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Neurocognitive Profiles in Children With ADHD and Their Predictive Value for Functional Outcomes

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Abstract

Objective: We examined whether neurocognitive profiles could be distinguished in children with ADHD and typically developing (TD) children, and whether neurocognitive profiles predicted externalizing, social, and academic problems in children with ADHD. **Method:** Neurocognitive data of 81 children with ADHD and 71 TD children were subjected to confirmatory factor analysis. The resulting factors were used for community detection in the ADHD and TD group. **Results:** Four subgroups were detected in the ADHD group, characterized by (a) poor emotion recognition, (b) poor interference control, (c) slow processing speed, or (d) increased attentional lapses and fast processing speed. In the TD group, three subgroups were detected, closely resembling Subgroups (a) to (c). Neurocognitive subgroups in the ADHD sample did not differ in externalizing, social, and academic problems. **Conclusion:** We found a neurocognitive profile unique to ADHD. The clinical validity of neurocognitive profiling is questioned, given the lack of associations with functional outcomes. *(J. of Att. Dis. 2019; 23(13) 1567-1577)*

Keywords

ADHD, primary school-age children, community detection, neurocognitive functioning, functional outcomes

ADHD is the most common mental health disorder diagnosed in children and adolescents (Willcutt, 2012). The disorder is characterized by a persistent pattern of age-inappropriate levels of inattention and/or hyperactivity-impulsivity (American Psychiatric Association, 2013) and is etiologically heterogeneous (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Sonuga-Barke & Halperin, 2010). Previous work showed that several distinct etiological pathways can be distinguished in ADHD (e.g., Sonuga-Barke, Bitsakou, & Thompson, 2010), suggesting the existence of more homogeneous subgroups within the disorder. One approach to detect which subgroups can be distinguished derives from graph theory. Graph theory is used to model relations between objects within a network (Reichardt & Bornholdt, 2007). In clinical research, community detection procedures are used to examine the relations between individuals. This approach is data driven and enables to detect which individuals share similar characteristics (Fair, Bathula, Nikolas, & Nigg, 2012). In the past, latent class analysis was applied to distinguish subgroups within the ADHD population based on, for instance, symptom profiles (Rohde et al., 2001). However, none of these approaches could classify all children with ADHD within distinct subgroups. Furthermore, these approaches target phenotypical presentations of ADHD, while it seems more logical to explore different endophenotypes within the disorder when trying to gain more insight into the etiology of ADHD. Endophenotypes are thought to be more directly related to etiological factors such as genetic alterations and environmental risk factors. Previous work has identified several neurocognitive pathways for ADHD, that are well-grounded in neuroscience (Castellanos & Tannock, 2002). These pathways involved (a) a specific abnormality in reward-related circuitry related to abnormalities in (ventral) frontostriatal brain areas, (b) deficits in temporal processing involving the basal ganglia and cerebellum, and (c) deficits in working memory related to abnormalities in (dorsal) frontostriatal areas (Castellanos & Tannock, 2002).

Recently, a paradigm shift took place from identifying subgroups of children with ADHD based on a single neurocognitive deficit, toward identifying subgroups that share a neurocognitive profile, acknowledging that neurocognitive

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functioning consists of a complex interplay of strengths and weaknesses. In recent years, researchers have focused on the construction and analysis of psychopathology networks, based on the proposition that psychopathology symptoms causally influence each other (Borsboom & Cramer, 2013). The same holds for neurocognitive functioning in children with ADHD, as (reciprocal) interactions between neurocognitive domains have been found in ADHD (Sonuga-Barke et al., 2010). Moreover, children with ADHD often show impairments across multiple neurocognitive domains (Nigg et al., 2005). Using association networks enables to detect which neurocognitive features interact with each other. A model of clusters of neurocognitive functions that interact with each other provides more information about distinct etiological trajectories that are shared by a subgroup, than examining single neurocognitive risk factors for a disorder (Borsboom & Cramer, 2013). Neurocognitive measures apparently do not one-to-one translate into symptoms of ADHD (Van Lieshout, Luman, Buitelaar, Rommelse, & Oosterlaan, 2013). Therefore, a network approach for neurocognitive functioning may be valuable to provide more insight into multiple etiological pathways in ADHD (e.g., Nigg et al., 2005; Sonuga-Barke et al., 2010). More specifically, it was argued that distinct neurocognitive profiles could be indicative of separate neurobiological pathways. Thus far, in three studies community detection procedures were applied to distinguish subgroups of individuals with ADHD based on neurocognitive endophenotypes, all showing distinct neurocognitive profiles (Fair et al., 2012; Mostert et al., 2015; Van Hulst, De Zeeuw, & Durston, 2015).

