THE HEALTH HAZARDS OF FLUORIDATION: A RE-EXAMINATION

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SUMMARY

This paper re-examines the evidence that low doses of fluoride — such as ingested daily from artificially fluoridated drinking water — can cause dental fluorosis, skeletal fluorosis and hypersensitivity reactions in some people.

***Fluoride dose**. When drinking water is fluoridated, so that it contains about 1 part per million (ppm) fluoride, high fluoride doses (compared with average doses) are absorbed by formula-fed babies, children who drink water-based beverages, outdoor workers, long-distance runners, people with diabetes insipidus and people with malfunctioning kidneys, amongst others. It is such high-risk groups which require protection against environmental chemicals such as fluoride, rather than just the 'average person'. Specifically, about 1% of adults ingest 5.5 to 7.0 mg of fluoride per day (without tea), and heavy tea-drinkers consume an additional 1.0 to 7.5 mg per day. The doses, in mg per day, received from fluoridated water by formula-fed infants are 4 to 6 times the doses from fluoride supplements currently recommended by American Academy of Pediatrics and the Australian National Health and Medical Research Council for infants in unfluoridated areas. Formula-fed infants ingest 100 times the natural fluoride dose ingested by breastfed infants.*

***Dental fluorosis**, a specific type of dental mottling, is the only fluoride-induced disease for which dose guidelines are available. Dental fluorosis of grades 1 and 2 is found in some children exposed to 0.1 mg fluoride per kg body mass per day (and even less) during the period of enamel formation. Doses 20% to 80% higher than this are ingested by formula-fed infants in fluoridated areas, so it is not surprising that the prevalence and severity of dental fluorosis are increasing in fluoridated areas. Since dental fluorosis is caused by damage to the enamel-forming cells, it is a sign of physiological damage rather than a purely cosmetic effect.*

***Skeletal fluorosis**, a disease of the bones and ligaments which is primarily a hazard to older people, has been reported in at least 9 published papers to occur in communities in 5 countries where natural fluoride concentrations in drinking*

water range from 0.7 ppm to 2.5 ppm. In several of these reports, severe clinical symptoms have been observed at these low fluoride concentrations. Although most cases of skeletal fluorosis have been reported from tropical countries, there is no basis for assuming that the patients were necessarily malnourished, or had a high fluoride intake from food, or had an enormous water intake. Indeed, there is explicit evidence that the first two of these factors were not significant in several locations. The scarcity of reports of skeletal fluorosis from temperate countries should not be grounds for complacency, because, in temperate countries:

- *Few doctors are trained to recognise skeletal fluorosis; early stages of the disease can be mistaken for arthritis.*
- *The few studies of the prevalence of the disease which have been carried out are inadequate in design.*
- *High-risk groups, such as people with malfunctioning kidneys and people with high water intake, have not been given proper consideration.*
- *In most artificially fluoridated communities, there has not yet been enough time for the development of many cases.*
- *In most patients treated with 9-27 mg fluoride per day for osteoporosis, the period of treatment has been too short for the development of skeletal fluorosis.*

***Hypersensitivity or intolerance reactions** are well-known in patients receiving as little as 9 mg fluoride per day from osteoporosis therapy. Since some people who are not osteoporosis patients also have a total fluoride intake of over 9 mg per day in fluoridated areas, some of them are likely to be experiencing reactions to fluoridated water. Several physicians and dental researchers have reported adverse reactions to 1 ppm fluoridated drinking water and to 1 mg per day fluoride supplements. Some of these observations are based on blind and double-blind challenges. But there has been no study capable of determining the prevalence of these reactions in fluoridated versus unfluoridated communities. Rebuttals of reports of adverse reactions to fluoride are not usually based on scientific research, but rather on executive statements and endorsements of fluoridation by certain 'authorities', and on suppression of the physicians and scientists reporting the reactions.*

INTRODUCTION

This paper re-examines the evidence that low doses of fluoride — such as ingested daily from artificially fluoridated drinking water — can cause three different diseases in people: dental fluorosis, skeletal fluorosis and hypersensitivity reactions. It does not attempt to review the issues of whether fluoride is mutagenic or carcinogenic, which would require a separate dedicated review paper.

Most previous assessments and reviews of the 'safety' (i.e. health hazards) of water fluoridation, which have been written from a pro-fluoridation perspective, have only considered the health implications for the 'average person'[1,2]. Indeed, this is a natural consequence of focusing on fluoride concentrations in drinking water rather than on the range of daily doses actually ingested by people.

However, the proper approach to risk assessment in toxicology and radiation protection is to identify the high-risk groups in the community and to set safety standards for daily doses with sufficient margin to protect them with a high degree of certainty. If only the risks to the 'average' member of the community are known, a "safety factor" of 10 is normally used to allow for high-risk groups. Any other major uncertainties are compensated for by increasing the "safety factor"[3].

Such an approach has not been followed for water fluoridation, which was introduced long before the growth in awareness of environmental chemical and physical health hazards of the 1970s. In this paper, an early step is taken towards reform, by estimating the fluoride intake of some of the high-risk groups and by reviewing the published scientific literature which shows that there can be no safety margin for members of some of these groups if they consume water fluoridated at 1 ppm (1 mg fluoride per litre water). The most obvious high-risk groups are those which consume large daily doses of tapwater.

