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CONTENTS

Continued next page

DISCUSSION SECTION

MISSING EVIDENCE REVEALED

"... for truth is truth
To the end of reckoning."
Shakespeare, Measure for Measure, V.1.45-46

In this issue two previously missing pieces of information are presented that help solve the fluoride puzzle. The first of these items (pp 261-266) summarizes a report by Joel Griffiths and Chris Bryson about recently declassified US government documents revealing a close connection between water fluoridation and the Manhattan Project of World War II to build the atomic bomb. The other item (pp 270-271), drawn from an Internet contribution, reveals and corrects a long-standing computational error concerning the daily fluoride intake found by Kaj Roholm to produce various stages of skeletal fluorosis.

Ever since its inception over fifty years ago, water fluoridation has caused many to wonder why the effort to promote it has been so intense and why opposition to it has been treated so harshly. Griffiths and Bryson shed light on this question by documenting a long-suspected link between fluoridation and official US interests involving far more than public health policy. Ironically, the key individual at the heart of fluoride toxicology studies during the Manhattan Project was also responsible for the calculation error about the daily fluoride intake needed to cause skeletal fluorosis.

During production of uranium hexafluoride for isotope separation and enrichment to make atomic weapons, serious neurotoxic effects became evident among workers exposed to the fluoride chemicals. Heading up the investigation of these effects was the late Professor Harold C Hodge of the University of Rochester, New York. Hodge called for research on central nervous system (CNS) effects of fluoride, and this research probably took place, but the reports are missing from the repository archives. Also, strangely missing from the files is the transcript of a high-level conference on fluoride metabolism held during World War II, whose participants included not only Hodge but also Dr David B Ast, director of the Newburgh NY fluoridation project, and Dr H Trendley Dean, the "father of fluoridation" in the US Public Health Service.

In 1944 air-borne fluoride emissions from the du Pont company's classified uranium hexafluoride operations in New Jersey laid waste to farms downwind, blighting orchards and causing serious illness in both animals and humans. Hodge and Ast investigated the damage and agreed that it was serious. After the war the farmers filed lawsuits, creating consternation at the highest levels of the US government, and prompting strenuous efforts by federal officials to prevent these suits from succeeding. For his part, Hodge urged telling the public that fluoride is good for the teeth, and lectures for this purpose were then given throughout the country and elsewhere.

The need for research on long-term effects of small daily doses of fluoride led to Hodge chairing a committee that recommended the Newburgh NY pilot fluoridation study, which was headed by Ast and secretly steered by Project F (fluoride) A-bomb scientists. Blood and placenta samples from Newburgh citizens

were sent to Rochester for testing under the direction of Hodge, but none of these connections to the Manhattan Project were ever disclosed until now.

The Internet presentation describes the unit conversion error made by Hodge in 1953 in his extrapolation of Roholm's data on the daily fluoride intake per body weight found to produce skeletal fluorosis in Danish cryolite workers in the 1930s. Long unrecognized and widely cited, Hodge's incorrect calculation has given a false sense of safety about many typical levels of fluoride exposure and intake. Only in 1979 did Hodge finally publish a corrected version of his calculation, but the erroneous figure is still quoted.

After his retirement, Hodge continued to serve in advisory roles on fluoride research. At the Forsyth Dental Research Institute in Boston, where Dr Phyllis Mullenix was investigating CNS effects of fluoride in rats, Hodge visited her as a consultant, but he never mentioned his previous engagement in such research on fluoride neurotoxicity in connection with the Manhattan Project.

In the light of what we now know about the US government's desire to minimize concern over the toxic effects of fluoride in order to protect the Manhattan Project, it is understandable but not excusable that dental health authorities cooperated to mislead a trusting public. Unfortunately, we still do not have the whole story, because freedom of information requests for numerous additional important fluoride documents have been stonewalled. Moreover, the principal player in this drama, Harold Hodge, is no longer alive to answer many of the remaining questions. Even so, we can remain confident that what is true about fluoride will be "truth to the end of reckoning."

Bill Wilson
Editorial Assistant

[We apologize to Society members and readers for the late appearance of this "November" issue, caused by the illness and hospitalization, following a tragic family bereavement, of our usual editor, Dr John Colquhoun. With the help of the co-editors it has been published. I accept responsibility for any shortcomings. B W]

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CHARACTERISTICS OF BONE CHAR RELATED TO EFFICACY OF FLUORIDE REMOVAL FROM HIGHLY-FLUORIDATED WATER

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Bangkok, Thailand and New York, USA

SUMMARY: Bone char, prepared from commercially available bone meal by steaming, drying and calcining at 600°C, is one of the systems used to reduce the levels of fluoride (F) in drinking water to less harmful levels. The purpose of this study was to determine factors which will increase the efficacy of bone char to remove F from fluoridated water. Polyvinyl chloride pipes were prepared with bone char calcined at different temperatures (400, 600, 800°C). Bone char regenerated using either an acid or alkaline method was also used to prepare the columns. Naturally fluoridated water in Thailand was poured at the top of the column and defluoridated water (effluent) collected at the bottom. The F, calcium and phosphate contents and the pH of the effluent were determined. Bone char materials, before and after the processes of defluoridation, were characterized using x-ray diffraction and infrared absorption spectroscopy. Results demonstrated: 1) the capacity of the bone char to remove F was inversely related to the crystallinity (reflecting crystal size) of the bone apatite which in turn was related to the calcination temperature (*i.e.*, the higher the temperature of calcinations, the greater the crystallinity of the bone apatite; the greater the crystallinity the lower the capacity); 2) the alkaline method of regeneration was more efficient than the acid method in terms of the amount of F removed from the bone char; 3) the crystallinity of the bone char increased after defluoridation procedure. It is concluded that the removal of F from the fluoridated water may occur by a dissolution-reprecipitation process resulting in the formation of (F,OH)-apatite. This reaction may be a major mechanism operating during the defluoridation of water using bone char. The results suggest that heating at low temperature (< 600°C) and regeneration using the alkaline method will maximize the efficacy of bone char in removing F from naturally fluoridated water.

Key words: Apatite; Bone; Defluoridation.

INTRODUCTION

The caries-inhibiting effect of F ions is the rationale for the Public Health program of water fluoridation with 1 ppm fluoride (F) in several countries, especially the United States.[1] However, reported incidences of dental and skeletal fluorosis have been attributed to levels of F ions in drinking waters.[2-7]

Several defluoridating systems have been developed to reduce the levels of F in drinking water to less harmful levels. These systems have included: lime softening, alum, alum and lime, activated alumina, activated carbon, natural bone, bone char, bone char and charcoal, synthetic bone (mixture of tricalcium phosphate and hydroxyapatite), bauxite, ion-exchange resins, electrodialysis, and osmosis.[5,8-10] Evaluation of the efficacy of these systems in terms of capacity for F removal, commercial availability, cost-effectiveness, maintenance requirements, feasibility for big and small water plants, indicated the superiority of

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bone char compared to the other systems.[5] The filtration system using bone char was modified and improved by Phantumvanit *et al*[10] and this system has been recommended for domestic use.

A visual examination of the bone char used for the modified procedure showed non-uniformity in the color of the bone particles: ranging from black, dark and light shades of grey, and white. This non-uniformity in color suggests non-uniformity in the temperature of heating probably due to unequal heat distribution in the large oven.

The purpose of this study was to determine factors which will increase the efficacy of bone char to remove F from fluoridated water. The factors investigated were the calcining temperature (*i.e.*, effects of heat treatment on the physicochemical properties of the bone char) and the regeneration method (acid *vs* alkaline).

MATERIALS AND METHODS

Preparation of the bone char: Commercially available bone meal, usually sold for use as fertilizer or animal feeding, was used for this study. The bone meal consisted of mixed cow and pig bones, crushed to particle sizes ranging from 40 to 60 mesh, steamed and dried. For use in defluoridating system, the bone meal is usually calcined in a high temperature electric furnace at 600°C for 30 minutes.[10]

Preparation of the defluoridator column: The column is prepared as schematically described in Figure 1. The column measuring 75 cm long and 9 cm in diameter is prepared from a piece of polyvinyl chloride pipe. The filter column is prepared by placing in a plastic bag (measuring 10-12 cm in diameter and 80 cm in length) the following: bottom layer of crushed charcoal (300g), middle layer of bone char (1000g), covered with a top layer of approximately 200g of clean pebbles to prevent floating of the bone char. A hole is cut at each of the two corners at the base of the plastic bag to allow passage of the filtered water on to the receptacle.

Preparation of column using bone char calcined at different temperatures: Visual analyses of the bone char after calcining at 600°C for 30 minutes showed non-uniformity of color, varying from black to gray to white, although most areas were gray. This color variation was attributed to the possibility of uneven heating in the oven.[11] To determine the effect of calcination temperature on the efficacy of the bone char to remove F from the drinking water, several columns were prepared using bone meal sintered at 400°C, 600°C and 800°C.

Defluoridation procedure: Naturally fluoridated water from a well source is poured at the top of the column and defluoridated water (effluent) is collected at the bottom (Figure 1). The flow rate through the column was four liters of fluoridated water per hour.

Regeneration of the bone char: Regeneration of the bone char was made by either an acid or alkaline method.[12] The **acid method** consisted of passing through the column three liters of an acid (pH 3) solution containing 0.14M $CaCl_2$ + 0.08M NaH_2PO_4 for each cycle.[13] The **alkaline method** consisted of passing through the column four liters of a basic (pH 12) 1N KOH solution for

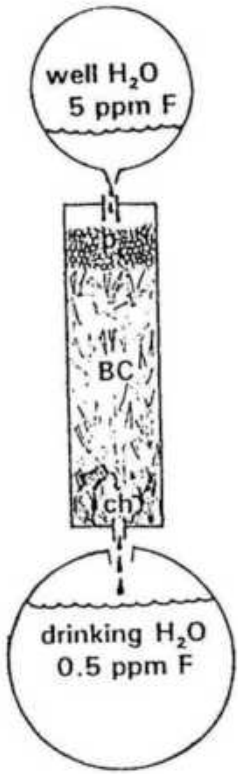

FIGURE 1. Schematic representation of the defluoridator system consisting of a defluoridating column containing small pebbles (p), bone char (BC) to remove the fluoride ions and activated charcoal (ch) as deodorizer. After going through the defluoridator system, the fluoridated well water containing 5 ppm F resulted in drinkable water with 0.5 ppm F.

each cycle. After each regeneration cycle, the concentrations of F, calcium and phosphate ions, and the pH of the effluent were determined. Following each regeneration cycle, either acid or alkaline, the column is washed with twenty liters of F-free water to remove either the extreme acidity or basicity of the solutions used for regeneration.

Analyses of the bone char: Bone meal calcined (heat-treated) at different temperatures (400, 600, 800°C) were analyzed before and after defluoridation procedures and before and after regeneration, using x-ray diffraction and infrared (IR) absorption spectroscopy. X-ray diffraction analyses were made using Phillips APD3520 x-ray diffractometer, x-rays generated from a copper target at 40 kV and 20 mA using Ni filter, slit system, scanning speed at 1/8 deg 2θ per minute, scanning from 25 to 35 deg 2θ. Line-broadening of the apatite (002) diffraction peak was taken as an indication of crystallinity (reflecting crystal

size): the broader the peak, the smaller the crystal size.[14] IR analyses were made on KBr pellets prepared by mixing together 2 mg of powdered bone char with 300 mg of KBr (IR grade) and pelletized using 12,000 psi. IR analyses were made on Perkin Elmer 983 double grating IR spectrometer, scanning frequency range from 4000 to 400 cm-1. Identification of phases were made according to earlier IR studies on apatites and related calcium phosphates.[15-18]

Fluoride analyses: Fluoride concentrations of the water before and after passing through the defluoridator column and before and after regeneration either by acid or alkali methods were determined using F ion selective electrode. Fluoride content of bone char after defluoridation and after two methods of regeneration were determined by dissolving the bone char in perchloric acid and analyzing for F with the ion-selective electrode using appropriate F standards.

RESULTS

Physicochemical properties of the bone char materials. Color changes observed after calcining or heating the bone meal at different temperatures were: black (ignited at 400°C); grey (ignited at 600°C); white (ignited at 800°C). All these colors were represented in the bone char materials prepared for the defluoridation system in the original study.[10] These color changes were similar to those observed upon heat-treatment of dentin.[8] X-ray diffraction (XRD) patterns showed differences in crystallinity as reflected by the broadening of the diffraction peaks (Figure 2): XRD of materials calcined at 800°C. (Figure 2D) showed the highest crystallinity, reflecting larger crystal size, compared to the untreated bone and those treated at lower temperatures (Figures 2A, 2B, 2C). No significant difference in crystallinity was observed between bone before and after calcining at 400°C (Figures 2A, 2B) except for lower background after calcination. The greater intensity and narrower apatite diffraction peaks of materials heated at 800°C reflects the crystal growth of bone apatite induced by heat treatment.[17]

The IR spectra of the bone meal materials before and after heat-treatment at 400°C were not significantly different in terms of the intensity of the CO_3 absorption bands. However, loss of the organic component (represented by the N-H absorption bands in the unheated bone) after heat-treatment at 400°C was apparent (Figure 3). The intensity of the CO_3 absorption bands (reflecting CO_3 concentration in the bone apatite) decreased with increasing temperature of calcination. For example, the intensity of the CO_3 absorption bands in the IR spectra of bone char heated at 800°C was lower than those in bone char heated at 600°C or at 400°C (Figure 3). The loss of CO_3 with heat treatment has also been observed with human enamel and dentin specimens.[8,17,19,20]

The XRD analyses of the bone char before and after acid or alkaline regeneration showed sharper diffraction peaks in the XRD patterns of bone char after acid regeneration compared to those after alkaline regeneration (Figure 4). No significant difference was observed between the XRD patterns before and after alkaline regeneration (Figures 4A *vs* 4C); while considerable differences were observed between the XRD patterns before and after acid regeneration (Figures 4B *vs* 4C).

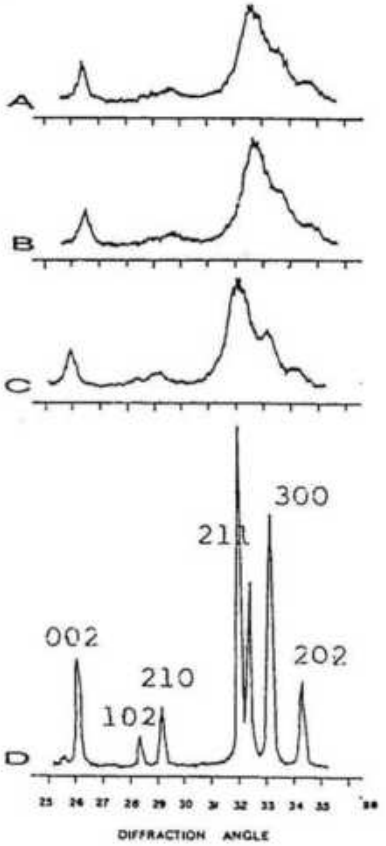

FIGURE 2 (above) X-ray diffraction (XRD) patterns of bone meal used in defluoridation system. Bone meal before calcination (A); after calcination at 400°C (B); 600°C (C) and 800°C (D). The XRD patterns demonstrate increase in crystallinity with increase in temperature of calcination reflecting increase in crystal size and decrease in surface area. The corresponding color changes observed were: (A) beige; (B) black; (C) grey; (D) white.

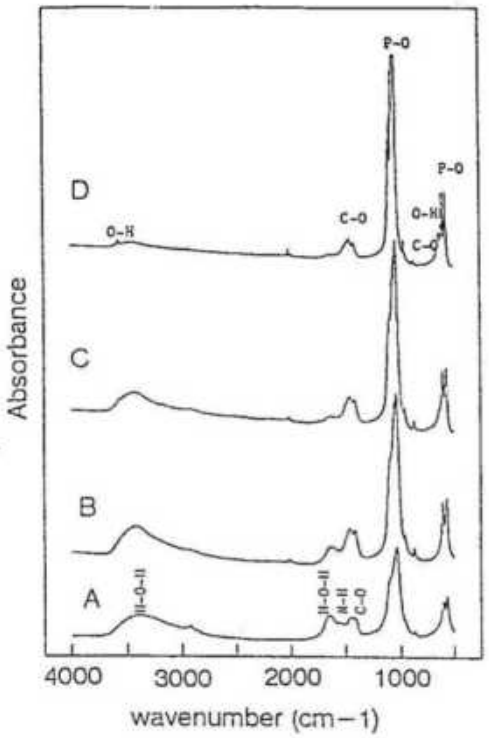

FIGURE 3 (top right) Infrared (IR) absorption spectra of bone meal used in defluoridation system before (A) and after calcination at 400°C (B); 600°C (C); and 800°C (D). The loss of the organic component is indicated by the disappearance of the N-H absorption bands (B vs A); the decrease in CO_3 content with calcination is demonstrated by the decreasing absorption intensity of C-O in (C) and (D) compared to (A) or (B).

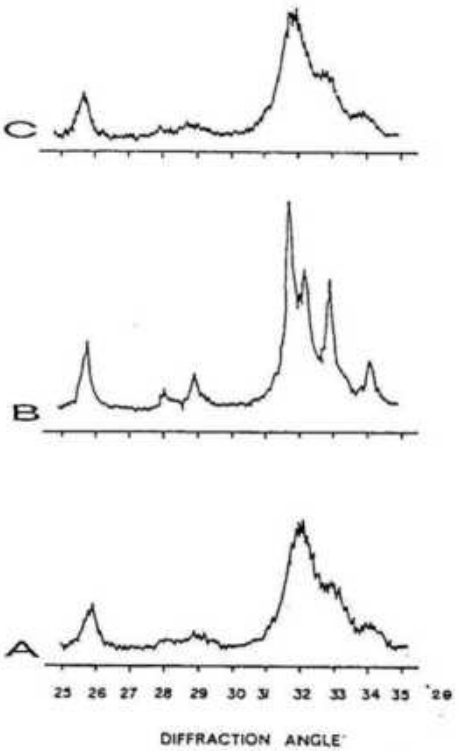

FIGURE 4 (right) XRD patterns of bone char before (A) and after acid (B) or alkaline (C) regeneration.

Similarly, differences were observed in the IR spectra of the bone char materials before and after acid and alkaline regeneration (Figure 5), principally in the intensity of the CO_3 absorption bands and in the resolution of the PO_4 absorption bands. The IR spectra of the bone char after acid regeneration, showed lower intensity of the CO_3 absorption bands and higher resolution of the PO_4 absorption bands. The lower ratio of the intensities of the CO_3 to PO_4 absorption bands indicate a decrease in CO_3 content of the bone apatite after acid regeneration of the bone char. The increase in resolution of the PO_4 absorption bands suggest an increase in crystallinity of the bone apatite after acid regeneration.

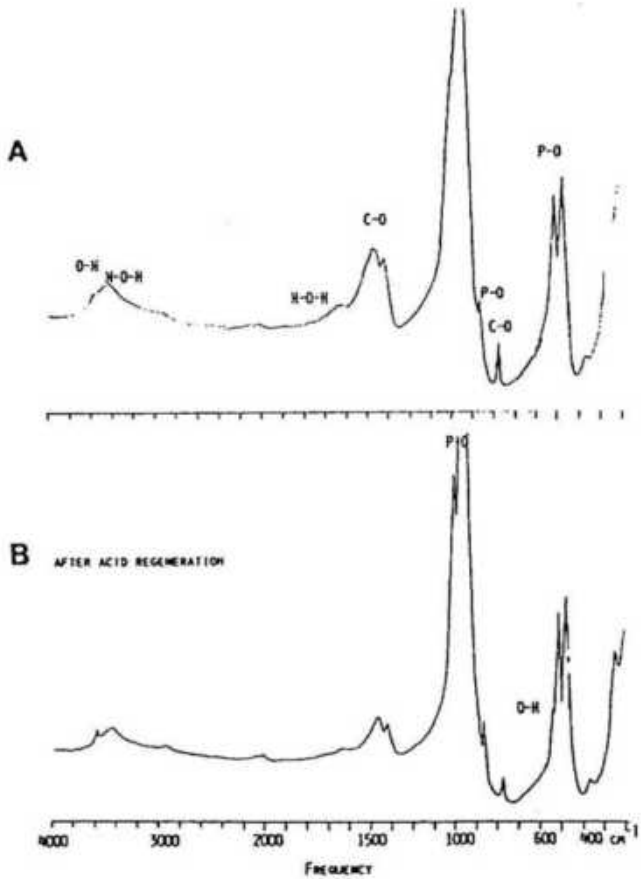

FIGURE 5. IR spectra of the bone char material before (A) and after (B) acid regeneration showing loss of CO_3 component of the bone apatite after acid regeneration suggesting dissolution and reprecipitation.

