# SPECIAL ARTICLE

# Annual Report to the Nation on the Status of Cancer (1973 Through 1998), Featuring Cancers With Recent Increasing Trends

Holly L. Howe, Phyllis A. Wingo, Michael J. Thun, Lynn A. G. Ries, Harry M. Rosenberg, Ellen G. Feigal, Brenda K. Edwards

Background: The American Cancer Society, the National Cancer Institute (NCI), the North American Association of Central Cancer Registries, and the Centers for Disease Control and Prevention, including the National Center for Health Statistics (NCHS), collaborate to provide an annual update on cancer occurrence and trends in the United States. This year's report contains a special feature that focuses on cancers with recent increasing trends. Methods: From 1992 through 1998, age-adjusted rates and annual percent changes are calculated for cancer incidence and underlying cause of death with the use of NCI incidence and NCHS mortality data. Joinpoint analysis, a model of joined line segments, is used to examine long-term trends for the four most common cancers and for those cancers with recent increasing trends in incidence or mortality. Statistically significant findings are based on a P value of .05 by use of a two-sided test. State-specific incidence and death rates for 1994 through 1998 are reported for major cancers. Results: From 1992 through 1998, total cancer death rates declined in males and females, while cancer incidence rates declined only in males. Incidence rates in females increased slightly, largely because of breast cancer increases that occurred in some older age groups, possibly as a result of increased early detection. Female lung cancer mortality, a major cause of death in women, continued to increase but more slowly than in earlier years. In addition, the incidence or mortality rate increased in 10 other sites, accounting for about 13% of total cancer incidence and mortality in the United States. Conclusions: Overall cancer incidence and death rates continued to decline in the United States. Future progress will require sustained improvements in cancer prevention, screening, and treatment. [J Natl Cancer Inst 2001;93:824–42]

The American Cancer Society (ACS), the National Cancer Institute (NCI), the North American Association of Central Cancer Registries (NAACCR), and the Centers for Disease Control and Prevention (CDC), including the National Center for Health Statistics (NCHS) and the Center for Chronic Disease Prevention and Health Promotion, collaborate to produce an annual report on the current burden of cancer in the United States. Four years ago, the initial report documented the first sustained decline in cancer death rates, a notable reversal in the increases documented since national record keeping was instituted in the 1930s (1). The second and third reports updated and confirmed these declines in both cancer incidence and death rates (2,3). Special features in these reports highlighted lung cancer and the tobacco epidemic (2) and opportunities to improve the prevention, early detection, and treatment of colorectal cancer (3). The current report examines trends from 1973 through 1998 in the incidence and death rates for the four most common cancers (breast, prostate, lung, and colon–rectum cancers), which comprise more than half the cancer burden. This report also features specific cancers for which incidence or death rates increased in one or more population subgroups during the period from 1992 through 1998.

# SUBJECTS AND METHODS

All statistics plus additional data and information on cancer incidence, mortality, and survival are available from the following Internet addresses: URL, www.seer.cancer.gov (NCI); www.naaccr.org/CINAPlus/index.html (NAACCR); www.cdc.gov/cancer/NPCR (CDC); www.cdc.gov/nchs/about/ major/dvs/mortdata.htm (NCHS); and www.cancer.org (ACS). More detailed information, figures, and methodology pertaining to this report are available at the NCI Internet address.

#### **Cancer Cases and Deaths**

Information on newly diagnosed cancer cases in the United States is based on data collected by registries in the NCI's Surveillance, Epidemiology, and End Results (SEER) Program (4) and the CDC's National Program of Cancer Registries (NPCR) (5). NAACCR evaluates and publishes data annually from registries in both programs (6).

For the long-term trend analyses, SEER incidence data for 1973 through 1998 were used. (These data are based on 10% of the U.S. population.) For the analyses of more recent trends, the SEER incidence data for 1992 through 1998 were used; these data are based on 14% of the U.S. population. For estimates of the proportion of cases and deaths contributed by each cancer, 1998 incidence data from SEER (covering 14% of the U.S. population) and 1998 U.S. mortality data were used. State incidence rates are provided for the four most common cancers from 30 registries that meet NAACCR criteria for highest quality data for 1994 through 1998 (6–8). Approximately 53% of the U.S. population is included in the NAACCR combined cancer incidence rates. All information on primary site and histology for incidence is converted to the International Classification of Diseases for Oncology, 2<sup>nd</sup> edition (9), with the use of site groups published previously (4).

Affiliations of authors: H. L. Howe, North American Association of Central Cancer Registries, Springfield, IL; P. A. Wingo, Division of Cancer Prevention and Control, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention (CDC), Atlanta, GA; M. J. Thun, Epidemiology and Surveillance Research Department, American Cancer Society, Atlanta; L. A. G. Ries, B. K. Edwards (Division of Cancer Control and Population Sciences), E. G. Feigal (Division of Cancer Treatment and Diagnosis), National Cancer Institute, Bethesda, MD; H. M. Rosenberg, Division of Vital Statistics, National Center for Health Statistics, CDC, Hyattsville, MD.

*Correspondence to:* Holly L. Howe, Ph.D., North American Association of Central Cancer Registries, Inc., 2121 W. White Oaks Dr., Springfield, IL 62704–6495 (e-mail: hhowe@naaccr.org).

See "Notes" following "References."

© Oxford University Press

Downloaded from https://academic.oup.com/jnci/article/93/11/824/2906147 by guest on 16 August 2022

Cancer deaths in the United States are reported to state vital statistics offices and are consolidated into a national database by NCHS through the National Vital Statistics System (NVSS) (10). NVSS codes the underlying cause of death according to the version of the International Classification of Diseases (ICD) in use in the United States at the time (11,12). A conversion algorithm ensures comparability between versions of ICD codes, and they are categorized according to SEER site groups (4).

# **Cancer Incidence and Death Rates**

Age-adjusted cancer incidence and death rates were computed by use of aggregated annual county resident population estimates from the U.S. Bureau of the Census. Population data for Hawaii were adjusted slightly for an over-count of white persons.

All rates were expressed per 100 000 population and were age-adjusted by the direct method to the 1970 U.S. standard million population. For this report, a rate was calculated when at least 20 cases or deaths occur in the category of interest. The term "all sites" refers to all sites combined (except basal and squamous cell carcinoma of the skin), including those not shown in the tables or figures.

This year's report features 12 cancers for which incidence or death rates increased from 1992 through 1998, in contrast to the decreasing trends noted for most cancers and for all cancer sites combined. For these cancers, either the SEER incidence rate (based on 14% of the population) or the U.S. death rate increased in either sex among the total, white, or black population. Age-specific trends were not used in the selection of the cancers with increasing trends. The interval of 1992 through 1998 was chosen because the decline in cancer incidence rates began in 1992 (1) and 1998 was the most recent year for which incidence and mortality data were available. Combining all data from 1992 through 1998 also improved the precision of point estimates and the data interpretability for all populations in the analyses (Hispanic, black, American Indian/Alaska Native [AI/AN], Asian/Pacific Islander [API], and white). State cancer incidence and death data for 1994 through 1998 were provided to maximize geographic coverage (for incidence data) and to include the most recent 5 years of available data.

#### **Statistical Analyses**

Annual percent change (APC) (4) was used to describe the recent trends from 1992 through 1998, with statistical significance assessed with the use of a twosided P = .05. The APC for the recent trends is the best measure for comparisons among groups because both the beginning and ending years are the same (3).

Long-term trends are better described by joinpoint analysis (JPA), a model of joined lines (straight lines on a log scale). JPA chooses a model of line segments, such that each is joined at points called a "joinpoint." Each joinpoint denotes a statistically significant change in trend (13). For JPA, the overall statistical significance level was set to P = .05, with a maximum of three joinpoints and four line segments allowed. An APC was used to describe the trend for each line segment. On the figures, lines represent predicted trends and symbols represent observed rates.

# RESULTS

# General Update on Overall Trends: All Sites Combined

Cancer incidence rates for all sites combined decreased from 1992 through 1998 among all persons in the United States (Fig. 1), primarily because of a decline of 2.9% per year in white males and 3.1% per year in black males (Table 1). Among females, cancer incidence rates increased 0.3% per year. During 1992 through 1998, overall cancer death rates declined 1.1% per year (Table 2; Fig. 1). The change was greater among males than among females (declines of 1.6% and 0.8% annually, respectively), with the greatest decrease in black males (decline of 2.0% per year).

Long-term trends in incidence and mortality were analyzed (Fig. 1; Table 1) both with and without the inclusion of prostate cancer to assess the effects of the sudden increase in diagnosis of prostate cancer on the overall cancer incidence and to help put recent trends in context. JPA revealed three trends in overall

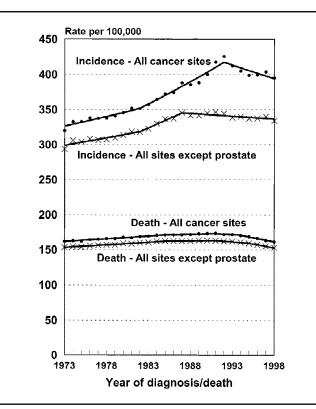


Fig. 1. All cancers and all cancers except prostate, incidence and death rates, joinpoint analyses for 1973 through 1998, all races, both sexes. Incidence data are from Surveillance, Epidemiology, and End Results Program areas covering 10% of the U.S. population. Death data are from the National Center for Health Statistics covering the entire U.S. population. Rates are per 100 000 persons and are age-adjusted to the 1970 U.S. standard million population.

cancer incidence rates from 1973 through 1998 for all sites, both sexes, and all races combined: Rates increased from 1973 through 1982, accelerated from 1982 through 1992, and declined from 1992 through 1998<sup>1</sup> (Fig. 1; Table 1). The exclusion of prostate cancer eliminates the sharp increase in cancer incidence that peaked in 1992 and suggests that the decrease in all other cancers actually began 5 years earlier (in 1987). The recent decline in cancer incidence is evident both with and without the inclusion of prostate cancer. Long-term mortality trends were similar regardless of the inclusion or exclusion of prostate cancer. When all sites are combined, death rates increased from 1973 through 1991, were level from 1991 through 1994, and declined 1.4% per year from 1994 through 1998 (Fig. 1; Table 2). The trends in cancer incidence and death rates varied by sex and race (Tables 1 and 2).

# Update on the Most Common Cancer Sites

Cancers of the breast, prostate, lung, and colon–rectum accounted for 55.9% of all 1998 SEER cases and 52.7% of all 1998 cancer deaths in the United States. Changes in the rates for these sites, therefore, have a strong influence on overall cancer trends. Results from incidence and mortality trend analyses for the top 10 cancers are provided on the NCI Internet site. The 1992 through 1998 trends in incidence rates for these cancers were level or were declining, with the exception of increasing trends in melanoma and in female breast cancer. Death rates decreased for all of the top 10 sites, except non-Hodgkin's lymphoma (NHL) and female lung cancer, both of which were increasing.

 Table 1. Cancer incidence rates and trends for 1992 through 1998 and joinpoint analyses for 1973 through 1998 for the most common cancers and the 12 cancers with recent increasing rates by site, sex, and race\*

