AFFIDAVIT of Dr. Paul Harry Connett April 2011

COURT DETAILS	
Court	NSW Land and Environment Court
Registry	Level 4, 225 Macquarie St. Sydney, NSW
Case number	40590/10
TITLE OF PROCEEDINGS	
Applicant	Al Oshlack
First Respondent	Rous Water
Second Respondent	Ballina Shire Council
Third Respondent	Lismore City Council
PREPARATION DETAILS	
Filed for	Applicant
Contact name and telephone	Al Oshlack – 0415 140 410

AFFIDAVIT of PROFESSOR PAUL HARRY CONNETT

I, Dr. Paul Harry Connett of 82 Judson Street, Canton, New York USA 13617, solemnly and sincerely affirms and declares that:

1 I am a graduate of Cambridge University and hold a Ph.D. in Chemistry from Dartmouth College. Since 1983 I taught Chemistry at St. Lawrence University in Canton, NY where I specialized in Environmental Chemistry and Toxicology. I retired in May 2006. Attached and marked as **Annexure 1** is a copy of my Curricula Vitae.

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Date……..

2 I am of legal age and competent to testify.

3 I have the authority to make the following statements and declarations.

4 I have no commercial affiliations.

5 The Applicant has provided me with a copy of SCHEDULE 7 of the Uniform Civil Procedures Act – "***Expert witness code of conduct"*** the Practice Direction which I have read and agree to comply with. Annexed and mark as **Annexure 2** is a copy of the Schedule 7.

6 I am deposing this affidavit in reply to the Respondents witnesses.

7 I have been involved for 25 years with Waste Management. I have given over two thousand pro bono presentations in forty-nine US states, seven provinces in Canada and fifty-two other countries on incineration and waste management.

8 In 2010, I gave two presentations on Zero Waste for Sustainability to the United Nations Commission for Sustainable Development in New York City. In October 2010, I gave a presentation before the agriculture committee of the European Parliament and in November in the British House of Commons. In Jan 2011, I participated in a parliamentary hearing in Scotland on the problems posed by waste incineration.

9 I have co-authored six peer reviewed and published papers on dioxin, co-edited the newsletter Waste Not, provided expert testimony in court cases, critiqued numerous health risk assessments and co-produced over fifty videos on waste issues including a ten part series on dioxin. I have received many awards from environmental and citizens' groups in recognition of my work.

10 I have researched the literature on fluoride's toxicity and the fluoridation debate, interviewed many leading researchers and critics of fluoridation on video.

11 I am the Executive Director of Fluoride Action Network distributing world wide over 1,000 bulletins examining and researching literature on fluoride's toxicity for over 15 years.

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Date……..

12 I have given invited presentations on the dangers of fluoridation to legislative and research bodies in Australia, Canada, Germany, Ireland, Israel, Japan, New Zealand, and the United Kingdom and the United States. The latter has included invited presentations to both the US Environmental Protection Agency (EPA) and the National Research Council. I was invited peer reviewer of the York Review (McDonagh et al., 2000). I was invited to give testimony to the Fluoridation Forum in Ireland (Oct, 2000) as well as to a health committee of the Irish parliament.

13 I am the lead author of the recently published book "*The Case Against Fluoride*" How Hazardous Waste Ended Up in Our Drinking Water and the Bad Science and the Powerful Politics That Keep It There."

14 My research interests are the Interaction of Metals with Biological Systems (chromium and lead), the Build-up of Dioxins in food chains, Health risk assessment, the problems and dangers of incineration, Resource management for a sustainable society and Toxicity of Fluoride and the dangers of Water Fluoridation.

15 A list of my publications appears in **Annexure 3.**

16 I have before me for the preparation of this affidavit the following documents:

a. *Rous Regional Water Supply- Fluoridation Plants Including the Ballina Shire Council Marom Creek Water Supply- Review of Environmental Factors* dated March 2010 (REF);

b. the approval under section 6 of the *Fluoridation of Public Water Supplies Act 1957* (Fluoridation Act), granted to the First Respondent by the Director-General of NSW Health, to fluoridate the water supply within nominated areas of the Lismore City Council Local Government Area which was published in the *NSW Government Gazette* on 14 December 2007 (Lismore Approval);

c. the direction under section 6A of the Fluoridation Act, issued to the First Respondent by the Director-General of NSW Health, to fluoridate the water supply within nominated areas of the Richmond Valley Council Local Government Area which was published in the *NSW Government Gazette* on 14 December 2007

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Date……

(Richmond Valley Direction); and

d. the approval under section 6 of the Fluoridation Act, granted to the Second Respondent by the Director-General of NSW Health, to fluoridate the water supply Within nominated areas of the Ballina Shire Council Local Government Area which was published in the *NSW Government Gazette* on 18 December 2009 (Ballina Approval)

e. Copy of the Applicant's Points of Claim.

f. The Respondents affidavits of:

   i. Dr John Colin Chapman, dated 29th Nov 2010 (**JC Affidavit**)

   ii. Dr Anthony S Blinkhorn, dated 9th Dec 2010 (**AB Affidavit**)

   iii. Assoc. Prof. Anthony Brown dated 21st Dec 2010 (**AMB Affidavit**)

   iv. Prof. John Spencer dated 7th Dec 2010 (**JS Affidavit**)

   v. Dr Fredrick Alan Clive Wright dated 30th Nov 2010 (**FW Affidavit**)

g. The Applicant's affidavits in chief:

   i. Prof. Mark Diesendorf on 7 October 2010 (**MD Affidavit**);

   ii. Dr John Alexander Ryan on 7 October 2010 (**JR Affidavit**); and

   iii. Dr Andrew Paul Harms on 9 October 2010 (**AH Affidavit**).

17 I understand that the First and Second Respondents propose to construct several fluoridation dosage plants in their geographic areas of responsibility and that they have approval from NSW Health (or have been directed by NSW Health) to dose their public water supply by adding fluorine at a target concentration level of 1.0mg/L

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Date……..

with an overall accuracy of +1-5% and within an operating range of not more than 1.5mg/L and not less than 0.9mg/L (Approved 2Concentration)

18 In this section of my affidavit, unless **I** say otherwise, where I refer to "fluoridation" **I** am specifically referring to the addition of fluorine to a public water supply at the Approved Concentration. Where **I** refer to "fluoridated water'', **I** am referring to water that has been the subject of fluoridation.

19 Before I address to affidavits of Drs. Blinkhorn, Wright, Brown, Spencer and Chapman, it would be helpful if I summarized my concerns about the practice of water fluoridation based on my 15 years of researching the matter. Such a summary should put my commentary on their affidavits into a larger context.

20 A detailed and comprehensive overview of my concerns is provided in the book I co-authored with James Beck, MD, PhD and Spedding Micklem, DPhil (Ocxon), entitled "*The Case Against Fluoride*" and published by Chelsea Green (White River Junction, Vermont) in October 2010. For the convenience of the court a pdf copy of this book is provided in **Annexure 4**)

21 Overview of my Evidence

A) **Fluoridation is a bad medical practice.** Once fluoride is added to water

i) it is impossible to control the dose (people drink very different amounts of water and also get fluoride from other sources);

ii) it is impossible to control who it goes to – it goes to everyone, including the very young, the very old, those with poor nutrition and in poor health;

iii) there is no one overseeing any side effects from this *"prescription"* either individual doctor or health agency and

iv) last, but not least, it is a violation of the individual's important fundamental right of *"informed consent to medication."*

B) **The evidence that fluoridation significantly reduces tooth decay (especially in the permanent teeth) is very weak.** A summary of

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Date……..

this evidence is given in chapters 6-8 in *The Case Against Fluoride*.

C) **There is no adequate margin of safety** (see chapter 20, *The Case Against Fluoride*) to protect everyone in fluoridated populations from the harm demonstrated in countries with moderate to high levels of natural fluoride in the water (see NRC, 2006 and (*The Case Against Fluoride*, chapters 13-19). For example, if routine assumptions and safety factors are applied to the finding that IQ is lowered at 1.9 ppm (Xiang et al., 2003a,b) a safe level to protect the whole population for this serious effect would be 0.19 mg/day. Such a dose would be obtained simply by drinking one glass of water at 0.7 ppm fluoride. The Xiang study is one of 24 studies that have found an association between moderate exposure to fluoride and lowered IQ in children (see chapter 15 in *The Case Against Fluoride*).

D) **The promotion of this practice by dental organizations and health agencies in fluoridated countries has been both unprofessional and unscientific.** Promotion is based largely on the "authority" of endorsements by professional bodies and government agencies, rather than clear citations from the primary literature. Endorsements are effective with the public but they don't constitute scientific evidence unless they are supported by careful review of the literature. Perhaps the most disturbing aspect of the lack of science on fluoridation promotion is to discover how few health studies have been conducted in fluoridated countries on harmful effects demonstrated elsewhere. This is particularly true of Australia. The absence of study does not prove the absence of harm. The only rational explanation for such irresponsible behavior on part of the health agencies in fluoridated countries is that for some reason it has become more important to protect this practice than to protect people from harm.

E) **The risks posed by fluoridation vastly outweigh the benefits.** Can any one seriously maintain that securing – at best- a reduction in tooth decay of 0.6 permanent tooth surfaces (Brunelle and Carlos, 1990), could warrant risking a child's mental development as well as the other health concerns documented in the National Research Council report (NRC, 2006) and our book (*The Case Against Fluoride*, chapters 13-19)?

F) **At the very least the Precautionary Principle should eliminate the**

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Date……

practice of water fluoridation. To proceed with this practice with so many unanswered questions on health issues is reckless.

Affidavit of Prof Anthony Blinkhorn (AB)

22 Paragraph 15(e), of the affidavit of AB writes:

> *"Many parts of Australia have been fluoridated for over 40 years and there is no evidence of any general health problems, despite detailed investigations and rigorous government surveillance.[9,10]"*

23 This statement is false and neither reference 9 nor 10 support such a claim. In reality there have been neither *"detailed investigations"* nor *"rigorous government surveillance"* of the *"general health"* of fluoridated communities in Australia, despite the long history of fluoridation in this country.

24 Neither reference 9 nor reference 10 support the claim of *"detailed investigations"* or *"rigorous government surveillance"* of the *"general health"* of fluoridated communities in Australia.

25 Reference 10 is listed as: Medical Research Council Working Group. *"Water Fluoridation and Health".* United Kingdom, Medical Research Council, London, UK, 2002. This is a report from a committee appointed by the Medical Research Council of the UK (2002) as a follow up to the York Review of 2000 (McDonagh et al., 2000). Neither the York Review nor the MRC (2002) report dealt with any health studies conducted in Australia.

26 Reference 9 is listed as: Armfield J. *"The extent of water fluoridation coverage in Australia".* Australian and New Zealand Journal of Public Health 2006; 30(6):581-582.

This is a short (less than two pages) article written by Jason Armfield a dental researcher from University of Adelaide. As the title suggests the article deals with the extent of each state's population covered by water fluoridation programs. There is no discussion of health studies and certainly contains no *"detailed investigations"* or *"rigorous government surveillance"* of the *"general health"* of fluoridated communities in Australia.

27 In 1991 NHMRC recommended that some basic studies be undertaken

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Date……..

(NHMRC, 1991)

28 On fluoride's impact on the bone, the NHMRC panel wrote:

> *"If skeletal fluorosis is occurring at all in Australians, it is likely to be slight, and it will most likely occur in those who drink large amounts of water, or whose renal function is impaired. Studies of bone fluoride collected at autopsy in selected individuals could provide needed reassurance that the current policy is not resulting in hazardous levels of accumulation in bone."*

29 In the twenty years (as of 2011) since that recommendation was made, no Australian health authority has sought such reassurance.

30 On the possibility that some individuals in the population might be particularly sensitive to fluoride the NHMRC (1991) panel wrote:

> *"It is desirable to explore in a rigorous fashion whether the vague constellation of symptoms which are claimed to result from ingestion of fluoridated water can be shown to be reproducibly developed in these "susceptible" individuals. These claims are being made with sufficient frequency to justify well-designed studies which can properly control for subject and observer bias."*

31 Not one health agency in Australia, in the twenty years (as of 2011) since this recommendation was made, has attempted any formal study on the matter. This, despite the fact that citizens have offered to be tested in this way.

32 Practically all the studies that have been conducted on fluoride in Australia have been conducted on one single tissue, the teeth. The preoccupation here has been on demonstrating the benefits of fluoridation (i.e. supporting the Australian government's promotion of this practice) with respect to reducing tooth decay. The only adverse effect examined in any depth also pertains to teeth and that is the issue of dental fluorosis. Dental fluorosis is damage to the growing enamel that can occur prior to the eruption of the secondary teeth. This damage is largely dismissed as a cosmetic effect by promoters of fluoridation, whereas opponents stress that it is an indicator of over-exposure to fluoride.

33 Another example demonstrating the apparent lack of interest by

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Date……..

Australian authorities in investigating possible health effects in a scientific and responsible manner has been their failure to use the severity of dental fluorosis as a non-invasive biomarker for exposure in epidemiological studies in children. This approach was used to investigate bone fractures in Mexico (Alarcon-Herrera et al., 2001). Many potential health effects in children could have been investigated using this simple and readily measured parameter, such as: bone defects and fractures; lowered IQ; behavioral changes; attention deficit syndrome; lowered thyroid function; lowered melatonin levels and the earlier onset of puberty.

34 As far as adults are concerned there has been a surprising lack of interest in pursuing the possibility that fluoride exposure could cause or exacerbate lowered thyroid function, even though Australia has areas with outright or borderline iodine deficiency. Also surprising has been the absence of any interest in pursuing a possible relationship between the number of years lived in a fluoridated community and the onset of arthritis considering that a) fluoride accumulates in the bone and can damage both the bone and cartilage and b) the first indications of fluoride's damage to the bone and ligaments are identical to the first symptoms of arthritis, namely stiffness of joints and pain in both the bone and the joints (NRC, 2006).

35 Paragraph 18(a), of the affidavit of AB states:

> *"There is no evidence that Fluoridation at the Approved Concentration causes osteoporosis, arthritis or bone fractures. Indeed, both Osteoporosis Australia[11] and Arthritis Australia[12] support fluoridation."*

36 There are several things wrong or misleading about this statement.

a. Before drawing any conclusions from a statement citing "no evidence" one needs to establish that conscientious efforts have been made to actually study the issue (or issues) in question. The absence of studies should not be used to conclude the absence of harm. In the case of Australia no primary studies have been conducted in any Australian community on osteoporosis, arthritis or bone fractures. Instead, all conclusions have been drawn from reviews of the literature conducted elsewhere. As far as arthritis

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Date……..

is concerned no primary study of a possible relationship between arthritis and living in a fluoridated community has been published in any fluoridated country.

b. As far as the primary literature on a possible relationship between exposure to fluoride and osteoporosis, arthritis or bone fractures is concerned, it is misleading for AB to emphasize "at the Approved Concentration." As far as causing harm is concerned the key parameter is not the concentration of fluoride in water (mg/liter) but rather the daily dose of fluoride an individual receives (mg/day) which depends on two things a) how much water individuals drink and b) how much fluoride they get from other sources. Here is a simple example to illustrate this point: someone who drinks four liters of water per day at 1 ppm would get more fluoride (4 mg/day) than someone who drank one liter of water at 3 ppm (3 mg/day). Thus it is important to review the literature of effects on bone (and other tissues) for a whole range of concentrations. An important study that did just that was the study by Li et al. (2001) in which the authors looked at both total fracture rate and hip fracture rates in six Chinese villages with well water ranging from less than 1 ppm to 8 ppm. From this and other studies hip fractures in the elderly may increase at levels ranging from 6 to 14 mg/day over a lifetime of exposure, and may even occur at levels as low as 3 mg/day. The 3 mg/day would be exceeded by someone drinking over three liters of water at 1 ppm daily. Some miners in Australia drink very large quantities of water to fight dehydration. Some drink over twelve liters of water a day. So AB's reference to "Approved Concentration" is highly misleading. Engineers can control the concentration of fluoride that they add at the public water works, although there have been accidents, but they can't control the dose of fluoride people consume: this depends on how much water they drink (as well as fluoride they get from other sources).

c. Endorsements by bodies such as Osteoporosis Australia or Arthritis Australia have to be treated with caution. Endorsements can be effective promotional tools but they cannot be treated as scientific evidence unless the endorsing bodies have themselves carried out their own *primary* research on the matter and published their findings. Of lesser scientific significance are reviews of the literature carried out by the endorsing bodies.

Signature……..……..Witnessed by……..

Date……..

Such reviews have to be judged on the quality of the data they are reviewing. I am not aware of any primary studies carried out by Osteoporosis Australia or Arthritis Australia negating a possible relationship among Australians living in fluoridated communities and osteoporosis, arthritis or bone fractures. Or even reviews by these same bodies of the literature on these health conditions conducted either in Australia or elsewhere. AB cites none. He merely states that each of these bodies has endorsed fluoridation.

37 Endorsements by such associations have to be treated with especial caution in a country where government health agencies have a very strong bias towards fluoridation, and even promote it aggressively. Observers have to be particularly wary when these associations have received either political or economic support from the government health agencies in question.

38 All we are told about the endorsements of fluoridation by Osteoporosis Australia and Arthritis Australia are the dates on which the endorsements were made, which were Nov 25 and Nov 21, 2008, respectively. Thus it is not clear whether these endorsements were accompanied by primary studies or reviews of the literature, or whether these bodies simply added their names to a list of endorsements solicited by a pro-fluoridation government agency or some other fluoridation promoting body. Such endorsements by various health and dental associations has been a standard way that fluoridation has been promoted since the all-important first endorsement made by the US Public Health Service in 1950. When that endorsement was made none of the fluoridation trials had been completed and practically no health studies of any significance had been published. Nevertheless a series of endorsements from bodies like the ADA, APHA, AMA etc quickly followed. For a description of this and other tactics used in the promotion of fluoridation see chapters 10, 22 and 23 in the, "*The Case Against Fluoride*.

39 Paragraph 18(a) of the affidavit of AB states:

> *"Fluoride has even been used to treat osteoporosis".*[13]

AB cites only one study (Reference 13) in which fluoride tablets were used to treat osteoporosis. However, such a citation is highly selective and therefore misleading. Many such trials were conducted over the

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Date……..

period from the 1970s to the1990s, and the overall conclusion was that while fluoride could counteract the loss of bone mineral density caused by osteoporosis the effect varied with the type of bone (e.g. trabecular versus cortical). The results as far as reducing bone fractures were concerned– especially for the hip – were very disappointing. In some cases hip fractures actually increased with this treatment (Bayley et al., 1990; Dambacher et al., 1986; Gerster et al., 1983; Gutteridge et al.,2002;1990; Hedlund and Gallagher, 1989; Inkovaara, et al.,1975; O'Duffy et al., 1986; Orcel et al.,1990; Riggs et al.,1990; Schnitzler et al., 1990.)

40 The general conclusion is that while fluoride at high daily doses can harden bones it also makes them more brittle (see chapter 17 "*The Case Against Fluoride*: Paragraphs 18(b,c,d) of the affidavit of AB examines the issue of bone cancer, and particularly osteosarcoma.

41 Paragraph 18(b) of the affidavit of AB states:

> *"The international academic community has investigated the potential links between fluoridation and cancer. To date, no robust scientific data supports the contention that bone cancer is associated with fluoridation."*

To state that there is *"no robust scientific data supports the contention that bone cancer is associated with fluoridation"* is misleading in my view. It would be more accurate to say that there is some evidence – of varying robustness –of a relation between an increase in osteosarcoma in male rats exposed to fluoride (NTP, 1990) and young boys exposed to fluoridated water [(Hoover (1991 a); Cohn (1992); Takahasi, (2001) and Bassin (2001, 2006)] but other animal studies (Maurer, 1990) and epidemiological studies (Hrudey et al. 1990; Mahoney et al., 1991; McGuire et al., 1991; Freni and Gaylor, 1992; Gelberg et al., 1995 and Moss et al., 1995) have not found this relationship.

42 The connection between osteosarcoma and fluoridation is highly plausible from a biological point of view (NRC, 2006) but demonstrating the relationship for a rare cancer like this can be problematic. The one study that deserves and has received the greatest attention in recent years is the study by Elise Bassin at the Harvard dental school. Bassin used a novel approach (in a carefully matched case control study) of investigating the actual years when boys were

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Date……..

exposed to fluoridated water. She found a 5-7 fold increased risk of succumbing to osteosarcoma by the age of 20, when the boys were exposed in their 6th, 7th and 8th years (Bassin, 2001). No one had previously (or since) investigated whether there might be a window of vulnerability when fluoride strikes bones (although Perry Cohn in his 1992 study had suggested this possibility). The 6th,7th and 8th years coincide with the boys' mid-childhood growth spurt. At this time the bones would be turning over very fast and the genetic material is more subject to damage during such periods of rapid turnover.

43 Bassin's study was published in 2001 as part of her doctoral thesis at Harvard (Bassin, 2001). Strangely her thesis adviser Professor Chester Douglass had several opportunities to discuss Bassin's findings with the public, with his peers and with his funders over the next few years but failed to do so even while proclaiming that his own work showed that there was no relationship with osteosarcoma. This is no trivial matter. If Bassin was correct then fluoridation could actually be killing some young men (osteosarcoma is frequently fatal).

44 Eventually, Bassin's doctoral thesis was found in the rare books section of one of the Harvard libraries and eventually was published in the journal "*Cancer Causes and Control*" (Bassin et al., 2006). The same issue of this journal carried a letter from Douglass claiming that Bassin's findings might be premature and his larger study – of which Bassin's study was a small subset –found no relationship with osteosarcoma (Joshipura and Douglass, 2006). Douglass promised his study for the summer of 2006, but it still has not appeared. It is now nearly 5 years overdue. Meanwhile, those who have examined Douglass's methodology have indicated that it cannot discount Bassin's thesis since his biometric of exposure – fluoride bone levels at biopsy or autopsy –being bio-accumulative cannot identify the fluoride exposure during the critical 6th, 7th and 8th years that are central to Bassin's thesis (Neurath and Connett, 2008).

45 Paragraph 18(c.C.l) of the affidavit of AB addresses these important events:

> In 2006, Bassin et al. published a paper linking osteosarcoma (a type of bone cancer) in men to water fluoridation.[19] However, the senior researcher in charge of the study (Douglass) was not an author on the paper and he expressed grave reservations about the scientific veracity of the

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Date……..

paper. He stated that the full data set did not support any association between cancer and fluoridation.[20]

46 In my view, this summary does little justice to the issue. Nor does it indicate that Chester Douglass may not be the most appropriate adjudicator of this issue. In a previous paper he co-authored on this subject (McGuire et al., 1991) that was published in the "*Journal of the American Dental Association",* the authors make it very clear how disastrous such a positive relationship with osteosarcoma, were it found, would be for the water fluoridation program. Here are excerpts from their paper:

> *"An incorrect inference implicating systemic fluoride carcinogenicity and its removal from our water systems would be detrimental to the oral health of most Americans, particularly those who cannot afford to pay for increasingly expensive restorative dental care . . .Because of its strengthening action, fluoride has been widely accepted as the responsible agent for the dramatic declines in the tooth decay rates of U.S. children and adolescents . . . A disruption in the delivery of fluoride through municipal water systems would increase decay rates over time . . . Linking of fluoride ingestion and cancer initiation could result in a large-scale defluoridation of municipal water systems under the Delaney clause." (McGuire et al., 1991)*

47 Luckily for those who believed in the fluoridation program, the authors of this 1991 study did not find that fluoridation was associated with an increase in osteosarcoma. In fact, they found that the very opposite might be the case, stating that *"fluoridation at recommended levels may provide a protective effect against* the *formation of osteosarcoma."* This speculative finding allowed the authors to reach the conclusion that they (and the JADA editors and the ADA) clearly wanted out of this study: *"Given present knowledge, every effort should be made to continue the practice* of *fluoridating community water supplies."* (McGuire et al., 1991)

48 It is interesting to note that several governments promoting fluoridation and their agencies (e.g. Health Canada, 2007, 2008 and the NHMRC, 2007) have also used Douglass's promise of a study, which has been neither peer-reviewed nor published, to negate or obfuscate Bassin's important finding, and in so doing have willfully ignored Douglass's bias on this matter. In addition to being a prominent promoter of fluoridation he is also a consultant for Colgate,

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Date……..

which has huge liabilities should exposure to fluoride cause osteosarcoma. Such behavior smacks more of a relentless effort to keep fluoridation program going at all costs, rather than a precautionary approach to protecting the health of the people. I examine this issue more carefully in chapter 18 of my book "*The Case Against Fluoride*."

