Journal of Autism and Developmental Disorders (2019) 49:5009–5022
https://doi.org/10.1007/s10803-019-04207-6

ORIGINAL PAPER

Differences in the Late Positive Potential and P300 to Emotional Faces in Individuals with Autism Spectrum Disorder

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Published online: 5 September 2019

Abstract

Despite evidence suggesting differences in early event-related potential (ERP) responses to social emotional stimuli, little is known about later stage ERP contributions to social emotional processing in individuals with autism spectrum disorder (ASD). Adults with and without ASD completed a facial emotion recognition task involving stimuli that varied by emotional intensity while electroencephalograms were recorded. Principal components analysis was used to examine P300 and late positive potential (LPP) modulation by emotional intensity. Results indicated that greater ASD symptomatology evinced heightened P300 to high relative to low intensity faces, then heightened LPP to low relative to high intensity faces. Findings suggest that adults with greater ASD symptomatology may demonstrate a lag in engagement in elaborative processing of low intensity faces.

Keywords Autism spectrum disorder · ERP · LPP · P300 · Emotion processing · Social cognition

Deficits in social communication are a hallmark of Autism Spectrum Disorder (ASD) (American Psychiatric Association 2013). Individuals with ASD demonstrate aberrant social-emotional processing, as evidenced by behavioral, neuroimaging, and psychophysiological research (Blau et al. 2007; Harms et al. 2010; Kohls et al. 2012). Indeed, research has demonstrated that individuals with ASD evince deficits in processing nonverbal socio-affective cues (Harms et al. 2010). To date, most of the electrophysiological studies using event-related potentials (ERPs) to examine social-emotional processing in individuals with ASD have focused on early-stage ERPs (Batty et al. 2011; Hileman et al. 2011; Lerner et al. 2013), which represent relatively automatic perceptual processes, and thus, do not probe differences in later affective and cognitive processes that may be most related to the elaborative processing of emotional stimuli. This study examines later-stage social-emotional processing in adults with ASD as compared to typically developing (TD) peers using principal components analysis (PCA).

The literature on social-emotional processing in individuals with ASD has often focused on facial emotion recognition as a paradigmatic social-emotional process (Harms et al. 2010; Lozier et al. 2014) with downstream implications for social functioning (Trevisan and Birmingham 2016). While there has been considerable debate concerning the magnitude and universality of emotion perception deficits associated with ASD (see Harms et al. 2010 and Jemel et al. 2006 for review), recent meta-analyses have substantiated behavioral deficits in facial emotion recognition (Lozier et al. 2014; Uljarevic and Hamilton 2013). Individuals with ASD perform below that of TD controls on tasks of facial emotion identification and recognition across all six basic emotions (i.e. happy, sad, anger, fear, disgust, and surprise;

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s10803-019-04207-6) contains supplementary material, which is available to authorized users.

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Ekman et al. 2013; Lozier et al. 2014) and demonstrate particular difficulties when making judgements related to subtle expressions of emotion (Humphreys et al. 2006; Rump et al. 2009). Moreover, developmental studies of facial emotion recognition have noted that these deficits increase with age, observing the greatest divergence between group performance trajectories in adulthood (Gepner et al. 2001; Greimel et al. 2014; Rump et al. 2009). Overall, this literature suggests that individuals with ASD do not develop the specialization or expertise necessary to perform at the level of TD controls on more demanding tasks of emotion recognition and identification (e.g. identification of subtle facial expressions or paradigms with brief stimulus presentation; Webb et al. 2017).

When distributing visual attention to emotionally expressive, static faces, adults with ASD have been observed to allocate less attention to the eye region (Corden et al. 2008; Hernandez et al. 2009; Spezio et al. 2007) and more attention to the mouth region (Neumann et al. 2006; Spezio et al. 2007) compared to TD controls. A reduction in fixation to the eye region has been particularly correlated with lower performance on emotion identification tasks (Boraston et al. 2008; Corden et al. 2008). This, coupled with the finding that individuals with ASD demonstrate reduced fixation to the internal features of the face and increased fixation to the non-featural regions of the face (Pelphrey et al. 2002), suggests that individuals with ASD may not be attending to the most important aspects of a face for recognition and discrimination. However, this finding of differential single face scanning of individuals with ASD has not always been replicated (Kirchner et al. 2011; Rutherford and Towns 2008).

In addition to difficulties in identifying affect from facial expressions, individuals with ASD may also respond differentially to the viewing of emotional facial expressions. For TD individuals, the viewing of emotive facial stimuli (both in vivo and via video) leads to an increase in physiological arousal (Blair and Cipolotti 2000; Riby et al. 2012). However, not unlike the literature on emotion recognition and identification, findings for individuals with ASD have been inconsistent. Studies of physiological arousal in response to emotional facial stimuli have reported findings consistent with both accounts of hyperarousal (Bal et al. 2010; Cohen et al. 2015; Hirstein et al. 2001) and hypoarousal (Hubert et al. 2009; Riby et al. 2012). These contradictory findings may be due in part to individual differences in baseline activity levels of the autonomic nervous system. There is evidence to suggest that some individuals (measured in children) with ASD exhibit low baseline levels of parasympathetic activity (Ming et al. 2005). Therefore, successful facial emotion recognition may not only require the ability to perceptually encode and identify varying facial expressions but also an ability to modulate one's own physiological response to the viewing of emotion-laden expressions.

