating that children with OCD demonstrate deficits specifically in executive tasks and nonverbal memory when compared to healthy controls. Beers et al. (1999), however, found no impairment in performance on frontal tests in a slightly younger sample. Andres et al. (2007) investigated performance on cognitive tasks in youth with OCD without comorbid psychiatric disorders, both including and excluding a younger sample (7–11 years). Including the younger sample, youth with OCD performed worse on tasks involving logical memory, visual memory, velocity, and visual organization. When younger children were removed from the analysis, group differences in logical memory were no longer evident. Chang et al. (2007) found deficits in spatial attention in children (ages 9–17) with OCD, but not in executive functioning, visual spatial function, or motor function. Using a slightly younger sample (mean age 12 years), Shin et al.

(2008) found evidence for executive dysfunction, specifically in set shifting. More recently, Ornstein et al. (2010) demonstrated deficits in cognitive flexibility and planning in rather young children (mean age 6 years) with OCD, similar to the deficits in executive functioning found in very young children by Petrefesa and Evans (2007), but failed to exhibit deficits in response inhibition or memory deficits, which have been found in adults. Another study also failed to find problems with inhibition in children with OCD but did note impaired sequence learning (Vloet et al., 2010) while another noted cognitive inflexibility (Britton et al., 2010). Though often contrasting, these collective findings appear to suggest that some cognitive deficits associated with OCD in adulthood (i.e., inhibition) may not appear until additional development of the frontal lobe occurs later in adolescence. Furthermore, it is possible that normative improvements in executive functioning may be delayed in youth with OCD as compared to their typically-developing peers. Such inferences may translate across OCRDs.

3.2. Cognitive outcomes in the other OCRDs in adults

While the majority of research looking at hoarding as a self-contained disorder is extremely recent, it has been proposed that adults who exhibit HD behaviors may show difficulty with focusing and maintaining attention (Hartl et al., 2005), as well as with decision-making about personal possessions (Frost et al., 1995; Steketee et al., 2003; Hartl et al., 2004b; Tolin et al., 2008). Limited evidence suggests that adults with HD exhibit difficulties in delayed visual and verbal recall, utilize less effective organizational strategies (Hartl et al., 2004b), show slowed reaction time and difficulties in spatial attention (Grisham et al., 2007), and demonstrate shortfalls in planning and problem solving abilities (Grisham et al., 2010). Some research has even suggested cognitive deficits in a nonclinical hoarding sample (Luchian et al., 2007). No literature yet exists exploring cognitive characteristics specific to children with HD. Research on cognitive performance in adults with HPD has shown evidence for an array of cognitive deficits including impulse control problems (e.g., reaction time, motor inhibition) and issues with attention (Keuthen et al., 1996; Stanley et al., 1997; Chamberlain et al., 2006; Bohne et al., 2008), though individuals with HPD may show fewer deficits than those with OCD (Chamberlain et al., 2006, 2007). One study found deficits in both OCD and HPD, albeit in different areas (i.e., impaired feedback integration in OCD and impaired response flexibility in HPD (Bohne et al., 2005). Akin to OCD, HPD has been speculated to present in a bimodal fashion (Swedo and Rapoport, 1991), with early-onset HPD manifesting during the preschool years and often resolving without intervention (O'Sullivan et al., 1997; Papadopoulos et al., 2003). Though discussions regarding early-onset HPD are at this point largely speculation due to limited research, comparisons with later-onset HPD may still provide informative results. Odlaug and colleagues (Lochner et al., 2012) compared adults whose symptoms began either earlier or later during childhood, finding greater impairment in response control in later-onset subjects, but greater set-shifting difficulties in early-onset subjects. At this time, no investigations have looked at cognitive functioning in HPD around the actual time of onset during childhood.

As discussed previously, comparisons of early- (prior to 11 years old) and late-onset (after 11 years old) PSP have also been conducted. Investigating differences in cognitive functioning between adults with either early- or late-onset PSP, Grant et al. (2012) found both groups to evidence worse inhibition; however, only individuals with late-onset PSP showed significant impairment in cognitive flexibility on a task of set-shifting. Earlier studies by the same authors found comparable results (Odlaug et al., 2010; Grant et al., 2011). Schuck et al. (2012) reported that adults with PSP exhibit slowed reaction times to images of flawed skin versus

healthy skin that was not seen in healthy controls. Adults with PSP have also been found to be more impulsive than healthy controls (Snorrason et al., 2011). Unfortunately, research investigating cognitive domains in pediatric presentations of PSP is scarce. Findings specific to adults with BDD have suggested aspects of executive functioning to be impaired (Hanes, 1998; Veale, 2004; Dunai et al., 2010). Deckersbach et al. (2000) found evidence of a deficit in memory strategies as evidenced by decreased performance on verbal and nonverbal learning and memory directories as compared to controls. The piecemeal performance demonstrated on these tasks was reminiscent of how individuals with BDD process faces, reflecting some of the neurological abnormalities discussed earlier. Much like with other OCRDs, exploration of potential cognitive aspects of BDD in child and adolescent samples has not been realized. Considering the evidence reviewed thus far, it is unavoidable to note how little is understood about the course and presentation of cognitive characteristics of pediatric OCRDs, particularly HPD, SPD, HD, and BDD. The evidence presented thus far clearly points to a need for further research in the area of cognitive functioning in OCRDs – both in a typical (i.e., comparison between disorders and healthy controls) and transdiagnostic (i.e., presence vs. absence of repetitive behaviors) fashion, as such research may better inform future treatment design and implementation.

