



- 173 Update: Influenza Activity —
- United States, 1999–2000 Season 178 Update: Surveillance for West Nile Virus in Overwintering Mosquitoes—
- New York, 2000 180 Update: Pulmonary Hemorrhage/ Hemosiderosis Among Infants — Cleveland, Ohio, 1993–1996
- 185 Notice to Readers

Update: Influenza Activity --- United States, 1999-2000 Season

Influenza activity in the United States increased substantially during mid-December 1999 and appears to have peaked during the weeks ending December 25 (week 51) through January 15 (week 2). Predominant viruses isolated this season have been influenza type A(H3N2) viruses, antigenically similar to the viruses that have predominated since the 1997–98 influenza season and were well matched to this season's vaccine. This report summarizes influenza activity in the United States during October 3, 1999–February 26, 2000*, and compares the current season with the five previous seasons.

For the week ending February 26 (week 8), 1% of overall patient visits to U.S. sentinel physicians were for influenza-like illness (ILI)[†]. During October 3–February 26, the percentage of patient visits for ILI peaked at 6% during the week ending January 1 (week 52). During the five influenza seasons from 1994–95 through 1998–99, peak percentages of patient visits to sentinel physicians for ILI ranged from 5% to 7%. The weeks with the highest percentage of patient visits for ILI ranged from week 50 to week 7.

For the week ending February 26, one state epidemiologist reported widespread[§] activity, and 10 reported regional activity. During October 3–February 26, the highest combined number of reports of either widespread or regional influenza activity by state and territorial epidemiologists was 44 during the week ending January 15 (week 2). During the previous five influenza seasons, the highest total numbers of state and territorial epidemiologists reporting either widespread or regional influenza activity during any week during each of the seasons ranged from 25 to 46. The weeks with the highest number of reports of widespread or regional activity ranged from week 1 to week 10.

The percentage of total deaths attributed to pneumonia and influenza (P&I) in the 122 Cities Mortality Reporting System (MRS) was 8.6% for the week ending February 26. This was above the epidemic threshold¹ of 7.6% for that week. During Octo-

^{*}The four components of the influenza surveillance system have been described (1).

[†] Defined as temperature \geq 100 F (\geq 37.8 C) plus cough or sore throat.

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

¹The 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.

ber 3–February 26, the percentage of deaths attributed to P&I peaked at 11.2% during the week ending January 22 (week 3) (Figure 1). During the previous five influenza seasons, peak percentages of deaths attributed to P&I in the 122 Cities MRS ranged from 7.6% to 9.1%. The weeks with peak percentages of deaths attributed to P&I ranged from week 3 to week 10. This season, P&I mortality has been above the epidemic threshold for 20 of the 21 weeks during October 3–February 26.

Since the week ending October 3, the World Health Organization collaborating laboratories and the National Respiratory and Enteric Virus Surveillance System laboratories in the United States have tested 73,576 respiratory specimens for influenza viruses; 12,651 (17%) tested positive. For the week ending February 26, of 1118 specimens tested for influenza virus, 111 (10%) tested positive. During October 3–February 26, the highest percentage of specimens testing positive for influenza viruses was 33% during the week ending December 25 (week 51). During the previous five influenza seasons, peak percentages of specimens testing positive for influenza viruses ranged from 19% to 34%. The weeks with peak percentages of specimens testing positive ranged from week 51 to week 6.

Of the 12,651 positive specimens reported since October 3, 12,622 (99.8%) were type A, and 29 (0.2%) were type B. Of the 3310 influenza A viruses subtyped as of February 26, 3266 (99%) were H3N2 viruses, and 44 (1%) were H1N1 viruses. CDC has





*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.

characterized antigenically 380 influenza viruses received from U.S. laboratories since October 3. Of the 359 antigenically characterized influenza A (H3N2) viruses, 336 (94%) were similar to the vaccine strain A/Sydney/05/97, and 23 (6%) showed somewhat reduced titers to ferret antisera produced against the A/Sydney/05/97 virus. This is the third consecutive winter that the influenza A/Sydney/05/97-like viruses have predominated in the United States and worldwide. All four of the antigenically characterized U.S. influenza type B viruses were similar to the B/Beijing/184/93-like virus. Of the 17 antigenically characterized influenza A(H1N1) viruses, one was similar to the vaccine strain A/Beijing/262/95, eight were similar to the A/Bayern/07/95 virus, and eight were related more closely to the antigenic variant A/New Caledonia/20/99. A/Bayern/07/95-like viruses are distinct antigenically from the A/Beijing/262/95-like viruses; however, the A/Beijing/262/95 vaccine strain produces high titers of antibodies that cross-react with A/Bayern/07/95-like viruses.

Reported by: Participating state and territorial epidemiologists and state public health laboratory directors. World Health Organization collaborating laboratories. National Respiratory and Enteric Virus Surveillance System laboratories. Sentinel Physicians Influenza Surveillance System. Surveillance Systems Br, Div of Public Health Surveillance and Informatics, Epidemiology Program Office; Mortality Statistics Br, Div of Vital Statistics, National Center for Health Statistics; WHO Collaborating Center for Reference and Research on Influenza, Respiratory and Enteric Virus Br, and Influenza Br, Div of Viral and Rickettsial Diseases, National Center for Infectious Diseases, CDC.

Editorial Note: During the 1999–2000 season, influenza A/Sydney/05/97 (H3N2)-like viruses have predominated, with peak activity occurring during weeks 51–2. Peak activity for this season occurred approximately 4–6 weeks earlier than peak activity during the 1994–95, 1997–98, and 1998–99 influenza seasons but at approximately the same time as the 1995–96 and 1996–97 seasons. Nationally, influenza activity appears to be decreasing. This season's peak percentage of patient visits to sentinel physicians for ILI, peak percentage of respiratory specimens testing positive for influenza viruses, and peak number of state and territorial epidemiologists reporting either widespread or regional influenza activity have been within the range seen during the previous five seasons (Figure 2). However, the peak percentage of deaths attributed to P&I in the 122 Cities MRS has been higher than levels seen during the previous five seasons.

The 122 Cities MRS is a voluntary mortality reporting system that provides weekly data throughout the year to estimate the percentage of total deaths attributed to P&I. Factors that affect the percentage of P&I deaths estimated by the 122 Cities MRS include 1) the incidence of influenza in the population, 2) the level of pre-existing immunity to circulating viruses in the general population (as a result of previous natural infection or influenza vaccination), 3) the virulence of circulating influenza viruses, 4) the proportion of the population with conditions placing them at high risk for complications and death attributable to influenza, 5) the incidence and virulence of other respiratory pathogens, and 6) methodologic factors (2,3). The specific combination of factors contributing to the increased percentage of deaths attributed to P&I this season is not clear; however, one contributing factor has been a change in the P&I case definition for the 122 Cities MRS (1).

Before the 1999–2000 season, vital statistics offices participating in the 122 Cities MRS were asked to report a death as a P&I death when pneumonia was listed in part I of the death certificate or when influenza was listed anywhere on the death certificate (part I or part II). However, this case definition did not allow P&I mortality cases to be identified



FIGURE 2. Results of three influenza surveillance systems*, by week and year — United States, 1994–2000

^{*}The four components of the influenza surveillance system have been described (1).

 $^{^{\}scriptscriptstyle \dagger}\, Defined$ as temperature ≥ 100 F (≥ 37.8 C) plus cough or sore throat.

⁵ Levels of influenza activity are 1) no activity; 2) sporadic—sporadically occurring ILI or cultureconfirmed influenza with no outbreaks detected; 3) regional—outbreaks of ILI or cultureconfirmed influenza in counties with a combined population of <50% of the state's population; and 4) widespread—outbreaks of ILI or culture-confirmed influenza in counties with a combined population of ≥50% of the state's population.

easily in computerized mortality systems, and an evaluation of the 122 Cities MRS conducted in 1999 showed that the case definition was not used consistently by all cities (CDC, unpublished data, 1999). Some large cities reported P&I deaths on the basis of underlying causes of death (CDC, unpublished data, 1999). In addition, in January 1999, CDC's National Center for Health Statistics (NCHS) implemented the *International Statistical Classification of Diseases and Related Public Health Problems, 10th Revision* (ICD-10) (1). Coding rules for the underlying cause of death for pneumonia in ICD-10 substantially differ from those in *International Classification of Diseases, Ninth Revision* (ICD-(4). Among cities that reported P&I deaths using underlying causes of death coded according to ICD-10, a substantial decrease in the number of reported P&I deaths was seen in the second week of January 1999 compared with the previous week (CDC, unpublished data, 1999).

In response to inconsistent use of the old case definition and the impact of the change from ICD-9 to ICD-10 on reporting to the 122 Cities MRS in some cities, CDC modified the 122 Cities MRS case definition for reporting P&I deaths for the 1999–2000 season. Cities were asked to report a death as a P&I death when either pneumonia or influenza was listed anywhere on the death certificate (2). The new case definition is simpler and more compatible with computerized mortality systems. Many cities have implemented the new 122 Cities MRS P&I case definition; some cities continue to use underlying cause of death data coded according to ICD-10 for reporting to the 122 Cities MRS. For cities using the new reporting case definition, the number of P&I deaths reported to the 122 Cities MRS would have been expected to increase.

The effect of the concurrent ICD-9 to ICD-10 change and reporting case definition change is unclear. To clarify the impact of these changes, CDC will continue to analyze data from the 122 Cities MRS and will compare the data with vital statistics data from the NCHS. In addition, CDC will continue to examine other possible causes of the increased P&I mortality reported to the 122 Cities MRS this season. The increased P&I mortality reported this season must be interpreted with caution because influenza activity levels detected by the other three influenza surveillance systems this season have been similar to those seen during the previous five seasons.

Influenza surveillance data collected by CDC are updated weekly from October through May. Summary reports are available through CDC's voice information system, telephone (888) 232-3228, fax (888) 232-3299 (request document number 361100), or through CDC's National Center for Infectious Diseases, Division of Viral and Rickettsial Diseases, Influenza Branch World-Wide Web site, http://www.cdc.gov/ncidod/diseases/ flu/weekly.htm.

