C

MMWR

Recommendations and Reports

April 27, 1990 / 39(RR-5);1-24

Viral Agents of Gastroenteritis Public Health Importance and Outbreak Management

This report was prepared by: Charles W. LeBaron, M.D. Navid P. Furutan, M.D.* Judy F. Lew, M.D. James R. Allen, Ph.D. Vera Gouvea, Ph.D. Christine Moe, Ph.D. Stephan S. Monroe, Ph.D. *Department of Pediatrics, Emory University Summary Each year, infectious gastroenteritis causes greater than 210,000 children in the United States to be hospitalized and 4-10 million children to die worldwide. Since the mid-1970s, knowledge has increased dramatically concerning the viral agents that are responsible for much of this public health burden. Rotavirus, the most common cause of diarrhea among children, infects virtually every child in the United States by the age of 4 years and causes potentially lethal dehydration in 0.75% of children less than 2 years of age. Other recently identified pathogens include the enteric adenoviruses, calicivirus, astrovirus, and the Norwalk family of agents. Conclusive diagnosis of these viruses requires electron microscopic examination of stool specimens, a laboratory technique that is available only at a few large centers, including CDC. Stool samples from an outbreak that are submitted to CDC for detection of viral pathology should be collected in bulk from 10 ill persons during their first 48 hours of illness, while feces are still liquid, and should be stored at 4 C (not frozen). Acute- and convalescent-phase serum samples should be collected from the same persons, plus from an equal number of controls, during the first week of illness and 3 weeks thereafter. Control measures for outbreaks of viral gastroenteritis should focus on the removal of an ongoing common source of infection (e.g., an ill food handler or the contamination of a water supply) and on the interruption of person-to-person transmission that can perpetuate an outbreak in a population after the common source has been removed. Because improvements in environmental hygiene may not be accompanied by reductions of endemic diarrhea caused by viruses, immunization may play an important role in future control; vaccine trials for rotavirus are in progress. In anticipation of vaccine development and use, CDC recently began national surveillance for the viral agents of gastroenteritis. Health-care facilities involved in the detection of rotavirus or the other viral agents of diarrhea can participate. INTRODUCTION

The public health burden of infectious diarrhea is substantial, particularly among children, both in the United States and worldwide. Each year in the United States greater than 210,000 children less than 5 years of age are hospitalized for gastroenteritis for an average of 4.5 days, at an annual inpatient cost of almost $1 billion (1). During the period 1973-1983, an average of 500 children in the United States died from diarrhea each year (2). Also, 25 work or school days/100 children are lost each year as a result of acute gastroenteritis (3), and approximately 14% of children in the United States are treated by a physician for rotavirus diarrhea alone (CDC, unpublished data). Worldwide, 3-5 billion cases of diarrhea occur, causing 5-10 million deaths annually (4).

Until the 1970s, diagnostic techniques for infectious diarrhea were limited to bacteria and protozoa, and an etiologic agent could be identified in a limited proportion of cases. Investigators had hypothesized, however, that viruses might account for many of the cases of unknown etiology. In 1972, in the examination of stool specimens, electron microscopy identified the Norwalk agent, the most common viral cause of gastroenteritis outbreaks among adults. In 1978, the same technique was used to detect rotavirus, the most common cause of severe diarrhea in children. Since that time, knowledge about these and other more recently discovered pathogens has increased dramatically. Unfortunately, diagnostic technology is insufficiently developed to permit determining the disease burden of each of the known viral pathogens. Even when diagnostic efforts are pursued aggressively, an agent cannot be identified for almost half of diarrheal cases (5).

Current priorities in enteric viral research include: 1) improving diagnostic capabilities for known pathogens to determine their endemic importance and their role in outbreaks; 2) identifying new agents for the 50% of diarrhea cases that are still of unknown etiology; and 3) determining the modes of transmission and the means to prevent disease, including the characteristics of natural immunity and effective vaccines.

As a result of rapid progress in these areas, etiologic identification may soon be possible in most cases of diarrhea, and within a few years a rotavirus vaccine may be licensed. Thus, public health and other health-care professionals should be familiar with the viral agents of gastroenteritis, their role in endemic disease, methods of diagnosis, and measures to manage outbreaks.

This document was prepared by the Viral Gastroenteritis Section, Respiratory and Enterovirus Branch (REB), Division of Viral and Rickettsial Diseases (DVRD), Center for Infectious Diseases (CID), and is intended primarily for the use of state and local health departments who investigate outbreaks. It may also be of use in academic settings, research groups, or other groups interested in

studying outbreaks of gastroenteritis. PATHOGENS: EPIDEMIOLOGIC AND CLINICAL FEATURES Rotavirus Endemic disease

Rotavirus is the most common cause of severe diarrhea among children. In the United States, approximately 3.5 million cases occur each year. A child has a 2% lifetime chance of being hospitalized for rotavirus diarrhea, which accounts for 35% of diarrheal hospital stays (1) and an estimated 75-125 childhood deaths annually (2). Worldwide, an estimated 140 million cases occur each year, causing almost 1 million deaths (6).

In the United States, the peak incidence of rotavirus diarrhea is among children 6 months-2 years of age, although in developing countries younger infants may be affected. By 4 years of age, most persons have been infected and are immune to the severe dehydrating syndrome, but a high inoculum or lowered immunity can produce milder illness among older children or adults. One-third of parents whose children are infected with rotavirus become ill (7), and rotavirus diarrhea can occur among travelers to developing nations, the elderly, and persons with debilitative or immunosuppressive conditions. In the United States, rotavirus activity is concentrated in the cooler months of the year (October-April). Clinical syndrome

In general, the incubation period is approximately 2 days and is followed by vomiting for 3 days and watery diarrhea for 3-8 days. Fever and abdominal pain occur frequently. Usually, there are no sequelae other than temporary intolerance of lactose; however, without adequate fluid replacement the syndrome can result in severe dehydration and death. Modes of transmission

A person with rotavirus diarrhea may excrete approximately 1 trillion infectious particles/milliliter of stool. Since the infective dose in a child can be as few as 10 particles (8), person-to-person transmission probably perpetuates endemic disease. Infectivity does not parallel the presence of symptoms. Asymptomatic rotavirus excretion has been reported among half of children the day before diarrhea starts and among one-third during the week after symptoms end (9). Many children can shed rotavirus and never become ill (10,11). When food or water is contaminated to the extent that it overcomes adult immunity, rotavirus diarrhea among adults may be relatively common. In Thailand, 5% of adult gastroenteritis has been found to be caused by rotavirus (12). Outbreak characteristics

Nosocomial rotavirus among pediatric populations is common; in one study, all children hospitalized for greater than 2 weeks during rotavirus season ultimately shed the virus (13). Rotavirus at day-care centers, in both endemic and outbreak form, is also common (9,14,15). Outbreaks in neonatal units are frequently reported, but infection among full-term infants is usually benign, perhaps because maternal antibody transferred during the third trimester protects against illness for the first 3-6 months of life; premature infants are at higher risk. Among adults, an outbreak arising from rotavirus contamination of a municipal water supply has been reported (16), and foodborne transmission was suspected in two other outbreaks involving banquets (CDC, unpublished data). Immunity

Infection generally confers long-term immunity to serious gastroenteritis (17), but asymptomatic or minimally symptomatic reinfection can occur throughout life. Immunity may wane among the elderly, rendering them susceptible again to severe disease. Atypical rotaviruses

Rotaviruses affecting humans were once thought to be limited to one antigenic family termed Group A, whereas other antigenic groups (B-E) were thought to be strictly zoonotic. In 1982, however, an epidemic of Group B rotavirus affected millions of persons in China (including adults, children, and neonates) (18), and since then outbreaks have recurred, although affecting fewer persons. Studies of immunoglobulin pools from Shanghai suggest that the Chinese population had been exposed to this pathogen in the past (19). Since Group B rotavirus is a common diarrheal pathogen for swine, and since all rotaviruses have a segmented genome (similar to influenza) capable of antigenic changes through reassortment of genes, investigators have hypothesized that this human Group B epidemic arose by a reassortment that allowed the swine virus to propagate in the human gut (20). Thus far, the Group B epidemic is not known to have spread beyond China, despite serologic evidence of human infection in Southeast Asia. Group C rotaviruses are also primarily swine pathogens but have been detected among humans in all parts of the world. Outbreaks have occurred in Japan and England, but the importance of Group C rotaviruses in endemic disease is unknown (21,22). Although both Group B and Group C rotaviruses in humans have been reported in the United States, serum studies suggest that exposure has been minimal in the past. With the U.S. population almost wholly susceptible, the extent to which these atypical rotaviruses represent a potential public health hazard in the United States is unclear. Adenovirus Endemic disease

Adenoviruses are widely recognized causes of respiratory, ocular, and genitourinary infections. However, serotypes 40 and 41 (previously called fastidious enteric adenoviruses) primarily affect the gut, contributing to 5%-20% of hospitalizations for childhood diarrhea in developed countries (23,24). Peak incidence is among children less than 2 years of age, but older children and adults may be infected, with or without symptoms. Infections occur throughout the year with no clear peaks (25). Other serotypes of adenovirus, particularly 31, have also been associated with diarrhea (26). Clinical syndrome

Incubation is between 3 and 10 days, with illness lasting greater than or equal to 1 week, longer than for other enteric viral pathogens (23,27). Diarrhea is more prominent than vomiting or fever, and respiratory symptoms are often present. Modes of transmission

Person-to-person transmission is presumably the principal mechanism for the spread of infection. Asymptomatic shedding has been documented, but generally infectivity parallels symptomatic disease (25). Food and water have not been reported as vehicles. Outbreak characteristics

Reported outbreaks have tended to occur in hospitals or day-care settings, and all have involved children. Adult contacts were infrequently affected (28,29). Immunity

Long-term immunity is thought to be acquired during childhood infection. Calicivirus Endemic disease

A British study suggested that approximately 3% of children hospitalized for diarrhea excrete calicivirus (30), and a U.S. study found approximately the same percentage (2.9%) for children with diarrhea in day-care centers (31). On the basis of antibody-prevalence studies of pooled immunoglobulin and serum samples from many parts of the world, most persons appear to have been infected by age 12, and the peak acquisition takes place between 3 months and 6 years (32,33). Seasonality is unknown. Clinical syndrome

The incubation period is 1-3 days, with illness lasting an average of 4 days. Vomiting and diarrhea are common, with upper respiratory symptoms and fever occurring less frequently. Infections in the elderly have also been documented. Modes of transmission

Person-to-person transmission is presumed to be essential for endemic disease, but contaminated shellfish, cold foods, and drinking water have been implicated as vehicles (34). Outbreak characteristics

Of seven calicivirus outbreaks reported in the literature since 1979, all occurred in institutional settings. Four outbreaks affected children: one in an orphanage and another at a school in Japan (35,36), and one in an infant-mother hospital unit and another at a school in England (37,38). Three outbreaks involved the elderly in nursing-home settings in England and Japan (39-41). Attack rates ranged from 50% to 70%. No calicivirus outbreak in the United States has been reported. Immunity

In the reported outbreaks, mothers of infected infants were rarely infected, suggesting that young adults retain effective immunity from earlier exposures, although the outbreaks among the elderly suggest that this immunity may wane with age. Astrovirus Endemic disease

Studies of hospitalized children suggest that astroviruses may account for 3%-5% of admissions for diarrhea (30). Children less than 7 years of age are principally affected, although adults can be infected and suffer mild disease. Antibody to all five serotypes of astrovirus was present in a study of pooled American gamma globulin from the United States (CDC, unpublished data), and 75% of British children have acquired antibody by the age of 10 years (42). Australian data suggest a winter peak (43). Clinical syndrome

The incubation period is between 24 and 36 hours, with illness lasting 1-4 days. Gastrointestinal symptoms are nonspecific, consisting of vomiting, diarrhea, fever, and abdominal pain. Astrovirus in ducks has been associated with hepatitis, but hepatic involvement has not been reported for humans. Modes of transmission

Although most transmission is probably person-to-person among children, contaminated water and shellfish have given rise to outbreaks in Britain (44). Asymptomatic shedding has been documented (45). Outbreak characteristics

Institutional settings account for four reported astroviral outbreaks from Britain and Japan: a kindergarten (46), a pediatric ward (47), and two nursing homes (39,48). In the kindergarten, a 50% attack rate was reported, with secondary transmission of the illness to families occurring in one-third of the cases. Immunity

The characteristics of immunity to astrovirus are unknown. Since reported outbreaks have involved only children and the elderly, young adults may have resistance to infection. Norwalk-like Viruses Endemic disease

The Norwalk virus is the representative agent of a heterogenous group of viruses, also called small round structured viruses (SRSVs) or the Norwalk-like family of agents. The antigenic interrelationships among the many members of this class are complex, and the agents are usually identified by the locale where an outbreak occurred (e.g., Hawaii, Snow Mountain, Montgomery County, Taunton, Amulree, Sapporo, and Otofuke). Although Norwalk-like viruses may play a role in endemic diarrhea, diagnostic technology is not yet sufficiently developed to assess this aspect of their disease burden. In the United States, illness is most commonly reported among persons of school age and older. Levels of antibody specific to the Norwalk agent are low during childhood but reach 50% by middle age (49). In developing countries, antibodies are acquired at an earlier age; peak incidence of illness may also occur among younger age groups than in developed nations (50). Clinical syndrome

The incubation period is 24-48 hours, and the mean duration of illness is 12-60 hours. Nausea is prominent, with vomiting, non-bloody diarrhea, and abdominal cramps occurring in most cases. These symptoms are experienced by all age groups, but diarrhea is relatively more prevalent among adults, whereas a higher proportion of children experience vomiting. From 25%-50% of affected persons also report headache, fever, chills, and myalgias. Adults have died during illness caused by Norwalk-like viruses, presumably from electrolyte imbalance. Late sequelae have not been reported, but the elderly often report persistence of constitutional symptoms for up to several weeks. Mode of transmission

Routes of transmission that have been documented include water, food (particularly shellfish and salads), aerosol, fomites, and person-to-person contact. Infectivity can last for as long as 2 days after resolution of symptoms (51). Presymptomatic shedding has been

suspected on epidemiologic grounds but not proven in volunteer studies. Outbreak characteristics

Numerous reports have described the course of outbreaks caused by Norwalk-like agents, usually involving adults and older children. The settings are diverse and include banquets, cruise ships, geriatric facilities, psychiatric wards, emergency rooms, cafeterias, recreational lakes, swimming pools, campgrounds, football teams, hotels, schools, dormitories, fast food restaurants, and others (52). Norwalk-like agents probably create a low background level of infection in a community until an infected individual contaminates a common source, and an explosive outbreak occurs. Although secondary cases can multiply the number of persons affected, outbreaks are generally limited to 1-2 weeks unless transmission is facilitated by a closed environment (e.g., a nursing home) or prolonged by renewal of the susceptible population (e.g., a new set of passengers on a cruise ship). Immunity

Studies of volunteers have documented the paradox that persons with the highest preexisting levels of Norwalk antibodies are at highest risk of developing symptomatic infection (49). Most persons' antibody levels against Norwalk virus rise after infection; these titers normally peak by the third week and persist until approximately the sixth week, after which they decline. Although preexisting antibody levels correlate with risk of symptomatic illness upon exposure to the virus, acutely elevated antibody levels appear to correlate with resistance to reinfection. The nature of resistance and susceptibility to the Norwalk-like agents is poorly understood. Other Viruses

Several other agents, listed below, have been implicated in viral gastroenteritis, but their public health importance is not yet clear. Pestivirus

A recent study showed that on an Arizona Indian reservation, 23% of specimens from children less than 2 years of age with gastroenteritis of unknown etiology were antigen-positive for pestivirus, compared with 3% of controls. Illness was relatively mild, and duration was 3 days (53). Antibody studies of serum samples from Arizona, Maryland, and Peru suggested that 30%-50% of children and adults had been infected, with peak exposure occurring at less than 2 years of age (54). Picobirnavirus

Reports from Brazil documented human cases of diarrhea caused by picobirnavirus, which had been thought to be a cause of diarrhea only in animals (55). The importance of this pathogen is unknown. Parvovirus

Parvovirus-like particles have been identified by electron microscopy in stool specimens of both well and ill persons in Britain (56). The relationship of these particles to disease is unclear, but they have been associated with shellfish-related outbreaks of gastroenteritis (57). Enteroviruses

Enteroviruses cause a wide spectrum of disease, in which gastroenteritis plays a minor role (58). Although the entry of polio, coxsackie, echo, or other enteroviruses through the gut may cause incidental mild diarrheal symptoms, the spread of the virus through the bloodstream to other organs (e.g., central nervous system, heart, pleura, pancreatic islets) produces major disease manifestations. Although reports have linked some enteroviruses to illnesses in which diarrhea was the sole symptom, an outbreak or case of gastroenteritis should not be attributed to an enterovirus merely because it was isolated in the stool of an affected person. Torovirus

Toroviruses are known causes of diarrhea among cattle, and identification in human specimens has been reported (59). Antibodies to the Breda strain of toroviruses were not present, however, in 100 human sera from Britain, and the torovirus' importance as a cause of human disease is unknown (60). Coronavirus

Coronaviruses are well-established causes of diarrhea in animals and respiratory disease in humans. These viruses have been identified in the stool of persons with gastroenteritis (usually children less than 2 years of age), but human controls have been found to shed them with higher frequency (61,62), raising doubt about their etiologic role in human diarrhea. Coronaviruses have been detected most frequently in the southwest; one group reported that more than two-thirds of diarrheal stools examined by electron microscopy over an 8-year period contained such viruses, although no comparison was made with specimens from well persons (63). Worldwide, coronaviruses have been detected at highest rates in situations of poor sanitation. METHODS OF VIRAL DETECTION Antigen Detection

Commercial antigen-detection kits for rotavirus are widely available, inexpensive, and permit rapid viral diagnosis. Only small amounts of stool are required for the tests, and samples may be frozen before testing. Kits vary widely in range of sensitivities (70%-100%) and specificities (50%-100%) (64,65). Newborns and breast-feeding children have particularly high false-positive rates. Such kits are most useful for childhood diarrhea during the normal rotavirus season; they have less diagnostic value in situations in which rotavirus is probably rare, as in community outbreaks involving adults or in outbreaks of pediatric diarrhea outside the rotavirus season. Confirmatory testing should be performed in any case in which rotavirus disease would be unusual (e.g., among children in the summer or among adults at any time) as well as periodically to validate the reliability of the assay employed.

A commercial kit for enteric adenoviruses is also available, but because adenoviral diarrhea affects mainly children less than 2 years of age and because outbreaks involving adults have never been reported, the diagnostic value outside the preschool-age group is also limited.

Antigen-detection systems have been used for research on calicivirus, Norwalk, Snow Mountain agent, and astrovirus. Rapid assays for

these and other agents are under development at the Viral Gastroenteritis Section, REB, DVRD, CID, CDC. Such techniques would allow the testing of large numbers of samples in a short period of time, an essential condition for determining the contribution of specific viruses to the incidence of diarrhea among populations and for identifying an etiologic agent during an outbreak. Antibody Detection

Persons infected with a viral agent of gastroenteritis will usually have a rise in antibodies to that virus.

For the Norwalk agent (the most commonly identified agent of outbreaks involving persons greater than 4 years of age), approximately half of adult Americans have preexisting IgG antibodies to the virus, so that a single specimen is insufficient to document recent infection. But if at least half of affected persons in an outbreak have a fourfold rise in specific antibody titers, the Norwalk agent can be designated as etiologic. Titers may begin to rise by the fifth day after onset of symptoms, peak at approximately the third week, and often begin to fall by the sixth week. Hence, the acute-phase serum should be drawn within the first week and the convalescent-phase serum during the third to sixth weeks. Diagnostic assays for IgM and IgA antibodies to Norwalk virus have been used on an experimental basis (66,67).

One disadvantage of serologic diagnosis is that patients are often reluctant to have serum drawn a month after a brief, self-limited illness. Furthermore, because this class of viruses cannot be cultivated, the supply of antigen for antibody testing is limited to a few research laboratories and cannot be offered for routine screening. In addition, antibodies can be detected to the Norwalk virus only, not the full spectrum of Norwalk-like agents that may cause disease. In a survey of 100 gastroenteritis outbreaks thought to be of viral origin submitted to CDC from 1985 to 1988, the Norwalk agent was identified by antibody rise (i.e., half or more of the persons showed a fourfold rise) in approximately 20% of outbreaks. Approximately 40% showed partial rises (less than half of the persons with a fourfold rise), suggesting that an antigenically related agent may have been involved. The remaining 40% showed no titer rises at all, indicating that an agent completely distinct from Norwalk virus caused the outbreak (68).

Adequate supplies of antigen are essential to any virus-testing system. Although rotavirus and astrovirus have been cultivated, the Norwalk family of viruses has proved resistant, and it may be several years before a panel of molecular diagnostic assays for known enteric viruses is available. Electron Microscopy

Under an electron microscope, a virus can be identified by its characteristic morphology in a stool specimen. The technique is highly specific but requires substantial resources. Since an electron microscope scans a field of approximately 1 millionth of a milliliter, there must be at least 1 million viruses/milliliter of stool for a detection to be made. Such levels of excretion are normally present only during the first 48 hours of viral diarrhea.

With a specialized technique called immune electron microscopy (IEM), the sensitivity of normal transmission electron microscopy can be improved 10-100 times. In one technique, the grid to be examined is coated with convalescent-phase serum before the stool specimen is applied; a high titer of virus-specific antibody tends to hold aggregates of homologous virus in the field, thereby enhancing diagnostic yield. Because reagents are scarce, this technique for diagnosing viral gastroenteritis is limited to a few centers in the United States. Other Techniques Culture

Rotavirus, enteric adenoviruses, and astrovirus can be cultured in research centers, but the techniques are not well suited for routine diagnosis. The other known major viral enteric pathogens cannot yet be cultivated. Enteroviruses can be cultivated but are not thought to be important causes of diarrhea. Electropherotyping

Rotavirus multiplies in the gut with such efficacy that, during infection, its genome dominates the ribonucleic acid (RNA) content of stool. When this RNA is extracted from stool and run on a gel, an electric field will cause the separate rotavirus genes to migrate in characteristic patterns. The presence of these patterns is diagnostic of rotaviral infection, and pattern variations provide insight into strain differences. Sensitivity is comparable with antigen detection (greater than 90% in the first few days of illness), and specificity is 100%; in addition, atypical rotaviruses (Groups B and C) can be detected, which is not possible with antigen-detection systems for rotavirus A. Electropherotyping has long been used as the principal diagnostic technique for rotavirus in many nations, but in the United States it is used mainly as a research tool. Hybridization probes

Dot-hybridization assays have been developed for rotavirus that are considerably more sensitive than conventional antigen-detection techniques and at least equally specific (69,70), but they are currently available in only a few research centers. Hybridization assays have also been developed for adenoviruses, but they are less sensitive than antigen-detection techniques (26). Polymerase chain reaction (PCR)

Enzymatic amplification of a viral gene to raise its concentration in a specimen to the level of detectability would represent an ideal diagnostic technique for viral gastroenteritis, because at present agents can be identified only when they are excreted at maximal levels (millions of viruses/milliliter of stool). The development of PCR reagents, however, requires cloning and sequencing of viral genes. Cloning and sequencing have been accomplished only with rotavirus, but PCR techniques are being actively developed for the other agents. ENDEMIC CONTROL

Although essential in outbreak management, improved environmental hygiene (i.e., food, water, and sanitation) may be ineffective in endemic control of some of the known viral agents of gastroenteritis (e.g., rotavirus), perhaps because person-to-person transmission is

the principal mechanism for the spread of infection. As a result, the population-based attack rate for these agents is thought to be the same (100%) in developed and developing countries, although disease caused by known agents tends to be acquired earlier in developing countries. The risk of death is highest in areas where medical care is least available and malnutrition is most prevalent.

Vaccines may prove to be the most effective method of endemic control. Because rotavirus is the leading cause of diarrheal mortality in the world and because natural infection appears to induce lifetime immunity, rotavirus has been the main focus of efforts at vaccine development: within 10 years of its discovery in 1973, trials were already under way. Although four major serotypes affect humans, three prototype vaccines have each been of a single serotype, and the results have been inconsistent (71-75). These inconsistencies have been ascribed by some investigators to differences between the vaccine serotype and the strain of rotavirus in the community. A large, multicenter, 2-year, efficacy trial of a vaccine that incorporates all four major serotypes that affect humans is now under way in the United States. The endemic disease incidence and the nature of immunity for the other viruses is less well defined, and vaccine development for them must await advances in knowledge of their epidemiology and molecular virology. ENVIRONMENTAL PROTECTION

Although person-to-person transmission is an important aspect of endemic disease, the initiating event for most outbreaks of viral gastroenteritis is contamination of a common source. In contrast to bacterial pathogens, enteric viruses cannot multiply outside their host; hence, the original inoculum into the common source determines infectivity. Food Shellfish

Shellfish that grow in fecally contaminated water concentrate enteric viruses in their tissues, and even harvests meeting bacteriologic standards of hygiene may contain viral agents. In addition, depuration (a technique in which shellfish are flushed with clean water treated with ultraviolet light) is less effective in viral than in bacterial decontamination (76). Finally, steaming for as long as 10 minutes may fail to inactivate all viral agents (77). Although boiling shellfish will inactivate viruses, such preparation is not popular with consumers.

The difficulties of assuring virus-free shellfish and the common preference for eating them raw have contributed to their prominent role in gastroenteritis outbreaks. Approximately 50% of Norwalk-confirmed foodborne outbreaks reported to CDC from 1976 to 1980 involved shellfish (52). A continent-wide epidemic of gastroenteritis in Australia in 1978 was attributed to oysters (78). Shellfish were implicated in 103 outbreaks of viral gastroenteritis in New York State in 1982, and similar outbreaks have occurred in the Northeast since then (79). Of 13 outbreaks of Norwalk virus documented in Britain during the period 1984-1985, seven were related to shellfish (57). Ill food handlers

When foods other than shellfish are implicated in viral gastroenteritis outbreaks, the contamination has usually taken place near the point of consumption. An ill food handler was identified in nine of the 15 documented Norwalk outbreaks reported to CDC from 1985 to 1988 for which adequate epidemiologic data were available (CDC, unpublished data). Foods that require handling and no subsequent cooking (e.g., salads) constitute the greatest risk. Among Norwalk-confirmed foodborne outbreaks from 1976 to 1980 that were not attributable to shellfish, salad was the most commonly implicated food (52). Other aspects of foodborne transmission

The long list of foods implicated in outbreaks of viral gastroenteritis reflects the variety of foods that are handled by food-service personnel and the low infectious dose (10-100 particles) of most viral agents of gastroenteritis, rather than peculiar viral tropisms. In contrast to the factors important in amplifying bacterial contamination, practices such as leaving foods unrefrigerated or warming them for prolonged periods are not direct risk factors for increased viral transmission because the viruses do not multiply outside the human host. Such practices, however, may be indicators of poor food hygiene in general.

The Norwalk agent can remain infective even if frozen for years or heated to 60 C for 30 minutes (80); however, cooking temperatures at boiling or above are probably adequate to inactivate Norwalk and most other enteric viral pathogens. Water

Outbreaks of viral gastroenteritis have been associated with various sources of contaminated water, including municipal water, well water, stream water, commercial ice, lake water, and pool water.

The most recent U. S. Environmental Protection Agency guidelines (June 29, 1989) for municipal water systems recommend residual chlorine concentrations of greater than or equal to 0.2 milligrams/liter (mg/L) (81), and in many localities peak levels of 5 mg/L are administered. Studies have documented that the Norwalk agent can remain highly infective despite 30-minute exposure to concentrations of chlorine as high as 6.25 mg/L, and levels of 10 mg/L appear necessary to inactivate it (82). This resistance may explain why the Norwalk agents are prominent in outbreaks of waterborne disease. Of 96 waterborne outbreaks with sufficient data reported to CDC from 1976 to 1979, 23% met epidemiologic criteria of a Norwalk virus outbreak (83), and subsequent surveillance data on waterborne outbreaks have been consistent with this finding. Of 38 serologically confirmed Norwalk virus outbreaks between 1976 and 1980, 13 were waterborne (52). Rotavirus, for which only one waterborne outbreak has been documented in the United States, is more sensitive to chlorine than the Norwalk agent and is inactivated by a 30-minute exposure to 3.75 mg/L (82). All viral agents of gastroenteritis are thought to be inactivated by boiling for 10 minutes. Surfaces

Because rotavirus can survive for several days on nonporous materials in conditions of low temperature and humidity, fomites may contribute to its nosocomial transmission (84). A recent study of a Norwalk viral outbreak on a cruise ship implicated toilets shared between staterooms as a risk factor for infection, suggesting that surfaces contaminated by Norwalk particles from spattered or

aerosolized material may play a role in transmission of Norwalk-like viruses (85). Data are lacking on the efficacy of disinfectants against Norwalk-like agents, but a number of germicidal chemicals have been shown in laboratory tests to be ineffective in reducing rotavirus activity (86-88). However, detergents do inactivate rotavirus (89) and should be used for laundering fecally contaminated linens and clothing. Thorough cleaning of environmental surfaces is required, as a minimum, to control spread of the viral agents of gastroenteritis. Hands

Hands that have been contaminated directly or from surfaces may be the most important means by which enteric viruses are transmitted. Because the active ingredients in some commercial handwashing preparations are ineffective against rotavirus (90), the use of special handwashing products is not indicated. Vigorous handwashing with soap, performed consistently at appropriate intervals, is necessary to control the spread of all enteric pathogens. Aerosols

Aerosolized or splattered Norwalk-like particles have been implicated in the transmission of gastroenteritis (91). Aerosolized rotavirus has caused diarrheal illness in mice (92), and airborne transmission of this agent among humans has been suspected. Studies are needed to address the efficacy of barrier precautions (e.g., face shields, respirators) in interrupting transmission of these agents. Zoonoses

Nearly all the agents of viral gastroenteritis in humans have related strains that can cause diarrhea in animal species. But these strains appear to be highly host-specific, and zoonotic transmission has not been documented as having an important role in human disease, either endemically or in outbreaks (93). TREATMENT

For most humans, viral gastroenteritis is a self-limited illness of a few days' duration, with virus replication restricted to the mucosa of the gut. The main risk is of dehydration and electolyte imbalance.

Children, in whom the risk of fluid loss is greatest, respond well to oral rehydration therapy (ORT). Hospitalization and treatment with intravenous fluids are required only for cases in which dehydration is severe, or in which the parent or caretaker cannot provide adequate oral rehydration. Analysis of geographic and demographic patterns of diarrheal mortality in the United States suggests that lack of access to medical care, rather than disease virulence, is a principal risk factor for death from gastroenteritis (2). Although infants with diarrhea may manifest subsequent mild lactose intolerance (often 10-14 days for rotavirus infection) (94), most infants completely recover. Breast milk may have a protective effect against bacterial or viral enteric infection, and most infants can be "fed through" an episode (95).

For adults, maintenance of good hydration is also important, particularly among the elderly and those receiving diuretic medication. In one study, bismuth subsalicylate reduced duration of Norwalk infection from 27 to 20 hours (96). SPECIAL HOSTS The Malnourished

The public health impact of diarrheal viruses is compounded in developing countries by the cycle of diarrhea and malnutrition: acute diarrhea converts a marginal nutritional status into undernourishment, thereby reducing resistance to infection, predisposing persons to chronic diarrhea, which leads to further undernourishment. Studies are needed to determine if interventions can interrupt this downward clinical spiral once it has started. Pregnant Women

Although dehydration and electrolyte imbalance from any cause can pose a risk to pregnancy, no evidence indicates that the viral agents of gastroenteritis constitute a particular threat. Other than in children with primary immunodeficiencies, viremic states from these agents are not known to occur in humans, and thus the risk of transplacental exposure, fetal demise, or malformation is probably low or nonexistent. Neonates

Data on neonatal infection and immunity are available principally for rotavirus, for which immunity appears to be transferred transplacentally from mother to fetus late in the third trimester, perhaps mainly in the final month of gestation. Consequently, for the term infant, rotaviral infection during the first month of life tends to be mild or asymptomatic, and only when maternal antibody levels wane between the third and sixth month of life does infection pose a significant risk of illness. Evidence from Australia suggests that neonatal infection with rotavirus may function as a vaccination--an asymptomatic infection producing subsequent immunity (17). The preterm infant, however, lacks adequate maternal antibody and appears to be at increased risk for early and symptomatic rotaviral infection. Data from Britain suggest that oral administration of immunoglobulin appears to protect against disease during outbreaks in nurseries and may shorten the duration of symptoms (97).

Outbreaks of necrotizing enterocolitis have occurred concurrently with outbreaks of rotavirus diarrhea (98,99). But annual patterns of mortality for necrotizing enterocolitis do not show the winter seasonality of rotavirus infections (100), and--if an association exists--other factors are probably important. The Elderly

Antibody levels to many viral pathogens, as well as total IgG levels, wane with age; therefore, the elderly are at risk for infections to which younger adults are resistant. Diuretic medications and debility can increase the risk of an adverse outcome in what otherwise might be a mild diarrheal episode. The role of rotavirus in deaths of the elderly from gastroenteritis is being investigated. Persons with Immunodeficiencies Acquired immunodeficiency syndrome (AIDS)

Chronic diarrhea is a common complication of AIDS, and lists of etiologic pathogens usually include Cryptosporidium, Isospora, atypical mycobacteria, Salmonella, and cytomegalovirus, among others. However, few studies have addressed the role of the viral agents of gastroenteritis among persons with AIDS. A recent antigen-detection study failed to detect rotavirus in the stool of 20 U.S. AIDS patients with diarrhea (101). In contrast, an Australian study that used electron microscopy (the diagnositc reference method) found rotavirus in 37% of specimens and adenovirus in 24% of specimens from 68 HIV-positive homosexual men with diarrhea (102). These viruses were detected more frequently than other microbial agents, were associated with higher degrees of immunocompromise, and were found less commonly in specimens from homosexual men who were HIV-negative or were HIV-positive but did not have diarrhea. Norwalk virus was detected in one instance, and no caliciviruses or astroviruses were identified. Immunity to most endemic viral agents of gastroenteritis appears to be acquired in childhood and may persist during early stages of adult AIDS. Children with AIDS may never acquire this immunity and thus may be at increased risk for persistent infection. Studies are needed to determine the role of viruses in diarrhea associated with pediatric AIDS. Other immunodeficiencies

Case reports have documented chronic diarrheal excretion of rotavirus, adenovirus, calicivirus, astrovirus, and other viruses among children with other immunodeficiencies, most notably severe combined immunodeficiency syndrome (SCIDS) (103,104). Rotaviral and adenoviral infections have been documented in one study involving recipients of bone marrow transplants, and such infections are associated with markedly increased mortality (105). Unusual forms of rotaviral infection can occur in persons with immunodeficiencies: antigen has been detected in serum, suggesting that viremia may occur (106), and unique genomic rearrangements have been noted among patients with T-cell deficiencies (107). The immunodeficient host and viral investigation

The effect of viral agents of gastroenteritis on persons with immunodeficiencies is of particular interest. Although immunization appears to offer the principal hope of endemic control, the nature of immunity to the viral agents of diarrhea is poorly understood, and lack of information in this area has hampered vaccine development. Interventions, such as immunoglobulin, that might prove successful in halting the chronic infection of immunocompromise might also prove useful in other situations, such as the chronic diarrhea of malnourishment. SPECIFIC SITUATIONS Nursing Homes and Residential Institutions

Protracted outbreaks of viral gastroenteritis have occurred in nursing homes and institutional residences. Risk factors in such settings include the enclosed living quarters in which most residents spend their time and the decreased personal hygiene among some residents because of incontinence, immobility, or reduced alertness. Although Norwalk-like agents are most commonly involved in nursing home outbreaks, viruses usually associated with childhood diarrhea, such ascalicivirus and astrovirus, have also been implicated etiologically in some outbreaks, suggesting that the waning immunity of the elderly may also be a predisposing factor. Cruise Ships and Camps

The close living quarters of ships and camp dormitories amplify opportunities for person-to-person transmission of viral agents. Gastroenteritis outbreaks would normally end after all susceptible persons have been infected; however, in such settings, new and uninfected populations usually arrive every 1 or 2 weeks, thereby renewing the epidemic. Norwalk viral outbreaks extending over five successive cruises have been documented (108). Pediatric Wards and Day-Care Facilities

Continuous close contact among unrelated children, some of whom may be ill, can accelerate the progression of endemic diarrhea through a small population into an outbreak. Nosocomial and day-care transmission of rotavirus during its peak season is particularly efficient and difficult to control. Calicivirus, adenovirus, astrovirus, and Norwalk-like particles are found more frequently in stool specimens from children in these settings than from children in other settings. CLINICAL CHARACTERISTICS OF VIRAL GASTROENTERITIS OUTBREAKS

In outbreaks of gastroenteritis, investigators often face the problem of having to take action before an etiologic agent can be identified. This problem particularly applies to outbreaks caused by viruses, since diagnosis can be delayed for months or longer. Distinguishing viral from bacterial or protozoal etiologies is sometimes difficult because of overlap in clinical syndromes. A study of 74 outbreaks of probable viral gastroenteritis that occurred from 1976 to 1980 showed that affected persons had the following symptoms: nausea, 79%; abdominal cramps, 71%; vomiting, 69%; diarrhea (never bloody), 66%; headache, 50%; fever, 37%; chills, 32%; myalgias, 26%; and sore throat, 18%. In greater than 90% of the 38 outbreaks in this survey documented to have been caused by the Norwalk virus, the average incubation period was 24-48 hours and the average duration of illness was 12-60 hours. Vomiting occurred among more than half of the persons in almost 90% of the outbreaks (52). Review of Norwalk-documented outbreaks reported to CDC from 1985 to 1988 confirms these patterns (CDC, unpublished data).

Outbreaks of viral gastroenteritis reported to CDC are usually community based, involving adults or older children; thus, the characteristics of these outbreaks are those of the Norwalk-like viruses that affect these age groups. Outbreaks involving preschool children are more likely to be caused by the agents of endemic childhood diarrhea (e.g., rotavirus, adenovirus, calicivirus, or astrovirus) and are often in institutional settings, such as pediatric wards or day-care centers. Although clinical syndromes differ somewhat among viral pathogens, common features of viral outbreaks involving this younger age group include a high rate of vomiting, an absence of blood in the stool, and a duration of illness that is usually less than or equal to 1 week. OUTBREAK CONTROL MEASURES

Most outbreaks of viral gastroenteritis are self-limited; however, certain factors create risks of intense or prolonged transmission that may require aggressive intervention. These risk factors include a closed environment (e.g., nursing home), a constantly renewing population of susceptible persons (e.g., children at camp), or persons at special risk (e.g., the elderly). Whatever the initial source of the outbreak, subsequent viral transmission is often person-to-person, with both direct fecal-oral and airborne transport probably involved.

Although interruption of this transmission may be difficult, the following measures may be helpful in controlling the spread of infection. Identify and Eliminate a Common Source

For Norwalk virus outbreaks, an ill food handler is a likely source, although water, ice, and shellfish are other common sources. When a water supply is thought to be contaminated with Norwalk virus, shock chlorine concentrations (greater than or equal to 10 mg/L for 30 minutes or longer) may be helpful. Prevent Employee Transmission of Illness

In many settings, employees (e.g., health-care providers, staff of day-care centers) are at highest risk for transmitting disease because of their many contacts with ill persons. Any staff member with symptoms that suggest infection should be excluded from contact with potentially susceptible persons for at least 2 days after resolution of illness. This exclusion is particularly important for food handlers, who also should not be involved in preparing food for the same period. Prevent Employee Acquisition of Illness

Personnel coming into direct contact with ill persons should wear disposable plastic gloves. When contamination of clothing with fecal material is possible, personnel should also wear gowns. Hands, which are the most likely means by which viral spread occurs, should be washed after each contact. The recommended procedure is to rub all surfaces of lathered hands together vigorously for at least 10 seconds, with plain soap or an antimicrobial-containing product, and then thoroughly rinse the hands under a stream of water. Since spattering or aerosols of infectious material may be involved in disease transmission, wearing of masks should be considered, particularly by persons who clean areas grossly contaminated by feces or vomitus. Use Safeguards with Laundry

Soiled linens and clothes should be handled as little as possible and with minimum agitation to prevent microbial contamination of the air and of persons handling the linen. Laundry should be transported in an enclosed and sanitary manner (e.g., in a plastic bag if the laundry is wet or moist), promptly machine washed with a detergent in water at the maximum cycle length, and then machine dried (109). Clean Soiled Surfaces

Because environmental surfaces in certain settings have been implicated in the transmission of enteric viruses, bathrooms and rooms occupied by ill persons should be kept visibly clean on a routine basis. Surfaces that have been soiled, especially by feces or vomitus, should first be cleaned of visible material and then disinfected with an appropriate commercial germicidal product according to the manufacturer's instructions. Feces and vomitus collected during the cleaning procedure should be promptly disposed of in a manner that prevents transfer of this material to other surfaces or persons. Persons performing these tasks should wear appropriate protective barriers (e.g., utility gloves--and if splashing is anticipated, a mask or face shield and garments such as a uniform, jumpsuit, or gown to protect street clothing). Minimize Contact Between Well and Ill Persons

When possible, ill persons should be separated from well persons until at least 2 days after resolution of symptoms. If nosocomial rotavirus is involved, this period should be longer--at least until the ill person's stool is negative by antigen detection, which may be greater than or equal to 1 week. In certain settings (e.g., camp, cruise ship, or nursing home), the clinic may function as a focus of transmission; persons with complaints of gastroenteritis should be seen by medical care personnel in the patient's living quarters, or at least in a separate area of the clinic. Stop Renewal of Susceptible Population

In situations in which the epidemic is extended by periodic renewal of the susceptible population (e.g., camps and cruise ships), consideration may have to be given to interrupting this process until the outbreak has ended completely. SPECIMEN COLLECTION DURING OUTBREAKS

Electron microscopy is necessary for complete viral diagnosis of an outbreak of gastroenteritis. Most of the electron microscopes in the United States are devoted to research or fee-related clinical diagnostic activity. Very few state laboratories have electron microscopes that can be used in the evaluation of outbreaks. Consequently, many state and local health departments submit outbreak specimens to CDC. Because of advances in the field of viral diagnostics, recommendations for specimen collection have changed substantially, and many investigators in the field may not be aware of these changes.

The following guidelines are designed to help outbreak investigators make the best use of CDC's viral laboratory facilities. Proper specimen collection is also critical for the diagnosis of bacterial and parasitic causes of gastroenteritis (see Appendix). Guidelines for Collecting Specimens for Viral Diagnosis Stool

- Collection in the first 48 hours. Presently, viral diagnosis of a stool sample can be made only when the level of excretion is approximately 1 million particles/ml. For many viruses, this level of excretion is present only during the first 2 days of illness, and occasionally during the third. If specimens are not collected during the first 2-3 days of illness, an agent is unlikely to be detected. Thus, appropriate specimens should be collected as soon as an outbreak occurs. Specimen collection should not await the results of epidemiologic and other investigations, since delay will almost certainly preclude a viral diagnosis. If information gathered subsequently indicates that a viral etiology is unlikely, the specimens can be discarded before the cost of testing is incurred.

