Scientific and Critical Analysis of the 2014 New Zealand Fluoridation Report

International Critique of the Royal Society of New Zealand/Office of the Prime Minister's Chief Science Advisor's Fluoridation Report: *Health effects of water fluoridation: A review of the scientific evidence*

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According to the University of York Centre for Reviews and Dissemination:

"Systematic reviews differ from other types of review in that they adhere to a strict scientific design in order to make them more comprehensive, to minimise the chance of bias, and so ensure their reliability. Rather than reflecting the views of the authors or being based on only a (possibly biased) selection of the published literature, they contain a comprehensive summary of the available evidence."

http://www.york.ac.uk/inst/crd/fluofaq.htm#q6

The following critique discusses how the Royal Society of New Zealand / Office of the Prime Minister's Chief Science Advisor's 2014 Fluoridation Report is not a systematic review.

This critique is provided for the benefit of those seeking reliable evidence based scientific information in the fluoridation debate

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For further facts about water fluoridation see www.fluoridefree.org.nz

Findings and recommendations

Failure to review health risks and identify harm

Sections of this critique focus on health risks and margin of safety considerations, which the report from the Royal Society of New Zealand and the Office of the Prime Minister's Chief Science Advisor claims are met, but elsewhere in the report identifies that they are not met.

Many examples are given in this critique of the lack of scientific rigor in the NZ report, including several inconsistencies and inaccuracies contained in the report. The report fails to acknowledge that both the safety and efficacy of fluoridation have been questioned for decades by scientists, physicians, dentists, and other professionals, based on the available evidence.

On the questions of risk, specifically those related to dental fluorosis, lead concentrations, IQ, osteosarcoma and kidney function, the NZ report appears unjustifiably complacent. The report identified large sections of the population of NZ who exceed the toxic limits for fluoride ingestion, including:

- Bottle fed infants
- Children under 8 years old
- People with impaired kidney function
- People who drink a lot of fluoridated water
- People who have high fluoride intake from other sources such as diet and toothpaste

Political Bias

The authors of the New Zealand report adopt a noticeably biased approach. The political bias of the fluoridation promoters is demonstrated in the NZ Official Information Act correspondence included.

The "elephant in the room" is that while decay rates have fallen in areas where fluoridation was implemented, it has also fallen in areas that were not fluoridated, often **at a faster rate**. This cannot in good faith be described as a robust and thorough review of fluoridation science by experts, as it fails to address conflicting evidence which is detailed in this critique.

Recommendations

Based on the findings of this critique, it is recommended that local councils suspend water fluoridation until central government conducts thorough safety studies. The burden of proof and guarantee of fluoridation safety lies with officials who enforce, promote and allow fluoridation. **As yet, no fluoridation safety studies have ever been conducted, anywhere in the world.**

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Scientific and Critical Analysis of the Royal Society/Prime Minister's Science Advisor's Office Fluoridation Report: *Health effects of water fluoridation: A review of the scientific evidence*

Introduction

The Report was sent to a selection of independent international experts for peer review. Two of these were members of the US National Research Council (NRC) review panel, whose report was published in 2006 following three years of reviewing the scientific literature. The reviewers were chosen for their particular expertise on the science around fluoridation, and their standing in the scientific community.

The first two reviews in this critique address the toxicity issue.

The first review was prepared by Dr Kathleen Thiessen, PhD, a risk assessment specialist on the NRC panel.

Dr Thiessen's critique has been peer reviewed and endorsed by Dr Hardy Limeback, PhD, the second former NRC panel member, and former Head of Preventive Dentistry, University of Toronto, and Dr James Beck MD, PhD, co-author of *The Case Against Fluoride*, a critically acclaimed contribution to the fluoridation debate.

The second review is by Dr Spedding Micklem, DPhil, co-author of *The Case Against Fluoride*. This critique has been peer reviewed and endorsed by Dr James Beck MD, PhD.

Dr Beck has also authorized publication of the following comment:

> "This report is a clear example of cherry picking, where only select studies that support the 'safe and effective' viewpoint were cited. It is far from a really critical review of the literature."

The third review is regarding tooth decay, and has been prepared by Wellington dentist, Dr Stan Litras, and has been peer reviewed by Dr Hardy Limeback and Dr Bruce Spittle, MB ChB DPM FRANZCP, Managing editor of the international journal *Fluoride.*

Review # 1 Dr Kathleen Thiessen PhD

Oak Ridge Center for Risk Analysis, Inc. NRC Panel member

Peer reviewed and endorsed by: Dr Hardy Limeback, B.Sc., Ph.D., D.D.S. Emeritus Professor, former Head of Preventive Dentistry, University of Toronto. Former president of the Canadian Association for Dental Research. NRC panel member

Dr James Beck MD, PhD co-author of *The Case Against Fluoride*

Comments on the RSNZ/OPMCSA report on "Health effects of water fluoridation: A review of the scientific evidence"

The following comments do not constitute a thorough critique of the RSNZ report. I have primarily tried to give examples of the lack of scientific rigor in the report, including several inconsistencies and inaccuracies contained in the report. Page numbers below refer to the RSNZ report unless otherwise stated.

(1) General comment

This report from the Royal Society of New Zealand and the Office of the Prime Minister's Chief Science Advisor in general falls short of the standards one expects for a "review of the scientific evidence" and instead seems to concentrate on demonstrating a consensus favoring community water fluoridation (CWF). For example, "the scientific consensus confirmed in this review" (p. 5); "Analysis of the peer-reviewed scientific literature reveals a clear consensus on the effectiveness of CWF" (p. 16); "the weight of peer-reviewed evidence supporting the benefits of water fluoridation at the levels used in New Zealand is substantial, and is not considered to be in dispute in the scientific literature" (p. 16); and "while the scientific consensus is that these [cancer, effects on cognitive development of children] are not significant risks" (p. 16).

The review mentions that "the effectiveness of CWF continues to be questioned by a small but vocal minority" (p. 16), but fails to acknowledge that both the safety and efficacy of CWF have been questioned for decades by scientists, physicians, dentists, and other professionals, based on the available evidence. For example, a 1944 editorial in the Journal of the American Dental Association stated that the current "knowledge of the subject certainly does not warrant the introduction of fluorine in community water supplies" and that "the potentialities for harm far outweigh those for good" (JADA 1944). The Director of Laboratories for the utilities department of the City of New York concluded that the "fluoridation of public water supplies is a hazardous procedure, people are bound to get hurt, it remains to find out how many and when" (Nesin 1956). When a former Principal Dental Officer of Auckland, New Zealand, compared decay rates for all children in all communities of the South Island, he found essentially no differences in tooth decay rates with respect to fluoridation status (Colquhoun 1997).

(2) Margin of safety

The report and the cover letter accompanying the report refer in several places to safety or to a margin of safety:

> The "safety margins are such that no subset of the population is at risk because of fluoridation." (Cover letter, p. 2)

> "The fluoride concentrations recommended for CWF have been set based on data from both animal toxicology studies and human epidemiological studies to provide a daily oral exposure that confers maximum benefit without appreciable risk of adverse effects." (pp. 4-5)

> "The amount of fluoride added to water in CWF programmes is set to minimise the risk of this condition [dental fluorosis] while still providing maximum protective benefit against tooth decay." (p. 6)

> "Community water fluoridation (CWF) entails an upward adjustment of the fluoride concentration in fluoride-poor water sources to a level that is considered optimal for dental health, yet broadly safe for the population that drinks the water." (p. 14)

In spite of these mentions of safety or a margin of safety, the report nevertheless indicates that many people exceed the supposedly "safe" levels:

> "In some cases the fluoride intake by these groups [formula-fed infants, young children who are likely to swallow toothpaste] can approach or exceed the currently recommended conservative upper intake level." (p. 6)

> ". . . there is a narrow range between optimal dental health effectiveness and a risk of mild dental fluorosis." (p. 10)

> Reconstituting infant formula with fluoridated water "can provide infants with fluoride at levels approaching or exceeding the recommended upper level for daily intake." (p. 25)

> ". . . infants who are exclusively fed formula made with water fluoridated at 1.0 mg/L will thus regularly exceed the current UL for fluoride." (p. 28)

If identifiable parts of the population predictably exceed the standards for fluoride intake, then the fluoride concentration in drinking water is too high and should be greatly lowered, so that there indeed exists a margin of safety between intake and a level at which health risks occur, and so that all subsets of the population are adequately protected.

(3) Adequacy of the standards for fluoride intake

In principle, the fluoride concentrations are set (in part) with respect to a demonstrated "safe" concentration. For New Zealand, this concentration is referred to as a "tolerable daily intake" (TDI), defined as "a daily oral exposure to the human population (including sensitive groups) that is estimated to be without an appreciable risk of deleterious effects during a lifetime" (p. 18) and which is "determined by applying a safety margin of several orders of magnitude" to a "no observed adverse effect level (NOAEL)" (p. 18).

The TDI appears to be based on the U.S. Environmental Protection Agency's Reference Dose (RfD), defined as "An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime" (EPA 2009). Properly understood, the RfD or TDI should not normally be exceeded by any individual, and any sort of allowable intake or, in this case, concentration in drinking water, should be set so that the RfD or TDI is not exceeded under ordinary circumstances. Individuals, including members of susceptible population subgroups ("sensitive subgroups"), should not normally have exposures in excess of the RfD or TDI.

For fluoride, the U.S. EPA has an existing RfD of 0.06 mg/kg/day (EPA 1989) and has proposed a new RfD of 0.08 mg/kg/day (EPA 2010). Another U.S. government organization, the Agency for Toxic Substances and Disease Registry, has a Minimal Risk Level (MRL, similar in concept to the USEPA's RfD) for fluoride of 0.05 mg/kg/day (ATSDR 2003). New Zealand has an "adequate intake" (AI) value for fluoride of 0.05 mg/kg/day and a "safe upper level of intake" (UL) of 0.1 mg/kg/day (p. 27, Table 2). Thus, New Zealand has set an "adequate" or "optimal" level of fluoride intake at or just below values considered by the U.S. government to be an upper level of "safe," and has set the "safe upper level of intake" above the U.S. values. The UL for older children and adults is based on an intake of 10 mg/day, considered a "NOAEL" for skeletal fluorosis (p. 26). The TDI, which is supposed to be set by "applying a safety margin of several orders of magnitude" to the NOAEL (p. 18), has in fact been set equal to the NOAEL, with no safety factor at all. There is only a factor of 2 between the AI and UL values (0.05 and 0.1 mg/kg/day; p. 27, Table 2). As pointed out above, some identifiable subsets of the population will have fluoride intakes that exceed the UL.

The report ignores entirely the central question of whether EPA's RfD values (old or new) and New Zealand's TDI are adequately protective. EPA's proposed (but not yet official) new RfD of 0.08 mg/kg/day was based on protection of the population from severe dental fluorosis (EPA 2010). However, in order to obtain this value, EPA inappropriately included an assumption of benefit in its risk assessment for fluoride, including the preservation of an intake of 0.05 mg/kg/day as desirable (based on IOM 1997) and exclusion of possible adverse health effects below an intake of 0.07 mg/kg/day (EPA 2010). In other words, EPA had to ignore other, more sensitive, adverse health effects ("known or anticipated adverse health effects"; EPA 2009) and the association of dental fluorosis (all levels) with increased risk of other adverse health effects (e.g., thyroid disease, lowered IQ, and bone fracture; Alarcón-Herrera et al. 2001; Zhao et al. 1996; Li et al. 1995; Lin et al.

1991; Desai et al. 1993; Yang et al. 1994; Jooste et al. 1999; Susheela et al. 2005). A number of adverse health effects can be expected to occur in at least some individuals when estimated average intakes of fluoride are around 0.05 mg/kg/day or higher (NRC 2006; 2009); in other words, a LOAEL for some adverse health effects is lower than EPA's new (or old) RfD, which is supposed to protect the population, including sensitive subgroups, from deleterious effects during a lifetime (EPA 2009; 2011). For persons with iodine deficiency (one example of a sensitive subgroup), average intakes as low as 0.01-0.03 mg/kg/day could produce effects (NRC 2006). Proper derivation of an RfD or TDI would consider these more sensitive endpoints and apply appropriate safety factors to obtain values much lower than those currently considered desirable by the New Zealand government.

(4) Effects of CWF in New Zealand

The RSNZ report states that "No severe form of fluorosis has ever been reported in New Zealand" (p. 6), "The prevalence of fluorosis of aesthetic concern is minimal in New Zealand, and is not different between fluoridated and non-fluoridated communities" (p. 56), and "Water fluoridation in New Zealand has been ongoing since the 1950s, with notable benefits to the oral health of its residents" (p. 55), while offering little documentation. However, the RSNZ has not even mentioned the reports by John Colquhoun, former Principal Dental Officer of Auckland, which report contrary evidence. For example: "When I obtained the decay rates for all children in all the fluoridated and all the nonfluoridated areas in that part of New Zealand [South Island], as well as the decay rates for all children in the recently defluoridated town, they revealed that there are virtually no differences in tooth decay rates related to fluoridation" (italics in the original) and "25 percent of children had dental fluorosis in fluoridated Auckland and around 3 percent had the severer (discolored or pitted) degree of the condition" (Colquhoun 1997).

(5) Carcinogenicity and genotoxicity

The RSNZ report states that "Multiple thorough systematic reviews conducted between 2000 and 2011 all concluded that based on the best available evidence, fluoride (at any level) could *not* be classified as carcinogenic in humans" (pp. 7, 46, italics in the report). The report is inaccurate to say that the U.S. National Research Council "could not" classify fluoride as carcinogenic to humans. While the U.S. National Research Council did not assign fluoride to a specific category of carcinogenicity (i.e., known, probable, or possible), the NRC committee did not consider either “insufficient information” or “clearly not carcinogenic” to be applicable. The committee report (NRC 2006) includes a discussion of how EPA establishes drinking water standards for known, probable, or possible carcinogens; such a discussion would not have been relevant had the committee not considered fluoride to be carcinogenic. The question remains how strongly carcinogenic fluoride is, and under what circumstances. The NRC (2006) specifically discussed the limitations of epidemiologic studies, especially ecologic studies (those in which group, rather than individual, measures of exposure and outcome are used), in detecting small increases in risk—in other words, most of the studies are not sensitive enough to identify small or moderate increases in cancer risk; therefore a

"negative" study does not necessarily mean that there is no risk (see also Cheng et al. 2007). In particular, a "negative" study that does not address a key condition involved in a "positive" finding (e.g., the failure to include age-specific, individual exposure or to separate young and old people in the analysis) cannot be considered evidence of no risk.

