

Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder

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Background: Individuals with autism exhibit impairments in face recognition, and neuroimaging studies have shown that individuals with autism exhibit abnormal patterns of brain activity during face processing. The current study examined the temporal characteristics of face processing in autism and their relation to behavior. **Method:** High-density event-related brain potentials (ERPs) were recorded to images of faces, inverted faces, and objects from 9 individuals with autism spectrum disorder (15–42 years old) and 14 typical individuals (16–37 years old). **Results:** With respect to a face-sensitive ERP component (N170), individuals with autism exhibited longer N170 latencies to faces than typical individuals but comparable latencies to objects. Typical individuals exhibited longer N170 latencies to inverted as compared to upright faces, whereas individuals with autism did not show differences in N170 latency to upright versus inverted faces. Neural speed of face processing, as reflected in N170 latency, correlated with performance on a face recognition task for individuals with autism. **Conclusions:** These data provide evidence for slowed neural speed of face processing in autism and highlight the role of speed of processing in face processing impairments in autism. **Key-words:** Event-related potential, N170, autism, face processing. **Abbreviations:** ERP: Event-related potential; N170: negative component at 170 milliseconds; ASD: Autism spectrum disorder.

Autism is a developmental disorder characterized by impairments in social interaction and communication and a restricted range of activities (American Psychiatric Association, 1994). These impairments likely relate to dysfunction of a brain system underlying social cognition (Bachevalier, 1994; Barbas, 1995; Baron-Cohen et al., 1999, 2000; Brothers, 1990; Damasio, 1994; Dawson, Meltzoff, Osterling, & Rinaldi, 1998; LeDoux, 1994). There is increasing evidence that autism involves an impairment in the visual processing of faces (Cipolotti, Robinson, Blair, & Frith, 1999; Hauck, Fein, Maltby, Waterhouse, & Feinstein, 1998; Jambaque, Mottron, Ponsot, & Chiron, 1998; Klin et al., 1999; Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Ozonoff, Pennington, & Rogers, 1990; Teunisse & DeGelder, 1994). One of the first symptoms evident in autism is a lack of attention to faces. In a study of home videotapes of first birthday parties, the failure to attend to others' faces was the best discriminator between one-year-olds with autism versus those with typical development (Osterling & Dawson, 1994). Research suggests that individuals with autism exhibit face processing difficulties throughout development. These individuals show face-specific deficits on simple discrimination tasks, such as picking the odd face out of a group of otherwise identical faces (Tantam, Monaghan, Nicholson, & Stirling, 1989), as well as more complex face recognition tasks (Boucher & Lewis, 1992; Boucher, Lewis, & Collis, 1998; de Gelder, Vroomen, & van der Heide, 1991).

These poor face discrimination and recognition abilities may stem from abnormal information processing strategies. Whereas typical individuals have more difficulty recognizing an inverted face, children with autism fail to demonstrate the 'face inversion effect' (Langdell, 1978). Children with autism are also better at recognizing individual facial features and partially obscured faces than typical children (Hobson, Ouston, & Lee, 1988; Tantam et al., 1989). These findings suggest that individuals with autism do not utilize configural processing strategies typically used for face processing (Elgar & Campbell, 2001; Freire, Lee, & Symons, 2000). Instead, individuals with autism may rely on feature-based processing (Joseph, 2001). These abnormal processing strategies may reflect dysfunctional face-processing brain circuitry.

Neuroimaging studies have provided information about brain activity associated with face processing in individuals with autism. Functional magnetic resonance imaging (fMRI) studies indicate that adolescents and adults with autism spectrum disorders show abnormal patterns of brain activation during face processing. While most typical individuals exhibit activation in the right fusiform gyrus when viewing faces, individuals with autism have been shown to exhibit irregular and inconsistent patterns of activation (Pierce, Muller, Ambrose, Allen, & Courchesne, 2001; Schultz et al., 2000). Dawson et al. (2002) examined event-related brain potentials (ERPs) in three- to four-year-old children during passive viewing of familiar and unfamiliar faces and

toys. Both typically developing children and children with developmental delay showed differential brain activity to familiar versus unfamiliar faces and toys. However, children with autism showed differential brain activity only for familiar versus unfamiliar toys. These studies indicate that by three to four years of age children with autism exhibit abnormalities in patterns of brain activity during face processing, and these abnormalities are not attributable to developmental delay alone.

Face processing offers potential insight into the nature and etiology of autism spectrum disorders. An interest in faces is evident very early in development. Neonates as young as one hour old preferentially attend to human faces (Johnson, Dziurawiec, Ellis, & Morton, 1991), and infants are more likely to smile in response to faces than other similarly complex forms of visual stimuli (Goren, Sarty, & Wu, 1975; Wolff, 1963). Infants are also capable of recognizing their mother's face within the first few days of life (Bushnell, Sai, & Mullin, 1989). These early forms of face processing are considered to be a developmental precursor to more sophisticated aspects of human social cognition and interaction (Baron-Cohen, 1995; Brothers, 1990; Cole, 1998; Perrett et al., 1990; Perrett, Hietanen, Oram, & Benson, 1992; Williams, Whiten, Suddendorf, & Perrett, 2001). The developmental primacy of face processing and its core role in social development underscore its importance in understanding abnormal social development in autism.

