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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.

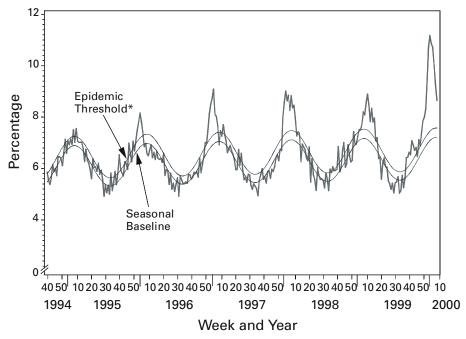
¹The epidemic threshold is 1.645 standard deviations above the seasonal baseline. The expected seasonal baseline is projected using a robust regression procedure in which a periodic regression model is applied to observed percentages of deaths from P&I since 1983.

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

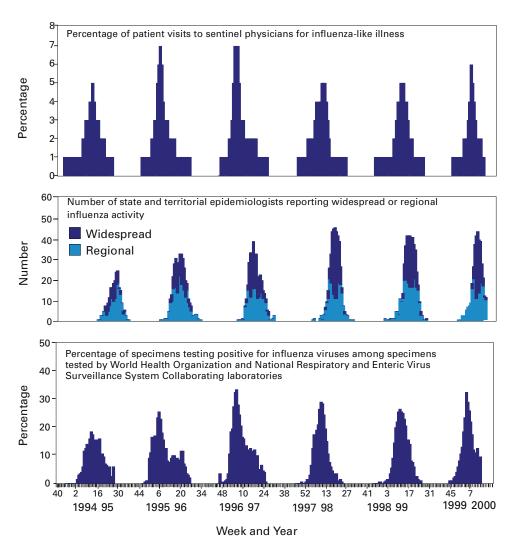


FIGURE 2. Results of three influenza surveillance systems*, by week and year — United States, 1994–2000

^{*}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.

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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.

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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.

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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."

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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	ntiretroviral drugs with the antimycobacterial drugs rifabutin 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.	Readers — 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.	Continued
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.

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Notice to Readers — Continued

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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			De	evice-as	sociate	d infect	tion rate	es
	No.	days patient	Device days*			Percentiles				
ICU/Type of infection	units	in ICU		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.

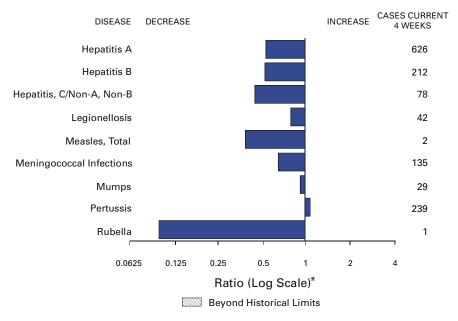
⁺ Device utilization ratio (device days divided by total number of days patient was in ICU).

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		-	HIV infection, pediatric*§	34
Brucellosis*		3	Plaque	2
Cholera		_	Poliomyelitis, paralytic	-
Congenital ru	bella syndrome	1	Psittacosis*	1
Cyclosporiasi		2	Rabies, human	-
Diphtheria		-	Rocky Mountain spotted fever (RMSF)	22
Encephalitis:	California* serogroup viral	1	Streptococcal disease, invasive Group A	486
•	eastern equine*	-	Streptococcal toxic-shock syndrome*	20
	St. Louis*	-	Syphilis, congenital ¹	-
	western equine*	-	Tetanus	2
Ehrlichiosis	human granulocytic (HGE)*	12	Toxic-shock syndrome	24
	human monocytic (HME)*	1	Trichinosis	1
Hansen Disea	se*	6	Typhoid fever	46
Hantavirus pu	Ilmonary syndrome*†.	-	Yellow fever	-
Hemolytic ure	emic syndrome, post-diarrheal*	8		

-: 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.

