2008-01-01

Face Processing: The N170 ERP Component in Autism

Camilla Marie Hileman
University of Miami, chileman@psy.miami.edu

UNIVERSITY OF MIAMI

FACE PROCESSING: THE N170 ERP COMPONENT IN AUTISM

By

Camilla M. Hileman

A THESIS

Submitted to the Faculty
of the University of Miami
in partial fulfillment of the requirements for
the degree of Master of Science

Coral Gables, Florida

May 2008

UNIVERSITY OF MIAMI

A thesis submitted in partial fulfillment of
the requirements for the degree of
Master of Science

FACE PROCESSING: THE N170 ERP COMPONENT IN AUTISM

Camilla M. Hileman

Approved:

____
Dr. Peter Mundy
Professor of Psychology

____
Dr. Terri A. Scandura
Dean of the Graduate School

____
Dr. Heather Henderson
Assistant Professor of Psychology

____
Dr. Jeffrey Brosco
Associate Professor of
Clinical Pediatrics

HILEMAN, CAMILLA M. (M.S., Psychology)
Face Processing: The N170 ERP Component in Autism (May 2008)

Abstract of a thesis at the University of Miami.

Thesis supervised by Professor Peter Mundy.
(74)

Face processing deficits appear early in autism and greatly impact subsequent development. In this paper, the N170 component, an event-related brain potential sensitive to face processing, is examined in children with autism and typical development. The N170 amplitude was sensitive to group differences, as children with typical development showed greater differentiation to upright vs. inverted stimuli and faces vs. vehicles than children with autism. The N170 was also delayed in children with autism. The N170 was not a sensitive marker of individual differences in social behavior and autistic symptomology, but the proceeding positive peak, the P1, was a sensitive marker of individual differences in children with typical development. Results suggest that children with autism and children with typical development employ different face processing strategies, even for the basic encoding of a face.

TABLE OF CONTENTS

CHAPTER 1: INTRODUCTION

Background Information

Autism is a complex developmental disability, characterized by qualitative impairments in social interaction and communication, as well as a restricted repertoire of behaviors and interests (American Psychiatric Association, 2000). Often, parents become concerned about their child's development when they notice a profound lack of reciprocal social interaction in their child. A disinterest in other people, poor eye contact, an absence of pointing, late speech development, and empty gaze are among the symptoms that parents frequently report as items of first concern (Gillberg et al., 1990). This disengagement from the social world is one of the clearest indicators of autism, leading many researchers to suspect that autism may be caused by a failure to orient to social stimuli, or a lack of motivation to experience the social world (e.g. Dawson & Lewy, 1989; Klin, 1991; Mundy & Burnette, 2005).

In the present study, the social orienting model was used as the theoretical backdrop to explore face processing in autism. Various social impairments may result from a social orienting deficit, but this study focused on face processing as a key social impairment that arises from this deficit. Face processing is particularly important to study because it appears early in development and becomes integral for subsequent development. In this study, the N170, an event related brain potential (ERP) component sensitive to face processing, was examined across children with typical development and autism. Using N170 amplitude and N170 latency, individual differences in social behavior and autistic symptomology were then explored.

The Social Orienting Model of Autism

Past research suggests that infants are predisposed to attend to certain aspects of the environment. These predispositions provide a starting point around which infants structure subsequent behavior and development (Mundy and Burnette, 2005). Typically developing infants seem to have a predisposition for social information processing (Blass, 1999), and the development of critical social skills, including joint attention, imitation, and face processing, builds upon this innate predisposition. In individuals with autism, this predisposition seems to be absent (Gillberg et al., 1990), and the development of subsequent social skills is adversely affected (Dawson & Lewy, 1989; Klin, 1991). Mundy and Burnette (2005) further note that abnormal social orienting may influence experience-expectant neural development (Greenough, Black, & Wallace, 1987). Through the neural pruning process, typical social processing pathways may be eliminated while atypical neural pathways remain. Thus, the initial social processing difficulty in autism may be further amplified by abnormal neural circuitry.

A multitude of studies show social orienting deficits in autism. Dawson, Meltzoff, Osterling, Rinaldi, and Brown (1998) examined the ability of individuals with autism, typical development, and Down Syndrome to orient to social stimuli (name called, hands clapping) and non-social stimuli (rattle, musical jack-in-the-box). They found that individuals with autism significantly more frequently failed to orient to all stimuli, but this impairment was most extreme for social stimuli. Furthermore, when individuals with autism did orient to the social stimuli, their response was significantly delayed. Although the results of this study support a social orienting deficit in autism, it is possible that individuals with autism were actually more familiar with the social stimuli and thus had a deficit in orienting to familiar stimuli. In a follow-up study, Dawson, Toth, et al. (2004) addressed this limitation. They presented a wide range of social and non-social stimuli, varying in familiarity, to individuals with autism spectrum disorder, developmental delay, and typical development. Once again, they found that individuals with autism significantly more frequently failed to orient to all stimuli, and this failure was most extreme for the social stimuli.

Lepisto et al. (2005) asked individuals with autism and typical development to pay attention to a soundless video while speech sounds and non-speech sounds played in the background. They measured the P3a, an ERP index of involuntarily orienting to sounds. For the autism group, the P3a was absent for pitch changes in the speech sounds but only slightly attenuated for pitch changes in the non-speech sounds. Lepisto et al. (2005) interpreted these findings to suggest that individuals with autism have a specific deficit in involuntarily orienting to speech sounds.

Leekam and Ramsden (2006) examined social orienting, joint attention, and their relation in autism. During a play interaction, the experimenter would make a bid for the child’s attention. Individuals with autism responded to significantly fewer bids than individuals with developmental delay, indicative of a social orienting impairment. Using the Early Social Communication Scales (ESCS) play interaction (Mundy & Hogan, 1996), individuals with autism also less frequently initiated joint attention with the experimenter. For individuals with autism, there was a significant correlation between social orienting and initiating joint attention, suggesting that impairments in social orienting may be associated with impairments in joint attention.

Rogers, Hepburn, Stackhouse, and Wehner (2003) went a step beyond social orienting and joint attention to examine imitation in autism. In this study, participants completed the Imitation Battery, an assessment in which the experimenter demonstrated certain actions and asked the participant to repeat those actions. Compared to participants with typical development and developmental delay, individuals with autism were significantly impaired on overall imitation, oral-facial imitation, and manual imitation. Participants were also evaluated on the ESCS, and initiating joint attention ability on the ESCS significantly correlated with oral-facial and object imitation ability. These studies suggest that imitation ability may build on joint attention ability, which may build on social orienting ability.

The above collection of studies demonstrates a social orienting impairment in autism and shows how this impairment can affect higher social functioning. Next, this paper will briefly discuss how a social orienting impairment can specifically affect face processing in autism. If an individual has difficulty orienting toward faces or has limited experience viewing faces, it is not surprising that he or she would have impaired face processing abilities. The following selection of studies will demonstrate some of the face processing difficulties often seen in individuals with autism.

Celani, Battacchi, and Arcidiacono (1999) conducted a two-part study of face processing in autism. In a delayed-matching task, they showed participants a target picture and then three sample pictures. The participant was required to pick the sample picture that either matched the facial expression or the identity of the target picture. When matching by facial expression, individuals with autism performed worse than individuals with Down Syndrome or typical development. When matching by identity, there were no differences among the three groups. In a sorting-by-preference task, participants were simultaneously presented with two faces or two nonsocial emotional situations of varying valence. They were asked to pick the face or the situation that they liked the best. In the face condition, individuals with autism made significantly more incongruent choices than controls; in the emotional situation condition, there were no significant differences among groups.

Baron-Cohen, Wheelwright, Hill, Raste, and Plumb (2001) administered the revised “Reading the Mind in the Eyes” Test to individuals with Asperger Syndrome or high-functioning autism and typical development. In this task, participants are presented with photographs of people’s eyes, and they are asked to identify the mental state of the eyes from four possible choices. Individuals with autism were significantly impaired on this task, but they were not impaired on a control task in which they were asked to identify gender. Furthermore, performance on this task was inversely correlated with score on the Autism-Spectrum Quotient, a measure of autistic symptomology. Baron-Cohen et al. (2001) concluded that individuals with autism have a deficit in processing mental state from the eyes and this deficit is most pronounced for individuals with severe autism.

The above studies show that individuals with autism have difficulty extracting emotions from faces. Adolphs, Sears, and Piven (2001) showed that individuals with autism also have difficulty making appropriate social judgments from faces. In this study, participants were asked to assess the trustworthiness of 100 faces. Individuals with autism performed significantly differently from controls on this task, as they gave abnormally high ratings to faces typically viewed as less trustworthy. In a control task, participants were asked to rate the likeability of adjectives that described personality attributes. Individuals with autism were not impaired on this control task.

Thus far, much research has been presented to support the idea of a social orienting deficit in autism. Social orienting is the foundation upon which more advanced social skills emerge, such as face processing, and impairments in social orienting can lead to impairments in these higher social skills. Many research studies have employed appropriate non-social control tasks, leading researchers to conclude that individuals with autism have a specific impairment in social orienting.

While the social orienting model of autism serves as the theoretical foundation for the research presented in this paper, it is important to note that not all researchers subscribe to this model. For example, Turati (2004) argues that infants' preference for faces is due to the perceptual features of faces, not social orienting. Turati believes that infants are attracted to all visual stimuli that share certain perceptual characteristics, such as up-down asymmetry, wherein more elements are located in the upper half of the stimulus than the lower half of the stimulus. Faces themselves may not be preferred stimuli; faces may merely show this set of preferred perceptual features. However, as social orienting has been demonstrated in multiple domains, not just face processing, Turati's explanation is unlikely.

Face Processing: Why is it Important?

Before describing a study that focuses on face processing in autism, it is necessary to understand why this line of research is important. This paper will emphasize three key reasons for researching face processing in autism. First, face processing deficits appear early in autism. Research shows that these deficits appear by 3 years of age, possibly earlier (Dawson, Webb, Carver, Panagiotides, & McPartland, 2004). Second, deficits in face processing adversely affect subsequent social development, such as the development of reciprocal social interaction. Lastly, deficits in face processing adversely affect subsequent development in general, from early world learning to speech perception. It is quite possible that face processing is a "pivotal" developmental skill, with changes in face processing leading to changes in many other critical areas (Koegel & Frea, 1993).

Dawson et al. (2002) examined brain responses to familiar faces, unfamiliar faces, familiar toys, and unfamiliar toys in participants aged 34-54 months. When presented with familiar and unfamiliar faces, typically developing children showed differential amplitudes for the P400, Nc, and PSW components while children with autism did not show differential amplitudes for these components. When presented with familiar and unfamiliar objects, both typically developing children and children with autism showed differential amplitudes for the P400 and Nc components. Thus, young children with autism appear to have a specific deficit in processing familiar and unfamiliar faces. This is not a general deficit in processing familiar and unfamiliar stimuli, as participants with autism did not show an atypical neural response to objects.

In a similar study, Dawson, Webb, et al. (2004) worked with children with autism spectrum disorder and typical development, ranging in age from 30 to 58 months. In this study, ERPs were recorded while participants passively viewed pictures of fearful and neutral faces. Children with typical development had both a larger N300 component and negative slow wave (NSW) following a fearful face than following a neutral face. Children with autism failed to show either of these differential brain responses. As indicated by these results, children with autism experience atypical neural responses to facial expressions at a young age.

Lastly, Anderson, Colombo, & Shaddy (2006) used a novel methodology to examine face processing in individuals with autism spectrum disorder, developmental delay, and typical development. Participants in this study were 4 years of age on average, and an eye-tracking system was used to measure pupillary response to face stimuli. Individuals with autism showed pupillary constriction to the face stimuli, while individuals with typical development and developmental delay showed pupillary dilation to the face stimuli. Again, young children with autism did not demonstrate the typical physiological response to face stimuli. As briefly demonstrated above, many studies employing diverse methodologies have repeatedly found a face processing abnormality in young children with autism.

Difficulty with face processing may lead to subsequent difficulties in social development. In an intriguing qualitative study, Cole (2001) draws upon the experience of facially disfigured persons to inform the development of social relationships. Cole recounts the experiences of people who are physically unable to form facial expressions, such as individuals with advanced Parkinson’s disease. While these individuals can understand and evaluate facial expressions, they are unable to modify their own facial expressions in response to another person's facial expressions. Similarly, due to face processing deficits, individuals with autism often fail to modify their facial expressions in response to another person's facial expressions. In his research, Cole notes that people are reluctant to actively engage with non-responsive social partners. He recounts the social dynamics of a Parkinson's Disease Society meeting:

> People would ask these facially impoverished people questions that demanded 'yes, no' answers rather than more expansive ones, so avoiding an engaged conversation. There was a definite movement away from these people at the meetings. Perhaps people did not get any feedback from the patients and so did not feel engaged and encouraged. (p. 60)

Due to face processing abnormalities, children with autism may not have the same opportunities to experience reciprocal social interaction, thereby adversely affecting their social development.

Stormark and Braarud (2004) further investigated the effects of face processing impairment by using a face-to-face interaction between infants and mothers. In this study, the infant and mother interacted via a closed-circuit TV system. The infant's gaze behavior was measured while the infant and the mother interacted in real-time and again while the infant and the mother reacted to a video replay of the other's behavior. Infants gazed significantly more frequently at their mother during the real-time condition than the replay condition. This study shows that facial expressions provide infants with clues about the reciprocity of social interaction, and infants modify their behavior accordingly. Infants who can't process facial expressions may not learn how to appropriately modify their social behavior in response to a social partner.

Working with an economically disadvantaged sample, Izard et al. (2001) administered two face processing tasks to children at age 5. This study used hierarchical regression to determine that children's face processing knowledge at age 5 was a significant predictor of their social skills, behavior problems, and academic competence at age 9, controlling for verbal ability and temperament. As Izard et al. (2001) note, face processing ability is critical for prosocial behavior. A lack of social skills in the school setting can further result in poor school adjustment and poor teacher rapport. Thus, face processing deficits may ultimately lead to difficulties in both social and nonsocial domains.

