

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**

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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.

Lastly, the correspondents state that high concentrations of manganese in Mosgiel water could account for the lack of IQ differences. It is important to note that manganese has importance for human development, as it is involved in more than 300 enzymatic processes,⁶ and that it is not considered to be very toxic when consumed as a normal part of the diet.⁷ For children aged one to three years, the tolerable upper limit for manganese has been reported as 300 milligrams per day⁶ (most of which is sourced from food). Manganese does occur in Mosgiel water, but the mean concentration of the source water is between 0.002 and 0.005 milligrams per liter, with the exception of higher concentrations of about 0.2 to 0.5 milligrams per liter from the Old Borough Bore (which provides about 8% of Mosgiel water). Mosgiel's water has manganese concentrations that are below the maximum acceptable level of 0.4 milligrams per liter.

As we showed in our original report, and subsequent analyses described herein, we observed no evidence of a detrimental effect on IQ from fluoride at the levels used in CWF. As a further way of identifying study members with high fluoride exposure, we tested for IQ deficits for study members with dental fluorosis, and no IQ differences were observed. It is worth pointing out, however, that we have observed significantly fewer caries-affected teeth in both childhood and adulthood among those who resided in CWF areas as children. **AJPH**

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