VU Research Portal

How specific are executive functioning deficits in attention hyperactivity disorder and autism?

Geurts, H.M.; Verte, S; Oosterlaan, J.; Roeyers, R.; Sergeant, J.A.

published in

Journal of Child Psychology and Psychiatry
2003

DOI (link to publisher)

[10.1111/j.1469-7610.2004.00276.x](https://doi.org/10.1111/j.1469-7610.2004.00276.x)

document version

Publisher's PDF, also known as Version of record

[Link to publication in VU Research Portal](#)

citation for published version (APA)

Geurts, H. M., Verte, S., Oosterlaan, J., Roeyers, R., & Sergeant, J. A. (2003). How specific are executive functioning deficits in attention hyperactivity disorder and autism? *Journal of Child Psychology and Psychiatry*, 45(4), 836-855. <https://doi.org/10.1111/j.1469-7610.2004.00276.x>

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal ?

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

E-mail address:

vuresearchportal.ub@vu.nl

How specific are executive functioning deficits in attention deficit hyperactivity disorder and autism?

Hilde M. Geurts,¹ Sylvie Verté,² Jaap Oosterlaan,¹ Herbert Roeyers,²
and Joseph A. Sergeant¹

¹Department of Clinical Neuropsychology, Vrije Universiteit Amsterdam, The Netherlands;

²Department of Psychology, Developmental Disorders, Ghent University, Belgium

Background: The objective of this study is to identify intact and deficient cognitive processes in children with attention deficit hyperactivity disorder (ADHD) and children with high functioning autism (HFA). **Method:** Three rigorously diagnosed groups of children aged between 6 and 12 years (54 ADHD, 41 HFA, and 41 normal controls) were tested on a wide range of tasks related to five major domains of executive functioning (EF): inhibition, visual working memory, planning, cognitive flexibility, and verbal fluency. In addition, the role of comorbid oppositional defiant disorder (ODD) and comorbid conduct disorder (CD) in ADHD was investigated by directly comparing 20 children with ADHD and 34 children with comorbid ADHD + ODD/CD. **Results:** ADHD was associated with EF deficits in inhibiting a prepotent response and verbal fluency. Children with HFA demonstrated deficits in all EF domains, except interference control and working memory. The HFA group showed more difficulties than the ADHD group with planning and cognitive flexibility. The comorbid ADHD + ODD/CD group did not show a distinctive pattern of performance on the EF tests compared to the ADHD group. **Conclusion:** The present study indicates that children with HFA exhibit more generalised and profound problems with EF tasks compared to children with ADHD. **Keywords:** ADHD, autism, executive functions.

Two major developmental disorders, attention deficit hyperactivity disorder (ADHD) and autism, have been associated with executive functioning (EF) deficits (Barkley, 1997a, b; Pennington & Ozonoff, 1996; Russell, 1997). Although there are many definitions of EF (Eslinger, 1996), executive functions (EFs) are commonly described as mental control processes that enable self-control (Denckla, 1996; Lezak, 1995; Pennington & Ozonoff, 1996) and are necessary to maintain an appropriate problem-solving set for the attainment of a future goal (Welsh & Pennington, 1988). EFs encompass different meta-cognitive domains such as response inhibition, working memory, cognitive flexibility (set shifting), planning, and fluency (Ozonoff, 1997; Pennington & Ozonoff, 1996; Reader, Harris, Schuerholz, & Denckla, 1994; Tranel, Anderson, & Benton, 1994).

Children with ADHD are characterised by symptoms of inattention, hyperactivity, and impulsivity (American Psychological Association [APA], 1994). The triad of characteristics that defines the syndrome of autism is the following: social abnormalities, communication abnormalities, and stereotyped repetitive patterns of behaviour (APA, 1994). There are different autism-like conditions, and these pervasive developmental disorders (PDD) are part of the broader phenotype of autism. Both ADHD and autism are characterised by behaviour similar to that found in patients with frontal lobe damage (Damasio & Maurer, 1978; Stuss & Benson, 1984). EFs are strongly related to the prefrontal cortex and its related networks (Cabeza & Nyberg, 2000; Fuster, 1997). Neural imaging studies show involvement of

prefrontal and connected structures in both ADHD (e.g., Faraone & Biederman, 1998; Hale, Hariri, & McCracken, 2000; Shaywitz, Fletcher, Pugh, Klorman, & Shaywitz, 1999) and autism (e.g., Bailey, Phillips, & Rutter, 1996; Chugani, 2000; Eliez & Reiss, 2000; Minshew, 1996). The above findings gave rise to the idea that ADHD and autism are associated with EF deficits.

Many theories consider an inhibition dysfunction as the core deficit in ADHD (Tannock, 1998). According to Barkley (1997a, b), poor behavioural inhibition is the central deficiency in ADHD. He argued that the inhibitory deficit causes secondary deficiencies in other EFs. Based on his theory, one might expect that children with ADHD would not only encounter problems with response inhibition but also with all other EFs, such as working memory, cognitive flexibility, planning, and fluency. On the basis of a review of studies of EF in ADHD, Pennington and Ozonoff (1996) concluded that the core problem in ADHD seems to be an inhibition deficit, and that the findings for other EFs were inconclusive.

Autism has been labelled as an executive disorder (Russell, 1997), because autism is associated with a number of EF deficits, especially in the domain of cognitive flexibility, planning, and working memory (Bishop, 1993; Hughes, Russell, & Robbins, 1994; Joseph, 1999; Ozonoff & Strayer, 1997; Robins, 1997). In contrast, inhibitory control seems to be relatively spared in autism (Ozonoff, 1997). Furthermore, Pennington and Ozonoff (1996) pointed out that the EF deficit in autism is more robust than

in ADHD, because the findings for deficits in different domains of EF were more conclusive.

If EF deficits are the primary and single cause of a disorder, this should imply that all children with this disorder have such deficits, that it is specific for this disorder, and that it is necessary and sufficient to cause the cardinal symptoms of the disorder (Pennington & Ozonoff, 1996). EF deficits are postulated as being the core cause in both ADHD and autism (e.g., Barkley, 1997a, b; Russell, 1997). This, so called, discriminant validity problem is partly solved, if there are differences in the kind of EF deficits or differences in the profoundness of a deficit in a particular EF domain. These differences might be a result of differences in the severity of brain damage or when or where the brain damage occurred. Pennington and Ozonoff (1996) postulated that childhood disorders could be distinguished by an EF profile (see also Ozonoff, 1997).

A plethora of studies have investigated EF deficits in ADHD and autism separately (see for reviews: Barkley, Grodzinsky, & Du Paul, 1992; Pennington & Ozonoff, 1996; Ozonoff, 1997; Sergeant, Geurts, & Oosterlaan, 2002). These reviews clearly show that findings are inconsistent for most EFs in both ADHD and autism. A direct comparison between children with ADHD and children with autism is needed to determine whether these two disorders differ in terms of their strengths and weaknesses on different domains of EF. The objective of the current study is to identify the combination of intact and deficient cognitive processes in ADHD and autism. This approach seeks to identify neuropsychological processes that can explain the nature of both ADHD and autism.

Two earlier studies have directly compared children with ADHD to children with autism on measures of EF (Nyden, Gillberg, Hjelmquist, & Heiman, 1999; Ozonoff & Jensen, 1999). Ozonoff and Jensen (1999) found a double dissociation between the two disorders. Children with autism showed difficulties in planning and cognitive flexibility, but not in inhibitory control, whereas children with ADHD showed the reverse pattern. Nyden et al. (1999) failed to replicate the double dissociation of Ozonoff and Jensen. Both ADHD and autism (Asperger syndrome) were associated with a response inhibition deficit and only children with ADHD showed deficits in flexibility. Both the Ozonoff and Jensen (1999) and the Nyden et al. (1999) studies show some methodological imperfections such as not excluding comorbid externalising disorders, not controlling for the non-EF demands of the EF tasks, and just covering a subset of EF domains. The current study improves on these imperfections.

The endeavour to identify a deficit that discriminates ADHD from autism requires that the groups chosen for study be clinically rigorously defined (Sergeant et al., 2002). Comorbid externalising disorders, including oppositional defiant disorder

(ODD) and conduct disorder (CD), are associated with EF deficits (e.g., Aronowitz et al., 1994; Hurt & Naglieri, 1992; Moffitt, Lynham, & Silva, 1994; Séguin, Boulerice, Harden, Tremblay, & Pihl, 1999; see for a review Sergeant et al., 2002). In order to conclude that possible EF deficits are related to symptoms of ADHD, comorbid externalising problems should be taken into account. The impact of comorbid ODD/CD on EF in children with ADHD is unclear. However, there is support for the idea that EF deficits in ADHD are independent of comorbid ODD/CD (Klorman et al., 1999; Nigg, Hinshaw, Carte, & Treuting, 1998). A recent study found evidence for EF deficits in children with ADHD with or without comorbid ODD/CD, but not in children with ODD/CD only (Clark, Prior, & Kinsella, 2000). These findings suggest that ADHD, but not ODD/CD, carries the EF deficit. Others have emphasised the importance of controlling for the comorbidity of ADHD with other externalising disorders (Pennington & Ozonoff, 1996; Oosterlaan, Logan, & Sergeant, 1998) because ODD or CD might enhance the EF deficits found. In the current study, we employed subgroup comparisons to study comorbidity in ADHD in terms of ODD/CD.

The current study improved on previous research by employing stringent controls for non-EF demands in the majority of the EF tasks. Performance on tests designed to measure EF are dependent on non-EF cognitive processes such as perception, attention, and memory (Eslinger, 1996). In order to conclude that poor performance on an EF task is due to an EF specific deficit, it is necessary to control for these non-EF demands (Denckla, 1996). Both the Ozonoff and Jensen study (1999) and the Nyden et al. study (1999) failed to control for non-EF demands in the EF measures used.

A unique feature of the current study is the broad array of EF domains that are covered. Pennington and Ozonoff (1996) suggested five EF domains: inhibition, working memory, planning, cognitive flexibility, and fluency. Most studies covered only a subset of the aforementioned EF domains.

To our knowledge the present study is the first study that directly compares children with ADHD and children with high functioning autism (HFA) on an extensive battery of EF measures that covers the major domains of EF. In the current paper, tasks were attributed to a given EF domain based on the original measurement purposes of the tasks and the nomenclature of EF domains as developed by Pennington and Ozonoff (1996, p. 53).

The current study has four goals. The first was to investigate the kind of EF deficits encountered by children with ADHD when compared to normal controls. One can argue that, according to Barkley's view (1997a, b), children with ADHD will encounter problems across all EF domains, because of their inhibitory control deficit, which leads to secondary impairments in all other EF domains.

The second goal was to find out which EF deficits children with HFA show in a direct comparison with normal controls. The EF hypothesis concerning the aetiology of autism (e.g., Russell, 1997) predicts that children with HFA will encounter problems across all EF domains. Other authors (e.g., Hughes et al., 1994; Joseph, 1999; Ozonoff & Strayer, 1997) predict specific deficits in cognitive flexibility, planning, and working memory, but not in inhibition (Ozonoff, 1997).

The third goal of the current study was to investigate whether children with ADHD and children with HFA differ in the severity and nature of their EF deficits by directly comparing these two groups of children to one another. Pennington and Ozonoff (1996) predicted that there would be a double dissociation between ADHD and HFA in relation to working memory and response inhibition. Children with ADHD will show inhibitory control deficits but will not encounter any difficulties with working memory tasks. Children with HFA are expected to show exactly the opposite pattern. In addition, children with HFA are expected to have more profound EF deficits, with the exception of inhibition, than children with ADHD (Pennington & Ozonoff, 1996; Sergeant et al., 2002).

Finally, the fourth goal of the study was to investigate whether it is possible to discriminate, at a neuropsychological level, children with ADHD from children with ADHD comorbid for ODD/CD. Children with ADHD + ODD/CD are at a greater risk than children with ADHD of having poor long-term outcomes and of having more profound neuropsychological deficits (e.g., Nigg & Hinshaw, 1998; Nigg et al., 1998). A direct comparison is needed to study whether ODD/CD enhance the EF deficits associated with ADHD (Oosterlaan et al., 1998). More profound deficits in the comorbid group would argue in favour of this hypothesis.

