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Date: 9/6/2011 8:54 AM
Subject: Prop 65 Carcinogen Identification Committee Meeting Submission

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September 5, 2011

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RE: Proposition 65 Carcinogen Identification Committee Meeting

Dear OEHHA,

The topic of fluoridation is one of the most politicized issues in health. The benefits of topically applied fluoride to treat oral infections and strengthen enamel to reduce carie formation is well established. The addition of fluoride to the municipal water supply is controversial because fluoride has been shown to kill human cells at very low concentrations and function as a mutagen/carcinogen at higher concentrations. Since fluoride concentrates in bone, it is not surprising that osteosarcoma has been associated with fluoride exposure. Increased incidence of bladder and lung cancers have been reported in fluoride industry workers.

Dental scientists at the University of North Carolina School of Dentisry recently published an article (May 2011) in the Journal of Dental Research confirming that fluoride ingestion "...can lead to disturbances of bone homeostasis (skeletal fluorosis, dental/enamel fluorosis).". They continue: "The severity of dental fluorosis is also dependent upon fluoride dose and the timing and duration of fluoride exposure. Fluoride's actions on bone cells predominate as anabolic effects both in vitro and in vivo. More recently, fluoride has been shown to induce osteoclastogenesis in mice. Fluorides appear to mediate their actions through the MAPK signaling pathway and can lead to changes in gene expression, cell stress, and cell death."(1).

As described above, skeletal & dental fluorosis is manifested in part through cell stress and cell death. The incidence of dental fluorosis in American teenagers was recently studied and posted on the CDC website in November 2010 (2). Take a guess at the incidence of fluorosis in American teens with the following multiple choice question:

In 2010, the incidence of irreversible fluorosis was found in what percentage of American children ages 12-15?:

A.) 1 out of 10,000
B.) 1 out of 1,000
C.) 1 out of 100
D.) 1 out of 10
E.) 1 out of 5
F.) 1 out of 2.4 (41%)

The correct answer is choice F, 41% of American teenagers were recently found to have dental fluorosis.

Meanwhile, in January of 2011 a similar survey of children in fluoridated Mexico City was published revealing a fluorosis incidence of 60% (3). Fluorosis is clearly not a rare toxic side effect of ingesting fluoride.

Last year, researchers from the College of Veterinary Medicine at the China Agricultural University set out to determine if sodium fluoride (NaF) influenced bone cells at very low concentrations (4). They used NaF at a concentration of 1 x 10(-5)M. Fluoridation enthusiasts have had us drinking 1 ppm for decades, which is a molar concentration of 5 x 10(-5)M. In their recently published study in Biochemical and Biophysical Research Communications, June 2011, Yang et. al. report that “NaF was found to reduce [bone] cell viability in a temporal and concentration dependent manner and promote apoptosis even at low concentrations (10(-5)M).”. They found that by using sophisticated methods of analysis in a controlled laboratory environment doses 5 times lower than those used in our drinking water are killing bone forming cells (osteoblasts) by triggering apoptosis. They noted alterations in the expression of bone cell survival genes bax and bcl-2 after exposure to these low concentrations of fluoride.

The issue of fluoride-induced oxidative stress on human osteoblast-like cell line (OS732 cells) and in vivo in rats was evaluated in an article by Liu et. al. published in October 2010. They reported “...inhibiting cell viability depended on fluoride-exposure concentration and period, both accompanied with active oxidative stress.” . Although the rat’s bone cells showed significant oxidative stress, that effect may have been lessened to some degree in rats because they make additional vitamin C in response to oxidative stress. The use of genetically modified rats that can not make the anti-oxidant vitamin C or the use of guinea pigs (naturally unable to make vitamin C) would have more closely resembled the effect of fluoride exposure in humans since we lack the ability to manufacture any vitamin C.

In 2006, Bassin et. al. from the Harvard School of Dental Medicine published evidence revealing a five-fold increase in the risk of developing osteosarcoma among teenage boys exposed to fluoridated water at ages 6, 7, and 8 (5).

