CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

November 19, 1993 / Vol. 42 / No. SS-5

MMWR
MORBIDITY AND MORTALITY WEEKLY REPORT

CDC Surveillance Summaries

Surveillance for Waterborne Disease Outbreaks — United States, 1991–1992

Silicosis Surveillance — Michigan, New Jersey, Ohio, and Wisconsin, 1987–1990

Sensitivity of Multiple-Cause Mortality Data for Surveillance of Deaths Associated with Head or Neck Injuries

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

The *MMWR* series of publications is published by the Epidemiology Program Office, Centers for Disease Control and Prevention (CDC), Public Health Service, U.S. Department of Health and Human Services, Atlanta, Georgia 30333.

SUGGESTED CITATION

General: Centers for Disease Control and Prevention. *CDC Surveillance Summaries*, November 19, 1993. *MMWR* 1993;42(No. SS-5).

Specific: [Author(s).] [Title of particular article.] In: *CDC Surveillance Summaries,* November 19, 1993. *MMWR* 1993;42(No. SS-5):[inclusive page numbers].

Copies can be purchased from Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402-9325. Telephone: (202) 783-3238.

Contents

Most Recent Reports Published in CDC Surveillance Summaries

Subject	Responsible CIO*	Most Recent Report
Abortion	NCCDPHP	1992; Vol. 41, No. SS-5
AIDS/HIV		
Distribution by Racial/Ethnic Group	NCID	1988; Vol. 37, No. SS-3
Among Black and Hispanic Children and Women of Childbearing Age	NCEHIC	1990; Vol. 39, No. SS-3
Behavioral Risk Factors	NCCDPHP	1991; Vol. 40, No. SS-4
Birth Defects		
B.D. Monitoring Program (see also Malformations)	NCEH	1993; Vol. 42, No. SS-1
Contribution of B.D. to Infant Mortality Among Minority Groups	NCEHIC	1990; Vol. 39, No. SS-3
Breast and Cervical Cancer	NCCDPHP	1992; Vol. 41, No. SS-2
Campylobacter	NCID	1988; Vol. 37, No. SS-2
Chancroid	NCPS	1992; Vol. 41, No. SS-3
Chlamydia	NCPS	1993; Vol. 42, No. SS-3
Cholera	NCID	1992; Vol. 41, No. SS-1
Coal Workers' Health (see also Mining)	NIOSH	1985; Vol. 34, No. 1SS
Congenital Malformations, Minority Groups	NCEHIC	1988; Vol. 37, No. SS-3
Contraception Practices	NCCDPHP	1992; Vol. 41, No. SS-4
Cytomegalovirus Disease, Congenital	NCID	1992; Vol. 41, No. SS-2
Dengue	NCID	1985; Vol. 34, No. 2SS
Dental Caries and Periodontal Disease Among Mexican-American Children	NCPS	1988; Vol. 37, No. SS-3
Diabetes Mellitus	NCCDPHP	1993; Vol. 42, No. SS-2
Dracunculiasis	NCID	1992; Vol. 41, No. SS-1
Ectopic Pregnancy	NCCDPHP	1990; Vol. 39, No. SS-4
Elderly, Hospitalizations Among	NCCDPHP	1991; Vol. 40, No. SS-1
Endometrial and Ovarian Cancers	EPO, NCCDPHP	1986; Vol. 35, No. 2SS
Escherichia coli O157	NCID	1991; Vol. 40, No. SS-1
Evacuation Camps	EPO	1992; Vol. 41, No. SS-4
Foodborne Disease	NCID	1990; Vol. 39, No. SS-1
Gonococcal Infection	NCPS, NCID	1984; Vol. 33, No. 4SS
Gonorrhea and Syphilis, Teenagers	NCPS	1993; Vol. 42, No. SS-3
Health Surveillance Systems	IHPO	1992; Vol. 41, No. SS-4
Hepatitis	NCID	1985; Vol. 34, No. 1SS
Hepatitis, Viral	NCID	1983; Vol. 32, No. 2SS
Homicide	NCEHIC	1992; Vol. 41, No. SS-3
Homicides, Black Males	NCEHIC	1988; Vol. 37, No. SS-1
Hysterectomy	NCCDPHP	1986; Vol. 35, No. 1SS
Infant Mortality (see also National Infant Mortality; Birth Defects; Postneonatal Mortality)	NCEHIC	1990; Vol. 39, No. SS-3
Influenza	NCID	1993; Vol. 42, No. SS-1
Injury		
Death Rates, Blacks and Whites	NCEHIC	1988; Vol. 37, No. SS-3
Drownings	NCEHIC	1988; Vol. 37, No. SS-1
Falls, Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Firearm-Related Deaths, Unintentional	NCEHIC	1988; Vol. 37, No. SS-1
Head & Neck	NCIPC	1993; Vol. 42, No. SS-5
In Developing Countries	NCEHIC	1992; Vol. 41, No. SS-1
In the Home, Persons <15 Years of Age	NCEHIC	1988; Vol. 37, No. SS-1

Abbreviations*

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

Most Recent Reports Published in CDC Surveillance Summaries — Continued

Subject	Responsible CIO*	Most Recent Report
Motor Vehicle-Related Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Objectives of Injury Control, State and Local	NCEHIC	1988; Vol. 37, No. SS-1
Objectives of Injury Control, National	NCEHIC	1988; Vol. 37, No. SS-1
Residential Fires, Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Tap Water Scalds	NCEHIC	1988; Vol. 37, No. SS-1
Lead Poisoning, Childhood	NCEHIC	1990; Vol. 39, No. SS-4
Low Birth Weight	NCCDPHP	1990; Vol. 39, No. SS-3
Malaria, Imported	NCID	1983; Vol. 32, No. 3SS
Malformations (see also Birth Defects)	NCEHIC	1985; Vol. 34, No. 2SS
Maternal Mortality	NCCDPHP	1991; Vol. 40, No. SS-2
Measles	NCPS	1992; Vol. 41, No. SS-6
Meningococcal Disease	NCID	1993; Vol. 42, No. SS-2
Mining (see also Coal Workers' Health)	NIOSH	1986; Vol. 35, No. 2SS
National Infant Mortality (see also Infant Mortality; Birth Defects)	NCCDPHP	1989; Vol. 38, No. SS-3
Neisseria gonorrhoeae, Antimicrobial Resistance in	NCPS	1993; Vol. 42, No. SS-3
Nosocomial Infection	NCID	1986; Vol. 35, No. 1SS
Occupational Injuries/Disease		
Among Loggers	NIOSH	1983; Vol. 32, No. 3SS
Hazards, Occupational	NIOSH	1985; Vol. 34, No. 2SS
In Meatpacking Industry	NIOSH	1985; Vol. 34, No. 1SS
Silicosis	NIOSH	1993; Vol. 42, No. SS-5
State Activities	NIOSH	1987; Vol. 36, No. SS-2
Treated in Hospital Emergency Rooms	NIOSH	1983; Vol. 32, No. 2SS
Ovarian Cancer (see Endometrial and Ovarian Cancers)		
Parasites, Intestinal	NCID	1991; Vol. 40, No. SS-4
Pediatric Nutrition	NCCDPHP	1992; Vol. 41, No. SS-7
Pelvic Inflammatory Disease	NCPS	1983; Vol. 32, No. 4SS
Pertussis	NCPS	1992; Vol. 41, No. SS-8
Plague	NCID	1985; Vol. 34, No. 2SS
Plague, American Indians	NCID	1988; Vol. 37, No. SS-3
Pneumoconiosis, Coal Miners	NIOSH	1983; Vol. 32, No. 1SS
Poliomyelitis	NCPS	1992; Vol. 41, No. SS-1
Postneonatal Mortality	NCCDPHP	1991; Vol. 40, No. SS-2
Pregnancy Nutrition	NCCDPHP	1992; Vol. 41, No. SS-7
Pregnancy, Teenage	NCCDPHP	1987; Vol. 36, No. 1SS
Psittacosis	NCID	1983; Vol. 32, No. 1SS
Rabies	NCID	1989; Vol. 38, No. SS-1
Racial/Ethnic Minority Groups	Various	1990; Vol. 39, No. SS-3
Respiratory Disease	NCEHIC	1992; Vol. 41, No. SS-4
Reye Syndrome	NCID	1984; Vol. 33, No. 3SS
Rocky Mountain Spotted Fever	NCID	1984; Vol. 33, No. 3SS
Rotavirus	NCID	1992; Vol. 41, No. SS-3
Rubella and Congenital Rubella	NCPS	1984; Vol. 33, No. 4SS
Salmonella	NCID	1988; Vol. 37, No. SS-2
Sexually Transmitted Diseases in Italy	NCPS	1992; Vol. 41, No. SS-1
Smoking	NCCDPHP	1990; Vol. 39, No. SS-3
Streptococcal Disease (Group B)	NCID	1992; Vol. 41, No. SS-6
Sudden Unexplained Death Syndrome Among Southeast Asian Refugees	NCEHIC, NCPS	1987; Vol. 36, No. 1SS
Suicides, Persons 15–24 Years of Age	NCEHIC	1988; Vol. 37, No. SS-1
Summer Mortality	NCEHIC	1983; Vol. 32, No. 1SS
Syphilis, Primary and Secondary	NCPS	1993; Vol. 42, No. SS-3
Tetanus	NCPS	1992; Vol. 41, No. SS-8
Toxic-Shock Syndrome	NCID	1984; Vol. 33, No. 3SS
Trichinosis	NCID	1991; Vol. 40, No. SS-3
Tubal Sterilization Among Women	NCCDPHP	1983; Vol. 32, No. 3SS
Tuberculosis	NCPS	1991; Vol. 40, No. SS-3
Waterborne Disease Outbreaks	NCID	1993; Vol. 42, No. SS-5
Years of Potential Life Lost	EPO	1992; Vol. 41, No. SS-6

Blank

Surveillance for Waterborne Disease Outbreaks—United States, 1991–1992

Anne C. Moore, M.D., Ph.D.[1]
Barbara L. Herwaldt, M.D., M.P.H.[1]
Gunther F. Craun, P.E., D.E.E., M.P.H.[2]
Rebecca L. Calderon, Ph.D.[3]
Anita K. Highsmith, M.S.[4]
Dennis D. Juranek, D.V.M., M.Sc.[1]

[1]*Parasitic Diseases Branch, Division of Parasitic Diseases, National Center for Infectious Diseases (NCID), CDC*
[2]*Global Consulting for Environmental Health, Radford, VA*
[3]*Health Studies Division, Health Effects Research Laboratory, U.S. Environmental Protection Agency*
[4]*Water Quality Laboratory, Scientific Resources Program, NCID, CDC*

Summary

Problem/Condition: Since 1971, CDC and the U.S. Environmental Protection Agency have maintained a collaborative surveillance program for collection and periodic reporting of data on the occurrence and causes of waterborne disease outbreaks.

Reporting Period Covered: January 1991 through December 1992.

Description of System: The surveillance system includes data about outbreaks associated with water intended for drinking and also about those associated with recreational water. State and local public health departments are the agencies with primary responsibility for the detection and investigation of outbreaks. State and territorial health departments report these outbreaks to CDC on a standard form.

Results: For the 2-year period 1991–1992, 17 states and territories reported 34 outbreaks associated with water intended for drinking. The outbreaks caused an estimated 17,464 persons to become ill. A protozoal parasite (*Giardia lamblia* or *Cryptosporidium*) was identified as the etiologic agent for seven of the 11 outbreaks for which an agent was determined. Five (71%) of the outbreaks caused by protozoa were associated with a surface-influenced groundwater source. One outbreak of cryptosporidiosis was associated with filtered and chlorinated surface water. *Shigella sonnei* and hepatitis A virus were implicated in one outbreak each; both were linked to consumption of contaminated well water. Two outbreaks due to acute chemical poisoning were reported; one had an associated fatality. No etiology was established for 23 (68%) of the 34 outbreaks, including the largest one reported during this period, in which an estimated 9,847 persons using a filtered surface water supply developed gastroenteritis. Most (76%) of the 34 outbreaks were associated with a well water source.

Twenty-one states reported 39 outbreaks associated with recreational water, in which an estimated 1,825 persons became ill. The most frequently reported illness was hot tub- or whirlpool-associated *Pseudomonas* dermatitis (12 outbreaks). Of 11 outbreaks of swimming-associated gastroenteritis, six were caused by *Giardia* or

Cryptosporidium, including three outbreaks associated with chlorinated, filtered pool water. The first reported outbreak of *Escherichia coli* O157:H7 infection associated with recreational exposure occurred during this period. Primary amebic meningoencephalitis, caused by *Naegleria fowleri* infection, resulted in six deaths.

Interpretation: The number of waterborne disease outbreaks reported per year has not changed substantially in the past 5 years. However, etiologic agents only recently associated with waterborne disease, such as *E. coli* O157:H7 and *Cryptosporidium*, are being reported more frequently and from new settings. Water quality data for outbreaks during the period 1991–1992 indicate that available water disinfection technology is not always in place or used reliably. However, the high percentage of outbreaks attributed to relatively chlorine-resistant protozoa suggests that improvements in monitoring and treatment of potable water may be needed.

Actions Taken: Surveillance data, which identify the types of water systems and their deficiencies and the etiologic agents associated with outbreaks, are used to evaluate the adequacy of current technologies for providing safe drinking and recreational water, establish research priorities, and assist in improving water quality regulations.

INTRODUCTION

The reporting of waterborne disease outbreaks (WBDOs) is voluntary in the United States. National statistics on outbreaks associated with water intended for drinking have been available since 1920 (*1*). Since 1971, CDC and the Environmental Protection Agency (EPA) have maintained a collaborative surveillance program with collection and periodic reporting of data on the occurrence and causes of waterborne outbreaks (*2,3*). This summary includes data for 1991 and 1992 and for previously unreported outbreaks in 1988 and 1989.

The surveillance program includes data for outbreaks associated with water intended for drinking and with recreational water. Previous summaries have reported data for foodborne outbreaks of gastroenteritis on oceangoing passenger vessels that call on U.S. ports. Because these data are not related to the goals of the surveillance program, they will no longer be included in these summaries.