In the study of Fair et al. (2012), subgroups of children with ADHD were characterized by either (a) high levels of response variability, (b) reduced working memory, memory span, and processing speed, (c) inaccurate temporal information processing, or (d) suboptimal arousal. The results of Van Hulst et al. (2015) in children with ADHD showed subgroups characterized by either (a) fast reaction times and high cognitive control, (b) poor cognitive control, or (c) slow and variable timing. Mostert et al. (2015) showed that in adults with ADHD, subgroups were either characterized by (a) impaired attention and inhibition, (b) impaired delay discounting, or (c) impaired fluency and memory. As all three studies used different selections of neurocognitive measures, profile characteristics differed across studies. In all studies, the detected profiles in the ADHD group were also observed in typically developing controls, with individuals with ADHD generally showing weaker performance (Fair et al., 2012; Mostert et al., 2015; Van Hulst et al., 2015). This finding suggests that individuals with ADHD reflect the extremes of normal neurocognitive heterogeneity.

One step forward in the approach of profiling is to include a more extensive set of measures that reflect core neurocognitive alterations in ADHD. One of the most consistently reported deficits in ADHD is an increase in intraindividual reaction time variability, including increased variability in responding and attentional lapses as measured by ex-Gaussian modeling (Tamm et al., 2012). However, thus far ex-Gaussian measures of intraindividual reaction time variability have not been addressed in neurocognitive profiling of children with ADHD. Another core deficit in ADHD, that has been omitted thus far in neurocognitive profiling studies, is the ability to recognize facial emotional expressions, a central aspect of social cognition (Shaw, Stringaris, Nigg, & Leibenluft, 2014).

One potential use of neurocognitive profiling in ADHD, that has not been addressed thus far, is to provide more insight into the clinical value of neurocognitive assessment in ADHD. ADHD is a clinically heterogeneous disorder, with great variance in functional outcomes across children. Although ADHD increases the risk of associated externalizing problems (including oppositional defiant disorder [ODD] and conduct disorder [CD]; Gillberg et al., 2004), social problems (McQuade & Hoza, 2008), and academic problems (Loe & Feldman, 2007), not all children with ADHD are impaired in terms of these functional outcomes. The heterogeneity in functional outcomes evokes to examine the differential risks leading to the heterogeneous outcomes, using more homogeneous subgroups in terms of neurocognitive strengths and weaknesses. We suggest that neurocognitive profiles may act as moderators, explaining heterogeneity in adverse outcomes. More specifically, we hypothesize that a subgroup of children with ADHD characterized by emotion recognition deficiencies has an increased risk of associated externalizing and social problems, as emotion recognition deficits have been found to increase the risk of ODD (Noordermeer et al., 2015), CD (Cadesky, Mota, & Schachar, 2000), and decreased social functioning (Trentacosta & Fine, 2010). Likewise, we hypothesize that a subgroup of children with ADHD characterized by deficiencies in cool executive functions (EF), including working memory and inhibitory control, has an increased risk of academic problems, given the role of EF in, for example, math performance (Antonini et al., 2016).

The current study sought to replicate previous work on community detection in samples of children with ADHD and typically developing (TD) children. In line with Fair et al. (2012), we expected to find distinct neurocognitive profiles based on children's performance on measures of cool EF (memory span, working memory, interference control). The present study extends previous research by adding ex-Gaussian parameters of intraindividual reaction time variability (processing speed, variability in responding, and lapses of attention), as well as a measure of social cognition (emotion recognition). In line with previous studies (Fair et al., 2012; Van Hulst et al., 2015), we expected to find similar neurocognitive profiles in children with ADHD and

TD children, with weaker performance for the ADHD group. We also examined whether the neurocognitive profiles within the ADHD group would reflect differential risks for functional outcomes: associated externalizing, social, and academic problems.

Method

Participants

Subjects were 81 children with ADHD (74% males) and 71 TD children (52% males), aged between 6 and 13 years. Inclusion criteria for the ADHD group were (a) a clinical diagnosis of ADHD according to *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) criteria, (b) confirmation of this diagnosis by the Diagnostic Interview Schedule for Children, fourth edition, administered to parents (DISC-IV-P; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), (c) significant ADHD symptoms, as indicated by scores >90th percentile on at least one of the ADHD scales (Inattention and Hyperactivity/Impulsivity scales) of the parent version of the Disruptive Behavior Disorder Rating Scale (DBDRS; Pelham, Gnagy, Greenslade, & Milich, 1992), and (d) pervasive ADHD symptoms, as indicated by scores >75th percentile on at least one of the ADHD scales of the teacher version of the DBDRS. Having a comorbid diagnosis (for example ODD) was no exclusion criterion, neither was treatment with stimulant medication. Children on stimulant medication (59% of the ADHD group) discontinued drug use 24 hr before testing, to allow complete washout. Inclusion criteria for the TD group were (a) absence of a clinical diagnosis of ADHD or ODD as obtained from parent information, and (b) scores <90th percentile on both parent- and teacher-rated ADHD scales of the DBDRS. Children with an IQ <70 were excluded.

Materials

Diagnostic assessment. Parents of children eligible for inclusion in the ADHD group were assessed with the ADHD section of the DISC-IV-P (Shaffer et al., 2000). The DISC-IV-P is a widely used standardized diagnostic interview for the assessment of *DSM-IV* childhood psychiatric disorders.

