

The neurophysiological correlates of face processing in adults and children with Asperger's syndrome

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Abstract

Past research has found evidence for face and emotional expression processing differences between individuals with Asperger's syndrome (AS) and neurotypical (NT) controls at both the neurological and behavioural levels. The aim of the present study was to examine the neurophysiological basis of emotional expression processing in children and adults with AS relative to age- and gender-matched NT controls. High-density event-related potentials were recorded during explicit processing of happy, sad, angry, scared, and neutral faces. Adults with AS were found to exhibit delayed P1 and N170 latencies and smaller N170 amplitudes in comparison to control subjects for all expressions. This may reflect impaired holistic and configural processing of faces in AS adults. However, these differences were not observed between AS and control children. This may result from incomplete development of the neuronal generators of these ERP components and/or early intervention.

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1. Introduction

For many of us, the ability to empathize and interact with others is an intuitive process and requires limited effort. However, for individuals with Asperger's syndrome (AS), relating to and understanding other human beings is often difficult. Asperger's syndrome is a neurodevelopmental disorder, mainly affecting non-verbal communication and sensory processing. People with AS also have restricted interests, exhibit repetitive and stereotyped behavioural responses, and enjoy routine. The symptomatology of AS is similar to autism, but without the associated language or cognitive delay (Attwood, 1998). For this reason, autism and AS are often classified as autistic spectrum disorders (ASDs), with AS at the higher end of the spectrum (Macintosh & Dissanayake, 2004).

However, although individuals with AS often want to interact with others, they experience great difficulty (Attwood, 1998; Birch, 2003; Miller, 2003). Two main theories for these interaction deficits are apparent in the autism literature. The first theory proposes that social deficits in ASD result from a general impairment in "theory of mind" (ToM), the ability to attribute thoughts and intentions to others. Past research has found evidence for impairment on first and second-order TOM tasks in children with autism, and on more complex ToM tasks in both children and adults with AS (Baron-Cohen, 1989; Baron-Cohen, Leslie, & Frith, 1985; Baron-Cohen, O'Riordan, Stone, Jones, & Plaisted, 1999; Happe, 1994). Similarly, deficits in joint attention and imitation—potential precursors in ToM development, have been documented in individuals with ASD (Charman, 2003; Williams, Whiten, Suddendorf, & Perrett, 2001).

The second theory is that social interaction difficulties in ASD may arise from a general deficit in central

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coherence, the ability to integrate local details into a coherent or ‘global’ whole (see Frith & Happe, 1994 for a review). This theory explains the processing style common to ASD, which is biased towards processing details over general meaning (Vermeulen, 2001). Furthermore, the use of local processing strategies to comprehend social interactions, which involve the simultaneous integration of visual, auditory, tactile, and even olfactory information, would leave an individual with AS at a serious disadvantage.

Past research has found evidence for face processing differences in individuals with AS relative to NTs. For example, whereas faces are mainly processed holistically (as perceptual wholes) in NTs, individuals on the autistic spectrum appear to favour a more feature-based, “analytical” approach. Evidence for this has been found using inverted faces, which are thought to be processed using predominantly analytical strategies (Tanaka & Farah, 1993; Yin, 1969). Inverting faces impairs face recognition in NTs through disruption of both configural (the ability to process spatial relationships between facial features) and holistic processing (Itier & Taylor, 2002). However, this procedure does not always impair face recognition in individuals with ASD who tend to show similar performance for recognition of upright and inverted faces (Hobson, Ouston, & Lee, 1988; Langdell, 1978). These findings suggest that the ability to process faces holistically and/or process facial configurations may be impaired in ASD, or that individuals with ASD prefer to use other face processing strategies (i.e., an analytical strategy). The latter explanation is probably has more empirical support, as recent research suggests that configural processing can occur in AS when face recognition is dependent on the mouth (Joseph & Tanaka, 2003).

Other studies have found evidence for abnormal processing of features in ASD. For example, past research has shown NTs fixate more on the eye than mouth region of faces while individuals with AS focus less on the eyes and instead devote greater attention to the mouth (Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Klin, Jones, Schultz, & Volkmar, 2003; Joseph & Tanaka, 2003). Furthermore, adults with AS have difficulty identifying complex emotional expressions from the eye region (Baron-Cohen, Wheelwright, & Jolliffe, 1997).

Evidence for face processing differences between individuals with ASD and NT controls are also found at a neurological level. Several studies have shown individuals with ASD to exhibit hypoactivation of the right fusiform gyrus during face processing, a region activated by extremely familiar stimuli (Hubl et al., 2003; Pierce, Muller, Ambrose, Allen, & Courchesne, 2001; Schultz et al., 2000). Decreased activity has also been observed in the superior temporal sulcus (STS), involved in the detection of biological motion such as eye gaze (Pierce

et al., 2001; Puce, Allison, Bentin, Gore, & McCarthy, 1998). Furthermore, increased activity in the inferior temporal and lateral occipital regions has been observed in individuals with ASD in response to faces. More importantly, these regions exhibit greater activation to objects in NT control subjects. Together, these findings provide further evidence that analytical processing strategies may be used to process faces in ASD (Hubl et al., 2003; Schultz et al., 2000).

Differences are also observed during emotional face processing in individuals with ASD relative to NT controls. For example, adults with ASD have been found to exhibit decreased activation of the left inferior frontal gyrus (IFG) and insula during identification of complex emotions from the eyes alone, and in the left middle frontal gyrus during recognition of fearful faces (Baron-Cohen, Ring, et al., 1999; Ogai et al., 2003). Furthermore, decreased amygdala activation has been observed in response to both emotional and neutral faces in ASD adults (Critchley et al., 2000; Pierce et al., 2001). Interestingly Carr, Iacoboni, Dubeau, Mazziotta, and Lenzi (2003) postulate that the STS, IFG, insula, and amygdala may incorporate a circuit involved in empathy, which may explain the ToM difficulties common to ASD.

A large number of studies have investigated the neurophysiological basis of face processing in NTs using event-related potentials (ERPs). The initial categorization of a stimulus as a face has been shown to occur as early as 100ms (the P1 component). A few (but not all) studies have found evidence that P1 may also reflect an early face processing stage. For example, some studies have observed P1 to be smaller and/or earlier to upright relative to inverted faces and objects (Taylor, Edmonds, McCarthy, & Allison, 2001; Itier & Taylor, 2002, 2004).

However, in contrast to P1, a negative deflection occurring around 170ms (between approximately 140 and 200ms) is consistently activated to faces, and is largest in amplitude over posterior temporal electrodes (Taylor, Batty, & Itier, 2004). Termed the N170, this component has been shown to be larger to human faces than to objects (furniture, flowers, etc.), animal faces and human hands (Bentin, Allison, Puce, Perez, & McCarthy, 1996). Several research groups have implicated the N170 component in configural and/or holistic processing. For example, N170 amplitude is delayed and/or larger in response to inverted relative to upright faces (Itier & Taylor, 2004; McPartland, Dawson, Webb, Panagiotides, & Carver, 2004; Itier & Taylor, 2004). Finally, some studies have observed that the P2 component are also sensitive to inverted faces, although this component has been examined less extensively than the N170 (Itier & Taylor, 2002; Rebai, Poiroux, Bernard, & Lalonde, 2001).

Developmental studies have found the N290 and P400 components in 12-month-old infants are

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