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Performance monitoring is altered in adult ADHD: a familial event-related potential investigation

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Abstract

Background—Attention deficit hyperactivity disorder (ADHD) is a common neurodevelopmental disorder that starts in childhood and frequently persists in adults. Electrophysiological studies in children with ADHD provide evidence for abnormal performance monitoring processes and the familiarity of these processes. It is not yet known if this abnormal processing is developmentally stable and shares familial influences with ADHD throughout the lifespan.

Method—We aimed to investigate event-related potential (ERP) indices of performance monitoring and their familiarity in adults with ADHD. An arrow flanker task was presented to 21 adults with ADHD, 20 first-degree relatives (fathers) of children with ADHD and 20 controls.

Results—Compared to the controls, both adults with ADHD and parents of children with ADHD displayed significantly weaker error and conflict monitoring, as indexed by the smaller error negativity (Ne) and the N2 components. These two components were highly correlated within each of the three groups ($r = 0.53 - 0.65$). The groups did not differ on the error positivity (Pe).

Conclusions—These findings closely resemble those previously found in children with ADHD, suggesting that abnormal conflict monitoring and early error processing is developmentally stable in ADHD and shares familial influences with ADHD in adults. The relationship between different indices of performance monitoring may suggest partly common underlying mechanisms or modulators.

Keywords

Action monitoring; ADHD; adult ADHD; ERP; error negativity; error positivity; N2; endophenotype

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Introduction

Attention deficit hyperactivity disorder (ADHD) is a childhood-onset, neurodevelopmental disorder, which frequently persists into adulthood, with around 15% meeting full criteria for ADHD at the age of 25 years (Faraone et al., 2006). ADHD is highly heritable with twin studies indicating that approximately 76% of phenotypic variance is accounted for by genetic influences (Faraone et al., 2005). Further evidence comes from family studies (Thapar et al., 1999; Faraone et al., 2005), which show increased rates of ADHD in all closely related family members of affected probands, including siblings (Faraone et al., 1991; Biederman et al., 1992) and parents (Biederman et al., 1992; Biederman et al., 1991). Functional candidate gene studies focusing on dopamine and related neurotransmitter pathways find convincing evidence for association with genetic variants within or close to the dopamine D4 and D5 receptor genes (Li et al., 2006) and suggestive evidence for a number of other genes, including the dopamine transporter (Thapar et al., 2007).

The relationship from genes to brain to behaviour is likely to be complex, with the effect of any single gene on behaviour expected to be small. The search for associations with neurobiological intermediate phenotypes or endophenotypes, which reflect more closely the underlying neurobiological mechanisms, has two potential advantages. First, it is feasible that specific genes may show greater effects in endophenotypes than behavioural phenotypes, providing improved measures for new gene discovery. Second, the study of endophenotypes is an essential step in elucidating the cognitive and neurobiological mechanisms that mediate genetic effects on behaviour (Gottesman & Gould, 2003; Tsuang & Faraone, 2000).

In ADHD, performance monitoring is a good candidate endophenotype as it has been associated with ADHD in numerous studies (see (Kuntsi et al., 2006) for a review) and is linked to dopaminergic functioning (Kramer et al., 2007; Frank et al., 2007; Holroyd & Coles, 2002; de Bruijn et al., 2006; de Bruijn et al., 2004; Zirnheld et al., 2004) in brain regions that have been implicated in ADHD (Carter et al., 1998; Gehring & Knight, 2000; Paloyelis et al., 2007). The process of performance monitoring is an essential prerequisite for adaptively altering behavior and decision making, and comprises error detection and conflict monitoring, functions that can be measured by their neurophysiological correlates (event-related potentials or ERPs). An ERP component that is associated with performance monitoring is the N2, a fronto-central negative amplitude that occurs between 200 and 400 ms after stimulus onset. The N2 was originally thought to index response inhibition (Falkenstein et al., 1999) as in go/no-go tasks there is an N2 enhancement in the no-go condition. More recent studies suggest that the N2 reflects a more general performance monitoring process, independent of response inhibition (Nieuwenhuis et al., 2003; Donkers & van Boxtel, 2004). Studies using continuous performance or go/no-go-tasks in children and adults with ADHD did not find differences in N2 between participants with ADHD and controls (Banaschewski et al., 2004; Overtom et al., 1998; Fallgatter et al., 2004). Yet tasks requiring a higher level of conflict monitoring, such as the stop task and flanker task, have elicited diminished N2 amplitudes or topographic N2 alteration in children with ADHD (Brandeis et al., 1998; Pliszka et al., 2000; Albrecht et al., 2005; Albrecht et al., 2008). The abnormality in conflict monitoring processes, as indexed by the N2, is therefore only elicited when there are increased demands on these processes. A recent investigation indicated a familial association between ADHD and the N2 in children, suggesting that the N2 may mediate genetic effects on ADHD behaviours (Albrecht et al., 2008). To date this has only been explored in relation to childhood ADHD, so it is not yet clear whether this represents a developmentally stable trait found in ADHD across the lifespan.

