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

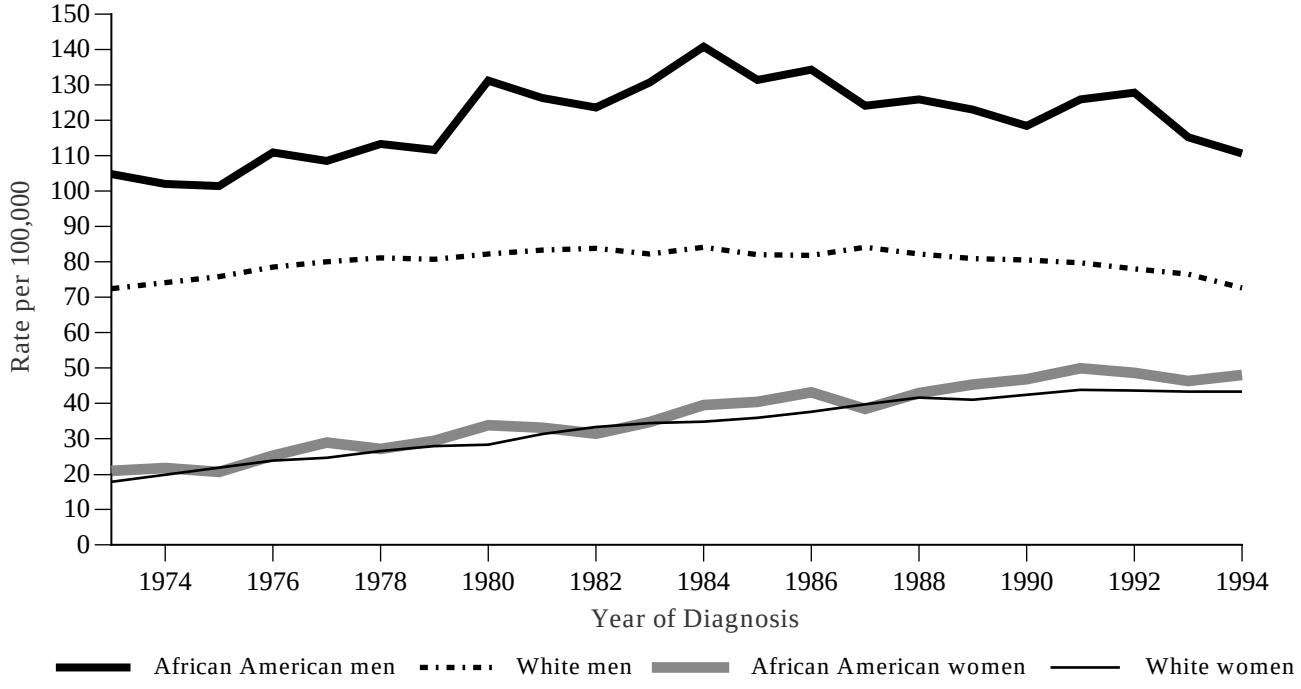
1992; Coultas 1994) and indoor and outdoor air pollutants (Samet 1993), also cause lung cancer. For example, synergism between smoking and radon and asbestos has been demonstrated in studies of worker groups (Saracci and Boffetta 1994).

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).

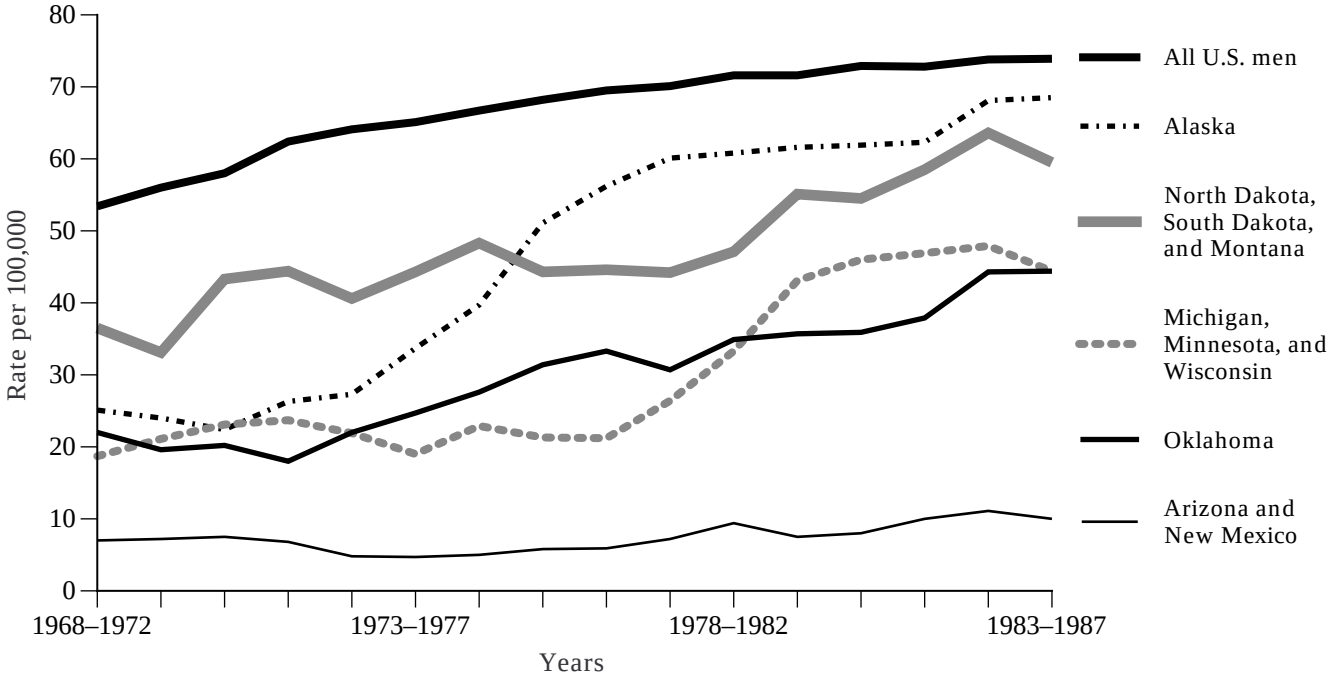
In summary, the higher rates of lung cancer observed among African American men are consistent with historical patterns of cigarette smoking in this century (Shopland 1995). In addition, African American men aged 40–54 years may be especially susceptible to lung carcinogens (Schwartz and Swanson 1997), 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*

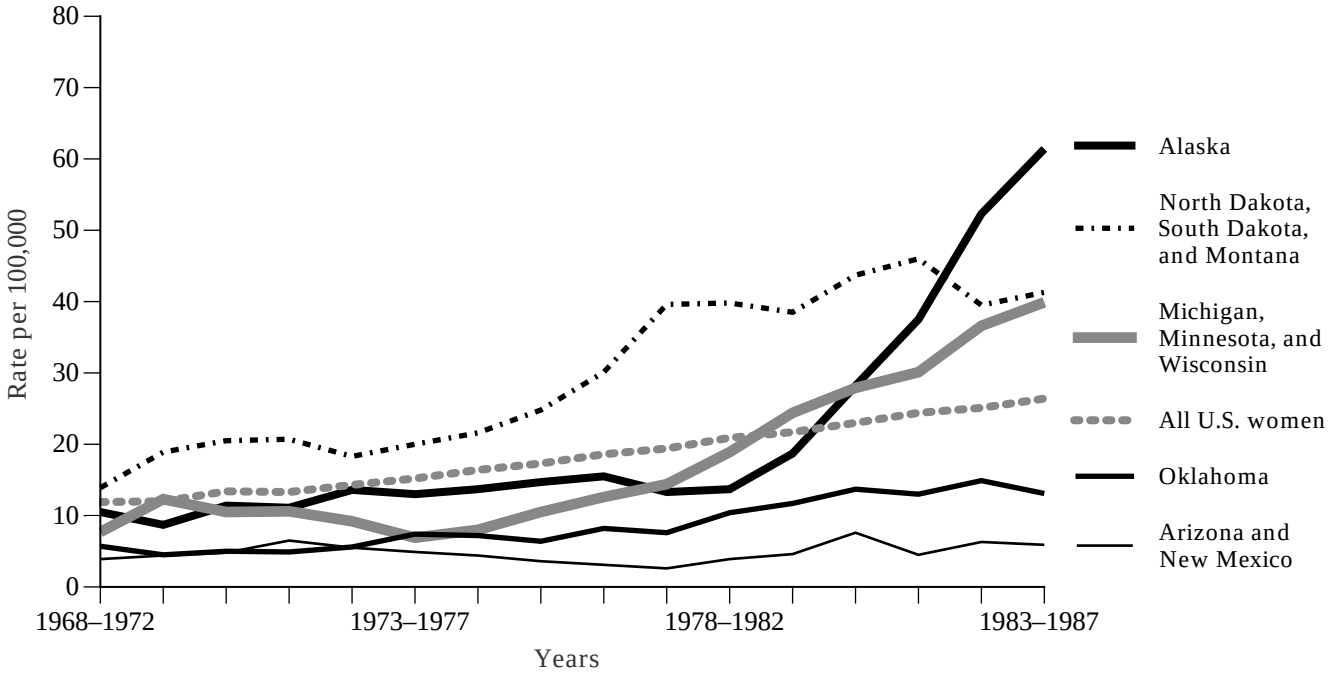


*Rates presented here were determined using midpoint population estimates for each 5-year time interval and were adjusted to the 1970 U.S. standard population.
Source: Valway 1992.

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

	Men		Women	
Areas	**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	560Δ	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

[Δ]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[Δ]	–**	–	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,[Δ] 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.

Hawaiian	Japanese	Korean	Vietnamese	White	Hispanic‡
11.7	7.0	–	11.6	14.6	8.9
–	3.3	–	–	5.8	2.7
–	2.1	NA	NA	3.8	2.7
–	0.8	NA	NA	1.5	0.7
10.9	8.7	–	–	9.8	8.0
8.7	7.3	7.6	–	7.4	6.9
12.8	8.5	NA	NA	9.7	7.1
9.1	6.7	NA	NA	6.9	5.2
20.5	30.5	48.9	25.8	10.2	15.3
13.0	15.3	19.1	25.8	4.4	8.0
14.4	17.4	NA	NA	6.1	8.4
12.8	9.3	NA	NA	2.8	4.2
–	13.7	10.4	–	31.7	15.8
–	4.1	–	–	7.8	4.3
–	2.0	NA	NA	5.8	2.8
–	1.2	NA	NA	1.7	0.9

[Δ]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.

Esophageal Cancer

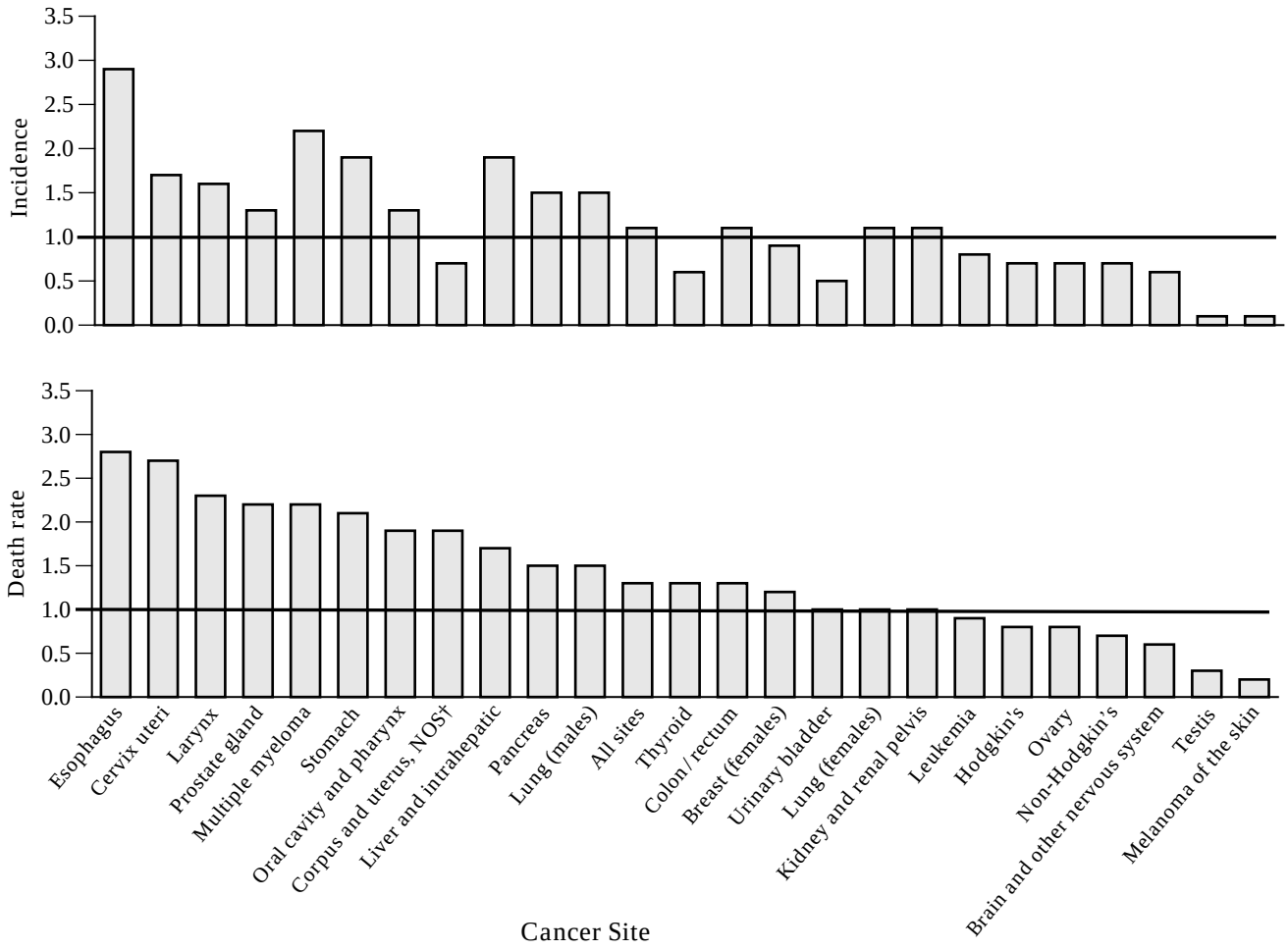
Esophageal cancer incidence and death rates in the United States are highest among African Americans (Tables 2 and 6) (NCI 1996b). To assess potential causes of the high rates of death from esophageal cancer found among African American men, Pottern and colleagues (1981) conducted a case-control study in Washington, D.C. After adjusting the data for alcohol consumption, they found that the relative risk of esophageal cancer among smokers was only marginally higher than among nonsmokers. In a more recent study, the risk for African American men of developing squamous cell carcinoma of the esophagus was significantly elevated for smokers, even after adjusting statistically for age, geographic area, alcohol consumption, and income (Brown et al. 1994).

Smoking mentholated cigarettes may also be a cause of the high and rising esophageal cancer rates among African Americans. In a case-control study of data from the American Health Foundation's ongoing tobacco study, Hebert and Kabat (1989) failed to show a consistent effect of smoking mentholated cigarettes on the risk of esophageal cancer among African Americans. Better designed studies are needed to adequately address this hypothesis.

Oral Cancer

Tobacco use and alcohol use are the predominant risk factors for cancers of the oral cavity and pharynx (commonly referred to as oral cancer) (USDHHS 1989b). African Americans have the highest oral

Figure 4. SEER* cancer incidence and U.S. death rates, 1988–1992, ratio of African American rate to white rate for all ages, by cancer site



*National Cancer Institute's Surveillance, Epidemiology, and End Results Program; rates are age-adjusted to the 1970 U.S. standard population.
†Not otherwise specified.
Source: Kosary et al. 1995.

cancer incidence and death rates in the United States (Tables 2 and 6) (NCI 1996b). Using underlying cause-of-death data compiled by NCHS and U.S. census population enumerations and intercensal population estimates, investigators found that from 1950 to 1990, the death rate for cancers of the oral cavity and pharynx (age-adjusted to the 1970 age distribution of the U.S. population) decreased for white men from 6.6 to 4.2 per 100,000 population. However, for African American men, the death rate increased from 4.8 in 1950 to 11.0 in 1980 and subsequently decreased slightly, to 9.8 in 1990. From 1980 through 1990, the rate for African American men was approximately twice as high as that for white men. The death rate for cancers of the oral cavity and pharynx for African American women exceeded the rate for white women for nearly all of the 41-year period. The death rate increased slightly for white women, from 1.5 to 1.6,

Table 7. Age-adjusted cancer death rates among American Indians and Alaska Natives at all 12 Indian Health Service areas, United States, 1984–1988

	Men			Women		
	All 12 areas			All 12 areas		
Primary cancer site	**N**	**Rate**	**U.S. rate***	**N**	**Rate**	**U.S. rate***
Oral cavity and pharynx	48	3.2	5.0	20	1.4	1.7
Digestive system						
Esophagus	41	3.0	5.8	16	1.2	1.5
Stomach	129	9.1	7.3	93	6.3	3.3
Respiratory system						
Larynx	15	1.1	2.6	5	0.3	0.5
Lung and bronchus	562	40.1†	74.2	296	21.4	27.3
Cervix uteri				126	7.6†	3.1
Urinary system						
Urinary bladder	18	1.3	5.8	12	0.9	1.7
Kidney and renal pelvis	80	5.6	4.8	44	3.2	2.2

Note: Rates per 100,000, age-adjusted to the 1970 U.S. standard population.
*1984–1988 U.S. cancer mortality rates for all races.
†Denotes a rate significantly different from the U.S. rate.
Source: Valway 1992.

and increased for African American women from 1.9 to 2.2 (CDC 1993a).

