



MWR

MORBIDITY AND MORTALITY WEEKLY REPORT

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Epidemiologic Notes and Reports

Unexplained CD4+ T-Lymphocyte Depletion in Persons Without Evident HIV Infection — United States

Since 1989, 21 persons with unexplained CD4+ T-lymphocyte depletion, but without evident human immunodeficiency virus (HIV) infection, have been described (1–12). These reports included persons who have resided in the United States and six other countries and who sought medical care for conditions often associated with immune deficiency. Some of these cases were also described at the VIII International Conference on AIDS/III STD World Congress in Amsterdam. In addition, CDC has received reports of five persons from three states who have had persistently low CD4+ T-cell levels but who have had no evidence of HIV infection or underlying disease processes or therapies known to be associated with T-cell depletion. In some of these five patients, opportunistic infections were diagnosed that frequently occur in persons with acquired immunodeficiency syndrome (AIDS). This report describes preliminary clinical and laboratory findings from an ongoing investigation by CDC of these five patients.*

Patient 1

In March 1991, a 70-year-old man developed *Pneumocystis carinii* pneumonia that was successfully treated with trimethoprim-sulfamethoxazole. Although serology for HIV antibody was negative, his CD4 count was 50 cells/µL. After this hospitalization, he developed a fungal infection of the groin that was treated with oral ketoconazole and topical antifungal medications. In April 1992, transitional cell carcinoma of the bladder, grade II, was diagnosed. As of July 1992, he was asymptomatic.

^{*}Single copies of this report will be available free until July 31, 1993, from the CDC National AIDS Clearinghouse, P.O. Box 6003, Rockville, MD 20849-6003.

CD4+ T-Lymphocyte Depletion - Continued

His family history and personal history were negative for immunodeficiency disease and for recurrent or unusual infections. His wife, who has remained healthy, is HIV seronegative and has a normal CD4 count. He did not report sexual contact with men or injecting-drug use. In 1987, he received 3 units of whole blood for a bleeding duodenal ulcer; follow-up investigation in 1992 indicated that all three blood donors were HIV type 1 (HIV-1) and HIV type 2 (HIV-2) seronegative, had normal CD4 counts, and were in good health.

Patient 2

In October 1984, a 38-year-old male health-care worker developed cryptococcal meningitis that was treated with a full course of amphotericin B and 5-fluorocytosine. In January 1985, he had an episode of localized herpes zoster. In July 1985, symptoms of meningitis recurred. A cryptococcoma was excised from his brain, and he was treated with another course of antifungal therapy. Since this hospitalization, he has been in generally good health except for a nonspecific skin rash that resolved, mild hypertension, and a grand mal seizure for which he takes phenytoin. In December 1987 and December 1988, his CD4 counts were 152 and 84 cells/µL, respectively. An enzyme immunoassay (EIA) test for HIV antibody was negative in April 1989. As of July 1992, he was asymptomatic.

There was no family history or personal history of immunodeficiency or unusual infections. He did not report sexual contact with men or injecting-drug use. He had not received blood transfusions, did not perform invasive procedures, and had no known parenteral or mucous membrane exposure to blood. He reported his spouse was in good health.

Patient 3

In October 1989, a 58-year-old woman developed acute cholecystitis and underwent cholecystectomy. She had a prolonged postoperative course complicated by nosocomial pneumonia of undetermined etiology and vaginal candidiasis and recovered with antibiotic therapy. An HIV EIA test was reported as positive during this hospitalization. In December 1989, although HIV serology by EIA was negative, a single p24 band on Western blot was observed; her CD4 count was 86 cells/µL. She remained asymptomatic; when reevaluated in January 1991, HIV serology was negative on two occasions, but her CD4 count remained low (103 cells/µL). As of July 1992, she was asymptomatic.

She had received multiple transfusions for hemorrhage during pregnancy during the 1950s and for menorrhagia in the late 1970s and early 1980s. There was no family history of immunodeficiency or unusual infections. She did not report injecting-drug use. She reported her spouse was in good health.

Patient 4

In November 1986, a 45-year-old man received treatment for disseminated molluscum contagiosum. In April 1989, a lung biopsy was performed for evaluation of a mass on chest radiograph. The lesion was consistent with a plaque secondary to asbestosis, although the man had no known history of asbestos exposure. In February and August 1989, HIV EIA serologies were negative; however, CD4 counts were 96 and 68 cells/µL, respectively. As of July 1992, he was asymptomatic.

His family and personal history were negative for immunodeficiency or for recurrent or unusual infections. He did not report sexual contact with men or injecting-drug use; he had not received blood transfusions. His spouse was in good health and had a normal CD4 count.

CD4 + T-Lymphocyte Depletion - Continued

Patient 5

In December 1983, a 70-year-old woman was hospitalized with disseminated cutaneous herpes zoster. In December 1988, she developed fever, cough, and pleurisy. On evaluation, mediastinal lymphadenopathy was found, and histoplasmosis was diagnosed by open lung biopsy. She was treated with amphotericin B and ketoconazole with resolution of both symptoms and lymphadenopathy. Although HIV serology was negative during this hospitalization, a CD4 count was 275 cells/ μ L. In February 1989, the count was 499 cells/ μ L.

In April 1991, she developed fever and cough, and a pulmonary infiltrate was indicated on chest radiograph; her CD4 count at that time was 199 cells/µL. Although her symptoms responded to ciprofloxacin, sputum cultures subsequently grew Mycobacterium avium-intracellulare (MAI). In September 1991, her pulmonary symptoms recurred, and a new pulmonary infiltrate was present on chest radiograph; MAI was again cultured from bronchial washings. Her symptoms resolved during treatment with ciprofloxacin, rifampin, and ethambutol. In January 1992, epigastric pain prompted a gastrointestinal evaluation and detection of an ulcerated mass lesion in the stomach. Histoplasmosis was found on biopsy, and Helicobacter pylori was cultured from the lesion. Fluconazole therapy was initiated with good response. As of July 1992, she was asymptomatic.

She had been in excellent health with no family history or personal history of immunodeficiency or unusual infections. She did not report injecting-drug use and had not received a transfusion. Her spouse died in 1984 with atherosclerotic cardiovascular disease and carcinoma of the kidney.

Laboratory Findings

Blood samples from each of the five patients have been tested at CDC, and low CD4 counts ($<300~cells/\mu L$) and negative HIV-1 and HIV-2 serologies (by EIA and Western blot) have been confirmed. Cocultures of peripheral blood mononuclear cells (PBMC) from patients 1–4 with normal PBMC and/or lymphoid cell lines were negative for cytopathicity, syncytia, and the generation of reverse transcriptase activity as measured by standard methods. Human T-cell lymphotropic virus (HTLV)-I and HTLV-II serologies were negative. Neither HIV-1– nor HIV-2–related DNA sequences were detected in blood of patients 1–4 by polymerase chain reaction (PCR). Results of studies of specimens from patient 5 are pending.

Reported by: Div of HIV/AIDS, National Center for Infectious Diseases, CDC.

Editorial Note: The clinical conditions of the patients described in this report vary considerably; however, these cases share three features: 1) persistently low CD4 + T-cell levels; 2) no evidence of HIV infection by serology, culture, or PCR analysis; and 3) infections that prompted physicians to consider HIV infection.

Review of available data on the 26 case-patients (including the five described in this report and 21 reported elsewhere) (1–12) does not indicate an epidemiologic linkage among the cases. Cases of unexplained CD4+ T-cell depletion have been reported from Australia (1), Denmark (2), England (3,4), France (5–7), Germany (8), Spain (9), and the United States (10–12). Of the 26 case-patients, five had received transfusions before onset of illness, five were homosexual men, and the remaining 16 had no known risk factors for HIV infection. In this report, follow-up investigation of the blood donors for patient 1 found that they were HIV-seronegative, immunologi-

CD4 + T-Lymphocyte Depletion - Continued

cally normal, and in good health. Two additional cases reported to CDC have been excluded because CD4+ T-cell depletion may have been related to chemotherapy.

Although infections with HIV-1 or HIV-2 have been associated with immunodeficiency of the type described in these patients, no evidence for infection with either virus has been documented. The cause of CD4+ T-lymphocyte depletion in the patients described in this report and in other reports is unknown; moreover, it is unknown whether these cases represent a single syndrome. However, there are at least two possible hypotheses to explain this abnormality. Persistent CD4+ T-cell depletion may occur in some patients as a response to certain infections or other exposures. Transient CD4+ T-cell depletion has been reported following some infections; whether this persists in some patients is unknown (13,14). Therefore, one possibility is that some or all of these case reports of unexplained CD4+ T-lymphocyte depletion are part of background occurrence that may only now be recognized because of the increased availability of T-cell phenotyping. A second possibility is that some of these cases may represent a different syndrome of immunodeficiency associated with CD4+ T-cell depletion.

Two of the recent preliminary reports of CD4 + T-cell depletion in patients who were HIV-1 and HIV-2 seronegative have suggested the presence of a retrovirus (11,12). The relation of these reports to the immunodeficiency detected in patients described in this and other reports is not known.

CDC and the National Institutes of Health (NIH) are collaborating with physicians, scientists, and public health officials to identify other cases and investigate this problem. NIH will assist in the characterization of the clinical, immunologic, and virologic features by collaborating with investigators and working through its network of grantees and contractors to collect, process, and distribute specimens and reference materials. CDC, in collaboration with NIH, will convene a meeting of investigators and public health officials in mid-August to discuss these cases and epidemiologic and laboratory investigations in progress.

Additional CDC epidemiologic and laboratory investigations regarding these cases are in progress. Health-care providers are requested to report to CDC through the AIDS surveillance section of their local or state health department patients who have 1) CD4+ T-lymphocyte depletion (absolute CD4+ T-cell level $<\!300$ cells/ μL or $<\!20\%$ on more than one determination), 2) no serologic evidence of HIV infection, and 3) no defined immunodeficiency or therapy associated with T-cell depletion. Although no cases have been reported in children, HIV-negative pediatric cases with unexplained depletion of CD4 cells (as defined by age-adjusted normal CD4 counts) should also be reported.

