

Scientific Evidence Continues to Support Fluoridation of Public Water Supplies

To the Editor.—I thank Paul Connett for the opportunity to provide further information about the benefits of water fluoridation.¹ I am not surprised by his unwillingness to accept previous reviews of the literature on fluoridation; this is a consistent aspect of his previous presentations on the subject.² Connett is a critic of fluoridation, having previously been critical of the York review of water fluoridation.³ McDonagh and Kleijnene, as well as Phipps, Orwoll, Mason, and Cauley, have responded to Connett's criticism.⁴ The title and purpose of my paper was to focus on the available evidence of the relationship between water fluoridation and the environment.⁵ Connett provides no specifics that refute any of the evidence I presented that there is no harm from water fluoridation.

However, Connett chooses to point out that I presented little evidence of the benefits of water fluoridation. The effectiveness of fluoridation has been the subject of extensive review. The York review⁶ found that the best available evidence suggests that fluoridation of drinking water supplies reduces caries prevalence, as measured by the proportion of children who are caries-free (a median of 14.6% more in fluoridated areas) and by the mean change in decayed, missing-due-to-extraction and filled primary and permanent teeth (median 2.25 fewer decayed teeth in fluoridated areas). The 2001 CDC review found that combined use of fluoride toothpaste and fluoridated water offers protection above either used alone.⁷

The WHO data (DMFT for 12-year-olds) referred to by Connett, as

graphically displayed by Chris Neurath, FAN (Fluoride Action Network),⁸ show that caries in selected countries has declined. This has not happened by accident, but by the availability of controlled fluoride concentrations in water, toothpaste and professionally applied fluorides in school-based and other public programs and dental offices. It should be remembered that the original studies of naturally occurring fluorides in drinking water and dental caries were conducted with 12–14-year-old children. These extra couple of years are particularly important in that permanent second molars do not erupt until age 11–13 years. This age group has been mainly studied because they are a generally compliant captive school-based population, even though the benefits of water fluoridation in permanent teeth are greater in older age groups.

The countries selected by Neurath do not all have descriptions in the accompanying chart. Data for Japan are plotted, but the description of Japan as a fluoridated or non-fluoridated country is omitted, even though the data for Japan show the highest caries experience (decayed, missing and filled teeth—DMFT—for 12-year-olds). There is no fluoridation in Japan (since 1972), and low availability of fluorides.⁹ In Australia, about 11.5 million people (nearly 2 in 3) have access to fluoridated water supplies in every capital city except Brisbane.¹⁰ However, Australia is not listed in the accompanying chart, although the DMFT data are graphed. Similarly, 50% of New Zealanders have access to fluoridated water but New Zealand is not included in the chart.¹¹

The countries listed by Neurath are those where the mean DMFT is less than 2; yet there are many more countries with low DMFT data. In fact there are 207 countries/areas listed where data were collected using somewhat different methods.¹² The reports for some countries are of different population groups from one time to another and are often not directly comparable. Reasons for the declines in different countries/ areas have been explained in a variety of ways.

For example in Iceland, in spite of an increase in sugar consumption in the form of sweets and soft drinks, the decline has been attributed to an increase in the use of fluoride toothpaste, fluoride rinse and varnish programs, and sealants, as well as changes in treatment philosophy and increased personnel resources.¹³ In sharp contrast, there are countries where caries experiences among 12-year-olds have apparently increased; Romanian children are among those with the most tooth decay (mean DMFT 7.3 in 1998).¹⁴

The 1991 review by the USPHS on fluorides clearly states the benefit of water fluoridation¹⁵:

Numerous studies, taken together, clearly establish a causal relationship between water fluoridation and the prevention of dental caries. While dental decay is reduced by fluoridated toothpaste and mouth rinses, professional fluoride treatments and fluoride dietary supplements, fluoridation of water is the most cost-effective method. It provides the greatest benefit to those who can least afford preventive and restorative dentistry and reduces dental disease, loss of teeth, time away from work or school, and anesthesia-related risks associated with dental treatment.

