Child Psychiatry Hum Dev (2017) 48:911–921
DOI 10.1007/s10578-017-0714-3

ORIGINAL ARTICLE

ASD Traits Among Youth with Obsessive–Compulsive Disorder

Donna L. Griffiths[1] · Lara J. Farrell[1] · Allison M. Waters[1] · Susan W. White[2]

Published online: 24 February 2017

Abstract Research has shown high rates of comorbid psychiatric disorders among samples of youth with obsessive–compulsive disorder (OCD) (Farrell et al., Psychiatry Res 199(2):115–123, 2012; Lewin et al., Psychiatry Res 178(2):317–322, 2010; POTS Team, J Am Med Assoc 292(16):1969–1976, 2004). Autism and autistic traits co-occur at high rates within clinical samples of youth with OCD (Ivarsson and Melin in J Anxiety Disord 22(6):969–978, 2008; Stewart et al. in Child Psychiatry Hum Dev 1–9, 2016). This study extends the literature by examining the relationship between ASD traits, family accommodation, and functional impairment in a sample of youth with OCD across a wide age range ($n=80$; aged 7–17 years). Results indicated that autistic traits, as measured by the social responsiveness scale (SRS), were elevated in 32.5% of youth (based on a T-score of 66T and above) relative to typically developing youth, as well as youth with non-autism-related psychiatric disorders (Constantino and Gruber in Social responsiveness scale, Western Psychogical Services, Torrance, 2012). Furthermore, 27.5% of youth scored within a moderate range (66T–75T) and 5% of youth scored within a severe range (76T or higher) on the SRS, typical of children with ASD (Constantino and Gruber in Social responsiveness scale, Western Psychogical Services, Torrance, 2012). Additionally, ASD traits were associated with greater functional impairment above OCD severity. Furthermore, family accommodation mediated the relationship between ASD traits and functional impairment. Implications of these findings are discussed in the context of clinical assessment and direction for further research.

Keywords OCD · Autism · Autistic traits · Functional impairment

✉ Lara J. Farrell
l.farrell@griffith.edu.au

Donna L. Griffiths
donna.griffiths@griffithuni.edu.au

[1] School of Applied Psychology, Behaviour Basis of Health and Menzies Health Institute, Griffith University, Gold Coast Campus, Southport, QLD 4222, Australia

[2] Department of Psychology, Virginia Polytechnic Institute and State University, 109 Williams Hall (0436), Blacksburg, VA 24061, USA

Introduction

Whilst the occurrence of high rates of comorbid psychiatric disorders such as anxiety and mood disorders in paediatric OCD has been well established (e.g., [1–3]), there is also emerging evidence for elevated rates of autism spectrum disorder (ASD) diagnosis and traits of ASD in paediatric OCD. For example, in a sample of children and adolescents ($n=109$) who had a primary diagnosis of OCD, Ivarsson and Melin [4] found that 8.26% of their sample met ASD diagnosis. Additionally, ASD traits were associated with a greater frequency of other comorbid disorders, such as tic disorders and attention deficit hyperactivity disorder (ADHD). In another study Stewart and colleagues [5] found elevated ASD traits in 36.2% of their clinical sample of young children ($n=127$; age 5–8 years) who had a primary diagnosis of OCD, as measured by the social responsiveness scale (SRS) [6]. In the adult literature, Anholt et al. [7] investigated the interrelations between OCD, ADHD, and autism symptoms in adult patients ($n=109$) with a primary diagnosis of OCD, and found that OCD patients presented with higher scores on standardised measures of ADHD and autism symptoms when compared to healthy controls ($n=87$). It is unclear whether elevated ASD traits

within OCD samples may be representative of an overlap in the clinical expression of each disorder, and/or point to shared underlying mechanisms or risk factors across these disorders. Thus, further research aimed at examining various subtypes of children with OCD (defined by comorbid symptoms and disorders) is important in order to develop more refined, conceptual models and approaches to treatment.

Clinical research has provided clear evidence that ASD co-occurs with OCD in both adult [8, 9] and paediatric samples [4]. However, given that the types of psychiatric comorbidity appears to differ across the developmental trajectory for patients with OCD [10], it is currently unclear whether ASD traits among OCD patients are generally elevated across development, or whether they are concentrated in younger samples. The prevalence of OCD among individuals with ASD has also been shown to be elevated when compared to typically developing peers; however, the precise rates of co-occurrence are unclear given that studies report estimates ranging from 1.5 to 81% (see [11]). In a systematic review across seven studies of participants with ASD, Neil and Sturmey [12] estimated the median prevalence of comorbid OCD to be 10% (range 1.47–37.2%). In a community sample ($n=109$), Leyfer et al. [11] found that 37% of children with autism also met DSM-IV [13] criteria for OCD. In two other previous studies based on DSM-IV-TR [14] criteria, the most common comorbid anxiety disorders found in youth with ASD were OCD and specific phobias (e.g., [15, 16]). Conversely, research suggests that 3–7% of children with OCD also meet diagnostic criteria for ASD, and the disorder is found to occur 6–14 times more often among OCD samples compared to the general population [17]. Recently, Griffiths et al. [18] reported rates of comorbid OCD and ASD at 21% within a clinical sample of ($n=117$) youth presenting for treatment of OCD. Therefore, while estimates of prevalence vary considerably across studies, there is evidence that these disorders co-occur at relatively high rates diagnostically and dimensionally in terms of trait severity.

Research to date has examined several child- and family-based correlates of OCD and ASD in youth, both with and without this particular comorbidity. For example, both OCD and ASD are frequently characterised by excessive repetitive behaviours, which may be accounted for by underlying deficits in neurological processes associated with response inhibition [19]. Furthermore, the presence of ASD traits in youth with OCD may be associated with a more severe OCD presentation and greater functional impairment. For example, Stewart and colleagues [5] found that autistic traits in young children (i.e., 5–8 years) as measured by the SRS were predictive of higher levels of OCD severity, as measured by the Children's Yale-Brown obsessive–compulsive scale (CY-BOCS) [20]. Similarly, Griffiths et al. [18] found that when compared to an age (7–17 years) and gender matched group of youth ($n=25$) with OCD without ASD, youth with comorbid ASD ($n=25$) displayed significantly greater functional impairment, had more comorbid disorders, and higher family accommodation. Additionally, they were more likely to have comorbid ADHD, and experience greater depressive symptoms and interpersonal difficulties.

