

# State of the Art

## Respiratory Diseases in Minorities of the United States

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The population of the United States includes many minority groups with diverse racial and ethnic heritages (table 1) (1, 2). The people in these groups have patterns of health, disease, and illness\* (4) that differ from those of the white majority of the United States. Conditions of the respiratory tract are no exception: even mortality data, a crude measure of disease impact, show some striking effects of race and ethnicity on the major diseases of the respiratory tract (tables 2 and 3) (5, 6). The purpose of this review is to provide clinicians and researchers with a comprehensive overview of respiratory diseases among the minority populations of the United States, including blacks, Hispanics, Asians-Pacific Islanders, and American Indians-Alaska Natives (7). The review addresses risk factors for the major respiratory diseases, the occurrence of these respiratory diseases in children and adults, and related clinical issues.

In the 1990 U.S. Census, minority groups constituted 24% of the population, or about 60 million people (table 1). Blacks were the largest group, followed by Hispanics, Asians-Pacific Islanders, and American Indians-Alaska Natives. Although these groups constitute a minority of the total population, their overall growth of 32% during the 10-yr period from 1980 to 1990 far exceeds the 6% increase among whites (table 1). Asians-Pacific Islanders had the largest growth during the period, with a 108% increase, Hispanics were second with 53%, followed by American Indians-Alaska Natives with 38% and blacks with 13%. The minority populations tend to be younger than the white majority and to have a smaller proportion of elderly.

Minority populations are special groups within the population that are distinct by racial or ethnic heritage. Race is a term that cannot be readily defined on a unified scientific basis; it incorporates notions of biologic and geographic origins (7, 8). Ethnic-

\*Health is defined as "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity." Disease is a physiologic/psychologic dysfunction, and illness is a subjective state of the person who feels aware of not being well (3).

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TABLE 1  
DISTRIBUTIONS OF RACE AND HISPANIC ORIGIN IN U.S.  
POPULATION AND PERCENT CHANGE, 1980 TO 1990\*

| Race                      | 1980<br>(n × 1,000) | 1990<br>(n × 1,000) | Change<br>from 1980<br>(%) |
|---------------------------|---------------------|---------------------|----------------------------|
| White                     | 188,372             | 199,686             | 6                          |
| Black                     | 26,495              | 29,986              | 13                         |
| American Indian†          | 1,420               | 1,959               | 38                         |
| Asian/Pacific<br>Islander | 3,501               | 7,274               | 108                        |
| All other                 | 6,758               | 9,805               | 45                         |
| Hispanic origin‡          | 14,609              | 22,354              | 53                         |

\* Source: 1980 (1) and 1990 (2) Censuses.

† Includes Eskimo and Aleut.

‡ Persons of Hispanic origin may be of any race.

ity, in contrast, refers to cultural identification and origins (4, 9). A single ethnic group may include peoples from different races; for example, Hispanics in the United States may be black, white, or members of another racial group. Race and ethnicity cannot be rigidly defined, nor can strict and fully valid criteria be set out for classifying individuals. Concepts of race and ethnicity are fluid, and they vary over time with changing societal perceptions.

Nevertheless, classification of race and ethnicity is necessary for demographic purposes. Hahn (7) has reviewed the classification of racial and ethnic groups in various federal health data bases. The current regulations for designating race and ethnicity were published in 1978 as Directive 15, "Race and Ethnic Standards for Federal Statistics and Administrative Reporting," by the Office of Management and Budget. The directive leaves race and ethnicity undefined while presenting rules for classifying persons into four racial groups (American Indian or Alaska Native, Asian or Pacific Islander, black, and white) and two ethnic categories (Hispanic origin and not of Hispanic origin). The distinction between race and ethnicity is poorly understood by the population; for example, a substantial proportion of Hispanics classify themselves as of "other race," rather than black or white.

Extensive research has been conducted on race and ethnicity and health and illness; see, for example, the reviews provided by Rothschild and Chapman 1981 (10), Harwood 1981 (9), Polednak 1989 (8), and Braithwaite and Taylor 1992 (11). Lacking a formal and standardized approach for assigning race and ethnicity, investigators have employed various approaches for classification such as self-identification, national origin, language use, family

characteristics and cultural practices, and biologic markers. Although misclassification is inherent in these research methods, strong effects of race and ethnicity on health, disease, and illness have been demonstrated.

The effects on health, disease, and illness of race and ethnicity may be mediated through a broad array of mechanisms that reflect genetic, environmental, social, and cultural influences. Although genetic factors are an appealing explanation for differences among groups of people, culture, degree of acculturation, and socioeconomic factors may be more significant determinants of health status. Simply attributing differences in health, disease, and illness to membership in particular groups may be inadequate for both research and clinical purposes.

In a discussion of the concepts of culture and acculturation, Helman (4) wrote "Culture is a set of guidelines, explicit and implicit, which individuals inherit as members of a particular society, and which tells them how to view the world, how to experience it emotionally, and how to behave in it in relation to other people, to supernatural forces or gods, and to the natural environment. It also provides them a way of transmitting these guidelines to the next generation by use of symbols, language, art and ritual." The influence of culture extends through all aspects of daily life, including beliefs and practices about health and illness. All societies include many different cultures, which may be defined operationally by historical, geographical, economic, social, and political elements (4).

Acculturation is one component of the multidimensional and bidirectional process of assimilation (12). In one conceptual model of assimilation, four subprocesses include changes at the psychologic (identification), cultural (acculturation), structural (integration), and biologic (amalgamation) levels. Acculturation is the process of incorporating some cultural attributes of a larger society. Indicators of acculturation might include birthplace, duration of residence in the United States for immigrants, patterns of language use, observance of traditional holidays, and other cultural practices.

Sociocultural factors and health and illness status of minority populations have been extensively examined and will not be specifically addressed in this review. The mechanisms by which sociocultural factors affect health are diverse and complex, extending from overt racism (13, 14) to far more subtle barriers as minority cultures interact with the majority culture. For readers seeking more information in this area, we suggest the following references for blacks (11, 15), Hispanics (16, 17), Asians-Pacific Islanders (18-21), and American Indians-Alaska Natives (22-24).