The RSNZ report dismisses the Harvard osteosarcoma study (Bassin et al. 2006) on the basis of a letter by Douglass and Joshipura (2006) that contained no actual data. Douglass approved Bassin's dissertation (Bassin 2001), on which her paper was based, and both Douglass and Joshipura were coauthors on an earlier paper by Bassin et al. (2004) describing the exposure analysis used in the study. The dissertation (Bassin 2001) and peer-reviewed paper (Bassin et al. 2006) contain essentially the same results. The key finding reported by Bassin et al. (2006) was an increased risk of osteosarcoma in young males, based on an age-specific analysis of fluoride exposure. Given this finding, studies that do not look at age-specific exposure of young males cannot be said to be negative.

Douglass and Joshipura (2006) mentioned, but did not provide, an analysis of the fluoride content of bone specimens from the osteosarcoma patients and a lack of association between bone fluoride concentration and excess risk of osteosarcoma; however, fluoride concentration in bones of diagnosed patients constitutes a measure of cumulative fluoride exposure which would not necessarily be expected to be correlated with the risk of osteosarcoma. Given that there is a "lag time" of a few years between onset of a cancer and its diagnosis, use of cumulative fluoride exposure until time of diagnosis is potentially misleading, as fluoride exposure during the last several years (during the "lag time" between initiation and diagnosis of a cancer) cannot have contributed to the initiation of a cancer but could have a significant effect on the estimate of cumulative fluoride exposure.

The RSNZ report mentions a later Harvard paper (Kim et al. 2011) which "reported that bone fluoride levels in these samples did not correlate with the occurrence of osteosarcoma" (p. 46). Kim et al. reported no significant difference in bone fluoride levels between cases and controls and no significant association between bone fluoride levels and osteosarcoma risk. The RSNZ report does not mention that Kim et al. (2011) specifically say that "if risk is related to exposures at a specific time in life, rather than total accumulated dose, this metric [bone fluoride content] would not be optimal," thus admitting that they did not address the key finding of Bassin et al. (2006). Comparison of the distributions of bone fluoride concentrations between cases and controls indicates that the ranges are not greatly different; the median was higher for the controls than the cases, which Kim et al. attribute to the older ages of the controls. Given that the median age of the controls is more than twice the median age of the cases (41.3 vs. 17.6), the obvious conclusion is not a lack of association between fluoride exposure and osteosarcoma, but considerably higher average exposure (by about a factor of 2) in cases and controls, in order to reach similar bone fluoride concentrations. Rather than refuting the work of Bassin et al., these findings by Kim et al. support an association between fluoride exposure and osteosarcoma.

In its discussion of animal studies of carcinogenicity (p. 45), the RSNZ report fails to point out that in most, if not all, of these studies, the fluoride exposures started after the age corresponding to the apparent most susceptible age in humans (based on

Bassin et al. 2006), and thus these animal studies may have completely missed the most important exposure period with respect to initiation of the majority of human osteosarcomas.

With respect to genotoxicity (p. 44), the RSNZ should be aware that *in vitro* genotoxic, cytogenetic, or transformational effects (i.e., positive results) have been observed in many studies at fluoride concentrations at or above about 5 mg/L (reviewed by NRC 2009). In addition, a recent paper by Zhang et al. (2009) describes a new testing system for potential carcinogens, based on induction of a DNA-damage response gene in a human cell line. Sodium fluoride tests positive in this system, as do a number of other known carcinogens, representing a variety of genotoxic and nongenotoxic carcinogenic mechanisms. Known noncarcinogens—chemicals not associated with carcinogenicity—did not test positive. For fluoride, a positive effect was seen at a fluoride concentration of about 0.5 mg/L, or a factor of 10 lower than in the other systems. A fluoride concentration of 0.5 mg/L in urine will routinely be exceeded by many people consuming fluoridated water (NRC 2006); for people with substantial fluoride intake, serum fluoride concentrations may also reach or exceed 0.5 mg/L. Acute fluoride exposures (e.g., accidental poisoning, fluoride overfeeds in drinking water systems) have resulted in fluoride concentrations in urine well in excess of 5 mg/L in a number of cases (e.g., Penman et al. 1997; Björnhagen et al. 2003; Vohra et al. 2008). Urine fluoride concentrations can also exceed 5 mg/L if chronic fluoride intake is above about 5-6 mg/day (less than New Zealand's upper level of intake for older children and adults; p. 27, Table 2). At intakes between New Zealand's "adequate" and "upper level" intakes, kidney and bladder cells are probably exposed to fluoride concentrations in the ranges at which genotoxic effects have been reported *in vitro*, especially when the more sensitive system of Zhang et al. (2009) is considered. Based on the results of Zhang et al. (2009), most tissues of the body are potentially at risk if serum fluoride concentrations reach or exceed 0.5 mg/L. In addition, cells in the vicinity of resorption sites in fluoride-containing bone are potentially exposed to very high fluoride concentrations in extracellular fluid (NRC 2006) and thus are also at risk for genotoxic effects.

(6) Neurotoxicity

The RSNZ report is not accurate in its characterization of the Choi et al. (2012) article on effects of fluoride on children's IQ. They indicate that Choi et al. found a "shift of less than one IQ point" (p. 7), and that "the standardised weighted mean difference in IQ scores between "exposed" and reference populations was only -0.45" (p. 49). In fact, the difference is about one- half (-0.45) of a standard deviation, or about 7 IQ points, not one-half of an IQ point. This was clarified in a letter to the journal in March 2013 (Choi et al. 2013); there was also a clarification for nontechnical readers in a September 5, 2012, correction to the original (July 25, 2012) press release from Harvard University.

While many of the articles included in Choi's meta-analysis had reference levels similar to CWF levels, and "high" levels somewhere above that, several studies had "high" levels within the legal limits for fluoride concentrations in drinking water in the U.S. One study had "high" at 0.88 mg/L, quite relevant to CWF. Also, studies

that have "reference" levels similar to or higher than CWF levels can say nothing about the safety of CWF. Rather, for something like neurotoxicity for which there is likely no threshold (the current U.S. assumption for lead exposure, for example), finding that sort of dose response ought to suggest the likelihood of a response at lower (e.g., CWF) levels compared to very low or negligible levels, and the importance of looking for possible effects at lower (CWF) levels is obvious. One extremely important finding by the NRC (2006) and then Choi et al. is the consistency of the effect. Even the one study in Choi's list that did not clearly show lower IQ still showed a tendency in that direction (just not statistically significant), and it certainly did not show clear absence of any effect.

The RSNZ report ignores the fact that Choi et al. (2012) excluded several studies from their meta-analysis because they used individual measures of exposure rather than group exposures-- in other words, some excluded studies might have been of better design than the ones that their meta-analysis could consider. There are also a few studies too recent to have been considered by Choi et al. but that should have been mentioned by the RSNZ report (e.g., Saxena et al. 2012; Seraj et al. 2012; Shivaprakash et al. 2011). While some of the neurotoxicity studies did not address confounders, some did handle them responsibly, a detail not mentioned in the RSNZ report.

The RSNZ report (p. 50) describes as "relatively high quality" a recent paper from New Zealand reporting no evidence for an effect of CWF on IQ (Broadbent et al. 2014). However, the assessment of exposure provided in that paper is inadequate and probably results in comparisons between groups with similar, or at least overlapping, exposures to fluoride. For example, children in the non-CWF group who received fluoride tablets probably had similar exposures to children in the CWF group. Broadbent et al. report that breastfeeding was associated with higher IQs but fail to point out that this effect was larger for CWF areas than non-CWF areas. (Fluoride concentrations in breast milk are quite low, regardless of the mother's fluoride intake.) Broadbent et al. defined breastfeeding as lasting at least 4 weeks, suggesting that further analysis, including duration of breastfeeding, might show a larger effect.

Both Broadbent et al. and the RSNZ report inaccurately state that no plausible mechanism exists for an effect of fluoride on IQ. The fact that no mechanism has been established reflects the absence of research effort, not the absence of a mechanism. One possible mechanism is reduction of maternal and/or infant thyroid function (NRC 2006). Others involve damage to the developing brain or disrupted neurochemistry (e.g., Blaylock and Strunecka 2009). Several studies have shown changes in brain chemistry in fetuses due to maternal fluoride exposures (Dong et al. 1997; Du et al. 2008; He et al. 2008; Yu et al. 2000; 2008).

(7) Significance of animal studies

The RSNZ report dismisses many of the animal studies as involving greatly higher fluoride intakes (or fluoride concentrations in drinking water) than those experienced by people with CWF (pp. 45, 49). However, animals require much higher exposures (5-20 times higher, or more; see NRC 2006; 2009) than

humans to achieve the same effects or similar fluoride concentrations in bone or serum. In other words, humans are considerably more sensitive to fluoride than are most animal species that have been studied. The animal studies cannot so easily be dismissed. This difference in sensitivity should have been discussed in the report.

(8) Endocrine effects

The RSNZ report mentions the extensive review of "potential fluoride effects on endocrine organs and hormones" by the U.S. National Research Council (p. 51), but they fail to mention that the NRC's report concluded that "fluoride affects normal endocrine function or response" and "Fluoride is therefore an endocrine disruptor" (NRC 2006). The RSNZ mentions a paper on childhood goitre in South Africa by Jooste et al. (1999) as included in the York review (p. 52). They have not mentioned the NRC's discussion of the same paper, specifically that the town with the lowest prevalence of goitre also had the lowest prevalence of "undernutrition." When that town is excluded from the analysis, a clear dose response is observed between goitre prevalence and fluoride concentration in drinking water.

(9) Monitoring of fluoride concentrations in water

The RSNZ indicates that "fluoridated drinking water supplies must be sampled at least weekly" (p. 24). It should be mentioned that the American Water Works Association recommends at least once per day (Lauer and Rubel 2004). The AWWA also mention the advantages of continuous monitors, in particular, having one equipped with an alarm to alert operators to a malfunction. Fluoride overfeeds do occur and can cause illness and even death (e.g., Gessner et al. 1994; Penman et al. 1997).

(10) CWF recommendations in the U.S.

The RSNZ report indicates that "optimally fluoridated" drinking water in the U.S. is now 0.7 mg/L (p. 54). However, while the U.S. Department of Health and Human Services proposed a new recommendation of 0.7 mg/L instead of the existing temperature-based recommendation of 0.7-1.2 mg/L (Federal Register 2011), this is not yet anything but a "proposed" new recommendation. As of this date (September 2014), this proposed recommendation has not become an official recommendation, and to the best of my knowledge has not had wide implementation in the U.S.

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Review # 2 Dr H.S. Micklem, DPhil

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NZ Report – Health effects of water fluoridation: A review of the scientific evidence

Critical comments

General

It appears that this report, or at least its summary, is written with a largely lay readership in mind. As such, it calls for particularly high standards of accuracy and integrity, so that readers may not be accidentally misled. The report measures up to these ideals with limited success.

Executive summary

In the executive summary the authors adopt a patronizing tone and a noticeably biased approach. For example, reviews and papers favorable to fluoridation are routinely described as "thorough" or "careful". This sounds more like propaganda than science and suggests that readers should look very critically at the report's conclusions.

Scope

These comments are confined to questions of risk, specifically those related to dental fluorosis, lead concentrations, IQ, osteosarcoma and renal function, where the report appears unjustifiably complacent.

Attitudes to risk

The Report is not alone in exhibiting an attitude to risk that baffles and angers many opponents of fluoridation. Proponents appear, or affect, not to understand the nature of this reaction, denouncing it as ignorant, emotional etc. Proponents tend to take the view that 'credible scientific evidence' does not support the view that fluoride poses any risk to consumers of fluoridated water; to be credible the evidence needs to be direct and incontrovertible. They think this is enough, and it might be enough if they were dealing with a prescription drug, where a sick patient might readily accept some risk of harm, and where considerable energy goes into identifying and recording side-effects. But as soon as something is put into the water supply, the game changes: millions are treated unnecessarily, there is little control over dose, no allowance for individual sensitivity, no individual consent and (despite frequent assertions to the

contrary) little monitoring of safety or attention to claimed ill effects. Opponents of fluoridation recognize this and view fluoridation from a toxicological angle, attaching some weight to animal studies and studies on populations drinking naturally fluoride-rich waters, and evaluating margins of safety. Proponents either do not realize that the game has changed or try to gloss over it. In either case, they adopt a default position that fluoridation is safe and are at pains, as in this report, to downplay any evidence to the contrary.

Dental fluorosis [Section 3.3] – an example of game-change denial

The report's discussion of fluorosis [Section 3.3] provides an example of what we might call game-change denial. The incidence of moderate fluorosis (2%) is described as 'rare' in 8 year olds. But note that side-effects of a drug are often listed as 'common' if they occur in more than 1% of cases. As the report goes on to show, moderate fluorosis is widely considered to be aesthetically unacceptable. That implies some 4-5 thousand aesthetically disadvantaged children and young adults in a largely fluoridated city such as Auckland[1]. A further 8000 must have 'mild' fluorosis, which many, including the York review [report refs 89, 90] and the British Fluoridation Society (BFS)[2] consider to be of aesthetic concern.