Given that ASD as a diagnosis and dimensionally (subclinical traits) has been associated with a poorer treatment response among youth with OCD [e.g., 18], further investigation in understanding this comorbidity is warranted. Notably, family accommodation (i.e., parental involvement in OCD symptoms and / or modification of family routines as a result of OCD symptoms) has been shown to mediate the relationship between symptom severity and functional impairment in children with OCD (e.g., [21–23]), and is also a robust predictor of attenuated treatment response in clinical trials of youth and adults with OCD [24, 25]. In terms of ASD, Mazefsky et al. [26] suggests that parental involvement is also elevated (i.e., parents tolerating the rigidity associated with ASD as a strategy to avoid emotional outbursts in their child), and may serve to perpetuate poor emotional regulation, increase risk for other psychopathology (such as anxiety, [27]) and as such may be an important process to target in treatment [27]. Considering that both impairment and family accommodation have been found to be elevated among youth with comorbid OCD and ASD [18], and moreover, that studies have suggested parental involvement might maintain emotional dysregulation among youth with ASD [26], an important area of enquiry is the role which family accommodation plays in exacerbating OCD related severity and impairment among youth with comorbid ASD traits. Indeed, one possibility which has not been examined to date is whether family accommodation mediates the association between ASD traits and OCD severity and impairment.

Aim

The aim of the study is to extend the findings of Stewart et al. [5] and Ivarsson and Melin [4] by examining ASD traits in a sample of youth with a primary diagnosis of OCD across a wide age range ($n=80$; aged 7–17 years) to (1) determine whether ASD traits as indicated by the SRS are elevated among both children (i.e., aged 7–12 years) and adolescents (aged 13–17 years) with OCD relative to the standardised, normative data of SRS-2 T-scores [6], and (2) determine if ASD traits are associated with OCD symptom severity and functional impairment. Furthermore, (3) we examined the potential mediating effects of family accommodation in the relationship between ASD traits and

OCD severity and impairment. The following hypotheses were tested: (1) ASD traits will be elevated among youth who have a primary diagnosis of OCD, relative to normative published data [6]; (2) Elevated ASD traits will be associated with increased OCD severity and impairment; (3) Family accommodation will mediate the relationship between ASD traits and OCD severity and functional impairment.

Method

Participants

Eighty participants aged 7–17years ($M = 12.30$, $SD = 2.68$) enrolled in clinical trials for OCD, conducted at Griffith University, from 2013 to 2016 were included in this study. Participants were recruited through community advertisements and self-referred into the programs. Youth were selected into the studies on the basis of a primary diagnosis of OCD, as per the fourth edition of the diagnostic and statistical manual of mental disorders (DSM-IV) [13], had at least one parent willing to attend all sessions, and if taking SRI medication prior to enrolment into the studies, were stabilised on the medication for 3 months, and the medication was not altered during the trial period. Four youth also reported a current diagnosis of ASD Level 1 in accordance to the DSM-5 [28], which had been made by the child's treating paediatrician. Exclusion criteria included psychosis, current suicidal ideation, intellectual disability, mental retardation, or currently receiving psychotherapy.

Measures

Demographics

Demographic data of age, gender, combined family income, medical and psychiatric history was collected by administering the Conners-March Developmental Questionnaire (CMDQ) [29].

The Anxiety Disorders Interview Schedule for Children Parent Version (ADIS-IVC/P). The ADIS-IV C/P [30] versions comprise two semi-structured, clinician-administered interviews, designed to assess anxiety and other childhood disorders, based on DSM-IV diagnostic criteria. They have been shown to have good interrater and retest reliability [31, 32], and have also demonstrated good sensitivity to treatment effects in both childhood anxiety [33–35] and paediatric OCD research [34, 36–38]. Diagnoses are based on symptom endorsement, as well as obtaining a distress/impairment severity rating (scale from 0 = not at all, to 8 = very, very much) from the child or parent being interviewed.

The ADIS-IV-P was administered to parents to ascertain a diagnosis of OCD and to verify secondary and tertiary comorbid diagnoses such as other anxiety disorders, mood disorders, externalising disorders and to screen for PDD. The individual diagnoses were assigned a clinical severity rating (CSR) based on clinician judgment and scored from 0 to 8, with a score of four indicating a clinically significant diagnosis. Independent inter-rater reliability across diagnostic levels of ADIS-P interviews and CSR ratings by our trained assessors have been previously established as excellent across individual published trials including the current sample (e.g., primary diagnosis $k = 1.0$; secondary diagnosis $k = 0.84$; tertiary diagnosis $k = 0.83$ [1]; and primary diagnosis $k = 1.0$; secondary diagnosis $k = 1.0$; tertiary diagnosis $k = 1.0$; [39]).

Children's Yale-Brown Obsessive–Compulsive Scale (CY-BOCS) The CY-BOCS [20] is an extensively used, clinician-rated, semi-structured interview, and was used to assesses symptom severity of obsessions and compulsions across five scales, and provides a total severity rating score. The five scales include: (a) time occupied by symptoms, (b) interference, (c) distress, (d) resistance, and (e) degree of control over symptoms. Ratings are based on 5-point Likert scales (0 = no symptoms, 4 = extreme symptoms), with total scores ranging from 0 to 40. Separate subtotals are calculated for severity of obsessions and compulsions. The clinical cut-off total scores are: (a) mild (8–15); (b) moderate (16–23); (c) severe (24–31); and extreme (32–40). The CY-BOCS includes another six items that assess insight, avoidance, indecisiveness, pathological responsibility, pathological slowness, and pathological doubting. The CY-BOCS has demonstrated excellent internal consistency ($r = .87$), good to excellent interrater reliability ($r = .66$ to .91 across subscales), and good convergent validity [20]. For the current study the CY-BOCS demonstrated good internal consistency ($r = .75$).

