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Amanita phalloides Mushroom Poisoning — Northern California, January 1997

The popular interest in gathering and eating uncultivated mushrooms has been associated with an increase in incidents of serious mushroom-related poisonings (*1*). From December 28, 1996, through January 6, 1997, nine persons in northern California required hospitalization after eating *Amanita phalloides* (i.e., "death cap") mushrooms; two of these persons died. Risks associated with eating these mushrooms result from a potent hepatotoxin. This report describes four cases of *A. phalloides* poisoning in patients admitted to a regional referral hospital in northern California during January 1997 and underscores that wild mushrooms should not be eaten unless identified as nonpoisonous by a mushroom expert.

Case 1. A 32-year-old man gathered and ate wild mushrooms that he believed were similar to other mushrooms he had previously gathered and eaten. Eight hours later, he developed vomiting and profuse diarrhea; he was admitted to a hospital 19 hours after ingestion. On admission, he was dehydrated, and laboratory findings included an aspartate aminotransferase (AST) level of 81 U/L (normal: 0–48 U/L), prothrombin time (PT) of 12.3 seconds (normal: 11.0–12.8 seconds), and bilirubin level of 0.9 mg/dL (normal: 0–0.3 mg/dL). He received intravenous fluids, intravenous penicillin, repeated oral doses of activated charcoal, and oral N-acetylcysteine. Although the diarrhea resolved after 24 hours, his PT and AST and bilirubin levels continued to rise. On the third day after eating the mushrooms, abnormal findings included an AST level of 2400 U/L, alanine aminotransferase (ALT) level of 4100 U/L (normal: 0–53 U/L), PT of >60 seconds, and total bilirubin level of 11 mg/dL. Six days after eating the mushrooms, his bilirubin level was 16 mg/dL, and his AST level had decreased to 355 U/L; he developed metabolic acidosis and hypotension. Seven days after eating the mushrooms, he developed hepatic encephalopathy, oliguric renal failure, and adult respiratory distress syndrome requiring intubation and mechanical ventilation. He died from multiple organ failure 9 days after eating the mushrooms. One mushroom cap remaining after the meal was identified as *A. phalloides*.

Case 2. A 42-year-old man developed vomiting and diarrhea 11 hours after eating wild mushrooms, and he was admitted to a hospital 14 hours after eating the mushrooms. His transaminase levels were elevated 24 hours after ingestion (AST and ALT levels both at 100 U/L); his PT was 12.1 seconds, and his bilirubin level was 0.2 mg/dL. His PT became prolonged the next day and peaked at 35 seconds on the fourth day.

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His transaminase levels also peaked on the fourth day (AST level of 3000 U/L and ALT level of 6000 U/L); his bilirubin level was 7.8 mg/dL. He was given repeated doses of activated charcoal and oral N-acetylcysteine. His transaminase levels and PT gradually decreased, and he was discharged on the seventh day after eating the mushrooms without sequelae.

Case 3. A 30-year-old man used a guidebook to assist in the collection of wild mushrooms. Twelve hours after eating the mushrooms he had gathered, he developed vomiting and severe diarrhea. He was admitted to a hospital 17 hours after ingestion because of orthostatic hypotension and dehydration. Abnormal laboratory findings indicated an AST level of 75 U/L, blood urea nitrogen level of 22 mg/dL (normal: 6–20 mg/dL), and creatinine level of 2.8 mg/dL (normal: 0.6–1.3 mg/dL). He was treated with intravenous fluids. Although renal function indicators were within normal limits 1 day after admission, his liver enzyme and PT levels began to increase; on the fourth day, transaminase levels peaked (AST level of 1900 U/L and ALT level of 2800 U/L), total bilirubin level was 1.6 mg/dL, and PT was 18 seconds. His clinical status continued to improve, and he was discharged 7 days after eating the mushrooms.

Case 4. A 68-year-old man ate mushrooms he had collected on a golf course. Two days after eating the mushrooms, he was admitted to a hospital because of diarrhea and weakness. His AST level was 630 U/L, and he had renal failure. On the third day after eating the mushrooms he required hemodialysis, and his transaminase levels and his PT continued to increase; on the fifth day, his AST level was 3500 U/L; ALT level, 4600 U/L; PT, 34 seconds; and bilirubin, 9.7 mg/dL. He developed hepatic encephalopathy and died 6 days after eating the mushrooms.

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Editorial Note: Ingestion of *A. phalloides* may account for approximately 90% of deaths attributable to mushroom ingestion worldwide (*1–5*); the proportion of cases of mushroom poisoning attributable to *A. phalloides* in the United States is unknown. In the United States, this species is found primarily in the cool coastal regions of the west coast, but it also grows in several other regions, including the mid-Atlantic coast and in the northeast (*1,2*). These mushrooms flourish in favorable weather conditions during the fall or the rainy season (*2,6*). The mature cap usually is metallic green but varies from light yellow to greenish-brown (*1–3*). *A. phalloides*, like most mushroom species, is not unique in appearance and can be mistaken for nonpoisonous species; it has no distinct taste or smell, and the toxins are not destroyed by cooking or drying (*3,5,6*). The principal toxins (amatoxins) are taken up by hepatocytes and interfere with messenger RNA synthesis, suppressing protein synthesis and resulting in severe acute hepatitis and possible liver failure. Radioimmunoassay of amatoxins can be obtained from serum and urine; the tests are performed at referral laboratories (*1,2*).

Since 1979, *A. phalloides* has been found in the region from northern California to Washington state, and since 1995, it has appeared in greater numbers because of abundant rainfall during winter months. During the winter of 1995–96, at least 13 persons in northern California were hospitalized for treatment of poisonings after eating *A. phalloides*; one patient died, and another required a liver transplant. The cluster of

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mushroom poisoning in northern California described in this report probably occurred because warm, heavy rainfall created optimal conditions for the growth of *A. phalloides* in unprecedented numbers. In addition, this mushroom grew in places where it had not grown before (e.g., backyards), which increased the likelihood that persons gathering these mushrooms could mistake them for a nonpoisonous species.

Patients may not associate their symptoms with ingestion of wild mushrooms because of the delayed onset. As illustrated by the cases described in this report, symptoms typically occur in a progression through three stages. During the first stage, which occurs 6–24 hours after ingestion, symptoms may include abdominal pain, nausea, vomiting, severe diarrhea, fever, tachycardia, hyperglycemia, hypotension, and electrolyte imbalance. During the second stage, which occurs during the next 24–48 hours, symptoms appear to abate even as hepatic and renal functions deteriorate. During the third stage, which occurs 3–5 days after the ingestion, hepatocellular damage and renal failure may progress, resulting in jaundice and hepatic coma (*1–5*). Possible sequelae include cardiomyopathy, coagulopathy, and seizures (*1,2,5*). Death from *A. phalloides* poisoning usually results from hepatic and/or renal failure and may occur 4–9 days after ingestion. Fatal outcomes are associated with age <10 years, a short latency between ingestion and onset of symptoms, and severe coagulopathy (*1,4*). The fatality rate among persons treated for *A. phalloides* poisoning is 20%–30% (*1,2,4*), and the median lethal dose is 0.1 mg to 0.3 mg of the toxin per kg of body weight (*1,5*).

A. phalloides poisoning has no specific antidote. The main treatment is vigorous intravenous fluid replacement and correction of electrolyte disturbances (*1–5*); correction of coagulopathy, if present, also may be indicated. Physicians should perform gastric lavage and administer repeated doses of activated charcoal to remove any unabsorbed *Amanita* and to interrupt the enterohepatic circulation of the toxin (*2,4,5*). Although some therapeutic regimens have included the administration of penicillin, cimetidine, silibinin, or N-acetylcysteine, these treatments have not been confirmed by clinical trials to be effective. Hemodialysis and hemoperfusion may be effective in removing the toxin if initiated within 24 hours of ingestion (*7*). The only definitive treatment may be liver transplantation once fulminant liver failure occurs (*1,2,4*).

Unintentional ingestion of *A. phalloides* can be prevented by ensuring that wild mushrooms are not eaten unless identified as nonpoisonous by a competent mycologist. Education campaigns should be established in areas where *A. phalloides* is common to educate the public about the potentially lethal consequences associated with eating uncultivated mushrooms. Field guides do not provide sufficient details to differentiate toxic from nontoxic species. Health-care providers should report cases of mushroom poisoning to poison-control centers; these centers can provide expertise in the clinical management of mushroom poisoning.

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Mushroom Poisoning — Continued

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As part of its commemoration of CDC's 50th anniversary, MMWR *is reprinting selected* MMWR *articles of historical interest to public health, accompanied by a current editorial note. Reprinted below is the first* MMWR *report of toxic-shock syndrome, which was published May 23, 1980.*

Epidemiologic Notes and Reports

Toxic-Shock Syndrome — United States

Cases of a newly recognized illness known as toxic-shock syndrome (*1*) have recently been reported to CDC by state health departments in Wisconsin, Minnesota, Illinois, Utah, and Idaho. Physicians in 8 other states have reported individual cases to CDC or to investigators at the University of Colorado, Denver.

Toxic-shock syndrome typically begins suddenly with high fever, vomiting, and profuse watery diarrhea, sometimes accompanied by sore throat, headache, and myalgias. The disease progresses to hypotensive shock within 48 hours, and the patient develops a diffuse, macular, erythematous rash with non-purulent conjunctivitis. Urine output is often decreased, and patients may be disoriented or combative. The adult respiratory distress syndrome or cardiac dysfunction may also be seen.

Laboratory studies reveal elevated blood urea nitrogen, serum creatinine, bilirubin, and creatine phosphokinase levels, and white blood cell counts with marked left shifts. Platelet counts are low in the first week of illness but are usually high in the second week.

Patients require large volumes of fluid to maintain perfusion and usually require intensive care. In the recovery phase, there is desquamation of at least the palms, soles, or digits and often of other skin areas as well.

Since October 1, 1979, 55 cases have been reported to CDC. Fifty-two of these (95%) have been in women. The mean age is 24.8 years, with a range of 13-52 years. Seven deaths have occurred, for a case-fatality ratio of 13%.

Of 40 patients in whom a menstrual history was obtained, 38 (95%) had onset of illness with the 5-day period following onset of menses. Two others had onset of illness 10 days after onset of menses. Moreover, 13 patients have had recurrence of symptoms with a subsequent menstrual period.

In 33 of 45 (73%) patients cultured, *Staphylococcus aureus* was isolated from the throat, cervix, vagina, or rectum. Four of 15 patients (27%) tested for *Herpesvirus hominis* had serologic or cultural evidence of herpes infection. No evidence for leptospirosis, Rocky Mountain spotted fever, viral exanthematous diseases, or streptococcal scarlet fever has been found in those patients in whom it has been looked for.

Toxic-Shock Syndrome — Continued

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Editorial Note: Toxic-shock syndrome is a serious disease of unknown etiology. It affects primarily young women of child-bearing age who have been previously healthy, and it has a case-fatality ratio for reported cases of 10%-15%. This ratio is probably high because severe cases are easier to recognize. In Wisconsin, where surveillance has been very active, the case-fatality ratio has been 3.2%. The incidence of the disease is not known but is apparently low. The increasing number of reported cases over the past 6 months is probably due to increasing recognition. In support of this theory, a review of medical charts in Wisconsin for the past 2 years revealed 6 cases fitting the case description that had not previously been recognized as toxic-shock syndrome.

The syndrome resembles Kawasaki disease (mucocutaneous lymph node syndrome) in several respects, namely fever, rash with subsequent desquamation, and cardiac involvement. However, shock, which is prominent in toxic-shock syndrome, is not usually seen in Kawasaki disease. The character of the rash is also different in the 2 diseases: it is a maculopapular one in Kawasaki disease but a non-papular, diffuse erythroderma in toxic-shock syndrome. Azotemia and thrombocytopenia are rarely seen in Kawasaki disease and are common in toxic-shock syndrome. Kawasaki disease classically occurs in children less than 5 years of age; some recently reported cases of "adult Kawasaki disease" (*2,3*) may actually be cases of toxic-shock syndrome.

