International Peer Review of the Royal Society/PM Science Advisor Office
Fluoridation Review

Dr Kathleen Theissen PhD
Environmental Risk Scientists
NRC Panel member

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

Kathleen M. Thiessen, Ph.D.
September 8, 2014

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 (less protective) 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 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 Strunceka 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 "fluorinated 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.

References


Chris Neurath  
Research Director  
American Environmental Health Studies Project

Comment:

My critique is only of the neurotoxicity section. I doubt I have time to do anything similar with the rest of the report. I've been working a lot on the neurotox issue recently so I am able to do that off the top of my head. The other issues would take a lot longer.

As Hardy said, this report is such a joke that it does not deserve the time spent to critique it in a scientific manner. It may well be a waste of our time. Instead we should just offer a summary of why it is biased and publicize that information on the FAN website, FAN Bulletins, and through other venues where people will actually read it. In fact, the NZ activists have already done a good job of this. I'm not sure a genuine scientific review of it is worthwhile. Why do a scientific review on a political/propaganda report?

Critique

Almost everywhere in the report, scientific evidence is selectively cited to produce a biased view of the evidence. The common term for this is “cherry picking”.

The section on neurotoxicity repeatedly claims that existing studies showing neurotoxicity in animals and humans were at exposure levels much higher than humans experience in places with artificial fluoridation. Specific studies are cherry picked which are claimed to have exposures 100-fold and even 230-fold higher than for humans drinking fluoridated water.

The exposure levels in the studies with the lowest exposure levels are not mentioned. Not even the average exposure level is mentioned, just the extreme high exposure studies. This isn’t just unscientific, it is intellectually dishonest.

Furthermore, as so often in works by fluoridation defenders, dose and concentration are confused. In toxicology, it is the dose to the organism that is much more relevant than the concentration in water. As should be obvious, a water concentration of 1 mg/L fluoride will produce a dose of 1 mg/day in a person who drinks 1 L/day, but a much higher dose of 4 mg/day in a person who drinks 4 L/day.

The NZ authors also apparently do not realize that rats and mice are much less sensitive to some of fluoride’s harmful effects than humans. Experts in this field have estimated that lab rodents are 5 to 20 times less sensitive than humans. [can cite Whitford and DenBesten]

The NZ report claims the human studies of neurotoxicity/IQ were in places with “very high” natural fluoride levels and that these levels were “much higher” than any level found in NZ. This claim is again based on cherry picking the evidence. In fact, one of the Chinese IQ studies had an average water concentration of 0.88 mg/L in the “high exposure” group. Nine human studies have had a water concentration less than 3 mg/L in the “high fluoride” group. All
nine of these studies showed a substantial drop in IQ in the “high fluoride” group that averaged 9 points. While a water concentration of 3 mg/L is indeed higher than 1 mg/L, there will be overlap in the doses that individual children receive because some children will drink 3 times as much water as the average. Therefore, some NZ children with artificially fluoridated water will likely ingest as much fluoride (dose in mg/day) as did some of the children in the studies which showed a 9 point IQ drop.

The most glaring error in the NZ review of the neurotoxicity evidence is the apparent claim that the Choi et al (2012) systematic review of IQ studies found an average drop of only 0.45 IQ points amongst the 27 studies. The authors and peer reviewers of the NZ report all seem to have incorrectly thought that Choi’s “standardised weighted mean difference” was in units of IQ points. The correct conversion gives a difference of 6 IQ points, which is more than an order of magnitude higher. The unit “standardised weighted mean difference” is a common way to report results of meta-analyses, so the NZ author’s inability to understand this measure indicates they have little experience reading meta-analyses. This gives us little assurance they have correctly interpreted other scientific papers they reviewed for their report. Furthermore, this same error was made by a number of fluoridation promoters when the Choi paper was first published, but Choi and her co-author Grandjean publicly corrected this misunderstanding within days of it coming to their attention. That was almost two years ago. Apparently the NZ report’s authors and peer reviewers never got that message and have relied on what newspaper reporters and fluoridation promoters have erroneously said about the Choi study.

