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Overlapping and Distinct Cognitive Impairments in Attention-Deficit/Hyperactivity and Autism Spectrum Disorder without Intellectual Disability

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Abstract

Attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are commonly comorbid, share genetic liability, and often exhibit overlapping cognitive impairments. Clarification of shared and distinct cognitive effects while considering comorbid symptoms across disorders has been lacking. In the current study, children ages 7–15 years assigned to three diagnostic groups:ADHD ($n = 509$), ASD ($n = 97$), and controls ($n = 301$) completed measures spanning the cognitive domains of attention/arousal, working memory, set-shifting, inhibition, and response variability. Specific processes contributing to response variability were examined using a drift diffusion model, which separately quantified drift rate (i.e., efficiency of information processing), boundary separation (i.e., speed-accuracy trade-offs), and non-decision time. Children with ADHD and ASD were impaired on attention/arousal, processing speed, working memory, and response inhibition, but did not differ from controls on measures of delayed reward discounting, set-shifting, or interference control. Overall, impairments in the ASD group were not attributable to ADHD symptoms using either continuous symptom measures or latent categorical grouping approaches. Similarly, impairments in the ADHD group were not attributable to ASD symptoms. When specific RT parameters were considered, children with ADHD and ASD shared impairments in drift rate. However, children with ASD were uniquely characterized by a wider boundary separation. Findings suggest a combination of overlapping and unique patterns of cognitive impairment for children with ASD as compared to those with ADHD, particularly when the processes underlying reaction time measures are considered separately.

Keywords Attention-deficit/hyperactivity disorder (ADHD) · Autism spectrum disorders (ASD) · Executive function · Neuropsychology · Reaction time variability · Diffusion model

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There is increasing recognition that many psychiatric symptom dimensions are shared across existing diagnostic boundaries, and the extent to which this indicates shared liability or etiology across disorders remains a major question (Insel et al. 2010; Sanislow et al. 2010). Despite conceptually distinct surface phenotypes, ADHD and ASD share overlapping features (Ronald et al. 2014)often enough to warrant clinical recognition in the DSM-5, co-occur at rates well above chance (Musser et al. 2014; Rommelse et al. 2011), and have overlapping genetic correlates (Rommelse et al. 2010; Ronald et al. 2008), suggesting they may be different manifestations of a shared liability. Crucially, for the focus of the current investigation, ADHD and ASD may also share some common cognitive impairments that can both confound and help clarify their clinical and phenomenological relationship.

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Executive Function in ASD and ADHD

Multiple cognitive domains have been proposed as relevant in ADHD and, to a lesser extent, in ASD, including: 1) response inhibition (Logan 1994; Nigg 2000); 2) interference control; 3) working memory (Baddeley 1996; De Luca et al. 2003; Engle 2002; Kane et al. 2007; Unsworth and Engle 2007), 4) set-shifting and 5) reward valuation, particularly delayed reward discounting (Demurie et al. 2012; Hulst et al. 2017; Mitchell 1999; Sagvolden et al. 1998).

Although there is heterogeneity in the ADHD population (Fair et al. 2012; Nigg et al. 2005), at the group level relatively consistent impairments have been found in individuals with ADHD in each of these domains (Huang-Pollock et al. 2012; Kofler et al. 2013; Willcutt et al. 2005). Deficits in several aspects of executive function have also been found in individuals with ASD relative to controls, particularly in set-shifting (Hill 2004; Ozonoff and Jensen 1999; Sanders et al. 2008).

Head-to-head comparisons of children with ASD and ADHD have yielded conflicting results as to whether executive function impairments and differences in reward processing are more pronounced in ADHD (Happe et al. 2006; Nyden et al. 2010) or in ASD (Geurts et al. 2004; Pennington and Ozonoff 1996). Individuals with ADHD may have greater difficulty with response inhibition and delayed reward discounting, whereas those with ASD may have more trouble with set-shifting and working memory (Corbett et al. 2009; Demurie et al. 2012; Gargaro et al. 2011; Happe et al. 2006; Hulst et al. 2017; Ozonoff and Jensen 1999; Sinzig et al. 2008). However, other studies also suggest that individuals with ADHD and ASD perform similarly (Geurts et al. 2004; Raymaekers et al. 2007; Semrud-Clikeman et al. 2010).

An obvious confound is that many children with ASD also experience clinically-significant ADHD symptoms, yet very few studies historically take this into account. One hypothesis is that the observed cognitive impairments in ASD may be related to comorbid ADHD symptoms rather than shared genetic liability or core mechanisms. Several studies have compared children with ASD and comorbid ADHD to those with ASD-only. However, this approach is limited because comparing categorical groups based on DSM diagnostic criteria does not account for sub-threshold but non-trivial ADHD symptoms that are often present in children with ASD. Some evidence using continuous symptom measures supports that impairments in response inhibition (Corbett et al. 2009; Yerys et al. 2013) and arousal (Corbett et al. 2009) in ASD may be driven by comorbid ADHD symptoms, whereas ASD symptoms may contribute unique variance to processing speed (Yerys et al. 2013). However, studies are small and have included tasks with emotional face stimuli (Yerys et al. 2013), which may be uniquely related to ASD traits in ways that non-emotional and non-face stimuli are not. Thus, it remains unclear whether the executive function problems attributed to ASD are truly due to shared liability or simply reflect comorbid ADHD symptoms.