Studies of face processing in autism can be useful in understanding the neural basis of autism, as a great deal is understood about the brain mechanisms underlying typical face processing. Insight into the brain basis of human face processing originates from the study of prosopagnosia (Bodamer, 1947). This visual disorder, an inability to recognize individual human faces, is caused by genetic or acquired lesions to the inferior occipitotemporal region (Damasio, Tranel, & Damasio, 1990; Meadows, 1974). Although it is most frequently observed with bilateral damage (Damasio, Damasio, & Van Hoesen, 1982), in cases in which unilateral damage results in prosopagnosia, the damage tends to be in the right hemisphere (De Renzi, 1986; Landis, Cummings, Christen, Borgen, & Imhof, 1986; Sergent & Signoret, 1992). Neuroimaging studies have further elucidated the brain regions involved in face processing. Both positron emission tomography (PET) and fMRI studies indicate that a portion of the occipitotemporal cortex, the right fusiform gyrus, is activated during perception of faces as compared to various non-face stimuli, inverted faces, or scrambled faces (Clark, Maisog, & Haxby, 1998; Haxby et al., 1994, 1999; Hoffman & Haxby, 2000; Kanwisher, McDermott, & Chun, 1997; Kanwisher, Tong, & Nakayama, 1998; McCarthy, Puce, Gore, & Allison, 1997; Puce, Allison, Asgari, Gore, & McCarthy, 1996; Puce, Allison, Gore, & McCarthy,

1995; Sergent, Ohta, & Macdonald, 1992; Wojciulik, Kanwisher, & Driver, 1998). Intra-cranial electrophysiological studies using subdural electrodes have also shown distinct patterns of electrophysiological activity in the fusiform and inferotemporal gyri during the viewing of face stimuli (Allison et al., 1994). These findings suggest important roles of the fusiform gyrus and inferotemporal gyri in face processing and provide a target, specified in terms of both function and neuroanatomy, to better understand social brain circuitry in autism.

Information regarding the temporal aspects of neural processing of faces can be ascertained through electroencephalographic recordings. Numerous ERP studies have found a distinct pattern of brain activity in response to human faces or face-related stimuli compared to non-face stimuli (e.g., furniture or cars). This pattern consists of a right-lateralized, negative peak over the lateral posterior regions of the scalp occurring about 170 milliseconds after stimulus presentation (the 'N170'; Bentin, Allison, Puce, Perez, & McCarthy, 1996; Eimer, 1998, 2000a, 2000b, 2000c; George, Evans, Fiori, Davidoff, & Renault, 1996). Research has shown that, though the N170 may be elicited by non-face stimuli, faces consistently evoke the shortest latencies and largest amplitudes (e.g., Rossion et al., 2000). The component varies in latency and amplitude in response to disruptions in the configuration of facial features, such as facial inversion or partial decomposition. The N170 exhibits enhanced amplitude and longer latencies to inverted faces relative to upright faces (Bentin et al., 1996; Eimer, 1998, 2000c). In most studies, the electrophysiological effects of inversion have been shown to be specific to faces, as compared to other comparably complex visual stimuli (Eimer, 2000b; Rebai, Poiroux, Bernard, & Lalonde, 2001; Rossion et al., 2000). The N170 is not affected by the familiarity of faces, and it is therefore interpreted as reflecting the earliest stages of face processing, 'structural encoding', that precede recognition processes (Eimer, 2000a; Bentin & Deouell, 2000). The latency of the N170 is the most sensitive indicator of disruption in early stages of face processing (Eimer, 1998, 2000a; Bentin & Deouell, 2000).

The current study used high-density ERP recordings (Tucker, 1993) to measure electrophysiological brain activity in high-functioning adolescents and adults with autism spectrum disorder and age and nonverbal IQ-matched typical individuals as they watched pictures of upright and inverted faces and objects. By analyzing brain activity to upright faces, non-face stimuli, and inverted faces, the current study sought to elucidate four aspects of face processing in autism. First, despite several neuroimaging studies demonstrating abnormal regional brain activity during face processing, no study has yet assessed the temporal characteristics of face processing in autism. The latency of the N170 provides

insight into the timing of the earliest stages of face processing, those associated with encoding the structural components of the face. We hypothesized that individuals with autism have an impairment in temporal processing of faces characterized by slower neural speed of early stage processing, as indexed by longer latencies to N170. Second, by assessing brain activity to upright versus inverted faces, we hoped to gain insight into whether individuals with autism show sensitivity to alterations in the configural information provided by faces, as indexed by the N170 and evidenced in typical individuals. We hypothesized that, as suggested by previous research, individuals with autism do not employ configural processing strategies and would, therefore, fail to exhibit differential patterns of brain activity to configurally altered, i.e., inverted, faces. Third, we were interested in examining whether individuals with autism show typical patterns of cortical specialization for face processing. Based on previous studies in our laboratory conducted with younger children (Webb, Dawson, & Shook, 2002), we hypothesized that individuals with autism would fail to show the characteristic pattern of right-hemisphere specialization and would display bilateral activation characteristically associated with immature processing (Taylor, Edmonds, McCarthy, & Allison, 2001). Fourth, we sought to explore relations between face processing speed, as assessed by ERP, and face recognition, as assessed by performance on a behavioral task. We hypothesized that disruptions in the temporal processing of faces, as reflected in slower N170 latency, would be correlated with poorer face recognition ability, as assessed on neurocognitive tasks.