ADHD symptom severity was assessed in both groups using the DBDRS, filled out by one of the parents and teacher (Pelham et al., 1992). The DBDRS measures the *DSM-IV* symptoms of ADHD (and other externalizing disorders) using a 4-point Likert scale. Scores on the Inattention and Hyperactivity/Impulsivity scales were used, with higher scores indicating worse symptoms.

Full-scale IQ was estimated using a short form of the Wechsler Intelligence Scale for Children–III (WISC-III; Wechsler, 1991), comprising the subtests Vocabulary, Arithmetic, Block Design, and Picture Arrangement. This short form of four subtests has an excellent reliability (r = .95) and validity (r' = .90) (Sattler, 2008).

Neurocognitive functioning. To measure verbal working memory, the backward condition of the Digit Span Task of the WISC-III was used (Wechsler, 1991). In the backward condition, the child is required to repeat in reversed order a sequence of numbers expressed verbally by the interviewer. There were seven sequence levels, starting from a span of two digits up to a span of eight digits. The product of the total number of correct responses and the highest obtained span served as measure of verbal working memory (Kessels, Van Zandvoort, Postma, Kappelle, & De Haan, 2000).

Visuospatial working memory was measured using the backward condition of the Grid Task (Bergman Nutley, Soderqvist, Bryde, Humphreys, & Klingberg, 2010), in which the child is required to repeat in reversed order a sequence of visual stimuli (yellow dots) presented on a computer screen in a four by four grid. The computer mouse was used to respond. There were nine sequence levels, starting from two up to a span of 10 dots. Each level consisted of Sublevel A, in which the sequence followed a logical pattern, and the more difficult Sublevel B, in which the sequence showed no clear pattern. The product of the total number of correct responses and the highest obtained span served as measure of visuospatial working memory (Kessels et al., 2000), with 0.5 point added to the highest obtained span when Sublevel B was reached (Bergman Nutley et al., 2010).

For interference control, processing speed, variability in responding, and lapses of attention, an adapted version of the Eriksen Flanker Task was used (Eriksen & Eriksen, 1974; Scheres et al., 2003). In each trial, a target stimulus (black arrow) appeared at the centre of a computer screen, pointing either to the left or right side, and the child was asked to press the spatially corresponding response button (left or right button). In the neutral condition, the target was flanked by two neutral items (rectangles), in the congruent condition by arrows pointing in the same direction, and in the incongruent condition by arrows pointing in the opposite direction. In total, 48 neutral, 48 congruent, and 48 incongruent trials were presented in random order. Difference scores between congruent and incongruent trials were calculated for latency on correct trials and accuracy, and served as measures of interference control (Mullane, Corkum, Klein, & McLaughlin, 2009). Further, individual response time distributions derived from the correct neutral trials were examined. The mean (mu) and standard deviation (sigma) of the normal component of the ex-Gaussian distribution served as measures of processing speed and variability in responding, and the mean plus standard deviation of the exponential component of the ex-Gaussian distribution (tau) as measure of attentional lapses (Whelan, 2010).

Recognition of facial emotional expressions was examined using the Children's Emotion Recognition Task (CERT), a computerized task developed for the current study. The CERT is an adaptation of a previously validated paradigm (Nowicki & Duke, 1994), but instead of using pictures of adult faces, the CERT consists of solely pictures of children's faces, to make the task more ecologically valid. The CERT contains hundred pictures of children's faces with neutral facial expressions, and four basic emotional expressions (happy, fear, anger, sadness); 20 stimuli per emotional expression, presented in random order. Pictures were selected from the validated National Institute of Mental Health Child Emotional Faces Picture Set database, based on their distinctive character (Egger et al., 2011). Upon presentation of the stimulus, participants had to indicate the corresponding emotion with a computer mouse: either angry, frightened, happy, sad, or neutral. The task was self-paced. For all five expressions, inverse efficiency scores were calculated, by dividing the mean reaction time by the proportion of correct responses (Townsend & Ashby, 1983).

Externalizing problems. Associated externalizing problems were examined using the ODD and CD scales of the parent- and teacher-rated DBDRS (Pelham et al., 1992).

Social functioning. Sociometric data were collected for all participating children (Coie & Dodge, 1983). All children in the classroom of a participant were required to nominate classroom peers they liked most (positive rating) and classroom peers they disliked most (negative rating). Children were free to nominate as many peers for both categories as they wanted. For each participant, we summed up the number of times the child was positively rated and the number of times the child was negatively rated. To adjust for classroom size, sum scores were divided by the number of classroom peers, yielding percentages of positive ratings and negative ratings, which were used as indicators of social acceptance and social rejection, respectively (Coie & Dodge, 1983).

Social problems (such as getting teased) were examined using the 11-item Social Problems scale of the Child Behavior Checklist (CBCL) and Teacher Rating Form (TRF), completed by parents and teachers, respectively (Achenbach, 1991). For both scales, items were rated on a 3-point Likert scale and summed up.

