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Epidemiologic Notes and Reports

Injuries Associated with Ultraviolet Tanning Devices — Wisconsin

In 1986 and 1987, the Radiation Protection Section, Wisconsin Division of Health, surveyed dermatologists, ophthalmologists, and emergency room personnel to better understand the occurrence of injuries caused by ultraviolet (UV) tanning devices in Wisconsin. Questionnaires were distributed to 43 of 106 practicing dermatologists in the state who were attending the annual meeting of the Wisconsin Dermatological Society in October 1986; 31 (72%) questionnaires were completed and returned. Thirteen (42%) dermatologists had treated a total of 65 patients for cutaneous burns resulting from suntanning devices during the preceding 12 months; the degree of the burns was not specified. Forty-two (65%) patients used bed/booth devices, and 23 (35%) used reflector lamps.

Of questionnaires sent to 132 Wisconsin ophthalmologists (of 260 patient-care ophthalmologists in the state) from a list provided by the state Ophthalmologic Society, 115 (87%) were completed and returned; 48 (42%) ophthalmologists completing the questionnaire had treated a total of 152 patients during the preceding 12-month period for eye injuries related to tanning devices. Injuries included corneal injuries (129 [85%] patients), both corneal and retinal injuries (four [3%]), and unspecified ocular injuries (19 [13%]). Thirty-seven (24%) patients reportedly wore safety goggles during their tanning sessions. The UV sources used by the patients were bed/booth devices (80 [53%] patients), reflector bulb lamps (26 [17%]), and mercury vapor lamps (16 [11%]); for 30 (20%), no source was indicated.

One hundred forty-one (47%) responses were received from questionnaires mailed to 301 emergency physicians and emergency rooms listed with the Wisconsin Division of the American College of Emergency Room Physicians. Forty-one (29%) respondents reported that in a 12-month period they had treated 155 patients for skin burns, including 105 (68%) first-degree burns and 39 (25%) second-degree burns; severity of burn was not indicated for 11 (7%) patients. The UV sources were

Tanning Devices — Continued

bed/booth devices (94 [61%]) patients), reflector lamp devices (54 [35%]), and natural light (two [1%]); sources were not indicated for five (3%) patients. Ninety (58%) patients were injured at commercial tanning facilities, and 58 (37%) were injured at home; for seven (5%), location was not indicated. Ninety-one (59%) patients were treated for eye injuries, and 57 (63%) of these were referred to ophthalmologists.

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Editorial Note: Artificial suntanning with UV light has become increasingly popular in Wisconsin and nationwide. Types of equipment range from small, single-bulb sunlamps used in the home to elaborate bed/booth equipment used in commercial tanning facilities. Although the number of suntanning devices used in Wisconsin is unknown, sales estimates from three major suppliers indicate that approximately 850 commercial tanning beds/booths were shipped there during 1980–1987. Estimates of the number of persons using these devices are not available; however, an estimated 2 million persons nationwide used 10,000 commercial tanning facilities in 1985. The National Electronic Injury Surveillance System, which is based on emergency room data, estimates that in 1986 approximately 700 U.S. burn injuries were related to suntan booths, and approximately 2600 burn injuries were related to sunlamps.*

Tanning is an adaptive response by the skin to protect the body from the damaging effects of UV radiation. UV radiation is composed of three spectra—UV-A (320–400 nanometers [nm]), the least energetic; UV-B (280–320 nm); and UV-C (<280 nm), the most energetic. Natural sunlight that penetrates the atmosphere is composed of UV-A and UV-B (*1*). Most tanning devices producing UV-A radiation also emit some UV-B radiation (*2*). Although UV-A radiation is less likely than UV-B to cause erythema, it can cause other adverse health effects to the skin, eyes, blood vessels, and immune system (*1,2*).

The eyes are highly susceptible to injury from UV radiation. Photokeratitis and conjunctivitis can occur within hours after exposure unless protective goggles are properly worn. UV radiation, in addition to promoting aging of the skin, is thought to promote the formation of cataracts (*3*).

A retrospective study conducted in Michigan demonstrated a changing trend in the causes of corneal burns (*4*). During the study period (July 1, 1985–July 1, 1986), 62 patients seen in two emergency rooms were treated for UV-light–induced corneal burns; 25 (40%) of these patients had been exposed at a commercial tanning facility. In previous years, burns associated with exposure to UV radiation from commercial tanning facilities were rare, but in this study the number of corneal burns increased concurrently with an increase in the number of these facilities in the area.

Exposure to UV radiation is associated with an increased risk of skin cancer. More than 500,000 cases of basal and squamous cell carcinoma of the skin occur each year in the United States (*5*). These skin cancers occur most often on sun-exposed areas of the body and are believed to be caused by exposure to UV radiation. Other evidence suggests that malignant melanoma also may be associated with sun exposure (*1*).

*Estimates are available from the U.S. Consumer Products Safety Commission, National Injury Information Clearinghouse, National Electronic Injury Surveillance System.

Tanning Devices – Continued

Because of the manner in which the surveys in Wisconsin were constructed, recall bias and multiple reporting of cases by different practitioners are probable. Questionnaires to emergency rooms and emergency physicians were designed to separate eye injuries from skin injuries. Because a person may have sustained both kinds of injuries, each of which would have been reported separately, overlap may also exist. Although the surveys do not reflect the total number of residents who have been acutely injured by using UV tanning devices in 1986–1987 or the risk factors associated with the use of these devices, survey results indicate that injuries associated with UV tanning devices can be severe enough to require medical attention. The extent of acute injuries associated with UV tanning devices may be underestimated, since medical assistance may not be sought for all injuries.

The long-term effects of UV tanning devices are not known (*5*); however, these devices have no known health benefits (*2,6*). Therefore, persons who choose to use these UV tanning devices should be aware of the potential risks and should follow the manufacturer's directions to minimize these risks. Protective goggles should be properly worn. Medications can increase photosensitivity, and persons on medication should consult their physician or pharmacist before using any tanning devices.

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Human Rabies – Oregon, 1989

On February 7, 1989, rabies was identified as the cause of death in an 18-year-old Mexican man who had died 4 days earlier of acute encephalitis in Oregon. He had no known exposure to the disease. This was the first case of human rabies in the United States since 1987 and the first in Oregon since 1978.

The patient was well until January 17, 1989, when he developed fever, nausea, vomiting, dyspnea, and cough. On January 22, he was treated at a local emergency room for bronchitis. On January 24, he went to another clinic with complaints of chills, myalgias, and sore throat and was diagnosed as having a viral upper respiratory illness. He was admitted to a Portland, Oregon, hospital on January 26 with fever, chills, and localized periumbilical pain suggesting acute appendicitis; during the next 2 days, the pain continued. Although his fever persisted, serial peripheral white cell counts remained normal. Ultrasound and two computerized axial tomography (CAT) scans of his abdomen were normal.

Rabies — Continued

On January 28, the patient developed vertigo and subsequent acute obtundation. CAT scan of his head was normal; however, examination of the cerebrospinal fluid (CSF) revealed a mild pleocytosis with 9 white blood cells/mm^3 (8% segmented polymorphonuclear cells, 78% lymphocytes, 10% macrophages, and 4% monocytes) and 10 red blood cells/mm^3. The CSF glucose level was 81 mg/dL, and protein was 39 mg/dL. Tests on spinal fluid, blood, urine, sputum, and stool were negative for bacterial, fungal, viral, and mycobacterial pathogens. An electroencephalogram revealed mild to moderate slowing of electrical activity and did not suggest herpes encephalitis. On January 30, he had areflexia of all his deep tendons and asymmetrical palsies of cranial nerves VII and XII; that day, the patient had a cardiopulmonary arrest. He died February 3.

