# MMR

MORBIDITY AND MORTALITY WEEKLY REPORT

- 157 Paralytic Shellfish Poisoning Massachusetts and Alaska, 1990
- 161 Cyanide Poisonings Associated with Over-the-Counter Medication — Washington State, 1991
- 169 Lead Poisoning among Bricklayers Washington State
- 171 Fetal Alcohol Syndrome Conference

### 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  $\mu g/100$  g in the raw mussels and 4280  $\mu g/100$  g in the cooked mussels (maximum safe level: 80  $\mu g/100$  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.

TABLE 1. Symptoms of paralytic shellfish poisoning from deep-water shellfish, by number of mussels consumed, onset time following ingestion, and duration of symptoms — Massachusetts, June 1990

				Duration (	hrs)
Patient no.	Symptoms	No. mussels consumed	Onset time (hrs)	Neurologic symptoms	Back pain
1	Vomiting; numbness and tingling around mouth (tongue); paresthesia of right arm; lower back pain.	4	21/2	12	72
2	Numbness of mouth (tongue and jaw); tingling of fingers and toes; lower back pain.	3–4	2	10	47
3	Vomiting; numbness of mouth (lips) and fingers; paresthesia of right arm (hand); lower back pain.	4–5	2	12	48
4	Numbness of face and arms; periorbital edema; paresthesias of legs; induced vomiting; lower back pain.	6	11/2	16	72
5*	Vomiting; numbness of throat; perioral numbness; tight feeling in chest; numbness of extremities (toes and fingers); lower back pain.	12	1	24	120
6*	Vomiting; severe paresthesias (arms and legs); numbness and tingling of mouth (jaws), arms, and extremities; loss of consciousness 2½ hours following onset; lower back pain.	18–24	1	48	120

<sup>\*</sup>Patient was hospitalized.

The implicated mussels had been harvested in an area of the Georges Bank where contamination of surf clams and sea scallops with saxitoxin had been detected through a deep-sea sampling survey conducted by the MDPH. The same area had been identified in a warning issued 2 weeks earlier by the MDPH and the National Marine Fisheries Service. The warning had been based on a report of a fisherman and his wife who had developed symptoms compatible with PSP after eating mussels obtained from that area. Because of the sampling survey and the first reported incident, the Georges Bank had been closed to harvesting of all shellfish except the adductor muscles of sea scallops shucked at sea. The closure notice had been sent to all appropriate Coast Guard stations and fishing vessels in the area; however, the six fishermen involved in the outbreak reported they had not received it.

#### Alaska

On June 26, 1990, a physician reported to the Alaska Department of Health and Social Services (ADHSS) that a Native Alaskan man had died after consuming shellfish collected from a beach on the Alaska Peninsula. On the evening of June 25, while aboard a fishing boat, the decedent had consumed 25–30 steamed butter clams and 2 teaspoons of butter clam broth. Within an hour, he complained of numbness

and tingling around his mouth, face, and fingers. Two hours later, he suffered a cardiopulmonary arrest; despite cardiopulmonary resuscitation efforts by emergency personnel, the patient died. Based on the symptoms reported, PSP was diagnosed. The patient's gastric contents contained 370  $\mu$ g/100 g of PSP toxin, and a sample of the butter clam broth from the meal contained 2650  $\mu$ g/100 g.

Two other crewmembers had also consumed butter clams. One developed numbness and tingling of the face and hands and dizziness approximately 1½ hours later and recovered uneventfully; the other had no symptoms. Four crewmembers from two other fishing boats also had shared the butter clams presumed to be the vehicle for illness; all four had symptoms consistent with PSP.

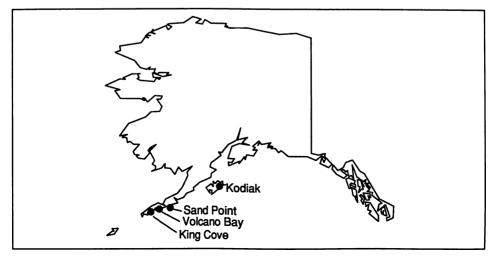
As a consequence of this episode, ADHSS identified three additional episodes in the region of the Alaska Peninsula and Kodiak Island during June 1990. Each episode involved consumption of shellfish collected from a different area (Figure 1). When aggregated, the four episodes constituted a PSP outbreak with 13 cases among 21 persons (attack rate: 62%) who had consumed the implicated shellfish.

The four episodes occurred during June 17–25. Onset of symptoms ranged from 0 to 2 hours (median: 1 hour) after consumption of shellfish. Duration of illness ranged from 1 to 24 hours (median: 7.5 hours). Seven (54%) persons sought medical care. Only the index patient died (case-fatality rate: 8%); the others recovered uneventfully.

Seven (54%) cases resulted from consumption of butter clams, and six (46%), from mussels. Shellfish were consumed raw, boiled, or steamed. Affected persons consumed three to 30 shellfish each (median: four shellfish).

Because shellfish from the four episodes were not available for testing for PSP toxin, samples were collected from the four sites where the shellfish had been harvested. Butter clam samples from Volcano Bay and King Cove contained 7750  $\mu$ g/100 g of PSP toxin, and mussel samples from Sand Point and Kodiak contained 1925–12,960  $\mu$ g/100 g.

FIGURE 1. Collection sites of shellfish implicated in an outbreak of paralytic shellfish poisoning — Alaska, June 1990



ADHSS, in conjunction with the Alaska Department of Environmental Conservation, issued a statewide press release warning of the risk for PSP for persons consuming shellfish collected from Alaskan beaches.

