DISCUSSION 235

CHANGING ONE’S MIND: AN EXAMINATION OF EVIDENCE FROM BOTH SIDES OF THE FLUORIDATION DEBATE

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Publication of John Colquhoun’s article on why he changed his mind about fluoridation along with a critique by Howard Pollick and Colquhoun’s reply provides an opportunity to re-examine the issues raised by this debate on fluoridation. The problem is to determine what evidence is sound and what is not. This analysis concerns the sixteen points raised by Pollick.

1. Printing error. In the interchange of views it was clarified that this first point was a non-issue and arose from a printing error.

2. Conspiracy to withhold information. There does not appear to be any matter of scientific evidence in dispute in this point.

3. Bias in the use of titles. Again no matter of scientific evidence appears to be under dispute on this point, but the published record speaks for itself.

4. Water fluoride levels. This point is the first involving the examination of scientific data. The point raised by Pollick was that “the work of Teotia in India” concerned areas with a very high fluoride beyond the recommended concentrations for water fluoridation. Colquhoun countered that the work had included both low and high fluoride areas.

Reference to the original article indicates clearly that the studies by Teotia and Teotia involving 400,300 children were done in areas including both endemic fluorosis with fluoride levels greater than 1 ppm and non-endemic fluorosis areas with fluoride levels less than or equal to 1 ppm. The total population of children surveyed was 800,750 of whom 400,300 volunteered for complete examination and inclusion in the study. In the nonendemic area the mean water fluoride level was $0.5 \pm 0.24$ ppm and contained 200,000 children while the endemic area had 200,300 children and a fluoride level of $4.19 \pm 2.03$ ppm. A subgroup of 23,270 children were studied involving 12,150 who lived in a low-fluoride area with a mean water fluoride level of $0.70 \pm 0.25$ ppm and 11,120 who lived in a high fluoride area with a mean water fluoride of $2.85 \pm 0.75$ ppm.

Since 1962, the “optimal” concentration of fluoride in drinking water for the United States has been set at 0.7-1.2 ppm depending on the mean temperature of the locality. Assessing the evidence suggests that it is incorrect to dismiss the work of Teotia and Teotia on the grounds that only areas with very high fluoride levels were involved.

5. Effects of nutrition on tooth decay. There does not appear to be any scientific data in dispute in this point with both Pollick and Colquhoun agreeing that nutrition may have a role in tooth decay. Pollick points to the place of sugar while Colquhoun says he does “not know the answer for sure” and wonders about fresh fruit, vegetables and cheese. However, the observation that influenced Colquhoun towards changing his mind, involving the decline in tooth decay commencing before the availability of fluoride and continuing even when fluoridation was fully implemented, is the central part of Colquhoun’s argument at this stage. This point is not addressed by Pollick.
6. **Benefits of fluoridation.** Pollick states that Colquhoun asserts that in all of the studies published there is bias in population selection and examiner diagnosis and that “most of the examiners were keen fluoridationists”. Colquhoun has replied that a study has not been produced which counters the statement that “It is just not possible to find a blind fluoridation study in which the fluoridated and nonfluoridated populations were similar and chosen randomly”. In his paper Colquhoun indicates that he considered most, rather than all, pro-fluoridation studies were not blind and that the examiners knew which children received fluoride.