Additional information on case reporting is available from the Surveillance Branch, Division of HIV/AIDS, National Center for Infectious Diseases, CDC (telephone [404] 639-2981). Reports of these cases will assist CDC and other public health agencies in developing a more precise case definition to examine this problem and providing a resource for investigators who are conducting etiologic studies. General inquiries about the cases described here as well as on HIV and AIDS should be directed to the CDC National AIDS Hotline, (800) 342-2437.

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 Gatenby PA. Reduced CD4+ T-cells and candidiasis in absence of HIV infection. Lancet 1989:1:1027–8.

CD4 + T-Lymphocyte Depletion - Continued

- 2. Hansen ER, Lisby S, Baadsgaard O, Ho VC, de Villiers EM, Vejlsgaard GL. Abnormal function of CD4 + helper/inducer T lymphocytes in a patient with widespread human papillomavirus type 3-related infection. Arch Dermatol 1990;126:1604–8.
- 3. Pankhurst C, Peakman M. Reduced CD4+ T-cells and severe oral candidiasis in absence of HIV infection. Lancet 1989;1:672.
- 4. Jowitt SN, Love EM, Liu Yin JA, Pumphrey RSH. CD4 lymphocytopenia without HIV infection in patient with cryptococcal infection. Lancet 1991;337:500–1.
- Cozon G, Greenland T, Revillard JP. Profound CD4+ lymphocytopenia in the absence of HIV infection in a patient with visceral leishmaniasis. N Engl J Med 1990;322:132.
- Seligmann M, Aractingi S, Oksenhendler E, Rabian C, Ferchal F, Gonnot G. CD4+ lymphocytopenia without HIV in patient with cryptococcal disease. Lancet 1991;337:57–8.
- Gautier V, Chanez P, Vendrell JP, et al. Unexplained CD4-positive T-cell deficiency in non-HIV
 patients presenting as a *Pneumocystis carinii* pneumonia. Clin Exp Allergy 1991:21:63–6.
- 8. Daus H, Schwarze G, Radtke H. Reduced CD4 + count, infections, and immune thrombocytopenia without HIV infection. Lancet 1989;2:559–60.
- 9. Castro A, Pedreira J, Soriano V, et al. Kaposi's sarcoma and disseminated tuberculosis in HIV-negative individual. Lancet 1992;339:868.
- Daar ES, Moudgil T, Ho DD. Persistently low T-helper (CD4+) lymphocyte counts in HIV-negative asymptomatic men. Western Society of Clinical Investigation meeting, February 1990.
- 11. Gupta S, Ribak CE, Gollapudi S, Kim CH, Salahuddin SZ. Detection of a human intracisternal retroviral particle associated with CD4+ T-cell deficiency. Proc Natl Acad Sci USA (in press).
- 12. Laurence J, Siegal FP, Schattner E, Gelman IH, Morse S. Acquired immune deficiency without evidence of infection with human immunodeficiency virus types 1 and 2. Lancet 1992 (in press).
- Scalzini A, Castelnuovo F, Puoti M, Cristini G. A case of cryptococcal meningoencephalitis and focal cerebral vasculitis with transient immunodeficiency. Acta Neurol (Napoli) 1990; 12:301–4.
- 14. Lehmann PF, Gibbons J, Senitzer D, Ribner BS, Freimer EH. T lymphocyte abnormalities in disseminated histoplasmosis. Am J Med 1983;75:790–4.

Current Trends

Arboviral Disease - United States, 1991

During 1991, state and local health departments reported 122 cases of human arboviral encephalitis to CDC. More than half (69) of the cases resulted from outbreaks of St. Louis encephalitis (SLE) in Arkansas and Texas. In addition, an epizootic of eastern equine encephalitis (EEE) extending from the Atlantic and Gulf coasts into the upper midwest caused sporadic human cases and a substantial loss of livestock. This report summarizes the reported cases of arboviral encephalitis in the United States during 1991 and underscores the continuing need for arbovirus surveillance and control.

St. Louis encephalitis. SLE activity in the United States during 1990—1991 was at the highest level since 1976 (Figure 1). From July through September 1991, 25 laboratory-confirmed SLE cases occurred in Pine Bluff, Arkansas, and 41 SLE cases occurred in Harris County (Houston), Texas, resulting in annual incidence rates of 44 and 1.5 cases per 100,000 population, respectively. Additional sporadic SLE cases were confirmed from Arkansas (three), California (three), Florida (one), Louisiana (one), North Carolina (one), Texas (two) and Washington (one). Travel histories suggest that two of the three California patients and the Washington patient contracted SLE during visits to Arizona and New Mexico.

Arboviral Disease - Continued

LaCrosse encephalitis. In 1991, 38 laboratory-confirmed cases of LaCrosse encephalitis (LAC) were reported from Illinois (13), Minnesota (four), North Carolina (one), Pennsylvania (one), and Wisconsin (19). Although LAC is generally the predominant cause of arboviral encephalitis in the United States, it is often undiagnosed and underreported.

Eastern equine encephalitis. In 1991, an EEE epizootic occurred among horses in states along the southeastern seaboard, with intense transmission occurring in Florida, Georgia, and South Carolina. The epizootic also extended into the midwestern states, causing deaths among horses in Ohio and Michigan. A cluster of five laboratory-confirmed human EEE cases occurred in northeastern Florida in June and July 1991, resulting in two deaths (1). Six additional human cases occurred in Georgia (two), Michigan (two), Louisiana (one), and South Carolina (one). For the first time, EEE virus was isolated from Aedes albopictus mosquitoes collected at a tire depot in central Florida (2,3).

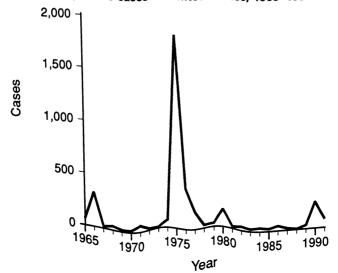
Western equine encephalitis. In 1991, one laboratory-confirmed human case of western equine encephalitis (WEE) occurred in Colorado. Since 1980, zero to 40 human cases of WEE have been reported annually, primarily from the western United States.

Reported by: Participating state epidemiologists, veterinarians, and vector-control directors. Div of Vector-Borne Infectious Diseases, National Center for Infectious Diseases, CDC.

Editorial Note: The isolation of EEE from a potential new mosquito vector (*Ae. albopictus*) and the increasing trend in human SLE cases that began in 1990 indicate the continuing need for arboviral surveillance and control in 1992 (2–4).

The last nationwide SLE epidemic in 1975 resulted in 1815 human cases in 31 states. This epidemic was preceded by an increase in cases in 1974, particularly in the southeastern United States. SLE virus is transmitted by the mosquito *Culex quinque-fasciatus* in the south-central and southeastern United States, except in Florida, where it is transmitted by *Cx. nigripalpus*. Virus activity generally peaks in August and

FIGURE 1. St. Louis encephalitis cases - United States, 1965-1991



Arboviral Disease - Continued

September. The 1991 SLE outbreak in Pine Bluff was the first reported from that community but was similar to outbreaks occurring in the Midwest in 1975 (5). During 1991, seroepidemiologic studies indicated that 10% of residents in Pine Bluff had antiflaviviral IgG (CDC, unpublished data, 1991), indicating unrecognized SLE transmission in this area.

Control measures that may reduce the risk for SLE infection include targeting vector-control efforts at open storm drains near housing developments, eliminating water-holding containers on premises, and reducing exposure to the vector mosquito at dusk and during the evening hours by staying indoors and using insect repellents. For those who must be outdoors in the evening, long-sleeved shirts and long pants reduce exposure to mosquito bites. Light-colored clothing is less attractive than dark clothes to most mosquitoes. Residents of areas where SLE is epizootic should repair window and door screens and avoid sitting on unscreened porches at dusk. In particular, prevention efforts should be aimed at elderly persons (>60 years old), who have the highest age-specific SLE attack rates during outbreaks (6). Although precise prediction of SLE epidemics is not possible, the increased level of SLE activity in the southern and southeastern United States during 1990–1991 suggests that continued SLE activity is likely in 1992.

LAC encephalitis, caused by an arbovirus of the California serogroup transmitted by *Ae. triseriatus*, is most common among young children. LAC virus is prevalent primarily in midwestern states; however, serosurveys and active surveillance programs have demonstrated this virus has a wide geographic distribution in the United States. LAC may be underreported in many areas and should be considered in the differential diagnosis of pediatric viral encephalitis in states where hardwood forests and woodlots are common.

Although fewer than 10 human cases of EEE are reported annually in the United States, this virus is associated with a high case-fatality rate. An effective equine vaccine is licensed in the United States and is recommended for livestock in areas where EEE transmission is known to occur. However, revaccination during a single transmission season may be necessary. Specific control measures to prevent human EEE cases are difficult to implement because the disease is rare, even during a major equine epizootic.

The recent detection of *Ae. albopictus* in the United States has prompted concern because of its potential for transmission of EEE virus. This mosquito was imported from Asia to Texas, where it was discovered in 1985, and has since become widespread in the central and southern United States (7). *Ae. albopictus* is an aggressive biter that thrives in both forest and suburban habitats. This species, therefore, potentially could serve as an important bridging vector for EEE virus from swamp habitats into populated areas, although there is no evidence to indicate this has occurred.

Clinicians should be encouraged to obtain acute and convalescent arboviral antibody titers on all suspected cases of arboviral encephalitis. Patients with arboviral encephalitis should be reported promptly to state and local public health authorities. References

- 1. CDC. St. Louis encephalitis outbreak Arkansas, 1991. MMWR 1991;40:605-7.
- CDC. Eastern equine encephalitis virus associated with Aedes albopictus Florida, 1991. MMWR 1992;41:115,121.
- Mitchell CJ, Niebylski ML, Smith GC, et al. Isolation of eastern equine encephalitis virus from Aedes albopictus in Florida. Science 1992;257:526–7.

Arboviral Disease - Continued

- 4. CDC. Update: St. Louis encephalitis Florida and Texas, 1990. MMWR 1990;39:756-9.
- CDC. Eastern equine encephalitis Florida, eastern United States, 1991. MMWR 1991;40: 533–5.
- Luby JP, Miller G, Gardner P, Pigford CA, Henderson BE, Eddins D. The epidemiology of St. Louis encephalitis in Houston, Texas, 1964. Am J Epi 1967;83:584–97.
- 7. Moore CG, Francy DB, Eliason DA, Monath TP. Aedes albopictus in the United States: rapid spread of a potential disease vector. J Am Mosq Control Assoc 1988;4:356–61.

