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Fluoride Action Network

January 8, 2016

Dr. Ruth Lunn,
Director, Office of the Report on Carcinogens
National Toxicology Program
[sent electronically to lunn@niehs.nih.gov]

Dear Dr. Lunn,

On behalf of the Fluoride Action Network (FAN), we are writing today in response to the National Toxicology Program's (NTP) nomination of fluoride as a substance to be reviewed for carcinogenicity. In its October 7, 2015 notice in the Federal Register, the NTP requested information on four subject areas relative to fluoride's nomination. We have provided detailed information on each of these four areas below.

NTP's nomination of fluoride for study of carcinogenicity is well warranted, particularly with respect to osteosarcoma and bladder cancer. The National Research Council (NRC) has stated that "fluoride appears to have the potential to initiate or promote cancers, particularly of the bone, but the evidence to date is tentative and mixed." [NRC 2006, p. 336]. Since NRC's review, additional studies relevant to fluoride carcinogenicity have been published. As discussed below, this research includes 19 *in vitro* studies, 19 animal studies, and 3 human studies reporting genotoxic effects from fluoride exposure.

Further, while a number of recent epidemiological studies have failed to find an association between fluoride and osteosarcoma [e.g., Gelberg 1995; Kim 2011; Levy 2012; Blakey 2014; Comber 2015; Young 2015], these studies have *major* limitations which may explain why they were unable to detect a positive association between fluoride and cancer. Reviews and reanalyses of these studies (which we have attached as Appendices 6-11) demonstrate that when these limitations are accounted for and corrected, the Gelberg [1995], Kim [2011], and Blakey [2014] studies actually *support* the age-specific relationship between fluoride and osteosarcoma first identified by Bassin in 2006. We urge the NTP to carefully consider these reviews, as they show that the current epidemiological evidence linking fluoride to childhood osteosarcoma is much stronger than currently recognized.

In addition to the reanalyses, we are attaching several comprehensive reviews of the fluoride/cancer literature, which include extensive discussions of Bassin's age-specific analysis; the biologic plausibility of fluoride being a carcinogen, particularly in bone; and the limitations of pre-2006 epidemiological research. These reviews can be found in Appendices 1-A, 1-B, 1-C, 2, and 5.

If there is *any* further information that FAN can provide that will assist NTP with its review—including providing electronic copies of papers cited in FAN's extensive research database—please do not hesitate to let us know.

Sincerely,

Michael Connett & Chris Neurath
Fluoride Action Network
www.FluorideAlert.Org

Submission by Fluoride Action Network (FAN) to National Toxicology Program (NTP) on Proposal to Review Carcinogenicity of Fluoride

Chris Neurath & Michael Connett
Fluoride Action Network (FAN)

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(1) Data on Current Production, Use Patterns, and Human Exposure.

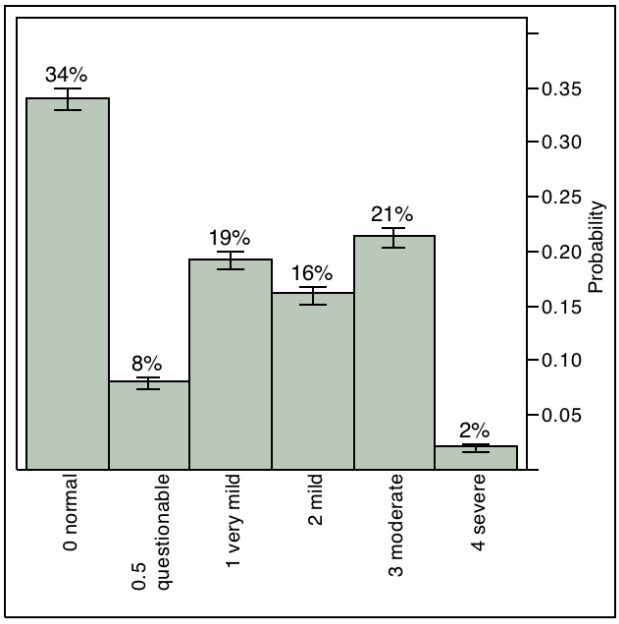
It is widely recognized that fluoride exposure has increased considerably over the past 70 years. This increase in exposure is reflected by the rising rates of dental fluorosis in U.S. children.

