

**MMWR**<sup>TM</sup>  
**MORBIDITY AND MORTALITY  
WEEKLY REPORT**

- 177 Update: Influenza Activity — United States, 1998–99 Season
- 181 Decrease in Infant Mortality and Sudden Infant Death Syndrome Among Northwest American Indians and Alaskan Natives — Pacific Northwest, 1985–1996
- 185 Preterm Singleton Births — United States, 1989–1996
- 189 Incidence of Foodborne Illnesses: Preliminary Data from the Foodborne Diseases Active Surveillance Network (FoodNet) — United States, 1998
- 194 Notice to Readers

**Update: Influenza Activity — United States, 1998–99 Season**

This report summarizes influenza activity in the United States from October 4, 1998, through February 27, 1999. It also presents results of an investigation of an influenza outbreak among staff and residents at one long-term-care facility (LTCF), and estimates the 1998–99 influenza vaccine effectiveness against the circulating influenza A(H3N2) viruses at that facility. Based on influenza surveillance data, influenza activity in the United States began to increase in mid-January 1999 and remained elevated in most regions of the country through the week ending February 27.

The percentage of patient visits to approximately 350 sentinel physicians for influenza-like illness (ILI) increased from baseline levels of 0–3% during the week ending January 23 and has remained elevated for 6 consecutive weeks. For the week ending February 27, 4% of patient visits were for ILI. Visits for ILI were above baseline levels in all influenza surveillance regions for the week ending February 27 except the mid-Atlantic and east south central regions, which had levels of 1% and 3%, respectively.

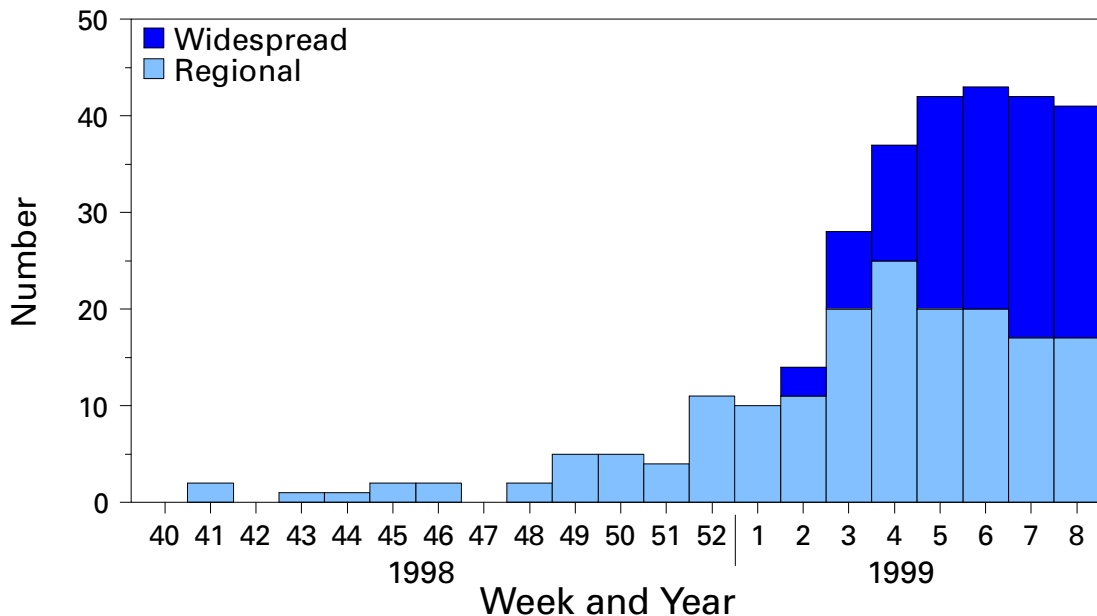
Since the week ending January 23, at least 25 states have reported either widespread or regional activity each week (Figure 1). The highest number of states reporting either widespread or regional activity during any 1 week was 43 during the week ending February 13. State and territorial epidemiologists in 41 states and the District of Columbia reported either widespread or regional influenza activity\* for the week ending February 27.

The percentage of deaths attributed to pneumonia and influenza (P&I) among 122 U.S. cities was 8.1% for the week ending February 27, which is above the epidemic threshold<sup>†</sup> of 7.5%. Mortality from P&I exceeded the epidemic threshold for 3 consecutive weeks beginning the week ending February 13.

From October 4, 1998 through February 27, 1999, the World Health Organization and the National Respiratory Enteric Virus Surveillance System collaborating

\* Levels of activity are 1) *no activity*; 2) *sporadic*—sporadically occurring ILI or culture-confirmed influenza with no outbreaks detected; 3) *regional*—outbreaks of ILI or culture-confirmed influenza in counties with a combined population of <50% of the state's total population; and 4) *widespread*—outbreaks of ILI or culture-confirmed influenza in counties with a combined population of ≥50% of the state's total population.

<sup>†</sup>The epidemic threshold is 1.645 standard deviations above the seasonal baseline. The expected seasonal baseline is projected using a robust regression procedure in which a periodic regression model is applied to observed percentages of deaths from P&I since 1983.

*Influenza Activity — Continued***FIGURE 1. Number of state and territorial epidemiologists reporting widespread or regional influenza activity\*, by week and year — United States, October 4, 1998–February 27, 1999**

\*Levels of activity are 1) *no activity*; 2) *sporadic*—sporadically occurring influenza-like illness (ILI) or culture-confirmed influenza with no outbreaks detected; 3) *regional*—outbreaks of ILI or culture-confirmed influenza in counties with a combined population of <50% of the state's total population; and 4) *widespread*—outbreaks of ILI or culture-confirmed influenza in counties with a combined population of  $\geq$ 50% of the state's total population.

laboratories in the United States reported detection of influenza in 6529 (12%) of 52,355 clinical specimens submitted for respiratory virus testing. Of the influenza-positive specimens, 5170 (79%) were type A and 1359 (21%) were type B. Of the 5170 influenza A isolates, 1275 (25%) were H3N2 viruses, 12 (0.2%) were H1N1 viruses, and 3883 (75%) were not subtyped. In the west north central, east north central, and east south central regions, 35%–46% of the influenza isolates were type B.

Of 169 influenza A(H3N2) isolates collected during October 4, 1998–February 27, 1999, that were antigenically characterized at CDC, all were characterized as A/Sydney/5/97-like viruses, the H3N2 virus strain contained in the 1998–99 influenza vaccine. Two influenza A(H1N1) isolates were characterized as A/Bayern/7/95-like viruses, antigenically distinct from A/Beijing/262/95, the 1998–99 H1N1 vaccine strain; however, A/Beijing/262/95 produced high titers of antibodies that cross-react with A/Bayern/7/95. All 51 influenza type B isolates were antigenically similar to B/Beijing/184/93, the recommended type B vaccine strain.

**Long-Term-Care Facility Outbreak**

The California Department of Health Services (CDHS) requires that all LTCFs report respiratory illness outbreaks to the state or local health department. As of February 27, CDHS had received five reports of culture-confirmed influenza outbreaks among the

*Influenza Activity — Continued*

approximately 1200 LTCFs in the state. Following is a result of an investigation of one of these outbreaks.

On December 31, 1998, a LTCF notified the Santa Clara County Public Health Department of an ILI outbreak among residents of two units in one of the facility's four buildings. Nasopharyngeal swab specimens from eight of 10 ill residents were positive for influenza A by direct fluorescent antibody testing. The outbreak investigation included active surveillance for ILI (temperature  $\geq 100$  F [ $\geq 38$  C] and cough or sore throat or rhinitis), viral culture of nasopharyngeal swab samples collected from selected ill residents and staff, and collection of vaccination and illness histories from residents and staff in the two affected units. Vaccine effectiveness against ILI was calculated as 1 minus relative risk.

Residents in this facility are assigned to different buildings according to the level of care required. The most debilitated residents, most of whom are bedridden and require complete care, reside in Building 1. During the fall, residents in all four buildings (n=524) received influenza vaccination, except residents with medical contraindications. Of the 1200 staff members offered vaccine, approximately 200 (17%) were vaccinated at the facility, and some may have been vaccinated by outside providers.

The first cases of ILI occurred during December 21–December 28, 1998, among five unvaccinated nurses who worked in two adjacent units in Building 1. From December 29, 1998, through January 17, 1999, additional ILI cases developed among residents and staff from those two units and others in Building 1 (Figure 2). Thirty-four (11%) of 309 staff members and 25 (13%) of 192 residents of Building 1 developed ILI. Three residents were hospitalized, and two died, including one who was not vaccinated because of a history of egg allergy. Forty-nine of the 50 residents (median age: 30 years [range: 13–87 years]) residing in the two initially affected units had been vaccinated before the outbreak compared with 12 (26%) of the 47 staff members (median age: 44 years [range: 20–68 years]).

Vaccine effectiveness against ILI was 72% (95% CI: -1.3–92.4) among the 47 staff members. Vaccine effectiveness was not estimated for residents because of the small number of unvaccinated persons. Four influenza A(H3N2) isolates obtained from ill residents were antigenically characterized as A/Sydney/5/97-like viruses.

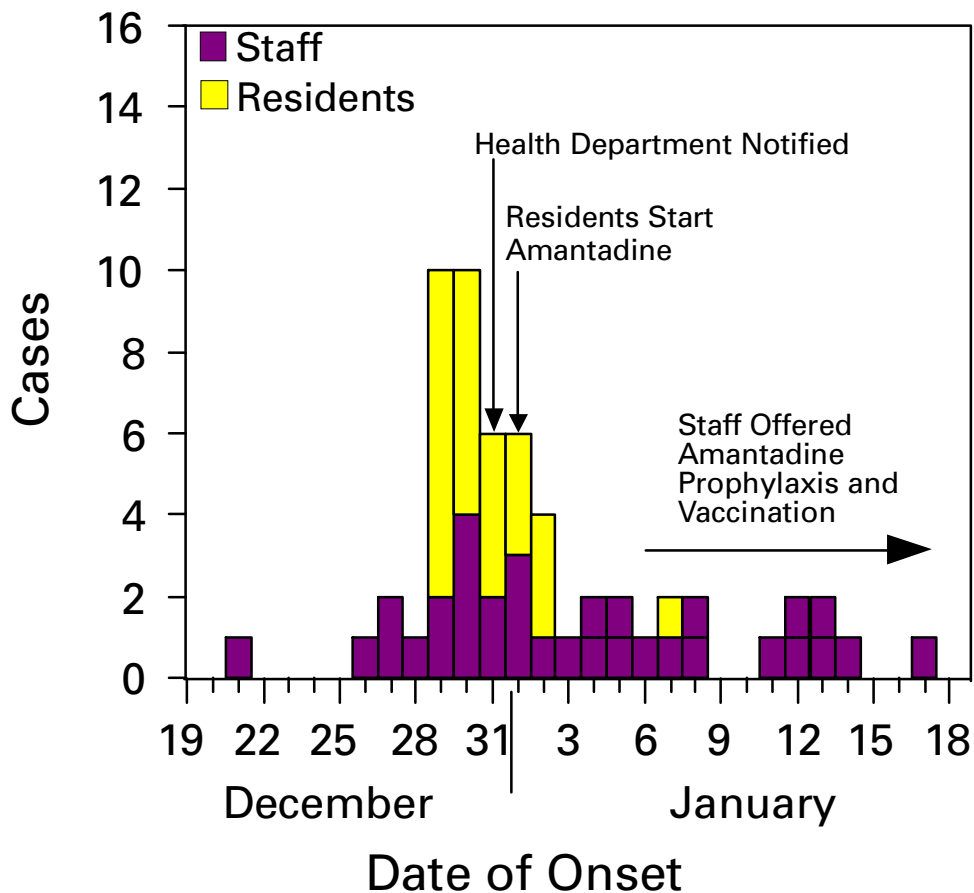
Outbreak-control measures included cohorting ill residents and initiating droplet precautions (1) and administering amantadine for prophylaxis of non-ill residents and treatment of ill residents. Unvaccinated staff were offered amantadine prophylaxis and influenza vaccine. Ill staff were discouraged from coming to work, and ill visitors were asked to postpone their visits.

