

Oculomotor capture in ADHD

S. Van der Stigchel

Department of Cognitive Psychology, Vrije Universiteit, Amsterdam, The Netherlands

N. N. J. Rommelse, J. B. Deijnen, C. J. A. Geldof, J. Witlox, J. Oosterlaan, and J. A. Sergeant

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

J. Theeuwes

Department of Cognitive Psychology, Vrije Universiteit, Amsterdam, The Netherlands

It is generally thought that deficits in response inhibition form an important area of dysfunction in patients with attention-deficit/hyperactivity disorder (ADHD). However, recent research using visual search paradigms seems to suggest that these inhibitory deficits do not extend towards inhibiting irrelevant distractors. Using an oculomotor capture task, the present study investigated whether boys with ADHD and their nonaffected brothers are impaired in suppressing reflexive eye movements to a task-irrelevant onset distractor. Results showed that boys with ADHD had slower responses than controls, but were as accurate in their eye movements as controls. Nonaffected brothers showed similar problems in the speed of responding as their affected brothers, which might suggest that this deficit relates to a familial risk for developing the disorder. Importantly, all three groups were equally captured by the distractor, which shows that boys with ADHD and their brothers are not more distracted by the distractor than are controls. Saccade latency and the proportion of intrusive saccades were related to continuous dimensions of ADHD symptoms, which suggests that these deficits are not simply present or absent, but rather indicate that the severity of these deficits relate to the severity of ADHD. The finding that boys with ADHD (and their nonaffected brothers) did not have problems inhibiting irrelevant distractors contradicts a general response inhibition deficiency in ADHD, which may be explained by the relatively independency of working memory in this type of response inhibition.

Attention-deficit/hyperactivity disorder (ADHD) is one of the most common psychiatric conditions of childhood, characterized by symptoms of inattention, hyperactivity, and impulsivity. It is estimated to affect around 5% of children, and its core symptoms are believed to persist into

adulthood for some of the patients (Tannock, 1998). Despite the relatively common occurrence of the disorder, the underlying mechanisms remain poorly understood. Response inhibition, among others, has been proposed as forming one of the primary deficits (Barkley, 1997; Barkley,

Correspondence should be addressed to Stefan Van der Stigchel, Department of Cognitive Psychology, Vrije Universiteit, Van Der Boerhorststraat 1, 1081 BT Amsterdam, The Netherlands. (E-mail: s.van.der.stigchel@psy.vu.nl)

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Grodzinsky, & Du Paul, 1992; Mostofsky, Lasker, Cutting, Denckla, & Zee, 2001; Oosterlaan, Logan, & Sergeant, 1998; Quay, 1997). It is hypothesized that the behaviour of patients with ADHD, compared to nonpatients, is more dominated by “stimulus-driven” or involuntary processes instead of “goal-driven” or voluntary processes (Barkley, 1997). Multiple different experimental paradigms and tasks have been used to test this hypothesis, all having in common that certain (inappropriate/irrelevant) behavioural responses need to be suppressed. Overall, children, adolescents, and adults with ADHD perform worse than normal controls on inhibition tasks (Hervey, Epstein, & Curry, 2004; Lijffijt, Kenemans, Verbaten, & Van Engeland, 2005; Nigg, 1999; Oosterlaan et al., 1998), supporting the hypothesis that deficits in response inhibition form an important area of dysfunction in patients with ADHD. However, even though all these paradigms and tasks are designed to measure inhibition processes, they can differ widely with respect to how response inhibition is actually conceptualized and measured (Nigg, 1999). This, in turn, can lead to ambiguous results, such as several inhibition tasks that correlate only modestly with each other (Scheres et al., 2004) or ADHD patients that perform abnormally on certain inhibition tasks but normally on other inhibition tasks (Epstein, Johnson, Varia, & Conners, 2001).

To further investigate response inhibition in children with ADHD, the present study focuses upon a specific method to assess inhibition, namely through the measurement of eye movements. Eye movements, or saccades, allow for an ideal measure of response inhibition, because a failure of inhibition is directly reflected in an erroneous eye movement response. A frequently used eye movement task in studying response inhibition in ADHD patients is the antisaccade task. In the antisaccade task, observers are presented with an abrupt visual onset in the periphery. Subsequently, they have to make an eye movement away from the onset location to its mirror opposite position (an “antisaccade”, Everling & Fischer, 1998; Hallet, 1978; Munoz & Everling, 2004).

These antisaccades typically have longer latencies than saccades towards the visual onset. Further, observers frequently make an erroneous saccade towards the onset location, an error referred to as a “prosaccade”. Successful performance on the antisaccade task requires two processes: response inhibition of an automatically evoked response to the onset location and the subsequent execution of a goal-driven eye movement to the mirror location of the onset. Research with children with ADHD has revealed that these children have higher reaction times on the antisaccade task and also an elevated proportion of erroneous prosaccades compared to controls (Klein, Raschke, & Brandenbusch, 2003; Mostofsky et al., 2001; Munoz, Armstrong, Hampton, & Moore, 2003). Because an erroneous prosaccade can be seen as a failure to inhibit the strong behavioural response to the onset, the higher error rate indicates that children with ADHD are less able to suppress inappropriate responses. These findings lend support for the presence of inhibition deficits in children with ADHD.

In contrast to these studies, results of visual search experiments with children with ADHD seem to be inconsistent with a response inhibition account. Visual search experiments typically involve detecting a target item (a unique shape, colour, or an onset element) amongst distractor items. These tasks can be used to investigate the efficiency of attentional selection required to select the target and to ignore the distractors by measuring the slope of the search function for a target as the number of distractors is varied. An increase in search slope is related to the inability to inhibit searched items. If the number of distractors is increased, children with ADHD are expected to show disproportionately longer search times than controls. Although children with ADHD were overall slower than controls (increase in intercept), there was no difference in the underlying search mechanisms, because the slope of the search function did not show any difference between the groups (Hazell et al., 1999; Karatekin & Asarnow, 1998a; Mason, Humphreys, & Kent, 2003, 2004; Sergeant & Scholten, 1985).

An explicit test of the response inhibition account is a version of a visual search paradigm in which on some trials a distracting object carrying a highly salient but irrelevant feature is presented ("singleton capture"). Visual search performance measured by manual reaction times is highly disrupted by the presence of this distractor, and reaction times to the target are longer in the presence of this distractor (Remington, Johnston, & Yantis, 1992; Theeuwes, 1992, 1994; Yantis & Egeth, 1999). A recent study with children with ADHD showed that the ability to ignore this irrelevant salient distractor is equal in ADHD and controls (Mason et al., 2004). These results are difficult to interpret in terms of differences in inhibitory control, because one would expect that the ADHD group would have larger interference from irrelevant distractors than would the control group if response inhibition is indeed affected in ADHD (Mason et al., 2003, 2004).

