

**Influenza Surveillance—United States,
1992–93 and 1993–94**

**Surveillance for Silicosis, 1993—Illinois,
Michigan, New Jersey, North Carolina,
Ohio, Texas, and Wisconsin**

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, GA 30333.

SUGGESTED CITATION

General: Centers for Disease Control and Prevention. *CDC Surveillance Summaries*, January 31, 1997. MMWR 1997;46(No. SS-1).
Specific: [Author(s)]. [Title of particular article]. In: *CDC Surveillance Summaries*, January 31, 1997. MMWR 1997;46(No. SS-1):[inclusive page numbers].

Centers for Disease Control and Prevention David Satcher, M.D., Ph.D.
Director

The production of this report as an *MMWR* serial publication was coordinated in:

Epidemiology Program Office..... Stephen B. Thacker, M.D., M.Sc.
Director

Richard A. Goodman, M.D., M.P.H.
Editor, MMWR Series

Scott F. Wetterhall, M.D., M.P.H.
Associate Editor, CDC Surveillance Summaries

Office of Scientific and Health Communications (Proposed)

CDC Surveillance Summaries Suzanne M. Hewitt, M.P.A.
Managing Editor

Ava W. Navin, M.A.
Project Editor

Peter M. Jenkins
Visual Information Specialist

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

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

Contents

Reports Published in <i>CDC Surveillance Summaries</i> Since January 1, 1985	ii
Influenza Surveillance—United States, 1992–93 and 1993–94.....	1
Introduction.....	2
Methods.....	2
Results	3
Discussion	10
References.....	11
Surveillance for Silicosis, 1993—Illinois, Michigan, New Jersey, North Carolina, Ohio, Texas, and Wisconsin	13
Introduction.....	14
Methods.....	15
Results	18
Discussion	23
References.....	27
State and Territorial Epidemiologists and Laboratory Directors.....	inside back cover

Reports Published in *CDC Surveillance Summaries* Since January 1, 1985

Subject	Responsible CIO/Agency*	Most Recent Report
Abortion	NCCDPHP	1996; Vol. 45, No. SS-3
AIDS/HIV		
Distribution by Racial/Ethnic Group	NCID	1988; Vol. 37, No. SS-3
Among Black & Hispanic Children & Women of Childbearing Age	NCEHIC	1990; Vol. 39, No. SS-3
Behavioral Risk Factors	NCCDPHP	1996; Vol. 45, No. SS-6
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 & Cervical Cancer	NCCDPHP	1992; Vol. 41, No. SS-2
<i>Campylobacter</i>	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
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	1994; Vol. 43, No. SS-2
Dental Caries & Periodontal Disease Among Mexican-American Children	NCPS	1988; Vol. 37, No. SS-3
Developmental Disabilities	NCEH	1996; Vol. 45, No. SS-2
Diabetes Mellitus	NCCDPHP	1993; Vol. 42, No. SS-2
Dracunculiasis	NCID	1992; Vol. 41, No. SS-1
Ectopic Pregnancy	NCCDPHP	1993; Vol. 42, No. SS-6
Elderly, Hospitalizations Among	NCCDPHP	1991; Vol. 40, No. SS-1
Endometrial & Ovarian Cancers	EPO, NCCDPHP	1986; Vol. 35, No. 2SS
<i>Escherichia coli</i> O157	NCID	1991; Vol. 40, No. SS-1
Evacuation Camps	EPO	1992; Vol. 41, No. SS-4
Family Planning Services at Title X Clinics	NCCDPHP	1995; Vol. 44, No. SS-2
Foodborne Disease	NCID	1996; Vol. 45, No. SS-5
Gonorrhea & Syphilis, Teenagers	NCPS	1993; Vol. 42, No. SS-3
Hazardous Substances Emergency Events	ATSDR	1994; Vol. 43, No. SS-2
Health Surveillance Systems	IHPO	1992; Vol. 41, No. SS-4
Hepatitis	NCID	1985; Vol. 34, No. 1SS
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	1997; Vol. 46, No. SS-1
Injury		
Death Rates, Blacks & 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

***Abbreviations**

ATSDR	Agency for Toxic Substances and Disease Registry
CIO	Centers/Institute/Offices
EPO	Epidemiology Program Office
IHPO	International Health Program Office
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
NCPS	National Center for Prevention Services
NIOSH	National Institute for Occupational Safety and Health
NIP	National Immunization Program

Reports Published in *CDC Surveillance Summaries* Since January 1, 1985 — Continued

Subject	Responsible CIO/Agency*	Most Recent Report
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
Motor Vehicle-Related Deaths	NCEHIC	1988; Vol. 37, No. SS-1
Objectives of Injury Control, State & 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	NCID	1995; Vol. 44, No. SS-5
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	NIOSH	1986; Vol. 35, No. 2SS
Mumps	NIP	1995; Vol. 44, No. SS-3
National Infant Mortality (see also Infant Mortality; Birth Defects)	NCCDPHP	1989; Vol. 38, No. SS-3
<i>Neisseria gonorrhoeae</i> , Antimicrobial Resistance in	NCPS	1993; Vol. 42, No. SS-3
Neural Tube Defects	NCEH	1995; Vol. 44, No. SS-4
Nosocomial Infection	NCID	1986; Vol. 35, No. 1SS
Occupational Injuries/Disease		
Asthma	NIOSH	1994; Vol. 43, No. SS-1
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
Parasites, Intestinal	NCID	1991; Vol. 40, No. SS-4
Pediatric Nutrition	NCCDPHP	1992; Vol. 41, No. SS-7
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
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	1993; Vol. 42, No. SS-6
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
Rotavirus	NCID	1992; Vol. 41, No. SS-3
<i>Salmonella</i>	NCID	1988; Vol. 37, No. SS-2
Sexually Transmitted Diseases in Italy	NCPS	1992; Vol. 41, No. SS-1
Silicosis	NIOSH	1997; Vol. 46, No. SS-1
Smoking	NCCDPHP	1990; Vol. 39, No. SS-3
Smoking-Attributable Mortality	NCCDPHP	1994; Vol. 43, No. SS-1
Tobacco Control Laws, State	NCCDPHP	1995; Vol. 44, No. SS-6
Tobacco-Use Behaviors	NCCDPHP	1994; Vol. 43, No. SS-3
Spina Bifida	NCEH	1996; Vol. 45, No. SS-2
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
Syphilis, Congenital	NCPS	1993; Vol. 42, No. SS-6
Syphilis, Primary & Secondary	NCPS	1993; Vol. 42, No. SS-3
Tetanus	NCPS	1992; Vol. 41, No. SS-8
Trichinosis	NCID	1991; Vol. 40, No. SS-3
Tuberculosis	NCPS	1991; Vol. 40, No. SS-3
Waterborne Disease Outbreaks	NCID	1996; Vol. 45, No. SS-1
Years of Potential Life Lost	EPO	1992; Vol. 41, No. SS-6
Youth Risk Behaviors	NCCDPHP	1996; Vol. 45, No. SS-4

Influenza Surveillance— United States, 1992–93 and 1993–94

Lynnette Brammer, M.P.H.

Keiji Fukuda, M.D., M.P.H.

Nancy Arden, M.N.

Leone M. Schmeltz

Lone Simonsen, Ph.D.

Ali Khan, M.D.

Helen L. Regnery, Ph.D.

Lawrence B. Schonberger, M.D., M.P.H.

Nancy J. Cox, Ph.D.

*Division of Viral and Rickettsial Diseases
National Center for Infectious Diseases*

Abstract

Problem/Condition: CDC conducts active surveillance annually from October through May on the emergence and spread of influenza virus variants and the impact of influenza-related morbidity and mortality. Influenza activity is also monitored throughout the year by passive surveillance.

Reporting Period Covered: This report summarizes U.S. influenza surveillance from October 1992 through May 1994.

Description of System: Influenza surveillance comprises four components, three of which provide weekly data from October through May: a) state and territorial epidemiologists provide estimates of local influenza activity; b) approximately 140 sentinel physicians report their total number of patient visits and the number of cases of influenza-like illness; and c) approximately 70 collaborating laboratories of the World Health Organization (WHO) report weekly influenza virus isolations and submit selected influenza isolates to CDC for antigenic analysis. Throughout the year, vital statistics offices of 121 cities report deaths related to pneumonia and influenza (P&I), providing an index of the impact of influenza on mortality.

Results: Influenza B viruses predominated during the 1992–93 influenza season, but influenza A(H3N2) isolates increased and were associated with outbreaks in nursing homes at the end of the season. The increase in influenza A(H3N2) activity was associated with a rise in P&I-related mortality. Preseason outbreaks of influenza A(H3N2) virus were reported during August and September 1993 in Louisiana. In the past, preseason outbreaks of influenza have been associated with earlier than usual epidemic-level activity. During the 1993–94 influenza season, activity rose during November and December and peaked earlier than usual, during the last week of December and the first week of January; influenza A(H3N2) viruses predominated.

Interpretation: The change in predominance from influenza B to influenza A in the spring of 1993 emphasizes the importance of annual influenza surveillance. Although influenza vaccine is effective against both influenza A and B, the antiviral drugs amantadine and rimantadine are effective only against influenza A. Outbreaks during the

summer of 1993 emphasize that influenza should be considered a possible cause of respiratory infections during summer and early autumn.

Actions Taken: Surveillance data were provided weekly throughout the influenza season to public health officials, WHO, and health-care providers.

INTRODUCTION

Influenza remains a cause of substantial morbidity and mortality in the United States (1). During influenza epidemics, visits to physicians, clinics, and emergency rooms, as well as hospitalizations caused by influenza-related complications, may increase. Persons ≥ 65 years of age and those with underlying chronic health conditions, including cardiovascular disease, pulmonary disease, and certain metabolic conditions are at increased risk for complications of influenza infection and are more likely than the general population to be hospitalized if infected. During major epidemics, hospitalization rates among the elderly and persons who have underlying chronic health problems may increase twofold to fivefold compared with nonepidemic periods (2). Influenza epidemics also are associated with increased mortality. Since 1968–69, the influenza virus subtype generally associated with the highest mortality has been influenza A(H3N2). During nine of 20 influenza seasons (from 1972–73 through 1991–92), $>20,000$ influenza-associated excess deaths occurred each season; during four of these seasons, $>40,000$ deaths occurred (CDC, unpublished data). Although there is season-to-season variability, in recent years $>90\%$ of influenza-associated deaths have occurred among persons ≥ 65 years of age (3).

Annual vaccination of persons at high risk for influenza-associated complications is the most effective means of reducing the impact of influenza and is recommended by the Advisory Committee on Immunization Practices for these persons and their frequent contacts (3). Typically, one or two of the three vaccine components are updated each year because influenza viruses undergo continual antigenic change. Eventually, the circulating viruses change enough that the previous year's vaccine offers diminished protection. Even when circulating viruses remain relatively unchanged, immunity induced by the influenza vaccine declines over time. Antiviral agents, which are effective only against type A influenza viruses, can be a useful adjunct to vaccination, but chemoprophylaxis is not a substitute for vaccination.

CDC conducts active influenza surveillance each year from October through May* to provide timely information to health-care providers and the general public regarding current influenza activity levels and circulating virus types. In addition, information about the worldwide circulation of virus strains forms the basis for selecting the optimal vaccine components for the following year. This report summarizes influenza activity in the United States for the influenza seasons from October 1992 through May 1994.

