

EDITOR'S NOTE

The two letters referred to at the beginning of the letter below were published in *Fluoride* 31(3) 153-157 August 1998.

In a separate letter from the National Academy of Sciences (NAS), also dated November 20, 1998, James Jensen, Director of the National Research Council Office of Congressional and Governmental Affairs of NAS, replied to an inquiry from Pennsylvania Senator Arlen Specter on behalf of one of his constituents, who wanted to know why my joint letter of October 15, 1997 to Dr. Bruce Alberts, President of NAS, had not received a reply. In his letter to Senator Specter, Mr. Jensen wrote:

“When Dr. Burgstahler’s letter on fluoridation [actually, it was about the proposed Dietary Reference Intake standards for fluoride and only indirectly about fluoridation] arrived at the Academy, a response was drafted but never sent out. There is little excuse for this, but this is what occurred. . . .

“Please accept our sincere apologies. There was no intent to show disrespect to your constituent.”

The letter below, therefore, although “drafted” earlier, was sent only after prompting from Senator Specter’s inquiry.

**NATIONAL ACADEMY OF SCIENCES
INSTITUTE OF MEDICINE**

2101 Constitution Avenue, Washington, D.C. 20418

November 20, 1998

Albert W. Burgstahler, Ph.D. and others
Professor of Chemistry
The University of Kansas
4035 Malott Hall
Lawrence, Kansas 66045

Dear Dr. Burgstahler:

We apologize for the delay in responding to your letters of October 15, 1997 and February 4, 1998, to each of us individually. At the time we had a very large number of inquiries and comments, and while letters were prepared in response to your letter along with the others, for some reason they did not reach you. The letter that we found in our files is reprinted below.

We want to thank you and your co-signers for your October 15, 1997 letter to us concerning the Food and Nutrition Board’s (FNB) recent report, *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D and Flu-*

oride. The publication of the report represents the initial report of a major new activity of the FNB: the development of a comprehensive set of reference values for nutrients and food components of possible benefit to health, that may not meet the traditional concept of a nutrient. If adequate scientific data exist that support a health benefit from the inclusion of these components in the diet, reference intakes will be established.

In replying to your letter, we have consulted with the Committee that produced the FNB report and asked them to review the important points that you raised concerning their report and the associated workshop, as well as to explain why they have reached the conclusions they reached despite the information you cite.

First, let us reassure you with regard to one concern. Nowhere in the report is it stated that fluoride is an essential nutrient. If any speaker or panel member at the September 23rd workshop referred to fluoride as such, they misspoke. As was stated in *Recommended Dietary Allowances 10th Edition*, which we published in 1989: "These contradictory results do not justify a classification of fluoride as an essential element, according to accepted standards. Nonetheless, because of its valuable effects on dental health, fluoride is a beneficial element for humans." Dr. Vernon Young, Chair of the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, stated this at the workshop's conclusion.

The adequate intake (AI) of fluoride for infants 0 to 6 months of age is set at 0.01 mg/day. As explained in Chapter I of the report, the average intake of a particular nutrient by full-term infants who are born to well-nourished mothers and exclusively fed human milk has been adopted as the basis for deriving an AI for all nutrients and other food components during the first 6 months of life. Using the human milk-fed infant as the model supports the recommendation that exclusive breast feeding is the preferred method of feeding for normal fullterm infants for the first 4 to 6 months of life - a recommendation shared by the Canadian Paediatric Society (Health Canada, 1990), the American Academy of Pediatrics (1982), and the Food and Nutrition Board's report *Nutrition During Lactation* (IOM, 1991). (Infants who are exclusively breast fed for the first six months of life would have a low fluoride intake, and yet scientific evidence showing that these infants are at greater risk for dental caries than formula-fed infants is lacking.) During the second six months of life and thereafter, the AI for fluoride from all sources is set at 0.05 mg/kg/day because it confers a high level of protection against dental caries and is associated with no known unwanted health effects.