References

- 1. CDC. Influenza activity—United States, 1999–2000 season. MMWR 1999;48:1039–42.
- Simonsen L, Schonberger LB, Stroup DF, Arden NH, Cox NJ. The impact of influenza on mortality in the USA. In: Brown LE, Hampson AW, Webster RG, eds. Options for the control of influenza III. Amsterdam, Netherlands: Elsevier Science BV, 1996:26–33.
- 3. Noble GR. Epidemiological and clinical aspects of influenza. In: Beare AS, ed. Basic and applied influenza research. Boca Raton, Florida: CRC Press, 1982:11–50.
- World Health Organization. International statistical classification of diseases and related public health problems, 10th revision. Geneva, Switzerland: World Health Organization, 1993.

Update: Surveillance for West Nile Virus in Overwintering Mosquitoes — New York, 2000

Following the 1999 West Nile encephalitis outbreak in New York, guidelines were developed to direct surveillance, prevention, and control efforts in the eastern United States (1). As recommended in the guidelines, the New York City and New York state departments of health developed comprehensive West Nile virus (WNV) surveillance and control programs, which included collecting overwintering *Culex* mosquitoes to determine whether WNV might persist throughout the winter and initiate a zoonotic transmission cycle in the spring of 2000. As part of this surveillance effort, adult *Culex* mosquitoes were collected from structures in New York City during January–February 2000 to determine whether overwintering mosquitoes were infected with WNV. This report summarizes the results of this analysis, which documented WNV RNA in some mosquito pools.

Mosquitoes were sought from sites within the city's storm and sanitary sewer system, historic sites at Fort Totten in northeastern Queens, hangars and other locations at the abandoned Flushing Airport, and utility rooms under the Whitestone Bridge and under municipal swimming pools. Collection sites were selected based on location of WNV-infected humans and mosquitoes during the 1999 outbreak (2). Mosquitoes were pooled and then tested for the presence of WNV using vero cell plaque assay (3) and a fluorogenic real-time polymerase chain reaction (PCR) assay (TaqManTM, Perkin-Elmer Biosystems, Foster City, California*) that focused on three different primer pairs: the envelope protein and the NS-1 and NS-5 regions (4).

No pools produced live virus isolates in the plaque assay. However, three of the 67 pools containing *Culex* spp. mosquitoes, all of which were collected from Fort Totten, reproducibly demonstrated low but detectable levels of WNV RNA.

Reported by: J Cooper, MPA, New York City Dept of Parks and Recreation; J Miller, MD, New York City Dept of Health; P Bennett, MPH, New York City Dept of Environmental Protection; D White, PhD, P Smith, State Epidemiologist, New York State Dept of Health. Arbovirus Diseases Br, Div of Vector-Borne Infectious Diseases, National Center for Infectious Diseases, CDC.

Editorial Note: The standard technique for detecting virus in mosquitoes is the cell culture plaque assay, which detects only live virus. The real-time PCR technique was first used to detect WNV RNA in mosquitoes in the outbreak investigation during September–November 1999, and produced results consistent with those obtained by plaque assay (CDC, unpublished data, 1999). This experimental assay is highly sensitive for detecting the nucleic acids of pathogens and represents a novel approach for detecting and quantifying viruses.

In the positive pools described in this report, the intensity of the TaqMan signal was in the range consistent with approximately one plaque forming unit (vero cell plaque assay equivalent) according to a standard curve generated in the assay. The ability to detect WNV RNA in the absence of infectious viral particles might be because 1) the virus titer in the overwintering mosquito may be near or below the detectable limits of the plaque assay method; 2) the virus may be noninfectious because of biologic changes in overwintering mosquitoes; 3) the virus may have been killed during the collection and processing of specimens; 4) noninfectious viral RNA may persist in the mosquitoes; or

^{*}The use of trade names is for identification only and does not imply endorsement by CDC or the U.S. Department of Health and Human Services.

West Nile Virus — Continued

5) the results were false positives. Attempts to isolate virus from these pools are continuing using other isolation systems.

It is unknown whether WNV will persist in the New York area. Overwintering mosquitoes were difficult to locate, and intact WNV has not been identified. Three fourths of all specimens were obtained from the Fort Totten site. Surveillance of overwintering mosquitoes will continue.

WNV can be transmitted from parent to offspring mosquitoes (5), and this vertical transmission has been documented in field populations of *Culex univittatus* in Kenya (6). The role of vertical transmission in the maintenance cycles of this virus is uncertain. A related flavivirus (St. Louis encephalitis virus) may persist through the winter in vertically infected, diapausing *Culex* mosquitoes, but it is probably a rare occurrence if it occurs in nature (7).

The findings in this report demonstrate the value of continued vigilance in detecting the re-emergence of WNV. Counties where WNV transmission occurred in 1999 should monitor closely for WNV and conduct mosquito-control activities in the spring to reduce the potential for recurrence and amplification of WNV. Mosquito-control activities include reducing the number of mosquito breeding sites, particularly around homes and suburban and urban areas, and applying larvicide to *Culex* larval habitats early.

In December 1999, CDC announced availability of funds to support WNV surveillance, prevention, and control programs. The 19 state and local health departments eligible to apply for these funds represent areas where WNV transmission already has occurred or where transmission would be more likely to occur based on bird migration patterns. The focus of these cooperative agreements enables state and local health departments to increase surveillance activities and enhance laboratory capacity for detecting WNV and other arboviruses. In 2000, surveillance activities will be focused on determining whether WNV survived the winter and, if so, to ascertain its geographic distribution along the Atlantic and Gulf coasts.

References

- 1. CDC. Guidelines for surveillance, prevention, and control of West Nile virus infection— United States. MMWR 2000;49:25–8.
- 2. CDC. Update: West Nile virus encephalitis—New York, 1999. MMWR 1999;48:944-6,955.
- Beaty BJ, Calisher CH, Shope RS. Arboviruses. In: Schmidt NJ, Emmons RW, eds. Diagnostic procedures for viral, rickettsial and chlamydial infections. Washington, DC: American Public Health Association, 1989:797–856.
- 4. Perkin-Elmer Biosystems. TaqMan[™] one-step RT-PCR master mix reagents kit protocol [Product information sheet]. Foster City, California: Perkin-Elmer Biosystems, 1999.
- Baqar S, Hayes CG, Murphy JR, Watts DM. Vertical transmission of West Nile virus by *Culex* and *Aedes* species mosquitoes. Am J Trop Med Hyg 1993;48:757–62.
- Miller BR, Nasci RS, Godsey MS, et al. First field evidence for natural vertical transmission of West Nile virus in *Culex univitattus* mosquitoes from Rift Valley Province, Kenya. Am J Trop Med Hyg 2000 (in press).
- Bailey CL, Eldridge BF, Hayes DE, Watts DM, Tammariello RF, Dalrymple JM. Isolation of St. Louis encephalitis virus from overwintering *Culex pipiens* mosquitoes. Science 1978; 199:1346–9.

Update: Pulmonary Hemorrhage/Hemosiderosis Among Infants — Cleveland, Ohio, 1993–1996

A review within CDC and by outside experts of an investigation of acute pulmonary hemorrhage/hemosiderosis in infants has identified shortcomings in the implementation and reporting of the investigation described in *MMWR* (1,2) and detailed in other scientific publications authored, in part, by CDC personnel (3–5). The reviews led CDC to conclude that a possible association between acute pulmonary hemorrhage/hemosiderosis in infants and exposure to molds, specifically *Stachybotrys chartarum*, commonly referred to by its synonym *Stachybotrys atra*, was not proven. This report describes the specific findings of these internal and external reviews.

Background

In December 1994 and January 1997, articles in *MMWR* described a cluster of 10* infants from Cleveland, Ohio, with acute idiopathic pulmonary hemorrhage, also referred to as pulmonary hemosiderosis (*1,2*). The children resided in seven contiguous postal tracts and had had one or more hemorrhagic episodes, resulting in one death, during January 1993–December 1994. Preliminary results of a CDC case-control study (*2*) indicated that hemorrhage was associated with 1) major household water damage during the 6 months before illness and 2) increased levels of measurable household fungi, including the toxin-producing mold *S. chartarum* (syn. *S. atra*).

These findings and the observation that tricothecene mycotoxins were produced in the laboratory by some *S. chartarum* isolates recovered from the homes of study subjects have been published and referenced in peer-reviewed scientific literature (3–9). The hypothesis from the findings of the investigation was that infant pulmonary hemorrhage may be caused by exposure to potent mycotoxins produced by *S. chartarum* or other fungi growing in moist household environments (4,5). The findings also were cited in environmental health guidelines (10,11), congressional testimony (12), and the popular media (13–16), and have been debated among industrial hygienists and other occupational and environmental health scientists (17–21). Despite caution that "further research is needed to determine...causal[*ity*] (4)," the findings have influenced closure of public buildings, cleanup and remediation, and litigation (16,22–28).

In June 1997, a CDC scientific task force, in a review of the agency's response to the problem, advised the CDC director that concerns about the role of *S. chartarum* in pulmonary hemorrhage needed to be addressed. In response, CDC convened a multidisciplinary internal group of senior scientists (working group) and sought the individual opinions of outside experts. The working group and the outside experts conducted separate reviews of the Cleveland investigation. The working group reviewed background literature, internal CDC documents, and published CDC reports; examined the data set; and interviewed the principal investigators. The external experts reviewed relevant literature, including internal CDC documents and the working group report, and invited additional consultants to address specific topics. The working group and the external consultants each concluded that further work is needed to better describe the clinical problem, its public health impact, and the factors that put infants at risk (*29,30*).

^{*}The first report (1) described eight infants identified through November 1994. Two additional infants, identified in December 1994, were added to the original study.

Pulmonary Hemorrhage — Continued

Case Identification

The reviewers had concerns about the characterization of the clinical problem as "hemosiderosis." The acute presentation in all 10 cases, the narrow age distribution (6 weeks to 6 months), and the absence of iron deficiency suggest that the illness described in the cluster of cases in Cleveland (1,3) is clinically distinct from idiopathic pulmonary hemosiderosis (IPH), the condition to which this cluster was linked (31). Hemosiderosis (i.e., hemosiderin-laden macrophages in the interstitium and alveolar spaces of the lung) is a pathologic finding indicative of pulmonary bleeding of any type, not a unique characteristic of a specific disease, etiology, or pathophysiologic process (32,33). Therefore, in referring to the cluster of cases in Cleveland, the working group defined that cluster as AIPH in infants. From the limited clinical and historic information available to the reviewers on cases added to the Cleveland series since the original cluster (D. Dearborn, Case Western Reserve Department of Pediatrics, personal communication, September 1999), the external consultants concluded that some of these additional cases (6), including several identified in a retrospective review of sudden infant death syndrome cases (2), do not conform to the clinical patterns of cases in the original cluster. Both groups of reviewers recognized limitations that precluded drawing conclusions about clinical or etiologic ties to IPH.

