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Contents

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Abbreviations

NCCDPHP	National Center for Chronic Disease Prevention and Health Promotion
NCEHIC	National Center for Environmental Health and Injury Control
NCID	National Center for Infectious Diseases
CIO	Centers/Institute/Offices
NCPS	National Center for Prevention Services
EPO	Epidemiology Program Office
NIOSH	National Institute for Occupational Safety and Health

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Waterborne-Disease Outbreaks, 1989–1990

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SUMMARY

For the 2-year period 1989-1990, 16 states reported 26 outbreaks due to water intended for drinking; an estimated total of 4,288 persons became ill in these outbreaks. Giardia lamblia *was implicated as the etiologic agent for seven of the 12 outbreaks in which an agent was identified. The outbreaks of giardiasis were all associated with ingestion of unfiltered surface water or surface-influenced groundwater. An outbreak with four deaths was attributed to* Escherichia coli *O157:H7, the only bacterial pathogen implicated in any of the outbreak investigations. An outbreak of remitting, relapsing diarrhea was associated with cyanobacteria (blue-green algae)-like bodies, whose role in causing diarrheal illness is being studied. Two outbreaks due to hepatitis A and one due to a Norwalk-like agent were associated with use of well water. Eighteen states reported a total of 30 outbreaks due to the use of recreational water, which resulted in illness for an estimated total of 1,062 persons. These 30 reports comprised 13 outbreaks of whirlpool- or hot tub-associated* Pseudomonas *folliculitis; 13 outbreaks of swimming-associated gastroenteritis, including five outbreaks of shigellosis; one outbreak of hepatitis A associated with a swimming pool; and three cases of primary amebic meningoencephalitis caused by* Naegleria. *The national surveillance of outbreaks of waterborne diseases, which has proceeded for 2 decades, continues to be a useful means for characterizing the epidemiology of waterborne diseases.*

INTRODUCTION

The reporting of waterborne-disease outbreaks (WBDOs) is voluntary in the United States. Information on the occurrence and causes of WBDOs is available from 1920 onward (*1*). Since 1971, CDC, in collaboration with the Environmental Protection Agency (EPA), has tabulated data concerning WBDOs separately from those for foodborne-disease outbreaks and has compiled these data in surveillance summaries. The two most recent surveillance reports summarized data from 1986 to 1988 and from 1985 (*2,3*). In 1989, responsibility for the surveillance system shifted within the National Center for Infectious Diseases, CDC, from the Division of Bacterial and Mycotic Diseases, Enteric Diseases Branch, to the Division of Parasitic Diseases, Parasitic Diseases Branch, primarily because of the prominent role of *Giardia lamblia* as an etiologic agent in WBDOs. This surveillance summary includes data for outbreaks in 1989 and 1990 and also for previously unreported outbreaks in 1988.

In addition to WBDOs associated with water intended for drinking, the surveillance summaries include data about a) outbreaks associated with exposure to water used for recreational purposes and b) outbreaks of gastroenteritis (whether waterborne or foodborne) on ocean-going passenger vessels that call on ports in the United States.

CDC's activities related to waterborne diseases have the following goals: a) to characterize the epidemiology of waterborne diseases; b) to identify the deficiencies in water systems and the etiologic agents that are associated with outbreaks, so that improved water systems can be designed; c) to teach public health personnel how to investigate WBDOs; and d) to collaborate with local, state, and other federal (e.g., EPA) and international agencies on initiatives to prevent waterborne diseases.

State health departments can request CDC and EPA to provide epidemiologic assistance in investigating WBDOs. In addition, CDC and EPA can be consulted about the engineering and environmental aspects of water treatment and about collecting large-volume water samples to identify viruses, parasites, and pathogenic bacteria.

METHODS

Sources of Data

State health departments report WBDOs to CDC on a standard form, which was revised in 1991 (CDC Form 52.12, Rev. 02-91; Figure 1). In November 1990, CDC personnel sent a letter to state and territorial epidemiologists requesting reports of all previously unreported outbreaks; many states had designated a person to coordinate the surveillance of WBDOs. States that did not respond to the letter were contacted by telephone. In addition, personnel from the Health Effects Research Laboratory of EPA contacted state water-supply agencies to obtain information about WBDOs.

As part of their request to enter a port, vessel masters of cruise ships must report the number of persons who visited the ship's physician because of diarrheal illness during the voyage. If 3% or more of the passengers on a 1-week voyage reported diarrheal illness, a quarantine officer and a member of CDC's Vessel Sanitation Program board and inspect the ship, and an epidemiologic investigation may be conducted. Data from these investigations are summarized here.

Definitions of terms

The surveillance system for WBDOs, like the system for foodborne-disease outbreaks, is unusual in that the unit of analysis is an outbreak of any of a variety of

waterborne diseases, rather than an individual case of a particular disease. The system's definition for a WBDO comprises two criteria: a) at least two persons must have experienced a similar illness after ingesting or using water intended for drinking or after being exposed to or unintentionally ingesting or inhaling fresh or marine water used for recreational purposes, and b) epidemiologic evidence must implicate the water as the source of the illness. The stipulation that at least two persons be ill is waived for single cases of chemical poisoning, if laboratory studies indicate that water was contaminated by the chemical, and for single cases of laboratory-confirmed primary amebic meningoencephalitis. If primary and secondary cases are distinguished on the outbreak report form, only primary cases are included in the case counts on the line listings.

Whirlpool- and hot tub-associated outbreaks of folliculitis due to *Pseudomonas* are included in the surveillance system, but wound infections caused by water-related organisms, such as *Aeromonas* species, are not. Outbreaks of Pontiac fever associated with whirlpools are listed, but outbreaks of Legionnaires' disease traditionally have not been included.

Community water systems are defined as public or investor-owned systems that serve large or small communities, subdivisions, or trailer parks with at least 15 service connections or 25 year-round residents. Noncommunity water systems serve institutions, industries, camps, parks, hotels, or service stations that may be used by the general public. Community and noncommunity water systems are classified as public water systems and are regulated under the Safe Drinking Water Act (PL 93-523) of 1974. Approximately 189,600 water systems in the United States are classified as public water systems (*4*); 31% of these are community water systems, which serve 91% of the U.S. population, and 69% are noncommunity water systems. The remaining water systems, which serve 9% of the U.S. population, are nonpublic or individual systems used by one or several residences or by persons traveling outside populated areas; they are generally wells or springs.

Deficiencies in water systems are classified as follows: 1 = untreated surface water (e.g., from rivers, streams, lakes, or reservoirs); 2 = untreated groundwater (e.g., from wells or springs); 3 = treatment deficiency (e.g., temporary interruption of disinfection, chronically inadequate disinfection, no filtration, or inadequate filtration); 4 = distribution system deficiency (e.g., a cross-connection, back siphonage, contamination of water mains during construction or repair, or contamination of a storage facility); and 5 = unknown or miscellaneous deficiency. In this surveillance summary, no outbreaks with miscellaneous deficiencies were reported. If more than one deficiency was reported for an outbreak, only the deficiency judged to be the most important is noted on the line listing (Tables 1 and 2).

Interpretation of Data

The data in this surveillance summary should be interpreted with caution; they cannot be used to determine the true incidence of WBDOs or the relative incidence of outbreaks of various etiologies. Presumably only a fraction of the actual total number of outbreaks is reported to CDC and EPA, but the extent of underreporting is unknown.

The likelihood that individual cases of illness will be epidemiologically linked and attributed to exposure to water varies considerably among locales and is dependent on such factors as consumer awareness, physician interest, and surveillance activities

of state and local health and environmental agencies. Therefore, the states that recognize and report the most WBDOs are not necessarily the states with the most outbreaks. Large outbreaks and those involving serious illnesses are most likely to come to the attention of health authorities. Outbreaks in community water systems are more likely to be reported than those in noncommunity water systems, since the latter serve primarily nonresidential areas and transient populations. Outbreaks in individual systems are the most underreported because they generally involve small numbers of persons.

The proportion of the total number of reported outbreaks that is attributed to a particular etiologic agent depends in part on the interests and expertise of the investigators. For example, epidemiologists and microbiologists interested in particular organisms, such as *Giardia lamblia* or Norwalk-like agents, are likely to obtain the necessary specimens and perform the necessary laboratory procedures to confirm the causative agent. Furthermore, the proportion of the total number of reported cases of waterborne diseases that is attributed to a particular etiologic agent can be substantially changed by the occurrence of a few large outbreaks due to that agent. The number of cases reported for an outbreak generally is an approximate figure; the method and accuracy of the approximation vary among outbreaks.

An outbreak is included in the surveillance summary if the data on the report form suggest that the outbreak was caused by exposure to contaminated water. The quality of the data implicating water, however, varies widely among the outbreaks. Factors influencing the quality of the data include the health department's budgetary, investigative, and laboratory resources and the timing of the investigation with respect to the course of the outbreak. Delayed recognition of outbreaks is a major impediment to timely investigations.

This surveillance summary incorporates a new classification system (Table 3) that indicates the strength of the evidence implicating water for each outbreak in the line listings (Tables 1, 2, and 4). Each outbreak (except outbreaks of *Pseudomonas* folliculitis and individual cases of primary amebic meningoencephalitis) was assigned a classification number (I through IV) that reflects both the kind of epidemiologic data and the presence or absence of water quality data on the outbreak report form. Epidemiologic data were given preeminence over water quality data; outbreaks without supporting epidemiologic data were not included in the surveillance summary, whereas some outbreaks without water quality data were included. The intent of the classification system is to provide the reader with additional information about the data that were available to implicate water. Classification numbers of II, III, and IV are not intended to reflect badly on the investigators, since not all outbreaks could or should have been investigated rigorously. On the other hand, a classification number of I does not necessarily imply that the investigation was optimal, but simply that both epidemiologic and water quality data implicated water.

RESULTS

Outbreaks due to Water Intended for Drinking

For the 2-year period 1989–1990, 16 states reported a total of 26 outbreaks due to water intended for drinking, which resulted in illness for an estimated total of 4,288 persons. Twenty (77%) outbreak investigations were classified as Class I (i.e., adequate epidemiologic and water-quality data were provided on the report form).

The individual outbreaks are listed by state in Table 1; in Table 5, data are summarized by etiologic agent and type of water system. The median size of the outbreaks was 54 persons (range, 3-1,000). Ten (38%) of the outbreaks were reported from three states: Pennsylvania (four), Missouri (three), and New York (three). Although Pennsylvania reported the most outbreaks, all were relatively small (the largest affected 63 persons). Overall, 12 outbreaks were reported for 1989 and 14 for 1990. Outbreaks began in each month except January and October; six (23%) began in July (Figure 2).

Giardia lamblia was implicated as the etiologic agent for seven of the 12 outbreaks for which an agent was identified. Outbreaks of waterborne giardiasis, which affected an estimated 697 persons, were reported from New York (three), Colorado (two), Vermont (one), and Alaska (one); they began in February (one), March (two), April (one), June (one), July (one), and August (one). *Giardia* was identified in stool specimens from ill persons for all seven outbreaks and also in water samples for four of the outbreaks. Four outbreaks were associated with community water systems and three with noncommunity water systems. Surface water supplies were implicated for six outbreaks, and in the other, the water source was a spring that was vulnerable to contamination above ground because of land erosion. Six outbreaks were associated with treatment deficiencies (chlorination that was not cysticidal; lack of filtration); the other outbreak, which occurred at a lodge, was associated with untreated river water that was used because the usual source of water (well water) was frozen.

The only outbreak attributed to a bacterial pathogen was also the outbreak associated with the most severe illness. A large outbreak (243 cases) due to *Escherichia coli* O157:H7, which had not previously been established to be a waterborne pathogen, occurred in a Missouri community (*5*). One-third of the ill persons had bloody diarrhea, 32 were hospitalized, two had hemolytic uremic syndrome, and four died. Multiresistant *E. coli* O157:H7 was isolated from stool specimens from ill persons, but not from water samples. However, an epidemiologic investigation implicated the community water system as the source of infection. Although unchlorinated well water was used by the community, the water distribution network was the likely source of contamination. Shortly before the outbreak peaked, two large broken water transmission pipes were repaired and more than 40 water meters were replaced without disinfection of the water system at the points of repair or replacement.

The first outbreak in this surveillance system associated with cyanobacteria (blue-green algae)-like bodies (CLB) occurred in a hospital in Chicago (*6*). Ill persons had remissions and relapses of explosive watery diarrhea. Light microscopic examination of stool specimens from ill persons demonstrated the presence of CLB, but no ova or parasites, and cultures were negative for common bacterial pathogens. Illness was associated with drinking water in a building for which open-air rooftop storage tanks were used to maintain water pressure. Before the outbreak, failure of the water pump used to fill the tanks caused both of the tanks to empty. Although CLB per se were not found in samples of the water, algae (primarily diatoms) were found in a storage tank.

Serologic testing implicated viral pathogens for three outbreaks associated with the use of well water. Hepatitis A was implicated in investigations of two outbreaks in Pennsylvania. One water supply, which was chlorinated, had a chlorinator malfunction; the other was untreated well water that was found to be contaminated

with fecal coliforms. Well-water samples were not tested for the presence of hepatitis A virus; however, the virus was recovered from well water in the investigation of a similar outbreak in 1982 in Georgia (*7*). A Norwalk-like agent was implicated as the cause of an outbreak that affected an estimated 900 persons at a new resort in Arizona; the outbreak was caused by effluent from the sewage treatment facility seeping directly into the resort's deep well through cracks in the subsurface rock (*8*).

