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**Surveillance for Diabetes Mellitus —
United States, 1980–1989**

and

**Laboratory-Based Surveillance for
Meningococcal Disease in Selected
Areas — United States, 1989–1991**

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

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Among Black and Hispanic Children and Women of Childbearing Age	NCEHIC	1990; Vol. 39, No. SS-3
Behavioral Risk Factors	NCCDPHP	1991; Vol. 40, No. SS-4
Birth Defects		
B.D. Monitoring Program (see also Malformations)	NCEH	1993; Vol. 42, No. SS-1
Contribution of B.D. to Infant Mortality Among Minority Groups	NCEHIC	1990; Vol. 39, No. SS-3
Breast and Cervical Cancer	NCCDPHP	1992; Vol. 41, No. SS-2
<i>Campylobacter</i>	NCID	1988; Vol. 37, No. SS-2
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Cholera	NCID	1992; Vol. 41, No. SS-1
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Cytomegalovirus Disease, Congenital	NCID	1992; Vol. 41, No. SS-2
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Dental Caries and Periodontal Disease Among Mexican-American Children	NCPS	1988; Vol. 37, No. SS-3
Diabetes Mellitus	NCCDPHP	1993; Vol. 42, No. SS-2
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Ectopic Pregnancy, Mortality	NCCDPHP	1987; Vol. 36, No. SS-2
Elderly, Hospitalizations Among	NCCDPHP	1991; Vol. 40, No. SS-1
Endometrial and Ovarian Cancers	EPO, NCCDPHP	1986; Vol. 35, No. 2SS
<i>Escherichia coli</i> O157	NCID	1991; Vol. 40, No. SS-1
Evacuation Camps	EPO	1992; Vol. 41, No. SS-4
Foodborne Disease	NCID	1990; Vol. 39, No. SS-1
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Gonorrhea and Salpingitis, Teenagers	NCPS, NCID	1983; Vol. 32, No. 3SS
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Firearm-Related Deaths, Unintentional	NCEHIC	1988; Vol. 37, No. SS-1
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*All abbreviations are listed at end of inventory. Readers should check individual summaries when more than one CIO is responsible.

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Maternal Mortality	NCCDPHP	1991; Vol. 40, No. SS-2
Measles	NCPS	1992; Vol. 41, No. SS-6
Meningococcal Disease	NCID	1993; Vol. 42, No. SS-2
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In Meatpacking Industry	NIOSH	1985; Vol. 34, No. 1SS
State Activities	NIOSH	1987; Vol. 36, No. SS-2
Treated in Hospital Emergency Rooms	NIOSH	1983; Vol. 32, No. 2SS
Ovarian Cancer (see Endometrial and Ovarian Cancers)		
Parasites, Intestinal	NCID	1991; Vol. 40, No. SS-4
Pediatric Nutrition	NCCDPHP	1992; Vol. 41, No. SS-7
Pelvic Inflammatory Disease	NCPS	1983; Vol. 32, No. 4SS
Pertussis	NCPS	1992; Vol. 41, No. SS-8
Plague	NCID	1985; Vol. 34, No. 2SS
Plague, American Indians	NCID	1988; Vol. 37, No. SS-3
Pneumoconiosis, Coal Miners	NIOSH	1983; Vol. 32, No. 1SS
Poliomyelitis	NCPS	1992; Vol. 41, No. SS-1
Postneonatal Mortality	NCCDPHP	1991; Vol. 40, No. SS-2
Pregnancy Nutrition	NCCDPHP	1992; Vol. 41, No. SS-7
Pregnancy, Teenage	NCCDPHP	1987; Vol. 36, No. 1SS
Psittacosis	NCID	1983; Vol. 32, No. 1SS
Rabies	NCID	1989; Vol. 38, No. SS-1
Racial/Ethnic Minority Groups	Various	1990; Vol. 39, No. SS-3
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Reye Syndrome	NCID	1984; Vol. 33, No. 3SS
Rocky Mountain Spotted Fever	NCID	1984; Vol. 33, No. 3SS
Rotavirus	NCID	1992; Vol. 41, No. SS-3
Rubella and Congenital Rubella	NCPS	1984; Vol. 33, No. 4SS
<i>Salmonella</i>	NCID	1988; Vol. 37, No. SS-2
Salpingitis (see Gonorrhea and Salpingitis)		
Sexually Transmitted Diseases in Italy	NCPS	1992; Vol. 41, No. SS-1
Smoking	NCCDPHP	1990; Vol. 39, No. SS-3
Streptococcal Disease (Group B)	NCID	1992; Vol. 41, No. SS-6
Sudden Unexplained Death Syndrome Among Southeast Asian Refugees	NCEHIC, NCPS	1987; Vol. 36, No. 1SS
Suicides, Persons 15–24 Years of Age	NCEHIC	1988; Vol. 37, No. SS-1
Summer Mortality	NCEHIC	1983; Vol. 32, No. 1SS
Syphilis	NCPS	1991; Vol. 40, No. SS-3
Tetanus	NCPS	1992; Vol. 41, No. SS-8
Toxic-Shock Syndrome	NCID	1984; Vol. 33, No. 3SS
Trichinosis	NCID	1991; Vol. 40, No. SS-3
Tubal Sterilization Among Women	NCCDPHP	1983; Vol. 32, No. 3SS
Tuberculosis	NCPS	1991; Vol. 40, No. SS-3
Water-Related Disease	NCID	1991; Vol. 40, No. SS-3
Years of Potential Life Lost	EPO	1992; Vol. 41, No. SS-6

Abbreviations

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

Surveillance for Diabetes Mellitus — United States, 1980–1989

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Abstract

Problem/Condition: In the United States, diabetes mellitus is the most important cause of lower-extremity amputation and end-stage renal disease; the major cause of blindness among working-age adults; a major cause of disability, premature mortality, congenital malformations, perinatal mortality, and health-care costs; and an important risk factor for the development of many other acute and chronic conditions (e.g., diabetic ketoacidosis, ischemic heart disease, stroke). Surveillance data describing diabetes and its complications are critical to increasing recognition of the public health burden of diabetes, formulating health-care policy, identifying high-risk groups, developing strategies to reduce the burden of this disease, and evaluating progress in disease prevention and control.

Reporting Period Covered: In this report, data are summarized from CDC's diabetes surveillance system; trends in diabetes and its complications are evaluated by age, sex, and race for the years 1980–1989.

Description of System: CDC has established an ongoing and evolving surveillance system to analyze and compile periodic, representative data on the disease burden of diabetes and its complications in the United States. Data sources currently include vital statistics, the National Health Interview Survey, the National Hospital Discharge Survey, and Medicare claims data for end-stage renal disease.

*All the authors, with the exception of Dr. Eberhardt, were affiliated at the time of this report with the Division of Diabetes Translation, National Center for Chronic Disease Prevention and Health Promotion. Dr. Eberhardt was affiliated with the Office of Analysis and Epidemiology, National Center for Health Statistics.

Results and Interpretation: In 1989, approximately 6.7 million persons in the United States reported that they had diabetes mellitus, and a similar number probably had this disabling chronic disease without being aware of it. The disease burden of diabetes and its complications is large and is likely to increase as the population grows older. Effective primary, secondary, and tertiary prevention strategies are needed, and these efforts need to be intensified among groups at highest risk, including blacks. Important gaps exist in periodic and representative data for describing the disease burden.

Actions Taken: CDC is assisting diabetes control programs in 26 states and one territory. These programs attempt to reduce the burden of diabetes by preventing blindness, lower-extremity amputations, cardiovascular disease, and adverse outcomes of pregnancy among persons with diabetes. Because of important limitations in measuring the burden of diabetes, CDC is exploring sources of surveillance data for blindness, adverse outcomes of pregnancy, and the public health burden of diabetes among minority groups.

INTRODUCTION

In the United States, diabetes mellitus is the most frequent cause of blindness among working-age adults; the most important cause of nontraumatic lower-extremity amputation (LEA) and end-stage renal disease (ESRD); a major cause of congenital malformations, perinatal mortality, disability, premature mortality, and increased health-care costs; and an important risk factor for the development of many other acute (e.g., diabetic ketoacidosis [DKA]) and chronic conditions (e.g., ischemic heart disease [IHD], stroke) (1-3). Diabetes and its complications shorten life span, limit normal daily activities, create disability, increase use of health-care services, and impose an economic burden on persons who have the disease.

Surveillance data describing the magnitude of the disease burden of diabetes and its complications can be used to identify high-risk groups, develop strategies to reduce the burden associated with diabetes, help formulate health-care policy, and evaluate progress in disease prevention and control. CDC has established an ongoing diabetes surveillance system to compile and analyze periodic, representative data on the disease burden associated with diabetes and its complications in the United States. Data sources for the surveillance system include vital statistics, the National Health Interview Survey (NHIS), the National Hospital Discharge Survey (NHDS), and Medicare claims data. These data are analyzed to estimate diabetes incidence, prevalence, mortality, DKA, ESRD, cardiovascular disease (CVD), LEA, hospitalization, and disability. The results of these analyses for the period 1980-1989 are summarized in this report and presented in greater detail in *Diabetes Surveillance, 1991*.*

**Diabetes Surveillance, 1991* is the second report from CDC's diabetes surveillance system. A copy of this report is available from the National Center for Chronic Disease Prevention and Health Promotion, Rhodes-1113, MS-K13, Centers for Disease Control and Prevention, 4770 Buford Highway, N.E., Atlanta, GA 30341-3724.

DATA ANALYSIS

For this report, trends were evaluated in diabetes and its complications by age, sex, and race. The presentation of results was generally limited to specific subgroups for which relatively stable estimates could be obtained. In most circumstances, race-specific results were evaluated only for whites and blacks because the number of persons in the sample surveys analyzed was not sufficient for stable estimates to be calculated for other racial groups.

Diabetes incidence and prevalence rates and some diabetes mortality rates were calculated by using estimates of the resident population of the United States as the denominator. These rates were standardized by age according to the direct method, with the 1980 United States resident population as the standard population. LEA and ESRD incidence, disability prevalence, rates of hospital discharge, and some mortality rates were calculated by using estimates of the number of persons known to have diabetes as the denominator. These rates for diabetic populations indicate the risk of various complications among persons with diabetes and are useful for comparing complication rates among subgroups of persons with diabetes. Rates for the diabetic population were age standardized according to the direct method, with the 1980 United States population of persons with diabetes as the standard population.

Most of the data are based on samples of the population and are thus estimates, with margins of error due to both sampling and nonsampling error. Because of these margins of error, estimates may fluctuate from year to year.

DATA SOURCES

Incidence and Prevalence Data

Data Source: National Health Interview Survey

The incidence and prevalence of self-reported diabetes were determined from the 1980–1989 NHIS, conducted by CDC's National Center for Health Statistics (NCHS). The NHIS, which has been conducted since 1957, is an annual household survey of approximately 120,000 civilian, noninstitutionalized United States residents. The survey provides information on the health of the United States population, including information on the prevalence and incidence of disease, the extent of disability, and the utilization of health-care services. The NHIS has a multistage probability design (4).