*Reported by: SH Cody, MD, AF Bolding, M Fenstersheib, MD, GS Olivas, PhD, Santa Clara County Public Health Dept; C O'Malley, PhD, N Smith, MD, Immunization Br, M Hendry, DSc, Respiratory, AIDS, and Support Section, Viral and Rickettsial Disease Laboratory; S Waterman, MD, State Epidemiologist, California Dept of Health Svcs. Participating state and territorial epidemiologists and state public health laboratory directors. National Respiratory Enteric Virus Surveillance System collaborating laboratories. World Health Organization collaborating laboratories. Sentinel Physicians Influenza Surveillance System. WHO Collaborating Center for Reference and Research on Influenza, Influenza Br, and Respiratory Enterovirus Br, Div of Viral and Rickettsial Diseases, National Center for Infectious Diseases, CDC.*

**Editorial Note:** Almost all states have reported either regional or widespread influenza activity this influenza season. Although only 21% of influenza isolates have been type B, influenza B viruses have been detected in all influenza surveillance regions.

*Influenza Activity — Continued*

**FIGURE 2. Influenza-like illness among residents and staff of a long-term-care facility — Santa Clara County, California, December 19, 1998–January 18, 1999**



Influenza A/Sydney/5/97 (H3N2)-like virus appears to be the predominant strain so far this influenza season.

The influenza A outbreak described in this report illustrates several points. First, influenza outbreaks can occur among highly vaccinated LTCF populations even in years when the vaccine is well matched to circulating virus strains (2,3); LTCFs should conduct surveillance to identify clusters of respiratory illness and should alert state or local health departments when clusters are identified. Second, early detection of influenza outbreaks and timely initiation of control measures, such as cohorting of ill residents, use of droplet precautions, and use of antiviral medications (amantadine or rimantadine) for prophylaxis or treatment of persons at high risk for influenza A-related complications, can limit the spread of disease (1,4). Amantadine and rimantadine are 70%–90% effective in preventing influenza A infections and can reduce severity and duration of symptoms from influenza A when administered within 48 hours of onset; however, these medications are not effective against influenza type B viruses (5). Chronic-care facilities should know which laboratories in their area perform rapid influenza A testing and should develop a plan to rapidly detect influenza A outbreaks and to administer antiviral medications if influenza is detected (4–7). Third, health-care workers can act as a vehicle for introducing influenza illness into LTCFs (3,7).

*Influenza Activity — Continued*

Because influenza infections can be severe in debilitated populations and because vaccine effectiveness is lower among LTCF residents (30%–40%) than in healthy adults (70%–90%), the Advisory Committee on Immunization Practices recommends that health-care workers and others caring for high-risk persons receive influenza vaccine annually (2,3,5,7). Health-care workers and family members should be educated about the potentially serious consequences of influenza illness for high-risk persons and the need to limit contact with these persons. When health-care workers and family members are ill, they should avoid contact with high-risk persons.

Influenza surveillance data collected by CDC are updated weekly throughout the influenza season. Summaries are available through CDC; telephone (888) 232-3228, or fax (888) 232-3299 (request document number 361100). Surveillance information also is available on the World-Wide Web at <<http://www.cdc.gov/ncidod/diseases/flu/weekly.htm>>.

*References*

1. Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53–80.
2. Drinka PJ, Gravenstein S, Krause P, Schilling M, Miller BA, Shult P. Outbreaks of influenza A and B in a highly immunized nursing home population. *J Fam Prac* 1997;45:509–14.
3. Coles FB, Balzano GJ, Morse DL. An outbreak of influenza A (H3N2) in a well immunized nursing home population. *J Am Geriatr Soc* 1992;40:589–92.
4. Leonardi GP, Leib H, Birkhead GS, Smith C, Costello P, Conron W. Comparison of rapid detection methods for influenza A virus and their value in health-care management of institutionalized geriatric patients. *J Clin Microbiol* 1994;32:70–4.
5. Advisory Committee on Immunization Practices. Prevention and control of influenza: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 1998;47(no. RR-6).
6. Gomolin IH, Leib HB, Arden NH, Sherman FT. Control of influenza outbreaks in the nursing home: guidelines for diagnosis and management. *J Am Geriatr Soc* 1995;43:71–4.
7. Atkinson WL, Arden NH, Patriarca PA, Norman L, Lui KJ, Gohd R. Amantadine prophylaxis during an institutional outbreak of type A influenza. *Arch Intern Med* 1986;146:1751–6.

### **Decrease in Infant Mortality and Sudden Infant Death Syndrome Among Northwest American Indians and Alaskan Natives — Pacific Northwest, 1985–1996**

Although the infant mortality rate (IMR) has steadily declined in the United States since the early 1900s, the rate varies among racial/ethnic populations (1). A goal of the national health objectives for 2010 is to eliminate racial/ethnic health disparities (U.S. Department of Health and Human Services, unpublished data, 1999). Historically, IMRs among American Indians and Alaskan Natives (AI/AN) have been high (2). In addition, IMRs have varied among AI/AN populations (3). To determine recent trends in infant mortality among Northwest AI/AN, the Northwest Portland Area Indian Health Board (NPAIHB) analyzed annual IMRs among AI/AN in Idaho, Oregon, and Washington. In addition, because sudden infant death syndrome (SIDS) is the major contributor to excess infant mortality in Northwest AI/AN (4,5), NPAIHB analyzed SIDS rates to determine whether the decline in SIDS rates in the United States also was occurring among Northwest AI/AN. This report summarizes the results of this

*Infant Mortality — Continued*

analysis and documents dramatic decreases in both SIDS and non-SIDS infant mortality.

Annual vital statistics data for 1985–1996 were analyzed from the state health departments of Idaho, Oregon, and Washington and from CDC. Numerators for IMRs were all resident deaths for which the decedent was aged <365 days and for which the death certificate was linked to a birth certificate on which the race of the mother was AI/AN, regardless of whether the death occurred in the same calendar year as the birth. Denominators for IMRs were all resident live-born infants for each year for which the race of the mother on the birth certificate was AI/AN. Comparison rates for SIDS and overall infant mortality for all other races (non-AI/AN) were calculated by subtracting the AI/AN births and infant deaths annually for each state from the all-races totals obtained from CDC. Hispanic ethnicity was not considered in the analysis. Annual rate changes were compared with combined rates for 1985–1988, 1989–1992, and 1993–1996. These periods were selected for comparison because of the introduction in 1993 of several programmatic initiatives that might have influenced IMRs among Northwest AI/AN. Deaths attributed to SIDS were those for which the underlying cause of death was listed as *International Classifications of Diseases, Ninth Revision*, code 798.0. Statistical analysis was conducted using chi square tests for trends using EpiInfo (6).

From 1985 through 1996, IMRs and SIDS rates decreased among Northwest AI/AN (Table 1). In particular, IMRs for Northwest AI/AN decreased from 20.0 per 1000 live-born infants during 1985–1988 to 7.7 during 1993–1996, a rate difference of 12.3 per 1000 population. During the same period, SIDS mortality rates decreased from 8.9 to 3.0, a rate difference of 5.9. Approximately half (48.0%) of the decline in AI/AN IMRs was attributable to the decline in SIDS.

For the same three time periods, IMRs and SIDS rates also decreased for non-AI/AN in Idaho, Oregon, and Washington. For non-AI/AN, IMR declined from 9.6 during 1985–1988 to 6.3 during 1993–1996, a rate difference of 3.3, and the SIDS rate decreased from 2.5 to 1.4, a rate difference of 1.1. Approximately one third of the decrease in infant mortality in non-AI/AN resulted from the decline in SIDS.

Annual SIDS rates and overall IMRs decreased substantially for both AI/AN and non-AI/AN during the study period (Figure 1). IMRs for Northwest AI/AN is approaching that for non-AI/AN in the same states. The small increase in deaths attributed to SIDS in 1996 did not differ significantly from the trend.

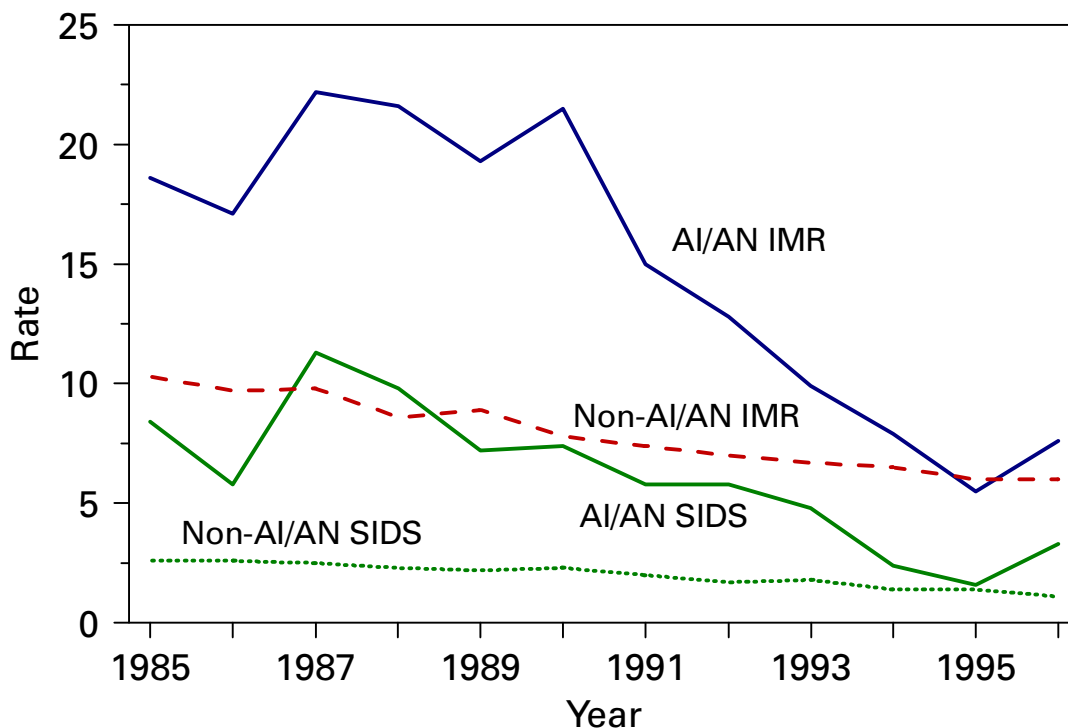
**TABLE 1. Number of sudden infant death syndrome (SIDS) and non-SIDS cases and SIDS rate among American Indians and Alaska Natives, by year—Idaho, Oregon, and Washington, 1985–1996**

Characteristic	1985– 1988	1989– 1993	1993– 1996
No. live-born infants	9,410	10,775	10,350
No. SIDS cases	84	71	31
No. Non-SIDS cases	104	115	49
SIDS rate*	8.9	6.6	3.0
Non-SIDS rate*	11.1	10.7	4.7

\*Per 1000 live-born infants.

## Infant Mortality — Continued

**FIGURE 1. Infant mortality rates (IMRs)\* and sudden infant death syndrome (SIDS) rates among American Indians and Alaskan Natives (AI/AN) and non-AI/AN — Idaho, Oregon, and Washington, 1985–1996**



\*Per 1000 live-born infants.

Reported by: LD Robertson, MD, Northwest Portland Area Indian Health Board, Portland, Oregon. LA DeRoo, MPH, JA Gaudino, MD, Washington State Dept of Health. CG Hahn, MD, Idaho Dept of Health and Welfare. KD Rosenberg, MD, Oregon Health Div.

**Editorial Note:** The findings in this report document a dramatic decline in IMR among Northwest AI/AN during 1985–1996. Decreases in both SIDS and non-SIDS cases were observed across each of the last two time periods, but decreases were greatest during 1993–1996. The decline in SIDS among Northwest AI/AN is consistent with, but of a greater magnitude than, the substantial decreases in SIDS nationally that have been attributed to the success of the national Back to Sleep campaign (7).

Multiple factors may have caused the decreases in SIDS and non-SIDS cases among Northwest AI/AN. Important risk factors that have been associated with SIDS include prone sleeping position and exposure to environmental tobacco smoke (ETS). In 1993, to reduce the risk for SIDS among Northwest AI/AN, the Portland Area Indian Health Service (IHS) (covering Idaho, Oregon, and Washington) initiated programs for parental education on nonprone infant sleep position and reduction of infant exposure to ETS. However, many Northwest AI/AN receive part or all of their health-care services outside the IHS health-care delivery system. As a result, the extent that Northwest AI/AN were exposed to these IHS programs is uncertain. As early as 1992, there was publicity in the Seattle area about increased risk for SIDS among infants sleeping prone, and in 1994 the national Back to Sleep program began. However, it is unknown

*Infant Mortality — Continued*

whether there were substantial changes in the prevalence of prone sleeping position or exposure to ETS among Northwest AI/AN during the time periods.

Factors that may have helped reduce non-SIDS IMRs among Northwest AI/AN include 1) structured activities by Portland area IHS programs initiated in 1993 to identify and manage high-risk pregnancies, 2) state programs such as the Washington State First Steps Medicaid expansion program for pregnant women and infants, 3) improved access to tertiary care for very low birth weight (<1500 g [ $<3$  lbs, 3 oz]) newborns, and 4) improvements in technology (e.g., introduction of surfactant use in neonatal intensive-care units).

