A SCIENTIFIC CRITIQUE OF THE FLUORIDATION FORUM REPORT, IRELAND 2002.

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Section 1.

Executive Summary

The undersigned scientists have been asked to review the Fluoridation Forum report published on September 10, 2002.

In our view, the report fails to provide a proper scientific review of the many health concerns raised about the practice of water fluoridation in Ireland, and elsewhere. Out of a total of 295 pages, only 17 pages (pp. 108-124) are devoted to health issues other than dental fluorosis. Of these, a heavy reliance is placed on reviewing "other reviews" some of which are dated. Incredibly, for a study which took two years, only 2 pages (pp. 122-3) are devoted to an independent analysis of specific health studies.

The report:

1) Fails to address important studies presented by two of us (Limeback and Connett) in testimony (e.g. the accumulationof fluoride in the pineal gland, Luke, 2001).

2) Fails to address many other important studies in the recent literature, including the studies of Masters and Coplan (1999, 2000), who have showed that the fluoridating reagent (hexafluorosilicic acid) used in Ireland has never been tested in animal testing and has been associated with increased levels of lead in children's blood as well as to an increase in violent behavior. The Forum's claim that this untested industrial grade fluoridating agent replaced sodium fluoride for technical reasons is invalid.

3) Fails to take a comprehensive look at the single health issue that it did examine (hip fractures). The Forum ignored the important of work of Li et al (2001) and Alarcon-Herrera et al (2001), and failed to acknowledge the significance of important clinical studies.

4) Demonstrates a weakness in their understanding of the basics of toxicology. For example, in their derivation of Tolerable Daily Intakes (TDI), the Forum made three mistakes. The authors failed to use the most sensitive endpoint of fluoride's toxicity (Varner et al, 1998). They failed to use an uncertainty factor for determining a TDI for children under 8 years of age, and they reported a TDI for children over 8 years of age which is twice the acutely toxic -- possibly lethal -- dose!

5) Fails to establish a significant clincial difference in dental decay between children living in fluoridated and non-fluoridated communities in Ireland and throughout largely non-fluoridated Europe. The Forum also failed to acknowledge the several modern studies which have found that dental decay has not increased when fluoridation has been halted in communities in Finland, Cuba, former East Germany and Canada (Maupome et al, 2001; Kunzel and Fischer, 1997, 2000; Kunzel et al, 2000 and Seppa et al, 2000).

6) Fails to deal convincingly with the issue of dental fluorosis, making several unsupportable assertions.

7) Fails to discuss the fact that certain individuals in a population are going to be more sensitive and more vulnerable to fluoride's toxic effects than others. By ignoring the plight of these individuals, the Forum authors are able to duck the implications of a policy which seeks to help some members of society, while hurting others. A similar argument applies to those individuals who suffer from mild, moderate and severe dental fluorosis.

8) Fails to provide the necessary precautionary advice to nursing mothers not to use infant formula made up with fluoridated tap water. Their failure in this respect is inconsistent with the Scientific Subcommittee of the FSAI (appendix 18) which recommends breastfeeding exclusively for the first four to six months of the baby's life. Even with the modified "optimal" level for water fluoridation lowered to 0.7 ppm, a bottle-fed baby would be getting 70 times more fluoride than a baby which is breast-fed. Not only is the Forum authors' assertion that there is no evidence of increased risk of dental fluorosis in babies drinking fluoridated water incorrect, but also there are far more serious risks to the baby which they are ignoring.

In our view, by failing to assess properly all the evidence available in the international scientific literature, the Forum wasted a valuable opportunity to fully engage the scientific case opposing fluoridation as proposed by the Irish Minister of Health when he invited submissions to the Forum. Moreover, the Forum failed to carefully weigh all the evidence. As scientists familiar with the literature on this matter we can only conclude that the aim of the authors of this report was not to study the evidence, but to find ways to get around it. The report's primary conclusion that there are no adverse health effects is not defendable, and in our view, is blatantly false.

Sadly, the omissions and failings in this report, and particularly, the authors' specious justification for using an industrial grade waste product (hexafluorosilicic acid) for the fluoridating agent, if considered on a purely scientific level, are so inexplicable that we are forced to look for other explanations for its weaknesses. The most logical conclusion is that the majority of the panel members (who worked in some capacity for the Irish government, or received their research funding from the same) were persuaded to produce a report in support of this long-standing government policy rather than freely and objectively analyzing the information which was made directly available to them in testimony as well as that available in the open scientific literature.

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Section 2.

Conclusions and Recommendations.

In our view, the Fluoridation Forum had several tasks. It first had to demonstrate that fluoridation was effective at reducing tooth decay in Ireland. It then had to address all the concerns that have been expressed about fluoride's harmful effects at the daily doses experienced in optimally fluoridated communities as well as the lifetime cumulative dose. If, after addressing these two tasks, there was uncertainty in either or both of the outcomes, then it had to offer some kind of balancing analysis which would demonstrate that any benefit found outweighed any possible health risks, with a sufficiency to overcome concerns about margin of safety considerations for a whole population.

Particular consideration should have been given to individuals who are likely to exhibit a wide range in sensitivity to any toxic substance, and who are also likely to experience a considerable range of exposure to fluoride from water and other sources, a sizeable number of whom are unable to avoid fluoridated water.

Some would argue, that no matter how strong the benefit and how weak the evidence of harm, the individual should still have the right to exercise "informed consent" in this matter and the Irish (or any) government should not be allowed to override this important human right, by enforcing medication on all. We agree with this ethical position. However, our main concern with this report is that the Forum has failed to demonstrate a significant benefit to teeth and failed to fully, or even partially, address many of the health concerns that have been raised.

Even the few concerns that the Forum authors did address were tackled inadequately and unscientifically. They ignored many studies, even ones that were actually presented to them in person, and those they didn't ignore they addressed superficially. In particular, they failed to use a "weight of evidence" approach which, in our view, is critical in a situation where a whole population is being exposed to a toxic substance, and where damage may not be realized until nearly a full lifetime of exposure. To wait until all the evidence is in simply means that a whole population will have received a lifetime of exposure. This is taking governmental arrogance to a whole new level of disdain.

In our view adding fluoride to the drinking water of every man, woman and child in society is akin to building a dam above a village. If something goes wrong with the dam it could flood the village. If those supporting fluoridation are wrong in their claims of safety millions of people could be adversely affected. That is why it is essential that those who review this policy leave no stone unturned in case there are flaws in the promoters' arguments. In toxicological terms that means taking into account every piece of scientific evidence however remotely relevant. The Forum should have examined biochemical studies; animal testing; clinical studies; all the evidence gleaned from countries which have high natural levels of fluoride (China, India and countries in Africa); the fluoride levels in the tissues and particularly the bones of people living in fluoridated communities, and epidemiological studies. These studies should be assessed for any need for caution. Those in charge, like the Minister of Health and Children, are appointed to look for red flags not to avoid them. That's their duty as public health officials. If they are unwilling to do their duty then they shouldn't be putting fluoride,

and certainly not untested industrial grade hexafluorosilicic acid, into the drinking water of their citizens.

Unfortunately, the Forum authors, like many government sponsored report authors before them, have not chosen the careful approach. Instead of looking under every stone, they have left whole mountains unscaled. They have missed out many important studies and lines of enquiry. Moreover, where red flags have been waved, instead of proper scrutiny and research, they have simply looked for other studies to nullify them. Their aim appears to have been not to study the evidence, but to find ways to get around it. In short, they are approaching this task in the same manner the chemical industry has defended a whole host of toxic substances in the past (e.g. tetraethyl lead, DDT, PCBs, dioxins and furans).

Underlining the Forum's failure to take its charge of protecting the public seriously, is their failure to address the fact that the industrial grade chemical (hexafluorosilicic acid) used to fluoridate Ireland's water has never been tested in long term toxicological studies. This is especially inexplicable in light of the fact that Masters and Coplan have shown (1999, 2000) that there is an association between the uptake of lead (from various sources) into children's blood and the use of these same fluoridating chemicals in the United States. Moreover, these same authors have found an association between their use and indices of violent and anti social behavior, which is entirely consistent with this greater uptake of lead.

What is particularly shocking about the Fluoridation Forum's failure, and other previous government sponsored reports, is the relatively simple task of extrapolating from solid clinical data to predict a serious outcome for the bones from lifetime exposure to fluoride. If one simply calculates, as we have done, the cumulative dose used in clinical studies for the treatment of patients with osteoporosis, from the administered daily dose (20-40 mg per day for 1 - 4 years) which resulted in the unintended increase in hip fracture rates, it is not difficult to conclude that these cumulative doses will be exceeded during lifetime exposure by people living in fluoridated communities (1.6 to 6.6 mg per day for 70 years or more).

Instead of doing these kind of calculations on cumulative doses, the fluoridation forum authors obscured the issue in two ways: 1) they continued to describe the clinical experience as "high dose" trials without acknowledging that the trials only ran for short (compared to lifetime) periods (1-4 years), and 2) failed to underline the significance of the fact that approximately 50% of every daily dose of fluoride accumulates in the bones. Such an accumulation cries out for cumulative dose calculations.

Perhaps, the clearest evidence that the practice and promotion of fluoridation has always been a non-scientific venture, is the fact that no government endorsing this practice, including the Irish government, has ever felt fit to call for, or financially support, the careful monitoring of fluoride levels in the bones of their citizenry. This, even while they have poured money into studies of dental caries and dental fluorosis and even money into studying the psychology of people who oppose fluoridation. The fluoridation forum fell into line once again by failing to call for the obvious.

Because the Fluoridation Forum has failed to demonstrate either the efficacy of fluoridation or its safety, or convincingly demonstrate that this is an issue over which the government still has the right to overrule the individual's right to "informed consent" to medication, it is time for the Irish government to halt this practice.