Toxic-shock syndrome was first recognized in 7 children aged 8-17 years, 3 of whom were boys (*1*). In 5 of the 7, *S. aureus* was isolated from the nasopharynx, vagina, or localized abscess. At that time it was hypothesized that the syndrome was caused by a toxin elaborated by the staphylococci. Although *S. aureus* was isolated from vaginal cultures in two-thirds of patients in the current report, no control study has been done to show that this prevalence is unusually high. The isolation of *Herpesvirus* in a small number of cases probably reflects stress-related recurrence of infection and not an etiologic role for the virus. CDC, in cooperation with a number of investigators, is setting up a nationwide case-control study to try to define the epidemiologic features and the cause of this disease.

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Toxic-Shock Syndrome — Continued

Editorial Note—1997: Although case reports of "Staphylococcal scarlet fever" had been published in the medical literature as far back as the 1920s, a 1978 report describing seven cases of what was named toxic-shock syndrome (TSS) heralded the apparent emergence of TSS in late 1979 and early 1980 (*1*). The report about TSS in the May 23, 1980, *MMWR* and the veritable landslide of studies of TSS that followed demonstrate the speed and effectiveness with which astute clinicians—together with public health officials, epidemiologists, and laboratory scientists—can respond to an "emerging" infectious disease threat. Did TSS truly "emerge" at that time, or did the intensive case-finding efforts of clinicians and epidemiologists in states such as Wisconsin and Minnesota simply make it appear to "emerge"? The limited data available from retrospective chart-review studies that were designed to identify TSS cases, whether previously diagnosed or not, clearly demonstrated that the number of cases of TSS in women of reproductive age increased beginning in the late 1970s (*2–4*). Cases of TSS in men also occurred during that time but at a low and stable rate. Thus, what "emerged" during late 1979–early 1980 was not all TSS, but TSS in reproductive-aged women, particularly menstruating women, as reflected in the dramatic data presented in the *MMWR* report—of the 55 reported cases, 95% occurred among women, and 95% of the cases among women for whom information was available had onset of their illness within the 5-day period following onset of menses.

The startling proportion of TSS cases identified during 1979–1980 among women who had onset during menstruation led investigators to focus on understanding the risk factors for development of menstrual TSS, rather than TSS in general. The wave of rapidly completed case-control studies of menstrual TSS that followed clearly demonstrated that use of various brands and styles of tampons was by far the most important risk factor for TSS during menstruation (*5–8*). Although the relative importance of absorbency, chemical composition, and other tampon-related factors in determining the risk for menstrual TSS has remained difficult to determine, the most plausible explanation for the "emergence" of menstrual TSS in the late 1970s was the manufacture and widespread use of more absorbent tampons made of a variety of materials not previously used in tampons. There is no evidence to suggest that changes in *Staphylococcus aureus*, the source of the toxin that causes TSS, were responsible for the emergence of menstrual TSS.

The week after the *MMWR* report appeared in May 1980, Dr. William Foege, the director of CDC at the time, testified before the Senate Subcommittee on Health regarding "toxic dumps." Given the widespread news media attention the *MMWR* report had received and a perceived connection between toxic dumps and toxic-shock syndrome, Dr. Foege also was asked about TSS at that hearing, and he optimistically promised "an answer" by the end of 1980. Although much more was learned about TSS during the years that followed (e.g., the biologically important properties of TSS toxin-I, the toxin responsible for most cases of TSS, particularly menstrual cases), in retrospect Dr. Foege was correct. From the public health point of view, before the end of 1980, enough was known about menstrual TSS based primarily on observational epidemiologic studies to promulgate recommendations (*9,10*) that led to a substantial reduction in the risk for menstrual TSS.

Perhaps less well known in the public health community is the important legal precedent that emerged from the civil litigation surrounding menstrual TSS. Faced with a large number of lawsuits filed by women with menstrual TSS, one of the tam-

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pon manufacturers filed suit to compel CDC to release the names and other personal identifiers of all women who had participated in the CDC case-control studies of menstrual TSS. Because the results of these studies (and hence the "collective evidence" of the study participants) were being introduced as evidence by women in their lawsuits against the manufacturer, the manufacturer argued that it had a fundamental legal right to know who these women were and even cross-examine them. Although the manufacturer had been given copies of all the data tapes and all the raw data forms from the studies (with identifiers removed) so its experts could reanalyze the results, the manufacturer also argued that it needed to re-interview the study participants several years after the case-control studies had been conducted to assess the extent to which bias had been introduced at the time of the original interviews (*11*). The federal appeals court decided that the manufacturer could not have access to the personal identifiers of the study participants. The court ruled that in furtherance of its mission to protect the public health, CDC must be able to "conduct probing scientific and social research supported by a population willing to submit to indepth questioning." The court further ruled that "disclosure of the names and addresses of … research participants could seriously damage this voluntary reporting" and that "even without an express guarantee of confidentiality there is still an expectation, not unjustified, that when highly personal and potentially embarrassing information is given for the sake of medical research, it will remain private" (*12*). Thus, the series of events that unfolded following the publication of the *MMWR* report not only led to an expeditious public health response to the emergence of menstrual TSS but to enhanced legal protection at the federal level of the public health research process.

1997 Editorial Note by: Arthur L Reingold, MD, University of California, Berkeley. Gene W Matthews, JD, Legal Advisor to CDC. Claire V Broome, MD, Deputy Director, CDC.

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Work-Related Aviation Fatalities — Alaska, 1990–1994

During 1990–1994, the annual occupational fatality rate in Alaska was 29.1 deaths per 100,000 workers, nearly six times the annual rate for the United States (5.1 per 100,000). In the United States, aviation-related fatality is the seventh leading cause of fatal occupational injury (*1*); however, in Alaska, this category is the second leading cause of occupational death. To characterize occupational aviation fatalities in Alaska, CDC analyzed all fatal occupational aviation crashes in Alaska during 1990–1994 (the most recent year for which complete data were available) and compared findings with overall patterns for the United States. This report summarizes the results of that study, which indicate that workers in Alaska are at increased risk for being killed in aircraft crashes when compared with all U.S. workers.

For all aircraft crashes during the study period, National Transportation Safety Board (NTSB) Accident Briefs were abstracted to obtain information about flight purpose, weather, aircraft, pilot, and probable cause. These reports were merged with records from the Alaska Occupational Injury Surveillance System, a database established and maintained by CDC's National Institute for Occupational Safety and Health, which includes information about cause of death, occupation of decedent, and circumstances associated with the crash. This study includes all occupational deaths related to commercial, military, and general aviation (i.e., all flying not involving military aircraft, scheduled airlines, and commuter or air-taxi service). For this analysis, an aircraft crash was defined as an incident in Alaska in which an aircraft in motion sustained substantial damage or an incident that resulted in injury or death to an aircraft occupant. An aircraft crash was categorized as occupational if at least one of the occupants in the aircraft was 1) working for pay or compensation; 2) working as a volunteer emergency medical technician, firefighter, or law enforcement officer; 3) traveling on business, including to and from customer/business contacts; or 4) engaging in a work activity in which the aircraft is the work environment. Denominator data for rates were based on 1990 U.S. Bureau of the Census and Alaska Department of Labor estimates.

During 1990–1994, a total of 876 aircraft crashes occurred in Alaska; of these, 405 (46%) were occupational. Overall, 106 (12%) crashes resulted in at least one fatality, and 69 (65%) of these were classified as occupational. Of these, 62 (90%) involved fixed-wing aircraft, and seven (10%) involved helicopters. Nearly all (61 [98%]) of the fixed-wing crashes involved propeller-driven aircraft; 54 (89%) were single-engine aircraft. No occupational fatalities occurred on scheduled commercial airline operations.

A total of 192 occupants were on board the 69 aircraft involved in the fatal occupational crashes; 149 (78%) of these occupants were killed. Of the 149 fatalities, 99 (66%) were occupation-related. The annual occupational fatality rate for pilots in Alaska was 268 per 100,000, 2.1 times higher than the U.S. pilot-specific rate of 126 per 100,000 (*1*). For all workers in Alaska, regardless of occupation, the death rate for work-related aircraft crashes was 8.3 per 100,000, 27.1 times higher than the U.S. rate of 0.3 per 100,000 (*1*).

The mean number of persons on board the aircraft was 2.8 (range: one–11); in 23 (33%) of these crashes, only the pilot was on board at the time of the crash. The mean age of the occupational decedents was 39 years (range: 20–68 years), and most (58 [59%]) were aged 30–44 years. In addition, nearly all (96 [97%]) deaths occurred

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among men. The most common cause of death was multiple impact injuries (48 [48%]), followed by head injuries (27 [27%]) and injuries to the chest (11 [11%]).

The takeoff and landing phases of flight together accounted for 228 (56%) occupational crashes, but for only eight (12%) of the fatal occupational crashes. Most (137 [60%]) of these crashes were associated with unimproved, off-airport sites (e.g., sandbars, mountain ridges, and meadows). Controlled flight into terrain during the cruise phase of flight (i.e., straight and level flying) or the maneuvering phase of flight (i.e., changing altitude or direction) together accounted for 46 (67%) fatal occupational crashes. The most common (28 [41%]) impact sites of fatal crashes were mountain sides and passes.

The Federal Aviation Administration (FAA) defines two categories of flying conditions based on meteorologic considerations. Instrument Meteorological Conditions (IMC) exist when visibility is <1 mile or the aircraft cannot be operated clear of clouds or overcast; in IMC, pilots must rely on instrumentation for navigation. Visual Meteorological Conditions (VMC) exist when visibility is ≥1 mile and pilots can use visual cues for navigation. In Alaska, crashes occurring under IMC were 5.3 times (95% confidence interval=3.5–7.9) more likely to be associated with a death than crashes in VMC.

NTSB determined that pilot error (defined as aircrew action or inaction that became a contributing cause or factor in the crash) was a cause in 53 (77%) of the fatal occupational aviation crashes in Alaska. In addition, 23 (33%) of the aircraft involved in fatal occupational incidents were not completely destroyed; however, only 22% of the occupants of these aircraft survived.

Reported by: Alaska Field Station, Div of Safety Research, National Institute for Occupational Safety and Health, CDC.

Editorial Note: When compared with risks for all U.S. workers, occupational aviation fatalities among workers in Alaska accounted for a disproportionate number of occupational fatalities in that state: workers in Alaska were 27 times more likely to be killed in an aircraft than were all U.S. workers. This increased risk reflects, in part, the greater use of aircraft for routine transportation in Alaska. Controlled flight into terrain during the transition from VMC to IMC was the most frequently identified cause of occupational crashes. This transition is a difficult flight task for pilots, and FAA regulations prohibit pilots of single-engine aircraft from flying in IMC while carrying passengers for compensation.

In Alaska, many pilots risk flying into potentially hazardous conditions because of the demand for reliable air service. In 1994, 70% of pilots in Alaska involved in the commuter and air-taxi industry reported inherent pressures in their flight operations, including self-induced pressures, mail-delivery responsibilities, and pressures from passengers, management, and other pilots (*2*). Approximately half of pilots surveyed reported having flown from VMC into IMC on at least one occasion, and 84% reported having inadvertently entered IMC on a VMC flight. Weather conditions in Alaska can change rapidly, and the vast distances between some weather reporting points often conceal substantial local variation in the weather. However, VMC flight into IMC usually involved poor pilot decision making (*3*).