49 Paragraph 18(d) of the affidavit of AB states:

> *"The NHMRC concluded that "there is no clear association between water fluoridation and overall cancer incidence or mortality. " "*[21]

50 The NHMRC comment here is contingent on denying the validity of the Bassin study. It should be noted that this statement comes after the NHMRC has used the promise of the Douglass study to discount the Bassin findings. The NHMRC states:

> *"The attention of the reader is drawn to a Letter to the Editor that appeared in the same issue of Cancer Causes and Controls by co-investigators on the larger Harvard study (Douglass & Joshipura, 2006). The authors point out that they had not been able to replicate the findings of Bassin and colleagues in the larger study that included prospective cases from the same 11 hospitals. Furthermore, the bone samples that were taken in the broader study corroborate a lack of association between the fluoride content in drinking water and osteosarcoma in the new cases. As Bassin and colleagues acknowledged, the shortcomings of their study mean that their results should be interpreted with caution pending publication of the larger study results." (NHMRC, 2007)*

51 There are several problems with these NHMRC conclusions. When the NHMRC states that Douglass was unable to replicate Bassin's findings in the larger cohort, they presented no evidence that Douglass had actually attempted to do so. For this Douglass would have had to calculate the risks involved to the whole cohort for exposure in each year including the critical 6th, 7th and 8th years. If they had done so the controversy could have been easily resolved by publishing a table of their results for the whole cohort. In five years such a table has not been published. Instead of doing this they have appeared to have taken a different tack of estimating cumulative fluoride bones levels at diagnosis or autopsy (Neurath and Connett, 2008). These cumulative measures while interesting cannot test Bassin's thesis. It is

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Date……..

surprising that the NHMRC did not recognize this inadequacy in Douglass's methodology before using it to conclude that *"there is no clear association between water fluoridation and overall cancer incidence or mortality."* If the association is unclear it is because Douglass has successfully been able to muddy the waters. Only he can unmuddy them, but after 5 years he has failed to do so. Meanwhile, Douglass's delay has served the purpose for those who wish to maintain that the relationship between fluoridation and both osteosarcoma remains *"unclear."*

52 Paragraph 18(e) of the affidavit of AB discusses "cancer in general." He states:

> *"The York Report concluded that there is no consistent evidence of an association between water fluoridation and morbidity/mortality due to cancer.[22] This conclusion is supported by a paper published in 2008 by Stewert (sic) [23] which stated that there is a significant body of evidence demonstrating that the consumption of optimally fluoridated water is not associated with a carcinogenic risk."*

53 Citing the York Review (McDonagh et al, 2000) here is hardly helpful since it was published 6 years before Bassin's study (Bassin et al., 2006). Citing the Stewart (2008) paper is also strange since it is not a specific review of fluoride's carcinogenic potential but rather deals with a procedural basis for qualitative cancer assessment. Fluoride is only mentioned once in this 26-page paper; it is mentioned in table 6. In a single line entry in this table for *"Drinking Fluoridated Water"* Stewart cites two references (270, 271) to support his conclusion that for *"bone cancer"* there is a *"marked body of evidence indicating lack of risk".* References 270 and 271, are studies by Takahashi et al. (2001) and Bassin et al. (2006), for which both authors published evidence for an association between drinking fluoridated water and bone cancer! One has to look into the references cited by Bassin to find epidemiological studies that have not found a relationship, but none of these examined the issue as a function of the years in which the patients were exposed to fluoride. Nor were these studies cited by Stewart.

54 Paragraph 18(f) of the affidavit of AB cites an opinion from a paper by Packham (1990) on which he comments in 18(g):

> *f) "From a global point of view our priorities may seem rather strange and a visiting Martian could be forgiven for asking why it is that on one part of the planet so much effort is devoted to the elimination of an*

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Date……..

unproven link between water quality and cancer; whilst elsewhere tens of thousands die daily from eradicable water borne diseases."

g) "The fact that water fluoridation continues to attract criticism in terms of its carcinogenic potential despite over 50 years of safe usage is a clear testimony to the concerns raised by Peckham that true water borne problems are being ignored whilst others which are not supported by mainstream science are continually reinvestigated."

55 This seems a pointless comparison since fluoridation does not compete for funds for eradicating waterborne diseases in developing countries. If it did so, it would be an argument for discontinuing fluoridation in favor of using the money in a more worthwhile cause.

56 Paragraph 20(c) of the affidavit of AB states:

"The NHMRC Nutrient Reference Value for Australia and New Zealand (2006) identifies 10 litres/day as the upper limit for consumption of fluoridated water for an average sized adult over the long term."

57 In consulting this document it appears that the NHMRC has derived this figure from the upper tolerance limit (UL) derived by the Food and Nutrition Board of the US Institute of Medicine (IOM, 1997). This was established (with considerable criticism from independent observers) as 10 mg/day for adults and children of 9 years of age and above. Only one end point was considered for this determination: skeletal fluorosis. However, despite the very poor and dated database used in this determination, the IOM applied an *"uncertainty factor"* of 1 to this value. This is quite extraordinary. Usually when extrapolating from a LOAEL (lowest observable adverse effect level) an uncertainty factor of 10 is applied to take into account the full variation of human susceptibility to any health end point expected in the whole population. This factor of 10 is sometimes referred to as protecting against *"intra-species variation."* If this safety factor was used the safe level to protect all citizens – young and old – from bone damage caused by accumulated fluoride would be 1 mg/day. Such a value would end water fluoridation forthwith.

58 This problem is compounded by the fact that the first symptoms of skeletal fluorosis are very similar to arthritis (stiff joints, aching joints and bones, NRC, 2006) for which there have been large

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Date……..

increases in the Australian and US populations over recent years. No attempt has been made to see if such increases are related to a lifetime's exposure to fluoridated water. It is incomprehensible then that the normal safety factor of 10 (or any safety factor) is jettisoned on the basis of the extremely limited database available.

59 However, if AB is right that someone drinking 10 liters of water a day will exceed this UL, then his argument about safety collapses because I am reliably informed by an executive in the mining industry in Western Australia that workers in this industry drink 10-12 liters of water a day.

60 This argument rests entirely on assuming the validity of there being only end point other than dental fluorosis, namely skeletal fluorosis. There are far more worrying end points that haven't even been discussed by AB even though they were brought up in the affidavits of Dr. Andrew Harms, Dr. Mark Diesendorf and Dr. John Ryan. These include fluoride's impact on the brain.

61 There have now been over 100 experiments indicating fluoride can damage animal brain and 24 human studies that indicate an association between the lowering of IQ in children and moderate exposures to fluoride in areas endemic for fluorosis in India, China, Iran and Mexico. (Ren et al., 1989 (2008); Qin et al., 1990 (2008); Lin, et al., 1991; Chen, et al., 1991 (2008); Guo, et al., 1991 (2008); An et al., 1992 (Chinese); Xu et al., 1994(Chinese); Li et al., 1995; Zhao, et al.,1996; Wang, et al., 1996 (2008); Yao et al., 1997 (Chinese); Zhang et al., 1998 (Chinese); Liu et al., 2000 (2008); Lu et al., 2000), Hong et al., 2001 (2008); Xiang et al., 2003a,b; Wang et al, 2005 (2008); Rocha-Amador et al.,2007; Seraj et al., 2007; Trivedi, et al., 2007; Wang et al., 2007; Fan et al.,2007 (Chinese); Li et al., 2008; Ding et al., 2011. Note: the second date in parenthesis indicates the date of the publication in English.

62 One study that I have pursued in some depth was the study by Xiang et al. (2003a,b). They found a lowering of IQ of 5-10 IQ points between the village with high fluoride levels (2.5-4.5 ppm fluoride) and the low fluoride village (<0.7 ppm). They controlled for many factors including lead exposure and iodine intake. In 2006, I visited the two villages where this research was carried out and later helped arrange a visit to the US by Xiang in order that he could share his

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Date……..

work with scientists there. Xiang estimated that the threshold for lowering IQ in this study was 1.9 ppm. I don't think there can be any question that the methodology was reasonable because an updated version of this study was peer reviewed and accepted for publication by *Environmental Health Perspectives,* the journal of the US National Institute of Environmental Health Sciences (NIEHS), which is part of the US Department of Health and Human Services (DHHS) which endorses fluoridation. A pre-publication copy appeared online in December 2010 (a pdf copy of Xiang's 2010 paper is attached in **Annexure 5**). Sadly this Xiang article was withdrawn when it became clear that some of the work, even though updated, had been published before - but it should be emphasized that it was not withdrawn because there was any weakness in the methodology.

63 If the routine calculations were performed on this data that usually go into risk assessments by government agencies it should become clear that there is absolutely no margin of safety to protect all children from this serious end point. This is how it would be done.

64 Let's assume that the children in this study were drinking one liter of water - then the threshold for IQ lowering was 1.9 mg/day. However, there were only about 300 children involved in the study group and we can't assume that the whole variation in sensitivity has been covered in a population like Australia or the US. As we have to protect millions of children we would normally apply the *"intra-species variation"* safety factor of 10 (as discussed above). So a safe level to protect every child (not just the average child) from fluoride's potential to interfere with mental development would be 0.19 mg/day. This would be exceeded by a child drinking less than one glass of water at 1 ppm!

65 Meanwhile, another study has just been published which adds further weight to this concern. In the *Journal of Hazardous Materials* Ding et al. (2011) found that lowering of IQ occurred within the range of 0.3 - 3 ppm (a pdf of this paper is attached in **Annexure 6**). They showed that there was a lowering of IQ of 0.59 points associated with each increase of 1 mg/liter of fluoride excreted in the urine of these children.

66 Paragraph 20(d), of the affidavit of AB states:

Signature……..……..Witnessed by……..

Date……..

"Health professionals have monitored the safety and effectiveness in drinking water for over half a century."

67 As indicated above health professionals have not monitored the safety of water fluoridation in Australia to any significant or comprehensive extent. The absence of study should not be read as the absence of harm. If AB is referring to monitoring of the safety of water fluoridation in other fluoridating countries, this is also misleading since there has been a serious dearth of primary health studies in *any* fluoridating country.

68 Paragraph 20(d) of the affidavit of AB continues

"In that time no ill effects on the general health of adults or children have been found."

69 Again, the absence of study should not be used to indicate or imply an absence of harm. More importantly, what is needed, in the absence of fluoridating countries conducting their own studies in artificially fluoridated communities, is a careful examination of health studies performed in countries like India and China that have experienced many health effects (especially bone damage and brain damage), in areas with moderate to high natural levels of fluoride in their water. The key determination then required is to see if there is an adequate margin of safety between the doses that are known to cause harm and the doses likely to be experienced in artificially fluoridated communities, sufficient to protect the whole population from harm. To date the largest review that has examined this worldwide database has been the 507-page review by the US National Research Council, "Fluoride in Drinking Water: An Examination of EPA's Standards" (NRC, 2006). Incredibly, this review has been largely ignored by Australian authorities and the health authorities in other fluoridating countries. The NHMRC (2007) dismissed its relevance to Australia in one sentence!

70 Paragraph 20(d) of the affidavit of AB continues

"The MD Affidavit implies that the medical and dental professionals are relying on personal opinion rather than science when supporting water fluoridation. This is certainly not the case as there is overwhelming evidence in peer reviewed papers that fluoridation is safe for all the population.[29]"

71 Reference 29 refers back to reference 14. Reference 14 is the York Review (National Health Service Centre for Systematic Reviews and

Signature……..……..Witnessed by……..

Date……..

Dissemination. *"A systematic review of public water fluoridation", "York Review"*. University of York, UK, 2000).

72 AB's claim that the York Review provided *"overwhelming evidence in peer-reviewed papers that fluoridation is safe for all the population"* is in sharp contrast to the conclusions of Professor Trevor Sheldon, who was the chairman of the advisory committee for the York review (McDonagh et al., 2000). In an open letter (which was subsequently read to the House of Lords) dated 3/1/2001, he stated:

> *"In my capacity of chair of the Advisory Group for the systematic review on the effects of water fluoridation recently conducted by the NHS Centre for Reviews and Dissemination the University of York and as its founding director, I am concerned that the results of the review have been widely misrepresented. The review was exceptional in this field in that it was conducted by an independent group to the highest international scientific standards and a summary has been published in the British Medical Journal. It is particularly worrying then that statements which mislead the public about the review's findings have been made in press releases and briefings by the British Dental Association, the British Medical Association, the National Alliance for Equity in Dental Health and the British Fluoridation Society. I should like to correct some of these errors." (Sheldon, 2001)*

73 **Two of the seven errors Sheldon attempted to correct, were:**

> **"3** *The review did not show water fluoridation to be safe. The quality of the research was too poor to establish with confidence whether or not there are potentially important adverse effects in addition to the high levels of fluorosis. The report recommended that more research was needed.*
>
> **"7** *The review team was surprised that in spite of the large number of studies carried out over several decades there is a dearth of reliable evidence with which to inform policy. Until high quality studies are undertaken providing more definite evidence, there will continue to be legitimate scientific controversy over the likely effects and costs of water fluoridation." (Sheldon, 2001) (A copy of the full letter is appended at* ***Annexure 7)***

74 Similar sentiments were echoed by Professor John Doull,

Signature……..……..Witnessed by……

Date……

the chairman of the important review by the National Research Council of 2006, in an interview he gave for a *Scientific American* article published in Jan 2008:

> *"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. I mean, when the U.S. surgeon general comes out and says this is one of the top 10 greatest achievements of the 20th century, that's a hard hurdle to get over. 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." (John Doull in Fagin, Scientific American, 2008)*

75 In my view, AB's use of the phrase *"there is overwhelming evidence in peer reviewed papers that fluoridation is safe for all the population"* is quite preposterous. On the one hand many key studies have not been performed in fluoridating countries (and that is especially so in Australia) and on the other, where studies have been done in countries that have high natural levels of fluoride, no attempt is being made to replicate them in fluoridating countries. Instead, most of the effort in fluoridating countries has gone into discrediting any study that indicates harm.

76 Here are a few of the key studies that have not been pursued in Australia:

An investigation into the possible relationship between consuming fluoridated water and:

a. lowered IQ in children;

b. bone defects and bone damage in children;

c. lowered thyroid function;

d. earlier onset of puberty;

e. melatonin levels in children and adults;

f. arthritis in adults;

g. hip fractures in adults;

Signature……..……..Witnessed by……

Date……..

77 Nor has any formal investigation taken place of the many anecdotal reports that some individuals are very sensitive to fluoride's toxic effects, even when the NHMRC (1991) panel recommended that they be carried out and individuals have offered themselves for such testing.

78 Nor have any attempts been made to assess fluoride levels in people's urine, plasma, hair or bone to gauge both short-term and long-term exposures to fluoride in citizens living in fluoridated communities for various lengths of time.

79 Nor has the non-invasive bio-marker of exposure to fluoride of the severity of dental fluorosis (prior to the eruption of the secondary teeth) been used to support epidemiological studies that could be undertaken to investigate a whole range of childhood concerns that might be related to fluoride exposure.

80 The lack of such studies on *any* public health matter would be unacceptable, but when that public health policy is being forced on communities without their consent it is doubly inexcusable.

81 I will now turn to the evidence of the benefits of fluoridation as articulated in AB's affidavit.

82 Paragraph 23(a) of the affidavit of AB states:

> *"The early studies into fluoridation were undertaken in a scientific manner and the effects of water fluoridation were so great that we can place a great deal of confidence in these early studies."*

83 The late Dr. Philip Sutton wrote a whole book devoted to analyzing these early studies and found the methodology used was very poor. Having read this book by Sutton I am inclined to give it more credence than the assertion offered by AB. Were an independent panel of scientists asked to do the same I think they too would side with Sutton (Sutton, 1998).

84 Paragraph 23(b) of the affidavit of AB states:

> *"...taking into account the huge number of studies which consistently showed a benefit from fluoridation, the York Report did accept that the evidence for the beneficial effects of fluoridation were*

Signature……..……..Witnessed by……

Date……..

overwhelming."

85 Professor Trevor Sheldon, the chairman of the York Review advisory panel, gives a more sober assessment of the York Review's findings in the letter I have already cited above. In point 1 of his letter he writes:

> ***"1*** *Whilst there is evidence that water fluoridation is effective at reducing caries, the quality of the studies was generally moderate and the size of the estimated benefit, only of the order of 15%, is far from 'massive'". (Sheldon, 2001)*

86 Paragraph 23(c) of the affidavit of AB claims:

> *"Fluoridation has been investigated in hundreds of research studies and the general comment that the population selection is unscientific is without any scientific foundation. The majority of studies published over the last 60 years have been scientifically rigorous."*

87 In actual fact, the York Review panel could find no grade A level studies and had to work with grade B level and grade C level studies. Specifically the York Review panel stated:

> *"No study used an analysis that would control for the frequency of sugar consumption or the number of erupted teeth per child" (York, p.24)*

88 The longitudinal studies reviewed by the York panel were published over a period of about 40 years. The savings ranged from -5% to plus 60% and the average saving was 15%. When this was translated into DMFT (Decayed Missing and Filled Teeth) it amounted to a saving of 2.25 DMFT. However, such figures (averages) derived over a *changing baseline* are questionable. Today WHO data indicates that 12 year olds have much less than 2.25 DMFT in numerous NON-fluoridated countries so it is difficult to find a physical reality to match the York review's findings here.

Signature……..……..Witnessed by……..

Date……..

89 Chapters 6-8 *The Case Against Fluoride* summarize the very weak evidence of benefit from water fluoridation (i.e. swallowing fluoride as opposed to topical applications). See **Annexure 4.**

90 Paragraph (26) AB does what a number of fluoridation promoters have done over the past 60 years of this practice and that is attempt to discredit the scientific credibility of fluoridation opponents. Citing Armfield's intemperate commentary from 2007 without citing my responses to that commentary is particularly egregious and prejudicial to the court in my view. My responses to Armfield's commentary appear in **Annexure 8.** Suffice it to say here that for someone who chastises the opponents of fluoridation as behaving unscientifically, the derogatory rhetoric Armfield applies to all opponents of fluoridation is hardly the behavior one has the right to expect of a scientist.

Affidavit of Dr Fredrick Allan Clive Wright

91 Paragraph 13(v) of the affidavit of Wright states:

> *"Water fluoridation has been practiced internationally for over 60 years, and in Australia and NSW for over 55 years. During this time, the safety and efficacy of water fluoridation has been re-evaluated many times. The most recent review and reaffirmation of water fluoridation from the peak Australian health authority, the National Health and Medical Research Council (NHMRC), was in 2007."*[4]

92 FW is correct in stating that the NHMRC has purportedly reviewed the safety and effectiveness of water fluoridation several times, most recently in 2007. However, as far as safety is concerned the fact is that in 55 years of fluoridation there have been virtually no primary health studies conducted in Australia. I discussed the absence of primary health studies in my responses to AB's affidavit above. Please see **Annexure 9** for my critique of the gross inadequacies of the NHMRC (2007) review.

93 Paragraph 13(vi) of the affidavit of FW states:

> *"Residents of metropolitan Sydney NSW have received fluoridated water at the Approved Concentration since 1968. There has been no*

Signature……..……..Witnessed by……..

Date……..

reported public health or environmental concerns regarding its impacts on residents or their environment in that 40 year period."

94 This comment is disingenuous since FW failed to disclose the fact that in the last 40 years no primary *"public health or environmental"* studies have been published in NSW by either government agencies or independent researchers. Paragraph 13(vii) of the affidavit of FW states:

"This public health measure has been exhaustively researched; more so than most other public health measures. Any new argument raised by opponents of fluoridation is investigated by NSW Health."

95 I refute both these claims. In my opinion, governments promoting and endorsing fluoridation from time to time conduct reviews of the literature (largely from overseas) for the suspected purpose of seeking continued endorsement of their fluoridation policy.

96 There have been two major exceptions to this matter in recent years. The York Review (McDonagh et al., 2000) and the US National Research Council review of 2006, *"Fluoride in Drinking Water: An examination of the EPA's Drinking Water Standards"* (NRC, 2006). The organizers of these reviews took steps to ensure as independent an enquiry as they could manage. Both panels drew attention to the fact that there is a very poor database from which to assert the safety of water fluoridation with any degree of confidence (See Trevor Sheldon's commentary of the York review in **Annexure 7** attached to this affidavit and Dr. John Doull's comments also cited above).

97 Key studies on many important health end points have simply not been attempted in fluoridating countries, particularly in Australia. The key studies not undertaken are summarized in my response to AB's affidavit above and in my critique of the NHMRC (2007) review in **Annexure 9** attached to this affidavit.

98 It is false for FW to claim that NSW Health responds to *"Any new argument raised by opponents of fluoridation."* NSW Health have not responded to the more than 20 studies that have indicated an association with lowered IQ in children and moderate exposure to fluoride in areas of the world endemic for fluorosis. NSW Health have not undertaken any health studies in relation to the possible association between fluoridation and arthritis in adults; hip fractures in

Signature……..……..Witnessed by……

Date……..

the elderly; lowered thyroid function; accumulation in the human pineal gland and reports of individual sensitivity to fluoride's toxicity and experience symptoms which disappear when the source of the fluoride is removed. I have seen no formal response to this literature by NSW Health or any willingness to conduct any primary studies of its own to investigate these concerns.

99 In support of paragraph 13 (viii), FW does not produce any primary study demonstrating or even investigating the safety of fluoridation in Australia. FW merely asserts a long list of endorsing agencies but this in no way can be regarded to constitute scientific evidence. The first major endorsement for water fluoridation came from the US Public health Service in 1950 before a single trial for effectiveness had been completed and before any significant health studies had been published. This endorsement was not science-based and the vast majority of the endorsements that followed were also not science-based but were based upon this US PHS endorsement.

100 The agencies, research groups or expert panels identified by FW at paragraph 13(b)(i-xiii) did not investigate nor monitor any issue other than fluoride's effect on teeth. The single focus appears to be on reducing tooth decay, with little or no attention being paid to any need to conduct primary studies on fluoride's impact on human health.

101 Paragraph 18 of FW comments on the prevalence of dental fluorosis in Australia failed to contain any tables identifying the different dental fluorosis rates by state, age, year surveyed and level of severity. The recognized impact of dental fluorosis does not appear to have been investigated in Australia in a systematic fashion.