CDC and EPA activities related to waterborne disease surveillance have the following goals: a) to characterize the epidemiology of waterborne diseases; b) to identify deficiencies in water systems and the etiologic agents causing outbreaks; c) to train public health personnel in investigating WBDOs; and d) to collaborate with local, state, and other federal and international agencies on initiatives to prevent waterborne disease. The data gathered through surveillance are useful for evaluating the adequacy of current treatment technologies for providing safe drinking and recreational water. Surveillance information influences research priorities and may lead to improved water quality regulations.

State health departments can request epidemiologic assistance from CDC in the investigation of 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 pathogenic bacteria, viruses, or parasites.

EPA REGULATIONS FOR WATER INTENDED FOR DRINKING

Public water systems are regulated under the Safe Drinking Water Act (SWDA) (PL 93-523) of 1974. The microbial content of drinking water is regulated by EPA through the Total Coliform Rule (54 FR 27544-27568) and the Surface Water Treatment Requirements (SWTR) (54 FR 27486-27541). A maximum contaminant level for total coliforms specifies the percentage of samples that may contain any coliforms during a month. The turbidity of finished water must meet specified maximum and monthly standards. All public systems using surface water or groundwater under the direct influence of surface water must provide disinfection. Systems must also filter the water unless they meet specific conditions, including source water quality criteria for turbidity and total or fecal coliforms and a watershed control program to minimize potential contamination by human enteric viruses and *Giardia* cysts. EPA is considering a groundwater disinfection rule and revisions to the SWTR.

METHODS

Sources of Data

State and local public health agencies are primarily responsible for the detection and investigation of disease outbreaks. State health departments voluntarily report WBDOs to CDC on a standard form (CDC form 52.12, Rev. 02-91). In December 1991 and 1992, CDC personnel sent requests for reports to the state and territorial epidemiologists or to persons designated as coordinators of WBDO surveillance. Personnel in states that did not respond to the letters were contacted by telephone. In addition, personnel from the EPA Health Effects Research Laboratory contacted state water supply agencies to obtain information about WBDOs.

Definition of Terms

The surveillance system for WBDOs differs from other systems in that the unit of analysis is an outbreak rather than an individual case of a particular disease. Two criteria must be met for an event to be defined as a WBDO. First, at least two persons must have experienced a similar illness after ingestion of water intended for drinking or after exposure to water used for recreational purposes. Second, epidemiologic evidence must implicate water as the source of the illness. The stipulation that at least two persons be ill is waived for single cases of laboratory-confirmed primary amebic meningoencephalitis and for single cases of chemical poisoning (if water quality data indicate contamination by the chemical). If primary and secondary cases are distinguished on the outbreak report form, only primary cases are included in the case counts.

Community and noncommunity public water systems are regulated under the SDWA. *Community water systems* are defined as public or investor-owned systems that serve large or small communities, subdivisions, or mobile-home parks with at least 15 service connections or 25 year-round residents. *Noncommunity water systems* serve institutions, industries, camps, parks, hotels, or businesses that may be used by the general public. Of the approximately 200,000 water systems in the U.S. classified as public, 30% (60,000) are community water systems and 70% (140,000) are noncommunity systems. Community water systems serve 91% of the U.S. population; the remaining 9% are served by nonpublic or individual systems, usually wells or

springs, used by one or several residences or by persons traveling outside populated areas.

Deficiencies in water systems are classified as follows:

1 = untreated surface water (e.g., from rivers, lakes, or reservoirs);
2 = untreated groundwater (e.g., from wells or springs);
3 = treatment deficiency (e.g., temporary interruption of disinfection, chronically inadequate disinfection, filtration absent or inadequate);
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.

If more than one deficiency was reported for an outbreak, only the most important is noted in the line listings. Outbreaks due to contamination of water or ice at the point of use (e.g., a contaminated serving container) are not included in the line listings.

Recreational waters are categorized as freshwater swimming pools, whirlpools, and naturally occurring fresh and marine surface waters. Although the surveillance system includes whirlpool- and hot tub-associated outbreaks of dermatitis due to *Pseudomonas*, it does not include wound infections caused by waterborne organisms, such as *Aeromonas* species.

Classification of Outbreaks

In this surveillance system, outbreaks are classified according to the strength of the evidence implicating water (Table 1). Each outbreak, except single cases of illness resulting from chemical poisoning and primary amebic meningoencephalitis, is classified (I through IV) based on the epidemiologic data and the presence or absence of water quality data on the report form. Epidemiologic data are weighted more heavily than water quality data. Thus, in this summary, some outbreaks without water quality data were included, but reports without supporting epidemiologic data were omitted. The classification numbers are included in the line listings to indicate what data were available. Classification numbers of II-IV do not necessarily imply that the investigations were flawed; the circumstances of each outbreak differ, and not all outbreaks can or should be rigorously investigated. A classification of I means that both epidemiologic and water quality data were reported but does not necessarily imply that the investigation was optimal.

RESULTS

Outbreaks Associated with Water Intended for Drinking

For the 2-year period 1991–1992, 17 states and territories reported 34 outbreaks associated with water intended for drinking. The outbreaks caused illness in an estimated 17,464 persons. Twenty-three outbreak reports (68%) were classified as Class I (i.e., adequate epidemiologic and water quality data were provided). Outbreaks are listed individually by state (Tables 2 and 3) and are tabulated by etiologic agent and type of water system (Table 4) and by water system deficiency (Table 5).

Fifteen outbreaks were reported for 1991 and 19 for 1992. No outbreaks were reported for the months of October, November, or December. The month with the most outbreaks (nine) was June (Figure 1). The median outbreak size was 57 persons (range, 1–9,847). Twelve (35%) of the outbreaks were reported from Pennsylvania.

Thirty-one (91%) of the outbreaks caused gastroenteritis. The other waterborne illnesses were chemical poisoning (two outbreaks) and hepatitis (one outbreak). Of the estimated 17,464 persons reported ill, 40 persons were hospitalized and one died. Of the hospitalized persons, 29 had acute gastrointestinal illness of unknown etiology (AGI), four had cryptosporidiosis, three had hepatitis A infection, two had giardiasis, one had nitrate intoxication, and one had fluoride intoxication. The reported death occurred after fluoride poisoning.

Etiologic Agents

A protozoal parasite (*Giardia lamblia* or *Cryptosporidium*) was identified as the causative agent in seven outbreaks, representing 21% of the 34 outbreaks and 64% of the 11 WBDOs for which an etiology was determined. The four outbreaks of giardiasis, which affected an estimated 123 persons, were reported from California, Idaho, Nevada, and Pennsylvania. The outbreaks occurred in March (two), July (one), and September (one). Two were associated with community water systems and two with noncommunity systems. In the Nevada outbreak, the community was supplied by unfiltered surface water that contained low levels of *Giardia* cysts; however, neither source nor tap water had detectable coliforms present. Chlorination of finished water had not been maintained consistently in this system. The other three giardiasis outbreaks were associated with groundwater supplies. In one of these, a cross-connection resulted in contaminated surface water entering a system using a spring water source. In another, coliforms were present in a water sample, but the source of

TABLE 1. Classification of investigations of waterborne disease outbreaks

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 historical 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 claim 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.

contamination of either the well or the underground storage tanks was not determined. No deficiency was identified in the remaining outbreak, which was associated with a chlorinated well water source. In this outbreak, consumption of well water was implicated epidemiologically; untreated water analyzed 3 months after the outbreak did not contain coliforms.

Three outbreaks of cryptosporidiosis, which resulted in illness in an estimated 3,551 persons, occurred in Oregon (two) and Pennsylvania (one). They began in February, May, and August, respectively. Although the two Oregon outbreaks occurred in geographically adjacent locations and may have overlapped in time, epidemiologic evidence suggests they were separate outbreaks. Because of some overlap in the affected populations, case counts for the two outbreaks (Table 3) have been added together.

Two distinct water supplies were implicated in the Oregon outbreaks. The first outbreak was associated with a disinfected spring water source that supplied a community of 80,000 people. Low numbers of *Cryptosporidium* oocysts were found in water samples obtained over a 2-week period, but none were found in numerous samples collected for 10 weeks thereafter. Sporadic low levels of coliforms, algae, and diatoms suggested that the spring may have been influenced by surface water.

The other Oregon outbreak was associated with inadequate filtration of a river water source. The river received waste water discharges, and water quality had deteriorated because of low stream flow during dry weather (*4*). The presence of *Cryptosporidium* oocysts in the filtered water was not confirmed. However, a reliable examination was not possible because the filtered water contained an excessive number of oocyst-sized particles (e.g., algae). The turbidity of the filtered effluent water was elevated; however, mean levels did not exceed the limits of the EPA SWTR. For both Oregon outbreaks, a review of water quality records showed that each system

TABLE 2. Outbreaks associated with water intended for drinking — United States, 1991 (N=15)*

State†	Month	Class§	Etiologic agent¶	No. cases	Type of system**	Deficiency††	Source	Setting
CA	Jul	I	*Giardia*	15	NC	4	spring	recreation area
IL	May	II	AGI	386	NC	5	well	school
MI	Jun	I	AGI	1,320	NC	2	well	campground
MI	Aug	I	AGI	33	NC	2	well	resort
MN	Jun	I	AGI	30	NC	2	well	campground
MN	Jul	I	AGI	30	NC	4	well	resort
MN	Aug	I	AGI	17	NC	2	well	restaurant
NM	Aug	I	AGI	38	NC	2	well	camp
PA	Jun	I	AGI	170	NC	3	well	picnic area
PA	Jul	I	AGI	8	NC	3	well	restaurant
PA	Sept	III	*Giardia*	13	NC	3	well	park
PA	Aug	I	*Cryptosporidium*	551	NC	3	well	picnic area
PA	Jun	I	AGI	300	NC	3	well	camp
PR	Aug	I	AGI	202	Com	4	river	penitentiary
PR	Aug	I	AGI	9,847	Com	3	river	community

*See Methods section for description of reporting variables.
†Includes territories.
§See Table 1 for class definitions.
¶AGI=acute gastrointestinal illness of unknown etiology.
**NC=noncommunity; Com=community.
††See Methods section for definitions of deficiencies.

had consistently met the coliform and turbidity maximum contaminant level (MCL) over the past several years.

The third outbreak of cryptosporidiosis was associated with a noncommunity well water source. Detection of coccidian oocysts that were the size and shape of *Cryptosporidium*, algae, and diatoms indicated that the well was influenced by surface water. Finished water in all three outbreaks was free of coliforms, and chlorine levels were probably sufficient to inactivate bacteria but not *Cryptosporidium* oocysts, which are highly chlorine resistant.

Nonparasitic infectious etiologies were identified for only two outbreaks. One, a WBDO caused by *Shigella sonnei* , which resulted in illness in an estimated 150 persons, occurred in a park in which an untreated spring-water source was contaminated with surface water. *Shigella* was demonstrated both in water samples and in stool specimens. An outbreak of serologically confirmed hepatitis A infection, associated with an untreated well water supply (individual household), was the only outbreak in which a viral pathogen was identified.

Two outbreaks due to acute chemical intoxication were reported. A case of methemoglobinemia (*5*) was due to ingestion of infant formula diluted with water that contained elevated levels of nitrate and copper. The water was from a shallow well that supplied an individual household equipped with a reverse-osmosis membrane filter. The filter reduced elevated nitrate-nitrogen levels in well water (58 mg/L) to a concentration in tap water (9.9 mg/L) that was close to the MCL. A large outbreak of acute fluoride poisoning caused illness in 262 persons and one fatality. Symptoms included nausea, vomiting, diarrhea, abdominal pain, numbness, and tingling. Serum chemistry abnormalities, including elevated lactic acid dehydrogenase and phosphorus levels and low magnesium levels, persisted for at least a week in some ill persons.

TABLE 3. Outbreaks associated with water intended for drinking — United States, 1992 (N=19)*

State	Month	Class†	Etiologic agent§	No. cases	Type of system¶	Deficiency	Source	Setting
AK	May	I	Fluoride**	262	Com	3	well	community
ID	Mar	III	*Giardia*	15	Com	2	well	trailer park
MN	Feb	I	AGI	250	NC	3	lake	restaurant
NV	Mar	I	*Giardia*	80	Com	3	lake	community
NY	Apr	III	AGI	107	NC	4	well	restaurant
NC	Jan	I	AGI	200	NC	2	well	restaurant
OH	Jun	III	AGI	129	NC	4	well	campground
OR	Feb	I	*Cryptosporidium*	††	Com	3	spring	community
OR	May	I	*Cryptosporidium*	††	Com	3	river	community
PA	Mar	III	AGI	5	NC	3	well	restaurant
PA	May	II	AGI	28	Com	5	river	park
PA	Jun	III	AGI	38	Ind	2	well	private home
PA	Jun	III	AGI	42	NC	3	well	camp
PA	May	I	AGI	50	NC	3	well	camp
PA	May	III	AGI	57	NC	3	well	camp
PA	Aug	I	AGI	80	NC	3	well	camp
WA	Jun	I	Hepatitis A	10	Ind	2	well	private home
WI	Jun	I	Nitrate	1	Ind	3	well	farm
WY	Jul	I	*Shigella sonnei*	150	NC	2	well	park

*See Methods section for description of reporting variables.
†See Table 1 for class definitions.
§AGI=acute gastrointestinal illness of unknown etiology.
¶NC=noncommunity; Com=community; Ind=individual.
**Resulted in one death.
††Total estimated number of cases for the Oregon outbreaks was 3,000; see text.

The outbreak was attributed to improperly installed equipment and to inadequate monitoring of the community water system, which resulted in fluoride levels in the water that were 12-fold higher than recommended.

In 23 WBDOs (68%), no etiologic agent was identified. Fourteen of these outbreaks of AGI included cases in which the symptom complex, incubation period, and duration of illness were consistent with a viral syndrome. In 15 of the outbreaks of AGI, stool specimens were negative for bacterial pathogens. Stool specimens were examined for ova and parasites in three investigations and for viral pathogens in only one. Coliforms were found in water samples for 19 (83%) of the AGI outbreaks, and chlorination deficiencies were associated with two others. In the largest AGI outbreak, an estimated 9,847 persons in Puerto Rico became ill when the water system resumed operation after an interruption due to drought. Waste water discharges into the river water source, temporary lack of chlorination, filtration deficiencies, and insufficient flushing of old water from pipes and tanks were identified as possible factors contributing to the outbreak.