Capacity to remove fluoride from the drinking water. The bone char materials prepared by calcination (heat-treatment) at different temperatures were shown to differ in their capacity to remove fluoride ions from the drinking water in terms of the number of liters of water which was defluoridated decreasing from a level about 5 ppm F before defluoridation to less than 1 ppm F after defluoridation (Figure 1, Table 1). Bone char calcined at 400°C (black) showed the highest capacity (560 liters of water); followed by the material calcined at 600°C (480 liters); while the material calcined at 800°C (white) showed the least (70 liters). The difference in the capacity between bone char prepared at 400°C and that at 600°C is about 15%, while the difference between the capacity of the bone char prepared at 400°C to that at 800°C is about 88%.

Table 1. Defluoridation capacity of bone char calcined at different temperatures

Bone Char Color	Calcination Temp (°C)	Defluoridation Cap (liters of 5 ppm F)
Black	400	560
Grey	600	480
White	800	70

Fluoride uptake: Analyses of fluoride content of the bone char after defluoridation showed that the capacity of the bone char material to remove fluoride from the drinking water was significantly influenced by the calcination or heat-treatment temperature (Table 2). The highest F uptake was observed in bone char material calcined at 400°C (0.56 wt% F) compared to that calcined at 600°C (0.46 wt% F) or at 800°C (0.06 wt% F). It was also observed that F uptake for the black bone char (prepared at 400°C) was highest at the upper layer compared to the middle and bottom layers of the bone char region (0.65 *vs* 0.52 %F); while no difference in the F uptake between upper and bottom layers were observed for the grey bone char (prepared at 600°C).

Table 2. Fluoride content (wt%) in bone char: after saturated defluoridation

Bone Char	F-content (%) in layers			
	Upper	Middle	Lower	Average
Black	0.65	0.52	0.52	0.56
Grey	0.47	0.46	0.46	0.46
White	0.06	0.06	0.06	0.06

Regeneration of bone char. The method of regeneration affected the efficiency to remove F from the bone char. An average of 78% of the F was removed using the alkali method compared to an average of 52% using the acid method after the first regeneration cycle (Table 3). In both methods the percentage of F removed was much less from the upper layer compared to that from the bottom layer of the bone char. However, although the alkali method removes more F from the bone char, this method permitted only 4 regeneration cycles before the bone char was completely saturated. In comparison, the acid method permitted 5 regeneration cycles.[12]

TABLE 3. Effect of regeneration on f content of bone char (BC)

Bone char		mgF/gBC	% F removed
before defluoridation		0.26	
after defluoridation		4.68	
after KOH regeneration:			
	top layer	1.42	69
	bottom layer	0.76	83
after acid regeneration:			
	top layer	3.61	23
	bottom layer	1.20	74

% F removed was calculated as follows: for KOH regeneration (1.42/4.68) x 100 = 69%; for acid regeneration: (3.61/4.68) x 100 = 23%.

Calcium, phosphate, fluoride concentrations and pH of effluent. Determination of calcium, phosphate and fluoride content of the effluent collected after acid regeneration showed higher levels of calcium and phosphate ions compared to the levels of these ions after alkaline regeneration (Table 4). This difference is expected because of the high concentrations of calcium and phosphate ions present in the acid solution used for regeneration. The F concentration were almost 50% higher in the effluent from the acid regeneration (4 ppm F) compared to those from the alkaline regeneration (2.4 ppm F). Calculations from the amounts of calcium and phosphate ions in the effluent after acid regeneration showed considerably lower Ca/P molar ratios than the original Ca/P molar ratio in the acid solution. The pH of the water after defluoridation changed from 7.5 to 7.7. The concentration of F ions decreased from 5 ppm to less than 1 ppm. No difference in the calcium and phosphate ions present in the fluoridated water was observed before and after defluoridation.

TABLE 4. Calcium (Ca), phosphate (P) and fluoride (F) contents (in ppm) and pH of the effluent after acid or alkaline regeneration of bone char

	Ca	P	F	Ca/P
Acid wash	78	92	4	0.66
Alkaline wash	0.4	0.008	2.4	-
Acid solution for regeneration	5.6	7.6	-	1.75

DISCUSSION

Bone char was shown to be an efficient system for defluoridation of water supply to make it safe for public consumption.[5,9] Particle size was shown to affect the efficiency of F removal in terms of F uptake of the bone char.[9] In this and a previously reported study,[11] color changes were shown to be related to calcination temperature: black (400°C), grey (500°C) and white (800°C). The different colors observed in bone char materials used in defluoridating system indicated that the temperature distribution in the oven is uneven, resulting in uneven heating of the bone meal. This study demonstrated that calcination temperature affects the crystallinity of the bone char material (Figure 1) which, in turn, affected its capacity to remove F ions from the fluoridated water: the lower the calcination temperature, the higher the capacity (Tables 1 and 3). This observation, reported by us earlier,[11] has been confirmed by recent subsequent observations.[21-23] The lower crystallinity (reflecting smaller crystal sizes) of the apatite in bone char heat-treated at 400°C and 600°C provide a much greater surface area for the absorption of F ions from the water and allows surface reactions to take place to a greater extent than apatite in bone chars heated at 800°C. That this is indeed the case is demonstrated by the higher F uptake and larger capacity (to remove F from the fluoridated water) of bone char materials heated at 400°C and 600°C compared to that of material heated at 800°C (Tables 1 and 2). Heat-induced changes in crystal property of apatite in bone char is more evident between 400°C and 800°C and between 600°C and 800°C than between 400°C and 600°C.

Improvement in crystallinity of the bone char after defluoridation suggests that a reaction has taken place between the bone apatite (which is principally a carbonate-apatite)[16-18] and the F^- ions from the fluoridated water. This reaction caused the formation of fluoridated apatite, (F,OH)-apatite, possibly on the surfaces of the bone char particles. A similar improvement in crystallinity of calf bone which has been suspended in F-containing solution has been previously observed.[24] It is possible that the effect on crystallinity would be more pronounced if it was possible to analyze only the materials on the surfaces of the bone char. In this study, materials for the XRD analyses included both the newly formed (F,OH)-apatite crystals on the surfaces of the bone char particles and the greater population of bone apatite crystals below the surfaces possibly containing much smaller amounts of F after defluoridation. The change in crystallinity was more pronounced in bone char materials obtained from the middle and bottom layers compared to that obtained from the top layer. The F concentration in the water presumably decreases as it flows down the defluoridator column.

The much higher crystallinity of the apatite in bone char after acid regeneration (Figure 4) suggests that dissolution and precipitation processes have taken place similar to previous observation on the dissolution of synthetic carbonate-containing F^- and OH-apatite.[25,26] The evidence for the dissolution is the high levels of calcium and phosphate ions obtained in the effluent after acid regeneration after accounting for the original concentration of these ions present in the acid solution used for regeneration (Table 3). The evidence for precipitation is

the considerable difference in crystallinity observed in the XRD patterns and IR spectra of the bone char before and after regeneration (Figsures 4 and 5). The lower CO_3 content (indicated by the lower intensity of the CO_3 absorption bands) of the acid-regenerated bone char suggests the dissolution of CO_3-rich apatite and precipitation of CO_3-poor apatite.[25,26] This precipitation is due to the reaction among the calcium and phosphate ions (present in the acid solution used for regeneration and produced by the partial dissolution of the bone apatite) and the F ions from the water. It is known that F ions facilitate the formation of $(F,OH)^-$ or F-apatite even under very acid conditions which normally would promote the formation of acid calcium phosphates, *e.g.*, dicalcium phosphate dihydrate, DCPD.[17] Based on the fact that bone mineral is principally a carbonate hydroxyapatite,[16,17] the dissolution/precipitation reaction involved in water defluoridation using bone char may be represented according to the reaction below:

$$(Ca,Mg,Na,X)_{10}(PO_4,HPO_4,CO_3)_6(OH,Cl)_2 \text{ ---(1)--->}$$

(bone char apatite)

$$Ca^{2+} + Mg^{2+} + Na^+ + X^+ + HPO_4^{2-} + PO_4^{3-} + HCO_{3-} + OH^- + Cl^- + {}^*F^- \text{ --(2)-->}$$

(in solution) (*from water)

$$Ca_{10}(PO_4)_6(F,OH)_2 \quad (\pm CaF_2)$$

(F,OH)-apatite

The initial reaction (1) involves a partial dissolution of bone apatite under acid conditions; the second reaction (2) involves a reaction with the F ions from the fluoridated water resulting in the precipitation of (F,OH)-apatite which can form under acid or basic conditions.[16,20] CaF_2 may also form, depending on the F^- ion concentration.[17,21,25]

The formation of calcium fluoride, CaF_2, or 'CaF_2-like' materials have been observed *in vitro* and *in vivo* when enamel, dentin or synthetic apatite react with high levels of fluoride ions.[27-29]

In these water defluoridation experiments, CaF_2 was not observed using x-ray diffraction analysis. However, small amounts of CaF_2, especially 'CaF_2-like' materials of low crystallinity may easily escape detection by x-ray diffraction analyses. The higher amount of fluoride removed by the alkaline method suggests that CaF_2 or 'CaF_2-like' materials may have been formed since CaF_2 is known to be soluble in KOH.[30] F uptake resulting from surface ion exchange or adsorption may add to the high amounts of F removed by the alkaline (KOH) method.

The alkaline method of regeneration was observed to be more efficient than the acid method in terms of amount of fluoride removed from the bone char. However, in terms of the number of times the bone char in the column can be reused for defluoridation, the acid method of regeneration appeared to be more efficient. This may be due to the fact that the acid method promotes the removal of F from water by precipitation according to the reaction described above.

The present study, which is an extension of preliminary studies reported,[11] suggests that the formation of (F,OH)-apatite on surfaces of the bone char

particles may be a major mechanism operating during the defluoridation of water using bone char. Evidence for the formation of (F,OH)-apatite is the improvement in crystallinity of the bone apatite [24] coupled with F-uptake of the bone char (Table 3). Reactions resulting in the formation of (F,OH)-apatite have also been suggested in studies[21-23] reported subsequent to our initial report[11]. The experimental defluoridation system reported by Pearce and Larson[21,23] involved DCPD instead of bone char.

The lower Ca/P ratio of the effluent compared to that in the regenerating solution after acid regeneration (Table 4) suggests that some of the calcium and phosphate ions from the solutions were used in the precipitation of (F,OH)-apatite.[17,31]

This study suggests that the efficiency and efficacy of the bone char defluoridation system in removing F from fluoridated water may be maximized with uniform heating of the bone meal at lower temperature (<600°C) and regeneration of the char may be achieved by either the alkaline or acid method.

ACKNOWLEDGMENTS

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ENDEMIC FLUOROSIS IN SAN LUIS POTOSI, MEXICO IV. SOURCES OF FLUORIDE EXPOSURE

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SUMMARY: The high prevalence of dental fluorosis in San Luis Potosi (SLP) cannot be attributed only to the fluoride concentrations in drinking water. The present work was carried out to study fluoride levels in different kinds of beverages. This work assumes that these beverages were contaminated with fluoride as a result of using water with high fluoride content in their production. Different brands of bottled juice, bottled water and soft drinks were analyzed. Some brands showed higher fluoride levels than others. However, the most popular brands for bottled juice and bottled water were among those having the highest fluoride level. With these results, and applying the risk factor of boiling the water (which increases the fluoride concentration in direct proportion to the loss of volume), exposure doses to fluoride were estimated. The dose estimated for infants in SLP in their first semester of life was 0.31 mg/kg/day, and that for infants one year old was 0.26 mg/kg/day. These estimations agree well with the fact that infants born in SLP are heavily exposed to fluoride during the first year of their life. Also, they explain the excessive prevalence of dental fluorosis in areas of SLP with low levels of fluoride in drinking water.

Key words: Beverages; Fluoride content; Fluoride doses; Fluorosis.

INTRODUCTION

It has been shown that the drinking water consumed in the city of San Luis Potosi (SLP), Mexico, contains excessive quantities of natural fluoride.[1] Results obtained by the analysis of tap water showed that 61% of the samples collected in SLP had fluoride levels above the range of 0.7-1.2 ppm.[1] However, the high prevalence of dental fluorosis in SLP cannot be attributed only to the levels of fluoride in drinking water, as the prevalence of severe dental fluorosis is increased even in areas with normal fluoride concentrations.[1-2] Other risk factors must be considered in the case of SLP.[1-3]

Ambient temperature can play an important role. The mean annual temperature in SLP is 24.0°C. Thus a higher ingestion of water can be expected. Nevertheless, an increased ingestion does not totally explain the increment in dental fluorosis, since the ingestion of drinking water in SLP would need to be 2-3 times higher than normal, and 24.0°C is not a temperature that can be related to this increased ingestion of water.

The boiling of tap water is a method currently used for disinfection. Direct measurements have shown that, in boiled water, fluoride levels increased proportionally to the loss of volume.[1] Additionally, 91% of the population in SLP uses boiled water for reconstituting infant milk formulas.[1] This has been shown to increase the risk of dental fluorosis.[4,5] Food preparation with boiled tap

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water could also be identified as another source of fluoride. It has been reported that 92% of the SLP population prepared their food using tap water.[1]

Ambient temperature and the practice of boiling the tap water may be important factors that increased the exposure to fluoride. However, there is a necessity of studying other sources of fluoride in SLP, more related to the presence of the mineral in drinking water. Among these, in the present work we studied soft-drinks, bottled juice, and bottled water.

METHODS

Bottled water, bottled juice and soft drink samples were collected at different grocery stores all located in SLP. The most popular brands were analyzed in all cases. Fluoride levels were quantified by adding TISAB buffer to the samples just prior to the analysis with a sensitive specific ion electrode. As an internal quality control program, primary standard reference material was analyzed and spiked samples were examined in blind analysis. Our fluoride recovery was 104%.

The exposure doses were calculated using the following generic equation:

$$ED = \frac{C \times WI}{BW}$$

Where:

ED = exposure dose (mg/kg/day)
C = fluoride concentration (mg/L)
WI = water intake (L/day)
BW = body weight (kg)

The standard values that were useful in estimating exposure are shown in Table 5. For the calculations, we assumed chronic exposure and total bioavailability of fluoride in water.

RESULTS

Fluoride concentrations in bottled juice are presented in Table 1. It is shown that those having the higher fluoride levels were the most popular brands for infant consumption (brands 2 and 3). The rest of the brands were within the Mexican regulation for fluoride in bottled water of 0.7 mg/L.[6] Among the brands of bottled water (Table 2), the one most consumed in SLP was the only one above the Mexican regulation for bottled water. Fluoride levels in the rest of the brands were within the national guideline, even though in the upper limit of their respective range. Soft drinks were also analyzed for fluoride content. Results are shown in Table 3. Fluoride concentration in the most popular brand was lower than 0.7 mg/L. However, soft drinks distributed by two companies (number 1 and 2 in Table 3), had fluoride levels well above the Mexican regulation. Furthermore, the levels of fluoride in the soft drink with the highest concentration had remained stable between 1991 and 1996 (Table 4). Fluoride concentration did not depend on the flavor of the soft drink, as different presentations had the same fluoride level within a company. Therefore, the fluoride content in a soft drink is a condition for the fluoride level in the water source used by each company. With these data, the exposure doses to fluoride were calculated for infants and children in SLP (Table 5). The highest exposure dose was estimated for infants in their first semester of life (6 kg infant), and the dose decreased only 15% in infants one year old (10 kg infant). The lowest dose was calculated for those children exposed to the most popular brands of soft drinks and bottled water.

Table 1. Fluoride levels in bottled juice collected in SLP

Brand	No. of samples	Arithmetic mean	Standard deviation	Range
1	3	0.24	0.01	0.23 - 0.26
2*	4	2.57	0.31	2.28 - 2.87
3*	4	1.72	0.25	1.41 - 1.94
4	3	0.39	0.19	0.38 - 0.41
5	4	0.32	0.07	0.22 - 0.40

* Most popular brands for infant consumption. All the samples were apple juices.

Table 2. Fluoride levels in bottled water collected in SLP

Brand	No. of samples	Mean	S.D.	Range
1	4	nd	-	nd
2	4	nd	-	nd
3	4	0.29	0.01	0.27 -0.30
4*	4	0.93	0.09	0.84 - 1.01
5	4	0.31	0.01	0.30 -0.32
6	4	nd	-	nd
7	4	0.28	0.01	0.27-0.30

* Most popular brand. Tables 2-4: Mean = Arithmetic mean. S.D. = Standard Deviation.

Table 3. Fluoride levels in soft drinks from different companies located in SLP

Brand	No. of samples	Mean	S.D.	Range
1	6	3.56	0.10	3.36 - 3.71
2	5	3.04	0.14	2.85 - 3.21
3	4	0.36	0.01	0.35 - 0.37
4*	6	0.40	0.07	0.33 - 0.56

* Most popular brand.

Table 4. Fluoride levels in soft drinks from one company in SLP, 1991 and 1996

Brand	No. of samples	Mean	S.D.	Range
1991	5	3.73	0.05	3.64 - 3.81
1996	6	3.56	0.10	3.36 - 3.71

Table 5. Estimation of exposure doses for fluoride in SLP

Example	Source of water	L/day	Fluoride (mg /L)	Fluoride intake (mg/kg/day)	Total fluoride intake (mg/kg/day)
Infant (6 kg)	boiled water[1]	1.00	1.86[a]	0.31	0.31
Infant (10 kg)	juice	0.50	2.57[b]	0.128	0.26
	boiled water[1]	0.75	1.86[a]	0.139	
Children (20 kg)	soft drink	0.25	3.56[c]	0.044	0.10
	bottled water	0.75	0.93[d]	0.034	
	boiled water[2]	0.25	1.86[a]	0.023	
Children (20 kg)	soft drink	0.25	0.40[e]	0.005	0.06
	bottled water	0.75	0.93[d]	0.034	
	boiled water[2]	0.25	1.86[a]	0.023	

[1] The source of boiled water in infants is the bottled water used in the reconstitution of milk formulas. [2] The source of boiled water in children is the water used in soup preparation. [a] Considering that in boiled water fluoride levels increase proportionally to the loss of volume[1], the concentration in the most popular brand of bottled water (Table 2) was doubled. [b] From Table 1 in this work. [c] The highest fluoride concentration found in a soft drink (Table 3). [d] Fluoride level in the most popular brand of bottled water (Table 2). [e] Fluoride concentration in the most popular brand of soft drink (Table 3).

DISCUSSION

In some areas of SLP, the high prevalence of severe dental fluorosis cannot be assigned only to the exposure to fluoride in drinking water.[1,2] For example, a prevalence of 18.7% for severe fluorosis was found in children living in areas with fluoride levels in tap water lower than 0.7 mg/L.[1] Therefore, risk factors or the presence of additional sources of fluoride might explain these results.

The present work was designed in order to study fluoride levels in different beverages. The work assumes that these beverages were contaminated with fluoride as a result of using water with high fluoride content in their production.

The data obtained by analyzing different brands of bottled juice (Table 1), bottled water (Table 2), and soft drinks (Table 3), were variable. Some brands show higher fluoride levels than others. However, the most popular brands of bottled juice and bottled water were among those having the highest fluoride level.

With all these results, and applying the risk factor of boiling the water,[1] exposure doses to fluoride were estimated for different situations (Table 5). The dose estimated for infants in SLP in their first semester of life was 0.31 mg/kg/day; which is equivalent to the dose for a 20 kg child drinking one liter per day of a water source with fluoride levels of 6.0 mg/L. Following a similar comparison, the exposure dose calculated for infants one year old was 0.26 mg/kg/day, which is equivalent to the dose for a 20 kg child exposed to a water source of 5.0 mg/L. These estimations agree with the idea that infants born in SLP are heavily exposed to fluoride during the first year of their life. Therefore, these results explain the excessive prevalence of dental fluorosis in areas of SLP with low levels of fluoride in drinking water.

In conclusion, risk factors such as preparing food with boiled water and consuming some beverages prepared with water containing high fluoride concentrations, might explain the prevalence of dental fluorosis in SLP. Therefore, in SLP a preventive health program needs to be applied for the control of fluorosis. In addition to controlling the levels of fluoride in drinking water, there is a necessity to reduce fluoride levels in other sources such as bottled water, soft drinks and bottled juice mainly consumed by infants.

