

Understanding the Nature of Face Processing Impairment in Autism: Insights From Behavioral and Electrophysiological Studies

Geraldine Dawson, Sara Jane Webb, and James McPartland
University of Washington Autism Center
University of Washington

This article reviews behavioral and electrophysiological studies of face processing and discusses hypotheses for understanding the nature of face processing impairments in autism. Based on results of behavioral studies, this study demonstrates that individuals with autism have impaired face discrimination and recognition and use atypical strategies for processing faces characterized by reduced attention to the eyes and piecemeal rather than configural strategies. Based on results of electrophysiological studies, this article concludes that face processing impairments are present early in autism, by 3 years of age. Such studies have detected abnormalities in both early (N170 reflecting structural encoding) and late (NC reflecting recognition memory) stages of face processing. Event-related potential studies of young children and adults with autism have found slower speed of processing of faces, a failure to show the expected speed advantage of processing faces versus nonface stimuli, and atypical scalp topography suggesting abnormal cortical specialization for face processing. Other electrophysiological studies have suggested that autism is associated with early and late stage processing impairments of facial expressions of emotion (fear) and decreased perceptual binding as reflected in reduced gamma during face processing. This article describes two types of hypotheses—cognitive/perceptual and motivational/affective—that offer frameworks for understanding the nature of face processing impairments in autism. This article discusses implications for intervention.

Autism is a disorder characterized by impairments in social and communicative behavior and a restricted range of interests and behaviors. Although there is great variability in symptom severity and intellectual functioning among individ-

uals diagnosed with the disorder, all individuals with autism have difficulties in social interaction such as use of eye contact, engaging in reciprocal interactions, and responding to the emotional cues of others. Basic impairments, such as lack of attention to others and failure to orient to name, often appear within the 1st year of life (Werner, Dawson, Osterling, & Dinno, 2000). By age 2 to 3, impairments are evident in social orienting, eye contact, joint attention, imitation, responses to the emotional displays of others, and face recognition (Dawson, Carver, et al., 2002; Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998; Dawson, Toth, et al., 2004; Mundy, Sigman, Ungerer, & Sherman, 1986; Sigman, Kasari, Kwon, & Yirmiya, 1992).

Many of the early social impairments in autism, such as eye contact, joint attention, responses to emotional displays, and face recognition, involve the ability to attend to and process information from faces. As such, we believe that impairments in face processing may play a fundamental role in the dysfunction of the brain systems underlying the impairments in social cognition in autism. The neural systems that mediate face processing come on line very early in life. Thus, face processing impairments may be one of the earliest indicators of abnormal brain development in autism. For typically developing infants, faces have special significance and provide nonverbal information important for communication and survival (Darwin, 1872/1965). Face recognition ability is present during the first 6 months of life. A visual preference for faces (Goren, Sarty, & Wu, 1975) and the capacity for very rapid face recognition (Walton & Bower, 1993) are present at birth. By 4 months, infants recognize upright faces better than upside down faces (Fagan, 1972). By 6 months, infants show differential event-related brain potentials to familiar versus unfamiliar faces (de Haan & Nelson, 1997, 1999). Young infants are capable of abstracting direction of gaze, facial gestures, and expressions of emotion within the 1st year of life. These early developing abilities, particularly attention and response to gaze, are critical to successful joint attention and social orienting interactions. Face processing has also been posited as critical to the development of social relationships and theory of mind (e.g., Baron-Cohen, 1995; Perrett et al., 1990; Perrett, Hietanen, Oram, & Benson, 1992; Williams, Whiten, Suddendorf, & Perrett, 2001).

In this article, we have three goals: First, we review behavioral studies of face processing in individuals with autism. Second, we summarize findings from electrophysiological studies of face processing in autism. Third, we describe hypotheses regarding the nature of face processing impairments in autism, which integrate behavioral and electrophysiological findings.

BEHAVIORAL STUDIES OF FACE PROCESSING IN AUTISM

Autism typically is not diagnosed until about 3 years of age, although researchers are working to identify children by 15 to 18 months (Filipek et al., 1999). Because

there exist few observations of infants with autism, very little is known about the early development of face processing abilities of young children with autism. In a case study of Dawson, Osterling, Meltzoff, and Kuhl (2000), a young infant who was diagnosed with autism at 1 year of age and rediagnosed at 2 years of age was reported in early medical records as having “generally good eye contact, although at times he averted his eyes” and to have smiled responsively during the first 6 months. On four different evaluations from 9 to 13 months, eye contact was reported as a transfixed stare, poor, and within normal limits. The infant was reported to have reduced social responsiveness, and social interactions were described as aversive to the infant. In a retrospective study (Osterling & Dawson, 1994; also see Adrien et al., 1991) using videotapes of first birthday parties, the single best discriminator between infants who were later diagnosed with an autism spectrum disorder versus those with typical development was the failure to look at others. The toddler’s ability to use facial information such as gaze monitoring during joint attention is considered to be one of the critical discriminatory in early diagnosis of this disorder. Taken together, these findings suggest that failure to process faces in a normal manner might be one of the earliest measurable autism symptoms, emerging by 1 year of age and possibly earlier.

