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Neurotypical Adults employ Distinct Cognitive Mechanisms compared to Adults with ADHD during a Sustained Attention Task with Gestalt Stimuli

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Abstract

Sustained attention is a fundamental cognitive ability that influences various aspects of human functioning. Studies of the neural correlates of attention commonly treat sustained attention as an isolated construct, however in any ecological context, sustained attention interacts with other executive functions such as inhibition of interference and processing of complex hierarchical stimuli. We have thus constructed a protocol to probe the interplay between these cognitive processes during visual attention task. We contrast putative typical vs atypical attention by comparing 18 healthy participants with 53 adults with Attention-Deficit/Hyperactivity Disorder, for whom difficulties with sustained attention are a core symptom and thus constitute a natural experiment condition. Our behavioural and brain-imaging analyses demonstrate distinct neural patterns in bottom-up visual processing and attention allocation mechanisms in ADHD and Control groups, highlighting different cognitive strategies utilised by adults with ADHD and healthy participants in tasks requiring sustained attention.

Keywords: ADHD; attention deficit hyperactivity disorder; sustained attention; illusory contours; inhibition of interference, EEG.

Introduction

Sustained attention is continuous and self-directed awareness of a subset of stimuli, that occurs when responding to approximately uniform task stimulation (Robertson & O'Connell, 2010). Maintaining attention is a universal challenge for individuals, and variations in sustained attentional performance are to be expected. Studying deficits in sustained attention can help to pinpoint the underlying neural and cognitive processes, leading to a more nuanced understanding of sustained attention, which is of primary importance as clinical pathologies of attention are recognised as a growing problem in children and adults (for US Centres of Disease Control demographic statistics, see CHADD (2017)). The ability to sustain attention involves resisting neuro-energetic fatigue and distraction, both of which have been implicated in hypotheses of

ADHD and reflected in patients' EEG signal (Gumenyuk et al., 2005; Killeen, Russell, & Sergeant, 2013; Russell et al., 2006). Impairments in the ability to summon glucose for oxidative metabolism, crucial for restoring ion gradients in the fronto-parietal system, can also lead to increased likelihood of attentional lapses, slowed response times, and heightened variability (Killeen et al., 2013).

Measurement and testing of sustained attention is often done with a computerised continuous performance test (CPT). While 'gold standard' CPTs aim to isolate sustained attention as a top-down mechanism through controlled, repetitive classification tasks with limited perceptual complexity, it's essential to recognize that sustained attention is not isolated in ecological contexts. Sustained attention integrates with other executive functions and relies on lower-level perceptual processing. In this study, an exploration of two critical ecological attention demands is presented: (a) successful attending may require inhibition of interference; (b) natural target stimuli are complex and often noisy. To examine these interactions of attention, we conducted an experiment to probe ecological sustained attention and its neural correlates by measuring ADHD and healthy participants with high-resolution electroencephalography (EEG) while they perform a novel CPT, which uses gestalt-image targets, primed by Congruent or Incongruent distractors.

Methods

Participants

We recruited 53 adults (25 males, age $M=36.26$, $SD=10.22$) diagnosed with either ADHD ($n=44$) or ADD ($n=9$) – we refer to both as ADHD group – and 18 adults (6 males, age $M=32.78$, $SD=10.82$) with no diagnosed neuro-cognitive deficits or ongoing medication for ADHD/ADD as a healthy control group. All participants had normal or corrected-to-

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normal vision. The groups did not differ in terms of age, gender, or handedness. The control group size was about 40% of the size of the ADHD group, and thus the statistical power of our tests is not substantially affected by this size disparity according to White (2018).

Inclusion criteria for the ADHD group were: (1) pre-existing diagnosis of ADHD/ADD, (2) no neurological diagnoses, (3) age between 18 and 60, (4) scores on Adult ADHD Self Report Scale (Kessler et al., 2005) and Brown ADHD scale (L. Davenport & Davis, 2011) indicating the presence of ADHD, and (5) an IQ score of at least 80 using WAIS IV measured by a qualified psychologist (Wechsler, 2012). A consulting psychiatrist followed DIVA guidelines to confirm the existing ADHD/ADD diagnosis, or not.

