Performance monitoring and post-error adjustments in adults with attention-deficit/hyperactivity disorder: an EEG analysis

Ann-Christine Ehlis, PhD; Saskia Deppermann, MA; Andreas J. Fallgatter, MD

**Background:** Recently, research into attention-deficit/hyperactivity disorder (ADHD) has focused increasingly on its neurobiological underpinnings, revealing (among other things) frontal lobe alterations. Specifically, action-monitoring deficits have been described, including impaired behavioural adjustments following errors. Our aim was to examine the neurophysiological background of post-error behaviour alterations in an adult ADHD sample for the first time, hypothesizing that people with ADHD would differ from controls in neurophysiological markers of cognitive preparation and stimulus processing, specifically following errors. **Methods:** In total, 34 people with ADHD and 34 controls participated in an electroencephalography measurement while performing a flanker task. The final number of electroencephalography samples included in the analyses ranged from 23 to 28. We recorded event-related potentials for the erroneous response itself (error-related negativity) and for events following errors (intertrial interval: contingent negative variation; next flanker stimulus: P300). **Results:** Over frontal electrode sites, error-related negativity amplitudes were significantly reduced in people with ADHD across response conditions. Both groups showed reduced P300 amplitudes on flanker stimuli following errors. Moreover, during the intertrial interval, patients exhibited significantly reduced contingent negative variation, specifically following errors. At the behavioural level, we observed no significant group differences in post-error data. **Limitations:** Only adults were examined (no longitudinal data). **Conclusion:** Based on previous reports of post-error behavioural alterations in childhood samples, we conclude that people with ADHD develop compensatory strategies across the lifespan that lead to inconspicuous post-error behaviour in adulthood. Neurophysiologically, however, subtle alterations remain, indicating a perseverance of at least some frontal lobe deficits in people with ADHD who are partially medicated, particularly with respect to action-monitoring and post-error adaptation.
from medial prefrontal areas, including the anterior cingulate cortex. Finally, an ERN/Ne-like potential of smaller amplitude has also been found to occur after correct responses (correct-response negativity, CRN/Nc), possibly reflecting the monitoring of response conflict or the processing of uncertainty (e.g., regarding the correctness of one's behaviour).

Both children and adults with ADHD have been shown to exhibit diminished ERN/Ne and Pe amplitudes, although the results have been mixed, with some negative findings, particularly for the ERN/Ne. Functional imaging studies have confirmed alterations in the action-monitoring network of unmedicated patients with ADHD, with reduced activation in medial and lateral prefrontal areas on error trials. Moreover, in a behavioural study, Yordanova and colleagues found that adolescents with ADHD showed increased behavioural instability in trials following incorrect responses ("post-error trials"), with respect to increased error rates and reaction time (RT) variability, indicating difficulties with the behavioural adjustments usually initiated after response errors (see also Wiersema and colleagues, Schachar and colleagues, and Mohammed and colleagues). However, the neurobiological correlates underlying this altered post-error behaviour have rarely been investigated. The present study aimed at characterizing the neurobiological underpinnings of post-error behavioural alterations in adults with ADHD using ERPs.

In healthy participants, post-error behavioural adjustments have been observed as post-error slowing (increased RTs on trials following errors), reduction of interference and improvements in accuracy (see also Danielmeier and Ullsperger). The neurobiological basis of these behavioural adjustments might involve recruitment of lateral prefrontal areas to achieve post-error adaptation by modulating both response-related activity in the sensorimotor cortex and target processing in task-relevant sensory areas. Among the prefrontal areas that showed enhanced activation in post-error trials was the anterior prefrontal cortex, possibly due to modulations in attention allocation. In line with this interpretation, post-error RTs were found to correlate with the amplitude of the P300, a stimulus-triggered ERP that is closely linked to attentional processes.