Studies of face memory in autism have shown that by middle childhood, children with autism perform worse than mental age and chronological age matched peers on a number of face processing tasks. This includes tests of face discrimination (Tantam, Monaghan, Nicholson, & Stirling, 1989) and face recognition (Boucher & Lewis, 1992; Boucher, Lewis, & Collis, 1998; Gepner, de Gelder, & de Schonen, 1996; Klin et al., 1999). In comparison to typically developing children who show better memory performance for faces than nonface visual stimuli, children with autism perform comparably on face and nonface tasks (Serra et al., 2003) or show better performance on nonface tasks (e.g., memory for buildings) than on face tasks (Boucher & Lewis, 1992). Similarly, adults with autism perform worse than chronological and verbal IQ matched control groups on tests of face memory but perform similarly to verbal IQ matched groups in terms of memory for cats, horses, and bicycles. Memory for buildings and leaves is unimpaired in comparison to the chronological age group (Blair, Frith, Smith, Abell, & Cipolotti, 2002).

Several studies have suggested that individuals with autism process faces using abnormal strategies. By middle childhood, typically developing children are better at recognizing parts of a face when the parts are presented in the context of a whole face, perform better when recognition involves the eyes versus the mouth (Joseph & Tanaka, 2003), show a greater decrement in memory for inverted versus upright faces as compared with nonface visual stimuli, and attend to upright faces for longer lengths of time than inverted faces (van der Geest, Kemner, Verbaten, & van Engeland, 2002). Children with autism, in contrast, are better at recognizing isolated facial features and partially obscured faces than typical children (Hobson, Ouston, & Lee, 1988; Tantam et al., 1989) and show better performance on mem-

ory for the lower half of the face than the upper half during childhood (Langdell, 1978). Studies (Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Pelphrey et al., 2002; Trepagnier, Sebrechts, & Peterson, 2002) of visual attention to faces have indicated that individuals with autism exhibit reduced attention to the core features of the face, such as the eyes and nose, relative to typical individuals. One study (van der Geest et al., 2002) indicated that when viewing emotionally expressive faces, children with autism may exhibit more typical patterns of visual attention, fixating more on the eyes and mouth than other parts of the face. Individuals with autism recognize inverted faces better than control participants (Hobson, Ouston, & Lee, 1988b; Langdell, 1978) and spend equal time looking at inverted faces compared with upright faces (van der Geest et al., 2002). It has been suggested that this pattern of deficits represents a failure to process faces configurally (Elgar & Campbell, 2001), with an emphasis on local detail rather than global patterns (Happe, 1999).

In summary, several studies have documented impairments in face discrimination and recognition in individuals with autism. Furthermore, there is evidence that individuals with autism use atypical strategies for processing faces characterized by reduced attention to the eyes and piecemeal rather than holistic strategies.

ELECTROPHYSIOLOGICAL STUDIES OF FACE PROCESSING IN AUTISM

Although the results mentioned previously provide insight regarding the abilities of individuals with autism, they often rely on tests requiring verbal ability and as such are mostly conducted with older, higher functioning children. Electrophysiological studies provide information about the neural basis of face processing impairments and autism and do not require a verbal response, making them appropriate for younger, lower functioning children. Specifically, electroencephalogram (EEG), which refers to the ongoing electrical oscillations of the brain, and event-related potentials (ERPs), which refer to the average electrical signal recorded in relation to a specific timed event, can be used to address fundamental questions about the neural systems involved in face processing in typical and atypical populations. The EEG/ERP signal originates from the postsynaptic (dendritic) potentials of a population of synchronously firing neurons. The activity recorded at the scalp reflects the summation of neurons that are oriented perpendicular to the surface of the scalp, aligned in such a way as to produce a dipole field (a field with positive and negative charges between which current flows; see Coles & Rugg, 1995). EEG/ERPs are exquisitely sensitive to real time neural processes, providing detailed temporal resolution on the scale of milliseconds as to changes in neural state. EEG/ERPs are noninvasive, requiring only that the participant tolerate a damp sensor net or an electrode hat for relatively short periods of time, and they do

not necessarily require the participant to follow explicit directions or produce motor or verbal responses. Thus, the methodology can be used across the life span and with participants who have limited cognitive or communicative abilities. Such requirements are important for understanding the early stages of brain development and function in young children with autism.