Participants provided written informed consent before entering the study. The protocol followed the Declaration of Helsinki, and ethical approval was obtained from The Ethical Committee of the Hospital District of Helsinki and Uusimaa, 28/03/2012, 621/1999, 24 §.

PSICAT protocol

Primed Subjective Illusory Contour Attention Task (PSICAT) protocol retains the standard repetitive classification-task structure, and adds complex target stimuli preceded by either Congruent or Incongruent interference primers. Primers are attention-grabbing but irrelevant to the task, and in a strategic sense should be ignored, because only target-recognition determines performance. As targets, PSICAT uses the Kanizsa subjective contour illusion (Shape; see Figure 1), which is a perceived polygon induced by collinear ‘Pac-Man’ shapes at the vertices. When the Pac-Man shapes are not collinear, the stimulus forms a nonShape target. PSICAT is a two-alternative forced-choice task, where the response is a left or right hand button-press to the presence or absence of a Shape in Kanizsa stimuli. The classification required by the task involves discrimination of gestalt images from images with identical visual features but no gestalt property. Congruency of primers creates a task-irrelevant probe condition. Thus, PSICAT protocol consists of 2×2 conditions: Congruent Shape, Incongruent shape, Congruent nonShape, Incongruent nonShape. PSICAT consists of five blocks of 110 trials, with a one minute rest break between each block.

Example trials are shown in Figure 1. Each trial consists of a preparatory fixation cross, primer-target pair, and inter-trial interval (ITI). Fixation lasts 100 ms; primers are flashed for 150 ms; targets are held until the participant responds (750 ms on average in this study); and ITI is 500 ± 100 ms (varied to minimise presentation expectancy which reduces trial effects). Thus an average trial should last 1.5 sec, giving an estimated protocol duration of 22 minutes. PSICAT protocol was designed by Cowley (Cowley, 2018) and is available as an open-source code repository at the following url, allowing researchers to reuse and adapt it to their requirements. https://github.com/zenBen/Kanizsa_Prime/

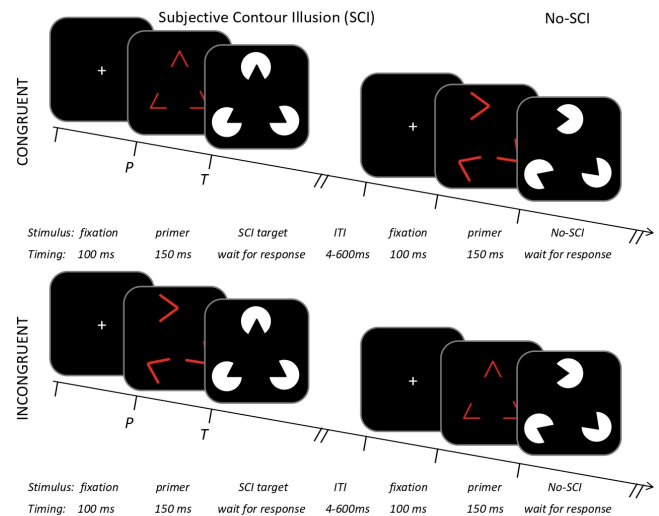


Figure 1: Schematic of the stimuli and protocol structure used in PSICAT. Stimuli are exemplified top left by a triangular Kanizsa subjective-contour-illusion target, displayed white-on-black; with the respective Congruent red-line primer. PSICAT protocol was designed by Cowley (Cowley, 2018).

Behavioral analysis

Exploratory analysis revealed the substantial decrease in RTs in the first block of experiment followed by relatively stable performance in subsequent blocks. We attribute this decline to participants’ adaptation to the experimental design and stimuli. Subsequently, we categorised our data into learning period (Block 1) and sustained performance period (Blocks 2 to 5). This paper primarily focuses on the sustained attention mechanisms which distinguish ADHD and Control groups, thus, the sustained performance period will be the one of interest.