The Report's authors are on morally, and also economically, shaky ground here, even if fluorosis is viewed merely as a cosmetic, not a health problem. They do not even mention the cost of veneers (up to $1750 per tooth)[3] and other treatment in their cost-benefit discussion [Section 3.2.5]. Perhaps aware of this, they attempt to exonerate fluoridated water from causing fluorosis by citing the NZ Oral Health Report (2010)[4] to claim that the incidence of moderate fluorosis in 8-30 year olds is no higher in fluoridated than in non-fluoridated areas. However, this was a relatively small survey and, as the 2009 report acknowledges, experience elsewhere has generally been that water fluoridation is associated with an increased incidence of fluorosis, including the grades of aesthetic concern (British Fluoridation Society, op.cit). The BFS suggests that the unusually high reported incidence of fluorosis in non-fluoridated areas of New Zealand may be attributable to supplement use. In other words, the suggestion that water fluoridation does not contribute to fluorosis is probably invalid. Moreover, Kanagaratnam et al[5] reported that 9 year old children who lived continuously in fluoridated areas of Auckland were 4.17 times as likely to have diffuse opacities as children who lived continuously in non-fluoridated areas.

In summary, some thousands of young people in New Zealand are involuntarily paying the price for an excessive load of fluoride that was imposed on them, largely via the public water supply, in early childhood. The NZ Government must surely have done these sums, but the report does not mention them.

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[1] For source and calculations see Appendix
[2] http://www.bfsweb.org/onemillion/05 One in a Million - Dental Fluorosis.pdf] Tables 1-3
[3] See Appendix
[4] https://www.health.govt.nz/system/files/documents/publications/our-oral-health-2010.pdf
[5] Kanagaratnam S1, Schluter P, Durward C, Mahood R, Mackay T Community Dent Oral Epidemiol. 2009 Jun;37(3):250-9. doi: 10.1111/j.1600-0528.2009.00465.x. Epub 2009 Mar 19.

[Section 3.3.5] pg 43. *"...adolescents actually preferred the whiteness associated with mild fluorosis [143]*". This is deceptive. What the cited paper actually states is that the subjects liked the appearance of a *complete set of artificially white teeth*. They did not like the whiteness associated with fluorosis: even TF1 and TF2 (very mildly fluorosed) teeth were rated lower than natural-coloured normal teeth.

Lead [Section 2.2.1]

Page 23. Reference is made to a report by Jackson et al [27]. Anyone unfamiliar with this contracted report would be hard put to trace it from the inadequate reference details given, even if two spelling mistakes were corrected. It can, however, be downloaded from the British Fluoridation Society's web site. Note that Jackson's 'determination' that "HFA used to fluoridate water is effectively 100% dissociated to form fluoride ion under water treatment conditions" was based on theoretical modeling and took no account of any real life data.

The following paragraph bizarrely suggests that a paper by Urbansky and Schock [33] published in 2000 criticized and fatally discredited papers published by Coplan et al and Maas et al seven years later in 2007.

Urbansky and Schock's paper actually referred to an earlier paper by Masters and Coplan (1999). Coplan et al (2007) [31] was a rebuttal of that with additional data showing that consumers of fluoridated water, particularly from poor communities, had raised concentrations of lead in their blood. The paper by Maas et al (2007) [32] described laboratory experiments showing that fluoride, added as NaF and particularly H2SiF6, enhanced the tendency of chlorine and chloramine to leach lead from brass plumbing components, providing a possible partial explanation for Coplan et al's findings. Urbansky's comments had no relevance to it. A more recent study in rats[6] showed that fluoride enhanced the uptake of lead into blood and bones, another possible factor in Coplan's results.

The studies by Coplan and Maas do not appear to have been challenged. Independent confirmation is needed, but meanwhile lead exposure is an important issue and their conclusions should be taken seriously. The superficial treatment of the subject by SCHER [34], on which the NZ report leans, did not even mention the Maas study. Whatever the truth of the matter, the NZ report is factually misleading here and does not justify its implied conclusion that fluoridation poses no added risk from lead.

Neurotoxicity and IQ [Section 4.4)

This is a superficial and unconvincing attempt to demolish the case that water fluoridation poses a developmental risk to human intelligence. I shall not discuss it in any detail, but make one general point. In most areas of science, the existence of so many studies almost all saying the same (important) thing would be treated with some attention and respect, even if individually most were not strong. The NZ report fails to

[6] Sawan RM, Leite GA, Saraiva MC, Barbosa F Jr, Tanus-Santos JE, Gerlach RF. Toxicology. 2010 Apr 30;271(1-2):21-6. doi: 10.1016/j.tox.2010.02.002. Epub 2010 Feb 25.

do that and even manages to misunderstand and mis-state the conclusions of a systematic review and meta-analysis of many of the studies by Choi et al. The authors made the rather elementary mistake of supposing that a standardized mean difference of 0.45 meant a reduction of less than half an IQ point and was therefore arguably negligible, whereas the actual reduction was almost 7 IQ points, and therefore far from negligible. It is hard to imagine how this mistake could have been made by anyone who had actually read the papers that are disparaged so casually.
Against all these studies the report sets a single inconclusive New Zealand study [176], and concludes (page 7) "that on the available evidence there is no appreciable effect on cognition arising from CWF". We must hope that the authors are right, but the question remains wide open. The issue is hardly trivial and the public is ill-served by such a poorly informed judgement.

Osteosarcoma [Section 4.2.3]

In discussing the paper by Bassin et al 2006 [159], the authors do not appear to understand what they are talking about. They have some excuse since the cited letter from Bassin's director [160] seemed designed to cause uncertainty and confusion and turned out to be factually incorrect. The letter claimed that a larger set of data failed to support Bassin's findings and promised a further paper "currently being prepared for publication". Nothing appeared for 5 years until the paper by Kim et al [161] was published. It did little to contradict Bassin's results, being relatively small, poorly controlled and, crucially, as Kim et al admitted, having no bearing on fluoride exposure during the critical age range identified by Bassin. Since then, as the Report states, Blakey et al [164] studied all (2566) osteosarcoma cases registered in the UK over a 25 year period in relation to the fluoridation status of their closely defined area of residence at the time of diagnosis. A range of potential confounders was taken into account. No association of osteosarcoma with the fluoride content of water was found. Unfortunately, however, no firm conclusions can be drawn from this for reasons that the authors acknowledge: the fluoride connection was only assessed on the place of residence at the time of diagnosis and no residential histories were available, nor any information about fluoride consumption during childhood. In addition, fluoride concentrations in artificially fluoridated waters were based only on information from 2004-06 and varied considerably with many being below 0.7mg/l. Comparable problems beset the other recent ecological studies cited by the Report. The fluoride/osteosarcoma question therefore remains unsettled; nothing has emerged that convincingly contradicts the conclusion of Bassin et al that boys go through a sensitive phase in childhood when exposure to fluoride in drinking water is associated with an increased frequency of osteosarcoma several years later.

Cardiovascular and renal effects [Section 4.5.3]

The Report raises some interesting points related to kidney disease and its cardiovascular consequences. We learn (page 9) that chronic kidney disease (CKD) is relatively common in New Zealand and that sufferers may be at risk of excess fluoride. "However," the Report states, "to date no adverse effects of CWF exposure in people with impaired kidney function have been documented." One meets this kind of statement rather often in reports and reviews. It can have three meanings: 1.

numerous studies have been reported, all considered negative; 2. no studies have been performed; 3. something in between. Critical readers tend to assume the second interpretation. Too often one is left guessing, but later this Report does eliminate interpretation 1, stating "However, the scarcity of data indicates that further studies are required." (page 59). In other words, we don't know.

With this in mind, we may turn to page 52 where the Report summarizes an interesting recent paper by Martin-Pardillos and colleagues [200]. The paper shows that partially nephrectomized rats (a model for human CKD) having 1.5 or 15mg/l fluoride in their drinking water show accelerated medial calcification of vascular smooth muscle. The authors of the paper suggest on the basis of these results that fluoridated water may pose a cardiovascular risk for CKD sufferers. The Report rightly states that the results need to be confirmed. The interesting question is, what should happen meanwhile? I suspect that most opponents of fluoridation would call for CKD sufferers to be warned to avoid tap water. Possibly the NZ health authorities have done so. If not, will they follow the "no adverse effects" message on page 9, or will they adopt a more precautionary approach to protect this group of patients from possible harm? And will further study of the matter be given any degree of priority?

END

Appendix – calculation of dental fluorosis incidence in Auckland (approximate numbers)

Auckland: total population = 1.4 million
- of which >95% fluoridated = 1.3 million
- of which conservatively 20% in 8-30 age group = 272,000
- of which 1.7% have 'moderate' dental fluorosis* = 4600
  and 3.0% have 'mild' fluorosis* = 8100

*Source: Our Oral Health: Key findings of the 2009 New Zealand Oral Health Survey. Ministry of Health, Wellington, 2010. p 171, Table 92.

Cost of veneers $650 - $1750 per tooth according to quality
http://www.clarencetam.co.nz/about_the_practice/standard_pricing.aspx

Review # 3 Dr Stan Litras BDS BSc

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Internationally peer reviewed by: Dr Hardy Limeback, B.Sc., Ph.D., D.D.S. Emeritus Professor, former Head of Preventive Dentistry, University of Toronto. Former president of the Canadian Association for Dental Research. NRC panel member

NZ Report – Health effects of water fluoridation: A review of the scientific evidence

Introduction

The findings and recommendations in the executive summary in my opinion overstate the benefits of water fluoridation and understate the potential risk to general health of individuals.

No effort was made in the selection of panel members to control for bias (1). The panel apparently simply reviewed a draft report undertaken over just a few months by a single literature researcher, Anne Bardsley, with unspecified previous familiarity of the subject, who was charged with selection and interpretation of papers. (1,2)

A poignant communication from Sir Skegg to Sir Gluckman, lamenting the number of scientists who were approached to be on the review panel, but declined to participate, reads:

"....I can understand why any reputable scientist would be reluctant to put their name to a report if they have not had the time to have a first-hand look." (2)

According to the University of York Centre for Reviews and Dissemination:

"Systematic reviews differ from other types of review in that they adhere to a strict scientific design in order to make them more comprehensive, to minimise the chance of bias, and so ensure their reliability. Rather than reflecting the views of the authors or being based on only a (possibly biased) selection of the published literature, they contain a comprehensive summary of the available evidence."

This cannot in good faith be described as a robust and thorough review of fluoridation science by experts, as it fails to address conflicting evidence as detailed in this critique. In my opinion it fails to meet its requirement of advising local government leaders as to the state of the science on fluoridation.

Similar reviews aimed at advising city councils concerned about the health risks posed to their communities, but which are conducted by a balanced and unbiased review panel, have reached the opposite conclusions and have resulted in rejection of fluoridation in their communities. (3)

Comments on the Understated Health Risks:

This report identified large sections of the population of NZ who exceed the toxic limits for fluoride ingestion, including bottle fed infants, children under 8 years old, people with impaired kidney function, people who drink a lot of water, and people who have high fluoride intake already from other sources such as diet and toothpaste.

Despite this, they conclude that:

"the safety margins are such that no subset of the population is at risk because of fluoridation"

---

a sweeping statement, which is totally unsupported by their own discussion, let alone the science.

The reviewers appear to ignore the risk of fluoride overdose at the individual level of ingestion (dose) as opposed to the fluoride levels in the water at the population level (concentration), despite WHO recommendations stressing that countries who deliberately fluoridate water supplies need to monitor people's total fluoride ingestion at the individual level in case of overdose. (4)

I will leave critiques of the risk to health sections of this review to scientists who are the true experts in the fields.

My comments will focus on the gross over statement of the purported benefits of fluoridation in our society, New Zealand, 2014.

Quotes from the New Zealand report are highlighted in red italics. Quotes from other sources are highlighted in green italics.

Comments on: "Evidence for benefits of water fluoridation"

"Analysis of evidence from a large number of epidemiological studies and thorough systematic reviews has confirmed a beneficial effect of CWF on oral health throughout the lifespan." PAGE 8

The observation in 1906 USA that Italian immigrants from a high fluoride region had discoloured and mottled teeth (subsequently called Dental Fluorosis), but no tooth decay, led to the assumption that fluoride reduces caries.

It was considered useful by public health authorities to put fluoride in the water supplies of populations en masse, based on the erroneous theory that fluoride incorporated into children's developing tooth enamel would make teeth more resistant to decay.

"ingested fluoride is incorporated into the developing enamel, making the teeth more resistant to decay." PAGE 7

It has been widely accepted since the 1990s that any effect on tooth decay from swallowing fluoride is insignificant or non-existent. To quote:

CDC 1999: "the effect of Fluoride is topical " (5)

J Featherstone 1999: "the systemic effect is, unfortunately, insignificant" (6)

Evidence given in support of water fluoridation has generally been weak, often suffering from lack of controls for a variety of confounding factors and research bias. (7)
The "elephant in the room" is that while decay rates fell in areas where fluoridation was implemented, it also fell in areas that weren't, often at a faster rate. (8)
Furthermore, cross sectional studies generally make comparisons on the basis of age and do not identify how long the teeth have actually been present in the mouth for each group.

There is evidence that teeth erupt later in areas exposed to water fluoridation (due to hardening of bone over the erupting tooth and hormonal effects), which of course means less time exposed to plaque acid and less decay at a particular age. (7)

If the actual time teeth have been present in the mouth is accounted for, the minor differences seen in cross sectional studies comparing fluoridated and unfluoridated communities disappear. (9)

Ministry of Health figures recorded every year in 5 year olds and year 8s (12-13 year olds) consistently show minimal or no differences between fluoridated and non-fluoridated areas of NZ. (10)

The highly acclaimed York Review found only 26 studies after 60 years of research which met even the minimum scientific standards to be considered reliable. Most of these were decades old and prior to the widespread use of fluoridated toothpaste.

Nevertheless their conclusion was that water fluoridation during that period did reduce the incidence of tooth decay, but only by about 14%, much less than the 60% quoted publicly by some health agencies and organizations.

Importantly, the York Review noted that the variation of tooth eruption times between fluoridated and unfluoridated areas was not taken into account. (7)

Furthermore, since the widespread use of fluoridated toothpaste from the late 1980s onwards, any perceived benefit from water fluoridation has become minimal at the community level.

