Obsessive-compulsive disorder in children and youth: Neurocognitive function in clinic and community samples.

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ABSTRACT

Background. Neurocognitive impairments are common in OCD, although not well studied in children and youth with the disorder. **Method**. Using the stop-signal task (SST), we measured response inhibition (stop-signal reaction time - SSRT), sustained attention (reaction time variability – RTV), reaction time (RT) and performance monitoring (post-error slowing - PES) in OCD cases and controls from two samples of children and youth. A Clinic OCD group (*n*=171, aged 7-17 years) was recruited from a specialty clinic after rigorous assessment. A typically developing (Clinic TD, *n*=157) group was enlisted through advertisement. A community OCD sample (Community OCD, *n*=147) and controls (Community TD *n*=13,832) were recruited at a science museum. We also identified a community group with high OCD traits without an OCD diagnosis (Community High Trait; *n*=125). **Results.** Clinic OCD participants had longer SSRT and greater RTV than Clinic TD. These effects were greater in younger OCD participants and, for SSRT, in those on medication for OCD. The Community OCD group did not differ from Controls but was like the Clinic OCD group in ADHD and ASD comorbidity and medication usage. The Community High Trait group had longer SSRT and atypical PES suggesting that symptom severity predicts neurocognitive function. No group differences were found in RT. **Conclusions**. In the largest study of neurocognitive performance in children with OCD to date, we found impaired response inhibition and sustained attention in OCD participants in comparison to typically developing peers. Performance was worse in younger OCD participants. In the community sample, participants with high OCD trait scores but no OCD diagnosis had impaired response inhibition and error processing, suggesting that OCD might be under-recognized.

Keywords: Stop-signal task; OCD; neurocognition; executive function; comorbidity

Obsessive-compulsive disorder (OCD) is a common and heritable condition characterized by unwanted, repetitive thoughts and behaviors (IOCDF-GC and OCGAS, 2018; Pauls et al., 2014). An extensive literature points to fronto-executive impairments manifest in neurocognitive deficits in response inhibition, flexibility, planning, working memory and error monitoring in the mechanism of OCD in adults (Robbins, Vaghi, & Banca, 2019). However, there is a substantial gap in our knowledge of neurocognitive function in OCD among children and youth (Gooskens et al., 2019; Negreiros et al., 2019; Ornstein, Arnold, Manassis, Mendlowitz, & Schachar, 2010; Woolley et al., 2008). Younger OCD patients are impaired in the ability to inhibit distractors (e.g., Stroop task; Abramovitch et al., 2021), but there has been far less research into other important neurocognitive processes such as response inhibition, sustained attention and error monitoring. In a recent meta-analysis of research using the stop-signal task (SST), a widely used measure of response inhibition (the ability to stop a response; Verbruggen, et al., 2019), only 4 of 21 studies involved individuals younger than 20 years of age (Mar, Townes, Pechlivanoglou, Arnold, & Schachar, 2021). Response inhibition longer in children and youth with OCD, but the impairment was half as large as what was observed in adults suggesting potentially critical differences between children and adults with OCD. Age effects in OCD could arise from genetic or neural factors (Van Grootheest, Cath, Beekman, & Boomsma, 2005; Piras et al., 2021), age-related cognitive deterioration in OCD (Andrés et al., 2008) or effects of intercurrent treatment (Skandali et al., 2018). Few studies in children and youth provide evidence about the effects of age, gender, comorbidity, and medication taken at the time of testing (e.g., SSRIs; Veselinović et al., 2019). Finally, most of our knowledge on neurocognitive function and OCD is based on samples recruited in specialty clinics, calling into question the generalizability of findings (Low

et al., 2008). Impairment in neurocognitive function may be overestimated in clinical samples because more severely affected individuals with multiple comorbidities are often referred to specialty clinics (Pearce & Richiardi, 2014).