The risks associated with smoking were similar for 194 African Americans and 871 whites participating in a 1984–1985 population-based, case-control study to assess tobacco use, alcohol use, and other risk factors for oral cancer in New Jersey, Atlanta, and two areas of California (Table 8) (Day et al. 1993). Calculations of attributable risks showed that the higher incidence of oral cancer among African Americans could be largely explained by tobacco and alcohol use. A case-control study of oral cancer among North Carolina women in 1975–1978 indicated a similar risk associated with smokeless tobacco use among African Americans and whites (Winn et al. 1981). Unfortunately, little information is available on the effects of smokeless tobacco use on oral cancer among members of the other racial/ethnic groups, even though the use of smokeless tobacco is a cause of oral cancer (USDHHS 1986b, 1989b).

Stomach Cancer

The incidence of stomach cancer in the United States is especially high in Asian Americans and Alaska Native men and intermediate for African Americans (Table 6) (NCI 1996b). The incidence of stomach cancer for persons of Japanese ancestry living in Hawaii is lower than for their counterparts in Japan and is increased by cigarette smoking (particularly for those who initiated at younger ages) (Nomura et al. 1995). In a case-control study conducted in south Louisiana, the risk of stomach cancer in African Americans was higher among smokers than among nonsmokers; in whites, the risk of stomach cancer was only slightly higher among smokers than among nonsmokers (Correa et al. 1985). In a more recent study, significant increases in stomach cancer were observed for African men and women who had ever smoked (Burns et al. 1995).

Table 8. Odds ratios (ORs) and 95% confidence intervals (CIs) for the risk of oral cancer associated with cigarette smoking, by race/ethnicity and smoking status, 1984–1985*

	African American		White	
Smoking status	**OR†**	**±CI**	**OR†**	**±CI**
Never smoked	1.0		1.0	
No. of cigarettes per day‡				
1–19	1.2	0.5–2.6	1.2	0.8–1.7
20–39	2.1	1.0–4.4	2.2	1.6–2.9
≥40	2.8	1.0–7.7	2.8	2.0–4.0
Years of cigarette smoking				
1–19	0.9	0.3–2.4	0.6	0.4–1.0
20–39	1.6	0.7–3.3	1.9	1.3–2.5
≥40	2.9	1.2–7.2	3.3	2.3–4.6
Age at smoking initiation (years)				
<17	1.8	0.8–3.9	2.0	1.4–2.7
17–24	1.7	0.8–3.8	1.9	1.4–2.6
≥25	1.2	0.4–3.6	2.2	1.4–3.5
Years since stopped smoking				
0 (never quit)	2.3	1.1–4.7	3.6	2.6–4.8
1–9	1.1	0.4–3.1	1.1	0.7–1.6
10–19	0.1	0.0–1.3	1.1	0.7–1.6
≥20	0.3	0.1–1.7	0.6	0.3–0.9

*Data from four population-based cancer registries in Los Angeles County and Santa Clara and San Mateo Counties near San Francisco-Oakland, metropolitan Atlanta, and the state of New Jersey.

†ORs are adjusted for alcohol consumption, gender, age, study location, and respondent status and are relative to persons who never smoked.

‡Usual number of cigarettes smoked daily when the persons smoked.

Source: Day et al. 1993.

Urinary Bladder Cancer

The incidence of urinary bladder cancer in the United States is highest for whites (Table 6) (NCI 1996b). Among men, mortality is highest for whites; among women, mortality is highest for African Americans (Tables 2 and 6) (NCI 1996b). Differences in bladder cancer risk associated with cigarette smoking for African Americans and whites have been examined in several case-control studies (Table 9), including the ongoing study conducted by the American Health Foundation (Harris et al. 1990), a population-based study conducted in the Detroit metropolitan area (Burns and Swanson 1991), and a population-based study carried out through SEER registries in 1978 (Hartge et al. 1993). In the American Health Foundation study, investigators found that although cigarette smoking was a significant risk factor for bladder cancer among both whites and African Americans, the data suggested a steeper exposure-response relationship among whites (with significant increased risk beginning at exposures of 20 pack-years) than among African American men (with increased risk beginning only after 60 pack-years). However, in a multivariate analysis of the data for men, the risk of bladder cancer did not differ by race. The other two studies showed similar findings for both whites and African Americans in the association between cigarette smoking and bladder cancer. In a smaller case-control study in Orange County, California, no significant interactions were found between smoking and race/ethnicity among whites, Hispanics, Asian Americans, or Pacific Islanders (Anton-Culver et al. 1993). Thus, information currently available suggests that smoking increases the risk of bladder cancer in a similar fashion among both whites and African Americans. In a cohort study of 7,995 Japanese American men who were living in Hawaii, the risk of bladder cancer was 2.9 times higher in current smokers than in nonsmokers (Chyou et al. 1993).

Aromatic amines, such as 4-aminobiphenyl, are considered causative chemical agents in cigarette smoke-induced bladder cancer (Bartsch et al. 1993). As with other potential carcinogens in tobacco smoke, aromatic amines require metabolic activation before interacting with DNA (Miller and Miller 1981). A competing chemical pathway (i.e., acetylation) exists and serves as a detoxification mechanism. Genotyping studies have characterized several variant alleles of the N-acetyltransferase gene, which can result in different rates of chemical acetylation. People who are slow acetylators have increased risk for bladder cancer (Hein 1988). Bell and colleagues (1993) determined

Table 9. Odds ratios for the risk of urinary bladder cancer associated with smoking, by gender, race/ethnicity, and smoking status

Reference, study type, and year	Smoking status	Men		Women	
		African American	White	African American	White
Harris et al. 1990	Never	1.0	1.0	1.0	1.0
Multicenter, hospital-based,	Former	1.6	2.1		1.3
1973–1985	Current	2.0	3.2	3.9*	3.2
Burns and Swanson 1991	Never	1.0	1.0	1.0	1.0
Detroit, population-based	Ever	3.0	2.3	3.8	2.4
	Pack-years				
	< 30	1.9	1.5	3.1	1.7
	30–59.9	4.0	2.6	3.8	2.9
	60–89.9	4.7	2.7	5.0	3.5
	> 90	4.8	3.0	5.2	2.7
Hartge et al. 1993	Never	1.0	1.0	1.0	1.0
SEER† registries,	Former				
population-based, 1978	Cigarettes smoked				
	< 20 per day	1.6	1.3	3.6	2.0
	≥ 20 per day	1.8	1.9	5.0	1.3
	Current				
	Cigarettes smoked				
	< 20 per day	2.2	2.1	1.7	2.0
	≥ 20 per day	4.5	3.0	2.1	3.1

*Ever smokers.
†National Cancer Institute's Surveillance, Epidemiology, and End Results Program.

that 41 percent of African Americans and 55 percent of whites were slow acetylators. A phenotyping study also found the highest percentage of slow acetylators among whites (54 percent), compared with African Americans (34 percent) and Asians (14 percent) (Yu et al. 1994).

In the 1994 study by Yu and colleagues, slow acetylators had higher levels of 3- and 4-aminobiphenyl-hemoglobin adducts, regardless of race and level of smoking (Yu et al. 1994). For African Americans, Asians, and whites, however, the levels of 3- and 4-aminobiphenyl-hemoglobin adducts increased proportionately more for cigarette smokers compared with nonsmokers than for slow acetylators compared to rapid acetylators. In a subsequent study by Yu and colleagues (1995), the slow acetylation phenotype combined with the null genotype of the gene *(GSTM1)* for a phase II detoxification enzyme (glutathione S-transferase) resulted in higher levels of 3- and 4-aminobiphenyl-hemoglobin adducts than did lower risk profiles (i.e., rapid acetylator and / or at least one functional *GSTM1* gene allele). The highest risk profile was seen in 27 percent of whites, 15 percent of African Americans, and 3 percent of Asians.

Several studies show that the highest levels of risk are experienced by smokers, because high levels of exposure to tobacco smoke overwhelm the various phenotypic traits. The differences in risks for various detoxification and activation pathways appear to be most significant among persons who did not smoke or who smoked at very low levels (Yu et al. 1994, 1995; Landi et al. 1996).

Chronic Obstructive Pulmonary Disease

In addition to causing lung cancer, tobacco smoking also causes several non-malignant diseases of the lung and increases the frequency of respiratory symptoms and illnesses (USDHHS 1989b, 1990). Chronic obstructive pulmonary disease (COPD) is a clinical term applied to persons with a permanent airflow obstruction associated with significant impairment (Samet 1989; USDHHS 1989b). Cigarette smokers with COPD have impaired breathing as a result of emphysema (air space enlargement and destruction) and damage to the airways (USDHHS 1984). These smokers also may have chronic bronchitis, which is the term used by epidemiologists and clinicians for chronic sputum production.

Longitudinal studies show that the development of COPD follows sustained excessive loss of ventilatory function of the lung caused by cigarette smoking (USDHHS 1984, 1990). The rate at which ventilatory function declines tends to increase with the amount smoked and to revert to the rate associated with aging after smoking cessation (USDHHS 1990). The frequency of chronic bronchitis is similarly related to smoking pattern.

African Americans

Data from several national surveys have been used to compare the prevalence of COPD among African Americans and whites. McWhorter and colleagues (1989) used data from the 1971–1975 National Health and Nutrition Examination Survey (NHANES I) and the 1982–1984 NHANES I Epidemiologic Follow-up Study (NHEFS) to determine the prevalence of COPD among 14,404 adults aged 25–74 years. African American race/ethnicity was associated with a lower risk for having COPD; 6.2 percent of whites and 3.2 percent of African Americans had COPD.

In the 1990 NHIS, the prevalence of self-reported chronic bronchitis was 55.2 per 1,000 African Americans aged 45–64 years and 42.7 per 1,000 African Americans aged 65 years and older (USDHHS 1991). The prevalence of self-reported emphysema was 3.6 per 1,000 middle-aged African Americans and 41.5 per 1,000 older African Americans. Compared with African Americans, whites in both age groups reported higher prevalences of chronic bronchitis (59.7 for those aged 45–64 years and 73.8 for those aged 65 years and older) and emphysema (13.8 for those aged 45–64 years and 46.1 for those aged 65 years and older). However, self-reports of chronic bronchitis and emphysema, without further validation, are probably subject to substantial misclassification.

African Americans are also less likely than whites to die of COPD (Evans et al. 1987; NCHS 1991). Evans and colleagues (1987) found that in 1982, the age-adjusted COPD death rate was 16.6 per 100,000 whites and 12.8 per 100,000 African Americans. Data for 1986–1988 also show lower death rates from COPD among African Americans than among whites (Desenclos and Hahn 1992). More recent data (Table 2) show that African American men have higher death rates (17.6) for chronic airway obstruction than men in the other three racial/ethnic minority groups, although their rates are lower than rates among white men (20.4). The same pattern is also evident for deaths due to bronchitis and emphysema. The rate of COPD mortality is unexpectedly low among African Americans, given their high prevalence of smoking and related high lung cancer rates. The reasons for this discrepancy remain to be explored. However, whites are more likely than African Americans to have ever smoked and to be former smokers (see Table 37 in Chapter 2). Mannino and colleagues (1997) have observed that death rates from obstructive lung disease relate to rates of ever smoking. These authors suggest that the differences in the race- and gender-specific relative rankings for obstructive lung disease and lung cancer may be because long-term former smokers are more likely to develop obstructive lung disease than lung cancer.

American Indians and Alaska Natives

Little information is available on the occurrence of COPD among American Indians and Alaska Natives. In a 1987 survey of approximately 6,500 American Indians and Alaska Natives aged 19 years and older, 2.4 percent of men and 1.4 percent of women reported having emphysema, compared with 2.7 percent of men and 2.3 percent of women in the general U.S. population (Johnson and Taylor 1991). Rhoades (1990) studied hospitalization and death rates for COPD in American Indians and Alaska Natives. Although the death rates for COPD were lower than from other competing causes, such as chronic liver disease, diabetes, and injuries, the hospitalization rates for COPD exceeded those for cancer and tuberculosis.

Additionally, hospitalization rates and death rates for COPD varied widely between geographic regions. The contribution of COPD to hospitalization rates ranged from 1.6 percent in the Navajo IHS area to 5.1 percent in the Bemidji area; COPD death rates per 100,000 ranged from 1.7 in the Albuquerque area to 10.3 in the Billings area (Rhoades 1990).

Between 1992 and 1994, COPD death rates among American Indian men were approximately two-thirds the rates among whites (Table 2). Data from the Alaska area indicate that from 1979 through 1986, COPD death rates per 100,000 were 31.6 for Alaska Native men, compared with 40.3 for white men in Alaska and 38.3 for men in the United States as a whole (Coultas et al. 1994). The COPD death rates per 100,000 were 22.3 for Alaska Native women, compared with 34.8 for white women in Alaska and 18.6 for women in the United States as a whole. Similarly, death rates for COPD in New Mexico (Samet et al. 1988b) reflect the nationwide pattern of lower rates of death among American Indians compared with whites and are consistent with the lower smoking prevalence among tribes in the southwestern United States (Sugarman et al. 1992). The high rates of COPD among Alaska Natives are probably related to the fact that rates of smoking among Alaska Natives are higher than rates among American Indians elsewhere, particularly in the Southwest.

Asian Americans and Pacific Islanders

Information on COPD morbidity and death among Asian Americans and Pacific Islanders is sparse. National mortality data indicate that the prevalence of deaths from bronchitis and emphysema is lower in this group than among African Americans and whites (Table 2); the death rate from chronic airways obstruction is lowest for Asian Americans and Pacific Islanders. Data from California show that from 1986 through 1987, the overall prevalence of COPD deaths among "Asian and other" persons was lower than among whites but varied widely for specific Asian American and Pacific Islander subgroups (Asian American Health Forum, Inc. 1990).

One of the oldest studies of Asian Americans—the Honolulu Heart Study, conducted in 1965—provides valuable age-related information on smoking and lung function among Japanese Americans. Of the 6,346 Japanese American men aged 46–68 years who underwent spirometric testing, 48 percent were current cigarette smokers, 25 percent were former smokers, and 27 percent had never smoked (Marcus et al. 1988). Airflow obstruction was found in 11.7 percent of the participants. The prevalence of airflow obstruction increased with age and with the amount smoked. For most age and smoking categories, the prevalence of airflow obstruction was lower among Japanese American men than among white men from Connecticut participating in the same study (Beck et al. 1981).

In another recent analysis of data from the Honolulu Heart Program, Japanese American men who continued to smoke showed steeper rates of decline in forced expiratory volume after one second (FEV_1), a measure of pulmonary function, compared with never smokers. Among continuing smokers, FEV_1 decline was significantly associated with duration of smoking. Additionally, the rate of decline in FEV_1 among former smokers became more like that of persons who had never smoked (Burchfiel et al. 1995), consistent with previous reports on the benefits of quitting smoking (USDHHS 1990). In another analysis of data from the same study, Sharp and colleagues (1994) found that a diet composed of large amounts of fish may protect the lungs against damage from cigarette smoking. However, fish consumption was not associated with pulmonary function at higher levels of cigarette smoking (>30 cigarettes / day).

Hispanics

In the 1982–1984 Hispanic Health and Nutrition Examination Survey (HHANES), Puerto Ricans (2.9 percent) had a higher prevalence of reported chronic bronchitis than Mexican Americans (1.7 percent) or Cuban Americans (1.7 percent) (Bang et al. 1990). Chronic airflow obstruction (assessed using spirometry) was present in less than 1 percent of Hispanic adults surveyed in a New Mexico community (Samet et al. 1988a). Similarly, investigators who surveyed Mexican Americans in Tucson, Arizona, found a relatively low prevalence of physician-diagnosed COPD or related diagnoses (Di Pede et al. 1991).

COPD has been reported to occur less frequently among Hispanics than among whites. Surveys in New Mexico have shown, for example, that physician-diagnosed chronic bronchitis or emphysema is less common among Hispanics than among whites (Samet et al. 1982, 1988a). Death rates from chronic obstructive lung diseases and allied conditions are also lower among Hispanics than among whites (Tables 2 and 4). Mortality data for New Mexico indicate that between 1958 and 1982, Hispanic men had a lower death rate from COPD than white men; however, from 1958 through 1982, the death rate from COPD rose

steeply among Hispanic men—from 5.0 per 100,000 in 1958–1962 to 30.1 per 100,000 in 1978–1982 (Samet et al. 1988b). During this same time, COPD death rates increased among Hispanic women but remained comparable to rates among white women (Samet et al. 1988b).

