Journal of Affective Disorders 266 (2020) 585–594

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Research paper

Clinical expression and treatment response among children with comorbid obsessive compulsive disorder and attention-deficit/hyperactivity disorder

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ARTICLE INFO

Keywords:
Paediatric
OCD
ADHD
Comorbidity
CBT

ABSTRACT

Background: Paediatric obsessive-compulsive disorder (OCD) is highly comorbid with other psychological disorders, including attention deficit/hyperactivity disorder (ADHD). Preliminary evidence suggests that youth with comorbid OCD and ADHD may experience greater impairments than children with other comorbidities; however, there is limited research examining the clinical expression and treatment response of these youth.

Methods: Youth (7 to 17 years) with a primary diagnosis of OCD and comorbid ADHD ($n = 40$) were compared a sample of age and gender matched youth with OCD and other comorbidity (without ADHD, $n = 40$). The study investigated symptoms, severity, functioning, comorbidity, family accommodation, in addition to parental psychopathology and rearing styles. Treatment response was investigated at post-treatment and six-month follow-up.

Results: Youth with comorbid OCD and ADHD had fewer sexual obsessions, higher rates of comorbidity, poorer executive functioning and higher family impairment. Families of comorbid youth engaged in significantly more accommodation and reported more negative rearing. Finally, comorbid youth were significantly less likely to be responders or remitters at post-treatment.

Limitations and Conclusions: Limitations include the cross-sectional design, relatively small clinical sample, and lack of an experimental control group of youth with ADHD without OCD. Current approaches to treatment may be improved for youth with comorbid OCD and ADHD by addressing cooccurring anxiety, behavioural difficulties, and maladaptive family accommodation and rearing. Moreover, given pronounced deficits in executive function, these youth may require a stronger initial dose of CBT to achieve an adequate response.

Paediatric obsessive-compulsive disorder (OCD) is a debilitating mental health disorder, affecting between 1% and 4% of children and adolescents (Douglass et al., 1995; Geller, 2006; Heyman et al., 2003; Rapoport et al., 2000; Shaffer et al., 1996; Valleni-Basile et al., 1994; Zohar, 1999). When present during childhood, OCD is associated with severe impairments, including disruption to family functioning and daily routine (Barrett et al., 2001; Cooper, 1996; Stewart et al., 2017), social relationships (Allsopp and Verduyn, 1990; Storch et al., 2006; Thomsen, 2000; Weidle et al., 2014), school functioning (Piacentini et al., 2003; Toro et al., 1992), and overall quality of life (Storch et al., 2007; Weidle et al., 2014). Furthermore, if left untreated, OCD in childhood tends to be chronic and unremitting (Palermo et al., 2011; Pinto et al., 2006; Storch et al., 2004).

A striking feature of paediatric OCD is high rates of comorbidity with other psychiatric disorders. Some studies have found that more than 80% of youth affected by OCD experience at least one other comorbid condition (e.g., Geller et al., 1996; Lewin et al., 2010; Storch et al., 2008; Swedo et al., 1989), and as many as 50 to 60% of children meet criteria for two or more comorbid conditions (Rasmussen and Eisen, 1990; Tanidir et al., 2015). In addition to anxiety and mood disorders, paediatric OCD often co-occurs with attention deficit/hyperactivity disorder (AD/HD), with up to 30% of children with OCD also concurrently meeting criteria for ADHD (Geller et al., 2002; Garcia, 2010; Masi et al., 2006; Storch et al., 2008). Characterised by a persistent pattern of excessive inattention (e.g., lack of attention to detail, distractible, disorganisation) and/or hyperactivity and impulsivity (e.g., fidgety, physically overactive, excessively talkative), ADHD is a neurodevelopmental disorder that has been found to be linked with OCD via shared genetic factors, such that comorbid OCD and ADHD may represent a unique familial subtype (Geller et al., 2007a; 2007b). While research into the unique clinical presentation of comorbid paediatric OCD and ADHD is limited, there is some evidence to suggest children with both disorders may be more impaired (Geller et al., 2003, 2002; Masi et al., 2006; Sukhodolsky et al., 2005),

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https://doi.org/10.1016/j.jad.2020.01.144
Received 20 October 2019; Received in revised form 10 December 2019; Accepted 25 January 2020
Available online 31 January 2020

and have a poorer response to current OCD treatments (e.g., Farrell et al., 2012; Storch et al., 2008), than youth with OCD and other comorbidities. However, currently there is limited research examining broader clinical parameters among these youth, such as OCD symptom subtypes, family variables (accommodation, rearing styles, parental distress), comorbidity patterns and executive functioning. A greater understanding of the clinical expression of OCD with comorbid ADHD, relative to comorbid OCD without ADHD is warranted in order to advance knowledge and improve treatments for these youth who are currently not achieving an adequate response to best-practice cognitive-behavioural therapy (CBT).

1. Clinical characteristics

Children with ADHD experience significant dysfunction and impairments across multiple domains of life including social, school, and family functioning (Barkley, 2002; DuPaul et al., 2001; Mariani and Barkley, 1997). Given that both OCD and ADHD are individually associated with impairment in functioning, it is not surprising that children with both diagnoses experience significant psychosocial adversity. Children with comorbid OCD and externalising disorders (including ADHD symptomatology), compared to children without an externalising disorder, have been found to experience significantly greater OCD symptom severity (Langley et al., 2010), earlier onset of OCD (Masi et al., 2006), poorer school and social functioning (Geller et al., 2003, 2002; Langley et al., 2010; Masi et al., 2006; Sukhodolsky et al., 2005), higher rates of comorbidity with other disorders (Masi et al., 2006), and higher levels of anxiety and depressive symptoms (Geller et al., 2004). In particular, children with OCD and ADHD are also more likely to present with Tic disorders, suggesting an underlying genetic risk for this clustering of disorders associated with cortico-striatal-thalamocortical circuits (Grados and Mathews, 2008; Huisman-van Dijk et al., 2016). Further, research has found that children with comorbid OCD and ADHD may differ in terms of OCD symptomology, with some studies finding OCD and comorbid ADHD is associated with more severe compulsive behaviours (Storch et al., 2010), and more frequent expression of somatic and hoarding symptoms (Frank et al., 2014; Geller et al., 2003; Hacker et al., 2016) relative to children with OCD, but withour ADHD.