	week	s enumų	j warch	4, 2000	, and w	arch 5,	1999 (91	IWEEK	1	
								Escherichia	<i>coli</i> O157:H7*	
	AIE		Chlan	<u></u>		oridiosis	NET		PHLIS	
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	511	352	3,680	3,616	6	10	16	29	15	28
Maine N.H.	6 5	5 13	221 133	124 181	1	1 1	1 3	1 1	1 3	- 1
Vt.	1	4	88	85	4	1	1	1	2	-
Mass. R.I.	370 17	238 20	1,506 370	1,535 375	- 1	6	5	17	3	14 1
Conn.	112	72	1,362	1,316	-	1	6	9	6	12
MID. ATLANTIC Upstate N.Y.	1,592 65	1,492 76	890 N	13,399 N	15 8	43 17	23 23	11 8	-	2
N.Y. City	986	835	-	6.540	4	21	-	1	-	1
N.J. Pa.	387 154	370 211	516 374	2,139 4,720	- 3	1 4	N	2 N	-	1
E.N. CENTRAL	590	489	14.000	18,105	17	45	22	35	6	22
Ohio	92 56	97 52	3,184 2.044	5,992 1,897	11	6	8	20	3	7
Ind. III.	353	231	3,701	4,633	3	6	2 8	6 4	1 -	7 3
Mich. Wis.	67 22	81 28	3,759 1,312	3,494 2,089	3	5 25	4 N	5 N	1 1	23
W.N. CENTRAL	151	161	4,115	7,151	8	19	56	34	26	27
Minn. Iowa	32 10	28 13	996 547	1,354 342	- 1	10 1	10 9	10 5	10 4	11 2
Mo. N. Dak.	70	84 3	902	3,266 161	3 1	4	30 2	2 2	8 1	2 1
S. Dak.	2	3	298	370	1	1	-	-	-	-
Nebr. Kans.	7 30	10 20	465 907	666 992	2	1 2	2 3	3 12	2 1	11
S. ATLANTIC	1,531	1,832	14,181	24,118	22	30	20	17	13	10
Del. Md.	26 153	31 252	500 971	524 2,289	- 1	4	5	1 1	1	-
D.C. Va.	112 115	69 102	507 2.001	N 2,484	-	3	- 4	- 5	U 4	U 2
W. Va.	6	14	219	411	-	-	1	-	1	1
N.C. S.C.	75 156	125 128	3,187 669	3,953 4,376	3	1	6 -	3 1	1	3 1
Ga. Fla.	183 705	207 904	2,523 3,604	4,878 5,203	12 6	21 1	2 2	1 5	3 3	U 3
E.S. CENTRAL	281	300	7,180	7,267	6	2	10	15	4	5
Ky. Tenn.	37 105	37 130	1,455 1,809	1,251 2,463	-	1 1	4 5	5 6	U 4	U 2
Ala. Miss.	92 47	69 64	2,169 1,747	2,501 1,052	6	-	1	2 2	-	2
W.S. CENTRAL	542	980	12,517	14,427	5	15	8	7	11	10
Ark. La.	20 92	34 67	554 2.232	862 1,436	1	- 12	2	2 3	1 6	2 2
Okla. Tex.	16 414	19 860	1,265 8,466	1,488 10,641	1 3	1	3 3	1 1	3 1	- 6
MOUNTAIN	213	207	3,400	5,930	9	22	22	12	4	10
Mont. Idaho	3	3 5	64	208 326	- 1	1 2	5 3	-	-	2
Wyo.	1	-	133	135	1	-	2	1	-	1
Colo. N. Mex.	52 26	56 9	602 334	1,230 903	1 1	2 10	7	3 1	1	1
Ariz. Utah	56 28	86 27	1,407 387	2,260 318	2 3	7 N	3 1	3 4	2 1	1 4
Nev.	28 44	21	543	550	-	-	1	-	-	1
PACIFIC Wash.	877 102	1,132 58	14,346 2,247	19,147 2,133	63 N	49 N	32 3	24 1	9	25 9
Oreg.	22	32	454	954	1	3	3	10	3 3	8