In 14 (54%) outbreaks, the etiology of acute gastrointestinal illness (AGI) was not determined; many of these outbreaks had features (e.g., symptom complex, incubation period, and duration of illness) suggestive of viral causes. For eight of these outbreaks, stool specimens from ill persons were examined and were found to be negative for bacterial pathogens (all eight outbreaks) and parasites (two outbreaks); no specimens were examined for viruses. Coliforms were detected during investigations of the water systems for 12 of the 14 outbreaks. The largest of these outbreaks, in which an estimated 1,000 persons became ill over a period of several months, occurred at a country club in Tennessee whose two wells were within 20 feet of a septic tank; the well water was heavily contaminated with coliforms.

Overall, noncommunity water systems accounted for 46% of the 26 outbreaks and 58% of the 4,288 cases, whereas community water systems accounted for 42% of the outbreaks and 39% of the cases (Tables 5 and 6 and Figure 3). Comparable percentages of outbreaks associated with noncommunity (42%) and community (36%) water systems occurred during summer months.

Thirteen (50%) of the 26 outbreaks were associated with well water (Figure 3). Distribution system deficiencies caused two (15%) of the 13 outbreaks. The well water was untreated in five (38%) outbreaks. Of the six (46%) outbreaks associated with inadequate or interrupted disinfection, chlorine was the disinfectant used by four of the water systems, and iodine and ultraviolet light were each used by one.

Three (12%) of the 26 outbreaks were associated with spring water; in two, the spring water emerged above ground and therefore was vulnerable to contamination, and in the other, untreated spring water was used to augment the usual water supply. Of the 10 (38%) outbreaks associated with the use of surface water, six (60%) were associated with treatment deficiencies; five of the water systems provided chlorination as the only treatment, and one provided both chlorination and filtration. Of the remaining four systems, two (20%) were associated with the use of untreated water, one (10%) with a distribution system deficiency, and one (10%) with an unknown deficiency.

One possible outbreak in 1989 was not included in the line listing because of limited data, but illness may have been caused by contaminated bottled water. Two persons, both of whom lived in the same household in Idaho and drank bottled water exclusively, became ill; stool specimens were not provided, nor was an epidemiologic investigation conducted. The ill persons stopped drinking the water after noting foreign objects in it and felt better within a few days. Water samples from previously unopened bottles had high standard plate counts, and diatoms were present. The water, which came from a river in Washington State, was chlorinated at a municipal plant and was passed through charcoal filters in the milk plant where it was bottled. However, the charcoal filters were bypassed on the day that the relevant water was bottled because they were clogged with diatoms.

Data regarding 12 other possible WBDOs reported to CDC were not included in this surveillance summary. In eight, either no supporting epidemiologic data or inade-

quate data were provided (i.e., the outbreak investigations did not meet the criteria for Class I, II, III, or IV); in two, water may have been contaminated at its point of use rather than at its source or in its distribution system (e.g., ice or water may have been contaminated by a sick handler); and in two, a foodborne etiology was as likely or more likely than a waterborne etiology.

Outbreaks Associated with Recreational Water Use

For the 2-year period 1989–1990, 18 states reported a total of 30 outbreaks associated with water used for recreational purposes; 14 outbreaks were reported for 1989 and 16 for 1990 (Table 4). Outbreaks began in each month except November; 10 (33%) began in July (Figure 2). The outbreaks, which caused illness for an estimated total of 1,062 persons, affected a median of 10 persons (range, 1-300). Of the 30 reported outbreaks, 13 were of folliculitis; another 13, gastroenteritis, five of which were shigellosis; one, hepatitis A; and three were individual cases of primary amebic meningoencephalitis.

The 13 outbreaks of folliculitis, which affected from two to 300 persons, were associated with the use of whirlpools or hot tubs; in two of the outbreaks, ill persons had used both a whirlpool and a swimming pool, and thus these two possible sources could not be differentiated. Only one of the 13 outbreaks occurred during the summer. In eight of the outbreak investigations, *Pseudomonas* was confirmed to be the etiologic agent; in the other five, the clinical syndrome was consistent with this etiology. In six of the investigations, low chlorine or bromine concentrations in the water were documented, and in one, a low water pH was also demonstrated.

Shigella sonnei was implicated as the etiologic agent for five outbreaks of gastroenteritis; three were associated with swimming in lakes, one with swimming in a pond (*9*), and one with playing in a wading pool. Crowded conditions or poor water exchange may have been contributing factors in at least three of the outbreaks. Secondary cases of shigellosis were noted for three of the outbreaks.

An outbreak of serologically confirmed hepatitis A at a commercial campground in Louisiana was the first reported outbreak of hepatitis A associated with use of a swimming pool (*10*). Water quality data were not available; however, the filtering system of the pool was designed in such a way that a cross-connection may have existed between the water-intake line and a sewage line.

Three boys were reported to have died from primary amebic meningoencephalitis due to *Naegleria*. The organism was identified in the cerebrospinal fluid of two of the boys and at autopsy for the third. One boy had swum in a commercial swimming area in a lake in Arkansas; the other two had swum both in a swimming pool and in a lake or pond in Texas.

Previously Unreported Outbreaks in 1988

After the surveillance summary for 1986–1988 was published, reports of seven additional outbreaks in 1988 were received, two associated with water intended for drinking and five with recreational exposures (Table 2).

An outbreak of chronic gastrointestinal illness (CGI), the second reported in this surveillance system, was associated with drinking well water from a community water system in Oklahoma. III persons had watery diarrhea for a median of 13 months. No etiologic agent was identified despite an extensive search for bacterial, parasitic, and viral pathogens. Coliforms were not found in routine water samples collected during the outbreak.

An outbreak of hepatitis A was associated with drinking fecally contaminated well water in a community in Washington. Although the source of contamination of the well was not identified, an old adjacent septic system was suspected to be leaking.

An outbreak of both Pontiac fever and *Pseudomonas* folliculitis was associated with the use of a whirlpool. Ill persons had fourfold rises in antibody titer to *Legionella pneumophila* serogroup 1; in addition, this organism and *Pseudomonas aeruginosa* were isolated from the whirlpool water.

During an investigation that began in 1988, high lead levels found among residents of rural areas on the big island of Hawaii were attributed to drinking water from rainwater catchment tanks sealed with lead-based paint. Information concerning this chemical exposure was not included in the line listing (Table 2) because the affected persons did not manifest signs of lead toxicity. However, four (6%) of 72 children under the age of 6 years who were tested had blood lead levels ≥15 μg/dl (none were ≥25 μg/dl), and nine (3%) of 305 persons ≥6 years old had levels ≥40 μg/dl (maximum of 73.2); all but one of these persons drank water that was heavily contaminated with lead (maximum of 4520 ppb).

Outbreaks on Cruise Ships

In 1989 and 1990, CDC personnel investigated 15 outbreaks of diarrheal illness on nine cruise ships (four in 1989 and five in 1990) that call on U.S. ports; one of these ships was an intrastate cruise liner. The median size of the 13 outbreaks with data that could be evaluated was 208 passengers (range, 38-412).

On one ship, two outbreaks occurred during the same 15-day cruise; both outbreaks were associated with exposures on land. On another ship, outbreaks occurred on two consecutive week-long cruises; the second outbreak, which was associated with eating freshly cut fruit items and stuffed eggs, was attributed to a Norwalk-like virus. On an additional ship, outbreaks occurred on two cruises 1 week apart. An outbreak occurred on a different ship from the same cruise line during the intervening week; cold seafood items containing scallops were implicated as the probable vehicles of the outbreaks, and enterotoxigenic *E. coli* was the probable etiologic agent. On another ship, outbreaks occurred on four consecutive week-long cruises; the attack rates progressively decreased. A Norwalk-like virus was found in stool specimens from ill passengers from the first two cruises, but the source of infection was not determined. In the first foodborne outbreak of shigellosis on a cruise ship investigated by CDC, German potato salad was implicated as the vehicle and multiresistant *Shigella flexneri* 4a as the etiologic agent (*11*). Consumption of water and ice was associated with illness in an unrelated shipboard outbreak, but food histories were not available, and the etiologic agent was not identified. Etiologic agents and vehicles were not identified in the other investigations.

DISCUSSION

Outbreaks Associated with Water Intended for Drinking

The numbers of outbreaks associated with water intended for drinking reported for 1989 (12) and 1990 (14) were comparable with the numbers previously reported for 1987 (15) and 1988 (13) (*2*). Additional outbreaks for 1989-1990 may be reported in the future, just as this surveillance summary includes data about two additional outbreaks for 1988. In contrast to the statistics for 1987–1990, at least 20 outbreaks per

year were reported for all previous years for which CDC and EPA have tabulated data (1971–1986) (Figures 4 and 5). The extent to which the decrease in the numbers of reported outbreaks (from a high of 53 in 1980) reflects a true decrease and not simply a reporting artifact is unknown and has been discussed previously (*2,3*). The requirement that outbreaks be investigated epidemiologically, which adds an element of complexity to the surveillance system, may be becoming more difficult to fulfill as the demands on health departments increase.

The relative proportions of outbreaks attributed to various types of water supplies and etiologic agents have remained fairly stable (Figures 4 and 5). Most outbreaks in 1989–1990 were associated with noncommunity or small community water systems, as has been noted for previous years (*12*). Only one outbreak occurred in a large metropolitan area (Chicago), but the outbreak was restricted to a hospital building and was due to a problem in the distribution system. The association of outbreaks with noncommunity or small community water systems may reflect the fact that large cities tend to have more sophisticated water treatment plants. On the other hand, sporadic cases of a waterborne disease that occur over a large metropolitan area may never be linked and may never be attributed to exposure to water, since ill persons in large cities are likely to consult different physicians.

Giardia lamblia was the most frequently identified etiologic agent in WBDOs for the 11th and 12th consecutive years. No outbreaks in 1989 or 1990 were attributed to *Cryptosporidium*, a protozoan parasite that is even more chlorine resistant than *Giardia*. A clinical case definition of acute giardiasis has been proposed that may prove useful to investigators of future outbreaks (*13*). Recently developed immunodiagnostics, such as monoclonal antibodies and enzyme-linked immunosorbent assays (*14*), may be useful for detecting *G. lamblia*-associated antigen in stool specimens and water samples during investigations of outbreaks.

As in the past, the outbreaks of giardiasis were associated with ingestion of unfiltered, inadequately chlorinated surface water or surface-influenced groundwater. *Giardia* (but not necessarily organisms even more resistant to chlorine) can be inactivated by disinfection without filtration, but only if stringent conditions are met and consistently maintained (*12,15,16*). The surface water treatment requirements (54 FR 27486-541, June 19, 1989) address regulations for treating surface water in public water systems and the criteria for exempting surface water from mandatory filtration. The addition of filtration to the water treatment process is an example of using multiple barriers to protect water supplies; treating sewage appropriately, preventing the contamination of watersheds, and using more than one water treatment process (e.g., disinfection and filtration of surface water) are all key elements in ensuring the availability of high-quality drinking water and preventing transmission of waterborne diseases (*12*).

During the period 1989–1990 only one WBDO was attributed to a bacterial pathogen, *E. coli* O157:H7; from 1971 to 1986, a mean of four outbreaks of bacterial diseases per year were reported. *E. coli* O157:H7 had never previously been associated with a WBDO in this surveillance system. However, waterborne transmission of this pathogen has been suspected previously (*17*), and it has been isolated from water (*18*). The severity of illness associated with this organism (*5*) reinforces the need to prevent WBDOs.

CLB, like *E. coli* O157:H7, had never previously been associated with a WBDO in this surveillance system. The role of CLB in causing diarrheal illness has not been

defined and is currently being investigated. These organisms, which are difficult to classify taxonomically, have been identified in stool specimens from patients around the world (*6,19,20*).

Four outbreaks reported in this surveillance summary (including one from 1988) were attributed to viral pathogens, either hepatitis A or a Norwalk-like agent. These outbreaks, which were all associated with well water, raise the issue of how groundwater becomes contaminated. The outbreak that occurred at a new resort in the southwest (in which a Norwalk-like agent was implicated) was particularly instructive (*8*). The resort's deep well and sewage treatment facility had been designed according to state-of-the-art technology, permitting the well and leach fields to be in close proximity. However, malfunction of two leach fields allowed sewage effluent to pass rapidly through the remaining three fields; the effluent then flowed unimpeded through channels in the compressed sandstone and limestone directly into the well.

The EPA has recently drafted criteria for disinfection requirements for public water systems using groundwater sources that are not under the direct influence of surface water (*21*). The draft rule and the proposed rule are expected to be available for public comment in 1992 and 1993, respectively; promulgation is expected in 1995.

