STATEMENT OF JAY HARRIS LEVY, D.D.S.
301 NE 65th Avenue
Portland, OR 97213
503-261-0916
jaylevy@easystreet.net

BEFORE THE PORTLAND CITY COUNCIL
City Hall - 1221 SW Fourth Avenue

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My name is Dr. Jay Levy. I've practiced dentistry for 30 years and have taught at NYU and OHSU dental schools. I trained as a postdoctoral fellow in neurophysiology and performed neurophysiologic research at OHSU. I have worked in public health dental clinics and treated children and adults with rampant tooth decay. Believe me, if fluoridation would actually improve public health I'd be the first to stand behind it.

I will address three key questions today.

Is fluoridation effective?

Conceived as a ten-year study to compare tooth decay rates in fluoridated and non-fluoridated cities the Grand Rapids-Muskegon trial is frequently cited by fluoridation promoters. However, the study was severely compromised when non-fluoridated Muskegon started fluoridating their water five-years into the trial. (1) This poorly designed study made no attempt to control for differences in socioeconomics. In fact, no statistical analysis was used at all. Declines in tooth decay in both cities mirrored national and international declines unrelated to fluoridation. I refer you to the paper "The Mystery of Declining Tooth Decay Rates" in the respected scientific journal Nature. (2)

In over fifty years of monitoring, the Newburgh-Kingston trial has failed to show an overall significant difference in tooth decay rates between these fluoridated and non-fluoridated cities. Unfortunately, fluoridated Newburgh has a significantly higher rate of dental fluorosis. (3,4)

Dental fluorosis is the visible manifestation of toxic overexposure to fluoride during developmental years. Severity ranges from white specs to pitted, dark brown stains in tooth enamel. Fluorotic enamel is brittle and prone to decay. Fluorosis rates in US teens have reached an astounding all time high of 41%, indicating that they are already ingesting high levels of fluoride from foods, bottled beverages and toothpaste. (5)

Commissioned by the British Health Department, the York Systematic Review of the fluoride literature was charged to "carry out an up to date expert scientific review of fluoride and health." It concluded: (6)

- "Given the level of interest surrounding the issue of public water fluoridation, it is surprising to find that little high quality research has been undertaken."
- "The failure of these studies to deal with potential confounding factors or to provide standard error data means that the ability to answer the objective is limited."

What is the quality of the fluoride used in fluoridation?

• 10% is medical grade.
• 90% is a toxic waste product of the phosphate fertilizer industry containing Hexafluorosilicic acid, arsenic, lead and cadmium. (7)
• According to EPA scientist Dr. William Hirzy: "If this stuff gets out into the air, it's a pollutant; if it gets into the river, it's a pollutant; if it gets into the lake it's a pollutant; but if it goes right into your drinking water system, it's not a pollutant…" (8)

Is fluoridation safe?

• 7000 EPA scientists and professional workers do not think so and called for a moratorium on all drinking water fluoridation programs.
• They have asked EPA management to recognize fluoride as posing a serious risk of causing cancer, neurotoxicity and reduced IQ. (9)

Dr. Arvid Carlsson who won the Nobel Prize in Medicine and Physiology noted that: "fluoridation is against all modern principles of pharmacology. It's obsolete. I don't think anybody in Sweden, not a single dentist, would bring up this question anymore." (10)

Thank you for your consideration.

CITATIONS

1. Grand Rapids Fluoridation Study: Results Pertaining to the Eleventh Year of Fluoridation. Arnold Jr., F.A. Am J Pub Health 5 539-545 (1957)

2. The Mystery of Declining Tooth Decay Rates. Diesendorf, M. Nature Jul 10-16 322(6075) 125-129 (1986)

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

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

5. Prevalence and severity of dental fluorosis in the United States, 1999-2004. Beltrán-Aguilar ED, Barker L, Dye BA. NCHS Data Brief. Nov;(53):1-8 (2010)

6. A systematic review of public water fluoridation. McDonagh M, Whiting P, Bradley M et al. York, Report number 18 University of York. (2000)

7. Hexafluorosilicic acid (hydrofluorosilicic acid), raw materials, manufacture, toxicity and public health concerns as an active ingredient in fluoridation of drinking water. Enviro Management Services. http://www.enviro.ie/downloads.html September (2012)

8. Statement of Dr. J. William Hirzy National Treasury Employees Union Chapter 280 before the subcommittee on wildlife, fisheries and drinking water. United States Senate June 29 (2000)

9. EPA Unions Call for Nationwide Moratorium on Fluoridation, Congressional Hearing on Adverse Effects, Youth Cancer Cover Up. Hirzy, J.W. NTEU Chapter 280 US EPA, National Headquarters August 19 (2005)

10. Water fluoridation "obsolete" according to nobel prize scientist. Connett, M. and Neurath, C. Fluoride Action Network http://www.fluoridealert.org/content/carlsson-interview/ October (2005)

The mystery of declining tooth decay

from Mark Diesendorf

Large temporal reductions in tooth decay, which cannot be attributed to fluoridation, have been observed in both unfluoridated and fluoridated areas of at least eight developed countries over the past thirty years. It is now time for a scientific re-examination of the alleged enormous benefits of fluoridation.

FLUORIDATION consists of raising the concentration of the fluoride ion F^- in water supplies to about 1 part per million (p.p.m.) with the aim of reducing dental caries (tooth decay) in children. In fluoridated areas, there are now many longitudinal (temporal) studies which record large reductions in the incidence of caries[1]. The results of these and of fixed time surveys have led to the 'fluoridation hypothesis', namely that the principal cause of these reductions is fluoridation.

Until the early 1980s, there had been comparatively few longitudinal studies of caries in unfluoridated communities. Only a small minority of the studies in fluoridated areas had regularly examined control populations, and there seemed to be little motivation to study other unfluoridated communities. But during the period 1979–81, especially in western Europe where there is little fluoridation, a number of dental examinations were made and compared with surveys carried out a decade or so before. It soon became clear that large reductions in caries had been occurring in unfluoridated areas (see below). The magnitudes of these reductions are generally comparable with those observed in fluoridated areas over similar periods of time.

In this article, these reductions are reviewed and attention is also drawn to a second category of caries reduction which cannot be explained by fluoridation. This category is observed in children described by proponents of fluoridation as having been 'optimally exposed', that is, children who have received water fluoridated at about 1 p.p.m. from birth. The observation is that caries is declining with time in 'optimally exposed' children of a given age. In some cases, the magnitudes of these reductions are much greater in percentage terms than the earlier reductions in the same area which had been attributed to fluoridation.

The problem of explaining the two categories of reduction goes well beyond the field of dentistry: contributions from nutritionists, immunologists, bacteriologists, epidemiologists and mathematical statisticians, amongst others, may be required.

Table 1 Studies reporting large reductions in dental caries in unfluoridated areas

Location		Years surveyed	References
Australia	Brisbane	1954, '77	2, 3
	Sydney	1961, '63, '67	4
Denmark	Various towns	1972, '79	53
Holland	The Hague	1969, '72, '75, '78	38
	Various towns	1965, '80	11
New Zealand	Auckland (parts)	1966, '74, '81	12
Norway	Various towns	1970, '80	54
Sweden	Various towns	1973, '78, '81	39
	North Sweden	1967, '77	55
United Kingdom	Bristol	1970, '79	56
	Bristol	1973, '79	56
	Devon	1971, '81	37
	Gloucestershire	Annually from 1964	37*
	Isle of Wight	1971, '80	57
	North-West England	1969, '80	58
	Scotland	1970, '80	59
	Shropshire	1970, '80	10
	Somerset	1975–79 annually	60
	Somerset	1963–79	61
United States	Dedham, Mass.	1958, '74	40
	Norwood, Mass.	1958, '72, '78	40
	Massachusetts: sample of schools	1951, '81	41
	Ohio	1972, '78	62

* Unpublished communication from J. Tee (1980), Area Dental Officer, Gloucestershire, to R. J. Anderson *et al.*[37]

Caries in unfluoridated areas

Table 1 lists over 20 studies which report substantial temporal reductions in caries in children's permanent teeth in unfluoridated areas of the developed world. In many of these cases, the magnitudes of these reductions are comparable with those observed in fluoridated areas and attributed to fluoridation.

Several of these studies give clues as to factors which are unlikely to be the main causes of the reductions. A comparison of the 1954 and 1977 dental health surveys in Brisbane[2,3] indicates to a reduction of about 50% in caries, as measured by the number of decayed, missing and filled permanent teeth (DMFT) per child and averaged over the age groups, in the 23-year period. The 1977 survey distinguished between children who took fluoride tablets regularly, irregularly or not at all. Although there were differences in caries incidences between the three categories (which could reflect factors unrelated to fluoride levels), even the "no tablet" group had on average 40% less caries experience than that recorded in 1954. So fluoride tablets were not the principal cause of the reductions observed in Brisbane.

The first Sydney study[4] showed that children with "naturally sound" teeth increased from 3.8% in 1961 to 20.2% in 1967 and 28% in 1972. The paper, which was titled enthusiastically "The Dental Health Revolution", was originally used widely to promote fluoridation in Australia. The authors stated that: "Almost certainly, the availability of fluoride both in tablet form and delivered through town water supplies has been the predominant factor. . . . These very large reductions represent a modern triumph of preventive health care"[4]. Yet the major proportion of the reported improvement had already occurred before Sydney was fluoridated in 1968. Moreover, no evidence was presented that fluoride tablets were widely used in the 1960s. Fluoride toothpaste was only introduced into Australia in 1967[3]. Although the index "naturally sound" teeth is unsuitable for more detailed

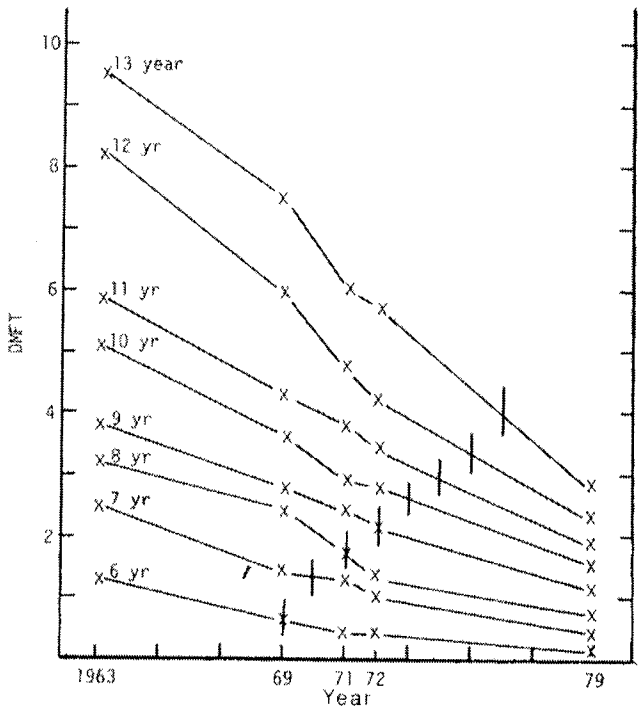

Fig. 1 Decline in caries, as measured by DMFT, in Tamworth, Australia, for children in age groups 6 years to 13 years. Data compiled from refs 14, 15. The vertical line cutting graph for each age group denotes year at which maximum possible benefit from fluoridation was reached. Tamworth was fluoridated in 1963.

studies which distinguish decayed, missing and filled teeth, the populations examined were very large (over 9,000 children at each examination) and the results clear-cut.

A second Sydney study[5] used the DMFT index, but was irrelevant for establishing any link with fluoridation, since it reported only on examinations in 1963 and 1982, but not around 1968 when Sydney was fluoridated. As in several other fluoridation studies, the key data were either not collected or not reported[6]. Although the two Sydney papers have an author in common (James S. Lawson, a senior officer of the New South Wales Health Commission), the second paper does not even cite the first. This suggests that, once it became clear that the first Sydney study contained evidence unfavourable to fluoridation, it was a source of embarrassment to some fluoridation proponents who are apparently trying to denigrate it.

However, independent confirmation of the large reductions in caries before fluoridation reported in the first Sydney study[4] is readily obtained by comparing the results of two surveys[7,8] separated by 20 years by Barnard. These surveys showed that the mean DIMF index ('I' denotes a permanent tooth which cannot be restored) for school children aged 13 and 14 declined from 11.0 in 1954–55 to 6.0 in 1972. The four years from 1968, when fluoridation commenced in Sydney, to 1972, would not have contributed significantly to the decline in caries prevalence in this age group[9].

The authors of one of the British studies[10] cited in Table 1 point out that sales of fluoride toothpaste in the United Kingdom were less than 5% of total sales in 1970, but rose to more than 95% of sales in 1977. They quote unpublished annual data from unfluoridated parts of Gloucestershire, collected from 1964 onwards, which show substantial improvements in children's teeth before the use of fluoride toothpaste became significant.

