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Neural Correlates of Face and Object Recognition in Young Children with Autism Spectrum Disorder, Developmental Delay, and Typical Development

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Abstract

This study utilized electroencephalographic recordings to examine whether young children with autism spectrum disorder (ASD) have impaired face recognition ability. High-density brain event-related potentials (ERPs) were recorded to photos of the child's mother's face versus an unfamiliar female face and photos of a favorite versus an unfamiliar toy from children with ASD, children with typical development, and children with developmental delay, all 3 to 4 years of age (N = 118). Typically developing children showed ERP amplitude differences in two components, P400 and Nc, to a familiar versus an unfamiliar face, and to a familiar versus an unfamiliar object. In contrast, children with ASD failed to show differences in ERPs to a familiar versus an unfamiliar face, but they did show P400 and Nc amplitude differences to a familiar versus an unfamiliar object. Developmentally delayed children showed significant ERP amplitude differences for the positive slow wave for both faces and objects. These data suggest that autism is associated with face recognition impairment that is manifest early in life.

INTRODUCTION

Autism is a developmental disorder characterized by qualitative impairments in social interaction and communication and a restricted range of activities. Individuals with autism have specific impairments in the processing of social and emotional information (Baron-Cohen, Tager-Flusberg, & Cohen, 1993; Davies, Bishop, Manstead, & Tantam, 1994; Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Hobson, Ouston, & Lee, 1988a, 1988b; Mundy, Sigman, Ungerer, & Sherman, 1986; Smith & Bryson, 1994; Teunisse & De-

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Gelder, 1994). Even on simple attention tasks, such as orienting to auditory stimuli, children with autism are less likely to orient to social stimuli (e.g., clapping) than to nonsocial stimuli (e.g., a rattle; Dawson, Meltzoff, Osterling, & Brown, 1998; Dawson et al., 2002). The basic nature of these impairments suggests that autism is related to dysfunction of brain regions specialized for early-stage processing of social information.

This study further explored the nature of early impairments in social cognition in autism. We were interested in assessing very young children's electrical brain responses to familiar and unfamiliar faces, and focused on face recognition in autism for three reasons. First, the profound disability in social cognition found in autism may be evident first in a failure to attend to faces. In a study of home videotapes of first birthday parties, the failure to attend to others' faces was the single best discriminator between 1-year-olds with autism versus those with typical development (Osterling & Dawson, 1994).

Second, face recognition impairments have been found in many studies of older individuals with autism. Klin et al. (1999) found that elementary school-age children with autism scored lower on face recognition tests than developmentally disabled children without autism. Boucher and Lewis (1992) found that children with autism were impaired compared with typically developing children on both picture-matching and picture-recognition tasks, and Boucher, Lewis, and Collis (1998) found that children with autism performed worse on face-recognition tasks than did children with learning disabilities. Adolescents and adults with autism also show impaired face recognition (Cipolotti, Robinson, Blair, & Frith, 1999; Hauk, Fein, Maltby, Waterhouse, & Feinstein, 1998; Jambaque, Mottron, Ponsot, & Chivron, 1998; Ozonoff, Pennington, & Rogers, 1990; Teunissse & DeGelder, 1994). Hobson et al. (1988a) and Langdell (1978) examined whether older individuals with autism show the "face-inversion effect" that has been demonstrated in normal individuals; that is, a superior ability to recognize upright as compared with inverted faces. In both studies, individuals with autism recognized inverted faces better than normal control participants, which suggests that they are not using the configural approach for processing upright faces used by normal individuals.

Third, neural systems that mediate face recognition appear to exist very early in life, offering the possibility that face recognition impairment may be one of the earliest indicators of abnormal brain development in autism. In normal infancy, the face holds particular significance and provides nonverbal information important for communication and survival (Darwin, 1872/1965). Face recognition ability is present during the first 6 months of life. A visual preference for faces (Goren, Sarty, & Wu, 1975) and the capacity for very rapid face recognition (Walton & Bower, 1993) are present at birth. By 4 months, infants recognize upright faces better than upside down faces (Fagan, 1972), and at 6 months, infants show differential event-related brain potentials to familiar versus unfamiliar faces (de Haann & Nelson, 1997, 1999).

Much is known about the neural systems that subserve face recognition in adult humans and primates. In monkeys, face-selective neurons have been found in the inferior temporal areas, TEa and TEm; the superior temporal sensory area; the amygdala; the ventral striatum (which receives input from the amygdala); and the inferior convexity (Baylis, Rolls, & Leonard, 1987; Desimone, Albright, Gross, & Bruce, 1984; Leonard, Rolls, Wilson, & Baylis, 1985; O Scalaidhe, Wilson, & Goldman-Rakic, 1997; Rolls, 1984, 1992; Williams, Rolls, Leonard, & Stern, 1993; Wilson, O Scalaide, & Goldman-Rakic, 1993). In functional magnetic resonance imaging (fMRI; Gauthier, Tarr, Anderson, Skudlarski, & Gore, 1999; Kanwisher, McDermott, & Chun, 1997; McCarthy, Puce, Gore, & Allison, 1997) studies of face recognition the fusiform gyrus is activated, typically more on the right than on the left. A recent fMRI study of high-functioning individuals with autism and Asperger syndrome

(Schultz et al., 2000) showed a failure to activate the fusiform face area during face processing. Damage to fusiform gyrus and to amygdala results in impaired face recognition (Aggleton, 1992; Damasio, Damasio, & Van Hoesen, 1982). Parts of the inferior and medial temporal cortex may work together to process faces (Nelson, 2001). For example, the anterior inferior temporal cortex and the superior temporal sulcus project to the lateral nucleus of the amygdala (Aggleton, Burton, & Passingham, 1980; Amaral, Price, Pitkanen, & Carmichael, 1992), with the amygdala responsible for assigning affective significance to faces, and thus affecting both attention and mnemonic aspects of face processing.

Previous studies of face recognition in autism (reviewed above) have used older individuals and required verbal instructions and responses. Our intent was to study face recognition in autism with both verbal and nonverbal children at a young age to better determine when such impairments emerge. To achieve this goal, high-density event-related potential (ERP) recordings (Tucker, 1993) were utilized to examine electrical brain activity to familiar and unfamiliar faces and objects in 3- to 4-year-old children with autism spectrum disorder (ASD) and comparison groups of children with developmental delay (DD) and typical development. In addition to offering spatial resolution on the scalp that is superior to conventional ERP recording methods, the dense-array ERP method is completely noninvasive and relatively easy to apply. This is in contrast to other brain imaging techniques, such as positron emission tomography (PET) or fMRI, which require injection of radioactive substances or require children to remain motionless for long periods of time. These latter methods are extremely limited in their applicability to severely impaired or very young children and have inferior temporal resolution. The high-density ERP method involves simply laying a light net of wet electrodes on the child's head and requires no abrasion of the scalp to obtain 64 simultaneous channels of electroencephalogram (EEG) activity. This method has been successfully used to study localized brain activity during speech perception in infants as young as 2 months of age (Dehaene-Lambertz & Dehaene, 1994). We found that such a method is ideal for testing hypotheses regarding brain function in young, normally developing infants and children with autism.

In a series of studies of young infants, Nelson and colleagues (e.g., Nelson & Collins, 1991, 1992) reported that distinct ERP patterns could be invoked to both familiar and unfamiliar faces. de Haan and Nelson (1997, 1999) found differential ERPs to a highly familiar face (i.e., the infant's mother's face) versus a dissimilar-looking unfamiliar female face in studies with 6-month-old infants. In the present study, a similar procedure was utilized with young children with ASD and comparison groups to assess early recognition of familiar faces and objects.

Three ERP components were examined. First, in de Haan and Nelson's (1997, 1999) studies of face processing in 6-month-olds, an early sensory component was observed over occipital scalp locations. de Haan and Nelson (1999) found that this P400 component peaked earlier over posterior scalp locations for faces than for objects, suggesting a temporal advantage in processing faces over objects. Differences in P400 amplitude were not found for familiar versus unfamiliar stimuli, however. Second, de Haan and Nelson (1997, 1999) found that the Nc component was larger in response to familiar faces and objects as compared with unfamiliar faces and objects. The Nc is a middle-latency negative component, which is maximal over frontal midline electrodes and has been associated with increased attention to salient stimuli (Courchesne, 1978; Nelson, 1994), as well as with recognition memory (de Haan & Nelson, 1997, 1999; Nelson, 1994). Third, late slow-wave activity also differed in response to familiar versus unfamiliar stimuli in de Haan and Nelson's study (1999). Positive slow-wave (PSW) activity over frontal scalp locations was larger for unfamiliar than for familiar stimuli. The PSW has been associated with memory processes. Given that it is likely that 6-month-olds are able to recognize their mother's face, the increased PSW

activity to the stranger may reflect updating of the memory trace for the unfamiliar stimulus (Nelson, 1994).

METHODS

Participants

Three groups of children participated in the present study: (1) 63 children with ASD who had diagnoses of either Autistic Disorder or Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), (2) 27 children with developmental delay (DD) without ASD, and (3) 28 children with typical development. Participants were recruited from 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 ed. (DSM-IV; American Psychiatric Association, 1994). In addition, clinicians made a clinical judgment of diagnosis based on the presence and/or 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. Children with DD and typically developing children were administered the ADOS-G. These children did not meet criteria for Autistic Disorder or PDD-NOS on the ADOS-G or based on DSM-IV criteria, nor did they show elevated symptoms on these measures.

Children were provided with a series of training and desensitization sessions (described below) to increase compliance with the ERP procedures. Despite extensive training, noncompliance was not uncommon, and therefore, interpretable ERP data were available for only a subset of the sample tested. Attrition levels were similar to many ERP studies with normal infants.