Academic functioning. Academic functioning was examined using data collected by teachers for the national pupil monitoring system, containing measures of reading comprehension (Staphorsius & Krom, 1998), spelling (de Wijs, Krom, & van Berkel, 2006), and mathematics (Janssen, Verhelst, Engelen, & Scheltens, 2010). These tests are administered twice a year and provide ability scores, which are standardized scores per academic domain, across the school grades.

Procedure

Children with ADHD were recruited from mental health outpatient clinics, through the parental association for children with behavioral problems, and through a university research website. The TD group was recruited from primary schools located throughout the country. Children were tested at their own school. Prior to participation, written informed consent was obtained of parents of all children, and of children ≥12 years. This study received approval from the local medical ethical committee (#NL39922.029.12).

Data Analysis

All statistical analyses were performed using R, version 3.2.1. All neurocognitive dependent variables were transformed into *z*-scores, to have all measures on the same metric scale, and if necessary reverse-scored, to ensure that for all variables higher scores were indicative of better performance.

The number of neurocognitive measures per domain was reduced by performing confirmatory factor analyses. Based on the model of Fair et al. (2012), it was hypothesized which of the neurocognitive measures were representative of the same latent factor. Our conceptual model consisted of seven latent factors: memory span, working memory, interference control, processing speed, variability in responding, lapses of attention, and emotion recognition. We acknowledged that there might be better fitting models, and therefore we also examined a six-factor model (6A) in which memory span and working memory were combined into one latent factor (memory); another six-factor model (6B) in which variability in responding and lapses of attention were combined into one latent factor (response time variability); and a five-factor model consisting of memory, response time variability, interference control, processing speed, and emotion recognition. Correlations between neurocognitive measures >.80 were interpreted as signs of multicollinearity (Field, Miles, & Field, 2012). Fit of all models was evaluated using chi-square (χ^2), comparative fit index (CFI), Tucker–Lewis index (TLI), root mean square error of approximation (RMSEA), and standardized root mean square residual (SRMR; Hu & Bentler, 1998). The most parsimonious model with adequate fit was selected and factor scores were calculated, serving as measures of neurocognitive functioning.

It was examined whether distinct neurocognitive profiles could be detected in the ADHD and TD group separately, using community detection procedures (see Fair et al. [2012] for a detailed description). Briefly, in each group, participants were assigned to detected subgroups using the Louvain algorithm (modularity_louvain_und_sign.m by Rubinov & Sporns, 2011)). For both groups, correlation matrices between subjects' neurocognitive factor scores

Table 1. Group Characteristics of the ADHD and TD Group.

	ADHD group (*n* = 81)	TD group (*n* = 71)	t/χ^2
Age in months, *M* (*SD*)	116.52 (19.96)	118.87 (20.68)	−.71, *ns*
Estimated IQ, *M* (*SD*)	100.23 (13.98)	104.24 (14.04)	−1.76, *ns*
Males, *n* (%)	60 (74.07)	37 (52.11)	7.90**
Parent-rated			
Inattention, *M* (*SD*)	17.56 (4.83)	3.34 (3.08)	21.88**
Hyperactivity/impulsivity, *M* (*SD*)	16.52 (5.87)	3.27 (2.73)	18.20**
Teacher-rated			
Inattention, *M* (*SD*)	14.72 (6.11)	1.87 (2.45)	17.40**
Hyperactivity/impulsivity, *M* (*SD*)	14.02 (7.15)	1.56 (2.31)	14.83**

Note. TD = typically developing; *ns* = not significant.
**$p < .01$.

were created, providing insight into connections between clusters of participants. Subgroup assignment of each participant was based on group assignment across 100 runs of the modularity algorithm, applying the most frequent group assignment (mode). Robustness of the community structure was determined based on the quality index (Q), with values >.40 being interpreted as indication of distinct subgroups (Fortunato & Barthelemy, 2007). For both the ADHD and the TD group, characteristics of the subgroups were examined by comparing the neurocognitive subgroups to each other, using analysis of variance (ANOVA), and post hoc comparisons per factor score. Visual inspection of the plots was carried out to examine whether the profiles were similar in the ADHD and TD group (Fair et al., 2012). In case of similar profiles, it was investigated whether children with ADHD differed from TD children in factor scores, using multivariate analysis of variance (MANOVA). These analyses were done for each profile separately. Post hoc comparisons tested group differences on the factor scores.

To study the predictive validity of the neurocognitive profiles, it was investigated whether the neurocognitive subgroups in the ADHD group differed from each other on measures of externalizing, social, and academic functioning, using ANOVA and post hoc comparisons between subgroups. To correct for multiple testing, the alpha level was adjusted according to the Bonferroni procedure per outcome domain: externalizing (four analyses, $p = .013$), social (four analyses, $p = .013$), and academic problems (three analyses, $p = .017$).

