

MORBIDITY AND MORTALITY WEEKLY REPORT

March 29, 1991 / Vol. 40 / No. 12

- 193 Fatal Pediatric Poisoning from Leaded Paint – Wisconsin, 1990
- 195 Characteristics of, and HIV Infection among, Women Served by Publicly Funded HIV Counseling and Testing Services – United States, 1989–1990
- 204 Tuberculosis Outbreak on Standing Rock Sioux Reservation – North Dakota and South Dakota, 1987–1990

Epidemiologic Notes and Reports

Fatal Pediatric Poisoning from Leaded Paint – Wisconsin, 1990

Although fatal lead poisoning among children occurs rarely in the United States, it represents a medical and public health emergency. This report summarizes the investigation of a child who died from poisoning associated with ingestion of lead-based paint.

On September 12, 1990, a 28-month-old Wisconsin boy was admitted to a hospital with a 4-day history of lethargy and reduced appetite. Although the child had no known past medical problems, his parents reported that he had eaten flaking paint. On initial neurologic examination, the child had extreme lethargy with facial palsy and gasping respirations, consistent with lead encephalopathy; laboratory results revealed severe lead toxicity and hematologic abnormalities (blood lead level [BLL] 144 μ g/dL; erythrocyte protoporphyrin level 593 μ g/dL; hemoglobin 8.1; and basophilic stippling). Despite chelation therapy with British antilewisite and calcium disodium edetate (CaNa₂-EDTA), the child developed seizures, became comatose, and died 26 hours after admission. An autopsy showed massive cerebral edema with uncal herniation. The intestines contained multiple roundworms (*Ascaris lumbricoides*) and flake-like material consistent with paint chips. Radiographs revealed prominent epiphyseal lines in the lower extremities, consistent with chronic lead exposure.

On September 20, staff from the Wisconsin Division of Health and the Waukesha County Health Department inspected the child's residence. The child and his parents had lived for at least 4 months on the second floor of a two-story, nonresidential structure built in 1923. The interior paint was badly deteriorated with paint chips visibly flaking from the walls and accumulating on floors, windowsills, and stairs. Eleven paint chip samples from the apartment ranged from 0.2% to 33.1% lead by weight (average: 9.1%); the U.S. Consumer Product Safety Commission (CPSC) permits a maximum of 0.06% lead in new residential paint.* House dust from the child's bedroom floor contained 3900 μ g lead/ft², and dust from a windowsill above the child's bed contained 31,128 μ g lead/ft².

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES / PUBLIC HEALTH SERVICE

^{*16} Code of Federal Regulations, part 1303. Ban of lead-containing paint and certain consumer products bearing lead-containing paint.

Lead Poisoning - Continued

higher than those proposed in recent guidelines issued by the U.S. Department of Housing and Urban Development (1), which recommend the maximum dust lead levels permissible before reoccupancy of a unit following lead paint abatement.

After the child's death, the parents moved and were unavailable for follow-up. The landlord has blocked access to the second floor and plans to eliminate the lead paint hazards in the building.

Reported by: J Schirmer, MS, HA Anderson, MD, State Environmental Epidemiologist, Div of Health, Wisconsin Dept of Health and Social Svcs; LA Saryan, PhD, Industrial Toxicology Laboratory, West Allis Memorial Hospital, West Allis, Wisconsin. Lead Poisoning Prevention Br, Center for Environmental Health and Injury Control, CDC.

Editorial Note: Lead encephalopathy usually is associated with a BLL >100 μ g/dL, although it has been reported at BLLs as low as 70 μ g/dL (2). As in this report, children with acute lead encephalopathy often have a recent history of prodromal symptoms, including anorexia, apathy, decreased play activity, hyperirritability, aggressiveness, poor coordination, and sporadic vomiting. Because lead encephalopathy in a child can rapidly progress to death, either a BLL \geq 70 μ g/dL in a child or the onset of encephalopathy constitutes an acute medical emergency.

At least four factors may account for the dramatic decline in the incidence of acute lead encephalopathy and childhood deaths from lead poisoning since the 1960s (3), including 1) increased screening of children at risk, 2) recognition of toxicity before the onset of life-threatening symptoms, 3) improvements in the treatment for lead poisoning, and 4) reduction of lead exposure from certain environmental sources. Although childhood deaths from poisoning associated with exposure to lead-based paint are now rare in the United States (4) (the most recently reported lead-based paint-associated death occurred in the mid-1970s [5]), subclinical toxicity is a widespread and persistent public health problem (6). BLLs as low as 10 μ g/dL, once considered safe, are now known to adversely affect cognitive development and behavior in children (7), with potentially long-term sequelae (8). In 1984, an estimated 3–4 million U.S. children had BLLs \geq 15 μ g/dL (6).

The primary source of high-dose lead exposure among children in U.S. urban areas is lead-based paint (6). Although CPSC banned lead-based paint for residential use in 1978, an estimated 12 million children <7 years of age reside in homes containing previously applied lead-based paint (6). Interior paints used before 1940 contained as much as 50% lead (9). Although children can ingest lead directly by eating paint chips, ingestion of lead-contaminated house dust and soil during normal mouthing and exploratory behaviors contributes substantially to elevating BLLs (10). The child reported in Wisconsin appeared to have been ingesting paint chips and was exposed to highly contaminated house dust.

All cases of lead poisoning are preventable. A national health objective for the year 2000 is to reduce the prevalence of children aged 6 months through 5 years with BLLs >15 μ g/dL to less than 500,000 and the prevalence of those with BLLs >25 μ g/dL to zero (*11*). Recently, several federal agencies responsible for housing, health, and the environment have focused attention on this problem and have set goals to abate lead-based paint in privately owned housing (*12*), reduce the number of children with elevated BLLs (*13*), and promote national efforts to eliminate childhood lead poisoning (*14*).

References

 Office of Public and Indian Housing. Lead-based paint: interim guidelines for hazard identification and abatement in public and Indian housing. Washington, DC: US Department of Housing and Urban Development, Office of Public and Indian Housing, 1990.