To begin to fill these research gaps, we studied children and youth from clinics and the community. The clinical sample consisted of children and youth with a diagnosis of OCD made in a specialty clinic using rigorous protocols (Clinic OCD) and a group of typically developing youth (Clinic TD) who were screened in the clinic. The community sample included individuals with a reported diagnosis of or treatment for OCD (Community OCD), a group from the community with extremely high scores for OCD traits (Community High Trait) but who did not report an OCD diagnosis, and a group of TD children (Community TD). We compared groups on measures of response inhibition, sustained attention, reaction time and performance monitoring (defined below) while taking gender, age, comorbidity, OCD severity and the use of medication into account. We hypothesized that Clinic OCD, Community OCD and Community High Trait groups would exhibit comparable neurocognitive function that would be worse than in TD controls.

METHOD

Participants

Clinic Participants

Participants with OCD or TD controls were recruited from tertiary care clinics as part of the Province of Ontario Neurodevelopmental Disorders (POND) Network. The Clinic OCD sample consisted of 171 participants (aged 7-17.9 years) that received an OCD diagnosis and

any other comorbid diagnosis after assessment by a clinician using DSM-5 criteria following a rigorous, clinical assessment by a psychologist or psychiatrist using a semi-structured clinical interview (Kiddie Schedule for Affective Disorders and Schizophrenia; Kaufman et al., 1997) and the Children's Yale-Brown Obsessive Compulsive Scale; CY-BOCS; Scahill et al., 1997) for OCD, Autism Diagnostic Observation Schedule–2 (ADOS) and Autism Diagnostic Interview–Revised (ADI-R; Lord et al., 2000) for ASD, and the Parent Interview for Child Symptoms (PICS; Ickowicz et al., 2006) for other comorbidities. Clinic TD participants (n= 157) were free of any mental illness.

Community Participants

The community participants were recruited from visitors (*n*=16,148, aged 7.0–17.9 years) to a public science museum (Crosbie et al., 2013). Parents reported diagnoses/treatment received for OCD, ADHD and ASD and rated behavioral traits of their children. When parent report was unavailable, we included self-report for participants 12+ years of age (*n*=2,909). We excluded 16 participants who reported a diagnosis of schizophrenia. The total sample was 16,019 participants. We measured OCD traits over the past 6 months using the Toronto Obsessive-Compulsive Rating Scale (TOCS; Park et al., 2016). The TOCS has 21 items scored on a seven-point Likert scale ranging from -3 ("far less often than others of the same age") to +3 ("far more often than others of the same age") with higher scores representing more OCD symptoms. We measured ADHD traits using the Strengths and Weaknesses of ADHD Symptoms and Normal Behavior Scale (SWAN; Burton et al., 2019, see Appendix S1 in the Supporting Information for details). We calculated standardized scores taking age, gender, and respondent into account. We also categorized scores of ≥2 on the TOCS or SWAN as an 'OCD symptom' or

'ADHD symptom' respectively. Both TOCS and SWAN scores show good reliability and validity (Burton et al., 2018).

Informants were asked about medication usage within the 24 hours prior to testing. Medications were categorized into those used to treat OCD (selective serotonin reuptake inhibitors - SSRIs, serotonin-norepinephrine reuptake Inhibitor - SNRIs, tricyclic antidepressant - Clomipramine), antipsychotics (e.g., risperidone) and stimulant/non-stimulant medications used to treat ADHD (e.g., methylphenidate or dextroamphetamines).

We classified participants as Community OCD if they reported a diagnosis of or treatment for OCD by a health practitioner. The High Trait group was defined as those with a t-score >70.5 (98%ile) standardized for age, gender, and respondent (parent/self) in the *absence* of a reported OCD diagnosis (*n*=125). Participants were classified as TD unless they met criteria for Community OCD or High Trait, reported a diagnosis of ASD or ADHD or had ADHD trait t-scores >66.4 (95%ile) for age, gender, and respondent (*n*=1,915). Comorbidity was defined as, and limited to, reported ASD or ADHD.