The findings in this report are subject to at least four limitations. First, infant race was defined using the CDC's National Center for Health Statistics definition of race for infant mortality (i.e., for calculation of rates, the infant is assigned the mother's race), which differs from the IHS method (i.e., considering the race of the infant as AI/AN if either the mother or father is AI/AN); thus, these findings cannot be directly compared with published IHS data. Second, determining race for AI/AN from vital statistics data is problematic (8); however, using linked records as in this analysis can minimize this problem (9). Third, diagnostic shift could have occurred, resulting in infant deaths that formerly would have been attributed to SIDS being ascribed to other causes. However, this possibility has been examined recently in other populations (7) and was not found to be a substantial factor. Finally, a small number of infant death records could not be linked to a birth certificate and were excluded.

More extensive analysis is needed to determine factors associated with the dramatic decreases in IMRs and SIDS rates among Northwest AI/AN. Further understanding of the protective factors would be useful for developing and implementing programs to reduce infant mortality in other AI/AN populations in which high rates of SIDS and non-SIDS cases have been documented (10).

*References*

1. MacDorman MF, Atkinson JO. Infant mortality statistics from the 1996 period linked birth/infant death data set. *Mon Vital Stat Rep* 1998;46(12).
2. Indian Health Service. Trends in Indian health, 1996. Rockville, Maryland: US Department of Health and Human Services, 1997;40.
3. Indian Health Service. Regional differences in Indian health, 1997. Rockville, Maryland: US Department of Health and Human Services, 1998;50.
4. Spiers PS, Santos V, Steinschneider A, Robertson LD. Race and risk for SIDS: final report to the Maternal and Child Health and Crippled Children's Services Research Grants Program. Springfield, Virginia: US Department of Health and Human Services, Public Health Service, 1988.
5. Irwin KL, Mannino S, Daling J. Sudden infant death syndrome in Washington state: why are Native American infants at greater risk than white infants? *J Pediatr* 1992;121:242-7.
6. Dean AG, Dean JA, Coulombier D, et al. Epi Info, version 6: a word processing, database, and statistics program for epidemiology on microcomputers [Software documentation]. Atlanta, Georgia: US Department of Health and Human Services, CDC, 1994.
7. CDC. Sudden infant death syndrome—United States, 1983-1994. *MMWR* 1996;40:859-63.
8. Frost F, Tollestrup K, Ross A, Sabota E, Kimball E. Correctness of racial coding of American Indians and Alaska Natives on the Washington state death certificate. *Am J Prev Med* 1994; 10:290-4.
9. CDC. Classification of American Indian race on birth and infant death certificates—California and Montana. *MMWR* 1990;42:220-3.
10. Oyen N, Bulterys M, Welty TK, Kraus J. Sudden unexplained infant deaths among American Indians and whites in North and South Dakota. *Paediatr Perinat Epidemiol* 1990;4:175-83.



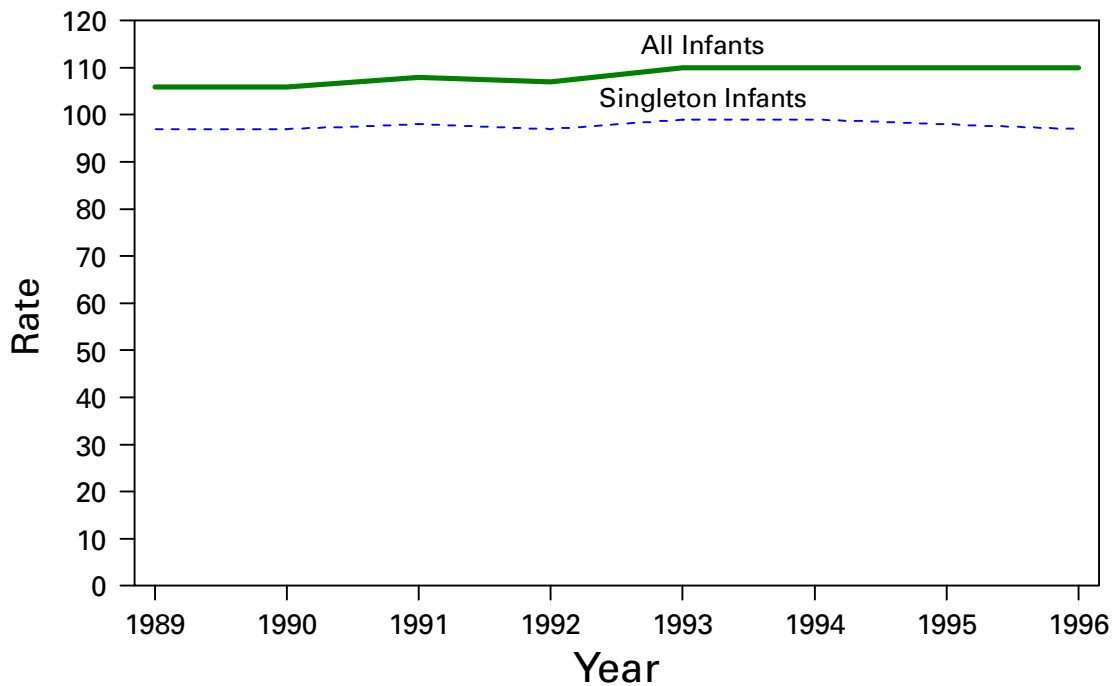
## Preterm Singleton Births — United States, 1989–1996

Preterm birth (birth at <37 completed weeks of gestation) is the second leading cause of neonatal mortality in the United States (1). Preterm birthrates differ by race; in 1996, black infants were 1.8 times more likely than white infants to be preterm (2). From 1989 through 1996, the overall rate of preterm birth (per 1000 live-born infants) increased 4% (2), and the rate of multiple births (e.g., twins, triplets, or other higher-order births) increased 19% (2). Multiple births are associated with preterm birth (3); trends in preterm births independent of the influence of multiple births have not been fully explored. To characterize race- and ethnicity-specific trends in preterm birth independent of multiple births, data from U.S. birth certificates for 1989–1996 were analyzed for singleton births only. This report summarizes the results of this analysis and indicates that although singleton preterm birthrates are stable overall, substantial changes in rates occurred in some racial/ethnic subgroups.

For this report, preterm birth was defined as a live birth occurring at 17–36 completed weeks of gestation and was subgrouped by weeks of gestation: moderately preterm (33–36 weeks), very preterm (29–32 weeks), extremely preterm (20–28 weeks), and ultra preterm (17–19 weeks). Gestational age was determined from information on the birth certificate by one of two methods (2,4): 1) the interval between the first day of the mother's last normal menstrual period (LMP) and the date of birth, or 2) a clinical estimate by the birth attendant of gestational age when the month or year of the LMP was missing or when the gestational age based on this date was inconsistent with the infant's birth weight. Approximately 1% of singleton infants were excluded because of missing or implausible estimates of gestational age. Infants were imputed as singletons for the 0.02% of live-born infants for which the number of fetuses in a given pregnancy was unreported. Maternal race and ethnicity were based on self-report and categorized as non-Hispanic white, non-Hispanic black, Hispanic, American Indian/Alaskan Native, or Asian/Pacific Islander. Stratification by gestational age was not performed for American Indians/Alaskan Natives and Asians/Pacific Islanders because the number of preterm births, when broken down into gestational age subgroups, was too small for meaningful analysis.

From 1989 through 1996, the preterm birthrate (per 1000 live-born infants) among singletons increased 0.3% (from 97.0 to 97.3) (Figure 1). Among moderately preterm singleton infants, the birthrate increased 2% (from 74.8 to 76.5). Among very preterm singleton infants, the birthrate decreased 8% (from 14.4 to 13.2) and among extremely preterm infants, decreased 4% (from 7.6 to 7.3) (Table 1). The singleton preterm birthrate increased 8% among non-Hispanic whites but decreased 10% among non-Hispanic blacks, 4% among Hispanics, 3% among American Indians/Alaskan Natives, and 2% among Asians/Pacific Islanders (Table 1). Among non-Hispanic whites, the moderately preterm birthrate increased 10%, and minor changes were observed in very and extremely preterm birthrates. Among non-Hispanic blacks and Hispanics, the preterm birthrate decreased in the moderately, very, and extremely preterm subgroups (Table 1).

Maternal factors that may affect observed trends in preterm birthrates were analyzed. The percentage of singleton infants born to women aged  $\geq 35$  years increased 43% (from 8.4% in 1989 to 12.0% in 1996), the percentage born to women who entered prenatal care during the first trimester increased 8% (from 75.6% to 81.8%), and the

*Preterm Singleton Births — Continued***FIGURE 1. Rate\* of preterm† birth among singleton infants — United States, 1989–1996**

\*Per 1000 live-born infants.

†<37 completed weeks of gestation.

percentage born to unmarried women increased 20% (from 27.0% to 32.5%). Similar trends were observed in all racial/ethnic groups.

To control for changes in maternal factors, preterm birthrates were directly standardized for each racial/ethnic group to the combined 1989 and 1996 singleton live birth distributions for maternal age, time of entry into prenatal care, and marital status. After standardization, the change from 1989 to 1996 in the preterm birthrate among non-Hispanic whites was 3.8 per 1000 live-born infants, 37% lower than the crude rate change of 6.0 (Table 2). For other racial/ethnic groups, the standardized rate was lower than the crude rate by 50% among non-Hispanic blacks, 29% among Hispanics, and 78% among American Indians/Alaskan Natives.

In addition to changes in maternal factors, changes in obstetric practices occurred during the study period that may have influenced preterm birthrates. For example, the percentage of singleton infants born to women whose labor was medically induced increased from 9.1% to 17.1%. To determine whether changes in preterm birthrates were independent of the change in induction practices, medically induced births were excluded from the analysis and rates were again standardized for maternal age, marital status, and time of entry into prenatal care. In this restricted group, the standardized preterm birthrate increased 9% among non-Hispanic whites, decreased 4% among non-Hispanic blacks, and changed <2% among Hispanics, American Indians/Alaskan Natives, and Asians/Pacific Islanders.

The proportion of births for which gestational age estimates were based on clinical evaluation increased slightly during the study period (from 3.6% in 1989 to

*Preterm Singleton Births — Continued***TABLE 1. Rate\* of preterm† birth among singleton infants, by maternal race/ethnicity‡, gestational age group, and year — United States, 1989 and 1996**

Race/Ethnicity/ Gestational age	1989	1996	% Change
<b>Non-Hispanic white</b>			
<20 weeks	0.1	0.1	7.7
20–28 weeks	4.8	4.9	2.1
29–32 weeks	9.9	9.9	0
33–36 weeks	60.0	65.9	9.8
<b>Total</b>	<b>74.8</b>	<b>80.8</b>	<b>8.0</b>
<b>Non-Hispanic black</b>			
<20 weeks	0.7	0.7	0
20–28 weeks	20.5	19.1	– 6.8
29–32 weeks	32.6	27.1	–16.9
33–36 weeks	126.6	115.6	– 8.7
<b>Total</b>	<b>180.4</b>	<b>162.5</b>	<b>– 9.9</b>
<b>Hispanic</b>			
<20 weeks	0.2	0.1	–23.5
20–28 weeks	6.5	6.4	– 1.5
29–32 weeks	14.5	13.4	– 7.6
33–36 weeks	83.3	80.8	– 3.0
<b>Total</b>	<b>104.5</b>	<b>100.7</b>	<b>– 3.6</b>
<b>American Indian/ Alaskan Native</b>	<b>112.9</b>	<b>109.7</b>	<b>– 2.8</b>
<b>Asian/Pacific Islander</b>	<b>94.8</b>	<b>92.6</b>	<b>– 2.3</b>
<b>All races</b>			
<20 weeks	0.2	0.2	– 4.2
20–28 weeks	7.6	7.3	– 3.9
29–32 weeks	14.4	13.2	– 8.3
33–36 weeks	74.8	76.5	2.3
<b>Total</b>	<b>97.0</b>	<b>97.3</b>	<b>0.3</b>

\*Per 1000 live-born infants, rounded to the nearest tenth.

†<37 completed weeks of gestation.

‡Stratification by gestational age was not performed for American Indians/Alaskan Natives and Asians/Pacific Islanders because the number of preterm births, when broken into gestational age subgroups, was too small for meaningful analysis.

