



VU Research Portal

What's in a game: The effect of social motivation on cognitive control in boys with ADHD and ASD

Geurts, H.M.; Luman, M.; van Meel, C.S.

published in

Journal of Child Psychology and Psychiatry
2008

DOI (link to publisher)

[10.1111/j.1469-7610.2008.01916.x](https://doi.org/10.1111/j.1469-7610.2008.01916.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., Luman, M., & van Meel, C. S. (2008). What's in a game: The effect of social motivation on cognitive control in boys with ADHD and ASD. *Journal of Child Psychology and Psychiatry*, 49(8), 848-857. <https://doi.org/10.1111/j.1469-7610.2008.01916.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

What's in a game: the effect of social motivation on interference control in boys with ADHD and autism spectrum disorders

Hilde M. Geurts¹, Mariolein Luman² and Catharina S. van Meel^{3,4}

¹Department of Psychonomics, Universiteit van Amsterdam, The Netherlands; ²Department of Clinical Neuropsychology, Vrije Universiteit, The Netherlands; ³Leiden Institute for Psychological Research, Leiden University, The Netherlands; ⁴Leiden Institute for Brain and Cognition, Leiden University, The Netherlands

Background: Children with attention deficit hyperactivity disorder (ADHD) and with autism spectrum disorders (ASD) are known to have cognitive control deficits. Some studies suggest that such deficits may be reduced when motivation is increased through tangible reinforcers. Whether these deficits can also be modulated by non-tangible reinforcers has hardly been studied. **Methods:** Therefore, the effect of social motivation on the ability to suppress irrelevant information (i.e., interference control) was investigated in 22 ADHD boys, 22 ASD boys, and 33 typically developing (TD) boys. An adapted Eriksen Flanker task was administered under a motivational condition in which the boys were told that they were competing with peers, and under a neutral condition in which standard instructions were given. **Results:** In comparison with TD boys, boys with ADHD were impaired even when no interference was present, while this was not the case for the ASD boys. All groups benefited from the motivation manipulation, i.e., their performance increased when they thought they were competing with peers. Although the boys with ADHD were still slower than TD boys when motivated, they performed as accurately as TD boys. Children with ASD also improved slightly in accuracy and response speed, but this did not reach significance. **Conclusion:** Children with ADHD are able to exert sufficient cognitive control when they are motivated, which is in line with the current models of ADHD. However, motivation seems to have a general effect on performance and is not solely related to cognitive control abilities. In contrast, this effect was not obtained in children with ASD. **Keywords:** Autism, ADHD, motivation, cognitive control.

The ability to monitor ongoing performance in a dynamically changing environment is an important aspect of cognitive control and goal-directed behaviour (e.g., Eslinger, 1996). Deficits in cognitive control have been observed in children with attention deficit hyperactivity disorders (ADHD) as well as children with autism spectrum disorders (ASD; e.g., Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2004; Happé, Booth, Charlton, & Hughes, 2006). Despite the apparent distinctiveness of these disorders, there is a striking co-occurrence of up to 75% (Sturm, Fernell, & Gillberg, 2004). Although a number of studies directly compared ADHD and ASD to disentangle the specific cognitive control deficits these children encounter, none of these studies concentrated on how these children may be able to overcome these deficits. Studying how cognitive control deficits can be modulated might give insight into the mechanisms that underlie successful clinical management and therapies in these developmental disorders. Therefore, the current study focused on the effect of motivation on cognitive control abilities in children with ADHD and ASD.

The dual pathway model of ADHD accounts for both the cognitive and motivational abnormalities seen in children with ADHD (Sonuga-Barke, 2002).

In this model two different neuro-developmental pathways can lead to ADHD: an executive dysfunction (i.e., cognitive control) pathway, linked to deficits in interference control, and a motivational dysfunction pathway, linked to suboptimal reinforcement processes in children with ADHD. It is suggested that these pathways show a non-reciprocal relationship, with the motivational pathway affecting the cognitive control pathway. According to this model, the pathways are linked to the fronto-dorsal striatal and the frontal-ventral striatal circuit (see also Dickstein, Bannon, Castellanos, & Milham, 2006; Nigg & Casey, 2005). Interestingly, exactly these brain circuits also come to the fore in studies concerning brain pathology in ASD (e.g., Bachevalier & Loveland, 2006; Mundy, 2003). Children with ASD show activation abnormalities in the fronto-dorsal circuit while performing cognitive control tasks (Kana, Keller, Minshew, & Just, 2006; Luna et al., 2002; Schmitz et al., 2006). Moreover, the amygdala and the orbito-frontal cortex, which are both implicated in the motivational pathway, have been shown to have a deviant activation pattern in people with ASD (e.g., see for an overview Amaral, Bauman, Mills, & Schumann, 2003; Bachevalier & Loveland, 2006; Schultz, 2005). Furthermore, structural abnormalities and metabolic changes have been reported in people with ASD for the brain

Conflict of interest statement: No conflicts declared.

© 2008 The Authors

Journal compilation © 2008 Association for Child and Adolescent Mental Health.

Published by Blackwell Publishing, 9600 Garsington Road, Oxford OX4 2DQ, UK and 350 Main Street, Malden, MA 02148, USA

regions involved in both circuitries (e.g., Bauman & Kemper, 2005; Carper & Courchesne, 2005; Endo et al., 2007; Murphy et al., 2002). Recently, it has been shown that adults with ASD show significantly greater activation, while performing a sustained attention task, within the anterior cingulate cortex when rewarded than control adults do (Schmitz et al., 2008). However, little is known about whether motivational factors actually affect cognitive control deficits in ASD.

Cognitive control is highly sensitive to motivational manipulations since reward or punishment affects performance on a broad range of tasks. Event-related potential (ERP) studies have demonstrated increased neural activity in networks supporting cognitive control as a function of motivational significance (Gehring, 1993; Pailing & Segalowitz, 2004). Motivation in such studies is usually manipulated using reinforcers such as tangible rewards and response costs. However, monitoring the consequences of one's actions is particularly crucial in the context of social interactions. The mere presence of another person enhances the performance on a wide range of tasks, including cognitive control tasks (e.g., Guerin, 1986; Hajcak, Moser, Yeung, & Simons, 2005; Huguet, Galvaing, Monteil, & Dumas, 1999; Kim, Iwaki, Uno, & Fujita, 2005), an effect called *social facilitation*. However, in both the ADHD and the ASD literature the focus is on tangible reinforcers and the effects of non-tangible reinforcers have been largely neglected. This is surprising as the dual pathway model of ADHD (Sonuga-Barke, 2002) focuses on a deficient motivational pathway in these children and this deficiency is probably not bound to motivational deficits in relation to tangible reinforcers only.

