# Lancet study links autistic symptoms, MMR vaccine

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ficiency"—a deficiency also seen in other gastrointestinal disorders. Noting that vitamin B12 is essential for normal myelination of cells in the central nervous system, a process that is not complete until around age 10, the researchers speculate that "B12 deficiency may... be a contributory factor in the developmental regression" seen in their subjects.

The researchers say it is possible that the association of gastrointestinal disorders and autistic-like symptoms in their subjects was merely chance. They argue, however, that "the uniformity of the intestinal pathological changes and the fact that previous studies have found intestinal dysfunction in children with autistic-spectrum disorders, suggests that the connection is real and reflects a unique disease process."

Wakefield and colleagues caution that they "did not prove an association" between the MMR vaccine and the behavioral and gastrointestinal abnormalities seen in their subjects. However, they say, rubella infection is a known risk factor for autism, and several researchers have tentatively linked the MMR vaccine to cases of autism. Furthermore, Wakefield et al. say, both the measles virus and the measles vaccine have been implicated as risk factors for the development of Crohn's disease, a disorder that causes gastrointestinal symptoms somewhat similar to those seen in Wakefield's subjects.

The good news, the researchers say, is that studies show that dietary restrictions can ameliorate some autistic symptoms. This, they say, "suggests a reversible element in this condition."

Dr. Wakefield, who is working on a treatment for affected children, will be speaking at our Defeat Autism Now! Conference in New Jersey, October 3 and 4, 1998

The report by Wakefield et al. was met with dismay by several researchers. Robert Chen and Frank De Stefano, also writing in *The Lancet*, questioned the findings, saying, "There is no report of detection of vaccine viruses in the bowel, brain, or any other tissue of the patients in Wakefield's report." Chen and De Stefano noted, further, that autism "was known well before MMR vaccine became available."

Researcher Edwin Cook commented, "there's nothing about this paper that tests whether MMR is related to autism, and I'm afraid that they try to make a circumstantial case." Cook argues, "It's more likely that there is a syndrome with a common age of onset and it is going to be associated in time with whatever is happening at that age."

In Britain, however, officials were sufficiently concerned by the new report to organize an investigation of the possible MMR/autism link, to be conducted by an independent panel set up by the Medical Research Council. In the meantime, Wakefield and colleagues are conducting virological studies which may help clarify the MMR vaccine's role, if any, in the gastrointestinal and behavioral symptoms seen in their subjects.

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"Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children," A. J. Wakefield, S. H. Murch, A. Anthony, J. Linnell, D. M. Casson, M. Malik, M. Berelowitz, A. P. Dhillon, M. A. Thomson, P. Harvey, A. Valentine, S. E. Davies, and J. A. Walker-Smith; *The Lancet*, Vol. 351, No. 9103, February 28, 1998, pp. 637-641. Address: Andrew J. Wakefield, Department of Medicine, Royal Free Hospital and School of Medicine, London NW3 2QG, UK.

—and—

"Measles vaccine's link with autism studied," Lian Murray, *London Times*, February 27, 1998.

—and—

"Report of new syndrome stirs controversy," Reuters News Service, February 27, 1998.

# New insights into causes of self-injury

The severe self-injury of many autistic and retarded individuals is horrifying but also intriguing. Currently there are two theories about why these individuals hit, bite, slap, or otherwise hurt themselves:

—The "pain theory" suggests that individuals who exhibit self-injurious behavior (SIB) have little sense of pain. According to this theory, naltrexone and similar drugs should reduce SIB because they heighten pain sensitivity by blocking the brain receptors for opioids, natural morphine-like substances produced by the body.

—The "addiction theory" suggests that self-injurious acts release a flood of opioids that provide a "fix" much like an addict's drugs. According to this theory, self-injurious individuals hurt themselves in order to achieve an opioid "high," and naltrexone works by eliminating this euphoria.

A new study by Curt Sandman and colleagues adds support to the second theory. Sandman et al. studied ten self-injurious retarded adults, measuring plasma levels of the opioid beta-endorphin immediately after episodes of self-injury. In addition, the researchers measured levels of the related peptide ACTH, which usually rises when beta-endorphin levels rise. Levels of these two substances were also measured during times when the subjects were not injuring themselves, and during both morning and afternoon hours (to account for normal daily fluctuations).

The researchers found that beta-endorphin levels rose immediately following an episode of self-injury, while ACTH levels did not. This finding is significant, they say, because of "the report of a remarkably similar dissociation between beta-endorphin and ACTH among heroin addicts in response to stress."

In another stage of this study, Sandman et al. gave naltrexone to the participants to determine the relationship between post-SIB beta-endorphin levels and the effectiveness of the drug. A significant majority of subjects responded positively to the drug, they say, and "patients with the largest increase in beta-endorphin after an episode of SIB had the greatest reduction in SIB when naltrexone was administered." This indicates, they say, that "circulating beta-endorphin predicts the central effects of opiate blocking agents in patients with SIB."

Sandman et al. say their findings are significant in light of biochemical similarities between their self-injurious subjects and heroin addicts.

The researchers conclude that the increased levels of beta-endorphin seen in their subjects following self-injury, in the absence of elevated ACTH, "may be consistent with the speculation that some patients engaged in self-injury to release beta-endorphin as an opioid 'fix'." If this is true, they say, "then these individuals may become 'addicted' to endogenous opioids, and SIB may be the self-destructive act to satisfy their addiction." The resistance of these behaviors to extinction, they say, is consistent with this theory.

Because the response of the subjects to naltrexone varied according to the degree of elevation of beta-endorphin, the researchers speculate that "patients who do not have elevated beta-endorphin after SIB are a different subgroup with different mechanisms controlling their behavior." This group, they say, may not respond to naltrexone treatment.

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"Dissociation of POMC peptides after self-injury predicts responses to centrally acting opiate blockers," Curt A. Sandman, William Hetrick, Derek V. Taylor, and Aleksandra Chicz-DeMet; *American Journal on Mental Retardation*, Vol. 102, No. 2, 1997, pp. 182-199. Address: Curt A. Sandman, Director of Research, 2501 Harbor Blvd., Costa Mesa, CA 92626.