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Submission to South Central Strategic Health Authority (SHA)

The Arguments Against the Proposal to Fluoridate Southampton

Introduction

It has taken me four solid days to prepare this submission. I do not have any illusions about the SHA paying serious attention to this work, because it is clear that the SHA made up its collective mind to fluoridate Southampton months ago. The brochure the SHA provided to the public on this matter revealed this most clearly. This brochure was as far from a balanced presentation on this controversial issue as it could possibly get. This same applies to the material (both the physical displays and the oral information) presented in the Open House meetings. Thus while in other ways the SHA has tried to give the appearance of impartiality, particularly the three "Question Time" type meetings held at the Football Stadium (which was conducted in a scrupulously fair fashion by the chair) in which I participated, all of this was part of a massive PR exercise to achieve a pre-ordained end: namely to fluoridate Southampton, regardless of scientific input or public opposition. On the matter of the "Question Time" meetings, I thought it was also interesting to note that we were told several times that the videotapes would be used to record the questions asked by the audience, but at no time were we told that the panel's *answers* would be considered in their deliberations. The final straw for me was seeing a postcard distributed at taxpayers' expense calling for people to respond to this proposal but only providing a single box for a "yes" vote. No box was provided for a "no" vote!

Now if this had all been part of an exercise by the British Fluoridation Society then perhaps one would have expected such blatant partisanship, however since this exercise is being funded by the British taxpayer I believe that it is inexcusable.

I have gone to the lengths I have to both participate in all three "Question Times" and present this written submission, not in the hope that the scientific evidence and the arguments I have presented will be considered seriously or even scientifically, but rather to present the very best case to members of the public that care about this matter, especially those, who like myself, believe that public health policy should be shaped by honest science combined with sound ethical principles. I also present it so that some future historian may actually find out that there were honest scientists who presented this case. I will leave it to them to pass judgment on why they think this solid case was ignored, as I expect it will be.

I will present my concerns in two parts.

In Section 1, I will present some key common sense, ethical and scientific questions that in my view need to be answered before decision makers or their advisers give the go ahead for fluoridating Southampton and other parts of Hampshire.

In Section 2, I will address each of these questions in turn and provide the necessary scientific documentation to support my arguments that this proposal be rejected.

I hope - in the rush to meet the deadline for this submission- I have been able to provide all the references cited in this submission. If any are missing it is highly likely that they can be found in the reference list for the NRC (2006) "Fluoride in Drinking Water: A Scientific Review of EPA's Standards" report, which is online at http://books.nap.edu/openbook.php?record_id=11571&page=268.

The references on the literature pertaining to fluoride's impact on the brain and on hip fractures are listed separately at the end of the reference list.

For those who would wish to explore more fully the impacts of fluoride on various organs, they will find extensive annotated discussions on each organ in the Health Effects Database (http://www.FluorideAlert.org/health/).

I have arranged for a copy of a book by Dr. Bruce Spittle entitled "Fluoride Fatigue" first published in 2008 to be forwarded to you. As it may arrive after the deadline for submissions the transcript can be viewed as a pdf file at http://www.pauapress.com/fluoride/files/1418.pdf. I would like this book to be considered as part of my submission. This book is an invaluable guide to the early reversible symptoms of fluoride poisoning which are suffered by a large number of people, but hitherto have gone largely unnoticed by the medical community in fluoridating countries. The updated version of this book contains very valuable information from Dr. A.K. Susheela who has spent the last 34 years studying the impacts of fluoride on the Indian population.

I am also sending by separate email a power point presentation outlining my arguments against fluoridation, which contains many figures, which provide a clearer illustration of some the arguments made in this submission.

Section 1. Key Questions for the SHA Panel to answer.

1) How convincing is the evidence that Southampton has a dental crisis that would warrant this kind of intervention?
2) How convincing is the evidence that swallowing fluoride reduces dental decay?
3) Is swallowing fluoride the best way of protecting the tooth enamel? Are there more appropriate delivery systems?
4) Have other communities demonstrated alternative methods of fighting tooth decay, which do not involve forcing a practice on people who may not want it?
5) Are children in the community already exceeding the "optimal dose" of fluoride as hypothesized by early promoters of fluoridation?
6) If ingested fluoride is necessary to protect children's teeth can the SHA explain why it is that the level of fluoride is so low in mothers' milk (0.004 ppm)? Did evolution screw up on the baby's first meal and nutritional requirements?
7) What is the evidence that has convinced you, or the experts upon whom you rely, that this program can be applied without causing any damage to the health of your citizens, especially infants and young children, other than an increase in the incidence of dental fluorosis?
8) What is the evidence that has convinced you, or the experts upon whom you rely, that a bottle fed infant will suffer no damage to its growing tissues when exposed to fluids containing fluoride at levels 250 times higher than the level in mothers milk (1 ppm versus 0.004 ppm)?
9) What is the evidence that has convinced you, or the experts upon whom you rely, that fluoride can damage the growing tooth cells (by some systemic mechanism) without damaging any other tissue in the child's developing body? How convincing is this evidence?
10) What is the evidence that has convinced you, or the experts upon whom you rely, that no one in your community is particularly sensitive or vulnerable to fluoride's known toxic effects? How convincing is this evidence?

11) Do you dispute the fact that fluoride - given in a sufficient dose - can cause a whole range of health effects from the very mild to the very serious? Do you dispute the fact that this has been demonstrated in hundreds of studies from India, China and other countries and communities that are exposed to high levels of natural fluoride in their water?
12) Please present a discussion of what is meant by "Margin of safety" for a toxic substance or "therapeutic index" for a pharmaceutical substance.
13) In your view, and the view of the experts on whom you rely, is there an adequate margin of safety between the doses or levels reported to cause adverse effects in the National Research Council (NRC, 2006) report, "Fluoride in Drinking Water: A Scientific Review of EPA's Standards," and the doses that people are likely to receive drinking fluoridated water (together with fluoride from other sources like dental products, pesticides etc) sufficient to protect everyone in your community?
14) There have now been 23 studies from 4 different countries (Iran, India, Mexico and China) that report an association between fluoride exposure and lowered IQ in children. What evidence has convinced you that all 23 studies can be safely ignored as suggested by Dr. Barry Cockcroft in his public testimony? Has Dr. Cockcroft, or his staff, produced a written scientific analysis supporting this claim?
15) What peer-reviewed and PRIMARY published studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and lowered IQ in fluoridated communities and convinced you that this is not a problem?
16) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and lowered thyroid function (including sub-clinical hypothyroidism) in fluoridated communities and convinced you that this is not a problem?
17) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have compared the levels of fluoride in the pineal glands of people living in fluoridated and non-fluoridated communities and convinced you that this is not a problem?
18) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have examined a possible relationship between a) fluoride exposure and melatonin levels and b) fluoride exposure and an earlier onset of puberty among children in fluoridated communities and convinced you that this is not a problem?
19) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and bone fractures in children in fluoridated communities and convinced you that this is not a problem?
20) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and arthritis in fluoridated communities and convinced you that this is not a problem?
21) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have convinced you that lifelong consumption of fluoridated water along with other sources of fluoride will not weaken the bones of the elderly and cause an increased rate of hip fractures, in fluoridated communities?
22) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and osteosarcoma in fluoridated communities, and convinced you that this is not a problem?
23) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have used the severity of dental fluorosis as a biomarker for epidemiological studies on children in fluoridated communities?
24) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have surveyed the population in the UK or any other fluoridated country, in a comprehensive fashion for the level of fluoride in their bones as a function of age, fluoridation status and other variables?
25) What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have surveyed the population in the UK or any other fluoridated

country, in a comprehensive fashion for the level of fluoride in their urine, as a function of age, diet, fluoridation status and other variables?

26) Are you satisfied that over the 60 year history of fluoridation that sufficient effort has been made by the governments, which promote this practice, to investigate possible health effects (in tissues other than the teeth) in fluoridated communities?
27) If this program moves forward are any health studies planned for the local community?
28) If this program moves forward will any compensation be given to children who develop very mild, mild, moderate or severe dental fluorosis? Will they be provided with free treatment for these conditions if desired?
29) If this program moves forward and some citizens complain of reversible symptoms, which elsewhere have been identified as being caused by fluoride (i.e. they cease when the source of fluoride is removed), will any steps be taken to investigate the matter scientifically?
30) Please discuss what you understand by the **Precautionary Principle**. Do you believe that in the context of the current scientific uncertainties about fluoridation's effectiveness and safety, and the availability of alternatives, that fluoridation is consistent with the Precautionary Principle?
31) Normally governments only use their **police power** to enforce medication on people when they are dealing with a life threatening contagious disease. Do you believe that this is the situation that confronts Southampton with respect to current dental decay levels?
32) Bearing in mind your responses to all of the above, are you convinced that the evidence of benefit from this practice is so strong, and the evidence of harm so weak, that it merits the application of governmental police power to force this practice on your citizens regardless of their views on the matter?
33) Could you summarize the evidence that has convinced you that there are no extra problems associated with using hexafluorosilicic acid (an industrial waste product) as a fluoridating chemical as opposed to pharmaceutical grade sodium fluoride?
34) If you are convinced that children's teeth in Southampton will benefit from ingesting fluoridated water please compare these two delivery systems: 1) adding contaminated hexafluorosilicic acid to the public water supply and 2) making fluoridated bottled water available in local supermarkets and chemists, and free for families of low income.
35) Are you prepared to do the whole community what an individual doctor is not allowed to do his or her individual patients: i.e. override the individual's right to **informed consent** to medication?

Section 2. Connett's answers and commentary on the Key Questions presented in Section 1.

How convincing is the evidence that the community has a dental crisis sufficiently serious to warrant such a controversial intervention?

I believe that Stephen Peckham's submission fully addresses this question and he convincingly demonstrates that there is no proven dental crisis in Southampton sufficient to warrant such an intervention.

Can swallowing fluoride reduce dental decay? How convincing is this evidence?

Clearly, if the promoters of fluoridation cannot convincingly demonstrate with bona fide primary research studies that fluoridation actually works to reduce tooth decay to a significant extent, then there is no justification for proceeding with this proposal. This is true even if no harm could be associated with the practice. It is even more so if potential harm can be demonstrated. The latter will be covered below. In this section I will start by demonstrating that the evidence that swallowing fluoride reduces tooth decay is extremely weak. The

following summary should be compared with the evidence proffered by proponents that fluoridation is effective.

1) **Fluoride is not an essential nutrient**. Fluoride is not an essential nutrient (NRC 1993 and IOM 1997). No disease has ever been linked to a fluoride deficiency. Humans can have perfectly good teeth without fluoride.
2) **No double blind studies.** There has never been a double blind trial demonstrating fluoridation's effectiveness. See the York Review (McDonagh et al., 2000) that could identify NO grade A studies, which have investigated fluoridation's effectiveness.
3) **Controlling for delayed eruption of teeth.** Not one single study purporting to demonstrate fluoridation's effectiveness has ever controlled for delayed eruption of teeth caused by fluoride (Feltman and Kosel, 1961; Komarek, et al. 2005).
4) **Primary versus secondary dentition.** Those promoting fluoridation usually do so using the data on primary dentition (deciduous teeth) rather than secondary dentition (permanent teeth). However, it is the latter which are more important since these are the teeth we hope to have for the rest of our lives.
5) **Cross-sectional versus Longitudinal studies.** The York Review (McDonagh et al., 2000) only looked at longitudinal studies. Cross-sectional studies are much larger and more convincing in indicating no or little benefit from ingesting fluoride to reduce tooth decay. Some of these are discussed below
6) **Baby bottle tooth decay.** Even promoters of fluoridation have conceded that fluoridation cannot prevent baby bottle tooth decay (BBTD) and this is the cause of the most distressing examples of tooth decay in infants often leading to extractions under anesthesia. BBTD is caused by babies sucking on sugared water, fruit juice (and even coca cola) for hours on end (Kelly et al., 1987; Barnes et al., 1992; Weinstein et al., 1992; Von Burg et al., 1995; Febres et al., 1997; Tang et al., 1997; Blen et al., 1999 and Kong, 1999).
7) **Pit and fissure decay.** Since 1950, it has been found that fluorides do little to prevent pit and fissure tooth decay, a fact that even the dental community has acknowledged (Seholle 1984; Gray 1987; PHS 1993; and Pinkham 1999). This is significant because pit and fissure tooth decay represents up to 85% of the tooth decay experienced by children today (Seholle 1984 and Gray 1987). Pit and fissure decay is best prevented with sealants.
8) **Decay rates were coming down before fluoridation was introduced and have continued after the benefits of the practice would have been maximized**. Modern research (e.g. Diesendorf 1986; Colquhoun 1997, and De Liefde, 1998) shows that decay rates were coming down before fluoridation was introduced and have continued to decline even after its benefits would have been maximized (see discussion on Diesendorf's 1986 paper below). Many other factors influence tooth decay. Some recent studies have found that tooth decay actually increases as the fluoride concentration in the water increases (Olsson 1979; Retief 1979; Mann 1987, 1990; Steelink 1992; Teotia 1994; Grobleri 2001; Awadia 2002 and Ekanayake 2002).
9) **Little difference between fluoridated and non-fluoridated communities**. There is very little evidence demonstrating a significant difference in the permanent teeth when comparing children living in fluoridated and non-fluoridated communities (Leverett, 1982; Diesendorf, 1986; Gray, 1987; Yiamouyiannis, 1990; Brunelle and Carlos, 1990; Spencer et al., 1996; deLiefde, 1997; Locker, 1999; Armfield & Spencer, 2004 and Pizzo et al., 2007). These studies are discussed further below.
10) **WHO data.** According to WHO data there is no significant difference in the rates of decline in decay in 12-year olds teeth over the period from the 1960s to the present, between fluoridated and non-fluoridated countries. The same set of data shows no significant difference today between fluoridated and non-fluoridated countries. See the figure at http://www.FluorideAlert.org/who-dmft.htm, which presents this data graphically. See also a similar graph presented in the article by Cheng et al, 2007 in the British Medical Journal.

