**Archival Report**

**Attentional Selection and Suppression in Children With Attention-Deficit/Hyperactivity Disorder**

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**ABSTRACT**

**BACKGROUND:** Attention-deficit/hyperactivity disorder (ADHD) is a prevalent neurodevelopmental disorder with prominent impairments in directing and sustaining attention. The aim of this study was to identify the neurophysiologic bases of attention deficits in ADHD, focusing on electroencephalography markers of attentional selection (posterior contralateral N2 [N2pc]) and suppression (distractor positivity [PD]).

**METHODS:** The electroencephalography data were collected from 135 children 9–15 years old with and without ADHD while they searched for a shape target in either the absence (experiment 1) or the presence (experiment 2) of a salient but irrelevant color distractor.

**RESULTS:** In experiment 1, the shape target elicited a smaller N2pc in children with ADHD (n = 38) compared with typically developing children (n = 36). The smaller N2pc amplitude predicted higher levels of inattentive symptoms in children with ADHD. Moreover, the target-elicited N2pc was followed by a positivity in typically developing children but not in children with ADHD. In experiment 2, the salient but irrelevant color distractor elicited a smaller PD component in children with ADHD (n = 32) compared with typically developing children (n = 29). The smaller PD predicted higher inattentive symptom severity as well as lower behavioral accuracy in children with ADHD.

**CONCLUSIONS:** The correlation between N2pc/PD amplitudes and ADHD symptom severity suggests that these signals of attentional selection and suppression may serve as potential candidates for neurophysiologic markers of ADHD. Our findings provide a neurophysiologic basis for the subjective reports of attention deficits in children with ADHD and highlight the importance of spatial attention impairments in ADHD.

**Keywords:** ADHD, Electroencephalography, N2pc, PD, Selection, Suppression

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Attention-deficit/hyperactivity disorder (ADHD) is characterized by developmentally inappropriate symptoms of inattention, hyperactivity, and impulsivity and affects ~5% of school-age children (1). Although the disorder is defined by subjective reports of attention deficits, the nature of any objective impairment of sustained or selective attention remains actively debated. Previous behavioral studies reported that individuals with ADHD had unimpaired selective attention and could effectively use top-down control to filter distractors (2–4). However, a series of electroencephalography (EEG) studies using spatial cuing paradigms found abnormal alterations in posterior alpha and frontal theta activity as well as their functional disconnection in response to a cue in children with ADHD (5–7). Additionally, several previous event-related potential (ERP) studies demonstrated that subjects with ADHD have deficits in both early sensory components (8) and subsequent response selection in some visual spatial tasks (9,10). These EEG and ERP findings imply the possible occurrence of spatial attention impairments in ADHD.

One ERP component known as posterior contralateral N2 (N2pc) is a well-characterized index of covert visual attentional selection (11–13). The presence of a reliable N2pc in response to salient but task-irrelevant visual objects has been interpreted as reflecting stimulus-driven bottom-up attentional capture (14,15), whereas modulation of the N2pc by task set and anticipation has been interpreted as evidence for attentional top-down control (16–22). A hallmark of selective attention is the active suppression of task-irrelevant, distracting information. Another ERP component known as distractor positivity (PD) is thought to be a neurophysiologic marker of this active suppression mechanism (21–28). This positivity is elicited when selective processing of the eliciting stimulus is to be avoided (22–27) or terminated (21,28).

To the best of our knowledge, neither N2pc nor PD has been studied in children with ADHD. Yet, *prima facie*, there is good reason to expect that these neurophysiologic markers could relate to the attention deficits of children with ADHD, which include distractibility and problems maintaining attentional focus. One recent study showed that the N2pc delay...
may be a neurocognitive endophenotype of adult ADHD (29); however, we cannot tell whether this N2pc pattern developed over the course of the disease or whether it would likewise be observable in children. The present study investigated 1) whether attention problems in children with ADHD could be partly explained by a reduced ability to modulate N2pc, P2, or both and 2) whether abnormalities in these components were related to symptom severity in children with ADHD. To that end, we conducted two visual search experiments to assess the characteristics of covert visual spatial attentional selection in children with ADHD and their ability to suppress salient but irrelevant distractors.