The frequency of pilot error in the incidents described in this report underscores the need for the development and introduction of Alaska-specific Aeronautical Decision Making (ADM) and judgement training (*3*). ADM is designed to assist pilots in making

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better decisions during potentially hazardous conditions, to avoid situations that require skill beyond their capabilities, and to reduce the number of judgement-related crashes. The FAA has proposed requiring ADM training for all levels of pilot certification in the United States (*4*).

NTSB has recommended that all pilots use protective equipment to reduce aviation fatalities (*5*). Helmets, energy-absorbing structures, padding the occupant's immediate environment, and use of shoulder restraints could reduce the number of aircraft-related occupational fatalities (*5,6*).

This analysis produced a descriptive characterization of the epidemiology of occupational aviation fatalities in Alaska. Additional efforts will be required to assess the association between other potential risk factors (e.g., carbon monoxide exposure, aging aircraft, pilot fatigue, and risk-taking) and occupational aviation fatalities. One important limitation of this analysis was the lack of accurate and reliable denominator data to control for exposure (i.e., flight hours). Fatal aircraft incident rates provided by the NTSB generally are presented as fatal incidents per 100,000 aircraft flight hours; however, these rates are based on national estimates, and these estimates cannot be applied to occupational aviation in Alaska.

In response to this study, the Alaska Interagency Working Group for the Prevention of Occupational Injuries has formed an aviation working group (including representatives from industry and state and federal agencies), to determine strategies for reducing such crashes. Ongoing activities include data collection and dissemination of information to local news media, industry, and educational and flight-safety organizations in Alaska.

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Prevalence of Aspirin Use to Prevent Heart Disease — Wisconsin, 1991, and Michigan, 1994

In the late 1980s, a series of secondary prevention trials documented that regular use of aspirin lowered the risk for myocardial infarction (MI) and nonfatal strokes in persons with cardiovascular disease (CVD) (*1,2*). Subsequently, a large randomized trial demonstrated that regular use of aspirin decreased the risk for MI by

Aspirin Use — Continued

approximately half in healthy male physicians with no history of CVD (*3*), suggesting a potential role for aspirin in primary prevention of heart disease. In 1989, the U.S. Preventive Services Task Force (USPSTF) recommended that regular low-dose aspirin should be considered for men aged ≥40 years who were at substantially increased risk for MI and who lacked contraindications to the drug (*4*). To assess the prevalence of self-reported, regular aspirin use to prevent heart disease among adults aged ≥45 years, both the Wisconsin and Michigan state health departments collected information in their Behavioral Risk Factor Surveillance System (BRFSS) surveys (in 1991 and 1994, respectively). This report summarizes the results of these surveys, which indicate that a high proportion of adults in those states used aspirin regularly to prevent heart disease.

The BRFSS is a random-digit–dialed survey of the U.S. civilian, noninstitutionalized population aged >18 years. In 1991, the Wisconsin BRFSS included the question "Do you take aspirin regularly to reduce your chances of having a heart attack?" In 1994, Michigan asked "Do you take aspirin daily or every other day to reduce your chance of a heart attack or stroke?" Responses were obtained from 548 and 1137 adults aged ≥45 years in Wisconsin and Michigan, respectively. The overall prevalence of aspirin use was 19.5% in Wisconsin in 1991 and 25.3% in Michigan in 1994. Because univariate results in each state were similar, the data were combined for more detailed analyses using SUDAAN. Statistical associations between explanatory variables and aspirin use were tested using the chi-square test of association. For those variables with an overall statistically significant association with aspirin use ($p<0.05$), pairwise comparisons of age-adjusted prevalence estimates were performed (Table 1). Age-adjusted estimates were calculated using the pooled age distribution from both data sets. A composite risk-score variable also was constructed using a combination of three risk factors—current smoking, overweight, and inactivity.

The overall prevalence of aspirin use in the combined data was 23.3% (Table 1). Prevalences increased directly with age from 16.0% of persons aged 45–54 years to 22.0%, 28.8% and 33.3% for persons aged 55–64, 65–74, and ≥75 years, respectively. Age-adjusted prevalences were higher for men (27.7%) than women (20.1%), current (25.5%) and former smokers (28.8%) than respondents who never smoked (18.0%) (Table 1), and persons who engaged in regular leisure-time physical activity (26.3%) than persons who were inactive (20.8%). There were no statistically significant associations between aspirin use and race, education, income, overweight, or composite risk-score. Prevalences were similar when the analysis was stratified by sex.

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Editorial Note: Approximately 40% of all deaths in the United States are attributed to CVD, and annual direct and indirect costs of CVD have been estimated to be $259 billion (*5*). In addition to population-based approaches to reducing CVD risk factors, prevention efforts should include efficacious and cost-effective therapies to both reduce the incidence of MI (primary prevention), and to prevent further cardiac events in persons who have had a CVD event (secondary prevention). Although the effectiveness of regular aspirin use for primary prevention has not been determined for the general population, aspirin use for secondary prevention has been documented to be effective and is widely recommended (*6*).

Aspirin Use — Continued

TABLE 1. Crude and age-adjusted prevalence* of aspirin use to prevent cardiovascular disease among adults aged ≥45 years, by selected demographics and risk factor groups — Wisconsin, 1991, and Michigan, 1994, Behavioral Risk Factor Surveillance System

Category	Sample size	Crude prevalence		Age-adjusted prevalence	
		%	SE†	%	SE
DEMOGRAPHICS					
State§					
Wisconsin	548	19.5	1.8	19.1	1.8
Michigan	1137	25.3	1.4	25.5¶	1.4
Age group yrs§					
45–54	577	16.0	1.6	—	—
55–64	434	22.0	2.0	—	—
65–74	424	28.8	2.3	—	—
≥75	250	33.3	3.2	—	—
Sex§					
Male	684	26.7	1.8	27.7**	1.8
Female	1001	20.4	1.4	20.1	1.3
Race††					
White	1513	23.5	1.2	23.4	1.1
Black	126	19.6	3.7	20.4	3.8
Other	43	28.4	8.3	34.2	8.3
Education					
Less than high school	327	25.5	2.5	24.5	3.0
High school graduate	653	23.0	1.7	23.1	1.7
Some college	344	23.2	2.5	24.5	2.4
College graduate	361	22.0	2.4	24.9	2.7
Income					
<$10,000	226	29.8	3.4	31.8	3.9
$10,000–$19,999	379	25.4	2.4	22.8	2.4
$20,000–$34,999	396	23.9	2.2	24.5	2.3
$35,000–$49,999	206	18.8	2.9	24.6	3.9
≥$50,000	309	19.7	2.4	27.9	4.4
Unknown/Refused	169	22.9	3.4	19.9	3.1
RISK FACTORS					
Smoking status§					
Current§§	349	21.1	2.3	25.5¶	3.0
Former§§	590	29.5	2.0	28.8¶	1.9
Never	741	18.8	1.5	18.0¶¶	1.5
Activity level§					
Active***	722	25.5	1.8	26.3¶	1.8
Inactive	949	21.2	1.4	20.8	1.4
Overweight†††					
No	1083	23.9	1.4	23.5	1.4
Yes	576	22.7	1.8	23.5	1.8
Risk-score§§§					
None	416	25.7	2.3	25.3	2.2
One	679	24.5	1.8	23.8	1.8
Two	478	19.2	1.9	19.9	1.9
Three	67	21.8	5.4	17.1	4.1
Total	**1685**	**23.3**	**1.1**	**—**	**—**

* Age-adjusted using the pooled age distribution from both data sets.
† Standard error.
§ Statistically significant chi-square test of association ($p<0.05$) between variable and aspirin use.
¶ $p<0.05$.
** $p<0.01$.
†† Numbers for races other than black and white were too small for meaningful analysis.
§§ Current—persons who reported having smoked at least 100 cigarettes during their lifetimes and who smoke now. Former—persons who reported having smoked at least 100 cigarettes and who do not smoke now.
¶¶ Reference group used for pairwise statistical testing of multiple level variable.
*** Persons who undertook leisure-time physical activity for at least 20 minutes three or more times a week during the previous month.
††† A body mass index of >27.3 for women and >27.8 for men.
§§§ The number of risk factors present where risk factors are current smoking, overweight, and physical inactivity.

Aspirin Use — Continued

Although the 1989 USPSTF guidelines were specific to high-risk men, the findings in this report indicate that a high proportion of women reported taking aspirin regularly, despite the absence of any specific recommendations about prophylactic aspirin use in women. Some physicians may be prescribing aspirin for their female patients despite the USPSTF recommendations, and some women may be deciding independently to initiate aspirin use.

The proportion of adults in this survey who reported taking aspirin to reduce their risk for heart disease was higher than in a similar study in New York (*7*), possibly reflecting differences in physician practice patterns or differences in the age structure of the two populations. Other factors related to the prevalence of aspirin use for heart disease prevention include the underlying prevalences of CVD risk factors, of preexisting CVD, and variations in public awareness about prophylactic aspirin use.

Although this study did not distinguish between aspirin use for primary or secondary prevention, some of the findings suggest that aspirin use was more common among health-conscious persons. For example, the prevalence of aspirin use was higher among physically active persons. However, prevalence of aspirin use was higher among the elderly, men, and current and former smokers, suggesting that aspirin may have been used for secondary prevention.

The findings in this report are subject to at least three limitations. First, data about regular aspirin use for heart disease prevention was self-reported. As a result, respondents may have overreported aspirin use if they confused prophylactic use with the use of aspirin-like drugs (e.g., ibuprofen) for reasons other than CVD prevention. Second, because aspirin use for primary or secondary prevention was not distinguished, the extent to which the results represent use for primary prevention or for therapy initiated following important cardiovascular events (e.g., MI or stroke) could not be determined. However, based on National Health Interview Survey findings, the prevalence of ischemic heart disease was 6.1% for U.S. adults aged 45–64 years and 15.3% for adults aged ≥65 years (*8*). By assuming that all patients with ischemic heart disease use aspirin regularly, most regular aspirin users in Wisconsin and Michigan probably were using this drug for primary prevention. Third, although the data were adjusted for age and separate analyses were performed for men and women, some of the findings may be confounded by unmeasured CVD risk factors (e.g., hypertension and high cholesterol).

Since collection of the BRFSS data in Wisconsin and Michigan, the second USPSTF report concluded that evidence was insufficient to recommend for or against prophylactic aspirin use for primary prevention of MI in asymptomatic men or women (*9*). Data were insufficient to determine whether the reduced risk for MI in low-risk men is outweighed by the potential risks for adverse effects associated with long-term aspirin use (e.g., gastrointestinal ulceration, hemorrhagic stroke, and sudden death) (*3,9*). The findings in this report indicate that substantial proportions of the populations in Wisconsin and Michigan used aspirin regularly to prevent heart disease, despite the lack of conclusive data on the relative benefits and harms when used for primary prevention. The state health departments in Michigan and Wisconsin are conducting studies to determine whether patients consult their physicians before initiating regular aspirin use for primary prevention of CVD and whether their prophylactic aspirin use is appropriate given their risk factor profile and possible contraindications.

Aspirin Use — Continued

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Suicide — Washington, 1980–1995

The ongoing assessment of health data and health data sources is essential to the development of effective prevention strategies for priority health issues. In Washington, assessment efforts include the analysis of suicide data. In 1995, suicide was the eighth leading cause of death in Washington (*1*), and most (58%) were firearm related. To determine trends in suicide during 1980–1995, the Injury Prevention Program of the Washington Department of Health (WDOH) analyzed death-certificate data. This report presents the findings of the analysis, which indicate that, while overall suicide rates in Washington remained relatively stable during 1980–1995, suicides became more common among persons aged 15–24 years and ≥75 years and less common among persons aged 25–74 years.