A number of variables can be abstracted from the raw EEG and the averaged ERP signal. From the underlying EEG, oscillation rate (measured in hertz or cycles per second) and power (contribution of each frequency to the signal) can be computed in regard to the activity over a time period (using a Fourier transformation) or in reference to a particular time point (using wavelet decomposition). For ERPs, most reports focus on the latency, amplitude, and scalp topography of the components. The timing of the peaks and troughs of the waveform is thought to reflect information-processing speed, whereas the amplitude (deviation from baseline activity) is thought to loosely reflect the amount of neural resources being devoted to that process. Differences in distribution are thought to reflect the location of the generator of the signal, albeit scalp potentials likely reflect the concurrent activity of a number of processes that summate on the scalp.

ERPs are particularly applicable to the study of face processing in autism because ERP studies of faces have been conducted with both infants and young children, a face-sensitive ERP component (N170) has been documented and studied across childhood, and an ERP signal can offer insight into stages of information processing. ERP studies have demonstrated that the ERP components reflect multiple stages of processing, including both early stage attentional and perceptual components and later slow wave components that are thought to reflect cognitive updating and memory. We begin by discussing the findings on the N170 component.

ERP Component, N170, Is Sensitive to Early Stage Face Processing

Faces evoke a distinct pattern of electrical brain activity. A number of researchers (Bentin, Allison, Puce, Perez, & McCarthy, 1996; Eimer, 1998, 2000a, 2000b, 2000c; George, Evans, Fiori, Davidoff, & Renault, 1996) have documented an ERP component that preferentially activates to faces, which is recorded over the posterior temporal lobe and is greater in the right than the left hemisphere. This component slopes negatively and, in adults, peaks at approximately 170 msec poststimulus onset. N170 is faster to faces and eyes alone than to inverted faces and nonface stimuli. The latency is sensitive to disruptions in early stage processing of faces but is not altered by facial familiarity or recognition processes (e.g., Bentin & Deouell, 2000; Eimer, 2000a). Facial inversion and partial decomposition of faces alters both the latency and amplitude of the component; these effects of inversion are specific to faces (Eimer, 2000b; Rebai,

Poiroux, Bernard, & Lalonde, 2001; Rossion et al., 2000). Facial movements, such as the eyes looking away and the mouth opening, also influence the N170 amplitude (Puce, Smith, & Allison, 2000).

Although functional magnetic resonance imaging studies have been useful in demonstrating abnormal regional brain activity during face processing in individuals with autism (R. Schultz et al., 2000), little is known about the temporal characteristics of neural processing of faces in autism. In the first published report of N170 in adolescents and adults with autism, McPartland, Dawson, Webb, Panagiotides, and Carver (2004) found an altered N170 pattern. Compared to cognitively matched typical individuals, high-functioning individuals with autism exhibited slower N170 latencies to faces than furniture and failed to show a face inversion effect. In addition, speed of face processing, as reflected in N170 latency, was found to be correlated with performance on a test of face recognition. The McPartland et al. ERP study provides evidence that early structural encoding of faces is disrupted in autism and is characterized by slower speed of information processing. Furthermore, based on the N170 scalp topography, there was evidence that a failure of normal right hemisphere specialization for faces exists in at least some individuals with autism. The presence of atypical cortical representation of face processing suggests that slower processing of faces in autism does not simply reflect a quantitative difference in timing of an otherwise normal neural circuit but rather an aberrant neural circuitry or missing neural processing modules resulting in less efficient processing strategies.

In typically developing individuals, the N170 undergoes a prolonged period of development. Taylor, Edmonds, McCarthy, and Allison (2001) and Taylor, McCarthy, Saliba, and Degiovanni (1999) have identified a precursor to the adult N170 in children between 4 and 15 years of age. Similar to the adult N170, this component is of greatest amplitude to eyes and upright faces and is sensitive to inversion. It is significantly slower than the adult N170, peaking at approximately 270 msec in 4- to 5-year-olds, and does not reach adult values (in terms of amplitude and latency) until late adolescence. Morphologically, the component is also more positive than the adult N170; it is a negative-going deflection recorded from the same electrode configuration, but it does not extend below baseline and thus may not have negative amplitude values. Webb, Bernier, Panagiotides, Paul and Dawson (2003) and Webb, Dawson, Bernier, and Panagiotides (in press) labeled this component the precursor N170 (prN170) and have studied it in 3- to 6-year-old children with autism and children with typical development. Unlike adults, 3- to 4-year-old children with typical development showed no amplitude difference between faces and objects. Typically developing children were found, however, to have faster responses (shorter prN170 latency) to faces than objects. In contrast, children with autism showed a larger prN170 to objects compared to typically developing children and showed shorter prN170 latency to objects than to faces (Webb, Dawson, et al., in press). In a study, Webb, Bernier, et al. (2003) conducted

with children who were 6 years of age, both typically developing children and children with autism showed the adult N170 pattern of greater negativity to faces than objects. Typical children continued to show faster responses to faces than objects, but children with autism showed no differences in their ERP latencies to faces versus objects (Webb, Bernier, et al., 2003). Thus, it appears that early on, autism is associated with a failure to show the normal speed advantage for processing faces.