Each year, a one-sixth subsample of NHIS respondents is asked whether anyone in the family has had diabetes in the past 12 months. If a household member has diabetes, the date of diagnosis is ascertained. In this report, diabetes prevalence was defined as the number of persons with diabetes. Diabetes incidence was defined as the number of persons who were diagnosed within the previous 12 months. Three-year moving averages were used to improve the precision of all estimates of incidence and of prevalence estimates among blacks.

Data Limitations: The NHIS underestimates the true prevalence of diabetes. About half of persons with diabetes are not aware that they have the disease (5). NHIS proxy respondents (i.e., household members responding for absent adult members) are also likely to underreport diabetes (5). In addition, the NHIS sample does not include per-

sons in institutional settings, who are more likely to have diabetes than are noninstitutionalized persons.

Mortality Data

Data Source: Multiple-Cause-of-Death Data

NCHS compiles and codes information on all deaths in the United States and releases annual multiple-cause-of-death data tapes. Data on these tapes include decedents' age, race, sex, and state of residence; the underlying cause of death; and contributing causes of death. Up to 20 causes of death for each decedent are coded according to the *International Classification of Diseases, Ninth Revision* (ICD-9).

Data tapes for the years 1980–1988 were used to extract information on deaths associated with diabetes (ICD-9 code 250) and to examine trends in diabetes as the underlying cause of death and as any listed cause of death. Among deaths for which diabetes was a listed cause, the analysis also examined deaths for which the corresponding underlying cause was DKA (ICD-9 code 250.1), major CVD (ICD-9 390–448), IHD (ICD-9 codes 410–414), or stroke (ICD-9 codes 430–434, 436–438).

Data Limitation: Diabetes is underreported on death certificates. Among persons known to have diabetes, only about 40% have diabetes listed as a cause of death and only 10% have diabetes recorded as the underlying cause of death (6,7). However, since underreporting is consistent over time and does not vary by race and sex, differences in temporal trends and relative differences between these groups are not likely to be attributable to reporting artifacts (8).

Hospitalization Data

Data Source: National Hospital Discharge Survey

Data from the 1980–1988 NHDS (9), also conducted by CDC's NCHS, were used to estimate diabetes-related hospital discharges. NHDS collects data on hospital discharges from a sample of short-stay, nonfederal hospitals in the United States. Data collected include information on patients' age, race, sex, and length of stay, up to seven diagnoses (one primary and six secondary diagnoses), and four surgical procedures.

The analysis estimated hospital discharges for which diabetes was the primary diagnosis (ICD-9 code 250) and for which diabetes was listed as any diagnosis. Among discharges with diabetes as a secondary diagnosis, discharges for which the primary diagnosis was DKA (ICD-9 code 250.1), major CVD (ICD-9 390–448), IHD (ICD-9 codes 410–414), or stroke (ICD-9 codes 430–434, 436–438) were estimated.

NHDS data were also used to examine the incidence of LEA. Incident cases were defined as discharges having diabetes as a listed diagnosis and an LEA (ICD-9 procedure code 84.1). Discharges with traumatic amputation procedural codes (ICD-9 procedure codes 895–897) were excluded.

Data Limitation: Hospitalizations related to diabetes may be underestimated by approximately 40% (10). Since NHDS samples hospital discharges and not persons, NHDS hospital discharge rates for diabetes-related diseases and procedures are influenced by persons who are hospitalized more than once for the same condition and hence may be sampled more than once.

End-Stage Renal Disease Data

Data Source: Management and Medical Information System, ESRD Program, Bureau of Data Management and Strategy, Health Care Financing Administration

ESRD is renal insufficiency requiring dialysis or kidney transplantation for survival. Because >90% of ESRD treatment in the United States is reimbursed by Medicare's ESRD program, 1980–1989 data from Medicare's medical information system were used to examine the incidence of ESRD attributed to diabetes mellitus (ESRD-DM). ESRD-DM incidence was defined as cases for which treatment was initiated for ESRD and for which diabetes was the primary cause of renal failure.

Data Limitations: Because ESRD-DM incidence was defined in terms of initiation of ESRD treatment, changes in incidence may be due to changes both in disease incidence and treatment practices. The latter may be influenced by changes in treatment availability and in the definition of treatment eligibility (11). In addition, the ascertainment of incident cases was incomplete, since Medicare reimburses only about 90% of ESRD treatment.

Disability Data

Data Source: National Health Interview Survey

Indicators of disability among persons with diabetes were derived from the 1983–1989 NHIS. Two of the major indicators of disability used in the NHIS are activity limitation and activity restriction. Activity limitation reflects a long-term reduction in activity resulting from one or more chronic diseases or impairments. Reduction in activity is measured in terms of activities normal for a person's age and sex group: "ordinary play" for children <5 years of age, "going to school" for children 5–17 years of age, "working at a job or business" or "keeping house" for persons 18–69 years of age, and independent performance of basic life activities (e.g., bathing, eating, shopping) for persons ≥70 years of age. Persons can be categorized as being a) unable to perform their major activity, b) able to perform their major activity but limited in the kind or amount of this activity, c) not limited in major activity but limited in other activities, and d) not limited in activity. This analysis examined persons unable to perform major activity (category a), limited in major activity (categories a–b), and limited in activity (categories a–c). Three-year moving averages were used to improve precision of the estimates.

The other major indicator of disability used in NHIS is activity restriction. This indicator refers to a reduction in activity due to either short-term or long-term conditions. Activity restriction is measured as school-loss days (for children ages 5–17), work-loss days (for currently employed persons ages 18–69), restricted-activity days (days in which persons limit their usual activities), and bed-rest days (inpatient hospital days and days in which a person stayed in bed for more than half a day because of illness or injury). The total number of restricted-activity days is the total number of days that a person experiences at least one of the above types of days. Because of small sample sizes, this analysis presents data in 3-year moving averages for total restricted-activity days and bed-rest days only.

Data Limitations: Although NHIS provides a stable source of annual estimates of disability, it does not sample the institutionalized United States population. Therefore, estimates of disability derived from NHIS underestimate the total amount of disability associated with diabetes.

Because NHIS changed the way it measured disability indicators in 1982, analysis of these indicators was limited to the years 1983–1989.

Population Data

Data Sources: 1980 Census estimates, 1981–1989 intercensal estimates, and National Health Interview Survey

Census estimates for 1980 and intercensal estimates for subsequent years were used to calculate rates (12). Estimates of the diabetic population (derived from NHIS) were used to calculate rates among persons with diabetes.

Data Limitations: For limitations of NHIS, see the above discussion of limitations under Incidence and Prevalence Data.

RESULTS

Prevalence and Incidence of Diabetes

In 1989, about 6.7 million persons in the United States (2.7% of the population) reported that they had diabetes. Although the prevalence of diabetes has increased since 1959, the overall rate of increase in the 1980s has slowed and reached a plateau (Table 1). Diabetes prevalence as estimated from NHIS data increased by 67% from 1959 through 1966, by 41% from 1966 through 1973, by 21% from 1973 through 1980, and by 4% from 1980 through 1989.

During the 1980s, the age-standardized prevalence of diabetes was higher among blacks than whites (Figure 1). Black males were the only group to show a marked increase (28%) in prevalence during the period 1980–1989. In 1989, the age-standardized prevalence of diabetes among blacks, regardless of sex, was approximately twice that for whites.

The number of new diabetes cases (i.e., diabetes incidence) averaged 648,000 per year. The incidence of diabetes increased in the early 1980s and then reached a plateau (Table 2). The lowest rates occurred among persons <45 years of age.

Mortality

Diabetes Mortality in the General Population

The annual number of deaths for which diabetes was listed as the underlying cause increased from 34,851 in 1980 to 40,368 in 1988. From 1982 through 1988, the age-standardized mortality rates for both black males and black females increased (by 23% and 11%, respectively) (Figure 2). The rates for white males and white females remained relatively constant.

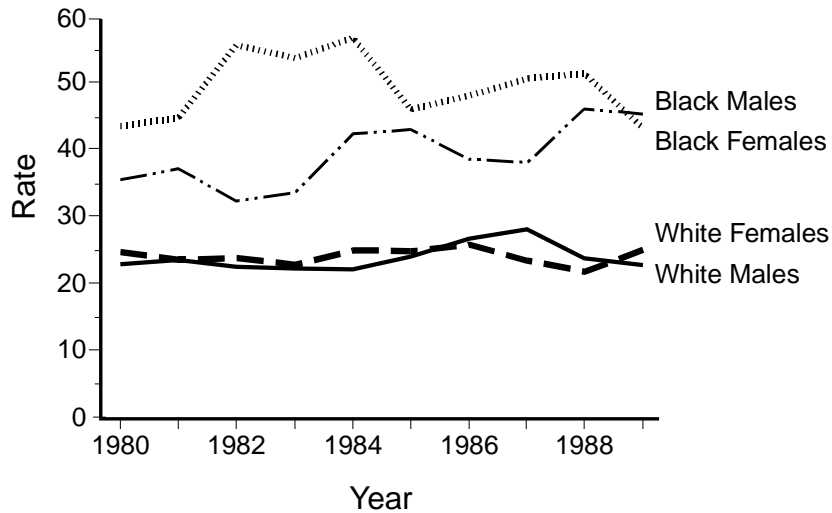
The annual number of deaths for which diabetes was listed as any cause of mortality (diabetes-related deaths) increased from 135,931 in 1980 to 157,265 in 1988. Race-sex temporal trends in age-standardized diabetes-related mortality were similar to those for diabetes as an underlying cause; mortality increased 21% among black

TABLE 1. Prevalence of diabetes per 1,000 population, by age group and year — United States, 1980–1989

Age (years)	Year										
	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	
≤44	Number*	1,010	952	984	1,002	994	1,038	1,047	1,364	1,092	1,224
	Rate	6.4	6.0	6.1	6.2	6.1	6.3	6.3	8.1	6.4	7.2
45–46	Number	2,389	2,545	2,567	2,600	2,360	2,331	2,849	2,553	2,460	2,707
	Rate	53.7	57.1	57.6	58.2	52.7	51.9	63.2	56.4	53.5	58.2
65–74	Number	1,522	1,364	1,525	1,304	1,756	1,849	1,592	1,703	1,704	1,632
	Rate	97.3	85.8	94.2	79.1	105.0	108.9	91.9	96.4	95.2	89.8
≥75	Number	861	948	810	874	1,090	1,101	1,284	1,195	1,094	1,096
	Rate	85.7	91.8	76.3	80.0	97.1	95.5	108.6	98.2	87.8	85.7
Total	Number	5,782	5,809	5,886	5,781	6,200	6,320	6,771	6,815	6,350	6,659
	Rate	25.4	25.1	25.2	24.5	25.9	26.2	27.8	27.7	25.6	26.6
Age-adjusted rate		25.4	25.3	25.4	24.7	26.0	26.2	27.9	27.6	25.4	26.3

*In thousands.

FIGURE 1. Age-standardized prevalence* of diabetes, by race, sex, and year — United States, 1980–1989



*Per 1,000 persons.

