Intellectual Deficits in Children with ADHD Beyond Central Executive and Non-Executive Functions†

Carin M. Tillmana,*, Gunilla Bohlina, Lin Sørensenb,c, Astri J. Lundervoldb,c

aDepartment of Psychology, Uppsala University, Uppsala, Sweden
bDepartment of Biological and Medical Psychology, University of Bergen, Bergen, Norway
cCentre for Child and Adolescent Mental Health, University of Bergen, Bergen, Norway

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Abstract

This study aimed to specify the deficit in intellectual ability in children with attention deficit hyperactivity disorder (ADHD), by studying the mediating role of impairments in central executive function (EF)-related components (working memory, inhibition, sustained attention) and non-EFs (short-term memory and processing speed). Two hundred and thirty children aged 8–11 years from a population-based sample were assigned to either the ADHD group, the clinical comparison group, or the normal comparison group. The results showed that children with ADHD had poorer fluid and crystallized intelligence, relative to both comparison groups. Further, regarding fluid intelligence, these deficits were not fully mediated by, but rather went beyond, poorer functioning on the studied EF-related components and non-EFs. We tentatively interpret these fluid deficits in children with ADHD as representing deficiencies in a general intellectual resource reflecting executive attentional processes. Concerning crystallized ability, in contrast, the deficit signified impairment in the studied cognitive functions, as indicated by the significant full mediation effect.

Keywords: ADHD; Intelligence; Executive functions (EF)

Introduction

Children and adolescents with attention deficit hyperactivity disorder (ADHD) have been shown to have impaired cognitive functions, including executive function (EF) components (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) and general intelligence (Fraizer, Demaree, & Youngstrom, 2004). Given that diverse EFs are also related to intelligence (Ackerman, Beier, & Boyle, 2005; Dempster, 1991; Salthouse, Atkinson, & Berish, 2003; Tillman, Bohlin, Sørensen, & Lundervold, in press), there are empirical interrelations between the three phenomena ADHD, intelligence, and EFs. This raises the question whether poor intellectual functioning in children with ADHD is mediated by specific cognitive deficits.

The concept of intelligence in the ADHD literature is part of the discussion of whether intellectual function should be statistically controlled for when examining possible etiological factors (Dennis et al., 2009). The literature that has focused more directly on intellectual function in children with ADHD generally reports impairment when the children are compared with controls (e.g., Barkley, 1997; Barkley, DuPaul, & McMurray, 1990; Fraizer et al., 2004; Goldstein, 1987a, 1987b; Kuntsi, Eley, Hughes, Asherson, Caspi, 2004; Loney, 1974; Mariani & Barkley, 1997; McGee, Williams, Moffitt, & Anderson, 1989; Sonuga-Barke et al., 2008), and a twin study by Kuntsi and colleagues (2004) suggested that the association between ADHD and lower IQ was mainly accounted for by shared genetic influences. Results from a meta-analysis concluded that

† Inter-correlations between the cognitive variables used in this study have been reported for descriptive purposes in a previous article (Tillman, Bohlin, Sørensen, & Lundervold, in press). The two manuscripts are otherwise concerned with different research questions.

* Corresponding author at: Department of Psychology, Uppsala University, PO Box 1225, SE-751 42 Uppsala, Sweden. Tel.: +46-18-471-21-03; fax: +46-18-471-21-23.

E-mail address: carin.tillman@psyk.uu.se (C.M. Tillman).

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children and adolescents with ADHD show as large (or even larger) intellectual as EF deficit (Fraizer et al., 2004). However, in a majority of the studies included in this meta-analysis, EF was measured using tasks that were also included in the measure of intelligence (e.g., the Digit Span task), preventing conclusions about which phenomenon was more affected. Nevertheless, based on their results, Fraizer and colleagues (2004) proposed that poorer intelligence should be seen as a “feature of the disorder” (p. 552). However, the authors did not explicitly define what they meant by “feature of the disorder”. Poorer intelligence in children with ADHD has elsewhere been suggested to be a direct effect of the symptoms of the disorder, to reflect impairments in working memory (WM), or to constitute a secondary symptom of the disorder (see e.g., Dennis et al., 2009; Nigg, 2006).

Deficits in central EF-related components such as inhibitory control functions, WM, and sustained attention are suggested to be some of the more specific neuropsychological disadvantages of children and adolescents with ADHD (Willcutt, Doyle et al., 2005). However, for sustained attention, the ADHD literature is rather inconsistent, regarding both operationalizations and results. According to Nigg (2006) as well as van der Meere and Sergeant (1988), sustained attention should be operationalized as performance deteriorations over the time course of a particular task (preferably a Continuous Performance Task [CPT]). Nigg (2006) has suggested that this type of attention deficit, measured in this way, is spared in most cases of ADHD. However, Swaab-Berneveld and colleagues (2000) found performance over time on a CPT to be the only measure differentiating between children with and without ADHD, and similar performance decrements were also reported by Epstein and colleagues (2003). It should also be mentioned that the categorization of sustained attention as being related to EF or not may be less straightforward compared with inhibition or WM. Barkley (1997) maintains that EF sets the stage for sustained attention, indicating that this attention function is related to EF (see also Miller & Cohen, 2001), whereas Nigg (2006) suggests that sustained attention is a non-EF. We have chosen to classify sustained attention as one of the EF-related components in this study.