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FLUOROSIS IN SOME TRIBAL VILLAGES OF DUNGARPUR DISTRICT OF RAJASTHAN, INDIA

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SUMMARY: The prevalences of dental and skeletal fluorosis were observed in fifteen tribal villages of the Dungarpur district of Rajasthan where the fluoride (F) concentration in drinking waters varies from 0.3 to 10.8 ppm. At 1.40 and 6.04 ppm mean F concentrations, 25.64% and 84.43% of school children (<16 years), and 23.91% and 96.87% of adults, respectively, were found to be affected with dental fluorosis. The maximum prevalence of dental fluorosis (76.43%) was observed in the 17 to 22 years age group. No significant correlation was found between dental fluorosis prevalence and gender. At these same F concentrations, 4.35% and 63.02% of adults (>21 years), respectively, were showing evidence of skeletal fluorosis. The prevalence of skeletal fluorosis was comparatively higher in males and increased with higher F level and age. None of the fluorotic subjects showed evidence of genu valgum syndrome or goitre. Radiological findings of other deformities in fluorotic subjects were also found.

Key Words: Dental fluorosis; Dungarpur district; Rajasthan; Skeletal fluorosis.

INTRODUCTION

Fluorosis is a world wide health problem and is endemic in those areas where the F content is high in drinking waters. In India, fluorosis is endemic in more than fifteen states.[1,2] In the state of Rajasthan more than 50% of the districts have high F levels (0.1 to 18.0 ppm) in their drinking/ground waters.[3-5] From southern Rajasthan, where tribal population is predominant, 0.3 to 10.8 ppm F concentration has also been reported.[6-8] Apart from a few studies,[9-13] no extensive surveys on endemic fluorosis have been conducted in Rajasthan state. Therefore, the present study was undertaken to determine the prevalences of dental and skeletal fluorosis at different F concentrations, ages and sex. Radiological examinations for other skeletal deformities in fluorotic subjects, and checks for goitre, were also carried out.

MATERIALS AND METHODS

The fifteen villages (Table 1) of Dungarpur Panchayat Samiti in Dungarpur district (Rajasthan) were selected as they have relatively high F concentrations in their drinking water, and the majority of the population is tribal.[6-8] Teeth of school children (<16 years) and adults, of both sexes, were examined for dental fluorosis. Mottling grade II was considered as a definite index of the prevalence of dental fluorosis.[14] Adults (>21 years), residing in these villages for more than fifteen years, were examined clinically for skeletal fluorosis as described elsewhere.[2,12] Simultaneously, from two to five clinically identified fluorotic subjects (>30 years) from each village were also radiologically examined for further evidence of skeletal fluorosis or other skeletal changes. Fluorotic subjects were also examined for evidence of generalised swelling of the thyroid gland (goitre).

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Table 1. Prevalence of dental fluorosis (DF)

Village	Fluoride concentration (ppm)		Prevalence (%) of dental fluorosis	
	Mean	Range	Children (< 16 yr)	Adults
Amaliya Fala	1.40	0.50-1.80	20/78 (25.64)	22/92 (23/91)
Doja	2.00	1.15-2.00	42/86 (48.84)	58/102 (56.86)
Selaj	2.00	1.50-2.50	32/68 (47.06)	46/85 (54.12)
Anturi	2.33	0.30-3.50	92/290 (31.72)	62/180 (34.44)
Batikada	2.35	1.25-3.10	72/130 (55.38)	73/120 (60.83)
Devsomnath	2.52	0.00-3.05	105/180 (58.33)	116/188 (61.70)
Masania	2.55	0.95-2.60	52/106 (49.06)	112/176 (63.64)
Dora	3.00	1.20-3.90	46/82 (56.10)	72/96 (75.00)
Dolwaniya Ka Oda	3.00	2.50-3.50	38/56 (67.86)	57/78 (73.08)
Palvasi	3.41	2.30-4.22	138/170 (81.18)	76/114 (66.67)
Jogiwara	3.43	0.95-4.30	85/98 (86.73)	96/106 (90.57)
Kolkhanda	4.46	4.20-4.65	113/121 (93.39)	98/106 (92.45)
Banda Ghanti	5.67	3.80-6.70	140/180 (77.78)	95/115 (82.61)
Hadmatiya	6.01	3.90-8.30	108/140 (77.14)	195/205 (95.12)
Pantali	6.04	2.40-10.80	141/167 (84.43)	186/192 (96.88)
Total			1224/1952(62.70)	1364/1955 (69.77)

Correlation coefficient between
1) Fluoride concentration and DF in children, r=+ 0.5074 (Positive correlation)
2) Fluoride concentration and DF in adults, r =+ 0.849 (Positive correlation)
3) Children and adults, r =+ 0.925 (Positive correlation)

Table 2. Prevalence of skeletal fluorosis (SF)

Village	Fluoride concentration (ppm)		Prevalence (%) of skeletal fluorosis	Crippling fluorosis
	Mean	Range		
Amaliya Fala	1.40	0.50-1.80	4/92 (4.35)	Negative
Doja	2.00	1.15-2.00	10/102 (9.80)	Negative
Selaj	2.00	1.50-2.50	4/85 (4.71)	Negative
Anturi	2.33	0.30-3.50	16/180 (8.89)	Negative
Batikada	2.35	1.25-3.10	16/120 (13.33)	Negative
Devsomnath	2.52	0.00-3.05	14/188 (7.45)	Negative
Masania	2.55	0.95-2.60	28/176 (15-91)	Negative
Dora	3.00	1.20-3.90	22/96 (22.92)	Positive
Dolwaniya Ka Oda	3.00	2.50-3.50	14/78 (17.95)	Positive
Palvasi	3.41	2.30-4.22	38/114 (33.33)	Positive
Jogiwara	3.43	0.95-4.30	27/106 (25.47)	Positive
Kolkhanda	4.46	4.20-4.65	52/106 (49.06)	Positive
Banda Ghanti	5.67	3.80-6.70	61/115 (53.04)	Positive
Hadmatiya	6.01	3.90-8.30	118/205 (57.56)	Positive
Pantali	6.04	2.40-10.80	121/192 (63.02)	Positive
Total			545/1955 (27.88)	

Correlation coefficient between
Fluoride concentration and SF, r = +0.976 (Positive Correlation)

RESULTS AND DISCUSSION

Dental Fluorosis: Out of 1952 school children and 1955 adults, 1224 (62.70%) and 1364 (69.77%) showed grade II dental fluorosis, respectively. At 1.40 ppm and 6.04 ppm mean F concentration its prevalence (%) in children was 25.64 and 84.43, and in adults 23.91 and 96.88, respectively. The maximum prevalence (76.43%) of dental fluorosis was observed in the 17-22 year age group and the minimum (58.98%) in the 5-10 year age group. However, little variation was found between the prevalence figures of the sexes. Statistically, the degree of correlation between F and prevalence of dental fluorosis in both children and adults was found to be positive (Table 1).

In India, enamel mottling at 0.5 ppm and 0.9-1.0 ppm F levels has been reported.[15,16] At 6.0 ppm F, 100% prevalence of dental fluorosis has also been reported.[17] From Rajasthan, 89.3% with dental fluorosis at 7.6 ppm and 100% at 3.8-5.0 ppm F concentrations have been observed.[9,10,12,14] These findings indicate that the prevalence of dental fluorosis is variable from place to place, although places have almost identical F concentrations in their drinking waters. In the present survey such findings have also been observed (Table 2). These findings suggest that, besides water fluoride concentration and exposure, other factors, such as dissolved salts in drinking waters and nutrition,[17-19] also affect the prevalence of dental fluorosis.

Skeletal Fluorosis: An overall 27.88% prevalence of skeletal fluorosis has been observed. None of children was found to be affected with skeletal fluorosis. The highest prevalence (63.02%) was found at 6.04 ppm, and the lowest (4.35%) at 1.40 ppm, mean F concentration. The prevalence of skeletal fluorosis at different F levels in the villages studied is shown in Table 2. Onset of skeletal fluorosis at 1.40 ppm was observed only in the higher age group(>40 years). Crippling fluorosis was found at and above 3.0 ppm F. Some skeletal fluorosis subjects had common deformities such as genu varum, kyphosis and invalidism. A few cases of paraplegia, quadriplegia and dwarfism have also been observed, in the higher age group (>45 years), at above 4.45 ppm mean F concentration. None of the fluorotic subjects showed evidence of genu valgum deformity or generalized swelling of the thyroid gland (goitre).

Several workers have reported skeletal and crippling fluorosis at F levels above 1 ppm and 3 ppm, respectively.[17] Moreover, other deformities have also been observed in endemic fluorosis areas. In the southern states of India, Andhra Pradesh, Karnataka and Tamil Nadu, where skeletal fluorosis is hyperendemic, cases of genu valgum deformity along with osteoporosis of long bones have been reported.[20,21] The findings on these deformities, which are not endemic in other parts of India, are still controversial and nonconclusive. The correlation between fluoride intoxication and thyroid function is also highly debatable. In India, some workers reported no goitre incidence,[1,22] while others observed it,[23] in endemic areas of fluorosis. In the present study not a single fluorotic subject showed evidence of goitre.

The prevalence of skeletal fluorosis also increased with increasing F concentration and age (Tables 2 and 3), and was relatively higher in males

(Table 3). Statistically, positive correlations were found between F concentration and skeletal fluorosis (r = + 0.976), age and males (r = + 0.99), age and females (r = + 0.197) and males and females (r = + 0.99).

Table 3. Skeletal Fluorosis (SF) in relation to age and sex

Age (yrs)	Males examined	SF (%)	Females examined	SF (%)
21-30	256	12 (4.69)	242	7 (2.89)
31-40	232	45 (19.39)	240	35 (14.58)
41-50	217	85 (39.17)	221	65 (29.41)
51-60	202	108 (53.46)	155	78 (50.32)
> 60	108	65 (60.18)	82	45 (54.88)
Total	1015	315 (31.03)	940	230 (24.47)

Correlation coefficient of SF between
1) Age and Males, r =+ 0.99 (Positive Correlation)
2) Age and Females, r = + 0.197 (Positive Correlation)
3) Males and Females, r = + 0.99 (Positive Correlation)

As F concentration in water increases, the prevalence of skeletal fluorosis affecting lower age groups increases also. However, there are varying prevalences of skeletal fluorosis in those villages where F level is almost identical (Table 2) due to age, sex and occupation.[17] The harder working males, predominantly farmers and manual labourers, ingest more water and consequently have a relatively higher prevalence of fluorosis. Males of the present villages also ingest excessive quantity of wines (local made) and tea which are additional sources of fluoride intoxication. The lower prevalence in women may be due to shorter stays in the villages because of marriage. Higher prevalence of skeletal fluorosis in higher age group is due to long exposure to F.

X-rays of cervical (Figure 1), lumbar-dorsal spine (Figure 2), forearm (Figure 3), pelvis (Figure 4), and rib cage (Figure 5) of the 2-3 fluorotic subjects of each village showed increased bone mass and density as well as exostoses, calcification of ligaments and interosseous membranes and osteosclerosis. These changes become more progressive with increase of age and F concentration. These findings further support evidence of skeletal fluorosis in these villages. Other radiological changes have also been observed as described elsewhere.[11,17]

In villages, where F levels in drinking water are high, appropriate defluoridation technique and health education should be encouraged to reduce dental and skeletal fluorosis. Furthermore, the present study significantly adds to the existing knowledge of fluorosis.

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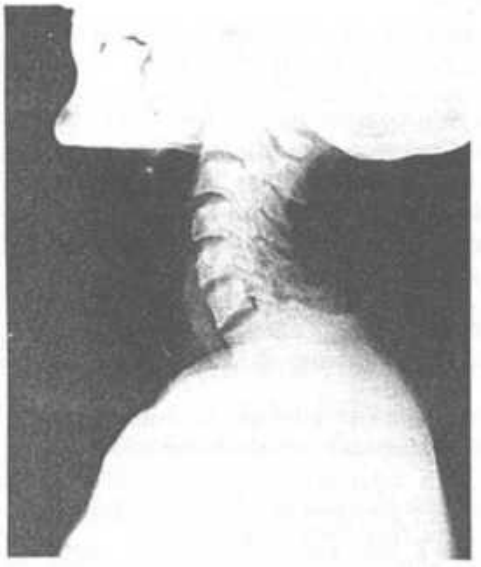


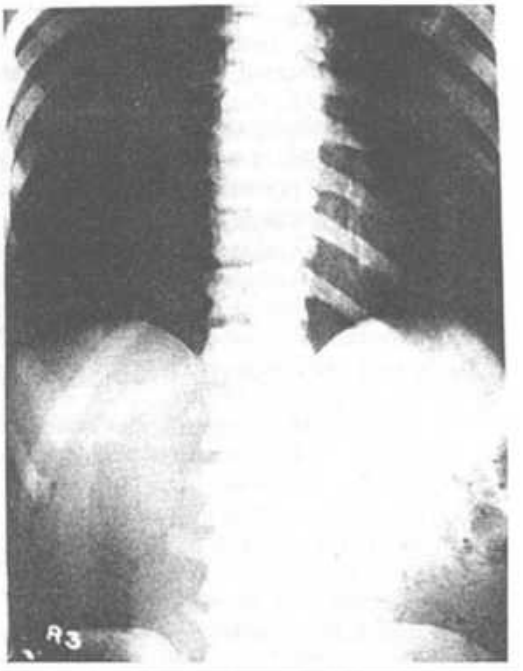


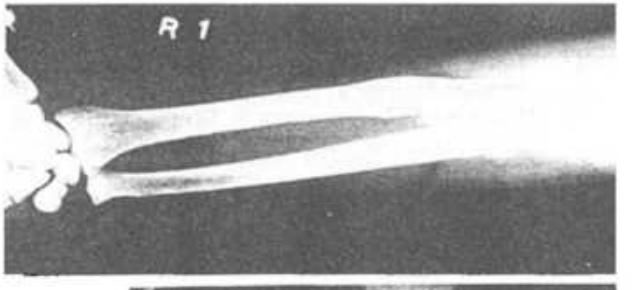
Figure 1 (Above left) Cervical

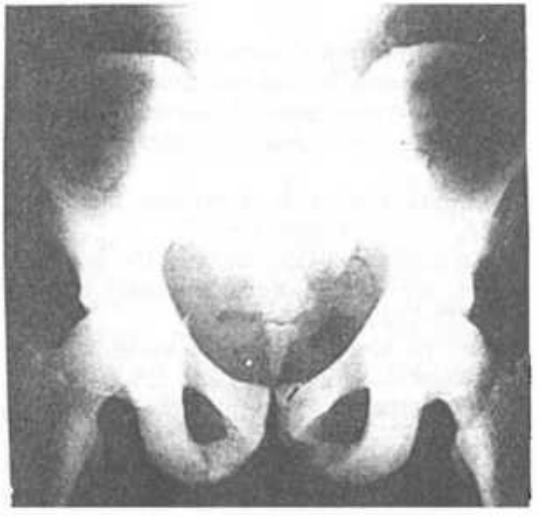
Figure 2 (Above right) Spine

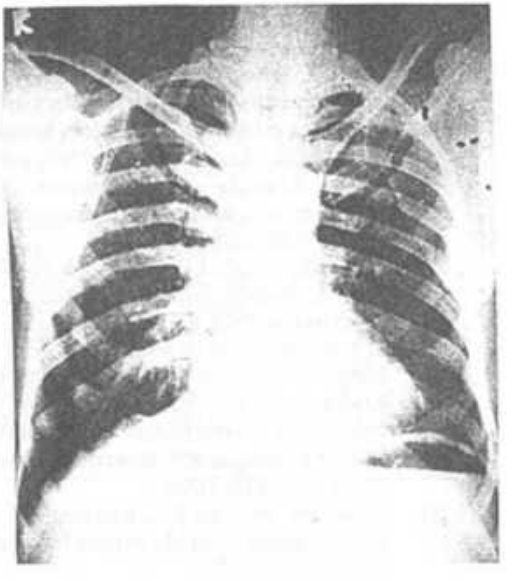
Figure 3 (Right) Forearm

Figure 4 (Below left) Pelvis

Figure 5 (Below right) Ribs







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THE CONTROL OF COAL-BURNING FLUOROSIS IN CHINA

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SUMMARY: After epidemiological investigations the widespread distribution, and main causes, of the coal-burning type of endemic fluorosis in China have been identified. Local residents absorb high doses of fluoride through the respiratory and/or digestive tract because of the incorrect use of high-fluoride coal in cooking, heating and drying of food. Effective preventive measures have been taken to minimise the harm, but more needs to be done.

Key words: China; Coal-burning fluorosis; Fluorosis control.

DISTRIBUTION OF FLUOROSIS CAUSED BY COAL-BURNING IN CHINA

By the end of the 1970s, the coal-burning type of fluorosis was identified as one kind of endemic fluoride poisoning found in mainland China.[1] According to 1995 statistics,[2] this type of fluorosis, in 201 counties of 13 provinces, autonomous regions and municipalities in China, contributed to 18,169,946 cases of dental fluorosis and 1,460,879 cases of skeletal fluorosis. There are 31.6l million people living in the endemic areas (Table 1).

The southern provinces of China such as Hunan, Sichuan, Yunnan, and Guizhou are the most severely affected fluorosis endemic areas. The prevalence rate of dental fluorosis is between 51% and 73% and there are many skeletal fluorosis cases in these areas.

In some northern provinces such as Shanxi, Shaanxi, Liaoning, Henan, and Beijing there are mild or moderate endemic areas with a prevalence rate of dental fluorosis between 13% and 45%, and less skeletal fluorosis.

TABLE 1. Coal-burning type fluorosis in China

Province	No. of counties	No. of villages	No. of house-holds	Population in endemic area	No. of cases of dental fluorosis	No. of cases of skeletal fluorosis
Beijing	2	569	41,912	428,500	59,600	0
Shanxi	20	3,506	622,819	2,476,300	1,126,028	31,866
Liaoning	2	4	498	2,400	1,056	177
Zhejiang	1	10	2,494	9,000	1,059	77
Jiangxi	6	399	61,302	289,100	81,154	1
Henan	12	2,435	187,388	1,026,500	375.334	361
Hubei	17	1,009	283.815	1,112,600	633,137	32,525
Hunan	25	1,741	588,336	2,309,200	1,196,510	73,565
Guangxi	2	518	43,056	216,500	82,900	5,800
Sichuan	54	4,386	1,141,359	3,899,900	1,941,598	136,045
Guizhou	37	11,171	2,849,298	14,197,000	10,381,760	649,143
Yuannan	14	9,424	969,835	4,679,400	2,109,888	423,818
Shaanxi	9	1,502	193,318	968,400	177,922	107,501
Totals	201	36,674	6.985,430	31,614,800	18,169,946	1,460,879

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MAIN PATHOGENIC CAUSES OF COAL BURNING TYPE FLUOROSIS

1. Residents in endemic areas use high fluoride content coal.

Coal is the main energy source for industry, agriculture and residents. Apart from large scale coal mines, there are many small coal pits spread over 13 provinces. Some of these are very shallow, even open pits, which are extremely easy to exploit and serve as a crucial energy source for local residents. The fluoride content of coal in endemic areas varies from ten to thousands of milligrams per kilogram, the highest up to 3000 mg/kg, the average being 200-1500 mg/kg.[1] These values greatly exceed the recommended international average of 80 mg/kg.[3] The coal in endemic areas may have contained high fluoride levels on formation. Residents burn the raw coal mixed with mud and sand which may also contain fluoride. However coal is considered the major fluorine source in endemic areas.

2. Residents in endemic areas burn the coal in improper ways.

Residents in the areas where the local coal has a high fluoride content use various stoves without lids or chimneys and even burn coal directly in the houses without a stove. The toxic fluoride emissions pollute the indoor air and the cereals and vegetables being dried in the room. This is the major cause of fluorosis in these areas. Most of the endemic areas in southern China are located in moist and cold mountainous regions, where grains and vegetables are harvested in the rainy season. In order to prevent grains from going mouldy, the residents have to hang the corn and vegetables in the room and burn coal to dry them[4] (see Table 2). Normally, washing of the polluted food fails to remove the smoke residues containing fluoride. Thus these southern residents ingest excessive fluoride by both respiratory and digestive routes, causing severe fluorosis.

TABLE 2. Fluorine content in two main kinds of food (Guizhou) (dry weight mg/kg)

	maize		hot pepper	
	fresh	toasted (7-10 days)	fresh	toasted (30 days)
Test area	0.26	26.30	0.65	310.50
Control area	0.13	0.70	-	2.48

However, in northern endemic areas, coal is not burned to dry grains and vegetables. Hence the intake of fluoride is mainly through the respiratory tract, resulting in mild fluorosis. It has been shown that the daily overall amount of fluoride intake correlates positively with dental fluorosis.[1]

3. The components of air pollutants in endemic area are very complex.

The burning of high fluoride coal releases many chemicals such as SO_2, SO_3, H_2SO_4, HF, SiF_4, NaF, CaF_2, Al_2O_3. These mainly fluoride emissions pollute indoor and outdoor air, and damage human health via the respiratory tract. There are differences, in severity and clinical manifestations, between coal-burning fluorosis and drinking water fluorosis. For example, the high content of aluminum in coal may be responsible for the severe bone deformations in endemic fluorosis areas of Guizhou province; while the sulphur in coal may affect the high prevalence of dental fluorosis in Hubei and Hebei provinces. It is important that further research is undertaken into the mechanisms by which chemical components of coal smoke cause fluorosis.