Reported by: K Sharifzadeh, DVM, N Ridley, MS, R Waskiewicz, MS, P Luongo, Div of Food and Drugs, GF Grady, MD, A DeMaria, MD, RJ Timperi, MPH, J Nassif, MS, Massachusetts Dept of Public Health. M Sugita, Kodiak Health Clinic; V Gehrman, King Cove Clinic; P Peterson, Sand Point Clinic; A Alexander, Cold Bay Clinic; R Barrett, K Ballentine, Alaska Dept of Environmental Conservation; JP Middaugh, MD, State Epidemiologist, Alaska Dept of Health and Social Svcs. I Somerset, MS, Food and Drug Administration. Enteric Diseases Br, Div of Bacterial and Mycotic Diseases, Center for Infectious Diseases; Div of Field Epidemiology, Epidemiology Program Office, CDC.

**Editorial Note:** The neurotoxins that cause PSP are among the most potent toxins known and can impair sensory, cerebellar, and motor functions. Saxitoxin is heat-stable and unaffected by standard cooking or steaming (1), is water-soluble, and can be concentrated in broth. Symptoms usually occur within 2 hours after ingestion of shellfish; high doses can lead to diaphragmatic paralysis, respiratory failure, and death (1,2).

The diagnosis of PSP is based on patient exposure history and clinical manifestations and on epidemiologic information. Predominant manifestations include paresthesia of the mouth and extremities, ataxia, dysphagia, and muscle paralysis (3–5); gastrointestinal symptoms are less common. Coma, total muscular paralysis, and respiratory arrest with death can occur. The prognosis is favorable for patients who survive beyond 12–18 hours (3). Because PSP has no specific treatment or antidote, treatment is supportive. Prompt evacuation of stomach contents may help by removing the remaining toxin-containing shellfish.

During 1973–1987, state health departments reported 19 PSP outbreaks (mean size: eight persons) to CDC's Foodborne Disease Outbreak Surveillance System. Outbreaks were caused by consumption of mussels, clams, oysters, scallops, and cockles. Outbreaks on the west coast have been reported from May through October, and on the east coast from August through October.

Most cases of PSP occur in individuals or small groups who gather shellfish for personal consumption. Although PSP has traditionally been considered a risk only in shellfish harvested from cold water, the incidence in tropical areas may be increasing: outbreaks have been reported recently from Central and South America, Asia, and the Pacific region (2,6).

The PSP-associated death in Alaska was the first in that state in >14 years. From 1976 through 1989, 42 PSP outbreaks (accounting for 94 cases) were documented in Alaska. Butter clams were implicated in 23 (55%) of the outbreaks. Other shellfish implicated in Alaskan outbreaks included mussels, cockles, steamer clams, sea snails, and razor clams. Thirty-one (74%) of the 42 outbreaks occurred during May–July.

Shellfish can become toxic when toxin-producing dinoflagellates create massive algal blooms known as "red tides." However, shellfish can become toxic even in the absence of such blooms; detoxification may require a month or more in clean waters.

To prevent outbreaks of PSP and other shellfish intoxications, samples of susceptible mollusks are periodically collected in the coastal states and tested for toxin by mouse bioassay. When toxin levels exceed 80  $\mu$ g/100 g, affected growing areas are quarantined, and sale of shellfish is prohibited. Warnings posted in shellfish-growing areas and on beaches and placed in the news media can alert the public to the hazard.

#### References

- 1. Hughes JM, Merson MH. Fish and shellfish poisoning. N Engl J Med 1976;295:1117-20.
- Rodrigue DC, Etzel RA, Hall S, et al. Lethal paralytic shellfish poisoning in Guatemala. Am J Trop Med Hyg 1990;42:267–71.
- 3. Eastaugh J, Shepherd S. Infectious and toxic syndromes from fish and shellfish consumption. Arch Intern Med 1989:149:1735–40.
- Sakamoto Y, Lockey RF, Krzanowski JJ. Shellfish and fish poisoning related to the toxic dinoflagellates. South Med J 1987;80:866–72.
- Wallace J. Disorders caused by venoms, bites, and stings. In: Isselbacher KJ, Adams RD, Braunwald E, Petersdorf RG, Wilson JD, eds. Harrison's principles of internal medicine. 9th ed. New York: McGraw-Hill Book Company, 1980:927.
- Maclean J, White A. Toxic dinoflagellate blooms in Asia: a growing concern. In: Anderson DM, White AW, Baden DG, eds. Toxic dinoflagellates. New York: Elsevier, 1985:517–30.

# Cyanide Poisonings Associated with Over-the-Counter Medication – Washington State, 1991

On February 2, 9, and 17, 1991, three persons in western Washington state, who had taken SUDAFED®\* 12-Hour capsules<sup>†</sup> (manufactured by Burroughs Wellcome Company) for nasal congestion, had onset of acute cyanide poisoning; two died. This report updates an ongoing investigation by the Food and Drug Administration (FDA) and other agencies.

Patient 1. On February 2, a previously healthy 28-year-old woman ingested one capsule of SUDAFED® 12-Hour purchased in Olympia. Within 1 minute, she collapsed. Paramedics who were called found her comatose. She was intubated and transported to a local hospital; on admission, she was comatose with a profound metabolic acidosis (pH: 7.0; lactate: 19 mmol/L; anion gap: 27 mmol/L). Following intensive supportive care for metabolic acidosis, hypotensive shock, and bradyarrhythmia, her acidosis resolved, her blood pressure stabilized, and she became conscious.

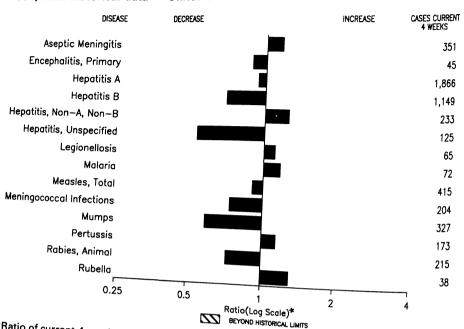
Based on the patient's elevated lactate level and on exclusion of other causes for her collapse, her physician suspected cyanide poisoning and submitted blood drawn shortly after admission for toxicologic analysis. It was positive for cyanide (6.14 mg/L) and pseudoephedrine. Suspecting poisoning, the physician obtained the patient's medications and observed that the lot number on the SUDAFED® box differed from the lot number on the blister pack.