TABLE 2  
AGE-ADJUSTED MORTALITY RATES\* FOR SELECTED PULMONARY DISEASES  
BY SEX, RACE, AND HISPANIC ORIGIN, UNITED STATES, 1986-8†

|                                    | Pneumonia and Influenza |       | COPD |       | Lung Cancer |       |
|------------------------------------|-------------------------|-------|------|-------|-------------|-------|
|                                    | Men                     | Women | Men  | Women | Men         | Women |
| Non-Hispanic whites‡               | 36.7                    | 25.3  | 47.8 | 21.0  | 80.7        | 32.1  |
| Non-Hispanic blacks‡               | 46.6                    | 24.9  | 34.1 | 10.2  | 104.6       | 30.3  |
| Hispanics, total‡                  | 29.8                    | 20.4  | 20.0 | 8.8   | 36.3        | 11.9  |
| Mexican                            | 16.0                    | 13.8  | 12.0 | 6.4   | 20.1        | 7.1   |
| Puerto Rican                       | 49.0                    | 32.9  | 32.8 | 15.5  | 42.3        | 15.8  |
| Cuban                              | 19.5                    | 11.8  | 15.2 | 5.4   | 40.6        | 6.8   |
| American Indians/Alaska<br>Natives | 41.9                    | 25.0  | 29.7 | 11.1  | 44.1        | 20.9  |
| Asians/Pacific Islanders           | 24.9                    | 15.4  | 18.8 | 5.6   | 38.2        | 16.7  |

\* Per 100,000.

† From CDC (4).

‡ Based on data from 18 states and the District of Columbia.

TABLE 3  
NUMBER OF DEATHS AND AGE-ADJUSTED MORTALITY RATES FOR SELECTED RESPIRATORY CAUSES: AMERICAN INDIANS (AI) AND ALASKA NATIVES (AN) IN THE IHS SERVICE AREA (1984-1988) AND THE U.S. ALL RACES (1986)\*

| Cause of Death<br>(ICD-9-CM Code)                             | Number of Deaths |                   | Mortality Rate per<br>100,000 Population |                   |
|---|------------------|-------------------|--|-------------------|
|   | AI/AN            | U.S.<br>All Races | AI/AN                                    | U.S.<br>All Races |
| Tuberculosis, respiratory system<br>(010-012)                 | 76               | 1,407             | 1.7                                      | 0.4               |
| Tuberculosis, other (013-018)                                 | 34               | 375               | 0.8                                      | 0.1               |
| Coccidioidomycosis (114)                                      | 5                | 82                | 0.1                                      | 0.0†              |
| Histoplasmosis (115)  | ‡                | 82                | -  | 0.0               |
| Blastomycosis (116)   | 3                | 27                | 0.1                                      | 0.0               |
| Cryptococcoses (117.5)  | 6                | 527               | 0.1                                      | 0.2               |
| Sarcoidosis (135)   | 1                | 397               | 0.0                                      | 0.2               |
| Malignant neoplasm of bronchus<br>and lung (162.2-162.9)      | 856              | 125,387           | 21.5                                     | 37.5              |
| Pneumococcal pneumonia (481)                                  | 56               | 2,812             | 1.2                                      | 0.6               |
| Pneumonia, organism unspecified<br>(486)                      | 743              | 52,236            | 14.1                                     | 9.8               |
| Pneumonia, all types (480-486)                                | 984              | 67,974            | 19.1                                     | 13.2              |
| Emphysema (492)   | 66               | 14,471            | 1.5                                      | 3.7               |
| Asthma (493)  | 47               | 3,955             | 1.1                                      | 1.2               |
| Chronic airway obstruction, not<br>elsewhere classified (496) | 364              | 53,513            | 8.1                                      | 12.8              |
| Idiopathic fibrosing alveolitis<br>(516.3)                    | 6                | 151               | 0.2                                      | 0.0               |
| Respiratory distress syndrome (769)                           | 93               | 3,408             | 1.3                                      | 1.4               |

\* Source: Indian Health Service (6).

† 0.0 = rounds to zero.

‡ - = represents zero.

Socioeconomic characteristics, among the most powerful determinants of health and disease (25-28), differ markedly among the racial and ethnic groups of the United States (table 4) (29). Level of income and education may impact directly and indirectly on health status (30, 31). Income is a determinant of access to health care, and frequently of the quantity and quality of health care available. Persons with low income, regardless of race or ethnicity, are more likely to be uninsured (32), to encounter delays or be denied care (33), to rely on hospital clinics and emergency rooms for health services (34), and to receive substandard care (35). Level of education may influence health beliefs and behaviors, including behavior in seeking health-care and treatment choices and compliance. The usual socioeconomic indicators, education and personal or household income, serve only as surrogates for more complex correlates of individuals within populations. Separating the effects of being in a minority group from the effects of socioeconomic status has proved difficult for many health and disease outcomes.

The racial and ethnic profile of physicians in the United States does not reflect the distribution of minority populations, and the resulting sociocultural discrepancies between patients and physicians may adversely affect access to care and the doctor-patient relationship (36). Except for Asians/Pacific Islanders, who contributed 14.3% of medical students during the 1991-92 academic year, all other minority groups were under-represented in medical schools (37) compared with the general population (table 1). Blacks constituted 6.5% of medical students, Hispanics 5.5%, and American Indians/Alaska Natives 0.4%. A similar pattern was seen in graduate medical education for 1990 (38).

Minority groups may also experience patterns of environmental exposures affecting the lung that differ from those experienced by the white majority (39). These differences may reflect residence location, housing characteristics, occupation, and lifestyle. The term "environmental equity" is now used to refer to the distribution of environmental risks across population groups (40). Minority populations may sustain a higher burden of risk from the con-

TABLE 4  
SOCIOECONOMIC CHARACTERISTICS OF MAJOR RACIAL AND ETHNIC GROUPS OF THE UNITED STATES, 1990\*

| Socioeconomic Characteristics | White    | Black    | Hispanic | Asian-Pacific Islander | American Indian/ Alaska Native |
|-------------------------------|----------|----------|----------|------------------------|--------------------------------|
| Education                     |          |          |          |                        |                                |
| ≥ High school graduate, %     | 77.9     | 63.1     | 49.8     | 77.5                   | 65.5                           |
| ≥ 4 years of college, %       | 21.5     | 11.4     | 9.2      | 36.6                   | 9.3                            |
| Household income, 1989        |          |          |          |                        |                                |
| Median                        | \$31,435 | \$19,758 | \$24,156 | \$36,784               | \$20,025                       |
| Poverty, 1989                 |          |          |          |                        |                                |
| Below poverty level, %        | 9.8      | 29.5     | 25.3     | 14.1                   | 30.9                           |

\* Source: 1990 Census (2).

fluence of exposures associated with residence in more polluted environments, housing that increases the burden of inhaled pollutants, jobs associated with toxic exposures, and higher rates of active and passive smoking.