METHODS

Sources of data for influenza surveillance during the 1992–93 and 1993–94 seasons were similar to those in previous years:

*Reports from the 121 Cities Surveillance System are monitored throughout the year.

State and Territorial Reports. Statewide or territorywide influenza activity, as assessed by the state or territorial epidemiologist, was reported weekly from October through May as either widespread (outbreaks of influenza-like illness [ILI]* or culture-confirmed influenza in counties having a combined population of $\geq 50\%$ of the state's population), regional (outbreaks of ILI or culture-confirmed influenza in counties having a combined population of $< 50\%$ of the state's total population), sporadic (sporadically occurring cases of ILI or culture-confirmed influenza, with no outbreaks detected), or no activity.

Sentinel Physician Surveillance Network. Each week from October through May, approximately 140 volunteer family-practice physicians reported the number of patient visits per week, the number of those patients examined for ILI,[†] and the number of hospitalizations for ILI or related complications, by age group. A subset of the physicians submitted nasal and throat swabs to a contract laboratory for virus isolation.

World Health Organization (WHO) Collaborating Laboratories. Each week from October through May, approximately 70 WHO collaborating laboratories in the United States reported the total number of specimens received for respiratory virus testing and the number of positive isolates of influenza A(H1N1), A(H3N2), A(not subtyped), or influenza B, by age group (< 1 , 1–4, 5–24, 25–44, 45–64, and ≥ 65 years of age, or unknown). Most WHO laboratories were in state or local health departments, with a few in universities or hospitals. A subset of the isolates obtained in these laboratories was submitted for complete antigenic characterization and antiviral resistance testing to the WHO Collaborating Center for Surveillance, Epidemiology and Control of Influenza, CDC.

121 Cities Surveillance System. Each week throughout the year, the vital statistics offices of 121 cities reported the total number of death certificates by week and the number of death certificates in which pneumonia was identified as the underlying cause of death or in which influenza was mentioned in any position on the certificate. These data were used to calculate a pneumonia and influenza (P&I) mortality curve. A periodic regression model that incorporated a robust regression procedure was applied to produce a seasonal baseline of P&I deaths since 1983 and to calculate "excess" deaths above the baseline. An increase of 1.645 standard deviations above the seasonal baseline of P&I deaths was considered the epidemic threshold (i.e., the point at which the observed proportion of deaths attributed to pneumonia or influenza was significantly higher than the deaths expected at that time of the year in the absence of substantial influenza-related mortality).

RESULTS

1992–93 Influenza Season

Reports from State and Territorial Epidemiologists

State and territorial epidemiologists first reported sustained regional influenza activity during the week ending December 19, 1992 (week 51), and widespread activity

*For this surveillance component, the case definition for "influenza-like illness" is left to the discretion of each state and territorial health department.

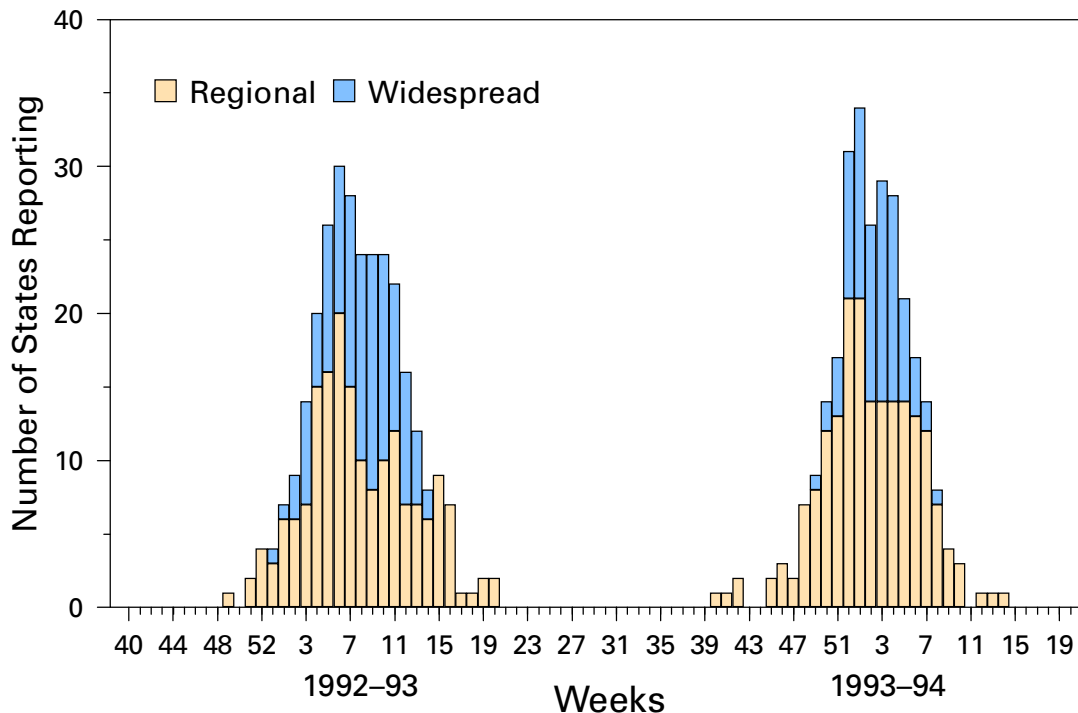
[†]"Influenza-like illness" is defined for this surveillance component as an illness characterized by fever ≥ 100 and cough or sore throat, in the absence of other confirmed diagnosis.

2 weeks later. This surveillance system component indicated that influenza activity in the United States peaked the week ending February 13, 1993 (week 6), when state and territorial epidemiologists reported regional or widespread activity in 30 states. Peak activity continued through the week ending February 20, 1993 (week 7), with 28 states reporting regional or widespread activity. No state reported widespread activity after the week ending April 10, 1993 (week 14). Regional activity was reported through the week ending May 22, 1993 (week 20) (Figure 1).

Sentinel Physicians Surveillance Network

Nationwide, the percentage of patient visits to sentinel physicians for ILI rose above baseline levels (0%–3%) during the week ending December 26, 1992 (week 52), peaked at 6% during the weeks ending February 6 and February 13, 1993 (weeks 5 and 6), and returned to baseline levels during the week ending March 20, 1993 (week 11) (Figure 2). On a regional level, the mountain region reported an earlier peak in activity and a higher percentage of patient visits due to ILI than the rest of the nation (Figure 3). Influenza activity in the New England region lagged behind the rest of the nation and peaked during the week ending March 13, 1993 (week 10).

FIGURE 1. Number of state and territorial health departments reporting influenza activity during the 1992–93 and 1993–94 influenza seasons, by week of report and extent of activity — United States

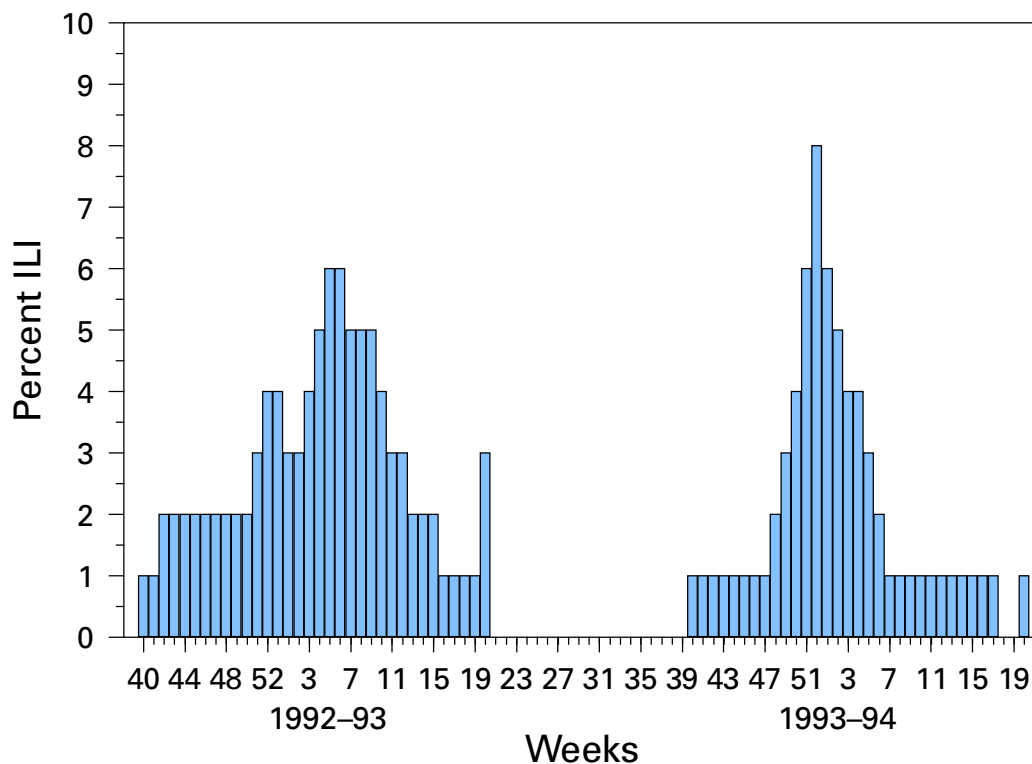


*Based on reports from 50 states and Washington D.C.

Widespread activity — outbreaks of influenza-like illness (ILI) or culture-confirmed influenza in counties having a combined population of $\geq 50\%$ of the state's population.

Regional activity — outbreaks of ILI or culture-confirmed influenza in counties having a combined population of $< 50\%$ of the state's total population.