Computerized death-certificate data and external cause-of-injury codes (E-codes) were used to identify all suicides (E950–E959) among Washington residents. Population data were derived from the 1980 and 1990 U.S. census and from intercensal and postcensal estimates from the Office of Management of Washington state. Contiguous age categories with similar death rates were grouped, and patterns within age groups were examined.

The average 1-year change in mortality was estimated using negative binomial regression in models that accounted for changes in the age, sex distribution, and size of the population. This regression method is useful for analyzing count data that do not meet the restrictive assumptions of Poisson models (*2*). Results are expressed as the overall percentage change in mortality from 1980 to 1995. Trends are presented graphically using robust locally weighted regression (*3*). Because suicide methods might change over time, trends in firearm-related suicides were compared with those in nonfirearm-related suicides.

Suicide — Continued

During 1980–1995, a total of 10,650 suicides occurred in Washington, representing an overall average rate of 14.2 per 100,000 population. The most common method of suicide was use of firearms (E950.0–E955.4) (56%), followed by poisoning (E950–E954) (23%), suffocation (E953) (13%), and other or unspecified means (8%). Most (78%) suicides occurred among males. Although the overall average rate of suicide in the total population remained relatively constant during the 16-year period, the rate of firearm-related suicide increased 8% (p=0.2), and the rate of suicide by other means decreased 15% (p<0.01) (Table 1). Changes in the overall suicide rate varied by age, increasing by 127% for children aged 5–14 years (all except one suicide in this age group during 1980–1995 occurred among children aged 10–14 years); by 16% for persons aged 15–24 years; and by 42% for persons aged ≥75 years (Figure 1). For persons aged 25–74 years, the rate declined substantially. The increase for children aged 5–14 years primarily reflected an increase in nonfirearm-related suicide, the increase for persons aged 15–24 years and ≥75 years reflected an increase in firearm-related suicide, and the decrease for persons aged 25–74 years reflected a decrease in both firearm-related and nonfirearm-related suicide (Figure 2).

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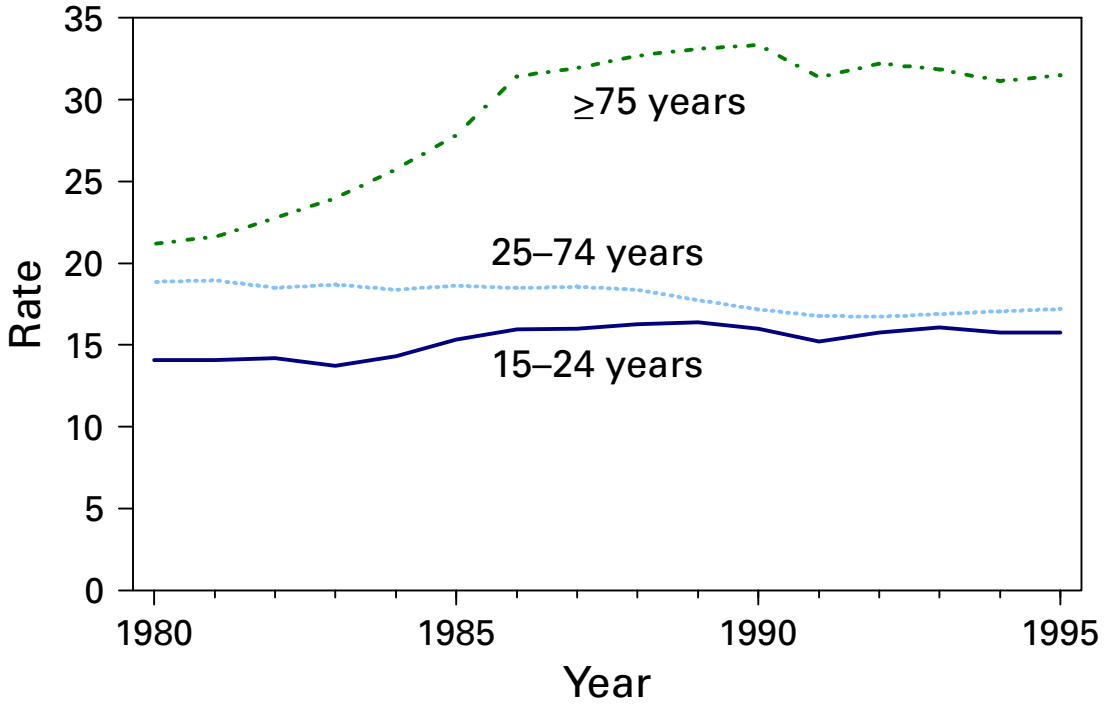
TABLE 1. Number and average rate* of suicides, 1980–1995, and overall percentage change in rate from 1980 to 1995, by age group and method of death — Washington

Age group (yrs)/ Method of death	Suicides		Overall change in rate from 1980 to 1995		
	No.	Average rate	(%)	(95% CI†)	p value§
5–14					
Firearm	56	0.5	+ 69%	(–30% to 310%)	0.2
Nonfirearm	47	0.4	+215%	(17% to 753%)	0.02
Total	**103**	**0.9**	**+127%**	**(15% to 349%)**	**0.02**
15–24					
Firearm	973	8.6	+ 46%	(14% to 87%)	<0.01
Nonfirearm	762	6.7	– 14%	(–34% to 13%)	0.3
Total	**1,735**	**15.4**	**+ 16%**	**(– 6% to 43%)**	**0.02**
25–74					
Firearm	4,200	9.7	– 11%	(–22% to 2%)	0.09
Nonfirearm	3,580	8.2	– 19%	(–29% to – 6%)	<0.01
Total	**7,780**	**17.9**	**– 17%**	**(–26% to – 7%)**	**<0.01**
≥75					
Firearm	705	20.2	+ 70%	(27% to 128%)	<0.001
Nonfirearm	327	9.4	– 5%	(–35% to 39%)	0.8
Total	**1,032**	**29.5**	**+ 42%**	**(10% to 83%)**	**<0.01**
All ages					
Firearm	**5,934**	**7.9**	**+ 8%**	**(– 4% to 21%)**	**0.2**
Nonfirearm	**4,716**	**6.3**	**– 15%**	**(–24% to – 4%)**	**<0.01**
Total	**10,650**	**14.2**	**– 3%**	**(–12% to 7%)**	**0.6**

*Per 100,000 person-years.
†Confidence interval.
§A test for linear trend in rates during 1980–1995.

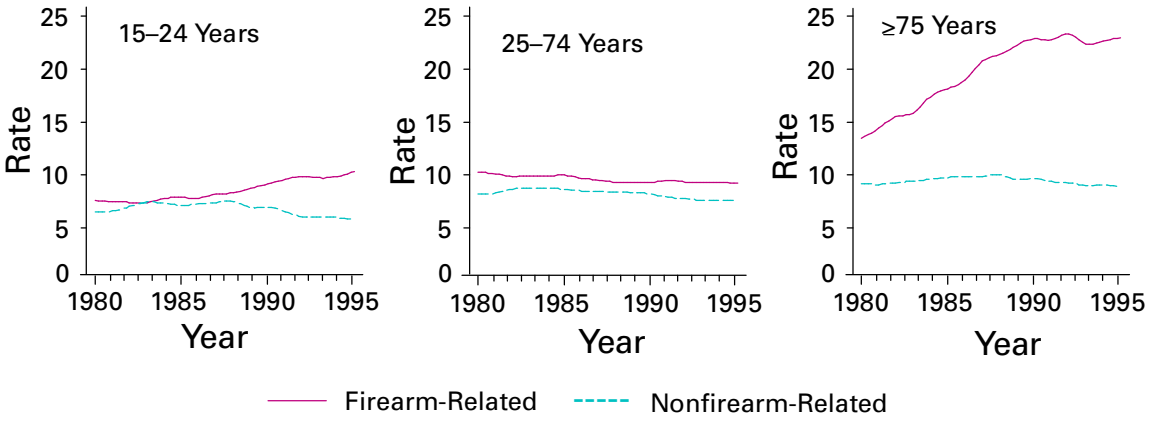
Suicide — Continued

FIGURE 1. Smoothed age-specific suicide rate*, by year — Washington, 1980–1995†



*Per 100,000 population.
†Rates for persons aged <15 years were not plotted because of small numbers.

FIGURE 2. Smoothed age-specific suicide rate*, by year and method — Washington, 1980–1995†



*Per 100,000 population.
†Rates for persons aged <15 years were not plotted because of small numbers.

Suicide — Continued

Editorial Note: The analysis by WDOH illustrates the usefulness of death-certificate data in assessing trends in suicide. Although overall suicide rates remained stable among residents of Washington during 1980–1995, age-specific analyses indicate that the rate of nonfirearm-related suicide increased significantly for children aged 5–14 years, and the rate of firearm-related suicide increased for persons aged 15–24 years and the elderly (aged ≥75 years). Suicide rates for persons aged 25–74 years declined, reflecting a decrease in both firearm-related and nonfirearm-related suicide. These findings can assist in identifying risk factors for suicide and high-risk groups; such analyses should be considered by other state and local health departments to better understand local suicide trends and guide prevention efforts.

The high proportion of firearm-related suicides in Washington is consistent with national patterns during the 1980s and 1990s (*4*). The increases in Washington in the overall rates of suicide for youths and for the elderly and in the rate of firearm-related suicide for persons aged ≥75 years also were consistent with national trends. Although reasons for these increasing trends in suicide are unknown, potential explanations include changes in the prevalence of depression, the use of more lethal methods, and changes in societal attitudes toward suicide among the elderly.

The findings in this analysis may have underestimated the true rate of suicide. The intent of some persons who commit suicide may be unknown or unrecognized; therefore, their deaths may not be reported as suicides. The magnitude of underreporting associated with these misclassification errors is unknown. In contrast, a previous report indicated that coding a nonsuicide death as a suicide probably is uncommon; in that study, 90% of deaths coded as suicides were coded correctly (*5*).

Routine collection of the circumstances of injury events may assist in more accurate coding of suicides on death certificates and in developing effective prevention strategies. In Washington, efforts to improve basic injury data collection include the reporting of firearm injury data to WDOH by all hospitals (admissions and emergency department visits), coroners, and medical examiners. In addition, WDOH is collecting information about the intent and circumstances of shootings and the types of firearms involved.

An important prevention measure for persons who are suicidal is to restrict access to highly lethal methods of suicide (*6*). For example, measures associated with reductions in suicide rates without compensatory increases in the use of other methods include removal of carbon monoxide from domestic gas (*7*), limiting the size of prescriptions to barbituates and other drugs commonly used in self-poisonings (*8*), and restricting access to handguns (*9*). In addition to means restrictions, other interventions for reducing the risk for suicide include 1) training of clergy, tribal leaders, school personnel, health-care professionals, and others who have contact with persons who may be contemplating suicide to recognize persons at risk for suicide and refer them for appropriate counseling; 2) educating the general public about warning signs for suicide and opportunities to seek help; 3) implementing screening programs for identifying and referring persons at highest risk for suicide; 4) improving access to or promoting crisis centers, hotlines, and peer support groups (including family and friends) for high-risk persons; and 5) implementing post-suicide actions to reduce the probability of cluster suicides (*5*). The effectiveness of each of these suicide-prevention strategies requires further assessment.

Suicide — Continued

WDOH, in collaboration with the University of Washington School of Nursing, has developed a Youth Suicide Prevention Plan (*10*) that includes a public education campaign to heighten awareness among adults about the increasing problem of youth suicide and to teach adults how to recognize common suicide warning signs and how to respond to youth who exhibit these signs. In addition, the program provides adults working with high-risk youth with information about effective screening and crisis-intervention strategies. The goals of this plan are to 1) prevent both fatal and nonfatal suicide behaviors among youth; 2) reduce the impact of suicide and suicidal behaviors on individuals, families, and communities; and 3) improve access to and availability of appropriate prevention services for at-risk persons and groups. Although this program is designed to prevent suicide among youths, some elements of the program may be useful to prevent suicide among the elderly.