The acquired immunodeficiency syndrome (AIDS) directly impacts on the development of infectious and other lung diseases (41) and is an increasing problem among minorities of the United States. Among blacks and Hispanics the numbers of persons with AIDS increased by 10.2 and 11.5% from 1990 to 1991, and declined by 0.5% among whites (42). However, a detailed description of AIDS and pulmonary disease complications in minorities is beyond the scope of this review, and AIDS will be considered only in the context of specific infectious lung diseases.

This review systematically covers the major respiratory diseases of childhood and adulthood in the principal minority populations of the United States. For each minority group, we address socioeconomic characteristics, risk factors for respiratory diseases, morbidity and mortality from respiratory diseases, and the prediction of lung function for clinical purposes. The data presented in this review have been extracted from a number of sources, including peer-reviewed publications, Vital Statistics, and a variety of state and federal reports. The approaches of these sources to classification of minority group membership are not uniform, and they are known to have limitations (7, 43). Our review is also limited by incomplete coverage of the respiratory diseases of interest across the minority populations considered. We offer research recommendations to address these gaps. Nevertheless, the data are sufficient to justify drawing out clinical implications. The review concludes with some suggestions with regard to the care of minority patients with respiratory diseases.

Although we emphasize major respiratory diseases, race and ethnicity probably have clinical significance for many other respiratory conditions. For example, it is probable that the frequency of sleep disorders varies among the racial and ethnic groups of the United States. Because of the high prevalence of obesity in blacks (26) and in Hispanics (44, 45), these groups are likely to be disproportionately affected by the adverse consequences of obesity on the pulmonary system (46). The prevalence of having a body weight of 20% or more above desirable body weight is much greater in black women (36%) than in white women (21%) (25). Preliminary data from a 5-yr study on the familial aggregation of sleep apnea showed that 31% of blacks younger than 45 yr of age had sleep apnea compared with 10% of whites (47). Overall, the prevalence of overweight is higher in Hispanics than in non-Hispanics in the United States (44), but it varies among the Hispanic groups. In the Hispanic Health and Nutrition Examination Survey conducted from 1982 to 1984, 11% of Cuban American men, 10% of Mexican American men, and 8% of Puerto Rican men were severely overweight (body mass index [weight in kg, height in meters squared] was 31.1 kg/m<sup>2</sup> or greater for men and 32.2 kg/m<sup>2</sup> or greater for women). The corresponding figures for women were 8% of Cuban Americans, 16% of Mexican Americans, and 14% of Puerto Ricans.

Patterns of alcohol use, also associated with adverse pulmonary consequences (48), vary among racial and ethnic groups. Alcoholism is a problem among the white majority and the minority populations of the United States (25). In a population-based survey conducted from 1980 to 1981 (25), the lifetime prevalence of alcoholism among male subjects was 30% for Hispanics, 23% for non-Hispanic whites, and 24% for blacks. The corresponding prevalence estimates for alcoholism among female subjects were lower at 4% for Hispanics, 5% for non-Hispanic whites, and 5% for blacks. Alcoholism is a particular concern among American

Indian groups; it is now regarded as a virtual pandemic, regularly being mentioned as the most pressing health problem faced by American Indian people. During 1986 to 1988 the age-adjusted death rate for American Indians directly attributed to alcohol was 32.7 per 100,000 population compared with 6.0 per 100,000 for U.S. All Races (49). For American Indians 45 to 54 yr of age, it was 78.9 per 100,000 compared with 15.8 per 100,000 for U.S. All Races.

As with alcohol, use of illicit drugs has adverse consequences on the pulmonary system (50), and the occurrence of illicit drug use may vary among the various racial and ethnic groups of the United States (25). Data from the National Household Survey on Drug Use obtained in 1988 suggest slight variations in the use of different illicit drugs among non-Hispanic whites, Hispanics, and blacks (25). The prevalence of ever using cocaine (including crack) was 10.8% for non-Hispanic whites, 11.0% for Hispanics, and 9.3% for blacks. The prevalence of ever using heroin was slightly higher among blacks (2.3%) and Hispanics (1.1%) than among whites (0.8%).

Many diseases that primarily affect organs other than the lung may be associated with pulmonary complications (51, 52). For some of these diseases, the available evidence suggests variation by race and ethnicity. For example, limited data suggest that the occurrence of systemic lupus erythematosus (SLE) is higher among blacks than among whites. During the period 1956 to 1965, the annual incidence of SLE among female whites living in New York City was 2.5 per 100,000 compared with 7.9 per 100,000 among female blacks (53). Similar results were found in Baltimore from 1970 to 1972, where white women had a prevalence of 3.9 per 100,000 compared with 10.5 per 100,000 among blacks (54). Blacks may also be at increased risk for scleroderma (55) and its associated pulmonary complications.

Variation that is not readily attributed to patterns of specific risk factors has also been observed in the occurrence of other diseases. For the period 1962 to 1984, mortality from thromboembolism in the United States was greater among nonwhites than among whites, and the differential in mortality was as high as 50% among persons 50 to 64 yr of age (56).

## BLACKS

The ancestors of most black Americans were brought to the United States from the west coast of Africa. More than 4 million of these people entered the country between 1619 and 1860 (57). In the early 17th century, they came as indentured servants, like many European immigrants, who were freed after seven years of service (58). However, as the need for free labor grew during the middle to late seventeenth century, the status of the African people changed from servant to slave, and they were denied all human rights. Although the Civil War ended slavery, the blacks were still denied civil rights, and they were compelled to live segregated and poverty-stricken lives. During and after the two world wars, many blacks moved to urban areas of the northern United States, but little changed in their living conditions (59). More recently, blacks have immigrated to the United States from Haiti and the Caribbean islands, introducing sociocultural and disease patterns that differ from those of blacks residing in the United States.

Because of the common African heritage of most blacks in the United States, the term African-American is often applied to them. In this review we use the term "black" for consistency with demographic standards and terminology in the medical literature. Blacks presently constitute approximately 12% of the U.S. population, making them the largest racial minority in the country (table 1).

Although blacks reside in all regions of the United States, their geographic concentration varies widely, with 53.0% living in the south, 20.1% in the north central areas, 18.3% in the northeast, and 8.6% in the west (25). In these regions, greater than 50% of blacks live in central cities (26). As a group, blacks are younger than whites. In 1990, the median age of blacks was 28.1 yr compared with 34.4 yr for the U.S. white population. Only 8.4% of blacks were 65 yr of age or older, compared with 13.9% of whites (60).

#### Socioeconomic Characteristics

Most indicators of the socioeconomic status of groups are lower in black than in white or other minority groups (table 4). In 1990, only Hispanics had a lower proportion of high school graduates than blacks, 49.8% and 63.1%, respectively, and the proportion of blacks with at least a college education was 47% lower than among whites. Income level is lowest among blacks, with approximately 30% of the black population living below the poverty level.

