Research article

Impaired neural discrimination of emotional speech prosody in children with autism spectrum disorder and language impairment

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HIGHLIGHTS

- Children with ASD encode naturally articulated words atypically.
- Cortical discrimination of emotional prosody is impaired in children with ASD.
- Children with ASD have deficits in involuntary orienting to prosodic changes.

ARTICLE INFO

ABSTRACT

Autism spectrum disorders (ASD) are characterized by deficient social and communication skills, including difficulties in perceiving speech prosody. The present study addressed processing of emotional prosodic changes (sad, scornful and commanding) in natural word stimuli in typically developed school-aged children and in children with ASD and language impairment. We found that the responses to a repetitive word were diminished in amplitude in the children with ASD, reflecting impaired speech encoding. Furthermore, the amplitude of the MMN/LDN component, reflecting cortical discrimination of sound changes, was diminished in the children with ASD for the scornful deviant. In addition, the amplitude of the P3a, reflecting involuntary orienting to attention-catching changes, was diminished in the children with ASD for the scornful deviant and tended to be smaller for the sad deviant. These results suggest that prosody processing in ASD is impaired at various levels of neural processing, including deficient pre-attentive discrimination and involuntary orientation to speech prosody.

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1. Introduction

Autism spectrum disorders (ASD) are characterized by deficient social and communication skills, and repetitive patterns of behavior [1,2]. Impaired Theory of Mind (ToM), referring to the ability to infer other people's mental states, can be considered as one of the core deficits behind these social and communicative impairments [3]. Speech prosody conveys information on ToM-related factors such as emotional state or intention of the speaker, as well as linguistic information, via pitch, intensity, and duration variations of speech [4]. This study explores processing of acoustic realizations of emotional prosody on low-level event related brain potentials (ERPs) in children with ASD.

Some behavioral studies have reported deficits in emotional speech prosody comprehension in ASD [5,6], whereas some other studies show no such deficits [6,7]. These conflicting results can be explained by differences in methodologies used: the variety of stimulus material (possibly requiring ToM to varying degrees), the clinical group, the age of participants, the experimental instructions [8], and the cognitive task demands [9]. Auditory ERPs are feasible for studying prosody perception in ASD as the recordings can be done in absence of participants' attention and performance [10]. Detection and encoding physical stimulus features are reflected by ERP waveforms of P1, N2 and N4 [11]. Mismatch negativity (MMN) is an ERP component reflecting pre-conscious detection of violations of auditory regularities [12; for MMN studies of speech sound discrimination in ASD, please see 13]. In children the MMN is often followed by a late discriminative negativity (LDN, or Late MMN) that is hypothesized to reflect more cognitive aspects of auditory change detection than the MMN [14]. When the stimulus deviation catches participants' attention the MMN is often followed by a

positive P3a indexing attention switch towards stimulus deviation [15].

Enhanced MMNs have been found in children with ASD (but no language or cognitive delays) to words pronounced with an angry voice occasionally occurring among tenderly-uttered words [16]. However, Kujala et al. [17], recording from adults with ASD MMN to occasional commanding, sad, or scornful deviants presented among a neutrally-uttered repetitive standard word, found a diminished MMN amplitude for the scornfully-uttered word. Furthermore, their MMN to the commanding deviant had a longer latency than the MMN of control participants [17]. These diminished MMN amplitudes and prolonged MMN latencies indicate impaired and sluggish low-level neural discrimination of prosodic features in participants with ASD [17]. The results of these two studies [16,17] are consistent with the notion of both hyper- and hyposensitive sensory processing in ASD [13,18,19], but may also be explained by differences in the age of the participants or the type of the stimuli [13].

We aimed to determine how children with ASD encode, discriminate and orient to prosodic changes among a stream of naturally articulated words. To this end auditory ERPs were recorded from 7 to 12 years old typically developed children and children with ASD using the same stimuli and paradigm as Kujala et al. [17]. On the basis of Kujala et al. [17] we hypothesized that participants with ASD would neurally discriminate the sad and scornful prosodic changes more poorly compared to control participants as reflected by a diminished MMN amplitude. However, on the basis of Korpilahti et al. [16] we expected that participants with ASD might react in a hypersensitive manner to the commanding deviant as reflected by an enhanced MMN.