4.7% in 1996). Because the method of determining gestational age may influence identification of a birth as preterm, an analysis was conducted that excluded births for which gestational age was clinically estimated. The standardized preterm birthrate for the study period increased 6.3% among non-Hispanic whites, decreased 5.0% among non-Hispanic blacks, and changed <2% among Hispanics, American Indians/Alaskan Natives, and Asians/Pacific Islanders.

*Reported by: Div of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion; Div of Applied Public Health Training, Epidemiology Program Office; Div of Vital Statistics, National Center for Health Statistics; and an EIS Officer, CDC.*

**Editorial Note:** The findings in this report indicate that preterm birthrates among singletons are stable; however, the overall rate masks differences in trends by race/ethnicity and among gestational age subgroups. The rate for singleton preterm births increased among non-Hispanic whites mainly because of an increase in the birthrate

*Preterm Singleton Births — Continued***TABLE 2. Crude and standardized rates\* of preterm† birth among singleton infants and change in rate, by maternal race/ethnicity — United States, 1989 and 1996**

Race/Ethnicity	Crude				Standardized <sup>§</sup>			
	Rate		Change 1989 to 1996		Rate		Change 1989 to 1996	
	1989	1996	Absolute	(%)	1989	1996	Absolute	(%)
Non-Hispanic white	74.8	80.8	6.0	( 8.0%)	81.4	85.2	3.8	( 4.6%)
Non-Hispanic black	180.4	162.5	17.9	(-9.9%)	154.6	145.6	9.0	(-5.8%)
Hispanic	104.5	100.7	3.8	(-3.6%)	99.8	97.1	2.7	(-2.8%)
American Indian/ Alaskan Native	112.9	109.7	3.2	(-2.8%)	101.3	102.0	0.7	( 0.7%)
Asian/Pacific Islander	94.8	92.6	2.2	(-2.3%)	102.5	99.3	3.2	(-3.1%)

\*Per 1000 live-born infants.

†&lt;37 completed weeks of gestation.

§Calculated by direct standardization using the combined 1989 and 1996 singleton live birth distributions for maternal age, entry into prenatal care, and marital status.

of moderately preterm infants. Among non-Hispanic blacks, the decline in moderately, very, and extremely preterm singleton births was substantial, and more modest declines were observed in overall preterm birthrates for Hispanics, American Indians/Alaskan Natives, and Asians/Pacific Islanders. The increase in singleton preterm birthrates among non-Hispanic whites and the decrease among non-Hispanic blacks are not explained entirely by changes in maternal age distribution, marital status, time of entry into prenatal care, induction rates, or use of clinical estimates of gestational age.

The findings in this study are subject to at least three limitations. First, LMP and clinical-based gestational age may be misclassified (e.g., because of imperfect maternal recall, postconception bleeding, delayed ovulation, or intervening early miscarriage); such errors may occur more frequently in some subpopulations, especially at shorter gestations (5). Second, changes in the reporting of preterm live births with the shortest gestations (ultra preterm) could have affected the preterm birthrates (6). However, these infants represent a small fraction of total preterm infants and do not contribute substantially to overall trends. Finally, because fetal deaths were not evaluated, the contribution of changes in fetal survival to the increase in preterm birthrates for non-Hispanic whites could not be assessed.

The disparity in preterm birthrates between blacks and whites is decreasing because of an increase in preterm births among non-Hispanic whites and a decrease among non-Hispanic blacks. The racial disparity in singleton preterm birth between non-Hispanic blacks and non-Hispanic whites decreased 17% from 1989 to 1996; however, in 1996, the risk for singleton preterm birth among blacks was still twice that for whites. Although many risk factors for preterm delivery have been identified, specific etiologies are not well characterized (7). In addition, many potential risk factors for preterm birth, such as urogenital tract infections (8) and history of subfertility or infertility (9) cannot be examined using the standard certificate of live birth. Additional studies exploring why preterm births are increasing among non-Hispanic whites and

*Preterm Singleton Births — Continued*

decreasing among non-Hispanic blacks may further understanding of how to prevent preterm birth.

*References*

1. Peters KD, Lochanek KD, Murphy SL. Deaths: final data for 1996. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, National Center for Health Statistics, 1998. National Vital Stat Rep 1998;47(9).
2. Ventura SJ, Martin JA, Curtin SC, Mathews TJ. Report of final natality statistics, 1996. Mon Vital Stat Rep 1998;46(11, suppl).
3. Keith LG, Cervantes A, Mazela J, Oleszczuk JJ, Papiernik E. Multiple births and preterm delivery. Prenat Neonat Med 1998;3:125–9.
4. National Center for Health Statistics. Instruction manual part 12, Computer edits for natality data, 1989. Hyattsville, Maryland: US Department of Health and Human Services, CDC, 1991.
5. Alexander GR, Himes JH, Kaufman RB, Mor J, Kogan M. A United States national reference for fetal growth. Obstet Gynecol 1996;87:163–8.
6. Phelan ST, Goldenberg R, Alexander G, Cliver SP. Perinatal mortality and its relationship to the reporting of low-birthweight infants. Am J Public Health 1998;88:1236–9.
7. Berkowitz GS, Papiernik E. Epidemiology of preterm birth. Epidemiol Rev 1993;15:414–43.
8. Fiscella K. Racial disparities in preterm births. The role of urogenital infections. Public Health Rep 1996;111:104–13.
9. Henriksen TB, Baird DD, Olsen J, Hedegaard M, Secher NJ, Wilcox AJ. Time to pregnancy and preterm delivery. Obstet Gynecol 1997;89:594–9.

### **Incidence of Foodborne Illnesses: Preliminary Data from the Foodborne Diseases Active Surveillance Network (FoodNet) — United States, 1998**

Estimates of the magnitude of foodborne illness in the United States have been imprecise. To quantify, better understand, and more precisely monitor foodborne illness, since 1996 the Foodborne Diseases Active Surveillance Network (FoodNet) has collected data to monitor nine foodborne diseases in selected U.S. sites (1). This report describes preliminary data from FoodNet surveillance for 1998 and compares findings with those for 1996 and 1997; compared with 1996, the overall incidence of the foodborne illnesses under surveillance during 1998 declined, particularly for salmonellosis and campylobacteriosis, and the data continued to demonstrate regional and seasonal differences in the reported incidence of diseases.

In 1996, active surveillance was initiated for culture-confirmed cases of *Campylobacter*, Shiga toxin-producing *Escherichia coli* O157, *Listeria*, *Salmonella*, *Shigella*, *Vibrio*, and *Yersinia* infections in Minnesota and Oregon and in selected counties in California, Connecticut, and Georgia. In 1997, surveillance for laboratory-confirmed cases of *Cryptosporidium* and *Cyclospora* infections were added. In 1998, active surveillance for these nine pathogens was initiated in selected counties in Maryland and New York. To identify cases, surveillance personnel contacted each clinical laboratory in their catchment areas either weekly or monthly, depending on the size of the clinical laboratory. Preliminary annual incidence was calculated using the number of cases reported by those laboratories for 1998 as the numerator and 1997 population estimates as the denominator (2); final incidence will be available once 1998 population estimates are available in mid-1999. All the rates contained in this article are

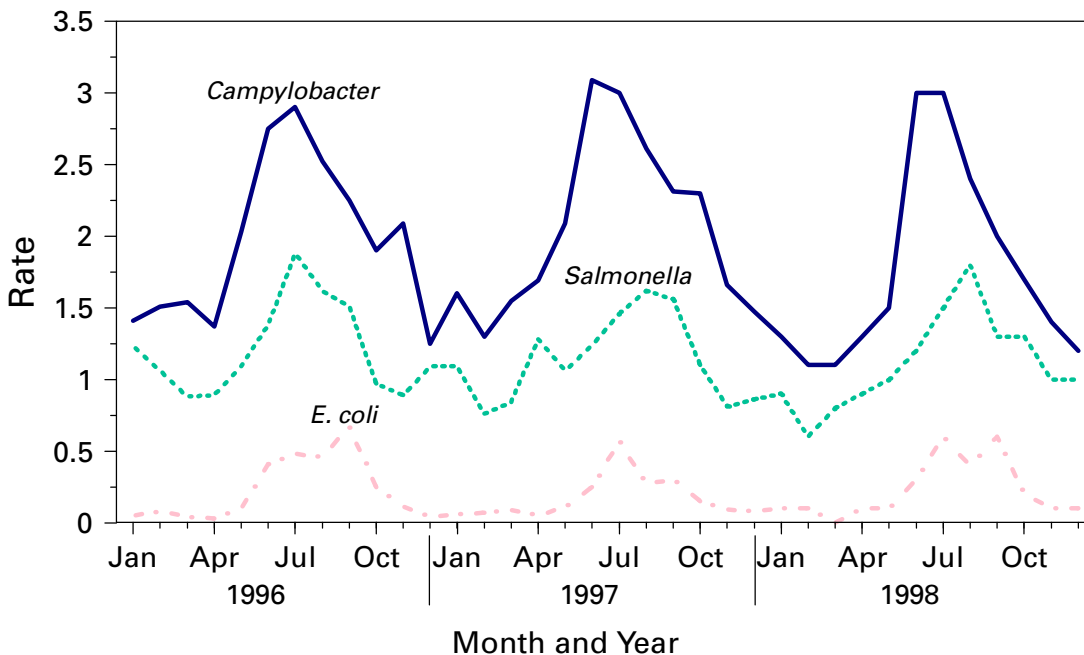
*FoodNet — Continued*

considered preliminary. Monthly incidence was calculated based on date of specimen collection.

**1998 Surveillance**

During 1998, 9787 laboratory-confirmed cases of nine diseases under surveillance were identified: 4031 of campylobacteriosis, 2849 of salmonellosis, 1483 of shigellosis, 565 of cryptosporidiosis, 508 of *E. coli* O157 infections, 186 of yersiniosis, 106 of listeriosis, 50 of *Vibrio* infections, and nine of cyclosporiasis. Among the 2670 *Salmonella* isolates serotyped, 808 (30%) were serotype Typhimurium, 406 (15%) were serotype Enteritidis (SE), and 168 (6%) were serotype Heidelberg; 179 (6%) were untyped. Isolation rates varied by season for several pathogens: 46% of *E. coli* O157, 41% of *Campylobacter*, and 35% of *Salmonella* were isolated during June–August (Figure 1). Fifty percent of cyclosporiasis cases and 33% of cryptosporidiosis cases were identified during June–August. Yersiniosis was more likely to occur during winter months, with 41% of cases reported in January, February, or December. *Listeria*, not usually tested for in stool, was isolated from normally sterile sites, including blood and cerebrospinal fluid, in 93% of reported listeriosis cases. In 8% of yersiniosis cases, 7% of salmonellosis cases, and  $\leq 1\%$  of shigellosis and campylobacteriosis cases, the organism was isolated from normally sterile sites.

**FIGURE 1. Rate\* of laboratory-confirmed infections with selected pathogens detected by the Foodborne Diseases Active Surveillance Network (FoodNet)<sup>†</sup> — United States, 1996–1998**



\*Per 100,000 population.

<sup>†</sup>In 1996, active surveillance was initiated for culture-confirmed cases of *Campylobacter*, *Salmonella*, *Shigella*, and Shiga toxin-producing *Escherichia coli* O157 infections in Minnesota and Oregon and selected counties in California, Connecticut, and Georgia. Data presented in this figure are from the original FoodNet sites only.

