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Examining executive functioning in children with autism spectrum disorder, attention deficit hyperactivity disorder and typical development

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Abstract

Executive functioning (EF) is an overarching term that refers to neuropsychological processes that enable physical, cognitive, and emotional self-control. Deficits in EF are often present in neurodevelopmental disorders, but the specificity of EF deficits and direct comparison across disorders is rare. The current study investigated EF in 7 to 12 year old children with autism spectrum disorder (ASD), attention deficit hyperactivity disorder (ADHD) and typical development using a comprehensive battery of measures assessing EF, including response inhibition, working memory, cognitive flexibility, planning, fluency and vigilance. The ADHD group exhibited deficits in vigilance, inhibition and working memory relative to the typical group; however, they did not consistently demonstrate problems on the remaining EF measures. Children with ASD showed significant deficits in vigilance compared to the typical group, and significant differences in response inhibition, cognitive flexibility/switching, and working memory compared to both groups. These results lend support for previous findings that show children with autism demonstrate generalized and profound impairment in EF. In addition, the observed deficits in vigilance and inhibitory control suggest that a significant number of children with ASD present with cognitive profiles consistent with ADHD.

Keywords

attention; inhibition; comorbidity; working memory; CANTAB; IVA; vigilance

1. Introduction

Executive function (EF) is an overarching term that refers to mental control processes that enable physical, cognitive, and emotional self-control (Denckla, 1996; Lezak, 1995; Pennington & Ozonoff, 1996) and are necessary to maintain effective goal-directed behavior (Welsh, 1988). Executive functions generally include response inhibition, working memory,

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cognitive flexibility (set shifting), planning and fluency (Ozonoff & Strayer, 1997; Pennington & Ozonoff, 1996). Deficits in EF are frequently observed in neurodevelopmental disorders, including autism and attention deficit hyperactivity disorder (ADHD).

Autism is a severe neurodevelopmental disorder characterized by impairment in communication, reciprocal social interaction, and a markedly restricted repertoire of activities and interests (APA, 2000). The symptoms of autism fall on a continuum of severity referred to as autism spectrum disorder (ASD), which include autistic disorder, Asperger syndrome, and pervasive developmental disorder-not otherwise specified (PDD-NOS). Autism is often further divided into those with mental retardation and those functioning in the average or above average range of intelligence (often called high functioning autism or HFA). In addition to the core features, deficits in EF have been widely reported (e.g., (Geurts et al., 2004; Goldberg et al., 2005; Hughes, 1994; Ozonoff et al., 1991; Pennington & Ozonoff, 1996)). The primacy of EF deficits in autism (Russell, 1997) especially in terms of planning, cognitive flexibility and working memory, remains an ongoing debate (for a review see (Hill, 2004)).

ADHD is also a neurologically mediated disorder that exists on a continuum. ADHD is characterized by varying degrees of inattention, impulsivity and hyperactive behavior (APA, 2000). ADHD is further divided into those individuals meeting symptom criteria in all the aforementioned areas (Combined type), those primarily evidencing attention problems (Predominantly-inattentive type) and those with mostly hyperactive and impulsive symptoms (Predominantly-hyperactive-impulsive type). Significant EF deficits in individuals with ADHD have been reported; however, there is still some inconsistency regarding particular impairments in domains of functioning. In a comprehensive meta-analysis, Willcutt, et al 2005, (Willcutt et al., 2005) found that studies most consistently report response inhibition and vigilance deficits in ADHD. Other impairments have been found in working memory (e.g., (Kempton et al., 1999; Rhodes et al., 2005)), planning (e.g., (Kempton et al., 1999; Rhodes et al., 2005)), and flexibility (Vance et al., 2003).

Few studies have directly compared EF across ADHD and ASD groups and studies investigating these groups separately report inconsistent findings. Some have proposed that EF deficits are core to ASD (Russell, 1997) and ADHD (Barkley, 1997). Pennington and Ozonoff suggested that deficits in domains of EF could be disassociated across disorders resulting in distinct EF profiles (Pennington & Ozonoff, 1996), a notion that has received some foundational support. Specifically, impaired motor and response inhibition in ADHD is well supported (e.g., (Barkley et al., 1992; Geurts et al., 2004; Pennington & Ozonoff, 1996). Deficits in planning and set shifting have shown to be more pronounced in individuals with HFA than ADHD and typical development (Geurts et al., 2004; Ozonoff et al., 2004; Ozonoff & Strayer, 1997; Sergeant et al., 2002). Similarly, more impairment in HFA as compared to ADHD has also been demonstrated with verbal working memory (Pennington & Ozonoff, 1996) and spatial working memory (Goldberg et al., 2005; Landa & Goldberg, 2005).

Studies are beginning to emerge directly comparing autism and ADHD groups with their typically developing counterparts (Corbett & Constantine, 2006; Goldberg et al., 2005; Ozonoff & Jensen, 1999; Verte et al., 2006). These studies have generally found EF deficits across both diagnostic groups, with ostensibly more severe and global deficits in ASD. Deficits within the ADHD groups tend to be more consistently restricted to behavioral disinhibition and vigilance (Corbett & Constantine, 2006; Goldberg et al., 2005; Ozonoff & Jensen, 1999; Verte et al., 2006). However, there is evidence that individuals with ADHD may also have deficits in planning, set-shifting, and spatial working memory (e.g., (Kempton et al., 1999)).

