

Relationship Between Executive Functioning and Symptoms of Attention-Deficit/Hyperactivity Disorder and Autism Spectrum Disorder in 6–8 Year Old Children

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Abstract This study examined relationships between executive functioning (EF) and ADHD/ASD symptoms in 339 6–8 year-old children to characterise EF profiles associated with ADHD and ADHD + ASD. ADHD status was assessed using screening surveys and diagnostic interviews. ASD symptoms were measured using the Social Communication Questionnaire, and children completed assessments of EF. We found the EF profile of children with ADHD + ASD did not differ from ADHD-alone and that lower-order cognitive skills contributed significantly to EF. Dimensionally, ASD and inattention symptoms were differentially associated with EF, whereas hyperactivity symptoms were unrelated to EF. Differences between categorical and dimensional findings suggest it is important to use both diagnostic and symptom based approaches in clinical settings when assessing these children's functional abilities.

Keywords Attention-deficit/hyperactivity disorder · Autism spectrum disorder · Comorbidity · Child · Executive function

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is characterised by symptoms of inattention, hyperactivity and impulsivity. As many as 22–50 % of children with ADHD display clinically elevated symptoms of Autism Spectrum Disorder (ASD) (Kochhar et al. 2011; Reiersen et al. 2007; van der Meer et al. 2012). It has been postulated that children with ADHD and ASD share similar deficits in executive functioning (EF) (Bradshaw and Sheppard 2000; Pennington and Ozonoff 1996). A recent review suggests that children with ADHD may be differentiated from those with ASD by specific EF profiles, and that the hallmark of a comorbid disorder may be a combination of these profiles (Gargaro et al. 2011). Findings have been mixed regarding the EF deficits associated with ASD and ADHD, and only a small body of research has examined EF in children with comorbid ADHD/ASD in comparison to groups of children with ADHD or ASD alone. Thus, little is known about the interplay between EF and symptoms of ADHD and ASD, though it has been suggested that the EF deficits of the comorbid profile are likely to be greater than in either disorder alone (Reiersen 2011).

Executive functions are conceptualised as an intercorrelated system of higher-order cognitive processes (Miyake et al. 2000) that allow purposeful, goal-directed behaviour in novel or complex circumstances (Walsh 1988). They include planning and reasoning, cognitive flexibility, response inhibition and working memory. Executive skills emerge gradually throughout development, closely

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aligning with the hierarchical development and increased connectivity of the frontal lobes (Spencer-Smith and Anderson 2009; Welsh and Pennington 1988). EF is thought to be mediated by a diffuse neural network involving the prefrontal cortex, and extending to other regions including the brain stem, occipital, temporal and parietal lobes, as well as limbic and subcortical regions (Anderson 2002). As such, efficient EF relies not only on the integrity of the frontal lobes, but on wider brain functioning and lower-order cognitive processes such as perception, visuo-motor skills and auditory processing (Anderson 2002).

While no clear consensus has been achieved, ADHD and ASD are associated with structural and functional brain abnormalities in regions associated with EF (Spencer-Smith and Anderson 2009), including the prefrontal cortex, anterior cingulate, motor regions and basal ganglia (Bradshaw and Sheppard 2000). However, there are important neurobiological differences between ADHD and ASD; ADHD is associated with a delay in structural brain maturation, whereas ASD is characterised by early brain overgrowth (Anagnostou and Taylor 2011; Stanfield et al. 2008). Further, reduced grey matter in the right posterior cerebellum is specific to ADHD, while increased grey matter in the middle/superior temporal gyrus is specific to ASD (Lim et al. 2015). Noteworthy, children with comorbid symptoms of ADHD and ASD (ADHD + ASD) share ADHD-like abnormalities in the basal ganglia not present in ASD alone (Di Martino et al. 2013), and also share symptoms of inattention as observed in ADHD (Bink et al. 2015). Overlap of neurobiological traits and symptoms suggests that some degree of overlap is also likely in EF deficits between children with ADHD, ASD and ADHD + ASD.

Many studies have examined EF in ADHD and ASD cohorts (Pennington and Ozonoff 1996; Willcutt et al. 2005), with mixed findings, though deficits in inhibition, spatial working memory and reasoning are apparent in both ADHD and ASD (Bühler et al. 2011; Efron et al. 2014; Geurts et al. 2004; Goldberg et al. 2005; Happé et al. 2006; Matsuura et al. 2014; Semrud-Clikeman et al. 2010; Takeuchi et al. 2013; Tye et al. 2014). However, the disorders are dissociable: compared to ADHD, ASD appears more closely associated with deficits in planning, set-shifting, self-monitoring and organization (Geurts et al. 2004; Semrud-Clikeman et al. 2010; Yerys et al. 2009), whereas ADHD appears uniquely associated with verbal working memory deficits (Takeuchi et al. 2013; van der Meer et al. 2012).

Fewer studies have examined EF in children with ADHD + ASD in comparison to samples of children with ADHD or ASD alone, where children have been classified into categorical groups according to diagnosis (Bühler et al. 2011; Sinzig et al. 2008; Takeuchi et al. 2013; Tye et al.