Association of Household AIPH with Water Damage and Fungi

Both groups of reviewers concluded that the available evidence does not substantiate the reported epidemiologic associations—between household water damage and AIPH (3) or between household fungi and AIPH (4)—or any inferences regarding causality. The interpretation of water damage and its association with AIPH was considered to have been hampered by the limited descriptive information, by the lack of standard criteria for water damage, and by the absence of a standard protocol for inspecting and recording information from home to home. Similarly, assessment of exposure to fungi or mycotoxin also was difficult to interpret because the methods did not distinguish between contamination and clinically meaningful exposure. No isolates or serologic evidence of exposure to fungi or mycotoxin were obtained in individual case-infants.

Evaluation of Analysis Methods

Three factors, considered together, contributed to the groups' conclusions that *S. chartarum* was not clearly associated with AIPH:

1. The working group found that the reported odds ratio (OR) of 9.8 for a change of 10 colony-forming units (CFU) per m³ (4) was statistically unstable and potentially inflated. The estimate was very sensitive to at least three influential steps or strategies in the analysis. First, the mean airborne *S. chartarum* concentrations (CFU/m³) for each household were calculated incorrectly. Substituting the corrected means reduced the OR by 44% to 5.5. Second, the mean *S. chartarum* value (CFU/m³) was imputed in one case home.[†] The sample was collected many months after sampling in the other case homes and, along with all other household samples collected at the same time, produced unusually heavy growth of non-*Stachybotrys* fungi, suggesting important differences in sampling technique, laboratory

[†] An imputed value, 4 CFU/m³ (half the limit of detection divided by the number of plates), was used because colonies were detected on one or more of the plates, but were too few to count on the final platings and, therefore, recorded in the laboratory record as 0 CFU/m³.

Pulmonary Hemorrhage — Continued

procedure, or environmental conditions at the time of the sampling. Exclusion of this household from the analysis⁵ and correcting the means reduced the OR to 1.9. Third, matching on age in a small data set created an unstable OR. Subject age would not be expected to influence concurrent measurements of airborne fungi and did not correlate with the mean *S. chartarum* CFU/m³. Therefore, the strategy to match cases and controls based on age was unnecessary and potentially misleading. Analysis without the matching variable reduced the OR from 9.8 to 1.5.

- 2. Although the methods specified that sampling be done in a blinded manner (4), one investigator correctly inferred the identity of many case homes and wanted to be certain to identify culturable fungi in these homes if they were present. As a result, the investigator collected twice the number of air samples from case homes as were collected from control homes. In addition, investigators used aggressive, nonstandardized methods to generate artificial aerosols for sampling (e.g., vacuuming carpets and pounding on furnace ducts and furniture [4]), increasing the potential for differential exposure assessments of cases and controls if sampling were conducted in an unblinded manner.
- 3. Among homes classified as water damaged, the presence of any culturable airborne *S. chartarum* was identified in similar percentages of case and control homes (four of eight compared with three of seven) (CDC, unpublished data, February 1997). Although the numbers were small, this provided little evidence of a difference in the presence of airborne *S. chartarum* between water-damaged case and control homes. If the classifications of water damage were correct, this would suggest that water damage, or an unrecognized correlate of water damage, may be confounding any perceived association with *S. chartarum*.

Overall, the reviewers concluded that on the basis of these limitations the evidence from these studies was not of sufficient quality to support an association between *S. chartarum* and AIPH. In addition, the reviewers noted that evidence from other sources supporting a causal role of *S. chartarum* in AIPH is limited. First, AIPH is not consistent with historic accounts of animal and human illness caused by *S. chartarum* or related toxigenic fungi. Second, clusters of AIPH have not been reported in other flood-prone areas where growth of *S. chartarum* or other toxigenic fungi might be favored. Third, the mold-disease association observed in the Cleveland investigation was not observed in the investigation of a similar cluster in Chicago (*34*; CDC, unpublished data, May 1997).

Reported by: Office of the Director, CDC.

Editorial Note: On the basis of the findings and conclusions in the reports of the CDC internal working group and the individual opinions of the external consultants, CDC advises that conclusions regarding the possible association between cases of pulmonary hemorrhage/hemosiderosis in infants in Cleveland and household water damage or exposure to *S. chartarum* are not substantiated adequately by the scientific evidence produced in the CDC investigation (2–4). Serious shortcomings in the collection, analysis, and reporting of data resulted in inflated measures of association and restricted

[§] The working group's reported reanalysis used the value originally coded in the laboratory record (0 CFU/m³). The result was identical to that obtained by excluding the household from the analysis.

Pulmonary Hemorrhage — Continued

interpretation of the reports. The associations should be considered not proven; the etiology of AIPH is unresolved.

As a result of the reviews, CDC will implement the following:

- 1. CDC will continue to investigate cases of AIPH in infants, particularly when clusters of cases can be identified.
- 2. CDC will continue to consider possible associations between AIPH and many possible etiologies, including household water damage or exposure to environmental hydrophilic fungi/molds such as *S. chartarum*. Standardized protocols will be recommended for data collection and environmental assessment.
- 3. CDC will assist in implementation of surveillance for individual cases or clusters of cases of AIPH in infants.
- 4. In collaboration with pediatric pulmonary specialists and with state and local health officials, a consistent standard surveillance case definition will be developed for reporting.
- As part of future CDC investigations, CDC will enhance sampling and laboratory analytic methods to improve assessment of environmental exposures to molds/ fungi.

Copies of the report of the working group and a synthesis prepared by CDC of the reports individually submitted by the external experts can be accessed at http://www.cdc.gov/od/ads, then click on "Pulmonary Hemorrhage/Hemosiderosis Among Infants."

References

- 1. CDC. Acute pulmonary hemorrhage/hemosiderosis among infants—Cleveland, January 1993–November 1994. MMWR 1994;43:881–3.
- 2. CDC. Update: pulmonary hemorrhage/hemosiderosis among infants—Cleveland, Ohio, 1993–1996. MMWR 1997;46:33–5.
- 3. Montaña E, Etzel RA, Allan T, Horgan T, Dearborn DG. Environmental risk factors associated with pediatric idiopathic pulmonary hemorrhage and hemosiderosis in a Cleveland community. Pediatrics 1997;99:117–24.
- 4. Etzel RA, Montaña E, Sorenson WG, et al. Acute pulmonary hemorrhage in infants associated with exposure to *Stachybotrys atra* and other fungi. Arch Pediatr Adolesc Med 1998;152:757–62.
- Jarvis BB, Sorenson WG, Hintikka EL, et al. Study of toxin production by isolates of Stachybotrys chartarum and Memnoniella echinata isolated during a study of pulmonary hemosiderosis in infants. Appl Environ Microbiol 1998;64:3620–5.
- Dearborn DG, Yike I, Sorenson WG, Miller MJ, Etzel RA. Overview of investigations into pulmonary hemorrhage among infants in Cleveland, Ohio. Environ Health Perspect 1999;107(suppl 3):495–9.
- 7. Elidemir O, Colasurdo GN, Rossmann SN, Fan LL. Isolation of *Stachybotrys* from the lung of a child with pulmonary hemosiderosis. Pediatrics 1999;104:964–6.
- Flappan SM, Portnoy J, Jones P, Barnes C. Infant pulmonary hemorrhage in a suburban home with water damage and mold (*Stachybotrys atra*). Environ Health Perspect 1999;107:927–30.
- Etzel RA, Dearborn DG. Pulmonary hemorrhage among infants with exposure to toxigenic molds: an update. In: Johanning E, ed. Bioaerosols, fungi and mycotoxins: health effects, assessment, prevention and control. Albany, New York: Eastern New York Occupational and Environmental Health Center, 1999:79–83.
- American Academy of Pediatrics Committee on Environmental Health. Toxic effects of indoor molds. Pediatrics 1998;101:712–4.

Pulmonary Hemorrhage — Continued

- 11. Etzel RA, ed. Handbook of pediatric environmental health. Elk Grove Village, Illinois: American Academy of Pediatrics, 1999.
- Hearings before the House Appropriations Committee, Labor, Health & Human Services, & Education subcommittee, 104th Cong, 2nd Sess (1998) testimony of DG Dearborn, M.D.
- 13. Lyman F. A fungus among us: protecting kids from indoor molds. MSNBC Health News [serial online]; October 13, 1999.
- 14. Marino J. Death of innocents. Cleveland SCENE/News; March 25-31, 1999:9,11-3.
- 15. Davidson J, Mulvihill K. Sick schools. Good Housekeeping; May 1999:124-7,184,187.
- Mann A. Mold: a health alert. USA Weekend Online Special Reports [serial online]; December 5, 1999.
- 17. Dearborn DG. Cleveland cluster of infant pulmonary hemorrhage: a *Stachybotrys* connection? Invironment Professional, June 1997;3:1,4–6.
- 18. Light E. Is it time for an objective look at *Stachybotrys*? Invironment Professional, October 1997;3:1,4–6.
- Johanning E, Morey P. Health and safety hazards of fungi, mold and microbials. What you need to know about the hazards and your personal health and safety. Cleaning and Restoration; February 1998:18–30.
- 20. Light E, Gots R. Mold revisited: a rebuttal. Cleaning and Restoration; July 1998:22-6.
- 21. Page E, Trout D. Mycotoxins and building-related illness. J Occ Env Med 1998;40:761-2.
- 22. Holloway L. Poisonous mold shuts a renovated library on Staten Island. New York Times; October 4, 1997: Metropolitan Desk, B, 1.
- 23. Lii J. Mold forces 2 more branches of the public library to close. New York Times; October 26, 1997: Sunday late edition, S1, 33.
- 24. Holloway L. Families plagued by home-wrecking mold. New York Times; November 9, 1997:1, 39.
- 25. Contiguglia F. New day care center has fungus problem. Washington, DC: Roll Call; May 7, 1998:1, 30.
- Arena S. Mold's toxic, tenants say in \$8B suit. New York Daily News; May 18, 1999: News & Views Beat, http://www.nydailynews.com.
- Stapleton branch library reopens Saturday, February 28 [press release]. New York: New York Public Library; http://www.nypl.org/admin/pro/press/stapleton.html. Accessed March 6, 2000.
- 28. Hilts PJ. The mold scare: overblown or not. October 23, 1997: New York Times; House & Home/Style Desk, F, 10.
- CDC. Report of the CDC Working Group on Pulmonary Hemorrhage/Hemosiderosis. June 17, 1999. Available at http://www.cdc.gov/od/ads, click on "Pulmonary Hemorrhage/ Hemosiderosis Among Infants."
- 30. CDC. Reports of Members of the CDC External Expert Panel on Acute Idiopathic Pulmonary Hemorrhage in Infants: a synthesis. December 1999. Available at http://www.cdc.gov/ od/ads, click on "Pulmonary Hemorrhage/Hemosiderosis Among Infants."
- Levy J, Wilmott R. Pulmonary hemosiderosis. In: Hillman BC, ed. Pediatric respiratory disease: diagnosis and treatment. Philadelphia, Pennsylvania: WB Saunders Co., 1993: 543–9.
- Boat TM. Pulmonary hemorrhage and hemoptysis. In: Chernick V, Boat TF, Kendig EL, eds. Disorders of the respiratory tract in children. Philadelphia, Pennsylvania: WB Saunders Co., 1998:623–33.
- Bowman CM. Pulmonary hemosiderosis. In: Loughllin GM, Eigen H, eds. Respiratory disease in children. Baltimore, Maryland: Williams & Wilkins, 1994:417–20.
- 34. CDC. Acute pulmonary hemorrhage among infants—Chicago, April 1992–November 1994. MMWR 1995;44:67,73–4.