There is still controversy concerning the neural substrates of the N170 component. One hypothesis suggests that it reflects eye detection because the N170 shows the greatest amplitude and shortest latency in response to eyes (Bentin et al., 1996; Taylor et al., 1999, 2001). A second hypothesis is that it reflects early stage processing of whole faces similar to the structurally encoding stage in Bruce and Young's (1986) model of face processing. Because the N170 is sensitive (slowed) when the face is inverted (e.g., Bentin et al., 1996; Eimer, 1998) and shows increased activation (amplitude) in response to expertise categories (Goffaux, Gauthier, & Rossion, 2003; Tanaka & Curran, 2001; Tanaka, Luu, Weisbrod, & Kiefer, 1999), it might reflect early stage configural processing. Individuals with autism show reduced attention to eyes within the face and also fail to use configural strategies for face processing. In the next section, we discuss an EEG index that is sensitive to configural or holistic processing of visual stimuli.

EEG Gamma Activity Provides Another Neural Index of Early Stage Face Processing

EEG activation in the gamma range (30 Hz to 80 Hz) has been proposed to reflect neural processes involved in holistic processing or the perception of a whole or gestalt. Specifically, gamma activation has been proposed as the key mechanism by which the brain links together stimulus properties (e.g., Eckhorn, Reitboeck, Arndt, & Dicke, 1990; Gray, Engel, König, & Singer, 1990; Gray, König, Engel, & Singer, 1989; Milner, 1974). For example, gamma activity increases in the visual cortex during the perception of coherent objects and coherent motion (e.g., Müller et al., 1996; Müller, Elbert, & Rockstroh, 1997; Tallon, Bertrand, Bouchet, & Pernier, 1995; Tallon-Baudry, Bertrand, Delpuech, & Pernier, 1996). Increased gamma activity has also been found over frontal regions during tasks that require top-down feature selection processes (Hermann & Mecklinger, 2000), increased attentiveness (e.g., Müller, Gruber, & Keil, 2000), processing of upright faces (Keil, Müller, Ray, Gruber, & Elbert, 1999; Rodriguez et al., 1999), and emotional scenes (Müller, Keil, Gruber, & Elbert, 1999).

Individuals with autism have been found to exhibit abnormal gamma band activity during processing of upright and inverted faces (Grice et al., 2001). For typical individuals, upright faces evoked a larger increase in gamma activation than inverted faces. In contrast, individuals with autism showed equivalent gamma activation to upright and inverted faces. The decreased gamma activation to up-

right faces in the autism group as compared to the typical group suggests decreased perceptual binding and is consistent with an impairment in holistic/configural processing of faces in autism. Webb is currently replicating and extending Grice et al.'s study of gamma in children with autism.

ERPs Provide Clues Regarding Early Perception of Facial Expressions of Emotion

ERP studies are also informative in studying early perception of facial expression. Differential ERPs to distinct facial expressions of emotion have been shown in infants as young as 7 months of age (Nelson & de Haan, 1996), in children (de Haan, Nelson, Gunnar, & Tout, 1998), and in adults (Eimer & Holmes, 2002). To assess emotion perception in young children with autism, Dawson, Webb, Carver, Panagiotides, and McPartland (2004) showed 3- to 4-year-old children pictures of two faces: one depicting a neutral expression and the same face depicting a prototypic expression of fear. Children with autism exhibited significantly slower early (N300) brain responses to fear as compared to typically developing children. Children with autism also failed to show larger amplitude, negative slow wave responses to the fear face as was found in the typical children regardless of mental age. Dawson, Webb, et al. (2004) also found that the children with autism displayed aberrant scalp topography of their ERP responses to fearful faces. The delay in response to fear faces suggests that information processing speed is compromised, and the abnormal topography suggests failure of cortical specialization or atypical recruitment of cortical areas.