Based on these previous findings in people without ADHD, we focused our analyses on post-error ERPs related to both stimulus processing (P300 amplitude elicited by flanker stimuli following incorrect button-presses) and attentional/preparatory processes in the intertrial interval (ITI; contingent negative variation [CNV] following incorrect responses) in addition to the above-mentioned action-monitoring potentials (ERN/Ne and Pe on error trials). Using this design, we aimed at replicating previous findings of diminished action-monitoring in adults with ADHD as indicated by reduced ERN/Ne or Pe amplitudes or both; and investigating the neurophysiological basis of post-error behavioural alterations. For the latter, we hypothesized that the behavioural instability following errors that has been previously reported in children/adolescents with ADHD might be related to reduced target stimulus processing on trials following errors (P300) or compromised preparatory/attentional processes in the ITI (CNV). Both findings would be in line with results from an fMRI study indicating compensatory recruitment of the temporal cortex in children with ADHD, possibly to resolve stimulus conflict due to difficulties in the reallocation of attention on task-relevant information in post-error trials.

Methods

Participants

We enrolled 34 adult outpatients with ADHD and 34 healthy controls in the study, which took place between March 2014 and July 2015. For recruitment, we used notice-board postings at various places in Tübingen, Germany, and university-wide emails advertising the study. Inclusion criteria for the ADHD group were as follows: an ADHD diagnosis according to DSM-IV criteria; a score of $\geq 18$ on the German ADHD self-report scale ADHS-SB; and a score $\geq 30$ on a short form of the Wender Utah Rating Scale, retrospectively assessing ADHD childhood symptoms. Exclusion criteria were comorbid axis I disorders as assessed by the Structured Clinical Interview for DSM-IV (SCID), with the exception of mild to moderate depression (Beck Depression Inventory II score $< 28$) and specific phobias. Antisocial and borderline personality disorders were also excluded based on SCID-II and a short version of the Borderline Symptom List (cutoff score $> 32$). Controls were assessed for psychiatric and neurologic illnesses using SCID-I and general screening questionnaires, as well as an oral interview. All participants gave written informed consent. The study was carried out in accordance with the Declaration of Helsinki in its latest revision (64th World Medical Association General Assembly, Fortaleza, Brazil) and approved by the local ethics committee (University Hospital of Tübingen).

Paradigm

We adopted a modified version of the Eriksen flanker task based on Yordanova and colleagues and our own studies. Our version consisted of a target stimulus (arrow or triangle) pointing to the left or right, which was flanked by 2 more stimuli of the same type on each side, providing incongruent information by pointing either completely (all 4 flankers) or partly (2 of 4 flankers) in the opposite direction (Fig. 1). The resulting 8 types of flanker stimuli were presented in randomized order (by shuffling the stimulus array using Presentation software [Neurobehavioural Systems Inc.]) for 125 ms. Participants were to indicate the direction of the target stimulus by pressing a button (left or right finger) as quickly and accurately as possible; the shape (arrow or triangle) further determined the response hand, resulting in 4 response options. Additionally, we combined the flanker task with a Go/No-go instruction by presenting all stimuli in either blue or red. We switched the assignment of colour to instruction (Go/No-go) and stimulus shape to response hand (left/right) for each participant between 2 blocks of the experiment, and counterbalanced the sequence of events across participants.
After each button press, visual feedback appeared to indicate whether responses were correct, incorrect, or correct but slow (Fig. 1), although participants generally said that they did not need the external feedback to recognize errors (or even slow responses, especially for the second half of the experiment). We determined the RT threshold individually based on a 40-trial pretest: we used the individual median of RTs for correct responses during this pretest as the threshold for differentiating responses that were on time versus too slow, discarding the first 10 practice trials. Generally, a total of 400 trials were presented in 2 blocks with an ITI of 4000 ms; however, in some cases (2 in the ADHD group, 3 in the control group), up to 600 trials were presented if fewer than 10 errors were made during the first 400 trials. One participant in the ADHD group and 1 in the control group aborted the experiment after 321 and 387 trials, respectively. Post hoc analyses of the number of trials presented revealed that the groups did not differ significantly with respect to the total number of trials, Go/No-go stimuli or fully/partially incongruent flanker stimuli (all \( p > 0.2 \)).