11) **DHHS survey.** Osmunson has showed that according to the results of a questionnaire administered to parents in all 50 states in the US by the Department of Health and Human Services (DHHS), there is absolutely no relationship in the percentage of parents who responded "my child has very good or excellent teeth" and the percentage of the population in the state drinking fluoridated water (Osmunson, 2007). However, there is a very strong relation in all 50 states between the percentage of parents giving that answer and their income levels. Across the board 80% of high income parents gave that answer, but only about 60% of low income parents gave that answer (Osmunson, 2007).
12) **New York State survey**. In 2002-2004 the pro-fluoridation NY Department of Health (NYDOH) conducted a survey of tooth decay in third graders in NY State. When the tooth decay in third graders (average by county), was plotted against the percentage of each county drinking fluoridated water, linear regression shows there is no relationship. There was however a clear relationship with average county income levels. (NYDOH, 2007)
13) **Tooth decay and income levels.** What both 11) and 12) above clearly show is that there is a much stronger relationship between tooth decay and parent's income level than whether the child drinks fluoridated water or not. If parental income level is not very carefully controlled (and other factors like delayed eruption, diet, genetic, ethnic, cultural and educational differences, parental oversight, as well as the dental services available) almost any result can be obtained when comparing a fluoridated and non-fluoridated community.
14) **Birmingham versus Manchester.** In the three Question Times organized by the SHA, fluoridation proponents put a great deal of emphasis on a comparison of tooth decay in 5 year olds between Manchester and Birmingham. However on inspection of the published survey (not study) cited, this conclusion was based on just two numbers: the average decayed missing or filled teeth (dmfts) of the two cities in question. No information was given on all the other factors that might have influenced these two numbers other than fluoridation status. No mention is made of delayed eruption, or dietary, genetic, ethnic, cultural, parental oversight or educational differences as confounding variables. Nor, most importantly, was mentioned how much money was spent on dental services in each community or the number of interventions administered. In fact, some evidence suggests that money on these services (and the number of interventions) continues to rise in the Birmingham area (this has been documented by NPWA for Wolverhampton and Coventry) and continue to decline in the Manchester area. One or more of these factors listed above could explain these differences. What is needed is a bona fide scientific study where these factors are carefully controlled. Meanwhile, these comparisons look more like a self-serving and self-fulfilling prophesy on behalf of fluoridation promoters, than a genuine comparison of the effects of ingesting fluoride. There is also evidence in the US that commensurate with the introduction of fluoridation in some cities (e.g. San Antonio, Texas) the measure has been accompanied with other expensive measures to fight tooth decay.
15) **No overall pattern in UK survey.** Furthermore, as both Stephen Peckham and John Spottisworth pointed out at the public "Question Times", when the full list of dmfts for all the communities in the UK survey are examined no clear pattern emerges which shows a consistent superiority in fluoridated versus non-fluoridated towns in the UK. It is thus very misleading to focus on just two towns in a very long list.
16) **Early trials and Dean's 21-city study.** A great deal of the conviction that fluoridation works has been derived from Dean's famous 21-city study in 1942 and the early fluoridation trials in the US, Canada and New Zealand. However, both the legitimacy and the quality of the methodologies of these have been questioned.
17) **Dean's study has been questioned by Ziegelbecker**. Ziegelbecker, an Austrian statistician asked why Dean relied on the data from only 21 cities when he had data from many other cities at his disposal. When Ziegelburger added in all the data he could find from the US and Europe, which related tooth decay with fluoride levels in the water, the inverse relationship reported by Dean disappeared. However, when he examined the same data for dental fluorosis he found a very robust relationship. (Ziegelbecker, 1970).

Thus one relationship (fluoride and dental fluorosis) holds up over the “noise”, the other (fluoride and dental decay) does not.

18) **The early trials.** The trials conducted in 1945 -1955 in the US, and Canada, which helped to launch fluoridation, have been heavily criticized for their poor methodology and poor choice of control communities (De Stefano 1954; Sutton 1959, 1960 and 1996; Ziegelbecker 1970). According to Dr. Hubert Arnold, a statistician from the University of California at Davis, the early fluoridation trials "are especially rich in fallacies, improper design, invalid use of statistical methods, omissions of contrary data, and just plain muddle-headedness and ineptitude.” Proponents have never refuted Sutton’s monographs on this matter, even though they have tried. Sutton’s work was re-published in book form shortly before he died in 1996.

19) The Hastings-Napier trial a fraud. The Hastings-Napier trial in New Zealand conducted in the 1950s has shown to be fraudulent by Colquhoun and Mann. The control community (Napier) was dropped two years after the trial began and the huge drop in tooth decay found in Hastings was found to be due to an artifact involving a change in methodology used to characterize tooth decay before and after the trial (i.e. diagnosing tooth decay was less stringent at the end of the trial than at the beginning). The fact that the methodology was changed was NOT acknowledged by the authors in their published report (Colquhoun and Mann, 1986).

20) **Colquhoun.** If we shift to more modern times, a major development occurred in 1980. This was when Dr. John Colquhoun was sent by his superiors in New Zealand on a four-month world tour to investigate tooth decay in several different continents, including Australia, Asia, North America and Europe. He was asked to bring back with him evidence that would prove once and for all that fluoridation worked.

At the time, Colquhoun was the principle dental officer for Auckland, NZ’s largest city. Both as a dental officer and as a city councilor he had avidly and successfully promoted fluoridation throughout the country.

To his chagrin researchers behind the scenes reported to Colquhoun that they were not finding the difference in tooth decay between fluoridated and non-fluoridated communities that they had expected – in fact they weren’t finding any.

When Colquhoun returned to NZ he was given a summary of tooth decay for the whole of the country. NZ is a little unusual in this respect since under their national health service they monitor tooth decay for all children at the ages of 5 and 12. So this was not a sample survey but a complete record.

When he looked at the complete record of tooth decay in NZ, he found no difference in tooth decay between the fluoridated and non-fluoridated cities. If anything, the teeth were slightly better in the non-fluoridated communities.

When Colquhoun’s assistants reported to him the extensive amount of dental fluorosis occurring in fluoridated Auckland, he risked his pension by deciding to make the lack of fluoridation’s effectiveness public. To his enormous credit he spent the rest of his life trying to undo the damage he had done by reversing his position on fluoridation, and opposing it in any way he could. I interviewed John on videotape in Auckland in 1997 shortly before he died. (See videotape). He also summarized his experiences in the article “ Why I changed my mind on fluoridation.”

Colquhoun wrote up his findings in several published papers (Colquhoun 1984, 1985, 1987, 1990, 1992 and 1995) and after he retired he obtained a PhD (1987). His research thesis examined the history of fluoridation in New Zealand. He explained the reluctance of the dental community to change its paradigm on fluoridation’s safety and effectiveness in NZ using Thomas Kuhn’s famous analysis: “On the Nature of Scientific Revolution.” Colquhoun

summarized some of his experiences in this matter in a paper, “Why I changed My Mind on Fluoridation,” published in 1997.

18) Leverett (1982). Meanwhile, in the 1980s other articles began to appear in major journals like Science and Nature, pointing out there was little difference in tooth decay between fluoridated and non-fluoridated communities. The first was by **Leverett** in Science in 1982?

19) Diesendorf, 1986. The next significant paper came from Mark Diesendorf who published, “The Mystery of Declining Tooth Decay” in Nature in 1986. In Diesendorf showed that tooth decay was coming down before fluoridation was introduced into several Australian towns, and continued to decline long after any benefits would have been maximized. It is important to comment on this latter finding because it remains relevant – but often overlooked - today. I will use tooth decay in 12 year olds to explain. 12 years after fluoridation has begun in a city, all 12 year olds will have been exposed to fluoridated water for 12 years (i.e. their whole lives). For all subsequent years – ALL 12 year olds will also have been exposed to fluoridated water their whole lives. Any further drop in tooth decay cannot be ascribed to fluoridation. That has been maximized for all 12 year olds after 12 years of the program. Today, careless observers are using current data in fluoridating communities without referring back to the numbers ascertained at the maximization point.

20) Gray, 1987. Another article indicating little difference in tooth decay between fluoridated and non-fluoridated communities in British Columbia was published by Gray in 1987.

21) NIDR, 1986-87. At this point the National Institute of Dental Research (NIDR) stepped in and organized – at great expense to the US taxpayer - the largest survey of tooth decay ever undertaken in the US. They examined the teeth of nearly 40,000 children in 84 communities.

22) Yiammouyiannis, 1990. When Dr. John Yiammouyiannis (a well-known opponent of fluoridation) obtained the raw data from the NIDR he found that there was no statistical difference between the DMFTs (decayed missing and filled permanent teeth) of children between those who lived all their lives in a fluoridated community or a non-fluoridated community – across the whole age range from 5 to 17.

23) Brunelle and Carlos, 1990. Subsequently, two authors from the NIDR (Brunelle and Carlos) published their own analysis of the data. However, they decided to jack up the sensitivity of the study by analyzing tooth decay as DMFS (decayed missing filled permanent surfaces). All except the cutting teeth have 5 surfaces per tooth so this jacked up the sensitivity by nearly a factor of five. Even so they found very little difference between children who lived all their lives in a fluoridated community or a non-fluoridated community. The authors reported the average difference for 5-17 year olds as 18%. However, if one goes to Table 6 in their paper one finds that this average saving amounts to just was 0.6 of one tooth surface, and that is out of approximately 100 tooth surfaces in a child’s mouth. Nor did the authors subject this to any analysis to see if the result was statistically significant.

24) Spencer et al., 1996. Subsequently a large survey conducted in two states in Australia by Spencer et el. in 1996, found an even lower difference in average tooth decay in the permanent teeth, measured as DMFS, between children who had lived all their lives in fluoridated versus non-fluoridated communities. The found a difference of between 0.12 and 0.3 tooth surfaces.

25) De Liefde, 1998. In 1998, De Liefde in a survey of tooth decay in New Zealand described the difference in permanent tooth decay between fluoridated and non-fluoridated communities as “clinically meaningless.”

26) Locker, 1999. In a report prepared for the Ontario Ministry of Health & Long Term Care, Dr. David Locker, of the University of Toronto, reported, “The magnitude of [fluoridation’s]

effect is not large in absolute terms, is often not statistically significant, and may not be of clinical significance."

27) The halo effect. Some promoters of fluoridation have tried to rationalize these findings by claiming a halo effect. They suggest that children in non-fluoridated communities are getting fluoride from food and beverages prepared in fluoridated communities. However, this explanation cannot possibly be used to explain the lack of difference in tooth decay between fluoridated and non-fluoridated countries in Europe, because the vast majority is unfluoridated and thus there is little or no source of fluoridated beverages or foodstuffs in non-fluoridated countries.

28) CDC, 1999. Clues to more a likely explanation for the lack of difference in tooth decay between fluoridated and non-fluoridated communities came in 1999 when the longtime promoter of fluoridation, Centers for Disease Control and Prevention (CDC) conceded that promoters had got the mechanism of fluoride's action wrong for over 50 years. The early belief was that for a child to benefit from fluoride they had to absorb fluoride before their teeth had erupted so that fluoride would build up in the enamel and make it more resistant to acid attack when they eventually emerged into the oral cavity. But, in 1999, the CDC conceded - along with the majority of dental researchers - that not enough fluoride was absorbed into the enamel via this process to work, and concluded that the major benefits of fluoride are TOPICAL not SYSTEMIC. This is what the CDC stated:

> "Fluoride's caries-preventive properties initially were attributed to changes in enamel during tooth development because of the association between fluoride and cosmetic changes in enamel and a [false] belief that fluoride incorporated into enamel during tooth development would result in a more acid-resistant mineral.
> However, laboratory and epidemiologic research suggests that fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children."

Thus it is more likely that the universal availability of fluoridated toothpaste has done more to reduce tooth decay than swallowing fluoridated water. Moreover, as the benefits of fluoride are topical, and the risks are systemic, it makes more sense, for those who want to take the risks, to deliver the fluoride directly to the tooth in the form of toothpaste. Since swallowing fluoride is unnecessary, there is no reason to force people (against their will) to drink fluoride in their water supply. This position was clearly stated by Dr. Douglas Carnall, the associate editor of the British Medical Journal, shortly after the publication of the York Review. His editorial appears in Appendix 3.

29) Other explanations for the universal decline of tooth decay in industrialized countries. Other explanations for the decline in tooth decay in both fluoridated and non-fluoridated countries include, an increased standard of living. With this goes a better diet, with more fruit and vegetables, more cheese, refrigeration, more time for parents to supervise their kids' dental habits and more money to pay for dental services. Another possibility offered by deLiefde (1998) is the increased consumption of processed foods containing antibiotics as preservative agents. Some of these antibiotics she has argued would reduce the bacteria in the mouth responsible for converting sugars to acids.

