

MNWR™

MORBIDITY AND MORTALITY WEEKLY REPORT

- 205 Trichinellosis Associated with Eating Cougar Jerky — Idaho, 1995
- 207 Human Rabies — Connecticut, 1995
- 209 Traumatic Brain Injuries — Oklahoma, 1992–1994
- 211 Infant Mortality — U.S., 1993
- 215 Undervaccination for Hepatitis B
- 217 ACIP Recommendations

Outbreak of Trichinellosis Associated with Eating Cougar Jerky — Idaho, 1995

On March 3, 1995, the Idaho Department of Health and Welfare received a report from the North Central District Health Department (NCDHD) of trichinellosis in a man residing in Idaho County, Idaho (1994 population: 14,938). This report summarizes the epidemiologic and laboratory investigations of this index case and the related outbreak by NCDHD, the U.S. Department of Agriculture (USDA), and CDC.

During the second week of January, the index patient shot and killed a cougar (*Felis concolor*) near Elk City, Idaho. During January 15–18, he prepared jerky from the cougar meat by first soaking the meat in a brine solution made from table salt, then smoking the meat; however, he later reported the smoker never became more than warm. During the next 4 weeks, he distributed the meat to 14 other persons, all of whom ate the meat within days to 1 month after receipt.

On January 26, the man had onset of illness characterized by fever, myalgia, arthralgia, facial swelling, and fatigue. On examination by his physician, his total white blood cell count was 8500/mm³ (normal: 5000–10,000/mm³) with 48% eosinophils, 32% segmented neutrophils, 17% lymphocytes, and 3% monocytes. Based on these findings, trichinellosis was suspected, and he was referred to an infectious disease consultant.

Samples of cougar jerky examined at the Sacred Heart Medical Center Department of Laboratory Medicine in Spokane, Washington, on February 22 contained *Trichinella* larvae. Examination of a muscle biopsy obtained from the patient on February 23 revealed granulomatous myositis with eosinophils. Serum obtained March 9 tested positive for *Trichinella* antibody by bentonite flocculation (1:160) and enzyme-linked immunosorbent assay (ELISA) (3.707) at CDC. *Trichinella* larvae were identified in specimens of jerky and fresh frozen cougar muscle submitted to the USDA in March. Polymerase chain reaction was performed on live larvae recovered from the fresh frozen tissue, and results were consistent with the sylvatic genotypes *T. nativa* and *Trichinella* T6.

During March 3–April 10, NCDHD interviewed the 14 persons who had received jerky. A case of trichinellosis was defined as 1) a *Trichinella*-positive muscle biopsy or positive serologic test for trichinellosis in a patient with eosinophilia, fever, myalgia, and/or periorbital edema; or 2) either a positive serologic test for trichinellosis or eosinophilia, fever, myalgia, and/or periorbital edema in a person who had eaten the cougar jerky. Based on these criteria, nine additional cases were identified. Manifestations

Trichinellosis — Continued

among the 10 cases included myalgia (seven), fever (six), rash (three), weakness (three), and arthralgia (two). Seven of these persons were men; case-patients ranged in age from 25 to 52 years.

The index patient and seven others were treated with mebendazole. Persons who had received the jerky were educated by NCDHD on trichinellosis prevention.

Reported by: A Vollbrecht, D Sokolowski, North Central District Health Dept, Lewiston; W Hol-lipeter, MD, Grangeville; R Sigler, MD, Cottonwood; J Greenblatt, MD, State Epidemiologist, Idaho Dept of Health and Welfare. DE Anderson, Jr, PhD, Sacred Heart Medical Center, PJ Ten-nican, Infectious Diseases Northwest, Spokane, Washington. HR Gamble, PhD, D Zarlenga, Parasite Biology and Epidemiology Laboratory, US Dept of Agriculture. Div of Parasitic Diseases, National Center for Infectious Diseases; Div of Field Epidemiology, Epidemiology Program Office, CDC.

Editorial Note: Since trichinellosis was designated a reportable condition in 1947, the number of cases reported annually by state health departments has declined from an average of 400 cases per year in the late 1940s to 32 cases in 1994 (1,2). The proportion of cases associated with eating contaminated commercial pork has been declining since 1975, most likely because of laws prohibiting feeding offal to hogs, the increased use of home freezers, and the practice of thoroughly cooking pork (2).

Trichinella species are found in virtually all warm-blooded animals. As domestic swine-associated cases have decreased, the proportion of cases associated with eating wild game has increased, and cases have resulted from consumption of bear, wild boar, and walrus (3–5). This report is the first to document cougar as the source of trichinellosis in the United States. In this investigation, viable larvae were recovered from meat that had been frozen. Although most species of *Trichinella* are killed by freezing, results of the genomic DNA amplification performed at USDA suggest that the cougar isolate was either *T. nativa* or *Trichinella* T6, both freeze-resistant strains that have not been previously reported in Idaho.

To ensure that *Trichinella* are destroyed, meat should be thoroughly cooked. A temperature of 170 F (77 C) exceeds the thermal death point of the trichinae and usually is achieved if the meat is cooked until the color changes from pink or red to gray. Some brine solutions used for preparing jerky also may kill *Trichinella*; however, curing temperature and total duration of time at this temperature are important determinants in this process (6). Physicians should be aware of the continued presence of *Trichinella* sp. in commercial pork and wild game in the United States and should consider the diagnosis in any patient with an illness compatible with trichinosis and whose dietary preferences pose a risk for infection (2).

References

1. Schantz PM. *Trichinosis* in the United States, 1947–1981. *Food Technol* 1983;37:83–6.
2. CDC. *Trichinella spiralis* infection—United States, 1990. *MMWR* 1991;40:57–60.
3. Bailey TM, Schantz PM. Trends in the incidence and transmission patterns of human trichinosis in the United States, 1982–1986. *Rev Infect Dis* 1990;12:5–11.
4. MacLean JD, Viallet J, Law C, et al. Trichinosis in the Canadian arctic: report of five outbreaks and a new clinical syndrome. *J Infect Dis* 1989;160:513–20.
5. Woodard TL. An outbreak of trichinosis traced to Alaskan black bear meat. *Alaska Medicine* 1988;30:41–4.
6. Zimmerman WJ. Salt cure and drying time and temperature on viability of *Trichinella spiralis* in dry cured hams. *J Food Sci* 1971;36:58–65.

Human Rabies — Connecticut, 1995

On October 3, 1995, a 13-year-old girl who resided in Greenwich, Connecticut, died from rabies virus infection. This was the first case of human rabies reported in a Connecticut resident since 1932. This report summarizes the investigation of this case, which indicated a bat as the probable source of her exposure.

On September 18, the patient reported general fatigue, stiffness, tremors, and tingling in her left arm and hand. On September 22, she visited a local emergency department because of pain and tingling in her left arm and shoulder and a low-grade fever. Cervical radiculopathy was presumptively diagnosed and was attributed to her habit of carrying a heavy backpack; ibuprofen was prescribed. She was given a cervical collar and referred to a pediatric neurologist.

On September 25, because of continuing symptoms, she was evaluated by her pediatrician, who noted sensory changes on the left arm and face. She was again referred to a pediatric neurologist and, later that day, was admitted to a hospital because of complaints of fever, neck pain, and painful sensations along her left arm and left side of her face. On physical examination, her temperature was 100.0 F (37.8 C), and she was alert but anxious; there was moderate nuchal rigidity. The only abnormal neurologic finding was deviation of the uvula to the left. Laboratory findings included a peripheral white blood cell (WBC) count of 13,600/mm³ (normal: 5000–10,000/mm³) with 86% neutrophils, 10% lymphocytes, and 4% monocytes. Her cerebrospinal fluid (CSF) contained 2 red blood cells/mm³ (normal: 0/mm³) and 100 WBCs/mm³ (normal: 0–5/mm³) with 48% neutrophils, 40% lymphocytes, and 12% monocytes, total protein of 104 mg/dL (normal: <40 mg/dL), and glucose level of 53 mg/dL; serum glucose was 102 mg/dL (normal: 70–110 mg/dL).

The diagnosis on admission was possible Lyme meningoencephalitis with peripheral nerve involvement; treatment was initiated with intravenous ceftriaxone and dexamethasone. During the 24 hours following admission, she became intermittently drowsy then agitated, and occasionally was disoriented. Subsequent manifestations included deviation of her tongue to the right, anisocoria, and progressive weakness of the left arm. She also was observed to be apprehensive and had difficulty swallowing, accompanied by a prominent aversion to oral intake. Severe pharyngeal spasms were elicited by offering a drink of water. The diagnosis of rabies was considered, and the patient was placed in isolation. She became increasingly agitated; although she experienced tactile hallucinations (i.e., complaining of a sensation of insects in her mouth), she intermittently was lucid and self-reflective and apologized for her mood and hallucinations.

On September 26, the girl was transferred to the intensive-care unit, where she was intubated because of progressive bulbar dysfunction. Beginning September 27, she became progressively less responsive, and subsequently lapsed into a coma. On October 3, mechanical ventilation was withdrawn, and the patient died. No autopsy was performed.

Rabies was diagnosed on October 2 at the New York State Rabies Laboratory based on corneal impressions collected on October 1, which were positive for rabies virus by immunofluorescence, and based on rising rabies virus neutralizing antibody titers of 1:32, 1:64, and 1:512 in serum samples collected on September 25, 29, and October 2, respectively. The diagnosis was confirmed at CDC through extraction of RNA from

Human Rabies — Continued

saliva and corneal epithelia, which was reverse transcribed with rabies-specific primers and amplified using the polymerase chain reaction (PCR) assay. Nucleotide sequencing of the PCR products at CDC characterized the rabies virus as a variant associated with the silver-haired bat, *Lasionycterus noctivagans*.

The girl lived in a single-family dwelling in a wooded residential area in Greenwich. Although she denied a history of animal bites, multiple potential sources of animal contact were present in the home and surrounding environment; domestic animals with which she was known to have had contact were accounted for and were well. Following the diagnosis of rabies, the girl's mother and three siblings recalled that on approximately August 19, a bat flying inside the house struck at least one person; during this time, the girl was asleep in an upstairs bedroom. Inspection of the house and surrounding property by the Greenwich Department of Health on September 29 did not identify dead animals or evidence of bats.

Because of possible percutaneous or mucous membrane contact with the girl's secretions during September 10–October 3, rabies postexposure prophylaxis was administered to 83 persons who reported probable contact with the patient's saliva: 46 health-care workers, 29 children, four family members, three family friends intimately involved in the girl's care, and one other adult.