Brooks and Meltzoff (2005) investigated the impact of face processing deficits on another area of nonsocial development, language. In an infant-experimenter play interaction, the experimenter would make eye contact with the infant, turn her head, and look at a target. In one condition, the experimenter would close her eyes while turning toward the target; in a second condition, the experimenter would leave her eyes open. At ages 10 and 11 months, infants looked at the target significantly more frequently in the eyes open condition than in the eyes closed condition. This shows that infants already use face/eye cues to process an adult's gaze, not merely head turn cues. In addition, Brooks and Meltzoff (2005) showed that correct gaze and number of vocalizations at the 10-11 month play interaction significantly predicted language score on the Communicative Development Inventory at 14 and 18 months, particularly phrases understood, words understood, and total gestures. These results echo the Morales, Mundy, and Rojas (1998) finding that infants who can follow gaze at 6 months have better receptive vocabulary at 12 months and better expressive vocabulary at 18 and 24 months, as assessed by the

MacArthur Communicative Development Inventory. Face processing, particularly eye processing, may be integral to early word learning in infancy.

In a series of experiments, Hollich, Newman, and Jusczyk (2005) investigated the ability of infants to segment words in a speech stream. They found that infants were better at this task when presented with auditory speech synchronized with a visual face display, as opposed to auditory speech unsynchronized with a visual face display or auditory speech and a static visual face display. These results indicate that infants actively employ face processing to separate a stream of speech. Infants with a face processing deficit may have difficulty discriminating words in a speech stream.

From this overview, it is clear that face processing skills play an integral role in both social and general development. Since individuals with autism show face processing deficits at such an early age, these deficits may have a significant impact on subsequent development. With this theoretical framework in hand, the N170 ERP component will now be discussed as an electrophysiological measure of face processing.

The N170 ERP Component

This paper investigated the neural processing of face stimuli, specifically the N170 ERP component. The N170 component is thought to reflect the earliest stage of face processing, the structural encoding that occurs before a face has even been recognized. This component is a right-lateralized, negative peak that occurs approximately 170 milliseconds after stimulus presentation (Eimer, 2000). Three lines of research on the N170 component are particularly relevant to this study: the N170 component in adults, the N170 component in children, and the N170 component in individuals with face processing deficits. The preponderance of research focuses on the N170 component in adults, thus this line of research will be emphasized.

The N170 Component in Adults

Bentin, Allison, Puce, Perez, and McCarthy (1996) tested the specificity of this component by presenting pictures of human faces, animal faces, human hands, cars, and furniture to undergraduate and graduate students. The N170 component was larger for human faces than all other stimuli, suggesting that this component is indeed specific to human faces. This component was not elicited to the same degree by other faces (animal faces), other human body parts (human hands), or other stimuli in general (cars and furniture). In a follow-up experiment, participants were presented with upright and inverted faces and cars. Cars, regardless of orientation, did not elicit an N170 component. Compared to upright faces, there was a delayed and enhanced N170 response to inverted faces. These results suggest that N170 amplitude increases when the configural properties of a face are disturbed. The N170 for faces was larger in amplitude for the right hemisphere than the left hemisphere in both experiments, although this difference was only significant in the second experiment.

Rossion et al. (1999) further investigated the effect of face inversion on the N170 component. In this study, participants had a mean age of 25 and were presented with a prime face, a perceptual mask in which no faces could be seen, and then a target face. The prime and target faces shared the same orientation (both upright or both inverted), and the participant was asked to decide whether the two faces were identical or different.

Rossion et al. (1999) observed a delayed and increased amplitude for inverted faces versus upright faces. This inversion effect was significantly more pronounced in the right hemisphere than the left hemisphere. They offered two potential explanations for this inversion effect. First, inverted faces may be more difficult to process and may require the contribution of more neurons, thus leading to a larger ERP component. Second, inverted faces may activate both neurons in the face processing circuit and neurons in the object processing circuit, again leading to a larger ERP component.

In his research, Eimer (2000) tackled two additional questions regarding the N170 component. First, he investigated whether this response was affected by stimulus familiarity; second, he investigated whether this response was delayed and enhanced for inverted non-face stimuli. Participants, ranging in age from 18-30, were presented with upright and inverted pictures of houses, familiar faces, and unfamiliar faces. The N170 was not altered by the familiarity of a face, indicating that this component does indeed reflect the earliest stage of face processing. Consistent with the above studies, the N170 was found to be delayed and enhanced in response to inverted faces, and this effect was larger over the right hemisphere. Surprisingly, the N170 was found to be enhanced but not delayed for inverted houses. This research suggests that the increased N170 amplitude for inverted stimuli may not be specific to faces.

Rossion et al. (2000) further investigated the specificity of the enhanced N170 amplitude for inverted stimuli. In this study, participants had a mean age of 25 years, and they viewed photographs of upright and inverted faces, cars, shoes, chairs, houses, and Greebles. The N170 component was largest for the face stimuli, and it was delayed and enhanced for inverted faces. Contrary to the previously presented study, stimulus orientation did not affect the N170 component for non-face visual stimuli. As this result has been replicated, it seems most likely that the demonstrated inversion effect is specific to faces (Rebai, Poiroux, Bernard, & Lalonde, 2001).

Just as there is controversy about the specificity of the N170 inversion effect to faces, there is an overarching controversy about the specificity of the N170 component to faces. In a theoretical paper, Rossion, Curran, and Gauthier (2002) argue that the N170 component reflects expert processing, rather than face processing. Most research studies have presented face stimuli, for which humans are expert processors, and object stimuli, for which humans are not expert processors. Consequently, face processing and expert processing have been confounded in most research. In fMRI studies, the fusiform "face area" has been activated for both face and expert processing (Gauthier, Skudlarski, Gore, & Anderson, 2000), and it is distinctly possible that the N170 component is similarly activated for both face and expert processing.

In order to further investigate this phenomenon, Tanaka and Curran (2001) presented pictures of birds and dogs to individuals with visual expertise in bird or dog processing. Participants ranged in age from 32-57, and all participants had at least 10 years of experience in their field of expertise. Expert bird processors had a significantly larger N170 amplitude to birds while expert dog processors had a significantly larger N170 amplitude to dogs. Tanaka and Curran (2001) note that the N170 observed in response to objects of expertise was strikingly similar in timing and scalp location to the N170 observed in response to faces.

Gauthier and Curby (2005) also evaluated the N170 component as a marker of early processing for objects of expertise. They presented faces and cars to individuals with and without expertise in cars. For non-car experts, the N170 response was largest for faces, but, for car experts, the N170 response was largest for cars. Furthermore, the extent to which car processing interfered with typical face processing correlated with level of car expertise. While these research studies suggest that the N170 is indicative of expert processing, only a handful of studies presently offer empirical support for this idea. The preponderance of research still supports the theory that N170 is a face-specific component.

In sum, the adult literature suggests that the N170 is a face-specific, right-lateralized ERP component, although some researchers argue that the N170 is specific to expert processing, not face processing. The N170 is not influenced by face familiarity, indicating that this component reflects the earliest stages of face processing. The N170 is delayed and enhanced for inverted faces, and this inversion effect tends to be strongest over the right hemisphere. The inversion effect seems to be specific to faces, although some research suggests otherwise.

The N170 Component in Children

Research on the N170 component in children is limited and not always consistent with the adult literature. In general, the N170 component is assumed to be a face-specific component for children since it is a face-specific component for adults. While this is a logical assumption, little research has corroborated (or sought to corroborate) this assumption. Most research studies with children have examined the N170 in response to face stimuli only and have not used control nonsocial stimuli. Taylor, Edmonds, McCarthy, and Allison (2001) examined the N170 component in 90 children, ranging in age from 4-15. They found that the N170 was evoked in response to eyes, upright faces, and inverted faces while only a small N170 was evoked in response to scrambled faces and flowers. Thus, the N170 does appear to be a face-specific component in childhood. In this study, the N170 was lateralized to the left hemisphere in younger children and lateralized to the right hemisphere in older children.

Taylor, McCarthy, Saliba, and Degiovanni (1999) asked children ages 4-14 and adults to view a slideshow presentation of faces, cars, scrambled faces, scrambled cars, and butterflies. Participants were asked to press a button in response to the target stimuli, butterflies. In this study, the N170 component did not always extend below baseline in young children. N170 amplitude increased with age while N170 latency decreased with age. Furthermore, the N170 component was not lateralized to the right hemisphere in young children and not significantly lateralized to the right hemisphere in older children and adults.

Itier and Taylor (2004a) studied the development of the N170 component in children ages 8-15 and adults. Stimuli for this study included upright faces, inverted faces, and contrast-reversed faces, and participants were asked to recognize a specific target face from a stream of distracter faces. N170 amplitude did not increase across children, but N170 amplitude did significantly increase between children ages 14-15 and adults. N170 latency decreased across all age groups, but this decrease was most pronounced between children ages 8-9 and 10-11. In contrast to the previous study, the N170 was a right-lateralized component across all age groups. Itier and Taylor (2004b) also found a similar pattern of amplitude and latency results across development.

Itier and Taylor (2004a) and Itier and Taylor (2004b) both examined the N170 face inversion effect in childhood. Both studies found that the N170 was delayed for inverted faces across all age groups. Both studies also found that the N170 was enhanced for inverted faces only in older age groups. Itier and Taylor (2004a) found that this inversion effect began at ages 12-13 while Itier and Taylor (2004b) found that this inversion effect didn't clearly begin until adulthood.

Most recently, Batty and Taylor (2006) investigated the N170 component in response to emotional faces in children ages 4-15. In contrast to previous studies, this study found a very specific developmental pattern for the N170 amplitude. The N170 was shown to decrease in amplitude until 12-13 years and then increase in amplitude at 14-15 years. Similar to previous studies, N170 latency decreased with age, and this decrease was not continuous across age groups. Again, the N170 component was lateralized to the right hemisphere.

In sum, the child literature has not thoroughly tested the specificity of the N170 component to faces. However, the research that has employed non-social stimuli suggests that the N170 component is face-specific in childhood. The N170 appears to be right-lateralized in late childhood. The latency of the N170 component decreases with age, but this decrease may not be continuous across age groups. The amplitude of the N170 component generally increases with age, although there may be a more specific developmental pattern of increases and decreases. The N170 is delayed for inverted faces across all age groups while the N170 is only enhanced for inverted faces in older children and/or adults.

The N170 Component in Individuals with Face Processing Deficits

The N170 component will be described in both individuals with prosopagnosia and individuals with autism. Individuals with prosopagnosia have a specific impairment in face recognition without sensory or perceptual deficits to account for this impairment. Research on the N170 component in these populations is limited.

Eimer and McCarthy (1999) analyzed the N170 component in response to faces and houses in 1 adult with prosopagnosia and 24 control participants. For controls, the N170 response was typical, with an enhanced N170 to faces versus houses. The N170 component was absent for the individual with prosopagnosia. This individual showed behavioral deficits in structurally encoding a face, thus these results support the interpretation of N170 as an early face component for structural encoding.

Working with a different adult with prosopagnosia, Bentin, Deouell, and Soroker (1999) analyzed the N170 response to faces and objects. For undergraduate controls, the N170 was elicited for faces only; for the individual with prosopagnosia, the N170 was elicited for both faces and objects. In contrast to the participant in the above study, this participant only showed difficulties in face identification; the participant was able to adequately determine age, sex, and affect from a face. This qualitative difference may explain why the first individual with prosopagnosia failed to show an N170 response and the second individual with prosopagnosia showed an atypical N170 response.

O'Connor, Hamm, and Kirk (2005) investigated the N170 component in children with Asperger Syndrome, typically developing children, adults with Asperger Syndrome, and typically developing adults. Typically developing children had a mean age of 11.2 years, and children with Asperger Syndrome had a mean age of 11.6 years. In this study, participants were presented with a series of upright faces and asked to identify the expression of each face. Typically developing adults showed a significantly larger N170 amplitude and a significantly shorter N170 latency than adults with Asperger Syndrome. There were no significant differences in performance for children with typical development and children with Asperger Syndrome. Across groups, the N170 component was larger in the right hemisphere than the left hemisphere. These results are quite intriguing, as they suggest that electrophysiological abnormalities in early face processing in autism may not be apparent until late childhood or adulthood.

In another study, O'Connor, Hamm, and Kirk (2007) asked control adults and adults with Asperger Syndrome to discriminate between target and distracter stimuli. There were no group differences on N170 amplitude. Across groups, amplitude was right-lateralized and larger for eyes than mouths, faces, and objects. Controls had a significantly shorter N170 latency to eyes and mouths than individuals with Asperger Syndrome, and there were no group differences on N170 latency to objects.

Lastly, McPartland, Dawson, Webb, Panagiotides, and Carver (2004) investigated the N170 component in adults and adolescents with autism (ages 15-42) and adults and adolescents with typical development (ages 16-37). Participants viewed pictures of upright faces, upright pieces of furniture, inverted faces, and inverted pieces of furniture. For faces, the N170 latency was significantly longer for individuals with autism; for pieces of furniture, the N170 latency was not significantly different between groups. Furthermore, individuals with typical development showed a large difference in N170 latency for upright and inverted faces, while individuals with autism showed a minimal difference in latency for upright and inverted faces. These results suggest that individuals with autism may be less affected by a disruption in the configural properties of face stimuli. Across groups, N170 amplitude was greater for faces than furniture and greater for inverted faces than upright faces.

The McPartland et al. (2004) study was the only study to investigate how the N170 component related to social behavior in individuals with autism. For individuals with autism, fewer errors on a face recognition task significantly correlated with a longer N170 latency in the left hemisphere. In contrast, for individuals with typical development, fewer errors on a face recognition task marginally correlated with a shorter N170 latency in both hemispheres. These results suggest that individuals with autism may employ a qualitatively different face processing strategy from individuals with typical development.

In sum, the N170 component is abnormal for individuals with prosopagnosia, although the specific abnormalities of this component depend on the extant face processing abilities of the individual. Only one study has examined the N170 in children with autism and typical development, and this study has not shown group differences. In adults with autism, the N170 is delayed for faces and latency is less affected by face inversion. Some evidence suggests that a longer N170 latency is associated with better face recognition in autism. The N170 amplitude for faces may not be different across groups or may be slightly attenuated for adults with autism. The N170 component appears to be right-lateralized in both typical development and autism.