30) Armfield and Spencer, 2004. In 2004, Armfield and Spencer published a study, which investigated tooth decay in 10,000 children in South Australia. While they found a small difference in the primary teeth they found no statistically significant difference between tooth decay in the permanent teeth between those children who had drunk tank water (rain water) and those who drank fluoridated water. Armfield and Spencer claim that fluoridation opponents are misusing their study, however in their abstract the authors clearly state: "The

effect of consumption of nonpublic water on permanent caries experience was not significant."

31) Pizzo, 2007. In 2007, a team of Italian researchers from the University of Palermo, concluded from their review of the literature:

> "…it is now accepted that systemic fluoride plays a limited role in caries prevention. Several epidemiological studies conducted in fluoridated and non-fluoridated communities clearly indicate that CWF (community water fluoridation, PC) may be unnecessary for caries prevention, particularly in the industrialized countries where the caries level has become low."

32) When fluoridation is discontinued. Contrary to claims from proponents that when fluoridation is discontinued tooth decay goes up, several modern studies indicate the very opposite. Where fluoridation has been discontinued in communities from Canada, the former East Germany, Cuba and Finland, dental decay has not increased but has actually decreased (Maupome 2001; Kunzel and Fischer, 1997,2000; Kunzel 2000 and Seppa 2000).

33) A dental crisis in many fluoridated cities in the US. There have been numerous media reports of dental crises in US cities (e.g. Boston, Cincinnati, New York City), which have been fluoridated for over 20 years. This again demonstrates that there is a far greater (inverse) relationship between tooth decay and income level than with water fluoride levels.

Is swallowing fluoride the best way of protecting the tooth enamel? Are there more appropriate delivery systems?

The concession from many dental researchers and conceded by the CDC in 1999, that fluoride's major benefits accrue topically not systemically (see above), would suggest that topical applications like the use of universally available fluoridated toothpaste make more sense. There is no reason to force people (against their will) to drink fluoride in their water supply. This position was clearly stated by Dr. Douglas Carnall, the associate editor of the British Medical Journal, shortly after the publication of the York Review in 2000. His editorial appears in Appendix x. It was also recently echoed by Dr. Arvid Carlsson, who led the successful campaign against fluoridation in Sweden in the 1970s and won the Nobel Prize for Medicine in 2000. In an interview with Michael Connett he stated:

> "In pharmacology, if the effect is local (topical), it's awkward to use it in any other way than as a local treatment. I mean this is obvious.
> You have the teeth there, they're available for you, why drink the stuff?"

Have other communities demonstrated alternative methods of fighting tooth decay, which do not involve forcing fluoridation on people who don't want it?

The vast majority of European countries neither fluoridate their salt nor their water and yet according to WHO data (discussed above) their tooth decay is no worse than fluoridated countries and in some case better (see appendix 1). Clearly, a careful examination of the methods they have used need to be scrutinized before embarking on the more draconian and controversial approach of forcing fluoridation on new communities. One particularly impressive Swedish research report (Axelsson et al, 1993) needs to be examined very carefully in this respect.

Are children in Southampton already exceeding the "optimal dose" hypothesized by early promoters of fluoridation?

According to H. Trendley Dean and other early promoters of fluoridation, the so-called "optimal" level of fluoride for fighting tooth decay was 1 ppm fluoride in the water. At this level they thought

tooth decay would be lowered while causing only minimal levels of dental fluorosis. Specifically, they believed that at "optimal levels" of fluoride exposure approximately 10% of children would develop dental fluorosis, and only in its very mild form (NRC 1993, pp. 6-7). A major US survey has found 30% of children in optimally fluoridated areas had dental fluorosis on at least two teeth (Heller 1997), while smaller studies have found up to 80% of children impacted (Williams 1990; Lalumandier 1995 and Morgan 1998). The York Review estimates that up to 48% of children in optimally fluoridated areas worldwide have dental fluorosis in all forms and 12.5% with symptoms of aesthetic concern (McDonagh, 2000).

Thus, a very simple and scientific way to determine whether Southampton children are receiving above or below so-called "optimal" levels of fluoride is to see what percentage of children have dental fluorosis. If over 10% of the children are already being impacted in this way then according to the fluoridation hypothesis children in the community are already getting more than sufficient fluoride to fight tooth decay.

In the US today, over 20% of children in non-fluoridated communities have dental fluorosis so they are getting more than enough fluoride (Heller, 1997; CDC, 2005). Indeed, the problem is not under-exposure but over-exposure to fluoride. Is this the case in Southampton? For less money than has already been spent on this consultation process this issue could be resolved scientifically.

One of the reasons why we are seeing far higher dental fluorosis rates than anticipated by the early fluoridation promoters is because there are far more sources of fluoride available to us today then there were in 1945, when fluoridation began. Other sources of fluoride include food and beverages processed with fluoridated water (Kiritsy 1996 and Heilman 1999), fluoridated dental products (Bentley 1999 and Levy 1999), mechanically deboned meat (Fein 2001), teas (Levy 1999), and pesticide residues on food (Stannard 1991 and Burgstahler 1997).

What is the evidence that has convinced you, or the experts upon whom you rely, that this program can be applied without causing any damage to the health of your citizens, especially infants and young children, other than an increase in the incidence of dental fluorosis?

When fluoridation was launched in the US in 1945, there was one piece of evidence that was known, namely that exposure to fluoride would cause some children to develop dental fluorosis. This was also known to be a systemic effect. What was not known was if other developing tissues in the child's body were being affected systemically while the growing tooth cells were being damaged.

David Ast, was the chief dental officer for NY State and was one of the key architects of the important fluoridation trial in Newburgh versus Kingston NY (1945-55). This is what he wrote in 1944, before this trial began:

> "Are there any cumulative effects - beneficial or otherwise, on tissues and organs other than the teeth - of long continued ingestion of such small concentrations as 1.0 part per million of fluorine (sic) in water? Again, there is much presumptive evidence that there are no such effects: but, until that is demonstrated, the procedure outlined in this paper must be regarded as an investigation." *David Ast, "A plan to determine the practicability, efficacy and safety of fluorinating a communal water-supply, deficient in fluoride, to control dental caries, Oct 30, 1944*

Two adverse effects were observed during this Newburgh vs. Kingston trial. One was a statistically significant difference (about 2 to 1) in the number of cortical bone defects in the children from the fluoridated community compared to the non-fluoridated community. The other was that on average the young girls in the fluoridated community reached menstruation approximately 5 months earlier than the young girls in the non-fluoridated community. Neither

effect has been investigated in artificially fluoridated communities since this time (Schlesinger, et al., 1956).

As far as the effect on menstruation is concerned the absence of any official concern became extremely puzzling when Jennifer Luke discovered in 1997 that fluoride accumulated in the human pineal gland (Luke, 1997, 2001). This gland produces a hormone called melatonin, which acts like a biological clock in many timed events in human development. The levels of melatonin are thought to influence on the onset of puberty. In her PhD thesis, Luke presented evidence that the high fluoride treated gerbils had lowered melatonin levels and reached puberty earlier than the low fluoride treated animals. This part of her PhD thesis has yet to be published. No fluoridating country has financed or requested a study to reproduce Luke's findings. In 2002, a MRC committee gave a lower priority to such research than further investigations of dental fluorosis (MRC, 2002).

As far as the observed bone effects are concerned, bearing in mind that the cortical bone is the outside layer of the bone which is critical in protection against stress fractures (as opposed to compression fractures) it is surprising that no effort has been made (for over 50 years since the Newburgh- Kingston study) to investigate the rates of bone fractures in young children exposed to fluoridated water. However, in Mexico in 2001, Alarcon-Herrera and co-workers examined bone fractures in children living in a high fluoride area (1.5 – 5.5 ppm fluoride in water) and reported a linear correlation between the severity of dental fluorosis in children (a biomarker of the level of over-exposure to fluoride) and the incidence of bone fractures reported in local clinics. While spokespersons for governmental agencies promoting fluoridation have criticized the methodology used in this study, since 2001 not one single agency in fluoridated countries has sort to repeat this study, even though they are aware that dental fluorosis impacts over 30% of children in the US.

Even more disturbing is the fact that not one single health agency in fluoridated countries has sort, or financed, studies which would have used the severity of dental fluorosis as a biomarker to investigate a possible relationship between other childhood complaints and exposure to fluoride.

The Newburgh-Kingston study was the first evidence that promoters of fluoridation are capable of ignoring potentially important findings, while they continue to tell the public with confidence that this practice is "safe and effective." Sadly there are many more examples of this state of denial by fluoridation promoters which have occurred throughout is 60 year history.

What is the evidence that has convinced you, or the experts upon whom you rely, that fluoride can damage the growing tooth cells (by some systemic mechanism) without damaging any other tissue in the child's developing body? How convincing is this evidence?

Dental fluorosis means that a child has been overdosed on fluoride. While the mechanism by which the enamel is damaged is not definitively known, it appears fluorosis may be a result of either inhibited enzymes in the growing teeth (Dan Besten 1999), or through fluoride's interference with G-protein signaling mechanisms (Matsuo 1996). Whatever the mechanism, the outrageous assumption made by early promoters, without any evidence to support the assumption, was that fluoride could damage the growing tooth cells without damaging any other tissue in the body.

When the US Public Health Services endorsed fluoridation in 1950, not one trial of efficacy had been completed nor had they seriously investigated this question of other health effects. The shocking thing is that since 1950, neither the US nor other governments that promote this practice have seriously investigated the possibility that fluoride is damaging soft tissues.

They spend more time trying to discredit studies done overseas (i.e. in non-fluoridated countries) than they do investigating the matter in their own countries.

In the absence of health studies in many vital tissues in fluoridated countries, should we be concerned? If we look at fluoride's biochemistry, physiology and impacts on animals most toxicologists would say yes. This is some of what we know:

a) The level of fluoride put into water (1 ppm) is 250 times higher than normally found in mothers' milk (0.004 ppm) (NRC, 2006, Ekstrand 1981; Institute of Medicine 1997). There are no benefits, only risks, for infants ingesting this heightened level of fluoride at such an early age (this is an age where susceptibility to environmental toxins is particularly high).

b) Fluoride is a cumulative poison. On average, only 50% of the fluoride we ingest each day is excreted through the kidneys. The remainder accumulates in our bones, pineal gland, and other tissues. If the kidney is damaged, fluoride accumulation will increase, and with it, the likelihood of harm.

c) Fluoride is very biologically active even at low concentrations. It interferes with hydrogen bonding (Emsley 1981) and inhibits numerous enzymes (Waldbott 1978).

d) When complexed with aluminum, fluoride interferes with G-proteins (Bigay 1985, 1987). Such interactions give aluminum-fluoride complexes the potential to interfere with many hormonal and some neurochemical signals (Strunecka & Patocka 1999, Li 2003).

e) Fluoride has been shown to be mutagenic, cause chromosome damage and interfere with the enzymes involved with DNA repair in a variety of cell and tissue studies (Tsutsui 1984; Caspary 1987; Kishi 1993 and Mihashi 1996). Recent studies have also found a correlation between fluoride exposure and chromosome damage in humans (Sheth 1994; Wu 1995; Meng 1997 and Joseph 2000).

f) Fluoride forms complexes with a large number of metal ions, which include metals that are needed in the body (like calcium and magnesium) and metals (like lead and aluminum) that are toxic to the body. This can cause a variety of problems. For example, fluoride interferes with enzymes where magnesium is an important co-factor, and it can help facilitate the uptake of aluminum and lead into tissues where these metals wouldn't otherwise go (Mahaffey 1976; Allain 1996; Varner 1998).

g) Rats fed for one year with 1 ppm fluoride in their water, using either sodium fluoride or aluminum fluoride, had morphological changes to their kidneys and brains, an increased uptake of aluminum in the brain, and the formation of beta amyloid deposits which are associated with Alzheimer's disease (Varner 1998).

h) In 2000, Aluminum fluoride was nominated by the Environmental Protection Agency and National Institute of Environmental Health Sciences for testing by the National Toxicology Program. According to EPA and NIEHS, aluminum fluoride currently has a "high health research priority" due to its "known neurotoxicity" (BNA, 2000). If fluoride is added to water, which contains aluminum, than aluminum fluoride complexes will form.

i) Animal experiments show that fluoride accumulates in the brain and exposure alters mental behavior in a manner consistent with a neurotoxic agent (Mullenix 1995). Rats dosed prenatally demonstrated hyperactive behavior. Those dosed postnatally demonstrated hypoactivity (i.e. under activity or "couch potato" syndrome). More recent animal experiments have reported that fluoride can damage the brain (Wang 1997; Guan 1998; Varner 1998; Zhao 1998; Zhang 1999; Lu 2000; Shao 2000; Sun 2000; Bhatnagar 2002; Chen 2002, 2003; Long 2002; Shivarajashankara 2002a, b; Shashi 2003 and Zhai 2003) and impact memory,

learning and behavior (Paul 1998; Zhang 1999, 2001; Sun 2000; Ekambaram 2001; Bhatnagar 2002; Cagiano, 2007 and Chioca, 2007)
j) While studies conducted by FDA scientists in the US have failed to find reproductive effects in rats (Sprando 1996, 1997, 1998), studies from other countries have found a multitude of effects on reproduction in a whole range of animal species. At high doses fluoride wreaks havoc on the male reproductive system - it damages sperm and increases the rate of infertility in a number of different species (Kour 1980; Chinoy 1989; Chinoy 1991; Susheela 1991; Chinoy 1994; Kumar 1994; Narayana 1994a, b; Zhao 1995; Elbetieha 2000; Ghosh 2002 and Zakrzewska 2002).