Beyond Executive Function? Reaction Time Measures in ASD and ADHD

In addition to traditional measures of executive functioning, reaction time (RT) measures are of increasing interest in both ADHD and ASD as a window into the intersection of bottom-up information processing and cognitive control (Karalunas et al. 2014; Kofler et al. 2013; Rommelse et al. 2011; van der Meer et al. 2012). Increased RT variability in ADHD is well-established. In ASD, although RT variability has received theoretical attention as a putative trans-diagnostic phenotype (e.g., Rommelse et al. 2011), the empirical evidence for increased RT variability is less clear. A recent meta-analysis suggests that RT variablity in ASD may be driven by comorbid ADHD but highlights the need for additional studies, particularly those that account dimensionally for ADHD symptoms in the ASD group (Karalunas et al. 2014).

Another question is the extent to which RT impairments are driven by the same cognitive processes in both ADHD and ASD. RT speed and variability reflect the influence of multiple interacting processes (Ratcliff 2002) that traditional RT measures, such as mean and standard deviation, do not differentiate. The drift diffusion model provides one approach for separately quantifying the processes affecting the final RT and has been applied in the ADHD literature to clarify mechanisms of RT impairment. Briefly, the drift diffusion model (Ratcliff and Rouder 2016) assumes that how quickly a person responds is related both to the conservativeness of the criteria they have set for responding (called *boundary separation*, related to speed-accuracy trade-offs) and to the rate at which information is accumulated in favor of one of the response criterion (called *drift rate*). Processes not directly related to the response decision, such as stimulus encoding and motor response, are modeled by *non-decision time* (Ratcliff 2006).

In ADHD, RT impairments appear to be primarily driven by slow drift rates (i.e., slow, inefficient information processing) and to a lesser extent fast non-decision times, but are unrelated to differences in boundary separation (Huang-Pollock et al. 2012; Karalunas et al. 2014; Karalunas and Huang-Pollock 2013). The only study to-date applying this model in ASD found that children with ASD have wider boundary separations and slower non-decisions times than typically-developing children, but no differences in drift rates (Pirrone et al. 2017). Results suggest possibly unique mechanisms driving RT differences, but need to be confirmed in larger samples and direct comparisons between children with ASD and ADHD taking into account comorbid symptoms are needed.

Summary

The shared and unique cognitive characteristics of ADHD and ASD remain insufficiently characterized. Here, we examine overlapping and distinct neuropsychological profiles of children with ADHD and ASD and evaluate the effects of dimensional symptom measures on neuropsychological profiles for both groups. Based on the previous literature, we hypothesized that the ASD group would show greater impairment than the ADHD group on set-shifting and working memory, but would have similar or less impairment than children with ADHD on measures of arousal, inhibitory control, and reward discounting. We did not expect ASD symptoms to account for cognitive impairments in ADHD, but sought to clarify whether ADHD symptoms accounted for cognitive impairments in ASD. We further hypothesized that the ADHD and ASD groups would be equally impaired on measures of response variability but that these differences would be driven by different mechanisms: slow drift rate in ADHD and wide boundary separations in ASD.

Method

Overview of Procedure

All procedures were approved by the Oregon Health & Science University's Institutional Review Board. Parents provided written informed consent and children provided written assent. Participants were ages 7–15 years assigned to three diagnostic groups using screening and diagnostic procedures described below. The final sample included 509 children ADHD, 97 children with ASD, and 301 typically-developing controls. Table 1 shows participant demographics. Study data were collected and managed, in part, using RED Cap electronic data capture tools hosted at OHSU. It provides a secure web-based and intuitive interface and export capabilities (Harris et al. 2009).

Participant Recruitment: ADHD and Typically-Developing Controls ADHD and control children in the current study were part of an ongoing longitudinal study. Only the data from the first year of the study are included here to match available data for the ASD sample. Participants were recruited via mailings to commercial mailing lists and public advertisements. Families participated in a multi-gated procedure that included an initial phone screen. Parents of children who remained eligible upon completion of the initial phone screen were invited to complete the ADHD Rating Scale (DuPaul et al. 1998), Conner's Rating Scale, 3rd edition (Conners 2008), and the Strengths and Difficulties Questionnaire (Goodman 2001), and an in-person semi-structured diagnostic interview (Kiddie Schedule for Affective Disorders and Schizophrenia,

KSADS; Puig-Antich and Ryan 1986), while the child completed IQ screening and brief academic achievement testing.

A best estimate DSM-IV diagnosis was established by a multi-disciplinary diagnostic team. Blind to one another's ratings and to the subsequent cognitive test scores, they formed a diagnostic opinion based on all available information. Their agreement rate was excellent (ADHD diagnosis kappa = .88). Disagreements were conferenced and consensus reached. Cases where consensus was not readily achieved were excluded. Specific symptom counts for determining diagnosis followed DSM-IV cutoffs and are detailed in Supplemental materials.

Participant Recruitment: ASD ASD youth were recruited in a more targeted fashion. Children who met criteria for DSM-IV diagnosis of either Autistic Disorder, Asperger's Disorder, or Pervasive Developmental Disorder Not Otherwise Specified were recruited through the University's Autism Clinic in the Child Development and Rehabilitation Center, community support groups and outreach, and targeted mailings based on charted ICD-9 codes. After passing initial screening criteria, participants in the ASD group were administered the Autism Diagnostic Observation Schedule (Lord et al. 2001) and parents completed the Autism Diagnostic Interview–Revised (Rutter et al. 2003). Parents also completed the Social Responsiveness Scale, 2nd edition (SRS-2, Constantino and Gruber 2012), Children's Communication Checklist (Bishop 2003), and a detailed developmental history questionnaire. Parents had the option to provide any existing documentation regarding a previous ASD diagnosis including IEP's, chart records, and psychological evaluations. A multi-disciplinary diagnostic team that included three licensed clinicians then utilized all of the aforementioned materials to determine a consensus diagnosis.

