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Chapter 3
Health Consequences of Tobacco Use Among Four Racial/Ethnic Minority Groups

Introduction

The fact that cigarette smoking causes cancer, respiratory and cardiovascular diseases, and adverse pregnancy outcomes is well established (U.S. Department of Health and Human Services [USDHHS] 1989b). Evidence of the relationship between smoking and lung cancer began to accumulate as early as the late 1930s (Ochsner and DeBakey 1939; U.S. Department of Health, Education, and Welfare [USDHEW] 1964). In 1964, the first Surgeon General's report linking smoking to disease concluded that cigarette smoking was a cause of lung and laryngeal cancers in men and a probable cause of lung cancer in women. In more recent reports, the Surgeon General has concluded that cigarette smoking causes 87 percent of lung cancer deaths, 30 percent of all cancer deaths, 82 percent of chronic obstructive pulmonary disease (COPD) deaths, 21 percent of coronary heart disease (CHD) deaths, and 18 percent of deaths from stroke (USDHHS 1989b) as well as 21–39 percent of low-birth-weight births and 14 percent of preterm deliveries (USDHHS 1980, 1989b). In addition, passive or involuntary smoking causes lung cancer in healthy nonsmokers and respiratory problems in young children (USDHHS 1986a; U.S. Environmental Protection Agency 1992).

Despite this wealth of knowledge about the health consequences of smoking, few studies have examined the relationship between tobacco use and known health effects among racial/ethnic groups in the United States. Moreover, few databases include information on sufficient numbers of persons from racial/ethnic groups to allow such analyses.

Although sufficient data are often not available for these population subgroups, the objectives of this chapter are to assess the burden of smoking-related diseases among U.S. racial/ethnic groups, to examine racial/ethnic differences in tobacco-related morbidity and mortality when possible, and to review studies that have examined how the relationship between tobacco use and selected health outcomes may differ among racial/ethnic groups. For many of the adverse health outcomes and diseases presented in this chapter, smoking is one of many contributing factors. The focus in this chapter is on the disease burden related to smoking among four U.S. racial/ethnic minority groups (African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, and Hispanics); data on the contribution of cigarette smoking to any differences between groups are highlighted whenever available. A discussion of some relevant methodological issues is provided in the chapter appendix.

Lung Cancer

The 1964 Surgeon General's report on smoking and health concluded that "Cigarette smoking is causally related to lung cancer in men; the magnitude of the effect far outweighs all other factors. The data for women, though less extensive, point in the same direction" (USDHEW 1964). That conclusion was based on strong epidemiological evidence from case-control and cohort studies and supporting toxicological evidence. When reviewed against criteria for causality, the evidence was initially judged to be sufficient for men and a similar conclusion was subsequently reached for women (USDHHS 1980).

Since the 1964 Surgeon General's report, voluminous evidence has accumulated about the relationship between smoking and lung cancer (USDHHS 1989b; Wu-Williams and Samet 1994). The epidemiological studies consistently indicate that the risk of lung cancer increases with the number of cigarettes smoked and with the length of time a person smokes. Furthermore, evidence shows that in comparison with smokers of non-filtered cigarettes, smokers of filtered cigarettes have only slightly less risk of lung cancer (Wu-Williams and Samet 1994). Although a family history of lung cancer is associated with increased risk, the genetic basis for this association has not yet been determined (Economou et al. 1994). Environmental agents other than cigarette smoke, including certain occupational agents (Coultas and Samet

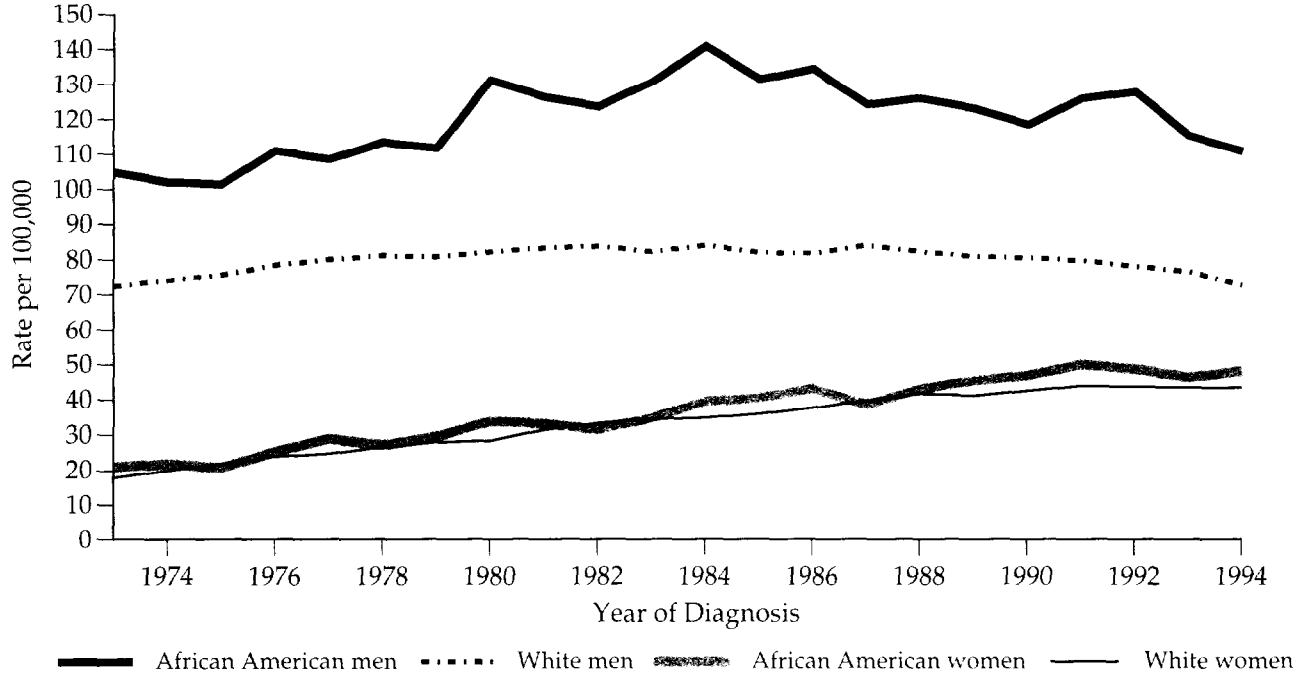
1992; Coultas 1994) and indoor and outdoor air pollutants (Samet 1993), also cause lung cancer. For example, synergism between smoking and radon and asbestos has been demonstrated in studies of worker groups (Saracci and Boffetta 1994).

Because nearly all cases of lung cancer are attributable to cigarette smoking, variations in lung cancer patterns between racial/ethnic groups most likely reflect differences in smoking patterns. Whenever more detailed information is available, it is included in the appropriate sections that follow.

African Americans

The population-based cancer registries operated by the National Cancer Institute's (NCI) Surveillance, Epidemiology, and End Results (SEER) Program provide cancer incidence data for several locations throughout the United States, including Connecticut, Hawaii, Iowa, New Mexico, and Utah and the metropolitan areas of Detroit, Atlanta, San Francisco/Oakland, and Seattle/Puget Sound. SEER data show that African American men have had consistently higher lung cancer incidence rates than white men since the 1970s (Figure 1) (Kosary et al. 1995). (SEER data cover about 10 percent of the U.S. population and are used frequently to estimate national cancer rates and trends.) Between 1950 and 1960, age-adjusted death rates for malignant neoplasms of the respiratory system (composed primarily of deaths from lung cancer) among African American men surpassed those among white men and have since remained higher, whereas death rates for African American women have remained fairly similar to those among white women, according to data from the National Vital Statistics System (Table 1) (National Center for Health Statistics [NCHS] 1997). Since 1990, respiratory cancer death rates declined substantially for African American men; among African American women, rates increased through 1990 and then leveled off. From 1992–1994, the age-adjusted death rate for cancer of the trachea, bronchus, and lung (generally referred to as lung

Figure 1. Incidence of cancer of the lung and bronchus, by race/ethnicity and gender, National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program, 1973–1994



Note: Age-adjusted to the 1970 standard U.S. population.
Sources: Adapted from Kosary et al. 1995; Ries et al. 1997.

