



Reduced Visual Evoked Potential Amplitude in Autistic Children with Co-Occurring Features of Attention-Deficit/Hyperactivity Disorder

Amanda Cremone-Caira^{1,2} · Yael Braverman^{2,3} · Gabrielle A. MacNaughton⁴ · Julia I. Nikolaeva⁵ · Susan Faja^{2,3}

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Abstract

Provided the significant overlap in features of autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD), there is a critical need to identify transdiagnostic markers that could meaningfully stratify subgroups. The objective of this study was to compare the visual evoked potential (VEP) between 30 autistic children, 17 autistic children with co-occurring ADHD presentation (ASD+ADHD), and 21 neurotypical children (NTC). Electroencephalography was recorded while children passively viewed a pattern-reversal stimulus. Mean amplitude of the P1 event-related potential was extracted from a midline occipital channel and compared between groups. P1 mean amplitude was reduced in the ASD+ADHD group compared to the ASD and NTC groups, indicating a distinct pattern of brain activity in autistic children with co-occurring ADHD features.

Keywords Neurodevelopmental disorders · Autism spectrum disorder (ASD) · Attention-deficit/hyperactivity disorder (ADHD) · Comorbidity · Visual evoked potential (VEP) · Event-related potential (ERP)

Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder marked by social and communication challenges as well as restricted, repetitive interests or behaviors (Center for Disease Control, 2021). Recent estimates suggest that up to 71% of children with a diagnosis of ASD have co-occurring symptoms of attention-deficit/hyperactivity disorder (ADHD; Kaat & Lecavalier, 2013; Rommelse et al., 2010; Salazar et al., 2015) – a neurodevelopmental

disorder characterized by inattention and/or hyperactivity and impulsivity (Center for Disease Control, 2021). Since the release of the DSM-5, ASD and ADHD have been recognized as common co-occurring conditions (American Psychiatric Association, 2013). However, less is known about the extent to which children with co-occurring diagnoses of ASD+ADHD are qualitatively different from their peers diagnosed with a singular diagnosis of either ASD or ADHD (Kern et al., 2015; van der Meer et al., 2012).

Variability in symptom presentation within and across both ASD and ADHD creates significant obstacles in distinguishing between these disorders (McPartland, 2017). Moreover, the high rates of co-occurrence between these already diverse disorders make clinical assessment and intervention recommendations even more difficult. Thus, there is a critical need for the development of robust transdiagnostic tools that can be used in samples with distinct and overlapping symptomatology subserving both disorders to classify subgroups who may have differing treatment needs or responses. Consistent with goals of the Research Domain Criteria (RDoC; Insel et al., 2010), we argue the characterization of functional constructs, rather than diagnostic groups, may elucidate transdiagnostic markers of singular and co-occurring conditions. In the context of neurodevelopmental disorders, neural markers of such constructs may

Amanda Cremone-Caira and Yael Braverman contributed equally to this work.

✉ Susan Faja
susan.faja@childrens.harvard.edu

¹ Department of Psychology, Assumption University, Worcester, USA

² Division of Developmental Medicine, Boston Children's Hospital, Harvard Medical School, 2 Brookline Place, Brookline, MA 02445, USA

³ Department of Neurology, Boston Children's Hospital, Boston, USA

⁴ Department of Psychology, University of California, Los Angeles, USA

⁵ Department of Communication Sciences and Disorders, Northwestern University, Evanston, USA

be especially useful, as biomarkers are objective and, consequently, less prone to the degree of subjectivity that comes with observational- and interview-based assessments. A growing number of studies have reported potential biomarkers related to ASD (Loth et al., 2016, 2017; McPartland, 2017), with emphasis on the utility of distinct, electrophysiological biomarkers of ASD and ADHD (Jeste et al., 2015). However, a limited number of studies have explored neural markers that differentiate overlapping symptoms of ASD and ADHD (Cañigual et al., 2021; Shephard et al., 2018, 2019).