FLUORIDE DOSE

In a fluoridated area, the main contributions to the fluoride dose, in the most common order of importance, are:

i. fluoride in tapwater used in the home (and also in the school, the place of employment and the restaurant) to prepare drinks, soups, home-made beer, ice, stews, cooked vegetables, etc.;

ii. fluoride added in the course of the commercial processing of food and beverages in fluoridated areas;

iii. fluoride which is already present naturally in the 'raw' primary produce (e.g. fruit, vegetables, grains, meat, fish, tea-leaves) before it is processed. The dose from this contribution is the same in fluoridated and unfluoridated areas, and is usually quite small (0.3-0.8 mg/day), except for people who eat large quantities of certain fish products or drink many cups of tea daily[4].

iv. fluoride in toothpaste and other regularly applied topical fluorides which are inadvertently ingested and/or absorbed through the buccal mucosa;

v. use of fluoride-containing nutritional supplements (e.g. vitamin pills), which are sometimes taken, contrary to official guidelines, in fluoridated areas.

Air pollution normally makes a very small contribution to the fluoride intake by members of the public, but for some workers exposed occupationally to fluoride (e.g. in aluminium smelting, fertiliser manufacture, steelworks and operating theatres) it can be the largest or second largest contribution[4].

The fluoride intake from items (i) and (ii) in fluoridated areas can be estimated from the recent survey of 'total water' and 'tapwater' intake by 26,000 people in the USA[5]. 'Total water', as defined in the survey, is the sum of 'tapwater' and 'intrinsic water'. 'Tapwater' corresponds to item (i) above. 'Intrinsic water' is water contained in foods and beverages at the time of purchase and so it contains both the 'biological water' of raw foods [i.e. see item (iii) above], which is usually very low in fluoride content, and any 'commercial water' added during manufacturing or processing [i.e. item (ii) above].

In the consumption of commercially processed food and beverages, there are two main cases:

(a) The person surveyed lives in a fluoridated community, but purchases food and beverages which have been mostly processed in unfluoridated areas. Then 'tapwater' plus item (iii) provide a good estimate of fluoride intake from food and beverages.

(b) The person surveyed lives in a fluoridated community, but purchases food and beverages which have been mostly processed in fluoridated areas. Then 'total water' plus item (iii) provide a good estimate of fluoride intake from food and beverages.

Both methods used in this survey, keeping a diary of food and drink consumed or recalling the intake over the previous 24 hours, tend to underestimate the varieties and quantities of foods and beverages consumed and hence underestimate the intake of 'total water' and 'tapwater'[5].

In the US survey[5] the average daily intake of all participants (excluding pregnant women, lactating women and breastfed children) was 2.07 ± 0.80 litres of 'total water', including 1.19 ± 0.70 litres of 'tapwater'[5]. The large values of the standard deviations demonstrate the importance of determining the high-consumption groups rather than simply focusing on the averages. The percentile distribution shows that 5% of participants had a daily 'total water' intake greater than 3.55 litres, and 5% had a 'total water' intake less than 1.01 litres. The corresponding values for 'tapwater' are 2.48 litres and 0.29 litres. These wide ranges show the difficulties of trying to medicate a whole community safely and effectively through the water supply.

For estimating the risk of diseases such as skeletal fluorosis it may be more appropriate to consider the top 1% of water drinkers. This group has a daily 'total water' intake greater than 4.65 litres. For adult males aged 20-64 years, the average intake is 2.5 litres, but 1% consume 5.5 litres or more of 'total water'.

For water fluoridated at 1 ppm, 1 litre contains 1 mg of fluoride. Natural fluoride in raw food [case (iii) above] could add daily another 0.3-0.8 mg[4]. Ingestion of fluoridated toothpaste could contribute 0.25 mg of fluoride on average, but up to 2 mg in some cases[6]. Among tea-drinking people, such as in most of the UK, Australia and New Zealand, there is an additional daily contribution of several mg. Even when made with unfluoridated water, the fluoride content of various teas ranges from 0.4 to 2.8 ppm, depending on the type of tea, quantity of tea-leaves per cup and length of time brewed[4]. For members of the top 1% of 'total water' consumers who are heavy tea-drinkers, possibly 80% of 'tapwater' consumed (about 2.7 litres or more) could be in the form of tea.

In summary, based on the comprehensive USA survey[5], it is likely that about 1% of the adult population in a fluoridated area ingest daily about 6-7.5 mg fluoride, excluding tea and fish. Heavy tea drinkers may consume a daily total of 7-15 mg fluoride. These are not lifetime averages, but serve to identify a high-risk group. Our results are similar to, but somewhat higher than, the earlier estimates of the Royal College of Physicians, which gave 'maximum' daily intakes of 6 mg (excluding tea) and 12.2 mg (with tea)[2]. In the light of our estimates, some of the very low traditional estimates of fluoride consumption (e.g. three estimates with 'maximum' daily intakes less than 3 mg included in Table 4 of the WHO report[4]) can be discarded as being unrealistic.

Children

For children aged 1-10 years, the mean daily intake of 'total water' is 1.56 litres, with the top 5% getting over 2.51 litres, the bottom 5% getting less than 0.84 litres and the top 1% getting over 3.01 litres. The corresponding values for 'tapwater' are: mean = 0.74 litres; top 5% over 1.52 litres; bottom 5% less than 0.19 litres; top 1% over 1.95 litres[5].