In addition to these EF-related components, more basal non-executive cognitive functions, such as processing speed (e.g., Cinnamon Bidwell, Willcutt, DeFries, & Pennington, 2007) and short-term memory (STM; Paule et al., 2000), have been suggested to be affected in children with ADHD, although the evidence for these dysfunctions are somewhat weaker than for EFs. It should be noted that poorer cognitive functions being associated with ADHD is not only supported by group differences found between ADHD and comparison groups but also by relatively strong relations using dimensional methods in normal samples (e.g., Berlin & Bohlin, 2002; Brocki & Bohlin, 2006; Kuntsi, Andreou, Ma, Borger, & van der Meere, 2005; Sonuga-Barke, Dalen, Daley, & Remington, 2002). Relations have been found primarily with the inattention symptom domain, rather than the hyperactive/impulsive symptom domain (e.g., Chhabildas, Pennington, & Willcutt, 2001; Martel, Nikolas, & Nigg, 2007; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005).

Interestingly, the EF-related components and non-EFs discussed earlier have not only been linked to ADHD but also to intelligence in both children and adults (Ackerman et al., 2005; Colom, Flores-Mendoza, Quiroga, & Privado, 2005; Dempster, 1991; Kail & Salthouse, 1994; Salthouse et al., 2003; Tillman et al., in press; Tillman, Nyberg, & Bohlin, 2008). However, the literature on the relations between specific EF-related components and intelligence is not conclusive. In their study on adults, Friedman and colleagues (2006) demonstrated that out of the EFs mental set-shifting, inhibition, and updating of WM, updating of WM was the only function showing an independent relation to intelligence. In contrast, Tillman et al. (in press) reported that several cognitive functions, including inhibitory functions and WM, were independently related to intelligence in children.

Although somewhat inconsistent, the literature thus indicates that the three phenomena ADHD, intelligence, and more specific cognitive functions (particularly EFs) are empirically interconnected. It should be noted that these phenomena have mostly been studied in bivariate relations, leaving open the potential mediating role of specific cognitive functions for the association between ADHD and impaired intellectual function. However, a study by Rommelse and colleagues (2008) is relevant to this issue. The results indicate that ADHD-related deficits in intelligence are not fully accounted for by the poorer WM and inhibitory abilities exhibited by these children. In line with this, Biederman and colleagues (2004) demonstrated that a group of children characterized by having both ADHD and EF deficits showed significantly poorer intelligence compared with a group of children having only EF deficits and not ADHD. Thus, these two studies suggest that there is something more to intellectual deficits in children with ADHD than poor EF. For a more conclusive answer, further studies are needed that encompass several cognitive functions, including non-EFs, that have been shown to be related to both ADHD and intelligence (e.g., Cinnamon Bidwell et al., 2007; Paule et al., 2000; Tillman et al., in press; Tillman et al., 2008; Willcutt, Pennington et al., 2005). Owing to comorbidity being the rule rather than the exception in cases of ADHD (e.g., Biederman, 2005; Schatz & Rostain, 2006), it is also important to be able to show clinical specificity of ADHD, an issue that has been overlooked in previous studies of the relation between EF deficits and intelligence in persons with ADHD (Biederman et al., 2004; Rommelse et al., 2008).

Following Cattell’s (1963) theory on intelligence, the complex concept of intellectual function should be further divided into fluid and crystallized subcomponents. Fluid intelligence, which is related to the solving of abstract-reasoning problems,
refers to the potential to adapt to new situations and form new ideas that depend on the ability to perceive relations and correlations. Crystallized abilities are composed of information that is acquired through education, experience, and socialization. Given the discrepancy in nature of these two intelligence types, it is crucial to investigate whether they would show different patterns of deficits in children with ADHD. The Verbal and Performance IQ scales as well as the Verbal Comprehension and Perceptual Organization Factor scores from the Wechsler (1992) scales may be used to define these two intelligence types. Several studies of ADHD have included these separate scales (e.g., Cook, Stein, Ellison, Unis, & Leventhal, 1995; Mayes & Calhoun, 2006; Nydén, Billstedt, Hjelmquist, & Gillberg, 2001; Shin & Lee, 2007; Yang, Jong, Chung, & Chen, 2004), but few have included normal controls. These few studies are included in the meta-analysis by Fraizer and colleagues (2004), suggesting that children with ADHD are impaired on both intelligence types, with some indications of a more severe deficit in verbal/crystallized intelligence (Chhabildas et al., 2001; Rommelse et al., 2008).

The Present Study

On the basis of findings suggesting that there are empirical interrelations between the three phenomena ADHD, intelligence, EF as well as some non-EFs, we aimed to specify the deficit in intellectual function in children with ADHD. By adopting a mediation perspective, we evaluated the strength of the “direct” effect that ADHD has on intelligence, as well as its “indirect” effect through potential mediators. A general illustration of the notion of direct and indirect effects is presented in Fig. 1a and b. This would yield information about whether or not the intellectual deficit in children with this disorder goes beyond deficits in specific EF components (WM, inhibition, and sustained attention) and non-EFs (STM and processing speed). Intellectual ability was evaluated in terms of both fluid and crystallized intelligence. The cognitive functions were studied separately as