Vol. 40 / No. 12

MMWR

Lead Poisoning – Continued

- Piomelli S, Rosen JF, Chisolm JJ, Graef JW. Management of childhood lead poisoning. J Pediatr 1984;105:523–32.
- 3. Lin-Fu JS. The evolution of childhood lead poisoning as a public health problem. In: Chisolm JJ, O'Hara DM, eds. Lead absorption in children: management, clinical and environmental aspects. Baltimore: Urban and Schwarzenberg, Inc, 1982:1–10.
- NCHS. Vital statistics mortality data, multiple cause-of-death detail [machine-readable public-use data tape]. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, 1979–1987.
- Klein R. Lead poisoning. In: Barness LA, ed. Advances in pediatrics. Vol 24. Chicago: Year Book Medical Publishers, 1977:103–32.
- Agency for Toxic Substances and Disease Registry. The nature and extent of lead poisoning in children in the United States: a report to Congress. Atlanta: US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, 1988.
- Mushak P, Davis JM, Crocetti AF, Grant LD. Prenatal and postnatal effects of low-level lead exposure: integrated summary of a report to the US Congress on childhood lead poisoning. Environ Res 1989;50:11–36.
- Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood, an 11-year follow-up report. N Engl J Med 1990;322:83–8.
- 9. National Academy of Sciences. Report of the Ad Hoc Committee to Evaluate the Hazard of Lead in Paint. Washington, DC: US Consumer Product Safety Commission, 1973:3.
- Charney E, Sayre J, Coulter M. Increased lead absorption in inner city children: where does the lead come from. Pediatrics 1980;65:226–31.
- 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.
- 12. Office of Policy Development and Research. Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing: a report to Congress. Washington, DC: US Department of Housing and Urban Development, Office of Policy Development and Research, 1990.
- Environmental Protection Agency. Strategy for reducing lead exposures. Washington, DC: US Environmental Protection Agency, 1991.
- 14. CDC. Strategic plan for the elimination of childhood lead poisoning. Atlanta: US Department of Health and Human Services, Public Health Service, 1991.

Current Trends

Characteristics of, and HIV Infection among, Women Served by Publicly Funded HIV Counseling and Testing Services – United States, 1989–1990

In 1990, the number of reported acquired immunodeficiency syndrome (AIDS) cases among women in the United States exceeded 15,000, an increase of 34% from 1989 (1). Public health surveillance of the human immunodeficiency virus (HIV)/AIDS epidemic has included monitoring of publicly funded voluntary counseling and testing (CT) programs, such as the voluntary client record system (representing 43% of all reported CT visits) that collects detailed information for each CT visit. This report summarizes findings based on information from the client record system for women who received public CT services during 1989 and 1990.

During 1989 and 1990, women accounted for approximately 1 million (48%) of the 2.2 million tests reported by all CT programs. Of these, 47% of tests were from white women; 35%, black women; and 16%, Hispanic women; in comparison, these groups account for 79%, 11%, and 6%, respectively, of the U.S. female population (2).

HIV Infection - Continued

Approximately 20,000 (2%) tests were positive for HIV antibody, including 0.9% among whites, 3.3% among blacks, and 3.7% among Hispanics.

Nearly all CT visits by women occurred in either sexually transmitted disease (STD) clinics (29%), HIV CT sites (29%), or women's (family planning and prenatal) clinics (28%). Of seropositive tests, 40% were from CT sites, 29% from STD clinics, and 8% from women's clinics. Drug-treatment centers accounted for 4% of all tests and 7% of all positive tests; however, the seropositivity rate among tests from drug-treatment centers (3.7%) was higher than tests from other sites (CT sites, 3.0%; STD clinics, 2.2%; and women's clinics, 0.7%) (Table 1).

Most (80%) women who were tested did not report HIV risk behavior (including 81% of blacks, 76% of Hispanics, and 69% of whites) (Figure 1, page 203). Of women who did not report HIV risk behavior, 1.0% were seropositive; however, seropositivity varied by race and ethnicity (1.9%, 1.0%, and 0.3% in black, Hispanic, and white women, respectively). Of seropositive women, 65% reported a specific risk behavior; 35% reported no risk behavior (22% among whites; 24%, Hispanics; and 44%, blacks).

Intravenous (IV)-drug use was reported by 8% of all women, compared with 31% of those who were seropositive. Among black, Hispanic, and white women who identified themselves as IV-drug users, seropositivity was 16.7%, 15.0%, and 3.8%, respectively. Of seropositive women, IV-drug use was reported by 43% and 32% of white and Hispanic women, respectively, compared with 26% of black women.

Women who were sex partners of persons at risk accounted for 13% of those tested, but 27% of all seropositive tests (20%, 30%, and 40% of all seropositive black, white, and Hispanic women, respectively). The overall seropositivity among women who were sex partners of persons at risk was 4.3% (1.5%, 8.2%, and 3.6% for whites, blacks, and Hispanics, respectively).

Reported by: HIV prevention programs of state and local health depts. Program Development/ Technical Support Section, Div of STD/HIV Prevention and Office of the Deputy Director (HIV), Center for Prevention Svcs, CDC.

Editorial Note: This assessment of findings at CT sites underscores the disproportionate impact of the HIV epidemic on minority populations in the United States (1). However, because these data reflect characteristics of women who receive services at public clinics (Table 1), they cannot be considered representative of all U.S. women. In addition, because these data are collected in service delivery settings, data regarding risk may be less reliable than those obtained during epidemiologic investigations, particularly for persons who initially report no HIV risk behavior.

Nearly half the HIV tests reported by publicly funded CT programs are from women, among whom blacks and Hispanics are disproportionately represented. Although 17% of all women in the United States are black or Hispanic (2), blacks and Hispanics accounted for 73% (52% and 21%, respectively) of reported AIDS cases among women (1). Because of the high prevalence of HIV infection and AIDS among these groups, community-based outreach programs should actively encourage women – especially minority women – to seek HIV-prevention services.

Sexual transmission of HIV is associated with certain STDs. In the United States, syphilis incidence is 50-fold greater among black women and 10-fold greater among Hispanic women than among white women (3). HIV infection and transmission have been epidemiologically linked with genital ulcer disease, including syphilis (3–5), suggesting that genital ulcer disease facilitates HIV transmission.

HIV Infection -

Continued

						Rac	e									
		White			Black			Hispanic			Other		Total			
	No.	HIV po	ositive	No.	HIV po	ositive	No.	HIV positive		No.	HIV positive		No.	HIV positive		
HIV test site	tests	No.	(%)	tests	No.	(%)	tests	No.	. (%)	tests	No.	(%)	tests	No.	(%)	
HIV CT site	72,844	859	(1.2)	21,457	1,406	(6.6)	20,763	1,242	(6.0)	2,170	36	(1.7)	117,234	3,543	(3.0)	
STD* clinic	42,569	304	(0.7)	62,454	1,779	(2.8)	11,285	466	(4.1)	1,287	16	(1.2)	117,595	2,565	(2.2)	
Drug-treatment center	8,703	186	(2.1)	5,674	344	(6.1)	2,663	102	(3.8)	210	1	(0.5)	17,250	633	(3.7)	
Family planning clinic	29,801	54	(0.2)	16,625	166	(1.0)	12,877	58	(0.5)	703	4	(0.6)	60,006	282	(0.5)	
Prenatal/ Obstetric clinic	21,029	51	(0.2)	23,328	348	(1.5)	9,249	62	(0.7)	1,163	8	(0.7)	54,769	469	(0.9)	
Prison	1,484	53	(3.6)	1,689	152	(9.0)	327	13	(4.0)	35	4	(11.4)	3,535	222	(6.3)	
Other	14,403	178	(1.2)	12,589	744	(5.9)	9,200	160	(1.7)	975	42	(4.3)	37,167	1,124	(3.0)	
Total	190,833	1,685	(0.9)	143,816	4,939	(3.4)	66,364	2,103	(3.2)	6,543	111	(1.7)	407,556	8,838	(2.2)	

 TABLE 1. Summary of HIV test results among women at publicly funded HIV counseling and testing (CT) sites, by race and testing site – United States, 1989–1990

*Sexually transmitted disease.