Expressed in terms of litres of 'total water' or 'tapwater' ingested per kg of body mass per day, the daily intake decreases with increasing age from age 1 year to adulthood, as shown in Table 1. In fluoridated areas, 1% of children aged 1-3 years consume daily over 0.14 mg fluoride per kg body mass from 'tapwater' alone. Of children aged 1-3 years who drink water-based beverages rather than fresh whole cow's milk (which contains less than 0.1 ppm fluoride[8]), about 1% consume daily over 0.2 mg per kg. For a 2-year-old with a mass of 12 kg, this corresponds to a daily dose of 2.4 mg fluoride. So, some young children and, as discussed below, formula-fed infants are high-risk groups for the health hazards of fluoride.

It is of interest to compare these values with the claim that the 'optimal' daily systemic fluoride dose to prevent caries in childhood is 0.05-0.07 mg per kg body mass[7]. The bottom row of Table 1 gives the water intakes for the broad age-group 1-10 years, which has been traditionally used for the calculation of 'optimal' fluoride doses. For children who do not drink much whole cow's milk and who live in a fluoridated area which obtains most of its manufactured beverages and foods from fluoridated areas, the average daily fluoride intake is about 0.075 mg per kg, which is greater than the 'optimal' dose, and about 5% of children ingest over double the 'optimal' dose. To these doses must be added that from ingested fluoride toothpaste.

Of course, it is a dubious procedure to average over children of ages 1-10 years, because the water consumption (and to some extent the body mass) each varies widely in a non-linear manner with increasing age. But the 1-3 year age-group in Table 1 has even higher fluoride doses than the 1-10 year group.

Infants

Infants (children younger than 1 year of age) are not covered adequately by the US survey[5] which has not recorded the intake of breastmilk. It is necessary to consider separately two extreme groups, breastfed and formula-fed infants. Breastmilk contains only about 0.01 ppm fluoride, whatever quantity of fluoride is ingested by the mother[9,10].

In other words, infants who are bottle-fed on powdered formula reconstituted with fluoridated water ingest 100 times the dose of fluoride ingested by breastfed babies.

Paediatricians recognise that babies in the first 6 months after birth ingest daily on average about 150 ml liquid per kg body mass, with a range of variation of about 120-180 ml per kg[11,12]. If the liquid is fluoridated water, this corresponds to 0.12-0.18 mg fluoride per kg. For a 6-month old infant of mass 8 kg, this yields a daily fluoride dose range of 0.96-1.44 mg, which is 4-6 times the daily doses currently recommended by American paediatricians for fluoride supplements in unfluoridated areas (0.25 mg for children aged 0-2 years[7]). Through the second 6 months after birth, babies' average fluid intake, per kg of body mass, decreases to about 120 ml per kg body mass, but this is compensated for to some extent by fluoride in supplementary solids. So, formula-fed infants in fluoridated areas are likely to have high fluoride intakes over the whole of the first year of life.

In comparison, earlier estimates[13,14] yielded much lower values of fluoride intake by infants. In obtaining their results Singer and Orphaug[13] underestimated the liquid intake of infants and only considered 'ready-to-feed' milk formula with flouride concentration $[F^-]$ = 0.68 ppm. Ophaug, Singer and Harland[14] aggregated together babies who drink fluoridated formula with those who drink cow's milk and with those who drink breastmilk, thus obtaining estimates of average values, which are useless for identifying the high consumption groups.

The most obvious health impact of the enormous unnatural fluoride doses ingested by formula-fed infants is the development of dental fluorosis in children. Also, because the uptake of fluoride by bones may be higher in infants and young children than in adults[15], the exposure of this group to fluoridated drinking water is a contributor to skeletal fluorosis in older people. But the impact of these doses on the developing immune system is unknown, although there are good reasons for examining this question further[16].

Table 1: 'Total water' and 'tapwater' intake* by age-group[5]

	'Total water'			'Tapwater'		
Age (yr)	Mean	Top 5%	Top 1%	Mean	Top 5%	Top 1%
1-3	100	>165	>209	47	>102	>141
4-6	78	>128	>164	38	> 81	>103
7-10	59	> 97	>121	27	> 55	> 71
11-14	42	> 72	> 89	20	> 42	> 55
15-19	34	> 56	> 71	16	> 35	> 46
20-44	32	> 55	> 71	19	> 38	> 53
45-64	34	> 57	> 73	22	> 42	> 58
1-10	75	>136	>179	36	> 79	>114

*In gm water/kg body mass/day. If all the water is fluoridated at 1ppm, these are the same as μg fluoride/kg body mass/day.

DENTAL FLUOROSIS

Dental fluorosis is a particular type of mottling of teeth which is caused only by the ingestion of fluoride during early childhood, while the teeth are still mineralising, before they erupt into the mouth. Dental fluorosis can usually be distinguished from other types of dental mottling by the characteristics that it is diffuse and has bilateral symmetry in the mouth. In its milder forms, which are ranked as grades 1 and 2 on Dean's classification index, dental fluorosis comprises opaque white patches which cover less than 50% of the enamel surface. The more severe grades, 3 and 4, can involve dark brown staining and pitting of the fluorosed enamel. Both the prevalence and severity of dental fluorosis increase with increasing fluoride concentration in drinking water or increasing doses from fluoride supplements[17].