Dawson, Webb, et al. (2004) were interested in whether speed of early stage processing of emotion is related to degree of social impairment. Thus, they examined correlations among individual differences in N300 latency to the fear faces and performance on behavioral tasks requiring social attention, which were administered on a different day from EEG testing. They also compared these correlations to those with control tasks involving attention to nonsocial stimuli. Of the measures of social attention, three measures were correlated with the speed of early ERP responses to a fear expression. Specifically, children who displayed a faster N300 latency to the fear face exhibited better joint attention, fewer social orienting errors, and more time spent looking at an experimenter expressing distress. In contrast, there was no association between N300 latency and performance on the nonsocial tasks. These findings suggest that slower information processing speed for emotional stimuli is associated with more severe social attention impairments in children with autism. If a child with autism fails to activate or is slowed in activating neural structures that are normally specialized for face processing, the stimulus information might not be correctly bound to information about the context of the facial display or other sensory input that accompanies it such as auditory or tactile information. Temporal asynchrony in

perceptual processing has consequences both for formation of neural circuits as well as the behaviors that result from them (Brock, Brown, Boucher, & Rippon, 2002; Dawson et al., 2003). This could result in the facial display being stored independently from the other events that accompany it and make it less accessible to other neural systems.

Electrophysiological Indexes of Later Stage Face Processing Provide Insight Into Impairments in Facial Recognition

Bruce and Young (1986) suggested that face recognition involves matching the structural encoding of the currently viewed face with previously stored structural codes (face recognition units), which in turn link to identity specific semantic representations. In this model, recognition is a process that occurs after the face has been encoded and processed as a face but is independent of the analyses of facial expression and speech. To some extent, ERP findings have supported this model. Although there is some suggestion that the N170 is modulated by top-down activations of face representations (Jemel, Pisani, Calabria, Crommelinck, & Bruyer, 2003), priming (Campanella et al., 2000), and category familiarity (Tanaka & Curran, 2001), face recognition or familiarity has not been shown to effect latency or amplitude (e.g., Bentin & Deouell, 2000; Eimer, 2000b; Henson et al., 2003; Jemel, Calabria, Delvenne, Crommelinck, & Bruyer, 2003; Mnatsakanian & Tarkka, 2003). Repetition of faces, such as in a priming experiment, does not alter the N170 but affects subsequent components such as the N250 recorded at inferior temporal lobe electrodes (repetition of famous faces; Schweinberger, Pickering, Jentsch, Burton, & Kaufmann, 2002) and late components peaking after 400 msec (Eimer, 2000b; Henson et al., 2003; Mnatsakanian & Tarkka, 2003).

It has been shown that typically developing infants and young children exhibit ERP differences when processing familiar (parents or classmates) versus unfamiliar faces and objects (familiar or favorite toys; e.g., Carver et al., 2003; de Haan & Nelson, 1999). In a study with 3- to 4-year-old children, Dawson, Carver, et al. (2002) found that typically developing children and mental age-matched children with idiopathic developmental delay showed differential ERPs to the unfamiliar face as compared to their mother's face and to a favorite toy as compared to an unfamiliar object at the posterior P400 component and the frontal Nc (typical children) or the Slow Wave (developmentally delayed children). Children with autism failed to show differential ERPs to their mother's face versus an unfamiliar face at either component but did show differential ERPs to a favorite versus an unfamiliar toy at the P400 and Nc (see Figure 1). These results suggest a specific impairment in representation of faces but not objects in autism.

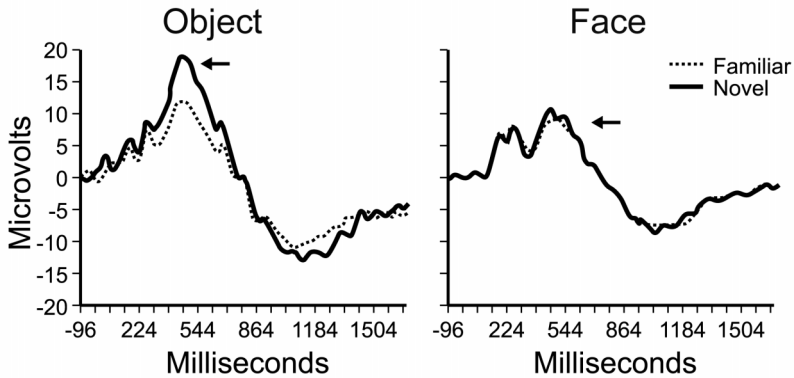


FIGURE 1 Event-related brain potentials to familiar faces and objects by 3- to 4-year-old children with autistic spectrum disorder. Based on data from Dawson, Carver, et al. (2002); reprinted with permission.

In summary, results of electrophysiological studies suggest that face processing impairments in autism are present early in life, at least by age 3 years. Such studies have detected impairments in both early (N170 reflecting structural encoding) and late (NC reflected recognition memory) stages of face processing. Furthermore, studies of young children, adolescents, and adults with autism consistently find slower speed of processing of faces and a failure to show the expected speed advantage of processing faces versus nonface stimuli. Such studies also have revealed atypical scalp topography suggesting abnormal cortical specialization for faces in autism. One study (Dawson, Webb, et al., 2004) conducted with young children with autism suggests both early and late stage processing impairments of facial expressions of emotion (fear). Finally, one study (Grice et al., 2001) of gamma activity suggests that face processing in autism is associated with decreased perceptual binding, which is consistent with behavioral findings of reduced holistic processing of faces. We next describe two types of hypotheses that offer frameworks for understanding the behavioral and electrophysiological findings on face processing in autism.

