Acta Psychologica 143 (2013) 71–78

Contents lists available at SciVerse ScienceDirect

Acta Psychologica

journal homepage: www.elsevier.com/ locate/actpsy

Interference control in adult ADHD: No evidence for interference control deficits if response speed is controlled by delta plots

Alexander Soutschek [a,*], Wolfgang Schwarzkopf [a], Kathrin Finke [a], Kristina Hennig-Fast [b], Hermann J. Müller [a], Michael Riedel [b], Hans-Jürgen Möller [b], Christian Sorg [c], Torsten Schubert [a,d]

[a] General and Experimental Psychology, Department of Psychology, Ludwig-Maximilians-Universität München, Munich, Germany
[b] Department of Psychiatry and Psychotherapy, Ludwig-Maximilians-Universität München, Munich, Germany
[c] Department of Psychiatry, Klinikum rechts der Isar, Technische Universität München, Munich, Germany
[d] Department of Psychology, Humboldt-Universität, Berlin, Germany

ARTICLE INFO

Article history:
Received 22 August 2012
Received in revised form 20 February 2013
Accepted 27 February 2013
Available online 27 March 2013

PsycINFO classification:
2346

Keywords:
Adult ADHD
Interference control
Stroop task
Delta plots

ABSTRACT

Several theoretical accounts assume that interference control deficits belong to the core symptoms of adult ADHD. However, findings of increased interference effects in adult ADHD patients compared with healthy adults may be confounded with the simultaneous finding of generally slower responses in the patient group. The current study compared the magnitude of the interference effect in the Stroop task between a group of adults with ADHD and a healthy adult control group in a procedure that accounted for differences in overall response speed by using delta plots. The amount of interference did not differ between patient and control group at comparable reaction time levels. These results challenge the conclusions of the previous studies, in that they indicate that interference control is not impaired in adult ADHD.

1. Introduction

Attention deficit hyperactivity disorder (ADHD) in adults represents a disorder which has been related to deficits in various cognitive domains, including attention and executive functions (e.g., Barkley, 1997; Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Faraone et al., 2000; Nigg, 2005). Several theoretical accounts consider impaired interference control to be one of the core deficits in adult ADHD patients (e.g., Barkley, 1997; Nigg, 2005). One important source of evidence is the finding of enhanced interference effects in the Stroop task. In this task, participants have to respond to the ink color of a color word and ignore its semantic meaning; the task-irrelevant semantics can either be congruent (e.g., "RED" written in red) or incongruent (e.g., "RED" written in blue) with the relevant ink color (Stroop, 1935). The reaction time (RT) difference between incongruent and congruent color-word combinations is referred to as "Stroop effect" and represents a widely used measure of resistance to interference. Increased interference effects displayed by adult ADHD patients in the color-word Stroop task (King, Colla, Brass, Heuser, & von Cramon, 2007; Taylor & Miller, 1997; Walker, Shores, Trollor, Lee, & Sachdev, 2000) and in other interference paradigms such as the counting Stroop (Bush et al., 1999) or the Flanker task (Lundervold et al., 2011) are commonly interpreted as an indicator of impaired cognitive control (for a review, see Boonstra et al., 2005), even though they are not always replicated (e.g., Banich et al., 2009; Marchetta, Hurks, Krabbendam, & Jolles, 2008).

Critically, however, the majority of studies that actually replicated larger interference effects also found adult ADHD patients to show slower overall mean RTs than healthy adults (Bush et al., 1999; King et al., 2007; Lundervold et al., 2011; Walker et al., 2000), whereas only one study reported a larger interference effect despite the absence of general performance slowing (Taylor & Miller, 1997). Importantly, the Stroop effect is known to be generally increased with longer overall RT levels (Bub, Masson, & Lalonde, 2006; Pratte, Rouder, Morey, & Feng, 2010). Therefore, larger interference effects in ADHD patients may not reflect impaired interference control, but rather be a by-product of their slowed overall RT performance (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Sergeant, 2005). In line with this possibility, one of the rare studies in which an ADHD and a healthy control group showed a comparable general RT level failed to reveal a significant difference between the interference effects in these groups (Banich et al., 2009). A similar discussion is on-going in the literature about executive function impairments in child ADHD: In particular, some studies reporting deficits in interference control (Homack & Riccio, 2004; Mullane, Corkum, Klein, McLaughlin, & Lawrence, 2011) and motor

* Corresponding author at: Department of Psychology, Ludwig-Maximilians-Universität München, Leopoldstr., 13, 80802 Munich, Germany. Tel.: +49 89 2180 16587.
E-mail address: Alexander.Soutschek@psy.lmu.de (A. Soutschek).

0001-6918/$ – see front matter © 2013 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.actpsy.2013.02.013

inhibition (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005) found that the general performance level, too, was impaired in children with ADHD compared to controls. In contrast, a study in which interference effects in an auditory Stroop task and a Simon task did not differ between a group of children with ADHD and a control group found only marginally significant or non-significant differences in general performance speed, respectively (Van Mourik et al., 2009). Thus, in both adult and child ADHD, interference control deficits appear to be correlated with general performance speed.

Clarification of whether specific interference deficits or more generalized RT slowing underlie the performance deficits of ADHD adults in the Stroop task is of particular significance from a theoretical point of view because it permits the notion of a cognitive control deficit in ADHD to be evaluated against alternative views. The notion of cognitive control impairments (Barkley, 1997; Nigg, 2005) would predict that, in adult ADHD patients, specific interference effects manifest over and above those explicable solely by overall RT slowing compared to healthy subjects. By contrast, if changes in RT behavior alone could explain the changes in Stroop task performance, this would suggest that the underlying impairments are not related to cognitive interference control in particular, but to more general deficits. Potential candidates that have been proposed in adult ADHD and that may play a role in any RT-based task are deficits in arousal adjustment (Sergeant, 2005), in response selection (Barkley, 1997; Castellanos et al., 2006), and/or in working memory processes (Finke et al., 2011). In fact, some accounts even consider the cognitive deficits in ADHD to be only "by-products" of underlying motivational or energetic dysfunctions (see Sonuga-Barke, Wiersema, van der Meere, & Roeyers, 2010, for a review).

Although the review of Boonstra et al. (2005) surmised that the increased Stroop interference effect in adult ADHD may be confounded by slower overall RTs because interference control deficits in ADHD appear to be positively correlated with slower response speed, the mere finding of a correlation between general RT speed and Stroop interference effects does not conclusively show that the slower mean RTs are the *cause* of the larger Stroop effect. For such a conclusion to be valid, it would be necessary to demonstrate that the amount of interference does not differ at equal RT levels between ADHD patients and healthy control subjects. Given this, the present study was designed to investigate whether adult ADHD patients would show a larger Stroop interference effect than demographically matched healthy control subjects when comparing performance at similar RT levels. If ADHD patients do show a larger effect, then this would provide further, conclusive support for the assumption of cognitive control deficits in ADHD. If not, this would suggest that larger Stroop effects in the ADHD group are brought about by the generally slower mean RTs, rather than by cognitive control deficits.