Ethical Considerations

Informed consent, and verbal assent when applicable, approved by the local institution's ethics review board, were obtained from all participants.

Measuring neurocognitive function

We measured neurocognitive function using the SST (Dupuis et al., 2019; see Appendix S1). We estimated response inhibition (stop-signal reaction time, SSRT) using interpolation

(Verbruggen, et al., 2019). Longer SSRT reflects worse inhibitory control. Lapses in attention were estimated from standard deviation of response times (RTV) across all go trials following a correct go response. Greater RTV indicates poorer ability to maintain arousal and attention (Karalunas et al., 2014). Mean reaction time on go responses (trials without stop signals) following a correct go response reflects speed of mental processing (Rommelse et al., 2020). We measured performance monitoring as slowing of RT on trials following unsuccessful (i.e., erroneous) stop-signal trials (post-error slowing, PES).

Statistical Analyses

Statistical analyses were performed using SAS 9.4 (SAS, 2012). We examined the effect of an OCD diagnosis on SSRT, RTV, RT and PES. We used a log transformation of all but the PES to correct their heteroscedasticity across age (higher variability at younger ages – e.g., community SSRT SD at age 6 was 198 ms compared to community SSRT SD at age 17 at 104 ms) and to derive a more parsimonious model of their non-linear decline with increasing age. Effects that are additive on the log transformed variables are multiplicative in the original (i.e., non-log transformed) scale. Hence, all effects are represented as % difference and as a result, the corresponding effect in milliseconds will be greater where predictors are associated with larger predicted values (e.g., longer SSRT at younger age) and smaller where predictors are associated with smaller predicted values. Raw scores are plotted in the figures and are presented in the Table S1 along with the age at which the models predict a control would perform at the same level. We examined the relationship of high OCD traits with SSRT, RT, RTV, and PES by comparing the performance of individuals with high trait scores but no OCD diagnosis to the TD group. We predicted a main effect of group on SSRT, RTV and PES and

tested for differences in group trajectories across age, for a total of 12 primary hypotheses (3 SST outcomes x 2 settings x 2 effects (age x group interaction and group main effect). We use a multiple comparison adjusted α of 0.05/12 = 0.004. Community models included siblings and therefore family was treated as a random effect. Models controlled for the effect of age, gender, and respondent where significant. We also examined the effect of SSRI medication on performance based on medication use in 24 hrs prior to assessment. We added ADHD trait scores (SWAN t-scores) to models with a significant group effect to test whether comorbid ADHD explained the group differences and included OCD trait scores (TOCS t-scores) to assess the impact of OCD severity. In a sensitivity analysis, we both controlled for comorbid disorders and ran the model on participants with no comorbid disorders. Three clinic participants were taking medications for OCD other than SSRIs and were not included in the OCD+SSRI medication group.

RESULTS

Group Characteristics

There were 147 participants (0.9% of total sample) in the Community OCD group. Prevalence of Community OCD was higher in males than females (*p*=.028, see Figure S1) and was slightly higher in self-report (1.9%, *m*age = 15.3) than parent-report participants (1.5%, *m*age = 13.4) and increased significantly with age (p<.0001). This increase was unlikely the result of over-reporting of OCD in older participants because older participants were no more likely to report a diagnosis of OCD in the absence of elevated trait or symptom scores or

medication treatment. There were 125 participants (0.8%) in the Community High Trait group, 171 in the Clinic OCD and 157 Clinic TD participants (Table 1).