102 Paragraph 20(c) of the affidavit of FW deposes that:

> *"During the development of a tooth, fluoride is incorporated in the developing enamel and into the crystal structure making the enamel more resistant to acid dissolution. This appears to be especially important in primary (baby) teeth."*

103 FW assertion is contradicted by the analysis presented by the US Centers for Disease Control and prevention (CDC) in 1999, where the authors state:

> *"Fluoride's caries-preventive properties initially were attributed to changes in enamel during tooth development because of the*

Signature……..……..Witnessed by……..

Date……..

association between fluoride and cosmetic changes in enamel and a belief that fluoride incorporated into enamel during tooth development would result in a more acid-resistant mineral. However, laboratory and epidemiologic research suggests that fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children (1)". (CDC, 1999)

104 In my view, this admission by the CDC – the primary promoter of fluoridation in the US – should have signaled the end of water fluoridation. If indeed the primary mode of action is TOPICAL why expose the whole body to this toxic substance through ingestion via water and why force it on people who don't want it, when topical treatments are readily and universally available in the form of fluoridated toothpaste? If the primary concern is children of low-income families then a more rational approach than the wasteful practice of water fluoridation (where most of the chemical gets flushed down the drain and goes nowhere near the teeth) would be to provide free toothbrushes and free toothpaste to these families.

105 Paragraph 20(e) of the affidavit of FW states that:

"The relative beneficial effects of pre- and post-eruptive exposure have been studied in Australian children: see Singh et al (2003)[34] and Singh and Spencer (2004)[35]. This research reported that exposure to fluoride before eruption reduced caries levels significantly, and that the benefits were enhanced with ongoing exposure to fluoride after tooth eruption."

106 Such studies might have been more convincing if the authors had controlled for delayed eruption of the teeth that is thought to be caused by fluoride ingestion (Komarek, et al., 2005). Without this control erroneous results can be observed. Fewer teeth erupted (in fluoridated communities) would mean less total tooth decay for the same age as the children in the non-fluoridated communities.

107 A study by Peiris et al., 2009, indicates that the dental age of children in Australia is 0.82 years later than UK children. As the UK is only has 10% of the population drinking fluoridated water, this 0.82 year delay may reflect a delayed eruption of teeth by fluoride. This is, of course, not conclusive but it is plausible. A delay of 0.82 years in tooth eruption would eliminate any of the so-called benefits in permanent teeth attributed to fluoridation found in

Signature……..……..Witnessed by……

Date……..

Australian studies.

108 Paragraph 22(a) of the affidavit of FW claims:

> *"In 2008, the ARCPOH (AUSTRLIAN RESEARCH CENTRE POPULATION ORAL HEALTH) reported research which examined the effectiveness of water fluoridation on children's dental health across four Australian States: Queensland; Victoria; Tasmania; and South Australia.[37] The study also considered, and controlled for, a number of additional factors associated with variation of dental caries. These factors included: individual tooth brushing history; use of other fluoride products; water and food consumption; use of infant formula; and socioeconomic status."*

109 The examination of data for each age range compared with the frequency of tooth decay for each state, indicated that for *no age range* or *all ages combined* was the tooth decay worse in unfluoridated Queensland, than in at least one of the fluoridated states (see Figures 1-6 below).

110 Paragraphs 22(b-e) FW affidavit purportedly provides evidence for the difference in tooth decay rates between fluoridated and non-fluoridated communities in Australia and elsewhere. I refute this assertion and rely on the evidence in **Annexure 4 (see chapters 6-8).** According to WHO figures there appears to no difference in tooth decay among 12-year-olds between fluoridated and non-fluoridated countries. In general, there is far stronger inverse relationship between tooth decay and income levels than there is with fluoridation status. Erroneous conclusions can be drawn when comparing two communities unless key confounding factors are carefully controlled. These include to the availability of local dental services and interventions, diet, sugar consumption, ethnicity and the potential of fluoride to delay the eruption of the teeth (Komarek et al., 2005).

111 Modern studies and reviews by Leverett (1982); Diesendorf (1986); Colquhoun (1984, 1985 and 1987); Gray (1987); Yiamouyiannis (1990); Brunelle and Carlos (1990); Spencer et al., (1996); de Liefde (1998); Locker (1999); Spencer and Armfield (2004); Komarek (2005) and Pizzo (2007) have reported little – if any- difference in tooth decay in the permanent teeth between fluoridated and non-fluoridated communities. Only Komarek (2005) adjusted for possible delayed eruption of the teeth and in doing so found no difference in permanent tooth decay between children taking fluoride supplements and those not taking supplements. Warren et al., 2009 was one of the few studies that looked at tooth decay as function of individual exposure to

Signature……..……..Witnessed by……

Date……..

fluoride as opposed to living in a fluoridated or non-fluoridated community). They found no relationship between tooth decay and individual exposure to fluoride. See more discussion on these studies in **Annexure 4, chapter 6-8.**

112 Average differences in the permanent teeth ranged from results which de Liefde (1998) described as "clinically meaningless" to a 0.6 of one tooth surface saved in the largest survey ever conducted in the US (Brunelle and Carlos, 1990). Spencer et al., (1996) found even less of a saving in permanent tooth surfaces in two states in Australia, 0.12-0.3 tooth surfaces respectively. In South Australia, Armfield and Spencer (2004) reported no significant difference in the permanent teeth between children who drank bottled water or tank water all their lives and those that drank fluoridated tap water.

113 In my view, these very small or no savings in tooth decay, cannot justify the health risks being taken in fluoridated communities. This is particularly so when one considers that there have now been 24 studies associating lowered IQ with modest exposure to fluoride (see paragraphs 61-63 above).

114 Paragraph 24(c) of the affidavit of FW states:

> *"The alternate gold standard for public health interventions are systematic reviews of population health studies and outcomes weighted by hierarchy of evidence and benefit of outcomes. There have been several recent systematic reviews on water fluoridation conducted in the USA, Australia, the United Kingdom and Ireland by authoritative bodies. Both individually and collectively, these systematic reviews provide substantial evidence of the effectiveness of water fluoridation under modern conditions: see NHMRC (2007), McDonagh et al (2000), Government of Ireland (2002) and Truman et al (2002)"[47].*

115 The list of reviews sited by FW excludes the most important review to date: namely, the review by the National Research Council (NRC, 2006) that I have discussed above.

116 In my opinion the report from the Fluoridation Forum in Ireland would be one of the shoddiest examples of government sponsored reviews undertaken on fluoridation of public water. I testified before this panel. The final review only identified one health effect (bone fractures) and provided only cursory comments on three primary studies. The forum completely ignored the hip fracture study I spent considerable time discussing with them. This study by Li et al. (2001) has been relied upon subsequently by the NRC (2006) and the WHO (2003) and is discussed in some

Signature……..……..Witnessed by……

Date……..

detail in chapter 17, "*The Case Against Fluoride*."

117 Paragraph 25(b) of the affidavit of FW states:

> *"In Australia, the systematic review conducted by the NHMRC in 2007,[51] not only assessed all of the studies examining the effectiveness of water fluoridation, but also those investigating its safety. With the exception of dental fluorosis (discussed in more detail in response to "Assertion 1" above), scientific studies have not found any credible link between water fluoridation at 1 ppm and adverse health effects."*

118 As I have stated the absence of studies does not mean the absence of harm. Key studies have not been done in Australia, and the NHMRC (2007) panel failed to properly review international studies on effects of fluoride on the brain, the thyroid and the pineal gland. Brain studies carried out by Xiang et al. (2003 a,b) found a lowering of IQ at 1.9 ppm. This offers no margin of safety from this end point for children drinking fluoridated water at 1 ppm (see paragraph 62)

119 Although the Xiang finding was not made at 1 ppm, that is merely a argumentative difference not a real or scientific one. A child drinking two liters of water a day at 1 ppm would get a higher dose of fluoride (2 mg/day) than a child drinking one liter of water a day at 1.9 ppm (1.9 mg/day). The NHMRC (2007) completely failed to give regard to the critical difference between concentration and dose, perpetuating a self-serving obfuscation that has continued for over 60 years in the promotion of this practice.

120 When the normal margin of safety factor is applied to the doses where a number of health effects have been observed, safety cannot be guaranteed for the whole population drinking uncontrolled amounts of fluoridated water. See the discussion on applying such a safety factor to the finding by Xiang et al., (2003a,b) of a lowering of IQ at 1.9 ppm in paragraph 66 above. The NHMRC (2007) review did not attempt a *"margin of safety"* analysis for any health end point.

121 Paragraph 26(e) FW repeats the arguments that AB used to counteract the possibility that fluoridation maybe contributing to arthritis and bone disease, by citing endorsements and comments from Arthritis Australia and Osteoporosis Australia. These endorsements were made without review of the literature and are not based on any primary study. Nor do they compensate for

Signature……..……..Witnessed by……

Date……..

the fact that no primary studies on these health concerns have ever been conducted in Australia.

122 In relation to kidney patients, who were identified in the Australian Drinking Water Guidelines 2004 as being more vulnerable to fluoride's toxicity because of their reduced ability to eliminate fluoride in the urine, statements from Kidney Health Australia (KHA) quoted in paragraph 26 (f)&(g) in FW's affidavit, should be disregarded as the KHA appears to have never published or sponsored any research on this issue in Australia.

123 Paragraphs 28(a-f) FW ignores the fact that 99% of water that is fluoridated is neither drunk nor goes any where near the teeth. This could only be considered *"cost effective"* because the fluoridating chemicals used are not pharmaceutical grade chemicals used in dental products but cheap industrial grade waste products from phosphate fertilizer companies.

Dr Anthony Maitland Brown's Affidavit

124 AMB is correct in his assertion Paragraph 14(c) that

> *" as with all toxic effects, dose is extremely important in considering effects of fluorides. Effects that occur at high doses may not occur at much lower doses."*

125 Consideration of high or low doses to be needs to be quantified using a margin of safety analysis, which was not done in "*A Systematic Review of the Efficacy and Safety of Fluoridation*" NHMRC (2007). In my opinion what is required is a demonstration that there is a sufficient margin of safety between the dose that have been shown to cause harm and the full range of doses that will be experienced by the whole population. That gap should also be sufficient to protect everyone across the full range of vulnerability to fluoride expected in any human population. For any toxic substance that range of vulnerability is expected to be at least ten-fold i.e, that some individuals will be 10 times more susceptible than others.

126 Paragraph 14(d) of the affidavit of AMB discusses the lethal dose of fluoride. This is not relevant to water fluoridation. No serious critics

Signature……..……..Witnessed by……

Date……..

of water fluoridation oppose the practice because they believe that drinking fluoridated water will deliver a one time lethal dose. It is the long term chronic effects that are of concern.

127 Paragraph 17, of the affidavit of AMB states:

> *"The NAS was tasked to review the United States Environment Protection Authority's (EPA) standard on fluoride in drinking water. Specifically, the NAS reviewed whether the EPA's current Maximum Contaminant Level Goal of 4 mg/L was adequate to protect children and others from adverse health effects. This is a maximum standard and represents a high concentration of fluoride. The NAS specifically did not consider artificial fluoridation at levels close to the Approved Concentration. Indeed, the NAS expressly states in its report that "(a)dressing questions of artificial fluoridation, economics, risk-benefit assessment, and water treatment technology was not part of the committee's charge."*

128 AMB in his affidavit is absolutely correct in this statement. However, the quote taken from the CDC and reproduced by AMB Paragraph 18 is highly misleading. The CDC has issued a statement in relation to the NAS Paper. In that statement, the CDC notes that the NAS Paper "*addresses the safety of high levels of fluoride in water that occur naturally, and does not question the use of lower levels of fluoride to prevent tooth decay.*"

129 While the NRC does not question the use of water fluoridation (as AMB made clear above, this was not its task) there is much data presented in this 507-page report that puts into question the safety of water fluoridation at 1 ppm. In particular, the NRC panel provides calculations in an exposure analysis in chapter 2 that indicate that subsets of the population are exceeding the EPA IRIS reference dose drinking water at 1 ppm. Other end points discussed by the NRC are occurring at concentrations in water that offer no adequate margin of safety to protect the whole population from harm. I have discussed one specific example of this above in connection with a lowering of IQ in children observed at 1.9 ppm (Xiang et al., 2003a,b) (see paragraph 62 above).

130 Paragraph 20(d) of the affidavit of AMB states:

> *"Skeletal fluorosis is a rare disease in Australia generally*

Signature……..……..Witnessed by……

Date……

and in areas where water is fluoridated at the Approved Concentration."

131 However, AMB offers no primary studies conducted in Australia that supports this proposition. There are none. He also fails to give regard to the fact that the first symptoms of skeletal fluorosis (stiff joints, aching bones and joints) are identical to arthritis. Australian health agencies are making no attempt to see if this condition is being caused by or exacerbated by fluoridation. None of AMB's responses Paragraphs 22 (a-e) to concerns about the possibility that fluoride lowers IQ in children at doses close to doses experienced in fluoridated communities do not bear up to scrutiny.

132 The claim Paragraph 22(a) AMB affidavit that:

"Reports of associations between intelligence and fluoride exposure in children come exclusively from China..."

is patently false. The facts are, there have been a number of studies not only from China but also India, Iran and Mexico, which report association between intelligence and fluoride exposure in children (see paragraph 61 et seq. above).

133 Paragraph 22(a) of the affidavit of AMB states the (Chinese studies) have taken place in communities that:

"have naturally fluoridated water often with very high levels that result in skeletal fluorosis."

is also false. The fact is, some of these studies took place in villages with relatively low levels of fluoride in the water (e.g. 2.5- 4.5 ppm, Xiang et al., 2003 a,b) and estimates indicate that the lowering may occur at 1.9 ppm (Xiang et al., 2003 a,b). The most recent study from Ding et al (2011) indicates a lowering of IQ in the range of 0.3 to 3 ppm (average 1.3 ppm).

134 Paragraph 22(b) of the affidavit of AMB further falsely states:

"These studies have usually been in Chinese and the details difficult to obtain."

In fact, several of the key studies have been available in English

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Date……..

for some time including Lin et al., 1991; Li et al, 1995; Zhao et al., 1996; Lu et al., 2000 and Xiang et al., 2003a,b. These five studies were reviewed in the NRC (2006) report. Other Chinese studies have subsequently been translated into English and have been made available in the journal *Fluoride* in 2008.

135 Paragraph 22(c) of the affidavit of AMB states:

> *"The studies have been criticised on many methodological grounds such as the methods of assessing intelligence and the controlling of confounding factors such as socioeconomic status and parental education."*

136 In his affidavit AMB does not cite a source for these criticisms. However, if he was aware of such critiques it is puzzling to understand why he states that the studies are only available in Chinese. AMB failed to state which studies were critiqued and in what language were the critiqued studies published?

137 Epidemiological studies are subject to criticism. It is very difficult to control for every possible confounding factor. However, one of the studies with the strongest methodology that controlled for many factors including lead exposure and iodine intake found a lowering at the level of 1.9 ppm (Xiang et al., 2003 a,b).

138 Paragraph 22(d) of the affidavit of AMB states:

> *"Although the studies purport to show some dose response effect between water fluoride levels and lower intelligence, the effects were at doses significantly greater than the Approved Concentration".*

139 In fact, Lin reported an effect at 0.9 ppm fluoride (Lin et al.,1991) ; Xiang reported an effect at 1.9 ppm (Xiang et al., 2003a.b) and more recently Ding et al. (2011) reported an effect within the range 0.3 to 3 ppm. In fact, Ding found a lowering of 0.59 IQ points for each 1 ppm fluoride excreted in the urine.

140 Needless to say, these effects are occurring at doses that will overlap with doses experienced by some children drinking uncontrolled amounts of water fluoridated at 1 ppm in Australia. Dr. Brown offers no comfort in this respect.

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Date……

141 Paragraph 22(e) of the affidavit of AMB states:

> *"Reviews of possible adverse health effects of fluoridation do not support a relationship with lowered intelligence in children at the Approved Concentration."*[14]

142 The single review that AMB cites to support this claim is the "*A Systematic Review of the Efficacy and Safety of Fluoridation*" NHMRC (2007), which in my opinion is both superficial and outdated in the treatment of this issue as I have discussed above and in my critique of this review in **Annexure 9.**

143 Paragraph 24(a) of the affidavit of AMB states that:

> *"The assertion that that fluoridated water can cause increased hip fracture rates in the aged at levels between 1 and 4 ppm misrepresents the findings of a study by Li et al from China.' This study found a doubling of the risk of hip fractures when people consume water with about 1.5 ppm fluoride and three times the risk of hip fractures when consuming water of greater than 4.3 ppm fluoride. However, the doubled risk of hip fractures at 1.5 ppm is not statistically significant and the authors found a "U" shaped relationship between the amount of fluoride in the water and fractures in general, with optimally fluoridated water (that is, water fluoridated at a concentration close to the Approved Concentration) actually conferring a protective effect against bone fractures."*

144 I have been aware of this report by Li et al, 2001, since receiving a pre-publication copy of the report from the lead author as part of my invited peer review of the York Review in 2000. It was also a key part of my invited oral testimony before the fluoridation Forum in Ireland in 2000. With my co-authors James Beck and Spedding Micklem we provide a thorough balanced analysis of this paper in chapter 17 of "*The Case Against Fluoride*." There are two interpretations of the Li et al. hip fracture data. One interpretation assumes that because the only statistically significant point at which hip fractures occurs was when the fluoride in the village well water exceeds 4.3 ppm, that this represents a threshold i.e. nothing happens to hip fracture rate between 1 ppm and 4.3 ppm, whereupon above this level the hip fracture rate abruptly triples. This interpretation – as articulated by AMB – ignores the approximately doubling of hip fracture rates,

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which occur in the two villages in which the water is between 1.5 and 3.5 ppm, because the increases were not found to be statistically significant. An alternative interpretation, which in my opinion is most likely is that the increase hip fracture rates between 1 ppm and 4.3 ppm, while not being statistically significant, actually represent a trend which is real. We believe that a trend is a more realistic representation of the data than a threshold at 4.3 ppm. In which case hip fractures may double at long-term exposure to 6 mg fluoride/day (a figure cited by the WHO in 2002) or even increase at doses as low as 3 mg fluoride/day; doses easily experienced by people living in fluoridated communities in Australia.

145 A simple way of summarizing AMB's comments in 24(b-d) is that the studies of hip fractures in fluoridated communities have been mixed and compounded by all the typical problems of ecological studies. That is what makes the Li et al., 2001 study very important from my point of view. The methodologies on hip fracture are no stronger or weaker than the studies that purport to demonstrate the effectiveness of fluoridation to reduce tooth decay. Apart from the study by Warren, 2009 (which examined tooth decay as a function of individual exposure) these studies are all of ecological design.

146 However, the notion that fluoride given a high enough dose weakens bones and makes them more brittle was given clinical support in the trials in which sodium fluoride were given to patients to treat osteoporosis (NRC, 2006). While doses of about 26 mg/day given a over a period of two or three years did harden the bones (i.e. increase bone mineral density) it also weakened the bones and made them more prone to fracture, especially the hip bone. While daily doses do not reach 26 mg/day, the cumulative doses (i.e. daily dose times number of days) experienced in these trials will be experienced over a lifetime for someone living in a fluoridated community. Data on bone fracture can be found at Chapter 17 of "*The Case Against Fluoride*."

147 Again this poses the question that since lifetime fluoride exposure is irreversible and hip fractures in the elderly are life-debilitating and even life-threatening (about 50% of the elderly impacted by a hip fracture never regain an independent existence and 25% are dead within a year) should these risks be taken to secure a very small (if any) reduction in tooth decay?

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Date……..

148 In Paragraph 26 it is not clear why AMB does not mention the study of Bachinskii et al., 1985, that found a lowering of thyroid function in otherwise normal people drinking water at 2.3 ppm. Such a finding offers an inadequate margin of safety for people drinking uncontrolled amounts of water fluoridated at 1 ppm and especially for those with borderline iodine deficiency for which there are many in Australia. Nor does AMB mention the whole chapter in the National Research Council review of 2006 dealing with fluoride's ability to disrupt the endocrine system including the thyroid gland (NRC, 2006, chapter 8). After 55 years of fluoridation in Australia no health agency has investigated the possibility that fluoridation is causing hypothyroidism or exacerbating the condition for those who are borderline. Nor has any Australian agency or researcher attempted to repeat Luke's findings that fluoride accumulates in the human pineal gland and lowers melatonin production in animals (Luke, 1997, 2001).

149 I agree with AMB's analysis of fluoride on G-proteins Paragraphs (25a-d). I would add that in the in vitro experiments G-proteins have been switched on at concentration that range from 20-200 ppm. Normally, these concentrations will not be reached in soft tissues, but they can easily be reached in the teeth, the bone, and other calcifying tissues like the pineal gland (Luke 1997, 2001). These concentrations will also be reached in the gum tissue during tooth-brushing with fluoridated toothpaste containing fluoride at 1000 ppm. G-protein mechanisms (amongst others) have been offered to explain both dental fluorosis (Matsuo et al., 1998) and fluoride-stimulated bone growth (Caverzasio,1998). A recent animal study Ge et al, (2011) explored which genes were up- or down-regulated in animal brain tissue treated with fluoride and found that G-proteins were among those up-regulated.

150 AMB addresses the issue of bone cancer and particularly osteosarcoma at Paragraphs 30(a-d) and in Paragraph 30(a) he states:

> *"osteosarcoma is a very rare cancer with only 6 cases reported in NSW children (0-14) in the five years 2004 - 2008."*[25]

151 I have no reason to doubt this figure, but how many teeth would you have to save to justify even one child dying from osteosarcoma if indeed

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Date……..

this cancer is related to fluoride exposure as some studies suggest (NTP, 1990; Hoover, 2001a; Cohn, 2002; Takahashi, 2001 and Bassin et al. 2006)?

152 Paragraph 30(e) of the affidavit of AMB concludes his discussion of fluoride and bone cancer by stating:

"Bone cancer is not an accepted adverse effect of fluoride exposure".

153 If however, he is referring to independent observers the jury is still out. The most robust study to date on the relation to fluoride to bone cancer has been that of Bassin et al. (2006) discussed above. The refutation of this study by Chester Douglass promised for the Summer 2006 has yet to appear (as of March 2011). If Bassin is correct, some young boys may be dying because of exposure to fluoridated water in their 6th to 8th years. But this serious outcome is being obfuscated by agencies like the NHMRC (2007) who are using Douglass's promised (un-peer-reviewed and unpublished) study to *"negate"* Bassin's findings.

Affidavit of Andrew John Spencer (Affidavit of AJS)

154 In paragraph [16] (a) of the AJS Affidavit , Prof Spencer writes that water, salt and milk fluoridation provide substantial protection against dental caries without causing "*unacceptable"* levels of dental fluorosis, as well as no other adverse health effects. However, Prof Spencer does not define what would be an *"acceptable"* level of fluorosis and what would be an *"unacceptable"* level. He also does not refer to any primary health studies conducted in Australia that have looked for adverse or even beneficial health effects on any organs or tissues in the body other than teeth. This is probably because, as we have discussed above, very few, if any, primary health studies have been conducted in Australia in the 50 plus years since fluoridation has been practiced in this country.

155 I will address my comments on the AJS affidavit under three headings: dental decay, dental fluorosis and other health effects.

i) Dental Decay

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Date……..