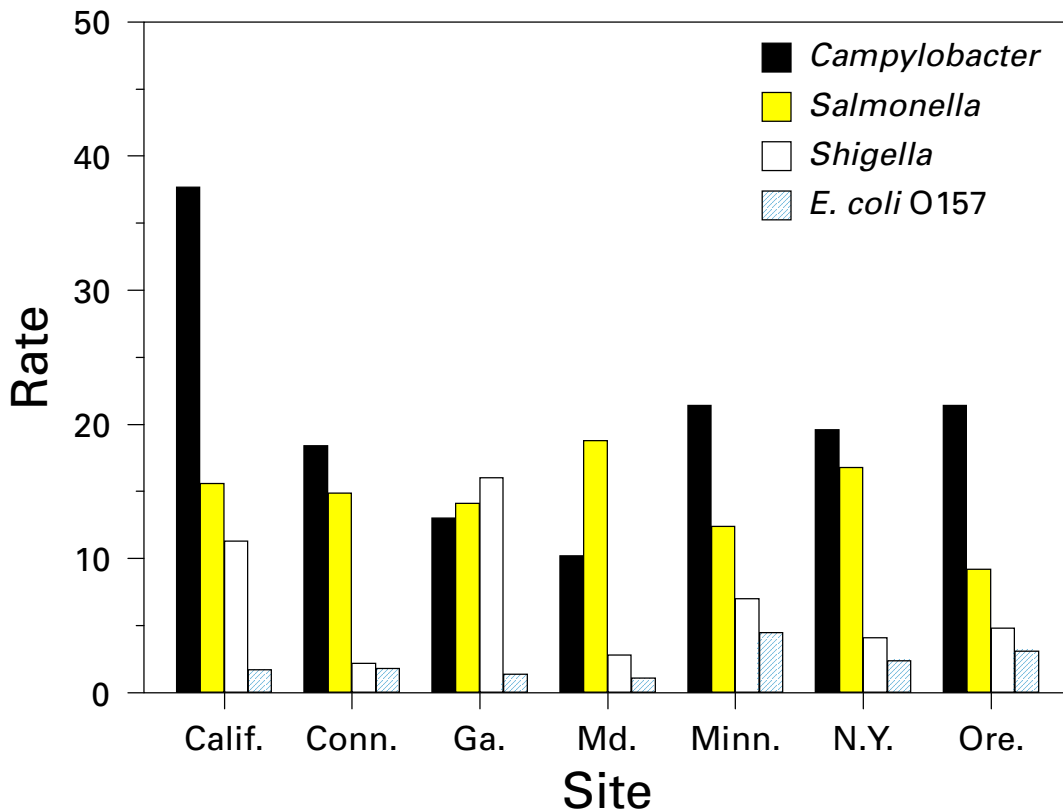
*FoodNet — Continued*

For all reporting sites, incidence was highest for campylobacteriosis (19.7 per 100,000 population), salmonellosis (13.9) and shigellosis (7.2). Substantial variation in incidence was observed among the sites for some pathogens (Figure 2). The incidence of campylobacteriosis ranged from 10.2 in Maryland to 37.7 in California. Although overall salmonellosis incidence was similar among the sites, the rates for infections with specific *Salmonella* serotypes varied; rates of infection with SE ranged from 0.7 in Georgia and New York to 5.1 in Maryland. Rates of infection with Typhimurium ranged from 3.1 in California and New York to 5.2 in Maryland. Shigellosis incidence ranged from 2.2 in Connecticut to 16.0 in Georgia. Incidence of *E. coli* O157 infections ranged from 1.1 in Maryland to 4.5 in Minnesota, and for yersiniosis ranged from 0.4 in New York to 1.6 in California and Georgia. The incidence of cryptosporidiosis ranged from 0.6 in Maryland to 3.7 in Minnesota.

**Comparison of Preliminary 1998 Data with 1996 and 1997 Data**

Comparing data from the five original FoodNet sites, overall incidence of laboratory-confirmed infections caused by the pathogens under surveillance declined from 1996 to 1998 (Table 1). Over this 3-year period, the largest decrease in bacterial pathogen-specific rates occurred in cases of infection caused by *Salmonella* (14.5 in

**FIGURE 2. Rate\* of laboratory-confirmed infections detected by the Foodborne Diseases Active Surveillance Network (FoodNet)<sup>†</sup>, by site — United States, 1998**



\*Per 100,000 population.

<sup>†</sup>Reporting was statewide in Minnesota and Oregon and from selected counties in California, Connecticut, Georgia, Maryland, and New York.

FoodNet — Continued

**TABLE 1. Rate\* of selected pathogens detected by the Foodborne Diseases Active Surveillance Network (FoodNet)<sup>†</sup>, at the five original sites, by year — United States, 1996–1998**

Organism	1996	1997	1998
<i>Campylobacter</i>	23.5	25.2	21.7
<i>Cryptosporidium</i>	§	2.7	2.5
<i>Cyclospora</i>	§	0.3	0
<i>Escherichia coli</i> O157	2.7	2.3	2.8
<i>Listeria</i>	0.5	0.5	0.5
<i>Salmonella</i>	14.5	13.6	12.4
<i>Shigella</i>	8.9	7.5	8.5
<i>Vibrio</i>	0.1	0.3	0.3
<i>Yersinia</i>	1.0	0.9	1.0
<b>Total</b>	<b>51.2</b>	<b>50.3<sup>¶</sup></b>	<b>47.2<sup>¶</sup></b>

\*Per 100,000 population.

<sup>†</sup>In 1996, active surveillance was initiated for laboratory-confirmed cases of *Campylobacter*, Shiga toxin-producing *E. coli* O157, *Listeria*, *Salmonella*, *Shigella*, *Vibrio*, and *Yersinia* infections in Minnesota and Oregon and in selected counties in California, Connecticut, and Georgia. In 1997, surveillance for laboratory-confirmed cases of *Cryptosporidium* and *Cyclospora* infections was initiated in Minnesota and Oregon and in selected counties in California and Connecticut. Data presented in this table are from these original FoodNet sites only.

<sup>§</sup>Not reported.

<sup>¶</sup>Excludes *Cryptosporidium* and *Cyclospora*.

1996 to 12.4 in 1998, a 14% decline). This decrease was particularly pronounced for SE, which decreased 44% (from 2.5 to 1.4). Campylobacteriosis rates increased 7% from 1996 to 1997 and then decreased 14% (from 25.2 to 21.7) from 1997 to 1998. After declining 15% from 1996 to 1997, rates of *E. coli* O157 infection increased 22% from 1997 to 1998 (from 2.3 to 2.8). Similarly, the incidence of shigellosis decreased 16% from 1996 to 1997, but increased 13% from 1997 to 1998 (from 7.5 to 8.5). The incidence of infections caused by *Vibrio*, which increased from 1996 to 1997, remained elevated in 1998. The incidence of listeriosis and yersiniosis remained essentially unchanged during the 3-year period. Comparing the data on parasitic diseases from 1997 to 1998 (using only the sites reporting in both years), decreases occurred in the incidence of illness caused by *Cryptosporidium*, which decreased 7% (from 2.7 to 2.5), and by *Cyclospora*, which decreased from 0.3 to 0. In 1998 compared with 1997, Georgia reported a slight overall increase in the combined incidence of illnesses caused by the seven bacterial pathogens under surveillance; California, Connecticut, Minnesota, and Oregon reported decreases.

Reported by: S Shallow, MPH, M Samuel, DrPH, A McNees, MPH, G Rothrock, MPH, California Emerging Infections Program, D Vugia, MD, S Waterman, MD, State Epidemiologist, California Dept of Health Svcs. T Fiorentino, MPH, R Marcus, MPH, G Kazi, MPH, School of Medicine, Yale Univ, New Haven; P Mshar, M Cartter, MD, J Hadler, MD, State Epidemiologist, Connecticut State Dept of Public Health. M Farley, MD, M Bardsley, MPH, S Segler, MPH, Emory Univ School of Medicine; J Koehler, DVM, P Blake, MD, K Toomey, MD, State Epidemiologist, Div of Public Health, Georgia Dept of Human Resources. M Pass, Johns Hopkins Univ School of Hygiene and Public Health, Baltimore; Y Wong Univ of Maryland Hospital, Baltimore; K Henning, L Gay, M Carter, D Dwyer, MD, State Epidemiologist, Maryland Dept of Health and Mental Hygiene. J Wicklund, MPH, C Hedberg, PhD, M Osterholm, PhD, State Epidemiologist, Minnesota Dept of Public Health. S Zanski, D Morse, MD, New York State Dept of Health. M Cassidy, T McGivern, R Stanton, B Shiferaw, MD, P Cieslak, MD, D Fleming, MD, State Epidemiologist, Oregon Health Div. Office of Public Health and Science, Food Safety and Inspection Svc, US Dept of Agriculture.



*FoodNet — Continued*

Center for Food Safety and Applied Nutrition, Food and Drug Administration. Foodborne and Diarrheal Diseases Br, Div of Bacterial and Mycotic Diseases; Epidemiology Br, Div of Parasitic Diseases; and Office of the Director, National Center for Infectious Diseases, CDC.

**Editorial Note:** Each year, millions of persons experience foodborne illness, though only a fraction seek medical care and an even smaller number submit laboratory specimens. FoodNet provides a precise measure of the laboratory-diagnosed cases of specific foodborne illnesses and performs additional surveys and studies to interpret trends over time. The 1998 FoodNet data indicate a decline in several of the major bacterial and parasitic causes of foodborne illness. These declines might in part reflect annual fluctuations in the incidence of foodborne illnesses and temporal variations in diagnostic practices. The trends also may reflect implementation of disease prevention efforts. The declines in salmonellosis and campylobacteriosis may reflect changes in meat and poultry processing plants in the United States mandated by the Pathogen Reduction and Hazard Analysis and Critical Control Points (HACCP) rule of the U.S. Department of Agriculture (USDA). HACCP consists of production process controls, standard sanitation procedures, and microbial testing (by both food-processing plants and USDA) designed to reduce foodborne illnesses by monitoring and decreasing microbial contamination in food processing plants. HACCP was implemented by the largest producers in the food industry in January 1998. The decline from 1996 to 1998 in the incidence of salmonellosis parallels the reported decline in the percentage of meat and poultry products tested at large, federally inspected processing plants that were positive for *Salmonella* (3).

Reasons for the decline in SE isolates remain under investigation. SE commonly has been associated with eating undercooked eggs (4), particularly in outbreaks. Implementation of an egg quality-assurance program with microbiologic testing and egg diversion (5) in some states may have contributed to the decline in reported cases of human illness caused by SE. This decline also might in part be explained by the decrease in the percentage of poultry products testing positive for *Salmonella* in large processing plants; recent evidence suggests that poultry meat might be a source of sporadic SE infections (6).

Other changes in rates of foodborne illness may be explained by known events. For example, the large reduction in cyclosporiasis follows restrictions on the import of raspberries into the United States after a large outbreak was traced to this food (7). The continued elevation in reported rates of *Vibrio* infections reflects several multi-state outbreaks of *V. parahaemolyticus* in 1997 and 1998 (8,9). However, the reasons for the changes in the incidence *E. coli* O157 infections from 1996 to 1998 are unclear. Additional surveillance data collected through FoodNet will help evaluate temporal trends in foodborne illnesses.

In 1998, the FoodNet catchment area included 20.5 million persons (based on 1997 estimates), 7.7% of the U.S. population. In 1999, the catchment area will include approximately 30 million persons (1997 estimates), with Georgia initiating statewide surveillance and New York adding counties to its catchment area. Tennessee, the eighth FoodNet site, also will begin collecting data from selected counties in 1999. The 1998 final FoodNet report will include final incidence figures and other information such as illness severity. Because the sites are likely to have had increases in population since 1997 (the increase from 1996 to 1997 was 1%), the 1998 rates most likely will

*FoodNet — Continued*

be slightly lower than the preliminary rates. FoodNet reports are available on the World-Wide Web at <<http://www.cdc.gov/ncidod/dbmd/foodnet/foodnet.htm>>.

*References*

1. CDC. The Foodborne Diseases Active Surveillance Network, 1996. *MMWR* 1997;46:258–61.
2. Bureau of the Census, Economics and Statistics Administration, US Department of Commerce. Population estimates. Available at <<http://www.census.gov/population/www/estimates/popest.html>>. Accessed October 1998.
3. Food Safety and Inspection Service. HACCP implementation: first year *Salmonella* test results—January 26, 1998 to January 25, 1999. Washington, DC: United States Department of Agriculture, Food Safety and Inspection Service, Office of Public Health and Science Publications, 1999. Available at <<http://www.fsis.usda.gov/OPHS/salmdata.htm>>.
4. St. Louis ME, Morse DL, Potter ME, et al. The emergence of grade A eggs as a major source of *Salmonella* enteritidis infection—new implications for control of salmonellosis. *JAMA* 1988; 259:2103–7.
5. Schlosser WD, Henzler DJ, Mason J, et al. The *Salmonella enterica* serovar Enteritidis pilot project. In: Saeed AM, ed. *Salmonella enterica* serovar Enteritidis in humans and animals: epidemiology, pathogenesis, and control. Ames, Iowa: Iowa State University Press, 1999: 353–65.
6. Kimura A, Reddy S, Marcus R, et al. Chicken, a newly identified risk factor for sporadic *Salmonella* serotype Enteritidis infections in the United States: a case-control study in FoodNet sites [Abstract]. In: Program and abstracts of the Infectious Diseases Society of America 36th Annual Meeting, Denver, Colorado, November 12–15, 1998:178.
7. CDC. Update: outbreaks of cyclosporiasis—United States, 1997. *MMWR* 1997;46:461–2.
8. CDC. Outbreak of *Vibrio parahaemolyticus* infections associated with eating raw oysters—Pacific Northwest, 1997. *MMWR* 1998;47:457–62.
9. CDC. Outbreak of *Vibrio parahaemolyticus* infection associated with eating raw oysters and clams harvested from Long Island Sound—Connecticut, New Jersey, and New York, 1998. *MMWR* 1999;48:48–51.

