University of Utrecht

Neuropsychology

Temporal Dynamics of Interference Inhibition in Adults with Autism Spectrum Disorder

Anne Janas

Studentnr.: 3729192

19-06-2012

Prof. Dr. Hilde Geurts

Dr. Leo Kannerhuis

Dr. Barbara Montagne

University of Utrecht

Abstract

In the present study activation and inhibition functioning in autism spectrum disorder (ASD) was investigated using the Simon task, with the dual process model as theoretical background. Temporal reaction time and accuracy changes, caused by the interference effect of the task, were examined with a delta plot analysis. It was expected that ASD participants would show a slower inhibition build up and overall deficits in inhibition compared with controls. Additionally, the hypothesis was tested that autism features, quantified by the Autism Quotient score, and Attention Deficit Hyperactivity Disorder (ADHD) features, quantified by the ADHD DSM-IV checklist scores, together were most predictive for the performance. Participants were male adults with ASD between 18 and 35 years (n=16) and healthy controls (n=16) matched on age and intelligence quotient. In line with the dual process model reaction time and accuracy changed over time. But contrary to the hypothesis, the interference effect decreased for both groups equally with increasing response time. Furthermore, only hyperactivity-impulsivity features and not ASD features were predictive for the inhibition performance. The negative predictive value of hyperactivity-impulsivity changed with increasing response time. This highlights the gain of a distributional analysis and also the importance of further investigation of the influence of comorbid ADHD symptoms on inhibition in ASD. In the current study ASD adults seemed to show the same inhibition and activation temporal dynamics on this interference task as healthy controls. More research with a purer and bigger sample and a faster event rate is needed to confirm this statement.

Self-regulation is necessary to successfully behave and communicate in social-emotional situations as inappropriate behavior has to be inhibited and appropriate reactions have to be activated. People with autism spectrum disorder (ASD)[1] seem to be impaired in these social-emotional self-regulation processes (Bachelavier & Loveland, 2006). Moreover, on non-social tasks ASD samples show more profound inhibitory problems than healthy controls (Corbett, Constatine, Hendren, Rocke, & Ozonoff, 2009; Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2004; Johnston, Madden, Bramham, & Russel, 2011; Verté, Geurts, Roeryers, Oosterlaan, & Sergeant, 2006). However, evidence has not been conclusive as other research groups reported comparable inhibition performances of ASD participants and controls (Eskes, Bryson, & McCormick, 1990; Goldberg et al., 2005; Happé, et al., 2006; Ozonoff & Jenssen, 1999). Given that within the same studies ASD samples performed deviant on some but not other inhibition tasks, a differentiation between impaired and spared aspects of inhibition in ASD has been suggested (Christ, Holt, White, & Green, 2007; Christ, Kester, Bodner, & Miles, 2011; Friedman & Miyake, 2004; Hill, 2004). Interference inhibition is one of the inhibition functions in ASD that is frequently labeled as impaired (Christ et al., 2007; Christ et al., 2011) and it was the subject of the current study. The aim of the present study was to explore inhibition processes of adults with ASD during an interference task in more detail and to rule out possible reasons for the so far inconclusive research outcomes.

One reason for the incompatible evidence might be the assumption of generalization of inhibition performances from ASD children to ASD adults. Although research has shown that inhibition functioning in persons with ASD can change with age (Happé, Booth, Charlton, & Hughes, 2006; Ozonoff & Jensen, 1999; for an exception see Ozonoff et al., 2004), only a very limited amount of studies has investigated inhibition in adults with ASD (Johnston, Madden, Bramham, & Russel, 2011; Raymaekers, van der Meere, & Roeyers, 2004). Therefore, the present study focused on providing empirical data of inhibition functioning in an ASD adult sample.

In addition, comorbid features of other disorders associated with inhibition problems as the attention deficit hyperactivity disorder (ADHD; Bush et al., 2008; Scheres et al., 2004) might have distorted earlier research outcomes. Although ASD and ADHD are classified as two independent syndromes according to the Diagnostic and Statistical Manual of Mental Disorders 4th edition (DSM-IV, American Psychiatric Association, 2000), 30-80% of children diagnosed with ASD have sufficient comorbid ADHD symptoms to meet the criteria of ADHD, with a possible persistency into adulthood (Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010). Children with ASD plus comorbid ADHD have been found to be more severely impaired in inhibition functioning on the Go/No-Go task than children

without comorbid symptoms (Sinzig, Morsch, Bruning, Schmidt, & Lehmkuhl, 2008). Consequently, deficiencies of participants with ASD on any kind of inhibition task might not only be attributed to ASD but also to possible comorbid ADHD symptoms. Unfortunately, most of the noted studies have not controlled for this covariate, but in the current study the possible influence of comorbid ADHD symptoms on interference inhibition in both the ASD and the control group was taken into count.

Furthermore, summing up individual measures and analyzing imprecise overall outcome measures might partly account for the mixed research outcomes. A more detailed measurement has to be used to deliver more persuasive evidence of inhibition functioning in ASD. For that reason, the current study zoomed in on the temporal dynamics of interference inhibition. For the first time in ASD research, a detailed inhibition investigation was carried out with a delta plot analysis, following Ridderinkhof, Scheres, Oosterlaan and Sergeant's (2005) footsteps who examined the temporal dynamics of inhibition in ADHD. The theoretical background of the delta plot analysis was the dual process model of response activation and selective response inhibition (De Jong, Liang, & Lauber, 1994) which is now pointed out before describing the current study in more detail.

Dual process model

So far the dual process model has successfully explained the interference effects of Flanker (Eriksen & Eriksen, 1974) and Simon (Simon, 1970) tasks (De Jong et al., 1994; Ridderinkhof, 2002; Ridderinkhof et al., 2005; Ridderinkhof, van den Wildenberg, Wijnen, & Burle, 2004; Speckman, Rouder, Morey, & Pratte, 2008) and is now introduced as the theoretical background for the current study. The basic concept of the model is the following: The presentation of a stimulus during a task activates two independent process routes. First, there is a deliberate response route which is based on the instructions of how to respond to the stimulus. Additionally, the direct activation route is automatically triggered by the features of the stimulus and is independent of any stimulus-response mapping instructions. Both activation routes converge at the level of the final response activation. The response of the dominantly activated pathway is executed (Ridderinkhof et al., 2004).

In an interference task, there are two different stimulus-response situations: congruent trials and incongruent trials. During a congruent trial the stimulus features trigger both the direct response route and the deliberate response route in a way that both routes activate the same response. This results in a fast and accurate response. During an incongruent trial the irrelevant features of the stimulus trigger the direct response route while the participant has to deliberately activate the second response route based on the stimulus-response mapping instructions. As a consequence two different response

activations compete for execution which results in a longer reaction time and more errors. In this competing situation the direct response activation can selectively be inhibited (Burle et al., 2002; Eimer & Schlaghecken, 1998), but this inhibition process needs some time to build up as the activation-suppression hypothesis states (Ridderinkhof, 2002). Consequentially, fast responses are most influenced by the direct activation of the response routes and slower responses are more affected by selective inhibition. Figure 1 shows the visualization of the dual process model for an interference task. The dual process model forms the theoretical background for the delta plot analysis which is outlined next.

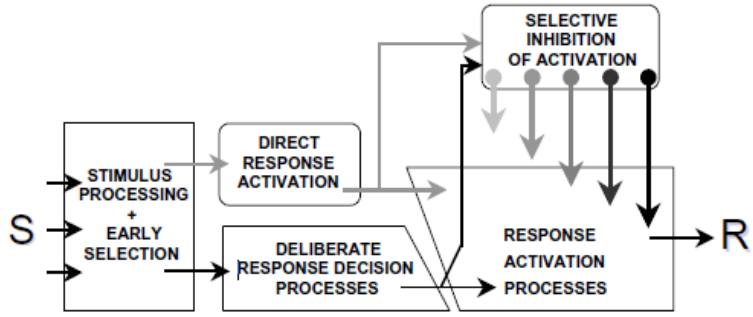

Figure 1: Visualization of the dual process model for an interference task. Picture taken from "Response Inhibition in Conflict Tasks is revealed in Delta Plots," by K.R. Ridderinkhof, W.P.M. van den Wildenberg, J. Wijnen and B. Burle, 2004, in M. Posner (Ed.), *Cognitive Neuroscience of Attention (p. 370).* NY: Guilford Press.

Delta plots analysis

To date, delta plot analyses have not been used to investigate inhibition in ASD. But as the dual process model and the activation-suppression hypothesis (Ridderinkhof, 2002) state, activation and inhibition change over time. It is, therefore, necessary to examine the temporal development of activation and inhibition in both groups instead of comparing overall measurements as researchers have done so far. By applying a distributional analysis it is possible to analyze the activation and inhibition of responses in relation to the reaction time at percentile levels, examining in detail where possible impairments of interference inhibition become clear (Speckman et al., 2008). For delta plots usually the difference in reaction time and the difference in accuracy between incongruent and congruent trails are calculated for different response times (the exact generation is further described in the method section; see also Ridderinkhof et al., 2004). These differences are plotted as a function of the mean reaction time, revealing a development of activation and inhibition of the response routes over time. The normal patterns of these delta plots for reaction time and accuracy differences are described first before discussing possible deviations on ASD delta plots.