(Continued on page 203)

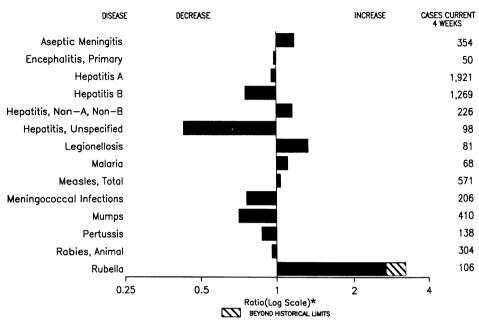


FIGURE I. Notifiable disease reports, comparison of 4-week totals ending March 23, 1991, with historical data - United States

*Ratio of current 4-week total to the mean of 15 4-week totals (from previous, comparable, and subsequent 4-week periods for the past 5 years). The point where the hatched area begins is 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 23, 1991 (12th Week)

	Cum. 1991		Cum. 1991
AIDS	9,984	Measles: imported	28
Anthrax	· ·	indigenous	1,232
Botulism: Foodborne	5	Plague	-
Infant	10	Poliomyelitis, Paralytic*	
Other	. 2	Psittacosis	19
Brucellosis	12	Rabies, human	-
Cholera	-	Syphilis, primary & secondary	9.224
Congenital rubella syndrome	4	Syphilis, congenital, age < 1 year	
Diphtheria	1 1	Tetanus	-
Encephalitis, post-infectious	14	Toxic shock syndrome	86
Gonorrhea	122,772	Trichinosis	
Haemophilus influenzae (invasive disease)	909	Tuberculosis	4,033
Hansen Disease	22	Tularemia	17
Leptospirosis	21	Typhoid fever	69
Lyme Disease	1,077	Typhus fever, tickborne (RMSF)	15
Lynno Bibbabb	1 1,077	(NIVISE)	

*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.

		-											
	AIDS	Aseptic Menin-	Encep	halitis	Gane	orrhea	н	epatitis	(Viral), by		Legionel-	Lyme	
Reporting Area		gitis	Primary	Post-in- fectious	Gond	nnea	A	В	NA,NB	Unspeci- fied	losis	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	9,984	1,118	124	14	122,772	158,636	5,678	3,472	744	315	252	1,077	
NEW ENGLAND	436	53	8		3,667	4,570	136	215	47	9	25	40	
Maine N.H.	15 11	3 3	3	-	23 72	61 58	4 15	3 6	2 2	-	- 1	2	
Vt. Mass.	6 243	5 18	3	:	14 1,459	18 1,657	6 74	1 179	1 40	-7	23	1 27	
R.I.	16	21	-	-	249	238	19	10	1	2	23	10	
Conn.	145	3	2	-	1,850	2,538	18	16	1	-	-	-	
MID. ATLANTIC Upstate N.Y.	2,949 470	156 72	6 5	4 3	13,438 2,591	21,816 3,242	422 273	296 146	50 32	9 3	64 24	876 761	
N.Y. City N.J.	1,613	9	-	•	3,197	9,516	25	6	-	-	3	-	
Pa.	569 297	- 75	1	1	2,643 5,007	3,550 5,508	49 75	77 67	8 10	6	7 30	115	
E.N. CENTRAL	597	193	31	4	20,622	30,753	542	409	168	16	44	40	
Ohio Ind.	102 48	70 22	8 5	1	5,456 2,462	9,517 2,621	117 102	98 52	45 1	6	22 3	26	
III.	307	25	4	2	6,658	9,076	137	21	3		1	-	
Mich. Wis.	93 47	68 8	13 1	-	5,402 644	7,551 1,988	91 95	141 97	29 90	10	12 6	14	
W.N. CENTRAL	313	76	7		6,569	8,435	781	140	76	6	14	6	
Minn.	67	12	5	-	696	1,060	87	11	5	ĩ	2	2	
lowa Mo.	22 160	20 28	-		454 4,125	649 4,876	18 176	7 108	5 65	- 4	- 7	4	
N. Dak.	18	-	-		11	39	12	2	-	ī	-		
S. Dak. Nebr.	14	47	2		102 404	49 381	344 122	1 8	-		3 2	-	
Kans.	32	5	-	-	777	1,381	22	3	1		-	-	
S. ATLANTIC	2,301	261	25	5	39,045	43,658	388	811	98	54	32	31	
Del. Md.	22 276	6 28	- 4		540 3,907	554 4,719	3 92	13 107	2 20	1 5	- 8	8 11	
D.C.	163	10	-	-	2,578	2,258	26	24	1	1	-	-	
Va. W. Va.	186 10	47 2	5 1	-	3,571 295	4,137 300	47 7	63 22	6 1	34	3	7	
N.C.	102	32	10		7,645	7,533	59	159	39	3	6	1 4	
S.C. Ga.	80 353	10 20	3	1	2,836 10,020	3,777	12 46	192	15	2	7	-	
Fla.	1,109	106	2	4	7,653	9,601 10,779	46 96	92 139	4 10	8	2 6	-	
E.S. CENTRAL	237	76	5		11,870	13,215	53	272	81	2	19	18	
Ky. Tenn.	34 70	20	2 2		1,143	1,520	8	51	5	2	11	11	
Ala.	62	16 27	1	-	4,265 3,448	3,989 4,690	32 12	190 31	73 3	-	6 2	4 3	
Miss.	71	13	-	-	3,014	3,016	1	•	-	-	-	-	
W.S. CENTRAL Ark.	901 42	68 26	9 1	:	13,839 1,718	15,883	699 102	307 17	19	44	9	13	
La.	131	20	1		3,082	2,141 2,867	102 36	60	1 1	2 1	1 3	7	
Okla. Tex.	27 701	1 33	3 4	-	1,477 7,562	1,456 9,419	96 465	75 155	13 4	6	4	6	
MOUNTAIN	302	50		1	2,411	3,445	1.035	239	4 34	35 66	1 26	2	
Mont.	5	2	-	-	19	29	40	22	1	3	1	-	
ldaho Wyo.	3 5	-	-	-	37 31	21 38	18 57	29 3	-		3	- 2	
Colo.	128	17	1	1	436	985	87	37	8	9	3	-	
N. Mex. Ariz.	25 56	6 17	-7	:	262 1,045	252 1,354	319 352	41 53	4	21 28	1	-	
Utah	19	2		-	89	110	89	10	6	20	4	-	
Nev.	61	6	-	-	492	656	73	44	11	-	5	-	
PACIFIC Wash.	1,948 120	185	25	-	11,311 914	16,861 1,602	1,622 148	783 113	171 34	109 6	19 1	51	
Oreg.	40	-			441	614	97	72	34 27	2	1	-	
Calif. Alaska	1,737 5	168 4	25	-	9,571	14,225	1,338	577	102	100	16	51	
Hawaii	46	13		-	193 192	304 116	30 9	6 15	7	1	1	-	
Guam	-	-	-	-	-	57	-	-	-	-	-	-	
P.R. V.I.	417 1	49	•	1	105 136	296 112	25	109	21	13	-	-	
Amer. Samoa	-	-	-	-	•	35	-	2	-	-	-	-	
C.N.M.I.	-	•	-	-	-	44	-	-	-	-	-	-	