Although the report acknowledges that most of the anti-caries effect attributed to fluoride occurs by topical exposure, it does not matter whether that exposure is from food, water or dental products. As you state, the prevalence of caries in some countries around the world without water fluoridation has declined over the years. This has been attributed to national dental hygiene programs and the use of fluoride in school-based prevention programs (rinses or tablets), as well as to the use of fluoridated toothpaste. These programs provide both systemic and topical fluoride exposures.

In following the model for the development of Tolerable Upper Intake Levels (ULs), as explained in Chapter 3 of the report, moderate enamel fluorosis was considered as the critical adverse effect in children under 9 years of age. As noted by Dean and coworkers some 60 years ago, mild enamel fluorosis was present in some residents of areas where water contained < 2 mg/liter of fluoride. At that time the diet, particularly the water, was the only significant source of fluoride so that the daily intake of fluoride could be estimated with reasonable accuracy. The average intake by children at risk was 0.05 mg/kg/day. The prevalences of both dental caries and fluorosis in these communities was low and there is no reason to expect that level of intake (from all sources) to produce different results today. Compared with Dean's findings, recent studies have revealed a higher prevalence of dental fluorosis in the United States and Canada, including a few cases of moderate fluorosis. However, analytical epidemiological studies have repeatedly shown that the major risk factors are ingestion due to the early use of fluoride toothpaste and/or the use of dietary supplements. Thus, the total fluoride intake by some children whose water is fluoridated is now higher than in Dean's time. This is the reason that age-specific intakes for total fluoride, including that from dental products, are based on 0.05 mg/kg/day. To the extent that this intake level is followed, the prevalence of dental fluorosis can be expected to decline while a healthy dentition is maintained.

Three recent reviews of the literature, Kaminsky et al., 1990; NRC, 1993; USPHS, 1991, attempted to identify adverse functional effects of fluoride ingestion in adults. Fluoride exposures included those associated with drinking water containing as much as 8 mg/liter of fluoride and the use of dental products. These reviews concluded that evidence linking chronic, high fluoride exposures with adverse effects such as cancer, including osteosarcoma, birth defects, genetic disorders, or bone fractures is either insufficient or highly contradictory. In addition, the majority of animal studies have shown no effect on cancer, birth defects, genetic disorders or bone strength of very high and long-term fluoride exposures. Thus, the primary adverse effects associated with chronic, excess fluoride intake are enamel fluorosis in children through 8 years of age and skeletal fluorosis in adolescents and adults over 8 years of age. In Hodge's 1979 article, he reported that evidence of crippling fluorosis "was not seen in communities in the United States where water supplies contained up to 20 ppm." In such communities daily fluoride intakes of 20 mg would not be uncommon.

Fluoride is continuously taken up by newly formed bone and released from older bone being resorbed. As long as intake remains constant, the concentration in bone tends to increase during life. It is not entirely clear why this happens but it may be due to the preferential resorption of bone crystallites that do not contain fluoride. In any event, in the United States and Canada, it is known that the development of skeletal fluorosis, even in earliest stages, has not occurred, even where the water fluoride concentrations have been in excess of 10 ppm.

In reviewing Kaj Roholm's classic 1937 report of bone changes among Danish cryolite workers, it was noted that Roholm reported no intake data for fluoride, apparently because the researchers were not able to measure airborne fluoride. On page 279, Roholm states: "It must be admitted that with respect to the important question of dose, that the observations available are sporadic and to some extent contradictory; in most of the spontaneous intoxications the intaken (his word) quantity of fluoride is not known at all." Later on page 319 Roholm states: "In man the disease (he is referring to crippling skeletal fluorosis) is probably caused by 0.20-0.35 mg fluoride daily per kg body weight." The reason for this estimate is not given. It is unfortunate that, in the absence of scientific data, these estimates were ever made. Although we are uncertain about the lower level of intake and time of exposure that causes clinically significant skeletal fluorosis, we do know that, at least for U.S. and Canadian citizens, intakes associated with water fluoride concentrations in excess of 10 ppm do not cause clinically significant skeletal fluorosis.

