

Deciphering interference control in adults with ADHD by using distribution analyses and electromyographic activity



Isabel Suarez ^a, B. Burle ^a, C. Tobon ^b, D. Pineda ^b, F. Lopera ^b, T. Hasbroucq ^a, L. Casini ^{a,*}

^a Aix-Marseille Université, CNRS, LNC UMR 7291, Laboratoire de Neurosciences Cognitives, FR 3C, Case C, 3 place Victor Hugo, 13331 Marseille cedex 3, France

^b Neurosciences Group, University of Antioquia, Torre 1 Piso 1, Medellín, Colombia

ARTICLE INFO

Article history:

Received 13 May 2014

Received in revised form 18 March 2015

Accepted 26 May 2015

Available online xxxx

PsycINFO classification:

3250

Keywords:

Adults with ADHD

Interference control

EMG

Distributional analyses

Sequential effects

ABSTRACT

A deficit in “interference control” is commonly found in adults with Attention Deficit Hyperactivity Disorder (ADHD). This has mainly been interpreted as difficulties in inhibiting inappropriate responses. However, interference control involves processes other than simply the ability to inhibit. Consequently, we used sophisticated analysis to decipher the additional processes of interference control in these patients.

We compared interference control between 16 adults with ADHD and 15 control adults performing a Simon task. In most studies, performance is generally reported in terms of mean error rates and reaction times (RTs). However, here we used distribution analyses of behavioral data, complemented by analyses of electromyographic (EMG) activity. This allowed us to better quantify the control of interference, specifically the part that remains hidden when pure correct trials are not distinguished from partial errors. Partial errors correspond to sub-threshold EMG bursts induced by incorrect responses that immediately precede a correct response. Moreover, besides “online” control, we also investigated cognitive control effects manifesting across consecutive trials.

The main findings were that adults with ADHD were slower and showed a larger interference effect in comparison to controls. However, the data revealed that the larger interference effect was due neither to higher impulse expression, nor to a deficit in inhibition but that these patients presented a larger interference effect than the controls after congruent trials.

We propose and discuss the hypothesis that the interference control deficit found in adults with ADHD is secondary to impairments in sustained attention.

© 2015 Published by Elsevier B.V.

1. Introduction

Attention Deficit/Hyperactivity Disorder (ADHD) is one of the most common developmental disorders diagnosed in childhood. It corresponds to symptoms of inattention, hyperactivity and impulsivity (APA, 2000). The persistence of this disorder into adulthood has been estimated at 70% (Spencer et al., 2007) leading to severe functional consequences such as underemployment, social status impairment, and risky behavior (Biederman and Faraone, 2005, 2006; Biederman et al., 1991; Moura et al., 2013). To better understand causes of these persistent behavioral difficulties, the present study focuses on young adults with ADHD.

ADHD has been described as a disorder of executive functioning by several authors (Barkley, 1997; Sergeant et al., 2002) and the predominant theories of neurocognitive deficit associated with ADHD more

particularly focus on impulsivity and deficits of behavioral inhibition as being at the origin of this disorder (Barkley, 1997; Nigg, 2001). To investigate both impulsivity and response inhibition, conflict tasks are particularly relevant. In such tasks, impulsive response triggered by irrelevant features of the stimulus sometimes may conflict with the response required by the rule associated with the task.

In general, poor performance is observed in adults with ADHD compared with controls in conflict tasks such as the Stroop task (King et al., 2007; Taylor and Daniel, 1997; Walker et al., 2000) or the Eriksen flanker task (Lundervold et al., 2011). Most of these studies revealed longer reaction times (RTs) and larger interference effects in adults with ADHD; rare studies only did not report larger interference effects (Banich et al., 2009; Soutschek et al., 2013). In the present study, we used the well-known Simon task (Simon, 1969). In this task, participants have to choose between a left- and a right-hand key press according to the color of a visual stimulus presented either to the left or the right of a central fixation point. Although stimulus position is irrelevant for the task, the performance, expressed both in terms of error rate and RT, is better when the required response spatially corresponds to the stimulus location (congruent association) than when it does not correspond (incongruent association), even though the stimulus location is irrelevant to the task. This effect is called the “Simon effect”

* Corresponding author at: LNC, FR 3C, Case C, 3 place Victor Hugo, 13331 Marseille cedex 3, France.

E-mail addresses: isabel.suarez-del-chiaro@etu.univ-amu.fr (I. Suarez), boris.burle@univ-amu.fr (B. Burle), cantobon@gmail.com (C. Tobon), dpineda12@hotmail.com (D. Pineda), flopera@une.net.co (F. Lopera), thierry.hasbroucq@univ-amu.fr (T. Hasbroucq), laurence.casini@univ-amu.fr (L. Casini).

or “interference effect” (Hedge and Marsh, 1975; Hommel, 2011; Simon, 1990). A widely accepted interpretation of the Simon effect is that the stimulus location automatically triggers a response impulse in the ipsilateral hand via a fast information processing route, while the relevant stimulus color must be translated into the required response according to task instructions via a slower controlled processing route (de Jong, Liang and Lauber, 1994; Kornblum, 1994; Kornblum et al., 1990; Proctor et al., 1995). When the stimulus–response association is congruent, the impulse triggered by the irrelevant stimulus location incidentally activates the required response, thus facilitating response processing. In contrast, when the stimulus–response association is incongruent, the impulse triggered by the irrelevant location activates the non-required response, which then competes with the required one. This competition is thought to be at the origin of the performance impairment.

Ridderinkhof (2002a,b) extended the dual-route hypothesis by proposing the “activation–suppression” model which incorporates the temporal dynamics of cognitive control and also allows the expression of impulse capture to be studied separately from inhibitory control. This model keeps the notion that the irrelevant location of the stimulus triggers the process of impulse capture, which is assumed to occur very quickly after the onset of the stimulus. It further adds an active suppression of the inappropriate impulse response, which reduces interference due to the incorrect action impulse; this second process is engaged more slowly (Ridderinkhof, 2002a). The strength of impulse capture, that is vulnerability to irrelevant features of the stimulus, should then be reflected in the proportion of fast errors, which can be evaluated by plotting accuracy rates against RT (known as conditional accuracy functions, CAF; Van den Wildenberg et al., 2010; Wylie et al., 2010; Wylie et al., 2012). On the other hand, inhibitory control should be more evident during slower responses, that is at the right side of the RT distribution when interference suppression has had time to build up. By plotting the difference between incongruent RTs and congruent RTs, which reflects the magnitude of interference effects, as a function of response speed (so-called delta plots), a pattern of reduced interference can be observed in the slowest segments of the RT distribution as the suppression mechanism becomes more fully engaged (de Jong et al., 1994; Ridderinkhof, 2002a,b). Consequently, according to the activation–suppression model, the slope of the Simon effect at the longest RTs provides a measure of inhibitory control over prepotent responses, whereas the slope of the CAF at the shortest RTs, for incongruent trials, provides a measure of the strength of impulse capture. Ridderinkhof et al. (2005) applied this method to investigate interference control in an Eriksen task in children with ADHD. They showed that performance deficits observed in these children involved response inhibition but not automatic response activation. Investigating whether this also holds for young adults with ADHD was the main goal of the present study. Two main differences with Ridderinkhof et al. (2005) study are to be noted. First, we used a Simon task because suppression is more difficult to evidence and less stable in the Eriksen task (Burle et al., 2014). Second, in Ridderinkhof’s study, as in most other studies of interference control, responses were classified simply as either correct or erroneous. However, analyses of response-related electromyographic (EMG) activity during such tasks reveals that committing an error is not an all-or-none process. Indeed, in about 20% of responses classified as correct responses, an early sub-threshold EMG burst recorded from the hand associated with the incorrect response precedes EMG activity related to the correct response (Fig. 1). Several lines of evidence from previous studies have indicated that these sub-threshold activities reflect suppression of erroneous impulses to prevent an incorrect response being made, rather than reflecting task-unrelated contractions (Allain et al., 2009; Hasbroucq et al., 1999; for a review, see also Van den Wildenberg et al., 2010) and such an EMG activity can be considered as reflecting “partial errors”. Therefore, among overt correct responses, two categories of trials may be distinguished: “pure correct” and “partial error” trials. In the same vein, it may be