Using VEP to Identify Differences Between Children with Neurodevelopmental Disorders

The visual evoked potential (VEP) is an exogenous neural response observed in the visual cortex following low-level visual stimulation. The utility of VEP in mapping differences between children with ASD and ASD + ADHD is threefold. First, the VEP can be reliably sampled from children regardless of age, language, or cognitive abilities (LeBlanc et al., 2015; Odom et al., 2016; Varcin et al., 2016). Pattern-reversal VEP is a passive procedure: a strong, robust signal is detected via electroencephalography (EEG) as participants passively view a flickering grid presented on a computer screen. Because children are not required to make verbal or behavioral responses, the VEP can be derived from samples with reduced verbal and cognitive abilities across age groups. The use of EEG – a noninvasive recording of neural activity in the cortex – is also an advantage of this paradigm, as EEG is relatively inexpensive and well-tolerated by young children with and without neurodevelopmental disorders (Webb et al., 2015).

Second, the VEP correlates with high-order cognitive functions often impaired in children with neurodevelopmental disorders. For example, among a well-characterized sample of toddlers from Bangladesh, amplitude of the P1 event-related potential (ERP) – elicited by the VEP task – was positively correlated with concurrent and prospective cognitive composite scores (as measured by the Mullen Scales of Early Learning) of visual reception, receptive language, and fine motor skills (Jensen et al., 2019). P1 amplitude was also concurrently associated with enhanced spatial and sustained attention in infants (Xie & Richards, 2017). Provided this evidence, the VEP may serve as a marker of cognitive functioning in diverse populations of children.

Third, VEP has been used to differentiate subgroups of autistic and non-autistic children as well as subgroups of children with genetic variants of ASD. Autistic children have reduced P1 amplitudes relative to NTC (Siper et al.,

2016 although see Milne 2011). Differences in P1 amplitude were also reported in single-gene variants of ASD including fragile X (increased VEP amplitude in adolescents and adults; Knott et al., 2014) and Rett syndrome (decreased VEP amplitude in mouse models; LeBlanc et al., 2015). Likewise, Leblanc and Nelson (2016) reported that children with a deletion of the chromosomal region 16p11.2 (16p CNV) – a chromosomal variant associated with increased likelihood of ASD (Kumar et al., 2008; McCarthy et al., 2009) – had increased P1 amplitude, relative to NTC. Together, these studies indicate that the VEP may differentiate autistic from neurotypical children (NTC) as well as subgroups of children with unknown genetic vulnerability, suggesting neural responses vary between diagnostic groups and individuals with the same diagnosis (Kovarski et al., 2019).

Although the VEP has been found to detect differential brain signals among groups of children with various neurodevelopmental conditions, the literature exploring the VEP in developmental populations with ADHD, specifically, is limited. Adolescents with ADHD were reported to have higher P1 amplitude elicited by a VEP, compared to those with bipolar mood disorder (Nazhvani et al., 2013). Moreover, P1 amplitude distinguished adolescents with ADHD, bipolar mood disorder, and NTC with 92% classification accuracy, providing evidence that the VEP can differentiate ADHD among other clinical disorders. Whether or not VEP P1 amplitude differs between children with ASD and ASD + ADHD is unknown.

Current Study

Studies comparing the VEP response in ASD and co-occurring ASD and ADHD are limited, leaving a critical gap in our understanding of the underlying neurological differences between prevalent neurodevelopmental disorders with shared features. As such, the current study compared the VEP response to a pattern-reversal stimulus between children with a singular diagnosis of ASD, combined ASD and ADHD symptomatology (ASD + ADHD), and NTC. The VEP was measured via the P1 ERP signal over the midline occipital channel Oz (Jensen et al., 2019; Mizrahi & Dorfman, 1980; Sayorwan et al., 2018). Consistent with prior reports among children with ASD with unknown genetic vulnerability (Siper et al., 2016; Kovarski et al., 2019), it was hypothesized that P1 amplitude would be reduced among ASD and ASD + ADHD children, relative to NTC.

Methods

Participants

As part of a larger study, VEP data was collected from 30 children with ASD (27 males, $M_{age} = 9.26 \pm 1.24$ years), 17 children with ASD+ADHD (13 males, $M_{age} = 9.47 \pm 1.42$ years), and 21 NTC without clinical concerns (17 males, $M_{age} = 8.77 \pm 1.28$ years). Across groups, exclusionary criteria included a history or current diagnosis of neurological disorders of known etiology and a history of serious head injury or physical impairment that would limit participation in the experimental tasks. Children whose caregivers had insufficient English language abilities were also excluded to maintain validity and reliability of study measures.