FIGURE 2. Percentage of visits to physicians' offices attributed to influenza-like illness (ILI) — United States, 1992–94



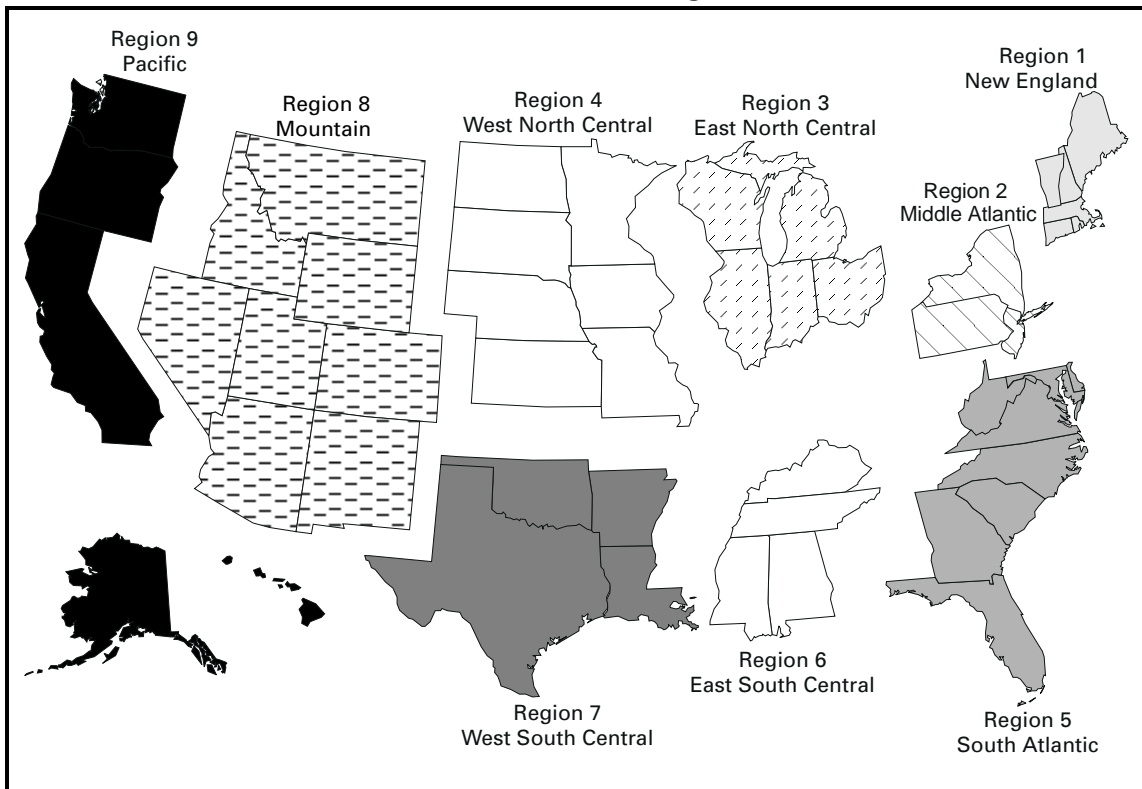
Patients in the age groups 5–24 years and 25–44 years accounted for 29% and 28%, respectively, of visits for ILI. Persons ≥ 65 years of age accounted for only 10% of physician visits for ILI but 50% of ILI-associated hospitalizations.

Ninety-eight physicians submitted 290 respiratory specimens for virus isolation, of which 58 (20%) were positive for influenza. Of these, 15 (26%) were influenza type A and 43 (74%) were influenza type B.

WHO Collaborating Laboratories

The WHO collaborating laboratories tested 36,452 specimens for respiratory viruses during the 1992–93 influenza season. From September 27, 1992, through May 22, 1993, 4,377 influenza viruses were isolated, of which 3,089 (71%) were influenza type B and 1,288 (29%) were influenza type A (Figure 4). Of the 717 influenza type A viruses that were subtyped, 645 (90%) were A(H3N2) and 72 (10%) were A(H1N1).

Overall, influenza activity as determined by the WHO collaborating laboratories peaked during the week ending February 13, 1993 (week 6), during which 410 influenza isolates were reported. Influenza B virus isolates were reported first during the week ending October 24, 1992 (week 43). The number of influenza B isolates increased during December and peaked during the week ending February 13, 1993 (week 6), but continued to be reported through the week ending May 22, 1993 (week 20). The first influenza A isolate was reported during the week ending November 7, 1992 (week 45), but isolates were not reported during each week until the week ending January 9,

FIGURE 3. United States influenza surveillance regions

1993 (week 1). The number of influenza A isolates peaked during the week ending March 13, 1993 (week 10) (Figure 5).

Nationally, influenza B viruses predominated overall, but influenza A isolates were reported more frequently than influenza B isolates from the week ending March 27, 1993 (week 12), through the week ending May 22, 1993 (week 20). Virus isolation patterns differed by region, however, with influenza A viruses predominating in New England and the Middle Atlantic region. Influenza B viruses were isolated more frequently than influenza A throughout the season in the East South Central region.

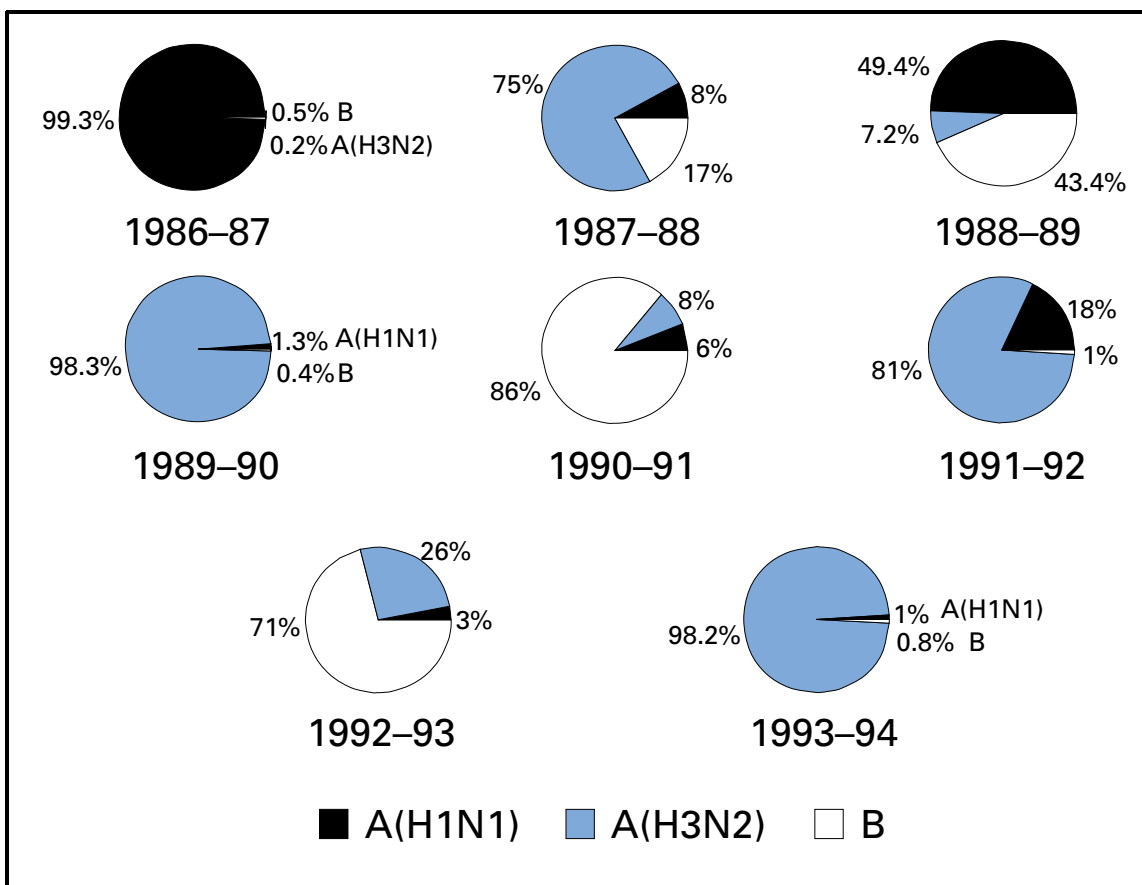
Virtually all influenza type B viruses isolated in the United States and antigenically characterized by CDC's WHO Collaborating Center for Surveillance, Epidemiology, and Control of Influenza were similar to B/Panama/45/90, the influenza B component of the 1992-93 influenza vaccine. All influenza A(H1N1) viruses were related to A/Texas/36/91, the H1N1 component of the 1992-93 vaccine, or the antigenically related A/Taiwan/01/86 strain. Most of the influenza A(H3N2) viruses isolated and characterized during the 1992-93 season were similar to the antigenic variant A/Beijing/32/92 included in the 1993-94 influenza vaccine; the remaining influenza A(H3N2) viruses were antigenically similar to the 1992-93 influenza vaccine component A/Beijing/353/89 (Table 1). Persons vaccinated with the 1992-93 influenza vaccine, which contained A/Beijing/353/89-like virus, demonstrated a diminished serologic response to A/Beijing/32/92-like viruses.

TABLE 1. Hemagglutination-inhibition reactions of influenza A(H3N2) viruses with antisera*

Viral antigen	Ferret antiserum	
	A/Beijing/353/89	A/Beijing/32/92
A/Beijing/353/89	320	80
A/Beijing/32/92	40	320

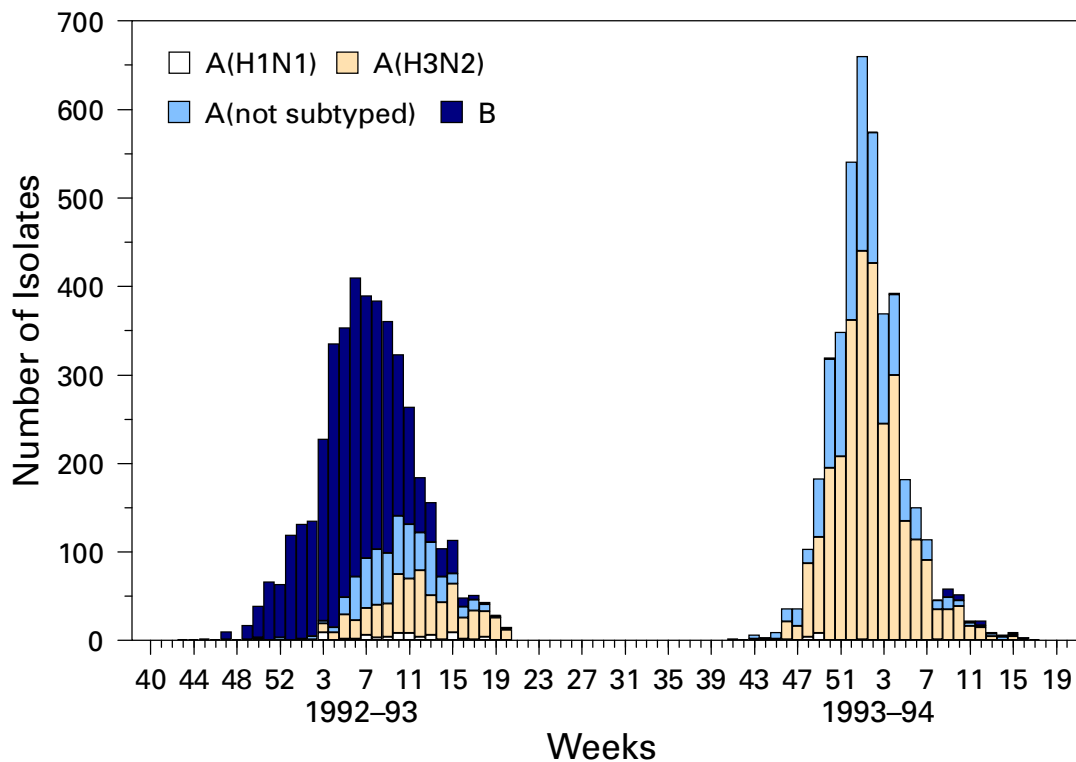
* A fourfold difference in hemagglutination-inhibition titers between two viruses is indicative of antigenic variation between viruses.

FIGURE 4. Influenza isolates* reported by the World Health Organization collaborating laboratories — United States, 1986–94



* Not all influenza A isolates were subtyped. The percentages were based on those subtyped.