The Hardwick et al study,6 mentioned by Pollick, does not contradict Colquhoun’s position that although the examiners were blind as to where the children came from, no evidence is given to show that, of the two areas which were compared, the choice as to which one would be fluoridated was made randomly. Whether this study is the best one to illustrate a beneficial effect for fluoridation is open to debate. It is not mentioned in the review by Newbrun7 in 1989 on the effectiveness of water fluoridation but is referred to in reviews by Beltran and Burt,8 1988, and Ripa,9 1993, as evidence of the benefit fluoride may give to teeth already erupted. Hardwick et al8 indicate that the primary aim was to test the null hypothesis that there would be no post-eruptive effect of artificial water fluoridation on the caries increments of 12-year old children over the following three years. A difficulty in the study was that of the 305 children in the fluoride group at the baseline examination only 47% or 144 were able to attend the fourth annual re-inspection. Another difficulty for the study, in demonstrating a beneficial effect for fluoride in reducing dental decay, was that although the control and fluoride groups of children showed “close agreement” in “mean initial DMFS and DMFT rates”, at the end of the study the figures given, in Table 1, show that the fluoride group did not have lower rates for either the DMFS or the DMFT (Mean ± SD; Control group DMFS 6.60 ± 4.80; Fluoride group DMFS 7.33 ± 6.47; Control group DMFT 4.27 ± 2.82; Fluoride group DMFT 4.58 ± 3.49). It is not clear that the Hardwick et al6 study shows that fluoridation of water supplies exerts a benefit systemically as well as topically. The authors saw “substantial topical effects on teeth already erupted at the start of fluoridation”, during the study period “fluoride dentifrices were used extensively”, and at the end of the four years the DMFS and DMFT rates were no better in the fluoride group than in the control group.

As has been shown elsewhere10 there is negligible benefit to be gained from fluoride ingestion and also evidence that fluoride acts topically. The mean level of fluoride in the saliva, after ingesting fluoridated water at a concentration of 1.2 ppm, is only 0.87 ± 0.047 μmol/L or 0.017 ± 0.001 ppm.11 Burt12 has noted that the evidence for a primarily topical cariostatic effect of fluoride has grown. He notes that “fluoride most effectively controls caries when a low concentration can be maintained consistently in the oral environment”.

Thus there is, in fact, wider acceptance of Colquhoun’s position that “there is negligible benefit from swallowing fluoride”. The study by Hardwick et al6 referred to by Pollick does not provide evidence to contradict this view.

7. **Dental fluorosis.** On this point Pollick gives the view that there is a “general consensus” that the increase in dental fluorosis is due to fluoride toothpaste and
tablets and that it is "generally accepted" that water fluoridation is not harmful in this way. No references are given whereas there are 22 references in the review article cited by Colquhoun. 13 Colquhoun includes a case history of a patient developing dental fluorosis with access to fluoridated water who did not use fluoridated toothpaste or tablets. In the absence of data, Pollick has not established a case to refute the evidence linking, in 11 communities, higher dental fluorosis rates in the range of 16-51% with water fluoride levels of 0.4-1.4 ppm, in contrast to, in 9 communities, dental fluorosis rates of 2-15% with water fluoride levels of 0.0-0.4 ppm. 13

8. Effects of fluoride on bone. Pollick indicates that studies, such as those reviewed by the National Research Council, 14 have found an association between bone fractures and fluoride. Colquhoun, in his paper, 1 notes that he expressed concern that this might arise back in 1984 at a time when his colleagues dismissed his need to worry. He related how his views on fluoridation were affected by the report 15 of hip fractures being associated with higher fluoride levels six years later. There appears to be a basic agreement that the effect of water fluoridation on hip fracture rates needs to be considered.

Whether one can accept the conclusion of the National Research Council "that there is no basis at this time to recommend EPA (the Environmental Protection Agency) lower the current standard for fluoride in drinking water" 14 (a maximum contaminant level of 4 ppm) would require a more detailed analysis of the evidence than is possible to provide here. Pollick suggests five of the ten studies looked at by the National Research Council 14 showed a positive association between fluoride and hip fracture. In reviewing the report Lee 16 found that seven of the ten showed a positive correlation and that there were reasons why the other three studies were not of value, such as lack of exposure to fluoride prior to the menopause and an insufficient number of subjects.

Pollick appears to be in agreement with Colquhoun that when fluoride is used in high doses to treat osteoporosis it has led to an increase in hip fractures. Thus on this point there appears to be a basic agreement between Pollick and Colquhoun that in some circumstances fluoride can contribute to hip fractures.

