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Review

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

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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.

Contents

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).

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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.

The brain's neural activity can also be studied by recording magnetic fields created by electrical currents. The magnetoencephalography (MEG) has a similar temporal resolution as the electroencephalography (EEG), but its spatial resolution is higher than that of the EEG. However, unlike the EEG, it is fairly insensitive to radial and deep activity sources (Hämäläinen et al., 1993). The MEG is well suited for investigating the tangential temporal-lobe sources of the auditory P1 (P1m/M50, the magnetic counterpart of the P1), N1 (N1m/M100), and MMN (MMNm). However, the frontal MMNm component, for example, is not visible in the MEG (Kujala et al., 2007).

3. Sound encoding in ASD

Somewhat conflicting results have been found concerning sound encoding in school-aged children with autism or AS. Vowels and their non-speech counterparts have elicited diminished P1, N2, and N4 responses (Lepistö et al., 2005), whereas another study (Čeponienė et al., 2003) reported only a trend for a diminished P1 in children with autism.

A study on AS (Lepistö et al., 2006), using the same stimuli and paradigm as the Lepistö et al. (2005) study on autism, reported no significant group differences except for a diminished N4 for vowels in AS. Furthermore, with the same paradigm, no group differences were found in adults (Lepistö et al., 2007). In contrast, in children with AS, sinusoidal tones elicited a P1 with prolonged peak latencies and N2 and N4 with diminished amplitudes (Jansson-Verkasalo et al., 2005), and tones and syllables elicited reduced P1 amplitudes (Jansson-Verkasalo et al., 2003).

MEG studies (Oram Cardy et al., 2004, 2005, 2008; Roberts et al., 2010) with groups including both children and teenagers (7–18 years old), have reported no amplitude differences for the P1m or N1m between individuals with and without ASD. The latencies of these responses were either longer in children with autism (Oram Cardy et al., 2008; Roberts et al., 2010) or not different from those of control participants (Oram Cardy et al., 2004, 2005; Tecchio et al., 2003). Furthermore, in about 10-year-old children with ASD, N1m peak latencies were prolonged for tones ranging from 200 to 1000 Hz, with the effect being more pronounced for the right than the left hemisphere (Roberts et al., 2010). A study including children with autism or AS, specific language impairment, and normal development showed an association between the severity of language deficiency and the P1m latency prolongation in the right hemisphere (Oram Cardy et al., 2008).

Based on these studies, one may conclude that in some stimulus conditions or subgroups, stimulus encoding is impaired in ASD. The results suggested either normal-like or diminished amplitudes in studies using EEG and delayed latencies in studies using MEG, indicating insufficient or sluggish encoding of sounds.

4. Sound discrimination in ASD

Quite consistent with clinical observations (Bonnel et al., 2003, 2010; Mottron et al., 2006; O'Neill and Jones, 1997), studies using the MMN have suggested both hypo- and hypersensitive sensory processing in ASD. Likely sources of inconsistencies include the stimuli used and the clinical features, cognitive abilities, or level of language skills of the ASD group.

4.1. MMN amplitude and latency changes

MMNs have indicated a hyposensitive discrimination of sound duration and a hypersensitive discrimination of frequency in children with ASD. MMNs were recorded for changes of vowel identity, duration, and frequency, and their non-speech counterparts in children (aged 7–11 years) with autism with no mental retardation (performance IQ over 75; Lepistö et al., 2005) and in children with AS (Lepistö et al., 2006). Duration changes in vowels and their non-speech counterparts elicited diminished MMNs in children with autism (Lepistö et al., 2005). In children with AS, the duration changes of vowel counterparts elicited diminished MMNs, whereas group differences for vowel duration changes did not reach statistical significance (Lepistö et al., 2006). Consistent with this, a duration discrimination test given to children with AS revealed that their hit rate was lower and their reaction times tended to be longer than in their controls (Lepistö et al., 2006). The MMN amplitudes for frequency changes in both speech and non-speech stimuli were, in turn, enhanced in autism (Lepistö et al., 2005) and for non-speech stimuli in AS (Lepistö et al., 2006). Adults with AS, investigated using the same stimuli and paradigm, showed MMN enhancements for both frequency and duration changes (Lepistö et al., 2007), suggesting that hypersensitive sound discrimination might predominate the neural basis of auditory perception in adults with AS.