Many of the studies in the Netherlands, reviewed by Kalsbeek[11], were carried out to evaluate the effectiveness of the school dental health programme. Temporal reductions in DMFT of about 50% occurred between 1970 and 1980, whether or not the children had taken part in the dental health education program. Kalsbeek also reviewed the use of fluoride tablets and toothpaste and concluded from the data that "factors other than the effects of different fluoride programmes must play a role."

The study in the partly fluoridated city of Auckland, New Zealand[12], examined the influence of social class (which reflects environmental and lifestyle factors, such as diet) as well as fluoridation on dental health as measured by the levels of dental treatment received by children. The paper showed that treatment levels have continued to decline in both fluoridated and unfluoridated parts of the city and that these reductions are related strongly to social class, there being less caries in the "above average social rank" group than in other children. Thus the main ethical argument for fluoridation, that it should assist the disadvantaged, is not borne out by this study.

Fluoridation's benefits

On 15 December 1980, the Dental Health Education and Research Foundation, one of the main fluoridation promoting bodies in New South Wales (NSW), issued a press release entitled, "Fluoridation dramatically cuts tooth decay in Tamworth"[13]. This document, which highlighted results of a study conducted by the Department of Preventive Dentistry, Sydney University, and the Health Commission of NSW, stated in part:

> Tamworth's water supply was fluoridated in 1963, and the last survey in the area was conducted in August 1979. It shows decay reductions ranging from 71% in 15-year-olds to 95% in 6-year-olds. . . . All those surveyed were continuous residents using town water.

The "95%" reduction actually corresponded to a reduction in DMFT from 1.3 in 1963 to 0.1 in 1979[14], which is 92%. The press release implied incorrectly that all this reduction was due to fluoridation. However, it has been claimed ever since

Table 2 Extent of fluoridation in Australia, 1977 and 1983

State or territory	Capital city	Year city fluoridated*	% Of state fluoridated† in 1977	% Of state fluoridated† in 1983
ACT	Canberra	1964	100	100
Tasmania	Hobart	1964	74	77
NSW	Sydney	1968	81	81
WA	Perth	1968	83	83
SA	Adelaide	1971	71	70
Victoria	Melbourne	1977	0.7 then 73	71
Queensland	Brisbane	Not fluoridated	10	5

* Each capital city has the majority of the population of its state or territory.
† That is, the percentage of population of state/territory which drinks fluoridated water. Data from Annual Reports of Director-General of Health, for example ref. 17.

the commencement of fluoridation that the maximum possible benefits from fluoridation are obtained in children who have drunk fluoridated water from birth. Six-year-olds would have done this by 1969, when, according to the published data[15], they had a DMFT index of 0.6. The further reduction in caries in optimally exposed 6-year-olds, observed in years following 1969, cannot be due to fluoridation.

Thus, one can say that at best fluoridation could have approximately halved the DMFT rate in 6-year-olds between 1963 and 1969. (Since there was no control population, one could also say that at worst fluoridation might have had no effect in that period.) But from 1969 to 1979, caries in 6-year-olds was reduced a further 83%, by some other factor(s) than fluoridation.

Figure 1 shows that the unknown factors caused in children of each age from 6 years to 9 years similar large reductions in caries. Unfortunately, there are no published data for Tamworth beyond 1979 or in the years between 1972 and 1979, and so it cannot be confirmed whether the large reductions observed[14,15] from 1972 to 1979 in children aged 10 to 15 were also due to these unknown factors.

A similar reduction beyond the maximum possible for fluoridation is observed for children of each age from 6 to 9 in the published data from Canberra[16], which cover the period from 1964, the stated year of fluoridation, to 1974. In particular, DMFT rates declined by 50% in 6-year-olds from 1970 to 1974 and by 54% in 7-year-olds from 1971 to 1974. These reductions in optimally exposed children cannot be due to fluoridation. Published post-1974 data are needed to check on further reductions in optimally exposed children aged over 9 years.

From 1977 onwards, data have been systematically collected from the school dental services in each Australian state and territory[9,17]. Table 2 shows the degree of fluoridation in each of these states/territories in 1977 and 1983 and also the dates of fluoridation of the capital cities of these regions. Each of these cities dominates the population of the state or territory in which it lies. The evidence presented in Fig. 2 and Table 2 suggests that states and territories which had been extensively fluoridated for at least 9 years before 1977 (Tasmania, Western Australia and New South Wales) had qualitatively similar large reductions in caries from 1977 to 1983 as a state which was only extensively fluoridated in 1977 (Victoria) and a state which had a small and declining fraction of fluoridation (Queensland). Although the results of the school dental health survey are recorded by age and state, the data have only been published[9,17,18] so far for ages 6–13 averaged in each state, or for each age for the whole of Australia. There is evidence that the use of fluoride toothpaste in Australia reached a high plateau around 1978, so these observed reductions in caries can be due neither to fluoride toothpaste[9] nor to fluoridated water.

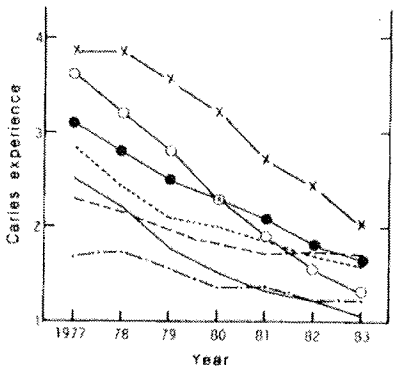

Fig. 2 Decline in the average number of (permanent) teeth per child with caries experience in each Australian state and the Australian Capital Territory as observed in school dental services[17]. 'Caries experience' can be one or more decayed, missing or filled teeth, and consists of an average for children aged 6–13 years. See Table 2 for information on the extent of fluoridation in each state/territory in 1977 and 1983 and the year when the main population centre of each state/territory was fluoridated. ×, Victoria; ○, Tasmania; ●, Queensland; – – – –, SA; ———, NSW; ——, WA; ·—·—·, ACT.

It is to be hoped that similar data on caries reductions in "optimally exposed" children will be sought in other fluoridated countries. In a region of Gloucestershire, United Kingdom where the main water supply was naturally fluoridated with 0.9 p.p.m. fluoride until 1972, reductions in caries of 51% were observed in 12-year-old children between 1964 and 1979[19]. Factors other than fluoridated water must have caused these reductions. After 1972, the main water supply was drawn from a bore with less than 0.2 p.p.m. fluoride, so a recent survey of caries there would be of great interest.

Benefits overestimated?

In some fluoridated areas (for example Tamworth, Australia), temporal reductions in caries have been wrongly credited to fluoridation. The magnitude of these reductions is similar in both fluoridated and unfluoridated areas, and is also generally comparable with that traditionally attributed to fluoridation. Can it be concluded that communities which prefer not to fluoridate, either because of concern about potential health hazards[20–25] or for ethical reasons (for example compulsory medication; medication with an uncontrolled dose), do not necessarily face higher levels of tooth decay than fluoridated communities? In other words, is it reasonable to ask whether it could be generally true that a major part of the benefits currently attributed to fluoridation is really due to other causes?

Such a hypothesis would seem to be possible in principle because it is well known that fluoridation is neither 'necessary' nor 'sufficient' (the words between inverted commas being used in the formal logic sense) for sound teeth; that is, some children can have sound teeth without fluoridation, and some children can have very decayed teeth even though they consume fluoridated water[25].

To confirm or refute the hypothesis, it is necessary (but not 'sufficient') to examine the absolute values of caries prevalence in fluoridated and unfluoridated areas. If it is true that the absolute values of caries prevalence in some unfluoridated areas are comparable with those in some unfluoridated areas of the same country, then the hypothesis is supported (but not proven), and there would be a strong case for the scientific re-examination of the epidemiological studies which appear to demonstrate large benefits from fluoridation.

The earliest set of studies comparing caries in fluoridated and unfluoridated areas were time-independent surveys of caries prevalence in areas with 'high' natural levels of fluoride in water supplies, conducted by H. T. Dean and others in the United States[26]. The surveys purported to show that there is an "inverse relationship" between caries and fluoride concentration. From the viewpoint of modern epidemiology, these early studies were rather primitive. They could be criticized for the virtual absence of quantitative, statistical methods, their nonrandom method of selecting data and the high sensitivity of the results to the way in which the study populations were grouped[25].

Results running counter to the alleged inverse relationship have been reported from time-independent surveys in naturally fluoridated locations in India[27], Sweden[28], Japan[29], the United States[30] and New Zealand[31,63]. The Japanese survey[29] found a minimum in caries prevalence in communities with water F-concentrations in the range 0.3–0.4 p.p.m.; above and below this range, caries prevalence increased rapidly.

These surveys[27–31] also selected their study regions nonrandomly. But recently Ziegelbecker[32] attempted to make a selection close to a random sample by considering 'all' available published data on caries prevalence in naturally fluoridated areas. His large data set, which includes Dean's as a sub-set, comprises 48,000 children aged 12–14 years drawn from 136 community water supplies in seven countries. He found essentially no correlation between caries and log of fluoride concentration. The surveys[27–32] are generally omitted from lists[1] of studies on the role of fluoridation in caries prevention.

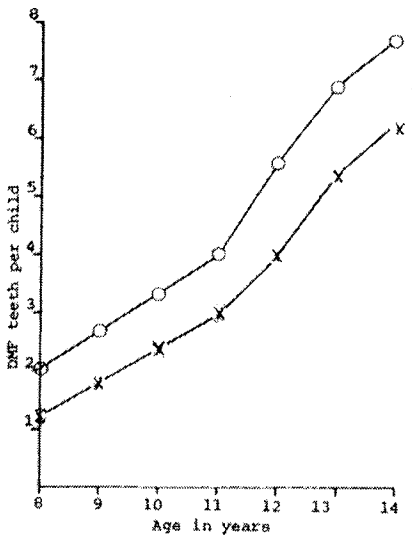

Fig. 3 The variation with age of decayed, missing and filled permanent teeth (DMFT) in fluoridated test towns (×) and unfluoridated control towns (○) in Britain, graphed from data published by the UK Department of Health[33]. Note that the rate of increase of DMFT is essentially the same in both groups. Children in the fluoridated areas have an average only one less cavity than children of the same age in the unfluoridated areas.

Further evidence can be drawn from Fig. 2. In 1983, the absolute value of caries prevalence in the Australian state of Queensland (which is only 5% fluoridated) was approximately equal to that in the states of Western Australia (83% fluoridated) and South Australia (70% fluoridated).

The classical British fluoridation trials at Watford and Gwalchmai were longitudinal controlled studies. In this regard they were better designed than the majority of other studies which have been conducted around the world. However, as in the case of almost all other surveys, the examinations were not 'blind'. The review of the British trials by the UK Department of Health after 11 years of fluoridation showed that children in fluoridated towns had approximately one less DMFT (that is, essentially one less cavity) than children of the same age in unfluoridated towns (see Fig. 3). The rate of increase in caries with age was the same in both populations[33].

Thus there are a number of counter-examples to the widely-held belief that "All studies show that communities where water contains about 1 p.p.m. fluoride have about 50% lower caries prevalence than communities where water has much less than 1 p.p.m. fluoride".

At this point the empirical data presented here may be summarized as follows. In the developed world:

(1) there have been large temporal reductions in caries in unfluoridated areas of at least eight countries;

(2) there have been large temporal reductions in several fluoridated areas which cannot be attributed to fluoridation;

(3) the absolute values of caries prevalence in several fluoridated areas are comparable with those in several unfluoridated regions of the same country.

Hence there is a case for scientific re-examination of the experimental design and statistical analysis of those studies which appear to prove or "demonstrate" that fluoridation causes large reductions in caries. Indeed the few re-examinations which have already been done confirm that there are grounds for concern.

The original justification for fluoridation in the United States, Britain, Canada, Australia, New Zealand and several other English-speaking countries was based almost entirely on the North American studies, which were of two kinds. The limitations of the first set, the time-independent surveys conducted in naturally fluoridated areas of the United States[26], have been referred to above.

The second set of North American studies consists of five longitudinal studies—carried out at Newburgh, Grand Rapids, Evanston and Brantford (two studies)—which commenced in the mid-1940s. Only three of them had controls for the full period of the study. These studies were criticized rigorously in a detailed monograph by Sutton[34], on the grounds of inadequate experimental design (for example, no 'blind' examinations and inadequate baseline measurement), poor or negligible statistical analysis and, in particular, failure to take account of large variations in caries prevalence observed in the control towns. The second edition of Sutton's monograph contains reprints of replies by authors of three of the North American studies and another author, together with Sutton's comments on these replies. It is difficult to avoid the conclusion that Sutton's critique still stands. Indeed, this was even the view of the pro-fluoridation Tasmanian Royal Commission[35]. Yet, in major, recent reviews of fluoridation, such as that by the British Royal College of Physicians[36], these North American studies are still referred to as providing the foundations for fluoridation, and Sutton's work[34] is not cited.