CONTROL MEASURES FOR COAL-BURNING TYPE FLUOROSIS

The cause of coal-burning fluorosis is relatively clear. At present the major control method is the popularization of new stoves to replace the traditional backward ones in endemic areas. These new stoves ensure that toxic emissions are discharged outdoors to be diluted, greatly reducing the concentration of fluoride and other toxic substances indoors. Nearly 20 kinds of new stoves used by residents in endemic areas could decrease the indoor concentration of fluoride and SO_2 by around 60-95% (Table 3).[5]

TABLE 3. Reduction of indoor air fluorine and sulphur by 3 kinds of improved stove

	Fluorine content (mg/m^3)				Reduced	SO_2 content (mg/m^3)				Reduced
	No. of samples	Range	$\bar{X}$	S	(%)	No. of samples	Range	$\bar{X}$	S	(%)
Before improved stove	6	0.058-0.103	0.071	0.016	-	6	2.0-72.5	27.25	26.82	-
Improved stove 1	6	0.015-0.033	0.24	0.008	67.1	6	0.4-4.1	1.40	1.41	94.9
Improved stove 2	6	0.017-0.028	0.21	0.005	70.3	6	0.3-2.0	1.00	0.64	96.3
Improved stove 3	6	0.014-0.036	0.24	0.009	66.0	6	0.3-3.0	1.62	1.06	94.1

Great progress has been made in improved stove installations in endemic areas since the successful experiment in selected units of Sichuan and Hubei provinces from 1987 to 1990. Now, there are 1,274,972 households using improved stoves in endemic areas, accounting for 18.25%, which benefited a population of 5,958,900 (Table 4).[2] However, the development is not balanced, for there are still seven provinces with new stove installations below 10%. In poor regions, it is difficult to fund the improved stoves project, so there are delays. In addition, only 58% of owners of new stoves use them all year round, due to little awareness of fluorosis and the constraints of traditional living habits, which reduce the effectiveness of the program.

TABLE 4. Progress of improved stove Installation

Province	No. of house-holds	No. of improved stoves	%	Population in endemic area	No. of persons receiving benefit	%
Beijing	41,912	624	1.49	428,500	7,000	1.63
Shanxi	622,819	433,682	69.62	2,476,300	1,665,900	67.27
Liaoning	498	209	41.97	2,400	800	33.33
Zhejiang	2,494	70	2.80	9,000	100	1.11
Jiangxi	61,302	5,290	8.63	289,100	22,800	7.89
Henan	187,388	17,056	9.10	1,026,500	77,600	7.56
Hubei	283,851	235,216	82.87	1,112,600	906,000	81.43
Hunan	588,336	13,756	2.34	2,309,200	74,400	3.22
Guangxi	43,056	10,490	24.36	216,500	52,500	24.25
Sichuan	1,141,359	238,359	20.88	3,899,900	968,900	24.84
Guizhou	2,849,289	39,513	1.39	14,197,000	194,400	1.37
Yunnan	969,835	276,409	28.50	4,679,400	1,969,400	42.09
Shaanxi	193,318	4,298	2.22	968,400	19,100	1.97
Total	6,985,430	1,274,972	18.25	31,614,800	5,958,900	18.85

THE TASKS AHEAD

1. To strengthen social mobilization and health education to teach all residents in endemic areas about the harm of fluorosis and its control methods.
2. To reduce the usage of coal with high fluoride and sulphur content, and to search for low fluoride coal or transport low-fluoride coal from other regions to the endemic area.
3. To speed up progress and consolidate achievements of stove improvement.
4. To research and develop simple devices suitable for drying grains in severe endemic areas.
5. To change the planting mode of corn; for example, by covering with a plastic membrane to allow earlier harvesting to avoid the rainy season.
6. To experiment with additives and defluoridation agents in affected regions.
7. To develop international cooperation. Further research is necessary into coal-burning fluorosis in China, which is severe and widespread. We would like to provide fields, personnel and laboratories for related agencies, nations and social organizations, to collaborate in the control and research of this kind of fluorosis.

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ENDEMIC FLUOROSIS IN MEXICO

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SUMMARY: The significant mining of fluorspar in Mexico is evidence of the presence of fluoride in the Mexican subsoil. Therefore, the appearance of endemic fluorosis in some areas of our country may be a consequence of the contamination of aquifers by fluorspar deposits. In endemic areas, fluoride levels in drinking water are higher than the Mexican guideline of 1.5 mg/L in both urban and rural locations. Furthermore, health risk effects related to fluoride exposure have been identified in at least two states. Nevertheless, endemic fluorosis is still an essentially unrecognized environmental health problem in Mexico, although it is affecting around five million people. Some improvements have been obtained by the introduction of educational programs and by the emerging of research programs in some universities. However, more work is needed in order to reduce the prevalent risks.

Key words: Endemic fluorosis, Mexico.

INTRODUCTION

Mexico has been an important producer of fluorspar. In 1980, the Mexican production of this mineral represented 20% of the total world production;[1] whereas in 1995, the 522,658 tons of fluorspar generated by Mexico, represented 13% of the worldwide production.[2] During 1995, Mexico occupied second place in the world, its production being greater than that of South Africa, France, or Spain, and was only lower than the Chinese production (Table 1).[2] It is interesting to note that, even though there has been a 43% decrease in Mexican production of fluorspar between 1980 and 1995,[1,2] the state of San Luis Potosi (SLP) remained the top producer in Mexico during this period, with an increase of 32% in its production.[1,2]

TABLE 1. Production of fluorspar in different countries

	1980		1995	
Country	Percent of total world production	Place	Percent of total world production	Place
Mexico	20	1	13	2
South Africa	11	3	6	3
China	9	5	51	1

In 1980 the former USSR was number 2 and Mongolia was number 4. Data presented in this Table were taken from reference 2.

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In Mexico, the entire mining of fluorspar is presently concentrated in two states (Table 2), both located in the north-central region of the country (see Figure). However, during 1980 the mining activities were more extensively distributed as five states were participating in them (Table 2 and Figure). Mining activities are direct evidence of the presence of fluorspar deposits in the Mexican subsoil. These deposits may be provoking the appearance of endemic hydrofluorosis in several Mexican states, as a result of the natural contamination of aquifers with fluoride.

TABLE 2. Mexican States with fluorspar mining

State	Percentage of Mexican Production 1980	1995
SLP	32	74
Guanajuato	30	-
Coahuila	19	26
Chihuahua	17	-
Durango	2	-

In 1980 the Mexican production of fluorspar was 916,455 tons. In 1995 the total production was 522,658 tons. Data in this Table were obtained from references 1 and 2.

FIGURE. Mexican states with endemic fluorosis areas
1 Sonora 2 Chihuahua 3 Coahuila 4 Durango
5 San Luis Potosi 6 Guanajuato 7 Aguascalientes

ENDEMIC HYDROFLUOROSIS

Fluoride levels in potable water sources were studied in different cities. Table 3 shows that those cities located near mining regions, like Durango and SLP, are among those having high fluoride levels. However, other cities, like Hermosillo, Aguascalientes and Salamanca, which are located distant from fluorspar mining areas, also have important concentrations of fluoride in drinking water. It is important to take into account that the mean fluoride levels reported in Table 3, are higher than the Mexican national guideline for fluoride in municipal water sources of 1.5 mg/L.[3] Furthermore, specific areas with very high fluoride levels were found in all the cities. For example, in the city of Hermosillo levels of up to 7.8 mg/L were detected (Table 3, value in the upper range).

TABLE 3. .Fluoride levels in potable water sources (mg/L) in urban areas

City	State	Population[a]	Mean[b]	SD[c]	Range
Hermosillo	Sonora	559,154	1.5	1.7	0.2 - 7.8
Aguascalientes	Aguascalientes	582,827	1.8	0.8	0.9 - 4.3
San Luis Potosi	San Luis Potosi	625,466	1.8	1.1	0.3 - 5.8
Salamanca[d]	Guanajuato	221,125	2.6	1.8	0.6 - 6.5
Durango	Durango	464,566	2.8	0.4	2.4 - 3.4

[a] Data from reference 4. [b] Arithmetical Mean. [c] Standard Deviation.
[d] Data from the Health Department. Fluoride levels in water were quantified with a sensitive specific ion electrode, as previously described.[10]

When compared to urban areas, some rural locations have higher mean fluoride levels in potable water sources (Table 4). Besides, they also present areas with very high concentrations of fluoride. For example, levels of up to 8.0 mg/L of fluoride in drinking water were detected at Abasolo (Table 4, value in the upper range).

TABLE 4. .Fluoride levels in potable water sources (mg/L) in rural locations

Location	State	Mean[a]	SD[b]	Range
Comarca Lagunera[c]	Durango/Coahuila	0.9	0.8	< 0.5 - 3.7
Villa de Reyes	San Luis Potosi	2.6	0.2	2.2 - 2.7
San Francisco[d]	Guanajuato	3.8	0.7	3.0 - 4.8
Abasolo[d]	Guanajuato	4.1	2.4	0.2 - 8.0
Puerto Peñasco	Sonora	4.5	1.1	3.7 - 6.7

[a] Arithmetical Mean. [b] Standard Deviation. [c] Data from reference 15.
[d] Data from the Health Department. Fluoride levels in water were quantified with a sensitive specific ion electrode, as previously described.[10]

Almost 2.5 million persons are exposed to fluoride, and there only in the cities studied in this work. However, the total amount of people at risk is certainly greater, as there is preliminary evidence of the presence of fluoride in water sources at other Mexican cities and in further rural areas. Considering all the locations, around 5 million persons may be exposed to higher than normal levels of fluoride in Mexico. This figure represents almost six percent of the total population.[4]

SOURCES OF FLUORIDE IN MEXICO

Potable water is the principal source of fluoride in Mexico; however, salt, soft-drinks and food are other important sources of this mineral.

A National Program of Salt Fluoridation was started in 1991, the goal of this program being to decrease the prevalence of dental caries in the Mexican population.[5] At the beginning of the program, it was declared that fluoridated salt would be distributed all over the country with the exception of those areas where fluoride levels in drinking water were higher than 0.7 mg/L.[6] However, fluoridated salt is being distributed in populations like SLP, which is a city with a mean fluoride concentration in potable water of 1.8 mg/L. Furthermore, people in these cities are consuming fluoridated salt, even though on their packages, a warning states the risk of consuming it in endemic areas. The amount of fluoride that was supposed to be present in the salt was 250 ± 50 mg/kg.[6] Nevertheless a study of 221 samples of salt registered a mean fluoride concentration of 48.7 mg/kg.[7] The authors of this report also found one sample with fluoride concentrations of up to 475 mg/kg and some samples with high levels of fluoride that were being distributed in packages without the warning.[7]

With reference to soft drinks, it is important to take into account that Mexico is one of the countries where more soft drinks are being consumed. Thus, the presence of fluoride in this source could become a health risk. In order to test this hypothesis, the fluoride content in the most popular brand of soft drinks was studied in a preliminary survey. Samples were collected in two Mexican mining states (SLP and Durango). The results showed differences in fluoride concentrations between the states (Table 5). Furthermore, fluoride levels were also distinct in samples of different brands collected in the same state. For example, in SLP, levels of fluoride up to 3.6 mg/L were found in some brands. The results can be explained, considering that soft drinks in Mexico are produced in each state by different companies; therefore, the fluoride content in each sample is a reflection of two factors: the fluoride concentration in the source of water used for the production of soft drinks and the availability of a treatment plant for the defluoridation of water in those companies involved in this activity.

TABLE 5. Fluoride levels in the most consumed brand of soft drinks in Mexico.

Source of the sample	F^- levels (mg/L) in the sample	Standard deviation
SLP	0.46	0.08
Durango	2.14	0.01
Control	0.60	0.05

Values are arithmetic means. Samples were collected in mining states located in endemic areas (SLP and Durango); or in a state located in a non-endemic region (Control). F^- levels in soft drinks were quantified with a sensitive specific ion electrode.

Thus, salt and soft drinks are important sources for fluoride exposure in Mexico. For instance, considering the mean daily amount of salt ingested by the Mexican population (8.0 g/day)[7], children in non-endemic areas, exposed to fluoridated salt, would be ingesting the amount of fluoride typical of an area with 2.0 mg/L of fluoride in water. Whereas, assuming a daily consumption of

one 250 mL - bottle of soft drink, the fluoride content in this source, would represent 10 % of the exposure dose for an adult living in the endemic area of Durango (without considering food sources).

FLUORIDE AND POVERTY

A possible relation between nutritional deficiencies and the severity of the effects caused by the exposure to fluoride, has been reported.[8] Considering that in Mexico, almost 12 % of the population lives in extreme poverty,[9] and taking into account that 64 % of those in extreme poverty conditions live in rural areas,[9] a small study was undertaken to verify the effects of fluoride (dental fluorosis) in a Mexican rural area with high levels of fluoride in drinking water.

Results depicted in Table 6 show that children living in the community of El Rosario have a higher prevalence of severe dental fluorosis when compared to children living in Villa de Reyes. This result was obtained despite the fact that both communities have similar fluoride concentrations in drinking water. For the analysis of the results, it is important to consider that urinary levels of fluoride were similar in children of both communities (Table 6), meaning that they were exposed to similar sources of fluoride. Taking into account that the social margination in El Rosario is higher than in Villa de Reyes, results in Table 6 confirmed the influence of social margination in the severity of the fluoride-induced effects.

TABLE 6. Correlation between margination ranking and fluoride effects in rural locations

Localities [a]	Margination [b]	Mean fluoride levels in potable water (mg/L)	Mean fluoride levels in urine (mg/L)	Prevalence of severe dental fluorosis [c]
Villa de Reyes	Low	2.6	5.3	43%
El Rosario	Medium	2.7	6.8	87%

[a] Both locations are localities in San Luis Potosi. [b] The margination ranking was established by the National Institute of Statistics, Geography and Information. [c] Fluoride levels in urinary samples and dental fluorosis were studied in children aged 8-17 years as previously described.[10]

FINAL COMMENTS

Endemic fluorosis is clearly present in some geographical areas of Mexico. Taking into account that this problem is affecting an important percentage of individuals, the introduction of health programs for risk reduction is a special need. Among the issues that require specific attention, we selected for a brief analysis the following:

To our knowledge, the only epidemiological study strictly projected to define the prevalence of dental fluorosis is the one which was performed in SLP.[10,11] In this study, the prevalence of total dental fluorosis found in SLP was 76% for children aged 11-13 years. In other cities or rural areas, like those of the state of Sonora, the figures obtained in preliminary analysis are similar to the one obtained in SLP. Therefore, more studies are needed in endemic areas, in order to verify the prevalence of dental fluorosis. Rural regions should deserve special attention, as the nutritional status may increase the risk of this fluoride-induced effect.

Skeletal fluorosis is a result of two factors: the dose and the time of exposure.[12,13] As to the dose, the concentrations of fluoride in drinking water in Mexico (Tables 3 and 4), are not as high as those found in areas with a high prevalence of crippling fluorosis.[12-13] And, in relation to the time of exposure, in many Mexican states the length of time of the exposure is less than 30 years, which is the age of the most contaminated wells. Therefore, what is needed for the particular conditions of Mexico, are studies aimed to define biomarkers for early signs of skeletal fluorosis. In SLP, we have reported some cases of preliminary skeletal fluorosis for which the diagnostic was made through x-rays and densitometry.[14] However the results were not conclusive. It would be interesting to test, in these cases, the usefulness of biochemical markers like osteocalcin levels in blood.

At least in the "Comarca Lagunera", a region located in the states of Durango and Coahuila, a correlation of 0.77 was found for arsenic and fluoride concentrations in well water.[15] Interestingly, in SLP, arsenic has also been found in samples collected from the aquifer contaminated with fluoride.[16] Therefore, it would be a matter of concern to study the toxicological interaction of both minerals in soft tissues. Arsenic and fluoride may have in common some mechanisms of toxicity. For example, arsenic increases lipid peroxidation[17] and fluoride effects are reduced by free radical scavengers[18].

In a different approach to the problem, and in a direction to decrease the exposure to fluoride, there is an urgent need to improve the enforcement of the law. In relation to soft drinks, in Mexico the guideline for bottled water is 0.7 mg/L,[19] and as shown in Table 5, some brands have higher levels than this guideline; whereas, in relation to fluorinated salt, a better distribution of it is advisable. This kind of salt has to be made unavailable in endemic fluorosis areas. The enforcement of the law requires education, as in our society, the benefits of using fluoride are more popular than its risks.

New laws would be an excellent aid for risk-reduction in sites with endemic fluorosis. For example, it would be interesting to set regional guidelines for municipal water sources which consider factors like local temperature or poverty ranking (nutritional deficiencies). In Mexico, it is a common mistake to apply the same national guideline for fluoride in municipal water in different regions. That is the case of SLP and Sonora. In both states the national guideline of 1.5 mg/L is being applied; however, the mean annual temperature in SLP is around 24°C, whereas in Sonora it is around 40°C. As a result of this difference in ambient temperature, the exposure to fluoride by drinking water is higher in Sonora.

In conclusion, endemic fluorosis still is an essentially unrecognized environmental health problem in Mexico. For this reason, educational programs in the area of fluoride-related health risks are becoming an important cue for the introduction of intervention programs in endemic areas. Some improvements have been obtained, but certainly more work is needed in order to reduce the prevalent risks.

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THE IMPACT OF INDUSTRIAL FLUORIDE FALLOUT ON FAUNAL SUCCESSION FOLLOWING SAND-MINING OF DRY SCLEROPHYLL FOREST AT TOMAGO NSW. 2. MYOBATRACHID FROG RECOLONIZATION

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Abstract from *Biological Conservation 82* (2) 137-146 1997 (November)

We examined the response of frog species, recolonising dry sclerophyll forest following sand-mining, to the added impact of fluoride pollution from an aluminium smelter. Pitfall trapping was used to survey a chronosequence of sites at 3, 8 and 20 years post-mining at three nominal levels of fluoride contamination (background low and high). Frog species, from the family *Myobatrachidae*, showed the following successional pattern of species replacement with increasing time after mining. *Limnodynastes dumerilii, Crinia spp. (tinnula* and *signifera), L. peronii, U. laevigata.* Frog species appear to enter the succession as their species-specific habitat requirements are met. Fluoride fallout, by affecting vegetation structure, alters the path of the succession of frog species. At 3 years post-mining, an increase in understorey density at the low fluoride level appeared to accelerate the succession of frog species. However, by 20 years past-mining, decreased canopy cover attributed to fluoride fallout is associated with a retardation of frog succession. There was little frog activity at extremely high levels of fluoride contamination, perhaps as a direct result of toxic effects.

Key Words: Australia; Disturbance; Faunal succession; Frogs; Industrial fluoride emission; Sand mining;.

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FLUORIDE-INDUCED LESIONS IN THE TEETH OF THE SHORT-TAILED FIELD VOLE (*MICROTUS AGRESTIS*): A DESCRIPTION OF THE DENTAL PATHOLOGY

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Abstract from *Journal of Morphology 232* (2) 155-167 1997 (May)

The effect of fluoride on the appearance of the teeth of the short-tailed field vole, *Microtus agrestis*, was investigated in both wild animals collected from field sites affected by different levels of industrial fluoride contamination and laboratory-reared animals consuming experimental grass diets of known fluoride concentration or with known fluoride concentrations in drinking water. The extent and severity of lesions on the surface and structure of both incisors and molars are described as six lesion types and related to the amount of biologically available orally ingested fluoride. In the incisors of voles consuming relatively low fluoride diets, lesions are mainly confined to those resulting from disruption

of enamel pigmentation expressing itself as concentric bands of pigmentation-free areas on incisor surfaces. The visible effects on molars at low fluoride levels are confined to minor alterations in surface appearance. At higher levels of available dietary fluoride, effects on enamel pigmentation are superseded by alterations in the formation, composition, and strength of both enamel and dentine. The incisors exhibit a marked to severe increase in the cutting tip erosion rates with comparable increases in the extent of abnormal surface changes (enamel hypoplasia) and loss of enamel pigmentation. The grinding surfaces of molars from animals exposed to high levels of dietary fluoride exhibit increasingly severe erosion of outer enamel regions, combined with cavitation and staining of the exposed central dentine. The mechanisms through which fluoride elicits increasingly visible and pathological alterations to the surface and subsurfaces of rodent teeth are discussed.