2. Material and methods

2.1. Participants

10 children with ASD fulfilling the ICD-10 [1] criteria for Childhood Autism and DSM-IV criteria for Autistic Disorder [2] participated in the experiment (9 boys; 1 left handed; mean age 10.5 years, sd 1.3, range 8.6–12.2 years). They were recruited from the Helsinki University Central Hospital and the Central Hospital of Central Finland and had a clinical ICD-10 diagnosis of Childhood Autism [1]. The diagnosis had been made by experienced clinicians working in multidisciplinary teams. The Autism Diagnostic Interview-Revised (ADI-R; [20,21] and the medical records were used to get supplementary diagnostic information. Due to timing/other personal reasons three families refused to participate in ADI-R interview. However, these children were included in the analysis as the validity of Finnish register-based diagnosis of autism is high; according to Lampi et al. [22] study, 96% of children with registered diagnosis of autism fulfilled the ADI-R diagnostic criteria as well. The mean total score of the ASD group on the Childhood Autism Rating Scale (CARS, [23]) was 32 points (range 29–36.5, sd 2.4; data unavailable from one participant). All the children were unmedicated, and based on the medical records had normal EEG, MRI, and chromosomes. Children with ASD were enrolled in special classes for children or for children with autism or learning problems.

The control group consisted of 13 age- and sex-matched participants recruited from elementary schools in the Helsinki area (12 boys; 1 left handed; mean age 10.0 years, 1.5, range 7.5–11.8 years). They had no past or present neurological disorder, language or learning difficulty, and no emotional problems. Furthermore, based on parental reports they had no family history of autism spectrum disorders or any other developmental or psychiatric disorder.

All participants in both groups were monolingual Finnish speakers with normal hearing.

The cognitive abilities of the children with ASD had been assessed in hospital with the Finnish version of the Wechsler Intelligence Scale for Children (WISC) III or IV [24,25], or the Leiter International Performance Scale-Revised [26]. The control children's cognitive abilities were assessed with WISC-III [24]. The mean performance IQ (PIQ) of the children with ASD was 100 (range 77–104, sd 10), and the mean verbal IQ (VIQ) 63 (range 46–98, sd 19; data not available on three children with very limited expressive language). The control participants' mean PIQ was 110 (range 85–136, sd 12), and the mean VIQ 117 (range 83–144, sd 17).The VIQ difference between the groups was high as expected ($F[1,19] = 43.6$, $P < 0.000$). The group difference in PIQ was also significant ($F[1,21] = 15.6$, $p > 0.001$). However, regression analyses computed separately for each group and contrast indicated no significant linear relationship between the PIQ and the ERP deflections in any condition, suggesting no influence of PIQ differences on group differences.

2.2. Stimuli and procedure

The stimuli were Finnish words (female name "Saara") uttered by a female speaker neutrally and with scornful, commanding, or sad voice. The stimuli were originally developed by Leinonen et al. [27] and used by Kujala et al. [17] and Lindström et al. [28] (For the acoustic parameters of the stimuli, see Lindström et al. [28]).The parents signed an informed consent and the children gave their assent prior to the experiment, which was carried out according to the Declaration of Helsinki and approved by the Ethical Committees of the Helsinki University Central Hospital and Central Hospital of Central Finland.

During the electroencephalogram (EEG) recordings eight stimulus blocks were presented, each containing 268 stimuli. Each block consisted of a frequently presented neutrally-uttered standard stimulus (79%), occasionally replaced with a commanding (7%), sad (7%), or scornful (7%) deviant. The stimuli were presented pseudorandomly with each deviant stimulus being preceded by at least two standard stimuli. The stimulus onset asynchrony (SOA) was 1300 ms. The stimuli were presented via loudspeakers OWI-202 (OWI Inc. CA., USA) at 56 dB (SPL) which were located in front of the child, on the left and right side of the screen that the children were watching during the recordings (at a 157 cm distance from participant's head, 108 cm apart from each other). During the experiment, the children sat in an armchair watching self-chosen soundless video in an electrically and acoustically shielded room and were instructed to pay no attention to the sounds. They were accompanied by their parent if necessary and video-monitored during the whole experiment, which took about an hour including breaks.