Fortunately, there is plenty of evidence in the dental literature that where communities have stopped fluoridation in recent years in Finland, Cuba, former East Germany and Canada, that tooth decay has not increased, but actually continued to decrease. Nor does halting fluoridation deprive anyone of fluoride who is capable of brushing their teeth, since fluoridated toothpaste is universally available. We would further argue that the money saved by the Irish government in halting this practice would be far better spent targeting vulnerable individuals and communities with better education in oral hygiene, free toothbrushes and toothpaste, better dietary information and better dental treatment services.

It is time for the Irish government to have the political courage to admit that this practice was, is, and always will be wrong. They will win far more respect taking this high road, than the low road of dragging science and common sense through the mud in a report like this.

1) The historical, geographical and political context of the report.

Ireland is one of the very few countries in the world which has water fluoridation mandated at the national level. This policy was enacted in 1963. As a consequence Ireland is one of a mere handful of countries worldwide with the majority of its citizens drinking water which has fluoride added. Virtually every country in Europe has abandoned the practice, some after lengthy trials.

In the 1990's there were attempts by the UK government to extend fluoridation into Northern Ireland beyond two suburbs of Belfast. This effort led to very widespread opposition across the political spectrum. The end result was that 25 out of the 26 councils in Northern Ireland rejected the proposal. The widespread and vocal opposition to fluoridation in the North raised questions from jurisdictions in the South. Eleven County Councils have gone on record as opposing the forced fluoridation of their local water supplies by central government.

In recent years citizens, Irish environmental and public health organizations, increasing numbers of dentists, and the media have raised many questions about the practice. These include:

1) Is it necessary to have the water fluoridated when fluoridated toothpaste is universally available?
2) Why is it that countries which do not fluoridate their water have teeth just as good if not better than those that do?
3) Why is it that when communities stop fluoridating their water, tooth decay does not go up but continues to go down, as it has been in most industrialized countries since World War II?
4) Why are the fluoridating chemicals used industrial waste products, and not pharmaceutical grade?
5) Is the prevalence of a number of health problems in Ireland -- such as irritable bowel syndrome, osteoporosis, osteoarthritis, hip fracture, bone cancer, Alzheimers disease -- related to lifelong exposure and bio-accumulation of fluoride?
6) Why has the government not systematically studied a possible relationship among these diseases and exposure to fluoride?
7) Why does the government feel that they can force fluoride on its people, even when some of these questions have not been fully answered?
8) Does any government have the right to override the citizen's right to "informed consent" to medication?

Because of these and other questions being raised, and the very unsatisfactory answers from the central government, a number of prominent politicians began to call for an end to fluoridation.

Eventually, this accumulating pressure forced a response from Mr. Micheal Martin, the Minister for Health and Children, who set up the Fluoridation Forum to review the policy. Key activists questioned whether this panel was created in an effort to protect the health of the Irish people, or to protect government policy.

2) Membership of the panel.

We note that of the 20 Forum panel members (listed p 14-15 of report), at least 10 work for the Irish government (national or regional), 2 are well known for their promotion of fluoridation (Professor John Clarkson and Professor Denis O'Mullane), and none specialize in toxicology. The fact that there was no one on the panel with specialist knowledge on toxicology goes a long way to explain how such an elementary mistake (see section 4.5) of presenting the tolerable daily intake for fluoride as 10 mg/kg/day, which is twice the acutely toxic (even lethal) dose, went unspotted, not once but twice!

3) Protecting health or protecting policy? Evidence of bias.

a) Use of conclusory statements.

Several times in the text, it is stated AS FACT that fluoridation reduces dental decay. Since this was presumably one of the points the panel was supposedly investigating, it is inappropriate to repeat this as a statement of fact, rather than a conclusion reached through an impartial examination of the evidence.

b) Chapter 7.

In Chapter 7, the Forum addresses the status of fluoridation worldwide. One can only describe their discussion as a "positive spin" on this matter. Instead of admitting that most countries in Europe do not fluoridate their water (e.g. Austria, Belgium, the Czech Republic, Denmark, Finland, France, Germany, Greece, Italy, Luxemburg, the Netherlands, Norway, Portugal and Sweden) they go to pains to inflate the number of countries around the world that do. Without citing a reference they tell us that "Approximately 317 million people in 39 countries benefit from artificially fluoridated water".

Without a citation it would be foolhardy to accept this number at face value. We can only assume it is based upon lists prepared by the British Fluoridation Society (BFS), since the BFS is the most commonly cited source for these statistics. However, it should be noted that the BFS list includes countries like Switzerland, New Guinea, and Fiji, which have only one city fluoridated, and other countries like Cuba and the Czech Republic which have stopped fluoridation, and countries like the Philippines where only the US military bases are fluoridated. In our view, it would be more appropriate to provide three lists:

(i) Those countries where the majority of citizens are drinking fluoridated water (i.e. Australia, Ireland, possibly Israel, New Zealand, Singapore and the United States). This list is a mere handful of the fluoridated countries listed by the BFS.

(ii) Those countries where 10% or more are drinking fluoridated water (e.g Canada, and the UK). This list is also very short.

(iii) Cities in otherwise unfluoridated countries which are fluoridated, e.g Basel in Switzerland.

Presented this way, it would become very clear to Irish citizens that they are in a very distinct minority worldwide and are a part of the 2% of Europe which is still fluoridated.

Other statements in this chapter do not stand up to scrutiny, and appear to be an attempt to minimize other countries outright rejection of water fluoridation. For example, their claim that, "The Government in the Netherlands did not persist with water fluoridation because it was unable to supply fluoridated and non-fluoridated water to adjacent towns depending on the decision reached by communities sharing the same water supply" is incorrect. It was not technical limitations that halted fluoridation in Netherlands, but a Supreme Court ruling on June 22, 1973, which stated that fluoridation had "no legal basis." When some politicians tried to amend the law to create a legal basis for fluoridation, "it became clear that there was not enough support from Parliament for this amendment and the proposal was withdrawn." These are the words of the Netherlands' Office of Drinking Water, January 26, 2002. (See letter at http://www.fluoridationfacts.com/c-netherlands.htm)

Likewise, the Forum's description of the Swedish battle over fluoridation is also very misleading. According to the Forum,

"A body established by the Swedish government advised that water fluoridation should proceed. A bill was prepared but not enacted."

Such a description gives little indication of the vigorous and articulate nature of the scientific opposition to fluoridation in Sweden. In fact, it is interesting to note that the scientist who led the successful opposition in Sweden was Dr. Arvid Carlsson who won the Nobel Prize for Medicine in 2000. According to Carlsson's written testimony to the Swedish Government in 1978,

"Water fluoridation also goes against leading principles of pharmacotherapy, which is progressing from a stereotyped medication of the type of 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. Not only in that the dose cannot be adapted to individual requirements. It is, in addition, based on a completely irrelevant factor, namely consumption of drinking water, which varies greatly between individuals and is, moreover, very poorly surveyed."

For this and other reasons, Carlsson stated in his testimony "I am quite convinced that water fluoridation, in a not-too-distant future, will be consigned to medical history."

Section 3

4) Health Concerns and key studies omitted. A critique of the Forum's Chapter 11.

4.1 Dental fluorosis.

The Forum's analysis of dental fluorosis is given a fuller treatment in section 5 below. What we found disturbing from the health perspectiven is that unlike fluoride's benefits to teeth, there is little discussion of how fluoride causes dental fluorosis and its relationship to other systemic health effects.

They acknowledge that this is a systemic effect but do not comment on the significance of this finding. They are content to focus only on the visible appearance of the phenomenon. It is described as a "cosmetic" problem; an "aesthetic" problem, even as a public perception which may require study.

They do not discuss the possibility that dental fluorosis is the first visible sign of fluoride's toxic effect on the body. Nor do they discuss the mechanism of how fluoride causes dental fluorosis. They do not discuss denBesten's work indicating that fluoride poisons one of the enzymes involved in the developing tooth (denBesten, 1999). Had they done so, it might have prompted them to consider what other enzymes fluoride might also be poisoning. Others have suggested that fluoride causes dental fluorosis by interfering with the thyroid gland and had they addressed this possibility it might have led them to consider the literature which deals with fluoride's ability to lower the activity of the thyroid gland of those suffering from hyperthyroidism, an issue which they do not address in the report (Galletti and Joyet, 1958, Bachinskii,1985).

4.2 The Alarcon-Herrera et al (2001) study.

One of the many papers not acknowledged, or reviewed, by the Fluoridation Forum was a report by Alarcon-Herrera et al (2001) which appeared in the May 2001 issue of Fluoride, together with an editorial on the same issue. In this study Alarcon-Herrera et al found a strong linear correlation between the severity of dental fluorosis and the frequency of bone fractures in children. The study examined children in an area in Mexico which is naturally high in fluoride. Thus, far from "dental fluorosis" being an inconvenient "cosmetic problem" it may be an indicator of early bone damage. This phenomenon is also covered in our discussion of fluoride and bone damage (section 4.10).

4.3 Tolerable Daily Intakes (TDI).

In their "background" introduction to this topic the authors refer only to the use of "human population" studies and animal studies in determining TDIs. They point out the limitation of human studies for this purpose, because of the usual lack of dose information and interference from exposure to other substances. However, in their discussion, they have neglected to include the use of clinical studies, where humans have been exposed to very precise doses of fluoride for known periods of time. These studies involved giving fluoride tablets to patients with osteoporosis in an effort to reduce

bone fractures (see our discussion of bone damage in section 4.10). Moreover, in determining TDIs they avoided using the most sensitive end point found to date, namely the impact on rat brain (Varner et al, 1998).

4.4 A TDI without uncertainty factor.

The authors define a tolerable daily intake (TDI) as "what can be ingested (daily) over a lifetime without appreciable health risk".

