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Update: Outbreaks of Cyclosporiasis — United States and Canada, 1997

Since April 1997, CDC has received reports of outbreaks of cyclosporiasis in the United States and Canada (*1,2*). As of June 11, there have been 21 clusters of cases of cyclosporiasis reported from eight states (California, Florida, Maryland, Nebraska, Nevada, New York, Rhode Island, and Texas) and one province in Canada (Ontario). These clusters were associated with events (e.g., receptions, banquets, or time-place–related exposures [meals in the same restaurant on the same day]) that occurred during March 19–May 25 and comprise approximately 140 laboratory-confirmed and 370 clinically defined cases of cyclosporiasis. In addition, four laboratory-confirmed and approximately 220 clinically defined cases have been reported among persons who, during March 29–April 5, were on a cruise ship that departed from Florida. Approximately 70 laboratory-confirmed sporadic cases (i.e., cases not associated with events, the cruise, or recent overseas travel) have been reported in the United States and Canada. The most recent laboratory-confirmed sporadic case occurred in a person who had onset of symptoms on June 3.

Fresh raspberries were served at 19 of the 21 events and were the only food in common to all 19 events, which occurred in April and May. At six of the 19 events, raspberries were the only type of berry served or were served separately from other berries; at 13 events, raspberries were included in mixtures of various types of berries. Eating the food item that included raspberries was significantly associated with risk for illness for seven of the 15 events for which epidemiologic data are currently available (including for three of the events at which raspberries were not served with other types of berries) and was associated with illness but not significantly for six events (i.e., all or nearly all ill persons ate the berry item that was served). The raspberries reportedly had been rinsed in water at 10 (71%) of the 14 events for which such information is available. Guatemala has been identified as one of the possible sources of raspberries for all eight events for which traceback data are currently available (i.e., Guatemala was the source of at least one of the shipments of raspberries that could have been used) and as the only possible source for at least one of these events and perhaps for two others for which the traceback investigations are ongoing.

Fresh raspberries were not served at two events in restaurants in Florida that have been associated with clusters of cases of cyclosporiasis (persons were exposed on March 19 and April 10, respectively, in two different cities). The first cluster was

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associated with eating mesclun (also known as spring mix, field greens, or baby greens—a mixture of various types of baby leaves of lettuce); the specific source of the implicated mesclun has not been determined. Mesclun also is suspected as the vehicle for the second cluster.

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Editorial Note: The investigations described in this report indicate that fresh raspberries imported from Guatemala are the probable vehicle of infection for most of the outbreaks of cyclosporiasis identified in 1997. There is no evidence of ongoing transmission of *Cyclospora* in association with mesclun, which was the vehicle for one, and possibly two, early outbreaks in March and April. In the spring and summer of 1996, an outbreak of cyclosporiasis in the United States and Canada was linked to eating raspberries imported from Guatemala (*3*). However, the mode of contamination of the raspberries implicated in that outbreak was not determined—in part because the methods for testing produce and other environmental samples for this emerging pathogen are insensitive and nonstandardized. No outbreaks of cyclosporiasis were reported in the United States in association with importation of raspberries from Guatemala during the fall and winter of 1996; however, cyclosporiasis is highly seasonal in some countries.

After the outbreak in 1996, the berry industry in Guatemala, in consultation with the Food and Drug Administration (FDA) and CDC, voluntarily implemented a Hazard Analysis and Critical Control Point system and improved water quality and sanitary conditions on individual farms (*3*). The occurrence of outbreaks in 1997 suggests either that some farms did not fully implement the control measures or that the contamination is associated with a source against which these measures were not directed.

At FDA's request, on May 30, 1997, the government of Guatemala and the Guatemalan Berries Commission announced their decision to voluntarily suspend exports of fresh raspberries to the United States (the last shipment was May 28). FDA is working with CDC, the government of Guatemala, and the Guatemalan Berries Commission to determine when exports can resume (*4*). Because of the relatively short shelf life, few, if any, fresh raspberries grown in Guatemala are available now for purchase and consumption in the United States. *Cyclospora* oocysts, like the oocysts of other coccidian parasites, are expected to be inactivated by temperature extremes (e.g., pasteurization or commercial freezing processes). The minimum time and temperature conditions required to inactivate *Cyclospora* oocysts by heating or freezing have not yet been determined.

Although exports of fresh raspberries from Guatemala to the United States have been suspended until further notice, cases of cyclosporiasis that are attributable to consumption of raspberries may continue to be identified by health-care providers and health departments. The average incubation period for cyclosporiasis is 1 week; if not treated with trimethoprim-sulfamethoxazole (*5*), illness can be protracted, with remitting and relapsing symptoms. Health-care providers should consider *Cyclospora* infection in persons with prolonged diarrheal illness and specifically request laboratory testing for this parasite. Cases should be reported to local and state health

Cyclosporiasis — Continued

departments; health departments that identify cases of cyclosporiasis should contact CDC's Division of Parasitic Diseases, National Center for Infectious Diseases, telephone (770) 488-7760. Newly identified clusters of cases should be investigated to identify the vehicles of infection and to trace the sources of implicated foods.

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Lactic Acidosis Traced to Thiamine Deficiency Related to Nationwide Shortage of Multivitamins for Total Parenteral Nutrition — United States, 1997

Since November 1996, there has been a nationwide shortage of intravenous (IV) multivitamins (MVIs) used in U.S. hospitals and home-health–care agencies for total parenteral nutrition (TPN). Patients receiving TPN without MVI supplementation are at risk for thiamine deficiency and life-threatening complications associated with severe deficiency of thiamine, a coenzyme necessary for oxidation of keto acids (Figure 1). This report describes three patients receiving TPN who had thiamine deficiency-related lactic acidosis in 1997 and presents recommendations for alternatives to parenteral MVI during the shortage.

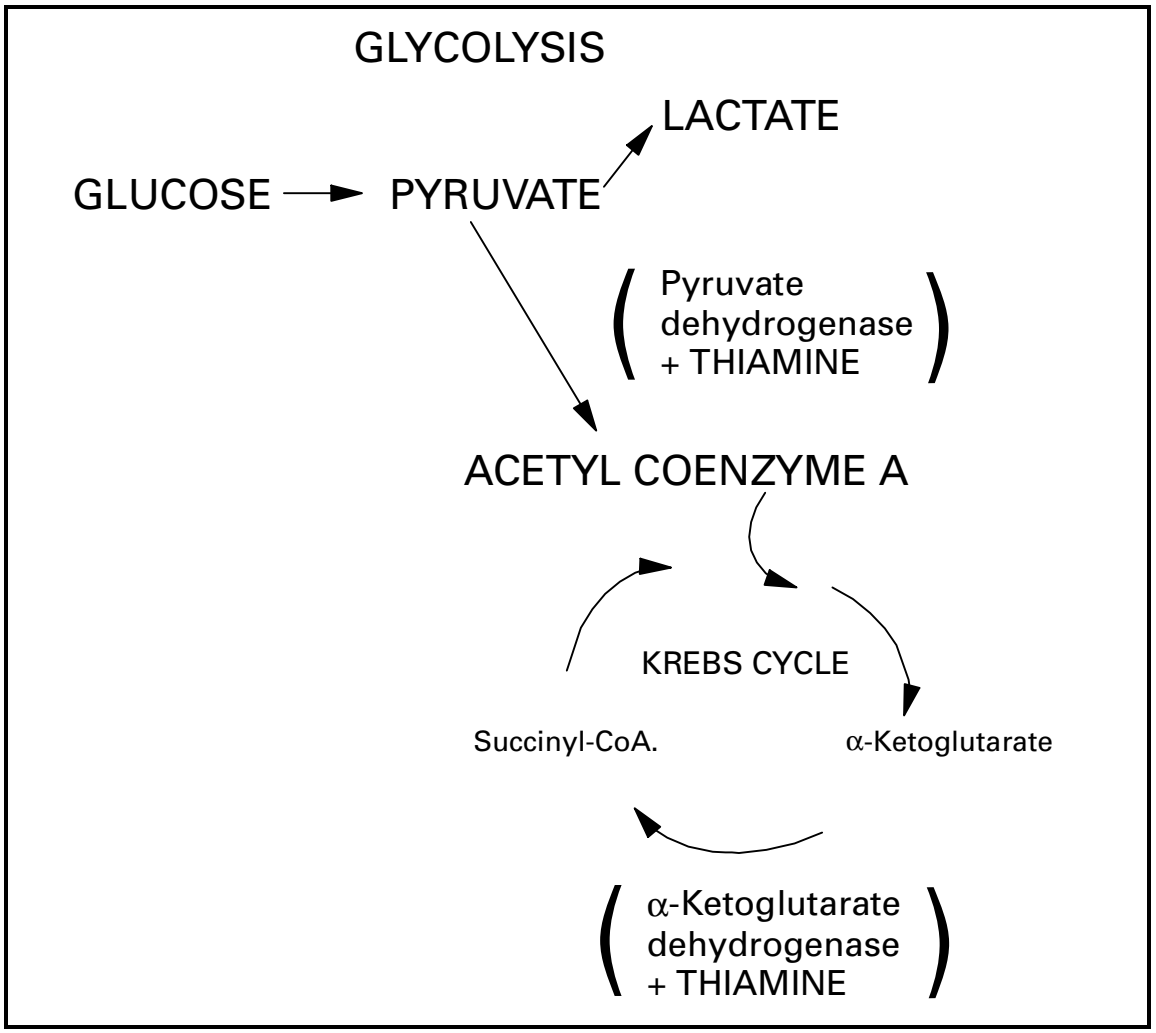
Case 1

On February 3, a 32-year-old man underwent a total coloproctectomy with ileostomy as treatment for fulminant ulcerative colitis. TPN was initiated immediately postoperatively and included 2087 mL per day of amino acids (92 g) and dextrose (382.5 g) with 21 g fat emulsion, electrolytes, and minerals per day; however, no MVIs were added to the solution because the hospital's supply was exhausted.

Attempts to introduce clear liquids orally on February 7 and 8 were unsuccessful because of persistent severe anorexia, nausea, and vomiting. On February 10, an upper gastrointestinal barium imaging study revealed delayed transit time, but no mechanical obstruction. During February 10–22, TPN was continued without MVIs. On February 22, the patient was lethargic and weak, and abnormal laboratory findings included severe acidosis (pH 6.87 [normal: 7.35–7.45]; HCO_3, 5 mEq/L [normal: 24–28 mEq/L]; pCO_2, 28 mm Hg [normal: 35–45 mm Hg]; pO_2, 131 mm Hg [normal: 80–100 mm Hg]; and base excess, –13 mEq/L), glucose level of 570 mg/dL (normal: 65–110 mg/dL), and serum lactic acid of 16 mmol/L (normal: 0.8–2.5 mmol/L); serum ketones were negative. Lactic acidosis of unknown etiology was diagnosed, and broad-spectrum antimicrobials were initiated after appropriate cultures were obtained. During the next 8 hours, 600 mEq/L of bicarbonate was administered with only modest elevation of pH (to 7.20) and no change in base excess (–16.2 mEq/L). Because

Lactic Acidosis — Continued

FIGURE 1. Thiamine requirements in the metabolic pathways of glucose*



*Source: Reference *1*.
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the patient's clinical condition continued to deteriorate, an exploratory laparotomy was performed; however, no focus of infection or bowel necrosis was found. An analysis for serum thiamine measured the lowest detectable level of 0.2 mg/dL (normal: 1.1–1.6 mg/dL), and 400 mg of thiamine was administered intravenously. Two hours later, a blood gas specimen contained a serum pH of 7.50 and an HCO_3 of 11.3 mEq/L. Acid/base and clinical status improved; a second dose of 400 mg thiamine was administered intravenously, and pH, pCO_2, and HCO_3 levels returned to normal.

Case 2

On March 10, an 11-year-old girl with chronic idiopathic intestinal pseudo-obstruction syndrome, maintained on home TPN, sought care following a 3–4 day history of abdominal pain, vomiting, and decreased ostomy output. Outpatient treatment with trimethoprim/sulfamethoxazole was initiated for suspected intestinal bacterial overgrowth. Because she did not improve, she was hospitalized on March 14.