As illustrated by the dual process model, the difference in reaction time between the trials is usually small for fast response times as responses are triggered by the automatic route in both trial types. With increasing mean response time the difference in reaction time between the incongruent and congruent trials increases: Responses on congruent trials are still fast due to the concord of automatic and deliberate response routes, but responses on incongruent trials slow down as the deliberate response route is activated too and competes for response execution. This increase in reaction time difference continues until reaching the transition point of the delta plot where enough time has passed to build up the selective inhibition of the automatic response. From this transition point on the difference in reaction time between the trial types decreases: Inhibition becomes stronger and the deliberate response route dominates more easily on incongruent trials, resulting in faster responses. This development is displayed by the plot: The more inhibition is exhibited, the more negative the gradient of the delta plot slope (Burle, Possamai, Vidal, Bonnet, & Hasbroucq, 2002). Thus, to assess inhibition differences between ASD and control participants the transition points and gradients of the reaction time delta plot slopes have to be compared between the groups.

Not only delta plots of reaction time differences reveal important information. Delta plots of differences in accuracy between the trial types visualize the necessary time to activate the deliberate response route (De Jong et al., 1994). The difference in accuracy is the largest on small reaction times when there are many errors on incongruent trials due to the dominating automatic response route. With increasing reaction time the deliberate response route gets more activated, resulting in more correct responses on incongruent trials. Therefore, the ascending slope of the first part of the plot displays the process of activating the deliberate response route and can be used in the present study to compare the activation processes between ASD participants and controls (Ridderinkhof, 2002).

The current study

In the present study inhibition was quantified by the Simon task (Simon, 1970), a spatial interference task. The Simon task, the Flanker task and the Stroop task (Stroop, 1935) have often been lumped together as measurements of interference of irrelevant stimulus information. However, differences in the cognitive models underlying the different tasks (Kornblum, Hasbroucq, & Osman, 1990), different associations between the tasks and active brain areas (Liu, Banich, Jacobson, & Tanabe, 2004) and differences in reaction time distributions between the tasks (Pratte, Rouder, Morey, & Feng, 2010; Speckman et al., 2008) have been outlined. Thus, although inhibition performances of ASD participants on these tasks have been compared with each other, these comparisons probably did not reveal a valid picture of interference inhibition. Hommel (2011) argued that the results of the Simon task

are most straightforward to interpret as other cognitive mechanisms as selective attention are controlled for. The Simon task is further described in the method section.

Besides the earlier described within-trial temporal dynamics of inhibition and activation which have frequently been detected in the Simon task (e.g. Burle et al., 2002; Ridderinkhof, 2002; Stins, Polderman, Boomsma, & de Geus, 2007), between-trial changes were of interest in the current study, too. The interference effect has been found to decrease when the preceding trial corresponded with the current trial (incongruent - incongruent or congruent - congruent) and to amplify when the preceding trial did not correspond (congruent - incongruent or incongruent – congruent; Hazeltine, Akçay, & Mordkoff, 2011; Stürmer, Leuthold, Soetens, Schröter, & Sommer, 2002), due to a possible priming effect of the preceding trial *(*Hommel, 2011; Stürmer & Leuthold, 2003). Thus, in the current study inhibition problems in ASD should get more obvious on incongruent trials following a congruent trial as more inhibition was necessary to restrain the primed automatic response route.

To control for the influence of comorbid ADHD symptoms on inhibition functioning, ASD and ADHD symptoms of all participants were quantified with respectively a score on the Dutch version of the Autism Quotient questionnaire (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001; Dutch Version: Hoekstra, Bartels, Cath, & Boomsma, 2008) and a score on the Dutch version of the ADHD DSM-IV rating scale (DuPaul, Power, Anastopolous, & Reid, 1998; Dutch version: Kooij & Buitelaar, 2000). First, the predictive value of the autism score for both reaction time and accuracy differences was checked. Next, the hyperactivity-impulsivity and inattention score were entered as predictive variables into the hierarchic regression model to investigate the additional effect of ADHD features on interference inhibition in ASD. According to Barkley's inhibitory theory (1997) the hyperactivity-impulsivity features of ADHD are associated with inhibitory problems while the inattentive ADHD type is not. However, research outcomes have not always supported this distinction (Houghton et al., 1999; Nigg, Blaskey, Huang-Pollock, & Cappley, 2002). In the present study the hyperactivity-impulsivity score was entered as second predictor and the inattention score as third predictor for inhibition performance on the Simon task.

Summing up, in the present study the Simon task was administered to both ASD and healthy control adults with the aim of exploring the groups' temporal dynamics of interference inhibition. The inhibition performance was analyzed by a delta plot analysis. It was hypothesized that ASD participants would show a longer build up time of selective inhibition of the automatic route and an overall less proficient inhibition compared with controls. Consequently, it was expected that the difference in reaction time between the trial types of ASD participants would be bigger than of controls at any time,

that the transition point would be reached later and that the decrease of the slope would be less steep for ASD participants. These differences between the groups were hypothesized to get more obvious on non-corresponding trial sequences. As correct responses on the Simon task depend on the inhibition of incorrect, fast responses, the accuracy on fast response trials was expected to be worse for ASD participants compared to controls. The impairment on interference inhibition was expected to be most predictable by a combination of high autism and high hyperactivity-impulsivity scores.

Method

Participants

Participants with ASD were recruited at different departments of the Dr. Leo Kannerhuis and by advertising on the website of the Dutch Autism Society. As controls, acquaintances of employees of the Dr. Leo Kannerhuis were approached. Besides the requirements of a male gender and an age between 18 – 35 years, the following exclusion criteria of the broader research program were used for all participants: an intelligence quotient (IQ) below 80, estimated by the performance on two subtests of the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 2005), history or a current record of cardiac disease and complaints, respiratory problems, liver- and/or kidney failure, a current diagnosis of depression and the use of beta-blockers or antidepressant medication. All participants were of Caucasian ethnicity.

Twenty-three male adults with ASD were tested. Data from one participant was excluded because the participant was not able to finish the Simon task due to a stress reaction. Consequently, the ASD group of the present study consisted of 22 male participants (age range: 18 – 35 years) of whom 5 were diagnosed with autism disorder, 5 with Asperger syndrome and 12 with a pervasive developmental disorder not otherwise specified (PDD-NOS).

For the control group 20 healthy male adults were tested. Due to an IQ-score of below 80, one participant was excluded, resulting in a group of 19 control participants (age range: 18 – 34 years).

To achieve a more pure ASD sample, the inclusion criteria for the sample for the delta plot analysis were tightened: Only ASD participants scoring 111 or higher on the AQ were included. The sample of the distributional analysis counted 16 males with ASD (age range: 18 – 32 years) and 16 control participants matched on age and IQ. This stricter sample is further referred to as the distributional sample and is only used in the delta plot analysis, while the bigger sample for the remaining analyses is called the regression sample. The samples did not differ significantly from each

other in age or intelligence ($F < 1$, *ns*). Nonetheless, the age and intelligence distributions are displayed for both samples individually in Table 1 to reveal a more detailed insight into the group descriptives.

Group descriptives

Table 1 provides also an overview of the composition of the questionnaire scores, divided into distributional sample and regression sample. Group differences were analyzed by ANOVA's, using an overall alpha level of .05.

Regression sample

The regression groups differed significantly on age and estimated IQ: controls were older and showed a higher estimated intelligence. Correlation analysis revealed that the IQ was not significantly correlated with the dependent variables of the Simon task (reaction time: $r = .16$, $p = .33$; accuracy: $r = .01$, $p = .96$) and age was only correlated with reaction time difference ($r = -.40$, $p = .01$) but not with difference in accuracy ($r = -.13$, $p = .41$).

As expected, the ASD group had a significantly higher AQ score than the controls. The ADHD comparison was based on twenty-one instead of twenty-two ASD participants given that one participant failed to complete this questionnaire. No group differences on the total score of attention deficit or hyperactivity-impulsivity were detected.

Distributional sample

The matched groups did not differ significantly on age, estimated IQ or any ADHD score. As expected, ASD participants scored significantly higher on the AQ questionnaire than controls.

Table 1

Group Descriptive and Mean Questionnaire Scores per Sample

	Group				
Regression sample	ASD (N=22)		Controls (N=19)		ANOVA
Measure	*Mean*	*SD*	*Mean*	*SD*	*F*
Age	22.69	3.92	26.04	4.83	6.04*
Estimated IQ	107.86	12.91	117.79	13.73	5.68*
Autism Quotient score	123.14	18.13	88.79	12.99	47.19**
Attention deficit score[1]	14.19	5.33	12.21	6.62	1.09
Hyperactivity-impulsivity score[1]	14.48	7.32	10.37	6.75	3.38
Distribution sample	ASD (N=16)		Controls (N=16)		ANOVA
Measure	*Mean*	*SD*	*Mean*	*SD*	*F*
Age	23.04	4.37	24.98	4.46	1.55
Estimated IQ	107.62	13.66	115.00	12.02	2.62
Autism Quotient score	131.75	12.46	89.56	13.60	83.67***
Attention deficit score	13.69	6.70	12.81	6.90	< 1
Hyperactivity-impulsivity score	14.25	9.00	11.69	6.49	< 1

Note. [1] ADHD scores are based on 21 ASD participants.
*p < .05. **p < .01. ***p < .001.