This was the conclusion of the European review of fluoridation, Scientific Committee on Health and Environmental Risks 2011 (11):

"In the 1970s, fluoridation of community drinking water, aimed at a particular section of the population, namely children, was a crude but useful public health measure of systemic fluoride treatment. However, the caries preventative effect of systemic fluoride treatment is rather poor (Ismael and Hasson 2008)".
SCHER 2011

"No obvious advantage appears in favour of water fluoridation compared with topical prevention. The effect of continued systemic exposure of fluoride from whatever source is questionable once the permanent teeth have erupted."
SCHER 2011

”This suggests that water fluoridation plays a relatively minor role in the improvement of dental health." SCHER 2011 (11)

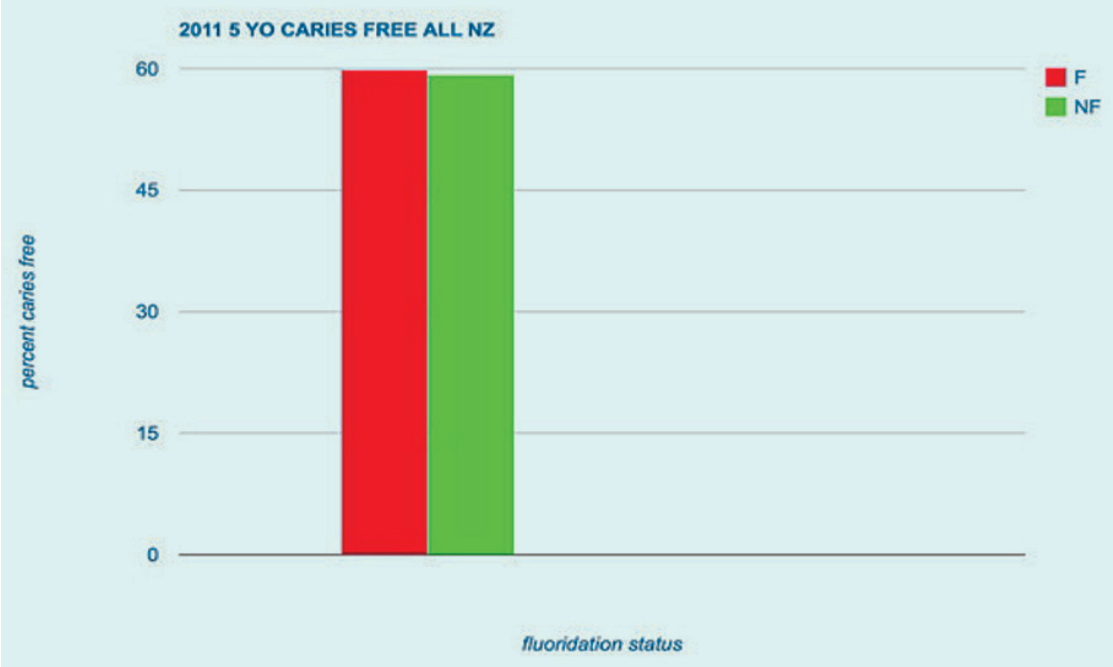
"SCHER agrees that topical application of fluoride is most effective in preventing tooth decay. Topical fluoride sustains the fluoride levels in the oral cavity and helps to prevent caries, with reduced systemic availability. The efficacy of population based policies, e.g. drinking water, milk or salt fluoridation, as regards the reduction of oral health social disparities, remains insufficiently substantiated." (12)

Just how minor a role water fluoridation plays at the community level is evident from the following graphs made from the www.health.govt website. (10)

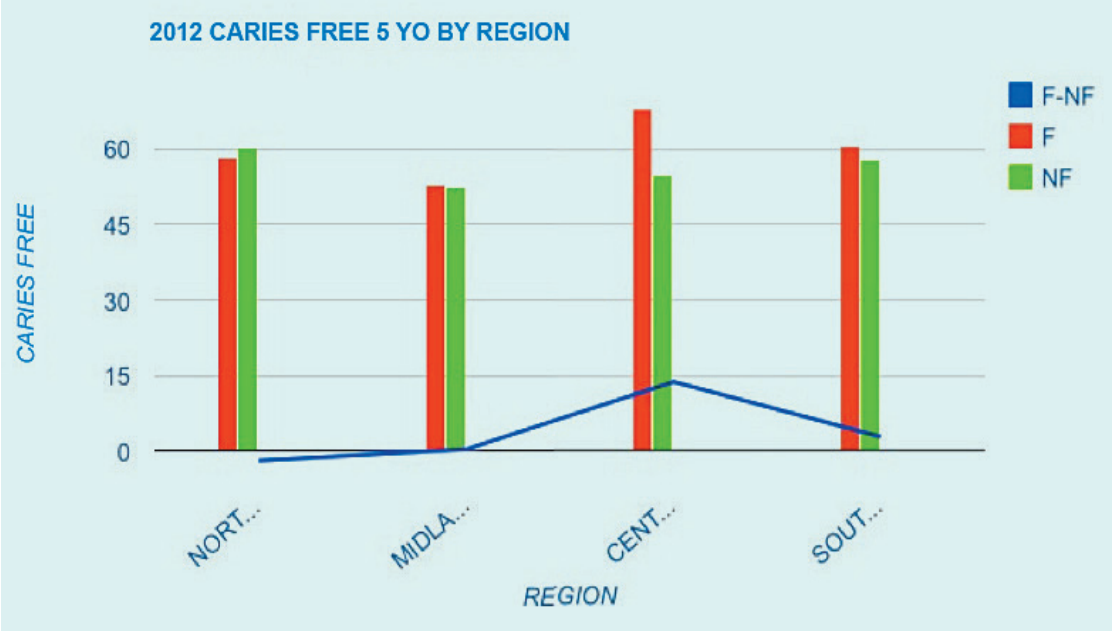
Number 44,653 5YO 44,659 Y8	2011 FLUORIDATED	2011 NOT FLUORIDATED	DIFFERENCE
5YO% CARIES FREE	59.91%	59.18%	0.73%
Y8 % CARIES FREE	55.17%	51.79%	3.38%
5YO dmft	1.77	1.9	0.13
Y8 DMFT	1.14	1.37	0.23

This 2011 real life child data shows that the difference in decay incidence and severity between children living in fluoridated and unfluoridated areas in NZ is insignificant.

Data sourced from: www.health.govt



Small apparent differences could be accounted for by other factors such as delayed eruption of teeth in fluoridated communities, therefore less time in the mouth exposed to plaque acids, ethnic distribution and urban/rural differences.



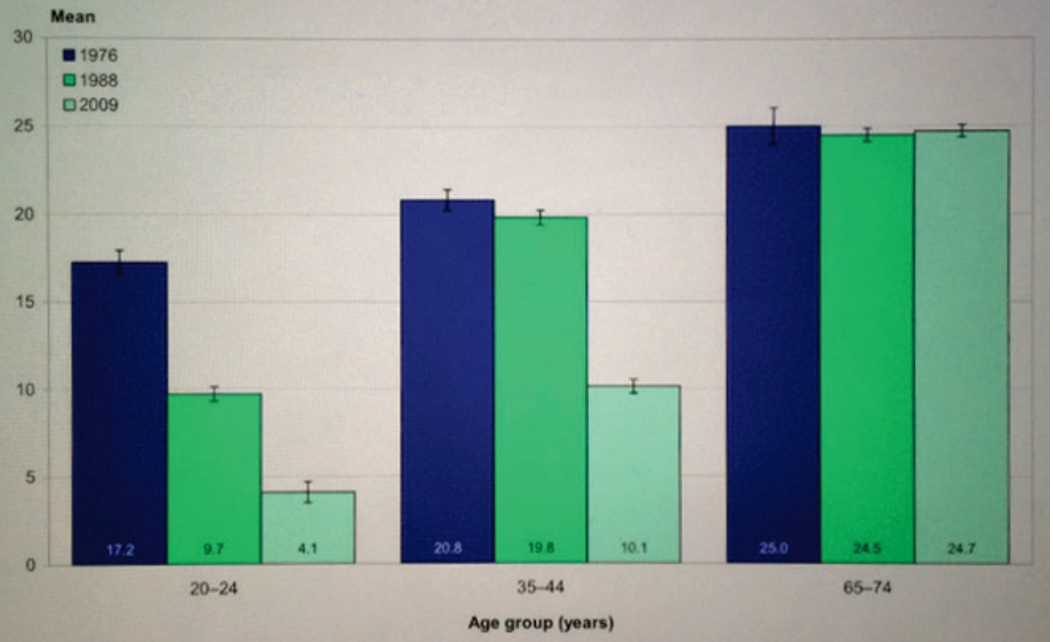
Data from the NZOHS 2010 do not support statements of a lifetime benefit, indicating that the action of fluoride is simply to delay the decay. (13)

The following figures, from the NZOHS, look at the senior adult age group and show that the lifetime decay experience is the same, regardless of the amount of time spent in a fluoridated community.

NZOHS 2010 data indicate that fluoridation only delays tooth decay; the lifetime experience is the same in the older age group irrespective of fluoridation history. No difference was found in DMFT in the 65-74 age group after water fluoridation was implemented in NZ.

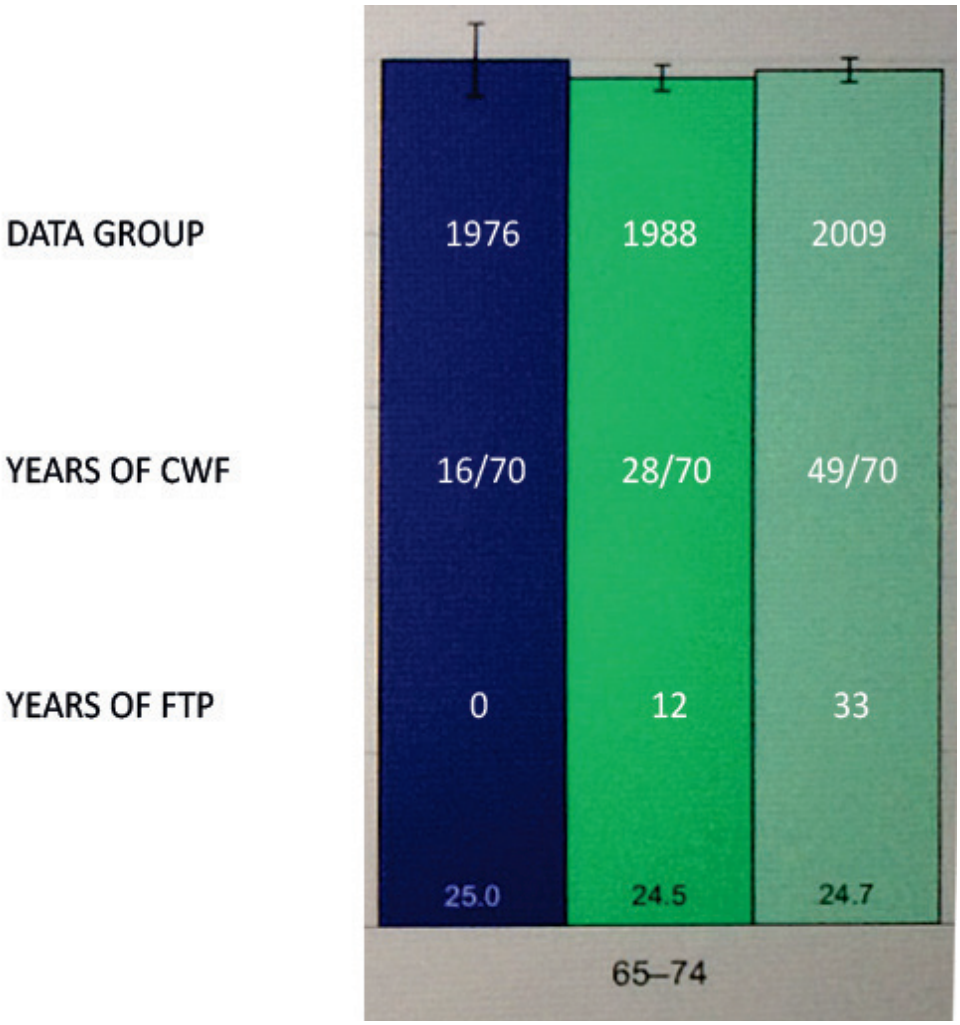
There was no significant change in the mean DMFT score in 65–74-year-old dentate adults from 1976 to 2009.

Figure 53: Mean number of decayed, missing or filled teeth (DMFT) per person, among dentate adults in 1976, 1988 and 2009, by age group (unadjusted mean)



Sources: 1976 Survey of Adult Oral Health, 1988 WHO Study of Oral Health Outcomes (NZ), 2009 New Zealand Oral Health Survey

An analysis of the 65-74 age group by proportion of life since fluoridation (CWF) was introduced and since fluoride toothpaste (FTP) was introduced:



Although we can see apparent benefit in younger age groups, we can see no lifetime benefit from CWF or FTP on this NZOHS data. 65-74 year olds in 2009 had the same lifetime decay experience as they did in 1976, despite having the benefit of fluoridated toothpaste and water fluoridation being available for over 30 years longer for half the population.

The York Review found there was no weight of evidence to support benefit in adults or in low SES groups, or increase of decay in cessation studies. (7)

"In New Zealand, significant differences in decay rates between fluoridated and non-fluoridated communities continue to exist, despite the fact that the majority of people use fluoride toothpastes.
These data come from multiple studies across different regions of the country conducted over the last 15 years, as well as from a national survey of the oral health status of New Zealanders conducted in 2009." PAGE 8

The reliability of NZ studies unfortunately suffers heavily from the weaknesses identified in the York Review in many cases. The studies cited in the review to support the efficacy of fluoridation are no exception.

1. The Hastings Experiment

This was a very questionable pilot study, which launched fluoridation in NZ in the early 1960s.

Designed to compare the decay rates in the communities of Napier (unfluoridated control) and Hastings (artificial fluoride added to water experimentally), it apparently became a case of adjusting the facts to fit the theory.

Decay was dropping faster in Napier and not in Hastings, so the researchers cut Napier out of the study.