In sum, in the present study, profiles of EF deficits for both children with ADHD and children with HFA were investigated in a large group of school-aged children by comparing these two clinical groups with a normal control group on five major domains of EF: inhibition, visual working memory, planning, cognitive flexibility, and verbal fluency. This study also compared a subgroup of children with ADHD to a subgroup of children with comorbid ADHD and ODD/CD.

Method

Participants

Three groups of children participated in this study: 54 children with ADHD, 41 children with HFA, and 41 normal controls (NC). All children were in the age range of 6 to 13 years. The children were selected through a recursive multi-method selection procedure. First, the diagnostic instruments that were used in the selection

procedure will be reported. Second, the selection procedure for each group will be described.

Diagnostic measures

Child Communication Checklist (CCC, Bishop, 1998; Dutch translation: Hartman et al., 1998). The CCC was developed to measure aspects of communicative impairments and covers mainly the pragmatic skills necessary in the use of social language. The CCC contains 70 items which are scored on a four-point scale (*does not apply*, *applies somewhat*, *definitely applies*, and *unable to judge*). The items are grouped in nine scales: (a) speech output: intelligibility and fluency, (b) syntax, (c) inappropriate initiation, (d) coherence, (e) stereotyped conversation, (f) use of conversational context, (g) conversational rapport, (h) social relationships, and (i) interests. The pragmatic composite score is an overall measure of pragmatic skills and consists of the sum of the scores on scale c to g. Lower scores on the CCC indicate impairment. Adequate psychometric properties have been reported (Bishop, 1998; Bishop & Baird, 2001). The CCC was used here to assess the pragmatic abilities of the children.

Disruptive Behaviour Disorder rating scale (DBD, Pelham, Gnagy, Greenslade, & Milich, 1992; Dutch translation: Oosterlaan, Scheres, Antrop, Roeyers, & Sergeant, 2000). The DBD was developed to measure externalising disorders. The DBD contains 42 items that are scored on a four-point scale (*not at all*, *just a little*, *pretty much*, and *very much*). The questionnaire contains four scales composed of the DSM-IV items for ADHD Inattentive subtype, ADHD Hyperactive/Impulsive subtype, ODD, and CD. The higher the score on the DBD, the more impaired the child is. Adequate psychometric properties have been reported (Oosterlaan et al., 2000). The DBD was used to make a first selection for the study of the children with externalising disorders.

Diagnostic Interview Schedule for Children for DSM-IV, parent version (DISC-IV, National Institute of Mental Health [NIMH], Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000; Dutch translation: Ferdinand, Van der Ende, & Mesman, 1998). The DISC-IV is a structured diagnostic interview. The current version is based on the DSM-IV (APA, 1994) and the ICD-10 (WHO, 1992). The following sections were used: disruptive behaviour disorders (ADHD, ODD, CD), obsessive-compulsive disorder (OCD; part of the anxiety disorders section), and tic disorders (part of the miscellaneous disorders section). The latter two parts were administrated to exclude comorbid OCD and TS in the clinical groups. The interview implements a stringent diagnostic algorithm and generates a categorical classification. Based on this algorithm, clinical group membership was established. Adequate reliability and validity have been reported for earlier versions of the DISC-IV (Schwab-Stone et al., 1996).

Revised Autism Diagnostic Interview (ADI-R, Le Couteur et al., 1989; Lord, 1997; Lord, Rutter, &

Le Couteur, 1994; Lord, Storoschuk, Rutter, & Pickles, 1993). The ADI-R is a comprehensive semi-structured interview for parents or principal caregivers that probes for symptoms of PDD. The ADI-R focuses primarily on: (1) qualitative impairment in social interactions; (2) qualitative impairment in communication; and (3) restricted, repetitive, and stereotypic patterns of behaviours, interests, and activities. The ADI-R covers a variety of behaviours that frequently occur in PDD. Parent responses are coded on a 4-point scale according to the quality and severity of symptoms (0 = *normal for developmental level*, 3 = *severely autistic*). Scores are summed in each of the three domains listed above. If scores for all three domains reach specified cut-offs, and if there is evidence of developmental abnormality before the age of 36 months, a DSM-IV (APA, 1994) or ICD-10 (WHO, 1992) diagnosis of PDD is suggested. The ADI-R is currently considered as the 'gold standard' diagnostic instrument for PDD (Filipek et al., 1999). The ADI-R was administered to confirm the diagnosis of PDD in the PDD groups and to exclude PDD in the ADHD groups.

Selection of the groups

ADHD and ADHD + ODD/CD. Children were recruited from two samples: a sample of children from parents affiliated with the national parent association of children with ADHD, and a sample of children attending 11 special educational services for children with extreme behavioural problems (Scheres, Oosterlaan, & Sergeant, 2001). Only 2.2% of Dutch children aged 6 to 12 years attend these special educational services (Central Office for Statistics, personal communication). A recursive multi-method five-stage selection procedure was used. Note that for some schools the order of stage 2 and stage 3 (see below) was reversed.

At stage 1, approximately 940 parents were sent information on the study, an informed consent form, and a booklet containing, among other questionnaires, the CCC and the DBD. The parents of 358 children completed the questionnaires. At stage 2, 252 teachers completed the booklet with these same questionnaires. Children obtaining a score at or above the 95th percentile on at least one of the four scales of the DBD (by *both* parent *and* teacher ratings) were included in the third stage. Only those children that evidenced symptoms of ADHD in both home and school settings were selected for the current study. In this way the *DSM-IV* pervasiveness criterion (APA, 1994) for ADHD was met. Children that evidenced symptoms of ODD/CD both at home and at school were also selected. Children were required not to use any medication.

At the third stage, four subtests of the Revised Wechsler Intelligence Scale for Children (WISC-R) were administered to 185 children to assess intelligence. These tests were Vocabulary, Arithmetic, Block Design, and Picture Arrangement. The estimation of the IQ as obtained by these four subtests correlates between $r = .93$ and $r = .95$ with Full Scale IQ (FSIQ; Groth-Marnat, 1997). Fifty-one children were excluded from the study because their estimated FSIQ was below 80.

At stage 4, the PDISC-IV was administered. This structured interview was used to confirm the group

assignment based on the DBD and to exclude children with OCD and children with TS. On the basis of the PDISC-IV, 21 of children were excluded from the study.

At stage 5, the ADI-R was administered for the assessment of PDD. Children were excluded from the current study, if they had scores above the specified cut-off at two or more of the three specified domains, and if the developmental abnormalities started before the age of three. Based on the results of the ADI-R, 22 children were excluded. In total, 64 children were included in the ADHD-group.

HFA. Sixty-eight children were recruited from institutions specialising in the care of children with autism. The selection procedure for HFA children consisted of four stages. Only children who were already diagnosed with autism and did not use medication (or did use medication that could be discontinued) were included at the first stage. All 68 parents and teachers of these children completed the informed consent form and the booklets containing the same questionnaires as used for the ADHD and ADHD + ODD/CD group. Two of those 68 children were excluded because they had epilepsy.

Second, an estimation of the FSIQ was obtained in the same way as for the ADHD and ADHD + ODD/CD group described above. Twelve children with an FSIQ below 80 were excluded from the study. In the third stage, the diagnosis of autism was verified using the ADI-R and in the fourth stage the PDISC-IV was administered to exclude children with comorbid TS. One child was excluded based on the ADI-R. Nineteen children were excluded based on the results of the PDISC-IV. Eight of the children were excluded from the study because they showed symptoms of TS. Eleven children had characteristics of ADHD combined subtype. Furthermore, four children without a prior diagnosis of PDD were included in the HFA group and four children with a clinical diagnosis of ADHD were included in the HFA group because of the outcomes of the diagnostic interviews. In total, 42 children were included in the HFA group.

NC. Approximately 400 parents of children from four regular schools located throughout the country received the booklet containing the same questionnaires as used for the clinical groups. Parents of 165 children signed the informed consent form and completed the questionnaires. Children were excluded from the study if (1) the parent or the teacher stated that the child had ever received a clinical diagnosis (e.g., a behavioural problem or a learning disability); or (2) their FSIQ estimate was below 80 as measured with the short version of the WISC-R; or (3) the score on one of the four scales of the parent or teacher DBD exceeded the 75th percentile. Forty-four children met inclusion criteria for the NC group. Because only 29 of these children were boys, 12 boys from another research sample (Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2003) who were recruited according to the aforementioned procedure were added to the current research sample. In this way the NC group was equal in size to the HFA group. A large difference in number of participants between groups can influence the outcome of the group comparisons (for details see Stevens, 1996, p. 249).

For the purpose of this study, girls were excluded from the analyses for all groups. Data analyses are reported for 136 children; 54 were assigned to the ADHD group (16 inattentive subtype, 2 hyperactive/impulsive subtype, and 36 combined subtype; 34 children with ADHD had comorbid ODD or CD), 41 to the HFA group, 41 to the NC group.

Table 1 provides the gender composition, ages, estimated FSIQs, rating scale, and interview scores for the groups. Group differences for these measures were studied using an overall alpha level of .05. Groups did not differ with respect to age, $F(2,133) < 1$, $p = .64$, $\eta^2 = .01$. However, groups did differ with respect to FSIQ, $F(2,133) = 9.00$, $p < .001$, $\eta^2 = .12$. Children with HFA and ADHD children had lower IQs than NC ($p = .001$).

In general, findings for the rating scale scores support the behavioural distinctiveness of the groups. First, as expected, parents and teachers of the NC group reported fewer problems than the clinical groups for all rating scales (see Table 1).

Second, children with an ADHD diagnosis obtained higher scores on the parent and teacher DBD hyperactivity/impulsivity scale than the HFA group. On the PDISC-IV, the ADHD group obtained higher ratings for ADHD inattention and hyperactivity/impulsivity than the HFA group. Although the ADHD group obtained higher ratings on the DBD inattention scale than the HFA group, this difference only reached statistical sig-

nificance for the parent ratings, but not for the teacher ratings.

Third, regarding the measures of symptoms of autism, children with HFA were poorer in pragmatic language use on the parent and teacher CCC when compared to the ADHD group. Interestingly, not just the HFA group, but also children with ADHD were poorer in pragmatic language use than the NC group. As expected, on the ADI-R, the HFA group showed the most autistic characteristics when compared to the ADHD group. Fourth, the ADHD group showed more ODD characteristics than the HFA group. However, the two groups could not be differentiated from each other in terms of CD characteristics as measured by the parent and teacher DBD and by the PDISC-IV.

Neuropsychological measures

Both EF and non-EF control tasks were administered in this study (see Table 2). The EF tasks were selected to measure the domains of EF as suggested by Pennington and Ozonoff (1996, p. 53). For each task the original measurement goal is noted. The tasks are never 'pure' measures of a *single* EF domain but are related to a number of domains. For a number of domains more than one task was included to get converging evidence, for a deficit in that domain or the absence of a deficit in that domain, independent of the task chosen (e.g.,

Table 1 Group means and standard deviations for gender, age, IQ, rating scales, and clinical interviews

Measure	Groups						Contrasts
	NC		ADHD		HFA		
	M	SD	M	SD	M	SD	
Age	9.1	1.7	9.3	2.0	9.4	1.8	<i>ns</i>
FSIQ	111.5	18.0	99.5	11.5	98.3	18.4	ADHD, HFA < NC
DBD parent							
Inattention	3.1	3.4	17.4	4.7	13.4	5.2	ADHD > HFA > NC
Hyperactivity/Impulsivity	2.3	2.5	16.5	5.4	12.4	5.3	ADHD > HFA > NC
ODD	1.3	1.8	10.4	4.6	7.9	4.4	ADHD > HFA > NC
CD	.1	.3	3.1	3.5	2.5	3.4	ADHD, HFA > NC
DBD teacher							
Inattention	2.3	2.7	11.6	5.0	10.6	5.0	ADHD, HFA > NC
Hyperactivity/Impulsivity	1.4	2.0	12.5	5.8	8.0	5.6	ADHD > HFA > NC
ODD	.1	.4	7.7	5.3	4.8	4.0	ADHD > HFA > NC
CD	.1	.2	1.9	2.0	1.1	1.6	ADHD, HFA > NC
CCC parent							
Pragmatic score (C-G)	153.9	5.9	132.4	13.2	121.4	13.1	NC > ADHD > HFA
CCC teacher							
Pragmatic score (C-G)	153.8	6.7	142.5	11.2	129.1	12.8	NC > ADHD > HFA
PDISC-IV							
ADHD inattentive	–	–	14.9	3.0	9.3	4.8	ADHD > HFA
ADHD hyperactive	–	–	13.2	4.2	6.5	4.1	ADHD > HFA
ODD symptoms	–	–	4.3	2.4	3.0	2.2	ADHD > HFA
CD symptoms	–	–	.8	1.3	.5	.7	<i>ns</i>
ADI-R							
Social Interaction	–	–	4.3	3.6	18.2	5.1	HFA > ADHD
Communication	–	–	4.0	3.1	15.2	3.5	HFA > ADHD
Repetitive/Stereotyped	–	–	1.2	1.5	6.9	2.5	HFA > ADHD

Note: The number of subjects differs for each dependent variable due to missing data and exclusion of outliers (see text). NC = Normal Controls; ADI-R = Revised Autism Diagnostic Interview; ADHD = Attention Deficit Hyperactivity Disorder; CCC = Child Communication Checklist; CD = Conduct Disorder; DBD = Disruptive Behavior Disorder scale; FSIQ = Full Scale IQ; HFA = Higher Functioning Autism; ODD = Oppositional Defiant Disorder; PDISC-IV = Diagnostic Interview Schedule for Children.