Based on this ADHD model (Sonuga-Barke, 2002), one would expect that all kinds of reinforcers would have a positive effect on the cognitive control abilities of these children. Indeed, numerous studies focusing on the effect of different types of tangible reinforcers have revealed that children with ADHD prefer immediate over delayed reward compared to typically developing (TD) children (Luman, Oosterlaan, & Sergeant, 2005). Moreover, there is some evidence that performance deficits in children with ADHD can be ameliorated when they are motivated by reinforcement contingencies, although the findings are mixed. However, in children with ADHD non-tangible reinforcers also seem to affect their behaviour, e.g., the presence of an observer had a positive effect on their performance in a vigilance task (Power, 1992). In contrast, little is known about reward processing in children with ASD (see also Schmitz et al., 2008). Yet, while children with ADHD had a preference for immediate rewards over delayed rewards, this was not the case for children with ASD (Antrop et al., 2006). Moreover, children with ASD seem to be sensitive to tangible reinforcers, but not to non-tangible reinforcers such as being praised (Garretson, Fein, & Waterhouse, 1990). The effect of

praising has, to our knowledge, never been studied in ADHD. Based on the dual pathway model we expect that children with ADHD will also be sensitive to non-tangible reinforcers. Moreover, we expect that this effect is specific for ADHD as children with ASD might be less sensitive to non-tangible reinforcers than children with ADHD.

To study this we employed an adapted version of the Eriksen Flanker task (Eriksen & Eriksen, 1974) as this is a frequently used cognitive control task to study interference control. With several different versions of this task it has consistently been shown that children with ADHD showed a larger so-called interference effect than TD children (e.g., Scheres et al., 2004; van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007). In contrast, children with ASD seemed to have a smaller interference effect (Henderson et al., 2006). Thus, children with ADHD and ASD might differ from each other in their ability to suppress irrelevant information. In order to determine which information processing stage is influenced most by motivation and whether this differs between the two disorders, both stimulus and response incongruent trials were included in the current study. In this manner, we were able to explore whether the clinical groups differ in interference deficits elicited by irrelevant responses and interference deficits from irrelevant stimuli. Moreover, we could study whether motivation has a specific effect on cognitive control or has a more general enhancing effect on information processing (Guerin, 1986).

Motivation was manipulated by using a competitive condition: letting the children believe that they were playing a game against other children. However, there were no competitors present. This motivational condition was compared to a neutral condition in which the task was administered under standard instructions. It was expected that the non-tangible reinforcer would increase the eagerness to perform well to a larger extent than when no specific reinforcer was given, in line with the dual pathway model (Sonuga-Barke, 2002). Even though none of the earlier studies using non-tangible reinforcers (Antrop et al., 2006; Garretson et al., 1990; Power, 1992) used participation in a (fake) competition as a reinforcer, we hypothesised that motivation (i.e., non-tangible reinforcer) would have a positive effect on cognitive control, and that this effect would be larger for children with ADHD relative to TD children (Power, 1992). In contrast, it was expected that children with ASD would not benefit from this motivator as much as children with ADHD (Antrop et al., 2006; Garretson et al., 1990).

Method

Participants

Seventy-seven boys aged 8 to 13 years participated in this study: 22 boys with a diagnosis of ADHD, 22 boys

with a diagnosis of an ASD, and 33 TD children. Only those boys who had an IQ above 70 as measured by the short version of the Dutch Wechsler Intelligence Scale for Children (WISC-III; Kort et al., 2002) were included. Two WISC-III subtests, Vocabulary and Block Design, were administered to assess intelligence. These subtests both have excellent reliability and correlate highly with Full Scale IQ (FSIQ; Sattler, 2001).

ADHD group. Only children with a prior independent DSM-IV-TR (American Psychiatric Association, 2000) diagnosis of ADHD were included. This diagnosis was checked in several ways. First, to verify the presence of ADHD characteristics all the parents filled out a questionnaire: the Disruptive Behaviour Disorder rating scale (DBD; Oosterlaan, Scheres, Antrop, Roeyers, & Sergeant, 2000; Pelham, Gnagy, Greenslade, & Milich, 1992). All boys had a parent DBD score that exceeded the 90th percentile on at least one of the ADHD scales. Second, most of the teachers of these children filled out the teacher DBD ($n = 21$) and all boys (except one) had a score above the 80th percentile (15 children had a score above the 90th percentile). Third, the ADHD, ODD, and CD sections of the Diagnostic Interview Schedule for Children for DSM-IV, parent version (PDISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) were administered. The PDISC-IV is a broadly used structured diagnostic interview. Based on a stringent diagnostic algorithm, including a check for the presence of cross-situational impairment, ADHD group member-

ship was established. Fourteen children with ADHD combined and 8 with ADHD inattentive subtype were included (6 with comorbid ODD and no comorbid CD).

ASD group. Only children with a prior independent DSM-IV-TR (American Psychiatric Association, 2000) diagnosis of ASD based on extensive diagnostic assessment by a multidisciplinary autism expert team were included. To verify the presence of ASD characteristics all the parents filled out the Children's Social Behaviour Questionnaire (CSBQ; Luteijn, Minderaa, & Jackson, 2002). In all the boys the CSBQ score confirmed the presence of ASD characteristics as they had a score above the 80th percentile (15 boys had a score above the 95th percentile). Two boys with autism, 5 with Asperger syndrome, and 15 boys with a pervasive developmental disorder-not otherwise specified (PDD-NOS) participated in this study. To assess whether these children might also fulfil the criteria for ADHD, the PDISC-IV was administered. Fifteen children indeed fulfilled the ADHD criteria (6 combined, 2 hyperactive/impulsive, and 7 inattentive subtype).

TD group. None of these children had clinical diagnoses of developmental disorders. Boys were excluded when they were diagnosed with disorders such as ADHD or ASD and also had a score on the parent DBD above the 90th percentile.