Updated Guidelines for the Use of Rifabutin or Rifampin for the Treatment and Prevention of Tuberculosis Among HIV-Infected Patients Taking Protease Inhibitors or Nonnucleoside Reverse Transcriptase Inhibitors

A previously published report provided guidelines for managing the pharmacologic interactions that can result when patients receive protease inhibitors and nonnucleoside reverse transcriptase inhibitors (NNRTIs) for treatment of human immunodeficiency virus (HIV) infection together with rifamycins for the treatment of tuberculosis (TB) (1). Protease inhibitors and NNRTIs are antiretroviral agents that are substrates that may inhibit or induce cytochrome P-450 isoenzymes (CYP450). Rifamycins are antituberculosis agents that induce CYP450 and may decrease substantially blood levels of the antiretroviral drugs. The pharmacologic interactions are called "drug-drug" because, in addition to the effect rifamycins have on protease inhibitors and NNRTIs, the antiretroviral agents may affect the blood levels of rifamycins. This notice presents updated data pertaining to drug-drug interactions between these agents and recommendations for their use from a group of CDC scientists and outside expert consultants (1).

The other class of antiretroviral agents available in the United States—nucleoside reverse transcriptase inhibitors (NRTIs) (zidovudine, didanosine, zalcitabine, stavudine, lamivudine, and the new drug abacavir [2])—are not metabolized by CYP450. Concurrent use of NRTIs and rifamycins is not contraindicated and does not require dose adjustments.

Drug regimens that include rifabutin instead of rifampin previously were suggested as the preferable alternative for the treatment of active TB among patients taking protease inhibitors or NNRTIS (1). The use of rifampin to treat active TB was specifically contraindicated for patients who take any of the protease inhibitors or NNRTIs, and the use of rifabutin was contraindicated for patients taking the protease inhibitor ritonavir or the NNRTI delavirdine. New data indicate that rifampin can be used for the treatment of active TB in three situations: 1) in a patient whose antiretroviral regimen includes the NNRTI efavirenz (3) and two NRTIs; 2) in a patient whose antiretroviral regimen includes the protease inhibitor ritonavir (4) and one or more NRTIs; or 3) in a patient whose antiretroviral regimen includes the combination of two protease inhibitors (5) (ritonavir and either saquinavir hard-gel capsule [HGC] or saquinavir soft-gel capsule [SGC]) (Table 1). In addition, the updated guidelines recommend substantially reducing the dose of rifabutin (150 mg two or three times per week) when it is administered to patients taking ritonavir ($\boldsymbol{6}$) (with or without saquinavir HGC or saquinavir SGC) and increasing the dose of rifabutin (either 450 mg or 600 mg daily or 600 mg two or three times per week) when rifabutin is used concurrently with efavirenz (Table 1) (7).

Of the available protease inhibitors, ritonavir has the highest potency in inhibiting CYP450 (1). The inhibition of this pathway increases plasma concentrations of other coadministered protease inhibitors, an interaction exploited in different combinations (e.g., ritonavir at low doses [400 mg twice per day] in combination with saquinavir [400 mg twice per day] substantially increases blood levels of saquinavir) (8). For patients treated with two protease inhibitors, the complexity of drug interactions is amplified, and

Antiretroviral	Use in combination with rifabutin	Use in combination with rifampin	Comments
Saquinavir*			
Hard-gel capsules (HGC)	Possibly†, if antiretroviral regimen also includes ritonavir	Possibly, if antiretroviral regimen also includes ritonavir	Coadministration of saquinavir SGC with usual-dose rifabutin (300 mg daily or two or three times per week) is a possibility. However, the pharmacokinetic data and clinical experience for this combination are limited.
Soft-gel capsules (SGC)	Probably ^s	Possibly, if antiretroviral regimen also includes ritonavir	The combination of saquinavir SGC or saquinavir HGC and ritonavir, coadministered with 1) usual-dose rifampin (600 mg daily or two or three times per week), or 2) reduced-dose rifabutin (150 mg two or three times per week) is a possibility. However, the pharmacokinetic data and clinical experience for these combinations are limited.
			Coadministration of saquinavir HGC or saquinavir SGC with rifampin (in the absence of ritonavir) is not recommended because rifampin markedly decreases concentrations of saquinavir.
Ritonavir	Probably	Probably	If the combination of ritonavir and rifabutin is used, then a substantially reduced-dose rifabutin regimen (150 mg two or three times per week) is recommended.
			Coadministration of ritonavir with usual-dose rifampin (600 mg daily or two or three times per week) is a possibility, though pharmacokinetic data and clinical experience are limited.
Indinavir	Yes	No	There is limited, but favorable, clinical experience with coadministration of indinavir [¶] with a reduced daily dose of rifabutin (150 mg) or with the usual dose of rifabutin (300 mg two or three times per week).
			Coadministration of indinavir with rifampin is not recommended because rifampin markedly decreases concentrations of indinavir.
Nelfinavir	Yes	No	There is limited, but favorable, clinical experience with coadministration of nelfinavir** with a reduced daily dose of rifabutin (150 mg) or with the usual dose of rifabutin (300 mg two or three times per week).

Yes	No	Coadministration of amprenavir with a reduced daily dose of rifabutin (150 mg) or with the usual dose of rifabutin (300 mg two or three times per week) is a possibility, but there is no published clinical experience.	Notice
		Coadministration of amprenavir with rifampin is not recommended because rifampin markedly decreases concentrations of amprenavir.	to Rea
Yes	Possibly	Coadministration of nevirapine with usual-dose rifabutin (300 mg daily or two or three times per week) is a possibility based on pharmacokinetic study data. However, there is no published clinical experience for this combination.	iders — Co
		Data are insufficient to assess whether dose adjustments are necessary when rifampin is coadministered with nevirapine. Therefore, rifampin and nevirapine should be used only in combination if clearly indicated and with careful monitoring.	ntinued
No	No	Contraindicated because of the marked decrease in concentrations of delavirdine when administered with either rifabutin or rifampin.	
Probably	Probably	Coadministration of efavirenz with increased-dose rifabutin (450 mg or 600 mg daily, or 600 mg two or three times per week) is a possibility, though there is no published clinical experience.	
		Coadministration of efavirenz ^{‡†} with usual-dose rifampin (600 mg daily or two or three times per week) is a possibility, though there is no published clinical experience.	

* Usual recommended doses are 400 mg two times per day for each of these protease inhibitors and 400 mg of ritonavir.

[†] Despite limited data and clinical experience, the use of this combination is potentially successful.

Amprenavir

Nevirapine

Delavirdine

Efavirenz

[§] Based on available data and clinical experience, the successful use of this combination is likely.

¹ Usual recommended dose is 800 mg every 8 hours. Some experts recommend increasing the indinavir dose to 1000 mg every 8 hours if indinavir is used in combination with rifabutin.

** Usual recommended dose is 750 mg three times per day or 1250 mg twice daily. Some experts recommend increasing the nelfinavir dose to 1000 mg if the threetimes-per-day dosing is used and nelfinavir is used in combination with rifabutin.

¹¹ Usual recommended dose is 600 mg daily. Some experts recommend increasing the efavirenz dose to 800 mg daily if efavirenz is used in combination with rifampin.

Notice to Readers — Continued

recommendations about dose modifications are difficult when rifamycins also are administered. However, if ritonavir (taken in doses ranging from 100 mg to 600 mg twice per day) is combined with any other protease inhibitor for HIV therapy, and the administration of rifabutin also becomes necessary, the need to use substantially reduced doses of rifabutin (150 mg two or three times per week) is certain. In comparison, for a patient who is undergoing treatment with saquinavir SGC (a relatively weak CYP450 inhibitor [1]) and two NRTIs, the usual dosage (300 mg daily or two or three times per week) of rifabutin should not be decreased (9). When both an inhibitor and an inducer of CYP450 are used with rifamycins (e.g., a protease inhibitor in combination with a NNRTI), a different complex interaction occurs and the appropriate drug-dose adjustments necessary to ensure optimum levels of both antiretroviral drugs and rifamycins are unknown.

Alternatively, for patients undergoing therapy with complex combinations of protease inhibitors or NNRTIs, the use of antituberculosis regimens containing no rifamycins can be considered. Isoniazid does not have an interactive effect with either the protease inhibitors or NNRTIs, and the use of a 9-month regimen of isoniazid is recommended as the preferred option for treatment for latent *Mycobacterium tuberculosis* infection (LTBI) (10). However, 2-month regimens of a rifamycin and pyrazinamide also are recommended for LTBI therapy (10). If these regimen options are chosen for HIV-infected patients with LTBI, the drug-drug interactions and dose adjustments for antiretroviral drugs and rifamycins apply. However, for HIV-infected patients with active TB, use of a treatment regimen that does not contain a rifamycin, although possible, may be suboptimal and usually is not recommended.

