

ERP Evidence of Atypical Face Processing in Young Children with Autism

Sara J. Webb · Geraldine Dawson · Raphael Bernier · Heracles Panagiotides

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Abstract Autism involves a basic impairment in social cognition. This study investigated early stage face processing in young children with autism by examining the face-sensitive early negative event-related brain potential component in 3–4 year old children with autism spectrum disorder (ASD), typical development, and developmental delay. Results indicated that children with ASD showed a slower electrical brain response to faces and a larger amplitude response to objects compared to children with typical development and developmental delay. These findings indicate that children with ASD have a disordered pattern of brain responses to faces and objects at an early age.

Keywords Autism · Event-related potentials · Faces · N170 · Children

Introduction

Recent evidence suggests that autism is a disorder characterized by impairments in face recognition (Dawson et al., 2002) and decreased attention to faces (Osterling &

Dawson, 1994, Osterling, Dawson, & Munson 2002). Several investigators have theorized that a lack of social motivation or social interest may reduce attention to faces early in development (e.g., Carver & Dawson, 2002; Klin et al., 1999). Further, aspects of face processing, such as emotion perception and eye gaze detection, are critical to the development of social relationships and theory of mind (e.g., Baron-Cohen, 1995).

Beginning during the neonatal period, infants demonstrate a preference for faces over other stimuli (Goren, Sarty & Wu, 1975; Johnson, Dziurawiec, Ellis, & Morton, 1991; Valenza, Simion, Cassia, & Umiltà, 1996) and prefer to look at their mother's face versus other female faces (Bushnell, Sai & Mullin, 1989). By 4 months of age, infants begin to show the inversion effect, better processing of upright faces compared with inverted faces (Fagan, 1972); by 6 months of age, they show differential event-related brain potentials to familiar versus unfamiliar faces (de Haan & Nelson, 1997) as well as to familiar and unfamiliar objects (de Haan & Nelson, 1999); and by 7 months infants are able to differentiate between a negative and neutral facial expression (Nelson & De Haan, 1996). While less is known about the development of face processing during the toddler period, Carver et al. (2003) found that ERP responses to familiar and novel faces shifted in response between 18 and 54 months, with the younger children demonstrating greater amplitude to the familiar face and older children demonstrating greater amplitude to the novel face. These results were interpreted as age-related differences in the perceived salience of the face of the primary caregiver versus and unfamiliar face.

Many of the early social impairments in autism, such as eye contact, joint attention, responses to emotional displays, and face recognition, involve the ability to attend to and process information from faces. Face processing

S. J. Webb (✉)
Psychiatry and Behavioral Sciences, Box 357920 CHDD,
Seattle, WA 98195, USA
e-mail: sjwebb@u.washington.edu

G. Dawson · H. Panagiotides
Center on Human Development and Disability, University of
Washington, Box 357920, Seattle, WA 98195, USA

R. Bernier · G. Dawson
Department of Psychology, University of Washington, Box
357920, Seattle, WA 98195, USA

abnormalities may be one of the earliest identifiable markers of autism (Dawson, Webb, & McPartland, 2005) as face processing and recognition are early developing abilities. Autism typically is not diagnosed until about 3 years of age, although researchers are working to identify children by 15–18 months (Filipek et al., 1999). Because there exist few observations of infants with autism, very little is known about the early development of face processing abilities of young children with autism. In a retrospective study using videotapes of first birthday parties, the single best discriminator between infants who were later diagnosed with an autism spectrum disorder versus those with typical development was the failure to look at others (Osterling & Dawson, 1994; also see Adrien et al., 1991). The toddler's ability to use facial information such as gaze monitoring during joint attention is considered to be one of the critical discriminatory factors in early diagnosis of this disorder.

Studies of face memory in autism have shown that by middle childhood, children with autism perform worse than mental age and chronological age matched peers on tests of face discrimination (Tantam, Monaghan, Nicholson, & Stirling, 1989) and face recognition (Boucher & Lewis, 1992; Boucher, Lewis, & Collis, 1998; Gepner, de Gelder, de Schonen, 1996; Klin et al., 1999). Several studies suggest that individuals with autism process faces using abnormal strategies. By middle childhood, typically developing children are better at recognizing parts of a face when the parts are presented in the context of a whole face, and they perform better when recognition involves the eyes versus the mouth (Joseph & Tanaka, 2003). Typically developing children also show a greater decrement in memory for inverted versus upright faces as compared with non-face visual stimuli, and attend to upright faces for longer lengths of time than inverted faces (van der Geest, Kemner, Verbaten, & van Engeland, 2002). Children with autism, in contrast, are better at recognizing isolated facial features and partially obscured faces than typical children (Hobson, Ouston & Lee, 1988; Tantam et al., 1989) and show better performance on memory for the lower half of the face than the upper half during childhood (Langdell, 1978). Studies of visual attention to faces indicate that individuals with autism exhibit reduced attention to the core features of the face, such as the eyes and nose, relative to typical individuals (Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Pelphrey et al., 2002; Trepagnier, Sebrechts, & Peterson, 2002).