Table 1 provides group characteristics for each of the three groups. The groups did not differ from each other with respect to age, $F(2,74) = 1.93, p = .15, \eta^2 = .05$

Table 1 Group means and standard deviations for gender, age, IQ, rating scales, and interview scores

Measure	Groups						Posthoc
	TD		ADHD		ASD		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Age	9.4	1.1	9.9	1.5	10.1	1.6	-
FSIQ	103.3	14.0	98.2	13.0	102.7	15.0	-
DBD parent							
Inattention	2.5	2.7	16.8	6.8	15.5	5.8	ADHD, ASD > TD ($p < .001$)
Hyperactivity/Impulsivity	2.8	2.9	16.3	5.3	13.4	7.3	ADHD, ASD > TD ($p < .001$)
ODD	1.9	2.4	8.5	5.1	7.5	5.3	ADHD, ASD > TD ($p < .001$)
CD	.3	.7	2.0	2.3	1.7	2.0	ADHD, ASD > TD ($p < .03$)
DBD teacher							
Inattention	2.4	4.3	11.3	6.8	11.2	6.2	ADHD, ASD > TD ($p < .001$)
Hyperactivity/Impulsivity	2.1	4.2	10.5	7.0	9.3	8.1	ADHD, ASD > TD ($p < .001$)
ODD	.7	1.7	5.6	5.3	5.4	5.5	ADHD, ASD > TD ($p < .001$)
CD	.3	1.6	1.3	1.3	1.6	2.9	-
CSBQ							
Not optimally tuned	2.8	2.8	9.1	3.3	11.8	5.8	ADHD, ASD > TD ($p < .001$)
Reduced social contacts	1.6	2.1	3.3	2.8	9.2	4.2	ASD > ADHD, TD ($p < .001$)
Orientation problems	.6	.9	7.0	3.8	8.0	4.6	ADHD, ASD > TD ($p < .001$)
Problems in understanding	2.2	1.9	5.4	3.0	8.8	2.6	ASD > ADHD > TD ($p < .001$)
Stereotype behavior	.8	1.8	5.8	4.0	6.6	4.0	ADHD, ASD > TD ($p < .001$)
Resistance to change	.6	.9	1.7	1.8	2.5	1.8	ASD > TD ($p < .001$); ADHD > TD ($p < .06$)
Total score	8.8	7.6	32.6	11.1	47.2	15.5	ASD > ADHD > TD ($p < .002$)
PDISC-IV							
ADHD inattentive	-	-	14.7	2.0	12.6	4.7	$p = .07$
ADHD hyperactive	-	-	12.5	4.5	8.5	4.8	ADHD > ASD ($p < .009$)
ODD	-	-	2.3	2.1	2.3	2.1	$p = .75$
CD	-	-	.5	.7	.2	.4	$p = .17$

Note. The number of subjects differs for some the CSBQ due to missing data: TD $n = 25$, ADHD $n = 20$, ASD $n = 22$. If we found a significant difference in the post-hoc testing we mention the highest p -value for these comparisons. ASD = autism spectrum disorders; ADHD = attention deficit hyperactivity disorder; CD = conduct disorder; DBD = Disruptive Behavior Disorder scale; FSIQ = Full Scale IQ; ODD = oppositional defiant disorder; PDISC-IV = Diagnostic Interview Schedule for Children; TD = typically developing controls.

and FSIQ, $F < 1$, $p = .39$, $\eta^2 = .03$. As expected, the parents of the children with ADHD or ASD reported problems when compared to TD children on all scales of the DBD and the CSBQ (see Table 1). Moreover, as stated by the parents, none of the boys of each of the three groups had a neurological, sensory or motor impairment, none had a comorbid learning disability or psychiatric disorders except oppositional defiant disorder (ODD) or conduct disorder (CD), and none of them used medication other than methylphenidate, which was to be discontinued at least 24 hours before testing (Greenhill, 1998).

Task

Spongebob-flanker task. This task is an adaptation of the Eriksen flanker paradigm (Eriksen, & Eriksen, 1974). The participants saw five horizontally arranged cartoon figures from the popular television series 'Spongebob' and had to identify a centrally presented cartoon figure (target) and ignore the distracter figures (flankers) presented on both sides of the target. There were four different cartoon figures: Spongebob, Sandy, Octo, and Patrick. There were three types of trials: congruent (CO), stimulus incongruent (SI), and response incongruent (RI). In a CO trial the flankers were the same as the target. A SI trial consisted of flankers that differed from the target, but both were mapped to the same response. In contrast, in an RI trial the flankers not only differed from the target, but they were also mapped to a different response.

The task started with 1 practice block of 24 CO trials, followed by 1 practice block with 76 mixed trials. If the participant's accuracy was less than 75%, one additional practice block of 76 trials was presented. This was followed by 6 experimental blocks of 88 trials each. Trial types were randomised within each block and within a block 50% of the trials were CO, 25% SI, and 25% RI (Van Veen, Cohen, Botvinick, Stenger, & Carter, 2001). Each trial started with a small fixation cross that was presented for 500 ms in the middle of the computer screen. At each stimulus presentation the flankers together with the central fixation cross appeared for 100 ms. This was followed with a screen with the target surrounded by the flankers. These disappeared from the screen as soon as the participants responded or when 2000 ms has passed (i.e., the response deadline). After this the screen turned black for 900 to 1100 ms due to jitter (see Figure 1).

The participants were instructed to respond with their index finger of one hand if the central figure was Spongebob or Sandy and with the middle finger of their other hand if the central figure was Octo or Patrick. The pictures of the cartoon figures were presented above the response buttons to help the participants to remember the stimulus-response mapping.

There were two motivational conditions in this task: a neutral condition in which standard instructions were given to respond as accurately and quickly as possible and a motivational condition in which the children were told that they were competing with peers. Each condition contained three blocks of trials.

Two dependent measures were calculated. First, the individual mean reaction times (MRTs) of the correct trials were calculated after removal of RTs faster than

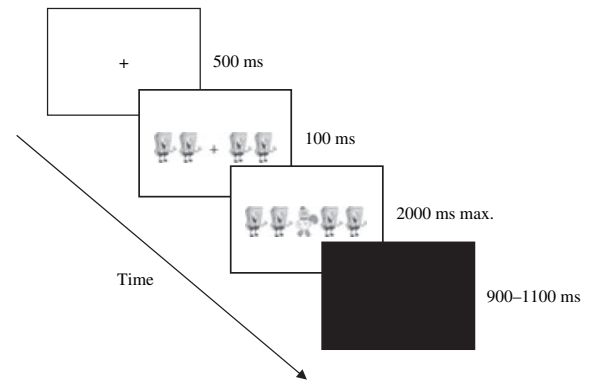


Figure 1 The Spongebob Flanker task is a modified version of the Eriksen Flanker task (Eriksen, & Eriksen, 1974). This figure illustrates the time course of this paradigm

100 ms on a subject-by-subject basis for each level. Second, the percentage of errors was calculated for each participant, which is the number errors divided by the total number of trials.