HYPOTHESES ACCOUNTING FOR FACE PROCESSING IMPAIRMENTS IN AUTISM

The development of brain circuits serving visual perception depends on a complex interaction between brain and experience. It was demonstrated (Katz & Shatz, 1996), for example, that the development of cortical–subcortical connections in the visual system relies on experience during both the prenatal period and postnatal periods. In contrast, the development of binocular vision depends primarily on postnatal

visual input (Hubel & Weisel, 1977). The development of general face perception abilities also occurs rapidly within the first months of life, with fine tuning likely taking place late through adolescence. In typical individuals, this is reflected in increased cortical specialization (i.e., right temporal lobe specialization for faces, e.g., de Schonen & Mathivet, 1989), increased neural speed (i.e., faster N170 with development; Taylor et al., 1999), and more efficient information-processing strategies (i.e., configural as opposed to featural processing; Diamond & Carey, 1986). Regardless of the “starter set,” visual experience is necessary for some aspects of face processing (de Schonen & Mathivet, 1989; Geldart, Mondloch, Mauer, de Schonen, & Brent, 2002; Grelotti, Gauthier, & Schultz, 2002). For example, infants with bilateral congenital cataracts who received limited visual experience for the first weeks of life have impaired recognition of facial identity even after years of corrected visual experience with faces (Geldart et al., 2002).

Several different models have been posited to explain the normal development of face processing and to account for its neurological underpinnings. A common assertion among many of these models is that face processing abilities are subserved by innate neural substrates that are (a) specialized for processing faces (e.g., a face processor in the fusiform gyrus) or (b) specialized for processing strategies that effectively apply to faces (e.g., configural processor; Kanwisher, 2000). The model put forward by Morton and Johnson (1991) posits that early face processing abilities are served by an innate subcortical neural system, which is replaced by a cortical system emerging by 6 months of age. The second system benefits from experience and accounts for the rapid changes in face processing abilities observed after 2 to 3 months of age.

In a contrasting model, Nelson (2001) suggested that rather than innate processing mechanisms specific to faces or processing strategy, there is the innate potential for cortical specialization for faces. In this model, domain general mechanisms, such as visual processing in the inferior temporal cortex, become domain specific with exposure (see Karmiloff-Smith, 1998); the face processing system may be an experience-expectant developmental system (Nelson, 2001). *Experience expectant* refers to a preparedness of the brain to receive special types of environmental stimulation during sensitive periods in which that type of stimulation is typically present. According to this theory, ample exposure to faces, a very reliable experience for most infants, drives specialization of a brain system that is likely necessary for normal development of face processing.

In the case of autism, the neural dysfunction resulting in face processing difficulties may occur at several levels, and several explanations can be posited. We discuss two types of explanations here:¹ (a) the fundamental impairment is percep-

¹Another type of explanation we do not discuss in this article is the hypothesis that autism involves a primary impairment in attention (Allen & Courchesne, 2001; Belmonte & Yurgelun-Todd, 2003; Burak, 1994). A combination of two dysfunctional systems (e.g., motivation and attention) is also a reasonable possibility.

tual/cognitive or (b) it is motivational/affective in nature. We discuss each of these hypotheses separately, although it should be noted that these hypotheses are not necessary mutually exclusive.

Perceptual/Cognitive Explanation of Face Processing Impairments

The perceptual/cognitive explanation of face processing impairments in autism includes hypotheses that autism involves (a) a fundamental general problem in perceptual binding as described previously; (b) an early onset, general, higher order perceptual/cognitive deficit that prevents the infant with autism from extracting perceptually relevant information from faces (e.g., prototype formation; Klinger and Dawson, 2001; Strauss, 2004); and/or (c) dysfunction of the specific neural mechanism that supports face processing, namely, the fusiform gyrus. We expand on the last one. In the fusiform hypothesis, face processing impairment would represent a primary deficit in autism. The dysfunction of the fusiform might be a neural deficit that was present at birth and thus could be screened for during early infancy. This deficit, early on, would be specific to faces, and we would expect that the typical pathway for processing faces would be inherently deficient. Remedial efforts might focus on compensatory strategies or require intensive stimulation of this circuit. The focus of the intervention would likely be to facilitate specific information-processing strategies such as teaching the child to attend to all features of the face and notice their configuration. Because the fusiform is part of a broader social brain circuit, a primary fusiform deficit would be expected to disrupt the general social brain circuitry and result in secondary deficits in abilities that rely on face perception such as joint attention, interpretation of emotional expression, and even speech perception.