While the results mentioned above provide insight regarding the abilities of individuals with autism, they often rely on tests requiring verbal ability and as such are mostly conducted with older, higher functioning children. Electrophysiological studies provide information about the neural basis of face processing impairments and autism and do not require a verbal response, making them appropriate

for younger, lower functioning children. Specifically, electroencephalogram (EEG), which refers to the ongoing electrical oscillations of the brain, and event-related potentials (ERPs), which refer to the average electrical signal recorded in relation to a specific timed event, can be used to address fundamental questions about the neural systems involved in face processing in typical and atypical populations. The EEG/ERP signal originates from the post-synaptic (dendritic) potentials of a population of synchronously firing neurons. The activity recorded at the scalp reflects the summation of neurons that are oriented perpendicular to the surface of the scalp, aligned in such a way as to produce a dipole field (a field with positive and negative charges between which current flows; see Coles & Rugg, 1995). EEG/ERPs are exquisitely sensitive to real time neural processes, providing detailed temporal resolution on the scale of milliseconds as to changes in neural state. EEG/ERPs are non-invasive, requiring only that the participant tolerate a damp sensor net or an electrode hat for relatively short periods of time, and they do not necessarily require the participant to follow explicit directions or produce motor or verbal responses. Thus, the methodology can be used across the lifespan and with participants who have limited cognitive or communicative abilities. Such requirements are important for understanding the early stages of brain development and function in young children with autism.

To address face processing abilities in young children with autism during the preschool period, two recent studies from our laboratory have used ERPs during passive viewing, similar to the infant ERP paradigms by de Haan and Nelson (1996, 1997, 1999). Dawson et al. (2002) found that children, age 3–4 years with autism, failed to show differential posterior P400 (approximately 400–500 ms after stimulus onset), anterior Nc (approximately 500–600 ms after stimulus onset) and posterior Slow Wave ERPs to their mother's face versus an unfamiliar face. The children with autism did show differential ERPs to a favorite versus an unfamiliar toy at the posterior P400 and the anterior Nc. In contrast, typically developing children and mental age-matched children with idiopathic developmental delay showed differential ERPs to both the unfamiliar face compared to the mother's face and the unfamiliar toy compared to the favorite toy at the P400 and Nc (children with typical development) and at the Slow wave (children with developmental delay). In separate analyses, the same group of children with autism¹ did not

¹ The same set of children participated in 3 ERP experiments in one session. The first two assessed familiar and novel face recognition and familiar and novel object recognition. The third assessed responses to a neutral and fearful face. Due to child fatigue, not all children had good data for all three experiments; thus the samples varied slightly on which children were included.

differentiate a face expressing a fear from a neutral expression at an early negative component (N300; latency 300 ms) or at the late slow wave component, both at posterior temporal leads. Children with typical development showed relatively greater responses to the fear face than the neutral at both components (Dawson, Webb, Carver, Panagiotides, & McPartland, 2004a).

While the ERP studies mentioned above have assessed face and object processing abilities, the ERP components that differentiated the two conditions were not components that are known to be specific to the early stage of face processing but instead are thought to relate to later stage cognitive processes (e.g., Nc, P400/P500, Slow Wave, see Nelson, 1996). In typical adults, the N170 component is thought to be related to early stage encoding of faces (Bentin, Allison, Puce, Perez, & McCarthy, 1996; Eimer, 1998, 2000). The N170 is maximal over posterior temporal areas (typically measured at electrodes T5 and T6), peaks at about 170 ms after stimulus onset, is faster and larger to face stimuli compared to non-face stimuli, and does not differ based on the familiarity of the face (Bentin, Deouell, & Soroker, 1999; Eimer, 2000). In a recent report, McPartland, Dawson, Webb, Carver, & Panagiotides, (2004) found that individuals with autism displayed slower N170 response to faces than to furniture and failed to show a face inversion effect, which in typical adults is characterized by a slower N170 to inverted than upright faces.

