

Shorts

briefed by Jule Klotter

Nasal Pathology in Patients with Chronic Airway Inflammation

A single acute exposure to dust, smoke, or chemicals such as acetic acid, ammonia, chlorine, ethylene oxide, and sulfur dioxide can cause an asthma-like condition known as reactive airways dysfunction syndrome (RADS). Bronchoscopic lung biopsies in patients with RADS show epithelial damage, chronic inflammation with lymphocytic infiltrates, and basement membrane (underlies the epithelium) thickening. Exposure to solvents and pesticides have caused a similar condition called reactive upper airways dysfunction syndrome (RUADS). A report in *Clinical Toxicology* by William J. Meggs, MD and colleagues at East Carolina University School of Medicine (Greenville, North Carolina) presents findings on the nasal pathology and ultrastructure of the nasal mucosa in 13 patients who developed RUADS after an occupational exposure to chlorine dioxide.

The researchers evaluated the patients five years after the chlorine dioxide exposure. They reviewed the patients' medical records and test results, had the patients fill out a standardized questionnaire concerning complaints of chemical sensitivities and lifestyle changes, and performed nasal examinations with a nasal telescope, and nasal biopsies. These results were compared to similar evaluations of three non-symptomatic volunteers. Nasal examination of the patients found abnormalities that included congestion, telangiectasia (dilation of capillaries producing an angioma, a usually benign tumor), paleness, cobblestoning, edema, and thick mucous. Nasal biopsies from the patients showed congestion and edema and mild to severe inflammation.

Nerve fibers were found in the biopsies of several patients; the greatest number of fibers appeared in the patient with severe inflammation. Electron micrographs from patients showed abnormal spaces between epithelial cells, while those from the normal patients did not. Detachment of respiratory epithelial cells from the basement membrane was also evident in some cases. Unlike patients with allergic rhinitis, the patients with RUADS showed an increase in lymphocytes and no increase in eosinophils.

Dr. Meggs and colleagues suggest that chronic airway disease may result from an interaction between the irritant chemical and sensory nerves, producing neurogenic inflammation (accounting for the presence of lymphocytes) as well as damage to the respiratory mucosa. Epithelial damage and the proliferation of nerve fibers may be the cause of sensitivity to low levels of chemical irritants.

"Nasal Pathology and Ultrastructure in Patients with Chronic Airway Inflammation (RADS and RUADS) Following an Irritant Exposure" by William J. Meggs, MD; Torik Elshelt, MD; W. James Metzger, MD; Marcus Alhernaz, MD; Richard M. Bloch, PhD. *Clinical Toxicology*, 34(4), 393-396 (1996).

PCBs and IQ

Although polychlorinated biphenyls (PCBs) have been banned in the U.S. since the 1970s, the chemicals are still found in soil and water, contaminating our food supply. Evidence indicates that PCBs contribute to impaired mental development in children. Joseph L. Jacobson and Sandra W. Jacobson studied 212 11-year-old children whose mothers had eaten fish from heavily-contaminated Lake Michigan. PCB levels in umbilical cord serum and in maternal serum and milk were measured at

birth for each child. (Disturbingly, the measured levels were only slightly higher than levels typically found in the general population.)

In an article published in the *New England Journal of Medicine* (Vol. 335, No. 11, September 12, 1996), the researchers reported that the children in the study were more likely to have low-average IQ scores than less-exposed children and to be at least two years behind in reading comprehension. Memory and the ability to focus and sustain attention were also impaired. In a *Science News* article by J. Raloff (Vol. 150, No. 11, September 14, 1996), Joseph Jacobson was quoted as saying: "I thought that once they reached a structured school environment, whatever minor handicaps [the children with high PCB exposures] had would be overcome. So I was quite surprised to find that, if anything, the effects were stronger and clearer at age 11 than they had been at age 4."

"Are banned PCBs still endangering mental health?" *CRIME Times*, Vol. 2, Number 4, 1996

Neurotoxicity and Fluoride

Dr. Phyllis Mullenix began studying the neurotoxicity of fluoride at the Forsyth Dental Center in 1987, at the request of the center's director, Dr. Jack Hein. Dr. Hein was the scientist responsible for adding monofluorophosphate (MFP) to toothpaste. When Dr. Mullenix and colleagues began investigating fluoride, they did not expect to find any problems. When their first rat experiment indicated that fluoride did indeed negatively affect brain function, they did not believe it. Instead of publishing their results, they repeated the study – more than once. They tried using more animals, different doses, sexes, ages and methods of administration.

In their 1995 paper titled "Neurotoxicity of sodium fluoride in rats" (*Neurotoxicol. Teratol.* 17: 169-177, 1995), they reported that rats exposed to fluoride as adults exhibited cognitive deficits, and rats exposed to fluoride prenatally showed hyperactivity. "Overall, we concluded that the rat study flagged potential for motor dysfunction, IQ deficits and/or learning disabilities in humans," Dr. Mullenix wrote in a letter to a US Army contractor, BSA Environmental Services.

Even though the fluoride levels that were added to the rats' drinking followed an animal model accepted by dental researchers, critics have claimed that the fluoride levels used in the experiment (75-125 ppm NaF) were too high, and therefore irrelevant. Dr. Mullenix explains that the doses they used produced plasma levels that equal the plasma levels found in humans who drink water containing 5-10 ppm fluoride. Children receiving topical applications of some dental fluoride gels have fluoride plasma levels that are 10 times greater, over an hour, than the rats.

Dr. Mullenix's research is by no means the only evidence of fluoride neurotoxicity. B. Spittle reviewed 60 years of case reports concerning neurological effects of fluoride ("Psychopharmacology of Fluoride: a review" *Int. Clin. Psychopharm.* 9:79-82, 1994). Dr. Mullenix says that common complaints in these case reports include impaired memory and concentration, lethargy, headache, depression, and confusion. A 1998 study by J.A. Varner and colleagues found that rats whose drinking water contained fluoride (1 ppm) had compromised blood brain barriers that permitted more