In the 1940's, children in communities with fluoridated drinking water had reductions in caries scores of

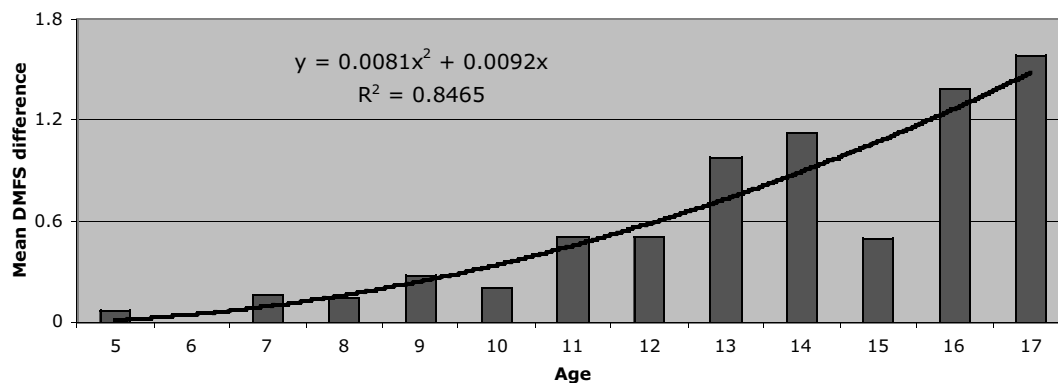


Figure 1—Data from Table 6 of Brunelle and Carlos.¹⁶ Difference in Mean Decayed, Missing Due to Caries and Filled Permanent Tooth Surfaces (DMFS) between Children with Continuous Exposure to Water Fluoridation and Those with No Such Exposure.

about 60 percent as compared to those living in non-fluoridated communities. Recent studies still reveal that caries scores are lower in naturally or adjusted fluoridated areas; however, the differences in caries scores between fluoridated and non-fluoridated areas are not as great as those observed in the 1940's. This apparent change is likely explained by the presence, in non-fluoridated areas, of fluoride in beverages, food, dental products, and dietary supplements.

Connett's view of the Brunelle and Carlos paper¹⁶ fails to take into account several factors. Firstly, no tests of statistical significance were applied to the data in that publication, so it wasn't a matter of the differences not being statistically significant. With sample sizes of this magnitude, small differences are usually statistically significant. Excluding children with a reported history of exposure to supplemental or topical fluorides, there was a 25% lower DMFS among a sample size of 3,574 children aged 5–17 who were continuously exposed to water fluoridation, compared with 2,380 children with no fluoridation exposure. This difference is in the permanent teeth only; greater differences have been found in the deciduous dentition (which was not the focus of the Brunelle and Carlos paper).

Second, the difference of 0.6 DMFS in favor of fluoridation was

for all children 5–17 years old combined. Very few 5-year-olds have any permanent teeth and the permanent teeth erupt into the mouth until about age 13 years (excluding the wisdom teeth). It also usually takes a few years for caries to develop to the point of being clinically evident. Thus, there are not 128 tooth surfaces at risk for all ages, as Connett claims. According to Burt and Eklund, "When caries first occurs today in the permanent dentition of young people, it does so predominantly in the first and second molars; it is now uncommon in all anterior teeth."¹⁷ This fact is primarily due to the availability of fluoride through water fluoridation and fluoride toothpaste in protecting the upper anterior teeth, and saliva flow that bathes the lower anteriors. While other teeth may develop caries, it can be postulated that there are 0–20 surfaces at risk before age 12 (first permanent molars) and 20–40 surfaces at risk from ages 13–17 years (first and second molars). Certainly, of those surfaces, the most at risk are the sealable pit and fissured occlusal surfaces of permanent first and second molars.

A further analysis of the difference in mean DMFS by age from the Brunelle and Carlos paper shows an increasing benefit of fluoridation with age, such that by age 17 there is a mean 1.5 surface benefit (Figure 1). It should be remem-

bered that saving one surface from decay means at least one filling and perhaps a root canal treatment or extraction or a variety of treatments in later years. It has been estimated that the lifetime cost per person of water fluoridation is less than the cost of a single filling.