Over half (54%) of all reported outbreaks for 1989-1990 were classified as outbreaks of AGI of unknown etiology. A substantial though variable proportion of outbreaks has been classified as such each year (Figure 5). In addition, an occasional outbreak—one in 1987 (*22*) and one in 1988 that is described here—has been classified as an outbreak of CGI of unknown etiology, a syndrome previously associated with consumption of raw milk (*23*). Some outbreaks were classified as being of unknown etiology after cursory investigations, others after rigorous investigations that were unrevealing. The clinical features of some of the illnesses suggest viral etiologies; however, clinical diagnosis is not very specific. Identification of the etiologic agents would provide insights into the adequacy of current water treatment processes. More timely investigations and increased availability of tests to identify viral agents and various novel pathogens may aid in elucidating the causes of these outbreaks. Even so, the isolation of coliforms from water samples during most investigations of outbreaks of AGI in 1989–1990 indicates that chlorine-sensitive organisms, not just relatively chlorine-resistant organisms such as viruses, were present in the water. Therefore, the readily available technology to eliminate chlorine-sensitive organisms should be applied more consistently and reliably.

The possible outbreak associated with bottled water was reported at a time of increased concern about the quality and safety of bottled water. Although EPA regulates public drinking water systems, the Food and Drug Administration sets standards for the quality of the bottled water that is sold in interstate commerce. The adequacy of the standards for bottled water and the means of enforcing the standards have recently been reviewed (*24*).

No chemical intoxications were reported for 1989 or 1990, but lead-contaminated drinking water in Hawaii in 1988 was associated with elevated blood lead levels. The investigation was prompted by concern that acid rain secondary to volcanic activity was causing lead to be leached from various materials. Recently, EPA promulgated a revised National Priority Drinking Water Regulation for lead in public water systems (56 FR 26459-564, June 7, 1991); the regulation requires lead levels to be $\leq$15 ppb in at least 90% of tap water samples from high-risk homes after the water has been

standing overnight in the household plumbing. Although this regulation technically applies only to public water systems, its underlying scientific rationale is relevant to all types of water systems. Of note, the waterborne-disease surveillance system is better equipped to detect acute chemical intoxications than the effects of chronic exposures to toxins.

Outbreaks Associated with Recreational Water Use

In 1989–1990, more WBDOs but fewer cases were attributed to recreational exposures than to ingestion of water intended for drinking. The extent of underrecognition and underreporting of outbreaks associated with recreational exposures is unknown. The newly revised outbreak report form (Figure 1) should facilitate reporting these outbreaks. Presumably many outbreaks of *Pseudomonas* folliculitis are not reported, since this is a relatively mild disease. Although most of the 13 reported outbreaks of folliculitis were relatively small, 300 persons became ill in one of the outbreaks. These outbreaks are preventable if the water in hot tubs and whirlpools is maintained at a pH of 7.2-7.8 with free residual chlorine levels from 2 to 5 mg/L, as specified in CDC's guidelines for public spas and hot tubs (*25*). Another risk of whirlpool use is Pontiac fever resulting from aerosolized antigens of *Legionella pneumophila* (*26,27*).

Swimming-associated outbreaks of shigellosis, which have been documented previously (*28-30*), continue to occur. Only a small inoculum of organisms is necessary to cause illness; therefore, even persons who do not swallow large volumes of water can become infected. The EPA has published criteria for evaluating the quality of fresh and marine recreational waters (*31,32*). The first outbreak of hepatitis A associated with use of a swimming pool was reported (*10*). Three persons with swimming-associated primary amebic meningoencephalitis due to *Naegleria fowleri* were reported, even though such case reports were not explicitly solicited. Sporadic cases of this fatal disease have been documented previously (*33*).

Outbreaks on Cruise Ships

The outbreaks in 1989–1990 aboard passenger cruise ships were notable because some occurred on consecutive cruises. This phenomenon has been observed previously (*34-36*). An earlier investigation of one of a series of outbreaks implicated vomitus in the transmission of a viral agent (*36*). The goal of CDC's Vessel Sanitation Program, which was established in 1975, is to prevent outbreaks of gastroenteritis on cruise ships (*37,38*).

CONCLUSION

Waterborne diseases in the United States are not associated with as much morbidity and mortality as they were earlier in this century. However, WBDOs continue to occur, sometimes even in relatively sophisticated community water systems. CDC and EPA have monitored the occurrence of WBDOs for two decades. The continued surveillance of WBDOs on a national level makes it possible to characterize the changing epidemiology of waterborne diseases and to identify the types of water systems, the water system deficiencies, and the etiologic agents associated with outbreaks. Agents only recently associated with WBDOs include *E. coli* O157:H7, CLB, and *Cryptosporidium* (*39*). Identification of such agents is important, since they may require new means of control. Persons investigating future

outbreaks also need to be aware that these organisms are possible etiologic agents, and therefore the laboratory investigations should be structured accordingly. Strengthened surveillance of WBDOs may be possible, since 46 (92%) of the states have designated persons to coordinate this activity. The ongoing challenge is to structure the surveillance system so that the data are applied to prevent outbreaks.

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TABLE 1. Outbreaks associated with water intended for drinking, United States, 1989-1990 (N = 26)*

State	Year	Month	Class†	Etiologic agent§	No. cases	Type of system¶	Deficiency	Source	Setting
AK	1990	Mar	II	*Giardia*	18	NC	1	River	Lodge
AL	1989	Sep	I	AGI	700	Com	2	Spring	Community
AR	1990	Jul	III	AGI	75	Ind	2	Well	Residence
AZ	1989	Apr	I	Norwalk-like	900	NC	3	Well	Resort
CA	1990	Feb	I	AGI	12	NC	3	Spring	Camp
CO	1989	Feb	I	*Giardia*	19	Com	3	River	Community
CO	1990	Aug	I	*Giardia*	123	Com	3	Spring	Community
ID	1989	Aug	III	AGI	31	Ind	1	Lake	Cabin
IL	1990	Jul	I	CLB (possible)	21	Com	4	Lake	Hospital
ME	1989	Apr	I	AGI	54	NC	3	Well	Restaurant
MN	1990	Feb	I	AGI	76	NC	2	Well	Resort
MN	1990	Aug	I	AGI	150	NC	4	Well	Resort
MO	1989	Dec	I	*Escherichia coli* O157:H7	243	Com	4	Well	Community**
MO	1990	May	I	AGI	109	Com	3	Lake	Community
MO	1990	Jul	I	AGI	52	NC	3	Well	Resort
NJ	1989	Jul	I	AGI	8	NC	3	Well	Camp††
NY	1989	Apr	I	*Giardia*	308	Com	3	Reservoir	Community
NY	1989	Jun	I	*Giardia*	152	NC	3	Reservoir	Prison
NY	1989	Jul	I	*Giardia*	53	Com	3	Lake	Community
PA	1989	Jul	I	AGI	50	NC	2	Well	Camp§§
PA	1990	May	III	Hepatitis A	22	Ind	2	Well	Group of 5 homes
PA	1990	Nov	III	Hepatitis A	3	Com	3	Well	Community
PA	1990	Dec	II	AGI	63	Com	5	Lake	Inn
TN	1990	May	I	AGI	1000	NC	3	Well	Country club
VT	1989	Jun	I	AGI	22	Com	2	Well	Housing development
VT	1990	Mar	I	*Giardia*	24	NC	3	Lake	Resort

*See Methods section for description of reporting variables.
†See Table 3 for class definitions.
§AGI = acute gastrointestinal illness of unknown etiology; CLB = cyanobacteria (blue-green algae)-like bodies.
¶NC = noncommunity; Com = community; Ind = individual.
**Resulted in 4 deaths.
††The vehicle of transmission was iced tea.
§§Two punches made with the water and watermelon kept cool in the water were implicated.

TABLE 2. Waterborne disease outbreaks not included in previous summaries, United States, 1988 (N = 7)*

A. Outbreaks associated with water intended for drinking

State	Month	Class†	Etiologic agent§	No. cases	Type of system¶	Deficiency	Source	Setting
OK	Nov	I	CGI	22	Com	3	Well	Community
WA	Sep	II	Hepatitis A	9	Com	3	Well	Trailer park

B. Outbreaks associated with recreational water use

State	Month	Class†	Illness	Etiologic agent§	No. cases	Source**	Setting
MD	Apr		Folliculitis	*Pseudomonas*	14	Whirlpool	Hotel
MN	Jan		Folliculitis; Pontiac fever	*Pseudomonas*; *L. pneumophila*	28	Whirlpool	Hotel
MN	Feb		Folliculitis	c/w *Pseudomonas*	34	Pool/whirlpool	Hotel
MN	Jun	II	Gastroenteritis	AGI	24	Lake	Swimming area
MN	Dec		Folliculitis	c/w *Pseudomonas*	18	Whirlpool	Resort

*See Methods section for description of reporting variables.
†See Table 3 for class definitions.
§CGI = chronic gastrointestinal illness of unknown etiology; c/w = consistent with; AGI = acute gastrointestinal illness of unknown etiology.
¶Com = community.
**The source is identified here as it was on the report form. If more than 1 source is listed (e.g., pool/whirlpool), both were possible sources.

TABLE 3. Classification of investigations

Class*	Epidemiologic data	Water quality data
I	**Adequate:†** (A) data were provided about exposed and unexposed persons; and (B) the relative risk or odds ratio was ⩾2, or the p-value was ⩽0.05.	**Provided and adequate:** could be historic information or laboratory data. Examples: the history that a chlorinator malfunctioned or a water main broke; no detectable free chlorine residual; the presence of coliforms in the water.
II	**Adequate.**	**Not provided or inadequate.** Example: stating that a lake was crowded.
III	**Provided, but limited:** (A) epidemiologic data were provided that did not meet the criteria for Class I; or (B) the statement was made that ill persons had no exposures in common besides water, but no data were provided.	**Provided and adequate.**
IV	**Provided, but limited.**	**Not provided or inadequate.**

* Classification was based on the epidemiologic and water quality data that were provided on the outbreak report form.
†Adequate to implicate water.

TABLE 4. Outbreaks associated with recreational water use, United States, 1989-1990 (N = 30)

State	Year	Month	Class*	Illness	Etiologic agent†	No. cases	Source§	Setting
AK	1989	May		Folliculitis	*Pseudomonas*	10	Hot tub	Resort
AR	1990	Jul		Amebic meningoencephalitis	*Naegleria*	1	Lake	Swimming area
LA	1989	Sep	II	Hepatitis	Hepatitis A	20	Pool	Campground
ME	1989	Feb		Folliculitis	*Pseudomonas*	3	Hot tub	Hotel
ME	1989	Jun		Folliculitis	*Pseudomonas*	5	Hot tub	Motel
ME	1989	Jul	III	Gastroenteritis	AGI	22	Lake	Park
MI	1989	Jul	I	Gastroenteritis	*Shigella sonnei*	65	Pond	Park
MN	1989	Mar		Folliculitis	c/w *Pseudomonas*	300	Whirlpool	Hotel
MN	1990	Jan		Folliculitis	c/w *Pseudomonas*	10	Pool/whirlpool	Motel
MN	1990	Jul	II	Gastroenteritis	AGI	18	Lake	Camp
MS	1990	Apr		Folliculitis	*Pseudomonas*	10	Hot tub	Motel
NC	1990	Jun	I	Gastroenteritis	*Shigella sonnei*	68	Lake	Recreation area
NJ	1989	Jun	I	Gastroenteritis	AGI	17	Lake	Park
NJ	1989	Jul	I	Gastroenteritis	AGI	26	Lake	Swimming area
NY	1990	Jul	IV	Gastroenteritis	*Shigella sonnei*	7	Lake	Park
OR	1990	Jul	III	Gastroenteritis	*Shigella sonnei*	9	Lake	Park
PA	1990	Jul	I	Gastroenteritis	AGI	60	Lake	Camp
SD	1990	Oct		Folliculitis	*Pseudomonas*	30	Whirlpool	Apt. clubhouse
TX	1989	Jul		Amebic meningoencephalitis	*Naegleria*	1	Pool/lake	Swimming area
TX	1989	Aug		Amebic meningoencephalitis	*Naegleria*	1	Pool/pond	Military base
TX	1990	Feb		Folliculitis	*Pseudomonas*	7	Pool/whirlpool	Hotel
VT	1990	Feb		Folliculitis	*Pseudomonas*	23	Whirlpool	Hotel
WA	1989	Mar		Folliculitis	c/w *Pseudomonas*	2	Hot tub	Private home
WA	1989	Dec		Folliculitis	c/w *Pseudomonas*	2	Hot tub	Private home
WA	1990	Jul	I	Gastroenteritis	AGI	244	Lake	Park
WA	1990	Oct		Folliculitis	c/w *Pseudomonas*	3	Hot tub	Private home
WI	1990	Jun	I	Gastroenteritis	AGI	79	Lake	Park
WI	1990	Aug	IV	Gastroenteritis	*Shigella sonnei*	10	Wading pool	Park
WY	1989	Jan		Folliculitis	*Pseudomonas*	5	Spa	Motel
WY	1990	Jun	III	Gastroenteritis	AGI	4	Pond	Information center

*See Table 3 for class definitions.
†AGI = acute gastrointestinal illness of unknown etiology; c/w = consistent with.
§In general, the source is identified here as it was on the report form. In some reports, however, the words whirlpool, hot tub, and spa were used interchangeably. If more than one source is listed (e.g., pool/whirlpool), both were possible sources.