What is the evidence that has convinced you, or the experts upon whom you rely, that no one in your community is particularly sensitive or vulnerable to fluoride's known toxic effects? How convincing is this evidence?

In an early study, which lasted 13 years, Feltman and Kosel (1961) gave patients 1 mg of fluoride per day. They were given either a tablet or a placebo. They found that about 1% of patients developed a range of neurological, dermatological and gastro-intestinal symptoms, which were reversed when the tablets were withdrawn. Numerous individuals in fluoridated communities have reported these same symptoms. George Waldbott and others have reported a number of case studies and double blind studies on this issue (Shea 1967, Waldbott 1978 and Moolenburg 1987). However, governments promoting fluoridation have ignored these findings and simply dismiss all such reports as "anecdotal." They have never attempted to put their dismissal on a scientific basis by carrying out scientific studies of their own. Particularly egregious in this matter is Australia where its own NHMRC in 1991 (which it touts as a promoter of fluoridation) recommended that the government carry out scientific studies to put the matter to rest one way or the other. Not one single health agency in Australia in the 17 years, since this recommendation was made, has done so. Recently, Dr. Bruce Spittle has published a book "Fluoride Poisoning" which goes into the issue of individual sensitivity to fluoride. I am arranging for a copy of this book to be forwarded to the SHA.

What has muddied the waters on this issue is the confusion between whether these individual responses are simply the tail end of the normal distribution of a range of sensitivity we can expect for any toxic substance or whether some people are actually allergic to fluoride (possibly by becoming sensitized to a whole range of chemicals via a toxic exposure incident). Supporting the former view is the fact that the symptoms reported are mimicked by symptoms experienced by those exposed to much higher doses either in clinical trials for the treatment of osteoporosis with sodium fluoride, or in areas of endemic fluorosis in India (Susheela, cited in Spittle, 2008).

According to the Agency for Toxic Substances and Disease Registry (ATSDR 1993), and other researchers (Juncos & Donadio 1972; Marier & Rose 1977 and Johnson 1979), certain subsets of the population may be particularly vulnerable to fluoride's toxic effects; these include: the elderly, diabetics and people with poor kidney function. Can you in good conscience force these people to ingest fluoride on a daily basis for their entire lives? One individual with impaired kidney function in the US developed skeletal fluorosis drinking water at 1.7 ppm (Juncos and Donadio, 1972).

Also vulnerable are those who suffer from malnutrition (e.g. calcium, magnesium, vitamin C, vitamin D and iodine deficiencies and protein poor diets). Those most likely to suffer from poor nutrition are the children from low-income families, who are precisely the people being targeted by new fluoridation programs. While being at heightened risk, poor families are less able to afford avoidance measures (e.g. bottled water or removal equipment). (Massler & Schour 1952; Marier & Rose 1977; Lin Fa-Fu 1991; Chen 1997; Teotia 1998).

Can the SHA in good conscience force any of these vulnerable people to ingest fluoride on a daily basis for their entire lives?

Do you dispute the fact that - given a sufficient dose - fluoride can cause a whole range of health effects from the very mild to the very serious? Do you dispute the fact that this has been demonstrated in hundreds of studies from India, China and other countries and communities, which are exposed to high natural levels of fluoride in their water?

It is very hard to see how any one can dispute this fact since the matter has been extremely well documented by the National Research Council in their 507 page report, "Fluoride in Drinking Water: A Scientific Review of EPA's Standards." This report has over 1000 references.

Please present a discussion of what is meant by "Margin of safety" for a toxic substance or "Therapeutic Index" for a pharmaceutical substance.

Such a discussion is critical before a sound decision can be made on whether to proceed with fluoridation or not. Proponents of fluoridation in Australia, New Zealand, the UK and the US have all dismissed the NRC report as being irrelevant to water fluoridation, claiming that it only applies to "high" exposure to fluoride. However, they do not explain what they mean by the word "high" or attempt to quantify the matter in any meaningful way. They also ignore chapter 2 which showed that some people consuming water at 1 ppm are exceeding the EPA's reference dose. To quantify this matter will require a) a careful examination of the NRC (2006) review to see the levels or doses adverse effects have been observed and b) a careful discussion of what MARGIN OF SAFETY is normally used by toxicologists, pharmacologists and regulatory officials when setting a safety margin sufficient to protect everyone from the full range of sensitivities to any toxic substance anticipated in any human population. Using the correct parlance, this safety factor is needed to protect for "intra-species variation." Pharmacologists use a similar concept when considering the possible dangers of pharmaceutical products. This is called the "therapeutic index" and is expressed as a ratio between the lowest toxic dose and the therapeutic dose.

Normally, the safety factor (Margin of Safety) applied when extrapolating from a no observable adverse effect level (NOAEL) is 10 and the safety factor applied to the lowest observable adverse effect level (LOAEL), if a NOAEL is not available, is 100. Has any evidence convinced you, or the experts upon whom you rely, that lower safety margins are acceptable in the case of this toxic substance and the end points discussed in the NRC (2006) report? How convincing is this evidence?

Sometimes a margin of safety lower than 10 or 100 is used if the data is collected in very large population studies where it is felt the full range of human sensitivity has been covered. However, in the case of fluoride, many of the studies have involved relatively small study groups that do not cover the full range of anticipated sensitivity in a large population.

In your view, and the view of the experts on whom you rely, is there an adequate margin of safety between the doses or the levels in the water where adverse health effects have been observed, as reported in the National Research Council review "Fluoride in Drinking Water" (NRC, 2006), and the doses that people are likely to receive drinking fluoridated water (together with fluoride from other sources like dental products, pesticides etc) sufficient to protect everyone in your community?

The people who need protecting include the very young, the very old, those with poor diet, including borderline iodine deficiency, those with poor kidney function (which reduces the ability to excrete fluoride) and those who consume above average quantities of water (athletes, diabetics etc) and bottle fed infants?

Even with safety margins much lower than 100 or 10, fluoridation would still be unacceptable, based upon the levels at which effects have been shown to occur. To continue promoting fluoridation when studies indicate that thyroid function is possibly lowered at 2.3 ppm

(Bachinskii et al., 1986); IQ in children might be lowered at levels as low as 1.8 ppm (Xiang et al, 2003) or at 0.88 ppm if there is borderline iodine deficiency (Lin et al, 1991) and hip fractures in the elderly may be doubled at levels as low as 1.5 ppm and tripled at levels over 4.3 ppm (Li et al., 2001), is reckless in my view.

There have now been 23 studies from 4 different countries (Iran, India, Mexico and China) which demonstrate a possible linkage between fluoride exposure and lowered IQ in children. What evidence has convinced you that all 23 studies can be safely ignored as suggested by Dr. Barry Cockcroft in his public testimony? Has Dr. Cockcroft, or his staff, produced a written scientific analysis supporting this claim?

The SHA has received a copy of all these studies except the five that have yet to be translated from the original Chinese. In the reference section all the references to studies on the brain including these 23 studies are provided in a single grouping.

Contrary to proponents' claims that this association has only been demonstrated at high fluoride levels, one of these studies indicates that IQ in children may be lowered at levels as low as 1 ppm (Xiang 2003 a, b) and another indicates that even very moderate levels of fluoride exposure (e.g. 0.9 ppm in the water) can exacerbate the neurological defects of iodine deficiency (Lin Fa-Fu 1991).

Often promoters of fluoridation see their task as merely fending of each new study that indicates harm. However, such an approach smacks of people dogmatically defending a practice rather than health professionals going about exhaustively investigating the slightest suggestion that the practice maybe causing harm. There is absolutely no a priori reason why we should expect that dosing people with a toxic substance, in uncontrolled doses, would not cause – or can never cause - harm. For instance, what rational person would dose a baby with fluoride at 250 times the level that nature intended, and not even investigate to see if any damage might have occurred. God provides no protective veil for stupid practices, no matter how well intentioned the instigators might have been.

One ugly fact can destroy a beautiful theory. It would appear that for over 60 years promoters have protected themselves against potential ugly facts by not looking for them. The answers to the next few questions should demonstrate this. There has been a huge absence of primary research studies in fluoridated countries, which have set out to examine a possible relationship between fluoridation and a number of serious health end points.

In the summer of 2008, two reports reviewed the published studies reporting an association of high fluoride exposure and reduced IQ. The fluoride levels in water in these studies range from 0.88 – 9.4 ppm.

In July 2008, Connett and Limeback presented a poster (#2205) entitled "Fluoride and its effect on human intelligence. A systematic review" at the International Association for Dental Research 83rd General Session and Exhibition. Toronto, Canada. This paper reviewed 20 papers investigating a possible connection between fluoride and lowered IQ and concluded that 18 of them showed a relationship and two did not.

In August 2008, Tang et al., published a meta analysis of 16 of the Chinese IQ studies in the journal Biol Trace Elem Res.

In addition to the studies that have reported an association between fluoride exposure and lowered IQ in children, there have been other studies from China, which add to the weight of evidence on fluoride's ability to damage the brain. These include studies which show that fluoride has damaged the brains in aborted fetuses in endemic fluorosis areas (Han et al, 1989; Du et al., 1992 and Yu et al., 1996) as well as studies which have shown behavioral

differences in adults exposed to high industrial levels of fluoride (Guo et al, 2001) and behavioral differences in neonates in endemic fluorosis areas (Li et al., 2004).

Backing up the studies on human behavior and intellectual development are a number of animal studies that have investigated the effect of fluoride on the learning, memory and behavior of animals. (Cagiano, 2007; Chioca, 2007; Sun, 2000; Zhang, 1999; Mullenix, 1995 and Wu, 1995).

While fluoridation promoters continue to ignore – or dismiss - these studies. Others are less cavalier. This is what Anna Choi and Phillipe Grandjean stated in a paper (Potentials for Developmental Fluoride Neurotoxicity) at the XXVIITH Conference of the International Society for Fluoride Research, October 9-12, 2007, Beijing China.

> "... In humans, only five substances have so far been documented as developmental neurotoxicants: lead, methylmercury, polychlorinated biphenyls, arsenic, and toluene. From this evidence, including our own studies on some of these substances, parallels may be drawn that suggest that fluoride could well belong to the same class of toxicants, but uncertainties remain. At least 200 industrial chemicals are known to cause brain toxicity in humans, mainly adults, and they must also be suspected to harm the developing brain. Because of the individual and societal importance of optimal brain function, recognition of developmental neurotoxicity is a public health priority, and further evidence on fluoride is needed."

What peer-reviewed and published studies can you, or the experts on whom you rely, cite which have examined the possible relationship between fluoride exposure and lowered IQ in <u>fluoridated</u> communities and convinced you that this is not a problem?

I am aware of only one small study in the US by Morgan et al. (1998) that looked at childhood behavior and an abstract of paper published in New Zealand. Is the SHA aware of any others?

The NRC (2006) has a whole chapter summarizing the evidence that fluoride can damage the brain (chapter 8).

Should the SHA ignore all this evidence?

What peer-reviewed and published studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and lowered thyroid function (including sub-clinical hypothyroidism) in <u>fluoridated</u> communities and convinced you that this is not a problem?

I am aware of a few studies that have investigated a possible relationship with goiter, but this is a really gross indicator of thyroid function. What needs investigating are the more subtle effects of lowered thyroid function. I am not aware of any studies in fluoridated countries that have investigated these or even sought to repeat the findings of Bachinskii et al (1986) who showed that thyroid function was altered in people drinking water containing 2.3 ppm.

In the first half of the 20th century, fluoride was prescribed by a number of European doctors to reduce the activity of the thyroid gland for those suffering from hyperthyroidism (over active thyroid) (Stecher 1960; Waldbott 1978). Moreover, the doses they used are easily reached in fluoridated communities today (Galleti and Joyet, 1958). With water fluoridation, we are forcing people to drink a thyroid-depressing medication that could, in turn, serve to promote higher levels of hypothyroidism (underactive thyroid) in the population, and all the subsequent problems related to this disorder. Such problems include depression, fatigue, weight gain, muscle and joint pains, increased cholesterol levels, and heart disease.

What makes this issue highly pertinent is that millions of people in fluoridated countries suffer from this condition, and probably millions more from pre-clinical hypothyroidism. Symptoms of sub-clinical hypothyroidism include: depression, fatigue, lethargy and obesity. In the US, in 1999, the second most prescribed drug of the year was Synthroid, which is a hormone replacement drug used to treat an underactive thyroid.

The NRC (2006) has a whole chapter on fluoride's impact on the endocrine system (see chapter 7). Can the SHA safely ignore all this evidence?

What peer-reviewed and published studies can you, or the experts on whom you rely, cite which have a) attempted to confirm the accumulation of fluoride in the human pineal gland; b) compared the level of melatonin in children living in fluoridated and non-fluoridated communities and c) examined a possible relationship between fluoride exposure and earlier onset of puberty among children in <u>fluoridated</u> communities, and convinced you that this is not a problem?

