Autism and Allergies

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relationship between food and mood when I reintroduced the cow's milk. Within an hour he was having a violent, self-destructive temper tantrum.

It is also important to point out that Tony got better when I first removed milk, but then he got worse. Other parents have had this same experience. It must have been some kind of withdrawal. About a month after I removed milk from Tony's diet permanently he began the worst temper tantrum of his life. It lasted nine days—but it was his last temper tantrum. When that horrible period was over his language and social skills began to improve dramatically. It was the beginning of a new life for him.

BUT IT WAS NOT AN OVERNIGHT MIRACLE! We had years of work ahead of us. The difference made by the removal of cow's milk was that our work paid off. Before that, we met nothing but dead ends.

Cow's milk is the primary offender in most of the cases I have become familiar with, but it is not the only one. Wheat and sugar come up almost as often. Some mothers were so impressed by the reaction their children had to Gummy Bears that they asked me to pass the information on. Test for Gummy Bears!

Finally, I have seen an interesting trend in the last year of talking to parents. At first, I heard horror stories of the resistance of doctors to the idea that behavior problems can be caused by allergy. Normally calm physicians yelled, "Don't you even think about it!," and "That's quackery!" to mothers who wanted help with elimination diets. But as time has gone by there has been a change. Maybe it is a coincidence or maybe it has something to do with the fact that Dr. Art Ulene supported me on the *Today Show*. But one doctor even said to a mother, "A year ago I would have told you it was nonsense to test an autistic child for allergy, but I'm beginning to think differently because of a couple of books that have come out." That child tested positive for a milk allergy and is now on that slow uphill course that took Tony home.

My goal is that someday the pediatric books will list allergy in the differential diagnosis of children with behavior disorders. But that could be a long way off. Until then, parents may have to play doctor themselves with the help of books like Dr. Crook's and support from each other. It's not easy. But take heart. As one pediatric allergist told me on a Pittsburgh talk show, "The truest test of allergy is to expose, withdraw the exposure, then expose again. If the results obtained by the mother's own observation differ from those obtained in the lab, we would always go with the mother." So you are your child's best hope, with or without the doctor. If there is a miracle to be had, you can make it happen.

**Send SASE to ARRI for list of allergy books.*

Role of genes in autism is explored

(Continued from page 2)

Tests are now available to help families with Fragile X relatives determine their risk (see box on page 2). However, there is no totally accurate test for Fragile X.

"Unfortunately, because of the limitations of carrier detection in DNA analysis and prenatal diagnosis, reproductive decisions can be particularly difficult," Cronister says.

Cronister recommends that both male and female siblings of males with Fragile X be tested for the disorder. "Aunts, uncles, cousins and other extended family members should also be tested if they are at risk for either carrying or being affected by the Fragile X gene," she says.

Many researchers suggest that autistic males be screened for Fragile X, especially if they show physical symptoms of the syndrome. These include large or prominent ears, hyperextensible finger joints, large testicles, a simian crease (single crease across the palm), and a long face. (However, if an autistic child has many siblings, cousins, uncles and aunts who are not mentally handicapped, Fragile X is unlikely.)

Diane R. Edwards et al. recently reported on two autistic girls with Fragile X, who showed only mild physical symptoms. They recommend that all girls with autism of unknown cause be screened for Fragile X, since its hereditary pattern and subtle expression in autistic girls "result in a marked underdiagnosis of the syndrome." Christopher Gillberg et al. also report on a set of identical twin girls with Fragile X and autism. These girls, too, did not show the typical physical signs of Fragile X.

Gene mapping: no answers yet

Some researchers are using highly sophisticated laboratory gene mapping techniques to attempt to isolate chromosome areas which may be linked to autism.

So far, there is no definite "marker" for autism. However, Spence et al. reported in 1985 that for families with at least one affected female, the most likely genetic linkage was with the "ABO" locus on chromosome 9, while in families with only affected males, the most likely linkage was with the esterase D locus on chromosome 13. Overall, haptoglobin (HP), on the #16 chromosome, showed the strongest linkage with autism.

Among the most important genetic markers are the HLA antigens (molecules produced by genes on the #6 chromosome, which give each person's cells a unique "fingerprint"). Various HLA antigens have been associated with specific disorders ranging from lupus to hay fever. (HLA antigens do not *cause* a disorder, but simply indicate that the gene for the disorder and the gene for the HLA antigen are "neighbors" on a particular chromosome.) However, Spence et al. found no association between specific HLA antigens and autism; also, they found that autistic siblings did not inherit similar HLA antigens to a greater degree than non-autistic siblings, making a connection between specific HLA antigens and autism unlikely.

Other scientists are studying biochemical abnormalities seen in many autistic children—for instance, high levels of the brain transmitter serotonin and reduced activity of the enzyme dopamine-B-hydroxylase (DBH)—to see if familial patterns can be found. Because the major gene for DBH is linked to the ABO locus on chromosome 9, Smalley et al. say researchers should "direct [their] attention to this region of chromosome 9 in investigating the genetics of at least one form of autism."

Smalley feels one fruitful avenue of research would be the study of non-retarded vs. retarded autistic people, and autistic people with and without minor physical abnormalities, to determine if there are distinct genetic differences between the groups.

Researchers Larry Burd and Jacob Kerbeshian have found evidence that one genetic subgroup of autism involves autistic or autistic-like children who also have both Tourette's Syndrome and hyperlexia. (Hyperlexics learn to "read" very early and decode words well, but have little comprehension of what they read). In a population of about 200,000 people, Burd and Kerbeshian identified five people with all three conditions, which they believe suggests genetic linkage. It is interesting, they say, that these individuals tended to make much better progress than autistic people without Tourette's Syndrome and hyperlexia.

Gene mapping and other techniques may some day tell us exactly what role genes play in autism. For now, researchers emphasize the need for parents of autistic children to receive adequate counseling about possible genetic risks.

"The assessment of a patient with autism should . . . include a careful and extensive family history," says researcher Susan Folstein in the *American Review of Medicine*. "Inquiry should be made not only about other autistic children in the family, but also about family members with any cognitive, reading, or language deficits. The family history should be extensive enough to discern patterns of inheritance that might be compatible with X linkage, such as other affected persons among the relatives of the autistic child's mother, and an absence of father-to-son transmission.

"If patterns of dominant inheritance with reduced penetrance are found," she says, "the possibility of tuberous sclerosis or neurofibromatosis should be considered."

While the overall risk of a parent of an autistic child having another affected child is between two and three percent, Folstein notes that "if there is no suggestion of other affected family members, and if the autistic child suffered severe perinatal injury or some infectious condition, the risk of recurrence is likely to be negligible."

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List of references available upon request. Please send SASE.