An examination has just been completed of the experimental design of all of the eight published fluoridation studies conducted in Australia. One (Tasmania) is a time-independent survey. Four (Townsville, Perth, Kalgoorlie and the second Sydney study) are longitudinal studies with only two examinations of the test group and either no control or only a single examination of a comparison group. The remaining three studies (Tamworth, Canberra and the first Sydney study) have several examinations of the test group, but no comparison group at all. Thus there has not been a single controlled longitudinal study in Australia. (M.D., to be published). Moreover, it has been shown above that three of the Australian studies (the first Sydney[4], Tamworth[14,15] and Canberra[16]) inadvertently provide evidence that some other factor(s) than fluoridation is/are playing an important role in the decline of caries prevalence.

Hence the hypothesis that fluoridation has very large benefits requires re-examination by epidemiologists, mathematical statisticians and others outside of the dental profession. The danger of failing to perform scientific research on the mechanisms underlying the large reductions in caries discussed in this paper is that the strong emphasis on fluoridation and fluorides may be distracting attention away from the real major factors. These factors could actually be driving a cyclical variation of caries with time[37]. It is possible that the condition of children's teeth could return to the poor state observed in the 1950s, even in the presence of a wide battery of F-treatments.

Causes of caries reductions

Many of the authors who reported the reductions in unfluoridated areas acknowledged that the explanation has not yet been determined scientifically[11,37-41]. It is after all much easier to perform a study which measures temporal changes in the prevalence of a multifactorial disease than to identify the causes of such changes.

Nevertheless, the authors of some of these studies have speculated that important causes of the reductions which they observe might be topical fluorides[38,53] (such as in toothpastes, rinses and gels), fluoride tablets[4,38], school dental health programmes[9], a lower *frequency* of sugar intake[39], the widespread use of antibiotics which may be suppressing *Streptococcus mutans* bacteria in the mouth[41], the increase in total fluoride intake from the environment[9,42], or a cyclical variation in time resulting from as yet unknown causes[37].

The present overview has revealed that several of the studies contain evidence against some of these proposed factors. We have seen that the Brisbane study[3] and

the Dutch review[11] suggest that fluoride tablets may not be important; the Sydney study[4], one of the British studies[10] and the Dutch review[11] each provides evidence against fluoride toothpaste; and the Dutch review[11] found no benefit in their school dental health education programmes.

Although there is evidence that fluoride toothpaste cannot be an important mechanism of caries reduction in some of the studies reported here, it must be stated that, unlike the case of fluoridation, there are also a few well-designed randomised controlled trials which demonstrate substantial reductions in caries from fluoride toothpaste[43]. Hence, the hypothesis can be made that topical fluorides sometimes improve children's teeth, although they are not necessary. So topical fluorides may comprise one of several factors contributing to the solution of the scientific problem of explaining the reduction in tooth decay.

Leverett[42] has speculated that the caries reductions in his smaller set of unfluoridated locations may be due to "an increase in fluoride in the food chain, especially from the use of fluoridated water in food processing, increased use of infant formulas with measurable fluoride content, and even unintentional ingestion of fluoride dentifrices." This hypothesis cannot explain the reductions in prefluoridation Sydney[4], or those in unfluoridated parts of Gloucestershire which started in the late 1960s[10]. The ingestion of fluoride toothpastes (and gels) by young children is well documented and could account for an intake of about 0.5 mg F^- per day in the very young[44]. But the food processing pathway is unlikely to be significant in western Europe where there is hardly any fluoridation, and infant formulas which are made up with unfloridated water will give only small contributions. Thus it appears that Leverett's hypothesis may at best be relevant to a minority of the studies listed in Table 1.

Here, the working hypothesis is presented that fluoridation and other systemic uses of fluoride, such as fluoride tablets, have at best a minor effect in reducing caries; that the main causes of the observed reductions in caries are changes in dietary patterns, possible changes in the immune status of populations and, under some circumstances, the use of topical fluorides. Indeed, a promising explanation is that the apparent benefit from fluorides is derived from their topical action. Then, since fluoridated water has a fluoride ion concentration 10^{-3} times that of fluoride toothpaste, its action in reducing caries is likely to be much weaker.

It is known that immunity plays a role in the development of caries, as it does with other diseases. Research is currently in progress to try to develop a vaccine against caries[45-47]. None of the data presented in the present paper provides evidence against immunity as a factor.

Dentists often argue against changes in dietary patterns as a major factor, on the grounds that sugar consumption has remained approximately constant in most developed countries over the past few decades. However, this is a simplistic argument. First, crude industry figures on total sales of sugar in developed countries contain no information on the distribution of sugar consumption with age and time of day. The form of sugar ingested—for example in canned food, soft drinks or processed cereals—may also be important. Second, tooth decay is increasing together with increases in sugar and other fermentable carbohydrates in the diet in several developing countries[48,49]. This was also the case with Australian aborigines, even when their water supplies consisted of bores containing fluoride at close to the "optimal" concentration for the local climate[50,51]. Third, there is more to diet than sugar. For instance, there is some evidence, even conceded occasionally by pro-fluoride bodies[52], that certain foods which do not contain fluorides (for example wholegrain cereals, nuts and dairy products) may protect against tooth decay. So the whole question of the relationship between total diet and tooth decay needs much greater input from nutritionists and dietitians.

Perhaps the real mystery of declining tooth decay is why so much effort has gone into poor quality research on fluoridation, instead of on the more fundamental questions of diet and immunity.

The main body of this research was performed while the author was a principal research scientist in the CSIRO Division of Mathematics and Statistics, Canberra. □

Mark Diesendorf is at the Human Sciences Program, Australian National University, GPO Box 4, Canberra ACT 2601, Australia.

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Enviro Management SERVICES

HEXAFLUOROSILICIC ACID

(HYDROFLUORSILICIC ACID)

Raw Materials, Manufacture, Toxicity and Public Health Concerns As an Active Ingredient in Fluoridation of Drinking Water

Prepared by:
Declan Waugh B.Sc. C.Env. MCIWEM. MIEMA. MCIWM
Member Chartered Institution of Water and Environmental Management
Member Chartered Institute of Waste Management
Member Institute of Environment Management and Assessment

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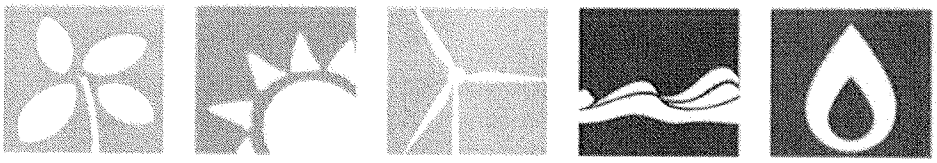
Any enquiries relating to this report should be referred to the author at the following email address: declan@enviro.ie
Tel: 023-8841933
Web: www.enviro.ie



HEXAFLUOROSILICIC ACID

CAS No: 16961-83-4

MOLECULAR: (H_2SiF_6)

Synonyms: Hydrofluorosilicic Acid, Hydrofluosilicic Acid, Hydrtosilicofluodie Acid, Fluorosilicic Acid, Silicofluoric Acid, Fluosilicic Acid

Raw materials

Calcium fluoride, Hydrofluoric acid, Silicon dioxide, Sulphuric acid, Celite

Preparation Products

Ammonium hexafluorosilicate, Sodium fluoroaluminate, Magnesium fluorosilicate, Potassium tetrafluoroborate, Potassium fluoride, Sodium tetrafluoroborate, MAGNESIUM HEXAFLUOROACETYLACETONATE DIHYDRATE, Chromic acid, Potassium fluorosilicate, Magnesium fluosilicate, Magnesium hexafluorosilicate hexahydrate, Sodium tripolyphosphate, Aluminium fluoride, Sodium fluorosilicate, CUPRIC FLUOROSILICATE, Trisodium hexafluoroaluminate, Ammonium fluoborate, Sodium fluoride, ZINC SILICOFLUORIDE, Lead.[1]

FLUORSPAR AND WATER FLUORIDATION CHEMICALS

Fluorspar (CaF2) is the most important fluoric containing mineral. About 52% of fluorspar consumption worldwide is used as starting material for the production of hydrofluoric acid; another 18% is used for aluminium fluoride, the fluxing agent in the aluminium industry; and 25 % for the steel industry as a flux to improve the fluidity of slag in steelmaking. Fluorspar is the commercial name for the mineral fluorite (calcium fluorite) and it is a major raw material source of fluorine. 25% of the fluorspar consumption of the European Union is produced by EU member states, mainly by Spain. A much larger amount is imported from states like China.[2]

Fluorspar deposits are primarly a byproduct of precious and base metal ores, such as lead, silver or zinc. Fluorspar deposits vary in mineral composition and are not pure. They contain large quantities of silica. Small quantities of rare earth elements (REE), strontium and other elements may substitute for calcium within the fluorspar crystal structure. Fluorspar is used directly or indirectly to manufacture such products as aluminium, gasoline, insulating foams, refrigerants, steel, and uranium fuel. It is used in the manufacture of Fluorocarbon chemicals, including fluoropolymers, chlorofluorocarbons (CFC's) , hydrochlorofluorocarbons (HCFC's), and hydrofluorocarbons (HFC's). CFC's, HCFC's, HFC's and Hydrofluoric acid (HF).

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[1] http://www.chemicalbook.com/ChemicalProductProperty_EN_CB3726895.htm

[2] Annex V to the Report of the Ad-hoc Working Group on defining critical raw materials, European Commission Enterprise and Industry Directorate General.

HF is used as the feedstock in the manufacture of a host of fluorine chemicals used in dielectrics, metallurgy, wood preservatives, herbicides, mouthwashes, dentifrices, plastics and water fluoridation.

One of its most common end-products is fluorosilicic acid or hexafluorosilicic acid also known as hydrofluosilicic acid, which is used in water fluoridation. HF is the primary feedstock for the manufacture of virtually all organic and inorganic fluorine-bearing chemicals and is also a key ingredient in the processing of aluminium and uranium. The remaining use of fluorspar consumption is as a flux in steelmaking, in iron and steel foundries, primary aluminium production, glass manufacture, enamels, welding rod coatings, cement production, and other uses or products such as the manufacture of Hexafluorosilicic acid.

Trace elements such as lead and arsenic are present in finished products. Hexafluorosilicic acid is sold as a concentrated solution that contains a significant concentration of HF(aq) to prevent dissociation and hydrolysis of the H_2SiF_6.

In North America many municipal authorities source the same product using recovered sulphuric acid from acid scrubbers to react with either fluorspar or phosphate rock.

How is it manufactured?

Before fluorspar can be used to make hydrofluoric acid, the raw ore must be physically concentrated and purified. Fluorspar is crushed, ground up and purified by froth flotation. First the lead and zinc sulphides are separated and the fluorspar treated with sulphuric acid forming hydrofluoric acid gas (HF).

The acid grade fluorspar typically contains at least 97 percent calcium fluoride, as well as silica, mixed metal oxides and a trace amounts of arsenic. The HF gas then begins a purification process involving the gas being cooled, purified by scrubbing and condensed.

The crude product may be diluted and sold as an approximately 70 percent hydrofluoric acid solution, or distilled to remove any remaining water and further reduce impurities, and sold as hexafluorosilicic acid (hydrofluorosilicic acid) typically made up to a concentration of 37 to 42 per cent.

The manufacturing process generates tailing waste consisting of lead and zinc sulfides, spent flotation reagents and corrosive process wastewater.

How does it vary from natural calcium fluoride?

Calcium fluoride occurs naturally in many places in groundwater. In trace amounts this is harmless. Many people, however, are getting high levels of fluoride from many sources beyond the trace amounts of calcium fluoride that are considered "safe".

Calcium fluoride is sparingly soluble under standard conditions in the stomach of the human body, in natural water it is insoluble. Calcium fluoride is excreted mainly through the bowel with up to 80% of that ingested being excreted.

In contrast soft waters that contain little appreciable polyvalent cations (calcium, magnesium, rare earth elements, iron, etc.) do not allow for the removal of fluorosilicicates and therefore expose an individual to more HF in the gut because the solution lacks "F buffering capacity.

Sodium fluorosilicate is excreted mainly through the bladder with up to 50% of that ingested in healthy adults being excreted. There is further concern regarding exposure to fluorosilicates in humans that may re-associate in the stomach or bladder in low pH environments. One such concern is they may be associated with increased cancer disease.

