

PAPER

Young children with autism show atypical brain responses to fearful versus neutral facial expressions of emotion

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Abstract

Evidence suggests that autism is associated with impaired emotion perception, but it is unknown how early such impairments are evident. Furthermore, most studies that have assessed emotion perception in children with autism have required verbal responses, making results difficult to interpret. This study utilized high-density event-related potentials (ERPs) to investigate whether 3–4-year-old children with autism spectrum disorder (ASD) show differential brain activity to fear versus neutral facial expressions. It has been shown that normal infants as young as 7 months of age show differential brain responses to faces expressing different emotions. ERPs were recorded while children passively viewed photos of an unfamiliar woman posing a neutral and a prototypic fear expression. The sample consisted of 29 3–4-year-old children with ASD and 22 chronological age-matched children with typical development. Typically developing children exhibited a larger early negative component (N300) to the fear than to the neutral face. In contrast, children with ASD did not show the difference in amplitude of this early ERP component to the fear versus neutral face. For a later component, typically developing children exhibited a larger negative slow wave (NSW) to the fear than to the neutral face, whereas children with autism did not show a differential NSW to the two stimuli. In children with ASD, faster speed of early processing (i.e. N300 latency) of the fear face was associated with better performance on tasks assessing social attention (social orienting, joint attention and attention to distress). These data suggest that children with ASD, as young as 3 years of age, show a disordered pattern of neural responses to emotional stimuli.

Introduction

Autism is a disorder characterized by specific impairments in processing social and emotional information (e.g. Baron-Cohen, Tager-Flusberg & Cohen, 1993; Dawson, Meltzoff, Osterling, Rinaldi & Brown, 1998b; Teunisse & DeGelder, 1994), with early-appearing impairments evident in social orienting (Dawson *et al.*, 1998b; Dawson, Toth, Abbott, Osterling, Munson, Estes & Liaw, 2002b), joint attention (Mundy, Sigman, Ungerer & Sherman, 1986; Dawson, Meltzoff, Osterling & Rinaldi, 1998a), responses to the emotional displays of others (Sigman, Kasari, Kwon & Yirmiya, 1992) and face recognition (Dawson, Carver, Meltzoff, Panagiotides, McPartland & Webb, 2002a; Klin, Sparrow, de Bilt, Cicchetti, Cohen & Volkmar, 1999). These social impairments, some of which are apparent by 1 year of age (Osterling & Dawson,

1994; Osterling, Dawson & Munson, 2002), suggest that autism is related to early dysfunction of brain circuitry involved in social cognition (Baron-Cohen, Ring, Wheelwright, Bullmore, Brammer, Simmons & Williams, 1999; Baron-Cohen, Ring, Bullmore, Wheelwright, Ashwin & Williams, 2000; Dawson, 1996).

In this paper, we explore further the nature of early impairments in social cognition in autism. We were interested in assessing very young children's electrical brain responses to an emotional facial expression. Processing the emotional expressions displayed by another individual involves attention to and perception of the face of that individual, as well as perception of the specific emotion and context of the event. By 3 to 4 years of age, children with ASD are already showing impairments in face processing (Dawson *et al.*, 2002a; Webb, Dawson & Shook, 2002). It is currently unknown whether emotion processing,

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specifically in the processing of a primary emotion (e.g. fear), is also disrupted at this early age, or whether such an impairment, if present, is related to impairments in other domains, such as social orienting, joint attention and attention to another's distress. To address these questions, this study utilized event-related potentials (ERPs) to examine early emotional perception in children with autism, and further examined whether individual differences in ERPs to emotional stimuli correlated with performance on tasks measuring social attention. We will first briefly review what is known about the normal development of neural substrates involved in face processing, and their disruption in autism. Then, we will review what is known about the development of neural substrates involved in emotional processing, and their potential role in autism.

Face processing and its neural substrates

In typically developing infants, the face holds particular significance and provides nonverbal information important for communication and survival (Darwin, 1872/1965). Visual preference for faces, and specifically preference for the mother's face, has been shown to be present within the first days of life (e.g. Goren, Sarty & Wu, 1975; Valenza, Simion, Cassia & Umiltá, 1996). Between the ages of 3 and 7 months, infants become better at distinguishing highly familiar faces from unfamiliar faces (e.g. de Haan & Nelson, 1997, 1999; Maurer & Salapatek, 1976; Pascalis, de Haan, Nelson & de Schonen, 1998), can categorize faces by gender (Cohen & Strauss, 1979; Yamaguchi, 2000), demonstrate the face inversion effect (Fagan, 1972; Webb & Nelson, 2001), and show a right hemisphere bias for processing faces (de Haan & Nelson, 1997; de Schonen, Gil de Diaz & Mathivet, 1986; de Schonen & Mathivet, 1989).

The system that subserves face recognition is thought to primarily involve the inferior and medial temporal cortex (see Nelson, 2001, for review). This includes, but is not limited to, the fusiform gyrus, amygdala and superior temporal sulcus; the specific involvement of each of these substrates may depend on the information being abstracted from the face. Functional magnetic resonance imaging (fMRI) studies suggest that the fusiform gyrus is more active during viewing of faces compared with non-face objects, scrambled faces, or inverted faces and is typically more active on the right than left (e.g. Gauthier, Tarr, Anderson, Skudlarski & Gore, 1999; Kanwisher, McDermott & Chun, 1997; McCarthy, Puce, Gore & Allison, 1997).

While the fusiform gyrus is preferentially active for encoding of faces, the amygdala and superior temporal

sulcus may be responsible for encoding social information derived from faces. The amygdala is thought to assign affective significance to faces, which would likely affect both attention and mnemonic aspects of face processing (Morris, Friston, Buchel, Frith, Young, Calder & Dolan, 1998). Functional MRI studies have demonstrated that regions surrounding the superior temporal sulcus (STS) are involved in processing face movements and in perception of gaze direction. The STS region is activated by a moving mouth and eyes (Puce, Allison, Bentin, Gore & McCarthy, 1998), dynamic changes in face identity (i.e. a face 'morphing' into another face) and fast dynamic changes in gaze direction (Hoffman, Phillips & Haxby, 2001). It has been suggested that this region, along with amygdala and orbitofrontal cortex, may form part of a neural system involved in social attention, social bonding, and use of facial gestures (Brothers & Ring, 1993; Kling & Steklis, 1976; Perrett, Harries, Mistlin & Hietanen, 1990; Perrett, Hietanen, Oram & Benson, 1992).

Face processing in autism

As discussed above, the neural systems that mediate face processing and recognition rapidly develop during the first 6 months of life. This precocious ability offers the possibility that face recognition impairments may be one of the earliest indicators of abnormal brain development in autism. While little is known about early face processing in children with autism, Osterling and Dawson (1994) found that attention to faces at 1 year of age was the single best discriminator between infants who were later diagnosed with autism and typically developing infants. In a recent study (Dawson *et al.*, 2002a), 3–4-year-old children with autism failed to show differential ERPs to their mother's face versus an unfamiliar face but did show differential ERPs to a favorite versus an unfamiliar toy. Typically developing children and mental age-matched children with idiopathic developmental delay showed differential ERPs to the unfamiliar face as compared to mother's face, and to a favorite toy as compared to an unfamiliar object. This suggests that very young children with autism have an impaired ability to process faces.

Emotional processing and its neural substrates

The face is a primary method of identification and association, with facial expressions serving as a means of communication. Children as young as 6 months of age are able to differentiate facial expressions of happy and

sad in a familiar person (Cohn, Campbell, Matias & Hopkins, 1990). By 12 months, typical children are able to use their mother's emotional expressions to guide their actions. During toddlerhood, typical children can recognize the primary emotions from facial and vocal expressions (Denham & Couchoud, 1990) and are beginning to label emotion states (Izard, 1971).

Differential event-related potentials (ERPs) to distinct facial expressions of emotion also have been shown in infants as young as 7 months of age (Nelson & de Haan, 1996). In the Nelson and de Haan study with infants, significant ERP differences in terms of amplitude were found in an early positive component (peak between 240 and 260 ms at midline leads), a midlatency negative component (peak between 530 and 560 ms) and a late positive component in response to a happy facial expression versus a fearful expression but not to angry versus fear expression. For children, four components have been shown to be influenced by the emotion of the stimulus: N170, P280, N400 and P700. At posterior-temporal leads, the N170 (peaking at 200 msec) was found to be faster to fear than angry and happy faces (de Haan, Nelson, Gunnar & Tout, 1998). The N170 in adults is thought to be a 'face' specific component, and is of greater amplitude and of shorter latency to face stimuli than to other types of stimuli (e.g. Bentin, Allison, Puce, Perez & McCarthy, 1996). However, work with adults has not found this component to differ in terms of amplitude or latency to fear versus neutral facial expressions (Eimer & Holmes, 2002). In two reports, the N400 at anterior leads was larger to angry expressions than to a happy or fear expression (Nelson & Nugent, 1990; de Haan *et al.*, 1998). The N400 is commonly found in children's visual ERPs and was interpreted as reflecting increased attention allocation to the negative emotional stimulus (de Haan *et al.*, 1998). The child P700 is thought to be analogous to the adult P300 and was found to be greater in amplitude to angry than neutral faces in typical children (Kestenbaum & Nelson, 1992) as well as maltreated children (Pollak, Cicchetti, Klorman & Brumaghim, 1997). In contrast to the work with children, in adults, fearful expressions elicit a greater fronto-central positive component at approximately 120 ms than do neutral faces (Eimer & Holmes, 2002). Combined, these reports suggest that emotion perception and recognition may influence the ERP wave at multiple time periods.

