

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 (an arthritis-like condition 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

^a Robert J. Carton, PhD, is an environmental scientist who has worked for over 30 years in the US federal government writing regulations, managing risk assessments on high priority toxic chemicals, and providing environmental oversight of medical research conducted by the government. From 1972–1992 he worked at the headquarters of the US Environmental Protection Agency in Washington, DC, and wrote the first regulations for controlling asbestos discharges from manufacturing plants. In 1985, as an official in the union for professionals at EPA, he became aware of possible scientific fraud in the development of the EPA standard for fluoride in drinking water and convinced the union to challenge EPA in court. From 1992–2002, he was Chief of Environmental Compliance for the US Army Medical Research and Materiel Command, Fort Detrick, MD. He successfully opposed attempts to put fluoride in the post’s drinking water. Since retiring from government service he has continued to work on exposing the unethical nature of government claims about fluoridation.

For correspondence: 11 Pond Hollow Road, Averill Park, NY 12018, USA.
Email:bcarton@verizon.net

of, EPA's 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 I 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 effect 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 summary²⁴ 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 AIF [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 AIF₃ in drinking water at 0.5, 5.0, and 50 mg/L for 45 weeks (approximately 60% of AIF₃ 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 AIF₃ 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 AIF₃ 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

AlF₃ in the brain with both AlF₃ and NaF, and significant damage to the brain at the low dose of 0.5 mg AlF₃/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, (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 exceeds the amount the average person 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 exceeds 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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