Dental fluorosis is the only adverse effect of low levels of fluoride for which dose guidelines are available. The most accurate ones are based on studies of children living in unfluoridated areas (where the fluoride concentration in drinking water was less than about 0.3 ppm), who ingested controlled doses of fluoride by taking supplements regularly. On the basis of these studies, it is stated that a substantial proportion of children suffer dental fluorosis of grades 1 and 2 (or the equivalent on other scales) when doses exceed 0.1 mg per kg per day[7,18]. Actually, dental fluorosis occurs in some children who receive less than this dosage[17,19]. Incidentally, it is claimed that the 'optimal' dosage to reduce subsequent tooth decay is 0.05 to 0.07 mg fluoride per kg body mass per day[7], but, as pointed out by Leverett[20], there is little direct evidence to support this claim. Even if it were correct, the safety margin between 0.1 and 0.07 mg per kg per day is very small. Indeed, the wide variation in dose from drinking fluoridated water makes it inevitable that a significant proportion of children in fluoridated areas will have dental fluorosis. It has been pointed out in the previous section that a high-risk group comprises infants who are fed on powdered formula reconstituted with fluoridated water. In fluoridated areas, these babies ingest 20-80% above the dose which is recognised officially as causing noticeable dental fluorosis in a community. So it is not surprising that dental fluorosis is prevalent in fluoridated areas. Moreover, as the deciduous teeth erupt, some infants are exposed to another significant fluoride source, fluoride toothpaste, much of which is swallowed by pre-school children[21], and can therefore be a contributor to dental fluorosis in permanent teeth[6]. For this reason, the Swedish Department of Health recommends that children aged less than 4 years should use unfluoridated toothpaste.

In the 1930s, in naturally fluoridated areas where [F^-] was about 1 ppm, Dean and coworkers observed dental fluorosis of grades 1 and 2 in about 10% of children, but no dental fluorosis of greater severity. However, there is recent evidence that the prevalence and severity of dental fluorosis have increased in both fluoridated and unfluoridated areas. In several recent American studies, the prevalence of all grades of dental fluorosis ranged from 26% to 51% in fluoridated areas, while the more "objectionable" grades 3 and 4 affected 1% to 2% of children[20,22,23]. For example, in 1981-82, Leverett found that the prevalence of dental fluorosis in fluoridated parts of New York State was 27%, compared with 4.4% in areas with 0.3 ppm fluoride or less[20]. In addition, 1% of children in the fluoridated area suffered from the more severe dental fluorosis of grade 3. In the naturally fluoridated town of Kewanee, Illinois, the overall prevalence of dental fluorosis in 1980 was 14.6%, compared with 12.2% four decades earlier[24]. But, the severity of dental fluorosis in Kewanee, as measured by the prevalence of grades 3 and 4, had risen from zero in 1940 to 2.4% in 1980[24]. More recently the prevalence of dental fluorosis in Kewanee has risen to 29%[25].

In New Zealand, 25% of children in fluoridated areas of Auckland have dental fluorosis[26-28] and 3.6% have discoloured or pitted enamel[26,27]. In fluoridated Hastings, the prevalence of dental fluorosis is 23%, but the percentage with 'objectionable' forms has not been reported[29].

Some increase in dental fluorosis is seen in unfluoridated areas, where it is associated with the use of fluoride supplements[18,30]. Generally the prevalence in unfluoridated areas is still much lower than in fluoridated areas. The main problem is in fluoridated areas, where the increase has been attributed primarily to increased fluoride in the food chain as the result of the use of fluoridated water for food processing[31].

Dental fluorosis is generally believed to be a result of disturbance or damage to the ameloblasts, the cells which are responsible for the development of the dental enamel. As such, it is an indicator of physiological damage or disease[17] rather than simply a "cosmetic effect".

SKELETAL FLUOROSIS

When fluoride is ingested, about 50% is excreted by the kidneys (provided that they are functioning properly) and almost all of the remainder is stored in the bones[4]. In infants and young children, there is evidence that considerably more than 50% is stored in the bones[15].

Skeletal fluorosis is a disease involving changes in the structure of bones and calcification of ligaments, which results from the chronic intake of fluoride over a period of years or decades. Early clinical stages resemble arthritis, with patients experiencing pain and stiffness in bones and joints. Restriction of movement occurs in the spine, mainly in the cervical region, but also in lower back, shoulder joint, hip and knee. Radiological symptoms may include osteosclerosis (thickening of the bones), periosteal bone formation (especially in the forearm) and calcification of the interosseous membrane. The advanced stages seen in some countries can involve rigidity of the spine, deformities such as kyphosis and exostosis, wasting of the muscles, and compression of nerves leading to paraplegia and quadriplegia in some cases.

Conventional wisdom

The conventional wisdom on skeletal fluorosis, in reports which have a pro-fluoridation perspective, is that there are no osteosclerotic changes in "temperate countries" when the fluoride concentration in drinking water, [F^-], is below 4 ppm[32], and that there are no "clinically significant" changes, except for dental fluorosis, when [F^- is below 8 ppm[33].

