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WHY EPA'S HEADQUARTERS UNION OF SCIENTISTS OPPOSES FLUORIDATION

The following documents why our union, formerly National Federation of Federal Employees Local 2050 and since April 1998 Chapter 280 of the National Treasury Employees Union, took the stand it did opposing fluoridation of drinking water supplies. Our union is comprised of and represents the approximately 1500 scientists, lawyers, engineers and other professional employees at EPA Headquarters here in Washington, D.C.

The union first became interested in this issue rather by accident. Like most Americans, including many physicians and dentists, most of our members had thought that fluoride's only effects were beneficial - reductions in tooth decay, etc. We too believed assurances of safety and effectiveness of water fluoridation .

Then, as EPA was engaged in revising its drinking water standard for fluoride in 1985, an employee came to the union with a complaint: he said he was being forced to write into the regulation a statement to the effect that EPA thought it was alright for children to have "funky" teeth. It was OK, EPA said, because it considered that condition to be only a cosmetic effect, not an adverse health effect. The reason for this EPA position was that it was under political pressure to set its health-based standard for fluoride at 4 mg/liter. At that level, EPA knew that a significant number of children develop moderate to severe dental fluorosis, but since it had deemed the effect as only cosmetic, EPA didn't have to set its health-based standard at a lower level to prevent it.

We tried to settle this ethics issue quietly, within the family, but EPA was unable or unwilling to resist external political pressure, and we took the fight public with a union amicus curiae brief in a lawsuit filed against EPA by a public interest group. The union has published on this initial involvement period in detail.\1

Since then our opposition to drinking water fluoridation has grown, based on the scientific literature documenting the increasingly out-of-control exposures to fluoride, the lack of benefit to dental health from ingestion of fluoride and the hazards to human health from such ingestion. These hazards include acute toxic hazard, such as to people with impaired kidney function, as well as chronic toxic hazards of gene mutations, cancer, reproductive effects, neurotoxicity, bone pathology and dental fluorosis. First, a review of recent neurotoxicity research results.

In 1995, Mullenix and co-workers \2 showed that rats given fluoride in drinking water at levels that

give rise to plasma fluoride concentrations in the range seen in humans suffer neurotoxic effects that vary according to when the rats were given the fluoride - as adult animals, as young animals, or through the placenta before birth. Those exposed before birth were born hyperactive and remained so throughout their lives. Those exposed as young or adult animals displayed depressed activity. Then in 1998, Guan and co-workers \3 gave doses similar to those used by the Mullenix research group to try to understand the mechanism(s) underlying the effects seen by the Mullenix group. Guan's group found that several key chemicals in the brain - those that form the membrane of brain cells - were substantially depleted in rats given fluoride, as compared to those who did not get fluoride.

Another 1998 publication by Varner, Jensen and others \4 reported on the brain- and kidney damaging effects in rats that were given fluoride in drinking water at the same level deemed "optimal" by pro-fluoridation groups, namely 1 part per million (1 ppm). Even more pronounced damage was seen in animals that got the fluoride in conjunction with aluminum. These results are especially disturbing because of the low dose level of fluoride that shows the toxic effect in rats - rats are more resistant to fluoride than humans. This latter statement is based on Mullenix's finding that it takes substantially more fluoride in the drinking water of rats than of humans to reach the same fluoride level in plasma. It is the level in plasma that determines how much fluoride is "seen" by particular tissues in the body. So when rats get 1 ppm in drinking water, their brains and kidneys are exposed to much less fluoride than humans getting 1 ppm, yet they are experiencing toxic effects. Thus we are compelled to consider the likelihood that humans are experiencing damage to their brains and kidneys at the "optimal" level of 1 ppm.

In support of this concern are results from two epidemiology studies from China\5,\6 that show decreases in I.Q. in children who get more fluoride than the control groups of children in each study. These decreases are about 5 to 10 I.Q. points in children aged 8 to 13 years.

