



N170 decoding response to Duchenne smile face in autism spectrum disorder children

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Abstract

Autism Spectrum Disorder (ASD) is characterized by significant social impairments thought to partially stem from anomalous neural reaction to facial cues. This study pioneers the exploration of how ASD children respond to a distinct Duchenne smile facial expression, employing the N170 event-related potential (ERP), to assess their neural responses. Our research aims to shed light on how ASD children process facial expressions, providing insights into the underlying neural mechanisms associated with emotions in this population. We recorded ERPs from 20 children, consisting of 10 with ASD and 10 control group counterparts. They viewed a series of Duchenne smile faces of a familiar female celebrity while their neural responses were recorded via EEG in a controlled, distraction-free environment. Our findings revealed significant differences during latency between ASD and the control group. During facial processing, ASD children presented a shorter latency for both faces than the control group. Source localization identified activation in the right hemisphere regions in the ASD children, particularly in the superior temporal and superior pole areas as well as the insula, which are associated with emotional processing. The control group activated areas linked to visual and cognitive functions. These findings suggest that ASD children may recognize faces quickly where their emphasis is placed on non-emotional facial features, but possibly due to an impaired emotional processing mechanism linked to a reduced emotional responsiveness. This research challenges prevailing studies where the ERP pattern in ASD suggesting potential overlap with other neurodevelopmental or psychiatric conditions, such as psychosis. Further investigation into possible comorbidities is warranted, thereby deepening our comprehension of ASD and its multifaceted neurobiological connections.

Keywords N170 · Autism spectrum disorder · Duchenne face smile · ERP · Insula · Temporal pole

Introduction

Autism spectrum disorder (ASD) is a type of neurodevelopmental disorder characterized by the difficulties in the perception of emotions, struggling with facial expression processing, appropriate social behavior (Vlamings et al., 2010) and restricted and repetitive patterns of thought and behavior. The studies conducted so far in face emotional processing in ASD have been focused on general faces and negative facial expressions, while, in contrast, the study of the Duchenne smile face processing has not yet been researched. The specific Duchenne smile is unique in its physiognomy, which is accurately characterized. It differs from an ordinary social smile by the flexing of the muscles at the sides of the mouth, and by the contraction of the zygomatic major muscle and the orbicularis oculi muscle. Additionally, small wrinkles are also formed around the eyes due to the contraction of the cheeks and the orbicularis

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oculi muscle (Feuerriegel et al., 2015). The Duchenne smile plays a crucial role in social interactions as a genuine emotional engagement and the distinct posed smile characterized by their genuine expressions of positive emotion and social engagement (Ekman et al., 1990; Gunnery & Ruben, 2016) which are essential for nonverbal communication and social attachment (Krumhuber et al., 2014). Research suggests that ASD individuals have difficulties interpreting emotional signals (Boraston et al., 2008), making the genuine positive expression Duchenne smile an important stimulus for studying emotional recognition in ASD. While negative emotions like anger or sadness have been extensively studied in ASD, positive expressions like Duchenne smiles, which are a key for meaningful social interactions, remains underexplored (Key et al., 2015). Also, many of the studies conducted in ASD consistently presented deficits in face recognition (Evers et al., 2015; Xavier et al., 2015; Whitaker et al., 2017; Wingenbach et al., 2017), exhibiting evidence of atypical neural response (Hudson et al., 2023; Parker et al., 2021; Kang et al., 2019), which is a common trait shared with psychosis (Tripoli et al., 2022).

When we talk about emotions, facial processing is one of the most crucial aspects for social cognition and appropriate neurological response to facial expressions, which is crucial for successful interpersonal relationships and appropriate social communication (Batty et al., 2011). Contrary to ASD, in typical development children, the ability to recognize and interpret face emotions information begins in infancy (Samaey et al., 2020). This process continues to develop social cognitive skills throughout adolescence, playing an important role in the language of the non-verbal emotional recognition task (Batty & Taylor, 2006; Herba et al., 2006; Meaux et al., 2014). This suggests that language is linked to some earlier physiological indicators of facial emotional processing (Leppänen & Nelson, 2009).