Several other studies also reported MMN results suggesting hypersensitive frequency processing in ASD. For example, shorter than normal MMN peak latencies were found for frequency changes of sinusoidal tones in 5–11-year-old children with autism (Gomot et al., 2002, 2011). Moreover, a shortened MMN latency followed by an enhanced P3a correlated with resistance to change, an important feature in this disorder (Gomot et al., 2011). This was evident in pronounced latency and amplitude abnormalities in a subgroup of autistic children who had the highest scores in reflecting intolerance to change on the Revised Behavior Summarized Evaluation Scale (Fig. 1).

However, a number of studies have reported hypo- rather than hypersensitive frequency discrimination in ASD, as reflected by diminished or delayed MMNs/MMNms for sound-frequency changes or changes of vowels (the formants of which are composed of sound frequencies). The MMNm for a tone frequency change (1000 vs. 1200 Hz) was absent in 8–32-year-old intellectually disabled and moderately or severely language-impaired autistic participants (Tecchio et al., 2003). For a similar frequency change, diminished MMNs were reported in 6–12-year-old children with autism matched with typically developing children by their non-verbal IQs (Dunn et al., 2008). However, these diminished MMNs might be explained by a noisy background due to the recordings being conducted while the video film had the sound on, since noise diminishes the MMN amplitude (Kujala and Brattico, 2009) and has a greater effect on the auditory perception of individuals with ASD than control participants (Alcantara et al., 2004).

However, delayed MMNs for tone-frequency changes (280 Hz vs. 320 Hz; Jansson-Verkasalo et al., 2005) and diminished MMNs for frequency changes of syllables (Kujala et al., 2010) were also found in about 11-year-old children with AS in silent EEG-recording conditions. Furthermore, delayed MMNm latencies were observed both for tone (300 vs. 700 Hz;) and vowel (/a/ vs. /u/) changes in 6–17-year-old intellectually non-disabled children with autism (Oram Cardy et al., 2004; Roberts et al., 2011). This delay was the most pronounced in children with language impairment (Roberts et al., 2011) (Fig. 2). In addition, in adults with autism with a performance IQ of 52–99, the MMNm latency for vowel changes was delayed in the left hemisphere (Kasai et al., 2004), and this delay correlated with the severity of autism.

The literature on the MMN in ASD seems to report variable and to some extent contradictory results. However, studies determining subgroups of ASD or carrying out correlation analyses between the MMN and behavioral test results indicate that the MMN seems to reflect the actual variation of the phenotypes in ASD (see, for example, Kasai et al., 2004; Gomot et al., 2011; Roberts et al., 2011).