2014; van der Meer et al. 2012, 2014; Yerys et al. 2009). Some studies report children with ADHD + ASD demonstrate similar deficits to ADHD—with both groups impaired compared to typically developing children—in response inhibition (Bühler et al. 2011; Takeuchi et al. 2013; Tye et al. 2014; Yerys et al. 2009), spatial working memory (Takeuchi et al. 2013; van der Meer et al. 2012; Yerys et al. 2009) and verbal working memory (Takeuchi et al. 2013; Yerys et al. 2009). In contrast, others have found children with ADHD + ASD do not differ from typically developing children across the same domains: response inhibition (Sinzig et al. 2008; van der Meer et al. 2012, 2014), spatial working memory (Sinzig et al. 2008; van der Meer et al. 2014), or verbal working memory (van der Meer et al. 2012, 2014). It is currently unclear whether deficits in reasoning are also evident in children with ADHD + ASD. Of note, no study to date has directly investigated the effects of lower-order functioning (e.g., auditory processing), which may impact efficient EF. While differentiating such lower-order deficits from specific EFs in these children is important for understanding neurocognitive profiles, it is also critical for designing appropriate interventions.

Only one study to date has reported on the ‘dimensional’ relationships between levels of ADHD or ASD symptoms and EF. In a clinical sample of 104 Japanese boys aged 6–15 years, Takeuchi et al. (2013) report that greater inattention symptoms are related to poorer inhibition and verbal working memory, but not to spatial working memory or interference control. The reverse was found for ASD symptoms: greater symptoms were not associated with worse inhibition and working memory, but were related to poorer interference control. In contrast, hyperactivity symptoms were unrelated to EF (Takeuchi et al. 2013). These findings provide provisional support for differential EF profiles for ADHD and ASD.

In summary, existing studies of EF in children with ADHD + ASD are limited by an almost exclusive use of clinical samples which tend to be biased towards males and those with more severe and comorbid mental health problems, and under-represent those with predominantly inattentive symptoms (Brassett-Harknett and Butler 2007), with some examining only boys (Takeuchi et al. 2013; Tye et al. 2014). Additionally, existing studies span broad age ranges, including children aged 5 years to adults of 22 years, and thus crossing multiple critical periods for EF development. Finally, with regard to measurement, the wide range of tools employed across studies limits the potential to generalise results.

The current study addresses these limitations by examining EF in a community-based sample of 6–8 year old children with ADHD + ASD compared to children with ADHD alone and to non-ADHD/ASD controls (hereafter

referred to as ‘controls’). We aimed to examine the categorical differences between groups in the EF domains of response inhibition, verbal working memory and reasoning, and the dimensional relationships between these EFs and symptoms of ADHD and ASD, as previous findings of categorical differences between children with ADHD and those with ADHD + ASD have been equivocal. Based on available literature we hypothesised that: (1) children with ADHD + ASD would demonstrate an EF profile similar to children with ADHD alone; (2) working memory ability would be impacted by auditory processing capacity; and (3) in line with Takeuchi et al. (2013), we predicted that inattentive, but not ASD, symptoms would be associated with poorer inhibition and verbal working memory, and that hyperactivity symptoms would be unrelated to EF. Finally, we aimed to explore whether ASD and inattention symptoms would be associated with reasoning ability.

Methods

Study Design and Participants

Participants were a subset of children participating in the Children’s Attention Project (CAP), a prospective, longitudinal community cohort, recruited from 43 government primary schools in Melbourne, Victoria (see Sciberras et al. 2013 for study protocol¹). Children were originally recruited during the second year of formal schooling in 2011 and 2012 following an ADHD screening and case confirmation procedure. Exclusion criteria were: intellectual disability, serious medical conditions, genetic disorders, moderate to severe sensory impairment, neurological problems, or parents with insufficient English language skills to complete interviews or questionnaires (Sciberras et al. 2013). For the current study, participants were further excluded if: (1) the child screened positive for ADHD, but were found negative for ADHD in the diagnostic interview ($n = 100$), due to our rigorous requirements for ADHD status, (2) they were included in the original control group (i.e. were negative for ADHD), though displayed significant symptoms of ASD ($n = 6$; not analysed separately due to low number); (3) if ASD symptom data were unavailable ($n = 25$); or (4) if the child was taking ADHD medication (e.g. methylphenidate, dexamphetamine and atomoxetine) at the time of assessment ($n = 21$) due to the potential effects of such medication on EF (Pietrzak et al. 2006).

Children were allocated to one of three groups as follows: (1) *control group*: children who screened negative for ADHD and did not meet criteria for either ADHD (i.e.

negative screen and negative diagnosis) or ASD (i.e. SCQ score < 15); (2) *ADHD group*: children who screened positive and met diagnostic criteria for ADHD during assessment, but scored below the clinical cut-off for ASD (i.e. SCQ score < 15); and (3) *ADHD + ASD group*: children who screened positive and met diagnostic criteria for ADHD during assessment, and also demonstrated clinically elevated symptoms of ASD (i.e. SCQ score ≥ 15). The final sample for this study consisted of 192 non-ADHD/ASD controls, 115 children with ADHD, and 32 children with ADHD + ASD.

