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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service
Agency for Toxic Substances
and Disease Registry

Memorandum

Date
August 11, 1987

From
Michael A. McGeehin
Senior Regional Representative
ATSDR - Region VIII

Subject
Health Consultation: Walkerville, Montana

To
Robert Duprey, Director
Hazardous Waste Management Division
EPA - Region VIII

REQUEST

The Environmental Protection Agency (EPA) has requested the assistance of the Agency for Toxic Substances and Disease Registry (ATSDR) in reviewing the sampling performed by the Emergency Response Branch on the Alice Mine/Sherman Ballfield Site in Walkerville, Montana. EPA is specifically interested in whether these sampling results represent a threat to the health of the community.

DOCUMENT REVIEWED

Preliminary Endangerment Assessment for the Alice Mine Site/Sherman Ballfield, Walkerville, Silver Bow County, Montana, and appendices.

BACKGROUND

From 1881 until 1959, the area around the town of Walkerville was the site of virtually continuous mining and milling activity. The Alice Mine was primarily mined for silver and the amalgamation process utilized large amounts of mercury. The waste materials from the mining, milling, and amalgamation processes were consolidated in dumps and piles, which are found throughout the community.

Additional contamination in the area include the Moulton, Lexington, and Paymaster mines. The Sherman Ballfield was created in the late 1930's as a recreational facility for the community's children. The ballfield was constructed by leveling off a section of the waste piles. The facility was used by the residents through 1986.

The Alice Mine/Sherman Ballfield site is a portion of the Silver Bow Creek National Priorities List (NPL) site. Although a Remedial Investigation/Feasibility Study is currently being developed, there will be little actual remediation work undertaken through this mechanism in the near future.

CDM has estimated that the Alice open pit dumps contain 5,560,000 cubic yards of material and cover approximately 46 acres.

SAMPLING

In March 1986, soil sampling of the Sherman Ballfield by the Montana Bureau of Mines and Geology indicated the following elevated levels of metals: 107 ppm of cadmium, 11,000 ppm of lead, and 2,500 ppm of mercury at a depth of 1.5-1.8 feet. Free elemental mercury was evident at this depth.

During November 1986, and January 1987, 611 soil samples were collected for the EPA to determine the extent of contamination within Walkerville. These soil samples were collected from 64 different locations determined by the presence of historically documented mill dump sites. These samples revealed extensive contamination throughout the area, particularly the Paymaster dump and the base of Sherman Ballfield. Maximum contaminant levels were: 522 ppm of arsenic, 71 ppm of cadmium, 14,200 ppm of lead (1.4%), and 86 ppm of mercury.

Of equal or greater importance were the drainage areas sampled. These results indicated the concentration of contaminants in the drainages suggesting extensive migration of metals through surface runoff, erosion, and wind entrainment. These drainage samples also indicate a potential source of mercury north of the site boundaries, at the site of the Margot Anne mine.

Elevated metal contamination was found in areas of frequent recreational use. The perimeter of the Sherman Ballfield yielded a maximum concentration of lead of 60,900 ppm (6.09%) and the Walkerville tennis courts (also constructed on mine wastes) showed 51,000 ppm maximum concentration of lead.

DISCUSSION

The population of Walkerville is approximately 850-900 people, with 70 to 100 homes being directly impacted by surface runoff and erosion. Without further information, however, the entire community may be considered to be affected by wind entrainment of the mining and milling waste and by direct exposure to the high levels of metals in the recreational areas.

There are no bodies of water in the study area. Therefore, surface water contamination is not an issue at this site, except for runoff from the waste piles at times of heavy rainfall or spring snow melt. The geological conditions present at Walkerville also eliminate groundwater from consideration as a pathway of contamination.

The high levels of lead found throughout the site are a major health concern, particularly with regard to the community's children. Soil and dust that contain lead are often an important source of lead exposure for children.

Children obtain lead from dust and soil as a result of their normal exploratory behavior (Baltrop, 1966; Sayre et al., 1974; Roels et al., 1976), coupled in some instances with pica. Because of these mouthing tendencies, children must be considered at risk of exposure both by inhalation of airborne lead-containing dust and by ingestion of deposited lead from soil and dust.

In general, lead in soil and dust appears to be responsible for blood lead levels in children increasing above background levels when the concentration in soil exceeds 500-1,000 ppm (CDC).

The most severe symptoms of acute lead poisoning may involve coma, seizures, and bizarre behavior. These symptoms are associated with extremely high blood lead levels (>100 ug/dl), however, and would not be expected to result from environmental exposure. According to the Centers for Disease Control, the levels represented by the sampling results in Walkerville would result in an increased body burden of lead for the children of the community.