In the present study, we used a version of the singleton capture paradigm in which the primary response was an eye movement, the so-called "oculomotor capture" task (Godijn & Theeuwes, 2002; Theeuwes, Kramer, Hahn, & Irwin, 1998). Participants viewed displays containing a number of red circles positioned on an imaginary circle around a central fixation point. After a fixed period, the target circle changed colour to grey. Upon the presentation of the target, on some trials an additional irrelevant red circle was presented with abrupt onset in the display. It is known from previous studies that, on a large proportion of trials in which the additional onset circle was presented, participants do not make an eye movement to the target element, but erroneously execute a saccade to the onset distractor element: The eyes are "captured" by the onset distractor (Godijn & Theeuwes, 2002; Theeuwes et al., 1998). Therefore, this paradigm allows for investigation whether ADHD patients are impaired in suppressing reflexive responses to a task-irrelevant distractor. If true, we should see an elevated proportion of "capture" trials in children with ADHD making an erroneous eye movement to the onset distractor. To our knowledge,

the present study is the first to investigate whether a reflexive task-irrelevant eye movement can be suppressed equally by ADHD and control children.

In addition to studying memory-guided saccades in boys with ADHD, we also studied these saccades in their nonaffected brothers. By including nonaffected siblings, it might be possible to discriminate between deficits that are part of a familial risk for having ADHD and between deficits that are caused by the presence of ADHD itself (Durstun et al., 2004). That is, seen from an etiological perspective, one would expect certain neuro(psycho)logical deficits to give rise to behavioural symptoms of inattention and hyperactivity-impulsivity, which are labelled as "ADHD". However, in reality the reverse is also possible: Being more hyperactive, impulsive, and inattentive may cause an abnormal performance on neuro(psycho)logical tasks. Nonaffected siblings may help in distinguishing between these two alternatives: Nonaffected siblings do not suffer from ADHD, which makes it unlikely that the possible neuro(psycho)logical dysfunctions observed in this group are a result of inattention and hyperactivity-impulsivity. It may be more likely that such deficits are a part of a familial (genetic and environmental) risk for having ADHD and form candidate endophenotypes: underlying vulnerability traits that heighten the risk for developing ADHD (Gottesman & Gould, 2003; Waldman, 2005). Previous research has shown that nonaffected siblings of children with ADHD have comparable problems with inhibition as their affected brothers (Rommelse et al., 2007a; Slaats-Willems, Swaab-Barneveld, de Sonneville, van der Meulen, & Buitelaar, 2003), which may also be observed on the oculomotor capture task.

The hypothesis was tested that (a) boys with ADHD and possibly their nonaffected brothers would be slower and less precise on the oculomotor capture task than would normal controls (main effect of group). Furthermore, it was examined (b) whether boys with ADHD and possibly their nonaffected brothers would be disproportionately slow and imprecise compared to controls when a

distractor was presented compared to when no distractor was presented (interaction group by distractor). Finally, it was tested (c) whether the task variables would relate to continuous dimensions of inattention and hyperactivity/impulsivity symptoms.

Method

Participants

Families with at least one child with the combined subtype of ADHD and at least one additional sibling (regardless of possible ADHD status) were recruited in order to participate in the Amsterdam part of the International Multicenter ADHD Genes study (IMAGE). The IMAGE project is an international collaborative study that aims to identify genes that increase the risk for ADHD using quantitative trait loci (QTL) linkage and association strategies (Brookes et al., 2006). Additional control families were recruited from primary and high schools from the same geographical regions as the participating ADHD families. Controls and their first-degree relatives had no formal or suspected ADHD diagnosis.

For the current study, brothers between 7 and 14 years old discordant for ADHD were selected from the Amsterdam IMAGE sample and were asked to take part in the eye movement study. Also, control boys between 7 and 14 years old that had previously participated in the IMAGE study were asked to take part. A total of 22 boys with the combined subtype of ADHD, 22 of their nonaffected brothers, and 20 control boys participated. All boys were of European Caucasian descent and were excluded if they had an IQ < 70, a diagnosis of autism, epilepsy, general learning difficulties, brain disorders, or known genetic disorders, such as Down syndrome or fragile X syndrome.

Both the boy already clinically diagnosed with ADHD as well as his nonaffected brother were similarly screened using the standard procedures of the IMAGE project described elsewhere (Brookes et al., 2006; Rommelse, Oosterlaan, Buitelaar, Faraone, & Sergeant, 2007b). Briefly, screening questionnaires (parent and teacher

Conners' long version rating scales, Conners, 1996, and parent and teacher strengths and difficulties questionnaires, SDQ, Goodman, 1997) were used to identify children with ADHD symptoms. T-scores ≥ 63 on the Conners' ADHD subscales (L, M, and N) and scores >90th percentile on the SDQ-hyperactivity scale were considered as clinical. For the child with ADHD, a semistructured, standardized, investigator-based interview was administered: the Parental Account of Children's Symptoms ("PACS", Taylor, Sandberg, Thorley, & Giles, 1991; Taylor, Schachar, Thorley, & Wieselberg, 1986). The PACS covers DSM-IV (*Diagnostic and Statistical manual of mental disorders—Fourth Edition*, American Psychiatric Association, 1994) symptoms of ADHD, conduct disorder, oppositional defiant disorder, anxiety, mood, and other internalizing disorders. The section on autistic behaviour traits was administered, if a clinical score (raw score ≥ 15) was obtained on the Social Communication Questionnaire (Berument, Rutter, Lord, Pickles, & Bailey, 1999). For details of the standardized algorithm that was applied to derive each of the 18 DSM-IV ADHD symptoms, readers are referred to Rommelse et al. (2007a). The Conners' long version for both parents and teachers was completed for control children. Control children had to obtain nonclinical scores on both the parent and teacher version (Conners' N-scale: T-score ≤ 62). Table 1 provides the characteristics of the three groups.