Informed consent was obtained from all participants and ethical approval was granted by the Human Research Ethics Committees of the Royal Children’s Hospital (#31056) and the Victorian Department of Education and Early Childhood Development (#2011_001095) (Sciberras et al. 2013).

Measures

Group Status

ADHD status Children were screened for ADHD using both the parent and teacher versions of the 10-item Conners 3 ADHD Index (Conners 2008). The Conners ADHD Index is a composite measure that assesses inattention, hyperactivity and impulsivity. This overall measure was used at the symptom screening stage, not as a specific measure of inattention or hyperactivity symptoms alone. The measure requires parents and teachers to rate the child’s behaviour within the past month on a 4-point scale from ‘not true at all’ to ‘very much true’. Both parent and teacher versions of the Conners 3 are considered valid and reliable ($\alpha = 0.71–0.98$). Children screened positive for ADHD if parents reported a current diagnosis, or if scores were greater than or equal to the 75th percentile for boys and the 80th percentile for girls on both parent and teacher indices. These cut off points were used based on pilot study data that showed consistency with DSM-IV ADHD criteria on the NIMH Diagnostic Interview Schedule for Children Version IV (DISC-IV) (Shaffer et al. 2000). If scores did not meet these thresholds and no ADHD diagnosis was reported, children were considered to screen negative (Sciberras et al. 2013). ADHD status was then confirmed via the DISC-IV, a structured interview assessing a range of psychiatric diagnoses according to DSM-IV criteria. The measure has good diagnostic validity and reliability (Shaffer et al. 2000). Children were considered positive for ADHD if they screened positive and were confirmed positive for ADHD via diagnostic interview.

ASD symptoms were measured using the Social Communication Questionnaire (SCQ) Lifetime Version (Rutter et al. 2003), which has good internal validity (Wei et al. 2015) and high correlation with the Autism Diagnostic

¹ Data on these children’s EF in relation to ASD status have not previously been reported.

Interview-Revised (ADI-R) on which it is based (Rutter et al. 2003). Parents responded ‘yes’ or ‘no’ to the 40 items in an interview led by researchers. The SCQ provides a total score with symptoms considered as clinically elevated when the total score is equal to or exceeds 15.

Executive Function

EF measures were selected to be appropriate for the study age group, and to include the skills outlined in Miyake’s (2000) EF model. All mean (M) and standard deviation (SD) values expressed in this section are based on the norms for each measure based on the child’s age.

Verbal working memory was assessed using Digit Span Backward from the Wechsler Intelligence Scale for Children, 4th Edition (Wechsler 2003) (WISC-IV; scaled to $M = 10$, $SD = 3$). The task requires children to repeat in reverse order a span of 2–9 numbers read aloud by the examiner. The task is considered reliable ($\alpha = 0.68$ – 0.83) in 6–8 year olds.

Non-verbal problem solving (reasoning) was assessed using Matrix Reasoning from the Wechsler Abbreviated Scale of Intelligence (Wechsler 1999) (WASI; T-score, $M = 50$, $SD = 10$). The task requires children to select the missing piece of a matrix from five options. The task has an average reliability coefficient of 0.87 in children, and good concurrent validity with other measures of intelligence and achievement.

Response inhibition was assessed using “Walk-Don’t-Walk” from the Test of Everyday Attention for Children (Manly et al. 1999) (TEA-Ch; $M = 10$, $SD = 3$). The task is completed using a laminated A4 sheet of paper and requires children to attend and respond to two auditory stimuli; a ‘beep’ signals children to mark the corresponding tile of a path, whereas a ‘beep’ followed by a ‘crash’ requires children to inhibit their response of marking tiles on the path. The task is reliable ($\alpha = 0.71$) and valid, with a regression coefficient of $b = 0.46$ within a structural equation model that fits all TEA-Ch items to three factors (Manly et al. 1999).

Lower-Order Cognitive Function

Auditory processing capacity was measured using Digit Span Forward from the WISC-IV (Wechsler 2003) (scaled to $M = 10$, $SD = 3$) and was employed to assess lower-order skills related to working memory. The task requires children to repeat a span of 2–9 numbers in the same order as read aloud by the examiner. The task is considered reliable ($\alpha = 0.79$ – 0.83) in 6–8 year olds.

Confounding Variables

Variables identified a priori as confounders included whether the child was positive (yes/no) for an internalising

