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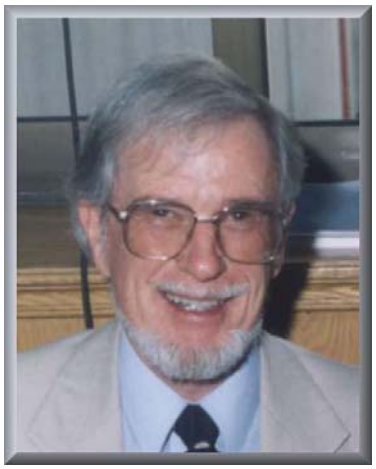
DISCUSSION SECTION

WHY I CHANGED MY MIND ABOUT WATER FLUORIDATION



John Colquhoun *

Former Advocate

To explain how I came to change my opinion about water fluoridation, I must go back to when I was an ardent advocate of the procedure. I now realize that I had learned, in my training in dentistry, only one side of the scientific controversy over fluoridation. I had been taught, and believed, that there was really no scientific case against fluoridation, and that only misinformed lay people and a few crackpot professionals were foolish enough to oppose it. I recall how, after I had been elected to a local government in Auckland (New Zealand's largest city, where I practised dentistry for many years and where I eventually became the Principal Dental Officer) I had fiercely - and, I now regret, rather arrogantly - poured scorn on another Council member (a lay person who had heard and accepted the case against fluoridation) and persuaded the Mayor and majority of my fellow councillors to agree to fluoridation of our water supply.

A few years later, when I had become the city's Principal Dental Officer, I published a paper in the New *Zealand Dental Journal* that reported how children's tooth decay had declined in the city following fluoridation of its water, to which I attributed the decline, pointing out that the greatest benefit appeared to be in low-income areas [1]. My duties as a public servant included supervision of the city's school dental clinics, which were part of a national School Dental Service which provided regular six-monthly dental treatment, with strictly enforced uniform diagnostic standards, to almost a11 (98 percent) school children up to the age of 12 or 13 years. I thus had access to treatment records, and therefore tooth decay rates, of virtually all the city's children. In the study I claimed that such treatment statistics "provide a valid measure of the dental health of our child population" [1]. That claim was accepted by my professional colleagues, and the study is cited in the official history of the New Zealand Dental Association [2].

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INFORMATION CONFIDED

I was so articulate and successful in my support of water fluoridation that my public service superiors in our capital city, Wellington, approached me and asked me to make fluoridation the subject of a world study tour in 1980 – after which I would become their expert on fluoridation and lead a campaign to promote fluoridation in those parts of New Zealand which had resisted having fluoride put into their drinking water.

Before I left on the tour my superiors confided to me that they were worried about some new evidence which had become available: information they had collected on the amount of treatment children were receiving in our school dental clinics seemed to show that tooth decay was declining just as much in places in New Zealand where fluoride had not been added to the water supply. But they felt sure that, when they had collected more detailed information, on *all* children (especially the oldest treated, 12-13 year age group) from *all* fluoridated and *all* nonfluoridated places [3] – in formation which they would start to collect while was I away on my tour – it would reveal that the teeth were better in the fluoridated places: not the 50 to 60 percent difference which we had always claimed resulted from fluoridation, but a significant difference nonetheless. They thought that the decline in tooth decay in the nonfluoridated places must have resulted from the use of fluoride toothpastes and fluoride supplements, and from fluoride applications to the children's teeth in dental clinics, which we had started at the same time as fluoridation. Being a keen fluoridationist, I readily accepted their explanation. Previously, of course, we had assured the public that the only really effective way to reduce tooth decay was to add fluoride to the water supply.

WORLD STUDY TOUR

My world study tour took me to North America, Britain, Europe, Asia, and Australia [4]. In the United States I discussed fluoridation with Ernest Newbrun in San Francisco, Brian Burt in Ann Arbor, dental scientists and officials like John Small in Bethesda near Washington, DC, and others at the Centers for Disease Control in Atlanta. I then proceeded to Britain, where I met Michael Lennon, John Beale, Andrew Rugg-Gunn, and Neil Jenkins, as well as many other scientists and public health officials in Britain and Europe. Although I visited only pro-fluoridation research centers and scientists, I came across the same situation which concerned my superiors in New Zealand. Tooth decay was declining without water fluoridation. Again I was assured, however, that more extensive and thorough surveys would show that fluoridation was the most effective and efficient way to reduce tooth decay. Such large-scale surveys, on very large numbers of children, were nearing completion in the United States, and the authorities conducting them promised to send me the results.

LESSON FROM HISTORY

I now realize that what my colleagues and I were doing was what the history of science shows all professionals do when their pet theory is confronted by disconcerting new evidence: they bend over backwards to explain away the new evidence. They try very hard to keep their theory intact – especially so if their own professional reputations depend on maintaining that theory. (Some time after I graduated in dentistry almost half a century ago, I also graduated in history studies, my special interest being the history of science – which may partly explain my re-examination of the fluoridation theory ahead of many of my fellow dentists.)

So I returned from my study tour reinforced in my pro-fluoridation beliefs by these reassurances from fluoridationists around the world. I expounded these beliefs to my superiors, and was duly appointed chairman of a national "Fluoridation Promotion

Committee." I was instructed to inform the public, and my fellow professionals, that water fluoridation resulted in better children's teeth, when compared with places with no fluoridation.

Surprise: Teeth Better Without Fluoridation?

Before complying, I looked at the new dental statistics that had been collected while I was away for my own Health District, Auckland. These were for all children attending school dental clinics – virtually the entire child population of Auckland. To my surprise, they showed that fewer fillings had been required in the nonfluoridated part of my district than in the fluoridated part. When I obtained the same statistics from the districts to the north and south of mine – that is, from "Greater Auckland," which contains a quarter of New Zealand's population – the picture was the same: tooth decay had declined, but there was virtually no difference in tooth decay rates between the fluoridated and non fluoridated places. In fact, teeth were slightly better in the nonfluoridated areas. I wondered why I had not been sent the statistics for the rest of New Zealand. When I requested them, they were sent to me with a warning that they were not to be made public. Those for 1981 showed that in most Health Districts the percentage of 12- and 13-year-old children who were free of tooth decay – that is, had perfect teeth – was greater in the non-fluoridated part of the district. Eventually the information was published [4].

Over the next few years these treatment statistics, collected for all children, showed that, when similar fluoridated and non-fluoridated areas were compared, child dental health continued to be slightly better in the non-fluoridated areas [5, 6]. My professional colleagues, still strongly defensive of fluoridation, now claimed that treatment statistics did not provide a valid measure of child dental health, thus reversing their previous acceptance of such a measure when it had appeared to support fluoridation.