Another troubling brain effect has recently surfaced: fluoride's interference with the function of the brain's pineal gland. The pineal gland produces melatonin which, among other roles, mediates the body's internal clock, doing such things as governing the onset of puberty. Jennifer Luke\7 has shown that fluoride accumulates in the pineal gland and inhibits its production of melatonin. She showed in test animals that this inhibition causes an earlier onset of sexual maturity, an effect reported in humans as well in 1956, as part of the Kingston/Newburgh study, which is discussed below. In fluoridated Newburgh, young girls experienced earlier onset of menstruation (on average, by six months) than girls in non-fluoridated Kingston \8.

From a risk assessment perspective, all these brain effect data are particularly compelling and disturbing because they are convergent.

We looked at the cancer data with alarm as well. There are epidemiology studies that are convergent with whole-animal and single-cell studies (dealing with the cancer hazard), just as the neurotoxicity research just mentioned all points in the same direction. EPA fired the Office of Drinking Water's chief toxicologist, Dr. William Marcus, who also was our local union's treasurer at the time, for refusing to remain silent on the cancer risk issue\9 . The judge who heard the lawsuit he brought against EPA over the firing made that finding - that EPA fired him over his fluoride work and not for the phony reason put forward by EPA management at his dismissal. Dr. Marcus won his lawsuit and is again at work at EPA. Documentation is available on request.

The type of cancer of particular concern with fluoride, although not the only type, is osteosarcoma, especially in males. The National Toxicology Program conducted a two-year study \10 in which rats and mice were given sodium fluoride in drinking water. The positive result of that study (in which malignancies in tissues other than bone were also observed), particularly in male rats, is

convergent with a host of data from tests showing fluoride's ability to cause mutations (a principal "trigger" mechanism for inducing a cell to become cancerous) e.g.\11a, b, c, d and data showing increases in osteosarcomas in young men in New Jersey \12 , Washington and Iowa \13 based on their drinking fluoridated water. It was his analysis, repeated statements about all these and other incriminating cancer data, and his requests for an independent, unbiased evaluation of them that got Dr. Marcus fired.

Bone pathology other than cancer is a concern as well. An excellent review of this issue was published by Diesendorf et al. in 1997 \14. Five epidemiology studies have shown a higher rate of hip fractures in fluoridated vs. non-fluoridated communities. \15a, b, c, d, e. Crippling skeletal fluorosis was the endpoint used by EPA to set its primary drinking water standard in 1986, and the ethical deficiencies in that standard setting process prompted our union to join the Natural Resources Defense Council in opposing the standard in court, as mentioned above.

Regarding the effectiveness of fluoride in reducing dental cavities, there has not been any double-blind study of fluoride's effectiveness as a caries preventative. There have been many, many small scale, selective publications on this issue that proponents cite to justify fluoridation, but the largest and most comprehensive study, one done by dentists trained by the National Institute of Dental Research, on over 39,000 school children aged 5-17 years, shows no significant differences (in terms of decayed, missing and filled teeth) among caries incidences in fluoridated, non-fluoridated and partially fluoridated communities.\16. The latest publication \17 on the fifty-year fluoridation experiment in two New York cities, Newburgh and Kingston, shows the same thing. The only significant difference in dental health between the two communities as a whole is that fluoridated Newburgh, N.Y. shows about twice the incidence of dental fluorosis (the first, visible sign of fluoride chronic toxicity) as seen in non-fluoridated Kingston.

John Colquhoun's publication on this point of efficacy is especially important\18. Dr. Colquhoun was Principal Dental Officer for Auckland, the largest city in New Zealand, and a staunch supporter of fluoridation - until he was given the task of looking at the world-wide data on fluoridation's effectiveness in preventing cavities. The paper is titled, "Why I changed My Mind About Water Fluoridation." In it Colquhoun provides details on how data were manipulated to support fluoridation in English speaking countries, especially the U.S. and New Zealand. This paper explains why an ethical public health professional was compelled to do a 180 degree turn on fluoridation.