The Current Study

In the current study, participants with autism and typical development (ages 9-17) watched a picture slideshow of upright vehicles, inverted vehicles, upright faces, and inverted faces. In order to ensure attention to the stimuli, they were asked to respectively count the number of female faces or the number of vehicles facing left. EEG activity was collected while participants viewed this slideshow, and the N170 component was subsequently analyzed.

This study had three primary objectives. (1) This study aimed to examine N170 amplitude and latency in children with autism. As far as this author is aware, this was first study to examine the N170 in children with autism ages 9-17. The autism group in O'Connor et al. (2005) had a mean age of 11.6 years with a standard deviation of 1.9 years, and the autism group in McPartland et al. (2004) had a mean age of 21.2 years with a standard deviation of 8.3 years. This study bridged the age gap between these studies by employing an autism group with a mean age of 13.3 years and a standard deviation of 2.8 years. (2) This study aimed to examine the inversion effect for faces in children with autism using the N170 component. McPartland et al. (2004) looked at this effect in older adolescents and adults with autism, but, as far as this author is aware, this was the first study to examine the N170 inversion effect in children with autism. (3) This study aimed to examine how the N170 component relates to individual differences in social behavior and autistic symptomology in children with autism. Again, McPartland et al. (2004) looked at this relation in older adolescents and adults with autism, but, as far as this author is aware, this was the first study to examine the relation between the N170 component and individual differences in social behavior and autistic symptomology in children with autism.

In the current sample, it was not clear whether the N170 would follow the adult pattern, which is more clearly documented in the literature, or the child pattern, which is just beginning to be documented in the literature. Thus, two sets of hypotheses were developed. One set of hypotheses described N170 predictions if the sample more closely followed the adult pattern, and a second set of hypotheses described N170 predictions if the sample more closely followed the child pattern.

If the N170 adhered to the adult pattern, the following results were expected: Individuals with autism would have a delayed latency and possibly an attenuated amplitude for faces. Individuals with typical development would show a stronger inversion effect for faces than individuals with autism. Across groups, the N170 would be lateralized to the right hemisphere.

If the N170 adhered to the child pattern, the following results were expected: There would be no group differences on N170 amplitude and latency. The N170 amplitude would increase with age, and the N170 latency would decrease with age. In general, the N170 component would be lateralized to the right hemisphere, although this lateralization effect may not hold for younger children. For all children, the N170 would be delayed for inverted faces. For older children, the N170 would also be enhanced for inverted faces.

Finally, N170 latency was expected to be related to individual differences, such that a shorter N170 latency for the control group and a longer N170 latency for the autism group would be correlated with less autistic symptomology and more social skills. This prediction is in line with the results from McPartland et al. (2004). It was not clear how N170 amplitude would relate to individual differences.

CHAPTER 2: METHODS

Participants

The initial sample included 32 participants with high-functioning autism and 31 control participants. Five children with autism were excluded from the final sample: 1 child was unable to finish the protocol, 1 child had missing data due to equipment malfunction, and 3 children did not meet the requirements for an autism diagnosis. Nine control children were excluded from the final sample: 3 children met the criteria for autism and 6 female children were excluded for matching purposes (see below for a full explanation). The final sample included 27 participants with high-functioning autism and 22 control participants.

Participants with high-functioning autism were recruited from the University of Miami Center for Autism and Related Disabilities. Participants with high-functioning autism were required to meet a cutoff score of 7 on the Autism Diagnostic Observation Schedule (ADOS; Lord, Rutter, Dilavore, & Risi, 1999) and a cutoff score of 15 on the Social Communication Questionnaire (SCQ; Berument, Rutter, Lord, Pickles, & Bailey, 1999). Since this was a high-functioning sample, participants who met the cutoff score on one measure and were within one point of the cutoff score on the other measure were also included in the sample. Participants with typical development were recruited from local public and private elementary, middle, and high schools. Control participants were excluded from the study if they met criteria for autism on either the ADOS or the SCQ. Additionally, all participants needed a verbal IQ greater than or equal to 70, as assessed by the Wechsler Intelligence Scale for Children (WISC) Verbal Comprehension Index. All participants were excluded from the study if they had significant sensory or motor impairments, a neurological disorder, or psychotic symptoms.

The recruitment procedure initially resulted in mismatched groups, with the control sample having more females, a higher average verbal IQ, and an older average age than the autism sample. To address this mismatching, 6 female control participants were removed from the sample. All female control participants were given a z-score for age and a z-score for verbal IQ. These two z-scores were averaged, and the 6 female participants with the highest average z-score were excluded from the final sample.

After this adjustment, the autism group was composed of 23 males and 4 females, while the control group was composed of 18 males and 4 females. In the autism group, 4 children had a co-morbid diagnosis of ADHD, 1 child had comorbid mood disorder and OCD, 1 child had comorbid anxiety and ADD, and 1 child had comorbid secondary pragmatic learning disability. In the control group, 1 child had a diagnosis of ADHD and OCD tendencies, 1 child had ADHD, 1 child had short term sequential memory disorder, and 1 child had ADD and a reading learning disability.

The autism group had a mean age of 159.56 (SD = 32.27) and a mean verbal IQ of 97.93 (SD = 14.43). The control group had a mean age of 172.73 (SD = 24.55) and a mean verbal IQ of 104.77 (SD = 12.76). Although the control group had a higher average verbal IQ and an older average age than the autism group, independent samples t-tests did not show significant differences between the two groups on age, verbal IQ, or sex. The ethnic distribution for the sample was: 59.2% Hispanic (29 participants), 26.5% White, Non-Hispanic (13 participants), 6.1% Black, Non-Hispanic (3 participants), 6.1% mixed

Hispanic (3 participants), and 2.0% no response (1 participant). Table 1 presents demographic and descriptive information on participants.

Participants were separated into a younger age group ($N = 25$) and an older age group ($N = 24$). 175 months, or 14.6 years, was chosen as the dividing age for two reasons. First, this dividing age allowed for a maximum sample size in both age groups. Second, recent developmental research on the N170 component suggests that N170 amplitude may have different patterns in participants younger than 14-15 years old and participants older than 14-15 years old (Batty & Taylor, 2006).

Psychological Testing

Autism Diagnostic Observation Schedule

Participants were administered the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 1999). The ADOS is a semi-structured observational assessment that examines language and communication, reciprocal social interaction, imagination and creativity, and restricted and repetitive behaviors and interests in order to assess symptoms of pervasive developmental disorders. For its diagnostic algorithm, the ADOS focuses on critical items that assess communication and reciprocal social interaction. The ADOS is divided into four age-appropriate modules; participants in this study either received Module 3 or Module 4. For both modules, the participant and the experimenter engage in certain play/discussion activities, and the participant is evaluated on a scale of 0 (not abnormal) to 3 (very abnormal). The modules discriminate among children with autism, children with pervasive developmental disorder – not otherwise specified, and children with non-spectrum disorders. In this study, the ADOS was used to discriminate between participants with autism and typical development. In addition, the ADOS Social Interaction Subscale and the ADOS Total were used as indicators of individual differences.

Behavior Assessment Scale for Children

Participants completed with Behavior Assessment Scale for Children: Self Report of Personality (BASC: SRP; Reynolds & Kamphaus, 1998). Parents of participants completed the Behavior Assessment Scale for Children: Parent Rating Scales (BASC: PRS; Reynolds & Kamphaus, 1998). The Social Stress and Interpersonal Relations Subscales of the BASC: SRP and the Social Skills Subscale of the BASC: PRS were used as indicators of individual differences in this study. The Social Stress Subscale contains 12 items with an alpha of 0.81, the Interpersonal Relations Subscale contains 10 items with an alpha of 0.84, and the Social Skills Subscale contains 14 items with an alpha of 0.87. These scales were standardized and normed on a national sample of 3,483 parents and 9,861 children, and they have established reliability and validity.

Children's Eyes Test

Participants completed the Children's Eyes Test (Baron-Cohen et al., 2001). In this test, participants view 28 pictures of the eye region of the face. They select one of four words that best describe what the person in the picture is thinking or feeling. Children receive one point for each correct selection, with their total score ranging from 0-28 points. This test has been shown to discriminate between children with autism and typical development. In this study, the Children's Eyes Test was used as an indicator of individual differences.

Social Communication Questionnaire

Parents of participants completed the Social Communication Questionnaire (SCQ; Berument et al., 1999). This questionnaire was developed from the forty critical items of the Autism Diagnostic Interview (ADI), and it focuses on social interaction, communication, and repetitive and stereotyped behaviors. The SCQ is comparable to the ADI in discriminating autism spectrum disorders from non-autism spectrum disorders, autism from mental retardation, and autism from other autism spectrum disorders. In this study, the SCQ was used to discriminate between participants with autism and typical development. In addition, the SCQ Social Interaction Subscale and the SCQ Total were used as indicators of individual differences.

Social Responsiveness Scale

Parents of participants completed the Social Responsiveness Scale (SRS; Constantino, 2004; Constantino et al., 2003). This is a 65-item questionnaire that inquires about the participant's social functioning in natural social situations. The SRS has good test-retest reliability (0.83 to 0.88). The SRS seems to be a valid measure of social functioning, as it is independent of IQ and satisfactorily distinguishes among children with autism, Asperger Syndrome, pervasive developmental disorder – not otherwise specified, and typical development (Constantino et al., 2003). In this study, the SRS total score was used as an indicator of individual differences.

Wechsler Intelligence Scale for Children – IV

Participants completed an abbreviated version of the Wechsler Intelligence Scale for Children – IV (WISC; Williams, Weiss, and Rolfhus, 2003a, 2003b). The WISC has been normed on over 2,000 children and yields standardized estimates for a Verbal Comprehension Index and a Perceptual Reasoning Index (Williams et al., 2003a). The Vocabulary Scale and the Similarities Scale of the Verbal Comprehension Index were administered, and the Block Design Scale and the New Matrix Reasoning Scale of the Perceptual Reasoning Index were administered. These subscales were selected for many reasons: they have the highest loadings on the Verbal Comprehension Index and the Perceptual Reasoning Index, they have the strongest test-retest reliabilities, they have the best internal consistency estimates, and they have the smallest standard errors of measurement (Williams et al., 2003b). In this study, the WISC Verbal Comprehension Index was used to ensure that all study participants had a verbal IQ of 70 or greater.

Stimuli

The stimuli for the EEG face processing task were gray-scale photographs of vehicles and faces. These photographs were matched on size, luminance, and background color (white). The same photograph was never shown twice, although some photographs shared common attributes. For example, the same person may have been shown smiling in one photograph and not smiling in a second photograph. The face stimuli were taken from the NimStim Face Stimulus Set, which may be viewed at: www.macbrain.org. The vehicle stimuli were taken from a database of vehicle pictures, which may be viewed at: www.cars.com.

In the overall presentation, four blocks of stimuli were presented randomly: upright vehicles, inverted vehicles, upright faces, and inverted faces. Within the vehicle blocks, four sub-blocks of stimuli were presented randomly: passenger cars, sports cars, SUVs, and pick-up trucks. Within the face blocks also, four sub-blocks of stimuli were presented randomly: angry faces, fear faces, neutral faces, and happy faces. Figure 1 shows example stimuli. Within each sub-block, 30 photographs were presented randomly. Each photograph was presented for 500 msec, followed by a blank screen for 500 msec. The E-Prime software was used for presentation purposes.

Procedure

In a previous experimental session, participants completed the ADOS, BASC: SRP, Children's Eyes Test, and WISC. Parents of participants completed the BASC:

PRS, SCQ, and SRS. Pertinent demographic information was also collected in a previous session. In the current experimental session, participants completed an EEG face processing task and a behavioral face processing task. These tasks were ordered randomly across participants. The current paper will only report on the results of the EEG face processing task.

Before EEG data collection, a 128-lead Geodesic sensor net was dipped into a potassium-chloride electrolyte solution and fitted to the participant's head. The electrodes on the cap were evenly spaced, covering the scalp from the left ear to the right ear and from the nasion to the inion. Impedances were kept below 50 kΩ. EEG was recorded continuously.

During the face processing task, participants were seated approximately 20 inches from the computer monitor used for stimulus presentation. At the beginning of each vehicles block, participants were asked to count the number of vehicles facing left. For the upright vehicles block, there were 61 vehicles facing left; for the inverted vehicles block, there were also 61 vehicles facing left. At the beginning of each faces block, participants were asked to count the number of female faces. For the upright faces block, there were 50 female faces; for the inverted faces block, there were 49 female faces. Participants were asked to count the stimuli to ensure that they were paying attention. At the end of each block, participants reported the number of stimuli that they had counted.

The EEG signal was amplified (x1000) and filtered (0.1 Hz high-pass filter and 100 Hz elliptical low-pass filter) using a preamplifier system. The conditioned signal was multiplexed and digitized at 250 Hz using an analog-to-digital converter and a Macintosh computer. All 128 channels were continuously recorded and streamed to the computer's hard drive. A Dell computer, interfaced and synchronized via serial port, used e-Prime software to generate the stimuli. Stimulus onset and offset was recorded in order to later segment the data. The Cz electrode was used as the reference during data collection (data was re-referenced to an average reference after data collection).

Data Editing and Reduction

Data was digitally filtered at a low-pass of 30 Hz to reduce artifacts caused by environmental noise. Data were segmented into 4 categories: upright faces, upright vehicles, inverted faces, and inverted vehicles. Artifact detection was conducted on all electrode channels and data segments. An electrode channel was marked bad if the fast average amplitude of the channel exceeded 200 μV, if the differential average amplitude of the channel exceeded 100 μV, if the channel had zero variance, or if the channel had been marked bad in over 25% of the segments. A segment was marked bad if that segment contained more than 15 bad channels. Next, all segments were manually reviewed for artifacts and/or eye movements. If any artifacts were detected during manual review, the segment was marked as bad. The data in bad channels was replaced with data interpolated from nearby good channels, using spherical splines (Electrical Geodesics, Inc., 2004).

For each participant, data were averaged across trials by stimulus type. Thus, each participant had 4 average segments: upright faces, upright vehicles, inverted faces, and inverted vehicles. After this averaging process, the segments were again manually reviewed. The data were re-referenced to an average reference and baseline corrected, with the baseline interval specified as 100 msec before the sample.