Group status (ASD, ASD+ADHD, and NTC) was based on criteria outlined by Cremonese-Caira et al. (2021). Children in the ASD groups were recruited based on caregiver-report of existing clinical diagnoses. Diagnostic status was then confirmed via standardized questionnaires and/or an assessment by a licensed clinician. Existing ASD diagnoses (for children in the ASD and ASD+ADHD groups) were confirmed via direct observation (Autism Diagnostic Observation Schedule – Second Edition Module 3; ADOS-2; Lord et al., 2012) and caregiver-report of symptoms (Autism Diagnostic Interview – Revised; ADI-R; Rutter et al., 2003). The ADOS-2 and ADI-R are standard measures of ASD characteristics and together capture a child's early developmental history and current abilities in the social, communication, and play domains. Based on all available information, a psychologist confirmed DSM-5 criteria for ASD were met. Children with a confirmed diagnosis of ASD and T-scores ≥ 65 on the ADHD subscale of the Child Behavior Checklist (CBCL; Achenbach & Rescorla 2001) were classified as having clinically significant ADHD symptoms and included

in the ASD+ADHD group, consistent with other studies (Andersen et al., 2013; Cremonese-Caira et al., 2019, 2021).

Children with significant, confounding clinical traits were also excluded from analyses. NTC children whose CBCL ADHD subscale T-score was ≥ 65 (to rule out significant ADHD symptoms) and/or Social Responsiveness Scale – Second Edition (SRS-2; Bruni 2014) Total T-score was > 75 (to rule out significant ASD symptoms) were also excluded from analyses.

Materials

VEP Paradigm

The VEP paradigm was administered on a PC using E-Prime 2 stimulus presentation software. The stimulus was a white and black grid presented in the center of the screen with each square in the grid subtending a 0.86×0.86 degree visual angle. The stimulus pattern was reversed every 500 ms for 100 trials (Fig. 1).

EEG

During the VEP paradigm, EEG was continuously recorded via Net Amps 400 (Electrical Geodesics, Inc.). A 128-channel Hydrocel Geodesic Sensor Net (GSN) was soaked in a potassium chloride electrolyte solution and placed on the participant's head following manufacturer specifications. Prior to the start of each recording, impedances were below $50 \text{ k}\Omega$. During recordings data were referenced to the vertex and a 4 kHz antialiasing hardware filter was set to a 500 Hz sampling rate.

Procedure

All procedures were approved by the Institutional Review Board at Boston Children's Hospital (Boston, MA, USA). Caregiver consent and child assent were obtained before data collection. As part of a larger study investigating executive function among school-aged children with ASD (Faja et al., 2022), VEP data were collected at the end of a 1-hour long EEG recording session. During the EEG session, children were seated in a chair positioned approximately 70 centimeters away from a computer monitor displaying the stimuli. The VEP paradigm lasted approximately 1 min. Throughout the EEG session, participant behavior was continuously monitored by a trained behavioral assistant (seated next to the participant to ensure participant comfort and attention) and another research assistant who subjectively tracked participant attention, gross motor movements, and excessive eye movements or blinks via a webcam affixed to the top of the computer screen.

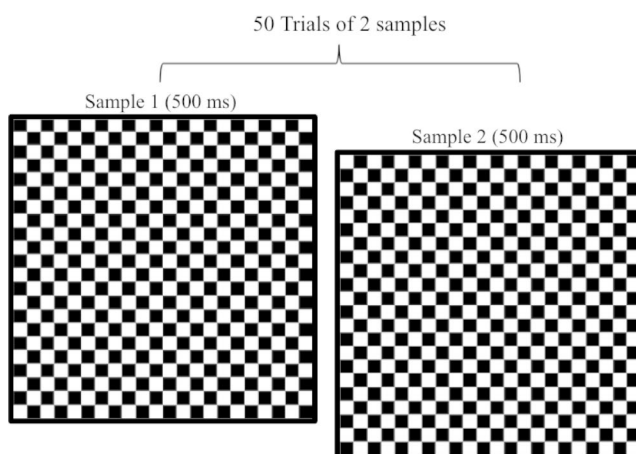


Fig. 1 Schematic of the VEP task
Note: *Ms* = *Milliseconds*

EEG Analysis

EEG data were re-filtered off-line using a highpass (0.1 Hz) and low-pass (30 Hz) filter (Kaiser-type FIR filter with 2 Hz rolloff). Data were then segmented using a 100 ms baseline immediately preceding stimulus onset and 400 ms period following stimulus onset. After segmentation, data were baseline corrected using the average of their respective baseline periods. Automated artifact detection excluded trials with the following parameters: (1) presence of an eye blink either tracked during acquisition or using the Netstation Eye Blink algorithm set at 220 μ V with an 80 ms moving average and (2) more than 10 channels with a fluctuation exceeding 140 μ V or less than 1 μ V with an 80 ms moving average or (3) detection of inattention during data collection. Data were then visually inspected and additional segments were excluded if they contained significant drift, eye movements or blinks, movement or mechanical artifacts.