TABLE II. Cases of selected notifiable diseases, United States, weeks ending March 23, 1991, and March 24, 1990 (12th Week)

N: Not notifiable

U: Unavailable

C.N.M.I.: Commonwealth of the Northern Mariana Islands

	Malaria		Meas	les (Rui	oeola)		Menin- gococcal	Mumps		Ι	Pertussi	•	Rubella		
Reporting Area	Malaria	Indigenous		Impo	orted*	Total	Infections						1		
	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	204	171	1,232	6	28	4,187	554	105	848	38	487	639	22	153	131
NEW ENGLAND Maine	13	1	4	-	2	101 27	49 4	:	10	19 -	55 5	80 1	:	1	2
N.H.	1		-	-	•	7	6	-	2	-	11	7	-	1	-
Vt. Mass.	1 7	1	3	-	-	1 2	8 24	-	-	18	1 36	2 65	:		-
R.I.	3	-	-	-	- 2	22	;		2		-	-	-	-	1
Conn.	1	- 102	1 545	-		42 444	7 56	- 7	6 62	1 2	2	5	-		1
MID. ATLANTIC Upstate N.Y.	17 6	102	545 1	-	:	229	31	6	35	1	54 27	148 117	21 20	75 68	2 1
N.Y. City	3	-	60	:	-	42	2 9	-	- 4	•	1	-	-	-	-
N.J. Pa.	5 3	101	85 399	-		18 155	9 14	1	4 23	1	26	11 20	1	7	1
E.N. CENTRAL	15		20	-	2	1,874	81	5	67	1	88	184	1	6	14
Ohio	4		-		ī	136	30	-	-	-	34	26	-	-	
Ind. III.	1 3	U	-	U	:	119 823	9 21	U	3 35	U	17 15	31 65	U	1 2	13
Mich.	7	-	18	-	-	265	16	5	26	1	18	27	1	3	-
Wis.	-	-	2	-	1	531	5	-	3	•	4	35	-	-	1
W.N. CENTRAL	3	1	2 1	-	1	101 28	23 6	-	39	1	34	20	-	3	-
Minn. Iowa	1	1	1	-	1	28	1	2	2 7	-	11 4	2	:	2	
Mo.	2	-	-	-	-	51	11	-	5	-	13	14	-	1	-
N. Dak. S. Dak.	-	-		-	:	-	1	:	-	:	1	1		-	
Nebr.	-	-	-	-	-	1	2	•	3	-	4	1	-	-	-
Kans.	-	-	-	-	-	-	2	-	22	1	1	1	•	-	-
S. ATLANTIC Del.	49	59 2	115 8	2	8	238 4	102	77 1	312 3	2	32	54 2	•	8	9
Md.	16	19	34	-	-	28	13	11	81	-	6	19		8	
D.C. Va.	3 8	- 6	- 7	- 2§	2	1 11	- 8	:	4	-	- 4	1	-	-	-
W. Va.	1	-	<u>'</u>	- 23	-	6	4	2	18 8	1	6	4		-	-
N.C. S.C.	1 4	-	12	-	2	3 1	25	3	65	-	7	9	-	-	-
Ga.	4	-	12	-	-	6	15 19	2	43 12	1	- 6	3 7		-	-
Fla.	12	32	54	-	6	178	18	58	78	-	3	4	-	-	9
E.S. CENTRAL	2	4	4	-	-	38	46	-	23	1	16	19	-	-	1
Ky. Tenn.	1	4	4	-		17	21 12	2	12	1	- 8	7	:	-	1
Ala.	1	-	-	-	-	4	13	-	2	-	8	10	-	-	-
Miss.	-	-	-	-	-	17	-	-	9	•	-	2	-	-	-
W.S. CENTRAL Ark.	12 1	-	-	-	5 5	302	32 7	7	121	-	12	5	-	1	-
La.	2	-	-	-	-	-	9	1	20 9	-	7	1	-	1	-
Okla. Tex.	1 8	-	-	-	:	39 263	4 12	6	1 91	-	5	4	-	-	-
MOUNTAIN	9	2	100	4	8	141	27			•		-	-	-	-
Mont.	1	-	- 100	4	-	141	4	8	46		77	59	-	1	8 5
Idaho	-	-	-	-	1	5	5	-	2	-	17	4	-	-	3
Wyo. Colo.	3		-	-	1	14	1	7	2 14	-	3 27	- 40	-	-	-
N. Mex.	1	2	68	-	2	34	4	N	N	-	12	2	-	-	-
Ariz. Utah	4		25	- 4†	4	63	5	1	20 8	-	8 10	7 3	-	-	-
Nev.	-	-	7	-	-	24	4	-	-	-	-	3	-	1	-
PACIFIC	84	2	442	-	2	948	138	1	168	12	119	70	-	58	95
Wash. Oreg.	6 1	- 1	- 5	-	:	33 91	12 13	N	40 N	6 4	22 20	20 7	-	-	-
Calif.	75		433	-	2	776	109	-	118	2	54	38	-	57	- 91
Alaska	2	1	- 4	2		46 2	4	1	4 6	-	4 19	- 5	-	•	- 4
Hawaii	2		4		-	2	-		U		19	D	-	1	4
Guam P.R.	-	U	-	U	1	101	11	U	- 5	U	6	4	U	1	-
V.I.	-	U	-	U	-	-	-	U	3	U	-	-	U	-	-
Amer. Samoa C.N.M.I.	-	U U	-	U U	:	-	-	U U	-	U U	-	:	U U	:	-
G.N.W.I.	-									<u> </u>		-		-	

