Biomedical/Education Update:

Mild autistic traits seen in some parents; "good gene" excess in autism?

Symptoms of autistic children are sometimes seen in very mild forms in their parents, according to two studies by Rebecca Landa et al., who say their findings indicate that such families have a "genetic liability" that may manifest itself in other family members as mild social or language disabilities.

The researchers first tested the ability of parents of autistic children, and a group of control subjects, to tell a narrative story. They found that a subgroup of the autistic parents (34%) produced stories that were rambling, incoherent, plotless, or otherwise indicative of defects in spontaneous narrative speech. In contrast, only 13% of controls produced such stories.

A second study by Landa et al. focused on the use of social language by parents of autistic children. After two-hour sessions with participants and controls, raters evaluated their conversational skills. Parents of autistic children, they report, had more abnormal scores on measures of disinhibition, awkward or inadequate expression, and odd verbal interaction (strange humor, inappropriate topics, etc.).

Landa et al. stress that their tests revealed mild defects which did not hinder the parents in forming friendships and social contacts.

Editor's note: An equally plausible hypothesis is that parents of autistic children, keenly aware of the parent-blaming attitudes of many professionals, became resentful and uncomfortable with the "psychiatric" tasks they were to perform.

Genetic liability or advantage?

English researcher Robin Clarke has an intriguing theory: that autism may result from an excess of normally beneficial genes. This, he says, would explain evidence that parents of classically autistic children tend to be highly intelligent and come from high socio-economic groups. Clarke's proposal is a variation of Bernard Rimland's 1964 theory that autism may result from a "double dose" of genes that permit intense mental concentration, and thus high levels of cognition and achievement.

Unlike Rimland, Clarke proposes that autism may result from an excess of genes or other factors that *inhibit* the expression of other genes. These factors, he says, may have "a quality-controlling effect, tending to suppress recently acquired idiosyncracies (which tend to be disadvantageous) and leaving those characteristics which have a relatively substantial history of advantageousness."

An excess of such genes, Clarke says, "would eliminate or suppress not only disadvantageous or neutral characteristics but also significantly advantageous, even vital ones." The suppression of idiosyncracies in appearance, he says, could explain why autistic children often are physically very attractive. And suppression of "newer" genes in the evolutionary process could cause defects in more recently evolved skills such as communication and socialization, while leading to the resurfacing of "old" prehuman genes that may cause such behaviors as posturing, flapping, and bursts of running, which are normal behaviors in animals.

Clarke proposes that classical autism is caused by such genetic factors, while environmental factors such as disease or pollution cause a less "pure" syndrome.

Editor's note: Despite the almost complete absence of overlap in the lists of references in our two works, Clarke's theory is remarkably similar in many ways to that presented in my 1964 book, **Infantile Autism: The Syndrome and its Implications for a Neural Theory of Behavior.**

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"Social language use in parents of autistic individuals," R. Landa, J. Piven, M.M. Wzorek, J.O. Gayle, G.A. Chase and S.E. Folstein; *Psychological Medicine*, 1992, 22, 245-254; and "Spontaneous narrative-discourse performance of parents of autistic individuals," *Journal of Speech and Hearing Research*, Vol. 34, December 1991, pp. 1339-1345. Address for both: Rebecca Landa, The Johns Hopkins University, School of Medicine, 600 North Wolfe Street, Meyer 2-181, Baltimore, MD 21205.

—and—

"A theory of general impairment of gene-expression manifesting as autism, Robin P. M. Clarke; *Person. individ. Diff.*, 1993, 14, 465-482. Address: Robin P. M. Clarke, 9 Augusta Road, Moseley, Birmingham, B13 8AJ, England.

Task variation in P.E.

Studies show that autistic students perform academic work better, and have fewer behavior problems, when they work on a variety of tasks during a class period rather than performing the same task over and over again until it is mastered. In addition, students perform better at academic activities when tasks they haven't mastered are interspersed with tasks they have already learned.

A new study indicates that both of these approaches—task variation, and interspersing previously mastered tasks—also increase the performance of autistic students in physical education programs.

Robert Weber and Joanne Thorpe evaluated the progress of 12 autistic P.E. students, ages 11 to 15, under two conditions:

—"constant task," in which only one task was presented repeatedly until the skill was learned, or until the class was over.

—task variation plus maintenance tasks. In this condition, students participated in six different tasks plus three previously learned tasks during their 45-minute P.E. sessions.

Six skills were taught to the students in each condition, but in the first condition each skill was mastered before the next was introduced.

The researchers found that the task variation approach, with interspersed maintenance skills, was superior to the constant task condition in teaching basic gross motor skills. "Randomly interspersed tasks being changed every two or three minutes appears to be highly effective because of the limited attention span of people with autism," they suggest, adding that "the use of maintenance tasks interspersed with new tasks may logically contribute to the students' retention of the skills previously learned."

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"Teaching children with autism through task variation in physical education," Robert Weber and Joanne Thorpe; *Exceptional Children*, September 1992, Vol. 59, No. 1, pp. 77-86. Address not given.

Immune attack could explain myelin defect

ARRI recently (6/4) reported research suggesting that autism involves a brainstem defect caused by faulty formation of myelin, the fatty coating that insulates the nerve cells forming the "white matter" of the brain and spinal cord. Now U.S. researcher Vijendra K. Singh and colleagues, who studied 33 autistic children, report finding signs in many of an immune system attack on myelin basic protein (MBP), a component of myelin.

Singh et al. found that 19 of their 33 subjects were positive for antibodies to MBP— a rate more than six times higher than in nondisabled and retarded control subjects they tested.

The researchers point out that autism and autoimmune disorders (in which the body attacks itself, as in multiple sclerosis and lupus) share several features: a tendency to occur in individuals with family histories of autoimmune disease; an increased incidence in one sex (in the case of autism, males); a possible link with viral infections; and evidence of association with certain forms of genes that are part of the major histocompatibility complex (the genes that differentiate "self" from "other," telling the body which cells to attack).

Singh et al. say that "if an immunological assault perhaps secondary to a virus infection were to occur prenatally or postnatally during infancy or early childhood, it could possibly result [in] poor myelination or abnormal function of the neuron-axon myelin."

The researchers note that "delayed or incomplete myelination in the corpus callosum (which is the largest myelinated area of the brain) has been suggested as the basis of auditory processing problems in some children with learning disabilities." Auditory processing deficits appear to play a major role in autism as well.

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"Antibodies to myelin basic protein in children with autistic behavior," Vijendra K. Singh, Reed P. Warren, J. Dennis Odell, W. Louise Warren, and Phyllis Cole; *Brain, Behavior, and Immunity*, in press. Address: Vijendra K. Singh, Biomedical Division, Center for Persons with Disabilities, Utah State University, Logan, UT 84322-6800.