Table 2 Overview of tasks and their dependent variables

Cognitive function	Tasks	Dependent measures
EF		
Inhibition	Change task Circle Drawing task Opposite Worlds of the TEA-Ch	SSRT Circle time difference TEA-Ch time difference
Working memory	Self-Ordered Pointing task	SoP beta errors
Planning	Tower of London	ToL beta score ToL beta decision time ToL beta execution time
Flexibility	Change task Wisconsin Card Sorting Test	Change MRT Change number of errors WCST percentage perseverative responses
Fluency	Verbal Fluency	Semantic number correct Letter number correct
Non-EF		
Response execution	Change task	MRT Response variability Number of errors
Short-term memory	Benton Visual Retention Test Corsi Block Tapping Test	BVRT number correct Corsi memory span
Categorisation	Categories of the SON-R	SON-R total score

Note: EF = executive function; BVRT = Benton Visual Retention Test; MRT = Mean Reaction Time; SON-R = Snijders-Oomen Non-verbal Intelligence Test; SoP = Self Ordered Pointing Task; SSRT = Stop Signal Reaction Time; TEA-Ch = Test of Every Day Attention for Children; ToL = Tower of London; WCST = Wisconsin Card Sorting Test.

inhibition, cognitive flexibility, and fluency). Note that for inhibition, three different tasks were included, each tapping a different form of inhibition (see Barkley, 1997a, 1997b; Nigg, 2001). Most of the tasks used here have been validated as measures of frontal lobe functioning, and prefrontal lobe functioning in particular, and were applied in recent EF research in at least one of the groups of interest.

EF tasks and dependent measures

Change Task (De Jong, Coles, & Logan, 1995; Logan & Burkell, 1986; Oosterlaan & Sergeant, 1998). The change task was included to measure: (1) inhibition of a prepotent response, (2) response execution, and (3) cognitive flexibility. The change task is an adapted version of the stop task. Several studies have found that performance on the stop signal task (Logan, 1994) is associated with right prefrontal cortex functioning (e.g., Band & Van Boxtel, 1999; Rubia et al., 1999).

The task consisted of two types of trials: go trials and stop trials. Trials were presented in blocks of 64 trials and the majority of the trials (75%) were go trials. Go trials required children to perform a two-choice reaction time task, the primary task. Subjects were required to indicate the position of an aircraft that was displayed to the left or right of a fixation point on a computer screen by pressing one of two buttons. Twenty-five per cent of the trials were stop trials. Stop trials were identical to go trials but in addition an auditory stop signal was presented, which directed children to (1) inhibit their response, and (2) immediately perform a different response, the change response. Stop signals were presented at four different 'stop signal intervals'. Specifically, tones were presented at 50, 200, 350 and 500 ms before the subject's expected response. The expected

moment of responding was estimated from the child's mean reaction time (MRT) in the preceding block of trials. A detailed description of the change task used in the present study is provided by Oosterlaan and Sergeant (1998).

The following dependent measures were derived from the change task: (1) Stop Signal Reaction Time (SSRT), a measure of the latency of the inhibitory process (Logan, 1994); (2) MRT, a measure of the latency of the response execution; (3) variability in the latency of the response execution process (response variability); (4) accuracy of responding as measures by the number of errors on the go trials (including both omission errors and commission errors); (5) change MRT as a measure of the latency of the set-shifting process; and (6) accuracy of cognitive flexibility (set shifting) as measured by the number of change response errors.

The reaction time data were processed as described by Oosterlaan et al. (1998; see also Oosterlaan & Sergeant, 1998). SSRT is an estimation of the stop signal reaction time and is not a directly observable measure like mean RT but is an estimation of the time a person needs to stop his or her response (Logan, 1994). SSRT calculations are based on the RT distribution and the probability of inhibition $p(i)$. MRT and $p(i)$ are both corrected on an individual basis. For each individual, RTs that were slower or faster than two SDs from the individual's mean were excluded for the calculation of the MRT. $P(i)$ was corrected for the number of non-responses using a formula described by Tannock, Schachar, Carr, Chajczyk, and Logan (1989). This is necessary because non-responses (omission errors) can occur on stop-trials and this will increase the probability of inhibition.

Circle Drawing Task (Bachorowski & Newman, 1985, 1990). The Circle Drawing Task was used as a

measure of inhibition of an ongoing response. The circle was 50.80 cm (20 in.) in diameter, drawn on a cardboard square, and covered with plexiglass. The circle had a small line indicating the starting and the finishing point of the tracing. The word START (in green ink) was printed on the right side of this line and the word STOP (in red ink) was printed on the left side. The task was administered under two conditions: first with neutral instructions ('trace the circle') followed by inhibition-instructions ('trace the circle again, but this time as slowly as you can'). A maximum of 12 minutes was allowed for both tracing conditions. Participants were not informed of this. The dependent variable in this task was the time used to trace the circle in the slow condition minus the tracing time in the neutral condition. The greater the time difference, the better a participant was able to inhibit (slow down) the continuous tracing response.

Opposite Worlds of the Test of Everyday Attention for Children (TEA-Ch; Manly et al., 2001). The Opposite Worlds is a subtest of the TEA-Ch. In the Opposite Worlds subtest the child is required to suppress an automatic or prepotent verbal response. This test is like the 'day' and 'night' test (Gerstadt, Hong, & Diamond, 1994; Passler, Isaac, & Hynd, 1985) where the children were also required to say the opposite of a logical response. There were two conditions in the TEA-Ch. First there was the Same World condition, where the child has to name the digits 1 and 2 that are scattered along a path. In the Opposite World condition the child was required to say 'one' when the child saw a 2 and 'two' when the child saw a 1. In this second condition, the child has to perform the task in a novel way and suppress the routine manner of performing it. The task was practised to make sure that the child had understood the task. There were four trials, first the Same World, followed twice by the Opposite World and finally the Same World was administered once again. The experimenter pointed with the index finger to the digits and the child was required to respond aloud. If the child committed an error, the experimenter did not move to the next digit until the child had corrected the error. The dependent variable was the difference between the mean time needed to complete the Opposite World conditions and the mean time needed to complete the Same World conditions.

Self-Ordered Pointing Task (Abstract Designs) (SoP, Petrides & Milner, 1982). The SoP was included to measure visual working memory capabilities. The SoP is one of the rare tests that have been validated as a relative selective frontal cortex measure, especially the mid-dorsolateral frontal cortex (Petrides, Alivisatos, Evans, & Meyer, 1993; Petrides & Milner, 1982).

Children were presented with four series of cards containing 6, 8, 10, and 12 abstract designs, respectively. The designs were relatively easy to distinguish from one another, but difficult to code verbally. The same set of designs was printed on each card, but the positions of these designs varied randomly from card to card. In the four series of cards, different designs were used. The 6 designs series was administered first, followed by the other series. For each series, children were presented with one card at a time. Children were

instructed to point to a different design on each of the cards. Children were informed that they could point to the designs in any order they wished, but without pointing to one of the designs more than once. Children were not allowed to respond to the same location on consecutive trials. Children were corrected when they made such a location repeat (but these location repeats were not counted as errors). Following the administration procedure of Petrides and Milner (1982), each series was presented three times in succession. Children were told to strive for accuracy and speed was not emphasised. A series of 3 designs was used for practice. Testing began only when subjects fully understood the instructions.

The demand on working memory increased as the number of designs on each card increased during the task. The dependent variable in this task was the number of errors (i.e., the number of times a design was responded to more than once). Difficulty level (6, 8, 10, 12 items) was taken into account in calculating the dependent variables. It was expected that there would be a linear relation between difficulty level and the dependent variables. Therefore, the regression coefficients (beta weight) for the dependent variable of the SoP was calculated for each individual, with difficulty level (four levels) being the predictor and number of errors the dependent variable. It was expected that, if children have a deficit in working memory, the regression coefficient for errors would be larger for such children compared to children without a working memory problem.

Tower of London (ToL; Krikorian, Bartok, & Gay, 1994). The ToL was selected to tap planning (Shallice, 1982). Several studies suggest that ToL performance relies heavily on frontal cortex functioning, especially the left frontal cortex (e.g., Baker et al., 1996; Dagher, Owen, Boecker, & Brooks, 1999; Rowe, Owen, Johnsrude, & Passingham, 2001).

Materials and procedures for administration and scoring were derived from Krikorian et al. (1994). Starting from a fixed arrangement of the three coloured balls (red, blue, and yellow) on two of the three pegs, the child was required to copy a series of depicted end-states by rearranging the balls. Upon presentation of a problem, participants were informed of the number of moves required to solve that problem correctly. A problem was solved correctly when the end-state was achieved in the required number of moves while avoiding errors. Children were encouraged not to initiate the first move until they were confident that they could execute the entire sequence of moves to solve the problem. A practice problem was presented to familiarise the child with the task. Thereafter, 12 problems of graded difficulty were presented. The demand for planning was manipulated by presenting problems that differ in the minimum number of moves required for solution. A maximum of three trials was allowed to solve each problem. Children were told to strive for accuracy.

Three measures were derived. The main dependent variable was the ToL score, which was calculated by assigning points based on the number of trials required to solve a problem. Three points were given if the problem was solved on the first trial, two points for successful solution on the second trial, and one point

for successful solution of the third trial. There were three difficulty levels: 2 or 3 moves necessary to solve the problem (lowest difficulty level), 4 moves (medium difficulty level), and 5 moves (highest difficulty level). Total item scores were calculated for each of the three difficulty levels. The maximum ToL score for each level of difficulty was 12 points.

Two temporal measures were derived for each level of difficulty: (1) planning time, which is the time between the presentation of a problem and the initiation of the first move on a trial (ball leaves peg), and (2) execution time, which is the time between the initiation of the first move and the completion of the final move on a trial. These measures were derived for the first attempt on each problem. The two temporal measures were applied as background variables in order to be able to interpret whether there was a difference in cognitive style or strategies during the task. Like in the SoP, difficulty level was taken into account in calculating the dependent measures. Again, it was expected that there would be a linear relation between difficulty level and the dependent variables. Therefore, the regression coefficients (beta weights) for the three dependent variables were calculated for each individual, with difficulty level (low, medium, and high) being the predictor, and ToL score, planning time, and execution time being the dependent variables, respectively.

Wisconsin Card Sorting Test (WCST, Grant & Berg, 1948; Heaton, 1981; Heaton, Chelune, Talley, Kay, & Curtiss, 1993). The WCST is a widely used measure to tap cognitive flexibility or set shifting. Several studies have found that WCST performance relies on the right dorso-lateral frontal cortex (e.g., Lombardi et al., 1999; Riehemann et al., 2001). However, some studies failed to find a neurological basis for performance on the WCST (e.g., Chase-Carmichael, Ris, Weber, & Schefft, 1999).