Little information is available on the risk of COPD among Hispanic smokers. In a 1979 respiratory disease survey of Hispanic and white residents of New Mexico's Bernalillo County, Samet and colleagues (1982) found that race/ethnicity was not a significant predictor of current or previous physician-diagnosed chronic bronchitis and emphysema and that no significant interaction existed between race/ethnicity and cigarette smoking. Hispanic ethnicity also was not a significant predictor of the symptoms of chronic cough, chronic phlegm, or persistent wheeze. Similarly, the results of a survey of Hispanics and whites in Tucson indicated that race/ethnicity was not a significant determinant of respiratory symptoms, after survey data were adjusted for cigarette smoking (Di Pede et al. 1991). However, a recent cross-sectional study of urban pregnant women indicated that the prevalence of either doctor-diagnosed asthma or persistent wheeze without asthma was lower among a heterogenous Hispanic population than among white women of similar socioeconomic background (these data were adjusted for cigarette smoking status, family history of asthma, educational level, household exposure to pets, and level of lung function). The authors did not conclude that their data provided evidence of biological protection from wheeze syndromes. An almost fivefold excess risk of persistent wheeze was detected in the total population of urban women who are current smokers (David et al. 1996).

Coronary Heart Disease

In 1994, cardiovascular diseases, comprising a diverse group of disorders including coronary heart disease (CHD), hypertension, stroke, and rheumatic heart disease, caused approximately 940,000 deaths in the United States (NCHS 1996a). The occurrence of specific cardiovascular diseases and their risk factors varies widely among the different racial/ethnic minority groups. Of the cardiovascular diseases, CHD is the single largest cause of death; it results in approximately 480,000 deaths annually in the United States. This section of the report focuses on CHD, which is also termed coronary artery disease or ischemic heart disease (IHD).

Coronary artery disease results from atherosclerosis of coronary arteries. Anatomical lesions become evident in young adults and are usually clinically manifest in the fifth through seventh decades as angina pectoris, myocardial infarction, and sudden cardiac death (Enos et al. 1986; Strong 1986). In this chapter, these clinical manifestations of coronary artery disease are collectively termed CHD.

Numerous non-modifiable and modifiable risk factors contribute to the development of CHD. The non-modifiable factors include aging, gender (men have greater risk), and family history of CHD. The major risk factors that are potentially modifiable include hypertension, cigarette smoking, obesity, hypercholesterolemia, diabetes mellitus, and physical inactivity (Smith and Pratt 1993). The 1983 Surgeon General's report on smoking and health concluded that "Cigarette smoking should be considered the most important of the known modifiable risk factors for coronary heart disease in the United States" (USDHHS 1983, p. iv).

African Americans

The first population-based epidemiological investigations of cardiovascular diseases in the United States that included substantial numbers of African American and white participants began in 1960 in Evans County, Georgia, and Charleston, South Carolina (Saunders 1991). Since 1960, follow-up data for these cohorts and a number of other epidemiological studies have provided information on the combined effects of race/ethnicity and various risk factors for cardiovascular disease. Consistent with findings for the general population, cigarette smoking increased risk of death from CHD among African Americans (Hames et al. 1993; Keil et al. 1995).

Tyroler and colleagues (1984) examined deaths from CHD among the Evans County men, who were followed from 1960 through 1980, and found that the overall rate of death from CHD was lower among African Americans than among whites, with a ratio of 0.86. For current and former smokers, the probability

of dying from all causes and from CHD was higher among whites with a low-socioeconomic status (on the basis of occupation, education, and source of income of the head of household) than among their African American counterparts. However, the analysis did not control for the number of cigarettes smoked, and the data were limited because of the small number of CHD deaths (31) among African Americans.

In the Charleston Heart Study of CHD death rates between 1960 and 1990, Keil and colleagues (1993) found that the age-adjusted, African American-to-white death rate ratios were 0.90 for men and 1.2 for women. After controlling for age and other cardiac risk factors, the researchers found that smoking was associated with a slightly higher risk of dying of CHD among African American men than among white men. White women had a slightly higher risk of dying of CHD than did African American women. These racial / ethnic group differences were not tested for statistical significance, however.

Other investigations that provide information on the risks for CHD and the modification of the effects of smoking, by race / ethnicity, include the Cancer Prevention Study I (CPS-I) (Garfinkel 1984), the NHEFS (Cooper and Ford 1992), the National Mortality Followback Survey (NMFS) (DeStefano and Newman 1993), and the ongoing study of Kaiser Permanente enrollees (Friedman et al. 1997). As part of the CPS-I, death patterns in the original cohort of one million people were described for 1959–1972. The observed-to-expected death rate ratios from CHD among African Americans and whites followed the same pattern as nationwide vital statistics described previously. Overall, the African American-to-white ratios of CHD deaths were 0.78 for men and 1.07 for women. Stratified analyses, by gender, of any effects that the amount of cigarettes smoked might have on CHD deaths showed little difference between African Americans and whites.

Participants in the NHANES I, conducted between 1971 and 1975, were reexamined between 1982 and 1984 as part of the NHEFS (Cooper and Ford 1992). Of the 12,599 participants in the follow-up survey, 10,741 were white and 1,858 were African American. The study showed that cumulative incidence rates of fatal CHD were higher among African Americans (6.2 percent of men and 3.7 percent of women) than among whites (5.6 percent of men and 2.6 percent of women). In contrast, cumulative incidence rates of nonfatal CHD were higher among whites (7.0 percent of men and 4.7 percent of women) than among African Americans (5.0 percent of men and 3.9 percent of women). The risk of new CHD events associated with cigarette smoking was similar among whites and African Americans. These results, however, are limited by the small number of new CHD events among African Americans and the low proportion (approximately 50 percent) of respondents for whom smoking information was collected at baseline.

In a case-control study of CHD deaths among African Americans and whites, DeStefano and Newman (1993) used data from the 1986 NMFS to identify case subjects (n = 803) and 1988 data from the BRFSS to identify control subjects (n = 25,398). When they compared the risk of death among smokers vs. persons who have never smoked (men aged 25–44 years and women aged 25–54 years), the investigators found that among persons without diabetes, African American smokers had a lower relative risk for CHD death than white smokers. However, the 95 percent confidence intervals associated with these odds ratios overlapped each other—an indication that the difference in risk was not statistically significant. In the Kaiser study, the risk of death from CHD has varied among African Americans and whites, but small numbers limit interpretation of these findings (Friedman et al. 1997).

American Indians and Alaska Natives

Most of the available data on CHD among American Indians and Alaska Natives have originated from studies of selected tribes, as reviewed by Young (1994). Investigations of heart disease in southwestern American Indians and Alaska Natives conducted several decades ago showed a low prevalence of CHD relative to the U.S. population and other racial / ethnic groups (Welty and Coulehan 1993). In a descriptive study of CHD deaths occurring from 1948 through 1952 among the Navajos, Smith (1957) found that the standardized death rate ratios for CHD among the Navajos compared with whites were 0.10 for men and 0.12 for women. Since then, numerous other regional investigations of CHD deaths and the incidence of CHD in other tribes of the United States and Canada have been reported. Overall, for studies conducted in the 1950s and 1960s, the ratios of CHD death rates among American Indians and Alaska Natives compared with nationwide rates have ranged from 0.1 to 0.5. An analysis of death statistics from the NCHS showed that crude CHD death rates for individuals classified as American Indians, Eskimos, or Aleuts declined from 100 per 100,000 in 1969–1971 to 67 per 100,000 for the years 1979–1981 (Gillum 1988). A review of New Mexico's vital statistics for 1958–1982 indicates that for American Indian men, CHD death

rates peaked at 101.7 per 100,000 between 1968 and 1972 and fell to 76.6 per 100,000 between 1978 and 1982 (Becker et al. 1988). For American Indian women, the CHD death rate peaked at 63.0 per 100,000 between 1963 and 1967 and declined to a low of 28.3 per 100,000 between 1978 and 1982.

In a recent analysis of mortality data for 1992–1994 (Table 2), the rate of death due to CHD was lower among American Indian and Alaska Native men (100.4) and women (45.9) than among white men (132.5) and women (62.9). The ratio of CHD death rates among American Indians and Alaska Natives compared with whites was .76 for men and .73 for women. The fact that these ratios are higher than ratios from earlier studies suggests that CHD deaths among American Indians and Alaska Natives may be increasing (Welty and Coulehan 1993; Young 1994).

Risk factors for cardiovascular disease were investigated recently in a large multi-tribal study of American Indians. The results showed that mean levels of total, low density lipoprotein, and high density lipoprotein cholesterol were lower in American Indians than in the U.S. general population. Prevalence of hypertension, non-insulin dependent diabetes mellitus, and obesity were very high, but varied considerably among tribes and geographic regions (Welty et al. 1995). A second study found that levels of serum cholesterol were lower in American Indian smokers who attended a stop smoking clinic than in African American and white smokers from population-based samples (Folsom et al. 1993). However, fibrinogen levels and the prevalence of abdominal obesity were higher in American Indian smokers than in African Americans and whites.

The IHS is another source of nationwide and regional health statistics on CHD deaths. Because the mortality data in IHS reports combine all cardiovascular diseases under "diseases of the heart" (IHS 1994b), this information cannot be compared directly with CHD data from other sources. Between 1989 and 1991, diseases of the heart accounted for 21.9 percent of deaths in all IHS areas, with a crude death rate of 115.1 per 100,000 (IHS 1994b). These data indicate cardiovascular diseases were the leading cause of death among American Indians. However, because Indian race / ethnicity was underreported on death certificates in several IHS areas, including California and Oklahoma as well as Portland, Oregon, this death rate may be incorrect.

Death rates from heart diseases vary widely among people in the 12 IHS areas. From 1989 through 1991, the rate of death from heart diseases per 100,000 was lowest in the Albuquerque area (88.0) and highest in the Aberdeen area (249.0) (IHS 1994a). These wide variations in deaths from diseases of the heart parallel the wide variations in the prevalence of cigarette smoking among the various tribes (Sugarman et al. 1992; Coultas et al. 1994) (see also Chapter 2). For example, in a 1985–1988 survey of adult American Indians in the southwestern United States, 18.1 percent of men and 14.7 percent of women reported current smoking, compared with 48.4 percent of men and 57.3 percent of women in the Plains states (Sugarman et al. 1992).

Data to assess the influence of tobacco use on the risk of cardiovascular disease among American Indians are extremely limited. One study has shown that cigarette smoking increases the risk for CHD among American Indians, after adjustment for other risk factors (Howard et al. 1995). In fact, most studies presented in this section describe cardiovascular disease morbidity and mortality without ever assessing the influence of tobacco use. Nevertheless, cardiovascular disease is the leading cause of death among American Indians and Alaska Natives (NCHS 1996b), and tobacco use is an important risk factor for this disease. More studies are needed to evaluate the independent effect of tobacco use on the risk of cardiovascular disease among American Indians and Alaska Natives.

Asian Americans and Pacific Islanders

Limited data are available on risk factors and CHD among Asian Americans and Pacific Islanders in the United States (Yu 1991). A recent study of nationwide mortality indicated that Asian Americans and Pacific Islanders have lower rates of death from CHD than whites (Table 2).

In an analysis of 1980 death rates in Los Angeles County, Frerichs and colleagues (1984) found that the age- and gender-adjusted death rates for cardiovascular diseases varied widely among Asian Americans and Pacific Islanders. Koreans had the lowest rate per 100,000 (82), and Japanese had the highest rate (162). These rates were substantially lower than the overall rate for the county population, with rate ratios of 0.26 for Koreans and 0.52 for Japanese. Specific data on CHD deaths and cigarette smoking prevalence were not available.

In another study, Reed and colleagues (1983) used death records from Hawaii to describe age-adjusted, gender-specific, and racial- and ethnic-specific rates of CHD deaths occurring from 1940 through 1978. For all racial / ethnic minority groups, CHD death rates were higher among men than among women. Death

rates and the temporal trends in deaths varied widely between the different groups, with the highest death rates among Native Hawaiians and the lowest among Japanese. Filipino men had the greatest increase in CHD death rates, surpassing the rates for whites in 1978. Although most of the other groups had declines in CHD death rates between 1960 and 1970, CHD death rates for Native Hawaiian men remained level.

In 1965, three cohorts of Japanese men were assembled in Japan, Honolulu, and San Francisco to investigate the differences in CHD deaths observed among Japanese men living in the three locales (Worth et al. 1975; Yano et al. 1988). From 1965 through 1972, Worth and colleagues (1975) found that age-specific death rates were highest among the San Francisco men, intermediate among those living in Honolulu, and lowest among those living in Japan. For example, among men 60–64 years of age, the annual CHD death rates per 1,000 were 4.9 in San Francisco, 3.9 in Honolulu, and 2.1 in Japan. Mortality data for 1965–1980 indicate that the age-adjusted CHD death rate ratio for men in Honolulu compared with men in Japan was 1.4 (Yano et al. 1988). The age-adjusted mean levels of most CHD risk factors, including cigarette smoking (measured in cigarette-years), were also higher among Honolulu men. After adjusting for these risk factors, the rate ratio for CHD declined to 1.17, indicating that more than half of the elevated CHD death rate was due to the higher mean levels of CHD risk factors among Honolulu men.

In the Honolulu Heart Program cohort, composed of 7,705 Japanese men 45–68 years of age living in Hawaii who had no evidence of CHD at enrollment between 1965 and 1968, numerous analyses were conducted to further examine predictors of CHD incidence and death (Reed et al. 1982, 1987; Yano et al. 1984; Benfante et al. 1991). A higher level of acculturation was found to be associated with CHD risk factors and incidence during the 1971–1979 follow-up (Reed et al. 1982). Men who were primarily Japanese in culture smoked an average of seven cigarettes per day, whereas men who were more acculturated smoked an average of 11 cigarettes per day. A similar pattern was seen for total CHD incidence, which was highest among the men who were more acculturated (62 per 1,000) and lowest among the men who were primarily Japanese in culture (35 per 1,000).

Yano and coworkers (1984) conducted detailed analyses of the relationship between risk factors and the incidence of CHD during a 10-year period, beginning after the enrollment period (1965–1968). Systolic blood pressure, number of cigarettes smoked, and cholesterol level were all independently associated with the occurrence of all CHD events. Alcohol consumption was found to be a protective factor. Subsequent analyses of 20-year follow-up data from the same study showed that cigarette smoking was independently associated, in a dose-response manner, with increased risk of CHD (fatal or nonfatal) and aortic aneurysm (Goldberg et al. 1995). The risk for angina was elevated in persons who smoked more than 20 cigarettes per day. Another analysis suggested that high levels of fish intake might limit the increased risk among heavy smokers, although these findings should be considered preliminary (Rodriguez et al. 1996). In addition, cigarette smoking was found to be independently associated with increased prevalence of myocardial lesions in Japanese men with minimal evidence of coronary atherosclerosis at autopsy (Burchfiel et al. 1996).

Hispanics

Because of incomplete data, the NCHS reported data from 1985 death certificates on decedents of Hispanic origin for only 17 states and the District of Columbia (NCHS 1996b). By 1990, data for 47 states and the District of Columbia were reported. The NCHS estimated that the 1990 reporting area encompassed 99.6 percent of the U.S. Hispanic population (NCHS 1996b). In 1993 and 1994, only Oklahoma did not provide information on Hispanic origin (NCHS 1996a,b).

Between 1992 and 1994, the overall rate of death from CHD in the United States was lower among Hispanics than among whites (Table 2). Among the various Hispanic subgroups, Puerto Rican men had the highest death rates per 100,000 (118.6); similarly, CHD death rates among Puerto Rican women (67.3) were higher than among Mexican (44.2) and Hispanic (42.4) women.

Nationwide death rates among Hispanics and whites have been estimated by using data collected by the U.S. Bureau of the Census as part of the Current Population Survey (CPS) (Sorlie et al. 1993). Baseline interview data were obtained between 1973 and 1985 from approximately 40,000 Hispanics and 660,000 non-Hispanics aged 25 years and older. Death rates for these two groups were ascertained up to nine years after the initial interview through the National Death Index. Age-adjusted death rate ratios for CHD were lower among Hispanics than among non-Hispanics (0.60 for men and 0.75 for women). Further details for the different Hispanic subgroups were not provided.

In addition to nationwide data on the occurrence of CHD among Hispanics, regional studies have been conducted in California (Schoen and Nelson 1981;

Frerichs et al. 1984), Colorado (Rewers et al. 1993), New Mexico (Buechley et al. 1979; Becker et al. 1988), and Texas (Stern and Gaskill 1978; Stern et al. 1987; Mitchell et al. 1991; Goff et al. 1993). In general, these investigations have consistently shown that Hispanic men have lower CHD death rates than white men, although the Colorado study found little evidence for lower CHD death rates among Hispanics without diabetes (Rewers et al. 1993).

The prevalence of angina was also found to be lower among Hispanics than among whites in a review of data from a sample of Mexican Americans participating in the 1982–1984 HHANES and of whites surveyed in the 1976–1980 NHANES II (LaCroix et al. 1989). Prevalence rates based on self-reports were 2.8 percent among Mexican American men and 3.9 percent among white men, and they were 5.4 percent among Mexican American women and 6.3 percent among white women. As with African Americans, no significant differences were observed in the distribution of cardiovascular disease risk factors among Mexican Americans with and without self-reported angina. The results of this survey were limited by the lack of smoking-specific analyses for Mexican Americans.

