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Surveillance for Waterborne-Disease Outbreaks — United States, 1995–1996

Cardiovascular Disease Risk Factors and Preventive Practices Among Adults — United States, 1994: A Behavioral Risk Factor Atlas

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
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*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

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Surveillance for Waterborne-Disease Outbreaks — United States, 1995–1996

Deborah A. Levy, Ph.D., M.P.H.[1,2]
Michelle S. Bens, M.P.H.[2]
Gunther F. Craun, M.P.H.[3]
Rebecca L. Calderon, Ph.D., M.P.H.[4]
Barbara L. Herwaldt, M.D., M.P.H.[2]
[1]*Epidemic Intelligence Service, Epidemiology Program Office, CDC*
[2]*Division of Parasitic Diseases, National Center for Infectious Diseases, CDC*
[3]*Gunther F. Craun & Associates, Staunton, Virginia*
[4]*Human Studies Division, National Health and Environmental Effects Laboratory, U.S. Environmental Protection Agency*

Abstract

Problem/Condition: Since 1971, CDC and the U.S. Environmental Protection Agency have maintained a collaborative surveillance system for collecting and periodically reporting data that relate to occurrences and causes of waterborne-disease outbreaks (WBDOs).

Reporting Period Covered: This summary includes data for January 1995 through December 1996 and previously unreported outbreaks in 1994.

Description of the System: The surveillance system includes data about outbreaks associated with drinking water and recreational water. State, territorial, and local public health departments are primarily responsible for detecting and investigating WBDOs and for voluntarily reporting them to CDC on a standard form.

Results: For the period 1995–1996, 13 states reported a total of 22 outbreaks associated with drinking water. These outbreaks caused an estimated total of 2,567 persons to become ill. No deaths were reported. The microbe or chemical that caused the outbreak was identified for 14 (63.6%) of the 22 outbreaks. *Giardia lamblia* and *Shigella sonnei* each caused two (9.1%) of the 22 outbreaks; *Escherichia coli* O157:H7, *Plesiomonas shigelloides*, and a small round structured virus were implicated for one outbreak (4.5%) each. One of the two outbreaks of giardiasis involved the largest number of cases, with an estimated 1,449 ill persons. Seven outbreaks (31.8% of 22) of chemical poisoning, which involved a total of 90 persons, were reported. Copper and nitrite were associated with two outbreaks (9.1% of 22) each and sodium hydroxide, chlorine, and concentrated liquid soap with one outbreak (4.5%) each. Eleven (50.0%) of the 22 outbreaks were linked to well water, eight in noncommunity and three in community systems.

Only three of the 10 outbreaks associated with community water systems were caused by problems at water treatment plants; the other seven resulted from problems in the water distribution systems and plumbing of individual facilities (e.g., a restaurant). Six of the seven outbreaks were associated with chemical contamination of the drinking water; the seventh outbreak was attributed to a small round structured virus. Four of the seven outbreaks occurred because of backflow or backsiphonage

through a cross-connection, and two occurred because of high levels of copper that leached into water after the installation of new plumbing. For three of the four outbreaks caused by contamination from a cross-connection, an improperly installed vacuum breaker or a faulty backflow prevention device was identified; no protection against backsiphonage was found for the fourth outbreak.

Thirty-seven outbreaks from 17 states were attributed to recreational water exposure and affected an estimated 9,129 persons, including 8,449 persons in two large outbreaks of cryptosporidiosis. Twenty-two (59.5%) of these 37 were outbreaks of gastroenteritis; nine (24.3%) were outbreaks of dermatitis; and six (16.2%) were single cases of primary amebic meningoencephalitis caused by *Naegleria fowleri*, all of which were fatal. The etiologic agent was identified for 33 (89.2%) of the 37 outbreaks. Six (27.3%) of the 22 outbreaks of gastroenteritis were caused by *Cryptosporidium parvum* and six (27.3%) by *E. coli* O157:H7. All of the latter were associated with unchlorinated water (i.e., in lakes) or inadequately chlorinated water (i.e., in a pool). Thirteen (59.1%) of these 22 outbreaks were associated with lake water, eight (36.4%) with swimming or wading pools, and one(4.5%) with a hot spring. Of the nine outbreaks of dermatitis, seven (77.8%) were outbreaks of *Pseudomonas* dermatitis associated with hot tubs, and two (22.2%) were lake-associated outbreaks of swimmer's itch caused by *Schistosoma* species.

Interpretation: WBDOs caused by *E. coli* O157:H7 were reported more frequently than in previous years and were associated primarily with recreational lake water. This finding suggests the need for better monitoring of water quality and identification of sources of contamination. Although protozoan parasites, especially *Cryptosporidium* and *Giardia*, were associated with fewer reported outbreaks than in previous years, they caused large outbreaks that affected a total of approximately 10,000 persons; all of the outbreaks of cryptosporidiosis were associated with recreational water, primarily swimming pools. Prevention of pool-associated outbreaks caused by chlorine-resistant parasites (e.g., *Cryptosporidium* and to a lesser extent *Giardia*) is particularly difficult because it requires improved filtration methods as well as education of patrons about hazards associated with fecal accidents, especially in pools frequented by diaper-aged children. The proportion of reported drinking water outbreaks associated with community water systems that were attributed to problems at water treatment plants has steadily declined since 1989 (i.e., 72.7% for 1989–1990, 62.5% for 1991–1992, 57.1% for 1993–1994, and 30.0% for 1995–1996). This decrease might reflect improvements in water treatment and in operation of plants. The outbreaks attributed to contamination in the distribution system suggest that efforts should be increased to prevent cross-connections, especially by installing and monitoring backflow prevention devices.

Actions Taken: Surveillance data that identify the types of water systems, 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. In addition, they are used to establish research priorities and can lead to improved water-quality regulations.

INTRODUCTION

Since 1920, national statistics on outbreaks associated with drinking water have been available (*1*). Since 1971, CDC, the U.S. Environmental Protection Agency (EPA), and the Council of State and Territorial Epidemiologists have maintained a collaborative surveillance system consisting of the collection and periodic reporting of data on the occurrences and causes of waterborne-disease outbreaks (WBDOs) (*2–4*). The surveillance system includes data about outbreaks associated with drinking and recreational water. This summary includes data for 1995 and 1996 and for previously unreported outbreaks in 1994.

CDC's and EPA's efforts related to waterborne-disease surveillance have the following goals: a) to characterize the epidemiology of WBDOs; b) to identify the etiologic agents that caused WBDOs and to determine why the outbreaks occurred; c) to train public health personnel in how to detect and investigate WBDOs; and d) to collaborate with local, state, federal, and international agencies on initiatives to prevent waterborne diseases. The data gathered through this surveillance system are useful for evaluating the adequacy of current technologies for providing safe drinking and recreational water. Surveillance information also influences research priorities and can lead to improved water-quality regulations.

EPA REGULATIONS FOR DRINKING WATER

Public water systems are regulated under the Safe Drinking Water Act of 1974 (*5*), as amended in 1986 (*6*) and 1996 (*7*). Microbial contamination is regulated under the Surface Water Treatment Rule of 1989 (*8*) and the Total Coliform Rule of 1989 (*8–10*). The Surface Water Treatment Rule includes regulations for filtration, disinfection, and turbidity, as well as treatment criteria for removing *Giardia lamblia*, viruses, *Legionella*, and heterotrophic plate-count bacteria. All public community and noncommunity water systems that use surface-water sources or groundwater sources under the direct influence of surface water are covered by the Surface Water Treatment Rule, which requires these water systems to disinfect their water and to maintain a disinfectant residual in their distribution system. Filtration must also be provided unless water sources meet specified criteria for water quality and source-water protection.

The Total Coliform Rule was promulgated specifically to identify public water systems that are contaminated or vulnerable to contamination. The total coliform group of organisms (see Glossary), which includes but is not limited to fecal coliforms and *Escherichia coli*, is used to indicate the possible presence or absence of pathogens and thus, provides a general indication of whether water is contaminated. The presence of fecal coliforms or *E. coli* provides stronger evidence than does a positive total coliform test of fecal contamination and the likely presence of pathogens (*9*).

Additional rules are being developed. The Enhanced Surface Water Treatment Rule, proposed July 29, 1994 (*11*), and the Ground Water Disinfection Rule, which has yet to be proposed, also will address the prevention of waterborne diseases. The Enhanced Surface Water Treatment Rule will propose changes to the Surface Water Treatment Rule to provide additional protection against *Cryptosporidium parvum* and other waterborne pathogens, including *Giardia* and viruses. The regulation will be im-

plemented in two stages: an interim rule and a final rule. Treatment requirements under the interim Enhanced Surface Water Treatment Rule are scheduled to be announced in late 1998.

The Ground Water Disinfection Rule will apply to both community and noncommunity water systems served by groundwater. Maximum contaminant level goals will be set for pathogens. A maximum contaminant level goal of zero will be set for viruses and possibly *Legionella*. No maximum contaminant level goal is expected for heterotrophic plate-count bacteria. In lieu of monitoring for pathogens, performance criteria for water treatment will be established that are expected to produce the desired reduction in levels of pathogens. Minimum levels of disinfection will be proposed for viruses and *Legionella*, if included in the new rule.

To fill gaps in existing data on the occurrence of microbial pathogens and other indicators of microbial contamination, the EPA promulgated the Information Collection Rule (*12*). This rule requires treatment plants that use surface water and supply communities of ≥100,000 persons or that use groundwater systems and supply communities of ≥50,000 persons to monitor their source water for specific microbes and chemicals beginning in July 1997. If the concentration of microbes or chemicals exceeds a predetermined threshold, then the utility must also monitor its finished water. Microbial monitoring is intended a) to provide data on the occurrence of pathogens and the effectiveness of treatment for the removal of pathogens and b) to evaluate the adequacy of the Surface Water Treatment Rule and the Total Coliform Rule. The Information Collection Rule requires utilities to monitor for the presence of *Cryptosporidium* and *Giardia*, total culturable viruses, and total and fecal coliforms or *E. coli* at least once a month for 18 months.

METHODS

Sources of Data

State, territorial, and local public health agencies have the primary responsibility for detecting and investigating WBDOs and voluntarily reporting them to CDC on a standard form (CDC form 52.12 [*4*]). CDC annually requests reports from state and territorial epidemiologists or from persons designated as the WBDO surveillance coordinators. When needed, additional information about water quality and treatment is obtained from the state's drinking water agency.

Definition of Terms*

The unit of analysis for the WBDO surveillance system 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, two or more persons must have experienced a similar illness after either ingestion of drinking water or exposure to water used for recreational purposes. This 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. Sec-

*Additional terms are defined in the glossary.

ond, epidemiologic evidence must implicate water as the probable source of the illness. Outbreaks caused by contamination of water or ice at the point of use (e.g., a contaminated water faucet or serving container) are not classified as WBDOs.

If primary cases (among persons exposed to contaminated water) and secondary cases (among persons who became ill after contact with primary case-patients) are distinguished on the outbreak report form, only primary cases are included in the total number of cases. If both actual and estimated case counts are included on the outbreak report form, the estimated case count is used if the study population was sampled randomly or the estimated count was calculated by using the attack rate.

Public water systems — classified as either community or noncommunity water systems — provide piped water to the public for general consumption and are regulated under the Safe Drinking Water Act. A community water system serves year-round residents of a community, subdivision, or mobile-home park that has ≥15 service connections or an average of ≥25 residents. A noncommunity water system is defined as any public water system that is not a community water system and is used by the general public for ≥60 days per year and has ≥15 service connections or serves an average of ≥25 persons. Noncommunity water systems are divided into nontransient and transient categories, with nontransient noncommunity water systems (e.g., in factories and schools) serving ≥25 persons ≥6 months of the year. Of the approximately 170,000 public water systems in the United States, 115,000 (68%) are noncommunity systems, serving transients (95,000 systems) and nontransients (20,000); 55,000 (32%) are community systems (EPA Safe Drinking Water Information System data base, 1998). Community water systems serve approximately 243 million persons in the United States (91% of the U.S. population); approximately 24 million persons (9%) rely on private or individual water systems, which are small systems, not owned or operated by a water utility, that serve <15 connections or <25 persons. In addition, millions of persons use noncommunity systems while traveling or working.

Each drinking water system associated with a WBDO is classified as having one of the following deficiencies:

1 = untreated surface water;
2 = untreated groundwater;
3 = treatment deficiency (e.g., temporary interruption of disinfection, chronically inadequate disinfection, and inadequate or no filtration);
4 = distribution system deficiency (e.g., cross-connection, contamination of water mains during construction or repair, and contamination of a storage facility); and
5 = unknown or miscellaneous deficiency (e.g., contaminated bottled water).

If more than one deficiency is noted on the report form for an outbreak, the deficiency that most likely caused the outbreak is noted.

Recreational waters include swimming pools, whirlpools, hot tubs, spas, water parks, and naturally occurring fresh and marine surface waters. Although the surveillance system includes whirlpool- and hot tub-associated outbreaks of dermatitis caused by *Pseudomonas aeruginosa*, it does not include wound infections resulting from waterborne organisms (e.g., *Aeromonas* species).

Classification of Outbreaks

This surveillance system classifies WBDOs according to the strength of the evidence implicating water (Table 1). The classification numbers (i.e., I–IV) are based on the epidemiologic and water-quality data provided on the report form. Epidemiologic data are weighted more heavily than water-quality data. Thus, although some outbreaks without water-quality data were included in this summary, reports without supporting epidemiologic data were excluded. Outbreaks of *Pseudomonas* dermatitis and single cases of primary amebic meningoencephalitis or of illness resulting from chemical poisoning are not classified according to this scheme.

A classification of I means that adequate epidemiologic and water-quality data were reported but does not necessarily imply that the investigation was optimal. 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.

RESULTS

1995–1996 Outbreaks Associated with Drinking Water

For the period 1995–1996, 13 states reported a total of 22 outbreaks associated with drinking water. Sixteen outbreaks were reported for 1995 and six for 1996. Wisconsin reported five outbreaks (22.7%), followed by Idaho, which reported three (13.6%). The outbreaks caused an estimated total of 2,567 persons to become ill. The median outbreak size was 22 persons (range: 1–1,449). No deaths were attributed to these outbreaks. Outbreaks were most common in the summer and fall months (Figure 1). Seventeen (77.3%) of the 22 outbreaks occurred during June through October.

Twelve (54.5%) of the 22 outbreaks were assigned a classification of I based on epidemiologic and water-quality data, three (13.6%) Class II, and six (27.3%) Class III; an individual case of chlorine poisoning was not classified. Outbreaks are listed individually by state (Tables 2 and 3) and are tabulated by the etiologic agent and type of water system (Table 4) and by the type of deficiency and type of water system (Table 5).

Etiologic Agents

Fifteen (68.2%) of the 22 outbreaks were of infectious or suspected infectious etiology, and seven (31.8%) were attributed to chemical poisoning. Two (9.1%) of the 22 outbreaks were caused by the parasite *Giardia*, four (18.2%) by bacteria, one (4.5%) by a small round structured virus, and eight (36.4%) were of unknown etiology (Figure 2).

Parasites. Two outbreaks in 1995 were caused by *Giardia,* one in Alaska and the other in New York. The outbreak of giardiasis in Alaska occurred in August, affected 10 persons, and was associated with untreated surface water. The outbreak in New York occurred in December, affected an estimated 1,449 persons, and was associated with surface water that was both chlorinated and filtered. A dose-response relation was found between consumption of municipal water and illness. No interruptions in chlori-

nation were identified at the water plant. Postfilter water turbidity readings, which serve as an index of the effectiveness of filtration, exceeded the regulated limit (i.e., 95% of water samples must be <0.5 nephelometric turbidity units [NTUs] for conventional and direct filtration) before and during the outbreak.

Bacteria. Four outbreaks were caused by bacteria: two were attributed to *Shigella sonnei*, one to *E. coli* O157:H7, and one to *Plesiomonas shigelloides*. The outbreak of shigellosis in Idaho, which affected 83 persons, occurred at a resort supplied by untreated well water, which became contaminated by sewage from a line that was draining poorly (*13*). The outbreak of shigellosis in Oklahoma, which affected 10 persons, was associated with tap water in a convenience store that was supplied by chlorinated well water. Although the factors contributing to contamination of the water were not determined, the water was thought to have been inadequately chlorinated.

The outbreak of *E. coli* O157:H7 infection occurred at a summer camp in Minnesota that was supplied by chlorinated spring water. Several of the 33 affected persons had stool cultures that also were positive for *Campylobacter jejuni* and *Salmonella* serotype London. Water samples from the spring and distribution system were positive for coliforms and *E. coli*. The contamination was attributed to flooding from heavy rains and to an improperly constructed spring.

The outbreak of *Plesiomonas shigelloides* infection occurred in a restaurant in New York that was supplied by a noncommunity water system. The outbreak affected 60 persons and is thought to be the largest outbreak of *Plesiomonas* infection reported in the United States (*14*). Chlorinated spring water that supplied a kitchen tap in the restaurant had a high coliform count, including *E. coli*, and the disinfectant residual was zero. The chlorinator was found to be depleted of disinfectant, and cultures of water from the river adjacent to the uncovered reservoir where treated water was stored grew *Plesiomonas*.

Viruses. One outbreak in 1995 was documented to have been caused by a virus, specifically a small round structured virus. The outbreak occurred in September at a high school in Wisconsin and affected 148 persons. The school received its drinking water from a community water supply. Contamination of the potable water system likely occurred from backsiphonage of water through hoses submerged in a flooded football field. However, the source of the virus on the field was not determined.

Chemicals. Seven outbreaks of chemical poisoning were reported, one of which was an individual case of chlorine poisoning. A person became ill immediately after drinking water obtained from a Florida restaurant's drive-through window; symptoms included a burning sensation in the throat and vomiting. Water samples obtained on the day of the incident had chlorine levels ranging from 1.2 mg/L to 4.7 mg/L (median: 4.5 mg/L). The source of the excessive levels of chlorine in the drinking water at the restaurant remained unclear.

In the two outbreaks of nitrite poisoning (i.e., in California and New Jersey), defective check valves for prevention of backflow allowed chemicals to contaminate drinking water. In California, three persons at a school became ill after consuming water from a system that had a double-check backflow prevention valve that did not

meet the industry standard and that allowed chemicals used to treat a cooling tower and chilling system for the school's air conditioning unit to contaminate the drinking water. Furthermore, the valve had rubber gaskets that were badly deteriorated. In New Jersey, the drinking water was contaminated with boiler-conditioning fluids through a faulty backflow check valve. Six persons developed acute onset of cyanosis and were diagnosed with methemoglobinemia caused by nitrites, which are strong oxidizing agents (*15*).

Elevated copper levels in tap water in Wisconsin were associated with gastrointestinal illness in at least 37 persons in private homes. The homes in this community had recently been built or remodeled, and new copper plumbing was thought to have contributed to the contamination of the water.

Thirty persons in Florida developed chemical burns in their mouths after they drank water contaminated with sodium hydroxide. The water became contaminated when an operator at the treatment plant unintentionally released the chemical into the water. The pH of the finished water was 11.8.

Thirteen persons at a health-care facility in lowa developed burning in their mouths and flu-like symptoms after drinking water contaminated with a concentrated liquid soap. A valve on the water supply hose to the soap dispenser had been left open, allowing the soap to enter the water system. Vacuum breakers to prevent backsiphonage were installed incorrectly at the soap dispensers.

Unidentified Etiologic Agent. The etiologic agent was not identified for eight (36.4%) of the 22 WBDOs associated with drinking water. The illnesses associated with at least four of these outbreaks had incubation periods, durations, and symptom complexes that were consistent with viral syndromes. For five of the six outbreaks for which testing was done, including three of the four outbreaks of suspected viral etiology, stool specimens were negative for bacterial and parasitic pathogens. One of these outbreaks (i.e., an outbreak in 1995 at the restaurant of a resort in Wisconsin) might have been caused by a rotavirus. The state laboratory reported identifying rotavirus in two of six stool specimens with an enzyme immunoassay. Eight stool specimens were tested for enteric pathogens and three for enteroviruses. However, no testing was done for Norwalk-like caliciviruses, which have been more commonly associated with WBDOs than the rotaviruses have been.

Of the eight outbreaks for which the etiologic agent was not identified, three outbreaks were associated with untreated well water, three with inadequate chlorination of unfiltered well water, and one with possible short-term cross-connection and backsiphonage problems in the distribution system. The other outbreak was associated with water from an outside tap at a waste-water treatment plant that was not marked as nonpotable. Even though the water was not intended for drinking, this outbreak was categorized as a drinking water outbreak for lack of a better category. Hikers accessed the tap by entering fenced property. The water from the tap was waste water that had been through a sedimentation process twice but still required additional treatment; the water had levels of fecal coliforms >23 most probable number/100 mL.

Water-Quality Data

Water-quality data (e.g., information about the presence of coliform bacteria, pathogens, or chemical contaminants) were available for 20 (90.9%) of the 22 out-

breaks. The relevant chemical was found in water samples for all seven outbreaks of chemical poisoning. Water samples were tested for coliform bacteria during the investigation of 13 (86.7%) of the 15 outbreaks that had a known or suspected infectious etiology and were positive for total coliforms or fecal coliforms for 11 (84.6%) of the 13 outbreaks. No information about the presence of coliforms was available for an outbreak of giardiasis and an outbreak of shigellosis. Coliforms were detected for three (75.0%) of the four bacterial outbreaks, and all eight outbreaks of unknown etiology, including one for which water samples collected 1 month after the outbreak (at a campground using untreated well water) did not exceed prescribed limits. Coliforms were not detected for two outbreaks. *Giardia*-like cysts and *Cryptosporidium*-like oocysts were found in a filtered, chlorinated water system during the investigation of an outbreak of giardiasis; but no coliforms were detected in water samples from the distribution system. During the investigation of an outbreak caused by a small round structured virus at a school, no coliforms could be detected 4 days after the water, which was chlorinated and filtered, had been contaminated by backsiphonage.

Water System and Water Source

Ten (45.5%) of the 22 WBDOs were associated with community systems, 10 (45.5%) with noncommunity systems, and two (9.1%) with individual water systems (Tables 4 and 5; Figure 2). Only three of the 10 outbreaks associated with community water systems were caused by problems at water treatment plants, and the other seven were the result of problems in the water distribution systems and plumbing of individual facilities (e.g., offices, schools, and restaurants). Six of these seven outbreaks were associated with chemical contamination of the drinking water. Two of the six outbreaks were attributed to nitrites that contaminated the water when check valves for prevention of backflow at the facilities malfunctioned; two resulted from copper leaching into the water after plumbing was installed in new homes; one occurred when concentrated liquid soap contaminated the water because of backsiphonage through an improperly installed vacuum breaker; and in one outbreak, the source of the excessive levels of chlorine in the drinking water at a restaurant remained unclear. The seventh outbreak was attributed to a small round structured virus that likely contaminated the drinking water when backsiphonage of water through hoses submerged in a flooded football field occurred. During 1995–1996, outbreaks in noncommunity systems were more likely than those in community systems to be associated with untreated water (40.0% versus 0.0%). Eight (80.0%) of the 10 outbreaks in noncommunity systems were associated with well-water sources, as were three (30.0%) of the 10 community outbreaks.

Of the 15 outbreaks with a known or suspected infectious etiology, nine (60.0%) were associated with well-water sources, three (20.0%) with surface-water sources, two (13.3%) with spring-water sources, and one (6.7%) with partially treated sewage. For four (44.4%) of the nine well-water systems, the water was untreated. For another four (44.4%), the identified deficiency was inadequate chlorination or interrupted disinfection (e.g., coliforms, which are chlorine sensitive, were present in tap water). For one (11.1%), the deficiency occurred in the distribution system. The identified deficiency for both outbreaks associated with spring-water systems was inadequate or interrupted chlorination. For the outbreaks associated with surface-water systems, the identified deficiencies were inadequate filtration, backsiphonage, and no treatment.

1995–1996 Outbreaks Associated with Recreational Water

For the period 1995–1996, 17 states reported 37 outbreaks associated with recreational water (Tables 6–8). Twenty-three outbreaks were reported for 1995 and 14 for 1996. The states that reported the most outbreaks were Minnesota (six outbreaks) and Texas (five outbreaks). The 37 outbreaks caused illness in an estimated 9,129 persons. The median outbreak size was 10 persons (range: 1–5,449). All but one of the 22 outbreaks of gastroenteritis occurred during the summer (Figure 1). The six cases of primary amebic meningoencephalitis, all of which were fatal, occurred in summer and early fall. Five (55.6%) of the nine outbreaks of dermatitis (i.e., rash or folliculitis), which all were associated with hot tubs and lakes, occurred during the relatively colder months of September through February.

Etiologic Agents

Thirty-three (89.2%) of the 37 recreational water outbreaks were of infectious etiology (Tables 6–8; Figure 3). Seven (31.8%) of the 22 outbreaks of gastroenteritis were caused by parasites, 10 (45.5%) by bacteria, one (4.5%) by a virus, and four (18.2%) were of unknown etiology (Tables 6 and 7).

Parasites. Six (27.3%) of the 22 outbreaks of gastroenteritis were caused by *Cryptosporidium* and one (4.5%) by *Giardia*. Six of these seven outbreaks were associated with swimming pools and water parks. In 1995, a large outbreak of cryptosporidiosis at a water park in Georgia caused an estimated 5,449 persons to become ill after a probable fecal accident in the children's pool. Some stools were positive for both *Cryptosporidium* and *Giardia*. Similarly, in 1996, an estimated 3,000 persons acquired cryptosporidiosis after visiting a water park in California. Again, a few of the stool specimens were also positive for *Giardia*. Park patrons were exposed to untreated water both at the swimming pool and when water from a jet-ski sprayed an audience watching a show.

At a swimming pool in Kansas, 24 persons acquired cryptosporidiosis, one of whom was hospitalized (*16*). Inadequate filtration and possible fecal accidents in the pool led to an outbreak of cryptosporidiosis associated with a shallow wading pool in Florida. Twenty-two persons became ill, six of whom were hospitalized. Other conditions that might have contributed to the outbreak included overcrowding of the area around the pool, an excessive number of swimmers, loss of water clarity, and an ozonator that was not operational. After heavy rains, runoff that contained cattle feces and that passed from a pasture into a lake led to an outbreak in Indiana in which three persons became ill with cryptosporidiosis. One of the stool specimens was also positive for *Giardia* cysts. Run-off containing livestock feces might have been responsible for an outbreak of cryptosporidiosis in Nebraska, which affected 14 persons.

Seventy-seven persons in Florida became ill after visiting a children's wading pool. Sixty persons had stool specimens that tested positive for *Giardia*, 17 had specimens that tested positive for *Cryptosporidium*, and eight had specimens that were positive for both organisms. The wading pool was supplied by municipal well water that was coagulated, settled, filtered, and disinfected with chlorine.