For the face study, of the initial sample of 63 children with ASD, 34 children (20 with Autistic Disorder and 14 with PDD-NOS) provided adequate artifact-free data (18 were not compliant, 10 provided too few artifact-free trials, and 1 experienced an equipment malfunction). Of the initial sample of 27 children with DD, 16 provided adequate artifact-free data (2 were not compliant, 8 provided too few artifact-free trials, and 1 experienced an equipment malfunction). Of the initial sample of 28 children with typical development, 19 provided adequate, artifact-free data (8 provided too few artifact-free trials, and 1 experienced an equipment malfunction).

Table 1 presents demographic and descriptive information, including gender, ethnicity, 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 three groups of children included in the final sample for the face study. The three groups did not differ in terms of gender, ethnicity, SES, or chronological age. As expected, the typical development group had a significantly higher mental age than did the ASD group, $t = 8.1$, $p < .001$, and the DD group, $t = 7.4$, $p < .001$. The ASD and DD groups did not differ in terms of their mental age, $t = .3$, $p = .803$.

For the object study, of the initial sample of 63 children with ASD, 33 children (20 with Autistic Disorder and 13 with PDD-NOS) provided adequate, artifact-free data (18 were not compliant, and 12 provided too few artifact-free trials). Of the initial sample of 27 children with DD, 17 children provided adequate, artifact-free data (1 was not compliant, 7 provided too few artifact-free trials, and 2 experienced an equipment malfunction). Of the initial sample of 28 children with typical development, 21 provided adequate, artifact-free data (1 was not compliant, and 6 provided too few artifact-free trials).

Table 2 presents demographic and descriptive information, including gender, ethnicity, SES, chronological age, and Early Learning Composite mental age, for the three groups of children included in the final sample for the object study. The three groups did not differ in terms of gender, ethnicity, SES, or chronological age. As expected, the typical development group had a significantly higher mental age than did the ASD group, $t = 8.6$, $p < .001$, and the DD group, $t = 7.1$, $p < .001$. The ASD and DD groups did not differ in terms of their mental age, $t = .6$, $p = .544$.

Stimuli

Face stimuli: Each child's mother's face was photographed by color digital camera against a light gray background. Each mother wore a gray scarf to obscure her neck and clothing neckline. Earrings and other jewelry were removed. Mothers assumed a neutral facial expression.

The image of each mother's face was matched with another, dissimilar female face selected from mothers of other children who participated in the study. The experimenter selected the unfamiliar face stimulus so that paired faces were of the same ethnicity and faces of mothers who wore glasses were paired with faces of other mothers who wore glasses. Otherwise, paired faces were chosen to be dissimilar in terms of hair color, hair style, eye color, face shape, and facial features (e.g., size of nose).

Object stimuli: Each participant's parent brought the child's favorite toy to the session. The toys did not have faces visible when photographed. Each toy was digitally photographed against a gray background. Because the size of toys varied, images of toys were graphically manipulated so that the perceptual sizes of the stimuli on the monitor on which they were presented were approximately equivalent. Each toy image was matched with another color-digitized image of a toy selected from toys brought in by other participants.

The choice of the unfamiliar object was made by an experimenter based on the following criteria: Paired objects were from the same category (i.e., both were toys). The unfamiliar toy was similar to the participant's toy in shape, color, and size but had a different function (e.g., if the child's favorite toy was a vehicle, the control toy was chosen to be similar in size, shape, and color, but was not another vehicle). Each child's parent confirmed that the child was not familiar with the comparison object.

Procedure

Training: Prior to data collection, each child received up to seven behavioral training sessions to acclimate the child to the testing setting and apparatus. During each training session, the child sat on the parent's lap in the position in which data collection would occur. One experimenter provided the child with both social and edible reinforcement while a second experimenter touched the child's head with different objects for time periods of increasing duration. Training began with touching the child's head with a tape measure for 5 s. Subsequently, within and across sessions, the time duration was increased using the following objects: a dry towel, a damp towel, a dry "practice" sensor net, and a damp "practice" sensor net. This procedure served to desensitize the child to tactile stimulation of the scalp. The goal for termination of training was toleration of the damp "practice" sensor net for approximately 40 s.

Data collection: 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, Inc.; Tucker, 1993) was placed on the child's head and fitted according to manufacturer's specifications after being dipped into a potassium chloride 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. Impedences were kept below 40 kΩ.

The face and object recognition studies were presented in counterbalanced order; half of the children viewed the face stimuli first, the other half of the children viewed the object stimuli first. For each study, familiar and unfamiliar stimuli were presented in a pseudorandom order. The stimuli were displayed on a 17-inch (43 cm) Apple color monitor. The stimulus frames were 32 cm (520 pixels) wide × 24 cm (420 pixels) high. In the face recognition condition, the faces were fitted within the frame and were displayed at 18 cm from the top of the head to the chin and 11 cm from cheek to cheek with ±1-cm variance in each direction. For the object recognition study, the stimuli were fitted within the same size frame. A break of approximately 3 min separated the face and object recognition studies.

A baseline recording of 130 ms preceded stimulus onset, and the stimulus appeared on the screen for 500 ms. Event-related potential data were recorded for an additional 1200 ms following stimulus offset. The intertrial interval varied randomly between 500 and 1200 ms. Data collection was terminated when the child had attended to 50 of each of the familiar and unfamiliar stimuli, or when the child was no longer tolerant of the procedure. 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.

Electroencephalogram recording: The EEG from the 64 channels was registered continuously. The signal was amplified and filtered via a preamplifier system (Electrical Geodesics, Inc.). The amplification was set at 1000× and filtering was done through a .1 Hz high-pass filter and a 100 Hz elliptical low-pass filter. The conditioned signal was multiplexed and digitized at 250 samples per second via an Analog-to-Digital converter (National Instruments PCI-1200) positioned in an Apple Macintosh computer dedicated to data collection. Data were recorded continuously and streamed to the computer's hard disk. A second computer generated the stimuli. The two computers were interfaced via one of their serial ports for precise synchronization. The timing of the stimulus onset and offset were registered together with the physiological record for offline segmentation of the data.

Data were collected using the vertex electrode as a reference, and were re-referenced offline to an average mastoid reference.

Data editing and reduction: Data were averaged using the Electrical Geodesics, Inc. program Averager. Signals from electrode sites were marked for rejection if the weighted running average exceeded 150 micro-volts for transit and 250 microvolts 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 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 averaging.

Transformations were applied to averaged data to correct for baseline shifts and to digitally filter data (low-pass Butterworth 20 Hz) to reduce environmental noise artifact. In addition, an algorithm that derived values from the neighboring sites by spline interpolation was used to replace electrodes for which more than 25% of trials were rejected by artifact. Participants for whom more than 10 channels required this replacement were excluded from further analyses. For the face study, an average of 2.83 (SD = 2.48) channels per participant were replaced for the ASD group, an average of 2.63 (SD = 2.17) channels were replaced for the typical development control group, and an average of 3.0 (SD = 2.5) channels were replaced for the DD group. Paired t tests revealed no significant differences in the number of channels replaced between groups, all ps > .05. For the object study, an average of 2.94 (SD = 2.68) channels per participant were replaced for the ASD group, an average of 3.95 (SD = 2.46) channels were replaced for the typical development group, and an average of 3.18 (SD = 2.29) channels were replaced for the DD group. There were no significant differences in the number of channels replaced between groups, all ps > .05. There were also no differences within groups between the number of channels replaced in the face study and in the object study, all ps > .05. All participants whose data were included in the final sample had at least nine artifact-free trials in each condition. Table 3 depicts the number of participants and average number of trials in each condition for each group.

Data analysis: Time windows for the hypothesized components of interest were chosen by visual inspection of data from individual participants, which ensured that for each participant the component of interest was captured in the time window. The time intervals used were 194 to 590 ms for the Nc component, 286 to 610 ms for the P400 component, and 670 to 1670 ms for the PSW component. In addition, for each component, the electrodes over which the component was apparent were identified. Electrode groups were identified that included anterior and posterior midline and lateral scalp locations. The ERP data was averaged over these electrodes for each component. The placement of electrodes in the geodesic sensor net system, and the electrodes over which data were averaged for each component are shown in Figure 1.

For each component of interest, the overall ANOVAs that included group (ASD versus comparison group) as a between factor, and condition (familiar versus unfamiliar) and hemisphere as within-subject factors are described first. Because only a subsample of children had adequate artifact-free data for both the face and object studies, face and object data were analyzed separately. Comparisons between the ASD and typical development groups, and between the ASD and DD groups were calculated separately, because only children with autism who were also mentally retarded were included in these analyses. Dependent variables for analyses of the P400 and Nc components were peak amplitude and latency. The dependent variable for the PSW component was mean amplitude.

RESULTS

Event-Related Potentials to Faces

P400—As seen in Figure 2, the P400 to unfamiliar faces had a posterior distribution, which was comparable across the three groups. Table 4 is a summary table showing means and standard deviations for P400 amplitude for all groups. Analyses of variance comparing the ASD and typical development groups revealed, for midline P400 amplitude, a main effect of condition at lateral leads, $F(1, 51) = 4.51$, $p < .05$, and a Group × Condition interaction, $F(1, 51) = 6.03$, $p < .05$. Post hoc analyses were conducted to interpret the interaction effect, as shown in Table 4. Typically developing children showed a significantly more positive midline P400 amplitude to the unfamiliar than to the familiar face (see Figure 3A). Typically developing children also showed a larger lateral P400 amplitude to the unfamiliar face than to the familiar face, but this effect was only marginally significant. Children with ASD showed no significant differences in P400 amplitude to the familiar versus unfamiliar face at all scalp locations assessed (see Table 4 and Figure 4A). There were no differences in P400 latency to familiar versus unfamiliar faces for either group, all ps > .10. Analyses of variance comparing the ASD versus DD groups for P400 amplitude and latency yielded no significant main effects or interactions, all ps > .05.