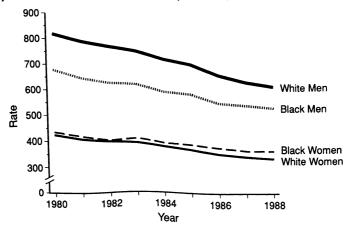
Trends in Ischemic Heart Disease Mortality — United States, 1980–1988

In 1989, approximately 500,000 persons died from ischemic heart disease (IHD), the leading cause of death in the United States (1). This report summarizes an analysis by CDC to characterize trends in IHD mortality in the United States from 1980 through 1988 (the latest year for which data are available), and emphasizes comparisons by race and sex, region, and state.

Public-use mortality data tapes compiled by CDC's National Center for Health Statistics and population estimates from the Bureau of the Census were used to calculate age-adjusted IHD death rates for persons aged ≥35 years, standardized to the 1980 U.S. population. Race-specific denominator data were available only for blacks and whites. IHD deaths were defined as those with the underlying cause of death listed on the death certificate as *International Classification of Diseases*, *Ninth Revision*, codes 410–414.

Age-adjusted IHD death rates for the U.S. population aged ≥35 years declined 24% – from 588.3 per 100,000 in 1980 to 448.8 per 100,000 in 1988. Although IHD death rates declined more rapidly for men than for women and for whites than for blacks, they declined for each of the four race-sex groups (Figure 1). The average annual decline (as estimated with a log linear model [2]) was 3.7% for white men, 3.1% for black men, 2.9% for white women, and 2.2% for black women.

FIGURE 1. Age-adjusted death rates* for ischemic heart disease among adults aged ≥35 years, by race and sex — United States, 1980–1988



^{*}Per 100,000 population, adjusted to the 1980 U.S. standard population.

Ischemic Heart Disease - Continued

During the 9-year period, overall rates of IHD mortality were highest in the Northeast, followed by the Midwest, the South, and the West. IHD death rates declined steadily in each of the four regions of the United States; the average annual decline was 3.9% in the Northeast, 3.2% in the Midwest, 3.1% in the West, and 2.9% in the South. Regional patterns in IHD mortality were similar by race and sex.

IHD death rates for the states varied substantially (Table 1, page 555). For both women and men, there was a more than twofold difference in the 1988 IHD death rate in the state with the highest rate compared with the state with the lowest (women: Hawaii, 184.0 per 100,000 compared with New York, 462.6; men: Hawaii, 316.4 per 100,000 compared with New York, 755.1). To determine whether state-to-state variation in IHD mortality was similar for men and women, the Spearman correlation coefficient was calculated between state-specific rates for women and men. The correlation coefficient was 0.93, indicating that the rank order of states by IHD mortality was similar for women and men.

Throughout the 9-year period, IHD death rates declined in each of the 50 states and the District of Columbia for both women and men (Table 1, page 555). The annual median rate of decline among the states was 3.0% for women and 3.8% for men. However, the percent change in IHD death rates varied widely from state to state for women (range: -7.5% to -1.3%) and men (range: -10.0% to -1.9%).

Reported by: Cardiovascular Health Br, Div of Chronic Disease Control and Community Intervention, National Center for Chronic Disease Prevention and Health Promotion, CDC.

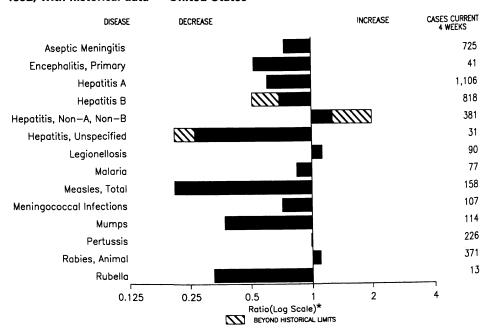
Editorial Note: The findings in this report indicate that, for persons aged ≥35 years, age-adjusted IHD death rates declined from 1980 through 1988; however, since 1976, the IHD death rates for white men have declined more rapidly (3). The factors contributing to the differential rates of decline are unclear but could include differences in 1) the incidence of coronary heart disease, 2) trends in risk factors for IHD, and 3) access to quality health care.

Although age-adjusted IHD death rates declined faster for men than for women, rates were consistently higher for men. In 1988, the IHD death rate for black men was 1.5 times greater than for black women, and the rate for white men was 1.8 times greater than for white women. These findings are consistent with previous reports noting excess IHD mortality among men and variations in the male-to-female ratio of IHD mortality by race, time period (4), country (5), and age group (6).

The declines in IHD mortality that occurred in all regions and states during 1980–1988 contrast dramatically with increases that occurred from the 1920s through the 1950s (7). The earliest declines in IHD mortality were observed in metropolitan areas, especially in the Northeast and Pacific West, and among communities with the most social and economic resources (8,9). Subsequently, the declines in IHD mortality occurred in nonmetropolitan areas, communities with fewer economic resources, and the southern region. Despite the declines in IHD mortality, death rates still varied substantially by region and state. The strong correlation of state-specific IHD death rates between women and men suggests that there is geographic variation in socioenvironmental conditions that influence the risk for IHD mortality similarly for women and men.

To improve strategies for reducing the burden of IHD mortality among all subgroups of the U.S. population, state-level factors that contribute to the geographic variations in IHD mortality should be identified. For example, the variations may

FIGURE I. Notifiable disease reports, comparison of 4-week totals ending July 25, 1992, with historical data — United States



^{*}Ratio of current 4-week total to the mean of 15 4-week totals (from previous, comparable, and subsequent 4-week periods for the past 5 years). The point where the hatched area begins is based on the mean and two standard deviations of these 4-week totals.

TABLE I. Summary — cases of specified notifiable diseases, United States, cumulative, week ending July 25, 1992 (30th Week)

	Cum. 1992		Cum. 1992
AIDS*	23,872	Measles: imported	96
Anthrax	20,072	indigenous	1,264
Botulism: Foodborne	9	Plaque	3
Infant	32	Poliomyelitis, Paralytic [†]	
Other	1	Psittacosis	49
Brucellosis	42	Rabies, human	
Cholera	42	Syphilis, primary & secondary	19,349
Congenital rubella syndrome	1 7	Syphilis, congenital, age < 1 year§	697
Diphtheria	l i	Tetanus	9
Encephalitis, post-infectious	86	Toxic shock syndrome	143
Gonorrhea	272,618	Trichinosis	17
Haemophilus influenzae (invasive disease)	861	Tuberculosis	12,142
Hansen Disease	97	Tularemia	79
Leptospirosis	1 18	Typhoid fever	189
Lyme Disease	2,895	Typhus fever, tickborne (RMSF)	185

^{*}Updated monthly; last update July 4, 1992.

'Two cases of suspected poliomyelitis have been reported in 1992; six of the nine suspected cases with onset in 1991 were confirmed and 5 of the 8 suspected cases with onset in 1990 were confirmed, and all were vaccine associated.

JJJJJJJJJJJJJDdates for first quarter 1992.

TABLE II. Cases of selected notifiable diseases, United States, weeks ending July 25, 1992, and July 27, 1991 (30th Week)

		Aseptic	Encen	halitis	T		He	epatitis ((Viral), by	type		1,
Reporting Area	AIDS*	Menin- gitis	Primary	Post-in- fectious	Gono	rrhea	Α	В	NA,NB	Unspeci- fied	Legionel- losis	Lyme Disease
	Cum. 1992	Cum. 1992	Cum. 1992	Cum. 1992	Cum. 1992	Cum. 1991	Cum. 1992	Cum. 1992	Cum. 1992	Cum. 1992	Cum. 1992	Cum. 1992
UNITED STATES	23,872	3,517	300	86	272,618	333,432	11,134	8,854	4,203	377	721	2,895
NEW ENGLAND	755	155	17	-	5,812	8,172	332	335	41	15	35	424 4
Maine N.H.	27 25	14 7	1 2		48 73	95 154	23 25	17 24	5 12	1	1 3	17
Vt.	11	8	2		15	30	5	8	6	-	2	3
Mass.	431	64	9	-	2,130 422	3,534 670	167 76	256 17	15 3	14	19 10	63 96
R.I. Conn.	61 200	62	3		3,124	3,689	36	13	-	-	-	241
MID. ATLANTIC	6,001	356	16	7	28,437	40,066	843	1,152	204	13	212	1,829
Upstate N.Y.	736	162	-	:	5,654	6,958	203 328	277 202	129 3	6	85 3	1,228 5
N.Y. City N.J.	3,309 1,214	70	4	1 -	9,320 4.013	15,441 6,612	122	288	52		24	193
Pa.	742	124	12	6	9,450	11,055	190	385	20	7	100	403
E.N. CENTRAL	2,192	492	79	26	50,515	61,202	1,609	1,334	735	24	160	65
Ohio	414	135 77	24 8	2 11	15,632 4,747	18,730 5,997	255 479	134 461	59 347	4 8	74 17	30 21
Ind. III.	222 979	104	26	6	16,323	18,463	285	134	39	4	11	6
Mich.	457	168	19	7	11,748	13,653	83 507	357 248	247 43	8	38 20	8
Wis.	120	8	2		2,065	4,359						405
W.N. CENTRAL	675	185 15	18 2	6	12,023 1,652	16,097 1,618	1,297 391	359 44	147 12	18 2	46 2	125 49
Minn. Iowa	120 50	27	-	3	892	1,147	22	22	4	2	14	10
Mo.	347	78	8	•	6,377 39	9,958 34	441 69	235 1	113 3	13 1	15 1	44 1
N. Dak. S. Dak.	1 6	1 6	1	1	99	197	179	3	-	-		
Nebr.	29	10	2	2	8	1,104	101	13	5	-	12	10
Kans.	122	48	5	-	2,956	2,039	94	41	10	-	2	11
S. ATLANTIC	5,678	678 26	61 6	35	87,265 989	101,716 1,449	695 24	1,482 141	585 119	56 1	106 16	220 95
Del. Md.	64 669	85	10		8,539	10,538	132	225	21	5	20	29
D.C.	417	14	1		3,829	5,623 9,915	12 61	47 105	233 23	20	7 10	1 52
Va. W. Va.	322 29	105 7	19 4	9	10,073 514	681	4	33	1	12	-	3
N.C.	370	79	17	-	14,463	20,259	58	247	58	-	18	22
S.C. Ga.	166 759	7 87	2		6,331 26,750	7,890 24,643	15 94	31 173	57	-	16 5	2
Fla.	2,882	268	2	26	15,777	20,718	295	480	73	18	14	16
E.S. CENTRAL	739	225	10		26,291	31,660	175	762	1,318	2	38	42
Ky.	105 227	73 49	7 1	-	2,732 8,218	3,347 11,338	47 80	46 638	3 1,303	-	16 16	14 23
Tenn. Ala.	272	62	i		8,805	8,908	27	75	11	1	6	5
Miss.	135	41	1	-	6,536	8,067	21	3	1	1	-	-
W.S. CENTRAL	2,174	447	30	4	30,921	38,183	1,071	1,146	75	93	11	68
Ark. La.	112 390	5 35	7 3	1	4,453 8,586	4,420 8,857	52 95	47 90	6 29	4 2	1	10 3
Okla.	147	-	2	2	3,127	3,869	121	114	24	3	5	16
Tex.	1,525	407	18	1	14,755	21,037	803	895	16	84	5	39
MOUNTAIN	686 12	125 2	13 1	4 1	6,859 60	7,101 64	1,612 48	401 23	156 25	33	57 9	4
Mont. Idaho	15	19			64	84	35	54	25	-	4	2
Wyo.	2	-	1	:	31	55	3	2	10		.1	1
Colo. N. Mex.	236 58	39 8	6 3	1	2,482 522	2,062 656	463 162	64 107	55 15	17 7	10 2	-
Ariz.	203	37	1	-	2,400	2,644	660	80	18	4	17	-
Utah Nev.	54 106	1 19	1	1	158 1,142	184 1,352	190 51	10 61	19 12	5	2 12	1
			56	4	24.495	29,235	3,500	1,883	942	123	56	110
PACIFIC Wash.	4,972 255	854	56 1	-	24,495	29,235	388	1,883	942 76	7	8	118 3
Oreg.	146		-	-	920	1,193	200	167	44	7	-	
Calif. Alaska	4,484 8	795 8	52 3	3	20,761 410	24,561 437	2,742 32	1,510 8	663 2	101 1	47	115
Hawaii	79	51	-	1	276	444	138	12	157	ż	1	-
Guam	-	2	-	-	45	5	5	1		6	-	1
P.R.	876	103	1	•	109	366	21	252	78	16	1	-
V.I. Amer. Samoa	2	-	-		61 24	259 28	2 1	5 1	:	-	-	
C.N.M.I,					45	37	i	•		-		