Apparatus

A Pentium IV computer with a processor speed of 2.3 GHz controlled the timing of the events and recorded response times. Displays were presented on an Iiyama 21" SVGA monitor with a resolution of 1,024 \times 768 pixels and an 85-Hz refresh rate. A second computer controlled the registration of eye movements' data online. Eye movements were registered by means of a video-based eye tracker (SR Research Ltd, Canada). The Eyelink II system has a 500-Hz temporal resolution and a spatial resolution of 0.01°. The system used an infrared video-based tracking technology to compute the pupil centre

Table 1. Sample characteristics

	Boys with ADHD (<i>n</i> = 22)		Nonaffected brothers (<i>n</i> = 22)		Control boys (<i>n</i> = 20)		<i>F</i> (2, 61)	<i>p</i>	Contrasts (<i>p</i> ≤ .05)
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			
Age in years	10.9	1.9	10.1	2.7	8.9	2.0	3.5	.04	1 > 3
IQ	95.5	11.3	104.9	14.9	108.0	9.6	6.0	<.001	1 < 2&3
<i>N</i> right handed	21		21		16		3.9 ^a	.15	
Conners' parent									
DSM-IV: Inattentive	66.2	7.3	45.7	4.9	44.2	3.6	104.4	<.001	1 > 2&3
DSM-IV: Hyperactive-impulsive	73.0	8.9	48.9	6.8	46.5	4.6	92.2	<.001	1 > 2&3
DSM-IV: Total	70.8	7.4	47.0	5.3	44.7	4.2	132.0	<.001	1 > 2&3
Conners' teacher									
DSM-IV: Inattentive	65.1	5.2	47.4	4.9	43.7	4.2	119.3	<.001	1 > 2&3
DSM-IV: Hyperactive-impulsive	67.5	8.7	47.5	5.6	43.7	2.1	88.9	<.001	1 > 2 > 3
DSM-IV: Total	67.6	6.1	47.3	5.1	43.3	3.2	140.6	<.001	1 > 2 > 3

Note: DSM-IV = Diagnostic and Statistical Manual of Mental Disorders (4th edition); ADHD = attention-deficit/hyperactivity disorder; *M* = mean; *SD* = standard deviation; 1 = boys with ADHD; 2 = nonaffected brothers; 3 = control boys. ^a χ^2 .

and pupil size of both eyes. An infrared head-mounting tracking system tracked head motion. Both eyes were monitored, but only data from the left eye were analysed. An eye movement was considered a saccade either when the movement velocity exceeded $35^\circ/\text{s}$ or when the movement acceleration exceeded $9,500^\circ/\text{s}^2$. Although the system compensates for head movements, the participant's head was stabilized using a chin rest. The distance between monitor and chin rest was 65 cm. Participants performed the experiment in a sound-attenuated and dimly lit room.

Stimuli

Figure 1 illustrates the display sequence. Participants viewed displays containing six equally spaced red circles (1.3° in diameter), presented on an imaginary circle with a radius of 9.6° . A star (0.4°) was presented in the centre of the display and was used for fixation. After 600 ms, one of the circles changed to grey, signalling the location to which a saccade had to be made (the target). The target was presented at a clock position of 1, 5, 7, or 11. The other circles did not change colour. On half the trials,

simultaneously with the target colour change, an additional red distractor appeared with an abrupt onset on the imaginary circle at a clock position

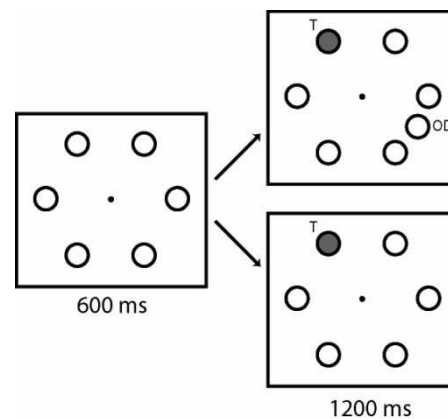


Figure 1. Example of the display sequence. Participants viewed displays containing six equally spaced red circles on an imaginary circle around a central fixation point. After 600 ms, one of the circles changed to grey, signalling the location to which a saccade had to be made (the target "T"). On half the trials, simultaneously with the target colour change, an additional red onset distractor ("OD") appeared with an abrupt onset on the imaginary circle. Participants were instructed to make an eye movement towards the target as quickly as possible.

of 2, 4, 8, or 10. The angular separation between target and onset distractor was always 150° . All objects were removed after 1,200 ms. The colours of the circles—red and grey—were made equiluminant, and the circles appeared on a black background.

Procedure and design

Testing of boys with ADHD, their brothers, and controls took place at the Vrije Universiteit, Amsterdam. Two tasks were administered in a random order: an oculomotor capture task (the current study) and a memory saccade task (described elsewhere). Psychostimulants were discontinued for at least 48 hours before testing took place (Pelham et al., 1999). At the end of the session, a gift worth approximately \$5 was given. The study had medical-ethical approval. Participants first received oral instructions accompanied by sketches of the task. They were instructed to fixate the centre fixation point until the target circle appeared and to then move their eyes to the corresponding location. The experiment consisted of a training session of 16 trials and an experimental session of two blocks of 40 trials. Each session started with a 9-point grid calibration procedure. Participants were required to saccade towards 9 fixation points sequentially appearing at random in a 3×3 grid. In addition, simultaneously fixating the centre fixation point and pressing the space bar recalibrated the system by zeroing the offset of the measuring device at the start of each trial. Each target location was equally probable. The sequence of trials was counterbalanced and randomized for each participant. Trials with and without the additional onset were mixed.

Full-scale IQ was estimated by four subtests of the Wechsler Intelligence Scale for Children—Third Edition (WISC-III) or Wechsler Adult Intelligence Scale—Third Edition (WAIS-III), depending on the child's age: Vocabulary, Similarities, Block Design, and Picture Completion (Wechsler, 2000, 2002). These subtests are known to correlate between .90–.95 with the full-scale IQ (Groth-Marnat, 1997).

Data analysis. Data were missing for four boys with ADHD and one control boy. Two of the files (one ADHD and one control) could not be used due to technical problems. Three of the boys with ADHD had to be excluded, because too few valid trials were present: Due to instable fixation, it was not possible to make a reliable recording of their eye movements.

Dependent measures for speed were (a) saccade latency and (b) total search time. Dependent measures for accuracy were (c) proportion saccades towards the target, (d) proportion saccades towards the distractor, and (e) proportion intrusive saccades (saccades directed neither at the target nor at the distractor). Saccade latency was defined as the interval between target onset and the initiation of a saccadic eye movement to the target. The saccade starting position had to be within 2° from the centre fixation point for the trial to be included. If the latency of the saccade was lower than 80 ms, higher than 800 ms, or further than two and a half standard deviations away from the participant's mean latency, the trial was removed from the analysis. Total search time was defined as the time necessary for the participant to make an eye movement close enough to the target (within 3° of visual angle from the centre of the target). If the total search time for the target or the distractor was more than 1,200 ms, the trial was excluded. Concerning accuracy, if the endpoint of the saccade had an angular deviation of less than 30° from the centre of the target or the distractor, the saccade was classified as initiated to the target or the distractor, respectively. Proportion of intrusive eye movements was defined as saccades directed nor at the target nor at the distractor and may be seen as reflecting general distractibility during the task.