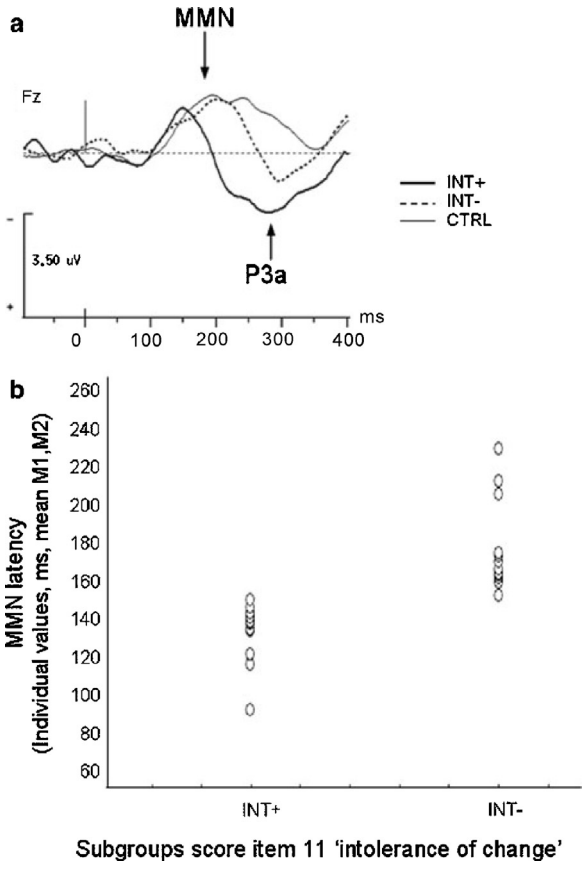

Fig. 1. Grand-mean MMN waveforms in the two subgroups with ASD and in control children. In children with ASD, the MMN latency was shorter and the P3a amplitude larger than in control children. These effects were more pronounced in the subgroup of children with ASD and a high intolerance to change (INT+) than in children with a low intolerance to change (INT−). The individual latency values are shown with circles for the two ASD groups in the bottom.
Adopted from Gomot et al. (2011).

4.2. Atypical MMN sources

The studies reviewed above also reported indications of atypical MMN generator sources in ASD. The MMN topography was generally more posterior in children with autism (Gomot et al., 2002; Jansson-Verkasalo et al., 2005; Lepistö et al., 2005, 2006) than in controls. This might be caused by either hypoactive frontal MMN generators or increased activity in posterior temporal-parietal areas. Gomot et al. (2002) studied children with autism using scalp-current density mapping, which improves the identification of local neural activities. These children had a posterior and more lateral scalp distribution than their controls for the frequency MMN, which could result from hypoactive frontal areas.

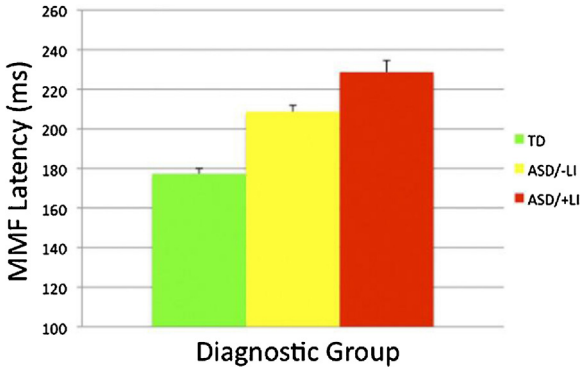

Fig. 2. MMNm latency collapsed across stimuli (vowels and tones) and cerebral hemispheres in children with and without autism. The latency was longer in children with autism than in their controls. The latencies were longest in children with autism having also a concurrent language impairment.
Adopted from Roberts et al. (2011).

These results are consistent with reports suggesting abnormal frontal-lobe metabolism (George et al., 1992; Zilbovicius et al., 1995) in autism. For example, in a longitudinal study measuring regional cerebral blood flow in children with autism, frontal hypoperfusion was found at the age of 3–4 years, which became normal-like by the age of 6–7 years. However, decreased blood flow has also been reported in adults with autism in the right, left, and midfrontal lobes with high-resolution brain single-photon emission tomography (George et al., 1992). The hypoperfusion of frontal areas might be associated with lower than normal neural activity, which could be associated with the diminished activation of frontal MMN generators.

Abnormally lateralized MMNs were also found in ASD. MMNs were generally diminished over the left and tended to be enhanced over the right hemisphere for speech and non-speech sounds in children with AS, whereas control children had a symmetrical MMN distribution (Lepistö et al., 2006). In adults with AS, in turn, the amplitudes were generally larger over the right and midline scalp areas than over the left hemisphere, whereas controls had a more symmetrical distribution (Lepistö et al., 2007). Consistent with these results suggesting abnormal left-hemisphere functions, left-hemisphere MMNm latencies were found to be prolonged in autism (Kasai et al., 2004).

However, MMN responses for prosodic changes in words were diminished over the right hemisphere in adults with AS, whereas the scalp distribution in control adults was relatively bilateral (Kujala et al., 2005). Consistent with this, another study (Korpilahti et al., 2007) reported right-hemisphere preponderant MMNs for prosodic changes in word stimuli in healthy children but symmetrically distributed MMNs in children with AS. Thus, it is evident that the neural assemblies processing auditory input are aberrantly distributed in ASD. The results suggest both left- and right-hemisphere dysfunctions as well as abnormal activity distribution in the front–back axis.