Pollick observes that the Subcommittee on Health Effects of Ingested Fluoride 17 "concluded that the weight of evidence indicates that bone strength is not adversely affected in animals that are fed a nutritionally adequate diet unless there is long-term ingestion of fluoride at concentrations of at least 50 mg/L in the drinking water or 50 mg/kg in the diet". However the exact quotation 17 qualifies the conclusion by referring to the "evidence currently available" and notes that recent reports from epidemiological studies of human populations have provided conflicting evidence on this subject and indicate the need for additional research.

Some reservations were held about the appropriateness of the methods used to cause bone fractures. Of the 15 animal studies reviewed, reduced bone strength was found in some of the experimental conditions in seven studies. 17 Higher bone fluoride levels occurred when the diet was low in calcium. The bone-ash fluoride levels reported, associated with reduced bone strength, were 640 ppm, 7,398 ppm, 12,600 ppm, 7,393 ppm, 5000 ppm, and 11,000 ppm. The presence of adequate amounts of calcium in the diet may reduce the rate of absorption of fluoride, but if the calcium intake is inadequate or the intake of fluoride is continued for a longer
period, the potential for fluoride to accumulate in bone and be associated with reduced bone strength has not been excluded by the animal studies. Arnala et al.\textsuperscript{18} found mean ashed trabecular bone fluoride level of $3.72 \pm 2.39$ mg/g ($3720 \pm 2390$ ppm) in 25 patients with hip fracture from a high fluoride area with a fluoride level in the water greater than 1.5 ppm.

Pollick notes that Colquhoun omitted mention in his 1997 paper\textsuperscript{1} of the successful results described in 1994 by Pak et al.\textsuperscript{19} with low-dose, slow-release sodium fluoride in the treatment of osteoporosis. This first report of the study gave the results of the use of intermittent slow-release sodium fluoride plus continuous calcium citrate administered for about 2.5 years. Colquhoun notes that the opinion that this is helpful is not shared by many other clinicians. Lee\textsuperscript{20} expresses such a viewpoint: that the fluoride treatment recommended by Pak et al. should be regarded as controlled osteofluorosis and that the 3.5 year trial described in the final report\textsuperscript{21} was insufficient to evaluate the potential increased risk of hip fracture.

A recent review\textsuperscript{22} noted that while the study by Pak et al.\textsuperscript{21} found a significant reduction in the rate of vertebral fracture, in three other studies\textsuperscript{23-25} the effect was small. In a randomized, placebo-controlled, two-year trial of sodium fluoride (50 mg a day) and monofluorophosphate (two doses) in 354 women with osteoporosis, fluoride therapy, as compared to placebo, had a large effect on bone mineral density in the lumbar spine (increase, 10.8% vs. 2.4%), but no effect on the rate of vertebral fractures\textsuperscript{26} Thus, even at relatively low doses, fluoride had little beneficial effect on fracture rates. It was noted that sodium fluoride causes stress fractures.\textsuperscript{23,26} In discussing the therapeutic choices for women most at risk for fractures no mention is made of a role for fluoride\textsuperscript{22} The hope offered by fluoride in 1994 for the treatment of osteoporosis appears to have passed.

9. Omission of paper with negative evidence on hip fractures. Pollick argues that while Colquhoun referred to two papers by the Finnish researchers Alhava et al.\textsuperscript{27} and Arnala et al.\textsuperscript{28} which noted fluoride accumulation in bones with fluoridation he omitted reference to a later paper by Arnala et al.\textsuperscript{18} in which no difference was found in the incidence of hip fractures between low-fluoride, fluoridated and high-fluoride areas.

No question involving the interpretation of scientific data appears to be at issue in this point. Colquhoun was recounting the factors which led him to change his mind about fluoridation, and it is understandable that studies not showing a relationship between fluoride and adverse effects would be less likely to have an influence.