Event-related brain potentials (ERP) are obtained using electroencephalography (EEG), which measures neural responses to specific emotional stimuli thru time sequences of human face perception (Miki et al., 2022). The N170 event-related potential is a negative waveform peaking a maximal amplitude over parietal-occipital sites approximately 170 msec after face stimulus presentation (Bentin et al., 1996). Also it is a marker that reflects the brain's early stages of processing visual stimuli, particularly, emotional face processing. This component is not just associated with the structural encoding of facial feature, but also demonstrates that N170 can be modulated by emotional content of faces, such as the Duchenne smile, and also is usually associated with faces-sensitive perception (Stephenson et al., 2020). It is identified with the earlier stage of face onset and reflects face structural encoding (Wynn et al., 2008). In the ASD literature, the N170 is one of the most studied

event-related potentials (ERP) because it is directly linked with the behavioral deficits in the face processing, and is considered one of the most hopeful biomarkers in ASD (Kang et al., 2017). Different ERP face processing studies conducted in ASD indicate that N170 latency to face stimuli is slower; however, not all studies observed these delays in this type of population (Dawson et al., 2005; Kang et al., 2018; McPartland et al., 2004), and some EEG studies even showed inconsistent results (Van der Donck et al., 2020). Recent studies have shown that the N170 ERP component is often delayed or reduced in amplitude in ASD children. This suggests that the neural processes involved in the recognition and processing of structural emotional faces are atypical with disruptions in the neural networks responsible for processing facial information in ASD children, which have been linked to impairments in the structural encoding of emotional faces, and leading to difficulties in accurately encoding and interpreting emotional expressions and potentially contributing to the difficulties these children face in social interactions (Jong et al., 2022; Yin et al., 2022; Matsuda et al., 2023; Chen et al., 2023).

According to our present understanding, only two EEG studies have employed stimuli containing the Duchenne smile face expression to examine variations in gaze patterns when discerning genuine and Duchenne smile face in individuals with Autism Spectrum Disorder (ASD). One of these studies focused on infants rather than children, and it suggested that exaggerated facial expressions may play a crucial role in supporting social information processing in high-risk ASD infant siblings (Key et al., 2015). The other study, conducted with adults, demonstrated difficulties in distinguishing between these types of facial expressions (Boraston et al., 2008).

The specific Duchenne smile is unique for the activation of the zygomatic major and orbicularis oculi muscles. This distinct posed smile is characterized by the genuine expressions of positive emotion and social engagement (Ekman et al., 1990). This expression is crucial in nonverbal communication and social interactions (Krumhuber et al., 2014). Research suggests that ASD individuals often struggle to interpret emotional signals (Boraston et al., 2008), making the genuine Duchenne smile a key stimulus for investigating emotion recognition deficits in this population due to its unique positive expression. While negative emotions like anger or sadness have been extensively studied in ASD, the Duchenne smile, which is also important for successful social interactions, remain underexplored (Key et al., 2015).

In this investigation, we present a pilot study where focus on the N170 component and we examine the connection between emotional decoding capacities and the recognition of normal and Duchenne smile facial expressions in ASD children while also exploring a potential existence in ASD

children of an anomalous overlap similar to those reported in psychosis.

Method

Participants

The sample used for the ERP consists of 20 subjects with the same socioeconomic status, education level and Caucasian race. The sample size is further discussed in the “[Limitations](#)” section.

The study involved an experimental group (ASD) of 10 children, comprising 6 boys and 4 girls, with ages ranging from 8 to 13 years old. A control group of 10 children, also consisting of 6 boys and 4 girls within the same age range of 8 to 13 years old, was included for comparison. The ASD children in our study had an average of intelligence quotient (IQ) of 89 ± 7.35 , whereas the control group exhibited an average IQ of 102 ± 5.33 . All children are from *Edith Stein School*, located in the city of Madrid, Spain. The diagnosis of Autism was conducted through a structured interview and the observation scale test for autism diagnosis (Ados-2) with an average of 18 ± 2.66 (Lord et al., 2012). None in both groups had any type of neurobiological or psychiatry disorders diagnosed. According to the primary caregivers, they were not aware of any visual, hearing or any other type of impairments.

Ethics procedure

The study complied with the ethical standards of the American Psychological Association and the procedures were approved by the Ethics and Clinical Research Committee of Madrid’s San Carlos Clinical Hospital, in compliance with the Declaration of Helsinki. We provided every school with written information about the study and obtained their written informed consent to participate. An informational talk and several group sessions were also arranged with principals and teachers and, after they approved the protocol, an informational talk and several group sessions were arranged with caregivers and teachers to explain the process and answer questions about the experiment, during which additional printed information was provided. After these sessions, and when parents expressed their interest in participating, we obtained the caregivers’ written informed consent to participate. We gave them written informed consent, parents and children were offered a detailed explanation of the procedures, risks and benefits of the experiment and given them several opportunities to ask questions to the researchers.

Each participant’s primary caregivers completed a screening measure that identified the eligibility of the children to participate in the study. Caregivers were contacted to schedule an appointment for their child to participate in the recording of data with the ERP and no child selected was excluded from the experiment.

Following the informational sessions, children were asked for their verbal assent. These children attend their school in the Autonomous Community of Madrid. No children had any neuropsychiatric or neuropsychiatric disorders.