TABLE II. (Cont'd.) Cases of selected notifiable diseases, United States, weeks ending March 23, 1991, and March 24, 1990 (12th Week)

*For measles only, imported cases includes both out-of-state and international importations. N: Not notifiable U: Unavailable [†]International [§]Out-of-state

Reporting Area		philis & Secondary)	Toxic- shock Syndrome	Tuber	culosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal	
	Cum. 1991	Cum. 1990	Cum. 1991	Cum. 1991	Cum. 1990	Cum. 1991	Cum. 1991	Cum. 1991	Cum. 1991	
UNITED STATES	TES 9,224 10,625 86		86	4,033	4,617	17	69	15	928	
NEW ENGLAND	256	444	6	101	85	-	6	2	1	
Maine N.H.	- 2	3 28	3 1	-	1			-	1	
Vt.	1	1	-	1	2			-	-	
Mass. R.I.	128 12	160 1	2	46 16	35 20	-	6	2	-	
Conn.	113	251	-	38	27			-	-	
MID. ATLANTIC	1,596	2,220	13	976	1,173	-	9	-	327	
Upstate N.Y. N.Y. City	103 733	141 1,238	7	43 653	118 753	-	3 2	-	101	
N.J.	297	365	-	183	157	-	4	-	146	
Pa.	463	476	6	97	145	-	•	-	80	
E.N. CENTRAL Ohio	888 129	626 110	15	488 78	433 57	1	7	-	14	
Ind.	22	6	10	18	23		1	-	3	
111.	391	240	2	278	216	:	:	-	3	
Mich. Wis.	221 125	170 100	3	85 29	121 16	1	5 1	-	2 6	
W.N. CENTRAL	164	87	17	111	104	3	2		117	
Minn.	19	31	7	19	19	-	2	-	43	
lowa Mo.	18 98	7 39	5 4	21 44	10 46	3	-	-	23 2	
N. Dak.		1	-	2	40	-	-	-	10	
S. Dak. Nebr.	1	- 2	1	11 3	4	•	•	-	29	
Kans.	27	7	-	11	12	-	-	-	4 6	
S. ATLANTIC	2,963	3,439	5	662	817	1	13	10	241	
Del.	31	51	ī	7	13	-	-	-	35	
Md. D.C.	257 155	289 162	-	58 40	73 21	-	5 1	1	85 3	
Va.	253	182	1	62	63	-	3	-	38	
W. Va. N.C.	4 448	4 404	- 3	22 91	13 101	-	1	- 8	20	
S.C.	365	214		75	109	-		-	18	
Ga. Fla.	732 718	767 1,366	-	131 176	125 299	1	2 1	1	36	
E.S. CENTRAL	1,018	901	4	293	305	2	1	-	6	
Ky.	17	18	4	293	305	1		2 1	20 4	
Tenn. Ala.	386 354	285 331	2	42 95	63	1	-	-	7	
Miss.	354 261	267	1	95 83	105 48	-		1	9	
W.S. CENTRAL	1,522	1,691	4	408	569	5	1	1	109	
Ark.	69	119	2	40	59	3	-	-	7	
La. Okla.	544 36	518 52	2	66 15	113 44	2	1	1	3 37	
Tex.	873	1,002	-	287	353	-		-	62	
MOUNTAIN	129	185	10	112	95	4	3	-	9	
Mont. Idaho	1 3	4	-	2	4	3	-	-	4	
Wyo.	3 1	4	-	1	1	1		-	1	
Colo.	15	15	1	6	6	-		-	1	
N. Mex. Ariz.	6 84	11 128	3 3	5 74	18 50		2		1 2	
Utah	3	2	3	13	-	-	-	-	-	
Nev.	16	25	-	11	15	-	1	•	-	
PACIFIC Wash.	688 33	1,032 105	12 1	882 59	1,036 61	1	28	-	90	
Oreg.	24	24	-	15	22	-	1	-	1	
Calif. Alaska	628 2	887 5	11	750	900 17	-	26	-	88	
Hawaii	1	11	-	10 48	36	-	1	-	1	
Guam	-	-	-	-	12	-	-	-	-	
P.R.	75	122	-	38	29	-	-	-	7	
V.I. Amer. Samoa	35	-	-	1	1 5	-	-	-	-	
C.N.M.I.	-	~	-	-	8	-	-		-	

TABLE II. (Cont'd.) Cases of selected notifiable diseases, United States, weeks ending March 23, 1991, and March 24, 1990 (12th Week)