Lactic Acidosis — Continued

A history obtained on admission revealed that she had been started on oral MVIs on January 16 because TPN supplemented with MVI was not available. However, on February 27, she had discontinued her oral MVI supplementation without notifying her physician or home-care provider. Physical examination revealed lethargy, pallor, 10% dehydration, orthostatic hypotension, tachycardia, hyperemic gingival mucosa, alterations in papillae on her tongue, and buccal mucosa ulcerations. Laboratory findings included a sodium level of 134 mmol/L (normal: 136–146 mmol/L); potassium, 2.7 mmol/L (normal: 3.5–5.3 mmol/L); blood urea nitrogen, 44 mg/dL (normal: 7–22 mg/dL); glucose, 154 mg/dL (normal: 70–100 mg/dL); and hemoglobin, 12.6 mg/dL (normal: 11.0–14.5 mg/dL). Rehydration therapy was continued with the home TPN formulation; however, she developed encephalopathy, generalized tremors, aphonia (hoarseness), progressive hyperglycemia, and acidosis with an increased anion gap. Vitamin deficiencies were confirmed with low red blood cell (RBC) transketolase activity of 0.17 IU/gm Hbg (normal: 0.75–1.3 IU/gm Hbg) and decreased levels of vitamin B_{12} (<100 pg/mL; normal: 200–1140 pg/mL) and 25-OH cholecalciferol (12.5 ng/mL; normal: 16–74 ng/mL) levels. Her RBC folate level was within normal limits, and lactate levels were not measured. Treatment with parenteral thiamine and other vitamin B supplementation improved her encephalopathy and tremors.

Case 3

On January 21, a 19-year-old man began to receive home TPN for treatment of gastrointestinal dysmotility associated with antecedent chronic cholecystitis and complications of abdominal surgeries. The initial TPN formula consisted of 2750 mL per day of amino acids (120 g) and dextrose (600 g) with 250 mL per day of 20% fat emulsion, electrolytes, minerals, vitamins, and trace elements.

On March 5, he was admitted to the hospital because of nonbloody diarrhea and fever. Findings on examination included an oral temperature of 102 F (39 C); pulse, 150 beats per minute; systolic blood pressure, 150 mm Hg; respiration rate, 20 per minute; oral thrush; an abnormally smooth tongue with decreased papillae; dry mucous membranes; diminished bowel sounds; left abdominal tenderness and rebound; rectal tenderness (without blood or abnormal mass); and a grade 2/6 systolic ejection murmur. His serum lactate level on admission was 16 mmol/L (normal: 0.93–1.65 mmol/L). The central IV catheter was removed and cultured, and TPN was temporarily discontinued. During the subsequent 5 days, the patient's neurologic status deteriorated markedly, and he became confused and complained of blurred vision, diplopia, and dyspnea. New findings included slurred speech, diminished deep tendon reflexes, ophthalmoplegia, and evidence of cortical blindness despite a normal fundoscopic examination. Magnetic resonance imaging (MRI) scan of the brain was consistent with Wernicke's encephalopathy.

As a result of the MRI findings, treatment was initiated with 100 mg per day of thiamine parenterally. The home TPN provider was contacted and reported that the patient did not receive IV MVIs during February 5–March 3 because of a national shortage. Within 24 hours after thiamine supplementation, the ophthalmoplegia and cortical blindness improved substantially. During the next 4–5 days, his mental status improved and his serum lactate level became normal.

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Lactic Acidosis — Continued

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Editorial Note: TPN may be used for short periods to treat severely ill patients and for prolonged periods for patients with chronic or permanent gastrointestinal failure. However, the decision to initiate TPN must be balanced against the potential risks for serious sequelae, including life-threatening lactic acidosis resulting from metabolic causes other than alcoholism (type B lactic acidosis). In 1989, three adults developed fatal severe lactic acidosis associated with acute thiamine deficiency while receiving TPN during a nationwide shortage of IV MVI preparations (*2*). The three cases in 1997 described in this report also were associated with a nationwide shortage of IV MVIs that began in November 1996. These cases were characterized by low thiamine levels and rapid reversal after a single dose of IV thiamine, as well as by initially refractory lactic acidosis, hyperglycemia, and absence of sepsis. The time for development of severe lactic acidosis in these and other reported episodes (range: 7–34 days) is consistent with the time required to deplete body stores of thiamine in healthy adults deprived of this vitamin. The large glucose load found in most TPN preparations results in additional metabolic needs for thiamine. To reduce the risk for complications related to thiamine deficiency, health-care providers should consider administration of thiamine if multivitamins are unavailable.

The current shortage of MVI supplement for TPN solution for adults first occurred in November 1996 after one of the U.S. distributors (Schein Industries, Florham Park, New Jersey*) of this product discontinued production of the supplement. Of the two remaining distributors, Astra USA (Westborough, Massachusetts) markets a supplement (MVI-12 Injection) that is identical to the product that was produced by Schein, and Fujisawa (Deerfield, Illinois) manufactures a preparation (Multi Vitamin Concentrate) that lacks three of the vitamins (folic acid, cyanocobalamin, and biotin) present in Schein or Astra's MVIs. Because Astra's supplier of the product (a contract manufacturer) has had and continues to have production difficulties, Astra has limited supplies of the product, which it is conserving for urgent/emergent situations. Fujisawa reports an increasing demand for their product.

The American Society for Parenteral and Enteral Nutrition (ASPEN) has recommended alternative options for parenteral MVI use in adults (see box). Because of reports of limited availability of IV MVIs for pediatric patients, ASPEN has recommended that supplements intended for neonates be reserved for use in this group (see box). Since the shortage began, the Food and Drug Administration has been working with Astra USA and ASPEN to identify alternative therapies and to identify alternate suppliers of acceptable product.

Physicians who prescribe TPN should recognize the potential risks for acute thiamine deficiency and lactic acidosis in patients who are not receiving adequate supplements. Until the manufacture of MVIs for TPN for adults increases, shortages of these products may continue. Patients who are receiving TPN for prolonged periods (>7 days) are at increased risk for lactic acidosis. Complications associated with inadequate MVIs for TPN should be reported to CDC's Hospital Infections Program, National Center for Infectious Diseases, telephone (404) 639-6413.

*Use of trade names and commercial sources is for identification only and does not imply endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Lactic Acidosis — Continued

Recommendations to Health-Care Providers to Reduce the Risk for Vitamin Deficiency in Adult and Pediatric Patients Receiving Total Parenteral Nutrition During Intravenous Multivitamin Shortages*

Recommendations for Adult Patients

1. **DO NOT** use pediatric parenteral multivitamins (MVIs) for adults.
2. Use oral vitamin preparations whenever possible.
3. Reserve use of MVI-12 Injection (Astra USA, Westborough, Massachusetts)† for patients receiving total parenteral nutrition (TPN) or those with a medical need for intravenous MVIs.
4. Ration MVI-12 (e.g., reduce the daily dose or give vitamins three times a week).
5. Use 5 mL of Multi Vitamin Concentrate (MVC) (Fujisawa, Deerfield, Illinois) three times a week along with intravenous supplementation of folic acid and monthly intramuscular or subcutaneous administration of cyanocobalamin (no parenteral biotin product is commercially available to use for supplementation). Because of the differences in the vitamin profiles of MVC and MVI-12, this provides an approximation of equivalency to MVI-12; health-care providers should remain vigilant for clinical signs of deficiency.
6. If an MVI preparation is needed and not available, then use individual vitamin preparations (oral or injectable). Optimally, patients should receive daily intravenous doses (unless otherwise clinically indicated) of 3–5 mg thiamine, 0.4–1.0 mg folate, 100 mg ascorbic acid, 5–10 mg pyridoxine, and 40–50 mg niacin. In the home setting, such patients should receive at a minimum 50 mg thiamine intravenously three times a week and folate three times a week. All patients should receive monthly doses of 100 mcg cyanocobalamin intramuscularly or subcutaneously.

Recommendations for Pediatric Patients§

1. Reserve use of MVI-Pediatric (Astra USA) for neonates.
2. MVI-Pediatric may deliver insufficient quantities of vitamin A in very-low–birthweight infants. Vitamin A supplementation in addition to MVI-Pediatric should be decided for each patient individually.
3. MVI-12 contains propylene glycol and similar quantities of polysorbate, which can cause adverse consequences:
   a. Polysorbate has been associated with hepatotoxicity resulting from use of intravenous vitamin E.
   b. Propylene glycol has been associated with hyperosmolality and seizures.
   c. Concomitant administration of drugs using propylene glycol as an excipient could increase the risk for toxicity.

   Therefore, to avoid propylene glycol toxicity, reserve MVI-Pediatric for use in low-birthweight infants (<3 lb 4 oz [<1500 g]) for which >2 weeks of TPN is anticipated.
4. Use adult MVIs for infants weighing ≥3 lb 4 oz (≥1500 g) and children. Supplementation with additional vitamins is necessary.

* Based on recommendations from the American Society for Parenteral and Enteral Nutrition (ASPEN).
† Use of trade names and commercial sources is for identification only and does not imply endorsement by the Public Health Service or the U.S. Department of Health and Human Services.
§ Because of the complex nature of the recommendations for pediatric patients receiving TPN, these recommendations are only a summary. Complete detailed recommendations are available from ASPEN, telephone (301) 587-6316 or e-mail aspen@access.digex.net.

Lactic Acidosis — Continued

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Heat-Related Deaths — Dallas, Wichita, and Cooke Counties, Texas, and United States, 1996

During July 2–8, 1996, high maximum daily temperatures in Dallas County, Texas, ranged from 101 F (38.3 C) to 106 F (41.1 C), and high maximum daily heat indexes (a measure of the effect of combined elements [e.g., heat and humidity] on the body) ranged from 105 F (40.6 C) to 112 F (44.4 C). Although guidelines for issuing heat advisories or warnings vary by geographic location and climate, the National Weather Service generally suggests issuing a heat advisory when a daytime heat index reaches ≥105 F (≥40.6 C), and a night time minimum ambient temperature of 80 F (26.7 C) persists for at least 48 hours. In Dallas County, the criterion used by the medical examiner's (ME's) office to designate a heat wave is ≥3 consecutive days of temperatures ≥100 F (37.8 C). This report describes four cases of heat-related death in Dallas, Wichita, and Cooke counties, Texas, in 1996; summarizes risk factors for this problem; and reviews measures to prevent heat-related morbidity and mortality. The findings in this report indicate that, although a large proportion of heat-related deaths occur during the summer and during heat waves, such deaths occur year-round.

For a death to be attributed to heat-related illness by the Dallas County ME's office (which serves as consultant for both Cooke and Wichita counties), a decedent must meet at least one of the following three criteria: 1) core body temperature is ≥105 F (≥40.6 C) at the time of or immediately following death, 2) there is substantial environmental or circumstantial evidence of heat as a contributor to death (e.g., decedent is found in a room with a high ambient temperature, windows closed, and no air conditioning), or 3) decedent is found in a decomposed condition without evidence of other cause of death, and the decedent was last seen alive during the heat-wave period.

Case 1. On February 21 (an exceptionally warm winter day [temperature approximately 90 F (32.2 C)]), a 10-month-old girl was left in a car in Dallas County at approximately 9:30 a.m.; she was discovered unresponsive at approximately 2:45 p.m. Despite the initiation of cardiopulmonary resuscitation, she could not be resuscitated. A core body temperature of 108 F (42.2 C) was recorded in the emergency department (ED), and the cause of death was listed as hyperthermia.

Case 2. On July 9, a 61-year-old female resident of Cooke County was found dead in her bedroom in a residence with no air conditioning. Although fans were operating in the room, the room temperature was 107 F (41.7 C); air moved by the fan was 104 F (40.0 C). Family members reported having heard the woman moving about at approximately 8 a.m., but she was not checked by family members until approximately noon. The family reported a possible history of diabetes, although this diagnosis could not be confirmed. The primary cause of death was listed as hyperthermia, and the secondary cause was listed as dilated cardiomyopathy.

Heat-Related Deaths — Continued

Case 3. On July 10, a 52-year-old man walked from the lawn he was mowing in Dallas County to a nearby residence and knocked on the back door. When the homeowner opened the door, the man collapsed onto the porch. An ambulance transported him to a hospital ED, where he died. On arrival in the ED, his core temperature was 107.1 F (41.7 C), which, before his death, was reduced to 101 F (38.3 C) with ice baths. The outside temperature at the time he collapsed was 109 F (42.8 C). The primary cause of death was listed as hyperthermia, and the secondary cause was listed as hypertensive and arteriosclerotic cardiovascular disease.

Case 4. On July 10, an 80-year-old female resident of Wichita County was discovered outdoors at 8 a.m. near shrubs she had been watering the previous day. Although her residence was air conditioned, the high temperature outside the day she died was 102 F (38.9 C). The primary cause of death was listed as hyperthermia. The secondary cause of death was listed as arteriosclerotic cardiovascular disease.