FIGURE 5. Influenza virus isolates reported by the World Health Organization collaborating laboratories — United States, 1992–93 and 1993–94 influenza seasons



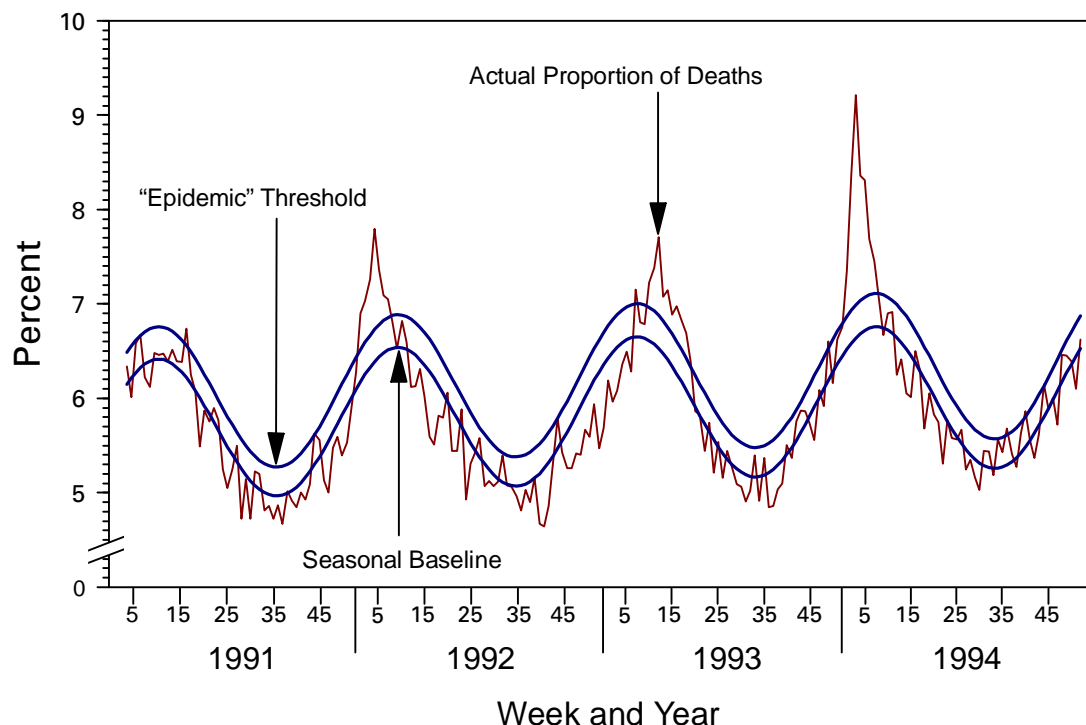
121 Cities Surveillance System

From October 3, 1992, through March 5, 1993, the proportion of deaths attributable to P&I slightly exceeded the epidemic threshold during the weeks ending October 24, 1992 (week 43), and February 20, 1993 (week 7). Sustained excess mortality began during the week ending March 13, 1993 (week 10), and continued for 10 weeks, through the week ending May 15, 1993 (week 19). The proportion of all deaths associated with P&I peaked at 7.7% during the week ending March 27, 1993 (week 12), which was substantially higher than the expected baseline of 6.46% for this week (Figure 6).

Outbreak Surveillance

The first reported culture-confirmed influenza B outbreak of the 1992–93 influenza season began during the week ending December 5, 1992 (week 45). The outbreak occurred among students in a high school in Washington State and was associated with an absenteeism rate of 12% (4). Most outbreaks reported early in the season were among school-age children; however, influenza B virus outbreaks also occurred among nursing-home residents (5). The increased circulation of influenza A(H3N2) viruses late in the influenza season was paralleled by increases in P&I-associated mortality and the number of outbreaks reported among nursing homes and other chronic-care facilities, particularly in areas of highest influenza A(H3N2) activity (New England, Mountain, Middle Atlantic, and South Atlantic regions) (6).

FIGURE 6. Percentage of deaths caused by pneumonia or influenza, 121 Cities Surveillance System — United States, 1991–94



1993–94 Influenza Season

Reports from State and Territorial Epidemiologists

State and territorial epidemiologists first reported sustained regional influenza activity during the week ending November 13, 1993 (week 45), followed by widespread activity 4 weeks later. This surveillance component demonstrated peak influenza activity in the United States during the week ending January 8, 1994 (week 1), when 33 states and the District of Columbia reported regional or widespread activity. No state reported widespread activity after the week ending February 26, 1994 (week 8). Regional activity was last reported for the week ending April 9, 1994 (week 14) (Figure 1).

Sentinel Physicians Surveillance Network

The percentage of patient visits to sentinel physicians for ILI rose above the baseline level during the week ending December 18, 1993 (week 50). Patient visits for ILI peaked at 8% during the week ending January 1, 1994 (week 52), and returned to baseline levels during the week ending February 5, 1994 (week 5) (Figure 2). The highest percentages (12%–14%) of office visits to sentinel physicians for ILI occurred in the East North Central, West South Central, and Pacific regions. Persons ≥ 65 years of age accounted for 8% of visits for ILI but 51% of associated hospitalizations.

Sentinel physicians submitted 349 nasal and throat swabs for respiratory virus testing; 92 (26%) were positive for influenza viruses. All positive specimens were influenza type A.

WHO Collaborating Laboratories

From September 12, 1993, through June 11, 1994, the WHO collaborating laboratories tested 36,764 specimens and identified 4,257 (12%) influenza isolates, of which 4,222 (99%) were influenza A and 35 (1%) were influenza B (Figure 4). Of these, 2,913 influenza A isolates were subtyped, and 2,898 (99%) were influenza A(H3N2).

The number of influenza A isolates peaked during the first week of January. Most of the antigenically characterized viruses were similar to the 1993–94 vaccine strain, A/Beijing/32/92, although some viruses were more similar to the antigenic variant A/Shangdong/09/93. Influenza B isolates associated with sporadic ILI cases were recovered throughout the season, with a slight increase in the number in March and April (Figure 5). All antigenically characterized influenza B isolates were similar to the vaccine strain, B/Panama/45/90. No reported isolations of influenza A(H1N1) were confirmed at CDC.

121 Cities Surveillance System

The proportion of deaths attributable to P&I exceeded the epidemic threshold for 10 consecutive weeks, beginning with the week ending December 25, 1993 (week 51), and continuing through the week ending February 26, 1994 (week 8). This was the earliest beginning of sustained excess P&I mortality in the previous 11 years. P&I mortality peaked at 9.2% during the week ending January 22, 1994 (week 3) (Figure 6).

Outbreak Surveillance

During August and early September 1993, the Louisiana Department of Health and Hospitals reported three pre-season outbreaks of ILI associated with influenza type A(H3N2). The first two outbreaks occurred among residents and staff of two nursing homes, and the third affected workers on a dredging barge in southern Louisiana. All three outbreaks were associated with high attack rates (range: 20% among staff of one nursing home and 64% among its residents) (7). Virologic and serologic evidence indicated that all three outbreaks were caused by A/Beijing/32/92(H3N2)-like viruses, which were first isolated in the United States during the 1992–93 season (8).

Outbreaks of ILI among school-age children were reported in early November 1993 in Wyoming and Montana and in mid-November in Idaho and were associated with influenza type A(H3N2) (9). Although most reported outbreaks were in schools, outbreaks occurred among persons in all age groups; high rates of absenteeism among workers were reported commonly during peak influenza activity. Outbreaks also occurred among residents of nursing homes (10).

DISCUSSION

The 1992–93 influenza season was dominated by influenza B, but increasing circulation of influenza A(H3N2) viruses toward the end of the season was associated with outbreaks caused by influenza A(H3N2) viruses in nursing homes. Early in the season, when influenza B viruses predominated, outbreaks were limited mainly to school-age children, and no excess mortality was observed. Sustained excess mortality first began during the week ending March 13, 1993 (week 10), and coincided with an increase in outbreaks among nursing homes and an increase in the number of influenza

A(H3N2) isolates. Since their emergence in 1968–69, influenza A(H3N2) viruses have been associated with excess influenza-associated mortality, particularly among the elderly. Most influenza A(H3N2) viruses isolated during the 1992–93 influenza season were antigenically related to A/Beijing/32/92, which first was detected in the United States in November 1992. This strain replaced A/Beijing/353/89 (the 1992–93 H3N2 influenza vaccine component) in the 1993–94 influenza vaccine. During the summer of 1993, sporadic influenza A(H3N2) activity occurred through June and three outbreaks of influenza A(H3N2) virus were detected in Louisiana during August and September. Influenza A(H3N2) predominated during the 1993–94 season, which began earlier than usual and was moderately severe.

At the end of an influenza season, an increase is often reported in the number of influenza isolates of a type or subtype different from the type/subtype that predominated during that season. Frequently, the emerging influenza virus type/subtype becomes predominant in the following season. For example, influenza B predominated during the 1992–93 season, but toward the end of that season influenza A(H3N2) activity increased. Sporadic cases of influenza A(H3N2) were reported during the summer months of 1993 and outbreaks of this strain occurred during August and September. Influenza A(H3N2) was the predominant virus during the 1993–94 season. Summer outbreaks of influenza sometimes have indicated an earlier than usual influenza season.

The variable epidemiology of influenza, as exemplified by these two seasons, emphasizes the importance of ongoing influenza surveillance to provide essential information to guide public health measures and interventions. Although vaccination against influenza is the most effective method of reducing the impact of influenza, the antiviral agents amantadine and rimantadine provide a useful adjunct for the prevention and treatment of influenza type A infections. Neither drug is effective against influenza type B viruses. Awareness among health-care providers of current influenza activity and circulating strains is necessary for reducing the impact of influenza and related complications.

The 1992–93 influenza vaccine contained A/Texas/36/91(H1N1), A/Beijing/353/89(H3N2), and B/Panama/45/90. For the 1993–94 season, the influenza A(H3N2) component was updated to the A/Beijing/32/92(H3N2) strain, and the influenza A(H1N1) and B components were retained. For 1994–95, the influenza A(H3N2) component in the vaccine was updated to A/Shangdong/09/93, and the influenza A(H1N1) and B components were retained.

References

1. Glezen WP. Serious morbidity and mortality associated with influenza epidemics. *Epidemiol Rev* 1982;4:25–44.
2. Barker WH. Excess pneumonia and influenza associated hospitalization during influenza epidemics in the United States, 1970–78. *Am J Public Health* 1986;76:761–5.
3. CDC. Prevention and control of influenza: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 1996;45(No. RR-5).
4. CDC. Update: influenza activity, United States and worldwide—1992–93 season. *MMWR* 1992;41:939,945.
5. CDC. Update: influenza activity—United States, 1992–93 season. *MMWR* 1993;42:51–3.
6. CDC. Update: influenza activity—United States, 1992–93 season. *MMWR* 1993;42:385–7.
7. CDC. Influenza A outbreaks—Louisiana, August 1993. *MMWR* 1993;42:689–92.
8. CDC. Update: influenza activity—United States and worldwide, and composition of the 1993–94 influenza vaccine. *MMWR* 1993;42:177–80.

9. CDC. Update: influenza activity—United States and Europe, 1993–94 season. MMWR 1993;42:909–11.
10. CDC. Update: influenza activity—United States and worldwide, 1993–94 season, and composition of the 1994–95 influenza vaccine. MMWR 1994;43:179–83.