The paper and pencil version of Grant and Berg (1948) was used here (see Heaton, 1981; Heaton et al., 1993). The dependent variable of interest was the percentage of perseverative responses. These percentages were calculated from the number of trials in which the child continued sorting by a previously correct rule despite negative feedback, and the total number of cards the child needed to complete the task. A perseverative response can be (1) an ambiguous answer; the stimulus card and response card match not only the correct sorting principle but also the perseverative principle; (2) a perseverative error; the child sorts according to the previous rule. A computer-based scoring program was used to calculate the dependent variables (Harris, 1990).

Verbal Fluency (Benton & Hamsher, 1978). An adaptation of the Controlled Word Association Task (COWAT) was used to measure the capacity to generate novel responses. Several studies have shown that verbal fluency tends to be associated with left prefrontal functioning (e.g., Frith, Friston, Liddle, & Frackowiak, 1991; Gaillard et al., 2000).

Children were required to name as many examples of a particular category as possible within a time limit of one minute. The categories were items from the semantic categories 'animals' and 'food', as well as

words beginning with the letters K and M. Children were instructed to exclude names of persons and the same word with a different suffix. If incorrect words were given, the children were briefly reminded of the rules. The dependent measures in this task were the total number of admissible words across the semantic categories 'animals' and 'food', as well as across the letters K and M.

Non-EF control tasks and dependent measures

Benton Visual Retention Test (BVRT, Sivan, 1992). The BVRT measures visuo-spatial abilities and immediate spatial memory abilities. This task was included to control for visual short-term memory in the SoP. The BVRT (form C) consists of 10 designs with each design containing one or more figures. Each of these designs was presented to the child for 10 seconds. The child was then required to reproduce the designs immediately after presentation of the designs (method A for administration). The number of correct designs was the dependent measure in this task (Sivan, 1992; Lezak, 1995).

Corsi Block Tapping Test (Corsi, 1972; Lezak, 1995; Milner, 1971; Schellig, 1997). The Corsi Block Tapping test (Corsi) was designed to test memory impairments in patients with temporal lobe damage. The test taps visuo-spatial memory-span (Berch, Krikorian, & Huha, 1998; Della Salla, Gray, Baddeley, Allamano, & Wilson, 1999) and was included to control for visual short-term memory in the SoP. The Corsi requires maintenance of spatial information but does not involve many explicit concurrent processing requirements, although the visuo-spatial sketchpad seem to be closely related to the central executive (Miyake, Friedman, Rettinger, Shah, & Hegarty, 2001). A detailed description of this task is provided by Schellig (1997). In short, in this task the child had to begin to copy a 3-block item, and the number of items was increased by one after a particular difficulty level was successfully completed. There were three trials for each difficulty level. The test ended after three consecutive errors within a particular difficulty level or after the 8-block items were administered. The dependent variable was the visual memory span of the child, which is defined as the difficulty level for which the child was able to finish at least two trials successfully.

Revised Wechsler Intelligence Scale for Children (WISC-R). Four subtests of the WISC-R were administered to all children: Vocabulary, Arithmetic, Picture Arrangement, and Block Design. These four subtests were used to estimate FSIQ.

Categories of the Snijders-Oomen Non-Verbal Intelligence Test Revised (SON-R 51/2-17; Snijders, Tellegen, & Laros, 1989; Tellegen & Laros, 1993). Categories is one of the subtests of the SON-R and measures semantic memory and the ability to categorise. This test was included for two reasons. First, Categories was used to control semantic memory capacities in verbal fluency. In previous research the fluency task has been used not only for tapping EF, but

also as a semantic memory task (e.g., Elwood, 1997; Rosen, 1980). Second, Categories was included to control for the ability to categorise, which is required in the WCST (Grant & Berg, 1948; Heaton, 1981).

In Categories the child was first shown three pictures and had to decide what the three pictured objects had in common. Next, five pictures were presented to the child and the child was required to choose those two pictures that depicted the same concept. After practising, a maximum of 27 items was administered. Items were divided into three different series. Each series was terminated when the child made two consecutive errors. The dependent variable was the number of correct items.

Procedure

All children were tested individually. Testing took place on three different occasions and tests were administered in a fixed order. During the first session, the WISC-R was administered. At the second testing session, the Circle Drawing Task, SoP, Verbal Fluency, WCST, and the BVRT were administered. One week later, the Change task, Corsi, Categories, ToL, TEA-Ch Opposite World, and the Beery were administered. Fifty-eight children from the clinical groups were on methylphenidate, but discontinued medication at least 20 hours prior to testing (Barkley, DuPaul, & Connor, 1999), allowing for a complete wash-out (Greenhill, 1998). Children discontinued the use of methylphenidate after their morning dose on the day before testing. All children received a small gift (worth approximately 1 USD) at the end of the study. The parents or caregivers were sent detailed reports on their child's performance on the tests.

Statistical analyses

Analyses focused on three group contrasts: (1) NC versus ADHD, (2) NC versus HFA, and (3) ADHD versus HFA. These three contrast analyses were performed separately for each EF domain. The alpha level was adjusted to compensate for the number of comparisons made. For each contrast, alpha was set at .01.

First, correlations were calculated between the dependent variables of the EF and non-EF tasks to investigate whether it is possible to reduce the number of dependent variables by creating composites.

Second, the dependent measures (EF and non-EF) were analysed using ANOVAs with group (3 levels) as the between-subject factor. When for one task there was more than one dependent variable, MANOVAs were conducted instead of ANOVAs.

Third, groups were compared on the EF measures, while controlling for FSIQ, age, and for performance on the non-EF control measures, with ANCOVAs and MANCOVAs. FSIQ was controlled for because there were significant group differences for FSIQ. Age was controlled for because EFs are still developing in the age range 6–12 and this might influence the outcome despite the fact that there were no group differences for age. In the current study, we did not control for ODD and CD in our group comparisons because by covarying ODD and CD we would remove a portion of variance that is actually associated with ADHD (Angold, Costello,

& Erkanli, 1999; Thapar, Harrington, & McGuffin, 2001). The correlation between ratings on the DBD ADHD-scales and DBD ODD-scale ranged from $r = .64$ to $r = .75$, and between the DBD ADHD-scales and the DBD CD-scale the correlations ranged from $r = .37$ to $r = .51$. Moreover, the level of ODD symptoms in the HFA group is also higher than in the NC group. The pattern of negative, hostile, and defiant behaviour that characterises ODD (APA, 1994) can also occur in children with HFA. By covarying for ODD we would remove a portion of variance that might be also specifically associated with HFA. In order to address the impact of comorbid ODD and CD in children with ADHD, we present an exploratory analysis in which children with ADHD and children with ADHD + ODD/CD were compared with one another.

Fourth, exploratory discriminant analyses were performed to investigate the contribution of EF and non-EF measures to possible differences between clinical groups and the NC group. In these analyses, dependent variables from the EF and non-EF tasks were used to predict group membership.

Missing data and outliers

Technical difficulties or the child refusing to do the task led to missing data. For each of the three groups and for each dependent measure, children with extreme scores were identified and discarded. Extreme scores were values more than three box plot lengths from the upper or lower edge of the box. In the MANOVAs and MANCOVAs only those participants were excluded that had extreme scores for more than one of the dependent measures. The number of missing cases (missing data and/or extreme cases) for the analyses ranged from zero to seven.

Results

The results of the data analyses are presented in Tables 3 and 4.

Correlations between dependent measures

The mean correlation between the dependent variables as derived from the EF tasks was rather low ($r = .15$, range $r = |.001| - r = |.63|$). There was little common variance between the EF-variables. This might indicate that the EF domains are more fractionated than unitary when measured with the current tasks. The mean correlation between the dependent variables as derived from the non-EF tasks was moderate ($r = .42$, range $r = |.26| - r = |.83|$). This implies that tasks within the non-EF domain share some variance. The mean correlation between the EF variables and non-EF variables was low ($r = .22$, range $r = |.02| - r = |.55|$), indicating that the EF domain and the non-EF domain are distinguishable from each other. However, the pattern of correlations does not demonstrate unequivocally that the EF and non-EF domains are independent, because the correlation between the EF and non-EF

Table 3 Group means and standard deviations for executive function tasks

Measure	Groups						Contrasts
	NC (<i>n</i> = 41)		ADHD (<i>n</i> = 54)		HFA (<i>n</i> = 41)		
	M	SD	M	SD	M	SD	
Inhibition							
SSRT	237.2	72.4	320.6	95.5	317.0	134.5	ADHD, HFA > NC
Circle time difference	104.4	76.2	92.5	80.7	58.4	55.5	HFA < NC
TEA-Ch time difference	4.0	3.0	3.9	2.9	5.4	4.3	<i>ns</i>
Working memory							
SoP beta errors	1.3	.7	1.4	1.0	1.8	1.0	<i>ns</i>
Planning							
ToL beta score	-1.2	.8	-1.7	1.7	-1.7	1.4	<i>ns</i>
ToL beta decision time	1.8	3.4	.6	2.4	.0	3.8	<i>ns</i>
ToL beta execution time	3.3	1.8	3.1	1.8	5.0	2.9	HFA > ADHD, NC
Flexibility							
Change MRT	514.2	76.4	553.7	89.6	586.9	120.5	HFA > NC
Change number of errors	7.4	7.2	11.6	9.1	12.1	9.8	<i>ns</i>
WCST percentage perseverative responses	14.1	6.9	16.2	8.0	22.4	11.7	HFA > ADHD, NC
Fluency							
Semantic number correct	33.8	7.7	29.3	7.9	26.5	8.0	ADHD, HFA < NC
Letter number correct	17.9	6.0	12.6	6.3	11.6	6.3	ADHD, HFA < NC

Note: The number of subjects differs for each dependent variable due to missing data and exclusion of outliers (see text). ADHD = Attention Deficit Hyperactivity Disorder; HFA = Higher Functioning Autism; NC = Normal Controls; MRT = Mean Reaction Time; SoP = Self Ordered Pointing Task; SSRT = Stop Signal Reaction Time; TEA-Ch = Test of Every Day Attention for Children; ToL = Tower of London; WCST = Wisconsin Card Sorting Test.

Table 4 Group means and standard deviations for control tasks

Measure	Groups						Contrasts
	NC		ADHD		HFA		
	M	SD	M	SD	M	SD	
Response execution							
MRT	487.1	100.7	498.0	129.4	536.5	126.7	<i>ns</i>
Response variability	112.2	39.3	138.7	55.9	144.2	51.1	HFA > NC
Number of errors	4.6	5.3	8.7	8.5	9.7	9.4	HFA > NC
Short-term memory							
Corsi memory span	5.0	.8	4.6	1.0	4.4	.9	HFA < NC
BVRT number correct	6.1	1.9	5.4	2.5	5.0	1.7	<i>ns</i>
Categorisation							
SON-R total score	13.7	5.4	11.1	4.2	11.7	4.0	ADHD < NC

Note: The number of subjects differs for each dependent variable due to missing data and exclusion of outliers (see text). ADHD = Attention Deficit Hyperactivity Disorder; BVRT = Benton Visual Retention Test; Corsi = Corsi Block Tapping Test; HFA = Higher Functioning Autism; MRT = Mean Reaction Time; NC = Normal Controls; SON-R = Snijders-Oomen Non-verbal Intelligence Test Revised.

measures is significantly higher ($r = .22$) than among the EF measures ($r = .15$). Moreover, the correlations do not justify the use of composite scores for one of the EF domains under study. Inspection of the correlation matrices for each of the groups separately did not alter our conclusions concerning the pattern of findings (ADHD: EF $r = .18$, non-EF $r = .41$, EF with non-EF $r = .24$; HFA: EF $r = .17$, non-EF $r = .35$, EF with non-EF $r = .22$; NC: EF $r = .19$, non-EF $r = .44$, EF with non-EF $r = .26$).

ADHD, HFA, and NC group comparisons

EF domains

Inhibition. As predicted, there was a main effect of group for SSRT, $F(2,129) = 8.78$, $p < .001$, $\eta^2 = .12$.