Several investigators also have examined the cardiovascular disease risk factor profiles of Hispanics (Mitchell et al. 1991; Shea et al. 1991; Winkleby et al. 1993). Shea and colleagues (1991) analyzed 1989 BRFSS data on 636 Hispanics, most of whom were Puerto Ricans, Dominicans, and Cubans living in New York City. Although the overall risk factor profile was high among these Hispanic subgroups, the prevalence of current cigarette smoking varied by level of education. Mitchell and colleagues (1991) obtained information on cardiovascular disease risk factors from 5,148 subjects, including 3,281 Mexican Americans, who participated in the San Antonio Heart Study from 1979 through 1988. The overall risk profiles were higher among Mexican Americans. For men of all ages, the prevalence of current smoking was higher among Mexican American men (36.7 percent) than among white men (30.4 percent). For women of all ages, however, the prevalence of current smoking was lower among Mexican American women (21.0 percent) than among white women (26.8 percent). For both men and women, the number of cigarettes smoked per day was consistently lower among Mexican Americans than among whites. More recently, Winkleby and colleagues (1993) examined the cardiovascular disease risk profiles of 756 Hispanics and 756 whites participating in California surveys from 1979 through 1990. Hispanics and whites were matched by age, gender, educational level, city of residence, and time of survey. Whites had a higher prevalence of smoking (34.2 percent) than Hispanics (24.0 percent), and they smoked more cigarettes per day (19.7) than Hispanics (11.4).

Few investigators have compared the risk of smoking-related CHD between Hispanics and members of other racial/ethnic groups. Mitchell and coworkers (1991) determined the 1979–1988 prevalence of myocardial infarction among 3,281 Mexican Americans and 1,867 whites who participated in the San Antonio Heart Study. On the basis of either electrocardiograms or self-reports, the risk of myocardial infarction among Mexican Americans compared with whites was 24 percent lower for men but 40 percent higher for women. Race/ethnicity did not appear to modify the risk for myocardial infarction.

Cerebrovascular Disease

Cerebrovascular disease is a major cause of mortality and morbidity in the United States every year. In 1994, a total of 153,306 deaths in the United States were caused by cerebrovascular disease (NCHS 1996a).

Stroke, the major form of cerebrovascular disease, results from an interruption of the arterial blood supply to the central nervous system, primarily the brain. Most commonly, the interruption of the arterial blood supply results from an occlusion of an artery in the brain by a thrombus, which may have resulted from atherosclerosis or blood clots from a diseased heart. A less common mechanism for development of stroke is rupture of a blood vessel in the brain. Other diagnoses under the general rubric of cerebrovascular disease include transient cerebral ischemia and cerebral arteriosclerosis.

As for CHD, risk factors for stroke may be divided into non-modifiable and modifiable characteristics. The non-modifiable factors include aging, gender, and family history of stroke. The major risk factors that are potentially modifiable include hypertension, hypercholesterolemia, diabetes mellitus, cigarette smoking, and heart disease (USDHHS 1989b).

African Americans

The rate of death from cerebrovascular disease in the United States is higher among African Americans than other racial/ethnic groups and whites (Table 2). For 1992–1994, the rate of death (per 100,000 population) from cerebrovascular disease was twice as high among African American men (53.1) as among white men (26.3) and almost twice as high among African American women (40.6) as among white women (22.6).

Similar patterns have been observed in studies of persons belonging to health plans. Klatsky and colleagues (1991) determined the incidence of hospitalization for cerebrovascular disease among 74,096 whites and 33,041 African Americans who were members of a prepaid health plan in northern California from 1978 through 1984. The relative risks for hospitalization for hemorrhagic cerebrovascular disease, cerebral thrombosis, and nonspecific cerebrovascular disease were higher among African Americans than among whites. Because hypertension is the strongest risk factor for stroke, the high prevalence of hypertension among African Americans partially explains this pattern (Braithwaite and Taylor 1992). Despite limited data on the link between smoking and stroke among African Americans, the high rate of cigarette smoking among African Americans (see Chapter 2) clearly appears to have played a significant role in elevating the risks of stroke in this population (USDHHS 1983).

American Indians and Alaska Natives

In recent years, age-adjusted death rates for cerebrovascular disease were slightly lower among American Indian and Alaska Native men and women than among white men and women (Table 2). For example, from 1992–1994, the age-adjusted death rate per 100,000 population for cerebrovascular disease was 23.9 for American Indian and Alaska Native men, 26.3 for white men, 21.1 for American Indian and Alaska Native women, and 22.6 for white women.

Young's (1994) recent review of the literature indicates that few investigations have focused on cerebrovascular disease among American Indians or Alaska Natives. Middaugh (1990) found little difference between the death rate from cerebrovascular disease among Alaska Natives and persons of other race/ethnicities, with death rate ratios of 1.13 for men and 1.03 for women. In a review of 1958–1987 vital statistics data from New Mexico, Kattapong and Becker (1993) observed lower rates of death from cerebrovascular disease among American Indians than among Hispanics and whites. For American Indian men, cerebrovascular disease death rates per 100,000 peaked at 70.1 between 1968 and 1972 and fell to 31.3 between 1983 and 1987. Cerebrovascular disease death rates for American Indian women also peaked at 55.7 between 1968 and 1972 and declined to a low of 19.3 between 1983 and 1987.

Asian Americans and Pacific Islanders

From 1992 through 1994, the age-adjusted death rate per 100,000 population for cerebrovascular disease was 29.3 for Asian American and Pacific Islander men, 26.3 for white men, 22.4 for Asian American and Pacific Islander women, and 22.6 for white women (Table 2).

In a study of stroke deaths occurring between 1965 and 1972 among Japanese men living in Japan, Honolulu, and San Francisco, age-specific stroke death rates were highest among men living in Japan (Worth et al. 1975). Among men 60–64 years of age, annual death rates per 1,000 men were 5.4 in Japan, compared with 2.5 in San Francisco and 1.1 in Honolulu. For CHD, however, the death rates in Japan were lower than rates in Honolulu and San Francisco. Data from the Honolulu Heart Program suggest that other risk or protective factors associated with a Japanese diet, such as high alcohol intake and low intake of food from animal sources, may play important roles in the development of stroke and CHD in Honolulu and Japan, along with smoking, older age, high systolic blood pressure, and high serum cholesterol and glucose levels (Reed 1990).

In a study of 1980 death rates among Asian Americans in Los Angeles, Frerichs and colleagues (1984) found that Koreans had the lowest age- and gender-adjusted death rate for cerebrovascular disease (48 per 100,000) and that Japanese had the highest rate (80 per 100,000). When the investigators compared the average age- and gender-adjusted death rates for these Asian Americans with rates for the entire county, the mortality ratio was 1.07 for Japanese and 0.65 for Koreans.

Cigarette smoking was found to be an independent risk factor for stroke among men of Japanese ancestry who participated in the Honolulu Heart Program (Abbott et al. 1986). For all types of stroke, the estimated relative risk of smoking, adjusted for age and other major risk factors, was 2.5. This risk decreased to 1.5 among men who quit smoking during the six-year follow-up period and increased to 3.5 among men who continued to smoke, indicating that cigarette smoking is a cause of stroke in Japanese men.

A subsequent analysis of participants in the Honolulu Heart Program indicated that cigarette smoking significantly increased the risk for thromboembolic stroke (Goldberg et al. 1995).

Hispanics

Studies about stroke among Hispanics have focused on the magnitude of this outcome in relation to other racial/ethnic groups. Between 1986 and 1988, the overall rate of death from cerebrovascular disease was lower among Hispanics than among whites in the United States (Desenclos and Hahn 1992). When cerebrovascular disease death rates for Hispanics and whites were compared, the mortality ratio for Hispanic men was 0.89, and the ratio for Hispanic women was 0.84. Of the different Hispanic subgroups, Mexican Americans had the highest death rates from cerebrovascular disease. Sorlie and colleagues (1993) had similar observations when they estimated death rates using census data collected between 1973 and 1985. Age-adjusted death rate ratios for cerebrovascular disease were lower among Hispanics than among whites (0.60 for men and 0.76 for women). No details were provided for the different Hispanic subgroups. In more recent years, age-adjusted death rates for cerebrovascular disease were slightly lower among Hispanic men and women than among white men and women. For example, from 1992–1994, the age-adjusted death rate per 100,000 population for cerebrovascular disease was 22.7 for Hispanic men, 26.3 for white men, 16.7 for Hispanic women, and 22.6 for white women (Table 2).

Regional studies in California (Frerichs et al. 1984), New Mexico (Kattapong and Becker 1993), and Texas (Stern and Gaskill 1978) provide further evidence that Hispanics have a lower risk of death from cerebrovascular disease than do whites and African Americans. Frerichs and colleagues (1984) compared 1980 death rates among the different racial/ethnic groups in Los Angeles County. The age- and gender-adjusted cerebrovascular disease death rates per 100,000 were 64 for Hispanics compared with 76 for whites (death rate ratio, 0.84) and 94 for African Americans (death rate ratio, 0.68).

After reviewing New Mexico vital statistics data for 1958–1987, Kattapong and Becker (1993) described time trends in deaths from cerebrovascular disease among Hispanics, whites, and American Indians. Except for the period 1983–1987, Hispanic men had lower death rates than white men. From 1983 to 1987, the ratio of death rates among Hispanic men (45.8 per 100,000) compared with the rate among white men (36.1 per 100,000) was 1.27. For women, the pattern of death rates was less consistent. From 1958 through 1972, Hispanic women had higher death rates than white women; between 1973 and 1982, they had lower rates; and from 1983 through 1987, Hispanic women had slightly higher death rates (43.1 per 100,000) than white women (39.3 per 100,000).

Stern and Gaskill (1978) examined temporal trends in stroke deaths from 1970 through 1976 among Hispanics and whites living in Bexar County, Texas, which includes San Antonio. Stroke deaths were generally lower among Hispanic women, but no significant difference was observed between the rates among men of either racial/ethnic group. Furthermore, no temporal trends in stroke deaths were evident for either gender or racial/ethnic group.

Cigarette smoking probably explains some of the risk of stroke among Hispanics. However, data to assess the strength of this relationship are not available. Because the data presented here suggest that stroke is a leading cause of morbidity and death among Hispanics (NCHS 1993), future studies should examine the specific role that cigarette smoking plays.

Smoking and Pregnancy

Smoking has long been known to be associated with poor outcomes for the infants of mothers who smoke. Mean infant birth weight and low birth weight (LBW) (<2,500 grams or <5.5 pounds) are often studied as measures of fetal morbidity because birth weight is easy to measure. LBW can result either from preterm delivery (<37 weeks' gestation) or from intrauterine growth retardation, but the distinction may be difficult to make. Smoking has been associated with an average decrease in birth weight of about 200 grams as well as LBW, preterm birth, perinatal mortality, and infant mortality (USDHHS 1980, 1989b; Malloy et al. 1988; English and Eskenazi 1992).

Evidence that the relationship between smoking and poor infant outcomes is causal has been strengthened by recent studies that used biomarkers of tobacco exposure, such as saliva and serum cotinine (Bardy et al. 1993; Li et al. 1993; English et al. 1994). Bardy and colleagues (1993) demonstrated a dose-response relationship between serum cotinine and decreased gestational age, decreased birth weight, and decreased crown-heel length.

The exact mechanisms whereby smoke exposure affects the fetus are poorly understood. Carbon monoxide, which impairs oxygen delivery to the fetus, and nicotine, which impairs placental blood flow, have been implicated as the causative substances in tobacco smoke (USDHHS 1980).

The infant outcomes most often studied have been LBW and infant mortality. Sudden infant death syndrome (SIDS) is an important component of infant mortality because it is the most common cause of death among infants older than one month of age. Available data show that LBW, infant mortality, and SIDS occur differentially in different racial/ethnic groups in the United States (Table 10) (Kleinman 1990; NCHS 1994). In general, whites have lower rates of these conditions and other racial/ethnic groups tend to have higher rates, but considerable variation exists.

Several studies have reported different effects of smoking on LBW, infant mortality, and SIDS across racial/ethnic minority groups. This section focuses only on those studies that have investigated potential racial/ethnic group differences in the relationship between smoking and infant outcomes.

Studies of Low Birth Weight

Nearly 25 years ago, the possibility was raised that smoking might have a differential effect on reproductive outcomes in different racial/ethnic groups (Lubs 1973). In a study of all singleton live births at Yale-New Haven Hospital in 1972, Lubs reported a difference in the effect of maternal smoking on LBW among 783 African American and 3,415 white women. A strong dose-response relationship was observed between the number of cigarettes smoked during pregnancy and infant LBW (defined as ≤2,500 grams for whites and ≤2,350 grams for African Americans). Among African American women, smoking 20 or more cigarettes per day was associated with a threefold increase in LBW, compared with only a twofold increase among white women. These racial/ethnic group differences were not explained by differences in age, prepregnancy weight, education, or marital status.

Several more recent studies also provide evidence for the possibility of a differential effect of smoking on LBW among white and African American women. English and colleagues (1994) used interview data from the Child Health and Development Studies, conducted from 1959 through 1966 in California. Stored serum samples were analyzed for cotinine, and the levels were compared with self-reported cigarette consumption and infant birth weight for 374 African American and 829 white pregnant smokers separately. African American pregnant smokers were found to have higher serum cotinine levels than white pregnant smokers after the data were controlled for smoking dose and demographic confounders. No racial/ethnic minority group difference was found in the rate of decrease in mean birth weight per given amount of cotinine in the serum of women who smoked. These data suggest that cigarette smoking may have a greater effect on birth weight among African Americans than among whites because higher cotinine levels are present in African American women than in white women who smoke the same amount; the higher cotinine levels may result from a greater intake of tobacco smoke per cigarette by African American women than by white women.

Li and colleagues (1993) found a differential effect of smoking reduction during pregnancy on infant birth weights among African American and white women. Study subjects were 803 participants in an experimental trial of smoking cessation for pregnant women in Alabama; self-reported smoking was validated with saliva cotinine. Reduction was defined as a minimum drop in saliva cotinine values between the baseline (early pregnancy) visit and the late pregnancy visit. Smoking reduction increased the birth weight of infants of both African American and white women, but racial/ethnic group differences were present. Among white women, a reduction in smoking increased infant birth weight regardless of the baseline cotinine value. However, among African American women with high baseline cotinine values, a reduction in smoking had no effect on infant birth weights. The authors suggested that high levels of cigarette smoking (as detected by high cotinine levels) early in pregnancy may have irreversible effects on African American infants.

Another recent study reported a differential effect of smoking on LBW (<2,500 grams) among multiparous African American and white women, but in the opposite direction (Neggers et al. 1994). Among African American women, the investigators found no significant difference in birth weight between smokers

Table 10. Rates of selected infant outcomes, by mother's race/ethnicity,* United States

Reference	Outcome/years	African American	American Indian and Alaska Native	Asian American and Pacific Islander				
				Total	Chinese	Japanese	Filipino	Other
NCHS, public use data tapes, 1992§	Low-birth-weight (<2,500 grams) rate per 100 live births, 1992	13.4	6.2	6.6	5.2	7.5	7.4	6.9
NCHS 1994§	Infant mortality rate per 1,000 live births, 1987	17.8	13.0	7.3	6.2	6.6	6.6	7.9
Kleinman 1990	Sudden infant death syndrome rate per 1,000 live births, 1983–1984	2.41	3.44	0.95	NA	NA	NA	NA

*The categories African American and white include persons of Hispanic and non-Hispanic origin. Conversely, persons of Hispanic origin may be included in other categories as well.
†Reported for selected states only; reporting areas for Hispanic origin vary by year.

and nonsmokers, whereas among white women, the infants of smokers weighed significantly less than those of nonsmokers. However, no information was available on the number or type of cigarettes smoked or the biomarker of exposure; these results were adjusted only for the mother's parity, age, height, and alcohol consumption as well as the infant's gender and gestational age at birth. In addition, the study was not designed to study the relationship between smoking and LBW but to determine whether the relationship between maternal triceps skinfold thickness and infant birth weight was modified by smoking and race/ethnicity.

Two studies have reported that smoking is related to an elevated risk of LBW among both African American and white women, but neither study found significant racial/ethnic group differences. In a population-based, case-control study of African American and white women delivering singleton infants without congenital anomalies in a large urban county of California, the Alameda County Low Birth Weight Study Group (1990) found that the risk of LBW associated with regular smoking throughout pregnancy was 3.0 (95 percent confidence interval [CI], 1.7–5.3) for white women and 3.6 (95 percent CI, 2.4–5.6) for African American women (adjusted for age, parity, prepregnancy weight, socioeconomic status, alcohol use, prior LBW birth, and prenatal care). Unfortunately, the authors were unable to adjust the data for the number of cigarettes smoked.

Castro and colleagues (1993) reported a study of maternal smoking and substance abuse during pregnancy and found similar associations between smoking during pregnancy and small size for gestational age (birth weight of less than the 10th percentile for gestational age) for African American and white women (odds ratio [OR] for African American women, 2.0 [95 percent CI, 1.3–3.1]; OR for white women, 2.4 [95 percent CI, 1.7–3.0]). These results were adjusted for maternal age, parity, marital status, insurance status, alcohol use, marijuana use, and other drug use; however, no information was available on the number of cigarettes smoked or the biomarker of exposure.

