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Event-Related Potential Study of Attention Regulation During Illusory Figure Categorization Task in ADHD, Autism Spectrum Disorder, and Typical Children

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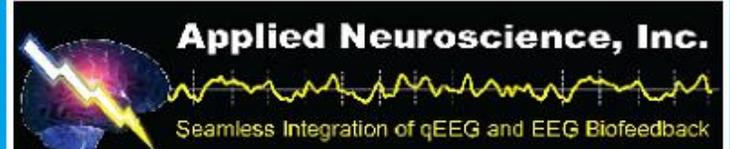
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EVENT-RELATED POTENTIAL STUDY OF ATTENTION REGULATION DURING ILLUSORY FIGURE CATEGORIZATION TASK IN ADHD, AUTISM SPECTRUM DISORDER, AND TYPICAL CHILDREN

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Autism spectrum disorders (ASD) and attention deficit/hyperactivity disorder (ADHD) are very common developmental disorder that share some similar symptoms of social, emotional, and attentional deficits. This study is aimed to help understand the differences and similarities of these deficits using analysis of dense-array event-related potentials (ERP) during an illusory figure recognition task. Although ADHD and ASD seem very distinct, they have been shown to share some similarities in their symptoms. Our hypothesis was that children with ASD will show less pronounced differences in ERP responses to target and nontarget stimuli as compared to typical children and, to a lesser extent, ADHD. Participants were children with ASD ($N=16$), ADHD ($N=16$), and controls ($N=16$). EEG was collected using a 128-channel EEG system. The task involved the recognition of a specific illusory shape, in this case a square or triangle, created by three or four inducer disks. There were no between-group differences in reaction time (RT) to target stimuli, but both ASD and ADHD committed more errors; specifically, the ASD group had statistically higher commission error rate than controls. Posterror RT in ASD group was exhibited in a posterror speeding rather than corrective RT slowing typical for the controls. The ASD group also demonstrated an attenuated error-related negativity as compared to ADHD and controls. The fronto-central P200, N200, and P300 were enhanced and less differentiated in response to target and nontarget figures in the ASD group. The same ERP components were marked by more prolonged latencies in the ADHD group as compared to both ASD and typical controls. The findings are interpreted according to the “minicolumnar” hypothesis proposing existence of neuropathological differences in ASD and ADHD, specifically minicolumnar number/width morphometry spectrum differences. In autism, a model of local hyperconnectivity and long-range hypoconnectivity explains many of the behavioral and cognitive deficits present in the condition, whereas the inverse arrangement of local hypoconnectivity and long-range hyperconnectivity in ADHD explains some deficits typical for this disorder. The current ERP study supports the proposed suggestion that some between-group differences could be manifested in the frontal ERP indices of executive functions during performance on an illusory figure categorization task.

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INTRODUCTION

According to diagnostic criteria enunciated in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev. [DSM-IV-TR]; American Psychiatric Association, 2000), both pervasive disorders of development (PDD) and attention deficit hyperactivity disorder (ADHD) are mutually exclusionary diagnoses. There is a growing consensus from clinicians, however, that behavioral characteristics of ADHD are observed in 14% to 78% of autism spectrum disorder (ASD) patients (Holtman, Bolte, & Poustka, 2007; Keen & Ward, 2004; Lee & Ousley, 2006; Leyfer et al., 2006; Reiersen, Constantino, Volk, & Todd, 2007; Ruggieri, 2006; Sinzig, Walter, & Doepfner, 2009; Yoshida & Uchiyama, 2004). These studies question the validity of comorbidity as an exclusionary criterion within current DSM-IV-TR guidelines and argue in favor of its revision for the upcoming DSM-V (Ruggieri, 2006).

Although behavioral characteristics of autism and ADHD may coexist, the more poignant question is whether both conditions share the same underlying pathophysiology. Without the presence of biomarkers, diagnosis based on observed behaviors is fraught with difficulties. Behaviors are nonspecific, tend to occur in clusters, and may be dependent or exacerbated by physical problems (e.g., urinary retention and pain), drug usage, and personal characteristics (e.g., age, gender, race, level of education). Furthermore, behavioral terms may serve as generic labels to describe a large variety of actions arising from diverse etiologies. The term *agitation*, for example, may be variously used when a patient hits, bites, demands attention, or exhibits repeating mannerisms. Behaviors in this regard may provide little if any insights as to causality.

Evidence derived from the neuropsychological domain relies primarily on behavioral assessments and may thus never be decisive in resolving the ongoing debate about the possible coexistence of autism and ADHD. A recent review of the literature by Gargaro and associates (Gargaro, Rinehart, Bradshaw, Tonge, & Sheppard, 2011) emphasized assessment of

executive functions as a potential probe for eliciting both similarities and differences between the aforementioned conditions. Gargaro et al. surmised that when using independent domains of executive functions, children with autism and those with ADHD exhibit opposite patterns (see also Pennington & Ozonoff, 1996). A recent study in adults similarly revealed potential dissociable features of executive functions between ADHD and ASD based on the Stroop, Matching Familiar Figures, and the Hayling sentence completion tests (Johnston, Madden, Bramham, & Russel, 2011). Not surprisingly, these authors conclude that evidence derived from executive functions assessments remains inconclusive and that further studies are needed (Gargaro et al., 2011; Johnston et al., 2011).

More decisive in settling the possibility of ASD/ADHD comorbidity is evidence derived from research domains other than neuropsychological assessments. Neuropharmacological interventions, for example, have shown that medications targeting hyperactivity and inattention in children with autism, when effective, won't reduce the core symptoms of autism (Hazell, 2007). Thus, core symptoms for both ASD and ADHD seem mediated by different pathophysiological mechanisms. More incisive still to the ongoing debate are data derived from neuroimaging studies. Brain size in autistic subjects appears, on average, to be increased (Stanfield et al., 2008) whereas those in ADHD exhibit a trend toward smaller volumes (Batty et al., 2010). Other investigators have also touched upon differences in gyral complexity, gray white matter parcellation, and size of the corpora callosa (Casanova, El-Baz, Giedd, Rumsey, Switala, 2010; Casanova et al., 2009; El-Baz et al., 2011; Wolosin, Richardson, Hennessey, Denckla, & Mostofsky, 2009). Strikingly, autistic patients, as compared to typical individuals, have larger brains but a smaller corpora callosa. At the opposite end of the spectrum, ADHD patients with smaller brains have a larger corpora callosa. These morphometric measurements imply differences in corticocortical connectivity emphasizing a

bias in short (i.e., arcuate) *versus* long projections (e.g., commissural fibers) that may help explain some of the behavioral manifestations observed in these conditions (Casanova, et al., 2010).