According to Roholm's toxicology research on fluorine intoxication pertaining to various inorganic fluorides:

H_2SiF_6 (Fluorosilicic Acid) **is 25 times MORE lethally toxic than CaF2 (Calcium Fluoride)**

NaF (Sodium Fluoride) **is 20 times MORE lethally toxic than CaF2 (Calcium Fluoride)**

Na_2SiF_6 (Sodium Fluorosilicate) **is 20 times MORE lethally toxic than CaF2 (Calcium Fluoride)**

AlF_3 (Aluminium Fluoride) **is 8.3 times MORE lethally toxic than CaF2 (Calcium Fluoride)**

According to Urbansky, a senior U.S. EPA chemist and expert on water fluoridation chemicals; such compounds may exist in artificially fluoridated drinking water as well as in low pH acidic environments within the human body (i.e. stomach and bladder) after consumption of fluoridated water.[3]

[3] Urbansky Eward Todd, PhD, Fate of Fluorosilicate Drinking Water Additives, Chemical Reviews, 2002, Vol. 102, No. 8

Comparative Toxicity of Inorganic Fluorides:

Extremely toxic

Hydrogen fluoride	HF
Silicon tetrafluoride	SiF_4
Hydrofluoroc acid	HF
Hydrofluorosilicic acid	H_2SiF_6

Very Toxic

Easily soluble fluorides and fluorosilicates

Sodium fluoride	NaF
Potassium Fluoride	KF
Ammonium fluoride	NH_4F
Sodium fluoride	Na_2SiF
Potassium Fluorosilicate	K_2SiF_6
Ammonium fluorosilicate	$(NH_4)_2SiF_6$

Moderately Toxic

Cryolite	Na_3AlF_6
Calcium fluoride	CaF_2

Source: Roholm K [1937]. Fluorine intoxication. A clinical hygiene study with a review of the literature and some experimental investigations. London, England: H.K. Lewis & Co.

WHY TOXICITY IS IMPORTANT

Hexafluorosilicic acid is classified as a health, physicochemical and/or ecotoxicological hazard, according to the National Occupational Health and Safety Commission (NOHSC) Approved Criteria for Classifying Hazardous Substances.

Safety standards for hexafluorosilicic acid and its derivative compounds are very important as little data is available examining the toxicological effects such compounds have on human health or the environment. What information that is available from limited clinical studies clearly demonstrates that 1 ppm of hexafluorosilicic acid ingested orally is the equivalent of 25ppm calcium fluoride.

The EU drinking water standard for naturally occurring calcium fluoride is 1.5ppm however the vast majority of drinking water in continental Europe is below 0.3ppm. The newly revised optimal fluoride level in the USA, recommended by the U.S. Department of Health and Human Services, is 0.7ppm. The Irish limit for artificially fluoridated water is 0.8ppm. The natural background level in surface and groundwater in Ireland is generally below 0.1ppm.

The drinking water standards were established for the much less toxic calcium fluoride which is listed as a moderately toxic compound compared to hexafluorosilicic acid, which is categorized as extremely toxic.

All synthetic fluorides are toxic, and naturally occurring calcium fluoride is benign in comparison (in trace amounts of course, since too much of a good thing is no longer a good thing). Sodium fluoride is the most expensive synthetic fluoride, and is used the least to treat in water supplies. It is no longer used in Ireland. In the late 1990's Ireland sourced its Hexafluorosilicic acid from Holland where it was derived from a byproduct waste from the phosphate fertilizer industry when phosphate rock is processed. It is now sourced from Fluorspar mineral rock.

The chemically synthesised more toxic substance is used in the treatment of drinking water due to cost. Even though sodium fluoride is the least toxic synthetic fluoride, this type has been studied extensively, and associated with many adverse health problems. It is well established that there is incomplete toxicological data available on Hexafluorosilicic Acid products used for water fluoridation.

No data is available from the manufacturer or marketers of Hexafluorosilicic acid on:

- Development toxicity
- Teratogenic effects
- Carcinogenic effects
- Mutagenic effects
- Toxicity to animals
- Chronic long term effects on humans
- Ecotoxicity
- Biodegradation

No comprehensive scientific studies have been undertaken on Hexafluorosilicic acid products used for water fluoridation.

Only incomplete studies and analyses exist to test or measure the various dissociated derivative compounds that may exist in treated water and no detailed toxicological assessments exist to demonstrate their safety for human consumption or environmental toxicity.

What is known however, is that people drinking soft water treated with silicofluoride chemicals will be exposed to much greater toxicological and general health risks than individuals drinking hard water treated to the same artificially high fluoride level.

No studies have ever been undertaken on the bioavailability of fluoride with varying source water chemistry in Ireland and no human health studies undertaken either. What is now known, however, is that the highest prevalence of certain diseases and ill health in Ireland (diseases that may be associated with fluoride toxicity) are predominantly located in areas with soft water that is artificially fluoridated.

It is noteworthy that Chapter 10 of the NRC report (NRC 2006a) reviewed available human and animal studies of carcinogenicity, in addition to genotoxicity studies for fluoride, and the committee unanimously concluded that "Fluoride appears to have the potential to initiate or promote cancers."

The U.S. EPA found that "(f)luoride affects thyroid and parathyroid function in humans and animals, elevating thyroid stimulating hormone levels, altering levels of the thyroid hormones T3 and T4, and increasing levels of parathyroid hormone and calcitonin. These changes can affect the rate of formation of bone tissue and the overall rate of bone growth. These effects of fluoride on thyroid function also may be related to the observed dose-related increase in thyroid tumors in animal studies."[4]

In 2009 the U.S. Office of Environmental Health Hazard Assessment (OEHHA) proposed Fluoride and fluoride salts for review by the Carcinogen Identification Committee (CIC).

The international respected SENES Oak Ridge Centre for Risk Analysis recommend in particular that silicofluorides (especially H_2SiF_6) commonly used to provide fluoride ion in municipal drinking water, should be included in this analysis.

When added to drinking water Hexafluorosilicic acid dissociates into free fluoride ions, it is now accepted that this reaction is not complete with the possibility of some silicofluoride compounds remaining present in drinking water.[5]

It is further known that the following fluorosilicate species may be present in treated water. However current analytical methodologies are not yet available to accurately measure or quantify the level of residual fluorosilicates or fluorosilicon complexes that may be present.

Table 6. Homoleptic and Heteroleptic Aquo-, Hydroxo-, Oxo-, and Fluorosilicate Species Proposed in, Reported in, or Inferred from the Literature (Gas Phase, Nonaqueous/Aqueous Liquid Phase, and/or Solid Phase)

coordination number of the Si^{IV} center	fluorosilicates	aquo/hydroxo /oxosilicates	aquo/hydroxo/ oxo/fluorosilicates
6	SiF_6^{2-} $HSiF_6^-$	$Si(OH)_4(H_2O)_2$	$SiF_5(H_2O)^-$ $SiF_5(OH)^{2-}$ $SiF_4(OH)_2^{2-}$ $SiF_4(H_2O)_2$ $SiF(OH)_2(H_2O)_3^+$
5	SiF_5^- $HSiF_5$	$Si(OH)_5^-$	$SiF_4(OH)^-$
4	SiF_4	$Si(OH)_4$ $SiO(OH)_3^-$ $SiO_2(OH)_2^{2-}$	$SiF_3(OH)$ $SiF_2(OH)_2$ $SiF(OH)_3$
3	none	$SiO(OH)_2$ $SiO_2(OH)^-$	$Si(OH)_2F^+$

[4] Evidence of the carcinogenicity of Fluoride and its salts, Reproductive and Cancer Hazard Assessment Branch Office of Environmental Health Hazard Assessment, Californnia Environmental Protection Agency. July 2011,

[5] Urbansky Eward Todd, Fate of Fluorosilicate Drinking Water Additives, Chemical Reviews, 2002, Vol. 102, No. 8

Once added to water it is established that the liberated fluoride ions will rapidly complex with other cations present in water. This increases their bioavailability in the human body when consumed while free fluoride ions will transform into hydrofluoric acid in the human stomach.

It is accepted by the U.S. EPA that "*concentrations of hexafluorosilicic acid may be present in the gastrointestinal tract* ***after consumption of fluoridated drinking water***".[6]

The existence of fluorosilicic acid compounds was also noted[7] by the EU Scientific Committee on Health and Environmental Risks (SCHER), when it published its 'Opinion on critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water' – 16 May 2011.

The National Research Council (NRC 2006, pp. 52-53) and Coplan et al. (2007) have discussed the available information on the chemistry and toxicology of these compounds, especially at low pH (e.g., use of fluoridated water in beverages such as tea, soft drinks, or reconstituted fruit juices), when their dissociation to free fluoride ion is probably not complete and individuals are exposed to silicofluorides as a by-product of water fluoridation.

The U.S. EPA have also highlighted that certain toxic fluoride complexes in particular aluminium, iron and other cations may be present in artificially fluoridated water. Dr. Urbansky a senior chemist in the U.S. EPA noted in particular that "*natural waters contain a number of metallic cations that can be ligated by fluoride.* ***Fluoride binds to*** *trivalent metal cations, such as iron(III) and aluminium, as well as divalent metal cations, such as* ***calcium*** *and* ***magnesium.*** *" **And** "much of the fluoride is in fact present as metal complexes, depending on the concentrations of the metal cations, the fluoride anion, and the hydrogen ion."*[8]

It is also now hypothesized that incomplete dissociated SiF residues may re-associate both at intra-gastric pH and in the bladder which are low ph environments[9] (thereby exposing the consumer to toxic harm) and during food preparation (low pH soft drinks) producing SiF species including silicon tetrafluoride, (SiF4), a known toxin. It is also believed that commercial SiFs are likely to be contaminated with fluosiloxanes.

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[6] Urbansky and Schock (2000) Working Document U.S. EPA

[7] Scientific Committee on Health and Environmental Risks, SCHER, Critical review of any new evidence on the hazard profile, health effects, and human exposure to fluoride and the fluoridating agents of drinking water. May 2011, Page 11

[8] Edward Todd Urbansky, Fate of Fluorosilicate Drinking Water Additives *United States Environmental Protection Agency, Office of Research and Development, National Risk Management Research Laboratory, Water Supply and Water Resources Division, Received January 29, 2002, Chem. Rev.* 2002, *102*, 2837-2854

[9] Ciavatta L, et al; "Fluorosilicate Equilibria in Acid Solution"; Polyhedron Vol 7 (18);1773-79;1988

POISONOUS SUBSTANCES IN DRINKING WATER

So we now know from scientific study that due to artificial fluoridation of water liberated fluoride ions will form metallic fluoride compounds such as aluminium fluoride.

It is well established that the treatment of drinking water with aluminium sulphate (ALUM) used as a coagulant in water treatment may result in increased aluminium levels in treated water.[10]

It is further acknowledged that fluoride ions have a strong tendency to form complexes with heavy metal ions such as aluminium fluoride in water. The toxic potential of inorganic fluorides is mainly associated with this behavior and the formation of insoluble fluorides such as aluminium fluoride (AlF_3).[11]

In Ireland the *POISONS REGULATIONS, 1982* lists *alkali metal fluorides* as poisons. By adding Hexafluorosilicic acid to water one is not only creating silicofluoride compounds but alkali metal fluorides compounds that are poisonous to public health.

Aluminium fluoride complexes are also created in the stomach at low pH where it acts in competition with hydrofluoric acid. Aluminium fluoride is far more bioavailable than is the free aluminium ion which is quantitatively eliminated out the GI tract. Animal studies have found that aluminium fluoride complexes (AlF3) in drinking water will result in increased Aluminium levels in the brain and kidney as well as causing significant changes to brain cellular structure and neuronal integrity.[12]

The addition of any substance that is capable of a deleterious or injurious effect upon health is a violation of the Fluoridation of Water Supplies Regulations 2007. Fully or partially dissociated silicofluoride compound may also cause a health hazard because the fluoride ion, the undissociated and the reassociated fluorosilicate and the arsenic and lead present in the chemical are all hazardous to fetal and infant central nervous system development and function.

The Supreme Court Judgment of Ryan v. A.G. (1965) specifically forbids the addition of any amount of substances to water that may be harmful to human health including lead or arsenic. Both arsenic and lead are known to be present in water fluoridation chemicals.

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[10] United Nations Environment Programme World Health Organization 1997 International Programme On Chemical Safety Environmental Health Criteria 194: Aluminium

[11] International Programme On Chemical Safety Environmental Health Criteria 36 Fluorine And Fluorides, WHO 1984

[12] Varner JA, Jensen KF, Horvath W, Isaacson RL.Chronic administration of aluminium-fluoride or sodium-fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity. Brain Res. 1998 Feb 16;784(1-2):284-98.

