

THE FLUORIDE ACTION NETWORK

FAN Bulletin #531: NRC, part 5: Industry's influence on the MCLG.

March 18, 2006.

Dear All,

In this bulletin, Michael Connett produces another tour de force exposing the fact that the US EPA, when determining the so-called "safe" drinking water standard for fluoride (i.e. the MCLG of 4 ppm) in 1985, relied heavily on studies from industry, or industry-funded scientists, and these were often very dated. The US EPA compounded this shoddy work by ignoring later studies by independent scientists.

Once again we hope - and expect - that the 12 scientists who have been picked to review this standard's appropriateness today will not fall into the trap of relying on scientific work tainted by economic interests.

Paul Connett

How Industry Influenced EPA's Fluoride Safety Standards

by Michael Connett

In a recent bulletin (see FAN #526), we detailed the intense, and ultimately successful, lobbying effort from the State of South Carolina, and various dental institutions, to thwart EPA's attempt to issue a safety standard for fluoride that would protect against moderate/severe dental fluorosis.

It wasn't, however, just South Carolina and dental institutions which influenced EPA's final decision. There was another important, albeit less visible, influence: industry, specifically those that emit large volumes of fluoride (e.g. aluminum, steel, fertilizer, and nuclear processing plants).

Industry's influence, while unquestionably more effective, did not come by way of lobbying or backroom deals. It came through funding the major studies used by EPA to determine the safe vs. toxic levels of fluoride exposure.

Evidence demonstrating industry's influence can be found by following the references contained in EPA's "Drinking Water Criteria Document on Fluoride." See:

<http://www.fluoridealert.org/health/epa/reports/criteria-doc1985.pdf>

The "Criteria Document", issued in October of 1985, provides the scientific basis for EPA's claim that 4 ppm fluoride in water is safe for everyone in the population for an entire lifetime of consumption.

On the face of it, the Criteria document – which is over 190 pages – appears to provide an extensive review of the scientific literature on fluoride toxicity. However, a careful review of the studies cited in the document reveals industry's indelible footprint.

Whereas studies by independent scientists are consistently ignored, studies funded by industries with overt conflicts of interest on fluoride toxicity, comprise the basis for many of the key conclusions in the report, including:

1. The toxic effects of fluoride on animals.
2. The threshold for fluoride-induced kidney damage.
3. The threshold for fluoride-induced thyroid damage.
4. The threshold for fluoride-induced bone changes.
5. The threshold for crippling skeletal fluorosis.

Let's address these one at a time:

1) The toxic effects of fluoride on animals:

(Effects on teeth, bone, reproduction, growth, and accumulation in soft tissues)

In its discussion of fluoride's effects on animals, EPA's Criteria Document relies heavily on research conducted on cattle by Dr. John Suttie at the University of Wisconsin and Dr. James Shupe from Utah State University.

Why did Suttie and Shupe study the impact of fluoride on cattle?

They studied it because, following World War II, farmers across America began suing fluoride-emitting industries for cattle poisonings that regularly occurred downwind of the fluoride emissions.

Industry's fluoride problem was not a small matter. According to a 1972 report from the US Department of Agriculture, "airborne fluorides have caused more worldwide damage to domestic animals than any other air pollutant."

Because of this damage, fluoride proved to be one of the most costly chemical liabilities to American industry from the 1940s through to the 1960s. According to Dr. Edward Groth, fluoride air pollution caused more damage claims to industry between 1957 and 1967 "than all 20 [nationally monitored air pollutants] combined." See: <http://fluoridealert.org/f-pollution.htm>

As a result of these lawsuits, industry was actively interested in determining how much fluoride cattle could be exposed to without experiencing untoward effects – information which they could use in court to defend themselves against farmers whose cattle were exposed to "safe" levels of fluoride. As noted by Frank Seamans, a lawyer at the Aluminum Company of America (ALCOA):

"[A]s soon as this problem was diagnosed and recognized, and we had researched the literature, we realized that there was very little solid information on the subject about what harm fluorides could do, what harm they did not do, and what the tolerance levels were for people, animals and vegetation. To close this gap and to provide solid information on which to build and to deal with this problem, research was encouraged and supported at the University of Wisconsin, Utah State, Stanford Research Institute, University of Tennessee, Kettering Institute, the Boyce Thompson Institute for Plant Research, and other noted scientific centers."

Seamans is referring here to the years following World War II when what he called the "sleeping giant" of fluoride lawsuits was "awakened."

To fight this 'awakened giant', industries such as ALCOA invested heavily in research (on cattle, on humans, and on vegetation) for over 30 years.