Whereas the incidence of dental fluorosis, in its mildest forms, was approximately 10% in the 1940s (NRC 1951); the CDC's 1999-2004 NHANES survey found that 41% of American adolescents had the condition [Beltrán-Aguilar 2010].

The rates of fluorosis have continued to rise since the early 2000s, as evidenced by the CDC's 2011-2012 NHANES survey, which found that 58% of adolescents now have the condition, with a staggering 21% of adolescents displaying *moderate* fluorosis on at least two teeth, up from 2% in 1999-2004. *Severe* fluorosis has also increased, from <1% to 2%.

The data for CDC's 2011-2012 NHANES survey, which we have summarized in Figure 1 below, can be accessed online at:
http://wwwn.cdc.gov/nchs/nhanes/search/nhanes11_12.aspx

Figure 1. NHANES 2011-2012, Distribution of Dean's Index Dental Fluorosis scores, unweighted. (Fluorosis score based on maximum degree found in at least 2 teeth)



Overview of Sources:

A comprehensive overview of current sources of fluoride exposure in the United States, and accompanying estimates of daily fluoride intakes, can be found in Chapter 2 (pages 23 to 88) of the NRC's 2006 report Fluoride in Drinking Water: A Scientific Review of EPA's Standards [NRC 2006].

Toothpaste:

While the NRC report provides a good overview of most fluoride sources, the NRC's estimate of daily exposures from toothpaste significantly understates fluoride exposure from this important source. The NRC estimated "typical" daily intakes of just 0.1 to 0.3 mg/day per day in children from *two brushings* with fluoride toothpaste (NRC 2006, Table 2-7), yet the average intakes reported in the scientific literature *greatly exceed* these estimates. Stephen Levy from the University of Iowa, for example, has estimated that children ingest an average of 0.3 grams of toothpaste per brushing, which equates to 0.3 – 0.45 mg of fluoride per brushing and thus *0.6 to 0.9 mg of fluoride per day* for children who brush twice [Levy 1993]. As Levy has stated, "Virtually all authors have noted that some children could ingest more fluoride from [toothpaste] alone than is recommended as a total daily fluoride ingestion." [Levy 1999].

For more recent data on fluoride intake from toothpaste, which further emphasizes the notably large quantities that many children now consume, we encourage NTP to review the recent work by Zohoori (2012, 2013), Oliveira (20913), and Strittholt (2015).

Fluoride Supplements

Fluoride supplements (i.e., pills and/or lozenges prescribed to replicate exposure to fluoridated water for children living in nonfluoridated areas) remain a significant source of daily fluoride intake. Some studies have found that children receiving the recommended doses of F supplements, who live in areas with low water F levels, can actually receive higher total F intakes than children living in areas with fluoridated water [Guha-Chowdhury 1996]. This finding means that caution must be exercised when interpreting studies that did not account for F supplement use, or total F intake, since exposure measures based on drinking water fluoride alone may seriously misclassify relevant F exposure.

Further, although fluoride supplements were only supposed to be prescribed in areas without fluoridated water, recent research has found that some dentists are prescribing these supplements to children, irrespective of the fluoride content of their water [Narendran 2006].

Tea

Tea plants readily absorb fluoride from soil. As a result, tea beverages invariably contain high levels of fluoride.

According to data from the U.S. Department of Agriculture, brewed black tea in the United States averages about 3 to 4 parts ppm fluoride [Pehrsson 2005, 2011]. Other published literature shows that commercial iced tea drinks typically contain between 1 and 4 ppm. [Whyte 2006].

Based on the high levels of fluoride in current tea products, a series of case reports over the past 10 years have identified cases of skeletal fluorosis among heavy-tea drinkers in the U.S. For a summary of and citations to these case reports, see: http://www.fluoridealert.org/studies/tea03/

Notably, however, there has yet to be any study of fluoride carcinogenicity that has considered tea-based fluoride exposures.