			Joinpoint analyses (1973–1998)										
Site	Average annual rate	APC†	Trend 1		Trend 2		Trend 3		Trend 4				
	(1992–1998)	(1992–1998)	Range of years	APC†	Range of years	APC†	Range of years	APC†	Range of years	APC†			
All sites	397.8	-1.1‡	1973–1982	0.8‡	1982–1992	1.7‡	1992-1998	-1.0‡					
Male	473.3	-2.7‡	1973-1989	1.4‡	1989-1992	5.2‡	1992-1995	-4.3‡	1995-1998	-0.8			
White	470.4	-2.9	1973–1989	1.4‡	1989–1992	5.2‡	1992-1995	-4.8‡	1995–1998	-0.8			
Black	596.8	-3.1‡	1973-1981	2.6‡	1981–1989	0.8‡	1989–1993	5.4‡	1993-1998	-4.2‡			
Female	345.2	0.3‡	1973–1980	0.1	1980–1987	1.6‡	1987–1998	0.3‡					
White Black	354.4 337.6	0.3 -0.1	1973–1980 1973–1991	0.2 1.2‡	1980–1987 1991–1998	1.7‡ -0.2	1987–1998	0.3‡					
Female breast	111.2	1.2‡	1973–1980	-0.7	1980–1987	3.8‡	1987–1998	0.5					
White	115.5	1.1‡	1973–1980	-0.6	1980–1987	3.9‡	1987–1998	0.4					
Black	101.5	0.1	1973–1979	-0.8	1979–1986	4.0‡	1986–1998	0.9‡					
Prostate	149.7	-5.1‡	1973–1988	2.9‡	1988–1992	17.5‡	1992–1995	-10.0‡	1995–1998	0.3			
White	144.6	-5.7‡	1973–1988	3.1‡	1988-1992	17.4‡	1992–1995	-11.3‡	1995–1998	0.4			
Black	234.2	-4.0‡	1973–1989	2.2‡	1989–1992	22.0‡	1992–1998	-4.0‡					
Lung	54.4	-1.6‡	1973-1981	3.0‡	1981-1991	0.9‡	1991-1998	-1.2‡					
Male	71.1	-2.7‡	1973–1981	1.9‡	1981–1991	-0.4‡	1991–1998	-2.4‡					
White	69.6	-2.7‡	1973–1981	1.7‡	1981–1991	-0.4‡	1991–1998	-2.5‡					
Black	107.2	-3.2‡	1973-1984	3.0‡	1984-1998	-1.8‡	1002 1001	2.1.1	1001 1000	0.0			
Female	41.8	-0.2	1973-1976	9.1‡	1976-1983	5.2‡	1983-1991	3.1‡	1991–1998	0.2			
White Black	43.6 45.7	-0.1 -0.3	1973–1982 1973–1990	6.5‡ 4.7‡	1982–1991 1990–1998	3.2‡ -0.7	1991–1998	0.4					
Colon-rectum	43.3	-0.7	1973–1985	0.8‡	1985–1995	-1.8‡	1995–1998	0.5					
Male	51.8	-1.1	1973–1986	1.1‡	1986–1995	-2.1‡	1995–1998	0.1					
White	51.4	-1.3‡	1973–1986	1.0‡	1986–1995	-2.2	1995–1998	0.1					
Black	57.7	-1.1	1973–1980	4.6‡	1980–1998	-0.1	1770 1770	011					
Female	36.7	-0.4	1973–1984	0.5‡	1984-1996	-1.6‡	1996-1998	2.5					
White	36.3	-0.4	1973-1984	0.5‡	1984-1995	-1.9	1995-1998	1.1					
Black	44.7	-0.3	1973-1980	2.9‡	1980–1998	-0.3							
Non-Hodgkin's lymphoma	15.7	0.1	1973–1991	3.5‡	1991-1998	0.4							
Male	19.5	-0.5	1973-1990	4.0‡	1990-1995	2.0	1995–1998	-3.3					
White	20.2	-0.8	1973–1979	2.4‡	1979–1988	5.0‡	1988-1995	1.7‡	1995–1998	-3.3‡			
Black	16.0	-0.1	1973–1998	4.1‡									
Female	12.5	1.0‡	1973–1988	2.9‡	1988–1998	1.4‡							
White Black	13.0 8.7	0.7 4.9‡	1973–1988 1973–1998	3.0‡ 3.0‡	1988–1998	1.3‡							
Liver and IBD <sup>†</sup>	4.3	4.4‡	1973–1983	0.8	1983-1998	4.4‡							
Male	6.6	4.0‡	1973–1984	1.7‡	1984–1998	4.4‡							
White	5.2	4.0‡	1973–1984	0.8	1984–1998	4.7‡							
Black	8.0	1.1	1973–1998	2.4‡	1701 1770								
Female	2.5	4.7‡	1973–1980	-1.8	1980-1998	3.9‡							
White	2.0	5.5‡	1973-1978	-4.8	1978-1998	3.1‡							
Black	2.8	4.1		§									
Esophagus	3.7	-0.3	1973–1998	0.5‡									
Male	6.2	-0.2	1973-1998	0.7‡	1000								
White	5.9	0.9	1973–1981	-0.4	1981–1998	1.8‡							
Black	11.8	-6.7‡	1973–1986	1.5	1986–1998	-4.9‡							
Female	1.7	-0.9	1973-1998	-0.2									
White Black	1.6 4.0	-0.7 -3.6	1973–1998 1973–1981	0.0 4.3	1981-1998	-2.3‡							
			1973–1981										
Melanoma Male	12.9 16.0	2.8‡ 2.8‡	1973–1981 1973–1985	6.1‡ 5.7‡	1981–1998 1985–1998	2.8‡ 3.0‡							
White	18.2	2.84 2.7‡	1973–1985	5.7÷ 7.7‡	1980–1998	3.04 3.9‡	1996-1998	-2.1					
Black	18.2	2.7÷ §	1775-1900	1.1÷ §	1700-1990	5.94	1770-1990	2.1					
Female	10.6	8 2.8‡	1973-1980	8 6.1‡	1980-1998	2.1‡							
White	12.3	2.9‡	1973–1980	6.7‡	1980–1998	2.3							
Black	0.7	§		§		04							
Acute myeloid leukemia	2.7	1.1	1973–1987	-0.9‡	1987–1998	2.1‡							
Male	3.3	1.8‡	1973-1988	-0.5	1988-1998	1.9‡							
White	3.5	1.7	1973-1988	-0.7‡	1988-1998	2.2‡							
Black	2.4	2.7		§									
Female	2.3	0.4	1973-1987	-1.1‡	1987-1998	2.4‡							
White	2.3	0.9	1973-1986	-1.4‡	1986-1998	2.3‡							
Black	2.1	-1.8		§									

(Table continues)

			Joinpoint analyses (1973–1998)										
	Average		Trend 1		Trend 2		Trend 3		Trend 4				
Site	annual rate (1992–1998)	APC† (1992–1998)	Range of years	APC†	Range of years	APC†	Range of years	APC†	Range of years	APC†			
Soft tissue (including heart)	2.4	2.0	1973–1990	0.5‡	1990–1998	2.4‡							
Male	2.8	1.0	1973-1998	0.9									
White	2.8	0.8	1973-1998	$0.8^{+}_{+}$									
Black	2.9	-0.1		ş									
Female	2.1	3.2	1973-1992	0.5	1992-1998	3.9‡							
White	2.1	3.3‡	1973–1998	1.2‡	1//2 1//0	0.77							
Black	2.3	2.1	1775 1770	§									
Thyroid	5.3	2.7‡	1973-1977	5.3‡	1977-1980	-6.4	1980–1998	2.5‡					
Male	2.9	0.1	1973-1977	6.5	1977-1980	-8.4	1980-1998	2.1‡					
White	3.0	0.5	1973-1977	7.6‡	1977-1980	-8.5	1980-1998	2.6‡					
Black	1.6	ş		§									
Female	7.7	3.7‡	1973-1977	5.0	1977-1980	-5.6	1980-1998	2.7‡					
White	7.8	3.9‡	1973-1988	1.1‡	1988-1998	3.6‡		-					
Black	4.2	2.8‡	1973-1988	-1.1	1988-1998	4.1‡							
Small intestine	1.3	0.9	1973-1998	2.6‡									
Male	1.6	-0.8	1973–1998	2.0‡ 2.7‡									
White	1.5	-1.2	1973–1998	$2.7 \pm 2.4 \pm$									
Black	2.7	-1.2	1975-1996										
Female	2.7		1973-1998	§									
		2.8‡		2.4‡									
White	1.1	1.8‡	1973-1998	2.2‡									
Black	1.8	ş		§									
Vulva	1.7	2.4‡	1973-1998	0.6‡									
White	1.8	2.5‡	1973-1998	0.7‡									
Black	1.6	2.3		§									
Peritoneum, omentum, and mesentery	0.4	11.2‡	1973–1988	2.3‡	1988–1998	11.0‡							
Male	0.2	-3.4		8									
White	0.2	ş		\$ \$ \$									
Black	8	ş		8									
Female	0.6	15.1‡		8									
White	0.7	14.9‡		ş									
Black	0.4	§		ş									

\*Incidence rates and trends for 1992–1998 are based on Surveillance, Epidemiology, and End Results (SEER) registries covering 14% of the U.S. population. Rates are per 100 000. Joinpoint analysis of trends for 1973–1998 allowed for up to three joinpoints and are based on data from SEER registries covering 10% of the U.S. population.

 $\dagger APC =$  annual percent change; IBD = intrahepatic bile duct.

 $\pm$ APC is statistically significantly different from zero (two-sided *P*<.05).

§No trend analysis was performed when there were too few cases in any year.

Breast (7.8% of 1998 deaths; 16.3% of 1998 cases). During the period from 1973 through 1998, female breast cancer incidence rates increased by more than 40%, from 82.6 in 1973 to 118.1 in 1998. Rates increased differentially by age and race. For white women, breast cancer incidence rates increased by 3.9% per year between 1980 and 1987 (Table 1; Fig. 2) and by 0.4% per year between 1987 and 1998. Breast cancer incidence rates for black women were lower than for white women; however, the increase for black women was similar to that for white women from 1979 through 1986 and larger for black women from 1986 through 1998 (Fig. 2; Table 1). Among white women and black women, trends in incidence rates varied by age (Fig. 3). Since the late 1980s, the increase in breast cancer incidence rates in white women was seen primarily in women 50-74 years old. Among black women 50 years old or older, incidence rates increased from 1973 through 1998, although it was more difficult to distinguish changes in the age-specific trends because of variability in annual rates resulting from small numbers of cases. The increases in incidence rates were limited

to early-stage breast cancer [American Joint Committee on Cancer stages I and II (14)] in both black women and white women, as well as *in situ* disease (data not shown). Increases from 1994 through 1998 in the incidence of stage II lymph node-positive breast cancer among white women 50–64 years old were seen, in contrast to the decline observed from 1984 through 1994. No increases in breast cancer incidence were apparent in either black or white women less than 50 years old. During 1992 through 1998, incidence rates increased in API women, while trends for AI/AN and Hispanic women were difficult to interpret because of small numbers of cases (data not shown).

Death rates from breast cancer decreased by 1.6% annually for all races combined from 1989 through 1995; the decrease then accelerated to a decline of 3.4% annually from 1995 through 1998 (Table 2). The breast cancer death rates declined among white women (Fig. 2), and the decrease was proportionately greater in women younger than 50 years of age than in older women (Fig. 4). For black women, the most recent death rates did not continue to increase, in contrast to the 1.3% annual

 Table 2. Cancer death rates and trends for 1992 through 1998 and joinpoint analyses for 1973 through 1998 for the most common cancers and the 12 cancers with recent increasing rates by site, sex, and race\*

			Joinpoint analyses (1973–1998)										
Site	Average annual rate	APC†	Trend 1		Trend 2		Trend 3		Trend 4				
	(1992–1998)	(1992–1998)	Range of years	APC†	Range of years	APC†	Range of years	APC†	Range of years	APC†			
All sites	167.8	-1.1‡	1973–1985	0.5‡	1985–1991	0.2‡	1991–1994	-0.5	1994–1998	-1.4‡			
Male	209.0	-1.6‡	1973-1980	0.7‡	1980-1990	0.2‡	1990-1994	-0.8‡	1994-1998	-1.9‡			
White	203.2	-1.5‡	1973-1979	0.7‡	1979-1990	0.2‡	1990-1994	-0.7‡	1994–1998	-1.8‡			
Black	297.7	-2.0‡	1973-1983	1.7‡	1983-1990	0.7‡	1990-1994	-0.9	1994–1998	-2.5‡			
Female	139.4	-0.8‡	1973-1990	0.5‡	1990-1995	-0.2	1995-1998	-1.3‡					
White	138.0	-0.8‡	1973-1990	0.5‡	1990-1995	-0.2	1995-1998	-1.4‡					
Black	166.6	-0.6‡	1973–1975	-1.2	1975–1991	0.9‡	1991–1998	-0.6‡					
Female breast White	24.7 24.3	-2.4‡ -2.7‡	1973–1979 1973–1990	-0.3	1979–1989 1990–1995	0.5	1989-1995	-1.6‡	1995–1998	-3.4‡			
Black	24.5 31.0	-2.7+ -0.6	1973–1990	0.2‡ 1.3‡	1990–1993	-2.0 -0.6	1995–1998	-3.6‡					
Prostate	24.5	-3.5‡	1973–1987	0.8‡	1987-1991	2.8‡	1991–1994	-1.2	1994–1998	-4.5‡			
White	24.5	-3.7‡	1973–1987	0.84 0.72	1987–1991	2.8+	1991–1994	-1.2 -1.4	1994–1998	-4.7‡			
Black	53.1	-2.3	1973–1988	1.7‡	1988–1993	2.6‡	1993–1998	-3.0‡	1774-1776				
Lung	49.1	-0.8‡	1973-1980	2.9‡	1980-1990	1.7‡	1990–1993	-0.2	1993-1998	-0.8‡			
Male	69.3	-1.9‡	1973–1980	2.94	1980–1990	$0.5^{+}$	1990–1993	-0.2 -1.8‡	1993-1998	-0.84			
White	67.8	-1.9	1973–1980	1.8	1980–1990	$0.5^{+}_{-}$	1990–1998	-1.7					
Black	96.2	-2.5	1973–1980	2.9‡	1980–1990	1.1‡	1990–1998	-2.2					
Female	90.2 34.0	-2.5+	1973–1982	2.94 6.1‡	1982–1990	1.1÷ 3.7‡	1990–1998	-2.2+ 0.9‡					
White	34.0		1973–1983		1983–1991 1982–1989				1993-1998	0.6+			
Black	33.6	0.8‡ 0.9‡	1973–1982	6.3‡ 6.6‡	1982–1989	4.1‡ 4.1‡	1989–1993 1990–1998	2.4‡ 1.1‡	1995-1998	0.6‡			
							1770-1776	1.1+					
Colon-rectum	17.1	-1.8‡	1973-1984	-0.6‡	1984-1998	-1.8‡	1007 1000	2.01					
Male	20.9	-2.1‡	1973–1979	0.4	1979–1987	-0.6‡	1987–1998	-2.0‡					
White	20.6	-2.3‡	1973-1978	0.4	1978–1986	-0.6‡	1986–1998	-2.1‡					
Black	27.3	-0.9‡	1973-1990	1.2‡	1990–1998	-0.9‡							
Female	14.3	-1.7‡	1973–1984	-1.1‡	1984–1998	-1.9‡							
White	13.9	-1.9‡	1973-1984	-1.2‡	1984–1998	-2.1‡							
Black	19.6	-0.6	1973–1985	0.5‡	1985–1998	-0.6‡							
Non-Hodgkin's lymphoma	6.8	1.1‡	1973–1977	-0.2	1977–1995	2.2‡	1995–1998	-0.2					
Male	8.4	1.1‡	1973–1977	-0.9	1977–1991	2.5‡	1991–1998	1.2‡					
White	8.7	1.1‡	1973–1977	-0.5	1977–1994	2.4‡	1994–1998	0.3					
Black	6.3	2.0	1973–1976	-3.7	1976-1998	2.5‡							
Female	5.5	1.0‡	1973–1975	-1.8	1975–1995	2.0‡	1995–1998	-0.1					
White Black	5.8 3.8	0.9 2.0‡	1973–1975 1973–1998	-1.9 2.4‡	1975–1995	2.0‡	1995–1998	-0.3					
Liver and IBD <sup>†</sup>	3.5	2.9‡	1973-1978	-1.4‡	1978–1987	1.7‡	1987-1995	4.0‡	1995–1998	2.1‡			
Male	5.1	3.0‡	1973–1978	-0.9	1978-1986	2.0‡	1986-1998	3.6‡					
White	4.6	3.0‡	1973–1977	-1.7	1977–1985	1.6‡	1985–1998	3.6‡					
Black	7.5	2.8‡	1973–1988	0.5	1988-1998	2.8‡							
Female	2.3	2.2‡	1973–1977	-2.7‡	1977–1988	1.2‡	1988-1995	4.2‡	1995–1998	0.3			
White	2.1	2.2‡	1973-1978	-2.1‡	1978–1988	1.1‡	1988–1995	4.0‡	1995–1998	0.5			
Black	3.0	1.8	1973–1998	1.6‡									
Esophagus	3.6	0.5‡	1973-1998	0.7	1075 1002	0.44	1082 1004	114	1004 1009	0.2			
Male White	6.3 5.8	0.5‡	1973–1975 1973–1982	2.9	1975-1983	0.6‡	1983–1994	1.1‡	1994–1998	0.2			
		1.6‡		0.6‡	1982-1998	1.9‡	1000 1000	2.04					
Black	12.4	-4.3‡	1973-1979	2.7‡	1979-1989	-0.6	1989–1998	-3.8‡					
Female	1.5	0.0	1973-1980	1.0‡	1980–1998	-0.2‡							
White Black	1.3 3.3	0.9 -3.4‡	1973–1998 1973–1984	0.3‡ 1.1‡	1984–1998	-2.1‡							
Melanoma	2.2	0.4	1973–1977	4.1‡	1977-1989	1.3‡	1989–1998	0.2					
Male	3.2	0.4	1973–1977	2.4‡	1987–1989	0.7‡	1909-1990	0.2					
White	3.2 3.6	$1.0^{+}$	1973–1987	2.4+ 2.6‡	1987–1998	0.74 0.84							
Black	0.4	-2.8	1973–1987	-1.2	1/0/-1990	0.04							
Female	1.5	-0.3	1973–1998	$-1.2_{+}$ 3.1‡	1979–1998	0.0							
White	1.5	-0.3	1973–1979	3.14	1979–1998	0.0							
Black	0.4	2.2	1973–1979	0.3	1717 1770	0.1							
Soft tissue (including heart)	1.3	1.0	1973–1976	-3.9	1976–1979	12.7‡	1979–1998	1.3‡					
Male	1.5	1.0	1973–1976	-5.4‡	1976–1979	12.7#	1979–1998	1.1‡					
White	1.4	0.7	1973–1976	-5.6‡	1976–1979	12.8‡	1979–1998	1.1‡					
Black	1.4	3.7‡	1973–1970	-5.0‡ 5.4‡	1982–1998	0.8	1717 1770	1.1+					
Female	1.4	0.8	1973–1982	-2.2	1976–1979	12.6	1979–1998	1.5‡					
White	1.2	1.3‡	1973–1976	-2.2 -2.7	1976–1979	12.0	1979–1998	1.4‡					
Black	1.7	-2.1	1973–1994	3.6‡	1994–1998	-3.4	1717 1770	1.74					
Ditter	1./	-2.1	1713-1774	5.04	177-1770	·J.+							

