Examining the Conners 3–Short Form Predictability on Comorbid ADHD Symptomatology in Children that have been Diagnosed with ASD

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Abstract

With the publication of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V) in 2013, newfound interest grew around the comorbidity of ASD and ADHD. However, the restructuring of both diagnosable descriptors has led to concerns surrounding prevalence rates, deliverable treatments, and identification of comorbid symptomatology. This study focuses on the use of the Conners, Third Edition-Parent Short Form [C3-P(S)]. In the current longitudinal study with 239 participants of typically developing youth (n=102) and youth with ASD (n=137), the C3-P(S) is administered alongside other screening and diagnostic tools. Using the data gathered from the measure alongside their diagnosis of ASD, the study aims to analyze if there is a significant difference between the C3-P(S) subscale scores of the TD and ASD groups. From there, this project serves to make predictions around whether participants present with a profile of co-occurring ADHD symptoms. A second goal is to observe which of the six subscales stands out the most in predicting ADHD comorbid symptomatology in participants who meet the criteria based on the C3-P(S). The results of this study found significant differences among all categories of the C3-P(S) between TD and ASD youth. A larger percentage of ASD children met criteria for ADHD as compared to their TD peers. Additionally, among participants who met screener criteria for ADHD, children with ASD scored significantly higher in the hyperactivity/impulsivity, learning problems, and peer relations subscales. These findings suggest potential predictability of differentiating ASD and ADHD symptomatology using the C3-P(S) if paired with more robust diagnostic tools.

Introduction

Both autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) have become more prevalent in recent decades and a significant amount of literature has been published on their diagnosis, symptoms, and treatment. Not until the publication of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V) in 2013 were clinicians and researchers able to properly characterize these two disorders in the context of each other (American Psychiatric Association, 2013). In the most recent decade, this publication has spurred newfound interest in how ASD and ADHD co-occur (Antshel and Russo, 2019).

As the world's expanding knowledge of neurodevelopmental disorders continues to grow, it has become exponentially more important to look at the crosstalk between each of these unique disorders and their symptoms. Focusing on the comorbid symptomatology of ASD and ADHD can provide scientists and clinicians with the necessary tools to tailor treatments and drug therapies to individuals who have been diagnosed with both conditions.

Brief Description of ASD

Affecting more than 5 million Americans, ASD impacts 1 in 36 children (Maenner et al., 2023). This prevalence is around 10% higher than it was almost a decade ago and is diagnosed in boys 4.3 more times than girls (Maenner et al., 2020). ASD, a neurodevelopmental condition, is characterized by two core features: impaired social communication and restricted, repetitive, or unusual sensory-motor behaviors (APA, 2013). The symptoms of ASD are most identifiable in children between the ages of two and three. The disorder is very heterogenous such that prognosis is influenced by age at diagnosis, sex assigned at birth, and demography (Hyman et al., 2020). Diagnosing this disorder is therefore challenging and many professionals are working to

improve the diagnostic accuracy of the many screening tools available (Thabtah and Peebles, 2019). Oftentimes, the most reliable method is clinically held interviews that may be inaccessible to many, so it is important to highlight tools that can be used to initially recognize the symptoms of ASD. Awareness of the disorder is especially essential to educate the public about its unique expression and create more effective pathways to treatments.

Among children with ASD, an estimated 33% are classified as having intellectual disability (Maenner et al., 2020). Breaking this percentage down, 39% of girls versus 32% of boys are diagnosed as having intellectual disability. The percentage is also higher among black and Hispanic children than their white counterparts. It is important to note that although intellectual disability is more highly diagnosed in black and Hispanic children, they are less likely to receive an early diagnosis (Maenner et al., 2020). Researchers and clinicians use a variety of screening measures and tools to understand the best paths for diagnosis and intervention in ASD; therefore, knowing how to identify the right measures to use is essential to differentiating the different appearances of ASD.

Brief Description of ADHD

With a diagnosis rate nationally of 8.4%, ADHD is one of the most common mental disorders in children and adolescents (Wolraich et al., 2019). The main characteristics of ADHD include excessive motor activity, inattention, and impulsiveness (APA, 2013). The DSM-V describes ADHD as a childhood-onset development disturbance that persists for at least six months and continues across home and social situations (APA, 2013). There are a variety of aspects such as demographics that could influence the initial diagnosis. For example, sex can

influence whether the hyperactivity of boys can be measured at a similar level to girls or if impulsivity is stronger in boys than girls (Wolraich et al., 2019).