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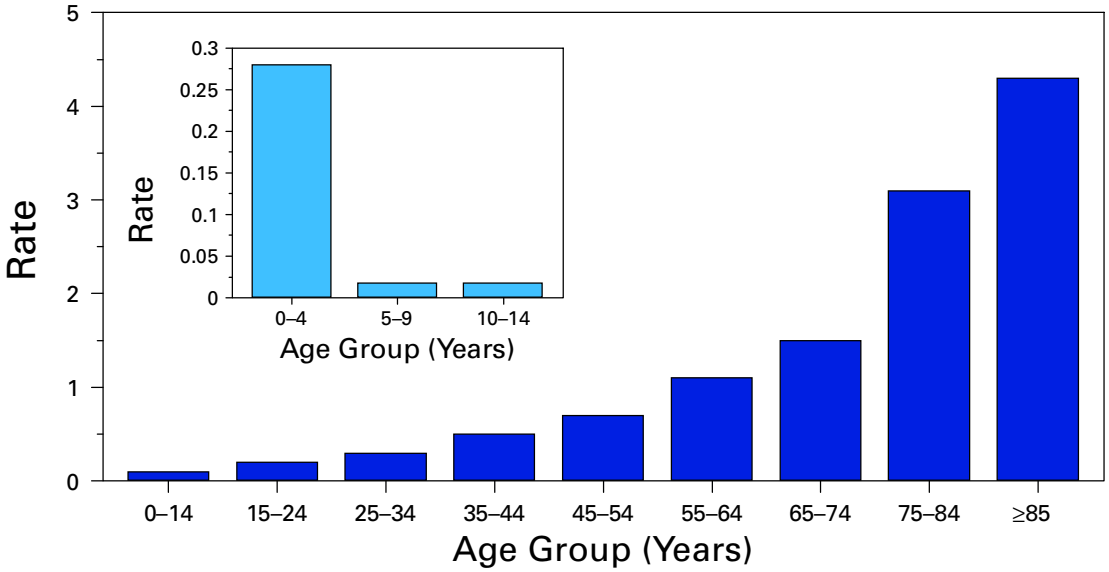
Editorial Note: During 1979–1994, the underlying cause of death for 5899 deaths in the United States was heat exposure*; of the 2383 persons for whom age data were available, persons aged ≥55 years accounted for 1471 (62%) heat-related deaths "due to weather conditions" (Figure 1). Of the 221 heat-related deaths in 1994, a total of 101 were "due to weather conditions." During 1979–1994, the four highest age-adjusted rates for heat-related deaths "due to weather conditions" occurred in Arizona, Arkansas, Kansas, and Missouri (range: 2.7–3.7 per 1 million population). Because several other causes of death (e.g., cardiovascular and respiratory diseases) also increase during heat waves (*1–3*), deaths attributed to hyperthermia represent only a portion of heat-related excess mortality. The criteria to determine which deaths are attributable to hot weather and heat-related illness vary by state and among individual MEs and coroners (*1,4,5*). Consequently, the effects of hot weather on human mortality probably are underestimated, and standard definitions are needed to accurately classify these deaths (*1,4,5*).

The cases described in this report illustrate the spectrum of factors and conditions associated with heat-related mortality, including age (the very young and the elderly), medical history (e.g., cardiovascular disease), social circumstance (e.g., living alone), and physical activity (e.g., exertion in exceptionally hot environments during either work or recreational activities) (*2,6*). Other factors associated with increased risk include alcohol consumption, chronic conditions (e.g., respiratory diseases), history of previous heatstroke, use of some medications (e.g., phenothiazines, butyrophenones, and thioxanthenes), and physical or mental impairment or bed confinement that interferes with ability to care for oneself (*2,4,6*). In addition to persons with risk factors, all persons may be at increased risk for fatal heatstroke if sufficiently exposed—even on

* *International Classification of Diseases, Ninth Revision* (ICD-9), code E900.0, "due to weather conditions" (deaths); code E900.1, "of man-made origin" (deaths); or code E900.9, "of unspecified origin" (deaths). These data were obtained from the Compressed Mortality File (CMF) of CDC's National Center for Health Statistics, which contains information from death certificates filed in the 50 states and the District of Columbia that have been prepared in accordance with external cause codes. CDC's Wide-ranging ONline Data for Epidemiologic Research computerized information system was used to access CMF data. All rates were standardized to the 1980 U.S. population.

Heat-Related Deaths — Continued

FIGURE 1. Average annual rate* of heat-related deaths,† by age group — United States, 1979–1994



*Per 1 million population.
†Underlying cause of death attributed to excessive heat exposure classified according to the *International Classification of Diseases, Ninth Revision* (ICD-9), as code E900.0, "due to weather conditions" (deaths).

exceptionally hot winter days (*4*). Because young children, the elderly, and the immobile may be unable to obtain and drink adequate fluids or to avoid hot environments, they are at greater risk for heat exhaustion or heatstroke (*2*). The use of some drugs may increase the risk for heat-related illness by interfering with the body's physical heat regulatory system (*2,4*); examples of such drugs are neuroleptics (e.g., antipsychotics or major tranquilizers) and medications with anticholinergic effects (e.g., tricyclic antidepressants, antihistamines, some antiparkinsonian agents, and some over-the-counter sleeping pills). Alcohol consumption may cause dehydration, which increases the risk for heat-related illness (*2*).

Adverse health conditions associated with high environmental temperatures include heatstroke, heat exhaustion, heat syncope, and heat cramps (*4*). Heatstroke is a medical emergency characterized by rapid onset and progression (within minutes) of the core body temperature to ≥105 F (≥ 40.4 C) and lethargy, disorientation, delirium, and coma (*4*). Heatstroke is often fatal despite expert medical care directed at rapidly lowering the body temperature (e.g., ice baths) (*4*). Manifestations of heat exhaustion, which is clinically more benign than heatstroke, include dizziness, weakness, or fatigue often following several days of sustained exposure to hot temperatures (*4*); treatment for heat exhaustion is directed at replacing fluids and electrolytes and may require hospitalization (*4*). Heat syncope and heat cramps are usually related to physical exertion during hot weather (*4*). Treatment of persons who lose consciousness as a result of heat syncope should include placement in a recumbent position and electrolyte replacement (*4*).

Heat-Related Deaths — Continued

Persons working in high temperatures—either indoors or outdoors—should take special precautions, including allowing 10–14 days to acclimate to an environment of high ambient temperature. Although adequate salt intake is important, salt tablets are not recommended and may even be hazardous for many persons (*4*). Even though the use of fans may increase comfort at temperatures <90 F (<32.2 C), fans are not protective against heatstroke in the presence of high temperatures (≥90 F [≥32.2 C]) and humidity (>35%) (*2,7*).

Measures for preventing heat-related illness and death include increasing time in air-conditioned environments, increasing nonalcoholic fluid intake, exercising only during cooler parts of the day, and taking cool-water baths (*2*). Persons whose fluid consumption is restricted for medical reasons should alter their fluid intake patterns only if advised by their physicians (*4*). The elderly should be encouraged and assisted in taking advantage of air-conditioned environments (e.g., shopping malls, public libraries, and heat-wave shelters), even if for only part of the day (*2,4,6*). Parents should be educated about the increased heat sensitivity of children aged <5 years (*4*). Prevention messages about how to avoid heat-related illness should be disseminated to the public as early as possible when exceptionally high temperatures are forecast. These messages can assist in reducing the risk for heat-related deaths, even though such deaths usually do not occur until the second or third day of a heat wave (*1,5*).

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Lyme Disease — United States, 1996

Lyme disease (LD) is caused by the tickborne spirochete *Borrelia burgdorferi* sensu lato and is the most common vectorborne disease in the United States. Surveillance for LD was initiated by CDC in 1982, and the Council of State and Territorial Epidemiologists designated it a nationally notifiable disease in January 1991. For surveillance purposes, LD is defined as the presence of an erythema migrans rash ≥5 cm in diameter or laboratory confirmation of infection with evidence of at least one manifestation of musculoskeletal, neurologic, or cardiovascular disease (*1*). This report summarizes the provisional number of cases of LD reported to CDC during 1996 and indicates that the number of cases reported to CDC was a record high.

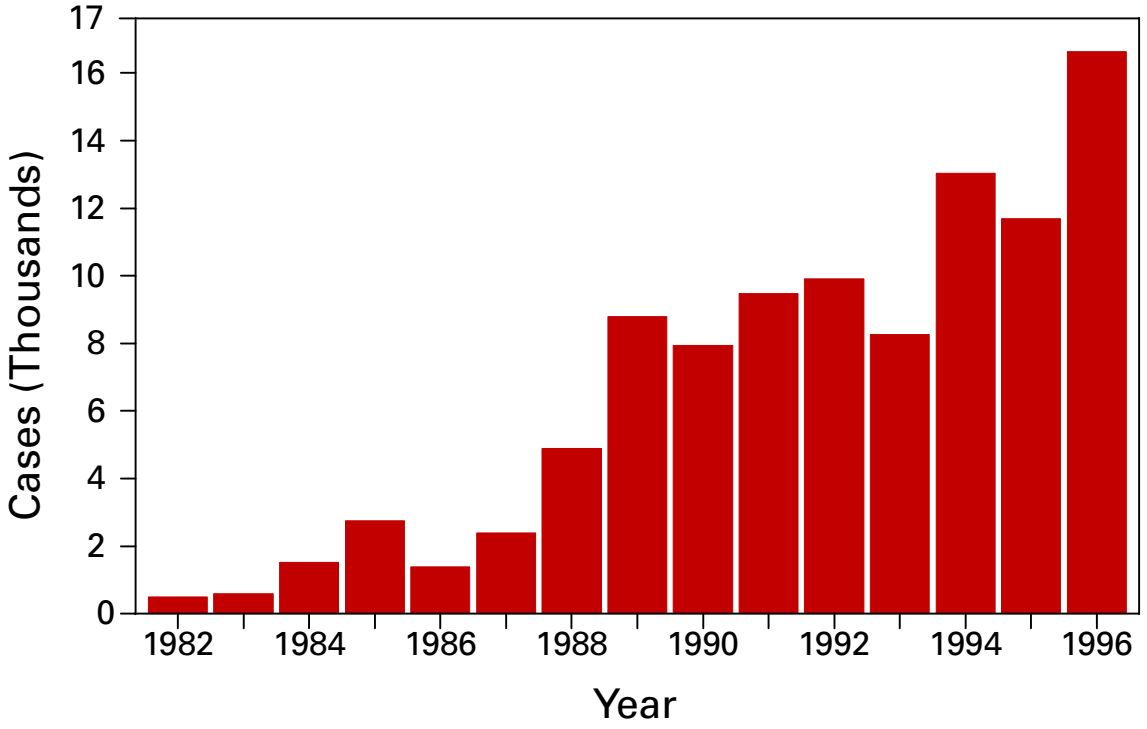
Lyme Disease — Continued

In 1996, a total of 16,461 cases of LD were reported to CDC by 45 states and the District of Columbia (overall incidence: 6.2 per 100,000 population*), representing a 41% increase from the 11,700 cases reported in 1995 and a 26% increase from the 13,043 cases reported in 1994 (Figure 1). As in previous years, most cases were reported from the Mid-Atlantic, Northeast, and North Central regions (Table 1). Eight states reported LD incidences that were higher than the overall national rate (Connecticut, 94.8; Rhode Island, 53.9; New York, 29.2; New Jersey, 27.4; Delaware, 23.9; Pennsylvania, 23.3; Maryland, 8.8; and Wisconsin, 7.7); these states accounted for 14,959 (91%) of the nationally reported cases. In 1996, zero cases were reported from five states (Alaska, Arizona, Colorado, Montana, and South Dakota).

Eighty-seven counties each reporting ≥20 cases accounted for 89% of all reported cases. Reported incidences were >100 per 100,000† in 18 counties in Connecticut, Maryland, Massachusetts, North Carolina, New Jersey, New York, Pennsylvania, Rhode Island, and Wisconsin; the highest reported county-specific incidence (1247.5 per 100,000) was in Nantucket County, Massachusetts (Figure 2). From 1995 to 1996, a total of 28 states reported increases in the number of cases, 16 states reported decreases, and seven states reported no change. Approximately 90% of the total increase in reported cases in 1996 occurred in five states (Connecticut, New Jersey, New York, Pennsylvania, and Rhode Island) where average annual LD incidence rates had exceeded the national average for the previous 5 years combined.

*State rates are based on 1996 population estimates.
†County rates are based on 1990 population estimates.

FIGURE 1. Number of reported cases of Lyme disease, by year — United States, 1982–1996*



*Data for 1996 are provisional.