**METHODS AND MATERIALS**

**Participants**

For this study, 170 children (95 with ADHD, 132 boys) were recruited. Written consent was obtained from all children and their parents according to the Declaration of Helsinki. The study was approved by the Ethics Committee of Peking University Institute of Mental Health. Data from 35 participants were discarded because of the high ratio of noise in the EEG signals or excessive horizontal and vertical eye movement (see the Supplement for the objective exclusion criteria). The group comparisons reported here are from the remaining 135 participants (70 with ADHD, 104 boys; 79% of the samples). Specifically, 38 drug-naive children with ADHD (32 boys) and 36 typically developing (TD) children (27 boys) participated in experiment 1. Participants in experiment 2 were 32 drug-naive children with ADHD (26 boys) and 29 TD children (19 boys). For both experiments, there were no significant differences between the groups in terms of age, IQ, and sex ratios (Table 1). Diagnosis of ADHD was based on DSM-IV criteria (Supplement).

**Search Paradigm**

The stimulus was a circular search array, consisting of 12 items positioned around the circle at a distance of 5° visual angle from the central fixation cross (Figure 1A). The stimulus in experiment 1 consisted of one circle (target) and 11 diamonds; experiment 2 consisted of a shape singleton target with a salient but irrelevant color singleton distractor (red or green) simultaneously presented. In both experiments, the tasks remained the same for the participants: participants were instructed to maintain their gaze at fixation and report the position of the target (upper or lower) but to ignore other extraneous items and distractors. The Supplement provides more specific paradigm details.

**ERP Recording and Analysis**

The EEG data were acquired from 128 channels (HydroCel Geodesic Sensor Net; Electrical Geodesics, Inc., Eugene, OR) with Net Station EEG Software. The impedance of all electrodes was kept below 50 kΩ during the data acquisition. All electrodes were physically referenced to Cz (fixed by the EGI system). The EEG data were amplified with a band pass of 0.01–400 Hz (half-power cutoff) and digitized online at 1000 Hz.

Offline EEG processing and analyses were performed using custom MATLAB (The Mathworks, Inc., Natick, MA) scripts and functions from the EEGLAB environment (30). The EEG data were band-pass filtered (half-power cutoff at 1–40 Hz) with a roll-off of 12 dB/octave (7) and then were re-referenced to the average of the left and right mastoid channels. Electrodes containing excessive artifact or high-amplitude, high-frequency muscle noise (>50% of total recording time) were excluded from further analysis. Data from the task blocks in each experiment were concatenated to form a continuous time series. This time series was subsequently inspected for outlier epochs encompassing gross movements and muscle artifacts, and these time series were removed. The trimmed data were then decomposed into maximally independent component processes using temporal independent component analysis decomposition via extended infomax. The components of independent component analysis associated with vertical eye movements were visually identified and removed according to their spatial, spectral, and temporal properties. The data were then segmented relative to stimulus onset (−200 to 600 ms), and the baseline preceding the stimulus (−200 to 0 ms) was subtracted. Epochs were then sorted according to target visual field (left, right) for each group of children.

To further control for horizontal eye movements, we rejected all segments with signals exceeding ± 50 μV at the difference waves of electrodes F9/10 during 200–400 ms before ERP averaging. To further control for eye blinking or closing during the presentation of stimulus, we also rejected all segments with signals exceeding ± 70 μV at electrodes F1/2 during 0–200 ms from the original segmented data before independent component analyses. Epochs contaminated by incorrect responses and responses faster than 200 ms or slower than 2000 ms were also excluded from the ERP averages. To assess whether any systematic horizontal electrooculography activity was present in the remaining data, we computed averaged F9/10 waveforms for left and right target trials. In all participants, residual activity was <2 μV, indicating that residual eye movements were less than ± 0.3° (31). An average of 21.3% of trials were rejected on the basis of artifacts for the final set of participants.