Next, channels that were marked as containing an artifact for more than 20% of trials were replaced using spherical spline interpolation which estimates voltages by imputing across channels. Data were then averaged individually and re-referenced offline to the average of all electrodes (excluding the four eye channels) using the polar average reference effect (PARE) correction. Data were baseline corrected once more to adjust for rejected trials then individually inspected for quality. The number of trials included in ERP analyses did not significantly differ between groups ($F(2,65)=0.32$, $p=0.73$; Table 1).

Consistent with prior literature, VEP was operationalized as the mean amplitude of the P1 ERP component derived from the midline occipital electrode Oz 70 ms after stimulus onset for 60 ms (i.e., 70 to 130 ms window). Mean amplitude was chosen due to its demonstrated robustness to noise, relative to peak amplitude (Clayson et al., 2013; Luck, 2014). Electrode Oz was selected due to greater signal to noise ratio, relative to lateralized electrodes O1 and O2 (Hamilton et al., 2021; Varcin et al., 2016). Consistent with previous literature (Jensen et al., 2019; Varcin et al., 2016), the 70 ms to 130 ms window was confirmed based on visual inspection of the ERP waveforms generated in our samples (see Fig. 2).

P1 mean amplitude was compared between groups using univariate ANOVA. Specifically, diagnostic group (ASD, ASD+ADHD, and NTC) was entered as the independent variable and mean P1 amplitude was entered as the dependent variable. In models with significant main effects, pairwise comparisons were run with Bonferroni correction.

Results

Participant demographics are reported in Table 1. Overall, age ($F(2,65)=1.51$, $p=0.23$, $\eta_p^2=0.04$) and sex distribution ($\chi^2(2, n=68)=1.65$, $p=0.44$) did not differ between diagnostic groups. Although all participants were verbal and had cognitive ability in the average or above average range, full-Scale IQ significantly differed by group ($F(2, 65)=5.58$, $p=0.01$, $\eta_p^2=0.15$). Post-hoc analyses indicated that the NTC group had significantly higher IQ than the ASD ($p=0.01$) and ASD+ADHD groups ($p=0.03$).

The ANOVA comparing mean amplitude of the P1 ERP component at Oz revealed a significant main effect of group, $F(2,65)=4.32$, $p=0.02$, $\eta_p^2=0.12$. Pairwise comparisons indicated that, relative to the ASD and NTC groups, P1 mean amplitude was significantly reduced in the ASD+ADHD group ($ps=0.05$ and 0.02 , Fig. 2).¹ Moreover, group differences in P1 mean amplitude remained significant when IQ was added as a covariate (main effect of group: $F(2,64)=3.65$, $p=0.03$, $\eta_p^2=0.10$; main effect of IQ: $F(1,64)=11.58$, $p=0.001$, $\eta_p^2=0.15$). However, when IQ was included in the model, P1 mean amplitude in the ASD+ADHD was significantly reduced only when compared to the ASD group ($p=0.03$), and not the NTC group ($p=0.24$).

Discussion

The primary aim of the current study was to compare the P1 ERP response to the VEP across groups of children with ASD, ASD+ADHD and NTC. In contrast to our hypothesis, our results indicate that P1 mean amplitude was reduced in the ASD+ADHD group compared to both ASD and NTC groups. Regarding clinical characterization, these results suggest that there are quantifiable differences in neural responses to low-level visual stimulation between groups of children diagnosed with singular and co-occurring neurodevelopmental disorders. Specifically, our ERP data suggest that children with ASD and co-occurring ADHD symptomatology (ASD+ADHD) have a different neural response to a basic pattern-reversal VEP task relative to their singular ASD diagnosis and NTC peers, as P1 mean amplitude was reduced in the ASD+ADHD group compared to the groups of children with ASD and those without clinical concerns.