Procedure

The Simon task was one of several administered tasks of a research program. Questionnaires for the program were filled in on the internet within three days after completing the tasks. The two questionnaires used in the present study plus the applied intelligence measurement are now described further.

Material

Autism questionnaire

The Dutch version of the Autism-Spectrum Quotient (AQ; Baron-Cohen et al., 2001; Dutch Version: Hoekstra et al., 2008) quantifies where participants are on the autism spectrum. It is specifically designed for self-report by adults with an average IQ or above. Participants rated on a 4-point scale (definitely agree – definitely disagree) in which degree the 50 statements apply to them. To generate a total score several items had to be recoded because of the negative formulation of the statements. The

validation study of the Dutch version of the AQ in a Dutch population by Hoekstra et al. (2008) revealed a discriminating cut-off score of 111. This cut-off score was based on a scoring mechanism where all scores were summed up, resulting in a total AQ score with a maximum of 200. The internal consistency and reliability of the Dutch version of this questionnaire are good, α = .71 and r = .78 (Hoekstra et al., 2008). In the present study only ASD participants scoring 111 or higher on the AQ were included in the distributional analysis.

ADHD questionnaire

The Dutch version of the ADHD DSM-IV rating scale (DuPaul et al., 1998; Dutch version: Kooij & Buitelaar, 2000) represents the factors inattention and hyperactivity-impulsivity of the DSM-IV criteria for ADHD. It was used in the current study to gather information about the prevalence of ADHD features in the participants. All participants rated the frequency of their experienced attention deficit and hyperactivity-impulsivity in the last six months for 23 items on a four point scale (0 = rarely or never, 1 = sometimes, 2 = often, 3 = very often). A sum score of attention deficit items and a sum score of hyperactivity and impulsivity items were generated for the present study. The reliability of the scale is good, the validity is moderate (Kooij et al., 2008).

Intelligence measures

The subtests 'Vocabulary' and 'Block Design' from the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 2005) were administered. The vocabulary subtest correlates highly with the verbal IQ score, while the block design subtest has a moderate correlation with the performance IQ score. The subtests together can give a valid and reliable estimation of the total IQ (Jeyakumar, Warringer, Raval, & Ahmad, 2004). In the current study participants were excluded if the estimated total IQ was below 80 because they might not have been able to fully understand and complete the questionnaires and tasks.

Simon task

The Simon task is a motor interference task administered on a computer. The rest mode of the screen was a grey background with a black square fixation point in the middle of the screen. The stimulus on each trial was either a green or blue circle. The interval between the appearing stimuli varied between 1750ms and 2250ms (with steps by 50ms and a mean of 2s).

Response keys were the 'Z'-key and '?'-key which had to be pressed on the keyboard with respectively the left or right index finger. The responses had to be made based on the color of the

stimulus, independent of its location. The correct response for a green stimulus was pressing the left-hand button. For a blue stimulus the correct response was made with the right-hand button. The maximum response time during the tasks was 1500ms. The stimulus disappeared as soon as the participant responded and was presented for 1500ms max. A sequence of events of the Simon task is illustrated in Figure 2.

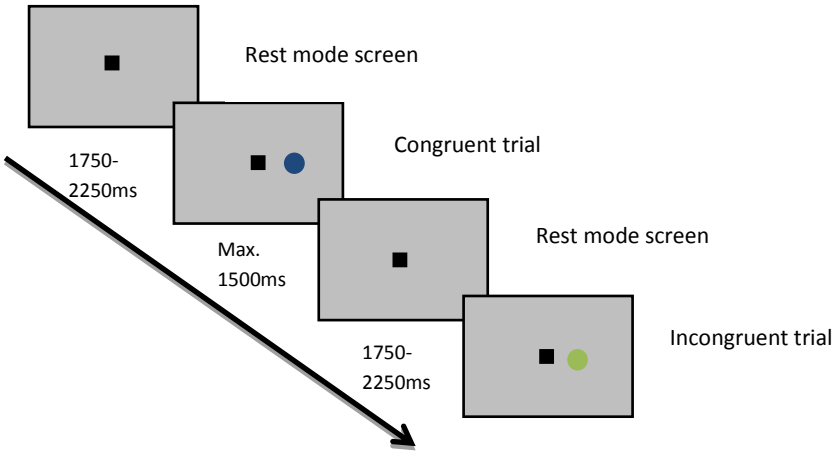

Figure 2: Sequence of events on an experimental trial in the Simon Task. Participants are trained to react with the right response key on a blue stimulus and the left response key on a green stimulus. In the congruent condition the side of display of the stimulus matches the side of the correct answer. In the incongruent condition there is a mismatch between the side of presentation of the stimulus and the side of response.

The experiment consisted of a practice and an experimental part. First, participants completed 72 practice trials to learn the association between color and response key. Here, the stimulus appeared in the middle of the screen and the participants were asked to respond as fast as possible according to the color of the stimulus.

The experimental part first consisted of twelve practice trials followed by four blocks of 60 experimental trials each. In each trial a blue or green colored stimulus appeared on the right or left side of the fixation point. Participants were always supposed to respond with the left key to green stimuli and with the right key to blue stimuli, independent of the stimulus location. In a congruent trial, a blue stimulus appeared on the right side of the screen or a green stimulus appeared on the left side of the screen, matching the side of the correct response. In an incongruent trial the green stimulus appeared on the right side of the screen or a blue stimulus appeared on the left side of the screen, opposite to the correct response side.

The color and the location of the stimulus were determined randomly, but with the restriction that each stimulus appeared equally often in both color and location. The congruent and incongruent

trials were evenly distributed on the left and right response key. The sequence of the trials was randomized by the computer. Between the blocks, there was the possibility to take a short break and to start the new block whenever ready. The duration of the whole Simon task varied between 20-30 minutes, depending on the participant's wish for breaks and need for instruction.

Statistical analyses

For the first four analyses the regression sample was used. Only the delta plot analysis was applied on the distributional sample.

First, it was controlled for differences between the groups on psychomotor speed which has been found to influence cognitive performances in ASD (Goldstein, Johnson, & Minshew, 2001). By an ANOVA the mean reaction time on the last 60 trials of the neutral practise part was compared between the groups. In addition, the correlation between the psychomotor speed and respectively the AQ, the hyperactivity-impulsivity and inattention score and all outcome measures of the Simon task was checked.

Second, it was explored whether the ASD and/or ADHD features predicted the interference inhibition on the Simon task and, therefore, whether they would have to be included as covariates in following analyses. By a multiple regression analysis the predictive values of the AQ score and the hyperactivity-impulsivity score and attention deficit score of the ADHD DSM-IV questionnaire were examined for the overall difference in accuracy and reaction time between the trial types for all participants.

Third, the interference effect and possible group differences on the overall measures were investigated to reveal outcome measures that can be compared with those of earlier research before focussing on the new type of analysis. The trial type (congruent vs. incongruent) as within-subject factor and group (ASD vs. controls) as between-subject factor were entered in a MANOVA to test the effect on the dependent variables mean response time and overall accuracy. This analysis was possibly repeated with ASD and/or ADHD features as covariates.

Fourth, the sequential effects for reaction time were explored by a second repeated measure ANOVA. The interference effect on different trial type sequences were compared with each other and between the groups, again repeated with a possible covariate, depending on the multiple regression analyses.

Fifth, the delta plot analysis was applied on the distributional sample: Delta plots for both reaction time and accuracy were generated based on the description from Ridderinkhof et al. (2005). For each participant the responses of the experimental trials were ranked ordered on reaction time, separately for the congruent and incongruent conditions. The responses were divided into five equal sized bins per condition whereof mean reaction time and overall accuracy were determined. The interference effect size was calculated by subtracting the reaction time/accuracy of the congruent trial from the reaction time/accuracy of the incongruent trail. This interference effect size was plotted as a function of mean response time per bin, resulting in the so called delta plots. The four slopes of these delta plots (slope 1 connecting the data points from bin 1 and bin 2, slope 2 connecting the data points from bin 2 and bin 3 etc.) were used for further analysis: Repeated measure analyses with the slopes as within-subject factor and group as between-subject factor were computed to analyze the temporal dynamics of the interference effect of both accuracy and reaction time. Next, differences between the groups on each of the four slopes of reaction time and the first slope of accuracy were explored by ANOVAs as had been suggested by Ridderinkhof et al. (2005). These two analyses were possibly repeated with ASD and/or ADHD features as covariates, depending on the outcome of the earlier multiple regression analysis.