Key words: Dental effects; Field vole; Industrial fluoride emission.
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ECOTOXICOLOGICAL EFFECTS OF FLUORINE DEPOSITS ON MICROBIAL BIOMASS AND ENZYME ACTIVITY IN GRASSLAND

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Abstract from *European Journal of Soil Science 48* (2) 329-335 1997 (June)

The influence of atmospheric fluoride deposits on the soil microbial biomass and its enzyme activities was investigated near the aluminium smelter at Ranshofen, Upper Austria. Soil samples at various distances from the emission source were analyzed for water-extractable F and microbial activity. The water-extractable F contents at the sites examined reflected the gradient of F exposure (10 to 189 mg F kg^{-1} dry soil). The microbial activities increased with distance from the emission source and were inversely correlated with the degree of F contamination. The linear correlation coefficients between the water-extractable F concentrations and the microbial biomass, dehydrogenase and arylsulphatase activity were r = -0.8, -0.86 and -0.84, respectively. In the most contaminated soil (up to 189 mg F kg^{-1}), the microbial activities were only 5-20% of those in the unpolluted soil. The microbial biomass and dehydrogenase activity decreased substantially where the concentration of F exceeded 100 mg kg^{-1}, whereas arylsulphatase activity was already inhibited at 20 mg kg^{-1}. The accumulation of organic matter near the smelter (123 mg F kg^{-1}) also indicated severe inhibition of the microbial activity by F. Our investigations show that the ratio of arylsulphatase to microbial biomass can be used as a sensitive index for evaluating environmental stress such as F contamination.

Key words: Atmospheric fluoride; Enzyme activity; Grassland; Microbial biomass; Soil.
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PREVENTION OF INDUSTRIALLY-INDUCED CATTLE AND SHEEP FLUOROSIS [French]

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Abstract from *Recueil de Medecine Veterinaire 173* (1-3) 53-58 1997 (January-March)

Two industrial plants emitting fluorides were built in an industrial area at Vlissingen, The Netherlands; one produced phosphorus from natural phosphates and the other produced aluminium through electrolysis. Control systems were installed to reduce emissions. An agreement was made between the farmers, manufacturers and provincial authorities of Zeeland to examine possible claims and develop measures for prevention. A 2 to 3 km area around the plants was designated unsuitable for agriculture and cattle-rearing. Beyond that area, contamination of pasture was measured every 15 days. About 30 differently contaminated herds were visited at regular intervals to evaluate fluoride intake and to initiate prevention hy distribution of rations containing 6 per cent aluminium sulfate to reduce fluoride bioavailability and replacing contaminated feed with fluoride-free feed. Pasture contamination was much higher in winter than in spring and summer; at the highest contaminated site, the average for autumn-winter was up to 100 ppm in the period 1970-1980, whereas it generally remained below 50 ppm in spring and summer. The levels recently decreased to half those values. Prevention of fluorosis has proven entirely successful: no damage compensation has been granted since 1985.

Key Words: Cattle; Damage compensation; Fluorosis; Industrial fluoride emmission; Prevention; Sheep.

Reprints: G Milhaud, Ecole Nationale Vétérinaire d'Alfort, Service Toxicologie, 7 Av du Géneral De Gaulle, F-94704 Maisons Alfort Cédex, France.

HYDROGEN FLUORIDE EFFECTS ON PLASMA MEMBRANE COMPOSITION AND ATPase ACTIVITY IN NEEDLES OF WHITE PINE *(PINUS STROBUS)* SEEDLINGS PRETREATED WITH 12 H PHOTOPERIOD

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Abstract from *Trees-Structure & Function 11* (4) 248-253 1997 (February)

Eastern white pine (*Pinus strobus L.*) seedlings were pretreated with 12 h photoperiod to induce dormancy. Dormant plants were fumigated with 0.5 ppb (0.4 $\mu g/m^{-3}$) or 2.0 ppb (1.6 $\mu g/m^{-3}$) hydrogen fluoride (HF) for 2-28 days. Plasma membranes were isolated from needles of treated and control seedlings to determine their chemical composition and ATPase activity. For all analyses, only those plants which did not show needle necrosis were selected. The amount of plasma membrane phospholipid expressed on a plasma membrane protein basis

was higher after 2 days in the 0.5 ppb HF treatment as compared to controls. After 2 days of 2.0 ppb HF treatment as well as after 8 and 28 days of both HF treatments phospholipid to protein ratios in fluoride treated seedlings were lower as compared to control levels. A decrease in sterol levels could be observed after 2 days in both HF treatments. A large increase in the ratio of sterols to proteins was observed in plasma membranes of eastern white pine seedlings treated with 0.5 ppb HF for 28 days. Increased sterol to phospholipid ratios were observed after 8 and 28 days in 0.5 ppb and after 2 and 8 days of 2.0 ppb HF treatment. A decrease in ATPase activity was observed after 8 days with both fluoride treatments. Drastic increase of ATPase activity was observed after 28 days of HF treated plants. Observed changes of sterol and phospholipid levels after only 2 days of fumigation suggest early fluoride effects on plasma membrane composition during plant dormancy.

Key Words: ATPase; Hydrogen fluoride; Lipids; Pine; Plasma membrane.
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FLUORIDE CONTENT AND MINERALIZATION OF RED DEER (CERVUS ELAPHUS) ANTLERS AND PEDICLES FROM FLUORIDE POLLUTED AND UNCONTAMINATED REGIONS

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Abstract from *Archives of Environmental Contamination and Toxicology 32* (2) 222-227 1997 (February)

Fluoride, calcium, and phosphorus content as well as ash percentage and ash density of primary antlers and pedicle bones were studied in nine yearling red deer stags from a fluoride polluted region in North Bohemia (Czech Republic) and in nine control animals from two uncontaminated areas in West Germany. Fluoride levels in antlers (845 ± 257 mg F^-/kg ash, mean ± SD) and pedicles (1,448 ± 461 mg F^-/kg ash) of the N-Bohemian specimens exceeded that of the controls (antlers: 206 ± 124 mg F^-/kg ash, pedicles: 322 ± 157 mg F^-/kg ash) by factors of 4.1 and 4.5, respectively. Antler and pedicle fluoride concentrations of the deer ($n = 18$) were closely correlated ($r = 0.975$, $p < 0.001$). Analyses of ash percentage and ash density revealed that the antlers of the N-Bohemian deer contained significantly less mineral and were significantly less dense than both their pedicles and the control antlers. In the pooled antler samples ($n = 18$), bone fluoride concentration was negatively correlated with ash density ($r = -0.826$, $p < 0.001$) and ash percentage ($r = -0.759$, $p < 0.001$), whereas non-significant positive correlations existed for the pooled pedicle samples. Ash percentage and ash density of the antlers and their corresponding pedicles were uncorrelated. It is concluded that increased fluoride exposure of deer leads to reduced mineral content and mineral density of antler bone and that it is the rapidity of their

growth and mineralization that makes antlers especially susceptible to fluoride action. Due to their ability to accumulate high amounts of fluoride during a defined, limited timespan and the apparently dose-dependent negative effect of fluoride on their density and mineral content, (primary) antlers can be recommended as monitoring tools for studying environmental pollution by fluorides.

Key words: Deer; Fluoride pollution; Mineralization.
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AVAILABILITY OF FLUORIDE TO PLANTS GROWN IN CONTAMINATED SOILS

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Abstract from *Plant and Soil 191* (1) 13-25 1997 (April)

Two pot experiments were carried out to study uptake of fluoride (F) in clover and grasses from soil. Fluoride concentrations in *Trifolium repens* (white clover) and *Lolium multiflorium* (ryegrass) were highly correlated with the amounts of H_2O- and 0.01 M $CaCl_2$-extractable F in soil when increasing amounts of NaF were added to two uncontaminated soils ($r = 0.95$-0.98, $p < 0.001$). The amounts of H_2O- or 0.01 M $CaCl_2$-extractable F did not explain the F concentrations to a similar extent in *Agrostis capillaris* (common bent) grown in 12 soils (Cambic Arenosols) collected from areas around the aluminium smelters at Ardal and Sunndal in Western Norway ($r=0.68$-0.78). This may be due to variation in soil pH and other soil properties in the 12 soils. Soil extraction with 1 M HCl did not estimate plant-available F in the soil as well as extraction with H_2O or 0.01 M $CaCl_2$. Fluoride and Al concentrations in the plant material were positively correlated in most cases. Fluoride and Ca concentrations in the plant material were negatively correlated in the first experiment. No consistent effects were found on the K or Mg concentrations in the plant material. The F accumulation in clover was higher than in the grasses. The uptake from soil by grasses was relatively low compared to the possible uptake from air around the aluminium smelters. The uptake of F in common bent did not exceed the recommended limit for F contents in pasture grass (30 mg kg^{-1}) from soil with 0.5-28 mg $F(H_2O)$ kg^{-1} soil. The concentration in ryegrass was about 50 mg F kg^{-1} when grown in a highly polluted soil (28 mg $F(H_2O)$ kg^{-1} soil). Concentrations in clover exceeded 30 mg F kg^{-1} even in moderately polluted soil (1.3-7 mg $F(H_2O)$ kg^{-1} soil). Liming resulted in slightly lower F concentrations in the plant material.

Key Words: Aluminium smelters; Bent; Clover; Industrial fluoride emission; Plant availability; Ryegrass; Soil fluoride.
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FLUORIDE TREATMENT INCREASED SERUM IGF-1, BONE TURNOVER, AND BONE MASS, BUT NOT BONE STRENGTH, IN RABBITS

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Abstract from *Calcified Tissue International 61* (1) 77-83 1997 (July)

We hypothesized that fluoride partly acts by changing the levels of circulating calcium-regulating hormones and skeletal growth factors. The effects of oral fluoride on 24 female, Dutch-Belted, young adult rabbits were studied. The rabbits were divided into two study groups, one control and the other receiving about 16 mg fluoride/rabbit/day in their drinking water. After 6 months of fluoride dosing, all rabbits were euthanized and bone and blood samples were taken for analyses. Fluoride treatment increased serum and bone fluoride levels by over an order of magnitude ($P < 0.001$), but did not affect body weight or the following serum biochemical variables: urea, creatinine, phosphorus, total protein, albumin, bilirubin, SGOT, or total alkaline phosphatase. No skeletal fluorosis or osteomalacia was observed histologically, nor did fluoride affect serum PTH or Vitamin D metabolites ($P < 0.4$). BAP was increased 37% ($P < 0.05$) by fluoride; serum TRAP was increased 42% ($P < 0.05$); serum IGF-1 was increased 40% ($P < 0.05$). Fluoride increased the vertebral BV/TV by 35% ($P < 0.05$) and tibial ash weight by 10% ($P < 0.05$). However, the increases in bone mass and bone formation were not reflected in improved bone strength. Fluoride decreased bone strength by about 19% in the L5 vertebra ($P < 0.01$) and 25% in the femoral neck ($P < 0.05$). X-ray diffraction showed altered mineral crystal thickness in fluoride-treated bones ($P < 0.001$) and there was a negative association between crystal width and fracture stress of the femur ($P < 0.02$). In conclusion, fluoride's effects on bone mass and bone turnover were not mediated by PTH. IGF-1 was increased by fluoride and was associated with increased bone turnover, but was not correlated with bone formation markers. High-dose fluoride treatment did not improve, but decreased, bone strength in rabbits, even in the absence of impaired mineralization.

Key words: Biomechanics; Bone; Fluoride treatment; Igf-1; Mineralization.
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ETIDRONATE VERSUS FLUORIDE FOR TREATMENT OF OSTEOPENIA IN PRIMARY BILIARY CIRRHOSIS: PRELIMINARY RESULTS AFTER 2 YEARS

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Abstract from *Gastroenterology 113* (1) 219-224 1997 (July)

Background and Aims: Because osteopenia increases morbidity of primary biliary cirrhosis (PBC), the effects of cyclical etidronate vs sodium fluoride on bone mass were compared in patients with PBC. **Methods**: Thirty-two women with PBC were randomly assigned to receive etidronate (400 mg/day during 14 days every 3 months) or fluoride (50 mg/day, enteric-coated tablets). Bone mineral density of the lumbar spine and proximal femur were measured initially and every 6 months. Bone fractures were also evaluated. **Results**: Sixteen patients were allocated into

each group, which were comparable with respect to the severity of PBC and osteopenia. Thirteen patients with etidronate and 10 patients with fluoride completed 2 years in the study. In the etidronate group, bone mineral density increased in the lumbar spine ($P = 0.02$) and did not change in the proximal femur. In the fluoride group, lumbar bone mineral density did not change but femoral bone mass decreased, particularly in the Ward's triangle. Two patients in the fluoride and none in the etidronate group developed new vertebral fractures, and the number of new nonvertebral fractures was similar in both groups. Neither treatment impaired liver function or cholestasis. **Conclusions**: Cyclical etidronate is more effective and better tolerated than sodium fluoride in preventing bone loss in PBC.

Key words: Etidronate; Osteopenia; Sodium fluoride; Primary biliary cirrhosis.

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DISTURBANCES OF THE SECRETORY STAGE OF AMELOGENESIS IN FLUOROSED DEER TEETH: A SCANNING ELECTRON-MICROSCOPIC STUDY

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Abstract from *Cell and Tissue Research 289* (1) 125-135 1997 (July)

Structural changes resulting from fluoride-induced disturbances of the secretory stage of amelogenesis were studied in fluorosed dental enamel of ten permanent premolars and molars from roe deer (*Cervus capreolus*) and red deer (*Cervus elaphus*). The fluorosed enamel exhibited surface hypoplasias of different depths and extents and an associated loss of its normal prism/interprism structure. The occurrence of such aprismatic enamel either was restricted to grossly accentuated and hypomineralized incremental (calciotraumatic) bands or affected more extended areas to the bottom of the hypoplastic lesions. The fluoride-induced disturbance of the secretory functions of the cells had thus been either temporary or permanent. Layers of aprismatic enamel were regarded as denoting periods of reduced enamel matrix formation by secretory ameloblasts lacking the distal, *i.e.*, the prism-forming, portions of their Tomes processes. Our observations also indicated that the transition from the presecretory to the secretory stage of amelogenesis could be affected by fluoride, thereby preventing the ameloblasts from achieving their normal secretory function and from establishing fully formed Tomes processes. Aprismatic enamel was formed throughout the secretory stage of amelogenesis at these locations. The most severe ameloblast reaction that could be deduced from our findings was an abrupt cessation of enamel matrix secretion. Some of the pathological changes observed in fluorosed deer enamel showed striking similarities to those reported in rodents after acute parenteral fluoride dosing. Thus, periods of especially elevated plasma-fluoride levels in chronically fluoride-stressed deer can cause a disruption in the function of secretory ameloblasts similar to that following acute fluoride dosing in rodents.

Key words: Cervidae; Dental fluorosis; Developmental defects; Enamel; Hypoplasia; Red deer, *Cervus elaphus;* Roe deer, *Capreolus capreolus (artiodactyla).*

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TWO TYPES OF INTRAORAL DISTRIBUTION OF FLUOROTIC ENAMEL

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Abstract from *Community Dentistry and Oral Epidemiology 25* (3) 251-255 1997 (June)

Different distributions of fluorotic dental enamel within the dentition have been described in the literature. This report describes two patterns of intraoral distribution. In nine Tanzanian low fluorosis communities with a prevalence of pitting fluorosis of less than 2% and in five moderate fluorosis communities with a prevalence of pitting fluorosis of 16-59%, incisors and first molars were the least affected teeth. In four high fluorosis communities with a prevalence of pitting fluorosis of 86-97%, maxillary incisors exhibited lower Thylstrup-Fejerskov Index values than the maxillary canines, premolars and molars. The mandibular teeth exhibited increasing Thylstrup-Fejerskov Index values from the anterior to the posterior region. The curves presenting the intraoral distribution of the severity of dental fluorosis corresponded with the curve presenting the completion time of primary enamel formation of the various tooth types, with the exception of the first molars in high fluorosis communities. The similarity of the curves suggests that the later in life enamel is completed, the higher is the severity of dental fluorosis. This relation seems to be explained by the prevailing feeding and dietary habits, which result in minimal intake of fluoride in the first 18 months of life during breastfeeding, followed by increasing fluoride ingestion in the following years through consumption of tea, seafish and F-containing magadi salt.

Key words: Dental fluorosis; Diet; Tanzania.

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UNACCEPTABLY HIGH LEVELS OF FLUORIDE IN COMMERCIAL PREPARATIONS OF SILVER FLUORIDE

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Abstract from *Australian Dental Journal 42* (1) 52-53 1997 (February)

Instead of expected fluoride ion concentrations of around 60,000 ppm, commercial preparations of 40 per cent aqueous silver fluoride were found to contain 120,000-127,000 ppm. Information received from the Western Australian Chemistry Centre which provided independent confirmation of the higher than expected [F] indicates that the currently available commercial preparations contain silver difluoride rather than silver fluoride.

In view of the potential of fluoride-containing products such as dentifrices (1,000-1,500 ppm F) and topical fluoride gels and solutions (6,000-12,000 ppm F) to cause adverse effects if excessive quantities are ingested, any product that contains 120,000 ppm [F] should be regarded as carrying a high risk of toxicity when used on young children.

Key words: Silver fluoride; Adverse effects.

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SAFETY ISSUES RELATED TO THE USE OF SILVER FLUORIDE IN PAEDIATRIC DENTISTRY

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Abstract from *Australian Dental Journal 42* (3) 166-168 1997 (June)

Due to its exceedingly high fluoride content, 40% silver fluoride solution has the potential to cause fluorosis when used in young children. *In vitro* testing conducted in the present investigation indicates that application of 40% silver fluoride to deep carious lesions or its use as a 'spot' application agent could result in 3 to 4 mg of fluoride reaching the systemic circulation.

As scientifically-based clinical trials on the safety of 40% silver fluoride have not been conducted, it would be appropriate for it to be withdrawn from further clinical use until proper testing and evaluation have been carried out. In view of the possibility that lower strength solutions of silver fluoride (1-4%) may be just as effective as 40% in 'arresting' deep caries, testing should focus on such solutions, particularly as the potential for toxicity from their fluoride content would be reduced by a factor of 10-40.

Key words: Fluorosis; Silver fluoride; Toxicity.

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THE START OF CARIES DECLINE AND RELATED FLUORIDE USE IN NORWAY

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Abstract from *European Journal of Oral Sciences 105* (1) 21-26 1997 (February)

The objective was to determine the start of caries decline and to estimate the use of fluorides about this time. The material consisted of service reports from the Norwegian municipal School Dental Services (SDS) and the county-based Public Dental Service (PDS), literature reports, private sales and marketing statistics, and a national health survey. The national data for the permanent dentition demonstrated a distinct decline in the average number of filled tooth surfaces from around 1970. Counties were grouped as "early", less than or equal to 1970, and "late", greater than or equal to 1971. The 5 "early" counties showed the decline to be evident in 1968. Several local districts reported caries reductions since 1963, following the implementation of fluoride programs. Altogether, there was a steady increase in the % of schoolchildren taking part in organized brushing or rinsing programs from 1960. In 1970, 60% of the schoolchildren regularly took part in the supervised fluoride programs. From September 1971, 0.1% fluoride dentifrices became generally available over the counter in retail stores. In 1972, 32-54% of all toothpaste delivered or sold was fluoridated. The distinct time interval between the caries decline and the availability of fluoridated toothpaste rules out the possibility of an interaction before 1971. The extensive activities with fluoride-based preventive programs in the SDS and PDS have probably been the major factors behind the decline in the late 60s/early 70s.

Key words: Dental caries; Epidemiology; Fluoride programs; Fluoride toothpaste.