Black men constitute a high proportion of the unskilled and the blue collar work forces. As a group, black workers are excluded from certain more desirable industries (61–63). This exclusion, coupled with a low entry level in the work force, results in their greater likelihood of assignment to the "dirty" jobs, involving exposures to dusts and fumes that are risk factors for increased respiratory symptoms and greater rates of lung function decline (64, 65) and for lung cancer (66, 67).

#### Smoking

Patterns of cigarette smoking by blacks have been well documented in national surveys. Data from National Health Interview Surveys (NHIS), covering 1965 through 1990, have consistently shown a higher overall prevalence of smoking among blacks than

among whites, but in recent years the gap has narrowed (figure 1) (68, 69). Smoking patterns and choices of cigarettes differ between blacks and whites. Among males, a higher proportion of blacks smoke than whites (1990 prevalence = 32.6% versus 27.9%); but among females, fewer blacks smoked than whites (1990 prevalence = 21.2% versus 23.5%). However, studies validating self-reports of smoking with carboxyhemoglobin levels suggest that available prevalence estimates may underestimate the true prevalence of smoking, particularly among males and nonwhites (70).

Black and white smokers also differ in the numbers and the types of cigarettes smoked (68, 71). Blacks smoke fewer cigarettes per day on average than do whites. In the 1985 NHIS, 63.5% of black smokers and 34.9% of white smokers consumed less than a pack a day (68). The cigarettes smoked by blacks are more often high-tar and mentholated brands (68). In a 1986 nationwide survey of tobacco use in the United States, 78.0% of blacks and 55.9% of whites reported smoking cigarettes with high tar yields (> 15 mg/cigarette). The contrast between blacks and whites was even greater for smoking menthol cigarettes: 75.5% and 23.1%, respectively. Although blacks smoke fewer cigarettes per day than do whites, their exposure to tobacco combustion products may not be lessened; it has been hypothesized that menthol cigarettes provide a sensation of cooling that may promote deep inhalation and thereby greater deposition of tobacco smoke (68). Black-white differences in nicotine metabolism have also been hypothesized. Wagenknecht (72) found median cotinine levels 30% higher in blacks than in whites in 1,545 current smokers 18 through 30 yr of age who participated in the Coronary Artery Risk Development in (Young) Adults Study (CARDIA). The difference persisted in a multiple regression analysis that controlled for number of cigarettes, nicotine content of cigarettes smoked, frequency of inhalation, and other factors. Slower nicotine metabolism is another possible explanation for the lower numbers of cigarettes smoked by blacks.

Novotny (73) examined socioeconomic and demographic factors that may contribute to the black-white differences in smoking pattern. Using data from 21,593 participants in the 1985 National Health Interview Survey, socioeconomic factors, educational level, marital status, and race were assessed in multivariate statistical models as predictors of ever smoking, quitting smoking, and being a heavy smoker. In these models, black race was not associated with ever smoking (odds ratio [OR] = 1.0, 95% confidence interval [CI] = 0.9–1.1), but it did predict not being a heavy smoker (OR = 0.3, 95% CI = 0.2–0.3) and being less likely to quit smoking (OR = 0.7, 95% CI = 0.6–0.9).

The balance between initiation of smoking and smoking cessation in a population determines the overall prevalence of current smokers. Blacks have tended to start smoking somewhat later in adolescence and young adulthood than do whites (68, 74, 75). In a nationwide survey conducted between 1985 and 1989 of 73,527 high school seniors 17 to 18 yr of age, Bachman and coworkers (75) found that the self-reported prevalence of smoking cigarettes in the previous 30 days was lower among male blacks (15.6%) than among male whites (29.8%) and lower among female blacks (13.3%) than among female whites (34.0%). Recent data have shown differing patterns of decline on initiation rates in a comparison of black and white youths. Using NHISs from 1974 through 1985, Fiore and coworkers (76) examined self-reports of current smoking among respondents 20 to 24 yr of age as an index of smoking initiation. During this period, overall smoking initiation declined 1.02% per year among blacks and 0.35% per year among whites. Rapid decline in smoking initiation among male blacks,

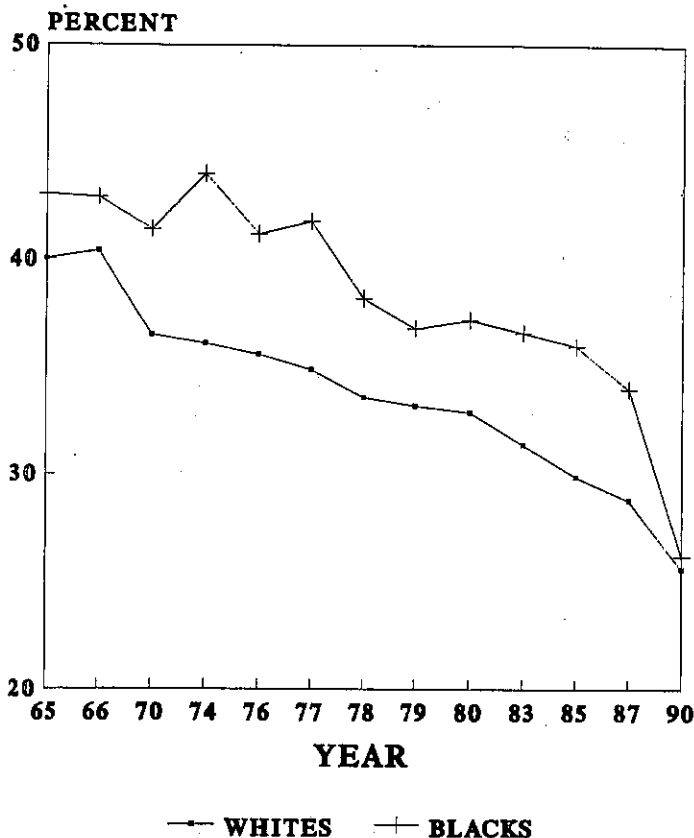


Figure 1. Prevalence of smoking in the United States among whites and blacks 20 years of age and older, 1965–1990 (68, 69).

2.24% per year, accounted for most of the black-white difference as initiation by female blacks declined by only 0.10% per year. Lower smoking initiation among blacks compared with whites was confirmed by Escobedo and coworkers (74) in a separate sample of 17,887 young adult participants 18 to 35 yr of age in the 1987 NHIS and the Hispanic Health and Nutrition Examination Survey (HHANES) conducted from 1982 through 1984. In these nationwide samples, smoking initiation, defined as the percentage of persons who started to smoke among persons who never smoked, was 40% among blacks and 47% among whites.