Lyme Disease — Continued

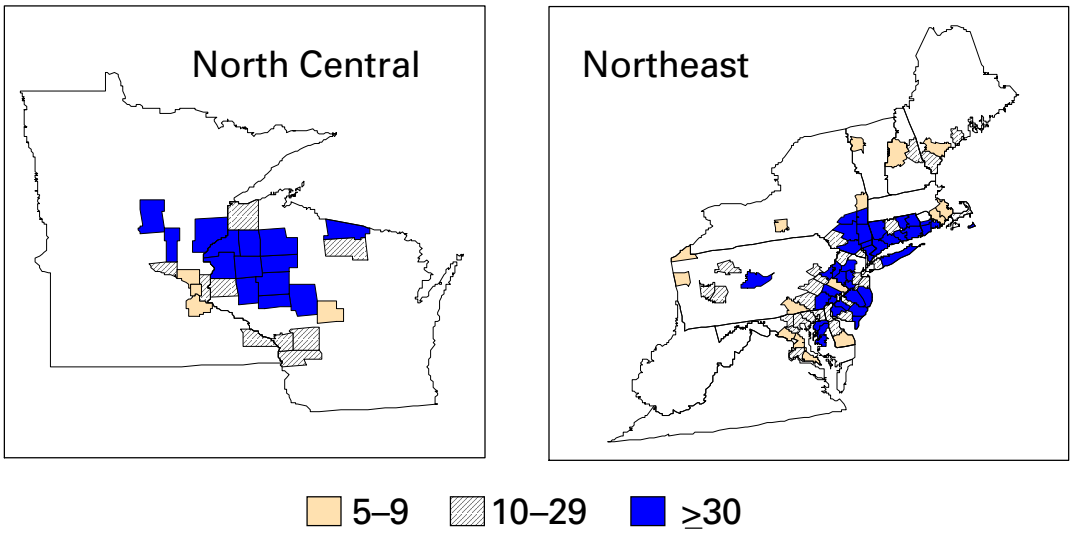
TABLE 1. Number of reported cases of Lyme disease, by state, 1991–1996*, and rate† of Lyme disease, 1996 — United States

State	1991	1992	1993	1994	1995	1996	Total	1996 Rate
Alabama	13	10	4	6	12	9	**54**	0.2
Alaska	0	0	0	0	0	0	**0**	0.0
Arizona	1	0	0	0	1	0	**2**	0.0
Arkansas	31	20	8	15	11	27	**112**	1.1
California	265	231	134	68	84	80	**862**	0.3
Colorado	1	0	0	1	0	0	**2**	0.0
Connecticut	1,192	1,760	1,350	2,030	1,548	3,104	**10,984**	94.8
Delaware	73	219	143	106	56	173	**770**	23.9
District of Columbia	5	3	2	9	3	3	**25**	0.6
Florida	35	24	30	28	17	55	**189**	0.4
Georgia	25	48	44	127	14	1	**259**	0.0
Hawaii	0	2	1	0	0	1	**4**	0.1
Idaho	2	2	2	3	0	2	**11**	0.2
Illinois	51	41	19	24	18	10	**163**	0.1
Indiana	16	22	32	19	19	32	**140**	0.5
Iowa	22	33	8	17	16	19	**115**	0.7
Kansas	22	18	54	17	23	36	**170**	1.4
Kentucky	44	28	16	24	16	26	**154**	0.7
Louisiana	6	7	3	4	9	9	**38**	0.2
Maine	15	16	18	33	45	61	**188**	4.9
Maryland	282	183	180	341	454	447	**1,887**	8.8
Massachusetts	265	223	148	247	189	321	**1,393**	5.3
Michigan	46	35	23	33	5	20	**162**	0.2
Minnesota	84	197	141	208	208	251	**1,089**	5.4
Mississippi	8	0	0	0	17	21	**46**	0.8
Missouri	207	150	108	102	53	52	**672**	1.0
Montana	0	0	0	0	0	0	**0**	0.0
Nebraska	25	22	6	3	6	5	**67**	0.3
Nevada	5	1	5	1	6	2	**20**	0.1
New Hampshire	38	44	15	30	28	47	**202**	4.0
New Jersey	915	688	786	1,533	1,703	2,190	**7,815**	27.4
New Mexico	3	2	2	5	1	1	**14**	0.1
New York	3,944	3,448	2,818	5,200	4,438	5,301	**25,149**	29.2
North Carolina	73	67	86	77	84	66	**453**	0.9
North Dakota	2	1	2	0	0	2	**7**	0.3
Ohio	112	32	30	45	30	32	**281**	0.3
Oklahoma	29	27	19	99	63	45	**282**	1.4
Oregon	5	13	8	6	20	19	**71**	0.6
Pennsylvania	718	1,173	1,085	1,438	1,562	2,814	**8,790**	23.3
Rhode Island	142	275	272	471	345	534	**2,039**	53.9
South Carolina	10	2	9	7	17	9	**54**	0.2
South Dakota	1	1	0	0	0	0	**2**	0.0
Tennessee	35	31	20	13	28	24	**151**	0.5
Texas	57	113	48	56	77	97	**448**	0.5
Utah	2	6	2	3	1	1	**15**	0.0
Vermont	7	9	12	16	9	26	**79**	4.4
Virginia	151	123	95	131	55	57	**612**	0.9
Washington	7	14	9	4	10	18	**62**	0.3
West Virginia	43	14	50	29	26	12	**174**	0.7
Wisconsin	424	525	401	409	369	396	**2,524**	7.7
Wyoming	11	5	9	5	4	3	**37**	0.6
Total	**9,470**	**9,908**	**8,257**	**13,043**	**11,700**	**16,461**	**68,839**	**6.2**

* Data for 1996 are provisional.
† Per 100,000 population.

Lyme Disease — Continued

FIGURE 2. Rate* of reported cases of Lyme disease, by county† — North Central and Northeast United States, 1996



*Per 100,000 population.
†Excludes counties with fewer than five reported cases.

Of 5298 cases for which information was available, 217 (4%) were reported as having been acquired outside of the United States, and 156 (3%) cases were reported as having been acquired in the United States but outside of the reporting state. The highest proportions of cases occurred among persons aged 0–14 years (3784 [23%]) and adults aged 40–79 years (7694 [47%]). Of 16,422 cases for which sex was reported, 8634 (53%) were male.

Reported by: State health depts. Bacterial Zoonoses Br, Div of Vector-Borne Infectious Diseases, National Center for Infectious Disease, CDC.

Editorial Note: LD continues to be an important emerging infection: geographic spread within states with endemic disease and intensified transmission of the LD spirochete in established foci of infection have been associated with increased numbers of reported cases in the United States. In the eastern United States, the patterns of human LD cases reflect the geographic distribution of *Ixodes scapularis*, also known as the black-legged or deer tick (*2,3*). Substantial annual fluctuations since 1992 in the number of reported cases in several northeastern states with endemic disease have been attributed, in part, to variations in *I. scapularis* density (*4,5*). The principal vector in western coastal states is *I. pacificus* (the western black-legged tick). LD also is transmitted by *Ixodes* spp. in Canada and in temperate areas of Eurasia, including Europe, Russia, northern People's Republic of China, and Japan (*6*).

Increases in reported LD cases in 1996 were limited to certain counties in some states, consistent with focal differences in the distribution and density of the tick vector. In both Connecticut and Rhode Island, the numbers of reported cases of LD increased statewide, although increases were greatest in coastal counties. In both states, this increase was associated with increased population densities of *I. scapularis* (K. Stafford, Connecticut Agricultural Experiment Station, and T. Mather, University of Rhode Island, personal communications, 1997). In New York, the greatest increases occurred in Dutchess County, where reported cases of LD nearly doubled

Lyme Disease — Continued

from 1995 (918) to 1996 (1832). Because an LD vaccine trial was being conducted in the area, some of this increase may have resulted from heightened awareness and reporting of LD. The number of reported cases was stable in other counties of New York with endemic disease, including Putnam, Suffolk, and Westchester counties. In New Jersey, eight counties with active surveillance reported higher rates than the remaining counties with passive surveillance systems.

Since 1991, state health departments in regions with endemic disease have been expanding their use of laboratory testing for assisting in LD surveillance. A positive laboratory result is required for reporting of persons with systemic manifestations of LD but is not required for persons with an erythema migrans rash ≥5 cm in diameter (i.e., early LD). Since August 1995, when CDC published recommendations for standardized two-step (enzyme immunoassay and Western immunoblot) serodiagnostic testing for LD (*7*), states have reported a shift toward use of the recommended two-step method in diagnostic laboratories. The impact of these changes in laboratory methods on LD surveillance is unknown.

The increase in reported LD cases in 1996 probably represents a combination of increased tick density, enhanced health-care provider awareness and reporting, and improved laboratory surveillance. In addition, case reporting has been enhanced through the availability of CDC resources for LD surveillance in eight states (Connecticut, Michigan, Minnesota, New Jersey, New York, Oregon, Rhode Island, and West Virginia).

Most LD cases respond well to appropriate antibiotic therapy; drugs of choice include amoxicillin, doxycycline, and ceftriaxone (*8*). Vaccines to prevent LD are under evaluation but are not yet available. Personal protection methods recommended for preventing cases of LD and other tickborne diseases (e.g., babesiosis, ehrlichiosis, and Rocky Mountain spotted fever) include wearing light-colored clothing (to more readily detect ticks), tucking long pants into socks, using insect repellents and acaricides according to label directions, and performing tick checks at least daily. The use of environmental modifications to residential properties (e.g., application of insecticides, use of deer fencing, and removal of leaf litter) also may help prevent LD.

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Malaria in an Immigrant and Travelers — Georgia, Vermont, and Tennessee, 1996

Each year, approximately 1000 cases of malaria are reported in the United States, nearly all among persons with histories of antecedent international travel. Failure to use appropriate measures to prevent infection when traveling in areas with endemic disease and delays in diagnosis and treatment can result in severe complications and death. This report presents three recent cases of malaria that illustrate the importance of following the fundamental measures for preventing malaria.

Case 1

In November 1996, a 37-year-old woman who was 35 weeks' pregnant was admitted to a hospital in Atlanta, Georgia, because of a 3-day history of fever, chills, dysuria, back pain, nausea, vomiting, and headache. She denied cough, night sweats, and weight loss. She had moved to Georgia from Honduras in 1994 and had not traveled outside the United States since arriving. On physical examination, her temperature was 102.2 F (39.0 C), and she had a firm gravid uterus. Laboratory findings included a hematocrit of 30% (normal: 34%–46%); white blood cell (WBC) count, 4400/mm^3 (normal: 4300–10,800/mm^3); and serum glucose, 203 mg/dL. Urinalysis showed 1+ protein, 3+ glucose, 2–5 WBCs, and two red blood cells (RBCs). She was admitted to the hospital with a diagnosis of a possible urinary tract infection and/or meningitis; following a lumbar puncture (analysis within normal limits) empirical treatment with cefazolin was initiated. However, her fever persisted and, on the fifth day of hospitalization, intraerythrocytic parasites consistent with malaria were detected in a peripheral blood smear.

Based on her move from Honduras in 1994 and microscopic examination of her blood smear, *Plasmodium vivax* infection was diagnosed; treatment was initiated with chloroquine 600 mg base PO, followed by 300 mg 6 hours later and daily for the next 2 days. She immediately defervesced and was discharged on chloroquine 300 mg weekly until delivery. Five weeks later and despite apparent gestational diabetes, she delivered a healthy baby without complications. She was prescribed primaquine 15 mg base PO for 14 days after finishing breast-feeding.

Case 2.

In October 1996, a 49-year-old man was admitted to a hospital in Vermont because of a 6-day history of nausea, vomiting, diarrhea, fever, and chills. Illness had begun 6 days earlier during a 5-week business trip to Kenya and southern Sudan. As a physician and an international health consultant, the patient had traveled frequently to areas with endemic malaria. In June 1996, he had had an episode of *P. falciparum* malaria and was treated with quinine. Although he had used chloroquine prophylaxis during previous travels, he had not taken any antimalarial drugs during the trip to Kenya and Sudan because of concerns with long-term chemoprophylaxis toxicity.

During the trip, he had noticed a foot ulcer and had cleaned the wound. Two days before departing for return to the United States, he had acute onset of vomiting, diarrhea, and fever, but initially attributed these symptoms to food poisoning. When the fever persisted and diaphoresis and chills occurred, he considered the possibility of malaria but did not seek treatment. His condition improved, but during a 1-day stop

Malaria — Continued

while in transit to the United States, fever and fatigue recurred. He deferred medical evaluation until his return to Vermont, where he was admitted to the hospital.