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SERUM IONIC FLUORIDE LEVELS IN HAEMODIALYSIS AND CONTINUOUS AMBULATORY PERITONEAL DIALYSIS PATIENTS

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Abstract from *Nephrology, Dialysis, Transplantation 12* (7) 1420-1424 July 1997

High serum fluoride (F^-) in patients with chronic renal failure (CRF) and end-stage renal disease (ESRD) is associated with risk of renal osteodystrophy and other bone changes. This study was done to determine F^- in normal healthy controls and patients with ESRD on haemodialysis (HD) or peritoneal dialysis (PD). Seventeen healthy controls (12 males, 5 females) and 39 ESRD patients on dialysis (17 males, 22 females) were recruited in the study in a community with 47.4 ± 3.28 μM/L (range 44-51 μM/L) of F^- content in drinking water. Control subjects showed a mean serum F^- concentration of 1.08 ± 0.350 μM/L. Males in control group showed slightly higher F^- levels (1.15 ± 0.334, range 0.55-1.9 μM/L) than females (0.92 ± 0.370, range 0.6-1.5 μM/L). Mean serum F^- concentration did not correlate significantly with age and sex among control subjects, whereas such correlation was observed in patients with ESRD on dialysis. Mean serum F^- concentration was significantly higher in patients on dialysis (2.67 ± 1.09, range 0.8-5.2 μM/L) than normal controls. When grouped according to sex, the mean serum F^- concentration in males (3.05 ± 1.04, range 1.8-5.2 μM/L) was significantly higher than females (2.38 ± 1.08, range 0.8-5.2 μM/L). When patients were grouped according to age, it was observed that F^- concentration was significantly higher in patients with age groups 21-70 (2.86 ± 1.05) than those with age group 13-20 years (1.42 ± 0.531). Thus F^- concentration correlated with age and sex, being higher in males and above 20 years. Despite appreciable clearance of F^- (39-90%) across the peritoneum, patients on continuous ambulatory PD (CAPD) showed higher serum F^- concentration than those on HD (3.1 ± 1.97 vs 2.5 ± 1.137 μM/L). Of the total 39 patients on dialysis 39% had their serum F^- concentration above 3.0 μM/L, posing the risk of renal osteodystrophy. Therefore we recommend frequent monitoring of serum F^- concentration in ESRD patients on CAPD/HD, particularly above age 20 years.

Key words: Dialysis; Haemodialysis; Ionic fluoride; Peritoneal dialysis; Renal disease; Renal osteodystrophy.

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LONG TERM FOLLOW UP OF IONIC PLASMA FLUORIDE LEVEL IN PATIENTS RECEIVING HEMODIALYSIS TREATMENT

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Abstract from *Clinica Chimica Acta 263* (1) 97-104 July 1997

The elimination half-life of fluoride is significantly increased in patients with chronic renal failure. This led us to conduct a study of variations of its plasma levels in 35 patients receiving dialysis treatment. In this population, there is a gaussian distribution of the values before and after the hemodialysis session,

with a significant decrease in the averages. Furthermore, there is a highly significant correlation between fluoride levels before and after the dialysis session ($P < 0.00001$), and also between the amount of time in hemodialysis (in months) and the average fluoride level before dialysis ($r = 0.624$; $P = 0.008$). The presence of a group of patients consuming fluoride waters such as Vichy St-Yorre Water was easily identified by their excessive fluoride levels (above 100 μg/L), which could have a tendency to increase the risks of this group.

Key Words: Hemodialysis; Plasma fluoride level; Vichy St-Yorre water.
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FLUORIDE CONCENTRATIONS OF INFANT FOODS

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Abstract from *Journal of the American Dental Association 128* (7) 857-863 July 1997

Infants who ingest high amounts of fluoride can be at risk of dental fluorosis. The authors analyzed the fluoride concentration of 238 commercially available infant foods. Fluoride concentrations ranged from 0.01 to 8.38 micrograms of fluoride per gram, with the highest fluoride concentrations found in infant foods containing chicken. Infant foods, especially those containing chicken, should be considered when determining total fluoride intake.

Key words: Fluoride content; Infant foods.
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ELECTROKINETIC BEHAVIOR OF FLUORIDE SALTS AS EXPLAINED FROM WATER STRUCTURE CONSIDERATIONS

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Abstract from *Journal of Colloid and Interface Science190* (1) 224-231 June 1 1997

Unlike the other silver halides, silver fluoride is positively charged in its saturated solution as determined by nonequilibrium electrophoresis measurements. In the absence of surface hydrolysis reactions, other fluoride salts (LiF, CaF_2, and MgF_2) also are positively charged in their saturated solutions. Furthermore, the electrokinetic behavior of these fluoride salts is rather insensitive to the fluoride ion activity in neutral or acidic solutions, and reversal of the sign of the surface charge by fluoride addition is not possible. Based on FTIR transmission spectra to describe the water structure of ionic solutions, in situ FTIR/internal reflection spectroscopy (FTIR/IRS) has been used to spectroscopically characterize interfacial water at fluoride salt surfaces. The experimental spectra were examined by consideration of the O-H stretching region (3000-3800 cm^{-1}) associated with the vibrational spectra of interfacial water. These results reveal a unique hydration state for fluorides and explain the anomalous

electrokinetic behavior of fluoride salts such as LiF, CaF_2, and MgF_2, which show an unexpected insensitivity to the fluoride ion concentration in solution. It appears that this insensitivity is due to the formation of strong hydrogen bonding of the fluoride ions with water molecules. This hydration state prevents the accommodation of excess fluoride ions at surface lattice sites and accounts for the observed electrokinetic behavior.

Key words: And ftir spectroscopy; Electrokinetic behavior; Fluoride salts; Ionic solids; Structure-breaking ions; Structure-making ions; Water structure.

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NEW METHOD FOR RETROSPECTIVE DETECTION OF EXPOSURE TO ORGANOPHOSPHORUS ANTICHOLINESTERASES: APPLICATION TO ALLEGED SARIN VICTIMS OF JAPANESE TERRORISTS

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Abstract from *Toxicology and Applied Pharmacology 146* (1) 156-161 1997 (September)

With regard to detection of exposure to anticholinesterase, the presently used methods have the disadvantage that they cannot detect either low-level exposures with certainty or the structure of the agent and the extent of poisoning. In principle, organophosphate-inhibited butyrylcholinesterase in human plasma is the most persistent and abundant source for biomonitoring of exposure to organophosphate anticholinesterases. Fluoride ions reactivate the inhibited enzyme readily at pH 4, converting the organophosphate moiety into the corresponding phosphofluoridate. Subsequent quantitation of the latter product provides a reliable, highly sensitive and retrospective method for detection of exposure to, or handling of, organophosphates such as nerve agents and organophosphorus pesticides. We applied the new procedure to serum samples from victims of the Tokyo subway attack by the AUM Shinriyko sect and from an earlier incident at Matsumoto. In serum of 10 of 11 victims from the Tokyo incident and of 2 of the 7 samples from the Matsumoto incident, reactivation with fluoride ions yielded sarin concentrations in the range of 0.2-4.1 ng/ml serum. Evidently, these victims had been exposed to an organophosphate with the structure $PriO(CH_3)P(O)X$, presumably with X = F (sarin). Several applications of the new procedure to establish nerve agent and/or organophosphate (OP) pesticide exposure can be envisaged, e.g., (i) in biomonitoring of exposure for health surveillance of those handling organophosphates, (ii) in cases of alleged exposure to nerve agents and/or OP pesticides in armed conflict situations or terrorist attacks, (iii) in medical treatment of intoxication, and (iv) in forensic cases against suspected terrorists that may have handled anticholinesterases.

Key words: Anticholinesterase; Fluoride ions; Sarin.

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DIETARY REFERENCE INTAKES
Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride

(Institute of Medicine, National Academy of Science, Washington DC 1997)

Reviewed by Richard G Foulkes BA MD•

This report, "Dietary Reference Intakes", in the words of its Preface "represents the initial report of a major new activity of the Food and Nutrition Board (FNB): the development of a comprehensive set of reference values for dietary nutrient intakes for the healthy population in the United States and Canada." A new activity that may refer to fluoride is "the examination of data about selected food components that have not been considered essential nutrients" (p vii).

With regard to fluoride, anyone familiar with previous US government reports on this subject [1,2] cannot avoid the conclusion that the report under review has as its objective the *justification* (or *rationalization*) of present high intake levels of fluoride by every age group, including pregnant and lactating females, that have been forced upon all by government policy. The report presents no new research to prove once and for all time in a way that can be replicated that fluoride (fluorine} is a nutrient that is "essential" for human growth and development. Neither the Food and Drug Administration (FDA) in 1978, nor the National Research Council/National Academy of Sciences {NRC/NAS) in 1989, has accepted the view that fluoride is an essential nutrient biologically for man.

This report is a review of *selected* references and contains errors, omissions, contradictions and biased manipulations of information. An adequate critique would have to be on a paragraph-by-paragraph basis thereby leading to the production of a lengthy volume. This would take resources not available to those whose cause is not favoured, as is the report under review, by the public purse.

It is not surprising to note that Health Canada is one of the contributors. A report prepared for Health Canada by a group of Canadian pro-fluoridation dental academicians was published in July 1994.[3] This effort resembles the present report in many ways. It was, in my view, an example of "tainted truth".[4]

To be specific: Does the Dietary Reference Intakes report present solid replicable evidence that fluoride is a nutrient to be placed alongside calcium, phosphorus, magnesium and vitamin D as a biochemically essential component of the human diet? Such positive evidence is a *sine qua non*. Without it, the proposals for a Recommended Daily Allowance (RDA), Estimated Average Requirement (EAR), Adequate Intake (AI), and Tolerable Upper Intake Level (UL) are without meaning. The UL, however, may be significant in estimating the possibilities of incurring adverse effects of fluoride which may be classified as a ubiquitous inessential component or a contaminant of the diet.

The report makes it clear (p 2-9) that "evidence on which to base an actual requirement (for fluoride) is scant . . . Because data are not available to determine an Estimated Average Requirement (EAR), the reference value that will be used for fluoride is the Adequate Intake (AI). The AI is based on estimated intakes that have been shown to reduce the occurrence of dental

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See also news release by EPA scientists and other professionals, page 258.

caries maximally in a population without causing unwanted side effects, including moderate fluorosis." (p 8-10).

To support this approach, the report presents the often used statement: "(the) ability (of fluoride) to inhibit and even reverse the initiation and progression of dental caries is well known. It also has the unique ability to stimulate new bone formation and, as such, it has been used as an experimental drug for the treatment of osteoporosis." (p 8-1)

As was the case in the Health Canada Report, no contemporary study could be found that effectively supports the belief in the cariostatic role. As a consequence, both reports are forced to present Dean's highly selective 1942 studies (and graphical representation) as the chief support. The authors of the Health Canada Report admitted that the 1986-87 survey of 39,000 plus US school children by the US National Institute for Dental Research (NIDR) was the most extensive contemporary study but showed no statistically significant difference between fluoridated and non-fluoridated groups.

The report under examination makes no such concession. Its authors stress the insignificant "18%" (sic) difference reported by Brunelle and Carlos[5] and manipulate the data in the Regional Survey Results[6] to obscure the finding that when the least fluoridated region (Region VII, "Pacific") was compared to the most highly fluoridated region (Region III, "Midwest") the *least* fluoridated population had the *most* children who were caries-free. The report fails to cite similar findings from Canada (Gray 1987)[7] and New Zealand (Colquhoun 1993)[8].

The authors of the report also deal with the effects which occur at very low intake of fluoride which they term "negative balance". They are forced to admit (p 8-10) that "no data document the effects of long-term negative balance on enamel, on salivary or plaque concentration or on caries development."

The "evidence" for the cariostatic activity of fluoride consists of the presentation of questionable data from the 1940s and of the manipulation of the results of contemporary surveys. There are no data that show detrimental effects of long-term absence of dietary fluoride. For these reasons, fluoride cannot be considered a nutrient along with such dietary components as calcium and vitamin D for which substances evidence is clear with regard to both positive contribution to health and negative physiological effects of long-term absence.

On the basis of their *belief* in the cariostatic action of fluoride, the authors of this report tabulate (Table S-5, p S-11) the "criteria and dietary reference intake values for fluoride by "life-stage group". The Adequate Intake (AI) for fluoride ranges from 0.01 mg/day for both sexes 0-6 months through 3.8 mg/day for males and 3.1 for females 19 to 70+ years. This includes 2.9-3.1 mg/day for both pregnant and lactating females. It is impossible to see these levels as "adequate" for anything but intoxication (poisoning) at some early stage for many individuals.

Of importance is the selective way in which the authors of this report derive the "Tolerable Upper Intake Levels (UL) by life-stage group" presented in Table S-6 (p S-12). The report states that these values are "set to protect the most sensitive individuals in the healthy general population (such as elderly

individuals who tend to have a decreased glomerular filtration rate)" (p S-11). The values range from 0.7 mg/day for 0-6 months to 10 mg/day for ages 9 years to 70 years, including pregnant and lactating females. There is contrary evidence even from sources cited by the authors, *e.g.* Hodge (1979),[9] who states that 10-25 mg/day over a period of 10-20 years can produce *crippling skeletal fluorosis*.

Important omissions are frequent in the selected studies used to produce these estimates. Enamel fluorosis is termed an "adverse cosmetic effect" rather than a "functional adverse effect", even in its most advanced forms (p 8-15). The authors refer to the papers by Fejerskov where his statements can be used to support their bias. They omit his description of the severity of the structural damage found in dental fluorosis[10] and his comment that "a daily dose of fluoride as low as 0.04 mg/kg body weight can result in dental fluorosis of the permanent dentition"[10] - presumably because they have selected an "optimal" daily level of 0.05 mg/kg body weight which, by comparison, is too high. They also ignore Fejerskov's comment that "a magic borderline below which the signs of dental fluorosis are totally absent for all people does not in reality exist."[11]

The report presents a case for increased bone density caused by fluoride but fails to discuss, in a meaningful way, the quality of bone produced and to take seriously the complicating factor of increased fracture of the femur (hip). With regard to the latter, the report plays down the effect on hip fractures of residence in fluoridated areas. Lee[12] points out that this relationship was shown to be positive in 7 out of 10 recent surveys comparing incidence relative to fluoridation status (including the results of two workshops held under the auspices of the NAS). In the same paper, Lee shows how the three negative studies deserve to be discarded.

The report mentions, but does not deal adequately with, the studies suggesting the "beneficial" effects on the primary teeth of the fetus in women receiving fluoride supplements. Leverett of the Eastman Dental Center, Rochester, New York, showed that Glenn's studies were incompetently carried out and that when he (Leverett) completed a well-designed study, the results showed conclusively that there was no difference in caries incidence or pregnancy outcome between the supplemented group and the control after five years.[13] Unfortunately, Leverett "on ethical grounds" gave both groups of children supplements after five years thereby sabotaging his own experiment. This experiment showed clearly the lack of benefit from strictly systemic use of fluoride.

There is no reference in the report to recently published work that suggests that low levels of fluoride delivered to the fetus through the placenta may cause brain damage resulting in behaviour disorder or low IQ.[14-16] They omit also recent evidence that fluoride may contribute to infertility.[17]

When the authors of this report discuss the important subject of skeletal fluorosis, they present confusing calculations for the daily amount and time interval. In some respects, for example in their rehashing of the controversial "findings" of the Bartlett-Cameron study, they appear to be caught in a time-warp. The report presents both the calculation, now known to be erroneous,

used in *Health Benefits and Risks,* 1991,[1] of "20-80 mg of fluoride per day for 10-20 years" and the now corrected value, found in the NRC/NAS Report of 1993,[2] of "10-20 mg of fluoride per day for 10-20 years." Because fluoride is cumulative, lower intake over a longer time period may result in crippling skeletal fluorosis. There are indications, therefore, that *both* the Adequate Intake (AI) and the Upper Intake Level (UL) values shown in Tables S5 and S6 of the report are set too high to prevent this serious complication.

The authors selected a study that showed a total fluoride intake for adults of 1.4-3.4 mg/day in fluoridated areas (1 mg/L) and 0.3-1.0 mg/day in non-fluoridated areas (<0.3 mg/L) (p 8-5). These estimates are *lower* than those presented elsewhere. For a 70 kg adult, a Health Canada survey[18] gives 3.22-4.06 mg/day in fluoridated and 2.24-2.45 mg/day in nonfluoridated areas; the US Public Health Service, in *Health Benefits and Risks,*[1] 2.1-9.1 mg/day in fluoridated (0.7.-1.2 mg/L) and 1.1-2.8 in non-fluoridated (< 0.3 mg/L). It may be shown from these intake estimates, especially for fluoridated populations, that whether the NAS (1993) estimate for skeletal fluorosis[2] or Roholm's 1937 findings for cryolite workers[19] are used, skeletal fluorosis (osteosclerosis) is a major possibility. Roholm's estimates can be used to show that a daily fluoride intake of 3.5-6.0 mg/day could produce "recognizable sclerosis" after 37 years of exposure and "severe sclerosis" around the age of 84. At the UL of 10 mg/day fluoride intake, this problem could occur after a shorter time interval.

The statement that "there was no evidence that dietary fluoride intake in the 1970s and 1980s had increased over the 1950s" is not in accord with common sense and not supported by research. Kintner[20] reviewed this to 1991 and estimated that total fluoride intake for an adult in the U.S. had increased from 0.45-0.55 mg/day prior to 1950 to a *mean* of 2.7 mg/day in fluoridated communities in 1991. Horowitz (1992)[21] recognized the increase for residents of *both* fluoridated and non-fluoridated areas. Recently, studies have shown elevations of fluoride in juices, juice-flavoured drinks and frozen concentrates reconstituted with distilled water, sold in the US (Kiritsy 1996)[22] and in California wines contaminated by the pesticide cryolite (Burgstahler 1997)[23].

CONCLUSION

Once again, a report on fluoride (and fluoridation) is presented that is more propaganda than science. Supporters of fluoridation and the users of other fluoride "technologies" such as the American Dental Association, will obtain comfort from the "confirmation" that the cariostatic effects of fluoride have been given recognition as an indicator for the establishment of Adequate Intake levels, in spite of the inability of the authors to find data to show that fluoride is an "essential nutrient" or that there is contemporary evidence to prove that the systemic use of fluoride is effective as a cariostatic agent. The "new" hypothesis concerning the "benefit" of topical use, presented at length in this report, appears to be more conjecture than reality, representing an adjustment to the failed concept of the past that "rationalized" mass medication via the drinking water supply.

The "confirmation" that dental fluorosis is an "adverse *cosmetic* effect" rather than an "adverse *functional* effect" will be hailed by fluoride promoters as it fits well the preconceived notion. The "reaffirmation" that new bone formation from fluoride ingestion is a positive finding rather than a harbinger of calamity to come will also receive favourable notice. The possibility of increased risk of hip fracture and of skeletal fluorosis is minimized, either by omission or down-grading present daily fluoride intake. The tables showing the Adequate Intake (AI) and Tolerable Upper Levels (UL) are calculated to defend the concept that current intakes are "necessary for good dental health" and are at levels that are "safe". This Institute of Medicine Report ignores a vast body of contrary evidence that undermines the seemingly sure positions that it seeks to impose on an unquestioning public. It makes a mockery of true science, which requires consideration of *all* relevant data *before* reaching conclusions.

There is *some* truth in this report; but, like the search for Waldo, the search for truth is made difficult to find amidst the background verbiage. What are these "truths"?

First, there is no evidence to show that fluoride is an "essential" nutrient or a "nutrient" as opposed to a "contaminant" of human diet.

Second, there is no solid evidence that the *systemic* use of fluoride is cariostatic. There is, also, little to support the "benefits" of topical application. Therefore, the term "Adequate Intake" is meaningless.

Finally, it cannot be hidden, even in this biased report, that there are many "red flags" that should serve as a warning that serious dental, bone and other adverse effects will become apparent as the future unfolds.

Note: The prepublication proof of the report, from the Institute of Medicine, National Academy of Sciences Press, Washington DC 1997, was used for this review.

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US ENVIRONMENTAL PROTECTION AGENCY SCIENTISTS CONDEMN NATIONAL ACADEMY OF SCIENCES REPORT ON FLUORIDE

The following statement was issued on September 23, 1997, on behalf of scientists, engineers, lawyers and other professionals at Headquarters of the US Environmental Protection Agency:

The document "Dietary Reference Intakes. Prepublication Copy" (DRI) is seriously flawed and deficient as an instrument to justify a public policy to establish fluoride as an "essential nutrient". The DRI document is rife with inadequacy, error and deceptive information, only some of which can be touched upon here.

For example, the only adverse effects of fluoride exposure discussed in DRI are enamel and skeletal fluorosis. These effects are only cursorily and deceptively touched upon, and no connection is made between them, as though they were independent effects and fluoride affinity for and damage to enamel is not a biochemical window on what is happening in bone.

While DRI lays out the parameters for conducting risk assessments in Chapter 3, it ignores application of those parameters particularly egregiously with respect to fluoride in purporting to establish a "tolerable upper intake level". One component of risk assessment is hazard identification, whose components include addressing evidence of adverse effects in humans. DRI attempts to deceive the public into believing the only identified adverse effects of fluoride exposure of significance are those mentioned above. The DRI document omits any mention of studies in humans showing increased risk of hip fractures and bone cancer and decreased IQ in children in areas with artificially fluoridated water or other sources of dietary fluoride that result in fluoride intakes that are below the "tolerable appear intake". Neither does the DRI document properly address use of animal data in the hazard and risk assessments on fluoride.

There are recent (1990-1995) animal data supporting concern for both cancer and central nervous system effects.