Nc—As seen in Figure 2, Nc was present concurrently with P400, but was maximal at anterior locations. For the typical development and ASD groups, Nc was slightly right lateralized; for the DD group, Nc amplitude was maximal at anterior midline electrodes. Table 5 is a summary table showing means and standard deviations for Nc amplitude for all groups. Analyses of variance comparing the ASD and typical development groups revealed, for Nc lateral amplitude, main effects of condition, $F(1, 51) = 7.49$, $p < .01$, and hemisphere, $F(1, 51) = 11.62$, $p < .001$, and a significant Group × Condition interaction, $F(1, 51) = 4.59$, $p < .05$. Nc lateral amplitude was significantly more negative for unfamiliar than familiar stimuli, and was larger over the right as compared with the left hemisphere. Post hoc analyses were conducted to interpret the interaction effect. Typically developing children showed a significantly larger Nc amplitude to the unfamiliar than to the familiar face (see Table 5 and Figure 3A). Children with ASD showed no significant differences in Nc amplitude to the familiar versus unfamiliar face at all scalp locations assessed. Analyses of variance comparing the ASD versus DD groups for Nc amplitude yielded, at lateral leads, a main effect of hemisphere, $F(1, 48) = 10.0$, $p < .01$. Nc amplitude was significantly larger over the right as compared with the left hemisphere. Analyses of variance conducted on Nc latency yielded no significant main effects or interactions, all ps > .05.

Positive slow wave—Table 6 is a summary table showing means and standard deviations for PSW mean amplitude for all groups. Analyses of variance comparing PSW mean amplitude for the ASD and typical development groups revealed a significant main effect of hemisphere, $F(1, 51) = 7.21$, $p < .01$. Positive slow wave was more positive over the right than the left hemisphere. As shown in Table 6, post hoc analyses showed that typically developing children displayed a larger midline PSW mean amplitude to the unfamiliar than to the familiar face at the midline scalp locations, but this effect did not reach statistical significance (see Figure 3A). Typically developing children did not show a difference in PSW mean amplitude at the lateral scalp locations. Analyses of variance comparing the ASD versus DD groups for PSW mean amplitude revealed, at lateral leads, a significant main effect of hemisphere (right larger than left), $F(1, 48) = 4.73$, $p < .05$; a significant Condition × Hemisphere interaction, $F(1, 48) = 9.02$, $p < .01$; and a significant Condition × Hemisphere by group interaction, $F(1, 48) = 8.62$, $p < .01$. Post hoc analyses were conducted to interpret the three-way interaction. As shown in Table 6 and Figure 5A, children with DD showed a significantly larger midline PSW mean amplitude in response to the familiar than

to the unfamiliar face. Follow-up analyses revealed that for the DD group, there was no difference in the PSW mean amplitude over the right hemisphere, $t(15) = -.53$, $p = .60$. Over the left hemisphere, however, the PSW mean amplitude was larger for familiar than for unfamiliar faces, $t(15) = 4.88$, $p < .05$.

Event-Related Potentials to Objects

P400—As seen in Figure 6, the P400 to objects had a posterior distribution that was similar for the typical development and ASD groups, but was slightly more right lateralized for the DD group. Table 4 is a summary table showing means and standard deviations for P400 amplitude for all groups. Analyses of variance comparing the ASD and typical development groups revealed, at lateral leads, a significant main effect of condition, $F(1, 52) = 11.54$, $p < .001$, and a significant Hemisphere × Group interaction, $F(1, 52) = 5.73$, $p < .05$. As shown in Table 4 and Figure 4B, post hoc analyses showed that both children with ASD and those with typical development (Figure 3B) displayed significantly larger P400 amplitudes in response to the unfamiliar than to the familiar object. Analyses of variance comparing the ASD and DD groups revealed a significant Condition × Group interaction at both midline, $F(1, 48) = 4.3$, $p < .05$, and lateral, $F(1, 48) = 4.84$, $p < .05$, leads, and a significant effect of hemisphere at lateral leads, $F(1, 48) = 9.4$, $p < .01$. Post hoc analyses are shown in Table 4. As can be seen in Figure 5B, children with DD showed no significant differences in P400 amplitude, all ps > .10. Analyses of variance yielded no significant main effects or interaction related to P400 latency to familiar versus unfamiliar objects, all ps > .10.

Nc—As seen in Figure 6, Nc was present concurrently with P400, but was maximal at anterior locations. For the typical development and DD groups, Nc was slightly right lateralized; for the ASD group, the Nc distribution was centered at midline anterior sites. Table 5 is a summary table showing means and standard deviations for Nc amplitude for all groups. Analyses of variance comparing the ASD and typical development groups revealed significant main effects of condition at both midline, $F(1, 52) = 6.71$, $p < .05$, and lateral, $F(1, 52) = 8.67$, $p < .01$, leads; a significant effect of hemisphere at lateral leads only, $F(1, 52) = 16.6$, $p < .001$; and a significant Hemisphere × Group interaction, $F(1, 52) = 9.49$, $p < .05$. As shown in Table 5, post hoc analyses showed that both children with ASD and those with typical development displayed a larger Nc amplitude in response to the unfamiliar as compared with the familiar object (this only approached statistical significance for the ASD group). For the typical development group only, Nc was larger over the right than the left hemisphere. Analyses of variance comparing the ASD and DD groups yielded a main effect of hemisphere, $F(1, 48) = 9.4$, $p < .05$, and a Condition × Group interaction, $F(1, 48) = 5.67$, $p < .05$. As shown in Table 5, post hoc analyses showed that whereas children with autism displayed a larger Nc amplitude to unfamiliar than to familiar objects, children with DD did not show this difference. Analyses of variance revealed no main effects or interactions related to Nc latency, all ps > .10.

Positive slow wave—Table 6 is a summary table showing means and standard deviations for PSW mean amplitude for all groups. Analyses of variance comparing PSW mean amplitude for the ASD versus typical development groups revealed no significant main effects or interaction. Similar ANOVAs comparing the ASD versus DD groups revealed a significant effect of group, $F(1, 48) = 6.46$, $p < .05$; a significant effect of condition, $F(1, 48) = 8.14$, $p < .01$; and a significant Condition × Group interaction, $F(1, 48) = 11.72$, $p = .001$. Positive slow-wave amplitude was larger for unfamiliar than for familiar objects, and children with ASD displayed a larger PSW mean amplitude compared with children with DD. As shown in Table 6 and Figure 5B, post hoc analyses showed that children with DD displayed significant larger lateral PSW mean amplitude to unfamiliar than to familiar objects, whereas children with ASD did not show this difference.

Summary

Table 7 summarizes the results pertaining to ERP amplitude differences for unfamiliar versus familiar faces and objects for the three groups of children. Children with ASD failed to show differential ERPs to the face stimuli, but did show P400 and Nc amplitude differences to object stimuli. Typically developing children showed ERP amplitude differences in all three components (P400, Nc, PSW) for the face stimuli, and in the P400 and Nc components for the object stimuli. For the DD children, ERP amplitude differences were found in the PSW component for both faces and objects. For the ASD group for all three components, the standard deviations of the ERP amplitudes for faces were smaller than those for the objects. Thus, greater interindividual ERP variability for faces is not likely to be an explanation for this pattern of findings.

DISCUSSION

In the present study, both children with typical development and those with DD showed differential ERP responses to their mother's versus an unfamiliar face and to a favorite versus an unfamiliar object. Typically developing children showed significantly larger P400 and Nc amplitudes to the unfamiliar face as compared with their mother's face, and to a favorite object as compared with an unfamiliar object. In 6-month-olds, de Haan and Nelson (1999) also found Nc amplitude differences for mother's versus an unfamiliar face and for a familiar versus unfamiliar object, although the Nc was larger for the familiar stimuli. The age difference between the 3- to 4-year-olds in the present study and the 6-month-olds in the de Haan and Nelson study might explain this difference. Young infants might devote greater attention to their mother's face, whereas for the older children, the reverse might be true.

Children with DD showed a larger PSW amplitude to their mother's face as compared with the unfamiliar face, and to the unfamiliar object as compared with a favorite object. Although the ERP pattern was different for the children with typical development versus DD, both groups showed differential ERPs to the unfamiliar versus familiar faces and objects. It is notable that the children with typical development showed differential processing of the familiar versus unfamiliar stimuli at the early, perceptual stages of processing, whereas the children with DD showed differential processing only at the later stages of processing.

In contrast with children with typical development and DD, children with ASD did not show a differential brain electrical response to their mother's versus an unfamiliar face, but did show larger P400 and Nc amplitudes to the unfamiliar object as compared with a favorite object. In fact, their ERPs to objects were quite similar to those of the chronological age-matched typical children; that is, larger P400 and Nc amplitude to the unfamiliar object at the lateral scalp locations. Thus, like the typical development children, for objects, the children with ASD showed differential brain activity at the early stages of processing. This is interesting in light of the fact that these children were matched to those with DD in terms of overall mental age and IQ level. These data add to the growing body of evidence indicating a selective impairment in social processing in autism.