Notably, our findings are inconsistent with a recent study that did *not* find group differences in P1 amplitude between children with ASD, ADHD, and ASD+ADHD (Cañigueral et al., 2021). However, Cañigueral and colleagues (2021) obtained P1 from a different VEP task that measured

¹ P1 latency did not significantly differ between groups (main effect: $F(2,65)=1.11$, $p=0.34$).

Table 1 Participant demographics

	NTC M (SD)	ASD M (SD)	ASD+ADHD M (SD)	Significance
Number of Participants	21	30	17	–
Age (Years)	8.77 (1.28)	9.26 (1.24)	9.47 (1.42)	NS
Sex (Male: Female)	17:4	27:3	13:4	NS
Race ^a (White: Non-White)	16:4	25:5	14:2	NS
Ethnicity ^a (Non-Latinx: Latinx)	18:2	29:1	15:1	NS
Average Household Income ^b (%)				NS
< \$20,000	–	3.4%	7.1%	
\$21,000 - \$35,000	–	10.3%	7.1%	
\$36,000 - \$50,000	–	6.9%	7.1%	
\$51,000 - \$65,000	–	3.4%	–	
\$66,000 - \$80,000	10.5%	10.3%	14.3%	
\$81,000 - \$100,000	5.3%	20.7%	28.6%	
\$101,000 - \$130,000	21.1%	13.8%	7.1%	
\$131,000 - \$160,000	31.6%	6.9%	7.1%	
> \$160,000	31.6%	24.1%	21.4%	
CBCL ADHD (T-score)	50.95 (2.06)	57.40 (3.82)	71.24 (3.15)	$F = 193.90^{***}$
WASI-2 FSIQ	116.76 (12.84)	106.30 (11.96)	106.71 (9.88)	$F = 5.58^{**}$
WASI-2 VCI	118.86 (18.41)	108.23 (12.82)	104.41 (10.39)	$F = 4.51^{**}$
WASI-2 PRI	111.48 (9.56)	102.90 (13.11)	107.71 (14.47)	NS
SRS-2 Total ^c	56.90 (3.05)	67.03 (8.61)	73.88 (6.62)	$F = 29.33^{***}$
Vineland-II Communication	114.29 (11.79)	94.63 (11.09)	86.94 (8.55)	$F = 34.34^{***}$
Vineland-II Daily Living	105.81 (10.86)	88.90 (10.06)	85.65 (9.27)	$F = 23.70^{***}$
Vineland-II Socialization	109.00 (10.49)	83.70 (9.18)	83.70 (9.18)	$F = 67.10^{***}$
Vineland-II Adaptive Behavior Composite	109.38 (8.63)	87.10 (8.58)	81.18 (6.87)	$F = 67.10^{***}$
ADOS-2 Total Comparison Score ^c	–	8.50 (1.55)	8.56 (1.32)	NS
ADI-R Social	–	17.33 (5.01)	18.35 (4.10)	NS
ADI-R Communication	–	15.97 (3.76)	15.88 (4.06)	NS
ADI-R RRBs	–	7.67 (2.62)	8.88 (2.06)	NS
Number of Trials Included in ERP Analysis	88.24 (7.94)	88.90 (8.60)	86.71 (11.11)	NS

Notes: *M* = mean; *SD* = standard deviation; *N/A* = not applicable; *NS* = non-significant ($p > 0.05$); *NTC* = neurotypical controls; *ASD* = autism spectrum disorder; *ADHD* = attention-deficit/hyperactivity disorder; *CBCL* = Child Behavior Checklist; *WASI-2* = Wechsler Abbreviated Scale of Intelligence, Second Edition; *FSIQ* = Full Scale Intelligence Quotient (four subtests); *VCI* = Verbal Comprehension Index; *PRI* = Perceptual Reasoning Index; *SRS-2* = Social Responsiveness Scale, Second Edition; *ADOS-2* = Autism Diagnostic Observation Schedule, Second Edition; *ADI-R* = Autism Diagnostic Observation Schedule-Revised; *RRBs* = restricted and repetitive behaviors

^aDue to missing data, reduced *NTC* and *ASD + ADHD* samples ($ns = 20$ and 16 , respectively)

^bDue to missing data, reduced *NTC*, *ASD*, and *ASD + ADHD* samples ($ns = 19$, 29 , and 14 , respectively)

