February 20, 2009

Agency for Toxic Substances and Disease Registry
ATTN: Records Center
1600 Clifton Road, NE, MS F-09
Atlanta, GA 30333

RE: Comments on the Public Health Assessment for K-25 and S-50 Uranium Fluoride Releases, Oak Ridge Reservation (USDOE) in Oak Ridge, Roane County, Tennessee (December 23, 2008)

Dear Sirs:

Attached are several comments on the ATSDR’s recent report on uranium and fluoride releases from the K-25 facilities near Oak Ridge, Tennessee. Please note that these comments deal only with fluoride issues and do not constitute an exhaustive review of the report (or even of the fluoride issues themselves).

I will be happy to discuss these comments further or to provide additional references.

Sincerely,

Kathleen M. Thiessen, Ph.D.
Senior Scientist
Comments on the Public Health Assessment for K-25 and S-50 Uranium Fluoride Releases, Oak Ridge Reservation (USDOE) in Oak Ridge, Roane County, Tennessee (Dec. 23, 2008)

Kathleen M. Thiessen, Ph.D.
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As a scientist with a risk assessment firm in Oak Ridge, I was involved in the Oak Ridge Dose Reconstruction project that was carried out during the 1990s to evaluate the potential effects on the off-site population of contaminants released from the Oak Ridge Reservation since the 1940s. I am also the author of an EPA document on the health effects of airborne fluorides (USEPA 1988) and one of the authors of a National Research Council report on fluoride toxicology (NRC 2006).

In January 2001, I sent a letter to the chair of the Oak Ridge Reservation Health Effects Subcommittee with copies to the ATSDR, pointing out that fluoride releases from the Oak Ridge Reservation had not been quantitatively assessed during the Oak Ridge Dose Reconstruction and that they needed to be quantitatively assessed. Therefore, I am pleased to see that some attention has finally been given to fluoride releases from the Oak Ridge Reservation. However, the current ATSDR report does not address all aspects of the Oak Ridge fluoride releases, nor does it consider all of the relevant information on fluoride toxicity.

The comments below are offered to assist the ATSDR in improving its assessment of Oak Ridge fluoride releases. Please note that these comments deal only with fluoride issues and do not constitute an exhaustive review of the report (or even of the fluoride issues themselves).

(1) In its responses to various public comments, the 2008 ATSDR report states that fluorine and fluoride compounds “are primarily associated with acute (short-term) health effects, whereas the state was interested in evaluating chronic exposures” (p. 138, response to comment 29) and “are primarily associated with acute exposures—they are not generally related to chronic, long-term health effects which the state was investigating” (p. 139, response to comment 30). These statements are not correct. In fact, the report from the Feasibility Study did not give a specific reason for the exclusion of fluorine or various fluoride compounds from any quantitative assessment. “Fluorine and fluoride compounds,” “Hydrofluoric acid,” Fluorine and various fluorides,” and “Chlorine trifluoride” were categorized as “acids/bases” for which collectively it was stated that the primary health effect is irritation, commonly associated with acute exposure (ChemRisk 1993b). However, at least for fluorine and fluoride chemicals, this statement is not correct.

While acute exposures to fluorine and fluoride are certainly a potential danger and deserve proper evaluation, fluorine and fluoride compounds are also associated with chronic, long-term effects from either inhalation or ingestion exposures. This was well known at the time of the K-25 and Y-12 releases (1944-1995) and certainly by the time of Phase I of the Oak Ridge Dose Reconstruction (the Feasibility Study, 1992-1993). See for example a 1937 monograph on fluoride toxicity (Roholm 1937), the cold-war era literature on fluoride toxicity (e.g., Hodge and
Smith 1965; various AEC studies), and an EPA health effects summary for airborne fluorides (USEPA 1988).

In addition, fluorine and fluoride compounds were released from both K-25 and Y-12 in massive quantities; the DOE (2000) states that the principal nonradionuclide emissions from the K-25 site included fluorine and HF. Even if the principal health effects for fluorine and fluorides were due solely to acute exposure, the magnitude of the releases should have suggested that further assessment was required. It is apparent from the annual environmental reports (1971-1985) that ATSDR cites and from material cited in the Feasibility Study report (ChemRisk 1993a) that the government considered fluorine/fluoride something important to monitor; this would not have been the case for a substance that was only an acute hazard or irritant.