I did not carry out the instruction to tell people that teeth were better in the fluoridated areas. Instead, I wrote to my American colleagues and asked them for the results of the large-scale surveys they had carried out there. I did not receive an answer. Some years later, Dr John Yiamouyiannis obtained the results by then collected by resorting to the U.S. Freedom of Information Act, which compelled the authorities to release them. The surveys showed that there is little or no differences in tooth decay rates between fluoridated and nonfluoridated places throughout America [7]. Another publication using the same database, apparently intended to counter that finding, reported that when a more precise measurement of decay was used, a small benefit from fluoridation was shown (20 percent fewer decayed tooth surfaces, which is really less than one cavity per child) [8]. Serious errors in that report, acknowledged but not corrected, have been pointed out, including a lack of statistical analysis and a failure to report the percentages of decay-free children in the fluoridated and nonfluoridated areas [7].

Other large-scale surveys from United States, from Missouri and Arizona, have since revealed the same picture: no real benefit to teeth from fluoride in drinking water [9, 10]. For example, Professor Steelink in Tucson, AZ, obtained information on the dental status of all schoolchildren -26,000 of them – as well as information on the fluoride content of Tucson water [10]. He found: "When we plotted the incidence of tooth decay versus fluoride content in a child's neighborhood drinking water, a positive correlation was revealed. In other words, the more fluoride a child drank, the more cavities appeared in the teeth" [11].

From other lands – Australia, Britain, Canada, Sri Lanka, Greece, Malta, Spain, Hungary, and India -a similar situation has been revealed: either little or no relation between water fluoride and tooth decay, or a positive one (more fluoride, more decay) [12-17]. For

example, over 30 years Professor Teotia and his team in India have examined the teeth of some 400,000 children. They found that tooth decay increases as fluoride intake increases. Tooth decay, they decided, results from a deficiency of calcium and an excess of fluoride [17].

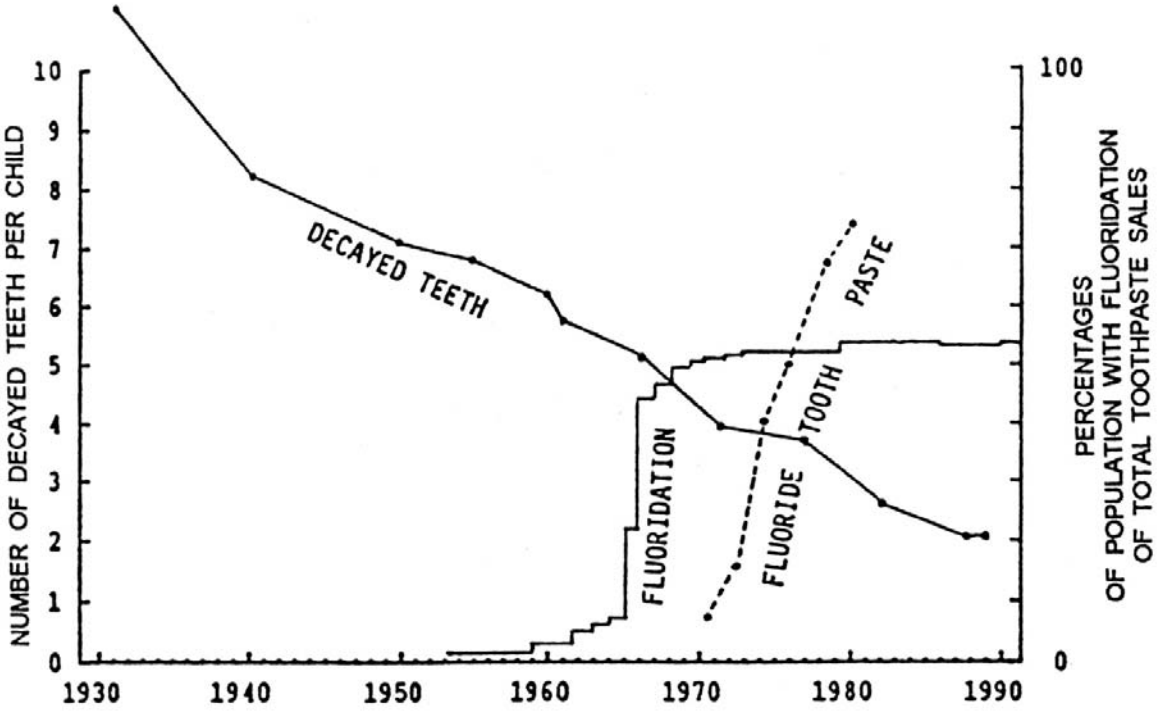

FIGURE 1 - 50-year decline in tooth decay of 5-year-olds.

SOURCE - Compiled from Health Department records of 5-year-olds' tooth decay 1930-1990, fluoridation, and fluoride toothpaste sales.

CAUSE OF DECLINE IN TOOTH DECAY

At first I thought, with my colleagues, that other uses of fluoride must have been the main cause of the decline in tooth decay throughout the western world. But what came to worry me about that argument was the fact that, in the nonfluoridated part of my city, where decay had also declined dramatically, very few children used fluoride toothpaste, many had not received fluoride applications to their teeth, and hardly any had been given fluoride tablets. So I obtained the national figures on tooth decay rates of five-year-olds from our dental clinics which had served large numbers of these children from the 1930s on [18]. They show that tooth decay had started to decline well before we had started to use fluorides (Fig. 1). Also, the decline has continued after all children had received fluoride all their lives, so the continuing decline could not be because of fluoride. The fewer figures available for older children are consistent with the above pattern of decline [18]. So fluorides, while possibly contributing, could not be the main cause of the reduction in tooth decay.

So what did cause this decline, which we find in most industrialized countries? I do not know the answer for sure, but we do know that after the Second World War there was a rise in the standard of living of many people. In my country there has been a tremendous increase in the consumption of fresh fruit and vegetables since the 1930s, assisted by the introduction of household refrigerators [19]. There has also been an eightfold increase in the consumption per head of cheese, which we now know has anti-decay properties [19, 20]. These nutritional

changes, accompanied by a continuing decline in tooth decay, started before the introduction of fluorides.

The influence of general nutrition in protection against tooth decay has been well described in the past [21], but is largely ignored by the fluoride enthusiasts, who insist that fluorides have been the main contributor to improved dental health. The increase in tooth decay in third-world countries, much of which has been attributed to worsening nutrition [22], lends support to the argument that improved nutrition in developed countries contributed to improved dental health.

Flawed Studies

The studies showing little if any benefit from fluoridation have been published since 1980. Are there contrary findings? Yes: many more studies, published in dental professional journals, claim that there is a benefit to teeth from water fluoride. An example is a recent study from New Zealand [23], carried out in the southernmost area of the country [23]. Throughout New Zealand there is a range of tooth decay rates, from very high to very low, occurring in both fluoridated and nonfluoridated areas. The same situation exists in other countries.