U: Unavailable

	1	All Cau	ises, B	y Age (Years)					All Causes, By Age (Years)						
Reporting Area	All	≥65	45-64	25-44	1-24	<1	P&I** Total	Reporting Area	All	≥65	45-64	_	1-24	<1	P&I** Total	
····	Ages								Ages							
NEW ENGLAND	687 175	483 110	116	61	18	9 3	54	S. ATLANTIC	1,364	861		121	43	35	76	
Boston, Mass. Bridgeport, Conn.	48	32	40 9	15 3	7	2	19 4	Atlanta, Ga.	169	101		16		3 4	11 12	
Cambridge, Mass.	24	17	5	2	-	-	2	Baltimore, Md. Charlotte, N.C.	155 116	93 69		17 11	6 2	2	5	
Fall River, Mass.	27	22	5	-	-	-		Jacksonville, Fla.	132	102		5		ī	8	
Hartford, Conn.	69	45		11	3	1	3	Miami, Fla.	94	59) 18	10	5	2	-	
Lowell, Mass. Lynn, Mass.	25 24	17 19	2 3	4	2	-	4	Norfolk, Va.	72	41				3	5	
New Bedford, Mass.	24	23		1		-	1	Richmond, Va. Savannah, Ga.	87 50	49 31		8		9 2	6 5	
New Haven, Conn.	61	35		10	1	1	2	St. Petersburg, Fla.	50 76	62		2		1	2	
Providence, R.I.	52	37	7	6	-	2	6	Tampa, Fla.	168	119		9		3	13	
Somerville, Mass.	5	5	-	-	;	-	-	Washington, D.C.	224	119				5	9	
Springfield, Mass. Waterbury, Conn.	54 34	46 24		1	1	-	4	Wilmington, Del.	21	16	6 3	2	-	-	-	
Worcester, Mass.	63	51	8	3	1	-	8	E.S. CENTRAL	824	555				13	61	
MID. ATLANTIC	2.766	1,763		295	86	75		Birmingham, Ala.	105	66		7	2	4	3	
Albany, N.Y.	2,700	35	545 14	295	2	2	154 4	Chattanooga, Tenn.	64	42		4		-	7 9	
Allentown, Pa.	13	11	2	-	-	-	-	Knoxville, Tenn. Louisville, Ky.	83 93	58 68		5 1		1	7	
Buffalo, N.Y.	107	73		5	3	4	10	Memphis, Tenn.	152	94		16	8		6	
Camden, N.J.	24	15		4	-	-	-	Mobile, Ala.	122	84		9	5	2	11	
Elizabeth, N.J. Erie, Pa.†	17 49	15 42		:	-	-	1	Montgomery, Ala.	43	27		4		1	3	
Jersey City, N.J.	49 61	38		- 6	-	1	3	Nashville, Tenn.	162	116	5 20	16	5	5	15	
New York City, N.Y.		802		183	37	40	54	W.S. CENTRAL	1,334	839		122	35	30	91	
Newark, N.J.	87	35	15	25	3	7	6	Austin, Tex.	67	40		11	3	-	6 2	
Paterson, N.J.	24	13	6	.4		1	-	Baton Rouge, La. Corpus Christi, Tex.	39 73	24 51		4	2	1	2	
Philadelphia, Pa.	597 68	399	112	47	22	17	41	Dallas, Tex.	203	119		23	5	1	5	
Pittsburgh, Pa.† Reading, Pa.	40	42 27	14 11	5 1	7	-	4 10	El Paso, Tex.	80	52		5	3	1	6	
Rochester, N.Y.	109	79	18	5	6	1	8	Ft. Worth, Tex.	84	50	20	10		2	4	
Schenectady, N.Y.	23	18	4	1	-	-	2	Houston, Tex.	340	189		31	14	18	31	
Scranton, Pa.†	28	19	5	2	1	1	1	Little Rock, Ark.	61	40		5 U	2 U	1 U	1 U	
Syracuse, N.Y. Trenton, N.J.	57 37	37 22	18	-	1	1	3	New Orleans, La.§ San Antonio, Tex.	U 215	U 157		15	1		16	
Utica, N.Y.	37 19	18	10 1	5	:	-	1	Shreveport, La.	55	38		4		1	10	
Yonkers, N.Y.	31	23	5	-	3		5	Tulsa, Okla.	117	79		10	3	5	8	
E.N. CENTRAL	2,471	1,571	487	203	111	99	149	MOUNTAIN	874	600		75	22	16	47	
Akron, Ohio	79	56	14	6	-	3	5	Albuquerque, N.M.	112	77		8		4	5 2	
Canton, Ohio	43	32		1		-	5	Colo. Springs, Colo. Denver, Colo.	49 107	29 63		9 10	2	5	12	
Chicago, III. Cincinnati, Ohio	518 153	211 100	123 31	86 12	69	29	21	Las Vegas, Nev.	150	102		12		1	5	
Cleveland, Ohio	162	110		15	2	8 7	19 4	Ogden, Utah	25	18		3	-	-	6	
Columbus, Ohio	200	133	32	17	11	÷	3	Phoenix, Ariz.	230	165		17	6	4	5	
Dayton, Ohio	130	107	14	7	1	1	12	Pueblo, Colo.	23	17		_	1	-	4	
Detroit, Mich.	272	172	56	15	8	21	10	Salt Lake City, Utah Tucson, Ariz.	48 130	29 100		5 11	2 3	2	6	
Evansville, Ind. Fort Wayne, Ind.	60 75	46 54	12 8	1	- 5	1	1									
Gary, Ind.	19	12	5	1	5	4	4	PACIFIC Berkeley, Calif.	2,262	1,529		219 1	71	57	159	
Grand Rapids, Mich.	75	57	6	5	4	3	6	Fresno, Calif.	18 70	13 45			3	5	7	
Indianapolis, Ind.	185	127	45	7	2	4	17	Glendale, Calif.	49	36		ĕ	1	ĭ	2	
Madison, Wis.	35	21	8	2	3	1	4	Honolulu, Hawaii	92	70		7	1	1	12	
Milwaukee, Wis. Peoria, III.	144 50	107 36	28 13	5 1	1	3	9	Long Beach, Calif.	83	61		4		4	11 43	
Rockford, III.	43	32	5	3	-	3	4	Los Angeles, Calif. Oakland, Calif.§	728 U	492 U		72 U	27 U	9 U	43 U	
South Bend, Ind.	38	29	8	ĭ	-	-	5	Pasadena, Calif.	39	29		4			1	
Toledo, Ohio	131	86	31	11	2	1	10	Portland, Oreg.	142	101		6	8	5	7	
Youngstown, Ohio	59	43	10	3	-	3	4	Sacramento, Calif.	168	119		7	5	6	15	
W.N. CENTRAL	855	613	152	34	24	32	71	San Diego, Calif.	153	94		24		5	16 6	
Des Moines, Iowa	68	53		1	1	6	7	San Francisco, Calif. San Jose, Calif.	. 175 196	104 126		32 16		3 9	17	
Duluth, Minn. Kansas City, Kans.	27	23 23		-	-	-	-	Seattle, Wash.	196	126		22		9	6	
Kansas City, Kans. Kansas City, Mo.	31 121	23 84		3 4	- 5	;	12	Spokane, Wash.	66	50		5		4	ĕ	
Lincoln, Nebr.	35	27	6	2	5		4	Tacoma, Wash.	96	63		10		1	10	
Minneapolis, Minn.	203	153	33	9	2	6	18	TOTAL	13,437 *	* 8,814	2,610	1,192	442	366	862	
Omaha, Nebr.	71	49		2	2	3	4			-,	-,- /•	.,				
St. Louis, Mo.	148	87	35	7	10	9	9									
St. Paul, Minn. Wichita, Kans.	82 69	64 50		2 4	1	1	10 7									
	03	50	12	4	3	•										

TABLE III. Deaths in 121 U.S. cities,* week ending March 23, 1991 (12th Week)

*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.

Included.
*Pneumonia and influenza.
*Pneumonia and influenza.
TBecause 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.
HTTotal includes unknown ages.
§Report for this week is unavailable (U).

HIV Infection - Continued

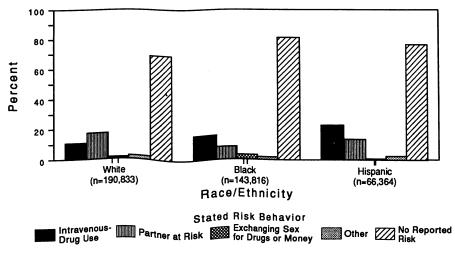
Previous documentation of the association between syphilis and transmission of HIV (3-5) suggests that syphilis contributes to heterosexual transmission of HIV in selected U.S. populations. However, because many women at risk for syphilis may not be aware of the associated risk for HIV, they may have reported no risk behaviors during pretest counseling. Accordingly, STD and HIV control programs should direct efforts to diagnosing and treating women who have syphilis or who are reported as sex partners of syphilis patients (6) and should ensure that these persons receive HIV CT.