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Toxigenic *Corynebacterium diphtheriae* — Northern Plains Indian Community, August–October 1996

Diphtheria was one of the most common causes of death among children during the prevaccine era. In 1921, a total of 206,939 cases of diphtheria were reported in the United States (incidence rate: 190 cases per 100,000 population), including 15,520 deaths (case-fatality rate: 7.5%). Since the introduction and widespread use of diphtheria toxoid beginning in the 1920s, respiratory diphtheria has been well controlled in the United States. However, diphtheria remained endemic in some states through the 1970s, with reported incidence rates of >1.0 per million population in six states (Alaska, Arizona, Montana, New Mexico, South Dakota, and Washington) (*1*). Since 1980, only respiratory diphtheria has been reportable in the United States. During 1980–1995, a total of 41 respiratory diphtheria cases were reported (*2*); of these, four (10%) were fatal, and all occurred in unvaccinated children. Five of the six culture-

Toxigenic Corynebacterium diphtheriae — *Continued*

positive diphtheria cases reported in the United States since 1988 have been associated with importation of *Corynebacterium diphtheriae*, an organism believed to have become rare or to have disappeared from the United States. This report describes a case of infection with toxigenic *C. diphtheriae* in an American Indian woman and presents the results of enhanced surveillance for diphtheria in the surrounding community. The findings suggest that *C. diphtheriae* continues to circulate in areas of the United States with previously endemic diphtheria.

Case Report

On June 1, 1996, a 62-year-old American Indian woman with a history of alcoholism and severe necrotizing skin ulcers on both legs was admitted to an Indian Health Service (IHS) hospital in South Dakota for treatment of alcohol intoxication and infected leg ulcers. She was treated with a course of ampicillin and received split-thickness skin grafts on both legs; she was discharged on June 19. A blood culture obtained from the patient on June 1 was sent to a regional reference laboratory, and *C. diphtheriae*, biotype mitis, was identified. At CDC's Diphtheria Laboratory, this isolate demonstrated weak toxigenicity. On admission, the patient's skin ulcers and throat were not swabbed. The patient had received a dose of adult formulation tetanus and diphtheria toxoid vaccine (Td) in 1984 and may have received an additional dose in 1994.

Enhanced Surveillance

In response to isolation of this organism, the South Dakota Department of Health (SDDOH), the Aberdeen Area Office of the IHS, and CDC initiated enhanced surveillance to evaluate the possibility of *C. diphtheriae* infections among other persons in the community where the patient lived. During August 1–October 7, all persons presenting to the IHS hospital and three satellite clinics for evaluation of pharyngitis, draining middle-ear infections, or skin ulcers were cultured for *C. diphtheriae* as part of their routine clinical care.

Specimens were obtained from 133 patients. Of the 133 swabs, 113 (85%) were collected from the oropharynx, 13 (10%) from skin ulcers or wounds, and seven (5%) from ear drainage. *C. diphtheriae* was isolated from the swabs from six (5%) of the 133 patients (Table 1). Ages of the six patients with culture-positive results ranged from 3 to 60 years; four were school-aged children (aged 6–15 years). Three were females. Five of the six patients reported sore throat, and one patient presented with otitis media. In one of the patients with culture-positive results (a 15-year-old female), a pharyngeal membrane was present at the time of her initial presentation. Five patients had been fully vaccinated with diphtheria toxoid, and one 8-year-old child had received three doses of diphtheria and tetanus toxoids and pertussis vaccine (DTP). In addition to *C. diphtheriae*, three patients had culture-positive test results for ß-hemolytic *Streptococcus* (one each of Group A, Group C, and Group G), and one patient had culture-positive test results for *C. pseudodiphtheriticum*. All six patients were treated with penicillin or a cephalosporin.

The primary-care providers of the six patients with culture-positive results were informed about surveillance findings, and local public health nurses and SDDOH staff investigated the household contacts of all these patients. Of the 14 household contacts from whom cultures were obtained, *C. diphtheriae* was isolated from four (29%) (Table 2). Three of the six patients had household contacts who had culture-positive test results. In two households, multiple *C. diphtheriae* biotypes were isolated from

Toxigenic Corynebacterium diphtheriae — *Continued*

TABLE 1. Patients with *Corynebacterium diphtheriae* isolates — Northern Plains Indian community, August–October 1996

Patient	Symptoms	Age (yrs)	Sex	Site of specimen collection	Biotype	Toxigenicity
Index patient	Leg ulcers	61	F	Blood	Mitis	Weakly toxigenic
1	Pharyngitis, labored breathing	3	F	Throat	Mitis	Weakly toxigenic
2	Suppurative otitis media	8	M	Ear	Gravis	Nontoxigenic
3	Exudative pharyngitis	8	M	Throat	Gravis	Toxigenic
4	Pharyngitis with membrane	15	F	Throat	Mitis	Toxigenic
5	Exudative tonsillitis	60	M	Throat	Gravis	Toxigenic
6	Tonsillitis, pharyngitis, fever	7	F	Throat	Gravis	Nontoxigenic

TABLE 2. Household contacts with *Corynebacterium diphtheriae* isolates — Northern Plains Indian community, August–October 1996

Contact	Relation to patients	Age (yrs)	Sex	Site of specimen collection	Biotype	Toxigenicity
1	Mother of patient 1	30	F	Throat	Gravis	Toxigenic
2	Sibling of patient 3	13	F	Throat	Mitis	Weakly toxigenic
3	Sibling of patient 3	11	F	Throat	Mitis	Toxigenic
4	Sibling of patient 4	13	M	Throat	Mitis	Toxigenic

family members. Household contacts received postexposure prophylaxis with penicillin and a dose of diphtheria toxoid-containing vaccine, regardless of their infection status.

Laboratory Results

Of the 10 positive isolates obtained from the six patients and the four household contacts, nine were from throat cultures, and one was from ear drainage. Eight isolates demonstrated toxigenicity by the Elek immunoprecipitation test and by polymerase chain reaction testing (PCR), which can detect both A and B subunits of the diphtheria toxin gene, *tox*. Of the 10 isolates, five were of the biotype mitis, and five were gravis. The toxigenic isolates were assayed by ribotyping and multilocus enzyme electrophoresis and compared with 10 *C. diphtheriae* isolates obtained from other patients in the same area during 1979–1983. Both molecular methods indicated that recent and older isolates from this area were genetically closely related to each other and differed from *C. diphtheriae* strains isolated either from other regions of the United States or from countries of the former Soviet Union affected by the ongoing diphtheria epidemic (*3*).

Reported by: T Welty, MD, C La Fromboise, MPH, J Dixon, DO, A Hurst, MD, D Mulder, DO, M Apostol, M Afraid of Bear, Aberdeen Area Office, Indian Health Service; L Volmer, L Schaeffer, W Anderson, J Judson, S Lance, DVM, State Epidemiologist, South Dakota State Dept of Health. Diphtheria Laboratory, Div of Bacterial and Mycotic Diseases, National Center for Infectious

Toxigenic Corynebacterium diphtheriae — *Continued*

Diseases; Infant Immunization Activity, Child Vaccine Preventable Disease Br, Epidemiology and Surveillance Div, National Immunization Program, CDC.

Editorial Note: During August–October 1996, six (5%) persons in an American Indian community in South Dakota were infected with *C. diphtheriae*; isolates from four of these persons were toxigenic. During 1971–1981, South Dakota had the highest average annual incidence (12.4 cases per million) of diphtheria (*1*). Molecular analysis suggests continuous presence of the organism in this community despite the absence of reported cases since 1976. The presence of two different biotypes in the same household suggests high rates of infection in the population.

The absence of reported cases of respiratory diphtheria in this South Dakota community since the late 1970s suggests a high level of vaccine-related or natural immunity in the population. The extent to which the pharyngitis in these patients was caused by *C. diphtheriae* or by other pathogens cannot be determined. Further evaluations are under way in the community to define factors associated with endemicity of *C. diphtheriae*, assess DTP vaccination coverage among children, and determine seroprevalence of diphtheria antibody among adults.

The presence of toxigenic *C. diphtheriae* in this community underscores the need to reemphasize the importance of timely vaccination against diphtheria among persons of all ages in the United States. Other *Corynebacterium* species may rarely produce diphtheria toxin but still cause a diphtheria-like disease in humans that is preventable through vaccination (*4*). Completing the routinely recommended childhood vaccination series for DTP (i.e., five doses at the recommended ages) and achieving high vaccination levels (>90%) among preschool-aged children is of particular importance in this community and in other communities where diphtheria was previously endemic. In addition, booster doses of Td vaccine every 10 years are recommended throughout adulthood. Efforts are under way to educate the public and health-care providers about the importance of vaccinations. Finally, surveillance should be enhanced in areas where diphtheria was previously endemic. Clinicians should consider diphtheria in the differential diagnosis of patients presenting with a sore throat; low-grade fever; and an adherent membrane of the tonsil(s), pharynx, and/or nose. Because the successful isolation of *C. diphtheriae* depends on rapid inoculation of special culture media, the laboratory should be notified as soon as the diagnosis is suspected. Whenever a diagnosis of diphtheria is strongly suspected, local public health officials should be notified immediately, and measures to prevent additional cases should be instituted (*5*).

As of January 1997, diphtheria antitoxin is no longer commercially available in the United States but may be obtained for treatment of suspected cases of diphtheria through the medical epidemiology staff of CDC's Child Vaccine Preventable Disease Branch, Epidemiology and Surveillance Division, National Immunization Program, telephone (404) 639-2889 (*6*).

References

1. Chen RT, Broome CV, Weinstein RA, Weaver R, Tsai TF. Diphtheria in the United States, 1971–81. Am J Public Health 1985;75:1393–7.
2. Bisgard KM, Hardy IRB, Popovic T, Strebel PM, Wharton M, Hadler SC. Virtual elimination of respiratory diphtheria in the United States [Abstract no. G12]. In: Abstracts of the 36th Interscience Conference on Antimicrobial Agents and Chemotherapy. Washington, DC: American Society for Microbiology, 1995:160.

Toxigenic Corynebacterium diphtheriae — *Continued*

3. CDC. Update: diphtheria epidemic—New Independent States of the Former Soviet Union, January 1995–March 1996. MMWR 1996;45:693–7.
4. CDC. Respiratory diphtheria caused by *Corynebacterium ulcerans*—Terre Haute, Indiana, 1996. MMWR 1997;46:330–6.
5. Farizo KM, Strebel PM, Chen RT, Kimbler A, Cleary TJ, Cochi SL. Fatal respiratory disease due to *Corynebacterium diphtheriae*: case report and review of guidelines for management, investigation, and control. Clin Infect Dis 1993;16:59–68.
6. CDC. Availability of diphtheria antitoxin through an investigational new drug protocol. MMWR 1997;46:380.

Notice to Readers

Publication of HIV-Prevention Bulletin for Health-Care Providers Regarding Advice to Persons Who Inject Illicit Drugs

Persons who inject illicit drugs are at risk for acquiring and transmitting bloodborne infections, including human immunodeficiency virus, hepatitis B, and hepatitis C. CDC, the Health Resources and Services Administration, the National Institute on Drug Abuse of the National Institutes of Health, and the Substance Abuse and Mental Health Services Administration have released a bulletin for health-care providers that contains the following provisional recommendations for persons who continue to inject illicit drugs:

1. Stop using and injecting drugs.
2. Enter and complete substance-abuse treatment, including relapse prevention.
3. If use of illicit injected drugs continues, take the following steps to reduce personal health risks and public health risks:
   a. Never reuse or "share" syringes, water, or drug-preparation equipment.
   b. Use only syringes obtained from a safe, reliable source (e.g., pharmacies).
   c. Use a new, sterile syringe to prepare and inject drugs.
   d. If possible, use sterile water to prepare drugs; otherwise use clean water from a reliable source (such as fresh tap water).
   e. Use a new or disinfected container ("cooker") and a new filter ("cotton") to prepare drugs.
   f. Clean the injection site before injection with a new alcohol swab.
   g. Safely dispose of syringes after one use.