In 1995, six cases of primary amebic meningoencephalitis were attributed to *Naegleria*. All six of the infected children, who ranged in age from 4 to 11 years, died.

Infection was acquired when the children swam in a shallow lake, river, pond, or canal. Five of the six cases were associated with exposure in Texas and one in Florida.

Bacteria. Ten (45.5%) of the 22 outbreaks of gastroenteritis were attributed to bacteria, and eight of these outbreaks were associated with lakes. Six outbreaks (27.3%) were caused by *E. coli* O157:H7 (*17*), three (13.6%) by *Shigella sonnei*, and one (4.5%) by *Salmonella* serotype Java. At a swimming pool in Georgia, 18 persons became ill after ingesting water contaminated with *E. coli* O157:H7. Inadequate chlorination was thought to have allowed the *E. coli* to multiply. Thirty-four persons became ill from five outbreaks of *E. coli* O157:H7 infection associated with lakes.

All three outbreaks of shigellosis were associated with lakes. In Colorado, a lake contaminated with human feces caused a total of 120 persons to become ill in two outbreaks. In Pennsylvania, soiled diapers were found near the implicated lake, and most of the laboratory-confirmed cases were in children aged <10 years who were playing in the sand close to the water.

Other. In an outbreak in Idaho in 1996, 55 persons became ill from infection with Norwalk virus. Lake water tested positive for coliforms, and the outbreak was presumptively attributed to fecal contamination by swimmers. No agent could be identified for four (18.2%) of the 22 outbreaks of gastroenteritis; all four of these outbreaks were associated with lakes.

An estimated 169 persons were affected in nine outbreaks of dermatitis that were associated with hot tubs or lakes. All nine outbreaks had a known or suspected infectious etiology (Table 8). *Pseudomonas aeruginosa* was confirmed as the etiologic agent for four of the seven *Pseudomonas* outbreaks and was suspected (based on the clinical syndrome) for the other three. *Schistosoma* sp. was the presumptive etiologic agent of the two outbreaks of swimmer's itch.

Previously Unreported Outbreaks

Reports of three previously unpublished WBDOs for 1994 also were received (Table 9). In the one outbreak associated with drinking water, well water at a small mobile-home park in Florida was inadequately treated, and coliforms were detected in samples of chlorinated water. Two persons were reported to have been ill, one of whom was hospitalized. The other two outbreaks were associated with recreational water and affected an estimated 312 persons. The etiologic agent was not identified for one of these two outbreaks. In the outbreak in Florida, 12 persons had gastroenteritis, two of whom were hospitalized. This outbreak occurred among children and adults who attended a birthday party at a park and swam in a lake, which was fed by an artesian well and had a history of high-quality water. During the investigation, samples of lake water had 80–230 total coliforms/mL and up to 40 fecal coliforms/mL. In a New Jersey state park, at least 300 cases of shigellosis were reported among bathers in the swimming area of a reservoir where numerous fecal accidents were reported and persons were seen rinsing diapers in lake water.

Outbreaks Not Classified as Waterborne-Disease Outbreaks

Outbreaks attributed to drinking water contaminated at its point of use, rather than at the source or in the distribution system, traditionally are not classified as WBDOs. Seven point-of-use outbreaks that affected a total of 200 persons were reported, one from 1991 and six from the 1995–1996 reporting period. Twenty-one persons became ill with symptoms suggestive of viral gastroenteritis after consuming ice at a picnic in New York in 1991. In Florida, consumption of water from an outdoor faucet equipped with a hose by attendees at a day camp resulted in 77 primary cases of cryptosporidiosis, as well as 24 probable secondary cases in household members (*18*). Portable water coolers were filled with water from the hose, which was also used for rinsing garbage cans. Water samples from the outdoor faucet were positive for total coliforms and *Cryptosporidium*, whereas samples collected at other sites at the school where the camp was held and at the municipal water plant were negative or below detectable limits for total coliforms, *E. coli*, and parasites. Feces were observed on several occasions near the faucet and attached hose; thus, the most likely source of contamination of the water was the hose nozzle.

Three outbreaks in the 1995–1996 reporting period were thought to be associated with consumption of contaminated ice. At a church festival in Wisconsin, 27 cases of *E. coli* O157:H7-related gastroenteritis were associated with consumption of ice from plastic water containers that had been filled with water and then frozen. The possible sources of contamination of the ice included a water faucet, which might have been contaminated while preparing ground beef, and the plastic water containers, which might have been contaminated when they were previously used to store the ground beef. Bagged ice was linked to infection with *Campylobacter* sp. in seven persons who became ill at a private home in Ohio. The third ice-related outbreak occurred in New Jersey and affected 39 persons who became ill with symptomatology suggestive of viral gastroenteritis after consuming ice that might have been contaminated with human sewage. In Florida, two persons became ill after consuming iced tea that had been left in a water cooler overnight. The acid in the tea caused the metal coils in the water cooler to corrode and release metals into the tea. Seventeen possible WBDOs that occurred during 1995–1996 were not included in this surveillance summary because of insufficient epidemiologic data (i.e., the outbreaks did not meet the criteria for Classes I–IV).

DISCUSSION

General Considerations About Surveillance Data for Waterborne-Disease Outbreaks

The waterborne-disease surveillance data, which identify the types of water systems, their deficiencies, and the respective etiologic agents associated with the outbreaks, are useful for evaluating the adequacy of current technologies for providing safe drinking and recreational water. However, the data in this surveillance summary are subject to at least one important limitation: they probably do not reflect the true incidence of WBDOs or the relative incidence of outbreaks caused by various etiologic agents. Not all WBDOs are recognized, investigated, and reported to CDC or

EPA; and the extent to which WBDOs are unrecognized and underreported is unknown.

The likelihood that individual cases of illness will be detected, epidemiologically linked, and associated with water varies considerably among locales and is dependent on factors such as public awareness, the likelihood that multiple ill persons consult the same rather than different health-care providers, the interest of health-care providers, availability of laboratory-testing facilities, local requirements for reporting cases of particular diseases, and surveillance and investigative activities and capacities of state and local health and environmental agencies. Therefore, the states that report the most outbreaks might not be those in which the most outbreaks occur. Recognition of WBDOs also is dependent on certain outbreak characteristics; outbreaks involving serious illness are most likely to receive the attention of health authorities. Outbreaks of acute diseases, particularly those characterized by a short incubation period, are more readily identified than those associated with disease from chronic, low-level exposure to an agent (e.g., a chemical). Outbreaks associated with community water systems are more likely to be recognized than those associated with noncommunity systems because the latter serve mostly nonresidential areas and transient populations. Outbreaks associated with individual systems are the most likely to be underreported because they generally involve relatively few persons.

The identification of the etiologic agent of a WBDO is dependent on the timely recognition of the outbreak so that appropriate clinical and environment samples can be obtained. The interests and expertise of investigators and the routine practices of local laboratories can also influence whether the etiologic agent is identified. For example, diarrheal stool specimens generally are examined for bacterial pathogens, but not for viruses. In most laboratories, testing for *Cryptosporidium* is done only if requested and is not included in routine stool examinations for ova and parasites (*19*). The water-quality data that are collected vary widely among outbreak investigations, depending on such factors as available fiscal, investigative, and laboratory resources. Furthermore, a few large outbreaks can substantially alter the relative proportion of cases of waterborne disease attributed to a particular agent. Finally, the number of reported cases is generally an approximate figure, and the method and accuracy of the approximation vary among outbreaks.

1995–1996 Outbreaks Associated with Drinking Water

The number of outbreaks reported for 1995 (i.e., 16) is comparable with those reported for each year during 1987–1994, except for an increase in 1992 (*2–4*; Figures 4 and 5). However, the number of outbreaks reported for 1996 (i.e., six) is much lower than for previous years. WBDO reports peaked during 1979–1983. The increase and subsequent decrease in the number of reports might reflect, at least in part, changes in surveillance activities (*20*). The decrease in the number of outbreaks reported for 1996 might indicate the beginning of a new trend or simply might reflect a reporting artifact.

The number of outbreaks attributed to various etiologic agents changed for this reporting period. The number caused by parasites decreased substantially from the previous reporting period (i.e., 1993 and 1994), during which 10 reported outbreaks were caused by parasites (i.e., five outbreaks each attributed to *Cryptosporidium* and

Giardia). For the 1995–1996 reporting period, only two outbreaks were caused by parasites. However, one of these two outbreaks (i.e., the outbreak of giardiasis in New York) affected the largest number of persons of any outbreak for this reporting period.

The outbreak of giardiasis in New York was associated with surface water that was filtered and disinfected with chlorine. Although no interruptions in chlorination at the water plant were identified, postfilter water turbidity readings, which serve as an index of the effectiveness of filtration, exceeded the regulated limit before and during the outbreak. The occurrence of the two outbreaks of giardiasis underscores the importance of requiring water systems to monitor turbidity, to meet turbidity standards, and to provide an adequate chlorine concentration and contact time (as specified by the Surface Water Treatment Rule) to inactivate *Giardia* and other organisms that are relatively chlorine-resistant, especially if the surface water is unfiltered (*21*). *Giardia* can be inactivated by disinfection without filtration, but only if stringent conditions are consistently maintained. Providing both filtration and chlorination is an example of using multiple barriers to protect water supplies. The outbreak of giardiasis in New York reportedly was the first attributed to a filtered municipal water system in that state and demonstrates that this organism remains a public health risk even in chlorinated and filtered water systems if levels of water turbidity are not consistently maintained (i.e., 95% of water samples must be <0.5 NTUs for conventional and direct filtration). Optimal filtration requires frequent, if not continuous, monitoring of the turbidity of the water both before filtration (i.e., after coagulation, flocculation, and/or settling) and after filtration.

No outbreaks in 1995 or 1996 were attributed to *Cryptosporidium*, a protozoan parasite that is >50-fold more chlorine-resistant than *Giardia*. More stringent EPA standards for acceptable turbidity values have become effective in all states since the outbreak of cryptosporidiosis in Milwaukee in 1993 (*22*). Many of the large water utilities have joined the Partnership For Safe Water (*23*), which is an American Water Works Association/EPA activity that helps treatment plants consistently achieve low water turbidity values, reducing the risk for outbreaks of cryptosporidiosis and giardiasis. The number of small utilities that have joined the partnership is unknown.

During 1995–1996, all four outbreaks caused by bacteria were associated with noncommunity systems that used either untreated well water or inadequately chlorinated surface or groundwater. Adequate, continuous disinfection of surface water and groundwater used for drinking water should reduce the occurrence of WBDOs, particularly for small systems in which intermittent contamination of wells and springs is difficult to detect or prevent. In addition, wells and springs should be protected from sources of contamination such as surface run-off, septic-tank drainage, and sewage discharges.

Unlike in recent years (i.e., 1991–1994), for which no viral outbreaks were reported, one reported outbreak in 1995 was documented to have been caused by a virus. Researchers used electron microscopy to identify a small round structured virus in stool specimens. Methods for detection of enteric viruses have improved greatly in recent years (*24*). State health departments should be encouraged to submit clinical specimens for viral testing, either at CDC or at state laboratories that have developed the capability to conduct these tests.

Seven outbreaks of chemical poisoning were reported to CDC for 1995–1996, which is similar to the number of outbreaks for the 1993–1994 reporting period (i.e., eight).

The two outbreaks associated with nitrite poisoning highlight the importance of preventing backflow when potable water systems are connected to boilers and water chillers; approved backflow prevention devices should be used and periodically monitored for effectiveness. EPA has established a maximum contaminant level of 1.0 mg/L for nitrites; this regulation applies to public water systems but not individual water systems. The two outbreaks of copper poisoning underscore the fact that corrosive water can cause leaching of metals from household plumbing and the water distribution system. EPA requires monitoring for copper (and lead) at the tap rather than at the treatment plant, and EPA's action level for copper is 1.3 mg/mL (*25*). The results of this monitoring are used to determine whether treatment to control corrosion is needed or is being applied properly. Occupants of new and older homes in communities that could have corrosive water might be able to reduce their risk for chemical poisoning by adequately flushing water through the household system before drinking the water, especially if the water has stood overnight.

Several reasons could help explain why waterborne chemical poisonings are rarely reported to CDC: a) most poisonings of this nature (e.g., those associated with the leaching of copper from plumbing systems) probably occur in private residences, affect relatively few persons and thus, might not come to the attention of public health officials; b) exposure to chemicals via drinking water can cause illness that is difficult to attribute to chemical intoxication, or it can cause nonspecific symptoms that are difficult to link to a specific chemical; and c) the mechanisms for detecting waterborne chemical poisonings and reporting them to the WBDO surveillance system are not as well established as they are for WBDOs caused by infectious agents. Future efforts should be tailored to improve the sensitivity of surveillance activities, the detection of associations between environmental releases or exposure incidents and individual health events, and the assessment of the public health burden associated with water-related chemical exposures.

As in previous reporting periods, except for 1993–1994, a large proportion (8 or 36.4%) of the WBDOs were of unknown etiology (Figures 3 and 4). Seven (87.5%) of these eight outbreaks were associated with groundwater sources, three (42.9%) of which involved untreated water; three (42.9%) a treatment deficiency; and one (14.3%) a deficiency in the distribution system. Of the eight outbreaks for which the etiologic agent was not identified, most (i.e., seven) were associated with noncommunity or individual systems; this finding reflects the difficulty of investigating outbreaks affecting the transient populations that use water from these systems.

The relative proportion of outbreaks associated with various types of water systems has remained fairly constant; the proportions associated with community water systems were 42.3% for 1989–1990, 23.5% for 1991–1992, 46.7% for 1993–1994, and 45.5% for 1995–1996. However, the proportion of reported outbreaks associated with community water systems that were attributed to problems at water treatment plants, and thus affected entire communities, has steadily declined since 1989 (i.e., 72.7% for 1989–1990, 62.5% for 1991–1992, 57.1% for 1993–1994, and 30.0% for 1995–1996). This decrease might reflect improvements in water-treatment practices and in operation of plants. For the 1995–1996 reporting period, most outbreaks (i.e., 7 of 10) associated with community water systems were the result of problems in the distribution system at individual facilities (e.g., a restaurant). Four of these seven outbreaks were associated with problems with backflow prevention devices (i.e., they had not been installed

or had been inappropriately installed or inadequately maintained). Unfortunately, such problems at individual facilities are not amenable to actions taken by treatment plants. However, they can be remedied by effective cross-connection control regulations that require inspection and testing. Monitoring, regulating, and standardizing the practices of the multitudinous individual facilities (e.g., offices, schools, and restaurants) in this country is a daunting task.

1995–1996 Outbreaks Associated with Recreational Water

The most frequently reported WBDOs caused by exposure to recreational water were outbreaks of gastroenteritis. Swimming and other recreational activities in which the unintentional ingestion of water can occur are known to increase the risk for gastrointestinal illness, even in nonoutbreak settings (*26,27*). The number of outbreaks of gastroenteritis for 1995–1996 (i.e., 22) was greater than reported for previous years (i.e, 14 each for 1993–1994 and 1991–1992, and 13 for 1989–1990).

Although the number of outbreaks caused by parasites decreased from 10 for the previous reporting period to seven for this period, two of these seven, both attributed to *Cryptosporidium*, accounted for a total of >8,000 ill persons. Each of these recreational water outbreaks affected more persons than the total number of persons affected by all of the reported drinking water outbreaks. In both of these outbreaks, which occurred at water parks, some stool specimens also tested positive for *Giardia*. The setting for six of the seven outbreaks was a swimming pool or water park. Investigators for only one of the six outbreaks reported that the pool water was inadequately chlorinated.

All six outbreaks of cryptosporidiosis reported for 1995–1996 were associated with recreational water (i.e., none with drinking water), five of which were associated with swimming pools. *Cryptosporidium*, and to a lesser extent *Giardia*, is resistant to disinfection by chlorine at levels generally used in swimming pools. Because *Cryptosporidium* oocysts measure only 4–6 μm in diameter, pool filtration systems that use sand or other granular materials (without the special chemical pretreatment that is commonly used by the drinking water industry) might not be effective in removing oocysts. Infection can occur after swallowing as few as 10–100 oocysts (*28,29*). Therefore, presumably swallowing a single mouthful of contaminated water could cause illness.

Prevention of recreational water-associated cryptosporidiosis is particularly difficult. Effective prevention measures require efforts to improve filtration methods and the design of pools and to educate patrons. The prolonged time required to filter all the water in a pool, problems in the design of pools that result in areas with poor water circulation (i.e., "dead spots"), and mixing of water from different pools during filtration increase the risk for cryptosporidiosis (*30*). Improving filtration systems, having specific pools designated for children to reduce the risk of contaminating the entire facility, and having separate filtration systems for the pools for children and those for adults might reduce risk. However, such changes can be costly, and the degree to which they reduce risk is unknown. Behavioral changes, such as showering before entering the pool and restricting access of diaper-aged children to certain pools, will require education of both patrons and facility management because few facilities currently enforce such measures. Development and enforcement of clear and

effective policies regarding fecal accidents in recreational water facilities is needed, but the effectiveness of various approaches in this regard is unclear and should be tested. Questions that still need to be addressed include a) how long should a pool be vacated after a fecal accident? b) is it beneficial to drain a pool after a fecal accident? and c) what hyperchlorination strategy should be used, especially for *Cryptosporidium*?

The number of reported outbreaks caused by bacteria increased from four during 1993–1994 to 10 during this reporting period. The same number of outbreaks of swimming-associated shigellosis (i.e., three) were reported during 1993–1994 and this reporting period. The probable source of the pathogen for the three outbreaks during 1995–1996, as for previous outbreaks, was fecal contamination of lake water by swimmers. Five of the six outbreaks caused by *E. coli* O157:H7 also were associated with lake water, which suggests the need for better monitoring of water quality and identification of sources of contamination; the sixth was associated with water from a swimming pool that was inadequately chlorinated. *E. coli* O157:H7, like *Shigella* spp., apparently has a low infectious dose (*31,32*). Thus, infection can be acquired by swallowing water with low concentrations of these bacteria. In contrast to the outbreaks caused by parasites, most of which were associated with adequately chlorinated water, all of the outbreaks attributed to bacteria were associated with unchlorinated water (i.e., in lakes) or inadequately chlorinated water (i.e., in pools). The outbreaks associated with swimming pools underline the critical need for maintaining adequate chlorination (*30*).

EPA has published criteria for evaluating the quality of both marine and fresh water used for recreation (*33,34*). Microbial monitoring has been recommended for recreational areas potentially contaminated by sewage. However, the health risk associated with various levels of fecal coliforms has not been established. Prevention efforts have focused on providing adequate bathroom facilities, including diaper-changing areas, at recreational areas and on limiting the number of swimmers per unit area. An additional important measure, although difficult to enforce, is to prevent persons (especially young, nontoilet-trained children) from entering recreational water if they are either experiencing or convalescing from a diarrheal illness.

For the period 1995–1996, most of the reported outbreaks of dermatitis associated with hot tubs were directly related to inadequate operation and maintenance procedures. Outbreaks of *Pseudomonas* dermatitis associated with hot tubs are preventable if water is maintained at a pH of 7.2–7.8 with free, residual chlorine levels in the range of 2.0–5.0 mg/L (*35*). A person's susceptibility and immersion time, along with the number of bathers per unit area, also could influence the risk for infection (*36*).

For the period 1995–1996, all six deaths associated with recreational water were caused by primary amebic meningoencephalitis, a rarely reported disease in the United States. Naegleria infections are generally acquired during the summer months, when the temperature of fresh water is favorable for multiplication of the organism (*37,38*).

CONCLUSIONS

Information from the nationwide surveillance of WBDOs is used to characterize the epidemiology of waterborne diseases in the United States. Data about the types of water systems and deficiencies associated with outbreaks are needed to evaluate the adequacy of current regulations for water treatment and monitoring of water quality. The identification of the etiologic agents of outbreaks is particularly critical because agents newly associated with WBDOs could require new methods of control. Trends in the incidence of WBDOs caused by various etiologic agents can lead to changes in policies or resource allotment.

For agents that are recognized as important waterborne pathogens, rapid recognition and control of WBDOs are facilitated by surveillance at the local and state levels. Close communication between local health departments and water utilities is crucial. For example, if epidemiologic evidence suggests the possibility of waterborne transmission, water utilities should be contacted promptly and asked about such factors as recent treatment deficiencies and changes in source-water quality. Similarly, local policies should be developed that specify the thresholds for reporting various water-quality data to health departments. Timely water testing and environmental investigations can facilitate the identification of an outbreak's etiologic agent and the correctable source(s) of water contamination, as well as establish whether control measures (e.g., boil-water advisories) are indicated.

Means of improving the surveillance system for WBDOs should be explored. The review of information that has been gathered through other mechanisms (e.g., issuances of boil-water advisories and computerized data on water quality) could facilitate the detection of WBDOs. Special epidemiologic studies are needed that supplement the findings of this surveillance system by addressing such issues as the public health importance of newly identified agents of waterborne disease, the effectiveness of prevention strategies in nonoutbreak settings, and the timeliness with which state and local health departments act in response to these pathogens.

State health departments can request epidemiologic assistance and laboratory testing from CDC for the investigation of WBDOs. 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 viruses and parasites, which require special methods for recovery. Requests for testing for viruses should be addressed to CDC' s Viral Gastroenteritis Section, Respiratory and Enterovirus Branch, Division of Viral and Rickettsial Diseases at (404) 639-3577. Requests for testing for parasites should be addressed to CDC's Division of Parasitic Diseases at (770) 488-7760.

Additional information is available from EPA's Safe Drinking Water Hotline (telephone [800] 426-4791; e-mail sdwa@epamail.epa.gov), CDC's Cryptosporidiosis Information Line of the Parasitic Diseases Information Line (voice telephone system [888] 232-3228, fax [888] 232-3299), and the CDC/National Center for Infectious Diseases' home page on the Internet at <http://www.cdc.gov/ncidod/ncid.htm>. Information about cryptosporidiosis is available at <http://www.cdc.gov/ncidod/dpd/list_crp.htm>. WBDOs should be reported to CDC's Division of Parasitic Diseases (telephone [770] 488-7760), and reports may be faxed to (770) 488-7761.

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Glossary

In this glossary, italicized terms that are not names of microorganisms are defined elsewhere in the glossary.

Action level: A specified concentration of a contaminant in water; if this concentration is reached or exceeded, certain actions (e.g., further treatment and monitoring) must be taken to comply with a drinking water regulation.

Boil-water advisory: A statement to the public advising persons to boil tap water before drinking it.

Class: Refer to the Classification of Outbreaks section in the text and to Table 1 for a comprehensive definition.

Coagulation: The process of adding chemicals to water to destabilize charges on naturally occurring particles to facilitate their subsequent aggregation and removal by *flocculation* and/or *filtration*.

Coliforms: All facultative anaerobic, gram-negative, nonsporeforming, rod-shaped bacteria that ferment lactose with gas and acid formation within 48 hours at 35 C.

Community water system: A *public water system* that serves year-round residents of a community, subdivision, or mobile-home park that has ≥15 service connections or an average of ≥25 residents.

Contact time: The length of time water is exposed to a disinfectant (e.g., chlorine contact time).

Cross-connection: Any actual or potential connection between a drinking water supply and a possible source of contamination or pollution (e.g., a waste-water line).

Cyst: The infectious stage of *Giardia lamblia* and some other protozoan parasites that has a protective wall, which facilitates survival in water and other environments.

Disinfection by-products: Chemicals formed in water through reactions between organic matter and disinfectants.

Distribution system: Water pipes, storage reservoirs, tanks, and other means used to deliver drinking water to consumers or to store it before delivery.

Excystation: The release of the internal (i.e., encysted) contents (e.g., trophozoites or sporozoites) from *cysts* or *oocysts*.

Fecal coliforms: Coliforms that grow and produce gas from lactose at 44.5 C in 24 hours.

Filter backwash: The water containing the material obtained by reversing the flow of water through a filter to dislodge the particles that have been retained on it.

Filtration: The process of removing suspended particles from water by passing it through one or more permeable membranes or media of small diameter (e.g., sand, anthracite, or diatomaceous earth).

Finished water: The water (i.e., drinking water) delivered to the *distribution system* after treatment, if any.

Flocculation: The water treatment process after *coagulation* that uses gentle stirring to cause suspended particles to form larger, aggregated masses (floc). The aggregates are removed from the water by a separation process (e.g., sedimentation, flotation, or *filtration*).

Free, residual chlorine level: The concentration of chlorine in water that is not combined with other constituents and thus serves as an effective disinfectant.

Groundwater system: A system that uses water extracted from the ground (i.e., a well or spring).

Heterotrophic microflora: Microorganisms that use organic material for energy and growth.

Individual water system: A small water system, not owned or operated by a *water utility*, that serves <15 residences or farms that do not have access to a *public water system*.

Maximum-contaminant level: The maximum permissible concentration (level) of a contaminant in water supplied to any user of a *public water system*.

Nephelometric turbidity units: The units in which the *turbidity* of a sample of water is measured when the degree to which light is scattered is assessed with a nephelometric turbidimeter.

Noncommunity water system: A *public water system* that a) serves an institution, industry, camp, park, hotel, or business that is used by the public for ≥60 days per year; b) has ≥15 service connections or serves an average of ≥25 persons; and c) is not a *community water system.*

Oocyst: The infectious stage of *Cryptosporidium parvum* and some other coccidian parasites that has a protective wall, which facilitates survival in water and other environments.

Public water system: A system, classified as either a *community* or a *noncommunity water system*, that provides piped water to the public for human consumption and is regulated under the Safe Drinking Water Act.

Raw water: Surface water or groundwater that has not been treated in any way.

Reverse osmosis: A filtration process that removes dissolved salts and metallic ions from water by forcing it through a semipermeable membrane. This process is also highly effective in removing microbes from water.

Siphonage: A reversal of the normal flow of water or other liquid caused by a negative-pressure gradient (e.g., within a water system).

Source water: Untreated water (i.e., raw water) used to produce drinking water.

Surface water: The water in lakes, rivers, reservoirs, and oceans.

Total coliforms: Nonfecal and fecal *coliforms* that are detected with a standard test.

Turbidity: The quality (e.g., of water) of having suspended matter (e.g., clay, silt, or plankton), which results in loss of clarity or transparency.

Untreated water: Refer to *raw water*.

Water-quality indicator: A microbial, chemical, or physical parameter that indicates the potential risk for infectious diseases associated with use of the water for drinking, bathing, or recreational purposes. The best indicator is one whose density or concentration correlates best with health hazards associated with a given type of hazard or pollution.

Water utility: A water provider that distributes drinking water to a community through a network of pipes.

Watershed: An area from which water drains to a single point; in a natural basin, the area contributing flow (i.e., water) to a given place or a given point on a stream.

Watershed-control program: A program to protect a watershed from sources of contamination or pollution.