Recently, Goldberg and colleagues (Goldberg et al., 2005) examined inhibition, planning, set shifting and working memory in a sample of children 8 to 12 years of age with HFA, ADHD

and typical development. Participants were carefully assessed to screen out comorbid impulsivity or hyperactivity in autism. Using a computerized battery (CANTAB® (Cambridge, 1996)), the study reported that response inhibition, planning, and set-shifting were similar across the three groups of ASD, ADHD and typical development and only impaired spatial working memory in the ADHD and HFA groups were reported (Goldberg et al., 2005). However, age and level of functioning on this measure may explain the limited sensitivity (Goldberg et al., 2005; Landa & Goldberg, 2005).

Since deficits in EF are present in several neurodevelopmental disorders, the issue of *discriminant validity* must be considered as to how disorders with different behavioral phenotype can share similar neuropathological substrates (Ozonoff & Jensen, 1999). Geurts (Geurts et al., 2004) expanded on this notion using a comprehensive neuropsychological battery in children between 6 and 12 years and reported that children with ADHD demonstrated EF deficits in inhibition and verbal fluency while children with HFA showed deficits across most of the EF measures.

Verte et al. (Verte et al., 2006) recently reported significant differences for children with ADHD and HFA in inhibition and response variability compared to children with Tourette Syndrome or typical development. Further, poorer inhibition and more response variability were associated with symptoms of ADHD, while poor working memory was associated with more symptoms of autism. Taken together, the majority of the studies conclude that children with ASD exhibit more pronounced deficits in EF than children with ADHD.

Converging evidence from a variety of methods including chart review (Goldstein & Schwebach, 2004), parent and teacher questionnaires (Gadow et al., 2004) and neuropsychological measures (Corbett & Constantine, 2006) conclude that a high percentage of children with ASD evidence symptoms of ADHD, some warranting a comorbid diagnosis. The etiology of both neurodevelopmental disorders has a strong genetic basis with heritability estimates for autism to be 0.9 (Baron-Cohen & Belmonte, 2005; Zafeiriou et al., 2006) and estimates for ADHD to be 0.7 (Faraone et al., 2005). Furthermore, there are some preliminary findings of a genetic linkage between these disorders at chromosomal locations 2q24 and 16p13 (Fisher et al., 2002; Ogdie et al., 2003; Smalley et al., 2005). Even without consideration of comorbid features, various neuroscientific models have highlighted the common behavioral features, biological pathways and neuroanatomical correlates between ASD and ADHD implicating the frontostriatal system including the frontal lobes and basal ganglia (Damasio & Maurer, 1978; Ozonoff & Jensen, 1999; Pennington & Ozonoff, 1996; Stuss & Benson, 1984). Structural and functional neuroimaging studies show frontal lobe dysfunction in autism (e.g., Carper & Courchesne, 2000, 2005; Courchesne et al., 2001; McAlonan et al., 2005; Muller et al., 2001), and ADHD (e.g., Faraone & Biederman, 1998; Kates et al., 2002; Lou et al., 1984; Mostofsky et al., 2002; Smith et al., 2006; Sowell et al., 2003; Zang et al., 2005)). These brain regions are important in EF, and, as discussed, both disorders have been associated with deficits in EF (e.g., Barkley, 1997; Goldberg et al., 2005; Pennington & Ozonoff, 1996; Russell, 1997)).

Furthermore, many children with ASD display ADHD symptomatology, suggesting that the disorders may share similar traits or are often comorbid (Corbett & Constantine, 2006; Gadow et al., 2004; Geurts et al., 2004; Ghaziuddin et al., 1992; Goldberg et al., 2005; Goldstein & Schwebach, 2004; Verte et al., 2006). Yet, the limited studies that have compared these two disorders generally *exclude* comorbid features (Geurts et al., 2004; Goldberg et al., 2005). Conversely, based on population-based investigation, it has been shown that children diagnosed with ADHD may show autistic traits (Reiersen et al., 2007), which punctuates the importance of investigating comorbid features.

Although we recognize the value of elucidating EF in clearly defined prototypical cases of autism and ADHD, this may not be representative or generalizable to many children on the spectrums of autism or ADHD. Thus, we conducted a comprehensive neuropsychological study to compare and contrast six domains of EF (response inhibition, working memory, flexibility/shifting, planning, fluency and vigilance), in children with ASD, ADHD and typical development deliberately allowing comorbid ADHD features in the children with ASD. We hypothesized that children with ASD would demonstrate greater impairment across a broad range of EF tasks. Simultaneously, we investigated the performance of EF and related it to the level of ADHD symptoms across these groups. We predicted that specific measures of vigilance and behavioral inhibition would be associated with ADHD symptoms across the groups.

2. Methods

2.1. Participants

The participants in this study included: 18 children with high functioning ($IQ > 70$) ASD (autism=12, Asperger=3, PDD-NOS=3); 18 children with ADHD (combined=16, primarily inattentive=1, primarily hyperactive/impulsive=1); and 18 typically developing children (TYP). The demographic information for the groups is presented in Table 1. Regarding medication, 7 ADHD participants were on stimulant medication, 1 of which was also on Clonidine, and 2 of which were on hormone medications. Of the ASD participants, 6 were on stimulants, neuroleptics, SSRIs or a combination of these. While stimulant medication was withheld for 24 hours prior to testing (Greenhill, (1998)), other longer term medications were not stopped for ethical reasons. Inclusion criteria for all participants consisted of having an $IQ \geq 70$, an absence of Fragile X or other serious neurological (e.g., seizures), psychiatric (e.g., Bipolar disorder) or medical conditions.