ADHD is a constantly changing disorder throughout development. The clinical presentation of the disorder is essential to consider longitudinally as children grow into adulthood. In younger children, inattentiveness in classrooms and at home is often one of the first and most obvious symptoms (Thomas et al., 2015). Specifically in school-aged children, the symptom characteristics are recognized in parent and teacher reports and a prominent clinical challenge is understanding how that interpretation transfers to differentiating between comorbid conditions (Leffa et al., 2022). More than half of children with ADHD will present with at least one other comorbidity (Leffa et al., 2022). Symptoms can be exacerbated when they co-occur with other problems such as anxiety and depression (Thomas et al., 2015). In adolescence, motor unrest gradually fades out while impulsivity and decision-making deficits persist. These problems can continue to result in poor academic performance and isolation that predict worse quality of life (Leffa et al., 2022).

Focus on Comorbid ASD+ADHD: Attentional Control and Cognitive Function

Since the description and acknowledgement of co-occurrence for ASD and ADHD in the DSM-V, new investigations have suggested that greater attention deficits exist among children with comorbid ASD and ADHD relative to those with ASD only (APA, 2013). Co-occurring mental health disorders are reported at higher numbers in people with ASD as compared to the general population (Lai et al., 2019). Specifically, regarding co-occurrences with ADHD, individuals are shown to have greater impairments of adaptive functioning, quality of life, and

cognitive functioning. Due to the high prevalence of these two disorders in younger children, it is essential to focus on this population.

Inattention, a symptom most often seen in individuals affected by ADHD, is characterized by trouble paying attention to detail, staying attentive and controlling distractions, and completing tasks or routines. Researchers hoping to assess the relationship between social functioning and inattention in children with ASD only, ADHD only, and comorbid ASD+ADHD, reviewed caregiver-reported data and retrospective medical records to identify the difference among groups (Ng et al., 2019). The study found that among participants with ASD+ADHD, differences in social functioning were significantly attributed to attention problems. This was also mirrored in participants with only ADHD, but not ASD only children.

For children who have been diagnosed with both ASD and ADHD, the symptom severity of hyperactivity/impulsivity is thought to match that of children with either only ADHD or only ASD. Hyperactivity/impulsivity symptoms are thought to involve emotional problems with self-control and cognitive restrictive behaviors (APA, 2013). In Ng et al. (2019), researchers assessed the associations between reported inhibition control and social impairment. The study found that hyperactivity/impulsivity problems consistently remained throughout all groups as a significant factor that impacts social functioning abilities. Another study noted that amongst co-occurring ASD+ADHD cases, the ADHD domain of the condition is best represented by hyperactivity/impulsivity, while the ASD domain is characterized by the social communication deficiency (Krakowski et al., 2022).

Learning problems can arise even in typically developing (TD) children, but it is often magnified by the co-occurrence of developmental disorders like ASD and ADHD. Cooper et al. (2014) was interested in how severity of ASD symptoms within a cohort of children diagnosed

with ADHD would impact academic performance and contribute to greater learning problems. Researchers found that greater ASD symptomatology within ADHD youths also contributed to a higher prevalence of low IQ and working memory challenges, both important components of learning problems.

Executive functioning (EF) serves as an umbrella concept that includes physical, cognitive, and emotional self-control which allow for inhibition, working memory, cognitive flexibility, and planning and goal setting (Corbett et al., 2009). Early studies suggest the deficits in EF is at the core of ASD and ADHD and can provide the insight necessary to differentiate between the two disorders (Corbett et al., 2009). Corbett et al. (2009) aimed to investigate EF deficits for ASD and ADHD compared to TD peers without controlling for ADHD symptoms. The diagnosis was performed using the DSM-IV (American Psychiatric Association, 1994). The results showed that deficits in inhibition in children with ASD suggests that they share cognitive profiles consistent with ADHD (Corbett et al., 2009). Overall, children who present with symptoms of both ASD and ADHD showed more impairment functionally than their TD peers.

An updated look at EF in the context of the two disorders, Lee et al. (2021) examined parent- and teacher-reported executive dysfunction to ASD and ADHD symptom severity. After analysis of the screened data from the Conner 3rd Edition-Parent Short Form (Conners, 2008), researchers found that performance in EF subcategories could predict ADHD symptom severity, but not ASD symptom severity (Lee et al., 2021). The study concludes that children diagnosed with ASD who display more severe ADHD symptoms may have greater executive dysfunction compared to children who only have ASD.