Analyses were conducted with and without IQ as a second covariate next to age, which revealed similar results. Therefore, analyses are presented without IQ as covariate. Alpha was set at .05. A natural log transformation was applied to ensure the normality of the variables. An analysis of variance (ANOVA) was used with group (three groups: boys with ADHD, nonaffected brothers, and control boys) and distractor (present versus

absent) as factors. Also the interaction between group and distractor was implemented in the model, in order to test whether the presentation of the distractor would enhance group differences. Since the proportion of saccades directed at the distractor could only be assessed in trials where a distractor was presented, this measure was compared between the groups without implementing the effect of distractor (present versus absent) and the interaction group by distractor. Age was implemented as a covariate, since it had a strong effect on the dependent measures, $F(1, 57) = 7.38$, $p = .009$ (older children were faster and more accurate than young children) and because the control group was younger than the ADHD group. The variance within families was not significant for all five dependent measures and was, therefore, not implemented as random factor in the model (saccade latency: $Wald Z = 0.83$, $p = .41$; total search time: $Wald Z = 0.60$, $p = .55$; proportion saccades towards the target: $Wald Z = 0.86$, $p = .39$; proportion saccades towards the distractor: $Wald Z = 1.03$, $p = .30$; proportion intrusive saccades: $Wald Z = 0.76$, $p = .45$).

Results: Group differences

Speed

Saccade latency. A significant effect of group was found for the latency of the saccade, $F(2, 111) = 3.35$, $p = .04$. Pairwise comparisons indicated that the boys with ADHD were significantly slower than normal boys, $p = .01$. Nonaffected brothers formed an intermediate group, since they did not differ from their affected brothers, $p = .29$, nor from controls, $p = .08$. There was also a significant effect of distractor, $F(1, 111) = 7.17$, $p = .009$, indicating that the saccade was significantly slower when a distractor was presented than when there was no distractor. No interaction was present between group and distractor, $F(2, 111) = 0.10$, $p = .91$, suggesting that the distractor comparably influenced the latency of the saccade in boys with ADHD, their nonaffected brothers, and normal boys (see Figure 2).

Total search time. There was a significant effect of group on the total search time, $F(2, 111) = 4.29$,

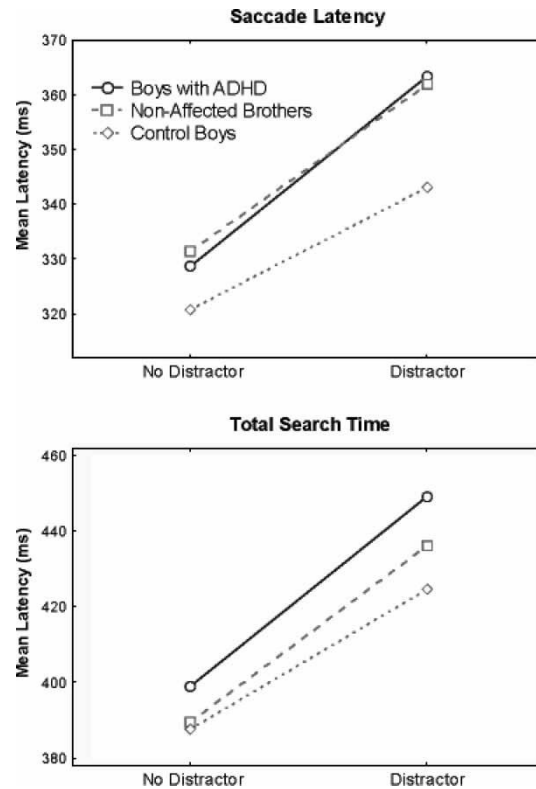


Figure 2. Speed (saccade latency and total search time) of saccades in boys with ADHD, their nonaffected brothers, and control boys on the oculomotor capture task.

$p = .02$. Pairwise comparisons revealed that boys with ADHD were slower than normal boys, $p = .004$. Nonaffected brothers formed, again, an intermediate group since they did not differ from their brothers with ADHD, $p = .06$, nor from controls, $p = .21$. A significant effect of distractor was found, $F(1, 111) = 15.44$, $p < .001$, but the interaction group by distractor was not significant, $F(2, 111) = 0.11$, $p = .90$ (see Figure 2).

Accuracy

Proportion saccades towards the target. Groups did not differ with respect to the proportion of saccades that was directed at the target, $F(2, 111) = 0.27$, $p = .76$. Boys with ADHD and their brothers performed as accurately as controls. As expected, a large effect of distractor was

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present, $F(1, 111) = 91.13$, $p = .001$, but the group by distractor interaction was not significant, $F(2, 111) = 0.20$, $p = .82$. This latter finding indicated that boys with ADHD (and their brothers) were not disproportionately inaccurate when a distractor was presented (see Figure 3).

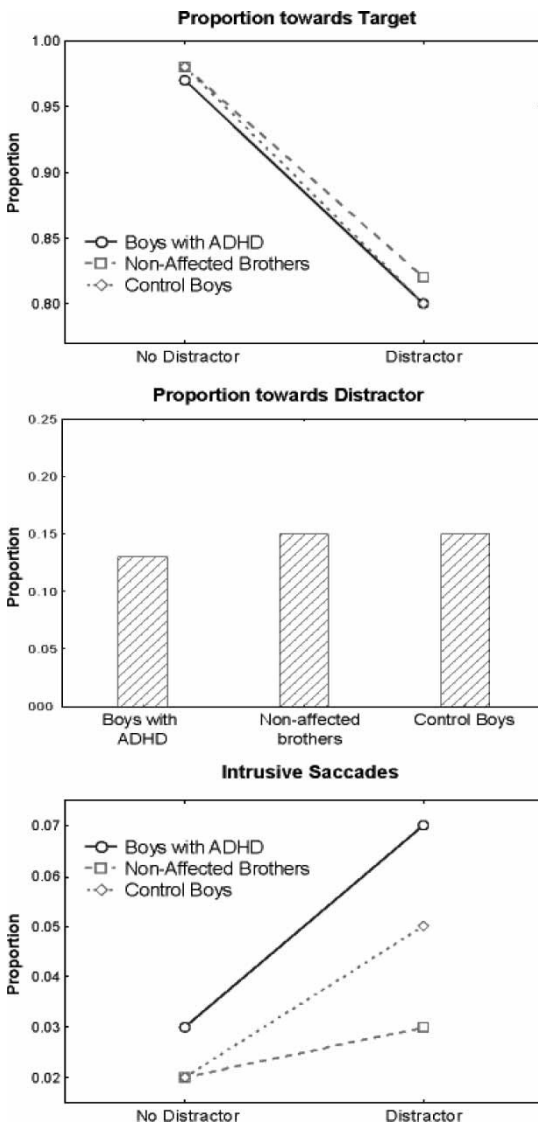


Figure 3. Accuracy (proportion saccades towards the target, towards the distractor and intrusive saccades) in boys with ADHD, their nonaffected brothers, and control boys on the oculomotor capture task.

Proportion saccades towards the distractor. Groups did not differ in the proportion of saccades towards the distractor, $F(2, 55) = 0.20$, $p = .82$. Boys with ADHD and their brothers made as many saccades toward the distractor as normal boys did.