What the pro-fluoride academics at our dental school did was to select from that southern area four communities: one nonfluoridated, two fluoridated, and another which had stopped fluoridation a few years earlier. Although information on decay rates in all these areas was available to them, from the school dental service, they chose for their study the one nonfluoridated community with the highest decay rate and two fluoridated ones with low decay rates, and compared these with the recently stopped fluoridated one, which happened to have medium decay rates (both before and after it had stopped fluoridation). The teeth of randomly selected samples of children from each community were examined. The chosen communities, of course, had not been randomly selected. The results, first published With much publicity in the news media, showed over 50 percent less tooth decay in the fluoridated communities, with the recently defluoridated town in a "middle" position (see left side of Fig. 2). When I obtained the decay rates for *all* children in *all* the fluoridated and *all* the nonfluoridated areas in that part of New Zealand, as well as the decay rates for *all* children in the recently defluoridated town, they revealed that there are virtually no differences in tooth decay rates related to fluoridation (see right side of Fig. 2).

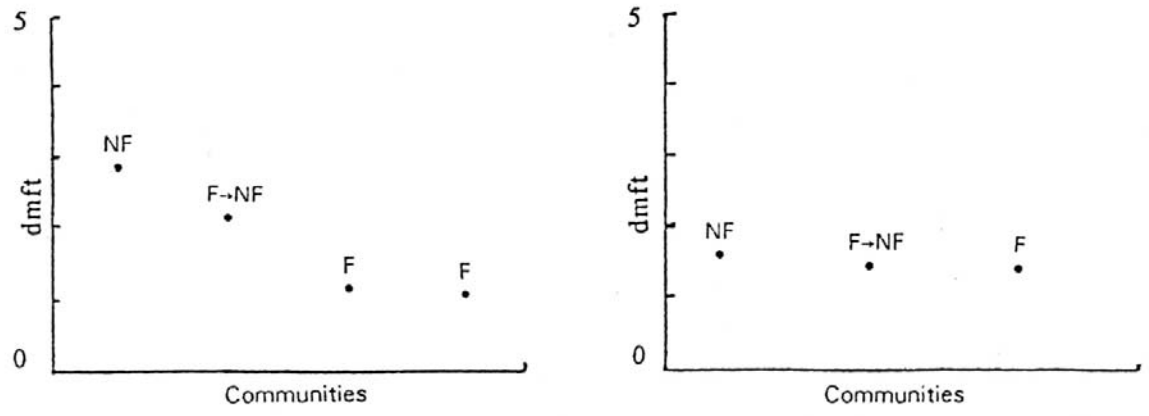

FIGURE 2. – Left: results of South Island dental survey of samples of 5-year-olds from selected areas. See [23]. Right: Results for all 5-year-olds in all nonfluoridated, fluoridated, and defluoridated areas. (School Dental Service records).

When I confronted the authors with this information, they retorted that the results of their study were consistent with other studies. And of course it is true that many similar studies have been published in the dental professional literature. It is easy to see how the consistent results are obtained: an appropriate selection of the communities being compared. There is another factor: most pro-fluoridation studies (including this New Zealand one) were not "blind" – that is, the examiners knew which children received fluoride and which did not. Diagnosis of tooth decay is a very subjective exercise, and most of the examiners were keen fluoridationists, so it is easy to see how their bias could affect their results. It is just not possible to find a blind fluoridation study in which the fluoridated and nonfluoridated populations were similar and chosen randomly.

EARLY FLAWED STUDIES

One of the early fluoridation studies listed in the textbooks is a New Zealand one, the "Hastings Fluoridation Experiment" (the term "experiment" was later dropped because the locals objected to being experimented on) [24]. I obtained the Health Department's fluoridation files under my own country's "Official Information" legislation. They revealed how a fluoridation trial can, in effect, be rigged [25]. The school dentists in the area of the experiment were instructed to change their method of diagnosing tooth decay, so that they recorded much less decay after fluoridation began. Before the experiment they had filled (and classified as "decayed") teeth with any small catch on the surface, before it had penetrated the outer enamel layer. After the experiment began, they filled (and classified as "decayed") only teeth with cavities which penetrated the outer enamel layer. It is easy to see why a sudden drop in the numbers of "decayed and filled" teeth occurred. This change in method of diagnosis was not reported in any of the published accounts of the experiment.

Another city, Napier, which was not fluoridated but had otherwise identical drinking water, was at first included in the experiment as an "ideal control" – to show how tooth decay did not decline the same as in fluoridated Hastings. But when tooth decay actually declined more in the nonfluoridated control city than in the fluoridated one, in spite of the instructions to find fewer cavities in the fluoridated one, the control was dropped and the experiment proceeded with no control. (The claimed excuse was that a previously unknown trace element, molybdenum, had been discovered in some of the soil of the control city, making tooth decay levels there unusually low [26], but this excuse is not supported by available information, from the files or elsewhere, on decay levels throughout New Zealand).

The initial sudden decline in tooth decay in the fluoridated city, plus the continuing decline which we now know was occurring everywhere else in New Zealand, were claimed to prove the success of fluoridation. These revelations from government files were published in the international environmental journal, *The Ecologist*, and presented in 1987 at the 56th Congress of the Australian and New Zealand Association for the Advancement of Science [27].

When I re-examined the classic fluoridation studies, which had been presented to me in the text books during my training, I found, as others had before me, that they also contained serious flaws [28-30]. The earliest set, which purported to show an inverse relationship between tooth decay prevalence and naturally occurring water fluoride concentrations, are flawed mainly by their nonrandom methods of selecting data. The later set, the "f1uoridation trials" at Newburgh, Grand Rapids, Evanston, and Brantford, displays inadequate baselines, negligible statistical analysis, and especially a failure to recognize large variations in tooth decay prevalence in the control communities. We really cannot know whether or not some of the tooth decay reductions reported in those early studies were due to water fluoride.

I do not believe that the selection and bias that apparently occurred was necessarily deliberate. Enthusiasts for a theory can fool themselves very often, and persuade themselves and others that their activities are genuinely scientific. I am also aware that, after 50 years of widespread acceptance and endorsement of fluoridation, many scholars (including the reviewers of this essay) may find it difficult to accept the claim that the original fluoridation studies were invalid. That is why some of us, who have reached that conclusion, have submitted an invitation to examine and discuss new and old evidence "in the hope that at least some kind of scholarly debate will ensue" [31].

However, whether or not the early studies were valid, new evidence strongly indicates that water fluoridation today is of little if any value. Moreover, it is now widely conceded that the main action of fluoride on teeth is a topical one (at the surface of the teeth), not a systemic one as previously thought, so that there is negligible benefit from swallowing fluoride [32].

Harm from Fluoridation

The other kind of evidence which changed my mind was that of *harm* from fluoridation. We had always assured the public that there was absolutely no possibility of any harm. We admitted that a small percentage of children would have a slight mottling of their teeth, caused by the fluoride, but this disturbance in the formation of tooth enamel would, we asserted, be very mild and was nothing to worry about. It was, we asserted, not really a sign of toxicity (which was how the early literature on clinical effects of fluoride had described it) but was only at most a slight, purely cosmetic change, and no threat to health. In fact, we claimed that only an expert could ever detect it.