The University of California, Davis Institutional Review Board (IRB) approved the study. The child's parent completed written informed consent and the child assented to participate in the study. Approximately one third of the children participated in a previous investigation (Corbett & Constantine, 2006). The diagnostic groups were recruited from the University of California, Davis M.I.N.D. (Medical Investigation of Neurodevelopmental Disorders) Institute. Thus, many of the participants already had a confirmed ASD diagnosis with the Autism Diagnostic Observation Schedule (Lord et al., 1999) and the Autism Diagnostic Interview (Lord et al., 1994). For children who were not already evaluated, the following diagnostic procedures were conducted. The diagnosis of autism spectrum disorder (i.e., Autistic Disorder, PDD-NOS, Asperger) was based on DSM-IV criteria (APA, 2000) and established by: 1) a previous diagnosis by either a psychologist, psychiatrist or behavioral pediatrician, 2) clinical judgment by a licensed clinical psychologist (bac), and 3) confirmation by a score on the social-communication scale of the ADOS within or above the autism spectrum threshold (Lord et al., 1999).

The diagnosis of ADHD was based on DSM-IV criteria (APA, 1994) established by: 1) a previous diagnosis of ADHD by either a psychologist, psychiatrist or behavioral pediatrician, 2) clinical judgment by a licensed clinical psychologist (bac), and 3) a semi-structured parent interview extracted from the *Diagnostic Interview Schedule for Children* (DISC) (Shaffer et al., 1996). The presence of autism symptoms was an exclusion for children who had a primary diagnosis of ADHD but none of the ADHD children had to be excluded based on this criteria. The typically developing children were selected based on age and gender and recruited from area schools and recreation centers, then screened via parent interview for the absence of neurodevelopmental disorders, including ASD and ADHD, using the DISC.

2.2. Procedures

Diagnostic and neuropsychological measures were completed in one visit using standardized procedures. A few of the participants were unable to complete every measure. Thus, analyses were completed with only those subjects who had data for the measures. Participants received minimal financial compensation and toys, and their parents/guardians were sent a letter summarizing the assessment results from the published, standardized measures.

2.3. Instruments

Autism Diagnostic Observation Schedule (ADOS) (Lord et al., 1999). The ADOS is comprised of semi-structured interactive activities designed to assess current behaviors indicative of autism involving social behavior, communicative functioning, and restricted activities (Lord et al., 1999). *Wechsler Abbreviated Intelligence Scale* (WASI; (Wechsler, 1999)). The WASI is a measure of general intelligence used to obtain an estimated IQ for inclusion/exclusion into the study.

The following dependent neuropsychological measures are conceptualized based on a previous theoretical model (Pennington & Ozonoff, 1996) and grouped into six EF domains (inhibition, working memory, flexibility, planning, and fluency, vigilance). In addition, a measure of ADHD symptoms was included.

Response Inhibition was measured using the IVA response control quotients and the D-KEFS Color Word Interference Test—The Integrated Visual and Auditory (IVA) Continuous Performance Test (CPT) (Sandford & Turner, 2000) was designed to help in the diagnosis and quantification of the symptoms of ADHD, but it has also been used across a variety of neurodevelopmental and psychiatric conditions. The IVA combines inattention (vigilance) and impulsivity in a counter-balanced design across both visual and auditory modalities. The Visual Response Control Quotient (VRCQ) and Auditory Response Control Quotient (ARCQ) are the primary dependent variables.

The *Dellis-Kaplan Executive Function System* (D-KEFS) (Dellis et al., 2001) consists of nine tests that measure a variety of EFs. The *D-KEFS Color-Word Interference Test* consists of four conditions including: color naming (word finding), word reading (reading speed), and inhibition (verbal inhibition) that expose the child to different reading conditions (fourth condition see Cognitive Flexibility/Switching below). The task is analogous to a Stroop test and performance is based on speed of completion. The inhibition portion requires the ability to verbally inhibit the more salient response of reading words in order to name the color of the discordant ink. These tasks are designed for children 8 years and older. As such, 34 of the 54 participants in the study were administered or able to complete these measures.

Working Memory was measured using the CANTAB Spatial Span and Spatial Working Memory subtests—The *Cambridge Neuropsychological Test Automated Battery* (CANTABexpedio) (Cambridge, 2002) assesses cognitive domains including attention, executive function, memory, processing speed, and visuospatial ability. *Spatial Span* (SSP) measures both forward and reverse spatial memory span. At the onset, white squares are displayed, some of which momentarily change color in an unpredictable pattern. The individual is required to touch the boxes in the same sequence order as they changed color. Throughout the task, the number of boxes in the sequence is increased, and the order and color change with each sequence to minimize interference. The total raw score was used as the dependent variable (Cambridge, 2002). *Spatial Working Memory* (SWM) measures the ability to maintain spatial information and to subsequently manipulate the presented items in working memory. The objective is to find a blue “token” in each of the boxes, then select and place them in an empty column. The individual must resist returning to a box where a token was previously found,

which constitutes an error. The number of boxes is progressively increased. For each trial, the color and position of the boxes are changed. The total spatial working memory between search errors (SWM Btwn Error) and strategy scores (SWM Strat) were used as dependent variables.