In their discussion of the standard way of determining TDIs, they rightly refer to the fact that a No Observable Adverse Effect Level (NOAEL) is first determined and then this is divided by an uncertainty factor (UF). However, when they derived their TDI for children up to age 8 years (0.05 mg/kg/day), they did not apply an uncertainty factor -- they simply took the NOAEL for mild fluorosis in permanent teeth. A tolerable upper intake (for children under 8 years have) is defined as 0.1 mg/kg/day, which is based on the level which causes moderate dental fluorosis, again without an uncertainty factor being used.

4.5 A huge mistake: a TDI which is enough to kill.

When the authors consider a TDI for children over 8 years and for adults, they state the following: "For children over 8 years and for adults (i.e. not at risk of dental fluorosis) a NOAEL of 10 mg F/kg/day is considered appropriate. In order to attain that level of exposure large amounts of water and toothpaste would need to be consumed over long periods."

A TDI of 10 mg/kg/day (this means a dose of 10 mg of fluoride per kilogram bodyweight per day) would mean that a 60 kg adult would be able to ingest 600 mg/day without harm. In reality, such a high dose would undoubtedly cause serious acute effects and possibly death. This point is made abundantly clear in the next section which deals with acute toxicity. Here a "probable toxic dose" (PTD) is defined as "the minimum dose that could cause toxic signs and symptoms including death and that should trigger immediate therapeutic intervention and hospitalization". The authors set this PTD at 5 mg/kg/day. In other words they defined a tolerable daily intake (10 mg/kg/day) as twice a probable toxic (and possibly lethal) dose (a PTD of 5mg/kg/day)! The same mistake is repeated in the Executive Summary, where the TDI and PTD appear on the same page.

Now clearly the authors have made a major mistake. What is disturbing is that for anyone familiar with toxicology this is a very easy mistake to spot. How many people on this panel proofread this report? Did no one know enough about toxicology to spot this sloppy mistake, which occurred not once, but twice!

If they appear to understand so little about TDIs, what other serious oversights did they make in the discussion of the toxicity and toxicology of fluoride? Moreover, since the Fluoridation Forum authors placed so much emphasis on reviewing other agency's reviews on fluoridation, it raises the question of how well they reviewed and understood these documents?

4.6 Margin of safety.

In our view, the authors should have provided a very clear discussion of the margin of safety of fluoride for the various end points of concern. This would have entailed telling us how many milligrams of fluoride per day is necessary to protect teeth (the therapeutic dose); how many milligrams of fluoride can cause a variety of undesirable end points (the toxic dose). Dividing the toxic dose by the therapeutic dose then gives the "margin of safety" for each end point. With these margins of safety in hand, they then should have compared them with estimated daily doses children and adults are getting in Ireland, to see how far they are edging into, or even exceeding, those safety margins. Such calculations would reveal that for several important endpoints the margin of safety, if indeed any exists at all, is ridiculously small, especially for a medication for which the dose cannot be controlled, and for one destined to a go to a whole population with a probable ten fold range of sensitivity to any toxic substance, including fluoride.

4.7 Fluoride's Bio-availability and the Pineal Gland.

In a short section on Fluoride Bio-availability, the FF authors state that, "the quantity of fluoride that could affect biological processes is very small as most of the fluoride retained in the body is sequestered in the bones and teeth." However, they fail to acknowledge a very important recent finding by Jennifer Luke (1997, 2001) that fluoride also accumulates in the human pineal gland.

The pineal gland is a small gland which is located between the two hemispheres of the brain. It is responsible for manufacturing a very important hormone called melatonin. This hormone acts like a biological clock. Its release and plasma concentrations are thought to control the timing of various biological events and cycles such as sleep patterns, the onset of puberty and aging. There are three things which make this little gland a target for fluoride accumulation: 1) It is not protected by the blood brain barrier 2) It has a very high perfusion rate by blood and 3) most importantly, it is a calcifying tissue, i.e. like the teeth and the bone it lays down the same crystals of calcium hydroxy apatite. Luke theorized that this gland would highly concentrate fluoride and when she examined the pineal glands from 11 corpses this is exactly what she found. The levels of fluoride on the crystals averaged about 9000 parts per million (ppm), which is extremely high. This work was part of her Ph.D. thesis (1997) and was published in the open literature in 2001.

The second part of her thesis involved examining the effect of fluoride on melatonin production in animals. She found that not only was melatonin production lowered, but the animals reached puberty earlier, as would be predicted. This part of her work has yet to be published in the open literature, although copies of her thesis have been made available to regulatory officials in the US.

What is inexcusable about the FF authors' failure to acknowledge this important work is that it was presented to them in October 2000, as part of Dr. Connett's testimony.

Their attention was also drawn to an interesting finding in the context of Luke's work. The second trial of fluoridation in the US occurred in Newburgh (fluoridated) and Kingston (unfluoridated control) and took place from 1945 to 1955. The health of the children was followed and reported in 1956 (Schlessinger et al). The authors found that the young girls were menstruating on average 5 months earlier in fluoridated Newburgh compared to unfluoridated Kingston. This is consistent with fluoride's ability to shorten the time to the onset of puberty.

We have to ask, why it was that the Irish Ministry of Health would go to the trouble of flying Dr. Connett from the US to Cork, Ireland, to give a presentation to this forum, if they subsequently

ignored the most important scientific findings he shared with them? If they felt that the work was unimportant, or premature, then surely they were, at least, obliged to acknowledge that the work exists and then give their reasons for ignoring it?

4.8 Second-hand science: Review of reviews.

Most of the remainder of Chapter 11 consists of reviews of reviews. Such an exercise might prove of some useful purpose if the reviews in question were conducted by independent or impartial panels. Most of them were not. Most of the Reviews have been commissioned, just like the Fluoridation Forum, by governments who practice fluoridation, and thus it is difficult to disentangle analyses designed to protect a policy from those which genuinely seek scientific and objective answers to serious questions about fluoride's toxicity and margin of safety. Such observations might be considered to be overly cynical were it not for the fact, that nearly all of these reviews have been highly selective in which literature they cite, and seem to consistently "overlook" the studies which indicate that there may be serious problems with this practice.

It is interesting to compare the Forum's summary of the York Review (pp. 119-120) with the comments of Douglas Carnall, the Associate Editor of the British Medical Journal, and Professor Trevor Sheldon, the chairman of the advisory panel to the York Review (see Appendix 3).

4.9 Key studies and key issues not covered in the Fluoridation Forum report.

Here are some of the key human and animal studies which were not covered by the Forum.

a) Masters and Coplan.

The most serious omission in our view is their failure to discuss the work by Masters and Coplan (1999, 2000) who have found an association between the blood lead levels in children (as well as violent behavior), and the use of hexafluorosilicic acid (and its sodium salt) to fluoridate water (but not the use of sodium fluoride).

This omission is striking because of the considerable time the Forum spent on discussing the use of hexafluorosilicic acid, the fluoridating agent used in Ireland. What Masters and Coplan have also brought out as a result to the responses to their work in the US (Urbansky and Schock, 2000), is that these agents have never been tested systematically for their long-term toxicology in animals (a fact that the US EPA has now acknowledged, see letter appendix 6). All the testing has been done on sodium fluoride. Moreover, when dealing with one rebuttal of their work by Urbansky and Schock (2000) they were able to refute the claim that when the hexafluorosilicate ion is diluted it breaks down completely to free fluoride ion and silica and hydrogen ions, quoting a study carried out in Germany in the 1970s (Westendorf, 1975). The importance of this has even been acknowledged by Urbansky himself since he heads up the team at the US EPA which is currently calling for research proposals to find out what silcofluoride complexes and other species are present in aqueous solutions of hexafluorosilicic acid. In other words, neither the US government nor the Irish government knows either the chemical nature or the toxicity of the chemical substance they are giving to a majority of their citizens in their drinking water.

b) Varner et al (1998).

Another key study missed by the Forum is an animal study where Varner and co-workers showed that rats fed fluoride at 1 ppm in their distilled and de-ionized drinking water (as either aluminum fluoride or sodium fluoride) for one year, had morphological changes to their kidneys and brains, a greater uptake of aluminum into their brains and the formation of amyloid plagues associated with Alzheimers disease. The authors hypothesize that fluoride facilitates the uptake of aluminum into the brain.

c) G-proteins.

It is also puzzling that the Forum authors make no acknowledgement of the fact that there are some 800 studies in the biochemical literature which indicate that fluoride in the presence of a trace amount of aluminium switches on G-proteins. G-proteins are the key molecules involved in getting messages that arrive at the outside of our tissues (such as water soluble hormones, growth factors and some neurotransmitters) across the membranes in order to excite secondary messengers on the inside of the tissues. Thus fluoride can short circuit very important signalling mechanisms in the body. This may prove important to unravelling fluoride's effects on the brain (Strunecka & Patocka, 1999).

d) Luke's work on the pineal gland.

Luke's work is discussed in section 4.7.

e) Other studies on the brain and human behavior and development.

The Fluoridation Forum authors dismiss concerns about fluoride's impact on the central nervous system in their review of the 1999 Canadian Review, where these authors state, "Studies from China claiming children exposed to high levels of fluoride had lower IQs than children exposed to low levels were found to be deeply flawed and provided no credible evidence that fluoride obtained from water or industrial pollution affects the intellectual development of children."

This second-hand dismissal of such an important concern is irresponsible.

While recognizing that some of the Chinese studies did not consider all the possible confounding variables, they do raise serious questions. While the study by Li et al (1995) examined the IQs of children exposed to fluoride air pollution from indoor coal burning, both Zhao et al (1996) and Lu et al (2000) examined the lowering of IQ in children drinking water with fluoride levels as low as 4 ppm. The work of Lin Fa-Fu et al (1991) is also of concern because in this study a lowering of IQ thought to be due to the impact of iodide deficiency was actually exacerbated by moderate fluoride exposure (less than 1 ppm). This and other studies (Varner et al 1998, Zhang et al , 1999) raise the possibility that we have to be concerned not just about fluoride's impact operating by itself but in conjunction with other metal ions, or in nutrient deficiency situations. Nor are these studies confined to China. Calderon et al (2000) found an impact of fluoride on "reaction time and visuospatial organization" in children in a study conducted in Mexico.