Having a plurality of behavioral coincidences between autism and other neurodevelopmental conditions is to be expected. This is what makes autism (as well as ADHD) a “pervasive” disorder: It affects many cognitive and behavioral domains. However, the coincidence and overlap of symptoms does not necessarily imply a similarity in underlying pathology. Previous studies have shown that ASD and ADHD fall at opposite ends of a spectrum from the standpoint of a number of neuropsychological and neuroimaging considerations (see previously). The present study focuses on the possibility of differing underlying mechanisms in both ASD and ADHD by implementing a series of electrophysiological studies aimed at comparing patterns of evoked cortical responses between these conditions.

Analysis of event-related potentials (ERP) is one of the most informative dynamic methods of investigation and monitoring of information-processing stages in the human brain. Different amplitude and latency characteristics of ERP waves at specified topographies reflect both early sensory perception processes and higher level processing including attention, cortical inhibition, memory update, and other cognitive activity (Duncan et al., 2009; Polich, 2007). ERP provide both a method of studying cognitive processes in normal typical subjects and a tool to assess differences in individuals with neurodevelopmental pathologies.

ERP studies of visual processing commonly employ an “oddball” discrimination task of selective attention in which the participant responds to an infrequent target stimulus among more frequent nontarget stimuli (Vohs et al., 2008). Most investigations into visual processing in ASD have focused on higher level, long-latency ERPs, like the P300 (Courchesne, Courchesne, Hicks, & Lincoln, 1985; Courchesne, Lincoln, Kilman, & Galambos, 1985; Courchesne, Lincoln, Yeung-Courchesne, Elmasian, & Grillon,

1989; Hoeksma, Kemner, Kenemans, & van Engeland, 2006; Kemner, van der Gaag, Verbaten, & van Engeland, 1999; Polich, 2007; Townsend et al., 2001; Verbaten, Roelofs, van Engeland, Kenemans, & Slangen, 1991). The P300 can be divided into the attention-orienting frontal P3a component and the sustained-attention centro-parietal P3b component (Katayama & Polich, 1998; Polich, 2003, 2007). The centro-parietal P300 amplitude (i.e., P3b) has been found to be similar (Courchesne, Courchesne, et al., 1985; Courchesne, Lincoln, et al., 1985; Courchesne et al., 1989; Hoeksma et al., 2006), reduced (Townsend et al., 2001; Verbaten et al., 1991), and augmented (Kemner et al., 1999) in ASD patients to target stimuli compared to controls. Nevertheless, an ERP signature for the autism spectrum disorders has not been identified.

There have been fewer studies on early-stage (i.e., 50–200 ms) visual processing in ASD (see Jeste & Nelson, 2009, for review). In our prior ERP study (Sokhadze, Baruth, Tasman, et al., 2010) on novelty processing in children with ASD, we reported that the ASD group showed significantly higher amplitudes and longer latencies of early ERP components (e.g., P100, N100) to novel distracter stimuli in both hemispheres at the frontal topography. The ASD group also showed prolonged latencies of late ERP components (e.g., N200, P3a) to novel distracter stimuli in both hemispheres. However, differences were more profound in the right hemisphere for both early and late ERP components. Our results indicate augmented and prolonged early frontal potentials and a delayed P3a component to novel stimuli, which suggest low selectivity in preprocessing and later-stage underactivation of integrative regions in the prefrontal cortices. At the centro-parietal topography, the ASD group showed significantly prolonged N100 latencies and reduced amplitudes of the N200 component to target stimuli as compared to controls. The latency of the P3b component was prolonged to novel distracters in the ASD group (Sokhadze, Baruth, Tasman, 2010).

Studies of P300 in ADHD have suggested that children with this diagnosis have attenuated P300 to both auditory and visual stimuli (reviewed in Barry, Johnstone, & Clarke, 2003). In the studies of P300 using continuous performance task in boys with ADHD, Klorman and colleagues (Klorman et al., 1983; Klorman et al., 1979) found that P300 was smaller only in the active condition. In children with ADHD, a decreased P300 at centro-parietal sites has been reported in conjunction with an augmentation at frontal sites (Johnstone & Barry, 1996; Johnstone, Barry, & Anderson, 2001). This result was observed more consistently in children with the combined type of ADHD as compared with the inattentive type, and in childhood than adolescent ADHD (Banaschewski et al., 2003; Banaschewski, Roessner, Dittmann, Santosh, & Rothenberger, 2004; Dimoska, Johnstone, Barry, & Clarke, 2003; Duncan et al., 2009; Smith, Johnstone, & Barry, 2004). In the ADHD population, a relatively small number of ERP studies had concentrated on visual selective attention. Some of these found a smaller early frontal negativity in ADHD as compared to normal controls, suggesting deficiencies in early attention processes (Jonkman, Kenemans, Kemner, Verbaten, & van Engeland, 2004; Satterfield, Schell, & Nicholas, 1994; Van der Stelt, van der Molen, Cunning, & Kok, 2001), whereas no abnormalities were found for the N200. For the P300, the findings were inconsistent, demonstrating no differences in amplitude, a smaller amplitude, or a deviation in scalp distribution (Dimoska et al., 2003; Jonkman et al., 1997; Jonkman et al., 2004; Smith et al., 2004). In the ADHD population, a relatively small number of ERP studies had concentrated on visual selective attention. Some of these found a smaller early frontal negativity in ADHD as compared to normal controls, suggesting deficiencies in early attention processes (Jonkman et al., 2004; Satterfield et al., 1994; Van der Stelt et al., 2001).

Studies using other attention paradigms (e.g., continuous performance, oddball and choice reaction time tasks), have provided evidence for smaller visually evoked

P300 amplitudes (Barry et al., 2003). In sum, several studies found reduced frontal amplitudes in ADHD, which can be taken as suggesting a deficit in selective attention. In autism, only a few studies reported a reduced ERP response to attended visual stimuli. Therefore, the majority of ERP studies have demonstrated altered visual P300 amplitudes in both ADHD and pervasive developmental disorders such as autism. However, these alterations do not seem to be specific markers.

Another important executive function that may differentiate ASD and ADHD is response monitoring and error correction capacity. The error-related negativity (ERN) is a negative-going waveform peaking 40–140 ms after an error response or a negative feedback stimulus (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring & Knight, 2000; Miltner, Braun, & Coles, 1997). This component is thought to reflect a mismatch between actual and intended actions or goals, and therefore occurs in response to unfavorable outcomes, response errors, response conflict, and decision uncertainty (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Further conscious error processing is thought to be reflected by the error Positivity (Pe), which is a positive-going potential following the ERN. Contrary to the ERN, this component does not emerge on trials where the subject is unaware of his or her committed error (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; O'Connell et al., 2007; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). Several studies have suggested that the Pe is a P3(b) response to the processing of errors (Davies, Segalowitz, & Gavin, 2004; Leuthold & Sommer, 1999; O'Connell et al., 2007; Overbeek et al., 2005).