- Ten diarrheal bulk specimens. Bulk samples (enough to fill a large stool cup) are preferred, and only those specimens loose enough to assume the shape of their containers are likely to yield positive results. Serial specimens from persons with acute,

frequent, high-volume diarrhea are particularly useful. The smaller the specimen and the more formed the stool, the lower the diagnostic yield. Rectal swabs are of little or no value. Specimens from at least 10 ill persons should be collected to maximize the chance that a diagnosis can be made. (The diagnostic yield is low when specimens from less than 10 persons are submitted.)

- Storage at +4 C. Because freezing may destroy the characteristic viral morphology that permits a diagnosis by electron microscopy, specimens should be kept at +4 C. Paired Serum Specimens (essential for diagnosis)

- Timing Acute: during the first week of symptoms

Convalescent: third to sixth week

- Number 10 pairs from ill persons (the same persons submitting stool specimens)10 pairs from well persons

- Quantity Adults: 10 ml

Children: 3 ml

- Storage Tubes containing no anticoagulant (tubes with red tops) should be used for collection. Sera should be spun off and frozen. If a centrifuge is not available, a clot should be allowed to form, and the serum should be decanted and frozen. If this step cannot be taken, the whole blood should be refrigerated, not frozen. Other Specimens

Viruses causing gastroenteritis cannot normally be detected in vomitus, water, food, or environmental samples. Although British researchers report electron microscope detection of virus in shellfish, no successful effort has yet been reported in the United States. Consultation

At any time during the course of an outbreak, the Viral Gastroenteritis Section, REB, DVRD, CID (telephone 404-639-3577), is available for advice and assistance.

Since the viruses involved in most outbreaks of gastroenteritis reported to CDC cannot be cultivated, the antigen and antibody reagents used in diagnosis are not easily renewable. Thus, CDC cannot routinely screen persons and must limit use of these reagents. The following information would help determine the best use of laboratory resources:

- Setting of the outbreak (e.g., nursing home, restaurant)

- Date of onset of outbreak

- Number of persons exposed and number ill

- Population characteristics (e.g., elderly, children)

- Incubation period

- Duration of illness

- Symptoms

    - percent vomiting

    - percent with diarrhea

    - percent with bloody diarrhea

    - other (e.g., fever, myalgia, malaise)

- Adverse outcomes (e.g., hospitalizations, deaths)

- Suspected common source (e.g., shellfish, other food, food handler, water, other)

- Ongoing problems Sending Specimens for Viral Diagnosis

If, after consultation, viral diagnostic services are considered to be useful, specimens may be shipped to the Viral Gastroenteritis Section, REB, DVRD, CID, following these guidelines:

- Each specimen should be labeled (waterproof) with a unique identifier firmly attached and should be accompanied by CDC Specimen Information Form 50.34.
- Stool specimens should be shipped as soon as they can be batched. Individual containers should be verified as leakproof and then enclosed in a plastic bag. The entire collection should be bagged in plastic and placed in a padded, insulated box with refrigerant packs.
- Frozen acute- and convalescent-phase serum samples should be batched and sent in a single shipment. Waterproof, padded, insulated boxes should be used, with dry ice added to maintain freezing. Whole blood samples should not be frozen, and refrigerant packs should be used instead of dry ice.
- Final notification should be made by telephone (404-639-3577) just before shipping.
- All shipments should be sent by overnight mail, due to arrive on a weekday, addressed to the Viral Gastroenteritis Section, REB, DVRD, CID, Mailstop G04, Centers for Disease Control, Atlanta, GA 30333. SURVEILLANCE

Few states maintain surveillance for the viral agents of gastroenteritis. In 1989, CDC began surveillance of rotavirus in anticipation of vaccine development and use. Postcard reports of monthly detections are submitted by almost 100 centers in the United States. Regular summaries of detection patterns will soon be issued to contributors, state public health departments, and other interested persons. Plans are under way for surveillance of the other agents of gastroenteritis, in collaboration with laboratories that use electron microscopy. States and centers interested in participating in these surveillance activities are invited to contact the Viral Gastroenteritis Section, REB, DVRD, CID, CDC.

References

1. Ho M, Glass RI, Pinsky PF, Anderson L. Rotavirus as a cause of

diarrheal morbidity and mortality in the United States. J Infect Dis 1988;158:1112-6.

2. Ho M, Glass RI, Pinsky PF, et al. Diarrheal deaths in American children: are they preventable? JAMA 1988;260:3281-5.

3. National Center for Health Statistics. Current estimates from the health interview survey. Series 10, no. 85, 1972. DHEW publication (HRA) 74-1512. Washington, DC: Government Printing Office, 1973.

4. Walsh JA, Warren KS. Selective primary health care: An interim strategy for disease control in developing countries. N Engl J Med 1979;302:967-74.

5. Kotloff KL, Wasserman SS, Steciak JY, et al. Acute diarrhea in Baltimore children attending an outpatient clinic. Pediatr Infect Dis J 1988;7:753-9.

6. Institute of Medicine. *Prospects for immunizing against rotavirus*. Washington, DC: National Academy Press, 1985.

7. Wenman WM, Hinde D, Feltham S, Gurwith M. Rotavirus infection in adults: results of a prospective family study. N Engl J Med 1979;301:303-6.

8. Ward RL, Bernstein DI, Young EC, Sherwood JR, Knowlton DR, Schiff GM. Human rotavirus studies in volunteers: determination of infectious dose and serological response to infection. J Infect Dis 1986;154:871-80.

9. Pickering LK, Bartlett III AV, Reves RR, Morrow A. Asymptomatic excretion of rotavirus before and after rotavirus diarhhea in children in day care centers. J Pediatr 1988;112:361-5.

10. Champsaur H, Questiaux E, Prevot J, et al. Rotavirus carriage, asymptomatic infection, and disease in the first two years of life. Part I: Virus shedding. J Infect Dis 1984;149:667-74.

11. Champsaur H, Henry-Amar M, Goldszimdt D, et al. Rotavirus carriage, asymptomatic infection, and disease in the first two years of life. Part II: Serological response. J Infect Dis 1984;149:675-82.

12. Echeverria P, Blacklow NR, Cukor GG, Vivulbandhitkit S, Changchawalit S, Boonthai P. Rotavirus as a cause of severe gastroenteritis in adults. J Clin Microbiol 1983;18:663-7.

13. Eiden JJ, Verleur DG, Vonderfcht SL, Yolken RH. Duration and pattern of asymptomatic rotavirus shedding by hospitalized children. Pediatr Infect Dis J 1988;7:564-9.

14. Bartlett III AV, Reves RR, Pickering LK. Rotavirus in infant-toddler day care centers: epidemiology relevant to disease control strategies. J Pediatr 1988;113:435-41.

15. Pickering LK, Bartlett III AV, Woodward WE. Acute infectious diarrhea among children in day care: epidemiology and control. Rev Infect Dis 1986;8:539-47.

16. Hopkins RS, Gaspard GB, Williams FPJ, Karlin RJ, Cukor G, Blacklow NR. A community waterborne gastroenteritis outbreak: evidence for rotavirus as the agent. Am J Public Health 1984;74:263-5.

17. Bishop RF, Barnes GL, Cipriani E, Lund JS. Clinical immunity after rotavirus infection: a prospective longitudinal study in young children. N Engl J Med 1983;309:72-6.

18. Hung T, Chen G, Wang C, et al. Waterborne outbreak of rotavirus diarrhea in adults in China caused by a novel rotavirus. Lancet 1984;i:1139-42.

19. Hung T, Chen G, Wang C, et al. Seroepidemiology and molecular epidemiology of the Chinese rotavirus. In: Bock G, Whelan J, eds. Novel diarrhoea viruses. (Ciba Foundation Symposium; 128). Chichester, UK: John Wiley & Sons, Ltd, 1987:49-62.

20. Bridger JC. Non-group-A rotaviruses. In: Viruses and the gut: proceedings of the Ninth BSG-SK&F International Workshop. Welwyn Garden City, UK: Smith Kline & French Laboratories, Ltd, 1988:79-81.

21. Bridger JC, Pedley S, McCrae MA. Group C rotaviruses in humans. J Clin Microbiol 1986;23:760-3.

22. Penaranda ME, Cubitt WD, Sinarachatanant P, et al. Group C rotavirus infections in patients with diarrhea in Thailand, Nepal, and England. J Infect Dis 1989;160:392-7.

23. Uhnoo I, Wadell G, Svenson L, Johansson ME. Importance of enteric adenoviruses 40 and 41 in acute gastroenteritis in infants and young children. J Clin Microbiol 1984;20:365-72.

24. Moffet HL, Schulenberger HK, Burkholder ER. Epidemiology and etiology of severe infantile diarrhea. J Pediatr 1968;72:1-14.

25. Kotloff KL, Losonsky GA, Morris JG, Wasserman SS, Singh-Naz N, Levine MM. Enteric adenovirus infection and childhood diarrhea: an epidemiologic study in three clinical settings. Pediatrics 1989;84:219-25.

26. Hammond GW, Hannan C, Yeh T, Fischer K, Mauthe G, Straus SE. DNA hybridization for diagnosis of enteric adenovirus infection from directly spotted human fecal specimens. J Clin Microbiol 1987;25:1881-5.

27. Wood DJ, Longhurst D, Killough RI, David TJ. One-year prospective cross-sectional study to assess the importance of group F adenovirus infection in children under 2 years admitted to hospital. J Med Virol 1988;26:429-35.

28. Chiba S, Nakata S, Nakamura I, et al. Outbreak of infantile gastroenteritis due to type 40 adenovirus. Lancet 1983;2:954-7.

29. Richmond SJ, Caul EO, Dunn SM, Ashley CR, Clarke SK, Seymour NR. An outbreak of gastroenteritis in young children caused by adenoviruses. Lancet 1979;i:1178-81.

30. Ellis ME, Watson B, Mandal BK, et al. Micro-organisms in gastroenteritis. Arch Dis Child 1984;59:848-55.

31. Matson DO, Estes MK, Glass RI, et al. Human calicivirus-associated diarrhea in children attending day care centers. J Infect Dis 1989;159:71-7.

32. Sakuma Y, Chiba S, Kogasaka R, et al. Prevalence of antibody to human calicivirus in general population of northern Japan. J Med Virol 1981;7:221-5.

33. Cubitt WD, McSwiggan DA. Seroepidemiological survey of the prevalence of antibodies to a strain of human calicivirus. J Med Virol 1987;21:361-8.

34. Cubitt WD. Caliciviruses. In: Farthing MJ, ed. Viruses and the gut: proceedings of the ninth BSG-SK&F international workshop. Welwyn Garden City, UK: Smith Kline & French Laboratories Ltd, 1988:82-4.

35. Chiba S, Sakuma Y, Kogasaka R, et al. An outbreak of gastroenteritis associated with calicivirus in an infant home. J Med Virol 1979;4:249-54.

36. Oishi I, Maeda A, Yamazaki K, Minekawa Y, Nishimura H, Kitaura T. Calicivirus detected in outbreaks of acute gastroenteritis in school children. Biken J 1980;23:163-8.

37. Cubitt WD, McSwiggan DA, Arstall S. An outbreak of calicivirus infection in a mother and baby unit. J Clin Pathol 1980;33:1095-8.

38. Cubitt WD, McSwiggan DA, Moore W. Winter vomiting disease caused by calicivirus. J Clin Pathol 1979;32:786-93.

39. Gary JJ, Wreghitt TG, Cubitt WD, Elliot PR. An outbreak of gastroenteritis in a home for the elderly associated with astrovirus type 1 and human calicivirus. J Med Virol 1987;23:377-81.

40. Humphrey TJ, Cruickshank JG, Cubitt WD. An outbreak of calicivirus associated gastroenteritis in an elderly persons home: a possible zoonosis? J Hyg 1984;93:293-9.

41. Cubitt WD, Moscovici OP PJ, Lebon PS AA. A new serotype of calicivirus associated with an outbreak of gastroenteritis in a residential home for the elderly. J Clin Pathol 1981;34:924-6.

42. Kurtz J, Lee T. Astrovirus gastroenteritis: age distribution of antibody. Med Microbiol Immunol 1978;166:227-30.

43. Grohmann G. Viral diarrhoea in Australia. In: Tzipori S, ed. Infectious diarrhoea in the young. Amsterdam: Excerpta Medica Elsevier, 1985:25-8.

44. Kurtz JB, Lee TW. Astrovirus: human and animal. In: Bock G, Whelan J, eds. Novel diarrhoea viruses. (Ciba Foundation Symposium; 128). Chichester, UK: John Wiley & Sons Ltd, 1987:92-107.

45. Ashley CR, Caul EO, Paver WK. Astrovirus-associated gastroenteritis in children. J Clin Pathol 1978;31:939-43.

46. Konno T, Suzuki H, Ishida N, Chiba R, Mochizuki K, Tsunoda A. Astrovirus-associated epidemic gastroenteritis in Japan. J Med Virol 1982;9:11-7.

47. Kurtz JB, Lee TW, Pickering D. Astrovirus associated gastroenteritis in a children's ward. J Clin Pathol 1977;30:948-52.

48. Oshiro LS, Haley CE, Roberto RR, et al. A 27-nm virus isolated during an outbreak of acute infectious non-bacterial gastroenteritis in a convalescent hospital: a possible new serotype. J Infect Dis 1981;143:791-5.

49. Blacklow NR, Cukor G, Bedigian MK, et al. Immune response and prevalence of antibody to Norwalk enteritis virus as determined by radioimmunoassay. J Clin Microbiol 1979;10:903-9.

50. Greenberg HB, Valdesuso JR, Kapikian AZ, et al. Prevalence of antibody to the Norwalk virus in various countries. Infect Immun 1979;26:270-3.

51. White KE, Osterholm MT, Mariotti JA, et al. A foodborne outbreak of Norwalk virus gastroenteritis: evidence for post-recovery transmission. Am J Epidemiol 1986;124:120-6.

52. Kaplan JE, Gary GW, Baron RC, et al. Epidemiology of Norwalk gastroenteritis and the role of Norwalk virus in outbreaks of acute nonbacterial gastroenteritis. Ann Intern Med 1982;96:756-61.

53. Yolken R, Leister F, Almeido-Hill J, Dubovi E, Reid R, Santosham M. Infantile gastroenteritis associated with excretion of pestivirus antigens. Lancet 1989;i:517-9.

54. Yolken R, Santosham M, Reid R, Dubovi E. Pestiviruses: major etiological agents of gastroenteritis in human infants and children? (Abstract). Clin Res 1988;36:80A.

55. Pereira HG, Fialho AM, Flewett TH, Teixeira JMS, Andrade ZP. Novel viruses in human faeces. Lancet 1988;ii:103-4.

56. Flewett TH, Davies H, Bryden AS, Robertson MJ. Diagnostic electron microscopy of faeces. II. Acute gastroenteritis associated with reovirus-like particles. J Clin Pathol 1974;27:608-14. 57. Appleton H. Small round viruses: classification and role in food-borne infections. In: Bock G, Whelan J, eds. Novel diarrhoea viruses. (Ciba Foundation Symposium; 128). Chichester, UK: John Wiley & Sons Ltd, 1987:108-25.

58. Birch CJ, Lewis FA, Kennett ML, Homola M, Pritchard H, Gust ID. A study of the prevalence of rotavirus infection in children with gastroenteritis admitted to an infectious disease hospital. J Med Virol 1977;1:69-77.

59. Beards GM, Green J, Hall C, Flewett TH, Lamouliatte F, Du Pasquier P. An enveloped virus in stools of children and adults that resembles the Breda virus of calves. Lancet 1984;i:1050-2.

60. Brown DWG, Beards GM, Flewett TH. Detection of Breda virus antigen and antibody in humans and animals by enzyme immunoassay. J Clin Microbiol 1987;25:637-40.

61. Kidd AH, Esrey SA, Ujfalusi MJ. Shedding of coronavirus-like particles by children in Lesotho. J Med Virol 1989;27:164-9.

62. Mathan M, Mathan VI, Swaminathan SP, Yesudoss S, Baker SJ. Pleomorphic virus-like particles in human faeces. Lancet 1975;i:1068-9.

63. Payne CM, Ray CG, Borduin V, Minnich LL, Lebowitz MD. An eight-year study of the viral agents of acute gastroenteritis in humans: ultrastructural observations and seasonal distrubution with a major emphasis on coronavirus-like particles. Diagn Microbiol Infect Dis 1986;5:39-54.

64. Dennehy PH, Gauntlett DR, Tente WE. Comparison of nine commercial immunoassays for the detection of rotavirus in fecal samples. J Clin Microbiol 1988;26:1630-4.

65. Thomas EE, Puterman ML, Kawano E, Curran M. Evaluation of seven immunoassays for detection of rotavirus in pediatric stool samples. J Clin Microbiol 1988;26:1189-93.

66. Erdman DD, Gary GW, Anderson LJ. Development and evaluation of an IgM capture enzyme immunoassay for diagnosis of recent Norwalk virus infection. J Virol Meth 1989;24:57-66.

67. Erdman DD, Gary GW, Anderson LJ. Serum Immunoglobulin: a response to Norwalk virus infection. J Clin Microbiol 1989;27:1417-8.

68. Glass RI, Monroe SS, Stine S, et al. Small round structured viruses: the Norwalk family of agents. In: Farthing MJG, ed. Viruses and the gut: Proceedings of the Ninth BSG-SK&F International Workshop. Welwyn Garden City, UK: Smith Kline & French Laboratories, 1988:87-90. References 69 through 109 may be obtained from the Viral Gastroenteritis Section, REB, DVRD, CID, Mailstop G04, Centers for Disease Control, Atlanta, GA 30333. APPENDIX

The following procedures are recommended for use by state and local health departments for investigation of outbreaks. Instructions are summarized for each category of pathogens.

State public health laboratories can provide diagnostic services for most bacterial and parasitic pathogens. No specimens should be sent to CDC for diagnostic testing unless the specific program concerned has been consulted.

Page converted: 08/05/98

This page last reviewed 5/2/01

Centers for Disease Control and Prevention
Morbidity and Mortality Weekly Report

CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

MMWR™

MORBIDITY AND MORTALITY WEEKLY REPORT

Published November 20, 1998, for
1997 / Vol. 46 / No. 54

Summary of Notifiable Diseases, United States

1997

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control
and Prevention (CDC)
Atlanta, Georgia 30333

Table of Contents

Foreword

MMWR Summary of Notifiable Diseases, United States, 1997

This publication contains summary tables of the official statistics for the reported occurrence of nationally notifiable diseases in the United States for 1997. These statistics are collected and compiled from reports to the National Notifiable Diseases Surveillance System (NNDSS), which is operated by CDC in collaboration with the Council of State and Territorial Epidemiologists (CSTE). Because the dates of onset or diagnosis for notifiable diseases are not always reported, these surveillance data are presented by the week they were reported to CDC by public health officials in state and territorial health departments. These data are finalized and published in the *MMWR Summary of Notifiable Diseases, United States* for use by state and local health departments; schools of medicine and public health; communications media; local, state, and federal agencies; and other agencies or persons interested in following the trends of reportable diseases in the United States. The annual publication of the *Summary* also documents which diseases are considered national priorities for notification and the annual number of cases of such diseases.

The Highlights section presents information on selected nationally notifiable and non-notifiable diseases to provide a context in which to interpret surveillance and disease-trend data and to provide further information on the epidemiology and prevention of selected diseases.

Part 1 contains information regarding morbidity for each of the diseases considered nationally notifiable during 1997. The tables provide the number of cases of notifiable diseases reported to CDC for 1997, as well as the distribution of cases by month and geographic location and by patient's age, sex, race, and Hispanic ethnicity. The data are final totals as of July 25, 1998, unless otherwise noted. Because no cases of anthrax or yellow fever were reported in the United States during 1997, these nationally notifiable diseases do not appear in the tables in Part 1. Nationally notifiable diseases that are reportable in fewer than 40 states also do not appear in these tables. In all tables, leprosy is listed as Hansen disease, and tickborne typhus fever is listed as Rocky Mountain spotted fever (RMSF).

Part 2 contains graphs and maps. These graphs and maps depict summary data for many of the notifiable diseases described in tabular form in Part 1.

Part 3 contains tables that list the number of cases of notifiable diseases reported to CDC since 1966. It also includes a table enumerating deaths associated with specified notifiable diseases reported to the National Center for Health Statistics, CDC during 1987–1996.

Background

As of January 1, 1997, 52 infectious diseases were designated as notifiable at the national level. A notifiable disease is one for which regular, frequent, and timely information regarding individual cases is considered necessary for the prevention and control of the disease. This section briefly summarizes the history of the reporting of nationally notifiable diseases in the United States.

In 1878, Congress authorized the U.S. Marine Hospital Service (i.e., the forerunner of the Public Health Service [PHS]) to collect morbidity reports regarding cholera, smallpox, plague, and yellow fever from U.S. consuls overseas. The intention was to use this information to institute quarantine measures to prevent the introduction and spread of these diseases into the United States. In 1879, a specific Congressional appropriation was made for the collection and publication of reports of these notifiable diseases. Congress expanded the authority for weekly reporting and publication of these reports in 1893 to include data from states and municipal authorities. To increase the uniformity of the data, Congress enacted a law in 1902 directing the Surgeon General to provide forms for the collection and compilation of data and for the publication of reports at the national level. In 1912, state and territorial health authorities — in conjunction with PHS — recommended immediate telegraphic reporting of five infectious diseases and the monthly reporting, by letter, of 10 additional diseases. The first annual summary of *The Notifiable Diseases* in 1912 included reports of 10 diseases from 19 states, the District of Columbia, and Hawaii. By 1928, all states, the District of Columbia, Hawaii, and Puerto Rico were participating in national reporting of 29 specified diseases. At their annual meeting in 1950, state and territorial health officers authorized the Conference of State and Territorial Epidemiologists (CSTE), whose purpose was to determine which diseases should be reported to PHS. In 1961, CDC assumed responsibility for the collection and publication of data concerning nationally notifiable diseases.

The list of nationally notifiable diseases is revised periodically. For example, a disease might be added to the list as a new pathogen emerges, or a disease might be deleted as its incidence declines. Public health officials at state health departments and CDC continue to collaborate in determining which diseases should be nationally notifiable. CSTE, with input from CDC, makes recommendations annually for additions and deletions. However, reporting of nationally notifiable diseases to CDC by the states is voluntary. Reporting currently is mandated (i.e., by legislation or regulation) only at the state and local level. Thus, the list of diseases considered notifiable varies slightly by state. All states generally report the internationally quarantinable diseases (i.e., cholera, plague, and yellow fever) in compliance with the World Health Organization's International Health Regulations.

The list of 52 infectious diseases designated as notifiable at the national level during 1997 is as follows:

The 52 Infectious Diseases Designated as Notifiable at the National Level During 1997

Acquired immunodeficiency syndrome	*Haemophilus influenzae* (Invasive Disease)	Rabies, animal
Anthrax	Hansen disease (leprosy)	Rabies, human
Botulism*	Hantavirus pulmonary syndrome	Rocky Mountain spotted fever
Brucellosis	Hemolytic uremic syndrome, post-diarrheal	Rubella
Chancroid*	Hepatitis A	Salmonellosis*
Chlamydia trachomatis, genital infection	Hepatitis B	Shigellosis*
Cholera	Hepatitis, C/non-A, non-B	Streptococcal disease, invasive, group A
Coccidioidomycosis*	HIV infection, pediatric	*Streptococcus pneumoniae*, drug-resistant*
Congenital rubella syndrome	Legionellosis	Streptococcal toxic-shock syndrome
Congenital syphilis	Lyme disease	Syphilis
Cryptosporidiosis	Malaria	Tetanus
Diphtheria	Measles (Rubeola)	Toxic-shock syndrome
Encephalitis, California	Meningococcal disease	Trichinosis
Encephalitis, eastern equine	Mumps	Tuberculosis
Encephalitis, St. Louis	Pertussis	Typhoid fever
Encephalitis, western equine	Plague	Yellow fever
Escherichia coli O157:H7	Poliomyelitis, paralytic	
Gonorrhea	Psittacosis	

NOTE: Although varicella is not a nationally notifiable disease, the Council of State and Territorial Epidemiologists recommends reporting of cases of this disease to CDC.
*Not currently published in the *MMWR* weekly tables.

Highlights for 1997

The Highlights section presents information on the public health importance of selected nationally notifiable and non-notifiable diseases, including a) domestic and international disease outbreaks; b) active surveillance findings; c) changes in data reporting practices; d) the impact of prevention programs; e) the emergence of antimicrobial resistance; and f) changes in immunization policies. This information is intended to provide a context in which to interpret surveillance and disease-trend data and to provide further information on the epidemiology and prevention of selected diseases.

Highlights for Selected Nationally Notifiable Diseases

Arboviral Encephalitis

The 1997 national total of 127 confirmed or probable California serogroup viral encephalitis cases (all of which were La Crosse encephalitis cases) is the fourth largest yearly total of such cases reported since 1964. The 73 case reports from West Virginia (57% of the national total) represent that state's largest total and an increase of 11% over its 1996 total. Much of the increase in reports from West Virginia may be attributable to this state's recent implementation of an active surveillance system for this disease. La Crosse encephalitis is endemic in the eastern United States, where it is associated with exposure to deciduous forests and *Aedes triseriatus* (the eastern treehole mosquito). A summertime/autumnal outbreak of St. Louis encephalitis in central Florida accounted for nine of the 13 cases reported nationally in 1997. The last major epidemic of St. Louis encephalitis in the United States (223 cases and 11 deaths) occurred in Florida in 1990. St. Louis encephalitis affects persons in portions of both the eastern and western United States. In Florida, the primary mosquito vector of St. Louis encephalitis virus is *Culex nigripalpus*. Fourteen cases of eastern equine encephalitis among humans were reported in 1997 from the South (12 cases), New England (one case), and the Upper Midwest (one case). Eastern equine encephalitis virus is typically transmitted to humans by various *Aedes* mosquito species. No cases of western equine encephalitis among humans have been reported nationally since 1994. The primary mosquito vector of western equine encephalitis virus in the western United States is *Culex tarsalis*.

Cryptosporidium

National reporting for cryptosporidiosis began in 1995 with 2,972 cases reported from 27 states. During 1996, as cryptosporidiosis became a reportable disease in an increased number of states, 2,426 cases were reported from 42 states. In 1997, a total of 2,566 cases were reported from 45 states. Because the diagnosis of cryptosporidiosis is often not considered, and because laboratories do not routinely test for *Cryptosporidium* infection, cryptosporidiosis continues to be underdiagnosed and underreported.

Diphtheria

Four cases of diphtheria were reported in the United States in 1997; two persons, both with localized mild illness, had culture-confirmed diphtheria. One confirmed case was caused by infection with a toxigenic strain of *Corynebacterium diphtheriae*, and was reported from a known endemic focus in South Dakota (*MMWR* 1997;46:506–10); one case caused by nontoxigenic *C. diphtheriae* was reported from Oregon. Two probable cases were reported from Nevada. Both case-patients had acute membranous pharyngitis; oropharyngeal specimens were positive for diphtheria toxin by polymerase chain reaction, but bacterial cultures of these specimens were negative.

In 1997, more than 7,000 cases of diphtheria were reported in an ongoing diphtheria epidemic in the New Independent States of the former Soviet Union. No importations were reported in the United States.

Haemophilus Influenzae (Invasive Disease)

In 1997, a total of 260 cases of *Haemophilus influenzae* (Hi) invasive disease among children aged <5 years were reported. (Data were provided by the National Immunization Program and were based on date of onset, not *MMWR* week.) An estimated 20,000 cases of *Haemophilus influenzae* type b (Hib) invasive disease among children occurred annually prior to Hib vaccine licensure in 1987. (*JAMA* 1993;269:221–6) The dramatic decline is attributed to the widespread administration of the Hib vaccine to preschool-aged children. Of the 260 cases, 201 (77%) isolates were serotyped, and 82 (41%) of the isolates for which serotype was known were type b. Of the 82 cases of Hib invasive disease reported in children aged <5 years, 42 (51%) were aged <6 months, which is too young to have completed a three-dose primary Hib vaccination. However, 27 (68%) of the 40 children who were old enough (aged ≥6 months) to have completed a three-dose primary series before they developed Hib invasive disease were incompletely vaccinated or their vaccination status was unknown. These cases might have been prevented with age-appropriate vaccination.

Hantavirus Pulmonary Syndrome

In 1997, a total of 21 cases of Hantavirus pulmonary syndrome (HPS) were reported. HPS is a pan-American viral zoonosis caused by Sin Nombre virus and other New World hantaviruses, which in the United States, include Bayou virus, Black Creek Canal virus, and New York-1 virus. The identified rodent reservoirs for Sin Nombre, New York-1, Black Creek Canal, and Bayou viruses are, respectively, *Peromyscus maniculatus* (deer mouse), *Peromyscus leucopus* (white-footed mouse), *Sigmodon hispidus* (cotton rat), and *Oryzomys palustris* (rice rat). Cases of HPS have been found in the continental United States, Canada, Argentina, Brazil, Chile, Paraguay, and Uruguay. As of March 31, 1998, national surveillance for HPS has identified 179 confirmed cases in 29 states (case-fatality ratio = 44.7%).

Hemolytic Uremic Syndrome

Post-diarrheal hemolytic uremic syndrome (HUS) is a life-threatening illness characterized by hemolytic anemia, thrombocytopenia, and renal injury. Nearly all cases in the United States are caused by infection with *Shiga* toxin-producing *Escherichia coli*, with serotype O157:H7 being predominant. In 1997, the second year of national reporting, 20 states reported 93 cases of post-diarrheal HUS to CDC. By comparison, 18

states reported 104 cases in 1996. The median age of patients was 4 years (range: 1–89 years), with females accounting for 62% of patients overall. Illness was seasonal, with 50% of cases occurring during July through September.

Hepatitis A

In 1996, the Advisory Committee on Immunization Practices (ACIP) issued recommendations for the prevention of hepatitis A through active or passive immunization (*MMWR* 1996;45[No. RR-15]). The report provides recommendations for use of the hepatitis A vaccines (i.e., HAVRIX®, manufactured by SmithKline Beecham Biologicals, and VAQTA®, manufactured by Merck & Company, Inc.). For communities with high rates of hepatitis A and periodic outbreaks (peak rates: 700 reported cases per 100,000 population), routine vaccination of children aged 2 years and catch-up vaccination of older children is recommended. To control outbreaks in communities with intermediate rates of hepatitis A (i.e., 50–200 reported cases per 100,000 population), vaccination programs targeting subpopulations with the highest rates of disease may be considered. In these communities, ongoing routine vaccination of young children should be implemented to prevent future outbreaks.

Hepatitis C

Hepatitis C virus (HCV) infection is the most common bloodborne infection in the United States. Based on data from the CDC Sentinel Counties Study of Viral Hepatitis, it is estimated that as many as 180,000 new HCV infections occurred each year during the 1980s. Since 1989, the annual number of new infections has declined by 80%. However, in 1996, data from the third National Health and Nutrition Examination Survey, conducted from 1988 through 1994, indicated that approximately 4 million Americans (1.8%) are infected with HCV. Many of these chronically infected persons might not be aware of their infection or be clinically ill, because symptoms of hepatitis C-related chronic liver disease might not develop for 10–20 years after infection. However, such persons can infect others and are at risk for chronic liver disease or other HCV-related chronic diseases. Cirrhosis develops in 10%–20% of persons with HCV-related chronic hepatitis during the first two decades after infection, and 8,000–12,000 persons die from HCV-related chronic liver disease each year. CDC recently published new guidelines for HCV prevention and control (*MMWR* 1998;47[No. RR-19]).

HIV Infection in Children and Infants

In 1997, reports based on AIDS surveillance data indicated substantial declines in perinatally acquired AIDS, reflecting declining perinatal HIV transmission. HIV surveillance data indicated that the increasing use of zidovudine was temporally associated with this substantial decline in perinatally acquired AIDS (*MMWR* 1997;46:1086–92). These data demonstrate success in nationwide efforts to implement Public Health Service guidelines for use of zidovudine to reduce perinatal HIV transmission (*MMWR* 1994;43[No. RR-11]); *MMWR* 1998;47[No. RR-2]) and routine, voluntary prenatal HIV testing (*MMWR* 1995;44[No. RR-7]). States that conduct surveillance of perinatally exposed and infected children can evaluate the impact of the guidelines more completely and document resources needed to care for perinatally exposed infants. In 1997, a total of 30 states conducted surveillance of HIV infection in children, reporting 258 HIV-infected children who had not progressed to AIDS and 200 children who had

AIDS. These states also received 2,238 new reports of perinatally exposed children who required follow up with health-care providers to determine their HIV infection status.

Measles

A total of 138 laboratory-confirmed cases of measles were reported to CDC in 1997, which is the lowest number of measles cases reported in one year and is less than half the previous record low. Of the 138 cases reported, 57 (41%) were international importations, and exposure to these cases resulted in 17 (12%) additional cases. Thus, 74 (54%) cases were associated with importation. An additional seven cases had virologic evidence suggesting an imported measles virus. Fifty-four (41%) measles patients were aged <5 years, 39 (28%) were aged 5–19 years, and 42 (30%) were aged ≥20 years. Thirty-two patients (23%) reported having been vaccinated; seven (5%) received two doses. A total of 13 outbreaks were reported, with the largest involving eight cases. In 1997, no confirmed measles cases were reported from 21 states, and fewer than five cases were reported from 20 states and the District of Columbia.

Plague

In 1997, four plague cases among humans were reported in the United States (two cases in California, one in Arizona, and one in Colorado). One case was fatal and, like two fatal cases that occurred in 1996, septicemic plague was diagnosed postmortem. Each of these cases, which occurred in plague-endemic areas, illustrates the need for health-care providers to maintain a high level of awareness about the risks of human plague. Of the 350 cases reported in the United States from 1970 through 1997, approximately 80% were reported from the southwestern states of New Mexico, Arizona, and Colorado; 9% were reported from California; and nine other western states reported limited numbers of cases. Plague also occurs in animal populations in four other western states that have not reported cases among humans, including Kansas, where *Yersinia pestis*-infected prairie dog fleas were identified in 1997. This is the first report of plague in an animal in Kansas since 1950; however, a nearby county in Oklahoma experienced one case among a person in 1991, and other Great Plains states have reported epizootic activity in recent years (*MMWR* 1994;43:242–6). Internationally, outbreaks of rat-associated plague occurred in the port city of Mahajanga, Madagascar from 1995 through 1997. These are the first port-related outbreaks to be reported from that country in decades. Researchers reported the first case of multidrug-resistant *Y. pestis* in 1997. This isolate, which was obtained in 1995 from a case in Madagascar, contained a plasmid that conferred resistance to antibiotics commonly prescribed for plague treatment or prophylaxis (e.g., streptomycin, chloramphenicol, and tetracycline) (*N Engl J Med* 1997;337:677–80, 702–4).

Poliomyelitis

In 1997, the Advisory Committee on Immunization Practices (ACIP) recommended a change in routine childhood vaccination policy for polio in the United States. The previously recommended schedule of four doses of attenuated oral poliovirus vaccine (OPV) was changed to a sequential schedule of two doses of inactivated poliovirus vaccine (IPV) followed by two doses of OPV for routine vaccination of children. Since

1980, a total of 147 cases have been reported, of which 139 were associated with the use of OPV. The last imported case was reported in 1993.

Streptococcal Disease, Invasive, Group A

According to reports from active surveillance programs in five states (i.e., California, Connecticut, Georgia, Minnesota, and Oregon), the incidence of invasive group A streptococcal disease during 1997 was 4.1 cases/100,000 population; disease incidence ranged from 2.2 to 5.1 cases/100,000 population among the surveillance areas. Streptococcal toxic shock syndrome and necrotizing fasciitis accounted for approximately 6.9% and 7.7% of invasive cases, respectively. Overall case-fatality among patients with invasive group A streptococcal disease was 13%; case-fatality rates were higher among patients with streptococcal toxic shock syndrome and necrotizing fasciitis (43% and 21%, respectively). Risk factors for invasive group A streptococcal disease include elderly age, HIV infection, diabetes, cancer, alcohol abuse, and varicella infection.

Streptococcus pneumoniae, Drug-Resistant

The proportion of drug-resistant *Streptococcus pneumoniae* isolates continues to increase, according to reports from active surveillance programs in seven states (i.e., California, Connecticut, Georgia, Maryland, Minnesota, Oregon, and Tennessee). During 1997, approximately 26% of pneumococcal isolates obtained from sterile sites were no longer susceptible to penicillin (mean inhibitory concentration [MIC] ≥0.1 µg/mL). In 1997, the proportion of all isolates with high-level penicillin resistance (MIC ≥2 µg/mL), increased from 12% in 1996 to 14.4%; a total of 7.2% of isolates had MICs ≥4 µg/mL compared with 5.4% in 1996. The resistant proportion varied widely by geographic region. To limit the contribution of unnecessary antimicrobial use to the spread of drug-resistant *S. pneumoniae*, CDC and the American Academy of Pediatrics issued recommendations for judicious use of antimicrobial agents for upper-respiratory-tract infections among children (*Pediatrics* 1998;101[suppl]). Educational materials concerning the principles of judicious antimicrobial use can be obtained by calling the National Center for Infectious Diseases at (404) 639-4702 for an order form.

Tetanus

Fifty cases of tetanus were reported in 1997. During 1995–1997, an average annual incidence of 41 cases were reported, the lowest ever reported since national tetanus surveillance began in 1947. The average annual incidence of 0.15 cases per million population represents a slight decline from the incidence of 0.2 cases per million population reported during 1991–1994.

Highlights for Selected Non-Notifiable Diseases

Cyclosporiasis

In 1997, several outbreaks of cyclosporiasis associated with various types of fresh produce (e.g., raspberries, mesclun lettuce, and basil) occurred in the United States. In the largest outbreak, which was associated with consumption of fresh raspberries, 41 clusters with a total of 762 cases (25% were laboratory confirmed) were reported by 13 states, the District of Columbia, and one province in Canada.

Dengue

Fifty-six laboratory-positive cases of dengue were imported into the United States in 1997 and diagnosed at the CDC Dengue Branch. This number represents a 30% increase from the number of laboratory-confirmed cases reported in 1996 (n=43). Similarly, the total number of dengue and dengue hemorrhagic fever (DHF) cases reported by Pan American Health Organization member countries in 1997 (n=364,945) was 46% higher than the 1996 total (n=250,707). Autochthonous dengue cases (n=3) were documented in south Texas again in 1997, underscoring the risk of dengue transmission in southern gulf coast states where mosquito vectors occur. After a 15-year absence, dengue cases were reported from Cuba in 1997. The municipality of Santiago de Cuba experienced an outbreak with 2,946 laboratory-diagnosed cases and 205 DHF cases, which resulted in 12 deaths.

HIV Infection in Adults

In June 1997, HIV-infection reporting for adults (i.e., persons aged ≥13 years) was added to the list of nationally notifiable diseases at a Council of State and Territorial Epidemiologists (CSTE) meeting. During 1997, reports based on acquired immunodeficiency syndrome (AIDS) surveillance data highlighted substantial declines in AIDS incidence and deaths. As a result of improvements in treatment and care of persons infected with the human immunodeficiency virus (HIV), surveillance of AIDS alone no longer accurately reflects the magnitude or direction of the epidemic. Data concerning persons in whom HIV infection is diagnosed before AIDS is diagnosed are needed to determine populations that could benefit from prevention and treatment services. CSTE recommends that all states and territories implement confidential HIV infection reporting based on methods that provide accurate and representative data for all persons confidentially diagnosed with HIV infection.

Influenza A (H5N1)

In May 1997, the first known case of disease among humans caused by influenza A (H5N1) virus occurred in a previously healthy 3-year-old child in Hong Kong; this child died from his illness. An additional 17 cases (including five deaths) were detected in November and December 1997. All cases occurred coincident with outbreaks of highly pathogenic avian influenza A (H5N1) virus among poultry. At the end of December, Hong Kong authorities initiated the slaughter of all chickens in Hong Kong and, since then, no additional cases of influenza A (H5N1) virus have been detected among humans despite enhanced surveillance. The pandemic potential of influenza A (H5N1) viruses remains unknown. No cases of H5N1 infection were reported in the United States.

Tularemia

Tularemia was removed from the nationally notifiable disease list in 1995. However, as of January 1998, a total of 36 states maintained tularemia as a notifiable condition. Based on a telephone survey of state departments of health conducted from 1995 through 1997, a total of 313 cases of tularemia were reported by 43 states (119 cases in 1995, 89 cases in 1996, and 105 cases in 1997). Of these, 155 (49%) were reported from Missouri, Oklahoma, Kansas, and Arkansas.

Vancomycin-Resistant Enterococci (VRE)

The magnitude and impact of vancomycin-resistant enterococci (VRE) in the United States are demonstrated by CDC's National Nosocomial Infections Surveillance (NNIS) system, which includes more than 275 U.S. hospitals. Additional data are available on the Internet at http://www.cdc.gov/ncidod/hip/Surveill/surveill.htm. During 1989–1997, the percentage of enterococci resistant to vancomycin isolated from patients in intensive care units with nosocomial infections increased from 0.4% to 23.2% (Table). The percentage of VRE isolated from patients in noncritical care units with nosocomial infections increased from 0.3% to 15.4%.

TABLE: Percentage of nosocomial enterococci reported as resistant to vancomycin, by health-care setting and year*

Year	Intensive care unit (ICU)†	Non-ICU†
1989	0.4	0.3
1990	1.5	0.8
1991	5.3	2.9
1992	7.1	2.9
1993	11.6	4.8
1994	13.6	9.0
1995	12.8	12.0
1996	16.6	11.6
1997	23.2	15.4

*N>2000 isolates for each year.
†P<0.0001, chi-square for linear trend.