FIGURE 1. Number of waterborne-disease outbreaks, by type of water, etiologic agent or illness, and month — United States, 1995–1996 (N = 59)

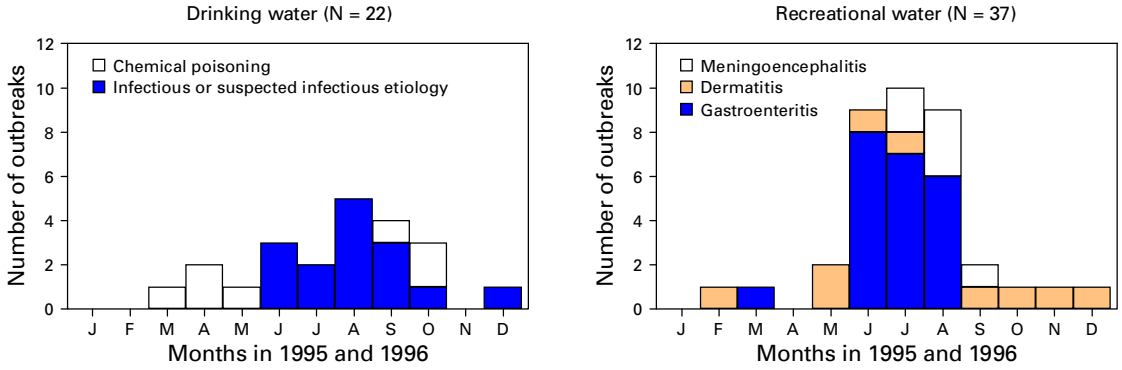


FIGURE 2. Waterborne-disease outbreaks associated with drinking water, by etiologic agent, water system, water source, and deficiency — United States, 1995–1996 (N = 22)

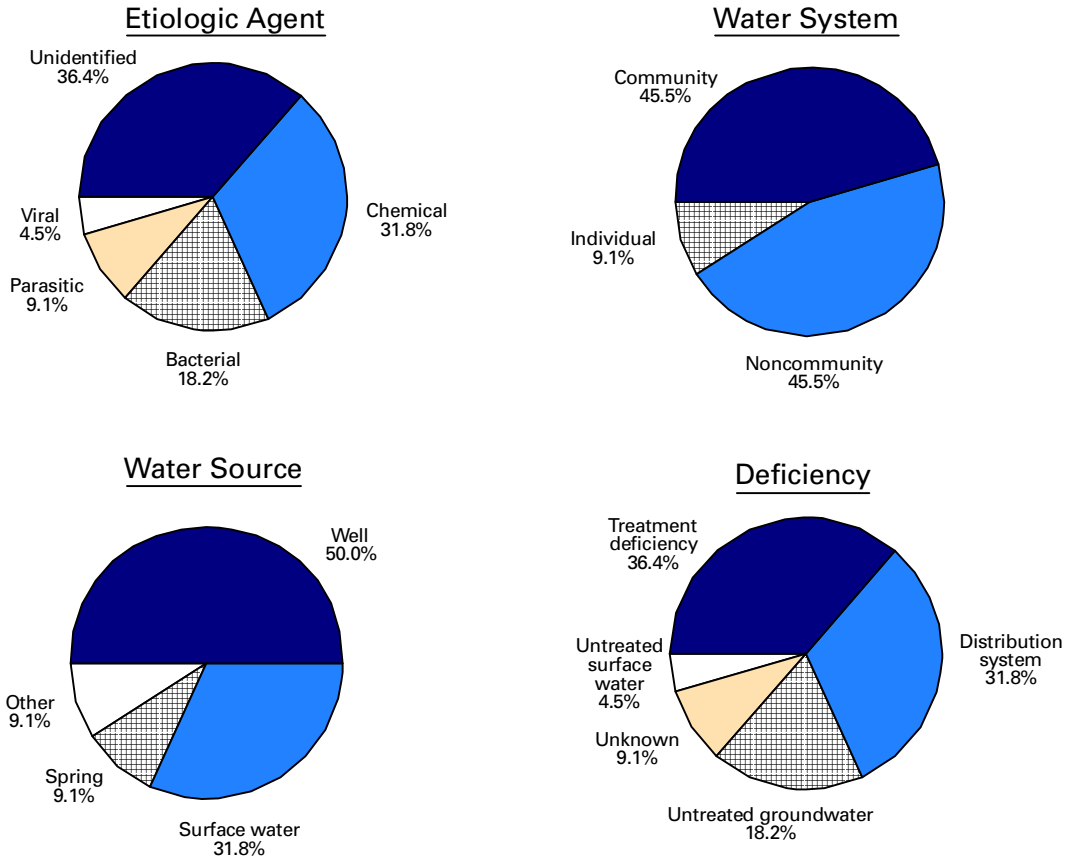
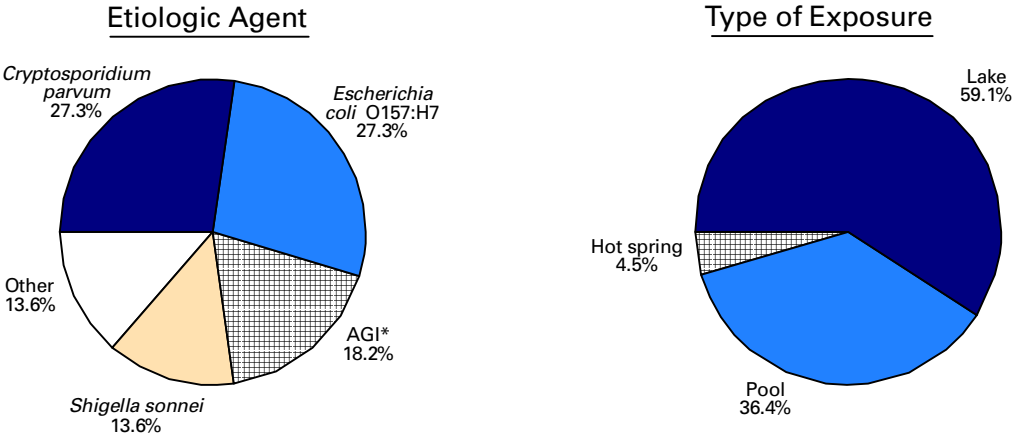
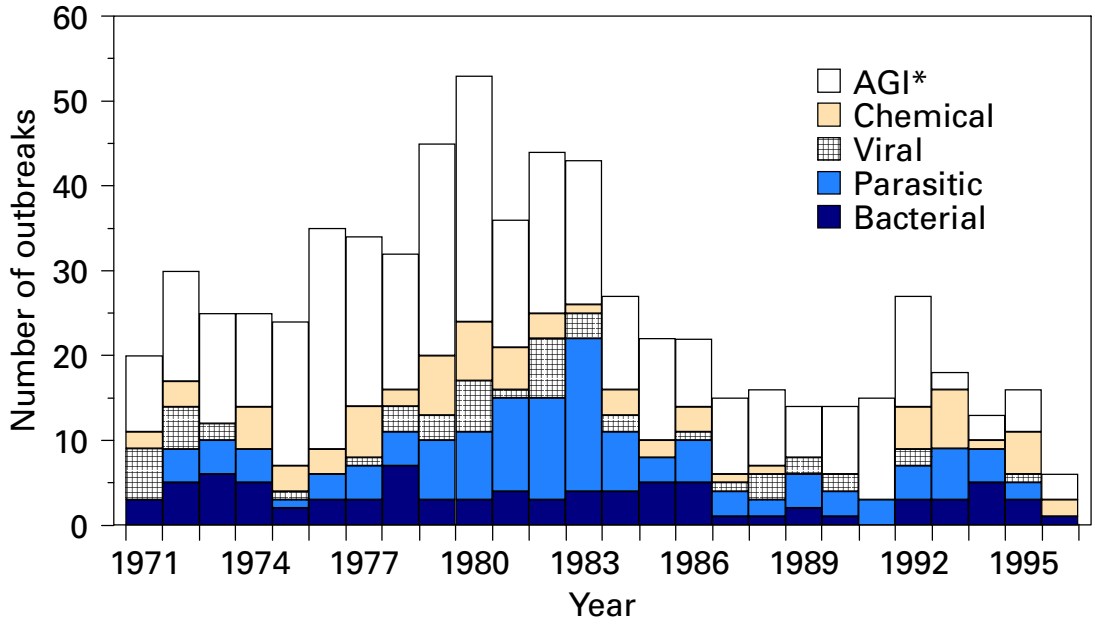


FIGURE 3. Waterborne-disease outbreaks of gastroenteritis associated with recreational water, by etiologic agent and type of exposure — United States, 1995–1996 (N = 22)



*Acute gastrointestinal illness of unknown etiology.

FIGURE 4. Number of waterborne-disease outbreaks associated with drinking water, by year and etiologic agent — United States, 1971–1996 (N = 674)



*Acute gastrointestinal illness of unknown etiology.

FIGURE 5. Number of waterborne-disease outbreaks associated with drinking water, by year and type of water system — United States, 1971–1996 (N = 674)

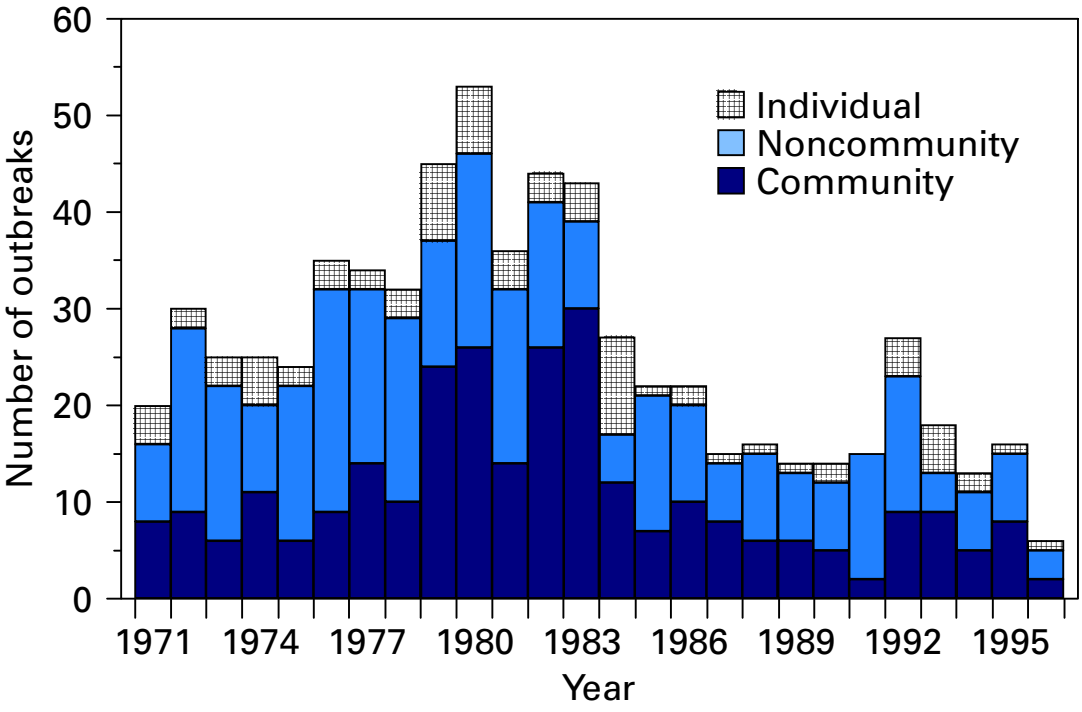


TABLE 1. Classification of investigations of waterborne-disease outbreaks — United States*

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 (e.g., the history that a chlorinator malfunctioned or a water main broke, no detectable free-chlorine residual, or the presence of coliforms in the water)
II	ADEQUATE	NOT PROVIDED OR INADEQUATE (e.g., 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

*Outbreaks of *Pseudomonas* dermatitis and single cases of primary amebic meningoencephalitis or of illness resulting from chemical poisoning are not classified according to this scheme.
†The classification is based on the epidemiologic and water-quality data that were provided on the form.
§Adequate data were provided to implicate water as the source of the outbreak.

TABLE 2. Waterborne-disease outbreaks associated with drinking water — United States, 1995 (N = 16)*

State	Month	Class†	Etiologic agent	No. cases	Type of system§	Deficiency¶	Source	Setting
Alaska	Aug	II	*Giardia lamblia*	10	Ind	1	Surface**	Rural area
Florida	Apr	—††	Chlorine	1	Com	5	Lake	Restaurant
Florida	May	III	Sodium hydroxide	30	Com	3	River	Water utility
Idaho	Aug	I	*Shigella sonnei*	83	NCom	2	Well	Resort
Idaho	Sept	I	AGI§§	18	Com	3	Well	Community
Iowa	Oct	III	Concentrated liquid soap	13	Com	4	Lake	Health-care facility
Minnesota	Jul	I	*Escherichia coli* O157:H7	33	NCom	3	Spring	Camp
Montana	Aug	II	AGI	450	NCom	2	Well	Campground
New York	Dec	I	*G. lamblia*	1,449	Com	3	Lake	Water utility
Oklahoma	Oct	II	*S. sonnei*	10	NCom	3	Well	Store
Pennsylvania	Aug	I	AGI	19	NCom	2	Well	Inn
South Dakota	Jun	I	AGI	48	NCom	2	Well	Camp
Wisconsin	Aug	III	AGI¶¶	26	NCom	3	Well	Restaurant
Wisconsin	Sept	I	Small round structured virus	148	Com	4	Lake	School
Wisconsin	Sept	I	Copper	22	Com	4	Well	Private home
Wisconsin	Oct	I	Copper	15	Com	4	Well	Private home

* Refer to the Methods section for a description of the reporting variables.
† Refer to Table 1 for information concerning the classification of outbreaks.
§ Com = community; NCom = noncommunity; Ind = individual; refer to the Methods section for definitions of the types of water systems.
¶ Refer to the Methods section for the classification of water-system deficiencies.
** Surface water from an unknown source.
†† Not applicable; see Table 1.
§§ AGI = acute gastrointestinal illness of unknown etiology.
¶¶ See text about the possibility that this outbreak was caused by a rotavirus.

TABLE 3. Waterborne-disease outbreaks associated with drinking water — United States, 1996 (N = 6)*

State	Month	Class†	Etiologic agent	No. cases	Type of system§	Deficiency¶	Source	Setting
California	Apr	III	Nitrite	3	Com	4	River	School
California	Sept	I	AGI**	8	Ind	5	Outside tap	Waste-water plant
Idaho	Jul	III	AGI	94	NCom	3	Well	Camp
New Jersey	Mar	I	Nitrite	6	Com	4	Mixed††	Office
New York	Jun	I	*Plesiomonas shigelloides*	60	NCom	3	Spring	Restaurant
Wisconsin	Jun	III	AGI	21	NCom	4	Well	Restaurant

* Refer to the Methods section for a description of the reporting variables.
† Refer to Table 1 for information concerning the classification of outbreaks.
§ Com = community; NCom = noncommunity; Ind = individual; refer to the Methods section for definitions of the types of water systems.
¶ Refer to the Methods section for the classification of water-system deficiencies.
** AGI = acute gastrointestinal illness of unknown etiology.
†† The source was both surface water and groundwater.

TABLE 4. Waterborne-disease outbreaks associated with drinking water, by etiologic agent and type of water system — United States, 1995–1996 (N = 22)*

Etiologic agent	Type of water system†						Total	
	Community		Noncommunity		Individual			
	Outbreaks	Cases	Outbreaks	Cases	Outbreaks	Cases	Outbreaks	Cases
AGI§	1	18	6	658	1	8	8	684
Giardia lamblia	1	1,449	0	0	1	10	2	1,459
Shigella sonnei	0	0	2	93	0	0	2	93
Copper	2	37	0	0	0	0	2	37
Nitrite	2	9	0	0	0	0	2	9
Small round structured virus	1	148	0	0	0	0	1	148
Plesiomonas shigelloides	0	0	1	60	0	0	1	60
Escherichia coli O157:H7	0	0	1	33	0	0	1	33
Sodium hydroxide	1	30	0	0	0	0	1	30
Concentrated liquid soap	1	13	0	0	0	0	1	13
Chlorine	1	1	0	0	0	0	1	1
Total	**10**	**1,705**	**10**	**844**	**2**	**18**	**22**	**2,567**
(Percentage¶)	(45.5)	(66.4)	(45.5)	(32.9)	(9.1)	(0.7)	(100.0)	(100.0)

*Ordered by total number of outbreaks and secondarily by total number of cases.
†Refer to the Methods section for definitions of the types of water systems.
§AGI = acute gastrointestinal illness of unknown etiology.
¶The percentage is based on 22 outbreaks or 2,567 cases.

TABLE 5. Waterborne-disease outbreaks associated with drinking water, by type of deficiency and type of water system — United States, 1995–1996 (N = 22)

Type of deficiency†	Type of water system*						Total	
	Community		Noncommunity		Individual			
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Untreated surface water	0	(0)	0	(0)	1	(50.0)	1	(4.5)
Untreated groundwater	0	(0)	4	(40.0)	0	(0)	4	(18.2)
Inadequate treatment	3	(30.0)	5	(50.0)	0	(0)	8	(36.4)
Distribution system	6	(60.0)	1	(10.0)	0	(0)	7	(31.8)
Miscellaneous or unknown	1	(10.0)	0	(0)	1	(50.0)	2	(9.1)
Total	**10**	**(100.0)**	**10**	**(100.0)**	**2**	**(100.0)**	**22**	**(100.0)**

*Refer to the Methods section for definitions of the types of water systems.
†Refer to the Methods section for the classification of water-system deficiencies.

TABLE 6. Waterborne-disease outbreaks of gastroenteritis and meningoencephalitis associated with recreational water — United States, 1995 (N = 17)

State	Month	Class*	Etiologic agent	Illness	No. cases	Source	Setting
Florida	Aug	—†	*Naegleria fowleri*	Meningoencephalitis	1	Canal	Canal
Georgia	Jul	I	*Cryptosporidium parvum*	Gastroenteritis	5,449	Pool	Water park
Idaho	Mar	I	*Salmonella* serotype Java	Gastroenteritis	3	Pool	Park
Illinois	Jun	I	*Escherichia coli* O157:H7	Gastroenteritis	12	Lake	Beach
Kansas	Jun	III	*C. parvum*	Gastroenteritis	24	Pool	Park
Minnesota	Jun	II	AGI§	Gastroenteritis	12	Lake	Beach
Minnesota	Jul	IV	*E. coli* O157:H7	Gastroenteritis	6	Lake	Beach
Minnesota	Jul	IV	*E. coli* O157:H7	Gastroenteritis	2	Lake	Beach
Nebraska	Jul	IV	*C. parvum*	Gastroenteritis	14	Pool	Water park
Pennsylvania	Aug	I	AGI	Gastroenteritis	17	Lake	Park
Pennsylvania	Aug	III	*Shigella sonnei*	Gastroenteritis	70	Lake	Beach
Texas	Jul	—	*N. fowleri*	Meningoencephalitis	1	River	River
Texas	Jul	—	*N. fowleri*	Meningoencephalitis	1	Pond	Pond
Texas	Aug	—	*N. fowleri*	Meningoencephalitis	1	Lake	Lake
Texas	Aug	—	*N. fowleri*	Meningoencephalitis	1	Lake	Lake
Texas	Sept	—	*N. fowleri*	Meningoencephalitis	1	Lake	Lake
Wisconsin	Jun	III	*E. coli* O157:H7	Gastroenteritis	8	Lake	Beach

*Refer to Table 1 for information concerning the classification of outbreaks.
†Not applicable; see Table 1.
§AGI = acute gastrointestinal illness of unknown etiology.

TABLE 7. Waterborne-disease outbreaks of gastroenteritis associated with recreational water — United States, 1996 (N = 11)

State	Month	Class*	Etiologic agent	No. cases	Source	Setting
California	Aug	II	*Cryptosporidium parvum*	3,000	Pool	Amusement park
Colorado	Jul	I	*Shigella sonnei*	39	Lake	Recreation area
Colorado	Jul	I	*S. sonnei*	81	Lake	Recreation area
Florida	Jun	I	*C. parvum*	22	Pool	Community
Florida	Jun	I	*Giardia lamblia*†	77	Pool	Community
Georgia	Jul	I	*Escherichia coli* O157:H7	18	Pool	Mobile-home park
Idaho	Jun	II	Norwalk	55	Hot spring	Camp
Indiana	Aug	IV	*C. parvum*	3	Lake	Beach
Indiana	Aug	IV	AGI§	4	Lake	Beach
Minnesota	Jun	IV	*E. coli* O157:H7	6	Lake	Beach
Oregon	Aug	IV	AGI	32	Lake	Camp

*Refer to Table 1 for information concerning the classification of outbreaks.
†Sixty persons had stool specimens that tested positive only for *G. lamblia;* 17 had specimens that tested positive only for *C. parvum;* and eight had specimens that were positive for both organisms.
§AGI = acute gastrointestinal illness of unknown etiology.

TABLE 8. Waterborne-disease outbreaks of dermatitis associated with recreational water — United States, 1995–1996 (N = 9)

State	Year	Month	Class*	Etiologic agent	No. cases	Source	Setting
Maine	1995	Dec	—†	*Pseudomonas aeruginosa*	10	Hot tub	Hotel
Minnesota	1995	May	—	*P. aeruginosa*	4	Hot tub	Hotel
Minnesota	1995	Oct	—	*P. aeruginosa*	6	Hot tub	Hotel
New Mexico	1995	Sept	—	*P. aeruginosa*	4	Hot tub	Apartment complex
Oregon	1996	Jun	III	*Schistosoma* sp.	71	Lake	Beach
Oregon	1996	Jul	III	*Schistosoma* sp.	50	Lake	Beach
Washington	1995	Feb	—	*P. aeruginosa*	2	Hot tub	Resort
Washington	1995	May	—	*P. aeruginosa*	5	Hot tub	Spa
Washington	1996	Nov	—	*P. aeruginosa*	17	Hot tub	Motel

*Refer to Table 1 for information concerning the classification of outbreaks.
†Not applicable; see Table 1.

TABLE 9. Waterborne-disease outbreaks of gastroenteritis associated with drinking or recreational water that were not included in the previous surveillance summaries — United States, 1994 (N = 3)*

State	Month	Year	Exposure†	Class§	Etiologic agent	No. cases	Type of system¶	Deficiency**	Source	Setting
Florida	Jun	1994	Wid	III	*Escherichia coli* O157:H7	2	NCom	3	Well	Mobile-home park
Florida	Jul	1994	Rec	I	AGI††	12	—§§	—§§	Lake	Park
New Jersey	Jun	1994	Rec	III	*Shigella sonnei*	300	—	—	Reservoir	Park

* Refer to the Methods section for a description of the reporting variables.
† Rec = recreational water; Wid = water intended for drinking.
§ Refer to Table 1 for information concerning the classification of outbreaks.
¶ Ncom = noncommunity; refer to the Methods section for definitions of the types of water systems.
** Refer to the Methods section for the classification of water-system deficiencies.
†† AGI = acute gastrointestinal illness of unknown etiology.
§§ Recreational water outbreaks are not categorized by type of system or water deficiency.

Behavioral Risk Factor Surveillance System
State & Territorial Coordinators

State/Territory	Coordinator
Alabama	Janice Cook, MBA
Alaska	Patricia Owen
Arizona	Brian Bender, MBA
Arkansas	John Senner, PhD
California	Bonnie Davis, PhD
Colorado	Marilyn Leff, MSPH
Connecticut	Mary Adams, MPH
Delaware	Fred Breukelman
District of Columbia	Cynthia Mitchell
Florida	Scott Hoecherl
Georgia	Linda Martin, MPH
Guam	Cynthia Naval
Hawaii	Alvin T. Onaka, PhD
Idaho	James Aydelotte
Illinois	Bruce Steiner, MS
Indiana	Kathy Horvath
Iowa	Andrew Wineski
Kansas	Michael Perry
Kentucky	Karen Asher
Louisiana	Ruth Jiles, PhD
Maine	Dorean Maines
Maryland	Alyse Weinstein, MA
Massachusetts	Daniel Brooks, MPH
Michigan	Harry McGee, MPH
Minnesota	Nagi Salem, PhD
Mississippi	Dick Johnson
Missouri	Theophile Murayi, PhD
Montana	Pete Feigley, PhD
Nebraska	Martha Metroka
Nevada	Emil DeJan, MPH
New Hampshire	Larry Powers, MA
New Jersey	Georgette Boeselager, MS
New Mexico	Wayne Honey, MPH
New York	Thomas A. Melnik, DrPH
North Carolina	Kristi Passaro, PhD
North Dakota	Jill Kaske, MPH
Ohio	Patricia Pullen
Oklahoma	Neil Hann, MPH
Oregon	Joyce Grant-Worley, MS
Pennsylvania	Linda Mann
Puerto Rico	Yvette Cintron, MPH
Rhode Island	Jana Hesser, PhD
South Carolina	Dennis Shepard
South Dakota	Mark Gildemaster
Tennessee	David Ridings
Texas	Ken Condon
Utah	Rebecca Giles
Vermont	Cheryl Roe, MS
Virgin Islands	Julia Sheen
Virginia	Linda Redman, MPH
Washington	Katrina Wynkoop-Simmons, PhD
West Virginia	Fred King
Wisconsin	Pamela Imm, MS
Wyoming	Menlo Futa, MA

Cardiovascular Disease Risk Factors and Preventive Practices Among Adults — United States, 1994: A Behavioral Risk Factor Atlas

Robert A. Hahn, Ph.D., M.P.H.[1]
Gregory W. Heath, D.H.Sc., M.P.H.[2]
Man-Huei Chang, M.P.H.[1]
Behavioral Risk Factor Surveillance System State Coordinators
[1]*Division of Prevention Research and Analytic Methods*
Epidemiology Program Office
[2]*Division of Adult and Community Health*
National Center for Chronic Disease Prevention and Health Promotion

Abstract

Problem/Conditions: Cardiovascular disease (CVD), including coronary heart disease (CHD) and stroke, is the leading cause of death in the United States, and state rates of CVD vary by state and by region of the country. Several behavioral risk factors (i.e., overweight, physical inactivity, smoking, hypertension, and diabetes mellitus) and preventive practices (i.e., weight loss and smoking cessation) are associated with the development of CVD and also vary geographically. This summary displays and analyzes geographic variation in the prevalences of selected CVD risk factors.

Reporting Period: 1994 (1992 for prevalence of hypertension).

Description of System: The Behavioral Risk Factor Surveillance System (BRFSS) is a state-based random-digit–dialing telephone survey of noninstitutionalized adults aged ≥18 years; 50 states and the District of Columbia participated in BRFSS in 1994, and 48 states and the District of Columbia participated in 1992.