(2) In the 2008 report, ATSDR has considered only fluoride releases associated with uranium releases, a set of measured fluoride concentrations in air (1971-1985), and estimates of airborne fluoride concentrations based on the uranium fluoride releases. Also, the report considers fluoride losses only from K-25. There is additional information in existence that should be considered in assessing fluoride exposures from Oak Ridge-area facilities.

First, fluoride in association with uranium was released from Y-12 as well as K-25. The ATSDR has assessed Y-12 uranium releases in a separate report, but has not addressed fluoride releases from Y-12. The estimated uranium releases from Y-12 over the entire period (1944-1995) were approximately 50,000 kg (ChemRisk 1999, p. D-5), as compared to about 16,000 kg of uranium released from K-25 over the same period (ChemRisk 1999, p. 2-27). Clearly, a considerable amount of fluoride could have been released from Y-12 as well as from K-25. In my 2001 letter, I provided a rough lower-bound estimate of 15,000 kg of F released from Y-12 and K-25 combined (from 1944-1995) as a part of the uranium fluoride releases, without considering releases of $F_2$, HF, or other fluoride compounds.

However, for K-25 in particular, it is known that large amounts of fluorine or fluoride (primarily as gaseous HF or $F_2$) were used and released apart from the uranium fluorides (DOE 2000). The Department of Energy describes the routine and accidental atmospheric releases of HF and $F_2$ from K-25 as having not been well documented, but with annual releases reaching 22,000 lb (10,000 kg) of $F_2$ in 1957 and 58,500 lb (27,000 kg) of HF in 1954 decreasing to 44,000 lb (20,000 kg) of HF in 1957 (DOE 2000, based on a 1957 study). Bryson (2004) cites a 1955 document from the Oak Ridge DOE Public Reading Room that provides an estimated cost of deliberately vented fluorine of $400,000 per year if recovery of the fluorine were not implemented; at a cost per pound of $0.65, this corresponds to 280,000 kg of fluorine per year. Fluorine/fluoride emissions from K-25 were reduced in the early 1960s and again in the late 1960s (DOE 2000) due to changes in plant processes and emissions control. Accidental releases of fluorine or fluorides include a release of 5000 lb (2300 kg) of HF in the 1974-1976 period (DOE 2000).

The ATSDR (2008) does not mention the DOE report or the documentation cited by Bryson (2004) and does not seem to have followed up on these estimates or information sources. Nor has the ATSDR attempted to estimate the possible exposure of members of the public to fluorine.
releases of this magnitude. The ATSDR (p. 97) mentions a correlation between the annual uranium releases and measured fluoride concentrations at the site perimeter (based on the 1971-1985 monitoring data) and the use of this correlation to estimate fluoride concentrations prior to the monitoring data; given the known large releases of HF and F₂ during the pre-1971 period (especially prior to the 1960s) apart from uranium releases, this approach seriously underestimates the off-site fluoride concentrations for earlier years.

The same annual environmental reports for 1971-1985 from which ATSDR obtained airborne fluoride concentrations also provide some measurements of fluoride in surface waters near K-25 and Y-12 (UCC 1972-1983; MMES 1984-1986). It ought to be worthy of mention that a number of these measurements (well after the 1950s when the peak releases from K-25 probably occurred) reach or exceed the fluoride concentrations reached with deliberate fluoridation of drinking water (up to 3.5 mg/L, vs. 1 mg/L typical of fluoridated water in Tennessee) even though background fluoride concentrations in surface waters in the Oak Ridge area are routinely very low (<0.3 mg/L; e.g., Morton 1962a; 1962b). While the surface waters near K-25 and Y-12 (Poplar Creek, East Fork Poplar Creek, Bear Creek) were not routinely used as drinking water sources for the population and thus might not have contributed significantly to off-site exposures of the public, fluoride concentrations in excess of 1 mg/L (up to 2.6 mg/L) were measured in the Clinch River in 1972 and 1973. Such measurements lend credence to the theories (e.g., Bryson 2004) that fluoridation of public water supplies was at least partially motivated, not for oral health benefits, but as an attempt to downplay the potential significance of fluoride releases to the environment from industrial and nuclear-related facilities. This issue therefore deserves discussion by the ATSDR.

