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Central Nervous System Damage from Fluorides
Phyllis J. Mullenix, Ph.D. September 14, 1998

It was 1982 when fluoride was first brought to my attention as a substance in need of investigation. At that time, I was in the Departments of Psychiatry at Boston's Children's Hospital and Neuropathology at the Harvard Medical School. My studies focused on detection procedures for neurotoxicity, and they typically considered a variety of environmental and therapeutic agents, i.e., radiation, lead, amphetamine, phenytoin, nitrous oxide.

Dr. John Hein, then Director of Forsyth's Dental Infirmary for Children in Boston, was interested in neurotoxicity studies and invited me to continue this research at Forsyth and to apply it to substances used in dentistry. Fluoride was prominent on his list.

Five years lapsed before our investigations of fluoride began. The delay was due to time spent on technological improvements, specifically development of a computer pattern recognition system for the objective quantification of behavior in an animal model. In early June of 1986, the Forsyth Dental Center was noted for this achievement in the Wall Street Journal and the Boston Herald, and applications of our research grew. The new technology enabled us to study the clinically recognized neurotoxicity associated with the treatment for childhood leukemia. Simultaneously, we started investigations of fluoride, the "safe and effective" treatment for dental caries.

Initially, the fluoride study sparked little interest, and in fact we were quite anxious to move on to something academically more exciting. Using an animal model developed for the study of dental fluorosis, we expected rats drinking fluoride-treated water would behave the same as matching controls. They did not. The scientific literature led us to believe that rats would easily tolerate 175 ppm fluoride in their drinking water. They did not. Reports in the literature indicated that fluoride would not cross the blood brain barrier. But it did. Prenatal exposure to fluoride was not supposed to permanently alter behavioral outcome. It did. Like walking into quicksand, our confidence that brain function was impervious to fluoride was sinking.

Our 1995 paper in Neurotoxicology and Teratology was the first laboratory study to demonstrate in vivo that central nervous system (CNS) function was vulnerable to fluoride, that the effects on behavior depended on the age at exposure and that fluoride accumulated in brain tissues. The behavioral changes common to weanling and adult exposures were different from those after prenatal exposure. Whereas prenatal exposure dispersed many behaviors as seen in drug-induced hyperactivity, weanling and adult exposures led to behavior-specific changes more related to cognitive deficits. Brain histology was not examined in this study, but we suggested that the effects on behavior were consistent with interrupted hippocampal development (a brain region generally linked with memory).

Establishing a threshold dose for effects on the CNS, in rats or humans, was not the intent of this initial investigation. Yet, one fact relevant to human exposure emerged quite clear. When rats consumed 75-125 ppm and humans 5-10 ppm fluoride in their respective drinking waters, the result was equivalent ranges of plasma fluoride levels. This range is observed with some treatments for osteoporosis, and it is exceeded ten times over, one hour after children receive topical applications of some dental fluoride gels. Thus, humans are being exposed to levels of fluoride we know alters behavior in rats.

We concluded that the rat study flagged potential for motor dysfunction, IQ deficits and/or learning disabilities in humans. Confident as we were, the data were only one piece of the puzzle, the overall picture was still emerging. Soon thereafter we learned of two epidemiological studies (Fluoride, 1995-1996) from China showing IQ deficits in children over-exposed to fluoride via drinking water or soot from burning coal. A recent review (International Clinical Psychopharmacology, 1994) listed case reports of CNS effects in humans excessively exposed to fluoride, information that spans almost 60 years. A common theme appeared in the reported effects: impaired memory and concentration, lethargy, headache, depression and confusion. The same theme was echoed in once classified reports about workers from the Manhatten Project. In all, our rat data seemed to fit a consistent picture.

1) A recent study in Brain Research demonstrated that chronic exposure to fluoride in drinking water of rats compromised neuronal (hippocampal) and cerebrovascular integrity (blood brain barrier) and increased aluminum concentrations in brain tissues.

2) Masters and Coplan have reported (International Journal of Environmental Studies, in press) that silicofluorides in fluoridated drinking water increased levels of lead in children's blood, a risk factor that predicts higher crime rates, ADD and learning disabilities.

3) Luke at the International Society for Fluoride Research (ISFR) meeting in August reported that fluoride accumulated in the human pineal gland, as much or more so than in bones and teeth, and the pineal gland's melatonin biosynthesis pathway is affected by fluoride.

4) Also at the ISFR meeting, I reported that the fluorinated steroid (dexamethasone) disrupts behavior in rats to a greater degree than does the nonfluorinated steroid (prednisolone). This finding matched results just completed in a study of children receiving steroids as a part of their treatment for childhood leukemia. Dexamethasone, compared to prednisolone, further reduced IQ, specifically impairing reading comprehension, arithmetic calculation and short-term working memory.

Exposure to fluoride goes well beyond that in our drinking water, toothpastes and mouth rinses. Fluoridation of water dictates that it is in food and processed beverages. Pesticides such as cryolite also increase fluoride content of foods. The trend toward fluorinating pharmaceuticals increases fluoride exposure via medication. Fluoride, in various compounds, plays a heavy role in occupational exposures and for people living in close proximity to industry, i.e., aluminum, steel, brick, glass, petroleum, etc. With exposure so common, we can no longer afford to ignore potential CNS consequences of fluoride.

I would be happy to answer questions about any of the above material.

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