Cognitive Flexibility/Switching was measured using the D-KEFS Total Switching Accuracy, CANTAB ID/ED Set Shifting, and Children's Color Trails

Test 2—The *D-KEFS Category Switching (DK T-Switch)*; (Dellis et al., 2001) condition is a measure of cognitive flexibility that requires the individual to shift between color naming, word reading and inhibition (see Color Word Interference Test above), and was used as a dependent variable for flexibility.

Intra-Extra Dimensional Set Shift (ID/ED); (Cambridge, 2002) is a test of rule acquisition and reversal that measures shifting and flexibility of attention, visual discrimination, attention set formation, and maintenance. The ID/ED task consists of colored shapes and white lines that increase in complexity throughout the test. Following 6 consecutive correct responses, the correct shape becomes the incorrect stimuli, and the formerly incorrect shape becomes the correct stimuli (intra-dimensional shift). Through Stage 7, it is the shape that determines which picture is correct. However, beginning with Stage 8, the line identifies the correct picture, (extra-dimensional shift) and cognitive flexibility is required. As this shift is the key measure of cognitive flexibility and many subjects were unable to complete Stage 8, only the number of errors committed in Stage 8 was used as the dependent variable. As directed in the CANTAB manual, if a subject did not complete Stage 8, 25 errors were assigned.

Children's Color Trails Test 1 and 2 (CCTT-1 & 2); (Llorente et al., 1998) are used to measure alternating and sustained visual attention, sequencing ability, psychomotor speed, cognitive flexibility and inhibition. It is analogous to Trail Making tests for adults but modified for children. The CCTT-1 requires the individual to rapidly connect different colored circles in the correct numerical order (1-2-3...). The CCTT-2 requires connecting circles numerically while switching between colored numbers. An Interference Index is obtained that rates the effect of interference in processing time needed to complete one task (CCTT2) that is more complex than another (CCTT1). The CCTT2 interference score was the primary dependent variable used for this measure.

Planning was measured using the CANTAB SOC—*Stockings of Cambridge (SOC)*; (Cambridge, 2002) is based on the "Tower of London" test and is a spatial planning task. Two views are shown, each with three colored balls which appear to be in "stockings" or stacks. The individual must repeat the pattern shown in the example view by touching the ball and then touching where the ball is to be moved. The individual's planning ability is based on how quickly and accurately the pattern is imitated. The SOC number of problems solved in minimum number of moves (SOC Min Moves), initial thinking (SOC Initial Thinking) and subsequent thinking (SOC Sub Thinking) were the dependent variables used.

Fluency was measured using the D-KEFS Letter Fluency and Category Fluency (see D-KEFS above)—The *D-KEFS Letter Fluency and Category Fluency Tests* (Dellis et al., 2001) provide information regarding the individual's ability to fluently retrieve words beginning with the same letter, and ability to retrieve lexical items from a designated category, respectively. The DK Letter and DK Category were the dependent variables used.

Vigilance was measured using the IVA VAQ and AAQ—The *IVA Visual Attention Quotient (VAQ) and Auditory Attention Quotient (AAQ)* (Sandford & Turner, 2000) are based on equal weights of Vigilance (inattention), Focus (speed of mental processing), and Speed (reaction time). The VAQ and AAQ were the primary dependent variables used for this measure (see IVA above).

ADHD Symptomology was measured using the *Conners' Parent Rating Scale-Revised* (Short) (CPRS-R:S) (Conners, 2001). The CPRS-R:S provides information about behaviors associated with attention and/or hyperactivity as well as oppositional behavior. The CPRS was used as an index of ADHD symptoms rather than for diagnostic purposes. The four domain Standard Scores were used as dependent measures and the ADHD Index (C-ADHD) was used as an index of symptomology.

2.4. Statistical analysis

Statistical analyses were performed using SPSS® (Narasis, 1993). As a first step, we did a multivariate analysis of variance, while covarying for IQ (MANCOVA) on the six EF domains. Secondly, of those results that were significant, independent Analyses of Variance (ANOVAs) were conducted on the dependent measures. The partial eta square was reported as an index of effect size. Subsequently, we conducted post hoc multiple pairwise comparisons using the Tukey HSD to control for overall Type 1 error and to determine what group comparisons were significant across the variables.

Simultaneously, we investigated the extent to which ADHD symptoms predict attention and EF performance across these groups using linear and stepwise multiple regression modeling. The predictor variables entered into the model were C-ADHD, diagnosis, IQ and age and the criterion variables were the EF variables previously shown to be statistically significant using ANOVA. We predicted that measures of vigilance and behavioral inhibition would be more predictive of ADHD across the groups. Exploratory analyses were conducted with MANCOVA excluding children with combined ASD and ADHD.

3. Results

Descriptive statistics for the 54 participants across the three groups are presented in Table 1. Chi square analysis demonstrated that the three groups did not differ relative to gender $\chi^2(2, N=54) = 14.52, P < 0.05$. Univariate ANOVA demonstrated that the three groups did not differ relative to age $F(2,51) = 0.04, P > 0.10$. There was a significant difference in Full Scale IQ $F(2,51) = 6.38, P < 0.005$. Post hoc planned comparisons revealed that this difference was due to the ASD group being significantly lower than both the ADHD $t(1,34) = 2.13, P < 0.05$ and TYP $t(1,34) = 3.31, P < 0.01$ groups. Subsequently, IQ was used as a covariate in the analyses.

