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ANATOMY AND NEUROBIOLOGY OF AUTISM

Development and neural bases of face recognition in autism

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Autism is a developmental disorder characterized by impairments in communication and social relationships, and a restricted range of activities. This disorder is associated with specific impairments in the processing of social and emotional information. The nature of these impairments in social processing suggests that autism involves dysfunction of brain regions responsible for early stage processing of social information. Here, we explore further the nature of early social processing impairments in autism, and discuss evidence that autism involves an early emerging impairment in face processing. We begin by discussing research on face recognition in autism. Next, we describe new data suggesting that this impairment is present early in children with autism. We then explore how a neural system for face recognition might emerge in normal development, and speculate on differences in the development of this system in autism.

Studies of older children with autism have demonstrated impairments in face recognition.[1-4] For example, Klin *et al*[3] found that school aged children with autism performed worse on face recognition tests than developmentally disabled children without autism. Boucher and Lewis[4] reported that children with autism performed worse on picture matching and recognition tasks than typically developing children. They found that children with autism were not impaired on similar tasks using pictures of buildings. Thus, face recognition impairments in autism do not appear to be the result of a general difficulty in visual discrimination. In a fMRI study of face processing,[5] adults with autism failed to show activation of the fusiform face area, an area typically activated when viewing faces.

In an event-related brain potentials (ERP) study of adolescents and adults with autism, McPartland, Dawson *et al*[6] found that the ERP component associated with face processing ('N170') differed between participants with autism and normal participants. This component is typically larger to faces than to non-faces,[7] and is especially prominent over the right hemisphere. In individuals with autism, N170 was larger for furniture than for faces, and was bilaterally distributed. The latency of the N170 component peaked was correlated with the participants' score on a face recognition task.

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In another study that used ERP to study face and object recognition, Dawson and colleagues found that an abnormality in face processing is present early in autism.[8] Three- to four-year-old children with autism and comparison groups of children with developmental delay (DD) and typical development (TD) were shown photos of their mother's face and an unfamiliar woman's face, and of their favorite and an unfamiliar object. Children with DD and TD showed differential ERP activity to familiar vs unfamiliar faces and objects. Children with autism showed differential brain activity only to objects. For these children, the latency of P400 to faces was correlated with severity of a core autism symptom, joint attention. Joint attention is an early emerging and fundamental impairment in children with autism. Increased P400 latency to faces was associated with greater joint attention impairment ($r = 0.63$, $P < 0.0001$). This suggests the possibility that impairments in joint attention may be related in part to a failure to adequately process information regarding faces. Certainly faces hold significant information, such as direction of eye gaze, which is critical for establishing joint attention.

Normal development of face recognition. Evidence suggests that complex interactions between biology and experience contribute to the development of visual systems. For example, Shatz and colleagues[9] have shown that development of cortical-subcortical connections in visual systems relies on both prenatal activity and visual experience. Animal studies indicate that visual input is important in the development of normal binocular vision and the formation of ocular dominance columns in primary visual cortex.[10] The formation of neural circuits is also dependent upon patterned cellular activity. The mechanism for this experience-related development is likely a change in the strength of synaptic connections. One likely candidate for this mechanism is long-term potentiation, in which glutamate receptors change their affinity in response to activity.

The system that develops for face recognition is thought to be a specialized system with at least some innate components. Newborns are capable of recognizing faces.[11,12] There is evidence for very early specialization of the right fusiform gyrus for face processing. Using PET to study neurologically impaired infants while they were presented with faces *vs* non-face stimuli, Mazoyer *et al*[13] reported activation of the right fusiform gyms, among other areas of the brain. Morton & Johnson14 argued that early face recognition abilities

are served by a subcortical neural system, which is replaced by a cortical system that emerges by 6 months of age, the latter being less fragile and more experience-dependent.

Subcortical-cortical changes in the face processing system may experience expectant developments.[15] 'Experience-expectant' refers to developmental processes that involve a readiness of the brain to receive specific types of information from the environment during sensitive periods during which specific types of information are reliably present. Exposure to faces is a reliable experience for most infants. It has been argued that over-proliferation of synapses during early post-natal life reflects the brain readiness to receive expected information during this period with the sub-set of synapses being selectively retained partly depending on experience. In this way, there is some flexibility in the development of the system (ie, there may be individual differences in the final product depending on specific experience), but provided that the expected experience occurs, the system develops normally. In the case of face recognition, the theory would predict that exposure to faces in development leads to the expected outcome, a neural system that is specialized for faces. This theory is supported by evidence that neural areas (eg, fusiform gyros) typically activated in studies of face perception can be activated by non-face tasks when the persons performing those tasks have special expertise in the category of stimuli being tested (eg, bird identification).[16] In other words, specialization of fusiform gyrus appears to influenced by experience and furthermore, expertise. Thus, in development, as humans gain experience and expertise with faces, the neural system for face processing becomes increasingly specialized.

Development of face recognition in autism. If face recognition proceeds along the lines of the development of other specialized networks in the visual system, experience should play an important role in its development, supporting the experience-expectant model of face development. With this in mind, what might be the nature of brain dysfunction contributing to face impairments in autism? There are at least three possibilities. First, it may be the case that the innate 'starter set' for face recognition may be absent, or may be different from that found in typically developing children. That is, there might be genetically based differences in the neural components of the face system that do not allow the system to form typically. The second possibility is that, because children with autism fail to attend to social stimuli, including faces, they do not develop the expertise-needed for a specialized face recognition system to develop. Dawson *et al*[8] have hypothesized that the impairment in social attention seen in autism is related to a difficulty in forming representations of the reward value of social stimuli. Representations regarding the anticipated reward value of a stimulus begin to motivate and direct attention by the second half of the first year of life. A lack of normal attention to social stimuli, including faces, might result in a kind of deprivation of normal learning experiences with faces. In this case, it may be that the face recognition system is intact, but has been co-opted for development of expertise in another area.

Finally, it may be that there is an interaction between these options. It may be that the anatomical components of the system are in place, but that experience with faces does not facilitate the development of the face recognition system for children with autism as it does for typically developing children. One potential mechanism for this failure would be impairments at the molecular level. If there are problems with neurotransmitter systems, synaptic activity may not occur in the expected way in response to experience, preventing the formation of a face-specific visual system. Alternatively, neurotransmitter systems may be intact, but the mechanism for using neurotransmitter activity to form and develop neural systems may be impaired.

These are clearly empirical questions. Future research will be needed to clarify the roles of genetics, environment, and the interactions between them in the development of the system for face recognition.

There are implications of the abnormal developmental trajectories discussed above for how we intervene with young children with autism. If it were shown that children with autism fail to develop a specialized system for face recognition because they do not attend to faces, one focus for intervention would be to motivate children with autism to look at and take an interest in faces by making faces rewarding to them. This is, in fact, the approach taken by present early behavioral interventions for children with autism. Children are explicitly directed by the examiner to 'Look at me' and then rewarded for doing so. Even if the problem for children with autism is not in their motivation to attend to faces, but rather a difference in the starter set, changing the environmental input by training children to become face experts might provide important stimulation to this neural system that might have a remedial effect. If children with autism have a fundamental impairment in the face recognition neural system that is unresponsive to intervention, then the focus of intervention should be in helping to develop compensatory strategies for remembering faces.

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