An erroneous response, in healthy individuals, is associated with a component called the error-related negativity (ERN) (Gehring et al., 1993) or the error-negativity (Ne) (Gehring et al., 1990). The specific functional significance of the Ne is still under debate. It may reflect mismatch (Gehring et al., 1993) or response conflict (Carter et al., 1998) between error and required responses. A number of studies have investigated the functional relationship between the Ne and the N2: while some suggest that they represent distinct neurophysiological processes (Falkenstein et al., 1999), (Ridderinkhof et al., 2002), others suggest they represent the same process of conflict monitoring (Yeung & Cohen, 2006). An additional component associated with error monitoring, the error positivity (Pe), has a more posterior distribution and is elicited after the Ne (Falkenstein et al., 1995). Although far less research has addressed the function of the Pe, it is elicited, unlike the Ne, only after full errors of which the subject is aware, which suggests that it represents conscious error-recognition processes (Nieuwenhuis et al., 2001; Hajcak et al., 2003; O'Connell et al., 2007).

Recent ERP studies have indicated abnormalities in the Ne and Pe in children with ADHD, but these processes have yet to be investigated in adult ADHD. In three studies that used stop signal and arrow flanker tasks, early error detection (Ne) was reduced in children with ADHD compared to controls (Liotti et al., 2005; van Meel et al., 2007; Albrecht et al., 2008). Further, initial evidence from the study of siblings of ADHD probands indicated shared familial influences on the Ne and ADHD (Albrecht et al., 2008). In contrast, two investigations using an arrow flanker task and a go/no-go task respectively, indicated normal Ne but deviant later error detection (Pe) in children with ADHD (Jonkman et al., 2007; Wiersma et al., 2005). Yet another study, using an oddball task, observed an enhanced Ne in children with ADHD, but comparable Pe (Burgio-Murphy et al., 2007). The contrasting findings have been attributed to the sensitivity of the Ne and Pe components to task-specific factors, such as task difficulty, the definition of an error in each of the studies (Jonkman et al., 2007) and differences in the number of error trials used in computation of these components (Jonkman et al., 2007). Overall, there is evidence for abnormal error monitoring in childhood ADHD, but the contrasting findings indicate that methodological differences between studies influence the extent that this abnormality is elicited.

The overall aim of this study was to investigate the familiarity of the ERP indices of performance monitoring deficits in ADHD and their persistence into adulthood. Using an arrow flanker task that provides adaptive feedback to ensure comparable accuracy in all participants (Albrecht et al., 2008), we addressed methodological shortcomings of some of the previous research while studying the key processes in a sample of adults with ADHD, first-degree relatives of ADHD probands (parents of children with an ADHD diagnosis) and healthy adult controls. To examine the performance accuracy in the two groups, we compared errors and reaction time in both congruent and incongruent conditions. We tested two main hypotheses. First, we predict that, based on previous findings in children using an identical arrow flanker task (Albrecht et al., 2008), adults with ADHD will have attenuated Ne but normal Pe components. This would indicate the presence of the same deficits in adults with ADHD as that seen in children with ADHD and suggest developmental stability of these processes when investigated under identical conditions. If the findings are similar when tested under identical conditions, it would also suggest that abnormalities in indices of error monitoring in ADHD are influenced by task-specific conditions. Further, we predict that the N2 component will be enhanced in the incongruent compared to the congruent conditions of this task and that this enhancement will be reduced in the ADHD participants compared to the controls, suggesting that conflict monitoring is abnormal in adult ADHD. Second, as parents of children with ADHD share 50% of their genes with their affected offspring, we hypothesise that the parents of children with ADHD will be significantly different from controls in these cognitive-neurophysiological parameters, indicating a

familial association between these parameters and ADHD in adults. Additionally, given the uncertainty regarding the extent to which the N2 and the Ne may reflect distinct or common underlying mechanisms (Falkenstein et al., 1999; Ridderinkhof et al., 2002; Yeung & Cohen, 2006), we aimed to investigate the relationship between these components for the task used here.