Interest in characterizing the cognitive processes underlying these observed behavioral and physiological responses has led to the examination of neural correlates pertaining to facial emotion recognition in individuals with ASD. Electrophysiological studies of emotional face processing typically examine the N170, an early event-related potential (ERP) peaking approximately 170 ms after stimulus onset, as a measure of facial processing that is modulated by emotional expressions (Blau et al. 2007). Many studies have concluded that individuals with ASD demonstrate differences in early stage perceptual processing of faces (i.e. decreased amplitude and longer latency of the N170) as compared to their TD peers (Batty et al. 2011; Hileman et al. 2011; Kang et al. 2017; O'Connor et al. 2005; Tye et al. 2014) and that this difference increases with age (Kang et al. 2017). However, similar to that of the behavioral data, this finding has not always been replicated (Faja et al. 2016; Webb et al. 2012). Notably, Webb et al. (2012) propose that the inclusion of a fixation cross on their stimuli, centrally located above the bridge of the nose, may have promoted an initial fixation to the eye region and subsequent attention allocation patterns akin to that of neurotypicals for their participants with ASD, thus minimizing any differences in the N170 response between their groups. However, individuals deliver preferential attention to different facial features for different emotions. While preferential attention is given to the eye region when viewing sad faces, preferential attention is given to the mouth region when viewing happy faces (Eisenbarth and Alpers 2011). Thus, adding an initial fixation to the eye-region may artificially change the natural face scanning pattern in participants. Additionally, differences in the N170 in adults with ASD as compared to TD adults have emerged on active facial emotion discrimination tasks (O'Connor et al. 2005) but not necessarily passive emotional face viewing tasks (Faja et al. 2016; Webb et al. 2012). This suggests that there is something specific to the processes engaged during active facial emotion discrimination tasks that differs between adults with and without ASD.

Facial stimuli are known to potentiate later ERPs that index sustained engagement and elaborative processing such as the late positive potential (LPP; Ferri et al. 2012; Wheatley et al. 2011), yet this component is understudied in ASD. The LPP peaks between 300 and 1000 ms after stimulus onset and is larger in response to salient emotional stimuli as compared to neutral stimuli in typically developing (TD) individuals (Hajcak et al. 2009). The LPP reflects motivated attention to emotional content, tracks salience of stimuli with larger LPP responses to more emotionally arousing stimuli, and is thought to represent the allocation of neural resources to salient emotional stimuli. So, while the N170 is linked to the structural encoding of faces, later-stage ERPs

are thought to represent more evaluative processes linked to stimulus content. Thus, the LPP is an especially useful tool for evaluating electrocortical response to active rating tasks.

In light of previous literatures suggesting blunted neural response during emotion labelling tasks (O'Connor et al. 2005) and decreased modulation of neural response to facial stimuli of varying intensity (Ashwin et al. 2007; Deeley et al. 2007), individuals with ASD may be less likely to demonstrate differential electrophysiological response to faces of varying emotion and emotional intensity. However, few studies have probed emotional face processing differences as a function of stimulus intensity (i.e. subtlety level of the emotional expression) in individuals with ASD (Lerner et al. 2013). Additionally, the few studies that have examined the LPP in individuals with ASD used top-down approaches to score the LPP (Benning et al. 2016; Luckhardt et al. 2017). While this approach is common in the literature, it is potentially problematic because the time course of the LPP overlaps with the P300 ERP component (Foti et al. 2009). While the P300 is sensitive to attention and modulated by emotional stimuli like the LPP, it peaks earlier than the LPP (about 300 ms) and has been differentiated from the LPP in previous studies (Keil et al. 2002; Mini et al. 1996). Nonetheless, while a recent meta-analysis indicates that the P300 is blunted in ASD (Cui et al. 2017), few studies have investigated the P300 in response to emotional faces in ASD. Thus, it is particularly important to tease apart the P300 and LPP to better understand the temporal dynamics of emotional processing in ASD. In contrast to top-down approaches to scoring ERPs, temporal-spatial principal components analysis (PCA) can be used as a data-driven approach to score ERP components by identifying latent components across electrode sites and time points, providing a systematic way to better tease apart sources of variability in the data.

In the present study, we addressed these gaps in the literature by examining ERP responses to an emotional face rating task in which faces vary by emotion and intensity via a data-driven analysis approach in adults with and without ASD. In doing so, we aimed to better tease apart the effects of P300 and the LPP PCA component scores in predicting ASD symptomatology. We anticipated that smaller magnitude of the (1) P300 and (2) LPP PCA component scores would be dimensionally associated with greater ASD symptomatology. Similarly, based on previous literature, we expected that individuals with elevated ASD symptoms would have significantly smaller (3) P300 (Cui et al. 2017) and (4) LPP PCA component score (Benning et al. 2016) to all emotional faces as compared to TD participants. Given that the P300 indexes attention to salient stimuli (Keil et al. 2002; Mini et al. 1996), we anticipated that (5) individuals with less ASD symptomatology dimensionally, or those in the TD group categorically, would demonstrate a larger P300 amplitude to high relative to low intensity emotional expressions. In line with past research suggesting blunted neural response to emotional faces and attenuated differentiation by emotional intensity in ASD (Ashwin et al. 2007; Benning et al. 2016; Deeley et al. 2007), we anticipated that participants with greater ASD symptomatology, or those in the elevated ASD symptoms group, would show attenuated P300 modulation by intensity. Drawing from the same literature, we would expect (6a) the same pattern for the LPP with participants with greater ASD symptomatology or participants in the elevated ASD symptoms group demonstrating attenuated LPP differentiation between high and low intensity emotional expressions. However, given the behavioral deficits observed in individuals with ASD while making judgements related to subtle facial expressions, we would predict (6b) the *opposite* pattern with *larger* amplitude to low relative to high intensity faces in the LPP as an index of greater sustained attention and effort due to higher task demands.

Methods

Participants

Participants included 22 adults with elevated ASD symptoms and 43 typically developing (TD) adults who consented under a University IRB. The group of individuals with elevated ASD symptoms was composed of individuals that met the clinical symptom cut-off (32 or above) on the Autism Spectrum Quotient (AQ; Baron-Cohen et al. 2001) or self-reported having ASD (including via previous diagnosis) and met the high-sensitivity cut-off on the AQ (26 or above; Woodbury-Smith et al. 2005). Five participants scored between 25 and 32 on the AQ and self-reported having ASD. These elevations in ASD symptoms are consistent with those found in adults who self-identify and/or meet diagnostic criteria for ASD in the broader (non-clinic referred) community (Bishop and Seltzer 2012). Our goal in selecting these inclusion criteria was to maximize ecological validity relative to adult populations that would present themselves as having ASD. While all participants in the elevated ASD symptoms group were assessed using the Autism Diagnostic Observation Schedule-Second Edition (ADOS-2; Lord et al. 2012) by a research-reliable administrator, meeting diagnostic criteria on the ADOS-2 was not required for inclusion in the study. Six participants in the elevated ASD symptoms group did not meet diagnostic cutoff on the ADOS-2 for ASD. The ASD phenotype is highly heterogeneous. Quantitative research has suggested that the social deficits associated with ASD are best represented as a continuous extension of typical social behaviour rather than categorically distinct (Constantino 2011; Constantino et al. 2004; Robinson et al. 2011). Therefore, we would expect

the ERP component factors to have a continuous rather than bimodal distribution in our sample. Thus, the present inclusion criteria were selected to maximize variability in ASD symptoms. The ADOS-2 Calibrated Severity Scores (CSS) were calculated for participants with elevated ASD symptoms. The ADOS-2-CSS is a measure of core symptom severity derived from the ADOS-2 algorithm.