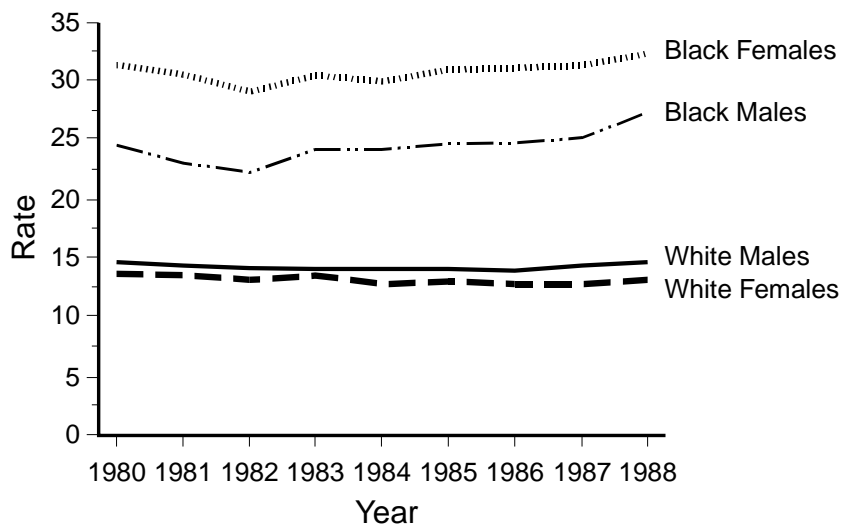
TABLE 2. Incidence* of diabetes per 1,000 population, by age group and year — United States, 1980–1989

Age (years)		Year									
		1980	1981	1982	1983	1984	1985	1986	1987	1988	1989
≤44	Number†	179	149	218	159	169	151	218	226	265	256
	Rate	1.07	1.18	1.11	1.14	0.99	1.10	1.21	1.35	1.50	1.56
45–64	Number	190	243	367	363	319	294	260	215	256	335
	Rate	4.94	6.07	7.34	7.91	7.34	6.54	5.73	5.29	5.90	6.45
≥65	Number	172	103	128	168	145	235	165	290	156	86
	Rate	5.65	5.44	5.25	5.68	6.91	6.73	8.34	8.18	6.18	4.18
Total	Number	541	495	713	690	833	879	644	731	678	677
	Rate	2.34	2.61	2.79	2.96	2.88	2.79	2.90	2.89	2.89	2.80
Age-adjusted rate		2.35	2.62	2.80	2.98	2.90	2.80	2.90	2.89	2.89	2.81

*Three-year moving average.

† In thousands.

FIGURE 2. Age-standardized mortality* for diabetes as underlying cause of death, by race, sex, and year — United States, 1980–1988



*Per 100,000 persons.

males (from 77.7 to 89.4 per 100,000) and 15% among black females (from 94.5 to 102.0 per 100,000). Diabetes-related mortality rates remained relatively constant for white males and white females.

From 1980 through 1988, diabetes-related mortality rates and mortality rates for diabetes as an underlying cause of death increased with age; the highest rates occurred among persons ≥ 85 years of age.

Diabetes Mortality in the Diabetic Population

When age-standardized mortality rates were calculated by using the number of persons known to have diabetes as the denominator, race-sex trends in age-standardized mortality were less clear. Age-standardized rates for diabetes as the underlying cause of death were lower in 1988 than in 1980 for white males, black males, and black females (Figure 3). White females were the only group whose rates were higher in 1988 than in 1980. Of the four race-sex groups examined, black males had the highest age-standardized rates.

Race-sex trends for diabetes-related mortality rates were similar to those for diabetes as the underlying cause of death. The highest age-standardized rates occurred among white males.

Hospitalizations

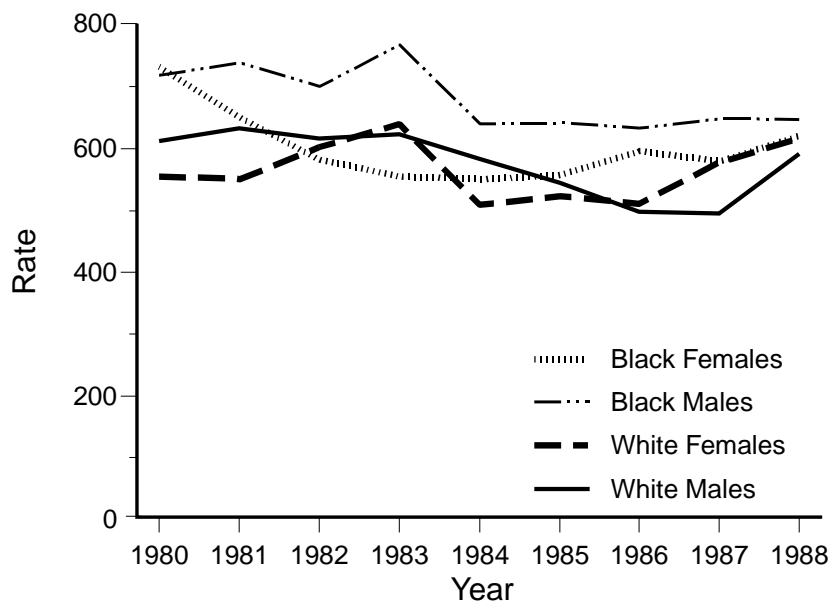
In 1988, diabetes was the primary diagnosis for 454,000 hospital discharges; it occurred as any one of seven listed diagnoses for 2.8 million hospital discharges. Age-standardized rates of hospital discharges for which diabetes was listed as the primary diagnosis increased slightly from 1980 through 1983 but then decreased and reached a plateau (Table 3). Age-specific rates were highest among persons < 45 years of age. The next highest rates were found among persons ≥ 75 years of age. The age groups of 45–64 years and 65–74 years had similar rates, which were the lowest in the analysis. Age-standardized rates were higher for blacks than whites, and black males had the highest rates (Figure 4).

In general, the diabetic population's age-standardized rates of hospital discharges that listed diabetes as a primary or secondary diagnosis showed temporal trends similar to those for diabetes as a primary diagnosis. However, age-specific rates increased with age. Among black females with diabetes, age-standardized rates increased 27% from 1983 through 1988 (from 324 to 411 per 1,000).

Cardiovascular Disease

In 1988, more than half of all diabetes-related deaths had major CVD listed as the underlying cause ($n=80,876$). Of these deaths from CVD, 61% ($n=49,433$) were attributable to IHD and 14% ($n=11,653$) to stroke. Age-specific rates for these diseases increase dramatically with age (Table 4). Among persons with diabetes, age-standardized mortality rates attributable to CVD, IHD, and stroke were lower in 1988 than in 1980; most of the decline occurred from 1983 through 1984. The declines for these 2 years were apparent among persons with diabetes who were ≥ 65 years of age. CVD mortality among persons with diabetes who were < 45 years of age tended to increase rather than decrease.

FIGURE 3. Age-standardized mortality* for diabetes as underlying cause of death among persons with diabetes, by race, sex, and year — United States, 1980–1988

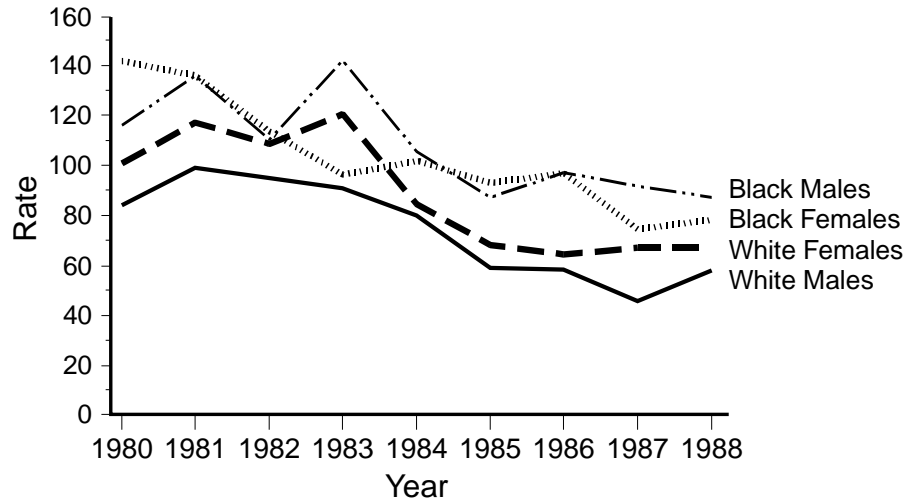


*Per 100,000 persons with diabetes.

TABLE 3. Hospital discharge rate for diabetes as primary diagnosis per 1,000 persons with diabetes, by age group and year — United States, 1980-1988

Age (years)	Year								
	1980	1981	1982	1983	1984	1985	1986	1987	1988
≤44	156.7	175.1	183.0	182.4	167.3	144.0	145.7	115.7	140.1
45-64	102.5	93.8	93.9	91.1	84.3	68.4	55.9	58.8	54.6
65-74	90.3	105.5	94.8	112.0	73.8	51.8	67.4	54.1	53.3
≥75	121.4	111.6	117.8	124.8	89.6	68.1	56.2	61.8	69.2
Total	111.6	112.8	112.3	116.7	95.6	75.9	72.5	69.5	71.5
Age-adjusted rate	111.6	113.7	113.3	117.5	96.8	77.2	74.6	67.9	71.3

FIGURE 4. Age-standardized rate* of hospital discharge with diabetes as primary diagnosis among persons with diabetes, by race, sex, and year — United States, 1980–1988



*Per 1,000 persons with diabetes.

TABLE 4. Mortality from major cardiovascular disease, ischemic heart disease, and stroke as underlying cause of death among persons with diabetes, per 100,000 diabetic population, by age group and year — United States, 1980–1988

Age (years)	Year								
	1980	1981	1982	1983	1984	1985	1986	1987	1988
Major cardiovascular disease									
≤44	82.6	90.3	87.3	95.3	106.3	95.9	106.1	83.5	102.6
45–64	624.9	597.4	579.4	586.4	646.1	657.0	533.8	591.8	602.1
65–74	1,512.5	1,669.3	1,502.1	1,813.7	1,360.3	1,318.2	1,502.3	1,440.5	1,421.4
≥75	4,273.2	3,820.6	4,566.4	4,407.8	3,560.0	3,571.6	3,106.7	3,341.9	3,721.7
Total	1,307.3	1,291.9	1,285.3	1,356.1	1,274.2	1,266.3	1,183.4	1,184.1	1,273.6
Age-adjusted rate	1,307.1	1,270.9	1,330.0	1,392.7	1,173.7	1,167.0	1,097.2	1,136.1	1,195.2
Ischemic heart disease									
<44	50.1	53.5	57.4	58.3	64.1	59.1	64.1	48.1	57.7
45–64	437.3	413.1	405.0	405.0	437.7	437.7	350.2	384.7	385.5
65–74	1,017.4	1,112.5	1,024.3	1,207.9	905.7	869.7	981.7	935.3	917.8
≥75	2,522.6	2,270.8	2,709.0	2,631.6	2,097.5	2,104.5	1,831.4	1,941.1	2,164.3
Total	833.0	821.5	824.6	862.8	802.2	792.4	735.4	727.7	778.4
Age-adjusted rate	832.9	811.0	850.4	887.3	742.8	733.5	687.0	702.7	733.3
Stroke									
<44	8.6	10.7	6.9	10.2	10.9	9.9	8.9	7.2	9.2
45–64	68.2	62.3	59.0	56.5	66.4	63.5	54.4	57.5	59.4
65–74	224.0	249.0	197.1	250.3	179.2	176.1	195.5	178.1	175.0
≥75	883.4	743.1	881.8	815.2	666.4	643.3	541.1	593.6	649.6
Total	220.2	208.7	199.4	206.9	194.9	188.7	172.9	171.5	183.5
Age-adjusted rate	220.2	203.8	208.8	212.4	175.7	170.1	156.1	160.3	169.0