Calif. Alaska	727	1,021 5	11,402 243	15,195 317	62	46	23	13	-	8
Hawaii	26	16		548	-	-	3	-	3	-
Guam P.R.	9 153	1 215	- 142	85 U	-	-	N	N 1	U U	U U
V.I. Amer. Samoa	6	3	-	Ŭ	-	U U	-	U U	Ŭ U	Ŭ U
C.N.M.I.	-	-	-	U U		U	-	U	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	rhea	Hep C/N	atitis A,NB	Legio	nellosis		rme ease
Reporting Area	Cum.	Cum. 1999	Cum.	Cum. 1999	Cum.	Cum. 1999	Cum.	Cum. 1999
UNITED STATES	2000 47,528	62,629	2000 301	590	2000 92	153	2000 476	746
NEW ENGLAND	1,132	1,301	-	2	5	11	60	153
Maine N.H.	12 13	9 15	-	-	2 1	2 1	- 15	1
/t.	4	11	-	1	- 1	3 2	-	- 70
Mass. R.I.	431 87	514 98	-	1	-	1	38	72
Conn.	585	654	-	-	1	2	7	80
MID. ATLANTIC Upstate N.Y.	1,212 648	7,561 806	3 3	21 11	15 6	40 8	335 115	429 85
N.Y. City	-	3,216	-	-	-	7	2	15
N.J. Pa.	302 262	1,280 2,259	-	10	9	5 20	218	100 229
E.N. CENTRAL	8,749	10,859	48	316	27	47	2	25
Ohio Ind.	1,837 931	2,881 1,173	-	-	16 3	14 1	2	8
11.	2,179 2,987	3,343	3 45	6 91	1 6	10	-	1 1
Mich. Wis.	2,987 815	2,519 943	40	219	1	13 9	Ū	15
W.N. CENTRAL	1,455	3,499	43	39	4	4	13	9
Minn. Iowa	404 121	520 144	-	-	1 1	2	2	1 2
Mo.	367	2,112	38	34	2	1	3	2
N. Dak. S. Dak.	45	9 30	-	-	-	-	-	1
Nebr. Kans.	141 377	301 383	1 4	1 4	-	1	- 8	- 3
S. ATLANTIC	10,795	19,133	11	36	21	18	48	91
Del. Md.	260 455	302 2.839	2	18	1 6	2	1 37	4 74
D.C.	427	1,310	-	-	-	-	-	1
Va. W.Va.	1,446 50	2,009 115	- 1	6 2	3 N	2 N	1 3	- 1
N.C. S.C.	3,097 574	3,540 2,150	5	8 1	2 2	4 4	4	11
Ga.	1,810	3,171	-	1	-	-	-	-
Fla.	2,676	3,697	3	-	7	4 9	2	-
E.S. CENTRAL Ky.	5,277 611	6,121 667	51 5	41 5	2	5	-	11 -
Tenn. Ala.	1,469 1,817	2,016 2,267	15 3	22 1	1 1	4	-	3 5
Miss.	1,380	1,171	28	13	-	-	-	3
W.S. CENTRAL	14,591	8,163	68	62	-	1	-	-
Ark. La.	319 9,531	408 1,453	1 31	2 47	-	- 1	-	-
Okla. Tex.	594 4,147	785 5,517	- 36	1 12	-	-	-	-
MOUNTAIN	1,420	1.706	46	46	8	12	1	1
Mont. daho	4	3 23	-	4	- 1	-	-	-
Nyo.	12	6	31	17	-	-	-	-
Colo. N. Mex.	648 62	372 180	7 4	4	4	1	-	- 1
Ariz.	440	853	4	10	-	1	1	-
Utah Nev.	52 202	34 235	-	1	3	5 4	-	-
PACIFIC	2,897	4,286	31	27	10	11	17	27
Wash. Dreg.	411 56	367 141	3 7	2 3	2 N	2 N	- 1	- 1
Calif.	2,401	3,620 62	21	22	8	9	16	26
Alaska Hawaii	29	62 96	-	-	-	-	Ň	Ň
Guam	-	15	-	-	-	-	- NI	-
P.R. /.I.	30	54 U	1 -	Ū	-	Ū	N -	N U
Amer. Samoa C.N.M.I.	-	U U	-	U U	-	U U	-	U U
N: Not notifiable	U: Unav	-	- : no report	-		0		Ŭ