The NZ report mis-interprets Choi et al’s statement that the difference in IQ between the low and high fluoride groups “may be within the measurement error of IQ testing”. But by this Choi meant that a 6 IQ point difference may be too small for a test on an individual to reliably determine. Epidemiological studies rely on looking at large numbers of people to overcome this type of measurement error that would apply to an IQ test of an individual. The majority of the studies reviewed by Choi et al found a statistically significant difference in IQ, which means the IQ measurement error for each individual was overcome by the large sample size.

The next claim about the Choi et al study is also incorrect, as would have been easily seen if the NZ report authors had actually read the Choi systematic review or the original scientific studies. It is claimed that the studies did not take into account other sources of fluoride exposure besides from drinking water and that other sources of fluoride exposure are widespread in China. In fact, most of the studies did consider other sources of exposure, such as from food dried over coal fires. There are only a few places in China where domestic fires use high-fluoride coal briquettes which contaminate food with fluoride. Most of the studies reviewed by Choi were conducted in places where this source of fluoride does not occur. Several were conducted in places where domestic coal burning was the main source of fluoride exposure. Those studies did also consider fluoride from drinking water. Industrial fluoride pollution of water or air was also ruled out in many of the studies because they were conducted in rural areas remote from any industry.

Thus in almost all studies, major alternative sources of fluoride exposure were ruled out or controlled for. The NZ report authors seem to have simply regurgitating false claims that have
been made by fluoridation proponents, rather than actually reading the scientific studies they claim to be reviewing.

The next criticism of the IQ studies from the NZ report is that “Most of the studies fail to consider the effects of lead, arsenic, iodine deficiency, socioeconomic status, or nutritional status of the children ….”

This is an important issue, so it should be considered carefully, rather than just painted in crude terms such as “most of the studies”.

First, several of the studies did consider each of these potentially confounding factors, and at least one group of researchers [lead by Xiang] considered all of them and more.

Second, it is crucial to understand that if a risk factor for IQ did not vary along with fluoride exposure, it could not confound the relationship between fluoride and IQ. Thus, it could not reduce the validity of the study. So, simply failing to assess these factors in a study does not mean the study was confounded and produced an invalid result.

Evidence from hydrogeological studies in China suggest that at a local level, natural fluoride concentrations in water are unlikely to be associated with lead, arsenic, or iodine levels [add references]. Therefore, it is unlikely that most of the 27 studies reviewed by Choi would have had the high fluoride group also exposed to higher lead or arsenic, or lower iodine (iodine protects against IQ loss).

Third, Choi herself makes the argument that the 27 studies were all independent of each other and conducted in widely different geographical locations, so it is unlikely that some unmeasured confounding factor could have biased 26 of 27 studies all in the same direction to make it appear that fluoride was lowering IQ. The overwhelming consistency of the results suggests that fluoride really is the cause of the IQ decline, rather than some unconsidered confounding factor.

Fourth, the issue of potential confounding in the Choi studies, is ironically glossed over when the NZ report describes a single recent study which found no effect of fluoride on IQ. This is the Broadbent et al 2014 study, done with a cohort of kids born in 1972-1973 in the Dunedin area of New Zealand and followed through adulthood. In this study, neither arsenic nor iodine exposure was considered. Even worse, lead data was collected for the cohort but was not considered by Broadbent in his study of the effect of fluoride on IQ. The cohort had data on several factors which are known to affect IQ, and which Broadbent criticized the Chinese studies as not considering, but Broadbent himself did not consider or try to control for these factors in his own study. Maternal IQ was such a factor, in addition to lead. In fact, of the 14 potentially confounding factors Broadbent criticized the Chinese studies for not considering, he only considered 4. The pot calling the kettle black.
Speaking of the Broadbent paper, the NZ Review devotes an entire paragraph to it, even though it is in a tiny minority of fluoride neurotoxicity studies which have not detected an adverse effect. Cherry picking again on the part of the NZ Review.