The management of HIV-infected patients taking protease inhibitors or NNRTIs and undergoing treatment for active TB with rifabutin or rifampin should be directed by, or conducted in consultation with, a physician with experience in the care of patients with these two diseases. This care should include close attention to the possibility of TB treatment failure, antiretroviral treatment failure, paradoxical reactions of TB, unique and synergistic side effects for all drugs used, and drug toxicities associated with increased serum concentrations of rifamycins.

Copies of these guidelines are available from CDC's National Center for HIV, STD, and TB Prevention, 1600 Clifton Road, N.E., Mailstop E-06, Atlanta, GA 30333, or from the CDC World-Wide Web site, http://www.cdc.gov/nchstp/tb.

References

- CDC. Prevention and treatment of tuberculosis among patients infected with human immunodeficiency virus: principles of therapy and revised recommendations. MMWR 1998;47(no. RR-20).
- 2. Glaxo Wellcome Inc. Abacavir package insert. Research Triangle Park, North Carolina: Glaxo Wellcome Inc., 1998.
- 3. Benedek IH, Joshi A, Fiske WD, et al. Pharmacokinetic interaction between efavirenz and rifampin in healthy volunteers [Abstract]. In: Program and abstracts of the 12th World AIDS Conference, Geneva, Switzerland, 1998.
- 4. Abbott Laboratories. Norvir package insert. Chicago, Illinois: Abbott Laboratories, 1999.
- 5. Veldkamp AI, Hoetelmans MW, Beijnen JH, Mulder JW, Meenhorst PL. Ritonavir enables combined therapy with rifampin and saquinavir. Clin Infect Dis 1999;29:1586.
- Gallicano K, Khaliq Y, Seguin I, et al. A pharmacokinetic study of intermittent rifabutin dosing with a combination of ritonavir and saquinavir in HIV patients [Abstract]. In: Program and abstracts of the 7th Conference on Retroviruses and Opportunistic Infections, San Francisco, California, 2000.

Notice to Readers — Continued

- Benedek IH, Fiske WD, White SJ, Stevenson D, Joseph JL, Kornhauser DM. Pharmacokinetic interaction between multiple doses of efavirenz and rifabutin in healthy volunteers [Abstract]. In: Program and abstracts of the 36th Annual Meeting of the Infectious Disease Society of America, Denver, Colorado, 1998.
- Dietrich MA, Butts JD, Raasch RH. HIV-1 protease inhibitors: a review. Infect Med 1999;16:716–38.
- 9. Jorga K, Buss NE. Pharmacokinetic drug interaction with saquinavir soft gelatin capsule [Abstract]. In: Program and abstracts of the 39th Interscience Conference on Antimicrobial Agents and Chemotherapy, San Francisco, California, 1999.
- 10. American Thoracic Society/CDC. Targeted tuberculin testing and treatment of latent tuberculosis infection. Am J Respir Crit Care Med 2000 (in press).

Errata: Vol. 49, No. 8

In the article "Monitoring Hospital-Acquired Infections to Promote Patient Safety— United States, 1990–1999," the data reported in Table 1 on page 151 were incorrect. Table 1 represents data from the National Nosocomial Infection Surveillance (NNIS) system for 1992–1999. The correct data for 1997–1999 are on page 190.

In the article "Corporate Action to Reduce Air Pollution—Atlanta, Georgia, 1998– 1999," on page 154 in the second paragraph there is a reference to Table 2. There was no Table 2 for that article.

		Total no. of			Device-associated infection rates							
	No.	days patient	Device			Percentiles						
ICU/Type of infection	units	in ICU	days*	DU⁺	Mean	10th	25th	50th	75th	90th		
Coronary		377,242										
Catheter-associated urinary tract infection [§]	79		192,226	0.51	5.6	0.9	2.6	4.5	8.1	12.3		
Central line-associated bloodstream infection [¶]	79		118,914	0.32	4.3	0.0	1.8	3.9	5.9	9.1		
Ventilator-associated pneumonia**	78		83,735	0.22	7.6	1.0	3.9	7.1	10.5	14.8		
Medical (nonsurgical)		651,356										
Catheter-associated urinary tract infection	107		483,209	0.74	6.5	2.0	3.6	6.1	8.3	10.6		
Central line-associated bloodstream infection	108		337,722	0.52	6.1	1.6	3.7	5.7	7.6	10.1		
Ventilator-associated pneumonia	107		322,825	0.50	6.6	1.9	3.3	6.3	8.2	12.2		
Pediatric		318,629										
Catheter-associated urinary tract infection	55		103,505	0.32	4.9	0.0	2.0	4.7	6.6	8.6		
Central line-associated bloodstream infection	56		145,532	0.46	7.7	1.5	3.7	6.8	9.5	12.1		
Ventilator-associated pneumonia	56		142,475	0.45	5.0	0.2	1.6	3.7	7.9	11.3		
Surgical		665,638										
Catheter-associated urinary tract infection	122		566,054	0.85	5.0	1.5	2.8	4.4	6.9	10.1		
Central line-associated bloodstream infection	122		444,040	0.67	5.4	1.1	2.3	4.9	6.9	9.9		
Ventilator-associated pneumonia	120		319,627	0.48	13.0	5.2	7.3	11.3	14.9	23.6		

 TABLE 1. Device-associated infection rates, by type of device and type of intensive care unit (ICU) — National Mathematical Nosocomial Infection Surveillance system, United States, 1997–1999
 Intensive care unit (ICU) — National Mathematical States, 1997–1999

* Number of days a urinary catheter, central line, or ventilator was used by all patients.

⁺ Device utilization ratio (device days divided by total number of days patient was in ICU).

⁵ Number of urinary catheter-associated urinary tract infections divided by number of days a urinary catheter was used multiplied by 1000.

¹ Number of central line-associated bloodstream infections divided by number of days a central line was used multiplied by 1000.

** Number of ventilator-associated cases of pneumonia divided by number of days a mechanical ventilator was used multiplied by 1000.





*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 4, 2000 (9th Week)

		Cum. 2000		Cum. 2000
Anthrax Brucellosis* Cholera Congenital rul Cyclosporiasis	bella syndrome *	- 3 - 1 2	HIV infection, pediatric* ^s Plague Poliomyelitis, paralytic Psittacosis* Rabies, human	34 2 - 1 -
Diphtheria Encephalitis:	California* serogroup viral eastern equine* St. Louis* western equine*	- 1 - -	Rocky Mountain spotted fever (RMSF) Streptococcal disease, invasive Group A Streptococcal toxic-shock syndrome* Syphilis, congenital ¹ Tetanus	22 486 20 - 2
Ehrlichiosis Hansen Diseas Hantavirus pu Hemolytic ure	human granulocytic (HGE)* human monocytic (HME)* se* Imonary syndrome*†. mic syndrome, post-diarrheal*	12 1 6 - 8	Toxic-shock syndrome Trichinosis Typhoid fever Yellow fever	24 1 46 -

-: 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).

⁵ 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 February 27, 2000. ¹Updated from reports to the Division of STD Prevention, NCHSTP.

							Escherichia coli 0157:H7*					
	AIE	os	Chlan	nydia ^s	Cryptos	poridiosis	NE	rss	PH	LIS		
Reporting Area	Cum. 2000 [†]	Cum. 1999	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999		
UNITED STATES	6,288	6,945	74,379	113,160	151	235	209	184	88	139		
NEW ENGLAND Maine N.H. Vt. Mass. R.I. Conn.	511 6 5 1 370 17 112	352 5 13 4 238 20 72	3,680 221 133 88 1,506 370 1,362	3,616 124 181 85 1,535 375 1,316	6 1 - 4 - 1	10 1 1 6 - 1	16 1 3 1 5 - 6	29 1 1 1 17 - 9	15 1 3 2 3 - 6	28 - 1 - 14 1 12		
MID. ATLANTIC Upstate N.Y. N.Y. City N.J. Pa.	1,592 65 986 387 154	1,492 76 835 370 211	890 N 516 374	13,399 N 6,540 2,139 4,720	15 8 4 - 3	43 17 21 1 4	23 23 - N	11 8 1 2 N		2 - 1 1 -		
E.N. CENTRAL Ohio Ind. III. Mich. Wis.	590 92 56 353 67 22	489 97 52 231 81 28	14,000 3,184 2,044 3,701 3,759 1,312	18,105 5,992 1,897 4,633 3,494 2,089	17 11 3 - 3	45 6 3 6 5 25	22 8 2 8 4 N	35 20 6 4 5 N	6 3 - 1 1	22 7 7 3 2 3		
W.N. CENTRAL Minn. Iowa Mo. N. Dak. S. Dak. Nebr. Kans.	151 32 10 70 - 2 7 30	161 28 13 84 3 3 10 20	4,115 996 547 902 - 298 465 907	7,151 1,354 342 3,266 161 370 666 992	8 - 1 3 1 1 2 -	19 10 1 - 1 1 2	56 10 9 30 2 - 2 3	34 10 5 2 2 - 3 12	26 10 4 8 1 - 2 1	27 11 2 2 1 - 11		
S. ATLANTIC Del. Md. D.C. Va. W. Va. N.C. S.C. Ga. Fla.	1,531 26 153 112 115 6 75 156 183 705	1,832 31 252 69 102 14 125 128 207 904	14,181 500 971 507 2,001 219 3,187 669 2,523 3,604	24,118 524 2,289 N 2,484 411 3,953 4,376 4,878 5,203	22 - - - - - - - - - - - - - - - - - -	30 - 4 3 - - 1 - 21 1	20 - - 4 1 6 - 2 2	17 1 - 5 - 3 1 1 5	13 - U 4 1 - 3 3	10 - - 2 1 3 1 U 3 3		
E.S. CENTRAL Ky. Tenn. Ala. Miss.	281 37 105 92 47	300 37 130 69 64	7,180 1,455 1,809 2,169 1,747	7,267 1,251 2,463 2,501 1,052	6 - - 6 -	2 1 - -	10 4 5 1	15 5 6 2 2	4 U 4 -	5 U 2 2 1		
W.S. CENTRAL Ark. La. Okla. Tex.	542 20 92 16 414	980 34 67 19 860	12,517 554 2,232 1,265 8,466	14,427 862 1,436 1,488 10,641	5 1 - 1 3	15 - 12 1 2	8 2 - 3 3	7 2 3 1 1	11 1 6 3 1	10 2 2 6		
MOUNTAIN Mont. Idaho Wyo. Colo. N. Mex. Ariz. Utah Nev.	213 3 1 52 26 56 28 44	207 3 56 9 86 27 21	3,470 64 133 602 334 1,407 387 543	5,930 208 326 1,230 903 2,260 318 550	9 - 1 1 1 2 3 -	22 1 2 10 7 N	22 5 3 7 - 3 1 1	12 - 1 3 1 3 4 -	4 - - 1 - 2 1 -	10 - 2 1 - 1 4 1		
PACIFIC Wash. Oreg. Calif. Alaska Hawaii	877 102 22 727 	1,132 58 32 1,021 5 16	14,346 2,247 454 11,402 243	19,147 2,133 954 15,195 317 548	63 N 1 62	49 N 3 46	32 3 3 23 - 3	24 1 10 13 -	9 3 - - 3	25 9 8 - -		
Guam P.R. V.I. Amer. Samoa C.N.M.I.	9 153 6 - -	1 215 3 -	142	85 U U U U	- - -	- U U U	N - - -	N 1 U U				