Single copies of the bulletin are available free of charge from the CDC National AIDS Clearinghouse, telephone (800) 458-5231 or (301) 217-0023. The bulletin is also available on the World-Wide Web at http://www.cdc.gov/nchstp/hiv_aids/pubs.htm.

Notice to Readers

Satellite Videoconference on Vancomycin-Resistant Enterococci

Vancomycin-Resistant Enterococci (VRE): Control of an Emerging Pathogen, a live satellite videoconference, will be broadcast Thursday, September 25, 1997, from 1 p.m. to 3:30 p.m. eastern daylight time. Cosponsors are CDC and the Public Health Training Network. This course is designed for physicians; nurses; infection-control

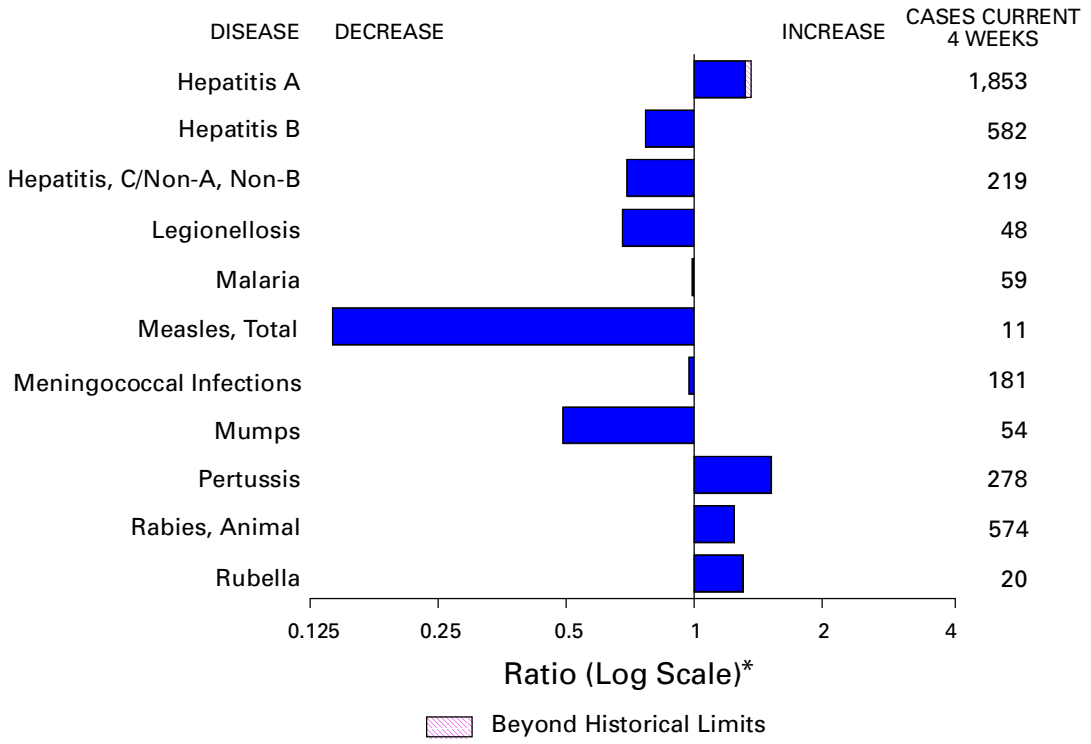
Notices to Readers — Continued

professionals; pharmacists; laboratorians; hospital administrators; and others who are involved with the detection, treatment, and control of VRE.

This course will provide an overview of the threat VRE infections cause to patients in health-care facilities. Participants will learn the methods of VRE detection, isolation, treatment, and prevention. In addition, experts will describe the magnitude, clinical impact, transmission methods, and risk factors for VRE. Continuing education credits will be awarded.

Registration information is available through the CDC fax information system, telephone (888) 232-3299 (CDC-FAXX), by requesting document number 564032.

FIGURE I. Selected notifiable disease reports, comparison of provisional 4-week totals ending May 31, 1997, with historical data — United States



*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 May 31, 1997 (22nd Week)

	Cum. 1997		Cum. 1997
Anthrax	-	Plague	1
Brucellosis	18	Poliomyelitis, paralytic	-
Cholera	2	Psittacosis	16
Congenital rubella syndrome	2	Rabies, human	2
Cryptosporidiosis*	470	Rocky Mountain spotted fever (RMSF)	57
Diphtheria	4	Streptococcal disease, invasive Group A	567
Encephalitis: California*	4	Streptococcal toxic-shock syndrome*	15
eastern equine*	-	Syphilis, congenital¶	62
St. Louis*	1	Tetanus	15
western equine*	-	Toxic-shock syndrome	45
Hansen Disease	45	Trichinosis	3
Hantavirus pulmonary syndrome*†	5	Typhoid fever	114
Hemolytic uremic syndrome, post-diarrheal*	17	Yellow fever	-
HIV infection, pediatric*§	92		

-:no reported cases
*Not notifiable in all states.
†Updated weekly from reports to the Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases (NCID).
§Updated monthly to the Division of HIV/AIDS Prevention–Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP), last update April 29, 1997.
¶Updated from reports to the Division of STD Prevention, NCHSTP.

TABLE II. Provisional cases of selected notifiable diseases, United States, weeks ending May 31, 1997, and June 1, 1996 (22nd Week)

Reporting Area	AIDS		Chlamydia		*Escherichia coli* O157:H7		Gonorrhea		Hepatitis C/NA,NB	
					NETSS†	PHLIS§				
	Cum. 1997*	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1997	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996
UNITED STATES	20,222	28,381	164,261	178,943	449	181	103,401	127,731	1,229	1,309
NEW ENGLAND	671	1,115	6,819	6,760	36	16	2,253	2,590	21	42
Maine	25	16	396	U	1	-	25	21	-	-
N.H.	8	31	305	310	2	-	53	62	4	2
Vt.	16	9	167	189	3	1	24	26	-	13
Mass.	282	549	3,008	2,693	26	15	938	879	14	24
R.I.	55	73	884	877	1	-	207	219	3	3
Conn.	285	437	2,059	2,691	3	-	1,006	1,383	-	-
MID. ATLANTIC	6,683	7,897	20,730	29,424	31	4	12,151	18,081	139	113
Upstate N.Y.	1,143	1,002	N	N	18	3	2,243	3,178	110	86
N.Y. City	3,308	4,490	10,958	15,654	5	-	4,826	6,937	-	2
N.J.	1,444	1,509	2,896	6,019	8	-	1,719	3,707	-	-
Pa.	788	896	6,876	7,751	N	1	3,363	4,259	29	25
E.N. CENTRAL	1,416	2,301	24,977	37,062	77	27	15,333	23,903	242	220
Ohio	270	526	5,639	8,522	24	10	3,615	5,969	7	4
Ind.	302	342	3,475	4,150	14	5	2,398	2,777	6	6
Ill.	509	984	4,703	10,414	19	-	2,302	6,859	20	47
Mich.	259	318	8,144	9,371	20	4	5,720	6,255	209	163
Wis.	76	131	3,016	4,605	N	8	1,298	2,043	-	-
W.N. CENTRAL	383	679	9,446	13,580	56	39	4,423	6,754	75	33
Minn.	79	126	U	2,293	29	20	U	1,659	2	-
Iowa	59	51	1,895	1,746	13	8	488	445	23	13
Mo.	150	320	4,692	5,779	5	8	3,092	3,461	32	11
N. Dak.	4	7	345	425	3	2	23	12	2	-
S. Dak.	2	7	509	569	1	-	53	80	-	-
Nebr.	35	49	421	822	3	-	119	166	1	4
Kans.	54	119	1,584	1,946	2	1	648	931	15	5
S. ATLANTIC	4,846	7,253	35,094	23,341	58	13	34,391	40,060	118	73
Del.	69	142	-	-	1	1	457	614	-	-
Md.	576	850	2,983	2,602	3	1	5,505	5,298	7	1
D.C.	282	456	N	N	-	-	1,319	64	-	-
Va.	421	395	4,738	4,954	N	5	3,342	4,092	8	7
W. Va.	27	50	1,411	889	N	-	409	301	7	7
N.C.	281	360	6,995	U	17	6	6,567	8,095	25	19
S.C.	270	383	5,173	U	1	-	4,621	4,672	19	14
Ga.	683	1,085	3,982	5,060	15	-	5,263	9,382	U	-
Fla.	2,237	3,532	9,812	9,787	21	-	6,908	7,542	52	25
E.S. CENTRAL	609	951	13,417	12,492	37	7	13,220	13,031	152	267
Ky.	60	153	2,754	2,910	11	-	1,628	1,731	8	13
Tenn.	285	352	5,134	5,385	19	7	4,275	4,642	89	219
Ala.	151	277	3,339	3,556	4	-	4,692	5,426	5	2
Miss.	113	169	2,190	641	3	-	2,625	1,232	50	33
W.S. CENTRAL	2,040	2,638	20,702	8,979	24	4	13,545	8,530	130	110
Ark.	83	121	519	685	2	1	1,044	1,710	-	3
La.	385	649	3,346	2,926	3	3	3,131	3,135	83	71
Okla.	116	100	3,096	2,935	1	-	1,948	1,857	4	1
Tex.	1,456	1,768	13,741	2,433	18	-	7,422	1,828	43	35
MOUNTAIN	601	797	10,293	15,241	48	25	2,988	4,826	155	273
Mont.	16	10	350	530	3	-	17	13	7	9
Idaho	18	19	616	654	11	1	45	38	21	70
Wyo.	11	2	223	297	4	-	25	13	60	85
Colo.	156	245	1,896	793	16	8	751	717	18	25
N. Mex.	58	45	1,477	1,669	4	3	547	366	27	35
Ariz.	158	233	3,922	9,460	N	10	1,199	3,196	16	27
Utah	41	88	685	628	7	-	88	122	3	11
Nev.	143	155	1,124	1,210	3	3	316	361	3	11
PACIFIC	2,973	4,750	22,783	32,064	82	43	5,097	9,956	197	178
Wash.	241	362	3,823	4,283	17	4	812	965	11	26
Oreg.	128	223	1,501	2,360	27	15	235	201	4	4
Calif.	2,570	4,074	16,257	24,264	35	21	3,690	8,374	113	65
Alaska	12	11	566	391	3	-	182	191	-	2
Hawaii	22	80	636	766	N	3	178	225	69	81
Guam	2	3	31	188	N	-	3	31	-	5
P.R.	520	423	N	N	22	U	258	212	45	72
V.I.	29	9	N	N	N	U	-	-	-	-
Amer. Samoa	-	-	-	-	N	U	-	-	-	-
C.N.M.I.	-	-	N	N	N	U	11	11	2	-

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

*Updated monthly to the Division of HIV/AIDS Prevention–Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention, last update April 29, 1997.

†National Electronic Telecommunications System for Surveillance.

§Public Health Laboratory Information System.