Procedures

Participants were scheduled for a 30 minutes visit and were accompanied by their primary caregiver. Each child sat in an armchair 75 cm in front of a 19” computer screen that presented the visual stimuli (images). They were asked to be as relaxed as possible. The room had a very dim light and was isolated from external noise. The setting of the room removed all distractions and allowed a complete concentration of the children on the monitor while the computer was recording neural responses 2 m in front of them. The study focuses exclusively in the children’s response during Duchenne smile and neutral emotional face-viewing experiment when the EEG data was collected. On top of this, another filled up questionnaire about the children’s mental and physical health were filled up for their caregivers during their visits to the lab.

The children were presented 10 times the following series of images: 10 images of the same Duchenne-smiling face, 1 transitional image of a tree, 10 images of the same Neutral face, and 1 transitional image of a tree. The smiling and neutral faces were from a female celebrity familiar to all the children. Both faces were shown over a black background in a frontal pose and cut to an oval shape to minimize other body cues. The faces had the same size, color palette, contrast, and brightness. The faces were presented in the middle of the screen for 250 ms, with a black screen of the same duration between every two images. In total, the children visualized 220 images and 220 inter-stimulus black screens for approximately 2 min.

Recordings and data processing

The data was collected using 64-channel EEG system ATI-Pentatek during the face-viewing paradigm. The children’s scalp was cleaned using the appropriate gel and after the electrodes cap place in their heads according to the international 10–20 standard system. Data was processed based on a reference mean, once acquisition with a band pass filter of 0.05 to 30 Hz with a sampling speed of 1024 Hz was obtained. During session recording, all electrode

impedances were maintained to be at or below 10 k Ω . Electrodes were placed on both mastoids as on-line references. Three electrodes' data (cantus, supraciliary and inferior palpebral muscles) were collocated in order to help identify eye movements. The "noise" created by eye and muscle movements was eliminated off-line. Noisy channels, identified by excessive artifacts, were replaced by an adjacent clean channels to ensure data quality and the remaining artifact-free epochs were averaged for each participant. Noisy epochs with more than 4 standard deviations were replaced by moderate linear interpolations of adjacent clean channels (Dmochowski et al., 2012) to average nearby clean channels for accuracy. Trials that contain excessive artifacts were excluded from the analysis if the amplitude exceeded $\pm 100 \mu\text{V}$ in any channel. The remaining artifact-free trials were averaged for each participant. After noise reduction, epochs of 500 ms, encompassing a 100 ms pre-stimuli and 400 ms post-stimuli were selected for analysis. The N170 negative component generated was analyzed 150–250 ms after the trigger and opening time window of 40 ms interval (–20 to +20 msec), centered around the peak highest positive amplitude measured in Pz electrode). This approach allowed us to measure neural activity, as reflected in the amplitude differences illustrated in Fig. 1. In addition to latency, the amplitude of the N170 component was calculated as the peak negative voltage relative to the baseline during the same time window. This measurement provides the intensity of neural responses as a complement for the latency, which indicates the timing of the response.

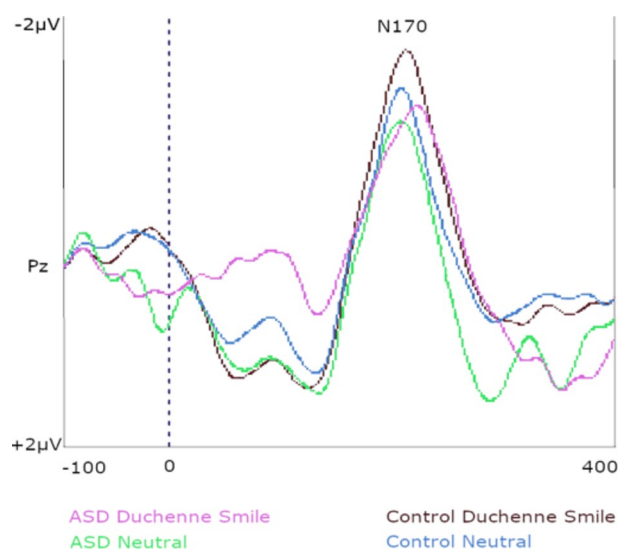


Fig. 1 Averaged event-related potential waveforms for the N170 Component at Pz Scalp Location. The x-axis represents time in milliseconds (ms), while the y-axis represents amplitude in microvolts (μV)

Measurements and statistical analyses

The comparative study, focused on a few independent variables associated with each face type (Duchenne smile vs. Neutral). Statistical analysis was performed using standard null-hypothesis significance testing with a fixed p-value of 0.05. The analysis employed standard comparative statistical techniques suitable for small-sized samples, as outlined below.

