Autism Research Review

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

Does tetanus play a role in autism?

Researchers have many theories about what causes autism, but Ellen Bolte recently advanced one of the most fascinating: that some cases of autism stem from chronic tetanus infections.

Studies show, Bolte says, that "very small amounts of tetanus neurotoxin injected directly into the brains of laboratory animals create a stereotyped behavior syndrome" including repetitive sniffing, gnawing, and running Animals infected with tetanus also show changes in behavior, learning ability, and memory. The same drugs that suppress stereotyped behaviors in autistic individuals, Bolte says, also suppress these behaviors in toxin-exposed animals. In addition, she notes, researchers use tetanus neurotoxin to cause epilepsy in animals—and epilepsy is a very common symptom in autism.

Bolte notes that tetanus infection can occur even in immunized individuals. Also, she notes, animal research demonstrates that tetanus infection of the intestinal tract—unlike infections in wounds—can lead to neurological symptoms without causing muscle spasms. Tetanus infection can occur in forms so subtle that many patients (22 percent, in one study) do not have recognizable symptoms, and chronic tetanus infection has been diagnosed in humans.

Bolte believes that intestinal tetanus infections may occur in children chronically exposed to antibiotics given to treat ear infections. She notes that these antibiotics can markedly increase the risk of infection of the intestinal tract by pathogenic bacteria, including the very common *Clostridium tetani*. A number of autism's features, Bolte says, could be explained by a sub-acute, chronic tetanus infection. Among them:

- Autism occurs in boys four times as often as in girls—a fact believed to indicate genetic influences. But Bolte notes that "tetanus infection is also documented to occur in males four times more often than in females."
- Autistic children often have too-high or too-low muscle tone, and some have difficulty chewing and swallowing. These probcontinued on page 6

DAN! Conference a huge success!

The Defeat Autism Now! (DAN!) Conference, held October 3 and 4 in Cherry Hill, New Jersey, was sold out weeks in advance. Speaker after speaker provided the audience with new and useful information. "Wonderful conference!" one attendee wrote. "I feel like I've been drinking from a fire hose!"

The highlight of the conference was the impassioned address by parent Victoria Beck, whose account of her uphill struggle to establish the autism-secretin connection brought a standing ovation from the crowd of 1,200.

"The best, most overwhelming, most articulate talk I've ever heard," said Maureen McDonnell, R.N., coordinator of the conference. Noting that there was hardly a dry eye in the house, McDonnell added, "the next time she speaks, I'll bring boxes of tissues to hand out to the audience."

While many other topics were also addressed, interest in secretin was especially high. Sudhir Gupta, M.D., Ph.D., provided an intriguing overview of the immunological aspects of secretin, while Sidney Baker, M.D., presented an analysis of his findings, based on the first 39 autistic children to whom he had given secretin. Popular demand resulted in an impromptu panel discussion by 11 physicians of their experiences with secretin.

Other highlights of the conference included an update on the studies in the UK of possible vaccine related autism, by Professor Andrew Wakefield, and the first public announcement of a biological marker for autism by chemist Alan Friedman, Ph.D. Sydney Walker, M.D., discussed his new book, *The Hyperactivity Hoax*.

Next DAN! Conference? Watch this space!

Prozac: remarkable changes in children with family histories of depression

A new study indicates that fluoxetine (Prozac) causes dramatic improvement in some autistic children—in particular, those with a family history of major affective disorders.

G. Robert DeLong and colleagues administered fluoxetine (0.2 to 1.4 mg/kg/day) to 37 autistic children between the ages of 2 and 7, in an open-label trial. Children with known causes of autism, such as chromosomal disorders, were excluded. At the time of the researchers' report, the children had been treated for periods ranging from 13 to 33 months.

The researchers say that eleven subjects had an excellent response to the drug, improving to the degree that they could attend regular education classes. Another eleven had a good response, but "remained identifiably autistic." Fifteen children did not improve or worsened, developing hyperactivity, agitation, or lethargy.

DeLong et al. say that the subjects who had a positive response to the drug showed improvements in mood and temperament (including increased happiness, fewer tantrums, less agitation, and more animation); increased ability to relate to others (including better eye contact and more initiation of social exchanges); cognitive improvements; and a reduction in stereotyped behaviors, fixations, and problems with transitions. In addition, remarkable improvements were seen in many children's language skills. These included increased complexity, accuracy, and appropriateness of language, greater desire to speak, and increases in both imitative and spontaneous speech.

Although nearly all of the children in the study were receiving other treatments, including other drugs and behavior modification therapy, the researchers say that "in those children for whom fluoxetine was effective, its

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