Finally, values for the P1 amplitude, N170 amplitude, and N170 latency were extracted from each average data segment. The P1 is the positive ERP component that occurs before the N170 at approximately 100 msec after stimulus presentation. Recent research suggests that this component is associated with face processing (Herrmann, Ehlis, Muehlberger, & Fallgatter, 2005; Marzi & Viggiano, 2007). Therefore, for the P1 component, four electrodes over the left occipital scalp (65, 66, 70, and 71) and four electrodes over the right occipital scalp (84, 85, 90, and 91) were analyzed. These electrode sites were intentionally selected, as they match the electrode sites in O'Connor et al. (2005). The maximum amplitude value 79-139 msec after stimulus presentation was identified for each electrode site, and these values were averaged to create a left hemisphere amplitude and a right hemisphere amplitude. For the N170 component, six electrodes over the left lateral posterior scalp (58, 59, 64, 65, 69, and 70) and six electrodes over the right lateral posterior scalp (90, 91, 92, 95, 96, and 97) were analyzed. These electrode sites were intentionally selected, as they match the electrode sites in McPartland et al. (2004). Figure 2 displays the selected electrode sites for P1 and N170. The minimum amplitude value 143-231 msec after stimulus presentation was identified for each electrode site, and these values were averaged to create a left hemisphere amplitude and a right hemisphere amplitude. Similarly, the time to the first negative peak 143-231 msec after stimulus presentation was determined for each electrode site, and these values were averaged to create a left hemisphere latency and a right hemisphere latency.

CHAPTER 3: RESULTS

Power

The following statistical analyses are conducted with 27 participants with high-functioning autism and 22 control participants. At a 0.05 level of significance for a two-tailed test with a moderate effect size of 0.5, power was equal to 0.40. Thus, there was a 40% probability that this study would correctly reject a false null hypothesis. At a 0.05 level of significance for a one-tailed test (taking into account a priori directional hypotheses), it was possible to detect correlations at or above $r = 0.275$. Previous research in this field has found significant results with comparable sample sizes (Dawson, Webb, et al., 2004; McPartland et al., 2004; Dawson et al., 2005), thus power was not anticipated to be a problem in conducting simple analyses. However, there was not sufficient power to conduct complex analyses.

Group Differences

While the groups were not significantly different on age, verbal IQ, or sex, the control group had an older average age and a higher average verbal IQ than the autism group. Correlation analyses revealed that age was significantly correlated with N170 amplitude and P1 amplitude measures and marginally correlated with N170 latency measures. Thus, age was taken into account in the following analyses. Correlation analyses revealed that verbal IQ was not correlated with N170 amplitude, N170 latency, or P1 amplitude. Thus, verbal IQ was not taken into account in the following analyses.

As has been previously noted in research on children (Taylor et al., 1999), the N170 component in this study differed from the adult literature in that it did not extend below baseline. Nevertheless, the N170 component was clearly visible in the grand averaged ERP waveforms to upright faces, upright vehicles, inverted faces, and inverted vehicles. Figure 3 displays these grand averaged waveforms for children with autism and typical development, and Figure 4 displays these grand averaged waveforms for younger children and older children.

Repeated measures ANOVAs were conducted on N170 amplitude, N170 latency, and P1 amplitude. Diagnostic group (autism vs. typical development) and dichotomous age (younger than 175 months vs. older than 175 months) were the between-subjects factors. Orientation (upright vs. inverted), stimulus type (face vs. vehicle), and hemisphere (left vs. right) were the within-subjects factors. Main effects and two-way interactions are reported below. Three-way interactions and higher were not considered because this study had insufficient power for the reliable interpretation of these interactions.

N170 Amplitude

In the following analyses, the N170 component frequently did not extend below baseline, and thus was often observed as a negative peak but with a positive amplitude. This complicates the interpretation of the N170 amplitude. In this study, a more positive value for the N170 was indicative of a more shallow (smaller amplitude) component, while a less positive value was indicative of a deeper (greater amplitude) component (see Figures 3 and 4).

Diagnostic Group Effects

Table 2 shows N170 amplitude values for all conditions. No main effects of diagnostic group on N170 amplitude were seen in this study. Nevertheless, one significant interaction and one marginal interaction involving diagnostic group were observed. There was a significant interaction between group and orientation on N170 amplitude, $F(1, 45) = 6.464$, $p = 0.015$, $\eta^2_p = 0.126$. Post hoc within group analyses indicated that the processing of upright stimuli was associated with significantly greater amplitudes (deeper N170 components) than the processing of inverted stimuli for children with autism, $F(1, 25) = 22.498$, $p < 0.001$, $\eta^2_p = 0.474$, and children with typical development, $F(1, 20) = 58.137$, $p < 0.001$, $\eta^2_p = 0.744$. This effect was stronger in children with typical development (see Figure 5), and the significant interaction reflected this effect size difference.

There was also a marginal interaction between group and stimulus type, $F(1, 45) = 3.504$, $p = 0.068$, $\eta^2_p = 0.072$. Post hoc within group analyses indicated that vehicle processing was associated with significantly greater amplitudes than face processing for children with typical development, $F(1, 20) = 12.642$, $p = 0.002$, $\eta^2_p = 0.387$, but not children with autism, $F(1, 25) = 0.001$, $p = 0.970$, $\eta^2_p < 0.001$ (see Figure 6).

Dichotomous Age Effects

The analyses revealed a main effect of dichotomous age on N170 amplitude, $F(1, 45) = 11.383$, $p = 0.002$, $\eta^2_p = 0.202$, such that older children had significantly greater N170 amplitudes (deeper N170 components) than younger children.

There was a significant interaction between dichotomous age and orientation, $F(1, 45) = 10.032$, $p = 0.003$, $\eta^2_p = 0.182$. Post hoc within group analyses indicated that the processing of upright stimuli was associated with significantly greater amplitudes than the processing of inverted stimuli for younger children, $F(1, 23) = 49.943$, $p < 0.001$, $\eta^2_p = 0.685$, and older children, $F(1, 22) = 29.648$, $p < 0.001$, $\eta^2_p = 0.574$. This effect was stronger in younger children, and the significant interaction reflected this effect size difference. Post hoc between group analyses indicated that older children displayed significantly greater amplitudes than younger children for the processing of upright stimuli, $F(1, 45) = 7.282$, $p = 0.010$, $\eta^2_p = 0.139$, and inverted stimuli, $F(1, 45) = 14.931$, $p < 0.001$, $\eta^2_p = 0.249$. This effect was stronger for inverted stimuli, and the significant interaction reflected this effect size difference.

There was also a marginal interaction between dichotomous age and hemisphere, $F(1, 45) = 3.250$, $p = 0.078$, $\eta^2_p = 0.067$. Post hoc within group analyses indicated that recordings from the left hemisphere yielded significantly greater amplitudes than recordings from the right hemisphere for younger children, $F(1, 23) = 6.077$, $p = 0.022$,

$\eta^2_p = 0.209$, but not older children, $F(1, 22) = 0.326$, $p = 0.574$, $\eta^2_p = 0.015$. Post hoc between group analyses indicated that older children had significantly greater amplitudes than younger children when the N170 was recorded from the left hemisphere, $F(1, 45) = 7.355$, $p = 0.009$, $\eta^2_p = 0.140$, and the right hemisphere, $F(1, 45) = 13.279$, $p = 0.001$, $\eta^2_p = 0.228$. This effect was stronger for the right hemisphere, and the significant interaction reflected this effect size difference.

Other Effects

Other effects included a main effect of orientation, $F(1, 45) = 78.527$, $p < 0.001$, $\eta^2_p = 0.636$, such that the processing of upright stimuli was associated with significantly greater N170 amplitudes (deeper N170 components) than the processing of inverted stimuli. There was a main effect of hemisphere, $F(1, 45) = 5.752$, $p = 0.021$, $\eta^2_p = 0.113$, such that recordings from the left hemisphere yielded significantly greater amplitudes than recordings from the right hemisphere. There was also a marginal main effect of stimulus type, $F(1, 45) = 3.285$, $p = 0.077$, $\eta^2_p = 0.068$, such that vehicle processing was associated with marginally greater amplitudes than face processing.

There was a significant interaction between orientation and stimulus type, $F(1, 45) = 6.861$, $p = 0.012$, $\eta^2_p = 0.132$. Post hoc analyses indicated that the processing of upright stimuli was associated with significantly greater amplitudes than the processing of inverted stimuli when the stimuli were faces, $F(1, 45) = 28.969$, $p < 0.001$, $\eta^2_p = 0.392$, and vehicles, $F(1, 45) = 78.081$, $p < 0.001$, $\eta^2_p = 0.634$. This effect was stronger for vehicles, and the significant interaction reflected this effect size difference. Post hoc analyses also indicated that vehicle processing was associated with significantly greater amplitudes than face processing when the stimuli were upright, $F(1, 45) = 11.025$, $p = 0.002$, $\eta^2_p = 0.197$, but not when the stimuli were inverted, $F(1, 45) = 0.027$, $p = 0.870$, $\eta^2_p = 0.001$.

There was also a significant interaction between orientation and hemisphere, $F(1, 45) = 5.549$, $p = 0.023$, $\eta^2_p = 0.110$. Post hoc analyses indicated that the processing of upright stimuli was associated with significantly greater amplitudes than the processing of inverted stimuli when the N170 was recorded from the left hemisphere, $F(1, 45) = 50.064$, $p < 0.001$, $\eta^2_p = 0.527$, and the right hemisphere, $F(1, 45) = 76.793$, $p < 0.001$, $\eta^2_p = 0.631$. This effect was stronger for the right hemisphere, and the significant interaction reflected this effect size difference. Post hoc analyses also indicated that recordings from the left hemisphere had significantly greater amplitudes than recordings from the right hemisphere when the stimuli were inverted, $F(1, 45) = 8.591$, $p = 0.005$, $\eta^2_p = 0.160$, but not when the stimuli were upright, $F(1, 45) = 2.599$, $p = 0.114$, $\eta^2_p = 0.055$.

N170 Latency

Diagnostic Group Effects

Table 3 shows N170 latency values for all conditions. Analyses revealed a main effect of diagnostic group, $F(1, 45) = 6.160$, $p = 0.017$, $\eta^2_p = 0.120$, such that children with autism had significantly longer N170 latencies than children with typical development (see Table 3). No other significant group effects were noted in the analysis of N170 latency.

Dichotomous Age Effects

There was a main effect of dichotomous age, $F(1, 45) = 5.429$, $p = 0.024$, $\eta^2_p = 0.108$, such that younger children had significantly longer N170 latencies than older children (see Table 3). No other significant age effects were noted in the analysis of N170 latency.

Other Effects

There was a main effect of orientation, $F(1, 45) = 26.452$, $p < 0.001$, $\eta^2_p = 0.370$, such that the processing of inverted stimuli was associated with significantly longer N170 latencies than the processing of upright stimuli. There was a main effect of stimulus type, $F(1, 45) = 73.612$, $p < 0.001$, $\eta^2_p = 0.621$, such that vehicle processing was associated with significantly longer N170 latencies than face processing. There was also a main effect of hemisphere, $F(1, 45) = 4.215$, $p = 0.046$, $\eta^2_p = 0.086$, such that recordings from the left hemisphere yielded significantly longer latencies than recordings from the right hemisphere.

There was a significant interaction between orientation and stimulus type, $F(1, 45) = 4.109$, $p = 0.049$, $\eta^2_p = 0.084$. Post hoc analyses indicated that the processing of inverted stimuli was associated with significantly longer latencies than the processing of upright stimuli when the stimuli were faces, $F(1, 45) = 23.578$, $p < 0.001$, $\eta^2_p = 0.344$, and vehicles, $F(1, 45) = 4.241$, $p = 0.045$, $\eta^2_p = 0.086$. This effect was stronger for faces, and the significant interaction reflected this effect size difference. Post hoc analyses also indicated that vehicle processing was associated with significantly longer latencies than face processing when the stimuli were upright, $F(1, 45) = 84.324$, $p < 0.001$, $\eta^2_p = 0.652$, and inverted, $F(1, 45) = 27.863$, $p < 0.001$, $\eta^2_p = 0.382$. This effect was stronger for upright stimuli, and the significant interaction reflected this effect size difference.

P1 Amplitude

Diagnostic Group Effects

Table 4 shows P1 amplitude values for all conditions. The N170 component may not have extended below baseline in this study because of the strength of the P1 component. No main effects of diagnostic group on P1 amplitude were observed in this study. Nevertheless, one interaction and one marginal interaction involving diagnostic group were observed. There was a significant interaction between group and orientation, $F(1, 45) = 5.142$, $p = 0.028$, $\eta^2_p = 0.103$. Post hoc within group analyses indicated that the processing of inverted stimuli was associated with significantly greater P1 amplitudes than the processing of upright stimuli for children with typical development, $F(1, 20) = 36.523$, $p < 0.001$, $\eta^2_p = 0.646$, but not children with autism, $F(1, 25) = 2.025$, $p = 0.167$, $\eta^2_p = 0.075$ (see Figure 7).

There was also a marginal interaction between group and hemisphere, $F(1, 45) = 3.974$, $p = 0.081$, $\eta^2_p = 0.081$. Post hoc within group analyses indicated that recordings from the right hemisphere yielded significantly greater amplitudes than recordings from the left hemisphere for children with typical development, $F(1, 20) = 22.663$, $p < 0.001$, $\eta^2_p = 0.531$, but not children with autism, $F(1, 25) = 0.518$, $p = 0.478$, $\eta^2_p = 0.020$ (see Figure 8).

Dichotomous Age Effects

There was a main effect of dichotomous age, $F(1, 45) = 15.092$, $p < 0.001$, $\eta^2_p = 0.251$, such that younger children had significantly greater P1 amplitudes than older children.

There was a significant interaction between dichotomous age and stimulus type, $F(1, 45) = 5.018$, $p = 0.030$, $\eta^2_p = 0.100$. Post hoc within group analyses indicated that face processing was associated with significantly greater P1 amplitudes than vehicle processing for older children, $F(1, 22) = 12.825$, $p = 0.002$, $\eta^2_p = 0.368$, but not younger children, $F(1, 23) = 0.410$, $p = 0.528$, $\eta^2_p = 0.018$. Post hoc between group analyses also indicated that younger children had significantly greater amplitudes than older children when the stimuli were faces, $F(1, 45) = 12.047$, $p = 0.001$, $\eta^2_p = 0.211$, and vehicles, $F(1, 45) = 17.664$, $p < 0.001$, $\eta^2_p = 0.282$. This effect was stronger for vehicles, and the significant interaction reflected this effect size difference.