Methods: Several different analyses were conducted: a) analysis of state risk factor and preventive practice prevalences by sex and race (i.e., black and white); b) mapping; c) cluster analysis; d) correlations of state prevalence rates by sex and race; and e) regression of state risk factor prevalences on state CHD and stroke mortality rates.

Results: Mapping the prevalence of selected CVD risk factors and preventive health practices indicates substantial geographic variation for black and white men and women, as confirmed by cluster analysis. Data for blacks are limited by small sample size, especially in western states. Geographic clustering is found for physical inactivity, smoking, and risk factor combinations. Risk factor prevalences are generally lower in the West and higher in the East. White men and white women are more similar in state risk factor rates than other race-sex pairs; white women and black women ranked second in similarity. State prevalences of physical inactivity and hypertension are strongly associated with state mortality rates of CVD.

Interpretation: Geographic patterns of risk factor prevalence suggest the presence (or absence) of sociocultural environments that promote (or inhibit) the given risk factor or preventive behavior. Because the risk factors examined in this summary are associated with CVD, further exploration of the reasons underlying observed geographic patterns might be useful. The BRFSS will continue to provide geographic data about

cardiovascular health behaviors with a possible emphasis on more data-based small-area analyses and mapping. This will permit states to more adequately monitor trends that affect the burden of CVD in their regions and the United States. Mapping also facilitates the exploration of patterns of morbidity, health-care use, and mortality, as well as the epidemiology of risk factors. Finally, by identifying those segments of the population with high levels of these risk factors and lower levels of the preventive health practices, public health personnel can better allocate resources and target intervention efforts for the prevention of CVD.

INTRODUCTION

Although age-adjusted cardiovascular mortality declined by 58% from 1950 through 1995 (*1*), cardiovascular disease (CVD), including stroke and coronary heart disease (CHD), remains a major public health problem in the United States. In 1995, CVD was the principal diagnosis in 5 million (16.2%) hospital patient discharge records in the United States (*2*) and was the leading cause of death, accounting for 38.7% of all deaths in the United States (*1*).

The prevalence, mortality, and health-care use associated with CVD in the United States vary substantially by geographic region and state (*2,3,4*). In 1994, stroke was 51% more prevalent in the South than in the Northeast, and CHD was 29% more prevalent in the South than in the West (*4*). In 1994, the ratios of the highest to the lowest age-adjusted state mortality rates for CHD and stroke in the United States were 4.1 and 4.4, respectively (*5*). Health-care use also varies substantially by region. In 1995, rates of hospital discharge for CVD were between 40% and 69% times greater in the Northeast, South, and Midwest than in the West (*2*). Geographic variations in CVD prevalence, mortality, and health-care use might correspond to differences in a) demographic or risk behavior profiles (e.g., smoking [*6*], physical inactivity [*7*], or risk factor combinations [*8*] among state residents); b) physical environment (e.g., excessive heat [9] and air pollution [10]); and c) social environment (e.g., laws taxing cigarettes or restricting cigarette use) (*11,12*).

This atlas displays distribution of major behavioral risk factors and preventive practices for CVD among black and white men and women in the 50 states and the District of Columbia. The atlas presents maps for five risk factors (i.e., overweight, physical inactivity, smoking, hypertension, and diabetes mellitus), risk factor combinations, and two preventive practices (i.e., weight loss and smoking cessation). Racial differences in risk factor prevalence are examined to facilitate exploration of well-recognized racial differences in health status. Only blacks and whites are examined in this analysis because of inadequate sample sizes in the data source for other populations. In this summary, we review the association of known behavioral risk factors and preventive practices with CVD and specify the criteria used to assess each risk factor analyzed. We then analyze the geographic clustering of CVD risk factors among states and examine the association of state risk factor prevalence rates with state rates of stroke mortality and heart disease mortality. Use of this atlas might facilitate the exploration of geographic patterns of CVD and of the risk factors as well. The atlas might also indicate the need for interventions to reduce cardiovascular risk factors in specific regions and enhance analysis of trends and evaluation of interventions.

Risk Factors

CHD risk factors analyzed in this report have been chosen for the reasons discussed in the following sections.

Overweight

Overweight is associated with high rates of CVD deaths, especially sudden death among men and congestive heart failure among women (*13*). The high death rate might occur largely as a consequence of the influence of overweight on blood pressure, blood lipid levels, and the onset of diabetes (*13*); however, a report from the Framingham Study indicates that overweight is also an independent risk factor for CVD (*14*). With rare exceptions, overweight develops from eating too much and exercising too little. The prevalence of overweight has increased substantially in the U.S. population during the last 10 years (*15*).

Physical Inactivity

A review of 43 epidemiologic studies in 1987 indicated that physical activity reduces the risk of CHD (*16*). The relative risk for CHD associated with physical inactivity is approximately 1.9, slightly lower than the relative risks associated with increased systolic blood pressure (2.1), cigarette smoking (2.5), and elevated serum cholesterol levels (2.4) (*17*). Several studies indicate that endurance exercise training among patients with documented CHD is associated with reduced morbidity and mortality (*18,19*) and that physical activity might improve the likelihood of survival from a myocardial infarction (i.e., heart attack) (*20*). In addition, evidence documents an association between regular, moderate-intensity physical activity and the lowering of several other risk factors for CVD, including blood lipid levels, resting blood pressure among persons with borderline hypertension, body composition and overweight, and glucose tolerance and insulin sensitivity (*21*).

Smoking

Evidence indicating cigarette smoking as a risk factor for CVD is substantial (*12*). Overall, smokers have a 70% greater level of CVD risk than nonsmokers; persons who smoke ≥2 packs of cigarettes per day have a two- to threefold greater risk for CVD (*17*). The risk for CVD also increases with greater depth of inhalation and with increasing years of smoking, although persons who stop smoking eventually reduce their risk for CVD to a level approaching that of nonsmokers (22). Cigarette smoking has been reported to act synergistically with other known risk factors for CVD (*23*).

Hypertension

High blood pressure is another major risk factor for CVD (*24*). Some evidence documents that blood pressure-related risk for CVD increases continuously from lowest to highest values for either systolic or diastolic blood pressure (*25*). Elevated blood pressure is often associated with other well-known risk factors, including dietary intake, elevated blood lipid levels, obesity, smoking, diabetes mellitus, and physical inactivity (*24*). The prevalence of hypertension has declined substantially in the last 20 years (*26*).

Diabetes Mellitus

The glucose intolerance that accompanies diabetes mellitus is a direct effect of overweight and is often associated with hypertriglyceridemia, hypertension, elevated LDL cholesterol, and depressed HDL (*27*). Some evidence documents that diabetes mellitus has a vasculotoxic effect, which is greatest for occlusive peripheral vascular disease; however, CHD and stroke are its most common manifestations (*27*). The risk for CVD is three times as high among diabetic women as it is among women without diabetes mellitus. Similarly, the risk for CVD is twice as high among diabetic men as it is among men without diabetes mellitus (*27*).

Risk Factor Combinations

Many CVD risk factors interact physiologically in the etiology of CVD (*28,29*). Persons with risk factor combinations are at an increased risk for CVD. Obesity is an example of a risk factor for CHD that influences other risk factors, including hyperlipidemia, hypertension, and diabetes mellitus. Physical inactivity has been related to obesity, lipid abnormalities, hypertension, and diabetes mellitus (*21,30*).

Preventive Practices

Several preventices practices have been demonstrated to reduce the risk of CHD. The preventive practices analyzed in this report have been chosen for the reasons discussed in the following sections.

Weight Loss

Weight control is a first step in the control of mild hypertension, hyperlipidemia, and impaired glucose tolerance and might eliminate the necessity of lifelong drug therapy for these conditions (*13*). Efforts to control weight generally have not been effective (*31*); however, researchers have demonstrated recently the effectiveness of combined programs of behavior modification of diet and exercise (*31,32*). Therapeutic approaches to weight control that emphasize increased physical activity have other benefits in addition to increasing caloric expenditure (*16*).

Smoking Cessation

Persons who stop smoking, especially before age 50 years, live longer than those who continue to smoke. After 15 years of abstinence from smoking, the risk for CVD approaches that of persons who have never smoked. Among persons with previously diagnosed myocardial infarction or stroke, smoking cessation reduces the risk for recurrent heart attack and death from stroke by 50% (*22*).

METHODS

Sampling

We used the Behavioral Risk Factor Surveillance System (BRFSS) data from 1992 and 1994. Data for 1992 were used to assess the prevalence of hypertension because data on hypertension in many states were not available in 1994. BRFSS data for 1995

Definitions of Risk Factors and Preventive Health Practices

- **Overweight**: Body mass index (BMI = weight [kg]/height [m^2]) ≥27.8 for men and ≥27.3 for women. These values approximate the sex specific 85th percentile of BMI that was estimated from NHANES II (*1*) for persons aged 20–29 years in the United States (*Healthy People 2000*, objective 2.3 [*33*]).
- **Physical Inactivity**: No reported exercise, recreation, or physical activities (other than regular job duties) during the previous month (*Healthy People 2000*, objective 1.5).
- **Smoking**: Current regular use of cigarettes by persons who have ever smoked at least 100 cigarettes (*Healthy People 2000*, objective 3.4).
- **Hypertension**: Survey participants having ever been told by a health professional that they have high blood pressure (*Healthy People 2000*, objective 15.5).
- **Diabetes Mellitus**: Survey participants having ever been told by a physician that they have diabetes (*Healthy People 2000*, objective 17.11).
- **Risk Factor Combinations**: Survey participants having ≥2 of the following risk factors: overweight, physical inactivity, smoking, and diabetes mellitus. Hypertension is not included because information on this risk factor was not available in 1994, the year for which risk factor combinations are examined.
- **Weight Loss**: Survey participants trying to lose or maintain or keep from gaining weight and who are either eating fewer calories or eating less fat or using physical activity or exercise to maintain, lose, or keep from gaining weight (*Healthy People 2000*, objective 2.7).
- **Smoking Cessation**: Survey participants ever having smoked 100 cigarettes and having quit smoking for ≥12 months (*Healthy People 2000*, objective 3.6).

were not used because they did not include information on physical activity. Arkansas and Wyoming did not participate in BRFSS in 1992; all 50 states and the District of Columbia participated in 1994. Using random-digit–dialing telephone survey techniques, participating states select a probability sample of their noninstitutionalized adult population (aged ≥18 years) with telephones. The Waksberg method (*34*), a multistage cluster-sampling design, was used in most states (37 states in 1994), whereas other states have chosen different sampling methods (e.g., simple-random or stratified sample designs) to meet their special needs. The standard BRFSS questionnaire includes questions from previously conducted national surveys (e.g., Health Promotion Disease Prevention Supplement to the National Health Interview Survey [*35*]). Modules of questions on additional topics are developed by CDC and added at the discretion of each state (*36*). BRFSS also contains basic demographic and socioeconomic information (e.g., age, race/ethnicity, family income, and educational attainment).

Analysis

This summary provides state risk factor prevalences estimated from the survey participant's probability of selection in each state, weighted by the distribution of the state population by age, race, and sex. Prevalence estimates for each risk factor include only participants who gave specific responses for this risk factor. State risk factor prevalence rates were not reported if the sample size for a given population (e.g., black women) was <50. Data were drawn from the BRFSS electronic source by using Statistical Analyses Software (SAS®) (37). *Epi-Info* (*38*) and *Epi-Map* (*39*) were applied to produce maps of the prevalence of risk factors and preventive practices in the 50 states and the District of Columbia. We do not present age-standardized state prevalences because we were interested in the burden of risk behavior in states. State prevalences were stratified into quartiles.

We examined three questions regarding the geographic distribution of risk factors. First, does a geographic pattern of the distribution of risk factors exist (e.g., higher rates in one region of the country than in another)? In addition to visual review, we used the Ohno method (*40*) in CLUSTER Software (*41*) to detect the presence of clustering of state prevalence rates of the risk factors and preventive practices for each race and sex group. The Ohno method uses a Chi-square test to determine whether the observed number of bordering states with similar rates is more than expected to occur by chance under the null hypothesis of no geographic clustering. Similar rates are defined as rates within the same quartile. We omitted from the cluster analysis noncontiguous states (i.e., Alaska and Hawaii) and states with unreliable prevalence rates (i.e., sample sizes <50).

Second, does race, sex, or some combination of these account for greater similarity in the distribution of state prevalence rates (e.g., are state prevalence rates for black women closer to those for white women or to those for black men)? For each risk factor and preventive practice and for all risk factors combined, we correlated state prevalence rates using Pearson correlation coefficients for each race-sex pair using the SAS® software. The analysis excluded states without prevalence estimates. We compared the magnitude of correlation for each race-sex pair among statistically significant correlations.

Third, does the distribution of risk factor prevalences among states correspond to state CVD mortality patterns? For each sex-race group, we conducted linear multiple regression analyses in which 1994 state mortality rates for all ages were the dependent variables and the independent variables were state behavioral risk factor prevalences. For heart disease, the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes were 390–8, 402, 404–29; for stroke, the ICD-9-CM codes were 430–8. We used mortality data from CDC's WONDER data system (*42*) and the Shapiro-Wilks statistic in SAS to assess the normality of mortality rates for regression analyses. Because state risk factor prevalences were not age-adjusted, we did not age-adjust mortality rates. We began with a model that included all of the risk factors and eliminated the risk factor with the highest p value in each succeeding model, until all p values were <0.05 in the final model. State risk factor prevalence rates were excluded from this analysis if the sample size for a given population was <50.

RESULTS

BRFSS sample sizes used for state prevalence estimates varied by risk factor. Excluding states for which race-sex group sample sizes were <50, the study sample included a maximum of 38,089 white men; 53,205 white women; 3,028 black men; and 5,989 black women.

Because of small sample sizes, 28–29 states among black men and 19–21 states among black women were excluded from analysis for the five study risk factors. The states with missing information among blacks varied by risk factor; in general, prevalence information was not available for many western and northeastern states. All states had sufficient sample sizes to calculate reliable prevalence estimates among whites for all risk factors, except as noted for hypertension, for which information was not available for Wyoming and Arkansas.

Prevalence of Risk Factor and Preventive Measures

Overweight

Median state prevalences of overweight are higher for blacks than for whites, higher for white men (28.5%) than for white women (24.9%), and higher for black women (42.6%) than for black men (37.0%) (Table 1). No clear geographic pattern of the prevalence of overweight among white men or black men exists (Figure 1a and c). Among white and black women, the higher prevalence rates are found in the northeastern quadrant of the nation (Figure 1b and d). No statistically determined clusters for this risk factor exist among whites, for whom clusters could be assessed.

Physical Inactivity

Median state prevalences of physical inactivity are higher for blacks than for whites and higher for women than for men (Table 1). State prevalences of physical inactivity among white men are generally higher in the East and lower in the West (Figure 2a). A similar pattern among white women is evident (Figure 2b) and is confirmed by significant clustering. Available data for blacks suggest that state prevalences of physical inactivity are generally higher in the South and lower in the North (Figures 2c and d); however, patterns are not clear because of insufficient data from several states.

Smoking

Median state prevalences of smoking are similar for blacks and whites but higher for men than for women (Table 1). Among white men, state prevalences of smoking are generally higher in the East and lower in the West, as confirmed by significant clustering (Figure 3a). Among white women, the geographic pattern is similar, but not as clearly defined as for white men (Figure 3b). Among black men, no clear geographic pattern exists (Figure 3c). Among black women, prevalences are generally higher in northern states and lower in southern states (Figure 3d).

Hypertension

Median state prevalences of hypertension are higher for blacks than for whites and higher for women than for men (Table 1). State prevalences of hypertension among white men are generally higher in the northeastern quadrant and lower in the West

(with the exceptions of Nevada and Arizona) (Figure 4a). No clear geographic patterns exist among white women, black men, or black women (Figure 4b, 4c, and 4d). No statistically determined clusters exist for this risk factor among whites, for whom clusters could be assessed.

Diabetes Mellitus

Median state prevalences of diabetes mellitus are higher for blacks than for whites, slightly higher for white men (4.0%) than for white women (3.7%), and slightly higher for black women (6.8%) than for black men (6.1%) (Table 1). State prevalences of diabetes mellitus among white men are generally higher in southern states (Figure 5a). State prevalences of diabetes mellitus among white women are generally higher in a band of states from Connecticut, Rhode Island, and Michigan to Texas, and lower in the West (Figure 5b). No apparent geographic pattern of state prevalences of diabetes mellitus exists among black men or black women (Figure 5c and d). No statistically determined clusters for this risk factor exist among whites, for whom clusters could be assessed.

Risk Factor Combinations

Median state prevalences of risk factor combinations are higher for blacks than for whites, higher for white men than for white women, and higher for black women than for black men (Table 1). Among the study sample for 1994, 40.1% have no risk factors, 38.2% have just one, 18.0% have just two, 3.4% have just three, and 0.2% have four risk factors. State prevalences of risk factor combinations among white men and white women are generally higher in the East and lower in the West; geographic patterns for both white men and white women are confirmed by significant clustering (Figure 6a and b). No clear geographic pattern of the state prevalences of risk factor combinations exists among black men and black women (Figure 6c and d).

Weight Loss

Median state prevalences of weight-loss practices are higher for whites than for blacks and higher for women than for men (Table 1). Prevalence rates are generally higher in the West for white men and white women (Figure 7a and b). No apparent geographic pattern of state prevalences of weight-loss practices exists among black men and women (Figure 7c and d). No statistically determined clusters for this risk factor exist among whites, for whom clusters could be assessed.

Smoking Cessation

Median state prevalences of smoking cessation are higher for whites than for blacks and higher for men than for women (Table 1). No apparent geographic pattern of state prevalences of smoking cessation exists among white men (Figure 8a). Among white women, state prevalences of smoking cessation are highest in coastal and border states (Figure 8b). No apparent geographic pattern of state prevalences of smoking cessation exists among black men and women (Figure 8c and d). No statistically determined clusters exist for this preventive practice among whites, for whom clusters could be assessed.

Associations Among State Risk Factor Prevalences by Race and Sex

Because of the small sample sizes of blacks in many western and northeastern states, comparison of similarity in state prevalences among white men, white women, black men, and black women is possible principally for the eastern states. Comparison of correlations of state risk factor profiles among all race-sex pairs indicates that white men and white women are most similar to each other; correlations of prevalence rates for all risk factors among whites are statistically significant ($p \leq 0.05$). Among significant correlations, second in similarity among race-sex pairs are white women and black women; this correlation is significant for overweight, physical inactivity, hypertension, weight loss, and smoking cessation — but not for diabetes mellitus. For diabetes mellitus, correlations are highest for the black men and white men pair. Among race-sex pairs, black men and black women are significantly correlated only for physical inactivity and weight loss; however, these correlations are lower (i.e., fourth and sixth out of six pairs, respectively) than those of most other race-sex pairs.

Associations Between State Risk Factor Prevalences and State CVD Mortality

Regression analysis of state risk factor prevalences on state rates of mortality from CHD and stroke among the four race-sex pairs are limited to states with sufficient sample sizes; thus, regression analyses of risk factors and mortality among blacks are restricted to a relatively small number of states. Mortality distributions are not significantly different from normal for CHD in any race-sex group and are significantly different from normal for stroke mortality only among black men. Thus, we do not transform the dependent variable in regression analyses. Analysis indicates that state rates of physical inactivity are predictive of state CHD mortality rates among each of the four race-sex groups. In addition, state rates of diabetes are predictive of state CHD mortality rates among white women, and state rates of hypertension are predictive of state CHD mortality rates among white men. State rates of hypertension are predictive of stroke mortality among white men and women, and state rates of physical inactivity are predictive of state rates of stroke mortality among white women. No

Regression models* of effects of state risk factor prevalences on CHD and stroke among adults — United States, 1994

Outcome
CHD Mortality
White men: CHD = –56.74 + 317.97 * INACTIVE + 1354.73 * HYPERTENSION
White women: CHD = 56.50 + 476.93 * INACTIVE + 2114.71 * DIABETES
Black men: CHD = 111.06 + 418.70 * INACTIVE
Black women: CHD = 41.46 + 424.76 * INACTIVE
Stroke mortality
White men: Stroke=12.87 + 37.94 * INACTIVE + 126.88 * HYPERTENSION
White women: Stroke= 12.76 + 290.24 * HYPERTENSION

*Included models with $p < 0.05$ for ≥ 1 risk factors.

risk factors are predictive of stroke mortality among black men or black women. State rates of overweight and smoking are not predictive of mortality from either cause.

DISCUSSION

This atlas of state prevalences of cardiovascular risk factors and preventive practices for white and black men and women in the United States indicates little geographic patterning for some risk factors and preventive practices (e.g., weight loss and smoking cessation), moderate patterning for others (e.g., diabetes mellitus and hypertension), and marked patterning for others (e.g., physical inactivity, smoking, and risk factor combinations). The presence (or absence) of geographic patterning among white men and white women is confirmed by cluster analysis.

The atlas indicates an eastern concentration of high state prevalences of several CVD risk factors (i.e., physical inactivity, smoking, and risk factor combinations) and low state prevalences of two preventive practices (i.e., weight loss and smoking cessation). Thus, risk factor prevalences are lower and preventive practice prevalences are higher among western states. Among blacks, such comparison is not possible because of inadequate information on most western states.

Correlation of state risk factor prevalences for four race-sex pairs indicates that among six possible comparisons, a) white men and white women are most similar in state risk factor profiles; b) white women and black women are second in similarity; and c) black men and black women are among the least similar. This finding suggests that neither race nor sex is uniformly a predictor of similarity in behavior among racial sex groups. The targeting of prevention messages might require specification of racial sex group combinations. These associations might be confounded (e.g., by age or socioeconomic position). For example, the proportion of persons aged ≥65 years in the West is smaller than proportions in the Midwest, Northeast, and South (*43*), and greater age might be associated with greater risk factor prevalence. However, comparison of state prevalence maps *unadjusted* for age (in this summary) and *adjusted* for age (not presented in this summary) reveals modified rates for some states with large proportions of younger or older populations (e.g., Alaska and Florida) but indicates little difference in overall national patterns. Regarding socioeconomic position, western states do not systematically exceed the national median of household income (*43*). However, western states do have higher proportions of the population who have completed high school (*44*). A plausible explanation of East-West prevalence differences is that the East and the West differ in their sociocultural environments related to risk factor avoidance, health promotion, and preventive behavior; however, no evidence supports this hypothesis.

Regression analysis indicates that state prevalences of some cardiovascular risk factors, particularly physical inactivity and hypertension, are predictive of mortality from CHD and stroke. Among whites, risk factor maps correspond to detailed maps of health-service areas with high mortality rates for CHD along the Mississippi and Ohio valleys; for blacks, insufficient information on risk factor prevalences in many states prevents comparison (*3*). Except for physical inactivity, little apparent correspondence exists between high state risk factor prevalences and high stroke mortality as depicted in detailed mortality maps. Although stroke mortality is highest in the Southeast and southeastern quadrant for all race-sex groups, high risk factor prevalences

other than physical inactivity are not predominantly concentrated in this region (*3*). Findings here differ from conclusions of a recent analysis of BRFSS data for 1991–1992 (*8*). However, our study included all persons aged ≥18 years (rather than only those aged 45–74 years), and our analysis was stratified by race as well as sex.

The data in this report indicate that, among blacks, higher prevalences of CHD risk factors (except for smoking) and lower prevalences of preventive practices exist. This finding is consistent with higher national rates of CHD morbidity and mortality among blacks. These risk factors can be changed or managed for healthier outcomes.

In addition to problems of misclassification associated with the ecological comparison of state prevalences and state mortality, between 1993 and 1994, approximately 2.7% of the U.S. population moved from one state to another, and national trends existed in interregional migration as well (*45*). Because rates of interstate migration have been similar or higher during the past 20 years, current state risk factor prevalence rates cannot be assumed to reflect the prevalences of risk factors among long-term state residents.

Several studies indicate the reliability or validity of BRFSS data. One study compared estimates from BRFSS telephone questions with measured physical characteristics for several cardiovascular risk factors (*46*). The validity of self-reported BMI was assessed using a cutoff value between the standards for men and women. Using measured height and weight as standards, researchers reported a sensitivity of 77% and a specificity of 99% for self-report of BMI among men and a sensitivity of 72% and a specificity of 99% for self-report of BMI among women. For cigarette smoking, as validated by lung capacity, researchers reported a sensitivity of 78% and a specificity of 97% among men and a sensitivity of 86% and a specificity of 96% among women. For diabetes, as validated by fasting serum glucose, researchers reported a sensitivity of 67% and a specificity of 98% among men and a sensitivity of 80% and a specificity of 98% among women. For hypertension, as validated by measurement, researchers reported a sensitivity of 40% and a specificity of 87% among men and a sensitivity of 46% and a specificity of 87% among women. The study population (in upper New York State) was 99% white and thus precluded racial comparisons of validity. Another study reported higher sensitivity and specificity for self-reported hypertension among both blacks and whites (*47*). One study compared self-reported cigarette consumption with estimates of cigarette sales from excise taxes and indicated self-reported consumption to be approximately 72% of true consumption (*48*). Another study indicated that BRFSS accurately reported smoking status, but substantially underreported obesity (*49*).

The prevalence estimates of self-reported health-risk behaviors in this analysis might be underestimated because data were collected through telephone interviews; previous studies indicate substantial differences in the characteristics of persons who reside in households without a telephone compared with those who reside in households with a telephone (*50*).

One problem with the local use of BRFSS, apparent in this summary, is the small sample sizes for blacks in approximately half the states. Available information on blacks in BRFSS does not correspond precisely with state populations of blacks. For example, the 1994 black population of Hawaii was 29,000 (*43*), and BRFSS has prevalence data on overweight among black men in Hawaii; whereas in Ohio, which had a black population of 1,235,000 (*43*), BRFSS sample size for black men was not large

enough to permit estimation of the prevalence of overweight. Achievement of reliable annual state information on blacks will require oversampling in states with small black populations. This additional information might facilitate understanding risk factors for cardiovascular disease among blacks and the design of appropriate prevention programs.

The primary intended use of this risk factor atlas is as a reference. It might indicate regions of the nation that are in particular need of risk factor reduction and health promotion programs. The atlas might also serve as a baseline for the analysis of trends and the assessment of intervention programs. It might serve to assist in planning for future data collection efforts (e.g., demonstrating where alternative methods might be beneficial in collecting information previously unavailable or unanalyzable). Finally, the atlas might serve to generate hypotheses and stimulate the development of risk factor epidemiology, which explores the causes and consequences of risk factor distributions in the population. Understanding the determinants for risk factors might facilitate their control for public health.