This then brings us back to John Suttie and James Shupe.

Suttie and Shupe were, by far, the biggest recipients of industry's financial investment in cattle research. Their research, published in countless papers, provided the scientific basis for nearly all state and federal air pollution standards on fluoride. The two scientists were also regular guests on the witness stand, where they made a habit of defending industry against claims from small farmers. See, for example, <http://www.fluoridealert.org/aluminum-industry.htm>

In EPA's Criteria Document, Suttie's and Shupe's papers are relied on heavily for issues ranging from fluoride's impact on teeth, bones, reproduction, growth, and accumulation

in soft tissues. If one accesses copies of the papers by Suttie and Shupe which EPA cites, one will find the following information:

Suttie 1957a,b; 1958; 1961:

“Supported in part by a grant from the Aluminum Company of America, Pittsburgh, Pennsylvania, on behalf of itself and the Aluminum Laboratories, Ltd., the American Smelting and Refining Co., the Kaiser Aluminum and Chemical Corp., the Monsanto Chemical Co., the Reynolds Metal Co., the Tennessee Valley Authority, the U.S. Steel Corp. of Delaware, and Westvaco, Chemical Division of Food Machinery and Chemical Corp.”

Shupe 1963:

"The study was supported in part by a grant-in-aid from Columbia Geneva Steel Division, United States Steel Corporation, Provo, Utah."

Perhaps just as telling as EPA's heavy reliance on Suttie's and Shupe's industry-funded research, was EPA's omission of any reference to Cornell scientists Dr. Lennart Krook and Dr. Ronald Minor.

Unlike Suttie and Shupe, Krook and Minor were not funded by any fluoride-polluting industry. Their research, published in the late 1970s and early 1980s, reported that cattle could be harmed at considerably lower levels of exposure than estimated by either Suttie or Shupe.

2) The threshold for fluoride-induced kidney damage:

In its Criteria Document, EPA repeatedly asserts that fluoride does not damage kidneys until the concentration of fluoride reaches 100 ppm in water. By assuming this statement to be true, EPA dismisses any possibility of fluoride-related kidney damage at the 4 ppm MCLG.

The reference which EPA cites to support this statement is a 1965 book co-authored by Dr. Harold Hodge and Dr. Frank Smith.

It is hard to overstate the importance of Drs. Hodge and Smith to current fluoride safety standards for humans. Their influence on the scientific understanding of how fluoride impacts human health rivals the influence Suttie and Shupe had on the understanding of how fluoride impacts cattle.

As with Suttie and Shupe, Hodge and Smith also had conflicts of interest on fluoride. In addition to their work at the University of Rochester, Hodge and Smith both worked for the Atomic Energy Commission (AEC).

Because the nuclear industry required massive amounts of fluoride to produce atomic weapons and energy, the AEC – and its contractors – were major sources of fluoride

pollution, both to downwind communities and the men who worked in the plants.

As detailed in Chris Bryson's book *The Fluoride Deception* (Seven Stories, 2004), Harold Hodge was one of the main AEC scientists researching the safety of fluoride compounds from the 1940s through to the 1970s. Hodge's research on fluoride was funded as a direct result of AEC's concern about the liability its contractors faced from fluoride-exposed workers and communities. See:

<http://www.fluoridealert.org/wn-414.htm> &
<http://www.fluoridealert.org/bryson.htm>

In addition to his research on fluoride, Hodge was also paid by the AEC to study the health effects of uranium and plutonium – chemicals of obvious concern to the nuclear industry. As part of this research, Hodge helped coordinate human radiation experiments at a hospital in Rochester, NY, where patients were unknowingly injected with plutonium to see how varying levels of plutonium would affect the human body. See:

<http://tinyurl.com/f9wq6>

The discovery of Hodge's involvement in the AEC's human radiation experiments has raised questions about his professional ethics. But what about his science?

Before addressing Hodge's statement about kidney damage, it is instructive to note some of the other demonstrably false claims that Hodge made during this period.

In the 1960s Hodge repeatedly asserted – based on his and Frank Smith's experiments on humans and animals – that people with kidney disease would NOT retain more fluoride in their body. Hodge claimed this to be true even for animals and humans with severe kidney disease. To quote:

“Serious kidney injury or disease does not interfere with fluoride excretion, e.g. in rabbits given near-fatal doses of uranium (a kidney poison), in rats poisoned with fluoride, in elderly patients and in children suffering from kidney disease” (Hodge 1963).