Before the statistical analysis, the data was evaluated to ensure it met the necessary criteria. The Shapiro-Wilk test was used to assess normality, and homogeneity of variances was tested using F-tests. For group comparisons, parametric tests like paired Student's t-test were employed when these assumptions were met. When these conditions were not fulfilled, a non-parametric method was applied to maintain the robustness of the analyses. When data values exceeding 2.5 standard deviations from the group mean, they were considered as anomalous and excluded from the analysis.

Within the N170 studies, the variables of interest were the N170 latency (ms) and N170 source localization in the neuroanatomical structures according to the projection areas of maximal intensity. Statistical measures for N170 latency included the calculation of grand averages and standard deviations of the population ($\text{avg} \pm \text{sdp}$), mean differences (MD), and the Shapiro-Wilk tests for normality to confirm that the data met the assumption of normal distribution. The statistical significance under normality was assessed using a one-tailed paired Student T-tests chosen due to its appropriateness for comparing means in paired samples when the direction of the effects is hypothesized a priori. This was paired with a previous F-test for equality of variance to verify that the assumption for equal variances between the groups was satisfied, ensuring the validity of the t-test.

The N170 negative component of ERP was estimated based on 64 recordings for all participants and localized in the brain through EEG inverse problem solution using Bayesian model averaging. This method can model uncertainty and can provide a robust estimation of the neural sources. Individual data was obtained using low-resolution electromagnetic tomography (Trujillo-Barreto et al., 2004) to calculate the electrical tomography of the brain. Model specifications involved restricting the solution to specific anatomical structures or combinations, facilitated by the Statistical Parametric Mapping (SPM) software package (MathWorks, Natick, United States) which is precise in handling complex neuroimaging data.

To identify statistically significant sources of P170, SPM was employed to establish maps based on a voxel-wise Hotelling T^2 distribution vs. zero (Carbonell et al., 2004). Probability maps were constrained to a false discovery rate of $q = 0.05$ (Lage-Castellanos et al., 2010) to mitigate false

positives, and presented as 3D activation images (Evans et al., 1993) providing reliable and accurate insights into the neural processes. Cortical projections were visualized according to the MNI coordinate system (Tzourio-Mazoyer et al., 2002).

Results

N170 wave morphology

The grand average of the cerebral ERP stimulus-synchronized waveforms associated with neutral and Duchenne smile faces were characterized by a negative N170 component of ERPs between 150 and 250 ms illustrated in Fig. 1. The amplitude suggests variability in the intensity of neural responses between both groups; the control group exhibited larger N170 amplitudes compared to the ASD group, while these children presented less distinction in amplitude between the different facial expression.

Table 1 Main neuroanatomical structures according to projection areas of maximal intensity in N170. AAL: Anatomical Atlas label corresponding to Probabilistic Brain Atlas (PBA). X, Y, Z: coordinates from PBA in three spatial axes. T²: Hotelling statistical test

N170	AAL	X	Y	Z	T ²	
Control DS	Cuneus L	-2	-70	23	28.897	
	Cuneus R	2	-76	22	26.726	
	Precuneus L	-1	-70	35	28.225	
	Precuneus R	4	-70	32	28.974	
	Calcarine L	-3	-70	15	28.501	
	Calcarine R	4	-70	12	27.953	
	Frontal Sup Medial R	2	47	4	24.059	
	Frontal Medial Orb R	2	53	-5	25.591	
	Cingulum Ant R	2	45	19	24.184	
	Cingulum Ant L	-2	45	18	24.016	
	Control N	Cuneus L	-2	-70	23	32.713
		Cuneus R	2	-76	22	29.386
		Precuneus L	-1	-70	35	31.259
		Precuneus R	4	-70	32	28.987
		Calcarine L	-3	-70	15	32.402
		Calcarine R	4	-70	12	29.132
Frontal Sup Medial L		-2	44	24	30.159	
Frontal Medial Orb L		-2	50	-8	31.701	
Cingulum Ant R		2	45	18	28.533	
Cingulum Ant L		-2	47	4	29.967	
ASD DS		Insula R	50	1	-1	7.254
		Temporal Sup R	50	0	-6	7.213
		Temporal Pole Sup R	50	2	-8	7.152
ASD N		Temporal Mid L	-58	-15	-5	20.409
		Temporal Sup L	-58	-16	4	19.128
		Cingulum Ant R	3	46	28	19.191
	Frontal Sup Medial R	3	47	31	18.058	
	Frontal Sup Medial L	2	46	25	20.277	
	Frontal Mid R	33	43	28	18.623	