TABLE II. (Cont'd.) Provisional cases of selected notifiable diseases, United States, weeks ending May 31, 1997, and June 1, 1996 (22nd Week)

Reporting Area	Legionellosis		Lyme Disease		Malaria		Syphilis (Primary & Secondary)		Tuberculosis		Rabies, Animal
	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997
UNITED STATES	341	308	1,098	1,787	483	485	3,366	4,967	5,721	7,293	2,986
NEW ENGLAND	24	17	230	255	17	14	62	69	169	158	467
Maine	1	1	3	3	1	3	-	-	11	11	94
N.H.	4	-	7	4	1	1	-	1	6	5	20
Vt.	3	2	3	-	2	2	-	-	3	-	80
Mass.	8	8	54	16	11	5	35	32	88	60	91
R.I.	4	6	37	21	2	3	-	1	13	20	9
Conn.	4	N	126	211	-	-	27	35	48	62	173
MID. ATLANTIC	56	66	669	1,334	114	145	154	226	1,265	1,266	644
Upstate N.Y.	13	14	106	638	24	28	16	35	177	138	477
N.Y. City	-	4	6	66	55	80	33	71	670	669	-
N.J.	7	7	139	173	24	28	61	74	255	282	61
Pa.	36	41	418	457	11	9	44	46	163	177	106
E.N. CENTRAL	123	113	22	18	34	61	291	833	652	806	58
Ohio	69	40	17	9	6	6	100	328	136	111	43
Ind.	18	29	5	6	4	6	71	108	64	81	8
Ill.	-	14	-	3	5	28	25	237	302	455	2
Mich.	31	20	-	-	16	11	45	71	102	125	5
Wis.	5	10	U	U	3	10	50	89	48	34	-
W.N. CENTRAL	31	18	11	42	16	13	54	187	192	206	180
Minn.	1	1	9	1	5	3	-	22	48	50	16
Iowa	9	2	-	5	8	2	3	13	20	25	69
Mo.	5	4	-	17	2	6	34	136	83	81	9
N. Dak.	2	-	-	-	-	-	-	-	4	2	22
S. Dak.	1	2	-	-	-	-	-	-	4	13	24
Nebr.	9	7	2	-	1	-	1	6	4	13	1
Kans.	4	2	-	19	-	2	16	10	29	22	39
S. ATLANTIC	53	35	106	71	122	80	1,399	1,631	1,267	1,320	1,265
Del.	4	2	1	38	2	2	11	16	7	22	29
Md.	16	5	79	6	36	21	391	263	124	107	222
D.C.	2	2	5	1	6	4	41	8	36	58	2
Va.	9	11	-	-	24	11	116	205	111	118	261
W. Va.	-	1	-	4	-	1	1	2	22	24	33
N.C.	6	3	7	16	6	10	303	471	149	159	393
S.C.	2	3	1	2	8	3	178	203	138	151	57
Ga.	-	-	1	-	12	8	229	298	232	270	128
Fla.	14	8	12	4	28	20	129	165	448	411	140
E.S. CENTRAL	10	19	27	24	14	13	774	1,163	450	571	112
Ky.	-	2	4	7	3	3	68	62	82	100	11
Tenn.	5	8	10	6	4	5	330	385	120	184	73
Ala.	1	2	2	1	4	2	206	241	173	185	28
Miss.	4	7	11	10	3	3	170	475	75	102	-
W.S. CENTRAL	6	2	9	11	5	11	466	495	140	841	119
Ark.	-	-	-	6	1	-	58	120	80	80	19
La.	1	-	1	-	4	1	174	236	-	3	1
Okla.	2	2	3	2	-	-	52	65	60	57	52
Tex.	3	-	5	3	-	10	182	74	-	701	47
MOUNTAIN	22	18	3	1	30	27	68	95	222	233	42
Mont.	1	1	-	-	2	2	-	-	7	7	8
Idaho	2	-	-	-	-	-	-	1	5	4	-
Wyo.	1	2	1	1	1	2	-	1	2	3	13
Colo.	4	6	1	-	15	14	2	17	44	43	-
N. Mex.	1	-	-	-	4	1	-	-	8	37	4
Ariz.	7	4	1	-	4	3	56	72	101	90	16
Utah	5	1	-	-	1	3	3	-	10	10	-
Nev.	1	4	-	-	3	2	7	4	45	39	1
PACIFIC	16	20	21	31	131	121	98	268	1,364	1,892	99
Wash.	4	1	1	1	8	8	6	3	87	113	-
Oreg.	-	-	8	9	10	8	3	4	60	76	1
Calif.	11	19	12	20	109	99	87	260	1,109	1,596	81
Alaska	-	-	-	-	2	2	1	-	36	39	17
Hawaii	1	-	-	1	2	4	1	1	72	68	-
Guam	-	1	-	-	-	-	-	3	5	45	-
P.R.	-	-	-	-	3	-	94	113	88	38	25
V.I.	-	-	-	-	-	-	-	-	-	-	-
Amer. Samoa	-	-	-	-	-	-	-	-	-	-	-
C.N.M.I.	-	-	-	-	-	-	4	1	-	-	-

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

TABLE III. Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending May 31, 1997, and June 1, 1996 (22nd Week)

Reporting Area	*H. influenzae*, invasive		Hepatitis (Viral), by type				Measles (Rubeola)					
			A		B		Indigenous		Imported†		Total	
	Cum. 1997*	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	1997	Cum. 1997	1997	Cum. 1997	Cum. 1997	Cum. 1996
UNITED STATES	482	519	11,217	11,032	3,418	3,773	3	34	2	17	51	179
NEW ENGLAND	28	13	243	133	73	85	-	-	-	-	-	10
Maine	3	-	37	11	5	2	-	-	-	-	-	-
N.H.	2	8	16	5	5	7	-	-	-	-	-	-
Vt.	-	-	6	3	2	6	-	-	-	-	-	1
Mass.	20	5	102	64	42	21	-	-	-	-	-	8
R.I.	2	-	24	5	8	5	-	-	-	-	-	-
Conn.	1	-	58	45	11	44	-	-	-	-	-	1
MID. ATLANTIC	53	104	795	746	451	623	-	7	-	4	11	12
Upstate N.Y.	3	26	107	160	94	137	-	1	-	3	4	4
N.Y. City	17	23	274	262	144	241	-	4	-	1	5	7
N.J.	23	30	146	160	104	121	-	1	-	-	1	-
Pa.	10	25	268	164	109	124	-	1	-	-	1	1
E.N. CENTRAL	67	86	1,188	1,050	383	471	-	4	-	2	6	12
Ohio	41	49	179	412	41	53	-	-	-	-	-	2
Ind.	8	4	130	143	40	63	-	-	-	-	-	-
Ill.	11	23	234	243	82	135	-	4	-	1	5	2
Mich.	6	5	580	154	206	178	-	-	-	1	1	2
Wis.	1	5	65	98	14	42	-	-	-	-	-	6
W.N. CENTRAL	21	18	813	829	219	192	-	9	1	2	11	15
Minn.	12	10	70	37	18	13	-	-	1	2	2	14
Iowa	2	3	113	179	32	21	-	-	-	-	-	-
Mo.	3	3	430	428	145	124	-	1	-	-	1	1
N. Dak.	-	-	9	22	1	-	-	-	-	-	-	-
S. Dak.	2	1	12	36	-	-	-	8	-	-	8	-
Nebr.	1	1	56	62	9	14	-	-	-	-	-	-
Kans.	1	-	123	65	14	20	-	-	-	-	-	-
S. ATLANTIC	107	95	671	404	485	471	-	1	1	3	4	4
Del.	-	1	11	6	2	2	-	-	-	-	-	1
Md.	39	32	121	86	75	73	-	-	-	1	1	-
D.C.	2	5	13	15	20	15	-	-	-	1	1	-
Va.	6	4	74	65	50	62	-	-	-	-	-	2
W. Va.	3	4	6	10	6	14	-	-	-	-	-	-
N.C.	15	14	94	51	94	129	-	-	1	1	1	-
S.C.	4	3	55	29	42	39	-	-	-	-	-	-
Ga.	18	26	117	15	47	7	-	-	-	-	-	-
Fla.	20	6	180	127	149	130	-	1	-	-	1	1
E.S. CENTRAL	33	18	292	742	297	361	-	-	-	-	-	-
Ky.	5	5	33	16	13	36	-	-	-	-	-	-
Tenn.	20	7	176	524	185	214	-	-	-	-	-	-
Ala.	8	5	48	96	31	24	-	-	-	-	-	-
Miss.	-	1	35	106	68	U	U	-	U	-	-	-
W.S. CENTRAL	23	20	2,463	1,779	441	327	-	3	-	1	4	2
Ark.	1	-	125	223	24	35	-	-	-	-	-	-
La.	3	1	87	58	46	50	-	-	-	-	-	-
Okla.	14	18	728	811	14	20	-	-	-	-	-	-
Tex.	5	1	1,523	687	357	222	-	3	-	1	4	2
MOUNTAIN	44	27	1,751	1,738	395	462	3	5	-	-	5	16
Mont.	-	-	47	60	5	4	-	-	-	-	-	-
Idaho	1	1	72	126	16	56	-	-	-	-	-	1
Wyo.	-	-	18	19	17	14	-	-	-	-	-	-
Colo.	6	5	198	159	84	55	-	-	-	-	-	5
N. Mex.	4	7	137	220	138	156	-	-	-	-	-	-
Ariz.	14	9	856	610	80	100	3	5	-	-	5	3
Utah	3	5	318	389	38	53	-	-	-	-	-	3
Nev.	16	-	105	155	17	24	U	-	U	-	-	4
PACIFIC	106	138	3,001	3,611	674	781	-	5	-	5	10	108
Wash.	2	1	220	237	30	47	-	-	-	-	-	36
Oreg.	19	19	162	509	51	51	-	-	-	-	-	4
Calif.	79	112	2,541	2,798	575	679	-	2	-	5	7	3
Alaska	1	4	19	28	12	2	-	-	-	-	-	63
Hawaii	5	2	59	39	6	2	-	3	-	-	3	2
Guam	-	-	-	3	1	-	U	-	U	-	-	-
P.R.	-	1	153	94	539	432	-	-	-	-	-	1
V.I.	-	-	-	-	-	-	U	-	U	-	-	-
Amer. Samoa	-	-	-	-	-	-	U	-	U	-	-	-
C.N.M.I.	4	10	1	1	19	5	U	1	U	-	1	-

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

*Of 102 cases among children aged <5 years, serotype was reported for 50 and of those, 19 were type b.