Reported by: JL Muñoz, MD, R Wolff, MD, A Jain, MD, J Sabino, MD, Westchester County Medical Center; G Jacquette, MD, M Rapoport, MD, Westchester County Dept of Health, Hawthorne, New York. J Lieberman, DVM, CC Baisley, BH Ward, MA, Greenwich Dept of Health, Greenwich; CR Brown, MD, Town of New Canaan, New Canaan, Connecticut; PM Stolte, AB Crowley, H Hastings, Nantucket Health Dept, Massachusetts. ML Cartter, MD, JL Hadler, MD, State Epidemiologist, Connecticut Dept of Public Health. TW French, PhD, Massachusetts Dept of Fisheries, Wildlife, and Environmental Law Enforcement, Westboro; S Kreindel, DVM, R Knowlton, Massachusetts Dept of Public Health. GS Birkhead, MD, JG Debbie, DVM, CA Hanlon, VMD, CV Trimarchi, DL Morse, MD, State Epidemiologist, New York State Dept of Health. Viral and Rickettsial Zoonoses Br, Div of Viral and Rickettsial Diseases, National Center for Infectious Diseases; Div of Field Epidemiology, Epidemiology Program Office, CDC.

Editorial Note: Since the 1950s, bats have accounted for an increasing proportion of variants of rabies virus transmitted from wildlife reservoirs to humans. The rabies virus variant identified in this case, and in a case in New York in 1993 (1), is associated with the silver-haired bat, a solitary, migratory species with a preferred habitat of old-growth forest. However, in neither of these cases was a clear history of bite exposure to a bat or any other animal established. Of the 28 cases of human rabies diagnosed in the United States since 1980, this case was the 15th to be associated with bats; 10 of the virus variants obtained from these 15 persons have been characterized as a silver-haired bat variant.

Bat rabies is enzootic in the United States, and cases have been reported from all 48 contiguous states (1). In Connecticut, of the 671 bats submitted to the state laboratory for testing during 1991–1995, a total of 47 (7%) were positive for rabies. Nine of the bats diagnosed with rabies in Connecticut during 1995 were sent to CDC for viral typing. Eight of the bats were infected with a variant associated with the common big brown bat (*Eptesicus fuscus*) and one bat was infected with a rabies virus variant associated with red bats (*Lasiurus borealis*). None of the bats were identified by species. In New York state, of the 6810 bats submitted to the state laboratory for rabies testing during 1988–1992, a total of 312 (4.6%) were positive for rabies; of these, approximately 90% were from *E. fuscus*. Only 25 of the submitted bats were silver-haired bats, of which only two were positive for rabies virus (2).

Human Rabies — Continued

The findings of the investigation of a recent case in Washington suggest that even apparently limited contact with rabid bats may be associated with rabies transmission (3). Because bites from bats may be very small, an exposure may not be recognized—particularly when an unattended child may not be able to accurately relate events to an adult.

The case described in this report and reports of similar cases (1,3,4) underscore the national recommendation that, in situations in which a bat is physically present and the person(s) cannot reasonably exclude the possibility of a bite exposure, post-exposure prophylaxis should be given unless prompt capture and testing of the bat has excluded rabies virus infection.

References

1. CDC. Human rabies—New York, 1993. MMWR 1993;42:799,805–6.
2. Childs JE, Trimarchi CV, Krebs JW. The epidemiology of bat rabies in New York state, 1988–92. *Epidemiol Infect* 1994;113:501–11.
3. CDC. Human rabies—Washington, 1995. MMWR 1995;44:625–7.
4. CDC. Human rabies—California, 1994. MMWR 1994;43:455–7.

Horseback-Riding–Associated Traumatic Brain Injuries — Oklahoma, 1992–1994

Each year, traumatic brain injury* (TBI) is associated with 52,000 deaths and accounts for one third of all injury deaths in the United States (2); in addition, approximately 80,000 persons who survive TBI incur some loss of function, residual disability, and increased medical-care needs because of these injuries (3). Major causes of TBI are motor-vehicle crashes, falls, assaults, and sports and recreational activities (3,4). During 1992–1993 in Oklahoma, horseback riding was the leading cause of sports-related TBI (5). To further characterize horseback-riding-associated TBIs, the Oklahoma State Department of Health (OSDH) and CDC analyzed these injuries for 1992–1994. This report summarizes the findings of this analysis.

In 1992, the OSDH established surveillance statewide in Oklahoma for all cases of TBI resulting in death or hospitalization. A case is defined as a TBI recorded on a medical examiner report or coded on a hospital discharge report as specified by *International Classification of Diseases, Ninth Revision*, codes N800.0–801.9, N803.0–804.9, and N850.0–854.1. Medical records of persons with TBI are reviewed by state injury-prevention service personnel at all 125 hospitals in the state either one, two, or four times per year (frequency depends on the size of the hospital). A standardized data-collection form is completed for each case. Information for all fatal cases is provided by the state Office of the Chief Medical Examiner, which investigates all trauma deaths. Horseback riding and other causes of injury are specified in the narrative of the medical record or medical examiner report.

During 1992–1994, a total of 9409 TBIs occurred in Oklahoma, of which 109 (1.2%), including three deaths, were associated with horseback riding; 23 other TBIs were at-

*Either 1) an injury to the head that is documented in a medical record, with one or more of the following conditions attributed to head injury: observed or self-reported decreased level of consciousness, amnesia, skull fracture, objective neurologic or neuropsychologic abnormality, or diagnosed intracranial lesion; or 2) death resulting from trauma, with head injury listed in the sequence of conditions that resulted in death on the death certificate, autopsy report, or medical examiner's report (1).

Traumatic Brain Injuries — Continued

tributable to horses but were not riding-associated. The numbers of these injuries were nearly equal among females (55) and males (54), and riders ranged in age from 3 years to 71 years (median: 30 years). Cases occurred more commonly during the spring (38) and summer (34) than winter (19) and autumn (18). Nearly one half (48%) of the riding-associated TBIs occurred on a Saturday or Sunday. Of 93 cases for which time of injury was known, 64 (69%) occurred between noon and 8 p.m. Of the 105 cases for which the mechanism of injury was specified, 100 (95%) involved riders who struck their heads either on the ground or a nearby object after falling from the horse, four (4%) who were kicked or rolled on by the horse after falling from the horse, and one (1%) who fell to the ground after his head struck a pole while riding. Of the 96 persons for whom information on type of activity was available, most (86 [90%]) were associated with recreational activity, and 10 (10%) were work-associated. A total of 107 persons were hospitalized (two persons died at the scene) and accounted for 388 hospital days (median duration of stay: 2 days).

Among the 106 survivors of riding-associated TBIs, 84 (79%) had one or more indicators of brain injury severity: 67 (63%) had loss of consciousness, 49 (46%) had post-traumatic amnesia, and 14 (13%) had persistent neurologic sequelae on discharge from the hospital (e.g., seizures or cognitive, hearing, vision, speech, and/or motor impairment). Among those hospitalized, the Abbreviated Injury Severity (AIS) scores[†] for the head region ranged from two (moderate) (64% of cases) to five (critical) (5% of cases). TBI was listed as the first (of a maximum of 10) discharge diagnosis for 90% of the hospitalized cases and as the first or second diagnosis for 99%.

Among the 23 TBIs attributable to horses not identified as riding-associated, 21 (91%) resulted from a direct kick to the head by a horse; one person died, and two others required cardiopulmonary resuscitation. Thirteen (57%) of these occurred among children aged ≤ 10 years. Of the 19 cases for which place of injury was specified, 15 occurred on a farm.

Reported by: P Archer, MPH, S Mallonee, MPH, S Lantis, MPH, Oklahoma State Dept of Health. Div of Acute Care, Rehabilitation Research, and Disability Prevention, National Center for Injury Prevention and Control, CDC.

Editorial Note: Even though only 1% of the TBIs in Oklahoma were associated with horseback riding, the medical burden of these injuries is substantial as reflected by the adverse neurologic outcomes and number of hospitalization days. In addition, the horseback-riding-associated TBIs described in this report probably underestimate the total number of all such injuries receiving medical care. For example, during 1991, 66% of persons in the United States who received medical care for TBIs were not hospitalized (4), and 79% of regular (i.e., six or more times per year) horseback riders who incurred a concussion or other nonfacial head injury were not hospitalized (6; CDC, unpublished data, 1995). In Oklahoma, only persons with TBIs who are hospitalized are identified by the surveillance system.

In Oklahoma, during 1992–1994, bicycle riding accounted for more than twice the number of TBIs as horseback riding (234 versus 109). However, the risk for injury during horseback riding probably was substantially greater than for bicycle riding: during 1989–1991, the rate of horseback-riding injury sufficiently severe to require hospital-

[†]AIS is an anatomic injury-severity scale ranging from one for minor injury to six for maximum injury.

Traumatic Brain Injuries — Continued

based emergency care was an estimated 28 per 100,000 riding hours (6), while the rate for bicycle-associated injuries was 3.7 per 100,000 riding hours (7).

The occurrence of most horseback-riding-associated TBIs during warm weather months, on weekends, and in the afternoon probably reflects greater ridership during those times. The high proportion of nonriding horse-related TBIs among children underscores the need for reducing the risk for direct contact with horses and the importance of using protective head gear for children who cannot be continuously supervised when near horses.

Horseback-riding-associated TBIs can be prevented by wearing protective helmets that meet the American Society of Testing and Materials (ASTM) standards (8). This measure is based on the documented effectiveness of helmet use for preventing bicycle-associated TBI by 88% (9). OSDH has included a recommendation for helmet use in the state's Strategic Plan for Injury Prevention and Control to promote helmet use among horseback riding clubs and organizations in Oklahoma. Additional efforts to prevent this problem should include direct assessment of the effectiveness of helmet use during horseback-riding activities, characterization of the biomechanics of horseback-riding-associated injury, educational programs in safe grooming and riding of horses, and careful matching of horses and supervision to the skill level of the rider.

References

1. Thurman DJ, Snieszek JE, Johnson D, Greenspan A, Smith SM. Guidelines for surveillance of central nervous system injury. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service, CDC, 1995.
2. Sosin DM, Snieszek JE, Waxweiler RJ. Trends in death associated with traumatic brain injury, 1979 through 1992. *JAMA* 1995;273:1778–80.
3. Kraus JF. Epidemiology of head injury. In: Cooper PR, ed. *Head injury*. 3rd ed. Baltimore, Maryland: Williams and Wilkins, 1993:1–25.
4. Sosin DM, Snieszek JS, Thurman DJ. Incidence of mild and moderate brain injury in the United States, 1991. *Brain Injury* 1996;10:47–54.
5. Lantis S. Horseback riding-related traumatic brain injuries. In: Injury Prevention Service. Injury update. Oklahoma City, Oklahoma: Oklahoma State Department of Health, Injury Prevention Service, April 1994.
6. Nelson DE, Rivara FP, Condie C, Smith SM. Injuries in equestrian sports. *The Physician and Sportsmedicine* 1992;22:53–60.
7. Rodgers GB, Tinsworth DK, Polen C, et al. Bicycle use and hazard patterns in the United States. Washington, DC: US Consumer Product Safety Commission, 1994.
8. American Academy of Pediatrics Committee on Sports Medicine and Fitness. Horseback riding and head injuries. *Pediatrics* 1992;89:512.
9. Thompson RS, Rivara FP, Thompson DC. A case-control study of the effectiveness of bicycle safety helmets. *N Engl J Med* 1989;320:1361–7.