**EEG recording and analysis**

We recorded EEG using a 32-channel DC-amplifier (Brain Products) and 23 Ag/AgCl ring electrodes placed according to the international 10/20 system, with 3 additional electrooculography electrodes. We used a frontocentral electrode position (FCz) as the recording reference (all impedances below 5 k\( \Omega \)) and Brain Vision Recorder (Brain Products) for data recording (sampling rate 1000 Hz; online filter 0.1–100 Hz). At the same time, we conducted near-infrared spectroscopy measurements using the ETG-4000 (Hitachi Medical Co.), the results of which will be reported elsewhere.

We analyzed ERPs using Vision Analyzer 2.0 (Brain Products). After visual inspection of the EEG raw data, we applied high-pass (0.1 Hz) and low-pass (50 Hz; 48 db/oct) filters before correction of eye-movement artifacts based on an algorithm implemented in the software. After that, data were re-referenced to an average reference and segmented according to the relevant conditions. For analysis of the ERN/Ne and Pe, we segmented the data in a response-locked manner, (i.e., relative to correct and incorrect button presses; segment

Fig. 1: Illustration of the flanker task and stimuli. Top: sequence of events within each flanker trial. The feedback always appeared exactly 1625 ms after the onset of the stimulus, with a fixed response window of 1000 ms. The 4 response keys are marked in yellow. Bottom: flanker stimuli containing information about the response hand (left or right, depending on the type of stimulus [triangle v. arrow]), the response finger (left or right, depending on the orientation of the central/target stimulus) and the action required (Go = button-press v. No-go = inhibition of motor response, depending on the colour). We switched assignment of stimulus type to response hand and colour to Go/No-go instruction between 2 blocks of the experiment for each participant (this example: Go = blue; left hand = triangle), and we counterbalanced the sequence of these assignments across participants.
Performance monitoring and post-error adjustments in adults with ADHD

The 2 groups did not differ significantly with respect to age, school education, distribution of sex, smoking status or handedness. Based on the Mehrfachwahl-Wortschatz-Intelligenztest, they also did not differ in terms of mean IQ (Table 1). As expected, people with ADHD had significantly higher scores on the hyperactivity and inattention subscale of the ASRS, as well as on the impulsivity subscale of the I7. Within the ADHD group, the following use of medication was reported: methylphenidate \((n = 6)\), dexamphetamine \((n = 1)\), atomoxetine \((n = 1)\) and bupropion \((n = 1)\). In all but 4 cases, ADHD medication was discontinued 2 to 7 days before the trial. Both groups consisted largely of university students \((n = 22\) controls, \(n = 24\) ADHD), but 11 from the control group and 2 from the ADHD group reported working as executive or regular employees; 2 from the ADHD group were unemployed; 3 from the ADHD group were self-employed; 1 from the ADHD group was a trainee; and 2 from the ADHD group were homemakers (information was missing for 1 member of the control group).

**Flanker task performance**

Groups did not differ significantly with respect to mean RTs; however, the ADHD group showed increased SD-RTs as a measure of response variability for all responses, as well as data between groups using \(t\) tests for independent samples or (in cases of non-normality) Mann–Whitney \(U\) tests. We conducted within-group comparisons using matched-samples \(t\) test or Wilcoxon test.

We analyzed EEG data using repeated-measures analyses of variance (ANOVAs), consisting of the between-subject factor of group (ADHD v. control) and within-subject factors of electrode position (ERN/Ne: Fz, FCz; P300: Cz, Pz) and condition (ERN/Ne: correct v. incorrect response; P300, CNV: post-error v. pre-correct trials). If the sphericity assumption was violated, we used Huynh–Feldt correction. We further analyzed significant interactions using post hoc \(t\) tests for matched or independent samples. Effect sizes were given as \(η^2\) (ANOVAs), Cohen's \(d\) \((t\) tests/group comparisons) and Cramer's \(V\) (crosstabs), respectively.