Linear mixed effects model (LMM) was used to analyse participant-wise reaction time (RT) change over time. We fitted LMM with RT as the DV, and group (ADHD, Control), block-wise Trials (bwTrial) (1 to 110), Block (2 to 5) and their interaction, as the predictors. RT variable with skewed distributions was log-transformed. Subjects’ ID was used as a random factor to account for potential variability between Subjects that is not explained by fixed effects. We also added a random intercept for the interaction between Block and Subject and bwTrial and Subject, allowing for variability in baseline performance across different combinations of Block and Subject and bwTrial and Subject, respectively. To compare the evolving variances between the groups across the time, we fitted LMM with SD as a DV, and group (ADHD, Control), block-wise Window (bwWindow) (1 to 87), Block (2 to 5) and their interaction, as the predictors. To obtain SDs we first applied the Subject-wise rolling window of SD across trials in each separate Block and calculated the mean of the SD windows across Subjects. SD variable with skewed

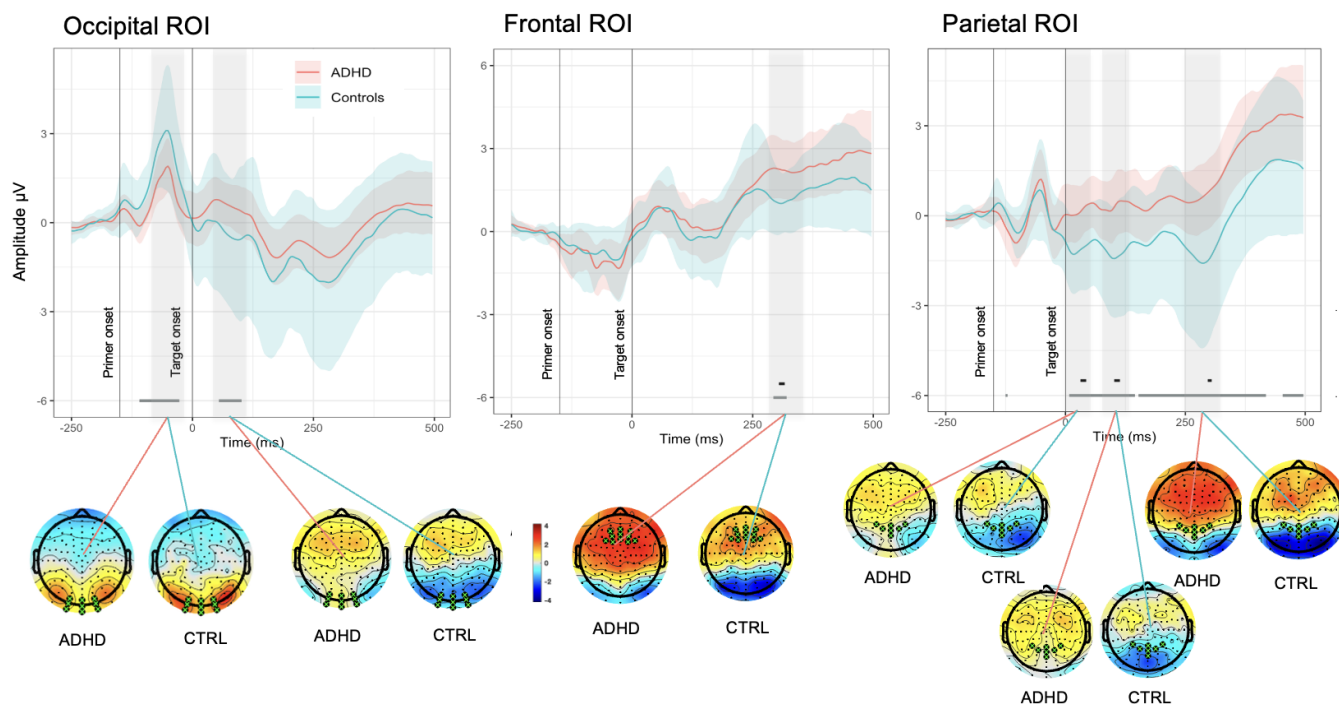


Figure 2: Spatial and temporal activations shown by grand average ERPs (calculated for ROIs) and associated whole-head scalp maps (calculated for the time window of curve testing). Each plot shows the average ERPs responses (solid lines) of the ADHD (red) and Control (blue) groups. The mean responses are surrounded by the MWE confidence bands of the time series. The grey significance lines at the bottom of each plot denote time points where group ERPs were drawn from different distributions. Black significance lines indicate time points where differences met a stricter significance criteria.

distribution was log-transformed. Random effects were used as in previous model for RT. Post-hoc pairwise comparisons between groups across different blocks were conducted using generalized linear hypotheses test ("glht" from the multcomp package). To account for multiple testing, we adjusted the p-values using Holm-Bonferroni method. Joint tests ("joint_tests" function from the emmeans package) were conducted to assess the combined significance of model coefficients in the LMM model.

The ex-Gaussian distribution has been proposed for modeling RTs, especially in ADHD patients who tend to have more abnormally slow responses which are associated in the tau component of the ex-Gaussian distribution (Lee, Mumford, Romero, & Lamme, 1998; Dawson, 1988; Burbeck & Luce, 1982). Thus, we advanced our analysis by calculating Subject-level ex-Gaussian parameters for RT data and fitted them in a LMM with mu, sigma and tau as the DV, and Group (ADHD, Control), Block (2 to 5) and their interaction, as the predictors. Tau variable with skewed distributions was log-transformed. We added a random intercept of Subjects' ID and a random slope for the Block.

