Letters to the Editor

To the Editor:

The recent paper by Burt and Beltran (1) claimed to respond to my published critiques of water fluoridation (2-11). The above authors omitted reference to more recent publications (8-11) and they appear to have not read the earlier ones carefully. As with their "response" to Dr. Diesendorf's thesis, they comment on only a small fraction of my case. They declined to discuss one of my main findings—that concerning the inexcusable omissions and manipulations of data in early studies which purported to "demonstrate" enormous benefits from water fluoridation (7,11). Replying to the "responses" as they appear in the Burt/Beltran paper:

1. They state, concerning my earlier data on social rank and fluoridation in Auckland (4,12): "When presented without explanation, they raise the question of whether the social class boundaries in the 1966 study and the 1981 census were the same. If they were not, then Colquhoun's social rank comparisons are invalid." If they had read my study, they should have known that it did give an explanation, describing the changes which occurred in the period, and how "it was found that suburbs were generally ranked as before (4)."

2. They then state: "A worse flaw came to light in a personal communication from Hunter . . . ," and give the wrong reference (4) for an alleged error (not of my making but in the Health Department's official statistics) affecting one figure in one table of some 1982 data published later (5). The unpublished allegation circulated by Hunter was not sent to me, or to the journal which had published the data. After lengthy correspondence, the New Zealand Health Department refused access to the records to verify or correct the alleged error. Eventually, a two-page editorial (13) fully related the events and correspondence, and stated: "These vague and misleading allegations were never directed to him or us, but disseminated throughout the world as if simple assertion validated them. While differences of opinion in scientific matters are normally addressed through letters in other published responses, fluoridation occupies a special status . . . The reader will have to judge whether the critique and the conduct of the New Zealand Health Department meet acceptable standards of scientific and professional integrity." In the correspondence the Health Department had conceded that my other published data (its own official statistics) were accurate. Yet, on the basis of one unpublished, unproven allegation, Burt and Beltran have concluded that "Colquhoun's data are questionable."

3. Burt and Beltran state, "Colquhoun defended the variety of the 'permanent fillings per child' measure . . . " and they then again use an unpublished personal communication, giving examples of diagnostic variability, in Auckland dental clinics, to cast doubt on my use of the measure. Had they read my study (4) carefully, they would have known that such variability, during a transitional period of changing diagnostic procedures, was specifically acknowledged and discussed in the study, which then pointed out: "But, at the time the information for this study was gathered, diagnostic criteria within the district under study were uniform."

4. Burt and Beltran then state, "It is likely, for example, that a higher proportion of preschoolchildren presented for care in the higher social rank areas than in the lower. If so, the measure of permanent fillings per child would naturally be depressed in the higher social rank areas." A reading of my study should have shown them that this very possibility was checked, for: "As in 1966 and 1974, these patient groups had similar age and sex distributions. Most of the children were primary school age (5–11 years) with smaller numbers of preschoolers and 11–13-year-olds spread evenly for both sexes throughout the social rank areas (4)."

5. Burt and Beltran then state, "The potentially different uses of fluoride toothpaste and supplements were not pursued." This question was pursued in my later study (8), where reports of fluoride tablet use, from my own and other studies, were discussed. The point does not strengthen the Burt and Beltran case. In the nonfluoridated part of Auckland, a low-income area, there was very little such fluoride use (8), yet the children required fewer fillings than in the whole varied-income area of fluoridated Auckland (4).

6. Burt and Beltran also state of my earlier study, "Any potential bias from ethnic differences in caries experience and attitudes to oral health was not pursued." This possibility was also pursued and discussed in my later paper (8); again, the evidence did not support the Burt and Beltran view.

7. Burt and Beltran state that other New Zealand studies (14-16) "demonstrate that DMFT scores in children from schools in fluoridated areas were 14–35 percent lower than in children from schools in nonfluoridated areas." But none of the cited studies do report differences as high as 35 percent. However, one of them (15), and other Health Department reports (17,18), have claimed, for the whole of New Zealand, 14 to 16 percent lower DMFT for 12- and 13-year-olds in fluoridated areas. Far from making "only passing reference to other studies," as Burt and Beltran claim, both my earlier and later studies specifically countered the above misleading claim: "The combined fluoridated areas, being mostly cities and large towns, are of higher income level than the combined unfluoridated areas, which are mostly rural and small towns" (4) and "the above comparisons are of different kinds of populations: the one largely of cities and large towns and the other largely of rural and small-town areas" (8).