A methodological tool that permits the magnitude of interference effects to be examined as a function of response speed is provided by delta plots. For the construction of delta plots, the interference effect is calculated separately for different percentiles (e.g., deciles) of the RT distribution of a given participant and plotted against the mean RTs of congruent and incongruent trials for the corresponding percentile (de Jong, Liang, & Lauber, 1994). The amount of interference is then taken as dependent variable (y-axis) and the mean RTs for the corresponding percentile as independent variable (x-axis). Previous studies investigating the time course of the Stroop effect with delta plots found that the Stroop effect increases with the RT level, that is: it is minimal for the fastest and maximal for the slowest percentiles within a subject (Bub et al., 2006; Pratte et al., 2010). This relationship has been explained within the framework of information accumulation models, which assume that a response decision is made when the accumulated information determining the response has reached a certain threshold (e.g., Ratcliff & Rouder, 1998; Usher & McClelland, 2001). Congruent stimuli may engender a higher accumulation rate (i.e., reach the response decision threshold faster) than incongruent stimuli because target and distractor information are related to the same response alternative. This in turn results in a larger difference between congruent and incongruent stimuli with increasing accumulation time, or, in other words, with increasing processing time needed to reach the decision threshold (Pratte et al., 2010). Importantly, this positive relationship between the magnitude of the Stroop effect and processing time implies that the increased Stroop effect in ADHD patients compared to controls may be caused by the generally increased RT level, rather than by specific interference control deficits in ADHD. Note that the positive delta plot slope (i.e., larger interference effects with increasing mean RT) in the Stroop task deviates from the shape of delta plots in some other interference paradigms like the Simon task, in which smaller congruency effects at the slowest compared to faster RT levels are found (Pratte et al., 2010; Ridderinkhof, 2002). These different delta plot slopes may be attributable to the different types of conflict and conflict resolution mechanisms engaged in the Stroop and the Simon task (Egner, Delano, & Hirsch, 2007; Soutschek, Müller & Schubert, 2013).

Based on these assumptions about the size of the Stroop effect at different response time levels, the present study aimed at comparing the Stroop effects between ADHD patients and healthy controls under conditions of controlled response time levels between these groups. Importantly, the delta plot technique allowed us to investigate the amount of the Stroop effect in the two experimental groups at comparable RT levels. If we do not find any group differences at comparable RT levels, then this would indicate that adult ADHD may not be related to interference control deficits.

In addition to investigating the Stroop effect in a given (or current) trial, we also examined cognitive control effects manifesting across consecutive trials. Although previous studies investigating interference control in ADHD have mainly focused on the Stroop effect as an indicator of interference control, the so-called "conflict adaptation effect" is often regarded as a more direct measure of cognitive control processes. This effect refers to the observation that the Stroop interference is reduced in the current (incongruent) trial episode n if this trial is preceded by an incongruent, versus a congruent, episode on trial $n - 1$. The standard explanation for this effect assumes that the detection of a conflict in trial $n - 1$ leads to the enhanced activation of cognitive control, as a result of which a conflict in the subsequent trial n is resolved more efficiently (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Egner, 2007; Kerns et al., 2004). The conflict adaptation effect therefore specifically reflects the reduction of interference resulting from reactive adjustments of cognitive control, rather than indicating only the amount of interference per se (Egner, 2007). To our knowledge, no study thus far has examined the conflict adaptation effect in adult ADHD. We examined the conflict adaptation effect to complement our comprehensive analysis of potential Stroop task indicators of interference control deficits in adult ADHD.

2. Methods

2.1. Participants

Twenty-one non-medicated adult ADHD patients (mean age = 34.14 years, age range 21–54 years, 8 female) were recruited at the Department of Psychiatry of the Ludwig-Maximilians-University Munich. They were tested a few days after the initial diagnostic assessment, which was carried out at a specialized adult ADHD outpatient clinic.

The diagnostic procedure necessary for including a patient in this study comprised different steps: Two psychiatric interviews (according to *DSM-IV*) were carried out independently by two psychiatrists of the ADHD outpatient clinic. Using a conservative criterion, patients were only included when both psychiatrists rated them as ADHD patients. Collateral information from different sources (e.g., school reports and third-party 'informants' such as parents or siblings) was obtained by a psychologist trained in ADHD assessment, in order to confirm childhood onset according to the obligatory *DSM-IV* symptoms for childhood ADHD. Patients were only included if descriptions of the respective symptoms were listed in the first elementary school reports (obtained

Table 1

	ADHD ($n = 21$)	Control ($n = 21$)	t
Sex (female/male)	8/13	8/13	
Age	34.14 (10.46) 21–54	32.90 (9.27) 22–50	.41
School (years)	11.86 (1.59) 9–13	11.71 (1.71) 9–13	.28
IQ (MWT-B)	107.33 (10.34) 93–130	113.29 (12.91) 94–136	1.65
CAARS-S subscales			
A	72.29 (8.16) 57–85	49.19 (5.85) 37–59	10.54*
B	63.52 (9.19) 46–84	47.29 (6.59) 34–58	6.58*
C	68.38 (10.59) 41–88	47.90 (6.17) 35–59	7.66*
D	64.86 (8.83) 42–79	44.90 (7.16) 34–60	8.04*
E	82.67 (7.86) 65–90	50.29 (6.22) 38–59	14.80*
F	68.81 (11.27) 48–86	50.14 (7.01) 39–59	6.44*
G	80.81 (9.55) 57–90	50.33 (6.81) 36–58	11.91*
H	74.10 (7.72) 60–88	50.10 (7.92) 34–59	9.45*
WURS	57.57 (13.87) 38–85	20.10 (12.46) 0–42	9.21*

Group demographics: Sex distribution, mean, SD, and range of age, attended school years, IQ. Mean T-values, SD, and range of subjective current symptoms and retrospective childhood symptoms for the ADHD group.

Abbreviations: School: Duration of education (in years); MWT-B: German Multiple-Choice Vocabulary Test (Lehrl et al., 1995); CAARS-S: Connors Adult ADHD Rating Scale Self-Rating (Conners Adult ADHD Rating Scales Self Report, CAARS; Conners et al., 2002); CAARS-subscales: A — inattention/memory problems; B — hyperactivity/restlessness; C — impulsivity/emotional instability; D — problems with self-concept; E — inattentive symptoms according to DSM-IV; F — hyperactive-impulsive symptoms according to DSM-IV; G — total ADHD symptoms according to DSM-IV; H — ADHD Index. WURS: Wender Utah Rating Scale (Wender Utah Rating Scale, WURS; Ward et al., 1993); ADHD: ADHD patients; Con: control participants.