Infant Mortality — United States, 1993

The 1993 final infant mortality (death before age 1 year) rate for the United States—8.4 infant deaths per 1000 live-born infants—was the lowest rate ever recorded and represented a decrease of 1.8% from the rate of 8.5 for 1992. Based on provisional data for 1994, the declining trend in infant mortality continued through 1994 (rate: 7.9)

Infant Mortality — Continued

(1). This report uses data from birth and death certificates compiled by CDC (2) to characterize infant mortality in 1993 and compares the findings with those for 1992.

Cause-of-death statistics are based on the underlying cause of death* reported on the death certificate by the attending physician, medical examiner, or coroner in a manner specified by the World Health Organization. Because race reflects differing distributions of several risk factors for infant death (e.g., low birthweight [LBW] [<2500 g (<5 lbs 9 oz) at birth]) and is useful for identifying groups at greatest risk for infant death, this analysis examines race-specific mortality rates. Numerators for infant mortality rates (infant deaths) were tabulated by race of infant; denominators for rates (live-born infants) were tabulated by race of mother. Rates are presented only for white and black infants because the Linked Birth/Infant Death Data Set (used to more accurately estimate infant mortality rates for other racial groups) was not available for 1992 and 1993. Numbers for white and black infants include both Hispanic and non-Hispanic infants.

In 1993, a total of 34,466 infants died in the United States, compared with 38,910 in 1992. The mortality rate for white infants in 1993 (6.8 per 1000) decreased 1.4% from the rate in 1992 (6.9), and the rate for black infants in 1993 (16.5) decreased 1.9% from the rate in 1992 (16.8).

Declines in race-specific rates also varied by age at death. From 1992 through 1993, the overall neonatal mortality (death before age 28 days) rate decreased 1.9% (5.4 to 5.3); for white infants, the rate remained constant (4.3), and for black infants, the rate decreased 1.0% (10.8 to 10.7). The overall postneonatal mortality (death at age 28 days–11 months) rate decreased 2.2% (3.1 to 3.0 per 1000); for white infants, the rate decreased 4% (2.6 to 2.5), and for black infants, 3.5% (6.0 to 5.8). In 1993, the risk for death during the first year of life for black infants remained 2.4 times greater than for white infants.

Among the 10 leading causes of infant death, the first four (congenital anomalies, sudden infant death syndrome, disorders relating to short gestation and unspecified LBW, and respiratory distress syndrome [RDS]) accounted for 54% of all infant deaths in 1993 (Table 1). For white infants, these causes also accounted for 54% of deaths, and for black infants, 51% of deaths.

From 1992 to 1993, the infant mortality rate decreased for seven of the 10 leading causes of infant death and increased for three causes (Table 1). For white infants, the rate also decreased for seven of the 10 causes; however, for blacks, the rate decreased for six of the 10 causes. Race-specific differences in infant mortality also were reflected in the rank order of the leading causes of infant death and in the rate of change of specific causes (Table 1). For white infants, the largest decrease in rates was for RDS (*International Classification of Diseases, Ninth Revision* [ICD-9], code 769) (15.5%), and the largest increase, disorders relating to short gestation and LBW (ICD-9 code 765) (16.1%). For black infants, the largest decrease was for pneumonia and influenza (ICD-9 codes 480–487) (16.8%), and the largest increase, infections specific to the perinatal period (ICD-9 code 771) (9.3%). The decrease in RDS for black infants was <1%.

Reported by: Div of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion; Mortality Statistics Br, Div of Vital Statistics, National Center for Health Statistics, CDC.

*Defined by the *International Classification of Diseases, Ninth Revision*, as "(a) the disease or injury which initiated the train of morbid events leading directly to death, or (b) the circumstances of the accident or violence which produced the fatal injury."

Infant Mortality — Continued

TABLE 1. Number of infant deaths*, mortality rate†, and percentage change from 1992 to 1993, and percentage of deaths attributed to the 10 leading causes of death, by race§ of mother and cause — United States, 1993

Race/ Rank order¶	Cause of death (ICD-9** codes)	No.	Rate	% Change in rate from 1992 to 1993	% Distribution
BLACK					
1	Disorders relating to short gestation and unspecified low birthweight (765)	2,021	306.7	2.0	18.6
2	Sudden infant death syndrome (798.0)	1,442	218.9	0.2	13.3
3	Congenital anomalies (740–759)	1,403	212.9	– 2.9	12.9
4	Respiratory distress syndrome (769)	686	104.1	– 0.6	6.3
5	Newborn affected by maternal complications of pregnancy (761)	452	68.6	– 0.9	4.2
6	Newborn affected by complications of placenta, cord, and membranes (762)	315	47.8	8.1	3.2
7	Infections specific to the perinatal period (771)	272	41.3	– 9.1	2.5
8	Accidents†† and adverse effects (E800–E949)	259	39.3	5.4	2.4
9	Pneumonia and influenza (480–487)	176	26.7	–16.8	1.6
10	Intrauterine hypoxia and birth asphyxia (768)	158	24.0	–12.7	1.5
	All other causes (residual)	3,703	562.0	– 4.1	34.0
All causes		10,887	1,652.4	– 1.9	100.0
WHITE					
1	Congenital anomalies	5,449	173.0	– 2.3	25.4
2	Sudden infant death syndrome	3,056	97.0	– 4.2	13.3
3	Disorders relating to short gestation and unspecified low birthweight	2,202	69.9	16.1	10.2
4	Respiratory distress syndrome	1,098	34.9	–15.5	6.3
5	Newborn affected by maternal complications of pregnancy	869	27.6	– 8.3	4.2
6	Newborn affected by complications of placenta, cord, and membranes	649	20.6	0	3.2
7	Accidents and adverse effects	599	19.0	14.5	2.4
8	Infections specific to the perinatal period	481	15.3	–14.5	2.2
9	Intrauterine hypoxia and birth asphyxia	371	11.8	– 6.4	1.7
10	Pneumonia and influenza	323	10.3	– 8.0	1.5
	All other causes (residual)	6,400	203.2	– 0.3	29.8
All causes		21,497	682.5	– 1.4	100.0
TOTAL §§					
1	Congenital anomalies	7,129	178.2	– 2.7	21.3
2	Sudden infant death syndrome	4,669	116.7	– 3.0	14.0
3	Disorders relating to short gestation and unspecified low birthweight	4,310	107.7	8.5	12.9
4	Respiratory distress syndrome	1,815	45.4	–10.6	5.4
5	Newborn affected by maternal complications of pregnancy	1,314	33.6	– 6.4	4.0
6	Newborn affected by complications of placenta, cord, and membranes	994	24.8	1.6	3.0
7	Accidents and adverse effects	898	22.4	11.4	2.7
8	Infections specific to the perinatal period	772	19.3	–13.1	2.3
9	Intrauterine hypoxia and birth asphyxia	549	13.7	– 9.3	1.6
10	Pneumonia and influenza	530	13.2	–10.8	1.6
	All other causes (residual)	10,457	261.4	– 1.7	31.2
All causes		33,466	836.6	– 1.8	100.0

*Death before age 1 year.

†Per 100,000 live-born infants in specified group.

§Race differences are given only for black and white infants because the Linked Birth/Infant Death Data Set (used to more accurately estimate infant mortality rates for other racial groups) was not available for 1992 and 1993. Hispanics and non-Hispanics are included in both racial groups.

¶Based on number of deaths.

** *International Classification of Diseases, Ninth Revision.*

†† When a death occurs under "accidental" circumstances, the preferred term within the public health community is "unintentional injury."

§§ Includes races other than black and white.

Infant Mortality — Continued

Editorial Note: The infant mortality rate is a standard index of health. In 1991 (the most recent year for which comparative data are available), the U.S. infant mortality rate ranked 24th among countries or geographic areas with a population of ≥ 1 million (3). Two national health objectives for the year 2000 are 1) to reduce the overall infant mortality rate to no more than 7 per 1000 (objective 14.1) and 2) to reduce the rate among black infants to no more than 11 per 1000 (objective 14.1a) (4). Based on the findings for 1993, attaining these goals will require an average annual decrease of 2.6% in the overall infant mortality rate and a 3.6% annual decrease in the rate for black infants.

In 1993, disorders relating to short gestation and unspecified LBW was the third leading cause of infant death overall, and the leading cause for black infants. During 1992–1993, mortality rates for this cause increased 8.4%, and during 1985–1993 increased 24%. However, based on data about infant birthweight or gestational age available in linked birth and infant death files, recent reductions in the misclassification of infant deaths to this cause may have obfuscated the true increase. If the number of deaths from disorders relating to short gestation and unspecified LBW continue to increase, the year 2000 objectives may not be attained.

The increased role of disorders relating to short gestation and unspecified LBW may reflect the 7.2% increase in LBW infants during 1992–1993 (5). A large portion of the increase in LBW infants has been attributed to increases in the rate of multiple births to white women and sustains a trend since 1980 (6). During 1985–1991, the proportion of deaths attributed to short gestation and unspecified LBW accounted for by multiple births increased from 5.6% to 11.5%—more than doubling for both white infants and black infants; however, this increase reflects primarily a large increase in the cause-specific infant mortality rate for whites (16.1%) (the rate for blacks increased only 2.0%). In comparison, during this period, infant mortality rates decreased for other causes associated with LBW (i.e., RDS, newborn affected by maternal complications of pregnancy, and congenital anomalies).

Although the total infant mortality rate declined in 1993, differences persisted in race-specific rates. This pattern underscores the need to distinguish between the factors associated with the decline and those associated with the race-specific differences. Reasons have not been determined for the lack of progress in reducing the infant mortality rates and race-specific differences associated with short gestation and LBW. Improved understanding of the heterogeneity of the risk factors that account for infant mortality and race-specific differentials associated with short gestation and LBW is essential for the development of prevention strategies aimed at reducing these differences. Differences in socioeconomic status and access to health care account for only a portion of the race-specific mortality differences (7,8), indicating the need for examination of information on other factors that may not be available from routinely collected data.