Contrast 1 and 2 showed that both the ADHD group and the HFA group had slower SSRTs compared to the NC group (both $p < .001$). The two clinical groups could not be differentiated from one another in terms of SSRT (contrast 3). These results were unchanged by covarying age and FSIQ.

A marginally significant main effect of group was found for inhibition time (circle time difference) as measured by the circle drawing task, $F(2,133) = 4.32$, $p = .015$, $\eta^2 = .06$. Contrast 2 showed that HFA children had smaller difference scores, indicating more problems with inhibition of an ongoing response than NC ($p = .006$). The other two contrasts did not reach statistical significance (contrast 1 and 3). Controlling for age and FSIQ did not alter the results.

There was no significant group difference for the interference score (time difference) as measured by the TEA-Ch, $F(2,132) = 2.61$, ns , $\eta^2 = .04$. Covarying age and FSIQ did not alter these findings.

Working memory. None of the groups could be differentiated from each other with respect to SoP beta errors, $F(2,136) = 3.63$, ns , $\eta^2 = .05$. This indicates that the increase in errors with increasing levels of difficulty on the SoP did not differ between the groups. These results were unchanged by covarying non-EF task performance (both BVRT and Corsi block tapping), age, and FSIQ.

Planning. A significant overall group effect was noted for the three dependent measures from the ToL, Wilks' $\Lambda = .84$, $F(6,260) = 3.87$, $p < .002$, $\eta^2 = .08$. Subsequent ANOVAs showed that increasing planning load did not differentiate between the groups on ToL beta score, $F(2,135) = 1.24$, ns , $\eta^2 = .02$ and ToL beta decision time, $F(2,136) < 1$, ns , $\eta^2 = .04$. In contrast, a significant group effect was evident for the ToL beta execution time, $F(2,135) = 10.27$, $p < .001$, $\eta^2 = .13$. Contrasts showed that children with HFA did need more execution time with increasing difficulty level to reach the same score as both NC and children with ADHD (contrast 2: $p = .001$, contrast 3: $p < .001$). Children with ADHD could not be differentiated from NC (contrast 1). The same results were obtained after covarying age and FSIQ.

Cognitive flexibility. There was a significant main effect of group among the two dependent measures regarding cognitive flexibility of the change task, Wilks' $\Lambda = .89$, $F(4,258) = 3.82$, $p < .01$, $\eta^2 = .06$. Subsequent ANOVAs revealed a significant group effect for change MRT, $F(2,133) = 5.72$, $p < .005$, $\eta^2 = .08$, but no significant effect of group for the numbers of errors, $F(2,133) = 3.69$, ns , $\eta^2 = .05$. Children with HFA were slower in their change MRT than controls ($p = .001$, contrast 2). The other two contrasts (1 and 3) for change MRT did not reach statistical significance. The MANCOVA, with age and FSIQ as covariates, revealed the same pattern of results.

The other cognitive flexibility task, the WCST, showed a significant main group effect for the percentage of perseverative responses, $F(2,130) = 9.12$, $p < .001$, $\eta^2 = .13$. Comparing the ADHD group and NC group did not reach statistical significance (contrast 1). Children with HFA had a higher percentage of perseverative responses than NC (contrast 2: $p = .001$). This was specific to the HFA group, since the HFA group had a higher percentage of perseverative responses than the ADHD group ($p = .001$, contrast 3). These results did not alter after covarying non-EF demands, age, and FSIQ.

Fluency. A significant main effect of group was evident among the two dependent measures of verbal fluency, Wilks' $\Lambda = .81$, $F(4,262) = 7.20$, $p < .001$,

$\eta^2 = .10$. Both the group effect for the semantic category, $F(2,135) = 8.93$, $p < .001$, $\eta^2 = .12$, and for the letter category, $F(2,135) = 12.61$, $p < .001$, $\eta^2 = .16$, were significant. For the semantic category, both the ADHD group and the HFA group gave fewer correct responses than the NC group (contrast 1: $p = .007$, contrast 2: $p < .001$) but the two groups could not be differentiated from each other (contrast 3). For letter fluency, a similar pattern of results was obtained (contrast 1: $p < .001$, contrast 2: $p < .001$, contrast 3: ns). Problems with generating novel responses in the verbal fluency tasks did not seem to be specific for one of the clinical groups. In general, the pattern of findings did not alter after covarying non-EF (SON-R), age, and FSIQ except for contrast 1 for the semantic category, which was no longer statistically significant. This alteration was due to covarying both FSIQ ($p = .001$) and age ($p = .009$), but not to the non-EF measure ($p = .050$).

The pattern results of the group comparisons on the EF-domains hardly altered when children with ADHD inattentive subtype were excluded from the analysis. The main difference was that on the WCST the ADHD group and the HFA group could no longer be differentiated from each other ($p = .022$).

Non-EF domains

Response execution. The MANOVA revealed a significant main effect of group on MRT, variability of responding, and number of errors, Wilks' $\Lambda = .86$, $F(6,256) = 3.48$, $p < .005$, $\eta^2 = .08$. There was no statistically significant effect for the speed of responding (MRT), $F(2,133) = 1.91$, ns , $\eta^2 = .03$. In contrast, for response variability and the number of errors there were significant group effects, $F(2,133) = 4.85$, $p < .01$, $\eta^2 = .07$ and $F(2,133) = 4.74$, $p = .01$, $\eta^2 = .07$, respectively. Compared with NC, HFA children showed greater variability in the speed of responding (contrast 2: $p = .005$) and were less accurate (number of errors: $p = .005$). After covarying age and FSIQ these findings fell shy of significance. The other contrasts were not significant.

Short-term memory. For the measures of visual short-term memory there were no significant group differences on the BVRT, $F(2,136) = 2.89$, ns , $\eta^2 = .04$ and a marginally significant main effect of group on the Corsi, $F(2,135) = 4.34$, $p = .015$, $\eta^2 = .06$. The HFA group performed more poorly than the NC group (contrast 2: $p = .004$). No other group differences were found (contrasts 1 and 3). After covarying age and FSIQ none of the contrasts was significant.

Categorisation. A marginally significant group effect was found for the categorisation task of the SON-R, $F(2,135) = 4.11$, $p = .019$, $\eta^2 = .06$. Children with ADHD made fewer correct responses on this task than NC (contrast 1, $p = .006$). None of the

other contrasts revealed a statistically significant difference between groups. After covarying age and FSIQ none of the contrasts was statistically significant.

Exploratory discriminant analysis

A discriminant analysis was conducted to determine whether a combination of EF and non-EF measures could predict group assignment. With a total number of 136 participants, the number of dependent variables to be entered was limited to a maximum of seven in order to obtain stable solutions for the discriminant functions (Stevens, 1996, p. 265). However, nine cases were excluded because one of the dependent variables was missing and, therefore, just six dependent variables were entered as a predictor. For each EF domain one dependent variable was selected. When a task or a domain generated more than one dependent variable, that dependent variable was selected which showed the most robust differences in the group comparisons. In the case of the verbal fluency measures, both measures were equal in discriminating between the groups. Therefore, an aggregated score was obtained by averaging the *z*-scores of these two dependent measures in this task. A *z*-score was calculated for each of the non-EF measures and an average of these *z*-scores was entered in the discriminant analysis. The *z*-scores of the following variables were included as predictors: SSRT, SoP beta errors, ToL beta execution time, WCST percentage perseverative responses, aggregated verbal fluency score, and aggregated non-EF score. The overall Wilks' lambda was significant, $\Lambda = .71$, $\chi^2(12, N = 127) = 42.06$, $p < .001$, indicating that the predictors differentiated between the three groups. In addition, the residual Wilks' lambda was significant, $\Lambda = .88$, $\chi^2(5, N = 127) = 16.20$, $p = .006$. This test indicated that the predictors differentiated significantly between the three groups on the second discriminant function, after taking account of the effects of the first discriminant function. The first discriminant function was strongly related to all measures except SSRT. However, SSRT showed the strongest relationship to the second discriminant function. The HFA group had the highest mean score ($M = .69$) on the first discriminant function, while the ADHD ($M = -.009$) and NC group ($M = -.55$) had lower mean scores. On the other hand, the NC group had the highest mean scores on the second discriminant function ($M = .38$), followed by HFA ($M = .23$) and ADHD ($M = -.44$).

Sixty-one per cent of the cases were correctly classified: 55% of the NC, 69% of the ADHD children, and 54% of the HFA children. In order to take into account chance agreement, the kappa coefficient was computed and a value of .40 was obtained. This indicates that the group prediction is moderately accurate. A value of 1 for Kappa indicates perfect

prediction, while a value of 0 indicates chance-level prediction. To determine how well the discriminant functions would predict a new sample, the percentage of children classified accurately was estimated using the cross-validation leave-one-out technique. With this technique, classification functions are derived based on all cases except one, after which the omitted case is classified. These results can be used to estimate how well the discriminant functions would predict a new sample. Fifty-six per cent were classified correctly.

When only the ADHD group and the HFA group were included in the discriminant analyses, 71% of the children were correctly classified (69% after cross-validation). The kappa coefficient indicated that the group prediction was moderately accurate with a value of .38. However, only 89 children were included in this analysis using 6 predictors. The ratio of the total sample size to the number of predictors is quite large and could lead to low reliability of the discriminant functions obtained. Therefore, these results need to be interpreted very cautiously.

Exploratory group comparisons: ADHD compared to ADHD + ODD/CD

To investigate whether children with ADHD and children with comorbid ADHD and ODD/CD differ from each other on the neuropsychological measures used in the current study, the ADHD group was split into two groups. Twenty children were diagnosed as ADHD only and 34 children were diagnosed as ADHD with comorbid ODD/CD. The mean age of both groups was 9.4 years ($SD = 1.95$) and 9.3 years ($SD = 1.9$), respectively, and mean estimated FSIQ was 99.0 ($SD = 12.3$) and 99.8 ($SD = 11.3$), respectively. The groups did not differ with respect to age ($F(1, 52) < 1$) or FSIQ ($F(1, 52) < 1$). Furthermore, as expected, the ADHD group obtained lower PDISC-IV scores than the ADHD + ODD/CD group for inattention, ODD, and CD. Although groups differed from each other on a behavioural level, there were no group differences for any of the EF ($.06 < p < .58$, range $\eta^2 .001-.063$) and non-EF measures ($.19 < p < .95$, range $\eta^2 .001-.033$).

Discussion

The major goal of the current study was to investigate if two major childhood disorders, HFA and ADHD, could be discriminated in terms of their profile of EF strengths and weaknesses. The secondary goal was to investigate whether comorbid ADHD + ODD/CD is a more severe form of ADHD.

The current results suggest that the hypothesised EF profiles for different clinical groups (Pennington & Ozonoff, 1996) may be less straightforward than anticipated. In line with the predictions, both the ADHD and the HFA groups exhibited EF deficits.

However, the results for the ADHD group do not support the theory of Barkley (1997a, b). Based on Barkley's model, it could be argued that children with ADHD have problems in all EF domains and that these difficulties would be specific for ADHD. The ADHD group showed difficulties in tasks related to only two areas of EF: inhibiting a prepotent response and verbal fluency. Moreover, according to Barkley an inhibition deficit is central to ADHD. Although an inhibition deficit for ADHD was found in the present study (like in, e.g., Nigg, 2001; Oosterlaan et al., 1998), this deficit was not specific to ADHD. Children with HFA also showed deficits in two domains of inhibition: inhibition of a prepotent response and inhibition of an ongoing response. Furthermore, the verbal fluency finding was not specific for ADHD (see also Sergeant et al., 2002).

The findings for the HFA group are partly in line with the EF hypothesis concerning the aetiology of autism (e.g., Russell, 1997). The prediction was made that children with HFA will encounter problems across all EF domains. The results indicated that children with HFA have difficulties in inhibiting a prepotent response, inhibiting an ongoing response, planning, cognitive flexibility, and verbal fluency. Contrary to predictions, children with HFA did not show problems on the working memory task. Another study also showed that children with autism could not be differentiated from NC on a broad range of working memory tasks (Ozonoff & Strayer, 2001).