N: Not notifiable U: Unavailable *Updated monthly; last update July 4, 1992.

TABLE II. (Cont'd.) Cases of selected notifiable diseases, United States, weeks ending July 25, 1992, and July 27, 1991 (30th Week)

	Malaria			les (Rubeola)			Menin-	١		Pertussis			5.1.1		
Reporting Area	Cum.	Indig	enous	Impo	orted*	Total	gococcal Infections	Mu	mps		Pertussi	S		Rubella	•
· · · · · · · · · · · · · · · · · · ·	1992	1992	Cum. 1992	1992	Cum. 1992	Cum. 1991	Cum. 1992	1992	Cum. 1992	1992	Cum. 1992	Cum. 1991	1992	Cum. 1992	Cum 1991
JNITED STATES	478	7	1,264	5	96	7,950	1,378	25	1,654	53	969		2	-	1,050
NEW ENGLAND	28	3	48		7	59	86	23	1,034	5		1,314	2	119	1,050
∕laine I.H.	3	2	2	-		2	7	·	-	1	86 4	200 45		6 1	•
łt.		-	15	-	•	5	5	-	2	2	26	17	-	-	
Mass. I.I.	14	-	11	-	3	27	3 35	:	2	-	1 36	3 116	-		
onn.	4 7	1	20	•	4	2	1	-	-	-	-		-	4	
AID. ATLANTIC	130		170	-		23	35	-	6	2	19	19	-	1	
Jpstate N.Y.	21		79	-	12 3	4,416 380	154 75	4 2	115 48	1	85 25	131	-	15 11	56 53
N.Y. City N.J.	69	•	42	-	8	1,575	14	-	18	-	25 11	74 17		- 11	
'a.	20 20	:	44 5	:	1	1,017	18	-	11	-	14	10	-	3	2
.N. CENTRAL	31		_			1,444	47	2	38	1	35	30	-	1	
Ohio	4		23	1 1†	13 6	77 3	213	-	214	2	71	254	-	7	17 14
nd. I.	9	•	20		•	1	55 32	-	82 7	1	32 14	68 47		:	
ı. ∕lich.	8 8	•	1	-	4	25	57	-	61	1	9	51	-	7	2
Vis.	2		2	-	2 1	39 9	53 16	•	56	-	5	23	-	•	- 4
V.N. CENTRAL	27	_	6	3	8			-	8	-	11	65	•	4	1
∕linn.	13	-	5	15	5	40 10	65 9	1	58 19	10 2	87 29	91 35	-	4	
owa //o.	2 8	-	•	2†	3	15	7	1	10	-	3	9	-	-	
l. Dak.			:	-	-	1	20	-	21	6	32	31	-	-	
. Dak.	1	-	-	-	-	-	1	-	2	-	8 5	2 3	-	:	
lebr. lans.	3	-	-	-	-	1	13	-	4	2	6	5		-	
. ATLANTIC		-	1	•	-	13	14	-	2	-	4	6	-	4	
el.	91 4	-	114 3	-	11	431	252	2	622	-	74	129	1	14	
∕ld.	27		9	-	7	21 165	2 26	:	4	-	1	-	-	5	
).C. /a.	7	-	-	-	-	- 103	20	1	60 5	-	16	30	-	1	
V. Va.	20	-	10	-	4	28	38	-	38	-	6	16	-	1	
v.c.	7	-	25	-	-	38	14 53	•	22 126	•	2 13	7 19	1		
S.C. Sa.	-	-	29	-	-	12	18	1	47		9	9		2	
la.	3 23	-	38	-	-	14	37	-	56	-	8	22	-	5	
S. CENTRAL	12	3		-		153	62	-	264	-	19	26	-	1	10
(y.	1	3	444 442		18 1	2	91	-	40	1	18	40	•	'.	
enn.	7	-	-	-	:	i	28 27	-	13	-	5	16	-	1	10
∖la. ∕liss.	4	-	2	-	-	-	27	-	7	1	12	23	-	:	
V.S. CENTRAL	47	-		-	17	•	9	-	20	-	1	1	•		
Ark.	17	-	366	-	-	127	102	3	288	1	35	33	•		
. 8.	1	-	-	-	-	5	9 24	-	6 15	1	9 2	3 9	-		
Okla. Tex.	4 12	•	11	•	-		13	-	15	·	24	15	•		
MOUNTAIN		•	355	-	•	122	56	3	252	-	-	6	-	5	
Mont.	11	-	4	-	7	949	69	7	101	11	193	139	-	-	
daho	-	-	:	-		385	12 8	-	2 3	2	1 23	2 20		1	
Nyo. Colo.	-	-	1	•	-	3	2	-	-	-	-	3	•	.:	
2010. N. Mex.	5 1		3	:	7	5 98	12 7	1	14	-	24 41	70 16	-	`.	
Ariz.	4	-	-	-	-	312	15	N	N 52	5 4	41 79	8	-	2	
Jtah Nev.	1	-	-	•	-	129	4	6	23	-	24	18	-	1	
PACIFIC		-	-	-	-	17	9	-	7	-	1	2		67	1
Wash.	131 7	1	89	1	20 10	1,849	346	8	206	22	320	297	1	6	
Oreg.	10	-	4	-	10	4 62	46 46	1 N	9 N	9 2	92 16	71 38	-	2	1
Calif. Alaska	106	-	46	1†	2	1,765	243	5	183	11	195	139	1	39	
Alaska Hawaii	1 7	1	8 31	:	1 6	1 17	6	-	1	-	3 14	12 37	-	20	
Guam	1	Ü	10		U	17	5	2	13		14	31	U	1	
P.R.	- '-	40	293	U	:	88	3	U	7 1	U	8	27	-	-	
V.I. Amer Sames	-	-			-	2	-	÷	17	-	-		-	-	
Amer. Samoa C.N.M.I.	•	Ū	-	Ū	-	24	-	Ū	-	ů	6 1	-	Ū		

^{*}For measles only, imported cases includes both out-of-state and international importations.

N: Not notifiable U: Unavailable †International fout-of-state

TABLE II. (Cont'd.) Cases of selected notifiable diseases, United States, weeks ending July 25, 1992, and July 27, 1991 (30th Week)