Patient 2. On February 9, a 40-year-old woman with a past history of hypertension and recent sinus problems was found unconscious on the floor of her bathroom shortly after she had entered the room. Paramedics detected a pulse and transported her to a local hospital; her condition deteriorated, and she died on February 11. An initial drug screen performed on hospital admission was positive for pseudo-ephedrine. Autopsy findings included cerebral edema with herniation, focal cerebellar hemorrhage, and pulmonary edema. Because no cause of death was determined and the autopsy findings were unexplained, the Tacoma County medical (Continued on page 167)

<sup>\*</sup>Use of trade names is for identification only and does not imply endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

<sup>&</sup>lt;sup>†</sup>A nonprescription medication for nasal decongestion; each capsule contains 120 mg of pseudoephedrine hypochloride.

FIGURE I. Notifiable disease reports, comparison of 4-week totals ending March 9, 1991, with historical data — United States



<sup>\*</sup>Ratio of current 4-week total to the mean of 15 4-week totals (from previous, comparable, and based on the mean and two standard deviations of these 4-week totals.

TABLE I. Summary — cases of specified notifiable diseases, United States, cumulative, week ending March 9, 1991 (10th Week)

	Cum. 1991		Cum. 1991
AIDS Anthrax Botulism: Foodborne Infant Other Brucellosis Cholera Congenital rubella syndrome Diphtheria Encephalitis, post-infectious Gonorrhea Haemophilus influenzae (invasive disease) Hansen Disease Leptospirosis Lyme Disease	7,344 9 9 - 12 3 1 7 104,660 541 15 17 343	Measles: imported indigenous Plague Poliomyelitis, Paralytic* Poittacosis Rabies, human Syphilis, primary & secondary Syphilis, congenital, age < 1 year Tetanus Toxic shock syndrome Trichinosis Tuberculosis Tularemia Typhoid fever Typhus fever, tickborne (RMSF)	22 855 12 7,958 - 71 71 3,244 12 57

<sup>\*</sup>No cases of suspected poliomyelitis have been reported in 1991; none of the 6 suspected cases in 1990 have been confirmed to date. Five of the 13 suspected cases in 1989 were confirmed and all were vaccine associated.

TABLE II. Cases of selected notifiable diseases, United States, weeks ending March 9, 1991, and March 10, 1990 (10th Week)

		Aseptic	Encephalitis				Н	epatitis	(Viral), by	type			
Reporting Area	AIDS	Menin- gitis	Primary	Post-in- fectious	Gono	rrhea	Α	В	NA,NB	Unspeci- fied	Legionel- losis	Lyme Disease	
	Cum. 1991	Cum. 1991	Cum. 1991	Cum. 1991	Cum. 1991	Cum. 1990	Cum. 1991	Cum. 1991	Cum. 1991	Cum. 1991	Cum. 1991	Cum. 1991	
UNITED STATES	7,344	919	96	7	104,660	132,010	4,595	2,695	597	258	194	343	
NEW ENGLAND	326	44	6	-	3,330	3,948	109	178	32	9	21	24	
Maine N.H.	15 10	2 2	3	-	20 56	51 41	4 9	1 11	1 2	-	1	2	
Vt.	6	2	-	-	12	14	5	1	1	-	-	1	
Mass. R.I.	167 13	16 21	1	-	1,289 216	1,470 198	61 15	147 7	28	7 2	20	15 6	
Conn.	115	- i	2	-	1,737	2,174	15	11	-	-	-	-	
MID. ATLANTIC	2,058	131	5	4	10,637	15,658	356	232	41	8	55	234	
Upstate N.Y. N.Y. City	309 1,035	57 9	4	3	2,117 1,918	2,563 8,188	222 25	113 6	31	3	20 3	224	
N.J. Pa.	472 242	-	:	:	2,264	2,737	40	47	5		6	10	
E.N. CENTRAL	593	65 157	1	1	4,338	2,170	69	66	5	5	26	-	
Ohio	102	157 55	21 6	2 1	19,606 5,456	26,462 7,889	414 104	319 75	150 39	10 4	31 17	14 6	
Ind. III.	48 306	21 19	5 3	1	2,233 6,215	2,555 8,071	93 68	53 10	1 2		3	-	
Mich.	91	57	7		5,135	6,267	68	112	24	6	10	8	
Wis.	46	5	-	-	567	1,680	81	69	84	-	1	•	
W.N. CENTRAL Minn.	219 44	62 11	8 5	-	5,597 586	7,188 841	728 77	94 7	43 5	4 1	9 2	3	
lowa	23	18	-	-	409	548	15	4	2	-	-	3	
Mo. N. Dak.	123	19	-	-	3,494	4,113 37	138 10	73	36	2 1	4	-	
S. Dak. Nebr.	-	6	3	- '	109	45	388	1	-	-	1	-	
Kans.	10 19	7 1	-		383 616	306 1,298	81 19	8 1	-		2	-	
S. ATLANTIC	1,667	209	18	1	32,557	38,011	296	662	75	50	24	24	
Del. Md.	12 165	4 24	4	-	451 3,245	465 4,237	4 72	14 86	1 17	4	7	6 9	
D.C.	132	8	-		2,189	1,738	19	20	-	1	-	-	
Va. W. Va.	126 8	44 2	3	-	3,012 248	3,745 249	40 5	52 17	3	35 3	2	5	
N.C. S.C.	71	31	7	-	6,190	6,429	55	153	31	-	6	4	
Ga.	78 216	7 16	2	1	2,548 8,341	3,267 8,757	10 25	156 76	15 3	2	4 1	-	
Fla.	859	73	2	•	6,333	9,124	66	88	5	5	4	-	
E.S. CENTRAL Ky.	176 33	70	5	-	8,885	10,718	45	199	68	2	14	15	
Tenn.	54	18 16	2 2	-	948 3.028	1,286 3,208	8 25	45 130	1 64	2	7 5	11 2	
Ala. Miss.	61 28	24 12	1	-	2,447	3,845	11 1	24	3	-	2	2	
W.S. CENTRAL	685	54	8	-	2,462	2,379	560	221	- 16	35	6	•	
Ark.	28	25	1	-	11,725 1,501	12,989 1,732	84	12	16	35 2	-	1 -	
La. Okla.	90 27	5 1	1 3	-	2,490 1,214	2,398 1,197	30 86	45 54	1 12	1 6	2 4	1	
Tex.	540	23	3	-	6,520	7,662	360	110	2	26	-	-	
MOUNTAIN Mont.	185	39	8	-	1,960	2,981	803	185	29	55	22	1	
Idaho	4 3	2	-	-	17 25	24 18	32 13	21 18	1	2	3	-	
Wyo. Colo.	3		-	-	24	32	56	3	-	-	-	1	
N. Mex.	80 11	12 5	1	-	379 222	842 209	60 253	32 23	8 2	6 18	3 1	-	
Ariz. Utah	37	13	7	•	821	1,243	277	43	4	24	7	-	
Nev.	11 36	2 5	-	-	70 402	85 528	61 51	6 39	4 10	5	4 4	-	
PACIFIC	1,435	153	17		10,363	14,055	1,284	605	143	85	12	27	
Wash. Oreg.	70 28		-	-	745	1,374	106	94	24	6	1		
Calif.	1,288	137	17	-	370 8,925	503 11,852	80 1,064	57 438	22 90	2 76	10	27	
Alaska Hawaii	3 46	4 12	-	-	160 163	231	26 8	5	6	1	1	-	
Guam		12	•	-	163	95 44	6	11	1	-	1	-	
P.R. V.I.	349	30	-	1	94	264	15	61	12	3	:	-	
Amer. Samoa	1	-	•	-	72	99 33	-	2	-	-		-	
C.N.M.I.	-	-		-		40	-	-	-			-	