Water Quality Data

Water quality data were obtained within 1 month of the WBDO for 30 (94%) of the 32 outbreaks that had a known or suspected infectious etiology. For all 30 of these WBDOs, water was tested for coliforms; in five outbreaks, samples were also examined for protozoa. Coliforms were noted for 24 outbreaks (80%). Overall, for outbreaks with bacterial, viral, or unknown etiologies, coliform testing was positive in 21 (88%). However, for protozoal outbreaks, finished water collected within a month of the outbreak contained coliforms in only two (33%) of six. Water was shown to contain the etiologic agent for four outbreaks: *Shigella* (one), *Giardia* (two), and *Cryptosporidium* (one).

Water Supply

Twenty-three (68%) of the 34 WBDOs were associated with noncommunity systems and only eight (24%) with community systems, but the outbreaks in community systems resulted in 77% of the total cases (Table 4, Figure 2). Outbreaks in community

TABLE 4. Outbreaks associated with water intended for drinking, by etiologic agent and type of water system — United States, 1991–1992 (N=34)

	Type of water system*							
	Community		Noncommunity		Individual		Total	
Agent†	Outbreaks	Cases	Outbreaks	Cases	Outbreaks	Cases	Outbreaks	Cases
AGI	3	10,077	19	3,252	1	38	23	13,367
Giardia	2	95	2	28	0	0	4	123
Cryptosporidium	2	3,000	1	551	0	0	3	3,551
Hepatitis A	0	0	0	0	1	10	1	10
Shigella sonnei	0	0	1	150	0	0	1	150
Nitrate	0	0	0	0	1	1	1	1
Fluoride	1	262	0	0	0	0	1	262
Total	**8**	**13,434**	**23**	**3,981**	**3**	**49**	**34**	**17,464**
(Percent§)	(24)	(77)	(68)	(23)	(9)	(<1)	(100)	(100)

*See Methods section for description of types of water systems.
†AGI=acute gastrointestinal illness of unknown etiology.
§The percentage of 34 outbreaks or of 17,464 cases.

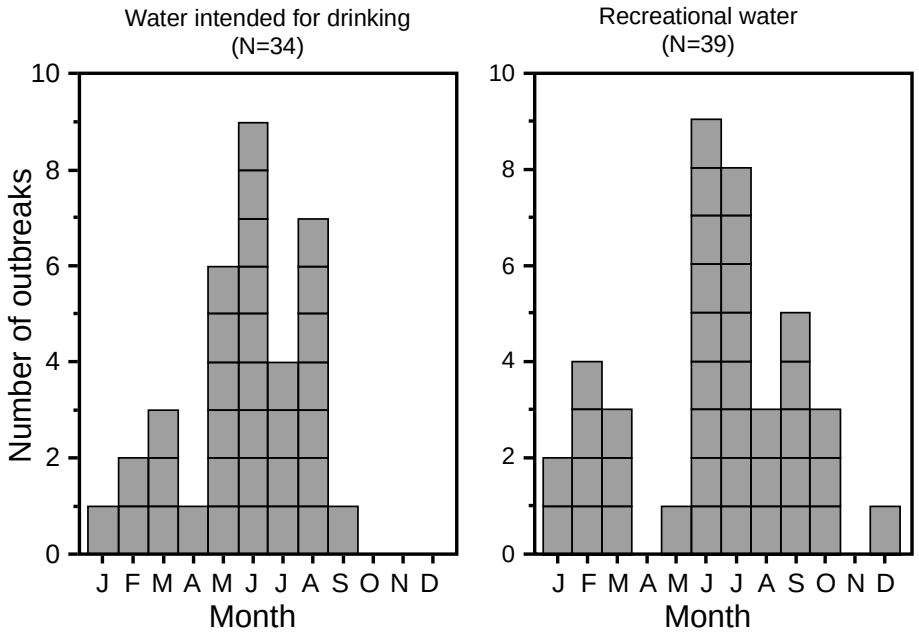
systems were primarily associated with surface water sources (63%). In contrast, only 4% of noncommunity outbreaks were associated with surface water.

Of the 34 outbreaks, 26 (76%) occurred in systems using well water. In 12 (46%) of the 26, the water was untreated. Inadequate or interrupted disinfection was the deficiency identified in another 12 (46%). Ten of these systems used chlorine disinfection; two systems used ultraviolet (UV) light. Of the remaining two outbreaks associated with treated well-water systems, one was attributed to a cross-connection with an unapproved supplemental pond water source, and no deficiency was reported for the other.

TABLE 5. Outbreaks associated with water intended for drinking, by type of deficiency and type of water system — United States, 1991–1992 (N=34)

	Type of water system							
	Community		Noncommunity		Individual		Total	
Type of deficiency	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Untreated surface water	0	(0)	0	(0)	0	(0)	0	(0)
Untreated groundwater	1	(13)	7	(30)	2	(67)	10	(29)
Treatment	5	(63)	11	(48)	1	(33)	17	(50)
Distribution system	1	(13)	4	(17)	0	(0)	5	(15)
Unknown	1	(13)	1	(4)	0	(0)	2	(6)
Total	**8**	**(100)**	**23**	**(100)**	**3**	**(100)**	**34**	**(100)**

FIGURE 1. Waterborne outbreaks, by month — United States, 1991–1992



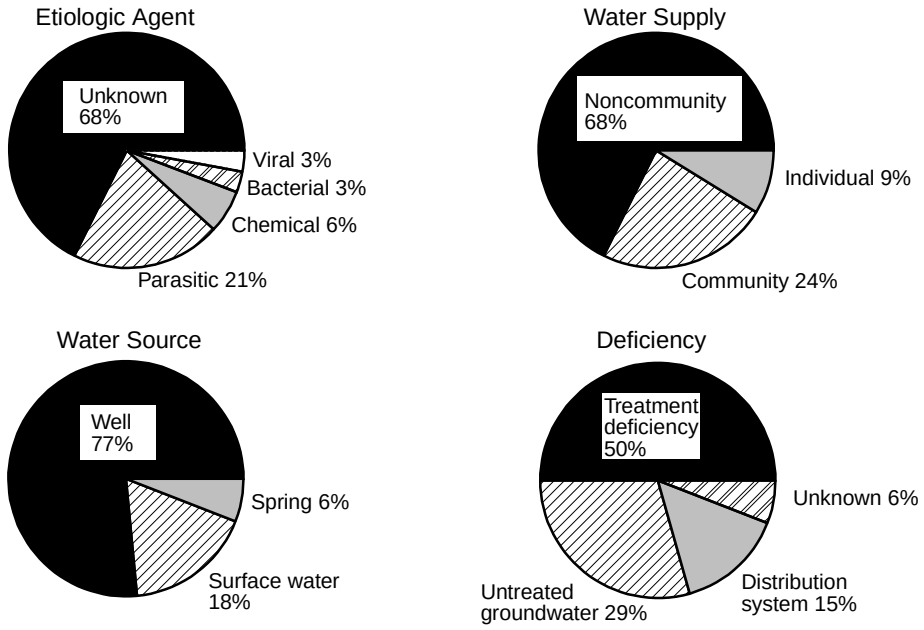
Water Source

In six (18%) of the 34 outbreaks, the water source was a lake or river (surface water). All systems provided chlorination, and four also provided filtration. In the filtered systems, distribution deficiencies were found for one outbreak, no deficiency was identified for one, and the other two were associated with poor filtration of water. During one of these latter outbreaks, the water was also temporarily not chlorinated. One of the unfiltered systems was preparing for an exemption from the filtration required by EPA's Surface Water Treatment Rule, and its raw water quality had been excellent (low turbidity, no coliforms) before the outbreak.

Two outbreaks (6%) were associated with spring water. A giardiasis outbreak occurred when a cross-connection at the water storage tanks allowed contaminated surface water to enter the distribution system. For the other outbreak, which was due to *Cryptosporidium*, evidence (i.e., presence of algae and diatoms) suggested that surface water had entered the spring water.

Outbreaks attributed to water contaminated at the point of use rather than at its source or in its distribution traditionally are not included in the line listings. CDC received five reports of such outbreaks, which caused an estimated 593 persons to become ill, including three who were hospitalized. Four of the five were outbreaks of AGI associated with contamination of a container (three outbreaks) or ice (one

FIGURE 2. Outbreaks associated with water intended for drinking — United States, 1991–1992 (N=34)*



*See Methods section for description of reporting variables.

outbreak). The other was an outbreak caused by Norwalk virus, which apparently contaminated an ice machine aboard an oceangoing passenger ship.

Outbreaks Associated with Recreational Water

For the period 1991–1992, 21 states reported a total of 39 outbreaks associated with water used for recreation (Tables 6 and 7). Thirty-three outbreaks were reported for 1991 and six for 1992. Two of the 21 states submitted 13 of the 39 reports: Washington (seven) and Minnesota (six). Outbreaks were reported for each month except April and November, but most outbreaks occurred in June (nine) or July (eight) (Figure 1).

The outbreaks caused illness in an estimated 1,825 persons. Median outbreak size was seven persons (range, 1–595). Reported illnesses included dermatitis (15 outbreaks), gastroenteritis (11), meningoencephalitis (six), Pontiac fever (four), conjunctivitis with otitis or pharyngitis (two), and leptospirosis (one). Twenty-one persons reportedly were hospitalized. The six deaths associated with recreational water exposure were all due to amebic meningoencephalitis.

Of the 15 outbreaks of dermatitis, which affected an estimated 292 persons, 12 (80%) were outbreaks of rash or folliculitis associated with hot tubs, whirlpools, or swimming pools. In eight of the 12, *Pseudomonas* was confirmed as the etiologic agent, and in the other four, the clinical syndrome was consistent with this etiology. In seven of the investigations, water sampling demonstrated low chlorine concentrations or the presence of *Pseudomonas*, or both. In the three dermatitis outbreaks not associated with *Pseudomonas*, a clinical syndrome consistent with schistosomal dermatitis (swimmer's itch) was noted. Two of these outbreaks were associated with swimming in lakes in Utah and Wyoming. The third was associated with ocean water in Delaware; local snails were found to contain cercariae of *Austrobilharzia variglandis*, an avian schistosome implicated as a cause of cercarial dermatitis (*6*).

The etiologic agent identified in six (55%) of the 11 outbreaks of gastroenteritis (Table 7, Figure 3) was a protozoal parasite, either *Giardia* (four) or *Cryptosporidium* (two). Three *Giardia* outbreaks were related to unintentional ingestion of untreated water from a lake (one) or small wading pools (two). The other three parasitic outbreaks were associated with community pools that were chlorinated and filtered. No treatment deficiencies in the pools were identified. In five of the parasitic outbreaks, no water sampling for protozoa was done. In the other, examination of filter backwash from the pool 3 months after the outbreak did not reveal an etiologic agent.

Two outbreaks of gastroenteritis of unknown etiology were reported. Three other outbreaks of gastroenteritis were attributed to bacterial pathogens; each outbreak was associated with swimming in a lake. *S. sonnei* was implicated for all three outbreaks. In one of the three, *Escherichia coli* O157:H7 was also implicated. This was the first reported outbreak of *E. coli* O157:H7 linked to recreational water. Bacterial subtyping indicated that lake-associated transmission of both pathogens continued for 3 weeks (personal communication, W. Keene). Poor water exchange was a contributing factor in at least two of the three outbreaks, and, for both of these, fecal coliforms in shallow lake water exceeded recommended state levels by several-fold.

Four outbreaks of hot tub- or whirlpool-associated Pontiac fever were reported. In three of the four, serologically confirmed *Legionella* infection was documented (*7*). For the other, although the clinical syndrome was consistent with Pontiac fever, serologic results were negative.

Two outbreaks of conjunctivitis were reported. Adenovirus serotype 3 was implicated (from clinical and water samples) in an outbreak of conjunctivitis, pharyngitis, and fever. Over a 2-month period, an estimated 595 persons became ill after swimming in an inadequately chlorinated pond (*8*). A swimming-pool-associated outbreak of conjunctivitis, otitis, and rash was caused by *Pseudomonas*.

An outbreak of leptospirosis was associated with swimming in a rural pond that was stagnant because of drought (*9*). *Leptospira interrogans* serovar *grippotyphosa* was demonstrated in urine specimens from patients and also from pond water; this was the first reported investigation in which the organism was cultured from both clinical and environmental samples.

The six cases of fatal primary amebic meningoencephalitis occurred during the summer and fall of 1991 (*10,11*). *Naegleria* was demonstrated in brain autopsy specimens from five cases and in cerebrospinal fluid from the sixth. Cases were associated with swimming in a lake (two), pond (one), or stream (one), or with facial immersion in a puddle during a fight (one). Another was related to bathing in a hot spring that had been associated with two previous cases (*10*).

Previously Unreported Outbreaks

Reports of three previously unpublished outbreaks from 1988 through 1989 were received (Table 8). The etiologic agent for all three was *S. sonnei*. In the two associated with water intended for drinking, an estimated 141 persons were reported ill and 13 were hospitalized. Both outbreaks occurred in noncommunity well-water systems. Although each system was equipped for chlorination and one also had filtration and UV capabilities, the equipment was not being used appropriately. In the WBDO associated with recreational water, an estimated 61 persons were ill and six hospitalized after swimming in a lake in which an ill child had defecated.