It is difficult to imagine how Hodge and Smith were unable to find increased fluoride retention with kidney disease. Virtually every study that has since examined the issue, has found that an increase in retention consistently occurs. It is, today, one of the most widely accepted facts in the entire fluoride debate. The fact that Hodge and Smith did not find the effect – in repeated studies - should raise eyebrows about the quality of their research.

This is not the only error, however, that should raise some eyebrows.

In 1956, Hodge claimed it was “impossible” for an accident with fluoridation equipment to cause acute fluoride poisoning. He further stated that a major fluoridation malfunction could occur everyday for 10 years and people would still not suffer “serious toxic consequences.” To quote:

“Sometimes the question is raised, What would happen if there were a mechanical breakdown at the fluoridation plant and all of one day’s supply of sodium fluoride or sodium silicofluoride were suddenly dumped into the water? If this large weight of fluoride could be dissolved, mixed and distributed within an hour, there would still be a factor of safety sufficient to predict that the water could be drunk for ten years or more without serious toxic consequences... it is clearly impossible to produce acute fluoride poisoning by water fluoridation.”

50 years after Hodge made this statement, it is now well known that water fluoridation accidents can, and do, result in acute poisoning – including death. For a list of documented poisonings since the 1970s, see:

<http://www.fluoridealert.org/health/accidents/fluoridation.html>

Hodge’s claim that 100 ppm is the lowest concentration of fluoride that can damage kidneys is similarly flawed. Hodge made the claim based again on his own animal research, as well as a review of other animal studies.

While Hodge’s claim was dubious when he made it (e.g. some animal studies had already reported effects at levels as low as 1 to 15 ppm, and human studies from India had reported effects at levels between 2.5 and 12 ppm), it can now be regarded as just plain false.

Animal research published in the past 10 years – including a long-term study by scientists at the EPA and a study by NIH-funded toxicologist Gary Whitford – has reported that fluoride can damage the kidney of animals at levels as low as 1 and 10 ppm, depending on the duration of the exposure (Varner 1998; Whitford 1999).

Of course, EPA can’t be faulted for not citing studies published after 1985. It can be faulted, however, for relying on Hodge’s 1965 claim without making any mention of a later 1975 study on monkeys (Manocha 1975) which found evidence of kidney damage at 5 ppm – a concentration twenty times lower than Hodge’s purported “threshold.”

3) The threshold for fluoride-induced thyroid damage:

In addition to relying on Hodge’s statement that fluoride can only damage the kidneys at 100 ppm, EPA’s Criteria Document also relies on Hodge’s statement that fluoride can only damage the thyroid at 50 ppm.

As with his claim about the kidneys, Hodge’s purported threshold for thyroid damage was questionable from the outset, and less tenable now.

As early as 1958, Galletti and Joyet published clinical evidence showing daily doses of just 2 to 10 mg fluoride could reduce the activity of the thyroid in individuals with hyperthyroidism. This study – and other clinical studies from European doctors reporting fluoride’s effectiveness at reducing thyroid activity among hyperthyroid patients – were ignored by EPA.

Also ignored was a 1985 Russian study by Bachinskii which reported that human thyroid function may be lowered by drinking 2.2 ppm fluoride.

More recent evidence, meanwhile, indicates yet further problems with Hodge's and EPA's 50 ppm threshold – particularly among people with iodine deficiencies. In 1991, a UNICEF-funded research team in China found that humans with iodine deficiencies may be impacted by fluoride levels as low as 0.9 ppm (Lin Fa-Fu 1991), while animal studies have reported that rats with iodine deficiency can be impacted by fluoride levels as low as 10 ppm (Guan 1988).

EPA's safety standards remain based to this day, however, on Harold Hodge's 1960s' assertion that fluoride can not damage the thyroid gland unless the concentration reaches 50 ppm.

4) The threshold for fluoride-induced bone changes (osteosclerosis):

In its Criteria Document, EPA attempts to estimate the threshold dose of fluoride that may cause detectable bone changes (osteosclerosis). To do so, EPA relies heavily on papers by Hodge & Smith (1970) and Dinman et al (1976).

According to the Hodge & Smith paper, bone changes do not occur in fluoride-exposed workers if their urine levels are below 5 ppm.

How did Hodge & Smith reach this conclusion?

A "personal communication" from Dudley Irwin.

Who was Dudley Irwin?

The Medical Director for the Aluminum Company of America (ALCOA).

According to the other paper, by Dinman et al (1976), bone changes do not occur in fluoride-exposed workers if the urine levels remain below 4 ppm.

Who were "Dinman et al"?

