

New Report Bolsters Fluoride-Cancer Link

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Bulletin #11: New Report Bolsters Fluoride-Cancer Link

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The evidence that fluoride may cause cancer has just become substantially stronger.

In the May 19th issue of the *Journal of the National Cancer Institute*, a 12-year follow-up study of workers in the cryolite industry confirms earlier reports of a link between occupational fluoride exposure and bladder & lung cancer.

In the May 19th paper, the authors - Dr. Philippe Grandjean & Jorgen Olsen – report:

"We previously reported the cancer morbidity from 1943 through 1987 for 422 male cryolite workers 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."

This study is extremely important for a number of reasons.

Relevance to Workers in Aluminum Industry

First of all, numerous studies have documented an increased cancer risk – particularly lung and bladder cancer - among workers in the aluminum industry (see references below). While workers in the aluminum industry are heavily exposed to fluorides, these studies have assumed that the main chemicals causing the cancer are Polycyclic Aromatic Hydrocarbons (PAH), which are also present in the workplace.

What makes Grandjean's & Olsen's study so important, therefore, is that the workers in the cryolite plant being studied were not exposed to PAH.

In October of 2002, while conducting a literature review of occupational fluoride hazards, I emailed Dr. Grandjean. I asked him: "At the cryolite plant, was there any exposure to PAH?"

On October 2, 2002, Dr. Grandjean emailed me back, stating:

"Thank you for asking this question, which is highly relevant since we found an increased incidence of bladder cancer. All cryolite plant processes were at room temperature, and there was no source of PAH other than some machinery and trucks entering and leaving the plant. We therefore concluded that there was no increased exposure to PAH among these workers. I realized too late that we should have included this information in the paper."

In their May 19th report, Grandjean and Olsen explicitly mention the absence of other carcinogens in the cryolite work place. To quote:

"Workers at the cryolite mill in Copenhagen, Denmark, are unique because of their exposure to high levels of fluoride dust and their virtual lack of exposure to other occupational toxicants or carcinogens."

It is difficult to overstate the importance of this observation, especially when considering that lung and bladder cancer are the two main cancers found in the aluminum industry, and that these are consistently

blamed on PAH, not fluoride. Yet, here we have a cryolite plant – with no PAH exposure – and the two main cancers observed are lung and bladder cancer.

Interestingly, the evidence for increased risk of lung and bladder cancer in the aluminum industry has become so strong that even ALCOA - the world's largest producer of aluminum – recently warned its workers of the problem.

In December of 1999, ALCOA sent a memo to its workforce across the globe, informing them that they were at increased risk of developing cancer – namely lung and bladder cancer. According to a December 17, 1999 report from The Associated Press:

“Aluminum manufacturer Alcoa is warning thousands of past and present employees that they may face a greater risk than previously believed of developing lung or bladder cancer.” See: <http://fluoridealert.org/pollution/1375.html>

In Australia,

“In December, 1999, Alcoa sent out about 3000 letters advising former employees to have medical checks for lung and bladder cancer.” See: <http://fluoridealert.org/pollution/1376.html>

Grandjean's and Olsen's research suggests that the fluoride exposure in the aluminum industry is, at the very least, a contributing factor to the increased incidence of these 2 cancers, and that it is misleading to simply focus on PAH.

Additional Evidence that Fluoride May Cause Cancer

Grandjean's and Olsen's observation of a possible fluoride/cancer link gains further support from recent studies examining fluoride's mutagenicity in humans. (Mutagenicity is an important issue to consider when determining if a chemical causes cancer, since many chemicals which produce mutagenic damage also cause cancer.)

Since 1994, 3 studies have been published which report an increased incidence of mutagenic damage in humans exposed to airborne fluorides (Meng 1995, 1997; Lazutka 1999), while 3 other studies have reported an increased incidence of mutagenic damage in humans drinking high levels of fluoride in water (Sheth 1994; Wu 1995; Joseph 2000).

The most common form of mutagenic effects reported in these studies has been a phenomenon known as "sister-chromatid exchange" (SCE).

Wu (1995) described the significance of SCE as follows:

"In recent years, SCE analysis has been considered to be a sensitive method for detecting DNA damage. There is a clear relationship between a substance's ability to induce DNA damage, mutate chromosomes, and cause cancers. The SCE frequency in the human body in peripheral blood lymphocytes is very steady, and does not vary with age or sex. Any increase of the SCE frequency is primarily due to chromosome damage. Thus using a method to detect SCE for exploring the toxicity and harm caused by fluoride is of great importance... The results in this paper showed an obvious increase in the SCE frequency of the patients with fluorosis, indicating that fluorine had some mutagenic effects, and could give rise to DNA damage."

The finding of increased SCE in fluoride-exposed humans has reinforced the possibility – as suggested by numerous laboratory ("in vitro") studies – that fluoride is a mutagenic agent. (See references below)

Again, this is important because most mutagens can also cause cancer.

Relevance to People Consuming Fluoridated Water

In their 1992 paper, Grandjean and colleagues discussed the possible significance of their findings of increased cancer risk in fluoride-exposed workers to people drinking fluoridated water. To quote:

"Should one assume that heavy occupational exposures to fluoride could cause an increased carcinogenic risk, an important question is whether such risk would also pertain to the universal exposure to fluoride at lower intake levels."