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TABLE 1. State-, race-, and sex-specific prevalence of selected characteristics — Behavioral Risk Factor Surveillance System, 1994 — Continued

State	Overweight				Physical inactivity				Smoking			
	White		Black		White		Black		White		Black	
	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)
Alabama	28.5	26.9	37.9	45.0	42.0	45.7	42.8	58.6	26.1	20.5	22.2	11.3
Alaska	31.0	28.0	—	—	20.6	21.7	—	—	30.1	24.1	—	—
Arizona	28.7	20.5	—	—	24.2	23.3	—	—	21.6	25.1	—	—
Arkansas	32.7	26.0	40.3	37.0	33.2	36.1	32.4	47.0	29.4	24.9	31.9	15.8
California	27.2	23.1	36.8	37.4	16.2	22.3	26.4	38.9	20.6	16.6	20.0	12.5
Colorado	22.3	16.3	—	—	14.6	17.1	—	—	24.3	21.6	—	—
Connecticut	30.1	20.4	—	—	16.9	24.6	—	40.3	20.8	18.8	—	24.6
Delaware	25.7	25.4	45.5	36.5	31.1	36.5	37.4	57.6	28.4	24.3	21.2	24.9
District of Columbia	12.8	10.6	22.2	41.7	42.4	38.9	50.3	56.3	15.4	7.3	18.6	16.5
Florida	25.3	22.2	35.6	36.6	23.7	29.1	34.4	43.0	26.8	22.9	18.2	16.6
Georgia	23.3	23.9	35.5	40.9	30.5	33.4	25.5	43.2	25.9	22.6	19.4	15.5
Hawaii	21.5	17.6	52.6	33.7	12.8	15.0	20.4	26.4	20.9	20.1	34.1	28.6
Idaho	29.0	28.8	—	—	22.0	21.8	—	—	18.7	19.8	—	—
Illinois	29.9	24.7	37.4	42.6	31.8	33.4	29.0	47.6	25.9	22.9	28.7	24.5
Indiana	30.1	28.5	39.9	54.5	25.0	32.7	34.6	45.6	27.9	23.1	26.6	20.6
Iowa	30.4	28.1	—	—	35.6	31.0	—	—	21.8	20.0	—	—
Kansas	28.5	19.1	—	—	34.1	33.9	—	—	23.9	20.1	—	—
kentucky	29.9	28.4	—	54.9	45.2	46.9	—	45.2	29.9	27.6	—	21.7
Louisiana	25.5	25.3	31.4	46.4	30.6	34.7	30.2	39.0	28.9	23.6	35.1	16.3
Maine	29.8	27.5	—	—	42.9	38.8	—	—	25.1	22.4	—	—
Maryland	27.6	23.8	34.5	41.2	26.7	29.8	30.6	41.7	21.1	20.1	25.4	18.3
Massachusetts	28.6	20.1	—	37.2	22.1	24.6	—	34.9	22.4	19.8	—	19.8
Michigan	31.9	27.6	37.2	51.6	19.5	24.3	20.2	39.2	24.8	25.9	22.6	20.8
Minnesota	29.6	24.9	—	39.4	21.3	21.9	—	19.2	21.4	21.3	—	25.1
Mississippi	29.4	24.5	46.1	46.1	35.5	36.7	37.8	46.8	27.9	20.6	26.2	13.1
Missouri	33.0	27.2	—	33.6	27.4	34.4	—	46.2	26.9	21.3	—	30.7
Montana	27.8	24.9	—	—	21.3	20.0	—	—	21.8	21.3	—	—
Nebraska	32.7	25.9	—	—	26.7	21.5	—	—	19.8	18.0	—	—
Nevada	31.2	23.8	—	—	18.6	24.1	—	—	32.1	26.8	—	—
New Hampshire	27.8	23.0	—	—	22.9	27.4	—	—	24.0	21.2	—	—
New Jersey	28.3	21.3	29.6	49.7	28.3	33.1	20.2	45.9	22.2	24.2	28.8	26.6
New Mexico	23.7	18.4	—	—	14.8	21.4	—	—	19.5	24.1	—	—
New York	28.5	24.3	32.6	42.5	32.5	36.3	34.7	50.9	22.8	20.6	23.5	19.1

TABLE 1. State-, race-, and sex-specific prevalence of selected characteristics — Behavioral Risk Factor Surveillance System, 1994 — Continued

State	Overweight				Physical inactivity				Smoking			
	White		Black		White		Black		White		Black	
	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)
North Carolina	32.0	25.9	37.6	48.2	38.8	39.9	49.6	62.9	29.5	25.5	37.9	22.5
North Dakota	30.2	23.8	—	—	34.2	29.5	—	—	19.9	19.5	—	—
Ohio	30.3	26.8	—	47.3	41.1	35.9	—	41.7	27.6	25.3	—	25.4
Oklahoma	24.8	24.6	—	34.5	27.6	30.9	—	27.9	24.6	21.5	—	30.7
Oregon	26.4	26.3	—	—	20.8	21.0	—	—	21.4	20.8	—	—
Pennsylvania	30.9	27.6	27.3	49.1	22.0	28.8	20.2	39.7	24.8	23.1	24.5	31.6
Rhode Island	25.8	23.7	—	—	22.1	30.4	—	—	23.9	21.0	—	—
South Carolina	25.1	25.1	39.7	49.1	28.4	29.5	31.6	43.3	30.4	21.9	21.8	15.5
South Dakota	28.1	27.7	—	—	29.1	31.9	—	—	20.1	19.8	—	—
Tennessee	26.8	25.4	27.9	46.2	38.6	39.9	37.6	47.9	28.8	26.0	24.1	21.6
Texas	33.1	24.9	—	51.6	24.6	27.9	—	32.6	23.4	20.7	—	19.4
Utah	21.6	26.3	—	—	18.2	22.9	—	—	16.7	14.2	—	—
Vermont	26.2	24.8	—	—	23.0	23.6	—	—	23.3	22.0	—	—
Virginia	28.3	20.4	46.4	39.0	18.4	23.1	31.4	33.0	27.5	23.6	28.4	22.6
Washington	26.8	25.6	—	—	16.2	19.4	—	—	23.9	19.4	—	—
West Virginia	32.2	31.0	—	—	43.2	47.3	—	—	27.7	26.8	—	—
Wisconsin	35.7	26.7	26.7	45.0	27.4	23.5	26.3	39.8	24.0	22.3	19.7	31.4
Wyoming	31.0	22.8	—	—	21.8	19.8	—	—	20.7	21.9	—	—
Median	**28.5**	**24.9**	**37.0**	**42.6**	**26.7**	**29.5**	**31.5**	**43.1**	**24.0**	**21.6**	**24.3**	**21.2**
Low	**12.8**	**10.6**	**22.2**	**33.6**	**12.8**	**15.0**	**20.2**	**19.2**	**15.4**	**7.3**	**18.2**	**11.3**
High	**35.7**	**31.0**	**52.6**	**54.9**	**45.2**	**47.3**	**50.3**	**62.9**	**32.1**	**27.6**	**37.9**	**31.6**

TABLE 1. State-, race-, and sex-specific prevalence of selected characteristics — Behavioral Risk Factor Surveillance System, 1994 — Continued

State	Hypertension*				Diabetes Mellitus				Multiple Risk Factors			
	White		Black		White		Black		White		Black	
	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)
Alabama	19.1	18.0	20.7	31.1	3.5	2.7	2.1	6.2	26.6	25.5	28.4	36.2
Alaska	14.8	20.4	—	—	3.2	2.8	—	—	19.9	17.7	—	—
Arizona	21.1	21.0	—	—	4.5	2.8	—	—	19.4	15.3	—	—
Arkansas	—	—	—	—	4.1	5.4	6.0	12.5	26.8	24.0	29.7	33.6
California	19.2	22.5	30.4	35.8	4.0	3.3	6.9	8.6	14.7	13.7	19.6	25.2
Colorado	15.6	19.4	—	—	3.5	2.6	—	—	13.5	10.7	—	—
Connecticut	21.3	23.9	—	29.5	3.8	5.4	—	6.7	14.5	13.7	—	29.6
Delaware	19.4	20.9	29.5	30.8	5.0	4.4	13.4	8.2	23.6	23.0	35.7	39.4
District of Columbia	13.0	15.4	22.1	25.9	2.7	1.9	5.0	5.7	15.6	9.2	23.1	36.6
Florida	19.7	21.6	31.8	32.7	5.0	4.2	7.6	6.4	17.7	18.3	24.6	29.9
Georgia	15.9	17.4	21.2	27.1	3.1	3.3	6.7	9.6	21.3	20.7	19.5	27.3
Hawaii	14.5	16.4	27.6	25.7	2.7	2.5	0.5	6.6	11.1	9.0	31.4	24.1
Idaho	14.3	22.9	—	—	3.9	4.6	—	—	17.5	16.4	—	—
Illinois	18.5	20.1	24.4	29.3	5.0	4.1	7.9	10.8	25.4	21.2	31.7	39.1
Indiana	21.0	27.3	24.2	34.1	4.1	4.5	2.0	6.1	21.8	22.0	28.6	37.7
Iowa	20.0	21.2	—	—	3.5	4.7	—	—	24.8	21.1	—	—
Kansas	18.2	23.9	—	—	4.8	3.1	—	—	25.4	17.5	—	—
kentucky	23.5	26.7	—	30.3	3.4	4.8	—	6.3	32.0	30.9	—	35.0
Louisiana	19.4	19.6	17.9	30.9	5.0	3.2	5.2	9.3	22.4	22.6	24.8	32.4
Maine	20.7	22.8	—	—	3.3	3.9	—	—	29.1	23.5	—	—
Maryland	17.1	20.1	19.6	30.4	4.5	4.2	6.3	7.1	18.2	18.8	24.2	30.9
Massachusetts	19.3	21.4	—	—	3.5	3.5	—	18.2	17.6	15.4	—	32.4
Michigan	24.0	21.7	28.4	27.1	4.0	5.3	3.5	4.7	18.9	20.7	19.6	32.3
Minnesota	20.7	22.7	—	—	3.5	4.0	—	4.5	18.6	16.2	—	17.1
Mississippi	21.2	24.9	30.7	44.8	5.3	3.2	6.6	10.4	27.6	19.4	36.4	32.7
Missouri	22.0	23.7	—	38.9	5.7	5.1	—	11.6	24.1	21.0	—	37.1
Montana	19.4	21.4	—	—	2.0	2.2	—	—	16.2	16.0	—	—
Nebraska	20.3	20.6	—	—	3.9	5.4	—	—	19.5	14.8	—	—
Nevada	21.4	21.1	—	30.5	4.9	3.1	—	—	19.6	17.7	—	—
New Hampshire	19.9	19.8	—	—	4.3	4.5	—	—	18.8	17.8	—	—
New Jersey	24.9	18.8	—	24.9	4.8	3.2	5.9	—	22.5	19.2	18.2	32.8
New Mexico	15.0	14.3	—	—	4.9	4.1	—	—	13.6	13.5	—	—
New York	20.5	19.2	19.2	38.5	3.4	3.5	7.1	6.8	20.2	20.9	24.4	33.9

TABLE 1. State-, race-, and sex-specific prevalence of selected characteristics — Behavioral Risk Factor Surveillance System, 1994 — Continued

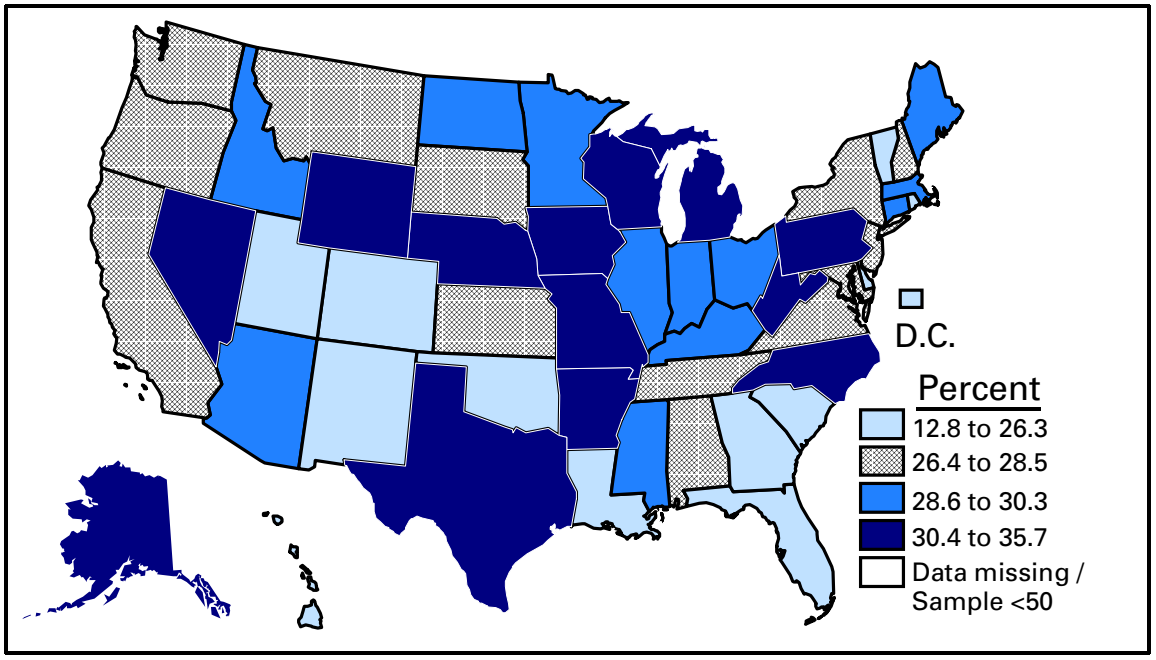
State	Hypertension*				Diabetes Mellitus				Multiple Risk Factors			
	White		Black		White		Black		White		Black	
	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)
North Carolina	19.0	17.3	13.0	27.5	4.2	3.4	5.5	6.1	30.5	25.9	39.7	42.9
North Dakota	17.3	21.4	—	—	3.9	3.3	—	—	22.6	17.4	—	—
Ohio	18.1	22.4	—	32.6	3.8	5.2	—	6.4	28.9	23.2	—	34.9
Oklahoma	20.8	25.3	—	—	2.4	2.9	—	3.3	19.5	18.5	—	26.0
Oregon	18.9	21.0	—	—	4.0	3.5	—	—	17.6	15.7	—	—
Pennsylvania	22.8	22.1	23.5	31.0	4.8	4.9	7.5	12.2	18.7	21.3	16.0	39.7
Rhode Island	23.4	22.1	—	—	4.4	4.7	—	—	18.4	18.3	—	—
South Carolina	18.5	24.7	27.1	34.4	6.2	4.3	6.9	8.2	22.3	18.7	27.5	30.5
South Dakota	15.5	19.3	—	—	4.9	3.2	—	—	19.6	19.5	—	—
Tennessee	20.5	24.0	24.7	27.4	4.9	4.9	6.4	11.0	28.7	26.1	21.3	38.1
Texas	17.3	18.8	24.8	29.1	5.5	4.7	—	1.1	22.4	17.9	—	26.4
Utah	16.9	20.4	—	—	4.1	3.6	—	—	13.5	13.3	—	—
Vermont	19.6	19.2	—	—	3.6	4.2	—	—	17.8	18.1	—	—
Virginia	16.9	19.7	25.2	37.2	2.9	3.7	5.6	12.6	18.8	15.8	32.5	31.4
Washington	19.2	19.9	—	—	4.1	3.6	—	—	15.4	14.0	—	—
West Virginia	22.9	24.5	—	—	4.2	6.1	—	—	28.2	33.1	—	—
Wisconsin	20.2	19.3	—	—	3.7	2.6	4.5	4.1	20.0	18.0	21.7	34.2
Wyoming	—	—	—	—	3.1	3.2	—	—	17.3	15.1	—	—
Median	**19.4**	**21.0**	**24.6**	**30.7**	**4.0**	**3.7**	**6.1**	**6.8**	**19.6**	**18.3**	**24.7**	**32.8**
Low	**13.0**	**14.3**	**13.0**	**24.9**	**2.0**	**1.9**	**0.5**	**1.1**	**11.1**	**9.0**	**16.0**	**17.1**
High	**24.9**	**27.3**	**31.8**	**44.8**	**6.2**	**6.1**	**13.4**	**18.2**	**32.0**	**33.1**	**39.7**	**42.9**

* Hypertension data for 1992 only.

TABLE 1. State-, race-, and sex-specific prevalence of selected characteristics — Behavioral Risk Factor Surveillance System, 1994 — Continued

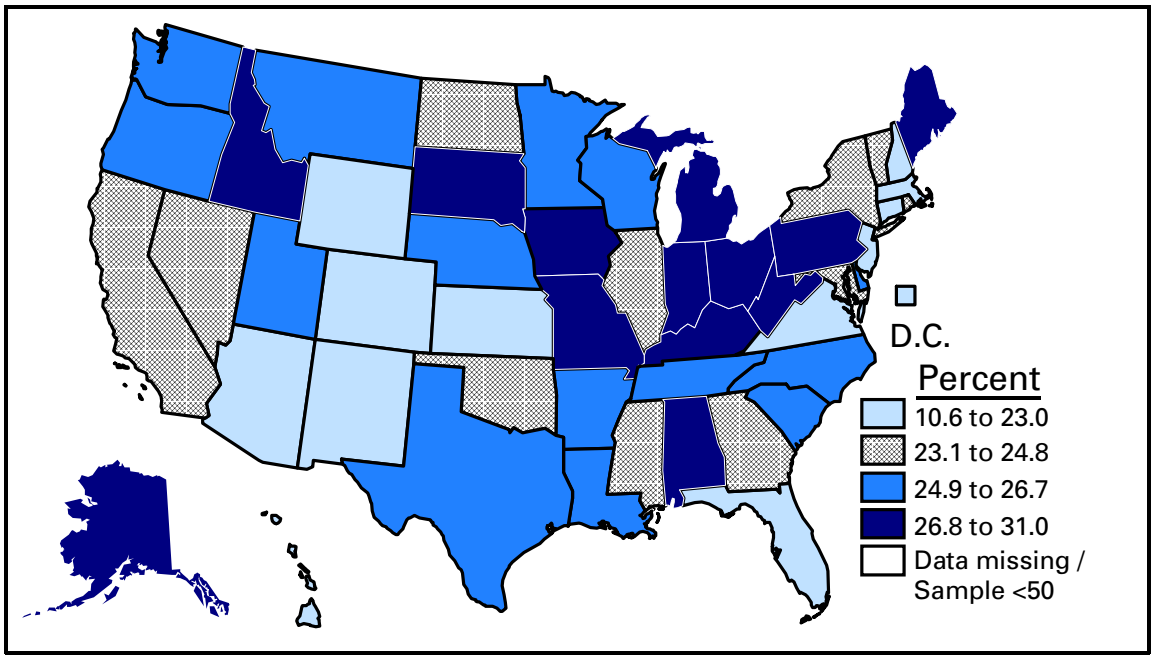
State	Weight Loss				Smoking Cessation			
	White		Black		White		Black	
	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)	Men (%)	Women (%)
Alabama	84.4	92.3	81.7	90.0	45.1	38.8	35.5	36.5
Alaska	90.5	94.1	—	—	29.6	36.3	—	—
Arizona	83.8	89.2	—	—	44.3	27.6	—	—
Arkansas	80.8	88.2	83.7	82.0	45.9	37.2	—	—
California	90.9	93.4	88.5	94.7	49.3	51.9	—	—
Colorado	89.4	92.7	—	—	49.1	41.6	—	—
Connecticut	80.6	89.3	—	—	53.4	53.3	—	—
Delaware	76.9	84.3	77.5	78.5	45.6	46.4	39.7	34.7
District of Columbia	82.0	82.5	67.3	81.2	40.7	58.3	30.6	32.7
Florida	84.5	88.7	83.5	89.1	48.7	47.1	38.5	29.9
Georgia	81.7	87.4	85.8	88.2	31.7	28.7	31.6	26.1
Hawaii	87.4	94.0	90.5	89.9	54.2	45.7	16.4	28.0
Idaho	81.5	91.5	—	—	31.8	23.1	—	—
Illinois	81.4	88.9	83.3	82.1	31.4	23.5	22.1	17.9
Indiana	87.0	91.5	—	93.4	45.7	38.4	—	—
Iowa	80.6	90.2	—	—	36.3	29.1	—	—
Kansas	87.0	91.3	—	—	46.5	37.9	—	—
kentucky	80.3	90.0	—	88.4	39.7	31.7	—	—
Louisiana	76.7	88.1	81.1	83.5	48.1	38.4	27.7	36.2
Maine	84.4	91.3	—	—	37.8	34.7	—	—
Maryland	85.2	92.4	85.3	90.2	48.9	45.1	32.2	37.8
Massachusetts	91.1	92.7	—	—	47.9	48.0	—	—
Michigan	87.5	91.6	88.5	89.8	48.2	39.4	—	33.4
Minnesota	85.0	89.2	—	—	51.6	44.5	—	—
Mississippi	71.2	85.2	68.6	86.7	48.2	39.2	33.2	22.0
Missouri	85.3	87.9	—	82.3	47.7	42.4	—	—
Montana	85.7	88.9	—	—	44.5	35.5	—	—
Nebraska	83.6	90.2	—	—	53.1	41.0	—	—
Nevada	79.5	90.2	—	—	41.9	38.2	—	—
New Hampshire	87.6	92.6	—	—	47.8	44.4	—	—
New Jersey	83.1	88.0	—	83.6	49.1	41.6	—	—
New Mexico	82.7	88.2	—	—	54.9	40.3	—	—
New York	81.5	87.0	77.0	84.2	47.6	43.4	—	28.0
North Carolina	77.2	86.5	68.6	82.0	43.4	37.6	25.2	21.9
North Dakota	80.9	89.4	—	—	54.4	41.1	—	—
Ohio	70.8	88.7	—	86.7	33.2	22.9	—	—
Oklahoma	75.7	82.6	—	80.1	36.4	38.6	—	—
Oregon	86.0	92.0	—	—	56.0	46.7	—	—
Pennsylvania	87.6	92.9	91.1	90.1	50.2	42.2	41.1	26.2
Rhode Island	88.4	90.3	—	—	50.4	49.2	—	—
South Carolina	74.2	81.6	76.5	79.0	13.4	21.2	23.9	14.8
South Dakota	83.8	89.9	—	—	52.8	37.1	—	—
Tennessee	82.2	87.6	84.1	83.9	35.5	30.1	26.2	31.1
Texas	85.6	90.4	—	75.2	49.4	38.8	—	—
Utah	88.6	93.0	—	—	42.8	32.3	—	—
Vermont	82.5	89.2	—	—	51.6	45.7	—	—
Virginia	85.1	90.0	87.2	95.3	44.2	38.9	35.8	23.2
Washington	87.5	90.9	—	—	51.4	50.7	—	—
West Virginia	79.3	87.9	—	—	35.1	23.8	—	—
Wisconsin	88.7	92.0	—	85.8	50.1	44.5	—	22.6
Wyoming	87.3	93.7	—	—	33.5	27.2	—	—
Median	**83.8**	**90.0**	**83.5**	**85.8**	**47.6**	**38.9**	**31.6**	**28.0**
Low	**70.8**	**81.6**	**67.3**	**75.2**	**13.4**	**21.2**	**16.4**	**14.8**
High	**91.1**	**94.1**	**91.1**	**95.3**	**56.0**	**58.3**	**41.1**	**37.8**

FIGURE 1a. Prevalence of overweight* among white men — Behavioral Risk Factor Surveillance System, 1994



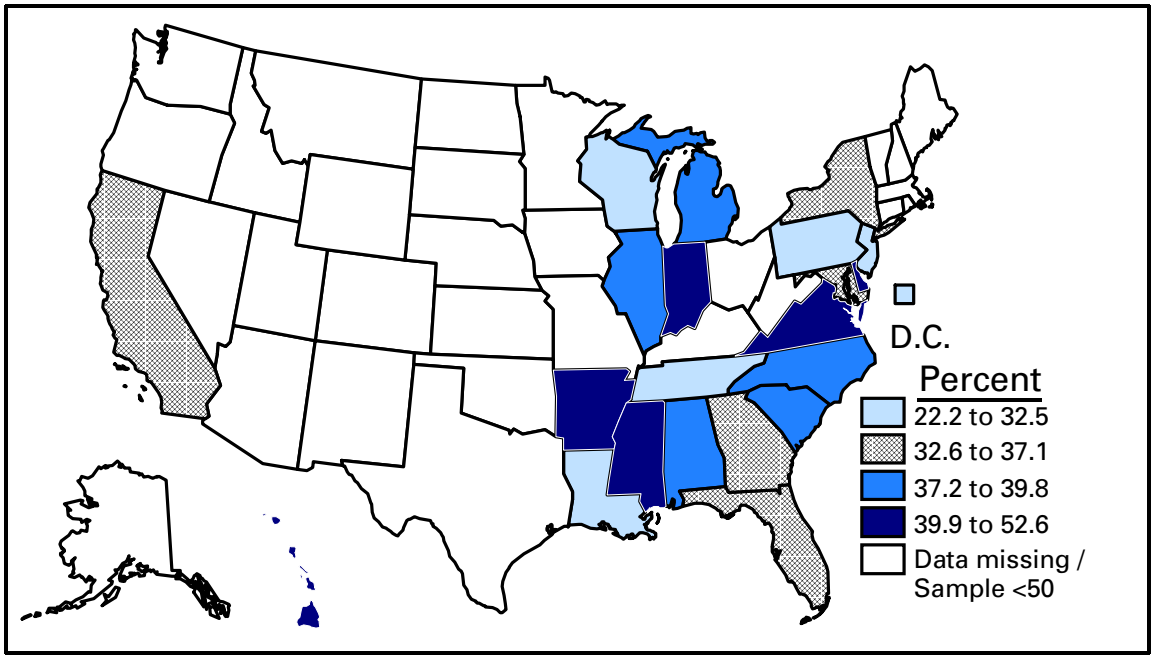
*Overweight: Body mass index (BMI = weight[kg]/height[m^2]) ≥27.8 for men and ≥27.3 for women.