Our study was funded entirely by the governments of the United States and Canada. The funding agencies were the National Institute of Health's National Heart, Lung, and Blood Institute; the Agricultural Research Services of the U.S. Department of Agriculture; U.S. Food and Drug Administration; and Health Canada.

We thank you and your co-signers for your careful reading of the report and interest in assuring its accuracy and completeness. Given the complexities of the issues the report considers, we are confident that much room remains for further objective inquiry. We have tried to give you some of the reasons for the Committee's conclusions. However, we hope that the report will lead to additional research on which to base dietary reference intakes - for both essential nutrients and other dietary constituents with documented health benefits.

Once again, we regret that this reply to your thoughtful letter did not reach you much earlier, when it was prepared.

Sincerely,

Bruce Alberts, Ph.D.
President,
National Academy of Sciences

Kenneth Shine
President,
Institute of Medicine

EDITOR'S COMMENT

On January 12, 1999, the following letter was faxed to Drs. Alberts and Shine:

Drs Bruce Alberts and Kenneth Shine
National Academy of Sciences & Institute of Medicine
2101 Constitution Avenue NW
Washington, DC 20418
Fax: (202) 334-2316

Dear Drs. Alberts and Shine,

Your letter of November 20, 1998 in response to my letters of October 15, 1997, and February 4, 1998, awaited me on my return late last month from an extended trip to the Far East beginning in mid-November. A related letter dated November 19, 1998, from Professor Gary M. Whitford had also arrived.

Next week I plan to attend the workshop scheduled for January 21 at the Academy. Several others who cosigned my October 15, 1997, letter also plan to be there.

In view of the gravity of the issues addressed in these letters, would it be possible for some of us to meet briefly with either or both of you before 11:00 a.m. on Wednesday, January 20, at the National Academy of Sciences?

I will be leaving Lawrence early Monday morning (January 18), so the favor of your early reply by fax or e-mail (addresses below) would be appreciated.

Yours sincerely,

Albert W. Burgstahler
Professor Emeritus of Chemistry
The University of Kansas

There was no reply to the above letter. Upon my arrival at the NAS headquarters in Washington on the morning of January 20, there was still no reply, and no meeting could be arranged with either Dr. Alberts or Dr. Shine.

As indicated at the end of my February 4, 1998 letter to Dr. Shine (see *Fluoride* 31(3) 157 August 1998), I also sent a copy of that letter to Professor Gary M. Whitford of the Medical College of Georgia, who was the ranking fluoride expert on the review panel. Then on February 17, 1998, I again wrote to Professor Whitford, specifically asking why his views, published prominently in the 1996 edition of his monograph, *Metabolism and Toxicity of Fluoride* (p. 138), that "crippling skeletal fluorosis occurs when 10-20 mg of flu-

oride have been ingested on a daily basis for at least 10 years," were set aside in favor of suggesting that a much higher level of fluoride intake is required to produce this effect.

Nine months later a reply finally arrived from Professor Whitford. Dated November 19, 1998, just one day before the date on the foregoing letter from Drs. Alberts and Shine, Professor Whitford stated in his letter: "During the course of assisting with the development of the DRIs [Dietary Reference Intakes],...I concluded that the literature does not support the likelihood [sic] of advanced skeletal fluorosis in U.S. residents whose daily intake does not exceed 10 mg." This statement, however, is contradicted by the evidence presented in my letter of February 4 to Dr. Shine.

It is also of interest that much of the material in Professor Whitford's letter to me is found in the November 20, 1998 letter from Drs. Alberts and Shine, including the passage from Roholm's treatise *Fluorine Intoxication—A Clinical Hygienic Study*, that a daily fluoride intake of 0.20-0.35 mg/kg body weight is likely to cause crippling skeletal fluorosis. But then Drs. Alberts and Shine go on and boldly assert: "...we do know that, at least for U.S. and Canadian citizens, intakes associated with water fluoride concentrations in excess of 10 ppm fluoride do not cause clinically significant skeletal fluorosis."