Questionnaires

The DBD contains four scales composed of the DSM-IV items for ADHD Inattentive subtype, ADHD Hyperactive/Impulsive subtype, Oppositional Defiant Disorder (ODD), and Conduct Disorder (CD). Adequate psychometric properties have been reported (Oosterlaan et al., 2000).

The CSBQ contains six scales: one related to behaviour and emotions not optimally tuned to the social situation, one to reduced social contacts and interests, one to orientation problems in time, place, or activity, one to difficulties in understanding social information, one to stereotypical behaviour, and one to fear of and resistance to changes. The total score of the CBSQ is the sum of the six scales scores and a score above 23 is indicative for the presence of ASD. Adequate psychometric properties have been reported (Hartman, Luteijn, Serra, & Minderaa, 2006; Luteijn et al., 2002).

Procedure

Children with ADHD and ASD were recruited via advertisement on the internet, the national parent association of children with developmental disorders, special educational services, and an institute specialising in the care of children with ASD. TD children were recruited from different community schools in the western part of the Netherlands.

After the parents had filled out an informed consent form and the questionnaires, the participants were tested. The task was part of a multi-centre study on cognition in ADHD and, in addition to the flanker task, two other tasks were administered as well as the two subtests of the WISC-III. Total duration of the study was 2.5 hours including breaks. The WISC-III subtests were administered in the same order for all children, but alternated with the experimental tasks. This is not identical to the standard WISC-III procedure; however, we were only interested in possible group differences in

IQ. The order of the experimental tasks was counter-balanced across participants. Within the Spongebob-flanker task the two motivational conditions were also counterbalanced across the participants. Between the two different motivational conditions of the Spongebob-flanker task there was a 10-minute break in which the children watched a Spongebob cartoon.

The task was practised first to make sure that the participants understood the instructions. The participants were asked to respond as quickly and accurately as they could, but accuracy was additionally stressed during the practice trials. In the neutral condition no additional instructions were given. In the game condition the children were told that they were competing against children from another school. A fake list, on which the names and scores of 10 (non-existent) participants were ranked based on their score, was shown to the children. The children were told that their name would be on a comparable list for their own school. At the end of the test session all children were individually debriefed. In the debriefing we explained that the game was not real. Almost all children said that they believed it was a real match, but that they were still willing to participate in future studies, even though they knew that we had not been telling the truth during this experiment. Only one child with ADHD did not believe that it was a real match, but did respond more accurately and quickly in the game condition.

Eighteen participants with ADHD and nine participants with ASD were on methylphenidate, but discontinued medication at least 24 hours before testing, allowing for a complete wash-out (Greenhill, 1998). All participants received a small gift (worth approximately 1 euro) at the end of the test session. The parents or caregivers were sent reports of the overall findings of the experiment. The study was approved by the local Ethics Committee.

Data analysis

There were no significant effects of the order in which the children received the motivational manipulations (MRT: $F(1,73) < 1$, $p = .364$, $\eta^2 = .01$; % errors F

(1,74) < 1 , $p = .44$, $\eta^2 = .01$), nor did order interact significantly with one of the other factors of interest (trial type and group). Therefore, order is not included as an additional within subject factor in the analyses. The dependent measures were subjected to separate repeated measures ANOVA with group (ADHD, ASD, and TD) as between-subject factor and trial type (CO, SI, and RI) and social motivation (neutral and motivation) as within-subject factors. Note that all analyses were conducted with and without the exclusion of outliers (three outliers: one boy with ADHD and two TD boys). However, since exclusion of outliers did not alter our main findings, we have presented all analyses with the inclusion of all participants.

Results

MRT: Trial type affected the speed of responding, $F(2,71) = 112.74$, $p < .001$, $\eta^2 = .76$. As expected, children were faster at the CO trials compared to the SI ($p < .001$) and RI ($p < .001$) trials. Moreover, children were faster in the SI trials than in the RI trials ($p < .001$). Children also responded significantly faster when they were believed that they were in competition with peers compared to the neutral condition, $F(1,72) = 18.37$, $p < .001$, $\eta^2 = .20$, implying that the motivational manipulation was successful. Moreover, there was a main effect of group, $F(2,72) = 4.21$, $p < .02$, $\eta^2 = .11$. Children with ADHD were overall slower compared to the ASD and TD group, while the other group comparisons did not reach significance.

There was no significant interaction between motivation and trial type, $F < 1$, $p = .39$, $\eta^2 = .03$, but the type of trials had a marginally significant differential effect on group, $F(4,142) = 1.97$, $p = .10$, $\eta^2 = .05$. Figure 2 shows that trial type had a less pronounced effect in the TD group than in the clinical groups. While the SI trials elicited similar MRTs to the RI trials in the TD group, $p = .15$, this was not