There were no significant differences between the number of valid trials (range, 169–190) for the ADHD and TD groups.
The N2pc and PD components were measured from the difference waves, whereby the waveform from the hemisphere ipsilateral to the stimulus of interest was subtracted from the waveform from the hemisphere contralateral to the stimulus. Both N2pc and PD were measured at the PO7 and PO8 electrode sites, where they were largest in the two experiments, respectively. The amplitudes of N2pc and PD were calculated as the mean value of a 20-ms window centered at the averaged peak between 200 and 300 ms and 300 and 400 ms, respectively. The averaged peak was based on a grand average across all subjects in both groups. The onset latency was the time at which the difference waveform reached 50% of its peak amplitude for each subject. Comparisons of N2pc and PD at the PO7 and PO8 electrode sites as well as behavioral performance (reaction time [RT], accuracy, RT variance) between the two groups were made using two-tailed independent sample t tests. All statistical results of amplitude analysis were confirmed with nonparametric permutation tests that do not depend on precisely defined measurement windows (Supplement). This permutation approach can provide an estimate of the probability that the observed response is due to random variation in the data, rather than a consistent physiologic response (32).

**EEG Predictors of Symptoms and Behavior**

To test whether the ERP neural indices were linked to behavioral responses and clinical assessment, we constructed a multiple regression model for each of the performance measures (accuracy, RT, RT variance) as well as the attention and hyperactivity/impulsivity subscales of the ADHD Rating Scale (ADHD-RS) for each group. The ERP measures were entered as continuous independent variables. Because behavioral performance is known to improve with age, we included age and sex as a continuous covariate of no interest. The model significance was tested using an F test; individual regression coefficients were evaluated using t tests, which were corrected for multiple comparisons using the false discovery rate method (33). The q value specifying the maximum false discovery rate was set to 0.1, such that no more than 10% false positive could be included.
N2pc and P0 in Children With ADHD

RESULTS

Experiment 1: Children With ADHD Have Deficits in Target Selection and Subsequent Suppression Process

We anticipated that attention would initially be deployed toward the target location, eliciting N2pc (11–20). We also predicted that the N2pc component would be followed by a contralateral positivity (P0) component, reflecting that attention is actively terminated after the completion of perception so that the brain can be prepared for the next target (21,28). Experiment 1 assessed the N2pc and the following P0 components to investigate whether children with ADHD have deficits in the ERP markers of attentional selection or in the subsequent suppression process.

Behavior. The children in the ADHD group were overall slower (t_{1,72} = 2.144, p < .039) and more variable in their RTs (t_{1,72} = 3.627, p < .001) (Figure 1B). The children with ADHD were also less accurate, but this was not significant (t_{1,72} = -1.252, p = .215). These findings are consistent with previous studies showing that RT slowing is found reliably in children as well as in adults with ADHD (2,29). The RT cost might reflect either genuine (age-independent) slowing or a compensatory speed-accuracy tradeoff strategy in ADHD.

ERPs. A reliable N2pc component was elicited 200–300 ms after stimulus onset in both groups, suggesting that the target appearance evoked a contralateralized neural effect in the children (Figure 2A, B). This effect can also be observed in the contralateral-minus-ipsilateral difference waveforms shown in Figure 2C and the topographic maps plotted in Figure 2D. One-sample t tests revealed that N2pc amplitude (254–274 ms) was significantly different from zero at PO7 and PO8 for the ADHD group (t_{37} = -5.566, p < .001) and the TD group (t_{36} = -8.199, p < .001). However, N2pc in children with ADHD was smaller than in TD children (t_{1,72} = 2.024, p < .047) (Figure 2C). We further analyzed the N2pc onset latency differences between the two groups. No significant effect was found between the ADHD and TD groups (t_{1,72} = -1.022, p > .310).

The N2pc in the TD group was followed by a positivity (beginning at ~300 ms) at contralateral relative to ipsilateral electrode sites (Figure 2B, C), reflecting an active suppression process that terminates the allocation of attention to the lateral target (21,28). However, the ADHD group did not show an obvious P0 component (Figure 2A, C). One-sample t tests confirmed that the P0 amplitude (345–365 ms) was significantly different from zero at PO7 and PO8 for the TD group (t_{36} = 2.188, p < .035) (Figure 2C), but not for the ADHD group (t_{37} = -1.428, p = .162) (Figure 2C), which resulted in the significant P0 difference between the two groups (t_{1,72} = -2.549, p < .013). To avoid any biases associated with choosing time windows, the further permutation analyses confirmed the N2pc and P0 results obtained with the conventional analyses (Figure 2E, F, and Supplemental Figure S1).