Although the possibility of rabies had been considered during hospitalization, specific diagnostic tests were not obtained until after the patient died. Direct fluorescent antibody staining of brain tissue collected at autopsy and submitted to the Oregon Public Health Laboratory was positive for rabies virus. Monoclonal antibody testing by CDC determined the antigenic pattern of the virus was the one found in areas of Latin America with enzootic canine rabies and in areas of California with enzootic skunk rabies.

During the 72 hours after diagnosis, extensive interviews were conducted with the patient's co-workers in Oregon, including two who originally traveled with him from Michoacán, Mexico. In March 1988, 11 months before onset of symptoms, the patient and his companions had driven by car from Michoacán through California to Oregon. Except for two trips to Washington in September and December of 1988, the patient had remained in northern Oregon, where he worked as an agricultural laborer. Interviews failed to identify a possible source of rabies exposure. Mexican health officials conducted an investigation in the patient's home area but found no additional information on possible exposures to rabies. Postexposure rabies prophylaxis was recommended for seven of his co-workers and two hospital workers who reported nonbite exposures to the patient's saliva.

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Editorial Note: As human rabies has decreased in the United States, the proportion of rabies patients with no known exposures to rabid animals has increased. Between 1960 and 1979, a source of infection was not identified in 16% (6/38) of U.S. rabies cases (*1*). Since 1980, the proportion has increased to 60% (6/10); none of the three most recent patients reported exposure (*2–7*). Of the 38 human cases during 1960–1979, rabies was diagnosed before death in 30 (79%) (*1*), in contrast to only 40% of the five most recent cases (*3–6*). Rabies is often not considered in the differential diagnosis in persons with no known recent exposure to animals.

It was unlikely that this patient's infection was acquired in Oregon for the following reasons. First, antigenic typing of the rabies virus, which can help determine the geographic source of infection (*8,9*), suggested that infection had occurred in areas of Latin America with enzootic dog rabies or areas of California with enzootic skunk rabies. Although the patient might have been bitten by a skunk during his 2-day trip

Rabies – Continued

through California, his traveling companions were unaware of such an event. Second, Oregon surveillance data since 1984 show that none of 33 skunks tested were positive for rabies. Based on this information and on the absence of reported indigenous skunk rabies in Oregon since 1966, Mexico was considered the most likely source of exposure. Regardless of whether the patient was exposed in Mexico or California, the incubation period would have exceeded 10 months.

For this patient, specific diagnostic tests for rabies might have been delayed because the initial clinical presentation suggested respiratory and gastrointestinal infection. Although respiratory tract infection is the most common diagnosis initially considered in patients with rabies, it was present in <20% of cases in one review (*1*).

Although only six cases of human-to-human rabies transmission – all in cornea transplant patients – have been well documented (*10–14*), there is a theoretical risk of human-to-human transmission (*10,15*) by bites or direct saliva contact to mucous membranes or broken skin. This risk, although low, was of sufficient concern that postexposure prophylaxis was recommended for nine persons in the Oregon case.

For this episode, only a small proportion of health-care workers and other persons received postexposure prophylaxis. In contrast, for the 10 U.S. cases from 1977 to 1979, an average of 49 contacts per patient were treated (*1*). An average of 92 contacts per case for four recent U.S. human rabies cases received prophylaxis (*3,4,6,7*). However, hospitals are moving toward the implementation of universal precautions (*16*); this practice may help explain why so few health-care workers in Oregon needed prophylaxis.

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Occupational and Paraoccupational Exposure to Lead — Colorado

On December 10, 1987, the Environmental and Occupational Disease Surveillance Project of the Colorado Department of Health (CDH) received a report that a 29-year-old man had a blood-lead level of 170 μg/dL, a level almost seven times the blood-lead concentration required to be reported to CDH (≥25 μg/dL). The patient had been hospitalized on November 15, 1987, for increasingly severe chronic abdominal pain and cramping. He had been hospitalized in 1986 for similar complaints and had been treated with cimetidine for a presumptive diagnosis of peptic ulcer disease.

The patient was discharged on November 17, 1987, after symptoms subsided. Later, his physician received reports of both an excessive blood-lead level (obtained during hospitalization) and a free erythrocyte protoporphyrin (FEP) level of 166.3 μg/dL (normal: <35 μg/dL). The patient was readmitted December 5, 1987, for chelation therapy with the use of ethylenediaminetetraacetic acid (EDTA), and he was discharged after his blood-lead level had fallen to 55 μg/dL. He was readmitted in January 1988 with a blood-lead level of 78 μg/dL and was again chelated with EDTA. Follow-up testing later that month showed his blood-lead level to be 20 μg/dL.

An occupational history revealed that since 1981 the patient had worked at a company that produces belt buckles, plaques, and awards. The patient was specifically involved in the manufacture of lead belt buckles and other lead products. His duties included pouring molten lead into a mold, removing the buckles from the mold, and grinding and smoothing imperfections in the product. In February 1988, the Tri-County Health Department (TCHD) conducted an environmental investigation of the patient's worksite. Investigators found that during the patient's 6-year employment at the company, ventilation for the melting and grinding areas was inadequate. A half-face respirator was available to the patient but was rarely used; furthermore, no routine maintenance was performed on the equipment, and inappropriate respirator filters were provided. During the site investigation, the grinding operation produced a visible cloud of particulate in the breathing zone of the operators, and a fine dust was found throughout the facility. Although environmental sampling was not done, the sampling of a similar operation in Colorado Springs (*1*) indicated that this type of grinding operation may produce lead exposures as high as 1900 μg/m^3 (Occupational Safety and Health Administration [OSHA] permissible exposure limit [PEL]: 50 μg/m^3 [*2*]).

In November 1987, after the discovery of this employee's elevated blood-lead level, the company terminated its lead-pouring operation. The company did not have a blood-lead–monitoring program; however, three of the five employees tested by CDH and TCHD in February 1988 had blood-lead levels exceeding 50 μg/dL (range, 14–58 μg/dL).* Seven employees at the facility, including the owners, refused blood-lead testing. Breathing-zone air samples collected in February 1988 contained lead particulate levels ranging from 35 μg/m^3 in the polishing room to 1121 μg/m^3 in the grinding area. These elevated lead levels were attributed to residual lead contamination from the terminated production process and to lead present in the copper, zinc, and nickel alloys used in other processes.

*OSHA regulations state that an employee whose confirmed blood-lead level exceeds 60 μg/dL must be removed from lead exposure; similarly, an employee whose average blood-lead level (measured on three occasions within 6 months) exceeds 50 μg/dL also must be removed from lead exposure (*2*).

Lead Exposure — Continued

Between February and August 1988, TCHD assisted the company in decreasing workplace lead exposures by introducing local exhaust ventilation at the grinding, polishing, and soldering operations. Follow-up blood sampling indicated that the blood-lead level of the employee who had the highest level during the first sampling (58 μg/dL) had dropped to 27 μg/dL, but another employee's blood-lead level remained elevated (52 μg/dL). Four air samples collected in the facility in August 1988 contained lead concentrations below the OSHA PEL (range, 20–44 $\mu g/m^3$). A fifth sample, obtained in the melting and grinding area, had a lead concentration of 140 $\mu g/m^3$; planned modifications to the ventilation system may reduce lead exposure to permissible levels.

The index patient's wife and three daughters were also screened for evidence of "paraoccupational exposure," which may occur when workers exposed to hazardous substances in their jobs carry the toxic materials home, usually on work clothing, thus exposing family members (*3*). The patient's children had blood-lead and/or FEP levels that exceeded CDC-recommended concentrations (*4*) (Table 1). Radiographs of his 4-year-old daughter showed dense metaphyses adjacent to the epiphyseal plates at the distal ulna and radius. (Increased metaphyseal density in long bones is indicative of excessive lead absorption in growing children [*4*].) All three children were given penicillamine chelation therapy. When retested, the 4-year-old girl had a blood-lead level of 41 μg/dL and was retreated.