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

	weeks e	ending M	arch 4, 20	000, and N	larch 5, 19	999 (9th W	eek)	
						Salmon	ellosis*	
	Mala			s, Animal		TSS		LIS
Reporting Area	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999
UNITED STATES	111	201	581	794	3,255	4,188	1,814	3,848
NEW ENGLAND	-	3	68	114	210	226	202	240
Maine N.H.	-	-	16 2	19 5	25 14	23 3	9 8	13 11
Vt.	1	-	4	18	5	10	3	10
Mass. R.I.	-	3	25	33 10	125 4	133 10	132 12	129 21
Conn.	-	-	21	29	37	47	38	56
MID. ATLANTIC	12	62	126	170	291	647	193	478
Upstate N.Y. N.Y. City	7 2	12 30	100 U	108 U	77 114	115 208	24 169	144 195
N.J.	-	15	14	37	-	161	-	135
Pa. E.N. CENTRAL	3 7	5 23	12 2	25	100 430	163 654	235	4 563
Ohio	2	2	2	1 -	140	148	70	114
Ind. III.	2	4 9	-	-	45 134	34 198	44	40 205
Mich.	3	5	-	1	71	162	88	143
Wis.	-	3	-	-	40	112	33	61
W.N. CENTRAL Minn.	4 2	7	53 18	114 15	197 42	228 53	142 42	257 92
lowa	-	2	7	16	17	30	11	28
Mo. N. Dak.	-	5	2 8	5 15	65 2	57 1	44 10	73 11
S. Dak. Nebr.	- 1	-	7	29 1	7 26	7 18	11 7	13 17
Kans.	i	-	11	33	38	62	17	23
S. ATLANTIC	34	47	251	277	564	741	367	695
Del. Md.	- 18	- 18	10 47	3 73	8 103	15 91	7 73	13 84
D.C. Va.	11	5	67	61	66	16 84	Ŭ 50	U 90
W. Va.	-	1	18	15	20	13	12	90 19
N.C. S.C.	4	3	52 14	62 11	132 55	170 38	67 41	141 53
Ga.	-	5	28	28	67	147	117	205
Fla.	1	8	15	24	113	167	-	90
E.S. CENTRAL Ky.	4 1	5 1	23 4	39 12	163 19	264 57	89 16	166 36
lenn.	- 3	2 2	16	18 9	40 70	75	47	75 47
Ala. Miss.	-	2 -	3	-	70 34	77 55	23 3	47 8
W.S. CENTRAL	1	9	8	14	192	297	238	396
Ark. La.	- 1	1 6	-	-	31 24	41 46	22 68	35 59
Okla.	-	1	8	14	23	32	18	14
Tex.	-	1	-	-	114	178	130	288
MOUNTAIN Mont.	8	10 1	27 9	20 8	296 11	294 3	210	285 1
ldaho Wyo.	-	1	14	5	21 6	10 2	-	15 5
Colo.	4	3	-	5 1	59	84	- 58	84
N. Mex. Ariz.	2	1 3	1 3	- 6	30 90	31 100	21 93	39 82
Utah	2	1	-	-	49	34	38	38
Nev.	-	-	-	-	30	30	-	21
PACIFIC Wash.	41 2	35 2	23	45	912 32	837 34	138 59	768 109
Oreg. Calif.	4 35	6 24	- 17	- 42	42 787	65 678	49	86 514
Alaska	-	-	6	3	12	6	2	4
Hawaii	-	3	-	-	39	54	28	55
Guam P.R.	-	-	- 6	-7	- 10	13 57	U U	U U
V.I.	-	Ŭ	-	U	-	U	Ŭ	Ŭ
Amer. Samoa C.N.M.I.	-	U U	-	U U	-	U U	U U	U U
N. Not notifiable	U·Unav	-	- no repor	-		-	-	-

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).

	WEEKS	Shigel		<i>, anu i</i>	1	ohilis	CCK/	
F	NET			HLIS		Secondary)	Tuber	culosis
Reporting Area	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999	Cum. 2000	Cum. 1999 [†]
UNITED STATES	1,961	2,136	780	1,175	1,135	1,188	1,050	1,870
NEW ENGLAND Maine N.H. Vt.	45 2 1 1	46 1 2 1	37 - 1	54 5 3	11 - -	12 - - 1	32 - 1	42 1 - -
Mass. R.I. Conn.	30 5 6	34 4 4	27 4 5	33 6 7	9 1 1	7 1 3	25 2 4	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.	134 35 19 62 - 1	106 14 2 70	71 32 14 18	91 19 3 60 1	15 2 6 5	51 2 1 44	64 24 7 27 -	59 31 - 22 - 2
S. Dak. Nebr. Kans.	12 5	8 12	4 3	4	- 1 1	- 1 3	3 2 1	2 1 3
S. ATLANTIC Del. Md. D.C. Va. W. Va. N.C.	159 - 18 - 12 1 13	303 5 18 11 13 3 45	41 - U 12 1 5	62 1 5 U 5 1 11	268 1 51 14 20 1 84	450 1 90 33 31 1 113	156 - 22 - - 7 26	238 4 32 8 17 7 45
S.C. Ga. Fla.	3 15 97	18 33 157	1 3 15	6 12 21	11 36 50	41 76 64	18 56 27	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.	183 22 1 24	138 3 2 2 30	46 - - 12	71 - 1 20	27 - - 3	30 - - - -	53 - - 6	48 - - - U
N. Mex. Ariz. Utah Nev.	22 70 5 39	13 72 10 6	12 17 5	6 31 10 2	3 19 - 2	30	12 15 5 15	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 U	20	43 U U U	- - - -	- - U U U
N: Not notifiable	U: Unav	ailable	-: no repo	rted cases				