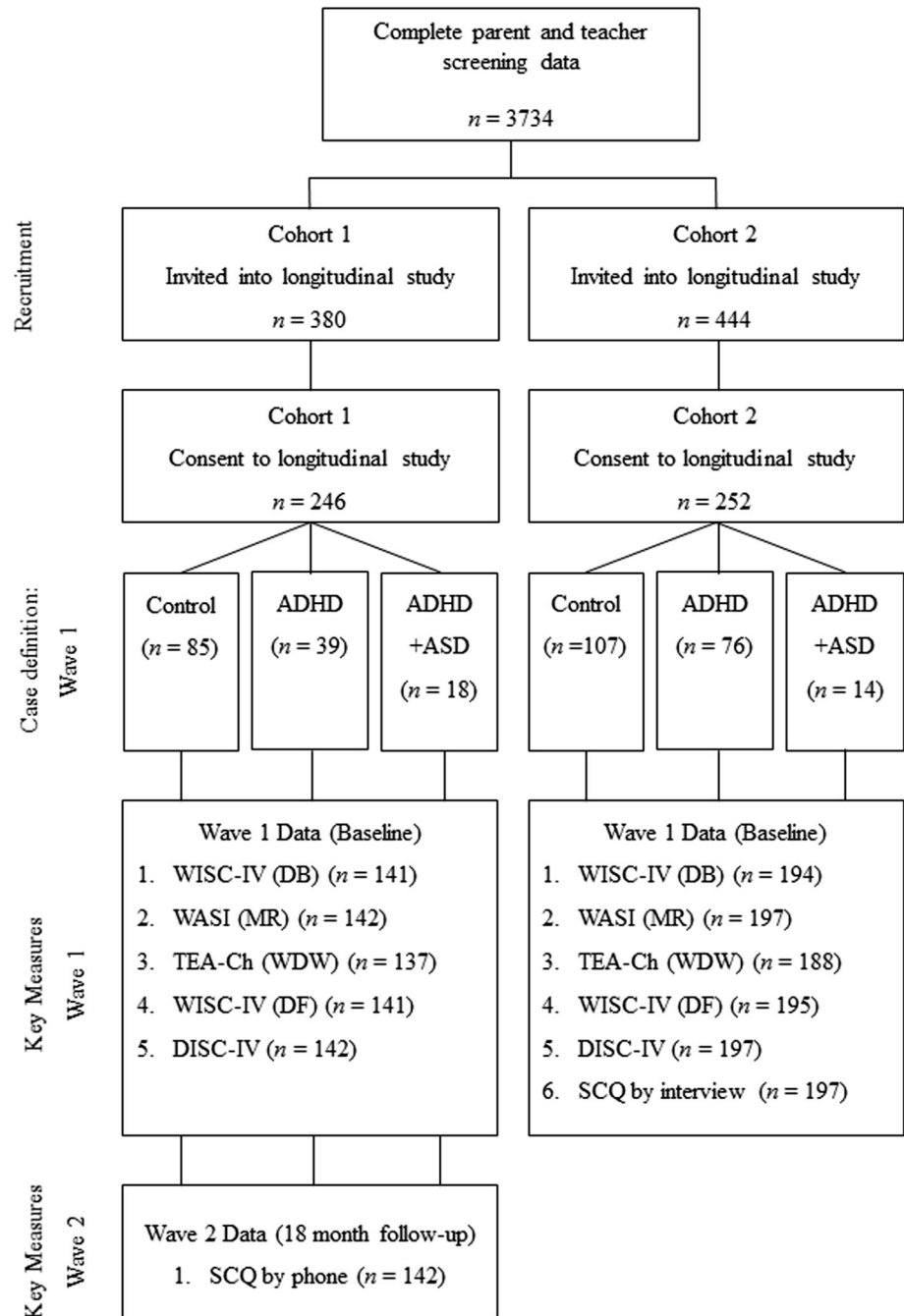
(e.g. generalised anxiety disorder, social phobia, specific phobia, panic disorder, obsessive compulsive disorder, post-traumatic stress disorder, tic disorder, separation anxiety disorder, major depressive disorder, and dysthymic disorder) or externalising disorder (e.g. oppositional defiant disorder, conduct disorder) in the past year as measured by the DISC-IV (Shaffer et al. 2000). Primary caregiver high-school completion (yes/no)—a proximal marker for socioeconomic status—was later included as a covariate as descriptive analyses revealed significant difference across groups. All cognitive measures were age-normed and no gender differences were observed between groups, therefore covariates for age and gender were not included.

Procedure

Full procedures have been reported elsewhere (Sciberras et al. 2013). Briefly, at baseline, children were screened for ADHD using parent- and teacher-reported Conners 3 ADHD Indices (Conners 2008). Children’s ADHD status was assessed via the DISC-IV interview with parents at home or at the child’s school. Direct neuropsychological cognitive assessments were conducted at the child’s school as part of a wider assessment of children’s cognitive, academic language and EF, plus height and weight measurements. Tests were administered in the same order for all participants and no counterbalancing occurred. Assessments ran for 60–90 min with no breaks and were conducted by trained researchers who were blind to child screening status (Sciberras et al. 2013). Parents completed the SCQ as part of the diagnostic interview at baseline for Cohort 2, and via phone interview at the 18-month follow-up for Cohort 1. See Fig. 1 for participant flowchart.