An alternative interpretation of the N2pc and P0 results might be that the ADHD group showed weaker sensory responses to the search array, which then lead to a smaller N2pc and the absence of the P0. We tested this explicitly by comparing the occipital P1, a positive potential occurring 100 ms after stimulus onset and indexing sensory visual processing. We used a repeated-measures analysis of variance with three factors—group (ADHD vs. TD), target side (left vs. right), and electrodes (O1 vs. O2)—to analyze the P1. However, neither the group effect nor its interaction with other factors was significant for either P1 amplitude and latency (Fs < 1).

EEG Predictors of Symptoms and Behavior. For each group, we constructed a multiple linear regression model with ERP measures (N2pc amplitude, P0 amplitude) as independent variables for each of the dependent variables (accuracy, RT, RTSD, and ADHD-RS inattention and hyperactivity/impulsivity subscales). As illustrated in Supplemental Table S1, the strongest relationship between the behavioral and ERP measures across children with ADHD was observed for the N2pc amplitude. A smaller N2pc amplitude predicted increased symptom severity on the inattention subscale of the ADHD-RS (r_{34} = -3.481, p < .001) (Figure 3A) but not on the hyperactivity/impulsivity subscale (r_{34} = -0.167, p > .869). No significant effect was found for the TD group (Supplemental Table S2). The following P0 amplitude showed no significant relationships with behavior and symptoms; this is likely because of its small amplitude values in experiment 1.

Experiment 2: Children With ADHD Have a Deficit in Active Distractor Suppression

We added a salient but irrelevant color singleton distractor to the search array in Experiment 2. The distractor singleton is chosen to pop out more than the target singleton, so that the distractor’s bottom-up salience and the observer’s top-down goal are in opposition and thus compete for attentional selection (34,35). If attention is miscaptured by the color distractor, the distractor should elicit an N2pc (14,22). In contrast, if the children are able to suppress the color distractor, the distractor should elicit a P0 or a small N2pc followed by a P0 (21–28). We aimed to provide more direct evidence that children with ADHD have a deficit in active distractor suppression.

Behavior. Because we primarily investigated the distractor suppression effect in experiment 2, we focused on trials with a lateral distractor and a midline target present in the following behavioral and ERP analysis (the results of trials with a midline distractor and a lateral target present are presented in the Supplement). Similar to in experiment 1, ADHD group performance was worse overall than TD group performance with slower RTs (t_{1,59} = 2.996, p < .004), lower accuracy (t_{1,59} = -4.039, p < .001), and more variability in RTs (t_{1,59} = 4.879, p < .001) (Figure 1B). These data further demonstrate that covert spatial attention is impaired in children with ADHD.

ERPs. A small distractor-elicited N2pc component is apparent at 200–300 ms followed by a pronounced P0 component 300–400 ms after visual search array onset in both groups (Figure 4A, B). One-sample t test revealed that the N2pc component (254–274 ms) did not reach significance in either
group (ADHD, \( t_{31} = -1.685, p > .102 \); TD, \( t_{28} = -1.569, p > .128 \)), but the PD component (308–328 ms) was significant for both groups (ADHD, \( t_{31} = 3.357, p < .002 \); TD, \( t_{28} = 5.857, p < .001 \)). We then focused on the PD difference between the two groups. The PD onset latency was nearly identical for the ADHD and TD groups (\( t_{1,59} = 0.249, p > .804 \)), but the PD amplitude in the ADHD group was much smaller than in the TD group (\( t_{1,59} = -2.745, p < .008 \) (Figure 4C, D). These results were confirmed by the further permutation analyses (Figure 4E, F, and Supplemental Figure S2).