Child OCD Impact Scale: Parent Report (COIS-P) The COIS-P [40] was used to assess the impact of OCD on psychosocial functioning from parent report. This measure assesses three domains of impairment (school, social, and family/home) using 20 items. Participants rate items on a 4-point Likert-scale. The parent separately responds to the prompt, *"In the past month, how much trouble have you [your child] had doing the following because of OCD?"* (e.g., writing in class, making new friends, getting dressed in the morning). Four additional items assess the global impact of OCD on school/work, home, social situations, and going out. Studies using the COIS have shown good internal consistencies for the three subscales and the total score (range $r = .78$ to .85), and good convergent validity between the COIS total score and the CY-BOCS ($r = .46$) [40]. For

the current study, internal consistency for the COIS-P total was $r = .95$, and for the subscales of school ($r = .91$), social ($r = .92$), and home ($r = .81$), demonstrating good to excellent internal consistency.

The Social Responsiveness Scale-Second Edition (SRS-2) The SRS-2 [6] is a 65-item parent report designed to measure deficits in social behaviour associated with ASD in children ages 4–18 years. The SRS-2 includes the original SRS for children (4–18 years) and has been extended for use on pre-school children (2.5–4.5 years) and adults from 19 years and above. In addition to a total score, the SRS-2 comprises five subscales: (1) Social awareness, (2) Social cognition, (3) Social communication, (4) Social motivation, and (5) Restricted interests and repetitive behaviour (RRB). Items are scored on a 4-point Likert-type scale, ranging from *not true* = 1, *sometimes true* = 2, *often true* = 3, to *almost always true* = 4. Sample items include: *"Expressions on his or her face don't match what she or she is saying", "Avoids people who want to be emotionally close to him/her", "Has an unusually narrow range of interests"*. SRS-2 has been used to gauge ASD traits dimensionally in both non-clinical and clinical, non-ASD samples [6]. In a recent test review, Bruni [41] reported that the SRS-2 [6] has strong internal consistency ($r = .94$ to $.96$), test–retest reliability based on the original SRS were found to be good ($r = .88$ to $.95$), interrater reliability ($r = .77$ for school age children), good predictive validity showing a sensitivity value of 0.92 and concurrent validity demonstrated high correlations between other rating scales of social behaviour and communications [41], for example, the social communication questionnaire [42] and the children's communication checklist [43]. Internal consistency for the total SRS-2 as measured by Cronbach's alpha was $r = .90$ for the current sample.

Family Accommodation Scale (FAS; [44]) The FAS was used to assesses the frequency and severity of parental accommodation to OCD on a 5-point scale (0 = *never/no accommodation* to 4 = *daily/extreme accommodation*). Total scores are created by summing eight of the 12 items. There is an additional item that rates parental distress associated with accommodation, and a further three items which assess the consequences of not participating in accommodation to their child's OCD behaviours. The psychometric properties have recently been demonstrated, showing good internal consistency, as well as good convergent and divergent validity [45]. The internal consistency for the FAS in the current study was excellent ($r = .90$).

Procedure

All procedures and protocols used in this study received institution ethics review board approval through the university human research ethics committee. Following referral into the various studies, participants were screened via a brief parent interview assessing for obsessive–compulsive symptomatology. If meeting inclusion eligibility, families attended an assessment at the university psychology clinic, conducted by postgraduate clinically trained psychologists. On attending this interview, the research aims were explained to all participants and written informed consent was gained from parents. Initial assessment involved ADIS-P interviews with parents via telephone and the CY-BOCS interview with children and parents in the clinic. Interviewers were trained in diagnostic interviews and CY-BOCS interviews through skills training workshops, observation of expert clinicians, and supervision of their interviewing skills by the principal investigator of the clinical trials. Assessment also included the completion of a number of self-report questionnaires, and various parent-report questionnaires. Pre- and post-treatment assessments were completed at various time points; however, for the purposes of this study, pre-treatment assessment data only was used. Children participated in an individual CBT protocol across various clinical trials which included standard treatment components typical of evidenced-based CBT for OCD and based on March and Mulle's (1998) [46] original protocol.

Results

Overview of Results

Firstly, the current characteristics of the sample are described via means and standard deviations which are presented across the key variables. In order to address hypothesis 1, the means and standard deviations for the current sample, on measures of OCD symptom severity, functional impairment and SRS T-scores are presented. Additionally, the SRS total T-Scores are compared to the standardised sample [6], and frequencies within each of the interpretative ranges are reported. Furthermore, a series of independent *t* tests were used to assess differences between the two age groups (7–12 years; 13–17 years) on the variables under investigation. Hypothesis 2 was examined using bi-variate correlations to determine the strength of the association of the variables under investigation, and simple regression analyses were used to assess the predictive value of the SRS total T-Score on OCD-related symptom severity and functional impairment. Finally, hypothesis 3 was examined using mediation analysis to assess the role of family accommodation in mediating the relationship between ASD traits, OCD severity and functional impairment.

Sample Characteristics

The mean age of participants was 12.30 years (*SD* 2.68), and 45.0% were male. The group was predominately described as White, not of Hispanic origin 90.0%, with a further 2.5% described as Black, not of Hispanic origin, 2.5% as Native Asian or Pacific Islander, and 1.3% as Aboriginal or Torres Strait Islander. A further 3.7% did not report their ethnicity. Combined family income was also examined, with 41.3% reporting a combined annual family income of above $100,000, 38.8% reporting an income between $60,001 and $100,000, 8.8% between $30,001 and $60,000, 1.3% less than $30,000, and 9.8% did not report the family income. Mother's education level was reported as tertiary level (degree or post graduate degree qualifications) 43.8%, secondary school 38.8%, technical and further education (TAFE; certificate or diploma qualifications, or vocational training) 7.5%, not reported 9.9%. Father's education level was reported as tertiary 40.0%, secondary 41.3%, TAFE or vocational training 5.0%, and not reported 13.7%. Sixty percent of participants were not on medication, 33.8% were on a SSRI, 1.3% on a stimulant medication, 3.8% were on another type of medication, 1.2% did not report medication type, and 5.0% were on two medication types.

All participants met criteria for a primary diagnosis of OCD. Of note, 5% also had a previous diagnosis of ASD, Level 1. On average, in addition to the OCD diagnosis, participants received two additional diagnoses ($M = 2.16$, $SD = 1.17$) (see Table 1). Overall, within the sample of youth, anxiety disorders were the most prevalent secondary diagnosis (e.g., 37.5% GAD, 21.3% social phobia, 17.5% specific phobia) and tertiary diagnosis (e.g., 18.8% specific phobia, 15.0% GAD, 10.0% social phobia). In relation to mood disorders 3.8% of the sample received a secondary diagnosis of dysthymia (2.5%) and major depression disorder (1.3%), and 3.8% of the sample presented with a tertiary diagnosis of dysthymia (1.3%) and MDD (2.5%). Disruptive behavioural disorders within the sample indicated that 2.5% of youth had ADHD/ADD and 3.8% ODD as a secondary diagnosis, 3.8% were diagnosed with ADHD/ADD and 3.8% with ODD as a tertiary diagnosis, as a fourth diagnosis 6.3% had ADHD/ADD, 2.5% had ODD, and 1.3% had conduct disorder, and 2.5% of the sample received a fifth diagnosis of ODD.