He notes that, at the time, the later paper by Arnala et al.\textsuperscript{18} had to be seen alongside a Finnish paper by Simonen and Laitinen\textsuperscript{29} suggesting fluoridation was associated with fewer hip fractures. His point that later studies on hip fractures with larger numbers of subjects were more relevant is reasonable. The 1986 paper by Arnala et al.\textsuperscript{18} had 461 subjects with hip fracture. This is fewer than the 651 patients with hip fracture in the 1993 study by Jacobsen et al.\textsuperscript{30} which Lee\textsuperscript{16} found to have an insufficient sample size to be able to detect important medical effects. In contrast the studies finding significant effects involved 541985 patients with hip fracture (Jacobsen et al. 1990\textsuperscript{15}) and 20393 patients with hip fracture (Cooper et al. 1991\textsuperscript{31}). Colquhoun has previously referred\textsuperscript{32} to the 1986 paper by
Arnala et al.\textsuperscript{18} Later studies with a much greater data base made it less important to cite it in his new paper under discussion. Hence there is no significance to the fact that it was omitted.

10. Fluoride and hip fractures. There does not appear to be any new point being made different from those raised in point 8.

11. Effects of fluoride on bone. Again no new issues are raised.

12. Association of fluoride and osteosarcoma. Pollick and Colquhoun agree that the appropriate reference for the study in which osteosarcomas were found in rats given fluoride was one referring to the National Toxicology Program study,\textsuperscript{33} rather than to the Procter and Gamble study\textsuperscript{34} in which the osteosarcomas found were not at a statistically significant level.

There does not appear to be any dispute about the term “equivocal evidence” of carcinogenicity. The Peer Review Panel\textsuperscript{35} indicated that equivocal evidence is a category for uncertain findings demonstrated by studies that are interpreted as showing a marginal increase of neoplasms that may be chemically related.

Although Colquhoun indicates that he was influenced by the finding of 3-7 fold increases in osteosarcoma rates in young males in fluoridated areas of New Jersey compared to non-fluoridated areas,\textsuperscript{36} there does not appear to be any basic difference in the interpretation of the findings. Pollick notes that Cohn\textsuperscript{37} stated there was insufficient basis to draw conclusions about whether osteosarcoma incidence and fluoridation were causally linked and, in the reference cited by Colquhoun, Cohn\textsuperscript{36} notes that “Because of the limitations of the study design and the small numbers of cases that occurred, this analysis does not imply a causal connection between fluoridation and osteosarcoma”. Colquhoun includes a question mark after the sub-title of Bone Cancer for this section of his paper indicating questions about the nature of the link still exist.

Questions about the sample studied by Gelberg et al.\textsuperscript{38} are raised by Lee\textsuperscript{39} because 67% of the patients were female whereas the majority of patients with osteosarcoma are usually male. Colquhoun notes that there is disagreement about whether it is safe to continue with water fluoridation while the nature of the associations between fluoridation and osteosarcoma are considered.

13. Effect of fluoride on testosterone synthesis. Pollick states that 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. In the reference given of Kanwar et al.\textsuperscript{40} it is noted that from 1 ppm to 200 ppm of fluoride, the degree of inhibition in testosterone synthesis seems to be dependent on fluoride concentration. In an \textit{in vitro} assay there was a noticeable, though marginal, inhibition even at 10 ppm, a significant marked fall at 100 ppm and maximal inhibition at 200 ppm. The highest reported levels of fluoride in soft tissues\textsuperscript{41} have been given as: brain 6.1 ppm, heart 8.1 ppm, pancreas 8.2 ppm, lung 17 ppm, thyroid 23.5 ppm, liver 61 ppm, lens 77.3 ppm, prostate 86 ppm, fat 145 ppm, hair 171 ppm, kidney 181 ppm, bladder 185 ppm, nails 186 ppm, skin 290 ppm and aorta 8400 ppm. The evidence suggests that inhibition of testosterone synthesis begins at 1 ppm of fluoride and a noticeable effect occurs with a level of 10 ppm.\textsuperscript{40} Although a reference to fluoride levels in the testes has not been found, the levels which may occur in other soft tissues
suggest that levels affecting testosterone production might occur. It thus appears to be fair comment that very low as well as high levels of fluoride might affect testosterone levels.