Reporting Area		ohilis Secondary)	Toxic- shock Syndrome	Tuber	culosis	Tula- remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies Anima
noporting Area	Cum. 1992	Cum. 1991	Cum. 1992	Cum. 1992	Cum. 1991	Cum. 1992	Cum. 1992	Cum. 1992	Cum. 1992
UNITED STATES	19,349	24,337	143	12,142	12,671	79	189	185	4,488
NEW ENGLAND	382	641	10	194	342	-	20	7	434
Maine	2	-	6	14 3	27 5	•	ī	-	1
N.H. Vt.	33 1	12 1	-	3	4	-		-	17
Mass.	182	300	3	74	167	-	12	4	4
R.I. Conn.	20 144	35 293	1	24 76	33 106	-	7	2 1	412
			19	2,851	2,989		, 51	13	1,272
MID. ATLANTIC Upstate N.Y.	2,899 191	4,370 395	8	200	2,303		7	3	673
N.Y. City	1,570	2,147	-	1,765	1,811	-	22	3	
N.J. Pa.	376 762	750 1,078	11	524 362	493 398	-	15 7	4 3	418 181
			40	1,231	1,268	1	21	18	79
E.N. CENTRAL Ohio	2,745 461	2,774 363	12	190	189		3	11	73
Ind.	159	84	9	99	106	-	1	3	9
III.	1,232	1,341	5	624	662	1	15 1	1	12 8
Mich. Wis.	557 336	668 318	14	271 47	255 56		i	3	43
W.N. CENTRAL	664	418	23	278	304	35	2	15	751
Minn.	47	45	5	71	59	•	ī		108
lowa	28	37	5	22	44	-	:		132
Mo. N. Dak.	506 1	290 1	3 1	126 2	127 6	27	1	13	7 102
S. Dak.	•	i		15	24	6		1	75
Nebr.	.1	9 35	3	13 29	11 33	1 1		1	8 319
Kans.	81		6						
S. ATLANTIC Del.	5,414 130	7,178 91	14 3	2,246 25	2,384 16	3	14	39 3	1,026 130
Md.	400	597	2	152	216	1	3	4	304
D.C.	249	448	:	77	117	-	1	1	11
Va. W. Va.	410 9	536 19	1	154 48	209 41	2	1	2 3	175 23
N.C.	1,383	1,094	ġ	283	325	-	:	19	13
S.C.	723	890	1	234	234 482	•	1	2	89
Ga. Fla.	1,090 1,020	1,761 1,742	1 2	498 775	482 744	-	8	3 2	211 70
E.S. CENTRAL	2,517	2,640	1	853	840	5	3	33	86
Ky.	83	48	-	222	193	ĭ		4	48
Tenn.	678	900	1	236	230	4	•	26	
Ala. Miss.	966 790	981 711	-	231 164	236 181	-	3	3	38
		4,385	1	1,231	1,446	17	6	51	465
W.S. CENTRAL Ark.	3,550 488	386		106	121	9		š.	19
La.	1,482	1,442	•	108	119	:	•	.:	
Okla.	166 1,414	110 2,447	1	87 930	104 1,102	8	6	43	227 219
Tex.		•		319	345	17	2	5	94
MOUNTAIN Mont.	227 7	339 5	11	319	345	8	-	2	12
Idaho	í	3	1	12	4	-	1	1	
Wyo.	_1	4		29	3 35	2 3	1	:	23 7
Colo. N. Mex.	32 24	54 21	4 2	29 47	45	4	:	i	5
Ariz.	115	216	2	144	185	-	•	:	44
Utah	6	5 31	2	46 41	30 40	:		1	1 2
Nev.	- 41						70	4	281
PACIFIC Wash.	951 49	1,592 108	24	2,939 175	2,753 173	1	4	•	281
Oreg.	49 26	45	1	77	62		-	1	
Calif.	867	1,431	23	2,520	2,357	1	63	3	269
Alaska Hawaii	4 5	4 4	-	31 136	43 118		3	-	12
		•	_	34	6	-	3	-	
Guam P.R.	2 181	275	-	135	109		i	-	31
V.I.	37	72	-	3	2	•	:	-	-
Amer. Samoa					2	-	1	•	-

U: Unavailable

TABLE III. Deaths in 121 U.S. cities,* week ending July 25, 1992 (30th Week)

								(30th Week)							
Reporting Area		All Cau	ıses, B	y Age	Years)		P&I [†]			All Cau	ises, B	y Age	Years)		P8
moporting Area	All Ages	≥65	45-64	25-44	1-24	<1	Total	Reporting Area	All Ages	≥65	45-64	25-44	1-24	<1	To
NEW ENGLAND Boston, Mass.	532	375	91	49	10	7	31	S. ATLANTIC	1,288	790	246	149	44	56	_
Bridgeport, Conn.	172 36	113 24		24	2	3	14	Atlanta, Ga.	182	97			7	14	
Cambridge, Mass.	20	17	1	3	-	1	2	Baltimore, Md.	218	130			9	11	
all River, Mass.	24	19	3	2	-	-	1	Charlotte, N.C.	99	63			5	2	
lartford, Conn.	35	26		i	1	-	1	Jacksonville, Fla.	111	72		9	1	2	
owell, Mass.	19	16		i		-	2	Miami, Fla.	99	67			6	-	
ynn, Mass.	9	6		ż	-	-	1	Norfolk, Va.	68	33			3	6	
New Bedford, Mass.	19	17		2		-		Richmond, Va.	92	66			2	3	
New Haven, Conn.	30	16	11		2	1	3	Savannah, Ga. St. Petersburg, Fla.	46	33			-	-	
rovidence, R.I.	33	24	4	4	ī	-		Tampa, Fla.	67	55			1	2	
omerville, Mass.	6	2	3	_	1		_	Washington, D.C.	152	96			2	9 7	
Springfield, Mass.	36	24		1		2	3	Wilmington, Del.	138	67			8	,	
Vaterbury, Conn.	36	28		3	2		ĭ	-	16	11		1	-	•	
Vorcester, Mass.	57	43	9	5	-	-	ġ	E.S. CENTRAL	757	500	161	54	28	14	
MID. ATLANTIC	2,482	1,530	499	320	71	60		Birmingham, Ala.	96	67	16		3	2	
libany, N.Y.	53	40	4	7	/1	62	113	Chattanooga, Tenn.		59			2	-	
Illentown, Pa.	20	14	5	í	-	2	2	Knoxville, Tenn.	87	62			3	1	
Buffalo, N.Y.	101	69	25	2	2	3	4	Lexington, Ky.	48	32		2	2	1	
amden, N.J.	33	17	7	6	3		2	Memphis, Tenn.	177	114			10	2	
lizabeth, N.J.	16	9	2	4		1	-	Mobile, Ala.	66	49			1		
rie, Pa.§	41	32	6	i	1	i	2	Montgomery, Ala.	52	28		. 5	1	1 7	
ersey City, N.J.	61	43	12	6			-	Nashville, Tenn.	143	89	31	10	6		
lew York City, N.Y.	1,272	759	262	179	34	38	46	W.S. CENTRAL	1,487	890	319	154	65	58	
lewark, N.J.	76	27	17	23	4	5	4	Austin, Tex.	60	43	11	3	2	1	
aterson, N.J.	25	12	7	5		ĭ		Baton Rouge, La.	59	29	12	6	5	7	
hiladelphia, Pa.	394	235	88	51	13	Ż	24	Corpus Christi, Tex.	51	34	8	4	5	-	
ittsburgh, Pa.§	62	40	12	6	2	2	4	Dallas, Tex.	205	105	46		9	10	
Reading, Pa.	13	10	2	1	-	-	1	El Paso, Tex.	64	39	18		3	1	
Rochester, N.Y.	119	85	16	11	7	-	9	Ft. Worth, Tex.	100	67	18		8	2	
Schenectady, N.Y. Scranton, Pa.§	25	20	3	2	-	-	-	Houston, Tex.	304	169	64		11	17	
Syracuse, N.Y.	23	20	2	1	-	-	3	Little Rock, Ark.	80	44	24		3	5 1	
renton, N.J.	79	53	16	5	4	1	6	New Orleans, La.	176	111	35	19	9	6	
Jtica, N.Y.	30 13	17 8	6	5	1	1	3	San Antonio, Tex. Shreveport, La.	233	153	45	20	9	2	
onkers, N.Y.	26	20	2 5	3	-	-	1	Tulsa, Okla.	51 104	37 59	8 30	4 8	1	6	
				1	-	-	2					_			
.N. CENTRAL	2,129	1,258	439	236	111	83	102	MOUNTAIN	848	544	165	84	31	24	
Akron, Ohio	87	62	11	10	1	3	-	Albuquerque, N.M.	83	54	17	6	3	3	
Canton, Ohio	38	27	. 8	3	-	-	4	Colo. Springs, Colo.		22	5	. 1	2		
Chicago, III. Cincinnati, Ohio	438	171	100	90	60	17	15	Denver, Colo.	122	65	28	18	7	4	
Cleveland, Ohio	126	81	29	4	6	6	11	Las Vegas, Nev. Ogden, Utah	169	107	40	17	2	3	
Columbus, Ohio	144 165	82		14	7	8	4	Phoenix, Ariz.	21	14	4	1	1	6	
Dayton, Ohio		84 74		25	7	2	8	Pueblo, Colo.	161	108	25	16	6	0	
Detroit, Mich.	117 236	136		16	:	3	6	Salt Lake City, Utah	20 102	16 57	2 18	1 18	1 6	3	
vansville, Ind.	43	36		23	8	18	4	Tucson, Ariz.	138	101	26	6	3	2	
ort Wayne, Ind.	52	41	6	1	-		1								
Sary, Ind.	22	11	4	2	3 5	1	2	PACIFIC	1,302	856	254	131	38	22	
Grand Rapids, Mich.	45	29		2	2	4	6 5	Berkeley, Calif.	23	16	4	1	1	1	
ndianapolis, Ind.	176	103		19	1	5	9	Fresno, Calif.	77	46	19	9	.1	2	
Madison, Wis.	51	37		4	i	-	10	Glendale, Calif.	Ū	U	U	ū	Ū	U	
Milwaukee, Wis.	126	93		6	3	4	6	Honolulu, Hawaii	57	37	11	7	2	2	
Peoria, III.	42	23		š	2	5	-	Long Beach, Calif.	83	53	18	6	4 U	ΰ	
Rockford, III.	40	32		2		1	2	Los Angeles, Calif. Pasadena, Calif.	U 22	U 12	U 5	U 3	ű	U	
South Bend, Ind.	46	32		2	3	4	4	Portland, Oreg.	118	85	27	4	2	1	
Toledo, Ohio	85	60		7	2	2	5	Sacramento, Calif.	147	107	25	11	4		
Youngstown, Ohio	50	44	. 3	3	-	-		San Diego, Calif.	140	79	26	20	7	7	
W.N. CENTRAL	753	554	111	54	15	10		San Francisco, Calif		84	30	36	4	4	
Des Moines, Iowa	69	55		4	10	19 1	34 4	San Jose, Calif.	158	102	44	8	2	2	
Duluth, Minn.	36	27		3	1	1	1	Santa Cruz, Calif.	31	25	3		3	-	
Kansas City, Kans.	28	21		1	3		1	Seattle, Wash.	143	100	25	12	4	2	
Kansas City, Mo.	123	86		19	2	2	7	Spokane, Wash.	49	38	5	5	1	-	
Lincoln, Nebr.	31	26			1	2	4	Tacoma, Wash.	96	72	12	9	2	1	
Minneapolis, Minn.	148	107		9	4	9	7	TOTAL	_						
Omaha, Nebr.	71	49		5	1	1	,	IJIAL	11,578	7,297	2,285	1,231	413	345	Ę
St. Louis, Mo.	122	93		8	ż	6	1								
St. Paul, Minn.	65	51		2	ī	-	6	1							
Wichita, Kans.	60	39	18			_	3	Ì							

