HANDOUT 1.4-A
10/11/16

October 11, 2016, SCVWD Meeting – Public Comment
Maureen Jones, Citizens for Safe Drinking Water, maureenj@pacbell.net

1. Letter, Nov. 2000. U.S. EPA to Roger Masters, Dartmouth College
   "To answer your first question on whether we have in our possession empirical scientific data on the effects of fluosilicic acid or sodium silicofluoride on health and behavior, our answer is no."
2. Letter, Mar. 2001. U.S. EPA to Roger Masters, Dartmouth College
3. In January, "several fluoride chemistry related research needs were identified."
4. Letter, Apr. 2002. U.S. EPA, Request for Assistance (RFA)
   Request for Assistance to investigate the reactions that take place when fluorosilicates are added to drinking water supplies.
5. 1937 *Comparative Toxicity of Inorganic Fluorides.*
   LD-50 shows Calcium Fluoride is 20 times less toxic for Guinea Pigs than any of the three fluoridation chemicals.
6. 1935 *Ohio Agricultural Bulletin 558.*
   "The toxicity of fluorine varied with the form of fluorine fed."
   "The availability of fluorine varied with the form of fluorine fed."
7. 1934 *Industrial and Engineering Chemistry*, U of Arizona.
   It took 5.4 per cent fluoride from calcium fluoride to cause death in animals in 9 to 11 days compared with 0.0004 percent from any of the fluosilicates to cause death in same length of time.
8. 2006 Review of U.S. NRC Report: *Fluoride in Drinking Water.*
   The 12-member committee of the NRC was instructed by EPA to:
   a. identify only health effects known with total certainty,
   b. not to identify a new MCLG,
   c. ***not to discuss silicofluorides***, used most for U.S. fluoridation.
9. 2014 *Internat. J of Occu and Environ Health. Vol 20 No. 2*
   Metal contaminants vary with each batch of fluorosilicic acid and create a regulatory blind spot that jeopardizes any safe use of fluoride additives.

Again, I request a copy of SCVWD's request(s) to their chemical supplier (or any potential supplier) and their responses, for any toxicological studies that show supplier is in compliance with ANSI/NSF Standard 60, General Requirements, Section 3.2.1.

Sincerely, Maureen Jones

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
NATIONAL RISK MANAGEMENT RESEARCH LABORATORY
CINCINNATI, OH 45268

November 16, 2000

OFFICE OF
RESEARCH AND DEVELOPMENT

Roger D. Masters
Research Professor of Government
Dartmouth College
Department of Government
6108 Silsby Hall
Hanover, New Hampshire 03755-3547

Dear Professor Masters:

We have received your letter dated September 27, 2000, requesting empirical scientific data we may have on the health effects of fluosilicic acid or sodium silicofluoride and manganese neurotoxicity.

To answer your first question on whether we have in our possession empirical scientific data on the effects of fluosilicic acid or sodium silicofluoride on health and behavior, our answer is no. Health effects research is primarily conducted by our National Health and Environmental Effects Research Laboratory (NHEERL). We have contacted our colleagues at NHEERL and they report that with the exception of some acute toxicity data, they were unable to find any information on the effects of silicofluorides on health and behavior.

In answer to your question on empirical information we may have on manganese neurotoxicity, NHEERL scientists forwarded to us several manuscripts with reference sections that contain information on the neurotoxicity of manganese. These are enclosed for your information.

I apologize for the delay in responding to your request and hope you find the enclosed information useful.

Sincerely,

Robert C. Thurnau

Robert C. Thurnau, Chief
Treatment Technology Evaluation Branch
Water Supply and Water Resources Division

Enclosures

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
NATIONAL RISK MANAGEMENT RESEARCH LABORATORY
CINCINNATI, OH 45268

March 15, 2001

OFFICE OF
RESEARCH AND DEVELOPMENT

Roger D. Masters, Ph.D
Research Professor of Government
Dartmouth College, 6108 Silsby Hall
Hanover, New Hampshire 03755-3547

Dear Dr. Masters:

Thank you for your letter dated December 19, 2000. I apologize for the delay in my response. To formulate a reply to you, it was necessary to coordinate with our program office in Washington.

A recent communication to you (November 29, 2000) from Jeanette Wiltse, Director, Health and Ecological Division indicated that the U.S. Environmental Protection Agency (EPA) endorsed the use of fluosilicate compounds for fluoridation of public drinking water supplies. Supporting the endorsement is the ANSI/NSF Standard 60, Drinking Water Chemicals – Health Effects. This is the official agency position on this matter.

In January, representatives from the Office of Research and Development (ORD) and the Offices of Science and Technology and Ground Water and Drinking Water met to discuss a number of water related issues including Fluoridation. Several fluoride chemistry related research needs were identified including; (1) accurate and precise values for the stability constants of mixed fluorohydroxo complexes with aluminum (III), iron (III) and other metal cations likely to be found under drinking water conditions and (2) a kinetic model for the dissociation and hydroloysis of fluosilicates and stepwise equilibrium constants for the partial hydrolysis products.

As a result of these discussions, ORD is exploring options to initiate research in the identified research areas.

If we have additional information on this issue, we will forward it to you.

Sincerely,

Robert C. Thurnau for

Sally C. Gutierrez
Director
Water Supply and Water Resources
Division

REQUEST FOR ASSISTANCE
Measurement of Fluorosilicates in Drinking Water

2002 over

1.0. INTRODUCTION

The improvement of the quality of the nation's drinking water is an important goal and research to extend the boundaries of knowledge in this area is a function that government serves well. The Water Supply and Water Resources Division (WSWRD) of the National Risk Management Research Laboratory (NRMRL) is involved in a wide range of efforts, one of which is the review and advancement of the science on regulated contaminants. Much of the nation's drinking water is fluoridated, and fluoride is regulated by the EPA at a maximum contaminant level of 4.0 mg L^{-1}. The division has completed a review of the scientific literature and has identified certain areas for which additional information is desired. The division seeks to promote the public welfare by researching basic liquid aqueous phase solution chemistry of regulated contaminants and soliciting competent researchers capable of completing research projects that fill identified gaps in the scientific literature. To that end, the division seeks to fund a proposal on the measurement of fluorosilicate species in drinking water.

2.0. RESEARCH OBJECTIVES

2.1. Background

Hexafluorosilicic acid (H_2SiF_6) and sodium hexafluorosilicate (Na_2SiF_6) are the most commonly used fluoridating agents by potable water systems in the U.S. These species dissociate and hydrolyze to produce fluoride anion (F^-). The release of fluoride proceeds through a complex, multi-step equilibrium process that is not well-understood. A variety of models have been proposed, and the speciation remains a matter of debate as does the existence of some fluorosilicates. A review of the relevant chemical literature detailing the complexities, disagreement, and scientific facts has been prepared by the EPA. This review is available to prospective applicants, and they are encouraged to request a copy prior to preparing a proposal.