None of the other employees who agreed to blood-lead screening had children at risk of paraoccupational exposure.

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Editorial Note: This report emphasizes the importance of occupational disease surveillance of sentinel health events, i.e., an unnecessary disease, disability, or untimely death that is occupationally related and whose occurrence may 1) provide the impetus for epidemiologic or industrial hygiene studies or 2) serve as a warning that materials substitution, engineering control, personal protection, or medical care may be required (*6*).

In February 1985, CDH instituted a network for surveillance of selected sentinel health events. This activity, which began as part of a cooperative agreement among CDH and CDC's National Institute for Occupational Safety and Health (NIOSH) and Center for Environmental Health and Injury Control, was designed to improve CDH's surveillance capacity. In this investigation, one case of lead poisoning reported through the CDH surveillance system resulted in the identification of other persons at

TABLE 1. Lead and FEP* levels for family members† of the index patient

Family member	Age (yrs)	Blood lead (μg/dL)	FEP (μg/dL)
Wife	25	15	36
Daughter	7	13	92
Daughter	6	29	305
Daughter	4	37	196

*Free erythrocyte protoporphyrin.

†Excessive absorption of lead in children is indicated by a blood-lead level ≥25 μg/dL. Lead toxicity is indicated by an elevated blood-lead level with an FEP of ≥35 μg/dL (*5*).

Lead Exposure — Continued

risk for lead exposure and the implementation of workplace controls to reduce lead exposure among employees and their families. Sentinel occupational health events such as the one reported here demonstrate the value of state programs that mandate routine reporting of elevated blood-lead levels by laboratories and physicians. They also illustrate the continuing problem of occupational and subsequent paraoccupational lead exposure (*5*).

The importance of obtaining an occupational history as part of a medical record is demonstrated by the index patient's first hospitalization and treatment for a presumed peptic ulcer. Recognition of the patient's occupational exposure to lead at that time may have accelerated diagnosis and treatment of lead poisoning and decreased his children's exposure to lead and its attendant risks of long-term neurobehavioral effects. Blood-lead levels of children exposed to lead through a parent's occupation are higher than those of unexposed controls (*3,7*). In this instance, clinical lead toxicity (CDC definition: ≥25 μg/dL blood lead, ≥35 μg/dL FEP [*4*]) developed in two

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TABLE I. Summary — cases of specified notifiable diseases, United States

Disease	19th Week Ending			Cumulative, 19th Week Ending		
	May 13, 1989	May 14, 1988	Median 1984-1988	May 13, 1989	May 14, 1988	Median 1984-1988
Acquired Immunodeficiency Syndrome (AIDS)	387	U*	174	12,287	11,103	4,585
Aseptic meningitis	86	90	84	1,457	1,482	1,482
Encephalitis: Primary (arthropod-borne & unspec)	16	15	16	222	251	302
Post-infectious	2	2	2	31	38	37
Gonorrhea: Civilian	9,505	11,791	14,562	232,119	241,459	291,875
Military	175	261	363	4,018	4,591	6,357
Hepatitis: Type A	581	478	428	12,196	9,011	8,067
Type B	568	458	500	7,796	7,817	9,037
Non A, Non B	28	48	76	830	958	1,261
Unspecified	46	29	94	934	781	1,743
Legionellosis	11	28	13	298	322	230
Leprosy	1	5	5	50	70	79
Malaria	17	12	12	374	243	257
Measles: Total†	311	109	109	4,002	1,021	1,237
Indigenous	296	100	100	3,765	902	1,100
Imported	15	9	12	237	119	137
Meningococcal infections	71	73	61	1,303	1,381	1,308
Mumps	71	252	117	2,062	2,230	1,529
Pertussis	84	26	31	707	787	755
Rubella (German measles)	3	2	21	118	76	181
Syphilis (Primary & Secondary): Civilian	575	693	523	14,268	13,632	10,279
Military	4	3	4	104	72	75
Toxic Shock syndrome	9	8	7	133	121	133
Tuberculosis	376	388	389	6,956	6,806	7,165
Tularemia	-	2	4	19	32	34
Typhoid Fever	6	10	7	147	136	109
Typhus fever, tick-borne (RMSF)	8	6	7	44	35	50
Rabies, animal	109	87	106	1,640	1,445	1,812

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1989		Cum. 1989
Anthrax	-	Leptospirosis	50
Botulism: Foodborne	6	Plague	-
Infant	3	Poliomyelitis, Paralytic	-
Other	3	Psittacosis (Ohio 1, Wash. 1)	32
Brucellosis (N.C. 1, Tex. 6)	20	Rabies, human	-
Cholera	-	Tetanus (Ala. 1, Calif. 1)	17
Congenital rubella syndrome	1	Trichinosis (Me. 1, Calif. 1)	12
Congenital syphilis, ages < 1 year	-		
Diphtheria	-		

*Because AIDS cases are not received weekly from all reporting areas, comparison of weekly figures may be misleading.
†Nine of the 311 reported cases for this week were imported from a foreign country or can be directly traceable to a known internationally imported case within two generations.

TABLE III. Cases of specified notifiable diseases, United States, weeks ending May 13, 1989 and May 14, 1988 (19th Week)