As postulated by Pennington and Ozonoff (1996), the EF deficit was most pronounced in children with HFA. However, the hypothesis of a double dissociation between ADHD and HFA was not confirmed here. Only two EF measures clearly discriminated between ADHD and HFA. Compared to children with ADHD, the HFA group showed more difficulties with cognitive flexibility and planning. The deficits in cognitive flexibility for children with HFA might be related to the stereotyped repetitive patterns of behaviour that are characteristic for autism (Happé & Frith, 1996) but not for ADHD. The present findings indicate that it is difficult to differentiate children with ADHD and children with HFA on the EF measures used here, although there are differences in both the quantity and the quality of the EF deficits across groups. The exploratory discriminant analysis was in line with these findings, because this analysis suggested that the EF measures in the present study are of modest utility in case identification in this sample (see, for similar results with inhibition measures, Nigg, 1999).

Although the non-EF domain was not a central part of the current investigation, it is noticeable that children with HFA encountered more difficulties with the non-EF tasks than children with ADHD. In the current study, HFA seems to be related to response-variability in particular. In the ADHD literature, the response-variability of children with ADHD is a

robust finding (Oosterlaan et al., 1998). Castellanos and Tannock (2002) recently argued that the essence of ADHD might be the temporal and contextual variability in symptom expression and performance. This variability might be related to difficulties with temporal processing and this, in turn, might be related to cerebellar dysfunctioning (Castellanos et al., 2002). However, Rutter and Bailey (1999) argued that one of the key features of the social abnormalities seen in autism is their timing, and also autism has been associated with cerebellar dysfunctioning (e.g., Courchesne et al., 1994). This implies that, in contrast to the hypothesis of Castellanos and Tannock (2002), response-variability may not be specific to ADHD.

The results of the current study are partly in line with those of Ozonoff and Jensen (1999) and Nyden et al. (1999). Ozonoff and Jensen concluded that children with autism have deficits in planning and flexibility and children with ADHD do not have such deficits but do show an inhibition deficit. In the Nyden et al. study, only ADHD was associated with deficits in cognitive flexibility and both groups showed deficits in response inhibition. Apart from the fact that the studies differ slightly in the type of tasks used and reported dependent measures, the current study and the two related studies mentioned above differ mainly in the way they dealt with comorbidity.

It is unclear whether findings of a number of studies may be explained in terms of comorbid disorders, such as ODD or CD (Pennington & Ozonoff, 1996; Sergeant et al., 2002). In the current study, the effect of comorbidity between ADHD and ODD or CD was addressed by comparing an ADHD-only with a comorbid ADHD + ODD/CD group. The ADHD + ODD/CD group did not show a distinctive pattern of performance on the EF tests in comparison with the ADHD group. This is in line with previous results in a meta-analysis in which children with ADHD only could not be differentiated from children with comorbid ADHD + ODD/CD on a task requiring inhibition of a prepotent response (Oosterlaan et al., 1998). Based on the current study, there is no evidence to support a distinction between ADHD and comorbid ADHD + ODD/CD. Interestingly, this conclusion is also in line with recent genetic research (Thapar et al., 2001). The findings of the current study, the Ozonoff and Jensen study (1999), and the Nyden et al. study (1999) are unlikely to be due to comorbid ODD or CD, but might be related to another possible impurity of their ADHD groups, namely PDD characteristics within the ADHD groups.

A related issue is how ADHD and autism may or may not overlap in their behavioural characteristics. Jensen, Larrieu, and Mack (1997) showed that children with PDD could not be differentiated from children with ADHD on scales related to hyperactivity and acting-out behaviour. The clinically

relevant group of children with PDD and ADHD has been underexposed in the recent literature. It is known that these two disorders often co-occur (Cohen & Volkmar, 1997; Ghaziuddin 2002). In the current study, of the 86 children with a prior clinical diagnosis of ADHD, 22 children were excluded because of PDD as measured with the ADI-R. This indicates that one-third of the original ADHD sample showed characteristics of the triad of behavioural symptoms of autism. This demonstrates that in clinical practice children with PDD often receive the diagnosis of ADHD. The presence of ADHD in a PDD sample, or PDD in an ADHD sample, may cause inconsistent results across studies. An interesting avenue for future research would be to investigate whether ADHD characteristics in children with PDD influence achievement on neuropsychological tasks.

The overlap between symptoms of ADHD and autism, the large comorbidity, and the finding that a given disorder may be a risk of developing another disorder, are all indications that there is a strong relationship between autism and ADHD (Bradshaw & Sheppard, 2000). In line with Pennington and Ozonoff (1996), Bradshaw and Sheppard (2000) argued that both autism and ADHD are neurodevelopmental fronto-striatal disorders. The fronto-striatal system encompasses the dorsolateral prefrontal cortex, lateral orbitofrontal cortex, anterior cingulate, supplementary motor area, and associated basal-ganglia structures. Bradshaw and Sheppard hypothesised that in ADHD other parts of the circuit may be disrupted than in autism. The robustness and profoundness of the EF deficits in autism might be due to a more severe disruption of the fronto-striatal system than in ADHD. Currently, a strong conclusion cannot be drawn concerning the specific dysfunctions of the fronto-striatal circuit in ADHD and autism (see for a review Eliez & Reiss, 2000). This is an area in need of urgent neuropsychological research.

The current study has some caveats. Within the field of EF it is known that operationalising the EF domain has a number of limitations. First, at the theoretical level, distinct relationships between the five EF domains have been claimed (e.g., Barkley, 1997a, b; Fuster, 1997; Miyake et al., 2000; Pennington, Bennetto, McAleer, & Roberts, 1996; Pennington & Ozonoff, 1996; Roberts & Pennington, 1996). One might speculate that the interrelationships might be unequal for different developmental disorders and might influence the relationship between the primary and secondary deficits found in ADHD and HFA. Future research is needed to address the interrelationships between the five EF domains.

Secondly, although most tasks applied in the current study are developed to measure a specific EF domain, they cannot be considered 'pure' measures of *one* EF domain (Denckla, 1996; Eslinger, 1996). We are well aware that our selection of tasks to cover the five domains of EF, based on Pennington and

Ozonoff (1996), is subject to enduring debate. This measurement problem has been tackled in the current study in three different ways. Firstly, by including some tasks that overlap in their EF demands in order to obtain converging evidence that there are problems with a particular EF domain. Secondly, by the inclusion of non-EF measures to control for the non-EF demands in the EF tasks employed. However, the tasks chosen as non-EF measures might include some EF demands as well. To cover the risk that by covarying for non-EF demands we would throw the baby out with the bathwater, we analysed the data both with and without covarying for non-EF control tasks. In general, results did not alter. Thirdly, by the inclusion of some information processing tasks such as the change task, the ToL, and the SoP, the EF process could be manipulated within subjects. Hence 'purer' measures of certain EF domains could be derived. However, even after controlling for non-EF demands and applying information processing tasks, the clinical groups did not show a double dissociation in their EF-profiles. This makes the results of this study noteworthy, given the high degree of methodological control.

Some might argue that the present findings are due to the heterogeneity of the ADHD group in terms of ADHD subtypes. In the current categorical clinical view, ADHD can be subdivided into three subtypes: ADHD predominantly inattentive subtype (ADHD-I), ADHD predominantly hyperactive/impulsive subtype (ADHD-H), and ADHD combined subtype (ADHD-C; APA, 1994). According to Barkley's theory (1997a, b), only ADHD-C and ADHD-H, but not ADHD-I, will be associated with EF deficits. One may argue, therefore, that the inability to find EF deficits across all EF domains is due to the inclusion of children with ADHD-I in the current sample of ADHD children. Indeed, some studies have shown that a deficit in EF was related to ADHD-C, but was not observed in ADHD-I (e.g., Klorman et al., 1999; Lockwood, Marcotte, & Stern, 2001; Nigg, Blaskey, Huang-Pollock, & Rappley, 2002), but none of these studies showed EF differences between the two subtypes across *all five* EF domains. Furthermore, a number of recent studies with large samples of children have failed to report reliable differences between ADHD-C and ADHD-I subtypes on diverse neuropsychological tasks (Barkley, Grodzinsky, & Du Paul, 1992; Chhabildas, Pennington, & Wilcutt, 2001; Faraone, Biederman, Weber, & Russell, 1998; Murphy, Barkley, & Bush, 2001). Nigg et al. (2002) suggested that although the subtypes differ in the extent and nature of their deficits, both subtypes still belong to the diagnostic entity of ADHD. In the current study (see Results section) we analysed all the data with and without children with ADHD inattentive subtype. The pattern of results hardly altered, although it was even more difficult to distinguish children with HFA from children with ADHD when the inattentive subtype was excluded. The

profile of impairments of children with ADHD when compared to normal controls did not alter at all.

The current findings do not imply that there are no EF profile differences between diagnostic groups as proposed by Pennington and Ozonoff (1996). However, attempting to pinpoint the precise strengths and weaknesses of HFA and ADHD with current EF tasks appears to have reached its limits. Future research should focus on the development of valid EF measures for children employing within subject manipulations (e.g., see Beveridge, Jarrold, & Pettit, 2002). Still, the notion that ADHD is *specifically* associated with EF deficits seems to be an oversimplification of the current state of affairs.

Acknowledgements

We thank Rinske van Dieren, Sonja Deden, Mirjam Jonkers, Margreet van Zelm, Kirsten Lamain, Linda van der Wösten, Sandra de Korte, Jukka Korpi, Rianne de Graaf, Mandy Abbring, Josje Arnoldi, Bregje Witjes, Marije Wertwijn, Nurit Tschochner, Veerle Rollé, Wendy Schelstraete, Marjolijn De Jonghe, and Lieve De Vos for their help in the data collection. Furthermore, we acknowledge Connor Dolan and Dirk Knol for their statistical advice.

Correspondence to

Hilde Geurts, Division of Psychonomics, University of Amsterdam, Roeterstraat 15, 1018 WB Amsterdam, The Netherlands; Email: h.m.geurts@uva.nl