Other bits of information not included by ATSDR include a fluoride vegetation damage study performed in 1957 (DOE 2000) and employee reports of detectable or “overpowering” onsite concentrations of fluorine and HF (DOE 2000). The DOE (2000) also mentions ambient air sampling for HF and fluorine having begun at K-25 in 1959. Vegetation monitoring for fluoride was routinely done at least for the 1971-1985 period. “Elevated concentrations” of fluoride were found in vegetation and some animals on the Oak Ridge Reservation in a 1979 study, well after the fluoride releases at K-25 had substantially decreased (DOE 1979).

(3) In its assessment of the significance of the fluoride releases (ATSDR 2008, pp. 97-98), ATSDR compares estimated airborne fluoride concentrations to concentrations associated with specific health effects. This approach is acceptable if the airborne fluoride is the only source of fluoride exposure or (primarily for acute exposures) the overwhelmingly predominant source of fluoride exposure. However, for long-term exposures and health effects, the total fluoride exposure from all sources (airborne, drinking water, food, dentifrices, etc.) must be considered. This has been stated many times (e.g., Chester et al. 1979; USEPA 1988; NRC 2006). OEHHA (2003) states that fluoride exposures at an acceptable level when a single route of exposure is considered in isolation might be deleterious for individuals with substantial fluoride exposures from other sources. In general, except for direct effects of inhaled fluoride on the respiratory tract, the body does not distinguish between inhaled and ingested fluoride, and it is the total exposure from all routes and sources that drives the health effects. Therefore, it is also necessary to assess K-25 and Y-12 fluoride releases in terms of their contribution to total fluoride...
exposures of off-site individuals (mg/kg/day), and to compare the total fluoride exposures to an appropriate reference exposure.

(4) ATSDR’s 2008 report considers skeletal fluorosis and dental fluorosis to be the effects of concern (e.g., pp. 122, 126-127), with skeletal fluorosis requiring very high intakes over many years and dental fluorosis being a cosmetic effect rather than a health effect. There are a number of additional effects of concern that should be considered, some of which can be expected at lower levels of fluoride exposure than either skeletal fluorosis or dental fluorosis.

On p. 127, in a response to a member of the public, the ATSDR refers to the EPA’s reference dose (0.06 mg/kg/day), which is intended to protect children from severe dental fluorosis, and dismisses its relevance for the K-25 assessment. ATSDR should be aware, first, that 0.06 mg/kg/day is not protective of dental fluorosis in general, and is not necessarily protective of severe dental fluorosis. Second, the National Research Council (2006) concluded that severe dental fluorosis is definitely an adverse health effect rather than merely a cosmetic effect, and even less severe forms of dental fluorosis should be avoided. Finally, the NRC (2006) cites a number of papers that show associations between the presence of dental fluorosis and an increased risk of other adverse health effects (e.g., reduced thyroid function, lowered IQ, increased fracture risk).

It should be noted that the ATSDR’s own Minimal Risk Level (MRL) for fluoride is also 0.06 mg/kg/day, based on an increased risk of bone fracture (ATSDR 2003). Both skeletal fluorosis and increased risk of bone fracture (from increased brittleness of bones due to incorporated fluoride) are relatively severe effects, requiring either high exposures or long exposure durations. The ATSDR should be evaluating more sensitive effects. For example, ATSDR’s toxicological profile for fluoride (ATSDR 2003) refers to an animal study of thyroid function that would give a lower MRL (value not given) than the MRL derived for bone fracture risk. On p. 122 in the K-25 assessment, in a response to a member of the public, the ATSDR (2008) states that “no evidence to date has suggested that fluoride is an endocrine disruptor, but some data from drinking water suggest that exposure to fluoride could potentially affect some endocrine glands” (citing ATSDR 2003). However, the National Research Council (2006) has concluded that fluoride is an endocrine disruptor, based on an extensive review, including studies not reviewed by ATSDR (2003).

For many health endpoints, a lowest observed effect level (not a no-effect level) for fluoride appears to be around 0.05 mg/kg/day (estimated average fluoride intake for a study group); for some endpoints such as altered thyroid function in persons with iodine deficiency, it appears to be even lower. Several population subgroups (e.g., persons with renal impairment) have been identified that are at higher than usual risk of adverse effects from fluoride exposure (NRC 2006). The NRC (2006) states that fluoride appears to have the potential to initiate or promote cancer, and several occupational studies (Grandjean et al. 1992; Grandjean and Olsen 2004; Romundstad et al. 2000) are consistent with an association between exposure to inhaled fluoride and bladder cancer. Finally, a recent paper (Taiwo et al. 2006) discusses increased risk of asthma with chronic exposure to inhaled fluoride.
References