In addition to the silicon(IV) present from the fluoridating agent, many natural water supplies contain soluble oxo- and hydroxosilicates, which further complicates the speciation. The EPA seeks information on the utility of techniques and methods for monitoring the species formed during the dissociation and hydrolysis of hexafluorosilicate as well as those species present once equilibrium is achieved. These data are expected to aid in the development of pharmacokinetic and toxicokinetic studies and to further the understanding of the fate of fluoride, including its interactions with other species in drinking water. As such, the results of this study will be of use to state agencies, water utilities, and other governmental or scientific bodies who seek to ensure the quality of the nation's drinking water supplies.

2.2. Objective

The primary objective of this RFA is to investigate the reactions that take place when fluorosilicates are added to drinking water supplies and what concentrations of which fluorosilicate species may monitored in finished drinking water supplies and what techniques may be used for such monitoring. A secondary objective of this RFA is to explore what spectroscopic or other techniques are most amenable to determination of equilibrium constants for fluorosilicate systems, which engage in multiple, simultaneous, and complex equilibria. A tertiary objective is to consider what techniques might be applied to kinetic and mechanistic studies of the dissociation and hydrolysis of hexafluorosilicate. Conditions in finished drinking water include total fluoride concentrations on the order of 20 μM and total silicon(IV) concentrations on the order of 300 μM. Collaboration by skilled experimentalists with expertise in inorganic chemistry and the analytical techniques is encouraged.

REQUEST FOR ASSISTANCE (RFA)

MEASUREMENT OF FLUOROSILICATES IN DRINKING WATER

Announcement date: April 25, 2002

An offeror must submit the application (original plus four copies) so as to be received by The application should be addressed as follows:

Edward T. Urbansky
U.S. Environmental Protection Agency
National Risk Management Research Laboratory
Water Supply and Water Resources Division
26 West Martin Luther King Drive, MS 681
Cincinnati, Ohio 45268

An application received after the above time and date will not be considered unless there is clear evidence that the application was mishandled by EPA after its timely receipt.

Questions regarding this RFA should be directed by electronic mail to **urbansky.edward@epa.gov**

Applicants will be notified by letter as to the disposition of their preapplication (accepted/not accepted) and informed of the identity of the successful proposal.

Proprietary Information: In accordance with 40 CFR 40.150, applications considered relevant to EPA research objectives will be viewed for technical merit by at least one reviewer within the EPA and at least two reviewers outside of the EPA. Therefore, proposals submitted in response to this competitive solicitation will not be considered if the applicant asserts a claim of confidentiality for technical information contained therein.

Table 7-1

Comparative Toxicity of Inorganic Fluorides[6]

Extremely Toxic	
Hydrogen fluoride (anhydrous)	HF
Silicon tetrafluoride	SiF_4
Hydrofluoric acid (aqueous)	HF
Hydrofluorosilicic acid	H_2SiF_6
Very Toxic	
Easily soluble fluorides and fluorosilicates	
Sodium fluoride	NaF
Potassium fluoride	KF
Ammonium fluoride	NH_4F
Sodium fluorosilicate	Na_2SiF_6
Potassium fluorosilicate	K_2SiF_6
Ammonium fluorosilicate	$(NH_4)_2SiF_6$
Moderately Toxic	
Poorly soluble (almost insoluble) fluorides	
Cryolite	Na_3AlF_6
Calcium fluoride	CaF_2

Table 7-2

Lethal Dose of Fluorides in Adult Guinea Pigs[7]

Compound	*Oral (mg/kg)*	*Subcutaneous (mg/kg)*
NaF	250	400
CaF_2	>5,000	>5,000
AlF_3	600	3,000
HF (aqueous)	80	100
H_2SiF_6	200	250
Na_2SiF_6	250	500
$Al_2(SiF_6)_3$	5,000	4,000

Table 7-2 shows the acute toxicity of the most important fluoride salts to guinea pigs, expressed in milligrams per kilogram of body weight. It also demonstrates the difference in the lethal dose following oral and subcutaneous administration.

7. Simonin, P., and Pierron A.: Toxicité brute des derivés fluorés. C.R.Séances Soc. Biol. Fil., 124:133-134, 1937.

In: Waldbott GL, M.D., AW Burgstahler, Ph.D., HL McKinney, Ph.D. Fluoridation: The Great Dilemma. Lawrence Kansas: Coronado Press, Inc., 1978.

COMPTES RENDUS HEBDOMADAIRES

DES SÉANCES ET MÉMOIRES

DE LA

SOCIÉTÉ DE BIOLOGIE

ET DE SES FILIALES ET ASSOCIÉES :

LES SOCIÉTÉS DE BIOLOGIE D'ALGER, DE BORDEAUX, LILLE, LYON, MARSEILLE, NANCY, STRASBOURG, ATHÈNES, BARCELONE, BELGRADE, MONTEVIDEO, MONTRÉAL;
LES SOCIÉTÉS DE BIOLOGIE ARGENTINE (BUENOS-AIRES, CORDOBA, ROSARIO), BELGE, BRÉSILIENNE (RIO DE JANEIRO, SAO PAULO) CHILIENNE (CONCEPCION, SANTIAGO), DANOISE, MEXICAINE, POLONAISE (LWOW, VARSOVIE, POZNAN), PORTUGAISE (LISBONNE, PORTO, COIMBRE), ROUMAINE (BUCAREST, CLUJ, JASSY), TCHÉCOSLOVAQUE, DE SUÈDE ET DE LETTONIE;
LA SOCIÉTÉ FRANCO-JAPONAISE DE BIOLOGIE.

(89e année)

ANNÉE 1937 - TOME I

(CENT VINGT-QUATRIÈME TOME DE LA COLLECTION)

PARIS

MASSON ET Cie, EDITEURS

LIBRAIRES DE L'ACADÉMIE DE MÉDECINE

120, BOULEVARD SAINT-GERMAIN (6e)

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1937

Kick, CH et al. "Fluorine in Animal Nutrition". Ohio Agricultural Experiment Station, Wooster, Ohio. *Bulletin 558.* November 1935.

Summary: Pages 73 & 74.

1. **The toxicity of fluorine varied with the form of fluorine fed.** Sodium fluoride was much more toxic to pigs, rats, and chicks than calcium fluoride when these two salts were fed at comparable fluorine levels. Rock phosphate, phosphatic limestone, and treble superphosphate occupied intermediate positions in this respect.