![Fig. 1. Schematic illustration of the different types of mediation effects investigated in the present study. (a) Simple mediation effect; (b) multiple mediation effect. IV = independent variable; Med = mediator; DV = dependent variable.](https://academic.oup.com/acn/article-abstract/24/8/769/3240/Intellectual-Deficits-in-Children-with-ADHD-Beyond/771)
well as together as potential mediators of intellectual function in children with ADHD. Performance of children diagnosed with ADHD was compared with that of children with other psychiatric diagnoses as well as children with no psychiatric diagnosis. On the basis of previous literature, we hypothesized that children with ADHD would have significantly poorer intelligence compared with the normal comparison group. On the basis of the extensive literature indicating strong relations between the three phenomena of ADHD, intelligence, and specific cognitive functions, we further expected to find significant partial mediation effects (i.e., significant indirect effects of ADHD on intelligence through the EFs and non-EFs). However, in that this issue is still explorative in nature, we did not state any predictions of whether or not there would be a full mediation effect (i.e., whether the direct effect would be significant). A full mediation (i.e., no significant direct effect) would indicate that deficits in intelligence do not go beyond deficits in the specific cognitive functions and thus that the intellectual deficits of these children cannot reveal any information additional to that provided by the specific cognitive deficits. In contrast, if only partial mediation were to be obtained, this would indicate that the deficits in intelligence would carry information over and above that carried by the specific cognitive functions. Inclusion of a clinical comparison group allowed us to study whether intellectual deficits were specific to children with ADHD rather than a correlate of psychiatric disorders in general. We also more specifically considered the role of comorbidity with other externalizing disorders as well as internalizing disorders. It should be noted that a categorical approach of studying ADHD will be adopted (i.e., presence of diagnosis vs. absence of diagnosis), and our results and interpretations will consequently only apply to this view.

Materials and Methods

Participants

The present study is part of the Bergen Child Study (BCS), a large Norwegian longitudinal population-based study of children attending 2nd to 4th grade (7–9 years of age) in schools in the Bergen (n = 9430) and Sund (n = 222) municipalities in October 2002. The protocol and population of the BCS have been described in detail in separate publications (Heiervang et al., 2007; Posserud, Lundervold, & Gillberg, 2009; Stormark, Heiervang, Lundervold, Heimann, & Gillberg, 2008), and only a short description will be given here. The first wave of the BCS included three stages. Stage 1: A screening questionnaire for behavior problems and psychiatric disorders was sent to parents and teachers of the whole 7–9-year-old population. The questionnaire included, among other instruments, the DSM-IV diagnostic criteria for ADHD and ODD (SNAP-IV; Swanson et al., 2001). Stage 2: Parents of all screen positive children (i.e., children with high scores on the questionnaire in Stage 1) and a random sample of screen negative children were interviewed according to the semi-structured development and well-being assessment (Goodman, Ford, Richards, Gatward, & Meltzer, 2000). Stage 3: Approximately 18 months after the Stage 1 questionnaire, 329 children together with their parents participated in an examination including a psychiatric diagnostic interview (the Kiddie-Sads-Present and Lifetime Version [K-SADS-PL]; Kaufman et al., 1997) and a neuropsychological test battery. In Stage 3, children were between 8 and 11 years old. The K-SADS-PL is a reliable semi-structured diagnostic interview designed to evaluate current and past episodes of psychopathology in children according to the DSM-IV criteria (Ambrosini, 2000; Kaufman et al., 1997). Diagnoses are scored as either definite, probable, in remission, or not present. A probable diagnosis is close to a definite diagnosis in symptom expression, that is, the child’s behavior fulfills all the diagnostic criteria except that carried by the specific cognitive functions. Inclusion of a clinical comparison group allowed us to study whether intellectual deficits were specific to children with ADHD rather than a correlate of psychiatric disorders in general. We also more specifically considered the role of comorbidity with other externalizing disorders as well as internalizing disorders. It should be noted that a categorical approach of studying ADHD will be adopted (i.e., presence of diagnosis vs. absence of diagnosis), and our results and interpretations will consequently only apply to this view.

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One hundred and nine (48%) of the children in the present sample received a definite psychiatric diagnosis according to the K-SADS-PL interview. Children who received a definite ADHD diagnosis, regardless of the presence of comorbid diagnosis/diagnoses, were assigned to the ADHD group ($n = 45$). Twenty-nine of these children had an ADHD-combined type diagnosis and 16 had an ADHD-inattentive type diagnosis. No children in the ADHD group were taking medication at the time of testing. Children with any definite diagnosis/diagnoses other than ADHD were assigned to the clinical comparison group ($n = 64$). Children with no diagnosis (neither definite nor probable) were assigned to the normal comparison group ($n = 121$). Seventy children (58%) in the normal comparison group had screened negative for mental health problems in Stage 1 (Stormark et al., 2008), and the rest had screened positive but without reaching a diagnostic level of symptoms according to the DSM-IV. There were more boys (64%) than girls in this sample. The age distribution was similar for boys ($M = 9.93$ years, $SD = 0.96$ years) and girls ($M = 9.76$ years, $SD = 0.89$ years). The ADHD group was significantly older than the two comparison groups, $F(2, 226) = 5.06, p < .01$. For more details on the groups, see Table 1. The study was approved by the Regional Committee for Medical and Health Research Ethics in Western Norway and by the Norwegian Data Inspectorate, Ombudsman for the Privacy in Research, Norwegian Social Science Data Services Ltd.

**Cognitive Tasks and Measures**

**Intelligence.** Fluid and crystallized intelligence were assessed by the sum of the age adjusted scaled scores on the Perceptual Organization and Verbal Comprehension Factors of the WISC-III (Wechsler, 1992). These factors do not include any explicit EF tasks (such as the Digit Span task, assessing WM). This is necessary if EF and intelligence are to be evaluated relatively independent of each other. The Verbal Comprehension Factor includes results on the Information, Similarities, Vocabulary, and Comprehension subtests. The Perceptual Organization Factor includes results on the Picture Completion, Picture Arrangement, Block Design, and Object Assembly subtests. It should be noted that these two factors are highly correlated with WISC-III full-scale IQ in this age range. However, as it has been suggested that the cognitive abilities and limitations