N170 latency

The N170 latency values for both groups were normally distributed. The N170 latency showed statistical differences between groups with a shorter latency in the experimental group and a greater difference for the neutral face: *Exp-Neutral* = 181.75 ± 15.83 ms, *Ctrl-Neutral* = 192.75 ± 11.97 ms, *MD* = -11 ms, *p* = 0.040, *Cohen's d* = 0.784, and there are no statistical differences for the Duchenne smile face: *Exp-Duchenne smile* = 182.50 ± 16.96 ms, *Ctrl-Duchenne smile* = 187.58 ± 10.18 ms, *MD* = -5.08 ms, *p* = 0.202, *Cohen's d* = 0.363. Bayesian analysis across both face types further supported these findings, with strong evidence for shorter latency in the experimental group: *Exp* = 182.13 ± 16.40 ms, *Ctrl* = 190.17 ± 11.12 ms, *MD* = -8.04 ms, 95% *CI*: [-14.32, -1.76]. Within-group comparison, the experimental group showed minimal latency differences and no statistical differences between the Duchenne smile and neutral face: *Exp-Duchenne smile* = 182.50 ± 16.96 ms, *Exp-Neutral* = 181.75 ± 15.83 ms, *MD* = -11 ms, *MD* = -0.75 ms, *p* = 0.430, *Cohen's d* = 0.046, while the control group showed statistical differences between both faces: *Ctrl-Duchenne smile* = 187.58 ± 10.18 ms, *Ctrl-Neutral* = 192.75 ± 11.97 ms, *MD* = -5.17 ms, *p* = 0.043, *Cohen's d* = 0.465.

N170 source localization

When we compared EEG responses, while the children visualized the Duchenne smile and neutral faces at the N170 using Hotelling T² test, we found significant differences in brain activation in N170. Table 1 and Fig. 2 shows the significant differences located in the parietal occipital areas, orbitofrontal and cingulum areas in the control group. In the experimental group, the greater activation areas were located in the temporal, orbitofrontal and insular areas.

Discussion

Our study reveals significant differences in N170 when examining the relationship between the decoding of visual emotional perception and the neural response during facial emotion processing in ASD children. While many previous studies have reported delayed N170 latency in ASD compared to the typically developing group (McPartland et al., 2004; Dawson et al., 2005; Kang et al., 2017), our findings show the opposite pattern, where ASD children presented shorter latency in both face types and statistically significant differences when comparing with the control group during neutral face processing and with Cohen's *d* approaching a large effect. These differences to prior literature may be due to methodological variations, but also our use of the

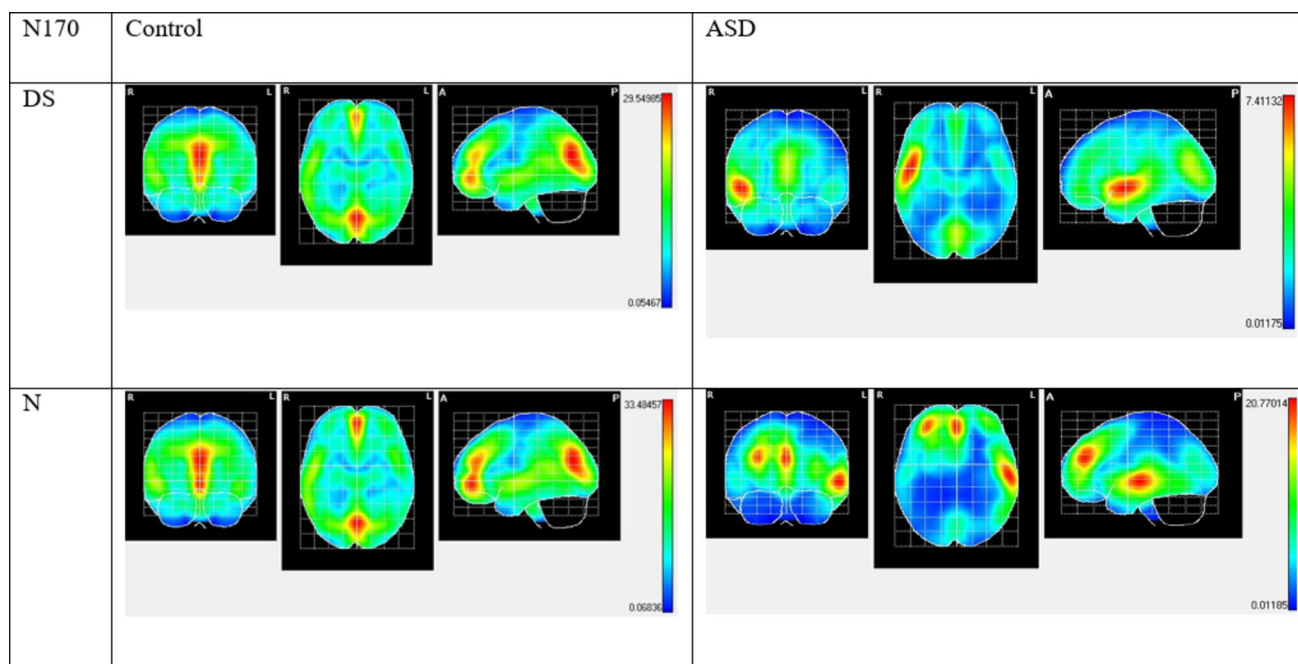


Fig. 2 N170 comparative mean source localization activation for DS and N faces in Control and ASD children

