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Research article

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

R. Lindström[a,*], T. Lepistö-Paisley[a,b], R. Vanhala[b], R. Alén[c], T. Kujala[a]

[a] Cognitive Brain Research Unit, Institute of Behavioural Sciences, University of Helsinki, Helsinki, Finland
[b] Department of Child Neurology, Helsinki University Central Hospital, Helsinki, Finland
[c] Department of Child Neurology, Central Finland Central Hospital, Jyväskylä, Finland

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.

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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.

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

* Corresponding author at: Cognitive Brain Research Unit, Institute of Behavioural Sciences, P.O. Box 9 FIN-00014, University of Helsinki, Finland.
E-mail address: riikka.h.lindstrom@helsinki.fi (R. Lindström).

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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]