Facial recognition impairment detected in autism

Autistic children respond to photos of their mothers in the same way that they respond to photos of strangers, a new study reveals, adding to evidence that autism involves defects in facial recognition.

Geraldine Dawson studied 34 autistic children, 21 non-disabled children, and 17 non-autistic, mentally retarded children, measuring their brainwave responses to photos. When she showed the children digitized photos of their mothers and strangers, Dawson reports, the non-disabled children and the retarded children recognized their mothers, but the autistic children did not. However, the autistic children did recognize the difference between photos of familiar and unfamiliar toys.

"Since all of the children in the study reacted similarly to toys and only the children with autism had problems with face recognition, it tells us autism is not a global problem," Dawson says. "Rather, it indicates an abnormality in those brain circuits responsible for social function. It highlights that autism is a disorder of the social brain."

Dawson reports that non-disabled children and those with mental retardation recognized their mothers, but autistic children did not. However, the autistic children did recognize the difference between photos of familiar and unfamiliar toys.

Dawson's research follows a report indicating that autistic individuals' brains respond abnormally when emotional facial expressions are viewed (see ARRI 14/4), and research indicating that the autistic brain processes faces as though they were objects (see ARRI 14/2).

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"Mother is just another face in the crowd to autistic children," press release, University of Washington, April 17, 2001. Dawson's research was reported at the annual meeting of the Society for Research in Child Development, April 19, 2001.

Breaking news (cont. from p. 1)

Chakrabarti, have just published a study in the *Journal of the American Medical Association* confirming the huge increase in the prevalence of autistic spectrum disorders reported in a number of other recent studies. Fombonne has been one of the most visible and outspoken adherents of the theory that the increase in prevalence is merely a reflection of greater awareness, not a genuine increase in numbers. (Details in next issue of the ARRI.) The study follows an April report by the Irish Society for Autism, which released preliminary survey data revealing an autism rate in Ireland of nearly 15 in 10,000 children—a threefold increase over the five per 10,000 rate found by a similar survey conducted only six years ago.

DAN! Conferences. The Spring 2001 DAN! Conference in Atlanta, Georga, was a sell-out. Plan to come to the Fall 2001 DAN! Conference in San Diego, October 5-7. Brochures will be mailed to all ARRI subscribers, or visit our web site at www.autismresearchinstitute.com.

Additional genes tagged as possible contributors to autism susceptibility

Research continues to uncover genes that may contribute to autism susceptibility. Among the investigations currently underway, in addition to those focusing on the reelin gene (see story in this issue), are:

• *Research into a genetic variant causing reduced adenosine deaminase (ADA) activity.* ADA plays an important role in the metabolism of purines, substances critical to the synthesis of RNA and DNA and to other cellular activities. Earlier this year (see ARRI 15/1), Antonio Persico et al. found that a gene variant known as ADA2, which appears to cause reduced ADA activity, was significantly more common in autistic subjects than in controls. Recently, N. Bottini et al., studying two Italian populations, came to a similar conclusion. The higher-than-expected frequency of the low-activity ADA gene in autistic subjects suggests, Bottini et al. say, that a genetically-determined reduction in ADA activity "may be a risk factor for the development of autism."

• *Research into the dopamine beta-hydroxylase (DBH) gene.* The DbetaH enzyme catalyzes the conversion of the neurotransmitter dopamine into another neurotransmitter, norepinephrine. A recent study by P.D. Robinson and colleagues found that in mothers with two or more autistic children, one type of DBH allele (DBH-), which appears to reduce serum DbetaH enzyme activity, occurs more commonly than in control subjects. In addition, mothers of two autistic sons were more likely to have two DBH- alleles than were controls, and DBH enzyme activity was reduced in the mothers as compared to controls. The researchers say their findings "suggest that lowered maternal serum DbetaH activity results in a suboptimal uterine environment (decreased norepinephrine relative to dopamine), which, in conjunction with genotypic susceptibility of the fetus, results in autistic spectrum disorder in some families."

• *Research into WNT2, one of a family of*
continued on page 6

Reelin gene variant linked to autism vulnerability

(continued from page 1)

hippocampi of subjects with schizophrenia, bipolar disorder, major depression, and lissencephaly (malformation of the brain, in which the brain surface is smooth rather than convoluted). Fatemi et al. speculate that different mutations may alter reelin production in varying ways, causing either profound disorders such as autism, lissencephaly, and schizophrenia, or milder problems such as depression or bipolar disorder, depending on the reelin levels associated with each mutation.

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"Reelin gene alleles and haplotypes as a factor predisposing to autistic disorder," A. M. Persico, L. D'Agruma, N. Maiorano, A. Totaro, R. Militerni, C. Bravaccio, T. H. Wassink, C. Schneider, R. Melmed, S. Trillo, F. Montecchi, M. Palermo, T. Pascucci, S. Puglisi-Allegra, K-L. Reichelt, M. Conciatori, R. Marino, C. C. Quattrocchi, A. Baldi, L. Zelante, P. Gasparini, and F. Keller, *Molecular Psychiatry*, Vol. 6, No. 2, March 2001, pp. 150-159. Address: Flavio Keller, Laboratory of Neuroscience, Libera Universita 'Campus BioMedico', Via Longoni 83, I-00155 Rome, Italy.

—and—

"Reduction in reelin immunoreactivity in hippocampus of subjects with schizophrenia, bipolar disorder and major depression," S. H. Fatemi, J. A. Earle, and T. McMenomy, *Molecular Psychiatry*, Vol. 5, No. 6, November 2000, pp. 654-663. Address: S. H. Fatemi, Department of Psychiatry, Division of Neuroscience Research, University of Minnesota Medical School, Box 392, Mayo Bldg., 420 Delaware St. SE, Minneapolis, MN 55455.

—and—

"Dysregulation of reelin and Bcl-2 in autistic cerebellum," S. H. Fatemi, J. M. Stary, A. Halt, and G. Realmuto, *Journal of Autism and Developmental Disorders*, in press. See address above.

—and—

"Reelin mutations in mouse and man: from reeler mouse to schizophrenia, mood disorders, autism and lissencephaly," S. H. Fatemi, *Molecular Psychiatry*, Vol. 6, 2001, pp. 129-133. See address above.

—and—

"A decrease of reelin expression as a putative vulnerability factor in schizophrenia," Francesco Impagnatiello, Alessandro R. Guidotti, Christine Pesold, Yogesh Dwivedi, Hector Caruncho, Maria G. Pisu, Doncho P. Uzunov, Neil R. Smalheiser, John M. Davis, Ghanshyam N. Pandey, George D. Pappas, Patricia Tueting, Rajiv P. Sharma, and Erminio Costa, *Proceedings of the National Academy of Sciences Online*, Vol. 95, No. 26, December 22, 1998, pp. 15718-15723.