FIGURE 1b. Prevalence of overweight* among white women — Behavioral Risk Factor Surveillance System, 1994



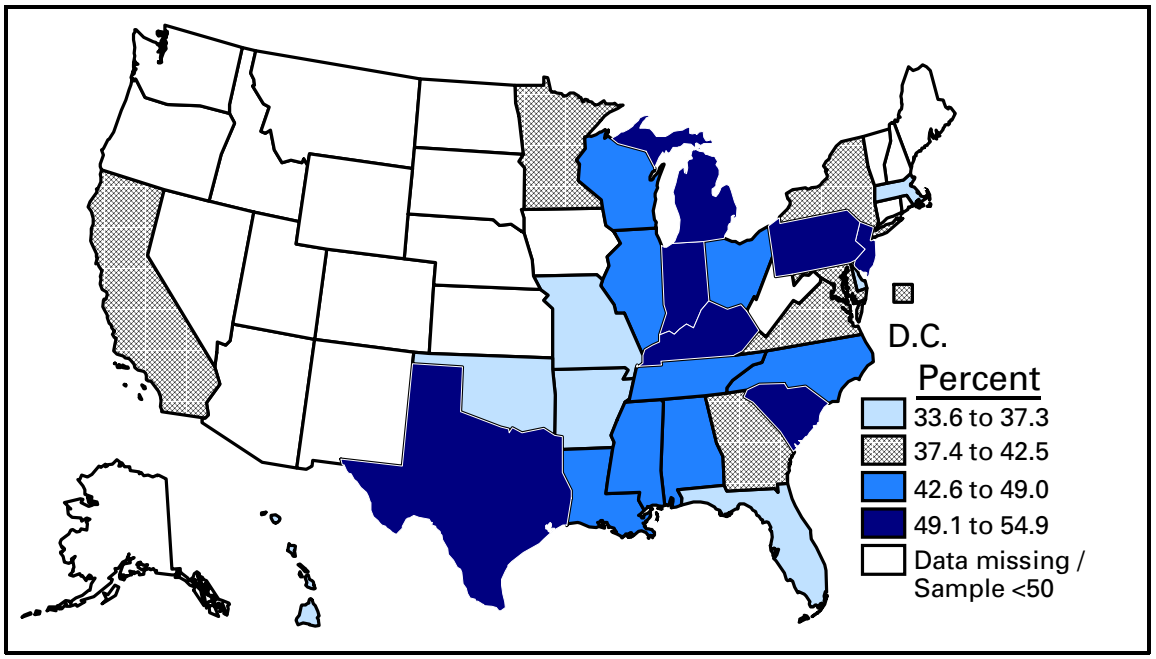
*Overweight: Body mass index (BMI = weight[kg]/height[m^2]) ≥27.8 for men and ≥27.3 for women.

FIGURE 1c. Prevalence of overweight* among black men — Behavioral Risk Factor Surveillance System, 1994



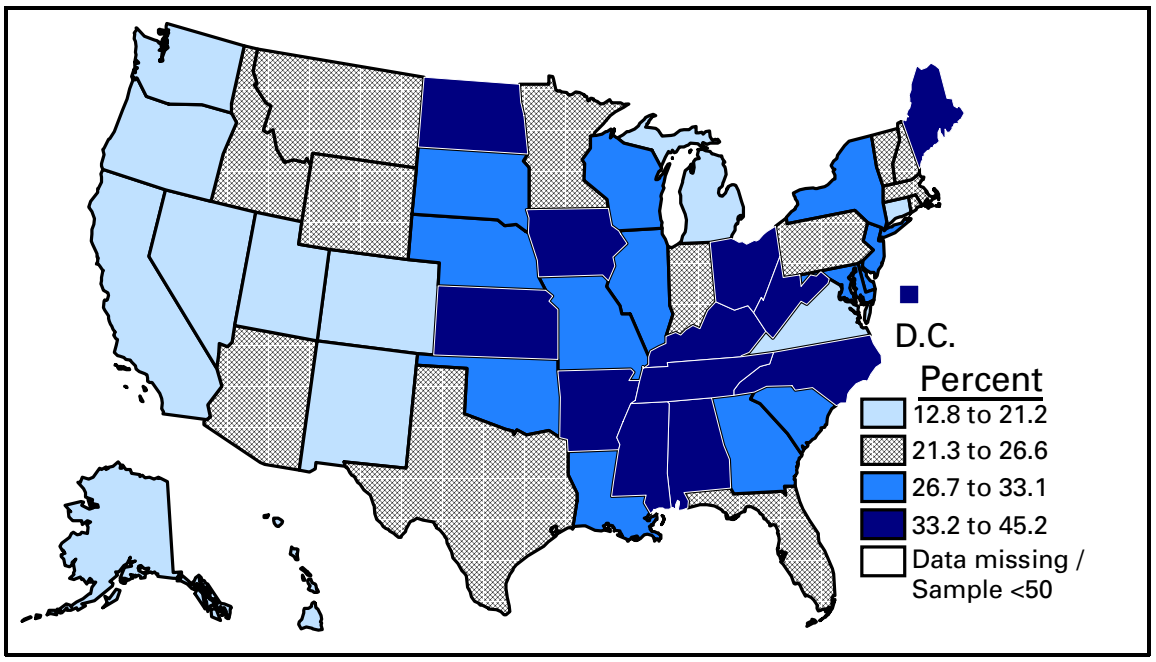
*Overweight: Body mass index (BMI = weight[kg]/height[m^2]) ≥27.8 for men and ≥27.3 for women.

FIGURE 1d. Prevalence of overweight* among black women — Behavioral Risk Factor Surveillance System, 1994



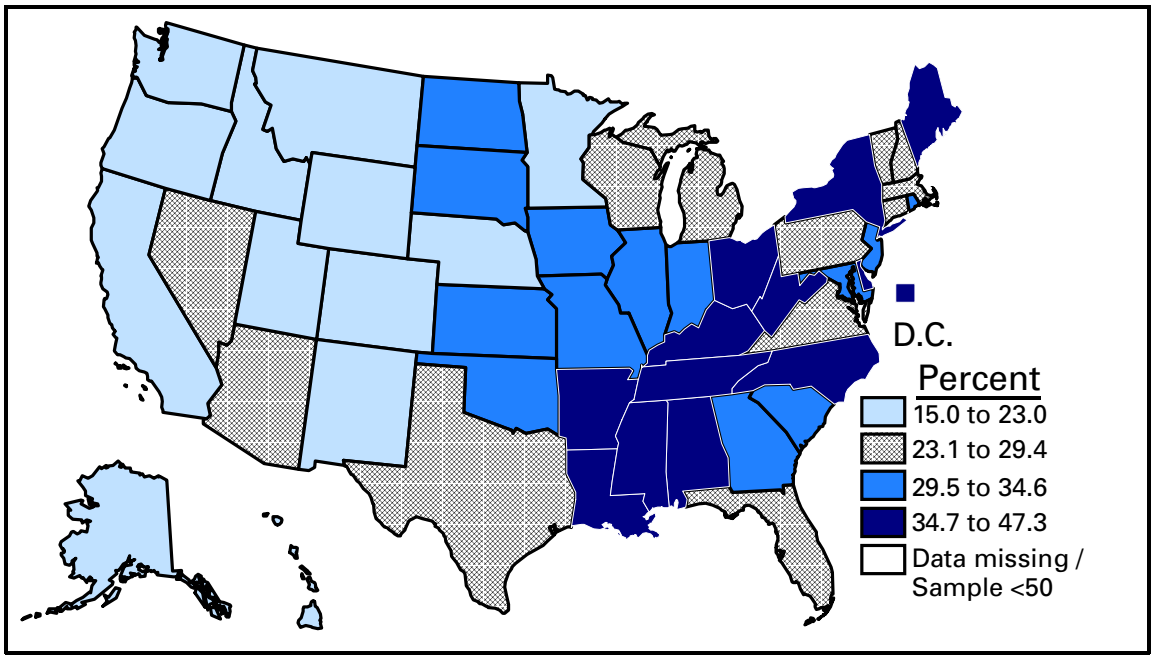
*Overweight: Body mass index (BMI = weight[kg]/height[m^2]) ≥27.8 for men and ≥27.3 for women.

FIGURE 2a. Prevalence of physical inactivity* among white men — Behavioral Risk Factor Surveillance System, 1994



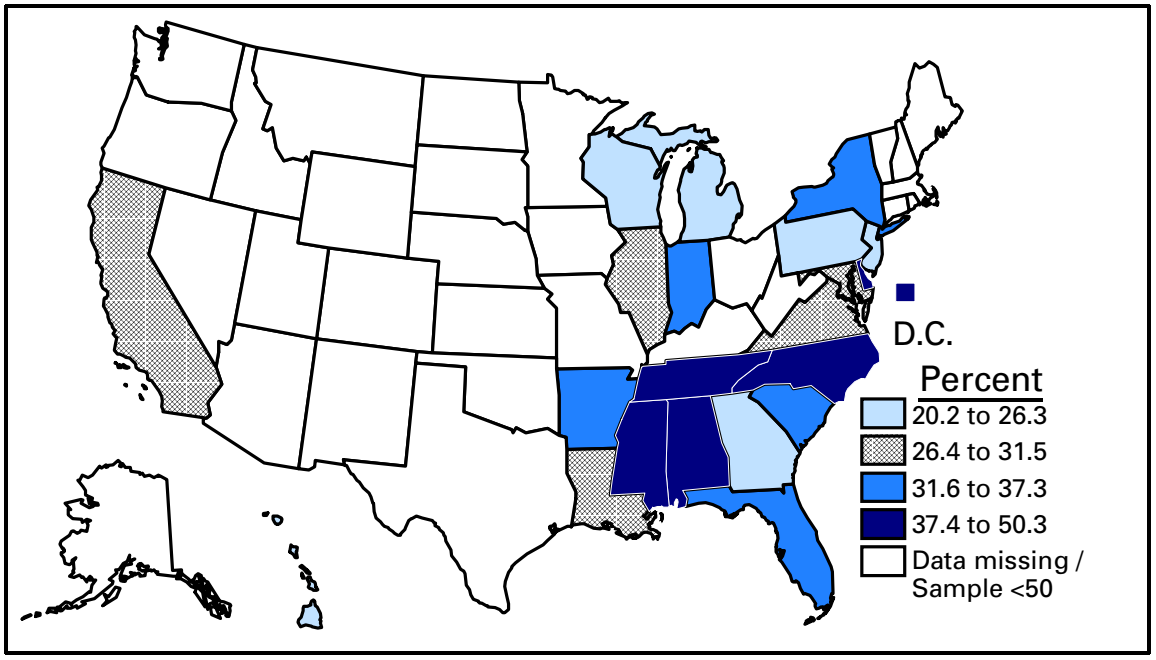
*Physical inactivity: No reported exercise, recreation, or physical activities (other than regular job duties) during the previous month.

FIGURE 2b. Prevalence of physical inactivity* among white women — Behavioral Risk Factor Surveillance System, 1994



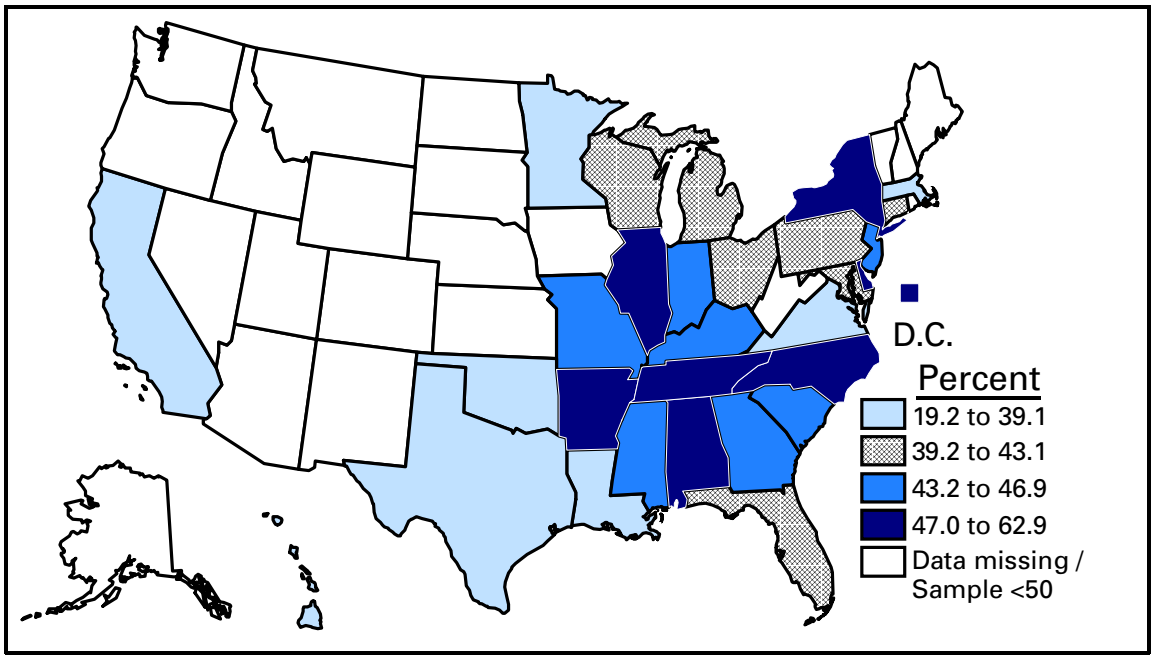
*Physical inactivity: No reported exercise, recreation, or physical activities (other than regular job duties) during the previous month.

FIGURE 2c. Prevalence of physical inactivity* among black men — Behavioral Risk Factor Surveillance System, 1994



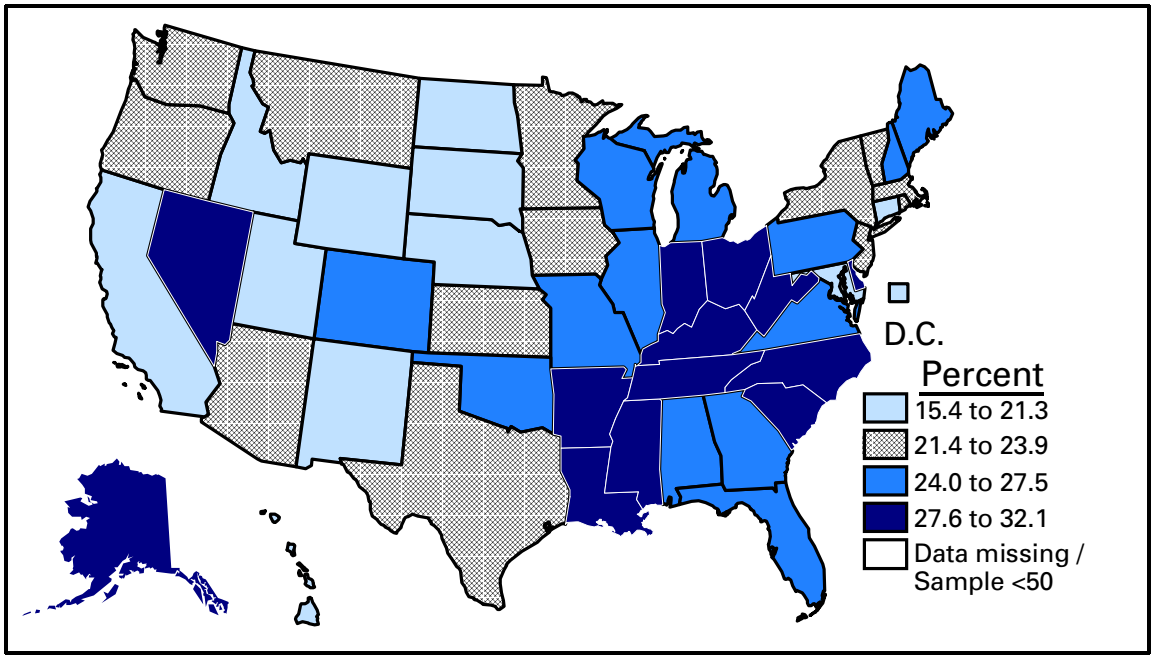
*Physical inactivity: No reported exercise, recreation, or physical activities (other than regular job duties) during the previous month.

FIGURE 2d. Prevalence of physical inactivity* among black women — Behavioral Risk Factor Surveillance System, 1994



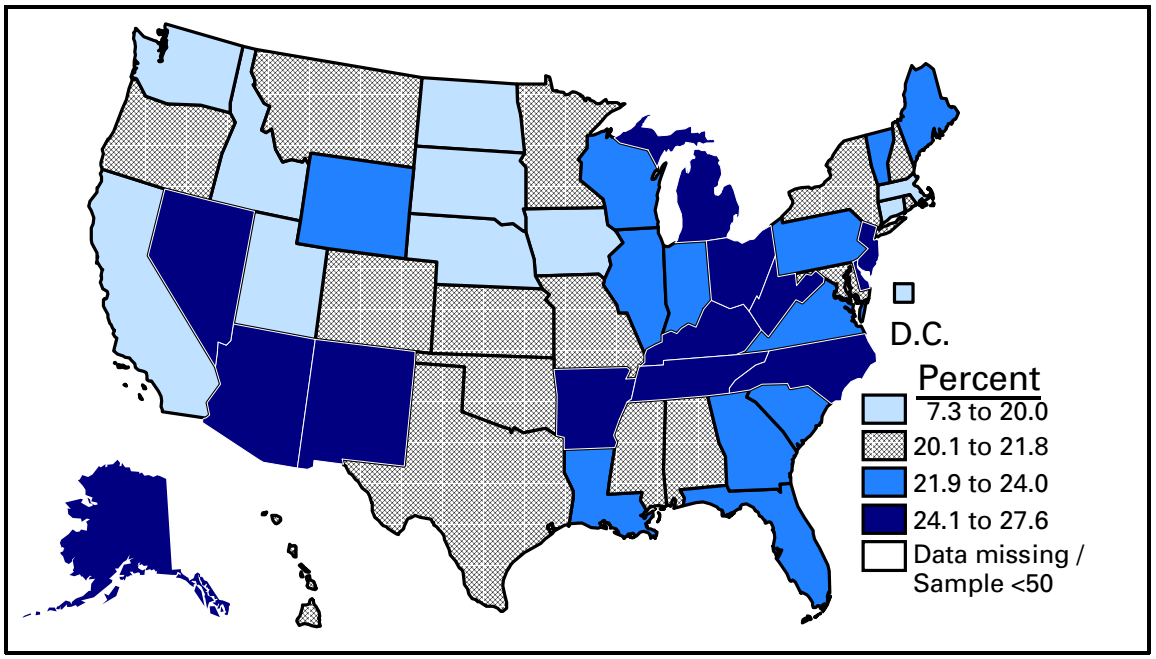
*Physical inactivity: No reported exercise, recreation, or physical activities (other than regular job duties) during the previous month.

FIGURE 3a. Prevalence of smoking* among white men — Behavioral Risk Factor Surveillance System, 1994



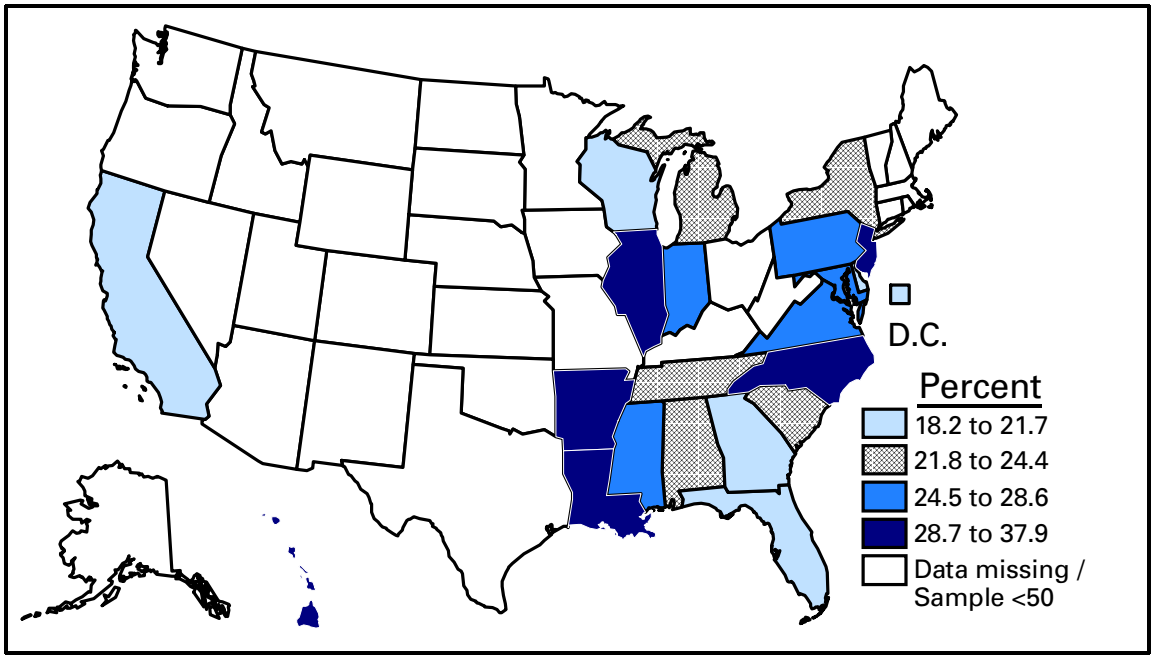
*Smoking: Current regular use of cigarettes by persons who have ever smoked at least 100 cigarettes.

FIGURE 3b. Prevalence of smoking* among white women — Behavioral Risk Factor Surveillance System, 1994



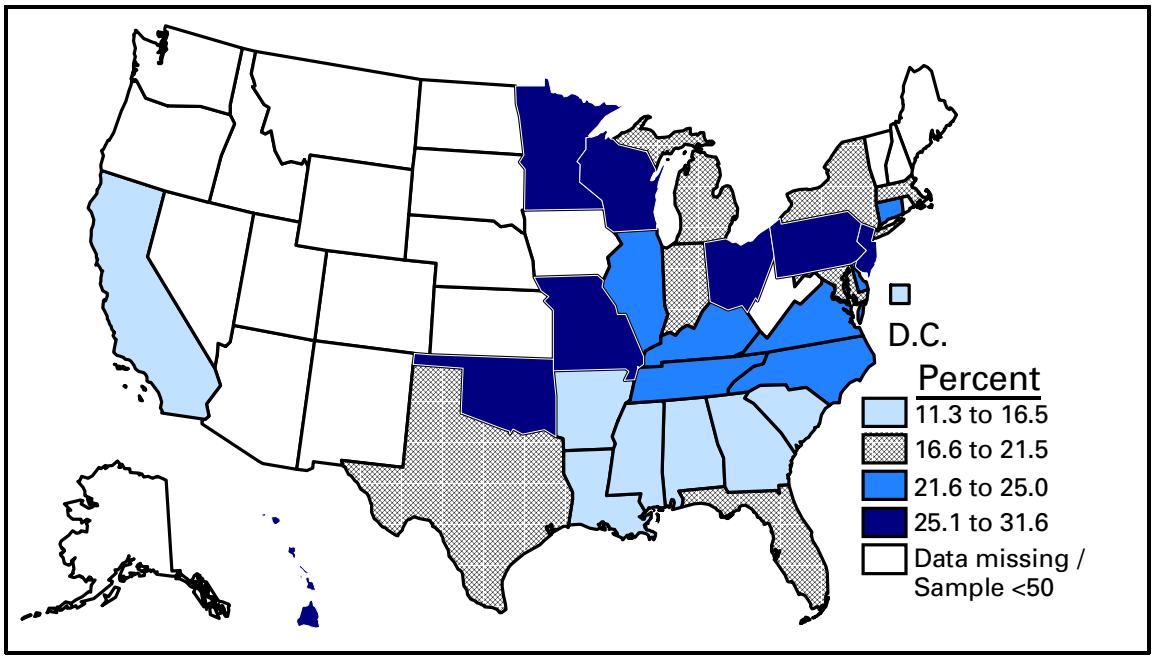
*Smoking: Current regular use of cigarettes by persons who have ever smoked at least 100 cigarettes.

FIGURE 3c. Prevalence of smoking* among black men — Behavioral Risk Factor Surveillance System, 1994



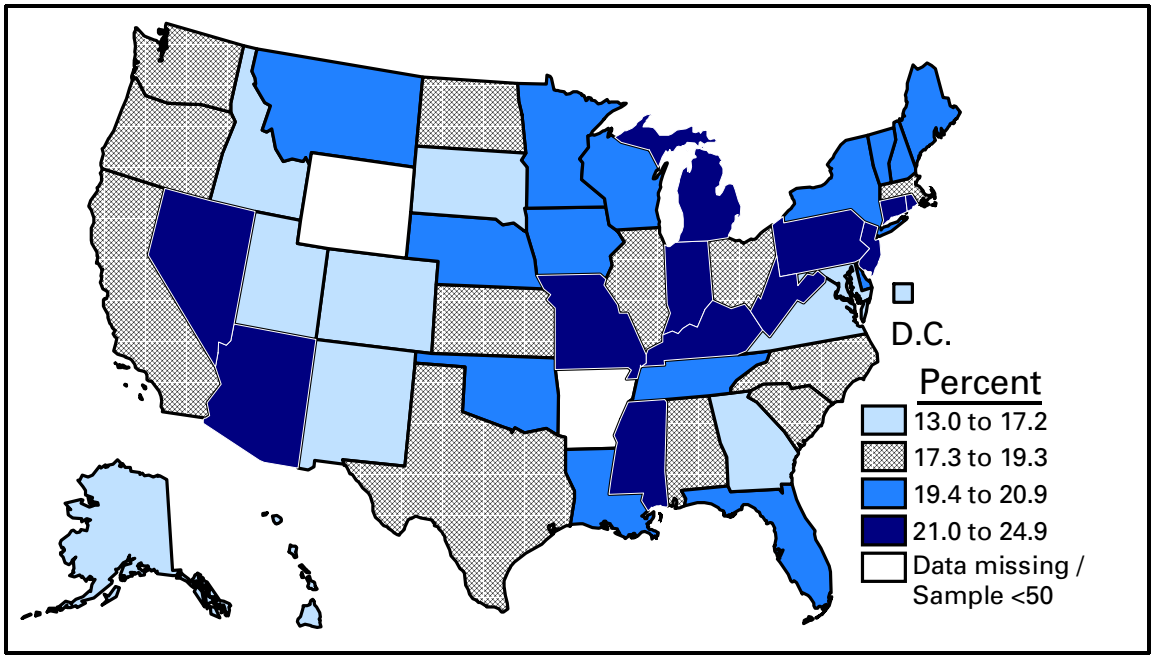
*Smoking: Current regular use of cigarettes by persons who have ever smoked at least 100 cigarettes.