Methods and Materials

Sample

21 male adults with ADHD, 20 male fathers of children with ADHD and 20 male healthy control adults participated in this study on the basis of informed consent. The joint South London and Maudsley and the Institute of Psychiatry NHS Research Ethics Committee approved this study (086/05). Age range was 18 to 56 years, with a mean age of 32.51 (SD=5.84) for the ADHD group, 45.90 (SD=4.15) for the parent group and 30.00 (SD=6.51) for the control group. A one-way ANOVA indicated a significant main effect of age [$F(1, 59) = 53.87, p < 0.001$] with post-hoc analyses showing no significant difference between the probands and controls [$p = 0.48$] but significant differences between the probands and parents [$p < 0.001$] and controls and parents [$p < 0.001$]. All participants had an IQ of 80 or above on the Wechsler Adult Intelligence Scale (WAIS-II) (Wechsler, 1997), with mean IQs of 118 (SD=10.00) for the ADHD group, 121 (SD=13.37) for the parent group and 122 (SD=12.10) for the control group, with no main effect of group on IQ [$F(2, 58) = 0.67, p = 0.52$].

Adults with ADHD were recruited from the National Adult ADHD Clinic at the Maudsley Hospital, where they had received diagnosis from a consultant psychiatrist who specialises in adult ADHD, following an in-depth clinical and psychological assessment. For the purposes of this study, diagnostic criteria for DSM-IV ADHD were applied using clinical interview data that enquired about each of the 18 ADHD symptom items in childhood and adulthood. In addition, individuals were only included if either the proband or an informant reported six or more DSM-IV items for both the hyperactive-impulsive and inattentive subscales in childhood, using the Barkley Adult ADHD rating scale for retrospective recall of childhood symptoms (Barkley & Murphy, 2005) and, in addition, six or more inattentive items from the Barkley Adult ADHD rating scale for current symptoms (Barkley & Murphy, 2005). Participants included in the study fulfilled criteria for DSM-IV combined subtype ADHD in childhood and either combined type ($n = 17$) or inattentive type ($n = 4$) as adults.

Exclusion criteria for the ADHD group included the presence of an Axis I or II co-morbid psychiatric diagnosis and taking any psychoactive medication other than stimulant medication for treatment of ADHD. A minimum of 48 hours medication-free period was required prior to the assessments. All participants were right handed, as determined by preferred writing hand, and had normal or corrected-to-normal vision.

The parent group was recruited from a database of families who had previously participated in the International Multicenter ADHD genetics project (IMAGE). All of the participating fathers had a biological child who received a research diagnosis of DSM-IV combined subtype ADHD (Chen et al., 2008). None of the fathers had a major psychiatric condition, history of substance abuse or previous head injury. Self-report data were collected on current and retrospective ADHD symptoms, using the Barkley Adult ADHD rating scales (Barkley & Murphy, 2005). One father had a rating scale diagnosis of adult ADHD, but was not excluded from the analyses, as the control samples were unselected for ADHD; the use of unselected samples enables unbiased estimates of the familial association between ADHD and secondary measures (Andreou et al., 2007).

Control participants were selected from a database of volunteers at the Institute of Psychiatry. They were selected if they had no major psychiatric conditions, substance abuse or previous head injury, and were matched with ADHD participants on age and gender. Self-report data were collected on current and retrospective ADHD symptoms, using the Barkley Adult ADHD rating scales. Based on the rating scale data, one of the control participants had above-threshold symptoms for the inattentive subtype in the current ratings and two had symptoms sufficient to qualify for combined subtype from the retrospective ratings. These individuals were not excluded from the analysis as they were only above threshold on self-report scales, they had never sought treatment for their symptoms and did not consider themselves impaired. Furthermore, as stated above, the use of unselected samples is beneficial for the estimation of familial associations. When tested, these individuals were not outliers on any of the ERP or performance variables and excluding them from analyses did not change any of the results. The mean score for the controls on the ADHD behaviour ratings was 8.70 (SD=8.30) for current and 5.90 (SD=5.11) for retrospective symptoms. For the ADHD group, the mean scores were 42.47 (SD=7.62) for current and 22.00 (SD=3.52) for retrospective symptoms. For the parent group, the mean scores were 12.10 (SD=8.81) for current and 7.90 (SD=6.84) for retrospective symptoms. The small difference in ADHD symptom scores between the parents and controls was not significant for either the current [$F(1,39)=1.58$, $p=0.22$] or retrospective [$F(1,39)=1.10$, $p=0.30$] ratings.