Focus on Comorbid ASD+ADHD: Social Functioning

Impaired social performance as well as the subcategory of defiance/aggression are commonly screened for in children with neurodevelopmental disorders (Satterfield et al., 1994). Pouw et al. (2013) served to differentiate between TD children and ASD children in terms of their correlation of aggression and empathy. The study found that there is a significant difference between how TD and ASD children experience defiance/aggression. For ASD children, aggression was a consequence of deficits in emotion regulation or poor environmental adjustment. Reactive aggression for TD children, however, came from the inhibiting role of empathy (Pouw et al., 2013).

Satterfield et al. (1994) aimed to evaluate how childhood levels of defiance in ADHD boys predicted their adolescent offender rates in comparison with a TD control group. When higher levels of defiance/aggression levels were seen in these younger ADHD children, researchers found that those boys were more likely to be arrested in the future. Even boys in the ADHD category who scored lower on defiance/aggression levels as compared to those in the TD group still were more likely to commit a felony.

Lawson et al. (2015) chose to test whether flexibility in ASD and inhibition in ADHD influenced more anxious/depressed or defiant/aggressive behavior. Researchers found that ASD predicted greater anxiety/depression, while ADHD predicted greater defiance/aggression. While the conditions lead to different reactions, their similar symptomatology can be used in the future for predicting comorbidity.

A big part of social functioning is an understanding of children's relationships with their peers. Peer relations help understand the severity of social functioning symptoms for ADHD and other related neurodevelopmental disorders (Waddington et al., 2018). Considering the

comorbidity of ASD and ADHD, Waddington et al. (2018) chose to investigate children with diagnoses and their unaffected siblings. The purpose was to compare emotion recognition and quantify the influence of the ASD+ADHD phenotype on symptom display. The study showed that participants consistently performed worse than their siblings. Among the categories of ASD only, ADHD only, and ASD+ADHD, the severity of their emotional recognition deficits was the same; therefore, making the role of differentiating between comorbidities much more difficult.

Rationale and Hypothesis

Previous research shows the complexity of understanding the interaction between ADHD and ASD symptomatology. It is then helpful to utilize diagnostic tools that can serve as a reliable first step in deciding whether a diagnosis of comorbidity is apparent. One of many rating scales used in clinical and research settings, the Conners 3–Parent Short Form (C3-P(S)) screens for a diagnosis of ADHD and its common comorbid conditions (Conners, 2008). Gomez et al. (2019) examined the accuracy of the Conners 3–Parent Short Form in ADHD and comorbid oppositional defiant disorder (ODD). Researchers studied clinic-referred children between the ages of 6-11 years with a diagnosis of both conditions. From their analysis, researchers concluded that the C3-P(S) can be used to discriminate between ADHD and ODD especially well in the hyperactivity/impulsivity and defiance/aggression subscales (Conners, 2008). This finding provides support for the continual use of this screening measure in the diagnosis of other comorbid conditions such as ASD and ADHD.

While the C3-P(S) cannot provide an official diagnosis of ADHD, it helps researchers to identify symptom presentation that may be consistent with a diagnosis and to compare profiles across groups. By using the measure in this project, the foundational evidence necessary in

determining whether the C3-P(S) predicts ADHD comorbid symptomatology will be examined. The specificity of the C3-P(S) and its individual subscales may allow for a deeper investigation into which symptoms best predict ADHD comorbidity.

In the current longitudinal study performed in the SENSE Lab with TD youth and youth with ASD during pubertal development, the C3-P(S) is administered alongside other screening and diagnostic tools (Corbett, 2017). Using the data gathered from the measure along with their diagnosis of ASD, the study aims to analyze if there is a significant difference between the C3-P(S) subscale scores of participants who occupy the TD group and those who belong to the ASD group. From there, this project serves to make predictions around whether participants present with a profile of co-occurring ADHD symptoms. A second goal is to observe which of the six subscales stands out the most in predicting ADHD comorbid symptomatology in participants who meet the criteria based on the C3-P(S).

Methods

Participants

The data was collected from the SENSE Lab's longitudinal study on pubertal development (Corbett, 2017). This project utilizes data from Year 1 enrollment, which includes children between the ages of 10 year 0 months to 13 years 11 months.