If the Broadbent study were truly a “relatively high quality” study, then the NZ Review might have been justified in giving it so much more attention than all of the studies which did find an effect from fluoride. While on the face of it, the Broadbent study appears relatively high quality, it has a fatal flaw. It failed to properly account for the major sources of fluoride besides water fluoridation. Unlike most of the Chinese studies, where water or coal burning were the sole major source of fluoride, in the Dunedin cohort, almost all the children used fluoridated toothpaste and some of them took fluoride supplements. Yet Broadbent never considered total fluoride exposure or simultaneously controlled for the three (or even two) of the fluoride sources.

Why is this such a serious weakness? Because at the time the mothers were pregnant and the children were growing up, it was common practice for doctors and dentists to advise that fluoride supplements be taken by those living in non-fluoridated areas, as a substitute source of fluoride. Likewise, anyone living in a fluoridated area would not be advised to take a fluoride supplement, otherwise they would receive too much fluoride. A study by researchers at Otago University in Dunedin estimated the total fluoride intake of children who lived in non-fluoridated areas who took the recommended fluoride supplements and compared it to those who lived in fluoridated areas and did not take the supplements. The two groups had indistinguishable total fluoride exposures. [Guha-Chowdhury et al 1996]

Thus, there may have been almost no difference in total fluoride exposure between Broadbent’s group that lived in fluoridated areas compared to those who lived in non-fluoridated areas. This lack of contrast in total fluoride exposure between Broadbent’s comparison groups could completely explain why he found no difference in IQ: they had no difference in total fluoride exposure!

This serious weakness of Broadbent’s study is never acknowledged by Broadbent, and fluoridation defenders have even erroneously claimed that Broadbent did control for all sources of fluoride.

What is even more stunning than failing to acknowledge this weakness, is that Broadbent could have very easily avoided it by simply looking at total fluoride or controlling for all fluoride sources simultaneously. It makes one wonder whether he did do the proper analyses, found that total fluoride did lower IQ, and decided to hide that by not reporting it. That would be tantamount to scientific misconduct.

In any case, the NZ Review’s emphasis on Broadbent’s study reveals they are either biased or incompetent, or both.

REFERENCES:

Comments:

Dr Hardy Limeback  
NRC Review Panel member  
Head of Preventive Dentistry, University of Toronto

This report is a clear example of cherry picking, where only select studies that support the 'safe and effective' viewpoint were cited.  
If is far from a REALLY critical review of the literature. It is NOT a meta analysis. It is no better scientifically than a dentist's review that rubber stamped by uncritical referees and published in a profluoridation dental journal.

I can't be bothered to show step by step where this review does not meet the standards of critical scientific analysis.  
It is a political document.

Chris is right on the ball in critiquing this review. I'm not sure many people will appreciate just how detailed he is.

I'm disgusted by how sloppy the NZ reviewers were.  
They were obviously politically motivated.

The effort to critiquing every paragraph of the NZ review is taxing but once it is done and posted on the website, it would be most useful for those people who want to take on the promoters of fluoridation who will undoubtedly use this review to support the profluoridation agenda and point to how unscientific, one-sided, politically motivated this review really is.

I would be happy to lend my name to the list of scientific reviewers of this critique.

Dr James Beck  
Co-author, The case Against Fluoride

I agree with the opinion that false and dishonest public comment in favour of fluoridation should be refuted. I agree despite being sick of it; I find myself writing targetted rebuttals sometimes several times a week. On the current New Zealand case I don't think others can do any better than Chris has done.

Unless there is some specific need for my intervention I would like to leave it with you, Spedding and Chris. It's always a shame, I think, to omit the ethical issue but it doesn't appear in the NZ abomination.

Spedding Micklem  
Co-author, The case Against Fluoride
I'll try to help with this though have not been focusing on fluoridation matters lately. Chris's analysis is excellent and I think he is right to point out that the report's incredible blooper on the Choi paper casts doubt on its authors' basic competence to interpret numerical data.