In addressing this question, Grandjean stated that the cryolite workers were exposed to roughly 10 times the level of fluoride ingested on a daily basis in fluoridated communities. "However," as he noted,

"the occupational exposure lasted only for a limited proportion of the workers' lifetime and would therefore correspond to a much lower daily uptake as an average for a lifespan... [I]t is not known whether any fluoride-associated cancer risk would be related to a long-term average uptake rather than to peak doses occurring at critical points of time."

While Grandjean doesn't state this himself, a margin of 10 (between the dose possibly causing cancer in the workers and the dose people now receive on a regular basis in fluoridated communities) is actually very small, and far smaller than a safety standard for fluoride would allow if fluoride was ruled a human carcinogen (and then treated like carcinogens are normally treated).

Hence, if Grandjean's observation of an increased rate of cancer among fluoride-exposed workers is accepted as a causal relationship, then the appropriate safety standard would indeed necessitate a substantial reduction of current fluoride exposures – not only among workers in industry, but among the general population as well. Therein lies the potential explosiveness of Grandjean's and Olsen's findings. For it's not just occupational standards that would be at stake.

Other policies at stake would include water fluoridation, tolerances for fluoride-based pesticides, and regulations on fluoridated dental products.

According, for example, to Dr. William Hirzy of the EPA, were fluoride to be classified as a "known", or even a "probable" carcinogen, then the entire policy of water fluoridation – at least in the United States – would be finished.

As Hirzy explains:

“All carcinogens have a Maximum Contaminant Level Goal (MCLG) of zero. That is the health based standard, the one EPA sets saying that at this level we anticipate no adverse health effects in the entire population with an adequate margin of safety. For carcinogens, the policy is no level of exposure is safe, so the MCLG is zero. If it’s zero, it means you can’t add any of this stuff to any water supply and that’s the end of fluoridation” (see: <http://www.fluoridealert.org/hirzy-interview.htm>)

It is apparent, therefore, that Dr. Grandjean’s study of cancer rates in Danish cryolite workers has implications that reach far beyond the confines of the Danish cryolite industry.

Summarizing Recent Evidence Supporting a Fluoride/Cancer Link

As noted above, the evidence supporting a link between fluoride and cancer includes:

1. An increased incidence of bladder and lung cancer among fluoride-exposed cryolite workers who were not exposed to PAH, the chemical assumed to be the cause of the increased rates of bladder and lung cancer in the aluminum industry (Grandjean 1985, 1992, 2004).
2. Increased incidence of mutagenic damage in humans exposed to elevated fluoride in air or water (Sheth 1994; Wu 1995; Meng 1995, 1997; Lazutka 1999; Joseph 2000).
3. Evidence of mutagenicity in laboratory (“in vitro”) studies. (Caspary 1987; Scott 1987; Kishi 1993; Khalil 1995; Mihashi 1996).

Additional evidence – not discussed above – also supporting a link between fluoride and cancer includes:

1. Dose-dependent increase of cancer (osteosarcoma) in target organ for fluoride accumulation (bone) in fluoride-treated male rats (NTP 1990), and an initially reported, but later downgraded, increase in

oral and liver tumors in the animals (Marcus 1990 – see: <http://www.fluorideaction.org/lancet-ntp.htm>).

2. Dose-dependent increase in rare bone tumors (albeit non-malignant) in fluoride-treated rats in Proctor & Gamble's animal bioassay, and the occurrence of 4 malignant bone tumors (albeit without statistical significance) in the fluoride treated animals (DHHS 1991; Maurer 1990).
3. Elevated bone cancer (osteosarcoma) rates among young males in fluoridated versus unfluoridated areas (albeit unrelated to the duration of fluoridation), based on national data from the National Cancer Institute (Hoover 1991), a smaller survey by the New Jersey Health Department (Cohn 1992), and a more recent national analysis (Takahashi 2001). Some epidemiological studies, however, have failed to find this relationship (Mahoney 1991; Freni 1992).

Finally, this is what Grandjean and colleagues had to say about using comparisons of cancer rates in fluoridated vs. unfluoridated to answer the question of whether fluoride causes cancer:

"[S]everal studies have shown that cancer mortality is similar in communities with or without water fluoridation. With regard to such cancer incidence data, however, the limitations of geographic comparisons must be acknowledged; the significance of individual risk factors is unknown, as is the level of individual fluoride exposure, including occupational exposures. With the distribution of processed food and beverages across fluoridation boundaries and with the widespread use of fluoride-supplemented dentrifices, the relative difference in daily fluoride absorption between fluoridated and nonfluoridated communities is likely to be small, thus further limiting the power of such epidemiological comparisons. Further, these ecological studies cannot exclude an increased cancer risk associated with occupational fluoride exposures" (Grandjean 1992).

Grandjean's comments here should serve to highlight the importance of using occupational studies, and animal studies, to determine if fluoride is a carcinogen - especially with today's fluoride-laden food supply masking the difference in fluoride intake between fluoridated and unfluoridated communities.

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