^{*}Mortality data in this table are voluntarily reported from 121 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

Included.
Theumonia and influenza.
SBecause of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week.
Complete counts will be available in 4 to 6 weeks.
Total includes unknown ages.
U: Unavailable

Ischemic Heart Disease - Continued

TABLE 1. Age-adjusted death rates* for ischemic heart disease among adults aged ≥35 years and average annual percentage change in rates, by sex and state — United States, 1980–1988

		Wome	n		Men	
State	1980	1988	% Change	1980	1988	% Change
Alabama	366.5	308.3	-2.5	719.1	553.4	-3.4
Alaska	236.5	214.7	-1.8	690.6	500.4	-2.7
Arizona	357.9	297.7	-2.7	695.6	548.4	-3.1
Arkansas	371.3	330.6	-1.5	741.2	593.7	-2.8
California	373.8	319.2	-1.9	688.8	531.3	-3.1
Colorado	314.3	291.2	-1.8	660.2	544.1	-2.7
Connecticut	410.7	310.2	-3.7	785.7	536.2	-4.7
Delaware	333.0	280.1	-1.5	688.1	502.6	-3.8
District of Columbia	385.0	217.8	-7.5	716.3	356.9	-10.0
Florida	359.6	311.2	-1.9	726.6	551.9	-3.3
Georgia	385.2	327.9	-2.1	793.3	596.3	-3.5
Hawaii	257.4	184.0	-4.3	513.0	316.4	-5.3
Idaho	340.8	268.3	-2.9	703.9	504.8	-4.1
Illinois	500.6	390.7	-3.0	861.1	652.2	-3.5
Indiana	437.3	356.5	-2.6	239.3	684.6	-2.7
lowa	375.5	284.2	-3.6	792.7	567.4	-4.5
Kansas	372.7	294.8	-3.0	778.1	558.3	-3.9
Kentucky	447.8	363.1	-2.7	908.5	683.4	-3.4
Louisiana	437.8	353.1	-3.3	812.2	635.0	-3.4
Maine	441.6	328.0	-3.1	843.2	623.0	-3.4
Maryland	340.3	251.2	-3.4	613.1	427.7	-4.2
Massachusetts	477.0	329.0	-4.4	906.2	603.9	-5.0
Michigan	472.7	402.0	-1.8	861.9	671.9	-3.1
Minnesota	354.4	265.0	-3.2	743.5	540.8	-4.1
Mississippi	370.8	320.6	-2.6	725.8	589.7	-3.5
Missouri	444.2	352.5	-2.6	827.6	628.8	-3.4
Montana	353.8	250.6	-3.9	686.5	487.6	-3.4 -4.3
Nebraska	364.5	281.6	-3.3	740.4	550.0	-3.8
Nevada	356.1	233.4	-4.7	668.3	409.4	-6.3
New Hampshire	416.3	322.1	-2.8	851.9	591.8	-4.5
New Jersey	528.1	396.3	-3.4	949.3	688.9	
New Mexico	279.3	190.8	-3.9	518.3	330.5	-4.0
New York	604.2	462.6	-3.4	1039.7		-5.3
North Carolina	379.5	339.5	-3.4 -1.3		755.1	-4.1
North Dakota	349.1	286.2	-1.3 -2.9	828.7	693.4	-2.5
Ohio	475.2	370.4	-2.9 -3.0	782.2	558.2	-4.0
				913.5	665.8	-4.0
Oklahoma	399.2	364.9	-1.6	835.2	678.7	-2.7
Oregon	355.6	292.6	-2.3	777.3	566.9	-3.5
Pennsylvania	466.2	345.3	-3.6	852.2	603.3	-4.0
Rhode Island	490.3	336.2	-3.8	947.6	613.2	-4.6
South Carolina	400.8	361.8	-1.4	786.6	657.9	-1.9
South Dakota	358.4	286.4	-3.0	824.9	630.6	-3.7
Tennessee	413.5	341.1	-2.4	806.9	645.1	-3.3
Texas	341.9	270.9	-3.1	654.7	488.5	-3.7
Utah	289.5	233.2	-3.5	649.1	425.1	-5.3
Vermont	391.0	277.9	-2.9	771.6	544.8	-4.1
Virginia	398.4	307.5	-3.2	805.4	601.3	-3.9
Washington	338.7	248.5	-4.1	666.4	498.3	-3.9
West Virginia	472.8	382.1	-2.5	883.7	623.8	-4.2
Wisconsin	436.4	331.5	-2.9	819.8	633.9	-3.0
Wyoming	351.6	234.7	-4.6	757.4	498.1	-4.4
Median	375.5	310.2	-3.0	782.2	567.4	-3.8

^{*}Per 100,000 population, adjusted to the 1980 U.S. standard population.

Ischemic Heart Disease - Continued

reflect state-to-state differences in factors such as the socioeconomic resources (10), quality and availability of health-care services, and prevalence of risk factors for IHD (e.g., cigarette smoking, dietary patterns, physical activity, hypertension, and hypercholesterolemia).

References

- NCHS. Advance report of final mortality statistics, 1989. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, 1992. (Monthly vital statistics report; vol 40, no. 8, suppl 2).
- 2. Kleinman JC. State trends in infant mortality, 1968–1983. Am J Public Health 1986;76:681-7.
- Sempos C, Cooper R, Kovar MG, McMillen M. Divergence of the recent trends in coronary mortality for the four major race-sex groups in the United States. Am J Public Health 1988;78:1422-7.
- Verbrugge LM. Recent trends in sex mortality differentials in the United States. Women Health 1980:5:17–37.
- Uemura K, Pisa Z. Recent trends in cardiovascular disease mortality in 27 industrialized countries. World Health Stat Q 1985;38:142–62.
- Wingard DL, Cohn BA, Kaplan GA, Cirillo PM, Cohen RD. Sex differentials in morbidity and mortality risks examined by age and cause in the same cohort. Am J Epidemiol 1989;130: 601–10.
- 7. Stallones RA. The rise and fall of ischemic heart disease. Sci Am 1980;243:53-9.
- Wing SB, Casper ML, Riggan W, et al. Socioenvironmental characteristics associated with the onset of decline of ischemic heart disease mortality in the United States. Am J Public Health 1988;78:923

 –6.
- 9. Wing SB, Barnett EM, Casper ML, Tyroler HA. Geographic and socioeconomic variation in the onset of decline of coronary heart disease mortality in white women: empirical findings and theoretical development. Am J Public Health 1992;82:204–9.
- Wing S, Dargent-Molina P, Casper M, Riggin W, Hayes CG, Tyroler HA. Changing association between community occupational structure and ischemic heart disease mortality in the United States. Lancet 1987;2:1067–70.

Pregnancy Risks Determined from Birth Certificate Data — United States, 1989

The 1989 revision of the "U.S. Standard Certificate of Live Birth" includes new items of information about medical and lifestyle risk factors related to pregnancy, birth, and method of delivery (1,2). This report presents data about three of these new items: maternal weight gain during pregnancy, smoking during pregnancy, and method of delivery. For this analysis, to allow for more in-depth comparisons, data were limited to the two largest racial groups (black and white) in the United States.

During 1989, information for approximately 3.9 million live-birth certificates was recorded; however, because the District of Columbia, Rhode Island, Texas, and Virginia did not implement the revised certificates until March or April 1989, data on the new topics for the first three or four months of that year for those areas were not available. Overall, 17% of the birth certificates did not report maternal weight gain, 8% did not report data on smoking and 5% did not report on method of delivery (among areas reporting these items).

Maternal Weight Gain

From 1974 through 1989, the guideline for weight gain during a normal pregnancy was 22–27 pounds (3); in 1990, the National Institute of Medicine set guidelines for minimum aternal weight gain at 25–35 pounds for average-sized women (4). Analysis of 1989

birth-certificate data indicated that approximately 17% of white mothers and 27% of black mothers with gestations ≥40 weeks gained ≤20 pounds; white mothers gained, on average, one half pound more than black mothers (Table 1). The risk for insufficient weight gain was highest among women aged ≥35 years, those with less than a high school education, unmarried women, and women whose attendant at delivery was not a physician or a midwife. For each variable examined, black mothers were more likely to have low weight gain than white mothers (Table 1).

Among infants with gestations \ge 40 weeks, the percentage of low birthweight (LBW) (i.e., <2500 g [<5.5 lbs]) declined as maternal weight gain during pregnancy increased from <16 pounds to \ge 40 pounds (2.8%–0.8% for white infants and 6.8%–1.8% for black infants). The percentage of infants weighing at least 3500 g (7 lbs 12 oz) increased with maternal weight gain (for white infants, 44%–67%, and for black infants, 25%–50%) (Table 2).

Smoking

Data from the 1989 birth certificates were used to identify maternal characteristics (e.g., educational attainment of mother and adequacy of prenatal care*) that predict variations in smoking practices. To ensure the validity of educational attainment as an analytic variable, this analysis was confined to mothers aged ≥20 years.

During 1989, 19% of women who gave birth reported tobacco use during pregnancy[†]. However, smoking levels varied substantially by mother's educational attainment and adequacy of prenatal care. During 1989, 73% of births to women aged ≥20 years were to mothers who had adequate care, 20% to those who had intermediate care, and 7% to those who had inadequate care. Mothers whose care was inadequate were twice as likely to have smoked as those who had adequate care (32% versus 16%).

The differences by adequacy of care persisted for mothers with ≥12 years of education. Among comparably educated mothers, those who had inadequate prenatal care were considerably more likely to smoke than mothers who had adequate care (Table 3). However, among women with 9–11 years of education, 41%–46% were smokers, regardless of level of prenatal care. In addition, among mothers receiving adequate care, those with 9–11 years of education were eight times as likely to have smoked as were college graduates (41% versus 5%).

Among mothers aged ≥20 years, reported prevalences of smoking varied by educational attainment and race. At most levels of care and education, white mothers were more likely to smoke than were black mothers. White mothers with 9–11 years of education were more likely than any other group to smoke (44%–48%), regardless of care. Among black mothers with 9–11 years of education, 36% smoked overall; however, smoking prevalences were substantially higher for those with inadequate care (46%) than for those with intermediate (35%) or adequate care (30%).