Source: NNIS System, Hospital Infections Program, National Center for Infectious Diseases

NOTIFIABLE DISEASES — Summary of reported cases, by month, United States, 1997

NAME	Total	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug	Sept.	Oct.	Nov	Dec	Unk
AIDS*	58,492	4,682	5,066	5,364	4,586	5,072	5,234	4,281	4,803	4,964	4,636	4,016	5,788	–
Botulism, total	132	9	5	8	2	14	9	19	16	8	8	20	14	–
Brucellosis	98	20	1	6	4	7	6	10	13	8	3	9	11	–
Chancroid†	243		65			80			58			40		–
Chlamydia†§	526,671		119,217			130,697			135,403			141,354		–
Cholera	6	–	–	–	–	1	–	–	2	–	2	1	–	–
Cryptosporidiosis	2,566	146	94	154	121	152	117	211	358	311	293	310	299	–
Diphtheria	4	–	–	2	1	–	1	–	–	–	–	–	–	–
Escherichia coli O157:H7	2,555	82	73	107	71	173	190	400	432	335	281	196	215	–
Gonorrhea†	324,907		74,417			76,126			87,378			86,986		–
Haemophilus influenzae, invasive	1,162	71	86	123	98	116	103	69	82	76	58	103	177	–
Hansen disease (leprosy)	122	6	4	12	11	12	5	4	7	11	2	19	29	–
Hepatitis A	30,021	1,716	2,184	2,885	2,033	3,124	2,163	2,091	2,628	2,517	2,526	2,524	3,630	–
Hepatitis B	10,416	696	637	947	736	1,022	774	731	955	809	735	923	1,451	–
Hepatitis, C/non-A non-B	3,816	273	257	322	246	384	291	304	370	319	242	312	496	–
Legionellosis	1,163	61	84	72	63	83	69	75	116	112	127	152	149	–
Lyme disease	12,801	512	254	390	293	612	724	1,638	3,197	1,944	1,057	988	1,192	–
Malaria	2,001	124	98	111	100	168	181	188	279	160	147	181	264	–
Measles (rubeola)	138	3	3	9	14	31	10	21	13	9	11	3	11	–
Meningococcal disease	3,308	138	348	469	282	360	248	175	184	171	168	230	535	–
Mumps	683	32	46	72	63	101	57	25	37	61	45	72	72	–
Pertussis (whooping cough)	6,564	607	403	512	537	475	404	393	543	475	397	740	1,078	–
Plague	4	–	–	–	–	1	1	–	–	1	–	1	–	–
Poliomyelitis, paralytic	3	1	–	–	–	1	–	–	–	–	–	–	1	–
Psittacosis	33	2	2	4	5	5	2	–	4	3	2	–	4	–
Rabies, animal	8,105	268	422	667	741	781	678	599	830	832	862	707	718	–
Rabies, human	2	–	–	1	–	–	–	–	–	–	–	–	1	–
Rocky Mountain spotted fever	409	20	7	14	11	24	58	54	87	48	45	25	16	–
Rubella (German measles)	181	10	4	7	10	30	34	36	7	10	17	1	15	–
Rubella, congenital syndrome	5	–	–	1	–	1	–	–	–	1	–	–	2	–
Salmonellosis	41,901	1,663	2,030	2,544	2,351	3,391	3,175	3,626	5,398	4,364	3,961	4,219	5,179	–
Shigellosis	23,117	1,572	1,200	1,301	1,064	1,615	1,522	1,694	2,717	2,166	2,100	2,792	3,374	–
Syphilis, total all stages†	46,540		11,872			13,007			11,371			10,290		–
Primary and secondary†	8,550		2,264			2,252			2,198			1,836		–
Congenital <1 year†	1,049		331			279			243			196		–
Tetanus	50	5	3	5	2	8	5	4	3	2	2	7	4	–
Toxic-shock syndrome	157	15	9	13	14	13	9	12	16	12	10	12	22	–
Trichinosis	13	5	–	–	–	–	–	–	4	–	–	–	4	–
Tuberculosis¶	19,851	794	1,285	1,630	1,790	1,813	1,553	1,697	1,644	1,583	1,601	1,442	3,019	–
Typhoid fever	365	9	20	28	17	33	25	23	43	44	35	36	52	–
Varicella (chickenpox)**	98,727	5,463	10,792	15,484	11,394	17,909	6,744	2,665	1,370	2,159	3,069	6,748	14,930	–

*The total number of acquired immunodeficiency syndrome (AIDS) cases includes all cases reported to the Division of HIV/AIDS Prevention — Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP) as of December 31, 1997.

†Cases were updated through the Division of Sexually Transmitted Diseases Prevention, NCHSTP, as of July 13, 1998.

§Chlamydia refers to genital infections caused by *C. trachomatis*.

¶Cases were updated through the Division of Tuberculosis Elimination, NCHSTP, as of April 15, 1998

**Not nationally notifiable.

NOTIFIABLE DISEASES — Reported cases, by geographic division and area, United States, 1997

Area	Total resident population (in thousands)	AIDS*	Botulism		Brucellosis	Chancroid†	*Chlamydia trachomatis* infection†
			Foodborne	Infant			
United States	**267,637**	**58,492**	**31**	**79**	**98**	**243**	**526,671**
New England	**13,379**	**2,372**	**–**	**–**	**1**	**4**	**18,433**
Maine	1,242	51	–	–	–	–	1,066
N H	1,173	55	–	–	–	–	816
Vt	589	29	–	–	–	NN	434
Mass	6,118	863	–	–	1	4	7,984
R I.	987	152	–	–	–	–	2,069
Conn	3,270	1,222	–	–	–	–	6,064
Mid Atlantic	**38,210**	**18,327**	**–**	**17**	**3**	**119**	**58,653**
Upstate N Y.	10,828	3,858	–	2	1	–	NN
N.Y City	7,309	9,331	–	–	–	119	28,468
N J.	8,053	3,226	–	3	–	–	10,347
Pa.	12,020	1,912	–	12	2	–	19,838
E.N. Central	**43,890**	**4,350**	**1**	**6**	**12**	**8**	**86,404**
Ohio	11,186	848	–	3	2	3	22,827
Ind	5,864	523	–	–	–	–	9,600
Ill	11,896	1,842	1	1	7	5	23,024
Mich	9,774	882	–	–	3	–	21,399
Wis	5,170	255	NA	2	NA	–	9,554
W.N Central	**18,571**	**1,166**	**–**	**–**	**7**	**–**	**32,968**
Minn.	4,686	214	–	–	–	–	6,631
Iowa	2,852	101	–	NN	4	–	4,907
Mo	5,402	577	–	–	2	–	12,308
N. Dak	641	13	–	–	NN	NN	902
S Dak.	738	11	–	–	–	–	1,450
Nebr	1,657	91	–	–	1	–	2,767
Kans.	2,595	159	–	–	–	–	4,003
S. Atlantic	**48,230**	**13,858**	**1**	**3**	**8**	**30**	**106,486**
Del	732	231	–	–	–	–	2,613
Md	5,094	1,875	–	–	–	1	13,763
D C	529	998	–	–	1	–	3,069
Va	6,734	1,175	–	–	1	1	11,615
W Va	1,816	130	–	2	–	–	3,108
N C	7,425	850	1	–	3	9	17,108
S C	3,760	779	–	–	–	15	12,511
Ga	7,486	1,722	–	1	1	1	15,911
Fla.	14,654	6,098	–	–	2	3	26,788
E S. Central	**16,326**	**2,062**	**–**	**–**	**2**	**2**	**35,437**
Ky	3,908	361	–	–	1	–	6,332
Tenn.	5,368	784	–	–	1	1	12,502
Ala	4,319	570	–	–	–	1	8,704
Miss	2,731	347	–	–	–	–	7,899
W S. Central	**29,631**	**6,337**	**1**	**11**	**20**	**57**	**72,139**
Ark	2,523	242	–	1	1	1	2,503
La	4,352	1,094	–	1	–	3	11,545
Okla	3,317	283	–	–	–	–	7,416
Tex	19,439	4,718	1	9	19	53	50,675
Mountain	**16,483**	**1,850**	**1**	**8**	**8**	**1**	**29,216**
Mont.	879	41	–	–	–	–	1,146
Idaho	1,210	52	–	2	–	–	1,709
Wyo	480	16	–	–	2	1	635
Colo	3,893	380	–	–	2	–	7,196
N Mex.	1,730	169	–	1	1	–	4,021
Ariz	4,555	448	1	2	3	–	10,783
Utah	2,059	152	–	2	–	–	1,774
Nev.	1,677	592	–	1	–	–	1,952
Pacific	**42,917**	**8,121**	**27**	**34**	**37**	**22**	**86,935**
Wash.	5,610	641	3	–	3	2	9,574
Oreg.	3,243	305	3	2	1	1	5,270
Calif	32,268	7,029	2	29	30	19	68,647
Alaska	609	52	19	–	–	–	1,615
Hawaii	1,187	94	–	3	3	–	1,829
Guam	145	2	–	–	–	–	368
PR	3,827	2,040	–	–	–	1	2,123
V I	114	99	NA	NA	NA	NA	14
American Samoa	60	–	NA	NA	NA	NA	NA
C.N M I	63	1	–	–	–	NA	NA

*Totals reported to Division of HIV/AIDS Prevention — Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP), as of December 31, 1997 Total includes 49 cases in persons with unknown state of residence
†Cases were updated through the Division of Sexually Transmitted Diseases Prevention, NCHSTP, as of July 13, 1998

NOTIFIABLE DISEASES — Reported cases, by geographic division and area, United States, 1997 (continued)

Area	Measles		Meningo-coccal disease	Mumps	Pertussis	Plague	Polio-myelitis, paralytic
	Indigenous	Imported*					
United States	**81**	**57**	**3,308**	**683**	**6,564**	**4**	**3**
New England	**11**	**8**	**209**	**14**	**1,096**	–	–
Maine	–	1	19	–	26	–	–
N.H.	1	–	17	1	150	–	–
Vt	–	–	4	–	283	–	–
Mass	10	6	100	4	582	–	–
R I	–	–	24	8	19	–	–
Conn	–	1	45	1	36	–	–
Mid. Atlantic	**18**	**9**	**357**	**66**	**503**	–	–
Upstate N.Y.	2	3	97	16	214	–	–
N.Y. City	8	3	57	4	78	–	–
N J.	3	–	75	8	14	–	–
Pa.	5	3	128	38	197	–	–
E.N. Central	**6**	**4**	**499**	**99**	**714**	–	–
Ohio	–	–	164	35	165	–	–
Ind	–	–	60	15	104	–	–
Ill.	6	1	156	17	155	–	–
Mich	–	2	72	28	71	–	–
Wis	–	1	47	4	219	NN	NN
W.N. Central	**14**	**3**	**248**	**19**	**890**	–	–
Minn	5	3	41	7	547	–	–
Iowa	–	–	47	10	207	–	–
Mo.	1	–	106	–	80	–	–
N. Dak.	–	–	2	–	2	–	–
S. Dak	8	–	6	–	5	–	–
Nebr.	–	–	20	1	16	–	–
Kans	–	–	26	1	33	–	–
S. Atlantic	**4**	**14**	**578**	**85**	**446**	–	**1**
Del	–	–	5	–	1	–	–
Md.	–	2	42	1	119	–	–
D C	–	2	12	–	3	–	–
Va.	–	1	60	21	59	–	–
W. Va.	1	–	19	–	6	–	–
N.C	–	2	97	12	118	–	–
S C.	–	1	64	11	32	–	–
Ga	–	1	108	11	18	–	–
Fla	3	5	171	29	90	–	1
E.S. Central	–	**1**	**242**	**34**	**159**	–	–
Ky.	–	–	50	3	74	–	–
Tenn	–	–	77	8	40	–	–
Ala.	–	1	85	9	34	–	–
Miss.	–	–	30	14	11	–	–
W S. Central	**3**	**5**	**335**	**98**	**376**	–	**1**
Ark.	–	–	38	3	62	–	–
La	–	–	57	17	21	–	–
Okla	–	1	45	3	60	–	–
Tex.	3	4	195	75	233	–	1
Mountain	**6**	**2**	**189**	**61**	**1,333**	**2**	–
Mont	–	–	8	–	18	–	–
Idaho	–	–	15	6	570	–	–
Wyo.	–	–	3	1	7	–	–
Colo.	–	–	51	3	415	1	–
N Mex.	–	–	31	NN	198	–	–
Ariz.	5	–	44	34	45	1	–
Utah	–	1	17	8	29	–	–
Nev	1	1	20	9	51	–	–
Pacific	**19**	**11**	**651**	**207**	**1,047**	**2**	**1**
Wash.	1	1	115	21	481	–	–
Oreg	–	–	124	NN	48	–	–
Calif.	16	8	402	151	483	2	1
Alaska	–	–	3	8	16	–	–
Hawaii	2	2	7	27	19	–	–
Guam	–	–	1	1	–	–	–
P.R	–	–	8	7	–	–	–
V I.	–	–	1	1	–	NA	–
American Samoa	NA	NA	NA	NA	NA	NA	NA
C N M I	1	–	–	4	–	–	–

*Imported cases include only those resulting from importation from other countries

NOTIFIABLE DISEASES — Reported cases, by geographic division and area, United States, 1997 (continued)

Area	Psitta-cosis	Rabies		RMSF*	Rubella		Salmonel-losis	Shigel-losis
		Animal	Human		Rubella	Cong. syndrome		
United States	**33**	**8,105**	**2**	**409**	**181**	**5**	**41,901**	**23,117**
New England	**1**	**1,257**	**–**	**5**	**6**	**–**	**2,348**	**592**
Maine	1	227	–	–	–	–	137	15
N H	–	49	–	–	–	–	151	54
Vt	–	113	–	–	–	–	88	11
Mass	–	282	–	1	1	–	1,259	316
R I	–	42	–	1	–	–	167	95
Conn	–	544	–	3	5	–	546	101
Mid. Atlantic	**5**	**1,722**	**–**	**39**	**40**	**–**	**6,505**	**3,168**
Upstate N.Y	3	1,264	–	8	11	–	1,649	801
N Y City	–	NA	–	6	29	–	1,796	956
N J	–	190	–	9	–	–	1,501	625
Pa	2	268	–	16	–	–	1,559	786
E N. Central	**4**	**203**	**–**	**19**	**6**	**–**	**6,207**	**2,552**
Ohio	–	116	–	12	–	–	1,545	835
Ind.	–	13	–	3	–	–	590	88
Ill	–	20	–	3	2	–	1,935	1,163
Mich	4	28	–	–	–	–	906	346
Wis.	NA	26	NA	1	4	NN	1,231	120
W.N Central	**2**	**537**	**–**	**35**	**2**	**–**	**2,287**	**908**
Minn	1	70	–	1	–	–	632	138
Iowa	–	160	–	1	–	–	297	90
Mo.	1	31	–	24	2	–	568	222
N Dak.	NN	91	–	–	–	–	69	10
S Dak.	–	94	–	2	–	–	90	31
Nebr	–	2	–	–	–	–	185	284
Kans.	–	89	–	7	–	–	446	133
S Atlantic	**7**	**3,109**	**–**	**136**	**79**	**1**	**8,475**	**4,499**
Del.	1	67	–	–	–	–	101	35
Md	1	603	–	20	–	–	1,231	423
D C	–	5	–	–	1	–	115	47
Va	–	678	–	23	1	–	1,120	416
W Va	–	89	–	3	–	–	133	27
N C.	1	879	–	35	59	–	1,226	387
S C	1	186	–	36	15	–	603	87
Ga	–	324	–	11	–	–	1,356	1,131
Fla	3	278	–	8	3	1	2,590	1,946
E.S. Central	**–**	**271**	**–**	**91**	**1**	**–**	**1,771**	**1,127**
Ky	–	29	–	5	–	–	373	449
Tenn	–	149	–	40	–	–	443	291
Ala.	–	88	–	9	1	–	470	272
Miss.	–	5	–	37	NN	–	485	115
W S. Central	**–**	**439**	**–**	**69**	**12**	**–**	**4,246**	**4,252**
Ark	–	56	–	31	–	–	445	273
La	–	7	–	5	–	–	617	182
Okla.	–	113	–	29	–	–	391	293
Tex.	–	263	–	4	12	–	2,793	3,504
Mountain	**3**	**197**	**1**	**12**	**7**	**1**	**2,587**	**1,913**
Mont.	–	52	1	4	–	–	63	11
Idaho	–	–	–	5	2	–	141	79
Wyo.	–	31	–	1	–	–	49	5
Colo	3	34	–	–	–	–	608	258
N. Mex	–	13	–	–	–	–	311	331
Ariz	–	53	–	1	5	1	853	1,076
Utah	–	6	–	1	–	–	271	101
Nev	–	8	–	–	–	–	291	52
Pacific	**11**	**370**	**1**	**3**	**28**	**3**	**7,475**	**4,106**
Wash	1	–	1	–	5	–	680	318
Oreg	2	14	–	1	–	–	368	189
Calif.	8	327	–	2	14	3	5,993	3,528
Alaska	–	29	–	–	–	NN	50	6
Hawaii	–	–	–	–	9	–	384	65
Guam	–	–	–	–	–	–	24	35
PR	–	71	–	–	–	–	838	70
VI	NA	NA	NA	NA	–	–	10	2
American Samoa	NA	NA	NA	NA	NA	NA	NA	NA
C N M I	–	–	–	–	–	–	43	34

*Rocky Mountain spotted fever.

NOTIFIABLE DISEASES — Summary of reported cases, by sex,* United States, 1997

NAME	Total	Male No.	Male (Rate)	Female No	Female (Rate)	Sex not stated
AIDS[†]	58,492	45,737	(35 23)	12,755	(9 42)	–
Botulism, total	132	55	(0.04)	73	(0.04)	4
Brucellosis	98	56	(0.04)	39	(0.03)	3
Chancroid[§]	243	157	(0 12)	69	(0 05)	17
Chlamydia[§¶]	526,671	–	(–)	436,366	(322 10)	2,663
Cholera	6	1	(0 00)	4	(0 00)	1
Cryptosporidiosis	2,566	1,331	(1 20)	1,200	(1 04)	35
Diphtheria	4	1	(0 00)	3	(0 00)	–
Escherichia coli O157:H7	2,555	1,161	(0.97)	1,317	(1 06)	77
Gonorrhea[§]	324,907	162,796	(125.41)	161,661	(119 33)	450
Haemophilus influenzae (Invasive Disease)	1,162	522	(0 40)	596	(0 44)	44
Hansen disease (leprosy)	122	64	(0 05)	32	(0.02)	26
Hepatitis A	30,021	16,599	(12.79)	10,969	(8 10)	2,453
Hepatitis B	10,416	6,115	(4.71)	4,045	(2 99)	256
Hepatitis, C/non-A non-B	3,816	2,424	(1.99)	1,354	(1.06)	38
Legionellosis	1,163	682	(0.53)	457	(0.34)	24
Lyme disease	12,801	6,703	(5.16)	6,016	(4 44)	82
Malaria	2,001	1,258	(0.97)	690	(0.51)	53
Measles (rubeola)	138	70	(0.05)	62	(0.05)	6
Meningococcal disease	3,308	1,662	(1.28)	1,583	(1.17)	63
Mumps	683	348	(0.27)	286	(0.22)	49
Pertussis (whooping cough)	6,564	3,036	(2.34)	3,468	(2.56)	60
Plague	4	1	(0 00)	2	(0.00)	1
Poliomyelitis, paralytic	3	1	(0 00)	2	(0.00)	–
Psittacosis	33	12	(0 01)	21	(0 02)	–
Rabies, human	2	2	(0 00)	–	(0 00)	–
Rocky Mountain spotted fever	409	248	(0 19)	157	(0 12)	4
Rubella (German measles)	181	109	(0.08)	67	(0.05)	5
Salmonellosis	41,901	16,716	(12.88)	17,477	(12.90)	7,708
Shigellosis	23,117	8,437	(6 50)	9,758	(7.20)	4,922
Syphilis, primary and secondary[§]	8,550	4,656	(3.59)	3,891	(2.87)	3
Tetanus	50	29	(0.02)	21	(0.02)	–
Toxic-shock syndrome	157	38	(0 03)	115	(0.09)	4
Trichinosis	13	6	(0 00)	7	(0.01)	–
Tuberculosis**	19,851	12,371	(9.53)	7,474	(5.52)	6
Typhoid fever	365	192	(0 15)	168	(0 12)	5

NOTE: Rates <0.01 after rounding are listed as 0.00.

*July 1, 1997, postcensal population estimates were used to calculate incidence rates per 100,000 population.

[†]The total number of acquired immunodeficiency syndrome (AIDS) cases includes all cases reported to the Division of HIV/AIDS Prevention — Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP) as of July 13, 1998

[§]Cases were updated through the Division of Sexually Transmitted Diseases Prevention, NCHSTP, as of December 31, 1997.

[¶]Chlamydia refers to genital infections caused by *C. trachomatis*. The rates for men are not presented because reporting for men is more limited than for women.

**Cases were updated through the Division of Tuberculosis Elimination, NCHSTP, as of April 15, 1998.

NOTIFIABLE DISEASES — Summary of reported cases, by race, United States, 1997

Name	Total	American Indian or Alaskan Native		Asian or Pacific Islander		Black		White		Other		Race not stated	
		No.	%	No.	%	No.	%	No.	%	No.	%	No	%
AIDS*	58,492	206	(<1)	446	(1)	27,018	(46)	20,188	(35)	–	(–)	10,634†	(18)
Botulism, total	132	19	(14)	6	(5)	7	(5)	71	(54)	–	(–)	29	(22)
Brucellosis	98	–	(–)	3	(3)	–	(–)	50	(51)	–	(–)	45	(46)
Chlamydia§	520,164	6,915	(1)	5,034	(1)	164,232	(32)	107,527	(21)	–	(–)	236,456†	(45)
Cholera	6	–	(–)	–	(–)	–	(–)	3	(50)	–	(–)	3	(50)
Cryptosporidiosis	2,566	249	(10)	23	(1)	196	(8)	1,262	(49)	1	(<1)	835	(33)
Diphtheria	4	2	(50)	–	(–)	–	(–)	2	(50)	–	(–)	–	(–)
Escherichia coli O157:H7	2,555	127	(5)	27	(1)	68	(3)	1,504	(59)	3	(<1)	826	(32)
Gonorrhea§	323,307	1,532	(<1)	1,021	(<1)	190,948	(59)	35,958	(11)	–	(–)	93,848†	(29)
Haemophilus influenzae (Invasive Disease)	1,162	67	(6)	20	(2)	162	(14)	685	(59)	1	(<1)	227	(20)
Hansen disease (leprosy)	122	–	(–)	33	(27)	7	(6)	30	(25)	–	(–)	52	(43)
Hepatitis A	30,021	528	(2)	445	(1)	2,013	(7)	17,819	(59)	69	(<1)	9,147	(30)
Hepatitis B	10,416	72	(1)	752	(7)	2,201	(21)	4,096	(39)	53	(1)	3,242	(31)
Hepatitis, C/non-A non-B	3,816	60	(2)	46	(1)	460	(12)	2,156	(56)	16	(<1)	1,078	(28)
Legionellosis	1,163	1	(<1)	7	(1)	97	(8)	809	(70)	–	(–)	249	(21)
Lyme disease	12,801	23	(<1)	86	(1)	185	(1)	9,645	(75)	27	(<1)	2,835	(22)
Malaria	2,001	1	(<1)	286	(14)	554	(28)	475	(24)	51	(3)	634	(32)
Measles (rubeola)	138	9	(7)	18	(13)	10	(7)	91	(66)	1	(1)	9	(7)
Meningococcal disease	3,308	41	(1)	35	(1)	553	(17)	2,090	(63)	9	(<1)	580	(18)
Mumps	683	1	(<1)	58	(8)	46	(7)	336	(49)	–	(–)	242	(35)
Pertussis (whooping cough)	6,564	205	(3)	66	(1)	332	(5)	4,079	(62)	9	(<1)	1,873	(29)
Plague	4	2	(50)	–	(–)	–	(–)	2	(50)	–	(–)	–	(–)
Poliomyelitis, paralytic	3	–	(–)	–	(–)	–	(–)	3	(100)	–	(–)	–	(–)
Psittacosis	33	–	(–)	–	(–)	–	(–)	25	(76)	–	(–)	8	(24)
Rabies, human	2	1	(50)	–	(–)	–	(–)	–	(–)	–	(–)	1	(50)
Rocky Mountain spotted fever	409	10	(2)	2	(<1)	19	(5)	303	(74)	–	(–)	75	(18)
Rubella (German measles)	181	4	(2)	14	(8)	7	(4)	73	(40)	4	(2)	79	(44)
Rubella, congenital syndrome	5	–	(–)	1	(20)	–	(–)	1	(20)	–	(–)	3	(60)
Salmonellosis	41,901	262	(1)	594	(1)	3,303	(8)	17,956	(43)	24	(<1)	19,762	(47)
Shigellosis	23,117	543	(2)	115	(<1)	3,055	(13)	8,739	(38)	23	(<1)	10,642	(46)
Syphilis, primary and secondary§	8,540	40	(<1)	32	(<1)	6,864	(80)	951	(11)	–	(–)	653†	(8)
Tetanus	50	10	(20)	–	(–)	3	(6)	33	(66)	1	(2)	3	(6)
Toxic-shock syndrome	157	1	(1)	3	(2)	13	(8)	117	(75)	–	(–)	23	(15)
Trichinosis	13	–	(–)	–	(–)	–	(–)	4	(31)	–	(–)	9	(69)
Tuberculosis¶	19,851	276	(1)	3,873	(20)	6,806	(34)	8,862	(45)	–	(–)	34	(<1)
Typhoid fever	365	2	(1)	114	(31)	27	(7)	56	(15)	19	(5)	147	(40)

*The total number of acquired immunodeficiency syndrome (AIDS) cases includes all cases reported to the Division of HIV/AIDS Prevention — Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP) as of December 31, 1997.

†Includes the following cases originally reported as Hispanic: 10,394 for AIDS; 62,716 for chlamydia, 13,990 for gonorrhea, and 450 for syphilis, primary and secondary.

§In addition to data collected through the National Electronic Telecommunications System for Surveillance (NETSS), some data concerning race are collected on aggregate forms different from those used for numbers of reported cases. Thus, the total number of cases reported on this table can differ slightly from other tables Cases were updated through the Division of Sexually Transmitted Diseases Prevention, NCHSTP, as of July 13, 1998. Data regarding race for 1997 are unavailable for chancroid.

¶Cases were updated through the Division of Tuberculosis Elimination, NCHSTP as of April 15, 1998

NOTIFIABLE DISEASES — Summary of reported cases, by ethnicity, United States, 1997

NAME	Total	Hispanic		Non-Hispanic		Ethnicity not stated	
		No	(%)	No.	(%)	No.	(%)
AIDS*	58,492	10,394	(18)	47,206	(81)	892†	(2)
Botulism, total	132	24	(18)	82	(62)	26	(20)
Brucellosis	98	59	(60)	15	(15)	24	(24)
Chlamydia§	520,164	62,716	(12)	271,759	(52)	185,689†	(36)
Cholera	6	3	(50)	1	(17)	2	(33)
Cryptosporidiosis	2,566	178	(7)	1,366	(53)	1,022	(40)
Diphtheria	4	–	(–)	3	(75)	1	(25)
Escherichia coli O157:H7	2,555	88	(3)	1,464	(57)	1,003	(39)
Gonorrhea§	323,307	13,990	(4)	226,906	(70)	82,411†	(25)
Haemophilus influenzae (Invasive Disease)	1,162	93	(8)	695	(60)	374	(32)
Hansen disease (leprosy)	122	35	(29)	51	(42)	36	(30)
Hepatitis A	30,021	6,828	(23)	13,341	(44)	9,852	(33)
Hepatitis B	10,416	940	(9)	5,264	(51)	4,212	(40)
Hepatitis, C/non-A non-B	3,816	475	(12)	1,721	(45)	1,620	(42)
Legionellosis	1,163	32	(3)	670	(58)	461	(40)
Lyme disease	12,801	140	(1)	7,750	(61)	4,911	(38)
Malaria	2,001	176	(9)	1,041	(52)	784	(39)
Measles (rubeola)	138	22	(16)	106	(77)	10	(7)
Meningococcal disease	3,308	311	(9)	2,023	(61)	974	(29)
Mumps	683	159	(23)	263	(39)	261	(38)
Pertussis (whooping cough)	6,564	594	(9)	3,444	(52)	2,526	(38)
Plague	4	–	(–)	4	(100)	–	(–)
Poliomyelitis, paralytic	3	2	(67)	1	(33)	–	(–)
Psittacosis	33	–	(–)	19	(58)	14	(42)
Rabies, human	2	–	(–)	–	(–)	2	(100)
Rocky Mountain spotted fever	409	4	(1)	253	(62)	152	(37)
Rubella (German measles)	181	109	(60)	46	(25)	26	(14)
Rubella, congenital syndrome	5	3	(60)	1	(20)	1	(20)
Salmonellosis	41,901	2,447	(6)	16,284	(39)	23,170	(55)
Shigellosis	23,117	3,427	(15)	8,051	(35)	11,639	(50)
Syphilis, primary and secondary§	8,540	450	(5)	7,815	(92)	275†	(3)
Tetanus	50	14	(28)	27	(54)	9	(18)
Toxic-shock syndrome	157	3	(2)	104	(66)	50	(32)
Trichinosis	13	–	(–)	4	(31)	9	(69)
Tuberculosis¶	19,851	4,228	(21)	15,586	(79)	37	(–)
Typhoid fever	365	56	(15)	181	(50)	128	(35)

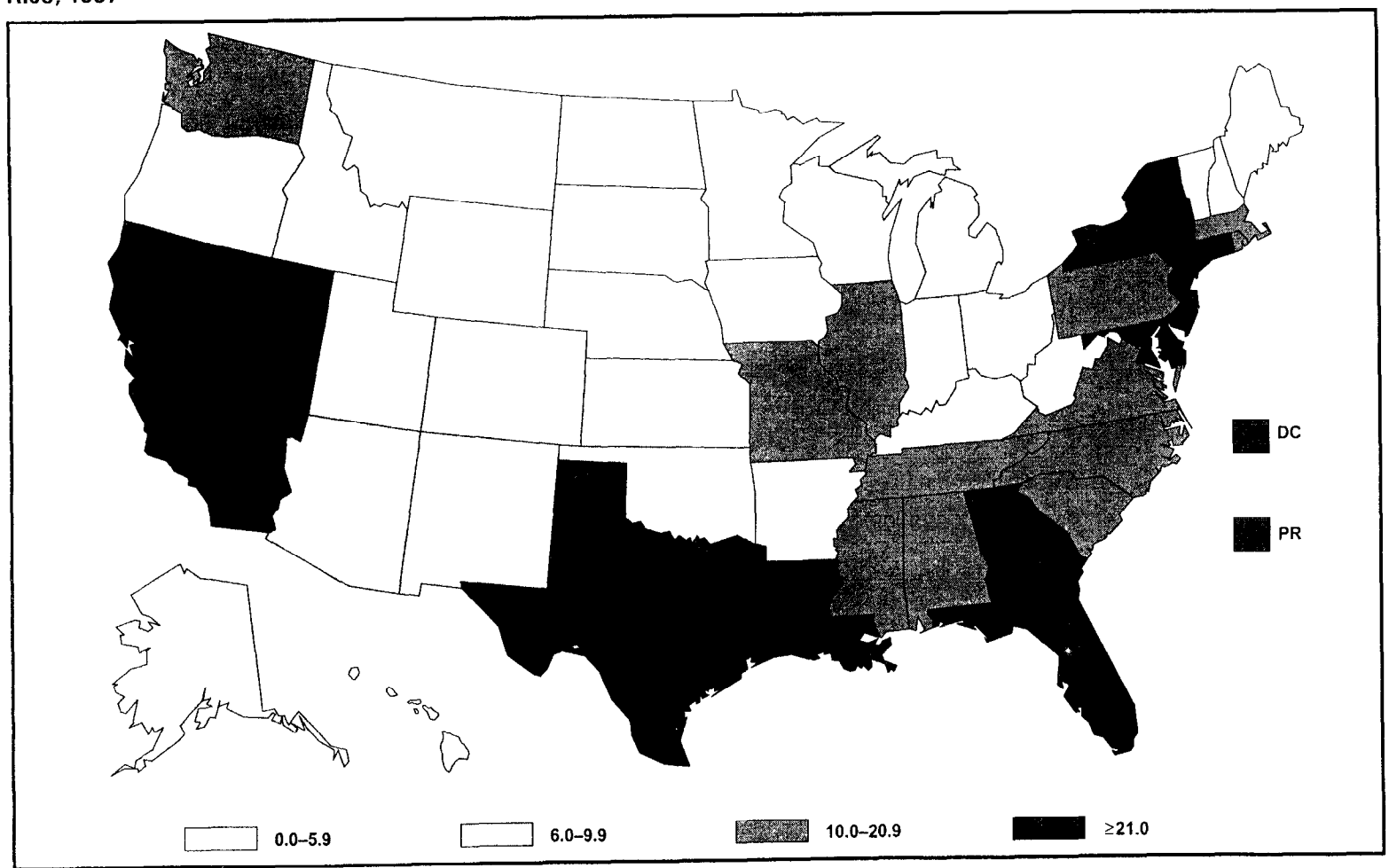
*The total number of acquired immunodeficiency syndrome (AIDS) cases includes all cases reported to the Division of HIV/AIDS Prevention — Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP) as of December 31, 1997.

†Ethnicity is not stated and includes cases originally reported as American Indian or Alaskan Native and Asian or Pacific Islander.

§In addition to data collected through the National Electronic Telecommunications System for Surveillance (NETSS), some data concerning ethnicity are collected on aggregate forms different from those used for numbers of reported cases. Thus, the total number of cases reported on this table can differ slightly from other tables. Cases were updated through the Division of Sexually Transmitted Diseases Prevention, NCHSTP, as of July 13, 1998. Data regarding ethnicity for 1997 are unavailable for chancroid.

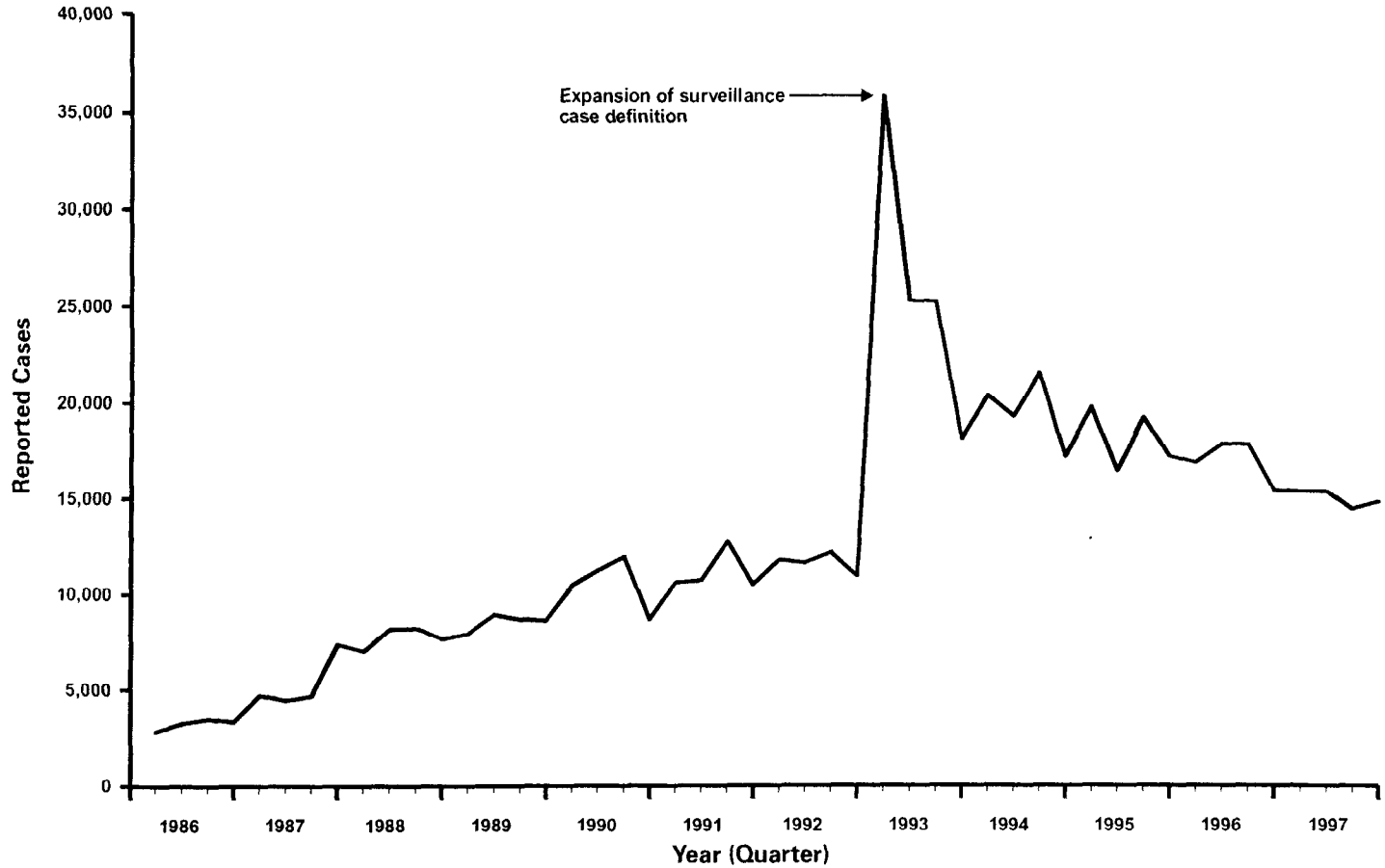
¶Cases were updated through the Division of Tuberculosis Elimination, NCHSTP, as of April 15, 1998.

ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) — reported cases per 100,000 population, United States and Puerto Rico, 1997



In 1997, the highest rates of reported AIDS cases per 100,000 population were in the northeastern, southeastern, and western states. Eighty-one percent (81%) of reported AIDS cases occurred among residents of large metropolitan areas (i.e., areas of ≥500,000 persons).

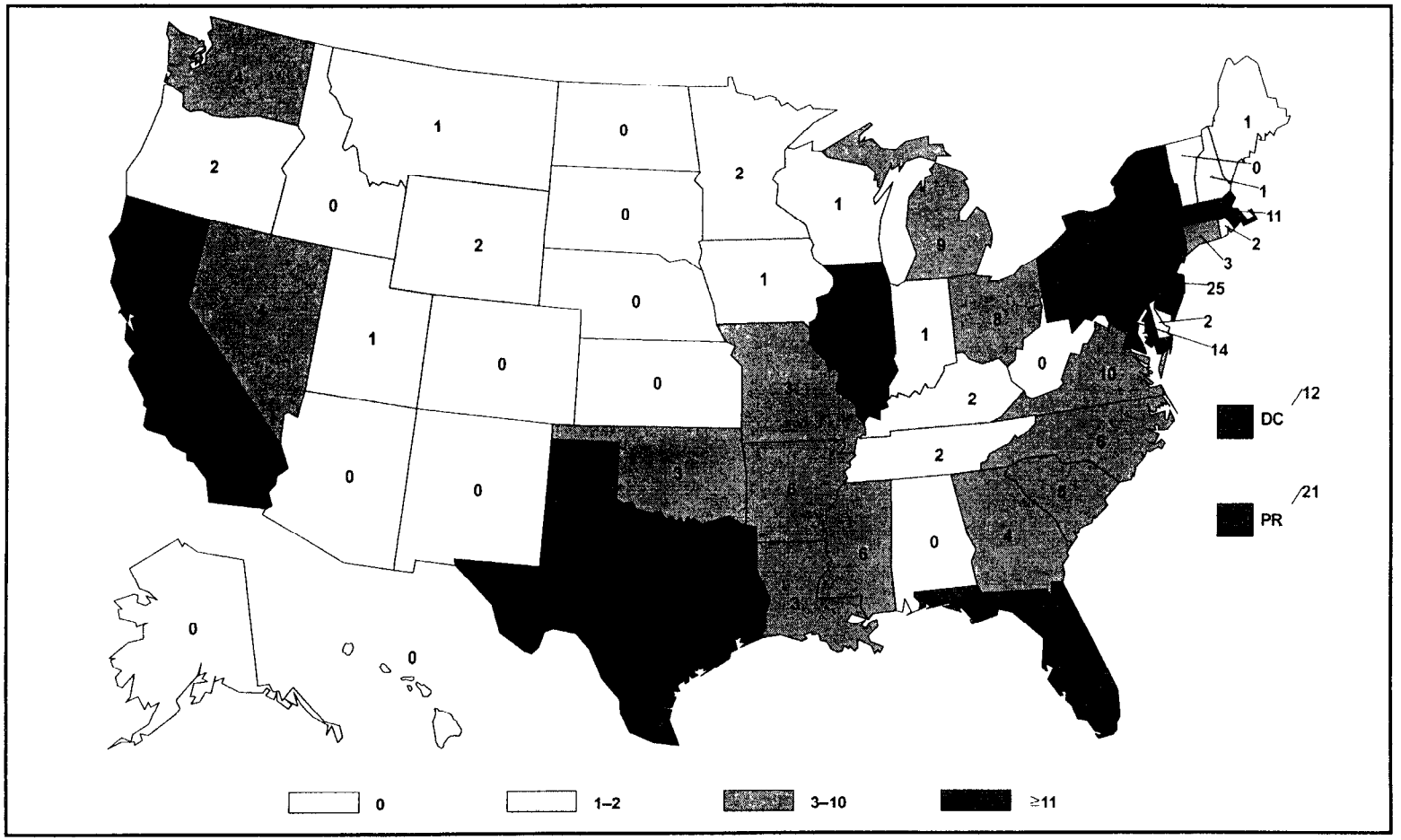
ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) — reported cases by quarter, United States,* 1986–1997



*Includes Guam, Puerto Rico, the U.S. Pacific Islands, and the U.S. Virgin Islands

The expansion of the AIDS surveillance case definition in 1993 resulted in a substantial increase in reported cases during that year. Since 1996, new treatments have slowed the progression from human immunodeficiency virus (HIV) infection to AIDS and from AIDS to death. Consequently, the number of new AIDS cases is declining, and the number of persons living with HIV infection and AIDS is increasing.

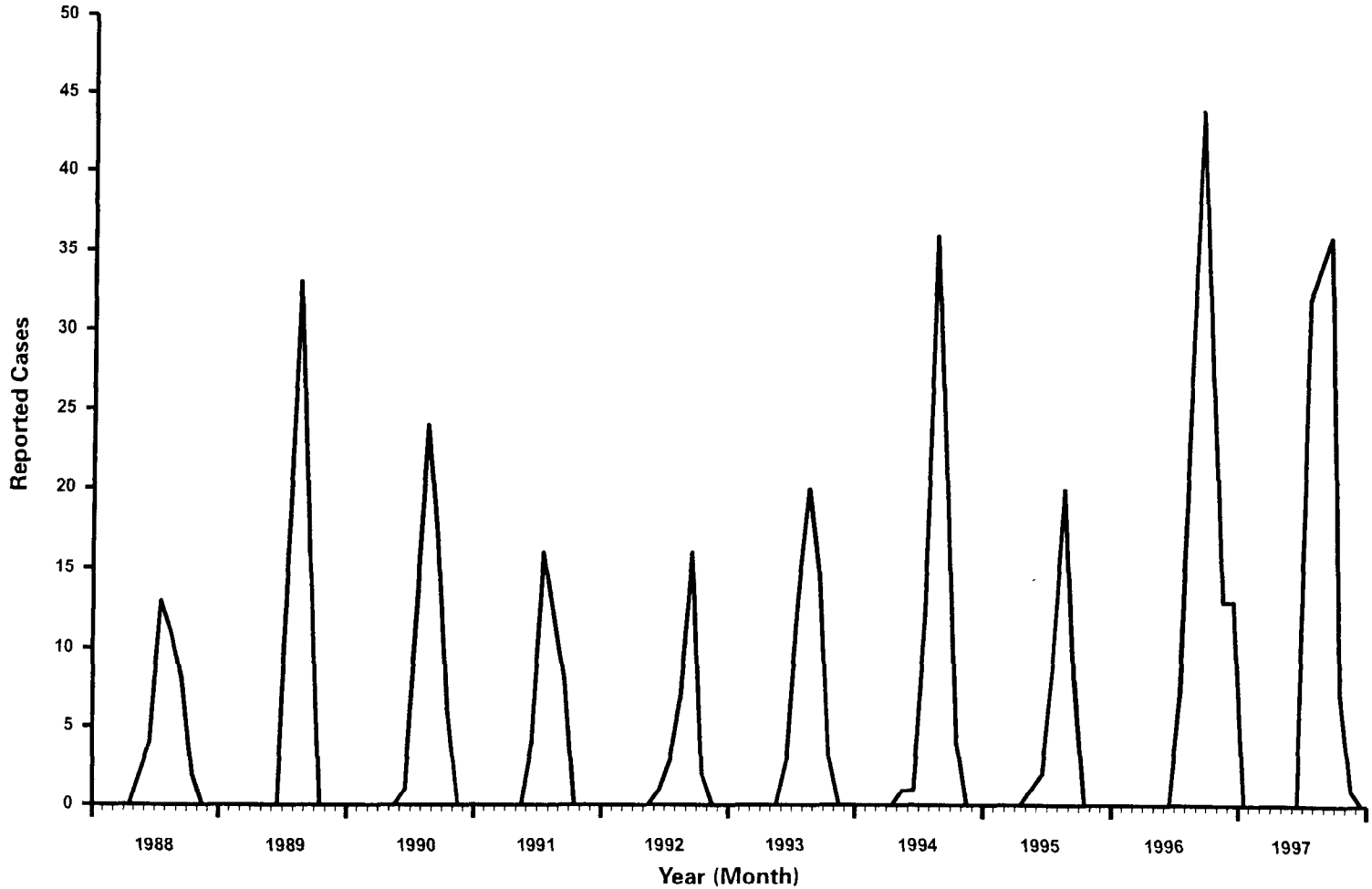
ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS) — reported pediatric cases,* United States and Puerto Rico, 1997



*Children and adolescents aged <13 years.