Excess evidence of bladder and lung cancers were described in fluoride industry workers by Philippe Grandjean & Jorgen Olsen in the 2004 May 19th edition of the Journal of the National Cancer Institute (6). The authors reported “We previously reported the cancer morbidity from 1943 through 1987 for 422 male cryolite workers [cryolite is sodium hexafluoroaluminate] employed for more than 6 months at the mill from 1924 through 1961. We observed excess incidences of primary cancer of the lungs and of urinary bladder tumors (including bladder papilloma)... We have now extended the follow-up of this cohort by 12 years, at the end of which the total percentage of cohort members who had died exceeded 90%. These findings amplify our previous observation of increased bladder cancer rates among cryolite workers... We therefore believe that fluoride should be considered a possible cause of bladder cancer and a contributory cause of primary lung cancer.”(6).

The issue of choice often emerges in free societies. Scandinavia has debated the topic of water fluoridation and banned water fluoridation in the early 1990’s because they considered it unethical to impose fluoridation on those who do not want to consume it. Perhaps they do not care about their children’s teeth? They cared enough to evaluate the consequences of discontinuation of fluoridation. In 2000, Seppä et. al. at the Institute of Dentistry at the University of Oulu in Finland published a paper entitled “Caries in the primary dentition, after discontinuation of water fluoridation, among children receiving comprehensive dental care.”. They reported the following: “Despite discontinuation of water fluoridation, no increase of caries frequency in primary teeth was observed in Kuopio within a three-year period.” (7). This study is reassuring but not surprising to those who read the dental research demonstrating that it is primarily poor dietary choices and the lack of basic dental hygiene that promotes carvities and gingivitis.

In summary, it is evident that fluoride is a powerful oxidizing agent that causes irreversible harm to human tissues at concentrations of 1 x 10(-5)M. The fluorosis statistics confirm that dental fluorosis is visible in approximately 1 out of 2 children exposed to fluoridation, and the damage is mitigated through free radical generating oxidative damage, a process which is known to increase the risk for cancer (8). Fluoride ingestion is not surprisingly associated with increased incidence of osteosarcoma in teenage boys and increased incidence of bladder and lung cancer in fluoride industry workers. Fluoride is undeniably a poison and it should be recognized as such for Proposition 65. Fluoride should not be ingested by humans at any concentration for any reason due to its persistent, human cell killing, and cancer cell promoting properties.

Sincerely,

Michael Powell, D.O.

1.) Everett ET. Fluoride's effects on the formation of teeth and bones, and the

influence of genetics. J Dent Res. 2011 May;90(5):552-60. Review. PubMed PMID: 20929720

2.) http://www.cdc.gov/nchs/data/databriefs/db53.pdf (see Figure 2). Beltrán-Aguilar ED, Barker L, Dye BA. Prevalence and severity of dental fluorosis in the United States, 1999-2004. NCHS Data Brief. 2010 Nov;(53):1-8. PubMed PMID: 21211168

3.) Jiménez-Farfán MD, Hernández-Guerrero JC, Juárez-López LA, Jacinto-Alemán LF, de la Fuente-Hernández J. Fluoride consumption and its impact on oral health. Int J Environ Res Public Health. 2011 Jan;8(1):148-60. PubMed PMID: 21318021

4.) Liu H, Sun JC, Zhao ZT, Zhang JM, Xu H, Li GS. Fluoride-Induced Oxidative Stress in Three-Dimensional Culture of OS732 Cells and Rats. Biol Trace Elem Res. 2010 Oct 23. PubMed PMID: 20972717

5.) Bassin EB, Wypij D, Davis RB, Mittleman MA. Age-specific fluoride exposure in drinking water and osteosarcoma (United States). Cancer Causes Control. 2006 May;17(4):421-8. PubMed PMID: 16596294.

6.) Grandjean P, Olsen JH. Extended follow-up of cancer incidence in fluoride-exposed workers. J Natl Cancer Inst. 2004 May 19;96(10): 802-3. PubMed PMID: 15150310.

7.) Seppä L, Kärkkäinen S, Hausen H. Caries in the primary dentition, after discontinuation of water fluoridation, among children receiving comprehensive dental care. Community Dent Oral Epidemiol. 2000 Aug;28(4):281-8. PubMed PMID: 10901407.

8.) Dreher D, Junod AF. Role of oxygen free radicals in cancer development. Eur J Cancer. 1996 Jan;32A(1):30-8. Review. PubMed PMID: 8695238.