Both Clinic and Community OCD groups had significantly higher TOCS t-scores than their respective controls. Clinic OCD cases had higher TOCS t-score than Community OCD cases (3.1-point difference, 95%CI: 1.2;5.1, *p*=.018). The High Trait group had significantly higher TOCS t-scores than either the Clinic or Community OCD groups (High Trait vs Community OCD: 11.2-point difference, 95%CI: 8.9;13.4, p<.0001; High Trait vs Clinic OCD: 8.1-point difference, 95%CI: 6.0;10.2, p<.0001). Clinic and Community OCD groups, but not the High Trait group, had significantly higher ADHD trait scores than Community or Clinic TD (Community OCD vs TD:7.6-point difference, 95%CI: 6.0;9.2, p<.0001; Clinic OCD vs TD: 11.5-point difference 95%CI: 9.1;14.0, p<.0001; High Trait vs TD: 0.1-point difference, 95%CI: -1.7;1.9, p=0.9). The median number of OCD symptoms was higher in the Clinic than Community OCD group and highest in the High Trait group. The Clinic, Community OCD and High Trait groups had few ADHD symptoms. Comorbidity of OCD with ADHD, ASD or both was common in Community, Clinic and High Trait groups (Table 2) with ADHD being the most common comorbid diagnosis.

About 30% of Clinic and Community OCD participants were taking an SSRI or SNRI (Table 3). Younger clinic OCD participants taking SSRIs had higher TOCS scores than those not taking SSRIs (Figure S2). ADHD medication was reported in 39% of the Clinic OCD cases with a comorbidity and 48% of the Community OCD cases with a comorbidity, of which 7% were taking a non-stimulant ADHD medication. Antipsychotic use was uncommon among OCD cases without a comorbidity (1-2%), but more common among those with ADHD or ASD comorbidity (9% of Clinic and 19% of Community cases). Medication use was rare in the Community High

Trait group except for those who reported comorbidity, 38% of whom were taking medication for ADHD.

SSRT

As shown in Figure 1, Community OCD and Community TD did not differ in SSRT (5.6% greater SSRT in Community OCD vs TD 95% CI: -3.1,15.0%, *p*=.2). The difference in SSRT between the High Trait group and the Community TD group was close to twice the difference between OCD and TD [10.4% greater SSRT in high trait vs TD (95%CI: 0.5,21.2%, *p*=.038], but the effect was not significant after controlling for multiple comparisons. There was no significant effect of comorbidity, age, SSRI, or stimulant medication on SSRT in the Community samples. Females had longer SSRT [4.0%, 95%CI: 2.3,5.8%, *p*<.0001] in both the Community OCD and High Trait groups.

In the clinic sample, there was a significant age by diagnosis interaction [F(1,312)=15.94, p<.0001] with greater group differences between younger participants than older participants. SSRI medication, the most reported medication, was associated with a 22.8% greater SSRT (p=.0007) but this did not eliminate the effect in unmedicated participants. Figure 1c shows SSRT for Clinic OCD and Clinic TD groups with the Clinic OCD group further divided into those who were and were not taking an SSRI. The significant age by group interaction [F(2,310)=11.06, *p*<.0001] remained after controlling for SSRI medication use. In young participants, SSRT was longer (worse) in Clinic OCD participants taking, rather than not taking, SSRIs, but both groups had longer SSRT than the Clinic TD group. Controlling for any medication at all, the effect remained in the unmedicated OCD group, indicating that the effect in this group was not driven by the few who were taking antipsychotic medications but no SSRIs. The

estimated difference from the model between TD and OCD at age 12.3 years (average age in our sample) was 45.5 ms. Among older participants, no differences in SSRT were noted whether participants were using SSRIs or not. There was no effect of gender comorbid disorders, or SWAN t-scores on SSRT in the clinic sample and the results did not change when we ran the models on participants with no comorbid disorders only. TOCS t-scores were significant predictors of SSRT in the clinic sample [TOCS by age interaction F(1,309)=8.81,p=.003] but not when together in the model with OCD diagnosis.

RT and RTV

Community OCD, High Trait and Community TD did not differ significantly in RTV (Figures 2a and 2b). By comparison, Clinic OCD had significantly greater RTV than Clinic TD (12.1% greater RTV, 95% CI: 4.6,20.1%, *p*=.001; Figure 2c). Age influenced RTV with older participants having lower RTV, but age did not affect the difference between Clinic OCD and Clinic TD. OCD medication, stimulant medication, SWAN t-scores, TOCS t-scores, and comorbid disorders had no significant impact on this pattern of results, and the effect remained when participants with comorbid disorders were excluded from the model. TOCS predicted RTV in the clinic sample (*p*=.020) but not when OCD diagnosis was in the model (*p* = 0.9). There was no difference in mean reaction time between OCD and TD groups in either sample (Figure S3).