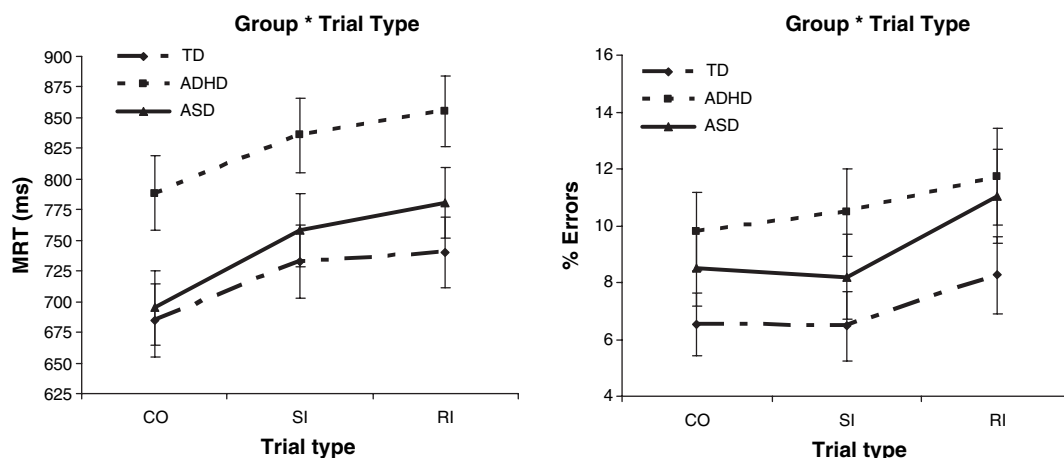


Figure 2 Mean reaction times (MRT in ms) and percentage of errors (% errors) per group for each trial type (left: MRT; right: % errors) Note. ADHD = attention deficit hyperactivity disorder; ASD = autism spectrum disorders; TD = typically developing children; CO = congruent; SI = stimulus incongruent; RI = response incongruent. Points represent the MRT for each specific trial type; vertical lines depict standard errors

the case for the clinical groups. The MRTs of the ADHD and ASD groups increased when the level of interference increased. Post-hoc tests confirmed these observations: In the ADHD group as well as the ASD group the CO trials resulted in the fastest MRTs, followed by the SI trials, while the RI trials resulted in the slowest MRTs (ADHD: CO < SI & RI $p < .001$, SI < RI $p < .04$; ASD: CO < SI & RI $p < .001$, SI < RI $p < .008$). None of the other interactions reached significance (group*motivation: $F < 1$, *ns*, $\eta^2 = .02$; group*motivation*trial type $F < 1$, *ns*, $\eta^2 = .02$). This suggested that the groups are not differentially affected by motivation.

Percentage errors

Trial type also affected accuracy, $F(2,72) = 12.09$, $p < .001$, $\eta^2 = .25$. All children were more accurate at the CO ($p < .001$) and SI trials ($p < .001$) compared to the RI trials. Moreover, the children were as accurate in the SI trials as compared to the CO trials ($p = .84$). Children were also more accurate when they believed that they were in competition with peers compared to the neutral condition, $F(1,73) = 9.59$, $p < .004$, $\eta^2 = .12$. This might imply that motivation had an effect on accuracy. Although visual inspection of Figures 2 and 3 (right panels) suggests that the children in the clinical groups were less accurate than the TD group, this was not significant, $F(2,73) = 1.92$, $p = .15$, $\eta^2 = .04$. Hence, there was no significant main effect of group.

Again, we did not observe a significant interaction between motivation and trial type, $F(2,72) = 1.78$, $p = .18$, $\eta^2 = .05$. Figure 3 (right panel) suggests that motivation had a more pronounced effect in both the ADHD and ASD groups than in the TD group. The interaction between motivation and group was indeed significant, $F(2,73) = 4.67$, $p < .02$, $\eta^2 = .11$. Children with ADHD did show a significant

improvement in accuracy in the motivational condition as compared to the neutral condition ($p < .008$). In contrast, the TD children did not improve as a result of motivation ($p = .79$). Hence, the percentage of errors was significantly higher for the ADHD group compared to the TD group in the neutral condition ($p < .005$), while in the motivational condition the children with ADHD performed as well as the TD group ($p = .17$). No differences were found between the ASD and TD groups in both the neutral ($p = .18$) and the motivational conditions ($p = .40$). Moreover, no significant differences were observed between the ASD and ADHD groups (neutral $p = .41$; motivation $p = .80$). None of the other interactions reached significance (group*trial type: $F < 1$, *ns*, $\eta^2 = .02$; group*motivation*trial type $F(4,144) = 1.15$, $p = .34$, $\eta^2 = .03$).

Discussion

The present findings showed that, when children with ADHD believed they were playing a game against other children, they were capable of implementing more adequate cognitive control in an interference control task, at least when the accuracy of performance was considered. Although children with ADHD still needed more time to accomplish the task, they achieved the same level of accuracy as TD children. To obtain this effect, the children were told only that they were participating in a competition and they were well aware that they could not win anything tangible. We interpret this finding as indicating that the motivation of the children with ADHD increased when they were in (a fake) competition with others. In previous motivational studies on ADHD, money or tokens were often used as reinforcers. These tangible reinforcers also improved the performance of the children with

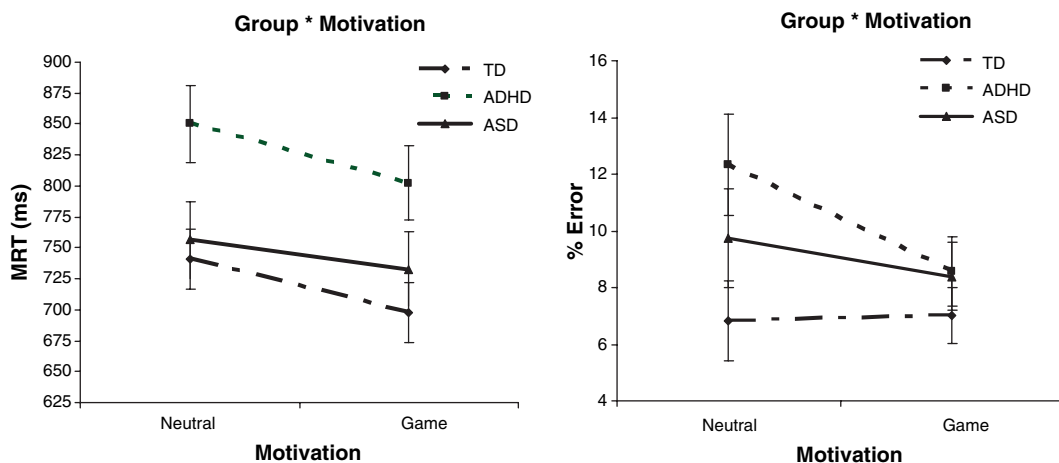


Figure 3 Mean reaction times (MRT in ms) and percentage of errors (% errors) per group for motivational condition (left: MRT; right: % errors) *Note.* ADHD = attention deficit hyperactivity disorder; ASD = autism spectrum disorders; TD = typically developing children; Game = game condition; Neutral = neutral condition. Points represent the MRT for each specific motivational condition; vertical lines depict standard errors

ADHD, but in most studies their performance level was still not similar to that of the TD children (Luman et al., 2005). Future studies should disentangle whether the effect of non-tangible reinforcers may be stronger than the effect of tangible reinforcers. Although ASD children seemed to be more eager to perform well in a competitive situation, they did not profit from the non-tangible reinforcer as much as children with ADHD.