2. Family functioning

In childhood, OCD is often associated with profound impairments to family life due to high rates of family accommodation to OCD symptoms, which has been found to be associated with hieghtened parental distress and burden (Lee et al., 2015). Moreover, studies highlight that greater parental distress predicts greater OCD severity and higher functional impairment (Storch et al., 2009), and poorer family functioning (i.e., blame, conflict, low cohesion) predicts poorer response to treatment (Peris et al., 2012a, 2012b)). Whilst there is minimal research dircetly examining the impact of comorbid ADHD and OCD on the family, comorbidity with disruptive behaviours has been found to be associated with greater impairment, including higher family conflict (Langley et al., 2010), higher family accommodation (Storch et al., 2010), and greater parental strain (Storch et al., 2009). Further, stuides which have examined coercive-disruptive behaviours (CDBs) in youth with OCD have found associations with hyperactivity, and with greater family accommodation, child OCD severity and impairement (Lebowitz et al., 2015). Given the increased strain on parents of youth with comorbid externalising disorders and/or symptoms, it is possible that this strain may have a negative impact on the quality of the parent-child relationships and parental rearing specifically. Indeed, families of youth with ADHD are oftentimes characterised by greater parental psychopathology, and less-positive parental rearing practices (i.e., higher overreativity, more criticism, and less rewarding of behaviours; Modesto-Lowe et al., 2008). Moreover, children with externalising disorders (without OCD) have been found to experience lower positive parental rearing (i.e., parental warmth) and greater negative parental rearing (i.e., overprotection and rejection) relative to children with other comorbidities (Muris et al., 1996). A study by Peris and colleagues (2012) examining maternal Expressed Emotion (EE) in paediatric OCD, defined as rearing high on criticism and overinvolvement, found that higher maternal EE was associated with higher externalising symptoms in children, as well as greater parental depression. To date however, there have been no studies which have examined associations of comorbid OCD and ADHD with parental rearing. Given the greater OCD severity and poorer response to treatment among youth with comorbid ADHD and externalising symptoms, examining parental rearing and family correlates may provide insights into broader contextual factors underlying the poorer response to evidence-based OCD treatments. Based on the aforementioned studies, it could be expected that parents of youth with comorbid OCD and ADHD may exhibit less-positive parental rearing styles, such as higher parental rejection and overprotection.

3. Executive function

Functional imaging studies in adult OCD support theory that the cortico-striato-thalamo-cortical (CSTC) is involved in the pathogenesis and expression of OCD (Saxena and Rauch, 2000), which is proposed to explain the observed associations between OCD and deficits in executive functions (Bannon et al., 2002; Chamberlain et al., 2007; Kuelz et al., 2004). However, findings across the neuropsychological literature in OCD are inconsistent and heterogeneous (Abramovitch et al., 2013; Kuelz et al., 2004), suggesting a range of potential moderating factors on these associations (e.g., Abramovitch et al., 2013). One such factor is comorbid ADHD which may account for more pronounced deficits across a range of indices of executive functions commonly associated with OCD. In contrast to neuropsychological studies with children with OCD, those conducted with children with ADHD are largely consistent and homogenous in observing deficits across a broad range of executive functions (i.e., Frazier et al., 2004). A poorer capacity to regulate behavioural and emotional responses and execute control across domains of meta-cognition (e.g., working memory, capacity to organize and plan) may result in even greater psych-social impairments for youth with comorbid OCD and ADHD.

4. Treatment response

CBT, including exposure with response prevention (ERP), either alone or in combination with SRI medication, is considered the gold-standard treatment for paediatric OCD (O'Kearney, 2007). However, despite best efforts, a large number of children and adolescents do not achieve complete remission following CBT. Comorbidity has consistently been found to be associated with poorer response to OCD treatment (Stewart et al., 2004; Storch et al., 2008), with several studies demonstrating that comorbid externalising disorders in particular are associated with an attenuated response (Farrell et al., 2012; Garcia, 2010; Ginsburg et al., 2008; Storch et al., 2008). For example, Storch and colleagues (2008) investigated the impact of comorbidity on response to CBT for OCD, and found that children with comorbid ADHD had significantly lower treatment response and remission rates compared to youth with other comorbidities. The response rates did not differ between youth with comorbid ADHD who were and were not on ADHD-focused medication (Storch et al., 2008). In a study of treatment response to group-CBT for paediatric OCD, Farrell and colleagues (2012) found that children with comorbid depression or pervasive developmental disorder fared no worse than children without comorbidity, yet children with comorbid ADHD showed significantly poorer treatment response and remission rates at 6-month follow-up. This body of research suggests that comorbid OCD and ADHD is a

particularly challenging presentation to treat and highlights the need to better understand the how these youth might differ in clinical expression of OCD (OCD symptoms, comorbidity), as well as in family functioning (parental rearing, distress and accommodation) and along indices of executive functioning, in order to improve current approaches to treatment.

