Autism Spectrum Disorders Expert Column

Face Processing in Individuals With Autism

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Introduction

Autism is a life-long neurodevelopmental disorder characterized by impairments in social and communicative abilities and a restricted range of interests and behaviors. Individuals with autism have profound difficulties with social interactions, which are manifested by impediments in the use of eye contact and problems with reciprocal social interactions and responses to emotional cues. Retrospective studies of home videotapes of first birthday parties of infants who were later diagnosed with autism document an early failure to actively attend to other people's faces and speech.[1,2] By age 2-3 years, children with autism exhibit a range of social dysfunction, including impairments in joint attention, imitation, and emotional responses.[3-7]

Impairments in face processing are well documented in autism. Such impairments are part of a more general dysfunction in complex social brain circuitry in persons with autism.[8] Because neural systems underlying face processing are present early in life, impairments in face processing likely represent a dysfunction of these very early brain systems. Typical neonates exhibit a visual preference for faces[9] and rapid face recognition.[10] At the age of 6 months, typically developing infants demonstrate specific brain responses, as measured by event-related potentials (ERPs), to familiar vs unfamiliar faces[11,12] and to fearful vs neutral facial expressions. These early face-processing skills are important for learning to interpret emotional expression and share attention with others.[13-16].

The Neural Basis of Face Processing

Evidence for face-processing impairments in autism comes from both behavioral and neuroimaging studies. In behavioral studies, elementary school-age children with autism have been found to perform worse than their mental age- and chronological age-matched peers on tests of face discrimination,[17] face recognition,[18-22] and emotion perception and recognition.[23]

In typically developing individuals, positron emission tomography and functional magnetic resonance imaging studies reveal that the right fusiform gyrus of the occipitotemporal cortex is more activated during perception of faces than various non-face stimuli, inverted faces, or scrambled faces.[24-33] The superior temporal sulcus is involved in processing face movements (eg, the eyes and mouth), and the amygdala is recruited when the face is familiar or expressing emotional content.[34] However, imaging studies indicate that the brains of individuals with autism fail to recruit brain regions specialized for faces;. for example, when a person with autism scans unfamiliar faces, the fusiform face area fails to activate, but rather, areas involved in processing of objects activate or idiosyncratic patterns of activation are evident.[35,36]

Faces themselves also elicit a characteristic pattern of electrical brain activity. In typical individuals from age 4 years to adult, an ERP component is seen that preferentially activates to faces.[37] This component, the "N170," is recorded over the posterior temporal lobe and is greater in the right than the left hemisphere.[38-43] The N170 is faster in response to faces and eyes alone than to inverted faces and non-face stimuli. Its latency is sensitive to disruptions in early-stage processing of faces but is not altered by familiarity of the face.[40,44] Facial movements, when the eyes look away or the mouth opens, for example, also influence the N170 amplitude.[45]

In a series of studies, Dawson and colleagues[4,5,46,47] found that children and adults with autism exhibit atypical ERPs to faces. They found that autistic children as young as 3 years of age exhibit atypical ERPs to faces and facial expressions, but not to objects.[4,47,48] Another study demonstrated that, compared with age-matched typical individuals, high-functioning adolescents and adults with autism exhibited longer N170 latencies to faces than to furniture, and the N170 failed to show a face inversion effect. Similar to findings in a study of 6-year-old children with autism,[47] scalp topography of the N170 did not show the normal right lateralized pattern, but rather was bilaterally distributed. On the basis of these studies, it appears that autism is associated with a life-long pattern of atypical neural responses to faces, which is characterized by a failure to show normal cortical specialization for face processing with slower than normal neural responses to faces.