It has been accused of being fraudulent, because not only was the methodology changed during the course of the study, the data recording protocols were changed, as were the MoH directives to school dental nurses criteria of when a tooth needed to be filled, and this was not accounted for by the researchers. (14,15)

Fluoridation defenders unsurprisingly no longer refer to this study, yet for decades it was taught in dental schools as the definitive justification for water fluoridation. Full critique attached as appendix 1.

2. Lee & Dennison 2004

These long standing DHB employees cherry picked two cities from the 1996 data to compare. Overall 1998 data show that had other areas been selected, the results would have been the opposite.

Even so, the absolute differences found were minimal, and again the study was seriously flawed by uncontrolled confounding factors and research bias. (16)
Full critique appendix 2.

3. Southland Study: Mackay and Thomson 2005

This review claims it shows children in fluoridated areas had about half the decay rates of those in unfluoridated areas.

In fact this was a very weak study, whose conclusions are not supported by the results presented. Children were examined in a mobile clinic and the teeth were not cleaned or dried for the examination.

Due to the small numbers, results were not statistically significant, and many confounding factors were uncontrolled.

In fact, the authors admit in the discussion that "there were no significant differences in deciduous caries prevalence or severity (or in the permanent caries prevalence) by socio-demographic characteristics or length of residence in fluoridated areas." (17)

4. 2010 NZ Oral Health Survey

This survey was a "snapshot in time" of New Zealander's oral health status in 2009, and was not designed as a scientific study on fluoridation. The writers make a number of disclaimers to this effect; yet still draw conclusions from data which is not statistically significant. (13)
Full critique appendix 3

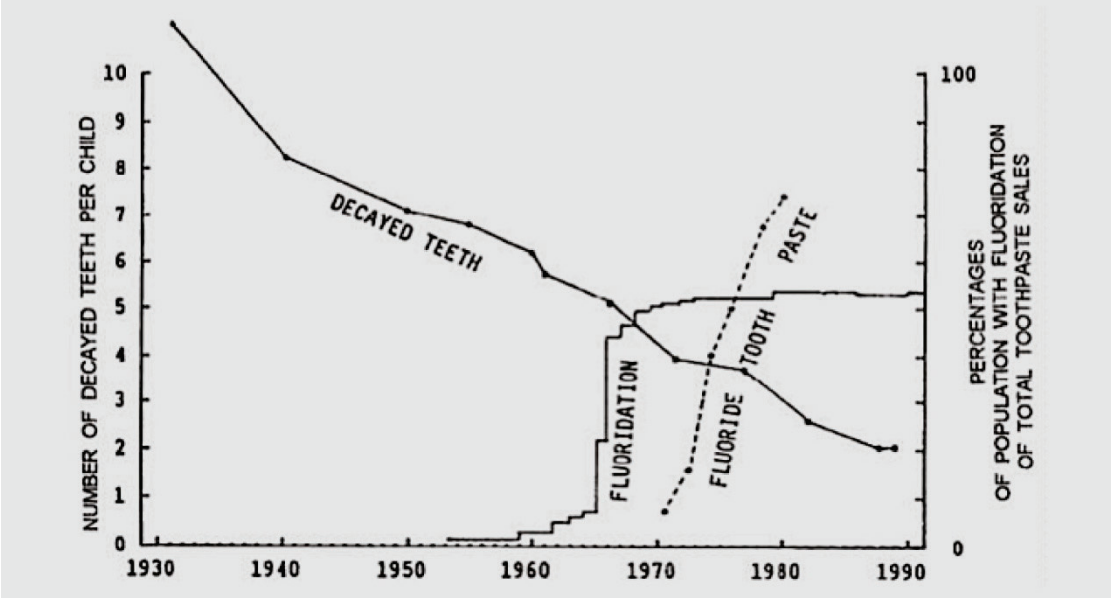
Evidence That Water Fluoridation is Ineffective:

There have been numerous NZ and overseas-published studies which indicate no significant differences in decay rates between fluoridated and unfluoridated areas.

These were overlooked in the present review.

1. New Zealand Studies:

Dr. John Colquhoun, a long serving Principal Dental Officer for the Auckland Health Board, was a leading advocate for water fluoridation for about 20 years.
In 1981 he changed his mind after he realized the data collected by his service proved that water fluoridation had no particular effect on tooth decay in NZ. He showed that the decay rate in NZ was declining before fluoride was introduced, and fluoridation had no apparent impact on this rate.



1. Graph of decline in NZ decay rate prior to fluoridation. Tooth decay was falling in NZ at the same rate even before fluoridation was in widespread use. Colquhoun J, (1984) New evidence on fluoridation. (18)

Dr. Betty de Liefde, who followed John Colquhoun as principal dental officer for the Health Department, made similar observations, and published a report in the NZDJ in 1989 concluding:

"the caries reducing effects of community water fluoridation are clinically insignificant" (19)

In an Auckland study comparing 9-year-old children attending schools in fluoridated and unfluoridated areas, Karaganatnam et al (2009) state:

"no significant relationship was found between residential fluoridation history and dental caries in the permanent dentition" Page 257 (20)

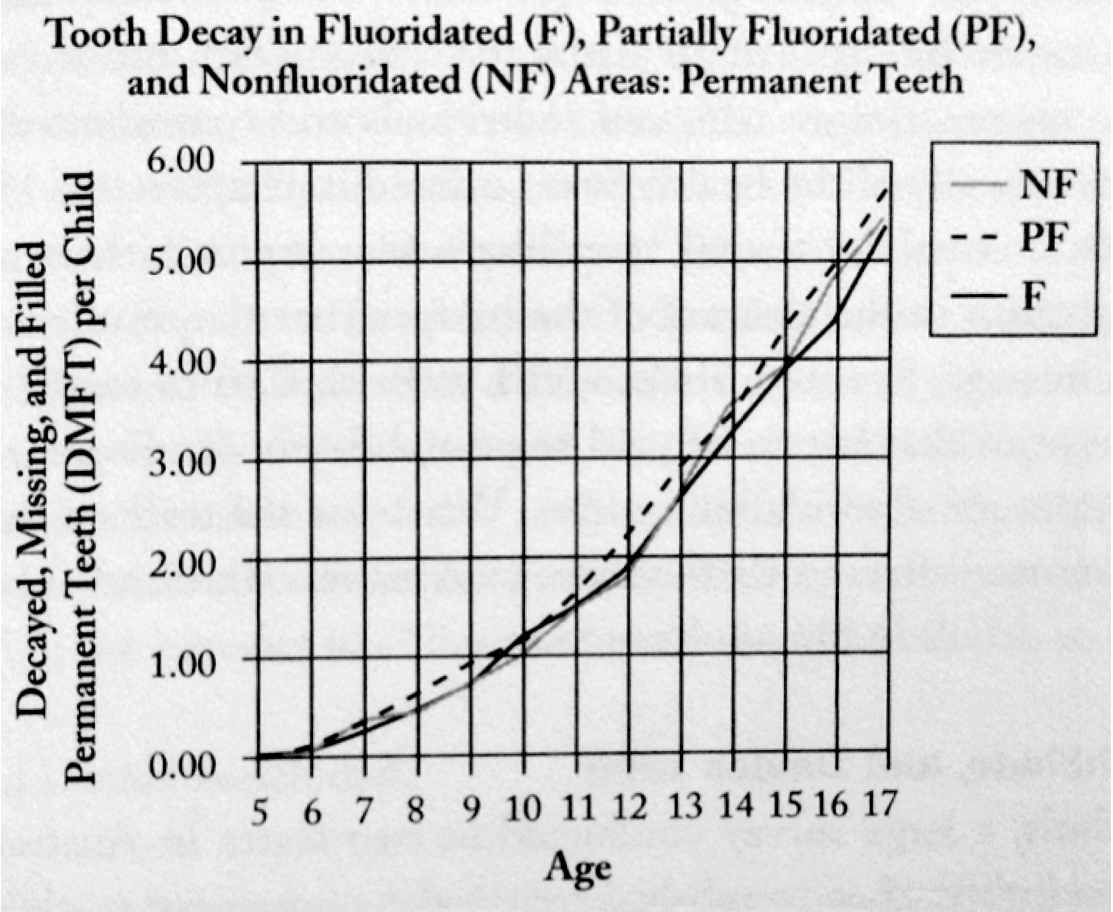
In their 2005 paper "Enamel defects and dental caries among Southland children", Thomson (a panel member of this present review) et al report:

"There were no significant differences in deciduous caries prevalence or severity (or in permanent caries prevalence) by sociodemographic characteristics or length of residence in fluoridated areas" Page 39

This is, in fact, what their research showed, however they oddly concluded that decay rates were halved by CWF. (17)

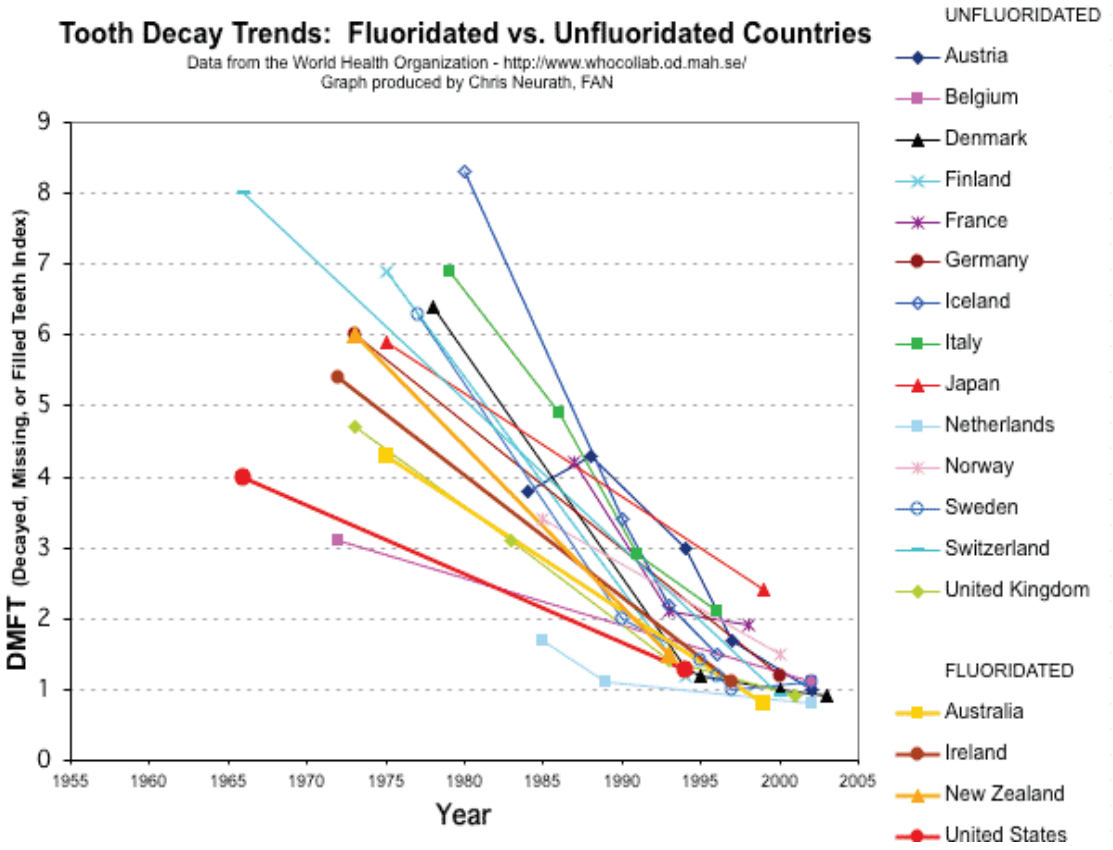
2. Overseas Studies:

The largest USA study also shows no difference in decay rates with or without water fluoridation.

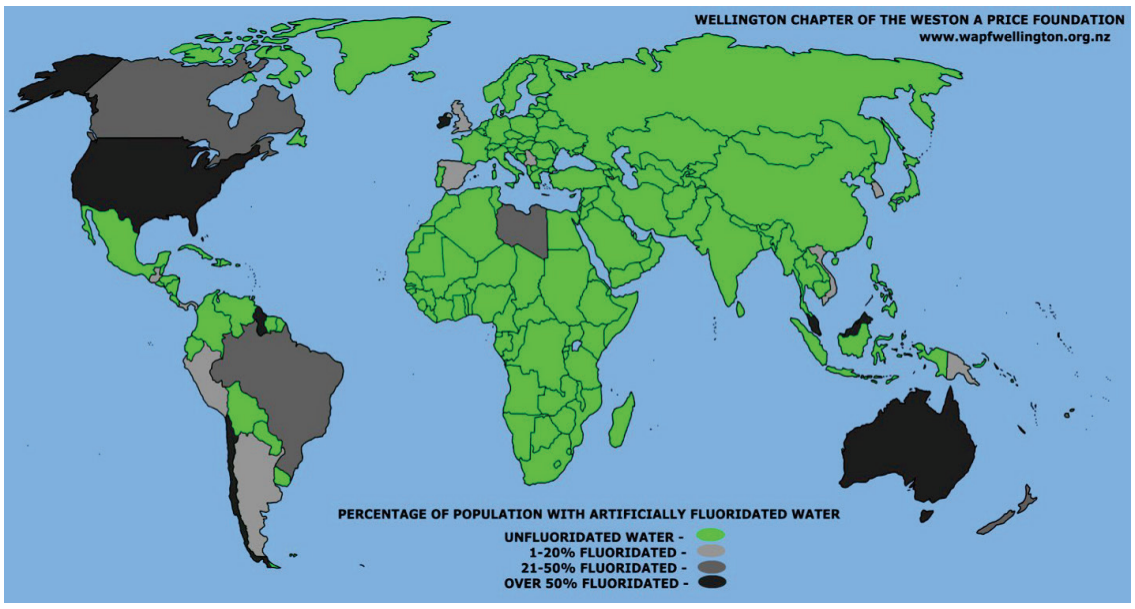


JA Yiamouyiannis water fluoridation and tooth decay: results from the 1986/87 National Survey of US schoolchildren (21)

Globally, fluoridation is seen to make no difference to reduced decay rates, there being no difference between the few countries which use artificial fluoridation, and those that don't. (8,7)



It must be noted that only a handful of countries fluoridate their water supplies, and fewer still fluoridate more than 10% of their population. More than half of all artificially fluoridated people live in the USA.