2.3. ERP recording and analysis

The EEG was continuously recorded (DC–104 Hz; sampling rate 512 Hz) with Biosemi Active Two Mk2 with a 64-channel active electrode set-up [29]; additional electrodes were placed at the mastoids. Vertical and horizontal eye movements were monitored with electrodes placed above and at the outer canthus of the left eye. The off-line reference electrode was attached to the tip of the nose. The continuous EEG was down-sampled to 256 Hz and off-line high-pass filtered (1 Hz) by using the EEGLAB toolbox [30]. The EEG was divided into 1100 ms long epochs, including a 100 ms pre-stimulus baseline. The epochs were baseline-corrected with respect to the mean voltage of this pre-stimulus period. Epochs with EEG changes exceeding $\pm 300\,\mu V$ at any electrode were discarded. An independent component analysis with run ICA [30]

algorithm was performed to remove blink artifacts (one component per participant). The original EEG data and ICA-corrected data of each child were manually compared to ensure that only the blink artifacts were removed with ICA algorithm. Thereafter, a 20-Hz low-pass filter was applied. The epochs with voltage changes exceeding 100 µV in the channels used in the statistical analyses were omitted. Responses distorted due to broken channels were discarded and the response from the neighboring channels were interpolated (0–3 electrodes/participant). ERPs were averaged in each condition separately for the standard and deviant stimuli. Difference waves were calculated by subtracting the ERPs elicited by the standard stimuli from the ERPs elicited by the deviant stimuli. The data were re-referenced to the average of the left and right mastoid recordings. The final data set consisted of, on average, 114 accepted deviant trials (range 71–146) for ASD children and 135 accepted deviant trials (range 81–149) for control children.

The peak latencies of the standard stimulus ERPs were identified at the central Cz electrode for each group separately. Thereafter, the mean amplitudes from the individual-participant ERPs were calculated over a ±25-ms time period centered at this grand-mean peak latency. The individual-participant peak latencies were identified at the Cz electrode with the following time windows: 1st peak as the maximum positivity between 100 and 300 ms; 2nd: the maximum negativity between 300 and 400 ms, 3rd: the maximum positivity between 400 and 600 ms and 4th: the maximum negativity between 600 and 800 ms.

The mean amplitudes and latencies of the responses elicited by the deviant sounds were identified correspondingly from deviant-standard difference waves at the Cz electrode with the following time windows: the MMN/LDN as the maximum negativity between 400 and 600 ms and the P3a as the maximum positivity between 600 and 900 ms.

2.4. Statistical analyses

The statistical significance of each deflection was analyzed with one-sample *t*-tests by comparing the mean amplitudes to zero at Cz (Table 1). Repeated measures analyses of variance (ANOVA) were used to investigate the between-group amplitude differences of ERPs that were significantly elicited in at least one of the groups and scalp distribution differences of ERPs that were significantly elicited in both groups. Latencies were analyzed with one-way ANOVAs.

Group differences of the amplitudes of each ERP deflection were analyzed with two-way ANOVAs (Group x Electrode: Fz, F3, F4, C3, Cz, C4). The scalp distribution differences of standard stimulus ERPs and P3a were investigated using three-way ANOVAs [Group x Anterior-Posterior (F3, Fz, F4/C3, Cz, C4/P3, Pz, P4) x Laterality (F3, C3, P3/Fz, Cz, Pz/F4, C4, P4)] and those of MMN/LDN were investigated using four-way ANOVAs [Group x Deviant x Anterior-Posterior (F3, Fz, F4/C3, Cz, C4/P3, Pz, P4) x Laterality (F3, C3, P3/Fz, Cz, Pz/F4, C4, P4)]. The latencies of each ERP deflection were analyzed separately with one-way ANOVAs.

For each ERP mean amplitude and latency ANOVAs the statistically significant main effects were further analyzed with Sidak post-hoc comparisons. Mauchly's Test of Sphericity served to test the assumption of equality of variances. Degrees of freedom were corrected with Huynh-Feldt correction when the assumption of sphericity was violated.

3. Results

The standard stimuli elicited an ERP consisting of four peaks (Fig. 1, Table 1). In controls the 1st, 3rd and 4th peak were significantly different from zero (1st peak: $t(12) = 11.27$, $p < 0.0001$; 2nd: $t(12) = 11.63$, $p < 0.0001$; 4th: $t(12) = -7.06$, $p < 0.0001$) and
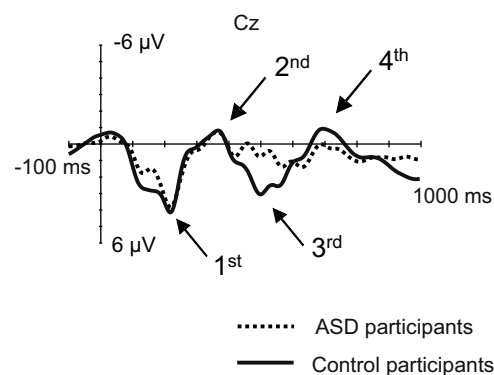

Fig. 1. The ERPs elicited by the standard stimulus at the Cz electrode. The onset of the sound is at 0 ms.

the 2nd marginally significant ($t(12) = -1.9$, $p = 0.08$). However, in children with ASD only the first peak was significant $t(9) = 10.6$, $p < 0.0001$). The amplitude of the 3rd ($F[1,21] = 25.6$, $p < 0.001$) and 4th $F[1,21] = 12.70$, $p < 0.01$) peak of these children was smaller than that of controls. The latencies of the peaks did not differ between the groups.