Reporting Area	AIDS	Aseptic Meningitis	Encephalitis		Gonorrhea (Civilian)		Hepatitis (Viral), by type				Legionellosis	Leprosy
			Primary	Post-infectious			A	B	NA,NB	Unspecified		
	Cum. 1989	Cum. 1989	Cum. 1989	Cum. 1989	Cum. 1989	Cum. 1988	Cum. 1989	Cum. 1989	Cum. 1989	Cum. 1989	Cum. 1989	Cum. 1989
UNITED STATES	12,287	1,457	222	31	232,119	241,459	12,196	7,796	830	934	298	50
NEW ENGLAND	493	63	7	2	6,741	7,294	270	409	37	37	22	4
Maine	30	3	3	-	105	165	4	18	3	1	3	-
N.H.	13	2	-	-	64	111	27	24	7	3	-	-
Vt.	5	1	-	-	24	60	13	30	4	-	-	-
Mass.	262	28	2	2	2,579	2,578	92	250	15	27	13	3
R.I.	25	20	-	-	514	658	16	36	3	2	6	-
Conn.	158	9	2	-	3,455	3,722	118	51	5	4	-	1
MID. ATLANTIC	3,478	203	40	3	33,092	38,314	1,635	1,203	80	126	79	5
Upstate N.Y.	493	85	11	2	5,734	4,393	397	274	35	4	26	1
N.Y. City	1,691	32	2	1	14,869	17,825	139	421	13	106	8	2
N.J.	846	1	27	-	5,116	5,400	165	213	11	5	12	1
Pa.	448	85	-	-	7,373	10,696	934	295	21	11	33	1
E.N. CENTRAL	986	226	72	1	40,485	38,876	714	1,029	88	35	78	1
Ohio	156	50	15	-	11,310	9,156	149	207	14	4	46	-
Ind.	185	52	19	-	2,920	3,028	40	154	14	12	15	1
Ill.	424	47	14	1	12,238	11,005	349	327	21	12	-	-
Mich.	187	67	19	-	11,532	12,397	129	248	27	7	13	-
Wis.	34	10	5	-	2,485	3,290	47	93	12	-	4	-
W.N. CENTRAL	271	56	7	2	10,893	9,625	342	287	28	5	7	1
Minn.	61	5	-	1	1,103	1,352	36	41	6	2	2	-
Iowa	24	12	2	-	945	750	30	16	7	-	2	-
Mo.	133	17	-	-	6,380	5,369	193	200	9	3	1	-
N. Dak.	3	3	1	-	42	73	3	9	3	-	-	-
S. Dak.	4	4	1	-	99	195	3	5	3	-	-	-
Nebr.	11	4	2	-	638	578	50	11	-	-	2	1
Kans.	35	11	1	1	1,686	1,308	27	5	-	-	-	-
S. ATLANTIC	2,580	318	28	6	65,557	67,198	1,005	1,544	118	127	39	-
Del.	41	10	1	-	1,043	974	18	59	1	1	3	-
Md.	282	34	6	1	7,408	7,064	231	301	13	14	10	-
D.C.	222	5	-	-	4,054	4,705	2	10	1	-	-	-
Va.	225	62	14	-	5,439	4,704	98	108	20	76	2	-
W. Va.	19	2	3	-	496	554	9	33	2	2	-	-
N.C.	157	44	-	1	9,740	10,103	185	395	40	-	12	-
S.C.	121	9	-	-	5,788	4,930	14	172	3	5	2	-
Ga.	357	21	1	-	13,013	13,388	130	149	7	4	4	-
Fla.	1,156	131	3	4	18,576	20,776	318	317	31	25	6	-
E.S. CENTRAL	301	138	13	1	19,286	18,650	130	564	61	1	10	-
Ky.	45	33	4	1	1,846	1,541	51	158	21	-	3	-
Tenn.	99	19	-	-	6,245	6,202	29	276	16	-	4	-
Ala.	90	68	9	-	6,146	6,246	29	85	21	1	3	-
Miss.	67	18	-	-	5,049	4,661	21	45	3	-	-	-
W.S. CENTRAL	1,013	107	24	2	25,279	27,162	1,400	726	52	211	18	11
Ark.	33	3	-	-	2,584	2,522	80	28	2	2	1	-
La.	157	14	5	-	5,427	5,694	112	129	5	1	4	-
Okla.	59	15	6	-	2,166	2,482	149	71	9	9	10	-
Tex.	764	75	13	2	15,102	16,464	1,059	498	36	199	3	11
MOUNTAIN	399	52	7	1	4,630	5,157	1,795	486	90	72	18	1
Mont.	1	1	-	-	71	151	16	17	1	1	2	1
Idaho	10	-	-	-	78	146	76	33	5	2	-	-
Wyo.	8	-	-	-	47	91	15	1	-	-	-	-
Colo.	139	17	2	1	1,077	1,206	257	78	32	36	2	-
N. Mex.	23	5	-	-	508	498	213	81	21	1	-	-
Ariz.	109	22	2	-	1,554	1,744	957	167	15	28	8	-
Utah	26	5	1	-	161	231	111	33	10	3	3	-
Nev.	83	2	2	-	1,134	1,090	150	76	6	1	3	-
PACIFIC	2,766	294	24	13	26,156	29,183	4,905	1,548	276	320	27	27
Wash.	198	-	-	1	2,159	2,451	1,018	283	74	17	5	1
Oreg.	100	-	-	-	1,086	1,099	815	145	32	6	1	1
Calif.	2,438	274	21	12	22,416	24,969	2,631	1,099	165	293	19	21
Alaska	5	1	2	-	330	398	380	19	5	2	1	-
Hawaii	25	19	1	-	165	266	61	2	-	2	1	4
Guam	-	-	-	-	-	53	-	-	-	-	-	-
P.R.	615	38	1	-	379	552	39	74	5	7	-	7
V.I.	16	-	-	-	199	144	-	4	-	-	-	-
Amer. Samoa	-	-	-	-	-	22	-	-	-	-	-	-
C.N.M.I.	-	-	-	-	-	19	-	-	-	-	-	-

N: Not notifiable U: Unavailable C.N.M.I.: Commonwealth of the Northern Mariana Islands

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending May 13, 1989 and May 14, 1988 (19th Week)

Reporting Area	Malaria	Measles (Rubeola)					Menin-gococcal Infections	Mumps		Pertussis			Rubella		
		Indigenous		Imported*		Total									
	Cum. 1989	1989	Cum. 1989	1989	Cum. 1989	Cum. 1988	Cum. 1989	1989	Cum. 1989	1989	Cum. 1989	Cum. 1988	1989	Cum. 1989	Cum. 1988
UNITED STATES	374	296	3,765	15	237	1,021	1,303	71	2,062	84	707	787	3	118	76
NEW ENGLAND	21	11	42	3	14	64	93	-	19	46	103	77	1	2	1
Maine	-	-	-	-	-	-	12	-	-	-	4	11	-	-	-
N.H.	1	-	1	-	-	56	10	-	10	-	5	22	-	-	-
Vt.	-	-	1	-	-	-	6	-	-	1	6	1	-	1	-
Mass.	13	-	9	3†§	12	1	42	-	8	44	83	33	1	1	-
R.I.	4	11	29	-	2	-	1	-	-	-	2	1	-	-	1
Conn.	3	-	2	-	-	7	22	-	1	1	3	9	-	-	-
MID. ATLANTIC	63	10	211	8	107	285	171	4	99	-	45	36	1	5	7
Upstate N.Y.	13	1	15	1†	77	6	52	4	47	-	25	21	-	1	1
N.Y. City	20	1	25	-	13	24	25	-	8	-	2	1	1	4	4
N.J.	13	-	112	-	-	15	39	-	11	-	14	4	-	-	1
Pa.	17	8	59	7†§	17	240	55	-	33	-	4	10	-	-	1
E.N. CENTRAL	19	124	688	-	38	56	160	3	203	-	35	94	1	16	21
Ohio	6	72	400	-	35	3	67	-	8	-	1	21	1	3	-
Ind.	3	-	17	-	-	-	18	-	18	-	8	38	-	-	-
Ill.	4	52	271	-	-	40	44	1	95	-	-	5	-	12	17
Mich.	4	-	-	-	1	13	24	2	69	-	19	16	-	-	4
Wis.	2	-	-	-	2	-	7	-	13	-	7	14	-	1	-
W.N. CENTRAL	8	-	248	-	2	-	35	1	263	-	17	35	-	2	-
Minn.	5	-	-	-	-	-	10	-	-	-	-	5	-	-	-
Iowa	-	-	-	-	1	-	-	1	13	-	6	14	-	-	-
Mo.	2	-	205	-	-	-	8	-	41	-	9	5	-	1	-
N. Dak.	1	-	-	-	-	-	-	-	-	-	-	6	-	-	-
S. Dak.	-	-	-	-	-	-	4	-	-	-	1	2	-	-	-
Nebr.	-	-	-	-	-	-	10	-	2	-	-	-	-	-	-
Kans.	-	-	43	-	1	-	3	-	207	-	1	3	-	1	-
S. ATLANTIC	65	4	246	-	15	203	212	12	321	3	60	76	-	4	3
Del.	1	-	34	-	1	-	2	-	-	-	-	3	-	-	-
Md.	14	-	6	-	6	4	33	-	151	1	6	17	-	2	-
D.C.	3	-	5	-	3	-	9	4	58	-	-	-	-	-	-
Va.	9	1	1	-	2	100	27	4	57	-	4	11	-	-	-
W. Va.	1	-	-	-	-	6	8	1	9	-	9	-	-	-	-
N.C.	10	3	159	-	-	1	30	2	12	-	15	24	-	1	-
S.C.	2	-	-	-	-	-	14	-	15	-	-	-	-	-	-
Ga.	4	-	-	-	-	-	37	-	3	2	8	14	-	-	-
Fla.	21	-	41	-	3	92	52	1	16	-	18	7	-	1	3
E.S. CENTRAL	4	5	22	-	-	40	35	2	78	1	30	13	-	1	-
Ky.	-	-	2	-	-	23	19	-	9	-	1	-	-	-	-
Tenn.	-	-	1	-	-	-	2	2	24	-	8	8	-	1	-
Ala.	2	5	19	-	-	-	11	-	6	1	21	3	-	-	-
Miss.	2	-	-	-	-	17	3	N	N	-	-	2	-	-	-
W.S. CENTRAL	18	132	1,912	-	23	9	103	45	810	-	22	35	-	11	3
Ark.	-	-	-	-	-	-	4	3	77	-	10	5	-	-	2
La.	1	-	6	-	-	-	20	23	286	-	4	6	-	5	-
Okla.	1	-	23	-	-	8	8	1	146	-	8	24	-	1	1
Tex.	16	132	1,883	-	23	1	71	18	301	-	-	-	-	5	-
MOUNTAIN	14	10	62	-	17	115	34	3	88	13	288	285	-	2	2
Mont.	-	-	12	-	1	-	1	-	2	-	-	1	-	1	-
Idaho	2	-	-	-	1	1	-	-	6	-	31	233	-	-	-
Wyo.	1	-	-	-	-	-	-	-	6	-	-	1	-	-	-
Colo.	1	7	28	-	1	114	13	1	7	1	18	7	-	-	1
N. Mex.	1	2	11	-	14	-	1	N	N	-	4	2	-	-	-
Ariz.	6	1	11	-	-	-	17	2	60	12	228	19	-	-	-
Utah	-	-	-	-	-	-	2	-	3	-	6	21	-	-	-
Nev.	3	-	-	-	-	-	-	-	4	-	1	1	-	1	1
PACIFIC	162	-	334	4	21	249	460	1	181	21	107	136	-	75	39
Wash.	9	-	6	-	10	-	43	-	15	1	23	29	-	-	-
Oreg.	8	-	-	4†§	4	3	32	N	N	-	4	4	-	1	-
Calif.	141	-	322	-	3	242	381	-	158	20	78	80	-	57	33
Alaska	2	-	-	-	-	-	3	-	-	-	-	3	-	-	-
Hawaii	2	-	6	-	4	4	1	1	8	-	2	20	-	17	6
Guam	-	U	-	U	-	1	-	U	-	U	-	-	U	-	1
P.R.	-	31	303	-	-	158	3	-	1	-	2	6	-	4	-
V.I.	-	U	-	U	-	-	-	U	6	U	-	-	U	-	-
Amer. Samoa	-	U	-	U	-	-	-	U	-	U	-	-	U	-	-
C.N.M.I.	-	U	-	U	-	-	-	U	-	U	-	-			