TABLE 5. Outbreaks associated with water intended for drinking, by etiologic agent and type of water system, United States, 1989-1990 (N=26)

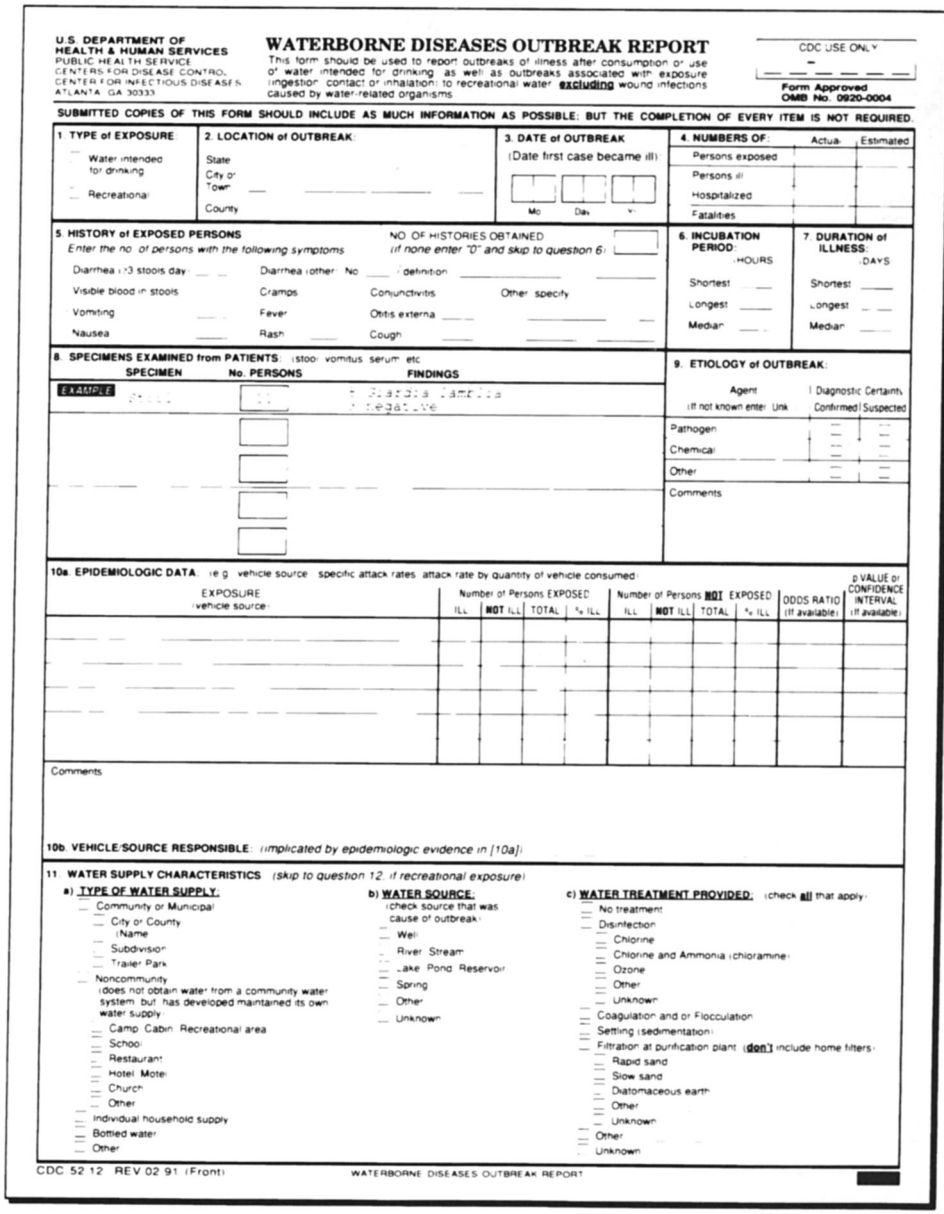
	Type of water system*							
	Community		Noncommunity		Individual		Total	
Agent†	Outbreaks	Cases	Outbreaks	Cases	Outbreaks	Cases	Outbreaks	Cases
AGI	4	894	8	1402	2	106	14	2402
Giardia	4	503	3	194	0	0	7	697
Hepatitis A	1	3	0	0	1	22	2	25
Norwalk-like	0	0	1	900	0	0	1	900
Escherichia coli O157:H7	1	243	0	0	0	0	1	243
CLB (possible)	1	21	0	0	0	0	1	21
Total	11	1664	12	2496	3	128	26	4288
Percent§	42	39	46	58	12	3	100	100

*See Methods section for descriptions of reporting variables.
†AGI = acute gastrointestinal illness of unknown etiology; CLB = cyanobacteria (blue-green algae)-like bodies.
§The percentage of 26 outbreaks or of 4,288 cases.

TABLE 6. Outbreaks associated with water intended for drinking, by type of deficiency and type of water system, United States, 1989-1990 (N=26)

	Type of water system Number (%)							
Type of deficiency	Community		Noncommunity		Individual		Total	
Untreated surface water	0	(0)	1	(18)	1	(33)	2	(8)
Untreated groundwater	2	(18)	2	(17)	2	(67)	6	(23)
Treatment	6	(55)	8	(67)	0	(0)	14	(54)
Distribution system	2	(18)	1	(8)	0	(0)	3	(12)
Unknown	1	(9)	0	(0)	0	(0)	1	(4)
Total	**11**	**(100)**	**12**	**(100)**	**3**	**(100)**	**26**	**(100)**

FIGURE 1. The newly revised report form (CDC Form 52.12, Rev. 02-91) for waterborne-disease outbreaks, both those associated with water intended for drinking and those associated with recreational water use.



U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
CENTER FOR INFECTIOUS DISEASES
ATLANTA, GA 30333

WATERBORNE DISEASES OUTBREAK REPORT

This form should be used to report outbreaks of illness after consumption or use of water intended for drinking, as well as outbreaks associated with exposure (ingestion, contact or inhalation) to recreational water **excluding** wound infections caused by water-related organisms.

CDC USE ONLY

Form Approved
OMB No. 0920-0004

SUBMITTED COPIES OF THIS FORM SHOULD INCLUDE AS MUCH INFORMATION AS POSSIBLE; BUT THE COMPLETION OF EVERY ITEM IS NOT REQUIRED.

1. TYPE of EXPOSURE:
- Water intended for drinking
- Recreational

2. LOCATION of OUTBREAK:
State
City or Town:
County

3. DATE of OUTBREAK (Date first case became ill):
Mo Day Yr

4. NUMBERS OF:

	Actual	Estimated
Persons exposed		
Persons ill		
Hospitalized		
Fatalities		

5. HISTORY of EXPOSED PERSONS
Enter the no. of persons with the following symptoms.
NO. OF HISTORIES OBTAINED: *(If none enter "0" and skip to question 6.)*

Diarrhea (>3 stools/day)	Diarrhea (other) No. ____ definition:		
Visible blood in stools	Cramps	Conjunctivitis	Other, specify
Vomiting	Fever	Otitis externa	
Nausea	Rash	Cough	

6. INCUBATION PERIOD: (HOURS)
Shortest
Longest
Median

7. DURATION of ILLNESS: (DAYS)
Shortest
Longest
Median

8. SPECIMENS EXAMINED from PATIENTS: (stool, vomitus, serum, etc.)

SPECIMEN	No. PERSONS	FINDINGS
EXAMPLE: Stool	10	7 Giardia lamblia; 3 negative

9. ETIOLOGY of OUTBREAK:

Agent (If not known enter Unk.)	Diagnostic Certainty: Confirmed	Suspected
Pathogen:		
Chemical:		
Other:		

Comments:

10a. EPIDEMIOLOGIC DATA: (e.g., vehicle source, specific attack rates, attack rate by quantity of vehicle consumed)

EXPOSURE (vehicle source)	Number of Persons EXPOSED: ILL	NOT ILL	TOTAL	% ILL	Number of Persons NOT EXPOSED: ILL	NOT ILL	TOTAL	% ILL	ODDS RATIO (If available)	p VALUE or CONFIDENCE INTERVAL (If available)

Comments:

10b. VEHICLE/SOURCE RESPONSIBLE: *(implicated by epidemiologic evidence in [10a])*

11. WATER SUPPLY CHARACTERISTICS *(skip to question 12 if recreational exposure)*

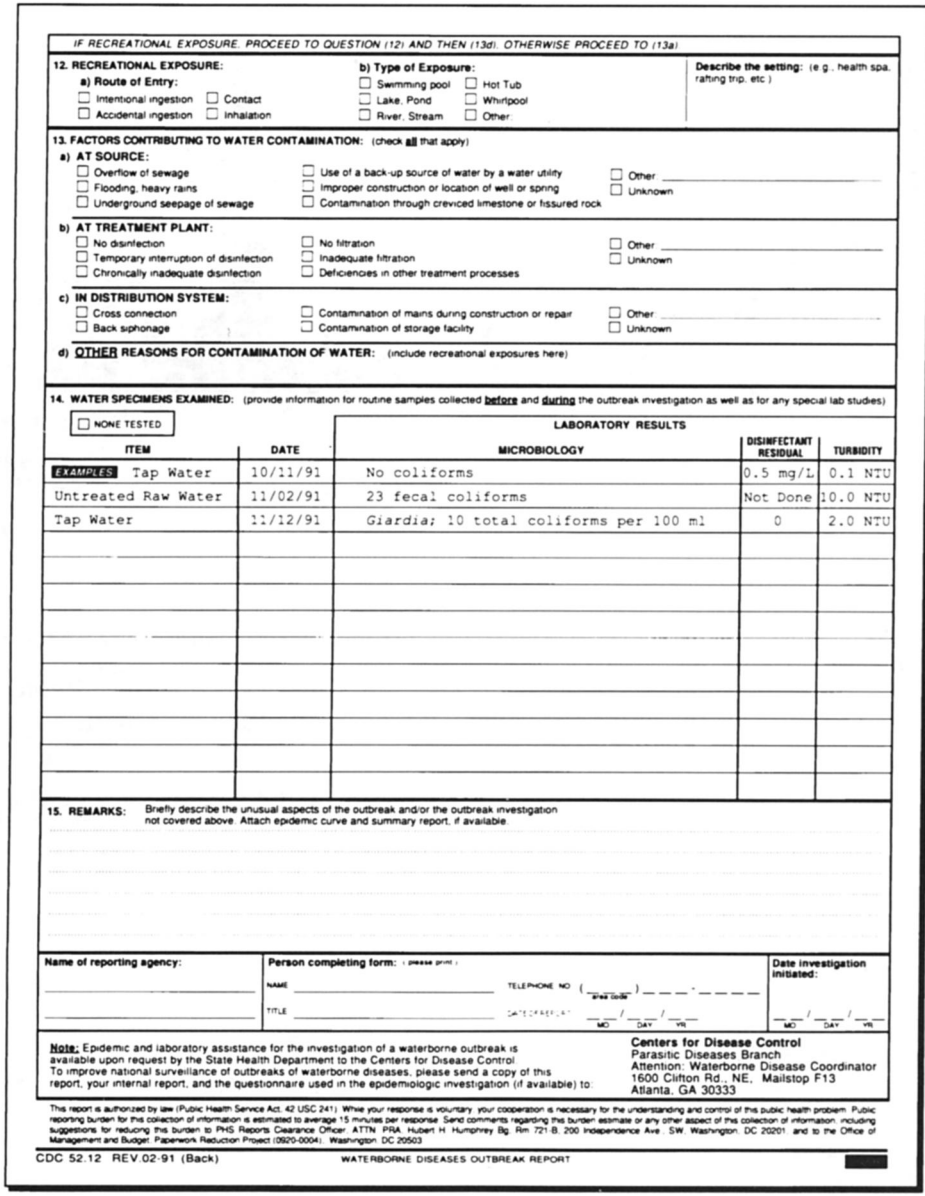
a) TYPE OF WATER SUPPLY:
- Community or Municipal
  - City or County (Name:
  - Subdivision
  - Trailer Park
- Noncommunity (does not obtain water from a community water system, but has developed maintained its own water supply)
  - Camp, Cabin, Recreational area
  - School
  - Restaurant
  - Hotel, Motel
  - Church
  - Other
- Individual household supply
- Bottled water
- Other

b) WATER SOURCE: (check source that was cause of outbreak)
- Well
- River, Stream
- Lake, Pond, Reservoir
- Spring
- Other
- Unknown

c) WATER TREATMENT PROVIDED: (check **all** that apply)
- No treatment
- Disinfection
  - Chlorine
  - Chlorine and Ammonia (chloramine)
  - Ozone
  - Other
  - Unknown
- Coagulation and or Flocculation
- Settling (sedimentation)
- Filtration at purification plant (**don't** include home filters)
  - Rapid sand
  - Slow sand
  - Diatomaceous earth
  - Other
  - Unknown
- Other
- Unknown

CDC 52.12 REV 02-91 (Front) WATERBORNE DISEASES OUTBREAK REPORT

FIGURE 1. The newly revised report form (CDC Form 52.12, Rev. 02-91) for waterborne-disease outbreaks, both those associated with water intended for drinking and those associated with recreational water use — *Continued*



IF RECREATIONAL EXPOSURE, PROCEED TO QUESTION (12) AND THEN (13d). OTHERWISE PROCEED TO (13a)

12. RECREATIONAL EXPOSURE:

a) Route of Entry:
☐ Intentional ingestion ☐ Contact
☐ Accidental ingestion ☐ Inhalation

b) Type of Exposure:
☐ Swimming pool ☐ Hot Tub
☐ Lake, Pond ☐ Whirlpool
☐ River, Stream ☐ Other

Describe the setting: (e.g., health spa, rafting trip, etc.)