Regardless of race, adequacy of prenatal care, or mother's educational level, babies born to mothers who smoked were at substantially elevated risk for LBW (Table 3). In addition, babies born to black women were substantially more likely to have LBW than were babies born to white women. For example, even among women with low risk for having LBW infants (i.e., nonsmoking mothers who were college

^{*}Adequacy of care as measured by the Kessner index provides a multidimensional measure that incorporates when prenatal care began, the total number of prenatal visits made by the mother, and the gestational age of the baby.

[†]Question as it appeared on 1989 live-birth certificate: "Tobacco use during pregnancy: yes/no."

TABLE 1. Percent distribution of live-born infants with gestations \geqslant 40 weeks, by maternal weight gain and selected characteristics of mothers — reporting areas, 1989

Characteristic	No.			Mater	nal weig	ht gain (pounds)		
of mother	births	<16	16–20	21–25	26–30	31–35	36-40	>40	Median
WHITE	1,335,840	7.0	9.7	15.3	21.4	16.1	13.5	17.0	30.7
Age (yrs)*							.0.0	17.0	30.7
<20	146,811	6.6	9.5	13.5	17.7	14.4	13.7	24.7	32.3
20–24	359,336	7.5	9.9	14.8	19.7	15.3	13.5	19.2	30.9
25–29	446,607	6.7	9.5	15.6	22.3	16.7	13.6	15.6	30.7
30–34 35–39	282,475	6.6	9.7	16.0	23.3	17.1	13.4	13.9	30.6
35–39 40–49	88,290	7.9	10.4	16.6	23.0	16.2	12.8	13.1	30.4
	12,321	10.4	12.1	17.2	22.0	14.5	11.5	12.4	30.1
Education (yrs) [†]									
0–11	223,884	10.2	11.7	14.8	18.1	13.5	12.2	19.5	30.5
12	473,530	7.8	10.0	15.1	20.4	15.5	13.3	18.0	30.7
13–15	249,118	6.2	9.2	15.5	22.1	16.7	14.0	16.3	30.8
≥16	235,540	3.9	8.2	16.3	25.4	18.9	14.3	12.9	30.8
Marital status*									
Married	1,100,592	6.7	9.5	15.5	22.0	40.5	40.0	404	30.7
Unmarried	235,248	8.5	10.7	14.4	22.0 18.4	16.5	13.6	16.1	
Attendant at	200,240	0.5	10.7	14.4	10.4	14.0	12.8	21.2	30.8
delivery*									
Physician	1 004 074								
Osteopathic	1,231,671	7.0	9.7	15.4	21.5	16.1	13.5	16.9	30.7
physician	45.670								
Certified nurse-	45,672	7.8	9.9	15.0	20.4	15.4	13.4	18.1	30.7
midwife	41,357	67		40.4					
Other midwife	7,367	6.7	8.6	13.4	19.9	17.1	14.8	19.5	31.6
Other	6,983	5.9 8.5	9.8	15.1	19.1	17.2	14.3	18.7	31.1
	•		11.4	15.7	20.8	14.7	12.6	16.3	30.5
BLACK	229,110	13.0	13.5	14.8	18.5	12.0	11.3	17.0	30.1
Age (yrs)*									
<20 20–24	54,089	12.6	14.6	15.5	18.2	11.6	11.0	16.7	29.7
25–24	75,845	12.8	13.5	14.6	18.6	11.8	11.3	17.4	30.2
30–34	57,036	12.7	12.5	14.3	18.5	12.5	11.8	17.5	30.3
35–34 35–39	30,483	13.4	13.2	14.7	18.4	12.5	11.1	16.6	30.2
40–49	10,047	15.5	14.1	14.6	18.9	11.6	10.4	14.8	28.7
· -	1,610	18.5	14.0	15.9	18.4	10.6	9.9	12.6	26.8
Education (yrs)†									
0–11	64,410	15.8	15.4	15.3	17.6	10.6	10.2	15.1	27.9
12	92,688	13.0	13.5	14.6	18.5	12.0	11.3	17.1	30.2
13–15	40,830	10.4	11.3	14.8	19.0	13.0	12.4	19.2	30.6
≥16	15,375	9.0	10.9	14.3	20.7	15.2	12.4	17.5	30.6
Marital status*									
Married	80,921	11.6	11.9	14.8	19.2	13.0	11.9	17.6	30.4
Unmarried	148,189	13.7	14.4	14.8	18.0	11.4	11.0	16.7	29.7
Attendant at	•				.0.0	11	11.0	10.7	20.7
delivery*									
Physician	210,132	12.9	13.5	14.8	18.5	12.0	11.0	17.0	20.1
Osteopathic	210,132	12.3	13.5	14.0	10.5	12.0	11.3	17.0	30.1
physician	5,280	11.5	12.8	14.6	17.6	12.8	12.1	18.5	30.4
Certified nurse-	2,200			14.0	17.0	12.0	12.1	10.5	30.4
midwife	10,082	13.6	12.7	14.7	17.7	12.4	11.6	17.2	30.1
Other midwife	417	11.2	14.0	16.4	18.9	13.2	11.2	15.1	30.1
Other	2,084	17.5	14.9	13.8	17.1	11.3	10.3	15.1	28.8
*Excludes data for Ca	lifornia. Louis	ana. Ne	braska, a					20.0	-6

Excludes data for California, Louisiana, Nebraska, and Oklahoma, which did not require reporting of weight gain during pregnancy.

Excludes data for California, Louisiana, Nebraska, New York (except for New York City), Oklahoma, and Washington, which did not require reporting of weight gain during pregnancy or educational attainment.

TABLE 2. Number and percent distribution of live-born infants with gestations ≥40 weeks, by birthweight according to maternal weight gain and race of mother - reporting areas, 1989*

Door of mother/			Matern	al weight gain	(pounds)			
Race of mother/ Birthweight (grams)	<16	16–20	21–25	26-30	31–35	36-40	>40	
				N	umber			Total
White	79,818	111,120	175,045	243,986	183,977	154,168	194,257	1,335,840 †
				Percent	distribution			
<2500	2.8	2.2	1.5	1.1	0.9	0.9	0.8	1.3
2500-2999	14.7	13.3	11.1	8.9	7.5	6.4	5.4	9.1
3000-3499	38.5	39.4	39.2	36.1	33.5	30.6	26.6	34.5
3500-3999	31.8	33.4	35.6	38.6	40.4	40.9	40.6	37.9
≥4000	12.1	11.7	12.6	15.2	17.7	21.2	26.6	17.2
				No	umber			Total
Black	23,026	23,938	26,254	32,758	21,306	20,063	30,182	229,110 [†]
				Percent	distribution			
<2500	6.8	5.4	3.7	3.0	2.4	2.3	1.8	3.8
2500-2999	25.5	23.4	20.8	17.7	15.7	13.4	11.0	18.3
3000-3499	42.4	43.8	44.6	44.2	42.9	40.9	36.6	42.1
3500-3999	20.6	22.3	25.1	27.8	30.6	33.0	35.7	27.6
≥4000	4.7	5.1	5.8	7.3	8.5	10.4	14.9	8.2

^{*}Excludes data for California, Louisiana, Nebraska, and Oklahoma, which did not require reporting of weight gain during pregnancy. †Includes births with weight gain not stated.

graduates and received adequate prenatal care), 8% of black infants and 4% of white infants had LBW.

Method of Delivery

In 1989, 23% of all births and 16% of births to mothers who had no previous cesarean were by cesarean delivery (Table 4). Overall and primary cesarean rates differed markedly by the mother's residence, age, parity, and educational attainment. Rates were highest for mothers who resided in the South, were in the oldest years of childbearing, were having their first child, and had ≥13 years of education. Rates were similar, however, by race.

At least half the infants were delivered by cesarean when eclampsia (52%), abruptio placenta (57%), fetal distress (63%), dysfunctional labor (64%), cord prolapse (68%), placenta previa (82%), malpresentation (84%), or cephalopelvic disproportion (98%) were diagnosed (1). The risk for cesarean increased with maternal weight gain during pregnancy, from approximately 21% for mothers who gained <31 pounds, to 29% for mothers who gained ≥41 pounds. Cesarean rates were highest for babies weighing <2500 g or ≥4000 g, for babies born prematurely, for twins, and for babies with certain abnormal conditions (e.g., hyaline membrane disease, meconium aspiration syndrome, or need for assisted ventilation).

TABLE 3. Percentage of mothers aged ≥20 years who smoked during pregnancy and percentage of low birthweight (LBW)* infants, by mother's smoking status, educational attainment, and race and adequacy of prenatal care[†] — reporting areas, 1989[§]

						% L	.BW		
	% Moth	ners who	smoked	-	Smoker	s	N	onsmok	ers
Adequacy of prenatal care/ Education of mother	All	White	Black	All	White	Black	All	White	Black
Adequate care [¶] 9–11 yrs 12 yrs 13–15 yrs ≥16 yrs	16.1	16.6	14.7	9.0	8.1	17.5	4.8	4.1	9.8
	40.5	43.6	30.3	10.7	9.3	19.5	6.6	5.2	11.5
	20.7	21.7	15.4	8.8	7.9	17.3	5.0	4.2	9.9
	12.4	12.7	11.0	8.4	7.5	16.3	4.7	4.0	9.7
	4.8	4.9	5.4	6.8	6.3	14.2	4.2	3.9	8.4
Intermediate care [¶] 9–11 yrs 12 yrs 13–15 yrs ≥16 yrs	24.8	26.6	21.6	12.2	10.2	20.1	6.7	5.3	11.2
	42.6	46.6	34.5	13.3	11.2	21.2	7.8	6.1	12.1
	26.6	29.5	20.1	11.5	9.6	19.5	7.0	5.4	11.3
	17.0	17.7	16.2	11.7	9.8	18.5	6.3	5.0	10.5
	6.0	5.9	9.5	12.5	10.1	22.6	5.8	5.1	10.1
Inadequate care [¶] 9–11 yrs 12 yrs 13–15 yrs ≥16 yrs	32.3	32.8	34.1	20.2	14.8	30.4	9.9	6.8	16.4
	46.3	47.6	46.1	20.6	14.9	30.9	11.5	7.8	18.2
	32.4	35.7	29.5	20.0	14.8	29.8	10.7	7.1	16.1
	24.4	24.5	25.8	19.3	13.7	28.8	9.4	6.6	14.5
	10.1	8.9	18.0	18.9	12.7	31.4	6.5	5.3	11.7
All levels of care ¹ 9–11 yrs 12 yrs 13–15 yrs ≥16 yrs	19.1	19.3	20.0	11.5	9.4	22.1	5.6	4.5	11.2
	42.2	45.1	36.2	13.7	11.0	24.3	7.9	5.9	13.3
	22.8	23.9	19.3	10.9	9.0	21.4	5.9	4.6	11.3
	13.7	13.8	13.9	10.1	8.3	19.4	5.3	4.3	10.4
	5.0	5.0	6.0	8.1	7.0	18.2	4.5	4.1	9.0

^{*}Birthweight <2500 a.