*For measles only, imported cases includes both out-of-state and international importations.

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

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending May 13, 1989 and May 14, 1988 (19th Week)

Reporting Area	Syphilis (Civilian) (Primary & Secondary)		Toxic-shock Syndrome	Tuberculosis		Tula-remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1989	Cum. 1988	Cum. 1989	Cum. 1989	Cum. 1988	Cum. 1989	Cum. 1989	Cum. 1989	Cum. 1989
UNITED STATES	14,268	13,632	133	6,956	6,806	19	147	44	1,640
NEW ENGLAND	608	376	4	165	133	-	10	1	2
Maine	5	5	2	3	3	-	-	-	1
N.H.	2	4	-	10	-	-	-	-	-
Vt.	-	-	-	2	-	-	-	-	-
Mass.	179	159	-	89	85	-	5	-	-
R.I.	14	12	-	22	11	-	4	1	-
Conn.	408	196	2	39	34	-	1	-	1
MID. ATLANTIC	2,624	2,809	24	1,378	1,244	1	40	4	221
Upstate N.Y.	295	184	3	98	206	-	4	2	4
N.Y. City	1,189	1,829	2	818	568	-	26	-	-
N.J.	491	309	7	210	224	-	7	-	-
Pa.	649	487	12	252	246	1	3	2	217
E.N. CENTRAL	547	425	16	800	788	1	18	7	30
Ohio	38	44	8	145	144	-	7	6	-
Ind.	23	21	3	69	86	-	1	1	2
Ill.	249	222	-	340	316	-	6	-	3
Mich.	217	123	5	206	193	-	3	-	4
Wis.	20	15	-	40	49	1	1	-	21
W.N. CENTRAL	122	86	23	204	182	3	4	2	220
Minn.	8	8	6	44	31	-	1	-	51
Iowa	15	10	4	28	14	-	2	1	63
Mo.	62	48	4	81	91	3	1	1	19
N. Dak.	1	1	-	6	4	-	-	-	11
S. Dak.	-	-	3	12	16	-	-	-	40
Nebr.	16	13	5	9	4	-	-	-	16
Kans.	20	6	1	24	22	-	-	-	20
S. ATLANTIC	5,421	4,829	11	1,485	1,512	1	11	20	496
Del.	66	52	-	19	17	-	2	-	13
Md.	283	264	-	138	161	-	1	4	126
D.C.	318	207	-	67	68	-	2	-	2
Va.	201	156	1	135	161	1	1	-	97
W. Va.	7	2	-	30	32	-	-	-	25
N.C.	338	277	4	147	109	-	2	13	-
S.C.	265	226	3	157	158	-	-	2	83
Ga.	1,137	791	2	197	224	-	-	1	86
Fla.	2,806	2,854	1	595	582	-	3	-	64
E.S. CENTRAL	962	738	2	593	565	2	1	6	142
Ky.	23	25	1	142	153	1	1	4	71
Tenn.	401	312	-	149	145	-	-	1	32
Ala.	328	215	1	180	180	-	-	1	39
Miss.	210	186	-	122	87	1	-	-	-
W.S. CENTRAL	1,926	1,441	9	786	856	7	6	2	279
Ark.	118	70	1	89	87	3	-	1	37
La.	439	259	-	95	122	-	1	-	4
Okla.	30	60	6	60	79	4	-	1	40
Tex.	1,339	1,052	2	542	568	-	5	-	198
MOUNTAIN	262	254	16	180	143	2	1	1	74
Mont.	-	2	-	5	-	-	-	-	34
Idaho	-	-	1	6	-	-	-	-	-
Wyo.	1	1	-	-	1	-	-	-	21
Colo.	46	36	4	12	20	1	-	1	-
N. Mex.	11	19	2	32	35	-	-	-	11
Ariz.	67	73	8	85	58	-	1	-	7
Utah	9	9	-	19	10	1	-	-	-
Nev.	128	114	1	21	19	-	-	-	1
PACIFIC	1,796	2,674	28	1,365	1,383	2	56	1	176
Wash.	91	85	1	73	78	-	2	-	-
Oreg.	113	104	-	49	47	-	4	1	-
Calif.	1,584	2,465	26	1,165	1,187	2	48	-	123
Alaska	3	6	-	17	13	-	-	-	53
Hawaii	5	14	1	61	58	-	2	-	-
Guam	-	1	-	-	7	-	-	-	-
P.R.	189	235	-	91	81	-	-	-	21
V.I.	1	1	-	3	3	-	-	-	-
Amer. Samoa	-	-	-	-	3	-	-	-	-
C.N.M.I.	-	1	-	-	8	-	-	-	-

U: Unavailable

TABLE IV. Deaths in 121 U.S. cities,* week ending May 13, 1989 (19th Week)