HARM TO TEETH

So it came as a shock to me when I discovered that in my own fluoridated city some children had teeth like those in Fig. 3. This kind of mottling answered the description of dental fluorosis (bilateral diffuse opacities along the growth lines of the enamel). Some of the children with these teeth had used fluoride toothpaste and swallowed much of it. But I could not find children with this kind of fluorosis in the nonfluoridated parts of my Health District, except in children who had been given fluoride tablets at that time.

I published my findings: 25 percent of children had dental fluorosis in fluoridated Auckland and around 3 percent had the severer (discolored or pitted) degree of the condition [33]. At first the authorities vigorously denied that fluoride was causing this unsightly mottling. However, the following year another Auckland study, intended to discount my finding, reported almost identical prevalences and severity, and recommended lowering the water fluoride level to below 1 ppm [34]. Others in New Zealand and the United States have reported similar findings. All these studies were reviewed in the journal of the International Society for Fluoride Research [35]. The same unhappy result of systemic administration of fluoride has been reported in children who received fluoride supplements [36]. As a result, in New Zealand as elsewhere, the doses of fluoride tablets were drastically reduced, and parents were warned to reduce the amount of fluoride toothpaste used by their children, and to caution them not swallow any. Fluoridationists would not at first admit that fluoridated water contributed to the unsightly mottling – though later, in some countries including New Zealand, they also recommended lowering the level of fluoride in the water. They still insist that the benefit to teeth outweighs any harm.

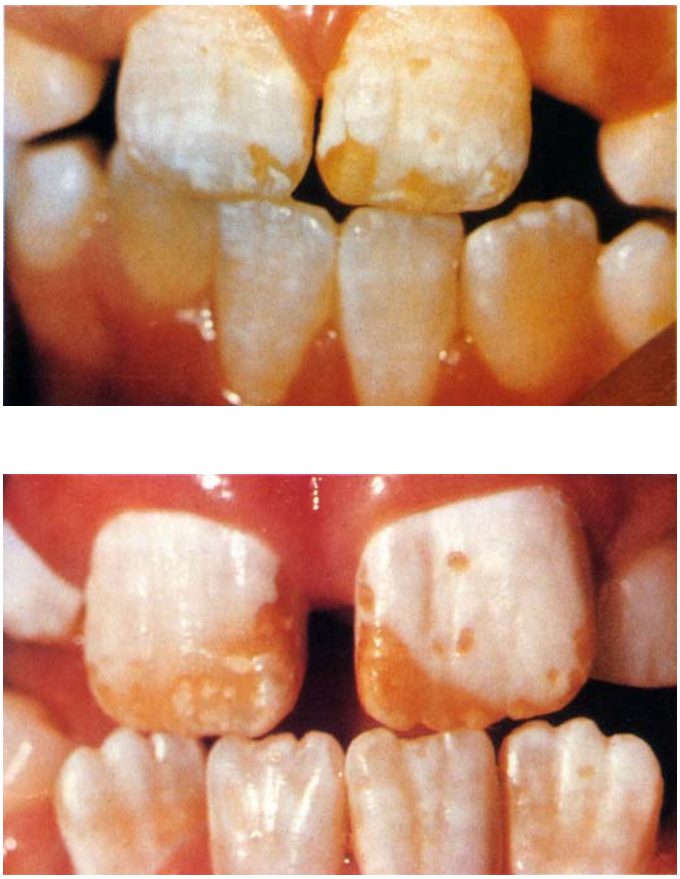

Figure 3. – Examples of dental fluorosis in 8- and 9-year old children who grew up in fluoridated Auckland, New Zealand.

WEAKENED BONES

Common sense should tell us that if a poison circulating in a child's body can damage the tooth-forming cells, then other harm also is likely. We had always admitted that fluoride in excess can damage bones, as well as teeth.

By 1983 I was thoroughly convinced that fluoridation caused more harm than good. I expressed the opinion that some of these children with dental fluorosis could, just possibly, have also suffered harm to their bones [Letter to Auckland Regional Authority, January 1984]. This opinion brought scorn and derision: there was absolutely no evidence, my dental colleagues asserted, of any other harm from low levels of fluoride intake, other than mottling of the teeth.

Six years later, the first study reporting an association between fluoridated water and hip fractures in the elderly was published [37]. It was a large-scale one. Computerization has made possible the accumulation of vast data banks of information on various diseases. Hip fracture rates have increased dramatically, independently of the increasing age of populations. Seven other studies have now reported this association between low water fluoride levels and hip fractures [38-44]. Have there been contrary findings? Yes; but most of the studies claiming no association are of small numbers of cases, over short periods of time, which one would not expect to show any association [45, 46]. Another, comparing a fluoridated and a nonfluoridated Canadian community, also found an association in males but not in females, which hardly proves there is no difference in all cases [47]. Our fluoridationists claim that the

studies which do show such an association are only epidemiological ones, not clinical ones, and so are not conclusive evidence.

But in addition to these epidemiological studies, clinical trials have demonstrated that when fluoride was used in an attempt to treat osteoporosis (in the belief it strengthened bones), it actually caused more hip fractures [48-52]. That is, when fluoride accumulates in bones, it weakens them. We have always known that only around half of any fluoride we swallow is excreted in our urine; the rest accumulates in our bones [53, 54]. But we believed that the accumulation would be insignificant at the low fluoride levels of fluoridated water. However, researchers in Finland during the 1980s reported that people who lived 10 years or more in that country's one fluoridated city, Kuopio, had accumulated extremely high levels of fluoride in their bones – thousands of parts per million – especially osteoporosis sufferers and people with impaired kidney function [55, 56]. After this research was published, Finland stopped fluoridation altogether. But that information has been ignored by our fluoridationists.

BONE CANCER?

An association with hip fracture is not the only evidence of harm to bones from fluoridation. Five years ago, animal experiments were reported of a fluoride-related incident of a rare bone cancer, osteosarcoma, in young male rats [57]. Why only the male animals got the bone cancer is not certain, but another study has reported that fluoride at very low levels can interfere with the male hormone, testosterone [58]. That hormone is involved in bone growth in males but not in females.

This finding was dismissed by fluoridation promoters as only "equivocal evidence," unlikely to be important for humans. But it has now been found that the same rare bone cancer has increased dramatically in young human males – teenage boys aged 9 to 19 – in fluoridated areas of America but not in the nonfluoridated areas [59]. The New Jersey Department of Health reported osteosarcoma rates were three to seven times higher in its fluoridated areas than in its nonfluoridated areas [60].

Once again, our fluoridationists are claiming that this evidence does not "conclusively" demonstrate that fluoride caused the cancers, and they cite small-scale studies indicating no association. One study claimed that fluoride might even be protective against osteosarcoma [61]; yet it included only 42 males in its 130 cases, which meant the cases were not typical of the disease, because osteosarcoma is routinely found to be more common in males. Also, the case-control method used was quite inappropriate, being based on an assumption that if ingested fluoride was the cause, osteosarcoma victims would require higher fluoride exposure than those without the disease. The possibility that such victims might be more susceptible to equal fluoride exposures was ignored. All these counter-claims have been subjected to critical scrutiny which suggests they are flawed [62, 63]. Nonetheless, the pro- fluoride lobbyists continue to insist that water fluoridation should continue because, in their view, the benefits to teeth outweigh the possibility of harm. Many dispute that assessment.