Table 1 Sample characteristics

Demographics	n^1 (%)
Child's gender	
Male	36 (45.0%)
Child's ethnicity	
White, not of Hispanic origin	72 (90.0%)
Black, not of Hispanic origin	2 (2.5%)
Native Asian or Pacific Islander	2 (2.5%)
Aboriginal or Torres Strait Islander	1 (1.3%)
Combined family income	
$30,000 and under	1 (1.3%)
$30,001–$60,000	7 (8.8%)
$60,001–$100,000	31 (38.8%)
$100,000 and above	33 (41.3%)
Mother's education level	
Secondary	31 (38.8%)
TAFE or college	6 (7.5%)
Tertiary	35 (43.8%)
Father's education level	
Secondary	33 (41.3%)
TAFE or college	4 (5.0%)
Tertiary	32 (40.0%)
Medication	
No medication	48 (60.0%)
SSRI	27 (33.8%)
Stimulant	1 (1.3%)
Other	3 (3.8%)
Comorbid Tic diagnosis	
Motor tics	3 (3.8%)
Vocal tics	1 (1.3%)
Both motor and vocal tics	1 (1.3%)
Comorbid ASD Level 1 diagnosis	4 (5.0%)
Comorbid diagnosis	
Secondary diagnosis	77 (96.3%)
Tertiary diagnosis	51 (63.7%)
Fourth diagnosis	32 (40.0%)
Fifth diagnosis	12 (15.0%)
Sixth diagnosis	1 (1.3%)

[1]Total sample size $N = 80$

Diagnostic Characteristics and Clinical Ratings (Hypothesis 1)

OCD Symptoms

The mean total CY-BOCS score for the sample was 25.73 (*SD* 4.57), indicating that on average OCD symptoms were within the severe range. Furthermore, 64.0% of the sample scored within the severe range (24–31), and 6.3% scored within the extreme range (32–40) on the CY-BOCS. Further examination of the data across two age groups ($n = 42$; age 7–12 years) and ($n = 36$; age 13–17 years) indicated that the mean total CY-BOCS scores (see Table 2) suggested that on average OCD symptoms in youth across these two age groups were within the severe range. Furthermore, 52.3% of children aged 7–12 years, and 77.7% of adolescents aged 13–17 years, scored within the severe range (24–31), and 4.5% of children,

Table 2 Symptom severity, functional impairment, family accommodation and autistic traits (SRS) for children, adolescents and total sample

	Children 7–12 years *M* (SD)	Adolescents 13–17 years *M* (SD)	Total sample 7–17 years *M* (SD)	*t*	*p*
Total symptom severity CY-BOCS	24.62 (4.46)	27.03 (4.40)	25.73 (4.57)	−2.39	0.02*
Total functional impairment COIS-P	39.21 (20.69)	50.08 (29.90)	44.16 (25.72)	−1.90	0.06
Family accommodation	19.09 (12.44)	23.18 (11.17)	20.90 (11.99)	−1.50	0.14
SRS total and subscales T-scores					
SRS social awareness	62.80 (10.68)	65.06 (9.59)	63.81 (10.20)	−0.99	0.33
SRS social cognition	57.34 (11.16)	58.97 (9.74)	58.08 (10.51)	−0.69	0.49
SRS social communication	57.80 (8.60)	58.31 (7.62)	58.03 (8.13)	−0.28	0.78
SRS social motivation	59.39 (11.11)	57.97 (8.97)	58.75 (10.17)	0.62	0.54
SRS restricted repetitive behaviour	59.30 (11.66)	63.08 (11.06)	61.00 (11.48)	−1.48	0.14
SRS total	59.75 (9.65)	61.22 (8.21)	60.41 (9.00)	−0.73	0.47
SRS standardised sample (4–18 years) across gender $n = 2025$			30.90 (25.30)		

and 8.3% of adolescents, scored within the severe range (32–40).

ASD Traits

Overall, of the combined sample (aged 7–17 years), 57.5% reported elevated ASD traits above the normal range on the SRS (i.e., T score above 60). However, using a more stringent cut-off (T score >65), 32.5% of the sample were within the moderate to severe range for ASD symptoms. To examine possible age differences, the SRS total T-score and subscale T-scores were calculated across two age ranges, i.e., 7–12 years (children) and 13–17 years (adolescents) by splitting the sample into two equal age groups, which closely correspond to periods of development defined as aligning with the onset of adolescence [47]. Means, standard deviations and SRS T-scores across age ranges, and school-age (4–18 years) SRS-2 standardised sample [6] are presented in Table 2. A series of independent *t*-tests were performed to assess differences between the two age groups on variables under investigation. A significant difference between the child group and the adolescent group was found on CY-BOCS symptom severity, $t(76) = -2.39$, $p = .02$, Cohen's $d = -0.54$, indicating that the adolescent group experienced greater overall OCD severity on the CY-BOCS compared to the child group. No significant age group differences were found on measures of functional impairment, family accommodation, or the SRS total T-score and the SRS subscales T-scores (Table 2).

The clinical cut-off on the SRS is a total T-score of 60. The frequency of children and adolescents falling into the various severity ranges on the SRS, based on T-scores is reported in Table 3. Of note, 26 children within the sample (32.5%) presented across the two intervals of either moderate (27.5%) or severe (5%) range on the SRS. Moreover, of this more severe subsample, the mean SRS T-score was 70.58 ($SD = 5.44$) indicating 2 standard deviations above the mean of the standardised sample.