Reporting Area	All Causes, By Age (Years)						P&I**
	All Ages	≥65	45-64	25-44	1-24	<1	Total
NEW ENGLAND	599	403	99	64	14	19	51
Boston, Mass.	194	111	37	30	5	11	24
Bridgeport, Conn.	39	29	5	4	-	1	1
Cambridge, Mass.	14	13	-	1	-	-	2
Fall River, Mass.	15	14	1	-	-	-	-
Hartford, Conn.	57	32	11	12	-	2	5
Lowell, Mass.	20	15	1	2	2	-	1
Lynn, Mass.	13	7	5	1	-	-	1
New Bedford, Mass.	16	13	1	1	-	1	-
New Haven, Conn.	44	30	9	4	1	-	5
Providence, R.I.	49	34	10	2	3	-	-
Somerville, Mass.	8	8	-	-	-	-	1
Springfield, Mass.	45	34	7	1	2	1	3
Waterbury, Conn.	30	22	5	3	-	-	6
Worcester, Mass.	55	41	7	3	1	3	2
MID. ATLANTIC	2,688	1,727	543	284	57	77	152
Albany, N.Y.	54	36	8	4	2	4	2
Allentown, Pa.	33	28	4	1	-	-	-
Buffalo, N.Y.	114	73	31	5	3	2	8
Camden, N.J.	48	33	9	3	1	2	-
Elizabeth, N.J.	17	12	4	-	1	-	2
Erie, Pa.†	48	36	9	3	-	-	4
Jersey City, N.J.	61	43	8	9	1	-	4
N.Y. City, N.Y.	1,359	814	285	186	32	42	48
Newark, N.J.	83	38	22	12	4	7	10
Paterson, N.J.	24	14	4	1	2	3	2
Philadelphia, Pa.	397	271	77	35	4	10	32
Pittsburgh, Pa.†	55	29	14	7	2	3	5
Reading, Pa.	34	30	4	-	-	-	5
Rochester, N.Y.	124	94	22	7	1	-	15
Schenectady, N.Y.	26	20	4	-	2	-	1
Scranton, Pa.†	24	21	2	-	-	1	4
Syracuse, N.Y.	110	75	23	8	1	3	3
Trenton, N.J.	26	19	4	2	1	-	3
Utica, N.Y.	20	14	5	1	-	-	2
Yonkers, N.Y.	31	27	4	-	-	-	2
E.N. CENTRAL	2,142	1,404	460	158	41	78	94
Akron, Ohio	56	34	12	6	-	4	-
Canton, Ohio	25	18	4	2	-	1	4
Chicago, Ill.§	564	362	125	45	10	22	16
Cincinnati, Ohio	141	93	30	7	3	8	15
Cleveland, Ohio	171	97	43	17	5	9	5
Columbus, Ohio	87	56	23	4	-	3	-
Dayton, Ohio	108	77	22	5	1	3	4
Detroit, Mich.	209	108	51	31	10	9	3
Evansville, Ind.	30	24	2	3	1	-	1
Fort Wayne, Ind.	58	36	15	3	3	1	4
Gary, Ind.	9	2	5	1	-	1	2
Grand Rapids, Mich.	66	52	9	2	-	3	7
Indianapolis, Ind.	142	92	32	11	2	5	5
Madison, Wis.	36	26	6	4	-	-	1
Milwaukee, Wis.	132	93	31	4	1	3	1
Peoria, Ill.	39	28	10	-	-	1	3
Rockford, Ill.	40	28	6	2	2	2	2
South Bend, Ind.	61	50	7	2	2	-	11
Toledo, Ohio	103	79	16	5	1	2	8
Youngstown, Ohio	65	49	11	4	-	1	2
W.N. CENTRAL	860	623	142	39	29	26	56
Des Moines, Iowa	70	47	13	4	4	2	3
Duluth, Minn.	34	26	6	1	1	-	4
Kansas City, Kans.	42	25	10	4	1	1	1
Kansas City, Mo.	120	87	21	3	4	5	14
Lincoln, Nebr.	34	30	1	2	-	1	2
Minneapolis, Minn.	276	196	44	17	12	7	20
Omaha, Nebr.	81	58	14	3	2	4	6
St. Louis, Mo.	127	95	18	5	4	5	2
St. Paul, Minn.	53	40	11	-	1	1	4
Wichita, Kans.§	23	19	4	-	-	-	-
S. ATLANTIC	1,334	804	262	163	46	56	57
Atlanta, Ga.	171	95	38	31	6	1	6
Baltimore, Md.	250	155	47	26	11	11	19
Charlotte, N.C.	77	44	14	13	3	3	3
Jacksonville, Fla.	117	74	28	7	6	2	5
Miami, Fla.	154	87	33	23	3	8	2
Norfolk, Va.	46	24	9	7	-	6	-
Richmond, Va.	96	57	16	9	5	8	11
Savannah, Ga.	43	33	6	3	1	-	4
St. Petersburg, Fla.	81	64	6	7	1	3	1
Tampa, Fla.	54	33	12	7	-	1	3
Washington, D.C.	208	112	43	29	10	13	2
Wilmington, Del.	37	26	10	1	-	-	1
E.S. CENTRAL	752	477	153	72	21	29	57
Birmingham, Ala.	115	75	15	14	3	8	3
Chattanooga, Tenn.	53	33	7	9	3	1	3
Knoxville, Tenn.	69	46	14	6	2	1	8
Louisville, Ky.	115	73	30	6	2	4	7
Memphis, Tenn.	197	126	47	15	7	2	24
Mobile, Ala.	39	18	8	9	-	4	1
Montgomery, Ala.	41	31	7	1	1	1	1
Nashville, Tenn.	123	75	25	12	3	8	10
W.S. CENTRAL	1,742	1,066	401	171	60	43	61
Austin, Tex.	77	50	15	5	5	2	6
Baton Rouge, La.	21	12	6	3	-	-	1
Corpus Christi, Tex.§	47	36	8	3	-	-	1
Dallas, Tex.	195	103	54	23	8	7	1
El Paso, Tex.	68	44	15	3	3	3	2
Fort Worth, Tex	74	43	17	7	5	2	4
Houston, Tex.§	734	436	169	89	24	16	18
Little Rock, Ark.	70	34	20	6	4	5	2
New Orleans, La.	104	57	27	11	5	4	-
San Antonio, Tex.	210	149	43	12	3	3	13
Shreveport, La.	50	39	9	1	1	-	6
Tulsa, Okla.	92	63	18	8	2	1	7
MOUNTAIN	706	461	147	62	18	18	43
Albuquerque, N. Mex.	86	57	22	5	2	-	4
Colo. Springs, Colo.	55	41	9	3	1	1	8
Denver, Colo.	93	59	13	9	5	7	5
Las Vegas, Nev.	113	76	26	9	2	-	9
Ogden, Utah	25	19	3	1	1	1	8
Phoenix, Ariz.	178	109	35	23	4	7	4
Pueblo, Colo.	23	17	5	1	-	-	-
Salt Lake City, Utah	37	21	11	3	1	1	1
Tucson, Ariz.	96	62	23	8	2	1	4
PACIFIC	2,057	1,335	403	196	59	55	122
Berkeley, Calif.	24	17	4	3	-	-	2
Fresno, Calif.	59	44	10	4	-	1	2
Glendale, Calif.	26	17	5	3	-	-	4
Honolulu, Hawaii	83	57	15	6	2	3	6
Long Beach, Calif.§	94	63	18	8	3	2	13
Los Angeles Calif.	593	372	117	67	22	8	24
Oakland, Calif.§	92	61	18	9	2	2	5
Pasadena, Calif.	24	17	3	2	-	2	-
Portland, Oreg.	160	118	27	3	3	9	5
Sacramento, Calif.	149	93	35	12	5	4	17
San Diego, Calif.	147	81	36	15	9	5	15
San Francisco, Calif.	154	86	34	25	3	6	7
San Jose, Calif.	176	127	32	13	1	3	8
Seattle, Wash.	146	92	25	18	6	5	4
Spokane, Wash.	71	51	13	4	1	2	7
Tacoma, Wash.	59	39	11	4	2	3	3
TOTAL	12,880††	8,300	2,610	1,209	345	401	693

*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.

**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.

††Total includes unknown ages.

§Data not available. Figures are estimates based on average of past available 4 weeks.

Lead Exposure — Continued

children as a consequence of their father's occupational lead exposure. Recent evidence suggests that adverse health effects of lead exposure are associated with lower blood-lead levels than was previously believed, particularly in children <6 years old (*8*). Thus, the levels of lead in the children in this investigation place them at increased risk for persistent neurobehavioral dysfunction.