What might the fact that young children with autism do not show differential brain responses to their mother's versus an unfamiliar face mean? One possibility is that there exists a genetically determined specialized system for face processing (Farah, Rabinowitz, Quinn, & Liu, 2000; Farah, Wilson, Drain, & Tanaka, 1998) and that autism involves a genetic abnormality that affects this system. Newborns are capable of recognizing faces (Johnson, Dziurawiec, Ellis, & Morton, 1991; Pascalis, de Schonen, Morton, Deruelle, & Fabre-Grent, 1995; Simion, Valenza, Umilta, & Barba, 1998), suggesting that face recognition is a very early capability. There is evidence for very early specialization of right

fusiform gyrus for face processing. Mazoyer et al. (1999) used PET to study neurologically impaired infants while they were presented with faces versus nonface stimuli, and found activation of the right fusiform gyrus, among other areas of the brain.

On the other hand, as Nelson (2001) and others (Morton & Johnson, 1991) have argued, both the fragility and nature of the early face recognition abilities suggest that they are served by a different neural system than the system that emerges later in the first year of life. Morton and Johnson (1991) hypothesize that early face processing abilities are served by a subcortical neural system, which is replaced by a cortical system that emerges by 6 months of age. The latter is less fragile and more experience dependent. These changes in the face processing system may reflect “experience expectant developments” (Nelson, 2001); that is, a readiness of the brain to receive specific types of information from the environment (Greenough, Black, & Wallace, 1987). This readiness occurs during sensitive periods during which specific types of information are reliably present for most individuals. Exposure to faces is a reliable experience for most human infants, and likely facilitates development of a neural system that is specialized for faces. Nelson (1993) found human infants superior to adults in discriminating monkey faces, suggesting that experience with human faces results in a “perceptual narrowing” similar to what is observed with speech perception (Doupe & Kuhl, 1999). Gauthier et al. (1999) showed that increased expertise in object recognition is associated with increased activation of the fusiform gyrus, a region reliably activated by face processing. These studies suggest that specialization of the fusiform gyrus is influenced by both experience and expertise.

Experience may also play a role in abnormal development of the face processing system in autism (Carver & Dawson, in press). We hypothesize that the abnormalities in face processing found in autism may be related to abnormalities in social attention, and, more specifically, that the neural mechanisms that naturally draw the normal infant’s attention to the eyes are dysfunctional in autism. Such mechanisms normally facilitate mutual gaze and the acquisition of knowledge about others’ intentions and facial expressions. Beginning early in life, in autism there may be a deprivation of critical experience-driven input that results from a failure to pay normal attention to faces, particularly the eye region.

Behavioral studies suggest that the most salient parts for face recognition are, in order of importance, eyes, mouth, and nose (see the review in Shepherd, 1981). Studies utilizing intracranial ERPs to face stimuli found the amplitude of the face-specific ERP component to decrease in the same order (Allison, Puce, Spencer, & McCarthy, 1999; McCarthy, Puce, Belger, & Allison, 1999). Eye-scanning studies in humans (Yarbus, 1967) and monkeys (Nahm, Perret, Amaral, & Albright, 1997) show that eyes and hair/forehead are scanned more frequently than the nose. Human infants focus on the eyes rather than the mouth (Haith, Bergman, & Moore, 1979). Using eye-tracking technology to measure visual fixations, Klin (2001) recently reported that adults with autism show abnormal patterns of attention when viewing naturalistic social scenes. The patterns include reduced attention to the eyes and increased attention to mouths, bodies, and objects.

Although little is known about how autism is manifest in early infancy, studies based on home videotape observations suggest that 8- to 10-month-old infants with autism can be distinguished from typically developing infants by their failure to orient to social stimuli (Werner, Dawson, Osterling, & Dinno, 2000). Interestingly, in a case study of an infant whose development was monitored from birth to 2 years of age, it was found that this infant showed normal social responses before about 6 months of age, but failed to develop anticipatory and intentional social responses that typically emerge in the second half of the first year, such as engaging in reciprocal imitative play (Dawson, Osterling, Meltzoff, & Kuhl, 2000). Although no conclusions can be based on one case study, these observations

suggest that lack of normal social attention by children with autism reflects an impairment in a neural system that comes on line during the second half of the first year. Dawson and colleagues (Dawson, Carver, & McPartland, 2000a, 2000b) have suggested that this lack of intentional and anticipatory social attention is related to a fundamental difficulty in forming representations of the reward value of social stimuli. Representations regarding the anticipated reward value of a stimulus begin to motivate and direct attention by the second half of the first year of life (Ruff & Rothbart, 1996). Establishing such representations regarding the anticipated reward value for social stimuli may be challenging for children with autism because social reward feedback (e.g., a smile in response to a behavior) is less predictable and more variable compared with nonsocial reward feedback (e.g., a sound in response to pushing a button; Dawson & Lewy, 1989). Gergely and Watson (1999) showed that in contrast to typically developing infants and toddlers, children with autism show a strong preference for highly contingent, nonvariable (i.e., perfect rather than imperfect) contingency feedback. The normal infant's attention is drawn to the imperfect contingent feedback that is characteristic of social interactions, whereas the child with autism is drawn to the less variable feedback of nonsocial stimuli (Dawson & Lewy, 1989; Gergely & Watson, 1999). This might result in a lack of attention to social stimuli, including faces, thereby creating a kind of deprivation of normal learning experiences with faces.

It is likely that social reward plays an important role in the consolidation of memories for emotional experiences and for emotionally laden stimuli, such as faces and facial expressions. The amygdala is necessary for assessing the emotional significance (reward value) of a stimulus. There is increasing evidence that the amygdala plays a role in enhancing memory for emotional stimuli. The physiological arousal experienced in association with emotional events or stimuli may be an important component in an amygdala-based memory system (Phelps & Anderson, 1997). Patients with amygdala damage do not show typical differential forgetting curves for arousing and nonarousing stimuli (arousing stimuli are recalled better). These patients have been shown to have intact recall for words that were emotional in meaning, but not arousing as measured by skin conductance response (Phelps, LaBar, & Spencer, 1997). If there exists amygdala dysfunction in autism, this might contribute to an impairment in memory consolidation for social stimuli, such as faces and facial expressions.

It is currently unknown whether there is a critical period for the development of a specialized face recognition system during which exposure and attention to faces are critical for normal development. Given that autism typically is not recognized until age 3 to 4 years, many children with this disorder might not have the necessary early experience for normal development of face processing. Early detection of autism is critically important so that the secondary effects of this disability can be avoided (Dawson, Ashman, & Carver, 2000). It is unknown whether very early intervention would prevent the full manifestations of autism. Evidence suggests that intensive early behavioral intervention can have a substantial impact on the outcome of children with autism (Dawson & Osterling, 1997; Lovaas, 1987; Rogers, 1998). Such evidence, however, rests on few studies, and only one controlled study; these studies require replication with other samples. Behavioral and electrophysiological studies of typical children suggest a protracted developmental course of the face recognition system, with substantial changes in face processing occurring from infancy through adolescence (Carey, 1992; Taylor, McCarthy, Saliba, & Degiovanni, 1999). It is possible that intervention started during the early preschool period or even later could significantly alter the developmental course of face processing in children with autism.

In conclusion, the present study suggests that an impairment in face recognition in autism exists by 3 to 4 years of age. Whether such an impairment can serve as an early behavioral

marker of autism, and whether such an impairment can be avoided by very early intervention are questions that await future research.

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References

Aggleton, JP. The functional effects of amygdala lesions in humans: A comparison with findings from monkeys. In: Aggleton, JP., editor. The amygdala: Neurobiological aspects of emotion, memory, and mental dysfunction. New York: Wiley-Liss; 1992. p. 485-503.

Aggleton JP, Burton MJ, Passingham RE. Cortical and subcortical afferents to the amygdala of the rhesus monkey (*Macaca mulatta*). Brain Research. 1980; 190:347–368. [PubMed: 6768425]

Allison T, Puce A, Spencer DD, McCarthy G. Electrophysiological studies of human face perception. I: Potentials generated in the occipitotemporal cortex by face and non-face stimuli. Cerebral Cortex. 1999; 9:415–430. [PubMed: 10450888]

Amaral, DG.; Price, JL.; Pitkanen, A.; Carmichael, T. Anatomical organization of the primate amygdaloid complex. In: Aggleton, JP., editor. The amygdala: Neurobiological aspects of emotion, memory, and mental dysfunction. New York: Wiley-Liss; 1992. p. 1-66.

American Psychiatric Association.. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 1994.

Baron-Cohen, S.; Tager-Flusberg, H.; Cohen, D. Understanding other minds: Perspectives from autism. Oxford, U.K: Oxford University Press; 1993.

Baylis GC, Rolls ET, Leonard CM. Functional subdivisions of the temporal lobe neocortex. The Journal of Neuroscience. 1987; 7:330–342. [PubMed: 3819816]

Boucher J, Lewis V. Unfamiliar face recognition in relatively able autistic children. Journal of Child Psychology and Psychiatry. 1992; 33:843–859. [PubMed: 1634592]

Boucher J, Lewis V, Collis G. Familiar face and voice matching and recognition in children with autism. Journal of Child Psychology and Psychiatry. 1998; 39:171–181. [PubMed: 9669230]

Carey S. Becoming a face expert. Philosophical Transactions of the Royal Society of London. 1992; 335:95–103. [PubMed: 1348143]

Carver L, Dawson G. Evidence for an early impairment in neural systems for face recognition in autism. Molecular Psychiatry. in press.