2. Rations containing excessive amounts of available fluorine reduced the growth and the feed consumption of pigs and chicks and definitely increased the feed requirement per unit of gain for the pig.

3. When the rations of pigs contained more than 0.029 per cent of fluorine as sodium fluoride or more than 0.033 per cent as rock phosphate, the bones were characterized by increased thickness, loss of normal color and luster, presence of exostoses, and a decreased breaking strength.

4. The weakened, thickened bones resulting from fluorine feeding contained normal percentages of ash, calcium, and phosphorus, increased amounts of magnesium and fluorine, and decreased percentages of carbonates. These changes were directly correlated with increased amounts of fluorine in the ration.

5. The inclusion of fluorine in the ration of the pig increased the thickness of the walls of the femurs but not of the mandibles. The increased thickness of the mandibles was due to an increase in the size of the medullary spaces. A change also occurred in the type of marrow present.

6. High-fluorine rations increased the width of the dental arch in pigs but not in rats.

7. The inclusion of excessive amounts of fluorine in the rations of pigs and rats caused hypoplasia of the enamel of the teeth. In pigs, the feeding of such rations over long periods of time caused the teeth to become so soft that they were worn down until in some cases the pulp cavities were exposed. The incisors of the rat became white in color and some were elongated with the occluding incisor worn down or broken off. These changes were accompanies by hypoplasia of the enamel. The dentin was similarly affected to a less degree.

8. The percentages of ash, calcium, phosphorus, magnesium, and carbon dioxide in the teeth were unaffected by the fluorine content of the ration but the percentage of fluorine was increased in direct proportion to the amount of this element present in the ration.

9. High levels of fluorine in the ration exerted no direct effect upon reproduction in rats or pigs but adversely affected lactation through decreased feed consumption.

10. Rations containing large amounts of fluorine caused an increased water consumption and a diuresis in pigs.

11. Sodium fluoride, calcium fluoride, and phosphatic limestone had no evident effect on the livers, kidneys, spleens, thyroids, or parathyroids of rats or on the livers or kidneys of pigs.

12. the addition of 1 per cent or more of rock phosphate to the ration of pigs caused a degeneration of the epithelium of the convoluted tabules and a fibrosis of the kidney. This did not occur in the case of the rat.

13. The feeding of sodium fluoride at levels of 0.05 per cent of the ration caused a retardation in the rate of growth of the rat but had no effect on the percentage of bone ash at maturity.

14. When fluorine as rock phosphate or sodium fluoride was fed at levels of 0.071 per cent, a retardation occurred in the calcification of the bones of rats at 5 and 10 weeks of age.

15. **The availability of fluorine varied with the form in which the fluorine was fed.** Approximately 30 per cent of the fluorine ingested in the form of rock phosphate, sodium flusilicate and sodium fluoride was retained in the body by the rat while none of the fluorine in the form of calcium fluoride was retained.

16. The effect of fluorine feeding on blood coagulability varied with the species. High-fluorine rations increased the rate of coagulation in the case of the chick and decreased it in the case of the rat.

17. For practical feeding purposes, rock phosphate may be fed to pigs at 0.5 per cent of the ration and to chicks at 2 per cent of the ration for short periods without danger of fluorine toxicity. If the animals are to be maintained on the ration for long periods of time (12 months or longer), the use of smaller amounts is recommended.

Bold added - MJ

Industrial
AND ENGINEERING
Chemistry

VOLUME 26
NUMBER 7

JULY
1934

HARRISON E. HOWE, EDITOR

Comparative Toxicity of Fluorine Compounds

MARGARET CAMMACK SMITH AND RUTH M. LEVERTON, University of Arizona, Tucson, Ariz.

The use of drinking water containing fluorine in concentrations of one part per million or more is recognized as the cause of mottled enamel, a defect of human teeth (12, 13, 15). The increasing use of fluorine compounds as spray insecticides has prompted this study of the comparative toxicity of different compounds of fluorine. The following compounds of fluorine are used: sodium, potassium, ammonium, and calcium fluorides; sodium, potassium, and barium fluosilicates; and natural cryolite (sodium aluminum fluoride). A comparison is made of their effect upon growth rate, food consumption, efficiency of utilization of food, reproduction, mortality, and teeth, when supplied to young albino rats at the same fluorine concentration. Wide differences in toxicity among these compounds are found when the effect upon growth, food utilization, and damage to the teeth are considered, which may or may not be a reflection of difference in their solubility. From the standpoint of initial damage to the teeth, however, all these compounds of fluorine are found to be equally toxic. Fourteen parts per million of fluorine (from any source) in the diet of the rats leaves a mark upon the rat incisors. The significance of these findings in relation to the spray residue problem and human mottled enamel is discussed.

REPORTS in the literature of the toxic effects of fluorides have been reviewed by McClure and Mitchell (*7*) and DeEds (*4*). Such indications of impaired health and development as subnormal rate of growth or decline in weight, poor appetite and lowered food intake, poor reproduction, and inferior bone and tooth structure have been reported to result from relatively low intakes of sodium fluoride. High levels of feeding have caused death. The harmful effects resulting from the use of rock phosphates as mineral supplements in farm animal rations have been believed to be due to their fluorine content (*1, 5, 10, 18, 19*).

In 1931 proof was established in this laboratory (*15*) that the dental defect of human teeth known as mottled enamel (Figure 1) which is endemic in many sections of the world is caused by the toxic action of fluorides present in the drinking water supply of the afflicted persons. An extensive survey of the conditions in Arizona (*18*) by means of which the concentration of fluorides in water supplies was correlated with the presence or absence of mottled enamel and its degree of severity if present, gave evidence that the use of drinking water having a fluorine concentration above 2.7[1] parts per million interfered with normal tooth development. The drinking of water in the usual amount which contains as little as 5[1] p. p. m. of fluorine has been found to cause a severe type of mottled enamel of the teeth, the enamel being pitted and corroded. Mottled teeth are not only disfiguring in appearance but are so defective in structure and strength that they often have to be replaced by false teeth at an early age.

Fluorine compounds are becoming more and more commonly used as spray insecticides in place of arsenicals because the toxic action of arsenicals is known and feared. It would appear that from the standpoint of public health, chronic intoxication from fluorides is little to be desired.

Sollmann, Schettler, and Wetzel (*17*) in 1921 studied the effect of fluorine intake upon the growth and food intake of albino rats. They varied the percentage of pure sodium fluoride in the ration from 0.0002 to 0.23 per cent for a 1- to 4-month period. A concentration of sodium fluoride greater than 10 mg. per kg. of rat per day, roughly 0.015 per cent of the ration, diminished food consumption and growth even though the food was equally palatable to the animals. Below this level no harmful effect of the fluoride was noted. In these studies, however, only growth and food consumption were used as criteria.