uniquely emotional Duchenne smile on a familiar face, which may have triggered more automatic visual processing. Additionally, Bayesian analysis provided strong evidence for shorter N170 latency in the ASD group compared to the control group, adding robustness to the observed group differences and supporting that the neural response differences are unlikely to be due to chance. These results suggest that their brains might not differentiate between emotional and neutral faces effectively, as a possible impairment development in their neural systems responsible for emotional recognition (Ewbank et al., 2017). Interestingly, when within-group comparison, ASD children showed minimal latency differences and no statistical differences between the Duchenne smile and neutral face processing, where the Cohen's *d* indicates an almost negligible effect. These findings may suggest an underlying deficit in their neural network responsible for differentiating emotions, resulting in a more consistent neural reaction across emotional stimuli (Kala et al., 2021).

These findings suggest a distinct cognitive processing approach in ASD children, with the N170 latency serving as one of the indicators of the timing of neural responses to face processing (Gliga & Haan, 2011). This pattern is possibly linked to differences in cognitive processing which indicates that ASD children may process facial structures differently. To ensure a more homogeneous sample and reduce variability, participants with co-occurring conditions, such as ADHD, were excluded from this study. Previous studies indicate how N170 latency is influenced by different facial emotional stimulus that can influence in their

latency and can affect their neural processing speed (Tye et al., 2013; Webb et al., 2017; Jones et al., 2021; Chen et al., 2020; Neuhaus et al., 2020). This is potentially due to the developmental and emotional factors affecting their cognitive encoding (Key & Corbett, 2019; Lagioia et al., 2010).

These results highlights the automatic nature of the N170 response, which represents an early stage of visual processing focused on facial attribute recognition, which sets a foundation for further cognitive and emotional evaluations. These examinations are probably influenced by cognitive empathy, defined as the understanding of emotions without necessarily experiencing them (De Jong et al., 2016). Literature suggests impairments in this cognitive empathy process in ASD (Ringshaw et al., 2022), which may limit the integration of effective empathetic responses.

Interestingly, some studies have observed a shorter latency on the psychotic spectrum during facial emotional processing (Martin et al., 2004) as in our study. Furthermore, there were no significant differences in latencies during emotional face processing when comparing the experimental and the control group (Wolfgang et al., 2012), which is in concordance with our results. When we scrutinize the literature review about ASD and psychosis spectrum, we observe that individuals with ASD and those experiencing primary psychosis often presented similar patterns of behavior and cognitive functioning, impeding to differentiate between symptoms of psychosis and those linked to ASD, therefore making the process of conducting a differential diagnosis more difficult (Crescenzo et al., 2019). On top of that, both disorders share epidemiological, environmental, and genetic

factors, and also present numerous similarities at the level of endophenotypes (Samaey et al., 2020).

An interesting ERP pilot study suggested that elevated callous-unemotional traits are linked to a modified neurophysiological pattern in children diagnosed with ASD (Tye et al., 2017). For this reason an interesting alternative hypothesis appears from neuroimaging studies, which points out the deviations from the predicted pattern of the ERP component (such as delays and/or short responses) could potentially not be unique to autism. Similar irregular N170 latency responses have been identified in different neurological and psychiatric conditions, notably in cases of primary psychosis. This suggests that these deviations can potentially indicate an impaired facial expression processing as a common symptom to these type of conditions, rather than representing a specific deficit to a particular disorder (Feuerriegel et al., 2015).

Instead of traditional amplitude measurements, we employed source localization to measure brain activity, focusing on neural activity, as illustrated in the source maps. The results consistently presented that the ASD children exhibited lower energy levels compared to the control group. Also, the most significant finding that we found in our study is the different neural pattern of the N170 source localization in ASD children, particularly during the processing of the Duchenne smile facial expressions. We observed a greater neuronal activity in the right hemisphere, specifically in the superior temporal, superior pole areas, and the insula. This activation pattern may suggest the presence of a compensatory cognitive mechanism, possibly as an adaptive response of the brain to deficits in emotional processing. It might indicate an alternative neural pathway that compensates for impaired emotional recognition. This finding aligns with previous research that suggests atypical hemispheric lateralization in ASD, where the right hemisphere may play a more prominent role in processing emotional stimuli (Wong et al., 2008) and confirms the significance of these differences through dipole source analysis (Sama et al., 2024). These regions, usually are associated with the processing of positive emotions, appear to take on an increased role in cognitive functions, possibly as a means to mitigate the challenges in the observer's ASD emotional processing (Eslinger et al., 2021).