*Notice to Readers***HIV Postexposure Prophylaxis Registry Closing**

Effective December 31, 1998, enrollment of new health-care workers (HCWs) in the Human Immunodeficiency Virus Postexposure Prophylaxis (HIV PEP) Registry ceased; the goals and objectives of the registry had been met. In addition, continuation of the registry appeared redundant with other ongoing surveillance programs.

The HIV PEP Registry was established in October 1996 as a prospective surveillance project to monitor adverse events associated with HIV PEP in HCWs after occupational HIV exposures. It was a collaborative project managed by CDC and two pharmaceutical companies, Glaxo Wellcome Inc. and Merck & Co., Inc.\* A designated third party, a contract research organization, responsible for registration and follow-up, served as the data coordination center.

The registry data have shown that HCWs for whom HIV PEP is prescribed have not reported unusual adverse events (i.e., those not included in the prescribing information or literature) with these treatments. Data suggest that careful counseling about

---

\* Use of trade names and commercial sources does not imply endorsement by the U.S. Department of Health and Human Services or CDC.

*Notice to Readers — Continued*

drug toxicity may be necessary to improve compliance with PEP among exposed HCWs. Six-week follow-up of enrolled HCWs will be completed.

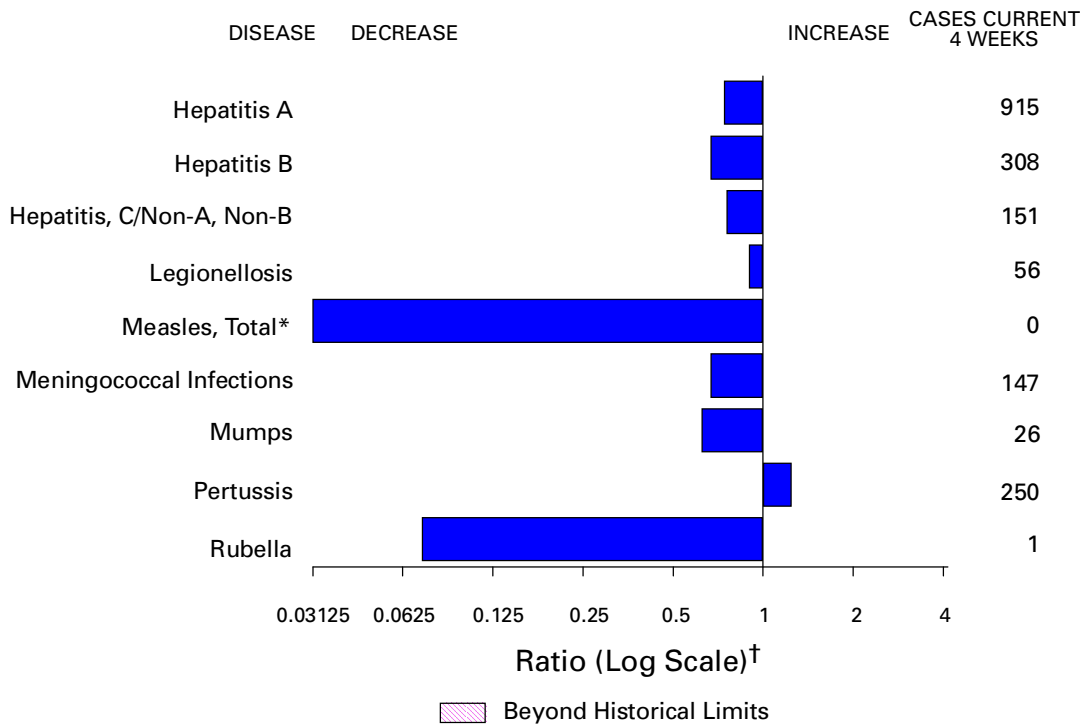
Additional information about the registry is available from the HIV PEP Registry, telephone (toll-free) (888) 737-4448 until June 30, 1999, and afterwards from CDC's Hospital Infections Program, telephone (404) 639-6425. Serious adverse events or product problems can be reported to the Food and Drug Administration's MedWatch program, telephone (800) 332-1088; fax (800) 332-0178; address: HF-2, FDA, 5600 Fishers Lane, Rockville, MD 20852-9787; or by the World-Wide Web, <<http://www.fda.gov/medwatch>>.

**Addenda: Vol. 48, No. 6**

In the report, "Farm Worker Illness Following Exposure to Carbofuran and Other Pesticides—Fresno County, California, 1998," on page 113 the first footnote should have indicated that resources for one of the surveillance programs also were provided by the U.S. Environmental Protection Agency. The footnote should read: "The California Department of Health Services (CDHS) participates in two pesticide illness prevention projects, for which CDC provided resources, that use case reports generated by these mandatory reporting requirements: the Sentinel Event Notification System for Occupational Risk (SENSOR) and Community Partners for Health Farming. The Office of Pesticide Programs, U.S. Environmental Protection Agency, also provided resources for the SENSOR program for pesticide-related illness in California."



**FIGURE I. Selected notifiable disease reports, comparison of provisional 4-week totals ending March 6, 1999, with historical data — United States**



\*No measles cases were reported for the current 4-week period, yielding a ratio for week 9 of zero (0).

† 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 — provisional cases of selected notifiable diseases, United States, cumulative, week ending March 6, 1999 (9th Week)**

	Cum. 1999		Cum. 1999
Anthrax	-	Plague	-
Brucellosis	8	Poliomyelitis, paralytic	-
Cholera	-	Psittacosis	4
Congenital rubella syndrome	-	Rabies, human	-
Cryptosporidiosis*	172	Rocky Mountain spotted fever (RMSF)	24
Diphtheria	-	Streptococcal disease, invasive Group A	241
Encephalitis: California*	1	Streptococcal toxic-shock syndrome*	5
eastern equine*	-	Syphilis, congenital <sup>¶</sup>	-
St. Louis*	-	Tetanus	2
western equine*	-	Toxic-shock syndrome	15
Hansen Disease	9	Trichinosis	1
Hantavirus pulmonary syndrome* <sup>†</sup>	1	Typhoid fever	32
Hemolytic uremic syndrome, post-diarrheal*	5	Yellow fever	-
HIV infection, pediatric* <sup>‡</sup>	7		

-:no reported cases

\*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 from reports to the Division of HIV/AIDS Prevention—Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP), last update January 24, 1999.

¶ Updated from reports to the Division of STD Prevention, NCHSTP.

**TABLE II. Provisional cases of selected notifiable diseases, United States, weeks ending March 6, 1999, and March 7, 1998 (9th Week)**

Reporting Area	AIDS		Chlamydia		<i>Escherichia coli</i> O157:H7		Gonorrhea		Hepatitis C/NA,NB	
	Cum. 1999*	Cum. 1998	Cum. 1999	Cum. 1998	NETSS†	PHLIS‡	Cum. 1999	Cum. 1998	Cum. 1999	Cum. 1998
					Cum. 1999	Cum. 1999				
UNITED STATES	3,137	7,332	78,510	98,646	174	72	45,259	58,752	396	634
NEW ENGLAND	158	198	2,614	3,770	26	23	819	1,087	40	19
Maine	3	4	124	177	1	-	9	8	-	-
N.H.	3	10	160	165	1	1	15	19	-	-
Vt.	-	8	85	55	1	-	10	1	1	2
Mass.	124	70	1,530	1,518	17	13	519	385	39	17
R.I.	9	22	375	430	-	-	98	59	-	-
Conn.	19	84	340	1,425	6	9	168	615	-	-
MID. ATLANTIC	489	2,103	12,199	14,320	11	1	6,559	8,255	24	53
Upstate N.Y.	17	299	N	N	9	-	453	1,025	20	49
N.Y. City	237	1,154	6,540	6,215	-	1	3,216	2,905	-	-
N.J.	162	284	1,041	2,018	2	-	672	1,260	-	-
Pa.	73	366	4,618	6,087	N	-	2,218	3,065	4	4
E.N. CENTRAL	179	509	12,728	14,902	33	8	8,855	11,345	92	87
Ohio	38	94	4,098	5,051	21	3	2,461	3,037	-	4
Ind.	25	79	-	-	5	-	726	1,135	-	2
Ill.	77	247	4,796	3,700	2	-	3,008	3,202	1	11
Mich.	22	57	3,345	3,713	5	2	2,436	3,018	91	70
Wis.	17	32	489	2,438	N	3	224	953	-	-
W.N. CENTRAL	110	147	2,879	6,175	30	12	1,078	2,550	2	74
Minn.	20	22	927	1,231	14	10	374	417	-	-
Iowa	3	9	325	650	5	2	141	182	-	2
Mo.	72	77	-	2,144	1	-	-	1,164	2	72
N. Dak.	-	3	-	168	2	-	-	16	-	-
S. Dak.	-	5	311	305	-	-	29	51	-	-
Nebr.	6	14	603	534	2	-	268	198	-	-
Kans.	9	17	713	1,143	6	-	266	522	-	-
S. ATLANTIC	883	1,855	19,166	18,889	22	7	15,108	15,117	34	18
Del.	13	36	524	400	1	-	302	265	-	-
Md.	81	239	1,260	1,311	2	-	1,871	1,397	16	2
D.C.	8	189	N	N	-	-	484	596	-	-
Va.	54	112	2,131	2,144	5	2	1,810	1,298	6	1
W. Va.	10	19	373	901	-	1	81	288	2	-
N.C.	69	107	3,949	3,606	3	2	3,547	3,140	-	5
S.C.	60	126	4,269	2,983	1	1	2,230	2,037	1	-
Ga.	111	228	2,408	4,283	1	-	1,682	3,440	1	6
Fla.	477	799	4,252	3,261	9	1	3,101	2,656	8	4
E.S. CENTRAL	157	289	5,965	6,787	13	1	5,332	6,633	24	17
Ky.	15	39	-	1,062	5	-	-	673	1	4
Tenn.	64	104	2,315	2,314	6	-	1,912	2,017	22	11
Ala.	31	86	2,504	1,689	2	-	2,269	2,235	1	2
Miss.	47	60	1,146	1,722	-	1	1,151	1,708	-	-
W.S. CENTRAL	532	885	5,203	13,572	5	1	3,741	8,603	18	12
Ark.	19	33	783	618	2	-	352	1,008	2	2
La.	27	148	2,926	2,133	1	1	2,599	1,872	7	-
Okla.	6	52	1,494	1,379	1	-	790	769	-	-
Tex.	480	652	-	9,442	1	-	-	4,954	9	10
MOUNTAIN	45	199	4,333	4,825	12	2	1,156	1,346	38	92
Mont.	-	8	208	158	-	-	3	8	4	4
Idaho	4	5	275	340	-	-	19	30	4	31
Wyo.	-	-	100	143	1	-	4	9	12	22
Colo.	26	39	1,161	1,181	3	1	320	469	4	7
N. Mex.	4	36	831	735	1	-	153	140	4	13
Ariz.	4	61	1,020	1,696	4	1	415	557	9	-
Utah	4	26	262	266	3	-	28	31	1	8
Nev.	3	24	476	306	-	-	214	102	-	7
PACIFIC	584	1,147	13,423	15,406	22	17	2,611	3,816	124	262
Wash.	29	73	2,123	1,870	1	4	366	327	2	2
Oreg.	15	31	732	1,024	8	8	97	152	-	1
Calif.	525	1,028	10,107	11,805	13	5	2,067	3,205	122	225
Alaska	5	-	264	341	-	-	48	55	-	-
Hawaii	10	15	197	366	-	-	33	77	-	34
Guam	1	-	-	47	N	-	-	4	-	-
P.R.	92	271	U	U	1	U	54	79	U	U
V.I.	-	8	N	N	N	U	U	U	U	U
Amer. Samoa	-	-	U	U	N	U	U	U	U	U
C.N.M.I.	-	-	N	N	N	U	-	7	-	-

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

\*Updated monthly from reports to the Division of HIV/AIDS Prevention—Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention, last update January 24, 1999.

†National Electronic Telecommunications System for Surveillance.

‡Public Health Laboratory Information System.