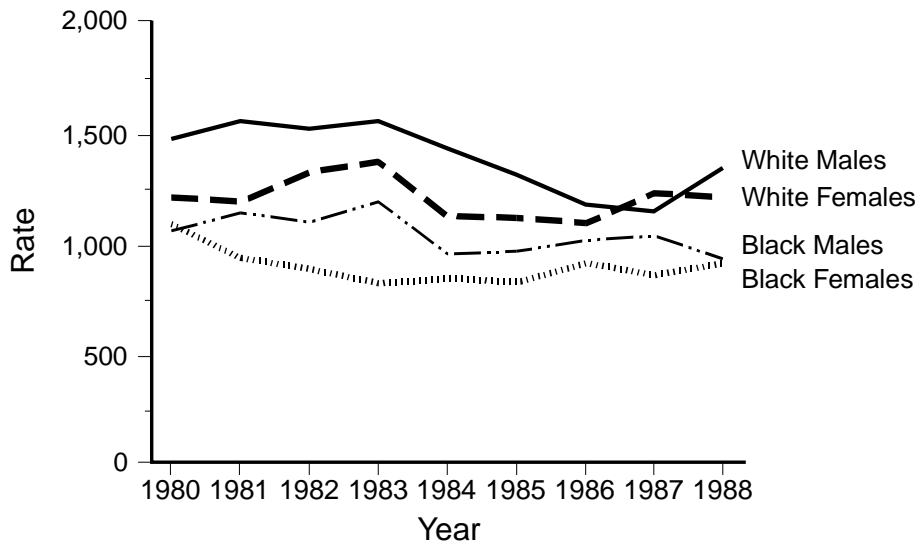
Of the four race-sex groups examined, white males had the highest age-standardized mortality rates for major CVD (Figure 5). Rates for black males, however, exceeded those for white males among persons <45 years of age. Although the age-standardized rates for white females were higher than those for black females, black females <65 years of age had higher rates than white females.

In 1988, 33% (908,000) of all diabetes-related hospitalizations listed major CVD as the primary diagnosis. Of these hospitalizations for CVD, 43% (n=386,000) were for IHD and 13% (n=114,000) were for stroke. Hospital discharge rates for major CVD and IHD among persons with diabetes increased from 1980 through 1988 (Figure 6). Although the trend for stroke was less clear, the rate was higher in 1988 than in 1980. In 1988, the age-standardized hospital discharge rates were 40% higher for major CVD, 42% higher for IHD, and 29% higher for stroke than in 1980. In general, hospital discharge rates for major CVD, IHD, and stroke increased with age. The rates of increase for major CVD and IHD were greater among persons <45 years of age.

Nontraumatic Lower-Extremity Amputation

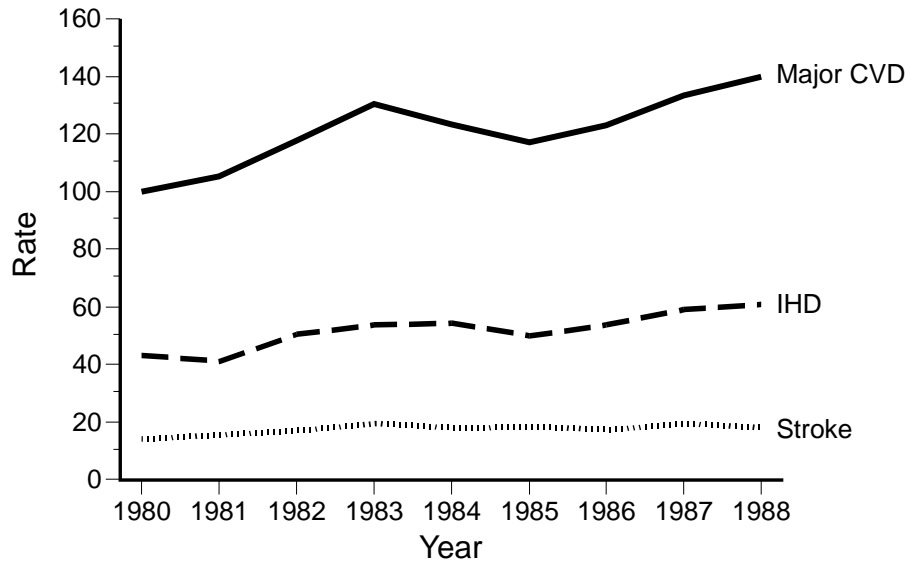
In 1988, 55,000 hospital discharges with LEA were reported among persons with diabetes (Table 5). This number represented approximately half of all hospital discharges with LEA. The incidence of LEA among persons with diabetes was relatively stable from 1980 through 1982, increased dramatically in 1983, and then leveled off (Table 5). Rates for LEA increased with age (Table 5) and were higher among males than among females and among blacks than among whites (Figure 7).

FIGURE 5. Age-standardized mortality* for major cardiovascular disease as underlying cause of death among persons with diabetes, by race, sex, and year — United States, 1980–1988



*Per 100,000 persons with diabetes.

FIGURE 6. Age-standardized rate* of hospital discharge for major cardiovascular disease (CVD), ischemic heart disease (IHD), and stroke as primary diagnosis among persons with diabetes, by year — United States, 1980–1988



*Per 1,000 persons with diabetes.

TABLE 5. Number of discharges for nontraumatic lower-extremity amputation and hospital discharge rate per 1,000 persons with diabetes, by age group and year — United States, 1980–1988

Age (years)		Year								
		1980	1981	1982	1983	1984	1985	1986	1987	1988
≤44	Number*	2	2	2	3	2	4	2	3	3
	Rate	1.6	1.6	1.6	2.8	2.4	4.2	2.3	2.1	2.5
45–64	Number	11	10	10	16	17	19	15	15	20
	Rate	4.7	3.8	3.9	6.1	7.2	8.1	5.4	5.8	8.0
65–74	Number	12	9	9	17	14	16	17	20	13
	Rate	7.6	6.4	6.0	13.1	8.0	8.6	10.5	12.0	7.5
≥75	Number	12	10	10	12	14	14	13	18	20
	Rate	13.5	10.5	12.6	13.2	12.9	12.4	10.2	14.8	18.0
Total	Number	36	30	31	47	47	53	48	56	55
	Rate	6.3	5.1	5.3	8.2	7.6	8.4	7.1	8.2	8.6
Age-adjusted rate		6.3	5.1	5.4	8.4	7.4	8.2	6.9	8.1	8.4

*In thousands.

Diabetic Ketoacidosis

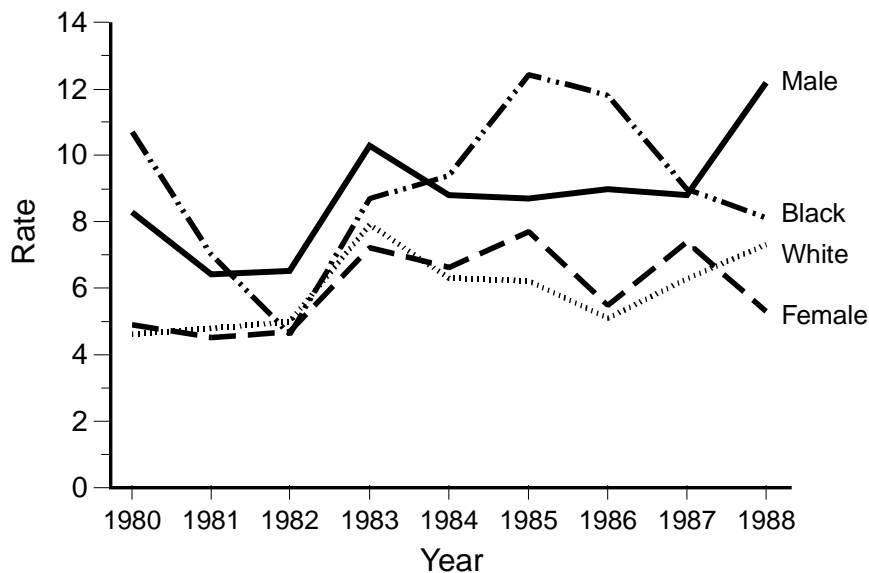
In 1988, DKA was the primary diagnosis for 84,000 hospital discharges. The age-standardized rates of hospital discharge for DKA increased from 1980 through 1984, decreased slightly in 1985, and then reached a plateau (Table 6). DKA hospital discharge rates were highest among persons <45 years of age. Among the race-sex groups examined, rates were higher among black males, followed by black females, white females, and white males. In 1988, the rate for black males was more than three times the rate for white males (26.8 vs. 7.9 per 1,000 diabetic population).

In 1988, DKA was listed as the underlying cause of 1,905 deaths. During the period 1980–1987, DKA mortality declined from 30.6 to 23.2 per 100,000 diabetic population. The rate then increased in 1988 to 27.7 per 100,000 persons with diabetes. The highest DKA mortality rates were among persons ≥ 75 years of age, followed by persons <45 years of age. Among the race-sex groups examined, age-standardized mortality rates were highest among black males, followed by black females and then by whites, with little difference between the sexes (Figure 8).

End-Stage Renal Disease

The number of new cases of ESRD-DM increased from 2,220 in 1980 to 13,332 in 1989. The age-standardized incidence rate of ESRD-DM among persons with diabetes increased more than fivefold, from 38.4 to 201.9 per 100,000 persons (Table 7). Age differences in incidence rates decreased during this decade. Rates were higher among

FIGURE 7. Age-standardized rate* of hospital discharge for nontraumatic lower-extremity amputation among persons with diabetes, by sex, race, and year — United States, 1980–1988

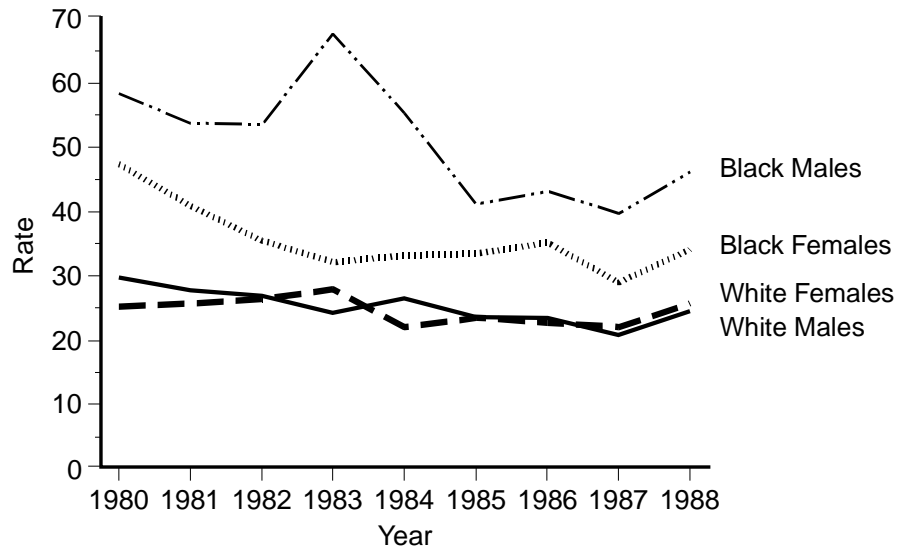


*Per 1,000 persons with diabetes.