5. Extracting the speech code in ASD

In addition to the generally abnormal pattern of auditory processing in ASD, individuals in this spectrum have various problems related to speech. It is remarkable how our speech system can rapidly and effortlessly analyze the speech of a person even when meeting for the first time, despite the enormous inter-individual variation in the way we speak or challenging conditions (e.g. noise). This is based on our speech system's ability to extract invariant features relevant for understanding speech (Bishop, 1997). This task may be challenging for individuals with autism, since they seem to have a detail-oriented style of processing information, suggested to result in enhanced low-level perceptual processing (Happé and Frith, 2006; Mottron et al., 2006). This suggestion is mainly based on observations made in the visual modality, indicating superior visual search, disembedding skills, and discrimination learning in individuals with autism (Dakin and Frith, 2005; Happé and Frith, 2006). The enhanced sensitivity to some acoustic features, observed in autism (Bonnel et al., 2003; Heaton, 2003, 2005; Mottron et al., 2000; O'Riordan and Passetti, 2006) and reflected in the neurophysiological responses of individuals with autism (Gomot et al., 2002, 2011; Lepistö et al., 2005, 2006) may be associated with this detail-oriented processing.

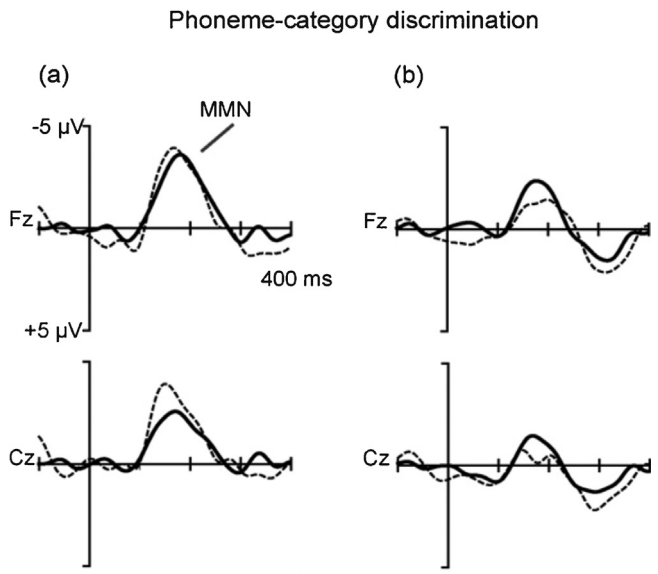

Fig. 3. Acoustic variance impairs the discrimination of phonetic categories in children with autism. The MMNs of these children (dashed line) and control children (continuous line) are shown in two conditions (a and b) and at a frontal (Fz) and central (Cz) scalp electrode. On the left (a), the MMNs were recorded for changes of vowels occurring in an otherwise homogenous stream of a repeated vowel. Consistent with previous studies, the children with autism have enhanced MMNs (Cz) in this condition. However, when the stream of vowels includes frequency variation (b), the MMN becomes smaller in these children than in their controls. This suggests that physical variation, normally present in speech, impairs categorical phoneme processing in children with autism.
Adapted from Lepistö et al. (2008).

The detail-oriented processing style and enhanced low-level discrimination abilities may cause auditory processing to be too focused on the intrinsic acoustical differences between speech sounds belonging to the same phoneme category (O'Riordan and Passetti, 2006). This may lead to difficulties in extracting relevant invariant phonetic features and forming proper phonetic categories, which may impair language development in autism.

The assumption of deficient abilities in phonetic invariance detection was tested by Lepistö et al. (2008). In their *varying-frequency condition*, sequences of a vowel with a continuously varying frequency were presented, along with occasional vowel-identity changes (e.g. from the repetitive /a/ to /u/). In the *constant-frequency condition*, the vowels were the same as in the varying-frequency condition, but the frequency of the stimuli was constant. In this condition, a centro-parietally enhanced MMN amplitude was found in children with autism (Fig. 3), consistent with the suggested superior low-level perceptual skills in autism (Mottron et al., 2006). However, in the *varying- frequency condition*, the opposite data pattern was observed, with the children with autism having a diminished response amplitude. This effect was specific to speech, since in the control conditions, no such effect was observed in MMNs for frequency changes when the identity of the repetitive vowel varied (i.e. the vowel category changed every time) vs. did not vary.