Social Motivational/Affective Explanation of Face Processing Impairments

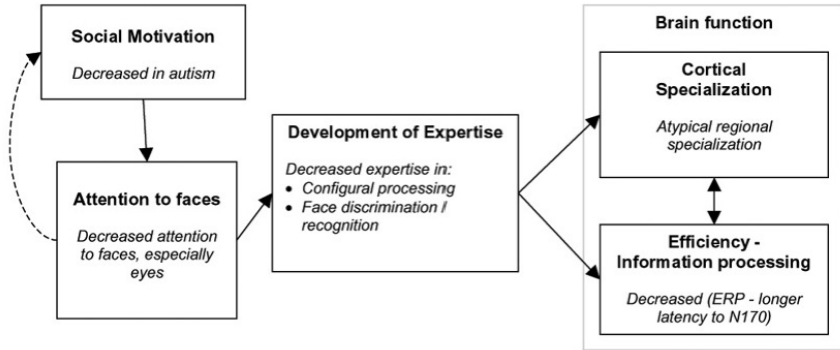
Alternatively, according to a social motivation hypothesis, the behavioral and electrophysiological indexes of face processing deficits described previously are secondary to a primary deficit in social motivation (Dawson, Carver, et al., 2002; Grelotti et al., 2002). The notion that individuals with autism lack social motivation is based partly on clinical observation; *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994) criteria for autism include “a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people” (p. 70) and “lack of social or emotional reciprocity” (p. 70). Observational studies have shown that young children with autism are less likely to smile when looking at their mothers (Dawson, Hill, Galpert, Spencer,

& Watson, 1990) and less likely to use positive affect during joint attention episodes (Kasari, Sigman, Mundy, & Yirmiya, 1990).

According to this hypothesis, an impairment in social motivation results in reduced attention to faces as well as to all other social stimuli such as the human voice, hand gestures, and so on. Dawson, Carver, et al. (2002) hypothesized that social motivational impairments in autism are related to a difficulty in forming representations of the reward value of social stimuli. We speculate that this difficulty might stem from abnormalities in either (a) the reward system per se (W. Schultz, 1998) or (b) neural systems that might be important for the perception of social reward such as ability to form representations of others as “like me,” that is, similar to self in some way (Decety, Chaminade, Grezes, & Meltzoff, 2002; Decety & Sommerville, 2003; Meltzoff & Brooks, 2001; Williams et al., 2001). With regard to the first option, one of the primary neural systems involved in processing reward information appears to be the dopamine system (W. Schultz, 1998). Research has demonstrated that dopaminergic projections to the striatum and frontal cortex, particularly the orbitofrontal cortex, play an important role in mediating the effects of reward on approach behavior. Representation of reward value in the orbitofrontal cortex appears to also depend on input from basolateral amygdala (Schoenbaum, Setlow, Saddoris, & Gallagher, 2003). Recent studies have shown that the dopamine reward system is also activated in response to social rewards including eye contact (Kampe, Frith, Dolan, & Frith, 2001). Dawson, Munson, et al. (2002) demonstrated that the severity of joint attention impairments in young children with autism is highly correlated with performance on neurocognitive tasks that tap the medial temporal lobe-orbitofrontal circuit (e.g., object discrimination reversal).

Representations regarding the anticipated reward value of a stimulus begin to motivate and direct attention by the second half of the 1st year of life. Reduced attention to faces and other stimuli, such as voices, would deprive the social brain of needed input for normal development and specialization. This lack of attention to social stimuli would result in a failure to become an expert processor. This is consistent with Grelotti et al.’s (2002) hypothesis that face processing is simply one example of expert processing as well as Nelson’s (2001) theory that experience drives cortical specialization for faces. This would further result in a failure of specialization of regions that typically mediate face processing and would be reflected in decreased cortical specialization and abnormal brain circuitry for face processing resulting in slower information-processing speed. The social motivation hypothesis as it pertains to the development of neural circuitry underlying face processing is depicted in Figure 2. Although the figure is depicted as being specific to face processing circuitry, we propose that it also applies to other social stimuli such as the perception of human speech.