FIGURE 3d. Prevalence of smoking* among black women — Behavioral Risk Factor Surveillance System, 1994



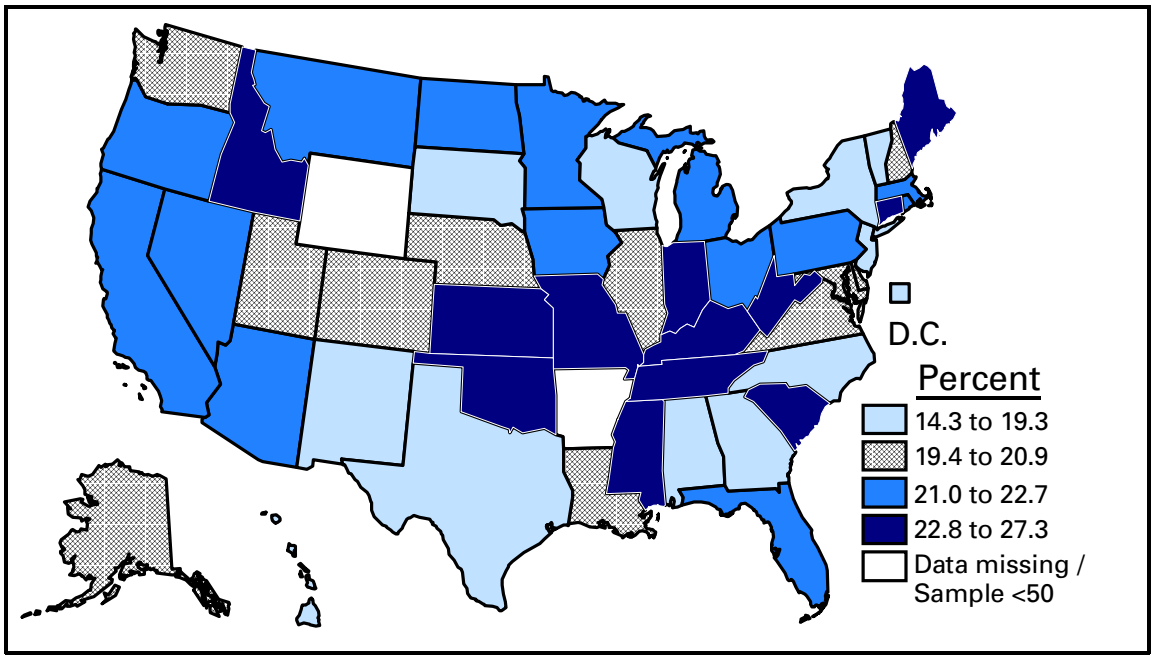
*Smoking: Current regular use of cigarettes by persons who have ever smoked at least 100 cigarettes.

FIGURE 4a. Prevalence of hypertension* among white men — Behavioral Risk Factor Surveillance System, 1992



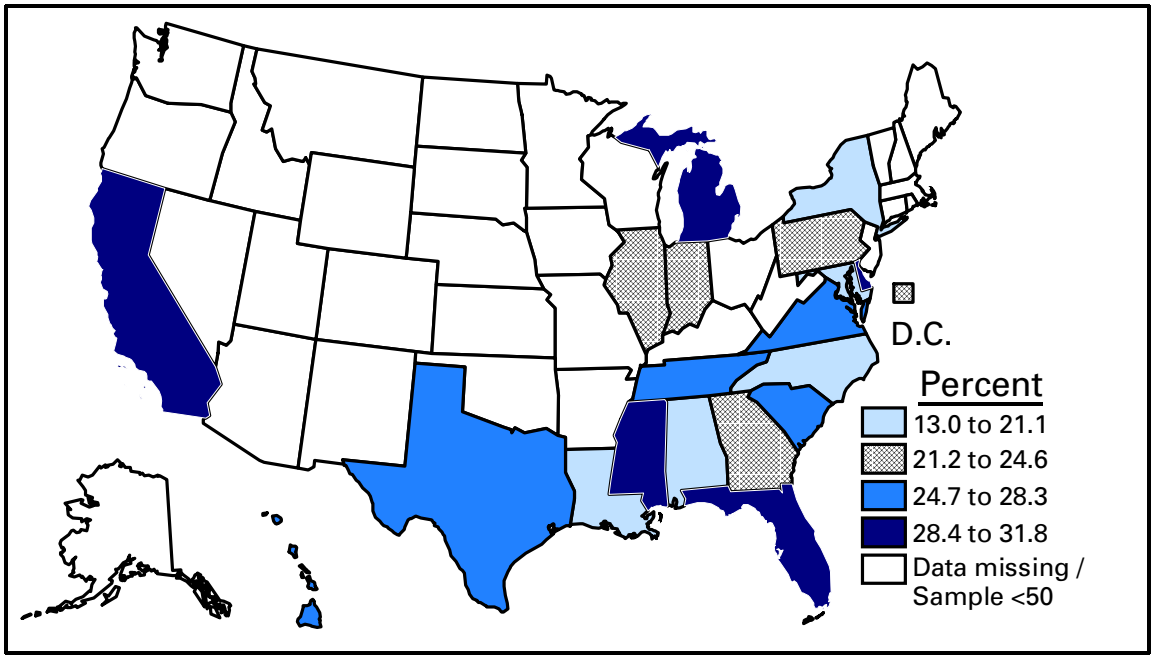
*Hypertension: Survey participants having ever been told by a health professional that they have high blood pressure.

FIGURE 4b. Prevalence of hypertension* among white women — Behavioral Risk Factor Surveillance System, 1992



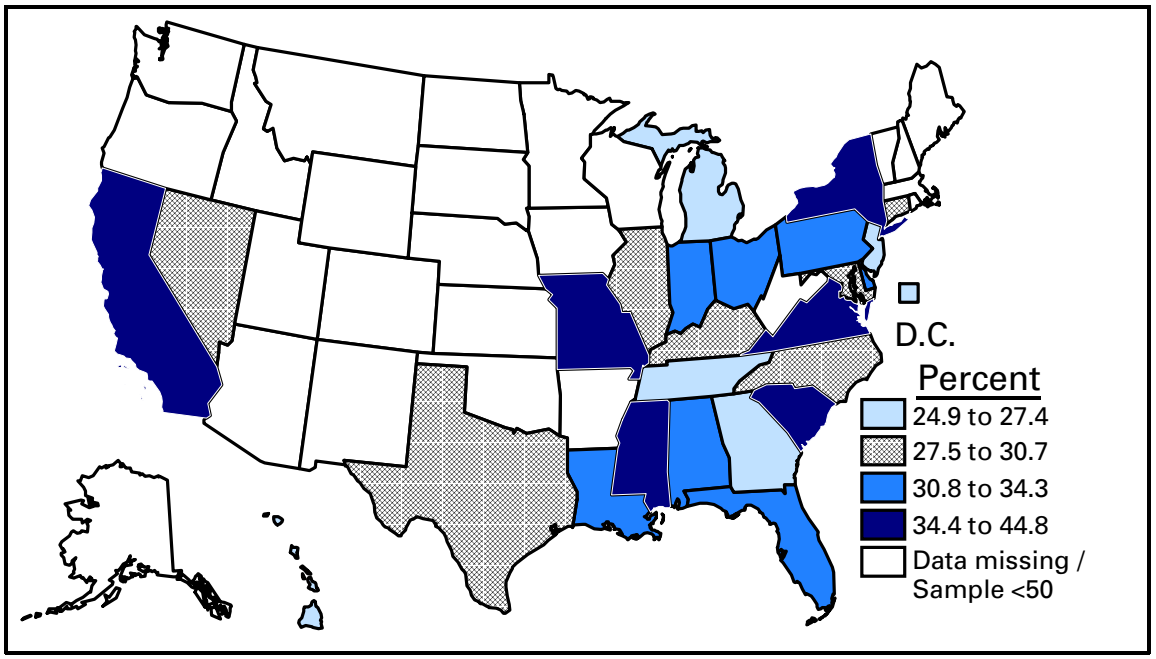
*Hypertension: Survey participants having ever been told by a health professional that they have high blood pressure.

FIGURE 4c. Prevalence of hypertension* among black men — Behavioral Risk Factor Surveillance System, 1992



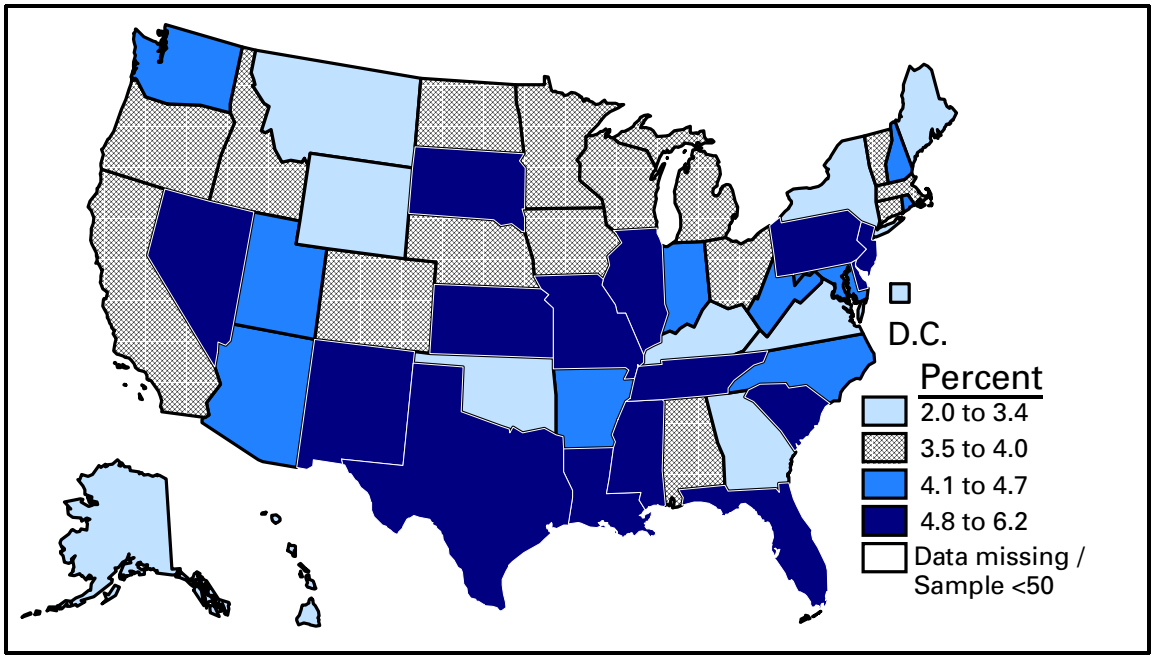
*Hypertension: Survey participants having ever been told by a health professional that they have high blood pressure.

FIGURE 4d. Prevalence of hypertension* among black women — Behavioral Risk Factor Surveillance System, 1992



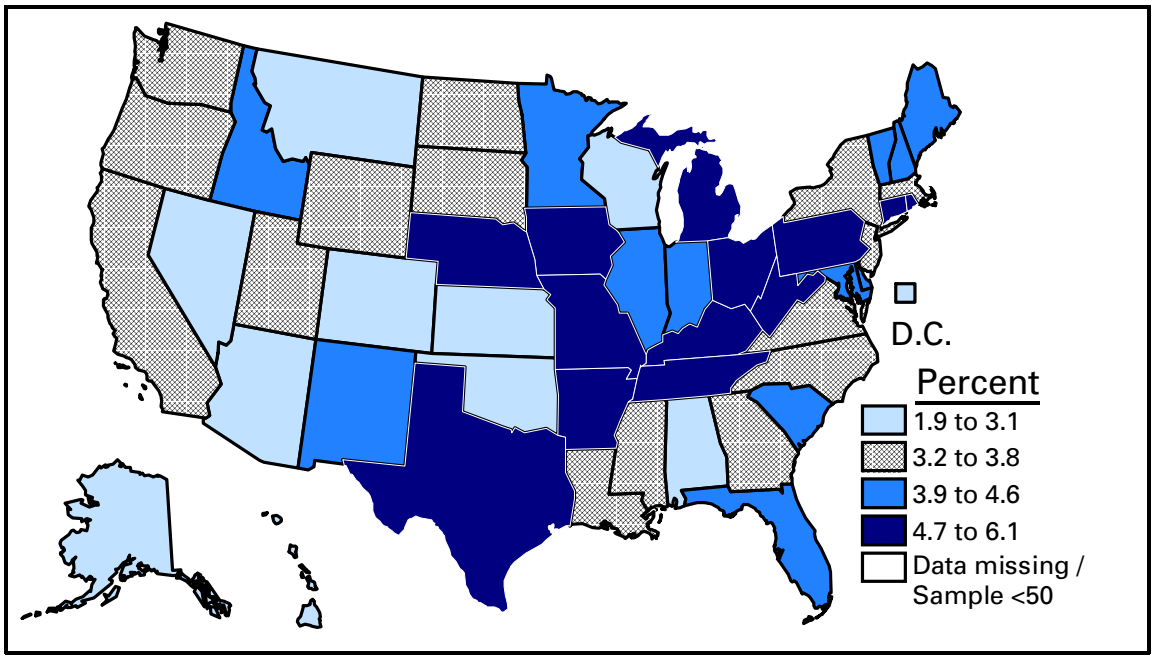
*Hypertension: Survey participants having ever been told by a health professional that they have high blood pressure.

FIGURE 5a. Prevalence of diabetes mellitus* among white men — Behavioral Risk Factor Surveillance System, 1994



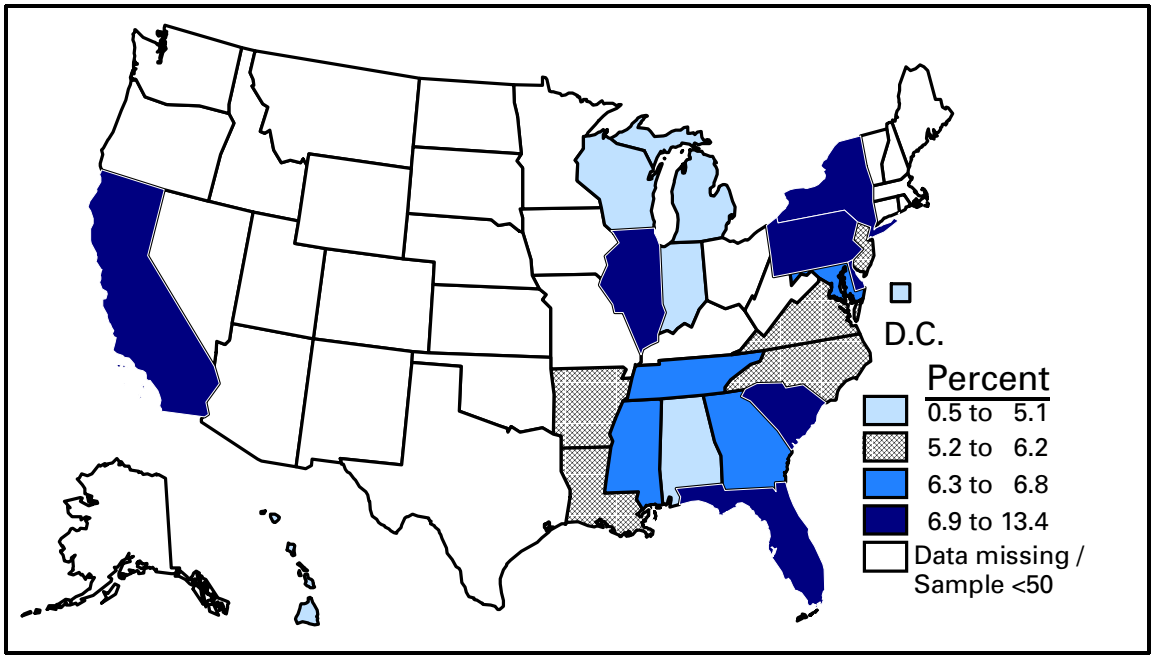
*Diabetes mellitus: Survey participants having ever been told by a physician that they have diabetes.

FIGURE 5b. Prevalence of diabetes mellitus* among white women — Behavioral Risk Factor Surveillance System, 1994



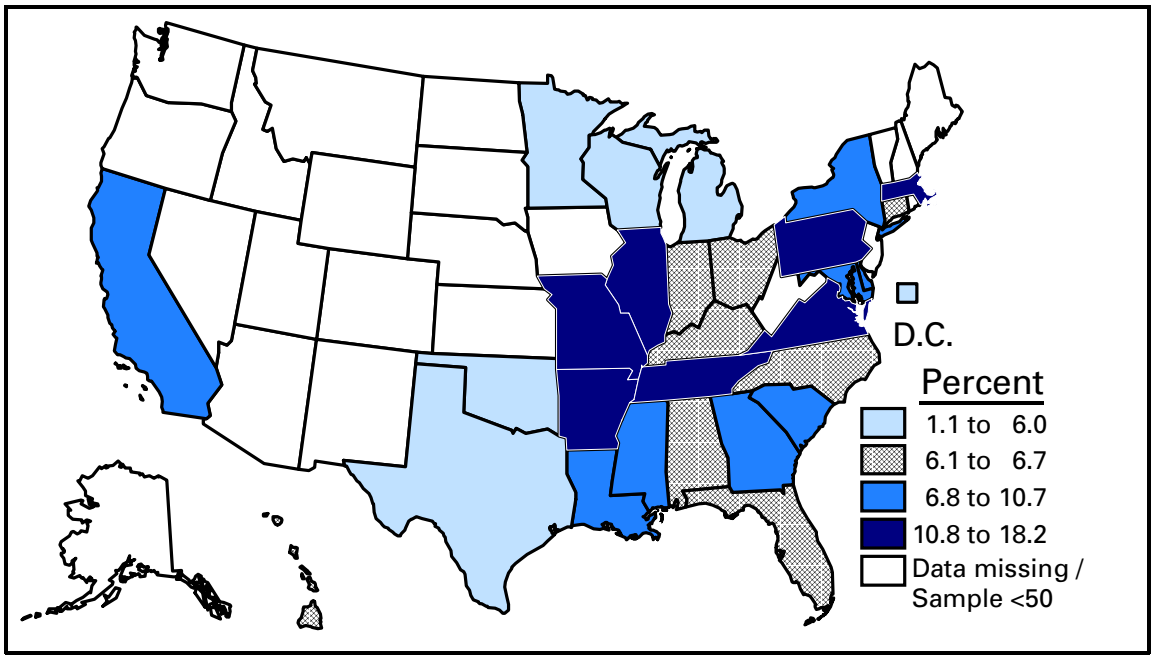
*Diabetes mellitus: Survey participants having ever been told by a physician that they have diabetes.

FIGURE 5c. Prevalence of diabetes mellitus* among black men — Behavioral Risk Factor Surveillance System, 1994



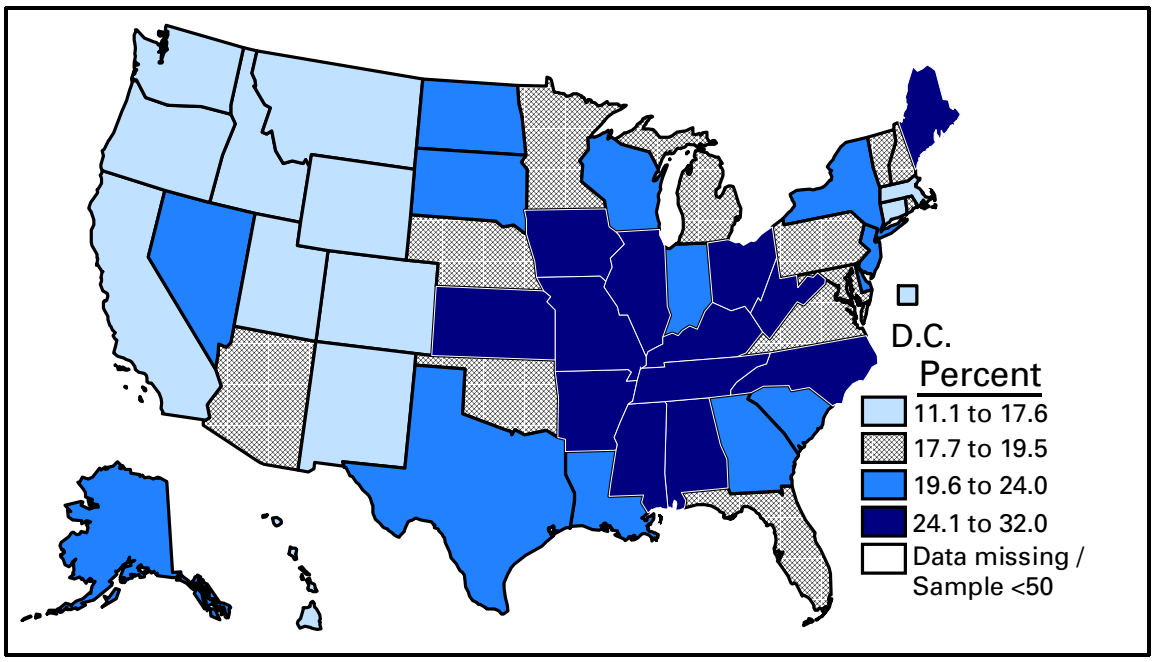
*Diabetes mellitus: Survey participants having ever been told by a physician that they have diabetes.

FIGURE 5d. Prevalence of diabetes mellitus* among black women — Behavioral Risk Factor Surveillance System, 1994



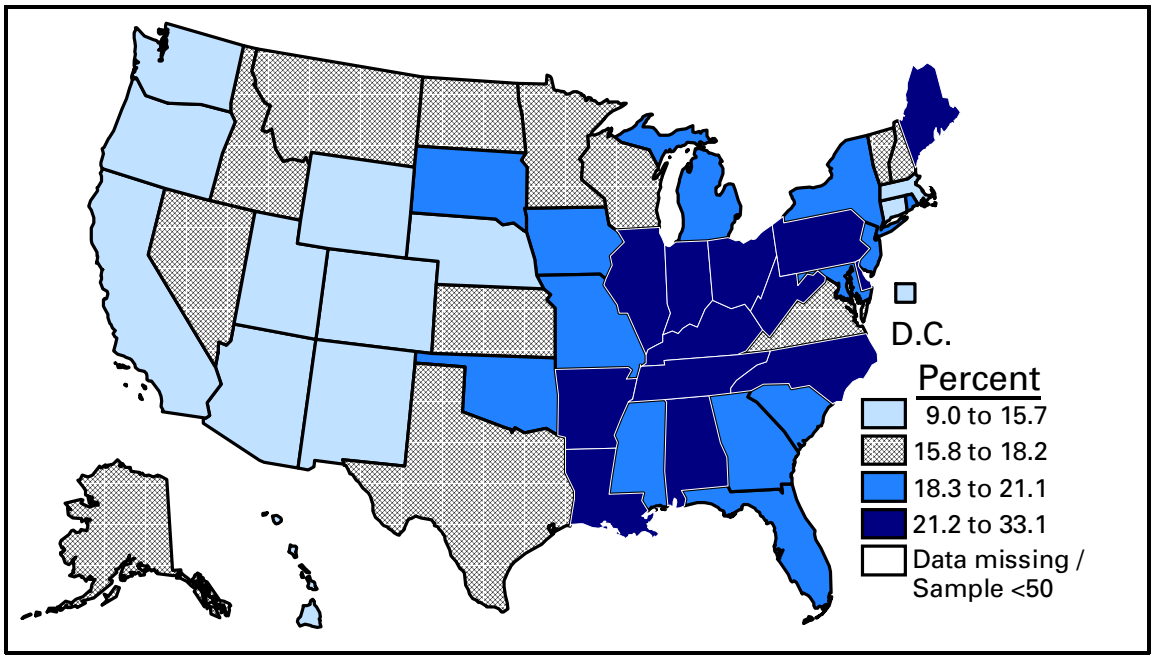
*Diabetes mellitus: Survey participants having ever been told by a physician that they have diabetes.

FIGURE 6a. Prevalence of risk factor combinations* among white men — Behavioral Risk Factor Surveillance System, 1994



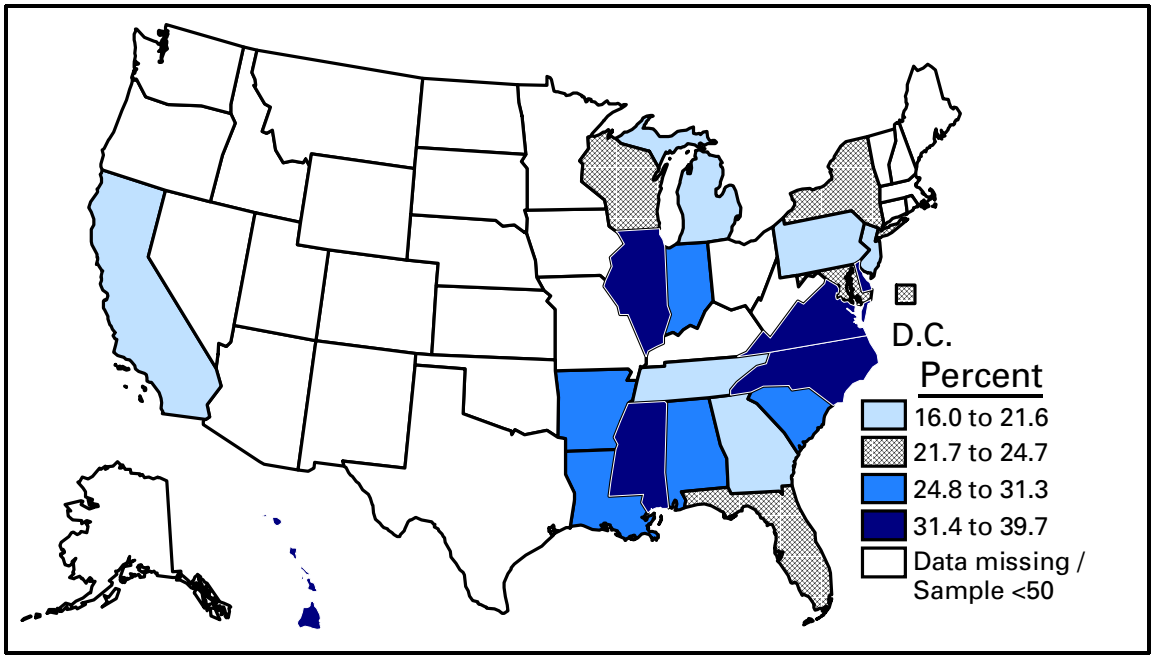
*Risk factor combinations: Survey participants having ≥2 of the following risk factors: overweight, physical inactivity, smoking, and diabetes mellitus.