Table 1. Death rates per 100,000 U.S. residents for malignant diseases of the respiratory system, by race/ethnicity and gender, United States, 1950–1995,* selected years

Race/ethnicity and gender	1950†	1960†	1970	1980	1985	1990	1992	1993	1994	1995
African American men										
All ages, age-adjusted	16.9	36.6	60.8	82.0	87.7	91.0	86.7	86.0	82.8	80.5
All ages, crude	14.3	31.1	51.2	70.8	75.5	77.8	74.7	74.7	72.5	71.2
American Indian or Alaska Native men‡										
All ages, age-adjusted	NA	NA	NA	23.2	28.4	29.7	31.7	31.0	31.1	32.7
All ages, crude	NA	NA	NA	15.7	19.6	21.1	23.1	23.1	23.0	25.1
Asian American or Pacific Islander men§										
All ages, age-adjusted	NA	NA	NA	27.6	26.9	26.8	27.4	28.4	28.0	25.8
All ages, crude	NA	NA	NA	22.9	21.3	21.7	23.0	23.8	23.9	22.4
Hispanic men△										
All ages, age-adjusted	NA	NA	NA	NA	24.0	27.7	24.4	25.1	24.8	25.2
All ages, crude	NA	NA	NA	NA	13.9	17.4	15.9	16.5	16.5	16.9
White men										
All ages, age-adjusted	21.6	34.6	49.9	58.0	58.7	59.0	56.7	56.3	54.8	53.7
All ages, crude	24.1	39.6	58.3	73.4	77.6	81.0	79.5	79.7	78.5	77.8
African American women										
All ages, age-adjusted	4.1	5.5	10.9	19.5	22.8	27.5	28.5	27.3	27.7	27.8
All ages, crude	3.4	4.9	10.1	19.3	23.5	29.2	30.9	30.2	30.8	31.3
American Indian or Alaska Native women‡										
All ages, age-adjusted	NA	NA	NA	8.1	11.1	13.5	15.5	16.1	17.7	16.4
All ages, crude	NA	NA	NA	6.4	9.2	11.3	13.4	14.6	16.5	15.5
Asian American or Pacific Islander women§										
All ages, age-adjusted	NA	NA	NA	9.5	9.2	11.3	11.1	11.7	11.2	13.0
All ages, crude	NA	NA	NA	8.4	8.2	10.6	11.1	11.7	11.4	13.6
Hispanic women△										
All ages, age-adjusted	NA	NA	NA	NA	6.7	8.7	8.4	8.2	8.5	8.2
All ages, crude	NA	NA	NA	NA	5.2	7.5	7.5	7.3	7.7	7.5
White women										
All ages, age-adjusted	4.6	5.1	10.1	18.2	22.7	26.5	27.4	27.6	27.7	27.9
All ages, crude	5.4	6.4	13.1	26.5	34.8	43.4	46.2	47.3	47.9	48.9

Note: Data in the table on African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, and whites include persons of Hispanic and non-Hispanic origin. Conversely, in this table, the data on Hispanic origin may include persons of any race.

*Age-adjusted to the 1940 U.S. standard population. Cause-of-death data are based on classifications from the then-current *International Classification of Diseases* (e.g., cause-of-death codes 160–165 for the Ninth Revision). Data for the 1980s are based on intercensal population estimates.

†Includes deaths of nonresidents of the United States.

‡Interpretation of trends should consider that population estimates for American Indians and Alaska Natives increased by 45 percent between 1980 and 1990 (because of better enumeration techniques in 1990 and an increased tendency for people to denote themselves as American Indian in 1990).

§Interpretation of trends should consider that the Asian population in the United States more than doubled between 1980 and 1990, primarily because of immigration.

△Because of incomplete data, the National Center for Health Statistics (NCHS) reports 1985 death certificate data on decedents of Hispanic origin for only 17 states and the District of Columbia. By 1990, data for 47 states and the District of Columbia were reported. NCHS estimates that the 1990 reporting area encompassed 99.6 percent of the U.S. Hispanic population. After 1992, only Oklahoma did not provide information on Hispanic origin.

NA = data not available.

Source: Adapted from National Center for Health Statistics 1997.

cancer) was highest for African American men (81.6 per 100,000 population) (Table 2); the lung cancer death rate for African American women (27.2 per 100,000) was similar to that for white women (27.9 per 100,000) and higher than that for any other racial/ethnic group. Among African Americans in 1993, the four leading causes of cancer death were lung cancer (26.1 percent of all cancer deaths), cancer of the colon and rectum (10.4 percent), prostate cancer (9.4 percent), and cancer of the female breast (8.3 percent) (Parker et al. 1997).

The higher lung cancer incidence and death rates among African American men have not been fully explained. Two ecological analyses of population-based incidence data for metropolitan areas have shown that the African American-white gradient in lung cancer occurrence among men was consistent with gradients in socioeconomic indicators (Devesa and Diamond 1983; Baquet et al. 1991) and that the difference in lung cancer disappeared when the data were adjusted for socioeconomic status. The authors of one paper (Baquet et al. 1991) surmised that the differences in smoking patterns associated with socioeconomic status accounted for the differences in lung cancer between white and African American men, whereas the authors of the other paper (Devesa and Diamond 1983) proposed that cigarette smoking and other environmental correlates of socioeconomic status, such as dietary habits or occupational exposure, may have accounted for their findings.

Data from several National Health Interview Surveys (NHISs) were used to conduct birth cohort analyses of cigarette smoking prevalence in the 1900s for African Americans and whites of both genders (Tolley et al. 1991; Shopland 1995). Older white men (those born before 1915) experienced higher peak smoking rates and slightly earlier ages of initiation than older African American men. For persons born after 1915, peak smoking rates and duration of smoking for African American men were slightly higher than those for white men. In addition, white male smokers were more likely than African American male smokers to quit smoking in the 1950s (when the early scientific studies on smoking and lung cancer were reported); African American male cohorts born after 1915 thus experienced a greater cumulative exposure to cigarette smoke. Reflecting these trends in smoking behavior, lung cancer mortality rates were initially higher for white men. The combination of less cessation, higher peak prevalence, and longer duration of smoking in African American men after the 1940s likely explains the observation that mortality rates for African American men began to exceed those for white men later in the century (Shopland 1995).

Lung cancer death rates have been much lower for women than for men (reflecting historically lower smoking prevalences) and have risen more slowly with age in the older birth cohorts. As rates for men began to decline in cohorts born after 1930, rates continued to rise among women, reflecting their slower adoption and increasing prevalence of cigarette smoking. African American and white women indicated similar patterns of smoking initiation, maintenance, and quitting; lung cancer death rates for African American and white women also have been similar (Tolley et al. 1991; Shopland 1995). These data are consistent with the interpretation that trends in smoking behavior are largely responsible for 20th century lung cancer mortality patterns for African Americans and whites. Tolley and colleagues (1991) further suggested that lung cancer rates among African American men and women may be slightly higher than those for white men and women, even after considering differences in their smoking behaviors.