"Various studies indicate that CWF has an additive effect over and above that of fluoride toothpaste and other sources of fluoride that are now in common use." Page 9

The review appears to be referring to a few weak studies to imply benefit, rather than the weight of evidence. In contrast, the more robust SCHER report states:

"No obvious advantage appears in favour of water fluoridation compared with topical prevention. The effect of continued systemic exposure of fluoridation from whatever source is questionable once the permanent teeth have erupted" SCHER 2011 (11)

An understanding of the mechanism by which fluoride is thought to reduce (or delay) tooth decay is a crucial factor, and the NZ report shows some confusion about this.

"It is now widely accepted that the action of fluoride is topical and that works by helping to slow down the decay process after it has started, that is, its action is in the early stage of decay, not on healthy enamel or established decay." (6)

"To this end, the constant bioavailability of fluoride in the saliva in low doses is desirable. Unfortunately, the increase of salivary fluoride levels from living in a 1ppm fluoridated area is too weak to have any noticeable effect." (22)

The CDC noted in 2001:

"The concentration of fluoride in ductal saliva, as it is secreted from salivary glands, is low – approximately 0.016 parts per million (ppm) in areas where drinking water is fluoridated and 0.006 in nonfluoridated areas. This concentration of fluoride is not likely to affect cariogenic activity." (23)

The required elevation of baseline levels only occurs after using fluoridated toothpaste or mouth rinse, a concentration of 1,000 ppm or more instead of 1 ppm from water. (24)

This elevation of fluoride levels in the saliva is transient and results from fluoride ions bound to oral tissues after using high strength topical agents being slowly released into pooled saliva.

Fluoride recirculated through the salivary ducts is of minimal contribution to pooled salivary levels, even after swallowing large doses as in fluoride toothpaste or tablets. (25)

"The burden of tooth decay is highest among the most deprived socioeconomic groups, and this is the segment of the population for which the benefits of CWF appear to be greatest." Page 9

This statement is clearly not aligned with the findings of the York Review and SCHER Review.

Increased decay rates among low SES groups are more strongly correlated to other factors such as parental education, family income, poor diet and reduced access to dental care. These factors are not improved by fluoridating the water supply, and there is no weight of evidence to support such a claim, which is why the major reviews conclude:

"The efficacy of population based policies, e.g. drinking water, milk or salt fluoridation, as regards the reductions of oral health social disparities, remains insufficiently substantiated." SCHER 2011 (11)

"There appears to be some evidence that water fluoridation reduces the inequalities in dental health across social classes in five and 12 year-olds, using the dmft/DMFT measure. This effect was not seen in the proportion of caries-free children among five year-olds. There was not sufficient data for the effects in children of other ages to investigate fully." York page 33 (7)

The argument that water fluoridation of the entire community is necessary in order to offer at least some benefit in decay reduction to young children in low SES circumstances, who do not brush their teeth, appears very unconvincing.

Aside from the minuscule elevation it contributes to pooled salivary fluoride, too weak to have any effect on caries, plaque growing on the teeth will block the penetration of fluoride to the enamel surface. (26) Furthermore, the caries balance in much of this demographic, with poor high sugar diets, would be heavily in favour of demineralization, so that any effect of fluoride would be further negated. (27)

It would be appropriate to note that tooth decay is caused by consumption of sugar and failure to remove plaque, it is not caused by fluoride deficiency. Efforts to close the gap that exists between SES classes tend to succeed far more when the focus is on diet and oral hygiene rather than on putting fluoride in the drinking water. (28)

NZ data show Maori, who are disproportionately higher represented in the low SES groups, are no closer to Europeans in fluoridated communities than they are in non-fluoridated.

2011 FIVE YEAR OLDS % Caries free	EURO	MAORI	GAP
FLUORIDATED	71.39	42.6	28.79
UNFLUORIDATED	66.26	39.3	26.96

Data sourced from: www.health.govt

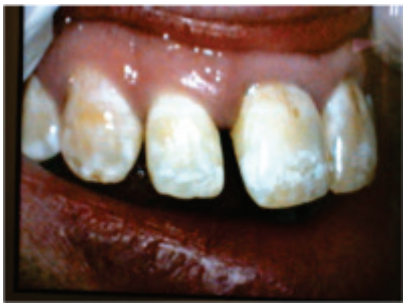
On the other hand, Maori are at greater risk of fluoride toxicity due to higher fluoride diets, lower calcium and lower vitamin D. (29)

Research in the USA indicating much higher levels of dental fluorosis and toxicity effects among African American and Latino groups have led to protests and civil unrest. (30)

Furthermore, communities which have stopped water fluoridation have shown no increased decay rate and in most cases, a reduction. (31,32)

In New Zealand, Timaru (ceased CWF in 1985), New Plymouth (2011) and Ashburton (2002), saw reductions in decay after cessation. Disappointingly such information has not been readily advertised by the Ministry of Health and generally is only released after OIA requests. (33)

Comments on: "Known effects of fluoride exposure – dental fluorosis"



"In the common, mild forms it is of minor or no cosmetic significance." Page 9

"No severe form of fluorosis has ever been reported in New Zealand." Page 9

While mild fluorosis is of minor cosmetic significance, it is a biomarker indicating fluoride overdose, and should raise questions of fluoride ingestion levels at the individual level and potential effects on other tissues and organs in the body, such as the thyroid or brain (34)

The review fails to mention the incidence of moderate fluorosis, which is a cosmetic problem requiring expensive treatment. In NZ it is around 5%, or 20,000 people.

The York Review estimated that up to 16% of fluorosis is of cosmetic concern.

"The risk for mild fluorosis that is associated with fluoride exposure is highest for formula-fed infants, and young children who are likely to swallow toothpaste. In some cases the fluoride intake by these groups can approach or exceed the currently recommended conservative upper intake level, but the rarity of cosmetically concerning dental fluorosis in New Zealand indicates that such excess intake is not generally a safety concern." Page 9

The "upper limit" is used in NZ and Australia to indicate how much fluoride you can give at the community level without causing unsightly dental fluorosis to more than 5% to 10% of the community. It addresses the visible (and undeniable) outward signs

of fluoride overdose, but takes no account of the doses at the individual level which can cause increased health risks with chronic ingestion.

Such measures are the Minimal Risk Level (MRL) and Reference Dose (Rf), which are respectively 0.05mg/kg and 0.06mg/kg of body weight/day as the toxic limit, and are generally much less than the UL in most age groups. (34)

"There is strong evidence that CWF is a cost effective use of ratepayer funds"

PAGE 13

One could ask whether funding for public health measures (and indeed liability) should be the responsibility of city councils instead of central government.

Studies on cost effectiveness are flawed and misleading, as they don't consider all the relevant factors and embellish the relevance of factors they do account for.

1. In calculating cost per head, they assume the whole community is gaining a (questionable) benefit, whereas in fact it appears that only a small section of the community are likely to have any significant benefit (young children who don't brush and don't eat much sugar). The cost should be divided by this number, not the entire population.

2. They often include costs for travel, loss of productivity, etc, which are not necessarily applicable.

3. They don't account for cost of PR campaigns, maintenance of equipment, staff payments.

4. They don't account for the cost of repairing fluorosis of aesthetic concern and emotional and social costs of this disfigurement.

5. They don't consider the cost of potential health issues: as fluoridation can increase the risk of conditions such as mental health disorders, diabetes, cardiovascular diseases, bone and joint diseases if they exceed chronic total fluoride intake limits.

6. They overstate the decay saving:
NZOHS & MOH data indicate 0.2 surfaces every 6 years, not 0.6 every year as in 2001 NZ study. (35) Furthermore, the aged population data presented above indicate zero lifetime decay reduction benefit.

7. If one assumes benefit, salt fluoridation is more cost effective as a community fluoridation measure then water fluoridation and has the added advantage that it can be targeted to at-risk groups. (36)

Concluding remarks

Just as individual research studies need to meet certain criteria in order to be considered reliable and relevant, so too do scientific reviews. The York Review 2000, NRC Review 2006 and SCHER Review 2011 do.

This one does not.

This report, far from being the robust definitive review that it repeatedly purports itself to be, falls well short of being a credible review of the conflicting science around fluoridation.

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APPENDIX 1: Critique of the Hastings Experiment
Written by Dr Paul Connett PH.D Chem, B.A. (Hons) Professor Emeritus in Environmental Chemistry at St. Lawrence University in Canton, NY

The Smoking Gun

This NZ fraud begins with a smoking gun letter (dated Oct. 12, 1962) from G. H. Leslie the director of NZ Government dentistry – who EIGHT years into the 10-year Hastings Napier trial (1954-64) complains that they cannot find the evidence to convincingly demonstrate a relationship between fluoridation and lower tooth decay.

12 October, 1962
Mr. Swann,
I have delayed acknowledging receipt of Dr. Roche's letter to you and replying to your minute in the hope that I would by now be able to give a positive reply to your enquiry. I still cannot. No one is more conscious than I am of the need for proof of the value of fluoridation in terms of reduced treatment. It is something which has been concerning me for a long time. It is only a matter of time before I will be asked questions and I must have an answer with meaning to a layman or I am going to be embarrassed and so is everyone else connected with fluoridation. But it is not easy to get. On the contrary it is proving extremely difficult. Mr. Espia is conferring with Mr. Bock and Mr. Ludwig and I am hopeful that in due course they will be able to make a practical suggestion.

I will certainly not rest easily until a simple method has been devised to prove the equation fluoridation = less fillings

(G.H. Leslie)

Director, Division of Dental Health

With this letter we have what amounts to a "smoking gun" as far as the inability of NZ dental officials and researchers to show the effectiveness of fluoridation – some eight years into the ten-year Hastings Napier fluoridation trial. The Mr. Ludwig, who Leslie refers to, is the lead researcher for this trial.

However, miraculously, two years later this trial was proclaimed a great success in demonstrating that fluoridation resulted in a large reduction in tooth decay (over 60%) and the result was used to push for fluoridation throughout the country. So how in the space of two years was this dramatic turnaround achieved?

The answer came from the late Dr. John Colquhoun, the former Chief Dental Officer for Auckland, who after retirement did a PhD thesis on the history of fluoridation in New Zealand (Colquhoun, 1987). As part of his doctoral research he was allowed access to the official files on the Hastings Napier trial (though according to his thesis advisor Professor Robert Mann, it became evident that some were incomplete, especially regarding Napier).
Based on these official files he was able to see how the deception was orchestrated.

In his thesis and in an article published in The Ecologist (Colquhoun and Mann, 1986) he showed that the massive reduction in tooth decay claimed for Hastings was a complete artifice.

According to Colquhoun the Hastings deception was in three parts:

1) After about two years the control city of Napier was dropped for bogus reasons.

2) The reduction in tooth decay claimed was based on comparing tooth decay in Hastings at the beginning and the end of the trial (and not a comparison between tooth decay in Hastings and Napier).

3) The method of diagnosing tooth decay was changed during the trial. Colquhoun describes this third aspect of the deception: "The school dentists in the area of the experiment were instructed to change their method of diagnosing tooth decay… Before the experiment they had filled (and classified as "decayed") teeth with any small catch on the surface, before it had penetrated the outer enamel layer. After the experiment began, they filled (and classified as "decayed") only teeth with cavities, which penetrated the outer enamel layer. It is easy to see why a sudden drop in the numbers of "decayed and filled" teeth occurred.
This change in method of diagnosis was not reported in any of the published accounts of the experiment."

What qualifies these activities as scientific fraud, in my view, is the last sentence: "This change in method of diagnosis was not reported in any of the published accounts of the experiment."

To the best of my knowledge the evidence that Colquhoun and Mann put forward for this rigged trial has never been refuted. In an email I received from Robert Mann (Dec 22, 2013), he wrote:

"I have never been aware of any attempt at rebuttal, let alone a refutation."

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APPENDIX 2: Written by Dr Paul Connett PH.D Chem, B.A. (Hons) Professor Emeritus in Environmental Chemistry at St. Lawrence University in Canton, NY

The Lee Dennison "Study"

This is a study of self-selected data with no blinding and little weight given to confounding factors. Clearly the York team would have rejected it.

Firstly, this study breaks the fundamental rule of scientific enquiry. Both Lee and Dennison are ardent pro fluoridationists, and selected data knowing what outcome they wanted. To avoid unconscious bias data must be selected "blind", unlike this study. Napier and Hastings were chosen for New Zealand's original study because of their demographic similarity, to minimize confounding factors.

Wellington, by contrast, is quite unsuitable for comparison with any other community: It has the highest educational and income levels in New Zealand, both of which correlate to good dental health. It would be extremely difficult to allow for confounding factors using Wellington statistics. We only need to look at statistics from nearby Hutt Valley to see this.

For Form 2 children, Hutt Valley Health records 1.10 DMFT in fluoridated areas and 1.01 DMFT in unfluoridated areas. It shows up in the Maori statistics too: 1.18 DMFT fluoridated, 0.72 DMFT unfluoridated (i.e. the unfluoridated areas have less decay than the fluoridated areas. Obviously something in that area is leading to better teeth (irrespective of fluoridation status).

In fact, Canterbury and Wellington are not a particularly good fit. For example, Wellington is much more urbanized than Canterbury, and there is a well-known correlation between urbanization and lower rates of decay.

Amongst the serious flaws in this study are the following:

The fluoridation history of the subjects was not known, nor whether they used fluoride toothpaste or not;

Two of the communities discontinued fluoridation (i.e. changed their fluoridation status) during the subjects' lifetime;

- There was no blinding;
- Data collection was not standardized;
- Examiner reliability was not ensured;
- Comparability of subjects was not assessed;
- Confounding factors were not eliminated;
- The mineral, particularly calcium and phosphorus, content of the respective water supplies was not determined

- The socio economic evaluations are completely unreliable. They use the 1996 TEFA ratings to assess the 1996 data. But the 1996 TEFAs were derived from 1991 census data. Moreover, TEFAs have been roundly criticized by schools for not accurately reflecting the socio economic status of their students. Not only were the TEFAs based on old data, but that data was derived from just a limited sampling of students (not the entire student population).