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

*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).

					1 5, 199		vveei	()				
	H. influ			epatitis (V	iral), by typ	е				les (Rubec		
	inva		A		В	-	Indiger		Impo		Total	
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	-	-	-	-	-	-	-
Mass. R.I.	8	9	9	14	-	11 2	-	-	-	-	-	-
Conn.	-	1	13	16	-	10	-	-	-	-	-	-
MID. ATLANTIC	23	35	84	192	65	138 23	-	-	-	-	-	-
Upstate N.Y. N.Y. City	13 5	14 9	46 38	43 59	11 54	40	-	-	-	-	-	
N.J. Pa.	4 1	11 1	-	27 63	-	21 54	-	-	-	-	-	-
E.N. CENTRAL	19	31	214	696	85	89		3			3	
Ohio	11	13	73	123	21	20	-	2	-	-	2	
Ind. III.	3 2	1 15	3 41	12 145	1	4	-	-	-	-	-	1
Mich. Wis.	3	2	92 5	402 14	63	59	-	1	-	-	1	-
WIS. W.N. CENTRAL	6	- 16	5 215	14	- 42	6 50	-	-	-	-	-	-
Minn.	-	4	18	4	-	3	-	-	-	-	-	-
lowa Mo.	2	3 3	21 119	20 101	7 22	8 26	-	-	-	-	-	-
N. Dak.	1	-	-	-	-	-	-	-	-	-	-	-
S. Dak. Nebr.	- 1	1 1	- 8	16	1 4	- 8	-	-	-	-	-	-
Kans.	2	4	49	18	8	5	-	-	-	-	-	-
S. ATLANTIC Del.	49	43	187	216	121	126	-	-	-	-	-	-
Md.	18	19	24	73	21	38	-	-	-	-	-	-
D.C. Va.	- 11	2	33	11 14	- 25	4 8	-	-	-	-	-	
W. Va.	1	1	19	1	-	-	-	-	-	-	-	-
N.C. S.C.	3 1	5 2	49 3	25 1	45 1	39 16	-	-	-	-	-	
Ga. Fla.	14 1	10 4	18 41	69 22	2 27	15 6	Ū	-	Ū	-	-	-
E.S. CENTRAL	8	15	68	83	45	82		_			_	
Ky.	3	3	2	15	2	6	-	-	-	-	-	-
Tenn. Ala.	3 2	5 5	21 14	35 21	28 5	42 17	-	-	-	-	-	-
Miss.	-	2	31	12	10	17	-	-	-	-	-	-
W.S. CENTRAL Ark.	13	17	282 30	429 6	35 8	91 10	-	-	-	-	-	2
La.	2	6	8	31	17	34	-	-	-	-	-	
Okla. Tex.	11	9 2	59 185	103 289	10	17 30	-	-	-	-	-	2
MOUNTAIN	26	27	130	304	60	79	-	-	-	-	-	-
Mont.	-	1	1	3	2	1	-	-	-	-	-	-
ldaho Wyo.	1 -	1 1	6 2	8 1	3	4	-	-	-	-	-	-
Colo. N. Mex.	9 8	1 6	36 17	60 5	18 13	15 25	-	-	-	-	-	-
Ariz.	7	14	50	183	19	17	-	-	-	-	-	-
Utah Nev.	1	3	9 9	15 29	2 3	7 10	-	-	-	-	-	-
PACIFIC	20	21	637	818	218	214	-	-	-	-	-	17
Wash. Oreg.	2 4	- 8	29 37	49 45	6 13	2 13	-	-	-	-	-	2 8
Calif.	4	12	568	721	196	192	-	-	-	-	-	7
Alaska Hawaii	1 9	1	3	2 1	2 1	4 3	-	-	-	-	-	-
Guam	-	-	-	2	-	2	-	-	-	-	-	-
P.R.	-	Ū	15	12	8	18	Ū	-	Ū	-	-	Ū
V.I. Amer. Samoa	-	U	-	UU	-	UU	U	-	U	-	-	U
C.N.M.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	gococcal		larch 5,	1000 (-				
	Dis Cum.	ease Cum.		Mumps Cum.	Cum.		Pertussis Cum.	Cum.		Rubella Cum.	Cum.