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

TABLE III. (Cont'd.) Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending May 31, 1997, and June 1, 1996 (22nd Week)

Reporting Area	Meningococcal Disease		Mumps			Pertussis			Rubella		
	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996
UNITED STATES	1,700	1,622	6	270	302	64	2,100	1,445	15	39	98
NEW ENGLAND	106	62	-	7	-	-	455	309	-	-	23
Maine	10	8	-	-	-	-	6	10	-	-	-
N.H.	9	1	-	-	-	-	58	17	-	-	-
Vt.	2	3	-	-	-	-	160	7	-	-	2
Mass.	57	21	-	2	-	-	214	272	-	-	19
R.I.	7	7	-	4	-	-	12	-	-	-	-
Conn.	21	22	-	1	-	-	5	3	-	-	2
MID. ATLANTIC	147	175	2	26	46	3	163	99	-	4	5
Upstate N.Y.	42	39	-	4	11	-	52	51	-	2	3
N.Y. City	24	26	-	-	13	1	40	16	-	2	1
N.J.	31	35	-	-	2	-	5	4	-	-	1
Pa.	50	75	2	22	20	2	66	28	-	-	-
E.N. CENTRAL	233	238	1	30	77	3	157	204	-	2	3
Ohio	98	81	1	13	27	2	65	69	-	-	-
Ind.	30	33	-	4	5	-	22	12	-	-	-
Ill.	67	71	-	7	15	1	24	53	-	-	1
Mich.	20	27	-	6	29	-	26	12	-	-	2
Wis.	18	26	-	-	1	-	20	58	-	2	-
W.N. CENTRAL	122	123	-	9	4	7	115	61	-	-	-
Minn.	12	14	-	3	1	7	74	40	-	-	-
Iowa	26	25	-	4	-	-	15	2	-	-	-
Mo.	63	52	-	-	1	-	16	12	-	-	-
N. Dak.	1	2	-	-	2	-	2	-	-	-	-
S. Dak.	4	4	-	-	-	-	1	1	-	-	-
Nebr.	5	12	-	2	-	-	2	2	-	-	-
Kans.	11	14	-	-	-	-	5	4	-	-	-
S. ATLANTIC	315	247	1	40	34	7	189	122	11	13	12
Del.	4	2	-	-	-	-	-	11	-	-	-
Md.	31	27	-	4	15	-	69	53	-	-	-
D.C.	1	6	-	-	-	-	2	-	-	-	1
Va.	27	30	-	4	3	-	19	5	-	1	-
W. Va.	12	10	-	-	-	1	4	2	-	-	-
N.C.	54	40	1	7	-	5	40	24	10	10	-
S.C.	40	33	-	9	5	-	8	1	-	1	1
Ga.	63	72	-	4	2	-	7	7	-	-	-
Fla.	83	27	-	12	9	1	40	19	1	1	10
E.S. CENTRAL	131	121	1	15	11	1	36	130	-	-	-
Ky.	34	19	-	2	-	-	2	112	-	-	-
Tenn.	45	36	-	3	1	-	14	11	-	-	-
Ala.	36	34	1	6	3	1	12	4	-	-	-
Miss.	16	32	U	4	7	U	8	3	U	-	N
W.S. CENTRAL	170	187	-	29	24	-	32	43	-	4	7
Ark.	24	26	-	-	-	-	5	2	-	-	-
La.	29	35	-	7	10	-	7	4	-	-	1
Okla.	22	18	-	-	-	-	5	4	-	-	-
Tex.	95	108	-	22	14	-	15	33	-	4	6
MOUNTAIN	104	101	-	34	13	25	648	155	2	4	5
Mont.	8	4	-	-	-	1	6	5	-	-	-
Idaho	7	12	-	2	-	15	480	56	1	1	2
Wyo.	-	3	-	1	-	-	4	-	-	-	-
Colo.	30	15	-	3	1	2	111	26	-	-	1
N. Mex.	18	20	N	N	N	6	31	29	-	-	-
Ariz.	23	26	-	22	1	-	10	11	1	3	1
Utah	12	9	-	4	2	1	4	5	-	-	-
Nev.	6	12	U	2	9	U	2	23	U	-	1
PACIFIC	372	368	1	80	93	18	305	322	2	12	43
Wash.	49	48	1	11	9	5	159	146	-	-	8
Oreg.	80	69	-	1	-	1	16	30	-	-	1
Calif.	240	245	-	57	68	12	123	135	2	7	32
Alaska	1	4	-	2	2	-	1	1	-	-	-
Hawaii	2	2	-	9	14	-	6	10	-	5	2
Guam	-	1	U	1	4	U	-	-	U	-	-
P.R.	8	8	-	4	1	-	-	2	-	-	-
V.I.	-	-	U	-	-	U	-	-	U	-	-
Amer. Samoa	-	-	U	-	-	U	-	-	U	-	-
C.N.M.I.	-	-	U	1	-	U	-	-	U	-	-

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

TABLE IV. Deaths in 122 U.S. cities,* week ending May 31, 1997 (22nd Week)

Reporting Area	All Causes, By Age (Years)						P&I† Total
	All Ages	>65	45-64	25-44	1-24	<1	
NEW ENGLAND	590	414	104	41	15	16	53
Boston, Mass.	144	86	33	12	7	6	20
Bridgeport, Conn.	41	31	7	2	1	-	3
Cambridge, Mass.	11	9	2	-	-	-	1
Fall River, Mass.	21	18	1	2	-	-	3
Hartford, Conn.	65	50	13	1	1	-	3
Lowell, Mass.	26	17	6	3	-	-	1
Lynn, Mass.	9	5	2	1	-	1	-
New Bedford, Mass.	24	22	2	-	-	-	1
New Haven, Conn.	46	31	6	5	1	3	1
Providence, R.I.	73	51	12	8	-	2	7
Somerville, Mass.	7	6	1	-	-	-	-
Springfield, Mass.	33	19	8	2	3	1	2
Waterbury, Conn.	31	25	4	1	-	1	3
Worcester, Mass.	59	44	7	4	2	2	8
MID. ATLANTIC	2,236	1,532	424	184	49	44	140
Albany, N.Y.	41	29	7	1	3	1	3
Allentown, Pa.	32	26	6	-	-	-	1
Buffalo, N.Y.	U	U	U	U	U	U	U
Camden, N.J.	36	18	11	2	4	1	2
Elizabeth, N.J.	16	14	2	-	-	-	-
Erie, Pa.	30	19	7	2	-	2	1
Jersey City, N.J.	40	25	13	2	-	-	2
New York City, N.Y.	1,126	776	216	98	20	16	49
Newark, N.J.	79	34	22	17	5	1	5
Paterson, N.J.	19	16	2	-	1	-	-
Philadelphia, Pa.	434	287	77	40	10	17	34
Pittsburgh, Pa.§	68	49	13	3	-	3	9
Reading, Pa.	22	17	1	1	3	-	3
Rochester, N.Y.	114	81	20	8	3	2	13
Schenectady, N.Y.	25	18	4	3	-	-	-
Scranton, Pa.	30	28	1	1	-	-	1
Syracuse, N.Y.	61	44	14	2	-	1	12
Trenton, N.J.	14	13	-	1	-	-	-
Utica, N.Y.	17	16	-	1	-	-	1
Yonkers, N.Y.	32	22	8	2	-	-	4
E.N. CENTRAL	1,936	1,330	356	149	57	41	133
Akron, Ohio	34	25	4	3	1	1	-
Canton, Ohio	41	30	8	3	-	-	4
Chicago, Ill.	442	270	79	62	18	12	34
Cincinnati, Ohio	115	82	17	6	5	5	21
Cleveland, Ohio	115	81	24	6	4	-	3
Columbus, Ohio	150	93	38	13	3	3	6
Dayton, Ohio	95	76	12	4	3	-	8
Detroit, Mich.	192	122	45	15	7	3	4
Evansville, Ind.	28	19	4	5	-	-	2
Fort Wayne, Ind.	42	33	7	2	-	-	3
Gary, Ind.	10	7	1	-	-	-	-
Grand Rapids, Mich.	60	45	13	1	-	1	6
Indianapolis, Ind.	188	128	41	9	6	4	13
Lansing, Mich.	42	27	9	-	2	4	4
Milwaukee, Wis.	109	87	15	3	2	2	10
Peoria, Ill.	35	28	2	4	-	1	-
Rockford, Ill.	48	31	8	6	2	1	1
South Bend, Ind.	49	40	6	1	-	2	4
Toledo, Ohio	85	66	13	5	1	-	7
Youngstown, Ohio	56	40	10	1	3	2	3
W.N. CENTRAL	601	424	111	28	11	14	40
Des Moines, Iowa	14	6	2	5	1	-	1
Duluth, Minn.	21	17	3	1	-	-	2
Kansas City, Kans.	21	15	3	-	-	3	-
Kansas City, Mo.	119	74	20	8	1	3	6
Lincoln, Nebr.	24	20	4	-	-	-	2
Minneapolis, Minn.	130	93	26	5	2	4	14
Omaha, Nebr.	64	41	19	-	2	2	4
St. Louis, Mo.	91	71	13	5	2	-	-
St. Paul, Minn.	50	38	6	2	2	2	5
Wichita, Kans.	67	49	15	2	1	-	6
S. ATLANTIC	1,135	715	227	125	40	26	60
Atlanta, Ga.	128	76	35	13	1	3	7
Baltimore, Md.	217	131	35	35	10	6	15
Charlotte, N.C.	90	59	21	7	2	1	3
Jacksonville, Fla.	107	70	18	13	3	3	2
Miami, Fla.	93	52	19	14	5	3	-
Norfolk, Va.	31	25	2	2	2	-	4
Richmond, Va.	53	39	8	4	2	-	4
Savannah, Ga.	48	37	8	-	2	1	7
St. Petersburg, Fla.	43	32	6	5	-	-	7
Tampa, Fla.	165	108	32	11	9	4	10
Washington, D.C.	147	76	41	21	4	5	1
Wilmington, Del.	13	10	2	-	-	-	-
E.S. CENTRAL	727	492	154	48	21	12	58
Birmingham, Ala.	121	86	19	9	5	2	9
Chattanooga, Tenn.	64	46	11	3	3	1	6
Knoxville, Tenn.	86	61	15	7	2	1	8
Lexington, Ky.	48	37	9	1	-	1	3
Memphis, Tenn.	199	133	47	9	6	4	24
Mobile, Ala.	51	31	11	8	1	-	-
Montgomery, Ala.	35	26	5	4	-	-	6
Nashville, Tenn.	123	72	37	7	4	3	2
W.S. CENTRAL	1,335	845	292	127	46	24	81
Austin, Tex.	77	56	9	9	3	-	5
Baton Rouge, La.	15	11	2	2	-	-	1
Corpus Christi, Tex.	44	23	12	3	2	3	7
Dallas, Tex.	161	92	41	20	5	3	3
El Paso, Tex.	85	53	20	8	2	2	3
Ft. Worth, Tex.	103	69	19	8	4	3	7
Houston, Tex.	400	235	101	41	16	7	29
Little Rock, Ark.	59	34	17	6	2	-	6
New Orleans, La.	83	48	21	9	4	1	-
San Antonio, Tex.	192	135	34	12	8	3	6
Shreveport, La.	40	29	6	4	-	1	6
Tulsa, Okla.	76	60	10	5	-	1	8
MOUNTAIN	788	507	158	70	40	13	38
Albuquerque, N.M.	101	58	29	8	5	1	2
Boise, Idaho	25	17	4	2	2	-	1
Colo. Springs, Colo.	42	35	4	2	1	-	-
Denver, Colo.	82	49	14	8	6	5	10
Las Vegas, Nev.	150	97	29	19	5	-	8
Ogden, Utah	41	30	8	2	1	-	3
Phoenix, Ariz.	133	78	32	11	8	4	6
Pueblo, Colo.	26	23	2	1	-	-	1
Salt Lake City, Utah	90	56	16	8	8	2	5
Tucson, Ariz.	98	64	20	9	4	1	2
PACIFIC	1,373	960	218	110	56	29	118
Berkeley, Calif.	20	13	3	3	-	1	-
Fresno, Calif.	51	28	12	3	4	4	3
Glendale, Calif.	14	11	1	1	1	-	2
Honolulu, Hawaii	57	37	14	5	-	1	6
Long Beach, Calif.	86	64	13	6	1	2	13
Los Angeles, Calif.	289	206	45	26	9	3	13
Pasadena, Calif.	25	21	2	1	-	1	2
Portland, Oreg.	107	75	16	6	5	5	11
Sacramento, Calif.	192	126	34	19	11	2	26
San Diego, Calif.	129	82	22	10	9	6	16
San Francisco, Calif.	U	U	U	U	U	U	U
San Jose, Calif.	144	103	21	12	5	3	12
Santa Cruz, Calif.	25	20	3	1	1	-	3
Seattle, Wash.	106	72	17	10	7	-	2
Spokane, Wash.	49	41	6	1	-	1	4
Tacoma, Wash.	79	61	9	6	3	-	5
TOTAL	10,721¶	7,219	2,044	882	335	219	721

U: Unavailable -: no reported cases

*Mortality data in this table are voluntarily reported from 122 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

†Pneumonia and influenza.

§Because of changes in reporting methods in this Pennsylvania city, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

¶Total includes unknown ages.

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