TABLE 6. Outbreaks of dermatitis associated with recreational water — United States, 1991–1992 (N=15)

State	Year	Month	Class*	Etiologic agent†	No. cases	Source	Setting
DE	1991	Oct	I	*c/w Schistosoma* sp.	30	ocean	beach
MN	1991	Feb		*Pseudomonas*	7	hot tub	private home
MN	1992	Sept		*Pseudomonas*	29	swimming pool	school
UT	1991	Jun	IV	*c/w Schistosoma* sp.	5	lake	swimming area
WA	1991	Jun		*Pseudomonas*	8	hot tub	private home
WA	1991	Dec		*Pseudomonas*	5	hot tub	apartment spa
WA	1991	Oct		*c/w Pseudomonas*	5	hot tub	private home
WA	1991	Sept		*c/w Pseudomonas*	2	hot tub	private home
WA	1991	Jul		*c/w Pseudomonas*	3	hot tub	private home
WA	1991	Feb		*Pseudomonas*	6	hot tub	private home
WI	1991	Jun		*Pseudomonas*	8	hot tub	private home
WI	1991	Mar		*c/w Pseudomonas*	45	whirlpool	motel
WI	1991	Feb		*Pseudomonas*	24	whirlpool	motel
WI	1992	Feb		*Pseudomonas*	10	whirlpool	motel
WY	1991	Jun	IV	*c/w Schistosoma* sp.	5	lake	park

*See Table 1 for class definitions.
†c/w=consistent with.

TABLE 7. Other outbreaks associated with recreational water — United States, 1991–1992 (N=24)

State*	Year	Month	Class†	Illness	Etiologic Agent§	No. cases	Source	Setting
CA	1991	Oct		meningoencephalitis	*Naegleria*¶	1	hot spring	recreation area
FL	1991	Aug		meningoencephalitis	*Naegleria*¶	1	puddle	rural area
GA	1991	Jul	I	gastroenteritis	*Giardia*	9	wading pool	day care center
GA	1991	Jul	II	gastroenteritis	*Giardia*	7	wading pool	day care center
GU	1991	Sept		meningoencephalitis	*Naegleria*¶	1	stream	rural area
ID	1992	Aug	II	gastroenteritis	*Cryptosporidium*	26	water slide	park
IL	1991	Jul	I	leptospirosis	*Leptospira*	6	pond	rural area
MD	1991	Jun	II	gastroenteritis	*Giardia*	14	swimming pool	park
MD	1992	Jul	III	gastroenteritis	AGI	15	creek	private home
MN	1992	Jan	I	otitis, conjunctivitis, and rash	*Pseudomonas*	35	swimming pool	hotel
MO	1991	Aug	II	gastroenteritis	AGI	61	dunking booth	fair
MT	1991	Mar	II	Pontiac fever	*Legionella*	4	hot tub	ski resort
NC	1991	Jun	I	pharyngitis	adenovirus 3	595	pond	camp
NC	1991	Sept		meningoencephalitis	*Naegleria*¶	1	pond	swimming area
NC	1991	Sept		meningoencephalitis	*Naegleria*¶	1	lake	swimming area
OR	1991	Jul	I	gastroenteritis	*E. coli* O157:H7**	80	lake	park
OR	1992	Jun	II	gastroenteritis	*Cryptosporidium*	500	wave pool	park
PA	1991	Jun	I	gastroenteritis	*Shigella sonnei*	203	lake	park
RI	1991	Jul	IV	gastroenteritis	*Shigella sonnei*	23	lake	swimming area
TX	1991	Jul		meningoencephalitis	*Naegleria*¶	1	lake	swimming area
VT	1991	Jan	II	Pontiac fever	*Legionella*	6	hot tub	ski resort
WA	1991	Jul	IV	gastroenteritis	*Giardia*	4	lake	campground
WI	1991	May	I	Pontiac fever	*Legionella*	6	whirlpool	motel
WI	1991	Mar	II	Pontiac fever	unknown	33	whirlpool	motel

*Includes territories.
†See Table 1 for class definitions.
§AGI=acute gastrointestinal illness of unknown etiology.
¶Resulted in one death.
**Mixed outbreak of *Escherichia coli* O157:H7 and *Shigella sonnei*.

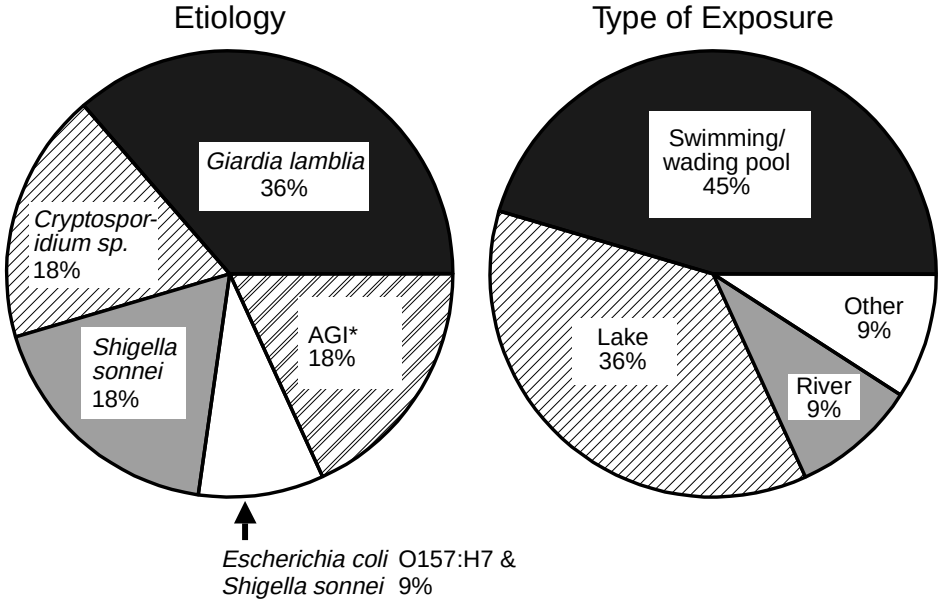
DISCUSSION

General Interpretation of Surveillance Data for Waterborne Disease Outbreaks

The data in this surveillance summary should be interpreted with care. They probably do not reflect the true incidence of WBDOs or the relative incidence of outbreaks caused by various etiologies. Only a fraction of WBDOs may be recognized, investigated, and/or reported to CDC or EPA, and the extent of underrecognition and underreporting is unknown.

The likelihood that individual cases of illness will be epidemiologically linked and associated with water varies considerably among locales and is dependent on factors such as consumer awareness, physician interest, and surveillance activities of state and local health and environmental agencies. Therefore, the states with the most outbreak reports are not necessarily the ones with the most outbreaks. Recognition of WBDOs is dependent on certain outbreak characteristics; outbreaks involving serious illness are most likely to come to the attention of health authorities. In cities, large outbreaks are more likely to be recognized than sporadic cases or small outbreaks in which ill persons may consult different physicians. Outbreaks occurring in community water systems are more likely to be recognized than those in noncommunity systems because the latter serve nonresidential areas and transient populations. Outbreaks in individual systems are the most likely to be underreported because they generally involve small numbers of persons. Outbreaks of acute disease are more readily

FIGURE 3. Outbreaks of gastroenteritis associated with recreational water use — United States, 1991–1992 (N=11)



*AGI=acute gastrointestinal illness of unknown etiology.

identified than those associated with disease from chronic, low-level exposure to a pathogen or chemical.

Identification of the etiologic agent of a WBDO is dependent on timely outbreak recognition so that appropriate clinical and environmental samples can be obtained. The interests and expertise of the investigators and the routine practices of local laboratories also influence whether the causative agent is identified. For example, diarrheal stool specimens generally are examined for bacterial pathogens but not viruses. In most laboratories, routine stool examination for ova and parasites does not include the special procedures needed to identify *Cryptosporidium*. Water quality data are also highly variable and depend on factors such as the health department's fiscal, investigative, and laboratory resources. Furthermore, a few large outbreaks may substantially alter the relative proportion of cases of waterborne disease attributed to a particular agent. The number of reported cases is generally an approximate figure, and the method and accuracy of the approximation vary among outbreaks.

1991–1992 Outbreaks Associated with Water Intended for Drinking

The total numbers of outbreaks reported for 1991 and 1992 are comparable with those reported for recent years: 15 and 19, respectively. With the addition of previously unreported outbreaks, 16 WBDOs have been reported for 1988, 13 for 1989, and 14 for 1990 (*2,3*). WBDO reports peaked during 1979–1983 (Figures 4 and 5); the increase and the subsequent decrease in reports may reflect, at least in part, changes in surveillance activity rather than deterioration or improvement in water systems (*12*).

During the years 1971–1990, comparable proportions of WBDOs were associated with noncommunity (45%) and community systems (43%) (*2,3,13*). However, during 1991–1992, substantially more noncommunity than community outbreaks (68% versus 24%) were reported. Although these data may not indicate a new trend, they may reflect increased public usage of noncommunity systems in recreational areas; June and July were the months with the most noncommunity outbreaks. Outbreaks in noncommunity systems were more likely than those in community systems to be associated with untreated water (44% versus 13%).

Protozoal parasites were the most frequently identified etiologic agents. From 1978 through 1991, *Giardia* was the most commonly implicated pathogen (*2*). However, in 1992, the same numbers of outbreaks of giardiasis and cryptosporidiosis were

TABLE 8. Waterborne outbreaks not included in previous summaries — United States, 1988–1989 (N=3)*

A. Outbreaks associated with water intended for drinking.

State	Year	Month	Class†	Etiologic agent	No. cases	Type of system§	Deficiency	Source	Setting
IL	1988	Jul	I	*Shigella sonnei*	11	NC	5	well	restaurant
PA	1989	Apr	I	*Shigella sonnei*	130	NC	3	well	restaurant

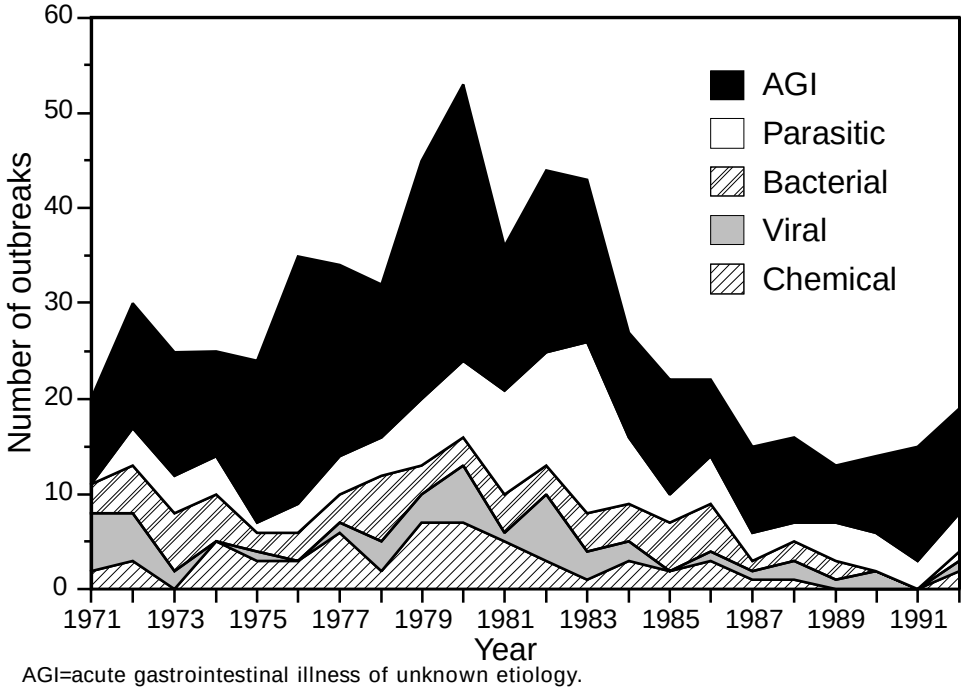
B. Outbreaks associated with recreational water.

State	Year	Month	Class†	Illness	Etiologic agent	No. cases	Source	Setting
IL	1988	Jun	I	gastroenteritis	*Shigella sonnei*	61	lake	beach club

*See Methods section for description of reporting variables.
†See Table 1 for class definitions.
§NC=noncommunity.

reported. The increased identification of cryptosporidiosis may be due to heightened awareness that the organism may cause WBDOs (*14–16*). However, outbreaks caused by *Cryptosporidium* are probably still underrecognized. An important factor in the recognition of the outbreaks in 1992 was routine screening of stool specimens for *Cryptosporidium* by certain local laboratories, which is not standard practice in most areas. The continued importance of waterborne cryptosporidiosis was recently underscored by an outbreak in Milwaukee (in March and April of 1993) that was the largest WBDO ever reported in the United States; an estimated 403,000 persons had watery diarrhea (personal communication, JP Davis). *Cryptosporidium* oocysts are widespread in U.S. raw water sources in both pristine and polluted areas (*17*). Water analysis at 66 U.S. and Canadian surface water treatment plants has revealed low levels of *Cryptosporidium* oocysts in up to 27% of drinking water samples (*18*), but the methods used do not assess viability or potential infectivity of the cysts. The risk posed by these low levels of oocysts for immunocompetent and immunocompromised persons is unknown. In the Oregon outbreak in February 1992, oocyst levels in environmental samples were low, and the attack rate (<5%) was also lower than the attack rates reported for *Cryptosporidium* outbreaks in which oocyst levels in water samples were more than 10-fold higher (*14*). More information is needed about the infective dose, the differences in virulence among strains, and whether acquired immunity is protective and long lasting.

FIGURE 4. Waterborne outbreaks, by year and etiologic agent — United States, 1971–1992 (N=609)

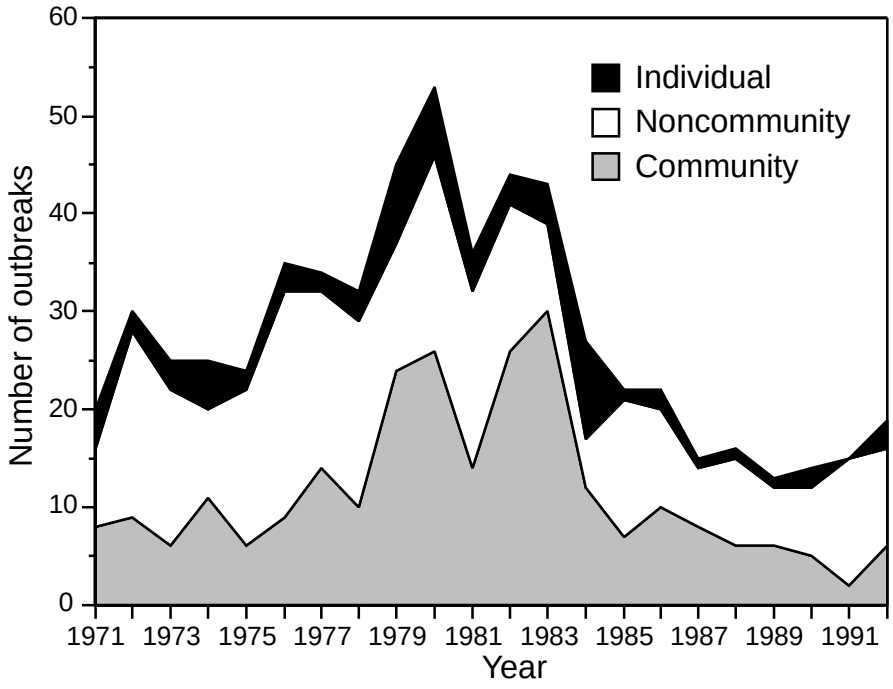


AGI=acute gastrointestinal illness of unknown etiology.