* $p < .001$.

at an age ≤ 7 years) and for a longer-term period in the following reports. In Germany, elementary school reports contain comprehensive descriptions of learning performance (e.g., participation in lessons, diligence with homework, accuracy in written reports), social behavior (e.g., impulsivity and aggression), and daily structure (e.g., forgetfulness and daydreaming), differentiated according to cognition, emotion, and motor behavior. Furthermore, prior psychiatric diagnoses, or third-party 'informants' (siblings), had to confirm that these symptoms were also displayed at home and that there had been no alternative suspected diagnosis. Two patients had already been diagnosed with ADHD in childhood, two had received ADHD medication during childhood (but not in adulthood). Finally, in an assessment of current (Conners Adult ADHD Rating Scales Self Report, CAARS; Conners, Erhardt, & Sparrow, 2002) and retrospective childhood symptoms (Wender Utah Rating Scale, WURS; Ward, Wender, & Reimherr, 1993), self-reports had to indicate ADHD since childhood.

Average ADHD symptom ratings in the ADHD patients (see Table 1) indicated severe subjective current impairments (all T-values > 60) and retrospective childhood ADHD symptoms (all ADHD patient values are above the cut-off value, i.e., ≥46; Ward et al., 1993). In accordance with previous reports on symptoms in adulthood (Biederman, 2005), inattentiveness ratings were especially pronounced (T-values > 70).

German versions of the Minnesota Multiphasic Personality Inventory (MMPI-2; Hathaway, McKinley, & Engel, 2000) and Personality Assessment Inventory (Groves & Engel, 2007) were used to exclude patients with other mental and personality disorders. Furthermore, patients with either prior or comorbid neurological disorders, bipolar disorder, schizophrenia, or other psychotic disorders, substance abuse or addiction other than nicotine within the last three months, or with an IQ below 85 were excluded.

Seven patients had a history of previous cannabis use and two were heavy smokers. Patients whose clinical picture was dominated by depressive symptoms were excluded. However, since depression and anxiety are frequent comorbid disorders in adult ADHD samples (Sprafkin, Gadow, Weiss, Schneider, & Nolan, 2007), secondary diagnoses (in addition to ADHD) had been given to six included patients with recurrent moderate depression (ICD-10 F 33.1; World Health Organization, 1992). Five of these patients took antidepressive medication, but none of them suffered from an acute major depression. Patients and control participants were asked to abstain from nicotine and caffeine at least 1 h prior to the application of the Stroop task. This was meant to ensure that, on the one hand, ADHD patients' performance could not profit from recent nicotine consumption and, on the other hand, that the results of heavy smokers were unlikely to be compromised by withdrawal effects (Heishman, Kleykamp, & Singleton, 2010).

Twenty-one participants with neither neurological nor psychiatric (inclusive drug addiction) history served as control group (mean age = 32.90 years, age range 22–50 years, 8 female) and were paid 8 euro/h for their participation. They were assessed with the Stroop task as well as the CAARS, the WURS, and the WST questionnaire. Age, gender, IQ (German Multiple-Choice Vocabulary Test; Lehrl, Triebig, & Fischer, 1995), and educational level were matched and did not differ significantly between the patient and control groups (all $ps > .1$). In contrast, subjective ADHD symptoms (CAARS — T-value and WURS scores) were significantly higher in the ADHD group than in the control group, all $ps < .001$ (see also Table 1). Informed consent according to the Declaration of Helsinki II was obtained from all participants. All had normal or corrected-to-normal vision.

2.2. Stimuli and apparatus

The experiment was run in a dimly lit sound-proof experimental cabin. The participants sat at a distance of approximately 50 cm from a 17 in. monitor on which stimuli were presented, controlled by Experimental Run Time System (ERTS; Berisoft) run on a standard PC. All stimuli were presented against a black background. We employed a color-word variant of the Stroop paradigm in which we presented three different color words ("BLAU", "ROT", and "GRÜN"; German for blue, red, and green) in blue, red, or green ink, resulting in either congruent (e.g., "BLAU" in blue ink) or incongruent (e.g., "BLAU" in red ink) color-word combinations.

2.3. Task and procedure

The task was to respond to the ink color of the presented words and ignore the semantic meaning. We instructed participants to respond by pressing the keys V (for blue words), B (for red words), and N (for green words) on a QWERTZ keyboard, using their right-hand index, middle, and ring finger, respectively.

Each trial started with the presentation of a white fixation cross on a black screen for 1500 ms, then the color word was presented for 2000 ms. Participants had to respond within stimulus presentation time. The next trial started immediately after the response. A block contained 66 congruent and 24 incongruent trials, which were presented in randomized order. We used a higher number of congruent than incongruent trials because this procedure has been shown to reliably produce strong interference effects (Kerns et al., 2004). We administered three blocks, resulting in a total of 270 trials.

2.4. Statistical analysis

We analyzed RTs and error rates of the Stroop task data. We removed all trials that contained either a repetition of the color or the word from the data set to control for any influence such stimulus repetitions might have on the conflict adaptation effect (Hommel, Proctor, & Vu, 2004; Kerns et al., 2004; Mayr, Awh, & Laurey, 2003). In addition, we conducted further analyses to control for potential negative priming effects (see below). For the RT analysis, we also removed all trials including or following an error.

To calculate the conflict adaptation effect, we differentiated between the effects of current trial congruency (denoted by upper-case *C*[congruent] vs. *I*[incongruent]) and previous trial congruency (denoted by lower-case *c* vs. *i*). We defined the conflict adaptation effect as the difference between the Stroop effects after previously congruent versus previously incongruent trials ($(cI - cC) - (iI - iC)$; Kerns et al., 2004). We tested the occurrence of conflict adaptation effects in the ADHD and the control group with one-tailed *t*-tests because we expected the congruency effect to be smaller after previously incongruent compared to after congruent trials.

The delta plot analysis was conducted as follows: We rank-ordered all RTs of each participant separately for congruent and incongruent trials and calculated the values for the 10th, 20th, 30th, 40th, 50th, 60th, 70th, 80th, and 90th percentiles (deciles). Next, we calculated the Stroop effect for each decile. The Stroop effect in the first decile, for example, was calculated by subtracting the RT for the first decile in congruent trials from the RT for the first decile in incongruent trials. In order to construct delta plots, we plotted the amount of the Stroop effect for each decile against the mean response speed for that decile, that is, the mean RT of congruent and incongruent trials.

In addition, we also computed a distributional analysis for the error congruency effect. For that purpose, we divided the RT distribution into five bins of equal size (quintiles) and then calculated the congruency effect in the error rates separately in the different RT bins (Ridderinkhof, 2002; Ridderinkhof, Scheres, Oosterlaan & Sergeant, 2005). In detail, we first subdivided the RTs in both correct and incorrect response trials into five bins of equal size, separately for congruent and incongruent trials. Then, we calculated the error congruency effect in each bin by subtracting the error rate in congruent trials from the error rate in incongruent trials in the corresponding bins.