One participant with ASD was excluded due to technical error during data collection. All TD adults were below cut-off on the AQ. One TD adult was excluded because they were not fluent in English, which impacted task completion. The final sample (Table 1) was composed of 21 individuals with ASD between 18 and 39 years of age (16 male; M_{age} = 26.312, SD_{age} = 7.361) and 42 TD individuals between 18 and 47 years of age (13 male; M_{age} = 21.123, SD_{age} = 4.706). Independent samples t-tests indicated that the elevated ASD symptoms group was older than the TD group [$t(61) = -3.398$, $p < .01$] and thus age was included as a covariate in our statistical models. Sex was also included as a covariate to account for the fact that the elevated ASD symptoms group was predominantly male whereas the TD group was predominantly female. All participants completed the Kaufman Brief Intelligence Test-Second Edition (KBIT-2; Kaufman and Kaufman 2004). For inclusion in the study, all participants had a FSIQ > 70 (no presence of intellectual disability). Groups did not differ by IQ [$t(61) = -1.462$, $p = .149$].

Table 1 Descriptive statistics for participants

	TD	ASD
N	42 (13 male)	21 (16 male)
Age		
Mean	21.12	26.31
SD	4.71	7.36
Range	18.14–47.71	18.35–39.98
FSIQ		
Mean	101.00	106.19
SD	12.36	15.02
Range	79–128	72–133
AQ		
Mean	18.12	35.81
SD	5.52	5.10
Range	7–30	27–45
ADOS-2 CSS		
Mean	–	5.81
SD	–	2.84
Range	–	1–10

FSIQ Full scale IQ, *AQ* Adult Autism Spectrum Quotient, *ADOS-2 CSS* Autism Diagnostic Observation Schedule-Second Edition Calibrated Severity Score, *ASD* Group with elevated ASD traits

EEG Task

Participants completed the facial expression subtests from the Diagnostic Analyses of Nonverbal Accuracy (DANVA-2), a standardized measure of facial emotion recognition, during simultaneous recording of EEG (Lerner et al. 2013; Nowicki 2004). Evidence suggests that individuals demonstrate an own-age bias in face perception, reflecting increased performance on tasks of facial recognition memory for own- compared to other-aged faces (Rhodes and Anastasi 2012). Thus, including both adult and child faces may increase the overall difficulty of the task inducing greater variability in behavioral accuracy and reduce the likelihood that any ceiling performance effects would be observed. Therefore, both the adult and child facial expression subtests of the DANVA 2 were used.

Stimuli were 48 naturalistic color photographs of males (24) and females (24), including the torso and head, that displayed either a high intensity (24) or low intensity (24) facial expression depicting one of four emotions (12 happy, 12 sad, 12, angry, and 12 fearful; Online Appendix A).The images included in the adult subtest (24) were of individuals all above the age of 18 while the images in the child subtest (24) were between the ages of 6 and 12 years. The intensity (high versus low) as well as the emotional valence of each stimulus face was determined by consensus ratings from a group of students (third grade to college aged). In order to be included in the final stimulus set, at least 80% of the coders (N = 185) had to agree on the emotion that was being conveyed (Nowicki 2004).

Digitized images of the photographs were superimposed on a black background and displayed on a computer screen at a visual angle of 22.5° (width) × 15.5° (height), with the face region occupying approximately 4.75° (width) × 6° (height) of visual angle. Each face was presented on the screen for a minimum of 1000 ms and a maximum of 3000 ms. Participants were instructed to identify the emotion presented on the screen (happy, sad, angry, or fearful) via a button box. This behavioral response cued trial advancement and a blank-screen inter-trial interval was presented for 1000 ms after each response. In the event that a participant did not provide a response within the 3000 ms time window, the picture of the face disappeared from the screen and the response options remained on the screen until the participant selected a response.

All trials were included in ERP analyses, regardless of emotion identification accuracy. Independent samples t-tests revealed that groups did not differ in the number of errors made on the DANVA-2 for the overall task, for high intensity faces, nor for low intensity faces (Online Appendix B; one TD participant was excluded from these analyses for data loss due to experimenter error). Participants in the TD group had significantly faster reaction time on the

overall task, to high intensity faces, and to low intensity faces. To probe these effects with a dimensional measure of ASD symptomatology, we conducted bivariate correlations between AQ score, DANVA-2 errors, and DANVA-2 reaction time. Higher AQ score was significantly associated with slower reaction time on the overall task ($r = .298$, $p < .05$) and to low intensity faces ($r = .293$, $p < .05$). AQ scores also marginally positively correlated with reaction time to high intensity faces ($r = .246$, $p = .05$). AQ score did not significantly correlate with errors on the DANVA-2 (all $p > .2$).