assumed that incorrect response activity occurs both for real errors and for partial errors but is successfully suppressed and corrected for partial errors only (Spieser et al., 2015). By consequence, the incorrect activation rate, calculated as the sum of partial errors and real errors, provides a better measure of the vulnerability to stimulus-driven incorrect response activation and the ratio between the number of partial errors to the number of all incorrect activations (real errors added to partial errors), called the “correction rate”, represents the ability to suppress incorrect responses (Burle et al., 2002), with a lower correction rate reflecting an impairment in response inhibition.

In summary, while in most of studies using the Simon task, performance has generally been reported in terms of mean reaction times (RT) solely, here we used distribution analyses of behavioral data complemented by analysis of EMG activity to better quantify the control of interference in adults with ADHD and, more specifically, that part which remains hidden when pure correct trials are not distinguished from partial errors. If inhibitory processes are impaired in patients with ADHD, this should be reflected by a shallower slope in the delta-plot and a lower correction rate, and a higher vulnerability to stimulus-driven incorrect response activation should be reflected by a steeper slope in the CAF and a higher incorrect activation rate.

Besides “online” control, we also investigated cognitive control effects manifesting across consecutive trials. Indeed, the so-called “conflict adaptation effect” is largely considered as another measure of cognitive control processes (Botvinick et al., 2001; Egner and Hirsch, 2005; Gratton et al., 1992; Mayr et al., 2003). This effect refers to the finding that the congruency effect largely depends on the nature of the previous trial: while the congruency effect is large after a congruent trial, it gets largely reduced or even disappears after an incongruent trial (Stürmer et al., 2002). It has been proposed that after incongruent trials, more challenging than congruent ones, cognitive control is enhanced leading to attention refocalization on the relevant attribute of stimuli during the next trial (Botvinick et al., 2001; Egner, 2007; Ullsperger et al., 2005), the nature of the signal triggering the refocalization remains unclear however (Burle et al., 2005). Such a refocalization of attention will not only reduce the interference in incongruent trials but also reduce facilitation in congruent ones. As a consequence, the difference between congruent and incongruent trials would get reduced after attention refocalization. Symmetrically, it is classically assumed that attention focalization being an effortful process, in the absence of challenging trials (and/or after the facilitatory effect of the irrelevant dimension in congruent trials), attention would broaden and the impact of the irrelevant dimension would be enhanced. In this case, correctly responding will be further facilitated in congruent trials, while a larger interference will occur for incongruent trials. Hence, a reduced interference effect after incongruent trials reflects the refocalization of attention, while an enlarged one effect after congruent trials reflects the relaxation of attention. Therefore, we also analyzed conflict adaptation effects in adults with ADHD.

2. Method

2.1. Participants

We tested two groups of subjects, recruited through the Science Investigation center of the University of Antioquia (Colombia): 16 ADHD adults (age 18–29, mean = 22.13, 14 males) and 15 healthy adults (age 19–25, mean = 21.68, 11 males). All participants gave informed consent to the experimental procedure, following the Helsinki declaration (1964). The study was approved by the Ethical Committee of the Investigation Center of the University of Antioquia (Colombia).

2.1.1. Selection procedure for the ADHD group

Adults in the ADHD group were recruited from the clinical database of the Neurosciences Clinic of the University of Antioquia

(Colombia). All participants in the present study were selected among patients who were diagnosed with ADHD as children, who continue to be observed by psychiatrists of the clinic, and for whom the diagnosis was again confirmed in young adulthood by two psychiatrists in the last two years.

Clinical diagnoses and definitions of ADHD (i.e., affected or non-affected, as defined by DSM-IV) used in this clinic have been extensively described in detail elsewhere (Palacio et al., 2004) and will be only briefly described here. For childhood diagnoses, structured diagnostic interviews were conducted in the Neurosciences Clinic by a team of professionals supervised by an expert psychiatrist who reviewed all interviews and conducted confirmatory clinical interviews with all participants. Parents underwent a full psychiatric structured interview regarding their offspring (Diagnostic Interview for Children and Adolescents-Revised Parents Version [DICA-IV-P], Spanish version translated with permission from [Reich, 2000]). Parents and teachers of school-age children also provided behavior rating scales. Final diagnoses were reached by a consensus on the basis of the results of structured interviews, collateral historical information, and clinical interviews for each family lineage member through the best estimate procedure (Reich, 2000) by a committee of four clinicians, all of whom have extensive experience with ADHD. Definitely affected subjects met full DSM-IV ADHD criteria during childhood, with onset before age 7 years, and with persistence of clearly impairing symptoms in more than one setting. In cases of discordance between an individual's self-report of symptoms and collateral reports, the supervising psychiatrist obtained further collateral information and probed more deeply for evidence of early impairment. For confirmation in young adulthood, patients received a structured interview for current functioning and evaluating symptoms of ADHD described in DSM-IV by two different psychiatrists.

For this study, in order for the ADHD group to be as homogenous as possible but also not reduced to only one symptom (impulsivity or attention), only adults who had been diagnosed with the combined ADHD type in childhood and with diagnosis confirmed in young adulthood were contacted and solicited for their participation.

It is important to note that all participants selected for this study had been under medication (methylphenidate) at different periods of their life but had not taken medication in the six months prior to the experiment.

2.1.2. Criteria for the control group

The control group was composed of young adults without ADHD recruited at the University of Antioquia (similar to most of patients). Diagnostic interviews were also conducted by the psychiatrists and/or neuropsychologists of the Neurosciences Clinic. Inclusion criteria were a lack of history of diagnosis of ADHD on the basis of a retrospective assessment of ADHD by self-completion of a 61-item Spanish version of the Wender Utah Rating Scale (WURS) (Rodríguez-Jiménez et al., 2001) and failure to meet the cut-off point.