<table>
<thead>
<tr>
<th>Table 1. Descriptive data as well as means and standard deviations of the cognitive variables for the ADHD group, clinical comparison group, and normal comparison group</th>
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<tbody>
<tr>
<td><strong>Group</strong></td>
</tr>
<tr>
<td>$n$</td>
</tr>
<tr>
<td>$M (SD)$ age in years</td>
</tr>
<tr>
<td>% girls</td>
</tr>
<tr>
<td>% children with more than one diagnosis</td>
</tr>
<tr>
<td>Specific diagnoses (% children)</td>
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<tr>
<td>ODD</td>
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<tr>
<td>CD</td>
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<tr>
<td>Depressive disorder</td>
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<tr>
<td>Anxiety disorder</td>
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<tr>
<td>Tourettes or chronic tics disorder</td>
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<tr>
<td>Stress disorder</td>
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<tr>
<td>Enuresis</td>
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<tr>
<td>Encopresis</td>
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<tr>
<td>Other psychiatric diagnosis</td>
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<tr>
<td>Cognitive variables</td>
</tr>
<tr>
<td>Fluid intelligence</td>
</tr>
<tr>
<td>Crystallized intelligence</td>
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<tr>
<td>WM*</td>
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<tr>
<td>Inhibition (Stroop)bc</td>
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<td>Sustained attentionbcd</td>
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<tr>
<td>STM*</td>
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<tr>
<td>Processing speed</td>
</tr>
</tbody>
</table>

*Notes:* Specific diagnoses are presented in percent out of the total $n$ in each group. ADHD = attention deficit hyperactivity disorder; ODD = oppositional defiant disorder; CD = conduct disorder; WM = working memory; STM = short-term memory.

*One child was excluded from this group due to being an outlier in the statistical analyses (see the Data Preparation and Statistical Analyses section).

*These variables comprise statistical residuals (see the Data Preparation and Statistical Analyses section).

*Higher values indicate worse performance.

*Owing to missing data, $n$ on this measure was 42 for the ADHD group and 116 for the normal comparison group.
of children with ADHD are best evaluated when separate intelligence factors are considered (e.g., Fiorello, Hale, McGarth, Ryan, & Quinn, 2002), it was regarded as important to separate Perceptual Organization and Verbal Comprehension Factors. The WISC-III is a reliable measure of intellectual ability in children aged 6–16 years. The Verbal Comprehension and Perceptual Organization Factors show split-half reliabilities of .94 and .90, respectively, averaged across all ages. The separate subtests included in these factors show reliability ranging from .69 to .87, demonstrating adequate internal consistency (Wechsler, 1992).

**EF Tasks.** Raw scores on the backward version of the Digit Span subtest from the WISC-III (Wechsler, 1992) were used to assess WM. This WISC-III subtest has a reliability of .85.

Inhibition was measured by interference scores derived from the Stroop task (Stroop, 1935) and the ANT. In the present paper and pencil version of the Stroop task (Lund-Johansen, Hugdahl, & Wester, 1996), the participant is presented with color patches or color words, printed either in compatible or incompatible ink color (e.g., the word “red” is printed in either red or blue) on separate sheets. The participant is instructed to say the ink color of each word or color patch, which produces a response conflict on the trials where the ink color is incompatible with the meaning of the word. Interference scores in the Stroop task were calculated by subtracting the number of errors made in the color patch condition from the number of errors made in the incompatible word-color condition (see Davidson, Amso, Anderson, and Diamond, 2006, for the validity of errors rather than time measures for assessing performance in children).

The ANT used in the present study is the original “child version” (Rueda et al., 2004). The test has four cue conditions (no cue, center, double, orienting) and three flanker conditions (congruent, incongruent, neutral) and has been described in detail elsewhere (Mezzacappa, 2004; Rueda et al., 2004). All combinations of conditions are randomly presented in three blocks of 48 trials each. The participants are instructed to indicate which direction a fish is pointing by pressing the left or right mouse button with their corresponding thumb. Sometimes the fish appears alone, and at other times in the middle of a row of five identical fishes. The participant is told to respond to the fish in the middle of the row. The flanking fish either points in the same direction (congruent) or in the opposite direction (incongruent) as the target fish in the middle. Test–retest reliability of the ANT has been demonstrated to be adequate, .77 (Fan, McCandliss, Sommer, Raz, & Posner, 2002), and error measures were shown to be more sensitive than reaction time measures in an earlier study from the BCS (Adólfsdóttir, Sørensen, & Lundervold, 2008). Interference scores in the ANT were calculated by subtracting the number of errors made in the congruent target-flanker condition from the number of errors made in the incongruent target-flanker condition, across all cue conditions. No interference scores in either the ANT or the Stroop task were negative, which indicates that more errors were made in the congruent conditions than in the incongruent conditions. The ANT and Stroop interference scores were significantly correlated ($r = .15, p < .05$), and they were aggregated after standardization into the inhibitory control composite measure that was used in the analyses, in which higher values indicated poorer inhibition.

Sustained attention was assessed by Conners’ CPT (Conners, 1994), in which participants are instructed to respond to all letters presented on the computer screen, except for the letter X, for 14 min. Split-half reliabilities for the different performance measures on this CPT range between .73 and .95 (Conners, 2000). In addition, test–retest reliabilities for a 3-month interval range between .55 and .84 (Conners, 2000). Both indices suggest adequate reliability for this test. The test is divided into six time blocks, which enables calculation of the slope of change in performance across time on task, in either a positive (i.e., increased values) or negative slope (i.e., decreased values). This has been suggested to be essential if conclusions are to be drawn regarding “sustained” attention (Nigg, 2006; van der Meere & Sergeant, 1988). The measures used in this study to assess sustained attention were standard error of reaction time on hits (RTSE), the slope of change of hit RT, and the slope of change of hit RTSE. These measures were significantly correlated ($r = .45–.63$), and they were aggregated, after standardization, to form the variable sustained attention, where higher values indicate poorer sustained attention.