(Table continues)

 Table 2 (continued). Cancer death rates and trends for 1992 through 1998 and joinpoint analyses for 1973 through 1998 for the most common cancers and the 12 cancers with recent increasing rates by site, sex, and race\*

			Joinpoint analyses (1973–1998)									
	Average annual rate	APC†	Trend 1		Trend 2		Trend 3		Trend 4			
Site	(1992-1998)	(1992–1998)	Range of years	APC†	Range of years	APC†	Range of years	APC†	Range of years	APC†		
Thyroid	0.3	-0.2	1973–1985	-2.4‡	1985-1998	0.1						
Male	0.3	1.1	1973-1984	-1.5‡	1984-1998	0.6						
White	0.3	1.2	1973-1977	4.2	1977-1983	-3.6	1983-1998	0.8‡				
Black	0.2	§	1973-1998	-1.4‡								
Female	0.4	-0.8	1973-1988	-2.6‡	1988-1998	0.3						
White	0.3	-0.9	1973-1988	-2.8	1988-1998	0.3						
Black	0.4	0.9	1973-1998	-1.3‡								
Small intestine	0.3	-0.2	1973-1998	0.5‡								
Male	0.4	0.7	1973-1998	0.7‡								
White	0.4	1.0	1973-1998	0.6‡								
Black	0.6	-2.0	1973-1998	1.5‡								
Female	0.3	-1.2‡	1973-1998	0.2‡								
White	0.3	-1.8‡	1973-1998	0.1								
Black	0.4	2.8	1973-1998	1.3‡								
Vulva	0.3	-1.2	1973-1984	-2.4‡	1984-1998	0.3						
White	0.3	-0.8	1973-1987	-2.0 <sup>‡</sup>	1987-1998	1.3‡						
Black	0.3	-5.8	1973-1979	-8.8‡	1979–1983	10.1	1983-1998	-3.6‡				
Peritoneum, omentum, and mesentery	0.1	8.2‡	1973–1977	-0.5	1977–1980	-16.5	1980–1988	0.6	1988–1998	7.3‡		
Male	0.1	-3.8	1973-1998	-2.2‡								
White	0.1	-4.0	1973-1998	$-2.0^{+}$								
Black	0.1	§		ş								
Female	0.2	13.0‡	1973-1977	-0.6	1977-1980	-18.9	1980-1988	0.9	1988-1998	12.0‡		
White	0.2	13.5‡	1973–1977	-0.4	1977-1980	-19.8	1980-1988	1.8	1988-1998	12.2‡		
Black	0.1	§		ş								

\*Rates are per 100 000 persons and are age-adjusted to the 1970 U.S. standard million population. Joinpoint analysis of trends allowed for up to three joinpoints. Death data are from the National Vital Statistics System of the National Center for Health Statistics; the data cover the entire U.S. population.

 $\dagger APC =$  annual percent change; IBD = intrahepatic bile duct.

‡APC is statistically significantly different from zero (two-sided P<.05).

§No trend analysis was performed when there were too few cases in any year.

increase between 1973 and 1991 (Fig. 2). Among black women, age-specific declines in death rates occurred among women younger than 75 years of age and were statistically significant only among women younger than 50 years of age.

Breast cancer was the most common cancer diagnosed among women in all five racial and ethnic populations studied (Fig. 5). It was the leading cause of cancer death among Hispanic women and was second to lung cancer among all other racial and ethnic female groups (Fig. 5). Across these populations, the breast cancer death rate was highest among black women. State-specific breast cancer incidence rates varied among white women (from 95.8 in West Virginia to 130.8 in Hawaii) and among black women (from 71.2 in New Mexico to 120.0 in the Atlanta metropolitan area) (Table 3). Breast cancer death rates for white women ranged from 20.1 in Colorado to 27.1 in New Jersey, while rates for black women were higher, ranging from 23.2 in Nevada to 38.9 in Washington, DC (Table 4).

**Prostate (5.9% of deaths; 14.8% of cases).** After a 2.9% annual increase in prostate cancer since 1973, a steeper increase began in 1988 with the introduction of serologic screening for prostate-specific antigen (PSA). Subsequently, incidence rates declined by 5.7% per year in white males from 1992 through 1998 (Table 1). Analysis of long-term trends suggested that the recent decline was not monotonic; there was an 11.3% annual decrease from 1992 through 1995 and a leveling of rates between 1995 and 1998. In black males, incidence declined by 4.0% per year throughout the 7-year period. Among the five

racial and ethnic populations, the incidence rate was highest among black males and lowest among AI/AN males (Fig. 5).

Death rates from prostate cancer have varied over time (Table 2). Among white men, prostate cancer death rates increased by 0.7% per year from 1973 through 1987, climbed by 2.9% per year between 1987 and 1991, stabilized between 1991 and 1994, and then declined by 4.7% per year through 1998. Among black men, death rates increased by 1.7% per year from 1973 to 1988 and 2.6% per year from 1988 to 1993, then declined by 3.0% per year through 1998 (Table 2).

Prostate cancer incidence and mortality varied widely by race/ethnicity and geographic region (Tables 3 and 4). Prostate cancer ranked first in incidence and second to lung cancer as the most common cause of cancer death among men across all five racial and ethnic populations (Fig. 5). The incidence rates ranged from 101.0 among white males in Kentucky to 262.6 among black males in the Atlanta metropolitan area (Table 3). For mortality, the U.S. rate was 21.7 in white males, ranging from 18.7 in Alaska to 25.9 in Wyoming (Table 4). Many states with the highest rates in white males clustered in the north central part of the United States (Wyoming, Wisconsin, Minnesota, Montana, Idaho, North Dakota, and South Dakota). Among black men, the death rates ranged from a low of 34.1 in Nevada to 64.2 in Delaware (Table 4). Most of the states with the highest death rates in black men were in the southeastern part of the United States (Alabama, Florida, Georgia, Mississippi, Tennessee, Virginia, North Carolina, and South Carolina).

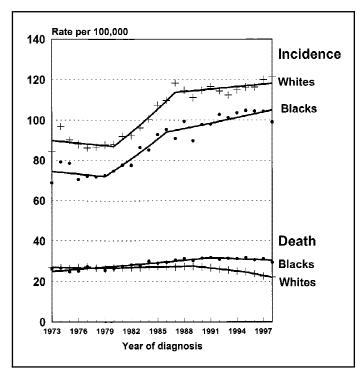


Fig. 2. Female breast cancer incidence and death rates by race, 1973 through 1998. Incidence data are from Surveillance, Epidemiology, and End Results Program areas covering 10% of the U.S. population. Death data are from the National Center for Health Statistics covering the entire U.S. population. Rates are per 100 000 females and are age-adjusted to the 1970 U.S. standard million population.

Lung (28.5% of deaths; 13.2% of cases). Overall, lung cancer incidence decreased 1.6% per year between 1992 and 1998 (Table 1), because of a decline in incidence among men (2.7% per year) and a leveling off of the rates among women. Long-term trends illustrate that women have lagged behind men in lung cancer incidence (Table 1) and mortality (Table 2). Lung cancer incidence rates began to decrease in 1981 in men but continued to increase until 1991 in women. Lung cancer mortality began to decrease in 1990 in men, but it continued to increase to at least 1998 in women. For 1992 through 1998, the death rate decreased by 1.9% per year among men, but it increased by 0.8% per year among women (Table 2). Lung cancer incidence and mortality in women showed a gradual slowing in the rates of increase over the past three decades.

Among men, the death rate from lung cancer for 1992 through 1998 exceeded that from other cancers in all five racial and ethnic populations (Fig. 5). The death rates ranged from 30.5 in Hispanic males and 33.8 in API males to 96.2 in black males. Among women, lung cancer was the leading cause of cancer mortality, except among Hispanics. The death rates ranged from 10.9 in Hispanic females to 34.6 in white females (Fig. 5). Sexand race-specific lung cancer incidence and death rates also varied widely across states (Tables 3 and 4). Incidence rates were lowest among white females in Utah (19.2) and highest among black males in Wisconsin (149.0) (Table 3). Death rates ranged from 14.1 among white females in Utah to 123.4 among black males in Wisconsin (Table 4).

**Colon–rectum (10.5% of deaths; 11.6% of cases).** Colorectal cancer was among the top four cancers in five racial/ethnic populations during 1992 through 1998 (Fig. 5). Colorectal cancer rates ranged from 22.8 in the black population to 10.2 in the

Hispanic population. Incidence rates for white males declined by 1.3% per year from 1992 through 1998 and remained constant for black males, black females, and white females over this time (Table 1). The long-term incidence rates of colorectal cancer in all persons increased until 1985, decreased 1.8% per year through 1995, and then stabilized through 1998 (Table 1). The ascending colon was the only colon subsite with increasing incidence rates during 1992 through 1998, primarily because of a 1.5% annual increase in females (data not shown). Death rates from colorectal cancer decreased from 1992 through 1998 in white males, white females, and black males, whereas rates in black females were stable. In the long-term trends, the decrease in death rates began earlier in women than in men and was larger in white than in black populations (Table 2).

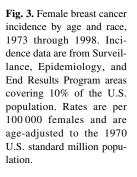
#### **Other Sites With Increasing Trends**

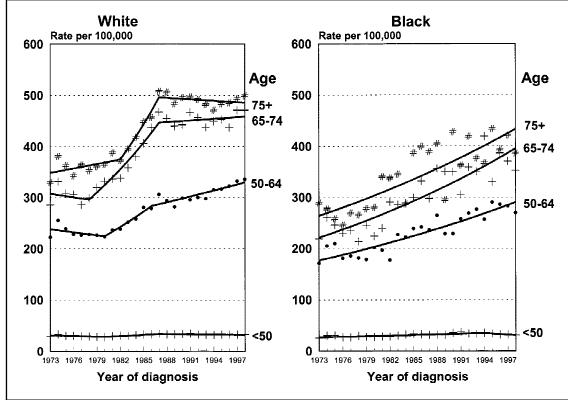
In addition to the recent rise in female breast cancer incidence rates and in the long-term increase in female lung cancer death rates, an increase in either incidence or mortality rates has been observed for 10 other cancer sites. These include NHL, melanoma, and cancer of the liver and intrahepatic bile duct (IBD), as well as relatively rare tumor types, such as cancers of the vulva and of the peritoneum, omentum, and mesentery. The results for each cancer are described in descending order of their contribution to total cancer deaths.

NHL (4.3% of deaths; 4.0% of cases). From 1992 through 1998, NHL incidence rates increased among black females (Table 1), primarily among those younger than 65 years old (data not shown). For all males, incidence rates were stable during the 7-year period (Table 1). The long-term trends in incidence rates increased from 1973 through 1998 by 3.0% per year for black females; incidence rates for white females increased by the same APC through 1988 but then slowed to an increase of 1.3% per year for the next 10 years. Incidence rates in black males increased 4.1% annually from 1973 through 1998; those in white males increased 2.4% per year in the 1970s, accelerated to an increase of 5.0% per year in the 1980s, and slowed to a 1.7% annual increase from 1988 through 1995, when the trend reversed to a decline of 3.3% per year from 1995 through 1998 (Table 1). During 1992 through 1998, death rates increased for white males and black females (Table 2). Analysis of long-term death rates in white males suggested that rates increased from 1977 through 1994 and were stable from 1994 through 1998 (Table 2).