Taylor, McCarthy, Saliba, and Degiovanni (1999) and Taylor, Edmonds, McCarthy, and Allison (2001) have identified a precursor to the adult N170, referred to here as the “precursor N170” in children between 4 and 15 years of age. Across childhood, this component was of greater amplitude to eyes and upright faces than to inverted faces (Taylor et al., 2001) similar to the adult N170. However, no direct comparison was made between upright faces and objects, such as cars (Taylor et al., 1999) in a way similar to that in adult reports (e.g., Bentin et al., 1996). The precursor N170 (prN170) is also significantly slower than the adult N170, peaking at approximately 270 ms in 4–5 year olds, and does not reach adult values (in terms of amplitude and latency) until late-adolescence. The response to upright faces also showed a slower development change in latency and amplitude compared to the other stimuli, which the authors interpreted as indicative of a longer developmental trajectory for configural processing of faces relative to featural processing of eyes alone (Taylor et al., 2001). Taken together, this work suggests that the precursor to a face specific component exists by 4 years of age, but that the component may differ in response properties as compared to adults.

To address whether children with autism have impairments in an early stage of face processing, this study utilized ERPs to examine the prN170 in 3–4 year old

children. This analysis represents a novel re-analysis of previously published ERP data that focused on familiarity and later ERP components (Dawson et al., 2002). Specifically, based on previous work with typical 4 year olds described above, we analyzed the early appearing negative component at posterior temporal leads between 230 and 390 ms in response to faces and objects in children with autism, developmental delay, and typical development.

Methods

Participants

Three groups of children between 33 and 54 months participated in this study: (a) 63 children with ASD, (b) 28 children with typical development (TYP), and (c) 37 children with idiopathic developmental delay (DD). For this analysis, 27 children with ASD, 18 children with TYP and 18 children with idiopathic DD provided adequate artifact free trials for both faces and objects. Of those children who were not included, 20 were non compliant (ASD 12, TYP 0, DD 8), 28 provided too few artifact free trials (ASD 12, TYP 8, DD 8), 3 experienced equipment malfunction (ASD 1, TYP 1, DD 1) and 4 (ASD 0, TYP 2, DD 2) did not have a visible developmental N170 component based on the criteria listed below. This analysis represents a novel re-analysis of previously published ERP data (Dawson et al., 2002); to be included in the re-analysis, subjects had to have good ERP data to both the face and object stimuli and thus fewer subjects were included.

ASD and DD group classifications were confirmed by standard diagnostic criteria including the Autism Diagnostic Observation Schedule- General (Lord, Rutter, Goode, & Heembergen, 1989) and Autism Diagnosis Interview-Revised (Lord, Rutter, & Le Couteur, 1994). 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. TYP children did not meet DSM-IV criteria for Autistic Disorder or PDD-NOS based on clinical judgment.

Mental age was assessed using the Mullen Scales of Early Learning (Mullen, 1997); Mullen’s data was only analyzed for those subjects with good ERP data. The TYP

group ($M = 48.1$ mos. $SD = 8$) had a higher mean mental age than the ASD group ($M = 28.7$ mos. $SD = 10$) and the DD group ($M = 28.9$ mos. $SD = 9$), $t(38) > 6.8$, $ps < .01$. The ASD group and DD had a similar mean mental age, $t(45) = -.07$, ns. Groups did not differ in chronological age (ASD $M = 45.2$ mos. $SD = 4$; TYP $M = 44.4$ mos. $SD = 7$; DD $M = 44.8$ mos. $SD = 5$).

Stimuli

The face stimuli consisted of two digital photos of faces, one familiar to the child (mother) and the other unfamiliar. The object stimuli consisted of digital photos of a familiar object and a perceptually similar unfamiliar object. Objects were the child's favorite toy at the time of testing (e.g., ball, bag, car, rattle, watering can) which was matched to a stimulus that was similar in terms of size, shape and type (monster truck matched with a firetruck or a backpack matched to a bag). The stimulus frames were 520 pixels wide by 420 pixels high and were presented for 500 ms. Faces were presented on the monitor at a size of 16 cm by 12 cm and subtended a visual angle of 12.2° by 9.15° . Because of the greater within category variability, objects were digitally sized so that the image display area approximated the area of the facial images. The visual angle of the objects ranged between 5 and 15° .