TABLE 6. Hospital discharge rate for diabetic ketoacidosis as primary diagnosis per 1,000 persons with diabetes, by age group and year — United States, 1980–1988

Age (years)	Year								
	1980	1981	1982	1983	1984	1985	1986	1987	1988
≤44	36.6	39.8	40.3	51.5	58.6	51.7	55.2	49.9	59.3
45–64	5.2	7.1	5.7	4.5	6.9	6.9	5.7	5.2	3.8
≥65	4.2	3.6	2.5	3.9	3.6	3.8	4.0	3.9	3.6
Total	10.3	11.0	10.2	12.4	13.7	12.8	12.6	13.6	13.2
Age-adjusted rate	10.3	11.3	10.4	12.4	14.6	13.4	13.6	12.5	13.4

FIGURE 8. Age-standardized mortality* for diabetic ketoacidosis as underlying cause of death among persons with diabetes, by race, sex, and year — United States, 1980–1988



*Per 100,000 persons with diabetes.

blacks than among whites (Figure 9). Among both races, males had higher rates than females until 1986, when rates for black females began to exceed those for black males.

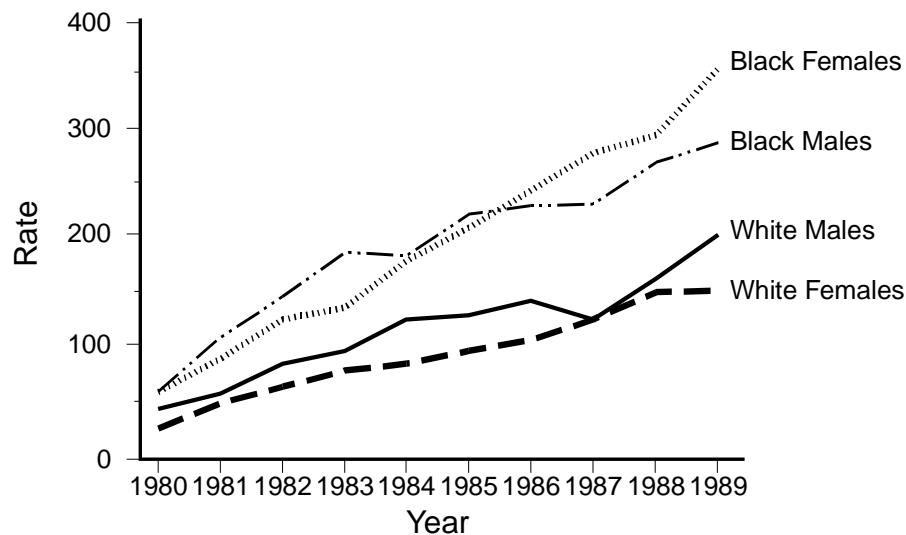
Disability

The number of persons with diabetes who reported that they were limited in activity increased from 3.1 million in 1983 to 3.3 million in 1989, approximately half of all persons with known diabetes (Figure 10). This proportion increased with age and tended to reach a plateau after age 64. In general, age-standardized rates of being limited in activity were greater among blacks than among whites and among females

TABLE 7. Rate of initiation of treatment for end-stage renal disease due to diabetes per 100,000 persons with diabetes, by age group and year — United States, 1980–1989

Age (years)	Year									
	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989
≤44	74.2	119.5	149.5	162.8	193.9	204.1	217.7	169.4	213.7	220.9
45–64	42.6	65.6	91.5	105.6	136.6	158.1	143.3	178.1	204.8	215.7
65–74	24.6	49.8	59.2	89.6	82.9	96.1	139.4	147.5	174.6	221.8
≥75	8.9	10.1	24.7	28.6	33.0	43.5	44.9	62.4	76.1	106.9
Total	38.4	61.7	83.6	100.3	112.4	127.5	135.2	148.4	176.1	200.2
Age-adjusted rate	38.4	62.6	83.2	99.9	117.1	132.7	140.6	151.3	179.2	201.9

FIGURE 9. Age-standardized incidence* of end-stage renal disease with diabetes as primary cause among persons with diabetes, by race, sex, and year — United States, 1980–1989



*Per 100,000 persons with diabetes.

than among males. From 1983 through 1989, rates decreased in all sex-race groups, except for white males.

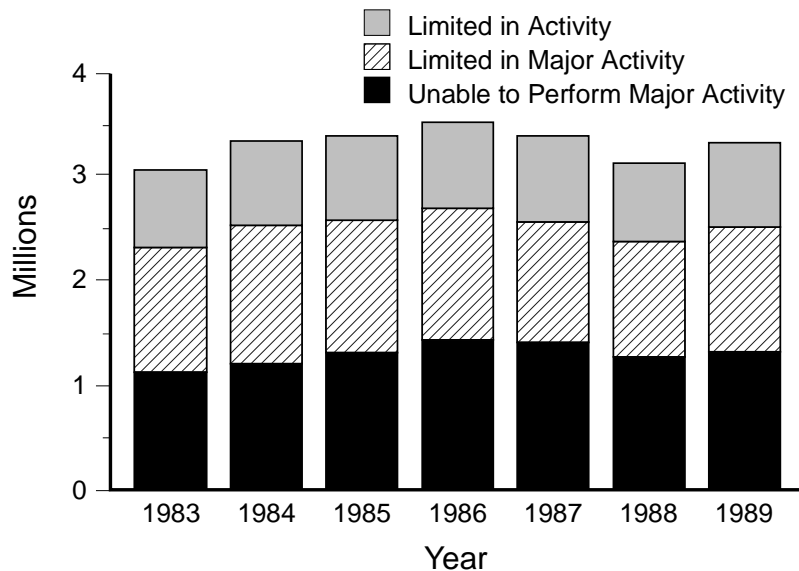
Approximately 40% of all persons with diabetes—and 75% of persons with diabetes who reported limitations—reported being limited in their major activity (Figure 10). About 20% of persons with diabetes reported that they were unable to perform their major activity.

The number of restricted-activity days among persons with diabetes increased from 199 million days in 1983 to 233 million days in 1989. During this period, persons with diabetes averaged 34 restricted-activity days per year; half of these days were bed-rest days. The average number of total restricted-activity days and bed-rest days was greater among blacks than whites (Figure 11).

DISCUSSION

Although the overall rate of increase in diabetes prevalence slowed and reached a plateau in the 1980s, the number of persons known to have diabetes increased by nearly 900,000 from 1980 through 1989. Because the prevalence of diabetes is associated with age, the number of persons with diabetes will continue to increase as the population ages, even if total population size and age-specific prevalence remain constant. Effective intervention strategies for preventing non-insulin-dependent diabetes mellitus (NIDDM), which accounts for 90%–95% of all prevalent cases, are urgently needed, particularly among groups at high risk for developing diabetes (e.g., blacks

FIGURE 10. Number* of persons with diabetes who report being limited in activity, limited in major activity, and unable to perform major activity, by year — United States, 1983–1989



*In millions; 3-year moving average.

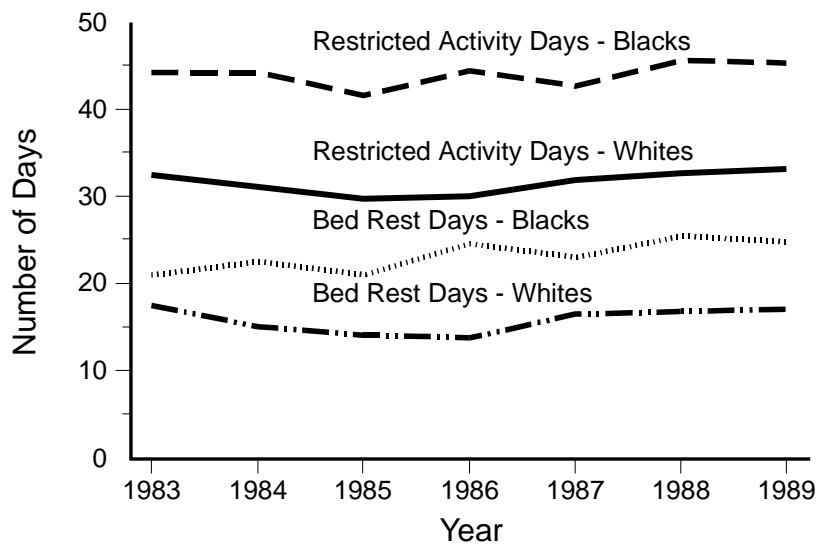
and other minority groups). These primary prevention strategies should focus on promoting healthful behaviors such as improved diet, exercise, and weight control.

Among persons with diabetes, major CVD accounted for more than half of all deaths and about one-third of all hospitalizations. Thus, the reduction of CVD risk factors among persons with diabetes could have a major effect on diabetes-related morbidity and mortality. Studies on the effectiveness of CVD risk-factor reduction among diabetic persons are needed. In the absence of such studies, prevention efforts should promote exercise, weight control, smoking prevention and cessation, hypertension prevention and blood pressure control, and lipid and glycemic control.

Temporal trends in hospital discharge rates varied by discharge diagnosis and must be interpreted with caution, because Medicare's implementation of the prospective reimbursement system in 1983 appears to have influenced hospitalization practices and disease reporting on discharge records. For example, the rate of LEA hospital discharges among persons with diabetes increased by >50% in 1983. This increase was probably due to increased reporting of diabetes among those having amputations rather than to increased incidence; the inclusion of diabetes as a contributing cause for LEA results in the assignment of that discharge to a higher reimbursement group.

The incidence of ESRD-DM increased dramatically in the 1980s. This increase may have been due to increases both in the incidence of ESRD-DM and in the use of treatment. Recommendations for preventing and slowing the progression of ESRD-DM among persons with diabetes include annual monitoring for early markers of renal disease among diabetic persons, controlling hypertension, and identifying and eliminating barriers to preventive care and treatment (13).

FIGURE 11. Age-standardized average number of restricted activity days and bed rest days* per person per year among persons with diabetes, by race and year — United States, 1983–1989



*Three-year moving average.

Many persons with diabetes have some degree of disability. Efforts to describe the disease burden of diabetes (and other chronic conditions) that do not consider disability will greatly underestimate the impact and burden of diabetes and its complications. Efforts to prevent disability among persons with diabetes should incorporate strategies to reduce the burden of diabetes and its complications, including CVD risk factor reduction, health promotion, improving access to health-care services and preventive care, patient and professional education, and incorporation of consensus standards of care (14) into health-care delivery systems. Diabetes control programs in 26 states and one territory are currently using these and other strategies to reduce the morbidity and mortality associated with diabetes among high-risk groups (e.g., the medically underserved and minority groups).