EEG measurement and analysis

EEG was measured using Biosemi ActiveTwo equipment with 128 active electrodes mounted on a cloth headcap with equiradial positions. The data was preprocessed using Com-

putational Testing Automated Preprocessing toolbox (CTAP) (Cowley, Korpela, & Torniaainen, 2017; Cowley & Korpela, 2018), based on EEGLAB (Delorme & Makeig, 2004) for Matlab. The data was then low-pass filtered at 45 Hz and high-pass filtered at 2 Hz. Each participant's continuous EEG and EOG data was decomposed using the FastICA algorithm (Hyvarinen, 1999). Independent Components (ICs) statistically similar to the CTAP-detected blinks were removed (Cowley et al., 2017). ERPs were generated from the EEG data using Matlab, time-locked to the target onset of hit trials only (excluding error trials). Continuous EEG was split into 750 ms epochs, with 100 ms pre-stimulus baseline, primer lasting 150 ms, and 500 ms after target stimulus onset. Epochs were baseline-corrected with respect to the mean voltage of the 100 ms period preceding the primer onset. After baseline-correction, a 20 Hz low-pass filter was applied for visualisation and testing. Analyses focused on three ROIs (denoted using 10-5 system labels mapped from the Biosemi 128 channel): frontal at F4, F4h, Fz, F3h, F3, AFF4h, AFz, AFF3h, parietal at P3, P1, CPPz, P2, P4, PO3, POz, PO4 and occipital I1, OI1, O1, POO3, POOz, Oz, OIz, Iz, I2, OI2, O2, POO4. The electrode locations were chosen a priori based on previous research (Hanslmayr, Sauseng, Doppelmayr, Schabus, & Klimesch, 2005; Hanslmayr et al., 2007; MacDonald, Lepage, Eden, & Eichenbaum, 2011; Mathewson, Grat-

ton, Fabiani, Beck, & Ro, 2009).

Minimum-width envelopes (MWEs) confidence bands were calculated to examine temporal characteristics of the ERP waveform and to define whether ERPs are drawn from separate distributions. To track the changes in processing over time we compared MWEs between ADHD and Control groups in the Block 2 vs Block 5. The set of topographic maps were built to represent the distribution of corresponding electrical potentials across the occipital, parietal and frontal regions of interest (ROI). We selected time ranges based on morphology of MWE waves in the corresponding areas.