In 1925 Schultz and Lamb (*11*) reported briefly that a level of 0.1 per cent sodium fluoride in the ration interfered with growth rate of rats and that an "unfavorable effect in reproduction begins at a level of about 0.025 per cent sodium fluoride." In the same year McCollum, Simmonds, and Becker (*8*) reported that, when sodium fluoride was added to an adequate mixed ration at the level of 0.05 per cent, the teeth of rats were observed to be abnormal in appearance and defective in structure.

In this laboratory (*14*) the feeding of sodium fluoride to rats at 0.025, 0.05, and 0.1 per cent of the ration has in each case interfered with the normal calcification of the teeth to an extent varying with the concentration. Even the lowest level of feeding (0.025 per cent) resulted in defective enamel of a rather severe type of the teeth of rats, dogs, and guinea pigs. It has been observed also that an effect upon the teeth of the

[1] Fluoride determination made by the modified Fairchild ferric chloride method described in Ariz. Expt. Sta. *Tech. Bull.* **43** (1932). Unpublished data at hand now indicate that a concentration of fluorine in water of as little as 1 p. p. m. as determined by the Willard or Foster methods of analysis is sufficient to cause mottled enamel of human teeth. A concentration of 2 p. p. m. is now found to be associated with mottled enamel of the more severe type.

differences are probably a reflection of the differences in solubility of the compound, although barium fluosilicate in spite of its relative insolubility has been found in every case to equal sodium fluoride in toxicity when fed at the same level of fluorine.

Fluorine is more toxic to the teeth of human beings than to rat incisors which grow at a more rapid rate. Small amounts of fluorine in excess of that usually occurring in food and water causes the damage to the developing teeth of human beings known as mottled enamel. More than five hundred water supplies have been analyzed and their fluorine contents correlated with the occurrence of mottled enamel among the users of these waters. The findings have shown that use of drinking water containing as little as 1 p. p. m. of fluorine produces mottled enamel (*12*, *13*). In other words, the permanent teeth of a child who drinks water containing 1 p. p. m. of fluorine during the period of formation of these teeth will be mottled when erupted. If we assume an average water intake of from four to eight glasses per day during that period, the average intake of fluorine from this source will be from 1 to 2 mg. per child per day. Analyses of apples which have been sprayed with barium fluosilicate have shown an average fluorine residue before washing of 5.6 p. p. m. (*9*) or approximately 2.5 mg. of fluorine per pound. A large apple a day alone, therefore, would provide as much fluorine as four glasses of water containing 1 p. p. m. of fluorine.

It appears to the authors, therefore, that the use of fluorine compounds for the control of insect damage to fruits and vegetables is fraught with danger, and unless steps are taken to control the use of these compounds or to insure their subsequent removal from the food material, mottled enamel will be more widespread and will no longer be only a sectional problem.

Conclusions

Retardation of the growth rate during a 6-week experimental period and a decrease in the efficiency of utilization of the food—i. e., a lower gain in weight per gram of food consumed—resulted when fluorine was incorporated in the diet at a 0.0226 percentage level when the source was sodium, potassium, or ammonium fluoride, or barium or sodium fluosilicate. Cryolite and calcium fluoride were found to be less toxic sources of fluorine from the standpoint of their effect upon growth rate and food utilization. It required ten times as much fluorine combined as cryolite and twenty times as much when combined as calcium fluoride to produce the slight stunting of growth which was caused by the inclusion of 0.0226 per cent fluorine combined as the sodium salt. The difference in apparent toxicity of these compounds was even greater when the amount of fluorine necessary to inhibit growth severely were considered. It required thirty times as much fluorine combined as cryolite than as sodium fluoride to reduce the growth rate to half that of the control non-fluorine-fed litter mates.

Fluorine interfered with the normal process of reproduction only at those levels of feeding which resulted in a stunting in growth of the female. No evidence of specific damage to the reproductive organs was evidenced.

A concentration of fluorine of 0.0904 per cent from sodium, potassium, and ammonium fluorides and from any of the fluosilicates studied, including barium, caused death of the animals in from 9 to 11 days. On the other hand, it required a concentration of 3.6 per cent fluorine from cryolite and 5.4 per cent from calcium fluoride to cause death in the same length of time. This meant a daily intake of approximately 40 mg. per kg. of fluorine from sodium fluoride as compared with 1900 and 3400 mg. per kg. from cryolite and calcium fluoride, respectively. Therefore from the standpoint of lethal concentrations and amount of fluorine necessary to cause growth inhibition, wide differences in toxicity of some of the compounds of fluorine were noted.

Fluorine in concentrations far below those necessary to interfere with growth or general health of the animal caused damage to the incisor teeth. Considered from the standpoint of the most severe type of damage to the teeth, again it was noted that it required more cryolite and calcium fluoride than any of the other compounds studied. At least ten times as much fluorine from cryolite and twenty times as much from calcium fluoride were necessary to cause corrosion and pitting of the enamel. However, all the compounds of fluorine studied, including the less soluble barium fluosilicate, cryolite, and calcium fluoride, left a mark characterized by the appearance of fine, lighter pigment lines on the incisor teeth when incorporated in the diet at a 0.0014 per cent level (14 p. p. m.) or fed separately in daily doses of 0.2 mg. fluorine to young rats or fed at the level of 1 mg. per kg. of body weight. Fluorine, regardless of its source, caused interference with normal tooth development at this level of feeding, but no discernible effect could be consistently noted when less than this amount was fed. The amount of fluorine required to cause initial damage to the rat incisors was so small that differences in solubility of the compounds were not a factor, and no differences in toxicity of fluorine from the various compounds studied could be noted.

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Received February 12, 1934.

Decrease of Fertilizer Prices in France. The French Ministry of Commerce has issued the following note:

In order to diminish the cost of living in foodstuffs, the government, endeavoring to reduce the cost of production of agricultural products, has just obtained from the industrialists concerned a very important decrease in the price of fertilizers.

The producers have agreed to make reductions of 6 per cent on superphosphates and of 8 per cent on potash fertilizers, against the assurance which was given them by the recent decree of April 28 (this decree requires individual import licenses for phosphate and potash fertilizers) that the importation of phosphate and potash fertilizers will be hereafter strictly limited to the indispensable quantities to provision the French market. It should be noted that a reduction of 6 per cent on nitrogenous fertilizers was already obtained by the government during the course of the present agricultural year.