Few studies have examined the relationship between smoking and LBW among Hispanic populations. Cohen and colleagues (1993) analyzed birth weight data on 19,571 Hispanic infants and 206,973 white infants (those whose mothers did not indicate they were of Hispanic origin) born in Massachusetts

	Hispanic[†]					
Total	**Mexican American**	**Puerto Rican**	**Cuban**	**Central and South American**	**Other[‡]**	**White**
6.4	6.0	8.8	6.0	5.6	7.5	5.9
8.2	8.0	9.9	7.1	7.8	8.7	8.2
NA	0.84	1.38	0.83	0.53	1.52	1.21

[‡]Includes persons of unknown Hispanic origin.
[§]Data calculated to one significant digit.
NA = data not available.

between 1987 and 1989 and found that the incidence of LBW ranged from a high of 73 per 1,000 Puerto Rican infants to a low of 32.2 per 1,000 Cuban infants. The crude percentage of LBW was higher for smokers than for nonsmokers in each racial/ethnic group; however, multivariate adjusted risks were not presented for racial/ethnic groups separately.

Several studies have demonstrated associations between smoking and LBW in specific racial/ethnic minority groups, including Puerto Ricans (Becerra and Smith 1988), Mexican Americans (Wolff et al. 1993), North American Indians (Godel et al. 1992), and African Americans (Jacobson et al. 1994; Johnson et al. 1994). In each instance, smoking was shown to be related to lower birth weight; however, these studies did not provide data on other racial/ethnic groups, which might have allowed comparisons.

The percentage of LBW (<2,500 grams) in the United States in 1993 was higher overall for smokers (11.8 percent) than for nonsmokers (6.6 percent) (NCHS 1996b). Although a higher percentage of white mothers (16.8) smoked during pregnancy than did African American mothers (12.7), African American women had a higher percentage (13.3) of LBW live births than white women (6.0) did in 1993. Age- and racial/ethnic-specific analyses of population data may be more revealing. Land and Stockbauer (1993), for example, found that the teenage-specific LBW rate for African Americans in Missouri dropped by 13.6 percent from 1978–1990, concomitant with a drop in cigarette smoking prevalence among young African American mothers. Analyses of individual data statistically controlled for confounding factors such as preterm deliveries and maternal parity, weight, and access to health care (USDHHS 1989a) would be preferable. The studies of individuals that are reported in this section provide more useful data than do population-based ecological comparisons on the relationship between cigarette smoking and the increased occurrence of LBW in various racial/ethnic groups.

Studies of Infant Mortality and Sudden Infant Death Syndrome

Only one study has examined the risks of smoking associated with overall fetal and infant mortality in specific racial/ethnic groups (Kleinman et al. 1988). The authors used data from Missouri live birth, fetal death, and infant death certificates for births during

Table 11. Risk of sudden infant death syndrome associated with smoking, by race/ethnicity, selected studies, United States

		African American		American Indian and Alaska Native		Asian American and Pacific Islander	
Reference	**Exposure/years**	**OR***	**CI†**	**OR**	**CI**	**OR**	**CI**
Li and Daling 1991‡	Active smoking 1984–1989	3.1	1.7–5.9	1.4	0.9–2.4	2.7	1.1–6.6
Schoendorf and Kiely 1992§	Passive exposure 1988	1.8	1.0–3.0	NA	NA	NA	NA
	Combined exposure 1988	3.1	2.3–4.2	NA	NA	NA	NA
Klonoff-Cohen et al.Δ 1995	Passive exposure 1989–1992	5.0	1.1–22.8	NA	NA	NA	NA

*OR = odds ratio.
†CI = 95% confidence interval.
‡Li and Daling assessed the risk, by mother's ethnicity, associated with active maternal smoking during pregnancy; ORs are adjusted for maternal age, marital status, prenatal care, parity, and birth weight.
§ Schoendorf and Kiely assessed the risk, by mother's ethnicity, associated with (1) passive smoking (maternal smoking after birth but not during pregnancy) and (2) combined exposure (maternal smoking during pregnancy and after birth); ORs are adjusted for maternal age, education, and marital status.
ΔKlonoff-Cohen et al. assessed the risk, by infant's ethnicity, associated with total passive smoke exposure from all adults (mother, father, live-in adults, and day-care providers); ORs are adjusted for birth weight, routine sleep position, medical conditions at birth, breast-feeding, prenatal care, and maternal smoking during pregnancy.
NA = data not available.

1979–1983 to examine the risk of mortality associated with smoking during pregnancy. They found no significant variation in the effects of smoking on African American and white women, with adjusted ORs ranging from 1.3 to 1.6, depending on parity and the amount smoked.

Three studies have examined the effects of smoking on SIDS in specific racial/ethnic minority groups (Table 11) (Li and Daling 1991; Schoendorf and Kiely 1992; Klonoff-Cohen et al. 1995). Li and Daling (1991) used data from Washington State birth records from 1984 through 1989, linked with infant death records. After adjusting the data for maternal age, marital status, prenatal care, parity, and birth weight, they found a statistically significant increased risk of SIDS associated with maternal smoking during pregnancy in all racial/ethnic groups except American Indians (Table 11). The ORs were not significantly different between groups, except between African Americans and American Indians. No information was available on the number of cigarettes smoked or the biomarker of exposure.

Schoendorf and Kiely (1992) used data from the 1988 National Maternal and Infant Health Survey to study the association between SIDS and maternal smoking (either passive [only after birth] or combined [during pregnancy and after birth]) among infants of normal birth weight. They found similar increased risks of SIDS among African American and white infants exposed to maternal smoking (Table 11), after adjusting the data for maternal age, education, and marital status. Although white mothers reported heavier smoking than African American mothers, the authors did not adjust their findings for the number of cigarettes smoked.

Hispanic		White	
OR	**CI**	**OR**	**CI**
5.5	1.4–22.0	2.2	1.8–2.6
NA	NA	3.1	2.3–4.2
NA	NA	1.8	1.0–3.0
2.6	0.9–7.3	3.4	1.6–7.2

Klonoff-Cohen and colleagues (1995) conducted a 1989–1992 case-control study of passive smoking and SIDS in five counties in southern California. The OR for SIDS associated with all types of passive smoke exposure combined was 3.50 (95 percent CI, 1.81–6.75), after adjustment for birth weight, routine sleep position, medical conditions at birth, breast-feeding, prenatal care, and maternal smoking during pregnancy. The evidence suggested a dose-response relationship, with an increased risk of SIDS associated with increased passive exposure to smoke. The authors also stratified the data by racial/ethnic group and found similar effects across groups (Table 11), although the results were not adjusted for the number of cigarettes smoked.

Health Problems Affecting Pregnant Women

Smoking is related to a variety of health problems affecting pregnant women, ranging from ectopic pregnancy to abruptio placentae (USDHHS 1980; Rosenberg 1987), but race- and ethnic-specific data are not generally available. In addition to exploring smoking's effects on fetuses and infants, future research should focus on the race- and ethnic-specific effects of smoking on the pregnant woman herself.

Implications

The question of whether race- and ethnic-specific differences exist in the relationship between smoking and infant outcomes has not been satisfactorily resolved. Many intriguing questions have been raised, but investigators have not yet determined the exact nature of such differences or what factors mediate them.

Comparative studies have been hampered by inconsistent and inadequate measurement of exposure. For example, few investigators have fully explored issues of dose of smoking such as the number of cigarettes smoked or the levels of biomarkers, although the amount of smoking during pregnancy does differ among racial/ethnic minority groups (see Chapter 2). Moreover, even though the timing of smoking during pregnancy may play a critical role in the development of LBW (Lieberman et al. 1994), few studies of LBW have separately assessed the effects of smoking during each trimester of pregnancy. Patterns of quitting and reducing smoking during pregnancy may in fact differ by race/ethnicity.

Racial/ethnic group differences in nicotine metabolism may also be important (Wagenknecht et al. 1990; English et al. 1994). African American pregnant smokers appear to have higher serum cotinine levels than white pregnant smokers when the data are controlled for nicotine dose (English et al. 1994). Thus, fetal exposure may be higher among African Americans than among whites for a given number of cigarettes smoked.

Racial/ethnic group differences in oxygen-carrying capacity may also play a role in mediating the effects of smoking. In 1973, Lubs suggested that the increased effects of smoking on birth weight among African American women might in part be explained by higher rates of sickle cell trait or glucose-6-phosphate dehydrogenase (G6PD) deficiency, which impair oxygen-carrying capacity (Lubs 1973). No published reports have examined Lubs's hypothesis. In addition, anemia, which is more prevalent among African American women, may be a risk factor for preterm delivery (Hogue and Yip 1989).

Future studies of smoking and pregnancy outcomes should consider racial/ethnic group differences in the timing of smoking during pregnancy, nicotine metabolism, and factors that affect oxygen-carrying capacity, such as sickle cell trait, G6PD deficiency, and anemia.

Summary of Health Consequences from Active Cigarette Smoking

Attempts to predict racial- and ethnic-specific rates of disease incidence and mortality from racial- and ethnic-specific cigarette smoking prevalences are of limited value, because other factors can also influence disease rates. When studies of individuals are conducted, the data lead to the conclusion that cigarette smoking is a major cause of disease and death in each of the four U.S. racial/ethnic minority groups studied in this report. These studies reveal few major differences in the risk ratios for various diseases. Limited epidemiological and biological data suggest that African Americans may be at an especially high level of risk for lung cancer. Although further research could clarify the nature of the interrelationships between cigarette smoking, other risk factors, potential modifying factors, racial/ethnic group membership, and various disease outcomes, it is clear that reducing tobacco use in each of the nation's racial/ethnic groups will reduce the incidence and mortality from several of the nation's leading causes of death and is a major public health goal to pursue.

Effects of Exposure to Environmental Tobacco Smoke

Environmental tobacco smoke (ETS) is the mixture of sidestream smoke and exhaled mainstream smoke that is produced by active smokers and then involuntarily inhaled by nonsmokers. Over the past decade, the adverse effects of ETS have been reported in the literature. The 1986 Surgeon General's report on smoking and health (USDHHS 1986a) concluded that the inhalation of ETS (labeled "involuntary smoking" in that report) is a cause of diseases, including lung cancer, in healthy nonsmokers and that the children of parents who smoke are more likely than the children of nonsmoking parents to have respiratory infections, respiratory symptoms, and abnormal maturation of lung function. Similar conclusions were also reached in 1986 by a committee of the National Research Council (1986). More recently, the U.S. Environmental Protection Agency (1992) assessed the risks associated with ETS, and the results reaffirmed that ETS is carcinogenic and that it exacerbates and may even cause childhood asthma. To date, racial/ethnic group differences in the adverse effects of ETS have not been investigated, although a number of studies have investigated racial/ethnic group differences in the level of exposure to ETS and in people's reactions to ETS.

Overpeck and Moss (1991) examined patterns of exposure to ETS among children five years of age and younger included in the 1988 NHIS and found that exposure varied by race/ethnicity and socioeconomic status (Table 12). African American children were the most likely to be exposed to ETS, whereas Hispanic children were the least likely to be exposed to ETS. Moreover, in the CARDIA (Coronary Artery Risk Development in [Young] Adults) study, the prevalence of exposure to ETS was significantly higher among African Americans (32 percent) than among whites (24 percent) (Wagenknecht et al. 1993). Overall, 28 percent of individuals 18–30 years of age were exposed to ETS, as detected by a serum cotinine level of 2–13 ng/mL. Adult survey data from the 1992 California Tobacco Survey show that Hispanics (21.3 percent) were most likely to report working around a cigarette smoker within the two weeks before the survey (Pierce et al. 1994). Asian Americans (13.2 percent) and African Americans (12.8 percent) reported being exposed to ETS at work in lower proportions than whites (17.9 percent). Data from the 1988 NHIS (CDC 1992) show that 40.3 percent of employed adults reported that cigarette smoking was allowed in their place of employment. The percentages of persons who reported experiencing discomfort caused by ETS exposure at work did not differ significantly by racial/ethnic group. In a 1992–1993 study of U.S. adults who worked indoors, Asian Americans and Pacific Islanders (51.4 percent) were the most likely and African Americans (43.3 percent) were the least likely to work under a completely smoke-free ETS policy (Gerlach et al. 1997). Since most studies suggest that differences exist in the ETS exposure of various racial/ethnic groups, studies to monitor the health effects of this exposure are needed.

Table 12. Exposure to household smoke among children 5 years of age and younger and percentage distribution, by level of exposure since birth and selected characteristics, United States, 1988

Characteristic	Number of children (in thousands)[†]	Percentage distribution*				
		Total	Not exposed since birth	Exposed since birth		
				Total[‡]	Current smoker in household	Former smoker in household
All children[§]	19,019	100.0	51.1 (0.9)	48.9 (0.9)	42.4 (0.9)	6.1 (0.4)
Ethnicity						
African American	2,759	100.0	41.5 (2.4)	58.5 (2.4)	51.3 (2.4)	6.7 (1.2)
White	15,575	100.0	51.9 (1.0)	48.1 (1.0)	41.6 (1.0)	6.1 (0.4)
Hispanic origin						
Non-Hispanic	16,923	100.0	50.4 (1.0)	49.6 (1.0)	43.2 (1.0)	6.0 (0.4)
Hispanic	2,096	100.0	56.4 (2.6)	43.6 (2.6)	35.8 (2.5)	6.9 (1.2)
Mexican American	1,006	100.0	60.7 (4.1)	39.3 (4.1)	31.8 (3.8)	6.5 (1.5)
Annual household income						
<$10,000	2,685	100.0	33.4 (2.1)	66.6 (2.1)	57.7 (2.3)	8.7 (1.1)
$10,000–$24,999	5,436	100.0	44.3 (1.5)	55.7 (1.5)	48.8 (1.6)	6.3 (0.7)
$25,000–$39,999	4,871	100.0	55.9 (1.7)	44.1 (1.7)	38.3 (1.6)	5.4 (0.7)
≥$40,000	4,149	100.0	65.7 (1.8)	34.3 (1.8)	29.5 (1.5)	4.6 (0.9)
Poverty status[Δ]						
In poverty	3,376	100.0	36.4 (2.1)	63.6 (2.1)	55.7 (2.3)	7.6 (1.0)
Not in poverty	14,582	100.0	54.8 (1.0)	45.2 (1.0)	39.2 (1.0)	5.6 (0.4)
Mother's education						
<12 years	3,279	100.0	33.3 (2.2)	66.7 (2.2)	61.2 (2.1)	5.1 (0.8)
12 years	8,014	100.0	44.5 (1.4)	55.5 (1.4)	47.9 (1.4)	7.3 (0.6)
>12 years	7,505	100.0	66.3 (1.2)	33.7 (1.2)	27.6 (1.1)	5.4 (0.6)
Place of residence						
Metropolitan statistical area	14,550	100.0	51.5 (1.0)	48.5 (1.0)	42.2 (1.1)	5.9 (0.4)
Central city	5,994	100.0	49.4 (1.4)	50.6 (1.4)	43.6 (1.5)	6.3 (0.6)
Not central city	8,556	100.0	52.9 (1.4)	47.1 (1.4)	41.1 (1.4)	5.6 (0.6)
Not metropolitan statistical area	4,469	100.0	49.7 (1.9)	50.3 (1.9)	43.1 (1.7)	6.8 (0.8)

*Figures in parentheses are standard errors of estimates.
[†]Excludes children whose exposure status is unknown.
[‡]Includes children exposed since birth whose period of exposure is unknown.
[§]Includes all other ethnicities, unknown household income, unknown poverty status, unknown education of mother, and unknown assessed health status.
[Δ]Poverty status determined in the National Health Interview Survey by family size, number of children, and household income by using 1987 poverty levels defined by the U.S. Bureau of the Census.
Source: Adapted from Overpeck and Moss 1991.

Effects of Smokeless Tobacco Use

Smokeless tobacco refers to moist oral snuff, dry oral and nasal snuff, and chewing tobacco. Smokeless tobacco is commonly used by youths, particularly those in rural areas, and it is highly addictive (USDHHS 1986b; Boyd and Glover 1989). Among the adverse health effects of smokeless tobacco use are oral cancer, oral leukoplakia (white mouth lesions that may be precancerous), gingival recession, periodontal diseases, elevated blood pressure, and increased risk for cardiovascular disease (NCI 1992; USDHHS 1994; Bolinder et al. 1994).

Few studies have examined the adverse health effects of smokeless tobacco use in racial/ethnic minority populations, and the research that has been conducted has been limited in several ways: (1) population-based, case-control studies rarely have sufficient numbers of racial/ethnic group members to allow group-specific analyses for groups other than African Americans (Blot et al. 1988; Day et al. 1993); (2) because the use of smokeless tobacco and associated health effects are relatively rare in most racial/ethnic groups, the feasibility of conducting prospective investigations is limited; and (3) smokeless tobacco users often report current or past use of other substances, such as cigarettes and alcohol, that are risk factors for health effects also associated with smokeless tobacco use, such as oral cancer (Blot et al. 1988; Mattson and Winn 1989). These multiple risk factors complicate or preclude analysis of the independent effects of smokeless tobacco use.