Correlations Among Variables and Regression Models (Hypothesis 2)

Correlations were assessed using Pearson's correlations coefficient (r) as a measure of the strength of the association between variables. No statistically significant correlation was found between the CY-BOCS total severity score and the COIS-P total impact score ($r = .16$, $p = .08$). The correlation between the CY-BOCS total severity score and the SRS total T-score was not statistically significant ($r = .13$, $p = .16$). However, the COIS-P total impact score (impairment) was positively and significantly correlated with the SRS total T-score ($r = .23$, $p = .02$). Importantly,

Table 3 SRS total T-score ranges for children, adolescents, and total sample

SRS total T-score ranges	Children 7–12 years *n* (%)	Adolescents 13–17 years *n* (%)	Total sample 7–17 years *n* (%)	*t*	*p*
Normal range (59T and below)	20 (45.5%)	14 (38.9%)	34 (42.5%)	0.59	0.56
Mild range (60T–65T)	10 (22.7%)	10 (27.8%)	20 (25.0%)	−0.51	0.61
Moderate range (66T–75T)	12 (27.3%)	10 (27.8%)	22 (27.5%)	−0.05	0.96
Severe range (76T or higher)	2 (4.5%)	2 (5.6%)	4 (5.0%)	−0.20	0.84

no significant correlations were found between age (as a continuous variable) and SRS total T-Score and subscales T-scores.

Using a simple regression model, the SRS total T-Scores were entered as a predictor of OCD-related impairment (COIS-P). SRS total T-Scores were a significant predictor of functional impairment as measured by the COIS-P, (R^2_{chg} = 0.05, F_{chg} (1, 77) = 4.03, p = .05, B = 0.63, SE = 0.32). These results suggest that autistic traits among youth with OCD account 5% of the variance in parent-reported functional impairment associated with OCD.

Mediation Among Variables (Hypothesis 3)

Given there was no significant relationship between ASD severity (SRS) and OCD symptom severity (C-YBOCS: model 1), mediation was only examined on impairment. The PROCESS macro provided by Hayes (2013) was used to assess the mediation model in this study (see Fig. 1), and the significance of the indirect path was assessed using bootstrapping methods (1000 samples). This software conducts regression analyses to assess the significance and magnitude of the *a, b, c*, and *ć* paths, and generates bias-corrected confidence intervals for the mediated pathway (*ab*). If the generated confidence interval does not span zero, the mediation effect is considered to be significant. The mediation model was tested using Process Model 4 [48], with SRS total T-score as the independent variable, total impact (COIS-P; model 2) as the dependent variable, and FAS as the mediator (see Fig. 1). For a full mediated effect to occur, direct pathways from the independent variable to the dependent variables should be non-significant after controlling for the mediator.

A significant association was found between SRS total T-scores and family accommodation (Path a), F (1,75) = 9.56, p = .003, B = 0.44, SE = 0.14, and between family accommodation and functional impairment (COIS-P; Path b) after controlling for SRS total, F (2,74) = 13.21, p = < 0.001, B = 0.99, SE = 0.21. However, no significant association was found between total SRS total and functional impairment (COIS-P; Path c'). These findings indicated that increased SRS total was associated with increased family accommodation, which in turn predicted increased functional impairment. After controlling for family accommodation, the association between SRS total and COIS-P became non-significant, F (1,75) = 3.14, p = .08, *B = 0.52, SE = 0.29* indicating a full mediation effect (see Table 4).

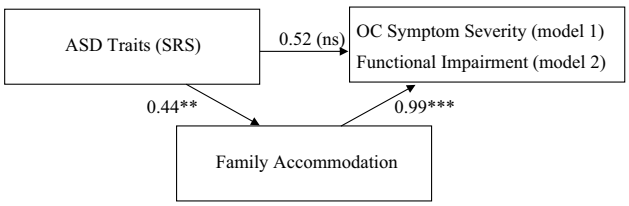

Fig. 1 Mediation model showing the pathway from ASD traits to OCD symptom severity and functional impairment with family accommodation as the mediator, **p < .01, ***p < .001

Discussion

This study extended the findings by Stewart et al. [5] who found that 36.2% of young children (n = 127; age 5–8 years) met clinically significant ASD traits (i.e., scoring 60 or higher) as measured on the SRS, and Ivarsson and Melin [4] who found that 8.26% of their sample met ASD diagnosis. The current study examined ASD traits using the same measure (SRS) across a broader age range of children and adolescents with OCD (n = 80; age 7–17 years) than previously published by Stewart et al. [5]. Furthermore, given that the clinical presentation of OCD differs across the developmental trajectory and is associated with different types of comorbidity at different ages [10], the sample was divided into two groups (i.e., 7–12 years and 13–17 years) to compare potential differences in the degree of ASD traits across younger children and adolescents. Three main hypotheses were tested: (1) that ASD traits are elevated among children and adolescents who have a primary diagnosis of OCD; (2) that elevated ASD traits will be associated with OCD severity and functional impairment; and, (3) that family accommodation will mediate the relationship between ASD traits and OCD severity and functional impairment.

Table 4 Mediation analysis on SRS and impact with family accommodation (FA) as the mediator

	B	*SE*	*β*	*p*	95% CI	
					LL	UL
Model 2, DV: impact (COIS-P)						
SRS total *T*-score and FA (path a)	0.44**	0.14	0.34	0.003	0.16	0.73
FA and impact (path b)	0.99***	0.21	0.51	0.000	0.59	1.41
SRS total *T*-Score and impact (path c')	0.52	0.29	0.20	0.081	−0.07	1.11

**p < .01, ***p < .001

To address the first hypothesis, this study found that approximately one-third of the sample (32.5%, $n=26$) exhibited moderate to severe levels of ASD traits. These results are indicative of clinically significant deficits in reciprocal social behaviour which are typically observed in children with autism spectrum disorders of moderate severity [6]. Similar levels of elevation on the SRS total T-scores were found across children (7–12 years) and adolescents (13–17 years). The elevated ASD traits in this study are aligned with previous research across both the adult and paediatric literature examining ASD traits among samples with diverse psychopathology, including anxiety, mood and behavioural disorders [49–52]. For example, Gilmore and colleagues [51] found that 66% of children with conduct disorder presenting to a general psychiatric clinic had impairment in pragmatic language and nonverbal communication behaviour similar in character and level to those in a comparison group of children with ASD. Pine and colleagues [49] found that patients with mood disorders exhibit higher scores on ASD symptom scales when compared to healthy youths. They demonstrated that youth with mood disorders exhibit impaired social reciprocity, language deficits, and behavioural rigidity, raising the question as to "whether ASD symptoms should be viewed as correlates of illness severity or of other nonspecific features of developmental psychopathologies" (p. 660).