TABLE II. (Cont'd.) Cases of selected notifiable diseases, United States, weeks ending March 9, 1991, and March 10, 1990 (10th Week)

	ľ	Γ				Menin-	T T T T T T T T T T T T T T T T T T T								
Reporting Area	Malaria	India	enous	sles (Rubeola)		gococcal	Mumps		Pertussis			Rubella			
Neporting Area	Cum. 1991	1991	Cum. 1991	1991	Cum. 1991	Cum. 1990	Cum. 1991	1991	Cum. 1991	1991	Cum. 1991	Cum. 1990	1991	Cum. 1991	Cum. 1990
UNITED STATES	161	227	855	2	22	3,335	413	116	657	43	424	563	20	77	80
NEW ENGLAND	11	-	1	-	2	86	43	2	9	6	35	76	1	1	1
Maine N.H.	i	-	-	-	-	26	4	-	-	1	5	1	-	-	-
Vt.	i		-	-		7 1	6 6	2	2	2	11 1	7 2	1	1 -	- :
Mass. R.I.	6	•	-	•	-	-	20	-	-	3	18	61	-	-	:
Conn.	2 1	-	1		2	16 36	7	:	2 5	:	-	- 5	-	-	1
MID. ATLANTIC Upstate N.Y.	15 4	95	342	-	:	300 174	43 24	4	51 25		50 24	145 117	6 6	9 6	2
N.Y. City	3	14	60	-	-	23	-	-	-		-	-	-	-	-
N.J. Pa.	5 3	81	31 251	:	-	14 89	7 12		2 24	-	1	9	•	3	1
E.N. CENTRAL	10	-	7		2	1,562	58			-	25	19	-		6
Ohio	3	-	<i>'</i> -	-	1	136	21	2	64	3	84 34	154 26	:	5	-
Ind. III.	1	-	-	-	-	48	9	-	3	-	16	31	-	1	-
Mich.	5	-	7	-	-	678 238	10 14	2	40 18	3	14 16	50 15	-	2	6
Wis.	-	-	-	-	1	462	4	-	3	-	4	32		-	-
W.N. CENTRAL	2	1	1	1	1	96	15	2	29	2	32	18	1	3	-
Minn. Iowa	1	1	1	1†	1	28 21	4	1	2 6	-	11	1	1	2	•
Mo.	1	-	-	-	-	47	5		2		4 12	1 12		1	-
N. Dak. S. Dak.	-	-	-	-	-	-	:	-	-	-	-	1	-	-	-
Nebr.	-	-	-	-		-	2 2	1	2	2	1 4	1 1	-		
Kans.	-	-	-	-	-	-	ī	-	17	-	-	i	-	-	-
S. ATLANTIC Del.	44	19	38	1	6	201	76	41	213	5	27	50	4	7	7
Md.	13	2	4 2	- 1§	1	4 22	10	4	-	-	-	2		7	-
D.C.	3	-	-			1	-	4	59 3	2	3	18 1	4	,	-
Va. W. Va.	7 1	1	1	-	-	11	8	2	14	1	3	4	-	-	-
N.C.	1	-	-	-		6 3	3 22	17	5 73	-	6 9	5	-	•	:
S.C. Ga.	4	-	12	-	-	1	7	14	39		-	6 3	- 1	-	-
Fla.	11	16	19	-	- 5	1 152	15 11	4	5		3	7	-	-	7
E.S. CENTRAL	1			_	٠	25			15	2	3	4	•	-	′
Ky.	-	-	-	-		25	34 15	3	21	2	17	15	•	-	:
Tenn. Ala.	1	-	-	-	-	15	7	3	11	1	10	4		-	-
Miss.	:	-	-	-	:	10	12	-	2 8	1	7	10	-	-	-
W.S. CENTRAL	10	_	-	-	5	150	25	_			-	1			
Ark. La.	1	-	-	-	5	-	6	18 2	89 8	3	12	5	1	1 1	-
Okla.	2 1	-	-	-	-	-	8	-	9	1	7	1		-	-
Tex.	6	-	-		:	39 111	2 9	16	1 71	2	5	4	-	-	
MOUNTAIN	8	24	94		4	81	18	1	28	9	-		•	•	
Mont. Idaho	-	-	-	-		-	3		- 28	9	69	51		1	-
Wyo.	-	-		-	1	2	3	-	2	8	17	2	-	-	-
Colo. N. Mex.	3	-		-	1	9	3	1	6	•	4 18	36	-	-	:
Ariz.	1 4	1 23	62 25	-	2	13	4	Ň	N		12	2	-		-
Utah	-		-	-	:	45	4		17 3	1	8	6	-	-	-
Nev.	-	-	7	-	•	12	1		-	:	10	2 3	-	1	Ĭ.
PACIFIC Wash.	60	88	372	-	2	834	101	43	153	13	98	49	7	50	64
Oreg.	5 1	1	1	- :	-	13	8	31	39	4	17	12	<i>'</i> -	-	-
Calif.	53	87	369	-	2	72 716	10 80	N 12	N 105	8	16	6	-	40	60
Alaska Hawaii	1	-	2	-	-	31	3	-	4	1 -	47 4	27	7	49	-
Guam		U	•		•	2	•	-	5	-	14	4	-	1	4
P.R.	-	-	-	U	1	44	3	U 2	-	U	-	-	U	-	-
V.I. Amer. Samoa	-	U	-	U		-	-	U	3 2	Ū	4	4	Ū	•	
C.N.M.I.	-	U	-	U	-	•	-	U	-	U	-	-	Ü	:	-
			•	J	-	•	-	U	-	U	-		ŭ	-	-