PES

Clinic and Community OCD groups did not differ in PES from TD controls. However, there was a significant age by group effect in the community sample [F(2,11E3)= 7.16, p=.0008] such that the High Trait group slowed significantly more than the Community TD group at younger ages and sped up after errors at older ages (Figure S4) Controlling for SWAN t-scores,

TOCS t-scores, or comorbidities did not eliminate this effect, and the effect remained when participants with comorbid disorders were excluded from the model.

DISCUSSION

As predicted, we found that the clinic OCD group had longer SSRT than TD controls, indicating slower, less efficient response inhibition. Younger individuals and those with highest OCD trait scores showed the greatest impairments. This effect diminished into late adolescence, where OCD and controls had similar performance. The age at which children participated in the study may be correlated with "time since diagnosis", with younger children more likely to be in earlier in their treatment of OCD, as evidenced by the more elevated TOCS scores in younger than older children taking SSRIs. Evidence of poor response inhibition in OCD contradicts studies that show no or a very limited difference in inhibitory control between children and youth with OCD and controls (e.g., Hybel et al., 2017). Response inhibition was worse in clinic OCD participants who were receiving SSRIs compared to OCD participants who were not, but this effect was evident only in the younger Clinic OCD participants. Worse inhibitory control in clinic cases on SSRIs was not a function of comorbidity with ADHD or ASD or with OCD trait severity. Worse inhibition in medicated OCD participants could reflect an adverse effect of medication, although previous studies have shown improvement in inhibition in adults on SSRIs (Skandali et al., 2018). SSRI treatment may also be a marker for more severe OCD. As predicted, we observed greater RTV in Clinic OCD compared to TD controls. The increase in RTV variability in OCD did not vary with medication use, comorbidity, gender, or age. Comorbidity was not significant in any of the models, controlling for SWAN did not change OCD effects, and the OCD group did not have particularly high ADHD symptom counts. Like other

studies, we did not find any main effects of OCD on RT (Rommelse et al., 2020) or evidence of atypical PES.

Greater OCD severity predicted worse inhibitory control but not over and above the effect of OCD. The absence of any within group effect of OCD severity in the presence of a between group effect suggests a threshold effect of severity on cognition. The question of whether OCD is best understood as the extreme of a widely distributed trait or as a category with boundaries is an important unanswered question.

We considered the clinical as well as statistical significance of observed impairments by examining age equivalence--the age at which the model predicts a control would perform at the same level. For example, a 12-year-old with OCD not using SSRI medication is predicted to have an SSRT of 272 ms – the same value of SSRT expected in a control at 10 years of age. Therefore, the observed deficit is equivalent to a 2-year lag in development. Another way to think of the magnitude of the observed impairment in response inhibition is braking in a car. A 20% increase in time to brake (the magnitude of the difference in SSRT in the current study) in a car going 65 mph would amount to 1 second longer for the car to stop, during which time it would have covered an additional 100 feet. if the car was going 65 mph. Apparently, small effects can have serious real-life circumstances.

Our results have implications for the pathophysiology of OCD in childhood. Response inhibition is familial, heritable, and stable, and is affected by stimulant medication and drugs used to treat OCD (Mcauley, Crosbie, Charach, & Schachar, 2013; Schachar, Forget-dubois, Dionne, Boivin, & Robaey, 2011; Tannock, Schachar, & Logan, 1995; Wilkins, 2009), although

drug effects on response inhibition and its clinical implications have not been adequately studied in OCD children and youth. RTV is also heritable (Finkel & Pedersen, 2014) and has been attributed to variation in energetic states (Sergeant, 2005), extinction processes (Sagvolden, Johansen, Aase, & Russell, 2005), or arousal (Tamm et al., 2012). However, slower, less efficient response inhibition and increased RTV are not unique to OCD, but are found in several disorders (Lipszyc & Schachar, 2010). Cognitive biomarkers that are shared by different disorders could point to sharing of genetic or other risk factors or may arise for different reasons in different disorders. To understand if neurocognitive impairment in OCD is prior to or causal to development of the disorder, mediation and moderation analyses of genetic variants, neurocognitive function and OCD traits or diagnosis could be helpful once clear genetic risks are established.