I have already mentioned above that Jennifer Luke found that fluoride accumulated in the human pineal gland (Luke, 2001) and that in animal studies she found it lowered melatonin production and shortened the time to puberty (Luke, 1997). As far as I am aware no fluoridating country has attempted to repeat either aspects of her work.

What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and bone fractures in children in <u>fluoridated</u> communities, and convinced you that this is not a problem?

Above I have already discussed the finding from one of the earliest trials of fluoridation (Newburgh versus Kingston, 1945-55) that fluoridation increased the rate of cortical bone defects. Because the cortical bone is key for the protection of fractures in bones it is important to check if there are increased rates of bone fractures in children living in fluoridated communities. I also mentioned Alarcon-Herrera et al.'s (2001) that showed a liner correlation between the severity of dental fluorosis and bone fractures. As far as I am aware no fluoridating country has attempted to repeat this work.

What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and osteosarcoma in fluoridated communities and convinced you that this is not a problem?

The possibility that fluoridation may increase the incidence of osteosarcoma (a rare but frequently fatal bone cancer) in young boys and men has a long and fascinating history. First, the "biological plausibility" of a fluoride-osteosarcoma link is widely acknowledged in the scientific literature.

Biological plausibility. The 3 key acknowledged mechanisms supporting the plausibility of a fluoride/osteosarcoma connection are:

1) The preponderance of laboratory evidence indicates that fluoride can be mutagenic when present at sufficient concentrations. Most mutagens are also carcinogens.
2) The bone is the principal site for fluoride accumulation within the body, and the rate of accumulation is increased during periods of bone development. Thus, the cells in the bone, particularly during the growth spurts, may be exposed to some of the highest fluoride concentrations in the body.

3) Fluoride is a 'mitogen' - meaning it can stimulate the proliferation of bone-forming cells (osteoblasts). Osteosarcoma is a cancer caused by an abnormal proliferation of the osteoblasts.

Hence, fluoride's ability to induce mutagenic damage in fluoride-rich environments coupled with its ability to stimulate proliferation of osteoblasts provides a compelling biological basis by which fluoride could cause, or contribute to, osteosarcoma.

According to the authors of the NRC (2006):

> "Principles of cell biology indicate that stimuli for rapid cell division increase the risks for some of the dividing cells to become malignant, either by inducing random transforming events or by unmasking malignant cells that previously were in nondividing states." (NRC, 2006, p.275)

According to Bassin (2006):

> " It is biologically plausible that fluoride affects the incidence rate of osteosarcoma, and that this effect would be strongest during periods of growth, particularly in males. First, approximately 99% of fluoride in the human body is contained in the skeleton with about 50% of the daily ingested fluoride being deposited directly into calcified tissue (bone or dentition). Second, fluoride acts as a mitogen, increasing the proliferation of osteoblasts and its uptake in bone increases during periods of rapid skeletal growth. In the young, the hydroxyapatite structure of bone mineral exists as many extremely small crystals each surrounded by an ion-rich hydration shell, providing a greater surface area for fluoride exchange to occur."

An historical overview.

Newburgh/Kingston trial. While the cortical bone defects observed in the Newburgh/Kingston trial (1945-55) did not prompt any research into any possible relationship between fluoridation and bone fractures in children (see discussion above), it did prompt some questions on a possible relationship with osteosarcoma. Dr. Caffey, the doctor who examined the X-rays of the children in this study, noted that the anatomical, gender and age distribution of these defects were remarkably similar to osteosarcoma. (Schlessinger et al., 1956; Caffey, 1955)

NAS review (1970). In 1970, an NAS review picked up on Caffey's comment and recommended that osteosarcoma rates in young males under 30 should be examined in fluoridated communities. This is what NAS author (Donald Taves) said:

> "There was an observation in the Kingston-Newburgh (Ast et al, 1956) study that was considered spurious and has never been followed up. There was a 13.5% incidence of cortical defects in bone in the fluoridated community but only 7.5% in the non-fluoridated community... Caffey (1955) noted that the age, sex, and anatomical distribution of these bone defects are `strikingly' similar to that of osteogenic sarcoma. While progression of cortical defects to malignancies has not been observed clinically, it would be important to have direct evidence that osteogenic sarcoma rates in males under 30 have not increased with fluoridation."

NTP animal study (1990). Nothing happened on the NAS recommendation until after a 1990 National Toxicological Program (NTP) cancer study on animals found a significant increase in osteosarcoma in MALE (but not female) rats exposed to fluoride (DHHS, 1991).

Commenting on these NTP findings a committee from the WHO made the following comment: "Such a (dose-dependent) trend associated with the occurrence of a rare tumour in

the tissue in which fluoride is known to accumulate cannot be casually dismissed." (WHO, 2002)

The NTP study has been the only government-sanctioned animal study to investigate if fluoride causes cancer. The initial review of this study also reported an increase in liver and oral cancers; however, all the non-bone cancers were later downgraded – with a questionable rationale - by a government-review panel (Marcus 1990). In light of the importance of this study, EPA Professional Headquarters Union has requested that Congress establish an independent review to examine the study's results (Hirzy 2000). Meanwhile, the NTP finally prompted the National Cancer Institute to review osteosarcoma rates in fluoridated communities in the US.

NCI Survey of the SEER registries for osteosarcoma. DHHS, 1991. The National Cancer Institute examined the SEER cancer registries (which cover above about 10% of the US population) for bone cancer. They found a greater increase in osteosarcoma in young males (but not for young females) in fluoridated versus non-fluoridated counties (Hoover et al, 1991 a)) However, the same authors, using a subset of the data claimed that they did not find that this increase was related to duration of exposure and discounted the original finding (Hoover et al., 1991 b). Today, more credibility is given to Hoover's first finding than his second (which supposedly discounted the first). This is largely because by the time he had used a subset of the data and divided it between 4 different age ranges of exposure, there were so few cases left in each grouping that the study lacked any statistical power.

Both Yiammouyiannis (1993) and Takahashi (2001) examined the same data base used by Hoover and found a significant increase in osteosarcoma in young males in fluoridated counties.

Cohn, 1992. In 1992, Cohn in NJ reported a significant increase in osteosarcoma in males in the fluoridated communities in three NJ counties – but again not for young females (Cohn, 1992). Most significantly Cohn suggested that there might be a time frame where young boys are particularly vulnerable to fluoride's carcinogenic effect.

Other studies. Other epidemiological studies of various sizes and quality have failed to find this relationship (Hrudy, 1990; Mahoney 1991; Freni 1992; McGuire, 1991; Gelberg, 1995; Moss, 1995). For a full review of these studies and other studies on this issue see the submission to the National Research Council by the Fluoride Action Network (Connett et al. (2005). Part 1 and Part 2).

Elise Bassin (2001). Elise Bassin is a dentist. She investigated a possible relationship between exposure to fluoride and osteosarcoma as part of her PhD thesis at the Harvard Dental School. Suspecting a possible time window of vulnerability for this problem, she examined osteosarcoma rates as a function of when the boys were exposed to fluoride (as Cohn had conjectured). In a matched case-control study, she found, in what she herself described as a "robust finding," that young boys exposed to fluoride between their 6th and 8th years (which corresponds to the mid-childhood growth spurt) had a 5 to 7 fold increased risk of succumbing to osteosarcoma by the age of 20. Her thesis was successfully defended in 2001.

Bassin's PhD thesis hidden from the scientific community. It is extraordinary that after Bassin's thesis was successfully defended in 2001, that it was neither followed up with a swift publication of her results nor any kind of statement made to warn the scientific community or the public about her findings. After all, if she was correct, a chemical being given daily to over 170 millions in their drinking water, might actually being killing people! If this finding had been made by industrial researchers on an industrial chemical and the authors had hidden the findings from government regulators they would have been in serious trouble.

Professor Chester Douglass. Professor Chester Douglass was Bassin's thesis adviser and signed off on her thesis. Clearly, he knew what she had found and knew the serious implications of her findings. However, even though he was given several opportunities to do so, Douglass neither warned his colleagues in professional meetings (e.g. a meeting organized by the British Fluoridation Society in 2002), nor the NRC panel nor his funders at the NIH (who had put over $ 1 million financing Douglass work). Instead of warnings he did the very opposite. He continued to assert that "his" work showed no significant association between fluoride and osteosarcoma. In his written comments to the NRC panel he even gave Bassin's thesis as a footnote, but without indicating that her findings contradicted what he was telling the panel.

Bassin's thesis discovered. Finally, Michael Connett, of the Fluoride Action Network, acting on a tip off, went to the Harvard Medical School Rare Books Room in January 2005 and "discovered" the "hidden" thesis. The resulting public release of this material, triggered a demand by the Environmental Working Group (www.ewg.org) for an official enquiry by the NIEHS into Douglass's behavior and a great deal of press attention to the "scandal." (See Begley, Wall Street Journal, July 22, 2005) The NIEHS gave the enquiry to Harvard. After a year, Harvard declared Douglass innocent of unethical behavior, stating that he did not "deliberately" hide Bassin's findings. Harvard has refused to provide any arguments supporting this finding despite repeated efforts to get them to do so.

Bassin (2006). Bassin's findings were finally published in May 2006 (Bassin et al, 2006). However, the same issue of the journal published a letter from Chester Douglass, downplaying the significance of her findings. It is interesting to contrast Douglass's failure to warn the public of Bassin's findings in 2001, with his need to warn the public that her findings were "premature" in 2006.

The Douglass letter. Douglass claimed that Bassin's findings were based on a subset of a larger cohort, and that the larger cohort did not support her thesis. This was strange because he provided no evidence that her methodology had, at that time, been applied to this larger cohort. Nor is it clear that it has ever been applied to the larger cohort. Douglass further claimed that his larger study (to be co-authored by Robert Hoover and Gary Whitford) would be published in the Summer of 2006. Over 2 and half years later it still has not appeared.

Douglass's methodology cannot refute Bassin. Those who have examined the methodology described by Douglass et al. have indicated that this work would fail to test the central thesis of Bassin's work (Neurath and Connett, 2008). This is because the biometric of exposure these authors are using - bone fluoride levels found at the time of diagnosis or autopsy - could not be used to ascertain exposure at the critical years (6- 8) determined by Bassin. This is because fluoride accumulates over the years – so a level say at 20 - gives no indication of the level of exposure at 6, 7 or 8. Moreover, for some bizarre reason the controls being used in the Douglass, Hoover & Whitford study are other bone cancers. Thus, this whole study would be invalidated if fluoride caused any of these other bone cancers, like Ewings Sarcoma, which is a distinct possibility.

Proponents use Douglass's letter to negate concern over the Bassin study. Despite the non-appearance of the promised Douglass et al. study and the limitations in the methodology he has used in terms of refuting Bassin's work, Douglass's letter is being used by fluoridation proponents in several countries, as if it was the final word on the issue. For those who insist on very high standards for the work they accept as evidence, using a "promise" in a letter to negate Bassin's findings is extraordinary.

This is how the Australian National Health and Medical Research Council (NHMRC) used the Douglass letter in the systematic review they published in 2007.

> "The attention of the reader is drawn to a Letter to the Editor that appeared in the same issue of Cancer Causes and Controls by co-investigators on the larger Harvard study (Douglass & Joshipura, 2006). The authors point out that they had not been able to replicate the findings of Bassin and colleagues in the larger study that included prospective cases from the same 11 hospitals. Furthermore, the bone samples that were taken in the broader study corroborate a lack of association between the fluoride content in drinking water and osteosarcoma in the new cases. As Bassin and colleagues acknowledged, the shortcomings of their study mean that their results should be interpreted with caution pending publication of the larger study results" (NHMRC, 2007, p.103)

The SHA used the Douglass letter in the following fashion in their public consultation brochure. You will notice that in this case the SHA authors do not make it clear that reference 13 is not study or "comprehensive review" but a letter promising a study!

> "Since 2006, fluoridation opponents have pointed to a study in the United States of America (12 – this is the Bassin study, PC) that appears to suggest a possible increase in osteosarcoma (bone cancer) rates in young males – but not females –living in fluoridated areas. However, this was part of a larger study (13 – this is the Douglass letter, PC) looking at many more osteosarcoma cases over a longer period of time and including an examination of bone samples. This more detailed and comprehensive review had found no link between water fluoride levels and osteosarcoma. The researchers therefore advised caution in selectively interpreting the results of the smaller study in isolation." (SHA brochure, 2008, pp 18-19)
>
> For more details on the fluoride-osteosarcoma issue see the Fluoride Action Network's submission to the National Research Council (Connett, Neurath and Connett, 2005, a and b).

What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have examined a possible relationship between fluoride exposure and arthritis in fluoridated communities and convinced you that this is not a problem?

Some of the early symptoms of skeletal fluorosis, a fluoride-induced bone and joint disease that impacts millions of people in India, China, and Africa, mimic the symptoms of arthritis (Singh 1963; Franke 1975; Teotia 1976; Carnow 1981; Czerwinski 1988; DHHS 1991). According to a review on fluoridation by Chemical & Engineering News, "Because some of the clinical symptoms mimic arthritis, the first two clinical phases of skeletal fluorosis could be easily misdiagnosed" (Hileman 1988). Few if any studies have been done to determine the extent of this misdiagnosis, and whether the high prevalence of arthritis in America (1 in 3 Americans have some form of arthritis - CDC, 2002) is related to our growing fluoride exposure, which is highly plausible. Nor has this plausible connection been investigated in any other fluoridating country. The causes of most forms of arthritis (e.g. osteoarthritis) are unknown.