The rate with which ASD traits were elevated in this study may also be a reflection of a more highly comorbid sample of youth with OCD relative to those typically reported in clinical trials. For example, this study was a highly complex, clinical sample, showing that 96.3% of participants had a secondary diagnosis, 63.7% had a tertiary diagnosis, and 40.0% had a fourth diagnosis, and extends previous findings in comorbidity research in OCD presentations. Prior research has found that around 80% of youth with OCD have a least one comorbid diagnosis [2, 3, 53] and around 50–60% have two or more psychiatric disorders during their lifetime [54]. Based on the findings of the current study, perhaps an argument could be made for initiating a higher threshold on ASD screening measures within OCD samples/cases, whilst simultaneously being mindful of likely comorbidity. This notion may provide the impetus for further research in the area, as well as provide clinicians with guidance on assessment considerations, given the potential for high levels of ASD traits within youth who have OCD.

Investigating the second hypothesis, no significant correlations were found between the CY-BOCS total symptom severity scores and the COIS-P total impact scores, or the CY-BOCS total symptom severity scores and the SRS total T-Scores, or the SRS subscales. These results lend support for findings by Lewin et al. [55] who found youth with comorbid ASD and OCD rate their OCD symptoms as equally distressing, time consuming, and disruptive as do youth without ASD. Hence, elevated ASD traits alone may not be associated with OCD symptom severity in youth with comorbid OCD and ASD, but suggests that comorbid ASD traits may be associated with greater impairment in functioning than OCD without ASD traits. Correspondingly, the COIS-P (OCD-related functional impairment) showed a positive and significant correlation between the SRS total T-score, and the SRS subscales T-scores of social cognition, social communication, social motivation and restricted, repetitive behaviours, suggesting that elevated ASD traits significantly impact young people's functioning when they have a diagnosis of OCD. These findings align with Griffiths et al. [18] who found that youth with comorbid OCD and ASD exhibited significantly greater functional impairment, higher levels of comorbid psychiatric disorders, including behavioural difficulties (e.g., ADHD), and higher levels of family accommodation, when compared to youth with OCD but without ASD. The results presented herein suggest that elevated ASD traits among youth with OCD were associated with greater impairment.

The third hypothesis was partially supported, by the finding that family accommodation mediated the relationship between ASD traits and functional impairment (COIS-P). These results suggest that ASD traits predict higher levels of family accommodation, which in turn mediates the degree of impairment experienced. Thus, family accommodation is associated with the level of functional impairment in youth with OCD, particularly in those that have an added vulnerability of clinical levels of ASD traits. Family accommodation has also been found to have the potential to limit the individual's opportunities to develop problem solving skills and in turn places undue strain on relationships with family members [23]. Therefore, examining the extent to which other parenting variables such as parent psychopathology (depression, anxiety and stress), and rearing styles characterised by emotional warmth, rejection, overprotection, and anxious rearing [56], may exert an influence over accommodating behaviours is suggested. Further understanding of the broader factors that increase family accommodation may be helpful in designing more targeted interventions to assist children and adolescents with OCD and elevated ASD traits. In sum, these findings support the notion that ASD traits within OCD samples of youth may reflect a shared clinical phenomenology, as well as point to family accommodation as a possible shared mechanisms of maintenance across the two disorders, or similarly support the view that the additive effects of comorbid disorders as suggested by Caron and Rutter [57] may have the potential to exacerbate impairment and dysfunction. Whilst the current study provides evidence of associations among these important variables, it does not address direction or causality amongst them. Future research with prospective

longitudinal designs may provide further insight into the precise nature of such associations and the development of comorbid symptoms over time.

The aim of the current study was to explore the role of dimensional ASD symptoms and traits on OCD severity and impairment, and as such, we did not use structured psychometrically validated interviews to specifically screen for a diagnosis of ASD. Despite this, the SRS-2 is routinely used to screen for clinical levels of ASD and along with all other measures in this study is a psychometrically robust tool, which was evidenced by excellent internal consistency for the total SRS-2 in the current sample. However, notwithstanding the SRS-2's robust psychometric properties, the findings from this study also highlighted potential difficulties of using the SRS-2 as a measure of autistic traits in youth with OCD. For instance, given that 57.5% of the currently sample fell above the clinical cut-off score of 60 T, these findings suggest that the SRS-2 may be too sensitive a measure to differentiate OCD from ASD. As such further investigation into the SRS's suitability as a screening measure for comorbid OCD and ASD is warranted.

Additionally, the sample in the current study comprised predominantly of mid-to-high socioeconomic status and high levels of education, thereby limiting generalisation of these findings. Although family accommodation explained a meaningful amount of variance between the SRS and OC functional impairment in this study, the cross-sectional design and exploratory nature of the mediation analysis limits the interpretation of these results to a bidirectional finding only.

Implications and Direction for Future Research

In conclusion, the findings from this study demonstrated that in this sample, 32.5% of children and adolescents who presented with a primary diagnosis of OCD also had elevated ASD traits to within a moderate and severe range as measured by SRS-2, and in turn experienced significantly more OCD-related functional impairment. Furthermore, these difficulties were mediated by accommodating behaviours within the family unit. Overall, the current study expands our understanding of the role of ASD traits within OCD paediatric populations, and highlights the importance for clinicians to appropriately screen for both disorders to assist diagnosis and to preclude diagnostic overshadowing. Further empirical research would be helpful in both adult and paediatric samples to increase our understanding of how widespread the prevalence of elevated ASD traits within OCD populations is. This research also raises the question of whether comorbid OCD and ASD is a subtype of OCD, and as such warrants different approaches to screening and treatment protocols to more accurately diagnose and address deficits in both disorders. Given the SRS-2 measure has the potential to be too sensitive in assessing ASD traits in youth with OCD, future research that examines the specificity of the SRS measure, and its ability to differentiate youth with OCD from ASD would be helpful, in order to determine its usefulness as a clinical screen for ASD among youth with OCD.

These findings suggest that for clinicians working with children who present with OCD, a comprehensive screening for ASD traits may be helpful to inform treatment protocols. Further understanding of the clinical presentation of OCD and the wide heterogeneity of this disorder would provide an opportunity to design more individualised conceptual models and approaches to evidence based treatments. This study also highlights the importance of addressing family accommodation early in treatment in order to improve functioning and optimise treatment outcomes.