Contrary to prediction, we did not observe impaired inhibition or increased RTV, RT or PES in Community OCD participants. The most likely explanation is that the Community OCD cases were somewhat less severe than clinic cases as was evident in their lower OCD trait and symptom scores. This possibility is supported by the fact that those in the high trait group showed a greater difference from controls in neurocognitive function than did the Community OCD group and the fact that OCD trait severity predicted worse function in the Clinic sample. One possibility is that community reported diagnosis is inaccurate, perhaps identifying individuals a different mental health concern without neurocognitive impairment. However, community and clinic OCD participants were remarkably similar in mean age, gender distribution, severity of OCD and comorbid ADHD traits, number of OCD symptoms, proportion

with comorbid ADHD and ASD diagnoses and medication usage. These similarities provide some support for further study of OCD identified in the general population using reported diagnoses.

The proportion of cases with high OCD traits but without a reported OCD diagnosis was comparable to previous reports in adult samples (de Bruijn, Beun, de Graaf, ten Have, & Denys, 2010; Ruscio, Stein, Chiu, & Kessler, 2010). Apparently, many children experience high OCD symptoms or traits but neither receive an OCD diagnosis nor are identified as requiring an assessment or care for OCD. Whether the high OCD trait participants in our study had undiagnosed OCD is unknown as we did not measure functional impairment and distress. However, the Community High Trait group showed longer SSRT (which did not survive correction for multiple significance testing) and atypical PES. Among the Community High Trait group, younger participants slowed more after errors than controls did, whereas older children with high OCD traits slowed less. In fact, they sped up. Post-error slowing reflects detection of and adjustment to errors, a critical neurocognitive skill for attaining life goals and correcting the kind of action errors seen in OCD (Dupuis et al., 2019; Hanna et al., 2018). Given that the high trait group had evidence of neurocognitive impairments, further study of this group is needed to determine whether the effect is driven by missed OCD diagnoses or by other conditions that may contribute to elevated TOCS scores but that were not measured in the present study.

The prevalence of OCD in the community increased with age (p<.0001) with a higher prevalence in males than females (p=.028), consistent with prevalence estimates of epidemiological studies in older people which use more rigorous diagnostic methods (Torres et al., 2006). We examined the possibility that prevalence was inflated among older participants because they were more likely than younger participants to be self-reporting their OCD

diagnosis. We found that older, self-report OCD participants were no more likely than younger, parent-report participants to report a diagnosis in the absence of high trait scores or OCD medication usage. This result adds to the limited number of population-based studies of OCD in children and adolescents (Osland, Arnold, & Pringsheim, 2018).

LIMITATIONS

The most obvious limitation was the definition of OCD in the community sample, the lack of a measure of functional impairment and possible comorbidities (e.g., anxiety and depression). We measured OCD traits over the previous 6 months. OCD traits tend to wax and wane - therefore, we do not know how performance would be affected by OCD state severity on the day of assessment. Additionally, both the community and clinic samples are likely to be shaped by hidden biases. Severely affected individuals may not attend science museum or be unable/willing to participate in our study. Specialty clinics might be biased toward more severe participants. We chose not to fit ex-Gaussian models to response time distributions. Ex-Gaussian models assume two-stages affecting response times -- one with a normal distribution and one with an exponential distribution. However, there are multiple stages to response (perception, decision, response) that are not distributed normally or exponentially. The current study was limited to four components to neurocognition. Further research into neurocognition in OCD is clearly needed.