TABLE II. Provisional cases of selected notifiable diseases, United States, weeks ending March 4, 2000, and March 5, 1999 (9th Week)

N: Not notifiable U: Unavailable -: no reported cases C.N.M.I.: Commonwealth of Northern Mariana Islands * Individual cases may be reported through both the National Electronic Telecommunications System for Surveillance (NETSS) and the Public

Health Laboratory Information System (PHLIS). Updated monthly from reports to the Division of HIV/AIDS Prevention–Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention, last update February 27, 2000.

⁵ Chlamydia refers to genital infections caused by C. trachomatis. Totals reported to the Division of STD Prevention, NCHSTP.

	Gono	orrhea	Her C/N	oatitis IA NB	Legio	nellosis	Ly Dis	rme
Benorting Area	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.	Cum.
UNITED STATES	47,528	62,629	301	590	<u>2000</u> 92	153	476	746
NEW ENGLAND Maine N.H. Vt. Mass. R.I. Conn.	1,132 12 13 4 431 87 585	1,301 9 15 11 514 98 654		2 - 1 1 -	5 2 1 - 1 - 1	11 2 1 3 2 1 2	60 - - - 38 - 7	153 1 - 72 - 80
MID. ATLANTIC Upstate N.Y. N.Y. City N.J. Pa.	1,212 648 302 262	7,561 806 3,216 1,280 2,259	3 3 - -	21 11 - 10	15 6 - 9	40 8 7 5 20	335 115 2 218	429 85 15 100 229
E.N. CENTRAL Ohio Ind. III. Mich. Wis.	8,749 1,837 931 2,179 2,987 815	10,859 2,881 1,173 3,343 2,519 943	48 - - 3 45 -	316 - - 91 219	27 16 3 1 6 1	47 14 10 13 9	2 2 - - U	25 8 - 1 1 15
W.N. CENTRAL Minn. Iowa Mo. N. Dak. S. Dak. Nebr. Kans.	1,455 404 121 367 - 45 141 377	3,499 520 144 2,112 9 30 301 383	43 - - 388 - - 1 4	39 - - 34 - - 1 4	4 1 2 - - -	4 - 2 1 - 1 -	13 2 - 3 - - 8	9 1 2 1 - - 3
S. ATLANTIC Del. Md. D.C. Va. W. Va. N.C. S.C. Ga. Fla.	10,795 260 455 427 1,446 50 3,097 574 1,810 2,676	19,133 302 2,839 1,310 2,009 115 3,540 2,150 3,171 3,697	11 - 2 - 1 5 - 3	36 - 18 - 2 8 1 1 -	21 1 6 3 N 2 2 7	18 2 2 7 8 7 8 7 8 4 4 4 4 4	48 1 37 1 3 4 - 2	91 4 74 1 - 1 11 - -
E.S. CENTRAL Ky. Tenn. Ala. Miss.	5,277 611 1,469 1,817 1,380	6,121 667 2,016 2,267 1,171	51 5 15 3 28	41 5 22 1 13	2 - 1 1 -	9 5 4 -		11 - 3 5 3
W.S. CENTRAL Ark. La. Okla. Tex.	14,591 319 9,531 594 4,147	8,163 408 1,453 785 5,517	68 1 31 - 36	62 2 47 1 12	- - - -	1 - 1 -		
MOUNTAIN Mont. Idaho Wyo. Colo. N. Mex. Ariz. Utah Nev.	1,420 4 12 648 62 440 52 202	1,706 3 23 6 372 180 853 34 235	46 - 31 7 4 4 -	46 4 17 4 6 10 1	8 - - 4 - - 3 -	12 - - 1 1 5 4	1 - - 1 -	1 - - 1 - -
PACIFIC Wash. Oreg. Calif. Alaska Hawaii	2,897 411 56 2,401 29	4,286 367 141 3,620 62 96	31 3 7 21 -	27 2 3 22	10 2 N 8 -	11 2 N 9 -	17 - 1 16 - N	27 1 26 N
Guam P.R. V.I. Amer. Samoa C.N.M.I.	30 - -	15 54 U U U	- 1 - -	U U U	- - - -	U U U	N - -	N U U U

TABLE II. (Cont'd) Provisional cases of selected notifiable diseases, United States, weeks ending March 4, 2000, and March 5, 1999 (9th Week)

N: Not notifiable

U: Unavailable

- : no reported cases

	Weeks	chang h	ui oli 4, 20					
						Salmo	nellosis*	
Benorting Area	Cum.	laria Cum.	Rabies Cum. 2000	s, Animal Cum. 1900	Cum.	Cum.	Cum.	ILIS Cum.
UNITED STATES	111	201	581	794	3.255	4,188	1.814	3.848
NEW ENGLAND Maine N.H. Vt.		3	68 16 2 4	114 19 5 18	210 25 14 5	226 23 3 10	202 9 8 3	240 13 11 10
R.I. Conn.	-	- -	25 - 21	33 10 29	125 4 37	133 10 47	132 12 38	129 21 56
MID. ATLANTIC Upstate N.Y. N.Y. City N.J. Pa.	12 7 2 3	62 12 30 15 5	126 100 U 14 12	170 108 U 37 25	291 77 114 100	647 115 208 161 163	193 24 169	478 144 195 135 4
E.N. CENTRAL Ohio Ind. III. Mich. Wis.	7 2 2 3	23 2 4 9 5 3	2 - - -	1 - - 1 -	430 140 45 134 71 40	654 148 34 198 162 112	235 70 44 - 88 33	563 114 40 205 143 61
W.N. CENTRAL Minn. Iowa Mo. N. Dak. S. Dak. Nebr. Kans.	4 - - - 1 1	7 - 5 - - -	53 18 7 2 8 7 - 11	114 15 16 5 15 29 1 33	197 42 17 65 2 7 26 38	228 53 30 57 1 7 18 62	142 42 11 44 10 11 7 17	257 92 28 73 11 13 17 23
S. ATLANTIC Del. Md. D.C. Va. W. Va. N.C. S.C. Ga. Fla.	34 18 11 4 - 1	47 - 18 5 7 1 3 - 5 8	251 10 47 - 18 52 14 28 15	277 3 - 61 15 62 11 28 24	564 8 103 - 66 20 132 55 67 113	741 15 91 16 84 13 170 38 147 167	367 7 50 12 67 41 117	695 13 84 U 90 19 141 53 205 90
E.S. CENTRAL Ky. Tenn. Ala. Miss.	4 1 - 3	5 1 2 2	23 4 16 3	39 12 18 9	163 19 40 70 34	264 57 75 77 55	89 16 47 23 3	166 36 75 47 8
W.S. CENTRAL Ark. La. Okla. Tex.	1 - 1 -	9 1 6 1 1	8 - - 8 -	14 - - 14 -	192 31 24 23 114	297 41 46 32 178	238 22 68 18 130	396 35 59 14 288
MOUNTAIN Mont. Idaho Wyo. Colo. N. Mex. Ariz. Utah Nev.	8 - 4 - 2 2	10 1 - 3 1 3 1 -	27 9 - 14 - 1 3 -	20 8 - 5 1 - 6 - -	296 11 21 6 59 30 90 49 30	294 3 10 2 84 31 100 34 30	210 - - 58 21 93 38 -	285 1 15 84 39 82 38 21
PACIFIC Wash. Oreg. Calif. Alaska Hawaii	41 2 4 35 -	35 2 6 24 - 3	23 - 17 6 -	45 - - 42 3 -	912 32 42 787 12 39	837 34 65 678 6 54	138 59 49 - 2 28	768 109 86 514 4 55
Guam P.R. V.I. Amer. Samoa C.N.M.I.	-	- - - - - - - - - - - - - - - - - - -	6	7 U U U	10 - - -	13 57 U U U	U U U U U	U U U U U

TABLE II. (Cont'd) Provisional cases of selected notifiable diseases, United States, weeks ending March 4, 2000, and March 5, 1999 (9th Week)

N: Not notifiable U: Unavailable -: no reported cases *Individual cases may be reported through both the National Electronic Telecommunications System for Surveillance (NETSS) and the Public Health Laboratory Information System (PHLIS).