13. FACTORS CONTRIBUTING TO WATER CONTAMINATION: (check **all** that apply)

a) AT SOURCE:
☐ Overflow of sewage
☐ Flooding, heavy rains
☐ Underground seepage of sewage
☐ Use of a back-up source of water by a water utility
☐ Improper construction or location of well or spring
☐ Contamination through creviced limestone or fissured rock
☐ Other _____
☐ Unknown

b) AT TREATMENT PLANT:
☐ No disinfection
☐ Temporary interruption of disinfection
☐ Chronically inadequate disinfection
☐ No filtration
☐ Inadequate filtration
☐ Deficiencies in other treatment processes
☐ Other _____
☐ Unknown

c) IN DISTRIBUTION SYSTEM:
☐ Cross connection
☐ Back siphonage
☐ Contamination of mains during construction or repair
☐ Contamination of storage facility
☐ Other _____
☐ Unknown

d) OTHER REASONS FOR CONTAMINATION OF WATER: (include recreational exposures here)

14. WATER SPECIMENS EXAMINED: (provide information for routine samples collected **before** and **during** the outbreak investigation as well as for any special lab studies)

☐ NONE TESTED

ITEM	DATE	LABORATORY RESULTS: MICROBIOLOGY	DISINFECTANT RESIDUAL	TURBIDITY
EXAMPLES Tap Water	10/11/91	No coliforms	0.5 mg/L	0.1 NTU
Untreated Raw Water	11/02/91	23 fecal coliforms	Not Done	10.0 NTU
Tap Water	11/12/91	*Giardia*; 10 total coliforms per 100 ml	0	2.0 NTU

15. REMARKS: Briefly describe the unusual aspects of the outbreak and/or the outbreak investigation not covered above. Attach epidemic curve and summary report, if available.

Name of reporting agency:

Person completing form: (please print)
NAME _____ TELEPHONE NO. (area code) ___-___-____
TITLE _____ DATE COMPLETED: ___/___/___ MO DAY YR

Date investigation initiated: ___/___/___ MO DAY YR

Note: Epidemic and laboratory assistance for the investigation of a waterborne outbreak is available upon request by the State Health Department to the Centers for Disease Control. To improve national surveillance of outbreaks of waterborne diseases, please send a copy of this report, your internal report, and the questionnaire used in the epidemiologic investigation (if available) to:

Centers for Disease Control
Parasitic Diseases Branch
Attention: Waterborne Disease Coordinator
1600 Clifton Rd., NE, Mailstop F13
Atlanta, GA 30333

This report is authorized by law (Public Health Service Act, 42 USC 241). While your response is voluntary, your cooperation is necessary for the understanding and control of this public health problem. Public reporting burden for this collection of information is estimated to average 15 minutes per response. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to PHS Reports Clearance Officer, ATTN: PRA, Hubert H. Humphrey Bg., Rm. 721-B, 200 Independence Ave., S.W., Washington, DC 20201; and to the Office of Management and Budget, Paperwork Reduction Project (0920-0004), Washington, DC 20503.

CDC 52.12 REV.02-91 (Back) WATERBORNE DISEASES OUTBREAK REPORT

FIGURE 2. Waterborne-disease outbreaks, by month, United States, 1989–1990

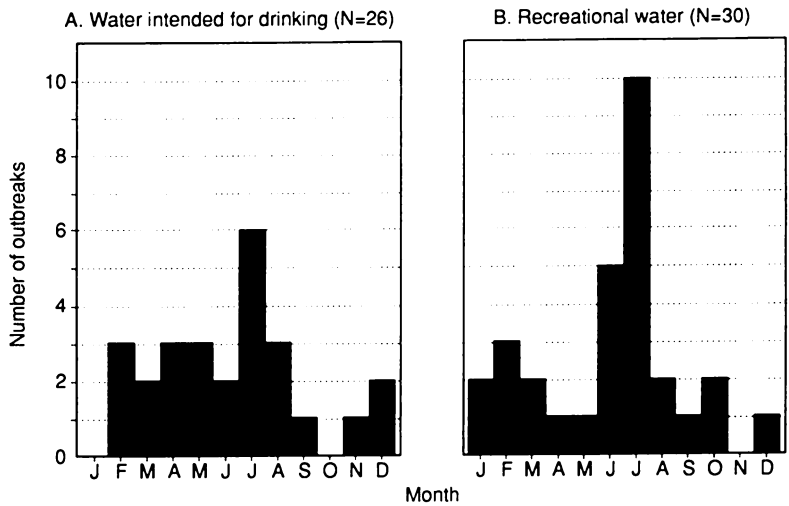
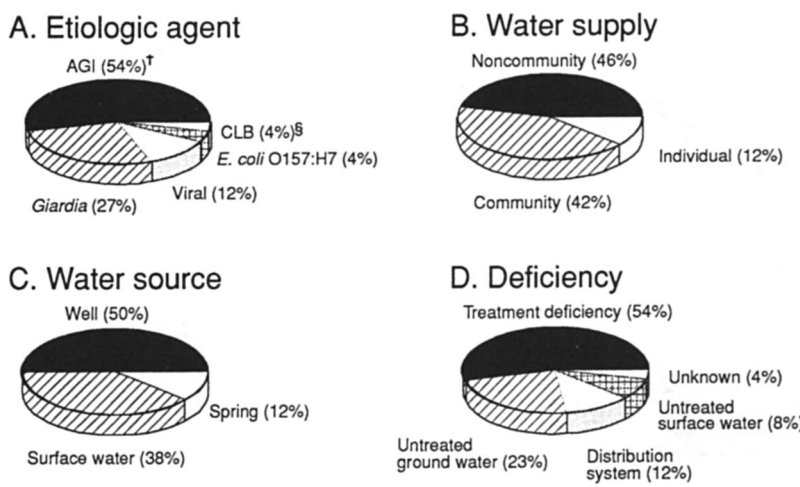
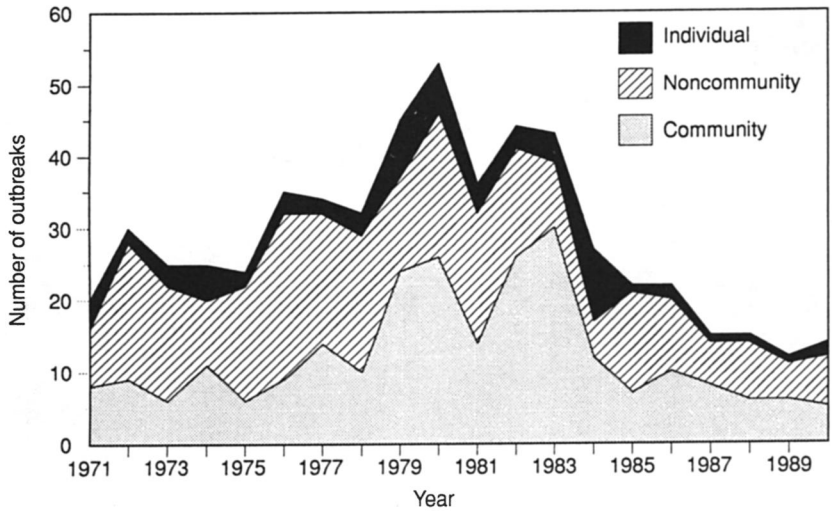


FIGURE 3. Outbreaks associated with water intended for drinking, United States, 1989–1990 (N = 26)*



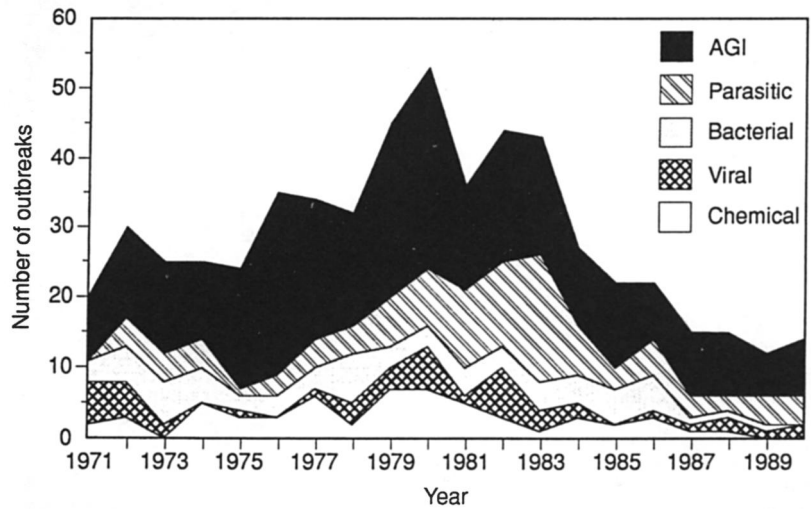
*See Methods section for description of reporting variables.
†AGI = acute gastrointestinal illness of unknown etiology.
§CLB = cyanobacteria (blue-green algae)-like bodies.

FIGURE 4. Waterborne-disease outbreaks, by year and type of water supply, United States, 1971–1990 (N = 573)*



*See Methods section for description of reporting variables.

FIGURE 5. Waterborne-disease outbreaks, by year and etiologic agent, United States, 1971–1990 (N = 573)*



*For convenience, the one outbreak (1990) associated with hard-to-classify CLB (cyanobacteria [blue-green algae]-like bodies) has been grouped here with the parasitic agents. The two outbreaks of chronic gastrointestinal illness, which occurred in 1987 and 1988, are grouped here with the outbreaks of AGI (acute gastrointestinal illness) of unknown etiology.

Tuberculosis Morbidity in the United States: Final Data, 1990

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Summary

The number of tuberculosis cases reported to CDC has been increasing since 1988, after a long historic decline. In 1990, 25,701 cases were reported, an increase of 9.4% over the 1989 figure and the largest annual increase since 1953. From 1985 to 1990, reported cases increased by 15.8%. Disproportionately greater increases in reported cases occurred among Hispanics, non-Hispanic blacks, and Asians/Pacific Islanders. In contrast, decreases were observed among non-Hispanic whites and American Indians/Alaskan Natives. By age, the largest increase in reported cases occurred in the 25- to 44-year age group; this increase may be largely attributable to rising numbers of tuberculosis cases among persons with human immunodeficiency virus infection or acquired immunodeficiency syndrome. Notable increases also occurred among children. The proportion of cases among foreign-born persons has risen steadily, from 21.6% in 1986 to 24.4% in 1990.

INTRODUCTION

Since 1953, all state and territorial health departments and the New York City Health Department have been submitting data on reported cases of tuberculosis, using a standardized case definition. At first, aggregate tabular reports were submitted to CDC, but since 1985 all reporting areas have submitted individual case reports to the Division of Tuberculosis Elimination (DTBE), National Center for Prevention Services, via the Report of a Verified Case of Tuberculosis (RVCT) system.

The number of tuberculosis cases reported to CDC had been declining an average of 5% per year until 1985. Since then, the number of tuberculosis cases reported has increased.

METHODS

DTBE receives RVCT data from 54 reporting areas: the 50 states, New York City, the District of Columbia, Puerto Rico, and the Virgin Islands. New case reports are edited weekly to verify diagnoses (*1*) and detect errors, which are referred back to the reporting areas for correction. In addition, reporting areas may initiate revisions. On a quarterly basis, the accumulated data are returned to the reporting areas, which may correct or update case reports. At year's end, all data are reviewed by the respective areas for revision before the national data are accepted as final.

This summary presents reported data for 1990 from all areas except Puerto Rico and the Virgin Islands. RVCT data were analyzed by age, sex, race/ethnicity, and country of origin of the persons with tuberculosis and by reporting area. Logistic

regression analysis was used to model tuberculosis incidence from 1953 through 1984, and the difference between predicted and observed incidence was used to estimate the number of tuberculosis cases in excess of historic trends for the period 1985–1990.

RESULTS

In 1990, the 50 states, New York City, and the District of Columbia reported 25,701 cases of tuberculosis–an increase of 9.4% (+2,206 cases) over the 1989 total of 23,495, which in turn was an increase of 4.7% (+1,059 cases) over the 1988 total. The tuberculosis incidence for 1990 was 10.3/100,000 population, compared with 9.5 for 1989 and 9.1 for 1988.

Thirty-one states reported increases in cases of tuberculosis in 1990 compared with 1989. The states with the largest increases were New York, which includes New York City (+974, +30.4%); California (+677, +16.1%); and Texas (+327, +17.1%). The largest increases reported in cities over 250,000 population were in New York City (+975, +38.3%), Los Angeles (+68, +7.8%), and Oakland (+62, +51.2%).

From 1985, the year with the lowest number of reported tuberculosis cases, to 1990, reported cases increased from 22,201 to 25,701 (+3,500, +15.8%). From incidence trends analysis, approximately 28,000 excess cases occurred from 1985 through 1990 (Figure 1).