The means and standard deviations and posthoc multiple pairwise comparisons (Tukey HSD) for the Conners Parent Rating Scale are reported in Table 2. The average range on this measure is defined by T scores from 40 to 60. ADHD was defined by a score greater than 1.5 standard deviations above the mean (Conners, 2001). Based on this criteria, all of the ADHD children met criteria, 8 of the ASD children met criteria, and none of the typical children met criteria.

The results of the MANCOVAs for each domain are presented below. The means and standard deviations and the results of the ANCOVAs of the EF measures grouped by domains are presented in Table 3. Posthoc analyses for each domain are presented below. Due to the concern regarding the possible effects of medication, analyses were conducted comparing the clinical groups between those with and without medication and there were no significant differences (all F 's < 1.0 and $P > 0.05$).

3.1. Response inhibition

Based on MANCOVA, there was a significant difference between the groups regarding Inhibition $F(6,56) = 3.99, P < 0.005$; Wilks' lambda = 0.548. Post hoc multiple pairwise comparisons (Tukey HSD) for the VRCQ showed significant differences between the ASD and TYP group and ASD and ADHD $F(2,51) = 5.33, P < 0.01$. The ARCQ revealed significant

differences between the ADHD and TYP group $F(2,51) = 4.59, P < 0.01$ and between the ASD and TYP groups $F(2,51) = 5.36, P < 0.01$. The ASD group demonstrated the lowest performance, followed by the ADHD group and then the TYP group. There were no differences between the ADHD and TYP groups on the DK-INH, but the ASD group performed significantly lower than the TYP group $F(2,34) = 6.20, P < 0.01$.

3.2. Working memory

The MANCOVA was significant between the groups for Working Memory $F(6,92) = 2.67, P < 0.05$, Wilks' $\lambda = 0.726$. There were significant differences between the ASD and TYP groups for SSP $F(2,48) = 4.72, P < 0.05$, and for SWM Btwn Errors $F(2,48) = 3.95, P < 0.05$, and SWM Strategy $F(2,48) = 3.97, P < 0.05$. There were significant differences between the ADHD and ASD children for SWM Btwn Errors $F(2,48) = 3.95, P < 0.01$ and SWM Strategy $F(2,48) = 3.97, P < 0.05$ with the ASD group performing more poorly. Regarding the SSP, there was a significant difference between the ADHD and TYP group $F(2,51) = 4.72, P < 0.05$.

3.3. Flexibility/Switching

There were significant differences across the groups for Switching $F(6,54) = 3.18, P < 0.01$; Wilks' $\lambda = 0.546$. For DK T-Switch, significant differences were found between the ADHD and ASD $F(2,33) = 7.56, P < 0.01$ and between the ASD and TYP group, $P < 0.001$. There were no significant differences for the CCTT2 or the ID/ED tasks across the groups.

3.4. Planning

There were no significant differences between the groups based on MANCOVA for Planning $F(6,56) = 1.73, P > 0.05$, Wilks' $\lambda = 0.79$, which included SOC Min Moves, SOC Initial Thinking and SOC Subsequent Thinking.

3.5. Fluency

There were no significant differences between the ASD and TYP groups for Fluency $F(4,58) = 2.38, P > 0.05$; Wilks' $\lambda = 0.738$, which included the DK-Letter and DK-Category measures.

3.6. Vigilance

Based on MANCOVA, there were significant differences in Vigilance between the groups $F(4,98) = 4.63, P < 0.01$; Wilks' $\lambda = 0.707$. Subsequently, significant differences were found between ADHD and TYP groups for AAQ $F(2,50) = 8.28, P < 0.001$, and VAQ $F(2,50) = 5.58, P < 0.01$ with the ADHD group performing more poorly. There were significant differences between the ASD and TYP groups on the IVA for AAQ $F(2,50) = 8.28, P < 0.001$, and VAQ $F(2,50) = 5.58, P < 0.01$.

3.7. Prediction and Exploratory analysis

Next, we used stepwise multiple regression to examine the relationship between the dependent variables and behavioral indices of ADHD symptoms. Stepwise multiple regression showed that the ADHD index (C-ADHD) was the first to enter the equation for AAQ explaining 28.5% of the variance ($t = 10.09, P < 0.001$), when IQ was also entered into the model it predicted 40.7% of the variance ($t = 3.24, P < 0.01$). For VAQ, the C-ADHD explained 27.5% of the variance individually ($t = 9.41, P < 0.05$) and when IQ ($t = 4.57$) and age ($t = 3.96$) were entered into the equation, 56.3% of the variance was explained (both $P < 0.05$). For D-KEFS Inhibition, diagnosis ($t = 13.30$) along with the ADHD index ($t = 2.47$) explained 35.4% of the variance ($P < 0.05$). Using linear regression analysis, the ADHD index predicted 16.1% and 11.1% of

the variance for ARCQ ($t=9.27$, $P<0.001$) and VRCQ ($t=7.96$, $P<0.05$), respectively. The ADHD index was not predictive for the remaining variables.