		Shigel	losis*		Sy	philis				
	NET	'SS	P	HLIS	(Primary 8	Secondary)	Tube	rculosis		
Reporting Area	2000	Cum. 1999	2000	1999	2000	1999	2000	1999 [†]		
UNITED STATES	1,961	2,136	780	1,175	1,135	1,188	1,050	1,870		
NEW ENGLAND Maine N.H. Vt. Mass. R.I. Conn.	45 2 1 30 5 6	46 1 2 1 34 4 4	37 1 27 4 5	54 5 33 6 7	11 - - 9 1 1	12 - 1 7 1 3	32 - 1 - 25 2 4	42 1 - 12 15 14		
MID. ATLANTIC Upstate N.Y. N.Y. City N.J. Pa.	103 58 32 13	162 34 55 45 28	54 3 50 1	103 18 44 41	11 6 2 3	44 6 18 13 7	202 14 123 59 6	296 14 149 74 59		
E.N. CENTRAL Ohio Ind. III. Mich. Wis.	312 20 49 94 141 8	400 141 15 148 44 52	104 4 9 - 88 3	179 13 8 142 3 13	174 10 81 48 23 12	170 18 40 79 26 7	126 19 6 89 6 6	182 55 14 78 25 10		
W.N. CENTRAL Minn. Iowa Mo. N. Dak. S. Dak. Nebr.	134 35 19 62 1 12	106 14 2 70 - - 8	71 32 14 18 - - 4	91 19 3 60 1 - 4	15 2 6 5 - 1	51 2 1 44 - 1	64 24 7 27 3 2	59 31 - 22 - 2 1		
Kans. S. ATLANTIC Del. Md. D.C. Va. W. Va. N.C. S.C. Ga. Fla.	5 159 - 12 1 13 3 15 97	12 303 5 18 11 13 3 45 18 33 157	3 41 - 4 U 12 1 5 1 3 15	4 62 1 5 U 5 1 11 6 12 21	1 268 1 14 20 1 84 11 36 50	3 450 1 90 33 31 1 113 41 76 64	1 156 - 22 - 7 26 18 86 27	3 238 4 32 8 17 7 45 64 57 4		
E.S. CENTRAL Ky. Tenn. Ala. Miss.	89 20 44 7 18	274 22 204 28 20	52 10 39 1 2	165 19 137 9	134 8 88 21 17	213 23 97 59 34	70 - 21 49 -	113 10 40 53 10		
W.S. CENTRAL Ark. La. Okla. Tex.	164 38 18 9 99	332 27 23 79 203	173 - 23 4 146	400 19 22 18 341	442 9 351 31 51	171 19 18 46 88	17 12 5 -	328 14 U 12 302		
MOUNTAIN Mont. Idaho Wyo. Colo. N. Mex. Ariz. Utah Nev.	183 22 1 24 22 70 5 39	138 3 2 30 13 72 10 6	46 - - 12 12 17 5 -	71 1 20 6 31 10 2	27 - - 3 3 19 - 2	30 - - - 30 -	53 - 6 12 15 5 15	48 - - U 7 17 10 14		
PACIFIC Wash. Oreg. Calif. Alaska Hawaii	772 141 70 550 2 9	375 10 8 345 - 12	202 162 35 - 5	50 27 11 - 12	53 8 1 44 -	47 5 1 40 - 1	330 33 282 3 12	564 22 17 491 6 28		
Guam P.R. V.I. Amer. Samoa C.N.M.I.	- 1 - -	2 6 U U U	U U U U	U U U U	20	43 U U U		- U U U		

TABLE II. (Cont'd) Provisional cases of selected notifiable diseases, United States, weeks ending March 4, 2000, and March 5, 1999 (9th Week)

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

*Individual cases may be reported through both the National Electronic Telecommunications System for Surveillance (NETSS) and the Public Health Laboratory Information System (PHLIS).

⁺Cumulative reports of provisional tuberculosis cases for 1999 are unavailable ("U") for some areas using the Tuberculosis Information System (TIMS).

H. ir		l. influenzae,		Hepatitis (Viral), by type					Measles (Rubeola)					
	inva	sive	A		В		Indige	nous	Impo	rted*	Tota	l		
Reporting Area	Cum. 2000†	Cum. 1999	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999	2000	Cum. 2000	2000	Cum. 2000	Cum. 2000	Cum. 1999		
UNITED STATES	176	221	1,849	2,933	677	894	-	3	-	-	3	20		
NEW ENGLAND	12	16	32	36	6	25	-	-	-	-	-	1		
Maine N.H.	2	1 2	1 7	2 4	1 3	2	-	-	-	-	-	- 1		
Vt.	2	3	2	-	2	-	-	-	-	-	-	-		
R.I.	8	9	9	14	-	2	-	-	-	-		-		
Conn.	-	1	13	16	-	10	-	-	-	-	-	-		
MID. ATLANTIC	23	35	84	192	65	138	-	-	-	-	-	-		
N.Y. City	5	9	38	43 59	54	23 40	-	-	-	-	-	-		
N.J. Pa	4	11 1	-	27 63	-	21 54	-	-	-	-	-	-		
E N CENTRAL	19	31	214	696	85	99 90	_	3	_	_	3	_		
Ohio	11	13	73	123	21	20		2	-	-	2	-		
Ind.	3	1 15	3 41	12 145	1	4	-	-	-	-	-	-		
Mich.	3	2	92	402	63	59	-	1	-	-	1	-		
Wis.	-	-	5	14	-	6	-	-	-	-	-	-		
W.N. CENTRAL Minn.	6	16 4	215 18	159 4	42	50 3	-	-	-	-	-	-		
lowa	-	3	21	20	7	8	-	-	-	-	-	-		
N. Dak.	2	- 3	-	101	- 22	26	-	-	-	-		-		
S. Dak.	-	1	-	- 16	1	-	-	-	-	-	-	-		
Kans.	2	4	49	18	8	5	-	-	-	-		-		
S. ATLANTIC	49	43	187	216	121	126	-	-	-	-	-	-		
Del. Md	- 18	- 19	- 24	- 73	21	- 38	-	-	-	-	-	-		
D.C.	-	-	-	11	-	4	-	-	-	-	-	-		
va. W. Va.	11	2	33 19	14	- 25	8		-	-	-	-	-		
N.C.	3	5	49	25	45	39	-	-	-	-	-	-		
Ga.	14	10	18	69	2	15	-	-	-	-	-	-		
Fla.	1	4	41	22	27	6	U	-	U	-	-	-		
E.S. CENTRAL	8	15	68 2	83 15	45	82	-	-	-	-	-	-		
Tenn.	3	5	21	35	28	42	-	-	-	-	-	-		
Ala. Miss.	2	5 2	14 31	21 12	5 10	17 17	-	-	1	-		-		
W.S. CENTRAL	13	17	282	429	35	91	-	-	-			2		
Ark.	-	-	30	6	8	10	-	-	-	-	-	-		
La. Okla.	11	ь 9	8 59	103	17	34 17	-	-	-	-	-	-		
Tex.	-	2	185	289	-	30	-	-	-	-	-	2		
MOUNTAIN	26	27	130	304	60	79 1	-	-	-	-	-	-		
Idaho	1	1	6	8	3	4		-	-	-	-	-		
Wyo. Colo	- 9	1	2 36	1	- 18	- 15	-	-	-	-	-	-		
N. Mex.	8	6	17	5	13	25	-	-	-	-	-	-		
Arız. Utah	1	14	50 9	183	19	1/		-	-	-	-	-		
Nev.	-	-	9	29	3	10	-	-	-	-	-	-		
PACIFIC	20	21	637	818	218	214	-	-	-	-	-	17		
Vvasn. Oreg.	2 4	8	29 37	49 45	13	13	-	-	-	-	-	2		
Calif.	4	12	568	721	196	192	-	-	-	-	-	7		
Hawaii	9	-	-	1	1	4 3	-	-	-	-	-	-		
Guam	-	-	-	2	-	2	-	-	-	-	-	-		
P.R. VI	-	ū	15	12 U	8	18 U	Ū	-	ū	-	-	Ū		
Amer. Samoa	-	Ŭ	-	Ŭ	-	Ŭ	Ŭ	-	Ŭ	-	-	Ŭ		
C.IN.IVI.I.	-	U	-	U	-	U	U	-	U	-	-	U		

TABLE III. Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending March 4, 2000, and March 5, 1999 (9th Week)

N: Not notifiable U: Unavailable - : no reported cases *For imported measles, cases include only those resulting from importation from other countries. *Of 44 cases among children aged <5 years, serotype was reported for 18 and of those, 3 were type b.

	Mening Dis	jococcal ease	Mumps			Pertussis			Rubella			
Reporting Area	Cum. 2000	Cum. 1999	2000	Cum. 2000	Cum. 1999	2000	Cum. 2000	Cum. 1999	2000	Cum. 2000	Cum. 1999	
UNITED STATES	410	470	3	64	71	73	597	789	-	5	3	
NEW ENGLAND Maine	25 2	25 3	-	-	3	9	135 7	100	-	1	1 -	
N.H. Vt	- 1	3	-	-	1	5 1	34 42	17 9	-	1	-	
Mass.	16	16	-	-	2	-	44	72	-	-	1	
K.I. Conn.	- 6	-	-	-	-	2	6	2	-	-	-	
MID. ATLANTIC	32	52	-	3	9	19	53	90	-	-	-	
Upstate N.Y.	8	8	-	1	2	8	32	55	-	-	-	
N.Y. City N.J.	8	20 13	-	-	-	-	-	2	-	-	-	
Pa.	8	11	-	2	4	11	21	23	-	-	-	
E.N. CENTRAL	49 12	72	1	6	7	9	122	94	-	-	-	
Ind.	13	20 6	-	-	2 -	2	108	50 4	-	-	-	
III. Miab	4	26	-	1	2	-	5	10 12	-	-	-	
Wis.	4	6	-	-	-	-	-	12	-	-	-	
W.N. CENTRAL	43	58	-	10	2	1	21	28	-	3	-	
Minn.	1	11	-	- 2	- 2	-	7	-	-	-	-	
Mo.	29	21	-	1	-	-	2	6	-	-	-	
N. Dak.	1	-	-	-	-	1	1	-	-	-	-	
Nebr.	1	3	-	4	-	-	-	1	-	÷	-	
Kans.	1	10	-	2	-	-	4	15	-	3	-	
S. AILANTIC Del.	75	60 1	1	8	9	5 1	43 1	50	-	-	-	
Md.	5	12	1	2	2	-	13	21	-	-	-	
Va.	12	5	-	- 1	2	2	3	7	-	-	-	
W.Va.	1 14	1	-	- 2	- 1	-	- 15	- 18	-	-	-	
S.C.	6	11	-	3	2	÷	9	4	-	-	-	
Ga. Fla.	17 20	14 7	Ū	-	- 1	2 U	2	-	Ū	-	-	
E S CENTRAL	19	39		1	1	-	12	20	-	-	-	
Ky.	4	8	-	-	-	-	7	4	-	-	-	
Ienn. Ala.	7	13	-	- 1	- 1	-	1	9	-	-	-	
Miss.	1	7	-	-	-	-	-	1	-	-	-	
W.S. CENTRAL	21	44	-	-	12	-	3	25	-	-	2	
Агк. La.	12	23	-	-	2	-	- 3	2	-	-	-	
Okla.	7	11	-	-	1 9	-	-	3 18	-	-	- 2	
MOUNTAIN	22	/9	_	3	5	16	153	160	_	1	-	
Mont.	-		-	-	-	-	1		-	-	-	
Idaho Wyo.	2	6 2	-	-	-	1	24	71	-	-	-	
Colo.	7	14	-	-	2	13	82	27	-	-	-	
Ariz.	4	7 15	-	-	- N	2	27 14	34	-	-	-	
Utah	3	3	-	-	2	-	4	18	-	1	-	
Nev.	-	Z 71	-	2	20	-		2	-	-	-	
Wash.	6	10	1	33 1	- 23	6	55 19	222	-	-	-	
Oreg.	13	17 27	N	N 21	N 19	-7	13 20	3 199	-	-	-	
Alaska	102	3	-	-	1	-	20	100	-	-	-	
Hawaii	2	4	-	1	4	1	1	8	-	-	-	
Guam PB	-	- 2	-	-	1	-	-	-	-	-	-	
V.I.	-	ບໍ່	U	-	U	U	-	U	U	-	U	
C.N.M.I.	-	U	U	-	U	U	-	U U	U	-	U	