2.1.3. Exclusion criteria for both groups

Exclusion criteria included 1/ a diagnosis of additional psychiatric (major depression, panic disorder, suicide risk, anxiety, substance abuse, psychoactive substance use, psychotic disorders) or neurological disorder on the basis of a Spanish version of the structured psychiatric interview mini mental test (M.I.N.I.), 2/ current or recent drug abuse (inferior to 6 months), and 3/ an intelligence quotient (IQ) inferior to 80, evaluated with the short-form of the Wechsler Adults Intelligence Scale (WAIS III) including four sub-tests: similarities and vocabulary tests to estimate verbal IQ, and picture completion and block design tests to estimate performance IQ. In addition to IQ measures, the working memory index was assessed using three items of the WAIS III (arithmetic, digit span, and letter-number sequencing).

2.2. Complementary neuropsychological assessments

In addition, our subjects performed a stop-task and a continuous performance test (CPT) task (Conners & Staff, 2000) to allow us to study correlations between indices measured in the Simon task and more classical and global measures of inhibition (obtained from the stop-task) and of sustained attention (obtained from the CPT task).

Briefly, in the CPT task used here, participants were required to press the space-bar as fast as possible for any letter appearing on the screen except for the letter "X" (7.5% of trials). Participants performed 2 blocks of 40 trials for each inter-stimulus interval (ISI of 1 s, 2 s, or 4 s). Sustained attention and distractibility were evaluated by mean RT, mean intra-subject RT variability and omission rate at the ISI of 4 s. Impulsivity was evaluated by the commission rate at the ISI of 1 s.

In the version of the stop-task we used here, participants had to choose between two hand button-presses in response to a visual stimulus (circle or square) appearing on the screen 500 ms after a central fixation point. They were required to respond as quickly as possible, and in 25% of trials, a stop-signal (a 600 Hz auditory tone) was presented shortly after the visual stimulus indicating that they had to withhold their response. The delay between the presentation of the visual stimulus and the onset of the stop-signal (stop-signal delay, SSD) was initially set to 250 ms and was adjusted up or down in 50 ms increments depending on the accuracy of the participants' response. Mean reaction time to the go-stimulus (go-RT) and stop signal reaction time (SSRT) were recorded, SSRT being used as an index of inhibitory motor control.

2.3. Procedure of Simon RT task

2.3.1. Stimuli and apparatus

Participants were seated comfortably in a dimly lit sound-shielded room, facing a black panel, which was located 1 m away and contained three light emitting diodes (LED). They were horizontally aligned and spaced 2.5 cm apart. The two lateral LEDs displayed the stimuli and both could be either red or green. The central LED was white and served as a fixation point.

Participants used their right and left thumbs to press two response buttons (3.5 cm in diameter, 9 cm in height) fixed on a table in front of them and spaced 30 cm apart. The subjects continuously kept the distal phalanx of the left thumb on the left button and the distal phalanx of the right thumb on the right button. The arms and the hypothenar eminences rested on the table as comfortably as possible. All stimuli and responses were controlled by a computer running t-scope (Stevens et al., 2006). RTs were recorded to the nearest millisecond.

2.3.2. Task and procedure

The participant's task was to respond as fast and as accurately as possible on the basis of the color of the stimulus. After the experimenter had verified that all instructions were understood, participants first performed 48 trials (24 trials of each type, congruent and incongruent, all randomized) to familiarize them with the task and allow them to stabilize their performance. Next, six experimental blocks were presented, each containing 96 trials. Blocks of trials were separated by 5 min.

Each trial started with a tone serving as a warning stimulus. After a variable delay (ranging from 250 ms to 600 ms) following the warning signal, one of the two lateral LEDs lighted on either green or red on the right or on the left of a white circle serving as a fixation point. Participants had to briefly press a response button as quickly as possible with the left or the right thumb according to the color of the LED. The color-response mapping was balanced across subjects.

There were 48 green and 48 red stimuli. For each color, there were two types of trials: 24 congruent trials (response side ipsilateral to stimulus side) and 24 incongruent trials (response side contralateral to the stimulus side).

2.4. EMG signal recording and processing

The EMG activity was recorded from the flexor pollicis brevis of each thumb by paired surface Ag/AgCl electrodes, 6 mm in diameter, fixed about 10 mm apart on the skin of the thenar eminence. This activity was amplified, filtered (low/high frequencies cut-off at 10 Hz/1 kHz), full-wave rectified, and digitized on-line (A/D rate 2 kHz). The EMG signal was continuously monitored by the experimenter to minimize background activity that could interfere with small bursts of muscular activation during the reaction period. If the signal became noisy, the experimenter immediately asked the subject to relax his/her muscles.

In this study, it was important to detect very small degrees of erroneous muscular activation. To this end, EMG traces were inspected visually and electromyographic onsets were hand-scored (see Burle et al., 2002). Although automated algorithms can be useful (Hodges and Bui, 1996; Van Boxtel et al., 1993) the ultimate standard, against which the accuracy of the different algorithms is rated, remains visual inspection.

2.5. Data analysis

First, correct trials were sorted into two categories, labeled “pure-correct” and “partial error” trials. “Partial errors” trials presented an activation of the agonist involved in the incorrect response preceding the activation of the agonist involved in the correct response. Importantly, to be classified as a “partial error”, the EMG signal deflection had to be phasic and to return to baseline (rest) level before the onset of the response-related EMG activity. Two variables were then calculated: the incorrect activation rate (IA) which corresponds to the proportion of both partial errors and real errors, and the correction rate (CR) which was defined as $CR = PE / (PE + E)$ where PE reflects the number of partial errors and E the number of real errors. In other words, the CR reflects the number of corrected incorrect activations divided by the overall number of incorrect activations (corrected or not).

Second, distribution analyses were performed for RTs and for accuracy rates. The cumulative density functions of trials were estimated for each participant and averaged through the so-called “vincentizing” procedure (Ratcliff, 1979; Vincent, 1912): RTs were rank ordered for each type of trial separately (CG trials and IG trials), and binned into deciles of equal frequencies (same number of trials). The mean of each bin was computed and equivalent bins were averaged across participants. Delta-plots were constructed by plotting the difference between incongruent and congruent bins, as a function of the mean of incongruent and congruent bin values (for more information, see Burle et al., 2002; Ridderinkhof, 2002a). In distributional accuracy analysis, pure correct trials were distinguished from partial errors. We therefore vincentized incorrect activations (real errors added to partial errors), rather than erroneous trials alone, as a function of the RT distribution and the resulting distributional analysis was designated a “conditional incorrect activation function” (CIAF) instead of “conditional accuracy function” (CAF). Pure correct, partial errors and erroneous trials were mixed together and the resulting distributions were vincentized as described above. For each bin, the proportion of “pure correct” trials was computed along with the mean RT of the bin. These data-pairs were averaged per bin through participants. This provides the mean incorrect activation rate as a function of increasing RT.