The valid data that are available, however, indicate that for men, the prevalence of smokeless tobacco use is highest among American Indians, Alaska Natives, and whites; for women, the prevalence is highest among American Indians, Alaska Natives, and African Americans (CDC 1993c). Data for 1989–1991 show that rates of death from cancers of the lip, oral cavity, and pharynx have been higher among African American men (7.8 per 100,000) than among Puerto Rican men (3.9 per 100,000), Asian American and Pacific Islander men (3.4 per 100,000), and white men (3.2 per 100,000) (Table 2) (NCHS, public use data tapes, 1989–1991; U.S. Bureau of the Census 1993).

In a case-control study, Winn and colleagues (1981) examined the estimated relative risk of oral and pharyngeal cancer associated with snuff-dipping among African American and white women in the southern United States. Although the relative risk was higher among white women (4.2) than among African American women (1.5), white women had dipped snuff for significantly longer periods and had consumed more snuff per week than African American women had. The relative risk for cancers of the gum and buccal mucosa increased with longer duration of snuff use, but this analysis was not conducted separately for African Americans and for whites.

A few studies of the health effects associated with smokeless tobacco use have been conducted among American Indian and Alaska Native populations. In a study of Navajo youths aged 14–19 years in New Mexico (Wolfe and Carlos 1987), 64 percent of the teenagers used smokeless tobacco products. Oral leukoplakia was found in 26 percent of smokeless tobacco users, representing a ninefold increase in risk when these youths were compared with those who did not use smokeless tobacco. The estimated relative risk of leukoplakia increased with duration and frequency of smokeless tobacco use. The investigators observed no apparent differences between users and nonusers of smokeless tobacco regarding gingival bleeding, calculus accumulation, or the extent or severity of gingival recession or loss of periodontal attachment.

In a survey of students in grades 7–12 attending schools on the Rosebud Sioux Reservation in South Dakota, more than one-third of the students reported regularly using smokeless tobacco (CDC 1988). Of these regular users, 37 percent had oral lesions (i.e., any white or red wrinkled area in the mouth or buccal mucosa). The students with oral lesions had used smokeless tobacco for a mean of 3.4 years, 6.6 times per day, and they had held each dip or chew for an average of 40 minutes. Students who used smokeless tobacco but did not have lesions had used the product for a mean of 2.5 years, 2.9 times per day, and they had held each dip or chew for an average of 30 minutes. This suggests a possible relationship between duration and intensity of smokeless tobacco use and the occurrence of oral lesions. The prevalence of oral lesions among nonusers of smokeless tobacco was not reported.

The 1986–1987 National Survey of Oral Health in U.S. School Children conducted oral clinical examinations on 17,027 children aged 12–17 years who provided information on their use of various tobacco products (Tomar et al. 1997). Smokeless tobacco lesions (defined by the authors as slight to heavy

wrinkling of the oral mucosa) were more common among white (2.0 percent) than among African American (0.2 percent) or Hispanic (0.8 percent) school children. In white males, the strongest correlates of lesions were, in order, current snuff use and current chewing tobacco use. Lesions were more common with increasing duration and frequency of smokeless tobacco use. Because of small sample sizes, analyses were not conducted on data for other racial/ethnic groups.

Nicotine Addiction and Racial/Ethnic Differences

Most smokers have difficulty quitting because they are addicted to nicotine (USDHHS 1988). An understanding of the role of nicotine addiction in determining smoking behavior could help clarify racial/ethnic differences in tobacco use and facilitate smoking cessation treatment. Nicotine addiction was reviewed extensively in the 1988 Surgeon General's report on smoking and health (USDHHS 1988). Concepts of addiction also have been reviewed in subsequent Surgeon General's reports (USDHHS 1989b, 1994). However, relatively little research has been conducted on racial/ethnic minority differences in nicotine addiction. This section provides a brief review of nicotine addiction and discusses the limited data on racial/ethnic differences and nicotine addiction.

Nature of Addiction

In the broadest sense, addiction (often used interchangeably with dependence) indicates a loss of control over drug-taking behavior. The World Health Organization describes drug dependence as "a behavioral pattern in which the use of a given psychoactive drug is given a sharply higher priority over other behaviors which once had a significantly higher value" (Edwards et al. 1982). In other words, drug use controls one's behavior to an extent considered detrimental to the individual or to society.

The criteria for drug dependence, described in the 1988 Surgeon General's report on smoking and health (Table 13) (USDHHS 1988), include highly controlled or compulsive use of a drug, the use of a drug that produces psychoactive effects, and evidence that drug-taking behavior is reinforced by the effects of the drug. Other criteria for drug dependence have been developed by the American Psychiatric Association [APA] (1994) for the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV™)* (Table 14). These criteria are quite specific and useful in diagnosing drug dependence in individual patients.

Pharmacologic Factors in Nicotine Addiction

Nicotine addiction, like all drug addictions, is a complex process involving the interplay of pharmacology, learned or conditioned factors, personality, social setting, and genetics (USDHHS 1988, 1994; Benowitz 1992a). The pharmacologic reasons for drug use include an enhancement of one's mood or functioning. Drugs produce such effects either directly or by relieving withdrawal symptoms. The pharmacologic factors involved in nicotine addiction work in several ways. For example, positive effects reported after smoking tobacco include pleasure, arousal, and relaxation as well as improved attention, reaction time, and performance of certain tasks. In addition, cigarette smoking has been cited as effective in relieving aversive emotional states, including reducing anxiety or stress, relieving hunger and preventing weight gain, and relieving nicotine withdrawal symptoms (Table 15) (Benowitz 1992a).

The pharmacology of nicotine addiction can be discussed in relation to several processes: (1) absorption, distribution, and elimination of nicotine in the body (pharmacokinetics); (2) pharmacologic effects of nicotine on target organs (pharmacodynamics); and (3) translation of pharmacologic effects into behavior. These processes are reviewed in the following sections, and racial/ethnic differences are discussed when information is available.

Absorption, Distribution, and Elimination of Nicotine in the Body

Nicotine from tobacco smoke is absorbed rapidly across the lungs' alveolar membranes and into the systemic circulation (Benowitz 1990). Following absorption from the lung, concentrations of nicotine in the blood rise quickly and peak at the completion of smoking. Concentrations of nicotine in arterial blood leaving the lungs and heart are several times higher than those measured in venous blood (Henningfield

Table 13. Criteria for drug dependence

Primary criteria
Highly controlled or compulsive use
Psychoactive effects
Drug-reinforced behavior
Additional criteria
Addictive behavior often involves—
stereotypic patterns of use
use despite harmful effects
relapse following abstinence
recurrent drug cravings
Dependence-producing drugs often produce—
tolerance
physical dependence
pleasant (euphoric) effects

Source: Adapted from U.S. Department of Health and Human Services 1988.

et al. 1993). Within 10 to 19 seconds after the start of a puff, nicotine is delivered to the brain. Rapid delivery of high concentrations of nicotine to the brain provides the possibility for rapid behavioral reinforcement from smoking and allows the smoker to control the concentration of nicotine in the brain and, hence, to modulate the pharmacologic effects of nicotine.

In contrast, the absorption of nicotine from smokeless tobacco is gradual, with blood levels peaking at the end of chewing tobacco or using snuff (Benowitz et al. 1988). Buccal-oral absorption results in a gradual increase in concentrations of nicotine in the brain, with relatively little arterial-venous disequilibrium. This pattern of absorption may provide a less intense pharmacologic reinforcement than that produced by smoke inhalation but is sufficient to produce addiction.

The level of nicotine in the body is determined by the balance of nicotine intake from tobacco and the rate of nicotine elimination from the body. Nicotine is eliminated primarily by hepatic metabolism, with a small amount (5–10 percent) excreted unchanged in the urine. The primary metabolite of nicotine is cotinine, which has been used as a measure of nicotine exposure (Benowitz 1996). Keenan and colleagues (1994, 1995) recently published preliminary data consistent with the hypothesis that cotinine has some psychoactive properties. These effects do not appear to be mediated by nicotine receptor agonism, but could play some role in nicotine addiction. The rate of metabolizing nicotine varies considerably from person to person (Benowitz et al. 1982). A person who metabolizes nicotine slowly would not need to take in as much nicotine to achieve a particular level of nicotine in the body as a person who metabolizes nicotine more rapidly. The level of nicotine in the body appears to be positively correlated with the degree of nicotine dependence and negatively correlated with the likelihood of successful cessation therapy (USDHHS 1988; Pomerleau et al. 1990; Sutherland et al. 1992).

Theoretically, racial / ethnic differences in the absorption, distribution, or elimination of nicotine could influence the likelihood of developing nicotine dependence (see Racial / Ethnic Differences in Nicotine Metabolites later in this chapter for further discussion of this topic).

Pharmacodynamics of Nicotine

Nicotine acts on nicotinic cholinergic receptors in the brain and other organs of the body, enhancing the release of neurotransmitters such as acetylcholine, norepinephrine, dopamine, beta-endorphin, and serotonin (USDHHS 1988). The physiologic consequences of nicotine intake include behavioral arousal and sympathetic neural activation (Table 15) (Benowitz 1992a). The release of specific neurotransmitters has been speculatively linked to the various reinforcing effects of nicotine (Pomerleau and Pomerleau 1984). For example, the enhanced release of dopamine and norepinephrine may be associated with pleasure as well as appetite suppression, the latter of which may contribute to lower body weight. The release of acetylcholine may be associated with improved performance of behavioral tasks and improved memory, whereas the release of beta-endorphin may be associated with reduced anxiety and tension.

Although smokers give different explanations for smoking, most agree that smoking produces arousal, particularly with the first few cigarettes of the day, and paradoxically, smoking can also be calming or relaxing, especially in stressful situations (Pomerleau and Pomerleau 1984; Benowitz 1992a). Consistent with reports of arousal, the smoking of cigarettes or the administration of nicotine is followed by electroencephalographic desynchronization, with an upward shift in the brain's dominant alpha frequency and decreased total alpha and theta power (Pickworth et al. 1989).

Table 14. American Psychiatric Association diagnostic criteria for substance dependence

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three or more of the following consequences, occurring at any time in the same 12-month period:
Tolerance, as defined by either—
need for markedly increased amounts of the substance to achieve intoxication or desired effect or markedly diminished effect with continued use of the same amount of the substance.
Withdrawal, as manifested by either—
the characteristic withdrawal syndrome* for the substance or the same (or a closely related) substance being taken to relieve or avoid withdrawal symptoms.
Consumption of the substance in larger amounts or over a longer period than was intended.
Having a persistent desire to cut down or control substance use or unsuccessfully trying to do so.
Spending a great deal of time in activities necessary to obtain the substance (e.g., visiting multiple doctors or driving long distances), use the substance (e.g., chain-smoking), or recover from its effects.
Giving up or reducing important social, occupational, or recreational activities because of substance use.
Continuing to use the substance, despite the knowledge that one has a persistent or recurrent physical or psychological problem likely caused or exacerbated by the substance (e.g., current cocaine use despite recognition of cocaine-induced depression or continued drinking despite recognition that an ulcer was worsened by alcohol consumption).

*The characteristic withdrawal syndrome for nicotine refers to the daily use of nicotine for at least several weeks and abrupt cessation of nicotine use, or reduction in the amount of nicotine used, followed within 24 hours by four or more of the following signs: dysphoric or depressed mood; insomnia; irritability, frustration, or anger; anxiety; difficulty concentrating; restlessness; decreased heart rate; increased appetite or weight gain.

Source: Adapted from American Psychiatric Association 1994.

Several researchers have studied the effects of cigarette smoking and nicotine administration on the behavior of smokers who have abstained from tobacco use (abstinent smokers) (USDHHS 1988; Hughes et al. 1990; Warburton 1990; Le Houezec and Benowitz 1991; Heishman et al. 1994). Many of these studies have shown that nicotine restores tobacco-abstinence-related deficits in attention and short-term memory and decreases reaction time (Peeke and Peeke 1984; USDHHS 1988; Snyder et al. 1989; Snyder and Henningfield 1989; Warburton 1990; Levin 1992; Pritchard et al. 1992). Nicotine also may increase a person's vigilance in performing repetitive tasks and increase selective attention in abstinent smokers. The effects of nicotine on the cognitive functioning of non-smokers have not been clearly identified (USDHHS 1988; Heishman et al. 1994). Smokers commonly report pleasure, mental stimulation, and reduction of stress after smoking a cigarette (McKennell 1970; Russell et al. 1974).

Cigarette smoking and nicotine also have sympathomimetic action, producing brief increases in blood pressure, heart rate, and cardiac output with cutaneous vasoconstriction (Benowitz 1988). Nicotine causes muscle relaxation by stimulating discharge of the Renshaw cells and pulmonary afferent nerves, which inhibit motor neuron activity and relax certain muscles. However, not all muscles are relaxed; increased electromyographic activity and tonicity of the large upper-back muscles (trapezius) have been observed after smoking (Fagerström and Götestam 1977).

Table 15. Human pharmacology of nicotine

Primary effects*	Withdrawal symptoms
Pleasure	Irritability, restlessness
Arousal, enhanced vigilance	Drowsiness
Improved task performance	Difficulty concentrating, impaired task performance
Relief of anxiety	Anxiety
Reduced hunger	Hunger
Body weight reduction	Weight gain
	Sleep disturbance
	Cravings or strong urges for nicotine
Electroencephalogram desynchronization	
Increased circulating levels of catecholamines, vasopressin, growth hormone, adrenocorticotropic hormone (ACTH), cortisol, prolactin, beta-endorphin	Decreased catecholamine excretion†
Increased metabolic rate	
Lipolysis, increased free fatty acids	
Heart rate acceleration	Heart rate slowing†
Cutaneous and coronary vasoconstriction	
Increased cardiac output	
Increased blood pressure	
Skeletal muscle relaxation	

*Some of these effects are related in part to relief of withdrawal symptoms.
†May represent a return to baseline rather than true withdrawal.
Source: Benowitz 1992a.

Genetic differences in the number of nicotinic receptors and pharmacologic responses to nicotine have been well demonstrated in animals (Marks et al. 1991). Genetic differences in pharmacologic responses to nicotine could underlie different susceptibilities to nicotine addiction, as appears to be the case for certain types of alcohol addiction (Hughes 1986; Cloninger 1987; Carmelli et al. 1992). Genetic susceptibility may vary by ancestry of origin (for example, sickle cell disease and African American ancestry). Genetic differences in nicotine responsiveness associated with ancestry of origin remain to be explored.

Tolerance, Withdrawal, and Addictive Tobacco Use

With prolonged or repeated exposure to nicotine, neurologic changes (neuroadaptation) occur. In animals, chronic nicotine exposure results in an increased number of nicotinic receptors in the brain (Marks et al. 1985). During the course of these changes, the smoker develops more brain nicotinic receptors and an increased tolerance to the various effects of nicotine. For example, previous studies have shown that at autopsy, the number of nicotinic receptors was greater in the brains of cigarette smokers than in those of nonsmokers (Benwell et al. 1988). Smokers develop substantial tolerance to the behavioral arousal and cardiovascular effects of nicotine in the course of a single day (Benowitz et al. 1989b). They can regain sensitivity to the effects of nicotine, at least in part, after overnight abstinence from smoking.

As a consequence of these neurologic changes, nicotine withdrawal symptoms appear when nicotine use is abruptly stopped (Table 16) (Hughes and Hatsukami 1992). Withdrawal symptoms include restlessness, irritability, anxiety, drowsiness, impatience, confusion, impaired concentration, and depression (Hughes et al. 1990). Some abstaining smokers gain weight, and others have impaired performance measures, such as reaction time. Many abstaining

Table 16. Incidence* of nicotine withdrawal symptoms, United States

Symptom	Clinic attendees (%)	Self-quitters (%)
Anxiety	87	49
Irritability	80	38
Difficulty concentrating	73	43
Restlessness	71	46
Hunger	67	53
Craving	62	37
Nocturnal awakenings	24	39
Depression	NA	31

*Percentage of subjects with postcessation ratings greater than precessation ratings 2 days after they quit smoking.
NA = data not available.
Sources: Hughes 1992; Hughes and Hatsukami 1992. Adapted from Hughes and Hatsukami 1992.

smokers have a strong craving to smoke a cigarette. Most of the withdrawal symptoms reach maximal intensity 24 to 48 hours after cessation and gradually diminish in intensity within three to four weeks (Gross and Stitzer 1989; Hughes et al. 1990), although some individuals experience longer lasting symptoms (USDHHS 1988). These symptoms, which also appear after quitting the use of smokeless tobacco (CDC 1994) or nicotine gum, are relieved following the administration of nicotine—a strong indication that the withdrawal symptoms are related to the effects of nicotine.