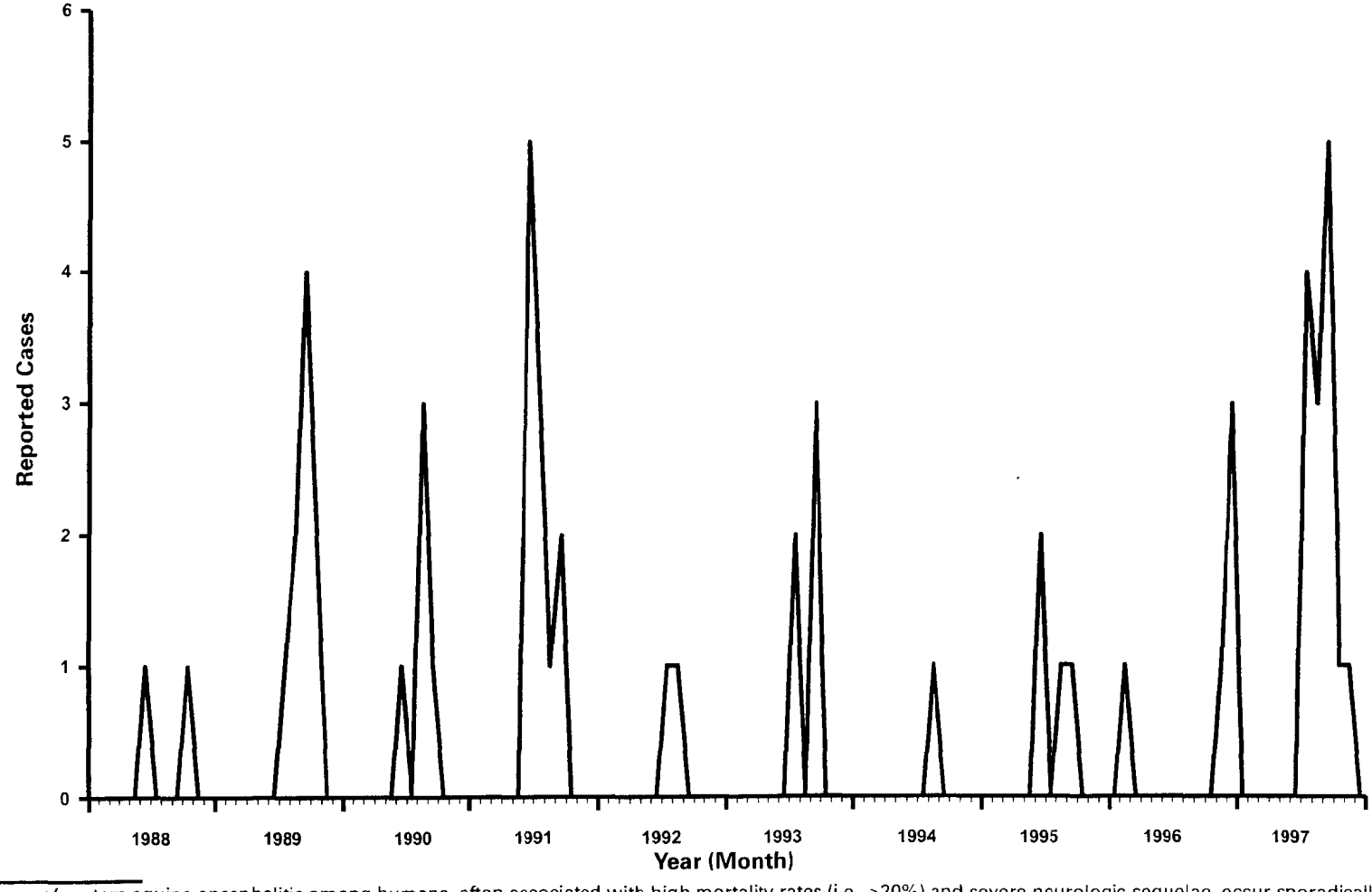
Trends in AIDS incidence among children continued to demonstrate the dramatic success of efforts to reduce perinatal (i.e., mother-to-child) human immunodeficiency virus (HIV) transmission. From 1992 through 1996, the number of perinatally acquired cases declined 43%. Despite these declines, new perinatally acquired AIDS cases continue to occur among very young children who are disproportionally from racial and ethnic minority populations. Intensified efforts are needed to prevent HIV infection among women and to provide early prenatal care and treatment to HIV-infected women.

ARBOVIRAL INFECTIONS (of the central nervous system) — reported laboratory-confirmed cases caused by California serogroup viruses, by month of onset, United States, 1988–1997



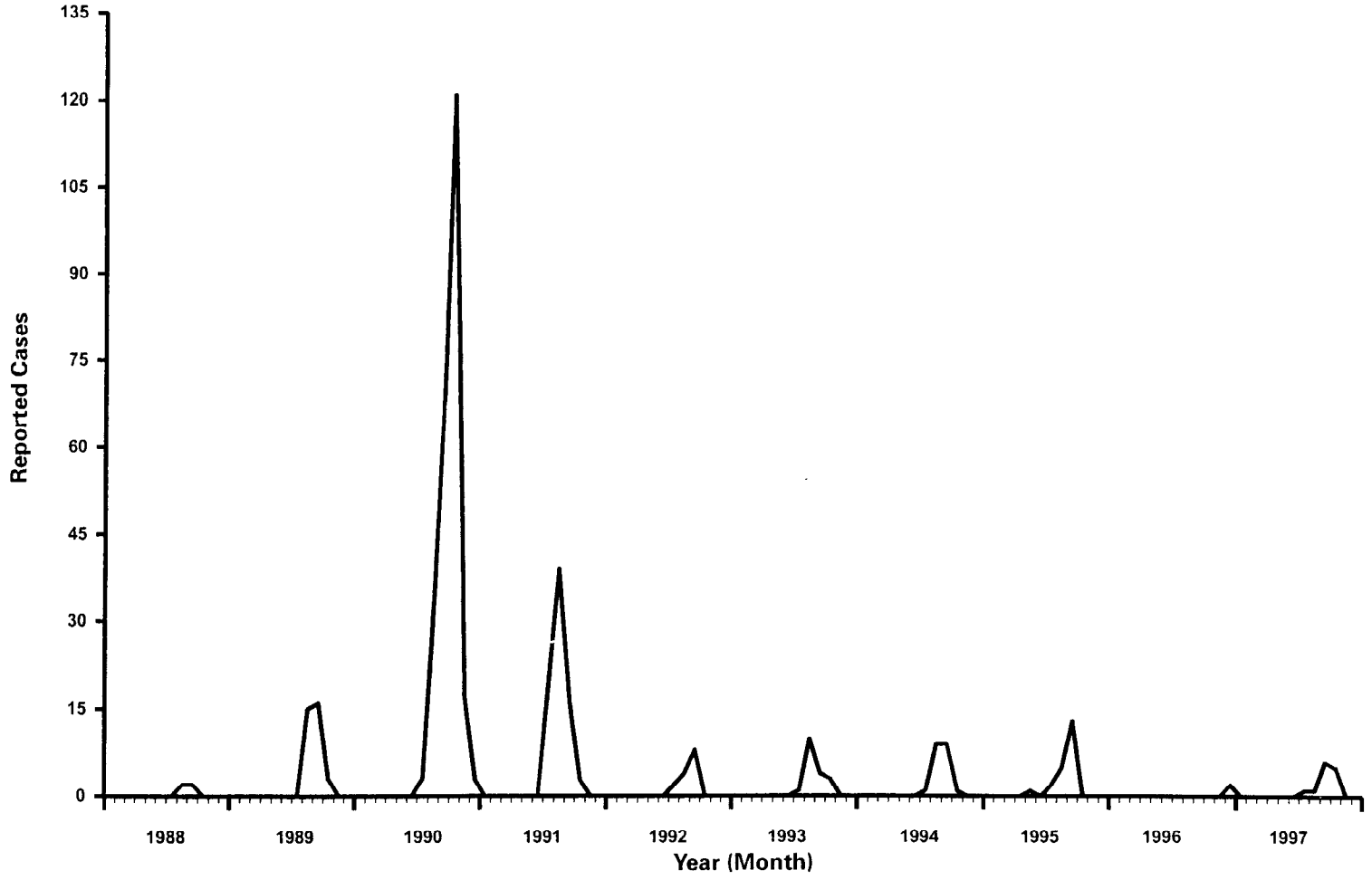
California serogroup viruses (mainly LaCrosse virus in the eastern United States) are an endemic cause of encephalitis, especially in children. The 1997 national total of 127 reported LaCrosse encephalitis cases is the fourth largest yearly total reported since 1964.

ARBOVIRAL INFECTIONS (of the central nervous system) — reported laboratory-confirmed cases caused by eastern equine encephalitis virus, by month of onset, United States, 1988–1997



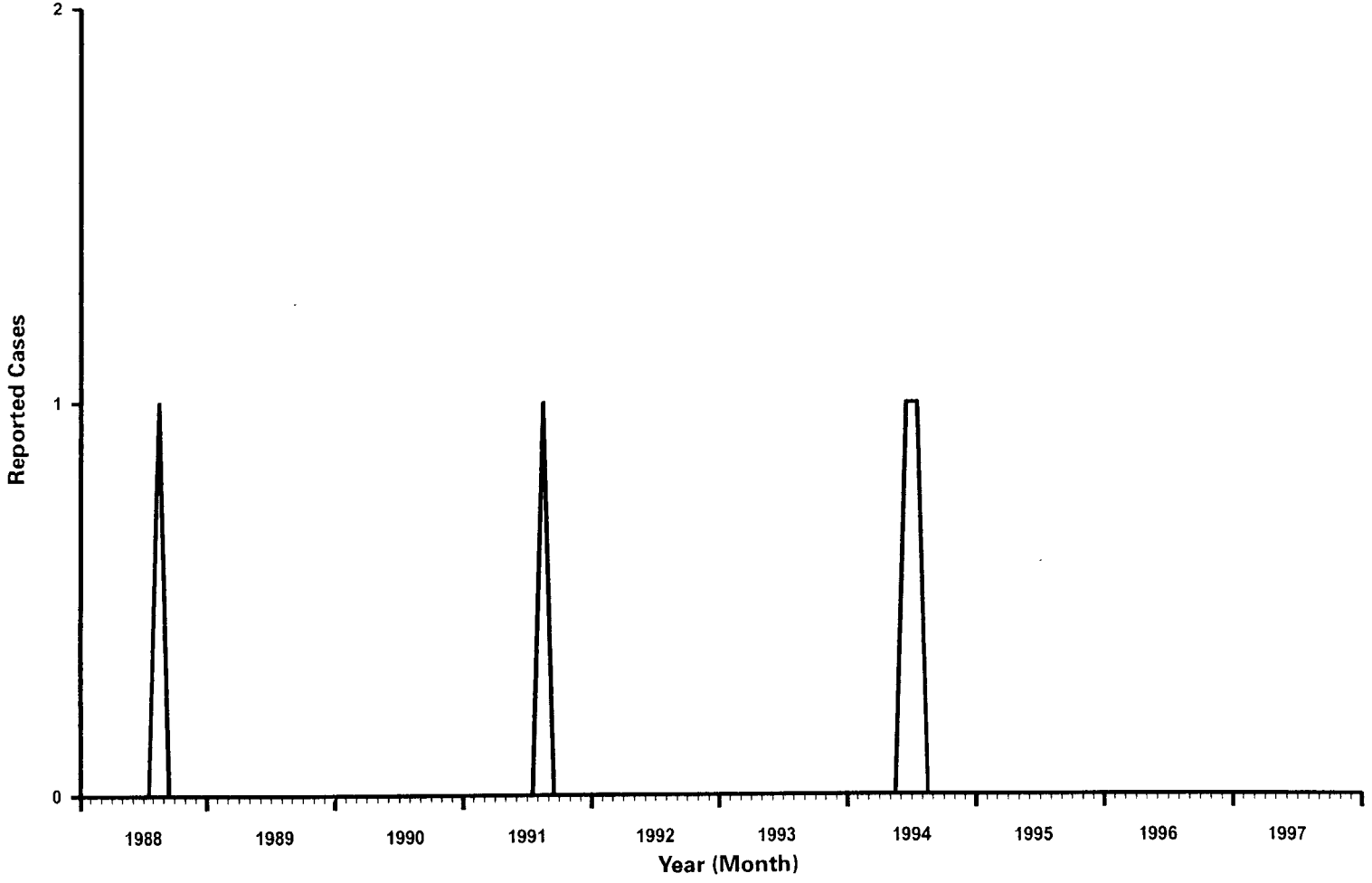
Cases of eastern equine encephalitis among humans, often associated with high mortality rates (i.e., >20%) and severe neurologic sequelae, occur sporadically in the eastern United States. The 1997 national total of 14 cases is the largest yearly total reported since 1983.

ARBOVIRAL INFECTIONS (of the central nervous system) — reported laboratory-confirmed cases caused by St. Louis encephalitis virus, by month of onset, United States, 1988–1997



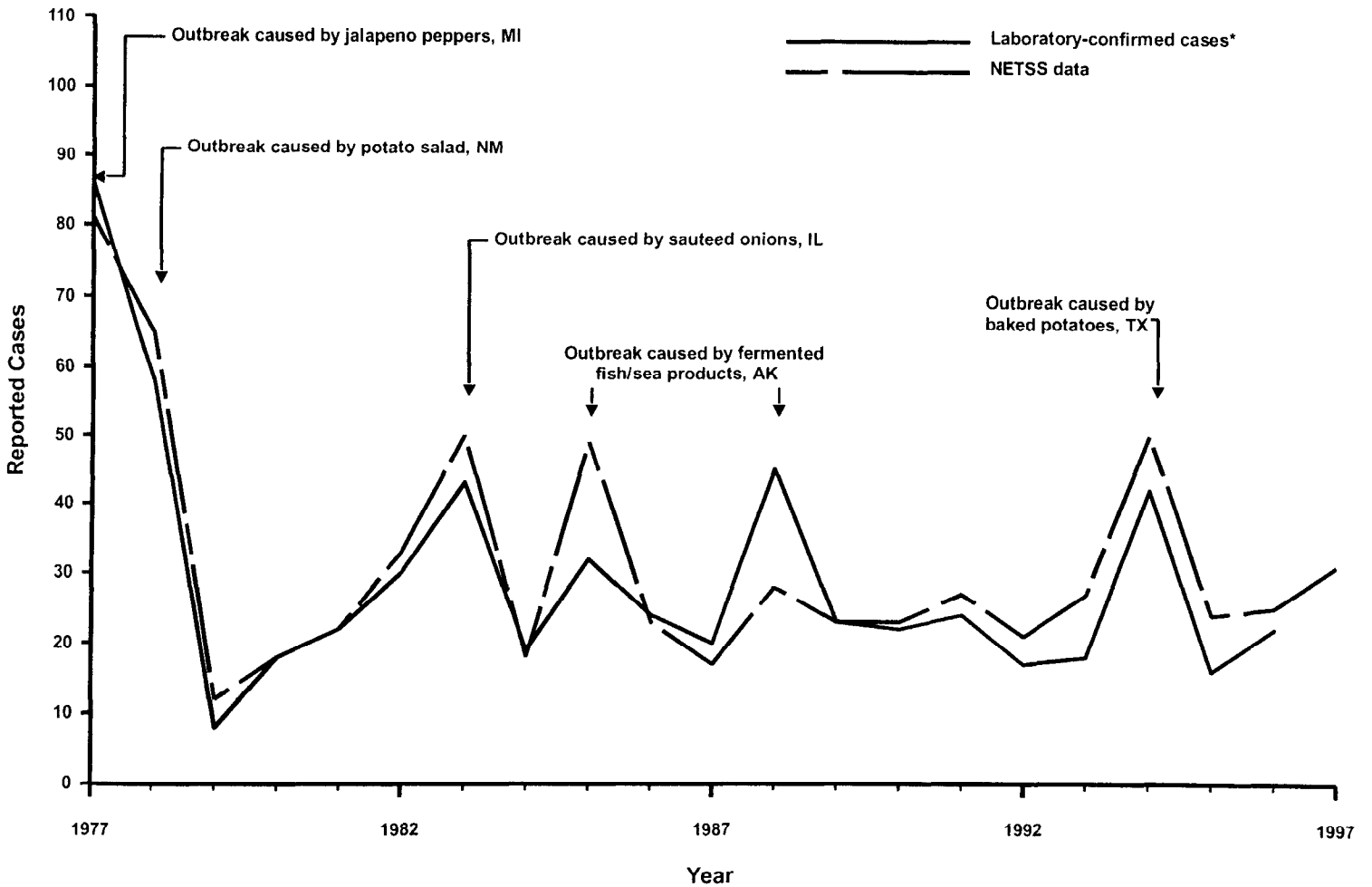
St. Louis encephalitis virus continues to be the primary cause of epidemic viral encephalitis in the United States. The most recent major epidemic occurred in Florida in 1990.

ARBOVIRAL INFECTIONS (of the central nervous system) — reported laboratory-confirmed cases caused by western equine encephalitis virus, by month of onset, United States, 1988–1997



The most recent epidemic of western equine encephalitis occurred in 1987 in Colorado, where 30 cases were reported.

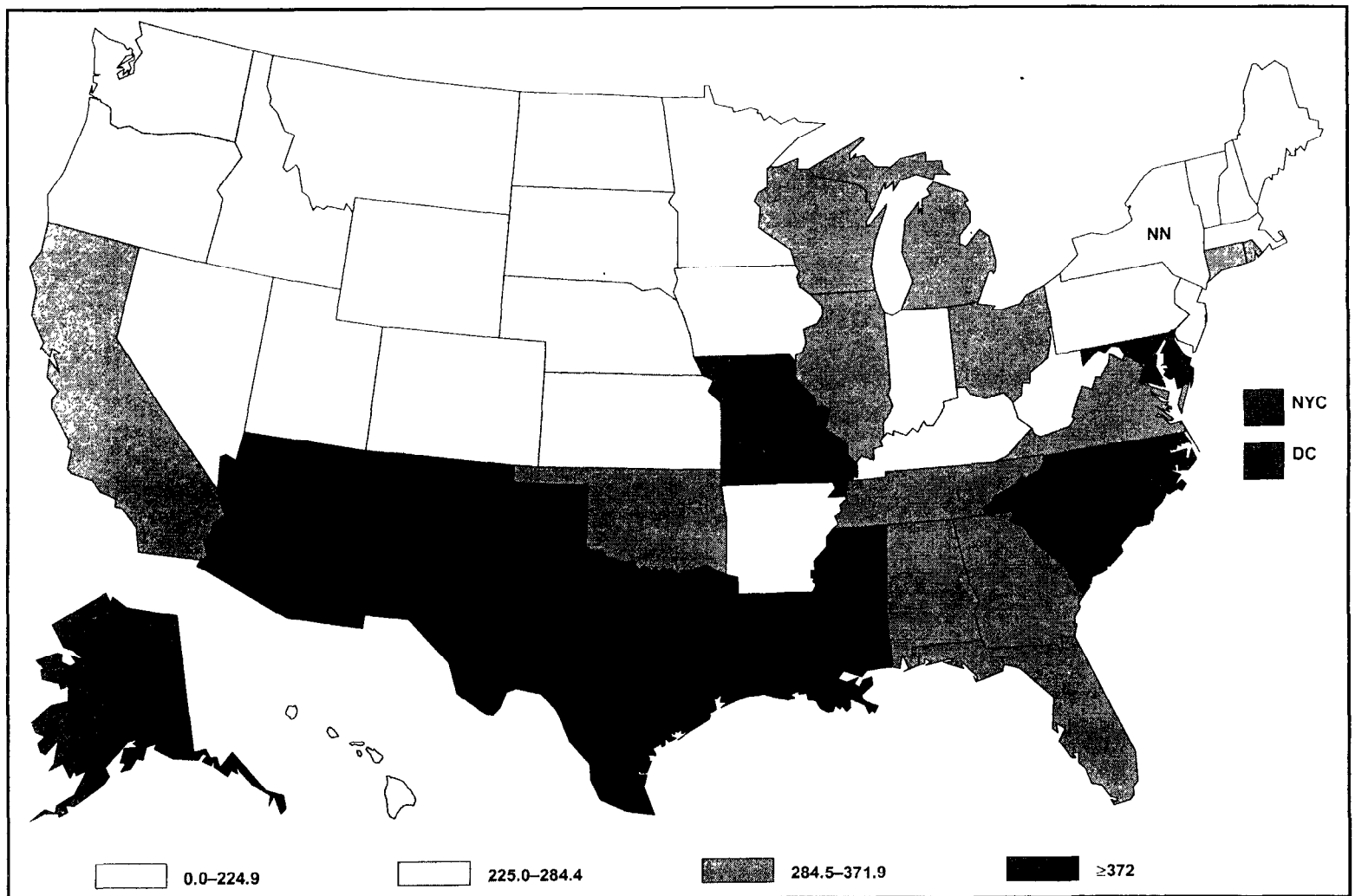
BOTULISM (foodborne) — by year, United States, 1977–1997



* Data from annual survey of state epidemiologists and directors of state public health laboratories Data are not yet available for 1997

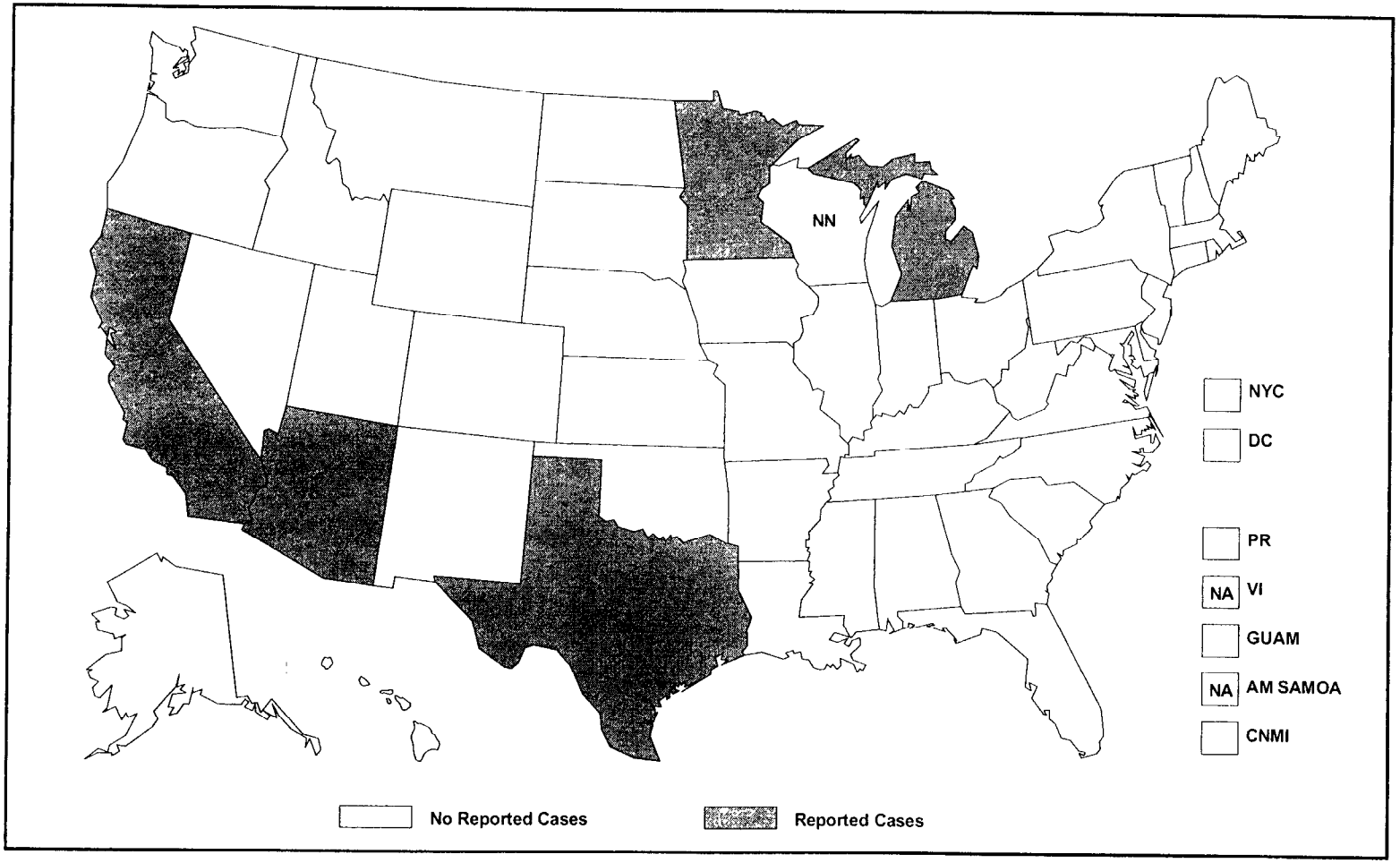
Although they occur infrequently, outbreaks of foodborne botulism can rapidly kill many affected persons. Such outbreaks require prompt and effective communication between clinicians and public health officials.

CHLAMYDIA — reported cases among women per 100,000 population, United States, 1997



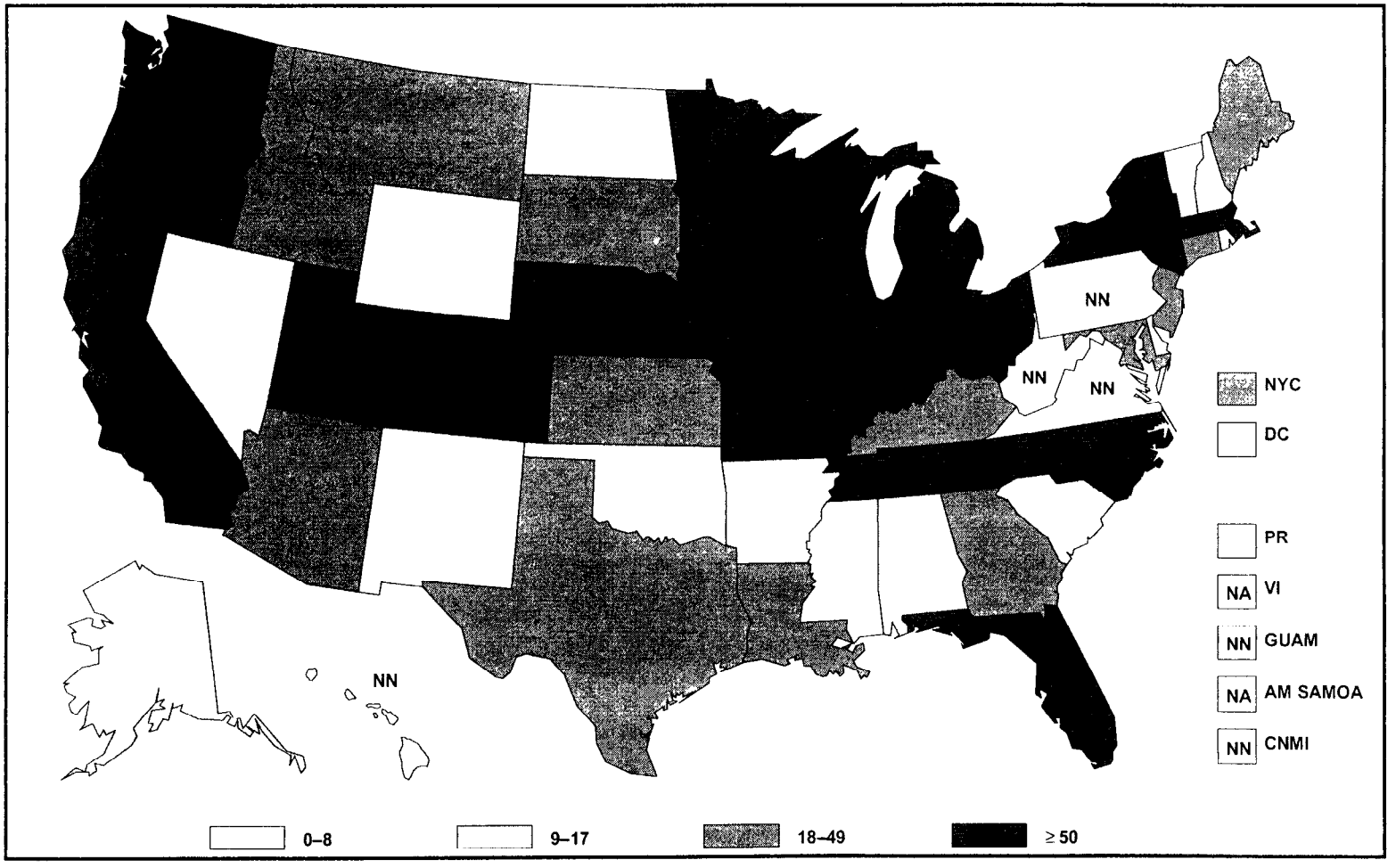
In 1997, the chlamydia rate among women was 322.1 cases per 100,000 population. The rates for men are not presented because reporting for men is more limited than it is for women.

CHOLERA — reported cases, United States and territories, 1997



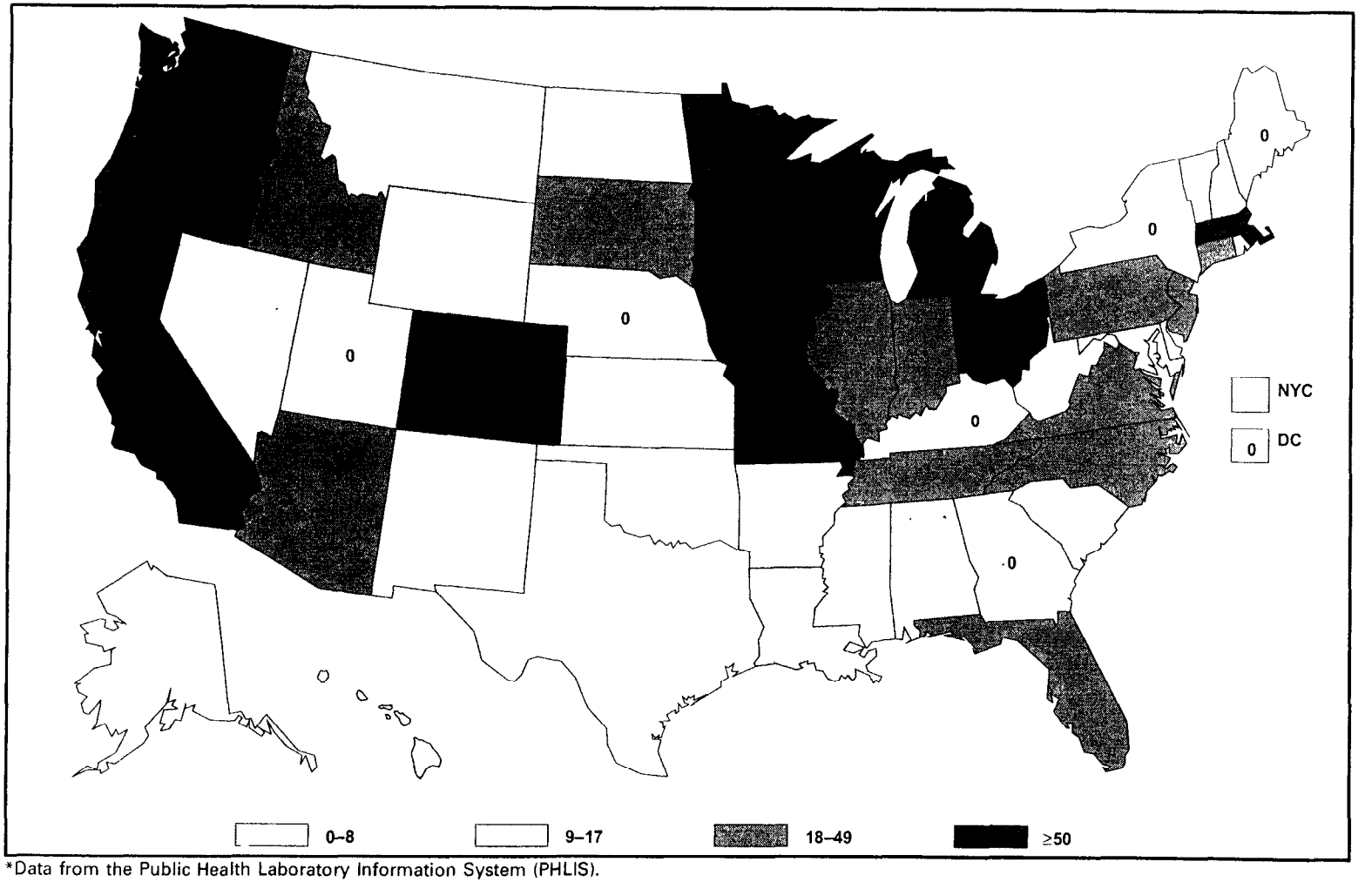
In recent years, cholera has been primarily a disease of travelers to Latin America, Asia, and Africa, although cases are occasionally acquired from contaminated food in the United States.

ESCHERICHIA COLI O157:H7 — reported cases, United States and territories, 1997



The number of states in which *E. coli* O157:H7 infection is a notifiable disease increased from 44 in 1996 to 46 in 1997. However, because <60% of clinical laboratories routinely test all stools — or even all bloody stools — for *E. coli* O157:H7, many infections are not recognized or reported.

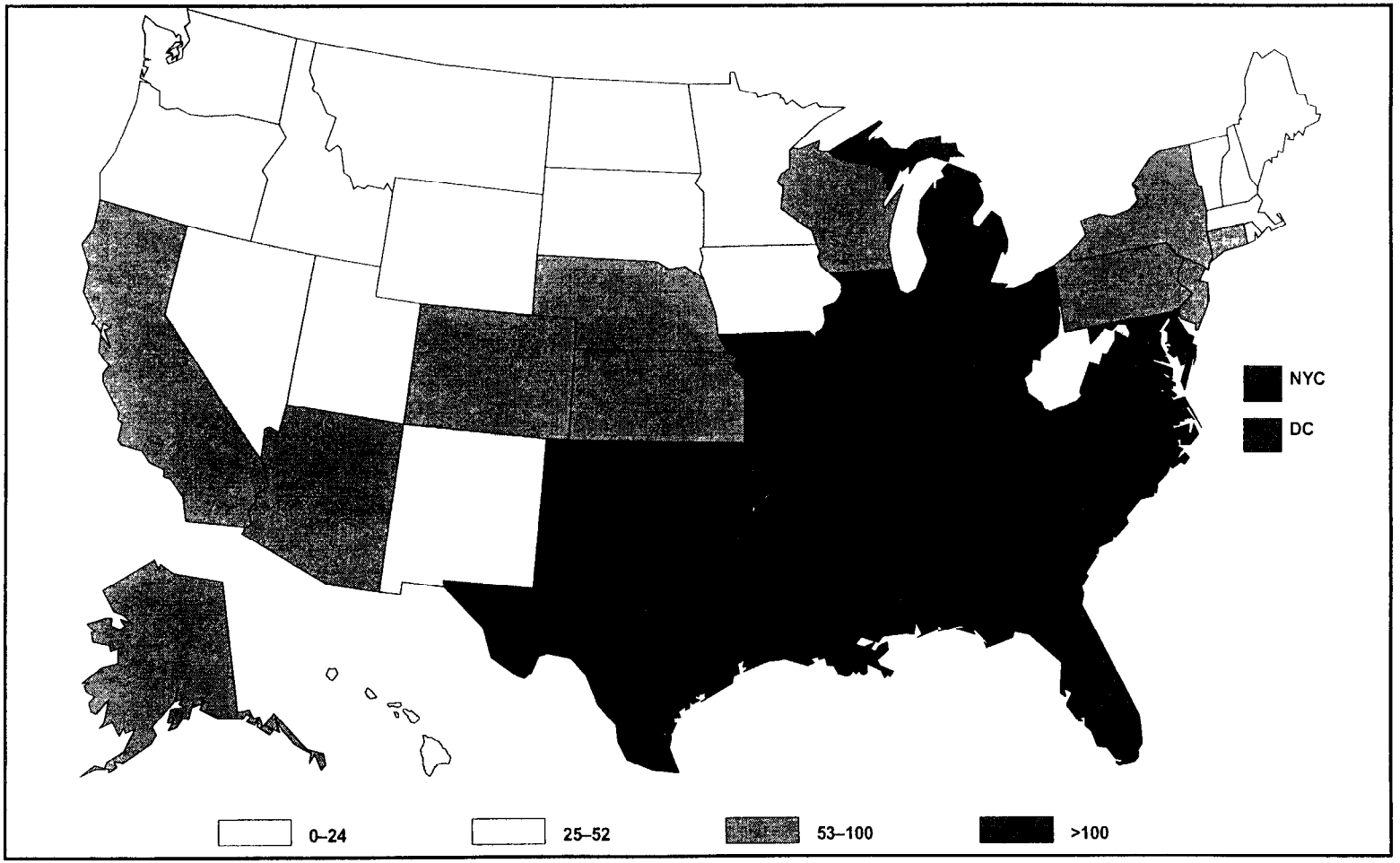
ESCHERICHIA COLI O157:H7 — reported isolates,* United States, 1997



*Data from the Public Health Laboratory Information System (PHLIS).

Only *E. coli* O157:H7 isolates that are confirmed by a state public health laboratory are reported to PHLIS. Many public health laboratories are now able to subtype isolates using pulsed-field gel electrophoresis, a procedure that facilitates comparison of strains among states.

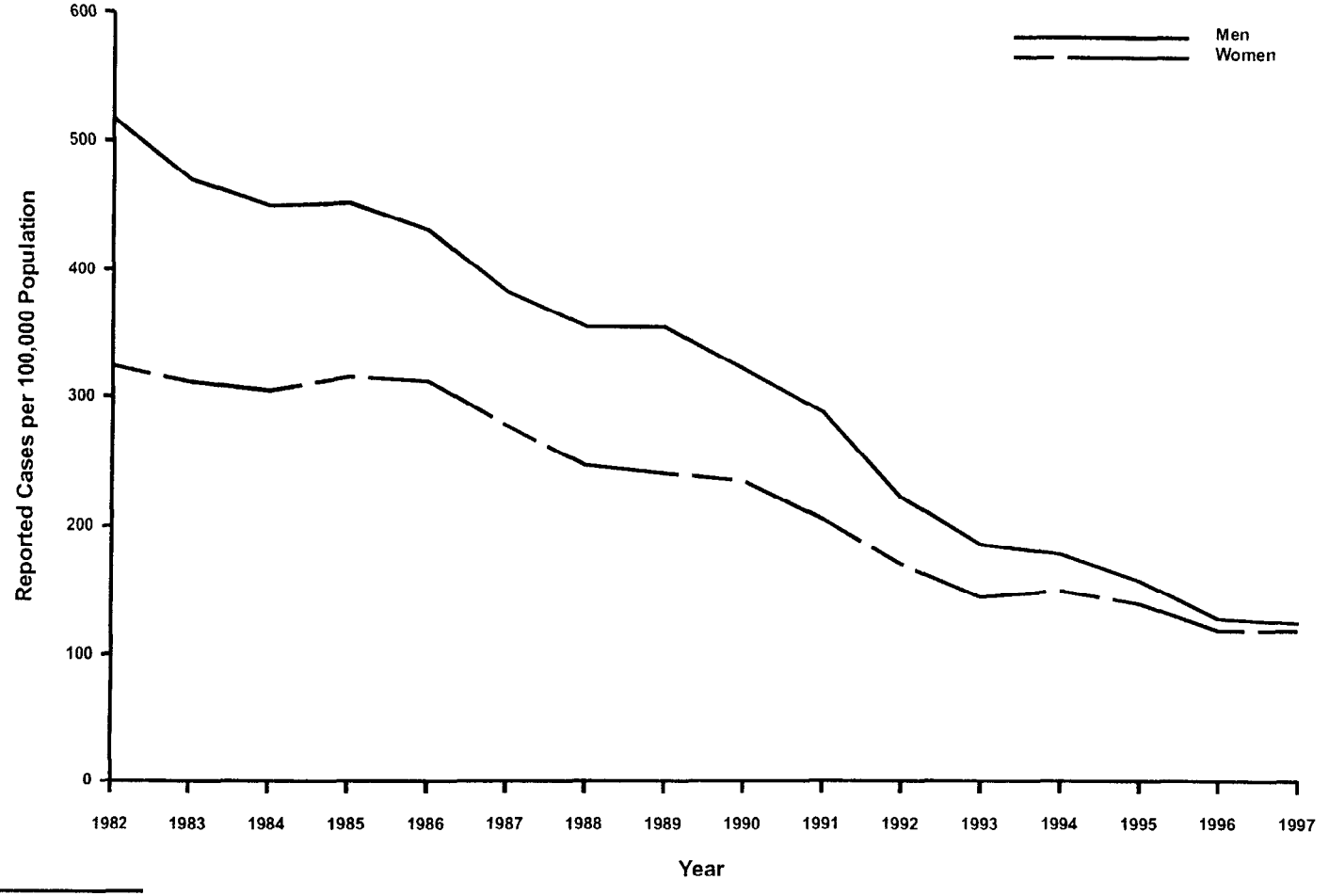
GONORRHEA — reported cases per 100,000 population, United States, 1997



NOTE The revised *Healthy People 2000* objective is ≤100 per 100,000 population.

The overall U.S. rate of gonorrhea in 1997 was 121.4 per 100,000 population; 30 states reported gonorrhea rates below the revised *Healthy People 2000* national objective.