This investigation illustrates how state and local public health agencies and employers can collaborate to control an occupational health problem. As a result of continued public awareness that many occupational illnesses and injuries can be prevented, state and local health departments have intensified their occupational health surveillance, including programs directed at specific hazards. Through a new program, the Sentinel Event Notification System for Occupational Risks (SENSOR), NIOSH supports demonstration projects in 10 state health departments† to improve state and local capacity to detect and investigate events such as the one described here. The SENSOR program has targeted specific conditions (lead poisoning, carpal tunnel syndrome, occupational asthma, hypersensitivity pneumonitis, silicosis, pesticide poisoning, pneumoconiosis, and occupational burns) for surveillance and follow-up. Information about this program is available from Surveillance Coordinating Activity, Office of the Director, NIOSH, Mailstop D26, CDC, Atlanta, GA 30333.

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†California, Colorado, Massachusetts, Michigan, New Jersey, New York, Ohio, Oregon, Texas, Wisconsin.

Endrin Poisoning Associated with Taquito Ingestion — California

In mid-March 1988, three family members in Orange County, California, became dizzy and nauseated within 1 hour of eating taquitos, a snack consisting of a corn tortilla wrapped around a meat filling. Two of the three subsequently had multiple grand mal seizures. The taquitos, a commercial product sold frozen in sealed plastic bags of 48, had been purchased 5 days earlier.

After receiving the reports of illness, the County of Orange Health Care Agency (COHCA) requested that the product be removed from the shelves of the store where the implicated bag was purchased. Several remaining taquitos from the implicated

Endrin Poisoning – Continued

bag were tested by the U.S. Department of Agriculture (USDA), Food Safety and Inspection Service, and found to contain endrin, a pesticide known to cause seizures. Samples of taquitos removed from the store and tested in USDA laboratories were negative for pesticides. The USDA reviewed the operations of the plant where the taquitos were produced, but no evidence of the pesticide was found. However, 90 cases of taquitos were destroyed by the plant owner as a precautionary measure. The USDA concluded that the poisonings were an isolated incident and closed the case April 20, 1988.

Subsequently, in September 1988, COHCA was informed of a 17-year-old boy who, in mid-March, had four seizures 30 minutes after eating taquitos purchased from the same store. After the seizures in March, he had been diagnosed as epileptic and begun on long-term anticonvulsants. At a hearing to determine the 17-year-old's continued eligibility for a driver's license, the hearing officer remarked that he had presided at a similar case (that of the father of the index family) the week before. (In California, seizures and loss of consciousness are reportable conditions for the purpose of determining eligibility for a driver's license.) This new information implicated a second bag of taquitos and indicated that other illnesses may have been misdiagnosed, resulting in serious medical and social consequences. Therefore, the investigation was reopened by the COHCA and the State of California.

A state-issued press release and a mailing by the store to over 40,000 customers generated 100 calls to the local health department. As a result of this publicity, two additional persons who had suffered seizures ≤12 hours after eating taquitos were identified. All five seizure patients had eaten taquitos purchased from the same discount store within a 5-day period in March. Families other than the index family had no remaining taquitos.

California Department of Food and Agriculture laboratories confirmed the presence of endrin in leftover taquitos from the index family. Endrin was present in the tortillas but not in the meat filling. The store, the manufacturing plant, and the manufacturer's suppliers were thoroughly inspected, but no source of endrin was found.

Because of the limited nature of the outbreak and failure to find evidence of contamination in the plant inspections, the California Department of Health Services suspects deliberate tampering as the cause of the outbreak.

Reported by: T Prendergast, MD, B Peck, Public Health, County of Orange Health Care Agency, Santa Ana; staff, California Dept of Food and Agriculture Laboratories; RJ Jackson, MD, T Slagle, PhD, KW Kizer, MD, DO Lyman, MD, State Epidemiologist, California Dept of Health Svcs. Food Safety and Inspection Svc, US Dept of Agriculture. Div of Field Svcs, Epidemiology Program Office; Div of Environmental Hazards and Health Effects, Center for Environmental Health and Injury Control, CDC.

Editorial Note: Endrin is an extremely toxic chlorinated hydrocarbon pesticide in the family of pesticides that includes DDT, heptachlor, aldrin, and dieldrin. Symptoms of endrin poisoning include dizziness, nausea, tremors, leg weakness, disorientation, and tonic-clonic seizures (*1*). Endrin has been responsible for more than 1200 cases of illness and 45 deaths during outbreaks of foodborne poisonings in Wales (*2*), Qatar and Saudi Arabia (*3*), and Pakistan (*4*).

The Orange County episode is the first reported outbreak of endrin poisoning in humans in the United States. The registration of endrin for agricultural purposes in the United States was withdrawn in 1984 (*5*), and it has not been manufactured in this

Endrin Poisoning – Continued

country since that time. Nevertheless, supplies of endrin remain in this country, and other countries continue to use and manufacture the chemical.

Pesticides can cause serious illness even among nonoccupationally exposed persons. Pesticide-related illness from a commercial product should prompt a rapid and aggressive investigation to identify cases, sources, and appropriate control measures including, if necessary, a product recall.

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Perspectives in Disease Prevention and Health Promotion

Dental Caries in Schoolchildren – Utah

During fall 1987, as part of routine surveillance of oral health status, the Dental Health Bureau, Utah Department of Health, surveyed Utah schoolchildren aged 8–12 years. The survey showed differences in histories of dental caries among three groups of children: those who had received optimal levels of systemic fluoride for their entire lives, for some portion of their lives, or not at all.

The stratified sample of 1507 children was selected so that all 12 health districts of the state were represented in percentages equal to the student population. Within each health district, classrooms were selected randomly. Parents completed consent forms that included questions about their children's residential history and other fluoride-related questions, e.g., daily use of supplements. The 957 children whose parents/guardians had completed consent forms (64% of all pupils in the selected classrooms) were examined by a dentist; for 938, sufficient information was available from the parents/guardians for investigators to estimate lifetime fluoride histories.

Children were categorized as having had lifetime fluoride, partial fluoride, and no fluoride. The 110 children in the lifetime-fluoride category reportedly had received optimally fluoridated water or a daily fluoride supplement at home for at least 6 months of every year of life or for all but 1 year of life. The 563 children in the partial-fluoride category reportedly had consumed fluoridated water or a daily fluoride supplement at home for as long as 6 months in a single year but not long enough to meet the criteria for the lifetime-fluoride category. The 265 children in the no-fluoride category reportedly had never received fluoridated water or a daily fluoride supplement at home for as long as 6 months in a single year.

One dentist completed all clinical examinations following the protocol and criteria used for prevalence surveys conducted by the National Institute of Dental Research (NIDR) (*1*). Using artificial light, compressed air, mirror, and explorer, the examiner assessed the decayed, missing, and filled surfaces of permanent teeth (DMFS) and

Dental Caries — Continued

the presence of dental sealants on occlusal surfaces of permanent teeth. A child was classified as having sealants if at least one permanent tooth had an intact sealant.

Examination showed dental caries in the permanent dentitions of 477 (50%) of the children (Table 1). Of those who received no fluoride, 54% had experienced tooth decay, compared with 36% of those with lifetime fluoride exposure. Among those surveyed, children with no exposure to systemic fluoride were more likely to have had dental caries in permanent teeth than children with a lifetime exposure to fluoride (prevalence ratio [PR] = 1.5, 95% confidence interval [CI] = 1.1–1.9).

A similar pattern occurred when severity of dental disease was considered. Almost one third of the children with no systemic fluoride exposure but one fifth of children with lifetime exposure had experienced ≥3 DMFS (PR = 1.6, 95% CI = 1.1–2.4). Mean DMFS for the no-fluoride group (2.2) was 41.5% greater than that for the lifetime-fluoride group (1.3). In the entire sample, 31% of the children had dental sealants on one or more permanent teeth.