In regard to the underlying anatomical circuitry, studies of lesioned animals and brain damaged individuals suggest that the medial temporal lobe, especially the amygdala, is critical for social perception (Baron-Cohen *et al.*, 1999, 2000, Dawson, 1996; Bachevalier, 2000), the interpretation of emotional and social signals (Leonard, Rolls, Wilson & Baylis, 1985; Nakamura, Mikami &

Kubota, 1992; Perrett, Smith, Mistlin, Chitty, Head, Potter, Broennimann, Milner & Jeeves, 1985; Perrett *et al.*, 1992; Scott, Young, Calder, Hellowell, Aggleton & Johnson, 1997), recognition of faces and facial expressions (Aggleton, 1992; Jacobson, 1986), the ability to make accurate judgments about emotion expression (Breiter, Setoff, Whalen, Kennedy, Rauch, Buckner, Strauss, Hyman & Rosen, 1996; Adolphs, Tranel, Damasio & Damasio, 1994; Adolphs, Tranel, Hamann, Young, Calder, Phelps, Anderson, Lee & Damasio, 1999; Young, Hellowell, Van De Wal & Johnson, 1996), recognition of the affective significance of stimuli (Lane, Reiman, Bradley, Lang, Ahern, Davidson & Schwartz, 1997; LeDoux, 1987), perception of gaze direction (Brothers, Ring & Kling, 1990) and the use of facial gestures (Brothers & Ring, 1993; Kling & Steklis, 1976; Perrett *et al.*, 1990, 1992, in non-human primates). The amygdala is a collection of nuclei that receive sensory input from the somatosensory, visual, auditory and visceral systems. While there is still a great deal of debate about the amygdala's role in positive emotions (e.g. Adolphs *et al.*, 1999), a large body of research has demonstrated that the amygdala plays a central role in the perception of and recognition of fear by human adults (e.g. Adolphs *et al.*, 1994, 1999; Scott *et al.*, 1997) and children (Thomas, Drevets, Dahl, Ryan, Birmaher, Eccard, Axelson, Whalen & Casey, 2001a; Thomas, Drevets, Whalen, Eccard, Dahl, Ryan & Casey, 2001b) as well as in the expression of fear in animals (see LeDoux, 2000, for review). The amygdala becomes active when adults view faces with a fear expression (e.g. Breiter *et al.*, 1996) and when children view faces with either a neutral or fear expression (Thomas *et al.*, 2001a, 2001b). In adults, degree of amygdala activation is correlated with the intensity of the fear expression (Morris, Frith, Perrett, Rowland, Young, Calder & Dolan, 1996; Morris *et al.*, 1998; Phillips, Young, Senior, Brammer, Andrews, Calder, Bullmore, Perrett, Rowland, Williams, Gray & David, 1997). These responses are more likely to involve activity in the left amygdala (Breiter *et al.*, 1996; Morris *et al.*, 1996), but both the right and left amygdala habituate to fear over time (Breiter *et al.*, 1996).

Patients who have had their amygdala removed or have amygdala lesions show impaired ability to recognize and match certain emotions, identify eye gaze direction, imagine emotional expressions and interpret social signals from the face (e.g. Young *et al.*, 1996). These impairments are also present when the emotion is verbally expressed instead of visually expressed (Scott *et al.*, 1997), and are greater to fear than other emotions (Morris *et al.*, 1996; Scott *et al.*, 1997; Phillips *et al.*, 1997; Adolphs *et al.*, 1994; Breiter *et al.*, 1996; Ketter, Andreason, George, Lee, Gill, Parekh, Willis, Herscovitch & Post,

1996). Adolphs *et al.* (1994) found that a patient with bilateral destruction of the amygdala (Urbach-Weithe disease) had intact identity recognition but rated fear, anger and surprise as less intense and was unable to perceive similarities between emotion expressions.

Emotion processing in autism

Several theorists have argued that the ability to use or understand facial information is a core deficit in autism (Baron-Cohen, 1993; Dawson, Webb, Schellenberg, Aylward., Richards, Dager & Friedman, 2002c; Frith, 1989; Hobson, Ouston & Lee, 1989). Given that emotion is often displayed in the face, separating a deficit in facial processing from a deficit in understanding and recognizing facial expression is difficult. However, studies have shown intact facial expression recognition despite severe deficits in facial recognition in patients with prosopagnosia (Shuttleworth, Syring & Allen, 1982; Bruyer, Laterre, Seron, Feyereisen, Strypstein, Pierrard & Rectem, 1983), as well as the reverse pattern of a deficit in expression recognition but intact facial recognition in patients with amygdectomy (Adolphs *et al.*, 1994). This suggests that the two abilities can be dissociated at the neural and theoretical level (Bruce & Young, 1986).

Evidence suggests that individuals with autism are less proficient at matching emotional expressions (Celani, Battacchi & Arcidiacono, 1999; Hobson, Ouston & Lee, 1988a, 1988b; Hobson *et al.*, 1989; Loveland, Tunali Kotoski, Chen, Brelsford, Ortegon & Pearson 1995). Their ability to match expressions has been correlated with verbal ability, making these findings difficult to interpret (Buitelaar, van der Wees, Swaab-Barneveld & van der Gaag, 1999; Happe, 1994; Hobson *et al.*, 1989; Ozonoff, Pennington & Rogers, 1990). Loveland, Tunali Kotoski, Chen, Ortegon, Pearson, Brelsford and Gibbs (1997) found no differences between controls and children with autism on emotion identification tasks; there were differences between high and low functioning autism groups. High functioning individuals with autism could use affective information from both verbal and nonverbal sources in much the same way as individuals with a similar developmental level without autism. These results suggest that high functioning individuals may be able to use different strategies, such as their verbal abilities, to compensate for a deficit in emotion processing and that the degree of impairment may not be uniform across individuals with autism.

While little is known about the ability of very young children with autism to match and recognize facial expressions, it has been shown that children with autism fail to attend to the emotional expressions of others

(Dawson *et al.*, 2002b; Sigman *et al.*, 1992; Toth, Dawson, Munson, Abbott, Estes & Osterling, 2001). Adolphs, Damasio, Tranel and Damasio (1996) propose that during development, the child acquires the link between the facial expression of fear and the personal experience of fear. This would require a neural structure that can perceive a specific facial expression, as well as a structure that can link the facial expression (encode and retrieve) to the affective experience of that specific facial expression. Aggleton and Young (2000) have argued that the amygdala is critical for making such associations between a specific stimulus and the affective experiences intrinsically associated with that stimulus.

To assess the emotion perception abilities in young children with autism spectrum disorder, we used recordings of event-related potentials (ERPs) while the children were watching two faces: a face depicting a prototypic fear expression and the same face with a neutral expression. The emotion fear was chosen because it is a salient stimulus, is known to evoke differential ERP responses in infants and children, and is known to activate the amygdala in both children and adults. In addition, work by Sparks, Friedman, Shaw, Aylward, Echeland, Artru, Maravilla, Giedd, Munson, Dawson and Dager (2002) found that young children with strictly defined autism were found to have significantly increased amygdala volume, in excess of increased cerebral volume, compared to typical and developmentally delayed children. This suggests that the anatomical system involved in fear processing may be abnormal at an early age. As mentioned previously, differential ERPs to distinct facial expressions of emotion have been shown in infants as young as 7 months of age (Nelson & de Haan, 1996) as well as in children at 4 and 6 years of age (Nelson & Nugent, 1990; de Haan *et al.*, 1998). Similar to the infant study but in contrast to the previously mentioned child studies, which involved active identification of emotions, the paradigm that we employ in this experiment requires only passive viewing of a fearful and a neutral facial expression, without requiring any overt identification of the stimuli. Attention to the stimuli was carefully monitored, however. We compared the ERP patterns from 3–4-year-old children with ASD and chronologically age-matched typically developing children as well as a mental age-matched subset. Given the results described above, we hypothesized that typically developing children would show faster and larger responses to a fear expression than to a neutral face at both early and late components. In the present study, three possible patterns of results existed for children with autism. First, children with autism might not exhibit differential brain responses to the two expressions. If there was a failure to show differential activity at early

components, this suggests an impairment in visual attention or in the early stage of face perception. If there was a failure to show differential activity at later components, this might suggest difficulties updating or in explicit recognition of the stimulus. Second, the children with ASD might show differential responses, but their responses might differ in lateralization, speed or amplitude, or with respect to the component in which differences are found. For example, typical children may show differential responding at an earlier component than that found in the children with ASD or children with ASD may fail to show a right lateralized response to the neutral face. Third, children with ASD might show responses that do not differ from the comparison group. We also examined the relation between children's ERP responses to an emotional stimulus (fear face), and several tasks that require attention and processing of socioemotional information, namely, tasks tapping social orienting, joint attention and attention to the distress displayed by another person. This is one of the first studies to examine links between behavioral measures and event-related potential measures of emotional processing in children with autism.