Liver and IBD (2.3% of deaths; 1.2% of cases). From 1992 through 1998, the incidence rates of liver and IBD cancer increased among all males and females, and the magnitude of the rates was consistently higher in males than in females and in black populations than in white populations (Table 1). Death rates during this time also increased in males (all, white, and black) and in females (all and white) (Table 2). The recent increases in both incidence rates and death rates were part of a long-term pattern of increasing rates for this cancer (Tables 1 and 2). The incidence rates of cancer of the liver and IBD were 11.5 among API, 6.1 among Hispanic, 5.6 among AI/AN, 5.1 among blacks, and 3.4 among whites populations (Fig. 5).

**Esophagus (2.2% of deaths; 0.9% of cases).** The overall death rate from cancer of the esophagus increased from 1992 through 1998 because of a 1.6% annual increase in white men; however, rates in black men and in black women decreased over this period (Table 2). Trends in the incidence rates of esophageal



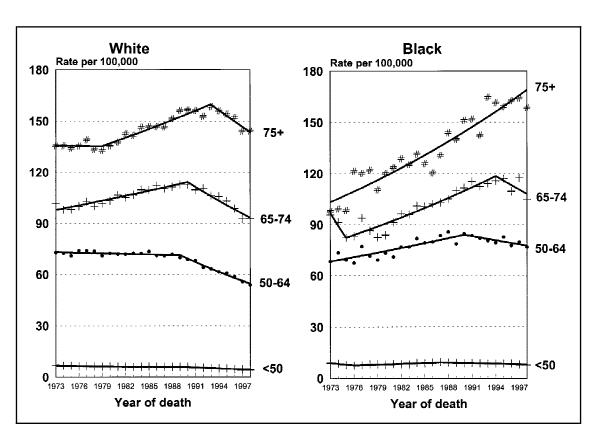


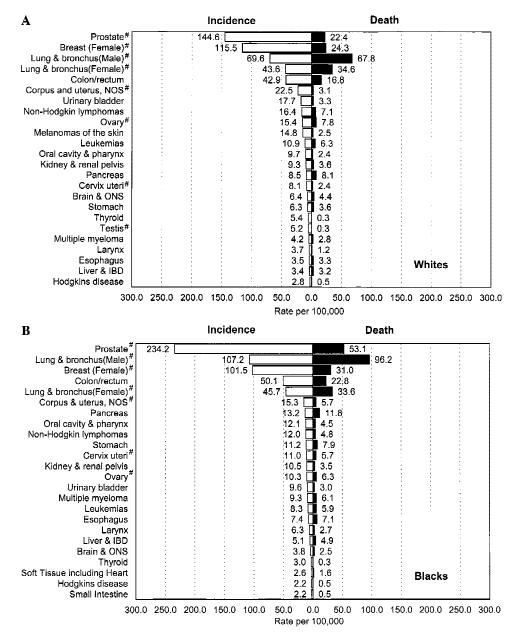
cancer during 1992 through 1998 differed by sex and race, with incidence rates decreasing among black men and remaining stable among other groups (Table 1). The incidence rates and death rates in black persons were nearly twice those in white persons (Fig. 5). The rates among API, AI/AN, and Hispanic

populations were lower than the rate in the white population and were nearly the same among the three groups. Similar racial and ethnic patterns were observed in death rates (Fig. 5).

The histologic distribution of esophageal cancer has also changed, influencing the overall trends of this cancer (data avail-

Fig. 4. Female breast cancer death rates by age and race, 1973 through 1998. Death data are from the National Center for Health Statistics covering the entire U.S. population. Rates are per 100 000 females and are age-adjusted to the 1970 U.S. standard million population.





**Fig. 5.** Cancer incidence and death rates by site and race/ethnicity, 1992 through 1998. Rates are per 100 000 persons and are age-adjusted to the 1970 U.S. standard million population. Incidence data are from Surveillance, Epidemiology, and End Results Program areas covering 14% of the U.S. population. Death data are from the National Center for Health Statistics covering the entire U.S.

population. A) Whites. B) Blacks. C) Asian and Pacific Islanders. D) American Indians and Alaska Natives. E) Hispanics. Hispanic is not mutually exclusive from whites, blacks, American Indians/Alaska Natives, and Asian/Pacific Islanders. <sup>#</sup>Rates are based on sex-specific data. NOS = not otherwise specified; ONS = other nervous system; IBD = intrahepatic bile duct.

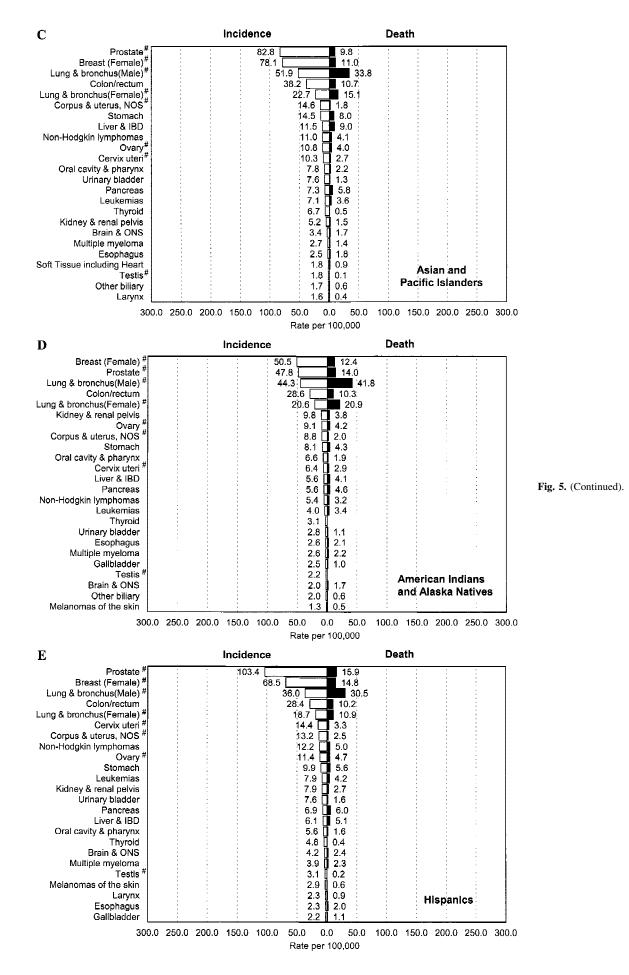
able on the Internet; *see* "Subjects and Methods" section). The incidence rates of squamous cell carcinoma of the upper esophagus have decreased gradually since the late 1970s, although they are higher in black than in white Americans and in men than in women. In contrast, the incidence rates of adenocarcinoma, which occurs in the lower esophagus near the gastroesophageal junction, have increased more steeply than those of any other cancer among men of both races (15).

**Melanoma (1.4% of deaths; 3.5% of cases).** From 1992 through 1998, the incidence rates of melanoma increased an average of 2.7% per year and 2.9% per year for both white males and white females, respectively (Table 1). The death rates during this period increased more slowly for white males (1.0% annu-

ally) and were stable for white females (Table 2). Melanoma is rare among black, API, AI/AN, and Hispanic populations.

Analyses of long-term trends in the incidence rates of melanoma indicated that sharp increases occurred in white males and white females from 1973 until the 1980s that were then followed by slower increases (Table 1). The rates of death from melanoma among white males rose by 2.6% per year from 1973 through 1987 and then by 0.8% annually from 1987 through 1998. Among white females, death rates increased by 3.2% per year in the 1970s and then stabilized through 1998 (Table 2).

Acute myeloid leukemia (AML) (1.3% of deaths; 0.8% of cases). From 1992 through 1998, the incidence of AML increased by 1.8% per year for all males (Table 1), with most of



						Lung				Colon and rectum				
	Female breast		Prostate		Μ	ale	Female		Male		Female			
States and areas	White	Black	White	Black	White	Black	White	Black	White	Black	White	Black		
Arizona	105.7	77.3	113.2	138.9	67.7	78.9	43.0	38.9	45.5	41.7	31.9	31.6		
California	117.2	97.1	124.3	198.7	65.8	93.8	45.6	45.8	48.0	54.8	33.9	42.4		
Greater Bay Area	130.6	92.4	130.6	180.4	62.0	92.9	45.8	45.6	49.8	52.8	35.6	36.8		
Los Angeles	110.0	99.4	124.6	205.3	57.6	93.9	39.0	43.8	46.6	56.5	32.5	44.7		
Colorado	110.5	83.4	131.1	163.4	56.9	70.1	35.1	41.8	44.5	47.7	32.6	36.7		
Connecticut	123.6	94.1	134.6	218.1	74.2	99.4	49.5	38.5	56.8	60.1	40.7	45.3		
Delaware	118.8	106.3	132.4	237.2	92.9	117.5	56.8	63.4	57.7	60.6	40.2	45.3		
Florida	112.1	88.0	124.9	201.5	90.1	101.3	54.6	36.7	58.4	56.9	41.4	45.0		
Georgia: Atlanta metropolitan area	117.1	120.0	147.4	262.6	79.8	117.6	46.6	40.1	45.7	56.7	34.2	49.9		
Hawaii	130.8	72.2	136.4	102.7	68.7		45.9		58.1		34.6			
Idaho	105.8		127.9		61.0		36.5		43.7		32.3			
Illinois	110.9	102.3	119.1	198.7	81.0	121.4	45.5	53.9	55.0	62.6	39.7	48.1		
Iowa	108.8	116.2	125.5	208.7	80.5	113.6	40.2	56.0	59.2	57.3	43.0	49.0		
Kentucky	101.4	103.0	101.0	151.3	120.7	140.3	58.0	67.0	55.5	65.5	40.1	50.3		
Louisiana	101.0	89.7	131.0	184.0	95.2	123.5	47.6	39.0	55.9	55.8	36.3	41.3		
Michigan: Detroit metropolitan area	111.6	102.8	149.2	256.3	85.1	118.5	51.8	52.1	55.7	59.1	36.9	46.3		
Montana	101.8		124.3		69.8		44.0		45.5		33.0			
Nebraska	108.6	103.0	127.4	175.4	72.9	107.9	36.4	57.2	56.0	48.0	38.0	41.1		
New Hampshire	115.1		127.4		76.5		49.3		57.1		40.1			
New Jersey	120.4	96.5	147.3	227.6	77.2	102.1	47.4	45.5	63.1	62.5	43.4	47.0		
New Mexico	104.5	71.2	127.1	121.3	54.2	58.0	32.7	31.3	41.8	42.2	30.0	32.8		
North Carolina	103.8	92.9	112.8	182.7	91.8	106.8	43.5	30.5	47.3	47.2	32.0	39.9		
Pennsylvania	108.2	101.4	129.5	238.2	79.5	122.0	41.7	58.6	60.5	63.4	42.0	44.5		
Rhode Island	117.0	86.8	143.8	167.8	92.2	88.2	53.8	57.6	62.7	45.6	44.0	45.2		
Utah	99.5		146.8	179.9	35.6		19.2		39.2		29.4			
Washington	121.0	99.0	132.8	186.6	73.4	91.3	51.4	44.6	47.8	56.0	35.3	38.9		
Seattle-Puget Sound, WA	127.4	105.3	134.0	200.2	73.4	98.7	53.2	47.4	48.9	59.3	35.2	40.5		
West Virginia	95.8	91.2	107.0	174.4	104.3	94.0	54.8	45.2	53.1	72.3	39.7	48.7		
Wisconsin	110.1	95.9	134.2	231.0	72.0	149.0	42.2	60.7	57.9	64.2	41.4	46.0		
Wyoming	98.7		146.6		61.6		39.8		45.1		33.4			
SEER Program <sup>†</sup>	116.6	102.0	133.4	222.9	67.7	103.7	43.7	45.6	50.5	56.7	35.9	44.6		
NPCR‡	111.4	95.1	125.3	200.4	78.8	108.7	46.6	44.9	53.7	57.2	38.0	43.8		
NAACCR combined§	111.6	96.6	126.7	206.7	78.0	109.3	46.2	45.1	53.6	57.3	37.9	44.3		

\*Rates are per 100 000 and are age-adjusted to the 1970 U.S. standard million population. Rates are suppressed when incidence counts are fewer than 20 in a 5-year period.

†SEER (Surveillance, Epidemiology, and End Results) Program rates are based on data from Greater Bay Area (California), Los Angeles (California), Atlanta metropolitan area (Georgia), Connecticut, Hawaii, Iowa, Detroit metropolitan area (Michigan), New Mexico, Utah, and Seattle–Puget Sound (Washington).

‡NPCR (National Program of Cancer Registries) rates are based on data from Arizona, California, Colorado, Delaware, Florida, Idaho, Illinois, Kentucky, Louisiana, Montana, Nebraska, New Hampshire, New Jersey, North Carolina, Pennsylvania, Rhode Island, Washington, West Virginia, Wisconsin, and Wyoming. \$NAACCR (North American Association of Central Cancer Registries) rates are based on data from 30 states and areas that meet standards of highest quality

for 1994–1998. (SEER metropolitan areas within included states are not duplicated in rates.) These states/areas cover approximately 53% of the U.S. population.

the increase occurring in males who were 65 years old or older (data not shown). This recent pattern was part of a longer trend of increasing rates since the late 1980s in white males. Data were insufficient to assess trends in death rates because of the lack of specificity of leukemias on the death certificate.