A recent animal study found significantly higher concentrations of lead in both blood and calcified tissues of animals exposed to both silicofluorides and lead (Sawan et al. 2010). The International Agency for Research on Cancer (IARC) classified inorganic lead as probably carcinogenic to humans (Group 2A) in 2006. The European Food Safety Authority (EFSA) have further identified developmental neurotoxicity in young children and cardiovascular effects and nephrotoxicity in adults as the critical effects of lead exposure.

The U.S EPA has categorised lead as a probable human carcinogen and to have no practical threshold with respect to neurotoxicity (EPA 2004)—in other words, there is considered to be no safe level of lead exposure, and the MCLG for lead is zero (EPA 2009).

It is known that Hexafluorosilicic acid contains lead, regardless of the quantity it is not acceptable to be adding lead to drinking water in any amount.

Furthermore apart from the carcinogenicity of fluoride itself, silicofluoride use is associated with increased blood levels of a human carcinogen, one that is also associated with neurotoxicity and developmental toxicity,[13]

HEXAFLUOROSILICIC ACID BANNED AS A BIOCIDAL PRODUCT IN THE EU.

Hexafluorosilicic acid is used for many industries including the textile, ceraminc, steel industry and as a biocidal product.

The same active chemical substance used for water fluoridation was banned as a biocidal substance by the EU in 2006 under Directive 98/8/EC. Hexafluorosilicic acid can no longer be used due to a lack of environmental and toxicological data to demonstrate that it is safe for humans or the environment.

Detailed information was sought by the EU on the toxicology of the substance to include toxicological and metabolic studies, ecotoxicological studies, reproductive toxicity, medical data including medical surveillance data, epidemiological studies on general population, skin sensitivity studies and allergenicity studies, carcinogenicity studies, mutagenicity studies, sub chronic toxicity studies and measures to protect humans and the environment.

No information was provided to the EU. The substance was subsequently removed as an authorized biocide within EU. The phase out date was set as 01/09/2006. The product remains in use in Ireland as the active substance for water fluoridation of drinking water supplies.

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[13] Kathleen M. Thiessen, Ph.D. SENES Oak Ridge, Inc., Center for Risk Analysis Report Submitted to the Cancer Hazard Assessment Branch of the EPA at the request of the International Academy of Oral Medicine and Toxicology (IAOMT) Sept 2011

STATEMENT OF DR. J. WILLIAM HIRZY

NATIONAL TREASURY EMPLOYEES UNION CHAPTER 280

**BEFORE THE SUBCOMMITTEE ON WILDLIFE,
FISHERIES AND DRINKING WATER UNITED STATES SENATE**

JUNE 29, 2000

Good morning Mr. Chairman and Members of the Subcommittee. I appreciate the opportunity to appear before this Subcommittee to present the views of the union, of which I am a Vice-President, on the subject of fluoridation of public water supplies.

Our union is comprised of and represents the professional employees at the headquarters location of the U.S. Environmental Protection Agency in Washington D.C. Our members include toxicologists, biologists, chemists, engineers, lawyers and others defined by law as "professionals." The work we do includes evaluation of toxicity, exposure and economic information for managements use in formulating public health and environmental protection policy.

I am not here as a representative of EPA, but rather as a representative of EPA headquarters professional employees, through their duly elected labor union. The union first got involved in this issue in 1985 as a matter of professional ethics. In 1997 we most recently voted to oppose fluoridation. Our opposition has strengthened since then.

Summary of Recommendations

1) We ask that you order an independent review of a cancer bioassay previously mandated by Congressional committee and subsequently performed by Battelle Memorial Institute with appropriate blinding and instructions that all reviewers independent determinations be reported to this Committee.

2) We ask that you order that the two waste products of the fertilizer industry that are now used in 90% of fluoridation programs, for which EPA states they are not able to identify any chronic studies, be used in any future toxicity studies, rather than a substitute chemical. Further, since federal agencies are actively advocating that each man woman and child drink, eat and bathe in these chemicals, silicofluorides should be placed at the head of the list for establishing a MCL that complies with the Safe Drinking Water Act. This means that the MCL be protective of the most sensitive of our population, including infants, with an appropriate margin of safety for ingestion over an entire lifetime.

3) We ask that you order an epidemiology study comparing children with dental fluorosis to those not displaying overdose during growth and development years for behavioral and other disorders.

4) We ask that you convene a joint Congressional Committee to give the only substance that is being mandated for ingestion throughout this country the full hearing that it deserves.

National Review of Fluoridation

The Subcommittees hearing today can only begin to get at the issues surrounding the policy of water fluoridation in the United States, a massive experiment that has been run on the American public, without informed consent, for over fifty years. The last Congressional hearings on this subject were held in 1977. Much knowledge has been gained in the intervening years. It is high time for a national review of this policy by a Joint Select Committee of Congress. New hearings should explore, at minimum, these points:

1) excessive and un-controlled fluoride exposures;
2) altered findings of a cancer bioassay;
3) the results and implications of recent brain effects research;
4) the "protected pollutant" status of fluoride within EPA;
5) the altered recommendations to EPA of a 1983 Surgeon Generals Panel on fluoride;
6) the results of a fifty-year experiment on fluoridation in two New York communities;
7) the findings of fact in three landmark lawsuits since 1978;
8) the findings and implications of recent research linking the predominant fluoridation chemical with elevated blood-lead levels in children and anti-social behavior; and
9) changing views among dental researchers on the efficacy of water fluoridation

Fluoride Exposures Are Excessive and Un-controlled

According to a study by the National Institute of Dental Research, 66 percent of Americas children in fluoridated communities show the visible sign of over-exposure and fluoride toxicity, dental fluorosis (1). That result is from a survey done in the mid-1980's and the figure today is undoubtedly much higher.

Centers for Disease Control and EPA claim that dental fluorosis is only a "cosmetic" effect. God did not create humans with fluorosed teeth. That effect occurs when children ingest more fluoride than their bodies can handle with the metabolic processes we were born with, and their teeth are damaged as a result. And not only their teeth. Childrens bones and other tissues, as well as their developing teeth are accumulating too much fluoride. We can see the effect on teeth.

Few researchers, if any, are looking for the effects of excessive fluoride exposure on bone and other tissues in American children. What has been reported so far in this connection is disturbing. One example is epidemiological evidence (2a, 2b) showing elevated bone cancer in young men related to consumption of fluoridated drinking water.

Without trying to ascribe a cause and effect relationship beforehand, we do know that American children in large numbers are afflicted with hyperactivity-attention deficit disorder, that autism seems to be on the rise, that bone fractures in young athletes and military personnel are on the rise, that earlier onset of puberty in young women is occurring. There are biologically plausible mechanisms described in peer-reviewed research on fluoride that can link some of these effects to fluoride exposures (e.g. 3,4,5,6). Considering the economic and human costs of these conditions, we believe that Congress should order epidemiology studies that use dental fluorosis as an index of exposure to determine if there are links between such effects and fluoride over-exposure.

In the interim, while this epidemiology is conducted, we believe that a national moratorium on water fluoridation should be instituted. There will be a hue and cry from some quarters, predicting increased dental caries, but Europe has about the same rate of dental caries as the U.S. (7) and most European countries do not fluoridate (8). I am submitting letters from European and Asian authorities on this point. There are studies in the U.S. of localities that have interrupted fluoridation with no discernable increase in dental caries rates (e.g., 9). And people who want the freedom of choice to continue to ingest fluoride can do so by other means.

Cancer Bioassay Findings

In 1990, the results of the National Toxicology Program cancer bioassay on sodium fluoride were published (10), the initial findings of which would have ended fluoridation. But a special commission was hastily convened to review the findings, resulting in the salvation of fluoridation through systematic down-grading of the evidence of carcinogenicity. The final, published version of the NTP report says that there is, "equivocal evidence of carcinogenicity in male rats," changed from "clear evidence of carcinogenicity in male rats."

The change prompted Dr. William Marcus, who was then Senior Science Adviser and Toxicologist in the Office of Drinking Water, to blow the whistle about the issue (22), which led to his firing by EPA. Dr. Marcus sued EPA, won his case and was reinstated with back pay, benefits and compensatory damages. I am submitting material from Dr. Marcus to the Subcommittee dealing with the cancer and neurotoxicity risks posed by fluoridation.

We believe the Subcommittee should call for an independent review of the tumor slides from the bioassay, as was called for by Dr. Marcus (22), with the results to be presented in a hearing before a Select Committee of the Congress. The scientists who conducted the original study, the original reviewers of the study, and the "review commission" members should be called, and an explanation given for the changed findings.

Brain Effects Research

Since 1994 there have been six publications that link fluoride exposure to direct adverse effects on the brain. Two epidemiology studies from China indicate depression of I.Q. in children (11,12). Another paper (3) shows a link between prenatal exposure of animals to

fluoride and subsequent birth of off-spring which are hyperactive throughout life. A 1998 paper shows brain and kidney damage in animals given the "optimal" dosage of fluoride, viz. one part per million (13). And another (14) shows decreased levels of a key substance in the brain that may explain the results in the other paper from that journal. Another publication (5) links fluoride dosing to adverse effects on the brains pineal gland and pre-mature onset of sexual maturity in animals. Earlier onset of menstruation of girls in fluoridated Newburg, New York has also been reported (6).

Given the national concern over incidence of attention deficit-hyperactivity disorder and autism in our children, we believe that the authors of these studies should be called before a Select Committee, along with those who have critiqued their studies, so the American public and the Congress can understand the implications of this work.

Fluoride as a Protected Pollutant

The classic example of EPAs protective treatment of this substance, recognized the world over and in the U.S. before the linguistic de-toxification campaign of the 1940's and 1950's as a major environmental pollutant, is the 1983 statement by EPAs then Deputy Assistant Administrator for Water, Rebecca Hanmer (15), that EPA views the use of hydrofluosilicic acid recovered from the waste stream of phosphate fertilizer manufacture as, "...an ideal solution to a long standing problem. By recovering by-product fluosilicic acid (sic) from fertilizer manufacturing, water and air pollution are minimized, and water authorities have a low-cost source of fluoride..."

In other words, the solution to pollution is dilution, as long as the pollutant is dumped straight into drinking water systems and not into rivers or the atmosphere. I am submitting a copy of her letter. Other Federal entities are also protective of fluoride. Congressman Calvert of the House Science Committee has sent letters of inquiry to EPA and other Federal entities on the matter of fluoride, answers to which have not yet been received.

We believe that EPA and other Federal officials should be called to testify on the manner in which fluoride has been protected. The union will be happy to assist the Congress in identifying targets for an inquiry. For instance, hydrofluosilicic acid does not appear on the Toxic Release Inventory list of chemicals, and there is a remarkable discrepancy among the Maximum Contaminant Levels for fluoride, arsenic and lead, given the relative toxicities of these substances.

Surgeon Generals Panel on Fluoride

We believe that EPA staff and managers should be called to testify, along with members of the 1983 Surgeon Generals panel and officials of the Department of Human Services, to explain how the original recommendations of the Surgeon Generals panel (16) were altered to allow EPA to set otherwise unjustifiable drinking water standards for fluoride.

Kingston and Newburg, New York

Results In 1998, the results of a fifty-year fluoridation experiment involving Kingston, New York (un-fluoridated) and Newburg, New York (fluoridated) were published (17). In summary, there is no overall significant difference in rates of dental decay in children in the two cities, but children in the fluoridated city show significantly higher rates of dental fluorosis than children in the un-fluoridated city.

We believe that the authors of this study and representatives of the Centers For Disease Control and EPA should be called before a Select Committee to explain the increase in dental fluorosis among American children and the implications of that increase for skeletal and other effects as the children mature, including bone cancer, stress fractures and arthritis.

Findings of Fact by Judges

In three landmark cases adjudicated since 1978 in Pennsylvania, Illinois and Texas (18), judges with no interest except finding fact and administering justice heard prolonged testimony from proponents and opponents of fluoridation and made dispassionate findings of fact. I cite one such instance here.

In November, 1978, Judge John Flaherty, now Chief Justice of the Supreme Court of Pennsylvania, issued findings in the case, Aitkenhead v. Borough of West View, tried before him in the Allegheny Court of Common Pleas. Testimony in the case filled 2800 transcript pages and fully elucidated the benefits and risks of water fluoridation as understood in 1978. Judge Flaherty issued an injunction against fluoridation in the case, but the injunction was overturned on jurisdictional grounds. His findings of fact were not disturbed by appellate action. Judge Flaherty, in a July, 1979 letter to the Mayor of Aukland New Zealand wrote the following about the case:

"In my view, the evidence is quite convincing that the addition of sodium fluoride to the public water supply at one part per million is extremely deleterious to the human body, and, a review of the evidence will disclose that there was no convincing evidence to the contrary...