Results

For group characteristics, see Table 1. Analyses showed that the ADHD and TD group did not differ significantly in mean age and IQ. The ADHD group showed more ADHD symptoms and consisted of considerably more males than the TD group. The DISC-IV-P indicated that 64 children met the *DSM-IV* criteria for the combined subtype of ADHD, 11 children for the predominantly inattentive subtype, and six children for the predominantly hyperactive-impulsive subtype.

Correlation analyses showed no sign of multicollinearity (*r*s = .10-.80). Fit indices were not satisfactory for the five-factor model, $\chi^2(68) = 147.5$, CFI = .92, TLI = .90, RMSEA = .09, SRMR = .06, and for six-factor model 6B, $\chi^2(63) = 137.6$, CFI = .93, TLI = .90, RMSEA = .09, SRMR = .06. All fit indices were satisfactory for the other two models: for six-factor model 6A, $\chi^2(65) = 101.0$, CFI = .97, TLI = .95, RMSEA = .06, SRMR = .06, and for the seven-factor model, $\chi^2(59) = 88.0$, CFI = .97, TLI = .96, RMSEA = .06, SRMR = .05. Out of the two well-fitting models, we selected the most parsimonious model, the six-factor model that is depicted in Figure 1.

Community detection analysis within the ADHD sample yielded four profiles (see Figure 2, panel a). The quality index ($Q = .45$) showed that the identified neurocognitive subgroups were strongly distinct from each other. Compared with the other subgroups, Subgroup 1 ($n = 20$) was characterized by poor emotion recognition, $F(3, 77) = 6.59$, $p < .01$; Subgroup 2 ($n = 9$) by poor interference control, $F(3, 77) = 6.49$, $p < .01$; and Subgroup 3 ($n = 26$) by slow processing speed, $F(3, 77) = 13.56$, $p < .01$. Subgroup 4 ($n = 26$) was characterized by increased attentional lapses, $F(3, 77) = 8.91$, $p < .01$, and fast processing speed, $F(3, 77) = 13.56$, $p < .01$, compared with Subgroups 1 and 3. Characteristics of the four subgroups are summarized in Table 2 and comparisons between the subgroups are shown in Table 3. There were no differences in IQ between subgroups. Subgroup 3 had less parent-rated symptoms of inattention compared with Subgroups 1 and 4 and less parent-rated symptoms of hyperactivity/impulsivity compared with Subgroup 1. Subgroup 4 had a higher mean age compared with Subgroups 1 and 3.

Community detection analysis within the TD sample yielded three profiles (see Figure 2, Panel b). The quality index ($Q = .49$) showed that the identified neurocognitive

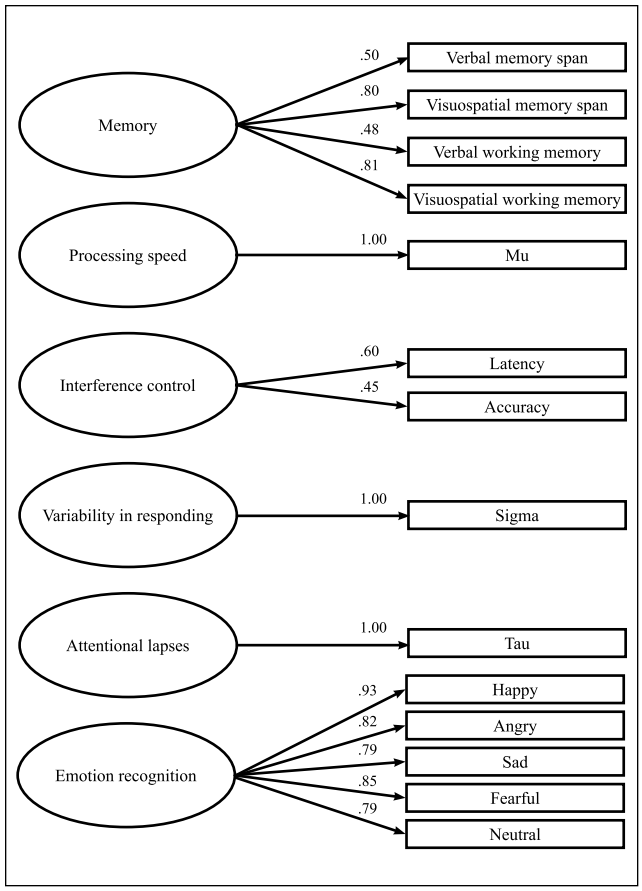

Figure 1. Overview of the six-factor model, created for data reduction.
Note. The numbers represent factor loadings.

subgroups were strongly distinct from each other. Upon visual inspection it is clear that the three subgroups in the TD group closely resembled the first three subgroups in the ADHD group, see Figure 2. Analyses using MANOVA showed that, for all three subgroups, children with ADHD showed weaker neurocognitive performance than TD children. Post hoc analyses showed that children with ADHD had weaker neurocognitive performance than the TD children on one to four factors scores per subgroup (see Figure 2 and Table 3). As the ADHD group consisted of considerably more males, analyses were rerun with gender as covariate. All group differences remained significant after adjusting for the effect of gender. The fourth subgroup obtained in the ADHD group, characterized by increased attentional lapses and fast processing speed, was not replicated in the TD group.