What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which has convinced you that lifelong consumption of fluoridated water along with other sources of fluoride will not weaken the bones of the elderly and cause an increased rate of hip fractures, in fluoridated communities?

Hip fracture is a very serious issue for the elderly, as a quarter of those who have a hip fracture die within a year of the operation, while 50 percent never regain an independent existence. If fluoridation were to increase the rates of hip fracture in the elderly it would be very serious indeed.

In my view the best way to resolve this issue is to use a "weight of evidence" approach which avails itself with all the evidence that can brought to bear on this matter: animal studies; mathematical modeling; clinical trials in addition to epidemiological studies. The NRC (2006) did do this and concluded that bones are weakened at levels lower than 4 ppm and that some individuals, especially above average water drinkers and those with poor kidney function, might have their bones weakened drinking water fluoridated as low as 1.5 ppm.

Unfortunately, neither the York Review (McDonagh et al, 2000) nor the more recent review by the NHMRC (2007) used this approach. Instead they limited themselves to epidemiological studies that examined fracture rates close to 1 ppm.

The dangers of limiting an assessment of this problem to epidemiological studies is that one is always looking at the past and by the time these show a definitive result, it is far too late for millions of people who have already been exposed. The use of animal studies, mathematical models and clinical trials allows one a better shot of protecting future generations, i.e. before the exposure has taken place.

Clinical trials. In some studies, where high doses of fluoride (average 26 mg per day) were used in trials to treat patients with osteoporosis in an effort to harden their bones and reduce fracture rates, it actually led to a HIGHER number of fractures, particularly hip fractures (Inkovaara 1975; Gerster 1983; Dambacher 1986; O'Duffy 1986; Hedlund 1989; Bayley 1990; Gutteridge 1990. 2002; Orcel 1990; Riggs 1990 and Schnitzler 1990). The cumulative doses used in these trials are exceeded by the lifetime cumulative doses being experienced by many people living in fluoridated communities.

Animal studies. A number of studies on several different animal species have shown that their bones are weakened at fluoride bone levels reached by people living in fluoridated communities.

Epidemiological studies. At least nineteen studies (three unpublished, including one abstract) since 1990 have examined the possible relationship of fluoride in water and hip fractures among the elderly. Eleven of these studies found an association, eight did not. Thus to claim, as the SHA brochure does, that there is no evidence that hip fractures are increased in fluoridated communities is inaccurate and misleading. An accurate statement is that the studies are "mixed." An annotated list of references to all 19 of these studies is given at the end of the list of references.

There is one study among these 19, which has been used by both proponents and opponents of fluoridation. This is the study by Li et al. (2001). This is a particularly strong study, in my view, because it looked at hip fractures in six different villages in China, which had six different levels of fluoride in their well water ranging from 0.23 ppm to 8 ppm. Other than this difference, the subject populations were highly homogeneous and stable sharing similar occupations, lifestyles, diet and free of many confounding variables like other sources of fluoride and hormone replacement therapy.

Proponents of fluoridation point out that if one looks at the two villages less than 1 ppm, there is lower frequency of all fractures combined than the control village at 1 ppm. They thus argue that this is evidence for the notion that fluoride – up to a certain level - actually strengthens bones. Opponents point out that this relationship did not apply to hip fractures and that for these the frequency for villages 3, 4,5 and 6 appears to increase in an approximately linear fashion. They further argue that even though the increased frequencies for villages 4 and 5, were not statistically significant, the frequency for the village at levels between 4.3 and 8 ppm, was. As the better fit of this data is linear rather than assuming a threshold at 4.3 ppm, this argues for the results for villages 4 and 5 to be real, even if they were not statistically significant.

The frequency of hip fractures in village 4 (fluoride level = 1.5 ppm), doubled compare to the control village 3 (fluoride level = 1 ppm). The frequency tripled for village 6 (F = 4.3- 8 ppm). This

would mean that the LOAEL for this study would have to be set at 1.5 ppm, for which the actual daily dose was estimated at 6.85 mg/day – a level that is exceeded by above average water drinkers in fluoridated communities. The NOAEL was not determined but has to be assumed to be between 1 and 1.5 ppm. Clearly based on this study there is no margin of safety for this effect adequate to protect the whole population.

This finding is further buttressed by the Kurttio et al. (1999) study from Finland showed an increase hip fracture rate in communities above 1.5 ppm. Meanwhile, the Alarcon-Herrera et al. (2001) study showed a linear increase in fractures of all kinds for both children and adults as a function of the severity of dental fluorosis – a condition that affects over 30% of children in the US.

There are other potentially serious consequences of accumulating fluoride in bones over a lifetime. The long half-life of fluoride in bones (estimated to be 20 years) means that there are lifelong ramifications from ingesting fluoride, not only for the bone itself but also the surrounding fluids. If the bone turnover is increased under certain health or fasting situations it may lead to a potentially problematic peak plasma level. Similarly if fluoride input is decreased it could lead to an increased release of fluoride. The issue of half-life and turnover was discussed in the NRC (2006) report:

> ... Fluoride concentrations in plasma are not homeostatically controlled (Whitford 1996). Chronic dosing leads to accumulation in bone and plasma (although it might not always be detectable in plasma.) Subsequent decreases in exposure cause fluoride to move back out of bone into body fluids, becoming subject to the same kinetics as newly absorbed fluoride. A study of Swiss aluminum workers found that fluoride bone concentrations decreased by 50% after 20 years. The average bone ash concentration in the workers was about 6,400 mg/kg at the end of exposure, estimated via regression (Baud et al. 1978). The bone concentration found in these workers is similar to that found in long-term consumers of drinking water containing fluoride in the range of 2-4 mg/L (discussed later in this chapter). Twenty years might not represent a true half-life. Recent pharmacokinetic models (see below) are nonlinear, suggesting that elimination rates might be concentration dependent (page 92).

What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have surveyed the population in the UK or any other fluoridated country, in a comprehensive fashion for the level of fluoride in their bones as a function of age, fluoridation status and other variables?

The fluoridation program has been very poorly monitored. There has never been a comprehensive analysis of the fluoride levels in the bones, blood, or urine of the American people or the citizens of other fluoridated countries. Based on the sparse data that has become available, however, it is increasingly evident that some people in the population – particularly people with kidney disease - are accumulating fluoride levels that have been associated with harm to both animals and humans, particularly harm to bone.

When my son and I investigated the literature to see how many human bone samples had been measured for fluoride for citizens living in fluoridated communities, we found less than 2000 measurements worldwide. Essentially, we are flying blind on a key measure of fluoride exposure in fluoridated communities. Without collecting such data epidemiologic studies on bone are highly limited.

What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have surveyed the population in the UK or any other fluoridated country, in

a comprehensive fashion for the level of fluoride in their urine, as a function of age, diet, fluoridation status and other variables?

> Measuring fluoride in urine is a much easier task than measuring fluoride in bone, and yet even this simple parameter of exposure – and possibly over-exposure is not being measured on a routing basis in fluoridated countries. Where independent researchers like Dr. Peter Mansfield have made measurements, the results in the UK are very disturbing. The urine levels are very high in the UK. Thus, even though UK only has 10% of the population fluoridated there is evidence that many people are being over-exposed to fluoride. This issue needs resolving as a matter of some urgency before more widespread fluoridation is introduced into Britain.

What peer-reviewed and published PRIMARY studies can you, or the experts on whom you rely, cite which have used the severity of dental fluorosis as a biomarker for epidemiological studies on children?

> It is well known that the severity of dental fluorosis indicates the level of over-exposure to fluoride to children prior to the eruption of their secondary teeth. Thus this presents an ideal – and OBVIOUS - biometric of exposure for epidemiological studies on children. I am not aware of any studies in fluoridated countries, with the single exception of Morgan et al (date) to have done this. The omission of such obvious studies is easier to explain on the basis that health authorities do want to find a problem with this program, than with the assumption that those in charge of overseeing our children's health are so grossly incompetent.
>
> This biometric has been used in studies from Mexico (Alarcon-Herrera et al., 2001) and China.

Are you satisfied that over the 60 year history of fluoridation that sufficient effort has been made by the governments which promote this practice, to investigate possible health effects (in tissues other than the teeth) in <u>fluoridated</u> communities?

> It is hard to believe that any person independent of the promotion of this practice could answer yes to this question. This is what Dr. John Doull, the chairman of the NRC (2006) report stated in an interview published in the January, 2008 issue of Scientific American, 2008:
>
> > "What the committee found is that we've gone with the status quo regarding fluoride for many years - for too long really - and now we need to take a fresh look … "In the scientific community people tend to think this is settled. I mean, when the US surgeon general comes out and says this is one of the top 10 greatest achievements of the 20th century, that's a hard hurdle to get over. "But when we looked at the studies that have been done, we found that many of these questions are unsettled and we have much less information than we should, considering how long this [fluoridation] has been going on."

This what Professor Trevor Sheldon, the chair of the Advisory Group for the York Review and the founding director of the NHS Centre for Reviews and Dissemination the University of York, said earlier in a letter to the House of Lords (3/1/2001),

> > "The review team was surprised that in spite of the large number of studies carried out over several decades there is a dearth of reliable evidence with which to inform policy. Until high quality studies are undertaken providing more definite evidence, there will continue to be legitimate scientific controversy over the likely effects and costs of water fluoridation."

Thus it is clear, to at least independent observers, that the fluoridation program lurches on from year to year without a convincing scientific demonstration of either its effectiveness or safety or

even an effort to investigate the matter in a scientific fashion. Instead of science in fluoridated countries we get promotion via a long list of endorsements, from associations and agencies, which are not on top of the primary literature and who take the word of government agencies on this issue, at face value. Meanwhile, these government agencies appear to have no interest in financing genuine scientific studies that could resolve some of the issues of concern. Not only is the practice of fluoridation a giant experiment, but also those who are conducting the experiment are not even collecting the data!

If this program moves forward are any health studies planned for the local community?

> I cannot say for certain what the answer to this question will be, but based upon the long history of this practice, the answer is probably no.

If this program moves forward will any compensation be given to children who develop very mild, mild, moderate or severe dental fluorosis? Will they be provided with free treatment for these conditions if desired?

> I cannot say for certain what the answer to these questions will be, but based upon the long history of this practice, the answer to both is probably no.

If this program moves forward and some citizens complain of reversible symptoms, which elsewhere have been identified as being caused by fluoride (i.e. they cease when the source of fluoride is removed), will any steps be taken to investigate the matter scientifically?

> I cannot say for certain what the answer to this question will be, but based upon the long history of this practice, the answer is probably no.

Please discuss what you understand by the <u>Precautionary Principle.</u> Do you believe that in the context of the current scientific uncertainties about fluoridation's effectiveness and safety, and the availability of alternatives, that fluoridation is consistent with the Precautionary Principle?

> The Precautionary Principle (PP) has come into play in Europe because it has been found in the case of the use of a number of harmful substances or practices that by the time it takes to get definitive scientific proof that the chemical or practice has caused harm, sufficient to withstand the assault of invested interests, it is too late for the millions whose health has been damaged irreversibly. This was the case with lead, benzene, asbestos and smoking. The PP acknowledges this problem and posits the notion that when there is reasonable doubt of the safety of substance or practice, we should err on the side of caution. Put simply, "if in doubt, leave it out."
>
> Clearly, unless the PP is to stymie all industrial progress, certain criteria should be met before the PP is invoked. I offer the following criteria to be considered in the case of water fluoridation:

1) Is the evidence of harm plausible and supported by a number of peer reviewed published studies?
2) If the harm is real is it serious? Are the effects reversible?
3) How good is the evidence that the benefit being sort is real?
4) How significant are the consequences if the practice is halted?
5) Are there cost effective alternatives to the practice?

I offer my answers below – in brackets.

1) Is the evidence of harm plausible (YES) and supported by a number of peer reviewed published studies? (YES)
2) If the harm is real is it serious? (YES) Are the effects reversible? (MANY ARE NOT)
3) How good is the evidence that the benefit being sort is real? (WEAK, AND WEAKER THAN THE EVIDENCE OF HARM)
4) How significant are the consequences if the practice is halted? (NOT VERY)
5) Are there cost effective alternatives to the practice? (YES)

I think careful answers to each of these questions reveals very clearly that fluoridation should be stopped based on the Precautionary Principle.

Normally governments only use their police power to enforce medication on people when they are dealing with a life threatening contagious disease. Do you believe that this is the situation that confronts Southampton with respect to current dental decay levels?

Tooth decay is not contagious in the larger community sense. It is possible for a mother to give to the baby streptococcus mutans, the bacteria which increases tooth decay by converting sugar into acids which attack the enamel, but it is hardly likely to spread to the larger community via this mechanism. Moreover, were it to do so, it would not lead to widespread deaths. In other words, water fluoridation is an unwarranted use of community or governmental police power, especially when it has been shown that in many European countries, as well as many towns in the UK, tooth decay can be reduced by alternatives that do not involve such use of police power.

Bearing in mind your responses to all of the above, are you convinced that the evidence of benefit from this practice is so strong, and the evidence of harm so weak, that it merits the application of governmental police power to force this practice on your citizens regardless of their views on the matter?