5. The present study

The aim of the present study was to examine the clinical expression (i.e., symptoms, comorbidity) and correlates (i.e., family variables, executive functioning) of comorbid OCD and ADHD in a sample of treatment-seeking children and adolescents with a primary diagnosis of OCD and comorbid ADHD (*OCD + ADHD group*), compared to an age- and gender-matched sample of youth with OCD and other comorbidity, but without ADHD (*OCD without ADHD*). Based on the literature reviewed, it was hypothesised that (1) relative to the OCD without ADHD, the OCD + ADHD group would be characterised by higher levels of OCD symptom severity, higher endorsement of somatic and hoarding symptoms, and greater OCD-related functional impairment; (2) the OCD + ADHD group would have higher overall comorbidity, as well as higher rates of anxiety and depressive symptoms and tic disorders, relative to the OCD without ADHD group; and (3) relative to the OCD without ADHD group, the OCD + ADHD group would exhibit greater deficits across indices of executive functioning (including behavioural regulation, metacognition, global executive function). It was hypothesised that there would also be group differences across a range of measures of family functioning, whereby (4) the OCD + ADHD group would score higher on family accommodation, parental psychopathology (depression, anxiety and stress symptoms), parental rejection and overprotection, and lower on parental warmth. Finally, in order to replicate previous findings of the attenuated response to CBT for youth with comorbid OCD and ADHD, this study examined patterns of treatment response and remission at post-treatment and at 6-month follow-up. It was predicted that (5) fewer participants in the comorbid OCD + ADHD group would be classified as a treatment responders and/or treatment remitters at post-treatment and 6-month follow-up compared to participants in the OCD without ADHD group.

6. Method

6.1. Participants

The participants for the current study included 40 children and adolescents aged 7 to 17 years (M = 12.18, SD = 2.85) with a primary diagnosis of OCD and comorbid ADHD, as well as an age- and gender-matched sample of 40 children and adolescents with OCD without ADHD (M = 12.13, SD = 2.85). Sixty-nine percent of the overall sample were male. The sample was drawn from consecutive referrals into cognitive-behavioural treatment studies being offered at Griffith University, Queensland, Australia. Across the studies, rates of comorbid OCD + ADHD ranged from 14% to 20% of the complete treatment seeking OCD sample. Participants were recruited into these research trials through community advertisements and self-referral.

For inclusion, participants had a primary diagnosis of OCD (DSM-IV; American Psychiatric Association, 2000), with or without comorbid diagnoses. Exclusion criteria included presence of psychosis, intellectual disability, or receiving concurrent psychotherapy. Participants were not excluded on the basis of medication, provided they were on a stable dose for 12 weeks prior to enrolment and throughout treatment. Overall, 51% of participants were on antidepressant medication for their OCD (59% in the OCD + ADHD group, and 46% in the OCD without ADHD group, no significance difference between groups, $p < .05$). Further, 20% of children in the ADHD group were also taking stimulant medication.

6.2. Measures

The Anxiety Disorders Interview Schedule - Parent Version (ADIS-P). The ADIS-P (Silverman and Albano, 1996) is a semi-structured, clinician-administered interview, designed to assess anxiety, mood, externalising and other childhood disorders including ADHD, based on DSM-IV diagnostic criteria. The ADIS-P has been shown to possess good inter-rater and retest reliability (Holmes et al., 2014; Silverman and Nelles, 1988; Silverman et al., 2001) and good sensitivity to treatment effects in both childhood anxiety (Kendall, 1994; Ollendick et al., 2009; Oar et al., 2015) and childhood OCD (P. Barrett et al., 2004; Farrell et al., 2012). The ADIS-P was administered to the child's parent/s. A Clinician Severity Rating (CSR), ranging from 0 to 8, with a score of 4 indicating a clinically significant diagnosis was assigned to each diagnosis. Independent inter-rater reliability has been excellent for the ADIS-P interviews and CSR ratings made by assessors involved in the trials from which the data is drawn (i.e., primary diagnosis κ = 1.0; secondary diagnosis κ = 0.84 – 1.0; tertiary diagnosis κ = 0.83 – 1.0; see Farrell et al., 2018; Farrell et al., 2013, 2012).

Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS). The CY-BOCS (Scahill et al., 1997) is a clinician-rated, semi-structured interview used for assessing OCD symptomology in children and adolescents. The CY-BOCS rates severity of obsessions and compulsions across five scales: time, interference, distress, resistance, and control over symptoms, and also provides a total score (0 to 40). The CY-BOCS has shown excellent internal consistency (r = 0.87), good to excellent inter-rater reliability (r = 0.66 to 0.91 across subscales), and good convergent validity (Scahill et al., 1997).

Child OCD Impact Scale - Parent Report (COIS-P). The COIS (Piacentini et al., 2001) is a self-report measure completed by parents to assess the impact of OCD on children's psychosocial functioning. This measure includes a total of 20 items assessing functioning across three domains: (1) school, (2) social, (3) family/home. Items are rated on a 4-point Likert-scales. Studies using the child COIS have shown excellent internal consistencies for the three subscales and the total score (range α = 0.78 to 0.85), and good convergent validity between the COIS total score and the CY-BOCS (r = 0.46) (Piacentini et al., 2001). The internal consistency within the current sample was α = 0.95.

Multidimensional Anxiety Scale for Children (MASC). The MASC (March 1997) is a 39-item self-report measure designed to assesses anxiety in children. Research has indicated that the MASC has good internal reliability and convergent validity (March et al., 1997). The internal consistency within the current sample was α = 0.92.

Children's Depression Inventory (CDI). The CDI (Kovacs, 1992) is a 27-item, self-report measure designed to assess symptoms of depression in children. The CDI has demonstrated good internal consistency across various age groups and genders (α = 0.83 to 0.89), stable item-total score product-moment correlations, and test-retest coefficients of 0.74 and 0.77 (Smucker et al., 1986), as well as demonstrated validity (Kovacs & Beck, 1977). The internal consistency within the current sample was α = 0.87.