Surveillance for Silicosis, 1993—Illinois, Michigan, New Jersey, North Carolina, Ohio, Texas, and Wisconsin

Roy Maxfield, M.S.¹

Celan Alo, M.D.¹

Mary Jo Reilly, M.S.²

Kenneth Rosenman, M.D.²

Doug Kalinowski, C.I.H.³

Martha Stanbury, M.S.P.H.⁴

David J. Valiante, C.I.H.⁴

Bill Jones, M.P.H, R.S.⁵

Susan Randolph, M.S.N., R.N., C.O.H.N.⁵

Edward Socie, M.S.⁶

Keith Gromen⁶

Adeline Migliozi, R.N., M.S.N., C.O.H.N.⁶

Teresa M. Willis⁷

Patricia Schnitzer, Ph.D.⁷

Dennis M. Perrotta, Ph.D.⁷

George Gruetzmacher, C.I.H, P.E.⁸

Henry Anderson, M.D.⁸

Ruth Ann Romero Jajosky, D.M.D., M.P.H.⁹

Robert M. Castellan, M.D., M.P.H.⁹

Steven Game, M.P.H.⁹

¹*Illinois Department of Public Health, Springfield, IL*

²*Michigan State University, East Lansing, MI*

³*Michigan Department of Consumer and Industry Services, Lansing, MI*

⁴*New Jersey Department of Health, Trenton, NJ*

⁵*North Carolina Department of Environment, Health and Natural Resources, Raleigh, NC*

⁶*Ohio Department of Health, Columbus, OH*

⁷*Texas Department of Health, Austin, TX*

⁸*Wisconsin Department of Health and Social Services, Madison, WI*

⁹*Division of Respiratory Disease Studies*

National Institute for Occupational Safety and Health (NIOSH), CDC

Abstract

Problem/Condition: Silicosis is an occupational respiratory disease caused by the inhalation of respirable dust containing crystalline silica. Public health surveillance programs to identify workers at risk for silicosis and target workplace-specific and other prevention efforts are currently being field-tested in seven U.S. states.

Reporting Period Covered: Confirmed cases ascertained by state health departments during the period January 1, 1993, through December 31, 1993; the cases and associated workplaces were followed through December 1994.

Description of Systems: As part of the Sentinel Event Notification System for Occupational Risks (SENSOR) program initiated by CDC's National Institute for Occupational Safety and Health (NIOSH), development of state-based surveillance and intervention programs for silicosis was initiated in 1987 in Michigan, New Jersey, Ohio, and Wisconsin and in 1992 in Illinois, North Carolina, and Texas.

Results: From January 1, 1993, through December 2, 1994, the SENSOR silicosis programs in Illinois, Michigan, New Jersey, North Carolina, Ohio, Texas, and Wisconsin confirmed 256 cases of silicosis that were initially ascertained in 1993. Overall, 185 (72%) were initially identified through review of hospital discharge data or through hospital reports of silicosis diagnoses; 188 (73%) were associated with silica exposure in manufacturing industries (e.g., foundries; stone, clay, glass, and concrete manufacturers; and industrial and commercial machinery manufacture). Overall, 42 (16%) cases were associated with silica exposure from sandblasting operations. Among the 193 confirmed cases for which information was available about duration of employment in jobs with potential exposure to silica, 37 (19%) were employed ≤ 10 years in such jobs and 156 (81%) were employed ≥ 11 years.

A total of 192 primary workplaces associated with potentially hazardous silica exposures were identified for the 256 confirmed silicosis cases. Of these, nine (5%) workplaces were inspected by state health department (SHD) industrial hygienists, 19 (10%) were referred to the Occupational Safety and Health Administration (OSHA) for follow-up, and seven (4%) were routinely monitored by the Mine Safety and Health Administration. Of the 157 (82%) remaining workplaces, follow-up activities determined that 82 were no longer in operation, eight were no longer using silica, 18 were assigned a lower priority for follow-up, six were associated with building trades and could not be inspected because of the transient nature of work in the construction industry, and 43 workplaces were not inspected for other reasons. Fourteen (7%) of the 192 workplaces were inspected. At 10 of the 14 workplaces, airborne levels of crystalline silica were measured; in nine, silica levels exceeded the NIOSH-recommended exposure level of 0.05 mg/m³ and in six, airborne silica levels also exceeded federal permissible exposure limits.

Actions Taken: Employee-specific and other preventive interventions have been initiated in response to reported cases. In addition, special silicosis prevention projects have been initiated in Michigan, New Jersey, North Carolina, Ohio, Texas, and Wisconsin. To facilitate the implementation of silicosis surveillance by other states, efforts are ongoing to identify and standardize core data needed by surveillance programs to describe cases and the workplaces where exposure occurred. These core variables will be incorporated into a user-friendly software system that states can use for data collection and reporting.

INTRODUCTION

Silicosis is an occupational lung disease that develops after dust containing respirable crystalline silica is inhaled and deposited in the lungs. It is both progressive and incurable, yet preventable. In response to high concentrations of crystalline silica,

disease can develop within a year after initial exposure. This acute form of the disease is associated with the accumulation of a proteinaceous fluid in the alveolar spaces of the lungs; it typically causes death within months of initial diagnosis. More commonly, chronic silicosis occurs after ≥ 10 years of relatively low, but still hazardous, exposure to crystalline silica. Chronic silicosis manifests as scarring of the lung tissue; it can be debilitating and cause death. In addition, silicosis is a risk factor for tuberculosis (1). The 1987 International Agency for Research on Cancer (IARC) Monograph on the Evaluation of Carcinogenic Risks to Humans classified crystalline silica as a probable human carcinogen (2). In October 1996, IARC reconvened a Monograph Working Group to reevaluate the carcinogenicity of silica, some silicates, and organic fibers. The Monograph Group concluded that inhaled crystalline silica in the form of quartz or cristobalite from occupational sources is carcinogenic to humans (IARC, unpublished data). In view of this assessment, persons occupationally exposed to crystalline silica in one or more of these two forms, regardless of whether they have silicosis, should be considered at potentially increased risk for lung cancer.

In 1987, CDC's National Institute for Occupational Safety and Health (NIOSH) awarded 5-year cooperative agreements to four state health departments (SHDs) (Michigan, New Jersey, Ohio, and Wisconsin) to develop and implement silicosis surveillance and preventive intervention projects under the Sentinel Event Notification Systems for Occupational Risks (SENSOR) program (3). The Michigan and New Jersey SENSOR projects were modeled after another NIOSH-supported cooperative agreement that began in 1984.* In an attempt to build on the experience gained by the states during the first 5-year phase of SENSOR, in 1992 these four states were granted additional 5-year cooperative agreements to continue refining and field-testing their approaches to silicosis surveillance and intervention. In 1992, three other states (Illinois, North Carolina, and Texas) were awarded SENSOR cooperative agreements to establish similar silicosis projects. This report summarizes data for confirmed silicosis cases ascertained during 1993 from the silicosis surveillance and intervention programs in all seven states.

METHODS

Surveillance

Case Report Ascertainment

All seven states with SENSOR silicosis programs actively solicit case reports from physicians likely to evaluate patients with silicosis, such as pulmonary and occupational medicine physicians and B-Readers (physicians trained and certified in the use of the International Labour Office system for classification of radiographs for pneumoconioses) (4,5).

Before 1993, physicians were required to report silicosis cases to SHDs in Michigan, New Jersey, Ohio, Texas, and Wisconsin (Table 1). Physicians were not required to report silicosis diagnoses in North Carolina until January 1, 1994, and in Illinois,

*NIOSH supported a cooperative agreement program for capacity building in occupational safety and health for state, territorial, and local health departments before the inception of the SENSOR program.

physician reporting of silicosis cases continues to be voluntary. The states with SENSOR silicosis programs actively solicit silicosis case reports from mandated and voluntary reporters (Table 1). North Carolina is the only state that did not begin to solicit case reports actively from physicians until after the end of the 1993 reporting period.

Although the original SENSOR model was based primarily on physician reporting (6), other data sources are utilized in each state for ascertaining silicosis cases. All seven SHDs review death certificate data. In Illinois, Michigan, and New Jersey, computerized hospital discharge data are reviewed to identify silicosis diagnoses. In addition, hospitals in Michigan and New Jersey are required to report hospital discharge diagnoses of silicosis directly to their SHDs. Although surveillance staff in Texas and Ohio do not have access to computerized hospital discharge data, staff in Texas review medical records of selected hospitals for diagnoses of silicosis, and, in 1993, Ohio began active solicitation of silicosis diagnosis reporting from hospitals (7). In addition, in Ohio and Michigan reports of workers' compensation claims are reviewed to identify potential cases. One source of data unique to North Carolina is the North Carolina Dusty Trades Program, a long-standing medical screening program, supported by the state health department, for workers exposed to silica and asbestos. Finally, in Michigan, New Jersey, and Ohio, cases have also been identified through selected surveys targeting silica-exposed coworkers of index cases.

Case Confirmation

In all seven states, demographic, work history, and medical information is collected about each reported silicosis case. This information is obtained through a combination of the initial case ascertainment source, a review of medical records, and follow-up telephone interviews with persons with cases or their next of kin. For SENSOR surveillance purposes, silicosis case confirmation requires a history of

TABLE 1. Year silicosis became reportable, mandated reporters, and year active solicitation efforts were initiated, by state, 1978–1994*

State	Year required to report	Mandated reporters	Year Reports Solicited: Sources of Reports
Illinois	NR	NA	1993: Physicians, hospitals
Michigan	1978	Physicians, hospitals, clinics, employers	1988: Physicians, hospitals, clinics, employers
New Jersey	1985: Hospitals 1990: Physicians	Hospitals, physicians	1985: Hospitals 1988: Physicians
North Carolina	1994	Physicians	1994: Physicians, hospitals
Ohio	1990	Physicians	1989: Physicians 1993: Hospitals
Texas	1985	Physicians	1992: Physicians
Wisconsin	—	Physicians	1988: Physicians

NR = silicosis not required to be reported

NA = category not applicable

—Specific year unknown, but before 1987

* Active solicitation efforts are facilitated through NIOSH-funded SENSOR programs, which began in 1987, or through capacity-building cooperative agreements, which were initiated in 1984 and were the model for the SENSOR program.

occupational exposure to airborne silica dust and a) a chest radiograph interpreted as characteristic of silicosis and/or b) lung histopathology characteristic of silicosis. These criteria have been modified from the previously published surveillance case definition for silicosis, which did not cite a requirement for a history of occupational exposure to airborne silica when the lung pathology criterion was used for case confirmation (3,8). To enhance the specificity of silicosis surveillance, some states also apply exclusion criteria for persons with occupational work histories of coal mining,* because coal workers' pneumoconiosis and silicosis have many clinical features in common.

The primary workplace associated with silica exposure for each worker is the job that is considered by surveillance staff as most likely responsible for the longest or highest potential exposure to silica. This assessment is based on a review of the detailed work history solicited for each case, combined with the surveillance staff's knowledge of high-risk industries, occupations, and work processes. Surveillance staff attempt to locate and contact all workplaces, both primary and secondary, that are potentially associated with silica exposure to collect information about current work practices and activities that may involve exposure to silica. This information, combined with information regarding nature of the disease (e.g., age of patient, severity of disease, number of years exposed) and number of cases reported in association with a specific workplace, assists surveillance staff in prioritizing workplace inspections by SHD industrial hygienists and in making appropriate referrals to consultative or enforcement agencies.