Again it is hard to believe that any rational person – outside those who actively promote fluoridation - could actually believe this.

Could you summarize the evidence that has convinced you that there are no extra problems associated with using hexafluorosilicic acid (an industrial waste product) as a fluoridating chemical as opposed to pharmaceutical grade sodium fluoride?

The chemicals used to fluoridate water in the US are not pharmaceutical grade. Instead, they come from the wet scrubbing systems of the superphosphate fertilizer industry. These chemicals (90% of which are sodium fluorosilicate and fluorosilicic acid) are classified hazardous wastes contaminated with various impurities.

These hazardous waste products are not allowed to be dumped into the sea by international law, nor can they be dumped locally because they are too concentrated. Once they are purchased by water departments they no longer have to meet the stringent legal requirements for handling hazardous waste in the US.

There is no question that these fluoridating agents are contaminated with other toxic contaminants. However, proponents claimed that by the time they are diluted by about 200,000 to 1 (to reach a concentration level for fluoride of 1 ppm), the contaminant levels would be below regulatory concern. However, this may not be true of arsenic. Recent testing by the National Sanitation Foundation suggest that the levels of arsenic in these chemicals are relatively high (up to 1.6 ppb after dilution into public water) and of potential concern (NSF 2000 and Wang 2000). There is also a concern about radioactive isotope levels. It should be noted that the same phosphate rock used by the superphosphate fertilizer industry has been used for mining uranium.

These hazardous wastes have not been tested comprehensively. The chemical usually tested in animal studies is pharmaceutical grade sodium fluoride, not industrial grade fluorosilicic acid.

Studies by Masters and Coplan (1999, 2000) show an association between the use of fluorosilicic acid (and its sodium salt) to fluoridate water and an increased uptake of lead into children's blood. Because of lead's acknowledged ability to damage the child's developing brain, this is a very serious finding yet it is being largely ignored by fluoridating countries.

More recent studies by Maas et al. (2006) indicate that fluoridating chemicals alone, and in conjunction with other chemicals added to water like chloramines, have the ability to increase the leaching of lead from brass fittings.

If you are convinced that children's teeth in Southampton will benefit from ingesting fluoridated water please compare these two delivery systems: 1) adding contaminated hexafluorosilicic acid to the public water supply and 2) making fluoridated bottled water available in local supermarkets and chemists, and free for families of low income.

Providing fluoridated water in supermarkets and chemists would have the following very clear advantages to adding fluoride to the public water supply:

a) It would allow the use of pharmaceutical grade sodium fluoride rather than an industrial grade chemical.
b) It would allow a much closer control over dose. 1 liter bottles of water fluoridated at 1 ppm fluoride could be provided and customers told to use only one liter of this and to use other water uses for any additional needs.
c) It would mean that you would not be forcing this on people who don't want it and/or don't need it.
d) People on low income could be provided this water for free.
e) It would drastically reduce the amount of toxic chemicals put into the environment. Only a small portion of the fluoride added to the public water supply goes anywhere near the teeth, most of it ends up flushing the toilet, cleaning the dishes, washing the car and watering the garden. All of this fluoride eventually ends up in the environment.

Are you prepared to do the whole community what an individual doctor is not allowed to do his or her individual patients: i.e. override <u>the individual's right to informed consent to medication?</u>

Clearly, many decision makers in the handful of countries, which practice fluoridation, are prepared to do this, but most countries are not. See the list of explanations offered why most European countries do not force fluoridation on their populations (see appendix 2).

Fluoridation defies one of the key mandates of medical practice. As Dr. Peter Mansfield, one of the advisory board members for the York Review stated:

> "No physician in his right senses would prescribe for a person he has never met, whose medical history he does not know, a substance which is intended to create bodily change, with the advice: 'Take as much as you like, but you will take it for the rest of your life because some children suffer from tooth decay.' It is a preposterous notion."

Proponents counteract such concerns by stressing how well they can control and monitor the CONCENTRATION of the fluoridating agent added to the water. However, as I pointed out several times at the public "Question Times" controlling concentration (mg/liter) is not the same

as controlling dose (mg/day). Once fluoride is put in the water it is impossible to control the dose each individual receives. This is because 1) some people (e.g. manual laborers, athletes, diabetics, and people with kidney disease) drink more water than others, and 2) we receive fluoride from sources other than the water supply.

As Arvid Carlsson pointed out as long ago as 1978:

> "I am quite convinced that water fluoridation, in a not-too-distant future, will be consigned to medical history...Water fluoridation goes against leading principles of pharmacotherapy, which is progressing from a stereotyped medication - of the type 1 tablet 3 times a day - to a much more individualized therapy as regards both dosage and selection of drugs. The addition of drugs to the drinking water means exactly the opposite of an individualized therapy"

The Union representing the scientists at US EPA headquarters in Washington DC is on record as opposing water fluoridation (Hirzy 1999). According to the Union's Senior Vice President, Dr. William Hirzy:

> "In summary, we hold that fluoridation is an unreasonable risk. That is, the toxicity of fluoride is so great and the purported benefits associated with it are so small - if there are any at all - that requiring every man, woman and child in America to ingest it borders on criminal behavior on the part of governments."

Conclusion.

In my view, both individually and collectively, the answers I have posed to the SHA and have addressed above reveal that fluoridation is unnecessary, unethical, ineffective and poses serious and significant health dangers.

At the very least there is a totally inadequate margin of safety to protect everyone in the population from the health effects revealed at higher doses around the world, and recorded in the National Research Council 2006 report "Fluoride in Drinking Water." Moreover, that argument has received massive augmentation since the NRC report was published in the form of 17 more studies than the NRC considered on fluoride exposure and lowered IQ in children.

The only way that fluoridating countries have been able to deny the health effects of fluoridation is that they simply do not conduct the relevant studies in their own countries, and instead use their time and skills to denigrate the studies – or the authors of the studies - done elsewhere. More or less the same tactics were used by both the lead industry and the tobacco industry to delay the day of reckoning on their products.

If one compares the number and quality of the studies that purport to demonstrate the benefits of fluoridation with the number and quality of the studies that purport to demonstrate its dangers, the case is overwhelming in favor of ending this practice. Just how much doubt is needed on just one of the health concerns identified above, to override a benefit, which when quantified in the largest survey ever conducted in the US (Brunelle and Carlos, 1990), amounts to less than one tooth surface (out of 128) in a child's mouth?

A simple application of the precautionary principle would make this practice unthinkable and makes the willingness of those in authority who would force it onto individuals without their informed consent the most glaring example of governmental arrogance imaginable.

When it comes to controversies surrounding toxic chemicals, invested interests traditionally do there very best to discount animal studies and quibble with epidemiological findings. We see these same tactics being used to defend fluoridation today.

In the past, political pressures have led government agencies to drag their feet on regulating asbestos, benzene, DDT, PCBs, tetraethyl lead, tobacco and dioxins. With fluoridation we have had a fifty-year delay.

Unfortunately, because government officials have put so much of their credibility on the line defending fluoridation, and because of the huge liabilities waiting in the wings if they admit that fluoridation has caused an increase in hip fracture, arthritis, bone cancer, brain disorders or thyroid problems, it will be very difficult for them to speak honestly and openly about the issue. But they must, not only to protect millions of people from unnecessary harm, but also to protect the notion that, at its core, public health policy must be based on sound science not political expediency.

For those who would call for further studies, I say fine. Take the fluoride out of the water first and then conduct all the studies you want. Again I would stress that at least 5 modern studies have shown that when fluoride is removed from the water tooth decay has not gone up.

For anyone not simply obeying orders, and who reads the scientific literature, it should be clear that the folly of fluoridation must end without further delay.

APPENDIX 1. World Health Organization Data

DMFT (Decayed, Missing & Filled teeth) Status for 12 year olds by Country

	DMFTs	Year	Status*
Australia	0.8	1998	More than 50% of water is fluoridated
Zurich, Switzerland	0.84	1998	Water is unfluoridated, but salt is fluoridated
Netherlands	0.9	1992-93	No water fluoridation or salt fluoridation
Sweden	0.9	1999	No water fluoridation or salt fluoridation
Denmark	0.9	2001	No water fluoridation or salt fluoridation
UK (England & Wales)	0.9	1996-97	11% of water supplies are fluoridated
Ireland	1.1	1997	More than 50% of water is fluoridated
Finland	1.1	1997	No water fluoridation or salt fluoridation
Germany	1.2	2000	No water fluoridation, but salt fluoridation is common
US	1.4	1988-91	More than 50% of water is fluoridated
Norway	1.5	1998	No water fluoridation or salt fluoridation
Iceland	1.5	1996	No water fluoridation or salt fluoridation
New Zealand	1.5	1993	More than 50% of water is fluoridated
Belgium	1.6	1998	No water fluoridation, but salt fluoridation is common
Austria	1.7	1997	No water fluoridation, but salt fluoridation is common
France	1.9	1998	No water fluoridation, but salt fluoridation is common

Data from WHO Oral Health Country/Area Profile Programme Department of Noncommunicable Diseases Surveillance/Oral Health WHO Collaborating Centre, Malmö University, Sweden http://www.whocollab.od.mah.se/euro.html

APPENDIX 2. Statements on fluoridation by governmental officials from several countries

Germany: "Generally, in Germany fluoridation of drinking water is forbidden. The relevant German law allows exceptions to the fluoridation ban on application. The argumentation of the Federal Ministry of Health against a general permission of fluoridation of drinking water is the problematic nature of compuls[ory] medication." (Gerda Hankel-Khan, Embassy of Federal Republic of Germany, September 16, 1999). http://www.fluoridealert.org/germany.jpeg

France: "Fluoride chemicals are not included in the list [of 'chemicals for drinking water treatment']. This is due to ethical as well as medical considerations." (Louis Sanchez, Directeur de la Protection de l'Environment, August 25, 2000). http://www.fluoridealert.org/france.jpeg

Belgium: "This water treatment has never been of use in Belgium and will never be (we hope so) into the future. The main reason for that is the fundamental position of the drinking water sector that it is not its task to deliver medicinal treatment to people. This is the sole responsibility of health services." (Chr. Legros, Directeur, Belgaqua, Brussels, Belgium, February 28, 2000). http://www.fluoridation.com/c-belgium.htm

Luxembourg: "Fluoride has never been added to the public water supplies in Luxembourg. In our views, the drinking water isn't the suitable way for medicinal treatment and that people needing an addition of fluoride can decide by their own to use the most appropriate way, like the intake of fluoride tablets, to cover their [daily] needs." (Jean-Marie RIES, Head, Water Department, Administration De L'Environment, May 3, 2000). http://www.fluoridealert.org/luxembourg.jpeg

Finland: "We do not favor or recommend fluoridation of drinking water. There are better ways of providing the fluoride our teeth need." (Paavo Poteri, Acting Managing Director, Helsinki Water, Finland, February 7, 2000). http://www.fluoridation.com/c-finland.htm

"Artificial fluoridation of drinking water supplies has been practiced in Finland only in one town, Kuopio, situated in eastern Finland and with a population of about 80,000 people (1.6% of the Finnish population). Fluoridation started in 1959 and finished in 1992 as a result of the resistance of local population. The most usual grounds for the resistance presented in this context were an individual's right to drinking water without additional chemicals used for the medication of limited population groups. A concept of "force-feeding" was also mentioned.

Drinking water fluoridation is not prohibited in Finland but no municipalities have turned out to be willing to practice it. Water suppliers, naturally, have always been against dosing of fluoride chemicals into water." (Leena Hiisvirta, M.Sc., Chief Engineer, Ministry of Social Affairs and Health, Finland, January 12, 1996.) http://www.fluoridealert.org/finland.jpeg

Denmark: "We are pleased to inform you that according to the Danish Ministry of Environment and Energy, toxic fluorides have never been added to the public water supplies. Consequently, no Danish city has ever been fluoridated." (Klaus Werner, Royal Danish Embassy, Washington DC, December 22, 1999). http://www.fluoridation.com/c-denmark.htm

Norway: "In Norway we had a rather intense discussion on this subject some 20 years ago, and the conclusion was that drinking water should not be fluoridated." (Truls Krogh & Toril Hofshagen, Folkehelsa Statens institutt for folkeheise (National Institute of Public Health) Oslo, Norway, March 1, 2000). http://www.fluoridation.com/c-norway.htm

Sweden: "Drinking water fluoridation is not allowed in Sweden...New scientific documentation or changes in dental health situation that could alter the conclusions of the Commission have not been shown." (Gunnar Guzikowski, Chief Government Inspector, Livsmedels Verket -- National Food Administration Drinking Water Division, Sweden, February 28, 2000). http://www.fluoridation.com/c-sweden.htm

Netherlands: "From the end of the 1960s until the beginning of the 1970s drinking water in various places in the Netherlands was fluoridated to prevent caries. However, in its judgment of 22 June 1973 in case No. 10683 (Budding and co. versus the City of Amsterdam) the Supreme Court (Hoge Road) ruled there was no legal basis for fluoridation. After that judgment, amendment to the Water Supply Act was prepared to provide a legal basis for fluoridation. During the process it became clear that there was not enough support from Parlement [sic] for this

amendment and the proposal was withdrawn." (Wilfred Reinhold, Legal Advisor, Directorate Drinking Water, Netherlands, January 15, 2000). http://www.fluoridation.com/c-netherlands.htm

Northern Ireland: "The water supply in Northern Ireland has never been artificially fluoridated except in 2 small localities where fluoride was added to the water for about 30 years up to last year. Fluoridation ceased at these locations for operational reasons. At this time, there are no plans to commence fluoridation of water supplies in Northern Ireland." (C.J. Grimes, Department for Regional Development, Belfast, November 6, 2000). http://www.fluoridealert.org/Northern-Ireland.jpeg

Austria: "Toxic fluorides have never been added to the public water supplies in Austria." (M. Eisenhut, Head of Water Department, Osterreichische Yereinigung fur das Gas-und Wasserfach Schubertring 14, A-1015 Wien, Austria, February 17, 2000). http://www.fluoridation.com/c-austria.htm

Czech Republic: "Since 1993, drinking water has not been treated with fluoride in public water supplies throughout the Czech Republic. Although fluoridation of drinking water has not actually been proscribed it is not under consideration because this form of supplementation is considered as follows:

(a) uneconomical (only 0.54% of water suitable for drinking is used as such; the remainder is employed for hygiene etc. Furthermore, an increasing amount of consumers (particularly children) are using bottled water for drinking (underground water usually with fluor)

(b) unecological (environmental load by a foreign substance)

(c) unethical ("forced medication")

(d) toxicologically and physiologically debatable (fluoridation represents an untargeted form of supplementation which disregards actual individual intake and requirements and may lead to excessive health-threatening intake in certain population groups; [and] complexation of fluor in water into non biological active forms of fluor." (Dr. B. Havlik, Ministerstvo Zdravotnictvi Ceske Republiky, October 14, 1999). http://www.fluoridealert.org/czech.jpeg

APPENDIX 3. Statement of Douglas Carnall, Associate Editor of the British Medical Journal, published on the BMJ website (http://www.bmj.com) on the day that the BMJ published the York Review on Fluoridation (McDonagh et al., 2000).