Task and stimuli

The flanker task was based on the Eriksen flanker paradigm (Eriksen & Schultz, 1979) (Figure 1) and consisted of ten blocks of 40 trials. Columns of black arrowheads (equilateral triangles with 18 mm edge length at 3 positions with 23 mm distance centre to centre) were presented in the centre of a computer monitor, against light grey background at 120 cm viewing distance. On every trial, the fixation mark in the centre of the screen was replaced by the stimuli. The flankers (two arrowheads pointing to the same direction above and below the position of the fixation mark) were presented 100 ms before the target arrowhead appeared between the flankers (for another 150 ms). Participants were seated on an adjustable chair in an acoustically shielded, video-monitored room and had to press response buttons with the index-finger of their hand corresponding to the direction indicated by the target.

On congruent trials, flanker and target arrowheads pointed in the same direction; on incongruent trials, they pointed in opposite directions. A trial was presented every 1650 ms, and the task took approximately 13 minutes. Congruent versus incongruent and the direction of responses (left versus right) were counter-balanced and randomised. Written feedback was given at the end of each block so that both speed and accuracy were emphasised. This meant that if participants made more than 10% errors on congruent or more than 40% errors on incongruent trials, they were instructed to slow down. In case of less than 10% errors in the congruent and less than 40% errors in incongruent trials, they were instructed to perform faster; otherwise, the participant was told to continue in exactly the same way. Two practice blocks with 24 trials each were administered before the real task and comprehension ascertained prior to task performance. Where necessary, participants were told to minimise eye movements or blinks. The task was run in between two other tasks (not reported here).

Scoring overt performance

Performance measures for congruent and incongruent stimuli were number of errors, target reaction time (MRT, i.e. mean latency of responding in ms after target onset), within-subject variability in reaction times (SD-RT), and the coefficient of reaction time variability (CV, i.e. SD-RT/MRT).

ERP recording and processing

The ERPs were recorded with a sample rate of 500 Hz and cut-off frequencies of 0.1–30 Hz via Nihon Kohden Ag/AgCl cup electrodes (impedances kept below 5 kOhm) fixed to the scalp with electrolyte gel at electrode positions, which included the 19 standard electrodes of the 10–20 system, FCz as recording reference, and a ground electrode placed at the forehead using calibrated technical zero baselines and a Neuroscan recording system. Vertical and horizontal electrooculograms (EOGs) were simultaneously recorded from electrodes above and below the left eye and at the outer canthi. The EEG was analysed using Brainvision Analyzer (Version 1.05) and after down-sampling to 256 Hz, was corrected for horizontal and vertical (blinks) eye movements using the Gratton and Coles method (Gratton et al., 1983). Trials with remaining artifacts exceeding $\pm 100 \mu\text{V}$ in any channel were rejected from the digitally lowpass-filtered (0.1 to 30 Hz, 24 dB/oct) data before averaging. All trials were inspected visually to detect additional subtle artifacts. Segments were averaged separately for each participant in three different response conditions: (1) stimulus-locked incongruent correct trials, (2) stimulus-locked congruent correct trials, and (3) response-locked incongruent incorrect trials. All averages were free from residual artifacts and contained a minimum of 20 accepted sweeps. The ERPs were transformed to the average reference for all subsequent computations (Lehmann, 1987). Maps of the topographical scalp distribution of electrical brain activity were spline interpolated between the electrode locations. Calibrated zero baselines were used (instead of prestimulus-baseline corrections) to avoid distorting the map topographies (Brandeis & Lehmann, 1986; Lehmann, 1987).

Statistical analyses

For the analysis of performance data, measures of error rates and intra-individual reaction time variability, SD-RT and CV, had pronounced heterogeneity of variance and skewed distributions therefore, these data were transformed using square root (errors) and inverse transforms (SD-RT and CV). Along with MRT, these data were analysed using repeated measures analyses of variance (group-by-congruency).