The current findings are in line with recent models of ADHD suggesting that deficits in cognitive control in children with ADHD can be reduced when motivational significance is high (Nigg & Casey, 2005; Sonuga-Barke, 2002). However, the findings also suggest that the motivational effect is not specific for cognitive control but has a more general effect on basic information processing, as the children improved in all trial types. This is in line with the finding that, for example, the social facilitation effect has been observed in a broad range of tasks and is not restricted to cognitive control tasks (Guerin, 1986). Thus, the children with ADHD even improved, when motivated, when hardly any cognitive control was needed to perform the task adequately. Motivation might ameliorate a more general underlying process such as the arousal level of children (see also Sergeant, 2005).

The findings also showed that children with ASD profited from a motivator in the interference task, although the effect was only marginally significant ($p = .08$, $\eta^2 = .15$). A positive impact of external motivation in ASD is in line with earlier studies that found that children with ASD benefit from tangible reinforcers in clinical behavioural modification programmes to reduce their dysfunctional behaviour (e.g., Lovaas, 1987; Matson, Benavidez, Compton, Paclawskyj, & Baglio, 1996). In contrast to our study, the only other study that systematically manipulated different types of reinforcers in ASD found that non-tangible reinforcers hardly affected the performance of the children with ASD (Garretson et al., 1990). The non-tangible reinforcers in the Garretson study consisted of compliments ('Good work!'), which is different from the current motivational manipulation. However, clinicians who treat children with ASD (high functioning) often report that these children are very eager to perform well and are disappointed when they think they have performed more poorly than others. This is only anecdotal evidence and begs for systematic studies, but it does suggest that the children might be sensitive to the idea that they are competing. In addition, owing to the presence of comorbid ADHD in most of the children in the ASD group we cannot separate effects due to ADHD and those due to ASD. Future research is warranted to determine which type of (tangible or non-tangible) reinforcers may be most effective in ASD when no ADHD characteristics are present.

Characteristic for the children with ADHD was that they responded extremely slowly even when they

had to respond to CO trials. This is in line with previous studies which have shown that children with ADHD needed more time to complete a task than TD children (e.g., Lijffijt, Kenemans, Verbaten, & van Engeland, 2005). These longer RTs have been associated with the attentional deficits that are characteristic of these children (e.g., Johnson et al., 2007). The decrease in MRT in the motivational condition suggests that these attention problems may be ameliorated when children are motivated to perform well and, as stated earlier, this motivational effect seems not to be solely related to cognitive control and is thus probably a-specific.

The current findings of cognitive control problems in both disorders are in line with previous studies (e.g., Geurts et al., 2004; Happé et al., 2006). The response speed of both clinical groups was affected more when there was response competition as compared to stimulus competition. In contrast, the response speed of the TD boys was affected when there was interference, but this was independent of the level of interference. However, the accuracy across trial types was similar for each of the three groups. All children were as accurate in the CO condition as in the SI condition but less accurate in the RI condition, which is in line with earlier findings (Van Veen & Carter, 2002). So even though children with ADHD and ASD adjusted their behaviour when there was more interference by slowing down, i.e., by drawing on cognitive control, this is not sufficient to enhance their accuracy.

A caveat of the current study might be that the ASD group was a heterogeneous group whose the clinical diagnosis was not confirmed with the currently widely used gold standard instruments, although the diagnoses were confirmed by the ASD parent questionnaire. An earlier study on interference control found no interference effects in children with ASD (Henderson et al., 2006), which is in contrast to our study. Possibly, in our study, comorbid ADHD in the ASD group increased the deteriorating impact of interference in this group, as interference problems in ADHD have been well established (e.g., Jonkman et al., 1999; Scheres et al., 2004; van Meel et al., 2007). Please note that in the Henderson et al. study (2006) no report was given regarding the presence or absence of ADHD characteristics in the participating ASD children. Another caveat might be that the ADHD groups consisted of children with different ADHD subtypes, while some suggest that these subtypes differ in their cognitive control abilities (e.g., Nigg, Blaskey, Huang-Pollock, & Rappley, 2002), although others cannot distinguish these subtypes on a broad range of cognitive control tasks (e.g., Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2005). The small number of children of the different subtypes excluded the possibility exploring whether these subtypes differed in their response to the applied task. A third caveat might be that children perceived competition with peers

differently, which makes it hard to quantify the 'amount' of reinforcement given or received by an individual child. Even though all children said that they were convinced that they were participating in a competition, this does not suggest that they all experienced the possibility of winning a competition as a reinforcer. However, the same could be argued when using tokens or money, as children might differ in how they perceive the value of these tangible reinforcers. The implicit assumption is that this would not be systematically different between groups. In a former study in which we actually asked children how much they enjoyed winning or losing money, no group differences were observed between ADHD and TD children (van Meel, Oosterlaan, Helsenfeld & Sergeant, 2005). In the current study we did not assess the subjective experience of the children. However, one could argue that owing to the presence of an ADHD or ASD diagnosis, children value money or winning a competition differently (see also Luman et al., 2005), which is exactly what we wished to disentangle.

In both disorders the cognitive control brain circuitries, fronto-dorsal striatal and frontal-ventral striatal, are activated atypically during cognitive control tasks (e.g., Konrad, Neufang, Hanisch, Fink, & Herpertz Dahlmann, 2006; Schmitz et al., 2006, 2008). However, the disorders might be different in how and when these circuits are disrupted. Both groups differ clinically from each other and, for instance, both groups show abnormalities in an ERP component that seems to be related to cognitive control (the error-related negativity [ERN], e.g., Henderson et al., 2006; van Meel et al., 2007), but no direct comparisons between these two groups have been made. Interestingly, this ERN seems to be generated by the anterior cingulate cortex which is thought to be of great importance for cognition-motivation interactions (Bush, Luu, & Posner, 2000; Holroyd & Coles, 2002). As motivation might have a general effect on cognitive processing and, at least in the current study, does not target solely cognitive control processes, other ERP components are probably affected by motivation. Comparisons between the two clinical groups are needed to disentangle how the (dys)functioning of the underlying brain network related to cognitive control and motivation results in similarities in cognitive control deficits and differences in the response to (social) reinforcers.