8. Burt and Beltran then state that in three other recent studies in New Zealand (19–21), "mean DMFT and dmft scores were 29–42 percent lower than in children from fluoridated areas." Of two of those studies (19,21) I had commented: "They were studies of primary teeth of very small samples—for 5-year-olds as few as 12, while there was more decay, not less, among upper class 7-year-olds" (8). Of the other study (20), I explained that it "reported a benefit to 9-year-olds, but the wholly urban fluoridated sample was compared with a partly rural and small-town sample. It did not consider socio-economic factors . . . " (8). I also pointed out: "None of these recent studies reported differences in caries-free percentages. Such small-sample studies are not supported by the present and another study of the permanent teeth of large populations of older children. If the above studies did demonstrate a fluoridation benefit to younger children, any lasting benefit must be small" (8).

By citing such inadequate and limited sets of data Burt and Beltran reinforce my main finding: that large surveys and population studies show that the benefits claimed for fluoridation are exaggerated. For example, dental data obtained for 98 percent of all New Zealand 12- and 13-year-olds before enrolling at high school, showed that more of them were free of dental decay in the largest nonfluoridated center (Christchurch) than in any of the other fluoridated, main population centers (8). Similar data from Canada (22,23), Denmark (24,25), and Australia (26-28) have been published. "Diminishing returns" and "population shifts" cannot explain away all such data. Dental data obtained from the entire population, and very large samples of five-year-old new patients in the New Zealand School Dental Service suggest that dental decay had started to decline before the introduction of fluoridation and other uses of fluoride (9). Diesendorf's critique (26) drew attention to such reductions, unrelated to fluoride, in other lands.

In their criticisms of both Diesendorf's and my evidence, Burt and Beltran can make a plausible-sounding case only by ignoring large sections of our presentations, and by misrepresenting those sections which they attempt to refute.

John Colquhoun, BDS, DipEd, PhD
Education Department
The University of Auckland
Auckland, New Zealand

References

1. Burt BA, Beltran ED. Water fluoridation: a response to critics in Australia and New Zealand. J Public Health Dent 1988;48(4):214-9.
2. Colquhoun J. Disfiguring dental fluorosis in Auckland, New Zealand. Fluoride 1984 Oct;17:234-42.
3. Colquhoun J. New evidence on fluoridation. Soc Sci Med 1984;19:1239-46.
4. Colquhoun J. Influence of social class and fluoridation on child dental health. Community Dent Oral Epidemiol 1985 Feb;13:37-41.
5. Colquhoun J. Fluoridation in New Zealand: new evidence. Part One. Am Lab 1985 May;17:66-72.
6. Colquhoun J. Fluoridation in New Zealand: new evidence. Part Two. Am Lab 1985 Jun;17:98-102.
7. Colquhoun J, Mann R. The Hastings fluoridation experiment: science or swindle? The Ecologist 1986;16(6):243-8.
8. Colquhoun J. Child dental health differences in New Zealand. Community Health Studies 1987;11:85-90.
9. Colquhoun J. Decline in primary tooth decay in New Zealand. Community Health Studies 1988;12:187-91.
10. Colquhoun J. The decline in primary tooth decay in New Zealand before the use of fluorides. Fluoride 1988;21:1-4.
11. Colquhoun J, Mann R. The Hastings fluoridation experiment: postscript. The Ecologist 1987;17(2):125.
12. Colquhoun J. The influence of social rank and fluoridation on dental treatment requirements. NZ Dent J 1977;73:146-8.
13. Editorial. Am Lab 1986 Sept;18(9):8,10.
14. Hunter PBV. The prevalence of dental caries in 5-year-old children in 1977 and 1982. NZ Dent J 1984 Jan;80:14-6.
15. Hunter PBV. The prevalence of dental caries in 12- and 13-year-old New Zealand children in 1977 and 1982. NZ Dent J 1884 Jan;80:16-8.
16. Hunter PBV. The prevalence of dental caries in 8- and 9-year-old New Zealand children. NZ Dent J 1985 Jan;81:23-5.
17. Annual Report, NZ Department of Health. Wellington: Govt Printer, 1985.
18. de Liefde B, Ritchie GR. Evaluation of dental public health in New Zealand. NZ Dent J 1984 Jan;80:8-14.
19. Evans RW, Beck DJ, Brown H, Silva PA. Relationship between fluoridation and socioeconomic status on dental caries experience in 5-year-old New Zealand children. Community Dent Oral Epidemiol 1984 Feb;12:5-9.
20. de Liefde B, Herbison GP. Prevalence of developmental defects of enamel and dental caries in New Zealand children receiving differing fluoride supplementation. Community Dent Oral Epidemiol 1985 Jun;13:164-7.
21. Fergusson DM, Horwood LJ. Relationships between exposure to additional fluoride, social background, and dental health in 7-year-old children. Community Dent Oral Epidemiol 1986 Feb:48-52.
22. Johnston DW, Grainger RM, Ryan RK. The decline of dental caries in Ontario schoolchildren. J Can Dent Assoc 1986 May;52:411-7.
23. Gray AS. Fluoridation: time for a new baseline? J Can Dent Assoc 1987 Oct;53:763-5.
24. Bille J, Hesselgren K, Thylstrup A. Dental caries in Danish 7-, 11-, and 13-year-old children in 1963, 1972 and 1981. Caries Res 1986;20:534-42.
25. Thylstrup A, Bille J, Bruun C. Caries prevalence in Danish children living in areas with low and optimal levels of natural water fluoride. Caries Res 1982;16:413-20.
26. Diesendorf M. The mystery of declining tooth decay. Nature 1986 Jul;322:125-9.
27. Diesendorf M. A re-examination of Australian fluoridation trials. Search 1986;17:256-62.
28. Diesendorf M. Misleading publicity for a fluoridation trial. New Zealand Med J 1988 Dec;101:832-3.