Using an exploratory approach we investigated the influence of ADHD within the ASD group by removing these subjects. The ASD group was divided into those without ADHD ($N=10$) and with ADHD ($N=8$) based on the ADHD index (≥ 65), and MANCOVAs were conducted excluding the ASD/ADHD participants on domains which more consistently discriminate ADHD group; namely Inhibition (ARCQ, VRCQ) and Vigilance (AAQ, VAQ). The MANCOVA with all subjects included was highly significant for Inhibition ($F(4,98) = 3.81$, $P = 0.006$, Wilks' lambda = 0.75). However, the exploratory MANCOVA without the ASD/ADHD group fell to trend level $F(4, 82)=2.24$, $P=0.07$; Wilks' lambda = 0.81 (See ARCQ Figure 1a) suggesting that the ASD/ADHD comorbid group significantly contributed to differences in these domains. The original Vigilance MANCOVA $F(4,98) = 4.63$, $P<0.0001$; Wilks' lambda = 0.71 conducted without the ASD/ADHD group was reduced but remained statistically significant $F(4,82) = 4.241$, $P=0.004$; Wilks' lambda = 0.687 suggesting that the results could not entirely be explained by ADHD symptoms (See AAQ Figure 1b). It is important to note, however, that this approach resulted in unequal groups and a smaller sample size, which reduced the power to detect differences.

4. Discussion

The overarching goal of this investigation was to profile EF deficits for two major childhood disorders, ASD and ADHD, compared to children with typical development while not controlling for ADHD symptoms. The initial aim was accomplished by assessing performance using a comprehensive neuropsychological battery of EF measures across six domains (response inhibition, vigilance, working memory, flexibility/shifting, planning and fluency). We confirmed our hypothesis that children with ASD demonstrate pervasive impairment across a broad range of EF tasks. Specifically, children with ASD showed poor performance relative to the typical group in inhibition, working memory, flexibility/shifting and vigilance. The ASD performed more poorly than the ADHD group in regards to inhibition, working memory, and flexibility. There were no significant differences observed on measures of planning and fluency across the groups. As can be seen in Table 3, the ASD group consistently showed more impairment than the control or ADHD group on all of the aforementioned EF measures. Thus, the current investigation supports previous findings that children with ASD demonstrate generalized and profound impairment in EF skills (Geurts et al., 2004;Goldberg et al., 2005). It is also consistent with recent studies in autism reporting working memory deficits (Goldberg et al., 2005;Landa & Goldberg, 2005;Pennington & Ozonoff, 1996;Verte et al., 2006), as well as set-shifting deficits (Hughes, 1994;Ozonoff et al., 2004;Ozonoff & Strayer, 1997;Sergeant et al., 2002). The finding that children with ASD performed more poorly than children with ADHD on measures of flexibility has also been previously reported (Geurts et al., 2004). Conversely, it has been shown that some ASD subjects have less severe and persistent EF deficits than ADHD children (Happé et al., 2006). It appears, however, that the majority of the ASD subjects had Asperger syndrome (81%) compared to the current study in which the majority had autism and few ASD participants had Asperger syndrome (17%). Thus, developmental and diagnostic issues likely serve as important distinctions in EF within ASD.

The ADHD group exhibited deficits in vigilance and response inhibition when compared to the TYP group corroborating our previous findings (Corbett & Constantine, 2006) and consistent with a meta-analytic review showing these as the most consistently reported domains of executive dysfunction in ADHD (Willcutt et al., 2005). Other comparative investigations report similar and specific deficits in inhibition (Geurts et al., 2004; Happé et al., 2006; Pennington & Ozonoff, 1996; Verte et al., 2006). Our ADHD group also showed some impairment in working memory, however, they did not show statistically significant deficits

in the remaining areas of EF. The current findings are in contrast to previous studies, which found impairments in working memory, planning, and attentional set-shifting (Kempton et al., 1999; Rhodes et al., 2005, 2006; Vance et al., 2003). Although the sensitivity of some of the measures may be called into question (Goldberg et al., 2005), the results in the current investigation are consistent with the notion that children with ADHD demonstrate variable deficits on neuropsychological measures of EF (Doyle et al., 2000; Pennington & Ozonoff, 1996; Rhodes et al., 2005, 2006; Vance et al., 2003; Willcutt et al., 2005). Further, it was shown that such variability, as in our own investigation, is not attributed to medication (Doyle et al., 2000).

While EF deficits are associated with ADHD, they appear to be merely part of the etiology contributing to the complex cognitive and behavioral profile (Willcutt et al., 2005). In consideration of the heterogeneity, more recent neuropsychological models are emerging suggesting that there may be additive or interactive effects arising from multiple neural networks contributing to the complexity of the symptom profile of ADHD (e.g., (Nigg et al., 2005; Sergeant et al., 2003; Sonuga-Barke, 2005)). Further, it has been suggested that studies of both ADHD and autism need to take into account the overlapping symptoms of these neurodevelopmental disorders (Verte et al., 2006); thus, a more dimensional (symptom profile) rather than a categorical approach (diagnostic grouping) may be warranted (Frazier et al., 2007).