**Soft (connective) tissue including heart (0.7% of deaths; 0.6% of cases).** The designation of soft tissue refers to the anatomic site and not to the type of tumor. Although more than 90% of soft-tissue tumors are soft-tissue sarcomas, sarcomas of specific sites are classified to the specific anatomic site and not to soft tissue. The incidence rate of cancers of the soft tissue, including heart, increased 3.3% per year among white females from 1992 through 1998, while that for the other race–sex groups were stable (Table 1). Death rates increased during this period for white females (1.3% annually) and black males (3.7% annually) (Table 2). The long-term incidence trend in all persons increased 0.5% annually from 1973 through 1990 and accelerated to increase by 2.4% per year from 1990 through 1998. In contrast, the long-term mortality trend increased by 12.7% annually in all persons from 1976 through 1979 and then slowed to a 1.3% annual increase through 1998.

Thyroid (0.4% of deaths; 1.5% of cases). The incidence rates for thyroid cancer for 1992 through 1998 were 2.5 times higher in women than in men. The overall incidence rate of thyroid cancer increased 2.7% per year during 1992 through 1998, reflecting annual increases in white (3.9%) and black (2.8%) women (Table 1). The increases occurred among 20- to 54-year-old females of both races and older white females (data not shown). Long-term trends of incidence rates in white women increased 1.1% per year from 1973 through 1988 and then accelerated to increase by 3.6% per year through 1998. Rates for black females were stable through 1988, at which time the rates began to increase by 4.1% per year through 1998. Death rates remained level during the recent 7-year period (Table 2); however, among white males, long-term death rates have increased by 0.8% annually from 1983 through 1998. The lowest incidence rates occurred in black persons (3.0) and in AI/AN populations (3.1), the highest rate was among the API population

Table 4. Average annual cancer death rates for selected cancer sites by sex, race, and state, 1994 through 1998\*

						Lu	ıng		Colon and rectum					
	Female breast		Pro	state	М	ale	Fer	nale	М	ale	Fer	nale		
State	White	Black	White	Black	White	Black	White	Black	White	Black	White	Black		
Alabama	20.6	28.2	21.2	58.3	83.6	90.4	34.3	24.4	17.5	24.7	11.7	16.9		
Alaska	21.3		18.7		56.1		41.0		17.4		10.9			
Arizona	21.8	27.3	21.1	42.3	58.0	81.5	33.0	31.4	17.5	21.9	11.9	17.8		
Arkansas	21.1	29.3	22.1	53.8	91.2	115.4	37.9	28.9	19.1	26.6	13.3	20.2		
California	24.2	30.5	20.7	43.0	53.3	79.3	34.8	37.2	17.6	24.0	12.2	18.1		
Colorado	20.1	28.6	21.2	40.8	46.6	59.4	26.0	29.0	17.2	19.2	11.8	15.3		
Connecticut	24.8	31.7	20.1	49.0	57.2	76.6	34.4	29.5	19.8	22.9	13.3	15.3		
Delaware	26.2	30.1	22.6	64.2	77.6	97.1	42.2	50.3	21.2	24.8	15.8	24.5		
Washington, DC	21.6	38.9	20.4	50.5	44.6	94.8	23.3	41.3	13.9	29.7	12.6	19.7		
Florida	22.8	27.7	19.7	55.2	66.9	86.2	37.2	27.4	18.7	23.9	12.8	18.5		
Georgia	21.9	30.3	22.4	59.8	80.9	95.9	35.8	24.2	17.1	24.5	11.6	18.3		
Hawaii	25.5		23.7		55.9		34.9		19.6		11.8			
Idaho	21.9		24.4		51.4		27.9		17.1		11.8			
Illinois	25.1	33.7	21.6	51.5	66.3	106.7	34.1	44.1	22.0	31.1	14.3	21.8		
Indiana	24.2	33.1	22.7	49.9	77.9	102.9	37.2	45.3	21.3	30.8	14.9	25.9		
Iowa	23.1	29.7	22.9	46.3	64.2	106.4	30.6	35.5	21.3	33.9	15.4	20.7		
Kansas	22.2	33.3	21.0	52.4	64.7	84.0	31.8	31.8	18.7	34.5	12.7	20.1		
Kentucky	23.2	34.0	22.6	49.6	98.8	117.0	43.8	53.0	22.6	29.7	15.3	20.1		
Louisiana	23.4	31.7	22.6	51.5	80.8	113.0	38.0	34.4	21.3	29.6	13.8	19.3		
Maine	23.9	51.7	23.5	51.5	74.0	115.0	41.6	54.4	22.1	27.0	16.0	17.5		
Maryland	25.1	32.2	23.5	56.0	68.4	95.2	39.4	38.6	22.3	30.7	14.9	21.2		
Massachusetts	25.8	28.9	22.2	46.0	64.2	68.7	37.2	33.3	22.3	29.4	14.9	18.0		
Michigan	23.8	28.9 31.4	22.2	40.0	66.1	94.1	37.2	36.8	23.0	29.4	13.5	20.0		
Minnesota	23.9	28.7	21.8	57.2	52.6	94.1	29.7	35.2	18.9	26.9	12.7	20.0		
Mississippi	23.2	30.3	24.5	58.3	32.0 87.7	92.8 106.7	35.1	27.0	18.9	20.9	12.7	18.4		
	20.3	30.3 32.7	21.0	50.6	87.7 77.1	106.7	37.8	44.1	20.5	29.3	14.1	21.3		
Missouri	23.1 22.0	52.7	20.4 24.3	50.0	55.7	100.0	32.7	44.1	20.3 18.7	29.5	14.1	21.5		
Montana		20.7		41.0		00.7		41 7		25 1		21.0		
Nebraska	23.2	29.7	19.9	41.2	60.4	90.7	28.0	41.7	22.1	35.1	14.1	21.0		
Nevada	23.2	23.2	22.9	34.1	69.0	79.0	48.4	36.9	21.5	27.6	13.9	18.9		
New Hampshire	24.7	22.2	22.8	50.5	65.9	00.0	40.3	26.0	21.8	07.1	16.6	20.7		
New Jersey	27.1	32.3	22.0	52.5	61.4	88.0	35.7	36.8	23.9	27.1	15.8	20.7		
New Mexico	22.5	23.3	23.4	10.5	47.1	45.9	26.8	24.6	17.7	22.0	11.5	16.7		
New York	26.8	28.6	21.1	43.5	59.7	66.8	33.4	27.0	22.8	23.8	15.5	16.7		
North Carolina	21.9	32.5	21.9	62.5	80.4	102.0	33.9	26.1	19.1	25.2	12.8	20.6		
North Dakota	22.5		25.4	10.6	54.7		26.1		20.9		12.9			
Ohio	25.2	31.2	22.4	48.6	73.1	101.1	37.0	43.6	22.4	28.8	15.1	20.2		
Oklahoma	23.9	31.5	20.5	47.6	83.3	87.0	38.5	34.8	19.0	32.2	13.5	16.5		
Oregon	23.3	27.2	23.7	53.3	63.0	83.1	39.5	29.2	18.5		12.6	18.0		
Pennsylvania	25.3	32.6	22.3	53.8	66.8	107.0	32.2	47.0	23.0	29.9	15.6	20.8		
Rhode Island	26.3	27.6	23.4	50.1	74.3	81.9	37.9	48.8	24.7		14.7	23.8		
South Carolina	21.3	30.9	22.1	62.0	76.7	91.8	34.0	24.2	19.5	28.3	12.5	18.6		
South Dakota	20.8		24.1		57.1		26.0		20.7		14.5			
Tennessee	23.0	33.1	21.5	55.5	90.5	115.3	36.3	36.9	19.7	34.4	13.2	23.5		
Texas	21.8	30.8	21.7	52.0	68.7	105.8	33.5	34.4	19.0	29.8	12.3	20.2		
Utah	20.8		25.3		28.2	94.1	14.1		14.3		11.2			
Vermont	23.5		23.7		66.1		35.6		22.2		16.5			
Virginia	23.5	33.3	22.4	55.8	73.0	99.6	35.9	34.8	19.1	29.3	13.7	21.6		
Washington	23.1	29.9	21.2	43.8	59.6	78.5	38.8	35.2	17.7	29.5	12.6	14.6		
West Virginia	22.9	25.8	21.8	52.4	87.0	88.5	42.0	33.4	21.1	36.1	15.4	24.6		
Wisconsin	22.9	26.4	24.4	51.6	55.3	123.4	29.6	43.1	20.1	25.4	13.4	18.3		
Wyoming	23.2		25.9		53.2		31.6		18.5		15.6			
United States	23.8	30.9	21.7	52.1	66.6	94.0	34.9	33.9	20.1	27.2	13.7	19.5		

\*Death data are from the National Vital Statistics System of the National Center for Health Statistics; the data cover the entire U.S. population. Rates are per 100 000 persons and are age-adjusted to the 1970 U.S. standard million population. Rates are suppressed when death counts are fewer than 20 in a 5-year period.

(6.7), and white persons had intermediate rates (5.4). All of the racial and ethnic groups had low mortality rates.

1) and for both adenocarcinoma and carcinoid neoplasms (histologic data not shown).

Small intestine (0.2% of deaths; 0.3% of cases). The incidence rates of cancer of the small intestine increased, whereas the death rate decreased, in women from 1992 through 1998 (Tables 1 and 2). Long-term increases in both incidence and death rates were evident among men and women from 1973 through 1998, with the average annual increase in incidence rates more than five times larger than the increase in death rates. Long-term incidence rates increased for men and women (Table

**Vulva (0.1% of deaths; 0.3% of cases).** The incidence rates for cancer of the vulva increased 2.4% per year from 1992 through 1998 (Table 1). The increase occurred predominantly among females younger than age 65 years (data not shown) and involved both squamous cell tumors and tumors of other epithelial origin. Trends in the death rates from vulvar cancer were more variable than incidence trends (Table 2); death rates decreased from the mid-1970s to the mid-1980s and then remained

stable through 1998. Examination of the trend from the mid-1980s to 1998 by race revealed death rates increasing in white women and decreasing in black women.

**Peritoneum, omentum, and mesentery (0.1% of deaths; 0.1% of cases).** The incidence rates for cancers of the peritoneum, omentum, and mesentery increased in white women by 14.9% per year from 1992 through 1998 (Table 1). Data were insufficient to assess incidence trends during 1992 through 1998 in black females, white males, and black males. An increase of similar magnitude was seen in the death rates for white females for this same 7-year period (Table 2). The recent increases in both incidence rates and death rates were part of long-term increases since 1973 that accelerated after 1988 (Tables 1 and 2).

## DISCUSSION

#### **Major Cancer Sites**

**Breast.** Reasons for the recent (1992 through 1998) increase in breast cancer incidence rates may signify increased screening and early detection, since the increase was limited to early-stage disease. The increase was greatest among white women who were 50–64 years old, the population in whom mammography has been used most effectively to detect early-stage tumors (16,17). The increase in stage II lymph node-positive disease that began in 1994 may reflect changes in surgical examination of axillary lymph nodes and pathologic techniques. It is not known whether any of the increase in incidence was due to age and birth cohort changes in reproductive patterns (delayed childbearing and having fewer children) (18–20), recent hormone use (21–23), increases in obesity (24,25), or other, as yet unknown, risk factors.

The death rate from breast cancer continues to decline because of improvements in early detection and treatment. Regular mammography screening, which can detect early-stage breast cancers, reduces cancer mortality by about 16% for women 40– 49 years old and 25%–30% for women 50–69 years old (26). Clinical trials have demonstrated that 5-year treatment with tamoxifen reduces regional recurrence and the occurrence of contralateral tumors by about 50% (27). Tamoxifen is also beneficial as adjuvant therapy for estrogen receptor-positive tumors (28). Adjuvant therapy with polychemotherapy improves survival, particularly if it includes anthracycline-containing regimens (28). Clinical trials (29–32) have also shown that adjuvant radiotherapy reduces local recurrence and improves survival among breast cancer patients with four or more axillary lymph nodes involved.

**Prostate.** Following large increases in prostate cancer incidence due to the introduction of PSA screening in the late 1980s, the incidence trend has been stable from 1995 through 1998, while death rates have continued to decline for both white males and black males. Much of the geographic variation in prostate cancer incidence rates reflects differences in PSA screening (33,34), with regions of high PSA screening penetration having higher incidence rates, often because of the discovery of clinically insignificant tumors. The correlation between prostate cancer incidence and death rates across states is low (r = .32; P = .11, not statistically significant) in white men, illustrating the probable influence that PSA screening has on the magnitude of the prostate cancer incidence rate (34–36).

The impact of PSA screening on mortality is not yet known because other changes have occurred concurrently, such as the shift to earlier stages of disease at diagnosis and the concomitant revised recommendations to treat early-stage disease more aggressively, including with both hormone therapy and surgery or radiation therapy. Clinical trials conducted during the past 10 years have demonstrated the efficacy of hormones in extending disease-free survival, despite the competing risks for mortality from other diseases in the older aged population of men with prostate cancer (37-40).

**Lung.** The decrease in lung cancer incidence and death rates in men and the slowing of the increase in women reflect reductions in tobacco smoking since the 1960s (41,42). During the period from 1991 through 1998, lung cancer death rates in women increased less rapidly than during earlier periods. Trends in incidence and death rates from lung cancer in women have followed the same patterns observed among men: Rates declined first among young women, remained level in women aged 60–69 years, and increased among older women (2). These trends follow the onset of regular cigarette smoking (and later, lung cancer) at younger ages, with progression of the lung cancer epidemic as birth cohorts age (43).

**Colon–rectum.** Following a long-term decline that began in the mid-1980s, incidence rates of colon–rectum cancer were essentially stable from 1995 through 1998. Increased use of effective early-detection methods, such as fecal occult blood test, sigmoidoscopy, colonoscopy, and barium enema, increases incidence in the short run and subsequently decreases mortality. However, because these tests can detect precancerous polyps and early-stage carcinomas, it is difficult to separate the effects of screening from true changes in the incidence of colorectal cancer. The full benefit of effective screening for colorectal cancer will only be realized if existing screening tools are widely used; currently, they are underutilized (*44*).