Two outbreaks caused by acute chemical intoxication were reported. Methemoglobinemia has been previously documented after ingestion of water contaminated with nitrates, often from agricultural fertilizers (*19*). The EPA has established an MCL of 10 mg/L for nitrate-nitrogen, but the regulation applies only to public water systems. In this report, the reverse-osmosis filter reduced nitrate levels only to the MCL (*5*). Elevated copper levels in the water may have contributed to the development of methemoglobinemia by inducing emesis in exposed ill persons, resulting in elevation of gastric pH with subsequent growth of nitrate-reducing bacteria and increased nitrate-to-nitrite conversion in the stomach. The 1992 outbreak of acute fluoride intoxication is the largest reported to date, and clinical follow-up of ill persons showed that metabolic abnormalities and elevated urine and/or serum fluoride levels can persist for a week or more.

As in previous years (Figure 4), the majority of outbreaks (68%) during 1991–1992 were classified as AGI of unknown etiology. Although some outbreaks were rigorously investigated, for many of these, the search for a causative agent was limited or clinical specimens could not be or were not obtained in a timely manner. The likelihood of identifying an etiologic agent was equally low (approximately one-third) for surface water and groundwater sources. However, the agent was much more frequently identified for WBDOs in community systems (63%) than in noncommunity systems (17%),

FIGURE 5. Waterborne outbreaks, by year and type of water system — United States, 1971–1992 (N=609)*



*See Methods section for description of types of water systems.

emphasizing the difficulty of investigating outbreaks affecting the transient populations that use noncommunity water. Although the clinical features of illness in more than half of the AGI outbreaks suggest a viral etiology, clinical diagnosis is not specific; the group of outbreaks of AGI probably includes viral, bacterial, and parasitic etiologies. Availability of rapid diagnostic tests for viruses and newly emerging pathogens can aid in identifying the causative agents of these outbreaks. Information about the pathogens responsible for WBDOs is important for evaluating the adequacy of current water treatment processes and regulations. Nevertheless, the water quality data from AGI outbreaks suggest that available water disinfection technology is not always in place or used reliably; for 91% of these outbreaks, water sampling showed the presence of coliforms and/or deficiencies in chlorination.

During 1991–1992, 24 outbreaks (71%) were associated with contaminated untreated or inadequately treated groundwater. Adequate, continuous disinfection of groundwater should reduce the occurrence of WBDOs, particularly in small systems in which intermittent contamination of wells and springs is difficult to detect or prevent. In addition, wells and springs must be protected from sources of contamination such as surface runoff, septic tank drainage, and sewage discharges.

Two outbreaks were associated with treatment deficiencies in water systems using UV light for disinfection. UV light can be an effective disinfectant if properly applied, operated, and maintained. It may be effective for disinfecting bacteria and viruses but not protozoa (*20,21*). Reduced efficacy of UV systems for colored or turbid water has been noted (*22*), and UV light is not approved by EPA for use in surface water systems or in groundwater systems under the influence of surface water. For many of the outbreaks associated with inadequately treated water, chlorination was not maintained consistently. Adequate, consistent levels of chlorine are particularly important for disinfection of relatively chlorine-resistant organisms such as *Giardia* (*23*). Unfortunately, *Cryptosporidium* is highly resistant to disinfection by chlorine. Ozone is more effective than chlorine against *Cryptosporidium* (*24*), but no residual disinfectant is provided with this treatment. Ozone may not be suitable for small systems because of expense and the technical expertise required, and it is used as a disinfectant by <1% of the drinking water systems that serve populations of more than 10,000. Therefore, efforts to reduce the risk of waterborne giardiasis and cryptosporidiosis should focus on source protection and removal of cysts and oocysts by filtration.

The SWTR requires filtration of all but exceptionally well-protected surface water sources, including groundwater influenced by surface water. The SWTR was promulgated to reduce the risk of disease caused by waterborne protozoal parasites. Three protozoal outbreaks during 1991–1992 occurred in systems that were equipped with chlorine disinfection and met EPA coliform standards but were not equipped with filtration. EPA is considering an Information Collection Rule (ICR) that would require larger water utilities to monitor for *Cryptosporidium*, *Giardia*, and perhaps other pathogens in source water and, under certain conditions, in treated water for 18 months. Under the ICR, careful laboratory analysis and quality control would be needed, and the currently available laboratory methods do not assess infectivity of cysts or oocysts. Epidemiologic studies in conjunction with information on the occurrence of cysts and oocysts in source water will be helpful in assessing waterborne disease risks, determining the need for an enhanced Surface Water Treatment Rule, and determining whether specific monitoring for protozoal pathogens is necessary to supplement the total coliform MCL. The water quality data collected for WBDOs in the

years 1991–1992 indicated that coliforms were detected for 88% of the outbreaks with bacterial, viral, or unknown etiologies but only 33% of the protozoal outbreaks. These data suggest that the use of coliforms as indicators of water contamination is generally sound but may not be adequate to detect contamination by protozoa.

Four of the six surface water systems associated with WBDOs were equipped with filtration. In three of these outbreaks, raw water quality had deteriorated because of sewage effluents that were not appropriately diluted as a result of low stream flows during dry weather. During the outbreaks associated with these systems, filtration deficiencies were noted, with elevated turbidity in finished water. Decreased filtration efficiency combined with deterioration in raw water quality also contributed to the WBDO in Milwaukee (1993). Although turbidity measurements indicated inefficient operation of the filtration process for the water systems associated with the 1993 Milwaukee outbreak and one Oregon outbreak (May 1992), none of the then-existing EPA water quality regulations were violated. Outbreaks associated with filtered systems illustrate the importance of improved operation and monitoring of the filtration process and the necessity for multiple barriers; in addition to disinfection and filtration, protection of raw water quality is essential for preventing transmission of waterborne diseases.

1991–1992 Outbreaks Associated with Recreational Water Use

In the period 1991–1992, the most frequently reported WBDOs due to recreational water were outbreaks of dermatitis associated with hot tubs, whirlpools, and swimming pools. Although factors such as host susceptibility, immersion time, and number of bathers can influence acquisition of infection (*25*), in general, most outbreaks are directly related to inadequate operation and maintenance procedures. Outbreaks are preventable if water 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 (*26*). Pontiac fever due to aerosolized *Legionella pneumophila* is also associated with use of hot tubs and whirlpools (*27,28*).

The six deaths associated with recreational water were caused by primary amebic meningoencephalitis (PAM). The presence in fresh water of the etiologic agent, *Naegleria fowleri*, is related to water temperature. Cases of PAM in the United States are rare but generally have been acquired during summer months when exposure to warm water is highest (*29*). Although behavioral risk factors for PAM are unknown, three cases in 1991 occurred in persons who may have had an increased risk of inhaling water; two were in young children learning to swim and the third in a young man whose face was immersed during a fight.

Swimming and other recreational activities in which unintentional ingestion of water can occur are known to increase the risk of gastrointestinal illness, even in non-outbreak settings (*30,31*). The number of outbreaks of gastroenteritis due to inadvertent ingestion of water during swimming (Figure 3) was similar to the number previously reported (11 in 1991–1992 compared with 13 in 1989–1990). However, in contrast to past years, approximately half (55%) of the gastroenteritis outbreaks were attributed to protozoal parasites: *Giardia* (four) and *Cryptosporidium* (two). Cryptosporidiosis associated with recreational water has been previously reported (*32*) but probably is underrecognized. The two cryptosporidiosis outbreaks were identified because drinking water outbreaks of *Cryptosporidium* in the region earlier in the year had led to increased awareness of the need to consider this diagnosis. Three of the

outbreaks of protozoal gastroenteritis occurred in pools that were chlorinated and filtered; two were in settings (wave pool or water slide) with an increased risk of unintentional ingestion of water. Typical pool chlorination will disinfect *Giardia* cysts but may require more than 15 minutes, depending on temperature, pH, and chlorine concentration. Even though *Cryptosporidium* oocysts are resistant to disinfection by chlorine, they can be removed by most pool filtration systems. However, rates of filtration are generally slow, requiring up to 6 hours for a complete turnover of pool water. Therefore, water treatment does not ensure protection against protozoal infection in these settings.

Outbreaks of swimming-associated shigellosis, which have been documented previously (*2,33,34*), continue to be reported. The probable source of the pathogen in the three 1991–1992 outbreaks, as in past outbreaks, was fecal contamination of lake water by other swimmers. A contributing factor in at least two of the outbreaks may have been poor water exchange in the swimming area. In one outbreak, a history of having swallowed lake water was a risk factor for illness. Because the infectious dose of *Shigella* is low, infection may be acquired without swallowing large quantities of water (*35*). An outbreak of *E. coli* O157:H7 infection occurred in conjunction with one of the *Shigella* outbreaks. This is the first reported *E. coli* O157:H7 outbreak associated with recreational water exposure. This outbreak was recognized rapidly because *E. coli* O157:H7 infection had been made reportable by the state during the previous year. *E. coli* O157:H7, like *Shigella*, appears to have a low infectious dose (*36*). Furthermore, it can survive in water under certain conditions for long periods; in one study, only a 2-log reduction in bacterial counts was found to have occurred after 5 weeks at 5 C (*37*). The long period of transmission in this outbreak may have been due to reintroduction of the particular *E. coli* O157:H7 subtype into the swimming area, but it is more likely that it persisted in lake water for the duration of the outbreak.

The EPA has published criteria for evaluating the quality of fresh and marine recreational waters (*38,39*). Microbial monitoring has been recommended for recreational areas potentially contaminated by sewage. However, the value of routine monitoring of untreated water for fecal contamination due to bathers has not been established. Prevention efforts have focused on providing adequate toilet facilities at recreational areas and limiting the density of bathers.

CONCLUSION

Information from national WBDO surveillance is used to characterize the epidemiology of waterborne diseases in the United States. Data regarding the types of water systems and deficiencies associated with outbreaks are necessary to evaluate the adequacy of treatment regulations and current water quality monitoring. Identification of the etiologic agents of outbreaks is particularly critical because agents newly associated with waterborne outbreaks may require new methods of control. In recent years, *Cryptosporidium* and *E. coli* O157:H7 have been identified as important waterborne pathogens. Rapid recognition and control of several outbreaks in 1991–1992 were aided by surveillance for these agents at the local and state level. Maintaining the capabilities of local and state health departments to investigate outbreaks and to conduct surveillance is a key factor in waterborne disease control and prevention. In addition to outbreak investigation, epidemiologic studies are needed to evaluate the risk of waterborne disease; evidence suggests that a substantial proportion of

non-outbreak-related diarrheal illness may be associated with consumption of water that meets all current water quality standards (*40*).

References

1. Craun GF, ed. Waterborne diseases in the United States. Boca Raton, FL: CRC Press, 1986.
2. CDC. Waterborne disease outbreaks, 1989–1990. MMWR 1991;40(SS–3):1–21.
3. CDC. Waterborne disease outbreaks, 1986–1988. MMWR 1990;39(SS–2):1–13.
4. Leland D, McAnulty J, Keene W, Stevens G. A cryptosporidiosis outbreak in a filtered-water supply. J Am Water Works Assn 1993;85:34–42.
5. CDC. Methemoglobinemia in an infant—Wisconsin, 1992. MMWR 1993;42:217–9.
6. CDC. Cercarial dermatitis outbreak at a state park—Delaware, 1991. MMWR 1992;41:225–8.
7. Thomas DL, Mundy LM, Tucker PC. An outbreak of hot-tub legionellosis. Abstracts of the 1991 ICAAC. Abstr. no. 310. Chicago, IL: Sept-Oct 1991.
8. Outbreak of pharyngoconjunctival fever at a summer camp—North Carolina, 1991 (news). Infect Control Hosp Epidemiol 1992;13:499–500.
9. Jackson LA, Kaufmann AF, Adams WG, et al. Outbreak of leptospirosis associated with swimming. Pediatr Infect Dis J 1993;12:48–54.
10. California Department of Health Services. Primary amebic meningoencephalitis associated with a natural hot springs in San Bernadino County. California Morbidity 1992;13/14.
11. CDC. Primary amebic meningoencephalitis—North Carolina, 1991. MMWR 1992;41:437–40.
12. Craun GF, McGoldrick JL. Workshop on methods for investigation of waterborne disease outbreaks. Research Triangle Park, NC: US Environmental Protection Agency, 1990. EPA publication no. 600/9-90/021.
13. Craun GF. Waterborne disease outbreaks in the United States of America: causes and prevention. World Health Stat Q 1992;45:192–9.
14. Hayes EB, Matte TD, O'Brien TR, et al. Large community outbreak of cryptosporidiosis due to contamination of a filtered public water supply. N Engl J Med 1989;320:1372–6.
15. D'Antonio RG, Winn RE, Taylor JP, et al. A waterborne outbreak of cryptosporidiosis in normal hosts. Ann Intern Med 1985;103:886–8.
16. Richardson AJ, Frankenberg RA, Buck AC, et al. An outbreak of waterborne cryptosporidiosis in Swindon and Oxfordshire. Epidemiol Infect 1991;107:485–95.
17. Rose JB. Occurrence and significance of *Cryptosporidium* in water. J Am Water Works Assn 1988;80:53–8.
18. LeChevallier MW, Norton WD, Lee RG. *Giardia* and *Cryptosporidium* spp. in filtered drinking water supplies. Appl Environ Microbiol 1991;57:2617–21.
19. Johnson CJ, Bonrud PA, Dosch TL, et al. Fatal outcome of methemoglobinemia in an infant. JAMA 1987;257:2796–7.
20. Sommer R, Weber G, Cabaj A, Wekerle J, Keck G, Schauberger G. UV-inactivation of microorganisms in water. Zbl Hyg 1989;189:214–24.
21. Lorenzo-Lorenzo MJ, Ares-Mazas ME, Villacorta-Martinez de Maturana I, Duran-Oreiro D. Effect of ultraviolet disinfection of drinking water on the viability of *Cryptosporidium parvum* oocysts. J Parasitol 1993;79:67–70.
22. Carlson DA, Seabloom RW, DeWalle FB, et al. Ultraviolet disinfection of water for small water supplies. Cincinnati, OH: US Environmental Protection Agency, 1985; EPA publication no. 600/S2-85/092.
23. Hoff JC. Inactivation of microbiological agents by chemical disinfectants. US Environmental Protection Agency, 1986; EPA publication no. 600/2-86/067.
24. Korich DG, Mead JR, Madore MS, Sinclair NA, Sterling CR. Effects of ozone, chlorine dioxide, chlorine, and monochloramine on *Cryptosporidium parvum* oocyst viability. Appl Environ Microbiol 1990;56:1423–8.
25. Highsmith AK, McNamara AM. Microbiology of recreational and therapeutic whirlpools. Toxicity Assessment 1988;3:599–611.
26. CDC. Suggested health and safety guidelines for public spas and hot tubs. Atlanta: US Department of Health and Human Services, Public Health Service, 1981; DHHS publication no. 99-960.
27. Spitalny KC, Vogt RL, Orciari LA, Witherell LE, Etkind P, Novick LF. Pontiac fever associated with a whirlpool spa. Am J Epidemiol 1984;120:809–17.