Once again a weight of evidence approach is needed. These human findings have to be taken in conjunction with animal studies. Mullenix et al (1995) showed that rats exposed to fluoride developed behavioral effects typically produced by neurotoxic agents. Guan et al (1998) demonstrated an impact of fluoride on the membrane lipid levels in rat brain. Varner et al (1998) showed that fluoride, administered as either aluminum fluoride or sodium fluoride in rat's distilled and de-ionized drinking water at 1 ppm fluoride, damaged the brain and led to greater uptake of aluminum and the formation of amyloid plagues (see 4.9 b).

Why have the Forum authors ignored all these red flags? Who, for one moment, would countenance the administering of fluoride to children to achieve an almost imperceptible benefit to their teeth, if there are serious questions about its impact on their developing brains? The above information needs to be taken in conjunction with the fact that the level of fluoride in mothers' milk (0.01 ppm) is 100 times less than the levels put in the water (1 ppm). While the authors have largely confined their discussions of the impact of fluoride on young children to the aesthetics of dental fluorosis, there may be far more serious consequences that they (or the Reviews that they cite) have overlooked.

As long ago as 1978, Dr. Arvid Carlsson (Nobel Prize winner in Medicine, 2000) voiced similar concerns, when he said "One wonders what a 50-fold increase in the exposure to fluoride, such as occurs in infants bottle-fed with water diluted preparations, may mean for the development of the brain and other organs... Problems associated with this can only be solved by precise and comprehensive epidemiological studies in which, for example, breast-fed and bottle-fed babies are compared in localities with varying water fluoride content. No studies have yet been made."

Such arguments, persuaded the Swedish public and the Swedish parliament not to embark on the fluoridation experiment. The least one would expect from a country, like Ireland, is to very carefully collect the data on these kind of possible impacts. Sadly, they have not done so.

f) Freni's study on fertility, Freni (1994).

Another important study that the Forum authors ignored was Freni's (1994) study of lowered fertility rates in US counties with 3 ppm of fluoride or higher in their water. While 3 ppm is higher than the 1 ppm used to fluoridate water in Ireland, the results of this study pertains to issues of "margin of safety" and discussion of the total dose of fluoride where citizens are exposed to fluoridated water as well as other sources.

g) Many bone studies (see section 4.10).

h) Studies on the thyroid gland (Galletti and Joyet, 1958, Bachinskii, 1985).

i) The case studies of individuals who appear to be highly sensitive to fluoride.

The Forum neglected to discuss the numerous case studies reported by Waldbott (Waldbott et al, 1978) and Moolenburgh (Moolenburgh, 1987) in which individuals have reported various symptoms as a result of exposure to fluoridated water, which disappear when they stop drinking the water. While a number of the Reviews the Forum report cites have dismissed these findings, the dismissals have been of a somewhat cavalier nature, and not as a result of thorough scientific study. In this respect the Forum was remiss in failing to address the concerns raised about the possible contribution of fluoride exposure to the high incidence of irritable bowel syndrome in Ireland.

Section 4.

4.10 Fluoride and Bone Health.

The one issue for which the FF authors did attempt a semi-independent review was Fluoride and Bone Health. However, this section is marred by an unwillingness, or inability, to use a weight of evidence approach to this issue and their selective use of the literature.

a) Selective studies.

The Forum authors discuss two recent studies in which no increase in hip fracture associated with fluoride in water was found (Hillier et al, 2000, and Phipps et al, 2000) but fail to discuss another recent study that did (Li et al, 2001). Moreover, in discussing Phipps's findings, the Forum authors point out that fluoride exposure was associated with a decrease in hip fracture and vertebra fracture, after correcting for 13 variables, but failed to point out that she also found an increase in wrist fracture, which was only a thin hair off significance. In the York report, the wrist fracture finding was reported as significant (see Connett 2001). Dr. Connett discussed this situation with the Forum in October, 2000 but they chose to ignore his comments. Why?

As the Forum authors point out, increased hip fracture in the elderly is a very serious -- even life-threatening -- situation and the number of hip fractures in Ireland has approximately doubled over the last ten years. It is critically important therefore to examine all the evidence to see if it is possible that exposure to fluoride over a whole lifetime could lead to weakening of the bones.

b) Omitted studies.

Li et al (2001).

It is inexplicable that the Forum authors failed to discuss the study by Li et al (2001), whose findings Dr. Connett presented to the panel in his presentation in October 2000. At the time, the study was unpublished, but it has since been published in the Journal of Bone and Mineral Research, several months before the Fluoridation Forum report was finalized.

This study is important in our view because it provides a very convincing dose-response relationship between hip fracture rates in the elderly and the level of fluoride in their drinking water. It is also important because it helps us to define a margin of safety for this very serious outcome. Li et al (2001) compared the hip fracture rates for the elderly in six Chinese villages with fluoride at levels in their well water which ranged from 0.25 ppm to 8 ppm. Using the village at 1 ppm as control, they found that the rates of hip fracture doubled when the levels went over 1.5 ppm, a result which was not statistically significant, and tripled when they went over 4.5 ppm, a result which was statistically significant. Unfortunately, the York review (McDonagh et al, 2000) concentrated on comparing the rates of hip fracture in the village at 1 ppm, with the villages with less than 1 ppm and in this comparison Li et al found no statistical difference. In our view, that is taking a rather myopic view of the significance of this data. The fact that rates doubled over 1.5 ppm and tripled over 4.5 ppm gives us a clear indication that we are dealing with a health problem with a very narrow margin of safety: possibly, as low as 1.5! Moroever, as the researchers found that the consumption of this well water

was the dominant source of fluoride for these people, it raises the question of what happens to people with lifelong exposure to fluoride, not only from water but with other sources such as dental products; processed food and beverages prepared in fluoridated water; pesticide residues and air pollution.

c) Weight of evidence from bone studies.

Standing on its own, the study of Li et al (2001) is highly significant. It becomes even more so when considering the studies of Sowers et al (1991), which found an increase in hip fracture in a US community drinking water at 4 ppm fluoride, and Turner et al (1992), which found that an equivalent of 4 ppm fluoride in the drinking water of rats resulted in significantly weaker bones.

Meanwhile, it is now clear from numerous animal and clinical studies that fluoride decreases the strength of bone (Roholm 1937; Gedalia et al., 1964; Daley et al., 1967; Beary 1969; Wolinsky et al., 1972; Chan et al., 1973; Riggins et al., 1974; Inkovaara et al., 1975; Riggins et al., 1976; Gerster et al., 1983; Moskilde et al., 1987; Hedlund & Gallagher, 1989; Kragstrup et al., 1989b; Bayley et al., 1990; Gutteridge et al., 1990; Riggs et al., 1990; Schnitzler et al., 1990; Turner et al., 1992; Sogaard et al., 1994; Sogaard et al., 1995; Turner et al., 1996; Turner et al., 1997; Haguenauer et al., 2000; Gutteridge et al., 2002). (See appendix 4)

The question is not whether fluoride reduces the strength of bone, but, at what level? It is this question which the Forum should have addressed -- but didn't.

At least two things need to be investigated. First, the levels of fluoride that accumulate in the bones of people living, for various periods of time, in fluoridated communities, need to be determined. Second, the levels of fluoride in bone that have been found to decrease bone strength, in both animal and clinical trials, need to be assessed. Thereupon, if the concentration of fluoride in bone which has been found to decrease bone strength in animal and clinical studies equals or exceeds the level of fluoride now found in the bones of some people living their whole lives in fluoridated communities, then it is clear there is a potential problem

Unfortunately, the Forum gives no indication at all of how much fluoride is accumulating in the bones of the Irish people. While research into fluoride exposure has been undertaken, no official, comprehensive results have emerged in the open literature. This state of affairs can no longer be considered a mere oversight.

However, from the scraps of data that are available from other countries, there is definite cause for concern.

In 1980, Alhava et al (1980) measured the concentrations of fluoride in the bones of people who had lived in a fluoridated community in Finland for less than 20 years, and compared it to the levels of fluoride in people from a non-fluoridated area.

According to Alhava, the average level of fluoride found in the bones of women in the fluoridated area was 1,360 ppm (cortical) and 2,070 ppm (trabecular), with some of the women having as much as 4,000 ppm.

An earlier study by Parkins et al (1974), found a range of 1,295 to 5,745 ppm in the iliac crest bones of people living in a fluoridated area of the United States. The average level of fluoride was 2,824 ppm.

A more recent study by Richards et al (1994), from Denmark, found the level of fluoride in bone to range from 463 to 4,000 ppm, with the average level for women being 1,337 ppm and for men 1,181 ppm. What's striking about this study, is that the bones came from people living in a non-fluoridated area. Moreover, another study by the same team (Sogaard 1994), found fluoride concentrations in osteoporotic patients (before treatment) to be as high as 4,250 and 6,500 ppm!

Thus, based on the limited data currently available, fluoride concentrations in human bone from areas with 1 ppm fluoride or less in the water, ranges from as low as 500 to as high as 6,500 ppm.

To put these figures into perspective, the following is a list of fluoride concentrations which have been found to reduce the strength and/or quality of animal bone.

1,963 - 2,223 ppm in quail. (Chan et al., 1973)
1,704 ppm in pigs (cortical bone). (Kragstrup et al., 1989b)
2,826 ppm in pigs (trabecular bone). (Moskilde et al., 1987)
3,300-4,600 ppm in rats. (Sogaard et al., 1995)
4,500 ppm in rats. (Turner et al., 1993)

From these studies, it appears that the threshold at which fluoride reduces the strength and quality of bone is somewhere between 2,000 and 4,500 ppm.

However, whether the threshold is as low as 2,000 ppm or as high as 4,500 ppm, it is clear from recent human data that there will be people in the population exceeding this level. We find this deeply disturbing.