References

1. NCHS. Annual summary of births, marriages, divorces, and deaths: United States, 1993. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, 1994. (Monthly vital statistics report; vol 42, no. 13).
2. NCHS. Advance report of final mortality statistics, 1992. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, 1994. (Monthly vital statistics report; vol 43, no. 2, suppl).

Infant Mortality — Continued

3. NCHS. Health, United States, 1994. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, 1995; DHHS publication no. (PHS)95-1232.
4. Public Health Service. Healthy people 2000: national health promotion and disease prevention objectives—full report, with commentary. Washington, DC: US Department of Health and Human Services, Public Health Service, 1991; DHHS publication no. (PHS)91-50212.
5. NCHS. Advance report of final natality statistics, 1993. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, 1995. (Monthly vital statistics report; vol 44, no. 3, suppl).
6. Martin JA, Taffel SM. Current and future impact of rising multiple birth ratios on low birth weight. In: MetLife. Statistical bulletin. New York, New York: Metropolitan Life Insurance Co, 1995 (April–June issue).
7. Schoendorf KC, Hogue CJR, Kleinman JC, Rowley D. Mortality among infants of black as compared with white college-educated parents. *N Engl J Med* 1992;326:1522–6.
8. CDC. Poverty and infant mortality—United States, 1988. *MMWR* 1995;44:922–7.

Undervaccination for Hepatitis B Among Young Men Who Have Sex With Men — San Francisco and Berkeley, California, 1992–1993

The evaluation of efforts to prevent hepatitis B virus (HBV) infection in the United States requires accurate measures of hepatitis B vaccination coverage among children and adults at risk for infection (1). Although vaccination coverage among children is obtained by nationwide surveys (2), vaccination coverage among adults at risk for HBV infection has not been well characterized. To estimate hepatitis B vaccination coverage among young men who have sex with men (MSM) (a group known to be at high risk for HBV infection and for whom hepatitis B vaccine has been recommended since 1982), CDC analyzed serologic data from the 1992–1993 Young Men's Survey (YMS) conducted by the San Francisco Department of Public Health (3). This report summarizes the results of that analysis, which indicate low hepatitis B vaccination coverage among young MSM in the San Francisco Bay area.

YMS used a targeted sampling method to enroll MSM aged 17–22 years at selected public venues (e.g., high-traffic street corners, dance clubs, bars, and parks) in San Francisco and Berkeley, California (3). During periods of recruitment, all young men who entered a predetermined area (e.g., stretch of sidewalk) at sampled venues were approached by YMS recruiters. Men who appeared to be substantially older than age 22 years were not approached. Young men who accepted approaches were interviewed by recruiters to determine their eligibility (i.e., aged 17–22 years and residence in the San Francisco Bay area) and willingness to participate in YMS. Participants provided blood specimens and were interviewed for sexual, drug-use, and health-care use practices in a specially-equipped mobile van. Blood specimens were tested for hepatitis B surface antigen (HBsAg), and antibody to both HBsAg (anti-HBs) and hepatitis B core antigen (anti-HBc). Because YMS was designed to measure the prevalence of human immunodeficiency virus (HIV) infection and related risk behaviors among young MSM, hepatitis B vaccination histories were not obtained from participants.

During July 1992–April 1993, YMS recruiters approached approximately 2000 young men during 96 sampling events at 26 different venues. Of 1773 (89%) young men who agreed to be approached, 778 (44%) were determined eligible, of whom 474 (61%) enrolled in the survey. Among these participants, 385 (81%) reported having

Hepatitis B — Continued

had oral or anal sex with one or more men during the preceding 6 months. This analysis was restricted to these 385 men.

Among the 385 MSM, 77 (20%) had evidence of previous or current HBV infection, including 54 (14%) who were positive for both anti-HBs and anti-HBc, 18 (5%) who were positive for anti-HBc alone, and five (1%) who were positive for both HBsAg and anti-HBc. An additional 12 (3%) MSM were positive for anti-HBs alone, suggestive of hepatitis B vaccination. Among the 296 (77%) MSM who lacked evidence of vaccination or infection, 237 (80%) reported having had anal sex or having injected drugs during the preceding 6 months. Of these, 203 (86%) reported receiving care from one or more types of health-care providers, including private physicians; health maintenance organizations; hospitals; or school, community, or health department clinics.

Reported by: M Katz, MD, AIDS Office, San Francisco Dept of Public Health. Seroepidemiology Br, Div of HIV/AIDS Prevention, National Center for Prevention Svcs, CDC.

Editorial Note: Despite the availability of an effective vaccine, the findings in this report suggest that only 3% of young MSM sampled at selected locations in San Francisco and Berkeley during 1992–1993 were adequately vaccinated against hepatitis B. The results also indicate that most (86%) young MSM who were still at risk for HBV infection were not vaccinated, despite receiving medical services from one or more health-care providers.

These findings are consistent with previous reports documenting that health-care providers in a variety of settings miss opportunities to vaccinate clients at risk for HBV infection (1,4,5). For example, at a Houston outpatient clinic for HIV-infected patients, clinic staff failed to prescribe hepatitis B vaccine to all patients for whom the vaccine was clearly indicated (4), and among MSM sampled at a Boston community health center, 84% reported having never been vaccinated against hepatitis B (5).

The findings in this report are subject to at least two limitations. First, these findings are limited to young MSM from the San Francisco Bay area who attended sampled venues. Hepatitis B vaccination coverage may vary among different groups of young MSM in the San Francisco Bay area and among young MSM in other regions of the country. Second, vaccination coverage may be underestimated when determined from serologic data alone. For example, some participants who were vaccinated against hepatitis B may not have developed a satisfactory response, or vaccine-induced anti-HBs may have waned below detectable levels. However, underestimation attributable to vaccine nonresponse or waning immunity is unlikely because of the young age and presumed healthy status (35 participants were HIV-infected) of participants.

Vaccination against hepatitis B is the most effective means of preventing HBV infection. Health-care providers should intensify their efforts to identify MSM and other candidates for vaccination during routine health-care visits (1). Susceptible persons at risk for HBV should be vaccinated, and routine vaccination should be provided to all infants and unvaccinated adolescents aged 11–12 years in accordance with published guidelines (1,6,7). As an integral part of prevention activities, health educators should promote the benefits of vaccination against hepatitis B and refer for evaluation MSM and other persons at risk for HBV infection.

*Hepatitis B — Continued**References*

1. CDC. Hepatitis B virus: a comprehensive strategy for eliminating transmission in the United States through universal childhood vaccination—recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1991;40(no. RR-13).
2. CDC. State and national vaccination coverage levels among children aged 19–35 months—United States, April–December 1994. MMWR 1995;44:613–23.
3. Lemp GF, Hirozawa AM, Givertz D, et al. Seroprevalence of HIV and risk behaviors among young homosexual and bisexual men: the San Francisco/Berkeley young men's survey. JAMA 1994;272:449–54.
4. Rabeneck L, Risser JMH, Murray NGB, et al. Failure of providers to vaccinate HIV-infected men against hepatitis B: a missed opportunity. Am J Gastroenterol 1993;88:2015–8.
5. McCusker J, Hill EM, Mayer KH. Awareness and use of hepatitis B vaccine among homosexual male clients of a Boston community health center. Public Health Rep 1990;105:59–64.
6. CDC. Update: recommendations to prevent hepatitis B virus transmission—United States. MMWR 1995;44:574–5.
7. CDC. Adolescent immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP), the American Academy of Pediatrics (AAP), the American Academy of Family Physicians (AAFP), and the American Medical Association (AMA). MMWR 1996 (in press).

*Notice to Readers***Recommendations of the Advisory Committee on Immunization Practices:
Programmatic Strategies to Increase Vaccination Coverage
by Age 2 Years — Linkage of Vaccination and WIC Services**

This statement by the Advisory Committee on Immunization Practices (ACIP), in collaboration with the U.S. Department of Agriculture's (USDA's) Food and Consumer Service (FCS), presents programmatic strategies to increase vaccination rates among preschool-aged children. This is the first statement to recommend assessment of vaccination status and referral for needed vaccinations of children receiving services from the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC).

Although vaccination coverage levels of first-grade school entrants are >95%, during the 1989–1991 nationwide measles epidemic, coverage levels among urban children aged 2 years were commonly <50% (1). Based on studies conducted during the epidemic, 29%–63% of unvaccinated preschool-aged children with measles either were or had been enrolled in WIC.

WIC is a categorical federal grant program administered by the FCS through state health departments. The program provides supplemental foods, health-care referrals, and nutrition education to low-income pregnant, breastfeeding, or postpartum women; infants; and children aged <5 years. In 1995, approximately 1.8 million infants (44% of the U.S. birth cohort that year) and at least 5 million children aged <5 years participated in WIC monthly. In 1996, the total number of participants aged <5 years is expected to increase to approximately 6 million.

Assessment of vaccination status and referral for needed vaccinations linked with food-voucher issuance (more frequent visits for children not up-to-date and less fre-

ACIP Recommendations — Continued

quent visits for those up-to-date) has been evaluated in New York City and Chicago and is being evaluated in Dallas. Among New York City WIC participants aged 12–59 months, 14% had not been vaccinated against measles; of these unvaccinated children, 79% who received assessment and referral linked with food-voucher issuance were vaccinated against measles within 6 months, compared with 86% who received assessment and escort to the vaccine-delivery site and 54% who received assessment and referral only (2). In Chicago, among groups receiving assessment and referral linked with voucher issuance, the prevalence of up-to-date status at age 2 years increased 36–40 percentage points; among the group receiving assessment and referral only, the prevalence increased only 4 percentage points (3).

CDC and USDA recommend that state and local vaccination and WIC programs collaborate to ensure that young children receive assessment and referral services (4). This recommendation requires that a documented vaccination record (parental recall alone is unreliable) of each WIC client be carefully reviewed as frequently as possible—preferably at each WIC visit. Providers are encouraged to provide vaccination records to parents to facilitate accurate assessment by WIC staff. Clients needing vaccinations should be referred to their usual source of comprehensive health care or be vaccinated on-site if feasible. Programs are encouraged to consider a variety of integrated service-delivery strategies, including the use of incentives for fully vaccinated young children, and outreach for and tracking of undervaccinated children. When possible, WIC and vaccination programs should use automated assessment modules and areawide vaccination data registries to increase the efficiency of assessment.