156 Paragraph [16] (o) of the AJS Affidavit of Professor Spencer states, that the 1991 NHMRC reported an approximate 60% protective effect on DIMFT in 1977 and an approximate 40% protective effect in 1987 (DIMFT = Decayed, Indicated for extraction, Missing and Filled Teeth). However, in his own Spencer et al. 1996 paper, the authors reported that a 100 per cent lifetime of exposure to fluoridated water gave a protective effect of saving 2.30 dmfs (decayed missing an filled tooth surfaces on the *primary* or *deciduous* teeth) in Queensland and 1.80 dmfs in South Australia and only 0.30 DMFS (decayed, missing and filled surfaces on the *secondary* or *permanent* teeth) for Queensland and only 0.12 DMFS for South Australian children's permanent teeth. Considering that there are over 100 tooth surfaces in a child's mouth, a saving of only 0.12, or at best 0.30, permanent tooth *surface*s (there are five surfaces on the chewing teeth and four surfaces on the cutting teeth and a total of 128 tooth surfaces in a child's mouth when all the teeth have erupted) saved for a lifetime of consuming fluoridated water, is very, very small. It represents an *absolute* saving of far less than 1% of the permanent tooth surfaces. This *absolute* saving is much less impressive than the NHMRC, 1991 claimed 40 % reduction. The saving of 0.12 to 0.3 DMFS may have been even smaller had a delayed eruption of teeth due to fluoride ingestion been investigated (Komarek et al., 2005). In fact, a one-year delay (or less) in tooth eruption in fluoridated communities would almost certainly have eliminated these small benefits. When the issue of delayed eruption of teeth has been clarified it may turn out that the reported *"benefits"* ranging from 0 to 1 DMFS reported in other American, Australian and New Zealand studies, are simply an *artifact* of the delayed eruption caused by fluoride (Brunelle and Carlos, 1990; Spencer et al. 1996; De Liefde, 1998; Lee and Dennison, 2004; Mackay and Thomson 2005; Armfield, 2005; Armfield et al., 2009 and Armfield, 2010). In a study published by Peiris et al., 2009, it was shown that the dental age (DA) in Australia was some 0.82 years later than the dental age in the UK. It has not been established that this delay in dental age was due to fluoridation, but it must be a candidate explanation since Australia is largely fluoridated and in the UK only 10% of the population is drinking fluoridated water. Despite Komarek's study and Peiris's observations not one of the dental studies cited by Spencer claiming benefits from fluoridation have controlled for delayed eruption of the teeth. Nor is this a new issue. According to the authors of the UK York Review (McDonagh et al., 2000) who took a comprehensive look at all the published longitudinal studies on tooth decay as of 2000,*"No study*

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Date……..

used an analysis that would control for the frequency of sugar consumption or the number of erupted teeth per child" (York, p.24). It is surprising then that authors of recent Australian studies are still not attempting to control for this important factor.

157 In paragraph [16] (s) A and B, of the AJS Affidavit Prof Spencer refers to the 2005 paper by Jason Armfield "*Public water fluoridation and dental health in New South Wales."* AJS asserts that children in fluoridated areas had lower caries experience than fluoridated, regardless of socioeconomic disadvantage and indigenous status. However, when comparing several non-fluoridated areas and fluoridated NSW Health areas (as summarised in Table 1) there is on average less than one tooth difference in decayed deciduous teeth and even less in the permanent teeth.

Table 1

5-6 Year-old dmft (dmft = decayed, missing, filled BABY teeth)			
NSW Health Region Year 2000	Non-fluoridated	Fluoridated	Mean number of teeth difference in decay
Greater Murray	1.94	1.02	0.9 of a tooth
Hunter	1.90	0.75	1.2 of a tooth
Mid North Coast	1.53	0.96	0.7 of a tooth
Mid West - including Lithgow unfluoridated Bathurst fluoridated	1.11	1.14	No significant difference
New England	1.69	0.87	0.8 of a tooth
Northern Rivers	1.83	1.27	0.6 of a tooth
SW Sydney	0.83	0.93	No significant difference
Southern	1.37	0.89	0.5 of a tooth

11-12 Year-old DMFT (DMFT = decayed, missing, filled PERMANENT teeth			
NSW Health Region Year 2000	Non-fluoridated	Fluoridated	Mean number of teeth difference in decay
Greater Murray	0.59	0.35	0.2 of a tooth
Hunter	0.40	0.44	No significant difference
Mid North Coast	0.48	0.17	0.3 of a tooth

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Date……..

Mid West - including Lithgow unfluoridated Bathurst fluoridated	0.41	0.39	No significant difference
New England	0.43	0.29	No significant difference
Northern Rivers	0.66	0.56	No significant difference
SW Sydney	0.53	0.47	No significant difference
Southern	0.47	0.32	0.2 of a tooth

158 Similarly, the maximum difference in 11 – 12-year-old's permanent teeth was *only* 0.2 of a tooth surface when comparing children from non-fluoridated areas with fluoridated areas while also comparing the most disadvantaged children to the most advantaged by SEIFA (Socio – Economic Index for Areas).

159 Again, it is important to underline the fact that no control was made in this study for the possible delayed eruption of teeth in fluoridated areas and a delay of half a year would have eliminated these small differences.

160 Paragraph 16 (ii) (D) of the Affidavit of AJS refers to a paper titled *"Caries experience among children in fluoridated Townsville and non-fluoridated Brisbane" Aus NZ J Public Health"* 1996. Prof Spencer asserts that *"Caries rates were significantly lower for children in Townsville than in Brisbane children, both in the deciduous and permanent dentition (p 626 – 7) . Water fluoridation appeared to provide a substantial benefit for children in Townsville"*

161 However, an examination of the data (as reproduced in Table 2) shows that while the tooth decay differences, when expressed as a *percentage*, may appear impressive when expressed as an *absolute* difference they are not so, especially when considering that there are over 100 tooth surfaces in a child's mouth.

162 The authors themselves conceded that for children aged 6 to 12 years that there was an average difference of *only* 0.25 decayed permanent teeth surfaces when comparing the teeth of fluoridated Townsville children with non-fluoridated Brisbane children. Bearing in mind the very small savings in tooth decay here, it is disturbing that Queensland Health used the data from this study (see Table 2) and

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Date……..

placed newspaper advertisements across Queensland in early Dec 2007 claiming that Townsville children with fluoridation had 65% less tooth decay than Brisbane children without fluoridation (see **Annexure 10** for a copy of one of these advertisements). This was very misleading. The 65% figure was derived from just one age (7 years). This figure possibly represents the mathematical vagaries of comparing two small numbers (0.09 and 0.26) such that any small change in the 0.09 figure would make a large change to the percentage figure. Meanwhile, even if this 65% figure is real, the saving of 65% figure, used to justify mandatory fluoridation on the state, amounted to a saving of *only* 0.17 of one tooth surface in 7 year-olds. Moreover, as indicated above, such changes would have disappeared if there were a few months delay in the eruption of teeth in the fluoridated community.

Table 2
Caries Experience (decayed, missing or filled surfaces)

Age	Townsville mean dmfs	Brisbane mean dmfs	% Difference	Surfaces Difference
5	1.35	2.98	55	1.63
6	2.23	3.48	36	1.25
7	2.28	4.4	48	2.12
8	2.61	4.99	48	2.38
9	2.64	3.89	32	1.25
10	2.03	3.97	49	1.94

Age	Townsville mean DMFS	Brisbane mean DMFS	% Difference	Surfaces Difference
6	0.04	0.1	60	0.06
7	0.09	0.26	65	0.17
8	0.25	0.52	52	0.27
9	0.41	0.51	20	0.1
10	0.57	1.13	50	0.56
11	0.65	1.45	55	0.8
12	0.94	1.8	48	0.86

dmfs = number of decayed, missing or filled baby teeth surfaces per child.
DMFS = number of decayed, missing or filled permanent teeth surfaces per child.

Signature……..……..Witnessed by……

Date……..

163 Paragraphs 25 (a) and (b) of the Affidavit of AJS refer to the AIHW's report of the National Adult Oral Health Survey 2004- 2006 (NASOH 2004-2006) also known as AIHW "*Australia's Dental Generations".* When this report was publicly released, Prof Spencer, and Prof Gary Slade, who was also employed at the Australian Research Centre Population Oral Health (ARCPOH) at Adelaide University Dental School, went on national television news promoting water fluoridation on the basis that the *"fluoride generation"* had half the tooth decay of the generation before. This promotion however disregarded any contribution from fluoride toothpaste and several other confounders and also the issue that similar declines in tooth decay have been observed in other developed countries in the same time frame. Based on World Health Organization tooth decay data for 12-year-olds many *non-fluoridated* countries (graphically presented at www.FluorideAlert.org/who-dmft.htm) could have claimed that the current generation had half the tooth decay of the previous generation. It is hard to believe that both Spencer and Slade were unaware of the WHO database.

164 In Figures 2 to 8a comparison is made for tooth decay among the largely fluoridated states and territories and largely unfluoridated (when the data was collected) Queensland. It is striking to note that in no age category considered, or for all the ages combined, was the tooth decay in largely unfluoridated Queensland (<5%) at the top of the list of decay by state. Yet it was claims that tooth decay was much worse in Queensland, compared to the rest of Australia, that were used to push mandatory statewide fluoridation onto Queensland.

Figure 1 shows the percentage of the Australian population with a fluoridated water supply. These percentages were confirmed by the NHMRC in 2007 just after the data for the National Adult Oral Health survey 2004-06 had been collected. In figures 2 to 8, a comparison is made for tooth decay among the largely fluoridated states and territories and largely unfluoridated (when the data was collected) Queensland. It is striking to note that in no age category considered, or for all the ages combined, was the tooth decay in largely unfluoridated Queensland (<5%) at the top of the list of decay by state. Yet, it was based on claims that tooth decay was much worse in Queensland, compared to the rest of Australia, that mandatory

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Date……..

statewide fluoridation was forced onto Queensland. (The individual state and territory reports or the (Australian) National Adult Oral Health survey are available from http://www.arcpoh.adelaide.edu.au/project/distribution/NSAOH.html)

Figures 7 and 8 show that ARCPOH is aware that there is little difference in tooth decay between adults from all Australian States and Territories and Queensland, despite the latter not being substantially fluoridated.

Figure 1 Water fluoridation extent; sourced from 2007 NHMRC Public Statement map as reproduced here.
http://www.nhmrc.gov.au/_files_nhmrc/file/media/media/rel07/Fluoride_Flyer.pdf

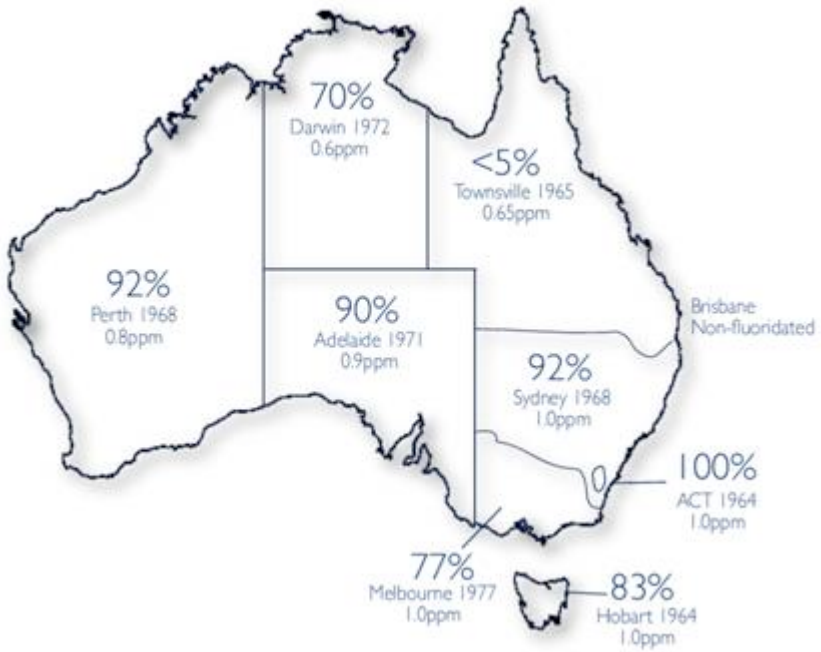

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Figure 2 – Tooth decay rates per age group by state with percent population with fluoridated water supply. Sourced from the individual state and territory reports of the (Australian) National Adult Oral Health survey 2004-06.

DMFT = Decayed. Missing and Filled Teeth due to decay					
	All ages	15-34 yrs	35-54yrs	≥ 55yrs	% Fluoridated
STATE	DMFT	DMFT	DMFT	DMFT	(NHMRC 2007)
ACT	11.0	2.6	12.9	22.7	100%
NSW	12.8	4.4	13.9	22.7	92%
NT	10.7	5.0	12.7	22.1	70%
QLD	13.1	5.0	14.6	23.1	<5%
SA	12.7	4.1	14.0	22.1	90%
TAS	13.4	4.9	14.1	23.4	83%
VIC	12.8	4.4	15.1	22.5	77%
WA	13.1	5.4	14.5	22.7	92%

Figure 3 – Tooth decay rates for the 15 – 34 year age group by state. Sourced from the individual state and territory reports of the (Australian) National Adult Oral Health survey 2004-06.

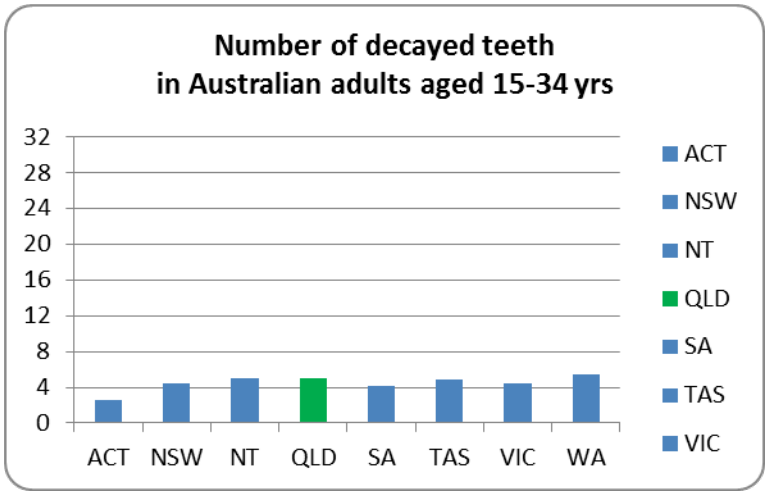

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Figure 4– Tooth decay rates for the 35 – 54 year age group by state. Sourced from the individual state and territory reports of the (Australian) National Adult Oral Health survey 2004-06.

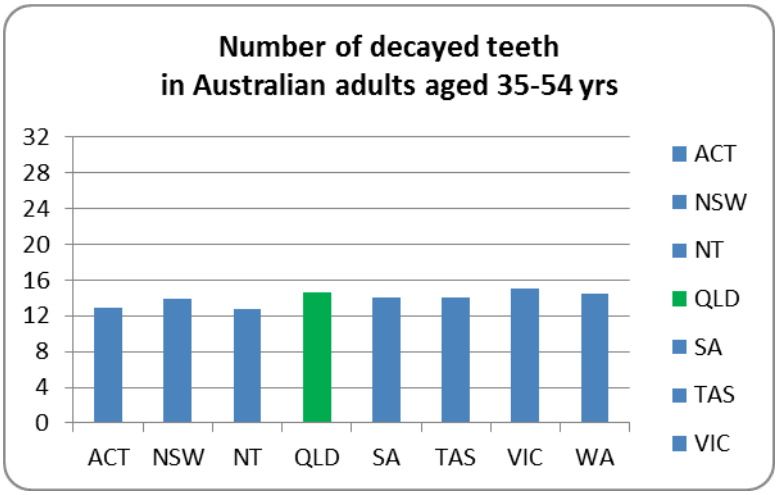

Figure 5 – Tooth decay rates for the 55 years and older age group by state. Sourced from the individual state and territory reports of the (Australian) National Adult Oral Health survey 2004-06.

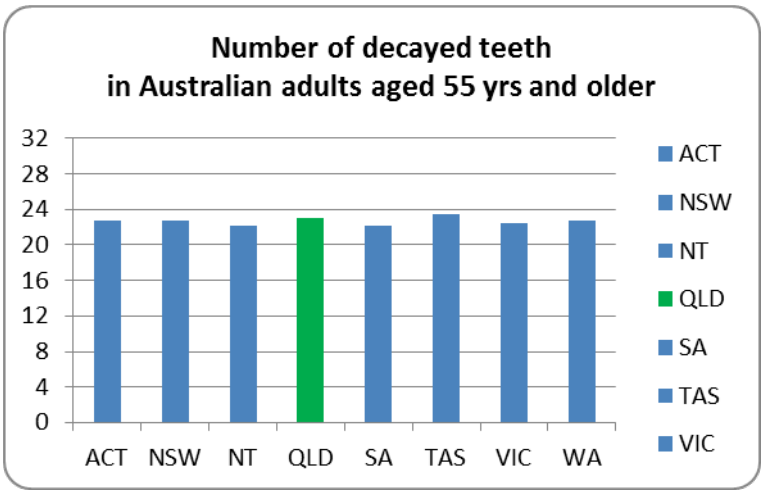

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Figure 6 – Tooth decay rates for all ages (15 – 99 years) by state. Sourced from the individual state and territory reports of the (Australian) National Adult Oral Health survey 2004-06.

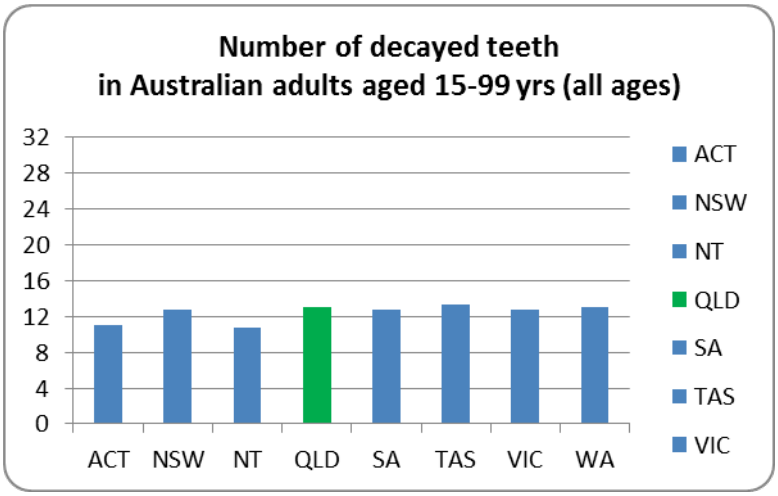

Figure 7 – Extracts from a presentation by Kaye Roberts-Thomson (Executive staff member ARCPOH) on the findings of the (Australian) National Adult Oral Health Survey 2004-06 http://www.arcpoh.adelaide.edu.au/workshop/NSAOH%20workshop%20Caries%20experience.pdf

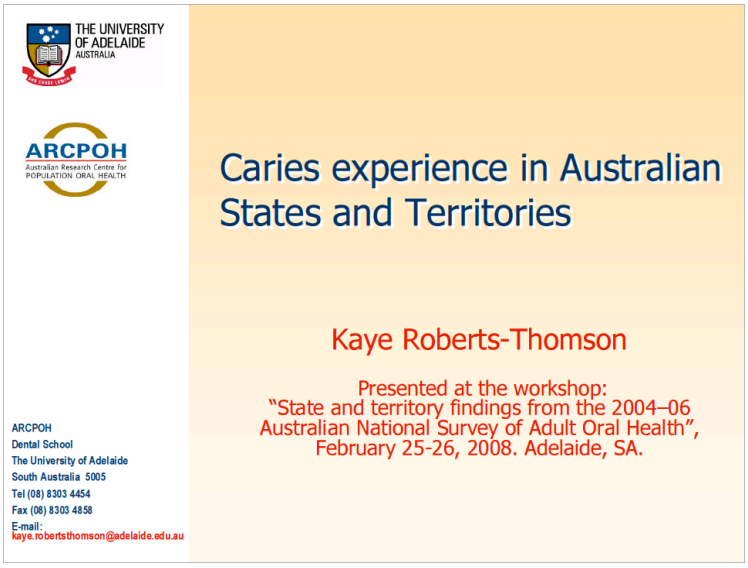

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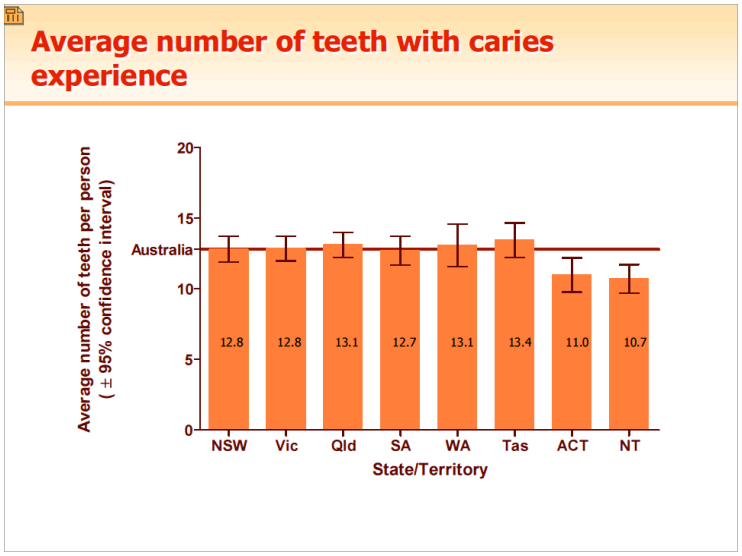
Figure 8 – Extracts from a presentation by Kaye Roberts-Thomson (Executive staff member ARCPOH) on the findings of the (Australian) National Adult Oral Health Survey 2004-06.



165 Paragraph 25(b) of the AJS Affidavit Prof Spencer seems to downplay the significance of these tooth decay comparisons between adults in the individual states and territories on the basis that the current place of residence does not classify an adult for their lifetime exposure to fluoridated water considering that residential mobility creates the risk of misclassification. However, this factor should have been discounted when comparing the one stand-out state of Queensland which had less than 5% of the population exposed to fluoridated water, when comparing Queensland adults to adults in all the heavily fluoridated states. While some Queenslanders may have grown up in other states and some Queensland adults may have gone to live in the heavily fluoridated states, it is unlikely that enough adults would have swapped their states of residence to totally invalidate these striking state to state comparisons.

166 Paragraphs 28, 29 and 30 of the AJS Affidavit refer to Prof Spencer's response to an assertion By Dr. Mark Diesendorf and others that a

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Date……

study he co-authored with Jason Armfield in 2004 titled *"Consumption of nonpublic water: implications for children's caries experience"* documented a lack of effectiveness with respect of fluoridation's protection of the *permanent* teeth.

167 In paragraph 30 (a) of the AJS Affidavit of AJS Prof Spencer responds to these assertions by saying that his research indicates that when children living in an area with fluoridated water substitute non- tap water for drinking, there is a significant association with increased caries experience in the deciduous teeth and a trend of increased caries experiences in the permanent teeth. I have examined this paper and I noted that the authors did find a significant difference in the baby teeth of 4 to 9 year children when comparing those who had always drunk fluoridated public water to unfluoridated non- public water, but that they did *not* find a *"significant difference"* when comparing decay in the permanent teeth of 10 to 15 year olds, between those who had consumed non – fluoridated water (bottled or tank water) for all of their lives, and those who consumed fluoridated water for all of their lives. They state this fact quite clearly in the abstract of their paper.

168 In the 4 to 9 year old grouping of children who had always drunk fluoridated water there was an average of 2.95 tooth surfaces in baby teeth that had been affected by decay. In this same age group children who had never drunk fluoridated water had an average of 4.47 tooth surfaces in baby teeth that had been affected by decay. Thus the saving in baby teeth amounted to 1.52 dmfs.