**Non-executive tasks.** Raw scores on the forward version of the Digit Span subtest of the WISC-III (Wechsler, 1992) were used to assess STM. This WISC-III subtest shows a split-half reliability of .85 (Wechsler, 1992). General processing speed was assessed by the sum of the age-adjusted scaled scores on the Processing Speed Factor of the WISC-III (Wechsler, 1992), comprising results on the Coding and Symbol Search subtests. The Processing Speed Factor has a test–retest reliability of .88 (Wechsler, 1992).

**Data Preparation and Statistical Analyses**

Multivariate outliers, identified by a Cook’s $D$-value higher than 1, were checked in all subsequently described analyses. Through this procedure, one participant in the normal comparison group was found to be an outlier in several multivariate relations and was therefore excluded from all analyses. Thus, the analyses were conducted with 120 children in the normal
comparison group. Data on sustained attention were missing for seven participants. We used age standardized scaled scores on the Verbal Comprehension, Perceptual Organization, and Processing Speed Factors from WISC-III. (Age-adjusted scaled scores for the separate forward and backward versions of the Digit Span subtest of the WISC-III are not available.) Although not all of the correlations between age and the variables that were not age-adjusted reached significance ($r = .01 - .13$), we still wanted to assure that all measures were treated uniformly with regard to potential age-related variance. Thus, to clear the other variables of age-related variance, statistical regression residuals were extracted in each variable by removing the variance shared with child age. These residuals were used in all subsequent analyses.

There was no evidence of heterogeneity of covariance among the variables, Box’s $M = 5.92, p = .44$. All potential mediators and control variables, except inhibition, showed homogeneity of regression, that is, no interactions between mediators and group were significant. For inhibition, the interaction with group was significant for fluid intelligence, $\beta = -.26, t = -4.10, p < .001$. By rescaling the inhibition variable into three response categories (based on the upper, middle, and lower third of the distribution), we achieved homogeneity of regression and this rescaled inhibition variable was used in the analyses of mediation.

Group differences in intelligence were studied using a regression analysis approach with dummy coded predictor variables representing the ADHD group, the clinical comparison group, and the normal comparison group. To evaluate group differences in intellectual abilities over and above those for specific cognitive functions, a mediation perspective was adopted. Mediation was studied following recommendations by Preacher and Hayes (2008). The absence of a significant “direct” effect from predictor variable to outcome variable was interpreted as the presence of a full mediation effect. The presence of indirect effects, indicating partial mediation effects, was investigated with bootstrapping techniques using the SPSS macro downloaded from the webpage accompanying Preacher and Hayes (2008). This bootstrapping procedure allows for testing of partial mediation as well as total mediation effect by multiple mediators. Five-thousand bootstrap resamples were used to calculate the indirect effects. Through this bootstrapping procedure, bias corrected and accelerated confidence intervals for the indirect effect of the predictor variable on the outcome variable, through the mediator(s), are calculated. If these confidence intervals do not include 0, the indirect effect is significant. As a first step in studying the mediation effects of specific cognitive functions in the intellectual deficits in children with ADHD, each one of the EF-related components and non-EFs was entered as a potential mediator in separate regression analyses, which were subjected to the bootstrapping procedure referenced earlier. In the subsequent step of the mediation analyses, we tested for the total effect of multiple mediators by entering all specific cognitive functions at the same time as potential mediators.

In order to evaluate the potential role of comorbidity in the obtained results, all analyses were rerun with the predictors being dummy coded variables representing the presence of ADHD, the presence of other externalizing disorders (ODD/CD), and the presence of internalizing disorders (i.e., depressive and anxiety disorders), as well as the interaction terms between the ADHD variable and each of the two other dummy variables. The role of comorbidity was evaluated both in terms of the significance of the interaction terms, signifying whether or not ADHD combined with another diagnosis would result in a worsened condition than simply adding together the effects of each disorder, and in terms of the contribution made by the ADHD dummy variable independently of the other diagnostic dummy variables, where a significant contribution would indicate that the results could not be explained by the presence of comorbid diagnoses.

**Results**

*Preparatory Analyses*

There were significant sex differences in fluid intelligence, WM, inhibition, STM, and processing speed ($t$ ranging $2.09 - 6.13, p < .05$), with girls performing at a higher level than boys. As a consequence, all subsequent analyses using any of these variables as outcome variable were controlled for sex.

To perform mediation analyses, it is necessary that the predictor variables, mediators, and outcome variables show significant interrelations. As shown in regression analyses using a dummy coded ADHD variable as predictor variable, each of the cognitive variables as outcome variables, and a dummy coded variable representing the clinical comparison group as a covariate, there were significant relations between ADHD and all cognitive functions, except inhibition (fluid intelligence, $\beta = -.33, t = -4.92, p < .001$; crystallized intelligence, $\beta = -.28, t = -4.18, p < .001$; WM, $\beta = -.14, t = -2.07, p < .05$; inhibition, $\beta = .09, t = 1.19, p > .05$; sustained attention, $\beta = .16, t = 2.31, p < .05$; STM, $\beta = -.15, t = -2.21, p < .05$; and processing speed, $\beta = -.26, t = -3.98, p < .001$). As there were no significant group differences on the composite inhibition variable, group differences were tested for the two individual inhibition measures. This showed a significant group difference for Stroop ($\beta = .18, t = 2.54, p < .05$), but not for ANT ($\beta = .09, t = 1.19, p > .05$), which means that only Stroop qualified as a mediator. Therefore, mediation analyses will primarily be performed using the Stroop measure, although
for completeness, results will be reported also for the composite measure. The Stroop measure was rescaled into three categories to achieve homogeneity of regression.