Methods

Participants

Two groups of children participated: (a) 63 children with autism spectrum disorder (ASD) who had diagnoses of either Autistic Disorder or Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS) and (b) 28 children with typical development (TYP). Participants were recruited from the University of Washington Autism Center, local parent advocacy groups, public schools, the Department of Developmental Disabilities, clinics, hospitals and the University of Washington Infant and Child Subject Pool. Exclusionary criteria included the presence of a neurological disorder of known etiology (e.g. Fragile X), significant sensory or motor impairment, major physical abnormalities, history of serious head injury, seizures and/or neurological

disease. In addition, children with typical development were excluded if they exhibited unusually high or low (+1 SD) cognitive ability as assessed by their composite score on the Mullen Scales of Early Learning (Mullen, 1997).

Children with ASD were administered a diagnostic evaluation consisting of the Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter & Le Couteur, 1994) and the Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord, Rutter, Goode & Heemsbergen, 1989). Both instruments assess the symptoms of Autistic Disorder listed in the *Diagnostic and statistical manual of mental disorders*, 4th edn (DSM-IV; American Psychiatric Association, 1994). In addition, experienced clinicians made a clinical judgment of diagnosis based on presence/absence of autism symptoms as defined in the DSM-IV. Diagnosis of autism was defined as meeting criteria for Autistic Disorder on the ADOS-G and ADI-R and meeting DSM-IV criteria for Autistic Disorder based on clinical judgment. Also, if a child received a diagnosis of Autistic Disorder on the ADOS-G and based on DSM-IV clinical diagnosis, and came within 2 points of meeting criteria on the ADI-R, the child was also considered to have Autistic Disorder. Diagnosis of PDD-NOS was defined as meeting criteria for PDD-NOS on the ADOS-G, meeting criteria for Autistic Disorder on the ADI-R or missing criteria on the ADI-R by 5 or fewer points, and meeting DSM-IV criteria for Autistic Disorder or PDD-NOS based on clinical judgment. Typically developing children did not meet DSM-IV criteria for Autistic Disorder or PDD-NOS based on clinical judgment.

ERP methods

Participants

Of the initial sample of 63 children with ASD, 29 children (18 with Autistic Disorder and 11 with PDD-NOS) provided adequate artifact-free ERP data (15 were not compliant and 19 provided too few artifact free trials). Of the initial sample of 28 children with typical development, 22 provided adequate, artifact-free ERP data (1 was not compliant and 5 provided too few artifact free trials). Table 1 presents demographic and descriptive

Table 1 Participant descriptive information and number of trials included for each group

Group	N (male)	SES (SD)	CA (mos) (SD)	MA (mos) (SD)	Trials in ave. (SD)	
					Neutral	Fear
ASD	29 (26)	46.0 (14)	44.8 (10) range: 30.0 to 58.7	27.8 (9) range: 15.30 to 46.8	21.9 (8)	22.9 (8)
Typical	22 (19)	52.2 (14)	43.7 (7) range: 31.5 to 57.4	48.4 (8) range: 33.8 to 64.3	27.5 (11)	27.6 (11)

information, including sex, socioeconomic status (SES) based on the Hollingshead Four Factor Index of Social Status (Hollingshead, 1975), chronological age and Early Learning Composite mental age, for the two groups of children included in the final sample. The groups did not differ in terms of sex, SES or chronological age. As expected, the typically developing group had a significantly higher mean mental age (MA) than the ASD group, $F(1, 49) = 67.1, p < .01$.

Because the typical group had a significantly higher mean mental age than the ASD group, we undertook a second set of analyses in which we analyzed only those individuals with a mental age between 29 and 46.75 months.¹ Of the original 29 children with ASD with good ERP data, 11 participants had mental ages in this range. The mean mental age for the MA-match ASD group was 37.7 months (range 29 to 46.75 months). Of the original 22 children with typical development and good ERP data, 11 participants had mental ages in this range; the mean mental age for the MA-match typical group was 41.2 months (range 33.75 to 46 months). There was no statistical difference in mental age for these two subgroups.

Stimuli

Stimuli consisted of digitized photos of a headshot of the same woman posing either a neutral expression or a prototypical fear expression (© Ekman & Friesen, 1976), which were presented in a pseudo-random order. The stimulus frames were 32 cm (520 pixels) wide by 24 cm (420 pixels) high. The emotion recognition study was presented after two other ERP studies reported elsewhere (Dawson *et al.*, 2002a).

ERP procedure

Prior to ERP data collection, each child received up to seven behavioral training sessions to acclimate the child to the testing setting and apparatus, as described in Dawson *et al.* (2002a). The child sat on the parent's lap in front of a table approximately 75 cm from the video monitor that delivered the stimulus in a sound-attenuated room. A large, trifold screen obscured the back of the monitor and the back part of the room from the child's view. The child's head was measured and the vertex was marked. An appropriate size 64 channel Geodesic sensor net (Electrical Geodesics Incorporated; Tucker, 1993) was placed on the child's head and fitted according to

manufacturer's specifications after being dipped into a KCl electrolyte solution. The 64 EEG electrodes covered a wide area on the scalp ranging from nasion to inion and from the right to the left ear arranged uniformly and symmetrically. Impedances were kept below 40 k Ω .

A baseline EEG recording of 100 msec preceded stimulus onset and the stimulus appeared on the screen for 500 msec. ERP data were recorded for an additional 1200 msec following stimulus offset. A variable inter-trial interval of 500 to 1000 msec was added so that the subject would not develop an expectation of when the next stimulus would appear. The total trial length was between 2400 and 2900 msec. An experimenter observed the child through a peephole in the trifold screen, and signaled the computer via button press when the child was not attending. Trials on which the child did not attend were removed. Data collection was terminated when the child had attended to 50 of each of the stimuli or when the child was no longer tolerant of the procedure.

EEG from the 64 channels was registered continuously. The signal was amplified and filtered via a preamplifier system (Electrical Geodesics Incorporated). The amplification was set at 1000X and filtering was done through a: 1 Hz high-pass filter and a 100 Hz elliptical low-pass filter. The signal was digitized at 250 samples per second. Data were collected using the vertex electrode as a reference, and were re-referenced off line to an average reference.

Data editing and reduction

Data were averaged by emotion (fear vs. neutral face); trials that included artifact were excluded. Signals from electrode sites were marked for rejection if signal amplitude exceeded 250 microvolts or a running average of activity exceeded 150 microvolts. A weighted running average is used with the thresholds set to 150 for transit and 250 for voltage. Running averages are analogous to using a band pass filter and reject both high-frequency noise and low-frequency drift. This method identifies the slope and rejects sharp transitions in the data. Trials during which electroocular (EOG) artifact, including eye blinks and movements, occurred were also excluded. EOG electrodes were located superior and horizontal to the eyes; artifact was defined as any activity exceeding 150 microvolts or a deviation in running averages of activity in superior eye channels exceeding 150 microvolts. Trials that had more than 10 electrode sites not meeting these criteria were not included in the average.

The data were then corrected for baseline shifts and low pass filtered at 20 Hz. In addition, an algorithm that derived values from the neighboring sites by spline interpolation was used to replace electrodes for which more

¹ This mental age range was chosen because it includes the greatest number of subjects from the original sample who have good ERP data and overlap in mental age.

than 25% of trials were rejected by artifact. Participants for whom more than 10 channels required this replacement were excluded from further analyses. An average of 3.4 channels ($SD = 2.4$) per participant were replaced for the ASD group. For the typically developing group, an average of 2.9 channels ($SD = 2.3$) were replaced. There was no significant difference in the number of channels replaced ($p > .1$).

Data analysis

Time windows for the components of interest were chosen by visual inspection of the grand-averaged data and the data from individual participants. Electrode groups were identified that included anterior and posterior lateral scalp locations. We have chosen to label the components based on the convention of first defining the direction of the peak (P = positive, N = negative) and then using the average latency of that component for further labeling (e.g. P200 refers to a positive component that peaks at approximately 200 msec), except for the Nc, which is a well-defined component present in infant and child ERP work (Nelson, 1994). Six components were analyzed: (1) the P200 was defined as the maximum positive peak between 150 and 270 msec at posterior leads; (2) the N300 was characterized as the most negative peak value between 270 and 370 ms at posterior leads; (3) the P300 was characterized as the most positive value between 270 and 370 ms at anterior leads; (4) the Nc was characterized as the negative peak between 270 and 710 ms at anterior leads; (5) the P500 was calculated as the most positive peak between 350 and 670 ms at posterior leads;² (6) the negative slow wave (NSW) was calculated as the average amplitude between 810 and 1170 ms at posterior leads. The amplitude and latency at which the peak values occurred for the P200, N300, P300, Nc and P500 was visually verified to occur within the specified temporal window for each subject at the specified leads by one of the authors (SW). The ERP data were then averaged over regions of interest for each component. Data used in the statistical analyses reflect the amplitude and latency of the peak value within a given time range (or average across the time window) for each individual; data presented in the tables are the average of these values. The placement of electrodes in the geodesic sensor net system and the electrodes over which data were averaged for each component are shown in

² Dawson *et al.* (2002a) called this component a P400 due to its latency range, between 400 and 450 msec. In contrast, the latencies found in this paper range from 480 to 530 ms. Although the component has the same distribution, we are using the ERP nomenclature of identifying the component based on the mean latency in this data set.