It is particularly disturbing when considering that 9 studies conducted since 1990 have found a significant association between fluoridated water and hip fracture (Jacobsen et al., 1990; Cooper et al., 1991; Keller 1991; Danielson et al., 1992; May & Wilson 1992; Jacobsen et al., 1992; Jacqmin Gadda et al., 1995; Kurttio et al., 1999; Hegmann et al., 2000).

An additional study also found an association between fluoridation and hip fracture (Suarez Almazor et al., 1993), although the authors dismiss the association since it was slight and only found in men.

Since it is important in public health policy to consider the worst-case scenario, it is essential that any discussion of fluoridation and bone, consider those individuals with inadequate nutrition, failing kidneys, and excessive thirst (and combinations thereof).

In the Forum report, there is no mention at all of how water fluoridation might affect the bones of people with any or all of these conditions. This despite the fact, now well established, that poor nutrition (particularly a deficiency of calcium) reduces the concentration at which fluoride reduces bone strength (Beary 1969; Riggins et al., 1974; Riggins et al., 1976); and that calcium deficiency, poor renal function, and excessive thirst (which often accompanies poor renal function) all serve to increase the bone's accumulation of fluoride -- sometimes dramatically so (Jackson 1955; Adams & Jowsey 1965; Call et al., 1965; Beary 1969; Juncos & Donadio 1972; Riggins et al., 1974; Spencer et al., 1980; Gerster et al., 1983; Noel et al., 1985; Welsch et al., 1990; Turner et al., 1996).

It is thus imperative for any analysis on how water fluoridation may affect bone to consider what lifetime exposure to fluoride might do to an individual with any or all of the above conditions.

The Forum's report doesn't come close to answering any of these questions. This despite the fact that many Irish people undoubtedly have some of these conditions.

Lastly, it is unfortunate that the Forum did not provide their own independent assessment of the numerous recent clinical trials which have examined how high-dose fluoride treatment affects bone. Instead, the Forum relies on yet another review (from the Australian Dental Journal) to serve as a substitute for their own analysis.

This represents yet another serious weakness and missed opportunity of the Forum report. For, in contrast to the ecological studies, the clinical trials provide the most thorough and scientific evidence on how fluoride affects human bone (see Inkovaara et al., 1975; Gerster et al., 1983; Vigorita & Suda 1983; Riggs 1984; Dambacher et al., 1986; O'Duffy et al., 1986; Kragstrup et al., 1989; Hedlund & Gallagher, 1989; Hodsman & Drost 1989; Bayley et al., 1990; Riggs, et al.; 1990; Kleerekoper et al., 1991; Fratzl et al, 1994; Schnitzler et al., 1986; Sogaard et al., 1994; Lundy et al., 1995; Pak et al., 1995; Patel et al., 1996; Balena et al., 1998; Haguenauer et al., 2000; Gutteridge et al., 2002). These studies provide an invaluable tool for guiding public health policy on the matter. (See appendix 4)

d) Cumulative dose versus daily dose.

The daily doses of fluoride used in the clinical trials are considerably higher than one would receive drinking fluoridated water. However, this is no reason to dismiss the relevance of the findings, particularly considering that the high doses used in these trials were given over very short periods of times (e.g. 1-5 years) compared to lifelong exposure (70 years or more) to fluoridated water (as well as other sources of fluoride).

More useful from a public health point of view would be a careful analysis which compares the total, cumulative dose of fluoride delivered in these trials versus the total, cumulative dose of fluoride one could expect to receive living one's whole life in a fluoridated area.

Such an analysis is not difficult to do. For example, if one multiplies the daily dosage (33.75 mg/day) of fluoride used in the Riggs study (1990) by the number of days in the year (365), and by the number of years in the study (4), one will find that the total dosage given in the trial was roughly 49,275 mg of fluoride.

According to Riggs, patients receiving this dosage had an increased rate of hip fracture. To receive the same amount of fluoride as delivered in the Riggs study, a person would need to consume an average of 2.7 mg of fluoride a day for 50 years, 2.25 mg/day for 60 years, 1.8 mg/day for 75 years, or 1.5 mg/day for 90 years.

What's striking is that all of these dosages (1.5 - 2.7 mg/day) are well within the current estimates for how much fluoride people living in fluoridated areas are now receiving. For instance, according to a 1991 review by the US Public Health Service (DHHS 1991), the average daily ingestion of fluoride in a fluoridated community ranges from roughly 1.6 to 6.6 mg/day.

More troubling is that other clinical trials have found an increase in bone fracture at dosages considerably lower than the Riggs study. For instance, the Hedlund and Gallagher (1989) study found an increase in hip fracture in patients receiving just 22.5 mg fluoride per day for just two years, not four.

Thus, the total fluoride dosage used in the Hedlund study (16,425 mg) was approximately a third of the dosage used in the Riggs study. To receive this same total amount of fluoride, a person would need only to consume 0.9 mg of fluoride per day for 50 years, 0.75 mg/day for 60 years, 0.6 mg/day for 75 years, and 0.5 mg/day for 90 years. Such doses (0.5-0.9 mg/day) are routinely, and often grossly, exceeded in fluoridated areas.

Although there are likely other factors to be considered when making such comparisons (i.e the pre-treatment accumulation of fluoride in the osteoporotic patients and the potential differences between rapid and gradual accumulation of fluoride), the compatibility of dosages between the short-term trials and long-term real-life exposures is a definite cause for concern.

It is revealing to look closely at the words the Forum report authors chose to dismiss concerns about bone. They state:

"Trials have shown that high doses of sodium fluoride substantially increased vertebral bone density, but this effect was not associated with lower rates of spinal fractures (114). This effect has only been seen when intake has been substantially higher than would be expected from fluoridation of water. Sodium fluoride as an anabolic substance was used in the past in the management of osteoporosis, but is no longer licensed in Ireland and Europe. It prolongs bone remodelling if given in twice the therapeutic dose. Experimental studies have shown that fluorotic bone is more resistant to compressive forces, but more easily fractured by torsional strains. Moderate doses of fluoride have been shown to increase bone strength in experimental animals and high doses of continued exposure decrease strength."

And later in their conclusion to the section on bone, they state:

"The use of fluoride in the treatment of osteoporosis was referred to above. The use of high doses of fluoride in the treatment of osteoporosis is no longer a therapeutic option. However, the role of low doses of fluoride, as is obtained in drinking water, is the subject of a systematic review (118)."

This truncated analysis does not begin to do justice to this issue, either qualitatively or quantitatively. In the first sentence above, you will note that the Forum authors talk about fluoride treatments not being associated with "lower rates of spinal fractures". However, what they do not state is that the fluoride treatment of osteoporotic patients has actually led to increased hip fracture rates, i.e. the very opposite result to that intended. The forum states that fluoride was used in the management of osteoporosis "but is no longer licensed in Ireland and Europe", but they don't tell us why. What they don't stress or even acknowledge is that this treatment is not licensed because it did not decrease hip fracture rates but often increased them.

In their concluding remarks, the Forum authors are extremely misleading when they state: "The use of high doses of fluoride in the treatment of osteoporosis is no longer a therapeutic option. However,

the role of low doses of fluoride, as is obtained in drinking water, is the subject of a systematic review."

Again, the authors do not stress that in clinical trials fluoride increased hip fracture rates; all we are told is that these treatments are "no longer a therapeutic option".

Instead of quantifying the comparison between the doses used in these clinical trials and the doses estimated for lifetime exposure to fluoride in optimally fluoridated communities, readers are simply told about the "high doses" used in the treatment of osteoporosis, and the "low doses of fluoride, as is obtained in drinking water". The juxtaposition of the words "high" and "low" in these two sentences is highly misleading. It obfuscates the fact that the "cumulative dose" is comparable, as we have demonstrated above.

In short, the Forum makes no attempt to analyze this clinical data in any toxicologically meaningful way. It simply implies that, since the dosages used in the trials were higher on a daily basis, that the trials have no relevance to water fluoridation. Such a conclusion -- especially without any supporting analysis -- is cavalier and crude.

e) Fluoride and bone damage in children.

There are two other studies not mentioned by the Forum, which point to the fact that fluoride might also damage the bones of children.

i) Schlessinger et al, (1956).

Schlessinger et al found a statistically significant increased incidence (13.5% versus 7.5%) of cortical bone defects in fluoridated Newburgh after ten years of fluoridation at 1 ppm (compared to children in unfluoridated Kingston). This was the first health study conducted in the US on artifically fluoridated water. According to a reviewer for a National Academy of Sciences (NAS) report published in 1977:

"Caffey (1955) noted that the age, sex and anatromical distribution of the 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" (NAS, "Drinking Water and Health", 1977, p. 388-9).

This comment is serious on two fronts. First, it confirms that bone damage was observed in young children as a result of drinking fluoridated water for 10 years and second, it raises a red flag of concern (as early as 1955) that fluoride might cause bone cancer in young men. The fact that increases in osteosarcoma has been subsequently found in both male rats treated with fluoride (NTP, 1990) and higher rates of osteosarcoma have been found in young men living in fluoridated communities in at least two epidemiological surveys (SEER, 1991; Cohn, 1992), should not have been so cavalierly dismissed as it has been by American and other authorities (Hoover, 1990, 1991, NRC 1993), and in a second-hand fashion in this Forum report.

ii) Alarcon-Herrera et al (2001).

This study has already been referred to above in the discussion of dental fluorosis. Alarcon Herera et al (2001) found that the incidence of bone fracture in children in an area in Mexico (which had naturally high levels of fluoride in the water) increased in a linear fashion with the severity of dental fluorosis. It is well established that dental fluorosis is a bio-marker for fluoride exposure.

f) No monitoring of fluoride levels in bone.