**Results**

**Sample characteristics**

<table>
<thead>
<tr>
<th>Group</th>
<th>Total</th>
<th>Controls</th>
<th>ADHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Handedness</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Statistical analysis**

At the behavioural level, we analyzed RTs, standard deviation of reaction times (SD-RTs) and error rates (overall, as well as for post-correct and post-error trials; for errors, we considered only incorrect button presses to Go stimuli). We determined traditional post-error slowing (PES) by subtracting RTs on trials after correct responses from RTs following errors. Additionally, we calculated a putatively more robust PES measure by defining pairs of RTs around each error, consisting of the individual pre-error \((E-1)\) and post-error \((E+1)\) trials. Based on these matched pairs, we calculated PES(robust) as the difference between the mean RTs on post-error and pre-error trials. We compared behavioural
for button-presses on trials following correct responses (Table 2). The 2 groups did not differ significantly with respect to the number of incorrect responses, although the ADHD group showed a statistical trend toward increased error rates overall, and for trials following correct responses. The 2 groups did not differ significantly with respect to the traditional index of PES; for PES(robust), the ADHD group tended to show increased values.

For within-group comparisons, both groups showed significant PES in terms of longer RTs on individually paired post-error versus pre-error trials (PES[robust], ADHD: $t_{33} = 5.48, p < 0.001; \text{control}: t_{33} = 2.15, p < 0.05$) and longer RTs on individually paired post-error versus pre-error trials (PES[robust], ADHD: $t_{33} = 5.98, p < 0.001; \text{control}: t_{33} = 5.65, p < 0.001$), respectively. Also in both groups, SD-RTs did not differ significantly on post-error versus post-correct trials (ADHD: $t_{33} = 0.82$, not significant; control: Wilcoxon $Z = 1.19$, not significant). Finally, both groups showed post-error adaptation in terms of fewer errors following incorrect responses versus correct responses (both $Z = 5.09, p < 0.001$). For ADHD symptom correlations, in controls, I7 impulsivity scores correlated significantly with SD-RTs following errors ($r = 0.447, p < 0.01$), indicating higher variability of the response behaviour following errors with increasing impulsivity.

**EEG data**

**Action-monitoring potentials**

For analysis of the ERN/Ne, a $2 \times 2 \times 2$ (condition $\times$ position $\times$ group) ANOVA revealed a significant main effect of group ($F_{1,33} = 4.89, p < 0.05, \eta^2 = 0.09$), with overall greater negativity in the control group ($-5.16 \pm 2.79 \mu V$) than in the ADHD group ($-3.55 \pm 2.50 \mu V$). We also found significant interactions of condition $\times$ position ($F_{1,33} = 42.06, p < 0.001, \eta^2 = 0.45$) and position $\times$ group ($F_{1,33} = 5.88, p < 0.05, \eta^2 = 0.10$). Post hoc $t$ tests showed a significant difference between conditions only for electrode position FCz, with significantly more negative amplitudes for errors ($-5.24 \pm 3.19 \mu V$) than for correct responses.
responses (−3.44 ± 3.62 µV; \( t_{52} = -3.55, p = 0.001 \)), while ERN/Ne amplitudes showed a similarly pronounced negativation for both conditions at Fz (\( t_{53} = 0.35 \), not significant; Fig. 2). For the position × group interaction, post hoc testing revealed a significant difference between groups only at Fz, with higher amplitudes in the control group (−5.46 ± 2.96 µV; \( t_{51} = 2.95, p < 0.01, d = 0.81 \)). We observed no between-group differences at FCz (\( t_{51} = 1.24, p > 0.9, d = 0.34 \); Fig. 2). The group difference at Fz occurred independent of the response condition for both errors (ERN/Ne) and correct responses (CRN/Nc).