3.3. Interaction of treatment and neurological aspects of pediatric OCRDs

Perhaps enforcing this argument for further research among pediatric populations, Flessner et al. (2010) indicated a worrisome interaction between existing cognitive dysfunction and treatment outcomes in a pediatric sample, specifying that worse executive functioning may impede the benefits of treatment. Other authors have suggested similar deleterious associations between cognitive deficits and treatment in both adults (Hollander et al., 1990) and children (March et al., 1990) while yet others have not found such a relationship (Leonard et al., 1989; Swedo et al., 1990). Andres et al. (2008) conducted the first study to investigate the stability of neuropsychological deficits in children and adolescents following treatment for OCD. The authors found that deficits – including verbal memory, visual memory, information processing speed, and response inhibition – appear to normalize with successful naturalistic treatment of symptoms. While minor improvements were found in a healthy control group, these improvements were not comparable to those children with OCD receiving treatment. Rettew et al. (1991) even indicated a link between performance on a task of memory and motor control and HPD symptoms severity in adults, as well as response to pharmacological treatment. In a review of literature on SSRI treatment for pediatric OCD, Rosenberg et al. (2001) also found evidence for improvements in neuroanatomical aberrations following successful treatment.

It has been suggested that the findings described above may be due to the plasticity of the brain during the developmental stages of life (Andres et al., 2008). Research among adults with OCD has generally shown disconcerting evidence for the persistence of cognitive deficits following successful treatment of symptoms (Nielen and den Boer, 2003; Chamberlain et al., 2005; Roh et al., 2005; Bannon et al., 2006) though some exceptions within the OCD (Moritz et al., 2001; Kuelz et al., 2006) as well as the PSP (Schuck et al., 2012) literature do exist. These stable deficits seen more often in adults may represent a missed opportunity for early intervention and supports the need for more research and better informed intervention closer to the age of onset for pediatric OCRDs, particularly with respect to cognitive deficits that may ultimately become treatment resistant. To our knowledge, no studies have examined the interaction between cognitive

functioning and treatment amongst other pediatric OCRDs. Despite obvious evidence implicating cognitive functioning and dysfunction in the OCRDs, the lack of conclusive evidence regarding the specific roles of such functions in the etiology and maintenance of these disorders is concerning.

4. Discussion and future directions

This paper, to our knowledge, is the first to review what literature exists on cognitive functioning in pediatric OCRDs. Abnormalities in neuroanatomical structures, among other things, have been clearly implicated in the presentation of OCRDs in adults. Of particular interest in the findings discussed in this review, is the evidence suggesting that differences in cognitive functioning may exist between adults and youth with OCRDs. An improved understanding of this potential phenomenon is crucial as research often relies on adult findings to explain features of psychopathology in children. The literature reviewed herein also suggests a number of cognitive domains that may impact the development of OCRDs in childhood as well as the maintenance of such disorders throughout the lifespan (i.e., executive functioning, memory, attention). More specific interrogations of the processes linked to a given disorder and across disorders during various developmental periods are needed. Clearly, there are a number of limitations within the current literature that must be addressed before significant advances in the field can be achieved.

What is both a strength and weakness of the literature is that much of the subsequent research has focused on areas of the brain or cognitive functioning that have already been associated with adulthood OCD, potentially overlooking other important areas of the brain or aspects of cognitive functioning that may be uniquely relevant to children with OCRDs. As such, the dearth of research on the other OCRDs as compared to OCD alone cannot be missed. Future research may greatly benefit from efforts to understand disorders such as the OCRDs on a more symptomatic level rather than separately based on individual diagnoses. Instead of focusing on OCD and tailoring further investigations based on previous findings, studies could include individuals with a variety of repetitive behaviors. Additionally, a measure capable of targeting repetitive behaviors of various presentations may allow for more highly inclusive research, as well as to foster a greater understanding of what may be fundamental aspects to understanding the pathophysiology of obsessive, compulsive, and repetitive or ritualistic behaviors across disorders. Such research would also meet with general trends in the field of psychiatry seeking increasingly dimensional approaches to clinical research.