TABLE III. (Cont'd) Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending March 4, 2000, and March 5, 1999 (9th Week)

N: Not notifiable

U: Unavailable

- : no reported cases

		All Cau	ises, By	Age (Ye	ears)		P&I [†]			All Cau	ises, By	/ Age (Y	ears)		P&I⁺
Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	Total	Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	Total
NEW ENGLAND Boston, Mass. Bridgeport, Conn. Cambridge, Mass. Hartford, Conn. Lowell, Mass. Lynn, Mass. New Bedford, Ma New Haven, Conn Providence, R.I. Somerville, Mass. Springfield, Mass	562 154 23 18 27 U 31 20 31 20 55 41 5 45 70 4 20 42	412 110 14 13 23 24 18 30 37 53 37 53 25	98 29 6 3 4 U 2 1 7 6 13 - 10	34 8 3 2 - U 5 1 3 - 2 1 5 2	11 5 - - U - 1 - 1 - 2	7 2 - - U - 2 1 -	835 4 7 6 U 2 3 2 4 2 - 8 8	S. ATLANTIC Atilanta, Ga. Baltimore, Md. Charlotte, N.C. Jacksonville, Fla Miami, Fla. Norfolk, Va. Richmond, Va. Savannah, Ga. St. Petersburg, I Tampa, Fla. Washington, D.e	1,468 U 276 125 . 149 104 67 78 Fla. 58 C. 328 I. U	997 U 190 78 106 82 52 41 57 45 161 185 U	280 U 50 24 32 17 12 16 13 8 29 79 U	120 U 27 14 6 5 6 2 5 4 12 39 U	33 U 6 6 3 - 1 3 - 4 10 U	34 U 3 2 - 3 3 3 1 3 1 3 U 0	126 U 39 14 14 9 5 7 4 3 24 7 U
Warcester, Mass. MID. ATLANTIC Albany, N.Y. Allentown, Pa. Buffalo, N.Y. Camden, N.J. Elizabeth, N.J. Erie, Pa.S	32 55 2,414 46 U 100 41 20 46	24 38 1,687 32 U 74 25 20 35	469 9 U 15 9 10	2 177 4 U 8 2 - 1	2 39 1 U 1 -	2 37 U 2 5	° 12 126 2 U 9 2 - 2	E.S. CENTRAL Birmingham, Al Chattanooga, Te Knoxville, Tenn. Lexington, Ky. Memphis, Tenn Mobile, Ala. Montgomery, A Nashville, Tenn.	966 a. 203 ann. 80 86 64 . 200 79 Ia. 64 190	695 147 64 65 45 136 61 47 130	175 37 11 14 13 40 12 8 40	62 16 5 4 1 13 5 5 13	19 2 2 4 1 4 4	15 1 3 7 - 3	106 29 14 6 2 22 5 7 21
Jersey City, N.J. New York City, N.J. Newark, N.J. Paterson, N.J. Philadelphia, Pa. Reading, Pa. Rochester, N.Y. Schenectady, N.Y. Scranton, Pa.§ Syracuse, N.Y. Trenton, N.J. Utica, N.Y. Yonkers, N.Y.	39 39 1, 1,227 U 19 459 55 29 131 23 25 131 23 25 111 25 111 25 111 25 111 25 111 21 21 21 21 21 21 21 21 2	24 854 U 9 290 40 23 98 11 21 90 90 19 16 U	10 222 U 8 121 9 4 25 5 3 14 4 1 U	3 102 2 36 5 2 5 1 1 3 2 - U	1 25 U - 9 - 1 - - 1 U	1 19 U 3 1 - 2 - 4 - U	31 U 2 20 3 6 6 5 1 1 1 6 5 1 U	W.S. CENTRAL Austin, Tex. Baton Rouge, La Corpus Christi, Dallas, Tex. El Paso, Tex. Ft. Worth, Tex. Houston, Tex. Little Rock, Ark. New Orleans, La San Antonio, Te Shreveport, La. Tulsa, Okla.	1,536 62 1. 43 Tex. 68 228 U 126 455 84 . U x. 269 58 143	1,017 45 19 47 148 U 80 292 51 U 196 41 98	311 12 13 9 49 U 27 86 21 U 57 9 28	123 2 7 4 18 U 9 53 7 U 10 3 10	44 32 45 U 4 14 3 U 4 32	41 248 U6102 225	112 1 2 6 8 U 6 38 3 U 33 7 8
E.N. CENTRAL Akron, Ohio Canton, Ohio Chicago, Ill. Cincinnati, Ohio Cleveland, Ohio Columbus, Ohio Dayton, Ohio Detroit, Mich. Evansville, Ind.	2,205 49 37 485 89 154 215 145 187 58	1,503 37 302 61 91 146 112 109 46	453 9 5 112 14 40 44 25 54 10	147 2 1 42 9 12 14 5 16 2	49 1 15 4 7 6 1 5	51 - 12 1 4 5 2 3 -	225 7 3 83 8 6 18 10 16 5	MOUNTAIN Albuquerque, N Boise, Idaho Colo. Springs, C Denver, Colo. Las Vegas, Nev. Ogden, Utah Phoenix, Ariz. Pueblo, Colo. Salt Lake City, U Tuceon Ariz.	1,006 I.M. 97 31 Colo. 48 126 195 20 195 32 Itah 84	736 80 20 40 89 138 17 135 26 61 130	161 11 4 18 41 32 5 11 32	64 4 3 1 12 12 - 15 1 6 10	25 1 4 1 3 4 - 7 - 1	20 1 2 4 - 6 5 2	91 13 2 3 18 14 - 13 2 10
Fort Wayne, Ind. Gary, Ind. Grand Rapids, Mid Lansing, Mich. Milwaukee, Wis. Peoria, III. Rockford, III. South Bend, Ind. Toledo, Ohio Youngstown, Ohi	70 21 205 46 132 46 69 95 0 U	53 11 43 142 30 86 43 52 41 68 U	9 8 5 40 13 2 2 10 5 16 U	6 1 2 9 2 6 1 7 2 8 U	- - - 3 - - 1 U	2 1 3 10 1 5 - - 2 U	91819793526U	PACIFIC Berkeley, Calif. Fresno, Calif. Glendale, Calif. Honolulu, Hawa Long Beach, Cal Los Angeles, Ca Pasadena, Calif. Portland, Oreg. Sacramento, Ca	2,251 19 169 38 ii 91 if. 79 lif. 796 170 lif. 163	1,633 15 127 28 69 51 577 20 128 111	32 402 27 8 14 17 137 4 29 34	124 1 8 2 3 8 52 - 10 8	4 51 7 1 2 18 1 2 5	2 39 1 - 4 12 12 1 5	216 23 1 7 12 57 6 11 20
W.N. CENTRAL Des Moines, Iowa Duluth, Minn. Kansas City, Kans. Kansas City, Mo. Lincoln, Nebr. Minneapolis, Min Omaha. Nebr.	864 101 55 . 35 93 32 n. 173 98	600 70 39 22 67 24 132 65	162 21 9 6 16 3 29 20	59 7 4 2 7 4 5 10	22 1 3 4 1 5 2	21 2 1 2 1 2 1 2 1	79 13 3 13 6 19 5	San Diego, Calif San Francisco, C San Jose, Calif. Santa Cruz, Calif Seattle, Wash. Spokane, Wash. Tacoma, Wash.	. 201 Calif. U 189 f. 31 132 . 45 102	137 U 135 27 89 36 83	37 U 39 3 30 9 12	6 U 9 1 9 7	9 U 5 - 1 - -	10 U 1 - 3 -	22 U 23 15 5 12
St. Louis, Mo. St. Paul, Minn. Wichita, Kans.	104 60 113	60 50 71	29 6 23	9 1 10	2	4 3 5	10 7	IUIAL	13,2721	9,280	2,511	910	293	265	1,164

TABLE IV. Deaths in 122 U.S. cities,* week ending March 4, 2000 (9th Week)

U: Unavailable -: no reported cases

U: Unavailable --: ho 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 Jose Aponte Paul Gangarosa, M.P.H. Gerald Jones David Nitschke Carol A. Worsham

CDC Operations Team Carol M. Knowles Deborah A. Adams Willie J. Anderson Patsy A. Hall Kathryn Snavely Sara Zywicki

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*. 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.

(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; eitation as to source however, is appropriated.

citation as to source, however, is appreciated.

Director, Centers for Disease	Acting Director,	Writers-Editors,				
Control and Prevention	Epidemiology Program Office	<i>MMWR</i> (weekly)				
Jeffrey P. Koplan, M.D., M.P.H.	Barbara R. Holloway, M.P.H.	Jill Crane				
Acting Deputy Director for Science	Editor, <i>MMWR</i> Series	David C. Johnson				
and Public Health, Centers for	John W. Ward, M.D.	Teresa F. Rutledge				
Disease Control and Prevention Lynne S. Wilcox, M.D., M.P.H.	Acting Managing Editor, <i>MMWR</i> (weeklγ) Caran R. Wilbanks	Desktop Publishing Lynda G. Cupell Morie M. Higgins Cheryle R. Reynolds				
\$11 S. Government Printing Office: 2000-533-206/08057 Begion IV						

Government Printing Office: eyn