See this review on the web at http://bmj.bmjjournals.com/cgi/content/full/321/7265/904/a

British Medical Journal, October 7, 2000, Reviews, Website of the week: Water fluoridation

Fluoridation was a controversial topic even before Kubrick's Base Commander Ripper railed against "the international communist conspiracy to sap and impurify all of our precious bodily fluids" in the 1964 film Dr Strangelove. This week's BMJ shouldn't precipitate a global holocaust, but it does seem that Base Commander Ripper may have had a point. The systematic review published this week (p 855) shows that much of the evidence for fluoridation was derived from low quality studies, that its benefits may have been overstated, and that the risk to benefit ratio for the development of the commonest side effect (dental fluorosis, or mottling of the teeth) is rather high.

Supplementary materials are available on the BMJ 's website and on that of the review's authors, enhancing the validity of the conclusions through transparency of process. For example, the "frequently asked questions" page of the site explains who comprised the advisory panel and how they were chosen ("balanced to include those for and against, as well as those who are neutral"), and the site includes the minutes of their meetings. You can also pick up all 279 references in

Word97 format, and tables of data in PDF. Such transparency is admirable and can only encourage rationality of debate.

Professionals who propose compulsory preventive measures for a whole population have a different weight of responsibility on their shoulders than those who respond to the requests of individuals for help. Previously neutral on the issue, I am now persuaded by the arguments that those who wish to take fluoride (like me) had better get it from toothpaste rather than the water supply (see www.derweb.co.uk/bfs/index.html and www.npwa.freeserve.co.uk/index.html for the two viewpoints).

Douglas Carnall
Associate Editor
British Medical Journal

APPENDIX 4. List of 14 Noble Prize winners who have opposed or expressed reservations about fluoridation.

1) Adolf Butenandt (Chemistry, 1939)
2) Arvid Carlsson (Medicine, 2000)
3) Hans von Euler-Chelpin (Chemistry, 1929).
4) Walter Rudolf Hess (Medicine, 1949)
5) Corneille Jean-François Heymans (Medicine, 1938)
6) Sir Cyril Norman Hinshelwood (Chemistry, 1956)
7) Joshua Lederberg (Medicine, 1958)
8) William P. Murphy (Medicine, 1934)
8) Giulio Natta (1963 Nobel Prize in Chemistry)
10) Sir Robert Robinson (Chemistry, 1947)
11) Nikolai Semenov (Chemistry, 1956)
12) James B. Sumner (Chemistry, 1946)
13) Hugo Theorell (Medicine, 1955)
14) Artturi Virtanen (Chemistry, 1945)

APPENDIX 5. Over 2000 Professionals call for an end to fluoridation worldwide. See http://www.FluorideAlert.org/professionals.statement.html

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(8) Wang G, Yang D, Jia, Wang H. (1996). A study of the IQ levels of four- to seven-year-old children in high fluoride areas. Endemic Diseases Bulletin, Vol. 11, No. 1, 60-6. February 1996.

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5 CHINESE STUDIES NOT YET TRANSLATED INTO ENGLISH:

The Tang et.al. study cited the following 5 studies as reporting lowered IQ from fluoride exposure. (FAN intends to translate these papers into English.)

(19) Title: Effect of high level of fluoride on children's intelligence. Authors: An JA, Mei SZ, Liu AP et al. Published in: Zhong Guo Di Fang Bing Fang Zhi Za Zhi 7(2):93–94. 1992. (in Chinese)

(20) Title: Effect of fluoride on children's intelligence. Authors: Xu YL, Lu CS, Zhang XN Published in: Di Fang Bing Tong Bao 9:83–84. 1994. (in Chinese)

(21) Title: Comparison of children's health and intelligence between the fluorosis area with altering water source and those without altering water source.

Authors: Yao LM, Deng Y, Yang SY et al (1997) Yu Fang Yi Xue Wen
Published in: Yu Fang Yi Xue Wen Xian Xin Xi 3(1):42–43. 1997. (in Chinese)

(22) Title: Effect of high level of fluoride and arsenium on children's intelligence. Authors: Zhang JW, Yao H, Chen Y. Zhong Guo Gong Gong Wei Sheng Xue Bao 17(2):119. 1998. (in Chinese)

(23) Title: Effect of high fluoride exposure on children intelligence.
Authors: Fan ZX, Dai HX, Bai AM et al. Published by: Huan Jing Yu Jian Kang Za Zhi 24(10):802–803. 2007. (in Chinese)

A meta-analysis of 16 Chinese IQ studies.

Tang Q-Q, Du J, Ma H-H, Jiang S-J, Zhou X-J.(2008) Fluoride and Children's Intelligence: A Meta-analysis. Biol Trace Elem Res. 2008 Aug 10. 2008.

2 CHINESE STUDIES that Connett & Limeback did not list as finding an association between fluoride exposure and reduced IQ:

Hu Y, Yu Z , Ding R. (1989) Research on the intellectual ability of 6-14 year old students in an area with endemic fluoride poisoning. Collection of papers and abstracts of 4th China Fluoride Research Association 6:73. 1989. (English translation not yet published)

Yang Y, Wang X, Guo X, Hu P. (1994) The effects of high levels of fluoride and iodine on child intellectual ability and the metabolism of fluoride and iodine. (English translation not yet published)

ANIMAL STUDIES – FLUORIDE'S EFFECT on LEARNING/MEMORY/BEHAVIOR:

Cagiano R, et al. (2007). Neurofunctional effects of developmental sodium fluoride exposure in rats. European Review for Medical and Pharmacological Sciences 11(4):211-24. [Pdf of Study]

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Sun ZR, et al. (2000). Effects of high fluoride drinking water on the cerebral functions of mice. Chinese Journal of Epidemiology 19: 262-263. [Chinese version | English translation]

Zhang Z, et al. (1999). [Effect of fluoride exposure on synaptic structure of brain areas related to learning-memory in mice] [Article in Chinese]. Journal of Hygiene Research 28(4):210-2. [Chinese version | English translation]

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Wu N, et al. (1995). Research on the abnormal behavior of rats exposed to fluoride. Chinese Journal of Control of Endemic Diseases 14(5):271. [Chinese version | English translation]

The 19 studies on the possible association of hip fracture and fluoridated-water.

a) Studies Reporting an Association between fluoridated water (1 ppm fluoride) & hip fracture.

1 a) Cooper C, et al. (1990). Water fluoride concentration and fracture of the proximal femur. Journal of Epidemiology and Community Health 44: 17-19.

1 b) Cooper C, et al. (1991). Water fluoridation and hip fracture. JAMA 266: 513-514 (letter, a reanalysis of data presented in 1990 paper).

2) Danielson C, et al. (1992). Hip fractures and fluoridation in Utah's elderly population. Journal of the American Medical Association 268: 746-748.

3) Hegmann KT, et al. (2000). The Effects of Fluoridation on Degenerative Joint Disease (DJD) and Hip Fractures. Abstract #71, of the 33rd Annual Meeting of the Society For Epidemiological research, June 15-17, 2000. Published in a Supplement of American Journal of Epidemiology P. S18.

4) Jacobsen SJ, et al. (1992). The association between water fluoridation and hip fracture among white women and men aged 65 years and older; a national ecologic study." Annals of Epidemiology 2: 617-626.

5) Jacobsen SJ, et al. (1990). Regional variation in the incidence of hip fracture: US white women aged 65 years and olders. JAMA 264(4): 500-2.

6 a) Jacqmin-Gadda H, et al. (1995). Fluorine concentration in drinking water and fractures in the elderly. JAMA 273: 775-776 (letter).

6 b) Jacqmin-Gadda H, et al. (1998). Risk factors for fractures in the elderly. Epidemiology 9(4): 417-423. (An elaboration of the 1995 study referred to in the JAMA letter).

7) Keller C. (1991) Fluorides in drinking water. Unpublished results. Discussed in Gordon, S.L. and Corbin, S.B,(1992) Summary of Workshop on Drinking Water Fluoride Influence on Hip Fracture on Bone Health. Osteoporosis International 2: 109-117.

8) Kurttio PN, et al. (1999). Exposure to natural fluoride in well water and hip fracture: A cohort analysis in Finland. American Journal of Epidemiology 150(8): 817-824.

9) May DS, Wilson MG. (1992). Hip fractures in relation to water fluoridation: an ecologic analysis. Unpublished data, discussed in Gordon SL, and Corbin SB. (1992). Summary of Workshop on Drinking Water Fluoride Influence on Hip Fracture on Bone Health. Osteoporosis International 2:109-117.

b) Studies reporting an association between water-fluoride levels higher than fluoridated water (4 ppm+) & hip fracture.

Li Y, et al. (2001). Effect of long-term exposure to fluoride in drinking water on risks of bone fractures. Journal of Bone and Mineral Research 16: 932-9.

Sowers M, et al. (1991). A prospective study of bone mineral content and fracture in communities with differential fluoride exposure. American Journal of Epidemiology 133: 649-660.

c) Studies Reporting No Association between water fluoride & hip fracture:

(Note that in 4 of these 8 studies, an association was actually found between fluoride and some form of fracture – e.g. wrist and hip. See notes and quotes below.)

Cauley J. et al. (1995). Effects of fluoridated drinking water on bone mass and fractures: the study of osteoporotic fractures. Journal of Bone and Mineral Research 10: 1076-86.

Feskanich D, et al. (1998). Use of toenail fluoride levels as an indicator for the risk of hip and forearm fractures in women. Epidemiology 9: 412-6.

While this study didn't find an association between water fluoride and hip fracture, it did find an association - albeit non-significant 1.6 (0.8-3.1) - between fluoride exposure and elevated rates of forearm fracture.

Hillier S, et al. (2000). Fluoride in drinking water and risk of hip fracture in the UK: a case control study. The Lancet 335: 265-2690.

Jacobsen SJ, et al. (1993). Hip Fracture Incidence Before and After the Fluoridation of the Public Water Supply, Rochester, Minnesota. American Journal of Public Health 83: 743-745.

Karagas MR, et al. (1996). Patterns of Fracture among the United States Elderly: Geographic and Fluoride Effects. Annals of Epidemiology 6: 209-216.

As with Feskanich (1998) this study didn't find an association between fluoridation & hip fracture, but it did find an association between fluoridation and distal forearm fracture, as well as proximal humerus fracture. "Independent of geographic effects, men in fluoridated areas had modestly higher rates of fractures of the distal forearm and proximal humerus than did men in nonfluoridated areas."

Lehmann R, et al. (1998). Drinking Water Fluoridation: Bone Mineral Density and Hip Fracture Incidence. Bone 22: 273-278.

Phipps KR, et al. (2000). Community water fluoridation, bone mineral density and fractures: prospective study of effects in older women. British Medical Journal 321: 860-4.

As with Feskanich (1998) and Karagas (1996), this study didn't find an association between water fluoride & hip fracture, but it did find an association between water fluoride and other types of fracture - in this case, wrist fracture. "There was a non-significant trend toward an increased risk of wrist fracture."

Suarez-Almazor M, et al. (1993). The fluoridation of drinking water and hip fracture hospitalization rates in two Canadian communities. American Journal of Public Health 83: 689-693.

While the authors of this study conclude there is no association between fluoridation and hip fracture, their own data reveals a statistically significant increase in hip fracture for men living in the fluoridated area. According to the authors, "although a statistically significant increase in the risk of hip fracture was observed among Edmonton men, this increase was relatively small (RR=1.12)."