ERP Procedure

Prior to ERP data collection, each child received behavioral training sessions to acclimate the child to testing (Dawson et al., 2002). ERPs were recorded from a 64 channel Geodesic sensor net (vertex reference and re-referenced off line to average reference). Impedances were below $40 \text{ k}\Omega$. ERP data were recorded at 250 Hz for 1800 ms, with a 100 ms baseline, 500 ms stimulus duration and recording, 1200 ms recording interval; a variable intertrial interval (500–1000 ms) was implemented between trials. During recording, amplification was set at $1000\times$ and band pass of 0.1 and 100 Hz was used. Data collection was terminated when the child had attended to 100 of each of the stimulus types or when the child was no longer attending. Trials were rejected if the child did not fixate on the picture, the signal amplitude exceeded 250 mV, a running average exceeded 150 mV, or electroocular artifact occurred. The data were corrected for baseline shifts and low pass filtered at 20 Hz.

The prN170 was measured at the right (44, 45, 47, 48) and left (27, 28, 31, and 32) posterior temporal electrodes, corresponding to the 10–20 system locations PO8, P6, P10, P8, P7, P5, P9, and PO7. As the comparison of interest for this analysis was the response to faces versus objects, the responses to the familiar and novel stimuli were collapsed

into one average for both faces and objects; this increases the signal to noise ratio of the data. The component was defined according to the following criteria: (1) a negative going peak between 230 and 390 ms; and (2) present at 2 out of the 4 right or left lead groups. The peak was identified by a computer algorithm (written by the fourth author), which defined the peak as the most negative sample point within the time window where the slope approached zero. (This procedure insures that the value is defined as the most negative point within a curve and not as the negative point at the window boundaries.) The accuracy of this identification procedure was visually confirmed by the first and third authors. Based on these criteria, and stated above under the subject section, an additional four children were not included in the analyses due to a lack of visible prN170 for faces only ($N = 1$ TYP), objects only ($N = 2$ DD), or for both stimuli ($N = 1$ TYP). The values at each lead (the point at which the peak was defined in terms of amplitude and latency) were then averaged to form a mean for the right and left hemisphere electrode groups. A repeated measures ANOVA was conducted; variables included Group (ASD versus DD or ASD versus TYP) as a between subject variable. This was done to account for the differences in mental age between the TYP group and the ASD and DD group and the similarities in ERP morphology between the ASD and TYP group in an earlier report (Dawson et al., 2002). To better understand the affect of autism and to equate group sizes, we compared two subgroups of the ASD group: (1) ASD-autism in which individuals met criteria for autism on both the ADI and ADOS, $n = 15$; and (2) ASD-PDD group in which individuals met ADI autism criteria and ADOS PDD-NOS criteria, $n = 12$. Both groups were compared to the DD group. Within subject factors included stimulus (face, object) and hemisphere (right, left). We did not correct for mental age in these analyses. Greenhouse-Geisser corrections were used to correct for sphericity.

Results

Mean values and standard deviations for each group by stimulus by hemisphere are presented in Table 1. Data used in the statistical analyses reflect the amplitude and latency of the peak value within the time window for each individual; data presented in the table are the averaged values across the group. In Figs. 1 and 2, ERP grand averaged waveforms were created for each group for faces and for objects. The grand average waveform is calculated by averaging the amplitude at each time point. Because of the difference in how the waveforms are graphically created versus the data used in statistical analyses, the values depicted in the figures may differ from the table. Figure 3

Table 1 Means and standard deviations (in parentheses) for the amplitude (in microvolts) and latency (in ms) responses to faces and objects for the ASD, DD and TYP groups

Group	Face Amp Right	Face Amp Left	Obj. Amp Right	Obj. Amp Left
ASD	0.39 (5)	0.42 (7)	5.02 (8)	2.72 (6)
ASD-Aut	0.22 (5)	1.91 (9)	4.79 (6)	2.76 (6)
ASD-Pdd	0.59 (6)	-1.45 (4)	5.30 (10)	2.68 (5)
DD	-0.13 (4)	-0.12 (4)	0.92 (7)	-0.68 (4)
TYP	-1.47 (5)	1.43 (3)	-0.69 (5)	1.38 (4)
Group	Face Latency Right	Face Latency Left	Obj. Latency Right	Obj. Latency Left
ASD	289 (28)	322 (30)	285 (30)	297 (35)
ASD-Aut	303 (31)	321 (27)	284 (33)	294 (31)
ASD-Pdd	293 (25)	323 (35)	286 (23)	302 (40)
DD	291 (31)	296 (33)	288 (35)	302 (35)
TYP	279 (26)	297 (20)	290 (35)	299 (36)

represents a graphical depiction of the mean latency responses in Table 1.