FIGURE 6b. Prevalence of risk factor combinations* among white women — Behavioral Risk Factor Surveillance System, 1994



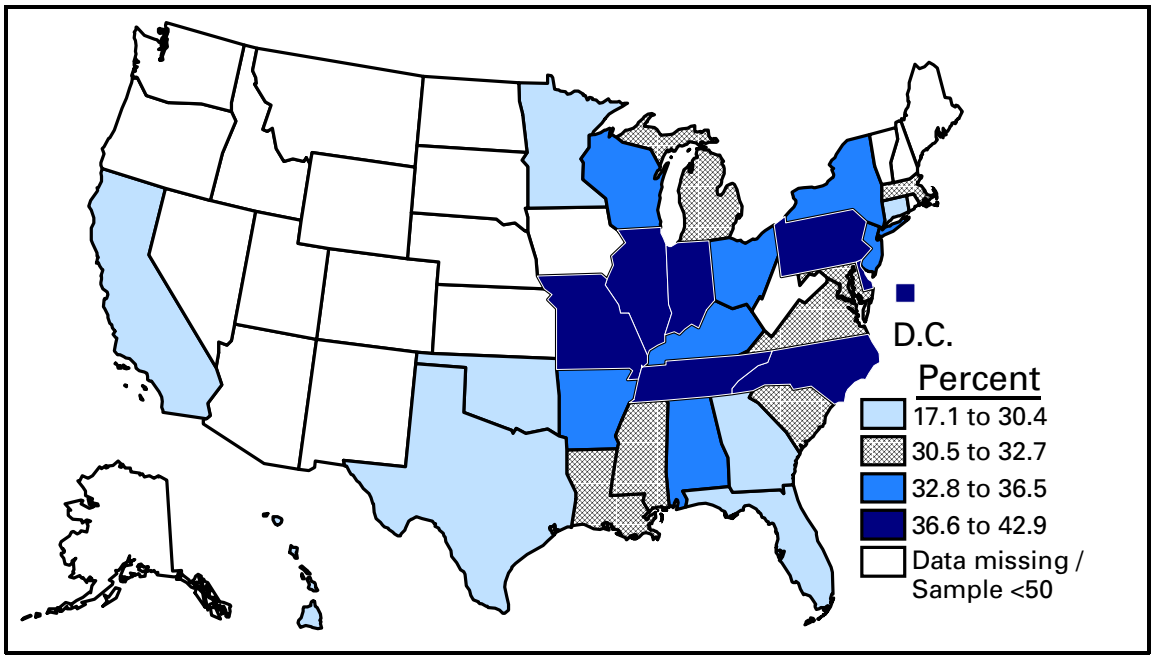
*Risk factor combinations: Survey participants having ≥2 of the following risk factors: overweight, physical inactivity, smoking, and diabetes mellitus.

FIGURE 6c. Prevalence of risk factor combinations* among black men — Behavioral Risk Factor Surveillance System, 1994



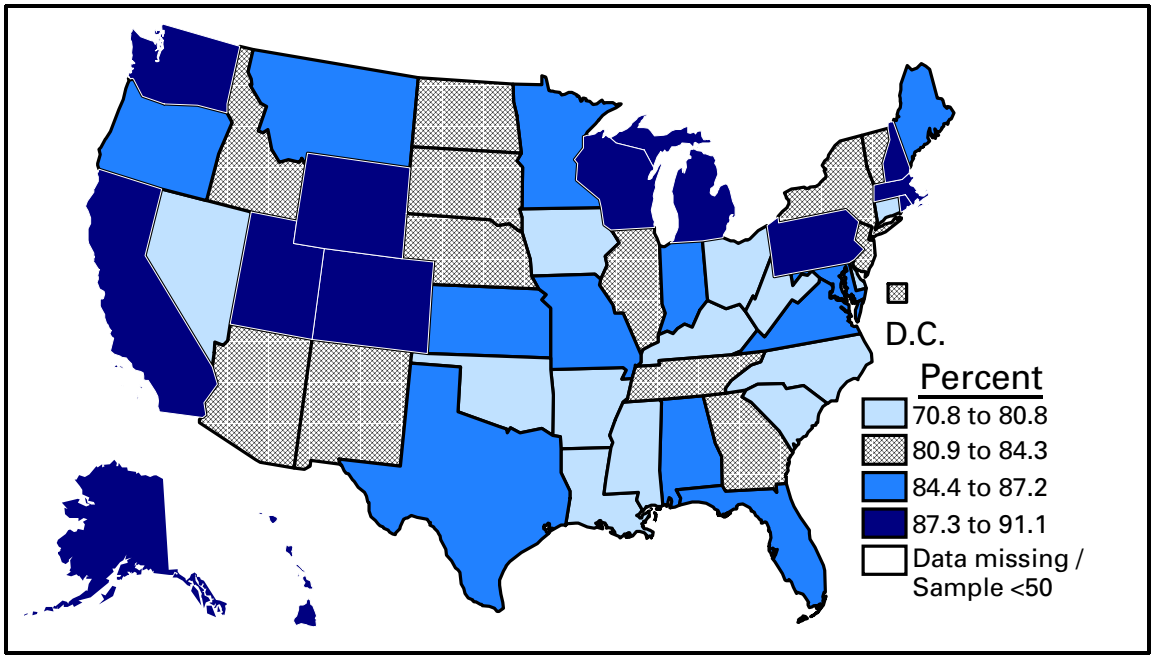
*Risk factor combinations: Survey participants having ≥2 of the following risk factors: overweight, physical inactivity, smoking, and diabetes mellitus.

FIGURE 6d. Prevalence of risk factor combinations* among black women — Behavioral Risk Factor Surveillance System, 1994



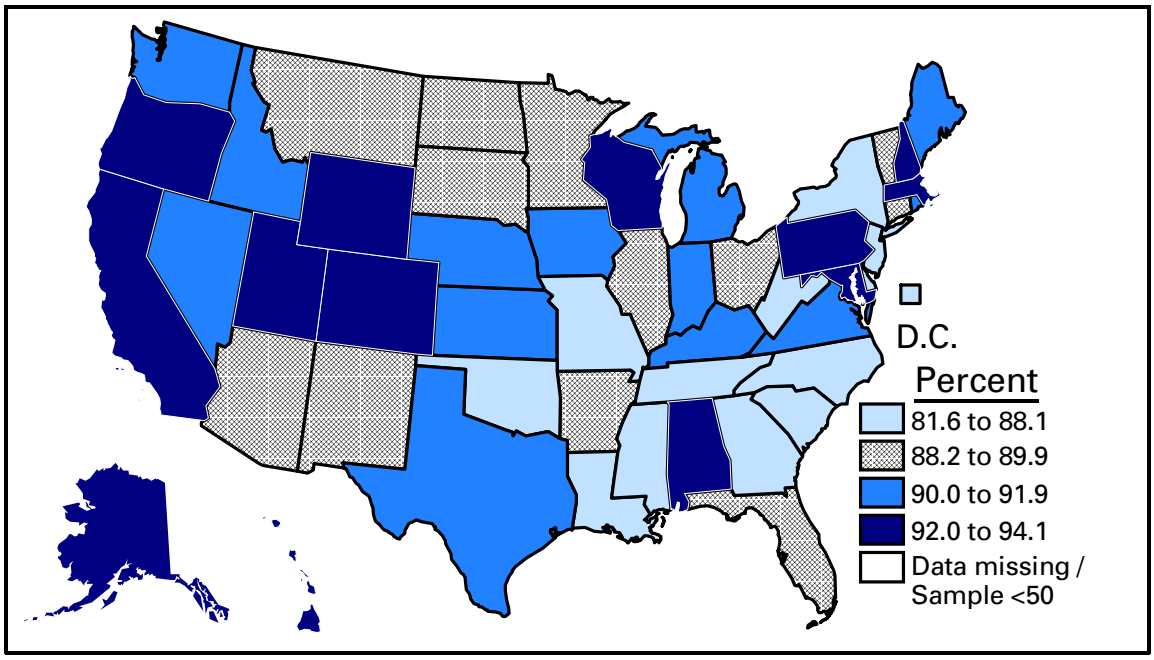
*Risk factor combinations: Survey participants having ≥2 of the following risk factors: overweight, physical inactivity, smoking, and diabetes mellitus.

FIGURE 7a. Prevalence of weight loss* among white men — Behavioral Risk Factor Surveillance System, 1994



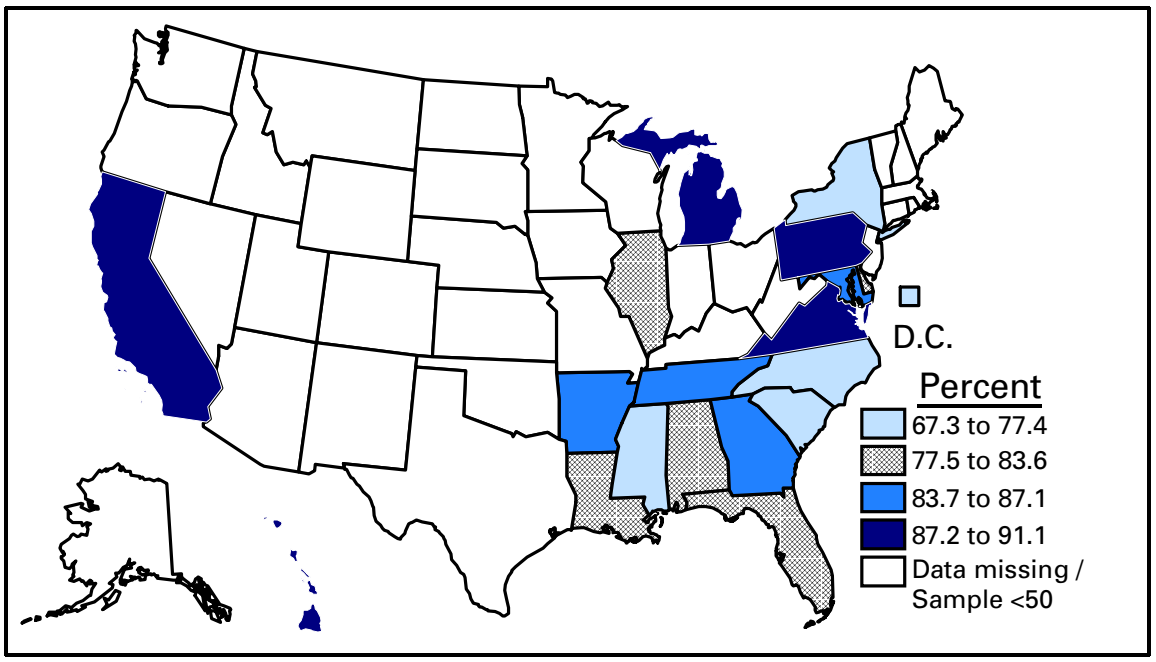
*Weight loss: Survey participants trying to lose or maintain or keep from gaining weight and who are either eating fewer calories or eating less fat or are using physical activity or exercise to maintain, lose, or keep from gaining weight.

FIGURE 7b. Prevalence of weight loss* among white women — Behavioral Risk Factor Surveillance System, 1994



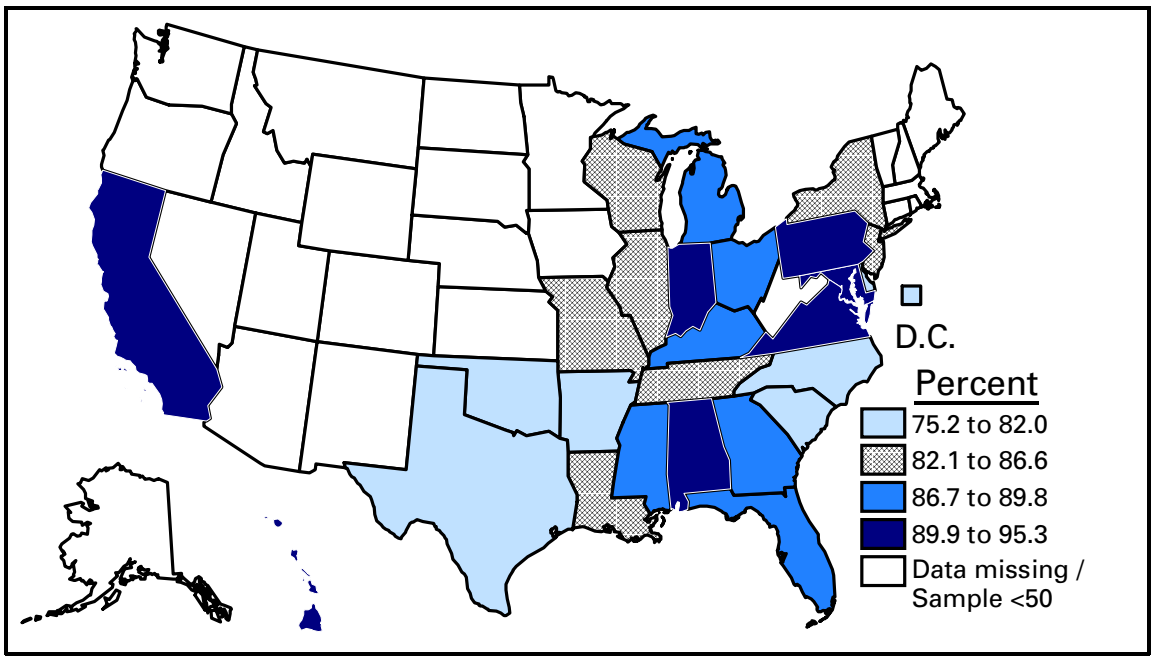
*Weight loss: Survey participants trying to lose or maintain or keep from gaining weight and who are either eating fewer calories or eating less fat or are using physical activity or exercise to maintain, lose, or keep from gaining weight.

FIGURE 7c. Prevalence of weight loss* among black men — Behavioral Risk Factor Surveillance System, 1994



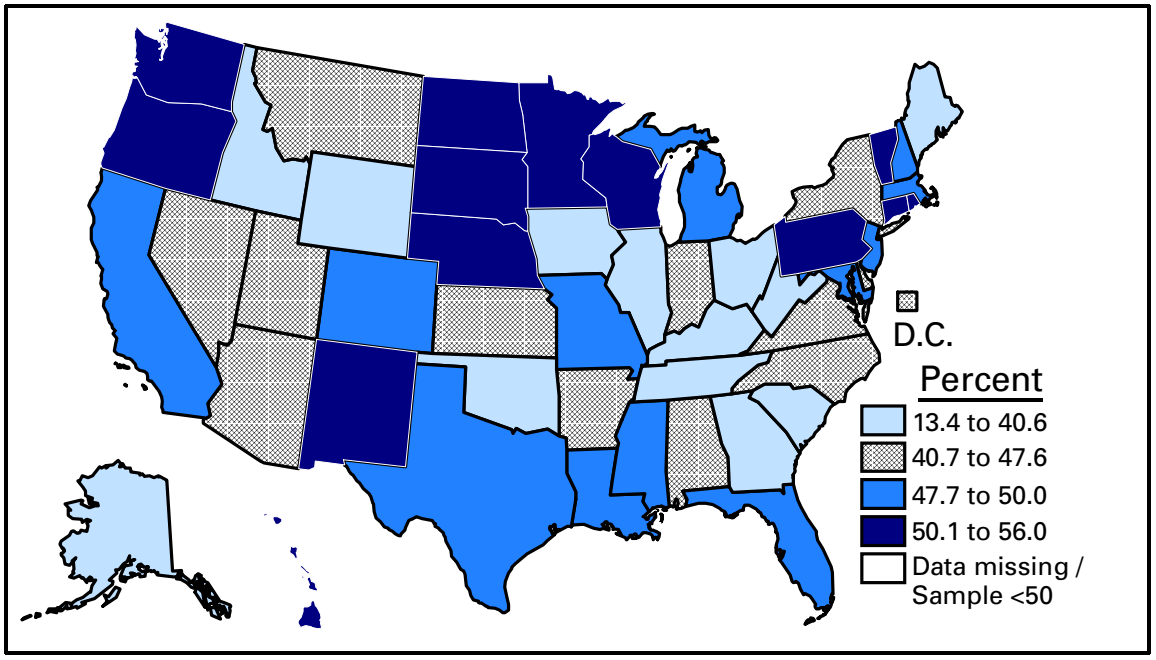
*Weight loss: Survey participants trying to lose or maintain or keep from gaining weight and who are either eating fewer calories or eating less fat or are using physical activity or exercise to maintain, lose, or keep from gaining weight.

FIGURE 7d. Prevalence of weight loss* among black women — Behavioral Risk Factor Surveillance System, 1994



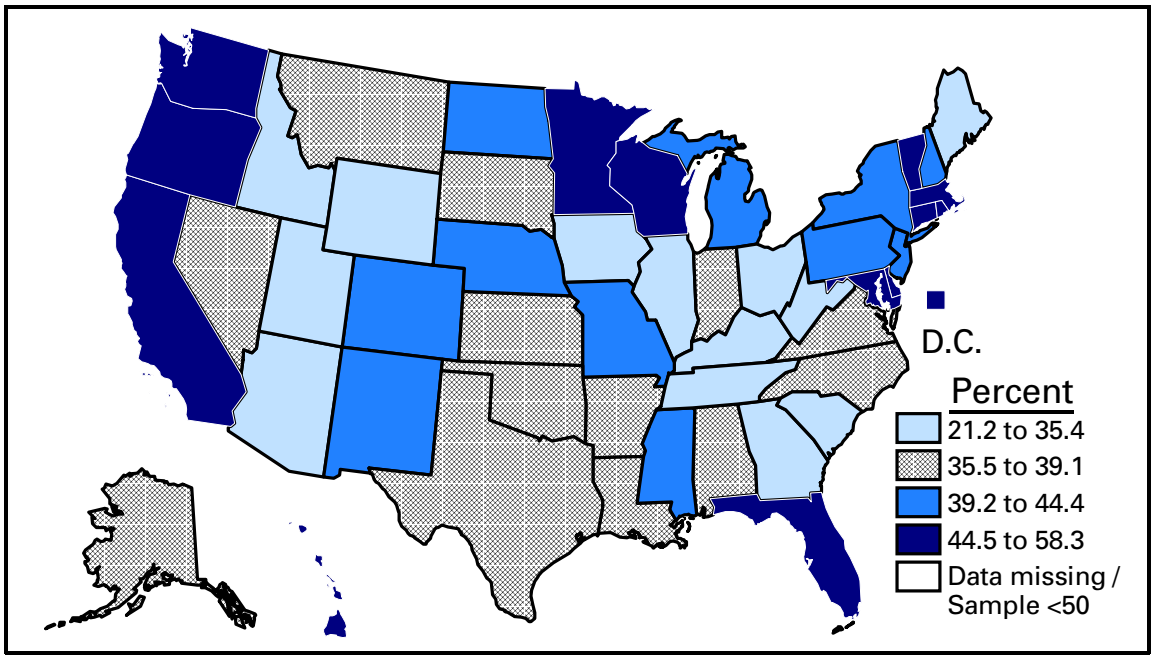
*Weight loss: Survey participants trying to lose or maintain or keep from gaining weight and who are either eating fewer calories or eating less fat or are using physical activity or exercise to maintain, lose, or keep from gaining weight.

FIGURE 8a. Prevalence of smoking cessation* among white men — Behavioral Risk Factor Surveillance System, 1994



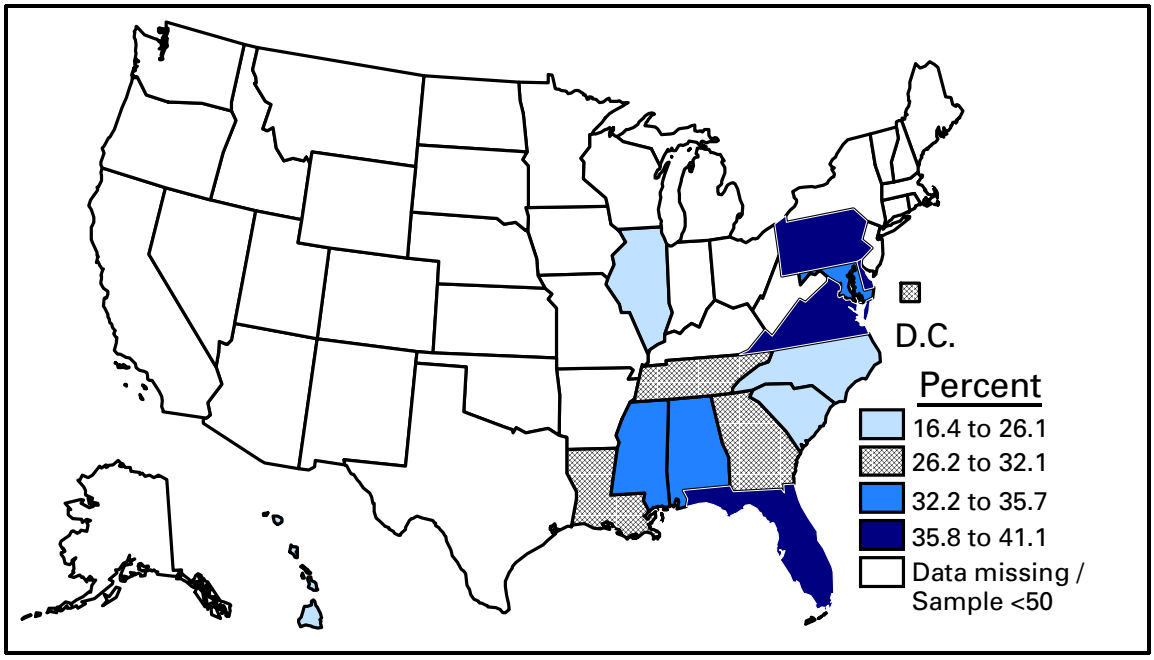
*Smoking cessation: Survey participants ever having smoked 100 cigarettes and having quit smoking for ≥12 months.

FIGURE 8b. Prevalence of smoking cessation* among white women — Behavioral Risk Factor Surveillance System, 1994



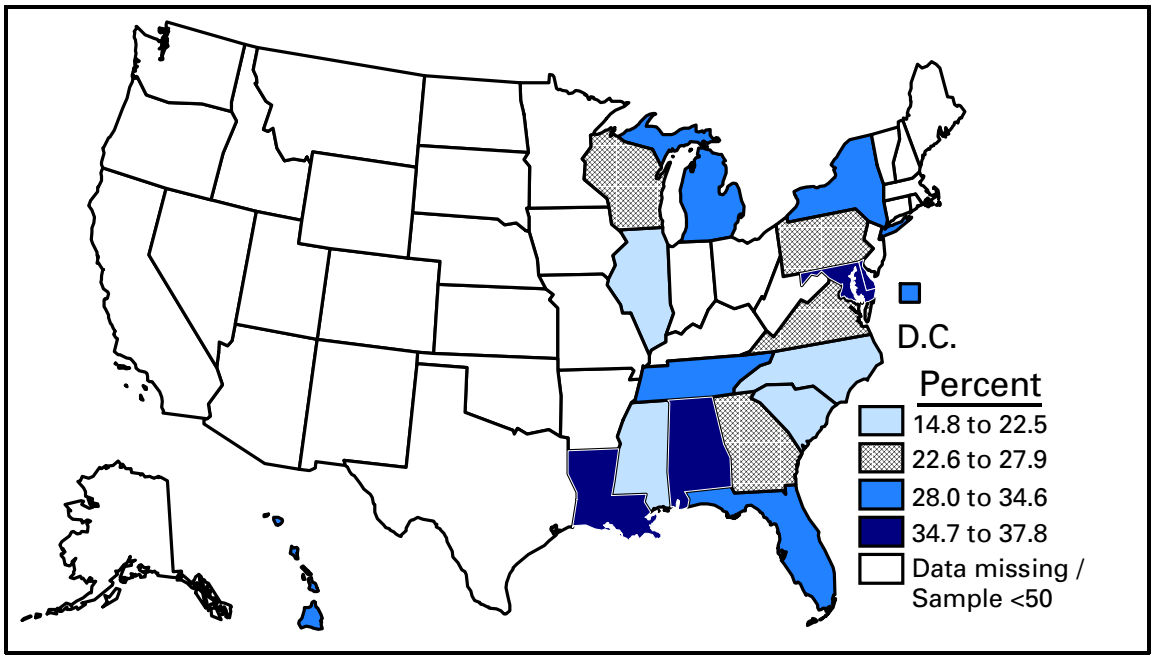
*Smoking cessation: Survey participants ever having smoked 100 cigarettes and having quit smoking for ≥12 months.

FIGURE 8c. Prevalence of smoking cessation* among black men — Behavioral Risk Factor Surveillance System, 1994



*Smoking cessation: Survey participants ever having smoked 100 cigarettes and having quit smoking for ≥12 months.

FIGURE 8d. Prevalence of smoking cessation* among black women — Behavioral Risk Factor Surveillance System, 1994



*Smoking cessation: Survey participants ever having smoked 100 cigarettes and having quit smoking for ≥12 months.

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 November 1998, and the laboratory directors listed below were in the positions shown as of November 1998.

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	Paul Kimsey, PhD
Colorado	Richard E. Hoffman, MD, MPH	Ronald L. Cada, DrPH
Connecticut	James L. Hadler, MD, MPH	Sanders F. Hawkins, PhD
Delaware	A. LeRoy Hathcock, PhD	Roy Almeida, DrPH
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	Paul Effler, MD, MPH	Vernon K. Miyamoto, PhD
Idaho	Christine G. Hahn, MD	Richard H. Hudson, PhD
Illinois	Byron J. Francis, MD, MPH	David F. Carpenter, PhD
Indiana	Gregory K. Steele, DrPH, MPH	David E. Nauth
Iowa	M. Patricia Quinlisk, MD, MPH	Mary J. R. Gilchrist, PhD
Kansas	Gianfranco Pezzino, MD, MPH	Roger H. Carlson, PhD
Kentucky	Glyn G. Caldwell, MD	Samuel Gregorio, DrPH (Acting)
Louisiana	Louise McFarland, DrPH	Henry B. Bradford, Jr, PhD
Maine	Kathleen F. Gensheimer, MD, MPH	John A. Krueger
Maryland	Diane M. Dwyer, MD, MPH	J. Mehsen Joseph, PhD
Massachusetts	Alfred DeMaria, Jr, MD	Ralph J. Timperi, MPH
Michigan	Mathew L. Boulton, MD, MPH	Robert Martin, DrPH, PhD
Minnesota	Michael T. Osterholm, PhD, MPH	Norman Crouch, PhD (Acting)
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	Mike Spence, MD
Nebraska	Thomas J. Safranek, MD	Steve Hinrichs, MD
Nevada	Randall L. Todd, DrPH	L.D. Brown, MD, MPH
New Hampshire	Jesse Greenblatt, MD, MPH	Veronica C. Malmberg, MSN
New Jersey	John H. Brook, MD, MPH	Thomas J. Domenico, PhD
New Mexico	C. Mack Sewell, DrPH, MS	David E. Mills, PhD
New York City	Benjamin A. Mojica, MD, MPH	Alex Ramon, MD, MPH
New York State	Perry F. Smith, MD	Ann Willey, PhD
North Carolina	J. Newton MacCormack, MD, MPH	Lou F. Turner, DrPH
North Dakota	Larry A. Shireley, MPH, MS	James D. Anders, MPH
Ohio	Thomas J. Halpin, MD, MPH	William Becker, DO
Oklahoma	J. Michael Crutcher, MD, MPH	Richard Baltaro, MD
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 Jr, PhD
South Carolina	James J. Gibson, MD, MPH	Harold Dowda, PhD
South Dakota	Susan E. Lance, DVM, PhD, MPH	Michael Smith
Tennessee	William L. Moore, Jr, 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	Robert B. Stroube, MD, MPH	James L. Pearson, DrPH
Washington	Juliet VanEenwyk, PhD (Acting)	Jon M. Counts, DrPH
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Wisconsin	Jeffrey P. Davis, MD	Ronald H. Laessig, PhD
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