Cipolotti L, Robinson G, Blair J, Frith U. Fractionation of visual memory: Evidence from a case with multiple neurodevelopmental impairments. Neuropsychologia. 1999; 37:455–465. [PubMed: 10215092]

Courchesne E. Neurophysiological correlates of cognitive development: Changes in long-latency event-related potentials from childhood to adulthood. Electroencephalography and Clinical Neurophysiology. 1978; 45:468–482. [PubMed: 81749]

Damasio AR, Damasio J, Van Hoesen GW. Prosopagnosia: Anatomic basis and behavioral mechanisms. Neurology. 1982; 32:331–341. [PubMed: 7199655]

Darwin, C. The expression of the emotions in man and animals. London, U.K: John Murray; 1965. (Original work published 1872)

Davies S, Bishop D, Manstead ASR, Tantam D. Face perception in children with autism and Asperger's syndrome. Journal of Child Psychology and Psychiatry. 1994; 35:1033–1057. [PubMed: 7995843]

Dawson G, Ashman S, Carver L. The role of early experience in shaping behavioral and brain development and its implications for social policy. Development and Psychopathology. 2000; 12:695–712. [PubMed: 11202040]

Dawson, G.; Carver, L.; McPartland, J. Role of ventromedial prefrontal dysfunction in the attentional impairments found in autism. Paper presented at the National Institutes of Health Conference on Attention and Intentionality in Autism; Newark, NJ. 2000a Mar.

Dawson, G.; Carver, L.; McPartland, J. Pre-frontal dysfunction in autism. Paper presented at the American Psychological Association meeting on Frontal Lobe Development; Philadelphia, PA. 2000b May.

Dawson, G.; Lewy, A. Arousal, attention and the socioemotional impairments of individuals with autism. In: Dawson, G., editor. Autism: Nature, diagnosis, and treatment. New York: Guilford Press; 1989. p. 49-74.

Dawson G, Meltzoff A, Osterling J, Brown E. Children with autism fail to orient to social stimuli. Journal of Autism and Developmental Disorders. 1998; 28:479–485. [PubMed: 9932234]

Dawson G, Meltzoff A, Osterling J, Rinaldi J. Neuropsychological correlates of early symptoms of autism. Child Development. 1998; 69:1277–1285.

Dawson, G.; Osterling, J. Early intervention in autism: Effectiveness and common elements of current approaches. In: Guralnick, M., editor. The effectiveness of early intervention: Second generation research. Baltimore, MD: Brookes; 1997. p. 307-326.

Dawson G, Osterling J, Meltzoff AN, Kuhl P. Case study of the development of an infant with autism from birth to 2 years of age. Journal of Applied Developmental Psychology. 2000; 21:299–313.

Dawson G, Toth K, Abbott R, Osterling J, Munson J, Estes A. Defining the early social attention impairments in autism: Social orienting, joint attention, and responses to emotions. 2002 Manuscript submitted for publication.

de Haan M, Nelson CA. Recognition of the mother's face by 6-month-old infants: A neurobehavioral study. Child Development. 1997; 68:187–210. [PubMed: 9179998]

de Haan M, Nelson CA. Electrocortical correlates of face and object recognition by 6-month-old infants. Developmental Psychology. 1999; 35:1113–1121. [PubMed: 10442879]

Dehaene-Lambertz G, Dehaene S. Speed and cerebral correlates of syllable discrimination in infants. Nature. 1994; 370:292–295. [PubMed: 8035876]

Desimone R, Albright TD, Gross CG, Bruce C. Stimulus-selective properties of inferior temporal neurons in the macaque. Journal of Neuroscience. 1984; 4:2051–2062. [PubMed: 6470767]

Doupe AJ, Kuhl PK. Birdsong and human speech: Common themes and mechanisms. Annual Review of Neuroscience. 1999; 22:567–631.

Fagan J. Infants' recognition memory for face. Journal of Experimental Child Psychology. 1972; 14:453–476. [PubMed: 4676459]

Farah MJ, Rabinowitz C, Quinn GE, Liu GT. Early commitment of neural substrates for face recognition. Cognitive Neuropsychology. 2000; 17:117–123. [PubMed: 20945175]

Farah MJ, Wilson KD, Drain JM, Tanaka JR. The inverted face inversion effect in prosopagnosia: Evidence for mandatory, face-specific perceptual mechanisms. Visual Research. 1995; 14:2089–2093.

Gauthier I, Tarr MJ, Anderson AW, Skudlarski P, Gore JC. Activation of the middle fusiform "face area" increases with expertise in recognizing novel objects. Nature Neuroscience. 1999; 2:568–573.

Gergely, G.; Watson, JS. Early socio-emotional development: Contingency perception and the social-biofeedback model. In: Rochate, P., editor. Early social cognition: Understanding others in the first months of life. Mahwah, NJ: Erlbaum; 1999. p. 101-136.

Goren C, Sarty M, Wu P. Visual following and pattern discrimination of face-like stimuli by newborn infants. Pediatrics. 1975; 56:544–549. [PubMed: 1165958]

Greenough WT, Black JE, Wallace CS. Experience and brain development. Child Development. 1987; 58:539–559. [PubMed: 3038480]

Haith MM, Bergman T, Moore MJ. Eye contact and face scanning in early infancy. Science. 1979; 198:853–854. [PubMed: 918670]

Hauk M, Fein D, Maltby N, Waterhouse L, Feinstein C. Memory for faces in children with autism. Child Neuropsychology. 1998; 4:187–198.

Hobson P, Ouston J, Lee A. What's in a face? The case of autism. British Journal of Psychology. 1988a; 79:441–453. [PubMed: 3208000]

Hobson P, Ouston J, Lee A. Emotion recognition in autism: Coordinating faces and voices. Psychological Medicine. 1988b; 18:911–923. [PubMed: 3270834]

Hollingshead, AB. Unpublished manuscript. Yale University; 1975. Form Factor Index of Social Status.

Jambaque I, Mottron L, Ponsot G, Chiron C. Autism and visual agnosia in a child with right occipital lobectomy. Journal of Neurology, Neurosurgery, and Psychiatry. 1998; 65:555–560.

Johnson MH, Dziurawiec S, Ellis J, Morton J. Newborns' preferential tracking of face-like stimuli and its subsequent decline. Cognition. 1991; 40:1–19. [PubMed: 1786670]

Kanwisher N, McDermott J, Chun MM. The fusiform face area: A module in human extrastriate cortex specialized for face perception. Journal of Neuroscience. 1997; 17:4302–4311. [PubMed: 9151747]

Klin, A. Eye-tracking of social stimuli in adults with autism. Paper presented at the meeting of the NICHD Collaborative Program of Excellence in Autism; New Haven, CT: Yale University; 2001 May.

Klin A, Sparrow SS, deBildt A, Cicchetti DV, Cohen DJ, Volkmar FR. A normed study of face recognition in autism and related disorders. Journal of Autism and Developmental Disorders. 1999; 29:499–508. [PubMed: 10638462]

Langdell T. Recognition of faces: An approach to the study of autism. Journal of Child Psychology and Psychiatry. 1978; 19:255–268. [PubMed: 681468]

Leonard CM, Rolls ET, Wilson FAW, Baylis GC. Neurons in the amygdala of the monkey with responses selective for faces. Behavioral Brain Research. 1985; 15:159–176.

Lord C, Rutter M, Le Couteur A. Autism Diagnostic Interview–Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. Journal of Autism and Developmental Disorders. 1994; 24:659–685. [PubMed: 7814313]

Lord C, Rutter ML, Goode S, Heemsbergen J. Autism Diagnostic Observation Schedule: A standardized observation of communicative and social behavior. Journal of Autism and Developmental Disorders. 1989; 19:185–212. [PubMed: 2745388]

Lovaas OI. Behavioral treatment and normal educational and intellectual functioning in young autistic children. Journal of Consulting and Clinical Psychology. 1987; 55:3–9. [PubMed: 3571656]

Mazoyer N, de Schonen S, Quinton O, Crivello F, Reutter B, Mazoyer B. Functional anatomy of face processing in two-month old alert children. Neuro-Image. 1999; 9:S346.

McCarthy G, Puce A, Belger A, Allison T. Electrophysiological studies of human face perception. II: Response properties of face-specific potentials generated in occipitotemporal cortex. Cerebral Cortex. 1999; 9:431–444. [PubMed: 10450889]

McCarthy G, Puce A, Gore JC, Allison T. Face-specific processing in the human fusiform gyrus. Journal of Cognitive Neuroscience. 1997; 8:605–610.

Morton J, Johnson MH. CONSPEC and CONLERN: A two-process theory of infant face recognition. Psychological Review. 1991; 2:164–181. [PubMed: 2047512]

Mullen, EM. Mullen Scales of Early Learning. Los Angeles, CA: Western Psychological Services; 1997.

Mundy P, Sigman M, Ungerer J, Sherman T. Defining the social deficits of autism: The contribution of nonverbal communication measures. Journal of Child Psychology and Psychiatry. 1986; 27:657–669. [PubMed: 3771682]

Nahm FKD, Perret A, Amaral D, Albright TD. How do monkeys look at faces? Journal of Cognitive Neuroscience. 1997; 9:611–623.

Nelson, CA. The recognition of facial expressions in infancy: Behavioral and electrophysiological correlates. In: de Boysson-Bardies, B.; de Schonen, S.; Jusczyk, P.; MacNeilage, P.; Morton, J., editors. Developmental neurocognition: Speech and face processing in the first year of life. Hingham, MA: Kluwer Academic Press; 1993. p. 187-193.

Nelson, CA. Neural correlates of recognition memory in the first postnatal year. In: Dawson, G.; Fisher, KW., editors. Human behavior and the developing brain. New York: Guilford Press; 1994. p. 269-313.

Nelson CA. The development and neural bases of face recognition. Infant and Child Development. 2001; 10:3–18.

Nelson CA, Collins PF. Event-related potential and looking time analysis of infants' responses to familiar and novel events: Implications for visual recognition memory. Developmental Psychology. 1991; 27:50–58.