Although children with increased body burden of lead are clinically asympto-

matic, it is likely that they have pervasive metabolic effects involving heme synthesis (Piomelli et al., 1982; Piomelli, 1973; Chisolm, 1978; Alessio et al.,1976), red cell nucleotide metabolism (Angle et al., 1980), renal function (Chisolm, 1968), and subclinical neurobehavioral effects (Needleman et al., 1979; Otto et al., 1982). Some of these metabolic and cellular effects of lead have been observed at blood lead concentrations <25 ug/dl.

It is important to emphasize that young children are particularly vulnerable to the effect of lead on neurophysiological performance.

Another potential health concern is the elevated levels of mercury found in the area. Chronic exposure to mercury vapor may effect the central nervous system (Friberg and Vostal, 1972). Early signs are nonspecific, but the syndrome includes one or more of the following clinical findings: tremor, thyroid enlargement, tachycardia, gingivitis, hematological changes and increased excretion of mercury in the urine. This syndrome and other symptoms of mercury vapor exposure have been chronicled primarily through chronic occupational exposure.

Cadmium is a toxic metal whose principal long term effects of low-level exposure are chronic obstructive pulmonary disease, emphysema, and chronic renal tubular disease (Nomiyama, 1980). Cadmium has been shown to be a teratogen in numerous animal studies.

Cadmium is much more readily taken up by plants than other metals. Therefore, plants grown in contaminated soil may contribute to dietary content and provide an additional pathway of exposure.

Long-term exposure to arsenic may result in liver injury, initially characterized by jaundice, and possibly progressing to cirrhosis. Persons chronically exposed to arsenic in drinking water in Chile have suffered peripheral vascular disease that may progress to gangrene of the lower extremities.

Numerous studies of occupationally exposed populations (pesticides and copper smelters) have shown an increase in lung cancer mortality. Populations living near emissions of arsenic to air may also have an increase in lung cancer mortality, but the studies have been inconclusive.

RECOMMENDATIONS

Based on the data provided, which have been discussed with ATSDR scientists in Atlanta, our agency feels that the levels of metal in the Walkerville may represent a threat to the health of the community. We recommend that:

1. Access by the population to the contaminated areas should be restricted, especially for children. The exposure pathways of direct ingestion of soils containing elevated metals and inhalation of contaminated dusts and soils should be minimized.

2. To assure that all of the extremely contaminated areas are addressed, ongoing sampling should be conducted during the removal action.

3. Residents should be informed concerning the dangers of storing elemental mercury in their homes. There have been anecdotal reports of loosely corked bottles of this element in some of the residences. This may represent an addi-

tional pathway of exposure to mercury vapor, albeit an easily resolved one.

4. A blood lead screening test should be offered to the children of the Walkerville community. With these levels of environmental contamination, some measure of possible lead body burden should be provided.

These recommendations are based on the data provided. They are subject to change as additional data become available. ATSDR will be available to review any additional data.

cc: George Buynoski, Chief, FOB, OEA
Steve von Allmen, HACA, OHA

REFERENCES

Alessio L, et al. Free erythrocyte protoporphyrin as an indicator of the biological effect of lead. Int Arch Occup Environ Health 37:73, 1976.

Angle CR, McIntire MS. Low level inhibition of erythrocyte pyrimidine nucleotidase. Environ Res 1978;17:296.

Angle CR, et al. Erythrocyte nucleotides in children: Increased blood lead. Pediatr Res 16:331, 1980.

Baltrop D. The presence of pica. Am J Dis Child 1966; 112:116-123.

Chisolm JJ Jr. The use of chelating agents in the treatment of acute and chronic lead intoxication in children. J Pediatr 1968;73(1);1-38.

Chisolm JJ Jr. Heme metabolites in blood and urine in relation to lead toxicity and their determination. Adv Clin Chem 20:225, 1978.

Friberg L., and Vostal J. Mercury in the environment-toxicological and epidemiologic appraisal. Chem Rub Co., Cleveland, 1982.

Needleman H, et al. Deficits in psychologic and classroom performance in children with elevated dentine lead levels. New Eng J Med 300:689, 1979.

Nomiyama K.: Recent progress and perspectives in cadmium health effects studies. Sci Total Environ, 14:199-232, 1980.

Otto DA, et al. Effects of low to moderate lead exposure on slow cortical potentials in young children. Neurobehav Toxicol Teratol 4:733, 1982.

Piomelli S, et al. Management of childhood lead poisoning. J Pediatr 1984; 105(4):523-532.

Piomelli S, et al. Threshold for lead damage to heme synthesis in urban children. Proc Natl Acad Sci USA 1982;79:3335-3339.

Roels H, et al. Impact of air pollution by lead on heme biosynthetic pathway in school age children. Arch Environ Health 1976;31(6):310-315.

Sayre JW, et al., House and hand dust as a potential source of childhood lead exposure. Am J Dis Child 1974;127:167.