Third, EMG activity was also used to determine the chronometric variables illustrated in Fig. 1. Reaction time was measured from the stimulus to the response button press. It was broken down into two components: premotor time (PMT, from stimulus to EMG onset) and motor time (MT, from EMG onset to button press). In partial error trials, two additional indices were defined: the incorrect activation latency (IAL) corresponding to the time between signal onset and incorrect EMG onset, and the correction time (CT) corresponding to the time between incorrect EMG onset and correct EMG onset.

For the analyses, percentages cannot be analyzed directly as the means and variances of percentages tend to be closely related.

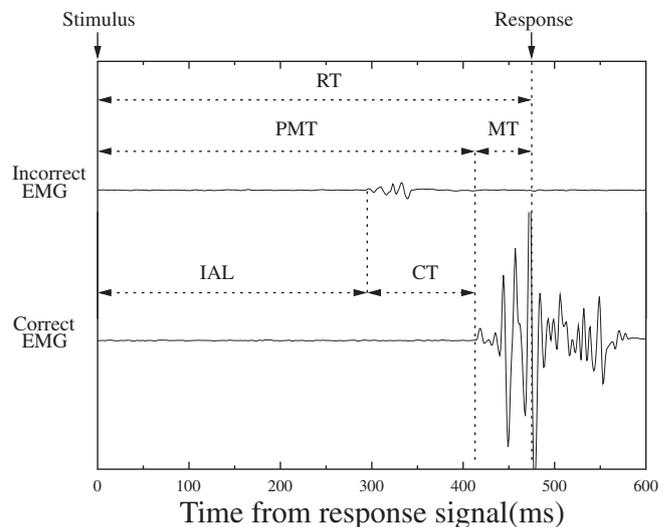


Fig. 1. Example of a “partial error” and chronometric indices. Electromyographic activity (EMG) from muscles controlling the incorrect (upper trace) and the correct (lower trace) responses. The vertical dashed line indicates the mechanical button-press response. The correct overt response was preceded by partial EMG activity in the muscle controlling the incorrect response, which was too weak to trigger an overt error. Chronometric indices are illustrated on the EMG activity in the involved (correct electromyographic activation) and non-involved (incorrect electromyographic activation) agonist muscles. RT: reaction time; PMT: premotor time (from the response signal to the correct electromyographic onset); MT: motor time (from the correct electromyographic onset to the mechanical response); IAL: incorrect activation latency (from the response signal to the onset of the electromyographic activity in the agonist muscle involved in the incorrect response); CT: correction time (from the incorrect electromyographic activation to the correct one).

Therefore, they were arcsine transformed (Winer, 1970) before being analyzed. A 2 way ANOVA with the between-subject factor Group (ADHD versus control) and the within-subject factor Congruency (congruent versus incongruent) was performed on the mean measures. For distribution analyses, the bin (1–10) was added as a third within-subject factor. Indeed, although delta-plots are shown for RT distributions, all statistical analyses were performed on the vincentized cumulative density functions.

3. Results

Performance of two adults with ADHD were not included in the statistical analyses because these two participants did not succeed in respecting task instructions. Analyses were thus performed with data of 15 control subjects and 14 adults with ADHD.

3.1. Demographic and neuropsychological variables

Differences between demographic characteristics of control and ADHD groups were tested using independent sample two-tailed *t* tests. As shown in Table 1, no significant differences existed in age, IQ scores, or working memory index. As expected, WURS scores were significantly different.

In the CPT task, adults with ADHD were significantly slower and more variable compared with control participants, but there was no difference between groups for the omission rate, nor for the commission rate. Concerning the stop-task, there was no difference between groups for the SSRT.

3.2. Simon task performance

The presentation of results will include four sections: 1/ overall mean RTs and error rates, 2/ distributional analyses, 3/ data coming from partial errors, and 4/ chronometric EMG indices.

Table 1

Demographic and neuropsychological variables for both groups (mean and standard deviation, SD). IQ = intellectual quotient; WMI = working memory index; WURS = Wender Utah Rating Scale. CPT = continuous performance test; RT = reaction time; ISI = inter stimulus interval; SSRT = stop signal reaction time.

	ADHD (N = 14) Mean ± SD	Control (N = 15) Mean ± SD	t value	p value
Age (years)	21.6 ± 4	22.2 ± 1.6	0.55	0.6
IQ	102.9 ± 10.6	100.9 ± 10.1	0.53	0.59
WMI	90.5 ± 8.7	95.8 ± 12.8	1.39	0.17
WURS	55.3 ± 10.9	18.7 ± 9.5	9.92	<0.0001
CPT task				
Mean RT in ms (ISI = 4 s)	427 ± 74	369 ± 49	2.59	<0.05
Mean intra-subject RT variability (ISI = 4 s)	79 ± 41	55 ± 14	2.09	<0.05
% of omission rate (ISI = 4 s)	0.53 ± 1.4	0.33 ± 1.8	0.45	0.65
% of commission rate (ISI = 1 s)	2.67 ± 2.5	1.83 ± 3.2	1.06	0.29
Stop signal task				
SSRT in ms	323 ± 40	330 ± 34	0.48	0.63

3.2.1. Mean RTs and error rates

Fig. 2A shows that mean RTs were longer for adults with ADHD (427 ms) compared with control adults (393 ms), $F(1, 27) = 6.75$, $p < 0.01$, and that RTs were shorter in congruent trials (394 ms) compared with incongruent trials (425 ms), $F(1, 27) = 125.33$, $p < 0.0001$. It can also be observed that the congruency effect was larger in ADHD group (36 ms) compared with control group (25 ms) as confirmed by the Group \times Congruency interaction, $F(1, 27) = 4.57$, $p < 0.05$.

As seen in Fig. 2B, patients with ADHD committed no more errors than control subjects, $F(1, 27) = 0.08$, $p = 0.77$, and as classically observed, error rate was larger in incongruent trials compared with congruent trials, $F(1, 27) = 24.42$, $p < 0.0001$. The Congruency \times Group interaction was not significant, $F(1, 27) = 0.20$, $p = 0.65$. Thus, overall performance measures suggest that compared with control adults, adults with ADHD are more sensitive to interference effects on response speed (but not accuracy) in the Simon task. These patterns could not be explained in terms of speed–accuracy trade-off.

In addition, there was a positive correlation (Pearson correlation) between the size of the Simon effect and the mean intra-subject RT variability on the CPT task, $r = 0.53$, $p < 0.05$, suggesting that subjects

that were more variable were also those that presented the largest Simon effect.

3.2.2. Distributional analyses

Fig. 3A displays delta-plots for RTs, representing the size of Simon effect as a function of deciles, for control and ADHD groups. The Simon effect decreased as RTs increased for both groups, as confirmed by the non-significant second order interaction, $F(9, 243) = 0.23$, $p = 0.98$. As seen in Fig. 3A, the slopes of the last segment of the delta-plot, which have been proposed to be a sensitive measure of inhibitory control, were not different between groups, $F(1, 27) = 2.01$, $p = 0.18$.