EEG Acquisition and Reduction

EEG data was recorded using a 32-channel BrainVision actiCAP (Brian Products) arranged in the international standard 10/20 system with Ag/AgCl active electrodes and an actiCHamp amplifier. Electrooculogram (EOG) was collected from four facial electrodes. Facial electrodes were placed approximately one centimeter above the right eye, below the right eye, to the right of the right eye, and to the left of the left eye. A small amount of SuperVisc electrolyte gel was applied to each electrode to reduce signal impedance to ≤ 15 kΩ. Data were recorded continuously using BrainVision Recorder software at a 500 Hz sampling rate. BrainVision Analyzer 2.1 was used for off-line data reduction. Data were re-referenced to the average of TP9 and TP10 and filtered with a Butterworth filter with a low cutoff of .1 Hz and a high cutoff of 30 Hz. Ocular correction using the Gratton and Coles algorithm (Gratton et al. 1983) and semi-automatic artifact rejection were completed on epochs defined 200 ms before and 1000 ms after stimulus onset. For the first phase of artifact rejection, the program identified artifacts on the basis of the following criteria: voltage step of greater than 50 μV/ms, a difference of values in intervals of more than 175 μV with an interval length of 400 ms, and activity in intervals of 100 ms lower than .5 μV. During the second phase of artifact rejection, program-identified artifacts were approved or rejected by a trained research assistant and each epoch was visually inspected for additional artifacts. After artifact rejection, the TD and elevated ASD symptoms groups did not differ significantly in the number of trials retained overall, in the high intensity emotional face condition, nor in the low intensity emotional face condition (all $p > .47$). The P300 signal stabilizes at 20 trials (Cohen and Polich 1997) and the LPP signal stabilizes at 10 trials (Moran et al. 2013), thus the threshold for inclusion in the final analyses was at least 20 usable trials in each of the high and low emotional intensity conditions. For the high intensity condition, a mean of 23.97 trials (SD = .252) were included in the analyses and, for the low intensity condition, a mean of 23.95 trials (SD = .378) were included. Data were baseline corrected against − 200 to 0 ms before stimulus onset. ERP's were averaged across stimulus conditions (emotion and intensity) and exported for PCA.

PCA

PCA identifies patterns of electrocortical activity by extracting linear combinations of data across time and electrode location. This analysis was performed using the Matlab ERP PCA Toolkit (version 2). Individual averages of each condition (high intensity and low intensity) were entered into the PCA matrix. First, a temporal PCA was performed using promax rotation. The PCA included all time points as variables and all participants, stimulus conditions, and recording sites as observations which generated linear combinations of temporal factors. Comparison of a scree plot of the data with a scree plot of random data indicated that 8 factors generated by the PCA accounted for a greater proportion of variance than the random dataset. Thus, 8 factors were retained for rotation using the covariance matrix and Kaiser normalization. These factors preserve spatial information and can be characterized by both the factor loadings (time course of the factor) and factor scores (value derived from the subject, condition, and recording site).

Next, a spatial PCA was performed on each temporal factor using Infomax rotation. At this stage, recording sites were used as variables and the subjects, conditions, and temporal factor scores were entered as observations. A scree plot of the data was again compared with a scree plot of random data and this comparison indicated that 3 spatial factors should be extracted for Infomax rotation. This yielded a total of 24 temporospatial factors. These factors were translated into voltages for interpretation.

Eight temporospatial factors each accounted for more than 1% of the variance and together accounted for 76.5% of the variance. Two temporospatial factors were similar temporally and spatially to P300 and LPP ERPs elicited in social-emotional processing tasks (Foti et al. 2009; Kujawa et al. 2013). Thus, these two factors were included in analyses (Fig. 1). The P300 factor was an early parietal positivity peaking at 250 ms which accounted for 5.86% of the overall variance. The LPP factor was a late occipital positivity peaking at 956 ms which accounted for 9.54% of the overall variance. For more information regarding the relationship between the PCA factors and top-down scored ERP components from the original data, please see Online Appendices C and D. Notably, no factor resembling the N170 that accounted for more than 1% of the variance in the data emerged in the PCA. The remaining 6 factors that accounted for more than 1% of the variance were not interpretable (i.e. likely related to noise in the waveform), were redundant with the identified components (i.e. appearing to be the dipole of the P300 and LPP components identified), or did not clearly represent a relevant ERP component.