Results

Behavioral results

LMM results reveal main effect of bwTrial ($t(26550)=3.52$, $p.\text{adj}<.01$) and three way interaction Group-Block4-bwTrial ($t(22240)=4.89$, $p.\text{adj}<.001$), but no main effect of Group or Block on RT. Post-hoc analysis based on generalized linear hypotheses test demonstrated that for ADHD group RTs in Block 2 and 3 were significantly different from Block 5 ($z = 3.43$, $p.\text{adj}<.05$, Cohen's $d = 0.10$ and $z = 3.13$, $p.\text{adj}<.05$, Cohen's $d = 0.09$, respectively). For Control group no differences were found in performance between blocks.

Our analysis of RT variability revealed the main effect of Group ($t(15510) = -6.34$, $p.\text{adj}<.001$), bwWindow ($t(15520) = 6.15$, $p.\text{adj}<.001$), interactions Group-Block3 ($t(15520) = 4.06$, $p.\text{adj}<.001$) and Group-Block4 ($t(15520) = -4.86$, $p.\text{adj}<.001$), and three way Group-Block4-bwWindow interaction ($t(15440) = 6.94$, $p.\text{adj}<.001$) on SD. The effect size for Group, as indicated by Cohen's d , was 0.39, suggesting a medium effect. Joint tests and post-hoc analysis revealed significant differences between the groups in SD in all Blocks (Block 2: $F(1, 13529) = 235.07$, $p.\text{adj}<.001$, Cohen's $d = 0.41$; Block 3: $F(1, 13535) = 119.30$, $p.\text{adj}<.001$, Cohen's $d = 0.32$; Block 4: $F(1, 14756) = 23.44$, $p.\text{adj}<.001$, Cohen's $d = 0.41$; Block 5: $F(1, 14670) = 49.97$, $p.\text{adj}<.001$, Cohen's $d = 0.45$).

Ex-Gaussian analysis of response timing variables demonstrated no main effect of Group on μ and σ . We found main marginal effect of Group on τ ($t(204) = -1.93$, $p<.01$). The effect size for Group, as indicated by Cohen's d , was 0.52, suggesting a medium effect. After adjusting for multiple comparisons, the observed differences in τ were no longer statistically significant.

Neural results

Analysis of MWEs in the occipital area of the brain revealed significantly reduced activity in reaction to primer in ADHD group in comparison with CTRLs 50-120 ms after the onset of primer (see Figure 2). Later on, de-synchronisation demonstrated by the Control group around 200-250 ms after the primer onset (50-100 ms after the target onset) drove the significant differences between ADHD and Controls in the corresponding time period. Comparison across the time of experiment revealed differences between the groups across

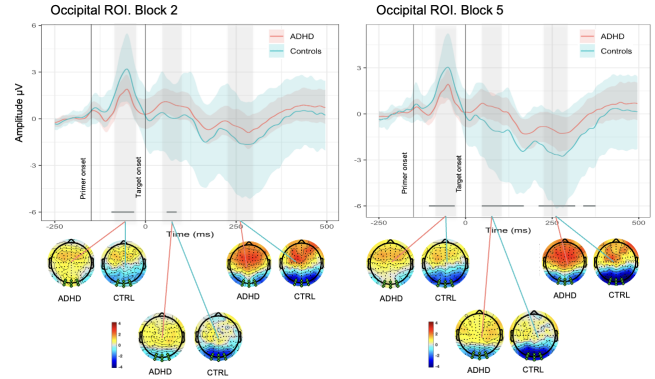


Figure 3: Spatial and temporal activations shown by grand average ERPs in Occipital ROI and associated whole-head scalp maps calculated for the time window of curve testing.

longer periods of epoch in Block 5 in comparison to Block 2. Topographic maps in occipital area demonstrated positive lateral activations in 65-125 ms after primer onset time range present in both groups but stronger for Controls. Stronger negative activation was evident in Control group in comparison with ADHD in 50-100, 140-190 and 230-300 ms time periods of interest. In the same time ranges Control group showed increased intensity of negative activation in Block 5 in comparison to Block 2 (see Figure 3).