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th edn). Washington, DC: American Psychiatric Association.
- Angold, A., Costello, J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, 40, 57–87.
- Aronowitz, B., Liebowitz, M., Hollander, E., Fazzini, E., Durlach-Misteli, C., Frenkel, M., Mosovich, S., Garfinkel, R., Saoud, J., DelBene, D., Cohen, L., Jaeger, A., & Rubin, L.A. (1994). Neuropsychiatric and neuropsychological findings in conduct disorder and attention-deficit hyperactivity disorder. *Journal of Neuropsychiatry and Clinical Neuroscience*, 6, 245–249.
- Bachorowski, J.A., & Newman, J.P. (1985). Impulsivity in adults: Motor inhibition and time-interval estimation. *Personality and Individual Differences*, 6, 133–136.
- Bachorowski, J.A., & Newman, J.P. (1990). Impulsive motor behavior: Effects of personality and goal salience. *Journal of Personality and Social Psychology*, 58, 512–518.
- Bailey, A., Phillips, W., & Rutter, M. (1996). Autism: Towards an integration of clinical, genetic, neuropsychological, and neurobiological perspectives. *Journal of Child Psychology and Psychiatry*, 37, 89–126.
- Baker, S.C., Rogers, R.D., Owen, A.M., Frith, C.D., Dolan, R.J., Frackowiak, R.S., & Robbins, T.W. (1996). Neural systems engaged by planning: A PET study of the Tower of London task. *Neuropsychologia*, 34, 515–526.
- Band, G.P., & Van Boxtel, G.J. (1999). Inhibitory motor control in stop paradigms: Review and reinterpretation of neural mechanisms. *Acta Psychologica*, 101, 179–211.
- Barkley, R.A. (1997a). Behavioural inhibition, sustained attention, and executive functions: Constructing a unifying theory of AD/HD. *Psychological Bulletin*, 121, 65–94.
- Barkley, R.A. (1997b). *ADHD and the nature of self-control*. New York: The Guilford Press.
- Barkley, R.A., DuPaul, G.J., & Connor, D.F. (1999). Stimulants. In J.S. Werry & C.J. Aman (Eds.), *Practitioner's guide to psychoactive drugs for children and adolescents* (2nd edn, pp. 213–247). New York: Plenum Publishing Corporation.
- Barkley, R.A., Grodzinsky, G., & DuPaul, G.J. (1992). Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. *Journal of Abnormal Child Psychology*, 20, 163–188.
- Benton, A.L., & Hamsher, K.S. (1978). *Multilingual Aphasia Examination* (Manual, revised). Iowa City, IA: University of Iowa.
- Berch, D.B., Krikorian, R., & Huha, E.M. (1998). The Corsi block-tapping task: Methodological and theoretical considerations. *Brain and Cognition*, 38, 317–338.
- Beveridge, M., Jarrold, C., & Pettit, E. (2002). An experimental approach to executive fingerprinting in young children. *Infant and Child Development*, 11, 107–123.
- Bishop, D.V. (1993). Annotation: Autism, executive functions and theory of mind: A neuropsychological perspective. *Journal of Child Psychology and Psychiatry*, 34, 279–293.
- Bishop, D.V. (1998). Development of the Children's Communication Checklist (CCC): A method for assessing qualitative aspects of communicative impairment in children. *Journal of Child Psychology and Psychiatry*, 39, 879–891.
- Bishop, D.V., & Baird, G. (2001). Parent and teacher report of pragmatic aspects of communication: Use of the children's communication checklist in a clinical setting. *Developmental Medicine and Child Neurology*, 43, 809–818.
- Bradshaw, J.L., & Sheppard, D.M. (2000). The neurodevelopmental frontostriatal disorders: Evolutionary adaptiveness and anomalous lateralization. *Brain and Language*, 73, 297–320.
- Cabeza, R., & Nyberg, L. (2000). Imaging cognition II: An empirical review of 275 PET and fMRI studies. *Journal of Cognitive Neuroscience*, 12, 1–47.
- Castellanos, F.X., Lee, P.P., Sharp, W., Jeffries, N.O., Greenstein, D.K., Clasen, L.S., Blumenthal, J.D., James, R.S., Ebens, C.L., Walter, J.M., Zijdenbos, A., Evans, A.C., Gied, J.N., & Rapoport, J.L. (2002). Developmental trajectories of brain volume abnormalities in children and adolescents with Attention-Deficit/Hyperactivity Disorder. *Journal of the American Medical Association*, 288, 1740–1748.

- Castellanos, F.X., & Tannock, R. (2002). Neuroscience of Attention-Deficit/Hyperactivity Disorder: The search for endophenotypes. *Nature Reviews Neuroscience*, 3, 617–628.
- Chhabildas, N., Pennington, B.F., & Willcutt, E.G. (2001). A comparison of the neuropsychological profiles of the DSM-IV subtypes. *Journal of Abnormal Child Psychology*, 29, 520–540.
- Chase-Carmichael, C.A., Ris, M.D., Weber, A.M., & Schefft, B.K. (1999). Neurologic validity of the Wisconsin Card Sorting Test with a pediatric population. *Clinical Neuropsychology*, 13, 405–413.
- Chugani, D.C. (2000). Autism. In M. Ernst & J.M. Rumsey (Eds.), *Functional neuroimaging in child psychiatry* (2nd edn, pp. 171–188). Cambridge: University Press.
- Clark, C., Prior, M., & Kinsella, G.J. (2000). Do executive function deficits differentiate between adolescents with ADHD and oppositional defiant/conduct disorder? A neuropsychological study using the Six Elements Test and Hayling Sentence Completion Test. *Journal of Abnormal Child Psychology*, 28, 403–414.
- Cohen, D.J., & Volkmar, F.R. (1997). *Handbook of autism and pervasive developmental disorders*. New York: John Wiley & Sons, Inc.
- Corsi, P.M. (1972). Human memory and the medial temporal region of the brain. *Dissertation Abstracts International*, 34, 819B.
- Courchesne, E., Townsend, J., Akshoomoff, N.A., Saitoh, O., Yeung-Courchesne, R., Lincoln, A.J., James, H.E., Haas, R.H., Schreibman, L., & Lau, L. (1994). Impairment in shifting attention in autistic and cerebellar patients. *Behavioral Neuroscience*, 108, 848–865.
- Dagher, A., Owen, A.M., Boecker, H., & Brooks, D.J. (1999). Mapping the network for planning: A correlational PET activation study with the Tower of London task. *Brain*, 122, 1973–1987.
- Damasio, A.R., & Maurer, R.G. (1978). A neurological model for childhood autism. *Archives of Neurology*, 35, 777–786.
- De Jong, R., Coles, M.G., & Logan, G.D. (1995). Strategies and mechanisms in nonselective and selective inhibitory motor control. *Journal of Experimental Psychology: Human Perception and Performance*, 21, 498–511.
- Della Salla S., Gray, C., Baddeley, A., Allamano, N., & Wilson, L. (1999). Pattern span: A tool for unwelding visuo-spatial memory. *Neuropsychologia*, 37, 1189–1199.
- Denckla, M.B. (1996). A theory and model of executive function: A neuropsychological perspective. In G.R. Lyon & N.A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 263–277). Baltimore, MD: Paul H. Brookes.
- Eliez, S., & Reiss, A.L. (2000). Annotation: MRI neuroimaging of childhood psychiatric disorders: A selective review. *Journal of Child Psychology and Psychiatry*, 41, 679–694.
- Elwood, R.W. (1997). Episodic and semantic memory components of verbal paired-associate learning. *Assessment*, 4, 73–77.
- Eslinger, P.J. (1996). Conceptualizing, describing, and measuring components of executive function. In G.R. Lyon & N.A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 263–277). Baltimore, MD: Paul H. Brookes.
- Faraone, S.V., & Biederman, J. (1998). Neurobiology of attention-deficit hyperactivity disorder. *Biological Psychiatry*, 44, 951–958.
- Faraone, S.V., Biederman, J., Weber, W., & Russell, R.L. (1998). Psychiatric, neuropsychological, and psychosocial features of DSM-IV subtypes of attention deficit/hyperactivity disorder. *Journal of the American Academy for Child and Adolescent Psychiatry*, 37, 185–193.
- Ferdinand, R.F., Van der Ende, J., & Mesman, J. (1998). *Diagnostic Interview Schedule for Children, DISC-IV*. Nederlandse vertaling [Dutch translation]. Unpublished manuscript. Sophia Kinderziekenhuis, Rotterdam.
- Filipek, P.A., Accardo, P.J., Baranek, G.T., Cook, E.H. Jr., Dawson, G., Gordon, B., Gravel, J.S., Johnson, C.P., Kallen, R.J., Levy, S.E., Minshew, N., Prizant, B.M., Rapin, I., Rogers, S.J., Stone, W.L., Teplin, S., Tuchman, R.F., & Volkmar, F.R. (1999). The screening and diagnosis of autistic spectrum disorders. *The Journal of Autism and Developmental Disorders*, 29, 439–484.
- Frith, C.D., Friston, K.J., Liddle, P.F., & Frackowiak, R.S. (1991). A PET study of word finding. *Neuropsychologia*, 29, 1137–1148.
- Fuster, J.M. (1997). *The prefrontal cortex; anatomy, physiology, and neuropsychology of the frontal lobe* (3rd edn). Philadelphia: Lippincott-Raven.
- Gaillard, W.D., Hertz-Pannier, L., Mott, S.H., Barnett, A.S., LeBihan, D., & Theodore, W.H. (2000). Functional anatomy of cognitive development: fMRI of verbal fluency in children and adults. *Neurology*, 54, 180–185.
- Gerstadt, C.L., Hong, Y.J., & Diamond, A. (1994). The relationship between cognition and action: Performance of children 2–7 years old on a Stroop-like day-night test. *Cognition*, 53, 129–153.
- Ghaziuddin, M. (2002). Asperger syndrome: Associated psychiatric and medical conditions. *Focus on Autism and Other Developmental Disorders*, 17, 138–144.
- Grant, D.A., & Berg, E.A. (1948). A behavioral analysis of degree of reinforcement and ease of shifting to new responses in a Weigel-type card-sorting problem. *Journal of Experimental Psychology*, 38, 404–411.
- Greenhill, L.L. (1998). Childhood attention deficit hyperactivity disorder: Pharmacological treatments. In P.E. Nathan & J. Gorman (Eds.), *A guide to treatments that work* (pp. 42–64). New York: Oxford University Press.
- Groth-Marnat, G. (1997). *Handbook of psychological assessment* (3rd edn). New York: John Wiley and Sons.
- Hale, T.S., Hariri, A.R., & McCracken, J.T. (2000). Attention-deficit/hyperactivity disorder: Perspectives from neuro-imaging. *Mental Retardation and Developmental Disabilities Research Reviews*, 6, 214–219.
- Happé, F., & Frith, U. (1996). The neuropsychology of autism. *Brain*, 119, 1377–1400.
- Harris, M.E. (1990). *Wisconsin Card Sorting Test: Scoring Program*. Odessa, FL: Psychological Assessment Resources, Inc.
- Hartman, C.A., Geurts, H.M., Verté, S., Roeyers, H., Sergeant, J.A., & Bishop, D.V.M. (1998). *De Drie C's*:

- Children's Communication Checklist* [The three C's: Children's Communication Checklist. Dutch translation]. Unpublished manuscript, Vrije Universiteit, Amsterdam.
- Heaton, R.K. (1981). *Wisconsin Card Sorting Test manual*. Odessa, FL: Psychological Assessment Resources.
- Heaton, R.K., Chelune, G.J., Talley, J.L., Kay, G.G., & Curtiss, G. (1993). *Wisconsin Card Sorting Test manual*. Odessa, FL: Psychological Assessment Resources, Inc.
- Hughes, C., Russell, J., & Robbins, T.W. (1994). Evidence for executive dysfunction in autism. *Neuropsychologia*, 32, 477–492.
- Hurt, J., & Naglieri, J.A. (1992). Performance of delinquent and nondelinquent males on planning, attention, simultaneous, and successive cognitive processing tasks. *Journal of Clinical Psychology*, 48, 120–128.
- Jensen, V.K., Larrieu, J.A., & Mack, K.K. (1997). Differential diagnosis between attention-deficit/hyperactivity disorder and pervasive developmental disorder-not otherwise specified. *Clinical Pediatrics*, 36, 555–561.
- Joseph, R.M. (1999). Neuropsychological frameworks for understanding autism. *International Review of Psychiatry*, 11, 309–325.
- Klorman, R., Hazel-Fernandez, L.A., Shaywitz, S.E., Fletcher, J.M., Marchione, K.E., Holahan, J.M., Stuebing, K.K., & Shaywitz, A. (1999). Executive functioning deficits in Attention-Deficit/Hyperactivity Disorder are independent of oppositional defiant or reading disorder. *The Journal of the American Academy of Child and Adolescent Psychiatry*, 38, 1148–1155.
- Krikorian, R., Bartok, J., & Gay, N. (1994). Tower of London procedure: A standard method and developmental data. *Journal of Clinical and Experimental Neuropsychology*, 16, 840–850.
- Le Couteur, A., Rutter, M., Lord, C., Rios, P., Robertson, S., Holdgrafer, M., & McLennan, J. (1989). Autism diagnostic interview: A standardized investigator-based instrument. *Journal of Autism and Developmental Disorders*, 19, 363–387.
- Lezak, M.D. (1995). *Neuropsychological assessment* (3rd edn). New York: Oxford University Press.
- Lockwood, K.A., Marcotte, A.C., & Stern, C. (2001). Differentiation of Attention-Deficit/Hyperactivity Disorder subtypes: Application of a neuropsychological model of attention. *Journal of Clinical and Experimental Neuropsychology*, 23, 317–330.
- Logan, G.D. (1994). On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In D. Dagenbach & T.H. Carr (Eds.), *Inhibitory processes in attention, memory and language* (pp. 189–239). San Diego, CA: Academic Press, Inc.
- Logan, G.D., & Burkell, J. (1986). Dependence and independence in responding to double stimulation: A comparison of stop, change, and dual-task paradigms. *Journal of Experimental Psychology*, 12, 549–563.
- Lombardi, W.J., Andreason, P.J., Sirocco, K.Y., Rio, D.E., Gross, R.E., Umhau, J.C., & Hommer, D.W. (1999). Wisconsin Card Sorting Test performance following head injury: Dorsolateral fronto-striatal circuit activity predicts perseveration. *Journal of Clinical and Experimental Neuropsychology*, 21, 2–16.
- Lord, C. (1997). Diagnostic instruments in autism spectrum disorders. In D.J. Cohen & F.R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd edn, pp. 460–483). New York: Wiley & Sons.
- Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview-Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 24, 659–685.
- Lord, C., Storoschuk, S., Rutter, M., & Pickles, A. (1993). Using the ADI-R to diagnose autism in preschool children. *Mental Health Journal*, 14, 234–252.
- Manly, T., Anderson, V., Nimmo-Smith, I., Turner, A., Watson, P., & Robertson, I.H. (2001). The differential assessment of children's attention: The Test of Everyday Attention for Children (TEA-Ch), normative sample and ADHD performance. *Journal of Child Psychology and Psychiatry*, 42, 1065–1081.
- Milner, B. (1971). Interhemispheric differences in the localization of psychological processes in man. *British Medical Bulletin*, 27, 272–277.
- Minshew, N.J. (1996). Brief report: Brain mechanisms in autism: Functional and structural abnormalities. *Journal of Autism and Developmental Disorders*, 26, 205–209.
- Miyake, A., Friedman, N.P., Emerson, M.J., Witzki, A.H., Howerter, A., & Wager, T.D. (2000). The unity and diversity of executive functions and their contributions to complex 'frontal lobe' tasks: A latent variable analysis. *Cognitive Psychology*, 41, 49–100.
- Miyake, A., Friedman, N.P., Rettinger, D.A., Shah, P., & Hegarty, M. (2001). How are visuo-spatial working memory, executive functioning, and spatial abilities related? A latent-variable analysis. *Journal of Experimental Psychology: General*, 130, 621–640.
- Moffitt, T.E., Lynam, D.R., & Silva, P.A. (1994). Neuropsychological tests predicting persistent male delinquency. *Criminology*, 32, 277–300.
- Murphy, K.R., Barkley, R.A., & Bush, T. (2001). Young adults with attention deficit hyperactivity disorder: Subtype differences in comorbidity, educational, and clinical history. *The Journal of Nervous and Mental Disease*, 190, 147–157.
- Nigg, J.T. (1999). The ADHD response-inhibition deficit as measured by the stop task: Replication with DSM-IV combined type, extension, and qualification. *Journal of Abnormal Child Psychology*, 27, 393–402.
- Nigg, J.T. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin*, 127, 571–598.
- Nigg, J.T., Blaskey, L., Huang-Pollack, C., & Rappey, M.D. (2002). Neuropsychological executive functions and ADHD DSM-IV subtypes. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41, 59–66.
- Nigg, J.T., & Hinshaw, S.P. (1998). Parent personality traits and psychopathology associated with antisocial behaviors in childhood attention-deficit hyperactivity disorder. *Journal of Child Psychology and Psychiatry*, 39, 145–159.
- Nigg, J.T., Hinshaw, S.P., Carte, E.T., & Treuting, J.J. (1998). Neuropsychological correlates of childhood

- attention-deficit/hyperactivity disorder: Explainable by comorbid disruptive behavior or reading problems? *Journal of Abnormal Psychology*, 107, 468–480.
- Nyden, A., Gillberg, C., Hjelmquist, E., & Heiman, M. (1999). Executive function/attention deficits in boys with Asperger syndrome, attention disorder and reading/writing disorder. *Autism*, 3, 213–228.
- Oosterlaan, J., Logan, G.D., & Sergeant, J.A. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD+CD, anxious, and control children: A meta-analysis of studies with the stop task. *Journal of Child Psychology and Psychiatry*, 39, 411–425.
- Oosterlaan, J., Scheres, A., Antrop, I., Roeyers, H., & Sergeant, J.A. (2000). *Vragenlijst voor Gedragsproblemen bij Kinderen (VuGK)*. Nederlandse bewerking van de Disruptive Behavior Disorders Rating Scale [Dutch translation of the Disruptive Behavior Disorders Rating Scale]. Lisse: Swets & Zeitlinger.
- Oosterlaan, J., & Sergeant, J.A. (1998). Response inhibition and response re-engagement in attention-deficit/hyperactivity disorder, disruptive, anxious and normal children. *Behavioral Brain Research*, 94, 33–43.
- Ozonoff, S. (1997). Components of executive function in autism and other disorders. In J. Russell (Ed.), *Autism as an executive disorder* (pp. 179–211). Oxford: Oxford University Press.
- Ozonoff, S., & Jensen, J. (1999). Brief report: Specific executive function profiles in three neurodevelopmental disorders. *Journal of Autism and Developmental Disorders*, 29, 171–177.
- Ozonoff, S., & Strayer, D.L. (1997). Inhibitory function in nonretarded children with autism. *Journal of Autism and Developmental Disorders*, 27, 59–77.
- Ozonoff, S., & Strayer, D.L. (2001). Further evidence for intact working memory in autism. *Journal of Autism and Developmental Disorders*, 31, 257–263.
- Passler, M.A., Isaac, W., & Hynd, G.W. (1985). Neuropsychological development of behavior attributed to frontal lobe functioning in children. *Developmental Neuropsychology*, 1, 349–370.
- Pelham, W., Gnagy, E.M., Greenslade, K.E., & Milich, R. (1992). Teacher ratings of DSM-III-R symptoms for the disruptive behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31, 210–218.
- Pennington, B.F., Bennetto, L., McAleer, O., & Roberts, R.J. (1996). Executive functions and working memory. In G.R. Lyon & N.A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 327–348). Baltimore: Paul H. Brookes Publishing Co.
- Pennington, B.F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 37, 51–87.
- Petrides, M., Alivisatos, B., Evans, A.C., & Meyer, E. (1993). Dissociation of human mid-dorsolateral from posterior dorsolateral frontal cortex in memory processing. *Proceedings of the National Academy of Sciences*, 90, 873–877.
- Petrides, M., & Milner, B. (1982). Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologia*, 20, 249–262.
- Reader, M.J., Harris, E.L., Schuerholz, L.J., & Denckla, M.B. (1994). Attention Deficit Hyperactivity Disorder and executive dysfunction. *Developmental Neuropsychology*, 10, 493–512.
- Riehemann, S., Volz, H.P., Stuetzer, P., Smesny, S., Gaser, C., & Sauer, H. (2001). Hypofrontality in neuroleptic-naïve schizophrenic patients during the Wisconsin Card Sorting Test: A fMRI study. *European Archives of Psychiatry and Clinical Neuroscience*, 251, 66–71.
- Roberts, R.J., & Pennington, B.F. (1996). An interactive framework for examining prefrontal cognitive processes. *Developmental Neuropsychology*, 12, 105–126.
- Robins, T.W. (1997). Integrating the neurobiological and neuropsychological dimensions of autism. In J. Russell (Ed.), *Autism as an executive disorder* (pp. 21–53). New York: Oxford University Press.
- Rosen, W.G. (1980). Verbal fluency in aging and dementia. *Journal of Clinical Neuropsychology*, 2, 135–146.
- Rowe, J.B., Owen, A.M., Johnsrude, I.S., & Passingham, R.E. (2001). Imaging the mental components of a planning task. *Neuropsychologia*, 39, 315–327.
- Rubia, K., Overmeyer, S., Taylor, E., Brammer, M., Williams, S., Simmons, A., & Bullmore, E.T. (1999). Hypofrontality in Attention Deficit Hyperactivity Disorder during higher-order motor control: A study with functional MRI. *American Journal of Psychiatry*, 156, 891–896.
- Russell, J. (1997). *Autism as an executive disorder*. Oxford: Oxford University Press.
- Rutter, M., & Bailey, A. (1999). Thinking and relationships: Mind and brain (some reflections on theory of mind and autism). In S. Baron-Cohen, H. Tager-Flusberg, & D.J. Cohen (Eds.), *Understanding other minds* (pp. 481–504). Oxford: Oxford University Press.
- Schellig, D. (1997). *Block-Tapping-Test*. Lisse: Swets & Zeitlinger.
- Scheres, A., Oosterlaan, J., & Sergeant, J.A. (2001). Response execution and inhibition in children with AD/HD and other disruptive disorders: The role of behavioural activation. *Journal of Child Psychology and Psychiatry*, 42, 347–357.
- Schwab-Stone, M.E., Shaffer, D., Dulcan, M.K., Jensen, P.S., Fisher, P., Bird, H.R., Goodman, S.H., Lahey, B.B., Lichtman, J.H., Canino, G., Rubio-Stipec, M., & Rae, D.S. (1996). Criterion validity of the NIMH Diagnostic Interview Schedule for Children Version 2.3 (DISC-2.3). *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 878–888.
- Séguin, J.R., Boulerice, B., Harden, P.W., Tremblay, R.E., & Pihl, R.O. (1999). Executive functions and physical aggression after controlling for attention deficit hyperactivity disorder, general memory, and IQ. *Journal of Child Psychology and Psychiatry*, 40, 1197–1208.
- Sergeant, J.A., Geurts, H.M., & Oosterlaan, J. (2002). How specific is a deficit of executive functioning for Attention-Deficit/Hyperactivity Disorder? *Behavioural Brain Research*, 130, 3–28.
- Shaffer, D., Fisher, P., Lucas, C.P., Dulcan, M.K., & Schwab-Stone, M.E. (2000). NIMH Diagnostic Interview Schedule for Children version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of*

- the American Academy of Child and Adolescent Psychiatry, 39, 28–38.
- Shallice, T. (1982). Specific impairments of planning. *Philosophical Transactions of the Royal Society of London*, 298, 199–209.
- Shaywitz, B.A., Fletcher, J.M., Pugh, K.R., Klorman, R., & Shaywitz, S.E. (1999). Progress in imaging attention deficit hyperactivity disorder. *Mental Retardation and Developmental Disabilities Research Reviews*, 5, 185–190.
- Sivan, A.B. (1992). *Benton Visual Retention Test* (5th edn). San Antonio, TX: The Psychological Corporation.
- Snijders, J. Th., Tellegen, P.J., & Laros, J.A. (1989). *Snijders-Oomen non-verbal intelligence tests: SON-R 5½–17. Manual and research report*. Groningen: Wolters-Noordhoff.
- Stevens, J. (1996). *Applied multivariate statistics for the social sciences*. New Jersey: Lawrence Erlbaum Associates, Inc.
- Stuss, D.T., & Benson, D.F. (1984). Neuropsychological studies of the frontal lobes. *Psychological Bulletin*, 95, 3–28.
- Tannock, R. (1998). Attention deficit hyperactivity disorder: Advances in cognitive, neurobiological, and genetic research. *Journal of Child Psychology and Psychiatry*, 39, 65–99.
- Tannock, R., Schachar, R.J., Carr, R.P., Chajczyk, D., & Logan, G.D. (1989). Effects of methylphenidate on inhibitory control in hyperactive children. *Journal of Abnormal Child Psychology*, 17, 473–491.
- Tellegen, P., & Laros, J. (1993). The construction and validation of a nonverbal test of intelligence: The revision of the Snijders-Oomen Tests. *European Journal of Psychological Assessment*, 9, 147–157.
- Thapar, A., Harrington, R., & McGuffin, P. (2001). Examining the comorbidity of ADHD-related behaviours and conduct problems using a twin study design. *British Journal of Psychiatry*, 179, 224–229.
- Tranel, D., Anderson, S.W., & Benton, A. (1994). Development of the concept of 'executive function' and its relationship to the frontal lobes. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology* (pp. 125–148). Amsterdam: Elsevier Science.
- Verté, S., Geurts, H.M., Roeyers, H., Oosterlaan, J., & Sergeant, J.A. (2003). *Executive functioning in children with autism and the syndrome of Gilles de la Tourette*. Manuscript in preparation.
- Welsh, M.C., & Pennington, B.F. (1988). Assessing frontal lobe functioning in children: Views from developmental psychology. *Developmental Neuropsychology*, 4, 199–230.
- World Health Organization. (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.