Using predictor variables, dummy coded as ADHD versus normal comparisons and ADHD versus clinical comparisons (corresponding to planned contrasts in analysis of variance), we further showed that the ADHD group performed significantly worse compared with both the normal comparison group and the clinical comparison group on fluid and crystallized intelligence, WM, inhibition (as assessed by the Stroop measure), sustained attention, STM, and processing speed (all \( p < .05 \)). Inter-correlations of the cognitive variables are presented in Table 2. All cognitive functions were significantly related to both fluid and crystallized intelligence. All interrelations of the specific cognitive functions, except for the relation between inhibition (either as assessed by the composite measure or the Stroop measure), and STM and WM, respectively, were significant.

**The Mediating Role of Specific Cognitive Functions in Intellectual Deficits**

Results from the bootstrapping analyses investigating direct and indirect effects of ADHD on intelligence are presented in Table 3 and Fig. 2. Each one of the EF-related components and non-EFs were significant mediators of the relation between ADHD and fluid and crystallized intelligence, respectively, as indicated by the confidence intervals of the indirect effects (Table 3). (Inhibition as assessed by only the Stroop measure was a significant mediator, whereas inhibition as assessed by the composite measure from the Stroop task and ANT was not.) The direct effect between ADHD and intelligence

<table>
<thead>
<tr>
<th>Table 2. Intercorrelations of all cognitive variables</th>
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<tr>
<td>Fluid intelligence</td>
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<tr>
<td>Fluid intelligence</td>
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<td>Crystallized intelligence</td>
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<td>WM</td>
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<td>Sustained attention</td>
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<td>STM</td>
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<td>Processing speed</td>
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Notes: \( n \) ranges from 222 to 229. The correlations presented changed minimally when controlling for group belongingness, indicating that the correlations were similar in the ADHD group, the clinical comparison group, and the normal comparison group. ADHD = attention deficit hyperactivity disorder; WM = working memory; STM = short-term memory.

\( a \)Correlations for the inhibition composite measure are presented within parenthesis.

\( *p < .05 \).

\( **p < .01 \).

\( ***p < .001 \).

<table>
<thead>
<tr>
<th>Table 3. Results from bootstrapping mediation analyses, regressing the ADHD dummy variable on fluid and crystallized intelligence</th>
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<td>Mediator</td>
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<tr>
<td>WM</td>
</tr>
<tr>
<td>Inhibition (Stroop measure)</td>
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<tr>
<td>Inhibition (composite score)</td>
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<td>Sustained attention</td>
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<td>STM</td>
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<td>Processing speed</td>
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Notes: \( n \) ranges from 222 to 229. The presence of another diagnosis than ADHD was controlled for in each analysis by entering a dummy coded variable representing “clinical comparisons/others”. In all analyses with fluid intelligence as outcome variable, sex was also controlled for (see the Data Preparation and Statistical Analyses section). ADHD = attention deficit hyperactivity disorder; WM = working memory; STM = short-term memory; CI = confidence interval.

\(^{a}\)Total effect of ADHD on fluid intelligence was -.301; total effect of ADHD on crystallized intelligence was -.263.

\( *p < .05 \).

\( **p < .01 \).

\( ***p < .001 \).
(both fluid and crystallized) was also significant in each of these cases. ADHD-related deficits in intelligence were thus not fully mediated by any one of the specific cognitive functions. Table 3 also shows that the mediators accounted for 4%–40% of the total effect of ADHD on fluid intelligence, and 10%–46% of the total effect of ADHD on crystallized intelligence. Inhibition accounted for the weakest mediation effects and processing speed for the strongest mediation effects in both types of intelligence. When evaluating the total mediation effect for all of these specific cognitive functions together (Fig. 2), the bootstrapping results showed that for crystallized intelligence, there was a significant full mediation effect, as evidenced by a significant total indirect effect through the mediators in combination with a non-significant direct effect of ADHD on crystallized intelligence. Sixty-five percent of the total effect of ADHD on crystallized intelligence was accounted for by all mediators together. Although the total indirect effect through the mediators was significant for fluid intelligence, there was also a significant direct effect of ADHD on fluid intelligence, thus refuting a full mediation effect. Forty-three percent of the total effect of ADHD on fluid intelligence was accounted for by all mediators together.

Comorbidity between ADHD and other externalizing disorders (i.e., ODD or CD) or internalizing disorders did not account for the results, as indicated by the fact that (a) the ADHD dummy variable neither interacted significantly with the ODD/CD dummy variable nor with the internalizing disorder dummy variable (all \( p > .10 \)), and (b) control for the two diagnostic dummy variables did not change conclusions about significant contributions of the ADHD dummy variable (all \( p < .05 \)).

**Discussion**

This study demonstrated that children diagnosed with ADHD have poorer fluid and crystallized intellectual ability relative to children diagnosed with another psychiatric disorder and to children with no psychiatric diagnosis. More interestingly, however, is our finding specifying this intellectual deficit by showing that it was only partially mediated by deficits in each one of the EF-related components and non-EFs, WM, inhibition, sustained attention, STM, and processing speed. Even when considering the total mediation effect of all of these potential mediators, a significant direct effect of ADHD on fluid intelligence was found. In contrast, regarding crystallized intelligence, the direct effect of ADHD was not significant when considering the total mediation effect of all of these cognitive functions, evidencing full mediation. Comorbidity with internalizing disorders or with the externalizing disorders ODD and CD did not change these results.