It should be noted that fluoride doses are not usually specified in the conventional wisdom, only concentrations. However, the US Environment Protection Agency and other regulatory agencies have stated that crippling skeletal fluorosis results from intakes of fluoride of 20 mg/day (i.e. 0.29 mg/kg/day for a typical 70kg adult) over periods of 20 years or more. But these agencies do not justify this figure, or give a figure for the dose causing crippling skeletal fluorosis over a *lifetime* of fluoride ingestion, or give a figure for skeletal fluorosis which produces clinical symptoms but is not crippling.

Contrary evidence

Clinically significant skeletal fluorosis has been reported where $[F^-]$ is below 4 ppm (mostly below 2.5 ppm) in at least 9 separate publications from 5 countries, including the USA, where there are naturally fluoridated areas:

India

In an extensive survey of skeletal fluorosis in 10 villages in Punjab, Jolly et al[34] reported that the main factors determining skeletal fluorosis were $[F^-]$ in drinking water and duration of exposure. Physical stress and other chemical constituents of the water were also factors; malnutrition was not. Fluoride intake from food was not significant in quantity. One village had an average $[F^-]$ of only 0.7 ppm, with a range 0.4-1.4 ppm. (N.B. 0.7 ppm is approximately equal to the so-called 'optimal' concentration of fluoride for that climate.) Yet 30% of 289 adults there showed clinical 'skeletal manifestations', such as restriction of movement of the spine, shoulder, hip and knee. But, from a subset of 165 adults x-rayed, only 6 people (3.6%) had radiological evidence of skeletal fluorosis.

In an earlier survey in Punjab, Singh, Jolly and Bansal[35] observed neurological complications (eg compression paraplegia) of skeletal fluorosis where $[F^-]$ =1.2 ppm and 1.3 ppm, and several severe non-neurological cases at 1.4-2.2 ppm. The people had a "moderately good diet".

In Andra Pradesh, Siddiqui[36] observed quadriplegia, as a complication of skeletal fluorosis, where $[F^-]$ =1.35 ppm and above.

In Uttar Pradesh, Misra et al[37] observed spinal cord compression, as a complication of skeletal fluorosis, where $[F^-]$ =2.4 ppm and above.

Sahara

Pinet & Pinet[38] observed osteosclerosis and calcification of ligaments where $[F^-]$ =1.5 ppm. In the range 1.5-4.0 ppm, there were 148 cases, including one paraplegia.

Qatar

Azar et al[39] observed osteosclerosis in 8 patients, referred for TB, who had been exposed to $[F^-]$ =0.8-1.1 ppm (piped water) or 0.8-3.5 ppm (well water). But the authors did not identify which patients drank from which type of water supply. There were apparently no clinical symptoms, except that most patients had marked dental fluorosis.

USA

Sauerbrunn et al[40] reported one case of severe osteosclerosis which was associated with polydipsia (excessive thirst) in a patient who had for many years consumed drinking water with $[F^-]$ =2.4-3.5 ppm. A post-mortem revealed a high fluoride content of the patient's bones. There was also a history of clinical symptoms: the patient had in 1950 complained of pain and cramping in the left leg and of weakness and stiffness of all limbs for several years; in 1954 spastic quadraparesis, wasting of muscles, etc were reported. Patient died in 1962 with severe respiratory distress and 'pneumonia'.

Juncos & Donadio[41] reported two cases of severe osteosclerosis with associated kidney damage and possible polydipsia, where $[F^-]$ = 1.7 ppm and 2.6 ppm. Both patients had dental fluorosis as well.

Japan

Hirata[42] reported skeletal changes and, in children before puberty, blood abnormalities, where $[F^-]$ was in the range 1-5 ppm.

Since most western medical schools do not teach their students how to recognise skeletal fluorosis, these examples could be just the tip of the iceberg.

Critique of evidence for the conventional wisdom

The principal reference which is purported to justify the conventional wisdom that skeletal fluorosis could not be a problem in fluoridated areas of western countries, is the paper by Stevenson and Watson (hereafter SW)[32]. In several pro-fluoridation reports the impression is given that the authors of this paper, two radiologists with some experience in the field of skeletal fluorosis, reviewed 170,000 x-rays of the spine and pelvis of patients living in Texas and Oklahoma. They could find only 23 cases of fluoride osteosclerosis and all of these cases, the reviews claim, came from areas where the drinking water contained at least 4 ppm fluoride.

However, a close reading of the original paper suggests a completely different interpretation of what was actually done. The authors state that: "...we reviewed the medical records on file at the Scott and White Clinic for the eleven year period from 1943 through 1953. A roentgenologic diagnosis of fluoride osteosclerosis was recorded on 23 patients' records."[32] So, SW may not have actually examined the 170,000 x-rays themselves. Instead, it appears that they only reviewed the 23 records for which unidentified physicians had previously recorded fluoride osteosclerosis. Since most medical schools in the USA do not teach the identification of skeletal fluorosis, one might ask whether any or all of the physicians had enough experience to identify more than the most obvious cases of fluoride osteosclerosis.

