Biomedical update:

More MRI evidence

More evidence of cerebellar defects in autism has been found by San Diego researcher James Murakami and colleagues (including Eric Courchesne, whose work was reported in ARRI Vol. 1, No. 1, and ARRI Vol. 2, No. 2).

The research team's most recent MRI scans, on 10 autistic subjects (including nine from the original study by Courchesne et al.), showed that in addition to having abnormally small cerebellar vermal lobes VI and VII—discovered in the earlier study—the subjects have smaller than normal cerebellar hemispheres. This finding adds support to autopsy studies showing a reduced number of cells not only in vermal lobules VI and VII, but also in the hemispheres of the cerebellum.

It appears, Murakami and colleagues say, that the defects seen in the MRI scans are due to a failure of neurons in these brain areas to develop properly, rather than to the atrophy of previously normal cells.

The cerebellum, a region of the brain at the base of the skull, coordinates motor activity and is directly or indirectly involved in speech and language, learning, attention, memory, emotional behavior, complex motivated behavior, and autonomic functions.

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"Reduced cerebellar hemisphere size and its relationship to vermal hypoplasia in autism," James W. Murakami, Eric Courchesne, Gary A. Press, Rachel Yeung-Courchesne, and John R. Hesselink; *Archives of Neurology*, Vol. 46, June 1989, pp. 689-694. Address: Eric Courchesne, Neuropsychology Research Laboratory, Children's Hospital Research Center, 8001 Frost Street, San Diego, CA 92123.

Diet changes reduce hyperactivity

According to a placebo cross-over study published in *Pediatrics*, more than half of 24 hyperactive boys improved when placed on a diet free of artificial colors and flavors, chocolate, MSG, preservatives, and caffeine. The researchers (Bonnie J. Kaplan et al.) also removed milk and other known "problem" foods from some children's diets.

Each child's behavior was rated by his parents during three phases:

1. a baseline period when the child ate home-cooked meals.
2. a placebo period when the child ate specially packaged food prepared by the researchers. Parents were not aware that these meals were nutritionally the same as the child's usual diet.
3. a four-week period when the children ate the experimental diet. These meals were pre-packaged by the researchers just as the placebo meals were.

Children on the experimental diet had significantly fewer behavior problems, slept through the night more often, and had less trouble falling asleep. Some decrease was also seen in bad breath and stuffy noses.

Because the children's blood levels of nutrients did not change during the study, the researchers say the improvements they saw were not due simply to the children eating a diet with more nutrients.

While they stress that all of the children in the study were hyperactive and difficult to manage even on the experimental diet, the researchers say their research "demonstrates a larger potential impact of diet than previously reported."

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"Dietary replacement in preschool-aged hyperactive boys," Bonnie J. Kaplan, Jane McNicol, Richard A. Conte, and H. K. Moghadam; *Pediatrics*, Vol. 83, No. 1, 1989, pp. 7-17. Address: Bonnie J. Kaplan, Department of Pediatrics, Alberta Children's Hospital, 1820 Richmond Road SW, Calgary, Alberta, T2T 5C7, Canada.

Retinal defect seen in more autistic individuals

Nearly half of 22 high-functioning people tested by Donnell J. Creel et al. had abnormal results on electroretinogram tests (exams which test the functioning of the retina of the eye). These results support earlier findings of retina abnormalities by the same researchers (ARRI Vol. 1, No. 1).

The abnormalities in these subjects resemble those seen in people with myotonic dystrophy, a hereditary muscle disorder, and the researchers speculate that "similar to some patients with myotonic dystrophy, a subset of autistics may have a low-level metabolic disturbance minimally detectable with the electroretinogram."

Only the rods in the eye, and not the cones, appear to be abnormal, and the researchers note that "the rod system is more sensitive to . . . minimal metabolic disturbance than is the cone system."

Another curious finding: many of the autistic subjects and their first-degree relatives had a youthful sheen on the retina; several parents had retinal sheens similar to those of teenagers. The researchers do not know if there is any connection between this normally healthy sign and the retinal abnormalities they found.

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"Abnormal electroretinograms in autism," Donnell J. Creel, Alan S. Crandall, Carmen Pingree and Edward R. Ritvo; *Clinical Vision Science*, Vol. 4, No. 1, 1989, pp. 85-88. Address: Donnell J. Creel, VA Medical Center, Salt Lake City, UT 84148.

Additional study reports success with lithium for manic symptoms

A case report by Ronald Steingard and Joseph Biederman adds more evidence that lithium can help autistic individuals with manic-depressive symptoms (see ARRI Vol. 2, No. 1).

One of the two individuals Steingard and Biederman treated had seasonal (fall/spring) manic episodes of agitation, aggression, self-injury, excessive laughter, and insomnia. High doses of chlorpromazine and haloperidol were relatively ineffective.

Since the 24-year-old autistic man began lithium four years ago, his self-injurious and aggressive behaviors have stopped, and his speech and social behavior have improved greatly. He also began sleeping through the night for the first time in 20 years. The lithium is still given with chlorpromazine, and withdrawing either drug leads to a recurrence of manic symptoms.

Steingard and Biederman also treated a nine-year-old autistic boy with manic symptoms and a family history of lithium-responsive manic depression. The boy, who began exhibiting manic behavior at age six, was overly elated, laughed excessively, and became agitated, aggressive, and self-injurious. Treatment with lithium (combined with his former medication, thioridazine) stopped the boy's inappropriate behaviors, outbursts, self-injury, and insomnia.

To help differentiate between manic-depressive symptoms and autistic symptoms, the researchers suggest comparing the individual's typical behaviors in "stable" periods with behavior changes in "unstable" periods. Symptoms which might indicate mania, they say, include increased intrusiveness, assaultive behavior, destructiveness, and self-injury; reduced appetite; and insomnia.

Because lithium can have major side effects, including thyroid suppression and kidney damage, most experts caution against treating children with the drug for more than six months unless absolutely necessary. Lithium is rarely used to treat children and adolescents, partly because it is difficult to make an accurate diagnosis of manic and/or depressive disorders at such a young age.

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"Lithium responsive manic-like symptoms in two individuals with autism and mental retardation," Ronald Steingard and Joseph Biederman; *Journal of the American Academy of Child and Adolescent Psychiatry*, 26/6, 1987, pp. 932-935. Address: Ronald Steingard, Pediatric Psychopharmacology Unit, ACC 625, Massachusetts General Hospital, Boston, MA 02114.

—and—

Getting Better, quarterly bulletin of the Huxley Institute, 1st quarter, 1989.