![Fig. 2](https://academic.oup.com/acn/article-abstract/24/8/769/3240/Intellectual-Deficits-in-Children-with-ADHD-Beyond/1)

**Fig. 2.** Results from mediation analyses investigating direct and indirect effects of ADHD on intelligence: (a) presents results for fluid intelligence; (b) presents results for crystallized intelligence. Confidence intervals (CI) not including 0 indicate a significant total indirect (mediation) effect. A significant direct effect refutes a full mediation effect. ADHD = attention deficit hyperactivity disorder; WM = working memory; STM = short-term memory; gF = fluid intelligence; gC = crystallized intelligence. The presence of another diagnosis than ADHD was controlled for in each analysis by entering a dummy coded variable representing “clinical comparisons/others”. In all analyses with fluid intelligence as outcome variable, sex was also controlled for (see Data Preparation and Statistical Analyses section). *p < .01. Superscript “a” indicates that the indirect effect for the inhibition composite measure was \(-.002\) and \(-.009\), respectively, for fluid and crystallized intelligence. The direct effect of ADHD on fluid intelligence was \( \beta = -.161, p < .01 \), and the direct effect of ADHD on crystallized intelligence was \( \beta = -.098, p > .05 \), when the inhibition composite measure was used as a mediator.
Our finding of poorer crystallized and fluid intelligence in children with ADHD compared with healthy comparison children is in line with a large body of research (e.g., Barkley, 1997; Kuntsi et al., 2004; Sonuga-Barke et al., 2008), including a meta-analysis (Fraizer et al., 2004). We extended this prior knowledge by showing that the effect on intellectual function was specific to children with ADHD and not due to a more general effect of having a psychiatric diagnosis. Further support for this comes from our secondary analyses showing no effects of disorders comorbid with ADHD.

What does the information gained in this study tell us about the nature of the deficits in intellectual ability found in children with ADHD? We approached this question by a mediation perspective and asked whether the relation between ADHD and intelligence could be mediated by specific cognitive functions. Worth noting first, and in line with our hypothesis, is that each one of the specific cognitive functions included in this study, partially mediated the relation between ADHD and intelligence. This suggests that specific cognitive functions do play some part in the poorer intellectual abilities of children with ADHD. It should be mentioned that the relatively strong mediating role of processing speed may have been magnified by the time restrictions inherent in several of the WISC-III subtests. However, none of these specific functions were alone able to fully explain the intellectual deficits found in children with ADHD. This finding contradicts the idea of intellectual deficits in children with ADHD being a reflection of impairments in WM (see e.g., Nigg, 2006), or any of the other separate cognitive functions included in the present study. These conclusions hold for both crystallized and fluid intelligence.

When taking a step further and looking at the total mediation effect of all cognitive functions included in this study, the results appear different for crystallized and fluid abilities. For fluid intelligence, it is especially noteworthy that although the total mediation effect was significant, the poorer intellectual abilities of children with ADHD were not fully mediated by the studied EF-related components and non-EFs. It is worth noting that this was true in spite of the fact that there were some overlapping time restrictions in the fluid intelligence variable and the processing speed variable. This means that there is some cognitive deficit represented in these children’s fluid abilities that could not be explained by the poorer EF-related components and non-EFs included in this study.

The present study holds several advantages compared with the Rommelse et al. (2008) study, in which a similar conclusion was drawn. We involved tasks of a wide range of potential mediators, including several EF-related components as well as non-EFs, and we also considered the role of comorbidity. Our findings thus extend theirs by showing that the entire fluid intellectual deficit in children with ADHD (a) could not be explained even if the variety of potential mediators was extended and (b) could not be accounted for by the comorbidity with other externalizing disorders or with internalizing disorders. It is important to note that the poorer WISC performance in children with ADHD could potentially reflect other factors in addition to poorer ability, for example, a general test-taking impairment that would extend to nearly all test-taking situations (e.g., Barkley, 1998; also see, Mahone et al., 2003). However, the direct effect of group on fluid intelligence after accounting for the mediators should constitute other performance factors (most likely, ability), as the variance pertaining to a general test-taking impairment should have been eliminated by the mediators.

Our interpretations of the results are based on a hierarchical view of intelligence (e.g., Carroll, 1993). According to this perspective, intelligence is thought to reflect multiple separate abilities working together, supplemented by a shared general resource. The fluid intellectual deficits in children with ADHD that could not be accounted for by specific cognitive functions could be interpreted to reflect a weakness in the general resource that supplements the specific, relatively independent abilities.

Regarding crystallized intelligence, there was a different pattern of results than for fluid intelligence. The relation with ADHD was generally weaker than for fluid intelligence, in contrast to findings from some previous studies (Chhabildas et al., 2001; Fraizer et al., 2004; Rommelse et al., 2008). This could be explained by the fact that the subtests Digit Span and Arithmetic, largely tapping WM, were included in verbal/crystallized intelligence measures in these previous studies. Although none of the cognitive functions studied here were able to by itself fully mediate the poorer crystallized ability in children with ADHD, all of these functions taken together did. In other words, children with ADHD do not seem to have poorer abilities to acquire information through education and experience, beyond those explained by deficits in several important EF-related components and non-EFs. These results are partly in line with the findings by Biederman and colleagues (2004), which showed that executive dysfunction played a major part in academic difficulties among children with ADHD. The differences found between the two intelligence types in the present study support earlier suggestions that a more representative picture of the cognitive functioning of a child with ADHD is yielded when different intelligence factors are viewed as separate, rather than constituting a unity (Fiorello et al., 2002; Schwean & Saklofske, 1998).