Henderson et al. (2006) were the first to conduct a study on performance monitoring in children diagnosed with ASD. They could not reveal overall differences in ERN amplitude between the ASD and typically developing groups, but they found that within the ASD group larger ERN amplitudes were predictive of a smaller impairment in social interaction as well as of decreased internalizing problems.

Performance studies have suggested deficits in error correction in autism. Russell and Jarrold (1998), for example, found that autistic children were more likely to fail correcting errors than controls, both when they were provided with visual feedback about their errors and when they had to detect their errors themselves. Moreover, Bogte, Flamma, van der Meere, and van Engeland (2007) found that a group of adult autistic subjects showed no post error slowing, whereas a control group did. These studies suggest decreased error awareness in autism, predicting decreased Pe amplitudes.

In our prior study (Sokhadze, Baruth, El-Baz, et al., 2010), we examined the possibility that children with ASD exhibit a deficiency in the processing of error, reflected by a reduction and delays in the ERN and Pe response-locked brain potentials. Our results showed that, as expected, ASD patients had high rate of errors in the visual oddball task with novel distracters. In addition, in neurodevelopmentally normal subjects, it has been observed that after an error has been committed, subjects show slower reaction time (RT) and decreased error rates. These changes have been interpreted as revealing changes in the speed-accuracy strategy of the subject possibly due to error-induced control processes and concomitant corrective adjustments. The patients with ASD showed opposite response: faster post-error RT instead of slowing down. We also found lower ERN amplitude and prolonged Pe in ASD compared to typical controls. The reduced ERN and altered Pe, along with a lack of posterror RT slowing in autism, was interpreted as an insensitivity to detect and monitor response errors and reduced ability of execute corrective actions (Sokhadze, Baruth, El-Baz, et al., 2010). Results were indicative of reduced error awareness, and a failure in adjustment in ASD when dealing with situations where erroneous responses may occur.

Findings on the ERN amplitude in ADHD are inconsistent. Two studies have found reduced ERN amplitudes in children with ADHD compared to typically developing children, suggesting that they have a deficit in

monitoring ongoing behavior (Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005; Van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007). Reduced Pe amplitudes in ADHD are in accordance with the findings of reduced posterror compensatory behavior, that is, the strategic RT slowing after the commission of errors (Schachar et al., 2004; Sergeant & Van der Meere, 1988; Wiersma, van der Meere, & Roeyers, 2005). Reduced error awareness may thus hamper children with ADHD in adequately adapting their behavior and consequently in learning from their mistakes.

The goal of this study was to investigate stimulus- and response-locked ERP during performance on a visual three-category oddball task with illusory figure stimuli in children with ASD, children with ADHD, and typically developing children. We proposed that behavioral and electrocortical evoked potentials will differentiate ASD, ADHD, and control groups. We expected to see more pronounced between group differences at the frontal topography, considering that both ADHD and ASD typically present executive function deficits. The study was guided by the "minicolumnar morphometry" hypothesis (Williams & Casanova, 2010) that considers ADHD and ASD as conditions with differing etiology of neurodevelopmental pathology.

METHODS

Participants

Participants with ASD (age range = 9–20 years) were recruited through the University of Louisville Weisskopf Child Evaluation Center. Diagnosis was made according to the *DSM-IV-TR* (APA, 2000) and further ascertained with the Autism Diagnostic Interview-Revised (Le Couteur, Lord, & Rutter, 2003). They also had a medical evaluation by a developmental pediatrician. All subjects had normal hearing based on past hearing screens. Participants either had normal vision or wore corrective lenses. Participants with a history of seizure disorder, significant hearing or visual impairment, a brain abnormality conclusive from imaging

studies, or an identified genetic disorder were excluded. All participants were high-functioning persons with ASD with Full-Scale IQ greater than 80 assessed using the Wechsler Intelligence Scale for Children, Fourth Edition (Wechsler, 2003) or the Wechsler Abbreviated Scale of Intelligence (Wechsler, 2004).

Controls were recruited through advertisements in the local media. All control participants were free of neurological or significant medical disorders; had normal hearing and vision; and were free of psychiatric, learning, or developmental disorders based on self- and parent reports. Subjects were screened for history of psychiatric or neurological diagnosis using the Structured Clinical Interview for *DSM-IV* Non-Patient Edition (First, Spitzer, Gibbon, & Williams, 2001). Participants within the control, ADHD, and autism groups were attempted to be matched by age, Full-Scale IQ, and socioeconomic status of their family. Socioeconomic status of ASD, ADHD, and control groups was compared based on parent education and annual household income. Participants in three groups had similar parent education levels.

Participating subjects and their parents (or legal guardians) were provided with full information about the study including the purpose, requirements, responsibilities, reimbursement, risks, benefits, alternatives, and role of the local Institutional Review Board. The consent and assent forms approved by the Institutional Review Board were reviewed and explained to all subjects who expressed interest to participate. All questions were answered before consent signature was requested. If the individual agreed to participate, she or he signed and dated the consent form and received a copy countersigned by the investigator who obtained consent.

The mean age of 16 participants who were enrolled in the ASD group was $12.6 \pm (SD) 2.3$ years (range = 9–17 years; 14 male, two female), and the mean age of the ADHD group was 13.2 ± 2.5 years ($N = 16$, range = 9–17 years; 14 male, two female). The mean age of the control (CNT) group ($N = 16$) was 14.6 ± 3.9 years (9–20 years; 13 male, three

female). The age difference between groups was not significant, $F(2, 45) = 1.91$, $p = .16$, ns). The mean Full-Scale IQ scores were 95.35 ± 19.11 for patients with ASD and 98.45 ± 9.77 for children with ADHD. The tests were Full-Scale IQ scores from the Wechsler Intelligence Scale for Children, Fourth Edition (Wechsler, 2003). Nine subjects from the ADHD group and 10 subjects from the ASD group were on medication. Children with ADHD were taking stimulants such as Ritalin (methylphenidate) or Adderall (dextro-amphetamine). Two children with ASD were taking stimulants (Ritalin, Concerta, Adderall, etc.), and eight were taking antidepressants (Prozac [fluoxetine], Zoloft [sertraline]) and mood stabilizers (Depakote [divalproex], Abilify [ariprazole]). Three children in the ASD group had comorbid mild mood disorders, and four had anxiety disorders. Two subjects from the ADHD group had comorbid mild mood disorders, and another two had anxiety disorders.

ERP Data Acquisition and Signal Processing

Electroencephalographic (EEG) data were acquired with a 128-channel Electrical Geodesics Inc. (EGI) system (v. 200) consisting of Geodesic Sensor Net electrodes, Net Amps, and Net Station software (Electrical Geodesics Inc., Eugene, OR). EEG data are sampled at 500 Hz and 0.1 1–200 Hz analog filtered. Impedances were kept under 40 K Ω . According to the *Technical Manual of EGI* (Electrical Geodesics, Inc., 2003), this Net Sensor electrode impedance level is sufficient for quality recording of EEG with this system. A study conducted by Ferree, Luu, Russell, and Tucker (2001) suggested that modern high input-impedance amplifiers and accurate digital filters for power noise provide excellent EEG signal collection with high scalp impedance (approximately 40 K Ω).

