Lead Poisoning among Bricklayers — Washington State

In May 1989, four members of an 11-man crew of bricklayers in western Washington state developed symptomatic lead poisoning while replacing the brick lining of an acid-accumulation tank at a paper mill. Peak blood lead levels (BLLs) for the four workers ranged from 88 to 123 μg/dL.* An investigation indicated the source of the lead exposure was a special brick mortar that contained 71% lead oxide and was formulated to resist the normally acidic environment of the tank.

The cylindrical acid-accumulation tank was 50 feet tall and 20 feet in diameter; the enclosed top had an access portal 3 feet in diameter. In April 1989, after removal of the old lining, bricklaying for the new lining began. The mortar was prepared outside of the tank by mixing dry mortar powder with water. The mixed mortar, along with new bricks, was then passed through the access portal to bricklayers working in the tank.

On May 4 (approximately 3 weeks after starting the job), the worker who mixed the mortar had onset of fatigue and abdominal pain and left work. His replacement developed headaches, chest pain, and abdominal pain within 2 days of assuming the mixing job. The workers independently sought medical care and reported the possibility of lead poisoning to their physicians on May 4 and 6, respectively. Elevated BLLs (112 μg/dL and 92 μg/dL, respectively) were documented in the two workers. In addition, the first worker was anemic (hematocrit of 31%), and he received a partial course of chelation therapy with oral penicillamine. There were no records of either case being reported to the local or state health departments.

Following recognition of these cases, the subcontractor who had employed the bricklayers introduced changes at the worksite, including improvement of safety training, construction of a separate shed for mixing mortar, provision of facilities to enable workers to shower before lunch and at the end of the work shift, and replacement of paper masks with appropriate forms of respiratory protection (including supplied-air respirators for use during mortar mixing). On May 11, the Washington Department of Labor and Industries (L&I), in response to a worker request, inspected the worksite and found no violations. Following the inspection, however, the project foreman became ill, was diagnosed with lead poisoning (initial BLL: 88 μg/dL), and, on May 15, was hospitalized for 2 days for chelation therapy with intravenous calcium ethylenediaminetetraacetic acid (EDTA). Finally, on May 19, a fourth crew member, who worked as both a bricklayer and a mortar mixer, became ill and consulted a physician; his highest BLL was 123 μg/dL, and he was treated with oral penicillamine. The relining operation was completed in June.

In August 1989, the University of Washington Occupational Medicine Program conducted a follow-up investigation of this outbreak after an affected worker was referred by L&I for independent medical examination. The investigation identified several deficiencies in the protection and monitoring of this group of workers, including the lack of air sampling at the worksite and the failure to test BLLs in workers who may have had excessive lead exposure but did not report symptoms.

*Under the Occupational Safety and Health Administration lead standard, BLLs exceeding 60 μg/dL on a single occasion or an average of 50 μg/dL on three separate occasions in a 6-month period require medical removal of the employee from the worksite (*1*).

Lead Poisoning — Continued

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Editorial Note: Acute lead poisoning in a worker whose job entails exposure to lead is considered an occupational sentinel health event (i.e., a condition that indicates both the failure to protect that worker from a preventable occupational illness and the existence of risk of similar illnesses for co-workers [*2*]). Adherence to appropriate workplace controls can prevent exposures to lead, while surveillance systems for identifying workers with elevated BLLs (ideally before they have become symptomatic) permit the targeting of intervention efforts (*3*). Treatment of lead poisoning always requires removal from exposure; chelation therapy (e.g., with intravenous EDTA) is generally reserved for symptomatic patients (*4*). State or local reporting and intervention systems can provide physicians access to consultation and expertise in diagnosis and treatment of clinical lead poisoning.

Environmental and biologic monitoring are usually necessary to evaluate the effectiveness of attempts to control exposure to lead. Consequently, the Occupational Safety and Health Administration (OSHA) lead standard for general industry specifies a permissible exposure level (PEL) of 50 $\mu g/m^3$ in air and mandates environmental and biologic monitoring under specified circumstances[†] (*1*). The construction industry, however, is exempt from the general industry standard; instead, it is covered by the OSHA standard for construction, which specifies a PEL of 200 $\mu g/m^3$ but contains no requirements for routine environmental or biologic monitoring (*5*).