The generally low caries experience in Australia is a function of the availability of fluorides. The small difference in caries experiences of children between fluoridated and non-fluoridated areas is related to the halo effect of those in non-fluoridated areas receiving fluoridated beverages and foods from neighboring fluoridated areas, especially with two-thirds of Australians having access to fluoridated water.¹⁸

Connett disregards the benefit of water fluoridation on the deciduous teeth in Armfield et al.¹⁹ Armfield states: "Deciduous caries experience was highest for children with the greatest lifetime consumption of tank and bottled water. For children who had lifetime availability of fluoridated tap water, the effect of consumption of nonpublic water was significant even after controlling for family income, residential location, age, sex and exposure to discretionary fluorides such as fluoridated toothpaste and fluoride tablets."

With regard to the Canadian report by Locker,²⁰ the full statement on reductions in dental caries in that executive summary states:

Although current studies of the effectiveness of water fluoridation have design weaknesses and methodological flaws, the balance of evidence suggests that rates of dental decay are lower in fluoridated than non-fluoridated communities. The magnitude of the effect is not large in absolute terms, is often not statistically significant and may not be of clinical significance. The effect tends to be more pronounced in the deciduous dentition. The effect tends to be maximized among children from the lower socioeconomic groups so that this section of the population may be the prime beneficiary. Canadian studies do not provide systematic evidence that water fluoridation is effective in reducing decay in contemporary child populations. The few studies of communities where fluoridation has been withdrawn do not suggest significant increases in dental caries as a result. More research is needed to document the benefits of fluoridation to adult and elderly populations in terms of reductions in coronal and root decay. Research is also needed to document improvements in the oral health-related quality of life that accrue to populations exposed to fluoridated water in order to enhance the credibility of this public health initiative.

Thus, taken as a whole, Locker's review of studies on reductions in dental caries shows the benefit of water fluoridation is greatest for the deciduous teeth and among children from poor families. There is nothing in Locker's report that indicates the time has come to stop fluoridation; on the contrary, fluoridation is of benefit and additional studies are needed to determine the extent of benefit in older populations.

Locker further asserts that:

[page 13] . . . Water fluoridation does more to enhance the maintenance of salivary levels of fluoride which are compatible with the inhibition of demineralization and promotion of remineralization of enamel than other types of fluoride vehicles (Lewis et al, 1994). It is for this reason that many authorities contend that water

fluoridation continues to be the fluoride technology of choice with respect to effectiveness, distribution, equity, compliance and costs.

The margin-of-safety issue on fluoride is paradoxical and perplexing to those who would like to see a greater level of reassurance that fluoride will not produce dental fluorosis. The fact is that the ecological relationship between fluoride and teeth is such that there will always be some individuals who will have very mild or mild dental fluorosis at fluoride levels in water that are most suitable for tooth decay prevention. The original pre-1945 studies prior to the introduction of water fluoridation and fluoride toothpaste and other forms of topical and systemic fluoride clearly show that at about 1 ppm fluoride in drinking water tooth decay and dental fluorosis are both minimal. In fact, 1 ppm was chosen to minimize the prevalence of fluorosis, while realizing that there would be even greater decay-preventing benefit at slightly higher concentrations. There is insufficient evidence of harm from having drinking water with 1 ppm fluoride, so that extending water fluoridation to all is appropriate. Since access to fluoride is the most beneficial method available to reduce the burden of tooth decay, a disease which can have serious consequences, then it behooves us, as a society, to provide such access for the greatest number, particularly in a cost-effective way that can benefit the most vulnerable. Further, since dental fluorosis is not a disease and does not have serious consequences, and water fluoridation contributes only a minimal amount of this condition, then the benefit-risk ratio is clearly in favor of water fluoridation.

It is important to remember that there are different degrees of severity of dental fluorosis; not all fluorosis is of esthetic concern. The York review found that the prevalence of fluorosis at a water fluoride level of 1.0 ppm was estimated to be 48%

(95% CI 40–57) and for fluorosis of aesthetic concern it was predicted to be 12.5% (95% CI 7.0–21.5).

The linear correlation between the severity of dental fluorosis and the frequency of bone fractures in children that Connett refers to is from a Mexican population where “nearly 84% of the wells that supply drinking water to inhabitants of the Guadiana Valley exceed the maximum fluoride concentration limits established by both Mexican and international regulations.”²¹ It is unclear what Connett is attempting to show, particularly given that those authors found the incidence of fractures to decrease at higher fluoride concentrations. The data from that study show that there were too few bone fractures for the data to be tested for statistical significance.