Materials and methods

Participants

Two groups of adolescents and adults participated in the study: 15 individuals with autism spectrum disorder (ASD) who had diagnoses of either Autistic Disorder or Asperger Syndrome and 21 neuropsychiatrically normal, medically healthy individuals. Participants were recruited from the University of Washington Autism Center, local parent advocacy groups, public schools, the Department of Developmental Disabilities, clinics, hospitals, and the University of Washington Psychology Department Infant and Child Subject Pool. Exclusionary criteria for participants with autism

included any associated known neurological, genetic, infectious, or metabolic disorder (e.g., tuberous sclerosis or fragile X, Norrie syndrome, neurofibromatosis), seizures, significant sensory or motor impairment, major psychiatric disorder other than autism, major physical abnormalities, serious head injury, or neurological disease. Exclusionary criteria for typical participants included birth or developmental abnormalities, brain trauma, current or past history of psychiatric or neurological disorder, medical disorder with implications for the central nervous system or requiring regular medication usage, family history of autism, developmental cognitive disorder, learning or language disability, neurological disorder of known etiology (e.g., fragile X), significant sensory or motor impairment, major physical abnormalities, or history of serious head injury.

All participants included in the autism group had been previously diagnosed with Autism or Asperger Syndrome based on a comprehensive multidisciplinary evaluation and administration of the Autism Diagnostic Interview – Revised (ADI-R; Lord, Rutter, & Le Couteur, 1995). The ADI-R is a parent-interview that assesses the symptoms of autism according to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV; American Psychiatric Association, 1994). In addition, the first author, who is experienced in the diagnosis of autism spectrum disorders, verified a clinical diagnosis of Autistic Disorder or Asperger Syndrome based on clinical observation of the presence of autism symptoms as defined in the DSM-IV.

Of the initial sample of 15 individuals with ASD, 9 (3 with Autistic Disorder and 6 with Asperger Syndrome) provided adequate artifact-free data (4 provided too few artifact-free trials, and 2 were inattentive). Of the initial sample of 21 typical participants, 14 provided adequate artifact-free data (7 provided too few artifact-free trials). Table 1 presents sample demographic and descriptive information, including sex, ethnicity, handedness, chronological age, and Wechsler performance IQ (Wechsler, 1991, 1997b) for both groups. Groups did not significantly differ on any of these variables. All participants had a performance IQ in the non-mentally retarded range.

EEG recording procedure

Stimuli. Stimuli consisted of gray-scale digital images presented on a computer monitor with a black background (Bentin et al., 1996; Martinez & Benavente, 1998; Samaria & Harter, 1994). Images were standardized in terms of size, background color (gray), and mean luminance. Stimuli were presented randomly in a single block composed of 60 each of four different

Table 1 Participant characteristics

	N	Male/Female	Ethnicity	Handed	Chronological age (years)			Performance IQ		
					Min	Max	M (SD)	Min	Max	M (SD)
ASD group	9	8/1	8 Caucasian 1 Other	7 Right 2 Left	15.1	42.5	21.2 (8.3)	80	131	103.6 (17.0)
Typical group	14	13/1	11 Caucasian 3 Other	14 Right 0 Left	16.4	37.7	24.6 (6.3)	72	133	106.6 (15.1)