From 1985 to 1990, reported tuberculosis cases increased in every racial/ethnic group except non-Hispanic whites and American Indians/Alaskan Natives (Table). Reported cases increased among Hispanics by 54.7%, among non-Hispanic blacks by 26.9%, and among Asians/Pacific Islanders by 19.6%. Cases decreased by 6.5% among American Indians/Alaskan Natives and by 7.3% among non-Hispanic whites.

Compared with 1985, the number of reported tuberculosis cases increased for all age groups except for those 65 and older (Table). The greatest increase occurred in the 25- to 44-year age group (+2,972, +44.0%). The age distribution of cases has been shifting toward the younger age groups, with notable increases among young adults and children under 15 years of age (Figure 2).

From 1986, the first year when all states reported the country of origin for persons with tuberculosis, to 1990, reported cases among foreign-born persons (persons born outside the United States and its territories) increased by 1,337 (+27.1%). Reported cases among foreign-born persons, as a fraction of the total cases, increased from 21.6% in 1986 to 24.4% in 1990. Of cases reported in 1990 among the foreign-born whose year of arrival was known (76%), 60% had been in the United States for 5 years or less.

DISCUSSION

In 1990, tuberculosis morbidity in the United States was characterized by an acceleration of the recent increase in cases, an increasingly greater fraction of cases occurring among some racial and ethnic minorities, an increase in cases among children, and an increase in the proportion of cases occurring among foreign-born persons. The 9.4% increase in reported tuberculosis cases in 1990 compared with 1989 marks the largest annual increase in reported tuberculosis morbidity since uniform national reporting began in 1953.

A variety of evidence supports the hypothesis that the epidemic of human immunodeficiency virus (HIV), the agent responsible for acquired immunodeficiency syndrome (AIDS), is contributing substantially to this observed rise in tuberculosis

cases (*2-5*). In a prospective study of tuberculin-positive injecting drug users who were followed an average of 22 months in a methadone treatment center, the incidence of tuberculosis was 7.9% per year among those who were initially HIV positive. No cases were observed in a control group of tuberculin-positive injecting drug users who were HIV negative (*6*). In HIV seroprevalence surveys at selected metropolitan tuberculosis clinics, the prevalence of HIV infection among new tuberculosis patients ranged from no HIV positivity at some sites to 46.3% at one (*7*). In states reporting the highest numbers of AIDS cases, the largest increases in tuberculosis cases have been reported among 25- to 44-year-old men (*8*), the age and sex group that also accounted for 68.4% of all AIDS patients reported to CDC through 1990 (*9*).

However, not all the estimated 28,000 excess tuberculosis cases may be directly attributable to the HIV epidemic. Reported increases have also been observed in age and racial/ethnic groups not characterized by large numbers of reported AIDS cases. The increase among Asians/Pacific Islanders, a group with a low observed incidence of AIDS (*8*), suggests that cases among persons from countries with a high prevalence of tuberculous infection contributed to the increases. In addition, increases in tuberculosis cases in the <15-year age group point to increased transmission of tuberculous infection in the United States, since most cases among children result from recent infection. Very likely, increases among persons of reproductive age have resulted in transmission of infection to the children who now manifest the disease. This trend of rising tuberculosis rates among children has serious implications for tuberculosis control programs in the future.

The Advisory Council for Elimination of Tuberculosis has developed a strategic plan, endorsed by the Secretary of Health and Human Services, for the elimination of tuberculosis from the United States by the year 2010 (*10*). Tuberculosis morbidity data from 1985 to 1990 suggest that substantial barriers jeopardize achievement of this goal. For the plan to succeed, tuberculosis control programs must focus increased resources on persons at high risk of tuberculous infection; such persons include contacts of persons recently diagnosed as having tuberculosis, members of racial and ethnic minorities, and the foreign-born. Persons with tuberculous infection who have conditions placing them at increased risk of active tuberculosis, such as HIV infection, also require special attention. Innovative methods are required to reach persons at greatest risk; cultural and social factors must also be taken into consideration. New technologies must be developed to speed and enhance diagnostic methods and to develop more effective treatment of tuberculosis and tuberculous infection. Without additions to existing intervention programs, the trends observed from 1985 through 1990 suggest that tuberculosis cases will continue to rise.

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2. Barnes PF, Bloch AB, Davidson PT, Snider DE. Tuberculosis in patients with human immunodeficiency virus infection. N Engl J Med 1991;234:1644–50
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5. CDC. Tuberculosis and human immunodeficiency virus infection: Recommendations of the Advisory Committee for Elimination of Tuberculosis (ACET). 1989;38:236–8,243–50.

6. Selwyn PA, Hartel D, Lewis VA, *et al*. A prospective study of the risk of tuberculosis among intravenous drug users with human immunodeficiency virus infection. N Engl J Med 1989;320:545–50.
7. CDC. National HIV seroprevalence surveys: summary of results: data from serosurveillance activities through 1989. Washington, D.C.: Government Printing Office, 1990. (DHHS publication no. HIV/CID/9-90/006.)
8. CDC. The HIV/AIDS epidemic: the first 10 years. MMWR 1991;40:357–69.
9. CDC. HIV/AIDS surveillance. U.S. AIDS cases reported through December 1990. January 1991:14.
10. CDC. A strategic plan for the elimination of tuberculosis in the United States. MMWR 1989;38:269–72.

Table 1. Reported cases and rates of tuberculosis by sex, age group, race/ethnicity, and country of origin – United States, 1985 and 1990

Characteristic	Cases		%	Rate*		%
	1985	1990	Change	1985	1990	Change
Totals	**22,201**	**25,701**	**+15.8**	**9.3**	**10.3**	**+10.8**
Sex						
Male	14,496	16,966	+17.0	12.5	14.0	+12.0
Female	7,704	8,729	+13.3	6.3	6.8	+7.9
Unknown	1	6	–†	NA§	NA	
Age						
0-4	789	936	+18.6	4.4	5.1	+15.9
5-14	472	660	+39.8	1.4	1.9	+35.7
15-24	1,672	1,867	+11.7	4.2	5.1	+21.4
25-44	6,758	9,730	+44.0	9.2	12.0	+30.4
45-64	6,138	6,365	+3.7	13.7	13.7	0.0
⩾65	6,356	6,115	–3.8	22.3	19.6	–12.1
Unknown	16	28	–	NA	NA	
Race/Ethnicity						
White, non-Hispanic	8,453	7,836	–7.3	4.5	4.2	–6.7
Black, non-Hispanic	7,592	9,634	+26.9	27.1	33.0	+21.8
Hispanic	3,092	4,782	+54.7	17.3	21.4	+23.7
Asian/Pacific Islander	2,530	3,027	+19.6	46.0	41.6	–9.6
American Indian/ Alaskan Native	397	371	–6.5	24.9	18.9	–24.1
Unknown/Other¶	137	51	–	NA	NA	
Country of origin	**					
Foreign-born	4,925	6,262	+27.1	NA	NA	
U.S.-born	17,712	18,997	+7.3	NA	NA	
Unknown	131	442	–	NA	NA	

* Per 100,000 population.
† –Not calculated.
§ NA = Denominator data not available.
¶ Includes blacks and whites of unknown ethnicity.
** Cases reported for 1986, the first year with uniform national reporting of country of origin for persons with tuberculosis.

FIGURE 1. Observed and expected tuberculosis cases, United States, 1980–1990

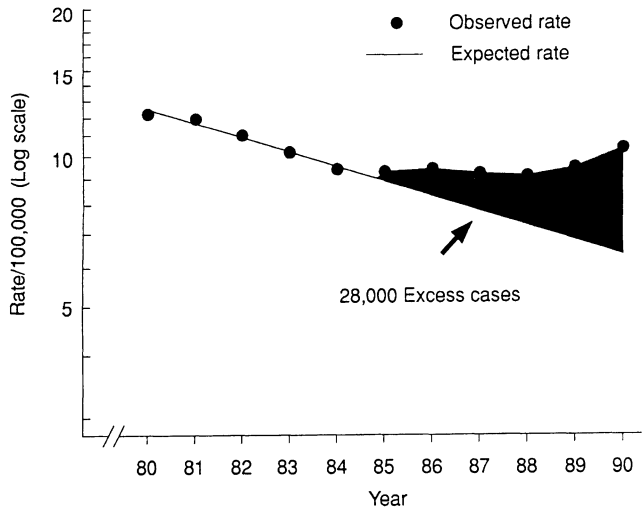
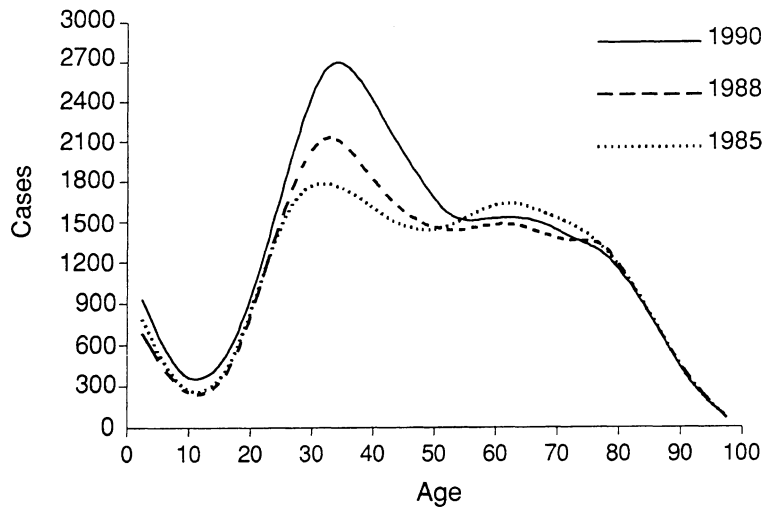


FIGURE 2. Age distribution of reported tuberculosis cases by year of report, 1985–1990



Regional and Temporal Trends in the Surveillance of Syphilis, United States, 1986–1990

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Summary

During the latter half of the 1980s, an epidemic of syphilis occurred throughout the United States. A comparison of regional rates of primary and secondary syphilis in 1990 indicated that the rates were highest in the South, followed by the Northeast, the West, and the Midwest. Primary and secondary syphilis rates from 1986 through 1990 exhibited different regional patterns. Rates of primary and secondary syphilis in the West peaked in 1987 and declined from 1987 to 1990. Rates increased in the Northeast and the South from 1986 to 1990, but the increase reached a plateau in the Northeast in 1990. Rates did not begin to increase in the Midwest until 1988. More detailed analyses of the syphilis epidemics in specific communities in each region are needed to better understand the regional patterns. A comparison of these findings across regions could be helpful in evaluating which sexually transmitted disease intervention and control programs are most effective during epidemic periods.

INTRODUCTION

Cases of primary and secondary syphilis, the most infectious stages of the disease, have been increasing annually in the United States since 1986. More than 50,000 cases were reported in 1990, the most cases reported since 1948. This epidemic of syphilis is occurring throughout all regions of the nation, but the magnitude and pattern of the epidemic have differed across regions during the past 5 years. To examine possible explanations for these regional differences, we analyzed data on reported cases of primary and secondary syphilis for 1986 through 1990.

METHODS

Summary data on cases of syphilis reported to state health departments from 1986 through 1990 were sent quarterly and annually to CDC in Atlanta, Georgia. The quarterly data from each state included total number of syphilis cases by gender, stage of disease (primary, secondary, early latent, and late latent), and source of report (public, private, or military). The annual data from each state included total number of primary and secondary syphilis cases by gender, racial/ethnic group (white, not of Hispanic origin; black, not of Hispanic origin; Hispanic; Asian/Pacific Islander; or American Indian/Alaskan Native), 5-year age group, and source of report.

Regional incidences of primary and secondary syphilis were calculated by using intercensal estimates of the population for 1986 through 1990 (*1*). States were grouped into the four regions of the United States as defined by the Bureau of the

Census: Northeast (Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont); South (Alabama, Arkansas, Delaware, District of Columbia, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia, West Virginia); Midwest (Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, Wisconsin); and West (Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, Wyoming). Because race-specific intercensal population estimates were available only for whites, blacks, and all others, primary and secondary syphilis cases among Hispanics were redistributed among these three race groups as follows: white (95%); black (2.5%); and all others (2.5%) (unpublished data, Bureau of the Census). Regional race-specific incidences were then calculated for whites, blacks, and all others. To facilitate comparisons, we standardized regional rates of primary and secondary syphilis by race, age, and gender by the direct method of standardization, using 1990 United States race-, age-, and gender-specific population estimates as the standard population (*1*).

RESULTS

In 1990, the incidences of primary and secondary syphilis were highest in the South, followed by the Northeast, the West, and the Midwest (Table 1). Across all regions, primary syphilis rates were higher among men than women, although rates of secondary syphilis were higher among women. Cases of primary and secondary syphilis represented approximately 23% of all reported syphilis cases in the West, 25% in the Northeast, almost 30% in the South, and approximately 32% in the Midwest.

During the period 1986–1990, the incidences of primary and secondary syphilis for men and women increased in the South (Figures 1 and 2). The incidences also increased in the Northeast but appear to have leveled off in 1990. In contrast, the primary and secondary syphilis incidences peaked in the West in 1987 and declined from 1988 to 1990. The rates in the Midwest did not begin to increase until 1988.