"Prior to hearing this case, I gave the matter of fluoridation little, if any, thought, but I received quite an education, and noted that the proponents of fluoridation do nothing more than try to impune (sic) the objectivity of those who oppose fluoridation."

In the Illinois decision, Judge Ronald Niemann concludes: "This record is barren of any credible and reputable scientific epidemiological studies and or analysis of statistical data which would support the Illinois Legislatures determination that fluoridation of the water supplies is both a safe and effective means of promoting public health."

Judge Anthony Farris in Texas found: "[That] the artificial fluoridation of public water supplies, such as contemplated by {Houston} City ordinance No. 80-2530 may cause or

contribute to the cause of cancer, genetic damage, intolerant reactions, and chronic toxicity, including dental mottling, in man; that the said artificial fluoridation may aggravate malnutrition and existing illness in man; and that the value of said artificial fluoridation is in some doubt as to reduction of tooth decay in man."

The significance of Judge Flahertys statement and his and the other two judges findings of fact is this: proponents of fluoridation are fond of reciting endorsement statements by authorities, such as those by CDC and the American Dental Association, both of which have long-standing commitments that are hard if not impossible to recant, on the safety and efficacy of fluoridation. Now come three truly independent servants of justice, the judges in these three cases, and they find that fluoridation of water supplies is not justified.

Proponents of fluoridation are absolutely right about one thing: there is no real controversy about fluoridation when the facts are heard by an open mind.

I am submitting a copy of the excerpted letter from Judge Flaherty and another letter referenced in it that was sent to Judge Flaherty by Dr. Peter Sammartino, then Chancellor of Fairleigh Dickenson University. I am also submitting a reprint copy of an article in the Spring 1999 issue of the Florida State University Journal of Land Use and Environmental Law by Jack Graham and Dr. Pierre Morin, titled "Highlights in North American Litigation During the Twentieth Century on Artificial Fluoridation of Public Water." Mr. Graham was chief litigator in the case before Judge Flaherty and in the other two cases (in Illinois and Texas).

We believe that Mr. Graham should be called before a Select Committee along with, if appropriate, the judges in these three cases who could relate their experience as trial judges in these cases.

Hydrofluosilicic Acid

There are no chronic toxicity data on the predominant chemical, hydrofluosilicic acid and its sodium salt, used to fluoridate American communities. Newly published studies (19) indicate a link between use of these chemicals and elevated level of lead in childrens blood and anti-social behavior. Material from the authors of these studies has been submitted by them independently.

We believe the authors of these papers and their critics should be called before a Select Committee to explain to you and the American people what these papers mean for continuation of the policy of fluoridation.

Changing Views on Efficacy and Risk

In recent years, two prominent dental researchers who were leaders of the pro-fluoridation movement announced reversals of their former positions because they concluded that water fluoridation is not an effective means of reducing dental caries and that it poses serious risks

to human health. The late Dr. John Colquhoun was Principal Dental Officer of Aukland, New Zealand, and he published his reasons for changing sides in 1997 (20). In 1999, Dr. Hardy Limeback, Head of Preventive Dentistry, University of Toronto, announced his change of views, then published a statement (21) dated April 2000. I am submitting a copy of Dr. Limebacks publications.
We believe that Dr. Limeback, along with fluoridation proponents who have not changed their minds, such as Drs. Ernest Newbrun and Herschel Horowitz, should be called before a Select Committee to testify on the reasons for their respective positions.

Thank you for you consideration, and I will be happy to take questions.

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NTEU CHAPTER 280 - U.S. ENVIRONMENTAL PROTECTION AGENCY, NATIONAL HEADQUARTERS
BEN FRANKLIN STATION, BOX 7672, WASHINGTON D.C. 20044 - PHONE 202-566-2788
INTERNET http://www.nteu280.org **E MAIL** Murphy.JamesJ@epa.gov

PRESS RELEASE FOR AUGUST 19, 2005

EPA Unions Call for Nationwide Moratorium on Fluoridation, Congressional Hearing on Adverse Effects, Youth Cancer Cover Up

Eleven EPA employee unions representing over 7000 environmental and public health professionals of the Civil Service have called for a moratorium on drinking water fluoridation programs across the country, and have asked EPA management to recognize fluoride as posing a serious risk of causing cancer in people. The unions acted following revelations of an apparent cover-up of evidence from Harvard School of Dental Medicine linking fluoridation with elevated risk of a fatal bone cancer in young boys.

The unions sent letters to key Congressional committees asking Congress to legislate a moratorium pending a review of all the science on the risks and benefits of fluoridation. The letters cited the weight of evidence supporting a classification of fluoride as a likely human carcinogen, which includes other epidemiology results similar to those in the Harvard study, animal studies, and biological reasons why fluoride can reasonably be expected to cause the bone cancer – osteosarcoma – seen in young boys and test animals. The unions also pointed out recent work by Richard Maas of the Environmental Quality Institute, University of North Carolina that links increases in lead levels in drinking water systems to use of silicofluoride fluoridating agents with chloramines disinfectant.

The letter to EPA Administrator Stephen Johnson asked him to issue a public warning in the form of an advanced notice of proposed rulemaking setting the health-based drinking water standard for fluoride at zero, as it is for all known or probable human carcinogens, pending a recommendation from a National Academy of Sciences' National Research Council committee. That committee's work is not expected to be done before 2006.

The unions also asked Congress and EPA's enforcement office, or the Department of Justice, to look into reasons why the Harvard study director, Chester Douglass, failed to report the seven-fold increased risk seen in the work he oversaw, and instead wrote to the National Institute of Environmental Health Sciences, the federal agency that funded the Harvard study, saying there was no link between fluoridation and osteosarcoma. Douglass sent the same negative report to the National Research Council committee studying possible changes in EPA's drinking water standards for fluoride.

The unions who signed the letters represent EPA employees from across the nation, including laboratory

scientists in Ohio, Oklahoma and Michigan, regulatory support scientists and other workers at EPA headquarters in Washington, D.C. and science and regulatory workers in Boston, New York, Philadelphia, Atlanta, and San Francisco. They are affiliated with the National Treasury Employees Union, the American Federation of Government Employees, Engineers and Scientists of California/International Federation of Professional and Technical Engineers, and the National Association of Government Employee/Service Employees International Union.

The unions' letter is online at http://nteu280.org/Issues/Fluoride/fluoridesummary.htm

FOR INFORMATION CONTACT:
Dr. William Hirzy, Vice-President
NTEU Chapter 280
Phone(cell) 202-285-0498

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John R. Lee, M.D.
9620 Bodega Hwy.
Sebastopol, CA 95472, USA

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WATER FLUORIDATION AND TOOTH DECAY: RESULTS FROM THE 1986-1987 NATIONAL SURVEY OF U.S. SCHOOLCHILDREN

by

John A. Yiamouyiannis*
Delaware, Ohio, USA

SUMMARY: Data from dental examinations of 39,207 schoolchildren, aged 5-17, in 84 areas throughout the United States are analyzed. Of these areas, 27 had been fluoridated for 17 years or more (F), 30 had never been fluoridated (NF), and 27 had been only partially fluoridated or fluoridated for less than 17 years (PF). No statistically significant differences were found in the decay rates of permanent teeth or the percentages of decay-free children in the F, NF and PF areas. However, among 5-year-olds, the decay rates of deciduous teeth were significantly lower in F than in NF areas.

KEY WORDS: Dental caries; Fluoridation; Schoolchildren; Tooth decay.

Introduction

It has become widely accepted among dental and public health professionals that fluoridation reduces tooth decay by one-half to two-thirds (1,2). However, recent studies by public health dentists in New Zealand, Canada and the United States have reported similar or lower tooth decay rates in nonfluoridated areas as compared to fluoridated areas (3-6). Moreover findings in the United States and worldwide show that, over the last 25 years, reductions in tooth decay rates in nonfluoridated areas are comparable to those in fluoridated areas (7-9).

From 1986 to 1987, dentists trained by the U.S. National Institute of Dental Research (NIDR) performed dental examinations on 39,207 schoolchildren, aged 5-17, in 84 areas throughout the United States. This survey allowed a comparison of tooth decay of large numbers of people from a large number of areas, some of which have been fluoridated and some of which have not.

Materials and Methods

Through the United States Freedom of Information Act, we obtained a printout of the dental records and a list of the 84 areas used in this survey. From these data, we calculated the number of decayed and filled deciduous teeth (dft) and the number of decayed, missing, and filled permanent teeth (DMFT) for each record and entered the resulting data into a computer. All calculations were triple-checked before being entered into the computer and all computer entries were double-checked.

By computer, each record (including the dft and DMFT scores of each student) was placed in the appropriate age group. For each of the 13 age

* Safe Water Foundation, 6439 Taggart Road, Delaware, Ohio 43015, USA.

groups, average dft and DMFT rates per child were determined for each of the 84 areas. Age-adjusted DMFT rates for 5- to 17-year-olds were calculated by adding the DMFT rates for each of the 13 age groups and dividing by 13 (10).

We obtained data regarding the fluoridation status of the areas surveyed from Natural Fluoride Content of Community Water Supplies, Fluoridation Census 1969, Fluoridation Census 1975, and Fluoridation Census 1985, all published by the U.S. Public Health Service. In some cases, local authorities were also contacted to determine the fluoridation status of an area.

Average DMFT (and dft) rates for the F, NF, and PF groups were calculated for each age. Average-age-adjusted DMFT (and dft) rates for the F, NF, and PF groups were calculated by taking the average of the age-adjusted rates for the respective groups (10).

The percentage of "caries-free" children was calculated for each age-group for each area. Age-adjusted "caries-free" rates were also calculated. A student was considered to be "caries-free" so long as they had no DMFT or dft. For example, a child who had lost all their teeth and no longer had any left to be decayed or filled would not be recorded as a "caries-free" student.

Through the United States Freedom of Information Act, we also obtained residence data for each of the above schoolchildren which allowed us to calculate tooth decay rates for those in F, NF, and PF areas who had lived at the same residence for their entire life.

The two-tailed t-test was used to determine 95% confidence intervals and to determine statistical significance (at the 95% confidence level). A two-sided Wilcoxon rank sum test (11) was used to determine whether there was a statistically significant difference (at the 95% confidence level) in the rank order of the DMFT rates of F and NF areas.

Results

Table 1 presents the number of students examined and the age-adjusted DMFT rate for each of the 84 areas in the order of increasing tooth decay rate. There is no statistically significant difference in the rank order of the age-adjusted DMFT rates of F and NF areas. As can be seen by examination of column 1, there is no clustering of fluoridated areas at the top of the table. In the quartile with the lowest age-adjusted DMFT rates, 9 are non-fluoridated, 3 are partially fluoridated, and 9 are fluoridated. In the quartile with the highest DMFT rates, 5 are nonfluoridated, 10 are partially fluoridated, and 6 are fluoridated. Table 1 also indicates that there is no biased geographical distribution of F and NF areas that is hiding some potential decay-preventive effect of water fluoridation.

There is no statistically significant difference between the average DMFT rates for the F and NF groups at any age (Figure 1). The average DMFT rates of the PF groups are higher than those of the F and NF groups at every age with the exception of 14-year-olds.

There is no statistically significant difference in the average-age-adjusted DMFT rates among the F, PF, and NF groups (Table 2). The average-age-

Figure 1

Tooth decay in fluoridated (F), partially fluoridated (PF), and non-fluoridated (NF) areas: Permanent Teeth.

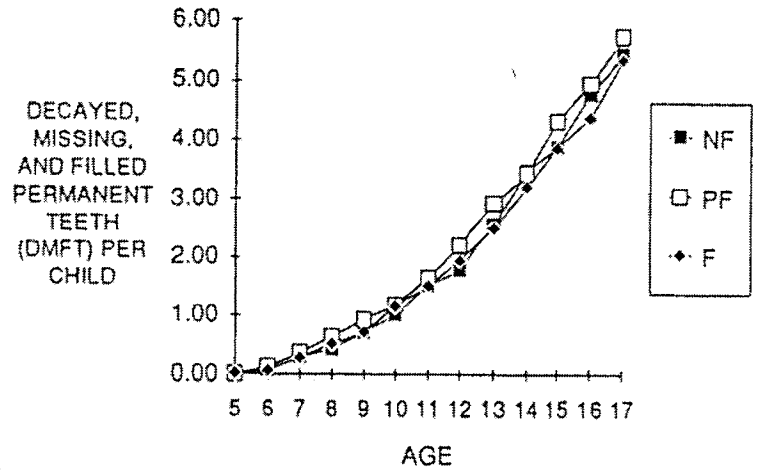


Table 2

Average-age-adjusted DMFT rates for 39,207 U.S. schoolchildren and 17,336 life-long resident schoolchildren in 84 areas throughout the United States. Standard deviations are given in parentheses.