Proportion intrusive saccades. A significant effect of group was found, $F(2, 111) = 3.44$, $p = .04$. Pairwise comparisons indicated that boys with ADHD made more intrusive saccades than their nonaffected brothers, $p = .01$, but not more than controls, $p = .07$. Nonaffected brothers also did not differ from controls, $p = .58$. A main effect of distractor was present, $F(1, 111) = 13.49$, $p < .001$, indicating that more intrusive saccades were made when a distractor was present. The group by distractor interaction was not significant, $F(2, 111) = 1.96$, $p = .16$ (see Figure 3).

Results: Relation task measures with continuous dimensions of ADHD symptomatology

The same mixed model was used as that described above, but the factor group was removed and replaced by one of the four task measures. A continuous measure of inattention (Conners' L-scale) and a continuous measure of hyperactivity/impulsivity (Conners' M-scale), averaged across parent and teacher, were separately used as the dependent measure.

Speed

Saccade latency. The latency of the saccade was significantly related to a continuous dimension of inattentive symptoms, $F(1, 114) = 6.37$, $p = .01$, as well as to a continuous dimension of hyperactivity/impulsivity symptoms, $F(1, 114) = 3.90$, $p = .05$. Slower saccades were associated with higher severity of ADHD symptoms.

Total search time. Total search time was significantly related to inattentive and hyperactive/impulsive symptoms, $F(1, 114) = 9.51$, $p = .003$, and $F(1, 114) = 6.61$, $p = .01$, respectively.

Longer search time was associated with more severe ADHD symptoms.

Accuracy

Proportion saccades towards the target. The proportion of saccades directed at the target was not associated with inattentive, nor with hyperactivity/impulsivity symptoms, $F(1, 114) = 0.001, p = .97$, and $F(1, 114) = 0.02, p = .88$, respectively.

Proportion saccades towards the distractor. The proportion of saccades directed at the distractor was also not associated with inattentive or hyperactive/impulsive symptoms, $F(1, 56) = 0.66, p = .42$, and $F(1, 56) = 1.90, p = .17$.

Proportion intrusive saccades. The proportion of intrusive saccades was not associated with symptoms of inattention, $F(1, 114) = 2.75, p = .10$, but was associated with symptoms of hyperactivity/impulsivity, $F(1, 114) = 5.79, p = .02$. Saccades directed neither at the target nor at the distractor do not seem to be related to inattentive problems, but rather seem to be specifically related

to hyperactivity/impulsivity. Means and standard deviations of the untransformed data are presented in Table 2.

Discussion

The present study investigated whether boys with ADHD and their nonaffected brothers were generally slower and less accurate than normal boys in an oculomotor capture task and whether they were disproportionately affected when an irrelevant onset was presented. By measuring eye movements, the response inhibition account could be directly tested, because a failure of response inhibition is directly reflected in an erroneous eye movement response to the onset distractor.

Results showed that boys with ADHD were slower than controls in the latency and total search time of the saccade to the target, irrespective of whether the onset distractor was present. However, boys with ADHD were not less accurate in making a saccade to the target. The finding that boys with ADHD were slower than controls in search replicates many search studies that have

Table 2. Means of the untransformed task variables

		Boys with ADHD (<i>n</i> = 18)		Nonaffected brothers (<i>n</i> = 22)		Control boys (<i>n</i> = 19)	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>Speed</i>							
Saccade latency ^a	No distractor	328.7	62.5	331.5	74.7	320.8	64.9
	Distractor	363.4	50.5	361.9	88.4	343.1	59.1
Total search time ^a	No distractor	399.0	70.3	389.4	76.1	387.6	66.8
	Distractor	449.2	49.3	436.2	90.9	424.8	64.2
<i>Accuracy</i>							
Proportion saccades to the target	No distractor	.97	.03	.98	.03	.98	.02
	Distractor	.80	.15	.82	.13	.80	.09
Proportion saccades to the distractor	No distractor	-	-	-	-	-	-
	Distractor	.13	.14	.15	.13	.15	.09
Proportion intrusive saccades	No distractor	.03	.03	.02	.03	.02	.02
	Distractor	.07	.05	.03	.03	.05	.04

Note: ADHD = Attention-deficit/hyperactivity disorder; *M* = mean; *SD* = standard deviation.

^aIn ms.

observed similar effects (Hooks, Milich, & Lorch, 1994; Karatekin & Asarnow, 1998a; Mason et al., 2003; Munoz et al., 2003; van der Meere & Sergeant, 1988). This effect is associated with a generalized deficit in the speed of responses (Schachar, Tannock, Marriot, & Logan, 1995).

As expected, all groups were overall slower in terms of saccade latency and search time when the target was accompanied by an irrelevant onset distractor. Also, the amount of capture was elevated when the distractor was present in that more saccades were directed to the distractor instead of to the target. This finding is in line with many studies that have used this type of oculomotor capture task (Godijn & Theeuwes, 2002; Kramer, Hahn, Irwin, & Theeuwes, 2000; Theeuwes et al., 1998; Theeuwes, Kramer, Hahn, Irwin, & Zelinsky, 1999). Importantly, however, the groups did not differ with respect to the influence of the distractor. The lack of interaction with group implies that children with ADHD were not disproportionately impaired in suppressing the distractor relative to the controls. This was underlined by the finding that groups did not differ with respect to the proportion of saccades towards the distractor. This indicates that the irrelevant distractor comparably influenced the latency of the saccade, the direction of the saccade, and the total search time in boys with ADHD and normal controls. This provides evidence for the idea that children with ADHD are not affected when the to-be-inhibited distractor is irrelevant. The results are also in line with visual search experiments in which the slope of the search function was similar for the ADHD and the control group (Mason et al., 2003, 2004; Sergeant & Scholten, 1985).

We also investigated whether the task variables were related to continuous dimensions of ADHD symptoms as measured by inattentive and hyperactivity/impulsivity symptoms. The latency of the saccade and the total search time were found to be related to both of these continuous dimensions of symptoms. Slower saccades and slower visual search were associated with higher severity of ADHD symptoms. These findings indicate that the overall lower speed of responding in children

with ADHD observed here and in various other paradigms (i.e., Hooks et al., 1994; Karatekin & Asarnow, 1998a; Mason et al., 2003; Munoz et al., 2003; Schachar et al., 1995; van der Meere & Sergeant, 1988) does not seem to be an all-or-none process, but rather indicates that the severity of the slowing is related to the severity of ADHD. The proportion of saccades towards the target and distractor was found to be unrelated to the tested dimensions. The proportion of intrusive saccades, however, was associated with symptoms of hyperactivity/impulsivity, though not with symptoms of inattention.