Fig. 3B displays distributional analyses for incorrect activation rates (CIAFs) for control and ADHD groups. The effect of congruency with response time was the same for both groups as confirmed by the absence of a second order interaction, $F(9, 243) = 0.71$, $p = 0.70$. More precisely, in incongruent trials accuracy rate increased for shorter RTs similarly for both groups. The slopes of the first segment of the plot, which have been proposed to be a sensitive measure of automatic response activation, were not different between groups, $F(1, 27) = 0.13$, $p = 0.71$.

3.2.3. Data from partial errors

Partial errors and errors were summed to provide an incorrect activation rate, which reflects the sensitivity of the subjects to the location of the stimulus. As observed in Table 2, incorrect activation rates were similar for ADHD and control groups, $F(1, 27) = 0.05$, $p = 0.81$. Incorrect activation rates were larger for incongruent than for congruent trials, $F(1, 27) = 0.36$, $p < 0.0001$, and there was no Group \times Congruency interaction, $F(1, 27) = 2.27$, $p = 0.14$.

Concerning the correction rate, there was no difference between groups, $F(1, 27) = 0.05$, $p = 0.81$, nor between congruency trials, $F(1, 27) = 0.03$, $p = 0.86$. The interaction Group \times Congruency was not significant, $F(1, 27) = 0.06$, $p = 0.81$.

3.2.4. Chronometric indices

Table 2 also presents mean premotor time (PMT), mean motor time (MT), mean incorrect activation latency (IAL) and mean correction time (CT) in congruent and incongruent trials for the ADHD and control groups. Premotor times (PMT) tended to be longer for adults with ADHD (307.5 ms) compared with control adults (280.5 ms), $F(1, 27) = 2.82$, $p = 0.10$. There was a congruency effect, $F(1, 27) = 135.71$, $p < 0.00001$, and a significant Congruency \times Group interaction, $F(1, 27) = 5.40$, $p < 0.05$, as observed for RTs. Concerning motor

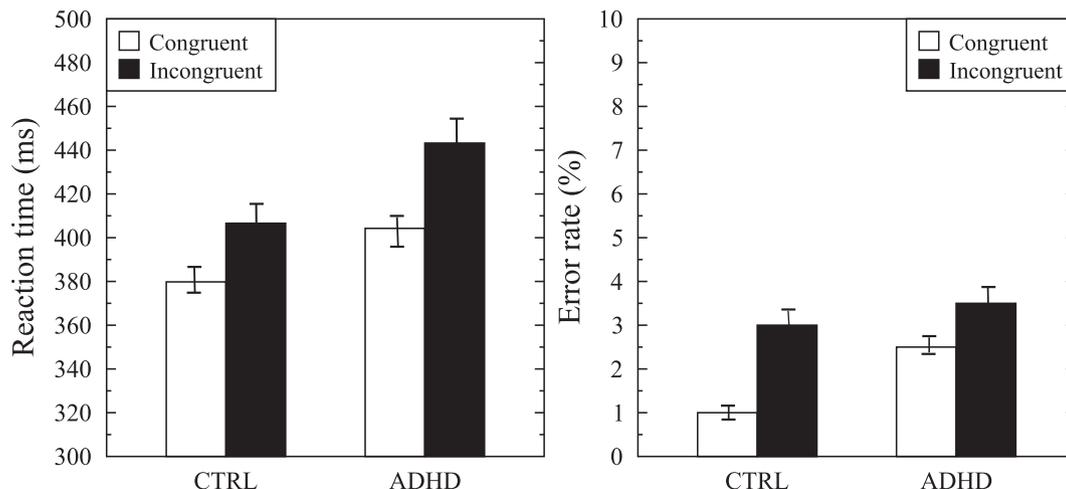


Fig. 2. Simon task. Overall mean RT (A) and mean error rate (B) for congruent (white bars) and incongruent (black bars) trials for both control and ADHD groups. Error bars are mean standard errors.

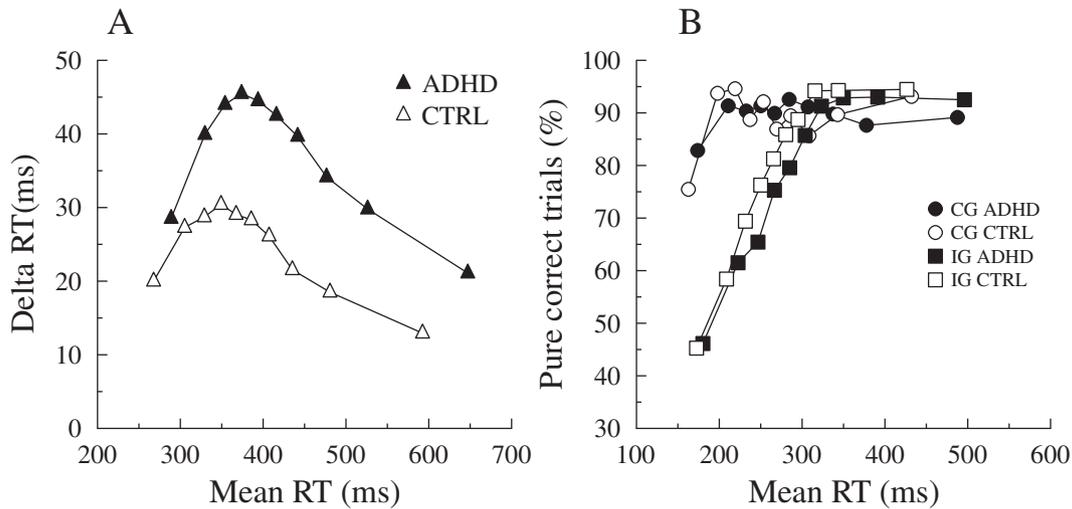


Fig. 3. Distributional analyses. (A) Delta plots showing Simon effect size as a function of response speed, expressed in reaction time (RT) quantile scores for control group (white triangles) and ADHD group (black triangles). (B) Conditional incorrect activation functions for congruent (circles) and incongruent (squares) trials for control (in white) and ADHD (in black) groups.

times (MT), there was no difference between groups, $F(1, 27) = 0.07$, $p = 0.79$, and no difference between congruent and incongruent trials, $F(1, 27) = 0.45$, $p = 0.5$.

Incorrect activation latency (IAL) tended to be longer in ADHD (259 ms) compared with the control group (232 ms), $F(1, 27) = 3.76$, $p = 0.06$, and shorter in incongruent trials than in congruent trials, $F(1, 27) = 26.18$, $p < 0.001$. No Group \times Congruency interaction was observed, $F(1, 27) = 0.56$, $p = 0.46$. Lastly, adults with ADHD tended to be longer to make a correct response after a partial error as compared with control participants (ADHD: CT = 271.5 ms; Control: CT = 248 ms; $F(1, 27) = 3.24$, $p = 0.08$). There was no effect of trial congruency, $F(1, 27) = 1.87$, $p = 0.18$, nor a significant Group \times Congruency interaction, $F(1, 27) = 0.47$, $p = 0.49$ on the correction time (CT).