The second difficulty is that SW's knowledge of skeletal fluorosis was apparently based on Roholm's reports on cryolite workers and Bishops's reports on phosphate workers (see refs 1 and 12 of SW). In these cases the workers received very large daily doses of fluoride by inhalation. SW did not seem to be aware of the early observations on endemic fluorosis from drinking water in India, which involved much lower daily doses and had somewhat different characteristics from those of industrial fluorosis. Even if SW had re-examined all 170,000 x-rays themselves, they would not have been using the most appropriate diagnostic criteria. In addition, they may not have been aware of the non-radiological 'skeletal manifestations' identified later by Jolly and co-workers[34] in India.

Consider the following warnings on the difficulties of diagnosis by experts in skeletal fluorosis from two different generations:

> "Dental fluorosis is easily recognised, but the skeletal abnormalities are not so obvious until the advanced stage of crippling fluorosis. However, radiological changes are discernable in the skeleton at a much earlier age, and provide the only means of diagnosing the early and relatively asymptomatic stage of fluorosis. These cases are young adults whose only complaints are vague pains noted most frequently in the small joints of hands and feet, the knee joints and those of the spine. *Such cases are frequent in the endemic area and may be misdiagnosed as rheumatoid or osteoarthritis. Such symptoms may be present prior to the development of definite radiological signs.*"[43] (emphasis added)

> "The difficulties in diagnosing skeletal fluorosis result from the questionable sensitivity of the x-ray techniques and from the non-specificity of the associated symptoms."[44]

> "Irregular bone was laid down along the attachment of muscles and tendons in the extremities as well as in joint capsules and interosseous membranes. The latter is particular helpful in *border-line cases where the density of the bones is not markedly increased*."[44] (emphasis added)

So, it is hardly surprising that SW concluded, amongst other things, that "In each case, adequate clinical examination failed to establish any relationship between the roentgenologic findings and the clinical diagnosis of the patient's condition." But, Singh and Jolly[34,35,43] in the 1960s and Czerwinski et al[44] in the 1980s are likely to have reached a different conclusion.

Thirdly, for 8 of the 23 identified cases of fluoride osteosclerosis in the SW paper, the fluoride concentration was actually unknown (see the notes under Table 1 of SW), and so it is misleading for reviewers to state that all 23 cases had fluoride concentration in drinking water of 4 ppm or more. Furthermore, in not one of the 23 patients was there a biopsy to determine the fluoride content of their bone.

In short, the study by Stevenson and Watson was conducted too early, by the wrong people, who had used incorrect criteria to identify endemic skeletal fluorosis, and did not have information on the fluoride concentrations or the doses ingested by one-third of the cases identified. There might still be some value in asking experts on endemic skeletal fluorosis (such as Susheela and co-workers at the All-India Institute of Medical Sciences, New Delhi) to re-examine the 170,000 x-rays, if the plates still exist. But even better would be a new survey, conducted by experts on skeletal fluorosis, on living people who could discuss their symptoms as well as receive radiological examinations.

The remaining studies which are occasionally quoted in support of the conventional wisdom in pro-fluoridation reviews can be dismissed on the grounds of small sample size, inconsistencies in results and exclusion of important data. In particular, Leone, Stevenson et al[33] studied "health", i.e. gross physical defects, in 116 subjects from Bartlett, Texas, with $[F^-]$ = 8 ppm. But, in this study even from comparison group, 121 subjects from Cameron, Texas, with $[F^-]$ of 0.4 ppm, there were 4 cases of osteosclerosis and 2 of coarsened trabeculation. There was no overlap in the sample with SW's group. A different account of the same study[45] listed only one bone change in Cameron.

A study by Zipkin et al[46] and Geever et al [47] of the pathology and biochemistry of bones of corpses in areas of USA with 1-4 ppm fluoride in drinking water considered the following tiny samples: 5 subjects had ingested 1 ppm for 12 years only; 27 subjects had 2.5 or 2.8 ppm; 4 subjects had about 4 ppm. People with chronic illness, including kidney disease, were excluded from the study. Since one of the main high-risk groups for skeletal fluorosis comprises people with chronic kidney disease, this was a worthless study scientifically.

How pro-fluoridation reports treat the evidence

The Tasmanian Royal Commission[48] seemed to attach significance to the notion that most reports of skeletal fluorosis were from what it called "native" populations. Furthermore, it considered only a small part of the available evidence summarised above and questioned whether the low observed fluoride concentrations had been correctly measured. But the present compilation of 9 separate reports of skeletal fluorosis, mostly with clinical manifestations, from 5 countries where fluoride concentrations were often below 2.5 ppm and always below 4 ppm, offers ample confirmation of the reality of the reported low fluoride concentrations.

The British Royal College of Physicians[2] attempted to diminish the evidence of skeletal fluorosis at low fluoride concentrations in drinking water by stating that it was mostly observed in "tropical" countries. But the absence of well designed studies to determine the prevalence of skeletal fluorosis in temperate countries, the absence of physicians trained to recognise skeletal fluorosis in temperate countries, and the wide range of tapwater intake in a population do not give grounds for confidence that skeletal fluorosis is somehow confined to tropical countries. It should be noted that the average consumption of tapwater in adults aged 20-64 in USA varies by about 23% between north and south and only by about 7% between summer and winter[5]. This suggests that the consumption of water in tropical countries may have been over-stated too. Furthermore, almost all the victims of skeletal fluorosis in tropical countries, reported above, would have been breastfed as infants, unlike the potential victims in temperate countries.