But clearly, the average fluoride intake of an adult drinking water containing more than 10 ppm fluoride *will* very likely exceed 10 mg/day and therefore, according to Professor Whitford, *would* create a risk for crippling skeletal fluorosis, even in the United States and Canada. Why residents of these two countries supposedly do not develop skeletal fluorosis from levels of fluoride intake that are well known to cause it elsewhere is deftly shoved aside by citing studies in the U.S. that did not report finding it.

Equally disturbing in the Alberts-Shine letter is the unexplained jump of an "adequate" fluoride intake of only 0.01 mg/day for infants up to age six months to 0.05 mg/kg body weight/day for the second six months of life and thereafter. By age six months, a baby weighing 6-8 kg would therefore have an "adequate" fluoride intake of 0.3 to 0.4 mg/day — a 30- to 40-fold increase from the first six months to the second six months of life after birth! No such huge increase is proposed for any other dietary component.

As pointed out by Dr. John Yiamouyiannis at the end of the following letter, this 0.05 mg/kg/day figure for fluoride appears to be based on an effort to justify or "sanctify" water fluoridation. Thus, an average daily total fluoride intake of 3.5-mg by a 70-kg adult drinking 1-ppm fluoridated water amounts to 3.5 mg/70 kg/day or 0.05 mg/kg/day. And this is sound "scientific" thinking by the U.S. National Academy of Sciences?

In the end, however, all these considerations are moot, since the basis for setting an "adequate intake" of fluoride rests on its alleged ability to prevent tooth decay. But since any such dental benefit from fluoride, to whatever extent it exists, is now known to be largely topical and not systemic (from ingestion), how can there even be a daily "adequate intake"?

Safe Water Foundation • 6439 Taggart Road • Delaware, Ohio 43015

Bruce Alberts, Ph.D.
President, National Academy of Sciences
Kenneth Shine, M.D.
President, Institute of Medicine
2101 Constitution Avenue NW
Washington, DC 20418

Dear Drs. Alberts and Shine:

A copy of your November 20, 1998 letter to Professor Albert Burgstahler has been sent to me for comment. If you actually believe what you wrote in that letter, I am sure you will welcome the following information.

I. In your letter, you cite the 10th edition of Recommended Dietary Allowances as stating that while fluoride cannot be classified as an essential element, [but] “because of its valuable effect on dental health [which you later define as its ‘anticaries effect’], fluoride is a beneficial element for humans.” You then state as a fact that the intake of .05 mg/kg/day of fluoride confers a high level of protection against dental caries. Implicit in your letter is the claim that fluoridation of water reduces tooth decay. You then explain that part of the reason for the decline in tooth decay in nonfluoridated areas is due to the administration of fluoride tablets.

However, as any informed professional would know, there is a general consensus among experts on both sides of the fluoride issue that swallowing fluoride does nothing to prevent tooth decay. In fact, the only proven effect that swallowing fluoride has on teeth is to poison ameloblasts and odontoblasts, resulting in dental fluorosis, the formation of imperfect or damaged enamel or dentine, respectively, and slowing down the eruption rate of deciduous teeth.

Evidence that swallowing fluoride does not prevent tooth decay

- In water

All the recent large-scale studies taken together show that fluoridation is ineffective in reducing the decay rate of permanent teeth.¹

Earlier studies claiming a reduction in tooth decay have already been discredited by those who have and/or still do promote fluoridation. For example, Dr. John Colquhoun, former Chief Dental Officer of Auckland, New Zealand and at one time the President of the Fluoridation Society conducted the largest tooth decay study and found no difference in tooth decay rates in fluoridated and nonfluoridated areas in New Zealand. Dr. A.S. Gray, former Chief Dental Officer of British Columbia, found that British Columbia, the province with the lowest fluoridation rate in Canada, also had the lowest tooth decay rate in Canada. And Dr. Elmer Green and Taimi Carnahan, two of the most ardent promoters of fluoridation, coauthored a study with two others that showed that there was no significant difference in the tooth decay rates in fluoridated New-

burgh and nonfluoridated Kingston (in 1986) despite the fact that these two cities were used to show that fluoridation reduced tooth decay rate by 70% (from 1945 to 1955) {J.V. Kumar, et al., "Trends in Dental Fluorosis and Dental Caries Prevalences in Newburgh and Kingston, NY", American Journal of Public Health, Volume 79, pp. 565-569 (1989)}.²

Even Dr. Hardy Limeback, the fluoride expert for the Canadian Dental Association admits that the ingestion of fluoride does nothing to reduce tooth decay. On February 14, 1998, he wrote:

You should be glad to know that I no longer take such a profluoride stand. I don't care if I alienate all my dental public health colleagues anymore on this whole issue and I realize just how much out on a limb I'm climbing in no longer supporting water fluoridation.