STD and HIV prevention programs need to maximize the proportion of high-risk women who receive comprehensive HIV risk assessment, accept pretest counseling, accept HIV testing, return for their test results, and receive posttest counseling. Based on the data in this report, a substantial proportion of women who seek HIV CT services at selected public sites may be at risk for HIV infection; however, many of these women may be unaware of, or unwilling to report, a specific risk behavior. Therefore, CT sites that serve women in areas with high prevalence of HIV seropositivity should routinely offer all clients HIV counseling and testing. In areas with low prevalence of HIV seropositivity, standardized, thorough risk assessments may assist in identifying a person's risks for HIV infection, and recommendations for HIV testing can be made based on the results of each assessment.

References

- 1. CDC. HIV/AIDS surveillance. Atlanta: US Department of Health and Human Services, Public Health Service, January 1991:9–14.
- Department of Commerce. Census of population, general population characteristics, United States summary. Washington, DC: US Department of Commerce, Bureau of the Census, May 1983:1–21.
- 3. Rolfs RT, Nakashima AK. Epidemiology of primary and secondary syphilis in the United States, 1981 through 1989. JAMA;264:1432-7.
- Cameron DW, Simonsen JN, D'Costa LJ, et al. Female to male transmission of human immunodeficiency virus type I: risk factor for seroconversion in men. Lancet 1989;2:403–7.

FIGURE 1. Percentage of tests for HIV among women at publicly funded counseling and testing sites who had known risk exposure, by race/ethnicity – United States, 1989–1990



HIV Infection - Continued

- Quinn TC, Cannon RO, Glasser D, et al. The association of syphilis with risk of human immunodeficiency virus infection in patients attending sexually transmitted disease clinics. Arch Intern Med 1990:150:1297–302.
- Toomey KE, Cates W Jr. Partner notification for the prevention of HIV infection. AIDS 1989;3(suppl 1):S57–62.

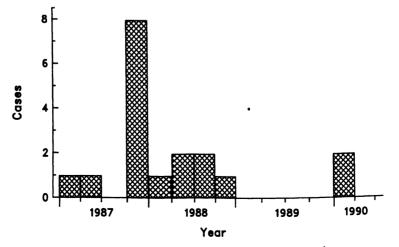
Topics in Minority Health

Tuberculosis Outbreak on Standing Rock Sioux Reservation – North Dakota and South Dakota, 1987–1990

From 1987 through 1988, a cluster of cases of tuberculosis (TB) occurred among persons residing on the Standing Rock Sioux Reservation (Figure 1); recognition of this cluster prompted an epidemiologic investigation by the Indian Health Service (IHS), Standing Rock Sioux Tribal Health and Community Health Programs, South Dakota State Department of Health, North Dakota State Department of Health, and CDC. This report summarizes the findings of that investigation and follow-up public health interventions.

Standing Rock Sioux Reservation, which comprises approximately 3640 square miles, encompasses Sioux County, North Dakota, and Corson County and portions of Dewey and Ziebach counties in South Dakota (1981 resident American Indian population: 7958). The IHS provides clinical and preventive health services for the tribal population on the reservation through a hospital at Fort Yates, North Dakota; a clinic at McLaughlin, South Dakota; and field clinics in three outlying communities. Five TB cases were diagnosed in persons on the reservation in 1986 (rate: 62.8 per 100,000 persons), 10 cases in 1987 (rate: 125.7 per 100,000), six cases in 1988 (rate: 75.4 per 100,000), and two cases in the first 5 months of 1990.

FIGURE 1. Reported tuberculosis cases,* by quarter of onset — Standing Rock Sioux Reservation, North Dakota and South Dakota, 1987–1990



*One case not shown occurred in a man who lived outside the reservation.

Tuberculosis – Continued

Sioux County

From May 1987 through January 1988, seven cases of TB were reported in residents of the adjacent towns of Solen and Cannonball, Sioux County. The investigation linked transmission from an adult source patient to five of his contacts. In October 1987, the source patient (index patient A), a man with diabetes mellitus who resided in Solen, was hospitalized with sputum-smear-positive, cavitary TB. He had a documented positive tuberculin skin-test reaction in 1967 and was evaluated for TB in 1977 because of chest pain and hemoptysis.

At the time of diagnosis in 1987, he had a history of heavy alcohol consumption and lived in a house with approximately 20 persons. Of 33 identified contacts, six were diagnosed with TB, including two children who lived in the same house with the index patient, a child and a woman who lived in Cannonball, a man in Solen, and a man who lived outside the reservation (not included in Figure 1). The *Mycobacterium tuberculosis* phage type for index patient A was 1 (7,13), the same as that of one of the children living in his house and that of the woman living in Cannonball. Phage typing was not done for the other four contact cases. Of his 20 contacts with no previous record of tuberculin skin-test positivity, seven (35%) were positive; all seven completed a course of directly observed preventive therapy.

A seventh case occurred in a man (index patient B) living in Solen. Although he was a contact of index patient A, his isolate had a different phage type (phage type 1). In 1981, he was tuberculin skin-test positive but did not adhere to preventive therapy. In 1984, when his wife had onset of smear-positive pulmonary TB, index patient B did not receive preventive treatment. In April 1987, when he was evaluated because of shortness of breath, a chest radiograph indicated cavitation in the right upper lobe; however, TB was not diagnosed by his health-care providers. He was not evaluated further until November 1987, when he was hospitalized for massive hemoptysis; at that time, a chest radiograph indicated multilobe infiltration, and the sputum smear was positive for acid-fast bacilli. Of his nine contacts with no previous record of a positive skin test, four (44%) had positive tests; all four completed a course of directly observed preventive therapy.

Corson County

During 1987, four TB cases were reported among residents in the town of Little Eagle, Corson County (70 miles from Solen); three occurred among brothers, and one epidemiologically unrelated case occurred in a woman.

In October 1987, pulmonary TB was diagnosed in a disabled, bedridden man (index patient C). In December 1987, a chest radiograph obtained from one of his brothers indicated a small patchy lesion in the right upper lobe; this brother had a history of tuberculous pleural effusion in 1955 for which he was treated with isoniazid (INH), para-aminosalicylic acid, and streptomycin for an unknown duration. Because of negative sputum smears and cultures for mycobacteria and the small radiographic extent of the lesion, he was considered an unlikely source of disease for the index patient. The most likely source was a second brother who, in December 1987, was diagnosed with sputum-smear-positive, cavitary TB; his *M. tuberculosis* phage type was 1 (7,13). Phage typing was not done for the index patient. The father, who was deceased, was the only other member of the family with a reported history of TB. Although preventive therapy had been prescribed previously for several of the family members, including the probable source patient, adherence could not be assessed.