We examined whether differences in neurocognitive profiles in the ADHD sample were related to differences in functional outcomes, but found no significant differences between the subgroups on measures of associated externalizing (Fs < 1.20, ps > .34), social (Fs < 3.20, ps > .03), and academic problems (Fs < 1.70, ps > .19).

Discussion

The aim of the current study was to gain more insight into neurocognitive profiles of children with ADHD. We found four distinct subgroups in the ADHD sample, with one subgroup characterized by poor interference control, one by slow processing speed, one by poor emotion recognition, and one by increased attentional lapses and fast processing speed. Our results partially replicate the findings of studies into neurocognitive profiling in children with ADHD, as our subgroup characterized by slow processing speed, was also found by Fair et al. (2012), and our subgroup characterized by fast processing speed also emerged in the study of Van Hulst et al. (2015). On the other hand, our slow processing speed subgroup was not characterized by an additional reduction in working memory and memory span, as found by Fair et al. (2012), and our high processing speed subgroup was not characterized by high cognitive control, as found by Van Hulst et al. (2015). Further, we observed two subgroups, characterized by poor interference control and poor emotion recognition, respectively, which are new findings. The latter is in accordance with the growing consideration that cool EF and social cognition are dissociable neurocognitive domains, with different etiological pathways at the neurobiological level (Zelazo & Carlson, 2012). Our results also confirm that heterogeneity in observed neurocognitive profiles across studies at least partially stems from using different sets of neurocognitive measures as dependent measures. This violation of measurement invariance, due to the selection of different neurocognitive constructs or similar constructs assessed by different instruments, limits the commensurability of neurocognitive profiling in ADHD. This hampers the possibility to derive final conclusions regarding the number and type of neurocognitive profiles being core to ADHD.

In line with earlier work (Fair et al., 2012; Van Hulst et al., 2015), we found neurocognitive subgroups in the ADHD group that were also observed in the TD group, with children with ADHD showing generally weaker neurocognitive performance compared with the TD children. These findings suggest that the heterogeneity in childhood ADHD is nested within the normal variation of neurocognitive functioning in children. Although children with ADHD reflected the extremes of normal neurocognitive heterogeneity on some neurocognitive factors within each subgroup, on other factors their performance overlapped with the performance of TD children. The latter might be explained by the large variance in neurocognitive performance in children with ADHD, showing that not all children with ADHD within a subgroup had weaker neurocognitive functioning on all factors.

Remarkably, in the TD group we did not find a subgroup characterized by fast processing speed and increased attentional