Black-white differences in trend were examined because diabetes and its complications disproportionately affect blacks and other minorities (1,3). Although genetic markers have not been identified for most forms of NIDDM, which accounts for 97% of incident cases, twin studies, studies of ancestral admixture, and other studies suggest that genetic factors play a strong role in the development of NIDDM (15-17). Not only are blacks and other racial/ethnic minorities more likely to develop diabetes, they are also at greater risk for many of the complications of diabetes (11,18,19). Our surveillance data indicate that blacks have higher rates of diabetes, of mortality with diabetes and DKA as underlying causes, of hospital discharges with diabetes and DKA as primary diagnoses, of LEA and ESRD-DM incidence, and of disability. It is unknown whether this increased risk of complications reflects more severe disease, barriers to health-care services, including preventive-care services, or the combination of these and other factors. Prevention efforts should be intensified in this high-risk population.

FUTURE DIRECTIONS

Although national data are available for many of the complications of diabetes, some important gaps remain. Diabetes is the leading cause of new cases of blindness among adults in the United States, but recent national data are not available for eye disease and blindness related to diabetes. Furthermore, although women with diabetes are known to be at increased risk for adverse outcomes of pregnancy, national data are not available for monitoring pregnancy outcomes among these women, nor are periodic, national data available on health-care practices and behaviors that could prevent many of the complications of diabetes. Periodic, representative data are also lacking for minority groups such as Hispanics and Native Americans, who are at increased risk for both diabetes and its complications. CDC is currently exploring possible data sources to address these important gaps.

References

1. Carter Center of Emory University. Closing the gap: the problem of diabetes mellitus in the United States. *Diabetes Care* 1985;8:391-406.
2. Center for Economic Studies in Medicine. Direct and indirect costs of diabetes in the United States in 1987. Alexandria, VA: American Diabetes Association, 1988.
3. Harris MI, Hamman RF, eds. *Diabetes in America*. Washington, DC: Department of Health and Human Services, NIH Publication No. 85-1468, 1985.
4. Massey JT, Moore TF, Parsons VL, Tadros W. Design and estimation for the National Health Interview Survey, 1985-94. Hyattsville, MD: National Center for Health Statistics. Vital and Health Statistics, Series 2, No. 110, 1989.

5. National Center for Health Statistics, Hadden WC, Harris MI. Prevalence of diagnosed diabetes, undiagnosed diabetes, and impaired glucose tolerance in adults 20–74 years of age, United States, 1976–80. Hyattsville, MD: National Center for Health Statistics. Vital and Health Statistics, Series 11, No. 237, 1987.
6. Harris MI, Entmacher PS. Chapter XXIX. Mortality from diabetes. In: Harris MI, Hamman RF, eds. Diabetes in America. Washington, DC: Department of Health and Human Services, NIH Publication No. 85–1468, 1985.
7. Ochi JW, Melton LJ, Palumbo PJ, Chu-Pin C. A population-based study of diabetes mortality. *Diabetes Care* 1985;8:224–9.
8. Wetterhall SF, Olson DR, DeStefano F, et al. Trends in diabetes and diabetic complications, 1980–1987. *Diabetes Care* 1992;15:960–7.
9. Graves EJ. Detailed diagnoses and procedures, National Hospital Discharge Survey, 1988. Hyattsville, MD: National Center for Health Statistics. Vital and Health Statistics, Series 13, No. 107, 1991.
10. Ford ES, Wetterhall SF. The validity of diabetes on hospital discharge diagnoses. *Diabetes* 1991;40(Suppl. 1):449a.
11. CDC. Diabetes surveillance, 1991. Atlanta: US Department of Health and Human Services, Public Health Service, 1992.
12. Irwin R. Intercensal estimates of the population by age, sex, race, 1980–1989. Alexandria, VA: Demo-Detail, 1989.
13. CDC. Incidence of treatment for end-stage renal disease attributed to diabetes mellitus — United States, 1980–1989. *MMWR* 1992;41:834–7.
14. CDC. The prevention and treatment of complications of diabetes: a guide for primary care practitioners. Atlanta: US Department of Health and Human Services, Public Health Service, 1991.
15. Zimmet P, Kirk RL, Serjeantson SW, King H. Genetic and environmental influence in the epidemiology of noninsulin-dependent diabetes mellitus: a global perspective. *Ann Acad Med (Singapore)* 1985;14:347–53.
16. Jarret RJ. Epidemiology and public health aspects of non-insulin-dependent diabetes mellitus. *Epidemiol Rev* 1989;11:151–71.
17. Stern MP. Primary prevention of type II diabetes mellitus. *Diabetes Care* 1991;14:399–410.
18. Stern MP, Haffner SM. Type II diabetes and its complications in Mexican Americans. *Diabetes Metab Rev* 1990;6:29–45.
19. Harris MI. Noninsulin-dependent diabetes mellitus in black and white Americans. *Diabetes Metab Rev* 1990;6:71–90.

Laboratory-Based Surveillance for Meningococcal Disease in Selected Areas, United States, 1989–1991

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Abstract

Problem/Condition: *Neisseria meningitidis* is a leading cause of bacterial meningitis and septicemia in the United States. Accurate surveillance for meningococcal disease is required to detect trends in patient characteristics, antibiotic resistance, and serogroup-specific incidence of disease.

Reporting Period Covered: January 1989 through December 1991.

Description of System: A case of meningococcal disease was defined by the isolation of *N. meningitidis* from a normally sterile site, such as blood or cerebrospinal fluid, in a resident of a surveillance area. Cases were reported by personnel in each hospital laboratory in the surveillance areas. The surveillance areas consisted of three counties in the San Francisco metropolitan area, eight counties in the Atlanta metropolitan area, four counties in Tennessee, and the entire state of Oklahoma.

Results: Age- and race-adjusted projections of the U.S. population suggest that approximately 2,600 cases of meningococcal disease occurred annually in the United States. The case-fatality rate was 12%. Incidence declined from 1.3/100,000 in 1989 to 0.9/100,000 in 1991. Seasonal variation occurred, with the highest attack rates in February and March and the lowest in September. The highest rates of disease were among infants, with 46% of cases affecting those ≤ 2 years of age. Males accounted for 55% of total cases, with an incidence of 1.2/100,000, compared with 1.0/100,000 among females (relative risk (RR) = 1.3, 95% confidence interval (CI) 1.0–1.6). The incidence was significantly higher among blacks (1.5/100,000) than whites (1.1/100,000) (RR = 1.4 [95% CI 1.1–1.8]). Serogroup B caused 46% of cases and serogroup C, 45%. Thirty-eight percent of isolates were reported to be resistant to sulfa; none were reported to be resistant to rifampin.

Interpretation: The decline in incidence of meningococcal disease from 1989 through 1991 cannot be explained by any change in public health control measures; this trend should be monitored by continued surveillance. The age, sex, and race distribution and seasonality of cases are consistent with previous reports. The proportion of *N. meningitidis* isolates resistant to sulfa continues to be substantial. A relatively small proportion of cases is potentially preventable by the use of the currently available polysaccharide vaccine, which induces protection against serogroups A, C, Y, and W135 and is effective only for persons >2 years of age.

Actions Taken: Current recommendations against the use of sulfa drugs for treatment or prophylaxis of meningococcal disease unless the organism is known to be sensitive to sulfa should be continued. Since resistance to rifampin is rarely reported, it continues to be the drug of choice for prophylaxis. The development of vaccines effective for infants and vaccines inducing protection against serogroup B would be expected to have a substantial impact on disease.

INTRODUCTION

Neisseria meningitidis is a leading cause of bacterial meningitis and septicemia in the United States (1). Although epidemic meningococcal disease continues to be a major public health problem in sub-Saharan Africa and other parts of the developing world, most disease in the United States is sporadic. Approximately half the cases in the United States are caused by serogroup B, with the highest attack rates in children <2 years of age (2). Efforts to control disease have been limited because the currently available quadrivalent meningococcal polysaccharide vaccine is effective only against serogroups A, C, Y, and W135 and is poorly immunogenic in children <2 years of age (3). Accurate surveillance for meningococcal disease is required to detect trends in a) patient characteristics, which may allow groups at higher risk of disease to be identified; b) antibiotic resistance, which influences the choice of antimicrobial agents for treatment and prophylaxis; and c) serogroup-specific incidence of disease, which influences the potential applications of the quadrivalent meningococcal polysaccharide vaccine. This report summarizes information from a laboratory-based surveillance system for invasive meningococcal disease conducted in a large U.S. population from January 1989 through December 1991.

METHODS

Laboratory-based surveillance, using methods previously described (1,2), was conducted in an aggregate population of 10.3 million persons (4.1% of the U.S. population), consisting of residents of three counties in the San Francisco metropolitan area, eight counties in the Atlanta metropolitan area, four counties in Tennessee, and the entire state of Oklahoma. Seventy-one percent of residents in the surveillance areas were white and 18% were black, compared with 80% and 12%, respectively, of the U.S. population. Surveillance was initiated in November 1988 and is ongoing; however, only cases reported from January 1, 1989, through December 31, 1991, are included in this report.

A case of meningococcal disease was defined by the isolation of *Neisseria meningitidis* from a normally sterile site, such as blood or cerebrospinal fluid (CSF), in a resident of a surveillance area. Cases were reported to surveillance workers by contacts in each hospital laboratory in the surveillance areas. A case report form with information about age, sex, race, outcome, and clinical syndrome of the patient, as well as the site of isolation, serogroup, and antibiotic sensitivities of the organism, was completed for each identified case. Hospital laboratory directors were asked to send *N. meningitidis* isolates to CDC for serogrouping; 65% of case isolates were received at CDC. To evaluate the sensitivity of reporting, hospitals were periodically audited by review of microbiology records. The audit completed in January 1990 identified 91 *N. meningitidis* cultures from sterile sites. Eighty-eight had been reported through the surveillance system, for a sensitivity of 96%.

Because race is a likely risk marker for meningococcal disease, data were analyzed by race, and the projected national incidence and annual number of cases based on incidence among surveillance area residents were adjusted for race. Races other than white or black were designated in <5% of records; therefore, only results of stratification by white and black race are reported.

Rates of disease were calculated by using 1990 population data from the U.S. Bureau of the Census. Fisher's exact test was used to assess statistical significance.

RESULTS

In the years 1989 through 1991 in the four surveillance areas, 332 cases of meningococcal disease were detected, for an average incidence of 1.1/100,000 population during this period. Based on this rate, when the data were adjusted for differences in racial distribution between the surveillance area population and the U.S. population, an estimated 2,600 cases of meningococcal disease occurred annually in the United States. The incidence was 1.3/100,000 in 1989, 1.0/100,000 in 1990, and 0.9/100,000 in 1991; this trend toward decreasing incidence with time was statistically significant (chi-square for linear trend, $p = 0.019$). Seasonal variation occurred, with the highest attack rates in February and March and the lowest in September (Figure 1). Of the 314 cases for which outcome information was available, 36 persons died, for a case-fatality rate of 12%. The case-fatality rate did not vary by sex, race, age, clinical syndrome, serogroup, or site of isolation of the organism.

The highest rates of disease occurred in infants <1 year of age, with a peak incidence of 26.4/100,000 population in infants <4 months of age (Figure 2). Twenty-nine percent of cases were in infants <1 year of age, 46% in children ≤ 2 years of age, and 25% in persons ≥ 30 years of age. The age distribution of cases did not vary by sex or race. Males accounted for 55% of total cases, with an incidence among males of 1.2/100,000, compared with 1.0/100,000 among females (RR = 1.3; 95% CI, 1.0–1.6). The incidence was significantly higher among blacks (1.5/100,000) than whites (1.1/100,000; RR = 1.4; 95% CI, 1.1–1.8).