<sup>\*</sup>For measles only, imported cases includes both out-of-state and international importations.

N: Not notifiable U: Unavailable †International \*Out-of-state

TABLE II. (Cont'd.) Cases of selected notifiable diseases, United States, weeks ending March 9, 1991, and March 10, 1990 (10th Week)

Reporting Area	Sy (Primary 8	philis k Secondary)	Toxic- shock Syndrome	Tubero	culosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies Anima Cum. 1991	
	Cum. 1991	Cum. 1990	Cum. 1991	Cum. 1991	Cum. 1990	Cum. 1991	Cum. 1991	Cum. 1991		
UNITED STATES	7,958	8,511	71	3,244	3,619	12	57	13	723	
NEW ENGLAND	220	362	5	74	66	-	6	2	1	
Maine N.H.	1	3 26	3 1	-	1	-	-	-	•	
Vt.	1	-	-	-	2		-	-	1 -	
Mass. R.I.	115 11	124 1	1	29	26	-	6	2	•	
Conn.	92	208	-	16 29	15 22	-	-	-		
MID. ATLANTIC	1,384	1,515	13	777	938		8	-	260	
Upstate N.Y. N.Y. City	103	102	7	21	99	-	2	-	79	
N.J.	624 249	1,039 325		553 133	602 114	-	2 4		117	
Pa.	408	49	6	70	123	-	-	-	64	
E.N. CENTRAL Ohio	815 101	571 94	13	399	358 48	1	7	-	8	
Ind.	20	94 6	9	68 16	48 23	-	1 -	-	1	
III. Mich.	384	227	1	229	176	:	-	-	1	
Wis.	210 100	161 83	3	62 24	97 14	1	5 1	•	2 4	
W.N. CENTRAL	143	69	16	78	82	1	1	-	103	
Minn.	15	20	7	10	16	-	i	-	35	
Iowa Mo.	15 84	6 33	4 4	17 29	7 35	1	-	:	20 2	
N. Dak.	-	1		2	3	-	-	-	9	
S. Dak. Nebr.	1 1	2	1	7 3	4 7	-	-	-	29 2	
Kans.	27	7		10	10	-	-	-	6	
S. ATLANTIC	2,501	2,989	4	483	567	-	9	8	196	
Del. Md.	21 235	43 240	1	7	10	-	- 5	1	33 70	
D.C.	138	153		34 35	54 14	-	-	!	2	
Va. W. Va.	199 4	143	1	49	40	-	1	-	34	
N.C.	368	2 337	2	18 70	9 73	-	1	6	15	
S.C. Ga.	313 615	178	-	53	92	-	-	-	10	
Fla.	608	767 1,126	-	100 117	86 189	-	2	1 -	29 3	
E.S. CENTRAL	794	630	1	204	262	2		2	12	
Ky. Tenn.	13	16	-	55	75	-	-	1	4	
Ala.	332 233	176 223	ī	- 81	63 83	2	-	1	8	
Miss.	216	215	-	68	41	-	•	-	-	
W.S. CENTRAL Ark,	1,310	1,342	2	342	453	4	-	1	62	
La.	69 436	102 410	1	32 66	47 94	3	-	•	4 3	
Okla. Tex.	30	46	1	15	35	1	-	1	20	
MOUNTAIN	775	784	•	229	277	-	-	-	35	
Mont.	120 1	164	8	75	67 4	3 2	3	-	7	
Idaho Wyo.	3	4	-	1	1	-		-	1	
Colo.	1 12	12	-	1	1	1	-	•	1	
N. Mex. Ariz.	6	11	3	6	16	-	-	-	i	
Utah	79 2	111 1	2 3	47 13	30	-	2	-	1	
Nev.	16	25	-	7	15	-	1	-	-	
PACIFIC Wash.	671	869	9	812	826	1	23	-	74	
Oreg.	33 21	84 20	-	36	53	1	-	-	-	
Calif. Alaska	615	752	9	13 720	18 711	-	1 21	-	1 73	
Hawaii	2	4 9	•	4	17	-	-	-		
Guam	_	3	-	39	27	-	1	-	-	
P.R. V.I.	65	105	-	15	9 29		-	-	6	
Amer. Samoa	8	-	-	1	1	-	-	-	-	
C.N.M.I.	-	-	-	-	5 7	-	-	-		

TABLE III. Deaths in 121 U.S. cities,\* week ending March 9, 1991 (10th Week)