Our investigation on the activation patterns in ASD children and the role of the right temporal lobe are in line with previous findings (McCarthy et al., 1997). We observed that the temporal areas activated in ASD children are consistent with the right lateralization typically associated with human face processing. These areas are well known for their involvement in face and visuospatial processing (Haxby et al., 2000) and also play a crucial role in emotion recognition (Raz et al., 2014). We observed a lack of activity in the

left temporal regions when ASD children were exposed to the Duchenne smile facial expressions, a contrasting pattern from their responses to neutral facial expressions.

Our literature review highlights several main symptoms associated with damage to the temporal lobe, including disturbances in visual perception, selective attention to auditory and visual inputs, and alterations in personality and affective behavior (Kolb & Wishaw, 2009). These symptoms are pertinent to our observations of neurological aberrations in domains related to emotions, sensory perception, and auditory processing in children with ASD. These abnormalities, attributed to cognitive impairments, are clear in the irregular activation patterns that we observed in the brain areas which are responsible for processing of human facial stimuli (Velikonja et al., 2019).

In our study we refer to these irregularities in the activation of areas who involved in lower-level visual processing, such as spatial and structural information processing, during face recognition tasks in ASD children (Chung & Son, 2020), which could be the result of a different pattern activation between both groups. Also, we observed that ASD children did not activate the cingulum areas during exposure to the Duchenne smiles, unlike the control group. The cingulum is crucial for social cognition, emotional processing, and motor control and comprises multiple sub-bundles with varied endpoints and functions (Amodio & Frith, 2006; Bush et al., 2000; Paus, 2001; Hau et al., 2019). This lack of activation suggests that ASD children might engage an autonomic output linked to an altered relationship between cingulum microstructure and executive functions, as a possible compensatory mechanism in their disorder (Hau et al., 2019).

Furthermore, our findings parallel patterns observed in conditions such as psychosis, where there is an activation increase of the spatial awareness-related to these brain regions, particularly in the right temporal lobe, during the N170 face recognition processing (Toal et al., 2009). This could indicate a similar and broader neurobiological commonality underlying these different conditions. Also, only the right temporal lobe is associated with spatial awareness, which includes the understanding of spatial relationships and recognizing landmarks (Karnath et al., 2001), and ASD may rely more on lower-level visual processing, such as spatial and structural information, during face recognitions tasks (Chung & Son, 2020). This could explain why individuals with autism may present different activation patterns during face recognition tasks. This pattern in ASD may be related to a focus on processing individuals features and spatial relationships with faces, as opposed to the more holistic and emotion-focused processing that is typically associated with the N170 response in neurotypical individuals (Khuntia et al., 2019). It is important to reiterate the

observation that ASD manifested a pattern of similarity with the psychosis neuroimaging commonalities suggest shared mechanism between both conditions (Moreau et al., 2021).

In addition, the right insula is primarily specialized in sensory functions, while the left insula is primarily associated with emotional functions (Yamada et al., 2016). Neuroimaging studies focusing on the insular cortex in children with ASD frequently report structural and functional abnormalities, particularly in the right insula. These abnormalities include important changes in gray matter within key functional regions engaged during face recognition tasks (Yamada et al., 2016; Ecker et al., 2013). The literature review related to ASD exhibits deficits in empathetic and emotional cues (Minio-Paluello et al., 2021; Vilas et al., 2021), which are associated with impairments in the previously mentioned brain areas. The right insula is an integral component of the brain's ventral frontoparietal attention network, responsible for automatically directing attention to salient stimuli in the environment (Uddin, 2015). Consequently, we postulate that activation of the right insula in individuals with reduced empathy can be clarified by its larger role in processing emotional and physiological responses. Even where empathy is less pronounced, the brain continues to use the appropriate insula to process the emotional and bodily facets of stimuli. This indicates the active involvement of the brain in understanding emotions and bodily reactions, even among individuals who do not manifest strong empathetic responses.