Exclusionary Criteria Children in all three diagnostic groups were excluded if they: were prescribed long-acting psychotropic medications; had neurological impairment, seizure history, head injury with loss of consciousness, other major medical conditions, or substance abuse; had a prior diagnosis of intellectual disability, or psychosis; were currently experiencing a major depressive episode; or had estimated IQ <70. Other comorbidities were assessed by the multi-disciplinary teams, but were not exclusionary in any of the diagnostic groups except that children in the ADHD and Control samples were excluded if they had a parent-reported history of ASD diagnosis or if the multi-disciplinary team identified a diagnosis of ASD.

Stimulant Medication Children with ASD or ADHD taking stimulant medications were included in the study but were required to be off medication for 24 (for short-acting preparations) to 48 h (for long-acting preparations) prior to testing.

Table 1 Participant characteristics

	Control	ASD	ADHD	p	Effect Size (d)[a]	Posthoc
N	301	97	509	–	–	
Sex (Male:Female)	146:155	84:13	359:150	< 0.001	–	ADHD > C; ASD > C; ASD > ADHD
Age in years (SD)	9.48 (1.59)	11.34 (2.49)	9.72 (1.59)	< 0.001	0.89, 0.15, 0.78	ASD > C; ASD > ADHD
Estimated IQ (SD)	115.28 (12.75)	99.53 (16.37)	108.27 (13.88)	< 0.001	1.07, .52, 0.58	ADHD < C; ASD < C; ASD < ADHD
ADHD-RS IA (SD)	44.73 (7.32)	63.54 (13.20)	72.17 (12.07)	< 0.001	1.76, 2.75, 0.68	ADHD > C; ASD > C; ASD < ADHD
ADHD-RS HI (SD)	45.11 (7.20)	60.07 (12.77)	67.59 (14.51)	< 0.001	1.44, 1.96, 0.55	ADHD > C; ASD > C; ASD < ADHD
ADHD-RS Total (SD)	44.61 (7.18)	62.82 (12.37)	71.48 (12.61)	< 0.001	1.81, 2.62, 0.69	ADHD > C; ASD > C; ASD < ADHD
SRS-2 Total (SD)+	45.54 (6.46)	77.93 (11.38)	55.38 (9.51)	< 0.001	3.50, 1.21, 2.15	ASD > ADHD > C

p-values indicate three-group significance test

ASD Autism spectrum disorder; ADHD Attention-deficit/hyperactivity disorder; IA Inattention; HI Hyperactive-Impulsive

ADHD-RS and SRS-2 scores reported are age- and sex-normed T-scores

[a] These values reflect the Cohen's d value for the Control/ASD, Control/ADHD, and ASD/ADHD comparison, respectively. Effects sizes presented are the absolute value of the Cohen's d value

Measures of Cognitive Function

A description of each cognitive task is below. Additional details of tasks, data cleaning and validity criteria, and rates of missing data are detailed in the Supplemental materials.

Working Memory Children completed a computerized version of a spatial working memory task identical to the spatial span task from the CANTAB (De Luca et al. 2003). Forward and backwards conditions were both administered. Children also completed the familiar Digit Span task from the WISC-IV, including both forward and backward conditions.

Set-Shifting The Trail making Test (Delis et al. 2001) from the Delis-Kaplan Executive Function System (D-KEFS) was used as a measure of set-shifting. The final dependent variable was the residual of Condition 4 (switching) score after partialing out performance on the number- (Condition 2) and letter- (Condition 3) sequencing conditions.

Interference Control Condition 3 of the Color-Word test (Delis et al. 2001) from the D-KEFS was used as a measure of interference control. The final dependent variable was the residual Condition 3 (inhibition) score after partialing out performance on the color naming (Condition 1) and word reading (Condition 2) control conditions.

Attention/Arousal A computerized Identical Pairs CPT (CPT-IP), similar to the A-X CPT design used by Nigg et al. (1996) was used to assess attention/arousal. Children viewed a series of four-digit numbers displayed one at a time in pseudo-random order for 200 ms followed by a 1500 ms blank screen. When two identical numbers appear back-to-back, the child pushed the response button. Target frequency was 20%, with an additional 20% "catch" trials (only one digit off— "hard" discriminations), and 60% "stim" or "non-target" trials (more than one digit different—"easy" discriminations). The task included a total of 300 stimuli and required about 10 min to complete. The primary

outcome measures were signal detection accuracy (d-prime [d']) on "catch" and "stim" trials.

Processing Speed The control conditions from the DKEFS (Delis et al. 2001) Trail making (Conditions 2 and 3) and Color Word (Conditions 1 and 2) tests were used as measures of processing speed.

Response Inhibition Response inhibition was evaluated based on a tracking version of the stop task (Logan 1994; Nigg 1999). On 75% of trials ("go" trials), children were asked to indicate with a key press whether an "X" or an "O" appeared in the center of the screen. On 25% of trials ("stop" trials), an auditory tone presented after the stimulus indicated that the child should not respond. After 32 practice trials, children completed 8 blocks of 64 trials.

Response Time and Response Variability Mean and standard deviation of RT on correct go trials from the stop task were calculated for each child. In addition, diffusion model parameters were estimated from the trial-by-trial RT time series using the Fast-dm modeling technique and the downloadable program from the author's website (Voss and Voss 2007), and as previously published (Huang-Pollock et al. 2012; Karalunas and Huang-Pollock 2013). Smaller absolute values of drift rate indicate slower/less efficient information processing. Smaller values of boundary separation indicate greater speed-accuracy trade-offs, and smaller non-decision times indicate faster non-decision time.