To the Editor:

A recent paper by Burt and Beltran (1), hereafter referred to as B&B, offered "a response" to my critique in *Nature* (2) and to an early version of Dr. John Colquhoun's critique of the alleged enormous benefits of water fluoridation. In their summary of the alleged shortcomings of my critique, B&B claimed that "Diesendorf employed an outdated view of how fluoride exerts its anticariogenic action" and took a number of quotations out of context.

Even if these claims were correct (and they are not; see below), they would not change the structure of my argument, which depends neither on the incompletely understood mechanism of action of fluoride nor on quotations of other authors' *opinions*.

My thesis is that the benefits of water fluoridation have been exaggerated. My case is actually presented in two complementary papers (2,3) and is based on four

separate points, each of which is supported by a significant body of scientific evidence published by authors who are almost all proponents of fluoridation:

1. The prevalence of dental caries in unfluoridated parts of the developed world had declined substantially over the past two to three decades (2). This decline has often involved a reduction of about 50 percent in DMFT, and in several cases (e.g., Sydney, Australia; Gloucestershire, UK; New Zealand; and Ontario, Canada) has commenced *before* water fluoridation (and the use of fluoride in other forms became significant (2,4,5).

2. The prevalence of dental caries in "optimally exposed" age groups of children in fluoridated areas has continued to decline long after the maximum possible benefit of water fluoridation could have been achieved (2,3). This additional decline has sometimes been attributed incorrectly to fluoridation.

3. Some of the major, quasiexperimental studies of human populations, which purport to prove or "demonstrate" the alleged enormous benefit of water fluoridation, are of poor scientific quality (3,6,7,8).

4. In Australia, New Zealand, the United States, Sweden, Japan, and Canada, the prevalences of caries in some unfluoridated regions are equal to or less than those in artificially or naturally fluoridated parts of the same countries (2,5,9,10).

Although my critique of the effectiveness of water fluoridation does not question the effectiveness of high-concentration (≧1,000 ppm fluoride) topical fluorides, it does point out that some of the large observed reductions in dental caries occurred before fluoride in any form was used widely, and so nonfluoride mechanisms must be playing a significant role.

B&B's comments are now examined for each of the above four points.

Caries Decline in Unfluoridated Areas

My critique (2) cites over 20 studies from eight developed countries. Further examples have been published from Canada (5,10) and France (11). The only matter of substance taken up by B&B is to question the evidence I put forward that caries declined substantially in Sydney, Australia, before fluoridation in 1968. This evidence comprises two independent studies by proponents of fluoridation.