Putting hydrofluorosilicic acid from smoke stack scrubbers to fluoridate the water for me was the breaking point.

Consider me still a pro-fluoride dentist but a converted antifuoridationist who now advocates that we stop putting toxic waste in Canadian water supplies – shame!

- In tablet or drop form

Contrary to your claims, fluoride tablets have been shown to be ineffective in reducing tooth decay in the Rand report sponsored by the Robert Wood Johnson Foundation (Robert Wood Johnson Foundation Special Report No.2, 1983) and in the scientific literature {H Kalsbeek, et al., Use of fluoride tablets and effect on prevalence of dental caries and dental fluorosis, Comm Dent Oral Epidemiol 20:243-245 (1992)}.

II. In your letter, you claim that part of the reason for the decline in tooth decay in nonfluoridated areas is due to the use of fluoride in school-based rinse programs.

Contradicting this unsubstantiated claim, a Rand Corporation study {Disney, et al., A case study in contesting the conventional wisdom. school-based mouthrinse programs in the USA, Comm Dent Oral Epidemiol 18:46-56 (1990)} has shown that topical fluoride exposure in school programs does not reduce tooth decay rates.

III. You acknowledge that "recent studies have revealed a higher prevalence of dental fluorosis" and claim that this is "due to the early use of fluoride toothpaste and/or the use of dietary supplements."

The absurdity of this can be seen by comparing the 1-2 mg per day children get from fluoridated water with the 1/4 mg per day of fluoride these same children get from ingesting toothpaste. While there are many studies showing the fluoridated water causes dental fluorosis, there are no studies showing that fluoride ingestion from fluoridated toothpaste by itself causes dental fluorosis. [This is not to deny, however, that the approximate 1/4 mg/day of fluoride con-

sumed from toothpaste might push the total fluoride intake over the top to cause dental fluorosis in marginal cases].

The fact that fluoridation causes dental fluorosis is obvious from your own NRC, 1993 report. Let's ask it some questions:

Does fluoridation result in dental fluorosis (fluoride-induced tooth damage)?

According to page 37 of your report: "...the prevalence of dental fluorosis in optimally fluoridated areas (both natural and added) in recent years ranged from 8% to 51%, compared with 3% to 26% in nonfluoridated areas."

Two things are obvious here: (1) we can safely assume that fluoride toothpaste usage is similar in both fluoridated and nonfluoridated areas and (2) we know that fluoride supplement use is higher in nonfluoridated areas – yet dental fluorosis rates are twice as high in fluoridated areas.

As a result, should we stop water fluoridation?

According to page 43 of your report: "Indeed, most dental researchers (Horowitz, 1991; Rozier, 1991 Szpunar and Burt, 1992) believe that the best approach to stabilizing the prevalence and severity of dental fluorosis is to control fluoride ingestion from foods, processed beverages, and dental products rather than reduce the recommended concentrations of fluoride in drinking water."

And on page 44 of your report it is stated: "Fluoride in foods and beverages processed with fluoridated water has long been suspected as a risk factor...."

But how can you control fluoride ingestion from foods and beverages processed with fluoridated water?

According to page 48 of your report: "applying such a policy would be formidable; reductions of fluoride in drinking water would be easier to administer, monitor, and evaluate."

The mission to fanatically support water fluoridation has apparently allowed you to disregard any form of logic, if necessary, in the production of your NRC, 1993 report.