REVIEW OF THE 2006 UNITED STATES NATIONAL RESEARCH COUNCIL REPORT: *FLUORIDE IN DRINKING WATER*

Robert J Carton[a]

Averill Park, NY, USA

SUMMARY: The recent report by a 12-member committee of the US National Research Council (NRC) examined the scientific basis for the Maximum Contaminant Level Goal (MCLG) of fluoride in drinking water promulgated in 1985 by the US Environmental Protection Agency (EPA). Due to misdirection by EPA management, who requested the report, the NRC committee identified only health effects known with total certainty. This is contrary to the intent of the Safe Drinking Water Act (SDWA), which requires the EPA to determine "whether any adverse effects can be reasonably anticipated, even though not proved to exist." Further misdirection by EPA consisted of instructing the committee not to identify a new MCLG—in other words, not to determine a safe level of fluoride in drinking water, and not to discuss silicofluorides, phosphate fertilizer manufacturing by-products used in most cities to fluoridate their water. Despite these restrictions, the committee broke new ground declaring severe dental fluorosis and moderate (stage II) skeletal fluorosis adverse health effects, and by noting that the current standard of 4 mg F/L in drinking water does not protect against bone fractures or severe dental fluorosis. Silicofluorides were said to need health effects testing. The NRC review includes extensive information on other possible health effects of fluoride, such as endocrine effects and effects on the brain. On the basis of this information and the proper interpretation of the SDWA, the following are all adverse health effects: moderate dental fluorosis, stage I skeletal fluorosis (arthritis with joint pain and stiffness), decreased thyroid function, and detrimental effects on the brain, especially in conjunction with aluminum. The amount of fluoride necessary to cause these effects to susceptible members of the population is at or below the dose received from current levels of fluoride recommended for water fluoridation. The recommended Maximum Contaminant Level Goal (MCLG) for fluoride in drinking water should be zero.

Keywords: Drinking water; US Environmental Protection Agency (USEPA); Exposure; Fluoride toxicity; Maximum Contaminant Level Goal (MCLG); National Research Council (NRC); Regulations; Risk assessment; Silicofluorides; Toxicity assessment.

INTRODUCTION

In 2002, the US Environmental Protection Agency (USEPA) asked the National Research Council (NRC) to independently evaluate the scientific basis of EPA's

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Maximum Contaminant Level Goal (MCLG) of 4 mg/L and the Secondary Maximum Contaminant Level (SMCL) of 2 mg/L of fluoride in drinking water. On March 22, 2006, NRC released its report in which it is clearly stated that the current MCLG does not prevent adverse health effects, and that the regulatory Maximum Contaminant Level (MCL) should be lowered.[1] Although the NRC committee was appointed to provide a balance of views on the safety of water fluoridation, it did not determine a fluoride level in drinking water that would protect against known or suspected adverse health effects with an adequate margin of safety. Instead, the committee deferred this analysis to EPA, which is required by the 1974 Safe Drinking Water Act (SDWA) to periodically review its standards. Indeed, according to Dr Hardy Limeback, one of the members of the committee, "We were clearly instructed to avoid trying to figure out a new MCLG."[2] Instead, according to another member of the committee, Dr Kathleen Thiessen, "We endeavored to provide a solid information basis for the conclusions that need to be drawn by EPA and others."[3]

This review analyzes whether or not the committee fully utilized its mandate and provided sufficient information to allow EPA to come to conclusions required by law.

EPA CHARGE TO THE COMMITTEE

The mandate of the committee, as explained by a representative of EPA at a public meeting held with NRC in August 2003, was to reevaluate the scientific basis of the 1986 MCL (Maximum Contaminant Level), and the SMCL (Secondary Maximum Contaminant Level—the guideline used to protect against adverse cosmetic dental effects).[4] The request to focus on the MCL was identical to the requirement for the previous 1993 report by NRC. However, transcripts of this meeting show that the committee requested and obtained a change in its mission from evaluating the enforceable MCL to the unenforceable MCLG (Maximum Contaminant Level Goal). This change removed the committee from evaluating an essentially political decision that requires judgments about feasibility and cost, to the more reasonable and possibly more satisfying evaluation of the scientific basis for the 1985 health goal.

In this connection it should be noted that the current MCL and MCLG for fluoride are both 4 mg/L. There is no requirement that they be the same. Other inorganic chemicals, such as arsenic and lead, have higher MCLs than MCLGs due to the difficulty and expense of treatment. The MCLGs for arsenic and lead are zero, while their MCLs are 0.010 mg/L and 0.015 mg/L, respectively.

Specifically excluded from the charge was the issue of artificial water fluoridation. EPA claimed this was a CDC (Centers for Disease Control) program, not under its jurisdiction. This view was clearly stated by another representative of EPA at a subsequent presentation in November 2003.[5] Similarly, silicofluorides, the chemicals used to achieve 1 mg/L of fluoride in 92% of all fluoridated drinking water supplies, were identified as off limits for analysis in this report. The EPA representative suggested that these chemicals would be better addressed as separate contaminants, presumably by a different committee. He noted that the dissociation of silicofluorides in water is under investigation at the University of Michigan (study now published).[6]

The committee discovered, however, that it was not possible to exclude discussion of these issues. The chapter on sources of fluoride exposure states: "The major dietary source of fluoride for most people in the United States is fluoridated municipal (community) drinking water."[7] In the chapter on the immune system, the report notes that "Machalinski et al. (2003) reported that four different human leukemic cell lines were more susceptible to the effects of sodium hexafluorosilicate, the compound most often used in fluoridation, than to NaF." The report also states: "The possibility of biological effects of SiF_6^{2-} [silicohexafluoride ion], as opposed to free fluoride ion, should be examined." There are numerous other references to fluoridation and silicofluorides, and even an entire page in the section on neurotoxicity is devoted to the neurotoxic effects of silicofluorides.

SCIENTIFIC REQUIREMENTS OF MCLG

In the August 2003 meeting, EPA explained in a general way the differences between the MCL and MCLG. The MCLG was discussed as the health goal that protects against adverse health effects and provides an adequate margin of safety. An important distinction, however, was left out of the discussion, namely, the amount of certainty necessary to establish the existence of an adverse health effect. According to Congress, Recommended MCLs (or MCLGs as they are now called) "are to represent non-enforceable health goals which are to be set at a level which assures 'that the health of persons will be protected against *known or anticipated* adverse effects [of the substance], allowing an adequate margin of safety'."[8] (Emphasis added in reference)

This means Congress intended that the administrator of EPA could determine that an adverse health effect existed without having to show total certainty. As discussed in the *amicus curiae* brief submitted by the EPA professionals union to a US District Court in 1986: "Moreover, the legislative history makes clear that 'the Administrator must decide whether any adverse effects *can be reasonably anticipated, even though not proved to exist*'."[9] (Emphasis added).