The degree of nicotine dependence is determined in part by the level of nicotine that accumulates in smokers. In general, the level of accumulated nicotine is proportional to the number of cigarettes smoked per day. Consistent with the concept of a daily tolerance-withdrawal cycle, a short duration of time between awakening and smoking the first cigarette is associated with a high degree of nicotine dependence (Heatherton et al. 1989). This presumably reflects an effort to relieve nicotine withdrawal symptoms. These two factors—the number of cigarettes smoked per day and the amount of time from awakening to smoking the first cigarette—are commonly used to assess the severity of nicotine dependence (Fagerström and Schneider 1989).

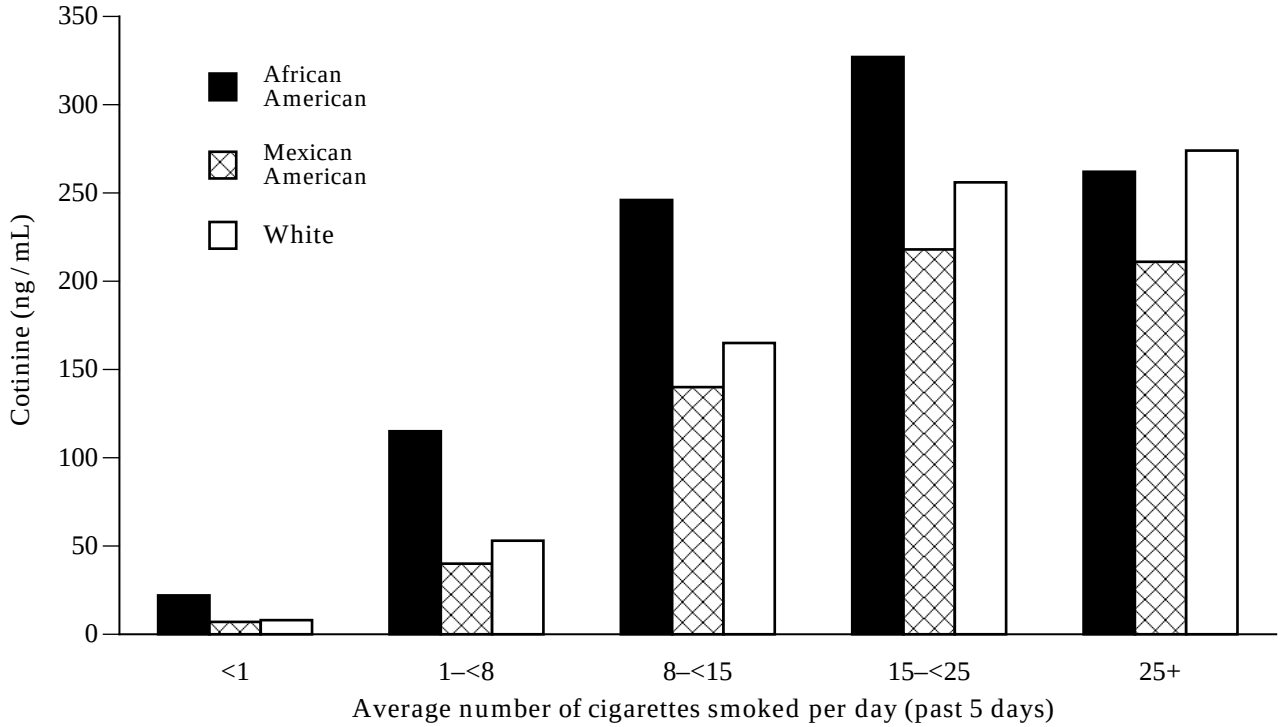
Level of Addiction

Assessments of the level of nicotine addiction help predict responses to nicotine and serve as a potential guideline for therapeutic approaches to smoking cessation. The professionals who design strategies to prevent tobacco use and treat persons with nicotine addiction need to understand the high level of addiction among cigarette smokers and to appreciate the group-specific cultural characteristics of the behavior and smokers' individual reasons for initiating, continuing, and quitting tobacco use (Krasnegor 1979; Grunberg and Acri 1991). The most widely used indexes of addiction levels are the number of cigarettes smoked per day, the serum nicotine or cotinine level, the Fagerström dependence questionnaire (Fagerström and Schneider 1989), and the diagnostic criteria of the *DSM-IV*™ (APA 1994). The Fagerström dependence questionnaire incorporates questions about the number of cigarettes smoked per day, the time between awakening and smoking the first cigarette of the day, as well as episodes in which the smoker lost control of smoking behavior (such as smoking at inappropriate times or in inappropriate places). The prevalence of smoking cessation—and conversely, the number of unsuccessful quit attempts—also reflects the level of addiction, at least in part. The brand of cigarette smoked might be expected to correlate with a person's level of dependence because high-yield cigarettes nominally deliver more nicotine per cigarette. However, in large surveys of smokers, only a modest relationship was found between yield (measured by a smoking machine) and levels of nicotine or cotinine in the body (Benowitz et al. 1986; Coultas et al. 1993). This is because people smoke differently than machines that are set to a standardized testing protocol—that is, they are able to take more frequent or deeper puffs, to smoke each cigarette more completely, to smoke more cigarettes per day, and to block ventilation holes in the cigarettes (Henningfield et al. 1994; NCI 1996a).

Racial/Ethnic Differences in Nicotine Metabolites

Evidence suggests that African Americans have higher cotinine levels per reported number of cigarettes smoked per day than whites (Wagenknecht et al. 1990; English et al. 1994; Clark et al. 1996a) (Figure 5). In Figure 5, the racial / ethnic minority group comparisons among those who smoked 25 or more cigarettes per day may be somewhat biased, because the average daily consumption for whites was substantially higher than that for African Americans and Mexican

Figure 5. Serum cotinine levels by number of cigarettes smoked daily for African Americans, Mexican Americans, and whites, National Health and Nutrition Examination Survey, United States, 1988–1991



Note: N = 2,136.
Source: National Center for Health Statistics, public use data tape, 1997.

Americans. Clark and colleagues (1996b) found no evidence that underreporting of daily cigarette consumption occurred more often in African American than in white smokers.

One possible explanation for the higher cotinine level among African Americans is that African Americans may absorb more nicotine from their cigarettes than whites (Benowitz et al. 1995). Greater absorption could result from several factors, including group-specific patterns of smoking behavior (i.e., more and deeper puffs per cigarette or longer retention of tobacco smoke in the lungs) (Benowitz et al. 1995). Additionally, menthol in cigarettes may facilitate absorption of cigarette smoke constituents (Jarvik et al. 1994; McCarthy et al. 1995; Clark et al. 1996a). However, the fact that African Americans smoke menthol cigarettes more commonly than whites do explains only a small percentage of their higher levels of cigarette smoke constituents (Wagenknecht et al. 1992; Ahijevych et al. 1996; Clark et al. 1996a).

Racial / ethnic differences in nicotine metabolism could influence the development of nicotine addiction. Several researchers have suggested that African Americans might metabolize cotinine differently than whites (Pattishall et al. 1985; Wagenknecht et al. 1990; English et al. 1994; Benowitz et al. 1995). Results of studies of nonsmokers support this hypothesis (Pattishall et al. 1985; Wagenknecht et al. 1993; Crawford et al. 1994; Knight et al. 1996; Pirkle et al. 1996). Most of these investigations (Pattishall et al. 1985; Crawford et al. 1994; Knight et al. 1996; Pirkle et al. 1996) reported that African Americans had higher cotinine levels than whites, even after ETS exposure and other factors were taken into account. These findings may be limited by the fact that no measures of tobacco smoke or nicotine concentrations in the air were obtained.

Based on a preliminary report of data for 40 African Americans and 39 white controls matched for age, gender, and cigarette consumption, Benowitz and colleagues (1995) reported that the disposition kinetics of nicotine were similar for both groups. For example, the percentage conversion of nicotine to cotinine was similar across groups. However, the clearance of cotinine was significantly lower for African Americans than for whites. Additionally, the average estimated intake of nicotine per cigarette smoked was 1.41 mg in African Americans and 1.09 mg in whites. This difference is of borderline statistical significance ($p = 0.07$) (Benowitz et al. 1995). African Americans took in 28 percent more nicotine per cigarette than would have been expected based on FTC yields; whites took in 9 percent more nicotine per cigarette than would have been expected based on FTC yields (Pérez-Stable et al., unpublished data).

Investigators have also found cotinine levels in African Americans that were higher than expected for the number of cigarettes smoked. Ahijevych and Wewers (1993) found an average salivary cotinine level of 402 ng/mL in African American women who smoked an average of 15 cigarettes per day. This level is much higher than the expected level found in other persons who smoked the same number of cigarettes. Clark and colleagues (1996b) reported that African American smokers smoked longer cigarettes and more of each cigarette than white smokers. However, because they smoked fewer cigarettes each day, African Americans smoked fewer total daily millimeters of cigarettes. Among young adults in the CARDIA study, African Americans (48 percent) were more likely than whites (36 percent) to report that a substantial amount of their cigarette burned without their smoking it (Wagenknecht et al. 1992). Also, in a study of 33 African American and white women, Ahijevych and colleagues (1996) did not find a racial/ethnic difference in total puff volume (per cigarette).

Pérez-Stable and colleagues (1990) reported that among Mexican Americans who were part of the 1982–1984 HHANES, cotinine levels were unexpectedly high in smokers reporting low levels of cigarette consumption. Higher-than-expected cotinine levels may reflect underreporting of smoking by Hispanics, but the possibility also exists that Hispanics absorb or metabolize nicotine differently than whites (Henningfield et al. 1990). However, recent data from NHANES III (Figure 5) indicate that, among persons who smoked at least one cigarette daily, Mexican American smokers had lower serum cotinine levels in each consumption category than African American and white smokers.

Racial/Ethnic Differences in Self-Reported Nicotine Dependence

The use of questionnaires to systematically investigate racial/ethnic differences in nicotine dependence has been limited. Data from the 1987 NHIS (Table 17) show that African Americans were more likely than whites and Hispanics to report smoking their first cigarette of the day within 10 minutes of awakening, although these differences tended to disappear among those who reported smoking 25 or more cigarettes per day (NCHS, public use data tapes, 1987). Telephone survey data on smoking, collected as part of the Community Intervention Trial (COMMIT) for Smoking Cessation, also indicate that African Americans were more likely than whites to smoke within 10 minutes of awakening (an indicator of nicotine dependence [USDHHS 1988]), even after the researchers controlled for the number of cigarettes smoked per day (Royce et al. 1993). Conversely, Andreski and Breslau (1993) conducted a study that used the dependence criteria of the *DSM-III*™ and found that, compared with African Americans, greater proportions of whites had symptoms of nicotine dependence. The researchers randomly selected 1,200 adults aged 21–30 years from the members of a health maintenance organization in southeast Michigan. Overall, 22.6 percent of the whites who smoked met the criteria for nicotine dependence, compared with 9.3 percent of the African Americans who smoked. Nicotine dependence was found to have a significant association with psychological distress, as measured by the Brief Symptom Inventory for smokers in both groups. Poor physical health was also associated with nicotine dependence, and this relationship was stronger among African Americans than among whites.

Kandel and colleagues (1997) used questions from the 1991, 1992, and 1993 (combined) National Household Surveys on Drug Abuse (NHSDAs) to develop a proxy measure of *DSM-IV*™ (APA 1994) dependence on various substances (including nicotine). Respondents were asked, for example, if they felt unable to reduce their use when they tried to cut down, experienced withdrawal symptoms (described in this survey as feeling sick because they stopped or cut down), felt that they needed or were dependent on the substance, and felt the need for larger amounts to obtain the same effect. This study used responses from 87,915 persons aged 12 years and older. Among persons who smoked during the previous year, whites were more likely than African Americans, Hispanics, and other racial/ethnic minority group members to be rated as dependent on nicotine. The authors

Table 17. Percentage of adult smokers* who reported that they smoked their first cigarette within 10 minutes and within 30 minutes of awakening, by race/ethnicity and number of cigarettes smoked per day, National Health Interview Survey, United States, 1987

	African Americans		Hispanics		Whites	
Characteristic	**%**	**±CI†**	**%**	**±CI**	**%**	**±CI**
1–14 cigarettes						
≤10 minutes	21.9	4.9	11.3	5.3	11.1	2.1
≤30 minutes	39.2	5.5	26.2	7.3	27.1	3.0
15–24 cigarettes						
≤10 minutes	51.7	8.4	32.7	10.3	36.9	2.4
≤30 minutes	77.6	5.9	61.3	10.3	68.4	2.5
≥25 cigarettes						
≤10 minutes	69.0	18.0	63.3	17.2	61.9	3.0
≤30 minutes	95.6	3.6	93.4	8.2	88.8	1.8

*Persons who reported smoking at least 100 cigarettes in their lives and who reported at the time of survey that they currently smoked.
†95% confidence interval.
Source: National Center for Health Statistics, public use data tapes, 1987.

acknowledged that their study was limited somewhat because the NHSDA indicators of dependence were not based on diagnostic interviews designed specifically to assess *DSM-IV*™ criteria. Nevertheless, the finding that whites were more likely to exhibit indicators of dependence than African Americans was consistent with that of Andreski and Breslau (1993). Further research is needed to resolve the apparent discrepancy for African Americans between studies that are based on the number of minutes to the first cigarette of the day and those that are based on *DSM-III*™ or *DSM-IV*™ criteria for dependence.

Navarro (1996) used population-based data from the 1990 California Tobacco Survey on white (n = 70,997) and Hispanic (n = 28,000) adults. Her analyses indicated that whites were significantly more likely than Hispanics to smoke on a daily basis and to smoke at least 15 cigarettes each day. Furthermore, among the daily smokers, whites were more likely than Hispanics to smoke a cigarette within 30 minutes of awakening. Among Hispanics, those who were less acculturated (i.e., who came from households where the language spoken in the household was not English) were significantly less likely than those who were more acculturated (i.e., who came from households where English was the language spoken) to be daily smokers and to smoke at least 15 cigarettes each day. Among Hispanics who were daily smokers, the percentage who smoked within 30 minutes of awakening did not differ significantly by level of acculturation.

Smoking to maintain a lower body weight is believed to contribute to tobacco dependence. In a survey of high school students in Memphis, Tennessee, Camp and colleagues (1993) found that more whites than African Americans believed that cigarette smoking could help them control their body weight. Among the high school students who smoked, 39 percent of white females and 12 percent of white males reported smoking to control their body weight, compared with none of the African American students.

A few studies have analyzed the perceptions that members of racial/ethnic groups have regarding the addictive nature of tobacco. In a San Francisco area study of 2,835 primary care patients who smoked, Vander Martin and colleagues (1990) found that whites smoked more cigarettes per day and were more likely to consider themselves addicted to cigarettes than African American, Asian American, and Hispanic smokers. Smoking within 15 minutes of awakening was least likely among Hispanic smokers but equally common among smokers in the other groups. In addition, African Americans and Hispanics were less likely than the others to believe that quitting smoking would lead to weight gain.

Most Americans of all races and ethnicities realize that cigarette smoking is addictive. In a survey of 2,092 adults in St. Louis and Kansas City, Missouri, Brownson and colleagues (1992) found that a similar number of whites (90.3 percent) and African Americans (88.5 percent) believed cigarette smoking was addictive. Results from the 1992–1993 CPS (see Chapter 5, Research and Development Limitations) showed that most members of the four racial / ethnic groups as well as whites agreed with the statements that cigarette smoking was an addiction or both a habit and an addiction (Table 18) (U.S. Bureau of the Census, NCI Tobacco Use Supplement, public use data tapes, 1992–1993). Minor differences across gender were observed, although smokers were somewhat less likely to agree with the statements. Approximately 5 percent of the Asian American and Hispanic smokers indicated that cigarette smoking was neither a habit nor an addiction, compared with 1.9 percent of white smokers.

Racial/Ethnic Differences in Quitting Smoking

Because nicotine is addictive, highly addicted smokers have great difficulty in quitting. Differences in quitting can be used as another measure of the level of dependence. Some studies have found that although a similar percentage of whites and African Americans have ever been smokers, the percentage of former smokers has been greater among whites (26.4 percent) than among African Americans (17.2 percent) (Novotny et al. 1988) (see also Chapter 2). Data for 1989 from the BRFSS indicate that the standardized prevalence of smoking cessation was 47 percent among whites vs. 39.1 percent among African Americans (prevalence of cessation was defined as the percentage of ever smokers who were former smokers) (CDC 1990). Similar findings were reported by Kabat and Wynder (1987), Hahn and colleagues (1990), and Geronimus and colleagues (1993). The 1991 NHIS Health Promotion and Disease Prevention supplement collected data on smokers who had quit for at least one day at the time of survey and for at least one month in the previous year (CDC 1993b). Hispanics (52.1 percent) and African Americans (48.7 percent) were more likely than whites (40.3 percent) to have quit smoking for one day. However, data on abstinence from smoking in the previous year showed that Hispanics (16.3 percent) and whites (14.0 percent) were more likely than African Americans (7.9 percent) to have quit smoking for one month or longer. Thus, African Americans were less likely than whites to maintain abstinence. This effect remained after the findings were controlled for socioeconomic status. In an unadjusted analysis of data from the Current Population Survey NCI Supplement, a similar pattern was observed, although the differences between African Americans and whites were slight (see Table 2 and African Americans, Quitting Behavior in Chapter 2).