The sample included 239 participants with the ASD group enrollment at 137 children (mean age = 11.43) and the TD group enrollment at 102 (mean age = 11.72). The ASD group included 35 females and 103 males, while the TD group included 44 females and 58 males. The racial and ethnic breakdown of the total sample included 7.9% African American, 83.6% White, and 8.2% Mixed. Demographic information is provided in **Table 1.**

Table 1

Demographic and Diagnostic Variables

	N	TD (*N* = 102)	ASD (*N* = 137)	Overall (*N* = 239)
Sex: male	161	0.57 (58/102)	0.75 (103/137)	0.67 (161/239)
Sex: female	79	0.43 (44/102)	0.26 (35/137)	0.33 (79/239)
Age	239	*M* = 11.72	*M* = 11.43	239
Race				
Caucasian		0.81 (111/137)	0.87 (86/102)	0.82 (197/239)
Black		0.12 (17/137)	0.02 (2/102)	0.08 (19/239)
Mixed Race		0.07 (9/137)	0.11 (11/102)	0.08 (20/239)

	N	TD		ASD	
		M	*SD*	*M*	*SD*
IQ	237	117.37	13.83	100.91	20.77
SCQ	236	2.51	2.68	17.36	8.34
ADOS total	137	-	-	12.57	4.58

Recruitment for this study spanned a broad community sample in the Southern United States covering a 200-mile radius that utilized medical and health-related services, clinics, research registries, regional autism/disability organizations, schools, and social media platforms. The inclusion criteria required an intelligence quotient (IQ) score of 70 or greater. The exclusion criteria included current use of medications known to alter the hypothalamic-pituitary-adrenal axis (ie. corticosteroids) or a medical condition known to impact pubertal development. After

completion of the screening portion of the study, 24 participants were excluded who did not meet inclusion criteria.

This study was approved by the Vanderbilt University Institutional Review Board and consistent with the Helsinki Declaration for research involving human participants. Written informed consent was given and obtained from parents as part of inclusion criteria, and research participants gave both verbal and written assent. All recruitment material was conducted using IRB approved flyers, emails, and clinic and health center systems.

Measures

The diagnosis of ASD was based on the Diagnostic and Statistical Manual-5 (APA, 2013). It was also confirmed through three different criteria: (1) a previous diagnosis by a psychologist, psychiatrist, or behavioral pediatrician with autism expertise; (2) current critical judgment, and (3) supported by the Autism Diagnostic Observation Schedule–2nd edition (ADOS-2) (Lord et al., 2012).

Autism Diagnostic Observation Schedule-2nd Edition (ADOS-2; Lord et al., 2012) is a semi-structured play and interview-based measure for the support of the diagnosis of ASD. The ADOS-2 was administered in a research setting by qualified clinical professionals. All ASD participants or suspected ASD participants were administered the ADOS-2 to corroborate diagnosis.

Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) is a general measure of intelligence, which can be used to estimate intellectual functioning. Test-retest reliability for full-scale estimated IQ is 0.95 (Wechsler, 1999).

Social Communication Questionnaire (SCQ; Rutter et al., 2003) is a 40-item parent-report measure of impairment in social communication associated with ASD. The SCQ was also used to screen typically developing children to confirm that scores were well below the ASD threshold (≥15 is suggestive of ASD, ≥22 is suggestive of autism). Discriminative ability to differentiate ASD from non-ASD ranges from 0.74 to 0.94.

Conners 3–Parent Short Form (C3-P(S); Conners, 2008) was also given during the enrollment session for each participant. The C3-P(S) screens for a diagnosis of ADHD and its common comorbid conditions. Previous literature has suggested that the C3-P(S) has been used to discriminate ADHD only and ADHD with a co-occurrence of ODD (Gomez et al., 2019). From their analysis, researchers concluded that the C3-P(S) has the potential to differentiate between the diagnosis of other comorbid conditions such as ASD and ADHD. The C3-P(S) contains the scores of each subscale of inattention (IA), hyperactivity/impulsivity (HI), learning problems (LP), executive functioning (EF), defiance/aggression (DF), and peer relations (PR). In the subscale categories, a *t*-score of over 60 may indicate that the child has symptoms of ADHD; a *t*-score between 60 and 70 indicates the child has moderate to severe symptoms; a *t*-score of over 65 indicates that the symptoms may fall into a clinical range and the child meets criteria for ADHD (Conners, 2008).