Missing data and outliers

In the individual reaction time distribution on the Simon task extreme scores that were four standard deviations above or below the mean reaction time of the participant, including missing scores, were excluded. The groups did not differ significantly on outliers or missing data in neither the distributional analysis sample, both $F < 1$, *ns,* nor the regression sample, respectively $F(1, 41) = 3.87$, $p = .06$; $F < 1$, *ns*. On group level, no outliers on reaction time or accuracy based on three standard errors were found. For the reaction time analysis all erroneous responses were excluded. After all corrections, a minimum of 200 out of 240 experimental trials were includable for each participant.

Results

Psychomotor speed

The groups did not differ in psychomotor speed ($F < 1$, *ns*). The psychomotor speed did not correlate with any of the further described outcome measures (all $p > .1$).

Multiple regression analysis

The variables AQ sum score and attention deficit total score and hyperactivity-impulsivity total score correlated with each other, but not very high, all $r = < .60$. Therefore, the assumption of no perfect multicollinearity was satisfied. The results of the multiple regression analyses are illustrated in Table 2.

The AQ sum score was neither significantly predictive for the difference in reaction time between the trials, $F(1, 39) = 1.87$, $p = .18$, nor for the difference in accuracy , $F < 1$, *ns*. The hierarchical model of the hyperactivity-impulsivity score and the attention deficit score revealed that none of the ADHD variables was significantly predictive for accuracy: hyperactivity-impulsivity: $F(1, 38) = 3.50$, $p = .07$, hyperactivity-impulsivity & inattention: $F(2, 37) = 1.85$, $p = .17$. For the difference in reaction time, hyperactivity-impulsivity was the only predictive factor, $R^2 = .20$, $F(1, 38) = 9.32$, $p = .004$. Adding attention deficit features had no significant effect on the explained variance $R^2 = .21$, $t = -.64$, $p = .53$.

A higher hyperactivity-impulsivity score predicted a smaller difference in reaction time between incongruent and congruent trials, $b = -1.06$, $t = -3.05$, $p = .004$. A correlation check revealed that the hyperactivity-impulsivity score was significantly correlated with the reaction time on incongruent trials, $r = -.35$, $p = .03$, but not on congruent ones, $r = -.22$, $p = .17$.

Thus, the hyperactivity-impulsivity score was the only predictive value for the difference in reaction time between the trial types. The negative predictive effect was the result of a decrease in reaction time on incongruent trials when the hyperactivity-impulsivity score increased. Therefore, the hyperactivity-impulsivity score was included as a covariate in all following analyses where reaction time difference was the dependent variable.

Table 2

Predictive Value of Questionnaire Scores for Reaction Time and Accuracy

	Dependent measures							
	Overall difference in reaction time				Overall difference in accuracy			
Predictor	*b*	*ß*	R^2	ΔR^2	*b*	*ß*	R^2	ΔR^2
AQ	-.16	-.21	.05	-	.10	.15	.02	-
HI	-1.06**	-.44	.20	-	.62	.29	.08	-
HI	-.90*	-.38*	.21	.01	.49	.23	.09	.01
AD	-.33	*-.12*			.26	.10		

Note. AQ = Autism Quotient, HI =Hyperactivity-Impulsivity, AD = Attention deficit, AQ; AQ, HI and AD were entered hierarchically. First, only AQ was entered. Second, a new model was started as AQ had no predictive value: HI was entered first and then AD was added, based on the hypotheses of the current study.
*p < .05. **p < .01.

Overall performance

The distributional sample and regression sample did not differ significantly from each other on overall measures (all $F < 1$, *ns*). Because the overall measures were used as dependent variables for the regression analysis, the results of this sample are described here.

The typical interference effect was confirmed as both groups were slower, $F(1, 39) = 107.56$, $p < .001$, $ES = .73$, and making more errors, $F(1, 39) = 12.18$, $p < .001$, $ES = .24$, on incongruent trials than on congruent ones. There was no interaction effect with group for neither reaction time nor accuracy (both $F < 1$, *ns*). Inserting hyperactivity-impulsivity features as a covariate did not change the results significantly.

These overall results suggest that both groups were equally influenced by the interference effect of the task and that there was no difference in overall reaction time or accuracy between the groups. The overall reaction time and accuracy of the groups are illustrated respectively in Figure 3 and Figure 4.

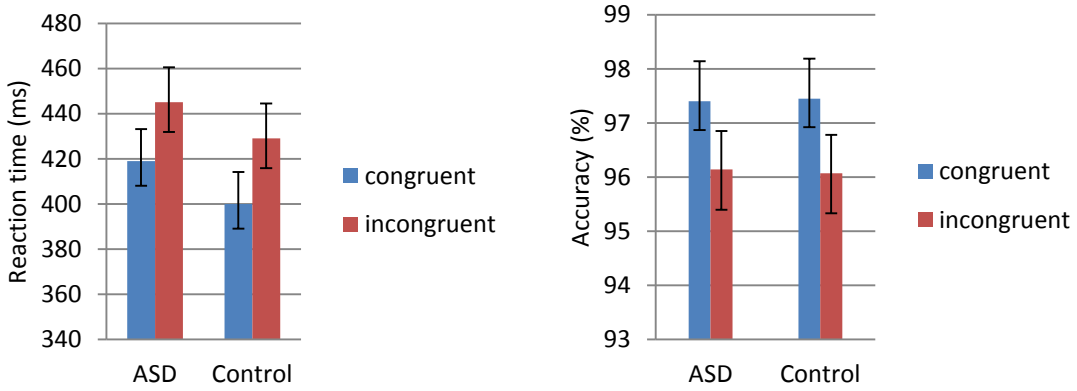

Figure 3 and 4: Overall effects of respectively reaction time and accuracy between the trial types and groups.

Sequential effect

A repeated measure ANOVA revealed a significant sequential effect, $F(3, 117) = 109.55$, $p < .001$, $ES = .74$ on reaction time. Pair wise comparisons showed that the reaction time difference between the trial types was the highest on incongruent trials proceeding a congruent trial ($M = 448.18$) and the lowest on congruent trials following a congruent trial ($M = 388.74$). They differed significantly (all $p < .001$) from each other and from the trials proceeding incongruent trials (congruent: $M = 423.62$, incongruent: $M = 422.25$). Trials proceeding incongruent trials did not differ from each other in reaction time difference, $p = .71$. There was no interaction effect with group, $F < 1$, *ns*. The replication of the analysis with hyperactivity-impulsivity as covariate revealed no significant different results. Thus,

although the interference effect on incongruent trials was strengthened after congruent trials as had been expected, the groups were still equally influenced by the interference effect.

Distributional analysis of the distributional sample

The differences in reaction time between incongruent and congruent trials changed significantly with increasing response time, $F(3, 90) = 4.12$, $p = .008$, $ES = .12$. The interaction effect with group was not significant, $F(3, 90) = 2.47$, $p = .07$, $ES = .08$.

The slopes of accuracy were not normally distributed. It was justified to run the analyses without having to transform the data because the Skewness and Kurtosis values were within a spectrum of -1.5 and 1.5 (Field, 2009). Mauchly's test indicated that the assumption of sphericity had been violated for accuracy, $X^2(5) = 21.73$, $p = .001$. Therefore, degrees of freedom were corrected using Huynh-Feldt correction estimates of sphericity ($\varepsilon = .78$). The differences in accuracy between incongruent and congruent trials changed significantly with increasing response time, $F(2.34, 70.06) = 7.20$, $p = .001$, $ES = .19$). No interaction effect with group was found, $F < 1$, *ns*.

The direct comparisons of each slope part of the reaction time delta plots and the first slope part of the accuracy plots between the groups revealed no significant differences between the groups: slope 1 RT: $F < 1$, *ns*; slope 2 RT: $F(1, 30) = 2.47$, $p = .13$; slope 3 RT: $F < 1$, *ns*; slope 4 RT: $F(1, 30) = 2.80$, $p = .11$; slope 1 accuracy: $F(1, 30) = 1.59$, $p = .22$.

These results suggest that differences in reaction time and accuracy between the trials types changed with increasing response time. These temporal changes of interference effect were not significantly different for ASD and control participants. The delta plots of the differences in reaction time and accuracy of both groups are respectively displayed in Figure 5 and Figure 6.

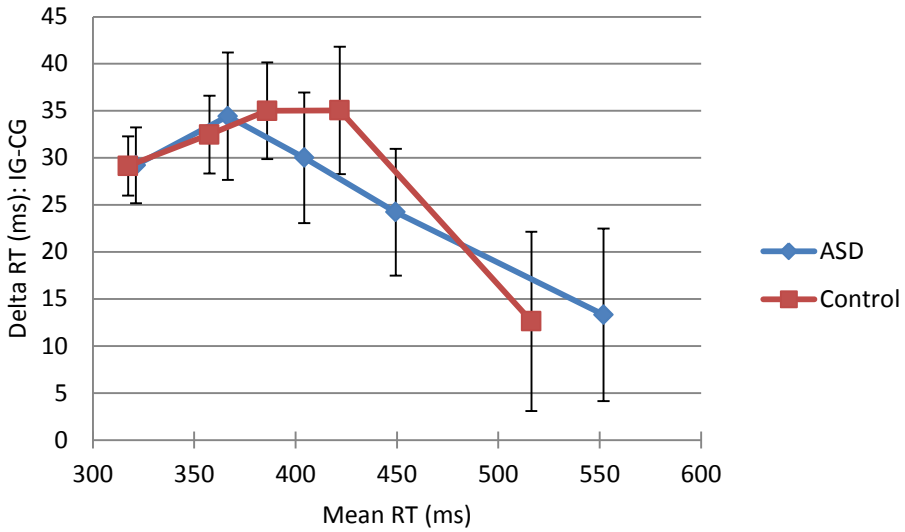

Figure 5: Difference in reaction time between the trial types as a function of response time. CG = congruent trails, IG = incongruent trails.