Statistical Analysis

Chi Square and ANOVA were used to compare sample characteristics (e.g. child sex, age, ASD and ADHD symptoms; family socioeconomic status) between children with ADHD + ASD, ADHD and controls (Table 1). For our first hypothesis, unadjusted and adjusted linear regressions were employed to examine differences in EF for children with ADHD + ASD compared to ADHD and compared to controls. Regressions were conducted with two dummy variables for group status as independent variables with ADHD + ASD as the reference group. Scores on each EF task were included as dependent variables. Regressions were subsequently re-run with ADHD as the reference group. For hypothesis 2, analyses for working memory were further adjusted including Digit Span Forward as an additional covariate to determine whether working memory was impacted by lower-order cognition. Finally, unadjusted and adjusted linear regressions were conducted to determine the

Fig. 1 Participant flowchart

dimensional relationships between EF and ASD, inattention and hyperactivity symptoms (Hypothesis 3). Regressions included ASD, inattention and hyperactivity symptom scales as independent variables and performance on each EF task as dependent variables.

For hypotheses 1 and 3, adjusted analyses included covariates for comorbid internalising and externalising disorders and primary caregiver high-school completion. In adjusted analyses, all cases with missing data were excluded; 5 controls, 7 ADHD and 1 ADHD + ASD. All were

due to missing parent education data. No models accounted for school clustering as variables in the current study were all at the individual level of analysis. Similarly, no models adjusted for IQ, as IQ is causal in cognitive functioning such that adjusting for IQ would remove warranted variation in EF outcomes (Dennis et al. 2009).

In adjusted analyses, the study had 80 % power to detect effect size differences of 0.34–0.35 between ADHD and controls, 0.55–0.57 between ADHD + ASD and controls, and 0.57–0.60 between ADHD + ASD and ADHD. For all

Table 1 Sample characteristics and demographics

	Symptom Group			<i>p</i>
	Control (<i>n</i> = 192)	ADHD (<i>n</i> = 115)	ADHD + ASD (<i>n</i> = 32)	
<i>Child characteristics</i>				
Male [<i>n</i> (%)]	123 (64 %)	75 (65 %)	25 (78 %)	0.30
Child age in years [mean (<i>SD</i>)]	7.3 (0.4)	7.2 (0.4)	7.5 (0.5)	0.002
Vocabulary ^a [mean (<i>SD</i>)]	50.0 (8.3)	45.2 (9.2)	40.9 (9.3)	<0.001
ASD symptoms ^b [mean (<i>SD</i>)]	4.9 (3.2)	6.9 (3.8)	20.9 (5.1)	<0.001
ADHD symptom severity [mean (<i>SD</i>)]	1.2 (2.0)	12.3 (4.8)	14.7 (3.4)	<0.001
<i>ADHD subtype [<i>n</i> (%)]</i>				
ADHD—combined	–	52 (45.2 %)	18 (56.3 %)	0.41
ADHD—inattentive	–	51 (44.4 %)	10 (31.3 %)	0.41
ADHD—hyperactive/impulsive	–	12 (10.4 %)	4 (12.5 %)	0.41
Internalising comorbidity in past year ^c [<i>n</i> (%)]	8 (4.2 %)	25 (21.7 %)	12 (37.5 %)	<0.001
Externalising comorbidity in past year ^c [<i>n</i> (%)]	14 (7.3 %)	56 (48.7 %)	18 (56.3 %)	<0.001
<i>Primary caregiver/family characteristics</i>				
SEIFA ^d [mean (<i>SD</i>)]	1015.6 (45.7)	1019.2 (41.8)	1001.7 (37.4)	0.14
Primary caregiver high school completion [<i>n</i> (%)]	153 (81.8 %)	70 (64.8 %)	20 (64.5 %)	0.002

p values for categorical variables refer to Chi square, and to ANOVA for continuous variables. Bolding denotes significant results

^a Wechsler Abbreviated Scale of Intelligence (WASI; T-score, *M* = 50, *SD* = 10)

^b Social Communication Questionnaire (SCQ)

^c Diagnostic Interview Schedule for Children Version IV (DISC-IV)

^d Socio-Economic Indexes for Areas (SEIFA) Disadvantage Index

comparisons, an alpha level of 0.05 was used and exact *p* values and effect sizes are reported. Although a number of statistical tests were performed, which may contribute to Type I error, we have been cautious in interpreting results rather than adjusting for multiple comparisons, as such adjustments increase Type II error and may be overly conservative for the interpretation of the results (Perneger 1998). Furthermore, Howell (2002) argues that correction for multiple comparisons is not warranted where a priori predictions are made. All analyses were conducted using Stata 13.0.

Results

Sample Characteristics and Demographics (Table 1)

Compared to other groups, children with ADHD + ASD were significantly older (*M* = 7.5, *SD* = 0.5), had lower vocabulary scores (*M* = 40.9, *SD* = 9.3), and were more likely to have had an internalising or externalising comorbidity in the past year (*n* = 12, 37.5 %; *n* = 18, 56.3 %, respectively). Primary caregivers of controls were more likely to have completed high school (*n* = 153, 81.8 %) than either ADHD or ADHD + ASD groups

(*n* = 70, 64.8 %; *n* = 20, 64.5 %, respectively). There were no significant group differences for gender or neighbourhood socio-economic disadvantage (Socio-Economic Indexes for Areas; SEIFA) (Australian Bureau of Statistics 2013). Similarly, the ADHD + ASD and ADHD groups did not differ by ADHD subtype.