Thus, the next aim of the study was to examine the relationship of ADHD symptoms to the neuropsychological measures. We hypothesized that ADHD symptoms would predict task performance on measures of vigilance and behavioral inhibition across the groups, and we confirmed our hypothesis. The results suggest that symptoms of ADHD are associated with poor performance on measures of visual and auditory vigilance and response inhibition as previously reported (Corbett & Constantine, 2006). Further, exploratory analysis excluding the ASD/ADHD children from the analysis provided support that symptoms of ADHD are especially associated with deficits in inhibitory control. Taken together, the results indicate that symptoms of ADHD are associated with inattention and inhibition deficits across the groups (Verte et al., 2006). The finding supports the utility of the IVA (Sandford & Turner, 2000) as a neuropsychological tool in identifying symptoms of poor vigilance and inhibitory control within and across neurodevelopmental disorders, including ADHD and ASD. These results also support the inclusion of a diagnostic parent report measure, such as the Conners (Conners, 2001) in an assessment battery as being able to assist in reliably capturing and classifying children with symptoms of ADHD in ASD.

The observed deficits in vigilance and inhibition in our ASD group provide additional evidence that a significant number of children with ASD present with ADHD-like cognitive impairments (Corbett & Constantine, 2006; Goldstein & Schwebach, 2004; Happe et al., 2006). Our results differ from some previous investigations (Goldberg et al., 2005) that do not report significant differences in vigilance and inhibition in ASD. The lack of replication may be due to distinctions in subject inclusion criteria. Goldberg (Goldberg et al., 2005) excluded subjects with ASD who had ADHD features, while we did not. This suggests that these deficits in our ASD group were due to comorbid ADHD symptoms, rather than fundamental deficits of autism. This notion is supported by the relatively high CPRS scores of our ASD subjects, two-thirds of whom fell in the at risk range or above on this measure.

Whether symptoms of ADHD seen in children with ASD represent an “ADHD-like” disorder unique to autism or represent a distinct co-occurring ADHD may have important treatment implications. It has been proposed that children with both sets of symptoms are more impaired functionally and may respond differently to treatment (Arnold et al., 2006; Ghaziuddin et al., 1992; Goldstein & Schwebach, 2004; Kadesjo & Gillberg, 2001; Posey et al., 2006).

Pharmacologic treatment reports for ADHD symptoms in children with ASD are mixed, with older studies suggesting poor results (Quintana et al., 1995) and more recent studies suggesting benefit in a subgroup of children (Stigler et al., 2004). Thus, the identification of subtype treatment predictors for medications and for cognitive and behavioral interventions may be helpful in treatment effectiveness and side effect avoidance. Future studies should determine if there is a subgroup of children who have ASD with ADHD symptoms that are more likely to respond to particular interventions.

4.1. Limitations

Despite these findings, there are important limitations to report. It is unclear if the current sample of subjects is truly representative of most children with ASD or ADHD. It is possible that some families enrolled the children with ASD in the study knowing that an investigation of ADHD was underway. As such, we may have enrolled a higher proportion of children with ADHD symptoms within ASD. Even so, recent reports showing the notable preponderance of ADHD symptoms in ASD (Corbett & Constantine, 2006; Gadow et al., 2004; Ghaziuddin et al., 1992; Goldberg et al., 2005; Goldstein & Schwebach, 2004; Happe et al., 2006) enhance the generalizability of our findings.

In addition, our investigation included a rather small sample size and multiple data points. In particular, the exploratory analysis was conducted with very few subjects and uneven groups; thus, it must be interpreted with extreme caution until the findings are replicated in larger samples. For clinical utility, we chose to use standardized instruments rather than conducting a factor analytic study merging domains of functioning; thus, there is an increased chance of Type 1 error. Our compromise was to conduct multivariate analysis followed up by analysis of variance on individual subtests. Further, we were unable to collect a medication naïve sample for this investigation. In an attempt to control for this, stimulant medication was withheld for 24 hours, which is a sufficient washout period (Greenhill, (1998)). Some investigations have shown benefit from stimulant medication on EF measures e.g., (Kempton et al., 1999; Vance et al., 2003), while other recent studies show modest or a lack of benefit on EF measures following stimulant use (Coghill et al., 2007; Rhodes et al., 2006). Other medications were few (ADHD = 3, ASD = 4) and evenly distributed across the two disorder groups. Even so, we conducted analyses across the clinical groups comparing those with and without medication and the results remained the same. Despite these efforts, we acknowledge that there may have been some modest effects on the results attributed to medication use in some of the participants. Finally, contradictory findings across studies may be, in part, explained by differences in inclusion criteria, age, level of functioning and task demands, which inadvertently contribute to inconsistencies in the literature.

4.2. Conclusion

Due to the converging evidence reporting a high prevalence of ADHD in ASD, a serious reconsideration is necessary regarding the current diagnostic practice of not providing a diagnosis of ADHD within a pervasive developmental disorder when appropriate (APA, 2000). The issues and challenges of diagnosing ADHD, especially within special populations, must be carefully considered (Barkley, 2003). The presence of symptoms from more than one neurodevelopmental disorder likely leads to exponential risks and greater treatment challenges. Additional research is needed to help delineate patterns of performance within and between ADHD and ASD. This comorbid profile of ADHD in ASD may represent a distinct phenotype in autism that requires further study. Neuropsychological models are beginning to emerge that could account for the heterogeneity of ASD and ADHD. Across both disorders there may be an additive or interactive effect stemming from dysfunction from various neural networks that contributes to the heterogeneous profile (e.g., (Castellanos et al., 2006; Nigg et al., 2005; Sergeant et al., 2003; Sonuga-Barke, 2005)). It may also be the case that a more dimensional

approach to understanding neurodevelopmental disorders is warranted (Frazier et al., 2007). It is our hope that elucidating the overlap and distinctions between ASD and ADHD profiles will enable clinicians and researchers to better assess, characterize and treat these complex disorders.

