Face recognition deficits: surprising new findings

Autistic individuals often have difficulty recognizing faces, and several reports have implicated deficits of a brain area known as the fusiform gyrus, which is associated with face processing. However, two new reports suggest that this explanation is inadequate.

Nouchine Hadjikhani and colleagues used functional magnetic resonance imaging to study facial perception in adults with autism spectrum disorders and nondisabled control subjects, showing the subjects pictures of faces, objects, and scrambled images. The researchers report, "Individuals with autism spectrum disorders activated the fusiform face area and other brain areas normally involved in face processing when they viewed faces as compared to non-face stimuli," indicating that the face-recognition deficits of autistic individuals do not stem simply from abnormalities of the fusiform gyrus.

This is consistent, Hadjikhani and colleagues say, with the fact that autistic individuals have milder face-processing deficits than those with prosopagnosia—"face blindness" known to be caused by defects of the fusiform gyrus—and with the fact that autistic individuals' deficits "appear to extend well beyond face identification to include a wide range of impairments in social perceptual processing."

In related research, Karen Pierce et al. report that functional MRI scans reveal "significant fusiform face area [FFA] activity in response to familiar and stranger faces in both autism and normal control groups." They also found that autistic individuals, like controls, show greater fusiform activity in response to pictures of familiar faces than in response to pictures of strangers, and exhibit normal right-hemisphere dominance during face processing. In addition, both groups exhibited a response to faces involving the posterior cingulate, amygdala and medial frontal lobes, although this response was more limited in autistic participants than in controls.

The researchers say their findings indicate that "dysfunction in the fusiform face area found in other studies of autism may reflect defects in systems that modulate the FFA, rather than the FFA itself."

Another research group, headed by James McPartland and Geraldine Dawson, recently analyzed event related potentials (voltage fluctuations associated with brain processing) in response to face processing in autistic and nondisabled individuals. They report that autistic individuals took longer to process faces, but not objects, than nondisabled subjects. In addition, while nondisabled subjects recognized right-side-up faces more quickly than upside-down faces, autistic individuals did not. Differences were also seen in brain lateralization, which the researchers say is "suggestive evidence of atypical cortical specialization for face processing."

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"Activation of the fusiform gyrus when individuals with autism spectrum disorder view faces," N. Hadjikhani, R. M. Joseph, J. Snyder, C. F. Chabris, J. Clark, S. Steele, L. McGrath, M. Vangel, I. Aharon, E. Feczko, G. J. Harris, and H. Tager-Flusberg, *Neuroimage*, Vol. 22, No. 3, July 2004, 1141-50. Address: Nouchine Hadjikhani, Martinos Center for Biomedical Imaging, Bldg. 36, First Ave., Room 417, Charlestown, MA 02129, nouchine@nmr.mgh.harvard.edu.

—and—

"The brain response to personally familiar faces in autism: findings of fusiform activity and beyond," K. Pierce, F. Haist, F. Sedaghat, and E. Courchesne. *Brain*, Vol. 127, No. 8, September 2004. Address: Karen Pierce, Department of Neurosciences, University of California at San Diego, La Jolla, CA 92093.

—and—

"Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder," J. McPartland, G. Dawson, S. J. Webb, H. Panagiotides, and L. J. Carver, *Journal of Child Psychology and Psychiatry*, Vol. 45, No. 7, 2004, 1235-45. Address: Geraldine Dawson, UW Autism Center, CHDD, Box 357920, University of Washington, Seattle, WA 98195, dawson@u.washington.edu.

Study detects high levels of anti-brain antibodies

Portuguese researchers studying 171 autistic individuals and their parents report that the autistic subjects exhibited high levels of antibodies against brain tissue—a finding that supports a growing body of evidence that autism involves an immune system attack on the brain. Susana Silva and colleagues say their analysis "did not provide any evidence that this reactivity was genetically determined."

One particular protein appeared to be involved in the autoimmune response, but the researchers were unable to identify it. The researchers suggest that the antibodies they detected "may represent the immune system's neuroprotective response to a previous brain injury occurring during neurodevelopment."

Editor's note: A more plausible explanation—and one that is consistent with the current autism epidemic that the researchers acknowledge in their paper—is that autism involves an autoimmune disruption stemming from environmental effects such as vaccination.

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"Autoantibody repertoires to brain tissue in autism nuclear families," S. C. Silva, C. Correia, C. Fesel, M. Barreto, A. M. Coutinho, C. Marques, T. S. Miguel, A. Ataide, C. Bento, L. Borges, G. Oliveira, and A. M. Vicente, *Journal of Neuroimmunology*, Vol. 152, No. 1-2, July 2004, 176-82. Address: Susana C. Silva, Instituto Gulbenkian de Ciencia, Rua da Quinta Grande 6, 2781-196 Oeiras, Portugal.

Autism and MMR vaccine

(continued from page 1)

in or after 1973 and diagnosed with pervasive developmental disorder between 1987 and 2001, as well as 4469 controls. The full medical records of a subset consisting of 300 of the individuals with PDD were checked to ensure a correct diagnosis, and they were matched to controls who did not develop PDD. The researchers report that among the entire group, the odds ratio for the association between having the MMR vaccine and having a pervasive developmental disorder was 0.86, suggesting that "MMR vaccination is not associated with an increased risk of pervasive developmental disorders." The findings were similar when the researchers studied only children with classic autism, restricted their data to children vaccinated before their third birthday, or used data only on children born before intensive media coverage regarding the MMR controversy began.

However, researcher Andrew Wakefield, who originally identified a link between MMR vaccination and a syndrome involving regressive autism and ileal-lymphoid-nodular hyperplasia, says the Smeeth study "has many failings" which invalidate its findings.

For example, Wakefield says, the researchers failed to identify children with regressive symptoms, and thus could not detect the subset of children who appear to develop normally before developing autism after MMR vaccination. Second, he says, the study numbers were far too small to be meaningful. Moreover, he says, only 25% of cases in this study were subjected to a review of case records to confirm a valid diagnosis. All of these problems, Wakefield says, "are of such significance as to invalidate the conclusion that MMR vaccine is not associated with onset of autism in children."

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"An investigation of the association between MMR vaccination and autism in Denmark," G. S. Goldman and F. E. Yazbak, *Journal of American Physicians and Surgeons*, Vol. 9, No. 3, Fall 2004, 70-75. Address: G. S. Goldman, Medical Veritas International, P.O. Box 847, Pearblossom, CA 93553.

—and—

"Scientists report start of MMR vaccinations linked to autism increase," *Schafer Autism Report*, September 7, 2004.

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"MMR vaccination and pervasive developmental disorders: a case-control study," L. Smeeth, C. Cook, E. Fombonne, L. Heavey, L. C. Rodrigues, P. G. Smith, and A. J. Hall, *The Lancet*, Vol. 364, No. 9438, September 11, 2004, 963-9. Address: L. Smeeth, Department of Epidemiology and Population Health, London School of Hygiene and Tropical Medicine, London, U.K., autism@lshtm.ac.uk.

—and—

"Dr. Wakefield responds to British study clearing MMR vaccines," news release, September 20, 2004.