As we have indicated above, fluoride may be damaging our bones at both the beginning and the end of our lives. What is particularly disturbing about the way governments of fluoridated countries have handled this issue, is that, despite the fact that they have known for many years that approximately 50% of the fluoride we ingest each day accumulates in our bones, there has been no systematic attempt in Ireland, or elsewhere, to track the level of fluoride in our bones as a function of age, geography, diet, health status and fluoridated water consumption. After nearly 40 years of fluoridation in Ireland, there should be a wealth of data; as the most recent international reviewers (York Review, 2000 and MRC, 2002) have indicated, there is none. While the Forum has recommended yet more studies on teeth, it has failed to recommend the collection of the most obvious and most basic data one would need to investigate the serious end point of bone damage. Why?

4.11 Failure to take into account total dose.

Another key weakness in the Forum report is the authors' failure to address the total dose of fluoride from all sources (See Stannard et al, 1991; Kritsky et al 1996 ; Turner et al 1998 ; Heilman et al 1999 ; Fein & Cerklewski 2001; Warnakulasuriya et al 2002.) . At times one gets the distinct impression that they see their task as exonerating water fluoridation of any harm, even though this means pointing the finger of blame at other sources of fluoride like toothpaste. For the average citizen, exactly which sources of fluoride is causing a problem is less relevant than the fact that many of our children are being over-exposed to fluoride from all sources combined. It is inexplicable that the Forum authors have not provided estimates of the total fluoride exposure to fluoride for both children and adults.

4.12 Failure to use weight of evidence approach.

In the section on bone strength (see 4.10) we stressed the failure of the Forum to combine the evidence that can be gleaned from animal, clinical and epidemiological studies. A similar failure to use "a weight of evidence" approach is revealed in many of the "Reviews" they have summarized. Two other examples are a repeated failure by government agencies to take a weight of evidence approach to a possible relationship between fluoride exposure and osteosarcoma in young men (see discussion in 4.10), and the impact of fluoride, in conjunction with aluminum and other ions, on the central nervous system (see Varner's work discussed in 4.9).

4.13 Failure to discuss the Precautionary Principle.

A definition of the Precautionary Principle was recently crafted by a group of scientists meeting in Racine, Wisconsin. They stated it this way: "When an activity raises threats of harm to the environment or human health, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically". Put more simply, it states, "If in doubt, leave it out."

If ever a policy should be forced to satisfy the precautionary principle it should be fluoridation, since this is the only time in human history (apart from a short experimentation with iodide) the public water supply has been used to deliver medication. If ever a policy screamed out for caution , this is it. What is being delivered to the whole population is a substance known to be highly toxic at moderate doses. The delivery system is incapable of monitoring individual response and doses cannot be controlled. Furthermore, the individual's normal right to "informed consent" to medication is being over-ridden.

The necessity for this precautionary principle has emerged because in the past it has been very difficult to prove convincingly that a persistent chemical has caused harm to workers or citizens. This is because by their very nature it is extremely difficult in epidemiological studies to control for all the complex and confounding variables in society. As some scientists have jokingly observed, "An epidemic is a health problem that even an epidemiologist can spot."

With the wisdom of hindsight, scientists have realized that by the time scientific proof has been obtained, which is robust enough to resist the most entrenched invested interest, it is too late for thousands or even millions of people who have meanwhile been damaged by exposure to the chemical of concern. The precautionary principle is a principle designed to help officials protect the public from this kind of damage.

In our view, there are five questions which should help to avoid exposing people to unnecessary risks from chemicals for which the toxicological and epidemiological data base is incomplete, as is the case with fluoride and the other chemicals like hexafluorosilicic acid used to fluoridate the public water supply. They are:

First question. Is the public being exposed to a chemical for which there is plausible evidence of harm?

Second question. How serious is this harm if it is found that indeed this chemical causes it?

Third question. How significant is the benefit being pursued?

Fourth question. Are there alternative approaches to pursuing this benefit?

Fifth question. Have all the people being exposed to these risks agreed to the exposure?

In our view, water fluoridation fails on all five questions.

Unfortunately, the Fluoridation Forum authors seem totally oblivious to any notion of a precautionary principle approach. Essentially, they are insisting that the practice of water fluoridation should continue until there is absolute proof of harm.

4.14 Failure to address Paul Connett's "50 Reasons".

In October of 2000, the Forum was presented with "50 Reasons to Oppose Fluoridation" by Dr. Paul Connett, professor of chemistry at St. Lawrence University. At the time, the Forum stated that it would respond to the 50 reasons, and indeed, soon set up a sub-committee to do so. For approximately 10 months, the Fluoridation Forum website reported on several updates of the progress this subcommittee was making in responding to this list. In September 2001, however, it was announced on the website that the subcommittee did not have the time to complete the task -- this despite the fact that they had almost a year to do so.

The only apparent reference to this matter in the Forum report comes in one line in the section labeled "Presentations and Submissions" in which the authors write: "One presenter requested a response to his submission and the response of the Forum to this request will be presented on the Forum website. The final Forum Report has taken account of the issues raised in the submission." (p23)

If readers check the "50 Reasons" (available at http://www.fluoridealert.org/50Reasons.htm or in appendix 2 of this report), they will find that very few of the concerns are addressed in the Forum's report, and none adequately.

4.15 The use of hexafluorosilicic acid instead of sodium fluoride (Chapter 10).

In Chapter 10, the Forum authors claim that the reasons for the switch from sodium fluoride (toxicologically tested) to hexafluorosilicic acid (not toxicologically tested) was made because,

"The sodium fluoride was very hygroscopic (water-absorbent) and as water treatment plants are by nature damp places there was a tendency for the powder to become solid, resulting in major difficulties measuring accurate amounts to add to the water. The dust from the powder was a serious health and safety threat to water plant workers."

According to Myron Coplan, an engineer with first-hand knowledge of fluoridating chemicals,

"This is an invalid, non-credible, specious argument. The dry powder, as delivered in bags, can easily be dissolved in plant water to a standard concentration (eg saturation), and stored in corrosion-resistant polyethylene tanks indefinitely without deterioration and used at any time, when called for.

"Such a solution, at known concentration, can easily be metered into the main water flow in a manner similar to the way hexafluorosilicic acid (HFSA) is metered into the main water flow. As a matter of fact, however, the saturated NaF solution would be far less hazardous to handle in the metering process. Spills and pump leaks, etc., would be easily collected and washed away without release of fluoride gases. The NaF solution itself would be far less corrosive at any concentration compared to the original HFSA and any dilution thereof. A saturated solution of NaF (4.2%) has a pH of 7.4, very slightly alkaline which can be handled easily in commonplace inexpensive available equipment such as pumps, valves and piping.

"Moreover, ANY prepared concentration of NaF could also be known precisely. In fact, the standard for calibrating the fluoride specific ion electrode used for quality control and other laboratory purposes is a weighed out amount of NaF added to a known volume of water. This is to be compared with the relatively imprecisely known HFSA concentration designated as "Not Less Than XX percent" of some total solids of some imprecisely known species of fluoride-bearing compounds. It is not and never could be used to prepare a standard solution of known fluoride concentration for laboratory test purposes.

"In short, the Fluoridation Forum Report in dealing with the fluoridating agent (HFSA) starts out with an unequivocal fabrication regarding an easily demonstrated fact. Was this the result of sheer ignorance by the authors of the Report? Were they deliberately misled by other so called "experts" intent on hiding the real motive behind disposing of fluosilicic acid in public water supplies, not only in Ireland, but also in Canada, Australia, the UK and US? In any case, given the fact that the entire validation of water fluoridation embodied in the Forum Report starts with this obfuscated, yet easily refuted basis for using HFSA, how can the rest of it be reliable?"

Section 5.

5) Inadequacies of the Forum's dental analysis.

5.1 Is fluoridation preventing dental caries?

The results of several Irish studies were summarized on pp. 100-103. As presented, the data appears to show that fluoridation had a very minor benefit in terms of reducing overall DMFT (Decayed, Missing and Filled Teeth) rates. However, there are problems with the data that the authors did not address.

1. How do they explain the drop in dental caries in subjects who were life-long residents in both the non-fluoridated and fluoridated areas? While this phenomenon is acknowledged on p. 102, it is claimed that "The reduction, however, was greatest in the fluoridation communities", resulting in a 23% overall difference in 12 year olds, or a net difference of 0.7 DMFT.

2. The authors did not correct for a delay in tooth eruption from fluoride ingestion (See Kunzel et al, 1976; Krook et al, 1983; Virtanen et al, 1994; Campagna et al, 1995; Limeback, 2002.) This was pointed out to Dr. O'Mullane when external input was sought but this seems to have been ignored altogether. If one uses the UK data as reported by Diesendorf in 1986, a simple calculation will indicate that the 0.7 DMFT "benefit" can be explained by a delay in tooth eruption.

3. The "benefits" from water fluoridation in adults, when delayed tooth eruption no longer has an effect, are minor. The use of DMFT as a tool for measuring dental decay is flawed in that it is affected by tooth loss from periodontal disease. No where in the report was this mentioned. Apart from one study (ref. 75), which was not a case-control study , the "benefits" from fluoridation for adults is minor. Many factors affect caries risk, such as mature onset diabetes (Narhi et al, 1996) and these must be taken into account when comparing populations.

4. A carefully conducted, randomized, prospective clinical trial on water fluoridation has never been conducted, not in Ireland, nor anywhere else in the world. This is the kind of clinical evidence that is required to approve drugs for human use. Why should placing a drug in the water be any different?

In the US, the net benefit from fluoridation 15 years ago was minor. According to Hunt et al (1989),

"Coronal caries incidence was significantly lower for people who had resided in fluoridated communities for more than 30 years (1.95 vs 1.33 surfaces). Root caries incidence was significantly less among residents for more than 40 years (0.56 vs 1.11 surfaces)."