Even if one grants as accurate the statement at page 8-15 in the prepublication copy of DRI, "Most research has indicated that an intake of at least 10 mg/day for 10 or more years is needed to produce clinical signs of the milder forms of the condition" (skeletal fluorosis), consider the simple mathematics of this "tolerable upper intake" level. That level is set at 10/mg/day for individuals aged 9 years and up. At age 39, the individual who has received the "tolerable upper intake" since age 9 will have accumulated 3 times the amount of fluoride needed, according to the DRI, to put him or her at high risk of skeletal fluorosis – not to mention bone fracture, cancer and decreased mental capacity

When a chemical manufacturer wants to make a new chemical to use, for example, as an additive in motor oil, all existing toxicological data must be presented to the Environmental Protection Agency for review of potential risks before manufacture and use can begin. In the DRI we see risk assessment principles, as applied to a major public policy issue, flouted – even the existence of a massive body of information on adverse effects of fluoride is

ignored, let alone discussed. And this for a chemical the National Academy recommends we purposely add to our diets, not our motor oil.

Furthermore, the claimed benefits from the "adequate intake" level have been shown to be based on biased or otherwise flawed studies. Not a single one of those studies was a randomized control trial.

In summary, our union members' review of the literature over the last 11 years has led us to conclude that a causal link exists between fluoride exposure and cancer, increased risk of hip fracture, and damage to the central nervous system. For the National Academy of Sciences to attempt to anoint this substance an "essential nutrient" is a travesty and a matter of shame for the US science community.

National Federation of Federal Employees Local 2050 represents and is comprised of scientists, lawyers, engineers and other professionals at Headquarters, US Environmental Protection Agency, Washington DC.

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On July 2, 1997, the above professionals had announced their unanimous decision to co-sponsor the campaign to reverse California's 1995 law making water fluoridation mandatory. In a letter announcing the decision Dr Hirzy stated:

It is our hope that our co-sponsorship will have a beneficial effect on the health and welfare of all Californians by helping to keep their drinking water free from a chemical substance for which there is substantial evidence of adverse health effects and, contrary to public perception, virtually no evidence of significant benefits.

These judgements are based, in part, on animal studies of the toxicity of fluoride coupled with the human epidemiology studies which corroborate them, and the studies of rates of decayed, missing and filled teeth in the United States (fluoridated and non-fluoridated communities) *versus* non-fluoridated European countries.

As professionals who are charged with assessing the safety of drinking water, we conclude that the health and welfare of the public is not served by the addition of this substance to the public water supply.

[The following is the text of a resolution approved by the City Council of Bellingham, Washington, USA, on September 15, 1997.]

RESOLUTION NO. 42-97
A RESOLUTION OF THE CITY OF BELLINGHAM RECOGNIZING THE IMPROPRIETY OF THE USE OF ITS PUBLIC WATER SYSTEM TO DISPENSE FLUORIDE TREATMENT TO THE GENERAL POPULATION

WHEREAS, fluoridation of all public water systems for the stated purpose of reducing tooth decay in the general population is the published goal of the Washington State Department of Health and of the US Public Health Service, and

WHEREAS, tooth decay is a personal health matter and does not pose a threat of contagion or life-threatening injury to the community, and

WHEREAS, the addition of fluoride to public water systems denies equal access to the common water supply to persons who cannot or do not wish to undergo fluoride treatment, and

WHEREAS, Local 2050 of the National Federation of Federal Employees, representing the 1,500 scientists and attorneys who evaluate US Environmental Protection Agency regulations, has found that the health and welfare of the public is not served by fluoridation,

NOW THEREFORE,

BE IT RESOLVED BY THE CITY COUNCIL OF THE CITY OF BELLINGHAM: That the City of Bellingham finds the use of its water system to dispense fluoride treatment to the general population to be improper.

BE IT FURTHER RESOLVED that this Resolution shall be forwarded to the Governor of the State of Washington with the request that state health policy promoting fluoridation be changed in light of the findings herein enumerated; and

BE IT FURTHER RESOLVED that this Resolution shall be forwarded to the elected Senators and Representatives of Washington Legislative Districts 40 and 42, of which the City of Bellingham is a part, with the request that said legislators consider the need for legislation to forbid state-mandated fluoridation and to secure for all the citizens of Washington the right of individual choice with regard to fluoride treatment.

Passed September 15, 1997

Address: City of Bellingham, 210 Lottie Street, Bellingham, Washington 98225, USA. Phone 360 676 6900 Fax 360 676 7693

FLUORIDE, TEETH AND THE ATOMIC BOMB

by Joel Griffiths and Chris Bryson

(Abstracted from the environmental newsletter *Waste Not #414*, September 1997)

The *Christian Science Monitor* commissioned this report in the spring of 1997, but it remained unpublished until the authors gave it to *Waste Not*, and others, with a short note: "use as you wish." Release of hundreds of declassified World War II and Atomic Bomb (Manhattan) Project documents told a disquieting story, that fluoridation of public drinking water was rooted in protecting the US atomic bomb program from litigation. Also brought out is the dirty politics involving dental and public health officials in the selling of fluoridation to a trusting public.

About the authors:

Joel Griffiths, a medical writer in New York City, is author of a book on radiation hazards and numerous articles for medical and popular publications. Chris Bryson holds a Masters degree from the Columbia University Graduate School of Journalism, and has worked for the British Broadcasting Corporation, *The Manchester Guardian*, *The Christian Science Monitor* and Public Television.

The Manhattan Atomic Bomb Project

Massive quantities of fluoride, one of the most toxic chemicals known, were used in the manufacture of bomb-grade uranium and plutonium for nuclear weapons throughout the Cold War. It was a leading health hazard of the U.S atomic bomb program for workers and nearby communities. A 1944 secret memo from Dr Harold C Hodge, then chief of fluoride toxicology studies for the University of Rochester division of the Manhattan Project, reported that clinical evidence suggests that the fluoride component of uranium hexafluoride may have a rather marked central nervous system effect. The memo is addressed to the head of the Manhattan Project's Medical Section, who immediately approved a program of animal research on CNS effects, because it was obvious that workers were being severely affected.. The proposal and the results of the CNS research, however, are missing from the files of the US National Archives. The chief librarian at the Atlanta branch of the US National Archives and Records Administration, where the memos were found, said they were probably still classified.

The du Pont fluoride emissions

In 1944, a severe fluoride pollution incident occurred downwind of the du Pont chemical factory in New Jersey, which was producing millions of pounds of fluoride for the Manhattan project. The owners of the farms downwind began to report in 1943 that their crops were blighted and they, their workers and animals were ill. The detailed symptoms are cardinal signs of fluoride toxicity; according to veterinary toxicologists. Taped interviews of the farmers' accounts were confirmed by Philip Sadtler, of Sadtler Laboratories of Philadelphia, who had personally conducted the initial investigation of the damage.

The New Jersey farmers, at the end of the war, sued du Pont and the Manhattan Project for fluoride damage. Secret documents reveal the seemingly trivial lawsuits shook the government. Manhattan Project chief Major General Groves personally directed secret meetings in Washington, with compulsory attendance by scores of scientists and officials from the US War Department, the Manhattan Project, the

Food and Drug Administration, the Agriculture and Justice Departments, the US Army's Chemical Warfare Service and Edgewood Arsenal, the Bureau of Standards, and du Pont lawyers. Declassified memos of the meetings reveal a secret mobilization of the full forces of the government to defeat the New Jersey farmers. These agencies "are making scientific investigations to obtain evidence which may be used to protect the interest of the Government at the trial of the suits brought by owners of peach orchards in ... New Jersey," stated a Manhattan Project officer in a memo copied to General Groves. In 1946 a Manhattan Project officer persuaded the Food and Drug Administration (FDA) to drop a threatened embargo of the region's produce because of "high fluoride content." The US Army's Chemical Warfare Service (CWS) took over testing of fluoride in the New Jersey area from the Department of Agriculture because "work done by the CWS would carry the greatest weight as evidence if lawsuits are started by the complainants." The A-bomb was seen as crucial for US leadership of the postwar world. The New Jersey fluoride lawsuits were a serious roadblock to that strategy.

Meanwhile, local citizens were in a panic about fluoride. The farmers' spokesman, Willard B Kille, was personally invited to dine with General Groves at his office at the War Department on March 26, 1946. Although he had been diagnosed with fluoride poisoning by his doctor, Kille departed the luncheon convinced of the government's good faith. Chief fluoride toxicologist Hodge suggested countering the local fear of fluoride through lectures on F toxicology and perhaps the usefulness of F in tooth health. Such lectures were indeed given not only to New Jersey citizens but to the rest of the nation throughout the Cold War.

The government's refusal to reveal how much fluoride du Pont had vented into the atmosphere during the war stymied the lawsuits. "Disclosure ... would be injurious to the military security of the United States," wrote Manhattan Project Major C A Taney Jr. The farmers were pacified with token financial settlements, according to interviews with descendants still living in the area. Angelo Giordano recalled that his father had received a settlement of about $200.

Fluoride safety studies

The farmers' claims of injury to their health triggered intensive secret bomb-program research on the health effects of fluoride. In a March 1 1946 secret memo, Hodge wrote to Colonel Warren about "problems associated with the question of fluoride contamination of the atmosphere in New Jersey ..." The fluoride studies were conducted with the same ethical mind-set, in which "national security" was paramount. The US government's conflict of interest and its motive to prove fluoride "safe" has not until now been made clear to the general public, civilian researchers, health professionals, or journalists. Fluoride safety studies were delegated to the prestigious upstate New York University of Rochester, which had housed a key wartime division of the Manhattan Project, studying the health effects of the new "special materials," such as uranium, plutonium, beryllium, and fluoride, being used to make the atomic bomb. That work continued after the war, with millions of dollars flowing from the Manhattan Project and its successor organization, the Atomic Energy Commission (AEC). Indeed, the bomb left an indelible imprint on all US science in the late 1940s and 50s. Up to 90% of federal funds for university research came from either the Defense Department or the AEC in this period, according to Noam Chomsky's 1996 book *The Cold War and the University*.

The University of Rochester medical school became a revolving door for senior bomb program scientists. Postwar faculty included Stafford Warren, the top medical officer of the Manhattan Project, and Harold Hodge, chief of fluoride research for the bomb program. Hodge directed the University of Rochester's classified fluoride studies, code-named Program F, which were conducted at its Atomic Energy Project (AEP) top-secret facility funded by the AEC and housed in Strong Memorial Hospital. It was there that one of the most notorious human radiation experiments of the Cold War took place, in which unsuspecting hospital patients were injected with toxic doses of radioactive plutonium. Revelation of this experiment in a Pulitzer prize-winning account by Eileen Wellsome led to a 1995 US Presidential investigation, and a multimillion-dollar cash settlement for victims.

A 1948 document details Program F's purpose to "supply evidence useful in the litigation arising from an alleged loss of a fruit crop several years ago, a number of problems have been opened. Since excessive blood fluoride levels were reported in human residents of the same area, our principal effort has been devoted to describing the relationship of blood fluorides to toxic effects." Lawyer Kittrell commented "This and other documents indicate that ... studies undertaken for litigation purposes by the defendants would not be considered scientifically acceptable today because of their inherent bias to prove the chemical safe." The reporters obtained an original secret version of a 1948 study published by Program F scientists in the *Journal of the American Dental Association* showing that evidence of adverse health effects from fluoride was censored by the US Atomic Energy Commission (AEC) for reasons of national security. Unfortunately, much of the proof of fluoride's safety rests on the work performed by Program F scientists at the University of Rochester. During the postwar period that university emerged as the leading academic center for establishing the safety of fluoride, as well as its effectiveness in reducing tooth decay, according to Dental School spokesperson William H Bowen MD. Bowen also said that Hodge was the key figure in this research and became a leading national proponent of fluoridating public drinking water.

Water fluoridation at Newburgh

The bomb program needed human studies, as they had needed human studies for plutonium, and adding fluoride to public water supplies provided one opportunity. Planning began in 1943 with the appointment of a special New York State Health Department committee chaired by Hodge, chief of fluoride toxicity studies for the Manhattan Project, to study the advisability of adding fluoride to Newburgh's drinking water. Subsequent members included Henry L Barnett, a captain in the Project's medical section, and John W Fertig, in 1944 with the office of Scientific Research and Development, the Pentagon group which sired the Manhattan Project. Their military affiliations were kept secret: Hodge was described as a pharmacologist, Barnett as a pediatrician. Placed in charge of the Newburgh project was David B Ast, chief dental officer of the State Health Department. Ast had participated in a key secret wartime conference on fluoride held by the Manhattan Project, and later worked with Hodge on the Project's investigation of human injury in the New Jersey incident, according to once-secret memos. The Newburgh Demonstration Project is considered the most extensive study of the health effects of fluoridation, supplying much of the evidence that low doses are safe for children's bones, and good for their teeth.

The committee recommended that Newburgh be fluoridated, selecting the types of medical studies to be done, and "provided expert guidance" for the duration of the experiment. The key question to be answered was: "Are there any cumulative effects – beneficial or otherwise, on tissues and organs other than the teeth of long-continued ingestion of such small concentrations ...? " This information was also sought by the bomb program, which would require long-continued exposure of workers and communities to fluoride throughout the Cold War. In May 1945, Newburgh's water was fluoridated, and over the next ten years its residents were studied by the State Health Department. In tandem, Program F conducted its own secret studies, focusing on the amounts of fluoride Newburgh citizens retained in their blood and tissues. Health Department personnel cooperated, shipping blood and placenta samples to the Program F team. The samples were collected by the Department's chief of pediatric studies at Newburgh. The final report of the Newburgh Demonstration Project, published in 1956 in the *Journal of the American Dental Association*, concluded that "small concentrations" of fluoride were safe for US citizens. Hodge delivered the biological proof, "based on work performed ... at the University of Rochester Atomic Energy Project".

News of the secret involvement of scientists from the atomic bomb program is now greeted with incredulity. "I'm shocked – beyond words," said present-day Newburgh Mayor Audrey Carey, commenting on these reporters' findings. "It reminds me of the Tuskegee experiment that was done on syphilis patients down in Alabama." Mayor Carey recalls, as a child in the early 1950s, doctors from the Newburgh fluoridation project studied her teeth, and a peculiar fusion of two finger bones on her left hand she had been born with. Today, adds Carey, her granddaughter has dental fluorosis on her front teeth. Mayor Carey wants answers from the government about the secret history of fluoride and the Newburgh fluoridation experiment. "I absolutely want to pursue it," she said. "It is appalling to do any kind of experimentation and study without people's knowledge and permission."

Contacted by these reporters, the director of the Newburgh experiment, David B Ast, denies knowledge of Manhattan Project scientists' involvement in blood and placenta sampling from Newburgh. Ast could not recall participating in the Manhattan Project's secret wartime conference on fluoride in January 1944, or going to New Jersey with Hodge to investigate human injury in the du Pont case in spite of the evidence of secret memos. A spokesperson for the University of Rochester Medical Center, Bob Loeb, confirmed that blood and tissue samples from Newburgh had been tested by the University's Dr Hodge. On the ethics of secretly studying US citizens to obtain information useful in litigation against the A-bomb program, he said, "that's a question we cannot answer", referring inquiries to the US Department of Energy (DOE), successor to the Atomic Energy Commission.

"Missing" documents

A spokesperson for the DOE in Washington, Jayne Brady, confirmed that a review of DOE files indicated that a "significant reason" for fluoride experiments conducted at the University of Rochester after the war was "impending litigation between the du Pont company and residents of New Jersey areas." However, she denied that DOE has documents to indicate that fluoride research was done to avoid lawsuits. She also denied that the DOE or predecessor agencies, especially

the Manhattan Project, authorized fluoride experiments to be performed on children in the 1940s. When told that the reporters had several documents that directly tied the Manhattan Project's successor agency at the University of Rochester, the AEP, to the Newburgh experiment, she later conceded that her search had been confined to radiation effects - "fluoride was not part of our research effort" - and that relevant documents may be in a classified collection at the DOE Oak Ridge National Laboratory known as the Records Holding Task Group. "This collection consists entirely of classified documents removed from other files for the purpose of classified document accountability many years ago," she said, and was "a rich source of documents for the human radiation experiments project."

The crucial question arising from this investigation is: Were adverse health findings from Newburgh and other bomb-program fluoride studies suppressed? All AEC-funded studies had to be declassified before publication in civilian medical and dental journals. Where are the original classified versions? The transcript of one of the major secret scientific conferences of WWII on "fluoride metabolism" is missing from the files of the US National Archives. Participants in the conference included key figures who promoted the safety of fluoride and water fluoridation to the public after the war – Harold Hodge of the Manhattan Project, David B Ast of the Newburgh Project, and US Public Health Service dentist H Trendley Dean, popularly known as the "father of fluoridation." "If it is missing from the files, it is probably still classified," National Archives librarians told these reporters. A 1944 WWII Manhattan Project classified report on water fluoridation is missing from the files of the University of Rochester Atomic Energy Project, the US National Archives, and the Nuclear Repository at the University of Tennessee, Knoxville. The next four numerically consecutive documents are also missing, while the remainder of the "MP-1500 series" is present. "Either those documents are still classified, or they've been 'disappeared' by the government" says Clifford Honicker, Executive Director of the American Environmental Health Studies Project in Knoxville, Tennessee, which provided key evidence in the public exposure and prosecution of US human radiation experiments. Seven pages have been cut out of a 1947 Rochester bomb-project notebook entitled "Du Pont litigation." "Most unusual," commented chief medical school archivist Chris Hoolihan. Similarly, Freedom of Information Act (FOIA) requests by these authors over a year ago with the DOE for hundreds of classified fluoride reports have failed to dislodge any. "We're behind" explained Amy Rothrock, FOIA officer for the Department of Energy at their Oak Ridge operations.

These reporters made what appears to be the first discovery of the original classified version of a fluoride safety study by bomb program scientists. A censored version of this study was later published in the August 1948 *Journal of the American Dental Association*. Comparison of the secret with the published version indicates that the US AEC did censor damaging information on fluoride, to the point of tragicomedy. This was a study of the dental and physical health of workers in a factory producing fluoride for the A-bomb program, conducted by a team of dentists from the Manhattan Project. The secret version reports that most of the men had no teeth left. The published version reports only that the men had fewer cavities. The secret version says the men had to wear rubber boots because the fluoride fumes disintegrated the nails in their shoes. The published

version does not mention this. The secret version says the fluoride may have acted similarly on the men's teeth, contributing to their toothlessness. The published version omits this statement. The published version concludes that "the men were unusually healthy; judged from both a medical and dental point of view."

Asked for comment on the early links of the Manhattan Project to water fluoridation, Dr Harold Slavkin, Director of the National Institute for Dental Research, the US agency which today funds fluoride research, said "I wasn't aware of any input from the Atomic Energy Commission." Nevertheless, he insisted, fluoride's efficacy and safety in the prevention of dental cavities over the last fifty years is well proved. "The motivation of a scientist is often different from the outcome," he reflected. "I do not hold a prejudice about where the knowledge comes from."

Recent CNS research

In the early 1990s Dr Phyllis Mullenix, former head of toxicology at Forsyth Dental Research Center in Boston, conducted animal studies which indicated that fluoride was a powerful central nervous system (CNS) toxin, and might adversely affect human brain functioning, even at low doses. The results were published in 1995 in a reputable peer-reviewed scientific journal. New epidemiological evidence from China adds support showing a correlation between fluoride exposure and diminished IQ in children. During her investigation, Mullenix could find virtually no previous US studies of fluoride's effects on the human brain. Her application for a grant to continue her CNS research was turned down by the US National Institutes of Health (NIH) panel, because "fluoride does not have central nervous system effects." Dr Antonio Noronha, an NIH scientific review advisor familiar with Dr Mullenix's grant request, says her proposal was rejected by a scientific peer-review group. He terms her claim of institutional bias against fluoride CNS research "farfetched" and he adds, "We strive very hard at NIH to make sure politics does not enter the picture." After reviewing the secret memos, Mullenix was "flabbergasted", continuing: "How could I be told by NIH that fluoride has no central nervous system effects when these documents were sitting there all the time?" She reasons that the Manhattan Project did do fluoride CNS studies, because the dangers to workers and the bomb program could not be ignored, but that the results were buried because they might create a difficult legal and public relations problem for the government.

Nearly fifty years later at the Forsyth Dental Center in Boston, Dr Mullenix was introduced to Hodge, who had been brought in to serve as a consultant on her CNS research. By then Hodge had achieved emeritus status as a world authority on fluoride safety, but "he never once mentioned the CNS work he had done for the Manhattan Project." Mullenix refuses to abandon the issue. After comparing the secret and published versions of the censored study, she commented: "This makes me ashamed to be a scientist." Of other Cold War-era fluoride safety studies, she asks, "Were they all done like this?"