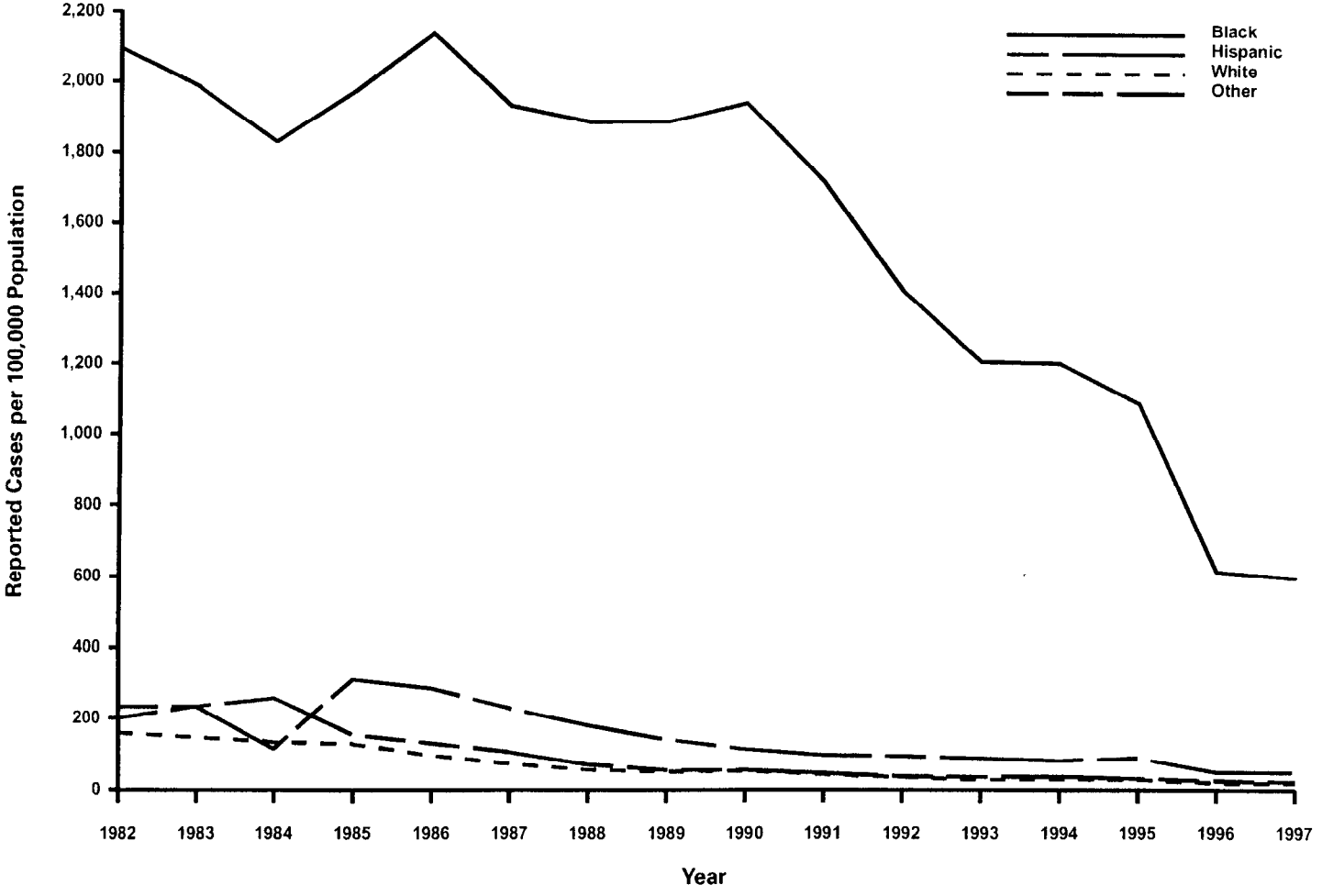
GONORRHEA — by sex, United States, 1982–1997



In 1997, the overall reported rate of gonorrhea in the United States was 121.4 per 100,000 population, similar to the rate of 122.8 in 1996. Among men, the rate decreased slightly from 128.5 per 100,000 population in 1996 to 125.4 in 1997. Among women, the rate increased slightly from 118.3 per 100,000 population in 1996 to 119.3 in 1997.*

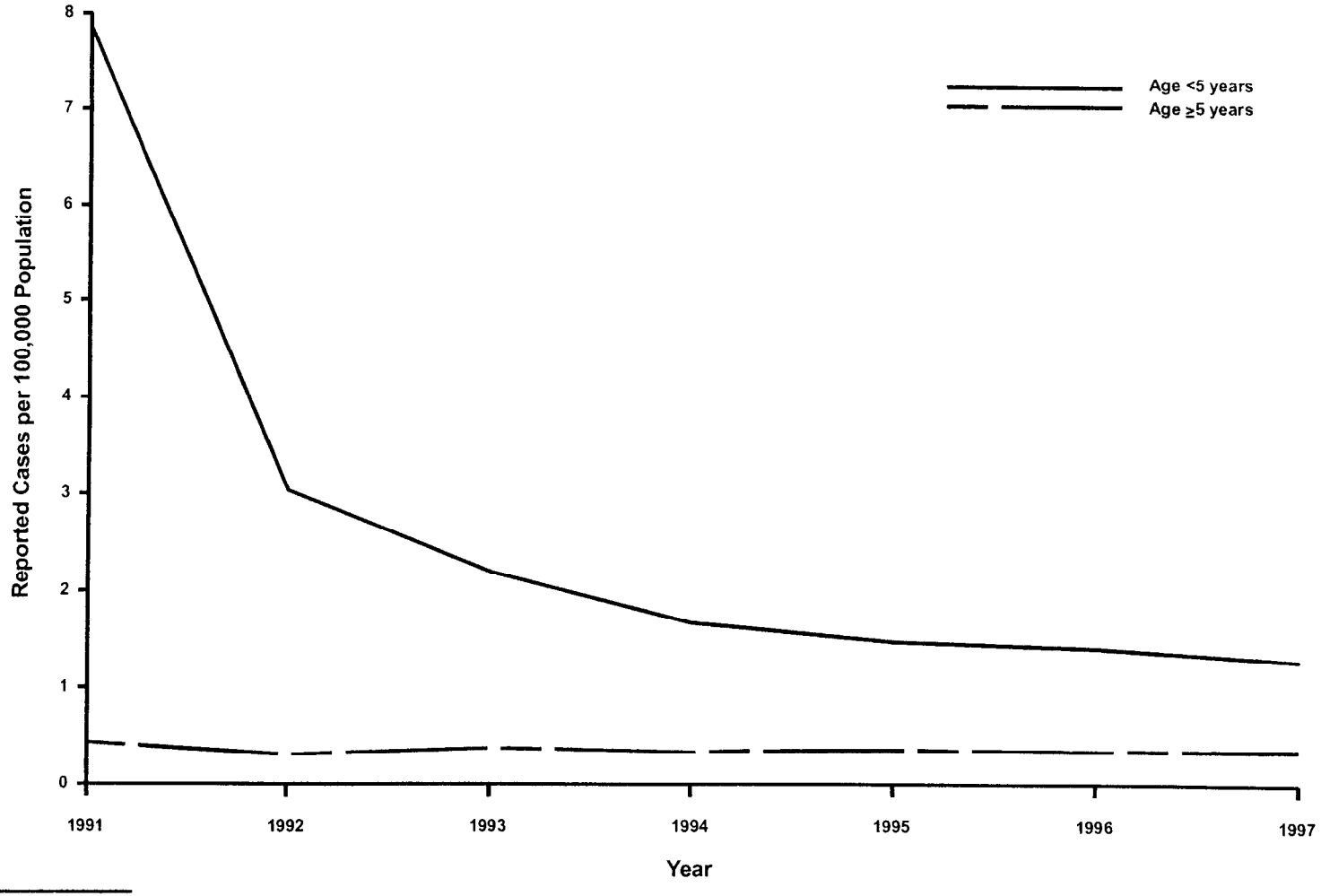
*Data source: Division of Sexually Transmitted Diseases Prevention, National Center for HIV, STD, and TB Prevention.

GONORRHEA — by race and ethnicity, United States, 1982–1997



In 1997, gonorrhea rates decreased or remained the same among all racial and ethnic groups. The only exception occurred among Asian/Pacific Islanders (included in the "other" race and ethnicity category).

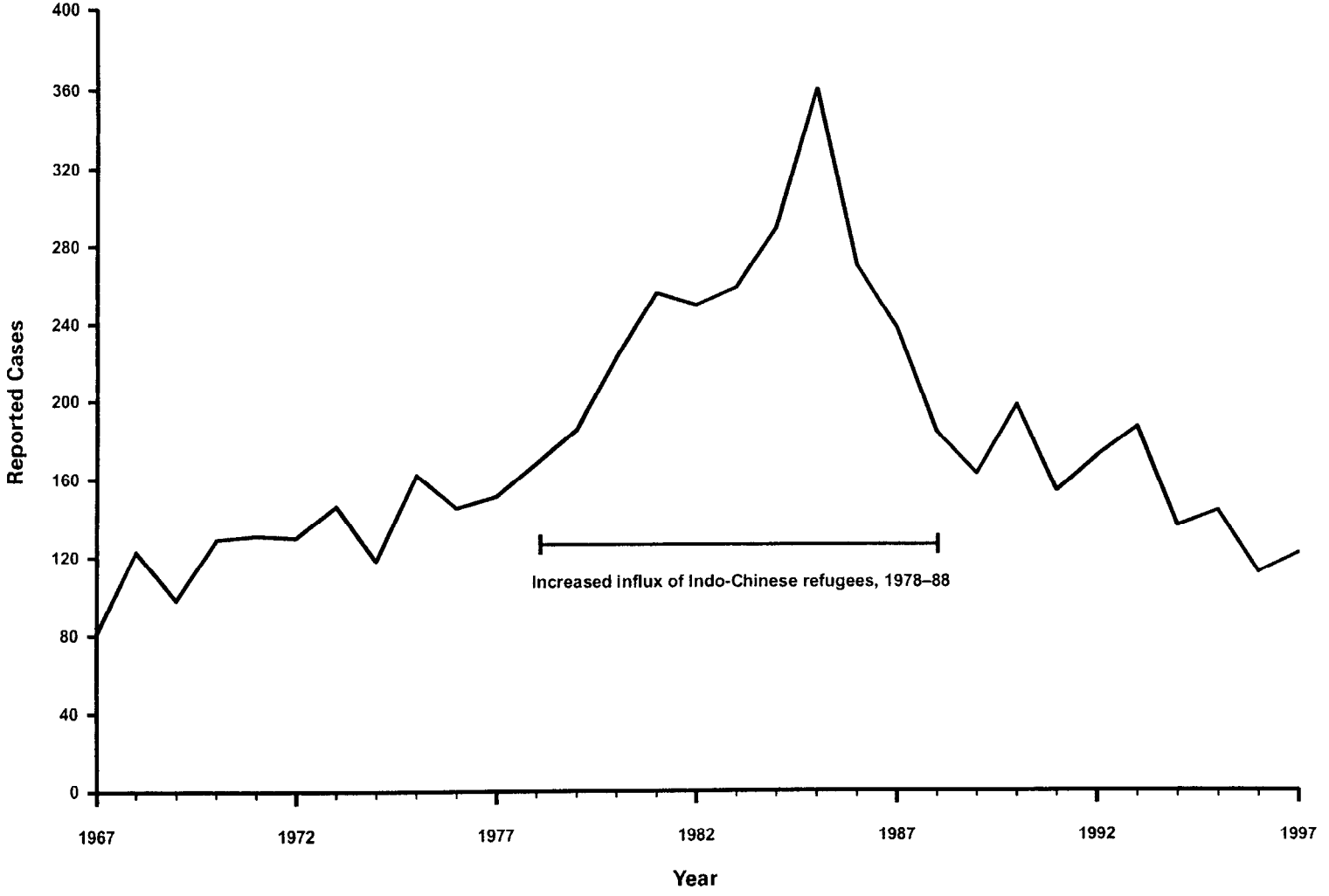
HAEMOPHILUS INFLUENZAE (Invasive Disease) — by age group, United States, 1991–1997



Before the introduction of the *Haemophilus influenzae* type b (Hib) vaccine in December 1987, the incidence of Hib invasive disease among children aged <5 years was estimated to be 60–110 per 100,000 population. In 1997, 260* cases of all serotypes of *H. influenzae* invasive disease among children aged <5 years were reported (incidence: 1.3 per 100,000 children); 82 (32%) cases were attributable to Hib (incidence: 0.4 per 100,000 children).

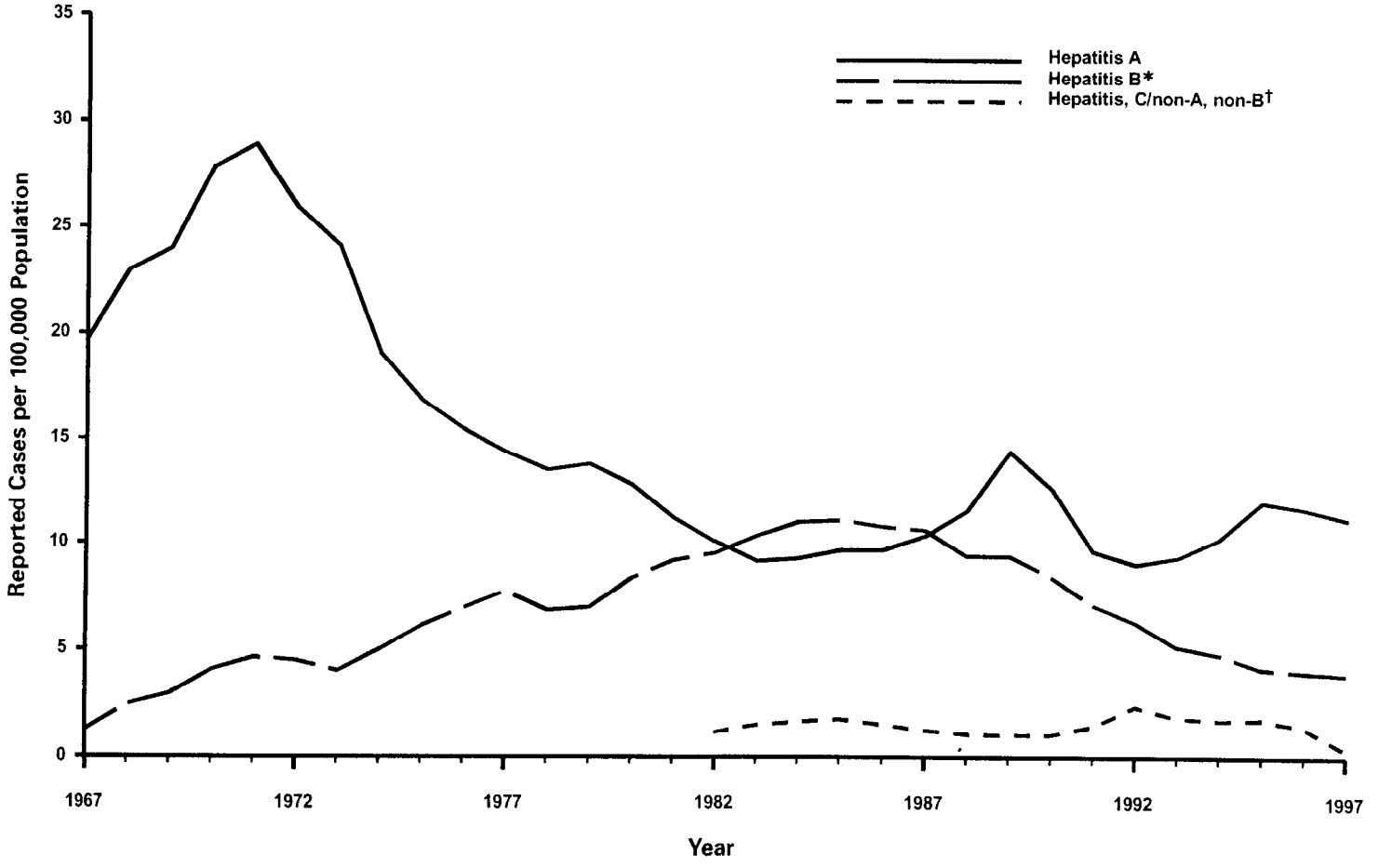
* Data source: National Immunization Program by date of onset.

HANSEN DISEASE (Leprosy) — by year, United States, 1967–1997



In 1997, a total of 122 cases of Hansen disease were reported in the United States. The number of cases peaked at 361 in 1985; since 1988, the number has remained relatively stable.

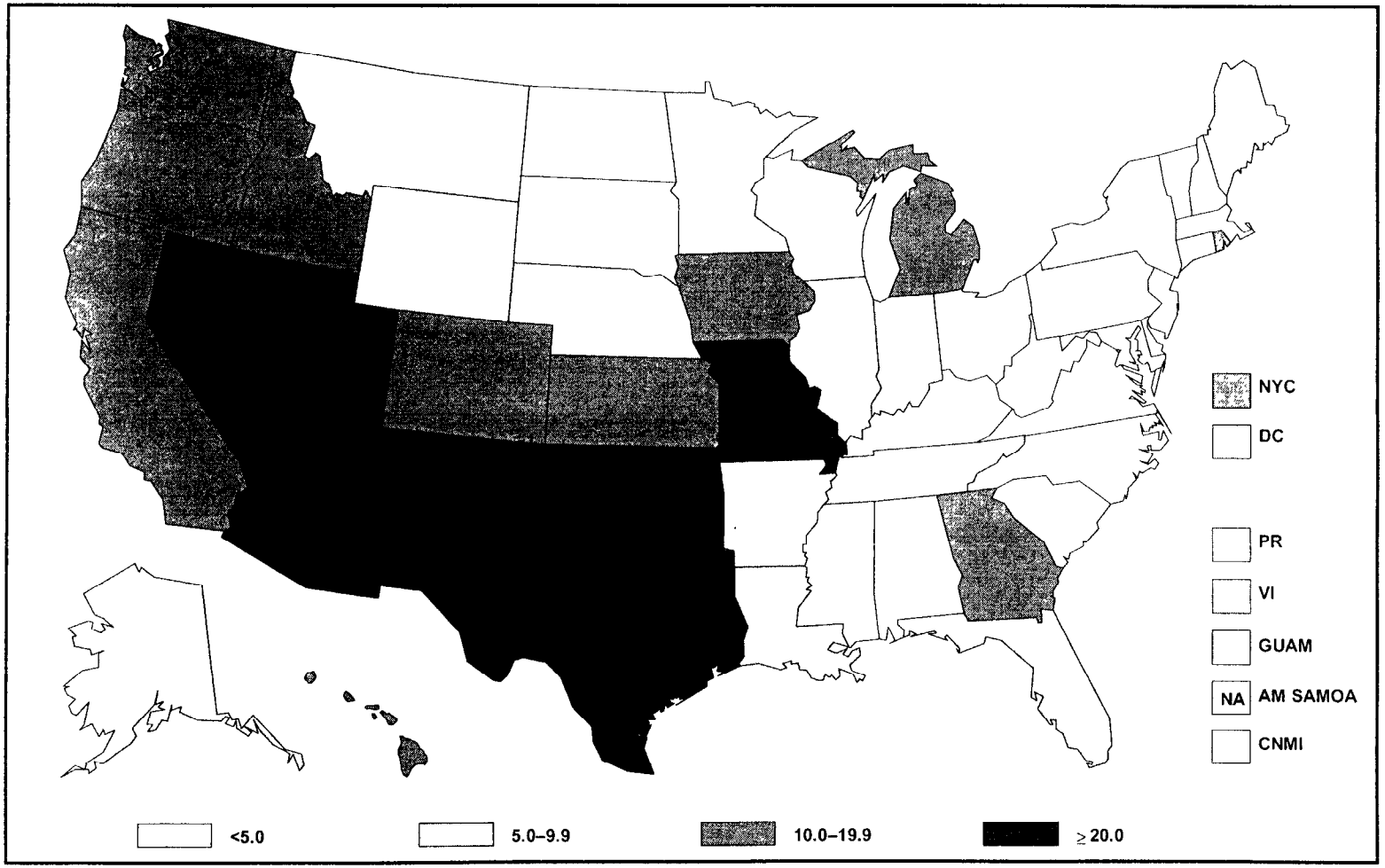
HEPATITIS — by year, United States, 1967–1997



*The first hepatitis B vaccine was licensed in June 1982.
†Anti-HCV antibody test was available as of May 1990.

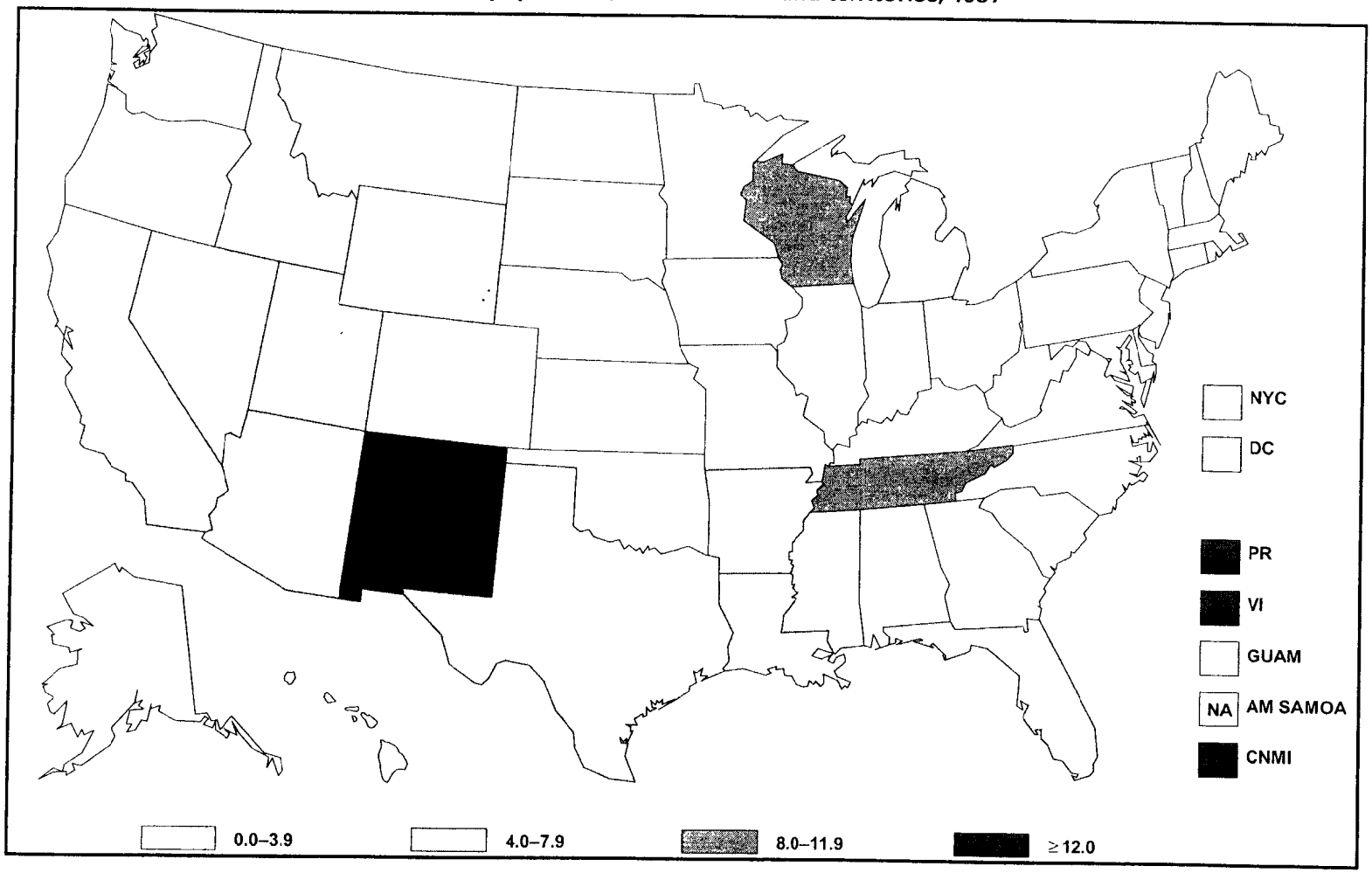
Hepatitis C/non-A, non-B is the most underreported type of viral hepatitis. Nonetheless, the increase observed in this type of hepatitis after 1990 is misleading because, in some states, reported cases have included those among persons identified in routine screening programs who were positive for antibody to hepatitis C virus but who did not have evidence of acute hepatitis.

HEPATITIS A — reported cases per 100,000 population, United States and territories, 1997



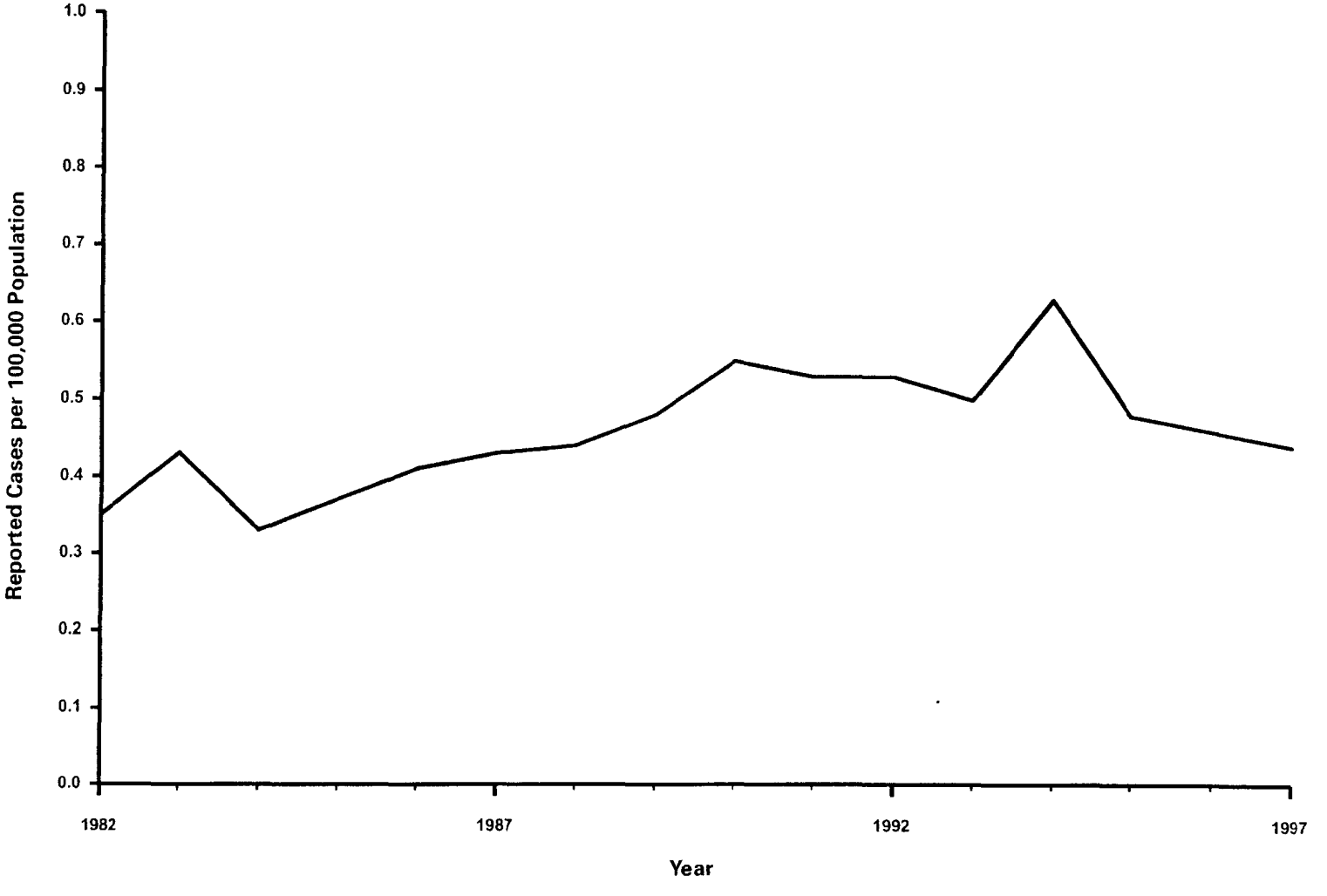
After reaching a rate of 12.1 cases per 100,000 population in 1995, the incidence of hepatitis A has declined slightly. In 1997, the rate of hepatitis A in the western United States was more than 2.5 times the average rate in other regions.

HEPATITIS B — reported cases per 100,000 population, United States and territories, 1997



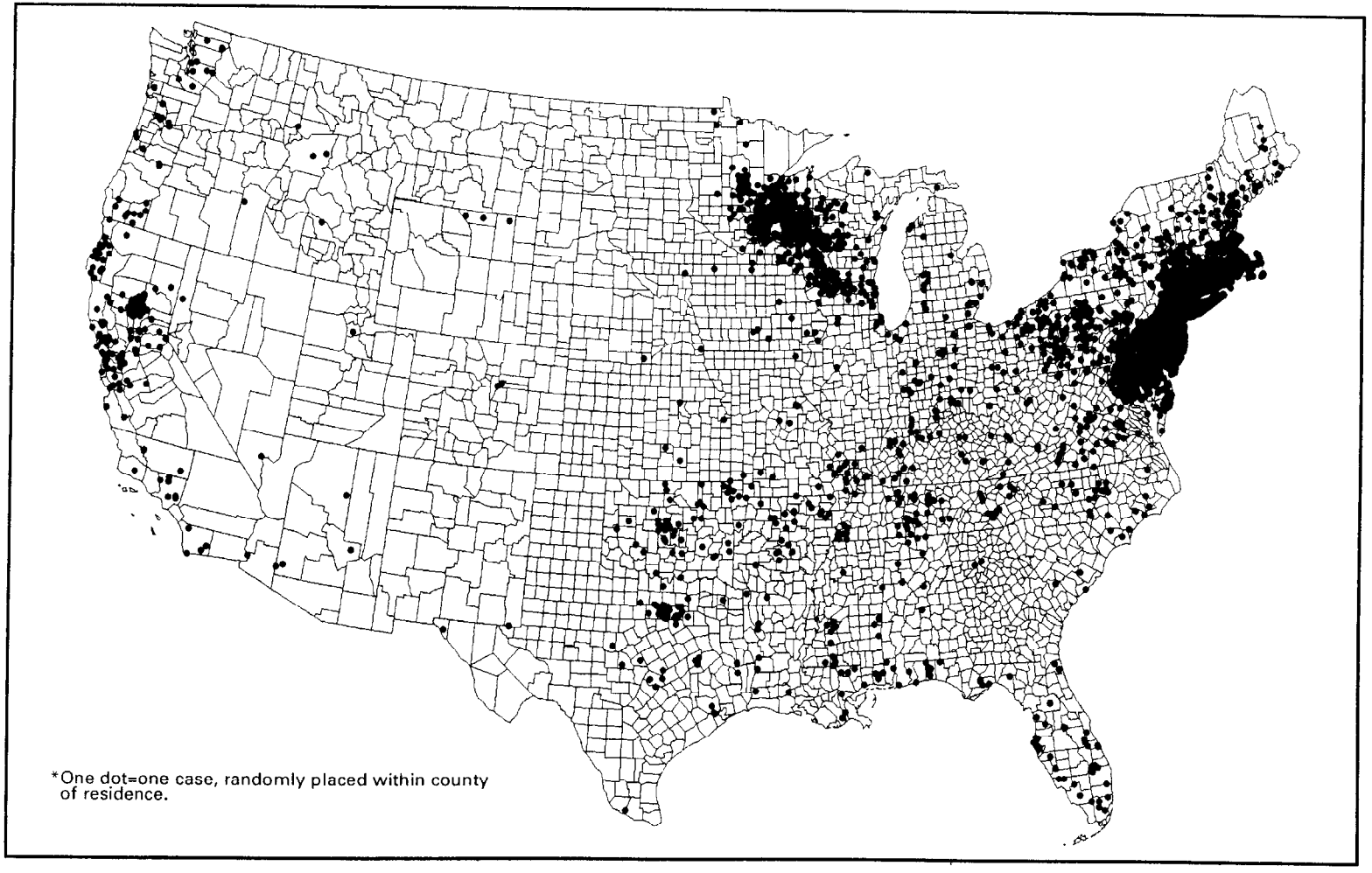
Hepatitis B continues to decline in most states, primarily because of a decrease in the number of cases among injecting-drug users and, to a lesser extent, because of a decline in cases associated with both male homosexual practices and heterosexual practices.

LEGIONELLOSIS — by year, United States, 1982–1997



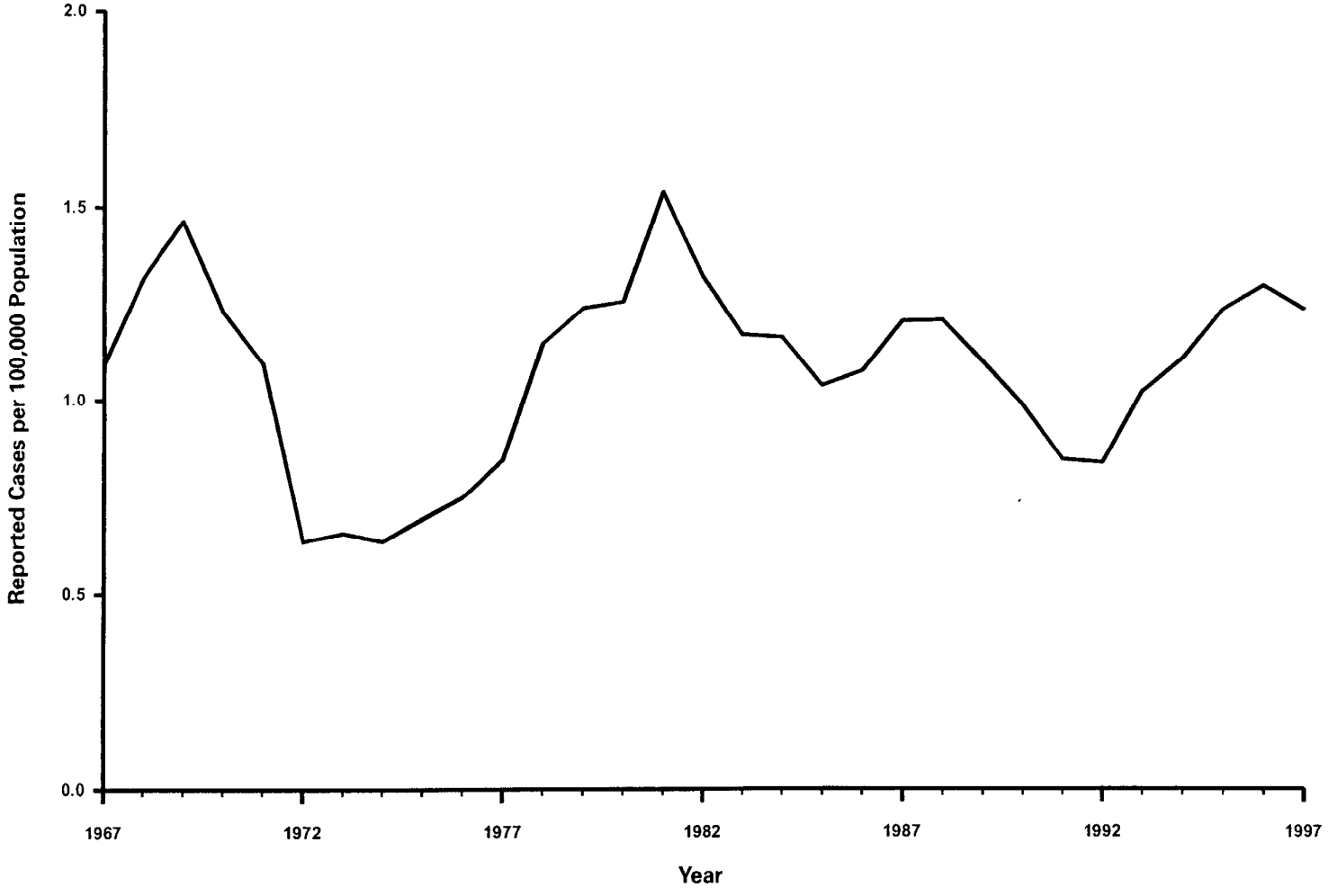
In 1997, the overall reported rate of legionellosis in the United States was 0.44 per 100,000 population. However, data from prospective, population-based studies of persons with pneumonia indicate that the actual rate of legionellosis is more than 10-fold this number.

LYME DISEASE — reported cases*, United States, 1997



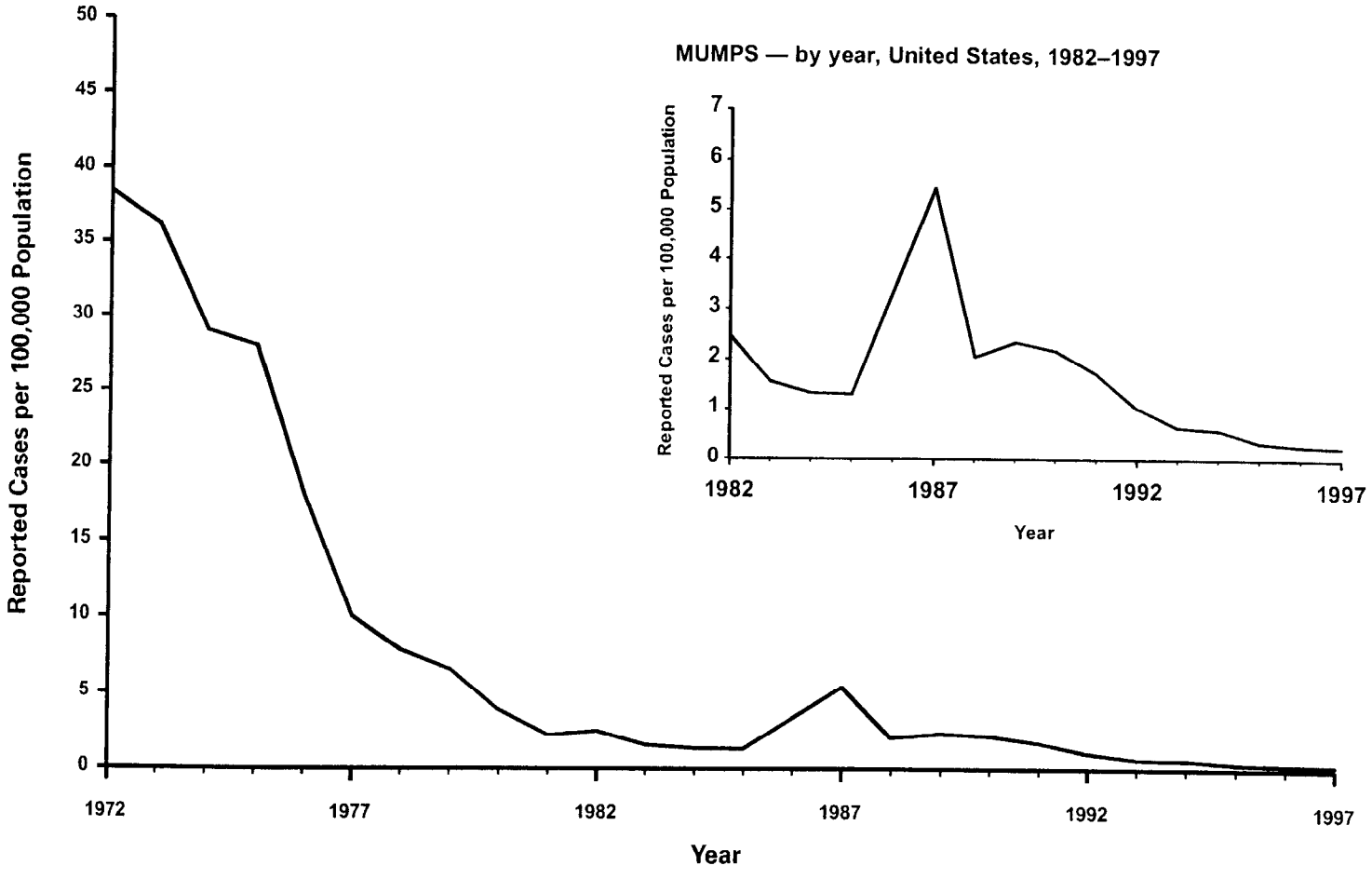
In 1997, a total of 12,801 cases of Lyme disease were reported by 46 states and the District of Columbia. The 10 states with the highest incidence of Lyme disease cases per 100,000 population were Connecticut, Rhode Island, New Jersey, New York, Pennsylvania, Delaware, Massachusetts, Wisconsin, Minnesota, and Maryland. These states accounted for 92% of the reported Lyme disease cases in 1997.

MENINGOCOCCAL DISEASE — by year, United States, 1967–1997



The overall rate of meningococcal disease remained constant over the past year. The proportion of cases in which the serogroup was reported increased from 19% in 1996 to 31% in 1997. Serogroup Y continues to cause disease in the United States. In 1997, serogroup Y accounted for 29% of cases in which the serogroup was reported. Most other cases were caused by serogroup B (32%) and serogroup C (31%).

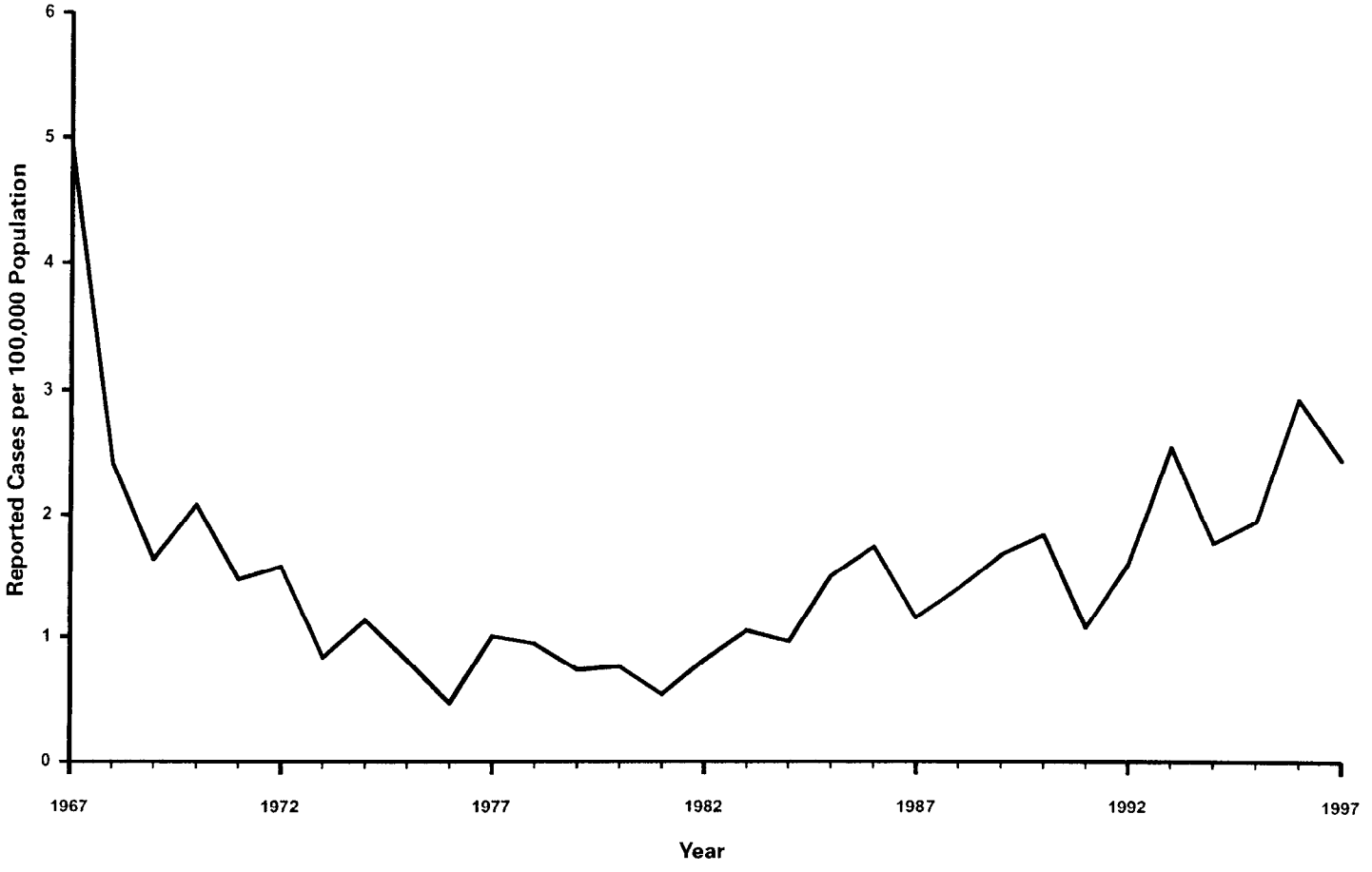
MUMPS — by year, United States, 1972–1997



NOTE: Mumps vaccine was licensed in December 1967.