Delayed Reward Valuation Delayed reward valuation was assessed using a computerized delay discounting questionnaire, based on the task described by Mitchell (1999) and Wilson et al. (2011). Children were presented a series of 91 questions in which they chose between (1) a varying amount of hypothetical money now ($0–$10.50 in $0.50 increments) and (2) a hypothetical $10.00 after a varying delay (7, 30, 90, or 180 days; e.g., $6.50 now or $10 in 30 days). Specific answers had no effect on the overall duration of the task; however, children's response time affected task length because the next question was not presented until the previous question was answered. The task took approximately 12 min. Valuation of delayed rewards was quantified as the discounting gradient (k) because prior publication indicated this best differentiates ADHD and control performance (Mitchell et al. 2015). Values were natural-log transformed to address non-normality.

Data Analytic Plan

All analyses were conducted in M plus v. 7.4 using the robust maximum likelihood estimator and full-information maximum likelihood methods to handle missing data. The nesting of children within families was handled using Mplus' cluster command.

Data Reduction In order to take full advantage of the multiple assessments of cognitive processes, confirmatory factor analysis (CFA, 1000 random starts and 50 integration points) was applied to create latent variables for: 1) *working memory* indicated by Spatial Span backward and forward and digit span forward and backward; 2) an *attention/arousal* indicated by d' on "catch" and "stim trials" for the CPT and the drift rate RT parameter; and 3) *processing speed* indicated by scores on Condition 1 and Condition 2 of Color Word and Condition 2 and Condition 3 of Trails. The measurement model fit the data well, χ^2 (39) = 164.70, p = .00, CFI = .96, TLI = .95, RMSEA = .06. Latent variables were used in between-group comparisons along with single indicators for response inhibition, interference control, switching, response variability, and delayed reward discounting for which there were not the multiple measures necessary to create latent variables. Single indicators for each diffusion model parameter were also compared.

Role of ADHD and ASD Symptoms in EF Impairment To assess the role of ADHD symptoms in ASD cognitive impairment and ASD features in ADHD cognitive impairment, two approaches were used: 1) covarying continuous dimensions and 2) comparing empirically-derived groups based on parent ratings. For our analyses using continuous dimensions, we first compared all diagnostic groups to each other. We then repeated the ASD-control group comparisons covarying: a) inattention and b) hyperactivity-impulsivity using T-scores from parent report on the well-validated ADHD Rating Scale (DuPaul et al. 1998). Because the ADHD group is necessarily defined by their ADHD symptoms, analyses covarying ADHD symptoms included only the ASD and typically-developing control participants.

Continuous ASD features (we avoid using the word symptoms because the SRS-2 is not a symptom checklist) based on parent ratings on the SRS-2 were available for a subset of ADHD and control participants in from the full sample (ADHD = 177, Control = 137). Children with and without SRS-2 did not differ in age, IQ, ADHD symptom severity, or sex distribution (all p > .10). Using this subsample, we repeated the ADHD-control group comparisons covarying the total T-score from the SRS-2 (Constantino and Gruber 2012). Because the ASD group is necessarily defined by their ASD features, analyses covarying ASD features included only the ADHD and typically-developing control participants.

For our analyses comparing empirically-derived groups based on parent ratings, we next conducted a latent profile analysis in the ASD sample using the age- and gender normed T-scores from the ADHD-RS (inattention, hyperactivity-impulsivity, total) to determine whether there were distinct ASD subgroups characterized by varying levels of ADHD symptoms. We repeated between-group comparisons using these empirically-derived groups. A latent profile analysis

Table 2 Neuropsychological tasks by diagnostic group

Cognitive Measure	Control	ASD	ADHD	Posthoc covarying Age	Effect Size (β)[a]	Posthoc covarying Age &IQ
Delay Discounting	−3.90 (2.08)	−4.15 (2.80)	−3.64 (2.61)	–	0.01, 0.07, 0.02	–
Arousal	0.19 (0.49)	0.19 (0.52)	−0.14 (0.47)	ASD = ADHD > C	0.22, 0.37, 0.11	ASD > ADHD > C
Speed	−1.47 (5.22)	1.80 (9.81)	0.54 (6.88)	ASD > ADHD > C	0.46, 0.24, 0.28	ASD > ADHD > C
Working Memory	.44 (1.10)	−0.20 (1.38)	−0.22 (1.10)	ASD < ADHD < C	0.48, 0.40, 0.18	ASD = ADHD < C
Switching	126.35 (51.80)	139.03 (64.38)	145.18 (60.43)	ADHD > C	0.18, 0.00, 0.13	ADHD > C
Interference Control	85.30 (24.68)	91.04 (50.81)	99.49 (31.76)	ADHD > C	0.14, 0.10, 0.15	ADHD > C
Response Inhibition	248.55 (78.84)	302.52 (122.26)	291.56 (115.06)	ASD = ADHD > C	0.33, 0.21, 0.10	ASD = ADHD > C
RT Variability	214.22 (72.05)	249.68 (106.21)	259.97 (92.86)	ASD > ADHD > C	0.36, 0.28, 0.13	ASD > ADHD > C
Drift Rate	3.15 (0.73)	3.00 (0.89)	2.72 (0.76)	ASD = ADHD < C	0.29, 0.30, 0.04	ASD = ADHD < C
Boundary Separation	1.55 (0.33)	1.71 (0.34)	1.56 (0.30)	ASD > C = ADHD	0.21, 0.00, 0.18	ASD > ADHD = C
Non-decision Time	0.47 (0.13)	0.45 (0.15)	0.44 (0.11)	ADHD < C	0.07, 0.12, 0.02	ADHD < C

ASD Autism spectrum disorder; ADHD Attention-deficit/hyperactivity disorder

Raw means for each group are reported. Post-hoc and effect sizes reflect findings of the final model including covariates

[a] These values reflect the absolute value of the standardized regression coefficient for the Control/ASD, Control/ADHD, and ASD/ADHD comparison, respectively

was also applied in the ADHD sample using T-scores from all subscales on the SRS-2 and between-group comparisons repeated using these empirically defined ADHD groups.