One study (Harris et al. 1993) showed a higher lung cancer risk among African Americans compared with whites who had the same level of cumulative exposure to cigarette smoking. In this 20-year case-control study, 2,678 cases of lung cancer were identified among white men, 238 cases among African American men, 1,394 cases among white women, and 113 among African American women; after adjusting the data for cumulative tar consumption and education, the researchers found that African Americans had a significantly higher risk of lung cancer. One limitation of this study is that it uses the Federal Trade Commission's (FTC's) estimates of tar yield to calculate cumulative tar consumption. The FTC's machines are set to parameters that have not changed for decades. Because humans smoke cigarettes differently than the machines used by the FTC, the validity of these measures has been called into question (NCI 1996a). In the Kaiser Permanente cohort study, the relative risks of lung cancer were approximately the same for African Americans and whites (Friedman et al. 1997). Dorgan and colleagues (1993) conducted a case-control study to assess race and gender differences in lung cancer, categorizing participants according to consumption of fruits and vegetables. Lung cancer risk was significantly increased for African Americans who currently smoked (compared with never smokers and former light smokers), regardless of the amount of vegetables consumed. These analyses were statistically adjusted for gender, age, education, occupation, passive smoking, and study phase.

In a recent population-based case-control study to compare the risks of lung cancer for African

Table 2. Age-adjusted death rates* for selected smoking-related causes of death, by race/ethnicity and gender, United States, 1992–1994

Disease Category (*ICD-9* code)†	African American		American Indian/ Alaska Native		Asian American/ Pacific Islander		White		Hispanic	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Cancer										
Lip, oral cavity, pharynx (140–149)	7.7	1.8	2.6	1.0	3.3	1.0	3.0	1.2	2.4	0.5
Esophagus (150)	11.4	3.0	3.2	0.5	2.7	0.5	4.4	0.9	2.8	0.4
Stomach (151)	9.5	4.1	4.9	2.6	8.9	5.1	3.9	1.7	6.2	3.1
Pancreas (157)	11.1	8.1	3.4	3.0	5.5	3.9	7.3	5.2	5.1	3.8
Larynx (161)	4.6	0.8	0.9	0.3	0.6	0.1	1.7	0.4	1.3	0.2
Trachea, bronchus, lung (162)	81.6	27.2	33.5	18.4	27.9	11.4	54.9	27.9	23.1	7.7
Cervix uteri (180)	NA	5.7	NA	3.0	NA	2.5	NA	2.2	NA	3.2
Bladder (188)	3.2	1.6	1.2	0.5	1.5	0.6	3.9	1.1	1.8	0.6
Kidney, other, unspecified urinary organs (189)	4.3	2.0	4.4	2.3	1.8	0.8	4.1	1.9	3.1	1.3
Cardiovascular diseases										
Coronary heart disease (410–414)	138.3	85.0	100.4	45.9	71.7	36.2	132.5	62.9	82.7	43.9
Cerebrovascular disease (430–438)	53.1	40.6	23.9	21.1	29.3	22.4	26.3	22.6	22.7	16.3
Respiratory diseases										
Bronchitis, emphysema (491–492)	4.7	1.6	2.8	1.9	2.9	0.9	6.2	3.8	2.4	0.9
Chronic airway obstruction, not elsewhere classified (496)	17.6	6.6	14.2	9.0	7.9	2.6	20.4	12.2	8.2	3.7

*Per 100,000, age-adjusted to the 1940 U.S. standard population. Estimates for Hispanics exclude data from New Hampshire for 1992 and from Oklahoma for 1992–1994.

†*International Classification of Diseases, Ninth Revision*, World Health Organization 1977.

NA = data not available.

Sources: National Center for Health Statistics, public use data tapes, 1992–1994; U.S. Bureau of the Census 1997.

Americans and whites across categories of cigarette smoking status, Schwartz and Swanson (1997) examined incident cases from the Occupational Cancer Incidence Surveillance Study. This study operates in conjunction with the Metropolitan Detroit Cancer Surveillance System, a participant in the NCI's SEER Program. The analyses were stratified by gender and statistically adjusted for age, education, and cigarette smoking behaviors. The overall risks of lung cancer (of all histological types) were similar for African

Americans and whites. Thus, race did not appear to be an independent predictor of lung cancer in the population as a whole. However, African Americans were more likely than whites to have developed squamous cell carcinoma. Additionally, African American men aged 40–54 years were 2–4 times more likely than white men of the same ages to have developed lung cancer (of several histological types). The authors concluded that the increased risks among younger African Americans may suggest a greater degree of susceptibility to lung carcinogens or greater exposure to other unidentified carcinogens and they called for further research on the topic.

Investigators have postulated that the more frequent smoking of menthol cigarettes by African Americans, compared with whites, contributes to their increased rate of lung cancer (Harris et al. 1993). In a recent experimental study of 12 persons after the amount of menthol injected into experimental cigarettes was increased, the amount of carbon monoxide exhaled by African American smokers also increased (Miller et al. 1994). In a comparison of smoking behavior associated with mentholated cigarettes and regular cigarettes among 29 subjects, McCarthy and colleagues (1995) found higher mean puff volume and higher puff frequency after participants smoked regular cigarettes than after they smoked mentholated cigarettes; however, no differences in mean expired carbon monoxide levels were found. Available data suggest that mentholated cigarettes are not smoked more intensely than regular cigarettes (Jarvik et al. 1994; Miller et al. 1994; McCarthy et al. 1995; Ahijevych et al. 1996). Thus, mentholated cigarettes may promote lung permeability and diffusibility of smoke constituents (Jarvik et al. 1994; McCarthy et al. 1995; Clark et al. 1996a).

Recent studies have examined the possible role of genetics in determining the risk of lung cancer among African Americans. Crofts and colleagues (1993) identified a restriction fragment length polymorphism (RFLP) in the gene (*CYP1A1*) that encodes the enzyme responsible for initiating metabolism of polyaromatic hydrocarbon compounds found in cigarette smoke (Guengerich 1992, 1993). In one study of African Americans, the risk of adenocarcinoma of the lung was higher for smokers with the *CYP1A1 RFLP* than for smokers who did not have this RFLP (Taioli et al. 1995). Two other studies, however, did not find an association between the presence of the variant allele in African Americans and increased lung cancer risk (Kelsey et al. 1994; London et al. 1995). Taioli and colleagues (1995) also found that persons who had adenocarcinoma with the African American *CYP1A1 RFLP* had lower lifetime cigarette consumption, as measured by pack-years, compared with those who had adenocarcinoma without the polymorphism. However, using a cutoff point of 35 pack-years, London and colleagues (1995) found no association between the variant *CYP1A1* variant allele and lung cancer risk based on smoking history. Additionally, a homozygous rare *CYP1A1* allele associated with the risk of lung cancer among persons from Japan (Kawajiri et al. 1990) was found more often in African Americans than in whites (Shields et al. 1993). However, in a small case-control study, no association was observed between the presence of this polymorphism and lung cancer risk (Shields et al. 1993).

Despite strong research interest in this area, scientists have been unable to consistently associate variant alleles with lung cancer susceptibility. The frequencies of the polymorphisms of interest appear to be low in United States populations studied thus far. Low frequencies of the alleles of interest suggest that future investigations must allow for an adequate sample size of the group under study and adjustment for factors such as smoking history and age. In addition, low frequency allelic affects may be negated or obscured by high tobacco exposure levels.

Two phenotypes were identified in African American and white persons representing poor and extensive extremes of glucuronidation (Richie et al. 1997). Glucuronidation is considered a detoxification pathway because it increases the water solubility of a chemical substrate and facilitates excretion (Goldstein and Faletto 1993). The ratio of conjugated metabolite to free metabolite of a tobacco-specific nitrosamine was 30 percent higher in the urine of white smokers than in African American smokers. This finding suggests that African Americans are at higher risk from nitrosamine exposure during smoking because of a decreased capacity to detoxify carcinogenic tobacco-specific nitrosamines. Hence, variability in glucuronosyltransferase activity, or in clearance of glucuronide conjugates, may represent another determinant of cancer risk.