Archival research by Clifford Honicker

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REPORT OF MEETING

In late October last year at Clark University in Worcester, Massachusetts, Drs Paul Connett, David Kennedy and Phyllis Mullenix gave presentations critical of water fluoridation, to assist the people of that US city to decide whether or not to fluoridate. Pro-fluoridation representatives, in accordance with their policy of not debating the fluoridation issue, had declined invitations to attend. On November 5, 1996, the citizens of Worcester decisively rejected fluoridation.

Dr Connett said that the only claimed benefit of fluoride was reduction of dental caries. He argued that the risks of dental fluorosis, interference of fluoride with hydrogen bonds and enzymes, skeletal fluorosis, and the susceptibility of a large subset of the population to fluoride, far outweighed any benefit.

Dr Kennedy said nine studies now show the positive association of increased hip fracture in people exposed for more than 20 years to fluoride. He also exhibited a graph for the whole population of the United States. This graph showed that the fluoridated, partially fluoridated and non-fluoridated populations data on age/dental caries were indistinguishable. He stated that the evidence for a positive association of fluoride to osteosarcoma was still equivocal. Kennedy agreed with Connett that we should not be experimenting and should wait until fluoride was proven safe.

Dr Phyllis Mullenix is co-author of a paper on the effects of fluoride on the central nervous system (CNS) of rats. The research indicated that fluoride accumulated in the brain and caused permanent alteration in rat behaviour. The rats exposed prenatally were hyperactive, while weanlings and adults showed cognitive deficits, exhibiting the "couch potato" syndrome.

She then described recently declassified documents from the Manhattan program (the project to create the atomic bomb). These showed that back in 1944 the effects of fluoride on the CNS were known and that affected workers' behaviour was sufficiently erratic to make them unsafe. Some of the affected workers exhibited the "couch potato" syndrome. Permission was granted to perform experiments on rats. Six months later this permission was withdrawn and it was ordered that whether experiments had started or not, they were not to proceed. It was 50 years later that Dr Mullenix did these experiments. She quoted research which stated that the symptoms of some fluoride exposed people looked very much like the chronic fatigue syndrome.

Coincidentally Dr H C Hodge, chief toxicologist on the Manhattan project, consulted in her department. He never discussed his previous work with her and it was only later that she discovered his involvement with the atomic project. Dr Mullenix described a declassified document written by Hodge, which he wrote after visiting a site badly affected by fluoride emissions. The document stated that people were poisoned by hydrogen fluoride, the fruit from the orchards were to be destroyed and that some windows had been etched by the fluoride. At the end of that letter Hodge suggested that an attempt should be made to allay the local people's fear of fluoride by suggesting it was useful for dental health. Dr Mullenix tellingly ended her presentation stating "So the next time someone comes up to you and says that fluoride is good for your teeth, I would just say no!"

CRITIQUE OF STUDY

(C Teo, W G Young, T J Daley and H Sauer Prior fluoridation in childhood affects dental caries and tooth wear in a south east Queensland population *Australian Dental Journal 42* (2) 92-102 1997)

The abstract of this study states: "Ninety-six South East Queensland subjects were studied. Their histories revealed three groups; a fluoride (F^-) in water supply, a F^- by supplement, and a non-fluoridated (non F^-) group. Significantly higher caries experience was found in the non-F^- group compared with F^- in water group and the F^- supplement group. No statistically significant difference in caries experience was found between the F^- in water and F^- supplement groups. Overall, tooth wear affected more sextants of the dentitions of non-fluoridated, high-caries subjects than of fluoridated low-caries subjects. Comparisons of wear patterns on sextants of the dentitions, between the fluoridated and non-fluoridated groups, revealed that in sextants where attrition was present no marked differences were discernible between the two groups. However, in most sextants where incisal, palatal, occlusal or non-occlusal erosion was found, this type of wear was commoner in non-fluoridated subjects. The exceptions were the mandibular molar sextants, where prior fluoride-exposure did not appear to protect against occlusal erosion patterns. This study showed that fluoride exposure during the first 12 years of life, which reduced dental caries in this population, may also protect teeth from wear to some extent."

The full study does not support the conclusions in the abstract. Of 120 patients preselected for tooth wear, after being specifically referred by dental practitioners for inclusion in the study, 96 with a known history of fluoride (F) exposure were retained. The majority of the group were not born in Queensland and "many had a background of loss of salivary protection due to occupation/sport dehydration or to drug-induced salivary hypofunction." The F exposure of the study group divided into: water F 25, supplementary F 24, and non-F group 47: *i.e.* 49 with F exposure and 47 without.

Table 1 details the selected groups, according to their F exposure, excluding toothpaste, during their first 12 years of life:

Age Range (Years)	F^- in water exposure	F^- supplement exposure	Non -F^-
10-34	19	22	17
35-49	6	1	21
50 plus	0	1	9
Totals	25	24	47

The aim of the study was to determine if early life F exposure reduced later life caries and tooth wear. Quotes from the study:

"In general, their oral hygiene methods reflected evidence of good instruction by dental personnel, and an awareness of the protective effects of fluoride."

"This was as expected in a population attending for regular dental care and hence referred by their dentists for inclusion in this study".

"In general, it was found that DMFS scores in all groups increased with age in a linear manner; however; this was most evident in the non-fluoridated group perhaps because 9 subjects over the age of 49 had not received fluoride".

"... in sextants where attrition was present no marked differences were discernible between the fluoridated and non-fluoridated groups".

"... in most sextants where incisal, palatal or occlusal and non-occlusion erosion was found, these patterns ... were commoner in non-fluoridated subjects".

Ten subjects over the age of 49 were excluded from the statistical comparisons of caries experience because most had not had fluoride exposure during the first 12 years of life. The data were grouped by age into 10-49 yrs, 10-29 yrs and 30-49 yrs for each treatment group. Student's t test was used to test significance of caries differences, and a relative risk test was performed on these data. The total 96 subjects then were divided into F exposed group (49) and non-F group (47). Both groups were subdivided by DMFS scores, above and below 10%, into high- and low-caries groups. For each sextant of the dentition, the percentage of treatments, showing all patterns of tooth wear at that site, were recorded for each subject and the data subjected to chi-square analysis.

Comment:

1. The F-exposed group of 49 were pooled for comparison with the non-F group of 47 in the analysis of tooth wear. The 50+yr subjects were included in their respective groups. The value of the results can be judged from the age disparity in Table 1: 8 of 49 were over the age of 35 in the F group; 30 of 47 were older than 35 in the non-F group. Caries and wear can increase with age.
2. Despite the decision to leave the 50+yr group out of the caries statistical analysis, Fig 11 of their study showed the DMFS experience of this group (8 non-F and 1 F supplement), presumably to enhance the authors' pro-F views.
3. It is not clear how many 10-12 year olds were included, who would appear to be ineligible for the study, given its aim.
4. Given the high rates of dental fluorosis in most fluoridated and even in non-fluoridated areas because of the "halo" effect, the total absence of dental fluorosis in this study group is unusual.
5. In this study, topical fluoride from toothpaste appears to have had little if any effect, a finding which is contrary to the prevailing view that it is topical rather than systemic fluoride that confers any substantial benefit.

The authors appear to have known what they wanted to find and had no difficulty doing so. This was a very unusual "cross section" study group. The non-random selection, the age differences between the sub groups, disparate origins of the subjects, extensive salivary hypofunction and small sample sizes make this study of little value. The only redeeming feature was the work on classification of tooth wear. It is a pity it could not have been applied to a more representative group.

Bill Wilson
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OFFICIAL "SAFE" FLUORIDE INTAKES BASED ON ARITHMETIC ERROR

What is the minimum intake of fluoride (F) which causes skeletal fluorosis, and how long is it before the onset of this disease? Kaj Roholm's 1937 study of industrial fluorosis showed that phases of skeletal fluorosis could occur, with an F intake of 0.2-0.35 mg/kg of body weight/day, after 2 yrs and 5 months for phase one; 4 yrs and 10 months for phase two and 11 yrs and 2 months for phase three (crippling skeletal fluorosis).[1]

Descriptions of the symptoms and range of F in bone ash for each clinical phase of skeletal fluorosis follow:-

Phase 1: sporadic pain; stiffness of joints; osteosclerosis of pelvis and vertebral column (6,000-7,000 ppm F in bone ash).

Phase 2: chronic joint pain; arthritic symptoms; slight calcification of ligaments; increased osteosclerosis/cancellous bones; with/without osteoporosis of long bones (7,500-9,000 ppm F in bone ash).

Phase 3: Crippling Skeletal Fluorosis: limitation of joint movement; calcification of ligaments/neck and vertebral column; crippling deformities of spine and major joints; muscle wasting; neurological defects/ compression of spinal cord (more than 8,400 ppm F in bone ash).

Approximately 50% of ingested fluoride is cleared by the kidneys.[2]

In 1953, Dr Harold C Hodge, the leading F toxicologist, applying Roholm's intake dosage range of 0.2-0.35 mg/kg/day to the range of weights of 100-229 lbs, concluded that 20-80 mg/day of fluoride intake for 10-20 years would be necessary to produce skeletal fluorosis. This result was published in 1953 by the National Academy of Sciences/National Research Council (NAS/NRC). In 1990, in response to queries about fluoride in a NAS/NRC publication,[3] NAS/NRC quoted Roholm as supporting evidence for the statement that "fluorosis occurs after years of daily exposure of 20-80 mg/day".

Unfortunately, Hodge had made an incredible blunder, because he had not corrected for pounds (lbs). He had calculated 0.2 mg x 100 (lbs) = 20 mg and 0.35 x 229 (lbs) = 80 mg giving a range of 20-80 mg/day.

This error was repeated in numerous subsequent publications purporting to report the range of intakes required to produce skeletal fluorosis. The erroneous statement was in the NAS/NRC book *Fluorides* of 1971. In 1991 NAS/NRC in a letter quoted additional supporting references: Hodge and Smith 1965 (In *Fluorine Chemistry*. Academic Press, New York); *Fluorides and Human Health*, WHO 1970; *Fluoride*, and *Fluorine and Fluorides*. WHO 1984. This letter also stated "The RDA subcommittee did not conduct experiments and come up with this range; rather, we reported this figure based on the work of others and on review papers." However, all the above reviews had accepted Hodge's miscalculation from Roholm's classic study. Thus all the above supporting references, which have been widely cited to support the safety of water fluoridation, contained the same erroneous information.

The corrected intake range for people between 100-229 lbs is 9.1-36.4 mg/day (based on 2.2 lbs/kg). Hodge partially corrected his error in a 1979 paper stating: "Crippling fluorosis as an occupational disease follows exposures estimated at 10 to over 25 mg of fluoride daily during periods of 10-20 years."[4] In a 1993

American Dental Association pamphlet, *Fluoridation Facts*, the incorrect dosage range was quoted but cited Hodge's paper of 1979. NAS/NRC finally quoted the corrected Hodge's dosage rate for skeletal fluorosis (SF) (10-25 mg/day of fluoride for 10-20 years) in 1993. It was also stated by NAS/NRC that "it is no longer feasible to estimate with reasonable accuracy the level of fluoride exposure simply on the basis of concentration in drinking water supply."[5].

Extrapolating from Roholm's original figures, it follows that for a 100 lb person, at less than 2.5 mg/day fluoride intake, stage 1 of SF can occur within 10 years. At this same dosage rate, stage 2 of SF can occur after 19 years and crippling skeletal fluorosis after 45 years. There is evidence that some people are ingesting at least 5 mg/day, in which case the stages of skeletal fluorosis can occur after 5, 10 and 23 years, respectively.

NAS has this year proposed to publish, for its sister organization the Institute of Medicine (IOM), "Dietary Reference Intakes", accepting Roholm's dosage range but relating these to early skeletal fluorosis. They then quoted: "... Advanced stages of skeletal fluorosis are associated with intakes of fluoride ranging from 20 to 80 mg/day for 10 or more years (Hodge and Smith 1977, WHO 1984)." The quotes may be accurate but the statements are false. NAS/IOM and other public health bodies must decide whether their task is to advance the health of the public or be apologists for the industries, which produce fluoride toxic wastes.

Hodge, NAS/NRC, and all other public health bodies, who quoted the incorrect dosage rates, were grossly negligent and, in some cases, deliberately misleading. They certainly misled by quoting the incorrect dosage rate for skeletal fluorosis, NOT crippling skeletal fluorosis.

Many reputations are at stake here, but the evidence is clear that the risks of fluoride far outweigh any minor benefit to teeth. Pride has to be swallowed and the precautionary principle applied. An increase in more severe skeletal fluorosis is due to erupt as older people enter the risk window. Water fluoridation should cease immediately and steps should be taken to reduce fluoride in food, drink, and dental products.

References

1 *Fluorine Intoxication. A Clinical-Hygienic Study.* H K Lewis, London 1937 pp 213-253.
2 *Review of Fluoride: Benefits and Risks.* US Public Health Service, 1991.
3 *Recommended Dietary Allowances.* 10th Edition. Food and Nutrition Board, National Academy of Sciences, 1990.
4 Hodge HC. The Safety of Fluoride Tablets or Drops. In: Johansen E, Taves DR, Olsen TO (Eds). *Continuing Evaluation of the Use of Fluorides.* American Association for the Advancement of Science, Selected Symposium 11. Westview Press. Boulder CO 1979.
5 *Health Effects of Ingested Fluoride.* Committee on Toxicology, National Research Council, National Academy of Sciences Press, Washington DC 1993.

[The above summary was compiled from information presented on the Internet (http://www.inter-view.net/home/sherrell/fluoride.htm) by Ms Darlene Sherrell, 793 South Lacey Lake Road Charlotte, MI 48813, USA. E-mail: sherrell@inter-view.net]

Information and discussion on fluoride can also be obtained from other Internet sites: *e.g.*
http://www.cadvision.com/fluoride/index.htm
http://www.sonic.net/~kryptox/fluoride.htm
http://www.trufax.org/menu/chem.html#fluorides

REJOINDER

I was fully aware that Dr Foulkes (*Fluoride 30* August pp 203-204) was referring to the vague symptoms, usually subjective and difficult to quantify, summarized as hypersensitivity or, less accurately, as allergy. This is a general phenomenon, as some people are hypersensitive to such diverse dietary items as alcohol, chocolate, tea, coffee, potatoes and tomatoes.[1] Apart from the symptoms produced by excessive coffee being probably due to caffeine, the active constituents are unknown.[1] I agree with Waldbott, cited by Spittle,[2] that hypersensitivity should be differentiated from the toxicity of the fluoride ion. I am puzzled by the quite numerous cases that Waldbott, Moolenburgh and others have found contrasting with the absence of any cases reported among the half million people on Tyneside who have been drinking fluoridated water for thirty years.

The difficulty about the statement of Fejerskov *et al*[3] on fluorosis, is that the lowest grades of fluorosis are considered by some to be an enhancement of the pearly appearance of the enamel. The results of the concentration as low as 0.04 mg/kilo of body weight are probably of this type and cannot be regarded as undesirable.

I cannot see why the difficulty in estimating accurately the acute toxic dose supports Dr Foulkes' contention about the non-existence of a minimum safe dose.

Next, Dr Foulkes suggests that paradoxical effects discussed by my sparring partner of forty years ago, Albert Schatz, may help my understanding of his belief about the minimum safe dose. Professor Schatz states confidently "the fact" (sic) "that low levels of fluoride exhibit paradoxical effects (as low-level radiation and low levels of many chemical compounds do" (*i.e.* low concentrations may have greater effects than higher concentrations) "makes it difficult to provide convincing evidence that fluoridation is safe" (*Fluoride 30* August p 200) but the evidence is unconvincing. When the SIDS rate, in areas with 0.1 ppm (curiously quoted as 0.1 g/m^3), varies from approximately 2.3 to 7.3/1000 live births can this wide range be represented by a single figure (5.3/1000 live births) on which the curve suggesting a paradoxical effect depends?[4] Also, this graph is based on the intake of fluoride from the water only[5] and ignores his own point about considering fluoride intake from all sources. Professor Schatz has, to use his own words, "disregarded the most basic tenet in forensic toxicology".[6] Professor Schatz seems to be using an unproven and disputed suggestion (that fluoride may be a factor in the cause of SIDS) to support another hypothetical idea (that fluoride shows paradoxical effects). I am unaware of paradoxical effects of the type Professor Schatz discusses in any other action of the fluoride ion but I can point out a different type of paradoxical action of fluoride: high doses are toxic but low doses have a qualitatively different and beneficial effect in reducing caries!

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References

1 Finn R, Cohen HN. "Food allergy": fact or fiction? *Lancet i* 426-428 1978.
2 Spittle B. Allergy and hypersensitivity to fluoride. *Fluoride 26* 267-273 1993.

3 Fejerskov O, Manji F, Baelum V. *Dental Fluorosis - a handbook for health workers*. Munksgaard, Copenhagen 1988 p 108.
4 Schatz A. Paradoxical effects: critique of a study. *Fluoride 30* 131-133 1997.
5 Mitchell EA, Thompson JMD, Borman DM. No association between fluoridation of water supplies and sudden infant death syndrome. *New Zealand Medical Journal 104* 500-501 1991. Abstracted in *Fluoride 30* 130 1997
6 Schatz A. Schatz rejoinder. *Fluoride 30* 201-202 1997.

FINAL WORDS

(Prof Jenkins and Dr Foulkes were each invited to submit a final summing up)

"What is truth? said jesting Pilate, and would not stay for an answer." Thus began the essay, *Of Truth*, by my favourite English essayist, Francis Bacon (1561-1626).[1] Bacon, using an example familiar to Christians, knew nothing of fluoridation; but he understood the issues exemplified by the discourse between Professor Jenkins and myself. Bacon knew that the fixing of a belief was a bondage "affecting freewill in thinking as well as acting". He understood that truth was found only through difficulty and labour and that its enjoyment is for the "sovereign good of human nature". He also understood that the truth of "civil business" is often made base by falsehood.

During my review of the literature on fluoridation, I have encountered many examples of "blind" belief. I have accused Professor Jenkins of this in previous correspondence. I am certain that both of us have discovered the arduous nature of obtaining data on which each of us has built our version of the truth. However, we are blind if we do not recognize the lies and deception offered to us by those whose "business" is dependent on the continuation: purveyors of the product used in the process; institutions and individuals whose reputations are at risk; and, those concerned about grants and legal suits.

While we struggle with these aspects of defining "truth", with our mountain of documents at our sides, we can, surely, agree to take a centre path and look at the ethical and moral issue of adding a known toxic chemical, a waste product of industry, to a medium that has to be used by all, without regard to age, sex, physical condition or known disease and without proper informed consent. And, this action for the avowed purpose of an alleged reduction in dental caries.

It should be obvious that on the "risk" side of fluoridation there are many "red flags" and legitimate cautions - even Professor Jenkins agrees that "more research" is required.

Neither of us is jesting. We must stay for an answer. While we must agree to disagree, for the moment, on the question as to whether fluoride is "an inessential nutrient" or "a contaminant", we must continue, in our separate ways to woo the truth and thereby win freedom from the bondage of belief that may obscure it.

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Reference

1 Bacon, F. *Essays*. J M Dent and Sons, London 1946 pp 3-5.

Dr Foulkes and I have not been able to convince each other about whether fluoride is a contaminant or an inessential food constituent with beneficial effects or whether a minimum safe dose does or does not exist. However, the exchange of views has been stimulating and has drawn attention to at least two other issues that need more research; the question of hypersensitivity to fluoride and Albert Schatz's belief that fluoride can act paradoxically.

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NATICK NEWS

The Fluoridation Study Committee of the town of Natick, Massachusetts, was a panel of impartial professionals charged with the task of answering the following question of the Board of Selectmen of the town:

"On the basis of the documentation provided to you by the proponents and opponents, do you believe that the potential side effects associated with fluoridating Natick's public water supply outweigh the potential benefits?"

The Study Committee conducted a thorough review of the scientific literature and presented its findings to the Board, along with the following conclusion and recommendation.

Conclusion

The Committee reached the firm conclusion that the risks of overexposure to fluoride far outweigh any current benefit of water fluoridation.

Recommendation

1. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the town of Natick NOT fluoridate the town water supply.

2. The Natick Fluoridation Study Committee unanimously and emphatically recommends that the Board of Selectmen take appropriate action to ensure that fluoridation of the town water supply does not take place.

COMMENT *ADDENDUM*

Burgstahler AW, Robinson MA. Fluoride in California wines and raisins. *Fluoride 30* (3) 142-146 1997

After publication of our report we learned that, owing to the apparent need to use cryolite in California vineyards because leaf-eating insects have become resistant to other pesticides, a new fluoride limit of 3 ppm has been agreed upon for California wines imported into certain countries.

AWB and MAR

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