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Figure 1.

Figures 1a and 1b. Scatterplots of the relationship between the C-ADHD (Conners ADHD Index) and the 1a. ARCQ (Auditory Response Control Quotient) and 1b. AAQ (Auditory Attention Quotient) across the groups: TYP = Typical, ADHD = ADHD, ASD = Autism Spectrum Disorder, and ASD/ADHD = Autism Spectrum Disorder with ADHD.

Table 1
Means and Standard Deviations for the Demographic Variables.

Variable	TYP		ADHD		ASD		F	p	Eta-squared
	M	(SD)	M	(SD)	M	(SD)			
Age	9.56	(1.81)	9.40	(1.98)	9.44	(1.96)	0.04	0.966	0.00
IQ	112.22	(14.84)	105.17	(12.82)	94.17	(17.79)	6.38	0.003	0.20

Note. TYP = 18 (12 males, 6 females), ADHD = 18 (12 males, 6 females), ASD = 18 (17 males, 1 female).

Table 2
Means and Standard Deviations for the Conners' Diagnostic Measure.

Variable	TYP		ADHD		ASD		F	p	Eta-squared
	M	(SD)	M	(SD)	M	(SD)			
C-OPP	48.78	(7.57)	65.89	(13.85)	55.78	(9.36)	11.87	0.000	0.32
C-INA	46.06	(6.83)	72.33	(9.27)	61.44	(8.45)	46.18	0.000	0.64
C-HYP	47.56	(5.70)	79.33	(10.53)	61.11	(12.94)	44.16	0.000	0.63
C-ADHD	45.72	(5.02)	76.22	(5.22)	64.78	(6.86)	128.96	0.000	0.83

Note. TYP = 18 (12 males, 6 females), ADHD = 18 (12 males, 6 females), ASD = 18 (17 males, 1 female), C = Conners Parent Rating Scale-Revised, C-OPP = Oppositional, C-INA = Inattention, C-HYP = Hyperactivity, ADHD = Conners' ADHD Index.

Table 3
Means and Standard Deviations and Analysis of Variance for the Executive Functioning and Attention Measures.

Measure	TYP		ADHD		ASD		p
	M	(SD)	M	(SD)	M	(SD)	
<u>Eta-squared</u>							
Response Inhibition							
VRCQ	94.50	18.37	83.28	21.96	65.33	24.90	<i>a,c</i> 0.33
ARCQ	91.56	19.29	72.89	18.38	67.44	19.75	<i>a,b</i> 0.21
DK INH	10.31	3.66	11.27	2.53	5.80	2.20	<i>a</i> 0.29
Working Memory							
SSP	5.59	1.37	4.67	1.14	3.94	1.39	<i>a,b</i> 0.16
SWM Bwn Errors	41.88	19.94	45.94	21.59	63.18	10.57	<i>a,c</i> 0.14
SWM Strategy	34.35	5.77	35.28	4.11	38.59	2.67	<i>a,c</i> 0.15
Cognitive Flexibility							
ID/ED-Shift	25.08	10.90	25.25	11.88	23.44	10.58	ns 0.01
DK T-Switch	11.83	2.73	12.75	3.36	7.22	4.18	<i>a,c</i> 0.34
CCTT2	96.67	15.13	86.58	18.72	74.56	18.19	ns 0.14
Planning							
SOC Min Moves	6.94	1.92	6.28	2.42	5.06	2.21	ns 0.06
SOC Initial Thinking	0.85	0.38	1.11	0.55	0.67	0.48	ns 0.13
SOC Sub Thinking	-0.15	0.75	-0.26	1.06	-0.49	0.64	ns 0.01
Fluency							
DK Letter	10.92	1.98	10.83	3.95	6.22	4.30	<i>a,c</i> 0.23
DK Category	12.08	2.72	11.83	4.24	8.44	2.65	ns 0.14
Vigilance							
VAQ	91.56	22.15	63.00	22.16	61.67	24.75	<i>a,b</i> 0.18
AAQ	94.39	21.23	65.12	19.17	64.06	20.04	<i>a,b</i> 0.25

^aNote: ASD vs. Typical, $p < 0.05$;

^bADHD vs. Typical, $p < 0.05$;

^cASD vs. ADHD, $p < 0.05$; ns, $p > 0.05$.

* N varies depending on test. VRCQ = Visual Response Control Quotient, ARCQ = Auditory Response Control Quotient, DK = D-KEFS, DK INH = Inhibition, SSP = Spatial Span, SWM = Spatial Working Memory, ID/ED-Shift = Extradimensional Shift errors, DK T-Switch = Total Switching, CCTT2 = Children's Color Trails Trial 2, SOC Min Moves = Stockings of Cambridge Minimum Moves, SOC Initial Thinking= Stockings of Cambridge Initial Thinking Time; SOC Sub Thinking= Stockings of Cambridge Subsequent Thinking Time; DK Letter = Letter Fluency, DK Category = Category Fluency, VAQ = Visual Attention Quotient, AAQ = Auditory Attention Quotient.