14. Effect of fluoride on intelligence. On this point Pollick states that Colquhoun misstates the facts of the research from China\textsuperscript{42,43} linking dental fluorosis in children with lower intelligence and that to suggest that children with dental fluorosis have on average lower intelligence scores is a gross misstatement of the facts.

Colquhoun in his paper said "Even more chilling is the evidence from China that children with dental fluorosis have on average lower intelligence scores". In the first reference given by Colquhoun, the paper by Li, Zhi and Gao\textsuperscript{42} a significantly lower Intelligence Quotient was found in children from areas with medium or severe degrees of dental fluorosis compared to children from areas without dental fluorosis or only slight dental fluorosis. In the other reference by Zhao, Liang, Zhang and Wu\textsuperscript{43} children in an area with a high rate of dental fluorosis, 86\%, had significantly lower intelligence than those in an area with a low rate of dental fluorosis, 9\%. The source of the fluoride in the study by Li et al,\textsuperscript{42} in the medium and severe fluorosis areas, was the coal used for cooking, heating and drying grain. In the study by Zhao et al\textsuperscript{43} the area with a high dental fluorosis rate had a water supply with a high fluoride level, 4.12 ppm.

The basic concern of intelligence being affected in some children with dental fluorosis accurately reflects the concerns raised in the studies and it is difficult to see it involving misstatement, or gross misstatement, of the facts of the research. Finding it chilling that fluoride might affect brain development is understandable given that fluoridation was initially promoted as being completely safe. The query about the relevance of the effect of excess fluoride on brain development, because in one of the studies it came from coal smoke rather than water, does not appear to be relevant. The concern is that if fluoride, from whatever source, affects other organs detrimentally as well as causing dental fluorosis, then water fluoridation, which is associated with significant dental fluorosis for many, may be unsafe.

15. Relevance of animal studies of the effect of fluoride on the brain. The point is made by Pollick that the study by Mullenix et al\textsuperscript{44} is irrelevant to water fluoridation because the pregnant rats studied were injected with fluoride, which would not occur with humans, and the weanling and adult rats then drank water with different concentrations of fluoride including, for adult rats, a period of six weeks with water at a concentration at least 100 times that recommended for water fluoridation.

Colquhoun indicates his concern that, because of accumulation within the body, lower doses taken for longer periods of time might have a similar effect to higher doses taken for a short period. He saw a model having been established with other toxins such as lead. In their paper Mullenix et al\textsuperscript{44} note that it was the fluoride levels in plasma, not fluoride levels of exposure, which best predicted effects on behaviour. Similar plasma fluoride levels of 0.076-0.25 ppm have been found in humans ingesting 5-10 ppm fluoride in drinking water and plasma levels as high as 0.28 to 0.43 ppm have been measured in children drinking water containing 16 ppm fluoride. Fasting serum fluoride levels of 0.2 to 0.3 ppm are used in the treatment of osteoporosis, and plasma fluoride levels as high as
1.44 ppm are found in children 1 hour after receiving topical applications of an acidulated phosphate fluoride (1.23%) gel.

Mullenix et al\textsuperscript{44} considered that because humans occasionally are exposed to high amounts of fluoride and plasma levels as high as those found in this rat study, neurotoxic risks deserve further evaluation. The closeness of the lower limit of 5 ppm, in the range of 5-10 ppm fluoride in drinking water which produced similar plasma levels to those found in the rats, to the maximum contaminant level for fluoride acceptable for drinking water of 4 ppm\textsuperscript{10} is a cause for concern. The evidence thus suggests that the adverse effects on brain function, possibly on that of the hippocampus in particular, are related to the plasma levels found rather than the method of administration.