Pharmaceuticals & Anesthetics

Many pharmaceuticals are currently made with organofluorine compounds. Some of these orgnanofluorine-based pharmaceuticals metabolize into inorganic fluoride, as reflected by elevated inorganic fluoride levels in blood and/or urine. The organofluorine pharmaceuticals that have thus far[1] been reported to metabolize into fluoride ion include: Cipro [Pradham 1995], the fluorinated anesthetics Isoflurane and Sevoflurane [Hoemberg 2012, Oc 2012]; Flecainide [Rimoli 1991]; niflumic acid [Gras-Champel 2003; Welsch 1990; Meunier 1980; Prost 1978]; and Voriconazole [Wermers 2011].

Of particular concern vis-à-vis carcinogenicity is the release of fluoride from fluorinated anesthetics (Isoflurane & Sevoflurane) during infancy and early childhood.

Fluoride Pesticides: Cryolite and Sulfuryl Fluoride

Fluoride chemicals (i.e., cryolite and sulfuryl fluoride) continue to be used as pesticidal agents in the United States.

Cyrolite is principally used as an insecticide on vineyards, which results in measurable fluoride contamination of wine, grape juice, and raisins. According to data from the U.S. Department of Agriculture, the average fluoride content of white grape juice is **2.13 ppm**, the average fluoride content of white wine is **2.02 ppm**, and the average fluoride content of red wine is **1.05** ppm. [USDA 2005].

[1] It is very likely that this list is incomplete as the potential for fluoride ion release from organofluorine pharmaceuticals has never been comprehensively studied.

Sulfuryl fluoride[2] is currently being widely used in the United States as a post-harvest fumigant. While most countries have banned the use of sulfuryl fluoride in food storing facilities, the United States now permits this use.

More disturbingly, the United States permits sulfuryl fluoride to be directly applied to certain foodstuffs. EPA has estimated that 100% of (non-organic) cocoa powder is fumigated with sulfuryl fluoride, 100% of (non-organic) dried beans, 99% of (non-organic) walnuts, 69% of (non-organic) dried fruits, 10% of (non-organic) tree nuts, 10% of almonds, and 3% of milled rice. Field tests performed by Dow AgroSciences show that the average residual fluoride contamination from the direct fumigation of these products ranges from 1 to 12.5 ppm.[3] See: http://fluoridealert.org/content/sf_exposure/

Fluoride Pollution

Hydrogen fluoride is one of the primary air emissions of the coal industry and is also emitted by other industries such as aluminum, chemical, glass and brickworks. According to the Toxics Release Inventory (TRI), U.S. power plants emitted 67 million pounds of hydrogen fluoride from 2010 to 2014. In total, U.S. industries emitted 104 million pounds of hydrogen fluoride during these years. See: http://fluoridealert.org/content/tri-rank-by-industry-for-hf-1994-2014/ . Further, recent research has identified the burning of biomass material as a major source of airborne F [Jayarathne 2014].

While current ambient air concentrations of fluoride (as HF or other air pollutants) generally do not contribute significantly to total F exposure, industrial fluoride emissions (airborne and/or waterborne) can be a significant source of exposure for those living close to fluoride-emitting industries. Further, the potential for enhanced fluoride toxicity when fluoride is inhaled in the form of fine particulate matter is a subject that remains almost entirely unexplored.

(2) PUBLISHED, ONGOING, OR PLANNED STUDIES RELATED TO EVALUATING ADVERSE HEALTH OUTCOMES.

The Fluoride Action Network (FAN) has created a uniquely comprehensive database ("Study Tracker") for published research on fluoride toxicity, including foreign studies that FAN has translated into English. This database is available online at: http://www.fluoridealert.org/studytracker/ .

We encourage NTP to utilize this database in order to obtain a more complete picture of the available research on fluoride carcinogenicity. We have full-text

[3] For a complete list of the fluoride residue limits that EPA permits on foods as a result of sulfuryl fluoride use, see: http://fluoridealert.org/content/fluoride-tolerances/

papers for the vast majority of studies cited in this database and are happy to provide electronic copies of these papers upon request.