But there is an even more fundamental flaw. To adequately correlate socio economic status with decay, you need to do so on a subject-by-subject basis. You need to associate a particular child's DMFS with that child's status, not give every child in the school the same average value (TEFA). As a specific demonstration of the unreliability of the socio economic assessments used in the study, Te Aro School (Wellington) was designated decile 3 in 2002 (lowest group) but decile 8 in 2003 (highest group). (see table below)

The ethnic evaluations are also worthless. Despite the fact that the study was not designed to assess differences based on ethnicity, the authors make broad statements based on inadequate data: "in the non-fluoridated group, the mean DMFSs score of Maori five year olds was double that of the "Other" group, and that for Pacific children, three times greater than the "Others". That non-fluoridated group is Canterbury, which has a low Maori/Pacific population.

Drawing conclusions from such a small sample is worthless. One must also question why South Canterbury was included in the Canterbury sample.

The obvious comparison would be between Wellington and Christchurch. Including South Canterbury makes the unfluoridated sample much larger than the fluoridated sample. Why was that done? It creates a suspicion that the higher decay rates of Timaru were added to pull down the Christchurch figures.

As an indication of the readiness of the authors to [edited], they quote Fergusson (1986), Stockwell (1990), Treasure (1994), Slade (1996), Jones (1999) and Riley (1999) as authorities but neglect to mention that the York Review (2000) said those studies were poor quality and could not be relied on.

But ultimately their conclusions actually showed there was not much difference anyway.

"RESULTS: Caries prevalence and severity was consistently lower for children in the fluoridated area for both age groups, and within all subgroups. Five year olds in the fluoridated area had 2.63 dmfs (sd, 5.88), and those in the non-fluoridated area 3.80 dmfs (sd, 6.79). For 12 year olds the respective figures were 1.39 DMFS (sd, 2.30) and 2.37 DMFS (sd, 3.46). Multivariable analysis confirmed the independent association between water fluoridation and better dental health."
See study.

This results in a saving of 1.17 decayed, missing of filled surfaces in 5 year olds and 1.09 decayed, missing or filled surfaces in 12 year olds. This is hardly any achievement.

Decile ratings of selected schools

	2002	2003
Lumsden School	7	3
Homebush School	5	9
Waihao Downs	6	10
Tapapa School	9	4
Te Moana School	9	5
Te Aro School	3	8
Glendowie School	3	7
Whangara School	3	7
Ngamatea School	4	9
Tahora School	4	8
Waingaro School	4	8
Rissington School	5	9
Glen Oroua School	4	10
Arthurs Pass School	7	1
Aberfeldy School	7	3
Whangaehu School	7	3
Kapuni School	8	3
Putorino School	8	3
Otuni School	9	2
Tiraumea School	9	3
Timaru Christian	9	4
Makarora Primary	10	5
South Wellington	5	8

APPENDIX 3: Critique of NZOHS 2010

This survey describes itself as a "snapshot" of the oral health of New Zealanders at a particular point in time.

It is important to note that it was not one of the objectives of the 2009 NZOHS to compare the oral health status of people by fluoridation status, and therefore the survey cannot be considered a fluoridation study as such. The following results are for a snapshot in time and constitute an ecological analysis based on current place of residence. As such, they do not take into consideration lifetime exposure to fluoridated and nonfluoridated water supplies. Individuals who currently live in fluoridated areas may have spent time in non-fluoridated areas, and the reverse is also true. Furthermore, there may be other confounding facts that have not been taken into consideration.

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While it yielded some useful information, it was not a scientifically designed piece of research into fluoridation, so it is invalid to draw any conclusions from it.
The authors repeatedly warn readers of this fact, for example in addition to a myriad of uncontrolled confounding factors, the sample size studied was statistically insignificant.

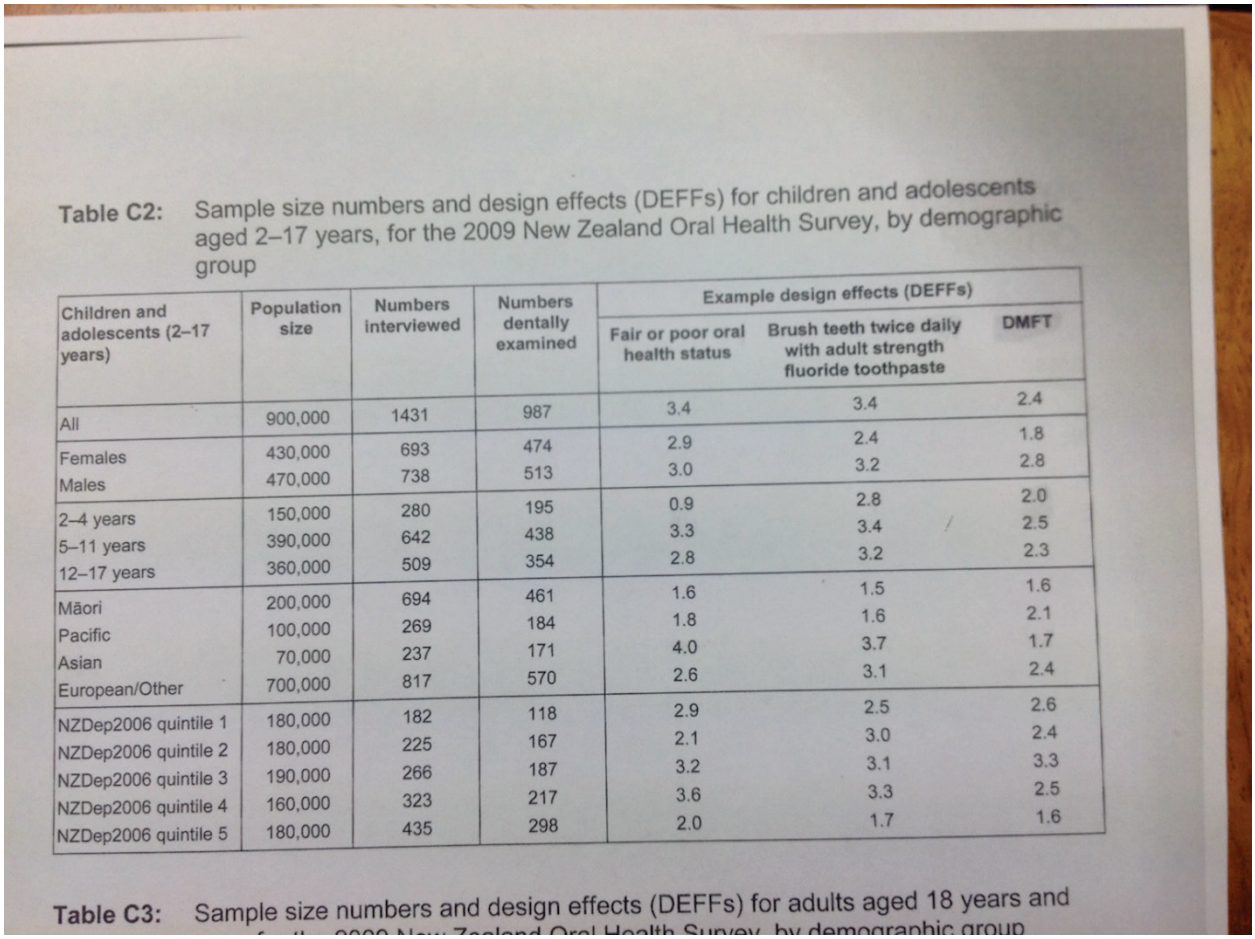

Table C2: Sample size numbers and design effects (DEFFs) for children and adolescents aged 2–17 years, for the 2009 New Zealand Oral Health Survey, by demographic group

Children and adolescents (2–17 years)	Population size	Numbers interviewed	Numbers dentally examined	Example design effects (DEFFs)		
				Fair or poor oral health status	Brush teeth twice daily with adult strength fluoride toothpaste	DMFT
All	900,000	1431	987	3.4	3.4	2.4
Females	430,000	693	474	2.9	2.4	1.8
Males	470,000	738	513	3.0	3.2	2.8
2–4 years	150,000	280	195	0.9	2.8	2.0
5–11 years	390,000	642	438	3.3	3.4	2.5
12–17 years	360,000	509	354	2.8	3.2	2.3
Māori	200,000	694	461	1.6	1.5	1.6
Pacific	100,000	269	184	1.8	1.6	2.1
Asian	70,000	237	171	4.0	3.7	1.7
European/Other	700,000	817	570	2.6	3.1	2.4
NZDep2006 quintile 1	180,000	182	118	2.9	2.5	2.6
NZDep2006 quintile 2	180,000	225	167	2.1	3.0	2.4
NZDep2006 quintile 3	190,000	266	187	3.2	3.1	3.3
NZDep2006 quintile 4	160,000	323	217	3.6	3.3	2.5
NZDep2006 quintile 5	180,000	435	298	2.0	1.7	1.6

Table C3: Sample size numbers and design effects (DEFFs) for adults aged 18 years and

It is profoundly inappropriate to use the NZOHS 2010 for advising policy on water fluoridation.

Analysis of Official Information request responses to: NZ Report – Health effects of water fluoridation: A review of the scientific evidence

Prepared on behalf of Fluoride Free NZ by G Mark Atkin BSc, LLB(Hons)

We have obtained information about the report's development from the Office of the Chief Science Advisor under the Official Information Act. The OIA requests were necessary after the office exhibited reluctance to our requests for transparency of information.

This information is not complete, and in places appears inconsistent. However, it is sufficient to raise serious concerns about the report's reliability, and the political bias observed by some of our reviewers. In all it tends to confirm the view that this is not a robust, objective, scientific report on the current science around water fluoridation, but was prepared to a political timeframe to meet a predetermined political outcome – to endorse water fluoridation as "safe and effective".

The methodology

Contrary to the methodology claimed in the report, (that the academic writer wrote the report which was then peer reviewed by the panel) the actual methodology was as follows:

- A panel of fluoridation advocates was hand-chosen by the pro-fluoridation chairs
- The panel members each prepared a summary of their views on the science in each member's area of claimed expertise.
- These summaries were given to the report author in a single one day session.
- The report author tidied these up, and added some further comments which she had prepared earlier. This was before she had received the bulk of the information.
- The author then gave this back to the panel to "review" their own work. It is therefore hardly surprising that they made few changes, they were only checking that the author had correctly relayed their own summaries.

Timeframes:

- 1 April – writer begins supplementary review (i.e. 6-8 weeks before receiving the bulk of the pre-written papers from the review panel).
- May 15-30 (pro-fluoridation) expert panel gives author 'state-of-the-science' briefing
- First draft complete 15 June (only 2-4 weeks to prepare this.)

Selection of the review panel

The review panel was established by the RSNZ with input from co-chairs. The approach was quoted as:

1. "Project co-chairs and science writer/coordinator to identify high-level headings for the review report and consult with Ministry of Health for their suggestions of issues needing coverage

2. RSNZ to invite experts onto panel in accordance with the identified headings and with the following minimum membership:

    a. Scientist - Public health Epidemiologist
    b. Scientist - Public health (dentistry specialty)
    c. Scientist - Toxicology
    d. Other relevant scientific experts ...
    e. Lay observer - a respected member of the public"

The responses received cannot identify any effort made to ensure the review panel had a balance of positions neutral, for, or against fluoridation on it (as the York and NRC reviews did), and evade the question of whether anyone meeting the expertise criteria, and with views opposing fluoridation, was ever approached.

Some of those approached declined to be involved, as follows:

On 5 April Prof Skegg wrote to Prof Gluckman:

> "As you will see below, however, [withheld] is questioning the feasibility of our approach… I can understand why any reputable scientist would be reluctant to put their name to a report if they have not had time to take a first-hand look at the evidence."

On 2 May Prof Skegg wrote (emphasis added):

> "You will have seen that [withheld] has agreed to join the panel. Unfortunately at the same time as adding a member, we have lost another. Professor [withheld] has just withdrawn: after reading the memorandum again, she felt she had insufficient expertise in the specific area and expressed reservations similar to [withheld]."

Selection of international reviewers

These were selected by the (pro-fluoridation) panel and vetted by the (pro-fluoridation) chairs. They are from fluoridated countries only. More professionals in fluoridated countries are biased in favour of fluoridation. However, thousands of professionals in fluoridated and non fluoridated countries have been very vocally opposed to fluoridation. See the thousands of signatures on the international Professionals Statement Against Fluoridation: http://fluoridealert.org/researchers/professionals-statement/

Selection of material to review

The following databases were used:

- Pubmed/Medline;
- Web of Science;
- Embase;
- Google Scholar;
- Cochrane library database;
- NFIS scanning was also checked (Reviews of scientific papers – lists new relevant studies up to June 2013).

Pubmed and Medline are known not to carry fluoride-adverse research, as identified by the NZ Public Health Commission in 1994/95.

The NFIS, is a now de-commissioned fluoridation support and co-ordination service, which was called the National Fluoridation Information Service (NFIS). It was a New Zealand Ministry of Health-funded consortium, contracted to "provide a proactive service that helps advance water fluoridation" (clause 2.2, clause 2.4 subclauses 3, 5, 6, 8, 9), and required to "not act in a way that may contradict or be inconsistent with Ministry policy on water fluoridation" (clause 2.3). In fact, the NFIS misrepresented itself, as it never provided objective unbiased information on fluoridation when it was active.

If one wanted the best available research on fluoride, as quickly as possible, the obvious place to look would surely be the only international journal dedicated to this subject. The journal is called *Fluoride* and was referenced heavily by the NRC review. While there is a reference in the Report's citations, the Chief Science Advisor's Office could not identify this as a journal that was identified for reference.