OTHER EVIDENCE OF HARM

There is much more evidence that tooth mottling is not the only harm caused by fluoridated water. Polish researchers, using a new computerized method of X-ray diagnosis, reported that boys with dental fluorosis also exhibit bone structure disturbances [64]. Even more chilling is the evidence from China that children with dental fluorosis have on average lower intelligence scores [65, 66]. This finding is supported by a recently published animal experiment in America, which showed that fluoride also accumulated in certain areas of the brain, affecting behavior and the ability to learn [67].

Endorsements Not Universal

Concerning the oft-repeated observation that fluoridation has enjoyed overwhelming scientific endorsement, one should remember that even strongly supported theories have eventually been revised or replaced. From the outset, distinguished and reputable scientists opposed fluoridation, in spite of considerable intimidation and pressure [68, 69].

Most of the world has rejected fluoridation. Only America where it originated, and countries under strong American influence persist in the practice. Denmark banned fluoridation when its National Agency for Environmental Protection, after consulting the widest possible range of scientific sources, pointed out that the long-term effects of low fluoride intakes on certain groups in the population (for example, persons with reduced kidney function), were insufficiently known [70]. Sweden also rejected fluoridation on the recommendation of a special Fluoride Commission, which included among its reasons that: 'The combined and long-term environmental effects of fluoride are insufficiently known" [71]. Holland banned fluoridation after a group of medical practitioners presented evidence that it caused reversible neuro-muscular and gastrointestinal harm to some individuals in the population [72].

Environmental scientists, as well as many others, tend to doubt fluoridation. In the United States, scientists employed by the Environmental Protection Agency have publicly disavowed support for their employer's pro-fluoridation policies [73]. The orthodox medical establishment, rather weak or even ignorant on environmental issues, persist in their support, as do most dentists, who tend to be almost fanatical about the subject. In English-speaking countries, unfortunately, the medical profession and its allied pharmaceutical lobby (the people who sell fluoride) seem to have more political influence than environmentalists.

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Discussion: Critique

The following **CRITICAL REVIEW**, by a professor at the School of Dentistry in San Francisco, has been sent to this journal. Dr Colquhoun's response follows.

WHY I CHANGED MY MIND ABOUT WATER FLUORIDATION by John Colquhoun

Perspectives in Biology and Medicine 41 (1) 29-44 Autumn 1997

Reviewed by Howard Pollick BDS MPH

I find this publication informational but biased in its details. I cannot conclude that water fluoridation is ineffective or harmful based on this paper, although Colquhoun attempts to persuade the reader of the correctness of his views. I base these conclusions on the following points.

1. In New Zealand there is a national School Dental Service that provided regular six-monthly dental treatment, with strictly enforced uniform diagnostic standards, to almost all (98 percent) school children up to the age of 12 or 13 years.

Over the next few years, these treatment statistics, collected for all children, showed that when similar fluoridated or unfluoridated areas were compared, child dental health continued to be slightly better in the fluoridated areas.

These are quotes from this paper which show that New Zealand has had a school-based dental treatment program and that, at least at one period of time Colquhoun acknowledges that fluoridation appeared to be of benefit. The school- based program provides treatment and prevention services, including application of fluorides and is a model of dental services that does not exist in many parts of the world, and not in California. It appears that Colquhoun has demonstrated that the school-based dental service, in part, has provided what fluoridation can provide. Colquhoun however does not address the issue of cost-effectiveness and presumably the dental service is far more expensive than fluoridation.

2. In comparing the work by Yiamouyiannis and that by Brunelle and Carlos of the U.S. 1986-87 national study on dental caries, Colquhoun states: 'Another publication using the same data base (Brunelle and Carlos) apparently intended to counter that findings (by Yiamouyiannis) reported that when a more precise measurement of decay was used, a small benefit from fluoridation was shown (20 percent fewer decayed tooth surfaces, which is really less than one cavity per child).' Since the publication by Yiamouyiannis includes an analysis of the publication by Brunelle and Carlos, then the journal Fluoride must have published Yiamouyiannis after the Journal of Dental Research published Brunelle and Carlos. Thus Colquhoun's conclusion that Brunelle and Carlos 'apparently intended to counter that findings (by Yiamouyiannis)' is misleading.

Colquhoun appears to have implied that because he didn't receive a reply from his American colleagues about the result of the national U.S. study and because Yiamouyiannis obtained the results 'resorting to the U.S. Freedom of Information Act' that the results would otherwise have not been published. On the contrary, they were published in 1989. (JA Brunelle. *Oral health of United States children, national and regional findings.* DBSS Publication No. (NIB) 89- 2247, U.S. Government Printing Office, Washington, DC, 1989.)

Thus it appears that Colquhoun fuels the fires of an alleged conspiracy at the highest levels of dental research in the U.S. to withhold information from the public and to bias the findings.

3. Colquhoun refers to some authors by name without title and others by name with titles, with those whose work supports that of Colquhoun with their title, for example 'Dr John Yiamouyiannis', 'Professor Steelink' and 'Professor Teotia', as if by adding their title they are afforded a higher place in Colquhoun's hierarchy. This indicates bias.

4. The work of Teotia in India concerns areas with very high fluoride beyond the recommended concentrations for water fluoridation.

5. Colquhoun suggests that the improved nutrition (without regard to sugar consumption) in most industrialized countries that has occurred since the 1930s and after World War II may be more important than fluorides in the prevention of tooth decay. However, he acknowledges that '1 do not know the answer for sure', and indeed does not refer to any epidemiologic studies that have shown that general nutrition is a greater factor than fluorides in the prevention of tooth decay in communities where people do not generally suffer from malnutrition.

Malnutrition that accompanies wartime periods and remote and isolated communities and countries such as New Zealand in the 1930's has been linked with increases and decreases in tooth decay prevalence, depending on the availability of sugar. Colquhoun rightly points to the increase in tooth decay in third-world countries, much of which has been attributed to worsening nutrition.

6. Colquhoun points out 'the studies showing little if any benefit from fluoridation have been published since 1980. Are there contrary findings? Yes: many more studies, published in dental professional journals, claim that there is a benefit to teeth from water fluoride.' However Colquhoun asserts that in all of the studies published in dental professional journals there is bias in population selection and examiner diagnosis and that 'most of the examiners were keen fluoridationists'.

It was my experience in recruiting and standardizing examiners for the California Oral Health Needs Assessment of Children, 1993-94, that none of the examiners could be described as 'keen fluoridationists'.

To overcome the potential of bias on the part of examiners, a study in England transported children from fluoridated and nonfluoridated communities to the examination site and had children wear a smock over their school uniforms so that the examiners would be 'blind' to where the children came from. That study found a benefit of fluoridation in the prevention of tooth decay in children who had been exposed to fluoridation for the first time after the age of 12 years. That study demonstrated that fluoridation does not have to be consumed from infancy to be of benefit. (Hardwick JL, Teasdale J, Bloodworh G. Caries increments over 4 years in children aged 12 at the start of water fluoridation. *British Dental Journal 153* (6) 217-222 1982).