169 The authors, after factoring in differences in household income, *"occupational prestige"* and education say the benefit found in baby teeth *"remained significant,"* although the effect size of this relationship was small. They did not however, publish the actual quantitative results for baby teeth after factoring in these differences.

170 A new publication in the Nov – Dec 2110 issue of the NSW Public Health Bulletin authored by Amit Arora and Robin Wendell Evans contains some of the latest information on tooth decay in NSW children. This study compares Lithgow, which will commence fluoridation in 2011 to the fluoridated communities of Bathurst and Orange. The paper describes these communities as being *"socioeconomically comparable."* This study actually shows that in

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2006, when the children were examined, that 6-year-old children from un-fluoridated Lithgow had less tooth decay in their baby teeth (dmft of 0.92) than those in fluoridated Bathurst and Orange, although the authors said the difference was not significant. Although the mean DMFT of 0.69 in the permanent teeth for Lithgow's 12 year old children was higher than Orange (DMFT 0.33) and Bathurst (0.29) The finding of a DMFT of only 0.69 is quite favourable when comparing with the latest published Australian Children's Dental Survey 2003- 2004, which found that the average DMFT was 1.03 for Australian 12-year-old children (the large majority living in fluoridated areas). Thus, a DMFT of 0.69 for Lithgow's 12 year olds is actually *lower* than the average DMFT for 12 year olds in the reported States and Territories results (NSW was withheld) in the 2003-2004 Australian survey. Regardless of this favorable result for Lithgow children's oral health, the Arora and Evans (2010) report is being used to force fluoridation on Lithgow residents by the combined actions of its council and NSW Health.

ii) Dental Fluorosis

171 Paragraph [16] (r)(i) of the AJS Affidavit refers to the NHMRC's recommendation in its 1991 report that it was necessary to establish a much more detailed and higher quality data base for the purpose of monitoring trends (including dental fluorosis). In paragraph [16] (l), Prof Spencer acknowledges that he was a member of this 1991 NHMRC review panel. Prof Spencer in listing his qualifications also acknowledges that in 1988 he established the Dental Statistics Research Unit. Thus, Prof Spencer was in a perfect position(s) to encourage an Australian wide child dental fluorosis survey to be undertaken. However, no Australian-wide *child* dental fluorosis studies have been published in the many years following the 1991 NHMRC recommendation. The Australian National Adult Oral Health Survey (2004- 2006) did collect data on *adults* in each state and territory. However, it is more than 5 years since that data was collected and it has not been made public. When the data is published, and providing a state comparison is made, it will be important to compare the rates of adult fluorosis in Queensland (which in 2004-2006 had less than 5% of its population exposed to fluoridated water) to the other (extensively fluoridated) Australian states and territories.

172 Paragraph 20 (n) of the Affidavit of AJS appears to infer that children

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Date……..

perceived their teeth with very mild fluorosis (TF1) as being more attractive than teeth with no fluorosis (TF0) or teeth with mild or moderate fluorosis (TF2+) .

173 Prof Spencer then generalized this observation to conclude that *"Dental Fluorosis did not have a negative impact on the perception of oral health and oral health- related quality of life of the South Australian children."* Without qualifying the words "*dental fluorosis"* in this sentence with the label *"very mild"* this seems to imply that children have experienced no negative impacts from having *any level* of dental fluorosis. This is highly misleading as the following discussion will demonstrate.

174 Moreover, this positive view of *very mild* dental fluorosis is totally inconsistent with Hoskin and Spencer's finding that children affected by *fluorosis* and their parents are able to perceive the presence of fluorosis at a *very mild* level. They also concluded that children with *mild* fluorosis showed a significant adverse psychological response to their dental appearance. This finding was presented as *"Personal perceptions of dental fluorosis in South Australian children"* at the Consensus Conference on Appropriate Fluoride Exposures for Infants and Children held in Perth, Western Australia, 1993 as reported in the 1998 *AIHW report "Review of Water Fluoridation: New Evidence in the 1990s."*

175 The claim that dental fluorosis is not an adverse effect of fluoridation and that it is *"merely cosmetic"* is at odds with the findings of Hoskin and Spencer.

176 According to Hoskin and Spencer, South Australian children 10- to 17-years-old were able to recognize *very mild* and *mild* fluorosis and register changes in satisfaction with the colour and appearance of teeth. Even mild changes were associated with psycho-behavioural impacts. Hoskin and Spencer asked eight questions on psycho-behavioural impact, such as embarrassment of teeth or self-consciousness because of the appearance of the teeth. The most dramatic finding was the strength of the association of TISF score with psycho-behavioural impact was similar to that of crowding and overbite, both considered key occlusal traits driving the demand for orthodontic care. Spencer AJ, et al. (1996). Water fluoridation in Australia. *Community Dental Health* 13(Suppl 2): 27-37. Although the

Signature……..……..Witnessed by……..

Date……..

Hoskin and Spencer findings were referred to by name in two publications, one for which Prof Spencer was lead author *(Spencer et al., 1996, Water Fluoridation In Australia)* and the other for which Prof Spencer was an executive staff member for the 1998 University of Adelaide report "*Review of Water Fluoridation: New Evidence in the 1990s."(AIHW 1998),* I have been unable to locate any published paper by Hoskin and Spencer. Thus, these findings, may or may not have been published.

177 The 1993 study by Perth dental researcher PJ Riordan *"Perceptions of Dental Fluorosis"* gathered the opinions of groups of 110 observers (parents, administrative staff, nutrition students, first year preclinical students and also 12 Dentists) with regard to the appearance of 28, 14 year old children who had degrees of fluorosis varying from TF scores 0 (no fluorosis) to 3 (moderate fluorosis).

178 Observations were made at a *"conversational"* distance from the subject and observers noted their responses onto a questionnaire. The questionnaire was designed to ascertain the feelings of the observers about the appearance of children's teeth and how they imagined different degrees of fluorosis might impact children. The results indicated that the low TF scores might not embarrass the children and that higher scores (TF = 3) could be a source of embarrassment, particularly for the girls. The higher fluorosis scores were seen by lay people as a sign of neglect on part of the children towards their teeth. The dentists felt that they would recommend treatment for children with TF scores of 3.

In this paper Riordan states:

> *…"there has been a tendency on the part of dental researchers to play down the negative esthetic consequences of fluorosis".*

Riordan also states

> *"Hitherto, the public has not been interested in dental fluorosis, but it can be expected that at some stage the issue will be raised in public debate. Proponents of fluoride-based preventive strategies would then be in a stronger position if it can be shown that most fluorosis in the community is not perceived to be a problem by the community"*

Signature……..……..Witnessed by……..

Date……..

179 In recent times, teenagers, girls particularly, are becoming increasing self-conscious about their appearance and it is recognized that self-consciousness about physical appearance impacts on self-esteem, which can have effects for a lifetime. In these terms, it is clear that *moderat*e fluorosis equates to harm to a child.

180 As one example of this see **Annexure 11** a submission to the Director Standing Committee on Social issues Legislative Council - Inquiry into Dental Services in NSW - Parliament House Sydney– Submission 242 from Mr Lewis Clyde James on the 19 /8/2005. This submission attests that dental fluorosis is harm to a child.

181 The 2007 NSW Child Dental Survey, which was published in 2009, shows that in 11 to 12 year olds 3.8%, or nearly 4 children out of 100, in fluoridated areas have fluorosis in the TF3 or TF4+ categories (i.e. moderate or clearly visible fluorosis) whereas, only 0.2 % of 11 to 12 year old children in non- fluoridated NSW areas had moderate dental fluorosis (TF3). It is of concern that nearly four children in a hundred may suffer from adverse effects on their self-esteem because of exposure to fluoridated water. It should be noted that fluorosis was only measured on one maxillary central permanent incisor per child. If "whole of mouth" fluorosis measurement was done, it may have yielded a higher percentage of children with moderate fluorosis.

182 From a historical perspective it is worth pointing out that in 1952 the "father of fluoridation" H. Trendley Dean, in testimony before the US Congress, said:

> *"We don't want any 'mild' [fluorosis] when we are talking about fluoridation. We don't want to go that high and we don't have to go that high . . . I don't want to recommend any fluoridation where you get any 'mild' [fluorosis]."* (Dean, 1952)

183 In more recent times, In an article published in the New York State Dental Journal in 2008, Elvir Dincer, DDS, concluded:

> *"that children's self-esteem is harmed by even mild fluorosis." (Dincer, 2008)*

Signature……..……..Witnessed by……

Date……..

iii) Other Health Risks

184 Paragraph 20 (s) of the Affidavit of AJS asserts that water fluoridation has been subject to great scrutiny with regard to health risks. As I have discussed above in my critique of both Blinkhorn's and Wright's affidavits Australia has produced very few, if any, primary studies examining the health of communities that have been fluoridated.

185 In addition, I would add that the NHMRC has never published or apparently investigated risks of the cumulative effect of fluoridation on people with impaired Kidney function, or the risks to people with impaired thyroid function.

186 Nor have there have been studies done that compared the rates of hypothyroidism in Queensland before it was fluoridated, to rates in the heavily fluoridated states.

187 Thus Prof Spencer's assertion that the only established outcome of fluoridation is dental fluorosis is not convincing since he offers no primary studies to support such an assertion. Moreover, as we have seen from the above analysis, his claim that most of the dental fluorosis is not of concern is actually refuted in studies he has himself co-authored. Prof Spencer's further claim that the most *"recent research"* has clouded judgment even about mild fluorosis being an adverse outcome, is also not supported by reference to the primary literature. But such nuances are somewhat trivial when we remember that there are now 24 studies that have associated exposure to fluoride to lowering of IQ in children.

<u>The Affidavit of Dr John Colin Chapman</u>

188 I note that Dr. Chapman refers to the SCHER report many times in his affidavit (see 23b; 27d; 27e,Table 1; 34d; 34g (twice); 34h; and 34j) however this report is only a *draft* not a *final* document. In his references this report is reported as a "pre-consultation opinion." It is unusual for experts to rely on draft documents when providing testimony. At the very least, in my view, he should have informed the

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Date……..

court that this was a *draft* not a *final* document.

189 Dr. Chapman in 14c) claims that his proposed interim guideline of 0.8 mg/L for fluoride will protect against any effect (chronic and acute) on 95% of all aquatic species over their entire life cycles with a 50% certainty. I am not convinced that this is the case and I am also concerned about the 5% of species (unknown) that may not be protected. These concerns are addressed in more detail below.

190 This 0.8 mg/L *"interim guideline"* does not compare well with other guidelines. For example, the Canadian Water Quality Guidelines for the Protection of Aquatic Life (Environment Canada, 2001) adopted an interim guideline of 0.12 mg/L for inorganic fluorides in fresh water. This guideline document states:

> *"An interim water quality guideline of 0.12 mg FIL is recommended for the protection of all stages of freshwater life against the adverse affects of total inorganic fluorides. It was derived by multiplying the lowest value from an acute, static toxicity study on the net-spinning caddisfly, Hydropsyche bronta, a LC50 of 11.5 mg F1L, by a safety factor of 0.01. The guideline was derived from an acute study, as no acceptable lower value from a chronic toxicity study was available. Due to a lack of required toxicity studies, neither full nor an interim water quality guideline for the protection of marine life could be developed at this time." (Environment Canada, 2001, last paragraph, p. vii)*

191 In 14 d) Dr. Chapman states that:

> *"if fluoridated drinking water were to be released directly into the environment, the chlorine which has been added to it for disinfection purpose would be far more toxic and would have a far greater effect on the environment than fluoride."*

I reject the relevance of this comment. Fluoridation is not being proposed as an *alternative* to chlorination. Whatever risks to the eco-system and human health that fluoride poses will be in addition to the risks posed by chlorination. Moreover, the justification for chlorination is far greater than the justification for fluoridation. Chlorination is used to fight disease-causing bacteria and other vectors and undoubtedly saves many lives, whereas fluoridation is used to fight tooth decay, a non-life threatening disease. In addition, as I have discussed above the effectiveness of this measure

Signature……..……..Witnessed by……

Date……..

is in serious question. What is not in question is that many other countries have avoided this practice without suffering higher levels of tooth decay than fluoridated countries (see World Health Organization data online and displayed graphically at www.FluorideAlert.org/who-dmft.htm.)

192 In 14 e) Dr. Chapman states that

> *"international literature also concludes that fluoridation of water at the Approved Concentration will pose negligible risks to organisms in the environment."*

My response. This statement is scientifically inaccurate. As discussed above there is no adequate margin of safety to protect the human species from several harmful effects, including the lowering of IQ in children. As far as aquatic species are concerned, the Canadian guideline of 0.12 mg/liter (Environment Canada, 2001) is 6-7 times lower than the standard that Dr. Chapman proposes (0.8 mg/L). Moreover, as discussed above, the "international literature" that Dr. Chapman cites includes a *draft* not a *final* document (SCHER, 2010).

193 In 15) Dr. Chapman states that:

> *'Scientific evidence supports the fluoridation of water supplies as safe for the environment…"*

194 My response is that Dr. Chapman does not cite any original (i.e. primary health or environmental) studies that have been conducted by NSW Health to support this position, especially the local ecology of the proposed Rous dosing plants. There has been no EIS done of the area.

195 Furthermore, Dr. Chapman's own statement in paragraph 23 (c) contradicts his claim, by stating:

> *" There are NO studies in the literature that I am aware of that examine fluoride uptake in aquatic organisms exposed to elevated levels of fluoride from drinking water or STP's in areas where water is fluoridated".*

Signature……..……..Witnessed by……..

Date……..

196 Dr. Chapman's statement that the *"Scientific evidence supports the fluoridation of water supplies as safe for the environment…"* (15c) fails to acknowledge the abundant evidence of the escalating distribution of inorganic fluorides into the human food chain and the environment. Moreover, more and more scientific evidence is revealing the real dangers of fluoride to many species including humans. For example:

> *"The abundance of inorganic fluorides in the environment is increasing because of their growing use in industrial and dental health products. Based on levels of inorganic fluorides in ambient air, drinking water, food, and household and dental products, the average total daily intake by humans is approximately 20% below the level at which adverse effects upon the skeleton are expected (Liteplo et al. 1994)." (Environment Canada, 2001, p.3)*

197 The US CDC has admitted that a huge number of children in the US have been over-exposed to fluoride. According to CDC (2010), 41% of ALL American children aged 12-15 are now afflicted with dental fluorosis. *"All children"* means that this figure is an average of children living in both fluoridated and non-fluoridated communities and thus it can be assumed that the percentage affected in fluoridated communities will be even higher than 41% and probably closer to 50%.

198 In 23 a) Dr. Chapman states that:

> *"the concentrations (of fluoride, PC) in unpolluted streams, rivers and lakes are generally in the range of 0.01 to 0.3 milligrams per liter (mg/L)"*

199 My response. The implication here is that if something occurs naturally in water it is safe. This is not the case. Arsenic occurs naturally in some water supplies, this does not make it safe, either for humans or aquatic species. What level of a mineral like fluoride ends up in the water is a vagary of which rocks the water has passed through, combined, in the case of surface water, with the dilution offered by rain water and melting snow. A better guide to what is safe – especially for a human baby or other mammal – is the level found in mothers' milk. This is extremely low (0.004 ppm in babies living in non-fluoridated communities, NRC, 2006, p. 40). Thus the safe level that Dr. Chapman proposes for aquatic species (0.8 mg/L) is actually

Signature……..……..Witnessed by……

Date……..

200 times the level found in mothers' milk.

200 In further response to Dr. Chapman's reference to background or natural levels of fluoride I would like to stress that there is nothing natural about the fluoride added to water. The fluoridating chemicals used in water fluoridation programs are derived from the wet scrubbing systems of the superphosphate industry. These wet scrubbing systems were designed to capture two very toxic gases (HF and SiF_4). For many years these gases decimated vegetation and crippled cattle in the vicinity of the plants. Fortunately a spray of water converts them into hexafluorosilicic acid (H_2SiF_6). When the scrubbing liquor reaches a concentration of about 23% it is sold to public water utilities to be used for water fluoridation. It is either used as the acid or as the sodium salt (Na_2SiF_6). These silicon fluorides do not occur naturally**;** they are man made in this scrubbing process. These chemicals (silico-fluorides) also contain many toxic contaminants including trace amounts of radioactive isotopes, because the same phosphate rock is also used as a source of uranium.

201 I agree with two statements cited by Dr. Chapman in 23 b), namely:

> *"there is a relatively narrow range between intakes associated with beneficial effects and exposures causing harmful effects" (WHO, 2002), and*
>
> *"Fluoride is not an essential element for human growth and development, and for most organisms in the environment." (SCHER, 2010, draft document).*

202 My response. Considering this relative narrow *"margin of safety"* for harmful effects, and with no support given to the notion that fluoride is essential for human growth (either by SCHER or numerous other scientific bodies), it is surprising that Dr. Chapman, or any one else, would support the unnecessary addition of this problematic element to the environment. As argued above, the most rational application of fluoride in fighting tooth decay is via topical application using fluoridated toothpaste. There is no need to flush over 99% of the water artificially fluoridated with industrial grade chemicals into the environment.

Signature……..……..Witnessed by……..

Date……..

203 I was not reassured by Dr. Chapman's comment in 23 c) that

> *"There are NO studies in the literature that I am aware of that examine fluoride uptake in aquatic organisms exposed to elevated levels of fluoride from drinking water or STP's in areas where water is fluoridated."*

204 Clearly this is a *major* problem and offers definitively no guarantee for the safety of aquatic and terrestrial biota. If there are no such studies then a claim of environmental safety is nothing more than speculation. In the 1970's DDT and Mercury in water are examples of such an oversight

205 In this matter we seem to be hostage to the kind of theoretical calculations provided by Dr. Chapman. In my view, such theorizing is no substitute for field data, especially field data in the local area where fluoridation is planned.

206 At paragraph 24 Dr. Chapman cites the fact that government agencies in Denmark and Sweden rejected water fluoridation in their countries partially because of the unknown threats it might pose to the environment and foodchains.

207 At paragraph 25a Dr. Chapman states that more information available on these unknown threats today than in 1977 and 1981 when these statements cited in 24) were made.

208 My response, I am not as convinced as Dr. Chapman that these issues have been resolved by more recent data (as he himself acknowledges in paragraph 23 c). In my view, the data available today is still far too limited to be as categorical as Dr. Chapman is prepared to be in claiming that his interim guideline of 0.8 mg/L will protect 95% of aquatic species over their total life cycles.

209 Again in paragraph 27(a) Dr. Chapman cites *"natural"* background levels of fluoride with the implication that they are safe for all species. See my comment above.

210 At paragraph 27(e) Dr. Chapman provides a table (Table 1) summarizing various toxic end points for a number of species. When one considers the millions of aquatic species that there are and the complexities of ecosystems, where interfering with the lifecycle of one

Signature……..……..Witnessed by……..

Date……..

species might have long term ramifications for others, I am not convinced that this list of studies is sufficiently comprehensive to generate the confidence that Dr. Chapman has in this matter. I also note that the number of references to these studies is limited and includes the SCHER report, which is still in a draft stage. I also note that there is only one reference to studies on frogs (WHO, 2002) and none for other amphibians. I am also concerned that many of the studies are probably *"fish tank"* studies and may not mimic real life conditions experienced in small creeks or estuaries in a very hot climate, subject to droughts.

1: Acute toxity of fluoride to freshwater fishes and aquatic invertebrates

Test species **(& no. of species)**	Test duration **(days)**	Range of LC50 or EC50; mg/L as Fluoride ion	End point	Review source
Algae **(8 spp)**	**4 - 15**	**50 – 266**	**EC50 – growth inhibition**	**Camargo (2003); WHO (2002); SCHER (2010)**
Macrophytes **(1 sp)**	**Not Recorded**	**> 60**	**EC50 – growth inhibition**	**WHO (2002)**
Crustaceans **(9 spp acute)**	**1 - 4**	**14.6 – 354**	**LC50 / EC50**	**Hickey et al (1989); Camargo (2003); WHO (2002); SCHER (2010); AQUIRE (2010)**
(2spp chronic)	**21 - 28**	**4 – 26**	**NOEC; LOEC; EC25**	**WHO (2002); SCHER (2010)**

Signature……..……..Witnessed by……

Date……

Insects **(10 spp)**	**4 - 6**	**11.5 – 124**	**LC50**	**Camargo (2003); Metcalfe et al (2003); AQUIRE (2010)**
Rotifer **(1 spp)**	**1**	**183**	**LC50**	**AQUIRE (2010)**
Molluscs **(5 spp acute)**	**4 – 7 d**	**53 – 157**	**LC50**	**AQUIRE (2010)**
(1sp chronic)	**56 d**	**2.8**	**LC50**	**WHO (2002)**
Fish **(6 spp acute)**	**4 – 8 d**	**51 - 830**	**LC50**	**Camargo (2003); Metcalfe et al (2003); AQUIRE (2010)**
(1 sp chronic)	**90 d**	**4**	**NOEC**	
Frogs **(1 spp)**	**30 d**	**50**	**LOEC mortality***	**WHO (2002)**

*** Unreliable figure as statistical analysis and endpoint unclear**

211 The doses needed to cause harm to the species listed in Table 1 may seem very large. They range from 2.8 to 830 ppm (mg/Liter). However, it should be noted that most of the end points observed are for LETHAL concentrations for 50% of the population of the particular species studied (LC50). That means that the researchers were examining what doses would kill half of their study group. There are many things to say about this. The concentrations would be considerably reduced if they were to look at the levels that kill 25%, 10% or even 1% of the sample. Moreover, as with humans, we are not just looking at doses that kill species outright, we are looking at doses

Signature……..……..Witnessed by……

Date……

that make living things sick, or disrupt their reproductive systems, or make them less likely to survive in a very complex ecosystem over an extended period of time. Thus it is necessary and important to look at other studies that have examined more subtle aspects of the survival of aquatic species subjected to fluoride exposure. In the following paragraphs (i-iii) I do that and the court should note how much lower the concentrations are that cause harm –i.e. much, much lower than the concentrations recorded in Table 1.

i) Levels below 1.5 mgF/L have been shown to have both lethal and other adverse effects on salmon (Court case involving Meader's Trout farm in Pocatello, Idaho, see Pocatello, 1961).

ii) According to Foulkes and Anderson (1994):

> *"In a field study, "Damkaer and Dey (1989) demonstrated that high salmon loss (Chinook and Coho) at John Day Dam on the Columbia River, 1982-1986, was caused by the inhibition of migration by fluoride contamination from an aluminum smelter 1.6 km above [upstream from, PC] the dam. The average daily discharge of fluoride in 1982 was 384 kg. This was associated, at the dam, with a fluoride concentration of 0.5 mg/L and a migration time of more than 150 hours and a 55% loss. In 1983, discharge was reduced to 107 kg/day. This was associated with a reduction of concentration to 0.17 mgF/L and the migration time to less than 28 hours with a loss of 11%. In 1985, fluoride discharge of 49 kg/day was accompanied by a concentration of 0.2 mgF/L and a salmonid loss of 5%.*
>
> *Damkaer and Dey (1989) "confirmed the cause-and-effect relationship by means of a two-choice flume for fluoride gradient salmon behaviour tests. These determined that the 'critical level' was 0.2 mgF/L."*

iii) Also according to Foulkes and Anderson (1994):

> *"The following studies indicate that fluoride at levels below 1.5 mg/L have lethal and other adverse effects on fish and other aquatic species. Delayed hatching of rainbow trout occurred at 1.5 mgF/L (Ellis et al., 1938); brown mussels died at 1.4 mgF/L (Hemens et al.,*

Signature……..……..Witnessed by……

Date……..