Here's how they're identified in the paper:

"From Aluminum Company of America. Dr. Dinman is Corporate Medical Director, Pittsburgh; Dr. Elder is Medical Director, Massena Operations, Massena, N.Y.; Mr. Bonney is Manager, Industrial Hygiene, Pittsburgh; Dr. Colwell is Vice President, Health and Environment, Pittsburgh. Dr. Bovard Tarentum, PA."

Thus, the two studies that EPA's Criteria Document cited for the threshold urinary levels causing bone changes came directly from ALCOA – a corporation with an overt conflict

of interest on the issue, and a corporation known to have withheld important evidence of fluoride toxicity in its workers (see Bryson 2004).

While relying on ALCOA's research, EPA's document fails to disclose the findings from Indian scientists which contradict ALCOA's conclusions (Siddiqui 1955; Singh 1961, 1963). Based on a series of comprehensive field surveys, the Indian scientists reported that skeletal fluorosis can occur in India among people with average urinary fluoride levels as low as 2.8 ppm.

The Indian scientists noted the contradiction between their findings and ALCOA's as early as 1963. According to Singh et al (1963):

“industrial workers whose urinary fluoride is less than 5 ppm do not develop osteosclerosis. However, in our series... there were many persons who excreted less than 5 ppm, yet had dense osteosclerosis.”

5) The threshold for crippling skeletal fluorosis:

EPA's safe water standard for fluoride is designed to prevent against only one fluoride-related effect: crippling skeletal fluorosis (a severe arthritic bone disorder). In its Criteria Document, EPA estimates the dose of fluoride that can cause crippling fluorosis by relying on the following two sets of data:

- a 1950 estimate by Harold Hodge and Gerald Cox of the dose that caused crippling fluorosis in cryolite workers (20 mg/day), and
- a series of studies conducted by the National Institute of Dental Research in the 1950s comparing the skeletal health of people living in high fluoride (4-8 ppm) vs low-fluoride areas of Texas and the Southwest.

Let's take the 2 sets of data one at a time:

A) Dose that causes crippling fluorosis:

EPA's Criteria Document cites 20 mg/day as the minimum dose of fluoride that may cause crippling fluorosis. While EPA cites this information as coming from the Surgeon General (1983), the scientists who first proposed this estimate are Gerald Cox and Harold Hodge (Cox & Hodge 1950).

We already know who Harold Hodge was – but what about Gerald Cox?

Gerald Cox was an ALCOA-funded scientist at the Mellon Institute in Pittsburgh. In 1939, after scientists from the American Water Works Association recommended a Maximum Contaminant Level for fluoride of 0.1 ppm (to prevent dental fluorosis), Cox argued that the “trend towards complete removal of fluoride may need some reversal.” Cox made the suggestion based on his own animal studies (funded by ALCOA) which suggested that rats given fluoride had stronger teeth.

In the 1950 paper which Cox co-authored with AEC's Hodge, the two scientists stated (based on a questionable interpretation of research conducted on industrial workers in 1937 and 1941) that 20 mg/day estimate would be the minimum dose of fluoride that could cause crippling fluorosis.

While Cox co-authored the 1950 paper, it was Harold Hodge who was ultimately the one who popularized the 20 mg/day estimate – thanks to continued repetition of the estimate in numerous publications for over 25 years.

Indeed, by the time the EPA based their standard on Hodge's and Cox's estimate, citing the Surgeon General (1983) as the source, they probably weren't even aware where the information came from, or how it was derived.

(For a full discussion of why 20 mg/day is an inappropriate threshold to use for crippling fluorosis, and bone damage, see:

<http://www.fluoridealert.org/health/epa/critiques/fan051216.pdf>)

B) Water fluoride levels that do not cause crippling fluorosis:

While the EPA Criteria Document cites 20 mg/day as a dose that can cause crippling fluorosis, it also cites 8 mg/L (ppm) fluoride in water as a level that would not be expected to cause crippling fluorosis, or any form of bone damage. To support this statement, EPA refers to a series of studies published in the 1950s and funded by the National Institute of Dental Research (Leone 1954, 1955; McClure 1954; Stevenson & Watson 1957).

There are several problems, however, with the EPA relying heavily on the NIDR's 1950s studies.

First, the studies have since been contradicted by later papers, including studies documenting crippling fluorosis in the US at 2 to 7 ppm (Sauerbrunn 1965; Goldman 1971); and a Mayo Clinic report documenting advanced skeletal fluorosis in kidney patients drinking 1.7-2.0 ppm (Johnson 1979). EPA's Criteria Document makes no reference to these more recent studies. (EPA conceded in an addendum to the report that they were not aware of the latter research while writing the document.)