Covariates Age was used as a covariate in all between-groups analyses. Groups also differed in IQ, however, covarying for characteristics (such as IQ) that differ between groups but are representative of the underlying populations can violate statistical assumptions and result in comparison at levels of the covariate that do not exist in the actual population (Dennis et al. 2009). For this reason, we report analyses without IQ as a covariate. For completeness, Table 2 shows changes in patterns of significance when IQ is covaried, which are minimal.

Multiple Comparisons A false discovery rate correction for multiple comparisons was applied (Benjamini and Hochberg 1995). Uncorrected p-values are reported in the text for ease of comparison with other studies; however, a corrected p-value threshold of $p = 0.042$ was used for interpreting all results (McDonald 2014).

Results

Means and SDs of cognitive measures are presented in Table 2 for diagnostic groups and in Table S2 for the empirically-derived subgroups. The 95% confidence intervals for the between-group effect sizes, which are generally in the small to medium range, are provided in Table S3 and S4. Neuropsychological profiles are depicted in Figs. 1, 2 and 3.

Executive Functioning

Continuous Symptoms As expected, children with ADHD performed more poorly than typically-developing controls on all executive function measures (all $p < 0.001$). However, there were no reliable differences in delayed reward discounting ($p = 0.067$). The pattern of results remained the same when controlling for ASD features as measured on the SRS-2, suggesting ASD features did not account for cognitive impairments in the ADHD group.

When comparing children with ASD to typically-developing children, the ASD group had lower arousal, slower processing speed, lower working memory, and worse response inhibition (all $p < 0.001$). There were no reliable group differences on tests of interference control, switching, or delayed reward discounting (all $p > .156$). The patterns of significance all remained the same when controlling for inattention and hyperactivity-impulsivity symptoms, suggesting ADHD symptoms did not account for cognitive impairments in the ASD group.

When directly comparing children with ASD and ADHD, children with ASD had slower speed and worse working memory than children with ADHD (all $p < 0.001$). There were no reliable difference between children with ADHD and ASD on measures of arousal, response inhibition, interference control, or switching (all $p > 0.05$).

Empirically-Defined Groups Children with ASD were split into empirically-defined groups based on their ADHD symptoms. The best-fitting LPA model[1] indicated two ASD groups: one

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[1] AIC = 2000.6, Sample Size Adjusted BIC = 1994.0, Vuong-Lo-Mendell-Rubin Likelihood Ratio Test for 1 versus 2 classes p < .001; Vuong-Lo-Mendell-Rubin Likelihood Ratio Test for 2 versus 3 classes p = .199

Fig. 1 Cognitive profiles for ADHD, control, ASD + ADHD, ASD-ADHD groups. Z-scores are plotted so that all variables are represented on the same scale. More negative scores indicate worse performance for all measures
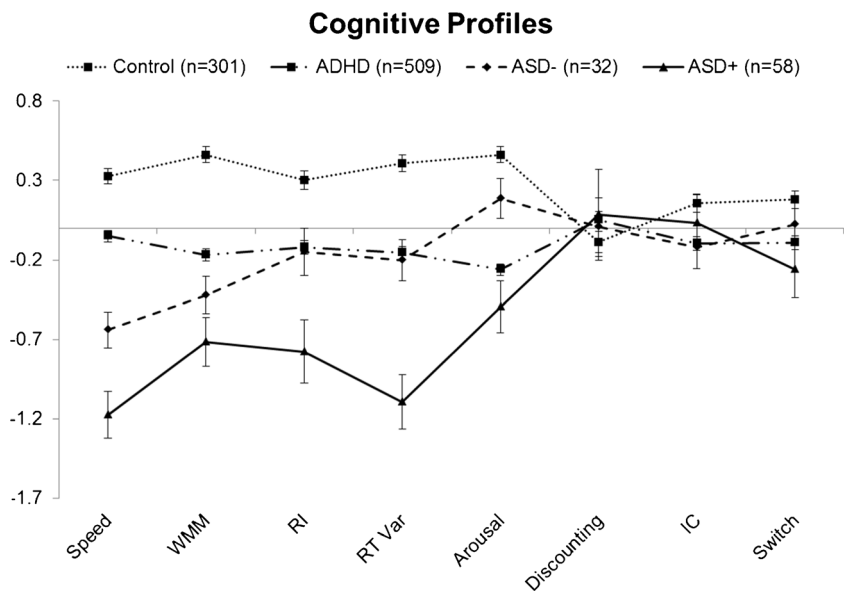
with ADHD symptoms in the non-clinical range similar to the typically-developing controls and a second group with both inattention and hyperactivity-impulsivity scores in the clinical range similar to the ADHD sample (see Table S1). Groups were heuristically labeled ASD-ADHD and ASD + ADHD.

When comparing executive function profiles using these empirically-defined ASD groups, the pattern of results was similar to analyses using continuous symptoms. Both the ASD-ADHD and ASD + ADHD groups had slower processing speed, worse working memory, and worse response inhibition than typically-developing controls (all $p < 0.01$). Although both ASD groups were impaired relative to typically-developing controls, children in the ASD + ADHD group were more impaired on measures of processing speed and response inhibition (all $p < 0.02$) than their ASD-ADHD counterparts. The two ASD subgroups did not differ in degree of working memory impairment ($p = .12$).