**TABLE II. (Cont'd.) Provisional cases of selected notifiable diseases, United States, weeks ending March 6, 1999, and March 7, 1998 (9th Week)**

Reporting Area	Legionellosis		Lyme Disease		Malaria		Syphilis (Primary & Secondary)		Tuberculosis		Rabies, Animal
	Cum. 1999	Cum. 1998	Cum. 1999	Cum. 1998	Cum. 1999	Cum. 1998	Cum. 1999	Cum. 1998	Cum. 1999*	Cum. 1998*	Cum. 1999
UNITED STATES	129	204	528	575	161	194	925	1,239	628	1,117	680
NEW ENGLAND	11	15	77	83	3	6	14	13	41	50	109
Maine	2	-	-	1	-	-	-	-	1	-	19
N.H.	1	2	-	1	-	-	-	1	-	-	4
Vt.	3	-	-	1	-	-	1	-	-	1	18
Mass.	2	5	65	22	3	6	9	11	12	23	31
R.I.	1	3	-	9	-	-	1	-	15	8	10
Conn.	2	5	12	49	-	-	3	1	13	18	27
MID. ATLANTIC	33	41	311	374	43	73	38	72	217	241	166
Upstate N.Y.	8	10	89	144	12	19	4	4	9	30	108
N.Y. City	-	10	1	10	10	40	14	7	132	152	U
N.J.	5	1	85	42	14	7	1	15	76	59	37
Pa.	20	20	136	178	7	7	19	46	U	U	21
E.N. CENTRAL	31	72	17	15	12	18	197	184	29	17	1
Ohio	14	20	11	10	2	1	17	36	U	U	-
Ind.	5	11	5	4	4	1	32	30	U	U	-
Ill.	-	14	-	-	-	-	9	122	65	U	U
Mich.	12	13	1	1	5	6	26	38	25	25	1
Wis.	-	14	U	U	1	1	-	15	4	17	-
W.N. CENTRAL	1	12	5	5	5	7	5	31	56	51	61
Minn.	-	-	1	-	-	1	-	1	32	18	17
Iowa	1	-	1	5	2	1	1	-	-	-	16
Mo.	-	6	-	-	3	4	-	20	19	28	-
N. Dak.	-	-	1	-	-	-	-	-	-	-	15
S. Dak.	-	-	-	-	-	-	-	-	2	-	-
Nebr.	-	6	-	-	-	-	1	4	1	-	1
Kans.	-	-	2	-	-	1	3	6	2	5	12
S. ATLANTIC	21	28	72	68	48	41	367	459	109	247	260
Del.	2	4	-	-	-	1	1	5	-	3	-
Md.	1	7	58	63	17	18	78	133	U	U	58
D.C.	-	2	1	3	5	2	10	14	8	19	-
Va.	2	3	-	-	7	4	27	39	9	30	61
W. Va.	N	N	-	-	1	-	1	-	7	12	15
N.C.	4	3	11	-	3	4	113	128	45	128	62
S.C.	4	3	-	-	-	-	43	47	40	55	11
Ga.	-	-	-	2	4	9	46	37	U	U	28
Fla.	8	6	2	-	11	3	48	56	U	U	25
E.S. CENTRAL	6	8	8	9	3	5	159	225	50	92	40
Ky.	2	4	-	-	-	-	-	24	U	U	13
Tenn.	4	2	3	5	2	3	89	114	U	U	18
Ala.	-	1	5	4	1	1	58	48	44	61	9
Miss.	-	1	-	-	-	1	12	39	6	31	-
W.S. CENTRAL	1	1	-	-	5	3	106	155	26	318	5
Ark.	-	-	-	-	-	-	19	17	14	5	-
La.	1	-	-	-	3	2	41	66	U	U	-
Okla.	-	-	-	-	1	-	46	9	12	20	5
Tex.	-	1	-	-	1	1	-	63	-	293	-
MOUNTAIN	11	11	1	1	8	10	14	46	21	45	18
Mont.	-	1	-	-	1	-	-	-	-	2	8
Idaho	-	-	-	-	1	1	-	-	-	-	-
Wyo.	-	-	-	-	-	-	-	-	-	1	5
Colo.	1	4	-	-	3	3	-	3	U	U	1
N. Mex.	1	1	1	-	1	3	-	4	6	8	-
Ariz.	1	-	-	-	2	2	13	34	U	U	4
Utah	4	4	-	-	-	1	-	2	9	6	-
Nev.	4	1	-	1	-	-	1	3	6	28	-
PACIFIC	14	16	37	20	34	31	25	54	79	56	20
Wash.	2	-	-	-	2	-	5	4	45	32	-
Oreg.	-	-	1	-	4	5	-	1	U	U	-
Calif.	12	16	36	20	27	26	19	49	U	U	18
Alaska	-	-	-	-	-	-	-	-	6	7	2
Hawaii	-	-	-	-	1	-	1	-	28	17	-
Guam	-	1	-	-	-	-	-	-	-	18	-
P.R.	-	-	-	-	-	-	43	36	-	6	7
V.I.	U	U	U	U	U	U	U	U	U	U	U
Amer. Samoa	U	U	U	U	U	U	U	U	U	U	U
C.N.M.I.	-	-	-	-	-	-	-	8	-	15	-

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

\*Cumulative reports of provisional tuberculosis cases for 1998 and 1999 are unavailable ("U") for some areas using the Tuberculosis Information Management System (TIMS).

**TABLE III. Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending March 6, 1999, and March 7, 1998 (9th Week)**

Reporting Area	<i>H. influenzae</i> , invasive		Hepatitis (Viral), by type				Measles (Rubeola)					
	Cum. 1999*	Cum. 1998	A		B		Indigenous		Imported <sup>†</sup>		Total	
			Cum. 1999	Cum. 1998	Cum. 1999	Cum. 1998	1999	Cum. 1999	1999	Cum. 1999	Cum. 1999	Cum. 1998
UNITED STATES	180	206	2,406	3,199	842	1,384	-	7	-	4	11	4
NEW ENGLAND	14	13	26	70	10	21	-	-	-	1	1	1
Maine	1	-	2	8	-	-	-	-	-	-	-	-
N.H.	2	1	4	3	2	2	-	-	-	1	1	-
Vt.	3	-	-	3	-	-	-	-	-	-	-	-
Mass.	8	12	7	17	6	11	-	-	-	-	-	1
R.I.	-	-	-	5	2	-	-	-	-	-	-	-
Conn.	-	-	13	34	-	8	-	-	-	-	-	-
MID. ATLANTIC	25	31	145	257	102	212	-	-	-	-	-	1
Upstate N.Y.	15	12	43	59	25	52	-	-	-	-	-	-
N.Y. City	-	10	17	97	12	56	-	-	-	-	-	-
N.J.	10	9	25	49	19	36	-	-	-	-	-	1
Pa.	-	-	60	52	46	68	-	-	-	-	-	-
E.N. CENTRAL	20	33	607	554	79	346	-	-	-	-	-	1
Ohio	13	15	125	71	19	13	-	-	-	-	-	-
Ind.	1	2	29	74	4	175	-	-	-	-	-	-
Ill.	5	15	60	146	-	49	-	-	-	-	-	-
Mich.	1	-	391	229	56	88	-	-	-	-	-	1
Wis.	-	1	2	34	-	21	-	-	-	-	-	-
W.N. CENTRAL	8	1	59	294	24	71	-	-	-	-	-	-
Minn.	2	-	4	5	4	2	-	-	-	-	-	-
Iowa	2	-	18	105	8	10	-	-	-	-	-	-
Mo.	-	-	16	151	4	51	-	-	-	-	-	-
N. Dak.	-	-	-	1	-	-	-	-	-	-	-	-
S. Dak.	1	-	-	1	-	1	-	-	-	-	-	-
Nebr.	1	-	13	4	6	2	-	-	-	-	-	-
Kans.	2	1	8	27	2	5	-	-	-	-	-	-
S. ATLANTIC	48	39	251	249	154	149	-	-	-	-	-	1
Del.	-	-	-	-	-	-	-	-	-	-	-	-
Md.	20	12	66	68	30	31	-	-	-	-	-	1
D.C.	-	-	11	10	4	2	-	-	-	-	-	-
Va.	2	5	14	32	8	13	-	-	-	-	-	-
W. Va.	1	1	1	-	-	-	-	-	-	-	-	-
N.C.	5	3	25	14	39	48	-	-	-	-	-	-
S.C.	2	-	1	7	16	-	-	-	-	-	-	-
Ga.	9	13	57	79	15	39	-	-	-	-	-	-
Fla.	9	5	76	39	42	16	-	-	-	-	-	-
E.S. CENTRAL	15	14	77	98	63	71	-	-	-	-	-	-
Ky.	2	3	6	2	7	3	-	-	-	-	-	-
Tenn.	8	6	49	51	42	55	-	-	-	-	-	-
Ala.	4	5	21	27	14	13	-	-	-	-	-	-
Miss.	1	-	1	18	-	-	-	-	-	-	-	-
W.S. CENTRAL	10	11	177	227	28	95	-	-	-	2	2	-
Ark.	-	-	6	6	7	20	-	-	-	-	-	-
La.	3	5	9	4	5	5	-	-	-	-	-	-
Okla.	5	4	59	82	8	7	-	-	-	-	-	-
Tex.	2	2	103	135	8	63	-	-	-	2	2	-
MOUNTAIN	25	38	248	571	84	139	-	1	-	-	1	-
Mont.	1	-	3	6	1	1	-	-	-	-	-	-
Idaho	1	-	8	40	4	4	-	-	-	-	-	-
Wyo.	1	-	1	10	-	1	U	-	U	-	-	-
Colo.	1	7	59	49	18	15	-	1	-	-	1	-
N. Mex.	6	-	5	35	33	50	-	-	-	-	-	-
Ariz.	11	19	131	347	11	36	-	-	-	-	-	-
Utah	4	2	12	36	7	16	-	-	-	-	-	-
Nev.	-	10	29	48	10	16	-	-	-	-	-	-
PACIFIC	15	26	816	879	298	280	-	6	-	1	7	-
Wash.	-	1	50	80	2	16	-	-	-	-	-	-
Oreg.	6	12	38	61	10	24	-	6	-	-	6	-
Calif.	8	10	725	725	282	233	-	-	-	1	1	-
Alaska	1	1	2	1	2	2	-	-	-	-	-	-
Hawaii	-	2	1	12	2	5	-	-	-	-	-	-
Guam	-	-	-	-	-	-	U	-	U	-	-	-
P.R.	-	1	9	6	13	81	-	-	-	-	-	-
V.I.	U	U	U	U	U	U	U	U	U	U	U	U
Amer. Samoa	U	U	U	U	U	U	U	U	U	U	U	U
C.N.M.I.	-	-	-	-	-	14	U	-	U	-	-	-

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

\*Of 33 cases among children aged <5 years, serotype was reported for 11 and of those, 2 were type b.

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



**TABLE III. (Cont'd.) Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending March 6, 1999, and March 7, 1998 (9th Week)**