Our findings showing that children with ADHD have fluid intellectual deficits beyond the impairment of specific cognitive functions have important practical, theoretical as well as clinical implications. First, based on our findings, future ADHD research should consider reevaluating the relevance of fluid intellectual ability in children with ADHD (see also Dennis et al., 2009). Our results demonstrate that fluid intelligence provides significant information about neuropsychological impairments in ADHD. Consequently, it deserves more attention than as a mere control variable, which today is how it is commonly viewed (for further arguments against control of IQ, see Dennis et al., 2009). It is also interesting to note that the deficits in fluid
intelligence found beyond those explained by the studied cognitive functions were similar in magnitude to those found for the EF-related components and non-EFs. It gives a hint as to the relevance of the information that could be gained from studying fluid abilities in children with ADHD. Second, the findings emphasize the importance of defining the nature of the general intellectual resource deficit. Here, the perspective presented by Garon, Bryson, and Smith (2008) in a broad review of the normal development of EF is interesting. They considered EF to be a unitary construct with partially dissociable components and suggested that what sets the stage for EF components to develop from simple to complex mechanisms is a broader attentional mechanism. This mechanism may be linked to, or equated with, the attention function denoted executive attention by other researchers (Miyake et al., 2000). Applied to our context, it may be such executive attentional functions that are reflected in the fluid deficits demonstrated by children with ADHD. Thus, deficits in this attentional function could be thought of as underlying these children’s weaknesses in separate EF components, but because of its broader scope, could also be reflected in the additional fluid deficits.

Third, based on the above interpretation, our findings may have important implications for the development of interventional EF training programs, such as that presented by Klingberg and colleagues (2005). As executive attentional processes are suggested to underlie the development of the EF components (Garon et al., 2008), intervention programs involving training of individual EF components, such as WM, may not be targeting the central processes that bring about EF deficits. Greater and more generalizable effects could perhaps be expected if the cognitive training instead aimed at the underlying deficit in executive attentional functions. However, before this hypothesis can be tested valid measurement of executive attention must be developed.

Strengths and Limitations

Although the EFs studied here as well as the tasks selected to represent the different cognitive constructs are selected based on sound theoretical and methodological grounds, we acknowledge the possibility that using a different set of tasks and tests would result in a different set of results. For example, mental set-shifting is one of few well-defined EF-related components (Miyake et al., 2000), not considered in the present study. However, since there is little empirical work linking this EF-component to both intelligence and ADHD, the relevance of this function as a mediator could potentially be down played. Further, although EF deficits are viewed as one of the strongest etiological factors of ADHD that have also been implicated in intelligence, there are other etiological factors, the inclusion of which could potentially result in a full mediation—for example, motivational deficits or delay aversion (e.g., Carlson & Tamm, 2000; Sonuga-Barke, 2005). However, also here is the empirical support for linking these factors to intelligence scarce. It could further be argued that the variables used as mediators in the present study would be similarly affected by, for example, a motivational deficit.

It is important to note that the issue of task selection also applies to the choice of intelligence tests. That is, the probability of obtaining a different pattern of results had a different intelligence test battery been used should be considered. This is for future studies to clarify. Further, regarding the separation of fluid and crystallized intelligence factors, other intelligence test batteries may be more advanced than the WISC-III. We therefore recommend future studies pursuing this issue to adopt batteries constructed along the theoretical lines of the Cattell–Horn–Carroll theory of cognitive abilities (e.g., McGrew & Woodcock, 2001), such as the Woodcock–Johnson III (Woodcock, McGrew, & Mather, 2001).

The community-based sample used in the present study is assumed to more validly represent the whole population of children with ADHD than more commonly used clinic referred samples. Clinical samples are usually biased toward inclusion of more severe ADHD cases, which could possibly represent a special subgroup of children with ADHD. Further, the fact that 41% of the children in our normal comparison group had initially been screened positive for mental health problems (although not reaching clinical significance) alleviates potential problems accompanied by having a “super-normal” comparison group. (Further, the conclusions drawn from our results were the same when the children with a probable diagnosis were included in the normal comparison group.) The fact that we were able to find significant group differences despite this group composition further strengthens our results.

Summary and Conclusions

Using a mediation approach, this study has revealed intriguing new information about what intellectual deficits in children with ADHD tell us about the disorder. We have shown that, regarding fluid intelligence, the intellectual deficits are not fully mediated by, but rather go beyond, weaknesses in central EF-related components and non-EFs, with regard to WM, inhibition, sustained attention, STM, and processing speed. We tentatively interpret these fluid deficits as reflecting deficiencies in a general intellectual resource of executive attentional processes (see e.g., Miyake et al., 2000). Concerning crystallized ability, in contrast, the deficit signifies poor abilities in these specific cognitive functions, as indicated by the absence of a
direct effect of ADHD on this type of intelligence. The differences between crystallized and fluid intelligence in ADHD-related impairments point to the value of studying these intelligence types separately, particularly in studies of ADHD. Knowledge about the implications of intellectual deficits in children with ADHD, such as that gained in the present study, is highly valuable in that variation in these abilities have been proposed to hold the key to understanding the heterogeneity of ADHD (Mill et al., 2006). Our interpretations also have crucial implications for interventional cognitive training programs in ADHD, by suggesting that such programs could be better off targeting an underlying deficit in executive attention rather than the resultant deficits in individual EF components. In conclusion, the results indicate that the significance of intelligence in ADHD should not be limited to a mere control variable in analyses studying etiological factors, but should rather be viewed as holding critical information about the disorder per se.

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Conflict of Interest

None declared.

References