Nelson CA, Collins PF. Neural and behavioral correlates of recognition memory in 4- and 8-month-old infants. Brain and Cognition. 1992; 19:105–121. [PubMed: 1605948]

O'Scalaidhe SP, Wilson FAW, Goldman-Rakic PS. Areal segregation of face-processing neurons in prefrontal cortex. Science. 1997; 278:1135–1138. [PubMed: 9353197]

Osterling J, Dawson G. Early recognition of children with autism: A study of first birthday home videotapes. Journal of Autism and Developmental Disorders. 1994; 24:247–257. [PubMed: 8050980]

Ozonoff S, Pennington B, Rogers S. Executive function deficits in high-functioning autistic individuals: Relationship to theory-of-mind. Journal of Child Psychology and Psychiatry. 1990; 32:1081–1105. [PubMed: 1787138]

Pascalis O, de Schonen S, Morton J, Deruelle C, Fabre-Grent M. Mother's face recognition by neonates: A replication and extension. Infant Behavior and Development. 1995; 18:79–95.

Phelps EA, Anderson AK. Emotional memory: What does the amygdala do? Current Biology. 1997; 7:311–314.

Phelps EA, LaBar KS, Spencer DD. Emotional memory following unilateral temporal lobectomy. Brain Cognition. 1997; 34:512–521.

Rogers S. Empirically supported treatment for young children with autism. Journal of Clinical Child Psychology. 1998; 27:168–179. [PubMed: 9648034]

Rolls ET. Neurons in the cortex of the temporal lobe and in the amygdala of the monkey with responses selective for faces. Human Neurobiology. 1984; 3:209–222. [PubMed: 6526707]

Rolls, ET. Neurophysiology and functions of the primate amygdala. In: Aggleton, JP., editor. The amygdala: Neurobiological aspects of emotion, memory, and mental dysfunction. New York: Wiley-Liss; 1992. p. 143-165.

Ruff, HA.; Rothbart, MK. Attention in early development: Themes and variations. New York: Oxford University Press; 1996.

Schultz RT, Gauthier I, Klin A, Fulbright RK, Anderson AW, Volkmar FR, Skudlarski P, Lacadie C, Cohen DJ, Gore JC. Abnormal ventral temporal cortical activity during face discrimination among individuals with autism and Asperger syndrome. Archives of General Psychiatry. 2000; 57:331–340. [PubMed: 10768694]

Shepherd, J. Social factors in face recognition. In: Davies, G.; Ellis, H.; Shepherd, J., editors. Perceiving and remembering faces. London: Academic Press; 1981. p. 55-79.

Simion F, Valenza E, Umilta C, Barba DB. Preferential orienting to faces in newborns: A temporal-nasal asymmetry. Journal of Experimental Psychology: Human Perception and Performance. 1998; 24:1399–1405. [PubMed: 9778830]

Smith IM, Bryson SE. Imitation and action in autism: A critical review. Psychology Bulletin. 1994; 116:259–273.

Taylor MK, McCarthy G, Saliba E, Degiovanni E. ERP evidence of developmental changes in processing of faces. Clinical Neurophysiology. 1999; 110:910–915. [PubMed: 10400205]

Teunisse J, DeGelder B. Do autistics have a generalized face processing deficit? International Journal of Neuroscience. 1994; 77:1–10. [PubMed: 7989155]

Tucker DM. Spatial sampling of head electrical fields: The geodesic sensor net. Electroencephalography and Clinical Neurophysiology. 1993; 87:154–163. [PubMed: 7691542]

Walton GE, Bower TGR. Newborns form "prototypes" in less than 1 minute. Psychological Science. 1993; 4:203–205.

Werner E, Dawson G, Osterling J, Dinno J. Recognition of autism before 1 year of age: A retrospective study based on home videotapes. Journal of Autism and Developmental Disorders. 2000; 30:157–162. [PubMed: 10832780]

Williams GV, Rolls ET, Leonard CM, Stern C. Neuronal responses in the ventral striatum of the behaving macaque. Behavioral Brain Research. 1993; 55:243–252.

Wilson FAW, O'Scalaidhe SP, Goldman-Rakic PS. Dissociation of object and spatial processing domains in primate prefrontal cortex. Science. 1993; 260:1955–1958. [PubMed: 8316836]

Yarbus, AL. Eye movements and vision. New York: Plenum Press; 1967.

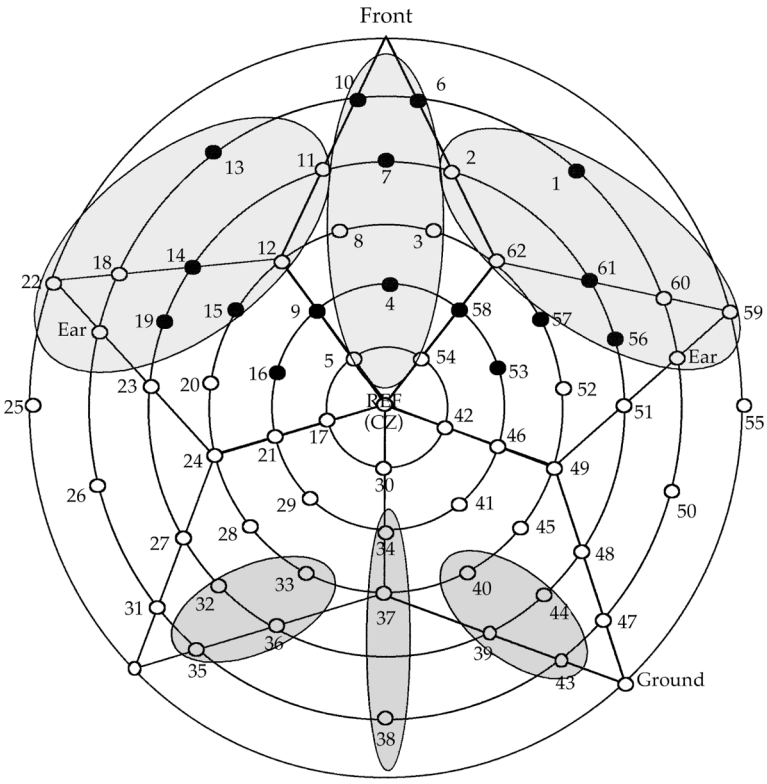

Figure 1.

Electrode groups over which data were averaged for each component (reference electrode during recording at location Cz). The Nc component is shown in the light-shaded areas (top). The P400 component is shown in the dark-shaded areas (bottom). Electrodes that are shaded black indicate the slow-wave component.

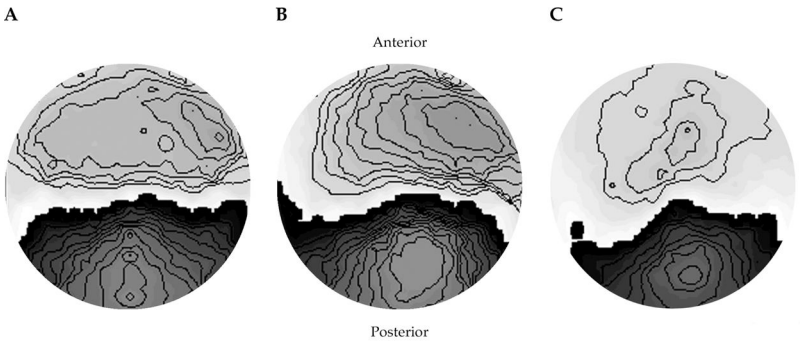

Figure 2.
Voltage maps of event-related potentials to unfamiliar faces at 450 ms for children with (A) autism spectrum disorder, (B) typical development, and (C) developmental delay.

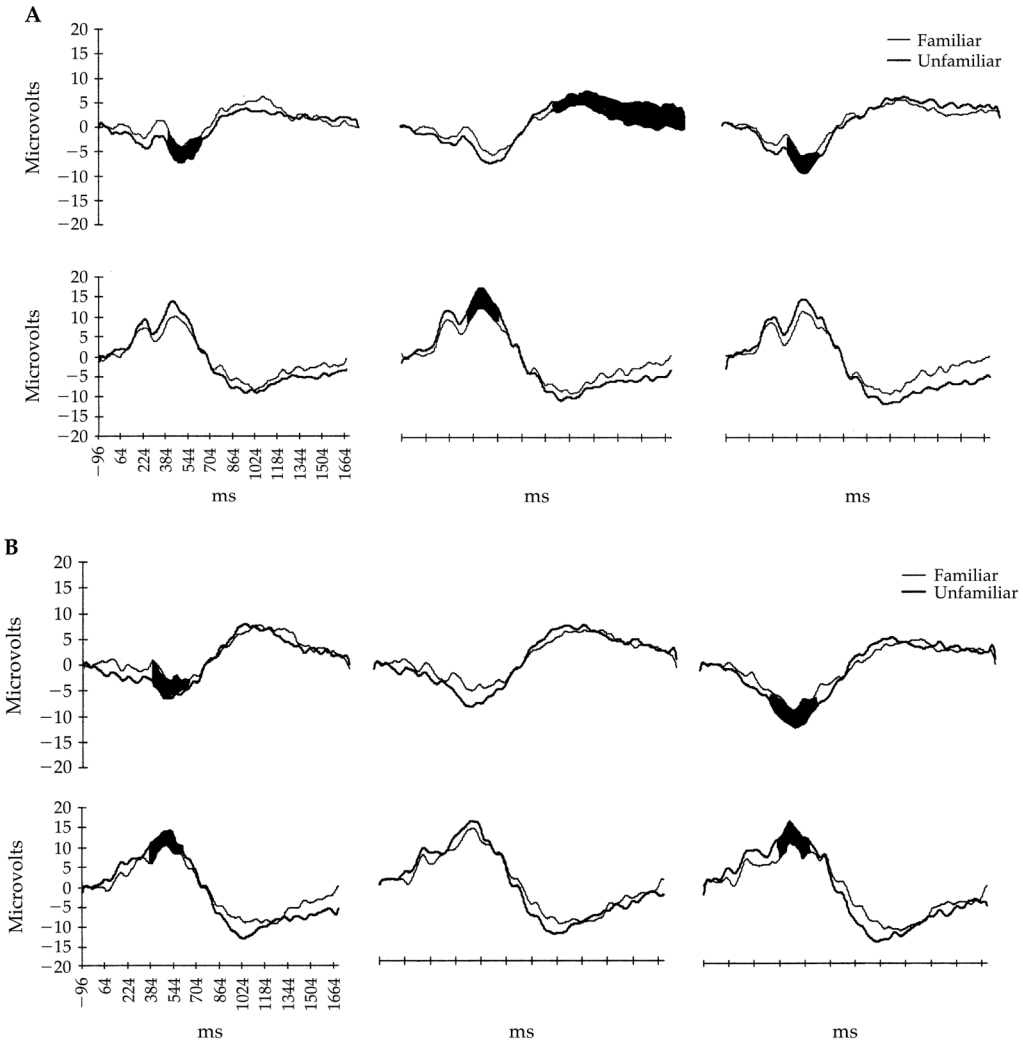

Figure 3.