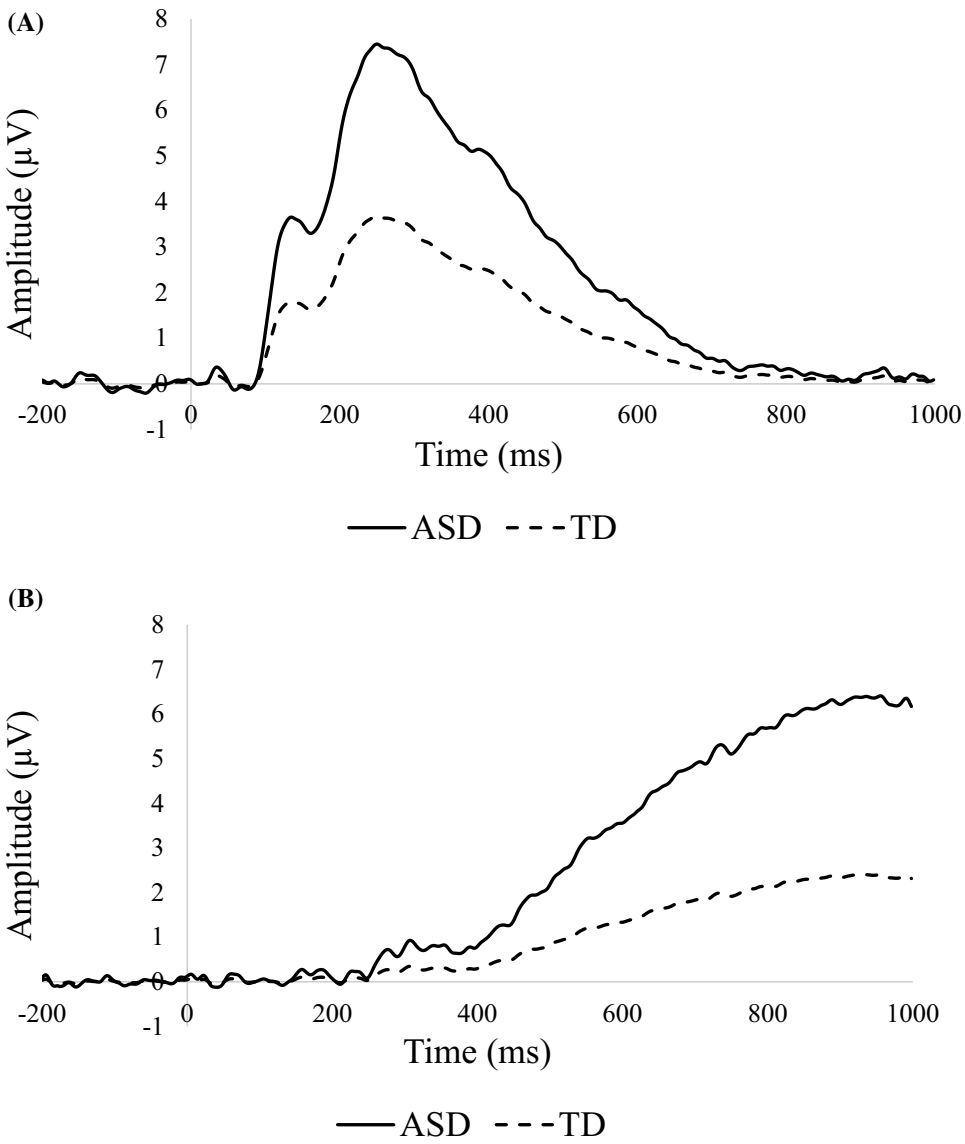

Fig. 1 *ASD* group with elevated autism spectrum disorder symptoms, *TD* typically developing. **a** Waveforms for the temporospatial factor representing the P300 by diagnostic group and **b** the temporospatial factor representing the LPP by diagnostic group

Data Analytic Plan

To evaluate our first hypothesis that smaller magnitude of the (1) P300 and (2) LPP would be associated with increased ASD symptoms, we conducted bivariate correlations between the magnitude of the ERPs and the AQ in the whole sample as well between the ERPs and the ADOS-CSS in the group with elevated ASD symptoms. We assessed our third and fourth hypotheses, that individuals with ASD would evince (3) blunted P300 amplitude and (4) blunted LPP amplitude to all emotional faces, by conducting two independent samples t-tests comparing P300 and LPP amplitudes to all emotional faces in participants with elevated ASD symptoms versus TD participants. To probe differences in modulation of the (5) P300 and (6) LPP by stimulus intensity we used generalized estimating equations (GEE) to evaluate interactions between the P300 or LPP and stimulus type in predicting dimensional ASD symptoms or categorical elevated ASD symptom status. PCA allows factors to be correlated and the P300 and LPP factors for each stimulus condition are moderately correlated. These components are likely correlated due to common-method variance and within-person variance. However non-independent observations violates the assumptions of the general linear model and thus we utilized GEE to evaluate the impact of the LPP and P300 in predicting ASD because this method assumes correlated independent factors (Hanley et al. 2003). Due to its estimation procedure, GEE is also ideal for samples of the present size, as it preserves more degrees of freedom than repeated measures ANOVA, and yields increased power as a function of the correlation of within-subject units of measurement.

For the GEE models, we compared unstructured, independent, and exchangeable working correlation matrices in

terms of their model fit. In all cases, the independent correlation matrix had the lowest QIC value (best model fit). In the first model, we used a linear link function to model AQ scores as the dependent variables. In the second model, we used a binary logistic link function to predict elevated ASD symptom status (0 = TD, 1 = ASD). We included intensity as nested within-subjects (i.e., participants) variables (coded 0 = low and 1 = high). We included LPP, P300, age, and sex as dimensional between subjects' factors. We included LPP*AQ score and P300*AQ score as interaction terms in the first model and LPP*elevated ASD symptom status and P300* elevated ASD symptom status as interaction terms in the second model. We adjusted for multiple comparisons using the Sidak correction.

Results

Dimensional Association Between Magnitude of P300 and LPP and ASD Symptoms

In contrast to hypotheses one and two, higher AQ scores were associated with greater magnitude of P300 ($r = .281$, $p < .05$) and LPP ($r = .312$, $p < .05$) responses to emotional faces (Fig. 2).

Due to the size of the ASD sample we were underpowered to report correlations with ADOS-2 scores in this subset of participants. That said, the correlations between the ADOS-2 CSS and the P300 ($r = .307$, $p = .176$) and LPP ($r = .221$, $p = .335$) components were in the same direction and of similar magnitude as the correlations between the ERP components and AQ scores (for P300 $r = .281$, $p < .05$ and for LPP $r = .312$, $p < .05$). Results of an *r*-to-*z* transformation to test the difference between two dependent correlations with one variable in common indicated that these *r* coefficients did not differ significantly (both $z < .55$ and both $p > .28$; Steiger 1980)—that is, the correlations of P300 and LPP with AQ scores did not differ from the correlations between these ERP components and ADOS-2 CSS scores.

Categorical Differences in P300 and LPP by Elevated ASD Symptom Status

In contrast with our third and fourth hypotheses that individuals with elevated ASD symptoms would evince blunted P300 and LPP responses to emotional faces, results indicated that the elevated ASD symptoms group exhibited significantly *larger* P300 response [$t(61) = -2.857$, $p < .01$] to emotional faces than the TD group (Fig. 1). Although the elevated ASD symptoms group did not significantly differ from the TD group in LPP response, there was a marginally significant effect in the same direction as the P300 results, with the elevated ASD symptoms group exhibiting a larger LPP response [$t(61) = -1.859$, $p = .068$; Fig. 1].