Precursor N170 Amplitude

ASD versus DD

There was a main effect of stimulus type $F(1,41) = 7.3$, $P = .01$, and a stimulus by group interaction, $F(1,41) = 5.5$, $P < .05$. As would be expected of a “n170” component, the response was more negative to faces ($mV = .14$ SD 6) than to objects ($mV = 2.0$ SD 7). There was no difference in prN170 amplitude to faces versus objects for the DD group (Face $M = .13$ mV; Object $M = .12$ mV), $P = ns$; Fig. 1A. In contrast, the ASD group had a larger difference in prN170 amplitude to faces versus objects (Face $M = .4$ mV; Object $M = 3.9$ mV), $P < .001$; Fig. 1B. This larger difference was due to the response to objects, which differed between the autism group and the control groups, Fig. 2B. The response to faces was similar in terms of amplitude across groups, as can be seen in 2A. There were no effects of hemisphere.

For the ASD-autism versus DD comparison, the main effect of stimulus type remained ($P < .05$) but the interaction between stimulus and group was non significant ($P = .1$). For the ASD-PDD versus DD comparison, both the stimulus main effect and the interaction were found ($ps < .05$). Displayed in Table 1, ASD-PDD group demonstrated a more negative prN170 to faces at left leads than the ASD-autism group; this would be considered “more normative” response given the adult profile of the N170.

ASD versus TYP

There was also a main effect of stimulus type $F(1,43) = 9.0$, $P < .01$, and a stimulus by group interaction, $F(1,43) = 6.0$, $P < .05$. Similar to the previous comparison, the TYP group responded similarly to faces and

objects, while the ASD group showed a more positive response to objects; Fig. 1B and C. There was an additional interaction between hemisphere and group, $F(1,43) = 5.6$, $P < .05$, in which the typical group showed a right lateralized response (right more negative than left), $F = 5.2$, $P < .05$, and the autism group showed a bilateral response, $P = ns$.