The genetically determined poor, intermediate, or enhanced debrisoquine metabolizer phenotype has been investigated as a risk factor for lung cancer. Homozygous dominant (extensive metabolizer) individuals were found more frequently among white lung cancer patients who smoked cigarettes than white control patients with COPD who smoked cigarettes (Ayesh et al. 1984). Caporaso and colleagues confirmed the association between the extensive debrisoquine metabolizer phenotype and lung cancer risk. In this study, almost equivalent numbers of extensive

metabolizers were found among African Americans (74 percent) and whites (73 percent) (Caporaso et al. 1990).

Another approach in assessing the possible role of genetics is using chromosome breaks to measure cancer susceptibility. One research group has developed an in vitro cytogenic assay that measures mutagen-induced chromosome breaks in short-term lymphocyte cultures. This approach has shown a relationship between mutagen sensitivity and elevated lung cancer. However, attempts to use this method as a predictive marker of racial/ethnic differences in cancer risk in African and Mexican Americans produced inconsistent results (Spitz et al. 1995; Strom et al. 1995; Wu et al. 1996).

Carcinogenesis can involve genotoxic mechanisms whereby chemical interactions at critical cellular sites go unrepaired. Alterations in certain genes, known as proto-oncogenes and tumor suppressor genes, are linked with cancer risk (Land et al. 1983; Marshall et al. 1984; Slamon et al. 1984; Klein and Klein 1985; Denissenko et al. 1996). Some gene alleles that are evaluated as markers of lung cancer risk vary in their distributions among African Americans and whites. For example, in a study of lung cancer cases and trauma victim controls, Weston and colleagues (1991) found rare Ha-ras-1 alleles more often in the lung tissue of African Americans (17 percent) than in whites (5 percent). For both groups, the prevalence of rare alleles among lung cancer patients was higher than among controls (23 percent for African American lung cancer cases, 15 percent for African American trauma victim controls, 6 percent for white lung cancer cases, and 2 percent for white trauma victim controls). These findings were confirmed in a second study (Weston et al. 1992). African American and white differences in distribution of alleles at the L-myc locus and p53 genotype have also been reported. The authors concluded that L-myc genotypes and p53 variants do not predict lung cancer risk (Weston et al. 1992).

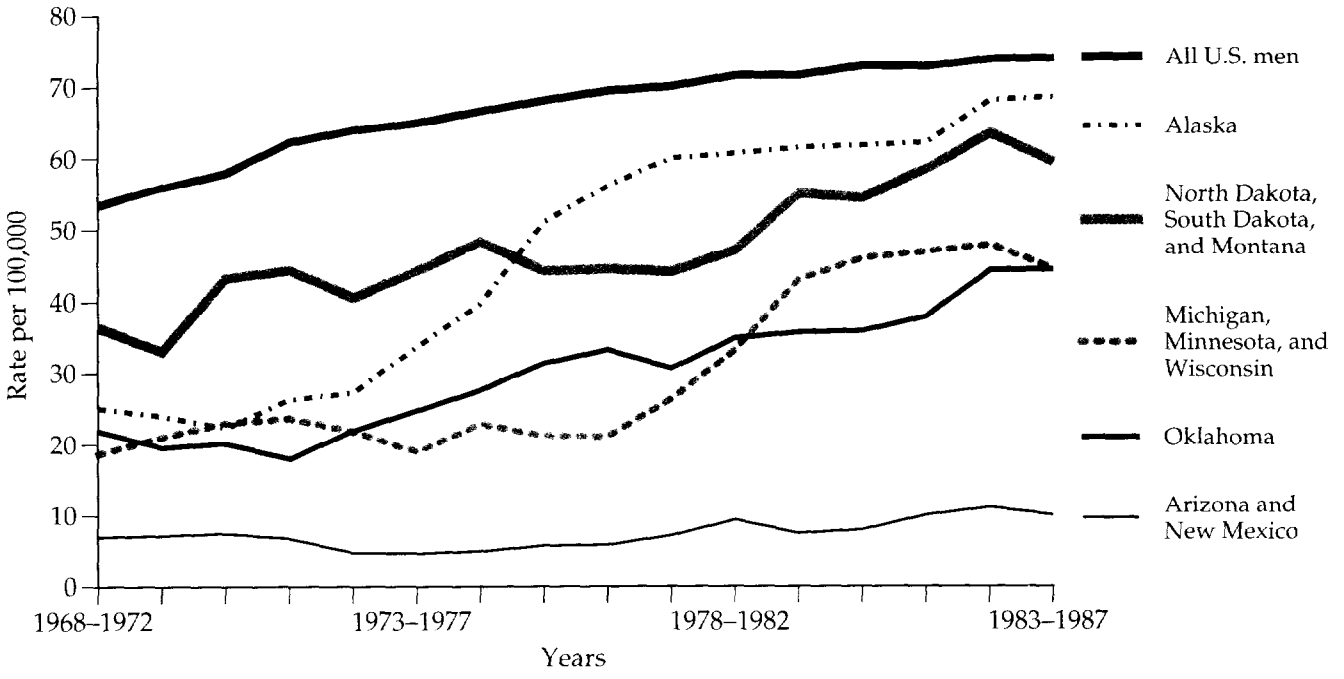
In summary, the higher rates of lung cancer observed among African American men are consistent with historical patterns of cigarette smoking in this century (Shopland 1995). In addition, African American men aged 40–54 years may be especially susceptible to lung carcinogens (Schwartz and Swanson 1997), perhaps because they detoxify them differently (Richie et al. 1997). A genetic role in racial and ethnic-specific risk for lung cancer cannot be ruled out, because some studies have shown that African American populations have increased frequencies of rare alleles associated with greater risks for developing lung cancer than whites. However, because of the low frequency of these alleles in the populations under study and the possibility of misclassification bias, studies have been inconclusive (Shields et al. 1993; Taioli et al. 1995). Further, African American smokers prefer mentholated cigarettes, and menthol may promote the absorption and diffusion of tobacco smoke constituents (Jarvik et al. 1994; McCarthy et al. 1995; Clark et al. 1996a). This hypothesis has received inconsistent support in the epidemiological literature. Kabat and Herbert (1991) found no relationship between menthol use and lung cancer risk; however, Sidney and colleagues (1995) suggested that smoking mentholated cigarettes increased the risk of lung cancer only in male smokers. Further research could clarify the nature of individual susceptibility and the possible role of mentholation. Reduction in cigarette smoking will undoubtedly lead to reduction in the risk of lung cancer for African Americans.

American Indians and Alaska Natives

Since the early 1900s, many studies have documented the low overall occurrence of cancer among American Indians compared with whites (Hoffman 1928; Smith et al. 1956; Smith 1957; Salsbury et al. 1959; Sievers and Cohen 1961; Kravetz 1964; Reichenbach 1967; Creagan and Fraumeni 1972; Dunham et al. 1973; Blot et al. 1975; Lanier et al. 1976; Samet et al. 1980, 1988b; Sorem 1985; Mahoney and Michalek 1991; Nutting et al. 1993). Investigations of lung cancer incidence and deaths have confirmed that lung cancer is less frequent among American Indians overall than among whites (Coultas et al. 1994). Between 1992 and 1994, age-adjusted death rates for lung cancer per 100,000 among American Indian and Alaska Native men (33.5) and women (18.4) were slightly higher than those among Asian American and Pacific Islanders as well as Hispanics, whereas they were lower than rates among African Americans and whites (Table 2) (NCHS, public use data tapes, 1992–1994; U.S. Bureau of the Census 1997). Mortality rates for malignant diseases of the respiratory system increased from 1980 through 1995 among American Indians and Alaska Natives (Table 1) (NCHS 1997).