This distinction was not explained to the committee. The committee had much broader leeway in determining health effects than they apparently knew. If they had known, their discussions could have indicated possible adverse health effects to sensitive members of the population at fluoride levels well below 1 mg/L. Moreover, contrary to the conclusions of the committee, no new research is necessary to make this determination. More research is necessary of course to understand more fully the chronic effects of fluorides, silicofluorides, and their interactions with other chemicals in and out of the body. Here, however, we are not limited and can therefore draw conclusions based on the Precautionary Principle as embodied in the requirements of the Safe Drinking Water Act as stated above.

If the committee had looked at the existing MCLG of 4 mg/L in light of the proper legal requirement, they might have asked the following questions:

1 What health effects can reasonably be anticipated to occur, although not proved to exist, to the most sensitive members of the population?

2 What is the lowest level at which these effects occur?

3 What margin of safety would be adequate given the level of certainty of the data?

A proper review of the scientific basis of the 1985 standard would answer these questions and compare them with the current standard.

FOCUS OF COMMITTEE

The committee apparently believed that it was their mission to identify only health effects known with total certainty. They also apparently believed that they should not identify the Lowest Observed Adverse Effect Level (LOAEL) at which these health effects can be found. Instead, they focused mainly on the safety of the numerical level of the current MCLG of 4 mg/L, and the SMCL of 2 mg/L.

To demonstrate the conclusions that are possible using the proper interpretation of the law, this review addresses the adverse health effect identified by the committee (severe dental fluorosis and bone fractures), and a number of other health effects discussed by them (skeletal fluorosis, endocrine effects, and effects on the brain).

DENTAL FLUOROSIS

The committee agreed that enamel fluorosis is a dose-related mottling of enamel, which is permanent once a child's teeth are formed. It is described as a toxic effect caused by fluoride interfering with ameloblasts in the developing tooth, resulting in a disruption of the process of enamel formation making it ever more porous. What is new in this analysis is the agreement by the committee that the most severe form of dental fluorosis is an adverse health effect, contradicting the official position of the Surgeon General and EPA in 1985, which claimed it is only cosmetic. While breaking new ground in this regard, the committee balked at including moderate dental fluorosis as an adverse health effect because of the lack of absolute certainty of the damage.

The committee stated that the available data are not adequate to categorize moderate enamel fluorosis as an adverse health effect on the basis of structural or psychological effects.[10] However, the weight of evidence of the possible adverse nature of this health effect appears to be sufficient to include it in the list of adverse health effects. The following statements from the report justify this assessment.

First: "In *moderate to severe* forms of fluorosis, porosity increases and lesions extend toward the inner enamel. After the tooth erupts, its porous areas may flake off, leaving enamel defects where debris and bacteria can be trapped. The opaque areas can become stained yellow to brown, with more severe structural damage possible, primarily in the form of pitting of the tooth surface."[11] (Emphasis added)

This statement suggests quite strongly that moderate dental fluorosis includes structural damage to tooth enamel, although not to the degree seen in severe dental fluorosis. As the report states: "One of the functions of tooth enamel is to protect the dentin and, ultimately, the pulp from decay and infection."[12] Thus the definite possibility exists of a detrimental effect on the tooth, which should be prevented.

Second: "It is reasonable to assume that some individuals will find moderate enamel fluorosis on front teeth to be detrimental to their appearance."[13]

One possible explanation for ignoring moderate fluorosis as an adverse health effect is that the level at which it may occur coincides with the level of artificial water fluoridation, 0.7–1.2 mg/L. Selecting severe fluorosis as an adverse health effect was a concession but not one the committee thought would occur at water fluoridation levels. In the report they give assurances that the occurrence of severe

fluorosis would be near zero below 2 mg F/L. The unspoken assumption here is that "near zero" is not sufficient to trigger a protective MCLG. This is contrary to the Safe Drinking Water Act, which does not allow for damage to occur to any fraction of the population.

The 1993 NAS review reported an incidence of severe dental fluorosis in 4 cities of approximately 0.1% at the levels of water fluoridation If this low incidence was found in only these 4 cities, irrespective of the incidence found in any other city or cities, this should be determined as the LOAEL and then a safety factor applied to allow for missing data and the wide variation in fluoride intake from sources other than drinking water. Taking moderate dental fluorosis into account, the MCLG would be lower than 0.7 mg/L.

Missing from the report is any indication of the minimal dosage necessary to cause moderate or severe dental fluorosis. There exists a determination by EPA in its Integrated Risk Information System (IRIS) database that the reference dosage, which would prevent objectionable dental fluorosis (moderate and severe), is 0.06 mg/kg/day.[14] This is slightly lower than what the Institute of Medicine (IOM) determined in 1997, or 0.10 mg/kg/day, which was pointed out in the NRC report. Interestingly the committee also noted that "infants (nursing and non-nursing) and children 1–2 years old would be at or above the IOM limits at a fluoride concentration of 1 mg/L."[15] These numbers are for the average child and do not represent the 99th percentile of exposure. Consequently, a recommendation should have been made to establish moderate fluorosis as an adverse health effect and an attempt made to calculate a fluoride concentration in water that would prevent children from getting that effect, using the 99th percentile as the target group. This was done by a consulting firm, Pacific Western Technologies, Ltd (PWT), for the US Army as part of an environmental assessment evaluating the possibility of fluoridating the water supply of Fort Detrick in Frederick, MD. PWT found that over 50% of children, between the ages of one and three-years-old, exceeded the EPA reference dosage of 0.06 mg/kg/day at the naturally occurring concentration in the Fort Detrick source water of 0.2 mg/L.[16] With only 0.2 mg/L in the drinking water, fluoride from all other sources consumed by a small child exceeded the EPA reference dose for a large fraction of that sub-population. This brought into question the wisdom of adding even more fluoride to their diet through water fluoridation at 1.0 mg/L.

BONE FRACTURES

The entire committee agreed, "Fluoride can weaken bone and increase the risk of fractures."[17] A majority of the committee believed that people exposed to 4 mg/L in their drinking water over their lifetime are likely to have an increase in bone fractures over those exposed to 1 mg/L. The summary of the report explains that the best study they reviewed actually found a risk of hip fracture above 1.5 mg/L, but this "study alone is not sufficient to judge fracture risk for people exposed to fluoride at 2 mg/L."[18]

This is not a necessary analysis, however, for the purposes of determining a new MCLG and for carrying out the purposes of the Safe Drinking Water Act. The biological certainty of fluoride weakening bone is demonstrated in clinical studies in humans and with animals. The report also says that there appears to be a gradient of effect between 1 and 4 mg/L, and that at 2 mg/L the evidence suggests

an increased risk of bone fracture. These statements could be used as a basis for setting an MCLG taking into account the need to protect susceptible individuals, such as those with high water intakes due to occupational necessity or medical condition. The report explains these exposure extremes in detail. What is not discussed is the magnitude of the safety factors necessary to insure protection from anticipated adverse health effects.