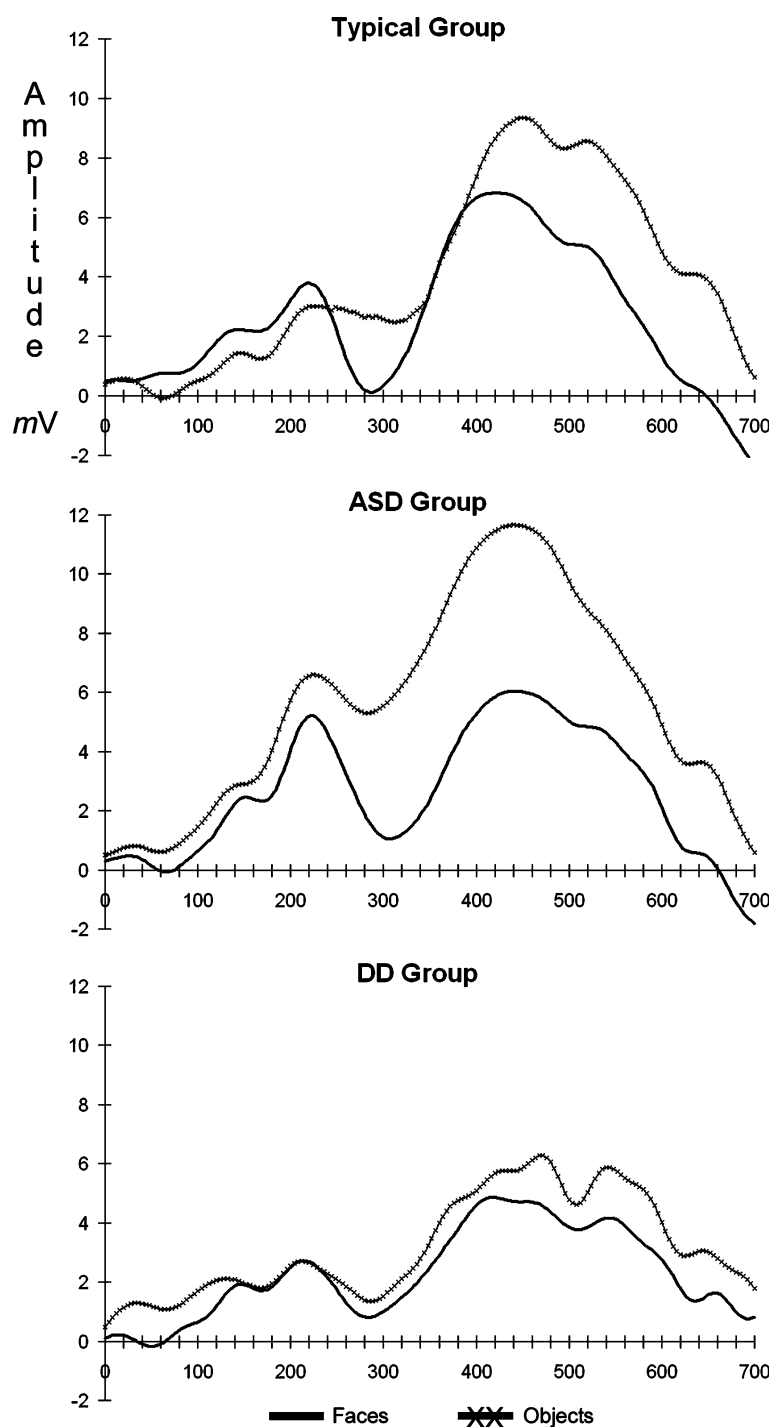
Precursor N170 Latency

ASD versus DD

Repeated measures ANOVA with prN170 latency as the dependent variable revealed a main effect of hemisphere, $F(1,41) = 7.5$, $P < .01$, and a stimulus by group interaction, $F(1,41) = 5.3$, $P < .05$. For both groups, the response was faster in the right than left hemisphere. Best illustrated in Figs. 1 and 3, the DD group showed no difference in prN170 latency to faces versus objects (Face $M = 293$ ms SD 27, Object $M = 295$ ms SD 31), $F = .04$, $P = ns$. In contrast, the autism group showed a faster response to the objects ($M = 290$ ms SD 23) than the faces ($M = 310$ ms SD 25), $F = 16.1$, $P < .001$. As seen in Fig. 3, this effect was driven by (1) a slower response to faces in the left hemisphere than the right ($P < .001$); (2) a slower response to faces than objects in the right hemisphere ($P < .01$); and (3) a slower response to faces than objects in the left hemisphere ($P < .01$).

When the group of children with ASD-autism were compared to the DD group, there was a main effect of hemisphere, $F(1,41) = 4.7$, $P < .05$ and a stimulus by group interaction, $F(1,41) = 5.2$, $P < .05$. In contrast, for the ASD-PDD versus DD group comparison, only the main effect of hemisphere remained, $F(1,41) = 5.9$, $P < .05$; the interaction of stimulus and group did not reach significance, $P = .1$. However, as seen in Table 1, the direction of effects for the ASD-autism and ASD-PDD group are similar.

Fig. 1 Grand averaged ERPs from posterior temporal electrodes (collapsed across hemisphere) for the ASD, Typical, and DD groups



ASD versus TYP

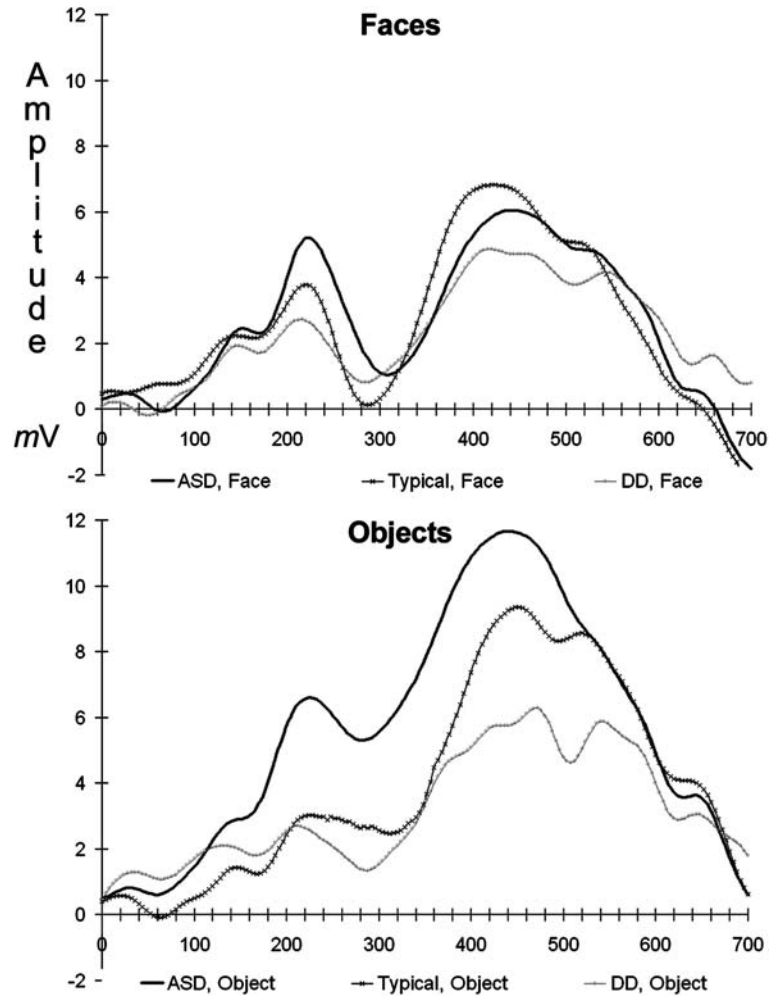
Similar to the ASD and DD comparison, for the ASD versus TYP comparison, there was main effect of hemisphere, $F(1,43) = 11.1, P < .01$, and a stimulus by group interaction, $F(1,43) = 10.4, P < .01$. For both groups, the response was faster in the right than left hemisphere. The TYP group showed a faster prN170 latency to faces versus objects. As seen in Table 1 and Fig. 3, this was driven by faster