	,						199	i (10th Week)	<b></b>							
Bonostina Asso							P&I**	P&I**	All Causes, By Age (Years)							
Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	Total	Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	Total	
NEW ENGLAND	708	509	117	52	14	16	62	S. ATLANTIC	1,497	943			41	51	91	
Boston, Mass. Bridgeport, Conn.	159 54	96 39	34 9	18 2	6 2	5 2	17 4	Atlanta, Ga.	193	116			3	7 5	5 39	
Cambridge, Mass.	29	25	4	- 2		-	1	Baltimore, Md. Charlotte, N.C.	296 106	196 70		27 9	11 1	6	39 7	
Fall River, Mass.	26	20	5	1		-		Jacksonville, Fla.	110	63			8	7	6	
Hartford, Conn.	79	56	11	9	1	2	5	Miami, Fla.	106	60		10	5	-	•	
Lowell, Mass. Lynn, Mass.	30 15	23 11	5 4	1	1	-	2	Norfolk, Va.	66	41				4	4	
New Bedford, Mass.	25	21	3	1	:	:	2	Richmond, Va.	69	44			1	4	3 2	
New Haven, Conn.	63	45	10	5	2	1	4	Savannah, Ga. St. Petersburg, Fla.	38 85	29 66			2		5	
Providence, R.I.	61	46	11	2		ż	8	Tampa, Fla.	166	118				2	14	
Somerville, Mass.	10 50	10	-	-	•	-	1	Washington, D.C.	221	106	54	38		16	6	
Springfield, Mass. Waterbury, Conn.	30	34 25	6 3	6 1	1	3	4	Wilmington, Del.	41	34	- 5	2	-	-	-	
Worcester, Mass.	77	58	12	6	1	1	2 10	E.S. CENTRAL	777	487	152		33	34	50	
	2.780	1.836	550	_	-			Birmingham, Ala.	132	75			7	6	3	
Albany, N.Y.	54	41	10	282	62 2	48 1	170 5	Chattanooga, Tenn.	54	37				1	4	
Allentown, Pa.	17	13	2	2	-	'.	1	Knoxville, Tenn. Louisville, Ky.	68 55	51 35				1	3	
Buffalo, N.Y.	149	110	27	8	2	2	10	Memphis, Tenn.	234	151				14	12	
Camden, N.J. Elizabeth, N.J.	31	14	11	6	-	-	-	Mobile, Ala.	27	15		3	2	3	2	
Erie, Pa.†	26 39	19 25	2 10	5 4	-	-	3	Montgomery, Ala.	38	23				-	5 18	
Jersey City, N.J.	51	35	13	3	•	•	6	Nashville, Tenn.	169	100	28			9		
New York City, N.Y.	1,464	926	281	187	42	28	66	W.S. CENTRAL	1,571	969			51	56	81 5	
Newark, N.J.	80	37	24	14	2	2	1	Austin, Tex.	74	50			2	1	2	
Paterson, N.J.	24	18	5	1		-	-	Baton Rouge, La.	33	23 30		4	1 2	:		
Philadelphia, Pa. Pittsburgh, Pa.†	398 58	262 43	88 11	30	10	7	27	Corpus Christi, Tex. Dallas, Tex.	44 233	138		31	8	11	7	
Reading, Pa.	48	38	6	2		2 1	4 9	El Paso, Tex.	90	58			1	4	4	
Rochester, N.Y.	113	80	26	6	1		14	Ft. Worth, Tex.	109	72	15	15		4	2 35	
Schenectady, N.Y.	25	18	5	1	1	-	1	Houston, Tex.	372	190			16	15 7	4	
Scranton, Pa.† Syracuse, N.Y.	29 79	25	4	:	-	-	1	Little Rock, Ark.	71	46 115				3		
Trenton, N.J.	79 42	59 26	11 10	4	1	4	6	New Orleans, La. San Antonio, Tex.	168 200	139			7	2	6	
Utica, N.Y.	22	20	2	4	1	1	11 3	Shreveport, La.	86	55		2	2	4	11	
Yonkers, N.Y.	31	27	2	2			2	Tulsa, Okla.	91	53	23	7	3	5	5	
E.N. CENTRAL	2,255	1,433	406	239	116	61	132	MOUNTAIN	897	600	163	86	24	24	57	
Akron, Ohio	54	41	9	3	110	1	132	Albuquerque, N.M.	113	80	17	10		1	9 5	
Canton, Ohio	43	36	5	2	-	-	5	Colo. Springs, Colo.	87	51				2	15	
Chicago, III.	447	174	99	92	69	13	14	Denver, Colo.	108	70		12 13		4	9	
Cincinnati, Ohio Cleveland, Ohio	154 149	105 88	30 36	10	4	5 5	13	Las Vegas, Nev. Ogden, Utah	163 27	103 20			1	-	2	
Columbus, Ohio	239	170	38	17 22	3 5	5 4	2 12	Phoenix, Ariz.	161	100	-		3	6	2	
Dayton, Ohio	90	61	17	- 6	1	5	4	Pueblo, Colo.	28	23	3	2	-	6	2	
Detroit, Mich.	247	147	49	31	10	10	9	Salt Lake City, Utah	53	33					10	
Evansville, Ind. Fort Wayne, Ind.	51	36	12	-	2	1	3	Tucson, Ariz.	157	120					140	
Gary, Ind.	51 23	39 9	6 6	1 8	5	-	6	PACIFIC	1,998	1,345		184	53 1	56 1	2	
Grand Rapids, Mich.	51	40	8	۰	1	2	3	Berkeley, Calif. Fresno, Calif.	19 104	16 70		3		5	10	
Indianapolis, Ind.	145	101	21	16	ż	5	9	Glendale, Calif.	31	24			2	1	1	
Madison, Wis.	37	25	5	4	1	2	ě	Honolulu, Hawaii	94	64			2	1	12 9	
Milwaukee, Wis. Peoria, III.	147	119	21	4	3	-	16	Long Beach, Calif.	75	43		6		7 8 U 4	13	
Rockford, III.	59 60	44 44		8	2	1	2	Los Angeles, Calif.	524	355		54	15 U	11	Ŭ	
South Bend, Ind.	49	39	8	2 1	3	2	8	Oakland, Calif.§	ŭ	U		U	2	4	2	
Toledo, Ohio	104	72		8	3	3	6	Pasadena, Calif. Portland, Oreg.	29 126	15 86				2	8	
Youngstown, Ohio	55	43		4	1	2	2	Sacramento, Calif.	160	107			4	4	27	
W.N. CENTRAL	904	653	167	52	16	16	71	San Diego, Calif.	170	102	33	21	6	7 4	16 4	
Des Moines, Iowa	111	77	24	6	3	1	13	San Francisco, Calif.	163	93				3	21	
Duluth, Minn.	32	26	5			i	2	San Jose, Calif.	180	134				5	4	
Kansas City, Kans.	28	19			-	1	2	Seattle, Wash. Spokane, Wash.	161 59	107 46		18 2		3	2	
Kansas City, Mo. Lincoln, Nebr.	123 41	91 32	16 8	10	1	5	5	Tacoma, Wash.	103	83				1	9	
Minneapolis, Minn.	241	32 166		15	6	2	3 24		13,387 <sup>†</sup>					362	854	
Omaha, Nebr.	81	62		2	2	1	6	I TOTAL	13,387	0,775	2,000	1,200	-410			
St. Louis, Mo.	121	84	23	8	2	4	3									
St. Paul, Minn.	70	53	11	4	1	1	9	1								
Wichita, Kans.	56	43	6	6	1	-	4	I								_