Second, the NIDR's research was conducted in a very politically charged climate, where the NIDR had a strong incentive to (retrospectively) prove fluoride's safety in water – since the agency had been promoting fluoridation programs across the country (with virtually no safety data) prior to initiating the research.

According to Dr. Edward Groth, a longtime senior scientist at Consumers Union who wrote a PhD thesis at Stanford University examining the water fluoridation controversy:

“The question of why there is not more American research on skeletal fluorosis is worth examination. I think there are several reasons. First and foremost is the

widespread perception that the disease does not occur in this country. That is a result of a few studies, done in the 1950's or 1960's for the most part, which effectively declared that the disease did not exist here. In contrast to the Indian research (and similar epidemiological studies in other countries), American studies were done with a political purpose -- to demonstrate that the fluoridation of water supplies would not pose any hazards to health. They were not done out of any strong scientific motivation to understand or prevent a potentially serious public health problem. By comparison to the other research... the US studies were very crude. They looked only for the most obvious and unambiguous signs of harm (i.e., crippling skeletal fluorosis, or clear osteosclerosis). There was evidently no concern with potential subclinical changes that could occur at doses lower than those that cause obvious, unambiguous stages of the disease.” See: <http://www.fluoridealert.org/health/epa/critiques/groth-1986.pdf>

Whatever the problems, however, with NIDR’s 1950s’ research, it can at least be said that it was not influenced by industry. Or can it?

According to investigative journalist, Chris Bryson, the line between the NIDR and industry in the 1950s was not nearly as clear and distinct as one might assume.

Over the past 10 years, Bryson has obtained a series of documents which show that the lead author of the NIDR’s studies – Dr. Nicholas Leone - worked closely during his research with a group of scientists and lawyers known as the “Fluorine Lawyers Committee.” Members of the Lawyers Committee, while representing different companies (e.g. ALCOA, Reynolds, etc), shared a common goal: to defeat lawsuits where fluoride-related health damage was being alleged.

According to the documents unearthed by Bryson, the NIDR’s Nicholas Leone communicated regularly with the lawyers about his findings, including mailing advanced copies of his papers for utilization in court, and coordinating with ALCOA’s Medical Director, Dudley Irwin, about how NIDR’s studies could be presented in a manner that would “best suit our purpose” (Bryson 2004).

Documents further reveal that the Fluorine Lawyers Committee was aware of the specific advantages of research coming from the NIDR, rather than industry. As noted in a 1956 letter from committee member, Robert Kehoe:

“The results of [NIDR] investigations are highly advantageous in that the problem exists outside of industry, thereby involving situations in which the economic factors tend to be of different type and significance than those which are often alleged to be active in the industrial world, and often involving investigators who are not subject to accusations of bias based on industrial associations.”

Aware of the “highly advantageous” nature of government (vs industry) research, Kehoe may have actually been the one to propose the idea of the NIDR studies in the first place.

In a May 1952 letter to the Public Health Service, Kehoe – writing on behalf of ALCOA,

Reynolds Metals, Kaiser Aluminum, and 5 other companies – requested the very type of research that Leone and the NIDR ended up conducting several years later. To quote:

“In a meeting a little while ago, the question was raised, naturally, as to the long-term influence of small quantities of fluorides, such as those which might be taken in with drinking water, both in areas in which fluorides occur in somewhat unusual concentrations in the drinking water as well as those areas in which fluorides are being added to community water supplies... I feel that I should transmit to you the opinions by this group and by the industries for whom they speak, not as a matter of their right to request any activity on the part of the Public Health Service, but rather as evidence of their interest in a broad problem of public health. That this interest has been aroused by their concern for the employees of their own companies, is a phenomenon which seems to me of some public consequence.”

According to Bryson, these and other documents from Kehoe’s collection, indicate that:

“the fluoride research of the National Institute of Dental Research, ostensibly conducted to prove water fluoridation ‘safe,’ was covertly performed in concert with industry, which was aware that the medical data would help their Fluorine Lawyers battle American pollution victims and workers in court.”

Conclusions:

EPA’s “Drinking Water Criteria Document on Fluoride” provides the scientific justification for current EPA safety standards on fluoride. Understanding the problems and limitations in this document can lend valuable insight into “how we got to where we are.”

While research conducted by industry-funded scientists is not inherently flawed or incorrect, it is commonly viewed as suspect and self-serving. In combing through EPA’s Criteria Document on Fluoride, industry’s science has left an indelible mark. Whether this influence was ultimately helpful, or detrimental, to EPA’s duty of enacting a standard that protects all members of society – including the most vulnerable - remains to be fully understood.

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