Previous research demonstrated that attentional capture fluctuates from trial to trial (36–38). If children are able to...
Supplemental Table S3, the impulsivity subscale (ERP measures (N2pc amplitude, PD amplitude) as independent variables. As illustrated in Supplemental Table S3, the strongest relationship between the behavioral and ERP measures across the children with ADHD was observed for the PD amplitude. A smaller PD amplitude predicted higher scores (higher symptom severity) on the inattention subscale \( t_{28} = -2.542, p < .017 \) (Figure 3B), but not the hyperactivity/impulsivity subscale \( t_{28} = -0.756, p = .456 \), of the ADHD-RS in the children with ADHD. Additionally, a smaller PD amplitude predicted lower accuracy in the children with ADHD \( t_{28} = 2.668, p < .013 \). No significant effect was found for the TD children (Supplemental Table S4). The distractor-elicited N2pc amplitude showed no significant relationships with behavior and symptoms, which is most likely because signal was too small in experiment 2.

**EEG Predictors of Symptoms and Behavior.** For each group, we constructed a multiple linear regression model with ERP measures (N2pc amplitude, PD amplitude) as independent variables. As illustrated in Supplemental Table S3, the strongest relationship between the behavioral and ERP measures across the children with ADHD was observed for the PD amplitude. A smaller PD amplitude predicted higher scores (higher symptom severity) on the inattention subscale \( t_{28} = -2.542, p < .017 \) (Figure 3B), but not the hyperactivity/impulsivity subscale \( t_{28} = -0.756, p = .456 \), of the ADHD-RS in the children with ADHD. Additionally, a smaller PD amplitude predicted lower accuracy in the children with ADHD \( t_{28} = 2.668, p < .013 \). No significant effect was found for the TD children (Supplemental Table S4). The distractor-elicited N2pc amplitude showed no significant relationships with behavior and symptoms, which is most likely because signal was too small in experiment 2.

**DISCUSSION**

In visual environments where multiple objects compete for attention, the challenge is to find relevant information and to ignore objects and events that are unrelated to current task goals. The present study investigated whether attention problems in ADHD are associated with deficits in attentional selection and suppression, as indexed by the N2pc and PD components, respectively. A classic pop-out visual search task was used in which children were instructed to covertly attend to a shape singleton target with the absence or presence of a task-irrelevant color singleton distractor. Analyses of behavioral performance and simultaneous EEG recordings revealed remarkable differences when comparing the ADHD and TD groups. Our data indicate that the attention problems in ADHD are at least in part related to poor covert visual spatial attention, possibly arising from deficits both in attentional selection and active suppression. The aberrant modulations of N2pc and PD were related to ADHD symptom severity and thus might be related to the neurophysiologic substrates of the disorder.

**Deficits of Attentional Selection in ADHD**

The N2pc component has been employed as a temporally precise measure of human attentional capture by salient stimuli during visual search [11–20,23]. As expected, the lateral shape singleton targets elicited a robust N2pc in both TD and ADHD groups, reflecting attentional selection of these task-relevant stimuli. However, children with ADHD showed smaller target-elicited N2pc, suggesting that children with ADHD did not deploy sufficient attention resources to the pop-out stimuli.

A salient visual stimulus such as a circle presented among numerous diamonds (a feature singleton) often elicits a rapid shift of spatial attention. Burra and Kerzel [39] demonstrated that N2pc amplitudes were larger when adults knew that the target shape remained the same in a block than when they knew that the target shape varied randomly from trial to trial. Our recent concurrent functional near-infrared spectroscopy–ERP study further demonstrated that even in simple pop-out search tasks, salience-driven singleton capture is not triggered in an entirely automatic bottom-up fashion but can be affected by top-down attentional control or anticipation for the target-relevant feature [20]. In the present study, the target remained a circle throughout experiment 1. Therefore, the target-elicited N2pc difference between the ADHD and TD groups might be related not only to pure bottom-up visual information processing [40] but also to top-down control. Prior work indicating preserved visuospatial orienting in ADHD [41] hinted at a top-down problem, consistent with the absence of group differences in the early (more bottom-up) P1 responses to encoding stimuli in this and other recent studies (7).