[†]Adequacy of prenatal care measured by Kessner index.

⁵Excludes data for California, Indiana, Louisiana, Nebraska, New York, Oklahoma, South Dakota, and Washington, which did not require reporting of either tobacco use during pregnancy or educational attainment of mother.

Includes births to mothers with 0-8 years of education.

TABLE 4. Rates of cesarean delivery and vaginal birth after a previous cesarean (VBAC), by selected maternal and infant characteristics — reporting areas, 1989*

		Rate				Rate	
Characteristic	Primary [†]	VBAC ⁵	Total*	Characteristic	Primary	VBAC	Total
MATERNAL							
Residence				Obstetric procedures			
Northeast	16.4	21.0	23.2	Electronic fetal monitoring	16.3	25.8	21.4
Midwest	15.0	19.2	22.0	Induction of labor	21.1	50.4	22.4
South	17.8	16.0	24.7	Stimulation of labor	17.4	58.3	18.6
West	14.9	20.1	21.2	Tocolysis	24.6	22.0	30.5
				Ultrasound ^{¶¶}	19.2	18.1	27.2
Race				Weight gain (lbs)			
White	16.2	18.6	23.2	<21	13.8	19.0	21.6
Black	15.8	19.7	22.0	21-30	14.1	20.5	21.1
				31-40	17.0	19.5	23.5
Age (yrs)				≥41	23.2	16.5	29.1
<20	15.0	24.0	16.9				
20–29	15.7	19.3	22.2	Total	16.1	18.9	22.8
30–39	17.2	18.0	26.5				
40–49	23.2	14.0	31.9	INFANT			
40 40	20.2		••	Birthweight (g)			
Parity				<1500	39.4	21.4	42.8
1st birth	24.8	26.0	24.9	<2500	29.4	18.6	34.2
2nd birth	8.9	18.6	22.9	2500-2999	14.8	19.0	21.3
≥3rd birth	8.6	19.1	19.7	3000-3999	14.0	19.3	21.0
2514 Birtii	0.0			≥4000	21.9	16.8	29.1
Education (yrs)**				1000			
0–11	13.0	19.3	18.8				
12	16.1	17.3	23.2	Weeks of gestation			
≥13	17.9	19.3	24.8	<37	22.6	18.2	28.5
0				37–39	14.4	14.1	24.1
Complications of labor				40	14.1	26.1	19.0
and/or delivery				41	17.2	28.5	21.0
Fever	34.3	38.8	36.4	⇒42	18.6	23.7	23.2
Premature	04.0	30.0	50.4		10.0	20.7	23.2
rupture of membrane	26.3	28.5	29.7				
	52.6	16.2	56.5	Plurality			
Abruptio placenta	78.4	3.3	82.2		45.0	40.0	
Placenta previa	78.4 41.4	40.3	42.3	Singleton Twin	15.3	19.2	22.1
Prolonged labor				I win	49.0	7.8	54.2
Dysfunctional labor	62.1	18.4	63.8	Ab.,			
Breech/malpresentation	82.6	5.1	83.9	Abnormal conditions			
Cephalopelvic				Hyaline membrane			
disproportion	97.5	0.9	97.7	disease/respiratory			
Cord prolapse ⁵⁵	66.2	10.8	68.0	distress syndrome	41.5	10.0	48.7
Fetal distress	61.1	16.8	62.9	Meconium aspiration			
				syndrome***	33.4	20.4	37.4
				Assisted ventilation,			
				<30 minutes ^{†††}	30.1	15.3	37.1
				Assisted vetilation,			
				≥30 minutes ^{†††}	43.8	13.0	49.0

^{*}Excludes data for Louisiana, Maryland, Nebraska, Nevada, and Oklahoma (except as noted), which did not require reporting of method of delivery.

Number of primary cesareans per 100 live births to women who had not had a previous cesarean.

⁵Number of VBAC per 100 live births to women with a previous cesarean.

Percentage of all live infants delivered by cesarean.

^{**}In addition, excludes data for New York (except for New York City) and Washington, which did not require reporting of educational attainment.

^{††}In addition, excludes data for Texas.

⁵⁵ In addition, excludes data for North Carolina.

[&]quot;In addition, excludes data for Illinois.

^{***}In addition, excludes data for New York (except for New York City).

^{***}In addition, excludes data for New York.

Less than 20% of infants born to mothers who had a previous cesarean were delivered vaginally (VBAC). VBAC rates were generally lowest for women comprising the groups with highest overall and primary cesarean rates.

Reported by: Div of Vital Statistics, National Center for Health Statistics, CDC.

Editorial Note: The findings in this report regarding maternal weight gain are consistent with a previous study, based on the 1980 National Natality Survey, that indicated substantial disparities in weight gain by race and sociodemographic status and that women with inadequate weight gain during pregnancy generally have lower weight live-born infants and a higher risk for a fetal death (5). Insufficient weight gain among high-risk women remains a public health concern because of its association with poor birth outcomes.

The large differences in maternal weight gain by race are consistent with the medical advice on weight gain reported by mothers (i.e., black women are more likely to report being advised to gain less weight than currently recommended, regardless of their age, education, or marital status) (6). Therefore, physicians and other health-care providers who advise on maternal weight gain during pregnancy should provide the recent guidelines of 28–40 pounds for women with low weight-for-height, 25–35 pounds for average weight-for-height women, and 15–25 pounds for women with high weight-for-height, for full-term pregnancies (4).

Results on smoking by women during pregnancy from this analysis are consistent with results from the 1988 National Maternal and Infant Health Survey, conducted by CDC's National Center for Health Statistics (NCHS). Smoking by women during pregnancy is associated with a variety of poor pregnancy outcomes, including higher rates of miscarriage, LBW, intrauterine growth retardation, and preterm birth (7). In turn, LBW and preterm birth are major predictors of infant mortality and infant and childhood morbidity. Therefore, intense intervention efforts to curtail smoking should be directed toward the group of pregnant women having the highest prevalence of smoking (i.e., mothers with less than a high school education).

The racial disparity in the incidence of LBW persisted even when births to women of the same education, prenatal care, and smoking status were compared. Therefore, determination of reasons for this difference will require further assessment of other lifestyle, nutritional, and environmental factors.

Since 1965, the National Hospital Discharge Survey (NHDS), conducted by NCHS, has provided national data on cesarean delivery rates. In this report, the cesarean delivery rates based on data from live-birth certificates are consistent with rates derived from the 1989 NHDS (1,8). The addition of the question regarding method of delivery to the 1989 revised "U.S. Standard Certificate of Live Birth" has provided public-health practitioners and health researchers with a basis for determining cesarean rates for a wider range of maternal demographic and health characteristics, as well as for smaller geographic areas and by health characteristics of the infant.

Since the early 1980s, the American College of Obstetricians and Gynecologists has encouraged an expanded use of VBAC to reduce the overall cesarean rate (9). Information now available from birth certificates will permit health professionals to more closely monitor changes in cesarean and VBAC rates and compare their rates to international, state, or small-area rates.

The findings in this report have at least two limitations. First, reporting may not be as complete as it could be in subsequent years because 1989 was the first year of reporting with this revised birth certificate and three states and the District of

Columbia did not begin reporting new data immediately. Second, for data on maternal weight gain, because prepregnancy weight and height are not recorded on the birth certificate, it was not possible to determine what proportion of women achieved the recommended levels for their weight-for-height; no other sources exist on maternal weight gain from health-care providers for comparison. Despite these limitations, the new data from the revised live birth certificates will facilitate tracking of results of public health initiatives designed to improve maternal health and pregnancy outcome.

References

- 1. NCHS. Advance report of new data from the 1989 birth certificate. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, CDC, 1992. (Vital and health statistics; vol 40, no. 12, suppl).
- Ventura SJ. New insights in maternal and infant health from the 1989 birth certificate [Abstract]. In: Program and abstracts of the 1992 annual meeting of the Population Association of America. Denver: Population Association of America, 1992:183.
- 3. American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Guidelines for perinatal care. Evanston, Illinois: American Academy of Pediatrics, 1983.
- Subcommittee on Nutritional Status and Weight Gain During Pregnancy, Institute of Medicine. Nutrition during pregnancy. Part I—weight gain. Part II—nutrient supplements. Washington, DC: National Academy of Sciences, 1990:11–2.
- Taffel SM, NCHS. Maternal weight gain and the outcome of pregnancy: United States, 1980. Hyattsville, Maryland: US Department of Health and Human Services, Public Health Service, 1986. (Vital and health statistics; vol 21, no. 44).
- Taffel SM, Keppel KG, Jones GK. Medical advice on maternal weight gain and actual weight gain: results from the 1988 National Maternal and Infant Health Survey [Abstract]. In: Proceedings of the Conference on Maternal Nutrition and Pregnancy Outcome (in press).
- CDC. Reducing the health consequences of smoking: 25 years of progress—a report of the Surgeon General. Rockville, Maryland: US Department of Health and Human Services, Public Health Service, 1989; DHHS publication no. (CDC)89-8411.
- 8. Taffel SM, Placek PJ, Moien M. Kosary CL. 1989 U.S. cesarean section rate steadies VBAC rate rises to nearly one in five. Birth 1991;18:73–7.
- Committee on Obstetrics, American College of Obstetricians and Gynecologists. Guidelines for vaginal delivery after a previous cesarean birth. Washington, DC: American College of Obstetricians and Gynecologists, 1988.

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Public Health Service
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HHS Publication No. (CDC) 92-8017