Behaviour Rating Inventory of Executive Function – Parent (BRIEF-P). The BRIEF-P (Gioia et al., 2000) is an 86-item parent-report measure of impairments in executive function in children and adolescents. Items comprise 3 index scores (and 8 clinical subscales), including Behaviour Regulation (comprised of Inhibit, Shift, and Emotional Control), Metacognition (Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor), and Global Executive Function (total of all 8 subscales). The scale has been shown to have high internal consistency (αs = 0.80 to 0.98) and test-retest reliability (rs = 0.82 for parents; Gioia et al., 2000). In this study, internal consistency was α = 0.94 Behaviour Regulation, α = 0.96 Metacognition, and α = 0.98 Global Executive Function.

Family Accommodation Scale (FAS). The FAS (Calvocoressi et al., 1995) was used to assess the frequency and severity of parental accommodation to OCD. The scale comprises 12 items scored on a 5-point

Table 1
Symptom severity, functional impairment, and symptom clusters across groups.

Clinical Measure	OCD + ADHD*N* = 40M (SD)/*n* (%)	OCD*N* = 40M (SD)/*n* (%)	t/χ^2	*p*	*d*
Age of OCD onset (years)	7.91 (3.08)	8.23 (3.72)	−0.70	.484	
OCD Severity					
CYBOCS Obsessions	12.95 (2.84)	12.30 (3.07)	0.97	.334	
CYBOCS Compulsions	13.15 (2.71)	13.05 (3.07)	0.16	.874	
CYBOCS Total	26.00 (5.10)	25.40 (5.59)	0.50	.617	
Functional Impairment (COIS-P)					
School	16.30 (10.26)	13.46 (9.45)	1.21	.232	
Social	15.20 (13.12)	13.41 (10.47)	1.64	.522	
Home and Family	19.46 (11.79)	14.24 (9.12)	2.11	.039*	0.48
Total Impairment	50.56 (30.79)	41.22 (25.74)	1.39	.169	
CY-BOCS Compulsions					
Washing/Cleaning	31 (78%)	34 (85%)	0.41	.521	
Checking	31 (78%)	32 (80%)	0.00	.955	
Repeating	25 (63%)	26 (65%)	0.00	.934	
Counting	12 (30%)	9 (23%)	0.69	.406	
Arranging/Symmetry	21 (53%)	20 (50%)	0.12	.732	
Hoarding/Saving	16 (40%)	9 (23%)	3.13	.063	
Superstitious Behaviours	12 (30%)	10 (25%)	0.33	.567	
Rituals Involving Others	32 (80%)	31 (78%)	0.25	.615	
Misc. Compulsions	32 (80%)	34 (85%)	0.13	.724	
CY-BOCS Obsessions					
Contamination	36 (90%)	30 (75%)	4.30	.039*	
Aggressive	30 (75%)	24 (60%)	2.61	.106	
Hoarding/Saving	13 (33%)	11 (28%)	0.32	.573	
Health-Related/Somatic	20 (50%)	23 (58%)	0.31	.579	
Religious/Moral	19 (48%)	19 (48%)	0.12	.727	
Magical/Superstitious	11 (28%)	12 (30%)	0.03	.861	
Sexual obsessions	4 (10%)	15 (38%)	6.36	0.012**	
Miscellaneous Obsessions	26 (65%)	28 (70%)	0.10	.750	

Note: *d* = Cohen's d effect size for t-tests. Holm-modified Bonferroni correction applied.
* $p < 0.05$.
** $p < .025$.

scale, with an additional item of parental distress associated with accommodation, and a further three items assessing the consequences of not participating in accommodation. Good internal consistency, as well as good convergent and divergent validity have recently been established in a psychometric evaluation of the measure (Flessner et al., 2011). The internal consistency within the current sample was $\alpha = 0.92$.

Egna Minnen Betraffande Uppfostran - Parent and Child (EMBU- P/C). The EMBU-P/C (Castro et al., 1993; Muris et al., 1996; Muris et al., 2003) is a widely used measure of parental rearing behaviours as perceived by the parent and child, respectively. It is comprised of 4 subscales with 10 items each, measuring four domains of rearing: (1) emotional warmth, (2) overprotection, (3) rejection and (4) anxious rearing. The EMBU-P and EMBU-C have been shown to have good internal consistency for emotional warmth, rejection and control attempts subscales (Castro et al., 1997) and has been recently validated for youth with OCD (Mathieu, Conlon, Waters, & Farrell, under review). The internal consistency within the current sample ranged from $\alpha = 0.66$ to 0.90.

Depression Anxiety and Stress Scale (DASS-21). The DASS-21 (Lovibond and Lovibond, 1995) was used to assess parental psychopathology of anxiety and depression. The measure is a 21 item self-report questionnaire designed to assess the severity of symptoms common to depression, anxiety and stress. Strong psychometric properties have been reported for the DASS-21 (Antony et al., 1998). Research has indicated excellent internal consistency (Cronbach's alpha 0.87 and above; (Antony et al., 1998; Sardá et al., 2008). The internal consistency for this sample was $\alpha = 0.92$.