In addition to including the published epidemiological literature on fluoride and cancer (which are discussed below and in the attached appendices), the database includes 118 studies that have examined the genotoxicity and mutagenicity of fluoride.[4] This includes 59 *in vitro* studies, which can be accessed at: http://tinyurl.com/ht7vkwt, 44 animal studies, which can be accessed at http://tinyurl.com/j6lpszg, and 15 human studies, which can be accessed at http://tinyurl.com/zdc5psx. The overwhelming majority of these studies have found genotoxic and/or mutagenic effects from fluoride exposures, including at non-cytotoxic concentrations.

The genotoxicity of fluoride supports the biologic plausibility of fluoride being a carcinogen. As the NTP noted in its prior bioassay of fluoride/cancer:

> "it would appear that sodium fluoride is genotoxic in a number of genetic toxicity assays, through as yet undetermined mechanisms. So, a neoplastic effect in a tissue that accumulates fluoride would appear possible." [Bucher 1990, p. 30-31]

Evidence linking fluoride to genotoxic effects has increased substantially in recent years, with 19 *in vitro* studies, 19 animal studies, and 3 human studies reporting effects since 2006. See: http://tinyurl.com/j5wf4ga

(3) Scientific Issues Important for Prioritizing and Assessing Adverse Health Outcomes

The majority of fluoride-cancer studies have focused on bone cancer, especially osteosarcoma. This is for biological plausibility reasons, as well as evidence from *in vitro* and animal studies. Bone accumulates 99% of the body's absorbed fluoride dose and reaches much higher F concentrations than any other tissue. Also, fluoride is known to stimulate osteobast activity, and has been found in numerous *in vitro*, as well as some *in vivo*, studies to induce genotoxicity or mutagenicity. Animal carcinogenicity studies have also found positive associations between fluoride and bone tumors, both malignant (osteosarcoma) and benign (osteoma). [Bucher 1991; Maurer 1993].

A few studies have looked at specific cancers other than bone cancers, or larger groupings of cancer types [Grandjean 1992, 2004; Takahashi 2001; Lynch 1984]. As discussed in Appendix 2, the occupational studies by Grandjean found a significant relationship between fluoride and bladder cancer in the absence of

[4] Many of the genotoxicity studies were published subsequent to the NRC's review in 2006, including 17 of the *in vitro* studies, 13 of the animal studies, and 2 of the human studies.

PAH exposure, thus suggesting that fluoride may be a contributing cause of the high rates of bladder cancer seen in the aluminum industry, where both PAH and fluoride exposures are high.

Our individual study reviews found that in most of the higher quality studies (case-control design) that purported to find no effect, when limitations were accounted for, they actually may have found a positive effect.

For most of the lower quality (ecological) studies that found no effect, we found that limitations can explain their inability to detect an effect.

Therefore, these studies do not provide strong evidence that fluoride is not carcinogenic. When all the evidence for carcinogenicity is taken together, and a weight-of-evidence evaluation is applied, we believe there is now sufficient evidence to classify fluoride as a probable or possible carcinogen, just as the NRC 2006 review concluded. Indeed, the two case-control studies published after the NRC 2006 review, by Bassin [2006] and Kim [2011], provide even more evidence that fluoride is carcinogenic than was available to the NRC 2006 review committee.

(3.1) Reviews of individual studies. We have prepared reviews of the more important human epidemiological studies of fluoride and cancer. Each of these reviews is attached as a separate appendix. We briefly summarize the findings from these individual reviews here but important details can be found only in the appendices themselves. Studies published before 2006 were reviewed in several submissions to the NRC 2006 committee which was preparing a comprehensive report on the toxicology of fluoride, including carcinogenicity. Those reviews of pre-2006 cancer studies are available in the following appendices:

APPENDIX 1-A. FAN submission to NRC 2006 on cancer, Part 1
APPENDIX 1-B. FAN submission to NRC 2006 on cancer, Part 2
APPENDIX 2. FAN submission to NRC 2006 on cancer [Grandjean 2004]
APPENDIX 3. Kenya study abstract [Neurath 2005a]
APPENDIX 4. Kenya study, full, with introduction [Neurath 2005b]

We have also included as an appendix a comprehensive independent review of the literature through 2011 by Dr. Kathleen Thiessen, a panelist on the NRC's fluoride review:

APPENDIX 5. Thiessen submission to California OEHHA, 2011

Of the higher quality human epidemiological studies to date, several have found a positive association between fluoride and cancer, specifically osteosarcoma [Hoover 1991; Cohn 1992; Bassin 2006]. These three studies suggest that fluoride's connection to osteosarcoma may be both age and gender-specific, with

the link most pronounced, if not limited to, adolescent males exposed to fluoride during their pre-adolescent years.