When preventable exposures to lead result in poisoning(s), effective surveillance systems are essential in preventing additional cases. Laboratory-based reporting systems, which rely on routine mandatory reporting of elevated BLLs by laboratories, can trigger timely follow-up and intervention activities. Advantages of these surveillance systems are that 1) implementation is straightforward because the systems rely on existing requirements and medical practice; 2) use of a laboratory test improves the reliability of case identification; 3) more precise targeting of prevention activities is possible; and 4) the health departments managing these systems can readily serve as resources for information on prevention, follow-up, and appropriate treatment of persons with lead toxicity. In conjunction with CDC's National Institute for Occupational Safety and Health (NIOSH), 14 states[§] have implemented or are developing laboratory-based systems for reporting elevated BLLs (*6*). These systems have been effective in identifying worksites with excess lead exposure and co-workers at risk for lead poisoning (*3,6*).

For at least two reasons, workers with excessive exposure to lead may not be identified until they are diagnosed with symptomatic lead poisoning. First, surveillance systems depend on compliance with requirements for medical monitoring of lead-exposed workers; consequently, in worksites that fail to perform routine medical monitoring, exposures may not be detected. Second, excessive exposures to lead in the construction industry are frequently undetected because medical monitoring is not required. In these circumstances, case reports submitted by physicians to organized surveillance and prevention programs can trigger a public health response.

[†]When airborne lead concentrations exceed 30 $\mu g/m^3$ (averaged during an 8-hour workshift), employers must provide an industrial hygiene program and medical surveillance (including monitoring of BLLs).

[§]Alabama, California, Colorado, Connecticut, Illinois, Maryland, Massachusetts, Michigan, New Jersey, New York, Oregon, Texas, Utah, and Wisconsin.

Lead Poisoning — Continued

Despite underreporting by physicians, such reports may be the only means for timely recognition of and response to lead poisoning in workers and other sentinel health events.

The national health objectives for the year 2000 have targeted the elimination of occupational exposures to lead that result in BLLs ≥25 μg/dL (*7*). To meet this objective, NIOSH encourages states to establish lead surveillance systems and advocates a coordinated approach involving federal, state, industry, labor, and trade groups.

References

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Notice to Readers

Fetal Alcohol Syndrome Conference

"Fetal Alcohol Syndrome and Other Congenital Alcohol Disorders: A National Conference on Surveillance and Prevention" will be held in Atlanta on April 1–3, 1991. The conference is cosponsored by CDC's Center for Environmental Health and Injury Control (CEHIC); the Indian Health Service; the Alcohol, Drug Abuse, and Mental Health Administration's Office for Substance Abuse Prevention and National Institute on Alcohol Abuse and Alcoholism; the Association for Retarded Citizens; the March of Dimes Birth Defects Foundation; and the National Organization for Fetal Alcohol Syndrome.

Information about the conference is available from the Division of Birth Defects and Developmental Disabilities, CEHIC, CDC, Mailstop F-37, 1600 Clifton Road, NE, Atlanta, GA 30333; telephone (404) 488-4707 or FTS 236-4707.

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Epidemiologic Notes and Reports

Paralytic Shellfish Poisoning — Massachusetts and Alaska, 1990

Paralytic shellfish poisoning (PSP) is a foodborne illness caused by consumption of shellfish or broth from cooked shellfish that contain either concentrated saxitoxin, an alkaloid neurotoxin, or related compounds. This report summarizes outbreaks of PSP that occurred in Massachusetts and Alaska in June 1990.

Massachusetts

On June 6, 1990, the Massachusetts Department of Public Health (MDPH) was notified that, on June 5, foodborne illness had occurred in six fishermen aboard a fishing boat in the Georges Bank area off the Nantucket coast. Onset of illness occurred after the men had eaten blue mussels (*Mytilus edulis*) harvested in deep water about 115 miles from the island of Nantucket.

The six men (age range: 24–47 years) developed symptoms 1–2½ hours after consuming the shellfish (Table 1). Symptoms included numbness of mouth (five men), vomiting (four), paresthesia of extremities (four), numbness and tingling of tongue (two), numbness of face (two), numbness of throat (one), and periorbital edema (one). In all six men, lower back pain occurred approximately 24 hours after onset. The median duration of neurologic symptoms was 14 hours, and for lower back pain, 3.3 days. Approximately 10 hours after onset, when the fishermen presented to a local hospital emergency room, four were recovering; however, two, including one who had recovered from loss of consciousness, required hospitalization for 2–3 days.

The six fishermen had boiled the mussels for approximately 90 minutes before consuming them with baked fish, boiled rice, boiled potatoes, green salad, and other food items. They did not consume alcoholic beverages with the implicated meal.

Laboratory examination of the uneaten mussels detected saxitoxin concentrations of 24,400 μg/100 g in the raw mussels and 4280 μg/100 g in the cooked mussels (maximum safe level: 80 μg/100 mg). The difference in the levels of PSP toxin between raw and cooked mussels suggested that much of the saxitoxin had dissipated into the boiling water.