Thus although the study by Mullenix et al\textsuperscript{44} may not have been intended, as noted by Pollick, to determine the effects of water fluoridation, its results could be seen as being particularly relevant to assessing whether the fluoridation of water was safe. The findings of Mullenix et al\textsuperscript{44} are consistent with clinical reports of chronic exposure to fluoride being associated with cerebral impairment, affecting particularly concentration and memory.\textsuperscript{45}

This position has been underlined by subsequent events and the publication of two papers\textsuperscript{46,47} by Isaacson, Varner, Jensen and Horvath on the effect, in rats, of the chronic administration of aluminium fluoride and sodium fluoride on neuronal and cerebrovascular integrity. In these studies, the chronic administration of aluminium fluoride or sodium fluoride in drinking water in rats resulted in distinct morphological alterations in the brain including effects on neurones and cerebrovasculature. The concentrations of aluminium fluoride, 0.5 ppm (as 0.5 ppm Al), and sodium fluoride, 2.1 ppm (as 2.1 ppm NaF), used gave fluoride ion concentrations of about 1 ppm, the level often used in water fluoridation.

Thus, with the benefit of hindsight, it is clear that Colquhoun was correct to be alarmed about the studies implicating fluoride as adversely affecting brain development, even though the source of the fluoride was not always in the drinking water but included injections\textsuperscript{44} (for pregnant rats) and coal smoke (in the study by Li et al\textsuperscript{42}).

16. Omission of details of dose and concentration in discussion on toxicology. Pollick argues that Colquhoun consistently alleges harm from fluoride without stating the dose or concentration of fluoride. Colquhoun indicates that low intakes for long periods might be as deleterious as high intakes for short periods. In his paper\textsuperscript{1}, when referring to harm from fluoride, he gives references which provide details of the fluoride exposure involved. For example, in the study by Zhao et al\textsuperscript{43}, on intelligence in children, the water fluoride concentrations in the two groups is given as 4.12 ppm and 0.91 ppm.

Colquhoun appears to be explaining what influenced him to change his mind from being an ardent advocate of water fluoridation, who fiercely poured scorn on a lay person who had heard and accepted the case against fluoridation\textsuperscript{1}, to one who was opposed to it. Given that there are space restrictions, which limit how much detail can be included from any paper referred to, it is appropriate to omit details about dose and concentration of fluoride where such information is readily available in the publications that are cited. In some instances, reference is made\textsuperscript{1} to "fluoridated Auckland" and "fluoridated areas of America" and the
meaning is conveyed that the level of fluoride in the water would be in the region of 1 ppm.

The reader, after considering the two sides of the debate, will be in a position to decide what their own view is. Colquhoun required some courage to change his mind. Somerset Maugham would not have had him in mind when he wrote, in Of Human Bondage, “Like all weak men, he laid an exaggerated stress on not changing one’s mind”.

REFERENCES
13 Colquhoun J. Disfiguring, or “white and strong”? Fluoride 23 104-111 1990.
17 Reference 14, pp 61-71.


Letter to the Editor

NATURAL AND ARTIFICIAL

I would very much like to see some explanation of the difference between natural calcium fluoride and the artificial fluorides used in water fluoridation.

Eileen Adelman

Managing Editor replies: There is a belief in some natural health circles that naturally occurring calcium fluoride is less harmful than other fluorides used for artificial water fluoridation. My understanding is that, in general, the fluoride ion is equally toxic, whatever its origin. In India and China naturally occurring fluorides cause much ill health, including crippling fluorosis. However, there are some differences:

1. The industrial waste products used for artificial water fluoridation are much more soluble than calcium fluoride, so are much more dangerous if there is an accidental overdosing of the water supply. Such accidents have occurred, causing sickness and sometimes deaths. However, at the relatively low concentrations occurring in artificially fluoridated water, calcium fluoride and the other fluorides are both soluble.

2. Sometimes naturally fluoridated water also contains minerals like calcium and magnesium, which are known to mitigate the toxic effects of the fluoride. Conversely, the industrial waste products used for water fluoridation almost always contain trace amounts of other toxins such as cadmium, lead and arsenic.

3. Recent research (see Editorial in this issue) indicates that silicofluorides, commonly used for fluoridation, are much more dangerous than previously realized, in that they increase the lead content of the water.