Differences in EF Between Children with ADHD + ASD, ADHD, and Controls (Table 2)

In unadjusted analyses, group status accounted for 7–9 % of variance in EF measures. There were no significant differences between children with ADHD and those with ADHD + ASD on any EF task; Digit Span Backward ($\beta = -0.02$, *p* = 0.84), Matrix Reasoning ($\beta = 0.11$, *p* = 0.25), and Walk-Don't-Walk ($\beta = -0.004$, *p* = 0.96). In contrast, the control group performed significantly better than children with ADHD + ASD across all EF tasks; Digit Span Backward ($\beta = 0.25$, *p* = 0.01), Matrix Reasoning ($\beta = 0.35$, *p* < 0.001), and Walk-Don't-Walk ($\beta = 0.30$, *p* = 0.002; Fig. 2). Subsequent analyses revealed the control group also performed significantly better than children with ADHD across all tasks; Digit Span Backward ($\beta = 0.27$, *p* < 0.001), Matrix Reasoning ($\beta = 0.24$, *p* < 0.001), and Walk-Don't-Walk ($\beta = 0.30$,

Table 2 Unadjusted and adjusted differences in executive functioning between children with ADHD + ASD, ADHD and controls

Outcomes	Unadjusted					Adjusted ^a						
	<i>R</i> ²	ADHD + ASD (<i>n</i> = 32)	ADHD ^b (<i>n</i> = 115)		Control (<i>n</i> = 192)	<i>R</i> ²	ADHD + ASD (<i>n</i> = 31)	ADHD ^b (<i>n</i> = 108)		Control (<i>n</i> = 187)		
			β	<i>p</i>				β	<i>p</i>	β	<i>p</i>	β
<i>Working Memory</i>												
Digit Span Backward ^c	0.07	Ref.	-0.02	0.84	0.25	0.010	0.08	Ref.	-0.006	0.95	0.18	0.091
<i>Reasoning</i>												
Matrix Reasoning ^d	0.07	Ref.	0.11	0.25	0.35	<0.001	0.10	Ref.	0.10	0.26	0.29	0.003
<i>Response Inhibition</i>												
Walk-Don't-Walk ^e	0.09	Ref.	-0.004	0.96	0.30	0.002	0.10	Ref.	-0.03	0.73	0.21	0.045

Bolding denotes significant results

^a Adjusted analyses covaried for comorbid internalising and externalising disorders, and primary caregiver high school completion. See in text results for the effects of an additional covariate for Digit Span Forward scores on working memory function

^b The ADHD group differed significantly from the control group for all tasks, in unadjusted ($p < 0.001$) and adjusted analyses ($p < 0.01$)

^c Wechsler Intelligence Scale for Children, 4th Edition (WISC-IV)

^d Wechsler Abbreviated Scale of Intelligence (WASI)

^e Test of Everyday Attention for Children (TEA-Ch)

$p < 0.001$). Most differences remained significant in adjusted analyses, though the difference between controls and children with ADHD + ASD attenuated for Digit Span Backward ($\beta = 0.18$, $p = 0.09$).

Analyses examining performance on Digit Span Backward (working memory) were further adjusted for Digit Span Forward (auditory processing capacity). Digit Span Forward was a significant factor in Digit Span Backward ability ($\beta = 0.32$, $p < 0.001$). With the inclusion of this covariate, the control group no longer differed from children with ADHD + ASD ($\beta = 0.06$, $p = 0.54$), and performed only marginally better than children with ADHD ($\beta = 0.12$, $p = 0.07$) where these differences were previously marginal and significant, respectively. Children with ADHD remained no different from children with ADHD + ASD ($\beta = -0.05$, $p = 0.56$).

Dimensional Relationship Between ADHD/ASD Symptoms and EF (Table 3)

In unadjusted analyses, ASD, inattention and hyperactivity symptoms jointly accounted for 7–10 % of variance in EF scores. Greater ASD symptoms were significantly related to poorer performance on Matrix Reasoning and Walk-Don't-Walk ($\beta = -0.14$, $p = 0.02$; $\beta = -0.13$, $p = 0.03$, respectively). Greater inattention symptoms were significantly related to poorer performance on Digit Span Backward and Matrix Reasoning ($\beta = -0.21$, $p = 0.01$; $\beta = -0.19$, $p = 0.02$, respectively), and marginally