Regarding brain–behaviour correlations, the amplitudes of the CRN/Nc at FCz showed a significant negative correlation with SD-RTs (\( r = -0.482, p < 0.01 \)), but only in the ADHD group (this numerically negative correlation indicates a greater response time variability with a more pronounced CRN/Nc—that is, stronger action-monitoring on correct trials). Moreover, also in the ADHD group only, CRN/Nc correlated significantly with ASRS hyperactivity scores (Fz; \( r = 0.544, p < 0.01 \)), indicating a more pronounced CRN/Nc with lower symptomatology. At a 1% significance level, no other correlations reached the statistical threshold.

**Processing of flanker stimuli**

For analysis of the P300 elicited by flanker stimuli (Fig. 3), a 2 × 2 × 2 (condition × position × group) ANOVA revealed a significant position × group interaction (\( F_{1,50} = 4.61, p < 0.05, \eta^2_p = 0.08 \)) and a main effect of condition (\( F_{1,50} = 10.98, p < 0.01, \eta^2_p = 0.18 \)), but no significant 3-way interaction (condition × position × group; \( F_{1,50} = 0.001, p > 0.9, \eta^2_p < 0.001 \)).

![Fig. 2: Illustration of ERN/Ne (and CRN/Nc) findings. Left side: grand averages of response-locked potentials elicited by correct button-presses (thin blue lines) and errors (bold red lines) at electrode positions Fz, FCz, Cz and Pz for controls (upper 4 panels) and people with ADHD (bottom 4 panels); y-axis with unit µV. For illustration purposes, we applied a 30 Hz low-pass filter (x-axis 0 marks the correct/error button-press). Topographical maps show the field distribution of the ERN/Ne (blue field, left) and subsequent Pe (red field, right). The middle panel on the right illustrates a significant group × position interaction indicated by the ANOVA on ERN/Ne amplitudes. **Indicates a significant group difference for mean correct/error potentials at electrode position Fz, \( p < 0.01 \). ADHD = attention-deficit/hyperactivity disorder; ANOVA = analysis of variance; CRN/Nc = correct response negativity; Cz = central electrode position; ERN/Ne = error-related negativity/error negativity; FCz = frontocentral electrode position; Fz = frontal electrode position; Pe = error positivity; Pz = parietal electrode position.](image-url)
Overall, participants showed significantly higher P300 amplitudes on flanker stimuli following correct trials (8.88 ± 3.72 µV) than following errors (7.98 ± 4.20 µV). With respect to the group × position interaction, the ADHD group did not differ significantly from controls at either Cz (8.88 ± 4.54 v. 10.52 ± 4.42 µV; t\textsubscript{50} = 1.32, p = 0.19, d = 0.37) or Pz (6.83 ± 3.23 v. 7.58 ± 3.16 µV; t\textsubscript{50} = 0.86, p = 0.40, d = 0.23). However, the increase in P300 amplitudes from Pz to Cz was significantly more pronounced in the control group than in the ADHD group (t\textsubscript{50} = 2.15, p < 0.01, d = 0.61), indicating a somewhat more frontal topography of the P300 in the control group.

Regarding brain–behaviour correlations, P300 amplitudes showed significant negative correlations with RTs in the ADHD group following slow responses (Cz: r = −0.570, p < 0.01; Pz: r = −0.474, p = 0.013). Similarly, SD-RTs tended to correlate negatively with P300 amplitudes in the ADHD group following both incorrect responses (Cz: r = −0.506, p < 0.01) and slow responses (Cz: r = −0.469, p = 0.014; Pz: r = −0.455, p = 0.017), indicating shorter RTs and smaller SD-RTs with higher amplitudes (but only after a suboptimal response outcome).