Other Effects

There was a main effect of orientation, $F(1, 45) = 20.087$, $p < 0.001$, $\eta^2_p = 0.309$, such that the processing of inverted stimuli was associated with significantly greater P1 amplitudes than the processing of upright stimuli. There was also a main effect of hemisphere, $F(1, 45) = 9.777$, $p = 0.003$, $\eta^2_p = 0.178$, such that recordings from the right hemisphere yielded significantly greater P1 amplitudes than recordings from the left hemisphere.

Individual Differences

Correlations between ERP measures (N170 amplitude, N170 latency, and P1 amplitude for upright faces, inverted faces, upright vehicles, and inverted vehicles) and autistic symptomology and social behavior measures were conducted separately for children with autism and children with typical development. The autistic symptomology and social behavior measures included the ADOS Social Interaction Subscale and ADOS Total Score; the BASC Self-Report (SRP) Social Stress and Interpersonal Relations Subscales; the BASC Parent-Report (PRS) Social Skills Subscale; the Children's Eyes Test of social cognition; the SCQ Social Interaction Subscale and SCQ Total Score; and the SRS Total Score. For the ERP measures, the right hemisphere and left hemisphere values were averaged, and these average values were used in correlation computations. For each ERP measure (N170 amplitude, N170 latency, and P1 amplitude), a total of 36 correlations were computed.

For children with autism, 0 correlations with N170 amplitude were significant, 1 correlation with N170 latency was significant, and 0 correlations with P1 amplitude were significant. Partial correlations were then performed with the same variables, controlling for continuous age. Taking age into consideration, 0 correlations with N170 amplitude were significant, 2 correlations with N170 latency were significant, and 0 correlations with P1 amplitude were significant. The number of significant correlations observed in these analyses did not exceed the number expected by chance. Therefore, little evidence was observed of a significant relation between ERP measures and social behavior and autistic symptomology measures for the autism group.

For children with typical development, 11 correlations with N170 amplitude were significant, 1 correlation with N170 latency was significant, and 13 correlations with P1 amplitude were significant. Partial correlations were again performed with the same variables, controlling for continuous age. Taking age into consideration, 2 correlations with N170 amplitude were significant, 0 correlations with N170 latency were significant, and 14 correlations with P1 amplitude were significant. For children with typical development, there was some evidence of a relation between N170 amplitude and social behavior and autistic symptomology measures, but this relation did not hold when age was considered. However, there was evidence of a significant relation between P1 amplitude and individual differences on social behavior and autistic symptomology measures in the typical development group, even when variance in age was considered (see Table 5).

Controlling for age, P1 amplitude was positively correlated with the ADOS Social Interaction Subscale, ADOS Total Score, and SCQ Social Interaction Subscale and negatively correlated with the Children's Eyes Test in children with typical development. Table 5 shows the partial correlations between P1 amplitude and these four measures in children with autism and typical development. Thus, children with typical development and large P1 amplitudes displayed evidence of less well developed or organized social skills on the ADOS and SCQ, as well as poorer performance on measures of social cognition. Although these data could not be explained in terms of age effects, they are consistent with the observation that younger and presumably less mature children tended to display greater P1 amplitudes in this study.

CHAPTER 4: DISCUSSION

Overview

The primary objectives of this research project focused on describing the N170 component in individuals with autism. However, as the data were analyzed, it became increasingly apparent that the N170 component could not be examined without also considering the proceeding positive peak, the P1 component. The P1 component seems to be an early index for processing visual stimuli, including but not limited to faces (Taylor et al., 2001). The P1 component has not been widely characterized, and the functional difference between the P1 component and the N170 component is not well understood. Itier and Taylor (2002) theorize that the P1 component reflects holistic processing, or the perception of a face as a face, while the N170 component reflects the relational processing of internal facial features. As the P1 component had a large, positive amplitude in this study, it may have influenced the amplitude of the N170 component.

Overall, the results adhered more closely to the predicted child pattern of results than the predicted adult pattern. The results for N170 latency and P1 amplitude were fairly straightforward and consistent with the literature while the results for N170 amplitude were less straightforward and consistent with the literature. Analyses of P1 amplitude and N170 amplitude indicated that individuals with autism did not differentiate among stimuli to the same degree as individuals with typical development. N170 was also delayed for children with autism. Finally, the P1 component was more indicative of individual differences than the N170 component. P1 amplitude was related to social behavior and autistic symptomology in children with typical development, but not children with autism.

As the P1 amplitude seems to have influenced the N170 amplitude, the P1 component will be discussed first to set the groundwork for later discussion of the N170 component. Similarly, age effects will be discussed before group effects to provide a foundation for the discussion of group effects. In the following discussion, not all results will be explicitly addressed; only the most pertinent and theoretically meaningful results will be discussed.

Group Differences

P1 Amplitude

P1 amplitude decreased considerably with age, as is consistent with previous research on children (Batty & Taylor, 2006; Itier & Taylor, 2004a, 2004b). Theory suggests that neural circuitry specific to face processing changes with age. Younger children tend to generally show face-preferential processing in the posterior ventral processing stream, while older children and adults tend to specifically show face-preferential processing in the fusiform face area (Gathers, Bhatt, Corbly, Farley, & Joseph, 2004; Passarotti, Paul, & Stiles, 2001). Thus, it seems likely that the high P1 amplitude in younger children is reflective of more effortful processing, while the low P1 amplitude in older children is reflective of less effortful processing. As face processing expertise increases, automaticity of processing increases, neuroanatomy becomes more specialized, and less mental effort is needed to recognize a face as a face.

In older children, P1 amplitude may reflect motivation level, rather than processing difficulty. For children with more established face processing skills, a high P1 amplitude may indicate greater interest and motivation in the stimulus. This theoretical supposition explains some interaction effects seen with P1 amplitude. For example, the interaction between age and stimulus type indicated that faces elicited a significantly larger amplitude than vehicles in older children, but not younger children. For younger children, processing difficulty may have trumped all effects of motivation. However, older children with more face processing experience may be showing a greater interest in face processing than vehicle processing, as indicated by a larger P1 amplitude.

P1 amplitude also references processing differences between children with autism and children with typical development. Children with typical development showed a greater P1 amplitude to inverted faces relative to upright faces, while children with autism did not show any evidence of differential processing. Inverted faces disrupt configural processing (Freire, Lee, & Symons, 2000), making inverted faces more difficult to process than upright faces. For individuals with typical development, the greater P1 amplitude to inverted faces relative to upright faces may reflect more effortful processing. Since individuals with autism do not show the same behavioral disadvantage for processing inverted faces (Langdell, 1978), it is not surprising that they also don't show a differential P1 amplitude to upright and inverted faces. Happé, Frith, and colleagues (e.g. Happé, 1999) suggest that individuals with autism have an abnormal processing style termed "weak central coherence". They argue that individuals with autism are biased toward local processing and have difficulty with configural processing. These results are consistent with the weak central coherence theory, as individuals with autism do not show an advantage (a lower P1 amplitude) for processing upright faces.

In this study, the P1 component was only lateralized to the right hemisphere in children with typical development, not children with autism. In older children with typical development, face processing typically occurs in the right hemisphere (Taylor, Batty, & Itier, 2004), suggesting that individuals with autism may have a right hemisphere deficit and/or may employ an atypical face processing strategy. If children with autism employ a qualitatively different face processing strategy from children with typical development, this may call into question the meaning of N170 in children with autism. The N170 may not index face processing in the same way within these two populations, and future studies that compare the N170 across different populations should be aware of this potential confound.

Interestingly, the P1 component in this study looked similar to the N170 component in the adult literature. The P1 in children may be capturing some of the same information that the N170 typically captures in adults. This suggests that the P1 is a more sensitive indicator of face encoding in childhood while the N170 is a more sensitive indicator of face encoding in adulthood. Regardless of its relation with N170, the P1 clearly marks early stages of face processing, and it is notable that children with autism and children with typical development show face processing differences even at this early stage.

N170 Amplitude

As previously noted, the N170 component generally did not extend below baseline. Thus, most of the amplitude values for this component were actually positive. The amplitude for the P1 component was unusually high, and this heightened amplitude may have carried over to the N170 component. It is not clear whether age effects on the N170 were simply the result of "carry over" age effects from the P1 or whether age independently influenced the N170.

Surprisingly, there was a marginal main effect of stimulus type on N170 such that vehicles had a greater (more negative) amplitude than faces. Since the N170 component is thought to be specific to face processing (Bentin et al., 1996), this was a most unexpected finding. The research on adults has systematically tested the specificity of the N170 component, but the research on children has only marginally addressed the specificity of this component. It is not clear whether the N170 is a face-specific component across development or whether the N170 becomes a face-specific component with development.

Consistent with previous studies (Itier & Taylor, 2004a; Taylor et al., 1999), N170 amplitude increased with age. This developmental effect, taken together with the surprising effect of stimulus type, may suggest that the N170 is reflective of expert processing, rather than face processing. As children gain more experience and expertise with face processing, the N170 component increases in amplitude and may then show a differential response to faces. Most adults are expert face processors, thus the N170 often appears specific to faces in adults; however, Rossion et al. (2002) argue that the N170 actually appears in response to any visual stimulus of expertise. Since the N170 component isn't fully mature until adulthood and there is limited evidence of the face-specificity of this component in childhood, the N170 may indeed index expert processing, rather than face processing.

In this study, age interacted with stimulus orientation and hemisphere. Past research has shown that both of these variables are influenced by development. Previous studies suggest that upright faces have a greater amplitude than inverted faces in younger children, but inverted faces have a greater amplitude than upright faces in older children and/or adults (Itier & Taylor, 2004a, 2004b; Taylor et al., 2001). Previous studies also suggest that younger children do not have a consistent hemisphere preference for face processing (Itier & Taylor, 2004b; Taylor et al., 1999; Taylor et al., 2001) while older children have a preference for the right hemisphere (Batty & Taylor, 2006; Itier & Taylor, 2004b; Taylor et al., 2001). The present study replicated both of these developmental effects for younger children and showed a trend toward replicating these developmental effects for older children. However, the developmental effects for older children were never fully realized in this study. There are a few possible explanations for this result.

First, in this study, P1 amplitude was significantly higher for inverted stimuli compared to upright stimuli and right hemisphere recordings compared to left hemisphere recordings. Since initial P1 amplitude isn't accounted for by N170 amplitude, this measure may be a biased indicator of amplitude. Second, most previous studies have employed a target detection task while the present study employed a passive counting task. This task may not have required the same degree of effortful processing as a target detection task, thus the developmental influences on amplitude weren't fully realized. Third, age was examined dichotomously in this sample. Younger children ranged in age from 108-175 months, and older children ranged in age from 175-209 months. This dichotomous measure of age may not have been sensitive enough to detect the full developmental course of N170 amplitude. Fourth, the sample may have been too young to show the full developmental pattern of N170. Since N170 amplitude continues to mature until adulthood (Itier & Taylor, 2004a; Itier & Taylor, 2004b), the full developmental course of N170 amplitude may only be realized when adults are included in the study.

As seen with the P1 component, children with typical development showed a differential N170 amplitude response to different stimuli (upright stimuli vs. inverted stimuli and faces vs. vehicles), while children with autism did not show this differential amplitude response. Children with autism don't seem to assign meaning to stimuli in the same way as children with typical development; they approach most stimuli with a steady level of interest and mental processing. In sharp contrast, children with typical development almost immediately (within 200 msec) adjust their level of interest and mental processing to match stimulus meaning.

It is difficult to clearly interpret diagnostic group differences in electrophysiology, as the neuroanatomical region of study is usually indicated from the typical development literature. Individuals with autism don't use the fusiform gyrus for face processing to the same extent as individuals with typical development (Pierce, Muller, Ambrose, Allen, & Courchesne, 2001), thus electrophysiological recordings are compared from an expert processing area in children with typical development to a non-expert processing area in children with autism. A non-expert face processing area may not be a valid or sensitive measure of face processing in autism, making it difficult to tease apart the effects of diagnostic group and neuroanatomical location. Although children with autism do not show differential stimulus processing in this study, it is important to note that the electrodes of interest were chosen from the typical development literature. While these electrode sites are sensitive to differential stimulus processing in typical development, they may not be sensitive to differential stimulus processing in autism.

N170 Latency

Corroborating previous studies, younger children had a longer N170 latency than older children (Batty & Taylor, 2006; Itier & Taylor, 2004a, 2004b; Taylor et al., 1999; Taylor et al., 2001), and individuals with autism had a longer N170 latency than individuals with typical development (O'Connor et al., 2005; O'Connor et al., 2007). In general, it is not uncommon for ERPs to be delayed in individuals with autism compared to individuals with typical development (Jansson-Verkasalo et al., 2003; Jansson-Verkasalo et al., 2005; Townsend et al., 2001), so this delayed latency effect may not be specific to the N170.

Research has demonstrated cortical underconnectivity in autism (Just, Cherkassky, Keller, Kana, & Minshew, 2007), and this underconnectivity may explain why individuals with autism show delayed ERPs. Reduced connectivity makes it difficult for cortical areas to quickly exchange information with one another. Complex information processing is particularly dependent on the efficient communication of different brain regions, thus delayed ERPs may be particularly apparent in complex information processing tasks, such as face processing tasks.

Individual Differences

For individuals with autism, there was no evidence that ERP measures were related to social behavior and autistic symptomology measures. For individuals with typical development, P1 amplitude was related to the ADOS Social Interaction Subscale, ADOS Total, SCQ Social Interaction Subscale, and the Children's Eyes Test, after controlling for age. These results suggest that the P1 and N170 components are not sensitive to individual differences in autism. However, the P1 component does seem to index individual differences in typical development. As P1 amplitude increased, social deficits and autistic symptomology increased and face/eye processing ability decreased. Children with autism and children with typical development had similar variances in their ERP measures, so variance differences cannot explain this divergent pattern of results. As noted earlier, the P1 component seems to be a more sensitive indicator of face processing in childhood than the N170 component.