3. Results

3.1. RT analysis

We carried out a 2 × 2 × 2 mixed-design analysis of variance (ANOVA) with the between-subject factor Group (ADHD vs. control) and the within-subject factor Current trial congruency (C vs. I) and Previous trial congruency (c vs. i). Due to the exclusion of stimulus repetition trials as well as error and post-error trials, a mean total trial number of 170 per participant was entered into the RT analysis. Results are illustrated in Fig. 1. This ANOVA yielded a significant main effect of group, with mean RTs being slower overall in the ADHD group (793 ms) than in the control group (597 ms), $F(1, 40) = 20.71$, $p < .001$, $\eta_p^2 = .341$. The main effect of current trial congruency was also significant, due to responses being slower overall in incongruent trials (857 ms) compared to congruent trials (644 ms), $F(1, 40) = 111.75$, $p < .001$, $\eta_p^2 = .736$ — that is, there was a Stroop effect of over 200 ms. Moreover, the Group × Current trial congruency interaction was significant, due to the ADHD group exhibiting a larger Stroop effect than the control group (258 ms vs. 168 ms), $F(1, 40) = 4.65$, $p < .05$, $\eta_p^2 = .104$. The effect of the current trial congruency interacted with the previous trial congruency, $F(1, 40) = 6.35$, $p < .05$, $\eta_p^2 = .137$, which is indicative of a conflict adaptation effect. Importantly, the conflict adaptation effect did not differ in magnitude between the ADHD and the control group, as evidenced by the non-significant Current trial congruency × Previous trial congruency × Group interaction, $F(1, 40) = .19$, $p = .67$, $\eta_p^2 = .005$. That is, there was a significant conflict adaptation effect both in the ADHD group $(cI - cC) - (iI - iC) = 63$ ms, $t(20) = 1.72$, $p < .05$, one-tailed, and in the control group $(cI - cC) - (iI - iC) = 44$ ms, $t(20) = 2.03$, $p < .05$, one-tailed, with no reliable difference in the effect magnitude between the two groups, $t(40) < 1$, $p > .67$.

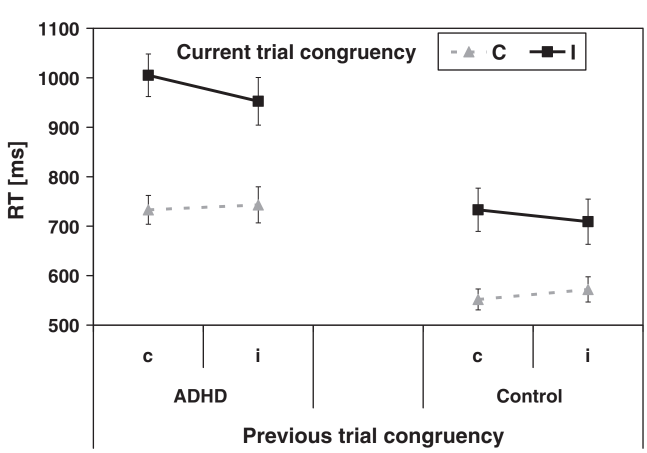

Fig. 1. Mean reaction times (RTs) in the Stroop task for the ADHD and control groups. Error bars indicate the standard error of the mean (C/c, congruent; I/i, incongruent).

For the analyses reported above, we had excluded all trials in which either the color or the word feature had been repeated (within-dimension repetitions) in order to control for potential feature repetition effects on conflict adaptation. Besides within-dimension repetitions, however, conflict adaptation might also be affected by negative priming, i.e. when the target feature becomes the distractor feature in the following trial, or vice versa (across-dimension repetitions). Note that we could not simultaneously control for both within-dimension repetitions and negative priming because this would have resulted in an exclusion of all iI trials in the variant of the Stroop paradigm we used. Therefore, we computed a separate analysis in which we excluded only across-dimension but not within-dimension repetition trials (see Soutschek, Strobach & Schubert, 2012): We found significant conflict adaptation effects both in the ADHD $(cI - cC) - (iI - iC) = 203$ ms, $t(20) = 4.22$, $p < .001$, and the control group $(cI - cC) - (iI - iC) = 67$ ms, $t(20) = 2.77$, $p < .05$, whereas conflict adaptation was more pronounced in the ADHD than in the control group, $t(40) = 2.52$, $p < .05$. The surprising finding of a larger conflict adaptation effect in the ADHD than in the control group may indicate that the benefit from within-dimension repetitions (which were not excluded in this analysis) was more pronounced in the patient than in the control group. Importantly, however, this finding is not compatible with the idea of interference control deficits in adult ADHD. In addition, we also tested whether conflict adaptation occurred when no repetition trials were excluded at all. Again, significant conflict adaptation effects occurred both in the ADHD, $(cI - cC) - (iI - iC) = 92$ ms, $t(20) = 3.11$, $p < .01$, and the control group $(cI - cC) - (iI - iC) = 41$ ms, $t(20) = 2.61$, $p < .05$, whereas we found no significant difference between ADHD and control group, $t(40) = 1.53$, $p > .13$. Taken together, the observed conflict adaptation pattern appears to be robust against the trial types included in the analysis.

3.2. RT delta plot analysis

In line with the previous studies, our analysis revealed a larger Stroop effect in the ADHD group than in the control group. However, the ADHD patients' performance was also characterized by slower mean RTs compared to the control group. As previous studies had shown that the size of the Stroop effect increases with slower responses (Pratte et al., 2010), the differences in Stroop interference between the ADHD and the control group may be attributable to the different RT levels in these groups. To test this hypothesis, we compared the Stroop effects in the ADHD and the control group by means of delta plots, which graph the size of the Stroop effect for different RT levels. To be able to compare similar RT levels between the ADHD and the control group, we calculated the mean Stroop effect for each decile and plotted it against the RT for the corresponding decile. This is illustrated in Fig. 2 in which the size of the Stroop effect (y-axis) for the decile of the two groups is plotted as a function of the mean RTs (x-axis) for the corresponding decile. As can be seen from Fig. 2, the whole distribution of the Stroop effect in the ADHD patient group is shifted to the up and to the right of the distribution of the control group, with the RT distribution being broader in the ADHD than in the control group due to some extremely slow responses occurring in the ADHD group (see also Leth-Steensen, Elbaz, & Douglas, 2000). This observation is corroborated by the results of a 2 (Group) × 9 (Decile) mixed-design ANOVA with the size of the Stroop effect as dependent variable. This analysis revealed a significant main effect of group, $F(1, 40) = 5.95$, $p < .05$, $\eta_p^2 = .129$, reflecting the generally larger Stroop effect in the ADHD group. Furthermore, the main effect of decile was significant, $F(8, 320) = 42.81$, $p < .001$, $\eta_p^2 = .517$, reflecting the fact that the Stroop effect was larger at slower, compared to faster, RTs in both groups. Importantly, the increase of the Stroop effect size with increasing RT levels did not differ between the ADHD and the control group, Group × Decile, $F(8, 320) < 1$, $p > .66$, $\eta_p^2 = .011$. Fig. 2 suggests that ADHD and control group show quite comparable amounts of Stroop interference at similar mean RTs. Importantly, the delta plot technique allowed testing this hypothesis in more detail by calculating post-hoc t-tests between the magnitudes of the Stroop effect in ADHD and control group at comparable RT levels. As it is illustrated by Fig. 2, the mean RTs of the patients in the first (552 ms), second (623 ms), third (677 ms), fifth (772 ms), and seventh deciles (920 ms ms) are comparable to the mean RTs of the controls in the third (536 ms), fifth (608 ms), seventh (694 ms), eighth (757 ms), and ninth deciles (891 ms), respectively (all *ps* > .42 between the corresponding deciles). Independent-samples *t*-tests validated that the size of the Stroop effect did not differ between ADHD and control group at these RT levels (i.e., first decile ADHD — third decile control: $t(40) < 1$, $p > .78$; second decile ADHD — fifth decile control: $t(40) < 1$, $p > .93$; third decile ADHD — seventh decile control: $t(40) < 1$, $p > .58$; fifth decile ADHD — eighth decile control: $t(40) < 1$, $p > .68$; seventh decile ADHD — ninth decile control: $t(40) < 1$, $p > .72$). In other words, ADHD and control group showed similar amounts of the Stroop effect at comparable RT levels.