Reporting Area	2000	1999	2000	2000	1999	2000	2000	1999	2000	2000	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	34 42	17 9	-	1	-
Vt. Mass.	16	16	-	-	2	1 -	44	9 72	-	-	- 1
R.I. Conn.	- 6	1	-	-	-	2 1	6 2	- 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 8	20 13	-	-	3	-	-	10 2	-	-	
Pa.	8	11	-	2	4	11	21	23	-	-	-
E.N. CENTRAL Ohio	49 13	72 26	1	6 3	7 2	9 6	122 108	94 56	-	-	-
Ind.	14	6	-	-	-	2	5	4	-	-	-
III. Mich.	4 14	26 8	- 1	1 2	2 3	- 1	5 4	10 12	-	-	-
Wis.	4	6	-	-	-	-	-	12	-	-	-
W.N. CENTRAL	43 1	58 11	-	10	2	1	21 7	28	-	3	-
Minn. Iowa	8	9	-	3	2	-	6	5	-	-	-
Mo. N. Dak.	29 1	21	-	1	-	- 1	2 1	6	-	-	-
S. Dak.	2	4	-	-	-	-	1	1	-	-	-
Nebr. Kans.	1 1	3 10	-	4 2	-	-	4	1 15	-	3	-
S. ATLANTIC	75	60	1	8	9	5	43	50	-	-	-
Del. Md.	- 5	1 12	- 1	- 2	- 2	1	1 13	- 21	-	-	-
D.C.	-	1	-	-	1	-	-	-	-	-	-
Va. W. Va.	12 1	5 1	-	1 -	2	2	3	7	-		
N.C. S.C.	14 6	8 11	-	2 3	1 2	-	15 9	18 4	-	-	-
Ga.	17	14	-	-	-	2	2	-	-	-	-
Fla.	20	7	U	-	1	U	-	-	U	-	-
E.S. CENTRAL Ky.	19 4	39 8	-	1	1	-	12 7	20 4	-	-	-
Ténn. Ala.	7 7	13 11	-	- 1	- 1	-	1 4	9 6	-	-	-
Miss.	1	7	-	-	-	-	-	1	-	-	-
W.S. CENTRAL	21	44	-	-	12	-	3	25	-	-	2
Ark. La.	2 12	8 23	-	-	2	-	3	2 2	-	-	-
Okla. Tex.	7	11 2	-	-	1 9	-	-	3 18	-	-	- 2
MOUNTAIN	22	49		3	5	16	153	160		- 1	2
Mont.	-	-	-	-	-	-	1	-	-	-	-
ldaho Wyo.	2	6 2	-	-	-	1	24	71 1	-	-	-
Cólo. N. Mex.	7 4	14 7	-	- 1	2 N	13 2	82 27	27 7	-	-	-
Ariz.	6	15	-	-	-	-	14	34	-	-	-
Utah Nev.	3	3 2	-	2	2 1	-	4 1	18 2	-	1	-
PACIFIC	124	71	1	33	23	14	55	222	-	-	-
Wash.	6 13	10 17	1 N	1 N	N	6	19 13	22	-	-	-
Oreg. Calif.	102	37	-	31	18	7	20	188	-	-	-
Alaska Hawaii	1 2	3 4	-	- 1	1 4	- 1	2 1	1 8	-	-	-
Guam	-	-	-	-	1	-	-	-	-	-	-
P.R. V.I.	-	2 U	Ū	-	U	Ū	-	Ū	Ū	-	Ū
Amer. Samoa	-	U	U	-	U	U	-	U	U	-	U
C.N.M.I. N: Not notifiable	-	U Iavailable	U	- no reporte	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 Causes, By Age (Years)				, 200		-	All Co.	ana Pi	/ Age (Y	(0010)				
Reporting Area	All		-	-			P&l [†] Total	Reporting Area	All						P&l⁺ Total
	Ages	≥65	45-64	25-44	1-24	<1			Ages	≥65		25-44	1-24	<1	
NEW ENGLAND Boston, Mass.	562 154	412 110	98 29	34 8	11 5	7 2	83 25	S. ATLANTIC Atlanta, Ga.	1,468 U	997 U	280 U	120 U	33 U	34 U	126 U