Nationally, lung cancer is the leading cause of cancer death among American Indians and Alaska Natives. Among those who died of cancer in 1993, the four leading causes of death were lung cancer (26.8 percent), cancer of the colon and rectum (8.9 percent), cancer of the female breast (6.3 percent), and prostate cancer (6.0 percent) (Parker et al. 1997). Additionally, lung cancer was the leading cause of cancer death among both men and women in 10 of the 12 Indian

Figure 2. Age-adjusted lung cancer death rates among American Indian and Alaska Native men in selected states compared with rates among all U.S. men, 1968–1987*

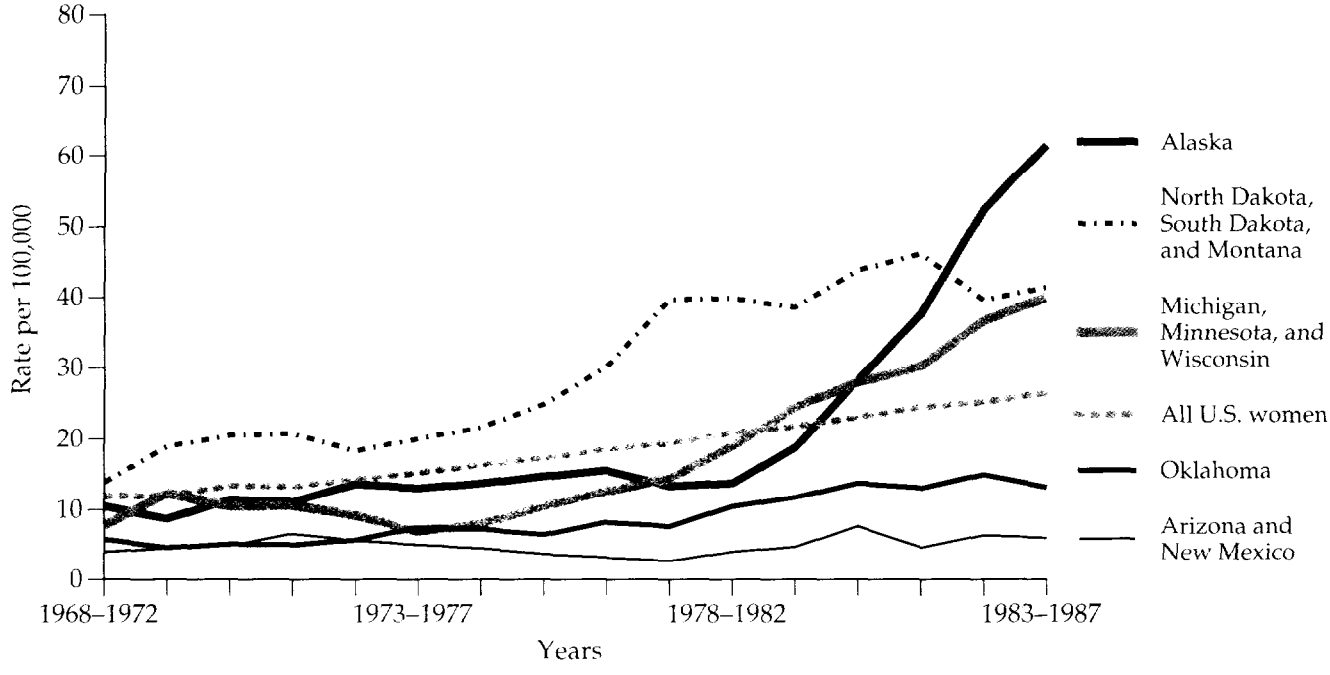


*Rates presented here were determined using midpoint population estimates for each 5-year time interval and were adjusted to the 1970 U.S. standard population.
Source: Valway 1992.

Health Service (IHS) areas (Arizona and New Mexico had low rates of lung cancer deaths) (Valway 1992). Lung cancer death rates among American Indians and Alaska Natives have been rising in most IHS areas (Figures 2 and 3) (Valway 1992); national death rates from malignant diseases of the respiratory system have also been increasing (Table 1).

Lung cancer death rates vary by IHS area. Specifically, American Indians in the Southwest have had the lowest lung cancer death rates, whereas American Indians in Alaska, North Dakota, South Dakota, and Montana have had rates nearly as high as those in the general U.S. population (Table 3, Figures 2 and 3) (Valway 1992). These differences are associated with variations in smoking among American Indians and Alaska Natives (Centers for Disease Control [CDC] 1987; Welty et al. 1993). In an analysis of data from the 1985–1988 Behavioral Risk Factor Surveillance System (BRFSS) on 1,055 American Indians, Sugarman and colleagues (1992) determined smoking prevalence for three groups of states that contained three specific IHS areas. In this study, the Plains states (Iowa, Minnesota, Montana, Nebraska, North Dakota, South Dakota, and Wisconsin) contained the Aberdeen, Bemidji, and Billings IHS areas; the West Coast states (California, Idaho, and Washington) contained the Portland and California IHS areas; and the Southwest states (Arizona, New Mexico, and Utah) contained the Albuquerque, Navajo, Tucson, and Phoenix IHS areas. Cigarette smoking prevalence rates were highest in the Plains states (48.4 percent for men and 57.3 percent for women), intermediate in the West Coast states (25.2 percent for men and 31.6 percent for women), and lowest in the Southwestern states (18.1 percent for men and 14.7 percent for women). These general geographic patterns of smoking prevalence paralleled patterns of lung cancer mortality (Table 3) (Valway 1992). The smoking prevalence estimates from the 1985–1988 BRFSS analyses may be imprecise because of relatively small samples. However, other analyses (American Indians and Alaska Natives, in Chapter 2; Welty et al. 1995) show similar patterns. Another

Figure 3. Age-adjusted lung cancer death rates among American Indian and Alaska Native women in selected states compared with rates among all U.S. women, 1968–1987*



*Rates presented here were determined using midpoint population estimates for each 5-year time interval and were adjusted to the 1970 U.S. standard population.
Source: Valway 1992.

potential limitation is that American Indians living in the California and Portland IHS areas may be more likely than American Indians from other IHS areas to be misclassified on death certificates as being of other racial/ethnic categories (Valway 1992), suggesting that death rates for American Indians may be underestimated in these areas (Sorlie et al. 1992).

Lanier and colleagues (1996) recently reported on lung cancer incidence rates for Alaska Native men and women. Lung cancer incidence was higher for Alaska Natives than it was for the general U.S. population. In addition, lung cancer was the most common incident cancer among men and the third most common incident cancer among women (after breast cancer and cancer of the colon/rectum). Lung cancer incidence increased substantially among Alaska Native men (by 93 percent) and women (by 241 percent) between 1969–1973 and 1989–1993. The authors concluded, "Reduction in tobacco use would result in the greatest decreases in cancer rates in this population" (p. 751).

Asian Americans and Pacific Islanders

Two issues should always be kept in mind when interpreting data about the health consequences of cigarette smoking among Asian Americans and Pacific Islanders: the diversity of this group and the paucity of data. The Asian American and Pacific Islander population of the United States includes approximately 32 national and racial/ethnic groups and nearly 500 languages and dialects. Although many of these persons were born in the United States, many others are recent immigrants (see Chapters 1 and 2); yet the national data do not indicate these distinctions. Environmental exposures experienced in Asia, such as women's exposure to smoke from cooking fuels, may influence lung cancer occurrence among recent immigrants (Coultas et al. 1994).

From 1980 through 1995, age-adjusted death rate for malignant neoplasms of the respiratory system (primarily deaths from lung cancer) among Asian

Table 3. Death rates for lung cancer among American Indians and Alaska Natives, by Indian Health Service (IHS) area, 1984–1988

Areas	Men		Women	
	N	Rate*	N	Rate*
U.S., all ethnicities		74.2		27.3
Nine IHS areas*†	307	38.5‡	203	27.2
All 12 IHS areas	562	40.1‡	296	21.4‡
Aberdeen	63	68.7	41	45.0‡
Alaska	80	75.5	62	68.5‡
Albuquerque	12	18.8‡	5	7.8‡
Bemidji	41	63.4‡	24	40.7‡
Billings	36	65.3	33	65.7‡
California†	33	33.2‡	8	6.6‡
Nashville	24	41.8‡	15	25.1
Navajo	25	11.4‡	7	4.0‡
Oklahoma†	167	46.0‡	55	14.0‡
Phoenix	20	17.2‡	13	11.5‡
Portland†	55	40.5‡	30	23.4
Tucson	6	25.9‡	3	13.5‡

*Per 100,000, age-adjusted to the 1970 U.S. standard population. Rates based on a small number of deaths should be interpreted with caution.
†The California, Oklahoma, and Portland IHS areas appear to have a problem with underreporting Indian ethnicity on death certificates; therefore, a separate total is presented for the nine other IHS areas, excluding these three areas.
‡Denotes a rate significantly different from the rate for the overall U.S. population.
Source: Valway 1992.