Perhaps most importantly, future research must take a more developmental approach to these disorders. The glaring lack of data on pediatric OCRD samples is again remarkable due to the early age of onset commonly observed in each phenotype. The consistent inconsistency in those findings described in this review further emphasize the need for continued investigation of cognitive functionality across age groups and manifestations of OCRDs, principally regarding the potential repercussions of developmental factors on the brain and cognitive characteristics. For instance, while deficits in executive functioning have been fairly regularly found in adults with OCD (Roth et al., 2005), similar conclusions have not been so consistently realized with OCD in children (Burdick et al., 2008; Chang et al., 2007; Zandt et al., 2009). Additionally, while other domains such as attention and memory have been implicated in many of the other OCRDs as well (Hanes, 1998; Stanley et al., 1997; Hartl et al., 2004a), such little research has been conducted in pediatric samples that

findings from the adult literature cannot reliably be generalized to pediatric samples. This is especially so due to what differences have been seen between adults and children in the neuroanatomy literature (Rosenberg and Keshavan, 1998; Smith et al., 2003) and in cognitive research conducted in children with OCD (Roth et al., 2005; Ornstein et al., 2010). As such, neurological approaches used to describe a common etiology currently remain unsatisfying. The absence of conclusive examinations of neurological aspects of these disorders in children is chiefly concerning considering the substantial incidence of childhood-onset in the OCRDs, especially as this is such a vulnerable and important population.

Additionally, almost all of the current literature on these topics reflects cross-sectional data, further reducing science's ability to truly understand the developmental nature of obsessive and compulsive behaviors. Increased research on pediatric samples will help greatly; however, the literature will continue to be limited until longitudinal research designs are normative. Longitudinal research will also likely strengthen the utility of linking these neurological aspects of the OCRDs to treatment. Despite the existence of several behavioral approaches to treatment for OCRDs that can be effective in both adults and children (Woods and Miltenberger, 1995; Piacentini et al., 2002; Abramowitz et al., 2003), relapse often remains a concern throughout the lifespan (Lerner et al., 1998; Deckersbach et al., 2002; Pallanti et al., 2002; Phillips et al., 2006; Ayers et al., 2011). Treatment response in pediatric OCD samples is also often fairly low (Pediatric Obsessive Compulsive Disorder Treatment Study Team, 2004) and access to and/or knowledge about effective treatments for other OCRDs is often lacking (Krishnan et al., 1985; Twohig et al., 2006). Considering the links between cognitive functioning and treatment outcomes mentioned above, understanding the roles, both positive and negative, that such functions serve in a given disorder or across disorders (i.e., obsessive-compulsive behaviors) is crucial and may initiate significant improvements in treatment design and implementation.

Targeted cognitive interventions, for example, might be beneficial in strengthening any deficits and thus improving neurological outcomes, hopefully directly impacting treatment outcomes such as symptom reduction and stable maintenance of gains. Such specific interventions may also be more easily implementable in daily life (e.g., via computer games) than many other treatments. A greater variety of types of effective treatments may even increase treatment adherence in youth, particularly in younger children who might find playing games to be more appealing than session by session psychotherapy. Unfortunately, no such interventions are currently supported for use in pediatric OCRDs; however, there is some evidence for the efficaciousness of cognitive training in ADHD (e.g., attentional training, working memory training; Toplak et al., 2008), another disorder marked by impulsivity. Further, a recent pilot study has suggested the efficacy of cognitive remediation in improving attention in adults with HD (DiMauro et al., 2014). Attentional bias training has also been suggested to be effective in reducing avoidance and compulsive behaviors in adults with symptoms of OCD (Najmi and Amir, 2010), another line of research that may prove beneficial if extended to pediatric samples. Alternatively, research in the field of cognitive neuroscience has suggested exciting links between neurostimulation (e.g., transcranial magnetic stimulation) and cognitive performance (e.g., Miniussi et al., 2010; Wagner et al., 2009). However, such practices have not yet been translated into therapeutic uses. Alternatively, deep brain stimulation has been found to alleviate symptoms of treatment resistant OCD in adults, often mollifying the negative cognitive effects of such symptomatology (e.g., Sturm et al., 2003; Greenberg et al., 2006). However, such invasive practices may not be best suited for pediatric

samples and further exploration of alternative options for the improvement of cognitive deficits in pediatric OCD is warranted.

Due in large part to the labors put forth by the NIMH, initiatives such as the RDoC will likely help to fuel and encourage more interest in transdiagnostic research. Efforts such as the creation and validation of an age-sensitive repetitive behaviors questionnaire may be a stepping stone towards an upsurge in research focusing on neurobiological and cognitive functioning across development in disorders such as the OCRDs and other phenotypes marked by repetitive, ritualistic, or habitual behavior. Much remains to be done in creating a more thorough conceptualization of what neurobiological and cognitive impairments underlie the OCRDs, across development and across various populations. It is our hope that research of this nature will lead to improved methods for diagnosis, case conceptualization, and treatment of children afflicted with these sometimes devastating psychiatric problems.

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