In sum, this is the first study on the effect of non-tangible reinforcement on cognitive performance in a direct comparison of children with ADHD and children with ASD. Our findings suggest that children with ADHD are able to exert sufficient cognitive control when they are motivated, using a motivational manipulation such as playing a game against other children. It seems that these children are sensitive to non-tangible reinforcers to a larger extent than TD children, although there might have been insufficient room for improvement for the TD

group. However, this is unlikely, as studies which used a similar paradigm obtained similar or even lower error rates in TD children, but still observed improvements (e.g., van Meel et al., 2007). In contrast, children with ASD do apply more cognitive control when motivated, but this is not sufficient to overcome their interference deficits and the observed effect might be due to the presence of children with a comorbid diagnosis of ADHD within this ASD group. This study is in need of replication but, for now, these findings support clinical interventions that do not just focus on tangible reinforcers, but also include non-tangible reinforcers.

Author note

We want to thank: the parents and children who participated in the current study, Bianca, Nina, and Tjerk for testing the children, and the GGZ Buit-enAmstel for making it possible to contact their patients.

Correspondence to

Hilde M. Geurts, Division of Psychonomics, University of Amsterdam, Roetersstraat 15, 1018 WB Amsterdam, The Netherlands; Tel: + 31 20 5256843; Fax: +31 20 6391656; Email: h.m.geurts@uva.nl

References

- Amaral, D.G., Bauman, M.D., & Schumann, C.M. (2003). The amygdala and autism: Implications from non-human primate studies. *Genes, Brain and Behavior*, 2, 295–302.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th edn, tr). Washington, DC: Author.
- Antrop, I., Stock, P., Verté, S., Wiersema, J.R., Baeyens, D., & Roeyers, H. (2006). ADHD and delay aversion: The influence of non-temporal stimulation on choice for delayed rewards. *Journal of Child Psychology and Psychiatry*, 47, 1152–1158.
- Bachevalier, J., & Loveland, K.A. (2006). The orbito-frontal-amygdala circuit and self-regulation of social-emotional behavior in autism. *Neuroscience and Biobehavioral Reviews*, 30, 97–117.
- Bauman, M.L., & Kemper, T.L. (2005). Neuroanatomic observations of the brain in autism: A review and future directions. *International Journal of Developmental Neuroscience*, 23, 183–187.
- Bush, G., Luu, P., & Posner, M.I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, 4, 215–222.
- Carper, R.A., & Courchesne, E. (2005). Localized enlargement of the frontal cortex in early autism. *Biological Psychiatry*, 57, 126–133.
- Dickstein, S.G., Bannon, K., Castellanos, F.X., & Milham, M.P. (2006). The neural correlates of attention deficit hyperactivity disorder: An ALE meta-analysis.

- Journal of Child Psychology and Psychiatry*, 47, 1051–1062.
- Endo, T., Shioiri, T., Kitamura, H., Kimura, T., Endo, S., Masuzawa, N., et al. (2007). Altered chemical metabolites in the amygdala–hippocampus region contribute to autistic symptoms of autism spectrum disorders. *Biological Psychiatry*, 62, 1030–1037.
- Eriksen, B.A., & Eriksen, C.W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception and Psychophysics*, 16, 143–149.
- 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.
- Garretson, H.B., Fein, D., & Waterhouse, L. (1990). Sustained attention in children with autism. *Journal of Autism and Developmental Disorders*, 20, 101–114.
- Gehring, W.J. (1993). The error-related negativity: Evidence for a neural mechanism for error-related processing. *Dissertation Abstracts International*, 53, 5090.
- Geurts, H.M., Verté, S., Oosterlaan, J., Roeyers, H., & Sergeant, J.A. (2004). How specific are executive functioning deficits in attention deficit hyperactivity disorder and autism? *Journal of Child Psychology and Psychiatry*, 45, 836–854.
- Geurts, H.M., Verté, S., Oosterlaan, J., Roeyers, H., & Sergeant, J.A. (2005). ADHD subtypes: Do they differ in their executive functioning profile? *Archives of Clinical Neuropsychology*, 20, 457–477.
- 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.
- Guerin, B. (1986). Mere presence effects in humans: A review. *Journal of Experimental Social Psychology*, 22, 38–77.
- Hajcak, G., Moser, J.S., Yeung, N., & Simons, R.F. (2005). On the ERN and the significance of errors. *Psychophysiology*, 42, 151–160.
- Happé, F., Booth, R., Charlton, R., & Hughes, C. (2006). Executive function deficits in autism spectrum disorders and attention-deficit/hyperactivity disorder: Examining profiles across domains and ages. *Brain and Cognition*, 61, 25–39.
- Hartman, C.A., Luteijn, E., Serra, M., & Minderaa, R.B. (2006). Refinement of the Children's Social Behavior Questionnaire. An instrument that describes the diverse problems seen in milder forms of PDD. *Journal of Autism and Developmental Disorders*, 36, 325–342.
- Henderson, H., Schwartz, C., Mundy, P., Burnette, C., Sutton, S., Zahka, N., & Pradella, A. (2006). Response monitoring, the error-related negativity, and differences in social behavior in autism. *Brain and Cognition*, 61, 96–109.
- Holroyd, C.B., & Coles, M.G.H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, 109, 679–709.
- Huguet, P., Galvaing, M.P., Monteil, J.M., & Dumas, F. (1999). Social presence effects in the Stroop task: Further evidence for an attentional view of social facilitation. *Journal of Personality and Social Psychology*, 77, 1011–1025.
- Johnson, K.A., Robertson, I.H., Kelly, S.P., Silk, T.J., Barry, E., Daibhis, A., Watchorn, A., Keavey, M., Fitzgerald, M., Gallagher, L., Gill, M., & Bellgrove, M.A. (2007). Dissociation in performance of children with ADHD and high-functioning autism on a task of sustained attention. *Neuropsychologia*, 45, 2234–2245.
- Jonkman, L.M., Kemner, C., Verbaten, M.N., van Engeland, H., Kenemans, J.L., Camfferman, G., Buitelaar, J.K., & Koelega, H.S. (1999). Perceptual and response interference in children with attention-deficit hyperactivity disorder, and the effects of methylphenidate. *Psychophysiology*, 36, 419–429.
- Kana, R.K., Keller, T.A., Minshew, N.J., & Just, M.A. (2006). Inhibitory control in high-functioning autism: Decreased activation and underconnectivity in inhibition networks. *Biological Psychiatry*, 62, 198–206.
- Luna, B., Minshew, N.J., Garver, K.E., Lazar, N.A., Thulborn, K.R., Eddy, W.F., & Sweeney, J.A. (2002). Neocortical system abnormalities in autism: An fMRI study of spatial working memory. *Neurology*, 59, 834–840.
- Kim, E.Y., Iwaki, N., Uno, H., & Fujita, T. (2005). Error-related negativity in children: Effect of an observer. *Developmental Neuropsychology*, 28, 871–883.
- Konrad, K., Neufang, S., Hanisch, C., Fink, G.R., & Herpertz Dahlmann, B. (2006). Dysfunctional attentional networks in children with attention deficit/hyperactivity disorder: Evidence from an event-related functional magnetic resonance imaging study. *Biological Psychiatry*, 59, 643–651.
- Kort, W., Compaan, E.L., Bleichrodt, N., Resing, W.C.M., Schittekatte, M., Bosmans, M., Vermeir, G., & Verhaeghe, P. (2002). *WISC-III NL. Handleiding*. London: The Psychological Corporation.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Lijffijt, M.J., Kenemans, L., Verbaten, M.N., & van Engeland, H. (2005). A meta-analytic review of stopping performance in ADHD: Deficient inhibitory motor control? *Journal of Abnormal Psychology*, 114, 216–222.
- Luman, M., Oosterlaan, J., & Sergeant, J.A. (2005). The impact of reinforcement contingencies on AD/HD: A review and theoretical appraisal. *Clinical Psychology Review*, 25, 183–213.
- Luteijn, E.F., Minderaa, R., & Jackson, S. (2002). *Vragenlijst voor Inventarisatie van Sociaal gedrag bij Kinderen (VISK), handleiding*. Lisse, The Netherlands: Swets Testpublishers.
- Matson, J.L., Benavidez, D.A., Compton, L.S., Paclawskyj, T., & Baglio, C. (1996). Behavioral treatment of autistic persons: A review of research from 1980 to the present. *Research in Developmental Disabilities*, 17, 433–465.
- Mundy, P. (2003). The neural basis of social impairments in autism: The role of the dorsal medial-frontal cortex and anterior cingulate system. *Journal of Child Psychology and Psychiatry*, 44, 793–809.
- Murphy, D.G.M., Critchley, H.D., Schmitz, N., McAlonan, G., van Amelsvoort, T., Robertson, D., et al.