		Total		Life-long	
	No. of Areas	No. of Students	DMFT	No. of Students	DMFT
Fluoridated	27	12,747	1.96 (0.415)	6,272	1.97 (0.465)
Partially Fluoridated	27	12,578	2.18 (0.465)	5,642	2.25 (0.470)
Nonfluoridated	30	13,882	1.99 (0.408)	5,422	2.05 (0.517)

adjusted DMFT rates in F and NF areas are 1.96 and 1.99, respectively. The 95% confidence interval for the DMFT rate in F areas minus the DMFT rate in NF areas is (-0.19, 0.25); thus we can rule out, with a certainty of 95%, the possibility that the DMFT rate in F areas is more than one-fourth of a tooth less than in the NF areas. We can also rule out, with a certainty of 95%, the possiblity that the DMFT rate in NF areas is more than one-fifth of a tooth less than in the F areas.

Table 1

The number of children examined and the average-age-adjusted DMFT, dft, and "caries-free" rates for 5- to 17-year-olds in each of the 84 areas in the order of increasing age-adjusted DMFT rate. F refers to areas fluoridated before 1970; PF refers to areas which are only partially fluoridated; PF(x) refers to areas fluoridated in the year "x"; NF refers to areas that are not fluoridated.

Water	Area	No.	DMFT	dft	Caries-free
NF	Buhler, KS	543	1.229	0.810	44.7%
F	El Paso, TX	451	1.321	0.777	43.5%
NF	Brooklyn, CT	410	1.420	0.693	47.6%
F	Richmond, VA	475	1.435	0.715	45.6%
F	Ft. Scott, KS	491	1.442	0.774	38.2%
F	Prince George, MD	443	1.491	0.539	48.0%
NF	Cloverdale, OR	354	1.494	0.872	40.4%
PF(71)	Alliance, OH	467	1.584	0.549	44.6%
NF	Martin Co., FL	440	1.587	0.677	41.0%
F	Andrews, TX	455	1.588	0.893	35.8%
NF	Coldspring, TX	406	1.589	1.144	33.8%
F	Tulsa, OK	504	1.602	1.075	35.5%
NF	Palm Beach, FL	476	1.613	0.896	34.5%
PF	Holcomb, MO	558	1.628	0.883	40.3%
NF	Kitsap, WA	564	1.635	0.769	42.9%
F	St. Louis, MO	491	1.638	0.711	39.1%
PF(82)	Houston, TX	488	1.662	0.819	41.8%
F	Clarksville, IN	428	1.678	0.747	40.4%
NF	Grand Island, NE	535	1.719	0.789	40.7%
F	Ft. Stockton, TX	415	1.722	0.891	33.4%
NF	San Antonio, TX	422	1.736	0.895	39.3%
F	Cherry Creek, CO	441	1.757	0.727	36.5%
F	Tuscaloosa, AI	475	1.809	0.963	32.0%
PF	Marion Co., FL	545	1.817	0.944	28.8%
F	Cleveland, OH	486	1.819	0.715	39.9%
NF	Allegany, MD	458	1.834	0.735	38.3%
PF(78)	Norwood, MA	434	1.841	0.640	39.9%
F	Alton, IL	511	1.859	0.843	37.6%
NF	Shamokin, PA	462	1.861	1.023	32.2%
NF	Lodi, CA	573	1.878	1.197	33.0%
PF	Bullock Creek, MI	472	1.879	0.766	36.7%
PF(82)	Marlboro, MA	386	1.885	0.613	40.8%
PF(81)	Allen, TX	445	1.905	0.674	38.7%
F	San Francisco, CA	456	1.908	1.031	36.3%
NF	E. Orange, NY	401	1.909	0.796	38.0%
PF(71/60)	Lincoln/Sudbury, MA	436	1.923	0.758	37.8%
NF	Conejo, CA	620	1.930	0.811	41.7%
NF	Lakewood, NJ	450	1.933	0.698	38.0%
F	New York City–2	336	1.953	0.812	34.9%
PF	Bethel, WA	540	1.956	1.072	34.3%
F	Beach Park, IL	518	1.970	0.878	35.2%
PF	Rising Star, TX	370	1.971	0.909	28.7%
F	Philipsburg, PA	499	1.983	0.982	33.2%

Table 1 (Continued)

Water	Area	No.	DMFT	dft	Caries-free
F	Lanett, AL	503	1.994	0.978	31.9%
PF(82)	Plainville, CT	436	2.006	0.795	39.3%
NF	Wichita, KS	496	2.036	0.878	33.5%
NF	Newark, NJ	494	2.038	0.869	35.9%
PF	Knox Co., TN	530	2.056	1.152	31.3%
NF	Los Angeles, CA	540	2.063	1.039	33.0%
F	Pittsburgh, PA	415	2.064	0.781	34.1%
PF(70)	Lincoln, NE	476	2.076	0.825	31.5%
NF	Newton, KS	464	2.083	1.225	31.1%
PF	Lakeshore, MI	486	2.088	0.781	32.6%
NF	New Paltz, NY	350	2.110	0.751	34.8%
F	Bemidgi, MN	485	2.124	1.001	29.3%
NF	Alpine, OR	397	2.133	0.974	36.7%
NF	Canon City, CO	463	2.160	1.118	33.1%
NF	Wyandank, NY	396	2.161	0.828	34.7%
NF	Millbrook, NY	332	2.179	0.716	32.2%
NF	Chowchilla, CA	551	2.181	1.073	33.0%
F	New York City–1	503	2.190	0.627	37.9%
PF(82)	Baltic, SD	487	2.193	0.974	27.8%
PF(71/74)	Blue Hill, NE	480	2.218	0.855	29.6%
NF	Crawford, PA	492	2.222	0.996	28.5%
PF(74)	New Orleans, LA	459	2.251	0.953	27.4%
PF(70)	Memphis, TN	464	2.253	0.763	33.1%
PF	Madison Co., MS	493	2.259	1.455	26.4%
F	Milwaukee, WI	478	2.349	0.909	29.9%
NF	Tooele, UT	519	2.372	1.458	24.3%
NF	Chicopee, MA	453	2.389	0.862	34.2%
PF	Cambria, PA	532	2.460	1.039	27.1%
PF(75)	Springfield, VT	444	2.489	0.838	32.1%
F	Dearborn, MI	491	2.496	1.167	26.3%
F	Maryville, TN	466	2.512	1.287	22.9%
PF(81)	Taunton, MA	445	2.515	0.903	31.0%
F	Greenville, MI	556	2.558	1.191	25.3%
PF	Hart/Pentwater, MI	455	2.584	1.344	24.1%
F	Philadelphia, PA	463	2.649	0.824	26.0%
PF	Sup. Union #47, VT	487	2.710	0.907	26.1%
NF	Cutler/Orosi, CA	528	2.796	1.742	19.2%
F	Brown City, MI	512	2.972	1.229	22.5%
PF(83)	Lawrence, MA	339	3.012	1.262	17.6%
NF	State of Hawaii	293	3.294	1.375	23.9%
PF	Concordia Co., LA	424	3.767	1.508	12.4%

To make certain that the absence of a statistically significant difference between the DMFT rates of schoolchildren living in F and NF areas was not the result of the mobility of schoolchildren, or their sex and racial compositions, DMFT rates were determined for 1.] those who spent their entire lives in one household and 2.] for white males and white females. The results in Table 2 show that for life-long residents, there is no statistically significant difference in average-age-adjusted DMFT rates in F and NF areas. In addition,

there are no statistically significant differences in tooth decay rates between permanent residents of F and NF areas at any age (Figure 2A). If water fluoridation were to have reduced tooth decay as measured by DMFT, tooth decay rates for life-long residents living in fluoridated areas should be lower than residents who had not spent their entire lives in these areas. This was not found to be the case. Figures 2B and 2C show that among white males

Figure 2A

Tooth decay in residents of fluoridated (F), nonfluoridated (NF), and partially fluoridated (PF) areas who lived their entire life in the same household.

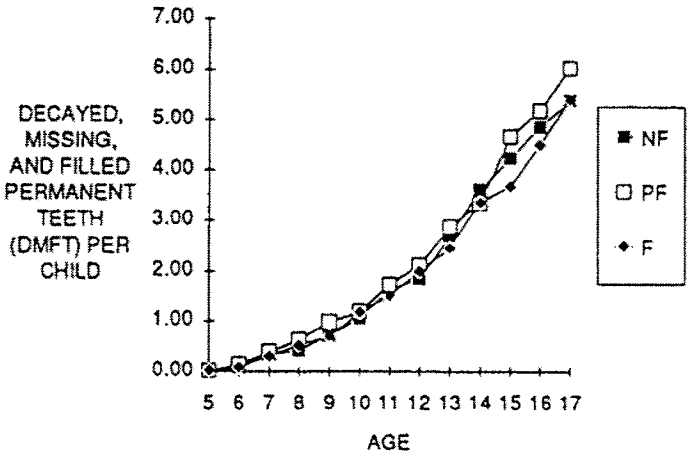


Figure 2B

Tooth decay rates of white males.

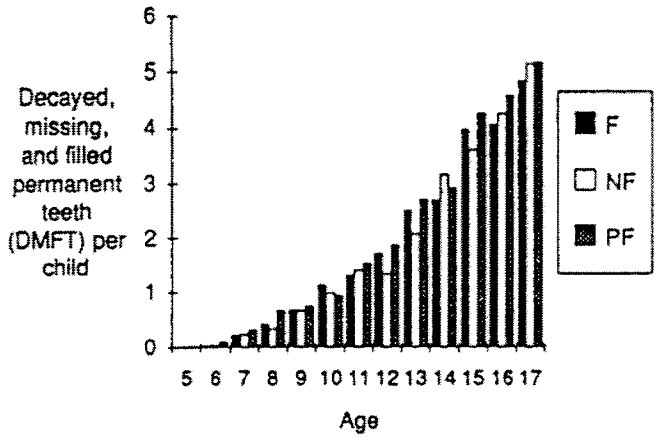
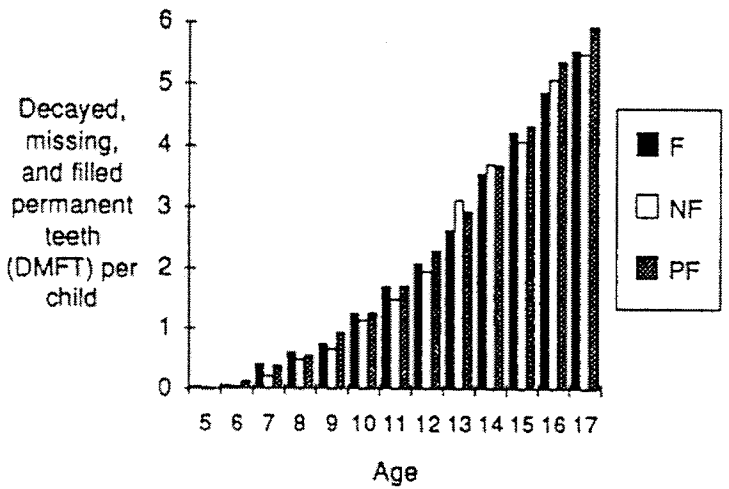


Figure 2C

Tooth decay rates of white females.

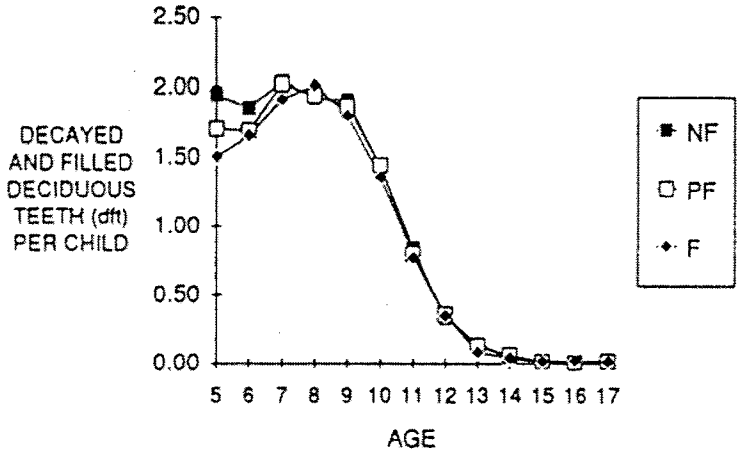


and white females (which make up about 70% of all the children studied), there is no significant difference in DMFT rates in the F and NF areas at any age group.