When compared to children with ADHD-only, both ASD groups had slower processing speed than the ADHD group ($p < 0.001$). The ASD + ADHD group also had lower working memory ($p < 0.001$) and worse response inhibition ($p = 0.01$) than children with ADHD-only, while the ASD-ADHD group performed similarly to children with ADHD-only on these measures (all $p > .12$). Overall, results indicate the cognitive impairments in ASD are not attributable to comorbid ADHD symptoms.

In contrast to results using continuous symptom dimensions, which suggested that arousal impairments in ASD were unrelated to ADHD symptoms, results from the categorical grouping approach suggested that arousal impairments in ASD were dependent on ADHD symptom profile. Results indicated that the ASD + ADHD and ASD-ADHD groups significantly differed from each other in arousal ($p < 0.001$). The ASD +

ADHD group performed similarly to children with ADHD on arousal measures ($p = .14$) and were significantly worse than controls ($p < 0.001$). In contrast, the ASD-ADHD performed similarly to typically-developing children ($p = .33$), and had significantly higher arousal than children with ADHD ($p < 0.001$).

The two ASD subgroups did not differ from a) children with ADHD, b) controls, or c) each other on delay reward discounting, interference control, or set-shifting ($p > .12$).

Children with ADHD were also split into empirically-defined groups based on their ASD features. The best-fitting LPA model[2] indicated two ADHD groups: one with ASD symptoms in the non-clinical range similar to the typically-developing controls and a second group with elevated SRS-2 scores across all domains. Groups were heuristically labeled ADHD-ASD and ADHD + ASD, although we note that SRS scores in the ADHD + ASD group were significantly lower than in the ASD group, consistent with our exclusion of children with an ASD diagnosis (see Table S1). The ADHD subgroups did not reliably differ from each other on any of the cognitive measures (all $p > 0.075$).

Reaction Time Parameters

Continuous Symptoms As expected, children with ADHD exhibited more reaction time variability than their typically-developing peers ($p = 0.007$). When parameters accounting for RT performance were considered separately, children with ADHD had slower drift rates and faster non-decision times

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[2] AIC = 7794.79, Sample Size Adjusted BIC = 7856.28, Vuong-Lo-Mendell-Rubin Likelihood Ratio Test for 1 versus 2 classes $p = .047$; Vuong-Lo-Mendell-Rubin Likelihood Ratio Test for 2 versus 3 classes $p = .521$

Fig. 2 Reaction time parameter profiles in the ADHD, control, ASD + ADHD, ASD-ADHD groups. Z-scores are plotted so that all variables are represented on the same scale. More negative scores indicate factors contributing to slower performance (i.e., less efficient information processing, wider boundary separation, and slower non-decision times)
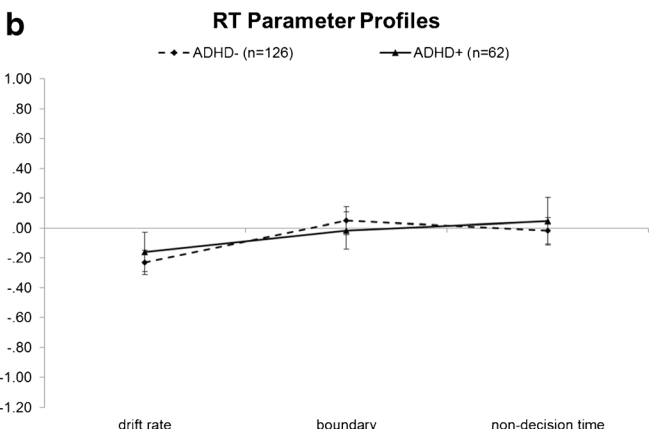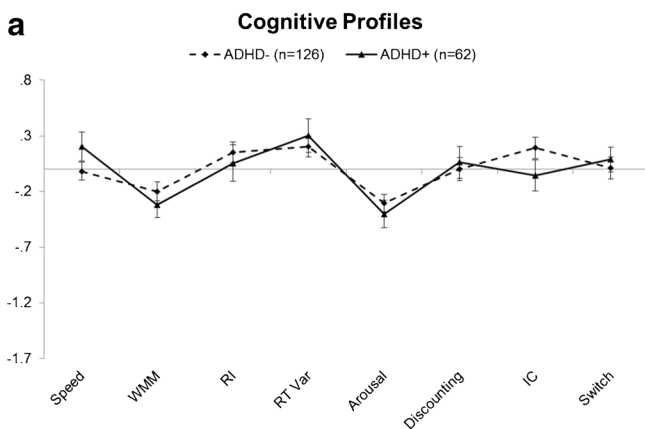
RT Parameter Profiles
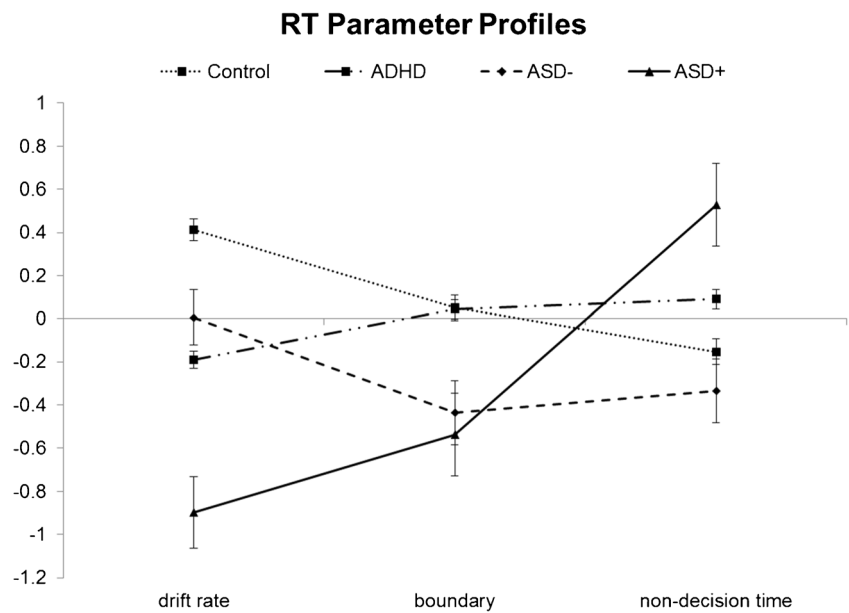
than typically-developing children (all $p < 0.001$), but groups did not reliably differ in boundary separation ($p = .950$). The patterns of significance all remained the same when controlling for ASD features.