Interestingly, the nonaffected brothers performed abnormally on the two variables that also dissociated their affected brothers from controls: the saccade latency and total search time. Nonaffected brothers formed an intermediate group: They did not differ from their affected brothers nor from control boys for both measures. This suggests that their intermediate slowing in visual search behaviour is related to a familial risk for having ADHD. Nonaffected brothers share certain genetic and environmental risk factors with their affected brother, because they share, on average, half of their genes with their affected brother and grow up in a similar environment (Gottesman & Gould, 2003; Waldman, 2005). Since a slower saccade latency and search time are not only present in children having the disorder but also in children carrying a familial risk for the disorder, this might imply that these deficits are related to genetic and/or shared environmental risk factors that also are related to the disorder. In contrast, the proportion of intrusive saccades did dissociate between children having the disorder and those only at risk: The children having ADHD made more intrusive saccades than their nonaffected brothers, suggesting irrelevant saccades to be caused by the presence of the disorder itself.

The current finding that boys with ADHD are equally influenced by an onset distractor as controls is inconsistent with a general response inhibition account of ADHD (Barkley, 1997). However, as noted before, the to-be-inhibited onset in the current paradigm was irrelevant and did not need

to be attended in order to correctly perform the task. Therefore, errors are purely stimulus driven, since neither the presence nor the location of the onset predicts anything about the target. Interestingly, previous experiments with the oculomotor capture paradigm have shown that the great majority of participants are unaware of the occurrence of onset (Theeuwes et al., 1998). In contrast, the status of the onset is fundamentally different in the antisaccade task in which children with ADHD perform worse than controls (Klein et al., 2003; Mostofsky et al., 2001; Munoz et al., 2003). Here, the onset is task relevant, since participants must direct their attention to the onset and use this object to direct their attention and eyes in the opposite direction. Errors in the antisaccade task are partly goal driven, because there is an explicit instruction not to look at the onset, but to saccade to the opposite direction. This difference between the two types of task might explain the inconsistent reports.

A similar explanation accounted for reported differences in response inhibition between different age groups in healthy individuals (Kramer, Gonzalez de Sather, & Cassavaugh, 2005; Kramer et al., 2000). Using the oculomotor capture and the antisaccade task, these authors showed that older adults do not have more difficulty inhibiting irrelevant onsets but do have more difficulty in suppressing relevant onsets than do younger adults (Kramer et al., 2000). Moreover, for a group of younger and older children it was found that they were not differently captured by the onset in the oculomotor capture task, whereas antisaccade performance improved with age (Kramer et al., 2005). These behavioural differences were explained by distinguishing two qualitatively different types of inhibition, with an automatic/implicit form of inhibition playing a central role in the oculomotor capture task, whereas an intentional/effortful inhibition mostly subserved performance in the antisaccade task (Kramer et al., 2005). On the basis of the current results, it can be concluded that boys with ADHD are unaffected in terms of the automatic/implicit form of inhibition, but have problems with intentional/effortful inhibition.

In line with Kramer and colleagues (2000), we believe that there might be differential involvement of working memory in both types of response inhibition. This memory-based account of saccadic inhibition entails that only intentional inhibition taps on working-memory performance. Evidence for this view comes from studies that observed poorer antisaccade performance when a concurrent working-memory task was executed (Mitchell, Macrea, & Gilchrist, 2002; Roberts, Hager, & Heron, 1994; Stuyven, Van der Goten, Vandierendonck, Claeys, & Crevits, 2000). On the other hand, visual search performance is unaffected when a working-memory task is simultaneously performed (Kane, Poole, Tuholski, & Engle, 2006; Woodman, Vogel, & Luck, 2001) indicating that automatic or implicit inhibition is unrelated to working memory. As it is known that children with ADHD have problems with working memory (Karatekin & Asarnow, 1998b; Kempton et al., 1999; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Oie, Sunde, & Rund, 1999), it might be that the deficits in intentional inhibition relate to working-memory problems. This might also explain the degraded performance of children with ADHD in tasks in which participants have to make a memory-guided saccade. Results show that children with ADHD have elevated anticipatory errors compared to controls (Mostofsky et al., 2001; Ross, Hommer, Breiger, Varley, & Radant, 1994). According to a memory-based account of saccadic inhibition, it is more difficult for children with ADHD to suppress the response to the location in memory because working memory plays an important role in this task as participants must maintain an internal representation of the target location throughout the trial.

Functionally, both forms of inhibition seem to be dissociable based on the involvement of working memory. Anatomically, both forms of inhibition might also be dissociable. Anatomical neuroimaging studies have revealed altered architecture of the prefrontal areas of ADHD participants (Castellanos et al., 1996; Yeo et al., 2003) and less activation compared to controls in frontal and cingulate regions (Aman & Carmichael, 1997;

Rubia et al., 1999; Zametkin et al., 1990). Problems in response inhibition in ADHD have generally been related to these deficits in frontal lobe structures (Castellanos, 2001; Mattes, 1980; Tannock, 1998), because response inhibition seems to depend largely on the frontal areas. Indeed, neurophysiological studies have identified various frontal areas that are active in the antisaccade task (Everling & Munoz, 2000; Funahashi, Chafee, & Goldman-Rakic, 1993). Interestingly, although frontal lesions produce impairments of goal-driven saccades, stimulus-driven saccades seem to be unimpaired (Deng, Goldberg, Segraves, Ungerleider, & Mishkin, 1986). Where the generation of goal-driven saccades largely depends on a frontal pathway, stimulus-driven (reflexive) saccades are thought to be dependent on a parietal eye field (PEF)–superior colliculus (SC) pathway (Pierrot-Deseilligny, Milea, & Muri, 2004). Because eye movements to the onset in the oculomotor capture task are thought to be purely stimulus driven, the dichotomy between goal-driven and stimulus-driven saccades could account for the findings of children with ADHD being equally influenced by the onset as compared to their normally developed peers.

To summarize, we have found that boys with ADHD were slower than control boys in an oculomotor capture task, but were not less accurate. Overall, children were slower and less accurate when a distractor was present, but this effect was comparable across groups, implying that children with ADHD were not impaired inhibiting an irrelevant distractor compared to controls. Slower search was found to be related to continuous dimensions of both inattentive and hyperactive/impulsive ADHD symptoms. Nonaffected brothers formed an intermediate group, showing comparable slowing in visual search to that of their affected brothers, which may suggest that behaviour was related to a familial risk for ADHD. In contrast, nonaffected brothers did not make more intrusive saccades than controls, suggesting intrusive saccades to be caused by the presence of the disorder itself. The finding that boys with ADHD (and their nonaffected brothers) do not seem to have problems inhibiting irrelevant

distractors contradicts a general response inhibition deficiency in ADHD, which may be explained by the relative independency of working memory in this type of response inhibition. We hypothesize that children with ADHD have problems with intentional/effortful inhibition in which working memory is involved, whereas their automatic/implicit inhibition, largely independent of working memory, seems relatively unaffected.