Today, after many years of fluoridated tooth paste use, one would likely find these differences eroded even further. Nowhere in the report is there an accounting of how much money has been spent on fluoridation in Ireland. After 30 years of water fluoridation, how many actual tooth surfaces were saved from decay? It is our contention that, if a delay in tooth eruption is factored in, the number of fillings saved per child is impossible to estimate unless the entire child population receives a dental examination. Even if a net statistically significant difference could be found in adults, it would be so small as to be clinically irrelevant.

5.2 Dental Fluorosis and the critical time of exposure.

According to a statement on p. 128 of the Forum report, "It would appear that the risk of dental fluorosis in the maxillary central incisors is low in the first 15 months of life."

To support this statement only one study is cited, that of Evans and Darwell (1995). While this study has been highly cited for trying to pinpoint the window of susceptibility, recent studies (Ishii and Suckling, 1991; Milsom et al 1996; Ismail et al, 1996; Bardsen and Bjortvatn, 1998; Brothwell and Limeback, 1999 and Fomon et al, 2000) show that exposure right from birth (during the first year) clearly increases the risk for dental fluorosis. Evansâ study may, therefore, be flawed (Burt, personal communication).

According to Bardsen and Bjorvatn:

"The findings indicate that early mineralizing teeth (central incisors and first molars) are highly susceptible to dental fluorosis if exposed to fluoride from the first and--to a lesser extent--also from the 2nd year of life."

According to Milsom et al:

"In light of these findings, it is worth considering the potential of the presence of enamel defects in deciduous molars in children aged 1 to 3 years as a predictor of the future appearance of similar lesions in their permanent incisors."

According to Ismail et al:

"The odds that a child had a maxillary central incisor with fluorosis were 5.69 (95% CI = 1.34, 24.15) times higher if exposure occurred during the first year of life compared with exposure after 1 year of age."

According to Brothwell and Limeback:

"Breast-feeding for 6 months or more may protect children from developing dental fluorosis in the permanent incisors."

According to Ishii and Suckling:

"Two 'at-risk' periods for the production of moderate or severe fluorosis were evident. One started at birth and ended early in tooth development, while the other started later and ended at eruption."

According to Fomon et al:

"We believe the most important measures that should be undertaken are (1) use, when feasible, of water low in fluoride for dilution of infant formulas; (2) adult supervision of toothbrushing by children younger than 5 years of age; and (3) changes in recommendations for administration of fluoride supplements so that such supplements are not given to infants and more stringent criteria are applied for administration to children."

5.3 Dental Fluorosis and Infant feeding.

On p.133 of the Forum report, the authors state that:

"It is recommended that parents continue to reconstitute infant formula with boiled tap water. Many brands of bottled water available in Ireland are not suitable for use in the reconstitution of infant formula due to the presence of salt and other substances which may be harmful to infants and young children."

It is hard to believe that this is a serious statement. Natural water has "salts" that may be harmful to the baby? Where are the studies to back this statement? What about the silicofluorides artificially added to tap water that are concentrated when boiled? The effect on infant development of these chemicals has never been tested. How can these chemicals be recommended as additives to infant formula over natural "salts" contained in bottled water?

On p. 134 of the report, the authors state that:

"An increase in the rate of breast feeding in this country would contribute significantly to a reduction of the occurrence of dental fluorosis."

If the Forum panel recognizes this to be true, then why promote dental fluorosis by continuing to recommend the use of infant formula made with boiled tap water which results in infant formula that has 100 times the level of fluoride as human breast milk?

If there is any doubt that infant formula made with fluoridated water increases dental fluorosis, whether at the old 1.0 ppm "optimal" level or the new 0.7 ppm "target" level, one only has to read the literature on the subject. The number of studies that have examined this problem is large. Why were the studies by Pendrys and Katz, 1989; Clark et al, 1994; Pendrys et al, 1994; Van Winkle et al, 1995; Grimaldo et al, 1995; Lewis and Limeback, 1996; Silva and Reynolds, 1996; Villa et al, 1998; Fomon and Ekstrand, 1999; Brothwell and Limeback, 1999; Pendrys, 2000; and Buzalef et al, 2001, ignored and not considered by the members of the fluoridation forum.

One must ask why this task was given to the Food Safety Authority of Ireland (FSAI) instead of being addressed by the Forum panel. Here are some key exerpts from some of these reports.

According to Buzalaf, 2001:

"Hence, to limit fluoride intakes to amounts <0.1 mg/kg/day, it is necessary to avoid use of fluoridated water (around 1 ppm) to dilute powdered infant formulas."

According to Pendrys, 2000:

"Enamel fluorosis in the optimally fluoridated study sample was attributed to early toothbrushing behaviors, inappropriate fluoride supplementation and the use of infant formula in the form of a powdered concentrate."

According to Fomon and Ekstrand, 1999:

"Many fewer infants are exposed to high F intakes from formula plus a supplement (recommended only for communities with water providing less than 0.3 ppm F) than from formula alone in communities with F content of 1 ppm in the drinking water."

According to Brothwell and Limeback, 1999:

"Breast-feeding for 6 months or more may protect children from developing dental fluorosis in the permanent incisors."

According to Villa et al, 1998:

"Subjects in Group I were 20.44 times more likely (95% CI: 5.00-93.48) to develop CMI fluorosis than children who were older than 24 months (Group III) when fluoridation began."

According to Silva and Reynolds, 1996:

"However, prolonged consumption (beyond 12 months of age) of infant formula reconstituted with optimally-fluoridated water could result in excessive amounts of fluoride being ingested during enamel development of the anterior permanent teeth and therefore may be a risk factor for fluorosis of these teeth."

According to Grimaldo et al, 1995:

"91% used infant formula reconstituted with boiled water." "Taking together all these results, three risk factors for human exposure to fluoride in SLP can be identified: ambient temperature, boiled water, and food preparation with boiled water."

According to Clark et al, 1994:

"Logistic regression analyses showed that the use of infant formula and parental educational attainment were significantly associated with the occurrence of dental fluorosis in the range of scores from 2 to 6."

According to Pendrys et al, 1994:

"Logistic regression analyses, which adjusted for confounding variables, revealed that mild-to moderate enamel fluorosis on early forming (Fluorosis Risk Index (FRI) classification I) enamel surfaces was strongly associated with both milk-based (odds ratio (OR) = 3.34, 95% confidence interval (CI) 1.38-8.07) and soy-based (OR = 7.16, 95% CI 1.35-37.89) infant formula use,"

According to Pendrys and Katz, 1989:

"An odds ratio of 1.7 associated with infant formula use was suggestive of an increased risk of enamel fluorosis"

5.4 Estimations of early childhood exposure to fluoride.

According to Appendix 18 of the Forum report, the Scientific Committee of the Food Safety Authority of Ireland (FSAI) made "estimates" of how much fluoride infants from birth to age 4 months were ingesting. Ireland has never actually measured these levels. This is unfortunate since comparing the Irish infants with those in one study carried out in Iowa US using a limited number of families is hardly enough scientific evidence to make the claim that the:

"maximum average intake of fluoride from infant formula reconstituted with fluoridated tap water over the first four months of life was estimated to be in the range of 0.105 mg/kg b.w./day to 0.712 mg/ kg b.w./day, depending on body weight."

If personal communication was made with Steven Levy, whose Iowan families are now the subjects in several fluoride intake studies, Dr. Levy could have informed the FSAI Scientific Committee of his latest results, which are now published (Levy et al, 2002). In this report, he and his co-workers indicate that:

"Results suggest that the middle of the first year of life is most important in fluorosis etiology for the primary dentition in this setting."

Why is this important? The FSAI Scientific Committee acknowledges that dental fluorosis in the primary dentition is a good predictor that dental fluorosis will occur in the permanent teeth. [p. 252, quoting Forsman (1977) and Walton & Messer (1981)]

And yet on p. 252, they state:

"On balance the Scientific Committee has taken the view that the most critical period for developing dental fluorosis of the permanent central incisors is between 15 and 30 months."

If these exposures (0.105 mg/kg b.w./day to 0.712 mg/ kg b.w./day) are confirmed with actual studies conducted in Ireland, then it would confirm that, on average, infants ingesting formula reconstituted with tap water are being exposed to a level of fluoride that is considered past the threshold that is safe for ameloblast function. A safe exposure level, where dental fluorisis is likely not to occur, is 0.050 mg/ kg b.w./day. Such a level can be determined from the work of Lewis and Limeback, 1996; Whitford, 1997 and Formon and Ekstrand, 1999. According to the latter authors:

"The addition of a F supplement of 0.25 mg/d for a 4 kg infant would increase the F intake by 63 micrograms.kg-1.d-1, resulting in a total intake of about 100 micrograms.kg-1.d-1, an intake in the range believed to be associated with development of fluorosis of the permanent teeth."

5.5 Conclusion on the Forum's dental analysis:

The report fails to demonstrate that over 30 years of fluoridation in Ireland has actually prevented tooth decay. Nor do the Forum authors attempt to put the Irish dental findings into the larger context of studies conducted elsewhere. For example, they do not mention the largest survey conducted in the US (Brunelle and Carlos, 1990) in which the authors could only find an average difference in tooth decay of 0.6 of one tooth surface out of 128 tooth surfaces for children (aged 5 -17

years) who had lived their whole lives in fluoridated communities compared to non-fluoridated ones. Even this minuscule difference was not shown to be statistically significant by the authors. Nor do they cite the work of Spencer et al (1996) who report an even smaller difference of 0.12 - 0.3 tooth surfaces in Australia. In New Zealand, de Liefde (1998) reports differences in tooth decay as being not clinically significant. Nor do they adequately address the fact that the vast majority of European countries have been able to achieve comparable, or lower, levels of dental decay as in Ireland, without fluoridating their water supplies. Finally, they do not acknowledge that where in recent years fluoridation has been halted in communities in Finland, Cuba, former East Germany and Canada, tooth decay rates have not gone up as predicted by promoters of fluoridation, but have actually gone down (Maupome et al, 2001; Kunzel and Fischer,1997,2000; Kunzel et al, 2000; and Seppa et al, 2000).