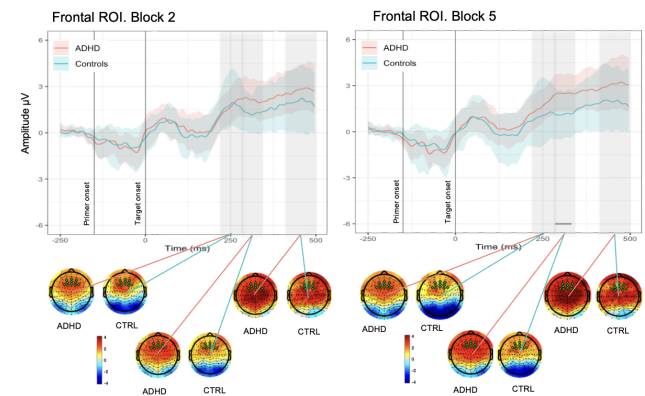


Figure 4: Spatial and temporal activations shown by grand average ERPs in Frontal ROI and associated whole-head scalp maps calculated for the time window of curve testing.

In the frontal ROI Controls demonstrate negative deflection peaking around 300 ms after target onset (see Figure 2). Weaker and delayed in latency peak is noticeable in the ADHD group, driving the significant differences between the groups. ADHD group demonstrated overall enhanced, although not significant, positive activity in comparison to Controls from 250 ms after the target onset. The comparison across blocks revealed differences between ADHDs and Controls in Block 5 and absence of such in Block 2. Topographic maps showed stronger positive activation within

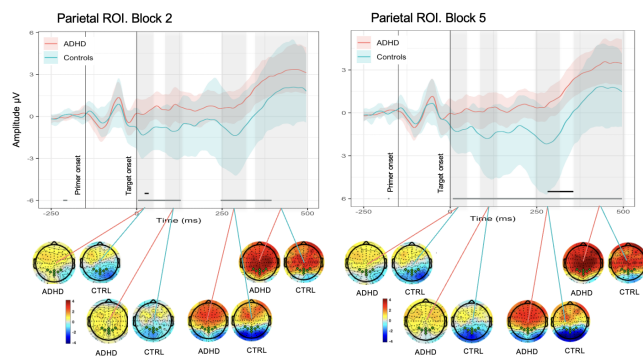


Figure 5: Spatial and temporal activations shown by grand average ERPs in Parietal ROI and associated whole-head scalp maps calculated for the time window of curve testing.

ADHD group rather than in Control in 220-290, 290-350 and 400-500 ms time ranges, though more prominent centrally in the latter one. Both groups slightly increased intensity of positive activation from Block 2 to Block 5 in two latest time ranges of interest (see Figure 4).

In parietal ROI differences between groups were present intermittently throughout the whole length of the epoch, with the most prominent ones corresponding to negative peaks in around 20, 110 and 300 ms demonstrated by Control group (see Figure 2). ADHD group had a similar morphology of the MWE wave but with delayed latency of peaks and weaker activation, especially for the last negative peak around 300 ms after target onset. Similar to the other ROIs, in parietal area differences between the groups were present across longer periods of epoch in Block 5 in comparison to Block 2. Topographic maps revealed negative activation present only in Control group in 0-50, 80-130 and 250-330 time ranges, which was stronger in Block 5 in comparison with Block 2. Positivity was prominent for ADHD patients in (350;500), while Controls demonstrated weaker activation in the same time range. Positivity intensified in Block 5 in comparison with Block 2 for ADHD group (see Figure 5).

Discussion

The current study investigated ecological sustained attention and its neural correlates in individuals with ADHD and healthy participants while they perform a novel CPT, which uses Kanizsa shape illusory contours. Our results revealed significant differences in neural and behavioral responses between ADHD and Control groups, highlighting distinct cognitive processes and compensatory mechanisms in individuals with ADHD.