Other higher quality human studies which did not find an effect of fluoride on osteosarcoma had serious limitations in their study designs and analyses [Gelberg 1994, 1995, Kim 2011]. These limitations included confounding, selection biases, non-relevant age groups, and failure to account for exposure timing effects. Reanalyses that correct for these problems show that both the Gelberg and Kim studies actually *support* an age-specific fluoride/osteosarcoma relationship. These reanalyses can be found in the following appendices:

APPENDIX 7-A. Gelberg 1995 review
APPENDIX 7-B. Gelberg 1995 review, calculations of age vs F exposure
APPENDIX 6-A. Kim 2011 review (includes discussion of Bassin 2006),
APPENDIX 6-B. Kim 2011 review, sensitivity analysis of fracture rates.

Recent ecological studies have also been reviewed individually. The most important of these is by Blakey [2014], which is discussed at length in the following appendices:

APPENDIX 8-A. Blakey/McNally 2014 review
APPENDIX 8-B. Blakey/McNally 2014 review, calculations of % misclassified

Blakey's study found no association between fluoride and bone cancers, but it also suffered from several serious limitations. They included limited exposure information, solely from drinking water F. The study was in Great Britain where F intake from tea can equal or exceed F intake from drinking water, so exposure misclassification may have been rather extreme. An additional problem with the exposure measurement was that it lacked a time history of exposure. We show how this likely led to differential misclassification, since many subjects who were considered fluoridated because their residence area was fluoridated in the mid 2000s, when water F levels were available, did not grow up in a fluoridated area, because many of the fluoridated areas in Great Britain only became fluoridated in the 1980s. This misclassification would have biased the results away from a positive finding, and could even have biased it to the extent that fluoridation exposure would have spuriously seemed to protect against bone cancers.

Corrections for these and other limitations suggest that, as with the Kim and Gelberg studies, the Blakey study may actually support the conclusion that fluoride intake is positively associated with an increased risk of osteosarcoma.

The individual review of Blakey 2014 in appendices 8-A and 8-B provide extensive details supporting these summary findings.

Three other recent ecological studies are also reviewed individually. All three of these studies found no association between F and osteosarcoma, but all three of them suffered limitations which could explain why they could not detect an effect. The individual reviews are in these appendices:

APPENDIX 9. Comber 2015 review
APPENDIX 10-A. Young 2015 review
APPENDIX 10-B. Young 2015 review, calculations of % misclassified
APPENDIX 11. Levy 2012 review

The Comber 2015 study was limited by its relatively small sample size, especially for the age and gender groups of most interest. It was based on residence at time of diagnosis so was unable to account for time history of exposure, and population mobility could have led to substantial exposure misclassification. The study did not attempt to control for several important potential confounders like socio-economic status (SES). Similarly to the Blakey study, Comber did not account for F sources besides drinking water, yet it was conducted in Ireland, which has an even higher tea consumption (and thus non-drinking water F source) than Great Britain. Tea consumption in Great Britain and Ireland is more than 10 times higher than in the US, so it must be considered as a very significant contributor to total F intake, in some cases outweighing F from drinking water.

The Young 2015 study was in many respects a replication of the Blakey 2014 study, using similar sources of exposure and outcome data, but it had a smaller sample size because of restrictions in geographical area and time period studied. Its limitations largely mirrored Blakey's, and thus its results suffered from similar problems and potential biases.