CONCLUSION

Impaired response inhibition and greater reaction time variability might play a role in the inability of people with OCD to identify and stop impulsive action and thought. Medication

use was associated with greater impairment and may be a marker of severity. Although impaired neurocognition was not evident in the community OCD sample, their characteristics and prevalence suggest that collection of OCD cases in the community, particularly based on trait scores, could make an important contribution to etiological studies in OCD. Performance monitoring requires further study in OCD.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Table S1. Predicted raw SST values and control age equivalence for significant group effects.

Figure S1. Prevalence of Community OCD and High Trait in the Community Sample by Age and Gender.

Figure S2. TOCS t-scores by Age and Medication Status in the Clinic Sample.

Figure S3. Reaction Time (RT) by Age in Community, Clinic, High Trait and TD Groups.

Figure S4. Post-Error Slowing (PES) by Age in Community, Clinic, High Trait and TD Groups.

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Key Points

- Limited research on neurocognition in children and youth with OCD but studies suggest possible age differences.
- Largest study to date shows impaired response inhibition and increase reaction time variability in children and youth with OCD.
- Neurocognitive problems might contribute to difficulty suppressing unwanted thoughts and fears (obsessions) and repetitive behaviors (compulsions) in OCD.

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Table 1

Age, OCD and ADHD Trait Scores for OCD and TD Groups (Means) in Clinic and Community Samples

			Age	TOCS [2] t-score	TOCS Symptom [3]	SWAN [4] t-score	SWAN Symptom[3]
Clinic		***n (%)***	***Mean (sd)***	***Mean (sd)***	***Median (IQR)***	***Mean (sd)***	***Median (IQR)***
TD	Males	96 (61)	11.8 (2.7)	41.5 (10.9)	0 (0;0)	44.7 (10.3)	0 (0;0)
	Females	61 (39)	11.9 (3.0)	40.7 (11.6)	0 (0;0)	42.6 (10.6)	0 (0;0)
OCD	Males	102 (60)	12.5 (2.6)	65.8 (9.2)	7 (4;11)	55.2 (12.0)	2 (0;7.9)
	Females	69 (40)	13.0 (2.5)	65.1 (9.0)	8 (3.6;11)	54.6 (12.0)	0 (0;4.5)
Community							
TD	Males	6,741 (49)	10.9 (2.6)	49.6 (9.6)	0 (0;2)	47.7 (8.5)	0 (0;2)
	Females	7,091 (51)	11.4 (2.9)	49.8 (9.7)	0 (0;2)	48.5 (8.8)	0 (0;1)
OCD	Males	82 (56)	12.9 (2.8)	61.9 (10.7)	5 (2;10)	57.4 (10.9)	4 (1;9)
	Females	65 (44)	13.5 (3.0)	62.5 (9.1)	6 (3;11)	52.4 (12.1)	1 (0;3)
High Trait[1]	Males	74 (59)	11.0 (2.8)	74.0 (3.2)	11 (9;14)	48.5 (13.1)	2 (0;6)
	Females	51 (41)	12.6 (3.3)	73.0 (2.1)	13 (12;15)	47.9 (13.4)	1 (0;3)

[1] Includes only those with a TOCS t-score >70.5 but no community diagnosis of OCD.

[2] TOCS =Toronto Obsessive-Compulsive Scale

[3] A symptom is defined as a score ≥2; IQR: Interquartile Range

[4] SWAN = Strengths and Weaknesses of ADHD Symptoms and Normal Behavior Rating Scale

Table 2

Comorbidity[1] for OCD and TD Groups (n, %) in Clinic and Community Samples

			No comorbidity	**+ ADHD [2]**	**+ ASD[3]**	**+ ADHD & ASD**
		n	***n (%)***	***n (%)***	***n (%)***	***n (%)***
Clinic						
OCD dx	Males	102	60 (59)	33 (32)	3 (3)	6 (6)
	Females	69	54 (78)	12 (17)	1 (1)	2 (3)
Community						
OCD dx	Males	82	26 (32)	42 (51)	5 (6)	9 (11)
	Females	65	47 (72)	18 (28)	0	0
High Trait	Males	74	53 (72)	12 (16)	7 (9)	2 (3)
	Females	51	46 (90)	1 (2)	3 (6)	1 (2)

[1] Comorbidity defined as and limited to ADHD and/or ASD.