The reference on fluoride and bone fracture supplied by Connett does not appear to support his cause.²² In that study, 8,266 male and female subjects ≥ 50 years of age were enrolled from six Chinese populations with water fluoride concentrations ranging from 0.25 to 7.97 ppm. Parameters evaluated included fluoride exposure, prevalence of bone fractures, demographics, medical history, physical activity, cigarette smoking, and alcohol consumption. The results confirmed that drinking water was the only major source of fluoride exposure in the study populations. The authors showed the prevalence of overall bone fracture was lowest in the population of 1.00–1.06 ppm fluoride in drinking water, which was significantly lower ($p < 0.05$) than that of the groups exposed to water fluoride levels ≥ 4.32 and ≤ 0.34 ppm. The prevalence of hip fractures was highest in the group with the highest water fluoride (4.32–7.97 ppm). The value was significantly higher than that of the population with 1.00–1.06 ppm water fluoride, which had the lowest prevalence rate.

There have been many studies of fluoride in water and hip fracture, with the consensus being that there

is no cause-and-effect relationship. Hellier et al have summarized those studies²³:

Most epidemiological evidence comes from ecological studies of hip fracture, but the results of such investigations have not been consistent. Some studies have suggested a positive association between the concentration of fluoride in water and incidence of fractures, but others have found no association or even an inverse relation. Only two studies have related the risk of hip fracture to fluoride ingestion in individuals rather than populations, and for one of these only a brief preliminary analysis has been published. The limited scope in ecological studies to adjust for the effects of potential confounding variables (such as physical activity, body build, cigarette smoking, dietary calcium intake, and reproductive variables) makes interpretation of their conflicting findings difficult.

With regard to any relationship between fluoride and IQ, the 2000 York review recommended a need for further high-quality research. As for the 2003 Chinese studies by Xiang et al. on fluoride and IQ, unfortunately I was not able to access those references from the journal *Fluoride*.

Connett presents no evidence that water fluoridation is harmful to the thyroid. However, he presents evidence of a reduction of thyroid function with access of up to 10 ppm fluoride in drinking water, in the 1985 Russian article.²⁴ Bachinskii et al. assert that: "Consumption of drinking water with elevated fluorine content (122 ± 5 $\mu\text{mol/L}$) (equivalent to 2.35 ppm) leads to stress of the functional status of the pituitary-thyroid system in healthy people." Note: for fluoride, 1 ppm = 52.6 $\mu\text{mol/L}$.

The study by Freni suggested that further research was indicated to investigate whether or not the fluoride effect on the fertility rate found at the county level also applies to individual women. Again, the fluoride levels specified in that

study, in excess of 3 ppm, exceed the recommended levels for community water fluoridation.²⁵

For previously reviewed information on the effect of water fluoridation on the kidneys, I turned to "Fluoridation Facts" from the American Dental Association²⁶:

Approximately 50% of the fluoride ingested daily is removed from the body by the kidneys. Because the kidneys are constantly exposed to various fluoride concentrations, any health effects caused by fluoride would likely manifest themselves in kidney cells. However, several large community-based studies of people with long-term exposure to drinking water with fluoride concentrations up to 8 ppm have failed to show an increase in kidney disease.

In a report issued in 1993 by the National Research Council, the Subcommittee on Health Effects of Ingested Fluoride stated that the threshold dose of fluoride in drinking water which causes kidney effects in animals is approximately 50 ppm—more than 12 times the maximum level allowed in drinking water by the Environmental Protection Agency. Therefore, they concluded that "ingestion of fluoride at currently recommended concentrations is not likely to produce kidney toxicity in humans. . . .

Many people with kidney failure depend on hemodialysis (treatment with an artificial kidney machine) for their existence. During hemodialysis, the patient's blood is exposed to large amounts of water each week (280–560 quarts). Therefore, procedures have been designed to ensure that the water utilized in the process contain a minimum of dissolved substances that could diffuse indiscriminately into the patient's bloodstream. Since the composition of water varies in different geographic locations in the United States, the U.S. Public Health Service recommends dialysis units use techniques such as reverse osmosis and de-ionization to remove excess iron, magnesium, aluminum, calcium, and other minerals, as well as fluoride, from tap water before the water is used for dialysis.