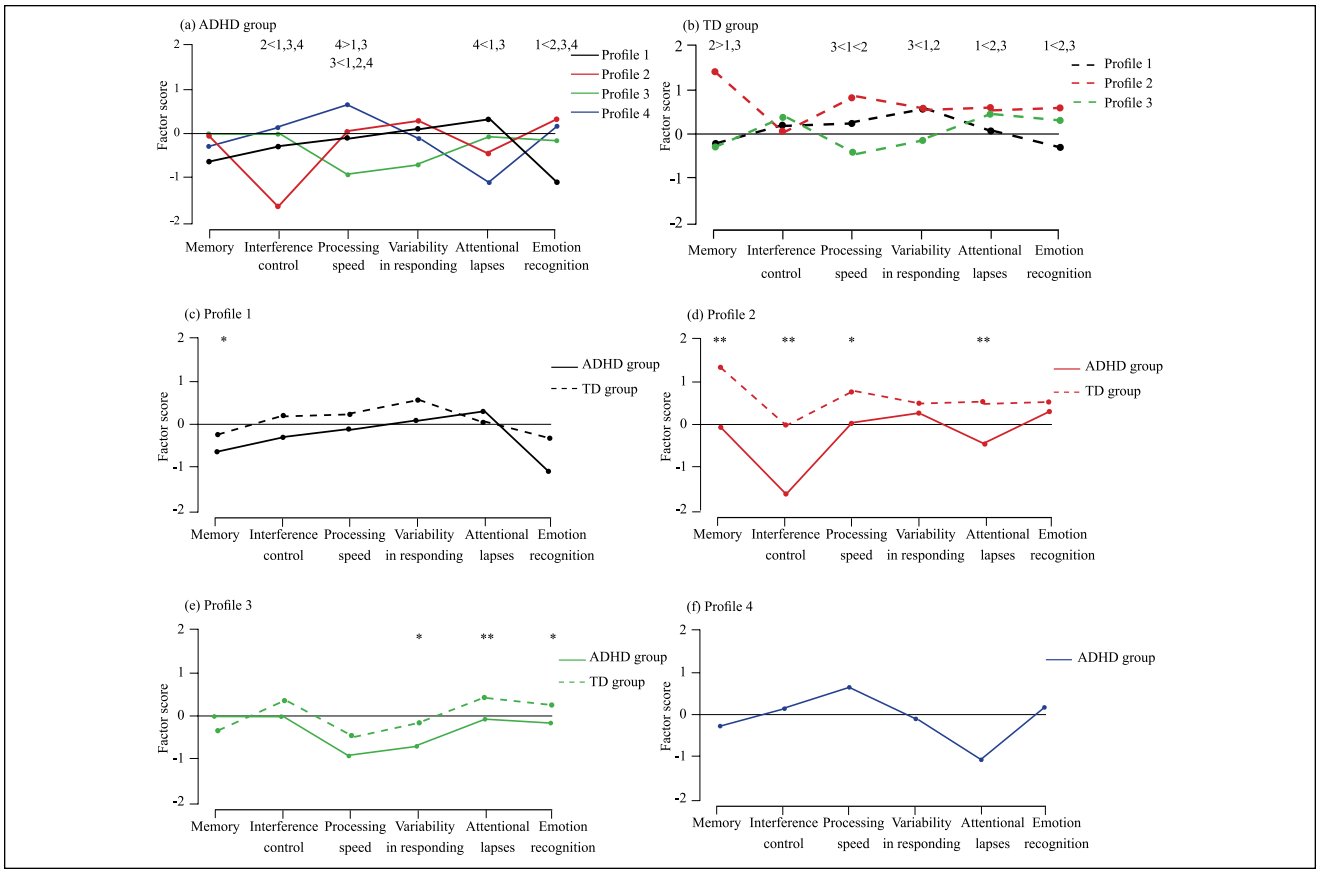

Figure 2. Neurocognitive profiles in children with ADHD (Panel a) and TD children (Panel b). In Panels c to e, a comparison in factor scores between the ADHD and TD group per profile is shown (*$p < .05$. **$p < .01$). Panel f shows a unique neurocognitive profile in the ADHD group.
Note. TD = typically developing.

Table 2. Characteristics of Neurocognitive Profiles of the ADHD Group.

	Profile 1: Poor emotion recognition (n = 20)	Profile 2: Poor interference control (n = 9)	Profile 3: Slow processing speed (n = 26)	Profile 4: Increased attentional lapses/ fast processing speed (n = 26)	F/χ^2	Pairwise comparison
Age in months	110.90 (19.22)	111.89 (23.51)	112.38 (18.54)	126.58 (17.81)	3.58*	4 > 1;4 > 3
Males, n (%)	17 (85)	9 (100)	15 (58)	19 (73)	8.04	1 = 2 = 3 = 4
Estimated IQ	100.10 (15.89)	99.00 (18.93)	102.38 (11.47)	98.62 (13.40)	0.34	1 = 2 = 3 = 4
ADHD symptoms (DBDRS)						
Inattention[a]	19.15 (5.32)	18.89 (4.65)	14.69 (3.91)	18.73 (4.29)	5.22**	3 < 1;3 < 4
Hyperactivity/impulsivity[a]	18.55 (5.70)	17.00 (6.56)	13.81 (4.95)	17.50 (5.95)	3.14*	3 < 1
Inattention[b]	15.30 (6.36)	14.67 (6.69)	13.27 (6.25)	15.73 (5.64)	0.78	1 = 2 = 3 = 4
Hyperactivity/impulsivity[b]	15.55 (5.93)	14.00 (10.37)	12.77 (6.81)	14.12 (7.25)	0.56	1 = 2 = 3 = 4

Note. DBDRS = Disruptive Behavior Disorder Rating Scale. Results are shown in M (SD).
[a]Parent-rated.
[b]Teacher-rated.
*$p < .05$. **$p < .01$.

lapses, suggesting this to be a unique neurocognitive subgroup in children with ADHD. This profile reflects a combination of fast responses at some moments, which could be indicative of impulsive responding (Hervey et al., 2006), and occasional lapses in attention at other moments. This neurocognitive profile could reflect one of many possible etiological pathways of

Table 3. Comparisons Between Children With ADHD and TD Children Per Neurocognitive Profile.