Averaged event-related potential waveforms at the anterior (top) and posterior (bottom), right hemisphere, midline, and left hemisphere scalp locations for familiar and unfamiliar (A) faces and (B) objects for children with typical development. Areas in which significant differences were found for familiar versus unfamiliar stimuli are shaded in black.

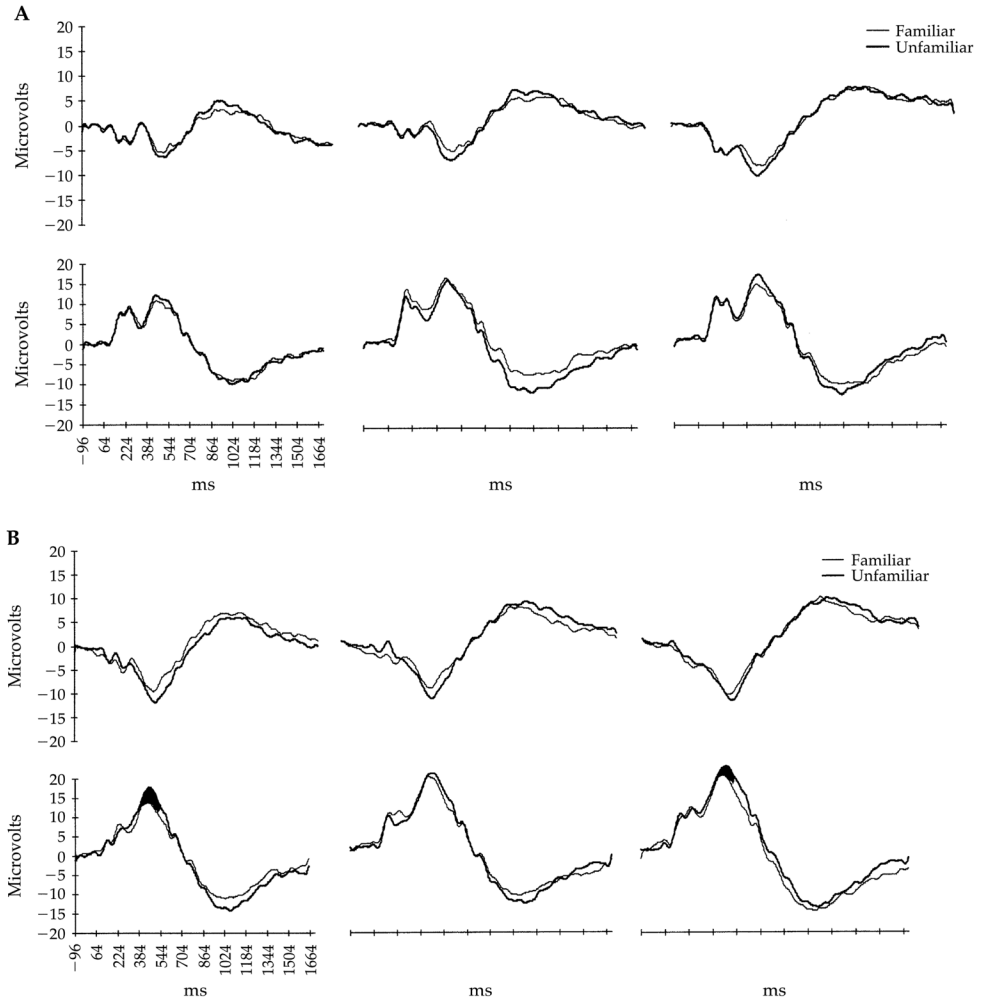

Figure 4.

Averaged event-related potential waveforms at the anterior (top) and posterior (bottom), right hemisphere, midline, and left hemisphere scalp locations for familiar and unfamiliar (A) faces and (B) objects for children with autism spectrum disorder. Areas in which significant differences were found for familiar versus unfamiliar stimuli are shaded in black.

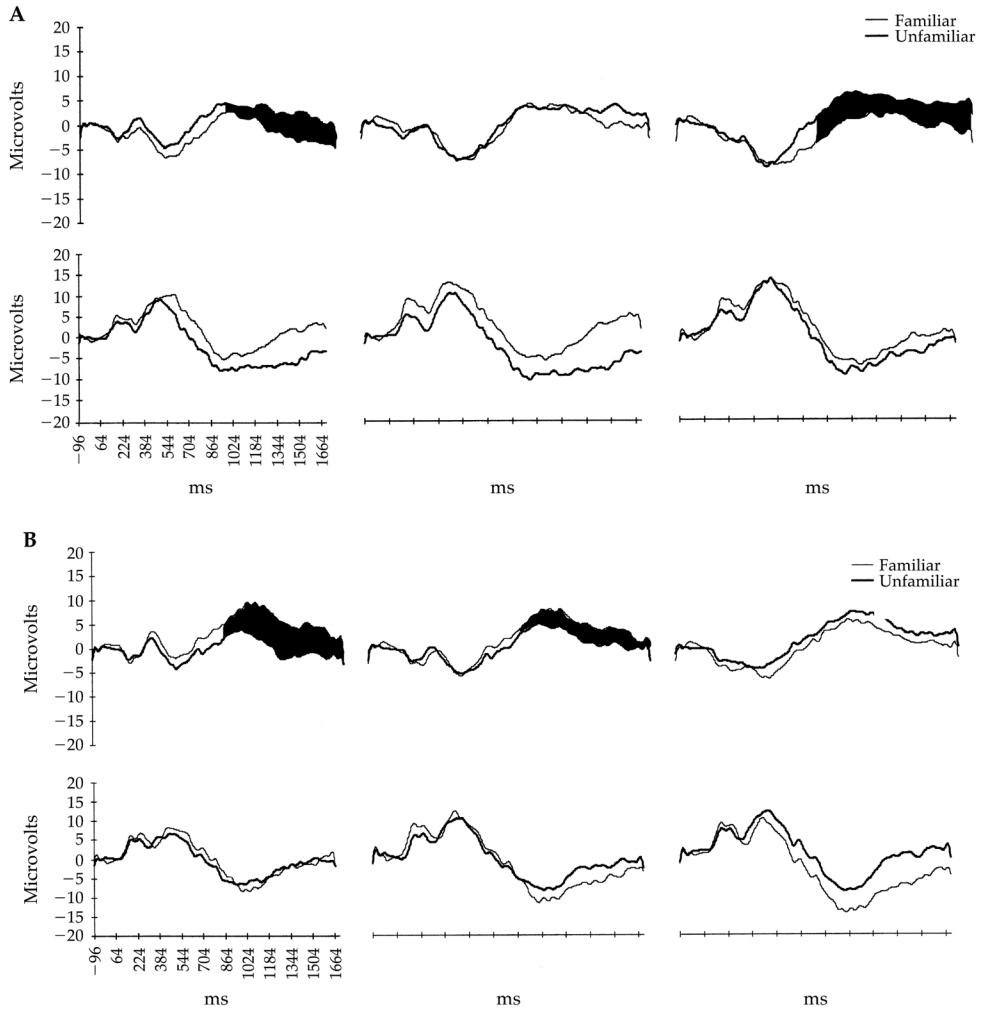

Figure 5.

Averaged event-related potential waveforms at the anterior (top) and posterior (bottom), right hemisphere, midline, and left hemisphere scalp locations for familiar and unfamiliar (A) faces and (B) objects for children with developmental delay. Areas in which significant differences were found for familiar versus unfamiliar stimuli are shaded in black.