3.2.5. Conflict adaptation effects

A $2 \times 2 \times 2$ mixed-design ANOVA including the between-subject factor Group (ADHS vs Control) and the within-subject factors Congruency (at trial n , Congruent vs Incongruent current trial) and trial $n - 1$ (Congruent vs Incongruent trial $n - 1$).

As illustrated in Fig. 4, there was a significant second order interaction Group \times Congruency \times trial $n - 1$, $F(1, 27) = 28.13$, $p < 0.00001$. After an IG trial $n - 1$, the Simon effect was reduced for both Control and ADHD groups and did not differ between the two groups, but after a CG trial $n - 1$, the Simon effect was larger in ADHD group (52 ms) compared with Control group (39 ms), $F(1, 27) = 7.88$, $p < 0.001$. More precisely, mean RT difference between Control and ADHD groups was larger for IG trials (41 ms) compared with CG trials

(29 ms), as confirmed by the Group \times Congruency interaction observed when considering $n - 1$ CG trials only, $F(1, 27) = 62.11$, $p < 0.0001$.

4. Discussion

The present study aimed to examine interference control in adults with ADHD engaged in a conflict task. Overall performance analyses revealed a larger interference effect (larger difference in RT between incongruent and congruent trials) in adults with ADHD than in control adults. These findings are consistent with results from other types of conflict task reporting stronger interference effects in adults with ADHD (Bush et al., 1999; King et al., 2007; Lundervold et al., 2011; Walker et al., 2000) and thus provide further support for the assumption of interference control deficits in adults with ADHD. However, our examination of indices from distributional analyses and EMG activity allowed us to investigate this deficit in interference control more deeply.

Interference control deficits can have three main causes: 1) a higher vulnerability to the location of the stimulus, which automatically activates the ipsilateral response via the direct route, 2) a dysfunction in the capacity to suppress this route or 3) a difficulty in refocalizing attention. The data we obtained in adults with ADHD did not support

Table 2

Mean incorrect activation rate, correction rate, and chronometric indices (PMT = premotor time, MT = motor time, IAL = incorrect activation latency, CT = correction time) for ADHD and control groups in CG (congruent) and IG (incongruent) trials. SD = standard deviation of the mean.

Congruency	ADHD		Control	
	CG Mean \pm SD	IG Mean \pm SD	CG Mean \pm SD	IG Mean \pm SD
Incorrect activation (%)	9 \pm 5.9	22 \pm 12.7	9 \pm 6.2	19 \pm 10.6
Correction rate (%)	77 \pm 20.6	80 \pm 13.7	79 \pm 22	79 \pm 20
PMT (in ms)	291 \pm 42	324 \pm 42	270 \pm 42	291 \pm 47
MT (in ms)	110 \pm 18	109 \pm 18	107 \pm 26	108 \pm 26
IAL (in ms)	273 \pm 45	245 \pm 32	242 \pm 43	222 \pm 36
CT (in ms)	265 \pm 48	278 \pm 27	246 \pm 48	250 \pm 28

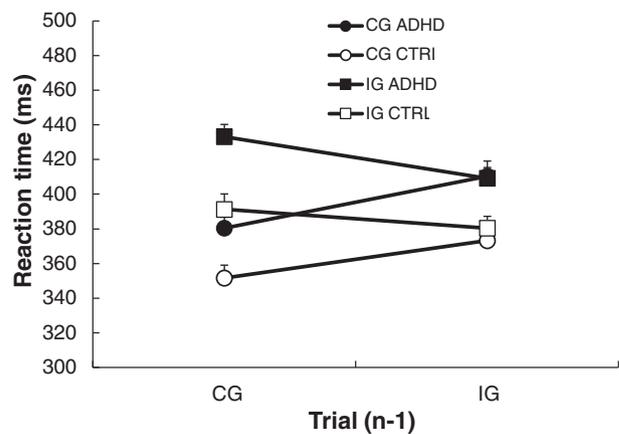


Fig. 4. Conflict adaptation effects. Group mean RT data for current trial compatibility are displayed depending on previous trial compatibility (CG = congruent, IG = incongruent) for control (in white) and ADHD (in black) groups. Error bars are mean standard errors.

the two first propositions. First, incorrect activation rates known to be an index of the expression of involuntary triggered erroneous responses were comparable in ADHD and control groups suggesting that ADHD patients were not more vulnerable to automatic activation of response than control adults. This finding was reinforced by data from distributional analysis of incorrect activation rates showing no difference between groups (see Fig. 3B). This suggests that even when taking account of partial errors, adults with ADHD were not more sensitive to impulse activation than controls. These results support previous data obtained in children with ADHD (Ridderinkhof et al., 2005). Second, analyses of RT distributions, and of the associated delta-plots, showed that the negative-going slopes of the delta-plots were similar for both groups of adults. In the context of the activation–suppression model (Ridderinkhof, 2002a), these findings indicate that adults with ADHD did not show a deficit in the selective inhibition of responses activated by stimulus position. Moreover, the correction rate, calculated from the partial errors defined by EMG activity, were similar for ADHD and control groups, suggesting that ADHD patients' ability to suppress impulses before they led to full performance errors was intact. Consistent with these results, data obtained from the Stop-task revealed no difference in the SSRT between groups suggesting a similar capacity to inhibit responses.

The study of the conflict adaptation effect is of great interest here, revealing the nature of the deficit. We observed a similar reduction of congruency effect after incongruent trials for both ADHD and control groups which indicates that the ability of adults with ADHD to refocus their attention after a challenging event is preserved. But after congruent trials, adults with ADHD presented a larger interference effect than the control group. The refocalization of attention being an effortful process, an enlarged interference effect after congruent trials reflects the relaxation of attention. The larger interference effect in the ADHD group hence suggests that these patients are less able to maintain an adequate level of attention focalization over time despite a preserved capacity to refocalise attention when necessary. Such a deficit in maintaining an appropriate level of attention over time may be the key explanation for performance of young adults with ADHD.