Summary

In the present study, high rates of autistic traits (i.e., 32.5%) as measured by the SRS were found in youth who presented to a university psychology clinic for assessment and treatment of a primary diagnosis of OCD, when compared to a standardised sample of typically developing youth [6]. Furthermore, SRS scores for these youth were found to be within the moderate to severe range which is typically found in children and adolescents with a diagnosis of ASD. Additionally, ASD traits were associated with significantly more OCD-related functional impairment across important domains of psychosocial functioning, and were mediated by family accommodation. The findings from this study indicated that OCD and autistic traits co-occurs at higher than expected rates in clinical samples, and highlights the importance for clinicians to appropriately screen for both disorders to assist diagnosis and inform treatment protocols. Further empirical research would be helpful to examine the prevalence of ASD traits in OCD populations.

References

1. Farrell L, Waters A, Milliner E, Ollendick T (2012) Comorbidity and treatment response in pediatric obsessive–compulsive disorder: a pilot study of group cognitive-behavioral treatment. Psychiatry Res 199(2):115–123
2. Lewin AB, Chang S, McCracken J, McQueen M, Piacentini J (2010) Comparison of clinical features among youth with tic disorders, obsessive–compulsive disorder (OCD), and both conditions. Psychiatry Res 178(2):317–322
3. POTS Team (2004) Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive–compulsive disorder: the pediatric OCD treatment

study (POTS) randomized controlled trial. J Am Med Assoc 292(16):1969–1976
4. Ivarsson T, Melin K (2008) Autism spectrum traits in children and adolescents with obsessive–compulsive disorder (OCD). J Anxiety Disord 22(6):969–978
5. Stewart E, Cancilliere MK, Freeman J, Wellen B, Garcia A, Sapyta J et al (2016) Elevated autism spectrum disorder traits in young children with OCD. Child Psychiatry Hum Dev 47(6):993–1001
6. Constantino JN, Gruber CP (2012) Social responsiveness scale, second edition (SRS-2). Western Psychogical Services, Torrance
7. Anholt GE, Cath DC, van Oppen P, Eikelenboom M, Smit JH, van Megen H et al (2010) Autism and ADHD symptoms in patients with OCD: are they associated with specific OC symptom dimensions or OC symptom severity? J Autism Dev Disord 40:580–589
8. Bejerot S, Nylander L, Lindström E (2001) Autistic traits in obsessive–compulsive disorder. Nord J Psychiatry 55(3):169–176
9. Cath DC, Ran N, Smit JH, Van Balkom AJLM, Comijs HC (2007) Symptom overlap between autism spectrum disorder, generalized social anxiety disorder and obsessive–compulsive disorder in adults: a preliminary case-controlled study. Psychopathology 41(2):101–110
10 Farrell L, Barrett P, Piacentini J (2006) Obsessive–compulsive disorder across the developmental trajectory: clinical correlates in children, adolescents and adults. Behav Chang 23:103–120
11. Leyfer OT, Folstein SE, Bacalman S, Davis NO, Dinh E, Morgan J et al (2006) Comorbid psychiatric disorders in children with autism: interview development and rates of disorders. J Autism Dev Disord 36(7):849–861
12. Neil N, Sturmey P (2014) Assessment and treatment of obsessions and compulsions in individuals with autism spectrum disorders: a systematic review. Rev J Autism Dev Disord 1(1):62–79
13. American Psychiatric Association (1994) Diagnostic and statistical manual of mental disorders: DSM-IV, vol 4th. American Psychiatric Association, Washington
14. American Psychiatric Association (2000) Diagnostic and statistical manual of mental disorders: DSM-IV-TR. vol 4th, text revision. American Psychiatric Association, Washington
15. Mukaddes NM, Fateh R (2010) High rates of psychiatric comorbidity in individuals with Asperger's disorder. World J Biol Psychiatry 11(2–2):486–492
16. Mukaddes NM, Hergüner S, Tanidir C (2010) Psychiatric disorders in individuals with high-functioning autism and Asperger's disorder: similarities and differences. World J Biol Psychiatry 11(8):964–971
17. Chasson GS, Timpano KR, Greenberg JL, Shaw A, Singer T, Wilhelm S (2011) Shared social competence impairment: another link between the obsessive–compulsive and autism spectrums? Clin Psychol Rev 31(4):653–662
18. Griffiths DL, Farrell LJ, Waters AM, White S (2016) Obsessive–compulsive disorder in autism: clinical expression and treatment response, in Manuscript submitted for publication
19. Zandt F, Prior M, Kyrios M (2009) Similarities and differences between children and adolescents with autism spectrum disorder and those with obsessive compulsive disorder: executive functioning and repetitive behaviour. Autism 13(1):43–57
20. Scahill L, Riddle MA, McSwiggin-Hardin M, Ort SI (1997) Children's Yale-Brown obsessive compulsive scale: reliability and validity. J Am Acad Child Adolesc Psychiatry 36(6):844–852
21. Caporino NE, Morgan J, Beckstead J, Phares V, Murphy TK, and Storch EA (2012) A structural equation analysis of family accommodation in pediatric obsessive-compulsive disorder. J Abnorm Child Psychol 40(1):133–143
22. Merlo LJ, Lehmkuhl HD, Geffken GR, Storch EA (2009) Decreased family accommodation associated with improved therapy outcome in pediatric obsessive-compulsive disorder. J Consult Clin Psychol 77(2):355–360
23. Storch EA, Geffken GR, Merlo, LJ, Jacob, ML, Murphy TK, Goodman WK et al (2007) Family accommodation in pediatric obsessive-compulsive disorder. J Clin Child Adolesc Psychol 36(2):207–216
24. Lebowitz ER, Panza KE, Su J, Bloch, MH (2012) Family accommodation in obsessive-compulsive disorder. Expert Rev Neurother 12(2):229–238
25. Garcia AM (2010) Predictors and moderators of treatment outcome in the pediatric obsessive compulsive treatment study (POTS I). J Am Acad Child Adolesc Psychiatry 49(10):1024–1033
26. Mazefsky CA, Herrington J, Siegel M, Scarpa A, Maddox BB, Scahill L et al (2013) The role of emotion regulation in autism spectrum disorder. J Am Acad Child Adolesc Psychiatry 52(7):679
27. White SW, Mazefsky CA, Dichter GS, Chiu PH (2014) Social-cognitive, physiological, and neural mechanisms underlying emotion regulation impairments: understanding anxiety in autism spectrum disorder. Int J Dev Neurosci 39:22–36
28. American Psychiatric Association (2013) Diagnostic and statistical manual of mental disorders, 5th edn. American Psychiatric Association, Washington
29. Conners CK, March JS (1996) Conners–March developmental questionnaire (CMDQ). Multi-Health Symstems Inc, Toronto
30. Silverman WK, Albano AM (1996) Anxiety disorders interview schedule for DSM-IV: parent interview schedule. Oxfor University Press, New York
31. Silverman WK, Nelles WB (1988) The anxiety disorders interview schedule for children. J Am Acad Child Adolesc Psychiatry 27(6):772–778
32. Silverman WK, Saavedra LM, Pina AA (2001) Test-retest reliability of anxiety symptoms and diagnoses with the anxiety disorders interview schedule for dsm-iv: child and parent versions. J Am Acad Child Adolesc Psychiatry 40(8):937–944
33. Kendall PC (1994) Treating anxiety disorders in children: results of a randomized clinical trial. J Consult Clin Psychol 62(1):100–110
34. Barrett PM, Dadds MR, Rapee RM (1996) Family treatment of childhood anxiety: a controlled trial. J Consult Clin Psychol 64(2):333–342
35. Ollendick TH, Ost LG, Reuterskiold L, Costa N, Cederlund R, Sirbu C et al (2009) One-session treatment of specific phobias in youth: a randomized clinical trial in the United States and Sweden. J Consult Clin Psychol 77(3):504–516
36. Knox LS, Albano AM, Barlow DH (1996) Parental involvement in the treatment of childhood obsessive compulsive disorder: a multiple-baseline examination incorporating parents. Behav Ther 27(1):93–114
37. Waters TL, Barrett PM, March JS (2001) Cognitive-behavioral family treatment of childhood obsessive–compulsive disorder: preliminary findings. Am J Psychother 55(3):372–387
38. Barrett P, Healy-Farrell L, March JS (2004) Cognitive-behavioral family treatment of childhood obsessive–compulsive disorder: a controlled trial. J Am Acad Child Adolesc Psychiatry 43(1):46–62
39. Farrell LJ, Waters AM, Boschen MJ, Hattingh L, McConnell H, Milliner EL et al (2013) Difficult-to-treat pediatric obsessive–compulsive disorder: feasibility and preliminary results of a randomized pilot trial of d-cycloserine-augmented behavior therapy. Depression Anxiety 30(8):723–731