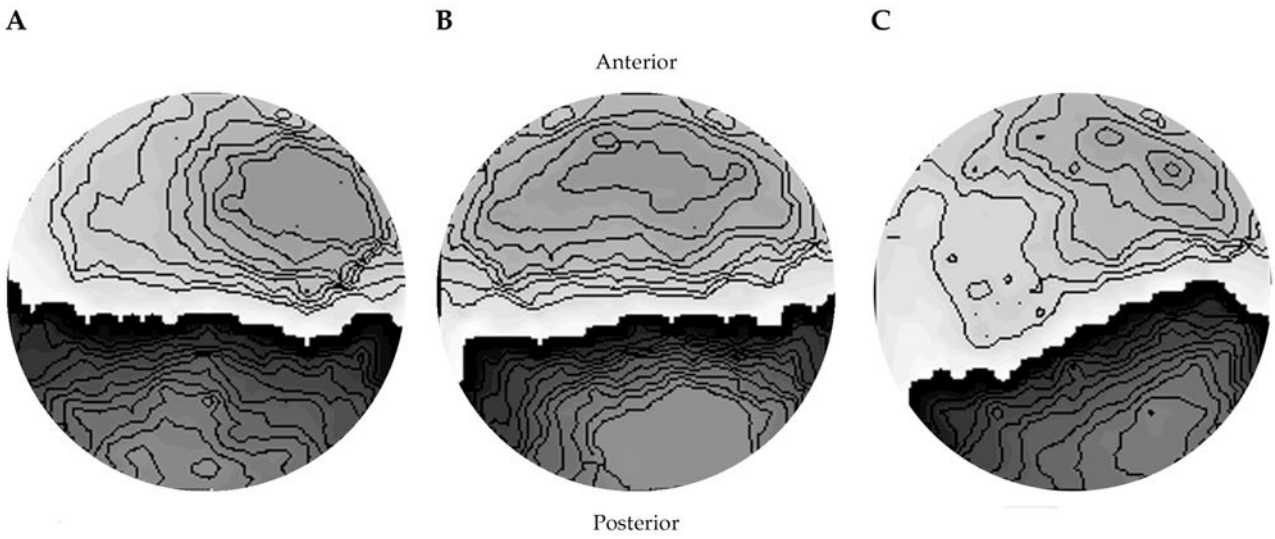

Figure 6.
Voltage maps of event-related potentials to unfamiliar objects at 490 ms for children with (A) autism spectrum disorder, (B) typical development, and (C) developmental delay.

Table 1

Face Study: Participant Descriptive Information

				Chronological Age (months)			Composite Mental Age (months)		
Group	***N*, Male: Female**	**Ethnicity**	**Socioeconomic Status (*SD*)**	**Minimum**	**Maximum**	***M* (*SD*)**	**Minimum**	**Maximum**	***M* (*SD*)**
Autism spectrum disorder	34, 30:4	25 White 9 Other	46.2 (12.6)	34.0	52.0	44.2 (4.2)	14.8	46.8	28.1 (9.6)
Typical development	19, 17:2	17 White 2 Other	53.1 (7.9)	34.0	55.0	45.4 (6.2)	39.0	58.5	48.4 (6.8)
Developmental delay	16, 10:6	9 White 7 Other	48.5 (11.0)	37.0	53.0	44.8 (4.9)	12.3	42.8	28.8 (8.9)

Table 2

Object Study: Participant Descriptive Information

				Chronological Age (months)			Composite Mental Age (months)		
Group	***N*, Male: Female**	**Ethnicity**	**Socioeconomic Status (*SD*)**	**Minimum**	**Maximum**	***M* (*SD*)**	**Minimum**	**Maximum**	***M* (*SD*)**
Autism spectrum disorder	33, 29:4	24 White 9 Other	47.6 (13.3)	34.0	50.0	43.5 (4.3)	14.8	46.8	28.6 (9.6)
Typical development	21, 18:3	19 White 2 Other	52.3 (8.6)	35.0	54.0	45.6 (5.8)	39.0	64.3	50.0 (7.6)
Developmental delay	17, 11:6	11 White 6 Other	47.7 (13.8)	37.0	53.0	45.1 (4.9)	12.3	42.8	30.4 (9.6)

Table 3

Average Trials by Condition

	Face Study				Object Study			
	Familiar Stimulus		Unfamiliar Stimulus		Familiar Stimulus		Unfamiliar Stimulus	
Group	***M* (*N*)**	***SD***	***M* (*N*)**	***SD***	***M* (*N*)**	***SD***	***M* (*N*)**	***SD***
Autism spectrum disorder	28.3 (34)	10.3	30.2 (34)	11.6	28.5 (33)	11.3	28.8 (33)	10.6
Typical development	34.7 (19)	11.4	35.2 (19)	10.8	28.6 (21)	11.1	28.7 (21)	13.3
Developmental delay	25.9 (16)	10.3	24.1 (16)	7.6	22.6 (17)	9.2	22.9 (17)	10.3

Table 4

Peak Amplitude in Microvolts of the P400 Component

Stimuli	Location	Condition	*M* (*SD*)	*F*	*p*
Autism spectrum disorder					
Face	Midline	Familiar	16.36 (9.73)	1.07	*ns*
		Unfamiliar	15.26 (11.56)		
	Lateral	Familiar	13.84 (9.68)	1.04	*ns*
		Unfamiliar	14.88 (11.73)		
Object	Midline	Familiar	17.10 (13.21)	.50	*ns*
		Unfamiliar	18.00 (11.48)		
	Lateral	Familiar	16.74 (12.03)	5.33	<.05
		Unfamiliar	19.23 (11.10)		
Typical development					
Face	Midline	Familiar	12.72 (5.93)	5.13	<.05
		Unfamiliar	16.01 (6.74)		
	Lateral	Familiar	11.27 (5.78)	4.05	.06
		Unfamiliar	13.71 (5.60)		
Object	Midline	Familiar	14.38 (10.74)	.76	*ns*
		Unfamiliar	15.85 (7.31)		
	Lateral	Familiar	12.65 (7.62)	7.14	<.01
		Unfamiliar	15.70 (6.75)		
Developmental delay					
Face	Midline	Familiar	10.41 (9.56)	.04	*ns*
		Unfamiliar	9.87 (9.65)		
	Lateral	Familiar	9.92 (7.64)	.03	*ns*
		Unfamiliar	10.25 (8.15)		
Object	Midline	Familiar	15.90 (14.11)	3.67	.07
		Unfamiliar	12.04 (11.46)		
	Lateral	Familiar	14.85 (11.14)	1.10	*ns*
		Unfamiliar	13.27 (10.82)		

Table 5

Peak Amplitude in Microvolts of the Nc Component

Stimuli	Location	Condition	*M* (*SD*)	*F*	*p*
Autism spectrum disorder					
Face	Midline	Familiar	−8.72 (7.42)	.29	*ns*
		Unfamiliar	−9.12 (6.39)		
	Lateral	Familiar	−9.92 (6.04)	.31	*ns*
		Unfamiliar	−10.22 (5.93)		
Object	Midline	Familiar	−10.70 (7.13)	3.24	.08
		Unfamiliar	−11.93 (6.63)		
	Lateral	Familiar	−11.41 (7.24)	3.84	.06
		Unfamiliar	−12.65 (6.35)		
Typical development					
Face	Midline	Familiar	−7.62 (3.82)	1.89	*ns*
		Unfamiliar	−9.26 (4.98)		
	Lateral	Familiar	−7.76 (4.19)	6.83	<.05
		Unfamiliar	−10.23 (4.58)		
Object	Midline	Familiar	−9.65 (3.41)	3.71	.07
		Unfamiliar	−11.18 (3.88)		
	Lateral	Familiar	−9.53 (4.68)	4.19	.05
		Unfamiliar	−11.70 (5.47)		
Developmental delay					
Face	Midline	Familiar	−7.60 (5.23)	.071	*ns*
		Unfamiliar	−7.37 (4.52)		
	Lateral	Familiar	−7.64 (5.46)	.26	*ns*
		Unfamiliar	−7.03 (4.84)		
Object	Midline	Familiar	−9.90 (7.13)	.07	*ns*
		Unfamiliar	−10.23 (6.63)		
	Lateral	Familiar	−10.68 (6.89)	2.16	*ns*
		Unfamiliar	−9.22 (5.74)		

Table 6

Mean Amplitude in Microvolts of the Positive Slow-Wave Component

Stimuli	Location	Condition	*M* (*SD*)	*F*	*p*
Autism spectrum disorder					
Face	Midline	Familiar	2.41 (5.08)	.01	*ns*
		Unfamiliar	2.29 (5.79)		
	Lateral	Familiar	1.72 (4.94)	.04	*ns*
		Unfamiliar	1.85 (4.23)		
Object	Midline	Familiar	3.98 (4.35)	.13	*ns*
		Unfamiliar	4.29 (4.68)		
	Lateral	Familiar	3.41 (4.30)	.25	*ns*
		Unfamiliar	3.06 (4.75)		
Typical development					
Face	Midline	Familiar	1.89 (4.31)	3.45	.08
		Unfamiliar	5.01 (5.92)		
	Lateral	Familiar	2.57 (3.92)	.002	*ns*
		Unfamiliar	2.54 (4.34)		
Object	Midline	Familiar	3.86 (6.41)	.007	*ns*
		Unfamiliar	4.05 (6.21)		
	Lateral	Familiar	2.55 (4.21)	.16	*ns*
		Unfamiliar	2.99 (4.54)		
Developmental delay					
Face	Midline	Familiar	4.81 (8.05)	4.87	<.05
		Unfamiliar	1.76 (4.75)		
	Lateral	Familiar	3.27 (4.81)	2.43	*ns*
		Unfamiliar	1.21 (4.85)		
Object	Midline	Familiar	1.36 (5.66)	.05	*ns*
		Unfamiliar	.94 (6.13)		
	Lateral	Familiar	−.71 (5.42)	13.82	<.01
		Unfamiliar	3.15 (5.34)		

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Table 7

Summary of Results for the P400, Nc, and PSW Components

	Face recognition			Object recognition		
Group	**P400**	**Nc**	**Slow Wave**	**P400**	**Nc**	**Slow Wave**
Autism spectrum disorder	—	—	—	*	+	—
Typical development	*	*	+	**	*	—
Development delay	—	—	*	+	—	**

* $p < .05$;

** $p < .01$;

+ $p < .10$.