In order to support the observation reported above which provided no evidence for interference control deficits in ADHD, we additionally compared the slopes of the delta plot curves between patient and control group. If ADHD patients suffered from specific interference control deficits, then the increase of the Stroop effect with slower response speed (that is, the slope of the delta plot curve) should be larger in the patient than in the control group. This is because the delta plot slope is thought to reflect the efficiency of interference control processes (Pratte et al., 2010). To test this prediction, we first computed the slopes of the individual delta plots by conducting a regression analysis for every single participant in which the magnitude of the interference effect and the mean RT in each decile were entered as criterion and predictor, respectively. The resulting beta weights of these regressions represented the delta plot slopes of the individual participants. In the next step, we calculated an independent-samples *t*-test to compare the delta plot slopes between ADHD patients and control subjects. Because the observed delta plot slopes did not significantly differ between the ADHD (mean slope = .49) and the control group (mean slope = .59), $t(40) = 1.03$, $p > .30$, the analysis of the delta plot slopes provided no evidence for interference control deficits in adult ADHD.

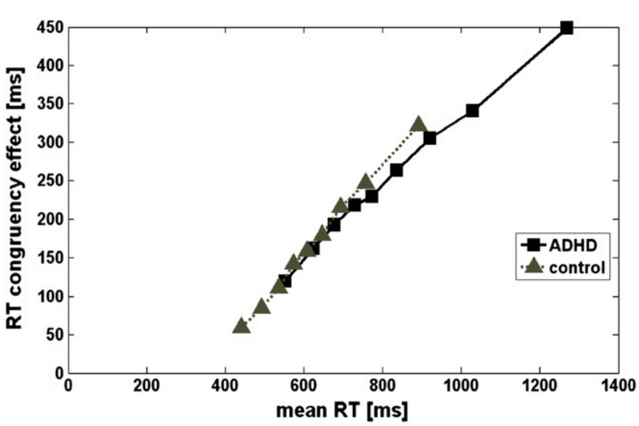

Fig. 2. Delta plot of the reaction time (RT) congruency effect in the ADHD and control groups.

3.3. Error analysis

We also analyzed error rates in a 2 × 2 × 2 mixed-design ANOVA including the same factors as in the RT analysis (for details, see Table 2). On average, 181 trials per participant were entered into the error analysis. ADHD patients tended to make more errors overall than healthy controls, $F(1, 40) = 3.45$, $p = .07$, $\eta_p^2 = .079$. The significant main effect of the Previous trial congruency indicated that error rates were larger after previously incongruent (6.4%) compared to the previously congruent trials (5.8%), $F(1, 40) = 7.14$, $p < .05$, $\eta_p^2 = .141$. In addition, there was a significant congruency effect, with more errors occurring in incongruent (11.6%) than in congruent trials (3.7%), $F(1, 40) = 25.72$, $p < .001$, $\eta_p^2 = .391$. This error congruency effect was modulated by the factor Group, $F(1, 40) = 5.73$, $p < .05$, $\eta_p^2 = .125$, reflecting a larger congruency effect in the ADHD group (11.2%) than in the control group (4.3%). Furthermore, there was a significant conflict adaptation effect in the error rates, $F(1, 40) = 5.69$, $p < .05$, $\eta_p^2 = .125$, indicating a reduced error congruency effect after incongruent (5.7%), compared to congruent (10.1%), trials. Again, the conflict adaptation effect did not differ in magnitude between the ADHD and the control group, $F(1, 40) = .78$, $p > .38$, $\eta_p^2 = .019$.

3.4. Distributional analysis of error congruency effects

We conducted a distributional analysis of the error congruency effect in order to examine the magnitude of the error congruency effect at different response time levels (see Fig. 3). For that purpose, we divided the RT distribution into five bins of equal size and calculated the error congruency effect in each RT bin separately for ADHD patients and controls. A 2 × 5 (Group × Bin) ANOVA revealed a tendency to a significant Group × Bin interaction, $F(4, 160) = 2.12$, $p < .08$, $\eta_p^2 = .052$, suggesting that the distributions of the error congruency effect differed between the ADHD and the control group. Comparing the congruency effects between these groups at each RT bin by independent-samples *t*-test, we found that ADHD patients showed a significantly larger error congruency effect than healthy controls only in the first bin (i.e., the fastest RTs), $t(40) = 2.62$, $p < .05$, whereas no significant difference occurred in all other bins, $ts < 1.03$, $ps > .30$. These findings indicate that ADHD patients showed a larger error congruency effect than healthy controls particularly in trials with very fast responses.

4. Discussion

In ADHD research, there is currently a debate as to whether or not adult ADHD patients suffer from deficits in interference control compared to healthy controls (e.g., Boonstra et al., 2005; Bush et al., 1999; King et al., 2007). Interestingly, the increased interference effect in the patient group seems to be correlated with slower overall response speed in this group (for an overview, see Boonstra et al., 2005). In line with these previous findings, the present study replicated both an

Table 2
Error rates in the Stroop task for ADHD and control group. Numbers in brackets denote the standard errors of mean (C/c, congruent; I/i, incongruent).

Previous trial congruency	C		I	
Current trial congruency	C	I	C	I
ADHD group	4.3 (1.0)	18.9 (3.0)	3.1 (1.0)	11.8 (3.2)
Control group	3.5 (1.1)	9.1 (1.7)	4.0 (1.6)	6.8 (2.2)

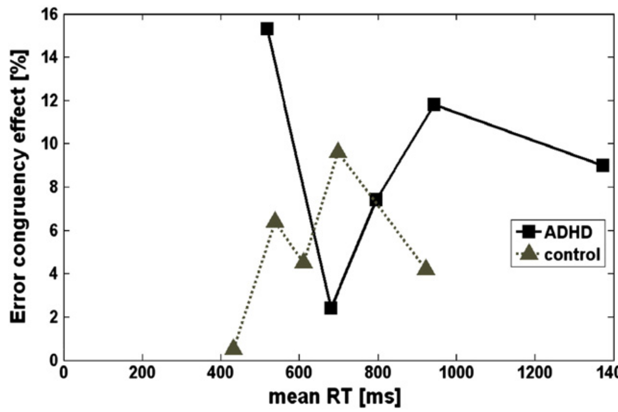

Fig. 3. Delta plot of the error rate congruency effect in the ADHD and control groups.