In this line, it is noticeable that most studies reporting larger interference effects in adults with ADHD also reported longer RTs in these patients (Bush et al., 1999; King et al., 2007; Lundervold et al., 2011; Soutschek et al., 2013). By contrast, one of the rare studies that did not report a significant difference between interference effects when comparing ADHD patients and control subjects also failed to report differences in RTs (Banich et al., 2009). Slower RTs have been found to be characteristic of ADHD responding in several different tasks (e.g., Alderson et al., 2007; Castellanos et al., 2005; Epstein et al., 2011; Johnson et al., 2007; Leth-Steensen et al., 2000; Lijffijt et al., 2005; Oosterlaan et al., 1998). Chronometric analyses indicated that motor times (MT) were not different between groups, refuting the idea that patients with ADHD are slowed at the motor execution level. Furthermore, all others indices, such as premotor time (PMT), incorrect activation latency (IAL) and correction time (CT) tended to be longer for adults with ADHD, suggesting that the slowing down affects different processes and is not restricted to some few specific stages. This fits with a dysfunctional sustained attention. If sustained attention level is lower in these patients, they will have more difficulties in quickly detecting the stimulus and all subsequent processes including incorrect activation correction in partial error trials will be delayed. Supporting this hypothesis, performance of adults with ADHD was impaired in the CPT task as compared to control adults, suggestive of deficits in sustained attention. Moreover correlations found between the Simon effect size and the mean intra-subject RT variability in the CPT task suggest that the patients presenting the largest Simon effect were also the most distracted in the CPT task, again favoring an underlying deficit in alertness and/or sustained attention. In the same line, some studies have shown that poorer information processing efficiency was found to be specific to ADHD (Rommelse et al., 2007; Salum et al., 2014).

Most of deficits associated with ADHD could be fully explained by a poor information processing due to a low sustained attention level.

To summarize, larger interference effects observed in adults with ADHD seems unlikely to reflect deficits such as higher sensitivity to external stimuli or lack of inhibition but seems to be due to difficulties in maintaining an appropriate level of sustained attention. This is at odds with data obtained in children with ADHD: Ridderinkhof et al. (2005), using delta plots, found deficits in response impulse suppression in children with ADHD, suggesting that deficits in executive function may partly differ between children and adults with ADHD. It is also notable that all adults participating in this study were medication-free for at least six months, meaning that even if they showed symptoms of ADHD, these symptoms did not prevent them to live without medication. It is possible that these patients had developed adaptive control strategies, explaining why the expression and inhibition of impulsivity were not affected in the Simon task. Another limitation of this study is the small sample sizes used which may have limited some statistical results, and especially since we discussed some null effects. But it should be noted that the conclusion that there was no difference between groups considering impulsivity and inhibition has been drawn from two different indices providing consistent findings. Nonetheless, these results need to be strengthened through further investigation.

5. Conclusions

Our results provided evidence that adults with ADHD did not present a greater degree of impulse expression, nor deficits in impulse suppression, when performing a Simon task. They did not present deficits in attention refocalization following challenging events neither. Instead, it seems that patients are less able in maintaining an appropriate level of sustained attention.

Acknowledgments

The authors warmly thank Jennifer Coull for their great help with English. This work was funded by the Centre National de la Recherche Scientifique (CNRS, France) and Aix-Marseille Université. It was also partly funded by the European Research Council under the European Community's Seventh Framework Program (FP7/2007–2013 Grant Agreement no. 241077).

References

- Alderson, R.M., Rapport, M.D., & Kofler, M.J. (2007). Attention-deficit/hyperactivity disorder and behavioural inhibition: a meta-analytic review of the stop-signal paradigm. *Journal of Abnormal Child Psychology*, 35, 745–758.
- Allain, S., Burle, B., Hasbroucq, T., & Vidal, F. (2009). Sequential adjustments before and after partial errors. *Psychonomic Bulletin Review*, 16, 356–362.
- American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th revised ed.). Washington, DC: American Psychiatric Association (DSM-IV-R).
- 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.A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin*, 121, 65–94.
- Biederman, J., & Faraone, S.V. (2005). Attention-deficit hyperactivity disorder. *Lancet*, 366, 237–248.
- Biederman, J., & Faraone, S.V. (2006). The effects of attention-deficit/hyperactivity disorder on employment and household income. *Medscape General Medicine*, 8, 12.
- Biederman, J., Newcorn, J., & Sprich, S. (1991). Comorbidity of Attention Deficit Hyperactivity Disorder with conduct, depressive, anxiety, and other disorders. *The American Journal of Psychiatry*, 148, 564–577.
- 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.
- Burle, B., Possamaï, 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.
- Burle, B., Allain, S., Vidal, F., & Hasbroucq, T. (2005). Sequential compatibility effects and cognitive control: does conflict really matter? *Journal of Experimental Psychology: Human Perception & Performance*, 31, 831–837.