**Inter-trial interval**

For analysis of the CNV in the ITI, a 2 × 2 × 2 ANOVA with the factors condition and group, as well as time bin (−2000 to −1000 ms vs. −1000 to 0 ms before the next stimulus) revealed a significant condition × group interaction (F\textsubscript{1,45} = 5.26, p < 0.05, η\textsuperscript{2} = 0.11) in addition to the expected main effect of time bin (F\textsubscript{1,45} = 7.36, p < 0.01, η\textsuperscript{2} = 0.14), with more negative CNV amplitudes as the duration of the ITI increased. Post hoc tests for the interaction showed a significantly more negative CNV amplitude in the control group compared to the ADHD group, specifically following errors (−1.36 ± 2.09 v. 0.53 ± 1.86 µV; t\textsubscript{45} = 3.29, p < 0.01, d = 0.96; Fig. 4), with comparable amplitudes following correct trials (−0.75 ± 2.85 v. −0.25 ± 2.51 µV; t\textsubscript{45} = 0.65, not significant, d = 0.19). At an uncorrected...
significance level, amplitudes of the later part of the CNV (second time bin) following slow responses correlated positively with RTs of the same condition in healthy controls ($r = 0.462, p = 0.026$), indicating slower RTs with more positive (i.e., smaller) CNV amplitudes.

**Discussion**

This study investigated neurophysiological correlates of action-monitoring and post-error adaptation in adult ADHD outpatients compared with age-matched controls.
using a modified flanker task. Specifically, we aimed at revealing the neurophysiological basis of post-error behavioural alterations previously observed in children/adolescents with ADHD.29 Partly replicating previous findings,19–22 we observed reduced amplitudes of action-monitoring potentials (ERN/Ne, CRN/Nc) in people with ADHD, indicating a diminished processing of response conflict for both correct and incorrect responses in this difficult, high-conflict task (inconsistencies with previous reports of partly negative ERN/Ne findings may depend on the task conditions; specifically, ERN/Ne amplitudes elicited by No-go/commission errors were repeatedly not found to differ between patients with ADHD and controls).23,25,26 We observed an unusually large early negativity following correct responses (CRN/Nc), which might be indicative of increased uncertainty of the participants or, possibly more likely, increased response conflict27 in our combined flanker–Go/No-go task, with 4 response options on Go trials alone. Interestingly, this action-monitoring component was associated with lower symptomatology (ASRS hyperactivity scores), but also with higher SD-RTs in the ADHD group, indicating increased response variability with more pronounced action-monitoring on correct trials.

Regarding post-error processes (the main topic of this study), we observed specific alterations in the ITI that were in line with our hypotheses. Reduced CNV amplitudes specifically following incorrect responses indicated compromised preparatory processes for the next trial after an error in patients with ADHD. At the same time, a general relationship between the CNV and response readiness on the next trial was confirmed by a tendency for quicker responses with higher CNV amplitudes in controls, particularly following slow responses. Based on previous data showing a strong link between the CNV and attention,28 we conclude that response errors seemed to have a negative effect on attentional processes related to the cognitive preparation for the next trial in the current sample of adults with ADHD.

However, despite this neurophysiological alteration, we found no behavioural indications of an altered post-error adaptation as previously described in younger ADHD populations.25,29,30 Specifically, both groups displayed PES (with PES[robust] showing a statistical trend for higher values in patients with ADHD) and made significantly fewer errors following incorrect responses compared with correct responses. Regarding more general behavioural measures, we replicated the well-known finding of enhanced SD-RTs in ADHD, suggesting an increased variability of RTs at the intraindividual level.29 Interestingly, in controls, impulsivity scores correlated positively with SD-RTs, showing an effect also at a subclinical level; the fact that this finding was particularly observed for post-error trials is in line with this study’s hypothesis of a specific effect of ADHD symptoms on post-error adaptation.