Patients with renal dysfunction and especially young children may have an increased requirement for water intake. However, there is no evidence of any risks to children with renal disease from fluoride at the doses recommended for the fluoridation of water supplies. There is no evidence that fluoride intake from sources other than water fluoridation, such as fluoride supplements, rinses, and toothpastes, poses any risk to patients with renal disease, once the normal precautions applying to the use of these products are carried out.²⁷

I believe that I have objectively reviewed the primary literature on water fluoridation that is available to me. In some cases I have referred to reviews of the literature compiled by others. In conclusion, I continue to find evidence that supports the use and expansion of water fluoridation to community water systems that have not yet been fluoridated. Water fluoridation is supported by many international organizations and continues to be a safe and effective public health program to reduce the burden of dental caries, particularly among the more vulnerable of our citizens.

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Responses to "Texaco and Its Consultants"

CHEVRON'S PERSPECTIVE

To the Editor:—We are writing in response to the open letter signed by 61 scientists that was published in the April–June 2005 issue of *The International Journal of Occupational and Environmental Health*. We welcome the opportunity for an open and honest discussion of the allegations that have been made against Chevron Corporation, the role of science in determining the legitimacy of those allegations and the appropriate channels of communication when discussing scientific data and opinions.

When research is first published and there are concerns about its quality, the research literature is an appropriate place to air scientific concerns, assuming the original work was published in an open journal. However, when the research is used to support an advocacy position or promote public policy, the pages of the scientific literature are no longer an adequate forum because the intended audience goes well beyond the scientific community. Therefore, for the matter at hand—the litigation facing Chevron in Ecuador—relying only on scientific journals as the arena

for discussion of the studies in question would be quite anomalous. Also, as we point out below, the fact that a number of the health studies referenced by the plaintiffs have not been published in the open literature provides another reason discussion of the studies must occur outside the realm of peer-reviewed journals. For the sake of putting this matter in the proper context, it is important to understand the background of this issue.

The litigation which is central to the issue here relates to environmental and health allegations made by a group of lawyers and activists on behalf of residents of the Oriente region of Ecuador, and concern the actions of a subsidiary of Texaco Inc. which was a minority partner and operator in an oil consortium with Petroecuador, the state oil company. (Chevron merged with Texaco in 2001.) The case began in May 2003 before the Superior Court in Nueva Loja, a small town in the Ecuadorian rainforest, and in August 2004 the Court commenced process to inspect 122 oil field sites to determine whether a remediation program carried out by Texaco Petroleum was effective, and whether there is any evidence of harmful

levels of petroleum-related contaminants in the areas remediated by Texaco Petroleum.

During the entire course of this litigation, the plaintiffs, led by their lawyers and supported by a number of activist groups, have waged an aggressive publicity campaign to win support for their efforts. This campaign has been characterized by misinformation, distortion and fabrication. Included in this publicity campaign has been the promotion of a handful of medical reports and studies that suggest, or attempt to establish, a link between these oilfield operations and health concerns in the region.

Chevron's communications efforts in regard to this litigation have primarily been in response to the publicity campaign carried out by the plaintiffs and their allies. This has included efforts by the company to address the allegations of health impacts, and respond to the characterizations made by the plaintiffs of the medical reports they have promoted.

The authors of the open letter characterize this as "an issue relevant to scientific integrity." We agree there is an issue of scientific integrity, since in our view, the manner in which the plaintiffs and their supporters have characterized the science in this matter threatens the integrity of the scientific process. Ironically, the very fact that the open letter is being promoted and circulated by the plaintiffs' supporters should be a clear indication of their own efforts to sway public opinion.

The manner in which we have communicated to the public on the issues associated with this litigation—including the matter of the health allegations—is not only appropriate, but necessary in order to restore balance and accuracy to the public's understanding of this issue. They deserve the opportunity to reach informed opinions on this matter, which is not possible through the one-sided and distorted portrayal of the facts by plaintiffs' supporters.