A comparison of MMNs between the conditions with stimulus variation revealed a very different processing pattern in the two groups. The children with autism had larger MMNs for frequency changes of vowels whose identity continuously varied than for vowel-identity changes when the same vowel varied in frequency. The results of the control children were exactly the opposite, indicating that the neural apparatus of children with typical development is more adept in extracting linguistically relevant than irrelevant features from sound streams. These results suggest a profoundly different stimulus-processing manner in autism and typical development, and indicate that even the low-level pre-attentive processing of speech is aberrant in autism.

6. Orienting to speech in ASD

While early feature analysis, which can be probed with the MMN, occurs even without consciousness, attentive processing is needed to understand the content of speech. Also in attention to speech and other social stimuli, individuals with ASD display an atypical data pattern. This is evident even in 32–52-month-old infants (Kuhl et al., 2005). When an oddball paradigm with a repetitive /wa/ and deviant /ba/ syllable was used for testing, a smaller MMN was found in infants with than without ASD (Kuhl et al., 2005). Furthermore, the infants preferring non-speech sounds, as judged from head-turning responses to speech and non-speech stimuli, had no MMNs whereas the MMNs of infants preferring speech did not differ from those of the controls.

This inattentiveness to speech is also evident in neural responses reflecting the orientation toward stimuli, such as the P3a, indicating that a stimulus has opened a channel to consciousness (Escera et al., 2000). Remarkable differences are present in this channel between individuals with ASD and typical development. Čeponienė et al. (2003), using vowels, their non-speech counterparts, and simple sinusoidal tones, recorded P3as for pitch changes of these stimuli in children with autism watching a video film. They found that the P3a was smaller in these children than in typically developing children for changes of vowels but not for changes of the two non-speech stimulus types (Fig. 4). Corroborating results were found by Lepistö et al. (2005) in children with autism and a similar tendency by Lepistö et al. (2006) in children with AS. In adults with AS, the P3a was larger to non-speech than speech changes, whereas no such effect was found in their controls (Lepistö et al., 2007). These results suggest that the central auditory system of individuals with ASD does not orient to speech sounds normally. However, in the studies reviewed above, the MMN amplitude for the speech sound changes was not diminished, suggesting that the result cannot be explained by a discrimination deficit at a lower level of processing.

Whitehouse and Bishop (2008) studied orienting to change in autism using speech and non-speech sounds in the same stimulus sequences. They found reduced P3as in autism for rare non-speech sounds when they were embedded in speech sound streams but not for rare speech sounds occurring among non-speech sounds, in which case the result was, in fact, the opposite. These results suggest that individuals with autism orient abnormally to even rare non-speech sounds if they occur among speech sounds. Thus, the nature of the sound stream affects the responsiveness to changes in autism. This suggests that auditory streams primarily composed of speech sounds are not processed as thoroughly as those of non-speech sounds in the auditory system in children with autism. This is supported by their diminished obligatory responses to repetitive speech but not to non-speech sounds, found by Whitehouse and Bishop (2008), indicating insufficient encoding of speech.

Whitehouse and Bishop (2008) also determined how voluntary attention modulates change-related responses in autism. They found that while smaller P3as were elicited by changes among unattended speech than by non-speech streams, these diminished P3a amplitudes were increased to the normal level when children with autism attended to auditory stimuli. However, the sample included high-functioning children with autism with conversational speech skills. Furthermore, only those children were included who could correctly detect the targets of the attentive task with an 80% accuracy, which led to the exclusion of almost half of the children from the study. Thus, their result concerns a subgroup