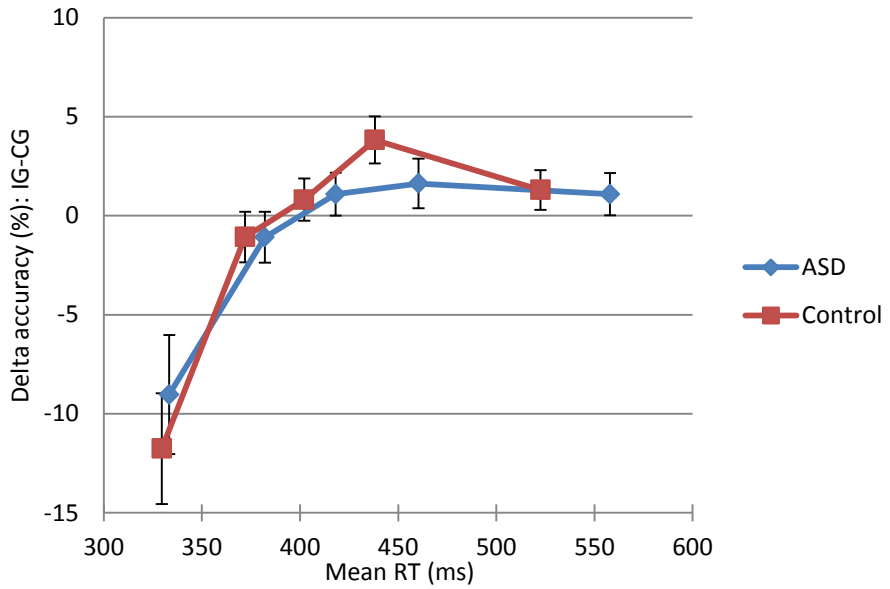

Figure 6: Difference in accuracy between the trial types as a function of response time. CG = congruent trials, IG = incongruent trials.

These analyses were repeated for the reaction time differences and added with hyperactivity-impulsivity features as covariate. The covariate changed the results of the repeated measure analysis significantly, as the temporal change of differences in reaction time between incongruent and congruent trials did not reach the significant level, $F(3, 87) = 2.35$, $p = .08$, $ES = .08$. The interaction effect with group was again not significant, $F(3, 87) = 2.34$, $p = .08$, $ES = .08$. The outcomes of the comparisons of each separate slope part between the groups were not significantly changed by the covariate. These

results suggest that hyperactivity-impulsivity features influence the temporal change of interference inhibition in all participants.

Discussion

The major aim of the present study was to investigate the temporal dynamics of inhibition in adults with ASD and healthy controls based on the interference effect of the Simon task. The dual process model predicts an increase of inhibition with increasing reaction time and, therefore, a decrease of interference effect. This was indeed found in the present study: the differences in reaction time and accuracy between incongruent and congruent trials changed significantly with increasing response time. Contradicting to the hypothesis, the temporal change of interference effect was the same for ASD participants and controls for both reaction time and accuracy, even when comparing each slope part separately. Also on past congruent trials, where the interference effect was expected and indeed found to be strengthened, both groups were again equally influenced by the effect and did not differ on inhibition functioning. These outcomes suggest that adults with ASD do not differ from healthy controls in response activation and interference inhibition. This corresponds with earlier studies which stated that inhibition problems in children do not necessarily persist into adulthood as they did not find any inhibition problems in adults with ASD compared with healthy controls (Happé et al., 2006; Johnston et al., 2011; Ozonoff & Jensen, 1999).

In line with the temporal development of inhibition, a remarkable shift of accuracy can be seen for both groups around a mean reaction time of 350-400ms. While below this reaction time the interference effect resulted in more errors on incongruent than on congruent trials, on higher reaction times the pattern reserved as more erroneous responses were observed on congruent trials than on incongruent ones. This is consistent with the finding by Eimer and Schlagenhecken (1998) who revealed that the priming effect in a task as the Simon task lasts only for about 200ms. After that a reversed activation trend can be seen. Consequently, the increase of errors on the congruent trials with increasing reaction time can be explained by inhibition of the initially primed and activated correct response (De Jong et al., 1994). This phenomenon is the same for both groups. Therefore, the groups show, again, no differences in inhibition function.

These results suggest that interference inhibition in adults with ASD might not be deviant at all and that both groups are equally influenced by the interference effect of the Simon task. However, visual inspection of the delta plot figures would lead one to argue the contrary: In ASD participants the inhibition seems to build up faster and reaches the threshold of inhibiting the direct activation route earlier than in controls. After reaching this threshold the negative gradient of ASD participants is less

steep than the gradient of controls, suggesting a less proficient inhibition in ASD (Burle et al., 2002). The absence of a significant interaction effect of time with group might be explained by the low power of the repeated measure analysis (1-ß = .596) as there was a 40% chance of missing a significant effect. A future study with a bigger sample is necessary to make a more powerful conclusion about differences in temporal dynamics of inhibition between ASD and control participants.

The second aim of the current study was to control for a possible influence of the frequently comorbid disorder ADHD (Rommelse et al., 2010). The predictive value of autism and ADHD features for the performance on the Simon task was examined. Contrary to the hypothesis autism features measured with the Autism Quotient did not have any predictive value. Hyperactivity-impulsivity assessed with the ADHD DSM-IV checklist was the only significant predictor for reaction time differences between the trial types. By adding hyperactivity-impulsivity features as covariate to the delta plot analysis of reaction time differences, the temporal change of the interference effect decreased until non-significant level. This decrease might partly, but not fully be explained by the automatic decrease of the main within-subject effect when adding a covariate to a repeated measure ANOVA (Thomas, Annaz, Ansari, Serif, Jarrold et al., 2009). More relevant, it might be that hyperactivity-impulsivity features are of different influence on inhibition on different response times. A quick check revealed that indeed hyperactivity-impulsivity features were significant predictors for the reaction time differences in the first four bins but not in the last bin (for more details see Table 1 in appendix). In contrast to Barkley's hypothesis (1937) that inhibition deficits are the key problem of the hyperactive and impulsive ADHD subtype, the interference effect was weaker instead of stronger in hyperactive, impulsive participants. The accuracy on these trials was not deviant and in addition independent of hyperactivity-impulsivity features, ruling out a speed/accuracy trade off explanation. One could state that the general higher activation level of hyperactive, impulsive participants implies a higher activation level of both response routes. Through the higher activation of the deliberate response route the threshold of a deliberate response on an incongruent trial might be reached earlier. This explanation is in line with the predictive differences of hyperactivity-impulsivity symptoms: the predictive value was strongest on bin 2 and 3 where the built up of selective inhibition of the automatic route is still in progress and a domination of the deliberate response route might be achieved by a higher activation level. For bin 5, hyperactivity-impulsivity was not predictive: with slow response time enough time has passed to inhibit the automatic response route completely so that the deliberate response route dominates also without higher activation level.

In contrast to this explanation, distributional analysis has revealed no difference in direct activation between ADHD and control participants on the Flanker task (Ridderinkhof et al., 2005) and Nigg (2001) stated that go response on Go/No-Go tasks are slow rather than fast in ADHD participants. However, the distribution of hyperactivity-impulsivity versus inattentive types in both of the before mentioned samples was not explored. Inattentive participants might have eliminated a potential difference. However, no differences were found comparing the combined and inattentive type of ADHD on the Stroop task (Houghton et al., 1999). Combined and inattentive ADHD groups even performed as well as controls on interference control, although ADHD participants showed a slowed response activation on the Stroop task (Nigg, Blaskey, Huang-Pollock & Cappley, 2002). But as has been noted before, performances on these interference tasks are not directly comparable to each other (for a short review, see Hommel, 2011). Summing up, the processes causing the negative relationship between hyperactivity-impulsivity features and inhibition of interference need to be examined further. However, it seems to be the case that hyperactivity-impulsivity features play a role in inhibition processes in both ASD and controls and that this influence changes with increasing reaction time.