Further, the Forum authors do not provide convincing evidence that fluoridated water, even at the new target level of 0.7 ppm, does not, and will not, cause dental fluorosis when used to make up infant formula.

Section 6.

Appendix 1 Biographical notes about signatories.

Albert Burgstahler, Ph.D., has been researching the fluoridation issue for over 30 years. He is co-author of "Fluoridation: the Great Dilemma" (Coronado Press, Lawrence, Kansas, 1978), is currently the editor of the journal "Fluoride", and is on the executive board of the International Society for Fluoride Research.

Robert J. Carton, Ph.D., has been researching the fluoridation issue for over 20 years. He was former President of Local 2050 of the National Federation of Federal Employees, the union representing all the professionals at the headquarters of the U.S. Environmental Protection Agency in Washington, D.C. Dr. Carton was involved in exposing the political pressures exerted to establish an unscientific drinking water standard (4 ppm) for fluoride.

Paul Connett, Ph.D., has been researching the fluoridation issue for over 6 years. He was an invited peer reviewer of the York Report (McDonagh et al, 2000) and testified before the Fluoridation Forum in October, 2000. Dr. Connett is a key organizer of the Fluoride Action Network which hosts the web page http://www.fluoridealert.org.

William Hirzy, Ph.D., is Vice-President of the National Treasury Employees Union, Chapter 280, which represents the professionals employed at the US EPA's headquarters in Washington, DC. This union has taken a position opposing water fluoridation. On June 29, 2000 Dr. Hirzy presented his Union's position in a statement he gave before the US Senate Subcommittee on Wildlife, Fisheries and Drinking Water (http://www.fluoridealert.org/testimony.htm).

Vyvyan Howard, MB, ChB, Ph.D., FRCPath., is a medically qualified toxico-pathologist. He is a past President of the Royal Microscopical Society. The primary research activity of his group concerns the action of toxic substances on the fetus and infant. He is currently investigating the differential neuro-developmental toxicity of various fluoride preparations. He wrote the foreword to the recent book on water fluoridation by Dr Barry Groves.

David C. Kennedy, DDS, is a Past-President and Fellow of the International Academy of Oral Medicine and Toxicology (IAOMT), the IAOMT's Fluoride Information Officer, and Special Project Consultant. In 1998 he and Dr. J. William Hirzy authored a risk assessment for ingested fluoride based upon the standard methodologies used by the US Environmental Protection Agency Global 86 program, full text available at http://www.saveteeth.org/, that demonstrated the current intake of fluoride already exceeds the minimum risk levels.

Hardy Limeback, Ph.D., DDS, is the former President of the Canadian Association for Dental Research, and one of Canada's leading fluoride researchers. He has acted as consultant to the Canadian Dental Association on fluoride and gave invited testimony to the Fluoridation Forum in October, 2000.

Roger Masters, Ph.D., has co-authored two papers with Myron Coplan (1999, 2000) which have shown an association of an increased uptake of lead into children's blood with the use of hexafluorosilicic acid (HFSA) and its sodium salt (NaSFA) as a fluoridating agent. Masters and

Coplan have also shown a relationship with the use of the HFSA and NaSFA and violent behavior in communities in the US.

Tohru Murakami, Ph.D., DDS, is vice president of the Japanese Society for Fluoride Research. He has translated many articles on fluoridation into Japanese and circulated them via the Japanese Journal of Fluoride Research.

Bruce Spittle, Ph.D., has extensively reviewed the literature on fluoride's impact on the central nervous system (Spittle, B. (1994), Psychopharmacology of fluoride: a review. International Clinical Psychopharmacology, 9, 79-82; and Spittle, B. (2000), Fluoride and Intelligence (Editorial), Fluoride Vol. 33 No. 2: 49-52).

A.K. Susheela, Ph.D., is currently the Executive Director of India's Fluorosis Research and Rural Development Foundation located in Delhi. From 1969-1997 she served on the Faculty of the All India Institute of Medical Sciences (AIIMS) in New Delhi. During her tenure there, she set up the Fluorosis Research Laboratories. From 1987-97, Professor Susheela was in charge of the Fluorosis Control Cell of the Rajiv Gandhi National Drinking Water Mission (Govt. of India) and established the Fluorosis Diagnostic Facility at the AIIMS Hospital. She has assisted State Governments, throughout India, in implementing Fluorosis control programmes. She was the President of the International Society for Fluoride Research (ISFR) for two consecutive terms during 1987 - 1991. She hosted the 13th International Conference of ISFR in New Delhi in November 1983. Professor Susheela is a Fellow of the National Academy of Medical Sciences and a Fellow of the Indian Academy of Sciences. During 1987, she won the prestigious Ranbaxy Research Foundation award in medical sciences. She has published many papers on the subject of fluorosis and recently summarized much of her work in a monograph.

Appendix 2. Dr Paul Connett's '50 Reasons to oppose fluoridation'.

See http://www.fluoridealert.org/50Reasons.htm

Appendix 3. Responses to the York Review.

See http://www.fluoridealert.org/Sheldon.htm

Appendix 4. A chronological listing of the animal, clinical and endemic studies of fluoride and bone, with quotations.

See http://www.slweb.org/f-bone.html

Appendix 5. A chronological listing of the epidemiological hip fracture studies.

Studies Reporting an Association between fluoridated water (< 1.2 ppm fluoride) & hip fracture.

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

b) Cooper C, et al. (1990). Water fluoride concentration and fracture of the proximal femur. J Epidemiol Community Health 44: 17-19.

"We found a significant positive correlation between fluoride levels and discharge rates for hip fracture. This relationship persisted for both women and men... Using an appropriately weighted regression model, there appears to be a positive ecologic association between fluoride levels of county water supplies and fracture discharge rates. This ecologic association is consistent with a recently published study and others currently in progress."

2) Danielson C, et al. (1992). Hip fractures and fluoridation in Utah's elderly population. Journal of the American Medical Association 268(6): 746-748.

"We found a small but significant increase in the risk of hip fracture in both men and women exposed to artificial fluoridation at 1 ppm, suggesting that low levels of fluoride may increase the risk of hip fracture in the elderly."

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 Am. J. Epid. P. S18.

This study found an age-specific, statistically-significant relationship between fluoridation and hip fracture in women 75-84 years old - RR = 1.43 (95% CI, 1.02-1.84). An increase in hip fractures was also found in women aged 85 and older - RR = 1.42 (CI, 0.98 - 1.87).

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.

"In order to assess the association between water fluoridation and hip fracture, we identified 129 counties across the United States considered to be exposed to public water fluoridation and 194 counties without exposure...There was a small statistically significant positive association between fracture rates and fluoridation. The relative risk (95% confidence interval) of fracture in fluoridated counties compared to nonfluoridated counties was 1.08 (1.06 to 1.10) for women and 1.17 (1.13 to 1.22) for men."

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

"This study examines the geographic distribution of hip fracture incidence in the United States at the county level. To this end, data are obtained from the Health Care Financing Administration (HCFA) and the Department of Veteran Affairs that identify all hospital discharges with a diagnosis of hip fracture for women aged 65 years and older for the period 1984 through 1987...After exclusions, 541,985 cases remained eligible for study... There is a weak positive association between the percent of county residents who receive fluoridated water and hip fracture incidence in the unadjusted analysis that is strengthened after adjustment."

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

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).

"We found a higher risk of hip fractures for subjects exposed to fluorine concentrations over 0.11 mg per liter but without a dose-effect relation."

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 Int. 2: 109-117.

" An ecologic study compared fracture rates in 216 counties with natural fluoride levels greater than 0.7 ppm with rates in 95 counties with naturally low fluoride (less than 0.4 PPM) in the drinking water. In general, with increasing dose of fluoride in the drinking water the hip fracture ratio also increased."

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.

"[A]mong younger women, those aged 50-64 years, higher fluoride levels increased the risk of hip fractures."

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 Inflruenbce on Hip Fracture on Bone Health. Osteoporosis Int. 2:109-117.

"The 1985 Fluoridation Census data were used for the 438 counties with populations over 100,000, which represents about 70% of the US population... The percentage of the population that received natural or adjusted fluoride (approximately 1 ppm) was estimated for each county. Medicare data for 1984-1987 were used to calculate the annual incidence of age adjusted hip fractures for white males and females age 65 and older. As the percentage of individuals exposed to fluoridated water increased within a county, the hip fracture rate generally rose for both sexes, but not in a smooth linear fashion... Adjustment for county latitude and longitude produced higher correlation values and significance for females and males."

Studies reporting an association between water-fluoride levels higher than fluoridated water (2 to 4 ppm) & hip fracture.

10) Li Y, et al. (2001). Effect of long-term exposure to fluoride in drinking water on risks of bone fractures. J Bone Miner Res.16(5):932-9.

"In general, the hip fracture prevalence was stable up to 1.06 ppm of fluoride and then appeared to rise, although it did not attain statistical significance until the water fluoride concentration reached 4.32 - 7.97 ppm... The prevalence of hip fractures was highest in the group with the highest water fluoride."

11) 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.

"Residence in the higher-fluoride community was associated with a significantly lower radial bone mass in premenopausal and postmenopausal women, an increased rate of radial bone mass loss in premenopausal women, and significantly more fractures among postmenopausal women."

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. See notes and quotes below.)

12) Cauley J. et al. (1995). Effects of fluoridated drinking water on bone mass and fractures: the study of osteoporotic fractures. J Bone Min Res 10(7): 1076-86.

13) 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(4): 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.

14) 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.

15) 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.

16) Karagas MR, et al. (1996). Patterns of Fracture among the United States Elderly: Geographic and Fluoride Effects. Ann. Epidemiol. 6 (3): 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."

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

18) 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." For a critique of this study, see http://www.fluoridealert.org/phipps.htm

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

Interestingly, while the authors of this study conclude that there is no association between fluoridation and hip fracture, their own data reveals a different picture. Namely, 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)."

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