In contrast, notably lower tooth decay rates were observed in the deci-

Figure 3

Tooth decay in fluoridated (F), Partially fluoridated (PF), and non-fluoridated (NF) areas: Deciduous Teeth.



duous teeth of young children living in F areas. The 5-, 6-, and 7-year-olds in the F group have dft rates 22%, 9% and 6% lower than those of the NF group, rspectively (Figure 3). Although the average-age-adjusted dft rates for F, NF, and PF groups were not significantly different statistically, they were higher for the NF groups (0.96 ±0.25) for the PF Groups (0.93 ±0.24), which in turn is slightly higher than the F group (0.89 ±0.19).

To focus in on dft rates among children 5-8, the eight areas which commenced water fluoridation between 1970 and 1978 were removed from the PF group and added to the F group. The 5-, 6-, and 7-year-olds in the new F (F*) group have dft rates 24%, 10%, and 10% lower than those of the NF group, respectively, and the dft rate of 5-year-olds in the F* group is significantly lower ($p < 0.05$) than that of the NF group.

Moreover among 5-, 6-, and 7-year-old life-long residents in the F* group, dft rates were 42%, 18% and 11% lower than those of the NF group, respectively, and the dft rate of 5-year-olds in the F* group was significantly lower ($p < 0.002$) than that of the NF group (Table 3). If water fluoridation were to have reduced tooth decay as measured by dft among 5-year-olds, tooth decay rates for life-long 5-year-old residents living in fluoridated areas should have been lower than those of residents who had not spent their entire lives in these areas. This was found to be the case. From Table 3, it can also be seen that this large and significant reduction disappears after a couple of years.

Fluoride may have caused a reduction in dft by delaying deciduous tooth eruption. This is consistent with the fact that the dft rate in the F and F* groups reaches a maximum later than in the NF group. Fluoride-induced delays in tooth eruption have been reviewed elsewhere (12,13) with contradictory conclusions, but more recent studies examining 5-year-olds have indicated delayed eruption that could account for such a difference in tooth decay rates (14).

The percentage of decay-free children in F, PF, and NF areas is 34.5%, 31.9%, and 35.1% respectively. There is no statisticaly significant difference between the average "caries-free" rates for the F and NF groups at any age (Figure 4).

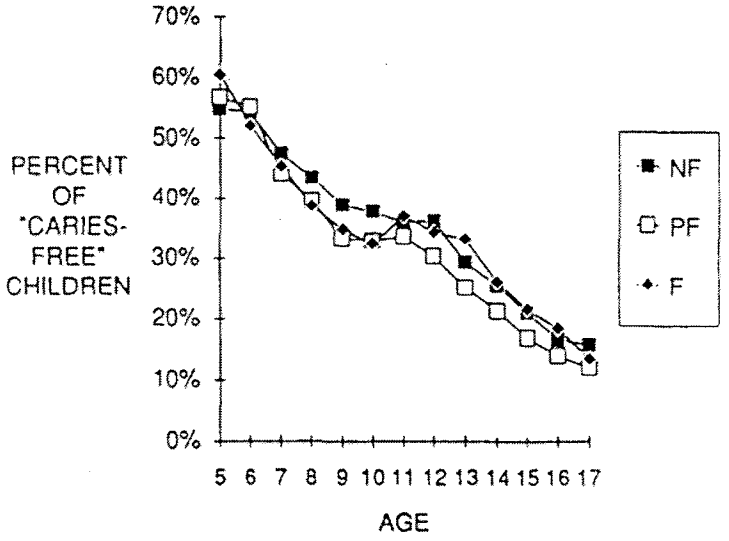
Table 3

Percentage change in dft rates in all residents and life-long residents of F and F* areas in comparison to NF areas.

	Total		Life-long	
Age	(NF-F)/NF	(NF-F*)/NF	(NF-F)/NF	(NF-F*)/NF
5	22%	24% ($p < 0.05$)	36% ($p < 0.02$)	42% ($p < 0.002$)
6	9%	10%	14%	18%
7	6%	10%	5%	11%
8	-4%	1%	-5%	1%

Figure 4

"Caries-free" rates in nonfluoridated (NF), partially fluoridated (PF), and fluoridated (F) areas.



Discussion

The data presented here are consistent with data reported elsewhere in large U.S. surveys. In 1977, the Rand Corporation examined the tooth decay rate of 25,000 children in (5 F and 5 NF) nonrandomly selected areas (15). In the three areas in their study that were included in the present study, we compared the tooth decay rates of 12-year-olds. There was good agreement between this study and theirs with regard to tooth decay rate, after converting DMFS (decayed, missing and filled permanent tooth surfaces) to DMFT (16) and considering the acknowledged 36% decrease in DMFS from 1979-1980 to 1986-1987 (17).

In 1983-1984, Hildebolt et al. (4) examined the tooth decay rates of over 6500 Missouri rural schoolchildren from grades 2 (average age 7.5) and 6 (average age 11.5). Among 6th graders living in the most intensively studied regions, the average DMFT+dft rate was 2.07 for those drinking nonfluoridated water and 2.17 for those drinking fluoridated water, compared to the DMFT+dft rate of 2.00 reported for 11-year-olds living in Holcomb, Missouri in our study.

In 1986, Kumar et al. examined 1446 schoolchildren aged 7-14 from Newburgh, New York (fluoridated in 1945) and cohorts from nonfluoridated Kingston, New York (18). The sample selection was nonrandom and had a response rate of only 50-65%. Nonetheless, the age-adjusted DMFT rates ob-

served (1.5 for fluoridated Newburgh and 2.0 for nonfluoridated Kingston) were in line with the corresponding values obtained in this study for communities in the area (1.5 for nonfluoridated New Paltz, New York and 1.7 for fluoridated New York City).

Conclusions

Does water fluoridation reduce tooth decay? i] This study and other recent studies (3-8) show that there is currently no significant difference in tooth decay rates in F and NF areas and that decreases in tooth decay rates over the last 25 years have been comparable regardless of fluoridation status; if this is true, there was no significant difference in the tooth decay rates between these areas 25 years ago. ii] From 1970 to the present, total fluoride intake studies indicate an average intake of 1-2 mg per day in nonfluoridated areas and 3-5 mg per day in fluoridated areas (19,20); thus, it is difficult to claim that the reason tooth decay differentials between fluoridated and nonfluoridated areas have disappeared is because the fluoride intakes in these areas are now similar. Furthermore, the substantially higher incidence of dental fluorosis in fluoridated areas confirms that residents in these areas are consuming substantially higher levels of fluoride than those living in nonfluoridated areas (21-23). iii] Dramatic reductions in tooth decay have occurred in developing countries where there is no water fluoridation and there is little reason to suspect that there would be elevated levels of fluoride in the food chain (7,9,24,25). iv] In addition to recent studies, a number of early studies have also shown no significant reduction in tooth decay as a result of water fluoridation (7,26-28). v] Serious questions have been raised regarding the reliability of earlier studies claiming that fluoridation causes a reduction in tooth decay (29).

Acknowledgements

I thank Kimberly Close-Hittle, Jerry Putnam, Margot Yiamouyiannis, and Opal Kuhn for their help in the calculation and verification of summary data as well as Jill Pitts and Chris Hiatt for their lightning fast speed in entering data into our computer. Without the generousity of Dr. Leo Roy, Dr. Reuben Benner, Dr. H. Charles Kaplan, Dr. Gerard Judd, Richard Barmakian, John C. Justice, Len Greenall, Mr. and Mrs. Andrew Yimoyines, Wini Silko, AIM International, Inc., and other patrons of the Center for Health Action and the Safe Water Foundation, the preparation and publication of this article would not have been possible. Finally, I thank Ray Fahey for correcting an error we had made in assigning the fluoridation status of E. Orange, NJ.

Addendum

Recently Brunelle (30), using the same database that we used, reported 26% fewer dfs (decayed and filled deciduous tooth surfaces) in children who had always resided in F communities than those who never lived in F communities. This finding agrees reasonably well with the data outlined in our Table 3, which shows a statistically significantly lower dft rate in life-long 5-year-old residents of fluoridated areas. However, by omission of age-specific data, the Brunelle study covers up the fact that this difference in tooth decay is no longer significant in 6-year-olds and disappears entirely among 8-year-olds.

Another recent study by Brunelle and Carlos (31), which also uses the same database that we used, reports a 17.7% lower DMFS rate in the F areas. This study has a number of major deficiencies which render the study of little or no value.

1. It contains extremely serious errors. For example, by a cursory inspection, we found two values that are off by 100% or more. In their Table 9, the DMFS figure for life-long F exposure residents of Region VII should be about 3, not 1.46 as reported. From their Table 3, the percent of 5-year-olds who have caries is 1.0%, not the 2.7% that can be calculated from the table (100%-97.3%). When I pointed out this error to Dr. Carlos, he admitted that only 19 out of the 1851 5-year-olds had caries: 19/1851 = 1%, but refused to make the correction (32).

2. It fails to report the tooth decay rates for each of the 84 geographical areas surveyed. This covers up the fact that there is no difference in the tooth decay rates of the fluoridated and nonfluoridated areas surveyed. The Brunelle/Carlos study even fails to list the areas studied. As a result, they produce misleading illustrations; for example, their Figure 3 implies that Arizona and New Mexico have the lowest tooth decay rates, when, in fact, not a single area was surveyed in either of the two states.

3. It fails to control for geographical differences in tooth decay rates by indiscriminantly and disproportionately bunching children from all parts of the country into 2 groups, F and NF.

4. It fails to do the statistical analysis (or even provide the data, i.e. the standard deviation and sample number) necessary to determine whether the values found for F and NF areas are significantly different. Our calculations show that even if their data were accurate, the 17.7% figure does not reflect a statistically significant difference between the F and NF groups.

5. It fails to report the data for the approximately 23,000 schoolchildren who were not life-time residents of either the F or NF areas (the PF group). If fluoridation reduced tooth decay, the DMFS rate of the PF group should have been greater than that of the F group and less than that of the NF group. Our data indicate that the PF group would have had a DMFS rate higher (although not significantly higher) than either the F or NF groups.

6. It fails to report the data for the percentages of decay-free children in F and NF areas. Our data indicate that had these calculations been done by Brunelle and Carlos, the results may have actually indicated better (although not significantly better) dental health in the NF areas.

Brunelle and Carlos, as well as their employer, the NIDR, have recently come under attack for presenting erroneous data and designing poor experiments which promoted the fluoride mouthrinse program (33). The apparent poor quality of their research regarding the 1986-1987 survey (30,31) is not an isolated case.

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Jay Harris Levy, DDS
511 SW 10th Suite 1102
Portland, OR 97205
503.222.2157
www.jayharrislevy.com
jayharrislevy@easystreet.net
JayHarrisLevy@gmail.com

Fax to:

Gayla Jennings
Deputy Auditor | Office of the City Auditor
City of Portland, Oregon
1221 SW 4th Avenue, Room 140
Portland, Oregon 97204-1987
Phone (503) 823-3560
gayla.jennings@portlandoregon.gov

PORTLAND CITY COUNCIL
COMMUNICATION REQUEST
Wednesday Council Meeting 9:30 AM

Council Meeting Date: December 19, 2012

Today's Date November 9, 2012

Name Jay Harris Levy, DDS

Address 301 NE 65th Avenue, Portland, OR 97213

Telephone 503-261-0916 Email jaylevy@easystreet.net

Reason for the request:
As a practicing dentist for 30 years, a former researcher and faculty member at OHSU School of Dentistry and having worked in public health dental clinics, I would like to present my perspective on water fluoridation to the Portland City Council.

(signed) Jay Harris Levy DDS

- Give your request to the Council Clerk's office by Thursday at 5:00 pm to sign up for the following Wednesday Meeting. Holiday deadline schedule is Wednesday at 5:00 pm. (See contact information below.)
- You will be placed on the Wednesday Agenda as a "Communication." Communications are the first item on the Agenda and are taken promptly at 9:30 a.m. A total of five Communications may be scheduled. Individuals must schedule their own Communication.
- You will have 3 minutes to speak and may also submit written testimony before or at the meeting.

Thank you for being an active participant in your City government.

Contact Information:

Karla Moore-Love, City Council Clerk
1221 SW 4th Ave, Room 140
Portland, OR 97204-1900
(503) 823-4086 Fax (503) 823-4571
email: Karla.Moore-Love@portlandoregon.gov

Sue Parsons, Council Clerk Assistant
1221 SW 4th Ave., Room 140
Portland, OR 97204-1900
(503) 823-4085 Fax (503) 823-4571
email: Susan.Parsons@portlandoregon.gov

1473 –

Request of Jay Harris Levy, DDS to address Council regarding water fluoridation (Communication)

DEC 19 2012

PLACED ON FILE

Filed DEC 14 2012

LaVonne Griffin-Valade
Auditor of the City of Portland

By _____

COMMISSIONERS VOTED AS FOLLOWS:		
	YEAS	NAYS
1. Fritz		
2. Fish		
3. Saltzman		
4. Leonard		
Adams		