Since 1990, the incidence of mumps has decreased steadily.

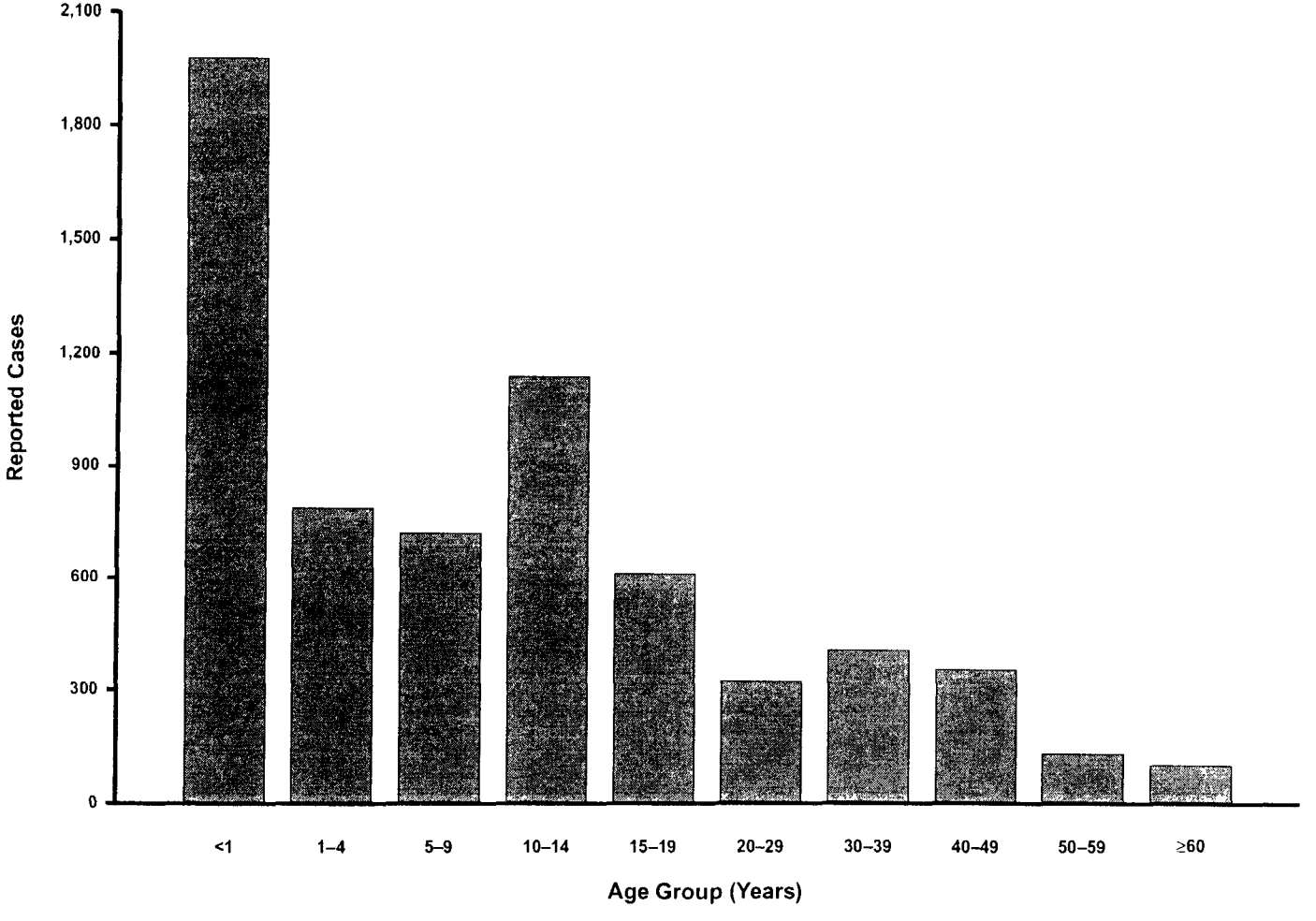
PERTUSSIS (Whooping Cough) — by year, United States, 1967–1997



NOTE: DTP vaccine was licensed in 1949.

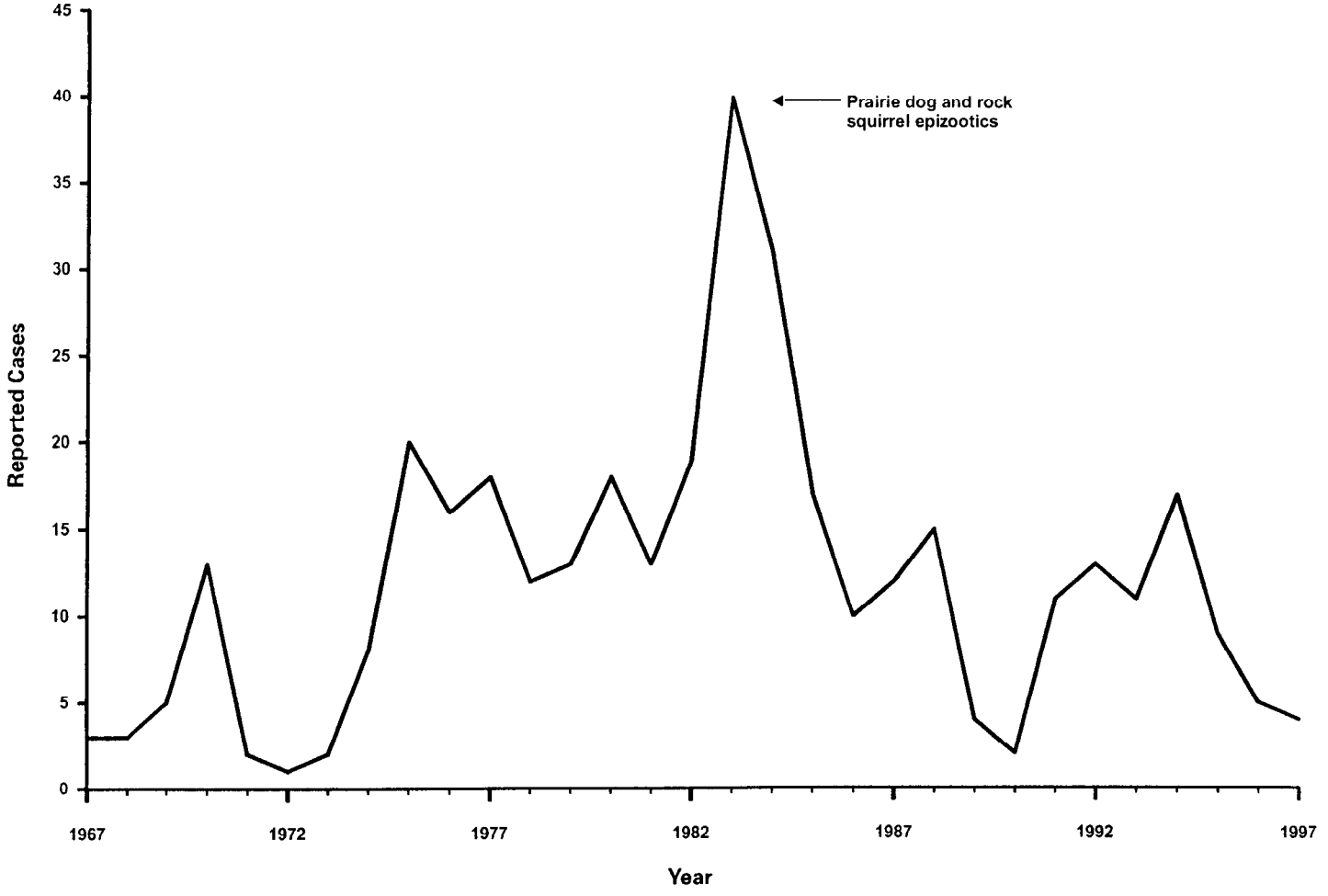
Pertussis epidemics occur every 3–4 years. During the last epidemic year (1996), the highest number of pertussis cases (7,796) since 1967 was reported with an incidence of 2.9 per 100,000 population. Since 1993, after each epidemic year, the number of reported cases has not returned to the baseline of the preepidemic year.

PERTUSSIS (Whooping Cough) — by age group, United States, 1997



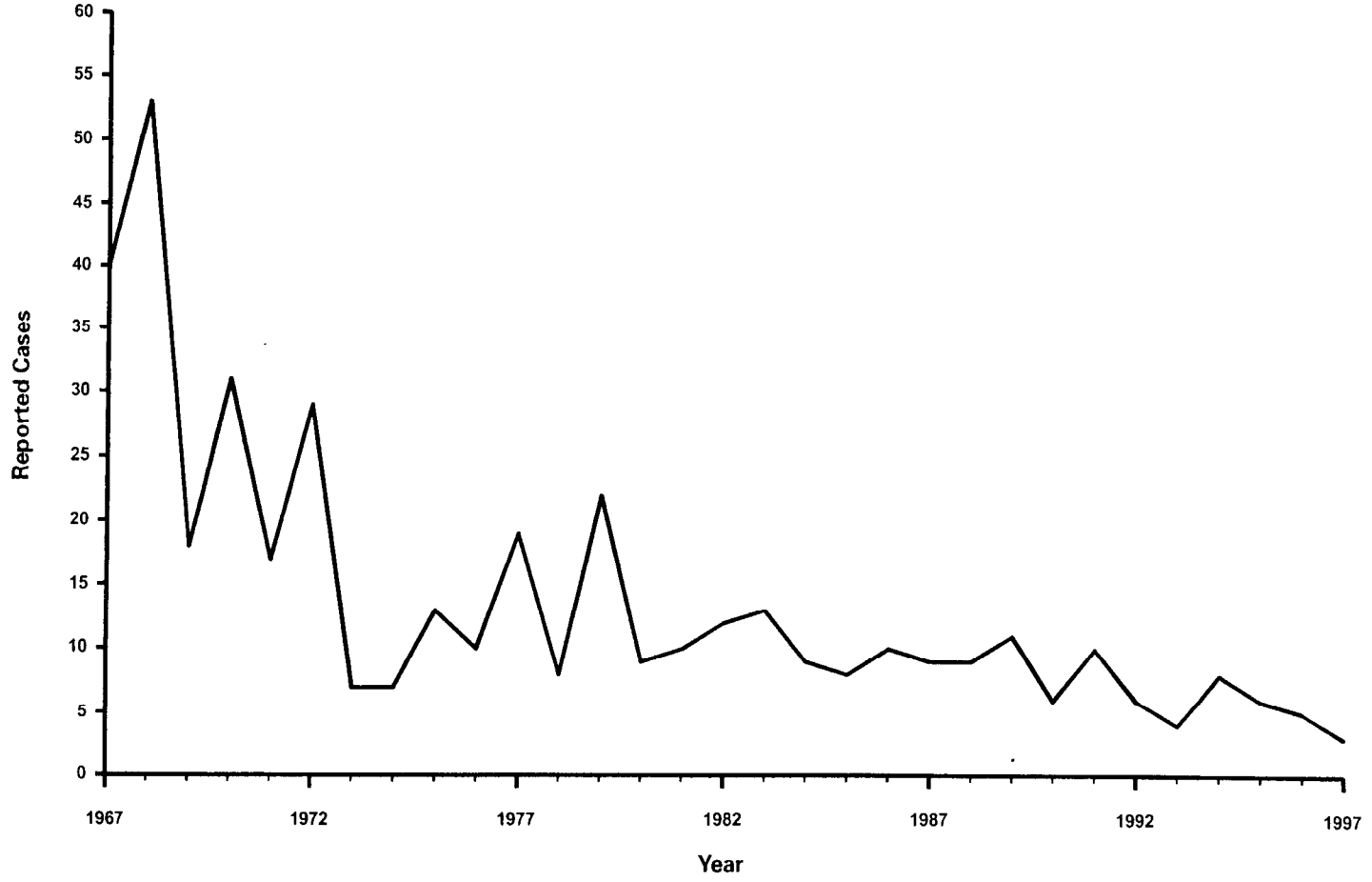
Although the highest number of reported cases continues to be among children aged <1 year, pertussis cases among adolescents and adults increasingly are being reported to CDC. In 1997, 46% of all reported pertussis cases occurred among persons aged ≥10 years. By comparison, during 1990–1992, 1993–1995, and 1996, the proportion of reported pertussis cases among persons aged ≥10 years was 24%, 29%, and 44%, respectively.

PLAGUE — among humans, by year, United States, 1967–1997



In 1997, four plague cases among humans were reported in the United States (two cases in California, one in Arizona, and one in Colorado). One case was fatal and diagnosed postmortem as septicemic plague.

POLIOMYELITIS (paralytic) — by year, United States, 1967–1997



NOTE Inactivated vaccine was licensed in 1955. Oral vaccine was licensed in 1961.

Of 142 cases of indigenously acquired paralytic poliomyelitis reported during 1980–1997, a total of 140 were associated with the administration of oral poliovirus vaccine (OPV). The remaining two cases were classified as indeterminate. To reduce the burden of poliomyelitis associated with the use of OPV, in January 1997, the Advisory Committee on Immunization Practices (ACIP) recommended a sequential schedule of two doses of inactivated poliovirus vaccine (IPV) followed by two doses of OPV.

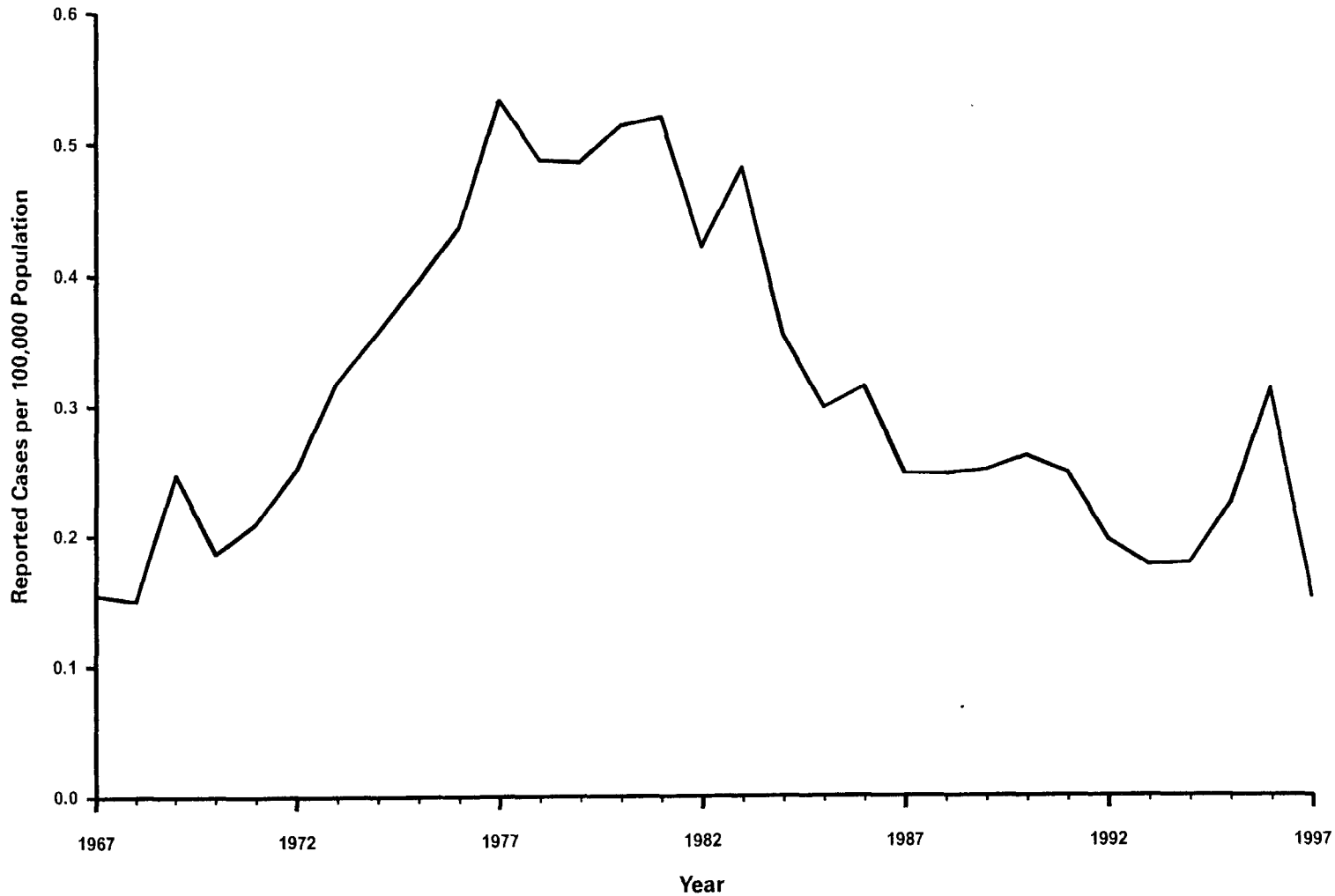
PSITTACOSIS — by year, United States, 1967–1997

The number of psittacosis cases can vary from year to year because of periodic outbreaks. The apparent increase in cases during the late 1970s to mid-1980s might reflect greater application of diagnostic tests for *Chlamydia* species in patients with respiratory illness. The lower number of cases in recent years might reflect both improved diagnostic testing for distinguishing *C. psittaci* from *C. pneumoniae* infections and improvement in control measures for *C. psittaci* infection in birds.

RABIES — wild and domestic animals, by year, United States and Puerto Rico, 1967–1997

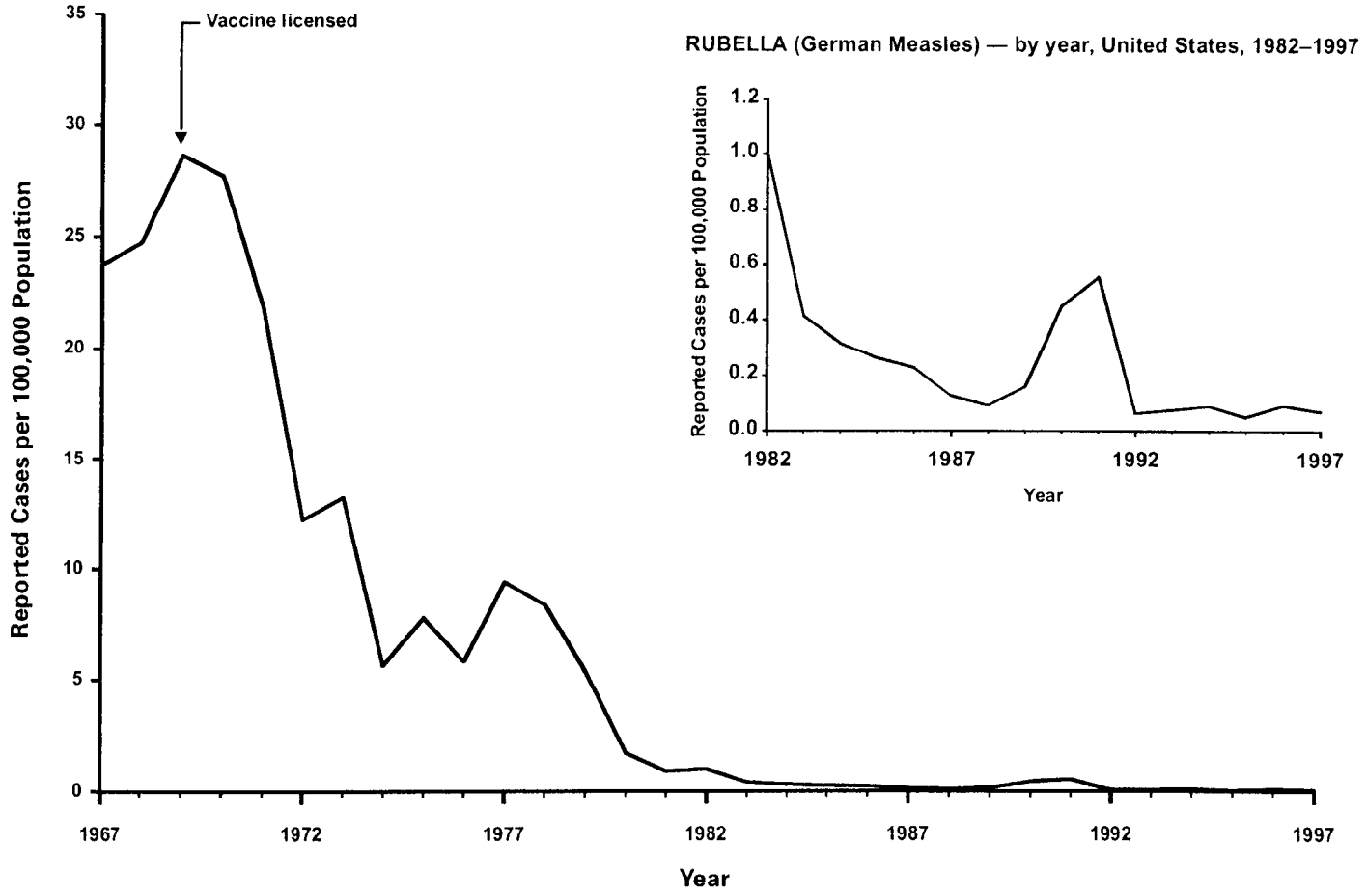
The resurgence of reported cases, following three consecutive years of decline, is primarily the result of cyclic or periodic reemergence of rabies, mainly among raccoons in the eastern United States. During 1997, populations variously decimated by previous epizootics again reached densities sufficient to support epizootic transmission of the disease.

ROCKY MOUNTAIN SPOTTED FEVER (RMSF) — by year, United States, 1967–1997



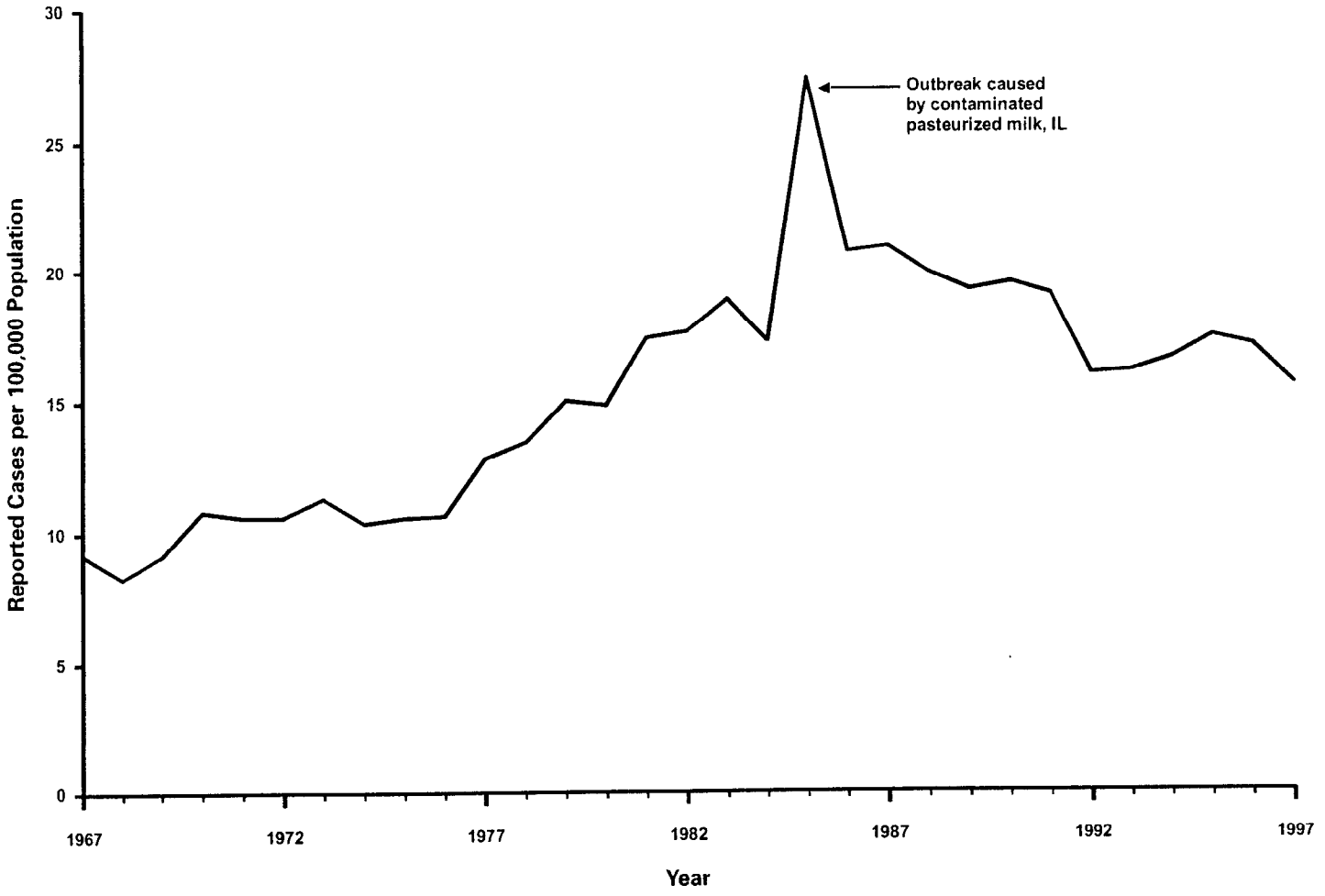
Changes in reported cases of Rocky Mountain spotted fever might reflect alterations to surveillance algorithms for this and other tickborne diseases. Biological factors (e.g., changes in tick populations resulting from fluctuating environmental conditions) also could be involved.

RUBELLA (German Measles) — by year, United States, 1967–1997



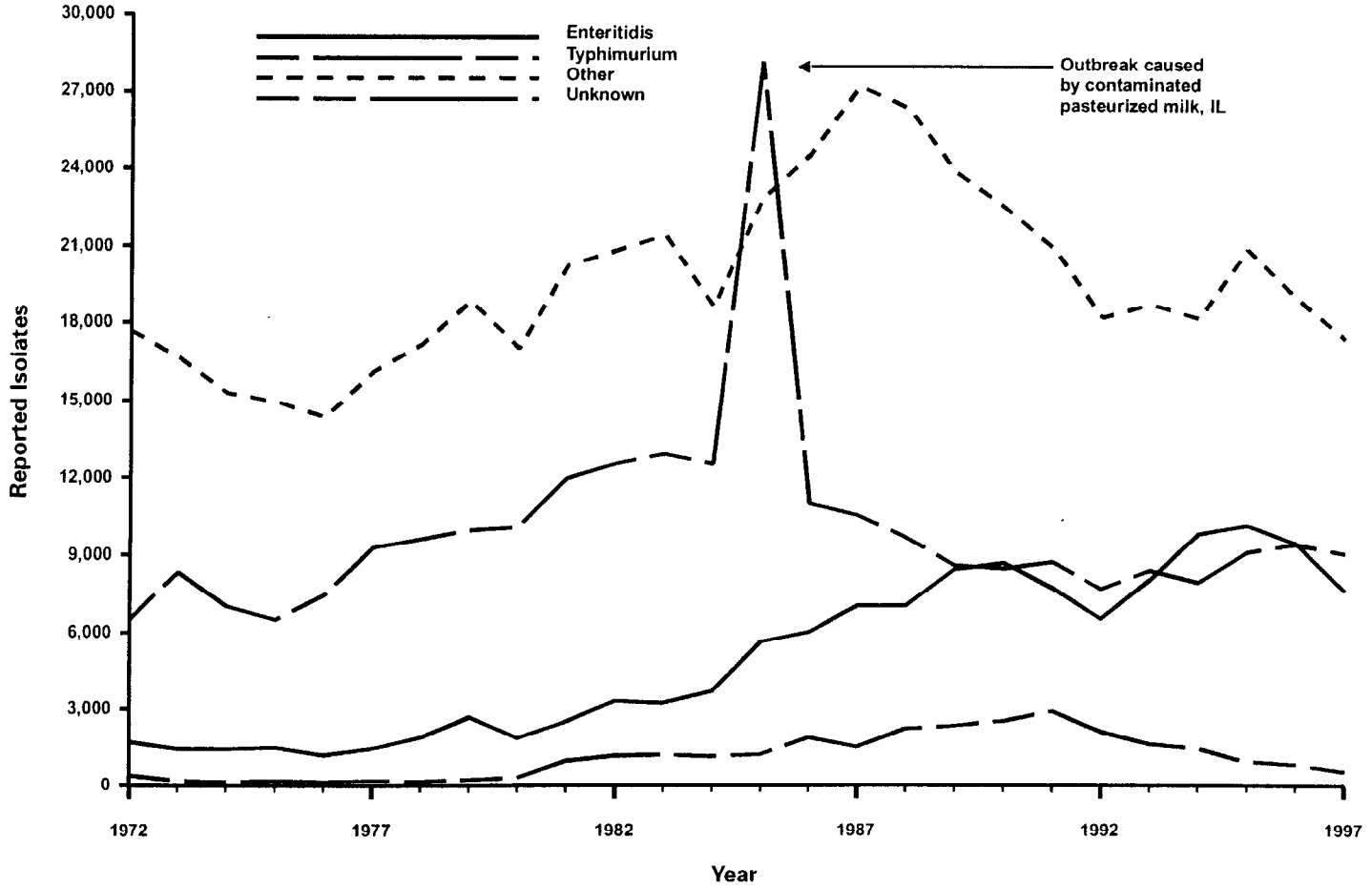
The incidence of reported rubella has decreased steadily. The highest proportion of cases is reported among persons aged >20 years.

SALMONELLOSIS (excluding Typhoid Fever) — by year, United States, 1967–1997



In 1997, *Salmonella* serotypes Typhimurium and Enteritidis together accounted for 46% of all salmonellosis reported in humans.

SALMONELLA — serotype of isolate by year,* United States, 1972–1997

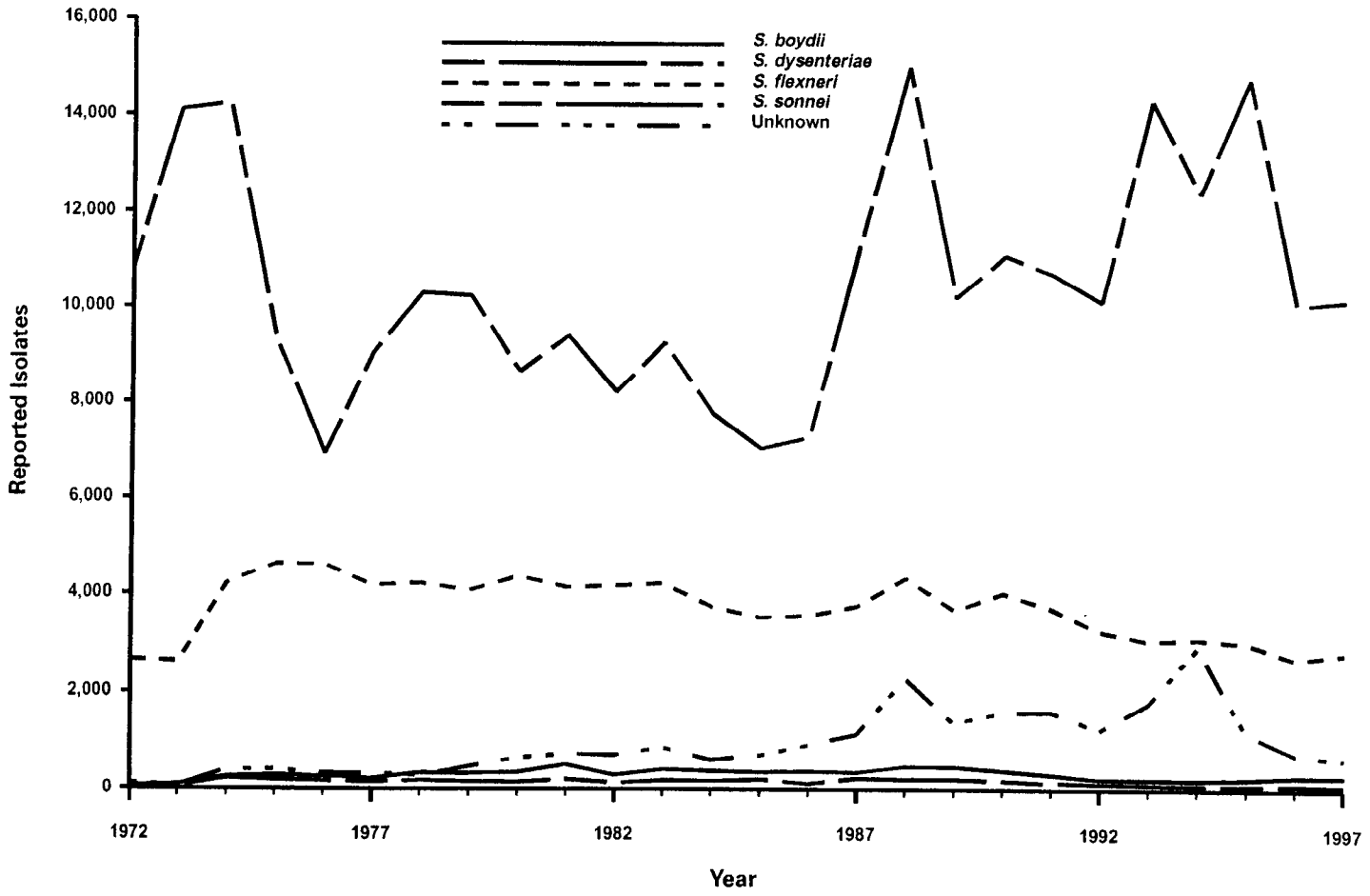


*Data from Public Health Laboratory Information System (PHLIS)

In 1997, Typhimurium was the most common ***Salmonella*** serotype isolated from humans; approximately 35% of all reported *S.* Typhimurium strains from humans are now resistant to five antimicrobial agents (i.e., ampicillin, chloramphenicol, sulfonamide, streptomycin, and tetracycline).

SHIGELLOSIS — by year, United States, 1967–1997

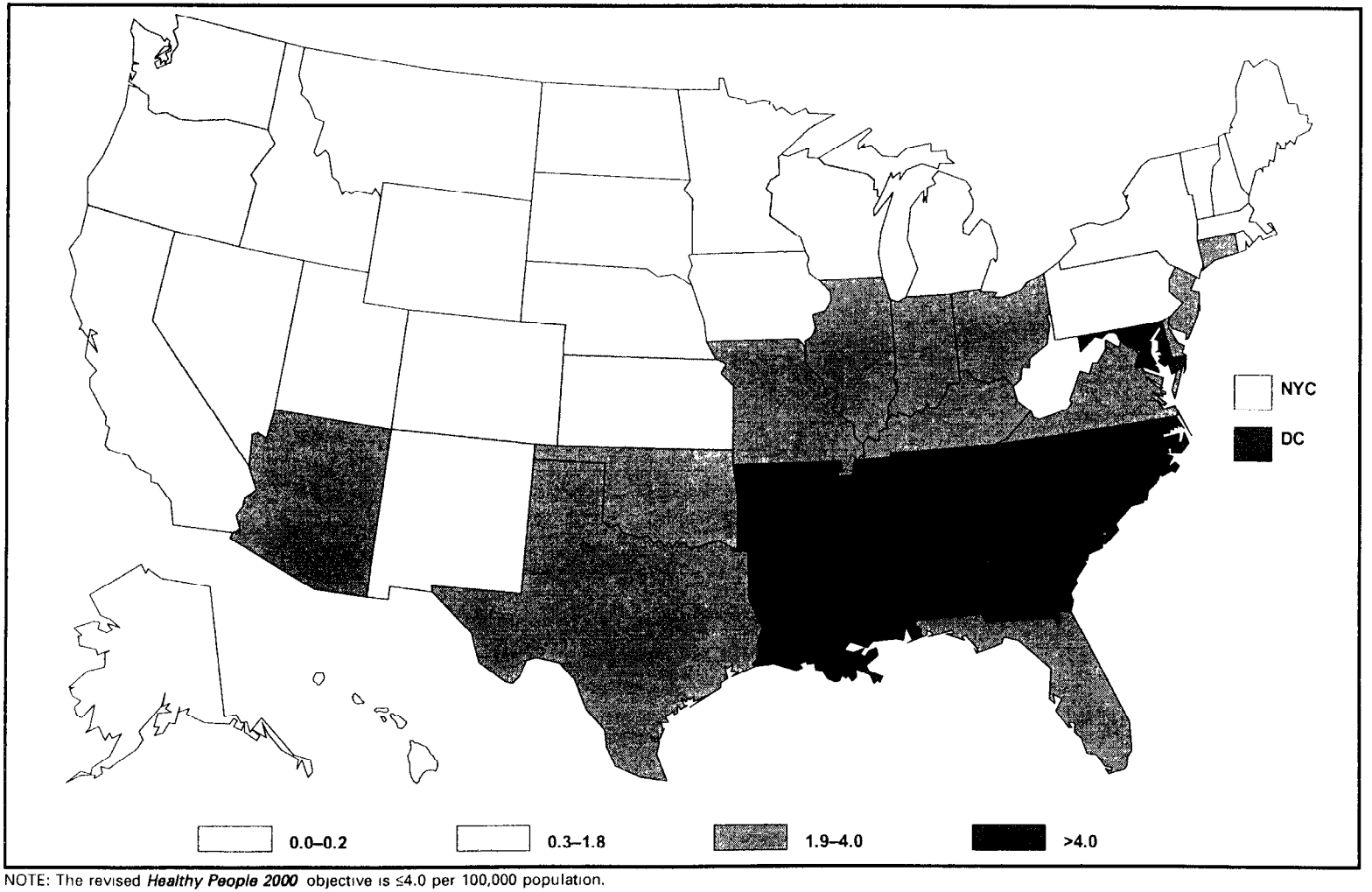
SHIGELLA — species of isolate by year,* United States, 1972–1997



* Data from Public Health Laboratory Information System (PHLIS).

Antimicrobial resistance among ***Shigella*** isolates has continued to increase: nearly 20% of ***Shigella*** isolates in the United States are resistant to both ampicillin and trimethoprim-sulfamethoxazole.

SYPHILIS (Primary and Secondary) — reported cases per 100,000 population, United States, 1997



NOTE: The revised ***Healthy People 2000*** objective is ≤4.0 per 100,000 population.

In 1997, the U.S. rate of primary and secondary syphilis of 3.2 per 100,000 population was below the revised national ***Healthy People 2000*** objective. Forty-one states reported rates below the national objective, and 12 states reported fewer than five cases.

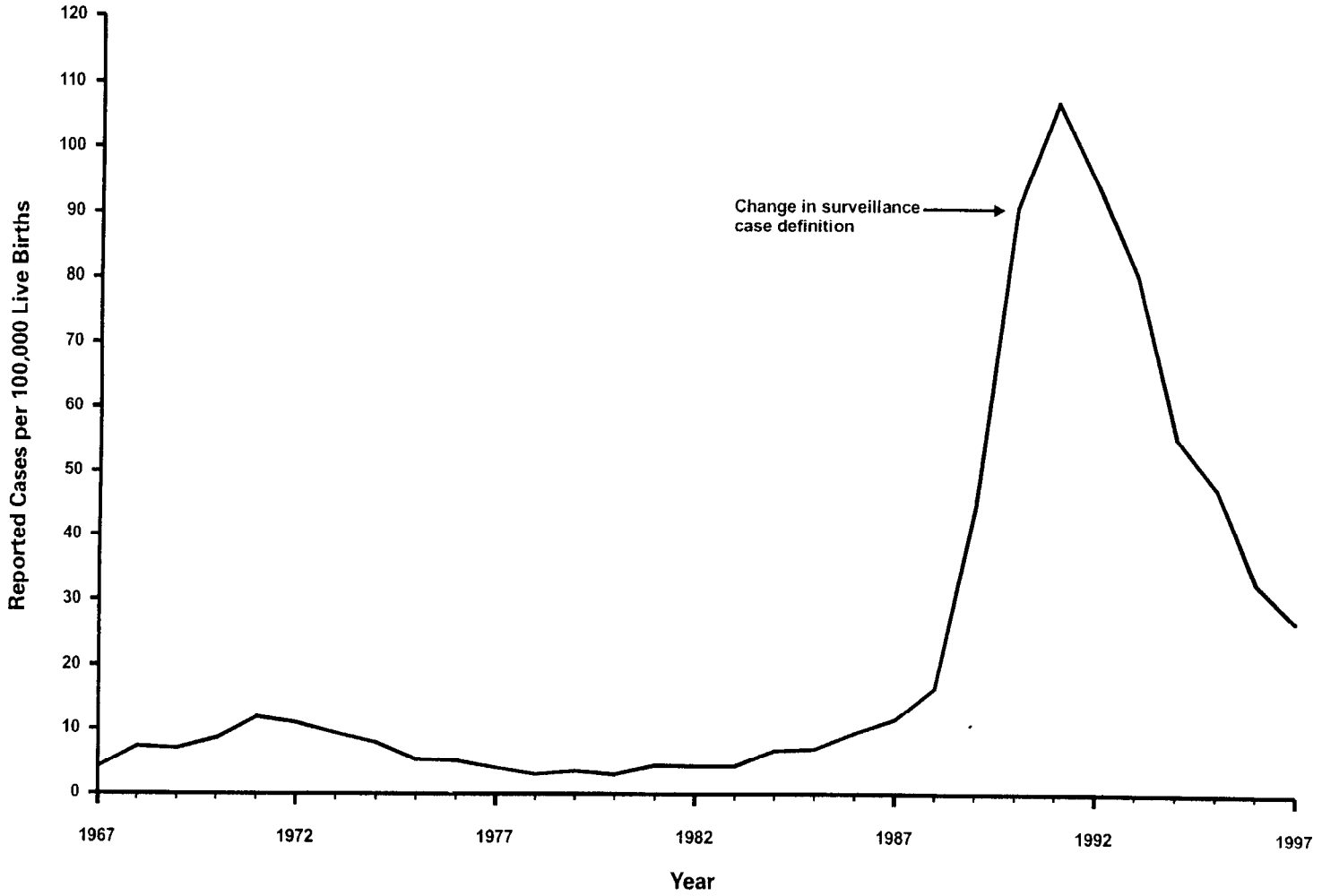
SYPHILIS (Primary and Secondary) — by sex, United States, 1982–1997

In 1997, the reported rate of primary and secondary syphilis in the United States continued to decline, with rates among both males and females below the ***Healthy People 2000*** objective of 4.0 per 100,000 population. Among men, the rate decreased from 4.7 per 100,000 population in 1996 to 3.6 in 1997. Among women, the rate decreased from 4.0 per 100,000 population in 1996 to 2.9 in 1997.