The Geodesic Sensor Net is a lightweight elastic thread structure containing Ag/AgCl electrodes housed in a synthetic sponge on a pedestal. The sponges are soaked in a KCl solution to render them conductive. EEG data

were recorded continuously. EEG channels with high impedance or visually detectable artifacts (e.g., channel drift, gross movement, etc.) were identified using Net Station event marker tools in “on-line” mode and removed in the “off-line” mode using Net Station Waveform Tools (NSWT). Stimulus-locked EEG data were segmented off-line into 1,000 ms epochs spanning 200 ms prestimulus to 800 ms poststimulus around the critical stimulus events, for example, in an oddball task: (a) rare target (Kanizsa square), (b) rare nontarget (Kanizsa triangle), (c) frequent nontarget (non-Kanizsa standards). Response-locked EEG data (for ERN and Pe analysis) are segmented off-line into 1,000 ms epochs spanning 500 ms prestimulus to 500 ms poststimulus around the critical stimulus events or commission error.

Data were digitally screened for artifacts (eye blinks, movements), and contaminated trials are removed using artifact rejection tools. The NSWT’s “Artifact Detection” module in “off-line” mode marked EEG channels “bad” if fast average amplitude exceeded $200\ \mu\text{V}$, differential average amplitude exceeds $100\ \mu\text{V}$, or if the channel has zero variance. Segments were marked “bad” if they contain more than 10 bad channels or if eye blinks or eye movements were detected ($>70\ \mu\text{V}$). After detection of bad channels, the NSWT’s “Bad channel replacement” function was used for the replacement of data in bad channels with data interpolated from the remaining good channels (or segments) using spherical splines (more information on interpolation methods used in EGI Net Station systems can be found in Fletcher, Kussmaul, & Mangun, 1996; Luu et al., 2001; Perrin, Pernier, Bertrand, Giard, & Echallier, 1987; Srinivasan, Tucker, & Murias, 1998).

The remaining data set was digitally filtered using 60 Hz Notch and 0.3–20 Hz bandpass filters, and were then segmented by condition and averaged to create ERPs. Averaged ERP data were baseline corrected and re-referenced into an average reference frame. All stimulus presentation and behavioral response collection was controlled by a PC computer running

E-prime software (Psychology Software Tools Inc., PA). Visual stimuli are presented on a 15-in. display. Manual responses were collected with a five-button keypad (Serial Box, Psychology Software Tools, Inc., Sharpsburg, PA).

Three Stimuli Visual Oddball with Illusory Kanizsa Figures

In this task, subjects responded with a button-press to rare (25% probability) Kanizsa squares (targets) among Kanizsa triangles (rare nontarget distracters, 25% probability) and non-Kanizsa figures (standards, 50% probability). The stimuli were presented for 250 ms with intertrial intervals varying in the range of 1,100 to 1,300 ms. A fixation point (cross) was presented during intertrial intervals (Figure 1). White figures were displayed on a black background on a flat monitor. Subjects were instructed to press the first button on a five-keypad with their right index finger when a target appears and to ignore nontarget Kanizsa or standard stimuli. The nontarget Kanizsa triangle was introduced to differentiate processing of Kanizsa figures and targets. The stimuli consisted of either three or four inducer disks, which are considered the shape feature, and they constitute either an illusory figure (square, triangle) or nonillusory figure (collinearity feature).

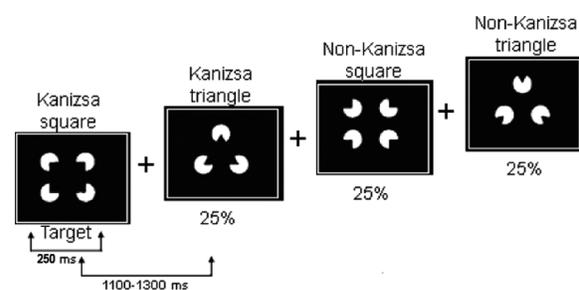


FIGURE 1. In this experiment we used Kanizsa and non-Kanizsa figures as stimulus material. In particular, the stimulus types are Kanizsa square (target), Kanizsa triangle, non-Kanizsa square, and non-Kanizsa triangle. The nontarget Kanizsa triangle is introduced to differentiate processing of Kanizsa figures and targets. The stimuli consist of either three or four inducer disks, which are considered the shape feature, and they either constitute an illusory figure (square, triangle) or not (collinearity feature).

Behavioral Measures

Behavioral response measures were mean reaction time (in milliseconds) and response accuracy (percentage of correct hits). Both commission and omission error rates were calculated.

ERP

Response-Locked ERPs Response-locked ERP dependent measures were adaptive mean amplitude and latency of two ERP peaks (i.e., ERN, Pe) within a temporal window across two region-of-interest (ROI) channel groups at the midline fronto-central area. Each ROI contained at least four electrodes. A list of ERP-dependent variables included stimulus-averaged amplitude and latency of the fronto-central ERP components: ERN (40–150 ms poststimulus) and error-related positivity (Pe; 100–200 ms).

The frontal and fronto-central ROIs for both ERN and Pe components included the following EGI channels: midline frontal and fronto-central ROI—Fz and FCz; and the extended fronto-central ROI contained five EEG sites—FCz, two left EGI channels 7 and 13 (between FCz and FC3 and C1) and two right EGI channels 113 and 107 (between FCz and FC2 and C2).

Stimulus-Locked ERPs Stimulus-locked ERP dependent measures were adaptive mean amplitude and latency of ERP peak (e.g., N100) within a temporal window across an ROI channel group. Each ROI contained at least four electrodes. A list of ERP dependent variables included stimulus-averaged amplitude and latency of the frontal ERP components: N100 (90–180 ms), P200 (180–280 ms), N200 (200–320 ms), and P300 (P3a, 300–500 ms), and the posterior (centro-parietal and parieto-occipital ROIs) ERP components N100 (80–180 ms), N200 (180–300 ms), and P300 (300–500 ms). The frontal (i.e., frontal and fronto-central) ROIs for N100, P200, N200, and P300 components included the following EGI channels: left ROI—EGI channel 29, F3, FC1, FC3; midline ROI—Fz, FCz, EGI channels 5, 12; right ROI—EGI channel 118, F4, FC2, FC4.

The parietal (i.e., centro-parietal and parieto-occipital) ROIs for N100, and P200 components included following EGI channels: left ROI—EGI channel 67, PO3, PO7, O1; right ROI—EGI channel 78, PO4, PO8, O2. Midline parietal (Pz) and parieto-occipital (POz) channels were used in combination with the left and right parieto-occipital ROIs to form a comprehensive parieto-occipital ROI containing 10 EEG channels. For centro-parietal N200 and P300 (P3b) were used channels P1, P3, PO3, EGI channel 54 and 67 (left) and P2, P4, PO4, EGI channels 78 and 80 (right). Midline parietal channels included Pz and POz.