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REFERENCES

- Aman, D. G., & Carmichael, B. D. (1997). High-resolution brain SPECT imaging in ADHD. *Annals of Clinical Psychiatry*, *9*, 81–86.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*, 65–94.
- Barkley, R. A., Grodzinsky, G., & Du Paul, 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.
- Berument, S. K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *British Journal of Psychiatry*, *175*, 444–451.
- Brookes, K., Xu, X., Chen, W., Zhou, K., Neale, B., Lowe, N., et al. (2006). The analysis of 51 genes in DSM-IV combined type attention deficit hyperactivity disorder: Association signals in DRD4, DAT1 and 16 other genes. *Molecular Psychiatry*, *11*, 934.
- Castellanos, F. X. (2001). Neural substrates of attention-deficit hyperactivity disorder. *Advances in Neurology*, *85*, 197–206.
- Castellanos, F. X., Giedd, J. N., Marsh, W. L., Hamburger, S. D., Vaituzis, A. C., Snell, J. W., et al. (1996). Quantitative brain magnetic resonance imaging in attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, *53*, 607–616.

- Conners, K. (1996). *Rating scales in ADHD*: Duke University Medical Center.
- Deng, S.-Y., Goldberg, M. E., Segraves, M. A., Ungerleider, L. G., & Mishkin, M. (1986). The effect of unilateral ablation of the frontal eye field on saccadic performance in the monkey. In E. L. Keller & D. S. Zee (Eds.), *Adaptive processes in visual and oculomotor systems* (pp. 201–208). Oxford, UK: Pergamon Press.
- Durstun, S., Hulshoff Pol, H. E., Schnack, H. G., Buitelaar, J. K., Steenhuis, M. P., Minderaa, R. B., et al. (2004). Magnetic resonance imaging of boys with attention-deficit/hyperactivity disorder and their unaffected siblings. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*, 332–340.
- Epstein, J. N., Johnson, D. E., Varia, I. M., & Conners, C. K. (2001). Neuropsychological assessment of response inhibition in adults with ADHD. *Journal of Clinical and Experimental Neuropsychology*, *23*, 362–371.
- Everling, S., & Fischer, B. (1998). The antisaccade: A review of basic research and clinical studies. *Neuropsychologia*, *36*, 885–899.
- Everling, S., & Munoz, D. P. (2000). Neuronal correlates for preparatory set associated with pro-saccades and anti-saccades in the primate frontal eye field. *Journal of Neuroscience*, *20*, 387–400.
- Funahashi, S., Chafee, M. V., & Goldman-Rakic, P. S. (1993). Prefrontal neuronal activity in rhesus monkeys performing a delayed anti-saccade task. *Nature*, *365*, 753–756.
- Godijn, R., & Theeuwes, J. (2002). Programming of endogenous and exogenous saccades: Evidence for a competitive integration model. *Journal of Experimental Psychology: Human Perception and Performance*, *28*, 1039–1054.
- Goodman, R. (1997). The strengths and difficulties questionnaire: A research note. *Journal of Child Psychology and Psychiatry*, *38*, 581–586.
- Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry*, *160*, 636–645.
- Groth-Marnat, G. (1997). *Handbook of psychological assessment* (3rd ed.). New York: Wiley.
- Hallet, P. E. (1978). Primary and secondary saccades to goals defined by instruction. *Vision Research*, *18*, 1279–1296.
- Hazell, P. L., Carr, V. J., Lewin, T. J., Dewis, S. A. M., Heathcote, D. M., & Brucki, B. M. (1999). Effortful and automatic processing in boys with ADHD and specific learning disorders. *Journal of Child Psychology and Psychiatry*, *40*, 275–286.
- Hervey, A. S., Epstein, J. N., & Curry, J. F. (2004). Neuropsychology of adults with attention-deficit/hyperactivity disorder: A meta-analytic review. *Neuropsychology*, *18*, 485–503.
- Hooks, K., Milich, R., & Lorch, E. P. (1994). Sustained and selective attention in boys with attention deficit hyperactivity disorder. *Journal of Clinical Child Psychology*, *23*, 69–77.
- Kane, M. J., Poole, B. J., Tuholski, S. W., & Engle, R. W. (2006). Working memory capacity and the top-down control of visual search: Exploring the boundaries of “executive attention”. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *32*, 749–777.
- Karatekin, C., & Asarnow, R. F. (1998a). Components of visual search in childhood onset schizophrenia and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*, *26*, 367–380.
- Karatekin, C., & Asarnow, R. F. (1998b). Working memory in childhood-onset schizophrenia and attention deficit/hyperactivity disorder. *Psychiatry Research*, *80*, 165–176.
- Kempton, S., Vance, A., Maruff, P., Luk, E., Costin, J., & Pantelis, C. (1999). Executive function and attention deficit hyperactivity disorder: Stimulant medication and better executive function performance in children. *Psychological Medicine*, *29*, 527–538.
- Klein, C., Raschke, A., & Brandenbusch, A. (2003). Development of pro- and antisaccades in children with attention-deficit hyperactivity disorder (ADHD) and healthy controls. *Psychophysiology*, *40*, 17–28.
- Kramer, A. F., Gonzalez de Sather, J. C. M., & Cassavaugh, N. D. (2005). Development of attentional and oculomotor control. *Developmental Psychology*, *41*, 760–772.
- Kramer, A. F., Hahn, S., Irwin, D. E., & Theeuwes, J. (2000). Age differences in the control of looking behavior: Do you know where your eyes have been? *Psychological Science*, *11*, 210–217.
- Lijffijt, M., Kenemans, J. 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*, *116*, 216–222.
- Martinussen, R., Hayden, J., Hogg-Johnson, S., & Tannock, R. (2005). A meta-analysis of working