Whereas from 1992 through 1998, the incidence rate for all cancers of the colon–rectum were stable, rates for cancer of the ascending colon increased. These tumors are beyond the reach of sigmoidoscopy. A shift toward more colon carcinomas of the ascending and transverse colon has been observed in the United States (45) and internationally (46,47). The change in anatomic distribution of colorectal tumors may indicate improvements in diagnosis of these tumors, combined with increased screening by sigmoidoscopy and removal of adenomatous polyps in the descending colon, rather than a true increase in the incidence of ascending colon cancers (47,48).

Colorectal cancer death rates continued to decline, a trend that began in the mid-1980s. For many years, the standard treatment for colorectal cancer after definitive surgery previously was single-agent 5-fluorouracil (5-FU); however, within the past 10 years, survival has improved with the combination of 5-FU and leucovorin (49). Detection of earlier stages of disease and more effective treatments have led to the decline in death rates.

#### **Other Sites With Increasing Trends**

**NHL.** The etiology of most NHL cases is unknown. Recent analyses (50) describe the biologic and clinical heterogeneity within the NHLs according to histology, suggesting that future epidemiologic investigations focus on NHL risks and causal factors according to subtype. Suspected risk factors include chronic antigenic stimulation of the immune system and immunosuppression, primary or acquired immunodeficiency diseases, and immunosuppression following transplantation. In the 1980s, increases in NHL incidence and mortality in males were asso-

ciated with the AIDS epidemic. Individuals occupationally exposed to chemicals, and perhaps phenoxy herbicides, also appear to be at increased risk (51). Recent increases in incidence and mortality among women cannot be explained.

Liver and IBD. While liver cancer ranks as the fifth most common cancer in the world (52), it is not a common tumor within the United States. Chronic infection with hepatitis B virus (HBV) increases the risk of developing liver cancer; this relationship is so strong that it has been determined to be causal. Liver cancer has one of the widest international variations in incidence of any cancer, and this variation correlates with geographic differences in the prevalence of chronic HBV infection (53). For example, immigrants to the United States from Asia have a high prevalence of chronic HBV infection and incidence of liver cancer (53). Hepatitis C virus (HCV) infection is reputed to be the major cause of liver cancer in areas with low prevalence of chronic HBV infection (53). Some proportion of the increase in liver cancer may also be attributed to HCV infection. Other risk factors for liver cancer include cirrhosis (a consequence of chronic HBV and HCV infections), chronic alcoholism, smoking, aflatoxin exposure, occupational vinyl chloride exposure (causing angiosarcoma of the liver), and oral contraceptive use (53).

**Esophagus.** The major risk factors for squamous cell carcinoma of the esophagus in the United States are tobacco and alcohol consumption, especially in combination (54). Risk factors for adenocarcinoma of the esophagus (55), usually preceded by the precursor lesion Barrett's esophagus (56), are obesity (15), gastroesophageal reflux (57), tobacco smoking (58,59), and perhaps the decreasing prevalence of *Helicobacter pylori* infection (60). Compared with other risk factors, the relative importance of obesity and its increasing prevalence in the U.S. population on the increasing rates of adenocarcinoma of the esophagus is not well understood (61,62). Most esophageal carcinomas are detected when the cancer can no longer be resected, which may account for the average survival of less than 1 year from the time of diagnosis (4).

**Melanoma.** Sunburn at any age is associated with an increased risk of melanoma (63), as is sporadic overexposure to the sun (64). Incidence rates are also higher among persons of higher socioeconomic status, most likely reflecting the association of sun exposure and socioeconomic status (64). A recent analysis (65) shows that, in the 1990s, age-specific incidence rates in women stabilized or declined for tumors at an advanced stage and increased more slowly, compared with rates in the 1970s and 1980s, for tumors of local stage. Earlier detection of melanoma in younger populations could be contributing to the increasing incidence trends.

AML. AML is the most common leukemia and occurs most frequently in young children and in the elderly (66). Cigarette smoking is associated with an increased risk of many types of leukemia (66,67). Exposure to benzene, an established carcinogen in cigarette smoke, is associated with an increased risk of AML (67), as are occupational and environmental exposures to ionizing radiation. Patients treated for multiple myeloma, various lymphomas, and breast and ovarian cancers have an increased risk of AML as a second malignancy, although no increased risk has been reported in cohort studies of children treated for acute lymphocytic leukemia (66). Other less wellknown risk factors may include hair dye use in both men and women and infection with human herpesvirus 6 (68). The increasing trend in incidence observed in this study could be attributable to a residual cohort effect of cigarette smoking in older men. Changes in coding practice and the clinical ability to identify this specific leukemia may also contribute to some of the increase.

Soft (connective) tissue, including heart. Ninety different morphologic types are included in soft-tissue cancers, with nearly all of them sarcomas. These tumors are rare, and it has been difficult to identify risks for any factors other than common etiologic agents, such as genetic susceptibility, ionizing radiation, occupational exposures to certain chemicals, and immuno-suppression (69). Some sarcomas, particularly those occurring early in life, are strongly influenced by genetic factors. Still others, such as neural sheath sarcomas, fibrosarcomas, and rhabdomyosarcomas, have both genetic and environmental etiologies (69). Exposure to phenoxy herbicides and related chlorophenols used for wood preservation has been associated with an increased risk of soft-tissue sarcomas (70–72). The reasons for incidence increases since 1992 are unknown.

**Small intestine.** Cancers of the small intestine make up approximately 2% of digestive tract cancers in the United States (73). Their rarity is surprising, given that the small intestine contributes 75% of the length and 90% of the absorptive area of the entire gastrointestinal tract and is one of the most rapidly proliferating tissues in the body (74). It is not known why the incidence rates and rates of death from cancers of the small intestine increased from 1973 through 1998. The long-term trends could partly reflect improved diagnosis, because incidence increased much more steeply than mortality and similar proportional increases occurred in adenocarcinomas of intestinal epithelial cells and enteroendocrine cells (also called carcinoid tumors).

**Thyroid.** Cancer of the thyroid is relatively uncommon. Fiveyear relative survival rates are higher than 90%, and death from thyroid cancer is rare. Risk is linked to ionizing radiation (75), to radiation treatment to the head or neck for benign conditions (75) and for Hodgkin's disease in children (76,77), and to radiation exposure among atomic bomb survivors (78). Other possible risk factors include certain benign thyroid diseases (76) and being a Filipino-born person living in the United States (79). Current smokers and alcohol consumers have lower risk than former or never users (80). A recent study (81) suggests that thyroid stimulation during both pregnancy and lactation is associated with a transient increase in the risk of papillary thyroid cancer among younger women.

Long-term survival for thyroid cancer patients is very good; however, it worsens with increasing age at diagnosis. Follicular and papillary carcinomas have the highest incidence in young women but have low mortality; they represent a different disease than anaplastic types or undifferentiated carcinomas, both of which occur among older patients and have high death rates (75). Thus, incidence and mortality statistics may measure these differences. A cause for the recent trend is unknown.

**Vulva.** The majority of vulva cancers are squamous cell in origin and occur in older women and women of low socioeconomic status (82). Epidemiologic study of this rare tumor is limited and has identified few consistent risk factors, although some evidence exists for a possible role of human papillomavirus and cigarette smoking. Although *in situ* disease was not the focus of the present report, increasing trends of *in situ* tumors and level rates of invasive disease for the 1970s and 1980s have

been noted by other investigators (82). In the present study, rates of *in situ* squamous cell tumors in white women increased from 1973 through 1996 and then leveled off. Among black women, rates of *in situ* squamous cell tumors increased by 7.9% per year from 1973 through 1998. Possible explanations for the observed trends include increased use of biopsy for suspicious vulva lesions, temporal changes in sexual behaviors, or a reporting artifact (83).

**Peritoneum, omentum, and mesentery.** Primary tumors of the peritoneum, omentum, and mesentery are rare, since cancers in these sites result largely from metastases from other sites (84,85). In females, accurate classification of a peritoneal tumor requires good visualization during surgery to distinguish tumors of the ovary from those of the peritoneum (86). Misclassification of these tumors has been reported (87-89). Thus, the increasing incidence trend in females could reflect better classification practices due to a gradual realization and greater understanding of the characterization of primary peritoneal serous tumors and better identification of borderline ovarian and primary mucinous tumors compared with metastatic tumors (86).

#### Limitations

Some of the statistically significant findings in analyses that test multiple comparisons may be due to chance alone. The analyses in this report examined site-specific trends among all nine combinations of race (all, black, and white) and sex (all, females, and males) and for both incidence (>100 cancers) and mortality (>70 causes of cancer death) and thus could include results that are statistically spurious.

Limitations in the trend statistics must also be considered. The APC assumes that rates increase or decrease at a constant rate over a time interval, an assumption that may not be true. Moreover, results of the JPA are not necessarily comparable to those in last year's annual report (3) because the addition of the 1998 data point may have altered the joinpoints, the direction or magnitude of the APC, or both. The JPA complicates comparisons across different groups that have different joinpoints and years of inflection. Moreover, the JPA is less able to discern changes in trends in the black population because rates are based on fewer cases or deaths than for the white population. However, the JPA is a flexible, and perhaps more accurate, approach than APC in identifying the years in which significant changes in trends occurred.

Another limitation is that, although the NAACCR combined rate covers more than 50% of the U.S. population, it may not reflect the United States as a whole. Progress in achieving nationwide coverage of the U.S. population has been substantial in the last 5 years. It is anticipated that at least 90% of the U.S. population will be covered within the next 5 years by population-based registries meeting quality standards.

Finally, our assessment of long-term cancer trends is limited to white and black populations only. While changing trends for other race and ethnic groups may exist, annual population counts before 1990 for these other populations are not available; thus, annual rates could not be calculated for these years. Furthermore, cancer incidence and death rates for some racial and ethnic populations may be limited by problems in ascertaining race and by the misreporting of race and ethnicity on the basic records from which information is collected on cancer incidence, deaths, and the population at risk (90-93). Studies (91,93) suggest that reporting race for the white and black population is generally reliable, but biases are more serious for some smaller populations, particularly American Indians. These biases can affect trends and comparisons among groups.

#### Strategies for the Future

Although it is encouraging that overall cancer incidence and death rates continue to decline in the United States, measures to sustain this progress must address the entire spectrum of prevention, early detection, and improved treatment and quality of life and must be aimed at reducing mortality among all populations. Except for breast and lung cancers in women, the other 10 cancers that increased in incidence or mortality from 1992 through 1998 are relatively uncommon, making up about 13% of the total cancer incidence and mortality in the United States. Prevention is a key strategy for reducing the national cancer burden. In particular, the single most critical determinant of future cancer incidence and mortality will be the ability to reduce tobacco use in all segments of the population. Effective tobacco reduction requires 1) full-scale, well-funded, sustained efforts to stop smoking initiation in young people and 2) application of effective and state-of-the-art smoking-cessation programs that combine behavioral and pharmacologic approaches to increase the quit rate of tobacco-addicted adults (94,95). Tobacco smoking causes an estimated 30% of cancer deaths (96). Both the avoidance and cessation of smoking effectively reduce the risk of many cancers.

Additional prevention opportunities could substantially reduce the burden from other cancers. For example, although melanoma rates in Australia remain the highest in the world, prevention programs in that country have been conducted for more than 20 years and have been effective in reducing melanoma incidence rates (97). The multistrategy programs address not only individual behaviors (use of protective clothing and sunscreen to limit sun exposure) and social norms (desirability of a suntan), but also social policies that increase sun protection and modify the environment. These social policies include increasing shade in public spaces, planting trees and constructing canopies on playgrounds, reducing workplace exposure, avoiding mid-day scheduling of sport activities, and providing inexpensive sunscreen without sales tax. Protective behavior is encouraged by media depictions of popular role models who demonstrate the recommended behaviors.

Childhood immunization against hepatitis B prevents chronic HBV infection and reduces the incidence of liver cancer (98–100). Chronic HBV infection beginning in childhood poses the highest risk of liver cancer. In the United States, most HBV infections occur in adults, yet the small proportion of infections that begin in childhood account for a high proportion of chronic HBV infections. Hepatitis B vaccination has been successfully incorporated into early childhood and adolescent immunizations in the United States. In addition, screening for carriers of  $\alpha$ -fetoprotein has been effective in detecting early-stage liver tumors and in improving survival in an HBV-infected AI/AN population (101). It remains unclear whether screening of other populations would be as effective (102). However, HBV infection is a significant global health problem.

A vaccine to prevent chronic HCV infection is needed, but its development poses major technical challenges due to viral complexity. The illness, mortality, and economic impact associated with HCV infection is projected to be substantial in the next 10–20 years (103). A randomized clinical trial is in progress to

evaluate the impact of ribavirin and interferon on reducing progression to cirrhosis and ultimately to liver cancer in individuals infected with hepatitis C.

A second strategy to reduce the cancer burden is to improve use of currently effective but underutilized cancer screening and to develop more effective screening technologies. For example, among the more common cancers, established procedures for early detection of colorectal cancers are underused in all population groups (44). Routine regular screening by mammography should also be increased, especially in women aged 65 years and older. Evidence from successful intervention projects demonstrates that they can be used to achieve higher rates of routine screening; however, the interventions must be more widely applied. A large, randomized clinical trial is currently testing whether digital mammography improves detection over screenfilm mammography. New colon cancer-detection technologies now in trials include novel fecal tests to detect cancer markers and virtual or computed tomography (CT)-colonography. Studies are in progress to determine the impact of PSA screening on survival and to clarify clinical guidelines for screening and treatment of prostate cancer. Spiral CT scanning is under evaluation as a screening tool for lung cancer. Information on these and on other clinical trials is available on the Internet (http:// cancernet.gov).