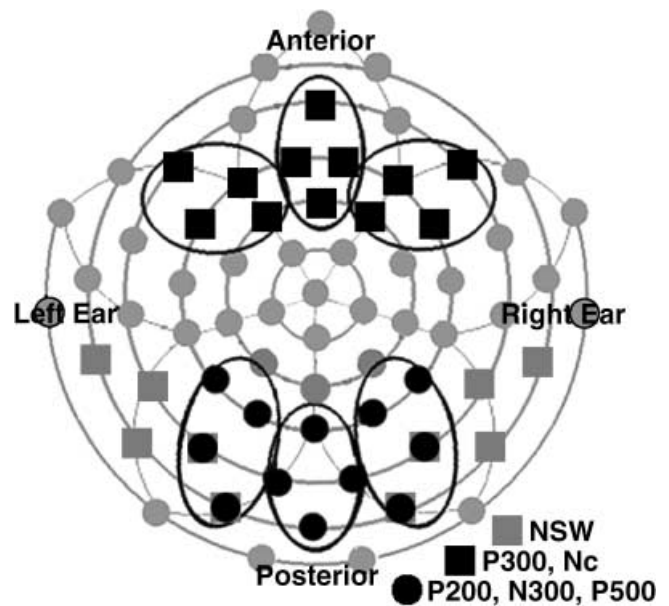


Figure 1 Topographical plot of electrodes included in the analysis.

Figure 1. ERP grand-averaged waveforms were created for each group per emotion. The grand-average waveform is calculated by averaging the amplitude at each time point. Because of the difference in how the waveforms are calculated versus the data used in statistical analyses, values displayed in the tables may differ from the figures.

For each component of interest, a Group (ASD versus TYP) by Emotion (neutral versus fear face), by electrode region (e.g. right, left, midline) repeated measures analysis of variance (ANOVA) was conducted. In order to determine if the results could be accounted for (a) by disruptions in general face processing ability for the ASD group, analyses were repeated using only the neutral face or (b) by a mental age differences between the groups, analyses were repeated using a subset of participants who were MA matched. Dependent variables consisted of the P200, N300, P300, P500 and Nc peak amplitude and latency. The dependent variable for the negative slow wave was mean amplitude over the time period of interest.

Behavioral tasks

Participants

The majority of children with ASD also were administered several behavioral tasks. Of the 29 children (18 with Autistic Disorder and 11 with PDD-NOS) who provided adequate artifact-free ERP data, 28 had valid data for a social and non-social orienting task, 26 for a joint attention task and 25 for an attention to distress task.

Procedure

On a separate day from ERP data collection, each child with ASD was administered the behavioral tasks. Detailed data from these tasks collected from a larger sample of ASD children, which included this sub sample, are also reported elsewhere (Dawson *et al.*, 2002b).

Social and non-social orienting task

The four social stimuli consisted of live sounds using the human voice and body: (a) humming a neutral tone, (b) calling the child's name, (c) snapping fingers and (d) patting hands on thighs. The four non-social stimuli consisted of mechanical sounds of inanimate objects activated by a human being: (a) a timer beeping, (b) a phone ringing, (c) a whistle blowing and (d) a tape recording of a car horn delivered while the examiner held a toy car. We do not have direct evidence whether the children made the distinction between social and non-social stimuli. Each stimulus was delivered three times with a 1-second inter-stimulus interval, for a total presentation of approximately 6 seconds. A decibel meter placed on the table next to the child for a random subset of participants confirmed that social and non-social stimuli were matched in terms of loudness. To control for acoustics, the same testing room was used for all children participating in this task. Stimulus order and location (behind vs. in front of the child, 30 degrees to right or left) were counterbalanced across participants. Delivery of the stimuli occurred only when the child was not already looking at the second examiner, and the examiner remained in each location for 15 seconds following delivery to allow for a delayed orienting response. The examiner assumed a neutral facial expression when delivering the social stimuli, and looked down at the floor when delivering the non-social stimuli, to ensure that the child's response was not unduly influenced by personal or social characteristics specific to the examiner.

Children's behavior was videotaped from behind a one-way mirror. The two examiners coded live whether the child looked toward the stimulus. An error was defined as a failure to turn eyes toward the stimulus within 15 seconds from delivery of the stimulus. The score was the proportion of stimuli to which the child oriented. Coding discrepancies, though rare, were resolved immediately following the task by viewing the videotape. An additional coding from videotape by coders blind with respect to hypotheses was obtained for a random subset of participants (19% of total sample). The intraclass correlation coefficient for the live versus videotape coding was .87.

Joint attention task

Based on a paradigm developed by Seibert and Hogan (1982), the child was presented with a variety of toys, the experimenter then tried to draw the child's attention to distal objects (three posters on the wall 90 degrees to the child's right and left, and 180 degrees behind the child, other toys). Intermittently, the examiner attracted the child's attention, then turned to point and gaze at each poster while stating the child's name three times. The score was the percentage on which the child accurately oriented with eyes and/or head turn beyond the examiner's finger and in the direction of the examiner's point and gaze. Initial inter-rater reliability was first assessed by independent paired ratings made from 15 practice tapes provided by Peter Mundy, University of Miami, who developed a behavioral coding system for this measure. Intraclass correlation coefficients for all scores derived were above .84. Reliability was then assessed by independent paired ratings made from videotapes of a randomly selected group of tapes (10% of total sample). Intraclass correlation coefficient across observers was .76 for the 'response to joint attention' score, which was used in this study.

Attention to distress versus humming task

The method used to measure attention to another's distress was based on that used by Sigman and colleagues (Sigman *et al.*, 1992). In this procedure, a familiar examiner allowed the child to play with either a wooden or plastic hammer and peg toy. The examiner then requested a turn and pretended to hurt him/herself by hitting one finger with the hammer. For 30 seconds, the examiner displayed facial and vocal expressions of distress ('crying') without using words, followed by a 10-second period of neutral affect with the examiner looking down. The examiner then showed the child his/her finger, stating, 'It is all better now.' In a second trial, the examiner again requested a turn with the toy and hit his/her finger, but this time displayed neutral affect while humming a neutral tone for 30 seconds, followed by 10 seconds of silence. The dependent variables were duration of gaze to the experimenter (time seconds) and degree of concern (4-point scale measuring affective involvement, facial expression, comforting behaviors); higher values reflect greater time looking toward the experimenter and greater degree of concern. Total time in half-seconds was calculated for each of these categories, as well as latency to first look at the examiner. Degree of concern was rated on a scale of 1 to 4 as follows: (1) shows no interest (did not look once at examiner), (2) shows some interest, but no concern (brief looking, neutral or positive facial expression), (3) shows

concern (facial expression shows concern, worry, or discomfort; looked at examiner more than briefly), and (4) showed intense affective involvement and/or comforting behaviors. Initial inter-rater reliability for the joint attention task was assessed by independent paired ratings on 15 subjects; intraclass correlation coefficients were above .84. Inter-rater reliability for the attention to distress task was assessed by independent paired ratings made from videotapes for a randomly selected group of participants in the study; intraclass correlation coefficients ranged from .79 to .99 for all variables.

Results

ERP data: comparisons of children with ASD with CA-matched typical children

P200, posterior leads

We labeled this component the P200 as it was a positive going component and peaked at approximately 210.3 msec (SD 24) in the TYP group and 220.3 msec (SD 20) in the ASD group, $F(1, 45) = 4.5, p < .05$. There was also a main effect of emotion, with responses to the fear face ($M = 210.8$ msec, SD 23) peaking faster than those to the neutral face ($M = 219.6$ msec, SD 22). When the responses to the neutral face were analyzed independently, there was a main effect of group, $F(1, 47) = 7.2, p = .01$, with the TYP group demonstrating faster responses ($M = 212.3$ msec, SD 23) than the ASD group ($M = 224.5$ msec, SD 19) to the neutral face.