1975) an alga (Porphyria tenera) was killed by a four-hour fumigation with fluoride with a critical concentration of 0.9 mgF/L (Ishio and Makagawa, 1971); and, levels below 0.1 mgF/L were shown to be lethal to the water flea, Daphnia magna (Dave, 1984). These latter two studies suggest that salmon species may be affected by fluoride induced reduction of food supply."

210 Groth (1975) had this to say about the laboratory studies that make up most of Table 1:

"there are serious problems with "laboratory" experiments as opposed to "field" studies."

211 Groth added that:

"the techniques of measurement may be inadequate to detect effects, and these may be at the population rather than individual level."

212 Carmargo (1995) makes a similar point to Groth's:

"Safe concentrations estimated via the multifactor probit analysis of sub lethal acute toxicity data should be validated by field investigations and chronic toxicity bioassays."

213 In paragraph 29 of JC he notes the concerns expressed in Dr. Mark Diensendorf's affidavit, that artificially fluoridated water contains:

"traces of arsenic and heavy metals (MD affidavit) and/or that the chemicals used to fluoridate water supply in the NSW Northern Rivers contain 20% w/w H_2SiF_6 (fluorosilicic acid) along with many toxic and carcinogenic contaminants including antimony, arsenic, beryllium, cadmium, chromium, lead and mercury."

214 My response. First is there no question that the contaminants that Dr. Diesendorf listed do occur in the chemicals used in fluoridation programs in Australia as chemical analysis demonstrates (see analyses in **Annexure 12**).

215 Moreover, the marine phosphate rock (i.e. *"ore")* used as feedstock in superphosphate manufacture is moderately variable in composition. It generally contains between 2 and 4 wt. % fluorine as a major contaminant, silica (chert), and also a range of other minerals which

Signature……..……..Witnessed by……

Date……

accumulate/form under reducing conditions in relatively shallow marine environments. Metallic sulfides (primarily of iron, with minor-trace zinc, cadmium, lead and mercury etc. + arsenic) and uranium oxide (UO_2, uraninite (var. comp.) + radioactive daughter nuclides) may also be present. The phosphorous is derived from decomposing organic material, which accumulates in these marine environments. The sedimentary product undergoes burial, diagenesis, dewatering, and lithification to form phosphate rock.

216 I will now respond to Dr. Chapman's criticism at paragraph 30 of his affidavit of Dr. Diesendorf's concerns.

217 At paragraph 30a Dr. Chapman states that the fluoridating chemical to be used in some of the plants in Rous water is sodium fluoride, but fails to acknowledge that the sodium fluoride used is also an industrial grade product containing some of the same toxic elements cited by Diesendorf.

218 Moreover, in 30a) Dr. Chapman acknowledges that for at least two of the water treatment plants "silico-fluoride dosing systems" will be used. Silico-fluoride is a generic name used for both H_2SiF_6 and its sodium salt, Na_2SiF_6 (described above). Both of these substances are contaminated hazardous waste products derived from the superphosphate industry and contain the toxic elements that Dr. Mark Diesendorf lists as well as trace amounts of radioactive isotopes.

219 At paragraph 30b Dr. Chapman claims that:

> *"section 8.1 of the Code of Practice provides that any impurities in the fluoridating agent 'shall not cause any health problems for consumers or result in non-compliance with the Australian Drinking Water Guidelines.'"*

220 My response: Without offering a list of primary studies that have established this claim, accepting this citation at face value adds little of scientific value to what amounts to a "legal" assertion. Moreover, the comment is directed towards human health not aquatic health. To deal adequately with Dr. Diesendorf's concerns, Dr Chapman should have examined the impacts on the aquatic ecosystem by each of these toxic contaminants at the concentrations reached after dilution of the parent fluoridating agent. Moreover, the concentrations used should be based on the 95% upper confidence level of the

Signature……..……..Witnessed by……..

Date……..

measurements made. Such measurements or estimations (from parent chemical levels) are seldom made public, and when they are they are usually not accompanied with any statistical analysis to enable independent observers to make a determination of the 95% upper confidence level.

221 Furthermore, Section "7 Environmental Safety" of the Code of Practice 2002, Section 7.1.1.1, Page 18, states:

> *"In the area of protection of the environment these Acts and Regulations will have precedence over the Fluoridation Act, Regulation and Code of Practice."*

In other words it is expected that full environmental investigation be undertaken to the fullest extent possible to guarantee safety of all species in the aquatic and terrestrial biota, including the health of humans.

222 As far as the human health impacts of these contaminants are concerned, in the US, the maximum contaminant level goals (MCLG) for both arsenic and lead are set at ZERO, because they are both human carcinogens. Put another way deliberately adding any level of arsenic or lead to the water supply will increase the cancer risk for the population. This could be serious. On occasions the arsenic level in the public water supply *(i.e. after* dilution of the fluoridating chemical) has exceeded 1 ppb (part per billion) (Wang et al., 2000). That has an estimated increased cancer risk for lifetime consumption of 1 in 1000.

223 Overall neither paragraphs 30(a) or (b) is responsive to the concerns expressed by Dr. Mark Diesendorf, cited in MD's paragraph 29).

224 In paragraph 31, Dr. Chapman acknowledges a concern expressed in the affidavit of Dr. Andrew Harms, about the ability of fluoridating chemicals to leach lead from pipes.

225 In paragraph 32a, Dr. Chapman says that he *"would be surprised if lead was even used in Australian water pipes."*

226 My response. Dr Chapman is almost certainly correct for pipes in people's houses today, or in the mains distribution system, however some lead pipes may still be present in older housing and possibly in

Signature……..……..Witnessed by……..

Date……..

some old distribution systems. However, a more likely source of lead today is either in some of the solders used in joining pipes or in brass fittings. Maas et al., (2005) showed that fluoridating chemicals in conjunction with some chlorinating agents like chloramine increase the leaching of lead from brass fittings. Moreover, two epidemiological studies by Masters and Coplan (1999) and Masters et al. (2000) found an increased lead uptake in children whose drinking water had been fluoridated with silico-fluorides. These observations while critiqued on theoretical grounds (Urbansky and Schock, 2000) have received some support from laboratory studies where Sawan et al., 2010 showed increased uptake of lead into plasma, teeth and bone when rats were given lead in the presence of hexafluorosilicic acid, compared to being given lead alone.

227 In paragraph 32(a), Dr. Chapman does acknowledge that fluoride solubilizes the aluminium ion. This could be important because it is known that aluminium is toxic for some aquatic species. Dr. Chapman does not pursue this matter. Locally, this is an issue of concern. Aluminium from soil and sediment dissolves in acidic water. This is of major concern in the Richmond river system, which suffers from large-scale acid sulphate events, which make waters acid and thus release toxic levels of aluminium. So we have two possible threats here: one, is the threat of aluminium itself on aquatic species and two, is that aluminium in conjunction with fluoride is more toxic than either alone. Aluminium forms a complex with fluoride that switches on G-proteins which are used to send hormonal, growth factor and neurotransmitter signals across membranes (Strunecka and Patocka, 1999; Strunecka, Strunecky and Patocka, 2002; Strunecka, Patocka and Connett, 2004)

228 In addition, fluoride is known to form complexes with many other metal ions (both toxic and non-toxic). In fact it forms complexes with every metal ion except the group I metals (lithium, sodium, potassium etc). Dr. Chapman provides no information about what effect this solubilization of other metal ions from rocks and soils might have on aquatic species.

229 Thus, the rejection by Dr. Chapman in paragraph 32(c) of Dr. Andrew Harms' concerns is without merit, since it appears in this case Dr. Chapman is unaware or fails to refer to the relevant literature.

Signature……..……..Witnessed by……..

Date……..

230 In paragraph 34 Dr. Chapman attempts to alleviate concerns about adding a known toxic substance into the environment. I will respond to each paragraph in 34 separately.

231 At paragraph 34 (a) Dr. Chapman correctly states the difference between toxicity and hazard. A toxic substance is only a hazard if it exceeds certain exposure levels and has to take into account factors that may modify toxicity and exposure levels. The process involved in moving from intrinsic toxicity of a substance to the hazard it presents, in a specific situation, is called risk assessment. We have already seen above some of the limitations of Dr. Chapman's risk assessment process. For example he did not deal with other contaminants in the fluoridating chemicals, and while he acknowledged the fact that fluoride increased the dissolution of aluminium from soils, he did not investigate the local ramifications of this fact.

232 In the paragraphs below I will discuss other limitations of Dr. Chapman's risk assessment, both in general terms and in terms of the specific locations where the fluoridation will take place.

233 Paragraph 34 (b) deals with dilution of the incoming water at the sewage treatment plant with other water supplies, possible absorption by biological treatment processes or precipitation as calcium fluoride by the addition of calcium sulfate. Dr. Chapman states:

> *"Although I do not expect fluoride ion to be strongly absorbed within the sewage treatment process, I also do not expect that the concentration of fluoride ion would remain at the level of 1 mg/L ("Approved Concentration") in STP effluent. Most of the reduction would be due to dilution from other waste streams, groundwater input, diversion of water elsewhere (eg watering gardens) and any rainfall input. Biological treatment, such as in secondary treatment plants, would also remove some fluoride (Osterman (1990)). This accords with the literature on this topic, discussed below in paragraph [34(d)]. In addition, any dosing with calcium sulfate would remove fluoride as calcium fluoride, which is relatively insoluble".*

234 In paragraph 34 (b) Dr. Chapman indicates that *"any dosing with calcium sulfate"* would remove fluoride as calcium fluoride. However, he doesn't give any details of calcium sulfate treatment. In fact, it is unrealistic to suggest that dosing with calcium sulfate (solubility of ~2100

Signature……..……..Witnessed by……

Date……..

mg/L at 20 ^{o}C (or ~ 615 ppm Calcium equiv.)) would significantly lower the dissolved fluoride concentration (already at such low levels) by precipitating relatively insoluble calcium fluoride (solubility of ~16 mg/L (or ~ 8 ppm Fluoride equiv.; Handbook of Physics and Chemistry (1962)). Fluoride in seawater is ~1.4 ppm where dissolved calcium, magnesium and sulfate are 400 ppm, 1300 ppm, and ~900 ppm, respectively.

235 Dr. Chapman states (34b) that

> *"most of the reduction of the (fluoride entering the sewage treatment plants) would be due to dilution from the other waste streams, groundwater input, diversion of water elsewhere (e.g. watering gardens) and any rainfall input."*

236 My response to 34 (b) is as follows:

i) Any diversion of the water via watering of gardens would not *dilute* the water entering or exiting the sewage plant, but only reduce the *total input* of fluoride into the sewage treatment plant. This diversion would also incur direct impacts on exposing growing plants and vegetable to *undiluted* fluoridated water. Such impacts were not discussed by Dr. Chapman. Nor did Dr. Chapman discuss other diversions of fluoridated water directly into the environment. There is some evidence that up to 10% of the water delivery system is lost through leakage. Dr. Chapman does not discuss the fate of such leaks or the impact of this undiluted fluoridated water on the environment.

ii) There will be little dilution from other waste streams flowing into the sewage treatment plants when there is little rain, thus diminishing any dilution via these sources. This area is subjected to intermittent droughts.

iii) Most of the information supporting Dr. Chapman's claim that the sewage plant will dilute the incoming fluoridated water to yield lower fluoride levels is derived from just two facilities, one in Sydney (NSW) (discussed in 34c) and the other in Montreal, Canada (discussed in 34 d). I will discuss each in turn.

237 In paragraph 34(c) Dr. Chapman tells us that he got his information on the Sydney plant from a conversation with Dr. Steve Blockwell at

Signature……..……..Witnessed by……..

Date……..

the Sydney Water Corporation (SWC), who informed him that

> *"SWC has measured fluoride once a year over 10 years in the effluent from about 10 plants that supply water for irrigation and other recycled water uses. The mean level of fluoride in effluent was 0.7 mg/L, with a range of 0.14 to 1.1 mg/L. Some of this may have been the natural component of fluoride or some industrial input."*

The SWC *"once a year"* measurements do not provide or define an accurate scientific assessment for the variation of fluoride levels throughout the rest of the year. Even if we can take such sparse measurements at face value, they are not reassuring when comparing with the Canadian guideline of 0.12 mg/L to protect aquatic species (Canadian Water Quality Guidelines 2001).

238 In paragraph 34 (d) Dr. Chapman provides some information about sewage outflow levels for Montreal, Canada. My response: to the best of my knowledge the city of Montreal has never been fluoridated so I am puzzled by this discussion. One community (Dorval) located on the edge of Montreal has been fluoridated and maybe the results have been derived from that community, but Dr. Chapman does not provide any details.

239 Further at paragraph 34 (d) Dr. Chapman cites studies by Osterman (1990) who observed:

> *"raw sewage from 7 fluoridated communities in Ontario, Canada contained 0.96 mg/L fluoride compared to 0.49 mg/L from 11 non-fluoridated communities."*

Again, this result is not reassuring when comparing with the Canadian guideline of 0.12 mg/L (Environment Canada, 2001) or even Dr. Chapman's own estimated safe guideline to protect aquatic species of 0.8 mg/L (see 34 (f), contradicting his own claim in 34 (h) that:

> *"the likely concentration of effluent from sewage treatment plants receiving fluoridated water is well below 'safe concentrations' reported for aquatic organisms" (WHO, 2002; Carmago et al., 2003).*

240 Foulkes and Anderson (1994) cite other effluent studies suggesting Dr. Chapman is being optimistic about fluoride reduction at sewage treatment plants. They state:

Signature……..……..Witnessed by……..

Date……..

"Masuda [1964, PC] studied a large number of cities and calculated the [fluoride, PC] concentrations in waste water that were in excess of the concentration present in the cities' water supplies. In raw sewage, this was 1.30 ppm; primary treatment reduced this slightly to 1.28 ppm; secondary treatment to 0.39 ppm. Singer and Armstrong [1977,PC] found 0.38 ppm in unfluoridated sewage and 1.16-1.25 ppm in fluoridated sewage."

241 With very little fluoride concentration reduction in sewage treatment plants Dr. Chapman's claims of safety largely relies upon the dilution and environmental interactions after the water leaves the sewage treatment plant. It is worth citing his last two paragraphs (i.e. 34 k and l) in full:

"34 (k) The remaining five plants discharge into freshwater streams of various sizes and/or have partial reuse of water onto land. The two Lismore plants discharge into Monaltrie Creek and Hollingsworth Creek for 2 — 2.5 km before discharging into the Wilsons River, where the 95th percentile flow is estimated at 41 MLA time. Hence, any added fluoride, after being reduced by around 50% through the STPs would be diluted by the natural river flow by a factor of between 5 and 8 times. The Casino STP, on Spring Grove Road, discharges to the freshwater section of the Richmond River, which also provides considerable dilution. The remaining STPs at Nimbin and Alstonville discharge to smaller creeks with lesser dilution, although there is a proportion of discharge to land. However, the water in the rivers in this region drain mostly basalt, which I would expect to produce water of hardness of soft to moderately hard. Hence the toxicity and environmental risk of fluoride would be further reduced.

34 (l) Hence the local receiving environment further underlines the general conclusion from literature and toxicity data that there is negligible risk to aquatic organisms from fluoridation of water supply."

242 My response to paragraphs 34 (k) and (l) is firstly to reject the outdated notion that *"the answer to pollution is dilution."* For example, while flowing streams can dilute, aquatic and other living species can bio-concentrate toxic substances like heavy metals and fluoride. Moreover, at certain times of the year – particularly during low rainfall and dry seasons - the dilution offered by the creeks and streams in the area could be very slight indeed. Before any firm conclusions can be made about the *"dilution factor"* very careful year-

Signature……..……..Witnessed by……

Date……

long calculations need to be made on the creeks and streams in the area destined to receive the fluoridated water. In other words, a full scientific environmental impact analysis needs to be carried out.

243 The information contained in Dr. Chapman's affidavit is inadequate for the purposes of evaluating the ecotoxicity of fluoride releases from the proposed fluoridation project by Rous Water. Some of the inadequacies reflect the limitations of Dr. Chapman's own analysis of the existing data, but it is also a reflection of the limited local data made available to him by the proponent of this project. The proponent does not estimate the amount of fluoride (plus any contaminants) that will be released to local creeks and estuaries on a yearly basis. Nor have background studies been provided of local aquatic systems likely to be affected.

244 It would appear that local waters are readily concentrated by evaporation during dry hot periods and limited rainfall. Fluoride levels can concentrate in pools in low flow creeks rather than dilute. Aquatic organisms become stressed by high water temperature, contaminants, lack of dissolved oxygen and accumulation of salts. Under these circumstances there may be little margin of safety from exposing sensitive and stressed species to elevated levels of fluoride.

245 There is also reason to believe that Dr. Chapman's estimations about swift dilution when the fluoride enters local flowing rivers and streams is overly optimistic. According to Foulkes and Anderson (1994):

> *"studies show that elevated concentrations in fresh water receiving fluoridated effluent may persist for some distance. Bahls [1973, PC] showed that the effluent from Bozeman Montana of 0.6-2.0 mgF/L, discharged into the East Galletin River did not return to the background level of 0.33 mgF/L for **5.3** km. Singer and Armstrong [1977, PC] reported that a distance of **16** km was required to return the Mississippi River to its background level of 0.2 mg/FL after receiving the effluent of 1.21 mgF/L from Minneapolis-St Paul."*

246 Dr. Chapman's assertion in paragraph 34 (k) that the basalt in the area will change the hardness of the water and ameliorate the toxicity of fluoride offers only one possible environmental interaction. Other fluoride interactions may actually increase the toxicity of the water

Signature……..……..Witnessed by……

Date……..

and the toxicity of the fluoride when considering fluoride's ability to enhance the dissolution of aluminum and other toxic metals from soils, rocks and sediments.

247 Dr. Chapman's claim that fluoride levels in the sewage effluent will be reduced *"by around 50%"* in local STPs (sewage treatment plants) is a best case scenario rather than a worst case scenario for the data he offered in paragraphs 34 (c) and (d).

248 In relation to aquatic species a series of citations are offered below, which in my view, indicate that Dr. Chapman's suggestions that 0.8 mg/L is a safe level sufficient to protect 95% of aquatic species throughout their entire lifecycles, is overly optimistic.

i) Camargo (2003), who Chapman cites several times, in a review of fluoride toxicity to aquatic organisms recommended a safe level for fluoride in freshwaters of below 0.5mg/L because of adverse effects at 0.5mg/L in sensitive fish and invertebrates in low ionic or soft waters.

ii) Camargo (2003) states:

" even though safe levels of fluoride for aquatic life have not been yet determined (USEPA, 1986), it should be evident from the data presented in this review that discharges from anthropogenic sources (i.e., fluoride pollution) may result in a serious ecological risk for aquatic organisms."

iii) Camargo (2003) also states:

"Regarding aquatic animals, most studies have concerned with acute/ short-term laboratory experiments and, consequently, more chronic/long-term studies would be needed to verify and improve safe levels of fluoride. Lastly, because inorganic fluoride bioaccumulates in aquatic producers (algae and macrophytes) as well as in aquatic consumers (invertebrates and fishes), studies on the biotransference and biomagnification of fluoride through food chains and food webs would have to be carried out." (Camargo 2003. Section 6, p. 261, last paragraph).

iv) Canadian Water Quality Guidelines (Environment Canada, 2001) indicate that there are extremely sensitive species to fluoride at

Signature……..……..Witnessed by……

Date……..

0.5 mg/L such as rainbow trout, salmon, algae, seaweeds, insects [e.g. caddis fly] and microorganisms and half that concentration can cause disruptions to their reproduction and growth size quality and quantity stating:

"The guideline value for inorganic fluorides is a numerical limit intended to protect all forms of aquatic life and all aspects of aquatic life cycles, including the most sensitive life stage of the most sensitive species over the long term." (Environment Canada, Section 1, Page 1 Paragraph 2)

v) Foulkes and Anderson (1994) also explain that the Canadian Province of British Columbia (BC) had *"recommended"* a *"guideline"* of 0.2 mg F/L to protect aquatic species but as of 1994 it had yet to become an enforceable regulation. However, the point is moot since it has been superseded by the new National Canadian Water Quality Guidelines (see vi).

vi) The current National Canadian Water Quality Guidelines (Environment Canada, 2001) is set at 0.12 mg/L designed to protect *all* forms of aquatic biota, not just 95% of them (as proposed in Dr. Chapman's guideline). Every organism is essential for a healthy thriving ecosystem.

vii) Dave (1984) showed that levels below 0.1 mgF/L were lethal to the water flea, Daphnia magna (cited in Foulkes and Anderson, 1994).

249 In my view the comment in **iv)** above draws attention to an underlining weakness in Dr. Chapman's analysis. He has focused on the fluoride concentrations causing a toxic or even lethal effect on a limited number of aquatic species. He fails to give regard to the subtleties of damaging a species that may be of critical importance in the life cycle of other species. The Canadian authorities appear to be far more aware of this matter. As follows:

"Inorganic fluorides at low concentrations in the aquatic environment can elicit slight effects that are statistically significant and ecologically relevant. Sensitive characteristics for fish include survival, growth, reproduction, and behavioural endpoints." (Environment Canada,

Signature……..……..Witnessed by……

Date……

2001, Section 8.2, Page 54 Paragraph 3).

"An interim water quality guideline of 0.12 mg F/L is recommended for the protection of all stages of freshwater life against the adverse affects of total inorganic fluorides. It was derived by multiplying the lowest value from an acute, static toxicity study on the net-spinning caddisfly, Hydropsyche bronta,by Camargo 1995, a LC50 of 11.5 mg F1L, by a safety factor of 0.01." (Environment Canada, 2001, Page vii, last Paragraph).

These Canadian concerns are further underlined by US researchers Rasmussen, Dana & Denson *'Status of Caddisflies (insecta:trichoptera) in Greatest Conservation Need in Florida 2008, by the Florida Fish and Wildlife Conservation Commission on the "net spinning caddisfly"*

"Trichoptera (caddisflies), the largest order of aquatic insects, contains over 12,000 extant species worldwide, approximately 1300 species in North America, and nearly 200 species in Florida. ***Most species are intolerant of pollution and are considered excellent biological indicators of freshwater ecosystem health****...However, to fully realize the utility of this group in conservation planning and implementation, it is important that as much information as possible is documented in regards to species geographic distributions, habitat associations, and overall population levels and trends." (emphasis added)*

250 The importance of Caddisfly in the Australian context is further confirmed by the "*Natural Heritage Trust"* C'wealth Govt. in: *'A Review of the Conservation Status Australian of Selected Non Marine Invertebrates'* p.114.

"There are approximately 600 species of Caddisflies in Australia, they are quite often overlooked, probably because of their typically drab colours and nocturnal behaviour. But they do play an important role in measuring environmental health. As larvae, they live under water and certain species have certain requirements for temperature, oxygen and other chemical levels, so an abundance or lack of a particular species can give a good indication of the quality of the water."

251 Dr Chapman deposes at paragraph 4 of his affidavit that:

Signature……..……..Witnessed by……..

Date……..

"I led the team that revised the toxicant guidelines for the Australian and New Zealand Guidelines for Fresh & Marine Water Quality in 2000 and I am involved in the current revision, commencing in 2009."