Limitations and future studies

Limitations of the present study might reveal other possible explanations of the results. First, the ASD sample for the distributional analyses was selected based on the score on the Autism Quotient questionnaire. The AQ is originally designed to measure autistic traits that may be continuously distributed in the population (Baron-Cohen et al., 2001). People diagnosed with ASD are the upper extreme of a normal distribution of specific traits in the population. Therefore, a cut-off score on the questionnaire needs to differentiate between 'normal' and 'extreme'. Which amount of ASD traits is experienced as normal is depending on cultural variables and the distribution of the traits in the population. Hence, a cut-off score needs to be validated in each population individually (Hurst, Mitchell, Kimbrel, Kwapil, & Nelson-Gray, 2007). The employability of the AQ for discriminating clinical samples from typical samples has only been investigated rarely (Wakabayashi, Baron-Cohen, Wheelwright, & Tojo, 2006; Woodbury-Smith et al., 2005) and only once in a Dutch sample with the now used Dutch version of the AQ (Hoekstra et al., 2008). While the cut-off score of 26 (based on the Baron-Cohen scoring method) had good discriminative validity in the UK population (Woodbury-Smith, Robinson, Wheelwright, & Baron-Cohen, 2005) and has frequently been used in the international literature (e.g. Austin, 2005; Hurst, Mitchell, Kimbrel, Kwapil, & Nelson-Gray, 2007), 54% of the current ASD sample scored below 26, in spite of a recently confirmed ASD diagnose. Also the distribution above and below this cut-off score of the Dutch ASD sample of the study by Hoekstra et al. (2008) shows the need for

culture dependent cut-off scores for the AQ. However, also the cut-off score by Hoekstra et al. (2008), based on a Dutch sample, might not reliably differentiate between ASD and control groups as 27% of the current ASD sample scored below this score. A quick check revealed that it were not only PDD-NOS diagnosed participants scoring below the cut-off by Hoekstra et al. (2008), but also participants with autism disorder or Asperger. In addition, the relationship between the AQ and valid diagnostic measures of autism has not been investigated yet. Therefore, one has to ask if the AQ was able to differentiate the present groups in a valid way.

Moreover, the AQ is a self-report instrument. Persons with autism were found to have difficulties to self-report their autism and comorbid symptoms due to problems in conversation and introspection (Capps, Kehres, & Sigman, 1998; Mazefsky, Kao, & Oswald, 2011; Williams, & Happé, 2010). Ketelaars et al. (2008) concluded that self-report questionnaires as the AQ are not adequate for differentiating less severe ASD patients from other patient groups. Their Dutch translation of the AQ did not differentiate between ASD and non-ASD patients although the ASD patients were all diagnosed based on a semi-structured interview, the Autism Diagnostic Interview – Revised (Lord, Rutter, & LeCouteur, 1994), taken from at least one of the parents and observations from the Autism Diagnostic Observation Schedule– Generic (Lord et al., 200). In sum, some might argue that our current sample might not have been a pure enough ASD group and given the limitations of the AQ it is not evident whether this is indeed the case. An impure sample might account for absent group differences and/or undetected predictive effects of autism for inhibition. In addition, an impure sample would not allow any concrete statements about inhibition in ASD.

As a second limitation the event rate of the stimuli was quite low with a between-stimulus interval of 1750ms to 2250ms. Consequences of the low event rate can be seen in the near-ceiling accuracy (M=96%). In addition, the low event rate might explain why the interference effect was not of bigger influence on the ASD group than on the controls. The ability to inhibit a response might depend on the event rate of stimuli as a non-optimal event rate requires an adaption of the own arousal state. This regulation has been hypothesized to be limited in ASD (Raymaekers, van der Meere, & Roeyers, 2004). However, evidence is mixed as adults but not children with ASD showed deviant performances on 1s event rates (Geurts, Begeer, & Stockmann, 2009; Raymaekers, Antrop, van der Meere, Wiersema, & Roeyers, 2007; Raymaekers et al., 2004). It does not seem to be clear if these contradictive results come forth of different outcome measures or a developmental change in arousal regulation (Geurts et al., 2009). On any account, a faster presentation rate around one second would be preferable in future research to investigate if interference inhibition problems in ASD adults get more obvious on fast event rate.

Third, as has been noted, the sample of ASD participants that met ASD criteria was quite small and the inability to find group-related differences might be based on a low power. In line with this argument a trend was detected for the interaction of the change of interference effect over time and the group condition (p = .067). Besides, as argued earlier, the sample might not have been a pure one which might have additionally decreased the chance of finding significant differences between the groups. Therefore, a bigger and more pure sample should be aimed at in future studies.

Summing up, the present study should be replicated with a bigger sample, using another instrument to differentiate validly between ASD and non-ASD participants and presenting the stimuli on a faster event rate. Besides correcting these limitations, future research should aim at investigating the reasons for the variability of results of ASD samples on different inhibition tasks. Different inhibition tasks might require inhibitory control on different levels. For example, Johnston et al. (2011) stated that Flanker and Stroop tasks require control at response selection level while the Stop or Go/No Go tasks might require inhibition of response execution. By investigating the temporal dynamics of inhibition on different tasks, one will be able to make more precise statements over inhibition performance on different functional levels and different reaction times, and over within- and between-trial executive changes of ASD samples. As an additional aspect of interest for future studies, the present study showed that hyperactivity-impulsivity features have a positive effect on interference inhibition and that this influence changes over time. More research is needed to investigate the role of hyperactivity-impulsivity symptoms in interference inhibition in ASD and healthy controls. The advantages of the distributional analysis when investigating inhibition functioning in clinical samples as ASD or ADHD became apparent in the present study and the study by Ridderinkhof et al. (2005) and researchers are encouraged to use delta plot analyses to compare inhibition performances in future studies.

References

American Psychiatric Association (2000). *Diagnostic and statistical manual of Mental Disorders* (4th ed., text revision). Washington, DC, American Psychiatric Association.

Austin, E. J. (2005). Personality correlates of the broader autism phenotype as assessed by the Autism-Spectrum Quotient (AQ). *Personality and Individual Differences, 38*, 451–460.

Bachelavier, J., & Loveland, K. A. (2006). The orbitofrontal–amygdala circuit and self-regulation of social–emotional behavior in autism. *Neuroscience and Biobehavioral Reviews, 30*, 97–117.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Construing a unifying theory of AD/HD. *Psychological Bulletin, 121*, 65-94.

Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from Asperger syndrome/high functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders, 31*, 5-17.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., Cohen, J. D., et al. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*, 624–652.

Bush, G., Spencer, T. J., Holmes, J., Shin, L. M., Valera, E. M., Seidman, L. J., et al. (2008). Functional magnetic resonance imaging of methylphenidate and placebo in attention-deficit/hyperactivity disorder during the multi-source interference task. *Archives of General Psychiatry, 65*, 102–114.

Burle, B., Possamai, C. A., Vidal, F., Bonnet, M., & Hasbroucq, T. (2002). Executive control in the Simon effect: an electromyographic and distributional analysis. *Psychological Research,66*, 324-336.

Capps, L., Kehres, J., & Sigman, M. (1998). Conversational abilities among children with autism and children with developmental delays. *Autism, 2(4),* 325-344.

Corbett, B. A., Constatine, L. J., Hendren, R., Rocke, D., & Ozonoff, S. (2009). Examining executive functioning in children with autism spectrum disorder, attention deficit hyperactivity disorder and typical development. *Psychiatry Research, 166 (2-3)*, 210-222.

De Jong, R., Liang, C., & Lauber, E. (1994). Conditional and unconditional automaticity: a dual process model of effects of spatial stimulus-response correspondence. *Journal of Experimental Psychology: Human Perception and Performance, 20(4),* 731-750.

DuPaul, G. J., Power, T. J., Anastopolous, A. D., & Reid, R. (1998). *ADHD Rating Scale –IV. Checklist, norms and clinical interpretation.* NY: The Guilford Press.

Eimer, M., & Schlaghecken, F. (1998). Effects of masked stimuli on motor activation: Behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception & Performance, 24*, 1737-1747.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception* & *Psychophysics*, *16*, 143–149.

Eskes, G. A., Bryson, S. E., & McCormick, T. A. (1990). Comprehension of concrete and abstract words in autistic children. *Journal of Autism and Developmental Disorders, 20,* 61–73.

Field, A. (2009). *Discovering Statistics using SPSS*. London, Sage.

Geurts, H. M., Begeer, S., & Stockmann, L. (2009). Brief report: Inhibitory control of socially relevant stimuli in children with high functioning autism. *Journal of Autism and Developmental Disorders, 39*, 1603-1607.

Geurts, H. M., Verté, S., Oosterlaan, J., Roeryers, 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(4*), 836-854.

Goldberg, M. C., Mostowsky, S. H., Cutting, L. E., Mahone, E. M., Astor B. C., Denckla, M. B., et al. (2005). Subtle executive impairment in children with autism and children with ADHD. *Journal of Autism and Developmental Disorders, 35,* 279-93.

Goldstein, G., Johnson, C. R., & Minshew, N. J. (2001). Attentional processes in autism. *Journal of Autism and Developmental Disorders, 31(4),* 433-440.

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, 26*, 25-39.

Hasbroucq, T., Possamai, C.-A., Bonnet, M., & Vidal, F. (1999). Effect of the irrelevant location of response signal on choice reaction time: an electromyographic study in man. *Psychophysiology, 36,* 552-526.

Hazeltine, E., Akçay, C., & Mordkoff, J. T. (2011). Keeping Simon simple: Examining the relationship between sequential modulations and feature repetitions with two stimuli, two locations and two responses. *Acta Psychologica, 136(2),* 245-252.