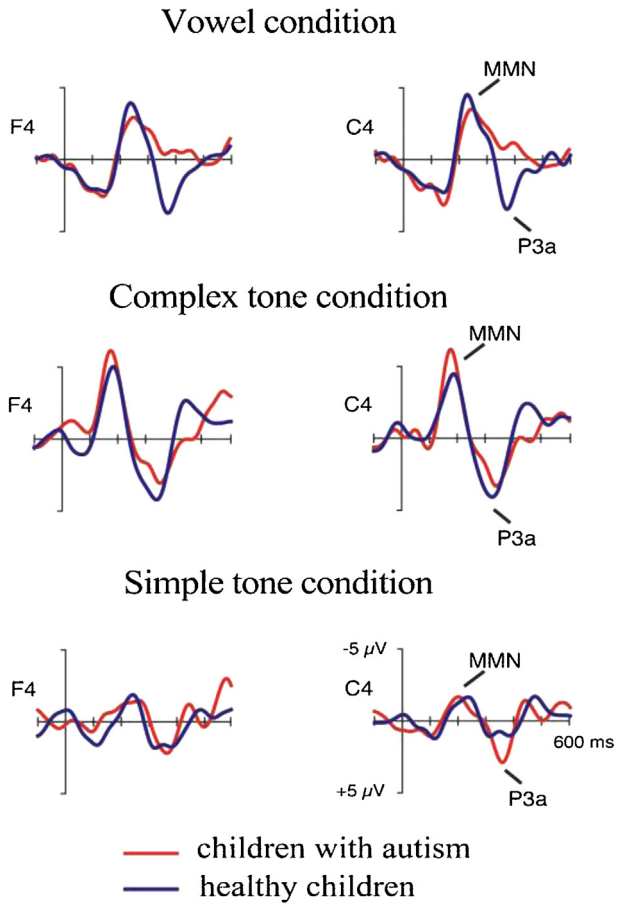

Fig. 4. Speech does not attract attention of the children with autism similarly to typically-developing children. Frequency changes of vowels and complex tones elicit robust P3a responses in typically developing children (blue line). Whereas children with autism (red line) have normal P3as for frequency changes of complex tones (bottom), their P3a is completely absent for frequency changes of vowels (top). This indicates impaired attention shifting to speech in autism.
Adopted from Čeponienė et al. (2003).

of children with autism having normal-like attentive abilities and cannot be generalized to the population with autism.

Taken together, the studies examining the orientation toward speech vs. non-speech sounds have shown atypical response patterns both in individuals with autism and AS. Their attention switches to speech-sound changes in speech streams are diminished (Čeponienė et al., 2003; Lepistö et al., 2005, 2006), whereas acoustic changes in non-speech sound streams cause normal-like or enhanced responses (e.g. Čeponienė et al., 2003; Lepistö et al., 2005, 2006, 2007; Gomot et al., 2011). When speech and non-speech stimuli are mixed, the most frequent stimulus type determines the reactivity to the stimuli, at least in some subgroups of ASD (Whitehouse and Bishop, 2008). This reactivity indicates that the channel for input that includes predominantly non-speech stimuli seems to be more open than that for speech input.

7. Conclusions

Neurophysiological approaches have shown that the pattern of auditory processing in ASD, involving both hypo- and hypersensitivity, originates from the early stages of neural processing. They have also shed novel light on the nature of speech-processing difficulties in these disorders, indicating defects in the fundamental low-level speech-sound analysis needed in perceiving speech fluently and orienting to speech.

The neural responses which reflect stimulus encoding (e.g. P1/P1m, N1/N1m) were found to be in ASD either diminished, delayed, or not significantly different from those of control participants. The abnormal sound-elicited responses do not seem to be related to stimulus material, since they were reported both for speech and non-speech sounds. Thus, auditory encoding is generally either insufficient or normal but not hypersensitive in ASD.

Atypical cortical sound discrimination in ASD was evident in all MMN parameters: latency, amplitude, and scalp distribution. The enhanced and faster MMNs in ASD (Gomot et al., 2002, 2011; Lepistö et al., 2005, 2006, 2008) are consistent with results showing superior performance in ASD in pitch discrimination and memory tests (Heaton et al., 2008). These results are also compatible with the theory of enhanced low-level perceptual processing in this disorder (Happé and Frith, 2006; Mottron et al., 2006). This type of processing is associated with detail-oriented perception, which may lead to difficulty in perceiving wholes without fully processing their fundamental parts. In practice, this may be reflected in superior perceptual skills, such as musical aptitude or absolute pitch processing. This processing style might, however, be inappropriate when one has to extract invariant information, for instance, from speech. This was supported by a study showing that in children with autism, the atypically large MMNs for vowel changes in repetitive stimulus sequences became suppressed when pitch variance was introduced to the sequences (Lepistö et al., 2008). In natural conditions, this might lead to difficulties in extracting the phonetic information relevant for effectively understanding speech.