My response: Dr Chapman states he led the team that revised, the abovementioned guidelines. The affidavit failed to disclose that the 2000 guidelines had insufficient evidence to determine the safe levels of fluoride on marine and aquatic species. The " *recommended guidelines"* for freshwater aquaculture in the Table 9.4.22 - Page 9.4.38 of the 2000 guidelines (as reproduced below) based on Tebbutt (1977) would indicate that the impact of fluoride has a much lower safety margin than expressed by Dr Chapman throughout his affidavit.

Table 9.4.22 Summary of the recommended water quality guidelines for fluoride

Group	Guideline mg/L	Comments	Reference
Recommended guidelines	<0.2 ND	freshwater saltwater	based on Tebbutt (1977)
Freshwater fish	0.2–1.0		Tebbutt (1977)
Edible bivalves	<0.025 <30.0	blue mussel Sydney rock oyster spat 20% growth reduction	Pankhurst et al. (1980) Nell & Livanos (1988)

ND: Not determined — insufficient information

252 Dr. Chapman's analysis does not sufficiently take into account either the local weather conditions or the physical parameters of local creeks and rivers. Furthermore, he does not take into account local sensitive species, some of which are very important for the local environment including fry, prawns and oysters as well as all the other species that support their life cycles. In the case of fry this would include the *"caddisfly",* discussed above.

253 Dr. Chapman does not address the issue of fluoride being retained in the sediments of streams and rivers or when it is carried to the estuary where it may persist for many years. (Carpenter, 1969). This retained fluoride may re-contaminate if dredging were to take place or during intense flooding events.

254 The addition of artificial fluoridated water into the Northern Rivers

Signature……..……..Witnessed by……

Date……

environment should not be considered in isolation but rather as contributing to the fluoride entering the environment from all sources combined. For example, a calculation should be made of the fluoride that washes out of farmland treated with phosphate fertilizer (which contains high levels of fluoride) as well as pesticides containing fluoride.

255 Superphosphates containing inorganic fluorides are used extensively in agriculture in the Rous water catchment. According to Kabata - Pendias and Pendias, 1984,

> "*the inorganic fluoride content of the superphosphate fertilizer used for farming contains as much as 38 mg of fluoride per kg contributing to an estimated accumulation of an additional 89 mg F/kg in soil over a ten year period. (cited in Environment Canada, 2001, Section 3.2, Page 12, Paragraph 1.)*

256 The Richmond River is already under stress and in recent years there have already been two significant fish kills after flooding events. The proposed additional extra fluoride from five fluoride dosing plants entering the aquatic and terrestrial biota has not been assessed.

257 Dr Chapman has failed to consider the impact of fluoridation on mammals. Above I have discussed the many studies indicating that fluoride can damage human health, and lower IQ in children also the NRC (2006).

258 There have been over 100 studies indicating that fluoride can damage animal brain (a full list of these studies can be found in Appendix 1 of my book *The Case Against Fluoride*, see annexure 4).

259 Dr Phyllis J Mullinex (Mullinex et al 1995) became well known in the field of neurotoxicology by using a sophisticated Computer Pattern Recognition System (she helped to develop) analyzing the effects of fluoride on rat brain (Mullenix et al., 1995). Since this study there have been many more animal studies on the brain and 24 studies that fluoride is associated with lowering of IQ in children, which is something that Mullenix predicted.

260 Mullenix's study was criticized because she used fairly high levels of fluoride in her experiment but Varner et al. 1998, found effects on rat brain at very low levels of exposure. They fed rats drinking water containing only 1 ppm fluoride for one year (either as aluminium

Signature……..……..Witnessed by……

Date……

fluoride or sodium fluoride). The animals developed kidney damage, brain damage and had a greater uptake of aluminium into the brain with the formation of beta-amyloid deposits that some researchers believe are associated with Alzheimer's disease. This is a remarkably low dose when one knows that it takes 5-10 times as much fluoride for a rat to reach the same plasma levels as a human. Such a result has implications for other small terrestrial native fauna should they be exposed to fluoridated water or discharges from the sewerage treatment works.

261 In relation to livestock consumption of water The Canadian Water Quality Guidelines 2001, at page 35 states:

> *"Cattle accumulate 10,000 mg F/kg of bone compared to normal levels of 1,500 mg F/kg (Warrington 1990). Dairy cattle have been found to be the most sensitive livestock to fluoride toxicity as they have high food and water uptake rates and long productive lives. This results in the maximal opportunity for fluorides to accumulate to harmful levels in dairy cows and eventually end up in future generations." (Environment Canada, 2001, Section 6, Page 35, Paragraph 2)*

262 In addition to farm animals there are concerns about fluoride's impact on domestic animals, especially dogs. According to Olga Naidenko, PhD, Senior Scientist, June 2009 in a commissioned report for the US Environmental Working Group, the levels of fluoride in many dog foods are very high. She stated:

> *"Eight major national brands marketed for both puppies and adults contained fluoride in amounts between 1.6 and 2.5 times higher than the Environmental Protection Agency's maximum legal dose in drinking water, and higher than amounts associated with bone cancer in young boys in a 2006 study by Harvard scientists (Bassin 2006). All 8 brands contain bone meal and animal byproducts, the likely source of the fluoride contamination".*

263 The dog is one of the few animals that succumbs to osteosarcoma, and larger dogs commonly lose strength in their rear legs. Fluoride is known to cause arthritic-like symptoms and is suspected of causing osteosarcoma in both rats and humans. With the possibility that dogs are getting high levels of fluoride in pet food especially pet food that

Signature……..……..Witnessed by……..

Date……..

contains bone-meal and with the further addition of fluoridated drinking water would only exacerbate their overall fluoride intake.

264 Tasmanian sheep farmers Sylvia and John Braim sued the Tasmanian government for the death of their pedigree cattle and sheep. Recorded veterinary diagnosis indicated that the animals died of acute fluoride poisoning with results that stomachs contained more than 250 ppm of fluoride. Some of the animals were still born and others had deformed hooves that were three times the normal size. **Annexure 13.**

265 Animals consuming large amounts of water especially, such as cattle and horses are vulnerable to skeletal and dental fluorosis from fluoridated water. One of the pioneers in researching the impacts of fluoride on farmyard animals was Dr. Lennart Krook, a veterinary pathologist at Cornell University. He investigated the mysterious illnesses affecting a number of quarter horses on Cathy and Wayne Justus's farm in Pagosa Springs, Colorado. Some of the horses died. It was only when Cathy and Wayne Justus changed the horses' water supply that the symptoms of the remaining animals began to clear up. Dr. Krook was able to show that the likely cause of the horses' ailments was fluoride. He had the bones analyzed for fluoride and they had very high levels. Photos of the horses' clearly fluorosed teeth and the deformities observed in the horses' hooves were consistent with fluorosis. This event led to the halting of fluoridation in Pagosa Springs. The Krook and Justus study was published in the journal *Fluoride* in 2006 (Krook and Justus, 2006). See also Justus and Krook (2006); Burgstahler (2006); Macicek and Krook , (2008); Choubisa (2010) and Spittle (2010). **Annexure 14** contains one of the Krook and Justus's papers on this issue.

266 Krook and Justus (2006) also noted that impaired conception occurred in mares after consuming fluoridated water for five years. Similarly, fluoride has been found to reduce *in vitro* male fertility in rats at a serum level of about 0.3 ppm F (Izquierdo-Vega et al., 2008) and *in vivo* fertility in several other animal species (rats, chinchillas, alligators, and caimans) when they drink municipal water fluoridated with fluorosilicic acid, H_2SiF_6 (Burgstahler et al., 2008).

267 Long et al. reviewed the fluoride toxicity in the male reproductive system in humans in 2009. These authors explained that the most

Signature……..……..Witnessed by……

Date……..

important consequences of environmental fluoride exposures were: "changes in the structure and functional behavior of spermatozoa, disruption of spermatogenesis, and disturbances of multiple hormone systems that impact male reproduction."

268 Long et al. (2009) further explained that, "There is evidence that fluoride interferes with spermatogenesis by depressing levels of epidermal growth factor and epidermal growth factor receptor, modifying G-protein signaling (discussed above), diminishing levels of testosterone and its androgen receptor and disturbing levels of estradiol. Furthermore, fluoride is also known to interfere with thyroid hormone metabolism (also discussed above), which directly and indirectly impacts not only spermatogenesis but also other reproductive functions. Although fluoride appears to exert its toxic effects in the male reproductive system through these pathways, the molecular details are still poorly understood. The growing evidence that fluoride overexposure leads to male reproductive toxicity through multiple pathways indicates that an assessment of chronic fluoride exposures in human and animal populations is urgently required."

269 It has been reported in the *Melbourne Age* on Feb 21st, 2010 that:

> *"Roos have become the victims of factory fluoride emissions. Scores of starving and pain-ridden kangaroos have been culled after developing tooth and bone deformities from breathing and ingesting fluoride emissions. Many more are believed to be suffering from growths that will kill them. The affected kangaroos are living near the Alcoa aluminium smelter in Portland, in the state's south-west, and the Austral Bricks factory at Craigieburn. Autopsies performed at Melbourne University on 49 kangaroos culled at Alcoa on a single day last year found all but one were suffering from fluorosis, which leads to excessive bone growths, or lesions, on joints in the paws, ankles and calves. It can also cause tooth and jaw deformities that hinder eating and foraging."*

270 According this report these kangaroos are a perfect example of the bio-magnification of fluorides. Not only were they breathing in air emissions but they were also exposed to fluoride consuming contaminated feed (grasses) and water.

Signature……..……..Witnessed by……..

Date……..

271 According to *Canadian National Water Quality Guidelines*:

> *"Terrestrial plants take up inorganic fluorides directly from air by their leaves or indirectly from rain or groundwater by their roots. Airborne inorganic fluorides are probably the most important source of inorganic fluorides for plants. Levels of inorganic fluorides in rainwater appear to be positively correlated with levels in plants (Kabata-Pendias and Pendias 1984). Animals take up inorganic fluorides by feeding on these plants or drinking water. Murray (1981) suggests a possible cycling from soils to vegetation and from vegetation to wildlife. Animal death and plant decomposition return fluorides to the soil and ultimately, to groundwater. In surface waters, inorganic fluorides are taken up by plants, and aquatic, semi-aquatic, and terrestrial biota." (Environment Canada, 2001, p.21)*

Conclusion

272 It is simplistic to assume that fluoride at 1 ppm, or even at 0.8 mg/L, as claimed by Dr. Chapman, will have a benign effect on either human health or the ecology of the receiving biota. Neither 0.8 nor 1 ppm is small. They are actually 200 and 250 times the level of fluoride in mothers' milk (NRC, 2006, p.40). Even though fluoridation of public water supplies is regulated and controlled at an approved *concentration* of 1ppm, giving the impression that at this concentration it is safe and effective, this completely misses the crucial point. Once fluoride is added to drinking there is no control of the dose received by humans, animals or fish. Without that control both short-term and long-term effects are theoretical and if nothing else the "Precautionary Principle" should insist that we avoid this unnecessary, unethical and highly problematic practice. In this respect India, China, Japan and the vast majority of European countries have demonstrated far more wisdom than the US, Australia and the handful of other countries that continue to fluoridate their water supplies.

273 It is puzzling why Australian authorities continue to force this practice on people despite the evidence that swallowing fluoride does little to fight tooth decay (fluoride's predominant mechanism of action in preventing tooth decay is topical not systemic, CDC, 1999) and that there is no adequate *margin of safety* to protect the whole population from known harmful effects' including lowering of IQ in

Signature……..……..Witnessed by……

Date……..

children at levels as low as 1.9 ppm (Xiang et al., 2003 a,b).

274 It is particularly disturbing that in Australia very little scientific effort has gone into conducting primary health studies on communities that have been fluoridated, nor investigated in a rigorous manner the many anecdotal reports that about 1% of the population may be highly sensitive to fluoride, nor made any effort to monitor exposure to fluoride by measuring fluoride in bones, plasma, urine, hair or nails. Not one among Drs. Blinkhorn, Wright, Brown, Spencer and Chapman, mentioned these serious shortcomings, even while extolling the "safety and effectiveness" of the program.

References:

1) **Twenty-four human studies that report an association of lowered IQ with fluoride exposure.**

1) Y. Chen, F. Han, Z. Zhou, et al., "Research on the Intellectual Development of Children in High Fluoride Areas," Fluoride 41, no. 2 (2008): 120–24, (originally published in 1991 in Chinese Journal of Control of Endemic Diseases), http://www.fluorideresearch.org/412/files/ FJ2008_v41_n2_p120-124.pdf.

2) Y. Ding, Y.Gao, H. Sun et al., "The relationships between low levels of urine fluoride on children's intelligence, dental fluorosis in endemic fluorosis areas in Hulunbuir, Inner Mongolia, China, J. Hazardous Materials (in press) 2011.

3) X. Guo, R. Wang, C. Cheng, et al., "A Preliminary Investigation of the IQs of 7–13 Year Old Children from an Area with Coal Burning-Related Fluoride Poisoning," Fluoride 41, no. 2 (2008): 125–28 (originally published in 1991 in Chinese Journal of Endemiology),

http://www.fluorideresearch.org/412/files/FJ2008_v41_n2_p125-128.pdf.

4) F. Hong, Y. Cao, D. Yang, and H. Wang, "Research on the Effects of Fluoride on Child Intellectual Development Under Different Environmental Conditions," Fluoride 41, no. 2 (2008): 156–60 (originally published in 2001 in Chinese

Signature……..……..Witnessed by……

Date……

Primary Health Care),

http://www.fluorideresearch.org/412/files/FJ2008_v41_n2_p156-160.pdf.

5) X. S. Li, J. L. Zhi, and R.O. Gao, "Effect of Fluoride Exposure on Intelligence in Children," Fluoride 28, no. 4 (1995): 189–92,

http://fluoridealert.org/scher/li-1995.pdf.

6) Y. Li, X. Jing, D. Chen, L. Lin, and Z. Wang, "Effects of Endemic Fluoride Poisoning on the Intellectual Development of Children in Baotou," Fluoride 41, no. 2 (2008): 161–64 (originally published in 2003 in Chinese Journal of Public Health Management),

http://www.fluorideresearch.org/412/files/FJ2008_v41_n2_p161-164.pdf.

7) F. F. Lin, Aihaiti, H. X. Zhao, et al., "The Relationship of a Low-Iodine and High-Fluoride Environment to Subclinical Cretinism in Xinjiang," Xinjiang Institute for Endemic Disease Control and Research; Office of Leading Group for Endemic Disease Control of Hetian Prefectural Committee of the Communist Party of China; and County Health and Epidemic Prevention Station, Yutian, Xinjiang, Iodine Deficiency Disorder Newsletter 7, (1991): 3,

http://fluoridealert.org/scher/lin-1991.pdf; also see

http://www.fluoridealert.org/IDD.htm.

8) S. Liu, Y. Lu, Z. Sun, et al., "Report on the Intellectual Ability of Children Living in High-Fluoride Water Areas," Fluoride 41, no. 2 (2008): 144–47 (originally published in 2000 in Chinese Journal of Control of Endemic Diseases), http://www.fluorideresearch.org/412/files/FJ2008_v41_n2_p144-147.pdf.

9) Y. Lu, Z. R. Sun, L. N. Wu, et al., "Effect of High-Fluoride Water on Intelligence in Children," Fluoride 33, no. 2 (2000): 74–78,

http://www.fluorideresearch.org/332/files/FJ2000_v33_n2_p74-78.pdf.

10) L. Qin, S. Huo, R. Chen, et al., "Using the Raven's Standard Progressive Matrices to Determine the Effects of the Level of Fluoride in Drinking Water on the

Signature……..……..Witnessed by……

Date……

Intellectual Ability of School-Age Children," Fluoride 41, no. 2 (2008): 115–19 (originally published in1990 in Chinese Journal of the Control of Endemic Disease),

http://www.fluorideresearch.org/412/files/FJ2008_v41_n2_p115-119.pdf.

11) D. Ren, K. Li, and D. Liu, "A Study of the Intellectual Ability of 8–14 Year-Old Children in High Fluoride, Low Iodine Areas," Fluoride 41, no. 4 (2008): 319–20 (originally published in 1989 in Chinese Journal of Control of Endemic Diseases),

http://www.fluorideresearch.org/414/files/FJ2008_v41_n4_p319-320.pdf.

12) D. Rocha-Amador, M. E. Navarro, L. Carrizales, et al., "Decreased Intelligence in Children and Exposure to Fluoride and Arsenic in Drinking Water," Cadernos de Saúde Pública 23, suppl. 4 (2007): S579–87.

13) B. Seraj, M. Shahrabi, M. Falahzade, et al., "Effect of High Fluoride Concentration in Drinking Water on Children's Intelligence," Journal of Dental Medicine 19, no. 2 (2007): 80–86. Note: English translation forwarded by lead author (B. Seraj, department of pediatric dentistry, faculty of dentistry, Tehran University of Medical Sciences), http://fluoridealert.org/scher/seraj-2007.trans.pdf.

14) M. H. Trivedi, R. J. Verma, N. J. Chinoy, et al., "Effect of High Fluoride Water on Intelligence of School Children in India," Fluoride 40, no. 3 (2007): 178–83,

http://www.fluorideresearch.org/403/files/FJ2007_v40_n3_p178-183.pdf.

15) G. Wang, D. Yang, F. Jia, and H. Wang, "A Study of the IQ Levels of Four- to Seven-Year-Old Children in High Fluoride Areas," Fluoride 41, no. 4 (2008): 340–43 (originally published in 1996 in Endemic Diseases Bulletin [China]),

http://www.fluorideresearch.org/414/files/FJ2008_v41_n4_p340-343.pdf.

16) S. Wang, H. Zhang, W. Fan, et al., "The Effects of Endemic Fluoride Poisoning Caused by Coal Burning on the Physical Development and Intelligence of Children," Fluoride 41, no. 4 (2008): 344–48 (originally published in 2005 in Journal of Applied Clinical Pediatrics [China]),

Signature……..……..Witnessed by……

Date……

http://www.fluorideresearch.org/414/files/FJ2008_v41_n4_p344-348.pdf.

17) S. X. Wang, Z. H. Wang, X. T. Cheng, et al., "Arsenic and Fluoride Exposure in Drinking Water: Children's IQ and Growth in Shanyin County, Shanxi Province, China," Environmental Health Perspectives 115, no. 4 (2007): 643–47, http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1852689/.

18) Q. Xiang, Y. Liang, L. Chen, et al., "Effect of Fluoride in Drinking Water on Children's Intelligence," Fluoride 36, no. 2 (2003): 84–94, http://www.fluorideresearch.org/362/files/FJ2003_v36_n2_p84-94.pdf.

Also see

Q. Xiang, Y. Liang, M. Zhou, and H. Zang, "Blood Lead of Children in Wamiao-Xinhuai Intelligence Study" (letter), Fluoride 36, no. 3 (2003):198–99, http://www.fluorideresearch.org/363/files/FJ2003_v36_n3_p198-199.pdf.

19) L. B. Zhao, G. H. Liang, D. N. Zhang, and X. R. Wu, "Effect of High-Fluoride Water Supply on Children's Intelligence," Fluoride 29, no. 4 (1996): 190–92, http://fluoridealert.org/scher/zhao-1996.pdf.

The following five Chinese I.Q. studies have not yet been translated:

20) J. A. An, S. Z. Mei, A. P. Liu, et al., "Effect of High Level of Fluoride on Children's Intelligence" (article in Chinese), Zhong Guo Di Fang Bing Fang Zhi Za Zhi 7, no. 2 (1992): 93–94.

21) Z. X. Fan, H. X. Dai, A. M. Bai, et al., "Effect of High Fluoride Exposure on Children's Intelligence" (article in Chinese), Huan Jing Yu Jian Kang Za Zhi 24, no. 10 (2007): 802–3.

22) Y. L. Xu, C. S. Lu, and X. N. Zhang, "Effect of Fluoride on Children's Intelligence" (article in Chinese), Di Fang Bing Tong Bao 9 (1994): 83–84.

23) L. M. Yao, Y. Deng, S. Y. Yang, et al., "Comparison of Children's Health and

Signature……..……..Witnessed by……

Date……

Intelligence Between the Fluorosis Area with Altering Water Source and Those without Altering Water

Source" (article in Chinese), Yu Fang Yi Xue Wen Xian Xin Xi 3, no. 1 (1997): 42–43.

24) J. W. Zhang, H. Yao, and Y. Chen, "Effect of High Level of Fluoride and Arsenium on Children's Intelligence" (article in Chinese), Zhong Guo Gong Gong Wei Sheng Xue Bao 17, no. 2 (1998): 119.

2) **Other references cited in Paul Connett's affidavit**

AIHW(Australian Institute Health and Welfare) (1998). Review of Water Fluoridation: New Evidence in the 1990s. Final report" (A Consultancy by Social & Preventative Dentistry, The University of Adelaide, for The Public Health Division, Victorian Dept of Human Services)

AIHW(Australian Institute Health and Welfare) (2009). *Australia's Dental Generations.* The National Survey of Adult Oral Health 2004- 2006"

Alarcon-Herrera MT, et al. (2001). Well water fluoride, dental fluorosis, bone fractures in the Guadiana Valley of Mexico. Fluoride. 34(2): 139-149. http://www.fluoride-journal.com/01-34-2/342-139.pdf

Arora, A. and Evans, RE (2010). Dental Caries in children: a comparison of one non- fluoridated and two fluoridated communities in NSW. NSW Public Health Bulletin: 2, 11-12.

Armfield JM, Spencer AJ (2004). Consumption of nonpublic water : implications for children's caries experience. Community Dent Oral Epidemiol: 32: 283-96

Armfield JM (2005).Public water fluoridation and dental health in New South Wales" Aust NZ Pub Health; 29; 477-83

Armfield J.(2006). The extent of water fluoridation coverage in Australia.

Signature……..……..Witnessed by……

Date……..

Australian and New Zealand Journal of Public Health; 30(6):581-582.

Armfield JM, Spencer AJ , Brennan DS (2009). Dental Health of Australia's teenagers and pre-teen children: The Child Dental Health Survey , Australia 2003-04 Dental Statistics and research series no 52 Cat no DEN 199 Canberra AIHW

Armfield JM (2010). Community effectiveness of water fluoridation in reducing children's dental disease" Public Health Report 125: 655-64

P. P. Bachinskii, O. A. Gutsalenko, N. D. Naryzhniuk, et al. (1985). Action of Fluoride on the Function of the Pituitary-thyroid System of Healthy Persons and Patients with Thyroid Disorders (article in Russian). Problemy Endokrinologii (Mosk) 31, no. 6 (1985): 25–29, English translation at http://www.fluoridealert.org/bachinskii.1985.pdf.

Bahls LL. (1973). Diatom community response to primary waste water effluent Journal Water Pollution Control Federation 45 134-144 (cited by Foulkes and Anderson, 1994).

E. B. Bassin (2001). *Association Between Fluoride in Drinking Water During Growth and Development and the Incidence of Osteosarcoma for Children and Adolescents.* DMSc thesis, Harvard School of Dental Medicine, Boston, Massachusetts, 2001.

Bassin EB, Wypij D, Davis RB, Mittleman MA (2006). Age-specific fluoride exposure in drinking water and osteosarcoma (United States). Cancer Causes and Control 17: 421-8.

T. A. Bayley, J. E. Harrison, T. M. Murray, et al (1990). Fluoride-Induced Fractures: Relation to Osteogenic Effect. Journal of Bone and Mineral Research 5, suppl. 1: S217–22.