On physical examination, his temperature was 99.8 F (37.7 C), and blood pressure was 103/54 supine and 80/46 sitting. Laboratory findings included hemoglobin of 9.3; WBC count, 7400/mm^3; platelets, 35,000/mm^3 (normal: 150,000–450,000/mm^3); blood urea nitrogen, 83; creatine, 3.7 mg/dL (normal: 0.8–1.3 mg/dL); total bilirubin, 1.8 mg/dL (normal: 0.2–1.5 mg/dL); and direct bilirubin, 1.4 mg/dL (normal: 0–0.4 mg/dL). Urinalysis showed 1+ protein and 1+ blood with granular casts and urate crystals. Examination of a blood smear detected *P. falciparum* parasites with a density of 1% of parasitized RBCs.

Treatment was initiated with intravenous quinidine and doxycycline. On the third day of hospitalization, the patient developed adult respiratory distress syndrome, which required intubation for 1 week; complications included barotrauma, acute tubular necrosis, and a nosocomial infection. Concurrently, the patient was diagnosed with and treated for *Entamoeba histolytica* diarrhea and *Staphylococcus aureus* infection in the foot ulcer. He recovered fully after an 18-day hospitalization.

Case 3

On March 25, 1996, a 64-year-old resident of Tennessee sought care at a local emergency department because of a 5-day history of intermittent fever, nausea, severe diarrhea, and generalized body aches. On March 20, he had returned from an annual trip to rural areas of Papua New Guinea, Philippines, and Myanmar, where malaria is endemic. During 18 previous trips, he had never taken malaria chemoprophylaxis, although he was aware of the recommendations for prophylaxis. Malaria was presumptively diagnosised, and he was admitted to the hospital.

His temperature on admission was 100 F (38 C) and pulse rate was 111 beats per minute. Laboratory findings included a white blood cell count of 2900/mm^3 (normal: 4300–10,800/mm^3); platelet count, 58,000/mm^3 (normal: 130,000–400,000/mm^3); hematocrit, 44% (normal: 42%–52%); and total bilirubin, 1.2 mg/dL (normal: <1.6 mg/dL). Peripheral blood smears obtained during the first 2 days of hospitalization showed ring forms diagnostic of *P. falciparum* with less than 1% parasitemia. He was treated with oral quinine and doxycycline. On the night of admission, he sustained a respiratory arrest and was placed in the intensive-care unit (ICU) and was resuscitated. His antimalarial medication was changed to intravenous quinidine and doxycycline. On March 27, the quinidine was discontinued because of prolongation of the QT interval on his electrocardiogram. His condition improved, and antimalarial treatment was discontinued after 7 days.

On April 2, the patient developed increasing respiratory difficulty and was transferred back to the ICU for suspected pulmonary edema and multilobar nosocomial pneumonia. Despite aggressive treatment, he died on April 6.

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Malaria — Continued

Editorial Note: Malaria remains a major cause of morbidity and mortality worldwide, with an estimated 300–500 million cases occurring annually (*1*). Factors associated with increased risks for or variations in the epidemiology of malaria include increasing international travel, changing patterns of travel (e.g., "adventure tourism" or immigration from malarious areas), and intensified antimalarial drug resistance (*2*).

The first two cases described in this report underscore important considerations regarding recognizing and promptly treating malaria in U.S. citizens and others returning from abroad or who are visiting the United States. Prompt diagnosis requires that malaria be included in the differential diagnosis of illness in a febrile patient with a recent history of travel to an area with endemic disease. Because some patients may not spontaneously mention a travel history, clinicians should ask about recent travel, particularly when evaluating febrile illnesses in international travelers, immigrants, migrant laborers, and international visitors. The patient's travel itinerary can provide key information for choosing appropriate drugs for antimalarial prophylaxis and malaria treatment. For example, since relapses of *P. vivax* can occur up to 4 years after the initial infection, for case 1, history of having lived in Honduras 2 years before the onset of illness was relevant to considering malaria.

The second and third cases illustrate the importance of antimalarial prophylaxis when traveling in a malarious area and the hazards associated with delaying diagnosis and treatment. Although malaria is most prevalent in rural areas of the tropics at elevations below 3282 feet (1000 meters), it is not limited to these areas. A careful review of a traveler's itinerary is necessary to determine the need for chemoprophylaxis and the most appropriate drug-treatment regimen. Prophylaxis should be started 1 week before travel (1–2 days for doxycycline) and continued throughout the stay in the malarious area and for 4 weeks after leaving the area. Although retinopathy has been reported after high doses of chloroquine for treatment of illnesses such as rheumatoid arthritis, this has not been documented to occur when chloroquine is used long-term for antimalarial prophylaxis.

When malaria is suspected, diagnosis and treatment should be initiated immediately. *P. falciparum* malaria often presents with nonspecific symptoms without the classical periodic fever. Because the multiplication cycle for this species is only 36–48 hours, the patient's clinical condition can deteriorate rapidly and, as in case 2, a delay of even as little as 6 hours can be critical. The potential sequelae of untreated *P. falciparum* malaria (e.g., adult respiratory distress syndrome, cerebral malaria, and renal failure) can be life-threatening. In addition, the estimated median cost ($12,516) of treating one case of severe *P. falciparum* infection contrasts sharply to the relatively inexpensive cost of a full prophylactic course of mefloquine ($48.00 to $56.00 for a median-length trip of 23 days) (*3*).

Information about malaria prophylaxis and treatment is available from CDC's Division of Parasitic Diseases, National Center for Infectious Diseases, telephone (770) 488-7760, from 8 a.m. to 4:30 p.m. eastern time, Monday through Friday, and (404) 639-2888 during other hours and on weekends. The automated information service (telephone [404] 332-4565) will fax documents containing information about general aspects of malaria, malaria in pregnant women and children, and prescription drugs used for malaria. International travel information is available on the World-Wide Web at http://www.cdc.gov/travel/travel.htm.

Malaria — Continued

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Sarcoidosis Among U.S. Navy Enlisted Men, 1965–1993

Sarcoidosis is a multisystem granulomatous disease of unknown etiology with highest incidence among young and middle-aged adults. In the United States, the risk for sarcoidosis is substantially higher among blacks than among other races (*1*,*2*); however, the reasons for this association are unknown. In response to the occurrence of a case of sarcoidosis in a U.S. Navy (USN) enlisted man, CDC's National Institute for Occupational Safety and Health (NIOSH) analyzed USN data on cases of sarcoidosis diagnosed among active-duty enlisted personnel during 1965–1993. This report summarizes the findings of this analysis, which indicate that the incidence of sarcoidosis declined among USN enlisted men during 1965–1993, particularly among blacks, and that the risk for sarcoidosis was statistically associated with the assignment of USN enlisted men to aircraft carriers.

In 1974, a 21-year-old black enlisted man had sarcoidosis diagnosed based on a chest radiograph indicating bilateral hilar adenopathy without parenchymal disease; noncaseating granulomata were present on lymph node biopsy. He had a history of shortness of breath, cough, and chest and joint pain, which he related to his work of grinding antiskid materials from aircraft carrier decks during the preceding 2 years. He received a medical discharge for sarcoidosis in 1975. In 1987, physicians at the U.S. Department of Veterans Affairs diagnosed pneumoconiosis in this patient after mineral-dust deposits were identified in a lung biopsy; the mineral-dust deposits were attributed to the same work exposures aboard the aircraft carrier. In October 1992, the patient asked the USN to request NIOSH to investigate whether his sarcoidosis diagnosis and other cases diagnosed in persons with whom he had served in the USN may have been associated with environmental exposures during their USN service. Because of the possibility of an association between risk for sarcoidosis-like illnesses and environmental exposures during service in the USN and because the underlying cause(s) of sarcoidosis is unknown, in December 1992 the USN requested that NIOSH evaluate the potential relation between sarcoidosis and the USN work environment.

NIOSH obtained records from the U.S. Naval Health Research Center (NHRC) for all incident cases of sarcoidosis (defined as diagnosis of "sarcoidosis" by a USN health-care provider) identified among white and black enlisted men while on active duty at any time from 1965 through 1993* (n=1121). Incidence rates were calculated using age-specific total denominator data for white and black enlisted men on active duty from 1971 through 1993 (denominator data were unavailable for the years before 1971). Numbers for other races were too small for meaningful analysis (no more than three incident cases of sarcoidosis were diagnosed among persons in any other racial

*The most recent year for which USN data were complete at the time the NIOSH analysis was started.

Sarcoidosis — Continued

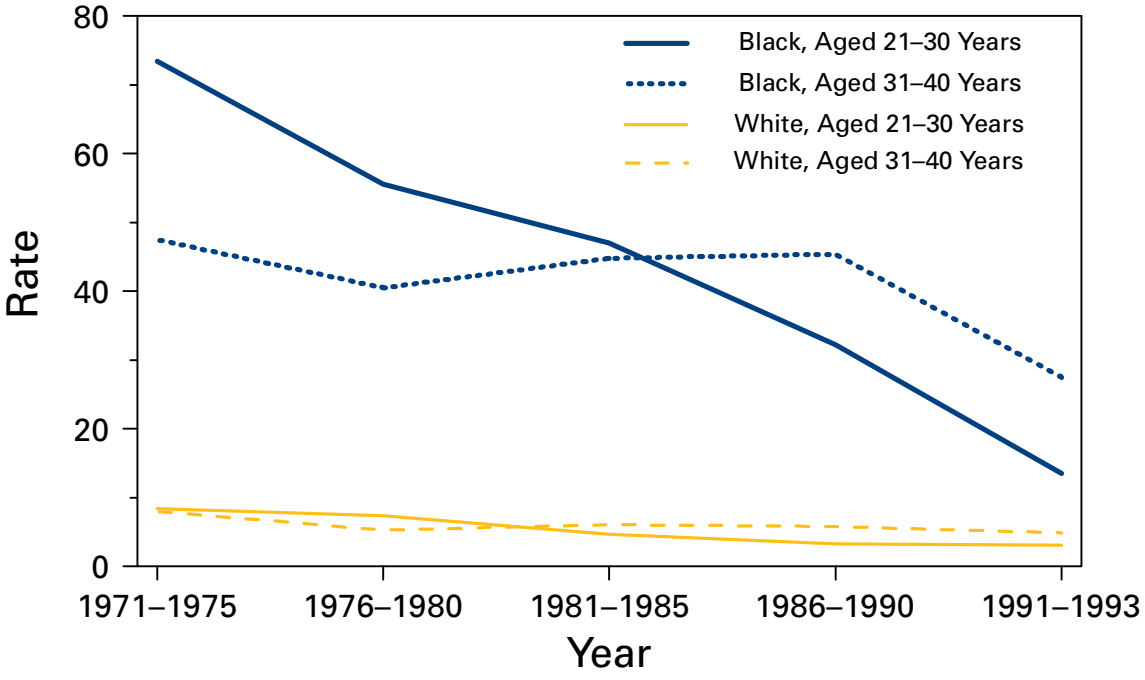
category); women were excluded because none had been assigned to aircraft carriers—an exposure of particular a priori interest—during 1965–1993.

During 1971–1993, the average annual age-specific incidence rate for sarcoidosis declined substantially among USN enlisted black men aged 21–30 years, from 73.3 per 100,000 to 13.2 per 100,000 (Figure 1). Rates for enlisted black men aged 31–40 years declined from 46.5 to 27.8, primarily during 1991–1993. During 1971–1993, rates for USN enlisted white men remained relatively stable.

To provide a basis for comparison with cases, a random sample of 10,000 controls was obtained from the NHRC records of enlisted personnel on active duty at any time during 1965–1993; of these, 9040 fulfilled the selection criteria of being men and either white or black. During this period (1965–1993), blacks accounted disproportionately for cases (47.8% of cases versus 11.4% of controls); in addition, USN men with sarcoidosis had served in the USN approximately twice as long as controls (mean tenure was 10.7 years [standard error (SE)=±0.2 years] versus 5.5 years [SE=±0.1 year], respectively) and had entered USN service an average of 5 years earlier (1971 [SE=±0.3] versus 1976 [SE=±0.1], respectively). Of the enlisted men in whom sarcoidosis had been diagnosed, 27% were discharged within 1 year of diagnosis.

Because specific codes for ship type and land assignment were generally not available until 1971, comparisons involving these variables were restricted to the 426 persons classified as cases and 4377 persons classified as controls who entered the USN after that date. Although 70% of case-patients and 66% of controls had ever served on ships, 26% of case-patients and 17% of controls had ever served specifically on aircraft

FIGURE 1. Average annual incidence rates* of sarcoidosis for U.S. Navy enlisted men, by race†, age group, and 5-year period§, 1971–1993



*Per 100,000 U.S. Navy enlisted men.
†Numbers for races other than black and white were too small for meaningful analysis.
§The period 1991–1993 comprises only 3 years.