The first, a longitudinal study by Lawson et al. (12), showed a rapid increase in the prevalence of "naturally sound" caries-free teeth in primary schoolchildren from 1961 (4%) to 1963 (14%) to 1967 (20%). Contrary to B&B, Lawson et al. (12) did *not* state that the results were based on "cursory dental screenings to identify children in need of treatment," etc. Furthermore, the question as to whether or not the sample was intended to be representative of all Sydney primary schoolchildren is irrelevant to the present debate, so long as the selection of schools was made consistently from the white-collar area, and it was for the pre-1974 observations. In summing up the validity of their data, Lawson et al. (12) stated: "There were variations between schools in the incidence of dental decay, but they all reflected the same pattern of improvement. Therefore, the information is relevant for making broad comparisons."

The independent supporting evidence in Sydney comprises two surveys by Barnard (13,14) of DMFT in 13–14-year-olds in 1954 and 1972 (which B&B list inaccurately as 1973). The point at issue is: could most of the very large observed reduction in DMFT of 5.0 or 45 percent have occurred in the four-year period from the commencement of fluoridation in 1968 to 1972? In my critique (2), I cited Dr. Lloyd Carr, then director of the Oral Health Unit in the Australian Department of Health and one of Australia's leading proponents of fluoridation, who stated that the four-year period would *not* have contributed significantly. The available Australian data support Carr's statement. For instance, in the city of Canberra (located about 300 km from Sydney), DMFT in 12-year-olds declined by only 0.77 (11%) over the four-year period following the commencement of fluoridation in 1964 (15).

Finally, quoting from an unpublished manuscript by an Australian fluoridation promoter, Graham Craig, B&B create the misleading impression that there exist standardized surveys showing that DMF rates in Sydney "were essentially stable from the mid-1950s through the mid-1960s." What B&B's informant, Craig, has actually done in his unpublished manuscript is to compare Barnard's 1954–55 survey of 13–14-year-olds with a 1963 survey of 12-year-olds conducted by a different set of authors, Burton et al. (16), on a totally different sample. It is difficult to draw any conclusion from this comparison of incompatible data sets. (In addition, B&B have described "1963" loosely as "the mid-1960s.")

B&B then provide a long, irrelevant discussion of papers I cited by Kalsbeek (17) and by Anderson et al. (18). These papers provide the data for two of my further examples of large declines in caries prevalence in unfluoridated areas. Nothing B&B say about these papers contradicts this central point or the data supporting it. Rather, B&B seem to be trying to create the impression that, in the course of making supplementary points, I have quoted the opinions of these authors out of context. To this end they quote larger sections than I was able to include in *Nature*. However, both B&B and I have quoted Kalsbeek and Anderson et al. correctly, and I am happy with B&B's choice of expanded quotations, which still bring out clearly the supplementary points I wished to make—namely, that (1) it is the opinion of some respected dental researchers that nonfluoride factors may have played a role in the caries decline, and (2) some reductions in caries occurred *before* fluoridation or the use of fluoride in any form were introduced. So the alleged "out-of-context quotations" are a nonissue.

Caries Decline in "Optimally Exposed" Age Groups

These declines can be identified in the published results of profluoridation dental researchers for fluoridated Tamworth and Canberra, Australia; Anglesey, UK; Karl-Marx-Stadt, East Germany; and in many other

fluoridated parts of the developed world. A number of proponents have attributed these declines incorrectly to water fluoridation. But because the declines occurred *after* the particular age groups of children had become "optimally exposed"—i.e., had first ingested fluoridated water from birth—it is impossible for water fluoridation to have been the cause (2).

This argument and conclusion are independent of the mechanism of action of fluoride in reducing tooth decay. B&B's discussion of mechanisms at this point distracts attention away from implications of the point, which are that claims that fluoridation was responsible for the observed declines in tooth decay in optimally exposed age groups are clear examples of the exaggeration of the effectiveness of water fluoridation.

In this light, I suggest that B&B read carefully my comments (2,19) on the misleading press release by Graham Craig and Noel Martin entitled "Fluoridation dramatically cuts tooth decay in Tamworth."

Poor Scientific Quality of Studies on Effectiveness

B&B ignore this point in their "response" to my work. In the section of their "response" that relates to Colquhoun's work, they mention the problem, but decline to discuss it.