IV. In you[r] letter, you claim to rely on "Three recent reviews of the literature, Kaminsky, et al., 1990; NRC, 1993; and USPHS, 1991."

Enclosed are critiques of Kaminsky, et al., 1990, NRC, 1993, and USPHS, 1991.

V. In your letter, you claim "the majority of animal studies³ have shown no effect on cancer, birth defects, genetic disorders, or bone strength".

I have enclosed a list of 10 animal studies that show that fluoride initiates tumors and/or cancer and/or promotes tumor growth rate and/or increases the cancer-causing potential of other carcinogens. Can you come up with 11 negative animal studies?

I have enclosed a list of 2 animal studies that show that fluoride has an effect on birth defects. Can you come up with 3 negative animal studies?

I have enclosed a list of 22 animal studies that show that fluoride causes genetic disorders. Can you come up with 23 negative animal studies? In your

own publication, NRC, 1993, on Table 6-2, you list six in vitro animal studies, five of which deal with the effect of fluoride on genetic disorders (aberrations) and all five of which found that fluoride exposure caused chromosomal damage. In table 6-6, you list four in vivo studies which deal with the effect of fluoride on genetic disorders (aberrations), two of which are listed as showing positive results and two of which are listed as showing negative results.⁴

While I don't have a large number of animal studies regarding the adverse effects of fluoride on bone strength of very high and long-term fluoride exposures, I do have a large number of human clinical and epidemiological studies, for a total of 34 references. Can you provide 35 negative studies?

VI. At this point, it must be obvious how preposterous is the statement made in your letter: "Thus, the primary adverse effects associated with chronic excess fluoride intake are [moderate] enamel fluorosis in children through 8 years of age and [crippling] skeletal fluorosis in adolescents and adults over 8 years of age."

But this statement not only covers up the well-documented findings that fluoride causes and/or promotes tumors and cancers as well as possibly enhancing the cancer-causing effects of other carcinogens⁵ and that fluoride causes genetic damage down to and including the levels proposed in your "adequate intake" recommendations, it also claims that you can recommend safe upper levels where children and adults can suffer from dental and skeletal fluorosis so long as the damage does not reach the level of moderate or crippling. Borrowing a word from Dr. Limeback - "shame!"

To make matters worse, the upper level for fluoride suggested, i.e. 2.2 mg per day for children 4-8 years of age, does not preclude moderate-to-severe dental fluorosis. Your own publication, NRC, 1993, table 2-5 shows that about 5-10% of these children will suffer from moderate-to-severe dental fluorosis.

It is astounding that in your letter, you never indicate what skeletal fluorosis is. Skeletal fluorosis simply refers to changes in the bone or tissues associated with the bone that result from the poisoning of fibroblasts, chondroblasts, and osteoblasts by fluoride.⁶ One form of skeletal fluorosis is osteoporosis, which can manifest itself in the form of increased hip fracture rates; and, at levels of about 1 mg/l in the drinking water, an increase in hip fracture rates has been reported in no less than four papers (two articles and two letters) published in JAMA since 1990.

By disturbing the protein extruded by blastic cells, fluoride can trigger autoimmune responses leading to rheumatoid arthritis (which can be crippling but will not be detectable in x-rays) or induce the calcification of ligaments, tendons, and cartilage which will only be observable in x-rays in the later stages. Both osteosclerosis and osteoid seams can be triggered by fluoride. None of the outdated studies (1941, 1954, 1955, 1957) cited by Gary Whitford are capable of detecting skeletal fluorosis even if they had been done today, due to lack of a control group in one, lack in numbers in three, and exclusive use of x-rays in one. On pages 50-53 of my book, *Fluoride, the Aging Factor*, I describe docu-

mented cases of skeletal fluorosis occurring among people consuming water containing fluoride at levels of 0.7 mg/l to 9.4 mg/l.⁷

Your Dietary Reference Intake values (DRIs) for calcium, phosphorus, magnesium, vitamin D, and fluoride are published in *Nutrition Today*, volume 32, pages 182-188. Looking at the mg/kg/day values of each of these substances relative to their 0-6 month value, each DRI goes down or stays the same after maturity, except for fluoride, which in contrast goes up about 3000%! Why? Just to sanctify fluoridation.