Inspection of the grand average waveforms revealed that both the effect of congruency on N2 components and the error-related negativity (Ne) were maximal at fronto-central electrodes (Figures 2 and 3). We analysed stimulus-locked N2 peaks scored at Fz 200–400 ms after the stimulus onset of correctly responded trials with ANOVA (factors: group, site (Fz, FCz) and congruency). One ADHD participant was excluded from the N2 analyses due to excessive movements. The average number of sweeps for the N2 in the control group was 183.75 (SD=12.22) for congruent stimuli and 153.05 (SD=10.79) for incongruent stimuli; in the ADHD group these were 161.76 (SD=42.87) for congruent stimuli and 134.86 (SD=39.83) for incongruent stimuli; and in the parent group 183.40 (SD=19.40) for congruent stimuli and 156.75 (SD=18.19) for incongruent stimuli. The N2 latency data were skewed and no transformations were successful (cubic, square, identity, square root, log, 1/square root, inverse, 1/square, 1/cubic); as such, these data were analysed with Generalized Estimating Equations (GEE) in a repeated measures design (group $\times$ congruency). GEE models estimate averages rather than the entire distribution of values, and hence are less restricted by distributional assumptions than other approaches to repeated measures analysis. This approach accounts for the correlation in performance on the two tasks; specifically, an exchangeable correlation structure was assumed to account for the within-subject correlation. This allowed the implementation of a group by congruency interaction to test group differences between conditions. Further information about this method is available in (McLoughlin et al., 2008). In order to test overall effects of group and interaction terms, we used the Wald chi2 test. We calculated effect sizes (d) for these data between ADHD and control participants using the difference of the marginal means from the GEE model, divided by the pooled standard deviation of the raw data.

The Ne was defined as the most negative peak 0–150 ms after an erroneous response on incongruent trials with respect to the preceding positivity (PNe, –100–20 ms), in order to obtain a more robust measure of this component (Nieuwenhuis et al., 2001; Falkenstein et al., 2001; Albrecht et al., 2008). The Ne was maximal at FCz. As the Ne data (latency and amplitude) were skewed and it was necessary to analyse the peak-to-peak (PNe-to-Ne) differences in a repeated-measures design, we used GEE analysis. Effect sizes for these GEE analyses were calculated as before. The Pe was the maximal positive peak between 200 and 500 ms at Cz. Amplitude and latency data of the Pe were analysed using a nonparametric Kruskal-Wallis test. As it is imperative to have sufficient number of trials without EEG artifacts to calculate the error-related components (>20), five participants were excluded from the ADHD group and one from the parent group due to insufficient number of error trials (<20). The average number of sweeps for the Ne and the Pe was 33.30 (SD=10.22) in the control group, 27.90 (SD=18.43) in the ADHD group and 27.55 (SD=12.14) in the parent group.

As IQ did not differ between groups, we did not include it in subsequent analyses. However, as age significantly differed between groups, we initially included as a covariate in all analyses and only report it when it was significant, as we dropped it from the analyses otherwise. We adopted a significance level of $p<0.05$ (two-tailed) throughout the analyses and report trends ($p=.09$).

Results

Performance data

Repeated measures analyses of variance indicated no overall group effect in terms of errors committed [$F(2, 57)=1.10$, $p=0.34$]. The data showed a significant effect of congruency [$F(1, 57)=434.75$, $p<0.0001$], with more errors being committed in incongruent than congruent trials (Table 1), yet no group-by-congruency interaction [$F(2, 57)=0.35$, $p=0.71$]. For MRT, we found a trend for group differences [$F(2, 57)=3.09$, $p=0.05$] and post-hoc analyses indicated a significant difference between ADHD participants and controls ($p=0.02$), a trend for a difference between parents and controls ($p=0.09$) but no difference between ADHD participants and parents ($p=0.52$). A strong effect of congruency on MRT emerged, with reaction times being longer for incongruent trials [$F(1, 57)=974.66$, $p<0.0001$; Table 1]. No significant interaction emerged between group and congruency [$F(2, 57)=1.33$, $p=0.27$].

Group differences emerged for SD-RT [$F(2, 57)=5.69$, $p<0.006$] with post-hoc analyses indicating a significant difference in this measure between ADHD participants and controls [$p<0.0005$] and a trend for a difference between ADHD participants and parents [$p=0.09$], yet no difference between controls and parents [$p=0.83$]. For SD-RT, we did not find an effect of congruency [$F(1, 57)=0.04$, $p=0.84$] or a group-by-congruency interaction [$F(2, 57)=0.67$, $p=0.51$]. Similarly, for CV a main group effect emerged [$F(2, 57)=5.90$, $p<0.005$] with significant differences between ADHD participants and controls [$p=0.01$] and parents [$p=0.02$] but no differences between these latter groups [$p=1.00$]. For CV, we found an increase in this measure for congruent trials [$F(1, 57)=61.80$, $p<0.0001$] but no group $\times$ congruency interaction [$F(2, 57)=1.69$, $p=0.19$].