Patterns of smoking cessation for blacks and whites also differ in these nationwide surveys (77). Fiore and coworkers (76) calculated the quit ratios of the proportion of ever smokers who have become former smokers in NHISs from 1974 through 1985. The quit ratio was 1.04% per year among male blacks; the corresponding annual changes were 0.67, 0.46, and 0.95% for male whites, female blacks, and female whites, respectively.

To characterize the different patterns of smoking and smoking cessation in blacks and whites, Royce and coworkers (78) surveyed 11,968 participants in the Community Intervention Trial for Smoking Cessation (COMMIT) on their motivation to stop smoking and degree of nicotine dependence. Overall, blacks were 1.2 times (95% CI = 1.14–1.27) more likely to make a serious quit attempt than whites. However, based on responses about the timing of their first cigarette upon awakening, blacks were more likely to smoke within 10 min of awakening even among light/moderate smokers (OR = 1.6, 95% CI = 1.39–1.76), suggesting a higher degree of nicotine dependence.

#### Respiratory Diseases of Childhood

**Hyaline membrane disease.** Little information is available on the occurrence of respiratory distress syndrome of the newborn and hyaline membrane disease among the nation's racial and ethnic groups. In selected case-series, the incidence of respiratory distress syndrome among black compared with among white newborns has been either the same (79) or lower (80, 81). However, the prognosis for black babies with hyaline membrane disease is worse than for white babies, and on the basis of nationwide mortality statistics, black babies have twice the mortality of white babies (82).

**Sudden infant death syndrome (SIDS).** SIDS is the leading cause of infant death beyond the neonatal period. Using data for the National Center for Health Statistics for 1987, Wegman (82) found that black babies were nearly twice as likely to die from SIDS as white babies. Similar data have been reported from California (83) and Cook County, Illinois (84). Nationwide for the period 1980 to 1988, the black-to-white ratio for SIDS declined from 2.2 to 1.8 (85).

**Cystic fibrosis.** Cystic fibrosis is rare in black children, who have an incidence of only 1 per 17,000 (86) compared with 1 per 2,000 in white children (87). The rarity of cystic fibrosis among black children is reflected in the low proportion of blacks followed at Cystic Fibrosis Centers in the United States. Nationally, 3.1% of patients with cystic fibrosis registered at these centers are black (88), although centers located in urban areas report that black patients constitute from 8% (89) to 14.5% of the total (90). Blacks have been reported to present two to three times more frequently than whites with pulmonary symptoms exclusively (89, 91). In a patient series from Johns Hopkins Hospital from 1971 through 1986 (89), blacks had fewer hospitalizations for pulmonary exacerbations, a higher mean FVC, and higher chest roentgenogram scores than did whites. Additionally, in that series blacks had a higher frequency of hyponatremic dehydration and peptic ulcer disease

(89). The different clinical presentations of cystic fibrosis between blacks and whites may be partially due to different patterns of genetic mutations (92).

#### Respiratory Diseases of Childhood and Adulthood

**Tuberculosis.** The occurrence of tuberculosis decreased in the early 1980s, but this infection is now, unfortunately, resurgent (93–95). This disease has occurred with greater frequency among all minorities (94, 96, 97); blacks and Hispanics have had the highest recent rates of increase. Furthermore, young blacks and Hispanics between 25 and 40 years of age have been disproportionately affected (93).

Statistics for 1990 show that 70% of the 25,701 cases in the United States were in minorities; of these, 54.1% were in non-Hispanic blacks, 26.8% in Hispanics, 17.0% in Asians/Pacific Islanders, and 2.1% in American Indians/Alaska Natives (94). In the age range of zero to 15 yr, 86% of cases were in minority children. Tuberculosis rates for 1985 to 1990 were highest in Miami, Atlanta, San Francisco, Newark, Tampa, and New York City (94). By county, cases in minorities were primarily localized to counties in the southeastern states, along the coasts, and in Texas.

Factors that probably contribute to the reemergence of tuberculosis in the United States include HIV infection, homelessness and poverty, substance abuse, and collapse of the public health system (94, 95, 98). These factors are strong determinants of the risk for tuberculosis. In addition, increasing evidence suggests that blacks are more susceptible than whites to the development of infection by *Mycobacterium tuberculosis* (99–101). McAdam (99) examined the risk of tuberculous infection among 1,343 homeless men in New York City from 1982 through 1988. In a multivariate statistical analysis that controlled for age, intravenous drug use, and length of residence in the shelter system, black race compared with white race had an independent positive effect on the risk for tuberculous infection (OR = 2.6, 95% CI = 1.7–4.0). Stead and colleagues (100) found similar results among black residents of nursing homes in Arkansas and among two prison populations. Possible explanations for the heightened susceptibility to tuberculous infection were recently reviewed by Stead (101), who emphasized the possibility of genetically determined resistance to tuberculosis through nonimmune mechanisms (102, 103).

Blacks also have apparently heightened susceptibility to the development of coccidioidomycosis, another disease for which cell-mediated immunity is a principal host defense. For example, fungal dissemination occurred in 18.7% of 230 ethnically identified, acutely infected, patients (104) from a severe 1977 dust storm in Central California (105). Exposure to *Coccidioides immitis* during the dust storm was considered widespread and "uniform" (104). Among 26 blacks, 53.8% developed disseminated coccidioidomycosis; the proportion among whites was 11.2%. However, the role of a racial predisposition has been challenged because of differences in nonracial factors and in the uniformity of exposures to *Coccidioides immitis*; that is, the nature of racial predisposition can only be determined as a function of the number of persons of each race who are infected (106).

**Pneumonia and influenza.** Pneumonia and influenza are a major cause of morbidity and the sixth leading cause of death in the United States. More than 3 million cases of pneumonia in children and adults occur annually in the United States, with more than 530,000 hospital admissions among persons 15 yr of age and older (107). Furthermore, mortality from pneumonia and influenza has increased by 26.8% from 1979 to 1988 (108). During the period from 1977 to 1988, 10,000 to 40,000 excess deaths were attributed to influenza (109).

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view Surveys suggest that childhood asthma became less severe during this time period for blacks and whites (118). In the 1981 survey 17.4% of black children were reportedly hospitalized in the previous year compared with 10.2% in the 1988 survey. The corresponding figures for whites were 12.8 and 9.7%. Other indicators of asthma morbidity were consistent.

As with hospitalization rates, mortality rates for asthma have increased in blacks and whites of all age groups since the late 1970s (114, 115, 125), with rates consistently higher in blacks than in whites. From 1980 to 1987, asthma mortality among blacks increased from 2.5 to 3.6 per 100,000; the corresponding increase for whites was from 1.1 to 1.5 per 100,000.