Statistical Data Analysis

Statistical analyses were performed on the subject-averaged behavioral and ERP data with the subject averages being the observations. The primary analysis model is the repeated measures analysis of variance (ANOVA), with dependent variables being RT, accuracy, error rate, and all the specific ERP components' amplitudes and latencies at selected ROIs. The data of stimulus-locked ERP dependent variable for each relevant ROI were analyzed using an ANOVA with the following factors (all within-participants): *stimulus* (target, standard, nontarget Kanizsa), *hemisphere* (left, right), and so on. The between-subject factor was *group* (ADHD, ASD, CNT). The data of each response-locked ERP dependent variable for relevant midline frontal ROI were analyzed using a one-way ANOVA. Post hoc analysis using a Tukey test was conducted where appropriate. A priori hypotheses were tested with student's *t* tests for two groups with unequal variance. In all ANOVAs, Greenhouse-Geisser corrected *p* values were employed where appropriate.

RESULTS

Behavioral Responses

RT to targets was not significantly different between groups of subjects (471 ± 89 ms in ASD vs. 434 ± 137 ms in ADHD vs. 466 ± 88

ms in CNT), $F(2, 45) = 0.54, p = .581, ns$). A difference in total error rate in the ASD and ADHD groups versus the CNT group was significant, $F(2, 45) = 4.63, p = .015$. A post hoc Tukey Honestly Significant Difference (HSD) test showed the following between group differences: $19.7 \pm 20.4\%$ in ASD versus $2.6 \pm 4.5\%$ in CNT ($p = .011$), but the ADHD versus CNT difference (9.24%) was not significant ($p = .236$). The percentage of commission errors was significantly lower in the typical children ($1.9 \pm 3.7\%$ in CNT vs. $16.8 \pm 19.7\%$ in ASD and $10.2 \pm 16.1\%$ in ADHD), $F(2, 45) = 4.04, p = .024$. Post hoc analysis showed an ASD versus CNT significant difference ($-14.9\%, p = .018$). The difference in omission errors ($2.8 \pm 3.4\%$ in ASD vs. $1.6 \pm 2.4\%$ in ADHD vs. $0.7 \pm 1.4\%$ in CNT) did not reach statistical significance, $F(2, 45) = 2.78, p = .073$. Mean posterror RT was different across groups, $F(2, 45) = 9.20, p < .001$; in particular, CNT and ADHD groups showed an increase of mean RT following committed errors (24.4 ± 65.2 ms in CNT and 7.6 ± 40.4 ms in ADHD), whereas the ASD group showed a decrease of posterror RTs (-52.6 ± 48.7 ms). This difference was confirmed by post hoc analysis both for ASD versus CNT (-77.0 ± 18.9 ms, $p = .001$) and ASD versus ADHD pairs (-60.0 ± 19.2 ms, $p = .009$). See Figure 2.

Response-Locked ERPs: Error-Related Negativity and Positivity

Amplitude. Amplitude of the ERN across a five frontal and fronto-central ROI showed significant differences ($-3.79 \pm 6.83 \mu\text{V}$ in ADHD, $0.44 \pm 7.29 \mu\text{V}$ in ASD, and $-5.81 \pm 5.12 \mu\text{V}$ in CNT), $F(2, 41) = 7.62, p = .002$. The ERN at midline fronto-central channel ROI showed the same effect, $F(2, 41) = 3.76, p = .031$. Post hoc analysis showed that the ERN amplitude in the ASD group as compared to controls was significantly less negative (-6.25 ms, $p = .001$). Amplitude of Pe between groups was not significantly different ($p = .36, ns$).

Latency. Latency of the ERN showed between group differences ($F(2, 41) = 3.44,$

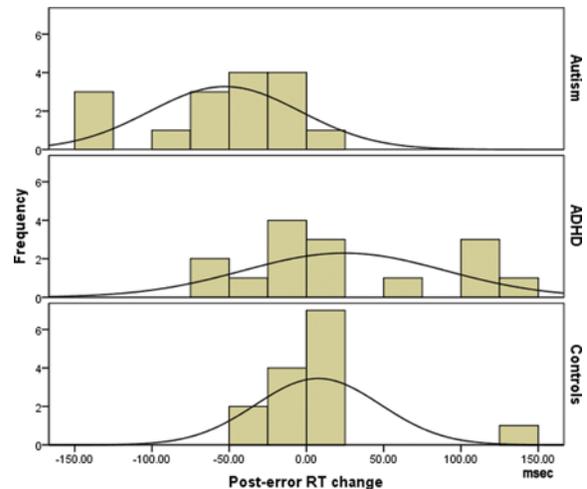


FIGURE 2. Histogram of distribution of individual posterror reaction time (RT) in children with autism, children with ADHD, and typically developing controls ($N = 16$ per group). Both ADHD and control groups demonstrate slower (positive) posterror RTs compared to correct response RTs. The ASD group shows speeding of posterror RTs with a negative peak of distribution curve. The ADHD shows high variability and relatively flat curve of posterror RTs as compared to controls. (Color figure available online.)

$p = 0.042$). Latency was prolonged in the ASD group (midline Fz-FCz ROI, 126 ± 32 ms in ASD vs. 89 ± 51 ms in CNT vs. 107 ± 28 ms in ADHD, $F(2, 41) = 4.97, p = 0.035$; five channel ROI respectively, 123 ± 22 ms vs. 88 ± 48 ms vs. 112 ± 36 in ADHD; $F(2, 42) = 3.50, p = 0.039$). Post hoc Tukey HSD test confirmed significant differences between ASD and CNT groups (Fz-FCz, $p = 0.032$; for 5 channel ROI $p = 0.035$). Latency of Pe across midline frontal and fronto-central channels did not yield any significant differences. See Figure 3.