- Burle, B., Spieser, L., Servant, M., & Hasbroucq, T. (2014). Distributional reaction time properties in the Eriksen task: marked differences or hidden similarities with the Simon task? *Psychonomics Bulletin & Review*, 21, 1003–1010.
- Bush, G., Frazier, J.A., Rauch, S.L., Seidman, L.J., Whalen, P.J., & Jenike, M.A. (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.S., Scheres, A., Di Martino, A., Hyde, C., & Walters, J.R. (2005). Varieties of attention-deficit/hyperactivity disorder-related intra-individual variability. *Biological Psychiatry*, 57, 1416–1423.
- Connors, C.K., & Staff, M.H.S. (2000). *Continuous Performance Test II: Computer Program for Windows Technical Guide and Software Manual*. 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, and Behavioural Neuroscience*, 7, 380–390.
- Egner, T., & Hirsch, J. (2005). Cognitive control mechanisms resolve conflict through cortical amplification of task-relevant information. *Nature Neuroscience*, 8, 1784–1790.
- Epstein, J.N., Langberg, J.M., Rosen, P.J., Graham, A., Narad, M.E., Antonini, T.N., et al. (2011). Evidence for higher reaction time variability for children with ADHD on a range of cognitive tasks including reward and event rate manipulations. *Neuropsychology*, 25, 427–441.
- Gratton, G., Coles, M.G., & Donchin, E. (1992). Optimizing the use of information: strategic control of activation of response. *Journal of Experimental Psychology: General*, 121, 480–506.
- Hasbroucq, T., Possamai, C.A., Bonnet, M., & Vidal, F. (1999). Effect of the irrelevant location of the response signal on choice reaction time: an electromyographic study in humans. *Psychophysiology*, 36, 522–526.
- Hedge, A., & Marsh, N. (1975). The effect of irrelevant spatial correspondences on two-choice response time. *Acta Psychologica*, 39, 427–439.
- Hodges, P.W., & Bui, B.H. (1996). A comparison of computer-based methods for the determination of onset of muscle contraction using electromyography. *Electroencephalography and Clinical Neurophysiology*, 101, 511–519.
- Hommel, B. (2011). The Simon effect as tool and heuristic. *Acta Psychologica*, 136, 189–202.
- Johnson, K.A., Kelly, S.P., Bellgrove, M.A., Barry, E., Cox, M., Gill, M., et al. (2007). Response variability in attention deficit hyperactivity disorder: evidence for neuropsychological heterogeneity. *Neuropsychologia*, 45, 630–638.
- King, J.A., Colla, M., Bras, 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.
- Kornblum, S. (1994). The way irrelevant dimensions are processed depends on what they overlap with: the case of Stroop- and Simon-like stimuli. *Psychological Research*, 56, 130–135.
- Kornblum, S., Hasbroucq, T., & Osman, A. (1990). Dimensional overlap: cognitive basis for stimulus–response compatibility—a model and taxonomy. *Psychological Review*, 97, 253–270.
- Leth-Steensen, C., Elbaz, Z.K., & Douglas, V.I. (2000). Mean response time, 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., & Engeland, H. (2005). A meta-analytic review of stopping performance inattention-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.
- Mayr, U., Awh, E., & Laurey, P. (2003). Conflict adaptation effects in the absence of executive control. *Nature Neuroscience*, 6, 450–452.
- Moura, H.F., Faller, S., Benzano, D., Szobot, C., von Diemen, L., Stolf, A.R., et al. (2013). The effects of ADHD in adult substance abusers. *Journal of Addictive Diseases*, 32, 252–262.
- Nigg, J.T. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin*, 127, 571–598.
- Oosterlaan, J., Logan, G.D., & Sergeant, J.A. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD, anxious and control children: a meta-analysis of studies with the stop-task. *Journal of Child Psychology and Psychiatry*, 39, 411–425.
- Palacio, J.D., Castellanos, F.X., Pineda, D.A., Lopera, F., Arcos-Burgos, M., Quiroz, Y.T., et al. (2004). Attention-deficit/hyperactivity disorder and comorbidities in 18 Paisa Colombian multigenerational families. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43, 1506–1515.
- Proctor, R.W., Lu, C.H., Wang, H., & Dutta, A. (1995). Activation of response codes by relevant and irrelevant stimulus information. *Acta Psychologica*, 90, 275–286.
- Ratcliff, R. (1979). Group reaction time distributions and analysis of distribution statistics. *Psychological Bulletin*, 86, 446–461.
- Reich, W. (2000). Diagnostic interview for children and adolescents (DICA). *Journal of American Academy of Child and Adolescent Psychiatry*, 39, 59–66.
- Ridderinkhof, K.R. (2002a). Activation and suppression in conflict tasks: empirical clarification through distributional analyses. In W. Prinz, & B. Hommel (Eds.), *Attention and Performance, Vol. XIX, Common Mechanisms in Perception and Action* (pp. 494–519). Oxford: Oxford University Press.
- Ridderinkhof, K.R. (2002b). Micro and macro-adjustments of task set: activation and suppression in conflict tasks. *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 ADHD that are eliminated by methylphenidate treatment. *Journal of Abnormal Psychology*, 114, 197.
- Rodríguez-Jiménez, R., Ponce, G., Monasor, R., Jiménez-Giménez, M., Pérez-Rojo, J.A., Rubio, G., et al. (2001). Validation in the adult Spanish population of the Wender Utah Rating Scale for the retrospective evaluation in adults of attention deficit/hyperactivity disorder in childhood. *Revista de Neurología*, 33, 138–144.
- Rommelse, N., Alting, M., de Sonneville, L., Buschgens, C., Buitelaar, J., Oosterlaan, J., et al. (2007). Are motor inhibition and cognitive flexibility dead ends in ADHD? *Journal of Abnormal Child Psychology*, 35, 957–967.
- Salum, G., Sergeant, J., Sonuga-Barke, E., Vandekerckhove, J., Gadelha, A., Pan, P., et al. (2014). Specificity of basic information processing and inhibitory control in attention deficit hyperactivity disorder. *Psychological Medicine*, 44, 617–631.
- Sergeant, J.A., Geurts, H., & Oosterlaan, J. (2002). How specific is a deficit of executive functioning for attention deficit/hyperactivity disorder? *Behavioural Brain Research*, 130, 3–28.
- Simon, J.R. (1969). Reactions toward the source of stimulation. *Journal of Experimental Psychology*, 81, 1974–1976.
- Simon, J.R. (1990). The effects of an irrelevant directional cue on human information processing. In R.W. Proctor, & T.G. Reeve (Eds.), *Stimulus-Response Compatibility: An Integrated Perspective* (pp. 31–86) (Amsterdam: North-Holland).
- Soutschek, A., Schwarzkopf, W., Finke, K., Hennig-Fast, K., Müller, H.J., Riedel, M., et al. (2013). Interference control in adult ADHD: no evidence for interference control deficits if response speed is controlled by delta plots. *Acta Psychologica*, 143, 71–78.
- Spencer, T.J., Biederman, J., & Mick, E. (2007). Attention-deficit/hyperactivity disorder: diagnosis, lifespan, comorbidities, and neurology. *Journal of Pediatric Psychology*, 32, 631–642.
- Spieser, L., van den Wildenberg, W.P.M., Hasbroucq, T., Ridderinkhof, K.R., & Burle, B. (2015). Controlling your impulses: electrical stimulation of the human supplementary motor complex prevents impulsive errors. *Journal of Neuroscience*, 35, 3010–3015.
- Stevens, M., Lammertyn, J., Verbruggen, F., & Vandierendonk, A. (2006). Tscope: a C library for programming cognitive experiments on the MS Windows platform. *Behavior Research Methods*, 38, 280–286.
- Stürmer, B., Leuthold, H., Soetens, E., Schröter, H., & Sommer, W. (2002). Control over location-based response activation in the Simon task: Behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance*, 28(6), 1345–1363.
- Taylor, C.J., & Daniel, C.M. (1997). Neuropsychological assessment of attention in ADHD adults. *Journal of Attention Disorders*, 2, 77–88.
- Ullsperger, M., Bylsma, L.M., & Botvinick, M.M. (2005). The conflict adaptation effect: it's not just priming. *Cognitive, Affective, and Behavioural Neuroscience*, 5, 467–472.
- Van Boxtel, G.J.M., Geraats, L.H.D., Van den Berg-Lenssen, M.M.C., & Brunia, C.H.M. (1993). Detection of EMG onset in ERP research. *Psychophysiology*, 30, 405–412.
- Van den Wildenberg, W.P.M., Wylie, S.A., Forstmann, B.U., Burle, B., Hasbroucq, T., & Ridderinkhof, K.R. (2010). To head or to heed? Beyond the surface of selective action inhibition: a review. *Frontiers in Human Neuroscience*, 4, 222.
- Vincent, S.B. (1912). The function of the vibrissae in the behavior of the white rat. *Behavioral Monograph*, 1(5).
- 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.
- Winer, B.J. (1970). *Statistical Principles in Experimental Design*. London: Mc Graw-Hill.
- Wylie, S.A., Ridderinkhof, R., Elias, W.J., Frysinger, R.C., Bashore, T.R., Downs, K.E., et al. (2010). Subthalamic nucleus stimulation influences expression and suppression of impulsive behavior in Parkinson's disease. *Brain*, 133, 3611–3624.
- Wylie, S.A., van den Wildenberg, W., Ridderinkhof, K.R., Claassen, D.O., Wooten, G.F., & Manning, C.A. (2012). Differential susceptibility to motor impulsivity among functional subtypes of Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, 83, 1149–1154.