Design and Procedure

During the screening visit of the study, participant eligibility was assessed at the university-based clinic. After participants were determined to have met the inclusion criteria, the participant and their caregiver were asked to complete the other measures, including the C3-P(S).

Statistical Analysis

The aim of this project is to first compare the ADHD symptom profiles of children with ASD to those who are typically developing. The statistical analysis was completed using SPSS software (version 28; IBM SPSS Statistics, IBM Corporation) and statistical significance was determined at $p < 0.05$ using two-tailed tests. Descriptive statistics were calculated by applying means and standard deviations for continuous variables. A one-way ANOVA was applied to each subscale score as dependent variables, and the diagnosis as the independent variable. Chi-squared analyses was employed to reinforce the criteria met in the ANOVA and mirrored the approach seen from Gomez et al. (2019). The conclusion from this primary investigation will lead to further evaluation of ADHD symptom profiles and identify those participants who meet criteria in the ADHD category as given by the C3-P(S)–scoring a total of 65 or greater.

To measure the second aim to determine which of the subdomains of the C3-P(S) is the most elevated, a regression analysis is applied to investigate the relationship between diagnosis groups that have met criteria for ADHD and the subscale scores as the predictors.

Results

Descriptive statistics of the recorded data from participant C3-P(S) are presented in **Table 2** and **Figure 1**. Inattention, hyperactivity/impulsivity, learning problems, and executive

function, defiance/aggression, and peer relations are subscales of the diagnostic tool. As seen, the means for the ASD group meet the ADHD criteria threshold of 65 or greater in each of the subscales; specifically, IA, HI, LP, EF, and PR, while the TD group does not meet the threshold in any subscale. It is important to note that the differential total numbers of completed subscales is due to caregivers not completing certain sections or missing questions that could not be verified after the initial visit.

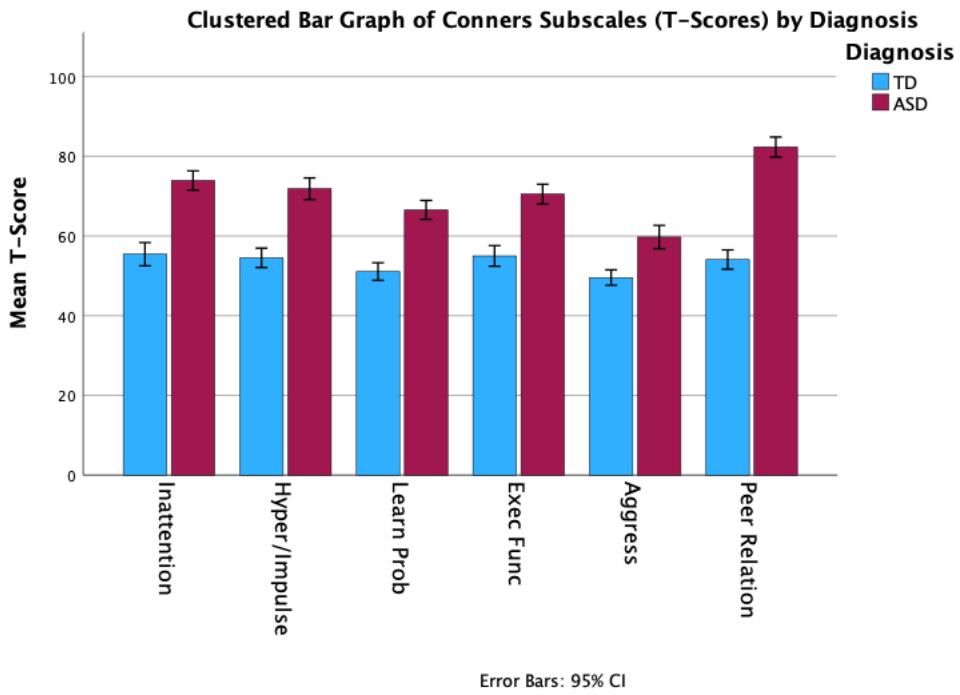
Table 2

Descriptives from C3-P(S)