40. Piacentini J, Jaffer M, Bergman RL, McCracken J (2001) Measuring impairment in childhood OCD: Psychometric properties of the COIS. In: Proceedings of the American Academy of Child and Adolescent Psychiatry Meeting
41. Bruni TP (2014) Test review: social responsiveness scale—second edition (SRS-2). J Psychoeduc Assess 32(4):365–369
42. Rutter M, Bailey A, Lord C (2001) Social communication questionnaire (SCQ). Western Psychology Services, Los Angeles
43. Bishop DVM (1998) Development of the children's communication checklist (CCC): a method for assessing qualitative aspects of communicative impairment in children. J Child Psychol Psychiatry Allied Discip 39(6):879–891
44. Calvocoressi L, Lewis B, Harris M, Trufan SJ, Goodman WK, McDougle CJ et al (1995) Family accommodation in obsessive–compulsive disorder. Am J Psychiatry 152(3):441–443
45. Flessner CA, Freeman JB, Sapyta J, Garcia A, Franklin ME, March JS et al (2011) Predictors of parental accommodation in pediatric obsessive–compulsive disorder: findings from the pediatric obsessive–compulsive disorder treatment study (POTS) trial. J Am Acad Child Adolesc Psychiatry 50(7):716–725
46. March JS, Mulle K (1998) OCD in children and adolescents: a cognitive-behavioral treatment manual. Guilford Press, New York
47. Edwards B (2014) Growing up in Australia: the longitudinal study of Australian children: entering adolescence and becoming a young adult. Fam Matters 95:5–14
48. Hayes AF (2013) An introduction to mediation, moderation, and conditional process analysis: a regression-based approach. Guilford Press, New York
49. Pine DS, Guyer AM, Goldwin M, Towbin KE, Leibenluft E (2008) Autism spectrum disorder scale scores in pediatric mood and anxiety disorders. J Am Acad Child Adolesc Psychiatry 47(6):652–661
50. Towbin KE, Pradella A, Gorrindo T, Pine DS, Leibenluft E (2005) Autism spectrum traits in children with mood and anxiety disorders. J child Adolesc Psychopharmacol 15(3):452–464
51. Gilmour J, Hill B, Place M, Skuse DH (2004) Social communication deficits in conduct disorder: a clinical and community survey. J Child Psychol Psychiatry 45(5):967–978
52. Liew SM, Thevaraja N, Hong RY, Magiati I (2015) The relationship between autistic traits and social anxiety, worry, obsessive–compulsive, and depressive symptoms: specific and non-specific mediators in a student sample. J Autism Dev Disord 45(3):858–872
53. Storch EA, Larson MJ, Merlo LJ, Keeley ML, Jacob ML, Geffken GR et al (2008) Comorbidity of pediatric obsessive–compulsive disorder and anxiety disorders: impact on symptom severity and impairment. J Psychopathol. Behav Assess 30(2):111–120
54. Rasmussen SA, Eisen JL (1990) Epidemiology of obsessive compulsive disorder. J Clin Psychiatry 51(2 Suppl.):10–13
55. Lewin AB, Wood JJ, Gunderson S, Murphy TK, Storch EA (2001) Phenomenology of comorbid autism spectrum and obsessive–compulsive disorders among children. J Dev Phys Disabil 23(6):543–553
56. Castro J, Toro J, Van der Ende J, Arrindell WA (1993) Exploring the feasibility of assessing perceived parental rearing styles in Spanish children with the EMBU. Int J Soc Psychiatry 39(1):47–57
57. Caron C, Rutter M (1991) Comorbidity in child psychopathology: concepts, issues and research strategies. J Child Psychol Psychiatry Allied Discip 32(7):1063–1080