Sincerely,

John Yiamouyiannis, Ph.D.
President

January 19, 1999

FOOTNOTES

- 1 See, for example, Yoshitsugu Imai, "Relation Between Fluoride Concentration in Drinking Water and Dental Caries in Japan", *Koku Eisei Gakkai Zasshi*, Volume 22, No. 2, pp. 144-196 (1972); R.M. Bell, et al., "Results of baseline dental exams in the national preventive dentistry demonstration program". R-2862-RWJ. Santa Monica, CA. Rand Corporation. 1982; J. Colquhoun, "Child Dental Health Differences in New Zealand", *Community Health Studies*, Volume 11, pp. 85-90 (1987). A.S. Gray, "Fluoridation: Time for a New Baseline?" *Journal of the Canadian Dental Association*, Volume 53, pp. 763-765 (1987); C.F. Hildebolt, et al., "Caries Prevalences among Geochemical Regions of Missouri", *American Journal of Physical Anthropology*, Volume 78, pp. 79-92, (1989); J.A. Yiamouyiannis "Water Fluoridation and Tooth Decay: Results from the 1986-1987 National Survey of U.S. Schoolchildren", *Fluoride* 1990; 23: 55-67.
- 2 Although there is good evidence that the decay rate of deciduous teeth is lower among 5-year-olds in fluoridated areas, this reduced tooth decay rate rapidly disappears among 6- and 7-year-olds and is no longer apparent in 8-year-olds. This temporary reduction may be due to the inhibitory effect of fluoride on tooth eruption which has already been reported (see for example S. S. Krylov and K. Pemrolyd, "Deciduous tooth eruption and fluorosis in the case of increased fluorine content in the drinking water", *Stomatologiia*, (Mosk) Volume 61, pp. 75-77 (1982)). In any event, there is no reason to believe that the intake of fluoride beyond the age of five would have any effect on the tooth decay rate.
- 3 I assume that you are referring to original studies (not reviews) that looked into these matters and are not just playing word games. It is obvious that there are hundreds of thousands of animal studies that have not found that fluoride causes e.g. genetic damage because there are hundreds of thousands of animal studies that have not looked at genetic damage and hundreds of thousands of animal

studies that have not looked at fluoride. I mention this only because when the National Cancer Institute was asked, during Congressional Hearings on fluoride and cancer in 1977, to produce studies showing that fluoride did not cause cancer, 11 of the 13 references they produced did not even deal with fluoride and cancer.

- 4 One of the two studies you claim shows negative results is Voroshilin, 1975, which is an apparent translation of his original paper in 1973, which I had translated. But even in the 1973 paper, the original English summary points out: "An increase from 1.24 (in the control) to 6.5% in the frequency of cells with structural chromosomal aberrations was observed in the bone marrow of albino rats after 5 months treatment with cryolite [at a 3.0 mg/m³ concentration. After a mixture of 0.5 mg/m³ of cryolite and 0.5 mg/m³ of hydrogen fluoride for five months, the increase was from 1.24 (in the control) to 5.9%. For comments on the second paper by Martin, 1979, see the footnote on page 66 of my book, Fluoride, the Aging Factor (enclosed).
- 5 C.A. Jones, et al., "Sodium fluoride promotes morphological transformation of Syrian hamster embryo cells", *Carcinogenesis*, Volume 9, pp. 2279-2284 (1988) and supported by the findings of R.N. Mukherjee and F.H. Sobels, The effect of fluoride and iodoacetamide on mutation by X-irradiation in mature Spermatzoa of *Drosophila*, *Mutation Research*, volume 6, 217-225 (1968).
- 6 not to mention secondary effects on bone that fluoride may have through its effects on the parathyroid.
- 7 Please explain to me what you mean in your final conclusion "...intakes associated with water fluoride concentrations in excess of 10 ppm do not cause clinically significant skeletal fluorosis."

NOTE: The foregoing letter has not received a reply or even an acknowledgement.