The lower smoking cessation rates among African Americans do not appear to result from a lack of desire to quit (Royce et al. 1993). In the COMMIT telephone survey, 46.0 percent of African American women and 44.4 percent of African American men stated that they wanted to quit smoking "a lot," compared with 35.0 percent of white women and 33.3 percent of white men. Thus, the lower prevalence of cessation among African Americans may be related to factors other than the desire to quit, such as the absence of culturally appropriate smoking cessation interventions, difficulties in accessing community resources for quitting smoking, and possibly a higher level of nicotine dependence as indicated by comparatively higher levels of cotinine when the data are controlled for the number of cigarettes smoked.

Addiction to Smokeless Tobacco

Considerable nicotine is absorbed from smokeless tobacco. An average systemic dose of nicotine is 3.6 mg for snuff, 4.6 mg for chewing tobacco, and 1.8 mg for cigarettes (Benowitz et al. 1988). Blood nicotine concentrations throughout the day are similar among smokers and those who use smokeless tobacco (Benowitz et al. 1989a). Plasma cotinine levels in regular smokeless tobacco users are often similar to the levels in cigarette smokers (Holm et al. 1992). Abstinence from smokeless tobacco use results in signs and symptoms of nicotine deprivation that are similar to those seen in smokers after they stop smoking (Hatsukami et al. 1987; CDC 1994). These symptoms are reversed by the use of tobacco or administration of nicotine gum. In a study of Swedish oral snuff users, many of the participants considered themselves addicted to snuff, and they reported having as much difficulty giving up smokeless tobacco use as was reported by cigarette smokers trying to quit smoking (Holm et al. 1992). Evidence also suggests that when regular snuff users are deprived of snuff, they will smoke cigarettes to satisfy their need for nicotine (Benowitz 1992b). However, no data are available on racial or ethnic differences in the level of addiction to smokeless tobacco.

Table 18. Percentage of men and women who considered smoking a habit or addiction,* overall and by smoking status, Current Population Survey, United States, 1992–1993

Characteristic	African Americans		American Indians/ Alaska Natives		Asian Americans/ Pacific Islanders		Hispanics		Whites	
	%†	±CI‡	%	±CI	%	±CI	%	±CI	%	±CI
Overall										
Habit	31.7	0.7	19.6	2.6	23.9	1.4	25.1	0.8	17.8	0.2
Addiction	19.8	0.6	19.6	2.6	17.8	1.2	26.3	0.8	21.9	0.2
Both	41.3	0.7	54.6	3.3	46.4	1.6	38.4	0.9	57.0	0.3
Men										
Habit	32.3	1.1	19.5	3.9	25.5	2.0	26.4	1.2	19.3	0.3
Addiction	20.4	0.9	21.4	4.0	18.4	1.8	26.7	1.2	22.0	0.3
Both	39.5	1.1	52.6	4.9	45.8	2.3	36.7	1.3	55.2	0.4
Women										
Habit	31.3	0.9	19.6	3.5	22.5	1.9	24.0	1.0	16.5	0.3
Addiction	19.5	0.8	18.1	3.4	17.2	1.7	25.9	1.1	21.9	0.3
Both	42.5	0.9	56.2	4.4	46.9	2.2	39.8	1.2	58.6	0.4
Nonsmokers										
Habit	29.8	0.8	18.3	3.3	21.7	1.4	23.5	0.8	16.4	0.2
Addiction	20.4	0.7	21.1	3.5	18.9	1.4	27.1	0.9	23.0	0.3
Both	42.9	0.8	54.6	4.2	47.5	1.8	39.4	1.0	57.7	0.3
Men										
Habit	30.3	1.3	19.8	5.3	22.2	2.2	24.6	1.3	18.0	0.4
Addiction	20.5	1.1	22.4	5.5	20.2	2.1	27.9	1.4	22.8	0.4
Both	41.6	1.4	51.4	6.6	48.1	2.6	38.0	1.5	56.1	0.5
Women										
Habit	29.6	1.0	17.3	4.2	21.3	1.9	22.7	1.1	15.0	0.3
Addiction	20.3	0.9	20.2	4.5	17.8	1.8	26.5	1.1	23.1	0.4
Both	43.7	1.1	56.8	5.5	47.0	2.4	40.4	1.3	59.0	0.4
Smokers										
Habit	36.6	1.4	21.5	4.4	36.0	3.9	32.7	2.0	22.1	0.5
Addiction	18.6	1.1	17.5	4.0	12.3	2.7	22.6	1.7	18.9	0.4
Both	37.2	1.4	54.4	5.3	40.9	4.0	34.1	2.0	55.2	0.6
Men										
Habit	36.4	2.0	19.4	5.9	36.6	4.7	32.3	2.5	22.9	0.7
Addiction	20.2	1.7	20.5	6.1	12.6	3.2	23.3	2.3	19.7	0.6
Both	35.1	2.0	53.6	7.5	38.3	4.7	32.8	2.5	53.0	0.8
Women										
Habit	36.7	1.9	23.7	6.4	34.6	7.1	33.2	3.1	21.2	0.7
Addiction	17.2	1.5	14.4	5.3	11.5	4.8	21.4	2.7	18.1	0.6
Both	39.0	1.9	55.2	7.5	47.0	7.5	36.1	3.2	57.3	0.8

*In response to the question, "Do you think smoking is a habit, an addiction, neither, or both?"
†Percentages in this table do not include all categories of responses and thus may not equal 100%.
‡95% confidence interval.
Source: U.S. Bureau of the Census, National Cancer Institute Tobacco Use Supplement, public use data tapes, 1992–1993.

Conclusions

1. Cigarette smoking is a major cause of disease and death in each of the four racial/ethnic groups studied in this report. African Americans currently bear the greatest health burden. Differences in the magnitude of disease risk are directly related to differences in patterns of smoking.
2. Although lung cancer incidence and death rates vary widely among the nation's racial/ethnic groups, lung cancer is the leading cause of cancer death for each of the racial/ethnic groups studied in this report. Before 1990, death rates from malignant neoplasms of the respiratory system increased among African American, Hispanic, and American Indian and Alaska Native men and women. From 1990 through 1995 death rates from respiratory cancers decreased substantially among African American men, leveled off among African American women, decreased slightly among Hispanic men and women, and increased among American Indian and Alaska Native men and women.
3. Rates of tobacco-related cancers (other than lung cancer) vary widely among members of racial/ethnic groups, and they are particularly high among African American men.
4. The effect of cigarette smoking (as reflected by biomarkers of tobacco exposure) on infant birth weight appears to be the same in African American and white women. As reported in previous Surgeon General's reports, cigarette smoking increases the risk of delivering a low-birth-weight infant.
5. No significant racial/ethnic group differences have been consistently demonstrated in the relationship between smoking and infant mortality or sudden infant death syndrome (SIDS); cigarette smoking has been associated with increased risk of SIDS and remains a probable cause of infant mortality.
6. Future research is needed and should focus on how tobacco use affects coronary heart disease, stroke, cancer, chronic obstructive pulmonary disease, and other respiratory diseases among members of racial/ethnic groups. Studies also are needed to determine how the health effects of smokeless tobacco use and exposure to environmental tobacco smoke vary across racial/ethnic minority groups.
7. Persons of all racial/ethnic backgrounds are vulnerable to becoming addicted to nicotine, and no consistent differences exist in the overall severity of addiction or symptoms of addiction across racial/ethnic groups.
8. Levels of serum cotinine (a biomarker of tobacco exposure) are higher in African American smokers than in white smokers for similar levels of daily cigarette consumption. Further research is needed to clarify the relationship between smoking practices and serum cotinine levels in U.S. racial/ethnic groups. Variables such as group-specific patterns of smoking behavior (e.g., number of puffs per cigarette, retention time of tobacco smoke in the lungs), rates of nicotine metabolism, and brand mentholation could be explored.

Appendix. Methodological Issues

It is important to review some methodological issues involved in collecting the data discussed in this chapter. These methodological problems affect the quality of the data and the type of conclusions that can be reached from studies conducted to date. Also, because cigarette smoking tends to be associated with other lifestyle risk factors that impact on health (e.g., Wingard et al. 1982; Vickers et al. 1990; Pérez-Stable et al. 1994), there is a need to control their co-occurrence in order to better understand the health effects of tobacco use.

Classification of Smoking Status

In investigating the health effects of smoking cigarettes and using other tobacco products, researchers typically obtain information from the subjects or surrogate respondents on the use of such products. Questionnaires usually cover cigarette smoking status (i.e., never, former, and current smoker), number of years of smoking and age at initiation of smoking, number of cigarettes smoked per day, and use of other tobacco products (e.g., pipes, cigars, and smokeless

tobacco). However, this information may not be fully valid, resulting in misclassification of exposure to cigarette smoking. A previous report of the Surgeon General reviewed the classification of cigarette smoking status and the consequences of misclassification (USDHHS 1990).

Misclassification of smoking information merits consideration in investigating tobacco use among racial / ethnic populations, because of the potential for bias in comparing the effects of smoking across racial / ethnic groups. To date, such bias has not been identified, although several studies show that Hispanics may underreport cigarette smoking. In a population-based survey in New Mexico, Coultas and colleagues (1988) compared self-reports of smoking against salivary cotinine level (a product of nicotine that has been used as a measure of exposure to nicotine) and end-tidal carbon monoxide concentration. Based on the questionnaire results, the age-standardized prevalence rates of current smoking were 30.9 and 27.1 percent for Hispanic men and women, respectively. After adjusting for cotinine and carbon monoxide levels, these percentages were 39.1 and 33.2. The rate of misclassification was greater in self-reported former smokers than in never smokers, but self-reported never smokers also had levels of cotinine and carbon monoxide indicative of active smoking.

Using information from the Hispanic Health and Nutrition Examination Survey (HHANES), Pérez-Stable and colleagues (1992) documented the misclassification of smoking status through comparisons of self-reports with serum cotinine levels. Among 65 Mexican American former smokers participating in the HHANES in 1982 through 1983, 7 (10.8 percent) had a cotinine level indicative of active smoking; among 124 reported never smokers, 5 (4 percent) were probably active smokers based on their cotinine levels. In a number of surveys, Hispanics, particularly Latino groups in the southwestern and western United States, have been found to smoke about one-half pack of cigarettes per day, compared with non-Hispanic whites who typically report smoking one pack per day (Coultas et al. 1994). Pérez-Stable and colleagues (1992) used data from 547 Mexican American participants in the HHANES to examine underreporting of cigarette consumption using the ratio of serum cotinine to self-reports of the number of cigarettes smoked per day as the "gold standard." This study found that among Mexican Americans, 20.4 percent of men and 24.7 percent of women who were self-reported smokers underreported smoking between one and nine cigarettes per day. Self-reported Mexican American smokers who reported smoking greater numbers of cigarettes per day underreported less frequently.

An analysis of the data from the Coronary Artery Risk Development in (Young) Adults Study (CARDIA) showed that there were higher rates of misclassification in terms of self-reported nonsmokers who had serum cotinine levels of at least 14 ng / mL among African Americans (5.7 percent) than among non-Hispanic whites (2.8 percent) (Wagenknecht et al. 1992). Alternative explanations for underreporting, such as more efficient smoking and differences in cotinine metabolism, could not be excluded.

Two additional studies examined the relationship between ancestry of origin and levels of biochemical markers in smokers. In a study of participants in CARDIA, African American smokers demonstrated higher cotinine levels than non-Hispanic white smokers after controlling for several dimensions of cigarette-smoking behavior (Wagenknecht et al. 1990). Lactose intolerance, which elevates breath hydrogen concentration, may increase the apparent level of expired air carbon monoxide, a readily measured marker of active smoking (McNeill et al. 1990). Lactose intolerance is common in a number of racial / ethnic groups, including Asian Americans and African Americans.

Classification of Race/Ethnicity

The data included in this chapter are derived from diverse sources, including vital statistics, cancer registries, and epidemiological studies on smoking. Race / ethnicity has been classified in these studies using various techniques, including designation on death certificate, classification according to cancer registry protocols, self-reports, birthplace, language use, and surname. The validity of each of these approaches is undoubtedly imperfect; moreover, validity varies across regions and over time. However, comprehensive assessments of the validity of racial / ethnic minority classification in various types of health data have not been reported.

The limited information available indicates some potential for misclassification. For example, Frost and colleagues (1992) compared the classification of "Native American," as recorded by the Seattle-Puget Sound registry of the Surveillance, Epidemiology, and End Results (SEER) Program against an Indian Health Service (IHS) registry of patients eligible for services. A substantial portion of patients with invasive cancer in the IHS registry were not similarly classified by the Seattle-Puget Sound cancer registry. Similarly, an injury registry for the state of Oregon undercounted those with injuries (Sugarman et al. 1993). Using data from the National Longitudinal Mortality Study, Sorlie and colleagues (1992) compared demographic characteristics reported on the CPS of the

U.S. Bureau of the Census with those characteristics reported on the death certificates for persons who died (during a seven-year follow-up period). Among 216 persons identified as American Indians or Alaska Natives by the CPS, only 159 (73.6 percent) were so classified on the death certificate. Similarly, the concordance rate for 272 persons classified by the CPS as Asian Americans or Pacific Islanders was 82.4 percent. Such disagreement suggests that current estimates of mortality rates for selected racial/ethnic groups are underestimated. However, in New Mexico, the classification of "American Indian" by the New Mexico Tumor Registry, also a participant in the SEER Program, closely corresponded with the classification by the state's Bureau of Vital Statistics (Eidson et al. 1994).

Another study in New Mexico also showed a high concordance between self-reported Hispanic race/ethnicity and the designation by the Bureau of Vital Statistics (Samet et al. 1988b). In the report by Sorlie and colleagues (1993), 10.3 percent (n = 62) of persons identified as Hispanics by the CPS were not classified as Hispanics on the death certificate. Surnames also have been used to classify Hispanic ethnicity, using either surname lists developed by the U.S. Bureau of the Census or name recognition algorithms (Howard et al. 1983; Wiggins and Samet 1993). Although studies in parts of the southwestern United States have shown a generally high validity for surname-based approaches for identifying Hispanic ethnicity, the sensitivity and specificity of the various Census Bureau lists have varied over time, and data from the Southwest cannot be readily generalized to other locales. In addition, surname lists tend to exclude women who marry non-Hispanic whites and who take their husband's last name and to exclude as well their children when given the father's non-Hispanic last name (Marín and Marín 1991).

These studies suggest that the validity of classification of race/ethnicity is likely to vary across locations and possibly by type of data. In interpreting health data for racial/ethnic populations, consideration should be given to the potential for misclassification of race/ethnicity and the consequences of any resulting bias.

Classification of Health Outcomes

Comparisons of disease occurrence among racial/ethnic groups also may be biased by differential patterns of disease diagnosis and labeling by race and ethnicity. Such differences may have multiple causes that reflect the complex sequence that begins with the development of symptoms and signs and extends to the labeling of an illness by a clinician or the statement of cause-of-death on a death certificate. Health beliefs and knowledge, ability to access and pay for medical care, the quality of care available, and differential patterns of care by race/ethnicity may all affect diagnoses of illnesses. A full review of these topics is beyond the scope of this report, but several examples are offered to illustrate the potential for differential patterns of classification of health outcomes by race/ethnicity.

Becker and colleagues (1990) examined the assignment of underlying cause of death to the category "symptoms, signs, and ill-defined conditions" in the *Manual of the International Classification of Diseases, Injuries and Causes of Death* (*ICD*). In the nation, the crude death rate for this non-specific category has paralleled the mortality rate in this category for African Americans. Becker and colleagues (1990) analyzed vital statistics data for New Mexico for 1958 through 1982 and calculated mortality rates for "symptoms, signs, and ill-defined conditions" by racial/ethnic group. The state mortality rates for Hispanics, non-Hispanic whites, and American Indians for this category exceeded the nationwide rates. Among the racial/ethnic minority groups in New Mexico, American Indians had particularly high mortality rates; for men, 8.4 percent of American Indian deaths were in this category versus 5.9 percent of Hispanic deaths and 5.0 percent of non-Hispanic white deaths. Similarly, mortality rates for cancers of ill-defined and unknown primary sites tend to be much higher in American Indians in several areas of the country than for all racial/ethnic groups combined (Valway 1992).

Recent comparisons of the evaluation and management of chest pain and coronary artery disease in African Americans and non-Hispanic whites further illustrate the potential for bias by race/ethnicity in diagnostic classification. In a study of patients presenting to an emergency room with chest pain, African Americans were less likely to be admitted and less likely to be sent to a coronary care unit once they were admitted (Johnson et al. 1993). The study also found that African Americans were as likely as non-Hispanic whites to have cardiac catheterization. In contrast, other studies, using Department of Veterans' Affairs, Medicare, and other large data bases, have shown that African Americans are less likely than non-Hispanic whites to have cardiac catheterization and invasive interventions for coronary artery disease (Wenneker and Epstein 1989; Udvarhelyi et al. 1992; Ayanian et al. 1993; Franks et al. 1993; Whittle et al. 1993; Peterson et al. 1994). These differential patterns of evaluation by race/ethnicity could introduce bias in investigations of tobacco smoking and coronary artery disease among African Americans and non-Hispanic whites by underestimating the effects of cigarette smoking on coronary artery disease.

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