28. Mangione EJ, Remis RS, Tait KA, et al. An outbreak of Pontiac fever related to whirlpool use, Michigan 1982. JAMA 1985;253:535–9.
29. Wellings FM, Amuso PT, Chang SL, Lewis AL. Isolation and identification of pathogenic *Naegleria* from Florida lakes. Appl Environ Microbiol 1977;34:661–7.
30. Calderon RL, Mood EW, Dufour AP. Health effects of swimmers and nonpoint sources of contaminated water. Int J Environ Health Res 1991;1:21–31.
31. Seyfried PL, Tobin RS, Brown NE, Ness PF. A prospective study of swimming-related illness: I. Swimming-associated health risk. Am J Public Health 1985;75:1068–70.
32. Sorvillo FJ, Fujioka K, Nahlen B, Tormey MP, Kebabjian R, Mascola L. Swimming-associated cryptosporidiosis. Am J Public Health 1992;82:742–4.
33. Sorvillo FJ, Waterman SH, Vogt JK, England B. Shigellosis associated with recreational water contact in Los Angeles County. Am J Trop Med Hyg 1988;38:613–7.
34. Makintubee S, Mallonee J, Istre GR. Shigellosis outbreak associated with swimming. Am J Public Health 1987;77:166–8.
35. DuPont H, Levine M, Hornick R, Formal S. Inoculum size in shigellosis and implications for expected mode of transmission. J Infect Dis 1989;159:1126–8.
36. Griffin PM, Tauxe RV. The epidemiology of infections caused by *Escherichia coli* O157:H7, other enterohemorrhagic *E. coli*, and the associated hemolytic uremic syndrome. Epidemiol Rev 1991;13:60–98.
37. Geldreich EE, Fox KR, Goodrich JA, Rice EW, Clark RM, Swerdlow DL. Searching for a water supply connection in the Cabool, Missouri, disease outbreak of *Escherichia coli* O157:H7. Water Research 1992;26:1127–37.
38. Dufour AP. Health effects criteria for fresh recreational waters. Research Triangle Park, North Carolina: US Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory, 1984; EPA publication no. 600/1-84-004.
39. Cabelli VJ. Health effects criteria for marine recreational waters. Research Triangle Park, North Carolina: US Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory, 1983; EPA publication no. 600/1-80-031.
40. Payment P, Richardson L, Siemiatycki J, Dewar R, Edwardes M, Franco E. A randomized trial to evaluate the risk of gastrointestinal disease due to consumption of drinking water meeting current microbiological standards. Am J Public Health 1991;81:703–8.

Silicosis Surveillance— Michigan, New Jersey, Ohio, and Wisconsin, 1987–1990

Mary J. Reilly, M.S.[1]
Kenneth D. Rosenman, M.D.[1]
Flint C. Watt, P.E.[2]
Martha J. Stanbury, M.S.P.H.[3]
David J. Valiante, M.S.[3]
Lesliann E. Helmus, M.S.[4]
Adeline A. Migliozzi, R.N., M.S.N., C.O.H.N.[4]
Henry A. Anderson, M.D.[5]
Lawrence Hanrahan, Ph.D.[5]
Ruth A. Jajosky, D.M.D., M.P.H.[6]
Karl J. Musgrave, D.V.M., M.P.H.[6]
Robert M. Castellan, M.D., M.P.H.[6]
Diana L. Ordin, M.D., M.P.H.[7]

[1]*Michigan State University, Lansing, MI.*
[2]*Michigan Department of Public Health, Lansing, MI.*
[3]*New Jersey Department of Health, Trenton, NJ.*
[4]*Ohio Department of Health and Social Services, Columbus, OH.*
[5]*Wisconsin Department of Health, Madison, WI.*
[6]*Division of Respiratory Disease Studies*
National Institute for Occupational Safety and Health (NIOSH), CDC
[7]*Division of Surveillance, Hazard Evaluations, and Field Studies, NIOSH, CDC*

Summary

Problem/Condition: Improved surveillance for silicosis is needed to target interventions to prevent this occupational lung disease caused by the inhalation of crystalline silica dust.

Reporting Period Covered: 1987–1990.

Description of Systems: State-based silicosis surveillance and intervention programs have been developed in Michigan, New Jersey, Ohio, and Wisconsin as part of the Sentinel Event Notification System for Occupational Risks (SENSOR) Program, initiated in 1987 by the National Institute for Occupational Safety and Health (NIOSH).

Results: From 1987 through 1990, the SENSOR program confirmed a total of 430 cases of silicosis reported from these four states. Overall, approximately 60% of these cases were in workers employed in primary metal industries, although the types of industries in which cases occurred varied by state. Some cases were attributable to relatively recent exposure, including new cases in seven persons first exposed since 1980 in New Jersey. Silicosis case reports have prompted measurement of respirable silica concentrations at 25 Michigan work sites, and 14 (56%) of these sites were found to have levels that exceeded the legally permissible exposure level.

Interpretation: The silicosis surveillance and intervention strategies piloted by state health departments in the NIOSH-funded SENSOR Program have demonstrated the feasibility and effectiveness of identifying specific silica-using work sites that need preventive intervention.
Actions Taken: On the basis of initial experience in these four states, NIOSH developed guidelines for state-based silicosis surveillance and awarded SENSOR cooperative agreements to three additional states where the applicability of these surveillance methods will be further evaluated.

INTRODUCTION

In 1987, 10 states were awarded 5-year cooperative agreements by CDC's National Institute for Occupational Safety and Health (NIOSH) to develop and implement surveillance systems for selected occupational conditions under the Sentinel Event Notification System for Occupational Risks (SENSOR) Program (*1*). The purpose of the SENSOR Program is to develop and implement case-based surveillance and preventive follow-up activities for selected occupational diseases and injuries (*1*). One of the diseases selected for SENSOR activities is silicosis, a fibrotic lung disease caused by inhalation of respirable crystalline silica dust. Although some cases occur after only a few years, most cases of silicosis occur after ≥20 years of occupational exposure. This report summarizes data from silicosis surveillance in the Michigan, New Jersey, Ohio, and Wisconsin SENSOR programs, solicited by state health departments during the period 1987–1990.

METHODS

Surveillance

Case Report Ascertainment

All four states have relied, at least in part, on reporting of silicosis by physicians for case ascertainment. In Ohio, physicians have been required by law to report this condition since 1953, but not until 1989 were occupational disease reports actively solicited. In Michigan, known or suspected work-related illnesses have been reportable by health professionals since 1978, but active solicitation of reports did not begin until 1988. In New Jersey, although physician reporting had been encouraged since 1983, mandated reporting by physicians did not begin until 1990. In Wisconsin, occupational disease reporting continues to be voluntary.

Other means of case ascertainment have varied by state. New Jersey and Michigan have complemented physician reports with cases identified by review of death certificates and hospital discharge data, as well as with selected follow-up investigations of workplaces where workers with index cases had been exposed to silica. Hospital discharge data account for three-fourths of all reports in each of these two states. In addition, Michigan has reviewed records of workers' compensation awards available from the Michigan Department of Labor. Ohio relies entirely on physician reports, but has actively solicited case reports from individual physicians of decedents identified initially through review of death certificate data. Wisconsin has complemented

physician reporting with ascertainment of cases through review of workers' compensation records, a procedure that accounts for half the confirmed cases in that state.

Case Confirmation

All four states collect demographic, work history, and medical information about each reported silicosis case from a combination of the initial case ascertainment source, review of medical records, and interview of cases and/or family members. Case confirmation requires a) a history of occupational exposure to silica and a chest radiograph classified by a "B" reader* as category 1/0 or greater profusion of small rounded opacities or b) a lung tissue biopsy indicating silicosis (*2*).

Preventive Intervention

Follow-up and prevention efforts, which vary among the participating states, include some or all of the following activities: a) checking with employer or examining other databases to determine if the workplace remains in operation; b) educational outreach regarding the health hazards of silica exposure to workers with silicosis, employees, employers, and physicians; c) workplace evaluations, including review of workplace industrial hygiene measurements and employee records, and silica air monitoring by SENSOR or Occupational Safety and Health Administration (OSHA) industrial hygienists; and d) referral to appropriate regulatory agencies if excessive exposures or hazardous work situations are found.

RESULTS

Epidemiology

From 1987 through 1990, the SENSOR program has confirmed a total of 430 cases of silicosis (Tables 1 and 2). By state, the average year of first occupational exposure to silica dust for workers with cases ranged from 1943 in Michigan to 1961 in Wisconsin. However, in all three states collecting information on year of first exposure (Michigan, New Jersey, and Wisconsin) there were workers with cases whose first exposure was after 1969 (n=23). This total includes seven new cases in New Jersey first exposed after 1980. The average duration of exposure for all confirmed cases was 26 years; 39 (10%) of confirmed cases had <10 years of occupational silica exposure, and 106 (27%) had <20 years.

Overall, approximately 60% of the persons affected by silicosis worked in primary metal industries (Table 2). This industry, which includes foundries, accounted for at least 70% of the confirmed cases in Michigan and Wisconsin. Stone, clay, glass, and concrete products (including ceramics) industries were the predominant source of exposure among the New Jersey cases. In Ohio, no single industry was predominant.

Workplace Follow-up

Detailed information on follow-up efforts in Michigan are illustrative of the potential effectiveness of state-based preventive interventions. The 249 cases of silicosis in

*"B" readers are physicians certified by NIOSH as proficient in classifying chest radiographs for pneumoconioses using the International Labour Office Classification for Radiographs of Pneumoconioses (*3*).

TABLE 1. Case-ascertainment sources for confirmed silicosis cases — Michigan, New Jersey, Ohio, and Wisconsin, Sentinel Events Notification System for Occupational Risks (SENSOR) programs, 1987–1990

	Michigan		New Jersey		Ohio*		Wisconsin		Total	
Source	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
Hospital discharge records	190	(76.3)	66	(74.2)	—†		—		**256**	**(59.5)**
Death certificates	25	(10.0)	1	(1.1)	—		—		**26**	**(6.0)**
Worker's compensation claims	17	(6.8)	—		—		25	(50.0)	**42**	**(9.8)**
Physician	14	(5.6)	3§	(3.4)	42	(100.0)	23	(46.0)	**82**	**(19.1)**
Other	3	(1.2)	19	(21.3)	—		2	(4.0)	**24**	**(5.6)**
Totals	**249**	**(100.0)**	**89**	**(100.0)**	**42**	**(100.0)**	**50**	**(100.0)**	**430**	**(100.0)**

*All cases in Ohio are reported by physicians, but some reports are actively solicited by the health department on the basis of death certificate review.
†State does not use this data source for surveillance of silicosis cases.
§One of these three cases was also ascertained from hospital discharge data.

TABLE 2. Industry reported as source of silica exposure for silicosis cases — Michigan, New Jersey, Ohio, and Wisconsin, Sentinel Events Notification System for Occupational Risks (SENSOR) programs, 1987–1990

	Michigan		New Jersey		Ohio		Wisconsin		Total	
Industry (SIC* code)	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
Manufacturing										
Primary metal industries (33)	195	(78.3)	15	(16.9)	8	(19.0)	35	(70.0)	253	(58.8)
Stone, clay, glass, and concrete products (32)	16	(6.4)	44	(49.4)	9	(21.0)	1	(2.0)	70	(16.3)
Miscellaneous (22,26,27,28,30,34,35,36,37,38)	17	(6.8)	12	(13.5)	10	(24.0)	6	(12.0)	45	(10.5)
Mining (10–14)	10	(4.0)	9	(10.1)	1	(2.4)	2	(4.0)	22	(5.1)
Construction (15–17)	4	(1.6)	7	(7.9)	0	—	0	—	11	(2.6)
Transportation and communication (42,46,47,48,49)	3	(1.2)	1	(1.1)	0	—	0	—	4	(0.9)
Services (73,76,77,80)	1	(0.4)	0	—	0	—	1	(2.0)	2	(0.5)
Trade (50,59)	0	—	1	(1.1)	0	—	0	—	1	(0.2)
Undetermined	3	(1.2)	0	—	14	(33.3)	5	(10.0)	22	(5.1)
Totals	**249**	**(100.0)**	**89**	**(100.0)**	**42**	**(100.0)**	**50**	**(100.0)**	**430**	**(100.0)**

*Standard industrial classification.