For children aged 8, 10, and 12 years, caries prevalence was higher for Utah than for a national sample (Figure 1) (*2*). Utah children with lifetime fluoride were least likely to have had dental caries in their permanent teeth (Figure 2). Although children who had received fluoride for a portion of their lives had a higher prevalence of dental caries than the lifetime-fluoride group, their caries prevalence was lower than that of children who had never received systemic fluoride.

Reported by: CM Fitzgerald, DDS, KL Zinner, Dental Health Bureau, CR Nichols, MPA, State Epidemiologist, Utah Dept of Health. Dental Disease Prevention Activity, Center for Prevention Svcs, CDC.

Editorial Note: Data from recent oral health surveys in national and state-based samples of children show a lower prevalence of dental caries than do those from similar earlier surveys (*1–3*; North Carolina Department of Human Resources, unpublished data). In general, the decreased prevalence has been attributed to increased use of fluorides, both systemic and topical, during the past several decades (*4–6*). As a public health measure, water fluoridation reaches all segments of the community cost-effectively.

In Utah, fluoridation of community water supplies is limited; only 2% of the population using public water supplies consumes optimally fluoridated water (*7*). For that reason, the Utah Department of Health urges physicians and dentists to prescribe fluoride supplements, which children should take daily during the first 14 years of life.

TABLE 1. Selected characteristics of schoolchildren, aged 8–12 years, by fluoride status — Utah, 1987

Characteristic	Entire sample (n = 957)		Fluoride status†					
			Lifetime (n = 110)		Partial (n = 563)		None (n = 265)	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Children who have had dental caries in their permanent teeth	477	(50)	40	(36)	281	(50)	144	(54)
Children with 3 or more DMFS*	258	(27)	23	(21)	147	(26)	81	(31)
Children with dental sealants	299	(31)	40	(36)	174	(31)	75	(28)

*DMFS = decayed, missing, and filled surfaces.
†The fluoride status was unknown for 19 children.

Dental Caries — Continued

In a recent survey, >90% of responding physicians reported that they prescribed fluoride supplements for children (*8*).

The fluoride histories and oral health status of children without consent forms (one third of the original sample) remain unknown. Among Utah respondents, 12% of children reportedly received systemic fluoride for their entire lifetimes; this proportion included those who consumed optimally fluoridated water as well as those who reportedly received supplements routinely. Approximately 60% of the sample were

FIGURE 1. Prevalence of dental caries in permanent teeth of children, by age — United States and Utah, 1987

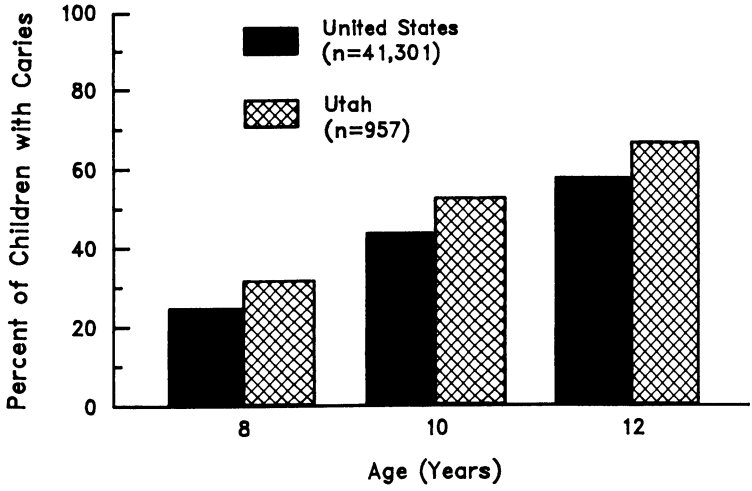
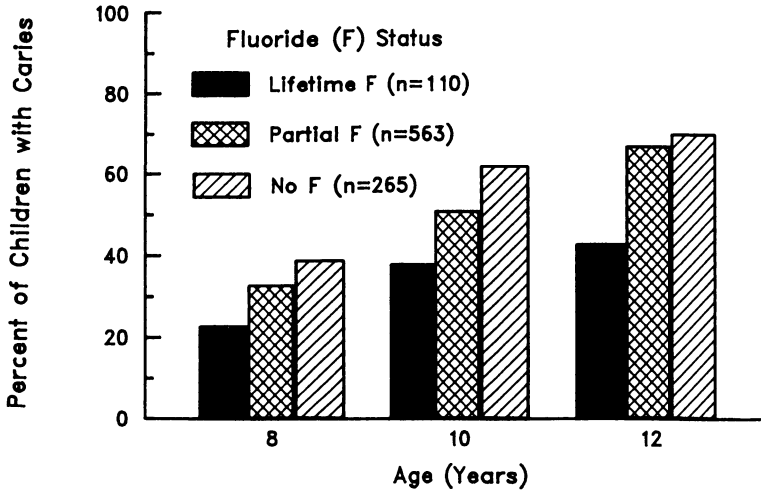


FIGURE 2. Prevalence of dental caries in permanent teeth of children, by age and fluoride status — Utah, 1987



Dental Caries — Continued

classified in the partial-fluoride category, and 28% of the Utah children had not received systemic fluoride for as long as 6 months. Children in all three groups may have ingested fluoride by swallowing small amounts of fluoride toothpaste (*4,9,10,*) or by consuming soft drinks and other food products manufactured in optimally fluoridated areas.

It is likely that the prevalence of dental caries in Utah would be higher had the health department not emphasized the need for dental sealants. The proportion of Utah children with dental sealants (31%) was higher than that found in recent surveys in Ohio (8%) (*3*) and North Carolina (10%) (North Carolina Department of Human Resources, unpublished data). Utah children without fluoride exposure were less likely to have sealants. Children from higher socioeconomic groups may be overrepresented in the lifetime-fluoride group because they would have been more likely to receive fluoride supplements throughout their lifetimes and to receive professional dental care, including the application of dental sealants.

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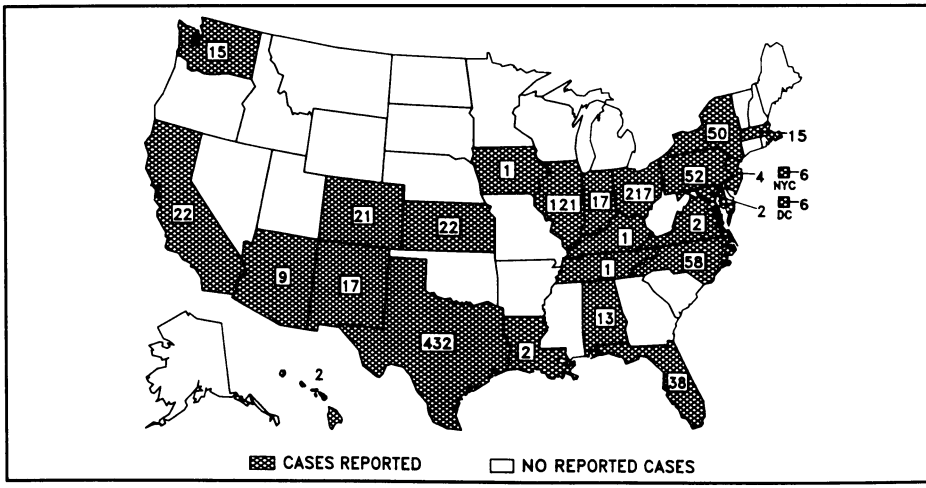
FIGURE I. Reported measles cases — United States, weeks 15–18, 1989



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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. The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: Editor, *Morbidity and Mortality Weekly Report*, Centers for Disease Control, Atlanta, Georgia 30333; telephone (404) 332-4555.

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