Equal or Less Caries in Unfluoridated Regions

B&B ignore the evidence presented in my paper, but attempt to explain a small part of the evidence from New Zealand. The principal response on this point will no doubt be handled by Dr. Colquhoun.

Significance of the Mechanism

In *Nature* (2), I referred loosely to high-concentration (≥1,000 ppm) topical fluorides as "topical fluorides" and the use of low-concentration (about 1 ppm) fluorides, which are intended to be ingested, as "systemic uses of fluoride." B&B are wrong in trying to deduce from this simple classification of intended method of fluoride delivery that "Diesendorf also adheres to the view that fluoridation is effective only because it is incorporated into developing enamel, a view which is long outdated." If B&B had read my paper more carefully, they would have seen that this false interpretation was explicitly contradicted on page 129, where I stated: "Indeed, a promising explanation is that the apparent benefit of fluorides is derived from their topical action. Then, since fluoridated water has a fluoride ion concentration 10^{-3} times that of fluoride toothpaste, its action in reducing caries is likely to be much weaker."

Since writing the *Nature* paper, I have found more empirical evidence, doubtless well known to B&B, which provides support for the previous statement and strongly suggests that there is negligible reduction in tooth decay from the systemic intake of fluoride:

1. In humans: No significant relationship has been demonstrated between caries experience of the individual and fluoride content of the enamel. Furthermore, the difference in fluoride content of the enamel in low and "optimal" areas is too small to produce a significant reduction in caries prevalence (20,21).

2. In the rat model: The anticaries effect of fluoride is primarily posteruptive (22,23). No caries reduction could be obtained from an implanted subcutaneous device that released fluoride slowly into bloodstream (23). The anticaries effect at 1 ppm fluoride is considerably smaller than at 10 ppm (22).

So, although my critique of fluoridation (2) does not depend on the mechanism of action of fluoride, the growing evidence that there are negligible *systemic* benefits from fluoride weakens further the case for fluoridating *drinking* water. The same (probably small) benefit would be obtained from mouthrinsing several times a day with a 1 ppm fluoride solution and then spitting the solution out.

Finally, since the view that fluoride acts by means of a systemic mechanism is "outdated," why are some dental associations still recommending fluoride supplements for breastfed infants?

Conclusions

B&B have offered only the image of a response to my critique of fluoridation, instead of a response of substance. My case is based on four separate points, each of which is supported by a significant and growing body of empirical data from (at least) several groups of researchers in several different countries. In their "response," B&B have only come to grips with one of the case studies (Sydney, Australia) supporting one of these points, and even in this case their objections have been easily refuted.

Mark Diesendorf, PhD
Human Sciences Program
Australian National University
Canberra ACT 2601, Australia

References

1. Burt BA, Beltran ED. Water fluoridation: a response to critics in Australia and New Zealand. J Public Health Dent 1988;48(4):214-9.
2. Diesendorf M. The mystery of declining tooth decay. Nature 1986;322:125-9.
3. Diesendorf M. A re-examination of Australian fluoridation trials. Search 1986;17:256-62.
4. Colquhoun J. Decline in primary tooth decay in New Zealand. Community Health Studies 1988;12:187-91.
5. Johnston DW, Grainger RM, Ryan RK. The decline of dental caries in Ontario schoolchildren. J Can Dent Assoc 1986;52:411-7.
6. Sutton PRN. Fluoridation: errors and omissions in experimental trials. 2nd edition. Melbourne, Australia: Melbourne University Press, 1960.
7. Colquhoun J, Mann R. The Hastings fluoridation experiment: science or swindle? The Ecologist 1986;16:243-8.
8. Diesendorf M. Anglesey fluoridation trials re-examined. Fluoride 1989;(in press).
9. Colquhoun J. Child dental health differences in New Zealand. Community Health Studies 1987;11:85-90.
10. Gray AS. Fluoridation, time for a new baseline? J Can Dent Assoc 1987;53:763-5.
11. Cahen PM, Turlot JC, Frank RM, Clement G, Seckler G. Comparative study of oral conditions in schoolchildren of Strasbourg, France, 1974-85. Community Dent Oral Epidemiol 1987;15:211-5.
12. Lawson JS, Brown JH, Oliver TI. The dental health revolution: the dramatic improvement in dental health of schoolchildren in the northern metropolitan region of New South Wales. Med J Aust 1978;1:124-5.