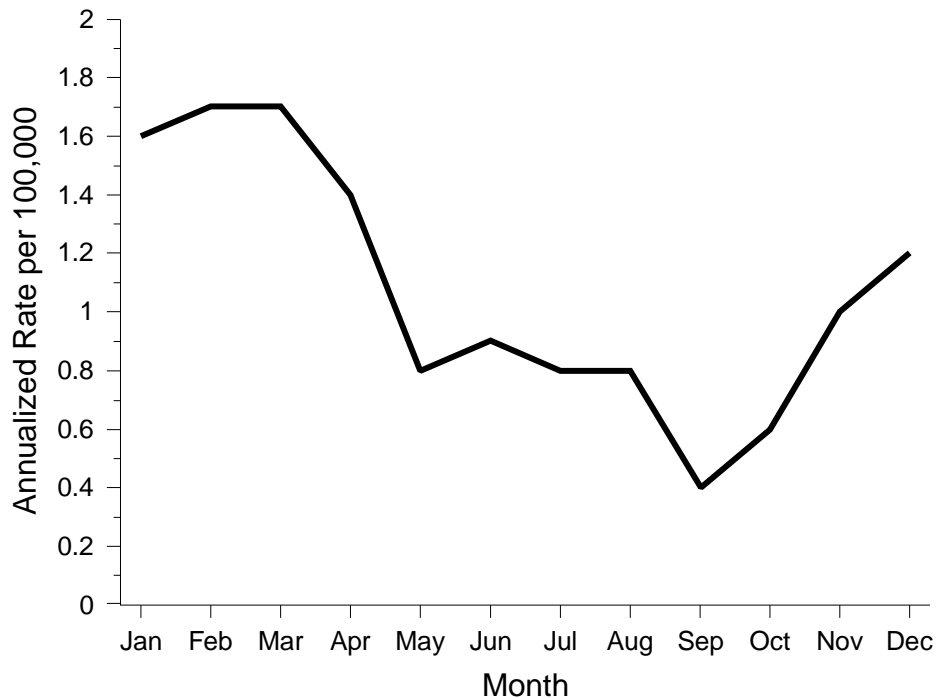
The average annual incidence of disease for the period 1989 through 1991 varied among the surveillance areas, with a peak of 1.3/100,000 in the San Francisco metropolitan area and a low of 0.6/100,000 in Oklahoma ($p < 0.001$). The incidence was higher among blacks than whites in all surveillance areas except Tennessee (Figure 3). The highest annual incidence, 1.9/100,000, occurred in San Francisco in 1989. An increased

rate of disease in the <2-year-old age group accounted for most of the increase in overall rate in this area during 1989. The attack rate in the <2-year-old age group in the San Francisco surveillance area was 23.0/100,000 in 1989, compared with 5.1/100,000 in 1990 and 10.2/100,000 in 1991. The cases among children ≤ 2 years old were not temporally clustered, and the serogroup distribution was similar to that reported for this age group in 1990 and 1991.

Meningitis, defined as *N. meningitidis* isolated from CSF and/or meningitis reported as the clinical syndrome on the case report form, accounted for 58% of cases, with an incidence of 0.6/100,000 population. The case-fatality rate for meningitic disease was 13% (25/192), which does not differ significantly from the case-fatality rate of 9% (13/140) for disease not meeting the above case definition for meningitic disease. The incidence of meningitic disease did not vary significantly by race; however, meningitis represented a higher proportion of total disease among whites (61%) than blacks (47%) ($p < 0.05$). The incidence of nonmeningitic disease declined after 12 months of age until age 20, when a secondary increase in incidence was seen that continued through the ≥ 60 age group (Figure 4). Nonmeningitic disease accounted for 63% of total disease in persons ≥ 30 years.

Meningitis was reported as the clinical syndrome on the case report form in 57% of cases and primary bacteremia in 49%. Other syndromes were much less common, with pneumonia reported in 4% of cases, otitis media in 2%, cellulitis in 0.6%, and arthritis in 0.3%. *N. meningitidis* was isolated from blood in 66% of cases, CSF in 51%,

FIGURE 1. Seasonal variation in incidence of meningococcal disease, selected U.S. areas, 1989–1991

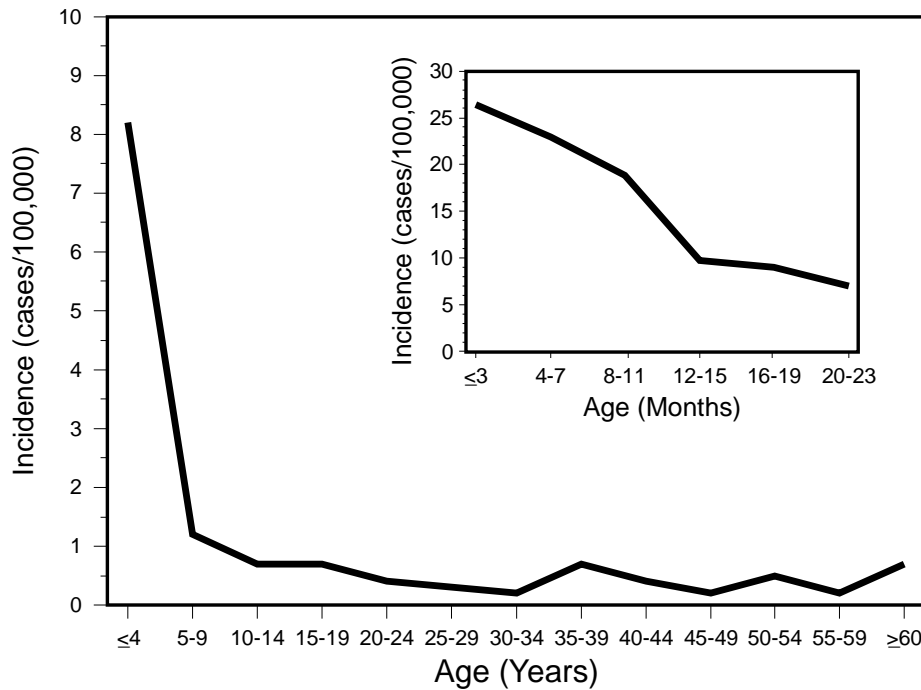


joint fluid in 1%, and pleural and peritoneal fluid in 0.3% each. More than one clinical syndrome or site of isolation was reported for some cases. The case-fatality rate when the organism was isolated from blood (regardless of whether it was also isolated from another site) was 11.5% (25/218); the case-fatality rate when the organism was isolated only from CSF was 9.3% (10/110).

A total of 217 isolates were serogrouped at CDC; serogroup information was collected locally and recorded on the case report form for 179. Serogroup data were available from either or both sources for 261 (79%) of cases. For 132 isolates, serogroup information was reported from both sources; the sources agreed in 118 (89%) cases. When a discrepancy occurred, the CDC results were used. The proportion of cases with serogroup information did not differ by race.

Serogroup B organisms accounted for 46% and serogroup C organisms for 45% of isolates for which serogroup information was available. W135, Y, and nontypeable serogroups accounted for 3%, 2%, and 2% of isolates, respectively. One isolate was reported to be serogroup A, and two others were reported as "other" serogroup; these isolates were not submitted to CDC for serogrouping. The estimated serogroup-specific incidences were 0.5/100,000 population for both serogroups B and C. Serogroup B disease accounted for 51% of disease among whites but for only 27% among blacks ($p < 0.01$). A significantly higher incidence of group C disease (1.0/100,000 vs. 0.4/100,000 [$p < 0.01$]) and a similar (not significantly different)

FIGURE 2. Incidence of meningococcal disease, by age group, selected U.S. areas, 1989-1991



incidence of group B disease (0.4/100,000 vs. 0.6/100,000; $p = 0.4$) occurred among blacks compared with whites.

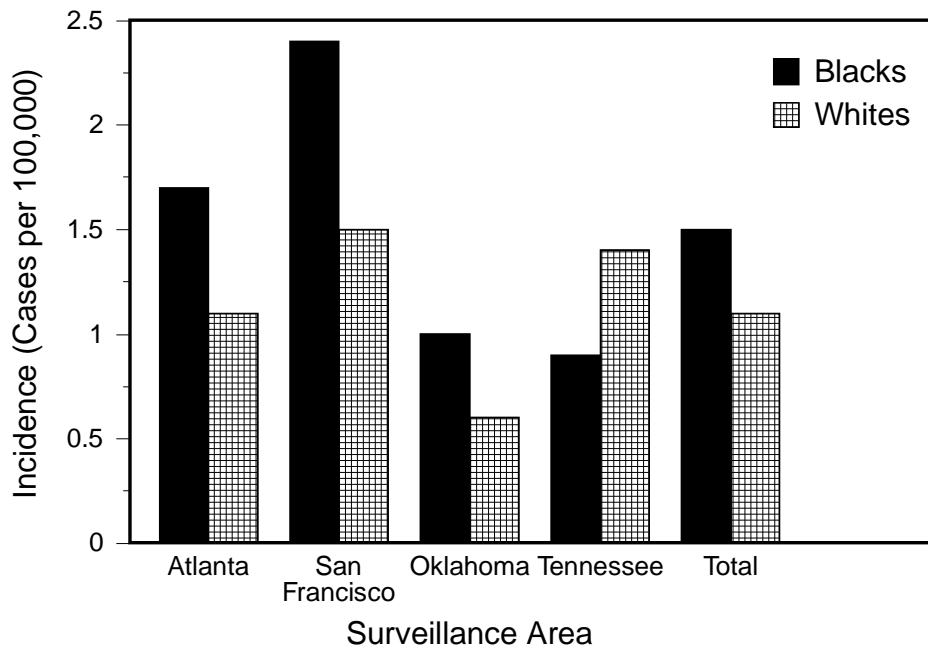
A significantly higher proportion of group C disease occurred in older age groups, with 69% of group C disease, compared with 37% of group B disease, in persons >2 years of age ($p < 0.001$) (Table 1). The incidence of group C disease varied by surveillance area, with rates of 0.4/100,000 and 0.2/100,000 in Tennessee and Oklahoma, compared with a rate of 0.7/100,000 both in Atlanta and San Francisco. Rates of group B disease varied less by surveillance area (Figure 5).

The proportion of meningitic and nonmeningitic disease caused by serogroups B and C was similar. Serogroups other than B and C accounted for four (44%) of nine pneumonia cases, compared with only 19 (6%) of 323 cases of all other syndromes ($p = 0.002$). Twenty-eight (38%) of the 73 isolates for which sulfa sensitivity was reported were reported to be resistant. Sulfa resistance did not vary by serogroup. None of the 42 isolates for which rifampin sensitivity was reported were found to be resistant.

DISCUSSION

The average annual rate of disease, 1.1/100,000 population, detected by this laboratory-based surveillance system in an aggregate population of 10 million persons from 1989 through 1991 was slightly lower than the rate of 1.3/100,000 population detected by a similar surveillance system in an aggregate population of 34 million in 1986 (2). The incidence declined substantially, from 1.3/100,000 in 1989 to 0.93/100,000 in 1991.