Brunelle JA, Carlos JP (1990). Recent trends in dental caries in U.S. children and the effect of water fluoridation. J. Dent. Res 69, (Special edition), 723-727.

Signature……..……..Witnessed by……..

Date……..

Available at http://www.fluoridealert.org/health/teeth/caries/nidr-dmfs.html

Burgstahler AW (2006). Failure to diagnose fluoride poisoning in horses caused by water fluoridation. Fluoride 39(1): 1-2.http://www.fluorideresearch.org/391/files/3911-2.pdf

Burgstahler AW, Freeman RF, Jacobs PN. Toxic effects of silicofluoridated water in chinchillas, caimans, alligators, and rats held in captivity. Fluoride 2008;40(1):83-8.

Camargo Julio, A (2002). Fluoride Toxicity to Aquatic Organisms: A Review Departamento Intruniversitario de Ecolgia Edificio de Ciencias, Universidad de Alcal-a, Alcal-a-de, Henares, Madrid E-28871, Spain. 23 August.

Camargo Julio, A, TW La Point (1995). Fluoride Toxicity to Aquatic Life: Proposal of Safe Concentrations for Five Species of Palaearctic Freshwater Invertebrates 23 Jan 1995.

Carpenter R (1969). Factors controlling the marine geochemistry of fluorine. Geochemical et Cosmochimica Acta, 33, 1153-1167.

CDC (1999 a) Centers for Disease Control and Prevention (CDC). 1999. Achievements in public health, 1900- 1999: Fluoridation of drinking water to prevent dental caries. Mortality and Morbidity Weekly Review. (MMWR). 48(41): 933-940 October 22, 1999. Available at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4841a1.htm

CDC (1999 b) Centers for Disease Control and Prevention, “Ten Great Public Health Achievements: United States, 1900–1999,” Morbidity and Mortality Weekly Report 48, no. 12 (April 2, 1999): 241–43, http://www.cdc.gov/mmwr/preview/mmwrhtml/00056796.htm.

Caverzasio J, Palmer G, Bonjour JP (1998). Fluoride: mode of action. Bone

Signature……..……..Witnessed by……

Date……

22(6):585-9.

Choubisa SL (2010).Osteo-dental fluorosis in domestic horses and donkeys in Rajasthan, India. Fluoride 43(1): 5-12.http://www.fluorideresearch.org/431a/files/FJ2010_v43_n1_p005-012.pdf

Code of Practice for the Fluoridation of Public Water Supplies Aug 2002, Fluoridation of Public Water Supplies Act 1957.

P. D. Cohn (1992). An Epidemiologic Report on Drinking Water and Fluoridation, New Jersey Department of Health, Environmental Health Service, November 8, 1992. Note: The original title of this report was A Brief Report on the Association of Drinking Water Fluoridation and the Incidence of Osteosarcoma Among Young Males. The word "osteosarcoma" was deleted from the title soon after the report was released; http://fluoridealert.org/cohn-1992.pdf.

Colquhoun J (1984). New evidence on fluoridation. Social Science & Medicine 19:1239-46.

Colquhoun J (1985). Influence of social class and fluoridation on child dental health. Community Dentistry and Oral Epidemiology 13:37-41.

Colquhoun J (1987). Child dental health differences in New Zealand. Community Health Studies 11:87-104.

P.H. Connett, J. Beck, H.S. Micklem. *The Case Against Fluoride: How Hazardous Waste Ended Up in Our Drinking Water and the Bad Science and Powerful Politics That Keep it There.* Chelsea green, White River Junction, VT, October, 2010.

M. A. Dambacher, J. Ittner, and P. Ruegsegger (1986). Long-Term Fluoride Therapy of Postmenopausal Osteoporosis. Bone 7 (3): 199–205.

Signature……..……..Witnessed by……

Date……

Damkaer DM, Dey DB (1989). Evidence for fluoride effects on salmon passage at John Day Dam, Columbia River, 1982-1986. North American Journal of Fisheries Management 9 154-162 (cited by Foulkes and Anderson, 1994).

Dave G (1984). Effects of fluoride on growth reproduction and survival in Daphnia magna. Comparative Biochemistry and Physiology 78c (2): 425-431 (cited by Foulkes and Anderson, 1994).

Dean HT (1952). HT Dean in: Testimony of Isadore Zipkin, Ph.D., National Institutes of Health, Bethesda, Md., in Chemicals in Foods and Cosmetics: Hearings Before the HouseSelect Committee to Investigate the Use of Chemicals in Foods and Cosmetics, House of Representatives, 82nd Congress (Washington, DC: U.S. Government Printing Office, 1952), 1652

E . Dincer, "Why Do I Have White Spots on My Front Teeth," New York State Dental Journal 74, no. 1 (2008): 58–60, http://www.nysdental.org/img/current-pdf/JrnlJan2008.pdf

Do LG, Spencer AJ, Ha DH (2009). Association between Dental Caries and Fluorosis among South Australian Children. Caries Res;43:366-373

B. de Liefde (1998). The decline of caries in New Zealand over the past 40 years. New Zealand Dental Journal. 94: 109-113

M. Diesendorf (1986). The mystery of declining tooth decay. Nature. 322:125-129. Available at http://www.fluoridealert.org/health/teeth/caries/diesendorf.html

C. W. Douglass and K. Joshipura (2006). Caution Needed in Fluoride and Osteosarcoma Study (letter). Cancer Causes & Control 17 (4): 481–82.

Environment Canada (2001). Canada Water Quality Guidelines for the protection of aquatic life: Inorganic fluorides. Scientific Supporting Document. Ecosystem Health: Science-based Solutions Report 1-1. National Guides and

Signature……..……..Witnessed by……..

Date……..

Standards Office, Environmental Quality branch, Environment Canada, Ottawa.

EIlis MM, Westfall BA, Ellis MD (1938). Determination of Water Quality Research Report 9. Fish and Wildlife Service, Department of Interior, Washington DC, pp 81-82 (cited by Foulkes and Anderson, 1994).

D. Fagin (2008). Second Thoughts on Fluoride. Scientific American 298 (1): 74–81. Excerpts at http://www.fluoridealert.org/sc.am.jan.2008.html.

Fluoridation Forum (2002) Dublin, Ireland: Stationery Office, 2002, page 110, http://www.fluoridealert.org/re/fluoridation.forum.2002.pdf.

Foulkes RG, Anderson AC (1994). Impact of artifical fluoridation on salmon species in the Northwest USA and British Columbia. Fluoride, 27(4): 220-226.

S. C. Freni and D. W. Gaylor (1994). International Trends in the Incidence of Bone Cancer Are Not Related to Drinking Water Fluoridation. Cancer 70 (3): 611–18.

K. H. Gelberg, E. F. Fitzgerald, S. Hwang, and R. Dubrow (1995). Fluoride Exposure and Childhood Osteosarcoma: A Case-Control Study. American Journal of Public Health 85(12): 1678–83, http://ajph.aphapublications.org/cgi/reprint/85/12/1678.pdf.

J. C. Gerster, S. A. Charhon, P. Jaeger, et al (1983). Bilateral Fractures of Femoral Neck in Patients with Moderate Renal Failure Receiving Fluoride for Spinal Osteoporosis. British Medical Journal (Clinical Research Edition) 287, no. 6394: 723–25.

Gray AS (1987). Fluoridation: time for a new base line? Journal of the Canadian Dental Association. 53(10): 763-5.

Groth III E (1975). An evaluation of the potential for ecological damage by

Signature……..……..Witnessed by……

Date……

chronic low-level environmental pollution by fluoride. Fluoride 6 (4) 224-240 (cited by Foulkes and Anderson, 1994).

D. H. Gutteridge, R. I. Price, G. N. Kent, et al (1990). Spontaneous Hip Fractures in Fluoride- Treated Patients: Potential Causative Factors. Journal of Bone and Mineral Research 5, suppl. 1: S205–15.

D. H. Gutteridge, G. O. Stewart, R. L. Prince, et al. (2002). A Randomized Trial of Sodium Fluoride (60 mg) +/- Estrogen in Postmenopausal Osteoporotic Vertebral Fractures: Increased Vertebral Fractures and Peripheral Bone Loss with Sodium Fluoride; Concurrent Estrogen Prevents Peripheral Loss, but Not Vertebral Fractures," Osteoporosis International 13(2): 158–70.

Health Canada (2008). Findings and Recommendations of the Fluoride Expert Panel (January 2007). April 2008, http://fluoridealert.org/re/canada.fluoride.expert.panel.2007.pdf.

Health Canada (2009). Fluoride in Drinking Water. Document for public comment. Guidelines for Canadian Drinking Water Quality: Guideline Technical Document. Prepared by the Federal-Provincial-Territorial Committee on Drinking Water, September 2009, http://fluoridealert.org/canada.2009.report.pdf

L. R. Hedlund and J. C. Gallagher (1989). Increased Incidence of Hip Fracture in Osteoporotic Women Treated with Sodium Fluoride. Journal of Bone and Mineral Research 4 (2): 223–25.

Hemens J, Warvick RJ, Oleff WD (1975). Effect of extended exposure to low fluoride concentration on estuarine fish and crustacea. Progress in water Technology 7 579-585 (cited by Foulkes and Anderson, 1994).

S. E. Hrudey, C. L. Soskolne, J. Berkel, and S. Fincham (1990). Drinking Water Fluoridation and Osteosarcoma. Canadian Journal of Public Health 81(6): 415–16.

Signature……..……..Witnessed by……

Date……..

J. Inkovaara, R. Heikinheimo, K. Jarvinen, et al. (1975). Prophylactic Fluoride Treatment and Aged Bones. British Medical Journal 3, no. 5975: 73–4.

Institute of Medicine (IOM). 1997. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board. National Academy Press. Available at http://www.nap.edu/openbook.php?isbn=0309063507

Ishio S, Makagawa H (1971). Cited in: Rose D. Marier J. Environmental Fluoride 1977. National Research Council of Canada, Ottawa 1977 p 30 ((cited by Foulkes and Anderson, 1994).

Izquierdo-Vega JA, Sánchez-Gutiérrez M, Del Razo LM (2008). Decreased *in vitro* fertility in male rats exposed to fluoride-induced oxidative stress damage and mitochondrial transmembrane potential loss. Toxicol Appl Pharmacol 2008 Mar 28. [Epub ahead of print].

Justus C and Krook LP (2006). Allergy in Horses from Artificially Fluoridated Water. Fluoride 39 (2): 89–94 http://www.fluorideresearch.org/392/files/39289-94.pdf.

Kennedy, D (2008). "*Poisoned Horses."* (DVD) International Academy of Oral Medicine and Toxicology. Excerpts are available at http://www .youtube.com/watch?v=t9RXfOuylWo.

Komárek A, Lesaffre E, Härkänen T, Declerck D, Virtanen JI (2005). A Bayesian analysis of multivariate doubly-interval-censored dental data. Biostatistics 6:145-55.

Krook, LP, Justus, C (2006). Fluoride poisoning of horses from artificially fluoridated drinking water. Fluoride 39(1):3-10. http://www.fluorideresearch.org/391/files/3913-10.pdf

Kabata - Pendias and H Pendias. *"Trace Elements in Soils & Plants,"* Chapter 12, Vol II, Fluorine. CBC Press Inc, Boca Raton, Florida, 1984.

Lee M and Dennison PJ (2004). Water fluoridation and dental caries in 5 and 12

Signature……..……..Witnessed by……

Date……

year old children from Canterbury and Wellington. New Zealand Dental Journal 100(1):10-5.

Leverett DH (1982). Fluorides and the changing prevalence of dental caries. Science. 217(4554):26-30.

Y. Li, C. Liang, C. W. Slemenda, et al. (2001).Effect of Long-term Exposure to Fluoride in Drinking Water on Risks of Bone Fractures. Journal of Bone and Mineral Research 16 (5): 932–39.

Locker D (1999). Benefits and risks of water fluoridation. An Update of the 1996 Federal-Provincial Sub-committee Report. Prepared for Ontario Ministry of Health and Long Term Care. Available at http://www.health.gov.on.ca/english/public/pub/ministry_reports/fluoridation/fluor.pdf

Long H, Jin Y, Lin M, Sun Y, Zhang L, Clinch C (2009). Fluoride toxicity in the male reproductive system. Fluoride 42(4):260-76.

Luke, J (2001). Fluoride Deposition in the Aged Human Pineal Gland. Caries Research 35 (2): 125–28.

Luke, J (1997). "The Effect of Fluoride on the Physiology of the Pineal Gland," PhD thesis, University of Surrey, Guildford, UK, 1997. Thesis online, with permission of author, at http://fluoridealert.org/luke-1997.pdf.

R. P. Maas, S. C. Patch, A. M. Christian, and M. J. Coplan (2007). Effects of Fluoridation and Disinfection Agent Combinations on Lead Leaching from Leaded-Brass Parts. Neurotoxicology 28(5): 1023–31.

Macicek P and Krook LP (2008). Fluorosis in horses drinking artificially fluoridated water. Fluoride, 41 (2):177-183 http://www.fluorideresearch.org/413/files/FJ2008_v41_n3_p177-183.pdf

Signature……..……..Witnessed by……

Date……

Mackay TD and Thomson WM (2005). Enamel defects and dental caries among Southland children. NZ Dental Journal. 101(2):35-43.

M. C. Mahoney, P. C. Nasca, W. S. Burnett, and J. M. Meius (1991). Bone Cancer Incidence Rates in New York State: Time Trends and Fluoridated Drinking Water. American Journal of Public Health 81, no. 4 (1991): 475–79, http://ajph.aphapublications.org/cgi/ reprint/81/4/475.pdf.

R. D. Masters and M. J. Coplan (1999). Water Treatment with Silicofluorides and Lead Toxicity. International Journal of Environmental Studies 56 (4): 435–49.

R. D. Masters, M. J. Coplan, B. T. Hone, and J. E. Dykes (2000). Association of Silicofluoride Treated Water with Elevated Blood Lead. Neurotoxicology 21 (6): 1091–99.

Masuda TT (1964). Persistence of fluoride from organic origins in waste waters. Developments in Industrial Microbiology, 5, 53-70 (cited by Foulkes and Anderson, 1994).

Matsuo S, Kiyomiya K, Kurebe M (1998). Mechanism of toxic action of fluoride in dental fluorosis: whether trimeric G proteins participate in the disturbance of intracellular transport of secretory ameloblast exposed to fluoride. Arch Toxicol. 72(12):798-806.

J. K. Maurer, M. C. Cheng, B. G. Boysen, and R. L. Anderson (1990). Two-Year Carcinogenicity Study of Sodium Fluoride in Rats. Journal of the National Cancer Institute 82 (3): 1118–26.

S. M. McGuire, E. D. Vanable, M. H. McGuire, J. A. Buckwalter, and C. W. Douglass (1991). Is There a Link between Fluoridated Water and Osteosarcoma? Journal of the American Dental Association 122(4): 38–45.

MRC (2002). Medical Research Council, Water Fluoridation and

Signature……..……..Witnessed by……

Date……

Health, Working Group Report, UK, September 2002, http://fluoridealert.org/re/mrc-2002.pdf.

M. E. Moss, M. S. Kanarek, H. A. Anderson, et al. (1995). Osteosarcoma, Seasonality, and Environmental Factors in Wisconsin, 1979–1989. Archives of Environmental Health 50 (3): 235–41.

P. J. Mullenix, P. K. Denbesten, A. Schunior, and W. J. Kernan (1995). Neurotoxicity of Sodium Fluoride in Rats. Neurotoxicology and Teratology 17, no. 2: 169–77.

Murray F (1981). Fluoride Cycles in an Estuarine Ecosystem. Sci. Tot. Environ. 17:223-241, cited Environment Canada, 2001.

O. Naidenko (2009). Dog Food Comparison Shows High Fluoride Levels. Environmental Working Group, June 2009, http://www.ewg.org/pets/fluorideindogfood.

C. Neurath and P. Connett, "Current Epidemiological Research on Link between Fluoride and Osteosarcoma," Fluoride 41, no. 3 (2008): 241–42 (abstracts from the XXVIIIth Conference of the International Society for Fluoride Research), http://www.fluorideresearch.org/413/files/FJ2008_v41_n3_p233-258.pdf.

NHMRC (1991). National Health and Medical Research Council, The Effectiveness of Water Fluoridation (Canberra: Australian Government Publishing Service, 1991), 142.

NHMRC (2007). National Health and Medical Research Council, A Systematic Review of the Efficacy and Safety of Fluoridation, reference no. EH41, Australian Government, December 27, 2007, http://www.nhmrc.gov.au/publications/synopses/eh41syn.htm.

NRC (2006). National Research Council of the National Academies, Fluoride in Drinking Water: A Scientific Review of EPA's Standards (Washington, DC:

Signature……..……..Witnessed by……

Date……

National Academies Press, 2006), http://books.nap.edu/openbook.php?record_id=11571.

NSW,(2007). Child Dental Health Survey, 2007. Centre for Oral Health Strategy NSW 2009

NTP (1990) National Toxicology Program. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Sodium Fluoride (CAS no. 7682-49-4) in F344/N Rats and B6C3F1 (Drinking Water Studies). Technical Report 393, NIH publ. no. 91-2848, National Institutes of Health, Public Health Service, U.S. Department of Health and Human Services, Research Triangle Park, NC, 1990.

J. D. O'Duffy, H. W. Wahner, W. M. O'Fallon, et al (1986). Mechanism of Acute Lower Extremity Pain Syndrome in Fluoride-Treated Osteoporotic Patients. American Journal of Medicine 80 (4): 561–66.

P. Orcel, M. C. de Vernejoul, A. Prier, et al (1990). Stress Fractures of the Lower Limbs in Osteoporotic Patients Treated with Fluoride. Journal of Bone and Mineral Research 5, suppl. 1: S191–94.

Osterman, JW (1990). Evaluating the impact of municipal water fluoridation on the aquatic environment. American Journal of Public Health, 80:1230-1235.

Peiris TS, Roberts GJ, Prabhu N (2009). Dental Age Assessment: a comparison of 4- to 24-year-olds in the United Kingdom and an Australian population International Journal of Paediatric Dentistry 19: 367–376

Pizzo G, Piscopo MR, Pizzo I, Giuliana G (2007). Community water fluoridation and caries prevention: a critical review. Clinical Oral Investigations 11(3):189-93.

Pocatello (1961). US Court Of Appeals, Ninth Circuit (Pocatello, Idaho) No 17059 (1961): Food and Machinery and Chemical Corporation and J R Simplot Co. vs W S and Ray Meader. Exhibit (Table 1) August 25 1961.

Signature……..……..Witnessed by……

Date……

B. L. Riggs, S. F. Hodgson, W. M. O'Fallon, et al. (1990). Effect of Fluoride Treatment on the Fracture Rate in Post-Menopausal Women with Osteoporosis. New England Journal of Medicine 322 (12): 802–9.

Riordan PJ (1993). Perceptions of Dental Fluorosis" J Dent Res 72 (9) 1268 – 1274

Rous Water (2009). Review of Environmental Factors, Report No. DC 09126, December, 2009, Public Works, Water Solutions.

Sawan, R. M. M.; Leite, G. A. S.; Saraiva, M. C. P.; Barbosa Jr, F.; Tanus-Santos, J. E.; Gerlach, R. F. (2010). Fluoride increases lead concentrations in whole blood and in calcified tissues from lead-exposed rats. Toxicology, 271 (1-2), 21-26.

Schluter PJ, Kanagaratnam S, Durwood CS, Mahood R (2008). Prevalence of enamel defects and dental caries among 9-year-old Auckland children. NZ Dental Journal 104(4): 145-152.

C. M. Schnitzler, J. R. Wing, K. A. Gear, and H. J. Robson, (1990). Bone Fragility of the Peripheral Skeleton during Fluoride Therapy for Osteoporosis. Clinical Orthopaedics no. 261: 268–75.

Sheldon T (2001). Open Letter by Professor Trevor Sheldon, DSc, FMedSci, of the University of York, Department of Health Sciences, Heslington, York, UK, January 3, 2001, http://www.appgaf.org.uk/archive/archive_letter_shel/.

Singer L. Armstrong WD. (1977). Fluoride in treated sewage and in rain and snow. Archives of Environmental Health, 32 21-23.

Slade GD, Spencer AJ, Stewart JF (1996). Caries experience among children in fluoridated Townsville and non- fluoridated Brisbane. Aus NZ J Public Health; 20: 623-629

Signature……..……..Witnessed by……

Date……

Spencer AJ, Slade GD, Davies M (1996). Water fluoridation in Australia. Community Dent Health. 13 Suppl 2:27-37. September.

Spittle B (2010). Fluoride toxicity and donkeys. (editorial) Fluoride 43(1): 4 http://www.fluorideresearch.org/431/files/FJ2010_v_43_n1_p004.pdf

Strunecka A, Patocka J. (1999). Pharmacological and toxicological effects of aluminofluoride complexes. Fluoride. 32(4):230-242. Full study at http://fluoridealert.org/re/strunecka-1999.pdf

Strunecka A, Strunecky O, Patocka J. (2002). Fluoride plus aluminum: useful tools in laboratory investigations, but messengers of false information. Physiological Research 51(6):557-64.

Strunecka A, Patoka J, Connett P, 2004. "Fluorine in Medicine." Journal of Applied Biomedicine 2: 141-150.

Sutton P. 1996. The Greatest Fraud: Fluoridation. Lorne, Australia: Kurunda Pty, Ltd.

Tasmania, 1989. The Supreme Court of Tasmania Court Case Dec, 1989, Hobart Register No. 1604 of 1987 Sylvia & John Braim vs Tasmanian Govt.

Urbansky ET, Schock MR. 2000. Can fluoridation affect lead(II) in portable water? Hexafluorosiliciate and fluoride in aqueous solution. Int J Environ Studies 57:597-637.

Varner JA, Jensen KF, Horvath W, Isaacson RL. 1998. Chronic administration of aluminum-fluoride and sodium-fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity. Brain Research. 784(-2):284-298. February 16. See extended excerpts at http://www.fluoride-journal.com/98-31-2/31291-95.htm

Signature……..……..Witnessed by……

Date……

C. Wang, D. B. Smith, and G. M. Huntley, 2000. "Treatment Chemicals Contribute to Arsenic Levels," Opflow (a journal of the American Water Works Association), October 2000.

J. J. Warren, S. M. Levy, B. Broffitt, et al., "Considerations on Optimal Fluoride Intake Using Dental Fluorosis and Dental Caries Outcomes—A Longitudinal Study," Journal of Public Health Dentistry 69, no. 2 (2009): 111–15.

Warrington P.D. 1990, Ambient Water Quality Criteria for Fluoride. Technical Appendix British Columbia Ministry of the Environment Victoria BC.

WHO (2002), World Health Organization, Fluorides, Environmental Health Criteria 227, International Programme on Chemical Safety, Geneva, Switzerland, 2002, http://www.inchem.org/documents/ehc/ehc/ehc227.htm.

JA Yiamouyiannis 1990. Water fluoridation and tooth decay: results from the 1986-87 national survey of U.S. schoolchildren. Fluoride. 23:55-67.

SWORN/AFFIRMED at: ……

Signature of deponent: ……

Signature of witness: ……

Name of witness: ……..

Address of witness: ……...

Capacity of witness: …… Justice of the Peace/Solicitor

Signature……..……..Witnessed by……..

Date……..

Date sworn/ affirmed: ……

NOTE : The deponent and witness must sign each page of the affidavit. See UCPR 35.7B (Civil Procedure, Schedule 7.)

Signature……..……..Witnessed by……

Date……..