Hill, E. L. (2004). Evaluating the theory of executive dysfunction in autism. *Developmental Review, 24,* 189-233.

Hoekstra, R. A., Bartels, M., & Cath, D. C. (2008). Factor Structure, Reliability and Criterion Validity of the Autism-Spectrum Quotient (AQ): A Study in Dutch Population and Patient Groups. *Journal of Autism and Developmental Disorders, 38*, 1555-1566.

Hommel, B. (2011). The Simon effect as tool and heuristic. *Acta Psychologica, 136,* 189–202.

Houghton, S., Douglas, G., West, J., Whiting, K., Wall, M., Langsford, S., et al. (1999). Differential patterns of executive function in children with attention-deficit hyperactivity disorder according to gender and subtype. *Journal of Child Neurology, 14*, 801–805.

Hurst, R. M., Mitchell, J. T., Kimbrel, N. A., Kwapil, T. K., & Nelson-Gray, R. O. (2007). Examination of the reliability and factor structure of the Autism Spectrum Quotient (AQ) in a non-clinical sample. *Personality and Individual Differences, 43*, 1938–1949.

Jeyakumar, S. L. E., Warringer, E. M., Raval V. V., & Ahmad, S. A. (2004). Balancing the need for reliability and time efficiency: Short forms of the wechsler adult intelligence scale-III. *Educational and Psychological Measurement, 64*, 71-87.

Johnston, K., Madden, A. K., Bramham, J., & Russell, A. J. (2011). Response inhibition in adults with autism spectrum disorder compared to attention deficit hyperactivity disorder. *Journal of Autism and Developmental Disorders, 41,* 903-912.

Ketelaars, C., Horwitz, E., Sytema, S., Bos, J., Wiersma, D., Minderaa, R., et al. (2008). Brief report: Adults with mild autism spectrum disorders (ASD): Scores on the Autism Spectrum Quotient (AQ) and comorbid psychopathology. *Journal of Autism Developmental Disorder, 38*, 176–180.

Kooij, J. J. S., Boonstra, A. M., Swinkels, S. H. N., Bekker, E. M., de Noord, I., & Buitelaar, J. K. (2008). Reliability, validity, and utility of instruments for self-report and informant report concerning symptoms of ADHD in adult patients. *Journal of Attention Disorders, 11*, 445-458.

Kooij, J. J. S., & Buitelaar, J. K. (2000). Zelfrapportage vragenlijst over aandachtsproblemen en hyperactiviteit (not published).

Kooij, J. J. S., Buitelaar, J. K., van den Oord, E. J., Furer, J. W., Rijnders, C. A. T. H., & Hodiamont, P. P. G., (2004). Internal and external validity of attention-deficit hyperactivity disorder in a population based sample of adults. *Psychological Medicine, 34,* 1-11.

Kornblum, S., Hasbroucq, T., & Osman, A. (1990). Dimensional overlap: Cognitive basis of stimulus–response compatibility—A model and taxonomy. *Psychological Review, 97,* 253-270.

Liu, X., Banich, M. T., Jacobson, B. L., & Tanabe, J. L. (2004). Common and distinct neural substrates of attentional control in an integrated Simon and spatial Stroop task as assessed by event-related fMRI. *NeuroImage, 22(3),* 1097-1106.

Lord, C., Risi, S., Lambrecht, L., Cook, E.H, Leventhal, B. L., DiLavore, P. C. et al. (2000). The Autism Diagnostic Observation Schedule–Generic: a standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders, 30(3),* 205-223.

Lord, C., Rutter, M., & LeCouteur, A. (1994). Autism Diagnostic Interview-Revised: a revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, *24(5),* 659-685.

Mazefsky, C. A., Kao, J., & Oswald, D. P. (2011). Preliminary evidence suggesting caution in the use of psychiatric self-report measures with adolescents with high-functioning autism spectrum disorders. *Research in Autism Spectrum Disorders, 5(1),* 164-174.

Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin, 127(5),* 571-598.

Nigg, J. T., Blaskey, L. G., Huang-Pollock, C. L., & Rappley, M. D. (2002). Neuropsychological executive functions and ADHD DSM-IV subtypes. *Journal of American Academy of Child and Adolescent Psychiatry, 41*, 59-66.

Nydén, A., Gillberg, C., Hjelmquits, E., & Heiman, M. (1999). Executive function/attention deficits in boys with Asperger Syndrome, attention disorder and reading/writing disorder. *Autism, 3,* 213-228.

Ozonoff, S., Cook, I., Coon, H., Dawson, G., Joseph, R. M., Klin, A., et al. (2004). Performance on Cambridge Neuropsychological Test Automated Battery subtests sensitive to frontal lobe function in people with autistic disorder: Evidence from the collaborative programs of excellence in autism network. *Journal of Autism and Developmental Disorders, 34(2),* 139-150.

Ozonoff, S., & Jenssen, J. (1999). Specific executive function profiles in three neurodevelopmental disorders. *Journal of Autism and Developmental Disorders, 29*, 171-177.

Ozonoff, S., Strayer, D. L., McMahon,W. M., & Filloux, F. (1994). Executive function abilities in autism and Tourette syndrome: An information processing approach. *Journal of Child Psychology and Psychiatry, 35*, 1015–1032.

Pennington, B., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 37(1),* 51-87.

Pratte, M. S., Rouder, J. N., Morey, R. D., & Feng, C. (2010). Exploring the differences in distributional properties between Stroop and Simon effects using delta plots. *Attention, Perception, & Psychophysics, 72 (7*), 2013-2025.

Raymaekers, R., van der Meere, J., & Roeyers, H. (2004). Event-rate manipulation and its effect on arousal modulation and response inhibition in adults with high functioning autism. *Journal of Clinical and Experimental Neuropsychology, 26*, 74-82.

Raymaekers, R., Antrop, I., van der Meere, J. J., Wiersema, J. R., & Roeyers, H. (2007). HFA and ADHD: A direct comparison on state regulation and response inhibition. *Journal of Clinical and Experimental Neuropsychology, 29 (4),* 418-427.

Ridderinkhof, K. R. (2002). Micro- and macro-adjustments of task set: activation and suppression in conflict task. *Psychological Research, 66,* 312-323.

Ridderinkhof, K. R., Scheres, A., Oosterlaan, J., & Sergeant, J. A. (2005). Delta plots in the study of individual differences: new tools reveal response inhibition deficits in AD/HD that are eliminated by Methylphenadite treatment. *Journal of Abnormal Psychology, 114(2)*, 197-215.

Ridderinkhof, K. R., van den Wildenberg, W. P. M., Wijnen, J., & Burle, B. (2004). Response inhibition in conflict tasks is revealed in delta plots. In M. Posner (Ed.), *Cognitive Neuroscience of Attention (p. 369-377).* NY: Guilford Press.

Rommelse, N. N. J., Franke, B., Geurts, H. M., Hartman, C.-A., & Buitelaar, J. K. (2010). Shared heritability of attention-deficit/hyperactivity disorder and autism spectrum disorder. *European Child and Adolescent Psychiatry, 19*, 281-295.

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.

Simon, J. R. (1970). Stereotypic reactions in information processing. In: L. E. Smith (ed.), *Psychology of motor learning*. Chicago, IL: The Athletic Institute.

Sinzig, J., Morsch, D., Bruning, N., Schmidt, M. H., & Lehmkuhl, G. (2008). Inhibition, flexibility, working memory and planning in autism spectrum disorder with and without comorbid ADHD-symptoms. *Child and Adolescent Psychiatry and Mental Health, 2(4).*Retrieved April 2, 2012, from http://www.capmh.com/content/2/1/4

Speckman, P. L., Rouder, J. N., Morey, R. D., & Pratte, M. S. (2008). Delta plots and coherent distribution orienting. *The American Statistican, 62(3)*, 262-266.

Stins, J. F., Polderman, J. C. T., Boomsma, D. I., & de Geus, E. J. C. (2007). Conditional accuracy in response interference tasks: Evidence from the Eriksen flanker task and the spatial conflict task. *Advances in Cognitive Psychology, 3(3),* 409–417.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, *18*, 643–662.

Stürmer, B., & Leuthold, H. (2003). Control over response priming in visuomotor processing: A lateralized event-related potential study. *Experimental Brain Research, 153*, 35-44.

Stürmer, B., Leuthold, H., Soetens, E., Schrö ter, H., & Sommer, W. (2002). Control over location-based priming in the Simon task: Behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance, 28(6),* 1345-1363.

Thomas, M. S. C., Annaz, D., Ansari, D., Serif, G., Jarrold, C., & Karmiloff-Smith, A. (2009). Using developmental trajectories to understand developmental disorders. *Journal of Speech, Language, and Hearing Research*, *52*, 336-358.

Verté, S., Geurts, H. M., Roeryers, H., Oosterlaan, J., & Sergeant, J. A. (2006). Executive functioning in children with an autism spectrum disorder: Can we differentiate within the spectrum? *Journal of Autism and Developmental Disorders, 36(3*), 351-372.