Preventive Intervention

Prevention efforts differ among the states, but generally include one or more of the following activities: a) follow-up interviews with persons with reported and/or confirmed silicosis or their next of kin; b) follow-up investigations to determine whether the workplace identified by the person with silicosis or his/her next of kin is currently active and engaged in work processes that have the potential for continuing hazardous silica exposure; c) educational outreach to persons with cases and their coworkers, employers, physicians, and unions; d) workplace inspections by SHD industrial hygienists or referrals to consultation programs such as that of the Occupational Safety and Health Administration (OSHA) to assess current silica exposure and to provide workplace-specific recommendations for reducing or eliminating hazardous silica exposure; and e) referrals to appropriate enforcement agencies—OSHA or the Mine Safety and Health Administration (MSHA)—if information obtained during the workplace follow-up indicates excessive exposures and/or hazardous work practices.

*Cases ordinarily meeting the silicosis surveillance case definition are excluded on the basis of a work history indicating >10 years of tenure in coal mining and <3 years of tenure in a silica-using industry. The coal mining exclusion criterion does not apply in Ohio if a coal mining occupation with high potential for silica exposure (e.g., roof bolter, driller, or motorman) is reported. North Carolina does not plan to apply coal mining exclusion criteria.

RESULTS

Epidemiology

The SENSOR silicosis programs in Illinois, Michigan, New Jersey, North Carolina, Ohio, Texas, and Wisconsin confirmed 256 cases of silicosis that were initially ascertained in 1993. Of these, 245 (96%) were male and 11 (4%) were female; 189 (74%) were white, 62 (24%) were black, three (1%) were of other races, and the race of two (0.8%) persons was unknown (Table 2). The race and sex distribution of cases is presented here because past differential hiring practices have put minority workers at greater risk than white workers of being placed in jobs with a high potential risk of exposure to airborne crystalline silica (9). Consequently, if adequate dust control measures are not properly employed, these workers are placed at greater risk for silicosis.*

The year of birth ranged from 1898 to 1968 (median=1924). By state, the median year of birth ranged from 1907 (n=2; range: 1905–1909) in North Carolina to 1943 (n=42; range: 1908–1968) in Texas.

Overall, 185 (72%) confirmed cases were initially identified through review of hospital discharge data or through hospital reports of silicosis diagnoses, 37 (15%) through physician reports, 26 (10%) through review of death certificate data, and eight (3%) through a combination of workers' compensation claims reports, other health-care providers, and referrals from other SHDs (Table 3).

Silica exposure in manufacturing industries accounted for 73% of the 256 confirmed cases (Table 4). The primary metal industries were associated with silica exposure in 86% of the cases in Wisconsin, 74% of the cases in Michigan, 46% of the cases in Ohio, and 30% of the cases in Illinois. Industries manufacturing stone, clay, glass, and concrete products accounted for 29% of the cases in New Jersey. In Texas, 21% of the cases were associated with the industrial and commercial machinery and computer equipment manufacturing industries.

Overall, 37 (14%) of the persons with cases had ≤ 10 years of employment in industries with potential for silica exposure, 89 (35%) had 11–30 years, 67 (26%) had >30 years, and for 63 (25%) this information is unavailable. The year of first reported exposure to airborne silica dust in an occupational setting ranged from 1917 to 1990 (median=1950).

Workplace Follow-up

Overall, 192 primary workplaces associated with potentially hazardous silica exposures were identified for the 256 confirmed silicosis cases (Table 5). Of these, nine

*This differential risk for silicosis is exemplified by the history of the construction of the Gauley Bridge Tunnel in southern West Virginia during 1930–31. Congressional reports revealed that 476 workers died of silicosis during the construction of the tunnel, and another 1,500 developed silicosis within a few years after the tunnel project was completed. The silica content of the rock drilled for the tunnel was high (up to 99.44% silica). Labor force figures indicate that of the nearly 5,000 men who worked on the tunnel project, more than half performed some work underground (i.e., inside the tunnel). Underground work, which included such jobs as rock drilling and mucking (removing pulverized rock left from drilling), placed a worker at high risk for silicosis. Black workers were largely unskilled, worked primarily as laborers, and were more likely than white workers to have worked underground. Although 66% of the labor force for the tunnel was made up of black males, this group composed 80% of the underground workers.

TABLE 2. Distribution of confirmed silicosis cases, by race, sex, and state — 1993

Race/Sex	IL*		MI*		NJ*		NC†§		OH*†		TX†		WI†		Total	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
White male	22	(73.3)	23	(46.0)	22	(78.6)	2	(100.0)	73	(75.3)	35	(83.3)	4	(57.1)	181	(70.7)
White female	—	(0.0)	1	(2.0)	1	(3.6)	—	(0.0)	4	(4.1)	1	(2.4)	1	(14.3)	8	(3.1)
Black male	7	(23.3)	24	(48.0)	3	(10.7)	—	(0.0)	18	(18.6)	6	(14.3)	1	(14.3)	59	(23.0)
Black female	—	(0.0)	—	(0.0)	2	(7.1)	—	(0.0)	1	(1.0)	—	(0.0)	—	(0.0)	3	(1.2)
Other race																
Male	1	(3.3)	2	(4.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	3	(1.2)
Female	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)
Unknown race																
Male	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	1	(1.0)	—	(0.0)	1	(14.3)	2	(0.8)
Female	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)
Total	30	(100.0)	50	(100.0)	28	(100.0)	2	(100.0)	97	(100.0)	42	(100.0)	7	(100.0)	256	(100.0)

* Solicits reports of silicosis diagnoses from hospitals.

† Does not have access to computerized hospital discharge data.

§ Did not solicit case reports from physicians and hospitals until 1994.

TABLE 3. Initial case ascertainment sources for confirmed silicosis cases reported to state health departments in 1993, by state and case identification source year*

Source/ Source year	IL [†]		MI [†]		NJ [†]		NC ^{§¶}		OH ^{†§}		TX [§]		WI [§]		Total	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Hospital discharges																
1976–1986	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	11	(11.3)	—	(0.0)	—	(0.0)	11	(4.3)
1987	3	(10.0)	—	(0.0)	—	(0.0)	—	(0.0)	2	(2.1)	—	(0.0)	—	(0.0)	5	(2.0)
1988	2	(6.7)	—	(0.0)	—	(0.0)	—	(0.0)	2	(2.1)	—	(0.0)	—	(0.0)	4	(1.6)
1989	5	(16.7)	2	(4.0)	2	(7.1)	—	(0.0)	2	(2.1)	—	(0.0)	—	(0.0)	11	(4.3)
1990	7	(23.3)	3	(6.0)	3	(10.7)	—	(0.0)	4	(4.1)	—	(0.0)	—	(0.0)	17	(6.6)
1991	4	(13.3)	4	(8.0)	3	(10.7)	—	(0.0)	33	(34.0)	—	(0.0)	—	(0.0)	44	(17.2)
1992	—	(0.0)	28	(56.0)	6	(21.4)	—	(0.0)	33	(34.0)	3	(7.1)	—	(0.0)	70	(27.3)
1993	2	(6.7)	—	(0.0)	9	(32.1)	—	(0.0)	6	(6.2)	6	(14.3)	—	(0.0)	23	(9.0)
Subtotal	23	(76.7)	37	(74.0)	23	(82.1)	—	(0.0)	93	(95.9)	9	(21.4)	—	(0.0)	185	(72.3)
Death certificates																
1988	1	(3.3)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	1	(0.4)
1989	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)
1990	1	(3.3)	—	(0.0)	1	(3.6)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	2	(0.8)
1991	1	(3.3)	—	(0.0)	1	(3.6)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	2	(0.8)
1992	1	(3.3)	5	(10.0)	1	(3.6)	—	(0.0)	—	(0.0)	4	(9.5)	—	(0.0)	11	(4.3)
1993	1	(3.3)	—	(0.0)	2	(7.1)	2	(100.0)	—	(0.0)	5	(11.9)	—	(0.0)	10	(3.9)
Subtotal	5	(16.6)	5	(10.0)	5	(17.9)	2	(100.0)	—	(0.0)	9	(21.4)	—	(0.0)	26	(10.2)
Physician reports																
1992	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	3	(42.9)	3	(1.2)
1993	—	(0.0)	5	(10.0)	—	(0.0)	—	(0.0)	2	(2.1)	23	(54.8)	4	(57.1)	34	(13.3)
Subtotal	—	(0.0)	5	(10.0)	—	(0.0)	—	(0.0)	2	(2.1)	23	(54.8)	7	(100.0)	37	(14.5)
Workers' Compensation																
1988	—	(0.0)	1	(2.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	1	(0.4)
1992	—	(0.0)	2	(4.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	2	(0.8)
Subtotal	—	(0.0)	3	(6.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	3	(1.2)
Other health-care provider																
1993	2	(6.7)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	1	(2.4)	—	(0.0)	3	(1.2)
SHD referrals																
1993	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	2	(2.1)	—	(0.0)	—	(0.0)	2	(0.8)
Total	30	(100.0)	50	(100.0)	28	(100.0)	2	(100.0)	97	(100.0)	42	(100.0)	7	(100.0)	256	(100.0)

* Source year represents the year of hospital discharge, death, diagnosis by health care reporter, or year of workers' compensation claim for silicosis.

† Solicits reports of silicosis diagnoses from hospitals.

§ Does not have access to computerized hospital discharge data.

¶ Did not solicit case reports from physicians and hospitals until 1994.

TABLE 4. Primary industry reported as source of silica exposure for silicosis cases, by state, 1993

Industry (SIC* code)	IL		MI		NJ		NC		OH		TX		WI		Total	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Manufacturing																
Primary metals (33)	9	(30.0)	37	(74.0)	6	(21.4)	—	(0.0)	45	(46.4)	—	(0.0)	6	(85.7)	103	(40.2)
Stone, clay, glass and concrete products (32)	3	(10.0)	2	(4.0)	8	(28.6)	1	(50.0)	19	(19.6)	—	(0.0)	1	(14.3)	34	(13.3)
Industrial and commercial machinery and computer equipment (35)	1	(3.3)	1	(2.0)	1	(3.6)	—	(0.0)	5	(5.2)	9	(21.4)	—	(0.0)	17	(6.6)
Fabricated metal products, except machinery and transportation equipment (34)	3	(10.0)	—	(0.0)	1	(3.6)	—	(0.0)	7	(7.2)	2	(4.8)	—	(0.0)	13	(5.1)
Other Manufacturing (28,30,36,37)	3	(10.0)	2	(4.0)	4	(14.3)	—	(0.0)	5	(5.2)	7	(16.7)	—	(0.0)	21	(8.2)
Subtotal Manufacturing	19	(63.3)	42	(84.0)	20	(71.4)	1	(50.0)	81	(83.5)	18	(42.9)	7	(100.0)	188	(73.4)
Construction																
Construction, special trade contractors (17)	2	(6.7)	3	(6.0)	1	(3.6)	—	(0.0)	7	(7.2)	3	(7.1)	—	(0.0)	16	(6.3)
Other Construction (15,16)	—	(0.0)	2	(4.0)	2	(7.1)	—	(0.0)	—	(0.0)	4	(9.5)	—	(0.0)	8	(3.1)
Subtotal Construction	2	(6.7)	5	(10.0)	3	(10.7)	—	(0.0)	7	(7.2)	7	(16.7)	—	(0.0)	24	(9.4)
Mining																
Mining and quarrying of nonmetallic minerals (14)	4	(13.3)	—	(0.0)	3	(10.7)	1	(50.0)	6	(6.2)	—	(0.0)	—	(0.0)	14	(5.5)
Other Mining (10,13)	1	(3.3)	2	(4.0)	1	(3.6)	—	(0.0)	—	(0.0)	1	(2.4)	—	(0.0)	5	(2.0)
Subtotal Mining	5	(16.6)	2	(4.0)	4	(14.3)	1	(50.0)	6	(6.2)	1	(2.4)	—	(0.0)	19	(7.5)
Other Industries (40,47,59,76,80)	4	(13.3)	—	(0.0)	1	(3.6)	—	(0.0)	—	(0.0)	—	(0.0)	—	(0.0)	5	(2.0)
Unknown Industries	—	(0.0)	1	(2.0)	—	(0.0)	—	(0.0)	3	(3.1)	16	(38.1)	—	(0.0)	20	(7.8)
Total	30	(100.0)	50	(100.0)	28	(100.0)	2	(100.0)	97	(100.0)	42	(100.0)	7	(100.0)	256	(100.0)