ERP data

Ne and Pe—GEE analysis indicated a main group effect for Ne amplitude measured peak-to-peak. [Wald $\chi^2(2)=7.52$, $p=0.02$, $d=0.59$] with higher amplitude in the control participants compared with the ADHD group [$z=2.72$, $p=0.007$] and the parents [$z=2.09$, $p=0.04$] (Table 2). Parents and ADHD participants did not significantly differ from each

other in amplitude of the Ne [$z=0.31$, $p=0.76$]. ANOVA indicated no significant group effects on the latency of the Ne [$F(2,45)=1.01$, $p=0.37$] (Table 2 and Figure 2). A Kruskal-Wallis test indicated no main group effect on the amplitude of the Pe component [$H(2)=2.65$, $p=0.27$, $d=0.11$] and no significant differences in latency between groups [$H(2)=3.96$, $p=0.14$, $d=0.44$; Table 2].

N2—The amplitude of the N2 was enhanced for incongruent compared to congruent items [$F(1,57)=45.72$, $p<0.001$; Table 3]. Further, this N2 congruency effect differed between groups [congruency $\times$ group, across both Fz and FCz; $F(1,57)=3.27$, $p=0.04$], being more pronounced in control participants than both ADHD ($p=0.02$) and parents ($p=0.001$) (Table 3). Parents and ADHD participants did not significantly differ from each other in N2 enhancement ($p=0.92$). The N2 enhancement was higher at Fz compared with FCz [$F(1,57)=33.49$, $p<0.0001$] and peaked between 311 and 361 ms for all groups (Table 3 and Figure 3). For latency of the N2, no main group effect [Wald $\chi^2(2)=1.35$, $p=0.51$, $d=0.38$] or a significant congruency $\times$ group interaction emerged [Wald $\chi^2(2)=0.43$, $p=0.81$]. The N2 peaked earlier for incongruent compared to congruent stimuli [$z=3.91$, $p<0.0001$, $d=0.66$; Table 3] and age was significant as a covariate [$z=2.11$, $p=0.03$].

N2 and Ne—Spearman's nonparametric correlation, with age partialled out, indicated significant relationships between the Ne and the N2 for controls [$r=0.65$, $p=0.003$], ADHD participants [$r=0.53$, $p=0.02$] and parents [$r=0.48$, $p=0.04$] and for all groups together [$r=0.58$, $p<0.0001$]. In order to test if this relationship was specific to the N2 and the Ne, we conducted additional correlational analysis between the N2 and the Pe, and the Ne and the Pe. Nonparametric correlations, with age partialled out, revealed no significant relationship between N2 and Pe for controls [$r=0.31$, $p=0.23$], ADHD participants [$r=-0.16$, $p=0.58$] or parents [$r=0.06$, $p=0.85$] or all groups together [$r=-0.04$, $p=0.80$]. Similarly, nonparametric correlations detected no relationship between Ne and Pe for controls [$r=0.16$, $p=0.53$], ADHD participants [$r=-0.45$, $p=0.09$] or parents [$r=0.43$, $p=0.09$] or all groups together [$r=0.17$, $p=0.24$].

Discussion

In a comparison of 21 adult males with ADHD, 20 age- and gender-matched controls and 20 fathers of children with ADHD, we investigated neurophysiological parameters of performance monitoring. The findings are strikingly similar to those found in children with ADHD and their siblings using an identical task (Albrecht et al., 2008). This indicates that the same deficits seen in children are also found in adults with ADHD, suggesting developmental stability and familiarity of abnormal performance monitoring across the lifespan.

The adaptive feedback procedure used in this version of the flanker task kept response accuracy constant at a pre-designated level; as such we were able to avoid confounds with speed-accuracy trade-off, equalising error rates between groups. A congruency effect emerged for both errors and RT, with more errors and slower RTs in the incongruent condition. Significant differences between the groups emerged for measures of RT variability measures, in that adults with ADHD were more variable in their responding than both parents and controls. As parents with ADHD were intermediate in their performance to ADHD participants and controls, these findings agree with previous research which suggests the potential of RT variability as an endophenotype for ADHD (Kuntsi & Stevenson, 2001).