American and Pacific Islander men remained fairly constant; this death rate for Asian American and Pacific Islander women increased slightly between 1980 and 1995 but was substantially lower than for men (Table 1) (NCHS 1997). Trends should be interpreted with caution because the large numbers of immigrants from Asia and the Pacific Islands that came to the United States during that time may have influenced both disease prevalence in and the age structure of this group. During 1992–1994, the age-adjusted death rate for lung cancer was 27.9 per 100,000 for Asian American and Pacific Islander men and 11.4 per 100,000 for women (Table 2). These rates were slightly higher than those for Hispanics and slightly lower than those for American Indians and Alaska Natives. In 1993, the four leading causes of cancer death among Asian Americans and Pacific Islanders were lung cancer (22.3 percent of all cancer deaths), cancer of the colon and rectum (10.4 percent), cancer of the liver and intrahepatic bile duct (8.6 percent), and stomach cancer (7.7 percent) (Parker et al. 1997).

Data on lung cancer for more specific subgroups have been published in several reports (Baquet et al. 1986; Ross et al. 1991; Zane et al. 1994; NCI 1996b). The most recent data are from NCI's SEER program and provide information for 1988–1992. This report includes incidence data from the nine areas included in the annual SEER reports (e.g., Kosary et al. 1995) and from Los Angeles, San Jose/Monterey, and the Alaska Area Native Health Service. Data on Hispanics are predominantly from Los Angeles, New Mexico, San Francisco, and San Jose/Monterey. Most Hispanics represented in SEER are Mexican Americans. Data on Asian Americans and Pacific Islanders are mainly from Los Angeles, Hawaii, San Francisco/Oakland, San Jose/Monterey, and Seattle/Puget Sound. Data on American Indians are from New Mexico; data from the Alaska Native Area Health Service provide information on Alaska Natives (NCI 1996b).

During 1988–1992, the age-adjusted (to the 1970 U.S. standard population) incidence per 100,000 population of lung cancer for men was 89.0 for Hawaiians, 70.9 for Vietnamese, 53.2 for Koreans, 52.6 for Filipinos, 52.1 for Chinese, and 43.0 for Japanese. For comparison purposes, the lung cancer incidence rates were 117.0 for African American men, 76.0 for white men, and 41.8 for Hispanic men. For women, the lung cancer incidence rates were 43.1 for Hawaiians, 31.2 for Vietnamese, 25.3 for Chinese, 17.5 for Filipinos, 16.0 for Koreans, and 15.2 for Japanese. In comparison, the lung cancer incidence rates were 44.2 for African American women, 41.5 for white women, and 19.5 for Hispanic women.

Age-adjusted lung cancer death rates during 1988–1992 were, per 100,000 men, 88.9 for Hawaiians, 40.1 for Chinese, 32.4 for Japanese, and 29.8 for Filipinos; mortality estimates were not available for Koreans and Vietnamese of either gender. In comparison, the lung cancer death rates were 105.6 for African American men, 72.6 for white men, and 32.4 for Hispanic men. For women, the lung cancer death rates were 44.1 for Hawaiians, 18.5 for Chinese, 12.9 for Japanese, and 10.0 for Filipinos. In comparison, the lung cancer death rates were 31.9 for white women, 31.5 for African American women, and 10.8 for Hispanic women (NCI 1996b). The lung cancer rates reflect gender differences in smoking rates among Asian American and Pacific Islander populations, as indicated by 1978–1995 data from the NHISs (see Chapter 2).

Several studies have identified high rates of lung cancer among Native Hawaiians. Data on lung cancer among Pacific Islanders from the Hawaii Tumor Registry indicate that Native Hawaiians have the highest lung cancer incidence rates among the islands' other racial/ethnic groups, including Japanese, Filipinos, and Chinese (Kolonel 1980; Hinds et al. 1981). Using medical records of lung cancer patients and data from a population-based survey, Hinds and colleagues (1981) assessed the risk of developing lung cancer associated with smoking among women in Hawaii. The risk for developing lung cancer among women who had ever smoked compared with those who had never smoked was substantially greater among Native Hawaiian women (tenfold higher) than among Japanese women (fivefold higher) and Chinese women (twofold higher). In a comparison of the risks of smoking among Native Hawaiians, Filipinos, Japanese, and Chinese in Hawaii, Le Marchand and colleagues (1992) found that Native Hawaiian men had the highest risk and that white and Filipino women had higher risks than Native Hawaiian women. The pattern of variation of smoking's effect on lung cancer was statistically significant for men. These differences persisted after variables for beta-carotene and cholesterol intake were included in the statistical model. The observation that the risk of lung cancer related to smoking may vary among subgroups requires further elucidation. In a cohort study of 7,961 Japanese American men who were living in Hawaii, the incidence of lung cancer was 11.4 times higher in current smokers than in persons who had never smoked; the risk for former smokers was 3.1 times higher than for never smokers (Chyou et al. 1993).

Hispanics

According to NCHS data from 1985 through 1995, the age-adjusted death rate for malignant neoplasms of the respiratory system (primarily deaths from lung cancer) among Hispanic men was about three times higher than that for Hispanic women (Table 1) (NCHS 1997). Trends should be interpreted with caution, because only 17 states and the District of Columbia contributed death certificate data on Hispanics for 1985; by 1990, however, 47 states and the District of Columbia, covering 99.6 percent of the U.S. Hispanic population, contributed relevant data (Table 1) (NCHS 1997). From 1992 through 1994, the age-adjusted death rate for cancer of the trachea, bronchus, and lung (generally referred to as lung cancer) was 23.1 per 100,000 for Hispanic men and 7.7 per 100,000 for Hispanic women (Table 2). Overall, lung cancer is the leading cause of cancer death among Hispanics. Among those who died of cancer in 1993, the four leading causes of death were lung cancer (17.9 percent), cancer of the colon and rectum (9.6 percent), cancer of the female breast (8.2 percent), and cancer of the liver and other biliary organs (6.0 percent) (Parker et al. 1997). Among Hispanic women, however, breast cancer mortality exceeds that of lung cancer (NCI 1996b).

National mortality data for 1992–1994 (Table 4) also indicate that rates of lung cancer per 100,000 were higher among Cuban men (33.7) than among Mexican American (28.3) and Puerto Rican men (21.9). Among women, little variation is evident across Hispanic subgroups (Table 4). An earlier nationwide analysis limited to foreign-born Cubans, Mexicans, and Puerto Ricans provided similar results for 1979–1981 (Rosenwaike 1987).

Some regional data suggest that rates of lung cancer among Hispanics increased rapidly. For example, New Mexico mortality data for 1958–1982 indicate that lung cancer death rates increased for successive birth cohorts of Hispanics (Samet et al. 1988b). Between 1958–1962 and 1978–1982, lung cancer death rates per 100,000 increased from 10.1 to 28.8 among Hispanic men and from 4.8 to 11.2 among Hispanic women (Samet et al. 1988b). However, lung cancer death rates among Hispanics remained below those of the general U.S. population. Moreover, between 1969–1971 and 1979–1981, lung cancer incidence rates doubled for persons with Spanish surnames (not necessarily all persons were Hispanic) residing in the Denver, Colorado, area (Savitz 1986).