<sup>\*</sup>Mortality data in this table are voluntarily reported from 121 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

<sup>\*\*</sup>Pneumonia and influenza.

\*\*Pneumonia and influenza.

\*\*Because of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week.

\*\*Complete counts will be available in 4 to 6 weeks.

<sup>††</sup>Total includes unknown ages.

<sup>§</sup>Report for this week is unavailable (U).

Cyanide Poisonings - Continued

examiner requested additional drug testing on the blood obtained at the time of hospital admission. The analysis detected cyanide (6.49 mg/L) and ephedrine/pseudoephedrine (0.52 mg/L) but was negative for alcohol and other drugs.

Patient 3. On February 17, a 44-year-old man with a 2-week history of sinus problems collapsed in his home within 2 minutes of ingesting one SUDAFED® 12-Hour capsule from a box purchased that day in Lacey, Washington. Paramedics transported him to a local hospital, where he was declared brain dead, and he was maintained on a respirator pending removal of his organs for transplantation. On February 19, his liver, kidneys, heart, and one lung were transplanted into five separate recipients. Although all admission blood samples had been discarded, a sample taken from the patient 28 hours after his collapse was positive for cyanide (0.34 mg/L). A blood sample taken at the time of transplantation on February 19 contained no measurable amount of cyanide (<0.1 mg/L). One of the transplant recipients died 8 days after transplantation from causes unrelated to cyanide poisoning.

On March 4, the Washington State Toxicology Laboratory retested blood samples from approximately 40 medical examiner and coroner (ME/C) cases from Pierce, King, and Snohomish counties for January and February 1991. Cases included those in which the decedent had exhibited any symptoms generally consistent with cyanide poisoning. All these samples were negative for cyanide.

On Saturday, March 2, Burroughs Wellcome Company recalled all boxes of SUDAFED® 12-Hour in the United States and notified the public of the potential cyanide contamination of those capsules.

Reported by: J Howard, MD, Pierce County Medical Examiner's Office, Tacoma; TH Pouw, MD, Olympia; J Arnold, Thurston County Coroner's Office, Olympia; B Logan, Washington State Toxicology Laboratory, Seattle; JM Kobayashi, MD, State Epidemiologist, Washington Dept of Health. J Davis, Region X, Food and Drug Administration. Surveillance and Programs Br, Div of Environmental Hazards and Health Effects, Center for Environmental Health and Injury Control, CDC.

Editorial Note: Cyanide is one of the most potent and rapidly acting poisons and can produce death within a few minutes of ingestion (1-4). Cyanide is toxic because it binds to and inactivates a mitochondrial enzyme, cytochrome oxidase, which is important in cellular respiration. The brain is the organ most sensitive to the toxic effects of cyanide, and death usually results from damage to neurons in the brainstem, with consequent respiratory arrest. A blood concentration >0.2 mg/L is toxic (2); fatal levels usually exceed 1 mg/L (2) but have ranged from 1.1 to 53.1 mg/L and from 0.4 to 230 mg/L in different reports (4). The lethal dose in adults is 200–300 mg of potassium cyanide (4,5).

Specific treatment for cyanide poisoning is directed at reducing the amount of free cyanide that can bind to cytochrome oxidase and at releasing cyanide already bound. This is accomplished by administration of sodium nitrite (300 mg for an adult) to produce methemoglobin, which competes with cytochrome oxidase for free cyanide, and administration of thiosulfate, which enhances the biotransformation of cyanide to thiosulfate, a less toxic compound (1–3). The cyanide concentrations in patients 1 and 2 were within the lethal range.

Before the poisonings in Washington, nine deaths were known to have resulted from deliberate tampering with over-the-counter medications; all involved cyanide. In 1982, seven persons died in metropolitan Chicago after ingesting acetaminophen capsules that contained cyanide (6). In 1986, two persons died in Seattle after

Cyanide Poisonings - Continued

ingesting cyanide-containing analgesic capsules (7). As a consequence of these incidents, many over-the-counter medications were repackaged to make them "tamper-resistant."

In the three poisonings reported here, all victims ingested SUDAFED® 12-Hour capsules, which are packaged with four tamper-resistant features: 1) a two-part plastic capsule that is sealed with a band; 2) encasement of the capsules in a blister pack consisting of a clear plastic film with a foil backing, which must be broken to use the capsule; 3) enclosure of the blister pack in a box sealed with a safety tab, which must be broken to open the box; and 4) identification of both the box and blister pack with code numbers, which should be the same. In three of the four packages in which cyanide has been found, the physical tamper-resistant features had been compromised; in all four, the code numbers on the box and blister pack did not match. The numbers on the blister packs, however, were the same, suggesting that someone had obtained a group of boxes from the same lot, removed the blister packs, substituted "look-alike" capsules containing cyanide in some of the compartments, and placed these altered blister packs in different boxes.