Michigan were associated with 144 silica-using workplaces. Sixty-one (42.4%) of these workplaces were no longer operating at the time of follow-up, 19 (13.2%) were located out of state, 11 (7.6%) no longer used silica, two (1.4%) involved multiple locations in the building trade, and four (2.8%) were unknown. The remaining 47 workplaces were targeted for follow-up inspection. Airborne silica concentrations measured at the first 25 worksites inspected exceeded the legally permissible exposure level at 14 (56%) and the NIOSH-recommended exposure limit at 17 (68%).

DISCUSSION

Silicosis is a chronic, nonmalignant lung disease caused by the inhalation of respirable crystalline silica dust. Despite longstanding knowledge about its cause, this preventable occupational lung disease continues to account for more than 300 deaths each year in the United States (*4*). Surveillance of silicosis has revealed neither the true burden nor the continuing risk of the disease (*5,6*), and prevention of silicosis will require improved surveillance in all 50 states (*7*). The findings in this report indicate that the silicosis surveillance strategies piloted by state health departments in the NIOSH-funded SENSOR Program are both feasible and useful. In these four states, the SENSOR Program has identified both large numbers of silicosis cases and high-risk workplaces and industries for targeting interventions and, by identifying cases resulting from relatively recent exposures, has clearly documented that silicosis is an ongoing problem.

SENSOR silicosis surveillance has identified multiple complementary sources for case ascertainment. Michigan and New Jersey have demonstrated that hospital discharge data, while underestimating the total number of patients with silicosis (*8*), can readily identify a considerable number of silicosis cases (*9,10*). Michigan, New Jersey, and Ohio have demonstrated that review of death certificate data can identify additional cases of silicosis.

Mandated case reporting by physicians, although incomplete, appears to provide the most timely case ascertainment, identifying more recently diagnosed cases. All four states have conducted outreach to certain specialty groups, primarily pulmonologists and occupational medicine specialists, to increase physician reporting of silicosis cases. Although Michigan and New Jersey have encouraged adoption of a national system for reporting aggregated readings by NIOSH-certified "B" readers (*11*), Michigan and Wisconsin have already begun providing pneumoconiosis classification of chest radiographs by "B" readers free of charge to physicians, companies, and individuals in those states. The utility and success of these targeting efforts have not been evaluated with regard to preventing silicosis and other pneumoconioses.

Although the surveillance data from each state tend to reflect the primary silica-using industries of that state, the data also have served to identify less-recognized occupational risks. For example, SENSOR surveillance has identified silicosis among workers employed in the dental supplies industry, a group that is not generally recognized as being at high risk for silicosis (*12*).

SENSOR data indicate that, despite a generally long latency period for this disease, follow-up of silicosis cases can help public health authorities identify ongoing hazardous exposures (*10*). The efficacy of such follow-up is illustrated by the findings of inspections in Michigan. Moreover, all states have found that workplace follow-up of a documented case often provides the employer particularly strong motivation to

control exposures through primary prevention strategies, such as material substitution and engineering controls. Thus, the case-based approach complements the usual industrywide approach to occupational hazard control (*9*).

The data initially reported to the SENSOR programs in Michigan, New Jersey, Ohio, and Wisconsin suggest that the SENSOR methods for case-based silicosis surveillance systems can be successfully implemented by state health departments, particularly in those states with mandated reporting requirements and/or state access to hospital discharge data (*6*). Based on the pioneering efforts of these four states, NIOSH has developed guidelines for state-based silicosis surveillance. New SENSOR cooperative agreements have been awarded to the four states that piloted these methods, as well as to three additional states—Illinois, North Carolina, and Texas—where the methods will be field tested to ascertain their applicability beyond the four original states. Based on the field-test results, a model surveillance system will be developed for implementation in all states interested in the prevention of silicosis.

References

1. Baker EL. Sentinel Event Notification System for Occupational Risks (SENSOR): the concept. Am J Public Health 1989;79(suppl):18–20.
2. CDC. Silicosis: cluster in sandblasters—Texas, and occupational surveillance for silicosis. MMWR 1990;39:433–7.
3. International Labour Office. Guidelines for the use of ILO International Classification of Radiographs of Pneumoconioses. Revised ed. Geneva, Switzerland: International Labour Office, 1980. Occupational safety and health series 22 (Rev.80).
4. National Institute for Occupational Safety and Health. Work-related lung disease surveillance report. Atlanta, GA: US Department of Health and Human Sevices, Public Health Service, CDC, 1991; DHHS (NIOSH) publication no. 91–113.
5. Windau J, Rosenman K, Anderson H, et al. The identification of occupational lung disease from hospital discharge data. J Occup Med 1991;33:1060–6.
6. National Research Council. Counting injuries and illnesses in the workplace: proposals for a better system. Washington, DC: National Academy Press, 1987.
7. Public Health Service. Healthy people 2000: national health promotion and disease prevention objectives. Washington, DC, 1991:305-6;DHHS publication no.(PHS) 91–50212.
8. Rosenman KD, Trimbath L, Stanbury M. Surveillance of occupational lung disease: comparison of hospital discharge data to physician reporting. Am J Public Health 1990;80:1257–8.
9. Rosenman KD. Use of hospital discharge data in the surveillance of occupational disease. Am J Ind Med 1988;13:281–9.
10. Valiante DJ, Rosenman KD. Does silicosis still occur? JAMA 1989;262:3003–7.
11. Rosenman KD, Valiante D. The identification of pneumoconiosis from hospital discharge data [letter]. JAMA 1990;263:3025.
12. Rosenman KD, Riley MJ, Watt FC. 1991 Annual report: silicosis in Michigan. Michigan State University/Michigan Department of Public Health. Lansing, Michigan: 1991.

Sensitivity of Multiple-Cause Mortality Data for Surveillance of Deaths Associated with Head or Neck Injuries

David E. Nelson, M.D., M.P.H.[1]
Jeffrey J. Sacks, M.D., M.P.H.[1]
R. Gibson Parrish, M.D.[2]
Daniel M. Sosin, M.D.[3]
Patricia McFeeley, M.D.[4]
Suzanne M. Smith, M.D., M.P.H.[1]

[1]*Division of Unintentional Injury Prevention, National Center for Injury Prevention and Control*
[2]*Surveillance and Information Systems Section, Surveillance and Programs Branch, Division of Environmental Hazards and Health Effects, National Center for Environmental Health*
[3]*State Branch, Division of Field Epidemiology, Epidemiology Program Office*
[4]*New Mexico Office of the Medical Investigator, Albuquerque, NM*

Summary

Problem/Condition: Multiple-cause mortality data was assessed as a source of information for surveillance of deaths associated with head or neck injuries.

Reporting Period Covered: 1985–1986

Description of System: Data on causes of death were abstracted from death certificates in New Mexico and coded according to criteria of the *International Classification of Diseases*, Ninth Revision (ICD-9). Deaths with an external cause-of-death (E) code as the underlying cause of death and one or more head or neck injury nature-of-condition (N) codes as contributing causes of death were considered head or neck injury deaths. These data were compared with data for head or neck injury deaths obtained from computerized records from the New Mexico Office of the Medical Investigator (OMI). Data for alcohol or drug use were abstracted from both systems.

Results: Of the 699 head or neck injury deaths coded by the New Mexico OMI system in 1985–1986, 536 were identified as head or neck injury deaths in multiple-cause mortality data (sensitivity = 76.7%). Firearms were the leading cause of head or neck injury deaths, followed by motor vehicles. Multiple-cause mortality data contained alcohol codes for only 3.7% of OMI records with blood alcohol concentrations ≥0.10 mg/dL and contained drug codes for none of the OMI records with positive toxicology tests for drugs.

Interpretation: The sensitivity of multiple-cause mortality data was relatively high for surveillance of head and neck injury deaths. This information source may be useful for conducting statewide surveillance for mortality from head injuries; however, the sensitivity and positive predictive value of these data require further assessment. Multiple-cause data substantially underestimate the extent of alcohol and drug involvement for head or neck injury deaths.

Actions Taken: The findings in this investigation have prompted further assessment of the usefulness of death certificate data for head injury surveillance.

INTRODUCTION

Although the incidence of head injuries has been estimated for local jurisdictions in the United States (*1–7*), surveillance at the state and national levels is needed to monitor such injuries and evaluate the impact of preventive measures. Some researchers have suggested that CDC's multiple-cause mortality data from death certificates could be used for such surveillance because the data are readily available and require no new collection system (*8*). Multiple-cause mortality data include underlying cause of death and up to 20 associated medical conditions, such as type of injury. Although no true standard exists for evaluating the validity of death certificate diagnoses, a medical examiner system is probably the best standard available because the cause of death in cases investigated by medical examiners is subject to more thorough investigation than are routine cases. Using 1985–1986 data from the New Mexico Office of the Medical Investigator* (OMI) as the standard, we compared the sensitivity of multiple-cause mortality data with data from deaths investigated by the OMI for ascertaining deaths caused by head or neck injuries.

METHODS

In New Mexico, by law, the OMI investigates deaths occurring on nonfederal lands that are of unknown cause or are sudden, violent, or untimely. (The OMI investigates similar types of deaths on federal lands only when invited by federal authorities). The chief medical investigator is a forensic pathologist who is responsible for appointing judicial district medical investigators. All district medical investigators are physicians.

Computerized OMI records were reviewed for all injury deaths among U.S. residents occurring in New Mexico during the period 1985–1986. All records with codes for subdural hematoma or head and neck injury were classified as head or neck injury deaths. In addition, records that might have included head or neck injury (OMI records coded as multiple injury, stab wound, gunshot wound, or child abuse) were reviewed manually; these deaths were classified as resulting from head or neck injury if there was any mention of injury to a head or neck anatomic structure. All head or neck injury deaths were categorized by external cause: motor vehicle, intentional and unintentional firearm discharge, fall, or other. Three injury epidemiologists reviewed and classified 27 records for which it was unclear whether the deaths were attributable to injury.

Head or neck injury deaths investigated by the OMI were classified as alcohol associated if tests indicated that the decedent had a blood alcohol concentration (BAC) ≥0.10 mg/dL; for records with more than one BAC, the highest recorded level was used for classification. Deaths were considered to be drug associated if toxicology tests indicated the presence of any narcotic, barbiturate, analgesic, sedative, antidepressant, stimulant, tranquilizer, cannabinoid, or other psychoactive substance.

*In New Mexico, medical examiners are known as medical investigators, and the state office responsible for investigating deaths is the Office of the Medical Investigator.

Using a broad definition for head or neck injury-associated deaths (Table 1), we reviewed multiple-cause mortality tapes for deaths among U.S. residents who died in New Mexico during the period 1985–1986. Multiple-cause mortality data are abstracted from death certificates and coded onto tapes according to criteria of the

TABLE 1. International Classification of Diseases, Ninth Revision (ICD-9), nature of condition (N) and external cause of death (E) codes used for multiple-cause mortality definitions

Nature of condition (N) codes	
Head injury	
800-804	Fracture of skull
830.0-830.1	Dislocation of jaw
850-854	Intracranial injury, excluding those with skull fracture
870-873	Open wound of ocular adnexa, eyeball, ear, other wound of head
905.0	Late effect of fracture of skull and face bones
907.0	Late effect of intracranial injury without mention of skull fracture
907.1	Late effects of injury to cranial nerve(s)
910	Superficial injury of face, neck, and scalp (except for eye)
918	Superficial injury of eye and adnexa
920	Contusion of face, scalp, and neck
921	Contusion of eye and adnexa
925.0	Crushing injury of face, scalp, and neck
950-951	Injury to optic nerve and pathways, injury to other cranial nerves
953.0	Injury to cervical nerve root
959.0	Injury, other and unspecified, to face and neck
Neck injury	
344.0	Quadriplegia
805.0-805.1	Fracture of cervical spine without mention of spinal cord injury
806.0-806.1	Fracture of cervical vertebral column with spinal cord injury
807.5-807.6	Fracture of larynx and trachea
839.0-839.1	Dislocation of cervical vertebra
847.0	Sprains and strains of neck
848.1-848.2	Sprains and strains of septal cartilage of nose, jaw, thyroid region
874	Open wound of neck
900	Injury to blood vessels of head and neck
905.1	Late effect of fracture of spine and trunk without mention of spinal cord injury
906.0	Late effect of open wound of head, neck, and trunk
907.2	Late effect of spinal cord injury
908.3	Late effect of injury to blood vessel of head, neck, and extremities
Alcohol use	
291	Alcoholic psychoses
303	Alcohol dependence syndrome
305.0	Alcohol abuse
790.3	Excessive blood level of alcohol
Drug use	
292	Drug psychoses
304	Drug dependence
305.2-305.9	Nondependent abuse of drugs
965	Poisoning by analgesics, antipyretics, and antirheumatics
967	Poisoning by sedatives and hypnotics
968.5	Poisoning by surface and infiltration anesthetics
969	Poisoning by psychotropic agents
970.0	Poisoning by analeptics
977.0	Poisoning by other and unspecified medicinal substances: dietetics
External cause-of-death (E) codes	
Motor vehicle:	810-825
Falls:	880–888
Firearms:	922.0-922.9, 955.0–955.4, 965.0–965.4, 970, 985.0–985.4
Other:	800-809, 826–879, 889–921, 923–954, 955.5–955.9, 956–964, 965.5–965.9, 966–969, 971–984, 985.5–985.9, 986–999

International Classification of Diseases, Ninth Revision (ICD-9) (*9*). We included all deaths with an external cause-of-death (E) code as the underlying cause and one or more nature-of-condition (N) code for head or neck injury as a contributing cause (Table 1). Categories for external cause were motor vehicle, unintentional or intentional firearm discharge, fall, or other. Multiple-cause mortality head or neck injury deaths were defined as alcohol or drug associated if certain codes were present (Table 1).