SKELETAL FLUOROSIS

The existing MCLG of 4 mg/L is based on the prevention of severe skeletal fluorosis, or Stage III skeletal fluorosis, as it is also known. The NRC committee expanded concerns for skeletal effects by including Stage II as an adverse health effect, declaring that: " . . . mobility is not *significantly* affected, but it is characterized by *sporadic pain, stiffness of joints*, and osteosclerosis of the pelvis and spine."[19] (Emphasis added)

Curiously, the reference to sporadic pain and stiffness of joints avoids the word "arthritis" used in describing the same clinical signs in Stage III. Nevertheless, arthritis could be used as a term to describe these symptoms. Rather than implying a specific etiology, arthritis is a general term for the presence in a joint of inflammation, the classical features of which are heat, swelling, redness and pain. Thus within the broad category of arthritis, in which it is implied that some but not necessarily all of the symptoms and signs of inflammation are present, the condition of Stage I skeletal fluorosis, due to exposure to fluoride, with the symptoms of joint pain and stiffness, may be placed alongside approximately 100 other forms of arthritis, with different etiologies, such as gout, osteoarthritis, rheumatoid arthritis, psoriatic arthropathy, ankylosing spondylitis, and postinfectious arthritis. Previously, only the effect of actual crippling was regarded by the NRC as an adverse health effect. Fluoride exposure, then, can now be officially listed as one of the causes of arthritis.

The committee had insufficient information to determine if Stage II or Stage III skeletal fluorosis was occurring in the US, and they failed to speculate on the possibility of the very large historical increase in cases of arthritis in the US being due to the ever-increasing amounts of fluoride exposure. Instead, they used a model they developed to estimate the possibility of Stage II occurring based on studies with known concentrations of fluoride in the drinking water and fluoride levels in bone. The model found that at 2 mg/L of fluoride in drinking water, the amounts of fluoride in bone ash from subjects exposed to these levels "fall within or exceed the ranges historically associated with Stage II and Stage III skeletal fluorosis . . ."[20] This indicates the likelihood that some individuals in the US *may* be experiencing Stage II and Stage III skeletal fluorosis at less than 2 mg/L despite the following statement by the committee: " . . . this comparison alone is insufficient for determining whether Stage II or Stage III skeletal fluorosis *is* a risk for populations exposed to fluoride at 4 mg/L."[21] (Emphasis added)

The key to understanding how the data should be evaluated goes back to the original legal mandate from Congress in setting standards (see above). Absolute proof is not needed to act when there are data showing possible harm. The possibility that harm may be occurring is more than justified based on the following additional analysis of the fluoride dose used to derive the current EPA standard of 4 mg/L.

According to EPA representatives at the August 2003 meeting with NRC, EPA claimed that the MCLG is based on the LOAEL of 20 mg/day for 20 years "from case studies in a limited number of kid [= child] studies of crippling clinical skeletal fluorosis." While differing substantially from previous assertions by EPA that the 1985 MCLG is based on a statement by Dr Harold C Hodge, this calculation does serve as a useful point of departure for looking at its implications for earlier stages of fluorosis. First, however, the actual lifetime dose needs to be calculated for Stage III skeletal fluorosis in order to deal with real life exposures. Thus, the 20 mg/day for 20 years should be multiplied by 20/70, where 70 is the average life expectancy. This results in a dose of 5.7 mg/day. Using the only in-depth study ever done on human exposure by Dr Kaj Roholm,[22] one can evaluate the possible doses necessary to cause the early stage of skeletal fluorosis as follows: Stage II occurred in Danish cryolite workers in approximately 1/2 of the time it took for workers to reach Stage III. Stage 1 occurred in 1/4 of the time. Thus we have the possibility of Stage I and Stage II occurring with a daily dose over a lifetime of 1.42 mg and 2.86 mg, respectively. These are both within the range of current fluoride exposures from all sources documented in the NRC report.

ENDOCRINE EFFECTS

The NRC report cites many endocrine effects of fluoride exposure, including decreased thyroid function, impaired glucose tolerance (Type II diabetes), and earlier sexual maturity. The Executive Summary of the report merely states that these effects are achievable with fluoride concentrations in drinking water of 4 mg/L or less.

Many details, however, can be found in the chapter on effects on the endocrine system. The summary at the end of the chapter explains the dosage necessary to affect thyroid function: "In humans effects on thyroid function were associated with fluoride exposures of 0.05–0.13 mg/kg/day when iodine intake was adequate and 0.01–0.03 mg/kg/day when iodine intake was inadequate . . ." [23] This simply means that for a 70-kg person (often called the "standard man"), fluoride doses as low as 3.5 mg/day for those with an adequate intake of iodine, and 0.7 mg/day for those with an inadequate intake of iodine may have an affect on the thyroid. The report also notes: "The recent decline in iodine intake in the United States could contribute to increased toxicity of fluoride for some individuals." Impaired glucose tolerance was identified as occurring in humans at levels as low as 0.07 mg/kg/day or 4.9 mg/day for a 70-kg man. Either of these effects could occur at water fluoridation levels of 1 mg/L to some people with the high water intakes identified in the report.

Moreover, the committee noted that some of the identified endocrine effects may not be adverse but are nonetheless grounds for concern because apparently even minor endocrine disruption may still cause adverse health effects. Given these possibilities, it is logical to base the MCLG on the lowest endocrine effects found for the most susceptible populations. If thyroid effects were used, this would mean that the total dose of fluoride from all sources should be less than 0.7 mg/day. This intake level covers susceptible people with iodine deficiency. Since the average American already exceeds this dose in the diet, the MCLG for fluoride in drinking water should be zero.

NEUROTOXICITY AND NEUROBEHAVIORAL EFFECTS

The committee also cited research indicating adverse health effects such as lower IQ in children, behavioral, and histopathological changes in the brains of laboratory animals (some of these resembling the brains of Alzheimer's patients), cerebral impairment of humans, and enhancement of effects in the presence of aluminum. The report concludes: "fluorides have the ability to interfere with the functions of the brain and the body by direct and indirect means." It also noted that many of the adverse effects of fluoride can be attributed to the formation of aluminum-fluoride complexes. The report provides a wealth of information showing the negative effects of fluoride on the brain but is often unduly cautious in drawing the appropriate conclusions. The summary[24] states: "A few epidemiological studies of Chinese populations have reported IQ deficits in children exposed to fluoride at 2.5 to 4 mg/L in drinking water." This information is said to "lack sufficient detail to fully assess their quality and relevance to US populations." However, the results are significant enough to "warrant additional study."