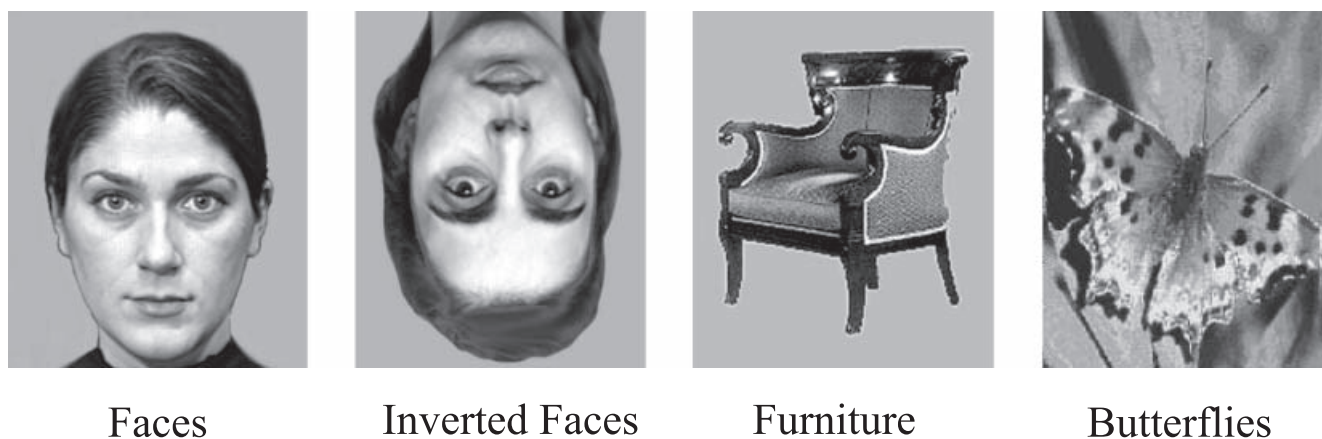


Figure 1 Example stimuli

stimulus categories: faces, inverted faces, pieces of furniture, and inverted pieces of furniture. Thirty butterflies were interspersed as target stimuli. To control for attention, participants were instructed to mentally count the number of butterflies, and participants who miscounted by a value of 10 or greater were deemed inattentive and excluded from analyses. Results to inverted pieces of furniture are not included in the current analyses. Examples of the stimuli used in the experiment are shown in Figure 1.

Data collection. EEG was recorded in an electrically shielded, sound-attenuated, darkened room. The participant was seated comfortably approximately 75 centimeters from the computer monitor that delivered the stimuli. A large, threefold screen obscured the back of the monitor and the back part of the room from the participant's view. A 128 lead Geodesic sensor net (Electrical Geodesics Incorporated; Tucker, 1993) was dipped into potassium-chloride electrolyte solution, placed on the participant's head, and fitted according to the manufacturer's specifications. The electrodes were evenly spaced and symmetrically covered the scalp from nasion to inion and from left to right ears. Impedances were kept below 40 k Ω .

ERP was recorded continuously throughout each stimulus presentation trial, consisting of a 130-millisecond baseline recording, a 500-millisecond stimulus presentation, and a 670-millisecond blank screen. Intertrial interval varied randomly between 500 and 1000 milliseconds, with an average of 750 milliseconds. Total testing time was approximately 10 minutes.

The EEG signal was amplified ($\times 1000$) and filtered (0.1 Hz high-pass filter and 100 Hz elliptical low-pass filter) via a preamplifier system (Electrical Geodesics Incorporated). The conditioned signal was multiplexed and digitized at 250 Hz using an analog-to-digital converter (National Instruments PCI-1200) and a dedicated Macintosh computer. All 128 channels were recorded continuously and streamed to the computer's hard disk. A second Macintosh computer, interfaced and synchronized via serial port, generated the stimuli. Stimulus onset and cessation were registered with the physiological record for off-line segmentation of the data. The vertex electrode was used as a reference, and

data were re-referenced to an average reference after data collection.

Data editing and reduction. Data were averaged for each subject by stimulus type across trials. Trials with artifacts were excluded from the averages. Each electrode's signal was checked for transients and maximum amplitude. A weighted running average was used with the thresholds set to 150 for transit and 250 for voltage. Running averages are analogous to using a band pass filter and reject both high-frequency noise and low-frequency drift. This method identifies the slope and rejects sharp transitions in the data. Trials during which eye movement occurred were also excluded. Subjects with fewer than 10 artifact-free trials were excluded from analysis. Averaged data were transformed to correct for baseline shifts and digitally filtered (low-pass Butterworth 30 Hz) to reduce environmental noise artifacts. Spline interpolation of data from neighboring sites was used to replace data from electrodes for which greater than 25 percent of the trials were rejected due to artifacts. Participants for whom more than 10 channels required replacement were excluded from further analyses. An average of 1.1 ($SD = .9$) channels per participant were replaced for the ASD group, and an average of 0.2 ($SD = .4$) channels per participant were replaced for the typical group.

Electrodes of interest were selected based on review of the literature and examination of grand averages and individual participant data. Corresponding to the semimedial electrode groups used in Halit, de Haan, and Johnson's (2000) study of ERPs to faces in typical adults, six electrodes over the left lateral posterior scalp (58, 59, 64, 65, 69, 70) and six electrodes over the right lateral posterior scalp (90, 91, 92, 95, 96, 97) were selected. These lead groups were also deemed most appropriate based on observed magnitude of the N170 at these electrodes and their correspondence to T5 and T6, the electrodes most commonly analyzed in previous studies (e.g., Bentin et al., 1996; Eimer, 1998, 2000a, 2000b, 2000c; Rossion et al., 2000). The layout of the Geodesic Sensor net and the electrodes of interest are shown in Figure 2. The time window for N170 was chosen by visual inspection of the grand average and data for individual participants. The time intervals used extended from 142 milliseconds after