Further on the point of the tide turning against drinking water fluoridation, statements are now coming from other dentists in the pro-fluoride camp who are starting to warn that topical fluoride (e.g. fluoride in tooth paste) is the only significantly beneficial way in which that substance affects dental health \19, \20, \21. However, if the concentrations of fluoride in the oral cavity are sufficient to inhibit bacterial enzymes and cause other bacteriostatic effects, then those concentrations are also capable of producing adverse effects in mammalian tissue, which likewise relies on enzyme systems. This statement is based not only on common sense, but also on results of mutation studies which show that fluoride can cause gene mutations in mammalian and lower order tissues at fluoride concentrations estimated to be present in the mouth from fluoridated tooth paste\22. Further, there were tumors of the oral cavity seen in the NTP cancer study mentioned above, further strengthening concern over the toxicity of topically applied fluoride.

In any event, a person can choose whether to use fluoridated tooth paste or not (although finding non-fluoridated kinds is getting harder and harder), but one cannot avoid fluoride when it is put into the public water supplies.

So, in addition to our concern over the toxicity of fluoride, we note the uncontrolled - and

apparently uncontrollable - exposures to fluoride that are occurring nationwide via drinking water, processed foods, fluoride pesticide residues and dental care products. A recent report in the lay media\23, that, according to the Centers for Disease Control, at least 22 percent of America's children now have dental fluorosis, is just one indication of this uncontrolled, excess exposure. The finding of nearly 12 percent incidence of dental fluorosis among children in un-fluoridated Kingston New York\17 is another. For governmental and other organizations to continue to push for more exposure in the face of current levels of over-exposure coupled with an increasing crescendo of adverse toxicity findings is irrational and irresponsible at best.

Thus, we took the stand that a policy which makes the public water supply a vehicle for disseminating this toxic and prophylactically useless (via ingestion, at any rate) substance is wrong.

We have also taken a direct step to protect the employees we represent from the risks of drinking fluoridated water. We applied EPA's risk control methodology, the Reference Dose, to the recent neurotoxicity data. The Reference Dose is the daily dose, expressed in milligrams of chemical per kilogram of body weight, that a person can receive over the long term with reasonable assurance of safety from adverse effects. Application of this methodology to the Varner et al.\4 data leads to a Reference Dose for fluoride of 0.000007 mg/kg-day. Persons who drink about one quart of fluoridated water from the public drinking water supply of the District of Columbia while at work receive about 0.01mg/kg-day from that source alone. This amount of fluoride is more than 100 times the Reference Dose. On the basis of these results the union filed a grievance, asking that EPA provide un-fluoridated drinking water to its employees.

The implication for the general public of these calculations is clear. Recent, peer-reviewed toxicity data, when applied to EPA's standard method for controlling risks from toxic chemicals, require an immediate halt to the use of the nation's drinking water reservoirs as disposal sites for the toxic waste of the phosphate fertilizer industry\24.

This document was prepared on behalf of the National Treasury Employees Union Chapter 280 by Chapter Senior Vice-President J. William Hirzy, Ph.D. For more information please call Dr. Hirzy at 202-260-4683. His E-mail address is <hirzy.john@epa.gov>

END NOTE LITERATURE CITATIONS

1.Applying the NAEP code of ethics to the Environmental Protection Agency and the fluoride in drinking water standard. Carton, R.J. and Hirzy, J.W. Proceedings of the 23rd Ann. Conf. of the National Association of Environmental Professionals. 20-24 June, 1998. GEN 51-61. On-line at URL http//:www.rvi.net/~fluoride/naep.cfm

2.Neurotoxicity of sodium fluoride in rats. Mullenix, P.J., Denbesten, P.K., Schunior, A. and Kernan, W.J. Neurotoxicol. Teratol. 17 169-177 (1995)

3. Influence of chronic fluorosis on membrane lipids in rat brain. Z.Z. Guan, Y.N. Wang, K.Q. Xiao, D.Y. Dai, Y.H. Chen, J.L. Liu, P. Sindelar and G. Dallner, Neurotoxicology and Teratology 20 537-542 (1998).

4. Chronic administration of aluminum- fluoride or sodium-fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity. Varner, J.A., Jensen, K.F., Horvath, W. And Isaacson, R.L. Brain Research 784 284-298 (1998).