**Figure 3.** Regression plot between the event-related potential components and ADHD symptom severity scores. (A) Scatter plots depicting the relationship between target-elicited N2pc amplitude and ADHD Rating Scale score on the inattentiveness subscale in experiment 1. (B) Scatter plots depicting the relationship between distractor-elicited PD amplitude and ADHD Rating Scale score on the inattentiveness subscale in experiment 2. ADHD, attention-deficit/hyperactivity disorder; ADHD-RS$_{\text{inatt}}$, inattention subscale of ADHD Rating Scale; N2pc, posterior contralateral N2; PD$_{\text{distr}}$, distractor positivity.
Deficits of Active Suppression in ADHD

The PD component has been interpreted as an ERP marker of top-down inhibition of attentional capture (21–28). In experiment 1, the N2pc elicited by shape singleton targets in the TD group was followed by a lateralized posterior ERP modulation of opposite polarity. This positivity is contralateral to the target, which was named as PT in the study by Jannati et al. (21). This component might reflect an active suppression process that terminates the allocation of attention to the lateral target in TD children (21,28). However, for children with ADHD, the target-evoked N2pc component simply faded away, with no subsequent positivity, reflecting a passive decay of attention after the completion of target processing (28). This abnormal pattern was also apparent in a recent study by Cross-Villasana et al. (29) that focused on the N2pc in the adults with ADHD (Figure 3 in Cross-Villasana et al.) but was not analyzed. In experiment 2, the salient but irrelevant distractor elicited an obvious PD component in both groups.
N2pc and P0 in Children With ADHD

However, the P0 was much smaller for children with ADHD, providing more direct evidence that children with ADHD have deficits in active suppression.

In the present study, we cannot be absolutely certain that the target positivity effect observed following the N2pc in experiment 1 reflects the same P0 effect observed for the task-irrelevant color distractor in experiment 2 (28). However, the identical polarity and similar scalp distributions of these effects make it quite likely that they reflect the same type of suppressive processes (42). This is the same suppression mechanism that is used to terminate attention after the completion of perception and is also used to prevent the orienting of attention to distractors (21,28). More importantly, children with ADHD lacked the typical positivity following the N2pc in experiment 1, whereas the children in the ADHD group in experiment 2 showed the abnormal P0 suppression when a salient distractor must be suppressed. The aberrant modulation of these two positive effects in children with ADHD across experiments 1 and 2 might share the same neural mechanisms.

Ability of Electrophysiologic Measures of Attention to Predict Symptom Severity and Behavioral Performance

Cross-Villasana et al. (29) found that N2pc latency was prolonged in ADHD adults with ADHD and was positively correlated with ADHD symptom ratings as well as slower behavioral responses. In our study, N2pc amplitude was reduced in children with ADHD, but N2pc latency seemed to be unaffected. The reduced target-elicited N2pc was correlated with higher inattentive symptom severity. Thus, there might be an ERP pattern that developed over the course of the disease. Children with ADHD are more impaired at sufficient attentional deployment to targets, whereas adults with ADHD are more impaired at the speed of attentional selection, although they may learn to compensate for attentional deployment problems over time.

The novel and most critical point from our study is that active suppression was impaired in children with ADHD, as indexed by a reduced P0, which predicted higher inattention symptom severity as well as lower behavioral accuracy. Thus, it appeared that both N2pc and P0 were associated with the inattentive symptom severity, suggesting that the ERP signals of attentional selection and suppression might reflect core mechanisms of ADHD. Our findings are encouraging and require replication in larger samples that can also be used to assess the potential clinical value of individual measures. The present study may contribute to providing an integrative framework for children with ADHD and, prospectively, to developing neural markers for this heterogeneous psychiatric disorder.

Conclusions

Our two ERP experiments provide novel, strong, and converging neurophysiologic evidence that the attentional problems in ADHD are at least in part related to poor covert visual spatial attention, possibly arising from deficits in both attentional selection (indexed by N2pc) and active suppression (indexed by P0). The correlation between N2pc/P0 amplitudes and ADHD symptom severity implies that these signals of attentional selection and suppression may serve as potential candidates for neurophysiologic markers of ADHD.

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ARTICLE INFORMATION

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