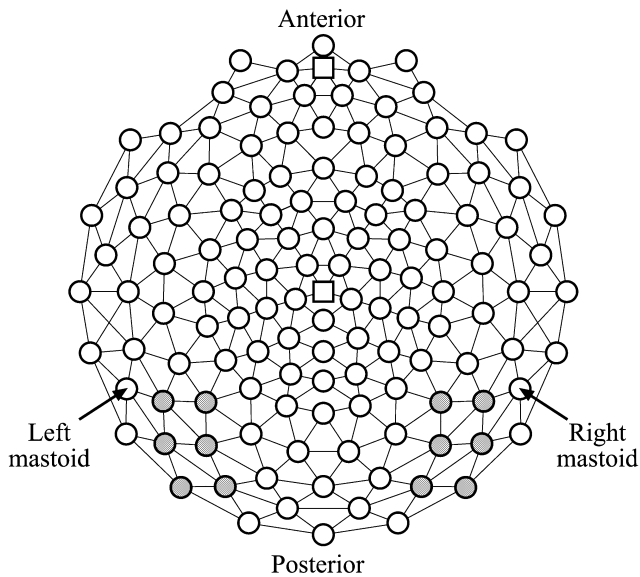


Figure 2 Electrode groups over which data were averaged for N170

stimulus presentation to 230 milliseconds after stimulus presentation. Peak and latency to peak were averaged across the specified electrodes within the specified time window and these values were extracted for each participant.

Neurocognitive testing

Recognition memory task. The Wechsler Memory Scale – Third Edition (WMS-III): Faces Subtest (Wechsler, 1997a) was used to assess immediate recognition memory for faces. In this standardized assessment, 24 faces are presented for a total of 2 seconds each, and the participant is asked to remember each one. Once the 24 faces have been presented, a second set of 48 faces is presented one at a time. For each face, the participant is to indicate if the face is one that he or she was asked to remember.

Data analysis. Number of errors out of 48 trials was computed for each participant. Pearson's product moment correlations were calculated between the number of errors and the ERP measures (N170 amplitude and latency for upright and inverted faces).

Results

ERP analyses

Figure 3 displays grand averaged ERP waveforms to faces, furniture, and inverted faces for typical individuals and individuals with ASD. Two separate analyses of the ERP variables were conducted: (1) faces versus non-face stimuli (furniture) and (2) upright faces versus inverted faces. For each comparison, separate univariate repeated measures analyses of variance (ANOVA) were conducted for

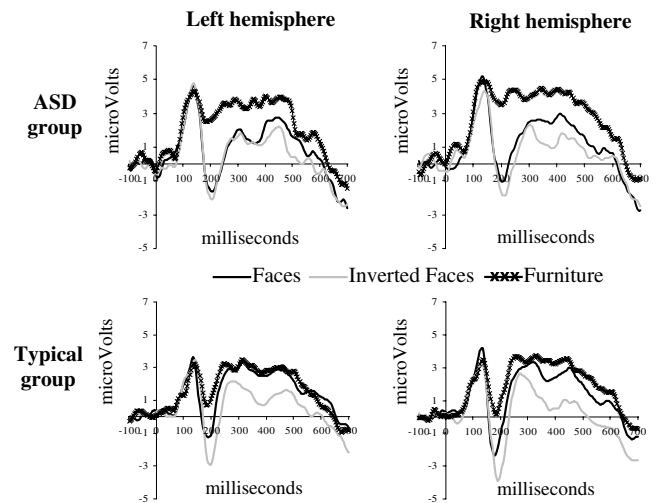


Figure 3 Waveforms for ASD and typical groups averaged across the six specified electrodes in each hemisphere. The figure depicts the grand average across all subjects in each group for upright faces, upright furniture, and inverted faces

Table 2 N170 latency

Hemisphere	Condition	<i>M</i> (milliseconds)	(<i>SD</i>)
Typical group			
Left	Faces	182.29	(16.9)
	Furniture	183.43	(21.6)
	Inverted faces	192.57	(15.9)
Right	Faces	180.57	(13.4)
	Furniture	186.86	(16.5)
	Inverted faces	192.00	(14.3)
ASD group			
Left	Faces	198.89	(14.9)
	Furniture	195.33	(22.7)
	Inverted faces	199.33	(12.6)
Right	Faces	200.67	(20.4)
	Furniture	186.44	(20.7)
	Inverted faces	206.89	(17.2)

N170 amplitude and N170 latency to peak. For each analysis, group was the between-subjects factor and condition (face vs. furniture; upright faces vs. inverted faces) and hemisphere (left, right) were within-subjects factors.

N170 latency. Table 2 shows N170 latency for all conditions and both hemispheres. ANOVA comparing N170 latencies to faces versus furniture revealed a significant group by condition interaction, $F(1, 21) = 4.52$, $p < .05$. To interpret this interaction effect, post-hoc analyses compared groups in terms of N170 latency to faces and furniture across hemispheres. For faces, the autism group exhibited a significantly longer N170 latency than that of the typical group ($t = 3.16$, $p < .01$). For furniture, groups did not differ in terms of N170 latency ($t = 0.77$, $p = .45$). This interaction effect is depicted in Figure 4.