^cDue to missing data, reduced *ASD + ADHD* sample ($n = 16$)

* $p < 0.05$; ** $p < 0.01$; *** $p \leq 0.001$

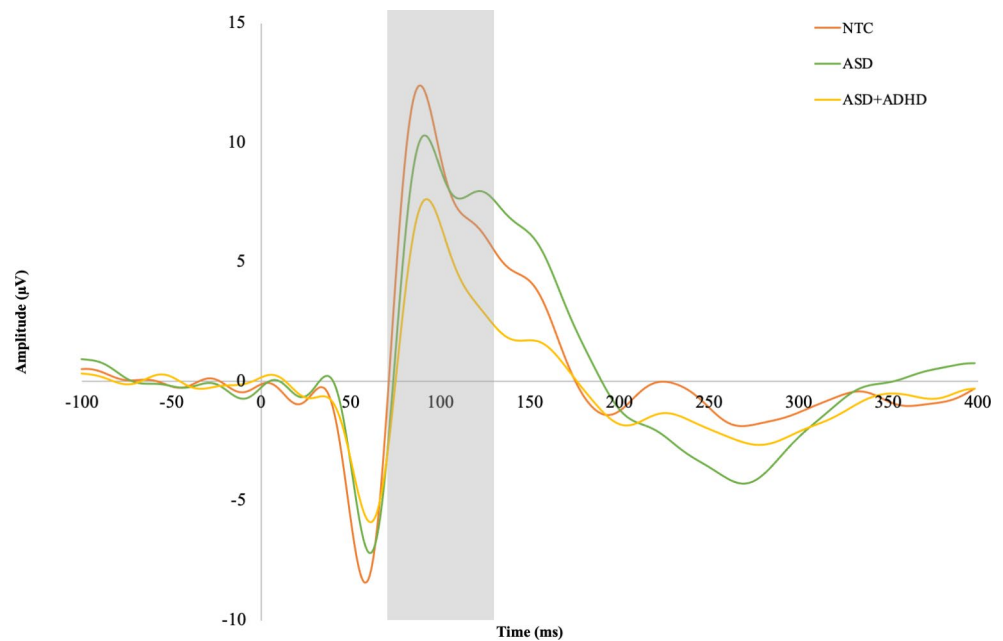
reaction time variability. In contrast, the VEP task used in the current study was intended to measure low-level visual sensory processing and required sustained visual attention for approximately 1 min. By design, our VEP task also did not require a cognitive or behavioral response. It is possible that these unique task demands may capture overlapping sensory and attention challenges characteristic of children with *ASD + ADHD* (Dellapiazza et al., 2021).

Similarly, we obtained inconsistent results with previous reports comparing the P1 between *ASD* and *NTC* groups

(Kovarski et al., 2019; Knoth et al., 2014; Siper et al., 2016). We hypothesize that the observed differences may be explained by (1) grouping parameters, as previous studies may have included children with co-occurring *ADHD* features in their *ASD* group and (2) use of mean rather than peak P1 amplitude (and/or peak-to-peak analysis methods; Siper et al., 2016). In our study, mean amplitude was selected due to its robustness to background noise (Clayson et al., 2013). By comparison, peak amplitude is impacted by differences in (1) noise level, (2) trial number, and (3)

Fig. 2 ERP Waveforms

Notes: ASD = autism spectrum disorder; ADHD = attention-deficit/hyperactivity disorder; NTC = neurotypical control; ms = milliseconds



trial-to-trial variability in latency within and across groups (see Luck 2014 Chap. 9 Supplement and Luck 2018).

In contrast to the ASD group in our sample, our ERP data suggest that children with co-occurring ASD and ADHD features have a distinct neural response to a basic pattern-reversal VEP task. We postulate that group differences in the VEP's neural response may reflect an atypical excitatory-inhibitory balance associated with impaired visual cortical responsiveness (LeBlanc et al., 2015). If this is the case, this low-level sensory task may be especially well-suited to detect neural differences within heterogeneous clinical groups. Further work is needed to test this theory directly.