National and regional vital statistics have shown that patterns of lung cancer incidence differ among Hispanics and whites throughout the United States (NCHS 1994). Much of the information available on lung cancer incidence has relied on the SEER Program, which for many years included only one subgroup of Hispanics—those residing in New Mexico.

Since the 1950s, descriptive studies of death have documented differing patterns of lung cancer among Hispanics and whites in the western and southwestern United States. In California, during the 1950s and 1960s, age-specific death rates from lung cancer among older Mexican-born women were two to three times the rates among California women of all ages (Buechley et al. 1957; Buell et al. 1968). Lung cancer death rates for women in Texas and New Mexico during the 1960s and 1970s showed a similar pattern of age-specific rates (Lee et al. 1976; Samet et al. 1980, 1988b), although Hispanic women in the West and Southwest have had lower overall lung cancer death rates than white women (Savitz 1986; Martin and Suarez 1987; Samet et al. 1988b; Bernstein and Ross 1991).

Table 4. Age-adjusted death rates* for selected smoking-related causes of death among Mexican Americans, Puerto Rican Americans, and Cuban Americans, United States, 1992–1994

Disease category (*ICD-9* code)†	Mexican		Puerto Rican		Cuban	
	Men	Women	Men	Women	Men	Women
Cancer						
Lip, oral cavity, pharynx (140–149)	2.0	0.4	5.5	0.9	3.3	0.7
Esophagus (150)	2.7	0.3	6.1	1.1	2.7	0.4
Stomach (151)	6.8	3.5	7.7	3.9	3.1	1.3
Pancreas (157)	5.4	4.3	5.0	3.6	5.0	4.1
Larynx (161)	1.1	0.1	2.6	0.3	2.2	0.1
Trachea, bronchus, lung (162)	21.9	8.0	28.3	9.6	33.7	8.9
Cervix uteri (180)	NA	3.7	NA	3.7	NA	1.6
Bladder (188)	1.4	0.5	2.1	1.0	3.5	0.5
Kidney, other, unspecified urinary organs (189)	3.7	1.6	1.9	1.0	2.7	1.0
Cardiovascular diseases						
Coronary heart disease (410–414)	82.3	44.2	118.6	67.3	95.2	42.4
Cerebrovascular disease (430–438)	25.5	18.9	27.3	16.5	17.1	11.5
Respiratory diseases						
Bronchitis, emphysema (491–492)	2.2	0.9	3.2	1.3	3.3	1.0
Chronic airway obstruction, not elsewhere classified (496)	7.6	3.7	10.5	5.3	9.1	3.1

*Per 100,000, age-adjusted to the 1940 U.S. standard population. Death rates are not available from New Hampshire for 1992 and from Oklahoma for 1992–1994. Due to limitations in the data, the population estimates for Oklahoma and New Hampshire were not subtracted from the denominator. Based on the 1990 Census, the number of persons of Hispanic origin from New Hampshire and Oklahoma represented about 0.04 percent of the U.S. Hispanic population.

†*International Classification of Diseases, Ninth Revision*, World Health Organization 1977.

NA = data not available.

Sources: National Center for Health Statistics, public use data tapes, 1992–1994; U.S. Bureau of the Census 1997.

In 1982 and 1983, lung cancer rates among Hispanic men and women in Florida also were lower than the rates among whites (Trapido et al. 1990a,b). More recent data (1981–1989) from Dade County, Florida, again show the incidence of lung cancer to be lower among Hispanic men than among white men and lower among Hispanic women than white women (Trapido et al. 1994a,b). Similarly, Mexican and Puerto Rican immigrants in Illinois have had lower standardized lung cancer death rates than whites (Mallin and Anderson 1988). In addition, lung cancer incidence and death rates have been much lower among Hispanic men than among white men in New Mexico (Samet et al. 1980), Texas (Lee et al. 1976), California (Menck et al. 1975; Bernstein and Ross 1991), Connecticut (Polednak 1993), and Colorado (Savitz 1986). Mortality data indicate that Puerto Ricans living on Long Island, New York, had slightly lower death rates for lung cancer than Puerto Ricans living elsewhere in the United States (except Puerto Rico) (Polednak 1991). However, Puerto Rican men and women residing on Long Island had lung cancer death rates that were three to four times the rates among Puerto Rico residents.

These lower rates of lung cancer among Hispanics appear to reflect differences in smoking between Hispanics and whites. The results of a 1980–1982 case-control study of lung cancer cases among Hispanics and whites residing in New Mexico indicate that the risks (adjusted for gender and age) across categories of smoking consumption among both groups were comparable (Table 5) (Humble et al. 1985). This finding suggests that the reduced rates of lung cancer deaths among Hispanics are attributable to their lower cigarette consumption (number of cigarettes smoked daily) and not to some other correlate of Hispanic race/ethnicity. In a mortality study conducted in Texas between 1970 and 1979 using age-standardized death rates, Holck and colleagues (1982) found that Mexican American women had stable lung cancer death rates (approximately 30 per 100,000), whereas white women had increasing rates of death from lung cancer. The lower lung cancer rates for Mexican American women were consistent with their lower prevalence of smoking (18.5 percent of Mexican American women vs. 31.6 percent of white women).

The elevated rates of lung cancer death among older Hispanic women in the West and Southwest have been attributed to a possible pattern of early initiation of smoking among women born in Mexico before 1900 as well as the custom of cooking indoors with an open fire (Buell et al. 1968; Lee et al. 1976). The findings of a 1980–1982 case-control study in New Mexico indicate that older Hispanic women smoked hand-rolled cigarettes, which may have contributed to the high lung cancer death rate among older Mexican American women (Humble et al. 1985).

Table 5. Odds ratios for the risk of lung cancer, by gender, race/ethnicity, and smoking status, case-control study, New Mexico,* 1980–1982

	Men	
Smoking status	**Hispanic**	**White**
Former smokers	8.0[†] (1.9–42.2)[‡]	7.2 (3.0–17.6)
Current smokers	11.6	9.2
<20 cigarettes per day	(2.7–61.5)	(3.3–25.8)
≥20 cigarettes per day	26.1 (5.6–146.6)	24.7 (10.0–59.9)
	Women	
	Hispanic	**White**
Former smokers	6.3[†] (1.5–27.8)	6.5 (2.8–15.4)
Current smokers	18.5	19.2
<20 cigarettes per day	(4.9–72.4)	(6.5–60.8)
≥20 cigarettes per day	36.9 (7.6–217.1)	16.0 (6.7–36.3)

*Mantel-Haenszel estimates of exposure odds ratios were calculated for two age strata: <65 years of age and ≥65 years of age. Odds ratios are relative to persons who never smoked.
[†]$p < 0.01$.
[‡]95% Cornfield confidence limits; unless otherwise indicated, $p < 0.0001$.
Source: Adapted from Humble et al. 1985.

Other Cancers

Cigarette smoking causes cancers of the lung, larynx, mouth, esophagus, and bladder; is a contributing factor for cancers of the pancreas, kidney, and cervix; and is associated with cancer of the stomach (USDHHS 1989b, 1990). Cigarette smoking is also suspected of contributing to colon cancer (Giovanucci et al. 1994), liver cancer (Doll et al. 1994), and acute myeloid leukemia (Siegel 1993). Little information is available on cigarette smoking as a risk factor for these cancers among members of racial/ethnic minority groups. In the annual Cancer Statistics Review of the SEER Program, cancer incidence and death rates are reported for African Americans and whites (Kosary et al. 1995). A special 1986 report provides more detailed information on African Americans and other ethnic groups for 1978–1981 (Baquet et al. 1986). A more recent report provides detailed information on several ethnic groups for 1988–1992 (NCI 1996b). Other population-based cancer registries are also beginning to contribute relevant information.