Subscale	Diagnosis	N	Mean	Std. Deviation	Std. Error
Inattention	**TD**	102	55.77	15.05	1.49
	ASD	130	74.51	13.667	1.199
	Total	232	66.27	17.034	1.118
Hyperactivity/Impulsivity	**TD**	101	54.51	12.268	1.221
	ASD	135	72.96	15.379	1.324
	Total	236	65.07	16.81	1.094
Learning Problems	**TD**	102	51.39	11.503	1.139
	ASD	135	66.89	13.19	1.135
	Total	237	60.22	14.647	0.951
Executive Function	**TD**	102	55.34	13.512	1.338
	ASD	137	71.1	13.796	1.179
	Total	239	64.38	15.724	1.017
Defiance/Aggression	**TD**	102	49.61	9.776	0.968
	ASD	137	60.09	16.261	1.389
	Total	239	55.62	14.787	0.957
Peer Relations	**TD**	102	54	12.213	1.209
	ASD	136	82.93	13.751	1.179
	Total	238	70.53	19.418	1.259

Figure 1

Mean T-Scores for Conners Subscales of TD and ASD Groups



Hypothesis 1.

To examine the prevalence of ADHD-related symptomatology between TD and ASD youths, this study aimed to investigate if there is a significant difference between the C3-P(S) subscale scores of participants in the two groups. A one-way ANOVA was chosen to best express the score difference between the TD and ASD groups. As shown in **Table 3**, there are differences for recorded scores for all subscales. Focusing on IA, we see that there is a highly significant difference between scores of the TD group and ASD group ($F[1, 230] = 98.216$, $p < 0.001$, $\eta^2 = 0.299$).

Table 3

One-Way ANOVA of Dependent Variables (Primary Subscales)

Subscale	Group Difference	df	F	Sig.	η2
Inattention	**Between Groups**	1	98.216	<0.001	0.299
	Within Groups	230			
	Total	231			
Hyperactivity/Impulsivity	**Between Groups**	1	98.432	<0.001	0.296
	Within Groups	234			
	Total	235			
Learning Problems	**Between Groups**	1	89.404	<0.001	0.276
	Within Groups	235			
	Total	236			
Executive Function	**Between Groups**	1	77.642	<0.001	0.247
	Within Groups	237			
	Total	238			
Defiance/Aggression	**Between Groups**	1	33.412	<0.001	0.124
	Within Groups	237			
	Total	238			
Peer Relations	**Between Groups**	1	283.552	<0.001	0.546
	Within Groups	236			
	Total	237			

Table 4 also provides results of the distribution of ASD and TD youth meeting ADHD criteria. A percentage breakdown by applying a chi-squared analysis was also effective to better interpret the significant difference between the TD and ASD groups. The findings show that 12.9% of the TD group met criteria for ADHD, while 62.5% of the ASD group predicted comorbid symptomatology. The reported chi-squared analysis of the data also showed significant differences ($\chi^2(1) = 57.652$, $p < 0.001$).