- (2002). Asperger syndrome: A proton magnetic resonance spectroscopy study of brain. *Archives of General Psychiatry*, 59, 885–892.
- Nigg, J.T., Blaskey, L., Huang-Pollock, C., & Rappley, 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., & Casey, B.J. (2005). An integrative theory of attention-deficit/hyperactivity disorder based on the cognitive and affective neurosciences. *Development and Psychopathology*, 17, 785–806.
- Oosterlaan, J., Scheres, A., Antrop, I., Roeyers, H., & Sergeant, J.A. (2000). *Vragenlijst voor Gedragsproblemen bij Kinderen (VuGK)*. Nederlandse bewerking van de DBD Rating Scale [Dutch translation of the DBD Rating Scale]. Lisse, The Netherlands: Swets & Zeitlinger.
- Pailing, P.E., & Segalowitz, S.J. (2004). The error-related negativity as a state and trait measure: Motivation, personality, and ERPs in response to errors. *Psychophysiology*, 41, 84–95.
- 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, 210–8.
- Power, T.J. (1992). Contextual factors in vigilance testing of children with ADHD. *Journal of Abnormal Child Psychology*, 20, 579–593.
- Sattler, J.M. (2001). *Assessment of children: Cognitive applications* (4th edn). San Diego, CA: Author.
- Scheres, A., Oosterlaan, J., Geurts, H., Morein Zamir, S., Meiran, N., Schut, H., Vlasveld, L., & Sergeant, J.A. (2004). Executive functioning in boys with ADHD: Primarily an inhibition deficit? *Archives of Clinical Neuropsychology*, 19, 569–594.
- Schmitz, N., Rubia, K., Daly, E., Smith, A., Williams, S., & Murphy, D.G.M. (2006). Neural correlates of executive function in autistic spectrum disorders. *Biological Psychiatry*, 59, 7–16.
- Schmitz, N., Rubia, K., van Amelsvoort, T., Daly, E., Smith, A., & Murphy, D.G. (2008). Neural correlates of reward in autism. *British Journal of Psychiatry*, 192, 19–24.
- Schultz, R.T. (2005). Developmental deficits in social perception in autism: The role of the amygdala and fusiform face area. *International Journal of Developmental Neuroscience*, 23, 125–141.
- Sergeant, J.A. (2005). Modeling attention-deficit/hyperactivity disorder: A critical appraisal of the cognitive-energetic model. *Biological Psychiatry*, 57, 1248–1255.
- 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.
- Sonuga-Barke, E.J. (2002). Psychological heterogeneity in AD/HD – a dual pathway model of behaviour and cognition. *Behavioural Brain Research*, 130, 29–36.
- Sturm, H., Fernell, E., & Gillberg, C. (2004). Autism spectrum disorders in children with normal intellectual levels: Associated impairments and subgroups. *Developmental Medicine and Child Neurology*, 46, 444–447.
- van Meel, C.S., Heslenfeld, D., Oosterlaan, J., & Sergeant, J.A. (2007). Adaptive control in ADHD: The role of error processing. *Psychiatry research*, 151, 211–220.
- van Meel, C.S., Oosterlaan, J., Heslenfeld, D.J., & Sergeant, J. A. (2005). Telling good from bad news: ADHD differentially affects processing of positive and negative feedback during guessing. *Neuropsychologia*, 43, 1946–1954.
- Van Veen, V., & Carter, C.S. (2002). The anterior cingulate as a conflict monitor: fMRI and ERP studies. *Physiology and Behavior*, 77, 477–482.
- Van Veen, V., Cohen, J.D., Botvinick, M.M., Stenger, V.A., & Carter, C.S. (2001). Anterior cingulate cortex, conflict monitoring, and levels of processing. *Neuroimage*, 14, 1302–1308.

Manuscript accepted 6 February 2008