Bridgeport, Conn	. 23	14	6	3	-	-	4	Baltimore, Md.	276	190	50	27	6	3	39
Cambridge, Mass Fall River, Mass.	. 18 27	13 23	3 4	2	-	-	7 6	Charlotte, N.C. Jacksonville, Fla	. 125 149	78 106	24 32	14 6	6 3	3 3 2	14 14
Hartford, Conn.	U 31	U 24	U 2	U 5	U	U	U 2	Miami, Fla. Norfolk, Va.	104 74	82 52	17 12	5 6	- 1	-	9
Lowell, Mass. Lynn, Mass.	20	18	1	1	-	-	3	Richmond, Va.	67	41	16	2	3	3 3	5 7
New Bedford, Ma New Haven, Conn		30 37	7 6	3	1	2	2 4	Savannah, Ga. St. Petersburg, F	78 la. 58	57 45	13 8	5 4	-	3 1	4 3
Providence, R.I.	70	53	13	2	1	1	2	Tampa, Fla.	209	161	29	12	4	3	24
Somerville, Mass Springfield, Mass		3 25	- 10	1 5	2	-	- 8	Washington, D.0 Wilmington, Del		185 U	79 U	39 U	10 U	13 U	7 U
Waterbury, Conn.	32	24	6	2	2	-2	8	E.S. CENTRAL	966	695	175	62	19	15	106
Worcester, Mass.	55	38	11	2			12	Birmingham, Ala	a. 203	147	37	16	2	1	29
MID. ATLANTIC Albany, N.Y.	2,414 46	1,687 32	469 9	177 4	39 1	37	126 2	Chattanooga, Te Knoxville, Tenn.	nn. 80 86	64 65	11 14	5 4	- 2	- 1	14 6
Allentown, Pa. Buffalo, N.Y.	Ú 100	U 74	Ú 15	U 8	U 1	U 2	Ū 9	Lexington, Ky.	64	45	13 40	1	2	3	2
Camden, N.J.	41	25	9	2	-	5	2	Memphis, Tenn. Mobile, Ala.	200 79	136 61	40	13 5	4	7	22 5 7
Elizabeth, N.J. Erie, Pa.§	20 46	20 35	- 10	- 1		-	-2	Montgomery, A Nashville, Tenn.	la. 64 190	47 130	8 40	5 13	4 4	-3	7 21
Jersey City, N.J.	39	24	10	3	1	1	-	W.S. CENTRAL	1,536	1,017	311	123	44	41	112
New York City, N.' Newark, N.J.	Y. 1,227 U	854 U	222 U	102 U	25 U	19 U	31 U	Austin, Tex.	62	45	12	2	3	-	1
Paterson, N.J. Philadelphia, Pa.	19 459	9 290	8 121	2 36	- 9	-3	2 30	Baton Rouge, La Corpus Christi, 1	. 43 Tex. 68	19 47	13 9	7 4	2 4	2 4	2 6
Pittsburgh, Pa.§	55	40	9	5	-	1	3	Dallas, Tex.	228	148 U	49 U	18 U	5 U	8 U	8 U
Reading, Pa. Rochester, N.Y.	29 131	23 98	4 25	2 5	-1	2	6 15	El Paso, Tex. Ft. Worth, Tex.	U 126	80	27	9	4	6	6
Schenectady, N.Y		17 21	5	1	-	-	1	Houston, Tex. Little Rock, Ark.	455 84	292 51	86 21	53 7	14 3	10 2	38 3
Scranton, Pa.§ Syracuse, N.Y.	111	90	14	3	-	4	16	New Orleans, La	. Ū	U	U	U	U	U	U
Trenton, N.J. Utica, N.Y.	25 18	19 16	4 1	2	-1	-	5 1	San Antonio, Te Shreveport, La.	x. 269 58	196 41	57 9	10 3	4 3	2	33 7
Yonkers, N.Y.	Ű	Ű	Ú	U	Ú	U	Ú	Tulsa, Ókla.	143	98	28	10	2	5	8