Many investigations have been conducted on risk factors for death from asthma (119, 120). The evidence from these studies indicates that mortality from asthma reflects the interplay of the severity of the underlying disease with personal characteristics, environmental factors, and characteristics of the available health care (126). Few studies have addressed black-white differences for this array of factors. Marder and coworkers (127) analyzed data from 205 patients 5 to 34 yr of age who died from asthma in Chicago from 1980 to 1988. For both blacks and whites, they found an inverse relationship between median family income in the census tract of residence at death and the mortality rate from asthma.

**Chronic obstructive pulmonary disease (COPD).** Compared with asthma, limited data are available on morbidity and mortality from COPD among blacks. McWhorter (117) used data from NHANES I, which was conducted from 1971 to 1975, and the NHANES I Epidemiologic Follow-up Survey, conducted from 1982 to 1984, to determine the prevalence and incidence of COPD among 14,404 adults 25 to 74 yr of age. In this sample, the prevalence of COPD was 6.2% among whites and 3.2% among blacks. Factors associated with an increased risk for the development of COPD during the follow-up period were increasing age (OR = 3.7, 95% CI = 3.0–4.6 for ages 65 to 74 yr compared with ages 25 to 34 yr), ever smoking (OR = 2.6, 95% CI = 2.2–3.0), and low income (OR = 1.3, 95% CI = 1.2–1.5). Black race was associated with a lower risk for COPD than was white race (OR = 0.6, 95% CI = 0.5–0.7).

The prevalence of COPD among blacks and whites in the United States was estimated in the 1990 NHIS using respondent reports on the presence of chronic bronchitis and of emphysema (128). Among blacks 45 to 64 yr of age and 65 yr of age and older, the prevalence of chronic bronchitis was 55.2 per 1,000 and 42.7 per 1,000, respectively. The corresponding figures for emphysema were 3.6 per 1,000, and 41.5 per 1,000. Compared with blacks, whites in both age groups reported higher frequencies of chronic bronchitis (45 to 64 years, 59.7 per 1,000, and 65 yr and older, 73.8 per 1,000) and emphysema (45 to 64 yr, 13.8 per 1,000, and 65 yr and older, 46.1 per 1,000).

Mortality statistics provide additional evidence of a higher frequency of COPD among whites than among blacks (115, 129, 130). Evans and Mullaly (115) summarized mortality statistics for COPD in the United States; in 1982 the age-adjusted mortality for whites was 16.6 per 100,000, and for blacks it was 12.8 per 100,000. A similar pattern of mortality was found in 18 states and the District of Columbia (table 2).

**Sarcoidosis.** Sarcoidosis occurs in all races and cultures. However, the incidence and prevalence of the disease varies widely from country to country and among population groups within countries (131). Morbidity and mortality from sarcoidosis are much greater for blacks than for whites in the United States (131, 132). Available data on occurrence have been obtained primarily from chest radiographic surveys of the general population for tubercu-

losis or from chest radiographs taken in military populations. Little recent information from these sources is available. This older information suggests an incidence of approximately 1 to 11 per 100,000 and a prevalence from 5 to 50 per 100,000 population (131, 133). The disease has been estimated to be 10 to 17 times more prevalent in blacks than in whites (134–136). The incidence is highest for young adults 25 to 40 yr of age, and higher in females than in males (132, 137).

Hospitalization data provide an index of morbidity associated with sarcoidosis. In 1981, there were more than 10,000 hospital discharges with a diagnosis of sarcoidosis in the United States among both blacks and whites. The black-to-white ratio of the age-adjusted discharge rates was 8.2 (132). However, because this disease is usually evaluated and managed in the outpatient setting, hospitalization data undoubtedly underestimate the morbidity of sarcoidosis (131).

In the United States, sarcoidosis is an infrequent cause of death. In 1982, only 339 deaths had sarcoidosis listed as the underlying cause and an additional 266 as a contributing cause (132). Mortality rates of all age groups other than 15 to 44 yr are approximately three times greater for blacks than for whites, but between the ages of 15 to 44 yr the rates are approximately 20 times greater. For 1979 to 1984, age-adjusted mortality rates per 100,000 population for sarcoidosis were 0.85 and 1.22 for black men and women, respectively, compared with 0.07 and 0.10 for white men and women (132). The major causes of death are irreversible fibrosis leading to cor pulmonale, pulmonary hemorrhage caused by aspergilloma, and sudden death from myocardial disease (138).

Although the well-documented geographic and racial variation in the occurrence of sarcoidosis has led to many hypotheses concerning genetic and environmental risk factors (131), little is known about the etiology of the disease and few epidemiologic studies have directly tested the various hypotheses (139). Some data suggest that cigarette smokers may have reduced risk of sarcoidosis (140, 141), with one study noting that nonsmokers were four times more likely than smokers to have sarcoidosis (140). Genetic susceptibility to sarcoidosis has been suggested by studies in families and in twin pairs (131, 142, 143). Reports from the United States and Europe have shown an increased frequency of A1, B8, and DR3 haplotypes in white patients (139, 144), and haplotype B8 has been associated with acute-onset disease and a good prognosis (145). Information on the influence of haplotype in black patients with sarcoidosis is conflicting. Some evidence suggests that the disease is associated with HLA-AW30 (146) and HLA-BW15, with sarcoidosis 5.5 times more frequent in persons with these antigens than in persons lacking them (147); however, other researchers have found no increase in the frequency of HLA in the A and B loci in blacks (148). It appears that HLA haplotypes affect the clinical expression of sarcoidosis, but to date there is inconsistent information on the effect of HLA haplotypes on disease susceptibility (144, 146, 149, 150).

Clinical patterns of sarcoidosis differ in whites and blacks. Blacks are more likely than whites to have multisystem disease, and they have a worse prognosis than do whites who have a similar presentation. In patients presenting with Stage I disease (bilateral hilar adenopathy with clear lung parenchyma), 50% of whites but only 12% of blacks have rapid recovery from the disease (138). On presentation, white patients are more likely to have asymptomatic pulmonary involvement or erythema nodosum, whereas blacks are more likely to have ocular symptoms or skin involvement other than erythema nodosum (138). In late-stage irreversible pulmonary fibrosis, the chest radiographic findings of small- and moderate-sized cavities are more frequent in black

For blacks, there is little specific information on morbidity from pneumonia or influenza. The annual incidence of pneumonia among children, estimated from the 1988 National Health Interview Survey, was 2.0 per 100 white children and 1.2 per 100 black children. A similar pattern for the incidence of influenza was found among adults participating in the same survey, 45.5 per 100 persons per year for whites and 26.8 per 100 persons per year for blacks (25). However, these statistics, based on subject reports of restricted activity or physician contact, may underestimate incidence, particularly for blacks.