increased interference effect and slower RTs in the ADHD, compared to the control, group. Importantly, however, the larger Stroop effect disappeared when we compared performance between the ADHD and the control group at similar response speed levels. In particular, the delta plot analysis revealed that the Stroop effect did not differ in magnitude between ADHD patients and controls at comparable RT levels. Since the Stroop effect generally increases with slower mean RTs (Bub et al., 2006; Pratte et al., 2010), we conclude that the generally slower RT performance of the ADHD patients can (at least to a large extent) explain the larger mean Stroop effect in that group. In other words, there is no need to assume that interference control, too, is impaired in ADHD. If the ADHD patients had suffered from deficits in interference control, then this group should have shown a larger Stroop effect, compared to the control group, at comparable RT levels as well. Note that there is evidence that color perception may be impaired in ADHD (Banaschewski et al., 2006). However, although this impairment might have contributed to the slower mean RTs in the ADHD than in the control group, it seemed to have no impact on the Stroop effect itself because the Stroop effect did not differ between ADHD and control group at comparable RT levels.

The current results are in line with the findings of the previous studies that failed to find a larger Stroop effect in ADHD when mean RTs were comparable between the patient and control groups (Banich et al., 2009) or when response speed was controlled for (Boonstra et al., 2005). However, in contrast to the findings of a correlational relationship between the magnitude of the Stroop effect and the general performance level, we directly tested for Stroop effect differences at comparable RT levels by using delta plots. The findings of the current analysis suggest that the increased interference effect in adult ADHD reported in the previous studies may be attributable to the slower overall RTs (Bush et al., 1999; King et al., 2007; Lundervold et al., 2011). A testable hypothesis that directly follows from our conclusion is that ADHD patients should show larger effect sizes than healthy controls in all experimental paradigms with positive-going delta plots (e.g., for word frequency effects in lexical decision tasks; see Rouder, Yue, Speckman, Pratte, & Province, 2010), whereas effects with negative-going delta plots (such as the Simon effect) should be reduced in ADHD relative to control samples.

It is important to note that slower mean RTs can be accounted for larger interference effects in ADHD patients relative to controls only if the delta plot of the applied interference paradigm has a positive slope. While a positive delta plot slope is well established for the Stroop task, the distributional properties of other interference paradigms used in ADHD research such as the counting Stroop task (Bush et al., 1999) have – to the best of our knowledge – not yet been examined. Thus, one should be careful with generalizing the results of our study to interference paradigms with unknown distributional properties.

As a further indicator of unimpaired interference control in the Stroop task, we found that the conflict adaptation effect was not reduced in the ADHD compared to the control group. Since the conflict adaptation effect measures control-triggered adjustments in interference processing, it is assumed to represent a more direct indicator of cognitive control than the Stroop effect itself (Botvinick et al., 2001; Egner, 2007). ADHD patients even showed a larger conflict adaptation effect than controls when only across-dimension repetitions were excluded from the data set, suggesting that the facilitatory effect of stimulus priming (induced by within-dimension repetitions) may have been more pronounced in the ADHD than in the control group. Summarizing our findings for the Stroop effect and the conflict adaptation effect, the present results suggest that interference control may not be impaired in adult ADHD.

The fact that an increased interference effect in adult ADHD is not necessarily indicative of deficits in interference control, but may rather be related to a generally slower RT level, raises the question as to how the slower RT level of the patients can be explained. Currently, several different accounts are discussed as potential explanations for the general performance deficits in ADHD: According to the recent dual-process models, for example, dysfunctional bottom-up factors like alertness/arousal or motivational factors contribute to the behavioral impairments in ADHD (Nigg & Casey, 2005; Sonuga-Barke, Wiersema, van der Meere, & Roeyers, 2010). According to the state regulation deficit account of ADHD, for example, ADHD patients show deficits in the context-dependent attribution of cognitive resources to the motor preparation stage (Sergeant, 2005; van der Meere, 2005), which may result in prolonged overall processing times. Our finding that seeming interference control deficits may be explicable by a general RT slowing would be consistent with such accounts of ADHD according to which the observed cognitive impairments are only secondary deficits originating from more basic motivational or energetic impairments. However, deficits in working memory functions, too, are discussed as causes for the impaired performance level in adult ADHD (Finke et al., 2011). The current data alone do not permit a decision to be made among these accounts of slower response speed in ADHD. For the purpose of the present study, however, the important result is that the increased Stroop effect in ADHD may be attributable to the slower response speed of ADHD patients, whereas the specific mechanisms underlying this slowing require further research.

A somewhat puzzling finding relates to the increased error congruency effect in the ADHD compared to the control group. However, the distributional analysis of the error congruency effects revealed ADHD patients to show a larger error congruency effect than healthy controls only in trials with very fast responses. This is consistent with the idea that errors in interference paradigms occur when the distractor information in incongruent trials automatically activates the wrong response alternative via a fast direct processing route (Ridderinkhof, 2002). Assuming that response activation via the direct route may be more pronounced in ADHD patients than in healthy controls, as suggested by Ridderinkhof et al. (2005), it would appear that the distractor information activated the incorrect response alternative more often in patients than in normal controls; and this in turn would result in an increased error congruency effect in fast trials. Accordingly, the observation of an increased error congruency effect in the patient group can be explained by the assumption of enhanced automatic direct-route activation in the ADHD compared to the control group, which affects the decisions only in trials with fast RTs. In contrast, the findings do not provide evidence for an impairment of interference control processes involved in the active resolution of processing conflicts.

Although the present results question the assumption of interference control deficits in ADHD, our findings do not imply that adult

ADHD patients do not suffer from impairments in the other domains of executive functions. This is so because the concept of executive functions represents a theoretical construct that includes several subcomponents such as interference control, working memory, and set shifting (Miyake et al., 2000); accordingly, ADHD might still be related to deficits in the other domains of executive functions than interference control. In fact, several studies found ADHD-related deficits also for working memory and set shifting processes (Burgess et al., 2010; Finke et al., 2011; King et al., 2007; Marchetta et al., 2008). Consequently, the results of the present study do not question the assumption of deficits in executive functions as a core syndrome of adult ADHD, but only the evidence for impaired interference control deriving from paradigms such as the Stroop task. It should also be noted that dissociable control mechanisms may be involved in resolving the different types of conflicts, e.g. in resolving stimulus-based conflicts in the Stroop task and response-based conflicts in the Simon task (Egner et al., 2007). Hence, despite our findings for the Stroop task, ADHD participants might conceivably still suffer from interference control deficits in the Simon task. In fact, there is evidence that response inhibition is impaired in adult ADHD (Lijffijt et al., 2005). Future research will have to clarify whether ADHD patients suffer of conflict-specific control deficits.