	MANOVA	Memory	Interference control	Processing speed	Variability in responding	Attentional lapses	Emotion recognition
Profile 1: Poor emotion recognition							
ADHD (*n* = 20)		−0.63 (0.62)	−0.27 (0.95)	−0.11 (0.78)	0.08 (0.95)	0.29 (0.64)	−1.12 (1.53)
TD (*n* = 15)		−0.23 (0.47)	0.25 (0.63)	0.22 (0.55)	0.57 (0.51)	0.05 (0.68)	−0.32 (0.47)
Difference (*F*)	2.59*	4.29*	3.45	1.91	3.16	1.15	3.78
Profile 2: Poor interference control							
ADHD (*n* = 9)		−0.07 (1.38)	−1.64 (1.64)	0.07 (0.89)	0.29 (1.04)	−0.45 (1.39)	0.31 (0.52)
TD (*n* = 24)		1.40 (0.89)	0.01 (0.70)	0.80 (0.68)	0.58 (0.63)	0.59 (0.56)	0.60 (0.58)
Difference (*F*)	3.29*	13.17**	16.92**	6.32*	0.94	9.72**	1.72
Profile 3: Slow processing speed							
ADHD (*n* = 26)		0.00 (1.00)	−0.01 (1.04)	−0.91 (1.02)	−0.70 (1.11)	−0.10 (0.82)	−0.14 (0.97)
TD (*n* = 32)		−0.32 (0.60)	0.38 (0.57)	−0.45 (0.77)	−0.16 (0.70)	0.49 (0.44)	0.28 (0.61)
Difference (*F*)	4.43**	2.26	3.25	3.80	5.11*	11.82**	4.08*
Profile 4: Increased attentional lapses/fast processing speed							
ADHD (*n* = 26)		−0.25 (0.64)	0.16 (1.03)	0.66 (0.84)	−0.12 (1.25)	−1.15 (1.21)	0.17 (0.86)

Note. MANOVA = multivariate analysis of variance; TD = typically developing. For each profile, values in the first two rows represent factor scores in *M* (*SD*).
*$p < .05$. **$p < .01$.

symptoms of hyperactivity/impulsivity and inattention. Our findings are in line with previous work on ex-Gaussian parameters, showing that children with the combined presentation of ADHD had fast processing speed and increased attentional lapses (Hervey et al., 2006; Tamm et al., 2012). This combination of fast responses and occasional attentional lapses is explained by complex brain activity during task performance. Greater right temporal-parietal junction activity and greater right inferior frontal gyrus activity have been found prior to fast responses, facilitating stimulus-triggered reorienting of attention after attentional lapses (Weissman, Roberts, Visscher, & Woldorff, 2006). Further, occasional attentional lapses have been related to reduced activity in the frontal cortex, prior to stimulus presentation (Weissman et al., 2006). Further research could focus on the underpinnings of the specificity of this reaction pattern in ADHD.

In terms of examining the clinical value of neurocognitive profiling in ADHD, our results showed no significant associations between any of the neurocognitive subgroups and measures of externalizing, social, and academic problems that are often found in ADHD. This absence of any predictive power questions the clinical relevance of classifying children with ADHD into neurocognitive subgroups using the current set of neuropsychological tasks. It thereby seems that using neurocognitive profiling currently does not provide more insight into the association between neurocognitive functioning and various functional outcomes in ADHD. It should be noted that our study was cross-sectional and therefore focused on the current externalizing, social, and academic problems of children with ADHD. To draw firm conclusions regarding the lack of predictive validity of neurocognitive profiles for future functioning, a longitudinal approach is required. Further, it is recommended to examine the association between neurocognitive profiles and a broader range of functional impairments, including learning disabilities (Barry, Lyman, & Klinger, 2002), repeating a grade (Barbaresi, Katusic, Colligan, Weaver, & Jacobsen, 2007), and peer rejection (McQuade & Hoza, 2008).

This observation of a lack of predictive validity of neurocognitive profiling is in line with general concerns regarding the limited (long-term) predictive validity of neurocognitive functioning for persistence of ADHD (Van Lieshout et al., 2013), or emergence of nicotine dependence or substance use disorders in ADHD (Groenman et al., 2015). Given the current lack of evidence of a pivotal role of neurocognitive functioning in the association between ADHD and various functional outcomes, it might be suggested that neurocognitive problems in ADHD are epiphenomena, potentially sharing the same etiological factors as the ADHD symptoms. Our lack of findings emphasizes the need to explore other factors than neurocognitive deficiencies that mediate or moderate the association between ADHD and associated social and academic problems. For instance, further research might focus on environmental factors, including family and parenting factors (Becker, Luebbe, & Langberg, 2012), to disentangle the true risk factors for associated social and academic problems in ADHD. This may enhance early detection of children with ADHD at risk and might open new roads for preventive interventions.

There are several limitations to the current study. First, although several aspects of neurocognitive functioning were included, our neurocognitive assessment is nonexhaustive

and did not tap into some important neurocognitive domains that have been found altered in ADHD, such as temporal information processing (Toplak, Dockstader, & Tannock, 2006) and reward sensitivity (Luman, Oosterlaan, & Sergeant, 2005). However, our selection of neurocognitive measures is larger and more diverse than the neurocognitive measures used in previous studies. Second, although the size of our sample of children with ADHD was similar to the sample (n = 96) of Van Hulst et al. (2015), replication in a larger sample of children with ADHD is warranted, as the subgroups had small sample sizes.

Conclusion

To conclude, in the current study we were able to replicate the results of previous work on subtyping children with ADHD based on neurocognitive functioning, showing that in children with ADHD neurocognitive subgroups can be found, that are also present in a group of TD children. We also found a unique subgroup in children with ADHD, characterized by fast processing speed and increased attentional lapses, which was not present in TD children. In the current study, no meaningful relationship was found between neurocognitive subgroups and functional outcomes.

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