6.3. Procedure

The current study received ethical approval through the university human research ethics committee. Participants were recruited through advertisements in school newspapers and local newspapers, as well as referrals from community health practitioners. Following referral, parents completed a brief telephone interview to determine eligibility. Diagnostic assessments were conducted by trained postgraduate-level clinicians with parents over the telephone. Final consensus on diagnostic severity and study eligibility was determined with the supervising clinical psychologist (LJF). Families were subsequently offered either group-based CBT treatment or intensive CBT delivered individually (depending on the trial for which they were recruited). The treatment protocols were based on empirically supported CBT for OCD (see March and Mulle, 1998) and included psychoeducation, cognitive restructuring, exposure with response prevention (ERP), and relapse prevention. There were no significant differences in the number of children across the comparison groups who received either individual (OCD + ADHD comorbid group $n = 8$; OCD without ADHD $n = 10$) or group CBT (OCD + ADHD group $n = 32$; OCD only $n = 30$), χ^2 (1, $N = 80$) = 0.36, $p = .55$. Previous randomised controlled trials demonstrated similar efficacy for CBT when delivered in either individual or group modality (Barrett et al., 2004).

6.4. Data analysis

Differences between groups on clinical characteristics of OCD were analysed using a series of *t*-tests and chi square tests. The baseline characteristics compared across groups included OCD symptom severity, CY-BOCS symptoms clusters, parent-reported functional impairment, number of comorbid diagnoses, anxiety symptoms, depressive symptoms, family accommodation, parental psychopathology (depression, anxiety, and stress), and parental rearing styles (warmth, rejection, over-protection, and anxious rearing). Differences in treatment response between groups were examined using t-tests and chi square tests, by comparing post-treatment response and remission status at post-treatment and 6-month follow-up. Treatment responders

were classified by a reduction of 35% or greater on CY-BOCS severity, and treatment remitters were classified based on a reduction of 55% or greater, with a CY-BOCS of 11 or less (Skarphedinsson et al., 2017). The Holm–modified Bonferroni method was for multiple comparisons, which controls the family-wise error rate (the probability that one or more Type I errors will occur) by adjusting the rejection criteria of each of the individual hypotheses, making alpha adjustments to families of variables. Alpha criterion for each significant finding is reported as a note within each table. Cohen's *d* is reported as an estimate of effect size.

6.5. Clinical characteristics

OCD symptom expression. Few group (OCD + ADHD vs. OCD without ADHD) differences were observed on OCD symptom expression (see Table 1). There were no significant difference between groups on average CYBOCS symptom severity, OCD-related school impairment, or OCD-related social impairment. Of four impairment areas, one group difference was found, with parents of youth with comorbid OCD + ADHD reporting significantly greater home/family impairment on average, than parents in the OCD without ADHD group. Across OCD symptom clusters, one difference was found; youth in the OCD + ADHD group were significantly *less* likely to endorse sexual obsessions but more likely to endorse contamination obsessions, than youth in OCD group.

Comorbidity. Group comparisons of comorbid diagnoses and symptoms of depression and anxiety are detailed in Table 2. The OCD + ADHD group had a significantly higher average number of comorbidities than children in the OCD group, after excluding OCD and ADHD from the analysis. Follow-up χ^2 tests showed that the groups did not differ significantly in terms of the proportion with comorbid anxiety disorder of mood disorder diagnoses. However, a higher proportion of children in the OCD + ADHD group experienced tics, as well as separation anxiety disorder (SAD), autism spectrum disorder (ASD) and/or oppositional defiance disorder (ODD) diagnoses. There were no significant group differences on symptoms of depression or anxiety, as measured by the CDI and MASC.

Executive function. As shown in Table 3, independent groups t-tests found that, on average, youth in the OCD + ADHD group, relative to the OCD group without ADHD, were rated significantly higher on executive function deficits across all three subscale of the BRIEF-P (behaviour regulation index, metacognition index, and global executive function).

Family functioning. No significant group differences were found on measures of parental psychopathology (see Table 4). However, significant differences were found between groups on family accommodation, whereby parents in the OCD + ADHD group reported significantly more family accommodation than parents in the OCD without ADHD group. Parents of children with OCD + ADHD also reported significantly higher levels of anxious rearing and rejection than parents of children in the OCD without ADHD group.

Table 2
T-tests and χ^2-tests comparing OCD + ADHD and OCD-without ADHD on comorbidity.

	ADHDM (SD) /*n* (%)	OCDM (SD) /*n* (%)	t/χ^2	*p*
No. Comorbid Diagnoses (excl. OCD/ADHD)	2.18 (1.60)	1.55 (0.98)	2.10	.039*
Comorbid Diagnoses				
Separation Anxiety Disorder	10 (25%)	3 (8%)	4.50	.034*
Social Phobia	12 (30%)	14 (35%)	0.23	.633
Specific Phobia	14 (35%)	10 (25%)	0.95	.329
Panic Disorder	0 (0%)	0 (0%)	–	–
Generalised Anxiety Disorder	18 (45%)	23 (58%)	1.25	.263
Dysthymic Disorder	0 (0%)	1 (3%)	1.01	.314
Major Depressive Disorder	3 (8%)	4 (10%)	0.16	.692
Presence of Motor or Vocal Tics	12 (30%)	4 (10%)	5.00	.025**
Tourette's Syndrome	3 (8%)	0 (0%)	3.12	.077
Autism Spectrum Disorder	10 (25%)	2 (5%)	6.26	.012***
Oppositional Defiance Disorder	7 (18%)	0 (0%)	7.67	.006****
Depression	59.29 (14.12)	57.14 (13.65)	0.65	.517
Anxiety	61.06 (13.13)	59.05 (13.16)	0.65	.518

Holm-modified Bonferroni correction applied.
* $p < .05$.
** $p \leq 0.025$,.
*** $p < 0.016$,.
**** $p < 0.012$.

6.6. Treatment response

Seventy children and their parents were assessed directly at post-treatment (OCD + ADHD group $n = 30$) and $n = 63$ children and their parents were assessed at 6-month follow-up (OCD + ADHD group $n = 31$). When the proportion of children were compared using χ^2, youth with OCD + ADHD were significantly less likely than youth with OCD without ADHD to be classified as treatment responders and remitters at post-treatment. Further, there was trend for fewer youth with OCD + ADHD to be deemed responders at 6 months follow-up. (see Table 5).