Stimulus-Locked ERPs

Frontal ERPs

N100. Amplitude of the frontal N100 to targets at midline and over the right hemisphere showed significant group differences (at midline ROI), $F(2, 45) = 4.62, p = .015$; right frontal ROI, $F(2, 45) = 3.65, p = .034$. Post hoc analysis showed that the midline N100 was significantly more negative in the ASD group compared to controls ($-3.31 \pm 1.99 \mu\text{V}$ vs. $-1.56 \pm 1.41 \mu\text{V}, p = .018$). The amplitude of

Error-related negativity (ERN) and positivity (Pe) in 3 groups (Control, Autism, ADHD) at the midline fronto-central site (FCz)

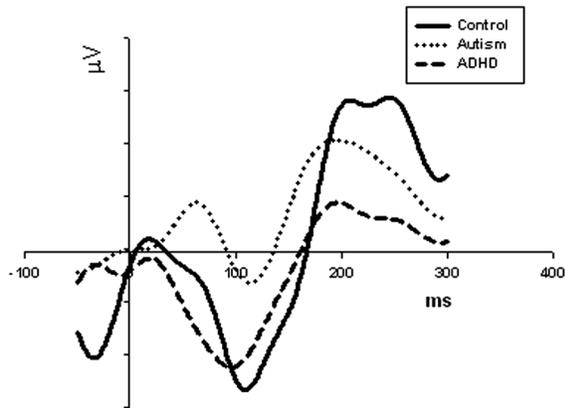


FIGURE 3. Error-related negativity (ERN) and positivity (Pe) from the frontal midline EEG site in three groups (control, ASD, ADHD). Grand average waveforms ($N=16$ per group) show more negative amplitude and shorter latency of the ERN in control and ADHD groups as compared to the ASD group.

the midline frontal N100 was also more negative to standards in the ASD group as compared to controls ($-3.07 \pm 1.99 \mu\text{V}$ vs. $-1.34 \pm 1.42 \mu\text{V}$, $p = .028$). Multiple post hoc comparisons showed that the amplitude of N100 at all frontal sites of interest across all three conditions (target Kanizsa, standard, rare nontarget Kanizsa) was significantly more negative in

the ASD ($-2.93 \mu\text{V}$ vs. $1.71 \mu\text{V}$ in ADHD vs. $-1.55 \mu\text{V}$ in CNT group ($p \leq .05$) as the ASD group exhibited comparable amplitudes to each category of stimuli. Latency of the frontal N100 to targets over the midline showed group differences, $F(2, 45) = 3.29$, $p = .046$, with shorter latency in the ASD and CNT groups. The latency in the ADHD group was delayed as compared to controls (147.3 ± 25.8 ms vs. 131.2 ± 10.3 ms, $p = .024$).

P200. No between-group differences were found in amplitude and latency of the frontal P200. See Figure 4.

N200. Amplitude of the midline frontal N200 did not show any between group differences. The latency of N200 to targets was significantly prolonged in the ADHD group compared to controls (at the midline 29.4 ± 10.4 ms difference, $p = .02$), whereas at the left frontal ROI, it was 26.2 ± 10.4 ms longer than in controls ($p = .04$) and 30.0 ± 10.4 ms longer than in the ASD group ($p = .016$).

P300 (P3a). Amplitude of the midline frontal P3a component showed clear group differences for all conditions, standards, $F(2, 45) = 4.45$, $p = .017$; nontarget Kanizsa, $F(2, 45) = 4.29$, $p = .02$; target Kanizsa, $F(2, 45) = 5.19$, $p = .009$. Post hoc analysis showed that the amplitude across all stimuli was lower

Frontal ERP to target (left) and non-target Kanizsa stimuli (N=16/per group)

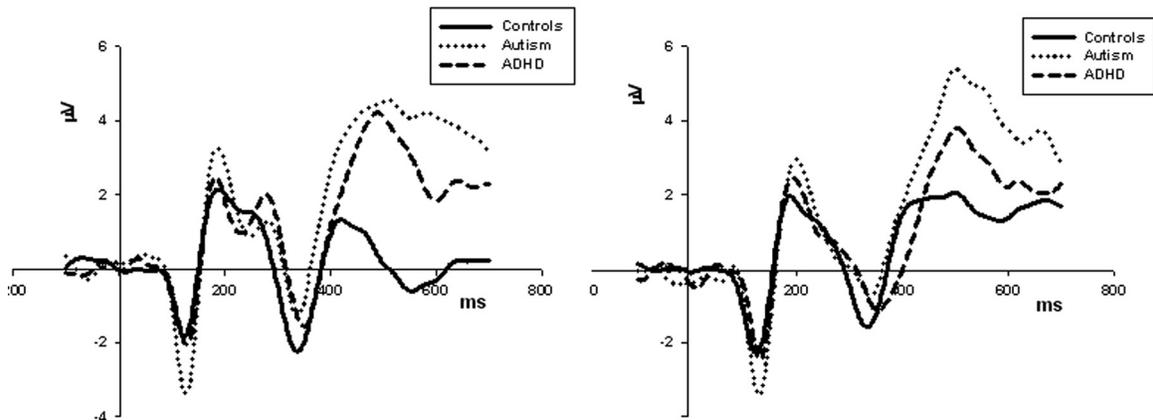


FIGURE 4. Grand average frontal ERP to target (left) and nontarget (right) Kanizsa stimuli in ASD, ADHD, and control groups ($N = 16$ per group). The ASD group shows more negative N100 component both to targets and non-targets, while the ADHD group has delayed N200 component both to target and nontarget stimuli as compared to controls. Both ASD and ADHD present higher and delayed P3a peak as compared to controls.

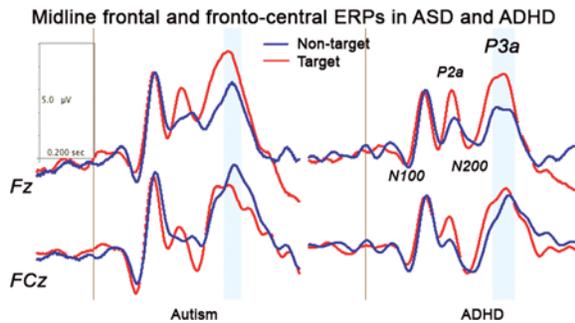


FIGURE 5. Midline frontal (Fz) and fronto-central (FCz) ERPs to target and nontarget Kanizsa stimuli in ASD and ADHD ($N = 16$ per group). (Color figure available online.)

in the control group as compared to both ASD ($-1.65 \pm 0.63 \mu\text{V}$, $p = .034$) and ADHD ($-2.33 \pm 0.69 \mu\text{V}$, $p = .002$) groups. For all stimuli, the group difference yielded a significantly lower amplitude in the controls compared to the ASD group only (standards, $-1.98 \pm 0.73 \mu\text{V}$, $p = .027$, rare nontargets, $-2.67 \pm 0.91 \mu\text{V}$, $p = .015$; targets, $-2.34 \pm 0.74 \mu\text{V}$, $p = .008$).

Latency of the frontal P3a at the midline showed significant group differences (standards, $F(2, 45) = 3.30$, $p = .045$; nontarget Kanizsa, $F(2, 45) = 6.75$, $p = .003$; targets, $F(2, 45) = 3.51$, $p = .038$). Post hoc analysis showed a significant delay of P3a latency in the ADHD group compared to controls in response to standard (-48.4 ± 19.7 ms, $p = .046$) and nontarget Kanizsa stimuli

(-61.3 ± 16.7 ms, $p = .002$). Similar differences were found for the left but not for the right frontal EEG recording sites. A Hemisphere (left, right) \times Group (CNT, ASD, ADHD) interaction was significant, $F(2, 45) = 3.28$, $p = .047$, and was expressed as a more profound delay of the P3a peak in the ADHD group over left hemisphere. A post hoc Tukey HSD test yielded a significantly prolonged latency in ADHD versus controls across all stimulus categories (52.7 ± 16.6 ms, $p = .008$). See Figure 5.