Improved early-detection approaches are also needed for less common cancers. Moreover, for many cancers, no effective screening tools are available. For example, early-stage cancers of the ovary are not reliably detected by existing screening tests. Populations at high risk for particular cancers might benefit from screening approaches beyond those recommended for the general population, such as improved approaches to detect adenocarcinoma of the esophagus among persons with Barrett's esophagus. Changes in the anatomic location of certain cancers should be considered in the periodic evaluation of screening practices. The NCI Cancer Genome Anatomy Project has uncovered molecules, proteins, genes, and other biologic substances that may be the earliest warning signs of normal cells turning cancerous. Research priorities are focusing on molecular targets and cancer imaging to identify treatments and earlydetection tools that are more effective.

A third strategy is to continue development of state-of-the-art diagnostic procedures and treatment. For breast cancer, trials are in progress of new preventive agents, such as raloxifene, and new therapeutics, such as the taxanes, her-2 neu, and angiogenesis inhibitors. For prostate cancer, a major challenge is to develop imaging techniques and biomarkers that will distinguish the aggressive tumors requiring treatment from those that are clinically insignificant. Techniques, such as cryosurgery, brachytherapy, and three-dimensional conformal radiotherapy, and new drugs for prostate cancer are under active investigation in clinical trials. For colorectal cancer, clinical studies are testing the use of cyclooxygenase 2 inhibitors for prevention and adjuvant treatment; other new treatments, such as oxaliplatin, monoclonal antibodies, angiogenesis inhibitors, and vaccines, also show promise. Irinotecan has been approved by the U.S. Food and Drug Administration for the treatment of patients with metastatic colorectal cancer (104) and is being evaluated in clinical trials for earlier stage disease. Information on these clinical trials is available on the Internet (http://cancernet.gov).

A fourth strategy is to identify and reduce disparities across diverse populations through dissemination of state-of-the-art

cancer care to all populations in order to increase survival, improve quality of life, and decrease mortality. Disparities in health and health care can result from socioeconomic factors, in conjunction with factors such as race, ethnicity, culture, and geography (urban-rural), among others. Widespread recognition of the existence of disparities and of the complexities involved in understanding their relation to health has led to more resources and programs to improve prevention, early detection, and treatment in all populations. From a clinical perspective, when diverse populations are included in clinical trials, all participants with equivalent stage of disease and state-of-the-art therapy have similar outcomes, regardless of their sociodemographic characteristics. The challenge is to improve access and use of cancer control measures across diverse populations, goals that have existed for a long time but for which new multifocused approaches are needed. Several national efforts have been initiated to broaden access of diverse patient populations to cancer information and clinical trials. Some examples include the Special Populations Networks for Cancer Awareness Research and Training, a network of institutions that will create and implement cancer control, prevention, treatment, research, and training programs in minority and underserved communities; a revamped national clinical trials program designed to facilitate enrolling patients into clinical trials; and a new national coverage decision from Medicare to cover routine care costs for patients enrolled in clinical trials. Major steps are being taken to make clinical trials more broadly accessible to physicians and patients. Training programs have also begun to increase the diversity of scientists in biomedical research and to enhance existing careers.

Coverage of cancer incidence data has expanded dramatically in the United States in the last decade, and completeness, accuracy, and timeliness have improved since 1995 (5,6,105-107). Cancer incidence data are becoming both national (in that they include a greater percentage of the U.S. population) and nationwide (in that local data are increasingly more available for all U.S. locations). A national cancer surveillance system can be more comprehensive, embracing the entire life cycle from birth to death. This surveillance system will be used to address the cancer information needs for healthy populations, newly diagnosed patients, patients receiving treatment, and those living with and dying of the disease. The goal is to use surveillance data in targeting and prioritizing populations so that prevention initiatives, early-detection programs, and research can be effectively focused and so that access to state-of-the-art treatment, quality of life, and palliative care for cancer patients and survivors can be improved.

## REFERENCES

- (1) Wingo PA, Ries LA, Rosenberg HM, Miller DS, Edwards BK. Cancer incidence and mortality, 1973–1995: a report card for the U.S. Cancer 1998;82:1197–207.
- (2) Wingo PA, Ries LA, Giovino GA, Miller DS, Rosenberg HM, Shopland DR, et al. Annual report to the nation on the status of cancer, 1973–1996, with a special section on lung cancer and tobacco smoking. J Natl Cancer Inst 1999;91:675–90.
- (3) Ries LA, Wingo PA, Miller DS, Howe HL, Weir HK, Rosenberg HM, et al. The annual report to the nation on the status of cancer, 1973–1997, with a special section on colorectal cancer. Cancer 2000;88:2398–424.
- (4) Ries L, Eisner M, Kosary C, Hankey B, Miller B, Edwards BK, editors. SEER cancer statistics review, 1973–97. Bethesda (MD): National Cancer Institute; 2000. NIH Publ No. 00–2789.

- (5) Cancer registries: the foundation for comprehensive cancer control. At-A-Glance 2000. Atlanta (GA): USDHHS, Centers for Disease Control and Prevention; 2000.
- (6) Howe HL, Chen VW, Hotes J, Wu XC, Correa C, editors. Cancer in North America, 1994–1998. Vol. I: Incidence. Springfield (IL): North American Association of Central Cancer Registries; April 2001.
- (7) Fulton JP, Howe HL. Evaluating the incidence-mortality ratios in estimating completeness of cancer registration. In: Howe HL, editor. Cancer incidence in North America, 1988–1991. Sacramento (CA): North American Association of Central Cancer Registries; April 1995.
- (8) Howe HL. Conclusions of the work group for high quality criteria for data use. NAACCR Narrative; Winter 2001. p. 8–9.
- (9) Percy C, Van Holten V, Muir C, editors. International Classification of Diseases for Oncology. 2<sup>nd</sup> ed. Geneva (Switzerland): World Health Organization; 1990.
- (10) National Center for Health Statistics. Vital statistics of the United States, 1950–1998. Vol. II: Mortality, parts A and B. Washington (DC): U.S. Public Health Service; 1954–2000.
- (11) U.S. Department of Health Education and Welfare, National Center for Health Statistics. Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death, adapted for use in the United States. 8<sup>th</sup> rev. Washington (DC): U.S. Govt Print Off; Public Health Service Publ No. 1693; 1967.
- (12) World Health Organization. Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death, based on the recommendations of the ninth revision, conference, 1975. Geneva (Switzerland): World Health Organization; 1977.
- (13) Kim HJ, Fay MP, Feuer EJ, Midthune DN. Permutation tests for joinpoint regression with applications to cancer rates. Stat Med 2000;19: 335–51.
- (14) Beahrs OH, Henson DE, Hutter RV, Myers MH. Manual for Staging of Cancer. 3<sup>rd</sup> ed. Philadelphia (PA): Lippincott, American Joint Committee on Cancer; 1988.
- (15) Blot WJ, McLaughlin JK. The changing epidemiology of esophageal cancer. Semin Oncol 1999;26(5 Suppl 15):2–8.
- (16) Ballard-Barbash R, Taplin SH, Yankaskas B, Ernster VL, Rosenberg R, Carney PA, et al. Breast Cancer Surveillance Consortium: a national mammography screening and outcomes database. Am J Radiol 1997;169: 1001–8.
- (17) Chu KC, Tarone RE, Kessler LG, Ries LA, Hankey BF, Miller BA, et al. Recent trends in U.S. breast cancer incidence, survival, and mortality rates. J Natl Cancer Inst 1996;88:1571–9.
- (18) White E. Projected changes in breast cancer incidence due to the trend toward delayed childbearing. Am J Public Health 1987;77:495–7.
- (19) Kelsey JL. Breast cancer epidemiology: summary and future directions. Epidemiol Rev 1993;15:256–63.
- (20) Hulka BS, Stark AT. Breast cancer: cause and prevention. Lancet 1995; 346:883–7.
- (21) Collaborative Group on Hormonal Factors in Breast Cancer. Breast cancer and hormone replacement therapy: collaborative reanalysis of data from 51 epidemiological studies of 52,705 women with breast cancer and 108,411 women without breast cancer. Lancet 1997;350:1047–59.
- (22) Colditz GA. Relationship between estrogen levels, use of hormone replacement therapy, and breast cancer. J Natl Cancer Inst 1998;90: 814–23.
- (23) Collaborative Group on Hormonal Factors in Breast Cancer. Breast cancer and hormonal contraceptives: collaborative reanalysis of individual data on 53,297 women with breast cancer and 100,239 women without breast cancer from 54 epidemiological studies. Lancet 1996;347:1713–27.
- (24) Huang Z, Willett WC, Colditz GA, Hunter DJ, Manson JE, Rosner B, et al. Waist circumference, waist : hip ratio, and risk of breast cancer in the Nurses' Health Study. Am J Epidemiol 1999;150:1316–24.
- (25) Huang Z, Hankinson SE, Colditz GA, Stampfer MJ, Hunter DJ, Manson JE, et al. Dual effects of weight and weight gain on breast cancer risk. JAMA 1997;278:1407–11.
- (26) Kerlikowske K. Efficacy of screening mammography among women aged 40 to 49 years and 50 to 69 years: comparison of relative and absolute benefit. J Natl Cancer Inst Monogr 1997;22:79–86.
- (27) Fisher B, Costantino JP, Wickerham DL, Redmond CK, Kavanah M, Cronin WM, et al. Tamoxifen for prevention of breast cancer: report of the

National Surgical Adjuvant Breast and Bowel Project P-1 study. J Natl Cancer Inst 1998;90:1371–88.

- (28) Early Breast Cancer Trialists' Collaborative Group. Tamoxifen for early breast cancer: an overview of the randomised trials. Lancet 1998;351: 1451–67.
- (29) Early Breast Cancer Trialists' Collaborative Group. Favourable and unfavourable effects on long-term survival of radiotherapy for early breast cancer: an overview of the randomised trials. Lancet 2000;355: 1757–70.
- (30) Ragaz J, Jackson SM, Le N, Plenderleith IH, Spinelli JJ, Basco VE, et al. Adjuvant radiotherapy and chemotherapy in node-positive premenopausal women with breast cancer. N Engl J Med 1997;337:956–62.
- (31) Overgaard M, Hansen PS, Overgaard J, Rose C, Andersson M, Bach F, et al. Postoperative radiotherapy in high-risk premenopausal women with breast cancer who receive adjuvant chemotherapy. Danish Breast Cancer Cooperative Group 82b Trial. N Engl J Med 1997;337:949–55.
- (32) Fowble B, Gray R, Gilchrist K, Goodman RL, Taylor S, Tormey DC. Identification of a subgroup of patients with breast cancer and histologically positive axillary nodes receiving adjuvant chemotherapy who may benefit from postoperative radiotherapy. J Clin Oncol 1988;6:1107–17.
- (33) Feuer EJ, Merrill RM, Hankey BF. Cancer surveillance series: interpreting trends in prostate cancer—part II: Cause of death misclassification and the recent rise and fall in prostate cancer mortality. J Natl Cancer Inst 1999;91:1025–32.
- (34) Hankey BF, Feuer EJ, Clegg LX, Hayes RB, Legler JM, Prorok PC, et al. Cancer surveillance series: interpreting trends in prostate cancer—part I: evidence of the effects of screening in recent prostate cancer incidence, mortality, and survival rates. J Natl Cancer Inst 1999;91:1017–24.
- (35) Legler JM, Feuer EJ, Potosky AL, Merrill RM, Kramer BK. The role of prostate-specific antigen (PSA) testing patterns in the recent prostate cancer incidence decline in the United States. Cancer Causes Control 1998; 9:519–27.
- (36) Etzioni R, Legler JM, Feuer EJ, Merrill RM, Cronin K, Hankey BF. Cancer surveillance series: interpreting trends in prostate cancer—part III: Quantifying the link between population prostate-specific antigen testing and recent declines in prostate cancer mortality. J Natl Cancer Inst 1999; 91:1033–9.
- (37) Pilepich MV, Caplan R, Byhardt RW, Lawton CA, Gallagher MJ, Mesic JB, et al. Phase III trial of androgen suppression using goserelin in unfavorable-prognosis carcinoma of the prostate treated with definitive radiotherapy: report of Radiation Therapy Oncology Group Protocol 85–31. J Clin Oncol 1997;15:1013–21.
- (38) Bolla M, Gonzalez D, Warde P, Dubois JB, Mirimanoff RO, Storme G, et al. Improved survival in patients with locally advanced prostate cancer treated with radiotherapy and goserelin. N Engl J Med 1997;337: 295–300.
- (39) Zagars GK, Johnson DE, von Eschenbach AC, Hussey DH. Adjuvant estrogen following radiation therapy for stage C adenocarcinoma of the prostate: long-term results of a prospective randomized study. Int J Radiat Oncol Biol Phys 1988;14:1085–91.
- (40) Granfors T, Modig H, Damber JE, Tomic R. Combined orchiectomy and external radiotherapy versus radiotherapy alone for nonmetastatic prostate cancer with or without pelvic lymph node involvement: a prospective randomized study. J Urol 1998;159:2030–4.
- (41) Shopland DR. Tobacco use and its contribution to early cancer mortality with a special emphasis on cigarette smoking. Environ Health Perspect 1995;103(Suppl 8):131–42.
- (42) Tolley HD, Crane L, Shipley N. Smoking prevalence and lung cancer death rates. In: Strategies to control tobacco use in the United States a blueprint for public health action in the 1990s. Smoking and tobacco control monograph No. 1. Rockville (MD): U.S. Department of Health and Human Services (DHHS), Public Health Service, National Institutes of Health, National Cancer Institute; 1992: NIH Publ No. 92–3316.
- (43) Burns DM, Lee L, Shen LZ, Gilpin E, Tolley HD, Vaughn J, et al. Cigarette smoking behavior in the United States. In: Changes in cigaretterelated disease risks and their implication for prevention and control. Smoking and tobacco control monograph No. 8. Bethesda (MD): U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute; 1997: NIH Publ No. 97–4213.

