Health Consultation

Federal Creosote Site (20JJ)
Manville, New Jersey

January 16, 1998

U.S. Department of Health and Human Services Agency for Toxic Substances and Disease Registry Division of Health Assessment and Consultation Atlanta, GA 30333.

Background and Statement of Issues

The Region II U.S Environmental Protection Agency (EPA) has requested that the Agency for Toxic Substances and Disease Registry (ATSDR) review sampling results from the Federal Creosote site in Manville, New Jersey to determine if the levels of contamination pose a threat to public health [1].

The Federal Creosote site is a 35-acre site that once housed a wood treating plant that operated until 1957. Railroad ties were treated with creosote at the site, and the waste was disposed of in two lagoons on site. A residential community consisting of 137 homes was constructed over the former wood treating facility beginning in the mid-1960s [2].

In April 1996, creosote contamination was discovered seeping into the sump in a basement of a home on site. In January 1997, creosote contamination was also discovered six feet below the surface where a sink hole had developed around a sewer pipe [2].

In May 1997, EPA conducted indoor air sampling to determine if volatile compounds were migrating into the homes on site. The EPA conducted sampling in 126 homes. Analytical results were evaluated using health based air action levels developed by ATSDR. According to EPA, the target compounds related to crossote were not detected at appreciable concentrations in the air inside the homes [3].

In May 1997, ATSDR provided a health consultation to the EPA on soil sampling results collected by a consultant for the Borough of Manville [4]. The sampling identified polycyclic aromatic hydrocarbons (PAHs), a constituent of creosote, at concentrations in the low percent range. ATSDR concluded that the PAHs detected in the subsurface soils posed a potential contact threat to area residents if the material were to be unearthed [4]. ATSDR recommended additional characterization of the contamination to include the collection of surface soil samples [4]. The resultant sampling is the subject of this health consultation.

In October 1997, EPA/ERT collected surface soil sample (0-3 inches) from over two dozen properties on site for PAH analysis [5]. Subsurface soil samples were also collected and analyzed for PAHs [4]. Sampling locations were selected based on historical information and input provided by residents on areas of suspected contamination.

Creosote material was identified at approximately 3 to 10 feet below the ground surface. There was no visual evidence of creosote material at the surface during sampling activities [6]. The state of the creosote waste in the subsurface ranged from a hardened material to a material with a liquid-like consistency [6]. Total PAH concentrations in the subsurface soils ranged from non-detect to 74,243 parts per million (ppm). The highest concentrations of

PAHs were detected at depth in the areas around the two former lagoons, or along the canals that transported the creosote waste from the former wood treating area.

Although there was no visual evidence of creosote contamination at the surface, soil at some properties had elevated levels of PAHs. The properties with the highest concentrations of PAHs in the surface soil are listed below:

Sample Location

Total PAHs (ppm)

Residential, Commercial,	Camplain Road- Valerie Drive- "Summit Bank"- Camplain Road-	758 ppm 504 ppm 120 ppm 79 ppm	total	PAHs
Residential,	Valerie Drive- Valerie Drive-	78 ppm 65 ppm		

The remainder of the residential properties had total PAH levels in the surface soil ranging from non-detect to 33 ppm. The surface soil sample from the residential property on Camplain Road (see above, 758 ppm) was collected at a swing set in the back yard.

The EPA has been concerned about the potential for contaminants at the site to impact two municipal wells located within 1/4-mile of the site. These wells serve as a potable water source for both the immediate community and the surrounding area. The EPA is planning to install monitoring wells at the site as an initial step to assess the groundwater exposure pathway [6]. In addition, the NJDEP recently collected samples from the Manville municipal wellfield (sampling results not available).

Discussion

The recent sampling conducted by EPA/ERT confirms earlier findings that show creosote, and its constituent PAHs, at elevated levels in the subsurface. As stated by ATSDR in a previous Health Consultation, if the creosote contamination is unearthed, it may pose an acute contact threat causing irritation and burning of the skin and eyes [4]. The ultraviolet rays of the sun may intensify the effects of the exposure.

Long-term exposure to creosote has been shown to induce skin cancer in animal studies, but evidence of cancer in humans is less definitive. Some studies of workers showed an association between long-term exposure to creosote and the development of skin cancer. However, other studies suggest there is no association between exposure to creosote and other coal tar products and cancer in humans [6]. Since creosote used for wood treatment contains a mixture of several carcinogenic PAHs, it is prudent to assume that there may be an increased risk of cancer associated with long-term exposure to creosote.

Non-cancerous adverse effects have also been associated with exposures to PAHs [7]. Mice fed high levels of the PAH, benzo(a)pyrene (B(a)P), during pregnancy had difficulty reproducing. Also, the offspring of mice fed high levels of B(a)P had an increased incidence of birth effects and decreased body weight [6]. There are no studies that indicate these effects occur in people, and the doses that cause these effects are considerably higher than those likely to be experienced through environmental exposures.

Exposure to PAHs in the soil is most likely to occur through incidental ingestion of soil or dust. Some dermal absorption of PAHs may occur through direct contact with skin. However, PAHs bind to organic matter in the soil, which decreases its bioavailability through skin absorption.

The following health evaluations are based on the highest concentrations of PAHs detected in on-site surface soils.