The ACIP recommends that WIC and vaccination programs assess regularly the vaccination coverage levels of WIC participants and develop new strategies and aggressive outreach procedures in those sites with coverage levels below 90%. In addition, vaccination programs and private providers are encouraged to refer eligible children to obtain WIC nutritional services. The linkage of WIC and vaccination services is expected to increase vaccination levels of high-risk preschoolers and to help sustain high coverage levels. CDC (in collaboration with WIC) is developing guidelines to implement this linkage, and in 1996, a portion of vaccination grant funds have been designated for the implementation.

References

1. Hutchins SS, Gindler JS, Atkinson WL, et al. Preschool children at high risk for measles: opportunities to vaccinate. *Am J Public Health* 1993;83:862–7.
2. Birkhead G, LeBaron CW, Parsons P, et al. The immunization of children enrolled in WIC: the impact of different strategies. *JAMA* 1995;274:312–6.
3. Hutchins SS, Rosenthal J. Results from WIC demonstration projects. In: *Proceedings of the 28th National Immunization Conference*. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service, CDC; 1995:1–4.
4. Ad Hoc Working Group for the Development of Standards for Pediatric Immunization Practices. Standards for pediatric immunization practices. *JAMA* 1993;269:1817–22.

Notice to Readers

Recommendations of the Advisory Committee on Immunization Practices: Programmatic Strategies to Increase Vaccination Rates — Assessment and Feedback of Provider-Based Vaccination Coverage Information

This statement by the Advisory Committee on Immunization Practices (ACIP) presents programmatic strategies to increase vaccination rates. This is the first statement to recommend the use of routine assessment and feedback of provider-based vaccination coverage information.

Routine assessment and feedback of vaccination rates obtained at the provider site is one of the most effective strategies for achieving high, sustainable vaccine coverage. For example, in 1986, the Immunization Program of the Division of Public Health, Georgia Department of Human Resources, initiated a program to assess annually the vaccination records of children enrolled in public health clinics to determine progress toward achieving the national goal of 90% series-complete coverage by age 2 years. During 1986–1994, series-complete vaccination rates at age 2 years among children in public health clinics increased from <40% to approximately 80%—an increase attributed to assessment and feedback, which motivated providers to develop and implement targeted interventions (1,2; CDC, unpublished data, 1996).

Assessment and feedback also has resulted in substantial increases in vaccination rates in public health clinics in Colorado, Illinois, Iowa, Kansas, and South Carolina (3–5; CDC, unpublished data, 1996). Assessments in private and managed-care provider settings in Arizona, Massachusetts, New York, and Washington suggest that this strategy also can improve rates in these settings (4,6–8). Beginning in 1995, all states receiving federal funds for vaccination programs have been required to conduct annual assessments of vaccination rates in public health clinics.* Managed-care organizations also have begun assessing vaccination rates by using data from the Health Plan Employer Data and Information Set (HEDIS) (9). Similarly, the Bureau of Primary Health Care, Health Resources and Services Administration, is introducing assessment and feedback of pediatric vaccination rates as part of quality improvement in community and migrant health centers.

CDC developed the Clinic Assessment Software Application (CASA) to assist in measuring vaccination rates in vaccination practices (10). Only documented antigen-specific vaccine doses and dates of vaccination are used in CASA (or computer vaccination-registry systems with CASA-like functions [11]) to determine provider vaccination rates for all routinely recommended vaccines. Reports produced by CASA also indicate the extent to which vaccination rates can be improved by administering multiple vaccines simultaneously, using accelerated vaccination schedules, encouraging parents to initiate the vaccination series on time, and contacting parents when children are due for or have missed vaccinations. Providers and clinic staff can receive feedback as soon as all vaccination data are entered into CASA. Effective assessment requires complete documentation of antigen-specific doses and dates of vaccination from current and previous providers' records.

*Public Law 103-333.

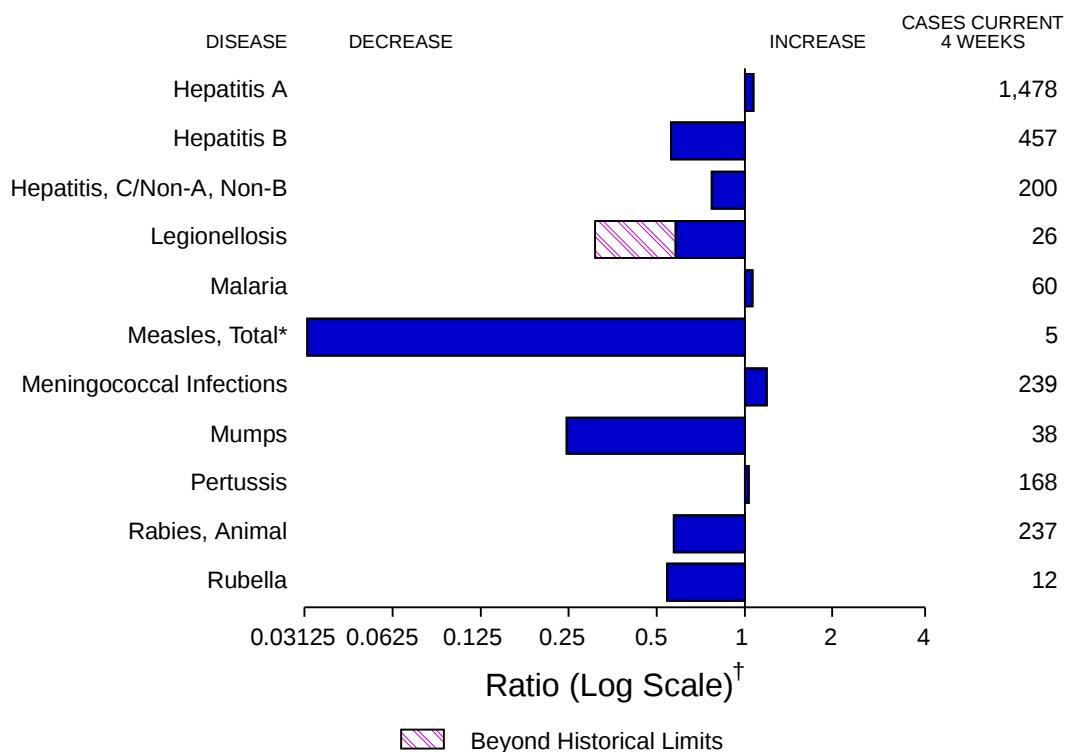
ACIP Recommendations — Continued

A goal of CDC is that vaccination coverage assessments be conducted at all provider sites (public and private) at regular intervals. Assessments provide data about vaccine-delivery practices and vaccine coverage that motivate and assist providers in developing and implementing practices to achieve optimal vaccination rates. Although the cost-effectiveness of vaccinations is well documented, assessment as a specific intervention has not been subjected to such analyses. However, implementation of regular assessments is expected to result in higher vaccination rates and, therefore, be cost-effective.

The ACIP recommends the regular assessment of vaccination rates for individual clinics or providers, including feedback about vaccine-delivery practices, to motivate providers and staff to improve vaccination practices. Implementation of these recommendations can contribute substantially to improving vaccination rates and sustaining high rates in all vaccine-provider sites.

References

1. CDC. Evaluation of vaccination strategies in public clinics—Georgia, 1985–1993. *MMWR* 1995;44:323–5.
2. Dini EF, Chaney M, Moolenaar RL, LeBaron CW. Information as intervention: how Georgia used vaccination coverage data to double public sector vaccination in seven years. *Journal of Public Health Management and Practice* 1996;2:45–9.
3. Galati RV. Assessments and incentives in the public sector: Illinois and Colorado. In: Proceedings of the 29th National Immunization Conference. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service, CDC, 1996 (in press).
4. Houts C, Warming J. Assessments with public and private providers. In: Proceedings of the 29th National Immunization Conference. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service, CDC, 1996 (in press).
5. Greene JE. Assessments using an immunization registry. In: Proceedings of the 29th National Immunization Conference. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service, CDC, 1996 (in press).
6. Thompson RS, Taplin SH, McAfee TA, et al. Primary and secondary prevention services in clinical practice: 20 years' experience in development, implementation, and evaluation. *JAMA* 1995;273:1130–5.
7. Bushnell C. Practice-based immunization assessments: the measures of change. In: Proceedings of the 29th National Immunization Conference. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service, CDC, 1996 (in press).
8. Morrow RW, Gooding AD, Clark C. Improving physicians' preventive health care behavior through peer review and financial incentives. *Archives of Family Medicine* 1995;4:165–9.
9. CDC. Use of a data-based approach by a health maintenance organization to identify and address physician barriers to pediatric vaccination—California, 1995. *MMWR* 1996;45:188–93.
10. CDC. Clinic Assessment Software Application (CASA): user's guide. Atlanta, Georgia: US Department of Health and Human Services, Public Health Service, CDC, 1994.
11. Frame PS. Computerized health maintenance tracking systems: a clinician's guide to necessary and optimal features: a report from the American Cancer Society Advisory Group on Preventive Health Care Reminder Systems. *J Am Board Fam Pract* 1995;8:221–9.

FIGURE I. Selected notifiable disease reports, comparison of 4-week totals ending March 9, 1996, with historical data — United States

*The large apparent decrease in the number of reported cases of measles (total) reflects dramatic fluctuations in the historical baseline.

[†]Ratio of current 4-week total to 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 selected notifiable diseases, United States, cumulative, week ending March 9, 1996 (10th Week)

	Cum. 1996		Cum. 1996
Anthrax	-	HIV infection, pediatric* [§]	49
Brucellosis	7	Plague	-
Cholera	-	Poliomyelitis, paralytic [¶]	-
Congenital rubella syndrome	-	Psittacosis	3
Cryptosporidiosis*	211	Rabies, human	-
Diphtheria	1	Rocky Mountain spotted fever (RMSF)	14
Encephalitis: California*	-	Streptococcal toxic-shock syndrome*	8
eastern equine*	1	Syphilis, congenital**	-
St. Louis*	-	Tetanus	2
western equine*	-	Toxic-shock syndrome	24
Hansen Disease	19	Trichinosis	6
Hantavirus pulmonary syndrome* [†]	-	Typhoid fever	31

*Not notifiable in all states.

[†] Updated weekly from reports to the Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases (NCID).

[§] Updated monthly to the Division of HIV/AIDS Prevention, National Center for Prevention Services (NCPS), last update February 27, 1996.

[¶] No suspected cases of polio reported for 1996.