Sarcoidosis — Continued

carriers. Additional analyses were conducted to examine the possible association of sarcoidosis with ship service and to control for potential confounders and investigate possible effect modification. For each case, a risk set was created that comprised all persons who had been born during the same 5-year period and who had survived without sarcoidosis beyond the age at which the corresponding case-patient had sarcoidosis diagnosed. Within each risk set, work history in the USN was truncated for persons in the control group when they reached the age at which their corresponding case-patient had sarcoidosis diagnosed. Cox regression with age as the time variable was conducted to examine associations between a USN diagnosis of sarcoidosis and the following variables: race, entry period of USN enlistment, type of ship assignment, and region of country where enlisted.[†] Region of enlistment was investigated because previous studies identified it as a predictor of sarcoidosis (*1*). The analysis indicated a statistically significant increased risk for blacks compared with whites (Table 1) and higher risks in earlier periods of entry, confirming patterns observed in the age-specific incidence rates (Figure 1). No association was identified between increased risk for sarcoidosis and ever having served on a ship; however, a statistically significant association was identified between increased risk for sarcoidosis and ever having served on USN aircraft carriers (risk ratio [RR]=1.5; 95% confidence interval [CI]=1.2–1.9). The RR for aircraft carrier service was higher for blacks (RR=1.7; 95% CI=1.3–2.3) than whites (RR=1.2; 95% CI=0.8–1.7), although the difference between these two RRs was not statistically significant. There was no indication that the risk for sarcoidosis was clustered around any specific aircraft carrier or period of entry. After adjusting for race, aircraft carrier exposure, and year of enlistment, an association was identified between increased risk for sarcoidosis and enlistment from the South Atlantic region (RR=2.1; 95% CI=1.6–2.7) and from the South Central region (RR=1.4; 95% CI=1.1–1.9)

[†] The states of enlistment were divided into four regions: Northeast, South Central, South Atlantic, and Other, with the included states as indicated. *Northeast*—Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont. *South Central*—Alabama, Arkansas, Kentucky, Louisiana, Mississippi, Oklahoma, Tennessee, and Texas. *South Atlantic*—Delaware, District of Columbia, Florida, Georgia, Maryland, North Carolina, South Carolina, Virginia, and West Virginia. *Other*—Alaska, Arizona, California, Colorado, Hawaii, Idaho, Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Montana, Nebraska, Nevada, New Mexico, North Dakota, Ohio, Oregon, South Dakota, Utah, Washington, Wisconsin, Wyoming, other U.S. possessions, and non-U.S. possessions.

TABLE 1. Regression analysis for diagnosis of sarcoidosis among U.S. Navy enlisted men who entered the service during 1971–1993

Model variables	Estimated coefficient	Standard error	p value	Risk ratio	(95% CI*)
Race[†]	2.10	(±0.10)	0.0001	8.19	(6.7–10.0)
Aircraft carrier	0.38	(±0.12)	0.00015	1.47	(1.2– 1.9)
Entry period 1[§]	0.54	(±0.37)	0.1417	1.72	(0.8– 3.5)
Entry period 2	0.45	(±0.35)	0.1983	1.57	(0.8– 3.1)
Entry period 3	0.48	(±0.31)	0.1271	1.61	(0.9– 3.0)

* Confidence interval.
[†] The races in this analysis are black and white. Numbers for other racial/ethnic groups were too small for meaningful analysis.
[§] Design variables that compare three time periods of entry to 1987–1993, based on 5-year intervals. Entry period 1=1972–1976; entry period 2=1977–1981; entry period 3=1982–1986.

Sarcoidosis — Continued

compared with the "Other" region. However, the effect of aircraft carrier assignment was neither confounded nor modified by region of enlistment.

Reported by: Div of Respiratory Disease Studies, and Div of Surveillance, Hazard Evaluation, and Field Studies, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Sarcoidosis is a multisystem granulomatous disease that typically presents with bilateral hilar lymphadenopathy, diffuse/nodular pulmonary infiltrates, and skin/ocular granulomata. The histology is characterized by noncaseating epithelioid-cell granulomas. Because there are no pathognomonic features, this definition is nonspecific, and sarcoidosis is a diagnosis of exclusion. Although the etiology of sarcoidosis is unknown, the epidemiology of the disease suggests that environmental or infectious agents could be contributory factors (*3*). For example, manifestations of sarcoidosis are more common during the winter and early spring. Health-care workers are disproportionately affected (*4,5*), and clusters of cases have been reported both in specific geographic regions (*1*) and among other occupational groups (e.g., firefighters) (*6*). Illnesses classified as sarcoidosis may represent a variety of discrete conditions with similar clinical presentations but varying etiologies, and other specific etiologies might be identified for what is currently reported as sarcoidosis. For example, a recent case report suggests an association between exposure to photocopier toner dust and sarcoidosis-like pulmonary disease (*7*). During the 1940s, several cases of "sarcoidosis" diagnosed among young women in the fluorescent light industry in Salem, Massachusetts, resulted in the recognition of beryllium exposure as a cause of "Salem sarcoid." Chronic beryllium disease is now considered a distinct diagnosis requiring specific immunologic testing (*8*). The higher risk for sarcoidosis among blacks (*1,2*) remains unexplained, and possible areas of further investigation include genetic predisposition and disproportionate exposure to environmental risk factors.

The limited data in this report indicate substantially higher sarcoidosis incidence rates for blacks than for whites enlisted in the USN and a clear decline in rates for blacks over time. The USN average annual sarcoidosis incidence rates per 100,000 for 1990–1993 (16.0 for black males and 2.5 for white males) were lower than average annual incidence rates for both black males (29.8) and white males (9.6) in a recently studied (1990–1994) population in Detroit, Michigan (*2*). However, reliable population-based rates over longer periods or for the United States are not available.

Reasons for the temporal changes in USN rates are unknown and could reflect unrecognized trends in the total U.S. population. The association of sarcoidosis with earlier entry period into the USN and the decline in incidence rates for blacks over time may indicate that exposures to etiologic factors (possibly including work-related exposures) were higher in the past and differentially affected blacks. The association of sarcoidosis with assignment to aircraft carriers also suggests an occupational factor, although ship assignment is only a crude surrogate for any specific exposures that might be causally related to the disease. However, secular changes in population characteristics, diagnostic and medical screening procedures, and case definition and diagnostic criteria may have affected the findings in this report and contributed to the observed decline in incidence of sarcoidosis. Such factors are particularly important for a disease such as sarcoidosis, which may be asymptomatic and remain undetected for long periods (*9,10*). In recent years, the USN has reduced the frequency of routine chest radiographs for enlisted personnel, which could explain some of the decline

Sarcoidosis — Continued

in sarcoidosis incidence rates over time but should not differentially affect rates for blacks and whites. The excess risk on aircraft carriers also may reflect increased detection rates from more frequent use of routine chest radiographs on aircraft carriers, which have large, well-equipped medical facilities. The findings in this report have been shared with the USN and the Department of Veterans Affairs; both are considering further action.

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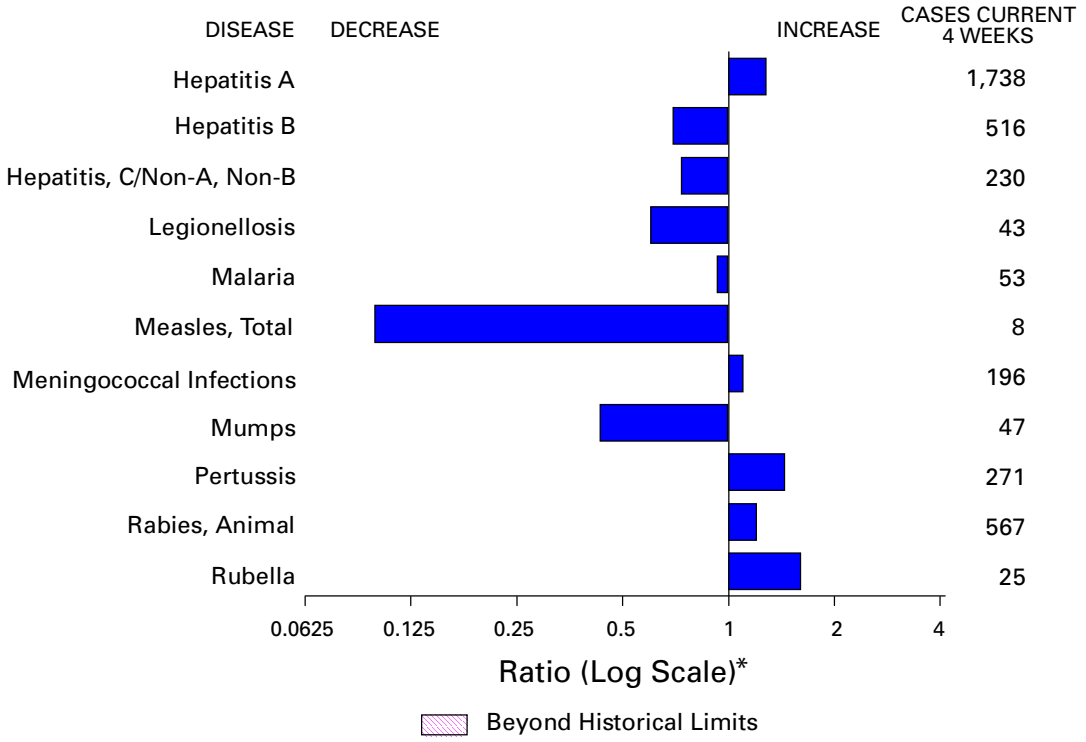
FIGURE I. Selected notifiable disease reports, comparison of provisional 4-week totals ending June 7, 1997, with historical data — United States



*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 June 7, 1997 (23rd Week)

	Cum. 1997		Cum. 1997
Anthrax	-	Plague	1
Brucellosis	21	Poliomyelitis, paralytic	-
Cholera	3	Psittacosis	19
Congenital rubella syndrome	2	Rabies, human	2
Cryptosporidiosis*	512	Rocky Mountain spotted fever (RMSF)	66
Diphtheria	4	Streptococcal disease, invasive Group A	599
Encephalitis: California*	4	Streptococcal toxic-shock syndrome*	16
eastern equine*	-	Syphilis, congenital¶	62
St. Louis*	1	Tetanus	15
western equine*	-	Toxic-shock syndrome	49
Hansen Disease	48	Trichinosis	4
Hantavirus pulmonary syndrome*†	5	Typhoid fever	124
Hemolytic uremic syndrome, post-diarrheal*	17	Yellow fever	-
HIV infection, pediatric*§	112		

-: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 May 27, 1997.
¶Updated from reports to the Division of STD Prevention, NCHSTP.