Children with ASD also exhibited more RT variability than typically-developing controls. The ASD group had slower drift rates than typically-developing controls ($p < 0.001$), similar to impairments observed in ADHD. However, children with ASD also had wider boundary separation ($p < 0.001$) and did not reliably differ from typically-developing controls in non-decision time ($p = 0.275$). The patterns of significance for ASD-control comparisons remained the same when controlling for inattention and hyperactivity-impulsivity symptoms.

When directly compared to children with ADHD, children with ASD had wider boundary separations ($p < 0.001$), but did not reliably differ in either drift rates or non-decision times.

Empirically-Defined Groups When children with ASD were split into empirically-defined groups based on their ADHD symptoms, the pattern of results was similar. Children in the ASD-ADHD and ASD + ADHD groups both exhibited greater reaction time variability than typically-developing controls. When RT parameters were considered separately, both ASD groups had slower drift rates (all $p < 0.011$) and wider boundary separations (all $p < 0.004$) than typically-developing controls. The ASD + ADHD group was more impaired than their ASD-ADHD counterparts on measures of overall RT variability and drift rate (all $p < 0.001$), but the two ASD groups did not reliably differ from each other in boundary separation ($p = 0.75$).

When compared to children with ADHD-only, the ASD + ADHD group was more variable overall and had slower drift rates (all $p < 0.001$), while the ASD-ADHD group did not

a **Cognitive Profiles**

b **RT Parameter Profiles**

Fig. 3 a) Cognitive and **b**) Reaction time parameters in the ADHD + ASD and ADHD-ASD groups. Z-scores are plotted so that all variables are represented on the same scale. More negative scores indicate worse performance on cognitive profile measures and factors contributing to slower performance for RTs (i.e., less efficient information processing, wider boundary separation, and slower non-decision times). The scales are intentionally kept the same as in Figs. 1 and 2 to facilitate visualization of group differences

reliability differ from the ADHD-only group (all $p < 0.15$). The ASD + ADHD group also had faster non-decision time than typically-developing controls, children with ADHD, or their ASD-ADHD counterparts (all $p < 0.03$), while the ASD-ADHD group had non-decision times similar to typically-developing controls ($p = 0.338$). Wider boundary separation again appeared to be specifically related to ASD. The ASD + ADHD and ASD-ADHD groups both had wider boundary separations than typically-developing controls or children with ADHD (all $p < 0.005$).

When children with ADHD were split into empirically-defined groups based on their ASD symptoms, the ADHD subgroups did not reliably differ from each other on any of the reaction time measures (all $p > .402$).

Discussion

The current study examined shared and distinct cognitive impairments in children with ADHD and ASD using both continuous symptom measures and empirically-defined categorical groups. Several cognitive impairments in ASD, including in processing speed, working memory, and response inhibition, were independent of ADHD symptoms. Similarly, impairments in ADHD were independent of ASD symptoms. Results support a model in which impairments in these areas are shared across disorders and are not attributable to comorbid symptom profiles.

Equally important, however, when specific measures contributing to RT performance were considered, children with ADHD and ASD had partially distinct profiles. Slow drift rates occurred in both clinical groups, suggesting that this is a shared liability marker similar to many of the executive functioning measures. In contrast, wider boundary separations, which are related to emphasizing accuracy over speed, were specific to the ASD group. Children with ASD who had elevated ADHD symptoms and those with normative ADHD symptom levels both had equally wide boundary separations. This is the first study to directly compare children with ASD and ADHD on these differentiated RT parameters. Wider boundary separation is not associated with ADHD (Karalunas et al. 2014; van Mourik et al. 2005), but has been found in ASD (as compared to typical development) in the only other study applying a diffusion model approach in this population (Pirrone et al. 2017). Pirrone et al., did not find slower drift rates in ASD, as we did here, but the sample included only a small sample of adults and used a perceptual discrimination task, either of which may account for differences in results. Overall, results confirm some specificity in features contributing to RT differences in ASD and ADHD. Put another way, although both groups are characterized by slower and more variable RTs on fast tasks, this is for partly distinct reasons. Wide boundary separation may be a specific feature of the ASD cognitive profile that could be used to differentiate diagnostic groups.

How might the present findings inform our conceptualization of these two disorders? ASD and ADHD are sometimes hypothesized to be different manifestations of a shared liability (van der Meer et al. 2012), and cognitive impairments have been proposed as indicators of this shared risk. The current results are consistent with the proposition that many cognitive deficits in ASD and ADHD reflect shared liability and are not simply accounted for by comorbid symptoms. Impairments in working memory, response inhibition, and speed of processing remained present in ASD even after accounting for continuous measures of ADHD symptoms. They occurred in children with ASD who had elevated ADHD symptoms, as well as in children with normative ADHD symptom levels. Although prior research and theory has focused on the role of ADHD symptoms in driving ASD cognitive impairments, we also confirm that comorbid ASD symptoms do not account for these cognitive performance deficits in ADHD. This further strengthens the argument that these impairments reflect shared liability and not comorbid symptom profiles. However, our results suggest important amendments to the shared liability proposal for RT measures (Rommelse et al. 2011). At least some RT differences in ASD are driven by distinct processes from those which drive the similar surface RT findings in ADHD.