The literature reporting hypo- vs. hypersensitive neural processing in ASD includes rather many conflicting results, for example, enhanced (e.g. Lepistö et al., 2005) vs. diminished MMN amplitudes (e.g. Dunn et al., 2008) and abnormally short (e.g. Gomot et al., 2011) vs. long MMN peak latencies (e.g. Roberts et al., 2011). This is likely to result from the heterogeneity of the ASD population, which is evident in the literature reporting results obtained with tests on perception, memory, and cognition (Heaton et al., 2008; Salmond et al., 2007). For example, enhanced frequency discrimination abilities were reported in 20% of individuals in a group of 72 adolescents with ASD (Jones et al., 2009). These notions are supported by findings suggesting that individuals with ASD having splinter skills may represent a distinct genetic group (Nurmi et al., 2003).

Studies testing autism traits and cognitive and language abilities along with MMN recordings have shown that an abnormal MMN pattern is associated with test measures reflecting the phenotype. For example, a diminished MMN for syllable changes was found in infants with autism having a preference for non-speech over speech stimuli but not in infants without such a preference (Kuhl et al., 2005). Faster MMN latencies, in turn, were found in a subgroup of children with autism with very high resistance to change (Gomot et al., 2002). In contrast, delayed MMN peak latencies were found in subgroups of ASD having the severest type of autism (Kasai et al., 2004) or concomitant language impairment (e.g. Roberts et al., 2011). These results are consistent with the notion on the relationship between cognitive abilities and the MMNs (Näätänen et al., 2011; Näätänen and Kujala, 2011). For example, children with severe autism who also have prolonged MMNms (Kasai et al., 2004), are usually intellectually disabled. In intellectual disability, in turn, MMNs have abnormally small amplitudes and long latencies (Ikeda et al., 2000). Also, the delayed MMNs in subgroups of ASD with the most severe language problems (Roberts et al., 2011) are consistent with studies on the MMN in groups with language deficits, that systematically report dampened or sluggish MMN (Kujala, 2007; Näätänen and Kujala, 2011).

Attention-switching functions, indicated by P3a following the MMN, are also aberrant in ASD. This was evident in the P3a elicited by speech and non-speech sound changes. Whereas the MMN

seems to be more affected by the type of sound change (e.g. duration or frequency) than by sound quality ("speechness") (Lepistö et al., 2005, 2006), attention-switching is affected by sound quality. P3as are absent or diminished for changes in speech-sound sequences but normal-like for those in non-speech sound sequences in ASD (Čeponienė et al., 2003; Lepistö et al., 2005, 2006). This tendency, if it exists in early childhood, may even hamper the development of communication and language skills. Typically developing infants have a preference for speech sounds, granting them a special status in auditory perception (Jusczyk and Bertoncini, 1988). This facilitates language learning by improving the selection of acoustic signals relevant for communication. If this bias toward speech is weak or absent, then the child is not efficiently processing speech signals, which can lead to underdeveloped neural speech representations and analysis. In fact, the severity of neural speech-processing symptoms and attention to social stimuli were shown to be connected in children with autism, and this connection is evident even in infancy (Kuhl et al., 2005).

Neurophysiological approaches have given important insight into to the neural basis of perception in ASD. Neurophysiological measures combined with careful clinical assessments have helped in understanding the phenotype variety in this spectrum. Involuntarily elicited neural responses, enabling the study of the neural basis of discrimination accuracy and attention shifting (Kujala et al., 2007), are invaluable for investigating challenging groups such as children with autism. Neurophysiological measures should be more extensively utilized, for instance, to determine the early stages of the development of children with ASD and the effects of intervention on the perceptual neural processes in this spectrum.

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