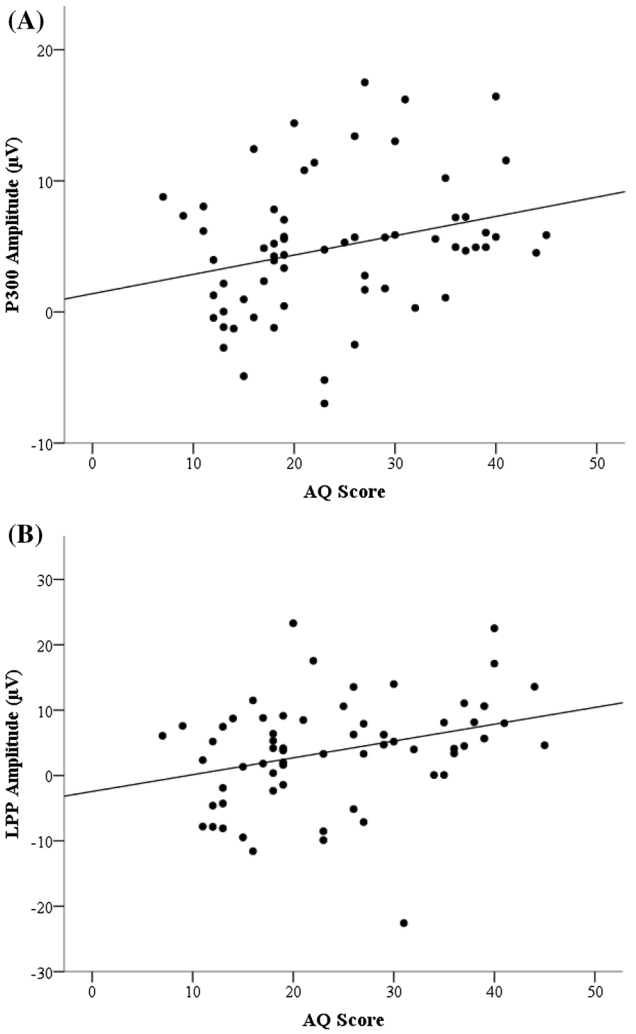

Fig. 2 Scatter plots depicting the correlation between **a** AQ score and P300 amplitude (r = .281, p < .05) and **b** AQ score and LPP amplitude (r = .312, p < .05)

Interactions Between P300, LPP, and Stimulus Type

Examination of the GEE model with P300 and LPP predicting AQ score while covarying age and sex indicated that there was a main effect of the LPP (*Wald's* $\chi^2 = 12.180$, $p < .001$) but not the P300 (*Wald's* $\chi^2 = .531$, $p = .47$) on the AQ, such that a larger LPP was associated with more ASD symptoms as measured by the AQ ($B = .254$, $p < .05$). The model also yielded a significant interaction between the P300 and emotional face intensity (*Wald's* $\chi^2 = 7.192$, $p < .01$). While the simple slopes did not significantly differ from zero (high intensity: $B = .312$, $p = .16$; low intensity: $B = -.003$, $p = .98$), individuals with greater ASD symptoms demonstrated a larger P300 to high relative to low

intensity faces. This finding is in contrast with hypothesis five, which predicted less P300 differentiation by intensity in individuals with increased ASD symptomatology. Additionally, there was a significant interaction between the LPP and emotional face intensity (*Wald's* $\chi^2 = 5.908$, $p < .05$) in predicting AQ score. Probing the simple slopes indicated that when emotions were of low intensity, a larger LPP significantly predicted a higher AQ score (*Wald's* $\chi^2 = 20.679$, $B = .462$, $p < .001$) relative to when emotions were of high intensity (*Wald's* $\chi^2 = 5.638$, $B = .254$, $p < .05$). Thus, larger LPP response to low intensity relative to high intensity faces predicted increased ASD symptoms (consistent with hypothesis 6b as opposed to hypothesis 6a).

Results of the GEE model examining the effects of P300 and LPP predicting diagnostic status while covarying age and sex revealed no significant main effect of the LPP (*Wald's* $^2 = 2.697$, $p = .101$) in predicting ASD status. A significant main effect of the P300 emerged (*Wald's* $^2 = 4.047$, $p < .05$) where the elevated ASD symptoms group had significantly larger P300 amplitudes than the TD group. The model yielded a significant interaction between the P300 and stimulus intensity (Fig. 3; *Wald's* $^2 = 4.361$, $p < .05$). Examining the simple slopes indicated that for high intensity faces, the association between the P300 and diagnostic status was significant (*Wald's* $^2 = 7.766$, OR = 1.232, $p < .01$) such that a larger P300 to high intensity emotional faces predicted elevated ASD symptoms status. There was no significant association between the P300 and diagnostic status for low intensity faces (*Wald's* $^2 = 1.155$, $p = .28$). Again, this finding is in contrast with hypothesis five. The interaction between the LPP and stimulus intensity was not significant (*Wald's* $^2 = 2.029$, $p = .154$).

Post-Hoc Analyses

We also examined whether the same pattern of results is observed when the TD and elevated ASD symptoms groups are matched on sex using FUZZY matching. While the results of the GEE model with P300 and LPP predicting AQ score remained the same, results of the GEE model with P300 and LPP predicting ASD status differed from the original analyses. Specifically, after matching for sex, there was a significant main effect of LPP in predicting ASD status such that a larger LPP predicted ASD status. In addition, the interaction between P300 and stimulus intensity predicting ASD status was no longer significant after matching for sex. For further description of these analyses please see Online Appendix E.

We ran the GEE models including only participants who met criteria for ASD on the ADOS-2 in the ASD group to examine whether significant findings remained after using more stringent diagnostic cut-offs. These analyses include the 42 TD participants and 15 participants in the ASD group. Results of the GEE model with P300 and LPP predicting AQ score after covarying age and sex including the ADOS-2 confirmed ASD group were consistent with the original analyses. Results of the GEE model with P300 and LPP predicting diagnostic status after covarying age and sex including the ADOS-2 confirmed ASD were largely consistent with the original analyses with one exception. The interaction between the P300 and stimulus intensity which was significant in the original analyses was no longer significant. A detailed description of these analyses can be found in Online Appendix E.

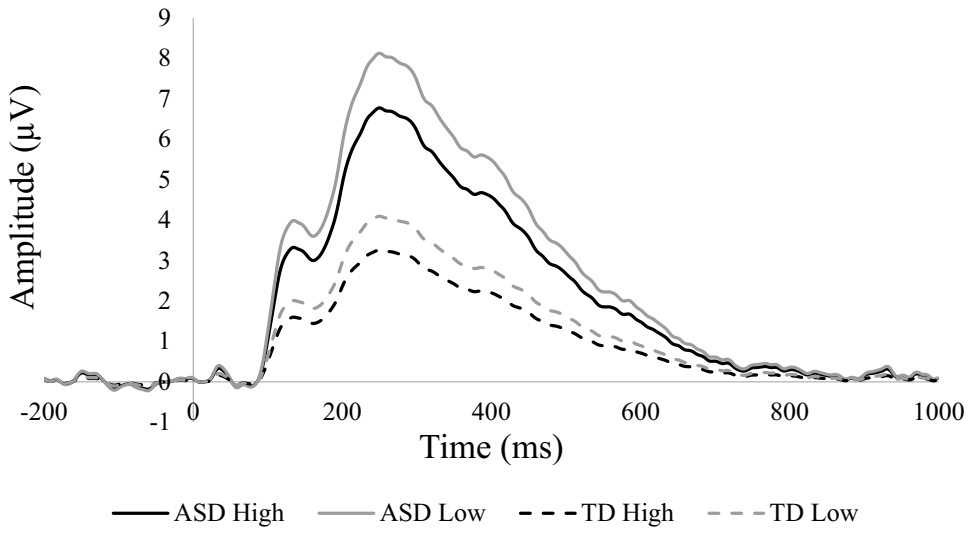

Fig. 3 *ASD* group with elevated ASD symptoms, *TD* typically-developing group. Waveforms for the temporospatial factors representing the P300 by stimulus intensity and diagnostic group