As in adult ADHD, interference control deficits in child ADHD are also often accompanied by a generally slower performance level (Homack & Riccio, 2004; Mullane, Corkum, Klein, McLaughlin, & Lawrence, 2011; Van Mourik et al., 2009). However, a meta-analysis found evidence for impaired interference control even when controlling for performance speed (Lansbergen, Kenemans, & van Engeland, 2007). Another study that investigated interference control in child ADHD by delta plots found that children with ADHD showed worse response inhibition performance than a healthy control group (Ridderinkhof et al., 2005). Despite our divergent findings for *adult* ADHD patients, control deficits may well be among the cognitive symptoms in *childhood* ADHD, given that the evidence for a relationship between interference control deficits and general performance speed in child ADHD is still pending and that cognitive deficits may partly differ between child and adult ADHD (Biederman, Mick, & Faraone, 2000). There is evidence that the development of prefrontal cortical areas (associated with cognitive control) is only delayed in children with ADHD compared to controls (Shaw et al., 2012), such that adults with ADHD may (at least partly) recover from the control deficits exhibited in childhood.

From a methodological point of view, our results underscore the usefulness of distributional analyses such as delta plots for comparing performance between (various) patient groups and healthy control subjects with different mean RT levels. This is of particular importance when comparing an effect whose magnitude is known (or suspected) to depend on general response speed. In such cases, the finding of differential effect sizes between two groups may lead to false conclusions if these groups also differ in their general response speed, owing to, for instance, differences in arousal or motor speed. Delta plots permit the magnitude of the respective effects to be compared between the groups at similar RT levels and so help to avoid false conclusions. Note that delta plots are not only helpful when it is already known that the magnitude of an effect depends on the RT level, but they also provide a useful tool for examining the distributional shape of an effect. In addition, delta plots should also be preferred to the use of performance-matched control groups in clinical research because they do not require the exclusion of control subjects from the data set. Thus, they can be recommended as a standard tool whenever performance in groups with different mean RTs is compared. Importantly, however, they require that the same cognitive processes are involved in slow and fast trials in an experimental task. In the context of the present study, the finding that the slopes of the delta plot segments did not significantly differ from each other suggests that this pre-condition was approximately met in our experiment.

5. Conclusions

Previous studies often considered the finding of a larger interference effect in adult ADHD patients compared to healthy controls as evidence for the interference control deficits in ADHD. The results of the present study reveal that this performance difference disappears when performance is compared at equal response speed levels. In addition, we found that also a more direct measure of cognitive control, namely, the conflict adaptation effect, did not differ between the ADHD and control groups. Our results therefore question the assumption of interference control deficits in ADHD. Moreover, the present study illustrates the importance of comparing cognitive deficits between patient and control groups in reaction time tasks with delta plots, which allow performance to be compared at similar levels of response speed.

Acknowledgments

The present research was supported by a grant of the German Research Foundation to T.S. as well as by a grant of CoTeSys (No. 439) to T.S. A.S. was supported by a PhD scholarship of the Bavarian Elite Aid Act (BayEFG). W.S. received Ph.D. scholarships by the Graduate School of Systemic Neurosciences (GSN) Munich and by the Bavarian Elite Aid Act (BayEFG). Prof. Dr. Möller has received grants or is a consultant for and on the speakership bureaus of AstraZeneca, Bristol-Myers Squibb, Eisai, Eli Lilly, GlaxoSmithKline, Janssen Cilag, Lundbeck, Merck, Novartis, Organon, Pfizer, Sanofi-Avantis, Schering-Plough, Schwabe, Sepracore, Servier and Wyeth. Correspondence concerning this article should be addressed to Alexander Soutschek, Ludwig-Maximilians-Universität München, Department of Psychology, Leopoldstr. 13, 80802 Munich, Germany. Electronic mail should be addressed to Alexander.Soutschek@psy.lmu.de.

References

Banaschewski, T., Ruppert, S., Tannock, R., Albrecht, B., Becker, A., Uebel, H., et al. (2006). Colour perception in ADHD. *Journal of Child Psychology and Psychiatry*, *47*, 568–572.

Banich, M. T., Burgess, G. C., Depue, B. E., Ruzic, L., Bidwell, L. C., Hitt-Laustsen, S., et al. (2009). The neural basis of sustained and transient attentional control in young adults with ADHD. *Neuropsychologia*, *47*, 3095–3104.

Barkley, R. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*, 65–94.

Biederman, J. (2005). Attention-deficit/hyperactivity disorder: A selective overview. *Biological Psychiatry*, *57*, 1215–1220.

Biederman, J., Mick, E., & Faraone, S. V. (2000). Age-dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. *The American Journal of Psychiatry*, *157*, 816–818.

Boonstra, A. M., Oosterlaan, J., Sergeant, J. A., & Buitelaar, J. K. (2005). Executive functioning in adult ADHD: A meta-analytic review. *Psychological Medicine*, *35*, 1097–1108.

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

Bub, D. N., Masson, M. E. J., & Lalonde, C. E. (2006). Cognitive control in children: Stroop interference and suppression of word reading. *Psychological Science*, *17*, 351–357.

Burgess, G. C., Depue, B. E., Ruzic, L., Willcutt, E. G., Du, Y. P., & Banich, M. T. (2010). Attentional control activation relates to working memory in attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *67*, 632–640.

Bush, G., Frazier, J. A., Rauch, S. L., Seidman, L. J., Whalen, P. J., Jenike, M. A., et al. (1999). Anterior cingulate cortex dysfunction in attention-deficit/hyperactivity disorder revealed by fMRI and the counting Stroop. *Biological Psychiatry*, *45*, 1542–1552.

Castellanos, F. X., Sonuga-Barke, E. J., Milham, M. P., & Tannock, R. (2006). Characterizing cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Sciences*, *10*, 117–123.

Conners, C. K., Erhardt, D., & Sparrow, E. (2002). *Conner's Adult ADHD Rating Scales (CAARS)*. North Tonawanda, NY: Multi Health Systems.

De Jong, R., Liang, C. -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*, 731–750.

Egner, T. (2007). Congruency sequence effects and cognitive control. *Cognitive, Affective, & Behavioral Neuroscience*, *7*, 380–390.

Egner, T., Delano, M., & Hirsch, J. (2007). Separate conflict-specific cognitive control mechanisms in the human brain. *NeuroImage*, *35*, 940–948.

Faraone, S. V., Biederman, J., Spencer, T., Wilens, T., Seidman, L. J., Mick, E., et al. (2000). Attention-deficit/hyperactivity disorder in adults: An overview. *Biological Psychiatry*, *48*, 9–20.

Finke, K., Schwarzkopf, W., Müller, U., Frodl, T., Müller, H. J., Schneider, W. X., et al. (2011). Disentangling the adult attention-deficit hyperactivity disorder endophenotype: Parametric measurement of attention. *Journal of Abnormal Psychology*, *120*, 890–901.

Groves, J. A., & Engel, R. R. (2007). The German adaptation and standardization of the Personality Assessment Inventory (PAI). *Journal of Personality Assessment*, *88*, 49–56.

Hathaway, S. R., McKinley, J. C., & Engel, R. (2000). *Minnesota-Multiphasic Personality Inventory 2 (MMPI-2)*. Bern: Huber.