Prior studies using continuous symptoms dimensions have similarly found that ADHD symptoms cannot explain ASD-related impairments in working memory (Corbett et al. 2009) and that there may be distinct contributions of ASD symptoms to RT measures (Yerys et al. 2013). However, current results are partially in contrast to findings that continuous ADHD symptoms predict response inhibition in both ASD and ADHD (Corbett et al. 2009; Yerys et al. 2013). Differences may be due to small samples (n~20/group) in prior studies, use of different stimuli, or differences in statistical approach. At least one prior study comparing children with ADHD and ASD has identified ADHD-specific differences in brain connectivity in regions often associated with response inhibition (Di Martino et al. 2013). This finding would not be predicted based on the shared cognitive impairment seen here; however, cognitive performance was not measured or related brain connectivity in the prior study and brain connectivity was not considered here. Future work combining these approaches will be important.

Two cognitive markers may also be specifically associated with ADHD symptoms but not ASD. Faster non-decision times, possibly related to a type of motor impulsivity, and low arousal occurred only in children with high levels of ADHD symptoms, but not in typically-developing controls or those with ASD but without ADHD symptoms. These impairments were unrelated to ASD symptoms in the ADHD group. Here, results were less clear cut, however, in that

differences were only found using categorical grouping approaches. Corbett et al. (2009), albeit in an small exploratory analysis, similarly found that the relationship of arousal measures to ADHD symptoms depended on whether categorical or continuous symptom measures were used. Additional studies using both categorical and continuous approaches are needed to clarify the specificity of these impairments to ADHD versus ASD. It may be that these effects are more dependent than other domains on how symptoms are viewed.

Clinically, recent review of the National Survey of Children's Health data found that ASD diagnosis is often delayed when ADHD symptoms are also present, and these delays are independent of ASD severity (Miodovnik et al. 2015). Thus, when comorbid ADHD symptoms are present, children with ASD may miss early intervention opportunities because of diagnostic uncertainty. Unique RT markers for ASD may offer one way to facilitate improved diagnosis for children with comorbid symptoms.

It is also clinically relevant, however, that effects for cognitive measures were generally in the small to medium range, suggesting substantial overlap of diagnostic groups with typical development. ADHD and ASD are both heterogeneous, and small to medium effects are consistent with prior research suggesting that only a subset of children with these disorders experience cognitive impairments. Future studies with sample sizes adequate to consider within-group heterogeneity in cognitive functioning in children with ASD will be important for determining how much of the ASD population these findings representor whether they may represent definable subgroups, such as we have suggested for ADHD (Fair et al. 2012; Nigg et al. 2005).

Several other caveats are also notable. Severity effects were observed for many measures. Even in cases where ADHD-only, ASD-ADHD, and ASD + ADHD were all impaired relative to typically-developing controls, the ASD + ADHD group was often the most impaired. This likely reflects the greater number of overall symptoms in this group, rather than a unique or distinct mechanism. Similar severity effects are often observed within ADHD, where greater symptom severity is associated with more cognitive impairment.

Cognitive abilities develop rapidly over middle childhood and adolescence(Best and Miller 2010; Karalunas et al. 2017; Miller et al. 2013) and whether the ASD and ADHD groups show distinct trajectories of cognitive development is unable to be evaluated in this cross-sectional study. Additionally, although our overall sample size was sufficient to handle the missing data observed, missing data was non-trivial in the ASD group. Children with ASD who completed all tasks were older than the children who did not, and so additional studies are needed to confirm that results are similar in younger samples as well.

Although there was variation within the sample, at the group-level the children with ASD included in the study had average IQ scores. This could have resulted in underestimation of cognitive effects and limits generalizability of results to children with ASD and intellectual disability. Exclusion of children on non-stimulant medications may also reduce generalizability of results, particularly for the ASD sample because of possible limited efficacy and potential for greater side effects in this diagnostic group (Davis and Kollins 2012).

Although children with ADHD were impaired on measures of interference control and switching, children with ASD were not, which stands in contrast to prior work suggesting that switching, in particular, may be a primary cognitive impairment in ASD. This may be related to the ASD sample being relatively high functioning, although this did not preclude finding impairments on many other cognitive measures. Alternatively, our approach of partialing out basic speed to isolate interference control and switching may account for this difference.

In addition, none of the groups here differed on measures of delayed reward discounting. Recent work from our group found that ADHD-related differences in delayed reward discounting on this task may be largest in early childhood, partially normalizing at the group-level by early adolescence (Karalunas et al. 2017). Our ASD sample was older than the other groups, which may have contributed to the lack of positive findings here. Alternatively, use of a task with real (rather than hypothetical) rewards may have better elicited group differences. These are both directions for future research.

Summary

Overall, current results indicate that cognitive impairments in working memory, response inhibition, and processing speed, as well as slow drift rates, may reflect shared liability in ADHD and ASD. In contrast, a conservative response style may be a unique marker of ASD pathology that helps partially differentiate the two disorders at the level of cognitive endophenotypes.

Compliance with Ethical Standards

Conflict of Interest   The authors have no conflicts of interest or financial disclosures to report.

Ethical Approval   All procedures were approved by the Oregon Health & Science University's Institutional Review Board.

Informed Consent   Parents provided written informed consent and children provided written assent.

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