Discussion

This study is the first to use a data-driven approach to examine differences in social-emotional processing as a function of stimulus intensity in adults with elevated ASD symptoms and TD adults. Importantly, this approach enabled us to better isolate the unique variance associated with the P300 and LPP components which tend to overlap spatially and temporally, as a function of dimensional ASD symptomatology and elevated ASD symptoms categorically.

In contrast with our hypotheses, results revealed that larger P300 and LPP amplitudes were associated with greater ASD symptomatology as well as categorically defined elevated ASD symptoms. This deviates from past research (Benning et al. 2016; Cui et al. 2017) which demonstrated that children with ASD evinced blunted LPP and P300 amplitude as compared to their TD peers. Larger LPP responses have been observed in response to explicit facial emotion processing tasks reflecting increased visual processing of facial stimuli in comparison to implicit facial emotion processing tasks (Van Strien et al. 2010). In this case, larger LPPs in individuals with greater ASD symptomatology may indicate a greater engagement in visual processing of faces to meet task demands and, therefore, that it may be especially effortful for individuals with ASD to identify emotions. Additionally, our original hypotheses regarding overall P300 and LPP effects were based on studies including youth participants (Benning et al. 2016; Cui et al. 2017). Therefore, the pattern of results that emerged in the present study may be specific to *adults* with elevated ASD symptoms. For instance, it could be that adults with elevated ASD symptoms have developed compensatory mechanisms for facial emotion processing that are contributing to a heightened P300 and LPP response relative to TD adults. Additionally, the LPP component elicited by the DANVA-2 task in this study was maximal at occipital sites whereas it typically emerges at centro-parietal sites. This difference in scalp distribution may also contribute to the pattern of effects. Future studies should aim to assess replicability of these findings longitudinally in individuals with varying levels of ASD symptomatology to clarify the trajectory of motivated attentional deficits across individuals.

The P300 differed as a function of stimulus intensity when predicting ASD symptoms dimensionally and elevated ASD symptom status categorically. A larger P300 to high relative to low intensity faces predicted greater ASD symptoms. In line with evidence that the P300 is reflective of heightened attention to salient stimuli, it could be that high intensity emotional faces elicit a larger P300 relative to low intensity faces. In relation to ASD, this effect may be associated with deficits in emotion regulation. Some research has suggested that individuals with ASD demonstrate decreased neural habituation in the amygdala (Kleinhans et al. 2009) and physiological hyperarousal (Bal et al. 2010; Cohen et al. 2015; Hirstein et al. 2001) while viewing emotional facial stimuli. It is possible that when presented with salient stimuli, individuals with ASD have impairments in their ability to regulate their emotional response. Ultimately, this electrophysiological difference could represent an early index of emotion dysregulation. Another possibility is that past social experiences contribute to the differential response to negative social information observed in adults with ASD compared to their TD peers. In fact, individuals with ASD are more likely to experience social rejection in childhood (Little 2002; Van Roekel et al. 2010). The increased prevalence of peer victimization in individuals with ASD may contribute to a heightened sensitivity to social information later on in life and ultimately shape their neural response to negative social stimuli.

In contrast to the effect found in the P300, larger LPP responses to low relative to high intensity emotional faces predicted increased ASD symptomatology. These results suggest that it is the LPP response to low intensity faces specifically that drives the differences in overall LPP as a function of ASD symptoms dimensionally. Importantly, this effect is not due to differences in age or P300 response. A relatively greater LPP to low intensity faces is consistent with literature suggesting individuals with ASD demonstrate aberrant processing and identification of subtle emotional expressions (Humphreys et al. 2006; Lerner et al. 2013; Rump et al. 2009). Findings from the present study suggest that individuals with heightened ASD symptomatology engage in increased elaborative processing of low intensity facial stimuli to decode these emotional expressions. Since this effect is present when ASD symptoms are scored dimensionally, it suggests that it is the subtle behavioral, cognitive, and social symptoms of ASD, rather than purely differences between diagnosed and clinical groups, that drives the association seen here. Future research should explore whether the LPP response to emotional faces may be useful as a neural marker of ASD symptom severity.

The results of this study suggest that individuals with more symptoms of ASD demonstrate differential facial processing as a function of intensity. Specifically, individuals with greater ASD symptoms demonstrate increased initial P300 amplitude to high intensity facial expressions but later increased LPP amplitude to low intensity facial expressions. As discussed above, the initial heightened P300 amplitude to high intensity faces may be indicative of a sensitivity to highly emotional faces. This is consistent with the notion that the P300 is reflective of context-updating in the presence of highly salient stimuli (Donchin and Coles 1988).

Following this heightened response to high intensity faces at approximately 300 ms, increased ASD symptoms is associated with an increased amplitude to low intensity faces at approximately 1000 ms. This later onset response to low intensity faces could be interpreted as a lag in responding to the low intensity facial expressions. In other words, it may take individuals with greater ASD symptomatology longer to engage in elaborative processing of low intensity emotional expressions than it does individuals with decreased ASD symptomatology.

Two key strengths of the present study are the data-driven approach to quantifying the P300 and the LPP and the robust statistical models employed to analyse the data. We used principal components analysis (PCA) to identify unique sources of variance in the EEG data and score the LPP and P300 in a way that maximized the variance accounted for by each—the first time this approach has been used in the study of these components in ASD populations. As a result, the scoring of the P300 and LPP components in this study was not biased by experimenter input, and was thereby maximally representative of distributions in the present sample. This method enabled us to more systematically isolate the P300 and LPP which frequently overlap spatially and temporally. Likewise, we employed a data analytic approach that is particularly robust to the presence of correlated parameters, thus furthering the reliability and generalizability of obtained estimates.