This, and other studies have shown that fluoridation of water supplies exerts a benefit systemically as well as topically. The salivary concentration of fluoride is higher in fluoridated areas, and the fluoride in saliva exerts a topical effect on teeth to remineralize the enamel to prevent tooth decay.

However, Colquhoun asserts that fluoridation is of 'little if any value' and 'there is negligible benefit from swallowing fluoride'.

7. Colquhoun in discussing fluorosis states: 'Some of these children with these teeth had used fluoride toothpaste and swallowed much of it. But I could not find children with this kind of fluorosis in the nonfluoridated parts of my Health District, except in children who had been given fluoride tablets at the recommended dose at the time.'

In this matter there is general consensus that recent increases in dental fluorosis are attributed to fluoride toothpaste being swallowed by very young children and by fluoride tablets being originally at too high a dose and also being inappropriately prescribed or ingested

more frequently than recommended. As a consequence, recommendations have been made to limit the amount of fluoride toothpaste used to a 'pea-size' amount and a lowering of the recommended dose of fluoride supplementation in nonfluoridated communities.

However the influence of fluoride in water fluoridation as being harmful in this way is not generally accepted.

8. The evidence on the role of water fluoridation and hip fracture has been the subject of a number of reviews. 'With respect to hip fractures and bone health, there is no scientific evidence for altering current public health policy on the use of fluorides for caries prevention'. (WHO Expert Committee on Oral Health Status and Fluoride Use. *Fluorides and Oral Health*, WHO technical report series #846, World Health Organization. 1994).

A 1993 review for the U.S. National Research Council addressed fluoride and bone fractures. Of six epidemiological studies using geographic comparison with no actual fluoride intake data, four found a weak association. Of two additional studies examining before and after fluoridation data, one found a negative association and the other no association. One additional essentially geographic comparison found increased risk of hip fracture at 4 mg/L and another where individual exposure data were collected showed no difference in risk.

With regard to animal studies, 'the subcommittee concluded that the weight of evidence indicates that bone strength is not adversely affected in animals that are fed a nutritiously adequate diet unless there is long-term ingestion of fluoride at concentrations of at least 50 mg/L of drinking water or 50 mg/kg in diet.' (National Academy Press:' *Health Effects of Ingested Fluoride;* Subcommittee on Health Effects of Ingested Fluoride; Committee on Toxicology, National Research Council; 1993.)

Colquhoun, in his review of fluoride and hip fractures, points to studies published in 1989-90 indicating high doses of fluoride used to treat osteoporosis 'actually caused more hip fractures.'

In recognition of the results of those studies, a lowered protocol of fluoride was used with successful results, which Colquhoun has not included in his 1997 publication. (CYC Pak et *al.* Slow-release sodium fluoride in the management of postmenopausal osteoporosis. Annals of Internal Medicine. April 15, 1994 Vol 120: No.8. p 625.) These authors concluded that their regimen of 'intermittent slow-release sodium fluoride plus continuous calcium citrate, administered for about 2.5 years, inhibits new vertebral fractures, increases the mean spinal bone mass without decreasing the radial shaft bone density, and is safe to use.' No patient in either experimental or control group developed microfractures, or blood loss anemia. These patients received 25 mg slow-release NaF twice daily in repeated 14 month cycles of 12 months on and 2 months off treatment, compared to a placebo, with both groups receiving 400 mg calcium twice daily.)

In recognition of the study by Pak et *al*, the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIM) issued a press release stating: This regimen (CYC Pak et *al.* Slow-release sodium fluoride in the management of post-menopausal osteoporosis. Annals of Internal Medicine. April 15, 1994 Vol120: No. 8. p 625.) supports the use of fluoride at high doses for this condition, but has no bearing on fluoridation of water supplies. It adds weight to the hypothesis that there is no positive association between fluoride intake and bone fracture. (National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIM): Fluoride offers hope for treating osteoporosis. Press Release. April 14, 1994.)

9. With regard to studies in Finland, the researchers who had examined autopsy samples of the anterior iliac crest in 1980 and found the highest fluoride content of bone ash from women with severe osteoporosis, also found in 1986 that hip fracture incidence was <u>not</u> affected by fluoridation (Arnala I, Alhava EM, Kivivuori R, Kauranen P. Hip fracture incidence not

affected by fluoridation. Osteofluorosis studied in Finland. *Acta Orthopaedica* 57 (4) 344-349 August 1986). However, Colquhoun did not include this later study in his publication.

Abstract: Iliac crest biopsies were taken from patients with hip fracture from a low-fluoride area (less than 0.3 ppm), from an area with fluoridated drinking water (1.0-1.2 ppm), and from a high-fluoride area (greater than 1.5 ppm). Fluoride content analysis and histomorphometry of bone were performed. The hip fracture incidence during 1972-1981 was studied in the same areas. The fluoride content of the bone samples correlated with drinking water fluoride. In patients with hip fracture, both osteomalacia and osteoporosis were common. In the high-fluoride area also osteofluorosis was found in many patients. Osteo-fluorosis may occur if the fluoride content of trabecular bone exceeds 4,000 ppm and either the volumetric density of osteoid or osteoid-covered trabecular bone surface is abnormally increased. There was no difference in incidence of hip fracture in the three areas.

10. A 1996 review on water fluoridation and osteoporotic fractures is included here as background on this subject: Hillier S, Inskip E, Coggon D, Cooper C. Water fluoridation and osteoporotic fracture. *Community Dental Health* Suppl 2 63-68 September 13 1996.

Abstract: Osteoporotic fractures constitute a major public health problem. These fractures typically occur at the hip, spine and distal forearm. Their pathogenesis is hetero-genous, with contributions from both bone strength and trauma. Water fluoridation has been widely proposed for its dental health benefits, but concerns have been raised about the balance of skeletal risks and benefits of this measure. Fluoride has potent effects on bone cell function, bone structure and bone strength. These effects are mediated by the incorporation of fluoride ions in bone crystals to form fluoroapatite, and through an increase in osteoblast activity. It is believed that a minimum serum fluoride level of 100 ng/m1 must be achieved before osteoblasts will be stimulated. Serum levels associated with drinking water fluoridated to 1 ppm are usually several times lower than this value, but may reach this threshold at concentrations of 4 ppm in the drinking water. Animal studies suggest no effect of low-level (0-3 ppm) fluoride intake on bone strength, but a possible decrease at higher levels. Sodium fluoride has been used to treat established osteoporosis for nearly 30 years. Recent trials of this agent, prescribed at high doses, have suggested that despite a marked increase in bone mineral density, there is no concomitant reduction in vertebral fracture incidence. Furthermore, the increase in bone density at the lumbar spine may be achieved at the expense of bone mineral in the peripheral cortical skeleton. As a consequence, high dose sodium fluoride (80 mg daily) is not currently used to treat osteoporosis. At lower doses, recent trials have suggested a beneficial effect on both bone density and fracture. The majority of epidemiological evidence regarding the effects of fluoridated drinking water on hip fracture incidence is based on ecological comparisons. Although one Finnish study suggested that hip fracture rates in a town with fluoridated water were lower than those in a matching town without fluoride, a later study failed to show differences. Ecological studies from the United States and Great Britain have, if anything, revealed a weak positive association between water fluoride concentration and hip fracture incidence. Two studies examining hip fracture rates before and after fluoridation yielded discordant results, and are complicated by underlying time trends in hip fracture incidence. Only two studies have attempted to examine the relationship between water fluoride concentration and fracture risk at an individual level. In one of these, women in as high fluoride community had double the fracture risk of women in a low fluoride community. In the other, there was no relationship between years of fluoride exposure and incidence of spine and non-spine fractures. In conclusion, the epidemiological evidence relating water fluoridation to hip fracture is based upon ecological comparisons and is inconclusive. However several studies suggest the possibility of a weak adverse effect, which warrants further exploration. Data on the relationship between fluoride intake and hip fracture risk at the individual level, and data relating fluoridation to bone mineral density are required. Until these become available, the burden of evidence suggesting that fluoridation might be a