**Updated quarterly from reports to the Division of STD Prevention, NCPS. First quarter 1996 is not yet available.

-: no reported cases

TABLE II. Cases of selected notifiable diseases, United States, weeks ending March 9, 1996, and March 11, 1995 (10th Week)

Reporting Area	AIDS*		Chlamydia	Escherichia coli O157:H7		Gonorrhea		Hepatitis C/NA,NB		Legionellosis	
	Cum. 1996	Cum. 1995		NETSS†	PHLIS§			Cum. 1996	Cum. 1995	Cum. 1996	Cum. 1995
			Cum. 1996	Cum. 1996	Cum. 1996	Cum. 1995					
UNITED STATES	10,058	11,636	35,978	115	35	50,238	75,110	583	754	114	200
NEW ENGLAND	454	601	1,869	14	3	1,098	1,113	8	17	3	2
Maine	8	15	-	2	-	6	10	-	-	1	-
N.H.	14	11	118	1	1	27	23	-	1	-	-
Vt.	5	1	-	3	2	17	5	4	1	-	-
Mass.	250	287	1,290	5	-	447	640	4	15	1	1
R.I.	17	28	461	2	-	124	116	-	-	1	1
Conn.	160	259	-	1	-	477	319	-	-	N	N
MID. ATLANTIC	2,863	3,074	5,118	18	9	3,774	8,622	68	67	25	22
Upstate N.Y.	324	251	N	11	6	423	1,898	47	24	7	5
N.Y. City	1,615	1,573	-	-	-	713	2,814	1	1	-	1
N.J.	554	805	1,154	4	-	499	748	16	34	2	6
Pa.	370	445	3,964	N	3	2,139	3,162	4	8	16	10
E.N. CENTRAL	822	1,145	7,852	14	2	8,838	15,542	64	64	41	73
Ohio	250	236	1,328	11	-	775	5,104	2	2	18	30
Ind.	91	103	1,789	2	-	1,455	1,521	3	-	9	13
Ill.	315	533	-	1	1	3,287	3,709	5	23	1	12
Mich.	108	215	4,095	-	1	2,909	3,862	54	39	12	9
Wis.	58	58	640	N	-	412	1,346	-	-	1	9
W.N. CENTRAL	254	298	3,946	14	12	3,188	4,207	72	16	8	22
Minn.	56	64	-	1	8	849	592	-	-	-	-
Iowa	23	14	549	4	1	197	293	46	2	2	3
Mo.	93	142	2,008	1	-	1,577	2,467	25	10	1	18
N. Dak.	-	-	-	1	1	-	7	-	-	-	-
S. Dak.	3	-	207	-	-	34	37	-	1	1	-
Nebr.	22	38	388	2	-	57	197	-	1	4	-
Kans.	57	40	794	5	2	474	614	1	2	-	1
S. ATLANTIC	2,485	2,761	9,318	9	1	20,903	22,183	23	56	11	39
Del.	72	69	-	-	-	301	415	-	-	-	-
Md.	198	348	1,030	N	-	2,611	2,797	-	2	2	9
D.C.	125	141	N	-	-	884	1,244	-	-	1	2
Va.	129	271	2,145	N	1	1,810	2,400	1	-	2	2
W. Va.	19	13	-	N	-	99	106	4	14	1	3
N.C.	34	161	-	4	-	4,171	5,084	8	16	3	7
S.C.	93	165	-	1	-	2,189	2,114	1	1	1	5
Ga.	446	439	2,462	1	-	5,601	3,942	-	9	-	5
Fla.	1,369	1,154	3,681	-	-	3,237	4,081	9	14	1	6
E.S. CENTRAL	360	383	1,135	5	1	4,612	9,012	82	292	11	7
Ky.	66	38	-	-	-	800	979	4	7	2	2
Tenn.	141	168	1,101	N	1	1,201	2,427	77	284	4	3
Ala.	90	104	-	-	-	2,446	3,793	1	1	-	1
Miss.	63	73	34	2	-	165	1,813	-	-	5	1
W.S. CENTRAL	956	1,014	1,258	5	1	2,909	6,601	55	22	-	3
Ark.	45	63	-	3	-	548	760	-	-	-	-
La.	225	257	-	N	1	1,570	2,436	9	7	-	1
Okla.	28	57	1,258	1	-	791	331	33	12	-	2
Tex.	658	637	-	1	-	-	3,074	13	3	-	-
MOUNTAIN	254	494	3,402	15	2	1,346	1,741	122	87	5	20
Mont.	3	8	-	-	-	4	23	5	3	-	2
Idaho	4	16	302	6	-	14	27	31	11	-	1
Wyo.	-	4	129	-	-	8	10	38	32	-	-
Colo.	85	214	-	5	2	392	547	4	19	4	11
N. Mex.	20	34	-	-	-	176	239	23	14	-	1
Ariz.	96	88	2,259	N	-	573	554	15	4	-	1
Utah	39	30	254	3	-	49	36	4	3	-	2
Nev.	7	100	458	1	-	130	305	2	1	1	2
PACIFIC	1,610	1,866	2,080	21	4	3,570	6,089	89	133	10	12
Wash.	141	147	1,801	4	4	470	526	15	28	-	-
Oreg.	103	79	-	8	-	64	82	2	6	-	-
Calif.	1,340	1,549	-	7	-	2,872	5,166	41	90	10	9
Alaska	3	29	N	-	-	87	188	2	1	-	-
Hawaii	23	62	267	N	-	77	127	29	8	-	3
Guam	3	-	-	N	-	-	18	-	-	-	-
P.R.	255	638	N	N	U	74	120	20	18	-	-
V.I.	1	10	N	N	U	-	8	-	-	-	-
Amer. Samoa	-	-	-	N	U	-	8	-	-	-	-
C.N.M.I.	-	-	N	N	U	7	5	-	-	-	-

N: Not notifiable U: Unavailable -: no reported cases C.N.M.I.: Commonwealth of Northern Mariana Islands

*Updated monthly to the Division of HIV/AIDS Prevention, National Center for Prevention Services, last update February 27, 1996.

†National Electronic Telecommunications System for Surveillance.

§Public Health Laboratory Information System.

TABLE II. (Cont'd.) Cases of selected notifiable diseases, United States, weeks ending March 9, 1996, and March 11, 1995 (10th Week)

Reporting Area	Lyme Disease		Malaria		Meningococcal Disease		Syphilis (Primary & Secondary)		Tuberculosis		Rabies, Animal	
	Cum. 1996	Cum. 1995	Cum. 1996	Cum. 1995	Cum. 1996	Cum. 1995	Cum. 1996	Cum. 1995	Cum. 1996	Cum. 1995	Cum. 1996	Cum. 1995
UNITED STATES	534	722	155	182	725	667	1,777	3,081	2,239	2,507	625	1,068
NEW ENGLAND	30	30	4	7	25	44	28	40	54	46	75	293
Maine	-	1	1	-	7	3	-	-	4	-	-	-
N.H.	-	2	-	1	1	8	-	1	2	1	9	40
Vt.	-	1	1	-	1	5	-	-	-	-	19	39
Mass.	8	4	2	-	8	13	16	14	22	19	21	138
R.I.	17	-	-	2	-	-	-	-	10	7	10	9
Conn.	5	22	-	4	8	15	12	25	16	19	16	67
MID. ATLANTIC	450	560	40	40	52	67	55	205	357	452	122	265
Upstate N.Y.	192	213	12	5	15	24	-	22	41	37	58	165
N.Y. City	128	28	19	19	5	8	18	117	191	251	-	-
N.J.	-	88	6	12	17	22	16	34	92	89	30	50
Pa.	130	231	3	4	15	13	21	32	33	75	34	50
E.N. CENTRAL	7	6	17	24	88	102	362	519	364	288	5	2
Ohio	5	4	3	1	42	27	151	178	57	38	2	1
Ind.	2	1	2	1	6	15	47	48	28	10	-	-
Ill.	-	1	3	18	25	31	95	189	232	171	-	1
Mich.	-	-	7	2	6	17	40	63	39	64	-	-
Wis.	-	-	2	2	9	12	29	41	8	5	3	-
W.N. CENTRAL	19	17	3	6	64	34	101	169	62	74	54	49
Minn.	-	-	-	3	3	6	25	8	14	15	3	5
Iowa	10	-	1	-	16	7	4	13	8	15	28	13
Mo.	-	7	1	3	24	14	69	144	24	30	4	7
N. Dak.	-	-	-	-	1	-	-	-	1	-	5	5
S. Dak.	-	-	-	-	2	-	-	-	6	-	10	12
Nebr.	-	-	-	-	8	2	3	4	-	-	1	-
Kans.	9	10	1	-	10	5	-	-	9	14	3	7
S. ATLANTIC	21	79	26	45	123	109	608	817	246	391	308	319
Del.	1	9	2	1	1	1	10	5	-	10	10	14
Md.	14	56	12	13	14	1	100	82	42	82	91	76
D.C.	-	-	1	3	2	1	26	33	11	19	-	1
Va.	-	2	5	9	15	11	85	126	1	6	75	58
W. Va.	2	5	-	-	4	-	1	-	16	15	11	17
N.C.	4	3	4	4	19	19	189	218	40	23	64	68
S.C.	-	4	-	-	17	13	80	110	38	60	6	21
Ga.	-	-	2	5	36	34	63	149	-	60	44	55
Fla.	-	-	-	10	15	29	54	94	98	116	7	9
E.S. CENTRAL	-	7	-	1	51	39	430	710	216	184	8	41
Ky.	-	1	-	-	8	13	33	47	42	35	-	3
Tenn.	-	4	-	-	3	6	84	148	44	72	-	22
Ala.	-	-	-	1	19	12	123	129	72	77	8	16
Miss.	-	2	-	-	21	8	190	386	58	-	-	-
W.S. CENTRAL	-	9	6	2	91	70	169	433	92	271	3	27
Ark.	-	-	-	1	10	7	44	95	15	30	-	15
La.	-	-	-	-	17	8	101	202	-	-	-	9
Okla.	-	9	-	-	4	9	24	33	14	32	3	3
Tex.	-	-	6	1	60	46	-	103	63	209	-	-
MOUNTAIN	-	1	12	12	52	53	23	62	83	81	9	8
Mont.	-	-	-	1	1	1	-	3	-	-	-	4
Idaho	-	-	1	-	6	2	1	-	3	2	-	-
Wyo.	-	-	1	-	3	1	1	-	-	-	5	-
Colo.	-	-	5	6	6	13	11	29	12	3	-	-
N. Mex.	-	-	1	3	12	14	-	10	7	17	1	-
Ariz.	-	-	1	1	17	18	7	11	48	51	2	4
Utah	-	-	2	1	3	2	-	2	-	7	-	-
Nev.	-	1	1	-	4	2	3	7	13	1	1	-
PACIFIC	7	13	47	45	179	149	1	126	765	720	41	64
Wash.	-	-	-	5	16	16	-	3	43	43	-	-
Oreg.	4	1	4	4	32	33	1	4	18	4	-	-
Calif.	3	12	40	32	126	99	-	119	662	625	37	62
Alaska	-	-	-	1	3	-	-	-	15	17	4	2
Hawaii	-	-	3	3	2	1	-	-	27	31	-	-
Guam	-	-	-	-	-	1	-	1	-	4	-	-
P.R.	-	-	-	-	-	9	33	55	-	-	6	11
V.I.	-	-	-	-	-	-	-	-	-	-	-	-
Amer. Samoa	-	-	-	-	-	-	-	-	-	2	-	-
C.N.M.I.	-	-	-	-	-	-	-	-	-	5	-	-