There was no noticed social emotion cognition in ASD children, located in the orbitofrontal cortex, according with literature (Rolls & Grabenhorst, 2008; Rempel-Clower, 2007). When this region (orbitofrontal) fails in the control and modulation of emotions' reaction, engendered by the limbic structures, it can cause abnormal facial expression processing and cognitive abnormalities (Mikhailova et al., 2021). For this reason, we speculate that the absence of activation in this area may present a dysfunction developmental pathology impairment, where the literature supports our observation, suggesting having found a disrupted signal transmission for social interactions and emotions in the orbitofrontal cortex areas in ASD (Liu et al., 2020). On top of this, studies showed that ASD children presented lower fractional anisotropy values that can suggest compromised microstructure or inclusive disrupted neural connections in their brain, (Ikuta et al., 2014). This can suggest an altered neural connectivity that could be related to difficulties in social cognition and emotion processing (Amodio & Frith, 2006; Bush et al., 2000; Ameis et al., 2013; Catani et al., 2016; Fitzgerald et al., 2019; Hau et al., 2019; Ikuta et al., 2014; Koolschijn et al., 2017).

Conclusion

In conclusion, children with ASD exhibited deficits in social cognitive functions characterized by reduced activation in the dorsal brain pathway and orbitofrontal areas. Our findings also identified alterations in the cingulum, functionally relevant to enhanced executive functioning that directly influences emotion regulation processes. The observed outcomes within the insula and the right temporal lobe are particularly noteworthy, suggesting potential alterations in neural processing linked to spatial perception and unique cognitive processes during face recognition tasks.

Our study reveals that brain functions and connectivity patterns in ASD children differ significantly from those in the typically developing population. This finding indicates a unique and complex neural dynamic in ASD, particularly in the context of the emotional processing. Contrary to what is typically observed in healthy individuals, these altered neural patterns in ASD may have parallels with conditions like psychosis and/or callous-unemotional traits (Carter et al., 2015; Chisholm et al., 2015; Kincaid et al., 2017; Ribolsi et al., 2022). These connections can potentially imply a shared underlying mechanisms between ASD and these conditions. On top of that some studies suggest that the essential multimodality of emotion recognition in ASD may have their origin in the early social information processing speed, despite the heterogeneous behavioral outcomes. These findings can suggest a new target for social emotional interventions in ASD (Keifer et al., 2019; Lerner et al., 2013) and understanding the neural mechanisms underlying these deficits could inform the development of brain stimulation technique to enhance emotional processing. For this reason it is crucial to further research this area to completely understand and address these complexities.

To summarize, our study initially indicated that children with ASD should be capable of perceiving the structural features of facial visual stimuli. However, our findings suggest a marked difficulty in effectively decoding and activating the corresponding brain regions when processing these visual cues. This challenge is probably linked to impaired emotional cognition in ASD, stemming from disrupted neural pathways. It is important to note that these disruptions likely involve complex interactions between different brain regions responsible for emotional processing. The observed alterations in brain function and cognitive impairment, and the unique neural circuitry in ASD, appear to contribute significantly to disrupted perception, especially in contexts that involve emotional processing such as the Duchenne smile face recognition. These findings illustrate the complex nature of neural processing in children with ASD, pointing out the importance of a detailed understanding of the relationship between neural circuitry and cognitive function. It

is important to focus on the observed ERP patterns in ASD, which can suggest a potential overlap with other neurodevelopmental or psychiatric conditions, such as psychosis. This observation warrants further investigation into possible comorbidities, thus deepening our understanding of ASD and its multifaceted neurobiological connections.

Limitations

This study has numerous strengths as an exploratory study which introduces a novel study of the Duchenne smile face neural responses in ASD children, a topic that has remained underexplored. But it also has some limitations, such as the sample size used, due to the challenges of recruiting ASD children that met the strict criteria like confirmed absence of comorbid in psychiatric disorders, and also remained seated and focused during the EEG-based ERP data collection and processing. It is important to note that the findings provided in this study point to a very valuable preliminary evidence for future investigations in large scale studies to confirm and expand on these results. Also, we consider that investigating this less explored topic in more depth is essential to understand the evolution of their neural mechanism over time which can help comprehend how these patterns evolve and how to respond to interventions.

While this study provides valuable preliminary evidence, the small sample size and gender composition may introduce certain limitations in the generalization of the results. In particular, the requirement for children with ASD to remain seated and focused may have favored the inclusion of participants with better behavioral regulation, potentially biasing the sample toward a specific subgroup within the autism spectrum. Additionally, the sample composition, with a distribution of six boys and four girls in the ASD group, could be a factor to consider in the interpretation of the findings, as previous studies have reported possible differences in emotional processing between genders. While the groups were matched for socioeconomic status, educational level, ethnicity, and without non diagnosed with psychiatric conditions, we did not conduct detailed individual assessments of their socioeconomic background nor subclinical symptoms, which could introduce minor unmeasured variability. Future studies with larger samples and more diverse representation will allow for a more robust understanding of these mechanisms.

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