The Levy 2012 study, which was conducted in the US, was based on relatively crude ecological groups—i.e., states. The outcome was average state-wide rates of osteosarcoma and the exposure measure was based on the percent of the state with fluoridated water. Levy did not control for potential confounders such as SES. Since exposure was based on state-level information for a single point in time, there is likely to be extensive exposure misclassification due to population mobility, which is very high in the US. No exposure information was available for sources of F exposure other than water fluoridation. Furthermore, in the US, there is a fairly large "diffusion effect" whereby people residing in a non-fluoridated area get substantial fluoride from foods and beverages produced in fluoridated areas, as well as meals eaten out in fluoridated areas. This diffusion effect also leads to exposure misclassification which will bias results toward a spurious null effect (no association between fluoride and osteosarcoma).

Due to these and other limitations described in detail in the individual reviews, the Comber 2015, Young 2015, and Levy 2012 studies do not provide strong evidence that fluoride is not a risk factor for osteosarcoma.

In the next subsections, we discuss important issues for evaluating studies on fluoride and cancer generally.

(3.2) What exposure measures were used? Most ecological studies and some case-control studies use artificial water fluoridation status at time of diagnosis. This measure ignores latency; exposure from sources other than home tap water, which can be substantial; variation in tap water consumption; population mobility; and age-specific effects. All of these limitations can impose substantial error, and in some cases, bias in the exposure estimates. Of those studies which use more refined measures of fluoride exposure, only one [Bassin 2006], a case-control study, has taken age-specific exposure into account in their exposure measure. Bassin's age-specific method implicitly also accounts for latency.

Two case-control studies have taken into account time history of water F exposure and other F sources, such as F supplements and swallowed F toothpaste, but not diet [Gelberg 1994, 1995; Bassin 2006]. However, these studies' estimates of F supplements and F toothpaste exposures have been based on subject recall in questionnaires and are unreliable in that they did not ask about doses and compliance with F supplements or amount of F toothpaste swallowed. Some studies have estimated bottled water use, but again, these estimates are based on subject recall and were only crudely measured. The level of F in the bottled water was typically not determined, and can range from near zero to 4 mg/L or higher. Of those studies that used fluoridated water as the measure of exposure, most assigned exposure status based on just whether the tap water was fluoriated or not. Only two used actual measured water F concentrations to refine their exposure measure [Bassin 2006, Blakey 2014].

Of the ecological studies, the geographical areas studied ranged from entire nations, to states/provinces, to counties, to small administrative areas. The larger areas had person-years of exposure in the millions per area, while the smallest had person-years of exposure in the thousands. Most of the ecological studies assigned "fluoridated" and "unfluoridated" status by the percent of the area fluoridated, with differing cutoffs in different studies. Such relatively crude exposure estimates in ecological studies will likely result in non-differential misclassification that will bias results to a null effect.

One study used bone F levels as a biomarker for F exposure [Kim 2011]. Bone F is a measure of cumulative lifetime exposure, so is not able to account for age-specific effects. For older subjects, it also has decreasing ability to capture childhood exposures, which may be the most susceptible time for childhood and teenage osteosarcomas. Furthermore, the Kim [2011] study did not account for latency.

It is worth noting that the Bassin [2006] study found a significant effect from exposure during a relatively narrow time window but not at other ages of exposure. The susceptible period was age 6-8, which was postulated to be due to the mid-childhood growth spurt, a developmental stage when bone remodelling is increased. No other studies have looked at age-specific exposures. Therefore, no other studies have been able to directly address Bassin's findings.

One study used the biomarker dental fluorosis as the measure of exposure [Neurath 2005a, 2005b]. Dental fluorosis presence and severity are strongly correlated with childhood total F intake from age 0-8 [Ziegelbecker 1981]. Fluorosis therefore provides a better estimate of total exposure during this age period than measures based on drinking water F and measures based on exposure at time of diagnosis.

(3.3) Confounding. Ecological studies are subject to the "ecological fallacy" since they are group level analyses rather than individual level. This results in difficulty controlling for confounding and interaction, which can lead to biases. Most ecological studies attempted to control for only a few potential confounders, such as age and gender. Case-control and cohort studies are better able to control for confounding. Most such studies did attempt to control for several potential confounders, including age, gender, socio-economic status (SES), sources of F exposure other than drinking water, and rural/urban. There are very few known risk factors for osteosarcoma, but each of these five factors have been identified.