Two recent investigations suggest that black race is associated with an increased occurrence of lower respiratory tract infections among children. To determine risk factors for acute lower respiratory tract infections and chronic respiratory symptoms, Margolis and coworkers (110) studied 396 infants from North Carolina in 1986 through 1988. Socioeconomic status was a strong predictor for acute lower respiratory tract infections, with an annual incidence of 1.41 episodes per child among infants whose head-of-household did not graduate from high school, 1.26 among infants whose head-of-household graduated from high school, and 0.67 among infants whose head-of-household had more than a high school education. Many factors were associated with persistent respiratory symptoms, including black race (RR = 1.75, 95% CI = 1.21–2.56), exposure to environmental tobacco smoke (RR = 1.85, 95% CI = 1.23–2.77), household crowding (RR = 2.53, 95% CI = 1.39–3.21), and bottle feeding (RR = 2.07, 95% CI = 1.32–3.24). Vadheim and coworkers (111) conducted a case-control study of invasive *Hemophilus influenzae* type b among 79 children aged 18 to 60 months who were identified in Los Angeles County from 1988 through 1989. Black maternal race was associated with an odds ratio of 3.47 (95% CI = 1.41–8.53) compared with that in Hispanics.

In comparison with whites, mortality from pneumonia and influenza is higher among black children and adults (table 2) (4). In 1987, mortality from pneumonia and influenza in black infants was 2.6 times that of white infants (82). For all age groups, overall mortality ratios for pneumonia in 1987 comparing blacks with whites were higher in male and female blacks, 1.58 and 1.26, respectively (25). The excess extends throughout the age spectrum: in 1988, black adults 45 to 64 yr of age had a mortality ratio of 2.7 for pneumonia compared with whites of the same age (112).

These differences in morbidity and mortality among blacks and whites may be partially explained by poverty and access to health care. Sick cell disease, discussed in greater detail in a subsequent section, may also contribute to morbidity and mortality from bacterial infections with polysaccharide-encapsulated organisms (113).

**Asthma.** In the United States, data from nationwide samples and survey populations indicate that asthma is a common disease among children and adults (114). Estimates of prevalence have varied widely, from 10 to 20 million nationwide, depending on the criteria for defining asthma: active asthma (point prevalence) or ever having asthma (cumulative prevalence) (115). Evans and Mullaly (115) estimated the cumulative prevalence of asthma from the Second National Health and Nutrition Examination Survey (NHANES II), 1976 to 1980, using reports of ever being told by a physician that the index subject had asthma and/or frequent problems with wheezing in the past 12 months as the definition. On the basis of these criteria, Evans and Mullaly estimated that approximately 21 million people 3 to 74 yr of age were previously or currently affected by asthma. This estimate probably overestimates the frequency of asthma because it is a cumulative prevalence and because the inclusion of persons with frequent wheezing is not specific for asthma.

Increasingly extensive data have accumulated during the past 10 years on the frequency of this common disease among the racial and ethnic groups of the United States (114). In all age groups the prevalence of asthma is higher in blacks than in whites (115–118). Gergen and coworkers (116) determined the cumulative prevalence of asthma among children from the NHANES II and found that 13.4% of black children and 9.7% of white children have ever had physician-diagnosed asthma or wheezing. McWhorter (117) used the more-restricted criterion of an affirmative response to the questions: "Has a doctor ever told you that you had asthma?" and "Do you still have it?" in analyzing data from adults 25 to 74 yr of age in the First National Health and Nutrition Examination Survey (NHANES I), 1971 to 1975. The prevalence of active asthma among blacks was 3.7%, and among whites it was 2.7%.

Using data from NHISs conducted in 1981 and 1988, Weitzman and coworkers (118) examined changes in the prevalence of asthma reported to the present in the previous 12 months. During the 7 yr of the surveys, prevalence increased among white children (2.7 to 4.1%), but it did not change among black children (5.3 to 5.1%).

Both genetic and environmental factors are considered to contribute to the development of asthma (119, 120). Several recent reports have described multivariate analyses that provide insight into the effect of black race on the prevalence of asthma in children (121, 122) and in adults (117). Schwartz and coworkers (122) analyzed data collected from 1971 through 1975 in the NHANES II from 5,672 children 6 months to 11 yr of age. In this sample, the overall prevalence of asthma was 7.2% among black children and 3.0% among white children. In a multivariate analysis, controlling for race, age, and sex, the odds ratio for asthma, comparing black with white children, was 2.5 (95% CI = 1.9–3.4). Other significant predictors of asthma, after controlling for race, age, and sex, were low birthweight (OR = 1.31, 95% CI = 1.00–1.72), young maternal age (OR = 1.43, 95% CI = 1.06–1.91), low family income (OR = 1.98, 95% CI = 1.27–3.05), and living in the central city (OR = 1.55, 95% CI = 1.13–2.13). This investigation lacked information on parental smoking. In a more recent national sample of 2- to 5-yr-olds, black race was associated with asthma occurrence (OR = 1.7, 95% CI = 1.1–2.8) after controlling for socioeconomic variables and maternal smoking (121).

Less information is available on risk factors for the development of asthma among adults (117). McWhorter (117) used data from the NHANES I, which was conducted from 1971 to 1975, and the NHANES I Epidemiologic Follow-up Survey, conducted from 1982 to 1984, to identify predictors of the onset of asthma among 14,404 adults, 25 to 74 yr of age. The unadjusted average annual incidence of asthma was 2.5 per 1,000 per year. Only female sex was a significant predictor of asthma (OR = 1.4, 95% CI = 1.1–1.9). Low income and black race were of borderline significance (OR = 1.3, 95% CI = 1.0–1.8 for both factors).