13. Barnard PD. NH&MRC Spec Rep Ser 1956;8:30-43. Canberra: National Health & Medical Research Council.
14. Barnard PD, Clements FW. Oral health care in Australia. Int Dent J 1976;26:320-6.
15. Carr LM. Fluoridation in Canberra. Part II. Aust Dent J 1972;17:335-9.
16. Burton, VJ, Rob MI, Craig GG, Lawson JS. Changes in the caries experience of 12-year-old Sydney schoolchildren between 1963 and 1982. Med J Aust 1984;140:405-7.
17. Kalsbeek H. Evidence of decrease in prevalence of dental caries in the Netherlands: an evaluation of epidemiological caries surveys on 4-6 and 11–15-year-old children, performed between 1965 and 1980. J Dent Res 1982;61(Spec Iss):1321-6.
18. Anderson RJ, Bradnock G, Beal JF, James PMC. The reduction of dental caries prevalence in English schoolchildren. J Dent Res 1982;61(Spec Iss):1311-6.
19. Diesendorf M. Misleading publicity for a fluoridation trial. New Zealand Med J 1988;101:832-3.
20. Fejerskov O, Thylstrup A, Larsen MJ. Rational use of fluorides in caries prevention. Acta Odontol Scand 1981;39:241-9.
21. Beltran ED, Burt BA. The pre- and posteruptive effects of fluoride in the caries decline. J Public Health Dent 1988;48(4):233-40.
22. Spuller RL, Beiraghi S, Rosen S, Beck FM. The effect of low levels of sodium fluoride in drinking water on the incidence of dental caries in rats. Caries Res 1986;20:556-8.
23. Mirth DB, Adderly DD, Monell-Torrens E, Amsbaugh SM, Li S-H, Bowen WH. Comparison of the cariostatic effect of topically and systemically administered controlled-release fluoride in the rat. Caries Res 1985;19:466-74.

To the Editor:

We appreciate receiving the comments from Dr. Diesendorf and Dr. Colquhoun on our recent paper. We agree with them that there is a good deal of research still required on the subject of water fluoridation, and like them we hope that it will continue to be carried out in an atmosphere of open-mindedness. One of us wrote some years ago: "The great tragedy of fluoridation as a public policy issue is that it cannot be debated publicly in a dispassionate manner . . There is, it seems, no middle ground" (1). While not much has changed in ten years, we think it is still worth trying to find that elusive middle ground.

Having stated that philosophy, Diesendorf and Colquhoun have raised some issues to which we should respond. Diesendorf states as his thesis that the benefits of water fluoridation have been exaggerated. If he is referring to the more extravagant claims made by some proponents of fluoridation down the years, we do not disagree; indeed, one of us stated this view years ago in the work cited above (1). In our recent paper (2), we chided Lawson et al. for making just such a claim. As a good scientist, Diesendorf no doubt accepts as part of his thesis that the criticisms of water fluoridation also have been exaggerated by some opponents.

Diesendorf states that his thesis is based on four issues, the first of which is that the decline in caries prevalence in the economically developed world began before significant use of fluoride. If he had read our previous work more carefully (to paraphrase both respondents), he would have seen that one of us presented the same thesis several years ago (3). If he had read further still, he would have seen it raised even before the caries decline was generally recognized (4). We maintain, however, that fluoride is likely to be the major reason for the sharp decline in recent years, if not the only one.

Diesendorf's second point, about the caries decline in "optimally exposed" groups, seems to repeat what he said in his 1986 paper. We do not agree with the statement in his letter that the observed declines are "clear examples" of the exaggeration of fluoridation's effectiveness. It is good practice in epidemiology to be cautious about before-after comparisons without controls, as in Tamworth, because of the several threats posed to internal validity of cause-and-effect conclusions (5). We discussed the Tamworth situation in our response, and stated that the "optimally exposed" philosophy depended on a reliance on the systemic action of fluoride. Perhaps we are missing something, but we cannot see that this argument is independent of the mechanisms of fluoride, and we do not agree that it "distracts" from Diesendorf's main point.