Non-cancer assessment:

The EPA and ATSDR have derived health-based guidelines for several PAHs. These guidelines are defined as estimates of a daily oral exposure of humans, including sensitive sub-populations, that are likely to be without an appreciable risk of deleterious effects (non-cancer). Such guidelines are not thresholds for toxicity, but are useful for screening to determine if more detailed evaluations are necessary. The EPA guidelines are referred to as a Reference Dose (RfD), and the ATSDR guidelines values are Minimal Risk Levels (MRLs).

Assuming a worst case scenario, a 3-4 year old child (weight-16 kg) ingesting 200 mg of soil per day while playing on the property with the swing set (total PAHs-758 ppm), the child would receive a dose of 0.0094 mg/kg/day. This dose is 10-100 times below any screening values used by ATSDR, and several thousand times lower than levels that have caused effects in animals.

Cancer Assessment:

Several PAHs have been shown to have a carcinogenic potential. The potencies of the individual PAHs vary, so mixtures of PAHs are evaluated by expressing their carcinogenic potential as B(a)P equivalents. The following two scenarios assess cancer risks for adults and children who are exposed to PAH mixtures.

1). 30-Year Exposure

The highest concentrations of PAHs at the site in B(a)P equivalents was 80.9 ppm [8]. This was derived from the surface soil sample that had a total PAH concentration of 504 ppm. Using standard default values to evaluate potential carcinogenic risk: 30 year

exposure to 80.9 ppm soil B(a)P equivalents (50 milligrams of contaminated soil ingested per day, 70 kilogram body weight), a cancer risk of 1.8E-04 is calculated. Calculation - (80.9 milligrams/kilogram) x (1/70 kilograms) x (50 milligrams soil/day) x (1 kilogram/106 milligrams) x (30/70) years = 0.000025 mg/kg/day; multiply by Cancer Slope Factor (7.3 mg/kg/day) 1 = 1.8E-04 estimated cancer risk. This is equivalent to 1.8 excess cancers per 10,000 exposed individuals.

2). 6-Year Exposure to Child

Again using standard default values to evaluate the potential carcinogenic risk: A 6-year exposure of a child to 80.9 ppm soil B(a)P equivalents (200 milligrams of contaminated soil ingested per day, 15 kilogram body weight), a cancer risk of 6.7E-04 is calculated. Calculation - (80.9 milligrams/kilogram) x (1/15 kilograms) x (200 milligrams soil/day) x (1 kilogram/106 milligrams) x (6/70) years = 0.000092 mg/kg/day; multiply by Cancer Slope Factor (7.3 mg/kg/day) = 6.7E-04 estimated cancer risk. This is equivalent to 6.7 excess cancers per 10,000 exposed individuals.

The cancer risks calculated above for the two scenarios are considered unacceptable for contamination left in place. However, these calculations are very conservative, and the actual risks are likely to be much lower. For example, the calculations assume (1) continuous exposure and does not take into account winter months when exposure to the soil is limited (2) assumes 100% absorption of the ingested PAHs, and (3) does not account for a vegetative cover (e.g. grass) that serves as a protective barrier to exposure.

The calculations made above were also based on only one surface soil sample per yard. Typically, several samples are required to assess a site. Further, since no visible creosote contamination has been noted at the surface, the sampling results may have been skewed by a piece of hardened creosote material, and may not be representative of the soil that is likely to be ingested. More surface soil data would provide a better indication of the extent of contamination, and the likelihood of exposure.

Groundwater Pathway:

Although PAHs bind to the soil and do not migrate via groundwater very readily, there is a potential for creosote and some of its volatile fractions to do so. The installation and sampling of groundwater wells will assess this potential exposure pathway.

Conclusions

Based on the information provided, ATSDR concludes the following.

- 1. PAHs were not detected in the surface soils on site at levels that pose an acute (short-term) health threat.
- 2. The creosote contamination detected in subsurface soil does not pose an immediate health threat unless the material is brought to the surface. If this should occur, the contaminated soils pose an acute health threat.
- 3. Some residential properties contained PAHs in surface soils at levels that represent a potential long-term health concern. However, sampling is limited to only one surface soil sample per property, and additional sampling will be required to assess the public health threat.

Recommendations

- 1. Collect additional surface soil samples in the residential yards. Ensure that sampling includes composite samples, as well as some discrete samples in areas of suspected contamination (e.g. adjacent to former lagoons or canals).
- Inform residents not to dig into areas of creosote contamination. If creosote contamination is unearthed, avoid contact with the skin.

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References

- [1] Verbal request from EPA Region II to ATSDR in December 1997.
- [2] March 27, 1997 Letter from NJDEP to Richard Caspe, U.S. EPA
- [3] EPA Memorandum From Raj Singhvi to Nick Magriples, OSC, September 9, 1997.
- [4] ATSDR Health Consultation, Federal Creosote Site, Timothy Walker, M.S., May 1, 1997.
- [5] EPA Federal Creosote Site Analysis, November 1997.
- [6] Verbal comments received by EPA Region II during a site visit by ATSDR on December 19, 1997.
- [7] ATSDR Toxicological Profile for Creosote, TP-90-09, December 1990.
- [8] EPA Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons, 1993.