Heishman, S. J., Kleykamp, B. A., & Singleton, E. G. (2010). Meta-analysis of the acute effects of nicotine and smoking on human performance. *Psychopharmacology*, *210*, 453–469.

Homack, S., & Riccio, C. A. (2004). A meta-analysis of the sensitivity and specificity of the Stroop Color and Word Test with children. *Archives of Clinical Neuropsychology*, *19*, 725–743.

Hommel, B., Proctor, R. W., & Vu, K. P. (2004). A feature-integration account of sequential effects in the Simon task. *Psychological Research*, *68*, 1–17.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*, 1023–1026.

King, J. A., Colla, M., Brass, M., Heuser, I., & von Cramon, D. Y. (2007). Inefficient cognitive control in adult ADHD: Evidence from trial-by-trial Stroop test and cued task switching performance. *Behavioral and Brain Functions*, *3*, 42.

Lansbergen, M. M., Kenemans, J. L., & van Engeland, H. (2007). Stroop interference and attention-deficit/hyperactivity disorder: A review and meta-analysis. *Neuropsychology*, *21*, 251–262.

Lehrl, S., Triebig, G., & Fischer, B. (1995). Multiple-choice vocabulary test MWT as a valid and short test to estimate premorbid intelligence. *Acta Neurologica Scandinavica*, *91*, 335–345.

Leth-Steensen, C., Elbaz, Z. K., & Douglas, V. I. (2000). Mean response times, variability, and skew in the responding of ADHD children: A response time distributional approach. *Acta Psychologica*, *104*, 167–190.

Lijffijt, M., Kenemans, J. L., Verbaten, M. N., & van Engeland, H. (2005). A meta-analytic review of stopping performance in attention-deficit/hyperactivity disorder: Deficient inhibitory motor control? *Journal of Abnormal Psychology*, *114*, 216–222.

Lundervold, A. J., Adolfsdottir, S., Halleland, H., Halmøy, A., Plessen, K., & Haavik, J. (2011). Attention Network test in adults with ADHD — The impact of affective fluctuations. *Behavioral and Brain Functions*, *7*, 27–34.

Marchetta, N. D. J., Hurks, P. P. M., Krabbendam, L., & Jolles, J. (2008). Interference control, working memory, concept shifting, and verbal fluency in adults with attention-deficit/hyperactivity disorder (ADHD). *Neuropsychology*, *22*, 74–84.

Mayr, U., Awh, E., & Laurey, P. (2003). Conflict adaptation effects do not require executive control. *Nature Neuroscience*, *6*, 450–452.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: A latent variable analysis. *Cognitive Psychology*, *41*, 49–100.

Mullane, J. C., Corkum, P. V., Klein, R. M., McLaughlin, E. N., & Lawrence, M. A. (2011). Alerting, orienting, and executive attention in children with ADHD. *Journal of Attention Disorders*, *15*, 310–320.

Nigg, J. T. (2005). Neuropsychologic theory and findings in attention-deficit/hyperactivity disorder: The state of the field and salient challenges for the coming decade. *Biological Psychiatry*, *57*, 1424–1435.

Nigg, J. T., & Casey, B. J. (2005). An integrative theory of attention-deficit/hyperactivity disorder based on the cognitive and affective neurosciences. *Development and Psychopathology*, *17*, 785–806.

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*, 2013–2052.

Ratcliff, R., & Rouder, J. N. (1998). Modeling response times for two-choice decisions. *Psychological Science*, *9*, 347–356.

Ridderinkhof, K. R. (2002). Activation and suppression in conflict tasks: Empirical clarification through distributional analyses. In W. Prinz, & B. Hommel (Eds.), *Common mechanisms in perception and action. Attention & performance*, *vol. XIX*. (pp. 494–519) Oxford: Oxford University Press.

Ridderinkhof, K. R., Scheres, A., Oosterlaan, J., & Sergeant, J. (2005). Distribution-analytical techniques in the study of ADHD: Delta plot analyses reveal deficits in response suppression that are eliminated by methylphenidate treatment. *Journal of Abnormal Psychology*, *114*, 197–215.

Rouder, J. N., Yue, Y., Speckman, P. L., Pratte, M. S., & Province, J. M. (2010). Gradual growth vs. shape invariance in perceptual decision making. *Psychological Review*, *117*, 1267–1274.

Sergeant, J. A. (2005). Modeling attention-deficit/hyperactivity disorder: A critical appraisal of the cognitive-energetic model. *Biological Psychiatry*, *57*, 1248–1255.

Shaw, P., Malek, M., Watson, B., Sharp, W., Evans, A., & Greenstein, D. (2012). Development of cortical surface area and gyrification in attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *72*, 191–197.

Sonuga-Barke, E. J. S., Wiersema, J. R., van der Meere, J. J., & Roeyers, H. (2010). Context-dependent dynamic processes in attention deficit/hyperactivity disorder: Differentiating common and unique effects of state regulation deficits and delay aversion. *Neuropsychology Review*, *20*, 86–102.

Soutschek, A., Müller, H. J., & Schubert, T. (2013). Conflict-specific effects of accessory stimuli on cognitive control in the Stroop and the Simon task. *Experimental Psychology*, *60*, 140–148.

Soutschek, A., Strobach, T., & Schubert, T. (2012). Working memory demands modulate cognitive control in the Stroop task. *Psychological Research* (Epub ahead of print).

Sprafkin, J., Gadow, K. D., Weiss, M. D., Schneider, J., & Nolan, E. E. (2007). Psychiatric comorbidity in ADHD symptom subtypes in clinic and community adults. *Journal of Attention Disorders*, *11*, 114–124.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, *8*, 643–662.

Taylor, C. J., & Miller, D. C. (1997). Neuropsychological assessment of attention in adults. *Journal of Attention Disorders*, *2*, 77–88.

Usher, M., & McClelland, J. L. (2001). On the time course of perceptual choice: The leaky competing accumulator model. *Psychological Review*, *108*, 550–592.

Van der Meere, J. J. (2005). State regulation and attention deficit hyperactivity disorder. In D. Gozal, & D. L. Molfese (Eds.), *Attention deficit hyperactivity disorder: From genes to patients* (pp. 162–213). Totowa: Humana.

Van Mourik, R., Papanikolau, A., van Gellicum-Bijlhout, J., van Oostenbruggen, J., Veugelers, D., Post-Uiterweer, A., et al. (2009). Interference control in children with attention deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology*, *37*, 293–303.

Walker, A. J., Shores, E. A., Trollor, J. N., Lee, T., & Sachdev, P. S. (2000). Neuropsychological functioning of adults with attention deficit hyperactivity disorder. *Journal of Clinical and Experimental Neuropsychology*, *22*, 115–124.

Ward, M. F., Wender, P. H., & Reimherr, F. W. (1993). The Wender Utah rating-scale — An aid in the retrospective diagnosis of childhood attention-deficit hyperactivity disorder. *The American Journal of Psychiatry*, *150*, 885–890.

World Health Organization (1992). *The ICD-10 classification of mental and behavioural disorders, clinical descriptions and diagnostic guidelines.* Genève: World Health Organization.