In the present sample of adult ADHD patients, we could not replicate increased error rates or SD-RTs specifically following errors as previously reported in a younger group (7 to 16 years of age).29 We interpret these findings to indicate a normalization of post-error adaptive strategies across the lifespan that leads to inconspicuous behavioural data in adults with ADHD (although the cross-sectional nature of the present study, focusing on a single age group, leaves this interpretation to be somewhat speculative). This conclusion is also in line with our previous findings suggesting a normalization of action-monitoring potentials and post-error adaptive behaviour in older adults with ADHD.21 It is also possible that people with ADHD develop compensatory strategies across the lifespan that allow them to optimize their response to unfavourable action outcomes, which may be particularly frequent as a result of their symptomatology. However, longitudinal data in optimally unmedicated patients are needed to confirm this interpretation. Alternatively, ITIs were long in the present study (4 s), possibly enabling patients to initiate behavioural adjustments that might not have occurred otherwise (because of the greater challenge shorter ITIs would have posed for adaptation/control capacity).29 Also, given the strict exclusion criteria for psychiatric comorbidities, we might have investigated an ADHD subsample that showed particularly good compensatory behaviour, as reflected in their relatively high-functioning state. Finally, behavioural effects may be subtler than assumed overall, since Plessen and colleagues reported reduced post-error adaptation in motor networks (fMRI data) without group differences at the behavioural level, also for children with ADHD.

With respect to attentional processes on stimuli following correct/incorrect responses, P300 amplitudes to flanker stimuli were significantly reduced for post-error versus post-correct trials, independent of diagnosis. Patients differed from controls only in a somewhat less frontal distribution of the P300 field (see Fallgatter and colleagues for data showing a similarly reduced “frontalization” of the P300 in people with ADHD on other Go/No-go tasks, possibly indicating reduced frontal lobe involvement). Overall reduced amplitudes of the flanker P300 on post-error trials indicate that both groups had mild difficulties reallocating their attention on task-relevant information following incorrect responses, an observation previously made specifically in ADHD samples.52 The notion that the flanker P300 seemed to be a good indicator of attentional processes was confirmed by partly significant brain-behaviour correlations in terms of quicker responses and less variable RTs with higher P300 amplitudes.

Limitations

We investigated only adults in the present study, which prevents any definite conclusions about developmental processes that might underlie changes in the neurocognitive characteristics of ADHD across the lifespan. Regarding the potential effect of sex, we included both female and male participants, whereas previous studies have focused on male participants only.29 For statistical analysis, we took a rather liberal approach in terms of corrections for multiple testing in this explorative study (especially given the relatively large number of tests performed for the correlation analyses). Future studies are needed to confirm whether the
findings can be replicated and to test the main hypotheses more stringently. Finally, because our sample was recruited from a relatively small university town using (among other options) university-wide emails, the sample might not be fully representative, and might also explain missing group differences in terms of general demographic and psychometric variables.

Conclusion
We conducted, to our knowledge, the first ERP study on post-error adaptation in adults with ADHD. Replicating previous findings of reduced action-monitoring potentials, a specific focus was on post-error processes in trials following incorrect responses. Here, post-error behavioural alterations previously reported in younger samples could not be replicated, possibly suggesting the development of compensatory strategies across the lifespan. Neurophysiologically, however, post-error alterations were still apparent, underlining the potentially endophenotypic nature of frontal lobe deficits in ADHD. Longitudinal data are needed to further investigate changes in error-monitoring and error-adaptation across the lifespan, as well as their contribution to ADHD symptoms and clinical outcomes.

Acknowledgements: This study was supported by IZKF Tübingen (Junior Research Group grant 2115-0-0). The authors thank Betti Schopp and Ramona Täglich for their proficient and dedicated technical assistance, and Stefanie Kettner for adapting the paradigm.

Affiliations: From the Department of Psychiatry and Psychotherapy, University of Tübingen, Tübingen, Germany (Ehlis, Deppermann, Fallgatter); the Centre for Integrative Neuroscience, University of Tübingen, Tübingen, Germany (Fallgatter); and the LEAD Graduate School and Research Network, University of Tübingen, Tübingen, Germany (Ehlis, Fallgatter).

Competing interests: None declared.

Contributors: A.-C. Ehlis and A. Fallgatter designed the study. S. Deppermann acquired and analyzed the data, which A.-C. Ehlis also analyzed. A.-C. Ehlis and S. Deppermann wrote the article, which all authors reviewed. All authors approved the final version to be published and can certify that no other individuals not listed as authors have made substantial contributions to the paper.

References