Several recently published sources of information on cancer among American Indians include an IHS

report, which describes regional differences in cancer deaths among American Indians in the United States for 1984–1988 and time trends for 1968–1987 (Valway 1992); two reports from the Alaska Area Native Health Service (Lanier et al. 1993, 1996), which describe cancer incidence in the state's Eskimo, Aleut, and Indian populations; and an NCI monograph that documents the status of the evidence on cancer and the need for additional research regarding cancer among American Indians and Alaska Natives (Burhansstipanov and Dresser 1993).

Table 6. Age-adjusted incidence and death rates* for selected smoking-related cancers, by race/ethnicity and gender, National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program, 1988–1992

Primary cancer site (*ICD-9* code)†	African American	Alaska Native	American Indian (New Mexico)	Chinese	Filipino
All sites					
Incidence rate,§ men	560¶	372	196	282	274
Incidence rate, women	326	348	180	213	224
Death rate,¶ men	319	225	123	139	105
Death rate, women	168	179	99	86	63
Cervix uteri (180)					
Incidence rate, women	13.2	15.8	9.9	7.3	9.6
Death rate, women	6.7	–**	–	2.6	2.4
Esophagus (150)					
Incidence rate, men	15.0	–	–	5.3	2.9
Incidence rate, women	4.4	–	–	–	–
Death rate, men	14.8	–	–	4.2	2.2
Death rate, women	3.7	–	–	–	–
Kidney and renal pelvis (189.0–189.1)					
Incidence rate, men	12.8	–	15.6	4.6	5.8
Incidence rate, women	6.0	–	–	2.3	2.8
Death rate, men	5.1	–	–	1.3	1.9
Death rate, women	2.2	–	–	0.9	–
Larynx (161)					
Incidence rate, men	12.7	–	–	2.8	2.4
Incidence rate, women	2.5	–	–	–	–
Death rate, men	5.6	–	–	0.9	–
Death rate, women	0.9	–	–	–	–
Lung and bronchus (162.2–162.9)					
Incidence rate, men	117.0	81.1	14.4	52.1	52.6
Incidence rate, women	44.2	50.6	–	25.3	17.5
Death rate, men	105.6	69.4	–	40.1	29.8
Death rate, women	31.5	45.3	–	18.5	10.0

*Rates per 100,000, age-adjusted to the 1970 U.S. standard population.
†U.S. Department of Health and Human Services 1989a.
‡Includes persons of other ethnic groups who designated themselves as of Hispanic origin.
§All incidence data are from five states: Connecticut, Hawaii, Iowa, New Mexico, and Utah; from six metropolitan areas: Atlanta (including 10 rural counties), Detroit, Los Angeles, San Francisco/Oakland, San Jose/Monterey, and Seattle/Puget Sound; and from the Alaska Area Native Health Service.

Death and incidence data both indicate marked heterogeneity of cancer occurrence among racial/ethnic groups in the United States, and this heterogeneity extends to the cancer sites associated with cigarette smoking. For example, SEER data indicate that African Americans have higher incidence and death rates than whites for a number of smoking-related cancer sites, including the oral cavity and pharynx, esophagus, cervix uteri, larynx, stomach, pancreas, and lung (Table 6; Figure 4) (Kosary et al. 1995; NCI 1996b). When the ratios of African American to white incidence and death rates exceed 1.0 in Figure 4, then African Americans

Hawaiian	Japanese	Korean	Vietnamese	White	Hispanic[†]
340	322	266	326	469	319
321	241	180	273	346	243
239	133	NA	NA	213	129
168	88	NA	NA	140	85
9.3	5.8	15.2	43.0	8.7	16.2
–	1.5	NA	NA	2.5	3.4
9.4	5.6	–	–	5.4	4.4
–	–	–	–	1.7	0.9
–	4.8	NA	NA	5.3	3.4
–	0.9	NA	NA	1.2	0.7
9.8	7.3	6.3	–	11.9	10.0
–	2.3	–	–	5.9	5.5
–	2.4	NA	NA	5.0	3.7
–	0.8	NA	NA	2.3	1.7
–	2.5	–	–	7.5	5.1
–	–	–	–	1.5	0.7
–	–	NA	NA	2.3	1.9
–	–	NA	NA	0.5	0.2
89.0	43.0	53.2	70.9	76.0	41.8
43.1	15.2	16.0	31.2	41.5	19.5
88.9	32.4	NA	NA	72.6	32.4
44.1	12.9	NA	NA	31.9	10.8

[△]Estimates for all cancer sites are rounded to the nearest integer.
[¶]National Center for Health Statistics, public use data tapes, 1988–1992, is the source for all death rates in this table. Death rates are U.S. mortality rates.
**A dash means that the rate was not calculated for fewer than 25 cases.
NA = data not available.
Source: National Cancer Institute 1996b; National Center for Health Statistics, public use data tapes, 1988–1992.

Table 6. Continued

Primary cancer site (*ICD-9* code)†	African American	Alaska Native	American Indian (New Mexico)	Chinese	Filipino
Oral cavity excluding nasopharynx (140.0–146.9; 148.0–149.9)					
Incidence rate,§ men	20.4△	–**	–	5.3	5.4
Incidence rate, women	5.8	–	–	2.3	5.3
Death rate, men	8.7	–	–	1.6	1.2
Death rate, women	2.1	–	–	0.7	1.3
Pancreas (157)					
Incidence rate, men	14.0	–	–	8.0	6.5
Incidence rate, women	11.5	–	–	4.9	6.0
Death rate,△ men	14.4	–	–	6.7	4.5
Death rate, women	10.4	–	–	5.1	3.5
Stomach (151)					
Incidence rate, men	17.9	27.2	–	15.7	8.5
Incidence rate, women	7.6	–	–	8.3	5.3
Death rate, men	13.6	–	–	10.5	3.6
Death rate, women	5.6	–	–	4.8	2.5
Urinary bladder (188)					
Incidence rate, men	15.2	–	–	13.0	8.3
Incidence rate, women	5.8	–	–	3.7	2.1
Death rate, men	4.8	–	–	2.0	1.2
Death rate, women	2.4	–	–	1.0	–

*Rates per 100,000, age-adjusted to the 1970 U.S. standard population.
†U.S. Department of Health and Human Services 1989a.
‡Includes persons of other ethnic groups who designated themselves as of Hispanic origin.
§All incidence data are from five states: Connecticut, Hawaii, Iowa, New Mexico, and Utah; from six metropolitan areas: Atlanta (including 10 rural counties), Detroit, Los Angeles, San Francisco/Oakland, San Jose/Monterey, and Seattle/Puget Sound; and from the Alaska Area Native Health Service.

experience excess morbidity and mortality from the cancers shown. Also, SEER data for 1988–1992 show that whites have higher rates of some cancers than Hispanics, Asian Americans, Pacific Islanders, American Indians, and Alaska Natives (Table 6) (NCI 1996b). U.S. mortality data for 1984–1988 show that American Indians have a lower mortality rate from lung cancer than the general U.S. population but a higher mortality rate from cervical cancer (Table 7) (Valway 1992).

Cervical Cancer

In a case-control Los Angeles County study of invasive cervical cancer that included 98 English-speaking case-control pairs and 102 Spanish-speaking pairs, Peters and colleagues (1986) found that the overall risk of such cancer was increased by cigarette smoking. The cervical cancer risk related to smoking was comparable in the two groups. In a more recent study of the risk factors for cervical dysplasia among Hispanic and white women in New Mexico (Becker et al. 1994a,b), cigarette smoking was significantly associated with high-grade cervical dysplasia among white women but not among Hispanic women; however, this difference in risk was not statistically significant. In addition, in a recent pilot study of American Indian women in the Albuquerque IHS area, Becker and colleagues (1993) found that cigarette smoking was associated with cervical dysplasia; however, the results were not statistically significant.