- memory impairments in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*, 377–384.
- Mason, D. J., Humphreys, G. W., & Kent, L. S. (2003). Exploring selective attention in ADHD: Visual search through space and time. *Journal of Child Psychology and Psychiatry*, *44*, 1158–1176.
- Mason, D. J., Humphreys, G. W., & Kent, L. S. (2004). Visual search, singleton capture, and the control of attentional set in ADHD. *Cognitive Neuropsychology*, *21*, 661–687.
- Mattes, J. A. (1980). The role of frontal lobe dysfunction in childhood hyperkinesia. *Comprehensive Psychiatry*, *21*, 358–369.
- Mitchell, J. P., Macrea, C. N., & Gilchrist, I. D. (2002). Working memory and the suppression of reflexive saccades. *Journal of Cognitive Neuroscience*, *14*, 95–103.
- Mostofsky, S. H., Lasker, A. G., Cutting, L. E., Denckla, M. B., & Zee, D. S. (2001). Oculomotor abnormalities in attention deficit hyperactivity disorder. A preliminary study. *Neurology*, *57*, 423–430.
- Munoz, D. P., Armstrong, I. T., Hampton, K. A., & Moore, K. D. (2003). Altered control of visual fixation and saccadic eye movements in attention-deficit hyperactivity disorder. *Journal of Neurophysiology*, *90*, 503–514.
- Munoz, D. P., & Everling, S. (2004). Look away: The anti-saccade task and the voluntary control of eye movement. *Nature Reviews Neuroscience*, *5*, 218–228.
- 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.
- Oie, M., Sunde, K., & Rund, B. R. (1999). Contrasts in memory functions between adolescents with schizophrenia or ADHD. *Neuropsychologia*, *37*, 1351–1358.
- 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. *The Journal of Child Psychology and Psychiatry and Allied Disciplines*, *39*, 411–425.
- Pelham, W. E., Aronoff, H. R., Midlam, J. K., Shapiro, C. J., Gnagy, E. M., Chronis, A. M., et al. (1999). A comparison of Ritalin and Adderall: Efficacy and time-course in children with attention-deficit/hyperactivity disorder. *Pediatrics*, *103*, e43.
- Pierrot-Deseilligny, C., Milea, D., & Muri, R. (2004). Eye movement control by the cerebral cortex. *Current Opinion in Neurology*, *17*, 17–25.
- Quay, H. C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*, *25*, 7–13.
- Remington, R. W., Johnston, J. C., & Yantis, S. (1992). Involuntary attentional capture by abrupt onsets. *Perception & Psychophysics*, *51*, 279–290.
- Roberts, R. J., Hager, L. D., & Heron, C. (1994). Prefrontal cognitive processes: Working memory and inhibition in the antisaccade task. *Journal of Experimental Psychology: General*, *123*, 374–393.
- Rommelse, N., Altink, M. A., Oosterlaan, J., Buschgens, C. J. M., Buitelaar, J., & Sergeant, J. (2007a). *Putting the executive and intelligence search lights on the ADHD endophenotype haystack: Efforts from a large neuropsychological study of ADHD and control families*. Manuscript submitted for publication.
- Rommelse, N., Oosterlaan, J., Buitelaar, J., Faraone, S. V., & Sergeant, J. (2007b). Time reproduction in children with ADHD and their non-affected siblings. *Journal of the American Academy of Child and Adolescent Psychiatry*, *46*, 582–590.
- Ross, R. G., Hommer, D., Breiger, D., Varley, C., & Radant, A. (1994). Eye movement task related to frontal lobe functioning in children with attention deficit disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *33*, 869–874.
- Rubia, K., Overmeyer, S., Taylor, E., Brammer, M., Williams, S. C. R., Simmons, A., et al. (1999). Hypofrontality in attention deficit hyperactivity disorder during higher-order motor control: A study with functional MRI. *American Journal of Psychiatry*, *156*, 891–896.
- Schachar, R., Tannock, R., Marriot, M., & Logan, G. D. (1995). Deficient inhibitory control in ADHD. *Journal of Abnormal Child Psychology*, *23*, 411–437.
- Scheres, A., Oosterlaan, J., Geurts, H., Morein-Zamir, S., Meiran, N., Schut, H., et al. (2004). Executive functioning in boys with ADHD: Primarily an inhibition deficit? *Archives of Clinical Neuropsychology*, *19*, 569–594.
- Sergeant, J., & Scholten, C. A. (1985). On data limitations in hyperactivity. *Journal of Child Psychology and Psychiatry*, *26*, 111–124.

- Slaats-Willemse, D., Swaab-Barneveld, H., de Sonneville, L., van der Meulen, E., & Buitelaar, J. (2003). Deficient response inhibition as a cognitive endophenotype of ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*, 1242–1248.
- Stuyven, E., Van der Goten, K., Vandierendonck, A., Claeys, K., & Crevits, L. (2000). The effect of cognitive load on saccadic eye movements. *Acta Psychologica*, *104*, 69–85.
- Tannock, R. (1998). Attention-deficit hyperactivity disorder: Advances in cognitive, neurobiological, and genetic researches. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *39*, 65–99.
- Taylor, E., Sandberg, S., Thorley, G., & Giles, S. (1991). *The epidemiology of childhood hyperactivity*. Oxford, UK: Oxford University Press.
- Taylor, E., Schachar, R., Thorley, G., & Wieselberg, M. (1986). Conduct disorder and hyperactivity: I. Separation of hyperactivity and antisocial conduct in British child psychiatric patients. *British Journal of Psychiatry*, *149*, 760–767.
- Theeuwes, J. (1992). Perceptual selectivity for color and form. *Perception & Psychophysics*, *51*, 599–606.
- Theeuwes, J. (1994). Stimulus-driven capture and attentional set: Selective search for color and visual abrupt onsets. *Journal of Experimental Psychology: Human Perception and Performance*, *20*, 799–806.
- Theeuwes, J., Kramer, A. F., Hahn, S., & Irwin, D. E. (1998). Our eyes do not always go where we want them to go: Capture of eyes by new objects. *Psychological Science*, *9*, 379–385.
- Theeuwes, J., Kramer, A. F., Hahn, S., Irwin, D. E., & Zelinsky, G. J. (1999). Influence of attentional capture on oculomotor control. *Journal of Experimental Psychology: Human Perception and Performance*, *25*, 1595–1608.
- van der Meere, J., & Sergeant, J. (1988). Focused attention in pervasively hyperactive children. *Journal of Abnormal Child Psychology*, *16*, 627–639.
- Waldman, I. D. (2005). Statistical approaches to complex phenotypes: Evaluating neuropsychological endophenotypes for attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *57*, 1347–1356.
- Wechsler, D. (2000). *WAIS-III Nederlandstalige bewerking. Technische handleiding*. London: The Psychological Corporation.
- Wechsler, D. (2002). *WISC-III Handleiding*. London: The Psychological Corporation.
- Woodman, G. F., Vogel, E. K., & Luck, S. J. (2001). Visual search remains efficient when visual working memory is full. *Psychological Science*, *12*, 219–224.
- Yantis, S., & Egeth, H. E. (1999). On the distinction between visual salience and stimulus-driven attentional capture. *Journal of Experimental Psychology: Human Perception and Performance*, *25*, 661–676.
- Yeo, R. A., Hill, D. E., Campbell, R. A., Vigil, J., Petropoulos, H., Hart, B., et al. (2003). Proton magnetic resonance spectroscopy investigation of the right frontal lobe in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*, 303–310.
- Zametkin, A. J., Nordahl, T. E., Gross, M., King, A. C., Semple, W., Rumsey, J., et al. (1990). Cerebral glucose metabolism in adults with hyperactivity of childhood onset. *New England Journal of Medicine*, *323*, 1361–1366.