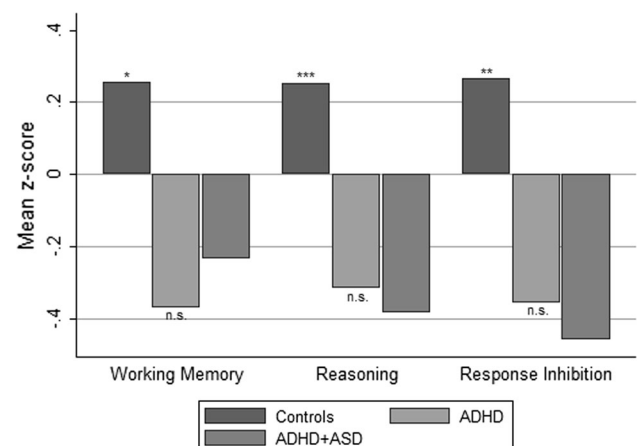


Fig. 2 Mean z-score for children with ADHD + ASD, ADHD and controls, for each executive function task. * Significantly different to children with ADHD + ASD at $p < 0.05$. ** Significantly different to children with ADHD + ASD at $p < 0.01$. *** Significantly different to children with ADHD + ASD at $p < 0.001$. n.s. not significantly different to children with ADHD + ASD ($p > 0.25$). Notes Working Memory as measured by the Wechsler Intelligence Scale for Children, 4th Edition (WISC; Digit Span Backward); Reasoning as measured by the Wechsler Abbreviated Scale of Intelligence (WASI; Matrix Reasoning); Response Inhibition as measured by the Test of Everyday Attention for Children (TEA-Ch; Walk-Don't-Walk)

related to poorer performance on Walk-Don't-Walk ($\beta = -0.14$, $p = 0.08$). Most findings were maintained in adjusted analyses, though associations between ASD and inattention symptoms and performance on Walk-Don't-

Table 3 Unadjusted and adjusted dimensional associations between ASD, inattention and hyperactivity symptoms and executive functioning in total sample

Outcomes	Unadjusted						Adjusted ^a							
	R ²	ASD (n = 339)		Inattention (n = 339)		Hyperactivity (n = 339)		R ²	ASD (n = 326)		Inattention (n = 326)		Hyperactivity (n = 326)	
		β	p	β	p	β	p		β	p	β	p	β	p
<i>Working memory</i>														
Digit Span Backward ^b	0.08	-0.03	0.62	-0.21	0.01	-0.07	0.38	0.09	-0.03	0.67	-0.20	0.02	0.01	0.87
<i>Reasoning</i>														
Matrix Reasoning ^c	0.10	-0.14	0.02	-0.19	0.02	-0.05	0.51	0.13	-0.15	0.02	-0.21	0.01	0.001	0.99
<i>Response inhibition</i>														
Walk-Don't-Walk ^d	0.07	-0.13	0.03	-0.14	0.08	-0.06	0.49	0.09	-0.12	0.06	-0.13	0.11	0.02	0.83

Bolding denotes significant results

^a Adjusted analyses covaried for comorbid internalising and externalising disorders, and primary caregiver high school completion

^b Wechsler Intelligence Scale for Children, 4th Edition (WISC-IV)

^c Wechsler Abbreviated Scale of Intelligence (WASI)

^d Test of Everyday Attention for Children (TEA-Ch)

Walk became marginal and non-significant, respectively ($\beta = -0.12$, $p = 0.06$; $\beta = -0.13$, $p = 0.11$, respectively). Hyperactivity symptoms were not significantly related to any EF measure in unadjusted or adjusted analyses.

Discussion

This study examined whether 6–8 year old children with ADHD + ASD demonstrate similar EF profiles to children with ADHD alone, and investigated the dimensional relationships between ASD, inattention and hyperactivity symptoms and children’s EF. This study had two main findings: Categorical analyses revealed no difference in EF between children with ADHD + ASD and those with ADHD, while dimensional analyses revealed that ASD, inattention and hyperactivity symptoms are differentially related to EF. Specifically, in categorical analyses, we found children with ADHD + ASD did not differ from children with ADHD on any comparison across the executive domains of response inhibition, reasoning and working memory. Children with ADHD + ASD did, however, display significant deficits compared to controls across all domains, though deficits in working memory attenuated in adjusted analyses. In relation to this latter finding, further analyses revealed auditory processing capacity was a significant factor in the working memory deficits observed in children with ADHD and ADHD + ASD, illustrating the importance of exploration of lower-order cognitive skills, when assessing EF. In dimensional analyses, we found greater ASD or inattention

symptoms were associated with poorer reasoning and response inhibition. Further, greater inattention symptoms were linked to poorer verbal working memory. In contrast, hyperactivity symptoms were not related to any EF tasks. These findings may reflect the increased sensitivity and power of considering dimensional relationships, as opposed to applying somewhat arbitrary clinical cut-points. Thus it is important to use both diagnostic and symptom based approaches in clinical settings when assessing these children’s functional abilities.

The current study provides support for claims that children with ADHD + ASD are likely to demonstrate the EF deficits associated with ADHD alone (Gargaro et al. 2011). Similarly to previous studies, children with ADHD + ASD demonstrated similar impairment to ADHD in response inhibition (Bühler et al. 2011; Takeuchi et al. 2013; Tye et al. 2014). The current study extended previous findings, revealing that children with ADHD + ASD display similar deficits in reasoning to children with ADHD alone.

Similarly to van der Meer et al’s. 2012 mixed clinical and community-based study, we found children with ADHD + ASD were not impaired in verbal working memory compared to controls after controlling for confounders, despite children with ADHD demonstrating a significant deficit. In contrast to these findings, verbal working memory deficits have been observed in small, clinical samples of children with ADHD + ASD (Takeuchi et al. 2013; Yerys et al. 2009), suggesting clinical samples may be more likely than community-based samples to report significant verbal working memory deficits in children with ADHD + ASD.