The mechanism we outlined in our companion paper (6) provides an explanation for the continuing decline of caries in fluoridated areas. Multiple exposures to fluoride in several forms could also be affecting disease prevalence, as could the improved availability of fluoride from toothpaste in recent years. Furthermore, if dentists are not filling the same early lesions that they once did then DMF data would certainly be influenced. If Diesendorf is really saying, as his letter suggests, that he finds the statements of Professors Martin and Craig to be extravagant, then he should discuss the issue with them, not us.

His third point is the "poor scientific quality of studies on effectiveness." Sutton's criticism of the first community trials in North America in 1945–46 (7) was quoted by Diesendorf in this context, and indeed it represents a scholarly and thorough critique of studies conducted before the science of community trials was refined. The main criticism we would make of Sutton's work, from the viewpoint of research design, is that he perhaps did not distinguish sufficiently between trivial issues and potentially more serious ones, though we will touch on the main issue of Sutton's work later. The first North American trials, however, were just the beginning of a host of studies, and focusing on these early studies (we have always called them "pioneering" rather than "classical") distracts from the main issue that there is a heavy body of evidence to favor water fluoridation.

Murray and Rugg-Gunn (8), in their response to Diesendorf, counted 95 studies that showed consistent results in favor of water fluoridation. Despite Colquhoun's criticism of the Napier–Hastings study and Diesendorf's view of the Australian fluoridation trials, that weight of evidence is extremely difficult to argue away.

On the fourth point, caries in nonfluoridated areas, there are many examples of DMF values in individual nonfluoridated communities being higher than those in individual fluoridated ones. Baseline mean DMFS values in the National Preventive Dentistry Demonstration Program in the United States (9) demonstrate this point, as shown here for Grade 5 children:

Fluoridated Sites		Nonfluoridated Sites	
El Paso	2.29	Wichita	3.67
New York	3.50	Tallahassee	3.86
Chattanooga	3.62	Pierce County	4.55
Minneapolis	3.75	Billerica	4.56
Hayward	4.55	Monroe	6.75

These data are not adjusted for socioeconomic status, racial and ethnic distributions, dietary and cultural practices, length of residence in the community, dental care received, nor for other exposures to fluoride; thus, the fact that there are individual nonfluoridated sites with lower DMFS values than those in fluoridated communities is not surprising. Similar situations have been described in Canada (10), and Colquhoun (11) concluded that the oral health of children in nonfluoridated Christchurch is no worse than in other fluoridated New Zealand cities. We had personal experience with the caries decline in nonfluoridated areas in a recent study on the caries–sugars relationship, where low caries increments were the norm (12). Overall, however, the evidence favors lower DMF values in fluoridated communities, well illustrated in a recent report from Ireland (13).

While wishing to avoid nitpicking (e.g., on what constitutes a longitudinal study), there are some additional minor points from the two letters that require comment. Diesendorf chided us for quoting other people's opinions (presumably referring to the personal communications we received from Craig and Hunter), yet uses an opinion from Carr (that four years of fluoridation would not have contributed significantly to a caries reduction) when it suits his thesis to do so. His use of the word "informant," in reference to our personal communications from Craig, seemed unnecessarily snide. Of course we sought other opinions from the countries concerned, just as Diesendorf prepared his letter in cahoots with Colquhoun.

Diesendorf states that our correcting of his quotes from Kalsbeek and Anderson are a "nonissue." We disagree; on rereading the various pieces, it still seems to us that Diesendorf misrepresented the comments these authors made. A final comment on Diesendorf's paper is that if his thesis is no more than saying that the benefits from water fluoridation have been exaggerated, then why give the paper a titillating title like "The Mystery of Declining Tooth Decay"?

We did not discuss Colquhoun's 1986 critique of the Napier–Hastings study in New Zealand because it was not directly relevant to the interpretation of his principal argument. Since our "informants" have given us another interpretation of the the circumstances of the Napier–Hastings study, either one side or the other is incorrect. It seems that the record could only be set straight by a dispassionate historian. We sympathize with Colquhoun in his long-running battles with the New Zealand Department of Health, but we feel that using information from officers in that department is not exactly an "unpublished, unproven allegation."