All the deviant stimuli elicited a MMN/LDN component within 500 ms from the stimulus onset (Fig. 2, Table 1), the amplitude which was statistically significant for each deviant stimulus in control children (scornful deviant: $t[12] = -6.36$, $p < 0.0001$; commanding: $t[12] = -3.36$, $p < 0.01$; sad: $t[12] = -6.38$, $p < 0.0001$), whereas in children with ASD it was significant only for the scornful ($t[9] = -2.85$, $p < 0.05$) and sad deviants ($t[9] = -3.16$, $p < 0.05$) (Table 1). A significant group main amplitude effect for the scornful deviant ($F[1,21] = 8.15$, $p < 0.01$) was found resulting from a diminished response in children with ASD. Scalp distribution comparisons of the MMN/LDN for the scornful and sad deviants showed a significant GROUP x DEVIANT x AP interaction effect ($F[3.06, 61.15] = 3.06$, $p < 0.05$). Post-hoc comparisons indicated that the MMN/LDN amplitude for the scornful deviant was diminished in children with ASD at frontal ($p < 0.01$) and central ($p < 0.05$) scalp areas. The latencies of the MMN/LDN did not differ between the groups.

All the deviant stimuli elicited a P3a component within 740 ms from stimulus onset in control children and 790 ms in ASD children from stimulus onset (Fig. 2, Table 1). In control children the P3a amplitude was statistically significant for the scornful ($t[12] = 5.87$, $p < 0.0001$) and sad ($t[12] = 5.42$, $p < 0.0001$) deviants and marginally significant for the commanding deviant ($t[12] = 2.1$, $p = 0.058$) (Table 2). In children with ASD the P3a amplitude was statistically significant for the commanding ($t[9] = 2.54$, $p < 0.05$) and sad ($t[9] = 3.54$, $p < 0.01$) deviants and marginally significant for the scornful ($t[9] = 2.21$, $p = 0.063$) deviant. A significant group main amplitude effect was found for the scornful deviant ($F[1,21] = 18.78$, $p > 0.001$), and a marginally significant one for the sad deviant ($F[1,21] = 2.30$, $p = 0.09$). Additionally, a marginally significant group x laterality interaction effect was found for the sad deviant ($F[1.72, 36.02] = 2.55$, $p = 0.09$), resulting from diminished amplitudes in children with ASD at the central electrodes ($p = 0.09$). The P3a latencies did not differ between the groups.

4. Discussion

Our study determined whether children with ASD have abnormalities in the low-level neural processing of emotional prosodic changes in natural speech by recording ERPs to word stimuli uttered neutrally, or with scornful, commanding, or sad voice. Firstly, impaired stimulus processing in ASD was evident in the smaller number of significantly elicited ERPs to the standard and

Table 1
Mean latencies and amplitudes (the standard deviation in brackets) of the brain responses at the Cz electrode.

Response	Stimulus type	ASD		Control	
		Amplitude μV (sd)	Latency ms (sd)	Amplitude μV (sd)	Latency ms (sd)
1st	Standard	3.36 (1)***	205.47 (29.36)	3.6 (1.15)***	207.63 (22.31)
2nd	Standard	−0.58 (1.42)	357.20 (21.85)	−0.6 (1.15)	360.58 (18)
3rd	Standard	0.54 (1.04)	514.45 (59.5)	2.79 (0.86)***	513.52 (35.43)
4th	Standard	0.16 (0.96)	704 (40.46)	−0.81(0.41)***	697.71 (32.70)
MMN/LDN					
	Scornful	−2.48 (2.76)*	510.55 (62.3)	−5.12 (2.9)***	493.7 (42.21)
	Commanding	−1.48 (2.94)	498.44 (55.03)	−1.92 (2.07)**	503 (37.47)
	Sad	−2,76 (2.76)*	469.36 (50.07)	−4.8 (2.7)***	453.12 (43.7)
P3a					
	Scornful	0.92 (1.37)	792.19 (63.94)	3.4 (2.12)***	750 (57.59)
	Commanding	1.71 (2.12)*	750.39 (62.81)	1.16 (2)	723.86 (69.59)
	Sad	1.56 (1.5)**	784.38 (61.46)	3.28 (2.18)***	748 (42.69)