E.N. CENTRAL	2,205 49	1,503	453	147	49 1	51	225	MOUNTAIN Albuquerque, N	1,006 .M. 97	736 80	161 11	64 4	25 1	20 1	91 13
Akron, Ohio Canton, Ohio	37	37 30	9 5	2 1	1	-	7 3	Boise, Idaho	31	20	4	3	4	-	2
Chicago, III. Cincinnati, Ohio	485 89	302 61	112 14	42 9	15 4	12 1	83 8	Colo. Springs, C Denver, Colo.	olo. 48 126	40 89	4 18	1 12	1 3	2 4	3 18
Cleveland, Ohio	154	91	40	12	7	4	6	Las Vegas, Nev. Ogden, Utah	195 20	138 17	41 3	12	4	-	14
Columbus, Ohio Dayton, Ohio	215 145	146 112	44 25	14 5	6 1	5 2	18 10	Phoenix, Ariz.	195	135	32	15	7	6	13
Detroit, Mich. Evansville, Ind.	187 58	109 46	54 10	16 2	5	23	16 5	Pueblo, Colo. Salt Lake City, U	32 tah 84	26 61	5 11	1 6	- 1	-5	2 10
Fort Wayne, Ind.	70	53	9	6	-	2	9	Tucson, Ariz.	178	130	32	10	4	2	16
Gary, Ind. Grand Rapids, Mi	21 ch. 53	11 43	8 5	1 2		1 3	1 8	PACIFIC	2,251	1,633	402	124	51	39	216
Indianapolis, Ind.	205	142	40	9	4	10	19	Berkeley, Calif. Fresno, Calif.	19 169	15 127	2 27	1 8	-7	1	23
Lansing, Mich. Milwaukee, Wis.	46 132	30 86	13 32	2 6	- 3	1 5	7 9	Glendale, Calif. Honolulu, Hawa	38 ii 91	28 69	8 14	2 3	- 1	-4	1 7
Peoria, III. Rockford, III.	46 69	43 52	2 10	1 7	-	-	3 5	Long Beach, Cal	if. 79	51	17	8	2	1	12
South Bend, Ind.	49	41	5	2	1	-	2	Los Angeles, Cal Pasadena, Calif.	if. 796 26	577 20	137 4	52	18 1	12 1	57 6
Toledo, Ohio Youngstown, Ohi	95 o U	68 U	16 U	8 U	1 U	2 U	6 U	Portland, Oreg.	170	128	29 34	10	2	1	11
W.N. CENTRAL	864	600	162	59	22	21	79	Sacramento, Ĉal San Diego, Calif	. 201	111 137	37	8 6	5 9	5 10	20 22
Des Moines, Iowa	101	70	21	7	1	2	13	San Francisco, C San Jose, Calif.	alif. U 189	U 135	U 39	U 9	U 5	U 1	U 23
Duluth, Minn. Kansas City, Kans	. 55 . 35	39 22	9 6	4 2	3 4	- 1	3 3	Santa Cruz, Calif	f. 31	27	3	1	-	-	2
Kansas City, Mo. Lincoln, Nebr.	93 32	67 24	16	7 4	1	2	13 6	Seattle, Wash. Spokane, Wash.	132 45	89 36	30 9	9	1	3	15 5
Minneapolis, Min	n. 173	132	29	5	5	2	19	Tacoma, Wash.	102	83	12	7	-	-	12
Omaha, Nebr. St. Louis, Mo.	98 104	65 60	20 29	10 9	2 2	1 4	5	TOTAL	13,272 [¶]	9,280	2,511	910	293	265	1,164
St. Paul, Minn.	60	50	6	1	-	3	10								
Wichita, Kans.	113	71	23	10	4	5	7								

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.

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