Limitations and Future Directions

An N170 did not emerge from the PCA, which was somewhat surprising given that the EEG task included facial stimuli. However, it could be that since a non-face comparison condition was not included in this experiment, there was not enough variability in the N170 component to differentiate trials such that a relevant component emerged in the PCA. In addition to including non-face stimuli, future studies should consider including neutral facial expression stimuli which would emphasize differences in processing emotional versus neutral social stimuli as well as non-facial stimuli. Furthermore, examining differences in facial versus non-facial social stimuli would highlight potential differences in social processing not specific to face processing.

Additionally, the ASD sample was primarily male. Given that ASD is 4.5 times more likely to occur in males (Centers for Disease Control and Prevention 2011), this study likewise yielded a relative oversampling of males; future research should consider whether there may be relevant gender differences in these obtained effects.

Not everyone in the elevated ASD symptoms group surpassed the clinical cutoff on the ADOS-2. Given the heterogeneity of the ASD phenotype, it was important to include individuals who self-reported past diagnoses or symptoms in the clinical range despite not meeting criteria on the ADOS-2. However, we may have under-sampled individuals with high levels of symptomatology. In our primary and post hoc analyses, we made every effort to control for subject characteristics that may have contributed to the pattern of results (i.e. age, sex, and subthreshold ASD symptoms). Although the overall pattern of results in the post hoc analyses matching for sex and in post hoc analyses using more stringent diagnostic thresholds were consistent with the primary results, there were some differences. Therefore, we cannot rule out the possibility that sex differences and differences related to diagnostic thresholds may have contributed to the categorical (though not continuous) results regarding the intensity × P300 interaction, and bear careful attention in future publications examining these effects. Future studies may seek to replicate the findings of the present study in samples with higher rates of ASD symptomatology.

There is a high rate of co-occurring psychiatric disorders in ASD, with anxiety disorders and ADHD representing the most common co-occurring conditions (Simonoff et al. 2008). While examining the impact of co-occurring disorders on the P300 and LPP was beyond the scope of the current project, future studies should consider the way in which emotion regulation, anxiety, inattention, and impulsivity affect the modulation of electrophysiological responses in individuals with and without ASD.

This study examined differences in the P300 and LPP in adults with and without ASD while they naturalistically viewed emotional faces. One limitation of the study is that it is difficult to ascertain the percentage of task time during which participants were looking directly at the facial stimuli. However, this is a common limitation of any visual task without an eye-tracking component. Likewise, there were enough trials in both groups to resolve stable ERP signals. While the addition of a crosshair may have promoted attention to the face, this would have artificially influenced the participants' gaze scanning pattern. Additionally, while the P300 is related to attention, it is also associated with salience. The addition of a crosshair to the facial stimuli could have affected the salience of the images and impacted the P300 signal.

Another potential limitation of the present study is that the PCA factors are not identical to extant ERPs derived from top-down scoring approaches. PCA components represent linear combinations of the data generated to maximize unique variance across components whereas ERPs scored from a top-down approach are derived based on previous literature and standards in the field. While PCA factors are not identical to ERPs scored using traditional methods, they do approximate them in terms of morphology and timing, and correlate with their corresponding raw components. Future

work should attempt to replicate and continue to examine PCA derived components in this population.

Past studies examining the LPP in response to emotional stimuli, such as IAPS images, find that the LPP is larger in response to emotionally arousing images (Hajcak et al. 2009). While all emotional images are more arousing than neutral images, negative images tend to be more arousing than positive images. We anticipated that participants would demonstrate a larger LPP to high intensity emotional expressions as these are more salient and arousing than low intensity facial expressions. However, individuals with ASD demonstrated a larger LPP to low intensity faces. One limitation of the stimuli used in the present study is that the emotional faces in the DANVA-2 task are overall less arousing than the IAPS images. Additionally, the distinction between high and low intensity facial emotions in this task is more subtle than the distinction between positive, negative, and neutral IAPS images. Therefore, distinguishing based on arousal may be more nuanced in the DANVA-2, and ultimately, task demands may account for differentiation in LPP response. Unfortunately, there were not enough trials to examine ERP modulation as a function of emotion type in the present study (only 12 stimuli of each emotion presented in the DANVA-2). Future studies should probe LPP differences by emotion via paradigms that include more facial emotion stimuli and a neutral face condition to serve as a control.

Finally, future studies should examine LPP response to social stimuli across age cohorts as well as longitudinally to identify when differences in motivated attention to social stimuli emerge and how these differences evolve with development.

Conclusions

Findings from the present study suggest that the nature of deficits in social-emotional processing in individuals with ASD may vary across stages of processing. Using a data-driven approach that allowed for the teasing apart of overlapping components, adults with greater ASD symptomatology demonstrated larger P300s to high intensity faces and larger LPPs to low intensity emotional faces as compared to individuals with lesser ASD symptomatology. This pattern of aberrant facial processing suggests a potential lag in engagement in elaborative processing of low intensity faces in individuals with greater ASD symptomatology. These findings emerged even in the presence of a data-driven ERP component extraction approach that allowed for more precise and sample-specific isolation of the P300 and LPP, and were evident when characterizing ASD dimensionally and categorically. Past studies have shown that individuals with ASD have deficits in early-stage processing of low-intensity emotions (Lerner et al. 2013) while the current study demonstrates a pattern of later-stage deficits related to stimulus intensity. It is essential to continue to investigate differences in social-emotional processing as a function of ASD symptomatology across different temporal stages to further clarify the conditions under which social emotional processing is derailed in individuals with ASD and other social deficits.

Acknowledgments This work was supported by the Alan Alda Fund for Communication.

Author Contributions All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by all authors. All authors read and approved the final manuscript.

Funding This study was funded by the Alan Alda Fund for Communication.

Compliance with Ethical Standards

Conflict of interest Cara Keifer, Kathryn Hauschild, Brady Nelson, Greg Hajcak, and Matthew Lerner declare that they have no conflicts of interest.

Ethical Approval All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed Consent Informed consent was obtained from all individual participants included in this study.

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