As predicted, Ne but not Pe was attenuated in the ADHD group compared to controls, which indicates abnormal initial error detection processes in adult ADHD. This is the first investigation, to our knowledge, into error monitoring in adult ADHD. It is in striking

agreement with similar investigations in childhood ADHD (Liotti et al., 2005; van Meel et al., 2007), most notably Albrecht et al. (2008) using identical procedures. Fathers of children with ADHD, although similar to controls in their self-rated behaviour, were significantly different from controls and more similar to the ADHD group in impairment in their error processing as indexed by the Ne. The familial association suggests that this abnormality is a trait that is influenced by susceptibility genes for ADHD. It is therefore likely that, as in children, the Ne indexes a genetically influenced endophenotype of adult ADHD.

Conversely, we did not observe a reduction of the Pe in adults with ADHD, yet the moderate effect size of 0.44 suggests that this might be significant given a larger sample size. The effect, though, is much smaller than that found for the Ne and the similarity in the pattern of findings to Albrecht et al. (2008) provides some evidence that the inconsistencies of previous findings in error monitoring in ADHD may be due to the sensitivity of these components to task-specific factors, such as emphasis on speed versus accuracy and error rate. We also observed the predicted group differences for conflict monitoring: an N2 enhancement for incongruent stimuli was highest in the control group, with attenuated amplitudes in both the ADHD and the parent groups. This suggests that, similar to the Ne, the N2 may also be a fruitful endophenotype for adult ADHD, as well as childhood ADHD (Albrecht et al., 2008).

The high correlations between the Ne and N2 suggest possible common influences on these processes. The Ne and the N2 components share sources in the anterior cingulate cortex (ACC) (Carter et al., 1998; Gehring & Knight, 2000), an area previously associated with both child and adult ADHD (Bush et al., 1999; Seidman et al., 2006). Although this area is functionally and structurally complex, it is involved in reward-based decision making (Bush et al., 2002) and is thought to be part of a lower level arousal system (Critchley et al., 2002). In light of the inconsistencies in the findings on performance monitoring in ADHD and the evidence of a possible common mechanism underlying both Ne and N2, we need also to consider the possibility that error and conflict monitoring deficits in ADHD could both be dependent on motivational and arousal states, which can be modified by task-specific conditions. Previous research has shown that factors that increase motivation or arousal, such as event rate (the presentation rate of stimuli) or rewards, can improve the performance of children with ADHD (Andreou et al., 2007; Konrad et al., 2000; Slusarek et al., 2001). There is evidence that the Ne is subject to influences by monetary incentives (Pailing & Segalowitz, 2004; Hajcak et al., 2005) but further research is required into separating the effects of these factors on performance monitoring in ADHD.

Since the Ne is linked to dopaminergic functioning (Kramer et al., 2007; Holroyd & Coles, 2002), these findings suggest a possible underlying neural mechanism for the familial influences on abnormal error monitoring in ADHD. Indeed, the dopamine D4 receptor (DRD4), which is a risk allele for ADHD (Thapar et al., 2007), has shown distinct effects on error monitoring processes (Kramer et al., 2007). The N2 has been linked to the COMT polymorphism (Kramer et al., 2007), previously identified as a possible risk allele for ADHD (Bellgrove et al., 2005) and, similar to the Ne, is related to dopaminergic functioning (Kramer et al., 2007).

To ensure the homogeneity of the sample and minimise the impact of potential confounding conditions, the participants with ADHD were selected to have no major comorbidities and we included males only. This highly selected group had slightly higher than expected IQs, yet they were well matched with the controls and parent group for IQ. Future studies are required to confirm these findings in more typical ADHD samples. To test if these ERP abnormalities are specific to ADHD, further studies are needed to investigate if they distinguish this disorder from other conditions, including overlapping neurodevelopmental

disorders and behavioural problems. The parent comparison sample was significantly older than both ADHD participants and controls; although age was not significant as a covariate for any measure, the results indicating familiarity on these processes need to be replicated in age-matched samples.