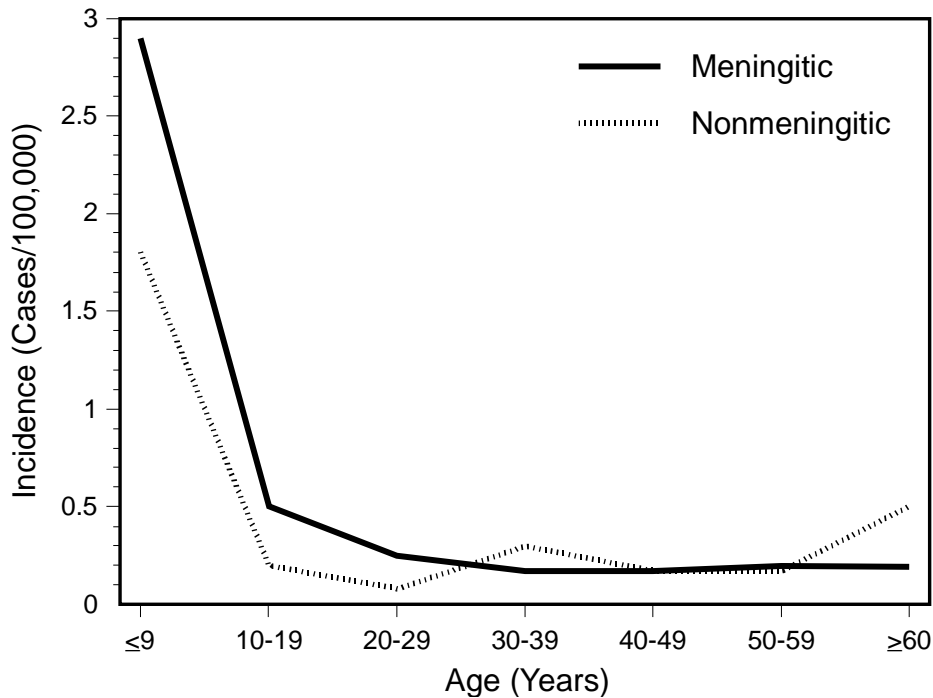
FIGURE 3. Incidence of meningococcal disease, by race, selected U.S. areas, 1989–1991



This decrease in the rate of disease cannot be attributed to public health control measures adopted during this period. The decline in incidence therefore could represent a natural fluctuation in disease incidence or could indicate a change in the occurrence of disease, possibly due to factors such as changes in the population's susceptibility to disease or in the organism's prevalence or virulence. The rate of meningococcal disease in the United States, as reported by CDC through the National Notifiable Diseases Surveillance System, also declined over a similar period, suggesting that the decrease was not limited to the geographic areas included in the laboratory-based surveillance system. The rate of meningococcal disease reported by CDC decreased from 1.2/100,000 population in 1988 to 1.1/100,000 population in 1989, 1.0/100,000 population in 1990, and 0.8/100,000 population in 1991 (chi-square test for linear trend, $p < 0.001$) (4). Continued surveillance for meningococcal disease is needed to determine the importance of this trend.

The age, sex, and race distribution and seasonality of cases reported by this laboratory-based surveillance system are consistent with previous reports. The higher attack rate among males, who accounted for 55% of total cases, was not found in 1986; however, higher rates of meningococcal meningitis in males have been reported previously (5,6). The 1986 laboratory-based surveillance system detected a higher rate of invasive meningococcal disease among blacks (2), and a report of bacterial meningitis passive surveillance data from 1978 through 1981 also noted higher rates of meningococcal meningitis among blacks compared with whites (5). Previous

FIGURE 4. Incidence of meningitic and nonmeningitic meningococcal disease, by age group, selected U.S. areas, 1989-1991



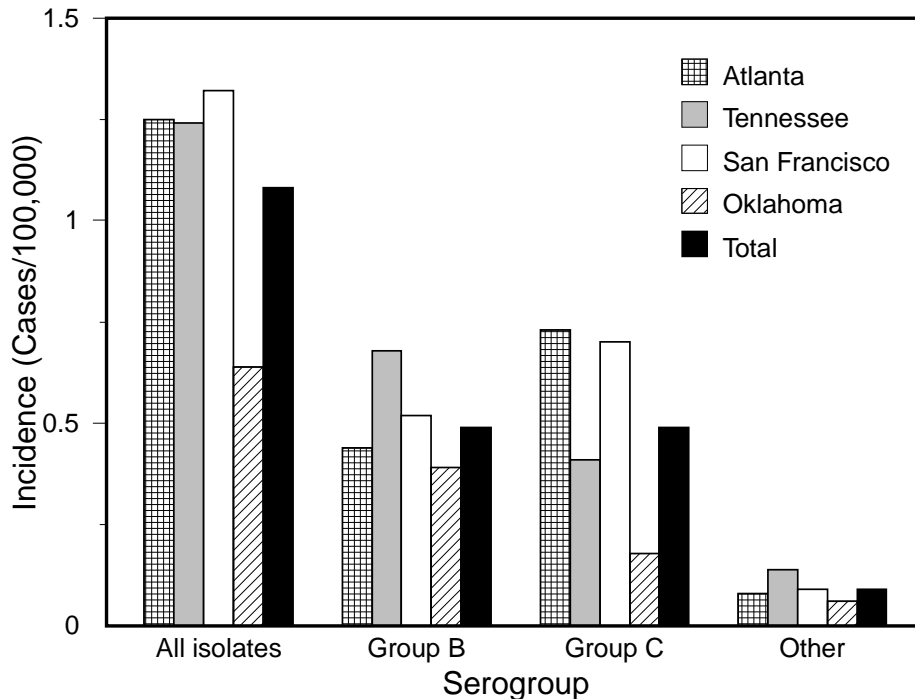
studies have reported a higher incidence of meningococcal disease among blacks than whites (2,5). Reasons for this difference are unclear; however, race is likely a risk marker rather than a risk factor for meningococcal disease. The race-specific variation in disease rates may reflect differences in factors such as household crowding, urban residence, or exposure to tobacco smoke. Risk markers may be useful for identifying groups to target with prevention efforts and may also suggest potential risk factors to investigate in future studies.

TABLE 1. Annual incidence and estimated number of cases of serogroups B and C meningococcal disease — United States, 1989–1991

Age group	Group B incidence*	Group C incidence*	Estimated no. of group B cases	Estimated no. of group C cases
≤2 years	7.5	3.1	822	382
3–5 years	0.58	1.9	62	203
6–17 years	0.20	0.50	83	208
18–29 years	0.12	0.25	58	117
≥30 years	0.21	0.19	288	268
All ages	0.51	0.46	1,313	1,178

*Incidence reported as cases/100,000 population per year.

FIGURE 5. Incidence of meningococcal disease, by serogroup, selected U.S. areas, 1989–1991



Serogroups B and C accounted for approximately equal proportions of disease; however, only 30% of cases in the ≤ 2 -year-old age group were due to serogroup C. Although serogroup C represented a higher proportion of total cases in older age groups, the incidence of disease in these age groups was lower; thus, the total estimated number of cases of group C disease in the > 2 -year-old age group is relatively small. Even if vaccination were 100% effective against group C disease and all persons > 2 years of age were routinely vaccinated with the polysaccharide vaccine, only 33% of meningococcal disease would be prevented. At current rates of disease, routine vaccination is unlikely to be a cost-effective preventive measure. However, vaccination of populations with clusters of disease (with high incident rates) is warranted, and further evaluation of the potential role of immunization for group C disease is under way. Development of vaccines that protect against serogroup B disease and are immunogenic in the age groups at highest risk of disease might improve the effectiveness of vaccination for meningococcal disease.

In 1986, a reported 61% of isolates were resistant to sulfa, with a significantly higher rate of resistance among group C compared with group B isolates (2). In contrast, in the present study the rate of resistant to sulfa was 37%; no difference was detected in resistance patterns by serogroup. Variation in reported sulfa resistance was also identified in data received through the CDC passive surveillance system from 1970 through 1980; the reported proportion of sulfa resistance varied from 12% to 67%, although it did not exceed 16% after 1974 and varied only from 11% to 16% from 1974 through 1980 (7). The prevalence of sulfa-resistant *N. meningitidis* is consistently high enough to warrant continuation of current recommendations against the use of sulfa drugs for treatment or prophylaxis of meningococcal disease unless the organism is known to be sensitive to sulfa. Since resistance to rifampin is rarely reported, it continues to be the drug of choice for prophylaxis.

The highest rates of both meningitic and nonmeningitic meningococcal disease occurred in infants < 1 year of age. A secondary peak was noted in nonmeningitic disease only after age 20, with an increase in rate that continued through persons ≥ 60 years of age (Figure 5). This secondary peak in incidence of nonmeningitic meningococcal disease was noted previously in a report of a laboratory-based surveillance system for invasive disease due to *N. meningitidis* (1). This report differed from the present study in noting a significantly higher case-fatality rate for nonmeningitic, compared with meningitic, meningococcal disease (17% vs. 12%).

Efforts to develop a serogroup B vaccine and protein conjugate C vaccines that would be immunogenic in infants are ongoing (8-10). Such vaccines, if incorporated into routine childhood vaccination schedules, would be expected to have a substantial impact on meningococcal disease, which remains an important cause of morbidity and mortality (an estimated 2,600 cases per year, 12% of which are fatal) in the United States.

References

1. Wenger JD, Hightower AW, Facklam RR, Gaventa S, Broome CV, the Bacterial Meningitis Study Group. Bacterial meningitis in the United States, 1986: report of a multistate surveillance study. *J Infect Dis* 1990;162:1316-23.
2. Pinner RW, Gellin BG, Bibb WF, et al. Meningococcal disease in the United States—1986. *J Infect Dis* 1991;164:368-74.
3. CDC. Meningococcal vaccines. *MMWR* 1985;34:255-9.

4. CDC. Summary of notifiable diseases, United States, 1991. *MMWR* 1992;40:58.
5. Schlech WF, Ward JI, Band JD, Hightower A, Fraser DW, Broome CV. Bacterial meningitis in the United States, 1978 through 1981. *JAMA* 1985;253:1749-54.
6. Fraser DW, Geil CC, Feldman RA. Bacterial meningitis in Bernalillo County, New Mexico: a comparison with three other American populations. *Am J Epidemiol* 1974;100:29-34.
7. Band JD, Chamberland ME, Platt T, Weaver RE, Thornsberry C, Fraser DW. Trends in meningococcal disease in the United States, 1975-1980. *J Infect Dis* 1983;148:754-8.
8. Frasch CE. Vaccines for prevention of meningococcal disease. *Clin Microbiol Rev* 1989;2(Suppl):S134-8.
9. Bjune F, Hoiby EA, Gronnesby JK, et al. Effect of outer membrane vesicle vaccine against group B meningococcal disease in Norway. *Lancet* 1991;338:1093-6.
10. de Moraes JC, Perkins BA, Camargo MC, et al. Protective efficacy of a serogroup B meningococcal vaccine in Sao Paulo, Brazil. *Lancet* 1992; 340:1074-8.

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State and Territorial Epidemiologists and Laboratory Directors are gratefully acknowledged for their contributions to this report. The epidemiologists listed below were in the positions shown as of May 5, 1993, and the laboratory directors listed below were in the positions shown as of April 1993.

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