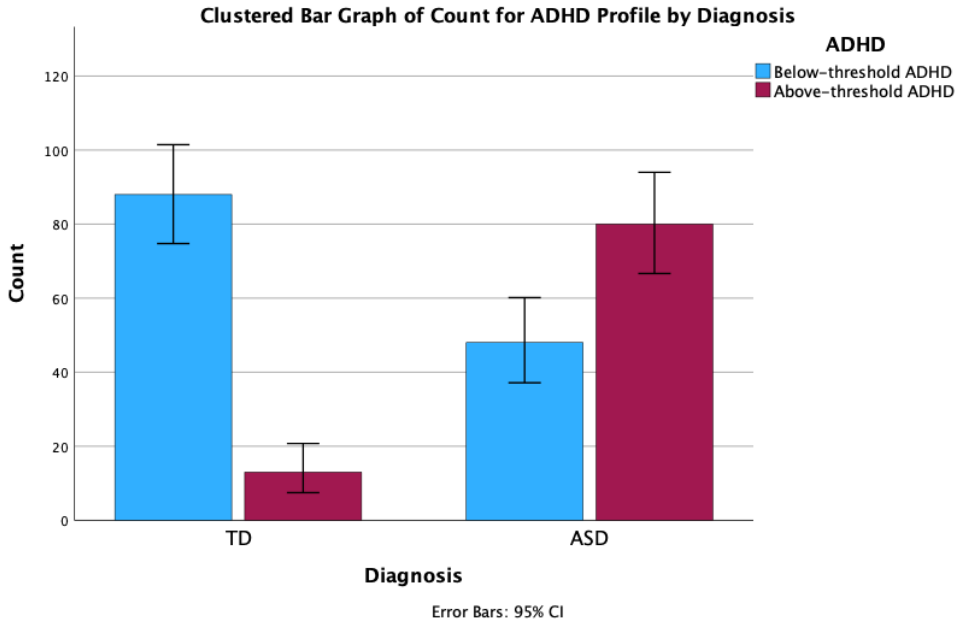
Table 4

Percentage Breakdown of Dependent Variables

Diagnosis		ADHD		Total
		t<65	t>65	
TD	Count	88	13	101
	% within diagnosis	87.10%	12.90%	100.00%
	% of Total	38.40%	5.70%	44.10%
ASD	Count	48	80	128
	% within diagnosis	37.50%	62.50%	100.00%
	% of Total	21.00%	34.90%	55.90%
Total	Count	136	93	229
	% within diagnosis	59.40%	40.60%	100.00%
	% of Total	59.40%	40.60%	100.00%

Figure 2

Percentage Participants Met Screener Criteria for ADHD by Diagnosis



Hypothesis 2.

A second question was to ask which predictors showed the most significant score difference among TD and ASD participants who met cut-off criteria for ADHD based on the C3-P(S). To better interpret the separate subscales of the C3-P(S), each category was designated as a predictor of the outcome, diagnosis, in a multiple regression analysis. **Table 6** shows there was an overall significant difference between predictor scores of TD and ASD groups who met cut-off criteria for ADHD ($R^2 = 0.601$, $F(6, 219) = 55.367$, $p < 0.001$). Taking a closer look at specific subscales in **Table 7**, there are a few predictors that are shown to contribute more to the overall difference between groups. The difference in IA scores of TD and ASD groups were not significant ($\beta = 0.029$, $t = 0.291$, $p = 0.771$). This was also mirrored in the EF and DA categories. However, ASD participants were found to score significantly higher in the HI subscale ($\beta = 0.152$, $t = 2.281$, $p = 0.024$). There was also a significant difference found in the LP subscale ($\beta = 0.172$, $t = 2.843$, $p = 0.005$). The PR predictor demonstrated the strongest effect as a predictor of diagnostic group ($\beta = 0.558$, $t = 10.955$, $p < 0.001$).

Table 6

Multivariate Regression for Dependent Variable (ADHD Criteria)

		R^2	df	F	Sig.
Diagnosis Predictor	**Regression**	0.603	6	55.367	<.001[b]
	Residual		219		
	Total		225		

Table 7

Coefficients Statistics for Predictors (Subscale Scores)

	Subscales	Standardized Coefficients Beta	t	Sig.
Predictors	Inattention	0.029	0.291	0.771
	Hyperactivity/Impulsivity	0.152	2.281	0.024
	Learning Problems	0.172	2.843	0.005
	Executive Function	0.006	0.077	0.939
	Defiance/Aggression	0.024	0.479	0.632
	Peer Relations	0.558	10.955	<.001

Discussion

Unique in its attempt to study specific diagnostic implications of the Conners 3rd Edition–Parent, Short form, this study aimed to investigate the predictability of comorbid symptomatology of ADHD in youth with ASD. The first hypothesis predicted significant differences between the subscale (IA, HI, LP, EF, DA, and PR) scores between the TD and ASD group. This hypothesis was supported, and significant differences in scores were found in each subcategory. There were also a higher percentage of ASD participants who met cut-off criteria for ADHD based on the C3-P(S), meaning that they scored above 65 in each subscale. However, there are implications to this conclusion, because the DSM-V has noted the similar symptomatology of the disorders; it is expected that there exist distinctive differences from TD peers (APA, 2013). It is, therefore, important to ask whether the percentage difference is practically significant and if it brings new understandings to existing literature. It is difficult to conclude based on this first research question whether the C3-P(S) is an effective measure to use

to predict comorbid symptomatology. As noted in an earlier study, the problems measured by each subscale remained consistent among ASD only, ADHD only, and ASD+ADHD groups (Ng et al., 2019). Future studies should cover a combination of diagnostic tools in addition to the C3-P(S) to create a more detailed understanding of predictability and diagnoses.

The second hypothesis investigated whether one or a few subscales showed the most significant elevated score difference between TD and ASD participants who met screener criteria for ADHD. From the results, the subscales of HI, LP, and PR were shown to be significantly higher in youth with ASD as compared to their TD peers. For HI, this conclusion deviated from that of previous literature. Ng et al. (20219) found that the scores remained consistent among ASD only and ADHD only children. This study, however, found that there was a significant deviation between TD and ASD participants who showed ADHD symptomatology based on the C3-P(S).