responses to faces than objects in the right hemisphere, $P = .05$. In contrast, as mentioned previously, the autism group showed a faster response to the objects than the faces.

Discussion

In this study, prN170 results for 3–4 year olds suggest some characteristics of the adult N170 component.

Fig. 2 Mean responses in ms for the TYP, ASD, and DD group for face and object responses at the right and left posterior temporal electrodes



Latency to peak of prN170

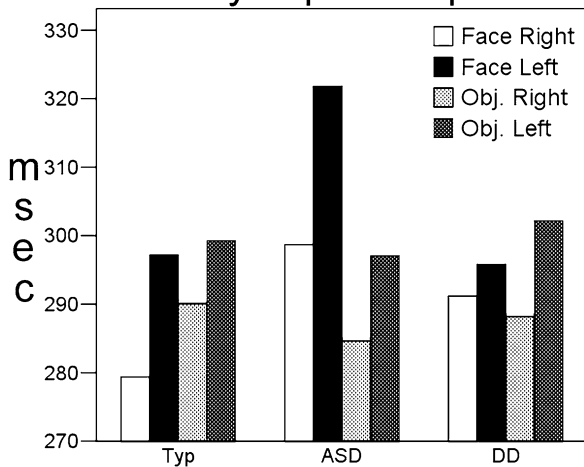


Fig. 3 Graphical presentation of the latency responses between the three groups

Collapsed across groups, children demonstrated a more negative response to faces than objects and faster responses in the right than left hemisphere. When considering the

specific characteristics of the N170, such as faster and more negative responses in the right hemisphere for faces than objects, the profile differed by group.

For the children with typical development, the latency of the response to faces was faster than that to objects, specifically in the right hemisphere. These findings are similar to those from a previous study of typically developing children of this age (Taylor et al., 1999, 2001). In contrast, the DD group and the ASD group did not show a face specific hemispheric response. The DD group also failed to show any differential response to faces and object for latency and for amplitude. The ASD group demonstrated the pattern of having a more negative response to faces than objects; but showed faster responses to objects than faces.

These ERP findings are suggestive of a number of conclusions. First, the prN170 may be quantitatively and qualitatively different in young children. Second, children with general developmental delay, the mental-age and chronological-age matched comparison group, did not show differential responses to faces and objects. Lastly, we

suggest that children with autism do have an atypical brain response to faces and objects, compared to children with developmental delay and typical development. We will address each of these statements below.

First, this analysis made use of data collected in the context of another study designed to address processing of facial and object familiarity. Children only saw two face stimuli and two object stimuli, not the multiple exemplars that have been used in previous N170 studies. The degree to which this manipulation affected the timing and the latency of the precursor N170 is unknown. In our report, the response to stimulus type was a within subject manipulation and highlights the relative difference in face and object processing within the individual and the group. Given the difficulty in assessing children with autism and developmental delay at this age, this report represents a novel first attempt at defining an early component that responds preferentially to faces; further work will be needed to assess the influence of this manipulation.

