Biomedical Update:

Malnutrition-caused eye disease a risk in autistic children

A new report cautions that the odd and restricted diets of many autistic individuals can lead to serious health problems.

Physicians Thomas Steinemann and Stephen Christiansen recently evaluated a five-year-old autistic patient with corneal ulcers. Questioning the boy's mother, the physicians learned that the child ate only bacon, muffins, and Kool-Aid, and refused to take vitamin supplements.

Tests revealed that the child had an extremely low level of vitamin A, and was suffering from xerophthalmia. This condition causes abnormal dryness of the eyes and, if untreated, can lead to blindness. In this case, Steinemann and Christiansen were able to successfully treat the child's vision problems.

Previously (see ARRI 7/4), physician Joseph Clark reported treating an autistic child who had both rickets and eye problems, caused by deficiencies of vitamins A and D.

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"Vitamin A deficiency and xerophthalmia in an autistic child," Thomas L. Steinemann and Stephen P. Christiansen; *Archives of Ophthalmology*, Vol. 116, March 1998, pp. 392-393. Address: Thomas L. Steinemann, Department of Ophthalmology, Jones Eye Institute, University of Arkansas, 4301 W. Markham Street, Mail Slot 523, Little Rock, AR 72205.

More on Prozac for autistic symptoms

A new study supports earlier findings that Prozac (fluoxetine) can reduce symptoms of autism in some individuals, but can also have adverse effects on behavior.

S. Hossein Fatemi and colleagues reviewed the charts of seven autistic teenagers and adults who had been treated with Prozac (20 to 80 mg per day) for periods ranging from 1 to 32 months. The subjects, three males and four females, ranged in age from 9 to 20.

The researchers report that behavioral tests given during treatment showed improvements in irritability, lethargy, stereotyped behaviors, and inappropriate speech. The "lethargy" category, they note, includes items related to social behavior, indicating that the drug improved subjects' social responsiveness.

Fatemi et al. note, however, that hyperactivity increased in four patients, and that two patients discontinued the treatment—one because of increased agitation, and another because of increased depression. Other reported side effects included temporary loss of appetite, and chronic vivid dreams.

The researchers say five previous studies have indicated that Prozac can reduce autistic symptoms. Three of these studies, they add, also noted increases in hyperactivity or anxiety.

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"Fluoxetine in treatment of adolescent patients with autism: a longitudinal open trial," S. Hossein Fatemi, George M. Realmuto, Lubna Khan, and Paul Thuras; *Journal of Autism and Developmental Disorders*, Vol. 28, No. 4, August 1998, pp. 303-307. Address: G. M. Realmuto, University of Minnesota, School of Medicine, Department of Psychiatry, 420 Delaware Street, Box 393 UMHC, Minneapolis, Minnesota 55455.

Chromosome 15: evidence mounts for role in autism

A report by Richard Schroer and colleagues adds to growing evidence that defects on chromosome 15 may cause a significant number of cases of autism.

Schroer et al. are involved in the South Carolina Autism Project, an in-depth study of the causes of autism. "Among the first 100 cases enrolled in the project," the researchers say, "abnormalities of chromosome 15 have emerged as the single most common cause [of autism]."

Four of the first 100 autistic subjects evaluated by the researchers had either duplications or deletions of areas of this chromosome. In all cases, the researchers say, the individuals inherited the defect from their mothers. This finding supports earlier research by Edwin Cook and colleagues (see ARRI 11/4), which found that maternally inherited duplications of one region of chromosome 15 can lead to autism, while similar duplications inherited from fathers do not.

Schroer and colleagues conclude that "duplications and deletions of the maternally derived chromosome 15 should be considered in all cases of autism, and particularly in those associated with mental retardation and seizures."

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"Autism and maternally derived aberrations of chromosome 15q," Richard J. Schroer, Mary C. Phelan, Ron C. Michaelis, Eric C. Crawford, Steven A. Skinner, Michael Cuccaro, Richard J. Simensen, Janet Bishop, Cindy Skinner, Don Fender, and Roger E. Stevenson; *American Journal of Medical Genetics*, Vol. 76, 1998, pp. 327-336. Address: Roger E. Stevenson, Greenwood Genetic Center, One Gregor Mendel Circle, Greenwood, SC 29646.

Another chromosome defect implicated

Although Fragile X syndrome and defects of chromosome 15 are the most commonly identified genetic defects in autistic children (see previous article), a number of other chromosome defects also are tentatively linked to autism. Researchers in Spain and Brazil recently added another candidate to the list: a defect on chromosome 22.

F. B. Assumpcão, Jr., recently identified a chromosome 22 alteration in a 13-year-old autistic male. In this case, one of the child's two copies of chromosome 22 was fused in a ring shape and was missing a segment. Although the child's mother was a schizophrenic who took psychotropic drugs during pregnancy, Assumpcão says that the nature of the child's symptoms suggests that the chromosome aberration is the cause.

F. Carratala and colleagues recently reported on a three-year-old, non-verbal autistic and retarded child whose chromosome analysis also revealed an aberration of chromosome 22. In this case, the test revealed a translocation between chromosome 20 and 22, and a deletion in the q11 region of chromosome 22.

"Although [such deletions] are responsible for the DiGeorge syndrome," the researchers note, "clinical, metabolic, and neurological image studies of the patient were inconsistent with this syndrome." The child studied by Carratala and colleagues had mildly unusual facial features, a concave chest, and a short thumb.

Assumpcão says his findings show the importance of clinical studies, and particularly genetic testing, of children with autistic symptoms.

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"Brief report: a case of chromosome 22 alteration associated with autistic syndrome," F. B. Assumpcão, Jr.; *Journal of Autism and Developmental Disorders*, Vol. 28, No. 3, June 1998, pp. 253-256. Address: F. B. Assumpcão, Jr., Child and Adolescent Center, Institute of Psychiatry, Medical School, São Paulo University, São Paulo, Brazil.

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"A patient with autistic disorder and a 20/22 chromosomal translocation," F. Carratala, F. Galan, M. Moya, X. Estivill, M. A. Pritchard, R. Llevadot, M. Nadal, and M. Gratacos; *Developmental Medicine and Child Neurology*, Vol. 40, No. 7, July 1998, pp. 492-495. Address: F. Carratala, Paediatric Department, Miguel Hernandez University, Elx, Alicante, Spain.

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