6.7. Effect of comorbidity on family variables and treatment response

As a post-hoc analysis to investigate the unique contribution of ADHD on the identified clinical correlates (family accommodation, executive function, parental rejection, anxious rearing, and home/family impairment) and treatment outcome (at 6-months follow-up), hierarchical regressions were conducted controlling for age, gender, ODD, ASD, and tics at Step 1 of the model. ADHD was entered at Step 2 of the model (see Tables 7 and 8). Results suggest that ADHD uniquely contributed to family accommodation, parental rejection, anxious rearing, global executive function, and percent reduction in OCD symptoms at 6-months follow-up. ADHD was not found to uniquely contribute to home/family impairment after accounting for age, gender, ODD, ASD, and tics (see Table 6)

7. Discussion

In the current study, we investigated the unique clinical expression and CBT treatment response of youth with comorbid OCD and ADHD relative to children with a diagnosis of OCD but not ADHD. Results of the current study suggest that the comorbid OCD and ADHD is relatively common, affecting between 14 and 20% of treatment seeking youth in the current study. Youth with both disorders may represent a specific subtype of OCD, characterised by high comorbidity and significant OCD-related impairments related to home and family life. In line with study hypotheses, parents of comorbid youth reported significantly greater home/family impairment, greater family accommodation of OCD symptoms, and more maladaptive parental rearing styles, including significantly greater parental rejection and anxious rearing. These findings highlight the strain on the families of children experiencing both OCD and ADHD, relative to families with a child with OCD but not ADHD. Given the difficulty that parents may have in determining what behaviours / impairments are associated with OCD versus ADHD, it is possible that the co-occurrence of ADHD symptoms inflates parents perceptions of OCD-related impairment and parental accommodation. Nevertheless, it is clear that comorbid youth present with greater difficulties in clinical expression, as well as impact on parents. Consistent with hypotheses, children with comorbid OCD and ADHD were also characterised as being more highly comorbid, with particularly high rates of separation anxiety, tics, autistic spectrum disorder (ASD) and oppositional defiant disorder (ODD). The results

Table 3
T-tests comparing OCD + ADHD and OCD without ADHD on executive function T-scores.

	ocd + adhd *m (sd)*	ocd without adhd *m (sd)*	*t*	*p*	*d*
Behaviour regulation index deficits	69.00 (12.19)	55.96 (10.91)	3.99	< 0.001	1.13
Metacognition deficits	67.58 (9.76)	55.00 (12.24)	3.99	< 0.001	1.14
Global executive function deficits	69.63 (10.87)	55.15 (11.56)	4.55	< 0.001	1.11

Holm-modified Bonferroni correction applied. All *p* were less than 0.001.
Note. For all three subscales, a higher score indicated more deficits.

Table 4
T-tests comparing OCD + ADHD and OCD without ADHD on family accommodation, parental psychopathology, and parental rearing styles.

	OCD + ADHD*M (SD)*	OCD without ADHD*M (SD)*	*t*	*P*	*d*
Family accommodation					
Total Accommodation	26.27 (11.92)	17.48 (11.33)	3.15	.002**	0.76
Parental psychopathology					
Depression	2.78 (3.33)	2.31 (2.58)	0.67	.507	
Anxiety	1.70 (2.88)	1.77 (2.41)	−0.11	.913	
Stress	6.65 (4.72)	6.63 (4.72)	0.02	.986	
Parental rearing (EMBU-P)					
Emotional warmth	33.47 (4.72)	33.74 (3.83)	−0.29	.774	
Overprotection	26.84 (3.68)	24.85 (4.18)	1.59	.118	
Rejection	18.04 (4.07)	15.42 (3.74)	2.77	.007**	0.67
Anxious rearing	25.73 (4.34)	21.42 (6.04)	2.30	.025*	0.82

Holm-modified Bonferroni correction applied.
* $p < .05$.
** $p < .01$.

Table 5
Treatment Response at Post-Treatment and Six-Month Follow-Up Across OCD + ADHD and OCD Groups.

Treatment response	OCD + ADHD*n* (%)	OCD without ADHD*n* (%)	χ^2	*p*
Responder status (Post)	17 (57%)	27 (82%)	4.72	.028*
Responder status (6-months)	16 (59%)	27 (79%)	2.94	.076
Remission status (Post)	9 (30%)	21 (63%)	7.13	.007**
Remission status (6-months)	10 (36%)	16 (47%)	0.81	.261

* $p < .05$.
** $p < .025$.

reported here are also in line with study hypotheses and past research highlighting an attenuated response to CBT for youth comorbid OCD and ADHD. Immediately following treatment, youth with comorbid OCD and ADHD were significantly less likely to be responders or re-mitters from their OCD.

The finding that children with comorbid OCD and ADHD were more likely to have higher rates of tic disorders and ASD, is in line with prior research highlighting a higher than expected rate of co-occurrence, suggesting shared neurobiological and genetic mechanisms (e.g., Pauls, 2008). Indeed, on the basis of the high co-occurrence, some researchers propose that this cluster of disorders may be best viewed as a spectrum of disorders with over-lapping symptoms and shared aetiologies, converging in dysfunctional cortico-striatal circuitry (Huisman-van Dijk et al., 2016;). These comorbidity findings align with a number of other studies among samples of children with OCD (Hanna, 1995; Ivarsson et al., 2008) who have reported similar clustering of OCD,

Table 6
Hierarchical multiple regression predicting Family Accommodation, Parental Rearing Styles, and Family Impairment.