N: Not notifiable

U: Unavailable

-: no reported cases

TABLE III. Cases of selected notifiable diseases preventable by vaccination, United States, weeks ending March 9, 1996, and March 11, 1995 (10th Week)

Reporting Area	<i>H. influenzae</i> , invasive		Hepatitis (viral), by type				Measles (Rubeola)			
	Cum. 1996*	Cum. 1995	A		B		Indigenous		Imported†	
			Cum. 1996	Cum. 1995	Cum. 1996	Cum. 1995	1996	Cum. 1996	1996	Cum. 1996
UNITED STATES	250	272	4,074	4,555	1,149	1,539	-	12	-	1
NEW ENGLAND	7	11	39	29	6	50	-	4	-	-
Maine	-	-	5	6	2	2	-	-	-	-
N.H.	5	1	3	1	-	4	-	-	-	-
Vt.	-	1	-	1	2	1	-	1	-	-
Mass.	2	3	22	7	1	10	-	3	-	-
R.I.	-	-	2	7	1	6	-	-	-	-
Conn.	-	6	7	7	-	27	-	-	-	-
MID. ATLANTIC	31	28	231	213	194	172	-	1	-	-
Upstate N.Y.	9	8	57	39	59	48	-	-	-	-
N.Y. City	2	4	147	90	117	29	-	1	-	-
N.J.	13	6	-	44	-	64	-	-	-	-
Pa.	7	10	27	40	18	31	-	-	-	-
E.N. CENTRAL	40	54	373	745	129	230	-	-	-	-
Ohio	25	30	200	432	21	19	-	-	-	-
Ind.	1	4	77	35	16	48	-	-	-	-
Ill.	12	17	24	150	10	68	-	-	-	-
Mich.	1	3	56	77	78	81	-	-	-	-
Wis.	1	-	16	51	4	14	-	-	-	-
W.N. CENTRAL	10	11	361	202	102	117	-	-	-	-
Minn.	-	3	7	12	2	5	-	-	-	-
Iowa	6	1	105	10	40	13	-	-	-	-
Mo.	4	6	163	146	43	87	-	-	-	-
N. Dak.	-	-	5	1	-	1	-	-	-	-
S. Dak.	-	-	20	1	-	-	-	-	-	-
Nebr.	-	-	32	14	3	7	-	-	-	-
Kans.	-	1	29	18	14	4	-	-	-	-
S. ATLANTIC	51	66	144	192	171	208	-	1	-	-
Del.	-	-	2	3	-	1	-	-	-	-
Md.	14	24	40	40	54	47	-	1	-	-
D.C.	-	-	5	1	3	8	-	-	-	-
Va.	2	9	21	37	25	15	-	-	-	-
W. Va.	-	1	4	6	6	13	-	-	-	-
N.C.	6	10	20	20	57	62	-	-	-	-
S.C.	2	-	16	5	6	7	-	-	-	-
Ga.	27	8	-	22	-	17	-	-	-	-
Fla.	-	14	36	58	20	38	-	-	-	-
E.S. CENTRAL	6	3	122	275	29	192	-	-	-	-
Ky.	2	1	5	18	12	20	-	-	-	-
Tenn.	-	-	19	211	6	145	-	-	-	-
Ala.	3	2	32	28	11	27	-	-	-	-
Miss.	1	-	66	18	-	-	-	-	-	-
W.S. CENTRAL	8	10	663	326	80	73	-	-	-	-
Ark.	-	1	115	14	9	1	-	-	-	-
La.	-	-	11	10	7	8	-	-	-	-
Okla.	8	7	340	109	19	14	-	-	-	-
Tex.	-	2	197	193	45	50	-	-	-	-
MOUNTAIN	27	28	634	834	156	114	-	-	-	-
Mont.	-	-	13	13	-	4	-	-	-	-
Idaho	1	1	93	94	22	18	-	-	-	-
Wyo.	9	1	5	27	5	2	-	-	-	-
Colo.	3	4	24	112	9	22	-	-	-	-
N. Mex.	6	5	117	173	79	39	-	-	-	-
Ariz.	4	6	179	168	14	13	-	-	-	-
Utah	2	3	167	219	20	11	-	-	-	-
Nev.	2	8	36	28	7	5	-	-	-	-
PACIFIC	70	61	1,507	1,739	282	383	-	6	-	1
Wash.	-	3	103	89	16	24	-	4	-	-
Oreg.	9	8	218	348	19	22	-	-	-	-
Calif.	59	48	1,147	1,271	244	331	-	1	-	-
Alaska	-	-	19	14	2	2	-	1	-	-
Hawaii	2	2	20	17	1	4	-	-	-	1
Guam	-	-	-	-	-	-	U	-	U	-
P.R.	-	3	16	5	82	33	-	-	-	-
V.I.	-	-	-	-	-	1	U	-	U	-
Amer. Samoa	-	-	-	4	-	-	U	-	U	-
C.N.M.I.	10	-	1	5	3	-	U	-	U	-

*Of 54 cases among children aged <5 years, serotype was reported for 13 and of those, 1 was type B.

†For imported measles, cases include only those resulting from importation from other countries.

N: Not notifiable U: Unavailable -: no reported cases

TABLE III. (Cont'd.) Cases of selected notifiable diseases preventable by vaccination, United States, weeks ending March 9, 1996, and March 11, 1995 (10th Week)

Reporting Area	Measles (Rubeola), cont'd.		Mumps			Pertussis			Rubella		
	Total		1996	Cum. 1996	Cum. 1995	1996	Cum. 1996	Cum. 1995	1996	Cum. 1996	Cum. 1995
	Cum. 1996	Cum. 1995									
UNITED STATES	13	104	9	105	151	52	358	510	1	25	12
NEW ENGLAND	4	3	-	-	3	4	60	79	-	2	2
Maine	-	-	-	-	2	-	2	8	-	-	-
N.H.	-	-	-	-	-	4	10	5	-	-	1
Vt.	1	-	-	-	-	-	6	2	-	-	-
Mass.	3	1	-	-	-	-	42	61	-	-	1
R.I.	-	2	-	-	-	-	-	-	-	-	-
Conn.	-	-	-	-	1	-	-	3	-	2	-
MID. ATLANTIC	1	1	2	14	22	10	48	42	-	3	-
Upstate N.Y.	-	-	-	5	6	3	31	24	-	2	-
N.Y. City	1	-	-	2	2	-	8	9	-	1	-
N.J.	-	1	-	-	3	-	-	3	-	-	-
Pa.	-	-	2	7	11	7	9	6	-	-	-
E.N. CENTRAL	-	-	2	28	24	-	47	57	-	-	-
Ohio	-	-	1	14	11	-	35	27	-	-	-
Ind.	-	-	1	5	4	-	3	6	-	-	-
Ill.	-	-	-	-	-	-	-	-	-	-	-
Mich.	-	-	-	9	9	-	7	22	-	-	-
Wis.	-	-	-	-	-	-	2	2	-	-	-
W.N. CENTRAL	-	1	-	2	10	1	3	20	-	-	-
Minn.	-	-	-	-	-	-	1	-	-	-	-
Iowa	-	-	-	-	1	1	2	1	-	-	-
Mo.	-	1	-	-	8	-	-	7	-	-	-
N. Dak.	-	-	-	2	-	-	-	1	-	-	-
S. Dak.	-	-	-	-	-	-	-	4	-	-	-
Nebr.	-	-	-	-	1	-	-	1	-	-	-
Kans.	-	-	-	-	-	-	-	6	-	-	-
S. ATLANTIC	1	-	1	11	23	2	22	42	-	-	1
Del.	-	-	-	-	-	-	-	2	-	-	-
Md.	1	-	1	5	5	1	16	-	-	-	-
D.C.	-	-	-	-	-	-	-	1	-	-	-
Va.	-	-	-	2	4	-	-	-	-	-	-
W. Va.	-	-	-	-	-	-	-	-	-	-	-
N.C.	-	-	-	-	10	-	-	30	-	-	-
S.C.	-	-	-	3	1	-	2	7	-	-	-
Ga.	-	-	-	1	-	-	1	-	-	-	-
Fla.	-	-	-	-	3	1	3	2	-	-	1
E.S. CENTRAL	-	-	2	5	5	1	7	14	-	-	-
Ky.	-	-	-	-	-	-	4	-	-	-	-
Tenn.	-	-	-	-	-	-	-	2	-	-	-
Ala.	-	-	-	3	2	-	1	12	-	-	-
Miss.	-	-	2	2	3	1	2	-	N	N	N
W.S. CENTRAL	-	-	-	3	9	-	3	13	-	-	1
Ark.	-	-	-	-	2	-	2	-	-	-	-
La.	-	-	-	3	2	-	1	-	-	-	-
Okla.	-	-	-	-	-	-	-	-	-	-	-
Tex.	-	-	-	-	5	-	-	13	-	-	1
MOUNTAIN	-	51	1	9	7	7	43	162	-	-	2
Mont.	-	-	-	-	-	-	2	2	-	-	-
Idaho	-	-	-	-	-	2	13	47	-	-	-
Wyo.	-	-	-	-	-	-	-	-	-	-	-
Colo.	-	17	-	-	-	4	4	32	-	-	-
N. Mex.	-	26	N	N	N	1	13	5	-	-	-
Ariz.	-	7	1	1	-	-	2	73	-	-	2
Utah	-	-	-	-	1	-	1	2	-	-	-
Nev.	-	1	-	8	6	-	8	1	-	-	-
PACIFIC	7	48	1	33	48	27	125	81	1	20	6
Wash.	4	-	-	2	2	-	10	10	-	1	-
Oreg.	-	-	N	N	N	1	16	1	-	-	-
Calif.	1	48	1	23	40	26	95	68	1	18	6
Alaska	1	-	-	1	5	-	-	-	-	-	-
Hawaii	1	-	-	7	1	-	4	2	-	1	-
Guam	-	-	U	-	-	U	-	-	U	-	-
P.R.	-	-	1	1	1	-	-	3	-	-	-
V.I.	-	-	U	-	1	U	-	-	U	-	-
Amer. Samoa	-	-	U	-	-	U	-	-	U	-	-
C.N.M.I.	-	-	U	-	-	U	-	-	U	-	-