[2] ADHD = Attention-deficit/hyperactivity disorder

[3] ASD = Autism spectrum disorder

Table 3

Medication Usage in OCD and TD Groups (n, %) in Clinic and Community Samples

			Medication		
			SSRI/SNRI	**ADHD[2]**	**Antipsychotic**
		n	***n (%)***	***n (%)***	***n (%)***
Clinic					
OCD dx	- comorbid	114	36 (32)	0	2 (2)
	+ comorbid	57	19 (33)[1]	22 (39)	5 (9)
Community					
OCD dx	- comorbid	73	20 (27)	0	1 (1)
	+ comorbid	74	19 (26)	35 (47)[3]	14 (19)
High trait only	- comorbid	99	2 (2)	0	0
	+ comorbid	26	1 (4)	10 (38)	0

[1] Includes one patient taking a tricyclic antidepressant for the treatment of OCD

[2] ADHD medications were stimulants, atomoxetine, clonidine and guanfacine

[3] 5/47 participants were taking a non-stimulant ADHD medication (cf clonidine, guanfacine).

Figures

Figure 1: Response Inhibition (SSRT) by Age in Community, Clinic, High Trait and TD Groups. For clarity, results for Community OCD (a) and High Trait (b) groups are shown separately. SSRT results for Clinic OCD are presented separately for those taking SSRI (c). Clinic OCD (with and without comorbidity) did not differ in SSRT. The bands around each line represent the 95% confidence interval. Data were grouped into age bins and simple means within age bins are represented as points to visually assess the general fit of the model.

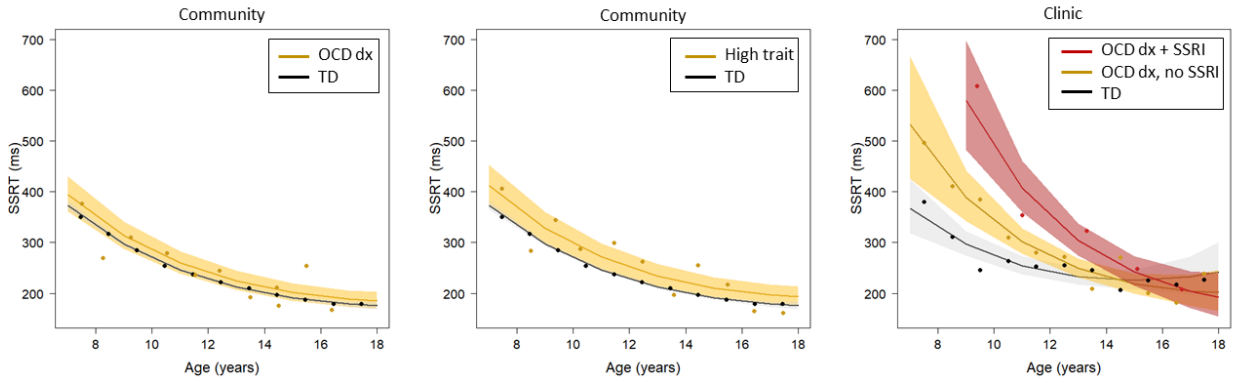

Figure 2: Reaction Time Variability (RTV) by Age in Community, Clinic, High Trait and TD Groups. Clinic OCD had significantly greater RTV than Clinic TD (12.1% greater SD, 95% CI: 4.6,20.1%, p=.001). The bands around each line represent the 95% confidence interval. Data were grouped into age bins and simple means within age bins are represented as points to visually assess the general fit of the model.