In 1990, the crude primary and secondary syphilis rate in the Northeast (22.3 cases per 100,000 population) was 33% lower than the comparable rate in the South (33.5 cases per 100,000 population) (Table 2). Standardization of these rates by race resulted in similar primary and secondary syphilis rates in the Northeast and South (22.4 and 23.3 cases per 100,000 population, respectively). On the other hand, even after the data were adjusted for racial differences, rates in the West were still approximately 30% lower than rates in the South and Northeast, and rates in the Midwest were 60% lower. Compared with the other regions, a much higher percentage of syphilis cases in the South (almost 85%) was reported from public sources (Table 3). The percentage of syphilis cases among blacks reported from public sources was higher than the percentage of cases among whites in all regions of the United States.

DISCUSSION

In 1990, primary and secondary syphilis rates were highest in the South and Northeast, with lower rates in the West and the lowest rates in the Midwest. These regional differences persisted even after the data were adjusted for differences in race-specific rates and different racial distributions of the regional populations.

It is unlikely that ascertainment bias could account for these regional differences. Specifically, the higher proportion of cases reported from public sources in the South might indicate a greater underreporting of cases from the private sector in that region. However, this would imply that the reported primary and secondary syphilis rates in the South were underestimated to a greater extent than in the other regions and that the regional differences were actually larger than reported. An evaluation of the completeness of syphilis surveillance is needed to determine the extent of underreporting in each region. The higher proportion of cases reported from public sources in the South may not reflect an ascertainment bias but simply a greater utilization of public clinics in that region.

Confounding by race, age, or gender also could not explain the regional differences in primary and secondary syphilis rates in 1990. Although the race distribution of the population did account for differences between rates in the South and Northeast, it did not account for the 30% and 60% lower rates of primary and secondary syphilis in the West and Midwest, respectively. Additional standardization of the rates by gender and age did not change these results.

The first evidence of the current syphilis epidemic was in the West, where primary and secondary syphilis rates began to increase in 1986. These rates peaked in that region in 1987 and decreased almost 41% between 1987 and 1990. The epidemic began in 1987 in the Northeast and the South. The rates in the Northeast increased 153% from 1986 to 1990. However, the rates increased less than 1% from 1989 to 1990, and preliminary 1991 data show a decrease in the number of primary and secondary syphilis cases being reported in that region. This decrease suggests that the epidemic may have peaked in the Northeast. The primary and secondary syphilis rates in the South increased 82% from 1986 to 1990, but preliminary 1991 data suggest that the epidemic may be leveling off. On the other hand, rates of primary and secondary syphilis did not begin to increase in the Midwest until 1988 and increased 188% from 1988 to 1990. Preliminary 1991 data continue to show an increase in the epidemic in the Midwest.

Because early and late latent stages represent syphilis of longer duration than primary and secondary stages, trends in early latent and late latent syphilis rates echo those of the primary and secondary stages, peaking somewhat later in time. If these patterns are true, primary and secondary syphilis cases may account for a lower proportion of total syphilis cases in the areas where the epidemic first started. In 1990, primary and secondary syphilis cases made up the lowest proportion of total syphilis cases in the West, where the epidemic peaked in 1987. In contrast, primary and secondary syphilis cases represented over 31% of the total 1990 syphilis cases in the Midwest, where the epidemic did not start until 1988.

Regional trends in syphilis rates represent a summation of trends in communities in each region. More detailed analyses of the patterns of syphilis on the community level are needed to better understand the regional syphilis epidemics and to determine to what extent sexually transmitted disease program activity affects syphilis reporting and thus syphilis surveillance. Furthermore, a comparison of the characteristics of the current epidemic with those of previous epidemics would be useful in identifying regional cyclical patterns in the incidence of this disease. These findings could be helpful in elucidating possible reasons for regional differences in syphilis morbidity levels and in evaluating sexually transmitted disease intervention and control programs in each region during epidemic periods.

Reference
1. Irwin R. 1980–1990 Intercensal population estimates by race, sex, and age (machine-readable data file). Alexandria, Virginia: Demo-Detail, 1991.

TABLE 1. Crude rates of syphilis per 100,000 population, by region, stage of disease, and gender, United States, 1990

Stage of disease / Gender	Region			
	South	Northeast	West	Midwest
Primary syphilis				
Male	21.5	14.3	8.7	4.8
Female	5.9	4.7	1.9	1.1
Total	13.5	9.3	5.3	2.9
Secondary syphilis				
Male	16.3	11.7	4.7	4.1
Female	23.5	14.2	5.9	5.0
Total	20.0	13.0	5.3	4.5
Primary and secondary syphilis				
Male	37.9	26.1	13.4	8.9
Female	29.4	18.9	7.8	6.1
Total	33.5	22.3	10.6	7.5
All stages*				
Male	122.4	95.6	52.0	26.5
Female	104.4	82.3	41.5	21.1
Total	113.2	88.6	46.7	23.7

*Includes all reported cases of primary, secondary, early latent, and late latent syphilis.

TABLE 2. Proportion of the population by race, crude race-specific primary and secondary (P&S) syphilis rates, and crude and standardized P&S rates in each region, United States, 1990

	White		Black		All other		All races		
Region	% of Pop.	Crude P&S rate	% of Pop.	Crude P&S rate	% of Pop.	Crude P&S rate	Crude P&S rate	Std. P&S rate*	Std. P&S ratio*
South	79.2	4.4	19.0	156.2	1.9	6.7	33.5	23.3	1.0(Ref)
Northeast	86.2	4.9	11.4	146.7	2.4	1.4	22.3	22.4	1.0
West	85.1	5.2	5.5	103.4	9.4	2.5	10.6	17.3	0.7
Midwest	88.2	1.0	10.0	65.5	1.8	1.2	7.5	9.0	0.4

*Standardized to the 1990 U.S. population by race (white, black, other).

TABLE 3. Percentages of primary and secondary syphilis cases reported from public sources, by region and race, United States, 1990

Region	White	Black	All other	All races
South	75.6	86.2	75.5	84.9
Northeast	44.5	63.2	37.8	56.9
West	63.3	69.8	64.8	66.0
Midwest	50.8	72.5	92.2	69.9

FIGURE 1. Primary and secondary syphilis rates among males, by region, United States, 1986–1990

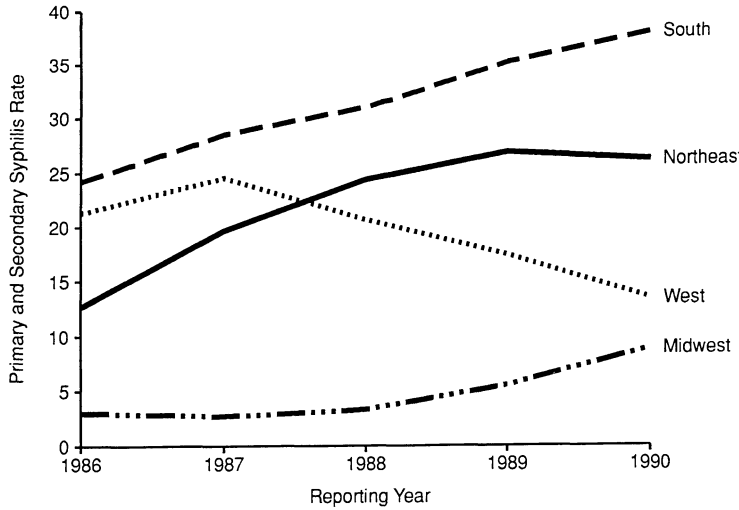
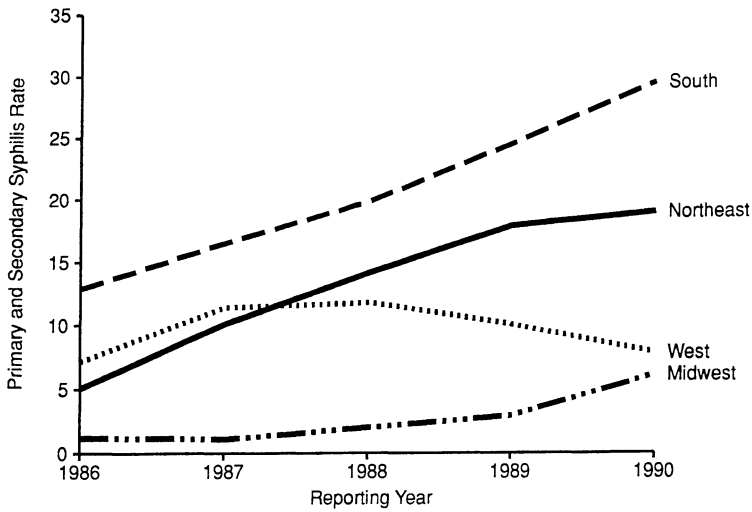


FIGURE 2. Primary and secondary syphilis rates among females, by region, United States, 1988–1990



Trichinosis Surveillance, United States, 1987-1990

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Summary

Since the Public Health Service began recording statistics on trichinosis in 1947, the number of cases reported by state health departments each year has declined. In the late 1940s, health departments reported an average of 400 cases and 10-15 deaths each year; from 1982 through 1986, the number declined to an average of 57 cases per year and a total of three deaths for the period.

From 1987 through 1990, 206 cases of trichinosis from 22 states, including 14 multiple-case outbreaks, were reported to CDC. In 1990, two large outbreaks associated with commercial pork accounted for 106 cases.

In the 192 instances in which a suspect food item was identified, pork was implicated in 144 (75%) cases, walrus meat in 34 (18%), and bear meat in 14 (7%). Sausage, the most frequently implicated pork product, was associated with 128 of the 139 cases for which a form of ingested pork was specified. Before 1990, the proportion of cases of trichinosis attributable to consumption of commercial pork had declined steadily. This decline was probably due to a combination of factors, including laws prohibiting the feeding of garbage to hogs, the increased use of home freezers, and the practice of thoroughly cooking pork.

Although the incidence of trichinosis has decreased substantially since national reporting was initiated in 1947, a dramatic increase in 1990, resulting from two large outbreaks, emphasizes the need for further education and control measures.

INTRODUCTION

Human trichinosis, an infection with worldwide distribution, is caused by tissue-dwelling roundworms of the species *Trichinella spiralis* . The organism is acquired by eating raw or inadequately cooked meat products containing encysted larvae. Abdominal pain and diarrhea, the first symptoms of trichinosis (the gastrointestinal phase), correspond to the maturation of the adult worms in the first week after ingestion. The classic symptoms of periorbital edema, fever, and myalgia occur when the larvae are encysting in the muscle, approximately 2-3 weeks after the infected meat is eaten. Pork products continue to be the major source of infection in the United States, although game animals are also a source of contaminated meat products.

From 1982 to 1986, an average of only 57 cases were reported each year with three associated deaths for the period, in contrast to an average of 402 cases each year and a total of 57 deaths reported from 1947 to 1951 (*1,2*). This decline is primarily attributed to legislation prohibiting the feeding of raw garbage to swine, widespread

freezing of pork, and increased public awareness of the dangers associated with eating inadequately cooked pork products (*3*). No apparent change in the surveillance system can account for this trend.

METHODS

State health departments report new cases of trichinosis by week to the National Morbidity Reporting Service. Supplemental epidemiologic information is submitted by the reporting state on Surveillance Case Report Forms (CDC Form 54.7, Rev 2-90) to the Division of Parasitic Diseases (DPD), National Center for Infectious Diseases, CDC. Additional cases are identified through reported results of trichinosis serologic tests performed by the Parasitic Diseases Branch, DPD, and through investigations conducted by DPD.

The CDC case definition for trichinosis is as follows:

1. *Trichinella* -positive muscle biopsy or a positive serologic test for trichinosis in a patient with one or more clinical symptoms compatible with trichinosis, such as eosinophilia, fever, myalgia, and periorbital edema.
   OR
2. In an outbreak, at least one person must meet criterion #1. Associated cases are defined by either a positive serologic test for trichinosis or one or more clinical symptoms compatible with trichinosis (such as eosinophilia, fever, myalgia, and periorbital edema) among persons who shared the epidemiologically implicated meal or ate the implicated meat product.

As in the past, cases reported by the states are not included in this report if they are not accompanied by written surveillance reports or do not fit the case definition. For the purposes of data analysis, commercial pork products are defined as pork purchased at supermarkets, butcher shops, wholesale meat suppliers, or public eating places. Noncommercial pork products are specifically identified according to source, such as hunting or trapping.

RESULTS

From 1987 through 1990, 206 cases of trichinosis from 22 states, including 14 multiple-case outbreaks, were reported to CDC (Figure 1). One hundred fifty-four (75%) of these cases occurred in Iowa (78 cases), Alaska (38), Virginia (16), California (11), and New Jersey (11) (Table 1). The 14 common-source outbreaks accounted for 171 (83%) of the 206 cases (Table 2). One hundred one (49%) cases occurred among males, 103 (50%) occurred among females, and gender was not reported in two cases. The mean age of patients was 35.4 years, ranging from 1 to 81 years (Figure 2).

In many previous years, trichinosis in the United States had exhibited a seasonal pattern, with a peak in December and January related to eating homemade pork sausage during the Christmas holidays. However, from 1987 through 1990, only 9% of cases occurred in December and January, in contrast to 49% of cases reported in these months in 1986. One hundred eleven (60%) of the 184 cases for which the month of onset was reported occurred from June to August. Ninety of these 111 cases resulted from one outbreak in Iowa associated with a single meal in July 1990.