To return to research critiques, any individual research study with humans can be criticized for departures from scientific rigor. Some such departures are trivial and unlikely to affect the results; others can be more serious. That is one reason why human epidemiological studies and clinical trials are replicated where possible. In that sense, Sutton's criticisms of the early fluoridation studies, as mentioned earlier, were fair enough. *But in determining public policy, the only question that remains after all criticisms like Sutton's have been presented is whether the identified problems were serious enough to invalidate the studies.* By Sutton's own testimony to the Tasmanian Royal Commission into Fluoridation of Public Water Supplies in 1968, they do not. While Diesendorf and Colquhoun are of course familiar with this testimony, other readers may be interested in it:

Question: Would you say these five trials did not succeed at all in showing that artificially fluoridated water at 1 ppm did in fact have an effect on the incidence and gravity of dental caries?

Sutton: No, I think they showed some effect, but how much effect I am not prepared to state; that is the trouble.

Question: Sufficient effect, if no ill effects were shown, to follow from the ingestion of water at 1 ppm to be a worthwhile public health measure?

Sutton: Yes, I think so. If there were no ill effects (14).

We return to where we began, with the plea that research in fluoridation be conducted objectively, rather than seeking to justify already entrenched positions. We know that sounds rather pious, but we will continue to press that hope. For now, all of us can only proceed on the weight of existing evidence. It is our opinion that this evidence favors water fluoridation as public policy, while Diesendorf and Colquhoun disagree. They have not changed our minds with their papers and letters, although they do raise some points that are worthy of consideration. We are sure that we have not changed their minds either, though for our part we modestly hope that we have raised some points for them to consider. Perhaps we all agree on more issues than we disagree on, and that is not a bad position from which to continue research into fluoridation as a public policy issue.

Brian A. Burt, BDS, MPH, PhD
Professor and Director
Eugenio D. Beltran, DDS, MPH, MS
Program in Dental Public Health
School of Public Health
University of Michigan
Ann Arbor, MI 48109

References

1. Burt BA. Fluoridation: greedy conspiracy or public boon? Chem & Eng News 1979 Oct;22:56-7.
2. Burt BA, Beltran ED. Water fluoridation: a response to critics in Australia and New Zealand. J Public Health Dent 1988 Fall;48:214-9.

3. Burt BA. The future of the caries decline. J Public Health 1985 Fall;45:261-9.
4. Burt BA. Influences for change in the dental health status of populations; an historical perspective. J Public Health Dent 1978 Fall;38:272-88.
5. Cook TD, Campbell DT. Quasi-experimentation; design and analysis issues for field settings. Boston: Houghton Mifflin, 1979;50-9.
6. Beltran ED, Burt BA. Pre- and posteruptive effects of fluoride in the caries decline. J Public Health Dent 1988 Fall;48:233-41.
7. Sutton PRN. Fluoridation: errors and omissions in experimental trials. 2nd ed. Melbourne: Melbourne University Press, 1960.
8. Murray JJ, Rugg-Gunn A. Fluoridation and declining decay; a reply to Diesendorf. London: The British Fluoridation Society, 1987.
9. Klein SP, Bohannan HM. The first year of field activities in the National Preventive Dentistry Demonstration Program. Santa Monica, CA: Rand, 1979, Rand Report No. R-2536/1-RWJ.
10. Gray AS. Fluoridation, time for a new base line? J Canad Dent Assoc 1987 Oct;53:763-5.
11. Colquhoun J. Child dental health differences in New Zealand. Comm Health Studies 1987;11:85-90.
12. Burt BA, Eklund SA, Morgan KJ, Larkin FE, Guire KE, Brown LO, Weintraub JA. The effects of sugars intake and frequency of ingestion on dental caries increment in a three-year longitudinal study. J Dent Res 1988 Nov;67:1422-9.
13. O'Mullane DM, Clarkson J, Holland T, O'Hickey S, Whelton H. Effectiveness of water fluoridation in the prevention of dental caries in Irish schoolchildren. Comm Dent Health 1988 Dec;5:331-44.
14. Crisp MP. Report of the Royal Commissioner into the fluoridation of public water supplies. Hobart, Tasmania: Government Printer, 1968;65.

SCHEDULED DATES OF FUTURE MEETINGS OF THE AMERICAN ASSOCIATION OF PUBLIC HEALTH DENTISTRY

1989 November 1–3 _____ Honolulu, HI
1990 October 11–12 _____ Boston, MA