SYPHILIS (Primary and Secondary) — by race and ethnicity, United States, 1982–1997

In 1997, primary and secondary syphilis rates for all racial and ethnic groups declined. In 1997, however, the rate for non-Hispanic blacks (i.e., 22.0 cases per 100,000 population) was 44-fold greater than that for non-Hispanic whites.

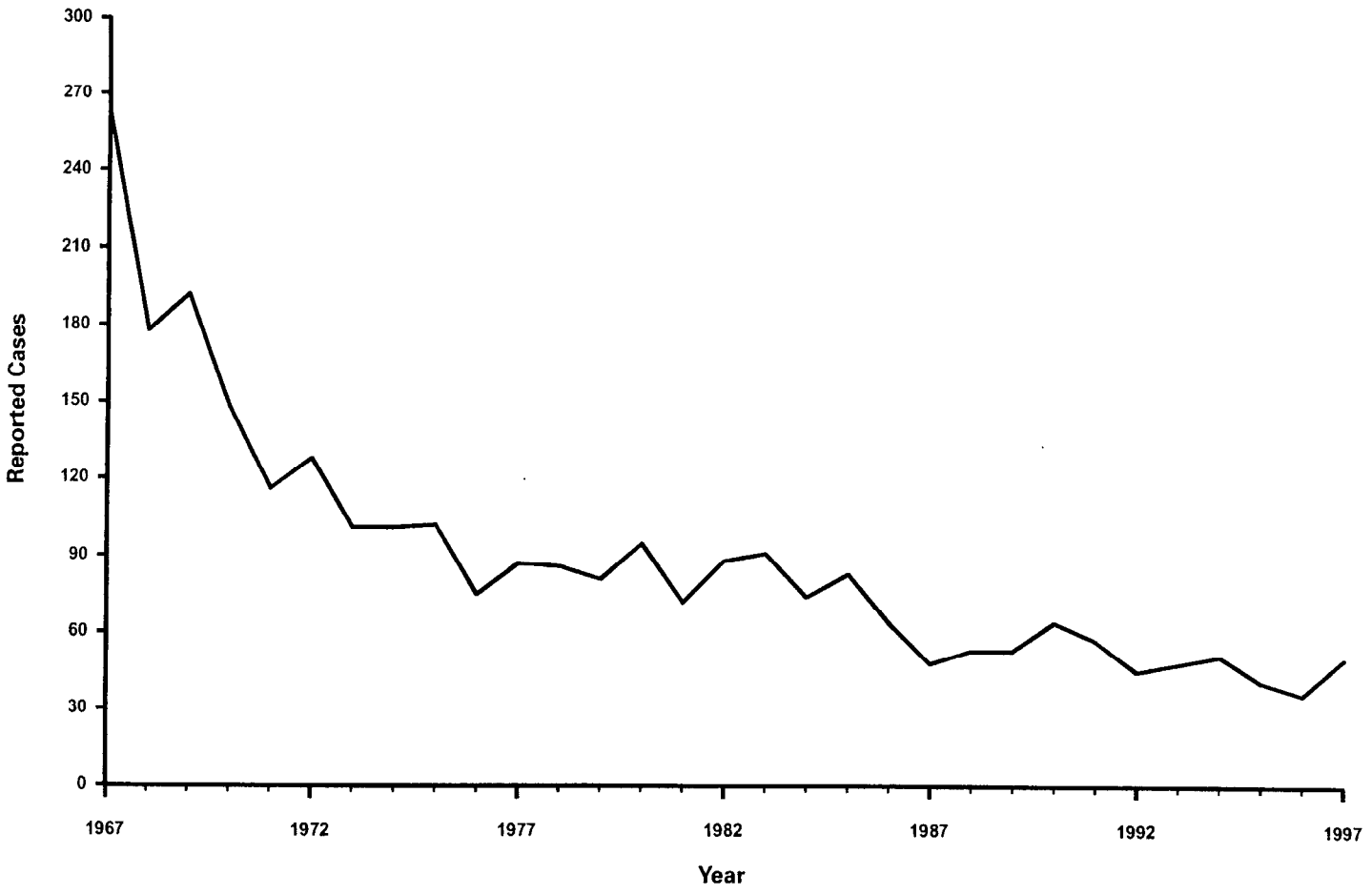
CONGENITAL SYPHILIS — among infants aged <1 year, United States, 1967–1997



The rate of congenital syphilis decreased from 32.9 cases per 100,000 live births in 1996 to 26.9 in 1997.*

*Data Source: Division of Sexually Transmitted Diseases Prevention, National Center for HIV, STD, and TB Prevention.

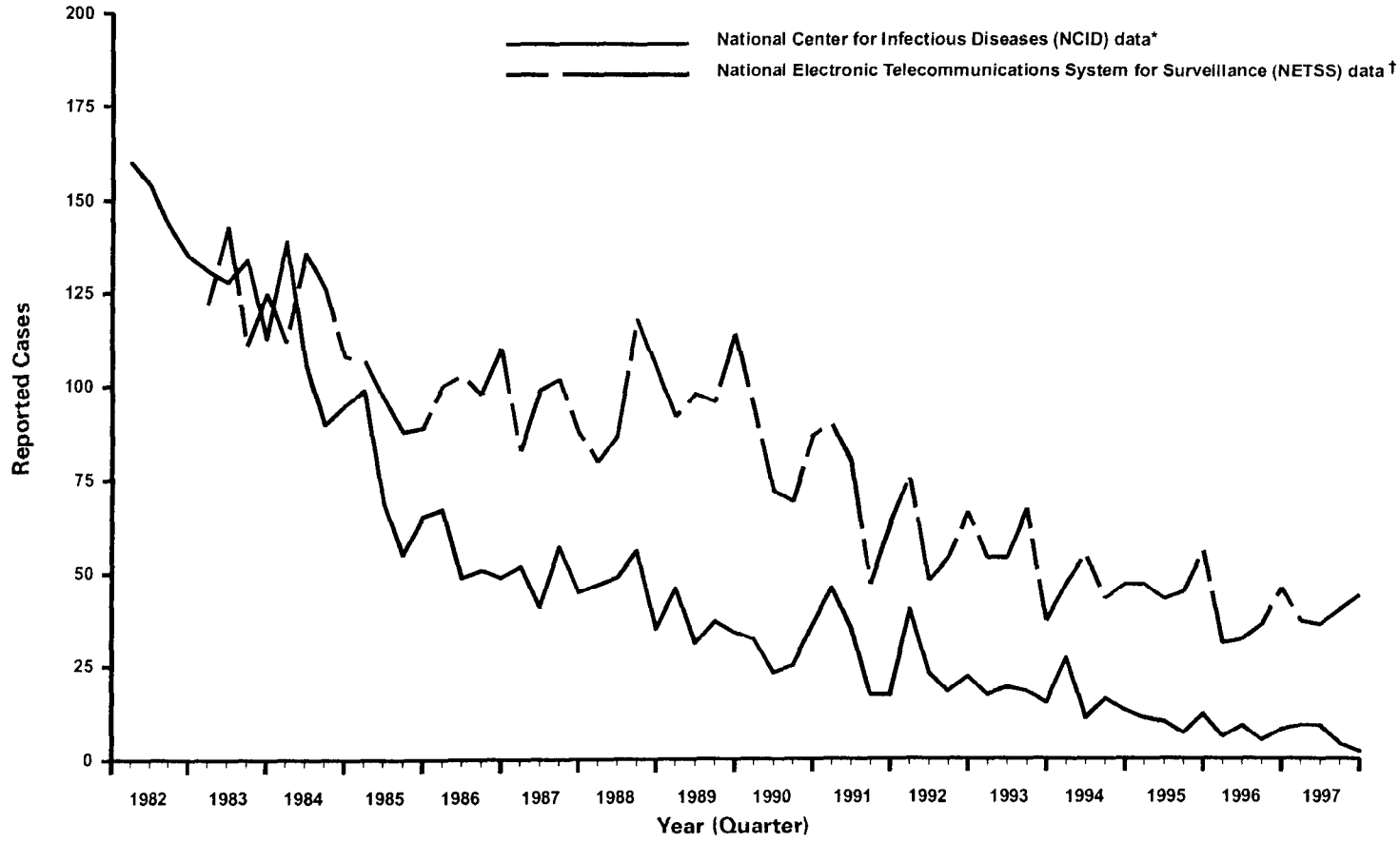
TETANUS — by year, United States, 1967–1997



NOTE: Tetanus toxoid was first available in 1933

Tetanus among persons aged <25 years has been targeted for elimination within the United States by the year 2000. From 1995 through 1997, 12 (9.7%) of 124 reported cases were among persons aged <25 years, including one case in a neonate and three cases that occurred among persons with religious objections to vaccination.

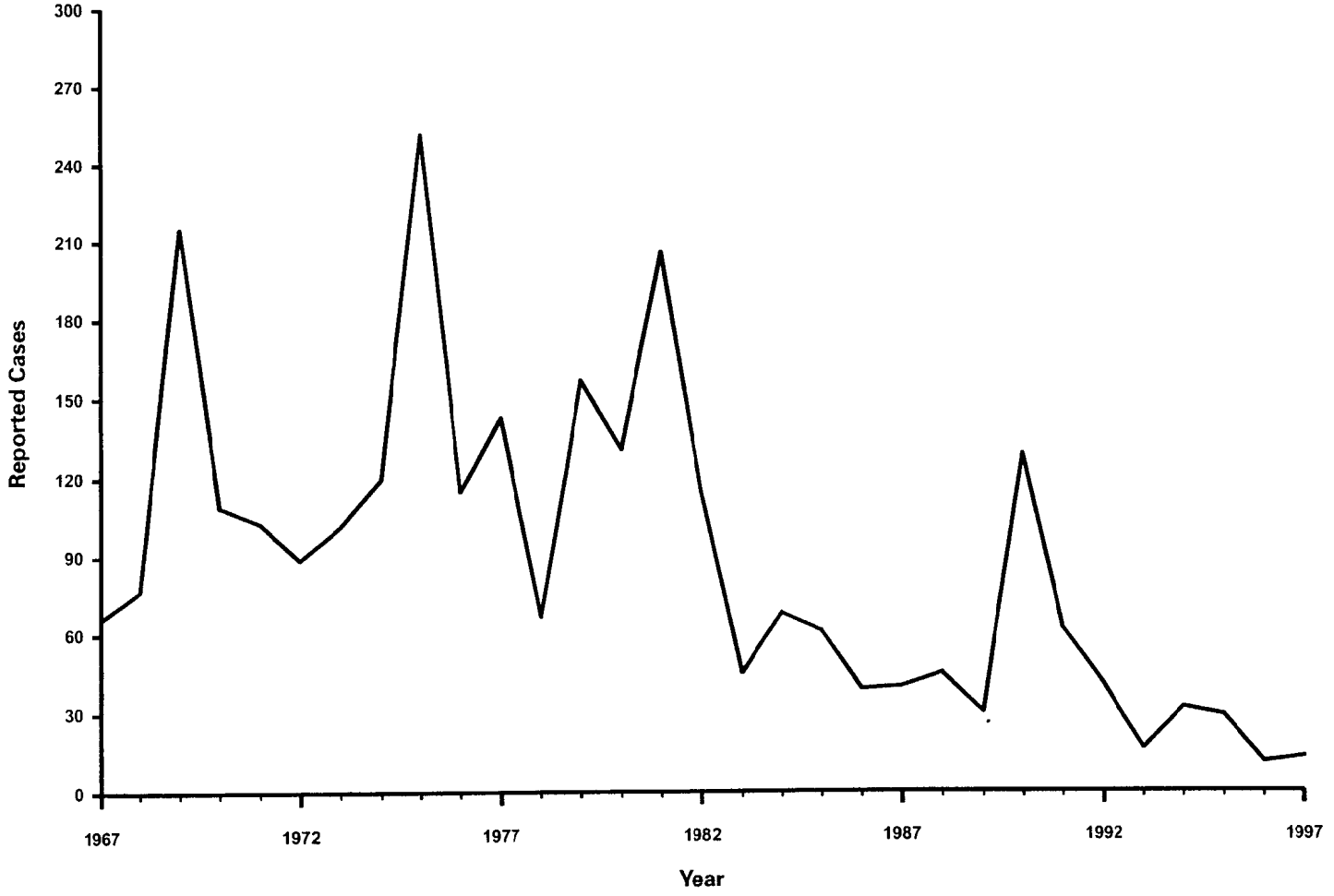
TOXIC-SHOCK SYNDROME (TSS) — by quarter, United States, 1982–1997



*Includes cases meeting the CDC definition for confirmed and probable cases for staphylococcal TSS (n=5,087)
†TSS data were first available through NETSS in 1983.

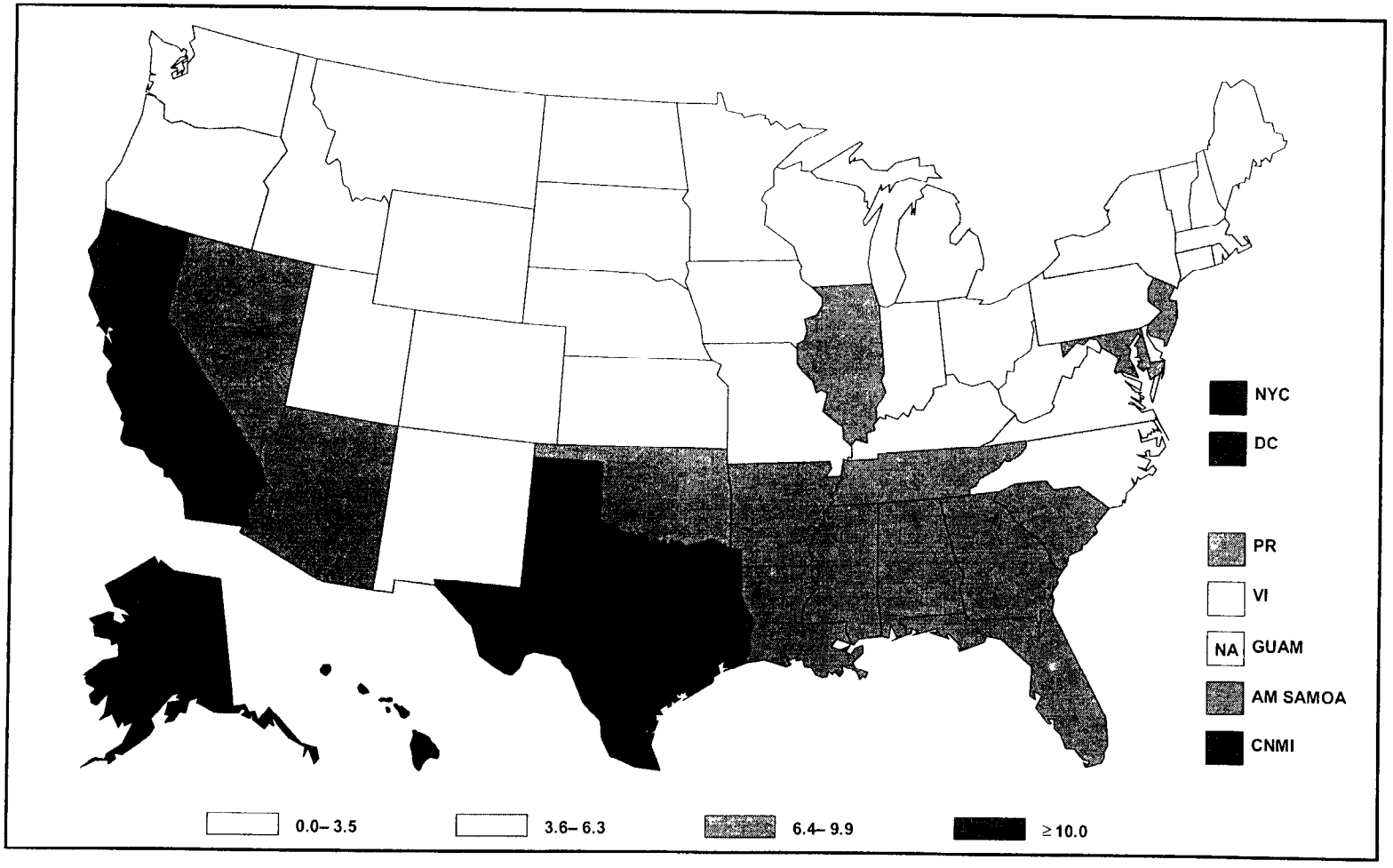
Although the number of cases of TSS reported through NETSS or NCID has not changed significantly over the last 5 years, trends of TSS should continue to be monitored, especially because new products (e.g., all-cotton tampons) and use patterns (e.g., using tampons overnight) have been introduced recently.

TRICHINOSIS — by year, United States, 1967–1997



In 1997, a total of 13 trichinellosis (trichinosis) cases were reported, remaining at the lowest levels ever reported.

TUBERCULOSIS — reported cases per 100,000 population, United States and territories, 1997

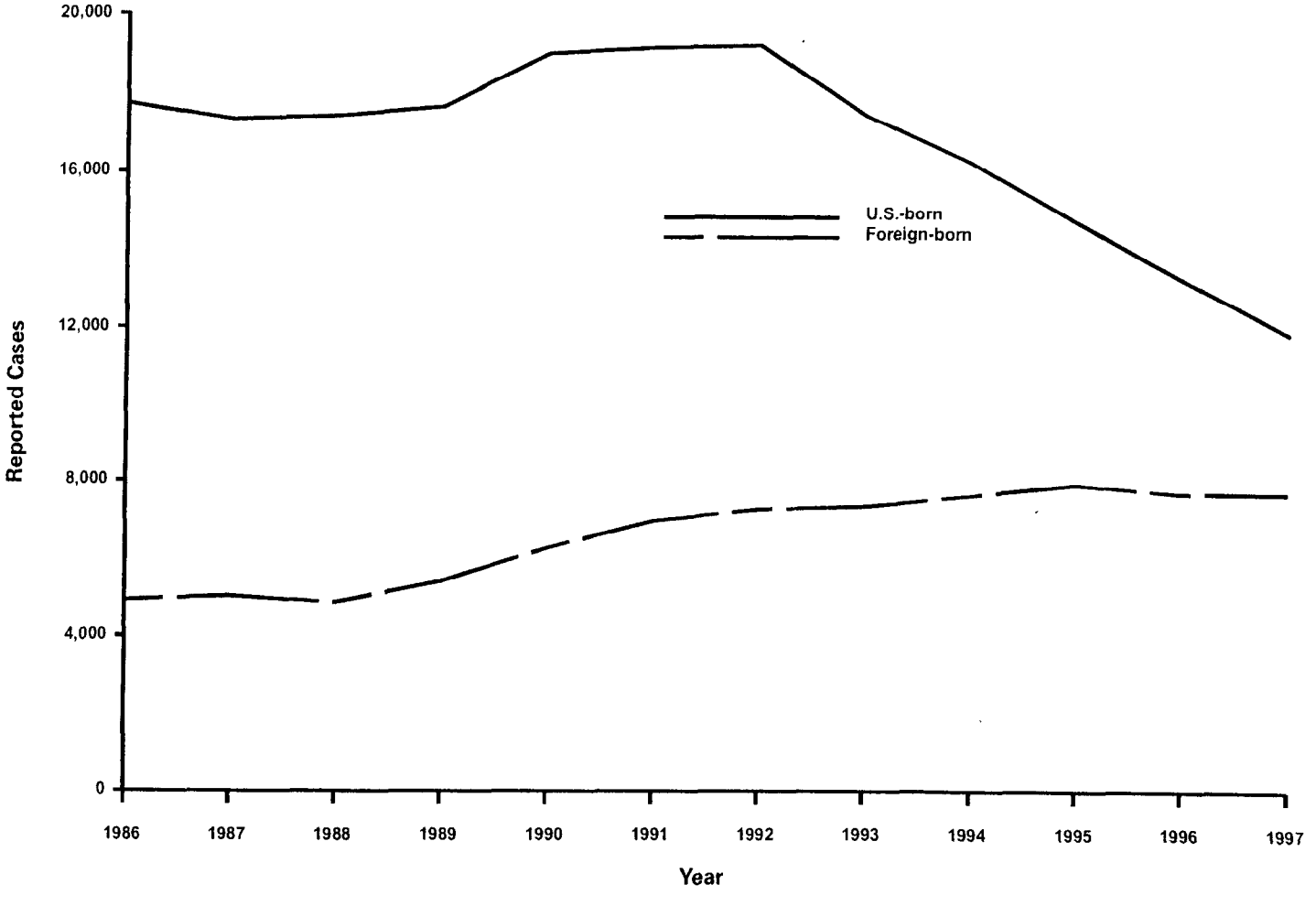


In 1997, a total of 18 states had tuberculosis rates of ≤3.5 cases per 100,000 population, which is the interim (i.e., Year 2000) tuberculosis incidence target for the elimination of tuberculosis by the year 2010.

TUBERCULOSIS — by year, United States, 1977–1997

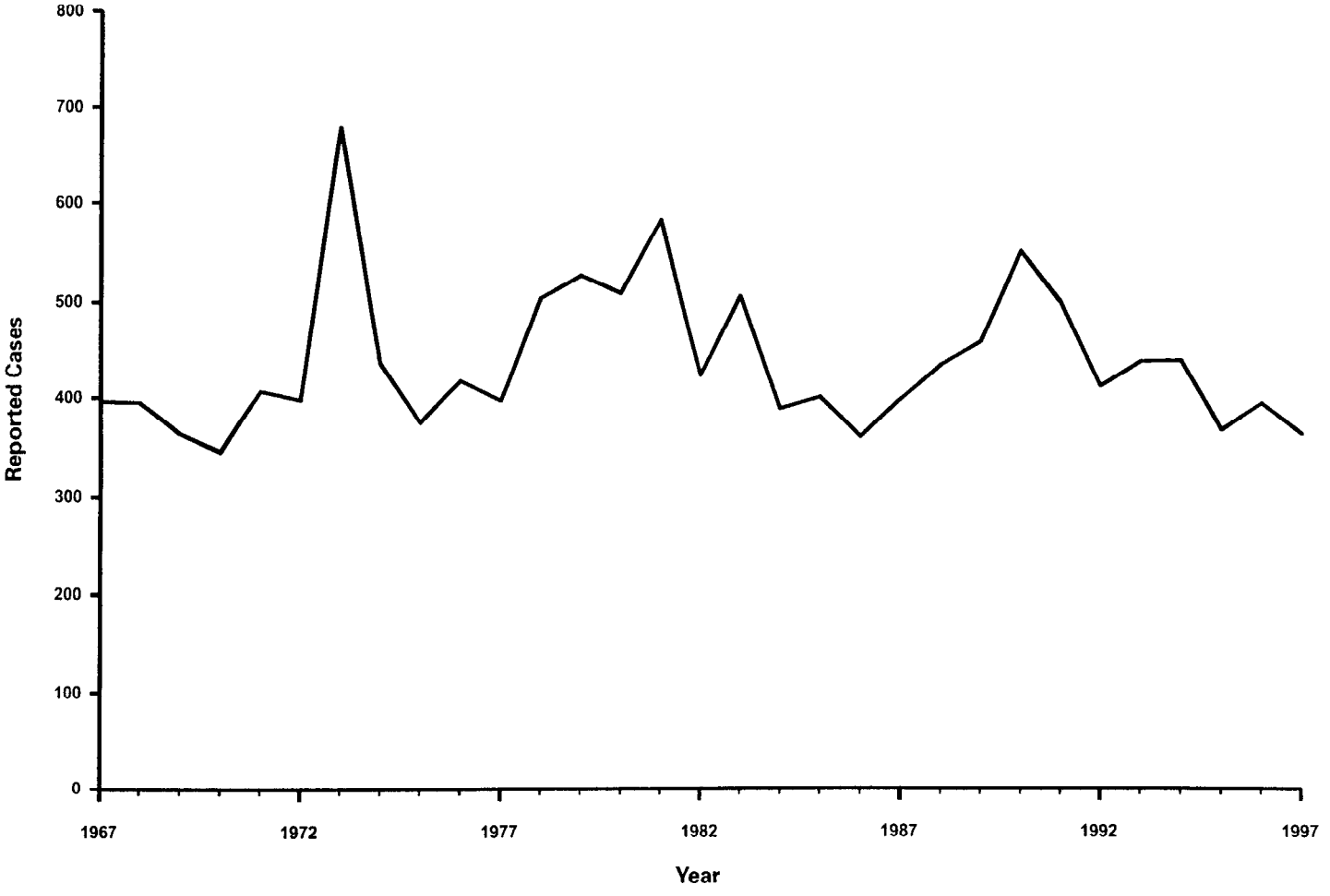
In 1997, a total of 19,851 cases of tuberculosis were reported to CDC, representing a 7% decrease from 1996.

TUBERCULOSIS — by year, among U.S.- and foreign-born persons, United States, 1986–1997



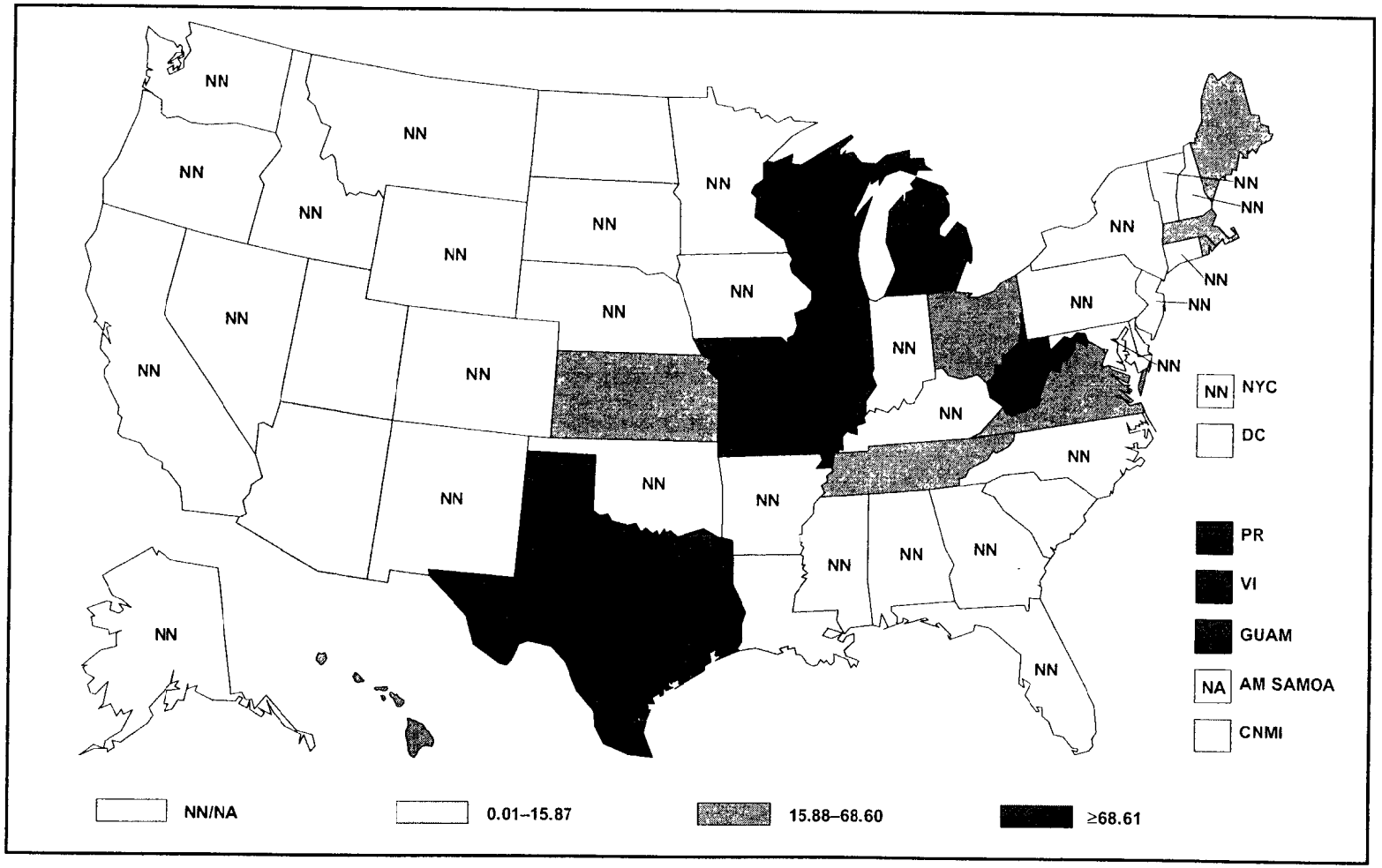
The number (and percentage) of tuberculosis cases among foreign-born persons in the United States has increased from 4,925 (21.6%) in 1986 to 7,702 (38.8%) in 1997.

TYPHOID FEVER — by year, United States, 1967–1997



Antimicrobial resistance among *Salmonella* serotype Typhi isolates has continued to increase, as has the proportion of typhoid fever cases that are preventable through immunization of travelers.

VARICELLA (Chickenpox) — reported cases per 100,000 population, United States and territories, 1997



Varicella is not a nationally notifiable disease; however, in 1997, 20 states, the District of Columbia, and four territories reported cases via the National Notifiable Diseases Surveillance System. This map reflects data from states where varicella is notifiable at the state level.

PART 3:

Historical Summary Tables

EXPLANATION OF SYMBOLS USED IN TABLES, GRAPHS, AND MAPS	
No reported cases	–

TABLE 1. NOTIFIABLE DISEASES — Summary of reported cases per 100,000 population, United States, 1988–1997

Disease	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997
AIDS*	12.61	13.58	16 72	17.32	17.83	40 20	30.07	27.20	25.21	21.85
Amebiasis	1.20	1.34	1.38	1 23	1.21	1 21	1 20	.	†.	.
Anthrax	0.00	–	–	–	0 00	–	–	–	–	–
Aseptic meningitis	2.94	4 14	4 77	6 26	5 18	5.39	3 71	.	†	.
Botulism, total (including wound and unsp)	0.03	0.04	0 04	0 05	0 04	0 04	0 06	0 04	0.05	0 05
Foodborne	0.01	0.01	0 01	0.01	0.00	0.01	0 02	0.01	0 01	0 02
Brucellosis	0 04	0.04	0 03	0 04	0 04	0 05	0 05	0.04	0 05	0.04
Chancroid	2 04	1.90	1.70	1.40	0 80	0 54	0.30	0.20	0.15	0 09
Chlamydia§	.	.	.	¶.	.	.	.	182 60	188.10	196 80
Cholera	0.00	–	0 00	0.01	0.04	0 00	0 02	0.01	0 01	0.01
Cryptosporidiosis	.	.	.	.	¶.	.	.	.	.	1.12
Diphtheria	0.00	0 00	0.00	0.00	0.00	–	0 00	–	0 01	0 01
Encephalitis, primary	0 36	0.40	0.54	0 40	0 30	0 36	0.28	.	†	.
Post-infectious	0.05	0.04	0.04	0.03	0 05	0.07	0 06	.	†	.
Escherichia coli O157:H7	.	.	.	¶.	.	.	0.82	1 01	1 18	1 04
Gonorrhea	298.74	297.36	276.60	249 48	201.60	172.40	168.40	149 50	122.80	121 40
Granuloma inguinale	0 00	0.00	0.00	0.01	0.00	0 00	0.00	.	†.	.
Haemophilus influenzae, invasive	.	.	¶	.	1.10	0 55	0.55	0.45	0.45	0 45	0 44
Hansen disease (leprosy)	0.07	0.07	0 08	0 06	0.07	0 07	0.05	0.06	0 05	0 05
Hepatitis A	11.60	14 43	12.64	9.67	9.06	9.40	10 29	12 13	11 70	11.22
Hepatitis B	9.43	9.43	8.48	7 14	6 32	5.18	4.81	4 19	4.01	3.90
Hepatitis, C/non-A, non-B**	1.07	1.02	1 03	1.42	2 36	1 86	1 78	1.78	1 41	1 43
Hepatitis, unspecified	1 00	0.93	0 67	0 50	0.35	0.24	0 17	.	†	.
Legionellosis	0.44	0.48	0.55	0 53	0 53	0 50	0 63	0.48	0.47	0 44
Leptospirosis	0 02	0.04	0.03	0 02	0.02	0.02	0.02	.	†.	.
Lyme disease	.	.	¶.	.	3.80	3 93	3 20	5.01	4 49	6.21	4.79
Lymphogranuloma venereum	0.07	0.08	0.10	0 19	0.10	0 10	0.10	.	†.	.
Malaria	0.45	0.51	0.52	0 51	0.43	0.55	0.47	0.55	0 68	0.75
Measles (rubeola)	1.38	7.33	11 17	3.82	0.88	0.12	0 37	0.12	0 20	0.06
Meningococcal disease	1.21	1.10	0.99	0 84	0 84	1 02	1.11	1.25	1 30	1.24
Mumps	2.05	2.34	2 17	1.72	1.03	0.66	0.60	0 35	0.29	0.27
Murine typhus fever	0.02	0.02	0 02	0.02	0.02	0 01	.	†	.	.
Pertussis (whooping cough)	1.40	1.67	1.84	1.08	1.60	2.55	1.77	1 97	2 94	2.46
Plague	0.01	0.00	0.00	0.00	0.01	0.00	0 01	0 00	0 01	0 01
Poliomyelitis, paralytic	0 00	0 00	0 00	0.00	0.00	0.00	0 00	0.00	0.01	0.01
Psittacosis	0.05	0.05	0 05	0 04	0.04	0.02	0.02	0 03	0 02	0.02
Rabies, human	0.00	0 00	0.00	0.00	0.00	0.00	0 00	0.00	0.01	0 01
Rheumatic fever, acute	0.14	0.13	0.09	0.12	0.06	0 08	0.09	.	†.	.
Rocky Mountain spotted fever	0.25	0.25	0.26	0.25	0.20	0.18	0 18	0.23	0.32	0.16
Rubella (German measles)	0.09	0.16	0.45	0.56	0.06	0.07	0 09	0.05	0.10	0.07
Salmonellosis, excluding typhoid fever	19.91	19.26	19.54	19.10	16.04	16.15	16 64	17 66	17.15	15 66
Shigellosis	12.46	10.07	10.89	9.34	9.38	12.48	11.44	12.32	9 80	8 64
Syphilis, primary and secondary	16.43	18.07	20.10	17 26	13.70	10.40	8.10	6.30	4 29	3.19
Total, all stages	42.37	44.94	53.80	51.69	45.30	39.70	32 00	26.20	19.97	17 39
Tetanus	0.02	0.02	0.03	0.02	0.02	0.02	0 02	0.02	0.02	0 02
Toxic-shock syndrome	0.16	0.16	0.13	0.11	0 10	0.08	0.10	0 07	0.06	0.06
Trichinosis	0.02	0 01	0 05	0.02	0.02	0.01	0.01	0 01	0.01	0 01
Tuberculosis	9.13	9.46	10.33	10.42	10.46	9.82	9 36	8.70	8.04	7.42
Tularemia	0.08	0.06	0.06	0.08	0.06	0.05	0.04	.	†.	.
Typhoid fever	0.18	0 19	0.22	0 20	0 16	0 17	0.17	0.14	0 15	0.14
Varicella (chickenpox)††	122 43	121.77	120.06	135.82	176 54	118.54	135.76	118 11	44 13	93.55
Yellow fever	–	–	–	–	–	–	–	–	0.01	–

NOTES. Data in the annual *Summary of Notifiable Diseases* might not match data in other CDC surveillance reports because of differences in the timing of reports, the source of the data, and the use of different case definitions. Rates <0.01 after rounding are listed as 0.00.

* Acquired immunodeficiency syndrome.
† No longer nationally notifiable
§ Chlamydia refers to genital infections caused by *C. trachomatis.*
¶ Not previously nationally notifiable.
** Anti-HCV antibody test became available May 1990.
†† Not nationally notifiable.

TABLE 2. NOTIFIABLE DISEASES — Summary of reported cases, United States, 1990–1997

Disease	1990	1991	1992	1993	1994	1995	1996	1997
AIDS*	41,595	43,672	45,472	103,691	78,279	71,547	66,885	58,492†
Amebiasis	3,328	2,989	2,942	2,970	2,983	§	§	§
Anthrax	–	–	1	–	–	–	–	–
Aseptic meningitis	11,852	14,526	12,223	12,848	8,932	§	§	§
Botulism, total (including wound and unsp.)	92	114	91	97	143	97	119	132
Foodborne	23	27	21	27	50	24	25	31
Infant	65	81	66	65	85	54	80	79
Brucellosis	85	104	105	120	119	98	112	98
Chancroid	4,212	3,476	1,886	1,399	773	606	386	243¶
Chlamydia**	††	††	††	††	††	477,638	498,884	526,671¶
Cholera	6	26	103	18	39	23	4	6
Cryptosporidiosis	††	††	††	††	††	††	††	2,566
Diphtheria	4	5	4	–	2	–	2	4
Encephalitis, primary	1,341	1,021	774	919	717	§	§	§
Post-infectious	105	82	129	170	143	§	§	§
Escherichia coli O157:H7	††	††	††	††	1,420	2,139	2,741	2,555
Gonorrhea	690,169	620,478	501,409	439,673	418,068	392,848	325,883	324,907¶
Granuloma inguinale	97	29	6	19	3	§	§	§
Haemophilus influenzae, invasive	††	2,764	1,412	1,419	1,174	1,180	1,170	1,162
Hansen disease (leprosy)	198	154	172	187	136	144	112	122
Hepatitis A	31,441	24,378	23,112	24,238	26,796	31,582	31,032	30,021
Hepatitis B	21,102	18,003	16,126	13,361	12,517	10,805	10,637	10,416
Hepatitis, C/non-A, non-B§§	2,553	3,582	6,010	4,786	4,470	4,576	3,716	3,816
Hepatitis, unspecified	1,671	1,260	884	627	444	§	§	§
Legionellosis	1,370	1,317	1,339	1,280	1,615	1,241	1,198	1,163
Leptospirosis	77	58	54	51	38	§	§	§
Lyme disease	††	9,465	9,895	8,257	13,043	11,700	16,455	12,801
Lymphogranuloma venereum	277	471	302	285	235	§	§	§
Malaria	1,292	1,278	1,087	1,411	1,229	1,419	1,800	2,001
Measles (rubeola)	27,786	9,643	2,237	312	963	309	508	138
Meningococcal disease	2,451	2,130	2,134	2,637	2,886	3,243	3,437	3,308
Mumps	5,292	4,264	2,572	1,692	1,537	906	751	683
Murine typhus fever	50	43	28	25	§	§	§	§

Pertussis (whooping cough)	4,570	2,719	4,083	6,586	4,617	5,137	7,796	6,564
Plague	2	11	13	10	17	9	5	4
Poliomyelitis, paralytic¶¶	6	10	6	4	8	6	5	3
Psittacosis	113	94	92	60	38	64	42	33
Rabies, animal	4,826	6,910	8,589	9,377	8,147	7,811	6,982	8,105
Rabies, human	1	3	1	3	6	5	3	2
Rheumatic fever, acute	108	127	75	112	112	.....	§	–
Rocky Mountain spotted fever	651	628	502	456	465	590	831	409
Rubella (German measles)	1,125	1,401	160	192	227	128	238	181
Rubella, congenital syndrome	11	47	11	5	7	6	4	5
Salmonellosis, excluding typhoid fever	48,603	48,154	40,912	41,641	43,323	45,970	45,471	41,901
Shigellosis	27,077	23,548	23,931	32,198	29,769	32,080	25,978	23,117
Syphilis, primary and secondary	50,223	42,935	33,973	26,498	20,627	16,500	11,387	8,550¶
Total, all stages	134,255	128,569	112,581	101,259	81,696	68,953	52,976	46,540¶
Tetanus	64	57	45	48	51	41	36	50
Toxic-shock syndrome	322	280	244	212	192	191	145	157
Trichinosis	129	62	41	16	32	29	11	13
Tuberculosis	25,701	26,283	26,673	25,313	24,361	22,860	21,337	19,851***
Tularemia	152	193	159	132	96	.....	§	–
Typhoid fever	552	501	414	440	441	369	396	365
Varicella (chickenpox)†††	173,099	147,076	158,364	134,722	151,219	120,624	83,511	98,727
Yellow fever	.....	.....	.....	§§§	.....	.....	1	–

NOTE Data in the annual *Summary of Notifiable Diseases* might not match data in other CDC surveillance reports because of differences in the timing of reports, the source of the data, and the use of different case definitions.

*Acquired immunodeficiency syndrome

†The total number of AIDS cases includes all cases reported to the Division of HIV/AIDS Prevention — Surveillance and Epidemiology, National Center for HIV, STD, and TB Prevention (NCHSTP) as of December 31, 1997.

§No longer nationally notifiable.

¶Cases were updated through the Division of Sexually Transmitted Diseases Prevention, NCHSTP, as of July 13, 1998.

**Chlamydia refers to genital infections caused by *C. trachomatis*.

††Not previously nationally notifiable.

§§Anti-HCV antibody test was available as of May 1990.

¶¶Numbers might not reflect changes because of retrospective case evaluations or late reports (see *MMWR* 1986;35:180–2).