Tuberculosis - Continued

No patients with TB were identified among 36 other contacts of index patient C and the probable source patient. However, of 15 contacts with no previous record of a positive tuberculin skin test, six (40%) were positive; all six completed a course of directly observed preventive therapy with INH.

The unrelated case occurred in a woman who was diagnosed with genitourinary TB in March 1987; the phage type was 2 (7,13).

Control and Prevention Response

Because of the high potential for TB transmission in the three communities, tuberculin skin-testing activities were expanded to schools and offered to the public. From October 1987 through February 1988, 724 persons were examined. Of these, 139 (19%) (including persons who were both tuberculin-positive and tuberculin-negative contacts, other persons with positive reactions, and persons who had previously had incomplete courses of preventive therapy) were placed on twice-weekly supervised preventive therapy; 137 persons completed at least 6 months of preventive therapy. One discontinued INH because of elevated liver enzymes; the other, who refused medication, subsequently had onset of TB and is currently receiving treatment. All persons with TB were treated with directly observed chemotherapy.

From February 1988 through May 1990, TB was diagnosed in seven other persons on the Standing Rock Sioux Reservation, four of whom were from Cannonball. Contact investigation did not indicate that any of these patients had been closely associated with any of the patients identified from October 1987 through January 1988, and no mycobacteriophage typing results of these cases were available.

Reported by: Standing Rock Sioux Tribal Health and Community Health Representative Programs; L Volmer, Communicable Disease Control Program, KA Senger, State Epidemiologist, South Dakota State Dept of Health. F Heer, Div of Disease Control, S McDonough, MD, State Epidemiologist, North Dakota State Dept of Health. HL Rieder, MD, Federal Office of Public Health, Bern, Switzerland. TK Welty, MD, JS Takehara, MPH, Aberdeen Area Indian Health Svc, Rapid City, South Dakota. D Dailey, FNP, Indian Health Svc Unit, Ft. Yates, North Dakota. Mycobacteriology Laboratory, Respiratory Diseases Br, Div of Bacterial and Mycotic Diseases, Center for Infectious Diseases; Div of Tuberculosis Elimination, Center for Prevention Svcs, CDC.

Editorial Note: Since 1975, when national reporting of TB among American Indians and Alaskan Natives began, the incidence of TB in these groups has progressively declined (1,2). In 1987, 317 cases were reported to CDC, a rate of 20.0 per 100,000-4.7 times higher than the rate of 4.3 per 100,000 in the non-Hispanic white population (3). In comparison, the 10 reported cases in persons on the Standing Rock Sioux Reservation in 1987 resulted in a case rate of 125.7 per 100,000 population.

As TB becomes located in focal geographic areas (4), small outbreaks of TB are more likely to be recognized and should prompt rapid intervention and containment. The investigation in this report suggested that three independent sources of infection developed almost concurrently and resulted in persons with secondary cases that were potentially infectious and in persons with asymptomatic tuberculous infection. All three source patients had documented prior tuberculous infection, and preventive therapy had previously been prescribed for two. In two cases, patient delay in seeking treatment accounted for the extensive disease at the time of the diagnosis; in the third case, the failure to prevent progression from early TB to extensive infectious disease was attributable to lack of follow-up of a suspicious radiograph.

Although the incidence of TB has declined during the past 35 years, decreased clinical consideration by physicians can lead to unnecessary delays in diagnosis. Noncompliance with treatment remains one of the most serious constraints in

Tuberculosis - Continued

effective TB control. Innovative ways to ensure adherence to prescribed treatment regimens, including fully supervised chemotherapy and preventive therapy (as was done in this outbreak), need to be implemented more widely, especially among high-risk populations (*5,6*).

The appropriate use of preventive therapy is crucial if TB is to be eliminated in the United States by the year 2010 (6). When taken as prescribed, INH preventive therapy is highly effective in preventing latent tuberculous infection from progressing to clinically apparent disease. The usual preventive therapy regimen is INH (10 mg/kg daily for children, 300 mg daily maximum for adults). The recommended duration of INH preventive treatment varies from 6 to 12 months of continuous therapy (7,8). To ensure that persons in high-risk groups comply with therapy, health-care personnel should, if necessary, directly observe the therapy must be directly observed and resources are inadequate for daily therapy (7,8). Patients should be thoroughly educated about the disease and its treatment and should be monitored for toxicity to medication by appropriately trained personnel (8). The IHS has issued guidelines for TB control (9,10), modeled after recommendations by the American Thoracic Society and CDC (7) and adjusted to IHS-specific needs.

References

- CDC. Tuberculosis among American Indians and Alaskan Natives-United States, 1985. MMWR 1987;36:493-5.
- 2. Rieder HL. Tuberculosis among American Indians of the contiguous United States. Public Health Rep 1989;104:653–7.
- 3. Rieder HL, Bloch AB, Cauthen GM, Kelly GD, Snider DE Jr. Tuberculosis in the United States. JAMA 1989;262:385–9.
- 4. Bloch AB, Rieder HL, Kelly GD, Cauthen GM, Hayden CH, Snider DE Jr. The epidemiology of tuberculosis in the United States. Semin Respir Infect 1989;4:157–70.
- Iseman MD, Sbarbaro JA, eds. National consensus conference on tuberculosis. Chest 1985; 87:125S–32S.
- 6. CDC/Advisory Committee for Elimination of Tuberculosis (ACET). A strategic plan for the elimination of tuberculosis in the United States. MMWR 1989;38(no. S-3).
- 7. American Thoracic Society/CDC. Treatment of tuberculosis and tuberculosis infection in adults and children. Am Rev Respir Dis 1986;134:355–63.
- CDC/Advisory Committee for Elimination of Tuberculosis (ACET). Screening for tuberculosis and tuberculous infection in high-risk populations and the use of preventive therapy for tuberculous infection in the United States: recommendations of the Advisory Committee for Elimination of Tuberculosis. MMWR 1990;39(no. RR-8).
- Welty TK, Helgerson S, Tempest B, Johannes P. Control of tuberculosis among American Indians and Alaska Natives. The Indian Health Service Primary Care Provider 1989;14:53–4.
- Welty TK, Follas R. IHS standards of care for tuberculosis. The Indian Health Service Primary Care Provider 1989;14:54–8.

PHS/CDC

PAID

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 cr 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: 1991-531-130/22060 Region IV

Penalty for Private Use \$300 Official Business Centers for Disease Control Public Health Service Atlanta, Georgia 30333 HEALTH AND HUMAN SERVICES DEPARTMENT OF HBE **POSTAGE & FEES** FIRST-CLASS MAIL Permit No. G-284

Redistribution using permit imprint is illegal.

×