Wakabayashi, A., Baron-Cohen, S., Wheelwright, S., & Tojo, Y. (2006). The Autism-Spectrum Quotient in Japan: Cross-cultural comparison. *Journal of Autism and Developmental Disorders, 36(2),* 263-70.

Wechsler, D. (2005). *Wechsler Adult Intelligence Scale – III NL herziene technische handleiding* (Revised technical manual). Amsterdam: Harcourt Test Publishers.

Williams, D., & Happé, F. (2010). Representing intentions in self and other: studies of autism and typical development. *Developmental Science, 13(2),* 307–319.

Woodbury-Smith, M. R., Robinson, J., Wheelwright, S., & Baron-Cohen, S. (2005). Screening adults for Asperger syndrome using the AQ: A preliminary study of its diagnostic validity in clinical practice. *Journal of Autism and Developmental Disorders, 35,* 331-335.

Appendix

Table 1

Predictive Value of Hyperactivity-Impulsivity Features on Reaction Time Differences per Bin

	Predictor		
	Hyperactivity-Impulsivity		
Dependent measure	*b*	*ß*	R^2
RT difference bin 1	-.76*	-.42*	.18*
RT difference bin 2	-1.30**	-.46**	.21**
RT difference bin 3	-1.43**	-.46**	.22**
RT difference bin 4	-1.34*	-.39*	.15*
RT difference bin 5	-.84	-.18	.03

Note. *p < .05. **p < .01.

	Nederlandse versie van de Autism Quotient vragenlijst Hoekstra, Bartels, Cath, & Boomsma (2008)	Geheel mee eens	Beetje mee eens	Beetje mee oneens	Geheel oneens
1.	Ik heb er een voorkeur voor om dingen samen met anderen te doen in plaats van alleen.	○	○	○	○
2.	Ik heb er een voorkeur voor om dingen steeds weer op dezelfde manier te doen	○	○	○	○
3.	Als ik mij iets probeer voor te stellen vind ik het erg makkelijk om mij een beeld voor de geest te halen.	○	○	○	○
4.	Ik ben vaak zo geobsedeerd door iets dat ik andere dingen uit het oog verlies.	○	○	○	○
5.	Ik hoor vaak kleine geluidjes als anderen niets horen.	○	○	○	○
6.	Nummerborden van auto's of andere informatie-reeksen vallen mij vaak op.	○	○	○	○
7.	Mensen zeggen regelmatig tegen me dat ik iets onbeleefds heb gezegd, terwijl ik het wel beleefd vond.	○	○	○	○
8.	Als ik een verhaal lees, kan ik me makkelijk voorstellen hoe de personages er uit zouden kunnen zien.	○	○	○	○
9.	Datums fascineren mij.	○	○	○	○
10.	In een groep kan ik makkelijk verschillende gesprekken tegelijk volgen.	○	○	○	○
11.	Sociaal contact gaat mij makkelijk af.	○	○	○	○
12.	Mij vallen details op die anderen over het hoofd zien.	○	○	○	○
13.	Ik ga liever naar de bibliotheek dan naar een feestje.	○	○	○	○
14.	Het bedenken van verhalen gaat mij makkelijk af.	○	○	○	○
15.	Ik voel me meer aangetrokken tot mensen dan tot voorwerpen.	○	○	○	○
16.	Ik heb meestal een diepgaande interesse in dingen, als ik me er niet mee bezig kan houden raak ik van streek.	○	○	○	○
17.	Ik vind sociaal gebabbel leuk.	○	○	○	○
18.	Als ik aan het woord ben krijgen anderen er geen woord tussen.	○	○	○	○
19.	Getallen fascineren mij.	○	○	○	○
20.	Als ik een verhaal aan het lezen ben, vind ik het moeilijk om te achterhalen waarom de personages iets doen.	○	○	○	○
21.	Het lezen van fictie vind ik niet zo interessant.	○	○	○	○
22.	Nieuwe vrienden maken vind ik moeilijk.	○	○	○	○
23.	Ik zie overal patronen in.	○	○	○	○
24.	Ik bezoek liever een theather dan een museum.	○	○	○	○

25.	Ik vind het niet erg als mijn dagelijkse routine wordt verstoord.	○	○	○	○
26.	Ik weet vaak niet hoe ik een gesprek gaande moet houden.	○	○	○	○
27.	Ik vind het makkelijk om 'tussen de regels door te lezen' als iemand tegen me praat.	○	○	○	○
28.	Ik richt mij meer op het totaalplaatje dan op de details.	○	○	○	○
29.	Ik ben slecht in het onthouden van telefoonnummers.	○	○	○	○
30.	Kleine veranderingen in situaties of in iemands uiterlijk vallen me vaak niet op.	○	○	○	○
31.	Ik merk het als mensen die naar me luisteren zich gaan vervelen.	○	○	○	○
32.	Meerdere dingen tegelijk doen gaat me makkelijk af.	○	○	○	○
33.	Tijdens een telefoongesprek weet ik niet wanneer het mijn beurt is.	○	○	○	○
34.	Ik houd er van om dingen spontaan te doen.	○	○	○	○
35.	Ik ben vaak de laatste die een grap begrijpt.	○	○	○	○
36.	Door naar iemands gezicht te kijken weet ik wat iemand denkt of voelt.	○	○	○	○
37.	Als ik onderbroken word, kan ik makkelijk verder gaan waar ik gebleven was.	○	○	○	○
38.	Ik ben goed in sociaal gebabbel.	○	○	○	○
39.	Ik krijg vaak te horen dat ik maar door blijf gaan over hetzelfde onderwerp.	○	○	○	○
40.	Toen ik jong was, vond ik het erg leuk om met andere kinderen spelletjes te spelen waarbij je moet doen alsof.	○	○	○	○
41.	Ik verzamel graag informatie over specifieke onderwerpen (bijvoorbeeld automerken, vogels, treinen, planten).	○	○	○	○
42.	Ik vind het moeilijk om mezelf in te leven in iemand anders.	○	○	○	○
43.	Ik vind het prettig om al mijn activiteiten nauwkeurig te plannen.	○	○	○	○
44.	Ik hou van sociale gelegenheden.	○	○	○	○
45.	Ik vind het moeilijk om er achter te komen wat mensen willen.	○	○	○	○
46.	Nieuwe situaties maken mij nerveus.	○	○	○	○
47.	Ik vind het leuk om nieuwe mensen te ontmoeten.	○	○	○	○
48.	Ik ben een goede diplomaat.	○	○	○	○
49.	Ik ben niet zo best in het onthouden van verjaardagen.	○	○	○	○
50.	Ik vind het erg makkelijk om spelletjes met kinderen te spelen waarin je moet doen alsof.	○	○	○	○

Zelf-rapportage vragenlijst over aandachtsproblemen en hyperactiviteit voor volwassenheid
Kooij & Buitelaar (1997)

Naam: …… Patiëntnr……
Geboortedatum: ……/ ……/ …….. Datum: ……/ ……./ …….

Omcirkel het getal dat het beste uw gedrag van de afgelopen zes maanden beschrijft.

Steeds één score aangeven (0, 1, 2 óf 3).
0 = nooit of zelden 1 = soms 2 = vaak 3 = erg vaak

1. Ik let onvoldoende op details bij mijn werk.	0 1 2 3
2. Wanneer ik zit, friemel ik met mijn handen of voeten.	0 1 2 3
3. Ik maak slordige fouten in mijn werk.	0 1 2 3
4. Ik zit te wiebelen en te draaien in mijn stoel.	0 1 2 3
5. Wanneer ik met iets bezig ben, kan ik er met mijn aandacht slecht bij blijven.	0 1 2 3
6. Ik sta snel op van mijn stoel in situaties waarin verwacht wordt dat ik netjes blijf zitten.	0 1 2 3
7. Ik luister slecht wanneer anderen iets tegen mij zeggen.	0 1 2 3
8. Ik voel me rusteloos.	0 1 2 3
9. Ik verveel me snel.	0 1 2 3
10. Ik heb moeite aanwijzingen op te volgen.	0 1 2 3
11. Karweitjes of werk waar ik aan begin, maak ik niet af.	0 1 2 3
12. Ik kan me moeilijk ontspannen in mijn vrije tijd.	0 1 2 3
13. In mijn vakantie of vrije tijd zoek ik een omgeving met drukte en lawaai.	0 1 2 3
14. Ik kan mijn bezigheden of taken moeilijk organiseren.	0 1 2 3
15. Ik ben voortdurend ‘in de weer’, alsof ik ‘door een motor word aangedreven’.	0 1 2 3
16. Ik probeer onder bezigheden uit te komen waarop ik me langere tijd moet concentreren.	0 1 2 3
17. Ik praat aan één stuk door.	0 1 2 3
18. Ik raak dingen kwijt die ik nodig heb voor taken of bezigheden.	0 1 2 3
19. Ik geef antwoord voordat vragen zijn afgemaakt.	0 1 2 3
20. Ik ben snel afgeleid.	0 1 2 3
21. Ik vind het moeilijk op mijn beurt te wachten.	0 1 2 3
22. Ik ben vergeetachtig bij alledaagse bezigheden.	0 1 2 3
23. Ik onderbreek anderen of val ze in de rede.	0 1 2 3