According to the analysis of speed and variability of performance, ADHD group performed at the same speed but with significantly less consistency in comparison with Controls. However, while Control group did not reduce its RTs over Blocks, ADHD group performance in Block 5 was significantly faster than performance in Block 2 and Block 3. Thus, ADHD group was gradually improving its perfor-

mance throughout the experiment, while Controls reached the plateau of performance already by Block 2.

Results of ex-Gaussian analysis revealed subtle differences between groups in the number of attentional lapses (tau parameter). Other variables (mu and sigma) did not demonstrate between-group differences. Presumably, ADHD group reaches equivalent performance speed at the expense of consistency of its responses, driven by occasional attentional lapses. A possible explanation is related to post-error performance, intended to improve performance on subsequent trials (Rabbitt, 1966). In our experiment this phenomenon might be represented as more focused performance after the period of mind wandering, which helps ADHDs to keep the overall performance on the same level as Controls. This result goes in line with previous research (Kofler et al., 2013; Russell et al., 2006; Tarantino, Cutini, Mogentale, & Bisiacchi, 2013; Epstein et al., 2010). According to the large-scale meta-analytic review of 319 studies individuals with ADHD did not evince slower mean RT after accounting for RT variability, whereas substantial deficits in RT variability persisted even after adjusting for mean RT (Kofler et al., 2013). In another study (Leth-Steensen, Elbaz, & Douglas, 2000) increased variability in a simple reaction time task in children with ADHD was primarily associated with heightened tau parameter, with no significant differences in mu and sigma compared to typically developing controls. Gmehlin et al. (2014) proposed that occasional prolonged RT indicated by tau parameter may be linked to deficits in sustained attention. It is thus crucial to stress the importance of ex-Gaussian distribution in comparison with conventional measures of central tendency.

Neural results provide insights about cognitive mechanisms underlying behavioral outcomes. In the occipital area of the brain ADHD demonstrated reduced activation in early ERP components in response to primers in comparison with Control group. According to the previous literature (Lee et al., 1998; De Valois, Albrecht, & Thorell, 1982; Pollen, 1989) approximately 40 ms after stimulus onset individual V1 neurons in occipital area recognize simplest object features and can discriminate small changes in visual orientations, special frequencies and colours. Smaller amplitudes in ADHD group in response to simple visual stimuli around 65-125 ms in our experiment might represent impaired bottom-up mechanisms in visual processing in adults with ADHD. While visual detection of object is an automatic process, full perceptual completion of gestalt stimuli implies awareness and feedback from higher visual areas back to lower ones. First, stimuli are recognised automatically in V1/V2, then sent forward to lateral occipital complex (LOC) to synthesise illusory contours, segment it and assign boundaries, and later sent back to V1/V2 to fill in details, complete the figure percept, and integrate contextual information (Koivisto, Railo, Revonsuo, Vanni, & Salminen-Vaparanta, 2011; Rao & Ballard, 1999). This two-stage model is also consonant with several studies (Keane, Paterno, Kastner, & Silverstein, 2016; Marini & Marzi, 2016; Wokke, Vandenbroucke, Scholte, & Lamme,

2013), which argue that the full perceptual completion of illusory contours happens only 160 ms after stimulus onset. In our experiment, differences between ADHD and Controls were found 200-250 ms after primer onset, which might represent the later discrimination stage of visual processing.