Consistent with this theory, our findings lend neurophysiological support to recent research by Dellapiazza et al. (2021) who reported that autistic children with co-occurring ADHD have unique sensory processing profiles compared to those without clinically significant ADHD features. Specifically, sensory processing correlated with attention problems in ASD and ASD+ADHD groups, which could be consistent with our ASD+ADHD group exhibiting significantly diminished neural responses during a sensory task (i.e., overall smaller P1 response). Such group differences may be helpful in developing targeted interventions that improve higher-order cognition, as sensory processes are antecedents of, and contribute to, functions that are often impaired among groups of autistic children including attention (Dellapiazza et al., 2021), executive function (Pastor-Cerezuela et al., 2020), and social interaction and communication (Thye et al., 2018). Although the neural P1 results reflected this behavioral pattern amongst our clinical groups, we did not have a suitable behavioral measure of sensory processing to evaluate in this sample. As such,

additional research is needed to characterize behavioral correlates of brain-based VEP differences in these groups.

Taken together, this collection of evidence suggests that neural responses to the VEP may be useful in stratifying subgroups of ASD children with unique sensory processing abilities. Consequently, VEP biomarkers could elucidate new mechanistic insights into sensory processing in co-occurring neurodevelopmental disorders and, therefore, inform new clinical targets for intervention, consistent with the aims of NIMH's RDoC (Insel et al., 2010).

Limitations and Future Directions

To our knowledge, this study was the first to compare neural correlates of the VEP response across groups of children with ASD and co-occurring ADHD symptom presentation. As such, replication studies are needed. The current sample was notably small. Although there was no significant difference between the ASD and NTC groups' P1 mean amplitudes, the ASD group's P1 amplitude was less than that of the NTC group, consistent with other work (Kovarski et al., 2019; Knoth et al., 2014; Siper et al., 2016). Therefore, future work should be conducted with a larger sample size to clarify these findings.

Moreover, the range of IQs obtained in each group also do not necessarily represent the broader population, as IQs were higher than average in the NTC group (see Table 1) and the sample was restricted to $IQ \geq 80$. Statistically, the difference in P1 mean amplitude between the ASD+ADHD and NTC groups was no longer significant when IQ was accounted for, suggesting that our findings may be influenced by intellectual differences across groups. Notably,

however, the main effect of group remained significant when controlling for IQ, as did the differences in P1 amplitude between the ASD and ASD + ADHD groups. However, as our IQ range was restricted, the effects of IQ in this sample should be considered with caution.

Future studies could also be improved by revised methodology. First, ADHD symptomatology should be evaluated with gold-standard clinical assessments – such as psychosocial interviews, neuropsychological testing, and direct behavioral observation – that can be used to replace or supplement the subjective caregiver-report measures used in the current study (Conners-3; Conners 2008 and CBCL). Additionally, the timing parameters of the VEP task should be altered to lengthen, and increase time between, trials to improve frequency resolution, as longer trials would afford sufficient frequency resolution to compute power at different points in time (also known as the event-related spectral perturbation (ERSP)) which could reveal how the time course of changes in power relate to the ERP. Our ERP waveforms (Fig. 2) also show that the ERP may be further decomposed into distinct components (e.g., P1a and P1b) that are observed in other studies (Kovarski et al., 2019) and should be explored in future research. Finally, additional research using neuroimaging tools with greater spatial resolution than EEG/ERP is needed to elucidate the neural etiology of both ASD and ADHD. To date, converging evidence suggests that both disorders may be characterized by abnormal structural and functional connectivity in the cortex and/or diminished synchronization of neural networks (see Kern et al., 2015 for review).

Conclusions

The present study indicates that unique neural correlates of low-level sensory processing are evident in ASD children with co-occurring ADHD symptom presentation (ASD + ADHD) in comparison to children with a singular diagnosis of ASD. The VEP is a robust measure of great utility in transdiagnostic research with neurodevelopmental disorders. As such, future research is warranted to determine if neural correlates derived from VEP could be useful stratification biomarkers for subgroups of ASD children with and without co-occurring symptomatology. Future studies may consider how neural correlates can be used to identify subgroups of children with unique sensory profiles and, consequently, the utility of targeting sensory processing challenges to improve psychosocial outcomes.

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Data Availability Some of the data is already publicly available in the National Database for Autism Research.

Declarations

Competing interests None of the authors have potential conflicts of interest to be disclosed.

Ethics Approval All procedures performed in this study were in accordance with the ethical standards of the Institutional Review Board at Boston Children's Hospital.

Consent to Participate and Publish: Written informed consent was obtained from the legal caregivers of all participants in this study.

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