5. Effect of high fluoride water supply on children's intelligence. Zhao, L.B., Liang, G.H., Zhang, D.N., and Wu, X.R. Fluoride 29 190-192 (1996)

6.. Effect of fluoride exposure on intelligence in children. Li, X.S., Zhi, J.L., and Gao, R.O. Fluoride 28 (1995).

7. Effect of fluoride on the physiology of the pineal gland. Luke, J.A. Caries Research 28 204 (1994).

8. Newburgh-Kingston caries-fluorine study XIII. Pediatric findings after ten years. Schlesinger, E.R., Overton, D.E., Chase, H.C., and Cantwell, K.T. JADA 52 296-306 (1956).

9. Memorandum dated May 1, 1990. Subject: Fluoride Conference to Review the NTP Draft Fluoride Report; From: Wm. L. Marcus, Senior Science Advisor ODW; To: Alan B. Hais, Acting Director Criteria & Standards Division ODW.

10. Toxicology and carcinogenesis studies of sodium fluoride in F344/N rats and B6C3F1 mice. NTP Report No. 393 (1991).

11a. Chromosome aberrations, sister chromatid exchanges, unscheduled DNA synthesis and morphological neoplastic transformation in Syrian hamster embryo cells. Tsutsui et al. Cancer Research 44 938-941 (1984).

11b. Cytotoxicity, chromosome aberrations and unscheduled DNA synthesis in cultured human diploid fibroblasts. Tsutsui et al. Mutation Research 139 193-198 (1984).

11c. Positive mouse lymphoma assay with and without S-9 activation; positive sister chromatid exchange in Chinese hamster ovary cells with and without S-9 activation; positive chromosome aberration without S-9 activation. Toxicology and carcinogenesis studies of sodium fluoride in F344/N rats and B6C3F1 mice. NTP Report No. 393 (1991).

11d. An increase in the number of Down's syndrome babies born to younger mothers in cities following fluoridation. Science and Public Policy 12 36-46 (1985).

12. A brief report on the association of drinking water fluoridation and the incidence of osteosarcoma among young males. Cohn, P.D. New Jersey Department of Health (1992).

13. Surveillance, epidemiology and end results (SEER) program. National Cancer Institute in Review of fluoride benefits and risks. Department of Health and Human Services. F1-F7 (1991).

14. New evidence on fluoridation. Diesendorf, M., Colquhoun, J., Spittle, B.J., Everingham, D.N., and Clutterbuck, F.W. Australian and New Zealand J. Public Health. 21 187-190 (1997).

15a. Regional variation in the incidence of hip fracture: U.S. white women aged 65 years and older. Jacobsen, S.J., Goldberg, J., Miles, ,T.P. et al. JAMA 264 500-502 (1990)

15b. Hip fracture and fluoridation in Utah's elderly population. Danielson, C., Lyon, J.L., Egger, M., and Goodenough, G.K. JAMA 268 746-748 (1992).

15c. The association between water fluoridation and hip fracture among white women and men aged 65 years and older: a national ecological study. Jacobsen, S.J., Goldberg, J., Cooper, C. and Lockwood, S.A. Ann. Epidemiol.2 617-626 (1992).

15d. Fluorine concentration is drinking water and fractures in the elderly [letter]. Jacqmin-Gadda,

H., Commenges, D. and Dartigues, J.F. JAMA 273 775-776 (1995).

15e. Water fluoridation and hip fracture [letter]. Cooper, C., Wickham, C.A.C., Barker, D.J.R. and Jacobson, S.J. JAMA 266 513-514 (1991).

16. Water fluoridation and tooth decay: Results from the 1986-1987 national survey of U.S. school children. Yiamouyannis, J. Fluoride 23 55-67 (1990).

17. Recommendations for fluoride use in children. Kumar, J.V. and Green, E.L. New York State Dent. J. (1998) 40-47.

18. Why I changed my mind about water fluoridation. Colquhoun, J. Perspectives in Biol. And Medicine 41 1-16 (1997).

19. A re-examination of the pre-eruptive and post-eruptive mechanism of the anti-caries effects of fluoride: is there any anti-caries benefit from swallowing fluoride? Limeback, H. Community Dent. Oral Epidemiol. 27 62-71 (1999).