* 1987 Standard Industrial Classification

TABLE 5. State health department follow-up of workplaces identified as being associated with silica exposure for 256 silicosis cases ascertained in 1993, by state*

Number of primary workplaces	IL		MI [†]		NJ		NC		OH		TX		WI		Total	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
No longer in operation	15	(50.0)	18	(54.5)	14	(58.3)	1	(50.0)	33	(45.2)	—	—	1	(16.7)	82	(42.7)
No longer using silica	2	(6.7)	—	—	4	(16.7)	—	—	2	(2.7)	—	—	—	—	8	(4.2)
Inspected by SHD	1	(3.3)	—	—	3	(12.5)	1	(50.0)	4	(5.5)	—	—	—	—	9	(4.7)
Referred to OSHA	—	—	5	(15.2)	—	—	—	—	2	(2.7)	7	(29.2)	5	(83.3)	19	(9.9)
Routinely inspected by MSHA	2	(6.7)	1	(3.0)	—	—	—	—	4	(5.5)	—	—	—	—	7	(3.6)
Assigned low priority for follow-up	10	(33.3)	—	—	—	—	—	—	8	(11.0)	—	—	—	—	18	(9.4)
Transient building trades workplace	—	—	5	(15.2)	—	—	—	—	1	(1.4)	—	—	—	—	6	(3.1)
Other [§]	—	—	4	(12.1)	3	(12.5)	—	—	19	(26.0)	17	(70.8)	—	—	43	(22.4)
Total	30	(100.0)	33	(100.0)	24	(100.0)	2	(100.0)	73	(100.0)	24	(100.0)	6	(100.0)	192	(100.0)

* Provisional data as of December 1994.

[†] Michigan OSHA is organizationally located within the Michigan Department of Consumer and Industry Services, formerly the Michigan Department of Health.

[§] For Michigan, this category includes two unspecified employers and two out-of-state workplaces.

For New Jersey, this category includes three out-of-state workplaces.

For Ohio, this category includes four workplaces to which entry was denied, five workplaces that SHD staff were unable to locate, and 10 workplaces for which follow-up is pending.

For Texas, this category includes 17 workplaces identified through review of hospital medical records or death certificates, and confirmation of workplace-specific information is incomplete.

—Indicates no workplaces in the category.

SHD = State health department.

OSHA = Occupational Safety and Health Administration.

MSHA = Mine Safety and Health Administration.

(5%) workplaces were inspected by SHD industrial hygienists, 19 (10%) were referred to OSHA for follow-up, and seven (4%) had routine monitoring by MSHA. Of the 157 (82%) remaining workplaces, follow-up activities determined that 82 workplaces were no longer in operation, eight were in operation but no longer using silica, 18 were assigned a lower priority for follow-up,* six were associated with building trades and could not be inspected because of the transient nature of work in the construction industry, and 43 workplaces were not inspected by SHD staff for other reasons (e.g., workplace-specific information was unavailable or had not been confirmed, workplaces could not be located or were located out of state, SHD staff were denied entry into the workplaces, or follow-up was pending).

Workplace inspections were conducted at 14 (7%) of the 192 workplaces. These 14 workplaces include nine workplaces inspected by SHD industrial hygienists and five workplaces (all foundries) that the Michigan OSHA[†] program inspected. The workplaces represented by the nine SHD inspections include a foundry in Illinois; a foundry, a sand mine, and a sanitary ware (i.e., vitreous china plumbing fixtures [SIC code 3261]) manufacturer in New Jersey; three foundries and a quarry in Ohio; and a quarry in North Carolina. At 10 of the 14 workplaces, airborne levels of crystalline silica were measured; in nine (90%) of these workplaces the silica levels exceeded the NIOSH-recommended exposure level (REL),[§] and in six (60%) workplaces airborne silica levels also exceeded federal permissible exposure limits (PEL)^{¶**} (Table 6).

DISCUSSION

Data aggregation for this report was based on the year a person with a confirmed case of silicosis was ascertained by the SHD SENSOR program and therefore depends on timing of access to data sources and intensity and completeness of surveillance and follow-up efforts. This method of data aggregation differs from the data aggregation methods used in previously published summary reports of SENSOR silicosis data (3,12,13). In previously published reports, data have been aggregated according to the source year—the year of a silicosis-related hospital discharge, death, year of a workers' compensation claim, or physician report. The method of data aggregation for this report was changed to facilitate administrative tracking of reported cases. The

*Criteria for prioritization of workplace follow-up include age of person with the case, disease severity, number of years employed at workplace with potential for exposure, and number of cases associated with workplace.

[†]The Michigan OSHA program is organizationally located within the Michigan Department of Health (recently renamed the Michigan Department of Consumer and Industry Services); therefore, the results of Michigan OSHA inspections are readily available to the Michigan SENSOR program.

[§]The NIOSH REL for respirable crystalline silica is 0.05 mg/m³ (or 50 µg/m³) as a time-weighted average (TWA) for up to 10 hours per day during a 40-hour work week. This REL is intended to prevent silicosis (10).

[¶]The current OSHA PEL as an 8-hour TWA for crystalline silica (as respirable quartz) is either 10 mg/m³ divided by the value "% silicon dioxide + 2" for general industry [29 Code of Federal Regulations (CFR) 1910.1000] or 250 million particles per cubic foot divided by the value "% silicon dioxide + 5" for construction [29 CFR 1926.55] and shipyard employment [29 CFR 1915.5] (11).

**For metal and nonmetal surface and underground mines, the MSHA PEL (in mg/m³) for respirable dust (30 CFR 56.500) is calculated by dividing 10 by the quantity "% crystalline silica + 2" (11).

TABLE 6. Distribution of 14 silica-related workplace inspections conducted by state health departments and Michigan OSHA and results of airborne silica level measurements, by state*†

Number of primary workplaces	IL		MI		NJ		NC		OH		TX		WI		Total	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Inspected	1	(3.3)	5	(15.2)	3	(12.5)	1	(50.0)	4	(5.5)	—	—	—	—	14	(7.3)
Inspected workplaces where																
Airborne silica levels measured	—		5		3		1		1		—		—		10	
Silica levels exceeded REL	—		5		3		NA		1		—		—		9	
Silica levels exceeded PEL	—		3		2		NA		1		—		—		6	

* Provisional data as of December 1994.

† The Michigan OSHA program is organizationally located within the Michigan Department of Health. Michigan OSHA inspection reports are readily available to the Michigan SENSOR program.

—Indicates no workplaces in this category.

NA = airborne silica measurements taken at this workplace did not exceed the federal permissible exposure limits or the NIOSH recommended exposure limit for respirable crystalline silica.

OSHA = Occupational Safety and Health Administration.

summary results in this report are therefore not directly comparable with those of previously published summary reports and should not be used to calculate silicosis incidence rates.

The methods for silicosis surveillance initially developed by Michigan, New Jersey, Ohio, and Wisconsin are being adopted by Illinois, North Carolina, and Texas. Overall findings demonstrate the importance of hospital discharge diagnoses for identification of silicosis cases. Only the Illinois, Michigan, and New Jersey SHDs have routine access to computerized hospital discharge data for silicosis case identification. Ohio and North Carolina, however, began to actively solicit hospital reports of silicosis diagnoses in 1993 and 1994, respectively. Although Wisconsin does not yet have access to hospital discharge data for 1993, surveillance staff in Wisconsin expect to review computerized hospital discharge tapes by 1997 for silicosis case identification. Although hospital discharge data are an important case ascertainment source, other case identification sources need to be developed, because many patients with silicosis may not be hospitalized for silicosis or may not mention a previous diagnosis of silicosis if hospitalized for another condition.

The year of initial silicosis diagnosis, the optimal year to use for silicosis incidence rate calculations, is not usually known by SHD surveillance staff. As an alternative, the Michigan SENSOR program has used the source year (previously defined) as the reference year for silicosis incidence rate calculations, since the source year represents the first time the surveillance program staff became aware of the silicosis diagnosis. Using the source year, the Michigan SENSOR program reports that, among counties where average annual silicosis incidence rates were compared for black and white workers, black workers had consistently higher incidence rates than white workers because of past differential hiring practices that increased this group's risk of exposure (see *Epidemiology*) (14). In Michigan, the overall average annual incidence rate for black males (14.3 cases per 100,000) ages ≥ 40 years was seven times higher than for white workers (2.1 cases per 100,000) in the same age group (denominator data from the 1990 U.S. census for men ages ≥ 40). Silicosis count data in Michigan indicate that the number of black males with silicosis is greater than that of silicosis cases in all other race and sex groups, reflecting past hiring practices in foundries, where black males were more likely to be given dustier jobs with increased likelihood of exposure to silica (14). Knowledge about high-risk race and sex groups can be useful for targeting public health prevention programs. For example, the Michigan SENSOR program has recommended that select counties (those with high silicosis incidence rates and numbers of cases) target tuberculosis screening tests for black males, because this group is at the highest risk for silicosis and because silicosis increases one's susceptibility to tuberculosis.

The word "sandblaster" was listed in the occupation narratives of 42 (16%) persons with confirmed cases of silicosis ascertained in 1993. At least one of these persons was first occupationally exposed to silica sand in abrasive blasting operations as late as 1983, 9 years after NIOSH recommended that materials containing >1% crystalline silica be prohibited as abrasive blasting materials and that less hazardous materials be used (10). Because of the continuing occurrence of severe silicosis associated with sandblasting, NIOSH published an Alert in 1992 reiterating this recommendation (15). Data were not available at the time of data aggregation regarding whether an individual had ever conducted sandblasting activities in the workplace and/or whether an

individual had ever worked in an uncontrolled plume of dust generated from sandblasting; therefore, other SENSOR silicosis cases ascertained in 1993 may be related to exposures generated during sandblasting activities. The risk of silicosis is high in workers exposed to sandblasting operations, and the quantity of dust generated by these operations is often difficult to control. Sandblasting also produces dust containing freshly fractured surfaces of crystalline silica, which appears to represent a greater hazard than crystalline silica dust characterized predominantly by aged cleavage surfaces (16).