	Family Accommodation			Parental Rejection			Anxious Rearing			Home and Family Impairment		
	B	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*
Step 1	$R^2 = 0.08$, $F(5,64) = 1.08$, $p = .380$			$R^2 = 0.07$, $F(5,63) = 0.89$, $p = .492$			$R^2 = 0.08$, $F(5,63) = 1.02$, $p = .414$			$R^2 = 0.10$, $F(5,66) = 1.45$, $p = .218$		
Age	−0.04	.54	−0.01	−0.11	0.18	−0.07	−0.30	0.25	−0.142	.34	.44	.09
Gender	−3.11	3.49	−0.12	1.29	1.15	.16	1.16	1.64	.096	−3.79	2.90	−0.17
Tics	−2.67	3.88	−0.09	0.05	1.24	.01	0.42	1.77	.030	4.88	3.46	.17
ODD	12.20	5.69	.28*	0.89	1.75	.07	−0.03	2.49	−0.002	2.58	4.58	.07
ASD	3.13	3.99	.10	2.04	1.34	.19	3.73	1.91	.241	3.81	3.81	.12
Step 2	$\Delta R^2 = 0.09$, $F_{chg}(1,63) = 6.63$, $p = .012$			$\Delta R^2 = 0.09$, $F_{chg}(1,62) = 6.68$, $p = .012$			$\Delta R^2 = 0.06$, $F_{chg}(1,62) = 4.19$, $p = .045$			$\Delta R^2 = 0.04$, $F_{chg}(1,65) = 3.07$, $p = .085$		
Age	0.05	.52	.01	−0.10	0.17	−0.07	−0.29	.247	−0.138	.32	.44	.09
Gender	−2.17	3.36	−0.08	1.56	1.11	.19	1.46	1.60	.121	−3.39	2.87	−0.15
Tics	−4.33	3.78	−0.14	−0.55	1.21	−0.06	−0.26	1.76	−0.02	3.64	3.48	.13
ODD	7.66	5.74	.17	−0.36	1.74	−0.03	−1.46	2.53	−0.08	.52	4.66	.02
ASD	0.53	3.97	.02	1.03	1.34	.10	2.57	1.95	.17	2.33	3.85	.07
ADHD	8.20	3.18	.33*	2.68	1.04	.34*	3.08	1.50	.27*	4.72	2.69	.22
Model R^2	$R^2 = 0.17$, $F(6,63) = 2.08$, $p = .068$			$R^2 = 0.16$, $F(6,62) = 1.92$, $p = .091$			$R^2 = 0.13$, $F(6,62) = 1.59$, $p = .165$			$R^2 = 0.14$, $F(6,65) = 1.76$, $p = .122$.		

Table 7
Hierarchical multiple regression predicting Global Executive Function and Percent Reduction in CYBOCS severity at 6-Months.

	Global Executive Function			CYBOCS% Reduction 6-Months		
	B	*SE B*	*β*	*B*	*SE B*	*β*
Step 1	$R^2 = 0.13$, $F(5,44) = 1.40$, $p = .242$	$R^2 = 0.26$, $F(5,43) = 3.08$, $p = .018$				
Age	−0.43	0.70	−0.09	0.62	1.49	.06
Gender	−2.18	4.10	−0.08	24.09	12.33	.30
Tics	4.35	7.07	.09	−20.05	10.83	−0.25
ODD	14.59	6.14	.36*	−25.67	17.54	−0.21
ASD	0.12	6.25	.00	−21.36	11.66	−0.25
Step 2	$\Delta R^2 = 0.23$, $F_{chg}(1,43) = 15.96$, $p = .000$			$\Delta R^2 = 0.08$, $F_{chg}(1,42) = 4.76$, $p = .035$		
Age	−0.49	.60	−0.10	0.48	1.43	.05
Gender	−0.35	3.57	−0.01	24.53	11.82	.30
Tics	−3.08	6.38	−0.06	−13.45	10.82	−0.17
ODD	7.93	5.56	.20	−20.02	17.02	−0.16
ASD	−3.29	5.46	−0.08	−13.90	11.69	−0.16
ADHD	14.26	3.57	.54***	−20.68	9.49	−0.31*
Model R^2	$R^2 = 0.37$, $F(6,43) = 4.23$, $p = .002$			$R^2 = 0.34$, $F(6,42) = 4.75$, $p = .006$		

* $p < .05$, ** $p < .01$,.
*** $p < .001$.

ASD, tics and ADHD. Despite this high rate of comorbid clustering, limited research has been conducted to date exploring the symptom level chrematistics of these youth.

In regards to the family, the findings of the current study highlight the importance of paying close attention to family processes when assessing and treating children with comorbid OCD and ADHD. Certainly, even without the presence of ADHD, family factors, including family accommodation and parental rearing, have been previously found to be associated with greater OCD severity and poorer response to CBT (e.g., Lavell et al., 2016; Peris et al., 2012). Greater family accommodation is also well established as a predictor of greater OCD severity and impairment among children, as well as a robust predictor of poorer treatment response (Lebowitz et al., 2012; Wu et al., 2016). Given the findings that comorbid ADHD appears associated with even higher family accommodation and greater parental rejection and anxious rearing, it is not surprising then that these youth report significantly more impairment at home and in family life, and have greater difficulty achieving an immediate treatment response, relative to youth without ADHD. Of note, this study did not find evidence that comorbid OCD and ADHD was associated with significantly greater OCD severity; suggesting that the associations found here are not simply a function of greater severity. Further investigation into family stress and burden, family accommodation, and parental rearing among youth with comorbid ADHD and OCD may explain the unique experiences for these children and their families, and inform components to address in treatment. For example, the current findings suggest that families of comorbid youth may benefit from additional family-focussed treatment modules which focus on targeting family accommodation of OCD and reducing potentially detrimental parenting practices, such as anxious rearing and/or harsh and rejecting rearing.

