

CARSTAIRS RESPONDS

Both Newbrun and Friedman's letters accuse me of opposing community water fluoridation (CWF). This was not the point of my article. My article examines the arguments being put forward by scientists in the 1930s, 1940s, and 1950s, to understand how the debate over CWF played out. Despite attempts to make science as bias-free as possible, historians and philosophers of science have shown that scientific research and debates are influenced by political, social, and cultural factors. My article maintains that evidence of the safety of water fluoridation mounted quickly and that early studies showed substantial reductions in cavities. I did not claim that water fluoridation is toxic nor was I trying to "legitimize the unsubstantiated claims of anti-fluoridationists" as Friedman argues.

I did outline the arguments of scientists who believed that there might be danger in consuming too much fluoride. I made the point that dentists were understandably keen to have a measure that would significantly decrease childhood caries, while also enhancing the scientific prestige of their profession. As a result, a number of activist dentists pushed for the approval of CWF before the long-term studies were completed. The quick approval of CWF by the American Dental Association, the American Medical Association and other bodies closed down the debate, making it seem that there was unanimous agreement in favor of CWF and that further research was unnecessary.

Proponents of CWF were quick to dismiss concerns about the long-term safety of consuming fluoridated water, and they accused their opponents of being paranoid cranks (à la Dr. Strangelove). This may have contributed to the situation York researchers uncovered in 2000, when they published a systemic review of the research into CWF, which expressed dismay over the quality of research in preceding decades.¹ In short, my article is about the scientific debate over CWF in the very early years of its implementation; I am not making claims about the value of CWF today.

Friedman argues that I critique the early fluoridation studies for not being blinded and claims that it would be impossible to blind a study of fluoridation. I agree. That being

said, I do think that lack of blinding may have influenced the results of these early studies, which showed the benefits of fluoridation to be much greater than later studies indicated.² Admittedly, later studies of CWF are complicated by the "halo" effect: people who live in communities that lack CWF still drink beverages made in places that have CWF, making it very difficult to determine the impact of CWF. Studies of the effectiveness of CWF are further complicated by the widespread use of fluoridated toothpastes. Moreover, children in both fluoridated and unfluoridated communities get far fewer cavities than before.^{3,4}

Newbrun compares the introduction of water fluoridation to the initial use of penicillin. This is a faulty comparison. Tooth decay, at least in most cases, is not a life-threatening condition. Penicillin, by contrast, saved lives with a short course of treatment, whereas CWF exposed entire populations to fluorides over the life course.

Fortunately, we all agree that there is very little evidence that CWF causes any long-term health problems. I still think that it is unfortunate that the debate over CWF grew so heated and partisan so quickly. People publishing work critical of CWF have accused the profluoridation lobby of blocking publication of their research.⁵ While I am skeptical of these claims, I think that unwillingness of many profluoridationists to broker any opposition to the measure had a stultifying effect on the research by making it unpleasant for any researcher who published work that could be interpreted as hostile to CWF. There were many reasons to implement CWF, especially in the 1950s, when children got far more cavities than they do today, but the measure was not entirely without risk, and a more extended debate would have served both science and the public well. **AJPH**

Catherine Carstairs, PhD

ABOUT THE AUTHOR

Catherine Carstairs is with the History Department, University of Guelph, Guelph, ON, Canada.

Correspondence should be sent to Catherine Carstairs, Associate Professor and Chair, History Department, University of Guelph, 50 Stone Road East, Guelph, ON N1G 2W1 Canada (e-mail: ccarstai@uoguelph.ca). Reprints can be ordered at <http://www.ajph.org> by clicking the "Reprints" link.

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STUDY INCAPABLE OF DETECTING IQ LOSS FROM FLUORIDE

The Broadbent et al.¹ article on fluoride and IQ has two serious weaknesses.

First, the question is not whether community water fluoridation (CWF) reduces IQ, but whether or not total fluoride intake reduces IQ. Broadbent et al. acknowledge that CWF likely represents less than 50% of total fluoride intake. Their study did not determine total fluoride exposure, nor did it simultaneously control for the three sources of fluoride intake: CWF, fluoride supplements, and fluoride toothpaste.

It is likely the Dunedin Cohort participants had a very narrow range of total fluoride exposure. This would severely limit the study's statistical power. More importantly, their 99 participants who had no CWF were precisely the participants most likely to have taken fluoride supplements. Guha-Chowdhury et al. found that Dunedin area children without CWF who took fluoride supplements would receive greater total fluoride than CWF children.²

We calculated total fluoride intake for the CWF and non-CWF Dunedin Cohort participants using publicly available data (available as a supplement to our article at <http://www.ajph.org>). We estimated that lifetime CWF children had mean total fluoride intake of 0.7 milligram per day while non-CWF averaged 0.5 milligram per day. This small difference can explain why Broadbent et al. would not find a statistically

significant difference in IQ, even if they had assessed total fluoride intake.