- (44) Trends in screening for colorectal cancer—United States, 1997 and 1999. MMWR Morb Mortal Wkly Rep 2001;50:162–6.
- (45) Troisi RJ, Freedman AN, Devesa SS. Incidence of colorectal carcinoma in the U.S.: an update of trends by gender, race, age, subsite, and stage, 1975–1994. Cancer 1999;85:1670–6.
- (46) Thorn M, Bergstrom R, Kressner U, Sparen P, Zack M, Ekbom A. Trends in colorectal cancer incidence in Sweden 1959–93 by gender, localization, time period, and birth cohort. Cancer Causes Control 1998;9:145–52.
- (47) Stewart RJ, Stewart AW, Turnbull PR, Isbister WH. Sex differences in subsite incidence of large-bowel cancer. Dis Colon Rectum 1983;26: 658–60.
- (48) Schub R, Steinheber FU. Rightward shift of colon cancer. A feature of the aging gut. J Clin Gastroenterol 1986;8:630–4.
- (49) O'Connell MJ, Mailliard JA, Kahn MJ, Macdonald JS, Haller DG, Mayer RJ, et al. Controlled trial of fluorouracil and low-dose leucovorin given for 6 months as postoperative adjuvant therapy for colon cancer. J Clin Oncol 1997;15:246–50.
- (50) Groves FD, Linet MS, Travis LB, Devesa SS. Cancer surveillance series: non-Hodgkin's lymphoma incidence by histologic subtype in the United States from 1978 through 1995. J Natl Cancer Inst 2000;92: 1240–51.
- (51) Scherr PA, Mueller NE. Non-Hodgkin's lymphomas. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. 2<sup>nd</sup> ed. New York (NY): Oxford University Press; 1996. p. 920–45.
- (52) Bosch FX, Ribes J. Epidemiology of liver cancer in Europe. Can J Gastroenterol 2000;14:621–30.
- (53) London WT, McGlynn KA. Liver cancer. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. New York (NY): Oxford University Press; 1996. p. 772–93.
- (54) U.S. Department of Health and Human Services. Reducing the health consequences of smoking: 25 years of progress. A report of the Surgeon General. Rockville (MD): U.S. Department of Health and Human Services (DHHS), Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1989: DHHS Publ No. (CDC)89–8411; 1989.
- (55) Devesa SS, Blot WJ, Fraumeni JF Jr. Changing patterns in the incidence of esophageal and gastric carcinoma in the United States. Cancer 1998; 83:2049–53.
- (56) Montesano R, Hainaut P, Hall J. The use of biomarkers to study pathogenesis and mechanisms of cancer: oesophagus and skin cancer as models. In: Toniolo P, Boffetta P, Shuker D, Rothman N, Hulka B, Pearce N, editors. Application of biomarkers in cancer epidemiology. Lyon (France): IARC Scientific Publications; 1997. p. 291–301.
- (57) Chow WH, Finkle WD, McLaughlin JK, Frankl H, Ziel HK, Fraumeni JF Jr. The relation of gastroesophageal reflux disease and its treatment to adenocarcinomas of the esophagus and gastric cardia. JAMA 1995;274: 474–7.
- (58) Gammon M, Schoenberg JB, Ahsan H, Risch H, Vaughan T, Chow WH, et al. Tobacco, alcohol, and socioeconomic status and adenocarcinomas of the esophagus and gastric cardia. J Natl Cancer Inst 1997;89: 1277–84.
- (59) Lagergren J, Bergstrom R, Lindgren A, Nyren O. Symptomatic gastroesophageal reflux as a risk factor for esophageal adenocarcinoma. N Engl J Med 1999;340:825–31.
- (60) Chow WH, Blaser MJ, Blot WJ, Gammon MD, Vaughan TL, Risch HA, et al. An inverse relation between cagA+ strains of *Helicobacter pylori* infection and risk of esophageal and gastric cardia adenocarcinoma. Cancer Res 1998;58:588–90.
- (61) Greenlee RT, Hill-Harmon MB, Murray T, Thun M. Cancer statistics, 2001. CA Cancer J Clin 2001;51:15–36.
- (62) Mokdad A, Serdula M, Dietz WH, Bowman B, Marks JS, Koplan J. The spread of the obesity epidemic in the United States, 1991–1998. JAMA 1999;282:1519–22.
- (63) Elwood JM, Jopson J. Melanoma and sun exposure: an overview of published studies. Int J Cancer 1997;73:198–203.
- (64) English DR, Armstrong BK, Kricker A, Fleming C. Sunlight and cancer. Cancer Causes Control 1997;8:271–83.
- (65) Jemal A, Devesa SS, Hartge P, Tucker MA. Cancer surveillance series: recent trends in cutaneous melanoma incidence among whites in the United States. J Natl Cancer Inst 2001;93:678–83.

- (66) Linet MS, Cartwright RA. The leukemias. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. 2<sup>nd</sup> ed. New York (NY): Oxford University Press, 1996; p. 841–92.
- (67) Korte JE, Hertz-Picciotto I, Schulz MR, Ball LM, Duell EJ. The contribution of benzene to smoking-induced leukemia. Environ Health Perspect 2000;108:333–9.
- (68) Gentile G, Mele A, Ragona G, Faggioni A, Zompetta C, Tosti ME, et al. Human herpes virus-6 seroprevalence and leukaemias: a case–control study. GIMEMA (Gruppo Italiano Malattie Ematologiche dell' Adulto). Br J Cancer 1999;80:1103–6.
- (69) Mack TM. Sarcomas and other malignancies of soft tissue, retroperitoneum, peritoneum, pleura, heart, mediastinum, and spleen. Cancer 1995;75(1 Suppl):211–44.
- (70) Wingren G, Fredrikson M, Brage HN, Nordenskjold B, Axelson O. Soft tissue sarcoma and occupational exposures. Cancer 1990;66: 806–11.
- (71) Eriksson M, Hardell L, Adami HO. Exposure to dioxins as a risk factor for soft tissue sarcoma: a population-based case–control study. J Natl Cancer Inst 1990;82:486–90.
- (72) Lynge E. Cancer in phenoxy herbicide manufacturing workers in Denmark, 1947–87—an update. Cancer Causes Control 1993;4:261–72.
- (73) Barquist E, Zinner M. Neoplasms of the small intestine, vermiform appendix, and peritoneum. In: Bast RC, Kufe DW, Pollack RE, et al, editors. Cancer medicine. Hamilton (ON): B. C. Decker; 2000. p. 1465–71.
- (74) Schottenfeld D, Islam SS. Cancers of the small intestine. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. New York (NY): Oxford University Press; 1996. p. 806–12.
- (75) Ron E. Thyroid cancer. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. 2<sup>nd</sup> ed. New York (NY): Oxford University Press; 1996. p. 1000–21.
- (76) Mack WJ, Preston-Martin S, Bernstein L, Qian D, Xiang M. Reproductive and hormonal risk factors for thyroid cancer in Los Angeles County females. Cancer Epidemiol Biomarkers Prev 1999;8:991–7.
- (77) Sklar C, Whitton J, Mertens A, Stovall M, Green D, Marina N, et al. Abnormalities of the thyroid in survivors of Hodgkin's disease: data from the Childhood Cancer Survivor Study. J Clin Endocrinol Metab 2000;85: 3227–32.
- (78) Thompson DE, Mabuchi K, Ron E, Soda M, Tokunaga M, Ochikubo S, et al. Cancer incidence in atomic bomb survivors. Part II: Solid tumors, 1958–1987. Radiat Res 1994;137(2 Suppl):S17–67.
- (79) Rossing MA, Schwartz SM, Weiss NS. Thyroid cancer incidence in Asian migrants to the United States and their descendants. Cancer Causes Control 1995;6:439–44.
- (80) Rossing MA, Cushing KL, Voigt LF, Wicklund KG, Daling JR. Risk of papillary thyroid cancer in women in relation to smoking and alcohol consumption. Epidemiology 2000;11:49–54.
- (81) Rossing MA, Voigt LF, Wicklund KG, Daling JR. Reproductive factors and risk of papillary thyroid cancer in women. Am J Epidemiol 2000; 151:765–72.
- (82) Daling JR, Sherman KJ. Cancers of the vulva and vagina. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. 2<sup>nd</sup> ed. New York (NY): Oxford University Press; 1996. p. 1117–29.
- (83) Sturgeon SR, Brinton LA, Devesa SS, Kurman RJ. *In situ* and invasive vulvar cancer incidence trends (1973 to 1987). Am J Obstet Gynecol 1992;166:1482–5.
- (84) Healy JC, Reznek RH. The peritoneum, mesenteries and omenta: normal anatomy and pathological processes. Eur Radiol 1998;8:886–900.
- (85) Hamrick-Turner JE, Chiechi MV, Abbitt PL, Ros PR. Neoplastic and inflammatory processes of the peritoneum, omentum, and mesentery: diagnosis with CT. Radiographics 1992;12:1051–68.
- (86) Weiss N, Cook LS, Farrow DC, Rosenblatt KA. Ovarian cancer. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. 2<sup>nd</sup> ed. New York (NY): Oxford University Press; 1996. p. 1040–57.
- (87) McGowan L, Norris HJ. The mistaken diagnosis of carcinoma of the ovary. Surg Gynecol Obstet 1991;173:211–5.
- (88) Scully RE, Salazar H. Problems in the histological typing of ovarian cancer. In: Stalsberg H, editor. An international survey of distributions of histologic types of tumours of the testis and ovary. Vol 75. Geneva (Switzerland); UICC Technical Report Series; 1983. p. 123–35.

- (89) Gitsch G, Tabery U, Feigl W, Breitenecker G. The differential diagnosis of primary peritoneal papillary tumors. Arch Gynecol Obstet 1992;251: 139–44.
- (90) National Center for Health Statistics. Vital statistics of the United States, 1994. Vol. II, Part A: Mortality, "Technical Appendix." Hyattsville (MD): National Center for Health Statistics; 1994.
- (91) Rosenberg HM, Maurer JD, Sorlie PD, Johnson NJ, MacDorman MF, Hoyert DL, et al. Quality of death rates by race and Hispanic origin: a summary of current research, 1999. Hyattsville (MD): National Center for Health Statistics. Vital and Health Statistics, Series 2, No. 128. September 1999.
- (92) Poe GS, Powell-Griner E, McLaughlin JK, Placek PJ, Thompson GB, Robinson K. Comparability of the death certificate and the 1986 National Mortality Followback Survey. Hyattsville (MD): National Center for Health Statistics. Vital Health Stat 1993;2:118.
- (93) Hogan, H. The 1990 post-enumeration survey: operations and results. J Am Stat Assoc 1993;88:1047–60.
- (94) Reducing tobacco use: a report of the Surgeon General. MMWR Recommendations and Reports December 22, 2000;49(RR16):1–27.
- (95) A clinical practice guideline for treating tobacco use and dependence: a U.S. Public Health Service Report. JAMA 2000;283:3244–54.
- (96) Doll R, Peto R. The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. J Natl Cancer Inst 1981;66: 1191–308.
- (97) Marks R. Melanoma prevention: is it possible to change a population's behavior in the sun? Pigment Cell Res 1994;7:104–6.
- (98) Hsu HM, Lu CF, Lee SC, Lin SR, Chen DS. Seroepidemiologic survey for hepatitis B virus infection in Taiwan: the effect of hepatitis B mass immunization. J Infect Dis 1999;179:367–70.
- (99) Harpaz R, McMahon BJ, Margolis HS, Shapiro CN, Havron D, Carpenter G, et al. Elimination of new chronic hepatitis B virus infections: result of the Alaska immunization program. J Infect Dis 2000;181:413–8.
- (100) Chang MH, Chen CJ, Lai MS, Hsu HM, Wu TC, Kong MS, et al. Universal hepatitis B vaccination in Taiwan and the incidence of hepatocellular carcinoma in children. Taiwan Childhood Hepatoma Study Group. N Engl J Med 1997;336:1855–9.

- (101) McMahon BJ, Bulkow L, Harpster A, Snowball M, Lanier A, Sacco F, et al. Screening for hepatocellular carcinoma in Alaska natives infected with chronic hepatitis B: a 16-year population-based study. Hepatology 2000; 32:842–6.
- (102) Margolis HS, Coleman PJ, Brown RE, Mast EE, Shinengold SH, Arevalo JA. Prevention of hepatitis B virus transmission by vaccination. An economic analysis of current recommendations. JAMA 1995;274: 1201–8.
- (103) Wong JB, McQuillan GM, McHutchison JG, Poynard T. Estimating future hepatitis C morbidity, mortality, and costs in the United States. Am J Public Health 2000;90:1562–9.
- (104) Saltz LB, Cox JV, Blanke C, Rosen LS, Fehrenbacher L, Moore MJ, et al. Irinotecan plus fluorouracil and leucovorin for metastatic colorectal cancer. Irinotecan Study Group. N Engl J Med 2000;343:905–14.
- (105) Swan J, Wingo PA, Clive R, West D, Miller D, Hutchison C, et al. Cancer surveillance in the U.S.: can we have a national system? Cancer 1998; 83:1282–91.
- (106) Hankey BF, Ries LA, Edwards BK. The Surveillance, Epidemiology and End Results Program: a national resource. Cancer Epidemiol Biomarkers Prev 1999;8:1117–21.
- (107) Miller M, Swan J. SEER doubles coverage by adding registries for four states [news]. J Natl Cancer Inst 2001;93:500.

# Notes

<sup>1</sup>An annual percent change (APC) for 1992 through 1998 based on the joinpoint analysis over the interval 1973 through 1998 may differ from an APC for the selected interval of 1992 through 1998 in column 2 of Tables 1 and 2.

We thank Danielle Harkins and Martin Krapcho, Information Management Services, Inc., Silver Spring, MD, for preparing the tables and graphs and Hal Margolis of the Centers for Disease Control and Prevention, Atlanta, GA, for assisting with the liver cancer strategies.

Manuscript received January 31, 2001; revised April 25, 2001; accepted April 26, 2001.