The Royal College of Physicians[2] also attempted to rationalise the observations in India at low values of $[F^-]$ by postulating the existence of additional large sources of fluoride intake in the diet. But there is no direct evidence to support this, and indeed in the extensive survey by Jolly et al[34] the fluoride content of food was shown to be insignificant.

The section on skeletal fluorosis in the Australian National Health and Medical Research Council's (NH&MRC) 1985 report on fluorides is devoted almost entirely to fluoride therapy for osteoporosis, currently a medically controversial therapy. Without citing any epidemiological studies,it states that:

> "There is a very low incidence of occurrence of skeletal fluorosis in osteoporotic patients treated with fluoride."[49]

But the report does not take into account the short periods of time over which these patients have been treated. A single case of skeletal fluorosis in an elderly woman who received 60 mg NaF (27 mg fluoride) daily for 3 years is mentioned. Since fluoride accumulates in bones approximately linearly with time, this could be roughly equivalent to the ingestion of 3 mg daily for 27 years, a modest total fluoride dose for some people living in fluoridated areas. Therefore, the alleged rarity of skeletal fluorosis among osteoporosis patients, most of whom receive from 9-27 mg fluoride per day, is not grounds for complacency. In Section 1 it was shown that some adults in fluoridated areas, who are not undergoing fluoride therapy, could be consuming at least 9 mg/day fluoride over periods of several decades.

The NH&MRC report also stated that:

> "Furthermore the development of skeletal fluorosis is virtually confined to tropical climates with very high fluoride levels in water, and in addition probably requires other factors (e.g. malnutrition) to contribute to its development."[49]

This is misleading, considering that the examples given under the heading "Contrary Evidence" (above) do not have 'very high' values of [F^-], that there are no adequate surveys from temperate countries and that malnutrition was not an important factor in some of our examples: e.g. "Most of our patients had a moderately good diet, perhaps somewhat deficient in animal proteins."[35] Jolly et al[34] also found that malnutrition was not a significant factor.

The New York State Department of Health (1989) report[50] placed much emphasis on the USA data, which has been found to be inadequate, and paid little attention to the extensive overseas data from predominantly tropical countries. Even so, the report admits that skeletal fluorosis could be a problem in people with malfunctioning kidneys in naturally fluoridated areas with [F^-] greater than 2 ppm, and recommends further study on the effects of fluoride on people with malfunctioning kidneys. However, it could be argued that any substance which is harmful to a significant number of people at 2 ppm must also be harmful to some people at 1 ppm. A safety factor of 2 is too small[3].

Discussion of skeletal fluorosis

The conventional wisdom — that there are no osteosclerotic changes when the fluoride concentration in drinking water is below 4 ppm and no "clinically significant" changes below 8 ppm — is wrong. Skeletal fluorosis with clinical indications has been reported for over 25 years in the refereed medical literature from at least 4 countries (including USA) from places where the fluoride content of drinking water is less than 2.5 ppm.

The observations of skeletal fluorosis are not limited to so-called 'native' populations (whatever that means scientifically) or to tropical countries, although there are more observations from the tropics. Considering that doctors in extensively fluoridated temperate countries like the USA are taught the conventional wisdom, the published results in temperate regions could be just the tip of the iceberg. It is likely that early stages of skeletal fluorosis have been misdiagnosed as rheumatoid or osteoarthritis in these countries. The few studies from naturally fluoridated areas of the USA, which are normally cited to bolster the conventional wisdom, either used inexperienced examiners, or questionable methods of diagnosis or examined very small samples of people.

Most of the major fluoridated cities were only fluoridated in the 1960s and early 1970s. Since fluoride accumulates in the bones over people's lifetimes, we would expect the main body of cases of skeletal fluorosis from artifical fluoridation to appear after the next 15-40 years. But, it is not too early to seek cases in population groups which are at high risk.

For people forced to consume drinking water fluoridated at about 1 ppm in temperate countries, most currently observable cases of skeletal fluorosis from fluoridated water are likely to be found amongst those with kidney malfunction and/or high water intake. Kidney disease is fairly common in the developed temperate countries. Its presence can more than compensate for reduced average water consumption in temperate climates. Analgesic abuse tends to affect both kidneys. I can find no scientific study determining the prevalence of skeletal fluorosis in people with malfunctioning kidneys.

What is the prevalence in people of average mass who live in fluoridated areas and throughout their lives are in the top 1% of drinkers of water? They may be at risk of skeletal fluorosis because as infants they were formula-fed on fluoridated water, as children they drank water-based beverages rather than fresh cow's milk, and as adults they had strenuous outdoor jobs or were long-distance runners. We cannot ignore infants who ingest about 0.18 mg fluoride/kg body mass/day (1.44 mg/day for 8 kg infants at age 6-months), then as children of age 1-10 years of average mass 21 kg ingest on average at least 0.11 mg/kg/day (about 2.3 mg/day), and then as adults of average mass 70 kg ingest at least 0.086 mg/kg/day (6 mg/day). We must also consider the prevalence of skeletal fluorosis in people who are consistently below average mass. In the pro-fluoridation literature, there is a tendency to regard people who are at high risk of adverse effects from fluoridation as abnormal and somehow unworthy of protection by the appropriate safety factors which are applied by toxicologists as a matter of course to other chemicals.