Models of the Development of Neural Systems Mediating Face Processing: Implications for Understanding and Treating Autism

Different models of typical face-processing systems development have been suggested, each having different implications for our understanding and treatment of face-processing impairments in autism. One model posits that face-processing abilities are subserved by innate neural substrates that are specialized for processing faces (eg, a face processor in the fusiform gyrus) or for strategies used for expert face processing (eg, configural processor).[49] Alternatively, Nelson[50] has argued that, although an innate *potential* for cortical specialization for faces exists, such innate mechanisms are domain general (eg, visual processing in the inferior temporal cortex later becomes domain specific as the individual becomes experienced with faces).[51] Research suggests that early visual experience does influence the development of face processing.[52,53] Infants with bilateral congenital cataracts who later have their sight restored show subtle impairments in face recognition even after years of visual experience with faces.[53]

Following from these 2 models are different possibilities regarding the nature of face-processing impairments in autism. One explanation is that face-processing impairments represent *perceptual/cognitive* impairments such as a problem of perceptual binding, a failure to extract perceptually relevant information from faces (eg, prototype formation[54,55]), or dysfunction of the specific neural mechanism that supports face processing, namely, the fusiform gyrus.

A second explanation is that face-processing impairments are secondary to faulty input to the neural systems that subserve face processing during early sensitive periods when such systems are being developed. According to the *social motivation* hypothesis,[4,56] a fundamental 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. Motivational impairments might stem from abnormalities in either the reward system, per se,[57,58] or neural systems that might be important for the perception of social reward, such as the ability to form representations of others as being "like me," similar to the self in some way.[16,59,60]

If the social motivation hypothesis is correct, it should be possible to prevent or at least ameliorate face-processing impairments in autism through early intervention. Most early intervention techniques involve directing children's attention to faces and speech, and rewarding them for doing so. In this way, social interactions, in general, become more rewarding and meaningful to the child. Investigators at the University of Washington Autism Center in Seattle are conducting a randomized study of early intensive behavioral intervention for toddlers with autism in which outcome measures will include ERPs to faces, facial expressions, and speech stimuli (phonemes). It is hoped that studies examining the effects of early intervention on children's behavior and brain activity will not only have important implications for devising improved interventions but will also help elucidate the nature of neural abnormalities in autism.

Evidence for Impairments in Face Processing in First-Degree Relatives

Numerous studies have shown that relatives of individuals with autism, including parents and siblings, exhibit higher-than-normal rates of autism-related impairments.[61-68] One strategy for discovering autism susceptibility genes is to define genetically related quantitative traits, or endophenotypes, related to autism. Face-processing impairments might be one of these traits. On standardized tests of visual processing ability, parents of children with autism were found to have decrements in face processing relative to other visual spatial abilities. Dawson, Webb, and associates[69] examined performance of 143 parents of children with autism on standardized Wechsler cognitive tasks assessing verbal (vocabulary, verbal comprehension), visual spatial (block design, object assembly), and face recognition (immediate memory for faces) abilities. The tasks were compared with those performed by a sample of 1250 adults from the general US population. Investigators found that the parents of children with autism demonstrated a significant decrement on the face recognition test compared with their visual spatial and verbal abilities. In another study,[69] high-density ERPs to faces and to chairs were recorded from parents of children with autism and from control adults with no family history of autism. Control adults demonstrated the expected right larger than left hemisphere N170 to faces, whereas parents of children with autism demonstrated reduced right hemisphere N170 amplitude to faces, resulting in bilaterally distributed ERPs to faces. Moreover, whereas control adults exhibited the expected pattern of faster N170 to upright faces than to upright chairs, parents of children with autism showed no significant difference in N170 latency to upright faces vs upright chairs. On the basis of these results, it is hypothesized that face processing might be a functional neural trait marker of genetic susceptibility to autism.

Conclusion

By discovering autism susceptibility genes, we may eventually be able to identify newborn infants at risk for autism. Very early intervention could provide appropriate stimulation during the period when social brain circuitry is first developing. In this way, prevention or at least significant reductions in severity and range of symptoms of autism might eventually be possible, especially for children without significant comorbid mental retardation. By understanding the nature of impairments of social brain circuitry in autism, we will be able to develop more targeted interventions, focusing on those aspects of early communicative and social behavior that are fundamental to the syndrome.

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