The best known risk factor for osteosarcoma is ionizing radiation, especially internal bone-seeking, alpha-emitting, radionuclides. Radium is the classic example, and was identified as early as the 1930s as a cause of osteosarcoma, in women exposed while painting watch dials with radium paint. Recently, limited evidence suggests that indoor radon may also be a risk factor for osteosarcoma [Wright 2004]. Radon is an alpha-emitter, but instead of concentrating in calcified bone tissue, it concentrates in fatty tissue including bone marrow.

Of the studies on fluoride and bone cancer, none have considered radionuclide exposure as a possible risk factor. Radionuclides may be potential confounders, since they can be associated with drinking water F levels, or other risk factors for osteosarcoma such as SES and rural/urban.

Genetic risk factors have not been considered in most fluoride-cancer studies, although a few have considered race. Blacks have a greater incidence rate of osteosarcoma than whites, so genetics probably is a risk factor.

(3.4) Selection bias, cancer outcomes, and other validity issues. Most human epidemiological studies have used cancer diagnosis from population-based cancer registries as their outcome effect. A few have used cancer

diagnosis from hospital recruited patients [Bassin 2006, Kim 2011]. Some cancer registries may have less reliable data than others. Hospital-based recruitment may raise questions of selection bias, and the possibility that such selection bias may lead to confounding [Rothman 1998].

A few ecological studies used the outcome of cancer mortality rather than cancer diagnosis. Mortality information was either from individual death certificates or statistical compilations of death certificates for a geographical area. Mortality and death certificate information is likely to be less reliable than cancer diagnosis information. Mortality rates can differ from diagnosis rates due to factors such as treatment effectiveness, which can vary over time, geography, and other factors.

One case-control study compared cases diagnosed with osteosarcoma to controls diagnosed with other types of bone cancer [Kim 2011]. If fluoride is associated with the other types of bone cancer, this study might not be able to detect an effect on osteosarcoma.

(4) NAMES OF SCIENTISTS WITH EXPERTISE OR KNOWLEDGE ABOUT THE SUBSTANCE

The following scientists have significant expertise and knowledge about the carcinogenicity of fluoride:

Philippe Grandjean, PhD: Dr. Grandjean is an adjunct professor at Harvard University School of Public Health. He is one of the world's experts on the health effects of fluoride, including carcinogenicity. He inherited Kaj Roholm's historic cohort of occupationally exposed works, and conducted numerous follow-up studies looking at long term health outcomes including cancer. Dr. Grandjean can be contacted by phone at: 617-384-8907 and email at: pgrand@hsph.harvard.edu. For further information, see: http://www.hsph.harvard.edu/philippe-grandjean/

Kathleen M. Thiessen PhD: Dr. Thiessen was a co-author of the National Research Council's 2006 report on fluoride. She has extensive knowledge of fluoride toxicity and exposure. Dr. Thiessen currently serves as Senior Scientist at the Oak Ridge Center for Risk Analysis. Dr. Thiessen can be contacted by email at kmt@orrisk.com. For further information, see: http://www.orrisk.com/thiessen_bio.html

Perry Cohn, PhD: Dr. Cohn has conducted several studies on osteosarcoma in relationship to fluoride and radionuclides. He is retired, but until recently was an epidemiologist at the New Jersey Public Health Department, specializing in

environmental contaminants, especially in drinking water. His phone number is 609-883-1152.

Judith Klotz, PhD: Dr. Klotz was a member of the NRC committee [2006] which reviewed the toxicity of fluoride. She was a lead author of the section on fluoride carcinogenicity. She was formerly an epidemiologist at the New Jersey Public Health Department and has experience in environmental cancer epidemiology. She is currently a professor at the University of Medicine and Dentistry of New Jersey's School of Public Health and can be contacted at judith.klotz@comcast.net. Her cv is available here: http://sph.rutgers.edu/departments/epidemiology/documents/cvs/KlotzJudith.pdf

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