Review of the fluoride toxicity information was deemed too difficult for this panel. On 5 April 2014 Prof Skegg wrote to Prof Gluckman:

> "As you will see below, however, [withheld] is questioning the feasibility of our approach. As you know, I have always had concerns that - whereas the benefits of fluoridation can be summarised succinctly - the literature on potential risks is vast and quite complex. I can understand why any reputable scientist would be reluctant to put their name to a report if they have not had time to take a first-hand look at the evidence… Do you envisage that we could present our report as a synthesis of reviews by reputable evidence-based groups in other countries…" (Note that this refers to politically biased profluoridation organisations such the National Health and Medical Research Council of Australia, as confirmed in another email, rather than the NRC Review, which represents the "state of the science" on fluoride toxicity as at 2006.)

On 10 April Prof Gluckman wrote to Prof Skegg:

> "The reality is that the bulk of these issues have been dealt with by major agencies/academies in recent years and of course a report produced in short order will rely heavily on those."

So the NZ Report is, on the toxicity question, nothing more than a summary of pro-fluoridation international reviews, and therefore does not provide any scientific authority in its own right; nor does it add anything new to the scientific debate on fluoridation.

The types of papers accepted

Papers linked to water fluoridation were assessed for relevance based on the criteria that they were:

- available in English;
- primarily focused on community water fluoridation or concerned with fluoride in levels used for CWF;
- from NZ or comparable context.

Bullet 2 excludes much relevant toxicological research. In particular it excludes all laboratory animal studies on toxicity – exactly the type of study used to test pharmaceuticals. This was the restriction imposed on the York Review, which is why it could not draw any conclusion about safety.

Conversely, these are exactly the studies used by the NRC Review, which is, to date, the definitive review on health risks from fluoridation. This review, conducted under the National Academy of Sciences, sometimes called "The World Court of Science" is ignored by fluoridation promoters because it was not ostensibly about fluoridation at 0.7 to 1ppm, even though many of the studies it included were on communities fluoridated at this level.

Bullet 3 is designed to exclude relevant research such as the Chinese IQ studies used in the Harvard meta-analysis, which is misrepresented in the NZ report.

Validity criteria included:

- robust design;
- adequate sample size;
- systematic data collection to ensure minimal bias;

We note that the Dunedin IQ study, given undue weight in the Report, does not meet these criteria. (This does not reflect on the larger study from which non-fluoride-related data was taken).

Another example where the report fails to meet these criteria is where it relies on a letter to the editor by Douglass and Joshipura to deny the findings of Dr Elize Bassin on osteosarcoma. This unscientific approach was previously taken by the Australian Government NHMRC 2007 Review. (The research promised in that letter did not address Bassin's core finding that the risk is associated with age of exposure at all, let alone disprove it (see below).

Statement on Dental Fluorosis

The two latest scientific studies in NZ show that dental fluorosis rates are twice as high in fluoridated communities as unfluoridated communities (approx. 30% vs approx. 15%). The Report states (emphasis added):

> "…the 2009 New Zealand Oral Health Survey, which showed the overall prevalence of moderate fluorosis to be very low. The survey indicated that fluorosis prevalence is not increasing, and that <u>levels of fluorosis are similar between fluoridated and non-fluoridated areas.</u>"

Note: as the survey was a 'snapshot in time" it cannot possibly identify trends.

The Survey report contains the following disclaimer at p167:

> "It is important to note that it was not one of the objectives of the 2009 NZOHS to compare the oral health status of people by fluoridation status, and therefore the survey cannot be considered a fluoridation study as such. The following results are for a snapshot in time, and constitute an ecological analysis based on current place of residence. As such, they do not take into consideration lifetime exposure to fluoridated and non-fluoridated water supplies. Individuals who currently live in fluoridated areas may have spent time in non-fluoridated areas, and the reverse is also true. Furthermore, there may be other confounding factors that have not been taken into account in this analysis, such as the usual reason for visiting a dental professional, and other sources of fluoride such as fluoride toothpaste."

We asked "(a) has the author read the published reports on the Southland (2005) and Auckland (2008) dental fluorosis studies? (b) If so, why does the author cite the 2009 Oral Health Survey on this subject, in light of its disclaimer on page 167, rather than bona fide research?"

The reply was:

> "The NZ Oral Health Survey is the most recent and comprehensive survey of oral health across the whole population of NZ, and is therefore an appropriate reference for a general statement on fluorosis in NZ."

So the Chief Science Advisor's Office has deliberately chosen a survey which itself states "should not be considered a fluoridation study" over *bona fide* science.

Systemic benefit or otherwise

We asked:

> "The author quotes Featherstone 1999 as authority for the claim that fluoride works systemically during tooth formation to harden enamel against decay. Why was there not acknowledgement in the report that the majority view currently is that systemic inclusion in tooth enamel has no significant effect on tooth decay, while a minority still cling to it? Instead the report gives the impression that such benefit is established fact, when the opposite is true. Is the author aware that the quoted paper in fact was the turning point that refuted that belief, in favour of topical effect? If so, why does she falsely quote this paper as endorsing the refuted theory of a systemic pre-eruptive mechanism?"

After acknowledging that the reference to Featherstone was a citation error, the reply went on:

> "The lead writer (and panelists) is aware that this paper is used by some to claim that fluoride has no systemic effect, but notes that this is not the conclusion drawn by the paper's author."

Featherstone actually says:

> "The level of fluoride incorporated into dental mineral by systemic ingestion is insufficient to play a significant role in caries prevention. The effect of systemically ingested fluoride on caries is minimal". (Extract - p31)
>
> "The topical effects of fluoride are over-riding, and the systemic incorporation of fluoride in the tooth mineral is unfortunately not of major benefit" (p37)
>
> "The role of systemically incorporated fluoride is of very limited value." (p37) And in the conclusions "(3) The systemic benefits of fluoride are minimal".

Dr Robin Whyman of the former NFIS states in his affidavit in the Sth Taranaki District Council fluoridation case:

> "Fluoride enhances enamel remineralisation. The effect is "topical". This means it works by the fluoride coming into physical contact with the biofilm [plaque on the teeth] and surface of the tooth enamel."

Featherstone also noted that a minimum of 0.03ppm fluoride is required in saliva. Although study results vary, the CDC's official position is that in a fluoridated community, the fluoride level in saliva is only 0.016ppm – too low to provide topical

benefit. The NZ Report mis-states this, saying that ductal saliva fluoride levels are always above the 0.03ppm required.
So it is NOT that "some [people] claim that fluoride has no systemic effect", but rather that in fact systemic pre-eruptively ingested fluoride has no significant role in caries prevention, but "some people" continue to cling to this now discredited theory of action.

Osteosarcoma

The Chief Science Advisor's Office claims that "All original research articles cited in the report were read in their entirety by Anne Bardsley".
Given that the main papers were processed by Bardsley in a 2 – 4 week window, it is difficult to see how she could have read and digested all the papers as well.
But if she did, Bardsley knows that, in relation to the osteosarcoma issue, the Douglass-Joshipura letter referred to *Kim et al* 2013, which confirms that its methodology does not address Bassin's core thesis of age-related risk in boys. The report refers to fluoride accumulation being shown by bone-fluoride levels, which is not related to osteosarcoma, but fails to state that this is irrelevant to Bassin's core finding. Further, there is also no legitimate scientific reason to cite the letter to the editor now that *Kim et at* has been published. It is worrying that this report continues to mislead the public in its dismissal of Bassin, as fluoridation promoters have done since 2006.

When asked what research addressed age-related exposure, the only specific reference given was to *Kharb et al*, (Kharb, S., Sandhu, R., & Kundu, Z. S. (2012). "Fluoride levels and osteosarcoma" *South Asian J Cancer, 1*(2), 76-77.) which does not address age-related exposure either, but does support a link between fluoridation and osteosarcoma. Oddly, there is no reference to the same authors' early, larger, study (Sandhu R, Lal H, Kundu ZS, Kharb S. "Serum fluoride and sialic acid levels in osteosarcoma" *Biol Trace Elem Res* 2011 Dec;144(1-3):1-5; E-published 2009), which supported a fluoride-osteosarcoma link even more strongly. In fact the researchers stated "this report proves a link between raised fluoride levels in serum and osteosarcoma".

As further evidence of bias in the report, the author refers to Bassin as "a small study", yet does not refer to Kim et al as such, even though it was a significantly smaller study than Bassin. Neither does the author mention that osteosarcoma is such a rare disease that Bassin's study is by far the biggest ever done. The reader is also mislead by the author referring to meaningless NZ statistics on the incidence of a comparatively tiny number of NZ osteosarcoma cases based on place of residence at the time the osteosarcoma was diagnosed, which is irrelevant to the age of exposure, as scientific evidence against Bassin.

Appendix: The stated methodology

Methodology:

1. Project co-chairs and science writer/ coordinator to identify high-level headings for the review report and consult with Ministry of Health for their suggestions of issues needing coverage

2. RSNZ to invite experts onto panel in accordance with the identified headings and with the following minimum membership:

    a. Scientist - Public health Epidemiologist
    b. Scientist - Public health (dentistry specialty)
    c. Scientist - Toxicology
    d. Other relevant scientific experts ...
    e. Lay observer - a respected member of the public

3. Royal Society NZ to convene one-day Expert Panel meeting to be attended by the science writer/ coordinator.

4. Panel members will be expected to present a state-of-the science briefing in their particular areas of expertise. The synthesis should include:
    a. What is known and not known
    b. Areas of consensus and any areas of debate in the literature c. ...

5. Science writer will:
    a. Summarise the Expert Panel briefings
    b. Supplement the briefings with independent review of the literature including any relevant Cochrane Systematic analyses.
    c. Prepare a synthesis report in accordance with the identified headings and/or any emerging headings recommended by the Expert Panel

6. Draft report to be circulated to Expert Panel for review and comment

7. Final draft report will be peer reviewed by two international experts to be identified by the Expert Panel and vetted by co-chairs

8. Peer reviewed report to be submitted to funders (Auckland City Council, Ministry of Health) and made publically accessible online at www.pmcsa.org.nz

Timeframe

April 1: Project Start
- Writer appointed and will start supplementary review of the literature co-chairs to identify key headings
- RSNZ to begin Expert Panel recruitment

April 17: Recruitment and appointment to Expert Panel completed

May 15-30: RSNZ convene Expert Panel for state-of-the-science briefing

June 15: first draft report circulated to Expert Panel for feedback

July 6: Report sent for international peer-review and review by Ministry of Health

July 30: Report finalized
August 7: Co-chairs' cover letter completed

August 15: Report provided to Ministry of Health and Councils

Aug 22: Report published

Review Comparison

Review	York	US National Research Council	NZ
Timeframe	1 year (1999-2000)	3 years (2003 – 2006)	3 months (April - July 2014)
Budget	£ 1 million	$US 6 million	$ NZ 50 thousand
Chair	Advisory panel: neutral; Review panel: pro-fluoridation	Pro-fluoridation	Two co-chairs, publicly committed to fluoridation
Panel makeup	There were two panels – a fully pro-fluoridation review panel and a mixed advisory panel: pro-, anti- and neutral.	13. Balanced: pro-, anti-, and neutral	5. All pro-fluoridation. Some panellists declined/resigned due to procedural concerns.
Methodology	Systematic review of original published research (approx 3,300). This was established by the UK Health Department "to prove once and for all the safety and effectiveness of fluoridation", as it was intended to launch a renewed push for expanding fluoridation. Excluded animal studies on toxicity, and medical case histories. Parameters were deliberately narrowed to exclude 100 case histories of fluoride-poisoned children received by the review Board.	Systematic review of original published research on adverse health effects of fluoride, from 1ppm upwards. 512 page report. Included animal studies on toxicity, and medical case histories. Focus was solely on health risks from fluoride; not claimed benefits.	Some original research purportedly reviewed. No record of what was rejected. No record of "anti-fluoridation" studies not cited. Pro-fluoridation panel members wrote their own summaries, gave these to the writer to consolidate, and peer-reviewed their own work. Co-chair Skegg admits that the research on toxicity is so 'vast and complex' that they could not possibly review it – second hand pro-fluoridation reviews were adopted instead, contrary to the statements in the public report. Excluded most studies on toxicity, just like York.
Transparency	Review was publicized before being conducted. Information provided openly to the public during the review. Pre-publication peer review included those opposed to fluoridation.	Open, transparent process. The existence and membership of the committee (including a short summary of the project) were all online. Parts of the first meetings were open to the	Conducted in secret, with no external input. Peer reviewers appear to have been selected for pro-fluoridation views. No attempt to have a balanced panel, and evasive when

Review	York	US National Research Council	NZ
		public and some public submissions were heard. Wide canvassing of external community for relevant research. Members of the public also submitted studies/papers for the NRC committee to include, and at least some of those were used.	asked what experts with views against fluoridation were approached.
Outcome	In spite of the bias with which it was established, this review presents a summary of the best available and most reliable evidence on the alleged efficacy of water fluoridation. "Given the level of interest surrounding the issue of public water fluoridation, it is surprising to find that little high quality research has been undertaken." No conclusive evidence on safety, or benefit to the poor. Evidence for general benefit in reducing tooth decay was based on few studies, of mediocre quality, with wide-ranging conclusions (including fluoride increasing tooth decay). "Legitimate scientific controversy will remain until better quality research is done."	The level (of natural fluoride only) allowed in the US of 4ppm is not safe. A promised but disallowed minority report would have recommended a maximum level of 0.4ppm until a truly safe level could be scientifically determined. The Chair stated in an interview for Scientific American "What the committee found is that we've gone with the status quo regarding fluoride for many years—for too long really—and now we need to take a fresh look . . . In the scientific community people tend to think this is settled. But when we looked at the studies that have been done, we found that many of these questions are unsettled and we have much less information than we should, considering how long this [fluoridation] has been going on."	Concluded that there was general consensus that fluoridation is 'safe and effective', as the two co-chairs had publicly proclaimed before the review. This was shortly after the Deputy Director of the National Poisons Centre, Michael Beasley, stated publicly that the "I think the jury is still out regarding the safety of Fluoride." Claimed there was an adequate margin of safety in spite of repeated statements throughout the report identifying there was not.