Reporting Area	Meningococcal Disease		Mumps			Pertussis			Rubella		
	Cum. 1999	Cum. 1998	1999	Cum. 1999	Cum. 1998	1999	Cum. 1999	Cum. 1998	1999	Cum. 1999	Cum. 1998
UNITED STATES	399	633	7	60	69	131	545	686	1	5	63
NEW ENGLAND	21	36	-	1	-	6	81	141	-	-	13
Maine	3	3	-	-	-	-	-	4	-	-	-
N.H.	-	1	-	1	-	3	17	14	-	-	-
Vt.	2	1	-	-	-	-	10	24	-	-	-
Mass.	15	14	-	-	-	3	54	96	-	-	1
R.I.	1	3	-	-	-	-	-	-	-	-	-
Conn.	-	14	-	-	-	-	-	3	-	-	12
MID. ATLANTIC	43	66	1	6	6	48	80	74	-	-	41
Upstate N.Y.	8	16	-	2	2	33	57	49	-	-	37
N.Y. City	13	10	-	-	4	-	-	4	-	-	-
N.J.	13	18	-	-	-	-	-	6	-	-	4
Pa.	9	22	1	4	-	15	23	15	-	-	-
E.N. CENTRAL	60	106	3	5	9	8	67	77	-	-	-
Ohio	29	39	1	2	6	6	56	31	-	-	-
Ind.	7	19	-	-	-	-	2	4	-	-	-
Ill.	17	23	-	-	-	-	-	1	-	-	-
Mich.	7	10	2	3	3	2	9	11	-	-	-
Wis.	-	15	-	-	-	-	-	30	-	-	-
W.N. CENTRAL	26	48	1	2	5	-	5	49	-	-	-
Minn.	2	-	-	-	4	-	-	28	-	-	-
Iowa	9	9	1	2	1	-	3	10	-	-	-
Mo.	6	24	-	-	-	-	1	5	-	-	-
N. Dak.	-	-	-	-	-	-	-	-	-	-	-
S. Dak.	4	4	-	-	-	-	1	-	-	-	-
Nebr.	2	1	-	-	-	-	-	2	-	-	-
Kans.	3	10	-	-	-	-	-	4	-	-	-
S. ATLANTIC	76	95	1	12	12	5	52	54	-	3	1
Del.	1	1	-	-	-	-	-	-	-	-	-
Md.	12	13	-	2	-	-	17	11	-	-	-
D.C.	1	-	-	1	-	-	-	-	-	-	-
Va.	5	10	1	2	2	-	7	-	-	-	-
W. Va.	1	3	-	-	-	-	-	-	-	-	-
N.C.	8	18	-	1	5	2	18	30	-	3	1
S.C.	11	10	-	2	3	1	4	5	-	-	-
Ga.	14	29	-	-	-	-	-	-	-	-	-
Fla.	23	11	-	4	2	2	6	8	-	-	-
E.S. CENTRAL	33	52	-	1	-	4	14	13	-	-	-
Ky.	10	9	-	-	-	-	1	-	-	-	-
Tenn.	11	18	-	-	-	3	9	4	-	-	-
Ala.	8	21	-	1	-	1	4	9	-	-	-
Miss.	4	4	-	-	-	-	-	-	-	-	-
W.S. CENTRAL	21	35	-	9	13	3	18	19	1	2	2
Ark.	7	6	-	-	-	-	3	3	-	-	-
La.	6	10	-	-	-	-	-	-	-	-	-
Okla.	7	15	-	1	-	-	2	-	-	-	-
Tex.	1	4	-	8	13	3	13	16	1	2	2
MOUNTAIN	39	46	-	4	4	21	124	134	-	-	5
Mont.	-	2	-	-	-	-	-	1	-	-	-
Idaho	5	2	-	-	-	6	72	62	-	-	-
Wyo.	1	3	U	-	1	U	1	-	U	-	-
Colo.	8	12	-	2	-	3	8	19	-	-	-
N. Mex.	7	6	N	N	N	-	7	39	-	-	1
Ariz.	13	17	-	-	1	11	18	7	-	-	1
Utah	3	3	-	1	-	-	16	3	-	-	2
Nev.	2	1	-	1	2	1	2	3	-	-	1
PACIFIC	80	149	1	20	20	36	104	125	-	-	1
Wash.	10	20	-	-	1	31	42	47	-	-	-
Oreg.	10	31	N	N	N	-	3	8	-	-	-
Calif.	53	95	1	18	12	5	58	70	-	-	1
Alaska	3	1	-	1	2	-	1	-	-	-	-
Hawaii	4	2	-	1	5	-	-	-	-	-	-
Guam	-	-	U	-	1	U	-	-	U	-	-
P.R.	1	-	-	-	-	-	-	2	-	-	-
V.I.	U	U	U	U	U	U	U	U	U	U	U
Amer. Samoa	U	U	U	U	U	U	U	U	U	U	U
C.N.M.I.	-	-	U	-	2	U	-	-	U	-	-

N: Not notifiable

U: Unavailable

-: no reported cases

**TABLE IV. Deaths in 122 U.S. cities,\* week ending  
March 6, 1999 (9th Week)**

Reporting Area	All Causes, By Age (Years)						P&J†	Total	Reporting Area	All Causes, By Age (Years)						P&J†	Total
	All Ages	>65	45-64	25-44	1-24	<1				All Ages	>65	45-64	25-44	1-24	<1		
NEW ENGLAND	602	443	113	31	9	6	86	S. ATLANTIC	1,305	917	234	105	24	24	102		
Boston, Mass.	193	128	49	10	3	3	26	Atlanta, Ga.	U	U	U	U	U	U	U		
Bridgeport, Conn.	41	30	10	1	-	-	5	Baltimore, Md.	216	142	46	17	6	4	36		
Cambridge, Mass.	25	23	1	1	-	-	2	Charlotte, N.C.	115	85	21	4	-	5	13		
Fall River, Mass.	47	39	5	3	-	-	3	Jacksonville, Fla.	178	130	27	16	2	3	7		
Hartford, Conn.	U	U	U	U	U	U	U	Miami, Fla.	115	74	25	10	4	2	-		
Lowell, Mass.	38	30	6	2	-	-	7	Norfolk, Va.	61	46	7	4	2	2	2		
Lynn, Mass.	22	17	-	2	3	-	1	Richmond, Va.	76	54	12	7	1	2	4		
New Bedford, Mass.	29	22	6	1	-	-	5	Savannah, Ga.	45	33	11	-	1	-	4		
New Haven, Conn.	50	35	9	3	1	2	9	St. Petersburg, Fla.	107	84	13	9	-	1	7		
Providence, R.I.	U	U	U	U	U	U	U	Tampa, Fla.	209	152	35	16	3	3	25		
Somerville, Mass.	9	7	2	-	-	-	-	Washington, D.C.	159	98	32	22	5	2	4		
Springfield, Mass.	55	41	10	4	-	-	11	Wilmington, Del.	24	19	5	-	-	-	-		
Waterbury, Conn.	20	15	3	-	1	1	1	E.S. CENTRAL	924	628	196	59	20	18	57		
Worcester, Mass.	73	56	12	4	1	-	16	Birmingham, Ala.	218	145	48	12	4	6	21		
MID. ATLANTIC	2,623	1,859	504	173	40	47	146	Chattanooga, Tenn.	50	35	13	-	1	1	3		
Albany, N.Y.	73	51	16	4	1	1	6	Knockville, Tenn.	90	71	14	2	2	1	2		
Allentown, Pa.	20	16	4	-	-	-	-	Lexington, Ky.	60	39	13	5	1	2	2		
Buffalo, N.Y.	108	78	20	7	1	2	3	Memphis, Tenn.	196	131	38	19	5	3	18		
Camden, N.J.	33	23	5	2	1	2	3	Mobile, Ala.	112	68	31	8	3	2	-		
Elizabeth, N.J.	14	11	2	1	-	-	-	Montgomery, Ala.	68	52	13	1	1	1	8		
Erie, Pa.	52	43	6	-	1	2	8	Nashville, Tenn.	130	87	26	12	3	2	3		
Jersey City, N.J.	63	43	13	6	-	1	-	W.S. CENTRAL	1,367	955	257	98	27	30	106		
New York City, N.Y.	1,268	899	245	85	20	19	31	Austin, Tex.	86	56	21	5	1	3	11		
Newark, N.J.	73	36	22	9	3	3	9	Baton Rouge, La.	105	69	21	9	2	4	6		
Paterson, N.J.	U	U	U	U	U	U	U	Corpus Christi, Tex.	68	52	11	2	2	1	8		
Philadelphia, Pa.	499	323	110	42	11	13	43	Dallas, Tex.	246	158	55	23	6	4	4		
Pittsburgh, Pa.‡	65	51	10	3	-	1	5	El Paso, Tex.	72	48	17	3	3	1	3		
Reading, Pa.	24	21	2	-	-	1	1	Ft. Worth, Tex.	124	99	12	9	2	2	16		
Rochester, N.Y.	107	83	18	5	-	1	16	Houston, Tex.	U	U	U	U	U	U	U		
Schenectady, N.Y.	35	30	3	2	-	-	3	Little Rock, Ark.	88	57	18	5	4	4	7		
Scranton, Pa.	32	29	2	-	1	-	1	New Orleans, La.	125	77	31	12	2	3	9		
Syracuse, N.Y.	104	85	16	3	-	-	15	San Antonio, Tex.	266	207	32	21	2	4	28		
Trenton, N.J.	29	15	9	3	1	1	2	Shreveport, La.	60	40	14	4	2	-	3		
Utica, N.Y.	24	22	1	1	-	-	-	Tulsa, Okla.	127	92	25	5	1	4	11		
Yonkers, N.Y.	U	U	U	U	U	U	U	MOUNTAIN	1,112	815	188	65	26	17	122		
E.N. CENTRAL	2,543	1,829	453	163	48	47	199	Albuquerque, N.M.	101	78	9	10	2	2	9		
Akron, Ohio	52	38	8	1	4	1	-	Boise, Idaho	44	35	8	1	-	-	2		
Canton, Ohio	42	33	7	1	-	1	5	Colo. Springs, Colo.	56	39	10	7	-	-	9		
Chicago, Ill.	508	340	102	44	9	10	39	Denver, Colo.	136	102	19	6	2	6	10		
Cincinnati, Ohio	93	61	24	7	1	-	11	Las Vegas, Nev.	253	172	58	13	8	2	24		
Cleveland, Ohio	185	131	29	14	6	5	7	Ogden, Utah	53	42	9	2	-	-	5		
Columbus, Ohio	246	178	40	20	5	3	29	Phoenix, Ariz.	81	54	17	3	5	2	-		
Dayton, Ohio	158	131	20	5	-	2	14	Pueblo, Colo.	23	18	2	2	1	-	-		
Detroit, Mich.	257	168	56	23	4	6	8	Salt Lake City, Utah	139	98	25	10	4	2	27		
Evansville, Ind.	55	49	5	1	-	-	4	Tucson, Ariz.	226	177	31	11	4	3	36		
Fort Wayne, Ind.	82	64	16	2	-	-	8	PACIFIC	2,299	1,720	375	123	40	39	252		
Gary, Ind.	30	14	11	5	-	-	1	Berkeley, Calif.	20	14	5	1	-	-	6		
Grand Rapids, Mich.	46	31	13	1	-	1	-	Fresno, Calif.	132	100	22	5	4	1	13		
Indianapolis, Ind.	367	268	55	21	12	11	32	Glendale, Calif.	24	20	4	-	-	-	-		
Lansing, Mich.	53	41	11	1	-	-	6	Honolulu, Hawaii	77	62	10	2	2	1	5		
Milwaukee, Wis.	127	89	27	7	3	1	12	Long Beach, Calif.	62	43	12	5	2	-	12		
Peoria, Ill.	45	35	6	2	-	2	-	Los Angeles, Calif.	544	412	85	29	11	7	34		
Rockford, Ill.	60	44	11	4	-	1	11	Pasadena, Calif.	32	24	5	1	-	2	5		
South Bend, Ind.	50	42	4	1	2	1	7	Portland, Oreg.	157	121	18	11	2	5	13		
Toledo, Ohio	U	U	U	U	U	U	U	Sacramento, Calif.	349	269	45	21	7	7	77		
Youngstown, Ohio	87	72	8	3	2	2	5	San Diego, Calif.	141	104	25	7	1	4	14		
W.N. CENTRAL	883	651	154	52	10	16	89	San Francisco, Calif.	159	117	29	7	3	3	17		
Des Moines, Iowa	U	U	U	U	U	U	U	San Jose, Calif.	195	139	36	15	3	2	23		
Duluth, Minn.	56	44	9	3	-	-	14	Santa Cruz, Calif.	30	22	5	1	1	1	3		
Kansas City, Kans.	U	U	U	U	U	U	U	Seattle, Wash.	175	129	30	11	-	5	10		
Kansas City, Mo.	142	107	25	6	2	2	20	Spokane, Wash.	68	51	15	-	1	1	9		
Lincoln, Nebr.	47	36	6	4	1	-	7	Tacoma, Wash.	134	93	29	7	3	-	11		
Minneapolis, Minn.	304	224	55	16	5	4	27	TOTAL	13,658†	9,817	2,474	869	244	244	1,159		
Omaha, Nebr.	125	80	32	9	1	3	13										
St. Louis, Mo.	114	83	14	11	1	5	-										
St. Paul, Minn.	95	77	13	3	-	2	8										
Wichita, Kans.	U	U	U	U	U	U	U										

U: Unavailable - : no reported cases

\*Mortality data in this table are voluntarily reported from 122 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 this Pennsylvania city, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

¶Total includes unknown ages.

**Contributors to the Production of the *MMWR* (Weekly)  
Weekly Notifiable Disease Morbidity Data and 122 Cities Mortality Data**

Samuel L. Groseclose, D.V.M., M.P.H.

***State Support Team***

Robert Fagan  
Scott Connolly  
Gerald Jones  
David Nitschke  
Carol A. Worsham

***CDC Operations Team***

Carol M. Knowles  
Deborah A. Adams  
Willie J. Anderson  
Patsy A. Hall  
Amy K. Henion

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 [listserv@listserv.cdc.gov](mailto:listserv@listserv.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 (888) 232-3228.

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  
Jeffrey P. Koplan, M.D., M.P.H.  
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  
John W. Ward, M.D.  
Managing Editor,  
*MMWR* (weekly)  
Karen L. Foster, M.A.

Writers-Editors,  
*MMWR* (weekly)  
Jill Crane  
David C. Johnson  
Teresa F. Rutledge  
Caran R. Wilbanks  
Desktop Publishing  
Morie M. Higgins  
Peter M. Jenkins