In the learning problems subcategory, an earlier study administered also found that ADHD worsened ASD symptomatology, which resulted in lower IQ and greater working memory deficits (Cooper et al. 2014). While the sample measured a population that slightly differs from that of the present investigation, the researchers' focus on comorbidity reinforces the elevated LP scores found in ASD participants as compared to their healthy controls.

Earlier literature has found discerning social functioning deficits associated with ASD versus ADHD especially difficult. This study, however, has shown that the C3-P(S) could be used as a screener for predicting which symptoms such as peer relations are elevated in ASD participants. Amongst the six subscales, PR showed the strongest difference in scores between the TD group and ASD group. This may suggest that an ASD diagnosis can magnify the dysfunction of social cognitive skills found in children. Waddington et al. (2018) found that

children with comorbid ASD+ADHD performed much worse in psychosocial measurement as compared to their healthy siblings. Emotional recognition deficits were the greatest causes of such dysfunction (Waddington et al., 2018).

There were no significant differences found in the inattention, executive function, and defiance/aggression subscales between TD and ASD children who met C3-P(S) ADHD cut-off criteria. The data differs from an earlier finding that found variability between children with ADHD and children with ASD+ADHD that accounted for the differences in social functioning (Ng et al., 2019). The C3-P(S) as a screening measure is less robust and unable to capture the subtleties of clinical presentation that other more intensive diagnostic interviews can and may not be able to parse through the nuances of this symptom affect. While both children with ASD and ADHD present with attention dysfunction, the mechanism by which it is expressed differs (Leitner, 2014). This difference in underlying attentional mechanisms may be attributed to the varied results.

The findings of this study also deviate from previous literature that found that working memory, a cognitive process under the EF umbrella, was significantly more impaired in individuals with both ASD+ADHD as compared to those diagnosed with ASD only (Colombi and Ghaziuddin, 2017). Though, it is important to note that this study was limited to participants only professionally diagnosed with ASD and typically developing children with reported ADHD symptoms based on the C3-P(S). The literature also noted that while slightly higher levels of behavioral control and anxiety were exhibited in the ASD+ADHD group, researchers were unable to identify a significant difference that accounted for the symptoms displayed (Colombi and Ghaziuddin, 2017). This reinforces the results established in the current investigation that

defiance/aggression scores do not vary significantly between TD and ASD participants who met threshold criteria for ADHD.

The implications of this study are important to consider knowing that one third of children with ASD potentially present with comorbid ADHD symptoms (Leitner, 2014). The additive issues that come from combined diagnoses can lead to adaptive problems and poorer quality of life that are not well understood due to the lack of an effective diagnostic tool to filter out this comorbidity (Leitner, 2014). Understanding first, as a caregiver and family member, that a child presents with both can help encourage more targeted treatments and intervention programs that improve outcomes.

The limitations of this study come from the lack of an official ADHD diagnosis group. It would be important and effective for future studies to include two additional sample groups: a diagnosed ADHD only group and a diagnosed ADHD+ASD group. This could provide insight on how ASD children with diagnosed ADHD compare with kids with ASD children with no diagnosed ADHD. If there is a difference between subscale scores then a differentiation between how comorbidity and the absence of it could be found. A question to consider and study in the future is: Are ASD and ASD+ADHD children performing differently on the C3-P(S) and other diagnostic/measurement tools?

Another limitation arises from the C3-P(S) itself. Although especially effective for its efficiency and understandable material, it is far from encompassing all the nuances and details of comorbid ASD and ADHD. As a “short form,” its main use is as a gateway to predicting how ADHD symptomatology exists in different participants. A more rigorous study design that includes the C3-P(S) along with a clinically administered interview of participants involved among other diagnostic tools is necessary.

Conclusion

Overall, the C3-P(S) subscale scores in all six categories of inattention, hyperactivity/impulsivity, learning problems, executive function, defiance/aggression, and peer relations were significantly different between the ASD and TD groups. In addition, a higher percentage of ASD youths met screener criteria for ADHD as compared to their TD peers. Among participants who met screener criteria for ADHD, ASD youths scored significantly higher than the TD group in the categories of inattention, learning problems, and peer relations. In summary, the C3-P(S) may play an effective initial role in identifying comorbid symptomatology of ADHD in children with ASD. Future directions should address pairing this screening tool with other more robust diagnostic measures as well as including official diagnosed ADHD and comorbid ASD+ADHD groups.

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