One hundred seventy-six (85%) patients reported at least one common symptom or sign of trichinosis: 159 (77%) had myalgia, 156 (76%) had fever, 132 (64%) had periorbital edema, and 97 (47%) had eosinophilia. One death was associated with acute trichinosis in 1987. One case of acute trichinosis involved a woman who was 16

weeks pregnant. The pregnancy was completed without complication, and the baby's physical examination at birth was normal.

The mean incubation period for the 152 cases for which the dates of consumption of the incriminated meat and the onset of symptoms were known was 20.5 days, with a range of 2 to 53 days. *Trichinella* serology was found to be positive in 110 (80%) of the 137 persons for whom the results were reported. Muscle biopsies were performed for 19 (9%) persons, and 18 of the biopsies were positive.

In the 192 instances in which a suspect food item was identified, pork was implicated in 144 (75%) cases, walrus meat in 34 (18%), and bear meat in 14 (7%) (Figure 3). Sausage, the most frequently implicated pork product, was associated with 128 of the 139 cases for which a form of ingested pork was specified (Table 3). The meat was examined in 55 cases and found positive for *Trichinella* larvae in 51 cases.

DISCUSSION

Although the incidence of trichinosis has decreased substantially since national reporting was initiated in 1947, a dramatic increase in 1990, resulting from two large outbreaks, emphasizes the continuing need for public education about the dangers of eating inadequately cooked pork. Despite this large increase in total cases reported in 1990, the mean number of cases per year for the period 1987–1990 was lower than the mean number of cases for the period 1982–1986 (51.5 vs. 57). If the 1990 Iowa outbreak had not occurred, the mean number of cases per year in 1987–1990 would have been 29 cases. Before 1990, the proportion of cases of trichinosis attributable to eating commercial pork had declined steadily (Figure 4). This decline was probably due to a combination of factors, including laws prohibiting the feeding of garbage to hogs, the increased use of home freezers, and the practice of thoroughly cooking pork. Many outbreaks in recent years have been associated with eating wild game, including bear, wild boar, and walrus (*2,4*).

The outbreak in Iowa was the fourth such event among Southeast Asian refugees in the United States in the past 15 years (*4,5*). The three previous outbreaks were related to eating undercooked pork that was not obtained from a commercial producer (*4,5*). Previous reports have emphasized that Southeast Asians are at particular risk for trichinosis because of their dietary habits, and the outbreak in 1990 supports this observation (*5*).

The outbreak in Virginia did not occur among persons of one distinct ethnic group but involved unrelated persons who obtained pork from the same supplier and routinely ate uncooked sausage (*4*). This outbreak emphasizes that not only Southeast Asians but any persons who eat undercooked pork are at risk for trichinosis.

The prevalence of *Trichinella* infection in commercial pork, as measured by examination of hogs at slaughterhouses, ranges from 0% to 0.7% (*6,7*). Any pork product sold as "ready to eat," which accounts for approximately 40% of the pork produced in the United States each year, must be made with trichina-free pork, or pork that has been adequately cooked or treated to kill trichina larvae. *Trichinella* larvae in pork are killed by freezing at -15 C for 21 days (longer if meat is >15 cm thick). However, because *Trichinella* larvae in wild game are often relatively resistant to freezing (*8*), cooking is the most reliable method of destroying *Trichinella* in any type of meat. A temperature of 170 F (77 C) is well above the thermal death point and is usually achieved if the meat is cooked until it is no longer pink (*9*).

Sporadic cases and outbreaks associated with eating commercial pork continue to occur. Health officials in areas with large populations of Southeast Asians should consider education programs directed at preventing trichinosis. Physicians need to be aware of the continued presence of *Trichinella spiralis* in commercial pork in the United States and should consider the diagnosis in any patient with an illness compatible with trichinosis whose dietary preferences put him or her at risk for acquiring this infection.

Recent analysis of cases reported in the United States from 1975 to 1989 revealed 26 cases acquired in association with foreign travel (*10*). This finding suggests that physicians must include trichinosis in their differential diagnosis of eosinophilia among persons returning from abroad and should also include trichinosis prevention measures in their pretravel counseling.

Public education will continue to play a prominent role in preventing illness among persons who eat wild animal meat. Even more important to the complete elimination of trichinosis in the United States are sustained efforts by state and local governments to enforce federal regulations regarding hog management, eliminate infected herds, and increase educational measures directed at both pork producers and the public.

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TABLE 1. Trichinosis cases, United States, 1987-1990

State	1987	1988	1989	1990	Total
Alaska	5	26	7	0	38
Arkansas	0	1	0	0	1
California	6	3	2	0	11
Colorado	0	0	0	1	1
Hawaii	0	1	1	0	2
Illinois	0	0	0	1	1
Iowa	0	0	0	78	78
Kansas	0	0	0	6	6
Massachusetts	0	1	6	0	7
Michigan	3	0	0	0	3
Minnesota	0	0	0	3	3
Missouri	0	0	1	0	1
New Jersey	9	0	2	0	11
New York (city)	1	0	0	0	1
New York (upstate)	0	2	2	1	5
North Carolina	1	0	1	0	2
Oklahoma	0	0	0	1	1
Pennsylvania	1	7	0	0	8
Virginia	0	0	0	16	16
Washington	1	0	4	0	5
West Virginia	0	0	1	0	1
Wisconsin	0	0	1	1	2
Unspecified	0	1	0	1	2
Total	**27**	**42**	**28**	**109**	**206**

TABLE 2. Multiple-case outbreaks of trichinosis, United States, 1987-1990

Year	State	Cases	Month of onset	Implicated meat
1987	New Jersey	9	March-April	Pork sausage
1987	Alaska	3	June	Polar bear
1987	California	2	July	Pork sausage
1987	California	2	December	Pork sausage
1988	Alaska	20	May-June	Walrus
1988	Alaska	7	June	Walrus
1988	California	2	November	Pork
1988	Pennsylvania	5	December	Black bear
1989	Alaska	2	June	Walrus
1989	Alaska	4	June	Walrus
1989	Massachusetts	5	June	Pork sausage
1989	Washington	4	November	Black bear
1990	Colorado, Iowa, Kansas, Minnesota, Wisconsin	90	July-August	Pork sausage*
1990	Virginia	16	November-December	Pork sausage
Total		**171**		

**som moo* – a Southeast Asian dish consisting of commercially purchased pork ground into sausage at home and eaten raw.

TABLE 3. Trichinosis cases by type and source of implicated meat product, United States, 1987-1990

Meat type	Cases	% of Total
Pork	125	60.7
Commercial	108	52.4
Sausage (*som moo*)*	90	43.7
Sausage	17	8.3
Roast	1	0.5
Direct from farm	15	7.3
Sausage	9	4.4
Chops	3	1.5
Roast	1	0.5
Unspecified	2	1.0
Hunted (Wild boar)	2	1.0
Sausage	1	0.5
Other meats	48	23.3
Bear meat	14	6.8
Walrus meat	34	16.5
Unknown	33	16.0
Total	**206**	**100.0**

*Southeast Asian pork sausage eaten raw, July-August 1990 outbreak

FIGURE 1. Reported trichinosis cases, United States, 1947–1990

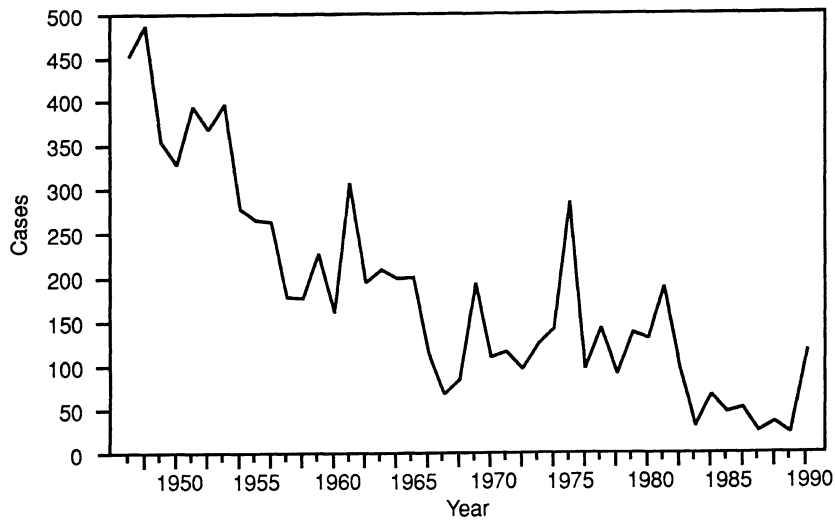


FIGURE 2. Trichinosis cases by gender and age, United States, 1987–1990

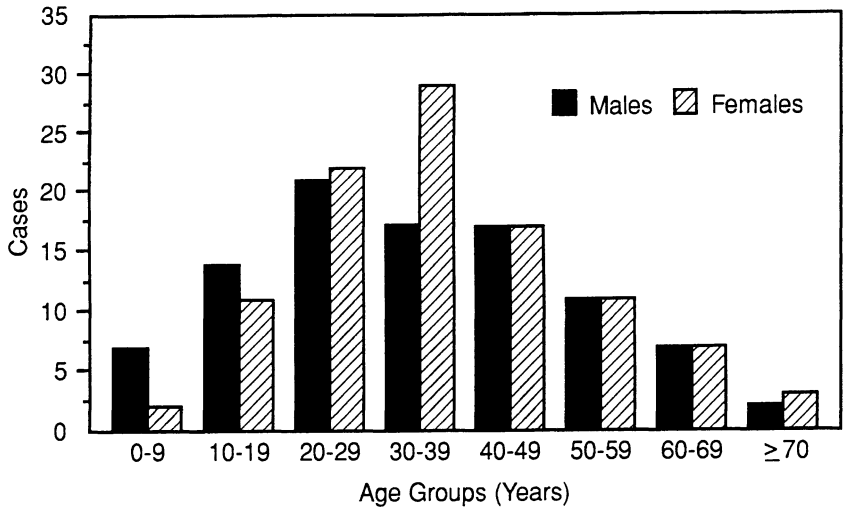


FIGURE 3. Trichinosis cases by month of onset, United States, 1987–1990

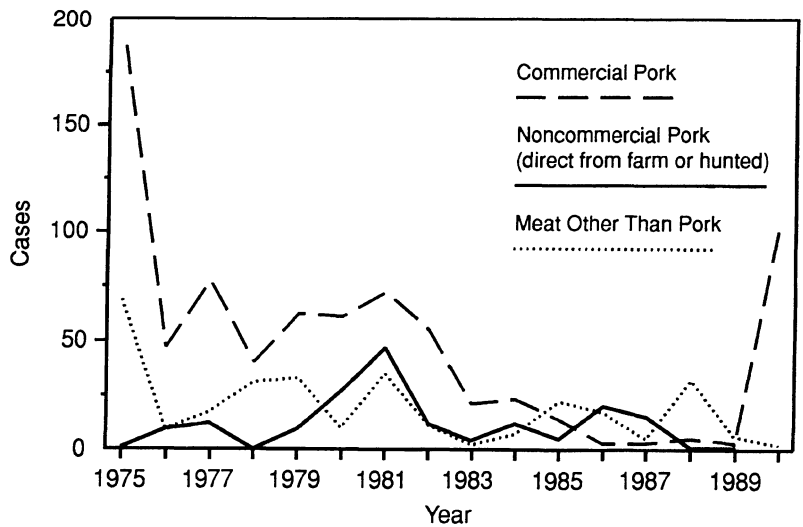
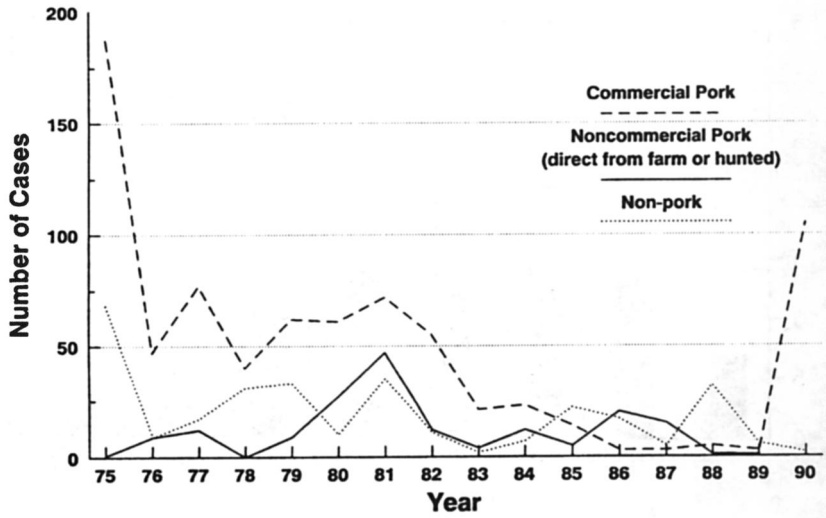


FIGURE 4. Trichinosis source of infection, United States, 1975–1990



State and Territorial Epidemiologists and Laboratory Directors

State and Territorial Epidemiologists and Laboratory Directors are gratefully acknowledged for their contributions to this report. The epidemiologists listed below were in the positions shown as of July 1991, and the laboratory directors listed below were in the positions shown as of April 1991.

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