Unlike previous studies, we directly examined the impact of lower-order cognitive functioning on EF by adjusting for auditory processing skills. Noteworthy from these analyses, significant deficits in working memory became marginal and non-significant for children with ADHD and those with ADHD + ASD, respectively. These results suggest that working memory deficits commonly detected in ADHD and ADHD + ASD may well be linked to auditory processing capacity, rather than reflecting a 'pure' EF deficit. Together, these findings highlight a potential benefit of pairing neuropsychological testing with wider cognitive assessment to comprehensively characterise deficits associated with ADHD and the comorbid profile.

Across all results, the magnitude of group differences was small, despite being significant, revealing that other factors are important in distinguishing children with ADHD from their typically developing peers. Not all children with ADHD display deficits in EF; one review notes that only half of children with ADHD are impaired on any given EF task (Willcutt et al. 2005). Such heterogeneity in findings supports the theoretical notion that there are multiple developmental pathways to ADHD including, but not limited to, executive dysfunction (Sonuga-Barke 2005).

Mirroring previous findings (Takeuchi et al. 2013), we found that greater inattention—but not greater ASD—symptoms were related to poorer verbal working memory; greater inattention symptoms were marginally related to poorer response inhibition; and hyperactivity symptoms were unrelated to EF. Of note, the marginal relationship between inattention symptoms and response inhibition did not hold in our adjusted analyses. We also demonstrated that greater ASD and greater inattention symptoms were linked to poorer reasoning. Extrapolating these findings, children with ADHD Inattentive type (ADHD-I) or Combined type (ADHD-C) may be at greater risk of cognitive impairments than children with ADHD Hyperactive/Impulsive type (ADHD-H/I), who are more likely to have behavioural problems (Shaywitz et al. 1995). In contrast to previous findings (Takeuchi et al. 2013), we found greater ASD symptoms were associated with poorer response inhibition. This difference may arise as Takeuchi and colleagues' 'ASD' group included participants with wider Pervasive Developmental Disorders, which likely led to greater variance in ability levels.

Strengths of the current study include the rigorous diagnosis of ADHD, the use of a range of EF measures, and the use of both categorical and dimensional analyses. This study uniquely employed a community-based sample of children within a narrow age-band and developmental stage to reduce ascertainment biases and variation due to developmental level. The study also directly investigated the effects of lower order functioning on observed EF

deficits. The study does have limitations. Whereas children's ADHD status was confirmed via diagnostic interview, we did not confirm ASD diagnosis, though measured children's ASD symptoms—similarly with inattention and hyperactivity symptoms—to examine dimensional relationships. SCQ scores were measured at different time-points for the two cohorts; 18 months after cognitive assessment for Cohort 1 and simultaneously with cognitive assessment for Cohort 2. It is possible that the relationship between ASD symptoms and EF may have changed during the delay in SCQ assessment for Cohort 1, however, ASD symptoms are considered fairly stable over time (St Pourcain et al. 2011). EF and lower-order cognitive measures were limited to those collected at baseline, which meant that measures of EF domains more closely linked to ASD, such as cognitive flexibility/fluency or planning (Sinzig et al. 2008; Yerys et al. 2009), and lower-order measures related to reasoning and response inhibition were unavailable. Finally, the small sample of children with ADHD + ASD may have contributed to reduced power to detect small differences between these children and those with ADHD alone.

Overall, this study found children with ADHD + ASD demonstrate many of the EF deficits observed in children with ADHD alone. Poorer auditory processing capacity was a significant factor for working memory deficits in children with ADHD and those with ADHD + ASD, highlighting that future research should examine the impact of lower-order cognitive deficits on EF. The study provides evidence that inattention, hyperactivity and ASD symptoms are differentially related to EF, and that it is important to use both diagnostic and symptom based approaches in clinical settings when assessing these children's functional abilities. Future studies may ultimately determine whether children with comorbid ADHD/ASD symptoms display the EF deficits associated with both ADHD and ASD, as well as the trajectories of children who display EF deficits at a young age.

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Authors' Contributions RN performed the statistical analysis, participated in the interpretation of data, and drafted the manuscript; JG assisted with the statistical analysis, interpretation of data and drafting the manuscript; ES participated in the design and coordination of the study, interpretation of data and drafting the manuscript; PH participated in the design of the study and revised the manuscript critically for important intellectual content; VA participated in the design of the study, interpretation of data, and drafting the manuscript. All authors read and approved the final manuscript.

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Compliance with Ethical Standards

Conflict of interest VA declares a financial disclosure and conflict of interest associated with the Test of Everyday Attention for Children (TEA-Ch), as she receives royalties from Pearson Publishing. All other authors declare that they have no conflict of interest.

Ethical Approval Ethical approval was granted by the Human Research Ethics Committees of the Royal Children's Hospital (#31056) and the Victorian Department of Education and Early Childhood Development (#2011_001095). All procedures performed in the study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed Consent Informed consent was obtained from all participants and ethical approval was granted by the Human Research Ethics Committees of the Royal Children's Hospital (#31056) and the Victorian Department of Education and Early Childhood Development (#2011_001095).

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