In 1993, three states with SENSOR silicosis programs—Michigan, New Jersey, and Ohio—developed special silicosis prevention projects to identify workplaces where abrasive blasting activities are conducted and to target these workplaces for intervention activities, including workplace surveys, industrial hygiene inspections, and distribution of educational literature regarding health hazards of silica exposure and practices to prevent silica exposure. With information gathered to date by NIOSH and New Jersey regarding silica sand substitutes, the New Jersey SHD prepared a fact sheet for employers aimed at promoting the use of alternatives to silica sand in abrasive blasting operations. The fact sheet includes information about the properties, equipment needs, specific applications, advantages, limitations, and cost of the major silica sand substitutes (aluminum oxide, baking soda, coal slag, corn cob granules, dry ice, garnet, glass beads, nickel slag, nut shells, olivine, plastic media, staurolite, and steel grit/shot). In addition, the fact sheet underscores the need for continued use of appropriate measures to control exposure, because the health effects of the substitutes have not yet been fully determined and because the substrate material being removed by abrasive blasting can contain silica or other hazardous materials (e.g., lead). The fact sheet has been shared with other states with SENSOR silicosis programs and has been distributed to 1,969 companies (1,795 in Michigan, 133 in New Jersey, and 41 in Ohio) identified as having a potential to conduct sandblasting operations.

A component of the silicosis prevention project in Michigan included evaluating whether the recommendations issued during initial SENSOR-prompted workplace inspections for silica exposure hazards had been implemented or had led to reduced airborne silica levels. To assess whether airborne silica levels had been reduced, industrial hygienists from Michigan OSHA reinspected 10 companies where airborne silica levels exceeded the OSHA PEL during the initial SENSOR-prompted inspections. Air sampling conducted during the reinspections showed substantially lower airborne silica levels—eight of the 10 workplaces (six foundries, a pottery, and a fabricated metal products manufacturer) had levels below the OSHA PEL for respirable crystalline silica. In the other two workplaces (both foundries), employees in jobs with high silica exposure were using supplied-air respirators or air-purifying respirators with high-efficiency particulate filters (17). The Michigan Department of Health (recently renamed the Michigan Department of Consumer and Industry Services) also sent letters to 10 additional companies who were not in violation of the OSHA PEL for respirable crystalline silica, inquiring whether they had followed the workplace-specific prevention recommendations made as a result of initial SENSOR-prompted inspections for silica. The companies receiving these inquiries included nine foundries and an abrasive products manufacturer. Results of the survey, reported elsewhere, indicate that most of the recommendations had been implemented (17). Although Michigan did not

validate the accuracy of this self-reported information, these results, together with the results of the reinspections, generally indicate that the prevention recommendations made by the Michigan SENSOR program are prompting silica hazard reduction.

Projects for SENSOR silicosis intervention activities also were developed by North Carolina, Texas, and Wisconsin. Although the activities and/or final reports from these projects are still in process, the primary focus of the activities in North Carolina and Wisconsin relates to assessing respirable silica exposures during road construction and maintenance activities. In Wisconsin, the primary activities of interest involve concrete and asphalt cutting operations; in North Carolina, the primary activities of interest involve rock drilling during road construction and abrasive blasting operations for bridge cleaning and maintenance. The Texas project involves a survey of companies engaged in the manufacture of cut stone and stone products to assess their potential for silica exposure, to determine the type of medical screening procedures implemented, and to identify opportunities for early case identification and silicosis prevention.

To facilitate the implementation of silicosis surveillance by additional states, efforts are in progress to refine and standardize core surveillance program elements needed to describe cases and their workplaces. Plans include the incorporation of core silicosis variables into a user-friendly software system, which can be used for data collection and reporting at the state level and for data aggregation across the states.

References

1. Cowie RL. The epidemiology of tuberculosis in gold miners with silicosis. *Am J Respir Crit Care Med* 1994;150:1460-2.
2. International Agency for Research on Cancer (1987). Overall evaluation of carcinogenicity: an updating of IARC Monographs Volumes 1-42 1987. Lyon: WHO, Supplement 7:341-3.
3. CDC. Silicosis Surveillance—Michigan, New Jersey, Ohio, and Wisconsin, 1987-1990. *MMWR* 1993; 42(No. SS-5):23-8.
4. Wagner GR, Attfield MD, Kennedy RD, Parker JE. The NIOSH B-reader certification program: an update report. *J Occup Med* 1992;34:879-84.
5. 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).
6. Baker EL. SENSOR: the concept. *Am J Public Health* 1989; 79 (suppl):18-20.
7. CDC. Occupational silicosis—Ohio, 1989-1994. *MMWR* 1995;44(4):61-4.
8. CDC. Silicosis: cluster in sandblasters—Texas, and occupational surveillance for silicosis. *MMWR* 1990;39:433-7.
9. Cherniac M. The Hawk's Nest incident: America's worst industrial disaster. Yale University Press: New Haven, 1986.
10. NIOSH. Criteria for a recommended standard: occupational exposure to crystalline silica. US Department of Health, Education, and Welfare, Public Health Service, CDC, 1974; HEW publication no. (NIOSH) 75-120.
11. CFR. Code of Federal Regulations. Washington, DC: US Government Printing Office, Office of the Federal Register.
12. NIOSH. Work-related lung disease surveillance report, 1994. US Department of Health and Human Services, Public Health Service, CDC, 1994; DHHS(NIOSH) publication no. 94-120.
13. NIOSH. Work-related lung disease surveillance report: supplement 1992. US Department of Health and Human Services, Public Health Service, CDC, 1992; DHHS(NIOSH) publication no. 91-113S.
14. Rosenman KD, Reilly MJ, Watt FC. 1995 Annual report on silicosis in Michigan. Lansing: Michigan Department of Health; 1995:6-7.
15. NIOSH. Request for assistance in preventing silicosis and deaths from sandblasting. Cincinnati, Ohio: 1992 DHHS(NIOSH) publication no. 92-102.

16. Castranova V. Generation of oxygen radicals and mechanism of injury prevention. *Environ Health Perspect* 1994;102:Suppl 10:65-8.
17. Rosenman KD, Reilly MJ, Watt FC. 1994 Annual report on silicosis in Michigan. Lansing: Michigan Department of Health; 1994: 24-5.

State and Territorial Epidemiologists and Laboratory Directors

State and Territorial Epidemiologists and Laboratory Directors are acknowledged for their contributions to *CDC Surveillance Summaries*. The epidemiologists listed below were in the positions shown as of December 1996, and the laboratory directors listed below were in the positions shown as of December 1996.

State/Territory	Epidemiologist	Laboratory Director
Alabama	John P. Lofgren, MD	William J. Callan, PhD
Alaska	John P. Middaugh, MD	Gregory V. Hayes, DrPH
Arizona	Robert W. England, Jr., MD, MPH	Barbara J. Erickson, PhD
Arkansas	Thomas C. McChesney, DVM	Michael G. Foreman
California	Stephen H. Waterman, MD, MPH	Michael G. Volz, PhD
Colorado	Richard E. Hoffman, MD, MPH	Robert Quillan, MS, MSPH
Connecticut	James L. Hadler, MD, MPH	Sanders F. Hawkins, PhD
Delaware	A. LeRoy Hathcock, PhD	Chris Zimmerman, MA (Acting)
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
Hawaii	Richard L. Vogt, MD	Vernon K. Miyamoto, PhD
Idaho	Jesse F. Greenblatt, MD, MPH	Richard H. Hudson, PhD
Illinois	Byron J. Francis, MD, MPH	David F. Carpenter, PhD
Indiana	Gregory K. Steele, DrPH, MPH	David E. Nauth (Acting)
Iowa	M. Patricia Quinlisk, MD, MPH	Mary J. R. Gilchrist, PhD
Kansas	Gianfranco Pezzino, MD, MPH	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, MPH	Philip W. Haines, DrPH
Maryland	Diane M. Dwyer, 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	Joe O. Graves, PhD
Missouri	H. Denny Donnell, Jr, MD, MPH	Eric C. Blank, DrPH
Montana	Todd A. Damrow, PhD, MPH	Douglas O. Abbott, PhD
Nebraska	Thomas J. Safranek, MD	John D. Blosser
Nevada	Randall L. Todd, DrPH	Arthur F. DiSalvo, MD
New Hampshire	Vacant	Veronica C. Malmberg, MSN
New Jersey	Faye Sorhage, DVM, MPH (Acting)	Marion Pierce
New Mexico	C. Mack Sewell, DrPH, MS	Loris W. Hughes, PhD
New York City	Benjamin A. Mojica, MD, MPH	Alex Ramon, MD, MPH
New York State	Perry F. Smith, MD	Lawrence S. Sturman, MD, PhD
North Carolina	J. Newton MacCormack, MD, MPH	Lou F. Turner, DrPH
North Dakota	Larry A. Shireley, MS, MPH	James D. Anders, MPH
Ohio	Thomas J. Halpin, MD, MPH	Leona Ayers, MD (Acting)
Oklahoma	J. Michael Crutcher, MD, MPH (Acting)	Garry L. McKee, PhD
Oregon	David W. Fleming, MD	Michael R. Skeels, PhD, MPH
Pennsylvania	James T. Rankin, Jr, DVM, PhD, MPH	Bruce Kleger, DrPH
Rhode Island	Utpala Bandyopadhyay, MD, MPH	Walter S. Combs, PhD
South Carolina	James J. Gibson, MD, MPH	Harold Dowda, PhD
South Dakota	Susan E. Lance, DVM, PhD, MPH	Vacant
Tennessee	William L. Moore, MD	Michael W. Kimberly, DrPH
Texas	Diane M. Simpson, MD, PhD	David L. Maserang, PhD
Utah	Craig R. Nichols, MPA	Charles D. Brokopp, DrPH
Vermont	Peter D. Galbraith, DMD, MPH	Burton W. Wilcke, Jr, PhD
Virginia	Grayson B. Miller, Jr, MD, MPH	James L. Pearson, DrPH
Washington	Paul Stehr-Green, DrPH, 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	Gayle L. Miller, DVM, MPH	Roy J. Almeida, DrPH
American Samoa	Joseph Tufa, DSM	—
Federated States of Micronesia	Vacant	—
Guam	Robert L. Haddock, DVM, MPH	—
Marshall Islands	Tom D. Kijner	—
Northern Mariana Islands	Jose L. Chong, MD	Isamu J. Abraham, DrPH
Palau	Jill McCready, MS, MPH	—
Puerto Rico	Carmen C. Deseda, MD, MPH	Jose Luis Miranda Arroyo, MD
Virgin Islands	Donna M. Green, 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 free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy on Friday of each week, send an e-mail message to lists@list.cdc.gov. The body content should read *subscribe mmwr-toc*. Electronic copy also is available from CDC's World-Wide Web server at <http://www.cdc.gov/> or from CDC's file transfer protocol server at <ftp.cdc.gov>. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 512-1800.

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 following Friday. Address inquiries about the *MMWR* Series, including material to be considered for publication, to: Editor, *MMWR* Series, Mailstop C-08, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone (404) 332-4555.

All material in the *MMWR* Series is in the public domain and may be used and reprinted without permission; citation as to source, however, is appreciated.

☆U.S. Government Printing Office: 1997-532-228/47055 Region IV