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The neural basis of aberrant speech and audition in autism spectrum disorders

T. Kujala^{a,b,*}, T. Lepistö^{b,c}, R. Näätänen^{b,d,e}

^a Cicero Learning, P.O. Box 9, FI-00014 University of Helsinki, Helsinki, Finland

^b Cognitive Brain Research Unit, Cognitive Science, Institute of Behavioral Sciences, P.O. Box 9, FI-00014 University of Helsinki, Helsinki, Finland

^c Department of Child Neurology, Hospital for Children and Adolescents, Lastenlinnantie 2, 00250 Helsinki, University of Helsinki, Finland

^d Institute of Psychology, University of Tartu, Tartu, Estonia

^e Centre of Integrative Neuroscience (CFIN), University of Aarhus, Aarhus, Denmark

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ABSTRACT

Autism spectrum disorders (ASD) are characterized by deficits in communication and social behavior and by narrow interests. Individuals belonging to this spectrum have abnormalities in various aspects of language, ranging from semantic-pragmatic deficits to the absence of speech. They also have aberrant perception, especially in the auditory domain, with both hypo- and hypersensitive features. Neurophysiological approaches with high temporal resolution have given novel insight into the processes underlying perception and language in ASD. Neurophysiological recordings, which are feasible for investigating infants and individuals with no speech, have shown that the representation of and attention to language has an abnormal developmental path in ASD. Even the basic mechanisms for fluent speech perception are degraded at a low level of neural speech analysis. Furthermore, neural correlates of perception and some traits typical of subgroups of individuals on this spectrum have helped in understanding the diversity on this spectrum.

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1. Introduction

Autism spectrum disorders (ASD) are characterized by deficits in communication and social behavior and by narrow interests. Individuals with ASD have abnormalities in various aspects of language, ranging from semantic-pragmatic deficits to the absence of speech. They also have aberrant perception, especially in the auditory domain, with both hypo- and hypersensitive features. Genes have a strong contribution to ASD. The concordance rates in monozygotic twins are 70–90%, whereas in dizygotic twins these rates are only 0–10% (Nickl-Jockschat and Michel, 2011). The neurobiological abnormalities in ASD include the atypical formation or elimination of neural connections, a lower than normal number of Purkinje cells, diminished neuronal size, and decreased dendritic branches (Nickl-Jockschat and Michel, 2011). Furthermore, brain growth during development is accelerated in autism (Sparks et al., 2002).

^{*} Corresponding author at: Cicero Learning, P.O. Box 9, FI-00014 University of Helsinki, Helsinki, Finland. Tel.: +358 9 19129838; fax: +358 9 19129450. *E-mail address:* teija.m.kujala@helsinki.fi (T. Kujala).

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ASD include neurodevelopmental variants, with the main diagnostic subgroups being autism and Asperger syndrome (AS) (WHO, 1993; American Psychiatric Association, 1994). The primary difference between autism and AS is in language development. Children with autism show significant delays and abnormalities in language development, with about half of them remaining nonverbal or with only little functional language (Gillberg and Coleman, 2000). Semantic-pragmatic deficits are widespread in autism (Rapin and Dunn, 2003), entailing, for instance, poor conversation skills and an impaired understanding of nonliteral language. Furthermore, deficits in conceiving and producing prosody are common (McCann and Peppe, 2003; Paul et al., 2005; Peppe et al., 2007). The development of language structure is rather normal in AS (WHO, 1993), whereas there are deficits in semantic-pragmatic skills, including deficient prosody (Adams et al., 2002; Shriberg et al., 2001), similarly as in autism.

Impairments in social interactions are considered to constitute the primary dysfunction in ASD, with deficits in communication being their secondary consequences (Mundy and Neal, 2001; Wing, 1988). However, impaired central auditory processing might also contribute to the language deficits observed in these disorders (Bomba and Pang, 2004; Rapin, 2002). This notion is supported by evidence suggesting that in autism structural and functional abnormalities exist in brain areas involved in language and auditory processing. For example, magnetic resonance imaging has revealed an abnormal asymmetry of frontal and temporal language areas (De Fosse et al., 2004; Herbert et al., 2002; Rojas et al., 2002). The volume of the left but not the right planum temporale was found to be reduced in adults with autism (Rojas et al., 2002). Furthermore, in children with autism, the volume of Broca's area in its right hemisphere homologue was larger in a language-impaired sample, whereas larger left Broca's area volumes were observed in a language unimpaired sample (De Fosse et al., 2004). Regional cerebral blood flow recordings during rest, in turn, indicated bilateral hypoperfusion in the superior temporal gyrus (Gendry Meresse et al., 2005; Ohnishi et al., 2000; Zilbovicius et al., 2000). Furthermore, listening to tones or completing sentence comprehension tasks showed diminished activation in the left-hemisphere language-related areas in autism (Boddaert et al., 2003, 2004; Müller et al., 1999).