In considering the impact of early experience on the development of face processing in autism, we are not suggesting that a simple lack of exposure to face is the critical problem. Infants with autism are likely to be exposed to faces espe-



Impairments in autism noted in italics

FIGURE 2 Social motivation hypothesis: effects of an impairment in social motivation on development of neural systems underlying face processing in autism. ERP = event-related potential.

cially during early months when infants are typically held and fed by their parents during face-to-face interactions. However, if the infant with autism does not find others inherently interesting or rewarding, then the infant might not be actively attending to the face and voice or perceiving the face within a larger social/affective context. In fact, recent evidence (Kuhl, Tsao, & Lui, 2003) suggests that simple exposure to stimuli does not necessarily facilitate the development of brain circuitry specialized for such stimuli. Evidence from studies of early infant speech perception suggests that perceiving speech within the context of a social interaction is important. For example, it has been well established that early in life, infants are capable of discerning differences among the phonetic units of all languages including native- and foreign-language sounds. Between 6 and 12 months of age, the ability to discriminate foreign language phonetic units sharply declines as the brain becomes proficient at perceiving speech (Kuhl et al., 1997). Kuhl, Tsao, and Liu (2003) investigated whether it was possible to reverse this decline in foreign language phonetic perception by exposing American infants to native Mandarin Chinese speakers. Kuhl et al. (2003) found that reversal in the decline of foreign language phonetic perception was achievable but only if exposure to speech occurred in the context of interpersonal interaction. Simple exposure via audiotapes did not affect the development of speech perception for nonnative language. In the case of autism, one can speculate that without an affectively rich social interaction, children with autism might not be actively attending to the facial and speech stimulation that is being provided to them, and therefore, such stimulation might not facilitate the normal development of the brain circuitry for faces and speech. In fact, in a sample of 3-

to 4-year-old children with autism, Kuhl, Coffey-Corina, Padden, and Dawson (2005) found that listening preferences in children with autism differed dramatically from those of typically developing and developmentally delayed children. Children with autism preferred listening to mechanical-sounding auditory signals (signals acoustically matched to speech referred to as “sine-wave analog”) rather than speech (motherese). The preference for the mechanical-sounding auditory signal was associated with lower language ability and abnormal ERPs to speech sounds. Children with autism who preferred motherese showed mismatch negativity (MMN) waveforms that were similar to those of typically developing children, with larger negative response to the deviant stimuli, indicating the brain’s detection of a change in the speech stimulus. Children with autism who preferred the mechanical-sounding auditory signal showed no differences between the MMN waveforms in response to two different syllables.

Implications for intervention. If the social motivation hypothesis is correct, it should also be possible to alter children’s attention to and experience with faces and speech through early intervention aimed to making social interaction more rewarding and meaningful to the child and then to examine the impact of the intervention on the development of face processing and brain responses to faces via ERP (Dawson & Zanolli, 2003). Increase use of eye contact, affective exchanges, and joint attention, which results from intervention, may improve the facial processing system. Indeed, interventions based on applied behavior analysis are designed the reward value of social stimuli through conditioned reinforcement. For example, the therapist’s face (a previously neutral stimulus) is deliberately paired with a reinforcer (usually access to food or toy). Via classical conditioning, the face then acquires reinforcer value. Thus, early intervention might facilitate the development of the face processing system in two ways: (a) by helping the child engage in meaningful social interactions that might lead to active attention to faces and (b) by altering the child’s motivational preferences for faces such that engaging in face-to-face interaction becomes more rewarding and frequent (Dawson & Zanolli, 2003).

The timing of intervention might also have important consequences on the plasticity of the face processing system. Alterations in social motivation and active attention to faces might result in a differential pattern of performance in children versus adults. For example, adults with autism might benefit from being trained to attend to faces and being taught explicit strategies for deriving information from the face. Although these interventions might result in better behavioral performance, it is unclear that they would result in alterations in patterns of neural activation. In other words, interventions might bolster compensatory processes but fail to “kick start” typical processing mechanisms. Conversely, given the plasticity in the developing brain, young children who receive early intervention might exhibit both improved behavioral performance (e.g., increases in eye

contact, joint attention, and face recognition) as well as normalized brain functioning (e.g., normal neural speed as reflected in face-related N170 and normal cortical specialization as reflected N170 scalp topography during face processing). Dawson and Webb are currently examining this possibility in an early intervention project with young children with autism.

CONCLUSION

In conclusion, electrophysiological studies of face processing in autism have provided important clues regarding the nature and development of social cognition impairments in this disorder. Electrophysical measures of brain activity are ideally suited for studying very young children. EEG/ERP studies have shown that the neural systems that mediate face processing are disrupted by age 3 in autism and persist into adulthood, that both early (encoding) and later (representation) stages of face processing are affected, that such impairments also are apparent in perception of facial expressions of emotion, and that slower speed of processing of faces is associated with more severe social behavioral impairments. Such studies have also suggested that abnormalities exist in the cortical specialization of such processes. We described two types of hypotheses, perceptual/cognitive and motivational/affective, that attempt to account for the abnormalities in face processing in autism. We discussed the implications of the social motivation hypothesis for early intervention in autism. Studies that examine the impact of early intervention on face processing in autism would not only have important implications for designing improved interventions but would also shed light on the nature of neural abnormalities in autism.

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