Children with comorbid OCD and ADHD experienced significantly greater deficits across major indices of executive functions relative to youth without ADHD, including elevated scores within clinical range (*T scores* >65) on the behavioural regulation index (i.e., one's ability to problem-solve or cognitively shift freely from one situation to another, and regulate his or her emotions), the metacognitive index (i.e., ones ability to plan, organise, self-monitor and sustain working memory), and on the overall global executive function index. Children without ADHD however, did not report overall deficits outside of normal range across the executive functioning indices measured by the BRIEF. These findings provide support for the role of comorbidity in potentially accounting for the largely inconsistent findings in neuropsychological studies among OCD samples to date (Abramovitch et al., 2013; Kuelz et al., 2004). The observed deficits in executive functions for youth with comorbid OCD and ADHD may account for greater overall impairment, and in part, may also be associated with poorer response to treatment. If these youth have significantly greater difficulty regulating emotions, as well as planning, organising and shifting attention, then they may have greater difficulty acquiring the skills to manage OCD symptoms during treatment. These deficits in executive functions (i.e., emotional regulation) may also underlie the elevated levels of family accommodation observed among comorbid youth with ADHD, whereby dysregulation and distress may drive coercive-disruptive behaviours (e.g., Lebowitz et al., 2015; Schuberth et al., 2018) and accommodation demands. Research that aims to examine mediating effects of executive function during CBT are needed to empirical address this possibility, as well as studies which aim to examine whether these deficits are corrected in response to CBT.

In line with prior research reporting an attenuated response to treatment among children with externalising symptoms and ADHD, youth in the current study with comorbid OCD and ADHD faired significantly worse at post-treatment than youth without ADHD (Farrell et al., 2012; Ginsburg et al., 2008; Storch et al., 2008). Indeed, youth with ADHD were less likely to be classified as treatment responders of remitters at post-treatment compared to youth without ADHD. While results suggest they may be just as likely to remit from CBT in the longer term (at 6-months follow-up), there also appeared a trend for fewer children with comorbid OCD and ADHD to respond at longer term follow-up relative to youth without ADHD. The mechanisms for poorer response associated with comorbid ADHD remain currently unknown. Certainly there are likely to be multiple factors that moderate longer term outcomes for these youth, including variables investigated here, such as family functioning, impact of other comorbidity, or the result of the significant neurocognitive deficits observed in this sample, all of which may play some role in this attenuated response. To improve response and remission rates, it is possible that these comorbid children may require a stronger dose of CBT, such as the provision of intensive sessions of longer duration (Farrell et al., 2016), more frequent sessions (bi-weekly instead of weekly), or booster sessions following the initial course of CBT in order to bolster their response to CBT. Future research examining mediators of change and moderators of response for youth with comorbid ADHD are needed in order to further explore this finding.

This study involved a carefully selected sample of youth, screened for the presence of both primary OCD and comorbid ADHD using gold standard, psychometrically robust, standardised assessments. A thorough and comprehensive assessment of clinical correlates was conducted in order to advance understanding on the clinical symptom expression of comorbid youth, relative to a more homogenous sample of youth with primary OCD. Limitations to the current study include the

cross-sectional design of the study, relatively small clinical sample resulting in compromised power, and lack of an experimental control group of youth with ADHD without OCD. Furthermore, the high rates of comorbidity (i.e., tics, ASD, ODD) observed in the comorbid OCD and ADHD group confounds the observed differences noted across groups. Studies with larger samples, additional control groups (e.g., ADHD alone, OCD and ADHD without ASD) would elucidate more precise information on the clinical correlates associated with ADHD, but not other frequently co-occurring conditions, such as tic disorders, or ASD.

The current study found a number of important differences in the clinical presentation and response to treatment among youth with comorbid ADHD and OCD. These findings highlight that youth with comorbid OCD and ADHD may have greater disruptions and impairments at home, characterised by greater family accommodation to symptoms, and may be more likely to experience more aversive / maladaptive parental rearing. These youth also appear to have significant deficits in their executive functions which may impede a good treatment response. Treatment for children with comorbid OCD and ADHD should involve the assessment of family functioning (including accommodation to symptoms and rearing) and ensure the inclusion of parents in therapy. Moreover, these youth may require a stronger dose of therapy (e.g., more sessions, longer sessions, or booster sessions) in order to improve treatment response following initial CBT. Given the heterogeneity in OCD expression in children and youth, and the far from optimal treatment response rates reported in the literature, greater understanding of OCD sub-types, response to current treatments and unique clinical correlates may help inform the development of more comprehensive developmental aetiological models, and drive the development of more personalised approaches to treatment, resulting in better long term outcomes for all youth. Author Statement

Funding

The current research was supported by funding from the Foundation for Children, Financial Markets grant scheme, as well as a National Health and Medical Research Council (NHMRC) project grant (Farrell APP1058025).

Author contributions

LJF designed the study, oversaw data collection, analysis and revising the paper. Author CL and EB assisted with data collection, literature searchers, data analysis and drafting / editing the first drafts of the manuscript. Authors AW and MZG contributed to the final drafting and revising the manuscript and contributed to the methodology and deigns. All authors contributed to and have approved the final manuscript. All authors warrant that the article is the authors' original work, hasn't received prior publication and isn't under consideration for publication elsewhere.

There are no further acknowledgements in relation to this research.

Declaration of Competing Interest

None.

Acknowledgements

The current research was supported by funding from the Foundation for Children, Financial Markets grant scheme, as well as a National Health and Medical Research Council (NHMRC) project grant (Farrell APP1058025).

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jad.2020.01.144.

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