risk factor for hip fracture is weak and not sufficient to retard the progress of the water fluoridation programs.

11. A 1997 review on osteoporosis, prevention, diagnosis and management is included here as background to this subject: Deal CL. Osteoporosis, prevention, diagnosis and management. American Journal of Medicine 102 (1A) 35S-39S January 27 1997.

Abstract: Osteoporosis is a public health scourge that is usually eminently preventable. Some risk factors, such as low calcium intake, vitamin D deficiency, and physical inactivity, are amenable to early interventions that will help maximise peak bone density. Other risk factors subject to modification are cigarette smoking and excessive consumption of protein, caffeine, and alcohol. Hip fractures are the most serious outcome of osteoporosis, with enormous personal and public health consequences. The ongoing Study of Osteoporotic Fractures has identified additional independent predictors of hip fracture risk, including maternal hip fractures, absence of significant weight gain since age 25, height, hyperthyroidism, use of long-acting benzodiazepines or anticonvulsants, spending < 4 hours per day on one's feet, inability to rise from a chair without using one's arms, poor visual depth perception and contrast sensitivity and tachycardia. In an individual perimenopausal woman, the risk of osteoporotic fracture and the urgency of oestrogen replacement therapy can be best estimated on the basis of bone mineral density as measured by dual-energy x-ray absorptiometry , coupled with the presence or absence of existing fractures and clinical risk factors evident from the history and physical examination. Estrogen, calcitonin and bisphosphonates have all proved effective in retarding postmenopausal bone loss and therefore reducing the risk of fracture. The use of sodium fluoride is more controversial, although a recent study has suggested a possible role for slow-release fluoride combined with high-dose calcium supplementation.

12. Colquhoun used a reference to state: Five years ago, animal experiments were reported of a fluoride-related incident of a rare bone cancer, called osteosarcoma, in young male rats. However that reference did not show any link between fluoride and cancer. It appears that there must have been an error in the publication of Colquhoun's paper.

The cited reference is: Maurer JR; Chang MC; Soysen AG; Anderson RL. Two- year carcinogenicity study of sodium fluoride in rats (see comments). *Journal of the National Cancer Institute* 82 (13) 1118-1126 July 4 1990.

Abstract: To determine the carcinogenic potential of sodium fluoride (NaF), we fed Sprague-Dawley rats a diet containing NaF for up to 99 weeks. Rats receiving NaF at a dose of 4, 10, or 25 mg/kg per day added to a low-fluoride diet were compared with controls receiving either a low-fluoride diet or laboratory chow. Each treatment group consisted of 70 rats of each sex. A 30% decrement in weight gain occurred at an NaF dose of 25 mg/kg per day. Evidence of fluoride toxicity was seen in the teeth, bones, and stomach, and the incidence and severity of these changes were related to the dose of NaF and the duration of exposure. Despite clear evidence of toxicity, NaF did not alter the incidence of preneoplastic and neoplastic lesions at any site in rats of either sex. Results from this study indicate that NaF is not carcinogenic in Sprague-Dawley rats.

The 'equivocal' finding that Colquhoun refers to is of a different rat study by the National Toxicology Program, and the designation 'equivocal' was based on the findings and not on any potential importance to humans.

Colquhoun's interpretation that: 'But now it has been found that the same rare bone cancer has increased dramatically in young human males -teenage boys aged nine to 19 -in the fluoridated areas of America but not in the unfluoridated areas' is different from that of the authors of that study. Indeed the authors of the New Jersey study referenced by Colquhoun stated: 'Therefore, taking both studies together (with reference to the other study by Hoover

RN, Devesa S, Cantor K and Fraumeni JF. Time trends for bone and joint cancers and osteosarcoma in the surveillance, epidemiology and end results (SEER) program. In: *Review of Fluoride. Benefits and Risks.* Appendix F. U .S. Department of Health and Human Services, Public Health Service. Washington DC. 1991.) there is insufficient basis to draw conclusions about whether osteosarcoma incidence and fluoridation are causally linked.' (FD Cohn. An *epidemiologic report on drinking water and fluoridation. Environmental Health Service.* New Jersey Department of Health, November 1992.)

Another reference that Colquhoun did not include, perhaps because it didn't support his thesis, is: Gelberg KH; Fitzgerald EF; Hwang SA; Dubrow R. Fluoride exposure and childhood osteosarcoma; a case control study. *American Journal of Public Health* 83 (12) 1678-1683 December 1995.

Abstract: OBJECTIVES. This study tests the hypothesis that fluoride exposure in a nonoccupational setting is a risk factor for childhood osteosarcoma. METHODS. A population-based case-control study was conducted among residents of New York State, excluding New York City. Case subjects (n = 130) were diagnosed with osteosarcoma between 1978 and 1988, at age 24 years or younger. Control subjects were matched to case subjects on year of birth and sex. Exposure information was obtained by a telephone interview with the subject, parent or both. RESULTS. Based on the parent's responses, total lifetime total lifetime fluoride exposure was not significantly associated with osteosarcoma among all subjects combined or among females. Protective trends were observed for fluoridated toothpaste, fluoride tablets, and dental fluoride treatment among all subjects and among males. Based on the subjects responses, no significant associations between fluoride exposure and osteosarcoma were observed. CONCLUSIONS. Fluoride exposure does not increase the risk of osteosarcoma and may be protective in males. The protective effect may not be directly due to fluoride exposure but to other factors associated with good dental hygiene. There is also biologic plausibility for a protective effect.

13. Additionally, only high levels of fluoride have been associated with reductions in testosterone levels in contrast to Colquhoun's assertion that 'very low levels can interfere with the male hormone testosterone.'