The report goes on to identify "a few animal studies" reporting alterations in the behavior of rodents. Limiting the impact of this statement, the committee concluded that the changes were not "substantial." They list "molecular, cellular, and anatomical changes in the nervous system . . . suggesting that functional changes could occur." More research is urged to "clarify the effects . . . on brain chemistry and function." Of particular concern is their statement: " . . . histopathological changes similar to those traditionally associated with Alzheimer's disease in people have been seen in rats chronically exposed to AlF [sic] (Varner et al. 1998)."[25]

Given these and many other examples, there is little doubt that fluoride affects the brain and that it enhances the uptake of aluminum in the brain. Human observations support the conclusion of brain effects, and animal studies allow dose levels causing these effects to be estimated for the purposes of developing an MCLG.

Exposure figures mentioned in this and other sections of the report often give only animal data. However, the committee suggested a way to convert such data to human exposures.[26] Apparently rats require 5 times the daily dose required by humans to arrive at the same serum concentrations. Thus, rats exposed to fluoride at 5 mg/L would achieve the same serum fluoride concentrations as humans exposed to 1 mg/L.

As noted in the report,[27] rats administered AlF_3 in drinking water at 0.5, 5.0, and 50 mg/L for 45 weeks (approximately 60% of AlF_3 is fluoride), all had significant damage in the hippocampus. An unusual number of deaths occurred at the lowest dose tested. A repeat of the test comparing AlF_3 at 0.5 mg/L and NaF at 2.1 mg/L for a test period of one year found that 6 out of 9 animals died in the AlF_3 group, 3 out of 9 of the NaF group died, and only 1 out of 9 control animals died. Both treated groups had twice as much aluminum in their brains as control animals. Leaving aside the unexplained deaths, there was a proven increase of AlF_3 in the brain with both AlF_3 and NaF, and significant damage to the brain at the low dose of 0.5 mg AlF_3/L, or approximately 0.3 mg F/L.

Two other studies were noted to have found the same pattern of neuronal degeneration. Thus, there exists a lowest observed effect level of 0.06 mg/L of

fluoride to develop an MCLG using the preventative approach of the Safe Drinking Water Act as mentioned earlier. (This figure of 0.06 mg/L is derived from the above 0.3 mg/L concentration of fluoride divided by the conversion factor for rats to humans of 5.) An appropriate safety factor does not have to be mentioned to see clearly that fluoridation at 1 mg/L cannot be considered acceptable for an MCLG.

CONCLUSIONS

The NRC committee's reevaluation of EPA's MCLG for fluoride in drinking water failed to identify a safe level of fluoride in drinking water. This failure can be attributed to misdirection by EPA of the intended goal of the effort. When the committee requested and received a change in its mandate from evaluating the MCL to the MCLG, EPA strangely omitted the key scientific criteria necessary for evaluating this standard. The committee should have been told to look for health effects that "can be reasonably anticipated, even though not proved to exist." As a result of this omission, the NRC panel focused only on end points that were totally certain and concluded that the current standard of 4 mg/L did not protect against bone fractures and severe dental fluorosis. For the first time in history, a committee of the NRC removed severe dental fluorosis from the benign category of cosmetic effects and added it to the list of adverse health effects. In addition, Stage II skeletal fluorosis was added to the list, but the committee was unable to state with absolute certainty that this was occurring at the current EPA standards.

This review applied the necessary criteria to some but not all of the adverse health effects discussed in the NRC report. The results are as follows:

1 Moderate dental fluorosis is an adverse health effect occurring at fluoride levels of 0.7–1.2 mg/L, the levels of water fluoridation.

2 The Lowest Observed Adverse Effect Level (LOAEL) for bone fractures is at least as low as 1.5 mg/L and may be lower than this figure.

3 Stage II and Stage III skeletal fluorosis may be occurring at levels less than 2 mg/L.

4 Stage I skeletal fluorosis, (arthritis, clinically manifested as pain and stiffness in joints) is an adverse health effect which may be occurring with a daily fluoride intake of 1.42 mg/day, which is less than the amount the average person already obtains in their diet in non-fluoridated areas. The Maximum Contaminant Level Goal (MCLG) should be zero.

5 Decreased thyroid function is an adverse health effect, particularly to individuals with inadequate dietary iodine. These individuals could be affected with a daily fluoride dose of 0.7 mg/day (for a "standard man"). Since this is less than the amount already in the diet, the MCLG should be zero.

6 Fluoride has adverse effects on the brain, especially in combination with aluminum. Seriously detrimental effects are known to occur in animals at a fluoride level of 0.3 mg/L in conjunction with aluminum. The goal for this effect should also be zero.

The committee should be applauded for their efforts in general and in particular for ignoring directives not to include discussions of water fluoridation and silicofluorides. Their recommendations for research should be taken seriously. EPA has sufficient information in this report to act immediately, using the appropriate criteria set forth in the Safe Drinking Water Act. Using the preventive public health intent of the law, the Maximum Contaminant Level Goal for fluoride in drinking water should be zero.

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A new perspective on metals and other contaminants in fluoridation chemicals* by Phyllis J. Mullenix / Andover, MA, USA / International Journal of Occupational and Environmental Health 2014 VOL.20 NO.2 157

Background: Fluoride additives contain metal contaminants that must be diluted to meet drinking water regulations. However, each raw additive batch supplied to water facilities does not come labeled with concentrations per contaminant. This omission distorts exposure profiles and the risks associated with accidents and routine use.

Objectives: This study provides an independent determination of the metal content of raw fluoride products.

Methods: Metal concentrations were analyzed in three hydrofluorosilicic acid (HFS) and four sodium fluoride (NaF) samples using inductively coupled plasma-atomic emission spectrometry. Arsenic levels were confirmed using graphite furnace atomic absorption analysis.

Results: Results show that metal content varies with batch, and all HFS samples contained arsenic (4.9–56.0 ppm) or arsenic in addition to lead (10.3 ppm). Two NaF samples contained barium (13.3–18.0 ppm) instead. All HFS (212–415 ppm) and NaF (3312–3630 ppm) additives contained a surprising amount of aluminum.

Conclusions: Such contaminant content creates a regulatory blind spot that jeopardizes any safe use of fluoride additives.

*Presented in part at the North Jersey District Water Supply Commission's Educational Seminar "Pharmaceutical Contaminants in the Water Treatment Industry," Wanaque, New Jersey, July 14, 2009.

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