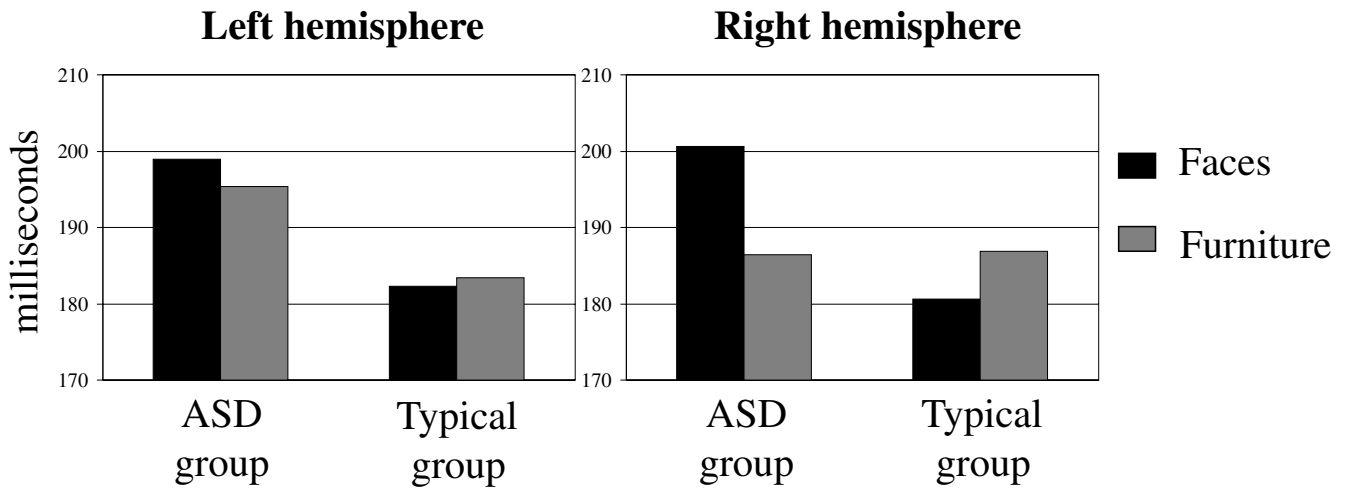


Figure 4 Latency of the N170 component to faces versus furniture

Analysis comparing N170 latencies to upright versus inverted faces revealed a main effect of orientation, $F(1, 21) = 15.68, p < .01$, a main effect of group, $F(1, 21) = 6.76, p < .05$, and a group by orientation interaction, $F(1, 21) = 4.41, p < .05$. Irrespective of group or hemisphere, inverted faces tended to elicit longer latencies than upright faces ($t = 4.21, p < .01$). Irrespective of hemisphere or condition, individuals with ASD tended to have longer N170 latencies to faces than typical individuals ($t = 2.60, p < .05$). Furthermore, whereas typical individuals showed a robust difference in latency to upright versus inverted faces (mean difference ~ 10.9 milliseconds), with longer latencies to inverted than upright faces in both hemispheres, individuals with ASD showed minimal difference in N170 latencies to upright versus inverted faces (mean difference ~ 3.3 milliseconds).

N170 amplitude. ANOVA comparing N170 amplitude to faces versus furniture revealed a main effect of condition, $F(1, 21) = 41.41, p < .01$. As shown in

Table 3 and Figure 3, across group and hemisphere, peak N170 amplitude was greater to faces compared to furniture. In terms of hemispheric lateralization, groups were not significantly different in cross-hemisphere differences in amplitude. Typical individuals exhibited a hemispheric difference in N170 amplitude (right–left hemisphere) of -0.57 microvolts, while individuals with ASD exhibited a difference of $.14$ microvolts.

A similar analysis comparing N170 amplitude to upright versus inverted faces revealed a main effect of orientation, $F(1, 21) = 11.35, p < .01$. Across group and hemisphere, peak N170 amplitude was greater to inverted faces compared to upright faces (see Table 3).

Face recognition test

Relative to typical participants, participants in the ASD group made significantly more errors on the face recognition test (7.9, 14.0, respectively; $t(21) = 2.84, p < .01$). As shown in Table 4, fewer errors made by individuals with ASD correlated with slower left hemisphere N170 to both upright and inverted faces ($r = -.73, p < .05$; $r = -.84, p < .01$, respectively). For typical individuals, correlations between N170 latency to upright faces and number of errors were marginally significant in both left and right hemispheres ($r = .48, p < .10$; $r = .47, p < .10$, respectively). Post-hoc regression analysis was conducted to analyze between-group differences in the relationship between task performance and N170 latency. In the left hemisphere, the correlation between task performance and N170 latency differed between groups for both upright faces ($t = 3.8, p < .01$) and inverted faces ($t = 4.7, p < .01$). In the right hemisphere, differences in correlation coefficients did not attain significance for upright faces ($t = 1.6, p = .13$) or inverted faces ($t = 1.8, p = .09$).