TABLE II. Provisional cases of selected notifiable diseases, United States, weeks ending June 7, 1997, and June 8, 1996 (23rd 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	25,284	28,368	173,088	187,383	497	217	108,249	132,965	1,331	1,572
NEW ENGLAND	903	1,115	7,166	7,264	38	18	2,390	2,674	24	43
Maine	25	16	428	158	2	-	25	8	-	-
N.H.	14	31	314	321	2	-	55	63	5	2
Vt.	18	9	178	198	3	1	24	26	-	14
Mass.	419	549	3,155	2,918	27	17	988	925	16	24
R.I.	71	73	917	906	1	-	213	230	3	3
Conn.	356	437	2,174	2,763	3	-	1,085	1,422	-	-
MID. ATLANTIC	8,301	7,896	23,110	30,732	31	4	13,313	18,521	145	123
Upstate N.Y.	1,358	1,001	N	N	19	3	2,306	3,188	112	93
N.Y. City	4,157	4,491	12,698	16,475	5	-	5,585	7,161	-	2
N.J.	1,773	1,509	3,050	6,226	7	-	1,796	3,815	-	-
Pa.	1,013	895	7,362	8,031	N	1	3,626	4,357	33	28
E.N. CENTRAL	1,687	2,301	25,657	38,748	83	27	15,791	24,926	246	234
Ohio	357	526	5,790	8,934	26	10	3,742	6,218	7	6
Ind.	329	342	3,652	4,264	15	5	2,504	2,866	7	6
Ill.	612	984	4,964	11,120	20	-	2,431	7,265	20	48
Mich.	306	318	8,235	9,641	22	4	5,816	6,449	212	174
Wis.	83	131	3,016	4,789	N	8	1,298	2,128	-	-
W.N. CENTRAL	469	678	9,979	13,981	70	42	4,633	6,922	84	38
Minn.	84	126	U	2,293	33	21	U	1,659	2	-
Iowa	67	51	2,048	1,833	15	8	550	464	26	16
Mo.	195	319	4,897	5,916	7	10	3,190	3,556	35	12
N. Dak.	5	7	365	425	3	2	23	12	2	-
S. Dak.	3	7	544	579	4	-	55	83	-	-
Nebr.	48	49	422	905	5	-	119	172	2	5
Kans.	67	119	1,703	2,030	3	1	696	976	17	5
S. ATLANTIC	6,203	7,254	36,456	24,436	63	19	35,623	41,512	123	76
Del.	111	142	-	-	1	2	477	634	-	-
Md.	734	850	3,207	2,715	3	1	5,795	5,455	9	1
D.C.	409	456	N	N	-	-	1,319	66	-	-
Va.	551	395	4,908	5,125	N	7	3,448	4,243	9	7
W. Va.	38	50	1,414	938	N	-	430	317	9	7
N.C.	361	360	7,438	U	17	9	7,005	8,359	27	20
S.C.	300	383	5,173	U	1	-	4,621	4,903	20	14
Ga.	850	1,085	4,031	5,351	19	-	5,305	9,737	U	-
Fla.	2,849	3,533	10,285	10,255	22	-	7,223	7,798	49	27
E.S. CENTRAL	810	951	14,287	13,154	40	7	14,073	13,916	154	277
Ky.	113	153	2,877	3,035	12	-	1,628	1,808	8	14
Tenn.	358	352	5,459	5,654	20	7	4,473	4,842	91	222
Ala.	194	277	3,389	3,720	5	-	4,801	5,711	5	2
Miss.	145	169	2,562	745	3	-	3,171	1,555	50	39
W.S. CENTRAL	2,596	2,635	20,895	9,473	26	4	13,774	8,858	167	119
Ark.	96	121	519	721	2	1	1,086	1,791	-	3
La.	476	647	3,468	3,026	4	3	3,279	3,196	96	72
Okla.	138	100	3,167	3,293	2	-	1,987	2,043	4	1
Tex.	1,886	1,767	13,741	2,433	18	-	7,422	1,828	67	43
MOUNTAIN	730	797	11,009	15,950	54	30	3,139	5,055	171	286
Mont.	18	10	450	550	3	-	18	13	7	9
Idaho	22	19	616	681	11	1	45	40	22	75
Wyo.	13	2	235	314	4	-	25	14	71	85
Colo.	180	245	1,896	866	19	10	751	774	20	25
N. Mex.	65	45	1,566	1,759	4	3	566	382	28	36
Ariz.	188	233	4,388	9,838	N	13	1,322	3,324	17	33
Utah	55	88	734	681	10	-	96	132	3	11
Nev.	189	155	1,124	1,261	3	3	316	376	3	12
PACIFIC	3,585	4,741	24,529	33,645	92	63	5,513	10,581	217	376
Wash.	288	362	4,048	4,438	18	4	862	989	12	29
Oreg.	144	223	1,618	2,477	29	34	249	245	4	4
Calif.	3,111	4,065	17,583	25,530	42	22	4,024	8,922	125	251
Alaska	16	11	602	405	3	-	183	195	-	2
Hawaii	26	80	678	795	N	3	195	230	76	90
Guam	2	3	31	197	N	-	3	31	-	5
P.R.	762	423	N	N	22	U	286	212	48	77
V.I.	36	9	N	N	N	U	-	-	-	-
Amer. Samoa	-	-	-	-	N	U	-	-	-	-
C.N.M.I.	1	-	N	N	N	U	16	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 May 27, 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 June 7, 1997, and June 8, 1996 (23rd 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	352	331	1,170	1,935	526	505	3,515	5,165	6,839	7,799	3,133
NEW ENGLAND	24	18	233	274	17	14	68	69	175	177	484
Maine	1	1	3	3	1	3	-	-	11	12	100
N.H.	4	-	7	5	1	1	-	1	6	5	20
Vt.	3	2	3	1	2	2	-	-	3	-	83
Mass.	8	9	57	17	11	5	37	32	94	74	97
R.I.	4	6	37	21	2	3	1	1	13	20	11
Conn.	4	N	126	227	-	-	30	35	48	66	173
MID. ATLANTIC	56	74	708	1,443	131	150	161	240	1,338	1,368	674
Upstate N.Y.	13	17	108	681	25	29	17	35	180	146	501
N.Y. City	-	4	9	74	64	82	33	74	713	704	-
N.J.	7	7	167	195	30	29	65	82	268	296	65
Pa.	36	46	424	493	12	10	46	49	177	222	108
E.N. CENTRAL	126	115	22	19	36	63	309	892	743	835	61
Ohio	70	40	17	9	6	6	104	344	143	126	46
Ind.	20	29	5	7	5	6	71	117	61	84	8
Ill.	-	14	-	3	5	30	25	244	380	462	2
Mich.	31	21	-	-	17	11	59	92	111	125	5
Wis.	5	11	U	U	3	10	50	95	48	38	-
W.N. CENTRAL	32	19	11	46	17	13	58	194	194	214	188
Minn.	1	1	9	1	5	3	U	22	48	53	17
Iowa	9	2	-	6	8	2	3	13	20	26	72
Mo.	6	5	-	19	2	6	36	141	85	83	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	-	20	1	2	18	12	29	24	43
S. ATLANTIC	55	37	121	78	129	80	1,450	1,685	1,346	1,411	1,321
Del.	4	2	6	40	2	2	14	17	11	22	30
Md.	15	5	85	8	42	21	393	275	131	109	234
D.C.	3	3	6	1	7	4	41	8	43	65	2
Va.	9	11	2	2	25	11	128	210	111	118	272
W. Va.	-	1	-	4	-	1	1	2	24	26	37
N.C.	6	3	7	16	6	10	320	485	166	182	406
S.C.	2	3	1	2	7	3	178	205	144	153	67
Ga.	-	-	1	-	12	8	242	312	233	296	124
Fla.	16	9	13	5	28	20	133	171	483	440	149
E.S. CENTRAL	12	21	29	28	14	13	825	1,203	463	610	121
Ky.	1	2	4	9	3	3	73	63	88	104	13
Tenn.	6	8	10	7	4	5	347	400	120	210	75
Ala.	1	2	4	1	4	2	214	249	175	188	33
Miss.	4	9	11	11	3	3	191	491	80	108	-
W.S. CENTRAL	6	2	11	12	5	11	472	512	781	948	124
Ark.	-	-	1	6	1	-	59	126	87	82	22
La.	1	-	1	-	4	1	176	245	-	3	1
Okla.	2	2	4	2	-	-	55	67	70	65	54
Tex.	3	-	5	4	-	10	182	74	624	798	47
MOUNTAIN	22	19	3	1	32	28	71	96	238	240	46
Mont.	1	1	-	-	2	2	-	-	7	7	9
Idaho	2	-	-	-	-	-	-	1	5	4	-
Wyo.	1	2	1	1	1	2	-	1	2	3	15
Colo.	4	6	1	-	15	14	2	17	50	43	-
N. Mex.	1	1	-	-	5	1	-	-	16	39	4
Ariz.	7	4	1	-	4	3	59	72	102	91	17
Utah	5	1	-	-	2	4	3	1	10	10	-
Nev.	1	4	-	-	3	2	7	4	46	43	1
PACIFIC	19	26	32	34	145	133	101	274	1,561	1,996	114
Wash.	5	1	1	1	8	7	6	3	90	118	-
Oreg.	-	-	9	9	10	9	3	5	66	78	2
Calif.	13	25	22	23	123	111	90	265	1,292	1,689	95
Alaska	-	-	-	-	2	2	1	-	36	39	17
Hawaii	1	-	-	1	2	4	1	1	77	72	-
Guam	-	1	-	-	-	-	-	3	5	45	-
P.R.	-	-	-	-	3	-	97	116	88	38	25
V.I.	-	-	-	-	-	-	-	-	-	-	-
Amer. Samoa	-	-	-	-	-	-	-	-	-	-	-
C.N.M.I.	-	-	-	-	-	-	5	1	-	-	-

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

TABLE III. Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending June 7, 1997, and June 8, 1996 (23rd 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	508	664	11,978	11,889	3,725	4,021	1	34	-	17	51	191
NEW ENGLAND	29	14	250	140	72	87	-	-	-	-	-	10
Maine	3	1	37	11	5	2	-	-	-	-	-	-
N.H.	2	8	17	5	5	7	-	-	-	-	-	-
Vt.	1	-	6	3	2	7	-	-	-	-	-	1
Mass.	20	5	108	69	41	21	-	-	-	-	-	8
R.I.	2	-	24	6	8	6	-	-	-	-	-	-
Conn.	1	-	58	46	11	44	-	-	-	-	-	1
MID. ATLANTIC	56	114	864	782	494	660	-	7	-	4	11	12
Upstate N.Y.	3	28	119	172	97	151	-	1	-	3	4	4
N.Y. City	18	27	298	268	166	245	-	4	-	1	5	7
N.J.	25	32	157	170	111	131	-	1	-	-	1	-
Pa.	10	27	290	172	120	133	-	1	-	-	1	1
E.N. CENTRAL	71	91	1,238	1,091	400	493	-	4	-	2	6	15
Ohio	42	49	189	422	41	57	-	-	-	-	-	2
Ind.	8	6	138	152	42	67	-	-	-	-	-	-
Ill.	13	25	234	257	82	141	-	4	-	1	5	2
Mich.	7	6	606	161	221	183	-	-	-	1	1	2
Wis.	1	5	71	99	14	45	-	-	-	-	-	9
W.N. CENTRAL	22	19	887	881	247	207	-	9	-	2	11	16
Minn.	12	10	72	48	18	19	-	-	-	2	2	14
Iowa	3	3	132	186	39	23	-	-	-	-	-	-
Mo.	3	3	461	449	164	129	-	1	-	-	1	1
N. Dak.	-	-	9	22	1	-	-	-	-	-	-	-
S. Dak.	2	1	12	37	-	-	-	8	-	-	8	-
Nebr.	1	1	70	64	8	14	-	-	-	-	-	-
Kans.	1	1	131	75	17	22	-	-	-	-	-	1
S. ATLANTIC	110	96	696	435	530	525	1	1	-	3	4	4
Del.	-	1	11	6	3	2	-	-	-	-	-	1
Md.	41	32	125	90	79	73	-	-	-	1	1	-
D.C.	2	5	14	15	21	15	-	-	-	1	1	-
Va.	6	4	79	66	55	65	-	-	-	-	-	2
W. Va.	3	4	6	10	8	14	-	-	-	-	-	-
N.C.	15	14	95	54	108	155	-	-	-	1	1	-
S.C.	4	3	58	29	48	40	-	-	-	-	-	-
Ga.	18	26	117	15	47	7	-	-	-	-	-	-
Fla.	21	7	191	150	161	154	1	1	-	-	1	1
E.S. CENTRAL	34	17	299	758	305	374	-	-	-	-	-	-
Ky.	5	5	35	16	15	36	-	-	-	-	-	-
Tenn.	21	6	180	536	191	222	-	-	-	-	-	-
Ala.	8	5	49	97	31	24	-	-	-	-	-	-
Miss.	-	1	35	109	68	U	U	-	U	-	-	-
W.S. CENTRAL	28	23	2,613	1,911	492	342	-	3	-	1	4	2
Ark.	1	-	128	226	28	38	-	-	-	-	-	-
La.	6	1	100	58	54	51	-	-	-	-	-	-
Okla.	16	20	770	876	14	22	-	-	-	-	-	-
Tex.	5	2	1,615	751	396	231	-	3	-	1	4	2
MOUNTAIN	49	29	1,831	1,880	416	485	-	5	-	-	5	22
Mont.	-	-	50	60	5	4	-	-	-	-	-	-
Idaho	1	1	73	127	16	58	-	-	-	-	-	1
Wyo.	-	-	18	19	18	14	-	-	-	-	-	-
Colo.	7	5	209	167	85	59	-	-	-	-	-	6
N. Mex.	6	7	143	225	142	158	-	-	-	-	-	-
Ariz.	16	11	895	702	86	112	-	5	-	-	5	8
Utah	3	5	338	416	47	54	-	-	-	-	-	3
Nev.	16	-	105	164	17	26	-	-	-	-	-	4
PACIFIC	109	261	3,300	4,011	769	848	-	5	-	5	10	110
Wash.	2	2	238	252	31	48	-	-	-	-	-	37
Oreg.	19	19	171	523	54	53	-	-	-	-	-	4
Calif.	82	234	2,811	3,167	666	740	-	2	-	5	7	4
Alaska	1	4	20	28	12	2	-	-	-	-	-	63
Hawaii	5	2	60	41	6	5	-	3	-	-	3	2
Guam	-	-	-	4	1	-	U	-	U	-	-	-
P.R.	-	1	157	101	592	466	-	-	-	-	-	1
V.I.	-	-	-	22	-	18	U	-	U	-	-	-
Amer. Samoa	-	-	-	-	-	-	U	-	U	-	-	-
C.N.M.I.	5	10	1	1	21	5	U	1	U	-	1	-