We find it interesting that the letter does not dispute the broad consensus, reached independently by established experts in the field of epidemiology and tropical medicine, that the health studies promoted by plaintiffs are flawed, biased and inconclusive. Indeed, in many instances the authors of these studies acknowledge limitations of their research, a detail the plaintiffs and their supporters conveniently overlook. Equally important, the open letter does not address the more plausible causes of health problems in the Oriente region of Ecuador.

To date, the scientific data and findings that Chevron's technical experts have presented to the Court are consistent with the view that the health concerns of the region are not associated with the oilfield operations, but rather, are more likely to be associated with other, non-oil-related concerns that have been well established. These data also demonstrate that the remediation program carried out by Texaco Petroleum was effective and in accordance with Ecuadorian and world standards for the time.

For the benefit of the public, including the scientific community, Chevron maintains a comprehensive Web site on this matter, <<http://www.texaco.com/sitelets/ecuador/en/>>, which includes summaries of the technical reports that have been submitted to the court, and the analyses conducted by the independent epidemiologists and physicians who reviewed the medical studies promoted by the plaintiffs.

We believe it is important to promote, and participate in, legitimate scientific and public dialogue on these important matters and to do so in an open manner.

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ELEVATING THE LEVEL OF SCIENTIFIC DISCOURSE

To the Editor:—Breilh et al.¹ have raised questions "relevant to scientific integrity" regarding a short scientific review that we wrote on three epidemiology reports on cancer rates in the Amazon region,² and some other events that transpired afterwards.³ Their letter suggests that reviewing a scientific study is itself a sociopolitical statement. As we stated in our review, we support fully the preservation of the Amazon forests. Also, we do not condone the use of excerpts from our review in a newspaper advertisement. On these two points, we have some common ground with Breilh and his colleagues. Nevertheless, we believe that it would be a misuse of science to use weak or flawed studies as a pretext to support the claim that the reason to preserve the Amazon environment is to avoid an adverse effect on cancer rates. The Amazon environment is worth preserving regardless of any impact on cancer rates. Even for the noble goal of preserving nature, however, we must be forthright about the difference between strong scientific evidence and weak scientific evidence. To do otherwise is to demean the science, and would defeat the purpose that Breilh et al. avow.

Breilh et al. had surprisingly little to say about our specific comments on the three papers. Instead, they criticized our review because we "never referred to industrial and environmental exposure records. . . ." We agreed to review three specific epidemiologic studies. We did not agree to conduct a comprehensive review of the effects of environmental contamination in the Amazon region, a topic outside our area of expertise. With regard to the substantive assessment of the papers, we note that the authors of the only published paper of the three that we reviewed wrote that "this ecologic study cannot lead to causal inference."⁴ We agree.

Breilh et al. also criticized our review because it was solicited by and submitted to Chevron-Texaco rather than submitted to a scientific journal. If our work had involved any new findings, we would have insisted, as is our policy, on the unfettered right to publish. But our letter to Chevron-Texaco was not new research. It was a short commentary on three reports that evidently were central to a legal dispute. Thus, Breilh et al. contend that Chevron-Texaco should not be allowed to request an expert evaluation of scientific work that bears on a dispute to which it is a party. They also imply that it is inappropriate to review scientific studies for corporations embroiled in legal concerns. Apparently, in their view, one side in a dispute can obtain advice but the other side may not. This lopsided view is especially odd in that,

among the names we recognize signing the letter with Dr. Breilh are some who have been regularly engaged as experts in such legal contests, and yet they did not recuse themselves from signing the letter.

Breilh et al. suggest that scientific dialogue should occur in journals rather than in the press. Nevertheless, they released their letter to the press before it was published. Furthermore, the usual practice for an editor of a journal receiving such negative comment would be to invite a response. Dr. LaDou did not do so. We note also that he is one of the authors of the letter. So much for high-minded principle.

We wish that the letter of Breilh et al. had been what it pretends to be, an appeal to elevate the level of scientific discourse. Its pugnacious tone, however, is not an auspicious beginning for any dialogue. We

think a substantive discussion is preferable to a polemic aimed at setting rules for who should be able to communicate with whom and under what circumstances.

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