14. With regard to studies from China that children with dental fluorosis have on average lower intelligence scores, Colquhoun misstates the facts of the research. In the study by Li (Li. Effect of fluoride exposure on intelligence in children. *Fluoride* 28 (4) pages 189-192 1995) there were four communities with a Community Fluorosis Index (CFI) of <0.4, 0.8, 2,5, and 3.2. Only in the two communities with the highest CFI were lower IQs found. This CFI index was developed by Dean (Dean HT .The investigation of physiological effects by the epidemiological method. In: Moulton FR (Ed). *Fluorine and Dental Health.* Am. Assoc. Adv. Sci., Washington 1942 pp 23-31). Dean stated that a CFI below 0.4 is of little or no public health concern; that the range from 0.4 to 0.6 is borderline; and that for indexes above 0.6 removal of excess fluoride from the water is recommended. Thus the communities where Li found lower intelligence scores were in areas of exceptionally high fluoride, apparently due to high fluoride coal being burned and inhaled, rather than in the water. Thus these findings are un- related to water fluoridation and to suggest, as Colquhoun does, that children with dental fluorosis have on average lower intelligence scores is a gross misstatement of the facts, since there are children with very mild and mild dental fluorosis, living in the low CFI communities where IQ scores were apparently normal.

15. Colquhoun supports his contention that fluoride is harmful by citing the reference by Mullenix et *al.* (Mullenix PJ, Denbesten PK, Schunior A, Kernan WJ. Neurotoxicity of sodium fluoride in rats. *Neurotoxicology and Teratology* 17 (2) 169-177 March-April 1995.) In this study pregnant rats were injected with very high doses of sodium fluoride at 0.13 mg/kg. This would be equivalent of a human dose for a pregnant woman of 6.5 mg, not to be

swallowed and therefore diluted by the body, but injected subcutaneously. This would be unconscionable in humans who are never injected with sodium fluoride. Then the weanlings (baby rats) drank water with 0, 75, 100, or 125 ppm F for 6 or 20 weeks, and the 3 month-old adults received water containing 100 ppm F for 6 weeks. Therefore all rats drank water with at least 100 times the recommended concentration for water fluoridation for extended periods of time. Thus this study was never intended to determine the effects of water fluoridation.

16. Colquhoun consistently alleges harm from fluoride without stating the dose or concentration of fluoride, which is absolutely essential in a discussion of the toxicology of fluoride.

This concludes my review.

RESPONSE TO CRITIQUE OF HOWARD POLLICK

John Colquhoun

Pollack argues mainly by quoting fluoridationist opinions, rather than evidence.

I here respond to each of his 16 points:

1. What I actually wrote was: "Over the next few years these treatment statistics, collected for all children, showed that when similar fluoridated and nonfluoridated areas were compared, child dental health continued to be slightly better in the nonfluoridated areas [5,6]. " The two references, from peer-reviewed journals, presented dental health data for the entire child population of (a) Greater Auckland, containing a quarter of New Zealand's population, and (b) the main population centers of New Zealand. Unfortunately, the last "nonfluoridated" word was misprinted as "fluoridated", quite altering the sentence's meaning. I have requested that a correction be published. If Pollick had read the two references he would have realized the sentence contained a misprint.

2. Pollick alleges I was "misleading" when I stated that the pro-fluoridation report of US Public Health Service authors Brunelle and Carlos was "apparently intended to counter" the Yiamouyiannis finding of no benefit from fluoridation. Yiamouyiannis' criticism of the Brunelle and Carlos paper was published as an addendum, when their attempt to refute the "no-benefit" finding had appeared. Obviously the fact that Yiamouyiannis had obtained the data showing no benefit from the US Public Health Service, using the Freedom of Information Act, would be known to Brunelle and Carlos when they prepared their paper. So my statement was not misleading.

3. Pollick alleges I was biased because I did not record the titles of profluoridation authors. When I listed the well-known fluoridationists whom I visited, early in my paper, no slight was intended. The content made clear that they were leading profluoridation experts.

4. Teotia's studies in India included low as well as high water fluoride areas.

5. Pollick agrees that nutrition is related to dental disease in developing countries, and offers no reason why the same should not apply in developed countries where, in poverty stricken areas, all diseases, including dental disease, are more prevalent than in affluent areas.

6. Pollick simply asserts his belief that fluoridation exerts both a systemic and topical dental benefit, but is unable to produce any study which counters my statement: "It is just not possible to find a blind fluoridation study in which the fluoridated and nonfluoridated populations were similar and chosen randomly."

7. Again, Pollick simply asserts his opinion (claiming "general consensus") that the fluoride in fluoridated water somehow does not cause dental fluorosis while the fluoride in swallowed

toothpaste and tablets does. In my paper I cited several studies which have reported higher fluorosis prevalences in fluoridated areas.

8. On the subject of hip fractures, Pollick quotes opinions expressed in "reviews" of pro-fluoridation authorities, but does not answer the points I made in my paper. His defense of high doses of fluoride for attempts to treat osteoporosis (citing the controversial Pak et *al* paper) is not an opinion shared by many other clinicians.

9. Pollick criticizes my failure to include, among my 73 references, a 1986 paper which reported no association between water fluoride and hip fractures. The paper was published when it was still being claimed that fluoride reduced hip fractures. Since then, the much more comprehensive studies reporting the association between hip fractures and fluoridation have been published.

10. The opinion, "the burden of evidence suggesting that fluoridation might be a risk factor for hip fracture is weak and not sufficient to retard the progress of the water fluoridation program", is also not shared by many other scientists.

11. It is difficult to see how an opinion on "possible role for slow-release fluoride combined with high-dose calcium supplementation" is related to the issue of mandatory fluoridation.

12. I agree that the National Toxicology Program study should be cited rather than the Maurer *et al* study, but that is a minor point. Pollick's quoting of the opinion that "there is insufficient basis to draw conclusions about whether osteosarcoma incidence and fluoridation are causally linked" does not alter the facts I presented: *viz.* Animal experiments, showing the rare bone cancer, osteosarcoma, occurred in male rats after fluoride ingestion were followed by reports of increased osteosarcoma in young human males in fluoridated areas but not in nonfluoridated areas.

Pollick asserts "Another reference that Colquhoun did not include, perhaps because it didn't support his thesis, is …" I not only cited the Gelberg *et al* study (reference 61) but also discussed its glaring faults (see p 114, p 40 in original).

13. The study I cited (Kanwar *et al* 1983) reported reduced testosterone levels at very low as well as high levels of fluoride.

14. Pollick alleges that I "grossly" misstated the facts of Chinese research on intelligence. A reading of the research will confirm the accuracy of my statements.

15. Pollick argues that, because only high intakes of fluoride caused intelligence deficits in rats in the Mullenix experiment, therefore the finding is unrelated to the issue of water fluoridation. Like most fluoridationists, he ignores the possibility that (as has been acknowledged with lead and other toxins) low intakes could have similar deleterious long term effects to the short term ones resulting from high intakes.

16. The same observation applies. However, the statement that I ignored "the dose or concentration of fluoride" comes strangely from one who advocates a measure which supplies an uncontrolled dose (depending on amount of water consumed) to entire populations.