Table 3 N170 amplitude

Hemisphere	Condition	M (microvolts)	(SD)
Typical group			
Left	Faces	-2.67	(2.7)
	Furniture	-0.03	(2.8)
	Inverted faces	-3.80	(2.9)
Right	Faces	-3.24	(3.0)
	Furniture	-0.58	(3.2)
	Inverted faces	-4.76	(3.5)
ASD group			
Left	Faces	-2.55	(2.9)
	Furniture	1.34	(3.1)
	Inverted faces	-3.07	(3.7)
Right	Faces	-2.41	(3.3)
	Furniture	1.99	(3.0)
	Inverted faces	-3.35	(4.7)

Table 4 Correlations between latency and face recognition errors

	Faces		Inverted faces	
	Left	Right	Left	Right
Typical group	.48, $p = .08$.47, $p = .09$.43, $p = .12$.43, $p = .12$
ASD group	-.73, $p = .03$	-.27, $p = .48$	-.84, $p = .01$	-.35, $p = .35$

Discussion and conclusion

The present study examined event-related brain potentials to faces, both upright and inverted, and objects (furniture) in individuals with autism spectrum disorder and typical individuals. Results from this study indicate that, compared to age and IQ-matched individuals with typical development, individuals with autism exhibit abnormal temporal processing of faces. Significant group differences in the latency of the face-sensitive ERP component, N170, were found. Specifically, as we predicted, regardless of orientation, individuals with autism spectrum disorder showed significantly longer N170 latencies to faces than typical individuals (approximately 18 milliseconds longer on average). Also consistent with our hypotheses, group differences in neural response to face inversion were found. Typical individuals showed a robust difference in latency to upright versus inverted faces, with longer latencies (approximately 11 milliseconds) to inverted than upright faces in both hemispheres. This indicates sensitivity to the configural aspects of the face and greater efficiency in processing faces as they are typically represented in a natural environment. In contrast, individuals with autism spectrum disorder showed minimal differences in N170 latencies to upright versus inverted faces (approximately 3 milliseconds), indicating less sensitivity to alteration in the configural properties of faces. Finally, suggestive evidence of atypical cortical specialization for face processing was found for individuals with autism. Although differences were not statistically significant, typical individuals exhibited a $-.57$ microvolt right-left lateralization difference in N170 amplitude to faces, while individuals with autism spectrum disorder exhibited a $.14$ microvolt right-left lateralization difference in N170 amplitude to faces. In summary, these results support the hypothesis that autism is associated with abnormal temporal processing of faces, demonstrated in this experiment by slower early-stage processing of faces.

Analyses examining the relation between individual differences in neural speed of processing of faces, as reflected by N170, and performance on a face recognition task showed different patterns of results for individuals with ASD and typical individuals. For individuals with autism spectrum disorder, slower left hemisphere N170 latency to both upright and inverted faces was associated with better face recognition ability. In contrast, typical individuals exhibited a marginally significant association in both

hemispheres between faster processing speed for upright faces and improved face recognition ability. This pattern suggests that not only is there a failure of normal right hemisphere advantage for face processing in autism, i.e., abnormal cortical specialization, but also that the strategies for improved performance in face processing in autism might be very different than those used by typical individuals. Furthermore, the finding that slower (rather than faster) speed of left (rather than right) hemisphere processing of faces correlated with improved task performance suggests that individuals with autism may be employing a qualitatively different processing strategy for faces.

The results of the current study emphasize the importance of speed of processing in understanding the neuropathology associated with face processing in autism spectrum disorders. Slowed processing of faces among individuals with autism may reflect a failure to develop expertise in face processing. Research shows that among individuals who have developed expertise with a particular class of objects, speed of processing is more rapid than among non-experts (e.g., Tanaka & Curran, 2001). A failure to develop face expertise in autism might be related to a lack of normal attention to faces during development. If this is the case, the brain systems typically subserving face processing might be immature and slower in autism. Indeed, research suggests that as humans progress from infancy through adulthood, they develop expertise for faces, and processing speed, as indexed by the N170, increases (Taylor et al., 2001). This hypothesis could be tested by training individuals with autism to develop expertise with faces and measuring whether N170 latency decreases among those who do so.

An alternative interpretation of the results is that they reflect a more general pattern of neuropathology among individuals with autism; abnormalities in processing speeds may relate to the broader core social impairments in autism. Navigating social interactions requires complex and rapid integration of information from multiple sensory modalities. For example, a simple interaction might consist of a mother leaning over her child, simultaneously smiling, speaking, and tickling the child's palm. To derive full meaning from this interaction, the infant's nervous system must appropriately integrate incoming visual, aural, and tactile information. Inconsistency or delay in processing speed within even a single modality could eliminate the perceived simultaneity among these inputs and thereby

disrupt contingencies crucial for associative learning. If, as evidenced in the current study, individuals with autism experience slowed or variable processing speeds in one or more sensory systems or with particular types of information (e.g., social information), it follows that disturbances might be evident in a variety of domains of function, as is the case in the disorder.