The amplitude of this component was greatest at posterior midline leads, $F(2, 44) = 16.3, p < .001$. There was an interaction of emotion by hemisphere by group, $F(2, 42) = 5.2, p < .05$. There were no significant differences between the ASD and TYP group for responses to the fear face. As seen in Table 2, the interaction was driven by the group differences in the responses to the neutral

Table 2 P200 amplitude (in microvolts) to fear face versus neutral face for the CA- and MA-matched analyses

Group	Hemisphere	Fear face	Neutral face
CA Typical	Right	7.2 (6)	6.1 (7)
	Left	6.6 (5)	8.0 (5)
CA ASD	Right	8.9 (7)	6.5 (5)
	Left	7.3 (7)	4.4 (8)
MA Typical	Right	5.9 (7)	4.6 (6)
	Left	7.4 (6)	5.3 (6)
MA ASD	Right	9.7 (7)	7.7 (4)
	Left	9.0 (6)	2.3 (7)

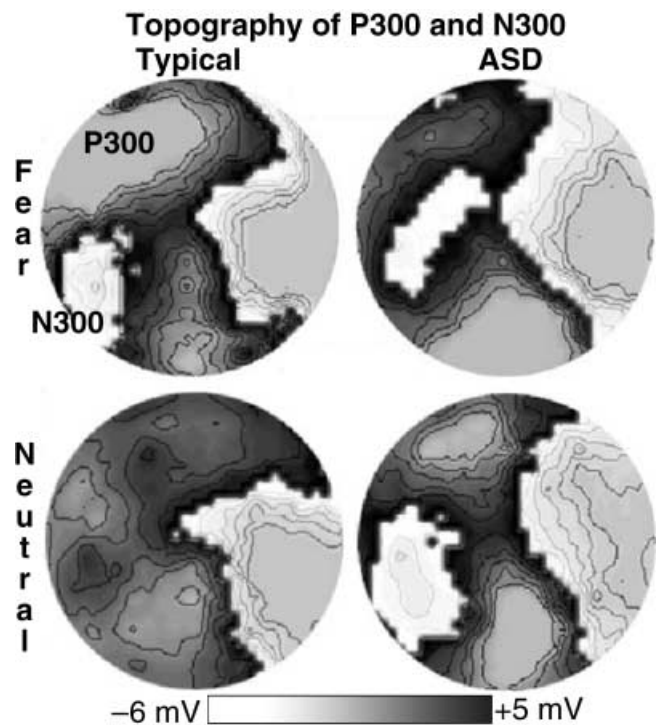


Figure 2 Topographical contour plot of the N300. This plot reflects the amplitude distribution at the peak of the N300.

faces and in particular, the significantly smaller amplitude from the ASD group to the neutral face at left leads.

N300, posterior leads

The N300 was a negative going component which peaked at 310.5 msec (SD 25) in the TYP group and 324.4 msec (SD 30) in the ASD group, $F(1, 45) = 5.0, p < .05$. For both groups, N300 latency was faster for the right ($M = 312.2$ ms, SD 30) than the left hemisphere ($M = 322.7$ ms, SD 28), $F(1, 45) = 7.2, p = .01$. In addition, a three-way interaction among group, hemisphere and emotion was found, $F(1, 45) = 5.7, p < .05$. In follow-up tests, typically developing children were found to exhibit a faster response to the fear versus neutral face and this difference was accentuated for the left hemisphere $F(1, 20) = 6.2, p < .05$, whereas children with ASD displayed no significant difference in N300 latency to the fear versus neutral face, $F = 2.1, p = ns$.

There were no main effects of group or emotion for N300 amplitude. There was a significant group by emotion interaction, $F(1, 45) = 5.1, p < .05$, however. As shown in Table 3, the typically developing group showed a larger N300 amplitude to a fear face than a neutral face, whereas for children with ASD there were no significant differences between the two stimuli.

Table 3 N300 amplitude (in microvolts) and latency (in milliseconds) to fear versus neutral face for the CA-matched analyses

Group	Hemisphere	Fear face	Neutral face
Typical	Right	-3.1 (6)	-1.3 (7)
	Left	-3.0 (5)	-0.5 (5)
ASD	Right	-1.2 (7)	-2.2 (6)
	Left	-2.1 (6)	-3.9 (7)
Typical	Right	306.5 (23)	309.0 (17)
	Left	303.2 (34)	323.5 (27)
ASD	Right	314.5 (32)	318.7 (32)
	Left	334.3 (29)	330.0 (28)

When responses to the neutral face alone were examined, the typically developing children showed a right hemisphere bias for the processing of neutral face, i.e. faster N300 latencies to the neutral face over the right hemisphere than the left, $t(20) = -2.3, p < .05$. The ASD group showed no right/left hemispheric differences in N300 latency to the neutral face, $p = ns$.

P300, anterior leads

The P300 was a positive going component that peaked at approximately 310 msec. The component was greater at midline leads, in terms of amplitude, than at the lateral leads, $F(2, 39) = 10.3, p < .001$. The ANOVA yielded no main effects or interaction involving emotion or group for either amplitude or latency.

Nc, anterior leads

Figures 3 and 4, respectively, show the ERP response for the ASD and TYP groups to the fear and neutral faces at the anterior leads where this component was most prominent. For both groups, Nc was of greater amplitude in the right than left hemisphere, $F(1, 41) = 9.3, p < .01$. The ANOVA yielded no main effects or interaction involving emotion or group for either Nc amplitude or Nc latency.

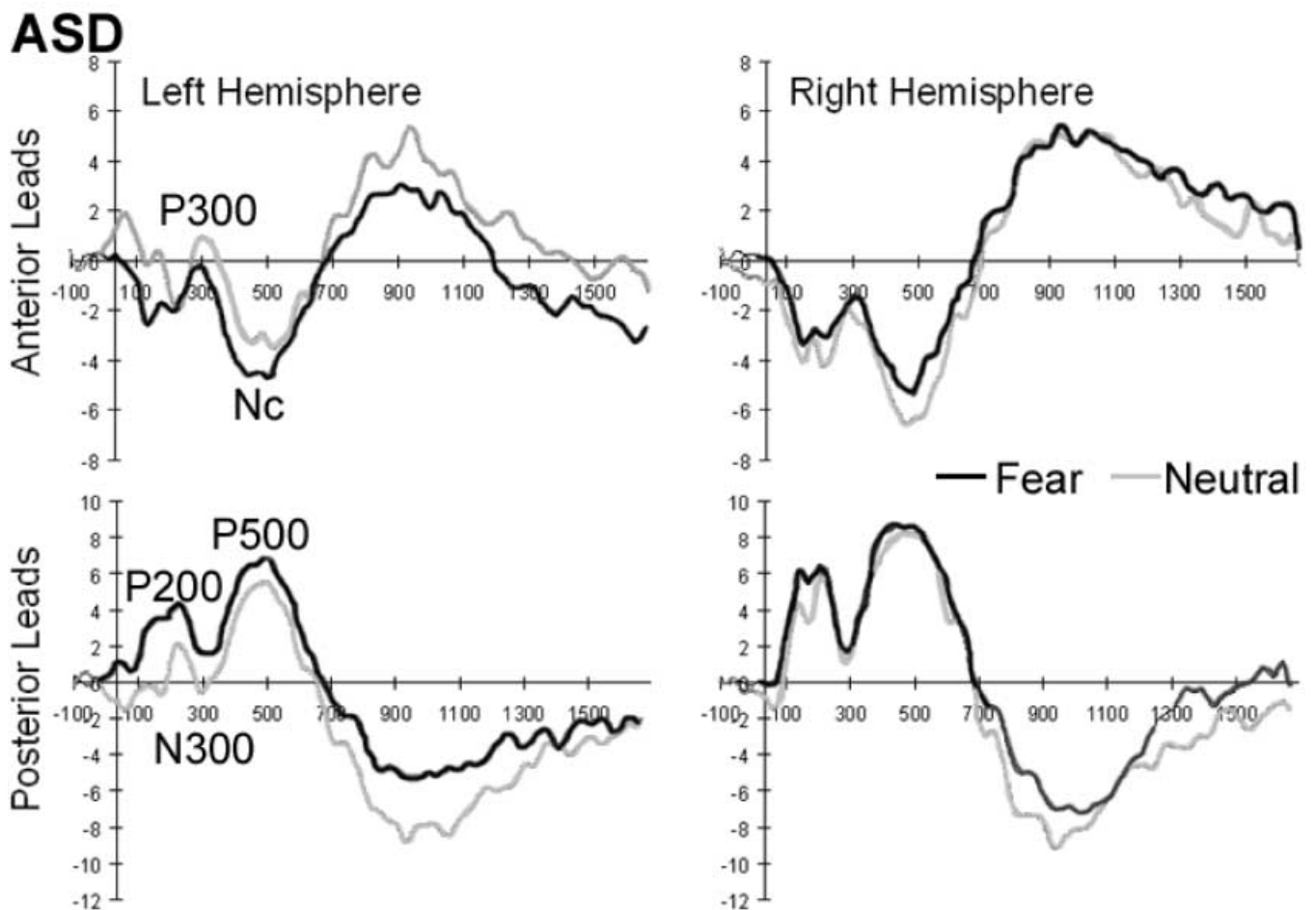


Figure 3 Grand mean ERP waveforms at anterior and posterior leads for ASD group.