If face processing is a primary deficit in autism, electrophysiological measures of face processing should be related to individual differences in social behavior and autistic symptomology. The absence of this relation suggests that face processing may not be a primary deficit in autism, or may not be *as* primary of a deficit in autism. While autism is clearly characterized by face processing difficulties, these difficulties may be secondary to a more central deficit, such as an impairment in social orienting.

Limitations and Future Directions

There were several limitations of this study. First, age was used as a dichotomous variable. Given the significant effect of development on N170, age may have been better used as a continuous variable. Dichotomizing age resulted in a loss of information, making it more difficult to detect specific developmental patterns. Second, the autism group and the typical development group were poorly matched in this study. Although there were no significant differences between groups, the typical development group tended to be older and have a higher verbal IQ than the autism group. It is difficult to evaluate group effects when the groups are only marginally matched. Third, sample size was small, resulting in a limited ability to detect between group differences.

Fourth, the N170 was evaluated over occipital-temporal brain regions. Some literature suggests that the N170 should be measured over occipital-temporal brain regions for adults and occipital-parietal brain regions for children (O'Connor et al., 2005). Thus, the data may have been more accurate with a different choice of electrode sites. Lastly, research suggests that the N170 component is bifed in young children, with the two divisions of the N170 merging into one component by 10 to 13 years of age (Taylor et al., 2004). The present study isolated the single most negative peak within the appropriate time window, thus bifed properties of the N170 were not investigated.

Future research should consider using the difference score between P1 amplitude and N170 amplitude as a more accurate measure of N170 amplitude. In this study, the difference score was not used, as the P1 component and the N170 component were analyzed from different sets of electrodes. In future research, however, the difference score would provide an estimate of N170 amplitude, independent of P1 amplitude. Future research also needs to examine the specificity of the N170 component to faces in childhood. A thorough investigation of the development of N170 may clarify whether N170 is a marker of face processing or expert processing. In addition, future developmental studies of face processing should consider focusing on the P1 component in childhood and the N170 component in adulthood.

Finally, the P1 and N170 components are only beginning to be investigated in autism. Future research needs to more definitively determine group differences on these components, as studies have found somewhat different results. In addition, both the P1 and N170 components should continue to be evaluated as potential markers for individual differences. If the P1 component and the N170 component are not related to individual differences in autism, it may be important to reconsider the role of face processing in this population.

REFERENCES

Adolphs, R., Sears, L., & Piven, J. (2001). Abnormal processing of social information from faces in autism. *Journal of Cognitive Neuroscience, 13,* 232-240.

American Psychiatric Association. (2000). *Diagnostic and statistic manual of mental disorders* (4th ed., TR). Washington, D.C.: American Psychiatric Association.

Anderson, C. J., Colombo, J., & Shaddy, D. J. (2006). Visual scanning and papillary responses in young children with autism spectrum disorder. *Journal of Clinical and Experimental Neuropsychology, 28,* 1238-1256.

Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The "Reading the Mind in the Eyes" Test Revised Version: A study with normal adults, and adults with Asperger Syndrome or high functioning autism. *Journal of Child Psychology and Psychiatry, 42,* 241-251.

Batty, M. & Taylor, M. J. (2006). The development of emotional face processing during childhood. *Developmental Science, 9,* 207-220.

Bentin, S., Allison, T., Puce, A., Perez, E., & McCarthy, G. (1996). Electrophysiological studies of face perception in humans. *Journal of Cognitive Neuroscience, 8,* 551-565.

Bentin, S., Deouell, L. Y., & Soroker, N. (1999). Selective visual streaming in face recognition: Evidence from developmental prosopagnosia. *NeuroReport, 10,* 823-827.

Berument, S., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *British Journal of Psychiatry, 175*, 444-451.

Blass, E. M. (1999). The ontogeny of human infant face recognition: Orogustatory, visual, and social influences. In Philippe Rochat (Ed.), *Early social cognition: Understanding others in the first months of life* (pp.35-65). Mahwah, NJ: Erlbaum.

Brooks, R. & Meltzoff, A. N. (2005). The development of gaze following and its relation to language. *Developmental Science, 8,* 535-543.

Celani, G., Battacchi, M. W., & Arcidiacono, L. (1999). The understanding of the emotional meaning of facial expressions in people with autism. *Journal of Autism and Developmental Disorders, 29,* 57-66.

Cole, J. (2001). Empathy needs a face. *Journal of Consciousness Studies, 8,* 51-68.

Constantino, J. (2004). *The Social Responsiveness Scale*. Los Angeles, Western Psychological Services.

Constantino, J. Davis, S., Todd, R., Schindler, M., Gross, M. M., Brophy, S. L., et al. (2003). Validation of a brief quantitative measure of autistic traits: Comparison of the Social Responsiveness Scale with the Autism Diagnostic Interview – Revised. *Journal of Autism and Developmental Disorders, 33,* 427-433.

Dawson, G., Carver, L., Meltzoff, A. N., Panagiotides, H., McPartland, J., & Webb, S. J. (2002). Neural correlates of face and object recognition in young children with autism spectrum disorder, developmental delay, and typical development. *Child Development, 73,* 700-717.

Dawson, G. & Lewy, A. (1989). Arousal, attention, and the social impairments of individuals with autism. In G. Dawson (Ed.), *Autism: Nature, diagnosis, and treatment* (pp. 49-74). New York: Guilford.

Dawson, G., Meltzoff, A. N., Osterling, J., Rinaldi, J., & Brown, E. (1998). Children with autism fail to orient to naturally occurring social stimuli. *Journal of Autism and Developmental Disorders, 28,* 479-485.

Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., et al. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology, 40,* 271-283.

Dawson, G., Webb, S. J., Carver, L., Panagiotides, H., & McPartland, J. (2004). Young children with autism show atypical brain responses to fearful versus neutral facial expressions of emotion. *Developmental Science, 7,* 340-359.

Dawson, G., Webb, S. J., Wijsman, E., Schellenberg, G., Estes, A., Munson, J., et al. (2005). Neurocognitive and electrophysiological evidence of altered face processing in parents of children with autism: Implications for a model of abnormal development of social brain circuitry. *Development and Psychopathology, 17,* 679-697.

Eimer, M. (2000). Effects of face inversion on the structural encoding and recognition of faces: Evidence from event-related brain potentials. *Cognitive Brain Research, 10,* 145-158.

Eimer, M. & McCarthy, R. A. (1999). Prosopagnosia and structural encoding of faces: Evidence from event-related potentials. *NeuroReport, 10,* 255-259.

Electrical Geodesics, Inc. (2004). Net Station Waveform Tools: Technical Manual. Eugene: Electrical Geodesics, Inc.

Freire, A., Lee, K., & Symons, L. A. (2000). The face-inversion effect as a deficit in the encoding of configural information: Direct evidence. *Perception, 29,* 159-170.

Gathers, A. D., Bhatt, R., Corbly, C. R., Farley, A. B., & Joseph, J. E. (2004). Developmental shifts in cortical loci for face and object recognition. *Neuroreport: For Rapid Communication of Neuroscience Research, 15,* 1549-1553.

Gauthier, I. & Curby, K. M. (2005). A perceptual traffic jam on highway N170: Interference between face and car expertise. *Current Directions in Psychological Science, 14,* 30-33.

Gauthier, I., Skudlarski, P., Gore, J. C., & Anderson, A. W. (2000). Expertise for cars and birds recruits brain areas involved in face recognition. *Nature Neuroscience, 3,* 191-197.

Gillberg, C., Ehlers, S., Schaumann, H., Jakobsson, G., Dahlgren, S. O., Lindblom, R., et al. (1990). Autism under age 3 years: A clinical study of 28 cases referred for autistic symptoms in infancy. *Journal of Child Psychology and Psychiatry, 31,* 921-934.

Greenough, W. T., Black, J. E., & Wallace, C. S. (1987). Experience and brain development. *Child Development, 58,* 539-559.

Happé, F. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences, 3,* 216-222.

Herrmann, M. J., Ehlis, A. C., Muehlberger, A., & Fallgatter, A. J. (2005). Source localization of early stages of face processing. *Brain Topography, 18,* 77-85.

Hollich, G., Newman, R. S., & Jusczyk, P. W. (2005). Infants' use of synchronized visual information to separate streams of speech. *Child Development, 76,* 598-613.

Itier, R. J. & Taylor, M. J. (2002). Inversion and contrast polarity reversal affect both encoding and recognition processes of unfamiliar faces: A repetition study using ERPs. *NeuroImage, 15,* 353-372.

Itier, R. J. & Taylor, M. J. (2004a). Effects of repetition and configural changes on the development of face recognition processes. *Developmental Science, 7,* 469-487.

Itier, R. J. & Taylor, M. J. (2004b). Face recognition memory and configural processing: A developmental ERP study using upright, inverted, and contrast-reversed faces. *Journal of Cognitive Neuroscience, 16,* 487-502.

Izard, C., Fine, S., Schultz, D., Mostow, A., Ackerman, B., & Youngstrom, E. (2001). Emotion knowledge as a predictor of social behavior and academic competence in children at risk. *Psychological Science, 12,* 18-23.

Jansson-Verkasalo, E., Ceponiene, R., Kielinen, M., Suominen, K., Jantti, V., Linna, S., et al. (2003). Deficient auditory processing in children with Asperger Syndrome, as indexed by event-related potentials. *Neuroscience Letters, 338,* 197-200.

Jansson-Verkasalo, E., Kujala, T., Jussila, K., Mattila, M. L., Moilanen, I., Naatanen, R., et al. (2005). Similarities in the phenotype of the auditory neural substrate in children with Asperger syndrome and their parents. *European Journal of Neuroscience, 22,* 986-990.

Just, M. A., Cherkassky, V. L., Keller, T. A., Kana, R. K., & Minshew, N. J. (2007). Functional and anatomical cortical underconnectivity in autism: Evidence from an fMRI study of an executive function task and corpus callosum morphometry. *Cerebral Cortex, 17,* 951-961.

Klin, A. (1991). Young autistic children's listening preferences in regard to speech: A possible characterization of the symptom of social withdrawal. *Journal of Autism and Developmental Disorders, 21,* 29-42.

Koegel, R. L. & Frea, W. D. (1993). Treatment of social behavior in autism through the modification of pivotal social skills. *Journal of Applied Behavior Analysis, 26,* 369-377.

Langdell, T. (1978). Recognition of faces: An approach to the study of autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 19,* 255-268.

Leekam, S. R. & Ramsden, C. A. H. (2006). Dyadic orienting and joint attention in preschool children with autism. *Journal of Autism and Developmental Disorders, 36,* 185-197.

Lepisto, T., Kujala, T., Vanhala, R., Alku, P., Huotilainen, M., & Naatanen, R. (2005). The discrimination of and orienting to speech and non-speech sounds in children with autism. *Brain Research, 1066,* 147-157.

Lord, C., Rutter, M., Dilavore, P., & Risi, S. (1999). *Manual: Autism Diagnostic Observation Schedule*. Los Angeles, CA: Western Psychological Services.

Marzi, T. & Viggiano, M. (2007). Interplay between familiarity and orientation in face processing: An ERP study. *International Journal of Psychophysiology, 65,* 182-192.

McPartland, J., Dawson, G., Webb, S. J., Panagiotides, H., & Carver, L. J. (2004). Event related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry, 45,* 1235-1245.

Morales, M., Mundy, P., & Rojas, J. (1998). Following the direction of gaze and language development in 6-month-olds. *Infant Behavior and Development*, 21, 373-377.

Mundy, P. & Burnette, C. (2005). Joint attention and neurodevelopmental models of autism. In Fred R. Volkmar, Rhea Paul, Ami Klin, & Donald Cohen (Eds.), *Handbook of autism and pervasive developmental disorders: Vol. 1. Diagnosis, development, neurobiology, and behavior* (3rd ed., pp. 650-681). Hoboken: Wiley.

Mundy, P. & Hogan, A. (1996). *A preliminary manual for the abridged Early Social Communication Scales (ESCS).* Unpublished manual, University of Miami.

O'Connor, K., Hamm, J. P., Kirk, I. J. (2005). The neuropsychological correlates of face processing in adults and children with Asperger's syndrome. *Brain and Cognition, 59,* 82-95.

O'Connor, K., Hamm, J. P., & Kirk, I. J. (2007). Neurophysiological responses to face, facial regions and objects in adults with Asperger's syndrome: An ERP investigation. *International Journal of Psychophysiology, 63,* 283-293.

Passarotti, A., Paul, B., & Stiles, J. (2001). Development affects the ventral and dorsal processing streams differently: An fMRI study on face and location processing in children, teenagers, and adults. *NeuroImage, 13,* S345.

Pierce, K., Muller, R. A., Ambrose, J., Allen, G., & Courchesne, E. (2001). Face processing occurs outside the fusiform 'face area' in autism: Evidence from functional MRI. *Brain: A Journal of Neurology, 124,* 2059-2073.

Rebai, M., Poiroux, S., Bernard, C., & Lalonde, R. (2001). Event-related potentials for category-specific information during passive viewing of faces and objects. *International Journal of Neuroscience, 106,* 209-226.

Reynolds, C. R. & Kamphaus, R. W. (1998). *Behavior Assessment System for Children.* American Guidance Service.

Rogers, S. J., Hepburn, S. L., Stackhouse, T., & Wehner, E. (2003). Imitation performance in toddlers with autism and those with other developmental disabilities. *Journal of Child Psychology and Psychiatry, 44,* 763-781.

Rossion, B., Curran, T., & Gauthier, I. (2002). A defense of the subordinate-level expertise account for the N170 component. *Cognition, 85,* 189-196.

Rossion, B., Delvenne, J. F., Debatisse, D., Goffaux, V., Bruyer, R., Crommelinck, M., et al. (1999). Spatio-temporal localization of the face inversion effect: An event related potentials study. *Biological Psychology, 50,* 173-189.

Rossion, B., Gauthier, I., Tarr, M. J., Despland, P., Bruyer, R., Linotte, S., et al. (2000). The N170 occipito-temporal component is delayed and enhanced to inverted faces but not to inverted objects: An electrophysiological account of face-specific processes in the human brain. *NeuroReport, 11,* 69-74.