Second, although Broadbent et al. criticized previous studies for failing to control for 15 potential confounders, their study failed to control for 11 of these, including important factors with available data. This is problematic because the study's non-CWF population came mainly from a single "satellite suburb": Mosgiel, New Zealand.^{3,4} This town used groundwater, whereas most of the CWF study population had surface water. Mosgiel's water was among the most corrosive in New Zealand and dissolved high levels of copper from plumbing and potentially also lead.⁵ Mean blood lead measured in the Dunedin Cohort was 11.1 µg/dl (SD ±4.91), sufficient to cause a loss of four IQ points, but was not considered in the Broadbent et al. study.^{6,7} Mosgiel's water also had high natural manganese levels, another suspected neurotoxin.^{8,9}

Data on the mothers' IQ and rural versus urban is also available for the Dunedin Cohort, but the study did not control for them. Mosgiel is more rural than the fluoridated area, potentially resulting in lower IQ in its children and their mothers.¹⁰

All these confounders would bias results away from an effect of fluoride on lowering IQ.

Confounding and the lack of contrast in total fluoride exposure may explain why no difference in IQ was found. **AJPH**

Bill Osmunson, DDS, MPH
Hardy Limeback, PhD, DDS
Chris Neurath, BSc

ABOUT THE AUTHORS

Bill Osmunson is a *Cosmetic Comprehensive Public Health Dentist, Neah Bay, WA*. Hardy Limeback is *Professor Emeritus, Faculty of Dentistry, University of Toronto, Toronto, Canada*. Chris Neurath is with the *American Environmental Health Studies Project, Lexington, MA*.

Correspondence should be sent to Bill Osmunson, PO Box 677, Neah Bay, WA 98357 (e-mail: bill@teachingsmiles.com). Reprints can be ordered at <http://www.ajph.org> by clicking the "Reprints" link.

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CONTRIBUTORS

All of the authors contributed equally to this letter.

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BROADBENT ET AL. RESPOND

The letter from Osmunson et al. raised some interesting questions about our article on community water fluoridation (CWF) and IQ. Specifically, we agree with the correspondents' assertion that children's total daily fluoride intake from CWF is insufficient to affect IQ. The correspondents asserted that, in our study, the difference in total fluoride intake between children living in CWF and non-CWF areas would have been only 0.2 milligrams per day. There are a number of problems with their aggregated calculations, but the number they reach is not far off our own estimate of an average difference of in total daily fluoride intake of 0.3 milligrams per day

through the first five years of life between study members from CWF versus non-CWF areas.

These differences are consistent with the wider literature. Guha-Chowdhury's work, used in the correspondents' calculations, estimated 0.2 milligrams per day greater total fluoride intake among children from CWF areas than non-CWF areas.^{1–3} Other researchers have estimated that the increase in fluoride intake among children aged one to three years attributable to CWF is 0.2 milligrams per day⁴ or 0.3 milligrams per day.⁵

Secondly, the correspondents mentioned data on total fluoride exposure from diet, toothpaste, and fluoride tablets. Originally, we controlled for these other sources of exposure (because our article was about CWF specifically), but since the correspondents agree that CWF is not an issue, we have now calculated estimates for total daily fluoride intake. For estimated total fluoride intake (taking into account the frequency of use of fluoride tablets and fluoride toothpaste), the mean was 0.9 milligrams per day (SD = 0.2), so there was adequate contrast to explore this in the context of the levels of fluoride used in caries control. We used these estimates of fluoride exposure in analysis, and this resulted in no meaningful change of significance, effect size, or direction in our original findings.

Thirdly, the correspondents refer to a Dunedin City Council map and assert that the study members from unfluoridated areas were exclusively from Mosgiel. This assumption is incorrect; the majority of these were from other locations across the wider Dunedin area. Nevertheless, we reran our analysis taking into account both suburb and distance from the Dunedin city center. This resulted in no meaningful change in terms of significance, effect size, or direction of our original findings.

Fourthly, the correspondents suggested that lead might be a confounder in this study, even though there was no association to be confounded. Nevertheless, we reran our analysis taking into account blood lead at age 11 years. This resulted in no meaningful change of significance, effect size, or direction in our original finding, including if we tested for estimated total fluoride intake.

This article has been cited by:

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