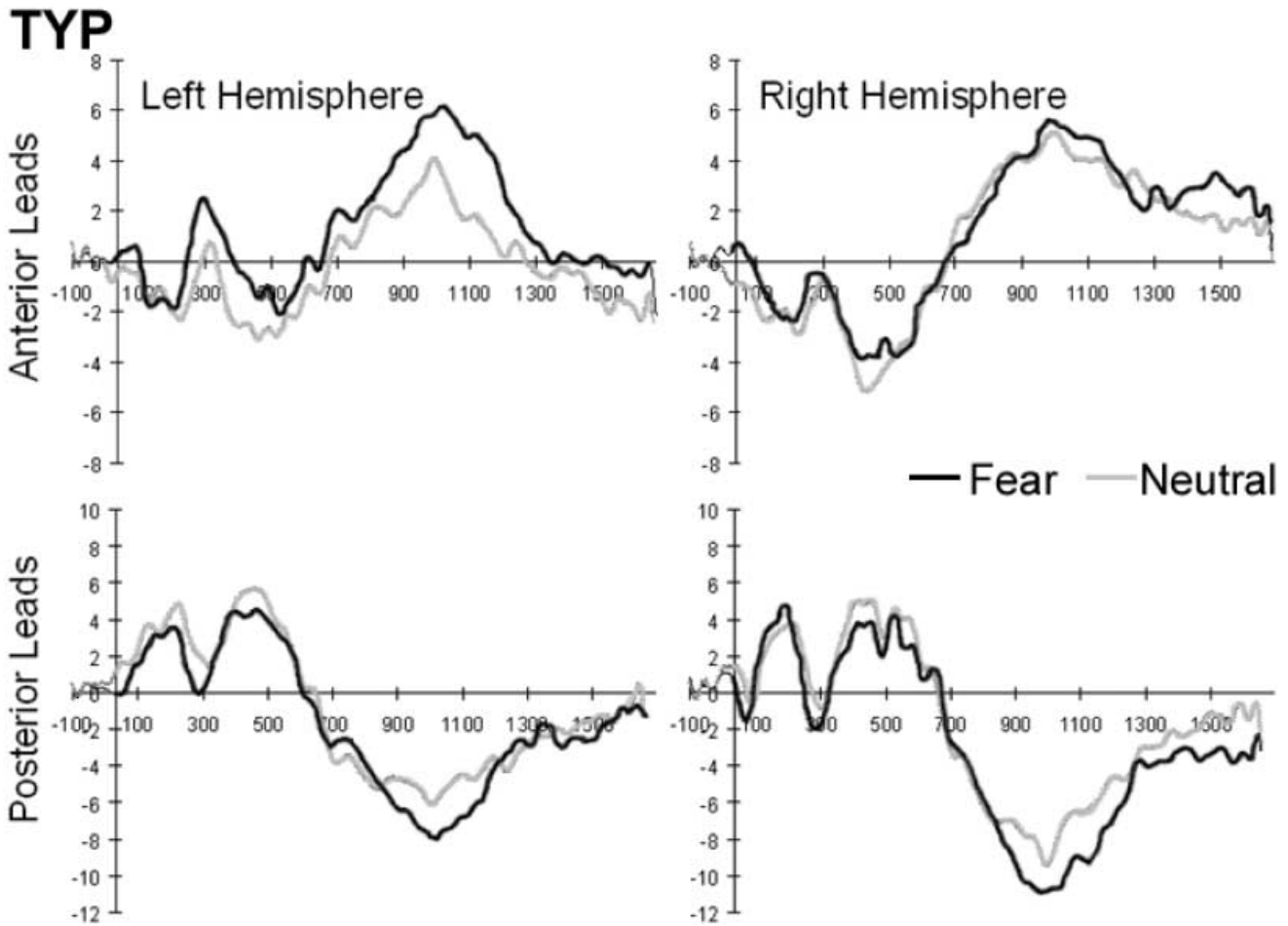


Figure 4 Grand mean ERP waveforms at anterior and posterior leads for typical group.

P500, posterior leads

The P500 had a posterior distribution, with greater amplitude in the right than left hemisphere, $F(1, 43) = 7.9$, $p < .01$; see Figure 3 for ASD responses and Figure 4 for TYP responses. There were no main effects of emotion or group for the amplitude or latency of this component.

Negative slow wave (NSW), posterior leads

The NSW had a posterior distribution, with greater activity in the right than left hemisphere, $F(1, 49) = 6.9$, $p = .01$. ANOVA of the NSW revealed no main effects of emotion or group for the amplitude of this component, but did reveal significant interactions between emotion and group, $F(1, 49) = 6.4$, $p = .01$, and between group and hemisphere, $F(1, 49) = 4.8$, $p < .05$. As seen in Table 4 typically developing children showed greater NSW amplitude to the fear than the neutral face ($p < .05$), whereas there were no differences in NSW amplitude to the fear versus neutral face for the ASD group ($p = ns$).

Table 4 Negative slow wave amplitude (in microvolts) to fear versus neutral face

Group	Fear	Neutral
Typical	-6.4 (5)	-4.3 (7)
ASD	-4.6 (8)	-6.1 (7)

ERP data: comparisons based on MA-matched sub samples

Group comparisons of the ERP were next made on the two subgroups of ASD and typical children who were matched on mental age.

P200, posterior leads

Similar to the CA-matched comparison, there was a main effect of emotion, with responses to the fear face ($M = 206.8$ msec, $SD = 25$) peaking faster than those to the neutral face ($M = 222.0$ msec, $SD = 21$), $F(1, 18) =$

Table 5 N300 amplitude (in microvolts) and latency (in milliseconds) for the MA-matched analyses

Group	Hemisphere	Fear	Neutral
Typical	Right	-4.6 (5)	-2.9 (8)
	Left	-5.4 (5)	-2.9 (5)
ASD	Right	-.19 (8)	-.37 (8)
	Left	-3.7 (6)	-5.7 (8)
Typical	Right	306.4 (18)	316.2 (13)
	Left	296.9 (40)	319.8 (21)
ASD	Right	302.7 (22)	307.5 (29)
	Left	331.5 (29)	331.5 (27)

15.8, $p < .001$. The effect of group did not reach significance in this analysis ($F = 64$, $p = ns$). For the amplitude of the P200, the emotion by hemisphere by group interaction was not found in this sub sample, although the values in Table 2 suggest a similar pattern of response. There was a main effect of emotion and hemisphere; responses to the fear face were greater in amplitude ($M = 8.8$ mV, $SD = 8$) than those to the neutral face ($M = 5.7$ mV, $SD = 6$), $F(1, 18) = 4.7$, $p < .05$ and greater at midline leads than at lateral leads, $F(2, 17) = 6.5$, $p < .01$. There was also an interaction between group and hemisphere, with responses from the ASD group of greater amplitude at right and midline leads, than those displayed by the TYP group, $F(2, 17) = 4.0$, $p < .05$.

N300, posterior leads

For N300 amplitude results, as shown in Table 5, there was an interaction between emotion and group, $F(1, 20) = 7.1$, $p < .05$ that paralleled the findings for the CA-matched group. Typically developing group showed a larger N300 amplitude to a fear face than a neutral face, whereas for children with ASD, there were no significant differences between the two stimuli. For the N300 latency effects, although the MA-matched subgroups showed a very similar pattern of results, only the effect of hemisphere was replicated, $F(1, 20) = 5.3$, $p < .05$. There was an interaction between group and hemisphere, $F(1, 20) = 8.3$, $p < .01$, in which the response from the ASD group was significantly slower than the typical group in the left hemisphere to both types of stimuli.

P300, anterior leads

There were no effects for the latency to peak of the P300 for the MA-matched groups; there was a main effect of emotion and lead for the amplitude of the component. The component was more positive to neutral faces ($M = 3.0$ mV, $SD = 5$) than to fear faces ($M = 1.2$ mV, $SD = 8$) and the response was greatest at midline leads,

$ps < .05$. This effect was driven by the ASD group, which demonstrated a significantly greater response to the neutral face ($M = 3.7$ mV, $SD = 5$) than to the fear face ($M = -1.1$ mV, $SD = 4$), $p < .05$. The TYP group demonstrated a greater response to the fear face ($M = 3.4$ mV, $SD = 5$) than to the neutral face ($M = 2.4$ mV, $SD = 4$), $p = ns$.

NC, anterior leads

The effect of hemisphere was similar to the CA analysis, with more negative responses in the right than left hemisphere, $p < .05$.

P500, posterior leads

There were no effects for this component.

NSW, posterior leads

For the negative slow wave findings, as found for the CA-matched comparison, the ANOVA revealed a group by emotion interaction, $F(1, 20) = 22.7$, $p < .01$ (M s and SD s for typical group to the fear face = -8.8 (6) and to the neutral face = -6.0 (9) and for the ASD group to the fear face = -0.8 (7) and to the neutral face = -7.5 (10)). Typically developing children showed greater NSW amplitude to the fear than to the neutral face, whereas the ASD children did not.

Summary of ERP findings

In summary, there were three patterns of effects that were significant for both the CA- and MA-matched analyses: (1) for both groups, the response to the fear face was faster than that to the neutral face at the P200 at posterior leads; (2) typically developing children showed a greater N300 response to the fear face as compared to the neutral face, whereas children with autism did not show this difference to the fear versus neutral face; (3) at a still later component (NSW), typically developing children again showed a larger amplitude response to the fear face compared to the neutral face, whereas children with ASD did not show such differences. The P200 amplitude and latency and N300 latency group differences for the CA analyses were not replicated in the MA-matched analyses, although the subgroup displayed the same pattern of results (Tables 2, 3, 5). This suggests that these findings, and in particular the latency results, may be influenced by the mental age difference between groups. In contrast, the P300 amplitude effects at anterior leads were found only in the MA-matched analyses but not in the larger group.

