Volume 11, No. 3 **Defeat Autism Now! Conference, Sept. 19-21 (see page 7)** 1997

Autism Research Review INTERNATIONAL

A quarterly publication of the Autism Research Institute

Reviewing biomedical and educational research in the field of autism and related disorders

'Thalidomide babies' alter theories about autism

Thousands of pregnant women who took the drug thalidomide for morning sickness during the 1960s gave birth to children with severe birth defects, including missing arms and legs. Now, these thalidomide victims are offering major new clues about the genesis of autism.

Patricia Rodier and colleagues were intrigued several years ago when they heard two ophthalmologists, who had been studying eye movements in thalidomide-affected adults, report that the rate of autism in these individuals was extremely high. Furthermore, the doctors said they were able to pinpoint the time at which the brain damage occurred that caused autistic symptoms. One third of individuals exposed to thalidomide between the 20th and 24th days of gestation developed autism, while *none* of those exposed to the drug during other periods of gestation became autistic. Thus, the critical period for autism in thalidomide-exposed children was during the development of the brain stem, the connecting link between the spinal cord and the rest of the brain.

Curious as to whether autistic people *not* exposed to thalidomide might also have damaged brain stems, Rodier et al. examined brain stem tissue from a deceased autistic woman. "The brain stem of the autistic case was abnormal in several ways," they note. First, the facial motor neurons were almost completely missing. Non-disabled individuals have about 9,000 of these neurons; the autistic woman had only about 400. The "superior olive," a brain structure involved in hearing, was also missing. In addition, the brain stem was significantly shorter than normal.

Brain stem damage, the researchers say, may explain many autistic symptoms. For instance, some autistic individuals appear expressionless. "It is not that some of these children with autism do not want to smile," the researchers hypothesize; "it is that they cannot smile," because neurons in the brain stem that control smiling are absent. Abnormal eye movements may also stem from a shortage of facial motor neurons.

Rodier et al. say early brain stem damage could explain both the high incidence of hearing problems in autism (because of abnormalities in brain stem areas associated with hearing) and the high incidence of ear malformations, because the ear begins forming around the 23rd day of gestation. (Data from one large study revealed that 42 percent of autistic subjects had "posteriorly rotated" ears, in comparison to only 18% of control subjects.)

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Rodier et al. say studies on rats show that genetic defects, specifically a defect in a gene called Hoxa-1, could cause the early brain stem damage seen in non-thalidomide-exposed autistic individuals. They add, however, that "while our working hypothesis is that the autopsy case's defects are genetic in origin, we must remember that her anomalies are similar to those of the thalidomide cases, and thus, may reflect an exposure to some injury." Whatever the cause, they stress, "the defect we have described could have arisen only at the time of neural tube closure," as the brain stem begins developing.

The researchers say the cerebellar defects seen in many autistic individuals are not surprising, even though the cerebellum hasn't even developed at the time their hypothesized autism-causing brain stem defect appears. "What we think happens," Rodier said, "...is a 'downstream' effect in which damage to one structure leads to damage to another structure, down the line." In other words, she says, "the loss of some early-forming cells, as we see in autism, not only deprives the individual

continued on page 7

Nutrient therapy leads to improvement

Nutritional therapy individually tailored to an autistic child's needs can result in significant improvements in behavior and health, according to a recent open clinical trial by H. Ron Isaacson et al.

Isaacson and colleagues evaluated 41 consecutive autistic or autistic-like patients between the ages of 3 and 27 who underwent nutrient therapy at the Pfeiffer Treatment Center in Illinois. The treatment, the researchers note, "is based upon an in-depth interview, physical exam, and approximately 80 laboratory analyses." These analyses revealed a high incidence of copper/zinc imbalances, pyroluria (a condition that can result in the depletion of vitamin B6 and zinc), lead and cadmium toxicity, malabsorption problems, and abnormal histamine levels.

Subjects underwent four or more months of therapy (generally receiving five to ten nutrients, including B6, magnesium, and dimethylglycine). Isaacson et al. report that following therapy, "Nine [22%] showed major improvement [and] 30 [73%] showed significant improvement while two [5%] showed little or no improvement." The greatest improvements were seen in health, thought processing, sleeping patterns, speech, tantrum reduction, socialization, and reduced repetitive behavior, while little change was seen in compulsive behaviors.

The researchers conclude that "nutrient therapy, as applied on an individual basis, is quite effective in ameliorating the symptoms of autism and related disorders." They also note that the high levels of pyroluria seen in study subjects may explain why vitamin B6 benefits about half of autistic individuals.

Noting that biochemical patterns varied widely among study subjects, the researchers say their results "demonstrate that autistic patients must be treated as individuals."

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"Autism: a retrospective outcome study of nutrient therapy," H. Ron Isaacson, Marsha M. Moran, Anmarie Hall, Bunny J. Harmon, and Mary A. Prekosovich; *Journal of Applied Nutrition*, Vol. 48, No. 4, 1996, pp. 110-118. Address: H. Ron Isaacson, Health Research Institute, 1804 Centre Point Drive, Suite 106, Naperville, IL 60563.