Medical update:

Retinal abnormalities may be autism marker

Decreased function of the rods (light-sensitive structures in the retina of the eye) has been detected in four autistic individuals studied by Dr. Edward Ritvo and fellow U.S. researchers. Three of the subjects have autistic relatives, and the fourth has a brother who may be autistic.

The researchers believe the study subjects may have a low- level metabolic disturbance which, in addition to other symptoms, may cause decreased rod function. This eye abnormality could serve as a biological marker for an autism subgroup.

The researchers speculate that their findings may help explain why autistic individuals frequently prefer to use peripheral (side) vision; avoid eye contact; spin and flap objects in their peripheral vision fields; line up objects; and insist on "sameness" in their environment.

They suggest that "electroretinograms [exams of the retina] be recorded whenever practical in connection with complete neurological evaluations on autistic patients."

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"Retinal Pathology in Autistic Children—A Possible Biological Marker for a Subtype?" (letter), Edward R. Ritvo, M.D., B. J. Freeman, Ph.D., Donnell Creel, Ph.D., Alan S. Crandall, M.D., Carmen Pingree, M.A., Robert Barr, B.A., and George Realmuto, M.D.; *Journal of the American Academy of Child Psychiatry*, 25:137, January 1986. Address: Edward R. Ritvo, M.D., Department of Psychiatry, University of California at Los Angeles, Los Angeles, California 90024.

Hemisphere reversal in autism?

In the normal human brain, language processing takes place predominantly in the left hemisphere. Some researchers believe the language problems seen in autism may be caused by the failure of the brain hemispheres to specialize, so that the left hemisphere never fully takes over speech processing. Others believe that in autism language processing is reversed, occurring in the right instead of the left hemisphere.

Recent tests of normal and autistic subjects' "evoked responses" (brain-wave reactions to stimuli—in this case language and non-language sounds) support the "reversal" theory. While both normal and autistic responses to the tests were asymmetrical—that is, one side of the brain dominated the other—most of the autistic subjects in the study seemed to process speech on the right, rather than the left, sides of their brains. Individuals with both reversal and a high level of asymmetry—indicating that their right hemispheres were even more dominant than in the other subjects—had the poorest language skills, while subjects with the best language skills generally processed speech in the left or "normal" hemisphere.

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"Hemispheric Specialization and the Language Abilities of Autistic Children," Geraldine Dawson, Sheila Phillips, Larry Galpert, and Charles Finley; *Child Development*, 1986, 57, pp. 1440-1453. Address: Geraldine Dawson, Department of Psychology, University of Washington, Seattle, Washington 98195.

Imipramine study: behaviors of retarded adults worsened

Researchers who administered imipramine, an antidepressant drug, to 10 profoundly retarded adults—five who showed depression-like symptoms, and five who were restless, hyperactive and aggressive—found that the behaviors of both groups worsened as a result of the medication.

Imipramine caused increased irritability, lethargy, social withdrawal, hyperactivity and noncompliance in the test subjects. While the depressed individuals became even less active than usual during the imipramine trials, the activity level of the hyperactive individuals increased.

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"Preliminary Study of Imipramine in Profoundly Retarded Residents," Michael G. Aman, Carolyn J. Teehan, Anthony J. White and C. Vaithianathan; *Journal of Autism and Developmental Disorders*, Vol. 16, No. 3, 1986, pp. 263-273. Address: Dr. Michael Aman, Department of Psychiatry, School of Medicine, University of Auckland, Private Bag, Auckland, New Zealand.

Additional fragile site linked to autism

Canadian researchers found two cases of fra(2)(q13)--a fragile area on the #2 chromosome--when studying a group of 20 autistic subjects, while no fragile sites on this chromosome were found in a control group of 20 normal subjects (Parul Jayaker et.al.). In both cases where the fragile site was discovered, a parent was found to have the same chromosome variation.

Like Fragile X, a restriction on the X chromosome, the fragile 2 site is folate-sensitive; that is, the abnormality is only detected in the laboratory when cells are grown in cultures deficient in folic acid.

The researchers concluded that "the finding of fra(2)(q13) in two patients was unexpected", saying that while the defect has been seen in children with mental retardation and multiple congenital anomalies, a direct connection has not yet been proven since these fragile sites also are found in unaffected relatives.

The researchers also found that fragile (16)(q23)--a non- inherited fragile site on chromosome 16--"is not a useful marker for autism," as it appeared in both autistic and control-group subjects.

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"Fra(2)(q13) and Inv(9)(p11q12) in Autism: Causal Relationship?", Parul Jayakar, Albert E. Chudley, Mano Ray, Jane A. Evans, Jack Perlov and Rox Wand; *American Journal of Medical Genetics*, 1986, 23:381-392. Address: Albert E. Chudley, M.D., Section of Clinical Genetics, Children's Hospital of Winnipeg, 840 Sherbrook Street, Winnipeg, Manitoba, Canada R3A 1S1.

Viral infections, neural problems linked to psychoses

After studying four children who developed childhood psychoses following viral infections, researchers in London theorize that in children with existing neurological problems, viral infections of the central nervous system may contribute to the development of schizophrenia or other psychiatric disorders.

Three of the children in the study developed schizophrenia immediately following infections (measles, rubella and varicella), while one was diagnosed as having manic depressive illness shortly after contracting recurring Herpes simplex. All four children were prepubertal, and histories in all cases included birth complications and early developmental delays.

The researchers believe the viral infections are implicated in the development of the psychoses because the psychoses appeared immediately following the viral illnesses; the subjects showed EEG changes consistent with past inflammation of the brain due to viral infection; and two of the children had raised viral titres, while the cerebro-spinal fluid of a third showed indications of viral infection.

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"Viruses, Neurodevelopmental Disorder and Childhood Psychosis," K. P. Nunn, B. Lask and M. Cohen; *Journal of Child Psychology and Psychiatry*, Vol. 27, No. 1, pp. 55-64, 1986. Address: Dr. Bryan Lask, Department of Psychological Medicine, Hospital for Sick Children, Great Ormond Street, London WC1N 3JH, U.K.