Questions remain regarding the nature of face processing impairments in autism and the role these impairments play in the overall syndrome. Inattention to human faces is the earliest emerging reliably documented impairment in individuals with autism spectrum disorders (Osterling & Dawson, 1994; Werner, Dawson, Osterling, & Dinno, 2000); however, it remains unclear to what degree face processing contributes to, rather than reflects, autistic symptomatology. Some authors have proposed that face processing impairments underlie core deficits in autism spectrum disorders. Trepagnier's gaze disruption hypothesis (1996, 1998) posits that acute, early-onset anxiety induces social withdrawal among infants with autism, thereby reducing exposure to faces during a critical period. Trepagnier hypothesizes that this failure to develop face expertise contributes to later significant social impairments associated with autism. However, it is unlikely that the profound social impairments experienced by individuals with autism can be fully attributable to problems with face processing. Children with autism who experience typical development followed by autistic regression have been shown to display appropriate attention to faces before the regression occurred (Werner, Dawson, Munson, Osterling, & McPartland, 2001). Furthermore, neuroanatomical studies of individuals with autism reflect abnormalities in distributed brain systems, several of which have been documented to occur prenatally in subcortical brain regions that are likely unrelated to face processing (Kemper & Bauman, 1998). These findings suggest that face processing impairments relate only to limited aspects of social dysfunction in autism.

An alternative hypothesis for the role of face processing impairments in autism has been put forward by other authors (Dawson et al., 2002; Grelotti, Gauthier, & Schultz, 2002), who posit that abnormalities in social attention fail to direct autistic infants' attention to human faces, and, consequently, individuals with autism fail to develop 'expertise' for processing human faces. According to such a model, initial inattentiveness to faces circumvents the natural development of expertise in face processing, creating problems at the basic stage of structural encoding and the temporal delays evidenced in the current study. Subsequent and more sophisticated stages of face processing, such as face recognition or interpretation of emotional expressions, could be affected if these delays in basic processing interfere with downstream transmission

of information, i.e., to a face recognition module or emotional expression interpretation circuit. This model also suggests that face processing abnormalities in autism might be affected at various points in the face processing pathway. For example, dysfunction of the amygdala, a limbic structure involved in both the social abnormalities in autism (Bachevalier, 1994) and in face recognition (Aggleton, 1992; Damasio et al., 1982), could affect face processing at its most basic level by inducing social inattentiveness early in development (resulting in a failure to develop face expertise). Amygdala dysfunction could also affect face processing at subsequent stages of processing through its independent involvement in face recognition circuitry. This model incorporates the direct developmental sequelae of face processing deficits (as per Trepagnier 1996, 1998) while acknowledging the contribution of independent difficulties in other domains of function and their independent and potentially additive or synergistic effects.

An alternative potential explanation for face processing anomalies in autism is that autism is associated with innate or early abnormalities in the fusiform gyrus or, more generally, in the inferotemporal cortex. Additional insight into this possibility can be provided by future studies investigating the development of expert processing. For example, if individuals with autism can demonstrate fusiform activation during processing of novel stimuli from an homogenous object class (e.g., 'Greebles'; Gauthier, Tarr, Anderson, Skudlarski, & Gore, 1999) or demonstrate inversion effects during visual processing of visual stimuli in which they possess expertise (e.g., objects related to preoccupations or favorite objects), it follows that the difficulties in face recognition might not be associated with dysfunction of the fusiform and configural processing circuitry, *per se*, but may instead be attributable to a face-specific lack of expertise related to inattention to human faces.

Yet another possible explanation for the face processing difficulties shown in individuals with autism may relate to the visual characteristics of human faces. Laeng and Caviness (2001) have proposed that prosopagnosia results from impairments in the visual processing of curved surfaces. The authors point out that volumetric and curved visual information are vital in processing and recognizing human faces. It is therefore possible that impairments in these domains could contribute to face processing abnormalities in autism. Human faces are also unusual in their degree of 'featural contiguity'; boundaries of individual facial features are not clearly demarcated, and individual judgments of the boundaries of one feature are interrelated with judgments regarding the boundaries of other features. This type of featural contiguity is also present among other forms of stimuli that show specialization in terms of behavioral effects and brain activity (e.g., Greebles, dogs, birds). In contrast, visual stimuli that offer clearer delineations among

individual features (e.g., houses, furniture) have not been consistently shown to exhibit specialization in brain function or behavior. Future research will need to examine visual processing among individuals with autism while accounting for these basic, low-level visual characteristics of stimuli.

Face processing is a vital area of research in understanding the nature of autism spectrum disorders and holds great promise for improving diagnostic methods and interventions. Early behavioral indices of face processing provide a potential marker for recognizing autism spectrum disorders during infancy. Future autism screening methods potentially could take advantage of the primacy of face preference and face recognition to assess children for autism at developmental stages in which more complex social and communicative behaviors have yet to emerge. Face processing also provides a viable avenue for intervention. Given the possibility of a critical period for the development of face processing expertise (LeGrand, Mondloch, Maurer, & Brent, 2001), it may be important to incorporate face processing strategies into early intervention programs for children with autism spectrum disorders.

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