John Colquhoun
SCHATZ: PARADOXICAL EFFECTS

I do not understand Neil Jenkins' two letters to the editor in *Fluoride* 30 (4) 1997. With respect to paradoxical effects, Neil has conveniently forgotten his own 1963 publication. I discussed it in an article in 1964. Neil not only reported anomalous results in his own research with fluoride but also referred to anomalous results which others got with fluoride. He called them "some unexplained anomalies."

Neil himself therefore conveniently disregarded his own data and erroneously concluded that "The solubility of enamel has been found to be related to its fluoride content."

I sent Neil a copy of everything I have ever published on fluoride and on paradoxical effects, including the 1964 article.

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Jenkins Replies

Al Schatz suggests that in my review of the relationship between the fluoride concentration of enamel and its solubility I have overlooked the existence of paradoxical effects. The results I quoted from Isaacs et al mostly consisted in showing that the higher the fluoride in enamel the lower is its solubility. The results on only two samples out of a total of 24, each pooled from 40 teeth, were unexpected, Which I still think can be regarded as "unexplained anomalies" which may occur in the collection of any experimental data.

The only results of Isaac et al that might be regarded as paradoxical refer to the four results comparing enamel from persons over 50 and under 20 years of age. The fluoride of the enamel is lower in the younger group (as expected) but in three out of the four comparisons the solubility is also lower. However, as Isaac et al point out, unknown changes may take place during aging which affect the solubility of enamel. Also, it is possible that dietary changes (for example, in trace elements) between the 1900s and the 1940s (when these teeth were forming) may influence the composition and solubility of enamel and over-ride the effect of fluoride.

I conclude that these results do not demonstrate a paradoxical effect of fluoride but show that many substances other than fluoride may affect enamel solubility. Provided that like is compared with like, the solubility of enamel has been found to be related to its fluoride concentration although this is only one means by which fluoride protects against caries.

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1 Jenkins GN. *Journal of Dental Research* 42 444 1963
I refer to the letter of Gillian Durham, New Zealand Director-General of Health, which criticized the article “New evidence on fluoridation” 2. To justify her allegation that the article lacks “veracity” and is selective, Durham refers to the 1994 Public Health Commission report Water fluoridation in New Zealand 3. That report itself states that it “was essentially written by one person with experience in particular areas only” and “places some reliance on the quality of previous reviews that have been conducted. It is important to acknowledge that there are limitations with some of the reviews. Some may have tended to place unwarranted weight on the findings of previous expert reviews and lack wide representation of all the areas requiring expertise.” This weakness is revealed by the report’s one-side reliance on small-scale pro-fluoridation studies to claim that fluoridation is cost-effective, rather than the various comprehensive studies which indicate little if any benefit. Yet Dr Durham implies that the PHC report is a watershed document and that only later papers have relevance.

However, the PHC report conceded that:

- “It is possible that there is a small increased risk of hip fracture associated with fluoridation”
- “a small increased risk of osteosarcoma in young men cannot be ruled out.”
- “Fluoride intake from food is likely to be relevant to skeletal fluorosis in some areas (especially when vegetables are grown in fluorotic soil)”
- “individuals with longstanding and severe renal disease are theoretically at risk in temperate countries at usual water fluoridation levels”
- “Skeletal fluorosis might be occurring in individuals with either a long term intake or a metabolic susceptibility.”

It was also admitted that skeletal fluorosis can occur at fluoride levels as low as 0.7-2.5 ppm, though it was qualified by the fact that the countries involved are nearly all tropical and therefore tend to have high water intakes. Many people in Australia and New Zealand also have high liquid intakes.

One has to question Durham’s judgement that the benefits of possibly preventing some dental caries outweigh the above admitted risks.

There has been recent publicity in New Zealand that dental caries is increasing with increasing poverty, in both fluoridated and nonfluoridated places. This fact disproves the assertion that fluoride is particularly advantageous to the lower socio-economic groups.

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