Most long-lasting differences between the groups, across the length of the epoch, were found in parietal ROI. Parietal cortex is playing an important role in spatial integration, allowing the brain to combine information from visual features to form a coherent spatial representation, and object recognition, helping individuals identify and categorize visual stimuli. In our experiment the largest in amplitude negative peak according to its location and latency might correspond to the N2pc component of ERPs. In line with its function, the amplitude of N2pc component response was related to the condition of Congruency rather than to Shape of the target, and was more prominent in Incongruent trials. While Control group demonstrated prominent negative deflection in the time range of interest, for ADHD group the N2pc component appeared to be less amplitudinal, creating statistical differences between the groups. N2pc amplitude reduction may represent salient stimulus categorization deficit in ADHD subjects (Lazzaro, Gordon, Whitmont, Meares, & Clarke, 2001; Kaur, Singh, Arun, Kaur, & Bajaj, 2019). Furthermore, there is evidence that ADHD patients have manifested significantly less theta rhythm and event-related desynchronisation during inhibition and response trials, during visual continuous performance test (Nejati & Ghayerin, n.d.). This fact goes in line with lengthly reduced activation among ADHD participants in our experiment.

Late non-lateralized ERP enhancements in frontal area of the brain are associated with processes such as decision-making, attention allocation, and cognitive control (P3 component; Kutas, McCarthy, and Donchin (1977); Kok (2001); Polich (2007)). P3 component has been shown to be consistently smaller in children and adults with ADHD than in Controls over different modalities (Hasler et al., 2016; Senderecka, Grabowska, Gerc, Szewczyk, & Chmylak, 2012; Spronk, Jonkman, & Kemner, 2008). The P3 amplitude reductions are linked with diminished facilitation of stimulus processing, attention deficits or inappropriate attentional resources allocation (Baijot, Deconinck, Slama, Massat, & Colin, 2013; Overtoom et al., 1998; Kaur et al., 2019). Some studies also revealed delayed latencies of both negative and positive ERP components in fronto-central area of the brain in individuals with ADHD (Sokhadze et al., 2012). For example, Sokhadze and colleagues in their ERP study of attentional regulation in ADHD, ASD and typical children during Kanizsa shape categorization task demonstrated that differences between ADHD and typical controls were mostly manifested in prolonged latencies of ERP. Although in our experiment delayed latencies in P3 ERP component found its confirmation, the activation itself was increased in ADHD group starting from 250 ms after target onset. This reflects the differences in how cognitive control mechanisms are modu-

lated in two groups during visual attention tasks. This result suggest a possible compensatory mechanism or neural efficiency strategy to cope with cognitive challenges. Interestingly, there were no differences between the groups in early stages of experiment, but they arose closer to the end of task. It seems that the need to utilize different cognitive mechanism grows over time.

In sum, deficient bottom-up visual processing in ADHD group might prevent them from successfully processing Kanizsa shape primers, which in turn leads to an observed deficit in the categorization of salient stimuli and processing of Congruency later on. This is reflected in weaker negative activations in comparison with Control group in parieto-occipital area in response to Incongruent trials. Nevertheless, ADHD group managed to maintain the same speed of performance as the Control group, presumably due to developed compensatory mechanisms. On behavioral level, ADHD demonstrate higher variability than the Control group, driven from occasional periods of mind wandering; on neural level ADHD group mobilize high-level attentional mechanism in a greater extent than the Controls do. It is reflected in increased positive activation in late ERP components in frontal area of the brain. Interestingly, on the latest stages of the experiment we found more neural differences between groups. We might presume that divergences in neural activity become increasingly pronounced over time as the need to employ distinct cognitive mechanisms rises in the ADHD group in order to behaviorally perform at the same level as the Controls.

Conclusion

The study demonstrates that adults with ADHD exhibit significant cognitive and neural differences compared to a Control group in processing visual stimuli during ecologically-valid sustained attention task. The differences between ADHD and Control groups were established both in bottom-up visual detection and more conscious top-down attentional mechanisms, requiring integration and categorisation of visual stimuli. The reason presumably lies in compensatory mechanism used by ADHD group to behaviorally perform on the same level as Control group. Over time neural activity divergences become more pronounced between the groups, indicating the growing need for ADHD group to employ distinct cognitive mechanisms over the length of experiment. This research contributes to a deeper understanding of the neurobiological underpinnings of visual processing and sustained attention, by leveraging a non-neurotypical population as natural control to provide contrast to the neurotypical performance.

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