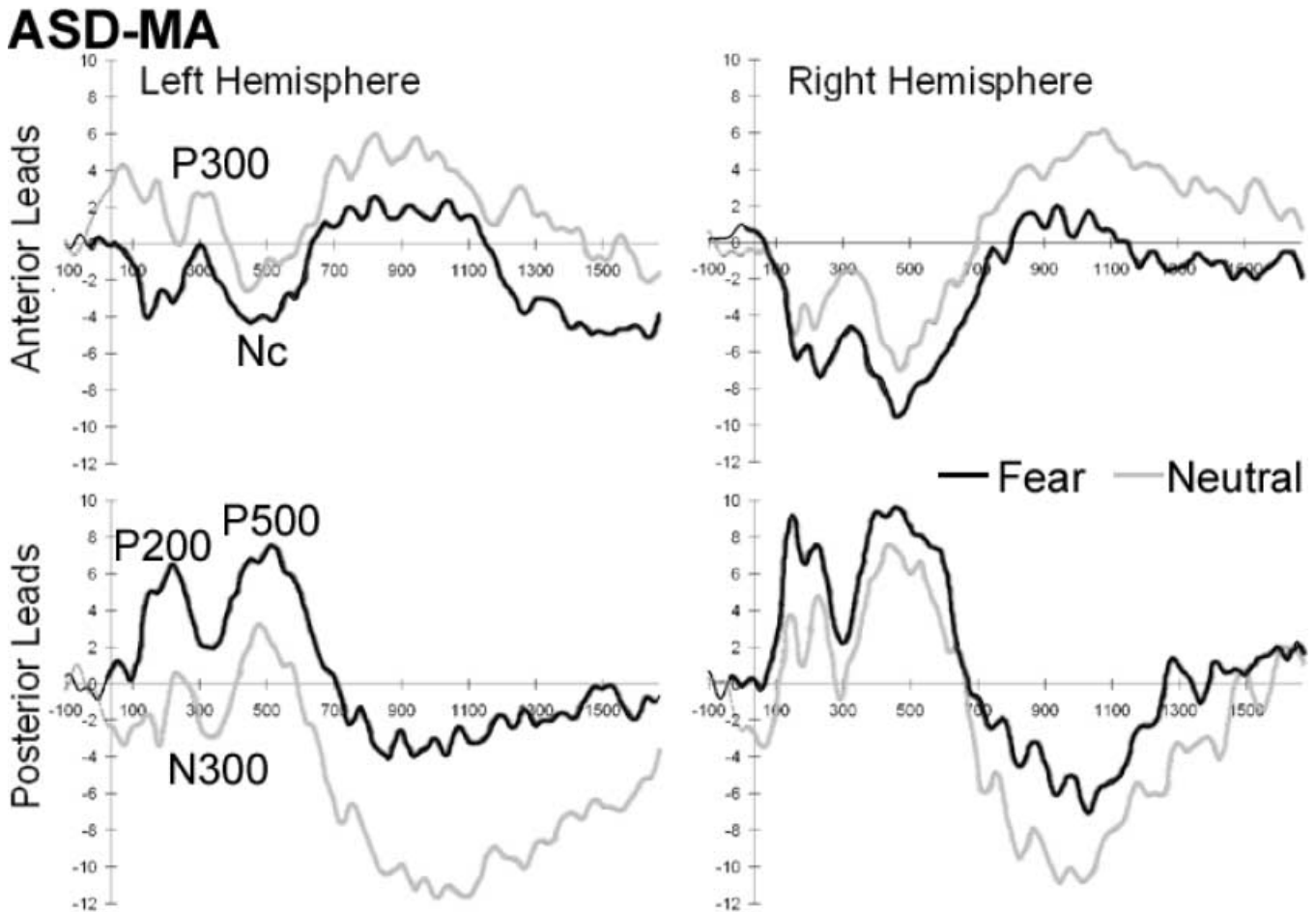


Figure 5 Grand mean ERP waveforms at anterior and posterior leads for ASD-MA-matched sub group ($N = 11$).

Behavioral tasks

In order to examine the relation between the behavioral tasks and the ERP measures to the fear face, we used those ERP measures in which differences between the ASD and TYP groups were found, including the P200 amplitude and latency, N300 amplitude and latency, P300 amplitude and NSW average amplitude. Pearson partial correlations were performed examining the relations between these ERP variables and the behavioral measures in the ASD group, controlling for IQ based on Mullen's Early Learning Composite mental age / chronological age in months, $M = 100$, $SD = 15$. The results showed that faster N300 latency to the fear face for the ASD group was associated with better joint attention ability, fewer social orienting errors, and longer time spent attending to experimenter expressing negative emotion (distress). NSW amplitude to the fear face (right hemisphere) was correlated with joint attention and non-social orienting. See Table 6 for a summary of the cor-

relation patterns for the N300. There were no significant correlations between the P200 and P300 responses to the fear face and the behavioral tasks.

Discussion

The 3- to 4-year-old typically developing preschoolers displayed a larger N300 response to a fear face than a neutral face. Typically developing children also showed a larger amplitude slow wave response (between 810 and 1170 msec) to the fear face compared to the neutral face. These results are similar to those of a previous study of typically developing children conducted by de Haan *et al.* (1998); however, they do differ in several areas. Primarily, our subjects were younger and we did not use an active detection task. Second, we neither detected a negative peak at anterior leads between 80 and 170 nor at posterior leads between 170 and 250 msec in our sample. However, we did analyze a positive peak between

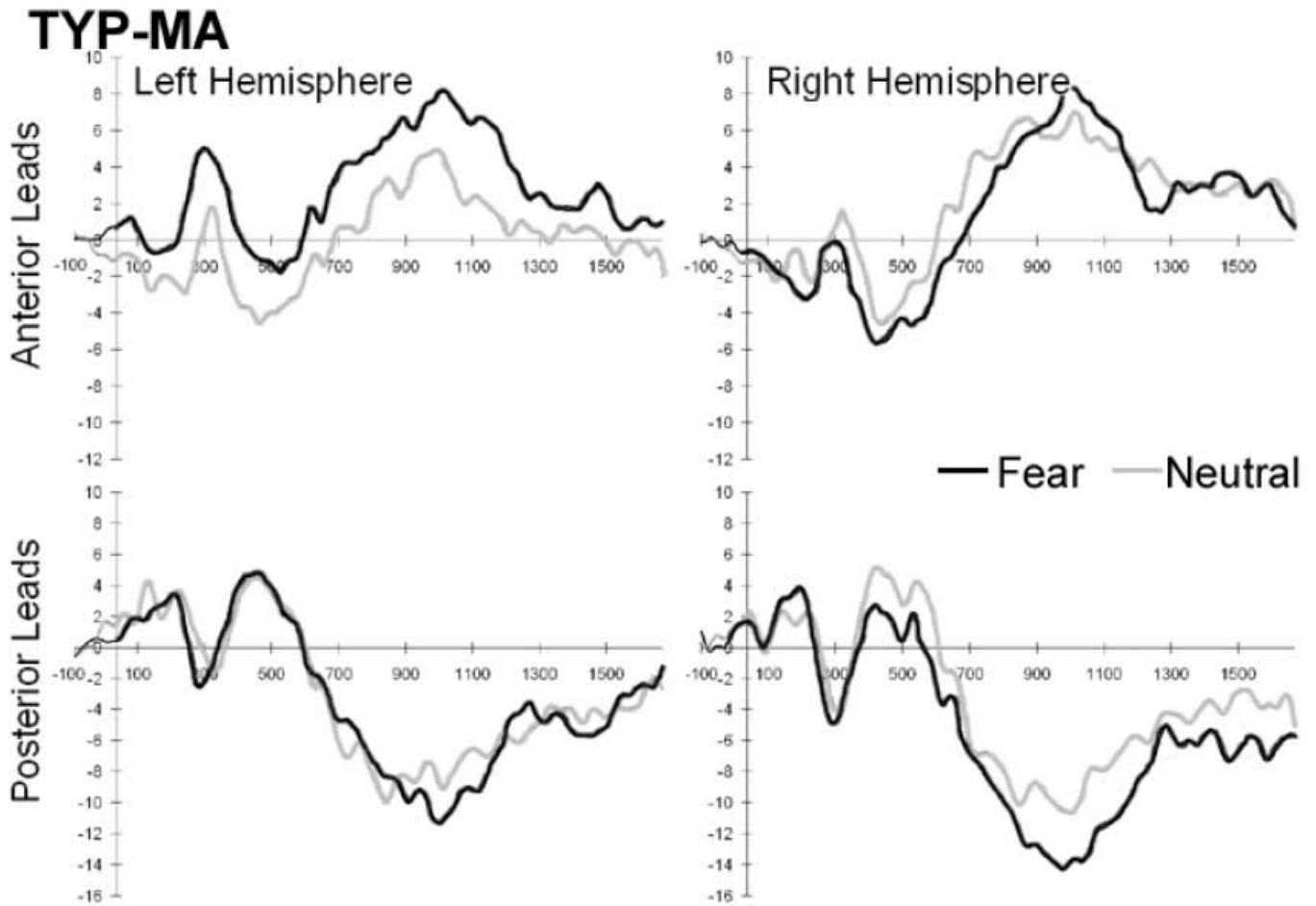


Figure 6 Grand mean ERP waveforms at anterior and posterior leads for typical-MA-matched sub group (N = 11).