Parietal ERPs

N100, P200, and N200. There were no significant between-group differences were found for amplitude and latency of the parieto-occipital N100 and P200 as well as parietal N200.

P300 (P3b). The amplitude of P3b did not show any statistically significant between-group differences. The latency of the P3b to targets at the right hemisphere showed group differences, $F(2, 45) = 4.45$, $p = .017$, but post hoc analysis confirmed only ASD versus CNT group differences (shorter latency in the ASD, -33.7 ± 11.3 ms, $p = .013$). At the same right centro-parietal site the ASD group also showed a marginally shorter latency in response to the rare Kanizsa nontargets (-26.7 ± 10.9 ms, $p = .049$) as compared to the ADHD group. See Figures 6 and 7.

Centro-parietal ERP to target (left) and non-target Kanizsa stimuli in ASD, ADHD and control subjects ($N=16$ /per group)

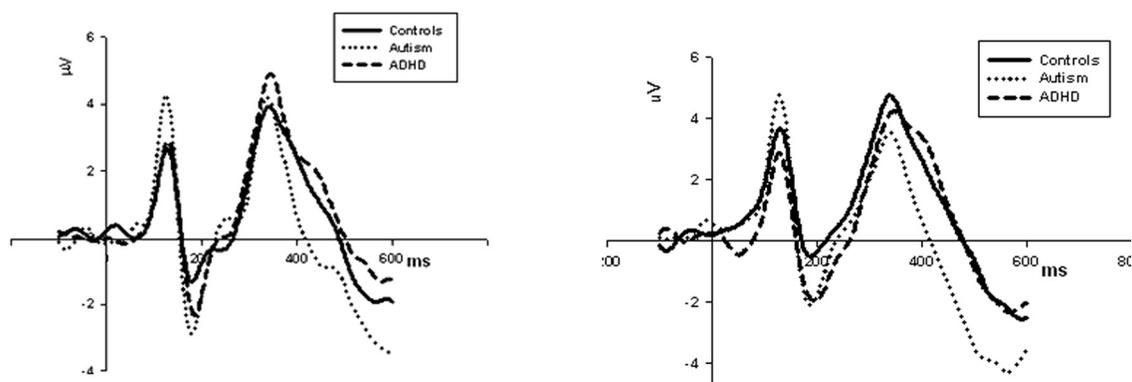


FIGURE 6. Centro-parietal ERP to target (left) and nontarget (right) Kanizsa stimuli in ASD, ADHD, and control subjects ($N = 16$ per group). The ASD group has shorter P3b latency as compared to controls.

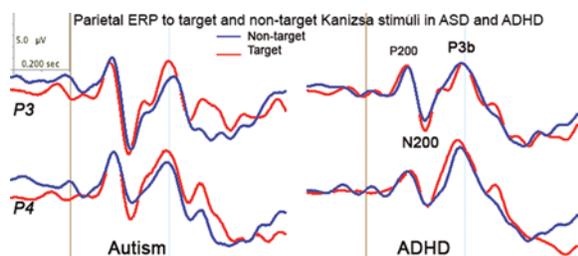


FIGURE 7. Parietal (P3,P4) ERP to target and non-target Kanizsa stimuli in ASD and ADHD. The latency of the P3b component is shorter in the ASD group. (Color figure available online.)

DISCUSSION

Our results show that children with ASD and ADHD do not differ on mean RT, but they commit more errors. Furthermore, children with ASD do not present normative posterror slowing of RT indicative of impaired error correction capacity. Response-locked ERN is also less negative and prolonged as compared to both ADHD and control groups. At the frontal topography, the ASD group showed higher early ERP peaks magnitude to nontarget stimuli (i.e., standards, nontarget Kanizsa figures) as compared to controls. The ADHD group showed delayed latency of the frontal N100, N200, and P3a to targets. The delayed P3a component in the ADHD was better manifested at the left hemisphere. At the posterior topography, the ASD group as compared to the control group showed shorter latency of P3b to both target and nontarget Kanizsa items.

In the ADHD group the latency of the P3b component to targets at the right hemisphere was prolonged as compared to the ASD group. Thus we found group differences predominantly in frontal ERP components indicating that the neurodevelopmental groups exhibit frontal function deficits. Most behavioral and ERP measures in this study show that the ASD group is significantly different from controls and on many measures also is to a lesser extent different from ADHD group. The more pronounced was the difference in reactivity to nontarget items. Autistic children showed excessive response to frequent standards and rare nontarget distracters. Differences between

ADHD and typical controls were minimal and were mostly manifested in prolonged latencies of ERP.

Shorter latency and higher amplitude of the early frontal negativity (N100) in the autism group with minimal differentiation of response magnitude to either target or nontarget stimuli is an interesting finding that replicates our earlier report (Sokhadze et al., 2009) where a different visual oddball task was used. Visual processing is based on a core system consisting of occipito-temporal regions in extrastriate visual cortex (Haxby, Hoffman, & Gobbini, 2002) although parietal (Posner & Petersen, 1990) and frontal (Clark, Fan, & Hillyard, 1995) regions also play a role in directing visual attention.

The visual N100 is similarly considered an index of stimulus discrimination (Hopf, Vogel, Woodman, Heinze, & Luck, 2002; Vogel & Luck, 2000). The N100 is generally defined within a time window starting as early as 70 ms poststimulus onset (Courchesne, Lincoln, et al., 1985) to as late as 180-ms poststimulus onset (Tendolkar et al., 2005). Over posterior electrode sites the visual N100 is probably generated by dipoles in lateral extrastriate cortex (Gomez-Gonzales, Clark, Fan, Luck, & Hillyard, 1994) with a contribution from parieto-occipital and occipito-temporal areas (Hopf et al., 2002; Yamazaki et al., 2000), whereas the visual N100 over frontal electrode sites most likely is reflective of frontal generators (Clark et al., 1995). The visual N100 generally is augmented during attentional stimulus processing, which is also known as the "N1-effect" (Hillyard, Hink, Schwent, & Picton, 1973), and is larger toward task-relevant target stimuli (Hillyard, Mangun, Woldorff, & Luck, 1995; Luck, Heinze, Mangun, & Hillyard, 1990).

The visual P200 over frontal electrode sites is generally found in a latency range of 180 to 320 ms poststimulus and has been reported in working memory and attention tasks. Kenemans, Kok, and Smulders (1993) described this frontal positivity as a component that indexes the hierarchical selection of task-relevant features for further processing.