N: Not notifiable

U: Unavailable

-: no reported cases

**TABLE IV. Deaths in 121 U.S. cities,* week ending
March 9, 1996 (10th Week)**

Reporting Area	All Causes, By Age (Years)						P&I†	Total	Reporting Area	All Causes, By Age (Years)						P&I†	Total
	All Ages	≥65	45-64	25-44	1-24	<1				All Ages	≥65	45-64	25-44	1-24	<1		
NEW ENGLAND	597	423	115	37	8	14	31		S. ATLANTIC	1,569	966	328	195	44	34	118	
Boston, Mass.	156	111	28	11	3	3	5		Atlanta, Ga.	171	100	38	21	4	8	7	
Bridgeport, Conn.	39	25	8	5	-	1	-		Baltimore, Md.	373	210	78	73	10	1	43	
Cambridge, Mass.	19	15	3	1	-	-	-		Charlotte, N.C.	91	60	18	7	1	4	8	
Fall River, Mass.	25	21	2	2	-	-	1		Jacksonville, Fla.	138	82	35	19	2	-	5	
Hartford, Conn.	73	42	20	5	2	4	1		Miami, Fla.	104	52	36	8	6	2	2	
Lowell, Mass.	30	25	3	2	-	-	1		Norfolk, Va.	54	36	9	3	-	6	4	
Lynn, Mass.	15	13	2	-	-	-	2		Richmond, Va.	71	50	13	5	-	3	4	
New Bedford, Mass.	20	15	4	-	-	1	-		Savannah, Ga.	53	34	11	2	4	2	8	
New Haven, Conn.	51	34	13	-	1	3	3		St. Petersburg, Fla.	56	42	6	6	2	-	5	
Providence, R.I.	35	26	6	3	-	-	4		Tampa, Fla.	197	143	30	18	4	2	22	
Somerville, Mass.	4	2	1	1	-	-	1		Washington, D.C.	242	142	50	33	11	6	10	
Springfield, Mass.	55	38	13	3	1	-	8		Wilmington, Del.	19	15	4	-	-	-	-	
Waterbury, Conn.	28	23	2	1	-	2	-		E.S. CENTRAL	850	577	169	63	20	21	81	
Worcester, Mass.	47	33	10	3	1	-	5		Birmingham, Ala.	99	60	22	9	2	6	1	
MID. ATLANTIC	2,492	1,629	485	271	63	43	125		Chattanooga, Tenn.	47	31	9	3	2	2	3	
Albany, N.Y.	40	31	5	3	1	-	1		Knoxville, Tenn.	78	57	14	4	1	2	14	
Allentown, Pa.	19	16	3	-	-	-	1		Lexington, Ky.	90	58	19	8	-	5	7	
Buffalo, N.Y.	80	62	14	2	1	1	-		Memphis, Tenn.	247	175	45	21	3	3	30	
Camden, N.J.	32	20	8	2	-	2	2		Mobile, Ala.	96	69	15	5	6	1	9	
Elizabeth, N.J.	21	14	5	2	-	-	-		Montgomery, Ala.	48	29	14	2	2	1	6	
Erie, Pa.‡	32	26	5	1	-	-	3		Nashville, Tenn.	145	98	31	11	4	1	11	
Jersey City, N.J.	46	30	12	3	1	-	2		W.S. CENTRAL	1,666	1,101	327	145	58	35	91	
New York City, N.Y.	1,226	792	234	151	31	18	55		Austin, Tex.	85	59	11	11	3	1	9	
Newark, N.J.	73	34	19	9	5	6	6		Baton Rouge, La.	52	31	8	8	4	1	3	
Paterson, N.J.	22	12	4	4	1	1	2		Corpus Christi, Tex.	45	33	7	1	1	3	2	
Philadelphia, Pa.	500	291	110	70	18	10	18		Dallas, Tex.	231	135	51	29	13	3	10	
Pittsburgh, Pa.§	47	30	9	4	2	2	3		El Paso, Tex.	100	67	18	10	4	1	4	
Reading, Pa.	13	11	-	1	1	-	4		Ft. Worth, Tex.	101	61	21	7	7	5	-	
Rochester, N.Y.	133	108	14	9	2	-	16		Houston, Tex.	366	242	84	29	8	3	28	
Schenectady, N.Y.	22	18	4	-	-	-	4		Little Rock, Ark.	97	67	18	7	3	2	6	
Scranton, Pa.§	29	23	5	1	-	-	-		New Orleans, La.	160	104	29	15	4	8	-	
Syracuse, N.Y.	82	58	19	3	-	2	4		San Antonio, Tex.	231	165	35	21	5	5	10	
Trenton, N.J.	28	19	7	2	-	-	2		Shreveport, La.	56	38	14	1	3	-	10	
Utica, N.Y.	20	16	3	1	-	-	-		Tulsa, Okla.	142	99	31	6	3	3	9	
Yonkers, N.Y.	27	18	5	3	-	1	2		MOUNTAIN	959	652	174	87	30	14	80	
E.N. CENTRAL	2,079	1,408	386	184	44	57	146		Albuquerque, N.M.	87	58	20	5	3	1	5	
Akron, Ohio	50	34	4	6	1	5	-		Colo. Springs, Colo.	62	38	16	7	1	-	3	
Canton, Ohio	38	31	3	2	1	1	6		Denver, Colo.	122	84	21	15	1	1	16	
Chicago, Ill.	422	242	104	50	11	15	40		Las Vegas, Nev.	202	132	44	14	8	4	13	
Cincinnati, Ohio	113	82	22	3	2	4	11		Ogden, Utah	31	25	5	1	-	-	1	
Cleveland, Ohio	166	102	36	19	3	6	2		Phoenix, Ariz.	181	122	26	18	8	5	20	
Columbus, Ohio	173	126	23	19	4	1	16		Pueblo, Colo.	27	20	4	1	2	-	3	
Dayton, Ohio	136	97	22	14	2	1	6		Salt Lake City, Utah	106	67	20	15	3	1	8	
Detroit, Mich.	200	135	37	18	5	5	11		Tucson, Ariz.	141	106	18	11	4	2	11	
Evansville, Ind.	41	30	6	3	1	1	5		PACIFIC	1,896	1,332	312	175	38	38	143	
Fort Wayne, Ind.	47	36	6	3	2	-	1		Berkeley, Calif.	21	16	5	-	-	-	1	
Gary, Ind.	U	U	U	U	U	U	U		Fresno, Calif.	114	88	17	4	4	1	9	
Grand Rapids, Mich.	61	42	9	5	2	3	6		Glendale, Calif.	29	21	5	2	-	1	2	
Indianapolis, Ind.	147	90	39	14	2	2	8		Honolulu, Hawaii	87	58	19	6	3	1	7	
Madison, Wis.	72	46	11	7	3	5	9		Long Beach, Calif.	90	58	9	11	8	4	12	
Milwaukee, Wis.	108	82	18	4	-	4	8		Los Angeles, Calif.	630	449	92	72	4	13	27	
Peoria, Ill.	40	30	5	2	1	2	5		Pasadena, Calif.	30	18	6	3	2	1	1	
Rockford, Ill.	37	22	9	4	1	1	4		Portland, Ore.	146	109	21	12	1	3	8	
South Bend, Ind.	43	37	5	1	-	-	3		Sacramento, Calif.	U	U	U	U	U	U	U	
Toledo, Ohio	115	92	18	4	1	-	5		San Diego, Calif.	162	110	28	16	4	4	24	
Youngstown, Ohio	70	52	9	6	2	1	-		San Francisco, Calif.	158	100	35	18	3	1	15	
W.N. CENTRAL	699	517	104	44	15	16	42		San Jose, Calif.	188	137	31	12	4	4	21	
Des Moines, Iowa	77	60	12	2	2	1	7		Santa Cruz, Calif.	35	28	5	2	-	-	6	
Duluth, Minn.	U	U	U	U	U	U	U		Seattle, Wash.	140	92	29	13	3	3	2	
Kansas City, Kans.	32	22	3	4	2	1	1		Spokane, Wash.	66	48	10	4	2	2	8	
Kansas City, Mo.	119	82	20	9	4	1	9		Tacoma, Wash.	U	U	U	U	U	U	U	
Lincoln, Nebr.	44	37	4	2	-	1	3		TOTAL	12,807†	8,605	2,400	1,201	320	272	857	
Minneapolis, Minn.	102	82	10	6	1	3	10										
Omaha, Nebr.	63	48	10	3	1	1	3										
St. Louis, Mo.	124	91	20	5	2	6	6										
St. Paul, Minn.	56	39	12	3	1	1	-										
Wichita, Kans.	82	56	13	10	2	1	3										

*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.

U: Unavailable - : no reported cases

Contributors to the Production of the *MMWR* (Weekly)

Weekly Notifiable Disease Morbidity Data and 121 Cities Mortality Data

Denise Koo, M.D., M.P.H.

Deborah A. Adams

Patsy A. Hall

Carol M. Knowles

Sarah H. Landis

Myra A. Montalbano

Graphics Support

Sandra L. Ford

Beverly J. Holland

Desktop Publishing

Jolene W. Altman

Morie M. Higgins

Peter M. Jenkins

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy on Friday of each week, send an e-mail message to lists@list.cdc.gov. The body content should read *subscribe mmwr-toc*. Electronic copy also is available from CDC's World-Wide Web server at <http://www.cdc.gov/> or from CDC's file transfer protocol server at <ftp.cdc.gov>. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 512-1800.

Data in the weekly *MMWR* 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 following Friday. Address inquiries about the *MMWR* Series, including material to be considered for publication, to: Editor, *MMWR* Series, Mailstop C-08, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone (404) 332-4555.

All material in the *MMWR* Series is in the public domain and may be used and reprinted without permission; citation as to source, however, is appreciated.

Director, Centers for Disease Control
and Prevention
David Satcher, M.D., Ph.D.
Deputy Director, Centers for Disease Control
and Prevention
Claire V. Broome, M.D.
Director, Epidemiology Program Office
Stephen B. Thacker, M.D., M.Sc.

Editor, *MMWR* Series
Richard A. Goodman, M.D., M.P.H.
Managing Editor, *MMWR* (weekly)
Karen L. Foster, M.A.
Writers-Editors, *MMWR* (weekly)
David C. Johnson
Darlene D. Rumph-Person
Caran R. Wilbanks

☆ U.S. Government Printing Office: 1996-733-175/27045 Region IV