Recent reports with adults also conflict in regard to the response of the N170 to familiarity and repetition. Reports with adults have suggested the N170 is: (1) affected by repetition (Caharel et al., 2002; Itier & Taylor, 2004; Jemel et al., 2003); (2) not affected by stimulus repetition (e.g., Begleiter, Porjesz & Wang, 1995; Schweinberger, Pfitzner & Sommer, 1995; Schweinberger, Pickering, Jentsch, Burton, & Kaufman 2002); and (3) not affected by familiarity (e.g., Bentin & Deouell, 2000; Eimer, 2000; Rossion et al., 1999). However, a later component, the N250 has been shown to be manipulated by familiarity and repetition (Schweinberger et al., 2002). In the grand averaged waveform (Figs. 1, 2), only one negative component existed within the window of interest. In addition, a visual inspection of the graphs in Dawson et al. (2002), which did compare a familiar to novel response, does not suggest a reliable presence of two negative components (prN170 and precursor N250) within the waveform prior to the P400. It is unclear whether the use of a different protocol might have resulted in different findings.

Second, in post hoc tests, children with developmental delay did not show differential responses to faces and objects either in respect to latency or to amplitude. While it is difficult to draw conclusions from null results, in Dawson et al. (2002), the same children with DD did show differential responses to familiar and novel faces and objects but did at the late slow wave component, which occurs between 670 and 1670 ms after stimulus onset. Nelson (1996) suggests that the slow wave component in infancy is reflective of stimulus updating and differs between repeating familiar, repeating novel, and unique novel stimuli (Nelson & Collins, 1991, 1992). Further, Snyder, Webb and Nelson (2002) found that the amplitude of slow wave component was affected by the number of

trials an infant had seen during an experiment. The lack of evidence of “early” component differentiation for the DD group in this report and Dawson et al. (2002) suggest several possibilities. Idiopathic developmental delay with no known genetic etiology, is a broad diagnostic category and may result in increased variability. While the mean results for the ASD-autism and ASD-PDD groups remained similar despite smaller group sizes, these groups also became more specific in terms of diagnostic definitions. Larger sample sizes may be needed to fully investigate responses from the DD group and to create meaningful subgroups.

Lastly, children with autism showed a reversal of the typical N170 pattern, with significant faster responses to objects than faces. These results primarily reflect latency delays in the left hemisphere. This result is similar to that found in Dawson et al. (2004b), which examined responses to fear faces. Responses to fear and neutral faces in children with autism were also significantly slower in the left hemisphere than the right; responses in the right hemisphere in children with ASD were similar to those of the typical children. These two reports utilize the same population of children, but employed different stimuli sets. For the autism group, the faster neural response to objects relative to faces could be interpreted as preferential or altered processing abilities for nonsocial as compared to social stimuli (Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Dawson et al., 2003). Further replication of these paradigms with another sample of children will be needed.

Measures of the amplitude of the prN170 also revealed interesting differences between the groups. Whereas all three groups showed a similar amplitude to faces, the children with ASD showed a more positive response to objects compared to the children with typical development and those with developmental delay and showed significant differences in the processing of faces and objects in regard to amplitude. The children with autism showed amplitude responses, in terms of face object differentiation, that are more similar to what is seen in typical adults. However, the pattern differs greatly from the two comparison groups, which did not show amplitude differences to faces and objects.

Given that children with autism showed relatively typical patterns of amplitude and latency responses to faces in the right hemisphere compared to the two control groups but showed abnormalities in speed of processing of faces in the left and amplitude responses to objects, we suggest that these patterns reflect abnormal cortical specialization. It is unknown whether these atypical responses in children with autism are the result of an innate disturbance in face processing or whether experiential effects related to a lack of attention to faces or greater attention to objects influence the development of the neural circuits involved in

face processing. The first possibility dictates that face processing abnormalities may represent a faulty “starter set” in the neural substrates that are to become specialized for faces (e.g., fusiform gyrus) or in the mechanisms that are needed to process faces effectively (e.g., configural processing circuitry) (Kanwisher, 2000). The second hypothesis is that face processing deficits arise from lack of experience with faces. Visual experience is necessary for some aspects of face processing (Geldart, Mondloch, Mauer, de Schonen, & Brent, 2002; Grelotti, Gauthier, & Schultz, 2002; de Schonen & Mathivet, 1989). While children with autism do not experience perceptual deprivation, it has been proposed that they may lack the social motivation to attend to stimuli such as faces (Dawson et al., 2002, 2005, Grelotti et al., 2002). This attention to faces may support the development of expertise and the organization of the requisite neural systems necessary to support aspects of face processing. Further research is needed to differentiate these theories.

This report represents a novel first attempt at defining an early component that responds preferentially to faces in young children. Children with autism differed from typical development and developmental delay both in amplitude and latency responses to faces and objects. Further work will be needed to assess the influence of the experimental design and the reliability of these results.

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