These cyanide poisonings suggest the need to reevaluate whether the current safeguards against tampering are adequate. In these and previous cyanide poisoning deaths, capsules have been the target for tampering. Some manufacturers have substituted larger tablets for capsules to make undetectable tampering more difficult. Consumers should carefully examine medication—especially nonprescription medication in capsule form—and its packaging for signs of tampering. Tampered medication should not be used and should be immediately provided to the FDA for investigation.

The rapid collection and dissemination of information on these cyanide poisonings relied on information-sharing among ME/Cs, public health agencies, and other organizations. In most jurisdictions, ME/Cs are not organizationally affiliated with public health departments (8); however, as demonstrated in this report, information from ME/Cs can complement investigations by public health officials.

#### References

- Smith RP. Toxic responses of the blood. In: Klaassen CD, Amdur MO, Doull J, eds. Toxicology. 3rd ed. New York: MacMillan, 1986:240–1.
- Rumack BH, Lovejoy FH. Clinical toxicology. In: Klaassen CD, Amdur MO, Doull J, eds. Toxicology. 3rd ed. New York: MacMillan, 1986:888–9.
- Klaassen CD. Nonmetallic environmental toxicants: air pollutants, solvents and vapors, and pesticides. In: Gilman AG, Goodman LS, Gilman A, eds. The pharmacological basis of therapeutics. 6th ed. New York: MacMillan, 1980:1651–2.
- Houts M, Baaselt RC, Cravey RH. Toxico-legal analysis of chemicals/drugs. Vol 3. Courtroom toxicology. New York: Matthew Bender, 1984:cyan-1-cyan-20.
- Agency for Toxic Substances and Disease Registry. Toxicological profile for cyanide. Atlanta: US Department of Health and Human Services, Public Health Service, 1989:15–16.
- Wolnik KA, Fricke FL, Bonnin E, Gaston CM, Satzger RD. The Tylenol tampering incident tracing the source. Anal Chem 1984;56:466A–8A, 470A, 474A.
- 7. Varnell RM, Stimac GK, Fligner CL. CT diagnosis of toxic brain injury in cyanide poisoning: considerations for forensic medicine. Am J Neuroradiol 1987;8:1063–6.
- Combs DL, Parrish RG, Ing R. Death investigation in the United States and Canada, 1990.
   Atlanta: US Department of Health and Human Services, Public Health Service, CDC, 1990.

## 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  $\mu$ g/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  $\mu$ g/dL and 92  $\mu$ g/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:  $88~\mu g/dL$ ), 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  $\mu g/dL$ , 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.

<sup>\*</sup>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

Reported by: H Stockbridge, MD, W Daniell, MD, Univ of Washington Occupational Medicine Program, Seattle, Washington. Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.

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/m³ 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/m³ 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<sup>§</sup> 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.

<sup>&</sup>lt;sup>†</sup>When airborne lead concentrations exceed 30 μg/m³ (averaged during an 8-hour workshift), employers must provide an industrial hygiene program and medical surveillance (including monitoring of BLLs).

<sup>&</sup>lt;sup>§</sup>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  $\ge$ 25  $\mu g/dL$  (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

- Office of the Federal Register. Code of federal regulations: occupational safety and health standards. Subpart Z: Toxic and hazardous substances—lead. Washington, DC: Office of the Federal Register, National Archives and Records Administration, 1985. (29 CFR § 1910.1025).
- Ruttstein DD, Mullan RJ, Frazier TM, Halperin WE, Melius JM, Sestito JP. Sentinel health events (occupational): a basis for physician recognition and public health surveillance. Am J Public Health 1983;73:1054–62.
- Maizlish N, Rudolph L, Sutton P, Jones J, Kizer K. Elevated blood lead in California adults, 1987: results of a statewide surveillance program based on laboratory reports. Am J Public Health 1990:80:931–4.
- 4. Rempel D. The lead-exposed worker, JAMA 1989;262:532-4.
- Office of the Federal Register. Code of federal regulations: safety and health regulations for construction. Subpart J: Welding and cutting—welding, cutting, and heating in way of preservative coatings. Washington, DC: Office of the Federal Register, National Archives and Records Administration, 1988. (29 CFR § 1926.354).
- CDC. Surveillance for occupational lead exposure United States, 1987. MMWR 1989;38: 642–6.
- Public Health Service. Healthy people 2000: national health promotion and disease prevention objectives. Washington, DC: US Department of Health and Human Services, Public Health Service, 1990; DHHS publication no. (PHS)90-50212.

#### 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.

The Morbidity and Mortality Weekly Report is prepared by the Centers for Disease Control, Atlanta, Georgia, and is available on a paid subscription basis from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, (202) 783-3238.

The data in this report are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday. Accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials, as well as matters pertaining to editorial or other textual considerations should be addressed to: Editor, Morbidity and Mortality Weekly Report, Mailstop C-08, Centers for Disease Control, Atlanta, Georgia 30333; telephone (404) 332-4555.

Director, Centers for Disease Control William L. Roper, M.D., M.P.H. Director, Epidemiology Program Office

Stephen B. Thacker, M.D., M.Sc.



Editor, MMWR Series Richard A. Goodman, M.D., M.P.H. Managing Editor Karen L. Foster, M.A.

☆U.S. Government Printing Office: 1991-531-130/22056 Region IV

Penalty for Private Use

Atlanta,

Public Health Service

HEALTH AND HUMAN SERVICES Centers for Disease Control , Georgia 30333

×

POSTAGE & FEES PAID FIRST-CLASS MAIL Permit No. G-284 PHS/CDC