The amplitudes significantly differing from the zero are marked with asterisks: *P < 0.05, **P < 0.01, and ***P < 0.001.
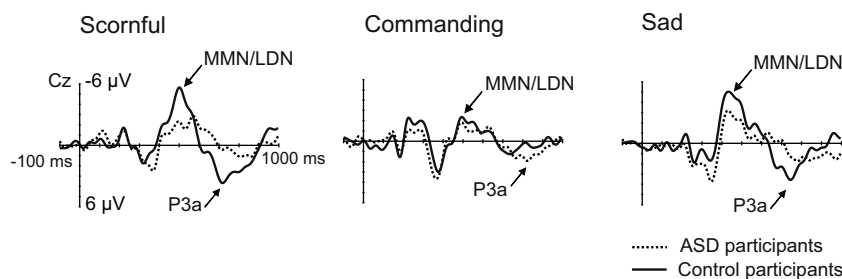
Fig. 2. The grand-mean difference waves for scornful, commanding, and sad deviants at the Cz electrode. The onset of the sound is at 0 ms.

deviant stimuli. Furthermore, smaller standard-stimulus ERPs (3rd and 4th peaks), MMN/LDN, and P3a components were found in children with ASD than in controls.

Our results of diminished standard stimulus ERPs [11] in children with ASD are consistent with previous studies reporting diminished N4 amplitudes in children with ASD with a language impairment [18] and those with ASD but no language or cognitive delays [19, see however, 13]. These results suggest speech encoding deficits in children with ASD.

The amplitude of the MMN/LDN, reflecting pre-attentive neural discrimination of acoustic [12] and prosodic changes [28], tended to be smaller in the participants with than without ASD for all deviant stimuli (Fig. 2). However, a significant group difference was only found for the scornful deviant. Our results are consistent with those of Kujala et al. [17] reporting diminished MMN amplitudes to this deviant, but not to the sad or commanding deviants in adults with ASD. The scornful deviant is likely to be hardest to discriminate from the neutral stimulus as suggested by Lindström et al. [28] study using the same stimuli in a discrimination test and reporting longest reaction times for the scornful deviant in typically developed school-aged children.

One could also speculate whether the impaired cortical discrimination of the scornful prosodic change in participants with ASD is associated with their deficient ToM. The commanding and sad stimuli could be classified as representing basic emotion category and requiring very little if any ToM, whereas the scornful stimuli might represent a social emotion category and thus requiring ToM [31]. This should be investigated in future studies determining the link between behavioral and neural emotional prosody perception and measures reflecting ToM.

Based on Korpilahti et al. [16] we expected hypersensitive reactions to the commanding deviant in ASD group. Instead, this change elicited no statistically significant MMN/LDN in these participants. Possibly, compared with our stimuli, the stimulus difference

between the angry vs. tender stimuli in Korpilahti et al. [16] study was acoustically more salient, causing enhanced responses in the ASD group. Furthermore, Korpilahti et al. [16] used only one (p = 0.15) deviant stimulus, possibly easier for the auditory system to detect than the three different deviant stimuli used in our and Kujala et al. [17] study (p for each deviant = 0.07). Finally, participants with ASD had no cognitive or language impairments in Korpilahti et al. [16] study, whereas our group consisted of children with ASD and language impairment.

Our results suggest that the neural basis of processing natural prosodic changes in ASD is deficient even at the early pre-attentive neural level. This can be expected to hamper further steps of prosody perception since these responses were shown to be associated with perceptual accuracy in number of studies [12]. This interpretation is supported by our finding of impaired involuntary orienting to prosodic changes in children with ASD. The amplitude of the P3a, reflecting an attention switch towards attention-capturing changes [15,28], was diminished in children with ASD for the scornful deviant and it tended to be diminished for the sad deviant, suggesting abnormal orientation to emotional speech sound changes. Possibly, the poor discrimination of emotional prosodic features as reflected by MMN leads to impaired orienting to emotional prosodic changes in children with ASD and language impairment.

To summarize, our results indicate that the processing of words and changes in prosodic information is impaired in school-aged children with ASD. Deficits were seen both at low-level speech sound encoding and discrimination, as well as in the subsequent attention switching processes. These prosody processing deficits emerging at various levels of information processing might contribute to social communication impairments in ASD and support the hypothesis that difficulties in social communication have low-level neurofunctional origins in ASD.

Acknowledgments

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