Stormark, K. M. & Braarud, H. C. (2004). Infants' sensitivity to social contingency: A "double video" study of face-to-face communication between 2- and 4-month-olds and their mothers. *Infant Behavior and Development, 27,* 195-203.

Tanaka, J. W. & Curran, T. (2001). A neural basis for expert object recognition. *Psychological Science, 12,* 43-47.

Taylor, M. J., Batty, M., & Itier, R. J. (2004). The faces of development: A review of early face processing over childhood. *Journal of Cognitive Neuroscience, 16,* 1426-1442.

Taylor, M. J., Edmonds, G. E., McCarthy, G., & Allison, T. (2001). Eyes first! Eye processing develops before face processing in children. *NeuroReport, 12,* 1671-1676.

Taylor, M. J., McCarthy, G., Saliba, E., & Degiovanni, E. (1999). ERP evidence of developmental changes in processing of faces. *Clinical Neurophysiology, 110,* 910-915.

Townsend, J., Westerfield, M., Leaver, E., Makeig, S., Jung, P., Pierce, K., et al. (2001). Event-related brain response abnormalities in autism: Evidence for impaired cerebello-frontal spatial attention networks. *Cognitive Brain Research, 11,* 127-145.

Turati, C. (2004). Why faces are not special to newborns: An alternative account of the face preference. *Current Directions in Psychological Science, 13,* 5-8.

Williams, P., Weiss, L., & Rolfhus, E. (2003a). *Wechsler Intelligence Scale for Children – IV, Technical Report 1, Theoretical Model & Test Blueprint.* San Antonio, TX: The Psychological Corporation.

Williams, P., Weiss, L., & Rolfhus, E. (2003b). *Wechsler Intelligence Scale for Children – IV, Technical Report 2, Psychometric Properties.* San Antonio, TX: The Psychological Corporation.

TABLES

Table 1

Participant characteristics

			Chronological Age (months)			Verbal IQ		
	N	M/F	Min	Max	*M* (*SD*)	Min	Max	*M* (*SD*)
Autism Group	27	23/4	113	209	159.56 (33.27)	77	130	97.93 (14.43)
Control Group	22	18/4	108	203	172.73 (24.55)	81	136	104.77 (12.76)

Table 2

N170 mean amplitude values in μV

	Autism		Typical Development	
	Younger	Older	Younger	Older
Left Hemisphere				
Upright Faces	3.33 (4.49)	0.54 (1.81)	3.64 (3.48)	2.33 (4.48)
Upright Vehicles	3.67 (4.95)	0.67 (2.62)	2.44 (5.48)	-0.22 (3.78)
Inverted Faces	4.78 (5.18)	1.67 (1.79)	6.20 (4.56)	2.64 (4.08)
Inverted Vehicles	5.66 (4.71)	1.46 (2.82)	6.75 (6.96)	2.42 (3.97)
Right Hemisphere				
Upright Faces	5.24 (5.98)	0.47 (2.70)	4.60 (4.38)	2.98 (4.64)
Upright Vehicles	4.63 (4.71)	0.50 (2.78)	3.54 (4.54)	-0.46 (4.42)
Inverted Faces	7.29 (6.32)	1.90 (2.55)	8.96 (4.74)	3.26 (4.72)
Inverted Vehicles	7.30 (4.26)	1.45 (2.84)	7.89 (6.11)	3.23 (4.42)

Note. Standard deviations are in brackets.

Table 3

N170 mean latency values in msec

	Autism		Typical Development	
	Younger	Older	Younger	Older
Left Hemisphere				
Upright Faces	180.71 (23.29)	173.52 (23.49)	177.78 (18.52)	161.69 (17.58)
Upright Vehicles	204.92 (15.74)	205.33 (17.66)	202.59 (18.30)	180.10 (20.43)
Inverted Faces	191.63 (21.54)	183.94 (26.35)	190.52 (20.86)	175.49 (28.01)
Inverted Vehicles	210.67 (16.70)	206.91 (22.93)	204.30 (21.21)	197.08 (25.28)
Right Hemisphere				
Upright Faces	172.29 (26.31)	180.00 (26.46)	168.81 (12.67)	158.77 (14.68)
Upright Vehicles	202.58 (17.94)	206.79 (22.48)	197.48 (17.32)	173.79 (17.17)
Inverted Faces	192.54 (23.79)	183.21 (28.29)	183.56 (13.93)	169.28 (21.70)
Inverted Vehicles	210.33 (21.52)	196.12 (29.12)	205.48 (12.93)	181.59 (19.90)

Note. Standard deviations are in brackets.

Table 4

P1 mean amplitude values in μV

	Autism		Typical Development	
	Younger	Older	Younger	Older
Left Hemisphere				
Upright Faces	19.46 (10.36)	10.50 (6.17)	16.76 (6.27)	12.11 (2.95)
Upright Vehicles	18.61 (8.65)	10.00 (6.83)	17.03 (7.68)	11.59 (3.16)
Inverted Faces	18.97 (9.47)	11.68 (6.11)	19.14 (6.55)	13.53 (3.25)
Inverted Vehicles	20.50 (9.22)	9.45 (6.10)	19.44 (8.82)	12.55 (3.29)
Right Hemisphere				
Upright Faces	19.42 (8.90)	10.88 (6.24)	19.10 (6.79)	14.42 (3.95)
Upright Vehicles	19.59 (7.79)	10.28 (6.25)	19.40 (7.48)	13.53 (4.43)
Inverted Faces	20.11 (8.26)	11.61 (6.42)	21.75 (6.75)	16.24 (4.29)
Inverted Vehicles	21.55 (7.72)	9.95 (6.44)	21.35 (7.63)	15.45 (4.44)

Note: Standard deviations are in brackets.

Table 5

Partial Correlations between P1 Amplitude Measures and Social Behavior and Autistic Symptomology Measures, Controlling for Age

	ADOS Social Interaction	ADOS Total	SCQ Social Interaction	Eyes Test
Autism				
Upright Faces	0.24	0.16	0.17	0.07
Inverted Faces	0.16	0.08	0.15	-0.01
Upright Vehicles	0.11	0.04	0.17	-0.04
Inverted Vehicles	0.08	0.02	0.26	-0.13
Typical Development				
Upright Faces	0.45*	0.45*	0.54*	-0.46*
Inverted Faces	0.55**	0.55*	0.53*	-0.45*
Upright Vehicles	0.54*	0.53*	0.48*	-0.47*
Inverted Vehicles	0.65**	0.61**	0.37	-0.38

Note: * $p < 0.05$, ** $p < 0.01$

FIGURES

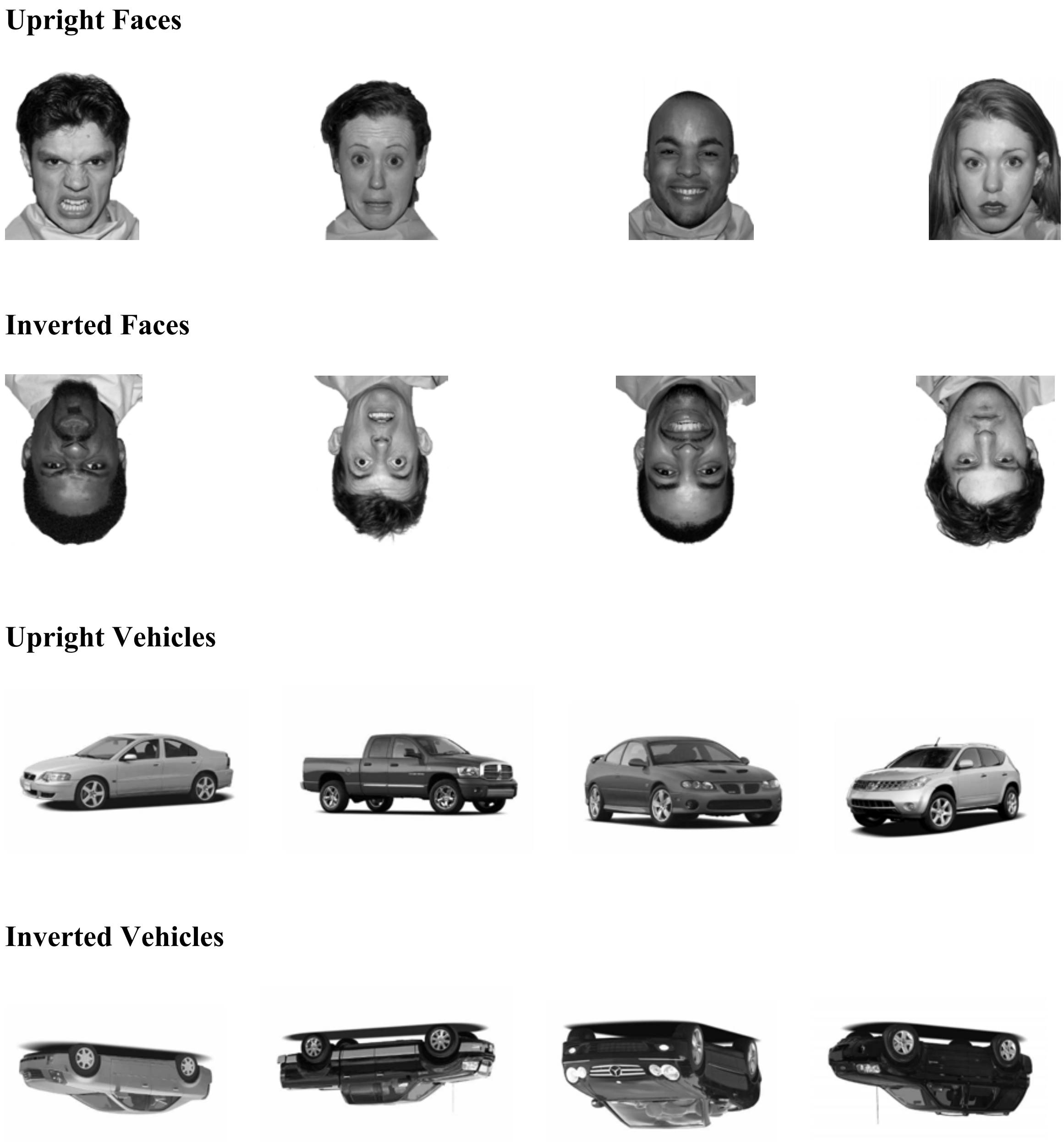

Figure 1. Example stimuli.

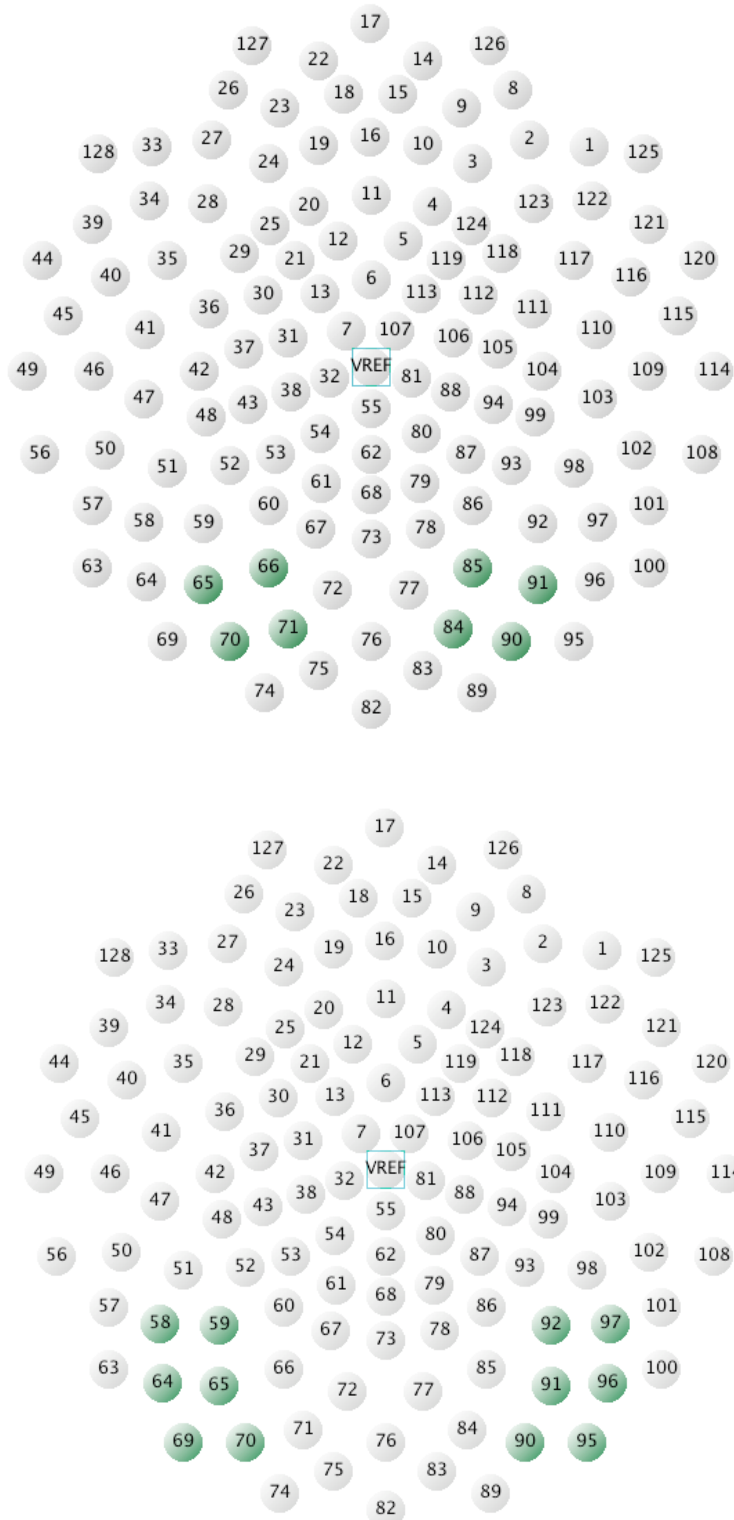

Figure 2. Electrode groups over which data were averaged for the P1 component (top) and the N170 component (bottom).