Table 6 Relations between N300 latency to the fear face and behavioral measures in children with ASD.

ERP measure	Social attention				Non-social attention		
	Joint attention	Social orienting	Attention to other's distress	Degree concern to other's distress	Non-social orienting	Attention to humming	Degree concern to humming
N300 Amplitude Right	ns	-.40*	ns	ns	ns	ns	ns
N300 Amplitude Left	.42*	ns	ns	ns	ns	ns	ns
N300 Latency Right	-.49*	ns	ns	ns	ns	ns	ns
N300 Latency Left	-.46*	-.43*	-.53**	ns	ns	ns	ns

Significance values: ** $p < .01$; * $p < .05$.

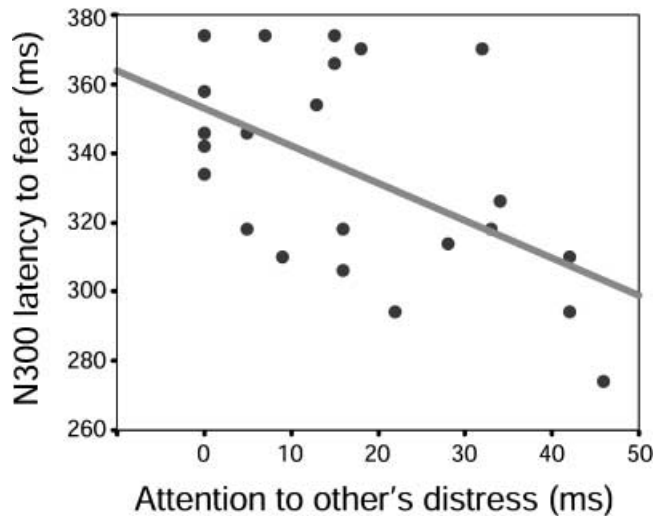


Figure 7 Scatter plot of relation between N300 latency to the fear face at left hemisphere leads and the time the child spent attending to an experimenter's distress.

170 and 270 msec (P200) and a negative peak between 270 and 370 msec (N300). Visual inspection of the graphs from the de Haan *et al.* study suggests that they did not have a component comparable to our P200. It is possible that our N300 is the same component as the de Haan *et al.* posterior N170, only temporally delayed. For the TYP group, this component was faster to fear faces (similar to de Haan *et al.*) and of more negative amplitude. If this component is similar to the developmental N170 that Taylor *et al.* (1999, 2001) describe as a face specific processing component, then this report suggests that facial expression, specifically fear, influences the early stage processing of faces in typically developing children and that the developmental N170 does not have the same response profile as the adult N170. As we did not include a non-face stimulus in this experiment, and thus have no direct evidence that this component preferentially activates to faces, this relation must be treated with caution. Third, it is unknown if the de Haan *et al.* P280 (220 to 370 msec) is similar to the anterior P300 reported in this paper. If the P300 is the same as the P280, there is less than 20 msec of developmental improvement in latency from 3–4 years and 5 years of age; in contrast, if the N300 is the same component as the N170, this component becomes 100 msec faster during this time period. Regardless, both reports do suggest that fear processing influences early components (within 300 msec of presentation) of the ERP in young typically developing children. Whether this is due to facilitated face processing or attention is unknown.

We tested three alternatives as to the possible ERP response pattern to fear for the children with autism: (1)

the children with ASD would show no differential responses between fear and neutral; (2) the children with ASD would show an alternative response pattern compared with the typical children; or (3) the ASD group would demonstrate responses that were similar to typically developing children. Unlike the typical group, children with ASD did not show a differential response to the fear and neutral face at the N300 or at the negative slow wave. If we limit our conclusions to the results that were significant for both the CA- and MA-matched analyses, these results support the first prediction, that children with ASD did not show differential amplitude responses to fear and neutral faces. One interpretation of these findings is that the fear stimulus does not facilitate attentional or early stage face processing circuits (N300). This would then have downstream consequences for perceptual or conceptual updating (NSW).

Do these results reflect a general disadvantage in processing faces for the ASD group or a specific disadvantage in processing of emotion? Because processing of facial expression requires general face processing, any abnormalities in the initial stages of face processing would likely disrupt processing of the emotion displayed on the face. Our results do suggest some abnormalities in the processing of neutral faces for the children with ASD. As seen in Table 2, the P200 amplitude response by the ASD group to the neutral face at left leads was significantly different from the response at right leads and from the typically developing children's responses. In addition, P200 and N300 latency (Tables 2, 3), *in general*, was slower for the ASD group compared to the typical group. The topography of the N300 (Figure 2) as well as the NSW was different wherein typical children exhibited a right hemisphere advantage (larger and faster neural response), whereas children with ASD did not. Indeed, in the present study, it is difficult to separate the process of perception of emotion from perception of face. In the ERP task, the children saw a neutral facial expression and a fearful facial expression within the same block of trials. For example, Thomas *et al.* (2001b) have suggested that neutral faces are ambiguous in their emotional meaning to children and thus also evoke an emotional processing system (i.e. the amygdala), thus one could interpret the paradigm as involving the processing of two types of emotional expressions. In addition, we did not have a non-face stimulus to compare these results with and thus do not have the means to dissociate a face processing component from a facial expression of emotion component. In any case, it is possible that the atypical responses to the neutral face shown by the children with ASD in the present study reflect abnormalities in general face processing, emotional processing, or both.

As a caveat, the degree to which these results can be generalized to other emotions is unknown. The amygdala does activate to other emotions, albeit to a lesser degree (e.g. Morris *et al.*, 1996). For other negative emotions, such as disgust, angry, and frightened, fMRI studies with adults have found that the insula (disgust; Phillips *et al.*, 1997), the medial frontal gyrus (angry), and inferior frontal gyrus (frightened; Kesler-West, Anderson, Smith, Avison, Davis, Kryscio & Blonder, 2001) as well as other areas were activated. In terms of ERP results, de Haan *et al.* (1998) found differential processing of emotions across three components: responses to the angry face were of greater amplitude at the N400; responses to the happy face were of greater amplitude at the P280 in the right hemisphere; and responses to the fear face were faster at the N170. This suggests that there may be some specificity to the pattern of results depending on which emotions are being compared.

Abnormalities in the amount of processing, measured by ERP amplitude, specifically a primary emotion such as fear, might have further consequences for social behavior. If the child is incorrectly processing the emotion by failing to activate structures that are normally specialized for emotion processing, the stimulus information may not be correctly bound to concurrent information about the context of the facial display or the auditory input that may accompany it. Failure to exceed threshold of activation for a region or a process may result in limited or reduced neural transmission according to Hebbian principles or may result in unspecified input to higher-level structures that might be used to further process the stimulus. These abnormalities could result in the facial display being stored independently from the other events that accompany it and may make it inaccessible to other systems.

In order to further investigate whether or not processing of a facial expression is correlated with degree of actual social impairment, we examined the relation between individual differences in ERP latency and amplitude to the fear stimulus and performance on tasks requiring social attention, as well as control tasks involving attention to non-social stimuli. This analysis is one of the first to directly look at the link between brain electrical activity and behavioral performance on two different tasks that might share a common neural circuitry. It has been proposed by a number of researchers (e.g. Baron-Cohen *et al.*, 2000; Brothers *et al.*, 1990; Dawson, 1996; Dawson *et al.*, 2002a, 2002b) that the amygdala is one of several regions that are abnormal in autism. Based on results from adult fMRI studies, which point to the amygdala's role in fear processing, we expect that the processing of fear in children also involves the amygdala and will be correlated with other social behaviors that involve amygdala circuitry.

Of the measures of social attention and emotional processing, three measures were correlated with the speed of early ERP responses to a fear expression. Specifically, faster N300 to the fear face was associated with improved performance on tasks requiring social attention. Children who displayed a faster N300 latency to the fear face exhibited better joint attention skill, fewer social orienting errors, and spent more time looking at an experimenter expressing distress. In contrast, there was no association between N300 latency as well as other components and performance on the non-social tasks. When we split the autism group in terms of joint attention ability, i.e. created a group who responded to the joint attention probes more than 50% of the time versus those who responded 50% or less to the probes, it was found that the children in the former group exhibited N300 latencies that were similar in duration to those exhibited by the typical group. The behavioral analysis did not include typically developing children; thus, the degree to which our results are specific to autism versus a more general characteristic is unknown. However, taken together, these results suggest that the speed of neural processing of facial expressions of fear is related to more complex social behaviors.

In conclusion, our results suggest that young children with autism display aberrant neural responses to both neutral and fearful faces. Whether this is reflected in a failure of cortical specialization or possibly atypical recruitment of cortical areas by 3 to 4 years of age is still unknown.

Acknowledgements

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