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

*Of 111 cases among children aged <5 years, serotype was reported for 54 and of those, 20 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 June 7, 1997, and June 8, 1996 (23rd 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	1,790	1,714	6	285	324	63	2,167	1,579	8	46	102
NEW ENGLAND	110	66	-	7	-	7	468	314	-	-	23
Maine	11	9	-	-	-	-	6	10	-	-	-
N.H.	10	2	-	-	-	3	61	19	-	-	-
Vt.	2	3	-	-	-	1	163	10	-	-	2
Mass.	59	23	-	2	-	3	221	272	-	-	19
R.I.	7	7	-	4	-	-	12	-	-	-	-
Conn.	21	22	-	1	-	-	5	3	-	-	2
MID. ATLANTIC	153	182	-	26	48	-	162	104	-	3	5
Upstate N.Y.	41	41	-	4	12	-	51	52	-	1	3
N.Y. City	27	28	-	-	13	-	40	16	-	2	1
N.J.	32	37	-	-	2	-	5	6	-	-	1
Pa.	53	76	-	22	21	-	66	30	-	-	-
E.N. CENTRAL	249	245	-	31	80	7	166	215	-	2	3
Ohio	101	84	-	13	27	-	65	72	-	-	-
Ind.	31	36	-	4	5	5	27	12	-	-	-
Ill.	73	72	-	7	15	-	24	53	-	-	1
Mich.	26	27	-	7	32	2	30	13	-	-	2
Wis.	18	26	-	-	1	-	20	65	-	2	-
W.N. CENTRAL	128	125	-	9	5	8	123	65	-	-	-
Minn.	12	14	-	3	1	8	82	42	-	-	-
Iowa	26	25	-	4	-	-	15	3	-	-	-
Mo.	69	54	-	-	2	-	16	13	-	-	-
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	326	258	1	41	42	13	198	149	8	21	12
Del.	4	2	-	-	-	-	-	13	-	-	-
Md.	31	27	-	4	15	2	72	54	-	-	-
D.C.	1	6	-	-	-	-	2	-	-	-	1
Va.	29	31	-	4	3	-	19	17	-	1	-
W. Va.	12	10	1	1	-	-	4	2	-	-	-
N.C.	55	44	-	7	8	6	46	27	-	10	-
S.C.	40	35	-	9	5	-	8	2	8	9	1
Ga.	65	74	-	4	2	-	7	7	-	-	-
Fla.	89	29	-	12	9	5	40	27	-	1	10
E.S. CENTRAL	136	126	-	15	14	1	37	131	-	-	-
Ky.	35	19	-	2	-	-	2	112	-	-	-
Tenn.	47	36	-	3	1	1	15	12	-	-	-
Ala.	38	36	-	6	3	-	12	4	-	-	-
Miss.	16	35	U	4	10	U	8	3	U	-	N
W.S. CENTRAL	193	204	-	29	27	6	38	46	-	4	7
Ark.	24	26	-	-	-	2	7	2	-	-	-
La.	33	35	-	7	10	4	11	4	-	-	1
Okla.	22	19	-	-	-	-	5	4	-	-	-
Tex.	114	124	-	22	17	-	15	36	-	4	6
MOUNTAIN	104	104	4	38	14	13	662	160	-	4	6
Mont.	8	4	-	-	-	1	7	5	-	-	-
Idaho	7	12	-	2	-	9	489	58	-	1	2
Wyo.	-	3	-	1	-	-	4	-	-	-	-
Colo.	30	17	-	3	2	1	113	27	-	-	2
N. Mex.	18	20	N	N	N	1	32	29	-	-	-
Ariz.	23	26	3	25	1	1	11	12	-	3	1
Utah	12	10	1	5	2	-	4	5	-	-	-
Nev.	6	12	-	2	9	-	2	24	-	-	1
PACIFIC	391	404	1	89	94	8	313	395	-	12	46
Wash.	50	49	1	12	9	4	163	150	-	-	8
Oreg.	82	70	-	1	-	-	16	26	-	-	1
Calif.	256	279	-	65	69	4	127	208	-	7	35
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	-	1	U	-	-	U	-	-
Amer. Samoa	-	-	U	-	-	U	-	-	U	-	-
C.N.M.I.	-	-	U	4	-	U	-	-	U	-	-

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

TABLE IV. Deaths in 122 U.S. cities,* week ending June 7, 1997 (23rd Week)

Reporting Area	All Causes, By Age (Years)						P&I† Total
	All Ages	>65	45-64	25-44	1-24	<1	
NEW ENGLAND	581	396	109	41	21	14	43
Boston, Mass.	157	110	34	11	2	-	19
Bridgeport, Conn.	22	15	3	2	2	-	-
Cambridge, Mass.	20	14	5	1	-	-	1
Fall River, Mass.	22	15	4	1	2	-	2
Hartford, Conn.	64	33	14	10	3	4	3
Lowell, Mass.	24	21	3	-	-	-	4
Lynn, Mass.	12	7	2	2	-	1	-
New Bedford, Mass.	17	11	6	-	-	-	2
New Haven, Conn.	32	19	8	-	2	3	1
Providence, R.I.	66	47	8	4	4	3	3
Somerville, Mass.	9	8	1	-	-	-	-
Springfield, Mass.	57	42	7	4	2	2	-
Waterbury, Conn.	26	18	5	1	2	-	2
Worcester, Mass.	53	36	9	5	2	1	6
MID. ATLANTIC	2,536	1,734	480	203	69	50	125
Albany, N.Y.	43	25	13	1	-	4	2
Allentown, Pa.	25	21	4	-	-	-	-
Buffalo, N.Y.	58	36	12	4	4	2	1
Camden, N.J.	44	32	6	1	4	1	2
Elizabeth, N.J.	19	9	6	3	-	1	1
Erie, Pa.	49	36	8	4	1	-	-
Jersey City, N.J.	30	16	5	5	2	2	-
New York City, N.Y.	1,195	816	237	96	27	19	48
Newark, N.J.	50	22	16	9	2	1	5
Paterson, N.J.	28	18	4	6	-	-	-
Philadelphia, Pa.	600	400	113	56	23	8	42
Pittsburgh, Pa.§	59	46	7	-	2	4	1
Reading, Pa.	8	5	2	1	-	-	-
Rochester, N.Y.	124	95	18	6	2	3	11
Schenectady, N.Y.	18	15	1	2	-	-	-
Scranton, Pa.	34	27	3	4	-	-	1
Syracuse, N.Y.	91	65	18	3	2	3	8
Trenton, N.J.	20	15	2	1	-	2	-
Utica, N.Y.	18	16	2	-	-	-	1
Yonkers, N.Y.	23	19	3	1	-	-	2
E.N. CENTRAL	2,194	1,512	413	172	49	46	140
Akron, Ohio	79	56	14	3	3	3	-
Canton, Ohio	36	29	6	1	-	-	1
Chicago, Ill.	409	243	101	46	4	13	30
Cincinnati, Ohio	105	79	15	4	4	3	5
Cleveland, Ohio	164	100	45	15	3	1	3
Columbus, Ohio	171	120	30	12	6	3	16
Dayton, Ohio	129	91	21	12	3	2	12
Detroit, Mich.	189	108	36	28	8	9	5
Evansville, Ind.	48	37	4	7	-	-	4
Fort Wayne, Ind.	81	63	16	2	-	-	4
Gary, Ind.	15	9	6	-	-	-	1
Grand Rapids, Mich.	60	40	12	4	2	2	8
Indianapolis, Ind.	225	165	36	15	5	4	12
Lansing, Mich.	46	33	6	3	3	1	6
Milwaukee, Wis.	118	93	18	4	1	2	11
Peoria, Ill.	50	37	10	2	1	-	2
Rockford, Ill.	55	40	9	4	1	1	3
South Bend, Ind.	56	43	9	3	1	-	3
Toledo, Ohio	89	69	13	4	3	-	11
Youngstown, Ohio	69	57	6	3	1	2	3
W.N. CENTRAL	646	474	98	35	19	19	40
Des Moines, Iowa	5	5	-	-	-	-	1
Duluth, Minn.	26	21	3	1	-	1	1
Kansas City, Kans.	38	24	4	6	3	1	-
Kansas City, Mo.	75	57	10	3	2	2	5
Lincoln, Nebr.	40	29	8	2	-	1	5
Minneapolis, Minn.	137	107	20	5	4	1	10
Omaha, Nebr.	82	54	18	2	3	5	6
St. Louis, Mo.	99	73	12	7	3	4	9
St. Paul, Minn.	64	47	9	5	2	1	3
Wichita, Kans.	80	57	14	4	2	3	-

Reporting Area	All Causes, By Age (Years)						P&I† Total
	All Ages	>65	45-64	25-44	1-24	<1	
S. ATLANTIC	1,244	756	271	151	32	31	70
Atlanta, Ga.	172	86	45	22	7	12	6
Baltimore, Md.	250	142	55	41	7	3	23
Charlotte, N.C.	90	55	19	10	3	2	7
Jacksonville, Fla.	143	94	29	13	3	4	6
Miami, Fla.	77	41	17	17	2	-	-
Norfolk, Va.	62	35	15	7	2	3	3
Richmond, Va.	74	55	8	8	1	2	1
Savannah, Ga.	29	17	8	4	-	-	2
St. Petersburg, Fla.	66	48	9	6	2	1	7
Tampa, Fla.	165	112	39	8	3	3	12
Washington, D.C.	102	65	19	15	2	1	3
Wilmington, Del.	14	6	8	-	-	-	-
E.S. CENTRAL	794	573	143	46	24	8	37
Birmingham, Ala.	173	118	35	15	3	2	17
Chattanooga, Tenn.	96	73	17	4	2	-	1
Knoxville, Tenn.	102	80	11	5	4	2	2
Lexington, Ky.	61	50	8	2	1	-	4
Memphis, Tenn.	90	66	17	5	2	-	6
Mobile, Ala.	91	61	19	5	4	2	-
Montgomery, Ala.	26	25	1	-	-	-	1
Nashville, Tenn.	155	100	35	10	8	2	6
W.S. CENTRAL	1,334	845	299	122	35	32	80
Austin, Tex.	42	24	7	6	3	2	2
Baton Rouge, La.	87	60	18	5	2	2	3
Corpus Christi, Tex.	40	24	12	3	1	-	4
Dallas, Tex.	194	110	47	25	7	5	4
El Paso, Tex.	64	45	11	5	3	-	4
Ft. Worth, Tex.	114	79	21	5	3	6	7
Houston, Tex.	356	223	86	37	5	5	26
Little Rock, Ark.	63	36	18	4	1	3	3
New Orleans, La.	U	U	U	U	U	U	U
San Antonio, Tex.	185	119	43	18	2	3	14
Shreveport, La.	51	31	14	2	3	1	3
Tulsa, Okla.	138	94	22	12	5	5	10
MOUNTAIN	923	608	183	85	28	18	64
Albuquerque, N.M.	107	74	17	10	4	2	1
Boise, Idaho	36	26	6	2	-	2	2
Colo. Springs, Colo.	52	29	13	9	-	1	3
Denver, Colo.	91	63	20	5	2	1	10
Las Vegas, Nev.	185	115	40	20	7	3	13
Ogden, Utah	25	22	-	3	-	-	2
Phoenix, Ariz.	186	117	40	19	6	3	10
Pueblo, Colo.	17	12	4	1	-	-	1
Salt Lake City, Utah	109	77	17	9	4	2	9
Tucson, Ariz.	115	73	26	7	5	4	13
PACIFIC	1,542	1,051	300	129	32	30	121
Berkeley, Calif.	14	7	5	2	-	-	1
Fresno, Calif.	86	60	13	6	1	6	3
Glendale, Calif.	26	22	4	-	-	-	2
Honolulu, Hawaii	75	53	15	3	1	3	5
Long Beach, Calif.	79	50	19	7	-	3	17
Los Angeles, Calif.	475	332	86	36	12	9	29
Pasadena, Calif.	U	U	U	U	U	U	U
Portland, Oreg.	88	53	25	9	1	-	6
Sacramento, Calif.	144	98	29	12	5	-	17
San Diego, Calif.	152	100	35	13	3	1	15
San Francisco, Calif.	111	74	21	12	2	2	10
San Jose, Calif.	U	U	U	U	U	U	U
Santa Cruz, Calif.	25	18	6	1	-	-	4
Seattle, Wash.	149	98	27	15	5	4	4
Spokane, Wash.	47	33	9	4	1	-	3
Tacoma, Wash.	71	53	6	9	1	2	5
TOTAL	11,794¶	7,949	2,296	984	309	248	720

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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