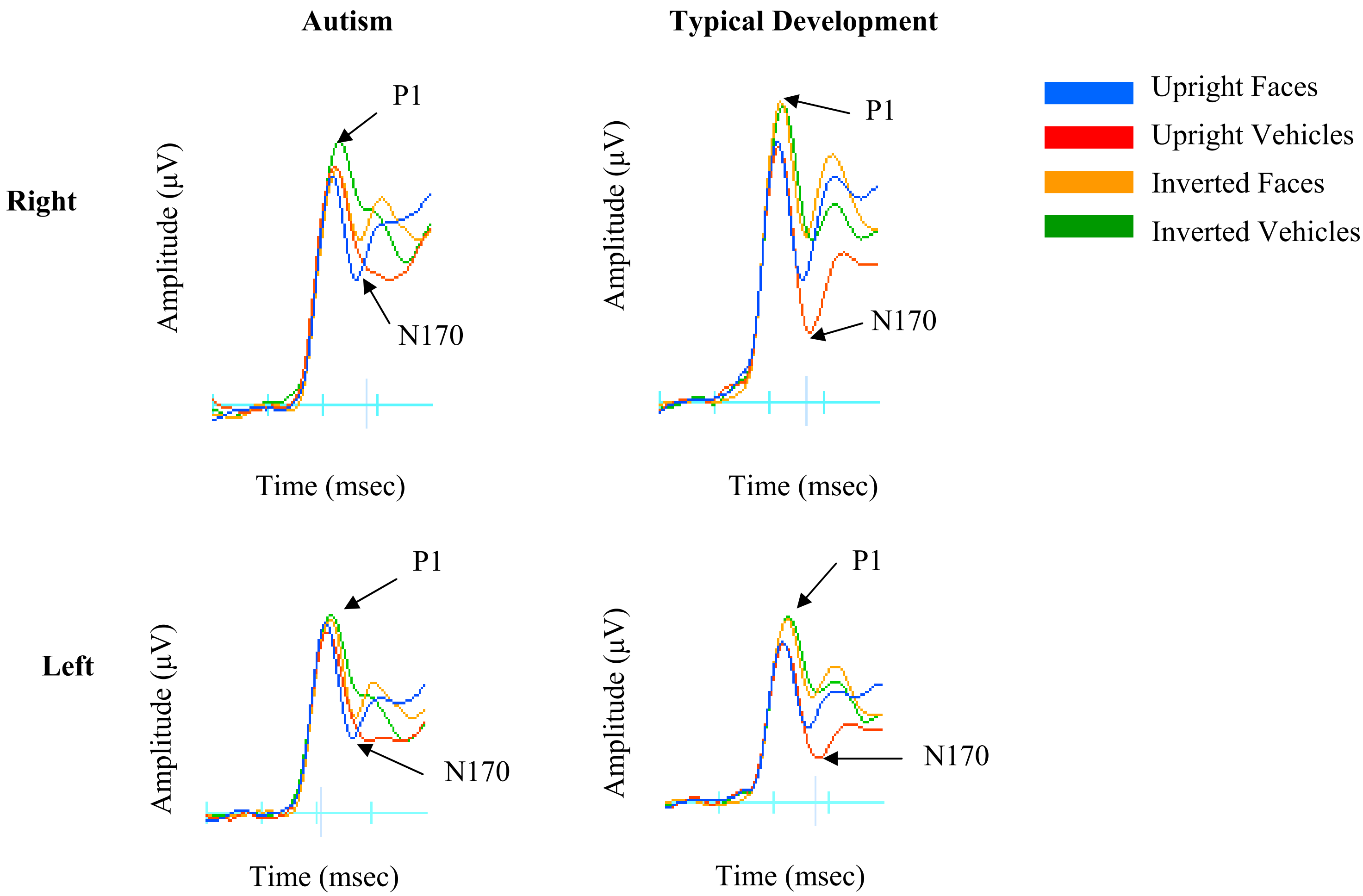

Figure 3. Grand averaged ERP waveforms to upright faces, upright vehicles, inverted faces, and inverted vehicles for children with autism and typical development. Electrode 91 is used as a prototypical electrode for the right hemisphere, and electrode 65 is used as a prototypical electrode for the left hemisphere. All waveforms have a 100 msec pre-stimulus and a 300 msec post-stimulus interval.

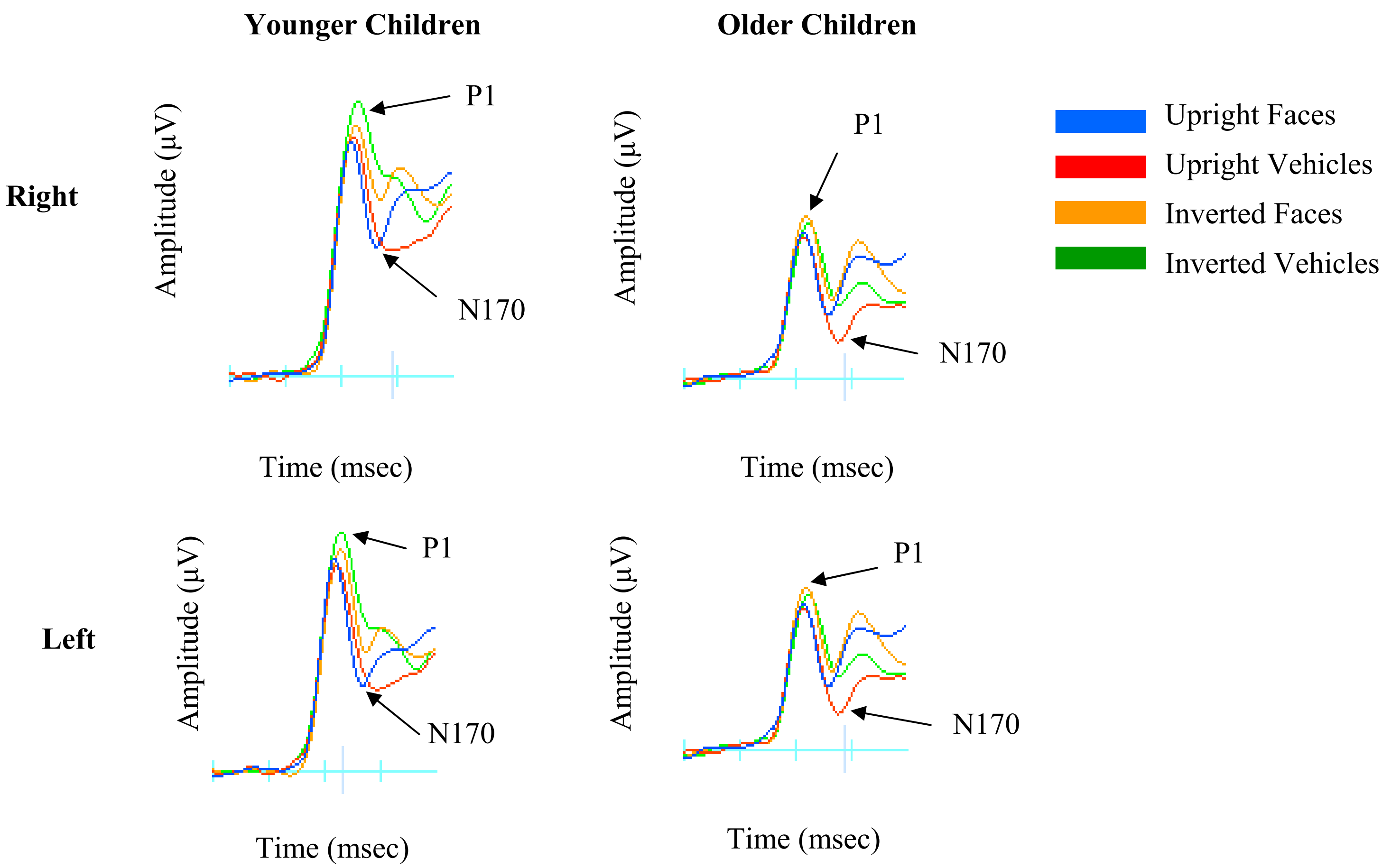

Figure 4. Grand averaged ERP waveforms waveforms to upright faces, upright vehicles, inverted faces, and inverted vehicles for younger children and older children. Electrode 91 is used as a prototypical electrode for the right hemisphere, and electrode 65 is used as a prototypical electrode for the left hemisphere. All waveforms have a 100 msec pre-stimulus and a 300 msec post-stimulus interval.

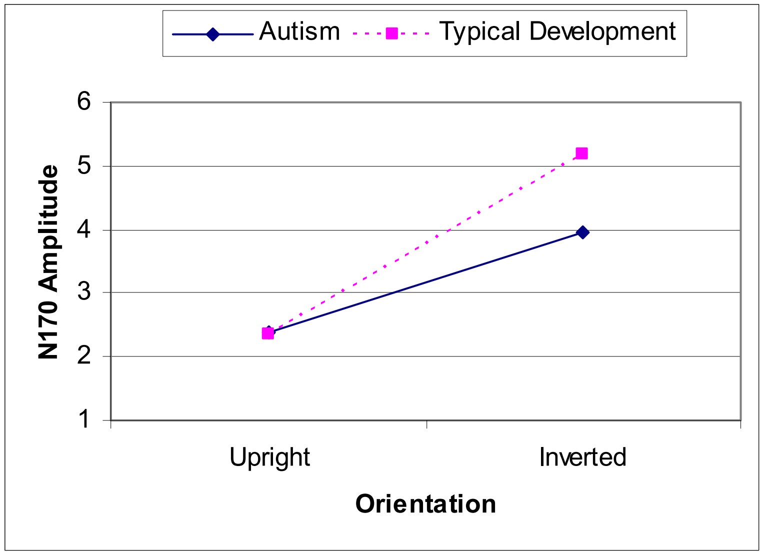

Figure 5. The interaction between group and orientation on N170 amplitude.

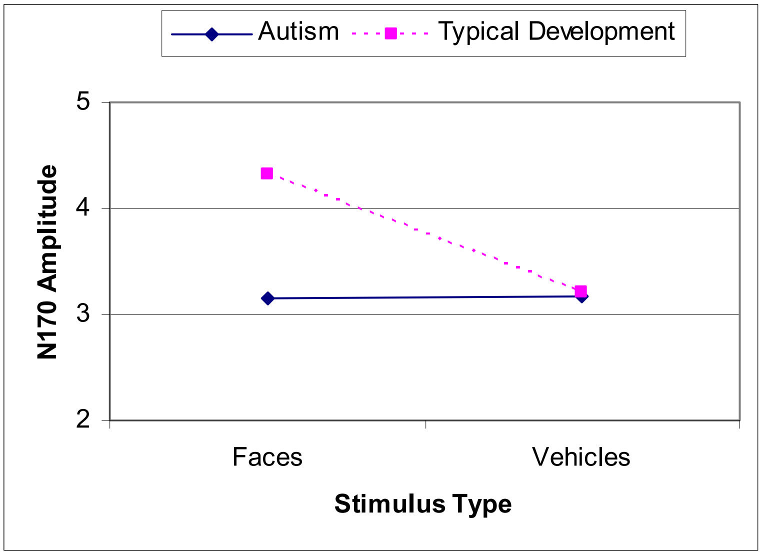

Figure 6. The marginal interaction between group and stimulus type on N170 amplitude.

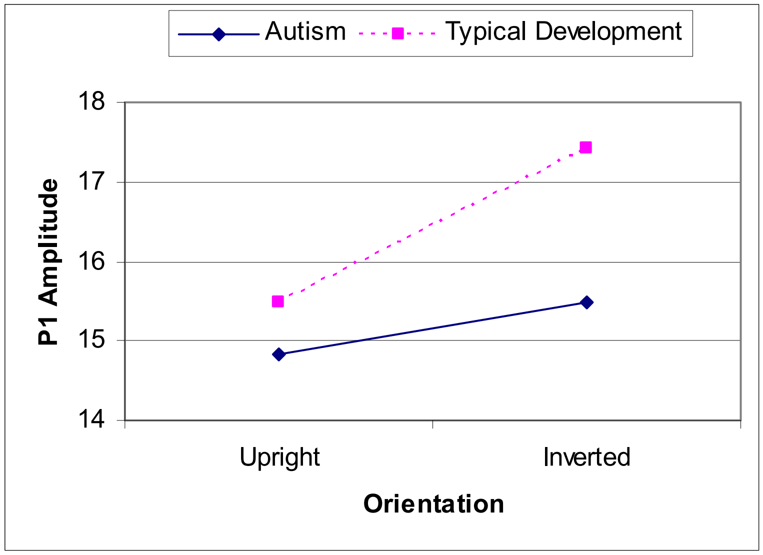

Figure 7. The interaction between group and orientation on P1 amplitude.

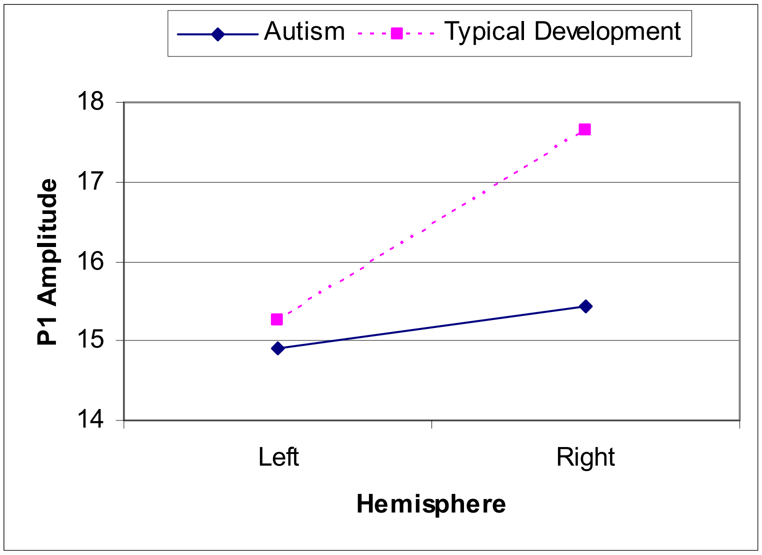

Figure 8. The marginal interaction between group and hemisphere on P1 amplitude.