***Cases were updated through the Division of Tuberculosis Elimination, NCHSTP, as of April 15, 1998 .

TABLE 3. NOTIFIABLE DISEASES — Summary of reported cases, United States, 1982–1989

Disease	1982	1983	1984	1985	1986	1987	1988	1989
AIDS*	†	†	4,445	8,249	12,932	21,070	31,001	33,722
Amebiasis	7,304	6,658	5,252	4,433	3,532	3,123	2,860	3,217
Anthrax	–	–	1	–	–	1	2	–
Aseptic meningitis	9,680	12,696	8,326	10,619	11,374	11,487	7,234	10,274
Botulism, total (including wound and unsp.)	97	133	123	122	109	82	84	89
Foodborne	§	§		49	23	17	28	23
Infant	§	§		70	79	59	50	60
Brucellosis	173	200	131	153	106	129	96	95
Chancroid	1,392	847	665	2,067	3,756	4,998	5,001	4,692
Cholera	–	1	1	4	23	6	8	–
Diphtheria	2	5	1	3	–	3	2	3
Encephalitis, primary¶	1,464	1,761	1,257	1,376	1,302	1,418	882	981
Post-infectious¶	36	34	108	161	124	121	121	88
Gonorrhea	960,633	900,435	878,556	911,419	900,868	780,905	719,536	733,151
Granuloma inguinale	17	24	30	44	61	22	11	7
Hansen disease (leprosy)	250	259	290	361	270	238	184	163
Hepatitis A	23,403	21,532	22,040	23,210	23,430	25,280	28,507	35,821
Hepatitis B	22,177	24,318	26,115	26,611	26,107	25,916	23,177	23,419
Hepatitis, C/non-A, non-B	†	3,470	3,871	4,184	3,634	2,999	2,619	2,529
Hepatitis, unspecified	8,564	7,149	5,531	5,517	3,940	3,102	2,470	2,306
Legionellosis**	654	852	750	830	980	1,038	1,085	1,190
Leptospirosis	100	61	40	57	41	43	54	93
Lymphogranuloma venereum	235	335	170	226	396	303	185	189
Malaria	1,056	813	1,007	1,049	1,123	944	1,099	1,277
Measles (rubeola)	1,714	1,497	2,587	2,822	6,282	3,655	3,396	18,193
Meningococcal disease	3,056	2,736	2,746	2,479	2,594	2,930	2,964	2,727
Mumps	5,270	3,355	3,021	2,982	7,790	12,848	4,866	5,712
Murine typhus fever	58	62	53	37	67	49	54	41
Pertussis (whooping cough)	1,895	2,463	2,276	3,589	4,195	2,823	3,450	4,157

Plague	19	40	31	17	10	12	15	4
Poliomyelitis, total	12	13	9	....	....	††	....	....
Paralytic	12	13	9	8	10	9	9	11
Psittacosis	152	142	172	119	224	98	114	116
Rabies, animal	6,212	5,878	5,567	5,565	5,504	4,658	4,651	4,724
Rabies, human	–	2	3	1	–	1	–	1
Rheumatic fever, acute	137	88	117	90	147	141	158	144
Rocky Mountain spotted fever	976	1,126	838	714	760	604	609	623
Rubella (German measles)	2,325	970	752	630	551	306	225	396
Rubella, congenital syndrome	7	22	5	–	14	5	6	3
Salmonellosis, excluding typhoid fever	40,936	44,250	40,861	65,347	49,984	50,916	48,948	47,812
Shigellosis	18,129	19,719	17,371	17,057	17,138	23,860	30,617	25,010
Syphilis, primary and secondary	33,613	32,698	28,607	27,131	27,883	35,147	40,117	44,540
Total, all stages	75,579	74,637	69,888	67,563	68,215	86,545	103,437	110,797
Tetanus	88	91	74	83	64	48	53	53
Toxic-shock syndrome	†	502	482	384	412	372	390	400
Trichinosis	115	45	68	61	39	40	45	30
Tuberculosis	25,520	23,846	22,255	22,201	22,768	22,517	22,436	23,495
Tularemia	275	310	291	177	170	214	201	152
Typhoid fever	425	507	390	402	362	400	436	460
Varicella (chickenpox)	167,423	177,462	221,983	178,162	183,243	213,196	192,857	185,441
Yellow fever	....	....	....	....	§§	....	....	....

NOTE. Data in the annual *Summary of Notifiable Diseases* might not match data in other CDC surveillance reports because of differences in the timing of reports, the source of the data, and the use of different case definitions.

*Acquired immunodeficiency syndrome

†Not previously nationally notifiable.

§Not reported as distinct categories during this period.

¶Beginning in 1984, data reflect change in categories for tabulating encephalitis reports that were recorded by date of record to state health departments. Data for previous years are from surveillance records reported by onset date.

**Beginning in 1982, data were recorded by date of report to the state health department Data for 1976–1981 are from surveillance records reported by onset date

††Categories other than paralytic are no longer reported.

TABLE 4. NOTIFIABLE DISEASES — Summary of reported cases, United States, 1974–1981

Disease	1974	1975	1976	1977	1978	1979	1980	1981
Amebiasis	2,743	2,775	2,906	3,044	3,937	4,107	5,271	6,632
Anthrax	2	2	2	–	6	–	1	–
Aseptic meningitis	3,197	4,475	3,510	4,789	6,573	8,754	8,028	9,547
Botulism, total (including wound and unsp.)	28	20	55	129	105	45	89	103
Brucellosis	240	310	296	232	179	215	183	185
Chancroid	945	700	628	455	521	840	788	850
Cholera	–	–	–	3	12	1	9	19
Diphtheria	272	307	128	84	76	59*	3	5
Encephalitis, primary	1,164	4,064	1,651	1,414	1,351	1,504	1,362	1,492
Post-infectious	218	237	175	119	78	84	40	43
Gonorrhea	906,121	999,937	1,001,994	1,002,219	1,013,436	1,004,058	1,004,029	990,864
Granuloma inguinale	47	60	71	75	72	76	51	66
Hansen disease (leprosy)	118	162	145	151	168	185	223	256
Hepatitis A	40,358	35,855	33,288	31,153	29,500	30,407	29,087	25,802
Hepatitis B	10,631	13,121	14,973	16,831	15,016	15,452	19,015	21,152
Hepatitis, unspecified	8,351	7,158	7,488	8,639	8,776	10,534	11,894	10,975
Legionellosis	†. . . .	.	235	359	761	593	475	408
Leptospirosis	68	93	73	71	110	94	85	82
Lymphogranuloma venereum	394	353	365	348	284	250	199	263
Malaria	293	373	471	547	731	894	2,062	1,388
Measles (rubeola)	22,094	24,374	41,126	57,345	26,871	13,597	13,506	3,124
Meningococcal disease	1,346	1,478	1,605	1,828	2,505	2,724	2,840	3,525
Mumps	59,128	59,647	38,492	21,436	16,817	14,225	8,576	4,941
Murine typhus fever	26	41	69	75	46	69	81	61
Pertussis (whooping cough)	2,402	1,738	1,010	2,177	2,063	1,623	1,730	1,248
Plague	8	20	16	18	12	13	18	13
Poliomyelitis, total	7	13	10	19	8	22	9	10
Paralytic[§]	7	13	10	19	8	22	9	10
Psittacosis	164	49	78	94	140	137	124	136
Rabies, animal	3,151	2,627	3,073	3,130	3,254	5,119	6,421	7,118
Rabies, human	–	2	2	2	4	4	–	2
Rheumatic fever, acute	2,431	2,854	1,865	1,738	851	629	432	264
Rocky Mountain spotted fever	754	844	937	1,153	1,063	1,070	1,163	1,192
Rubella (German measles)	11,917	16,652	12,491	20,395	18,269	11,795	3,904	2,077
Rubella, congenital syndrome	45	30	30	23	30	62	50	19
Salmonellosis, excluding typhoid fever	21,980	22,612	22,937	27,850	29,410	33,138	33,715	39,990
Shigellosis	22,600	16,584	13,140	16,052	19,511	20,135	19,041	19,859
Syphilis, primary and secondary	25,385	25,561	23,731	20,399	21,656	24,874	27,204	31,266
Total, all stages	83,771	80,356	71,761	64,621	64,875	67,049	68,832	72,799
Tetanus	101	102	75	87	86	81	95	72
Trichinosis	120	252	115	143	67	157	131	206
Tuberculosis[¶]	30,122	33,989	32,105	30,145	28,521	27,669	27,749	27,373
Tularemia	144	129	157	165	141	196	234	288
Typhoid fever	437	375	419	398	505	528	510	584
Varicella (chickenpox)	141,495	154,248	183,990	188,396	154,089	199,081	190,894	200,766
Yellow fever	. . . . .	. . . . .	. . . . .	. . . . .	. . . . .	. . . . .	. . . . .	. . . . .

NOTE: Data in the annual *Summary of Notifiable Diseases* might not match data in other CDC surveillance reports because of differences in the timing of reports, the source of the data, and the use of different case definitions.

*Cutaneous diphtheria is no longer nationally notifiable

†Not previously nationally notifiable.

§No cases of paralytic poliomyelitis caused by wild virus have been reported in the United States since 1979.

¶Case data subsequent to 1974 are not comparable with earlier years because of changes in reporting criteria that became effective in 1975

**Last indigenous case of yellow fever was reported in 1911; before 1996, the last imported case was reported in 1924.

TABLE 5. NOTIFIABLE DISEASES — Summary of reported cases, United States, 1966–1973

Disease	1966	1967	1968	1969	1970	1971	1972	1973
Amebiasis	2,921	3,157	3,005	2,915	2,888	2,752	2,199	2,235
Anthrax	5	2	3	4	2	5	2	2
Aseptic meningitis	3,058	3,082	4,494	3,672	6,480	5,176	4,634	4,846
Botulism	9	5	7	16	12	25	22	34
Brucellosis	262	265	218	235	213	183	196	202
Chancroid	838	784	845	1,104	1,416	1,320	1,414	1,165
Cholera	–	–	–	–	–	1	–	1
Diphtheria	209	219	260	241	435	215	152	228
Encephalitis, primary	2,121	1,478	1,781	1,613	1,580	1,524	1,059	1,613
Post-infectious	964	1,060	502	304	370	439	243	354
Gonorrhea	351,738	404,836	464,543	534,872	600,072	670,268	767,215	842,621
Granuloma inguinale	148	154	156	154	124	89	81	62
Hansen disease (leprosy)	109	81	123	98	129	131	130	146
Hepatitis A (infectious)	32,859	38,909	45,893	48,416	56,797	59,606	54,074	50,749
Hepatitis B (serum)	1,497	2,458	4,829	5,909	8,310	9,556	9,402	8,451
Leptospirosis	72	67	69	89	47	62	41	57
Lymphogranuloma venereum	308	371	485	520	612	692	756	408
Malaria	565	2,022	2,317	3,102	3,051	2,375	742	237
Measles (rubeola)	204,136	62,705	22,231	25,826	47,351	75,290	32,275	26,690
Meningococcal disease	3,381	2,161	2,623	2,951	2,505	2,262	1,323	1,378
Mumps	.....	..*	152,209	90,918	104,953	124,939	74,215	69,612
Murine typhus fever	33	52	36	36	27	23	18	32
Pertussis (whooping cough)	7,717	9,718	4,810	3,285	4,249	3,036	3,287	1,759
Plague	5	3	3	5	13	2	1	2
Poliomyelitis, total	113	41	53	20	33	21	31	8
Paralytic	106	40	53	18	31	17	29	7
Psittacosis	50	41	43	57	35	32	52	33
Rabies, animal	4,178	4,481	3,591	3,490	3,224	4,310	4,369	3,640
Rabies, human	1	2	1	1	3	2	2	1
Rheumatic fever, acute	4,472	3,985	3,470	3,229	3,227	2,793	2,614	2,560
Rocky Mountain spotted fever	268	305	298	498	380	432	523	668
Rubella (German measles)	46,975	46,888	49,371	57,686	56,552	45,086	25,507	27,804
Rubella, congenital syndrome	11	10	14	31	77	68	42	35
Salmonellosis, excluding typhoid fever	16,841	18,120	16,514	18,419	22,096	21,928	22,151	23,818
Shigellosis	11,888	13,474	12,180	11,946	13,845	16,143	20,207	22,642
Streptococcal sore throat and scarlet fever	427,752	453,351	435,013	450,008	433,405	.....	†.	.....
Syphilis, primary and secondary	21,414	21,053	19,019	19,130	21,982	23,783	24,429	24,825
Total, all stages	105,159	102,581	96,271	92,162	91,382	95,997	91,149	87,469
Tetanus	235	263	178	192	148	116	128	101
Trichinosis	115	66	77	215	109	103	89	102
Tuberculosis	47,767	45,647	42,623	39,120	37,137	35,217	32,882	30,998
Tularemia	208	184	186	149	172	187	152	171
Typhoid fever	378	396	395	364	346	407	398	680
Varicella (chickenpox)	.....	.....	.....	.....	.....	.....	164,114	182,927
Yellow fever	.....	.....	.....	.....	.....	.....	.....	.....

NOTE: Data in the annual *Summary of Notifiable Diseases* might not match data in other CDC surveillance reports because of differences in the timing of reports, the source of the data, and the use of different case definitions.

* Not previously nationally notifiable.

† No longer nationally notifiable.

§ Last indigenous case of yellow fever was reported in 1911; before 1996, the last imported case was reported in 1924

TABLE 6. NOTIFIABLE DISEASES — Deaths from selected diseases, United States, 1987–1996

Cause of Death	ICD*	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
AIDS†	*042-*044	13,468	16,602	22,082	25,188	29,555	33,566	37,267	42,114	43,115	31,130
Amebiasis	006	9	7	4	5	5	6	6	2	4	4
Anthrax	022	–	–	–	–	–	–	–	–	–	–
Aseptic meningitis	047.9	28	37	36	50	47	37	33	30	22	25
Botulism, foodborne	005.1	–	1	2	4	2	1	–	–	2	1
Brucellosis	023	1	2	–	–	–	–	1	–	1	–
Chancroid	099.0	–	–	–	–	1	–	–	–	–	–
Cholera	001	1	–	–	2	2	2	–	1	–	2
Diphtheria	032	1	–	–	1	–	1	–	–	1	–
Encephalitis, Eastern equine	062.2	–	–	1	1	1	1	1	–	1	1
Encephalitis, California	062 5	1	–	–	–	–	–	–	–	–	1
Encephalitis, St. Louis	062.3	2	–	–	13	9	2	1	3	6	–
Encephalitis, Western equine	062.1	1	–	–	–	–	–	–	–	–	–
Gonococcal infections	098	7	3	4	3	3	4	5	3	3	4
Granuloma inguinale	099.2	–	–	–	–	–	–	–	–	–	–
Haemophilus influenzae, invasive	041 5	25	25	16	16	17	16	7	5	12	7
Hansen disease (leprosy)	030	1	–	4	3	–	2	1	3	2	–
Hepatitis, viral, infectious (Hep A)	070.0, 070.1	77	70	88	76	71	82	95	97	142	121
Hepatitis, viral, serum (Hep B)	070.2, 070.3	595	621	711	816	912	903	1,041	1,120	1,027	1,082
Hepatitis, viral, other and unsp.	070.4-070.9	510	599	717	686	857	1,016	1,353	1,844	2,231	2,577
Leptospirosis	100	1	2	–	2	1	2	1	–	2	2
Lymphogranuloma venereum	099 1	–	–	2	2	1	–	2	–	–	–
Malaria	084	5	7	11	3	4	8	12	3	8	4
Measles (rubeola)	055	2	3	32	64	27	4	–	–	2	1
Meningococcal disease	036	258	278	273	215	198	201	260	276	273	290
Mumps	072	2	2	3	1	1	–	–	–	–	1
Murine typhus fever	081.0	–	–	1	–	–	–	–	–	–	–
Pertussis (whooping cough)	033	1	4	12	12	–	5	7	8	6	4
Plague	020	1	–	–	–	–	1	2	2	1	2
Poliomyelitis, total	045.0-045.9	–	1	–	–	1	–	–	–	1	–
Psittacosis	073	2	1	1	2	–	4	1	–	–	1
Rabies, human	071	1	–	1	1	3	1	1	3	3	3
Rheumatic fever, acute	390-392	42	76	70	66	89	100	153	191	159	114
Rubella (German measles)	056	–	1	4	8	1	1	–	–	1	–
Salmonellosis, incl. paratyphoid fever	002 1-002 9, 003	105	66	99	80	53	47	52	49	66	58
Shigellosis	004	13	8	16	10	10	8	5	13	8	5
Spotted fevers	082.0	21	20	10	20	13	13	5	9	8	6
Syphilis	090-097	98	85	105	106	93	91	80	79	65	73
Tetanus	037	16	17	9	11	11	9	11	9	5	1
Trichinosis	124	–	–	1	–	–	–	–	–	–	–
Tuberculosis (all forms)	010-018	1,755	1,921	1,970	1,810	1,713	1,705	1,631	1,478	1,336	1,202
Tularemia	021	4	2	1	1	2	3	–	–	2	–
Typhoid fever	002.0	2	–	–	1	1	–	–	1	–	1
Varicella (chickenpox)§	052	89	83	89	120	81	100	100	124	115	81
Yellow fever	01060	–	–	–	–	–	–	–	–	–	1

NOTE: Data in the annual *Summary of Notifiable Diseases* might not match data in other CDC surveillance reports because of differences in the timing of reports, the source of the data, and the use of different case definitions.

* Numbers in ICD column refer to the category numbers listed in the *International Classification of Diseases, Ninth Revision, 1975* (The asterisks in the ICD column pertain to the ICD code, not a footnote. They indicate that the numbers are not part of the ICD but were introduced for use in the United States.)

† Acquired immunodeficiency syndrome.

§ Varicella was taken off the nationally notifiable disease list in 1991 Many states continue to report these cases to CDC

Source. National Center for Health Statistics System, 1987–1996. Deaths are classified to the *ICD Ninth Revision.*

Bibliography

General

Niskar AS, Koo D. Differences in notifiable infectious disease morbidity among adult women — United States, 1992–1994. J Womens Health 1998;7:451–8.

CDC. Case definitions for infectious conditions under public health surveillance. MMWR 1997;46(No. RR-10). Available at <http://www.cdc.gov/epo/dphsi/casedef/cover97.htm>. Accessed November 19, 1998.

CDC. Sexually transmitted disease surveillance, 1996. Atlanta: US Department of Health and Human Services, Public Health Service, CDC, 1997.

CDC. Demographic differences in notifiable infectious disease morbidity—United States, 1992–1994. MMWR 1997;46:637–41.

CDC. National electronic telecommunications system for surveillance. Informational brochure July 1996. Available at <http://www.cdc.gov/epo/mmwr/other/netss/netss.html>. Accessed November 19, 1998.

CDC. Notifiable disease surveillance and notifiable disease statistics—United States, June 1946 and June 1996. MMWR 1996;45:530–7.

Koo D, Wetterhall S. History and current status of the National Notifiable Diseases Surveillance System. J Public Health Management and Practice 1996;2:4–10.

CDC. Ten leading nationally notifiable infectious diseases—United States, 1995. MMWR 1996;45:883–4.

Benenson AS. Control of communicable diseases in man. 16th ed. Washington, DC: American Public Health Association, 1995.

Martin SM, Bean NH. Data management issues for emerging diseases and new tools for managing surveillance and laboratory data. Emerg Infect Dis J 1995;1:124–8.

CDC. Manual of procedures for the reporting of nationally notifiable diseases to CDC. Atlanta: US Department of Health and Human Services, Public Health Service, CDC, 1995.

Teutsch SM, Churchill RE, eds. Principles and practice of public health surveillance. New York: Oxford University Press, 1994.

Thacker SB, Stroup DF. Future directions for comprehensive public health surveillance and health information systems in the United States. Am J Epidemiol 1994;140:383–97.

CDC. Use of race and ethnicity in public health surveillance. MMWR 1993;42(No. RR-10).

CDC. Mandatory reporting of infectious diseases by clinicians, and mandatory reporting of occupational diseases by clinicians. MMWR 1990;39(No. RR-9).

Thacker SB, Choi K, Brachman PS. The surveillance of infectious diseases. JAMA 1983;249:1181–5.

AIDS

CDC. Diagnosis and reporting of HIV and AIDS in states with integrated HIV and AIDS surveillance—United States, January 1994–June 1997. MMWR 1998;47:309–14.

CDC. Update: perinatally acquired HIV/AIDS—United States, 1997. MMWR 1997;46:1086–92.

CDC. Update: trends in AIDS incidence—United States, 1996. MMWR 1997;46:861–7.

CDC. HIV/AIDS Surveillance report—year-end edition. 1997;9(2).

Arboviral Infections (California serogroup viruses, eastern equine encephalitis, St. Louis encephalitis, western equine encephalitis)

CDC. Arboviral infections of the central nervous system—United States, 1996–1997. MMWR 1998;47:517–22.

Szumlas DE, Apperson CS, Hartig PC, Francy DB, Karabatsos N. Seroepidemiology of La Crosse virus infection in humans in western North Carolina. Am J Trop Med Hyg 1996;54:332–7.

Marfin AA, Bleed DM, Lofgren JP, et al. Epidemiologic aspects of a St. Louis encephalitis epidemic in Jefferson County, Arkansas, 1991. Am J Trop Med Hyg 1993;49:30–7.

Tsai TF. Arboviral infections: general considerations for prevention, diagnosis, and treatment in travelers. Seminars in Pediatric Infectious Diseases 1992;3:62–9.

Botulism

Angulo FJ, Getz J, Taylor JP, et al. A large outbreak of botulism: the hazardous baked potato. J Infect Dis 1998;178:172–7.

Shapiro RL, Hatheway C, Becher J, Swerdlow DL. Botulism surveillance and emergency response: a public health strategy for a global challenge. JAMA 1997;278:433–5.

Townes JM, Cieslak PR, Hatheway CL, et al. An outbreak of type A botulism associated with a commercial cheese sauce. Ann Intern Med 1996;125:558–63.

Chancroid

CDC. Chancroid detected by polymerase chain reaction—Jackson, Mississippi, 1994–1995. MMWR 1995;44:567,573–4.
DiCarlo RP, Armentor BS, Martin DH. Chancroid epidemiology in New Orleans men. J Infect Dis 1995;172:446–52.
CDC. Chancroid in the United States, 1981–1990: evidence for underreporting of cases. MMWR 1992;41(No. SS-3):57–61.

Chlamydia trachomatis Infection

CDC. *Chlamydia trachomatis* genital infections—United States, 1995. MMWR 1997;46:193–8.
Mertz KJ, Levine WC, Mosure DJ, Berman SM, Dorian KJ. Trends in the prevalence of chlamydial infections: the impact of community-wide testing. Sex Transm Dis 1997;24:169–75.
Mosure DJ, Berman S, Kleinbaum D, Halloran ME. Predictors of *Chlamydia trachomatis* infection among female adolescents: a longitudinal analysis. Am J Epidemiol 1996;144:997–1003.
CDC. Recommendations for the prevention and management of *Chlamydia trachomatis* infections, 1993. MMWR 1993;42(No. RR-12):1–39.

Cholera

Mahon BE, Mintz ED, Greene KD, Wells JG, Tauxe RV. Reported cholera in the United States, 1992–1994: a reflection of global changes in cholera epidemiology. JAMA 1996;276:307–12.
Wachsmuth IK, Blake PA, Olsvik O, eds. *Vibrio cholerae* and cholera: molecular to global perspectives. Washington, DC: American Society for Microbiology, 1994.
Blake PA. Epidemiology of cholera in the Americas. Gastroenterol Clin North Am 1993;22:639–60.
World Health Organization. Guidelines for cholera control. Geneva: World Health Organization, 1993.

Cryptosporidiosis

Kramer MH, Herwaldt BL, Craun GF, Calderon RL, Juranek DD. Surveillance for waterborne-disease outbreaks—United States, 1993–1994. MMWR 1996;45(No. SS-1).
Juranek DD. Cryptosporidiosis: sources of infection and guidelines for prevention. Clin Infect Dis 1995;21(suppl 1):S57–S61. Available at <http://www.cdc.gov/ncidod/diseases/crypto/sources.htm>. Accessed November 19, 1998.
CDC. Assessing the public health threat associated with waterborne cryptosporidiosis: report of a workshop. MMWR 1995;44(No. RR-6):1–19. Available at <ftp://ftp.cdc.gov/pub/Publications/mmwr/rr/rr4406.pdf>. Accessed November 19, 1998.

Cyclosporiasis

Soave R, Herwaldt BL, Relman DA. *Cyclospora*. Infect Dis Clin North Am 1998;12:1–12.
CDC. Update: outbreaks of cyclosporiasis—United States and Canada, 1997. MMWR 1997;46:521–3.
CDC. Outbreak of cyclosporiasis—northern Virginia–Washington, D.C.–Baltimore, Maryland, metropolitan area, 1997. MMWR 1997;46:689–91.
Herwaldt BL, Ackers ML, Cyclospora Working Group. An outbreak in 1996 of cyclosporiasis associated with imported raspberries. N Engl J Med 1997;336:1548–56.

Dengue

Rigau-Pérez JG, Gubler DJ, Vorndam AV, Clark GG. Dengue in travelers from the United States, 1986–1994. J Travel Med 1997;4:65–71.
Pinheiro FP, Corber SJ. Global situation of dengue and dengue haemorrhagic fever, and its emergence in the Americas. World Health Stat Q 1997;50:161–9.

Diphtheria

Bisgard K, Hardy I, Popovic T, et al. Respiratory diphtheria in the United States, 1980–1995. Am J Public Health 1998;88:787–91
Dittmann S. Epidemic diphtheria in the Newly Independent States of the former USSR—situation and lessons learned. Biologicals 1997;25:79–86.
CDC. Diphtheria acquired by U.S. citizens in the Russian Federation and Ukraine—1994. MMWR 1995;44:237,243–4

Drug-Resistant *Streptococcus pneumoniae*

Dowell SF. Principles of judicious use of antimicrobial agents for pediatric upper respiratory tract infections. Pediatrics 1998;101(suppl):S163–S184.

CDC. Prevention of pneumococcal disease: recommendations of the Advisory Committee on Immunization Practices. MMWR 1997;46(No. RR-8):1–24.

CDC. Defining the public health impact of drug-resistant *Streptococcus pneumoniae*: report of a working group. MMWR 1996;45(No. RR-1):1–20.

Butler JC, Hofmann J, Cetron MS, et al. The continued emergence of drug-resistant *Streptococcus pneumoniae* in the United States: an update from the Centers for Disease Control and Prevention's Pneumococcal Sentinel Surveillance System. J Infect Dis 1996;174:986–93.

Escherichia coli O157:H7, Hemolytic Uremic Syndrome

Bender JB, Hedberg CW, Besser JM, Boxrud DJ, MacDonald KL, Osterholm MT. Surveillance for *Escherichia coli* O157:H7 infections in Minnesota by molecular subtyping. N Engl J Med 1997;337:388–94.

Mahon BE, Griffin PM, Mead PS, Tauxe RV. Hemolytic uremic syndrome surveillance to monitor trends in infection with *Escherichia coli* O157:H7 and other shiga toxin-producing *E. coli*. Emerg Infect Dis 1997;3:409–12.

Slutsker L, Ries AA, Greene KD, Wells JG, Hutwagner L, Griffin PM. *Escherichia coli* O157:H7 diarrhea in the United States: clinical and epidemiologic features. Ann Intern Med 1997;126:505–13.

Boyce TG, Pemberton AG, Wells JG, Griffin PM. Screening for *Escherichia coli* O157:H7—a nationwide survey of clinical laboratories. J Clin Microbiol 1995;33:3275–7.

Gonorrhea

Fox KK, Knapp JS, Holmes KK, et al. Antimicrobial resistance in *Neisseria gonorrhoeae* in the United States, 1988–1994: the emergence of decreased susceptibility to the fluoroquinolones. J Infect Dis 1997;175:1396–403.

Gershman KA, Barrow JC. A tale of two sexually transmitted diseases: prevalences and predictors of chlamydia and gonorrhea in women attending Colorado family planning clinics. Sex Transm Dis 1996;23:481–8.

CDC. Surveillance for gonorrhea and primary and secondary syphilis among adolescents—United States, 1981–1991. MMWR 1993;42(No. SS-3):1–11.

Group A Streptococcal Disease

The Working Group on Prevention of Invasive Group A Streptococcal Infections. Prevention of invasive group A streptococcal disease among household contacts of case-patients: is prophylaxis warranted? JAMA 1998;279:1206–10.

CDC. Outbreak of invasive group A streptococcus associated with varicella in a childcare center—Boston, Mass. MMWR 1997;46:944–8.

Davies HD, McGeer A, Schwartz B, et al. A prospective, population-based study of invasive group A streptococcal infections, including toxic shock syndrome and the risk of secondary infections. N Engl J Med 1996;335:547–54.

Working Group on Severe Streptococcal Infections. Defining the group A streptococcal toxic shock syndrome: rationale and consensus definition. JAMA 1993;269:390–1.

Haemophilus influenzae (Invasive Disease)

Bisgard KM, Kao A, Leake J, Strebel PM, Perkins BA, Wharton M. *Haemophilus influenzae* invasive disease in the United States, 1994–1995: near disappearance of a child vaccine preventable disease. Emerg Infect Dis 1998;4:229–37

Schuchat A, Robinson K, Wenger JD, et al. Bacterial meningitis in the United States in 1995. New Engl J Med 1997;33:970–6.

Urwin G, Krohn JA, Deaver-Robinson K, et al. Invasive disease due to *Haemophilus influenzae* serogroup f: clinical and epidemiologic characteristics in the *H. influenzae* serotype b vaccine era. Clin Infect Dis 1996;22:1069–76.

CDC. Recommendations for the use of *Haemophilus* b conjugate vaccines and a combined diphtheria, tetanus, pertussis, and *Haemophilus* b vaccine: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1993;42(No. RR-13).

Hepatitis

CDC. Hepatitis surveillance report no. 56. Atlanta, GA: US Department of Health and Human Services, Public Health Service, CDC, 1996.

Alter MJ, Mares A, Hadler SC, Maynard JE. The effect of underreporting on the apparent incidence and epidemiology of acute viral hepatitis. Am J Epidemiol 1987;125:133–9.

Hepatitis A

Lemon SM, Shapiro CN. The value of immunization against hepatitis A. Infectious Agents and Disease 1994;1:38–49.

Shapiro CN, Coleman PJ, McQuillan GM, et al. Epidemiology of hepatitis A: seroepidemiology and risk groups in the U.S.A. Vaccine 1992;10(suppl 1):S59–S62.

Hepatitis B

Margolis HS, Alter MJ, Hadler SC. Hepatitis B: evolving epidemiology and implications for control. Semin Liver Dis 1991;11:84–92.

Hepatitis, C/Non-A, Non-B

CDC. Recommendations for prevention and control of hepatitis C virus (HCV) infection and HCV-related chronic disease. MMWR 1998;47(no. RR-19).

Alter MJ, Mast EE, Moyer LA, Margolis HS. Hepatitis C. Infect Dis Clin N Am 1998;12:13–26.

Alter MJ, Margolis HS, Krawczynski K, et al. The natural history of community-acquired hepatitis C in the United States. N Engl J Med 1992;327:1899–905.

Influenza A (H5N1)

Subbarao K, Klimov A, Katz J, et al. Characterization of an avian influenza A (H5N1) virus isolated from a child with a fatal respiratory illness. Science 1998;279:393–6.

Yuen KY, Chan PK, Peiris M, et al. Clinical features and rapid viral diagnosis of human disease associated with avian influenza A H5N1 virus. Lancet 1998;351:467–71.

CDC. Update: isolation of avian influenza A(H5N1) viruses from humans—Hong Kong, 1997–1998. MMWR 1998;46:1245–7.

Legionellosis

Fiore AE, Nuorti PJ, Levine OS, et al. Epidemic Legionnaires' disease two decades later: old sources, new diagnostic methods. Clin Infect Dis 1998;26:426–33.

Jernigan DB, Hofmann J, Cetron MS, et al. Outbreak of Legionnaires' disease among cruise ship passengers exposed to a contaminated whirlpool spa. Lancet 1996;347:494–9.

Keller DW, Hajjeh R, DeMaria A Jr, et al. Community outbreak of Legionnaires' disease: an investigation confirming the potential for cooling towers to transmit legionella species. Clin Infect Dis 1996;22:257–61.

Marston BJ, Lipman HB, Breiman RF. Surveillance for Legionnaires' disease: risk factors for morbidity and mortality. Arch Intern Med 1994;154:2417–22.

Lyme Disease

Dennis DT. Epidemiology, ecology, and prevention of Lyme disease. In: Rahn DW, Evans J, eds. Lyme disease. Philadelphia: American College of Physicians, 1998:7–34.

CDC. Lyme disease—United States, 1996. MMWR 1997:46:531–5.

CDC. Recommendations for test performance and interpretation from the Second National Conference on Serologic Diagnosis of Lyme disease. MMWR 1995;44:590–1.

Malaria

Lobel HO, Kozarsky PE. Update on prevention of malaria for travelers. JAMA 1997;278:1767–71.

Zucker JR. Changing patterns of autochthonous malaria transmission in the United States: a review of recent outbreaks. Emerg Infect Dis 1996;2:37–43.

Zucker JR, Campbell CC. Malaria: principles of prevention and treatment. Infect Dis Clin N Am 1993;7:547–67.

Measles

CDC. Measles, mumps and rubella—vaccine use and strategies for elimination of measles, rubella and congenital rubella syndrome and control of mumps: recommendations of the Advisory Committee on Immunization Practices. MMWR 1998;47(No. RR-7)1–48.

CDC. Measles—United States, 1997. MMWR 1998;47:273–6.

CDC. Measles—United States, 1996. MMWR 1997;46:242–6.

Meningococcal Disease

Rosenstein N, Levine O, Taylor JP, et al. Efficacy of meningococcal vaccine and barriers to vaccination. JAMA 1998;279:435–9.

Fischer M, Hedberg K, Cardosi P, et al. Tobacco smoke as a risk factor for meningococcal disease. Pediatr Infect Dis J 1997;16:979–83.

CDC. Control and prevention of meningococcal disease and control and prevention of serogroup C meningococcal disease: evaluation and management of suspected outbreaks: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1997;46(No. RR-5).

CDC. Laboratory-based surveillance for meningococcal disease in selected areas—United States, 1989–1991. MMWR 1993;42(No. SS-2):21–30.

Mumps

CDC. Mumps surveillance—United States, 1988–1993. MMWR 1995;44(No. SS-3):1–14.

Briss PA, Fehrs LJ, Parker RA, et al. Sustained transmission of mumps in a highly vaccinated population: assessment of primary vaccine failure and waning vaccine-induced immunity. J Infect Dis 1994;169:77–82.

Hersch BS, Fine PEM, Kent WK, et al. Mumps outbreak in a highly vaccinated population. J Pediatr 1991;119:187–93.

CDC. Mumps prevention. MMWR 1989;38:388–92,397–400.

Pertussis

Guris D, Bardenheier B, Brennan M, et al. Pertussis: a re-emerging disease among adolescents and adults in the U.S. [Abstract]. In: Proceedings of the International Conference on Emerging Infectious Diseases. Atlanta: CDC, CSTE, ASM and several others, 1998.

CDC. Manual for the surveillance of vaccine-preventable diseases. Atlanta: US Department of Health and Human Services, CDC, 1997. Available at <http://www.cdc.gov/nip/manual/vpd/vpd.htm>. Accessed November 19, 1998.

CDC. Pertussis vaccination: use of acellular pertussis vaccines among infants and young children: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1997;46(No. RR-7):1–25.

Plague

Gage KL. Plague. In: Hausler WJ, Sussman M, ed. 9th ed. Topley and Wilson's microbiology and microbial infections, Vol. 3, bacterial infections. London: Arnold 1998:885–903.

CDC. Prevention of plague: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1996;45(No. RR-14).

Poland JD, Quan TJ, Barnes AM. Plague. In: Beran GW, ed. CRC handbook series in zoonoses: section A — bacterial, rickettsial and mycotic diseases. Boca Raton, Florida: CRC Press, Inc., 1994:93–112.

Poliomyelitis

CDC. Paralytic poliomyelitis—United States, 1980–1994. MMWR 1997;46:79–83.

CDC. Poliomyelitis prevention in the United States: introduction of a sequential schedule of inactivated poliovirus vaccine followed by oral poliovirus vaccine–recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1997;46(No. RR-3):1–25.

Prevots DR, Strebel PM. Poliomyelitis prevention in the United States: new recommendations for routine childhood poliovirus vaccination place greater reliance on inactivated poliovirus vaccine. Pediat Ann 1997:26:378–83.

Psittacosis

Moroney JF, Guevara R, Iverson C, et al. Detection of chlamydiosis in a shipment of pet birds, leading to recognition of an outbreak of clinically mild psittacosis in humans. Clin Infect Dis 1998;26:1425–9.

Jorgensen DM. Gestational psittacosis in a Montana sheep rancher. Emerg Infect Dis 1997;3: 191–4

CDC. Compendium of psittacosis (chlamydiosis) control, 1997. MMWR 1997;46(No. RR-13):1–13.

Wong KH, Skelton SK, Daugharty H. Utility of complement fixation and microimmunofluorescence assays for detecting serologic responses in patients with clinically diagnosed psittacosis. J Clin Microbiol 1994;32:2417–21.

Rabies

CDC. Compendium of animal rabies control, 1998. MMWR 1998;47(No. RR-9).
Krebs JW, Smith JS, Rupprecht CE, Childs JE. Rabies surveillance in the United States during 1996. JAVMA 1997;211:1525–39.
CDC. Rabies prevention—United States, 1991: recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1991;40(No. RR-3):1–19.

Rocky Mountain Spotted Fever

Dalton MJ, Clarke MJ, Holman RC, et al. National surveillance for Rocky Mountain spotted fever, 1981–1992: epidemiologic summary and evaluation of risk factors for fatal outcome. Am J Trop Med Hyg 1995;52:405–13.
Salgo MP, Telzak EE, Currie B, et al. A focus of Rocky Mountain spotted fever within New York City. N Engl J Med 1988;318:1345–8.
Woodward TE. Rocky Mountain spotted fever: epidemiological and early clinical signs are keys to treatment and reduced mortality. J Infect Dis 1984;150:465–8.

Rubella

CDC. Rubella and congenital rubella syndrome—United States, 1994–1997. MMWR 1997;46: 350–4.
CDC. Rubella and congenital rubella syndrome—United States, January 1, 1991–May 7, 1994. MMWR 1994;43:391,397–401.
CDC. Rubella among crew members of commercial cruise ships. MMWR 1997;46:1247–50.

Salmonellosis

Mahon BE, Pönkä A, Hall WN, et al. An international outbreak of *Salmonella* infections caused by alfalfa sprouts grown from contaminated seeds. J Infect Dis 1997;175:876–82.
Mermin J, Hoar B, Angulo FJ. Iguanas and *Salmonella* marina infection in children: a reflection of the incidence of reptile-associated salmonellosis in the United States. Pediatrics 1997;99:399–402.
CDC. Multidrug-resistant *Salmonella* serotype Typhimurium—United States, 1996. MMWR 1997;46:308–10.
CDC. Outbreaks of *Salmonella* serotype Enteritidis infection associated with consumption of raw shell eggs—United States, 1994–1995. MMWR 1996;45:737–42.

Shigellosis

Sobel J, Cameron DN, Ismail J, et al. A prolonged outbreak of *Shigella sonnei* infections in traditionally observant Jewish communities in North America caused by a molecularly distinct bacterial subtype. J Infect Dis 1998;177:1405–8.
Mohle-Boetani JC, Stapleton M, Finger R, et al. Communitywide shigellosis: control of an outbreak and risk factors in child day-care centers. Am J Public Health 1995;85:812–6.
Ries AA, Wells JG, Olivola D, et al. Epidemic *Shigella dysenteriae* type 1 in Burundi: panresistance and implications for prevention. J Infect Dis 1994;169:1035–41.
Lee LA, Shapiro CN, Hargrett-Bean N, Tauxe RV. Hyperendemic shigellosis in the United States: a review of surveillance data for 1967–1988. J Infect Dis 1991;164:894–900.

Syphilis

St. Louis ME, Farley TA, Aral SO. Untangling the persistence of syphilis in the south. Sex Transm Dis 1996;23:1–4.
Nakashima AK, Rolfs RT, Flock ML, Kilmarx P, Greenspan JR. Epidemiology of syphilis in the United States, 1941–1993. Sex Transm Dis 1996;23:16–23.
CDC. Outbreak of primary and secondary syphilis—Baltimore City, Maryland, 1995. MMWR 1996;45:166–9.

Syphilis, Congenital

Risser WL, Hwang LY. Problems in the current case definitions of congenital syphilis. J Pediatr 1996;129:499–505.

Coles BF, Hipp SS, Silberstein GS, Chen JH. Congenital syphilis surveillance in upstate New York, 1989–1992: implications for prevention and clinical management. J Infect Dis. 1995;171:732–5.
CDC. Surveillance for geographic and secular trends in congenital syphilis—United States, 1983–1991. MMWR 1993;42(No. SS-6):59–71.
CDC. Guidelines for the prevention and control of congenital syphilis. MMWR 1988;37(No. S-1):1–13.

Tetanus

CDC. Tetanus surveillance—United States, 1995–1997. MMWR 1998;47(No. SS-2):1–13.
Craig AS, Reed GW, Mohon RT, et al. Neonatal tetanus in the United States: a sentinel event in the foreign-born. Pediatr Infect Dis J 1997;16:955–9.
CDC. Tetanus surveillance—United States, 1991–1994. MMWR 1997;46(No. SS-2):15–25.
Gergen PJ, McQuillan GM, Keily M, Ezzati-Rice TM, Sutter RW, Virella G. A population-based serologic survey of immunity to tetanus in the United States. N Engl J Med 1995;332:761–6.

Toxic-Shock Syndrome

Schuchat A, Broome CV. Toxic shock syndrome and tampons. Epidemiol Rev 1991;13:99–112.
CDC. Reduced incidence of menstrual toxic shock syndrome—United States, 1980–1990. MMWR 1990;39:421–3.
Gaventa S, Reingold AL, Hightower AW, et al. Active surveillance for toxic shock syndrome in the United States, 1986. Rev Infect Dis 1989;11(suppl):S28–S34.

Trichinellosis (Trichinosis)

CDC. Outbreak of trichinellosis associated with eating cougar jerky—Idaho, 1995. MMWR 1996;45:205–6.
McAuley JB, Michelson MK, Hightower AW, Engeran S, Wintermeyer LA, Schantz PM. A trichinosis outbreak among Southeast Asian refugees. Am J Epidemiol 1992;135:1404–10.
CDC. Trichinosis surveillance—United States, 1987–1990. MMWR 1991;40(No. SS-3):35–42.
Bailey TM, Schantz PM. Trends in the incidence and transmission patterns of human trichinosis in the United States, 1982–1986. Rev Infect Dis 1990;12:5–11.

Tuberculosis

CDC. Reported tuberculosis in the United States, 1997. Washington, DC: US Department of Health and Human Services, CDC, 1998.
CDC. Tuberculosis morbidity—United States, 1997. MMWR 1998;47:253–7.
CDC. Recommendations for counting reported tuberculosis cases. In: Reported tuberculosis in the United States, 1996. July 1997:61–8.
American Thoracic Society, CDC. Treatment of tuberculosis and tuberculosis infection in adults and children. Am J Respir Crit Care Med 1994;149:1359–74.

Typhoid Fever

Mermin JH, Townes JM, Gerber M, Dolan N, Mintz ED, Tauxe RV. Typhoid fever in the United States, 1985–1994: changing risks of international travel and increasing antimicrobial resistance. Arch Intern Med 1998;158:633–8.
CDC. Typhoid immunization: recommendations of the Advisory Committee on Immunization Practices. MMWR 1994;43(No. RR-14).
Woodruff BA, Pavia AT, Blake PA. A new look at typhoid vaccination: information for the practicing physician. JAMA 1991;265:756–9.

Varicella

CDC. Varicella-related deaths among children—United States, 1997. MMWR 1998;47:365–8.
CDC. Outbreak of invasive Group A Streptococcus associated with varicella in a childcare center—Boston, Massachusetts, 1997. MMWR 1997;46:944–9.
Izurieta HS, Strebel PM, Blake PA. Postlicensure effectiveness of varicella vaccine during an outbreak in a child care center. JAMA 1997;278:1495–9.
CDC. Prevention of varicella: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1996;45(No. RR-11).