20. Fluoride supplements for young children: an analysis of the literature focussing on benefits and risks. Riordan, P.J. Community Dent. Oral Epidemiol. 27 72-83 (1999).

21. Prevention and reversal of dental caries: role of low level fluoride. Featherstone, J.D. Community Dent. Oral Epidemiol. 27 31-40 (1999).

22. Appendix H. Review of fluoride benefits and risks. Department of Health and Human Services. H1-H6 (1991).

23.Some young children get too much fluoride. Parker-Pope, T. Wall Street Journal Dec. 21, 1998.

24. Letter from Rebecca Hanmer, Deputy Assistant Administrator for Water, to Leslie Russell re: EPA view on use of by-product fluosilicic (sic) acid as low cost source of fluoride to water authorities. March 30, 1983.

OTHER CITATIONS (This short list does not include the entire literature on fluoride effects)

a. Exposure to high fluoride concentrations in drinking water is associated with decreased birth rates. Freni, S.C. J. Toxicol. Environ. Health 42 109-121 (1994)

b. Ameliorative effects of reduced food-borne fluoride on reproduction in silver foxes. Eckerlin, R.H., Maylin, G.A., Krook, L., and Carmichael, D.T. Cornell Vet. 78 75-91 (1988).

c. Milk production of cows fed fluoride contaminated commercial feed. Eckerlin, R.H., Maylin, G.A., and Krook, L. Cornell Vet. 76 403-404 (1986).

d. Maternal-fetal transfer of fluoride in pregnant women. Calders, R., Chavine, J., Fermanian, J., Tortrat, D., and Laurent, A.M. Biol. Neonate 54 263-269 (1988).

e. Effects of fluoride on screech owl reproduction: teratological evaluation, growth, and blood chemistry in hatchlings. Hoffman, D.J., Pattee, O.H., and Wiemeyer, S.N. Toxicol. Lett. 26 19-24 (1985).

f. Fluoride intoxication in dairy calves. Maylin, G.A., Eckerlin, R.H., and Krook, L. Cornell Vet. 77 84-98 (1987).

g. Fluoride inhibition of protein synthesis. Holland, R.I. Cell Biol. Int. Rep. 3 701-705 (1979).

h. An unexpectedly strong hydrogen bond: ab initio calculations and spectroscopic studies of amide-fluoride systems. Emsley, J., Jones, D.J., Miller, J.M., Overill, R.E. and Waddilove, R.A. J. Am. Chem. Soc. 103 24-28 (1981).

i. The effect of sodium fluoride on the growth and differentiation of human fetal osteoblasts. Song, X.D., Zhang, W.Z., Li, L.Y., Pang, Z.L., and Tan, Y.B. Fluoride 21 149-158 (1988).

j. Modulation of phosphoinositide hydrolysis by NaF and aluminum in rat cortical slices. Jope, R.S. J. Neurochem. 51 1731-1736 (1988).

k. The crystal structure of fluoride-inhibited cytochrome c peroxidase. Edwards, S.L., Poulos, T.L., Kraut, J. J. Biol. Chem. 259 12984-12988 (1984).

l. Intracellular fluoride alters the kinetic properties of calcium currents facilitating the investigation of synaptic events in hippocampal neurons. Kay, A.R., Miles, R., and Wong, R.K.S. J. Neurosci. 6 2915-2920 (1986).

m. Fluoride intoxication: a clinical-hygienic study with a review of the literature and some experimental investigations. Roholm, K. H.K. Lewis Ltd (London) (1937).
n. Toxin-induced blood vessel inclusions caused by the chronic administration of aluminum and sodium fluoride and their implications for dementia. Isaacson, R.L., Varner, J.A., and Jensen, K. F. Ann. N.Y. Acad. Sci. 825 152-166 (1997).

o. Allergy and hypersensitivity to fluoride. Spittle, B. Fluoride 26 267-273 (1993)