There have been no properly conducted scientific studies to investigate the prevalence of skeletal fluorosis within the high-risk groups — only reassuring anecdotes from medical researchers and dentists with vested interests in the use of fluoride for therapy or as a preventive medicine.

HYPERSENSITIVITY

This section deals with those reactions to mg per day doses of ingested fluoride which are described as hypersensitivity, intolerance, allergy, or nonskeletal chronic fluoride intoxication.

It is generally accepted that some patients with osteoporosis, who ingest as little as 9 mg fluoride per day in the course of the controversial fluoride treatment, experience nausea, epigastric discomfort and, in some cases, vomiting after taking their tablets[2]. At least part of the gastric reactions appears to be the result of the irritant action of hydrogen fluoride[51]. (This statement is based on an experiment on 12 volunteers who had a single ingestion of 20mg fluoride: all 12 suffered damage to the gastric mucosa.[51])Some people who drink 1 ppm fluoridated water have total daily fluoride doses above 9 mg per day. Hence, some of these people are also likely to be suffering similar reactions to those of the osteoporosis patients. It is possible that the reactions to 9 mg fluoride per day taken at a single amount each day are more acute than when the same dose is spread out over 14-18 hours in drinking water, but this has not yet been established empirically.

Clinical reports of hypersensitivity to 1 ppm fluoride in drinking water or 1 mg per day in fluoride tablets have been published by Waldbott[52-56], Feltman[57,58], Shea[59], Grimbergen[60] and Petraborg[61,62]. These reactions include some of the following symptoms: gastrointestinal upsets (as described in the previous paragraph), skin rashes (also after bathing in fluoridated water), mouth sores (also from fluoridated toothpaste), migraine-like headaches, arthritic-like pains, dryness of the throat, excessive water consumption, frequent need to urinate, chronic fatigue, depression, nervousness and respiratory difficulties. These symptoms subside after discontinuation of the exposure to fluoride. Of course, such symptoms can also have other causes, but in several cases the association with fluoride has been confirmed by blind (e.g. refs.[55,56,58,59]) and double-blind[54,60] challenges. There have not been any studies designed specifically to determine the prevalence of hypersensitivity to fluoridated drinking water in the general community, but Feltman and Kosel's blind study of the effect of 1 mg per day fluoride in tablets on pregnant women and children in an unfluoridated area suggests that about 1% of the population may be hypersensitive[58].

The standard response of proponents of fluoridation and pro-fluoridation reports[2,4,49] has been to quote a statement by the Executive Board of the American Academy of Allergy that:

> "There is no evidence of allergy or intolerance to fluorides as used in the fluoridation of communal water supplies."

Waldbott, a Fellow of that Academy, has responded that:

> "Curiously, none of these prominent scientists had carried out research on the health effects of fluoride; no hearings were held on the subject and no inquiries were made of the members of the Academy regarding fluoride poisoning among their patients..... The bibliography did not include any of my original publications on chronic fluoride poisoning..."[56]

Clearly the statement by the Executive Board cannot be regarded as a scientific refutation of the published reports of hypersensitivity. To do this, rigorous double-blind studies would have to be conducted on large populations to determine the prevalence of fluoride-induced hypersensitivity.

Waldbott also pointed out that the Academy's statement was apparently made at the request of the pro-fluoridation US Public Health Service (USPHS) and that most of the members of the Executive Board of the Academy had received grants from the USPHS. Four members of the Executive Board actually received grants around the time that their statement was published[56]. So, a situation of conflict of interest could explain why the Executive Board's statement was issued.

Another response to reports of fluoride hypersensitivity has been to take the position that the reports should be ignored until those making them can produce theoretical mechanisms to explain their observations[4,49]. This position appears to be inconsistent with modern scientific method which places empirical observation before theories.

Yet another kind of response by proponents of fluoridation comprises attempts to misrepresent the evidence and to suppress the scientists and physicians who make these reports[63,65]. Unfortunately the Tasmanian report[48] seems to place credence on the personal and professional attacks on Waldbott, who was an allergist of international stature. This professional intimidation helps to explain why there have not been more published reports of hypersensitivity. But unscientific responses by proponents of fluoridation are inadequate to refute the existing clinical evidence of hypersensitivity.

The New York State Department of Health report admits that "some individuals may react idiosyncratically (*sic*) to fluoride; however, the prevalence of these reactions among the population is unknown. Further studies on possible hypersensitivity reactions to fluoride and the prevalence of these reactions among the general population are required."[50]

CONCLUSION

The fluoridation of drinking water delivers an uncontrolled dose of a chronically toxic substance to communities. In artificially fluoridated areas the prevalence and severity of dental fluorosis is increasing. Formula-fed infants are a high-risk group for dental fluorosis, because they can ingest 4-6 times the daily doses which American paediatricians recommend for fluoride supplements in unfluoridated areas. Reports of hypersensitivity reactions in the medical and dental literature have not been refuted scientifically. Skeletal fluorosis has been reported in naturally fluoridated areas of at least 5 countries where [F^-] ranges from 0.7 to 2.5 ppm. It is likely to become prevalent among high-risk groups (people with malfunctioning kidneys and/or high water intake) in artifically fluoridated areas as people who have been exposed to 1 ppm fluoridated water from infancy reach middle and old age.

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