The visual P200 over posterior regions has been studied less but likely is associated with generators in the primary visual cortex and extrastriate areas reflecting visual categorization processes. We could not find any significant group differences between ASD, ADHD, and controls on this measure. The visual N200 is a negative endogenous ERP component directly following the P200; it is mainly found in a latency range of 180 to 350 ms post-stimulus over centro-parietal scalp locations (Näätänen, Gaillard, & Mäntysalo, 1978; Näätänen, Schröger, Karakas, Tervaniemi, & Paavilainen, 1993) but can be isolated over frontal regions as well. The visual N200 component is associated with categorization, perceptual closure, and attention focusing ultimately signaling that a perceptual representation has been formed (Potts, Patel, & Azzam, 2004); it is enhanced if the presented stimulus contains a perceptual feature or attribute defining the target in the task. Over frontal channels the N200 can provide information about processes related to response conflict detection and processing, as well as inappropriate response inhibition (West, 2003; West, Bowry, & McConville, 2004). It is thought to originate from the anterior cingulate cortex (ACC) and prefrontal sources (Donkers & van Boxtel, 2004).

The P300 directly follows the N200 and is one of the most studied ERP components. It is elicited when a subject detects an unexpected (novel, rare) stimulus and consists of two components labeled P3a (fronto-central P300) and P3b (centro-parietal P300). The P3a (sometimes referred to as the novelty P300) is a fronto-central wave occurring within a time window of 300 to 520 ms; it reflects an aspect of the orienting response and has been related to evaluative attentional processes (Hruby & Marsalek, 2003; Polich, 2003). The ASD group shows clearly augmented and delayed frontal P3a that might result on impaired early differentiation of target and nontarget items (e.g., on N100 stage) and more effortful compensatory strategies involved for successful target identification and correct motor response selection. In general, the autistic group showed

prolonged latencies to standard and rare nontarget illusory figures. These results suggest that individuals with autism probably overprocess information needed for the successful differentiation of target and distracter stimuli. One of the possible explanations might be sought in the local hyperconnectivity hypothesis of autism.

The P3b is a centro-parietal wave occurring between 320 and 560 ms that has been linked to task-relevance and the decision-related character of the eliciting stimulus; it reflects memory-updating processes and/or processing closure (Picton, 1992). Source localization techniques have claimed that multiple brain areas are involved in the generation of the visual P3b: the hippocampus and parahippocampal areas, the insula, the temporal lobe, occipital cortex, and the thalamus (Goto, Brigell, & Parmeggiani, 1996; C. S. Herrmann & Knight, 2001; Mecklinger et al., 1998; Rogers, Basile, Papanicolaou, & Eisenberg, 1993). Most studies agree that the P3b has multiple dipole sources (Halgren, Marinkovic, & Chauvel, 1998; Knight, 1997; Townsend et al., 2001). Considering that most studies on P3b in ADHD report attenuated amplitude and prolonged latency of this cognitive component (Banaschewski et al., 2003; Banaschewski et al., 2004; Barry et al., 2003; Dimoska et al., 2003; Duncan et al., 2009; Jonkman et al., 2004; Satterfield et al., 1994; Smith et al., 2004; Van der Stelt et al., 2001), our finding of shorter latencies to targets in the ASD group definitely deserves interest and further studies. However, our study found only minimal group differences in posterior ERP components, as most ERP differences were at the anterior (frontal and fronto-central) topographies.

Comparison of behavioral (RT, accuracy, posterror slowing) and electrocortical (ERN/Pe) indices of error processing in children with ASD, ADHD, and in typical children allows to judge about behavioral self-regulation ability, which is so important in monitoring of ongoing behavior and adaptive control. Our recent study reported on several deficits in error monitoring function in autism (Sokhadze,

Baruth, El-Baz, et al., 2010). Several studies addressed neural correlates of error processing and behavioral performance monitoring measures in children and adults with ADHD (Burgio-Murphy et al., 2007; Groom et al., 2010; M. J. Hermann et al., 2010; Liotti et al., 2005). Furthermore, Groen et al. (2008) study used ERN/Pe using ERP technique considering error processing specifics as a useful method for dissociating ADHD from ASD and elucidating pharmacotherapy effects on performance monitoring in ADHD. Our prior study (Sokhadze, Baruth, El-Baz, 2010) also discusses error-processing measures as useful biomarkers of executive dysfunctions in children with ASD. The current study contributes to this investigation by adding ADHD group as a contrast (in addition to typically developing children).

The neuronal source of ERN has been localized in the ACC (see, for a review, Taylor, Stern, & Gehring, 2007). The ERN is hypothesized to reflect phasic ACC activity in response to reinforcement signals from the mesencephalic dopamine system that serves as a trigger for further processing of the event and further deliberate compensatory behavior (Holroyd & Coles, 2002).

Our results show significant differences both in behavioral and electrocortical responses between ASD, ADHD, and typical controls during performance on illusory figure test. The findings are interpreted according to the "minicolumnar" hypothesis proposing existence of neuropathological differences in ASD and ADHD, in particular minicolumnar number/width morphometry spectrum differences. In autism, a model of local hyperconnectivity and long-range hypoconnectivity explains many of the behavioral and cognitive deficits present in the condition, while the inverse arrangement of local hypoconnectivity and long-range hyperconnectivity in ADHD explains some deficits typical for this disorder (Williams & Casanova, 2010). According to Williams and Casanova, conditions like dyslexia, ADHD, and autism defined by the different intra- (i.e., local) and intercortical (i.e., long-range) connectivity should be considered

as polar extremes of their minicolumnar morphometry. Casanova, Buxhoeveden, and Brown (2002) proposed that minicolumns exist within a phenotypic spectrum that affects the inhibitory/excitatory ratio and hence flow of information in neocortical circuits (see also Rubenstein & Merzenich, 2003). Because local- and long-range cortical coordination is a finely tuned relationship of the signal-to-noise ratios, extremes of either edges of the spectrum can disrupt functionality and result in similar behavioral manifestations (e.g., attention deficits) despite opposing underlying etiologies in autism and ADHD. Following the hypothesis suggested in Williams and Casanova (2010) while considering dyslexia and autism conditions, it is possible to propose that ASD and ADHD are two conditions that share aspects which are also cortical inversions of one another and can be considered while trying to explain why some children with ASD may present with attention disorders similar to those seen typically in ADHD.

The current ERP study supports the proposed suggestion that some between-group differences could be manifested in the frontal ERP indices of executive functions during performance on an illusory figure categorization task. As it was stated in a recent review on ASD and ADHD phenotypes by Rommelse, Geurts, Franke, Buitelaar, and Hartman (2011),

Over the past decades, ASD and ADHD have been studied in isolation from each other, each disorder within its research tradition, networks of collaborating experts and theoretical frameworks, without much cross-fertilization. We argue that much can be gained when ASD and ADHD are studied together. (p. 1382)

Our study suggests that that looking for quantitative EEG and ERP biomarkers of executive function abnormalities and other behavioral performance deficits present in ASD and ADHD is a feasible research strategy that may contribute to better understanding of nosology of these two disorders.

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