Besides deficits in language and communication, autism and AS are characterized by aberrant perception (Dahlgren and Gillberg, 1989; Dunn et al., 2002; Gillberg and Coleman, 2000; Kern et al., 2006; Leekam et al., 2007; Kientz and Dunn, 1997; O'Neill and Jones, 1997; Talay-Ongan and Wood, 2000), which involves all sensory modalities, particularly the auditory one (Dahlgren and Gillberg, 1989). The aberrant auditory functions involve both hyper- and hyposensitivity. A child may seem to be inattentive to his/her name, may ignore loud sounds, or may seek auditory stimulation by producing sounds. Impaired phonological processing skills, as assessed with non-word repetition tasks, were also reported (Bishop et al., 2004; Kjelgaard and Tager-Flusberg, 2001). Furthermore, individuals with ASD have increased difficulty in perceiving speech in noisy environments (Alcantara et al., 2004).

Some behaviors in ASD, in turn, indicate auditory hypersensitivity. Individuals with ASD may become distressed by sounds, often trying to avoid sounds by covering their ears, and some of them appear to have superior hearing. Clinical observations have also reported relatively good musical skills in autism, such as case descriptions of musical savants with ASD (Heaton et al., 1999; Jones et al., 2009). In addition, some reports suggest an increased prevalence of absolute pitch (Rimland and Fein, 1988), superior pitch memory and pitch-discrimination skills (Bonnel et al., 2003; Heaton et al., 2001, 2008; Heaton, 2003, 2005; Khalfa et al., 2004; Mottron et al., 2000; O'Riordan and Passetti, 2006) in ASD. Individuals with ASD evidently form a very heterogeneous group with regard to their perceptual abilities. For example, enhanced frequency discrimination abilities were reported in 20% of individuals in a group of 72 adolescents with ASD (Jones et al., 2009). This heterogeneity could be explained by the complex genetic etiology of ASD, for example, autism has been suggested to be a manifestation of tens or even hundreds of genetic and genomic disorders (Betancur, 2011).

Children with ASD also exhibit abnormal orienting to sensory events. As early as at the age of 6–12 months, their social orienting was observed to be abnormal, whereas their reactions to non-social stimuli resembled those of typically developing infants (Maestro et al., 2002). Moreover, preschoolers with ASD show an abnormal orientation both to social and non-social auditory stimuli, in particular to social stimuli (Dawson et al., 2004). Furthermore, individuals with ASD prefer non-speech to speech sounds (Blackstock, 1978; Klin, 1991).

2. Neurophysiological responses as tools for ASD research

In order to understand the neural basis of complex disorders such as the autism spectrum, information provided by a wide range of brain research methods is needed. While hemodynamic methods, which are unmatched in spatial resolution, show brain regions that are normally or abnormally activated, neurophysiological approaches (event-related potentials, ERP; event-related magnetic fields, ERF) have the advantage of revealing stimulus-specific neural responsiveness with a temporal resolution of milliseconds. This approach enables the untangling of the different brain processes elicited by different stimuli and changes in the regularities of the stimulus environment (Näätänen et al., 2001; Winkler, 2007). Some of the neural responses are even elicited involuntarily, irrespective of the individual's primary task or direction of attention. These responses are very helpful for investigating the perception and cognition of individuals with limited or no communication ability, for example, those asleep, infants, aphasic patients, or children with autism.

Neural stimulus encoding can be investigated by recording a series of deflections elicited by stimuli. Typically, the most prominent response for a sound in adults is the N1, peaking at about 100 ms from stimulus onsets, offsets, or changes in stimulus energy (Näätänen and Picton, 1987). In children under 10 years of age, the sound-elicited response is somewhat different, lacking the N1 but including prominent P1, N2, and sometimes N4 responses (Sharma et al., 1997; Čeponienė et al., 2002).

A change in a repetitive sound sequence or a stimulus violating a regularity obeyed by auditory stimulation elicits the mismatch negativity (MMN) response (Näätänen et al., 1978; Winkler, 2007). The MMN, elicited even when an individual is engaged in some activity unrelated to the sounds, reflects low-level sound-discrimination accuracy, as it is small in amplitude for sound differences that are difficult to discriminate and large for easily-perceivable differences (Baldeweg, 2006; Kujala and Näätänen, 2010). The main neural MMN sources are located in the temporal and frontal areas (Kujala et al., 2007). The MMN elicited by acoustic changes is predominant in the right hemisphere, whereas native-language speech sound changes and other linguistically relevant changes usually induce stronger left than right hemisphere responses (Kujala et al., 2007; Näätänen et al., 1997; Tervaniemi and Hugdahl, 2003).

A salient change in the sound stream also elicits the P3a component, which is a sign of an attention switch toward the sounds (Escera et al., 2000). This component follows the MMN and indicates an attention switch caused by the stimulus change in the unattended sound stream (Schröger, 1996). The P3a amplitude is larger for distracting sounds than for minor sound changes. Download English Version:

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