In conclusion, in an investigation of ERP indices of performance monitoring in adult ADHD, we found a similar profile of altered processing deficits as previously identified in children with ADHD (Albrecht et al., 2008), indicating persistence across the lifespan. We also obtained evidence of familial influences on these processes in the parents of children with ADHD. Future research should include the longitudinal follow-up of these measures from childhood to adulthood, to specifically map the trajectory of these abnormalities in individuals with ADHD. Further research is also required to investigate the possibility of a common underlying neural mechanism affecting both the Ne and N2 and their relationship to other cognitive and neural processes implicated in ADHD, as well as investigating the role of specific genes in the association between ADHD and performance monitoring deficits.

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Figure 1.

Flanker arrowheads preceded the presentation of the central target arrowheads by 100 ms. Condition was either congruent or incongruent.

Figure 2. Response-locked error-related components of control participants (red), parents (green) and ADHD participants (black) with maps of error negativity (top) and error positivity bottom), plus t-maps for group comparisons (Controls versus ADHD participants and fathers, respectively).

Figure 3. Stimulus-locked N2 to incongruent correct responses. Response-locked averages of control (red), ADHD (black) participants and parents (green) at the respective group mean latency, plus t-maps for group comparisons (Controls versus ADHD participants and fathers, respectively).

Table 1

Means and standard deviations for performance data prior to transformations from the flanker task

	Controls (n=20)	Parents (n=20)	ADHD (n=21)
Errors, mean (SD)			
Congruent trials	4.35 (4.61)	3.30 (3.53)	5.15 (5.51)
Incongruent trials	35.70 (9.29)	30.55 (12.55)	35.10 (17.94)
MRT, mean (SD)			
Congruent trials	285.35 (34.53)	311.09 (31.38)	327.42 (78.40)
Incongruent trials	373.09 (42.13)	405.52 (38.84)	410.60 (74.87)
SD-RT, mean (SD)			
Congruent trials	65.45 (22.88)	70.59 (31.23)	95.12 (55.43)
Incongruent trials	63.00 (20.52)	71.79 (23.34)	93.99 (47.89)
CV, mean (SD)			
Congruent trials	0.23 (0.06)	0.23 (0.08)	0.28 (0.09)
Incongruent trials	0.17 (0.04)	0.17 (0.05)	0.22 (0.07)

MRT: mean reaction time in ms

SD-RT: within-subject variability in RTs in ms

CV: coefficient of variation (SD-RT/MRT)

Cohen's d=comparison between ADHD and control participants

Table 2

Mean amplitude (in μV) and latency (in ms) of response-locked components of error processing for controls, parents of children with ADHD and adults with ADHD.

	Controls (n=19)	Parents (n=16)	ADHD (n=16)
	Mean (SD)	Mean (SD)	Mean (SD)
Error Negativity at FCz			
Latency (ms)			
PNe	-1.09 (11.03)	-10.50 (33.20)	-19.79 (33.39)
Ne	79.36 (25.91)	96.19 (14.75)	85.42 (16.23)
Amplitude (μV)			
PNe	-0.11 (1.94)	0.85 (2.36)	-0.22 (2.36)
Ne	-9.05 (5.62)	-5.46 (4.61)	-5.05 (2.74)
Error Positivity at Cz			
Latency (ms)	266.65 (86.11)	288.82 (63.75)	275.52 (76.32)
Amplitude (μV)	8.36 (4.34)	5.71 (2.76)	6.60 (3.57)

PNe: positive peak preceding Ne; Cohen's d =comparison between ADHD and control participants

Table 3

Mean amplitude (in μV) and latency (in ms) of stimulus-related N2 to congruent and incongruent correct responses for controls, parents of children with ADHD and adults with ADHD.

Stimulus-locked N2	Controls (n=20)	Parents (n=20)	ADHD (n=21)
	Mean (SD)	Mean (SD)	Mean (SD)
Latency at Fz (ms)			
Congruent correct	329.69 (35.72)	361.91 (48.76)	345.61 (32.30)
Incongruent correct	311.72 (26.81)	337.50 (36.85)	321.24 (39.12)
Amplitude at Fz (μV)			
Congruent correct			
Fz	-3.38 (2.81)	-0.54 (2.11)	-1.97 (2.30)
FCz	-0.21 (2.27)	0.84 (2.10)	0.03 (2.13)
Incongruent correct			
Fz	-5.04 (2.89)	-1.52 (2.30)	-2.72 (2.94)
FCz	-3.37 (3.37)	-1.23 (2.88)	-1.30 (2.43)

Cohen's d =comparison between ADHD and control participants