To assess the sensitivity of multiple-cause mortality data for ascertaining head or neck injury deaths, we matched OMI and multiple-cause mortality records involving head or neck injuries by sex, race, age, and date of death. We included 30 records from the OMI system that matched with a corresponding record from the vital records system for each variable except age, after manual review showed matching birth dates but different ages.

Multiple-cause mortality data contain information about county of death, but do not indicate whether deaths occurred on federal or nonfederal land. (Federal lands are present in the majority of New Mexico counties.) As a result, the specificity and positive predictive value of multiple-cause mortality data for head or neck injuries could not be calculated.

RESULTS

Of 699 head or neck injury deaths included in the OMI system, 536 were also in multiple-cause mortality data, for an overall sensitivity of 76.7%. The sensitivity was highest for deaths coded for firearms and lowest for those coded for falls (Table 2). Deaths in the OMI system not identified by multiple-cause mortality data were similar by race and sex to those found in both systems, but differed significantly by age ($p<0.02$) (Table 3).

Most head or neck injury deaths included in multiple-cause mortality data were coded under five general rubrics (Table 4). Of the 536 deaths common to both systems, 486 (90.7%) were coded as due to intracranial injury (N850–N854) or open wound of the eye, ear, or head (N870–N873). (The majority of the latter were coded as N873.1—open wound of the scalp.) All 15 multiple-cause mortality records with neck injury codes also had head injury codes.

OMI records indicated that 460 (85.8%) of 536 persons had been tested for the presence of alcohol or drugs, 168 (36.5%) of whom had positive tests. The sensitivity of multiple-cause mortality data was 3.7% (5/134) for ascertaining deaths associated with

TABLE 2. Sensitivity of multiple-cause mortality data for identifying head or neck injury deaths in the Office of the Medical Investigator (OMI) records, New Mexico, by external cause of death — 1985–1986

External cause	No. records		Sensitivity
	OMI data	Multiple-cause mortality data	
Firearms	303	277	91.4%
Motor vehicle	247	172	69.6%
Other	96	63	65.6%
Fall	53	24	45.3%
Total	**699**	**536**	**76.7%**

BACs ≥0.10 mg/dL and no evidence of drug use, 0% for 17 deaths associated with drugs, and 0% for 17 deaths associated with both alcohol and drugs.

DISCUSSION

In the OMI system, head and neck injuries could not be separated. Because all deaths with neck injury codes reported in multiple-cause mortality data also had head injury codes, our study may be more representative of deaths resulting from head rather than neck injury.

Multiple-cause mortality data had fairly high sensitivity for ascertaining deaths reported in the OMI system that involved head or neck injury, especially for deaths associated with firearms. However, multiple-cause mortality data were not useful for identifying alcohol- and drug-associated deaths due to these injuries. Underreporting of alcohol use on death certificates has been noted before (*10*), but the extent of un-

TABLE 3. Demographic characteristics of persons with head or neck injury deaths identified in both the multiple-cause mortality and Office of the Medical Investigator (OMI) systems, compared with persons with head or neck injury deaths identified only by records — New Mexico, 1985–1986

	Identified in both systems		Identified only in OMI system		
Characteristic	N	(%)	N	(%)	P-value*
Median age (years)	30		35		0.02
Sex					
Male	417	(77.8)	121	(74.2)	
Female	119	(22.2)	42	(25.8)	0.40
Race					
White	471	(87.8)	138	(84.7)	
Black	10	(1.9)	2	(1.2)	0.35
Other	55	(10.3)	23	(14.1)	

*The Kruskal-Wallis and chi-square tests were used to compare a) median age and b) sex and race distributions, respectively.

TABLE 4. ICD-9* Nature-of-condition codes for 536 persons with head or neck injuries common to the multiple-cause mortality data and Office of the Medical Investigator systems — New Mexico, 1985–1986

	Number and percent of deaths with nature-of-condition code†				
Cause	Skull fracture (800-804)	Intracranial injury (850-854)	Open wound of eye, ear, or head (870-873)	Spinal cord injury (952.0)	Other and unspecified face and neck injury (959.0)
Firearms (n=277)	1 (0.4)	4 (1.4)	276 (99.6)	0 (0.0)	0 (0.0)
Motor vehicle (n=172)	4 (2.3)	169 (98.3)	0 (0.0)	1 (0.6)	10 (5.8)
Other (n=63)	4 (6.3)	52 (82.5)	15 (23.8)	0 (0.0)	3 (4.8)
Falls (n=24)	2 (8.3)	23 (95.8)	0 (0.0)	0 (0.0)	1 (4.2)

**International Classification of Diseases*, 9th Revision.
†Numbers in parentheses are row percents and total more than 100 because some death certificates had >1 nature-of-condition code; similarly, individual row numbers do not equal row totals because multiple codes were assigned to one event.

derreporting of alcohol use on death certificates with head or neck injury codes has not been previously reported. Death certifiers should obtain drug and alcohol tests and report the results on death certificates, even if delays in obtaining test results make it necessary to file supplemental reports.

In contrast to previous findings (*2,4–8*), firearms were the leading cause of head injury deaths. This finding underscores the importance of including open wounds of the eye, ear, or head when these deaths are defined, since most head injury deaths from firearms had one of those codes. In a study in which multiple-cause mortality data were used without the category for open wound of the eye, ear, or head, 315,328 deaths from head injury were found in the United States from 1979 through 1986 (*8*). When we reanalyzed the data and included these codes, we found an additional 102,404 deaths due to head injury, of which 97.0% contained an underlying cause of death attributed to firearms (*11*). This finding may indicate that nosologists use codes for open wounds to the eye, ear, or head inappropriately for head injury deaths, since these codes should be used for wounds that were incidental to intracranial injury (*9*).

The ICD codes used by researchers to define head injury have varied widely (*3,5–8*). Although we searched multiple-cause mortality records for a wide range of ICD-9 codes corresponding to head injury, we found that >90% of deaths due to head injury were coded as intracranial injury or open wound of the eye, ear, or head. Further exploration is needed to determine the adequacy of a surveillance system for head injury mortality that uses a simple case definition with these two categories.

Although multiple-cause mortality data underascertain head or neck injury deaths from falls, our results demonstrate that death certificate data may be appropriate and useful for monitoring head injury deaths. Determining the sensitivity and positive predictive value is needed in other localities so that the use of multiple-cause mortality data as a surveillance system for mortality from head injury can be fully assessed. If multiple-cause mortality data are found to have adequate sensitivity and positive predictive value, then, at a minimal cost, state health departments could use these data for head injury mortality surveillance to monitor patterns, set priorities, and evaluate the effects of efforts to prevent head injuries.

Acknowledgment

We thank Patricia Holmgreen, M.S., for assistance in data analysis and James Mercy, Ph.D., and Richard Sattin, M.D., for their editorial comments.

References

1. Kraus JF. Epidemiology of head injury. In: PR Cooper, ed. Head injury. Baltimore: Williams and Wilkins, 1987:1–19.
2. Annegers JF, Grabow JD, Kurland LT, Laws ER. The incidence, causes, and secular trends of head trauma in Olmsted County, Minnesota, 1935–1974. Neurology 1980;30:912–9.
3. Cooper KD, Tabaddor K, Hauser WA, et al. The epidemiology of head injury in the Bronx. Neuroepidemiology 1983;2:70–88.
4. Jagger J, Levine JI, Jane JA, Rimel RW. Epidemiologic features of head injury in a predominantly rural population. J Trauma 1984;24:40–4.
5. Klauber MR, Barrett-Connor E, Marshall LF, Bowers SA. The epidemiology of head injury: a prospective study of an entire community—San Diego County, California, 1978. Am J Epidemiol 1981;113:500–9.
6. Whitman S, Coonley-Hoganson R, Desai BT. Comparative head trauma experiences in two socioeconomically different Chicago-area communities: a population study. Am J Epidemiol 1984;119:570–80.

7. Kraus JF, Black MA, Hessol N, et al. The incidence of acute brain injury and serious impairment in a defined population. Am J Epidemiol 1984;119:186–201.
8. Sosin DM, Sacks JJ, Smith SM. Head injury-associated deaths in the United States, 1979–1986. JAMA 1989;262:2251–5.
9. Manual of international classification of diseases, injuries, and causes of death, 9th revision. Geneva: World Health Organization, 1977.
10. Pollock DA, et al.: Underreporting of alcohol-related mortality on death certificates of young US army veterans. JAMA 1987;258:345–8.
11. Sosin DM, Nelson DE, Sacks JJ. Head injury deaths: the enormity of firearms [letter]. JAMA 1992;268:791.

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 September 17, 1993, and the laboratory directors listed below were in the positions shown as of April 1993.

State/Territory	Epidemiologist	Laboratory Director
Alabama	Charles H. Woernle, MD, MPH	William J. Callan, PhD
Alaska	John P. Middaugh, MD	Katherine A. Kelley, DrPH
Arizona	Lawrence Sands, DO, MPH (Acting)	Barbara J. Erickson, PhD
Arkansas	Thomas C. McChesney, DVM	Robert L. Horn
California	George W. Rutherford, MD	Michael G. Volz, PhD
Colorado	Richard E. Hoffman, MD, MPH	Ronald L. Cada, DrPH
Connecticut	James L. Hadler, MD, MPH	Sanders F. Hawkins, PhD (Acting)
Delaware	A. LeRoy Hathcock, Jr., Ph.D.	Mahadeo P. Verma, PhD
District of Columbia	Martin E. Levy, MD, MPH	James B. Thomas, ScD
Florida	Richard S. Hopkins, MD, MSPH	E. Charles Hartwig, ScD
Georgia	Kathleen E. Toomey, MD, MPH	Elizabeth A. Franko, DrPH (Acting)
Hawaii	Richard L. Vogt, MD	Vernon K. Miyamoto, PhD
Idaho	Fritz R. Dixon, MD	Richard H. Hudson, PhD
Illinois	Byron J. Francis, MD, MPH	David F. Carpenter, PhD
Indiana	Mary Lou Fleissner, DrPH	Gregory V. Hayes, DrPH
Iowa	Laverne A. Wintermeyer, MD	W. J. Hausler, Jr, PhD
Kansas	Andrew R. Pelletier, MD	Roger H. Carlson, PhD
Kentucky	Reginald Finger, MD, MPH	Thomas E. Maxson, DrPH
Louisiana	Louise McFarland, DrPH	Henry B. Bradford, Jr, PhD
Maine	Kathleen F. Gensheimer, MD	Philip W. Haines, DrPH
Maryland	Ebenezer Israel, MD, MPH	J. Mehsen Joseph, PhD
Massachusetts	Alfred DeMaria, Jr, MD	Ralph J. Timperi, MPH
Michigan	Kenneth R. Wilcox, Jr, MD, DrPH	Robert Martin, DrPH
Minnesota	Michael T. Osterholm, PhD, MPH	Pauline Bouchard, JD, MPH
Mississippi	Mary Currier, MD, MPH	R. H. Andrews, MPH
Missouri	H. Denny Donnell, Jr, MD, MPH	Eric C. Blank, DrPH
Montana	Todd Damrow, PhD, MPH	Douglas Abbott, PhD
Nebraska	Thomas J. Safranek, MD	John Blosser
Nevada	Vacant	Arthur F. DiSalvo, MD
New Hampshire	M. Geoffrey Smith, MD, MPH	Veronica C. Malmberg
New Jersey	Kenneth C. Spitalny, MD	Shahiedy I. Shahied, PhD
New Mexico	C. Mack Sewell, DrPH, MS	Loris W. Hughes, PhD
New York City	Kelly J. Henning, MD (Acting)	Stanley Reimer
New York State	Guthrie Birkhead, MD, MPH	Lawrence Sturman, MD, PhD
North Carolina	J. Newton MacCormack, MD, MPH	Samuel N. Merritt, DrPH
North Dakota	Larry Shireley, MS, MPH	James L. Peerson, DrPH
Ohio	Thomas J. Halpin, MD, MPH	Gary D. Davidson, DrPH
Oklahoma	Paul Zenker, MD, MPH	Garry L. McKee, PhD
Oregon	David Fleming, MD	Charles D. Brokopp, DrPH
Pennsylvania	Dale R. Tavris, MD, MPH	Bruce Kieger, DrPH (Acting)
Rhode Island	Bela Matyas, MD, MPH	Raymond G. Lundgren, Jr., PhD
South Carolina	Dee Breeden, MD, MPH (Acting)	Harold Dowda, PhD
South Dakota	Kenneth A. Senger	Kathleen L. Meckstroth, DrPH
Tennessee	Robert H. Hutcheson, MD, MPH	Michael W. Kimberly, DrPH
Texas	Diane M. Simpson, MD, PhD	Charles E. Sweet, DrPH
Utah	Craig R. Nichols, MPA	A. Richard Melton, DrPH
Vermont	Robert Houseknecht, PhD	Burton W. Wilcke, Jr, PhD
Virginia	Grayson B. Miller, Jr, MD	D.B. Smit (Acting)
Washington	John Kobayashi, MD, MPH	Jon M. Counts, DrPH
West Virginia	Loretta E. Haddy, MA, MS	Frank W. Lambert, Jr, DrPH
Wisconsin	Jeffrey P. Davis, MD	Ronald H. Laessig, PhD
Wyoming	Stanley I. Music, MD, DTPH	Carl H. Blank, DrPH
American Samoa	Julia L. Lyons, MD, MPH	—
Federated States of Micronesia	Steven Auerbach, MD, MPH	—
Guam	Robert L. Haddock, DVM, MPH	Jeff Benjamin (Acting)
Marshall Islands	Tony de Brum	—
Northern Mariana Islands	Sean P. Flood, MD, MPH	—
Palau	Yugi Mesubed, MD	—
Puerto Rico	John V. Rullan, MD, MPH	Raul Baco Dapena
Virgin Islands	Alfred O. Heath, MD	Norbert Mantor, PhD

MMWR

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available on a paid subscription basis from the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 783-3238.

The data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday. Inquiries about the *MMWR* Series, including material to be considered for publication, should be directed to: Editor, *MMWR* Series, Mailstop C-08, Centers for Disease Control and Prevention, Atlanta, GA 30333; telephone (404) 332-4555.

☆**U.S. Government Printing Office: 1993-733-131/83042 Region IV**