Hospitalization rates reflect disease prevalence, disease severity, and patterns of health care access and medical practice. Nationwide data for both blacks and whites from the National Hospital Discharge Surveys, 1980 to 1987, have shown temporally increasing discharge rates for asthma, particularly among those 19 yr of age and younger and 65 yr of age and older (114). Black children had a greater increase in hospitalization than did white children (116). Investigations of hospitalizations for asthma among children and young adults in Maryland (123) and in New York City (124) have found that hospitalization rates are positively associated with black race and inversely associated with income. However, data on parental reports of hospitalizations and other indicators of asthma severity in the 1981 and 1988 National Health Inter-



TABLE 5  
INCIDENCE, MORTALITY, AND SURVIVAL FOR CANCER OF THE LUNG  
AND BRONCHUS BY RACE AND SEX\*

|                | Incidence* |         |                          | Mortality* |         |                          | Five-Year Survival |
|----------------|------------|---------|--------------------------|------------|---------|--------------------------|--------------------|
|                | 1973-74    | 1987-88 | Change<br>1973-88<br>(%) | 1973-74    | 1987-88 | Change<br>1973-88<br>(%) | 1981-87<br>(%)     |
| <b>Males</b>   |            |         |                          |            |         |                          |                    |
| White          | 73.2       | 82.2    | 12.2                     | 62.4       | 73.1    | 17.2                     | 11.8               |
| Black          | 103.0      | 120.0   | 16.5                     | 76.3       | 98.2    | 28.6                     | 9.9                |
| <b>Females</b> |            |         |                          |            |         |                          |                    |
| White          | 18.9       | 40.2    | 113.2                    | 13.8       | 29.2    | 111.2                    | 16.3               |
| Black          | 21.0       | 39.8    | 89.6                     | 13.8       | 27.7    | 100.7                    | 13.0               |

\* From: SEER 1991 (174).

† Rates are per 100,000 and are age-adjusted to the 1970 standard population.

smoking, occupational exposures, and other environmental factors are suspect (175-177). However, recent investigations of chromosomal patterns in patients with lung cancer suggest that rare alleles in blacks may represent a risk factor for lung cancer (178, 179).

#### Prediction of Lung Function

Anthropometric differences in thoracic height between U. S. blacks and whites responsible for differences in pulmonary function have been noted for more than a century. Black Civil War soldiers were found to have a vital capacity 11% less than that of white soldiers (180). An early study of lung function found vital capacity to be lower in black than in white men (181). Regression equations derived from white populations, using standing height as the measure of size, usually overpredict values measured in black subjects by approximately 12% for TLC, FEV<sub>1</sub>, and FVC, and by about 7% for FRC and residual volume (RV) (182). These differences persist after adjustments for age, stature, smoking, and other factors (183). Differing anthropometric characteristics of blacks and whites explain at least some of the differences in lung function. Anthropometric measurements show that blacks have smaller ratios of trunk size to height or leg length than do whites (184-186). Use of sitting height as an index of body size reduces but does not eliminate the racial differences in pulmonary function (184, 186-189).

Race-specific prediction equations should be used for blacks (190, 191). Although some researchers have argued that a "scaling" correction factor based on prediction equations for whites can be used to predict lung function in blacks (190), the availability of race-specific prediction equations makes such extrapolation unnecessary (192-194). A number of prediction equations are now available for black men and women, including predicted values for FEV<sub>1</sub> and FVC and for FEV<sub>1</sub>/FVC% (183). Such prediction equations should be used whenever possible; however, for a laboratory that only occasionally serves black subjects and has not assessed the adequacy of external equations for the local black population, a "scaling" correction factor may be applied to prediction equations for caucasians (190).

#### Summary

The patterns of risk factors and respiratory diseases in blacks differ markedly from those in whites. Cigarette smoking is more common among blacks, yet on average they smoke fewer cigarettes. The high prevalence of smoking is associated with a higher incidence of and mortality from lung cancer among blacks than among

whites. However, the frequency of COPD is unexpectedly low in blacks, given their prevalence of smoking. The discrepancy between the prevalence of smoking and the occurrence of smoking-related diseases remains unexplained.

For tuberculosis, pneumonia and influenza, asthma, and sarcoidosis, blacks have greater morbidity and mortality than do whites. Socioeconomic factors, for example poverty, access to health care, and environmental exposures, are certain to contribute to differences between blacks and whites in the occurrence of these diseases.

Little information is available on the role of genetic characteristics as a determinant of black-white differences in patterns of malignant and nonmalignant respiratory diseases.

#### HISPANICS

The designation Hispanic indicates a common heritage of language and culture that can ultimately be traced to the Spanish empire of centuries ago. Peoples with ancestral roots in areas previously under Spanish rule, including Mexico, Central America, South America, and some Caribbean islands, are considered Hispanic. The term Hispanic is used to refer to ethnicity with the commonality of language and cultural origins and not to race; persons of Hispanic origin can be of any race (195). In the 1980 Census, 55.6% of Hispanics (persons who responded "Yes" to the Hispanic origin question) characterized their race as white, 40% as "other," 2.7% as black, 1.1% as Asian/Pacific Islander, and 0.6% as American Indian (196). The majority of persons selecting "other" race wrote in designations such as Mexican, Puerto Rican, or other Hispanic-origin terms, reflecting the widespread confusion about the distinction between racial and ethnic-origin indicators. The terms Latino and Chicano have also been used in reference to persons of Latin American heritage (197); these terms have not been universally adopted, however (196). Although Spanish dominion left a common legacy of language and culture, present-day use of the term Hispanic hides the marked heterogeneity of Hispanics with regard to racial and cultural backgrounds. Demographically, Hispanics in the United States are generally grouped into five major populations: Mexican-Americans (62%; concentrated in Arizona, California, Colorado, New Mexico, and Texas), Puerto Rican-Americans (13%; concentrated in the New York City metropolitan area), Cuban-Americans (5%; concentrated in Florida), Central/South Americans (12%), and "other" Hispanics (8%) (198, 199). The group of other Hispanics includes the Hispanic populations of New Mexico and Colorado who combine Spanish,

(30%) than in white (13%) patients (151). Blacks with sarcoidosis have more frequent evidence of airways obstruction (152). Skin involvement other than erythema nodosum is seen in approximately 20% of black patients and 5% of whites (138), and skin plaques and nodules are more frequent and more persistent in black patients (153). The skin changes may be seriously disfiguring and are often associated with mucosal changes resulting in nasal obstruction, chronic sinusitis, and laryngeal and endobronchial disease causing airway obstruction (153). Ocular sarcoidosis is seen in approximately 15% of black patients and 5% of whites, with acute or chronic granulomatous uveitis being most common (138). Cystic bone changes, seen in approximately 14% of patients with sarcoidosis, are more common in blacks. Overall, chronic arthropathy is rare in sarcoidosis, but it is most often reported in black patients (138, 153). As much as 25% of black patients with sarcoidosis may have increased serum immunoglobins (138).

**Sickle cell disease.** Centuries prior to Herrick's description in *The Archives of Internal Medicine* in 1910, West Africans were conversant with the clinical manifestations of sickle cell disease, to which they gave specific tribal vernacular names (154, 155). Hemoglobin-S, the most common abnormal hemoglobin, has the highest incidence among black Africans and descendants of blacks who emigrated from Equatorial Africa. The prevalence of sickle cell disease in Africa varies from 40% in some mid-African tribes to near zero in regions remote from the equator. A prevalence as great as 25% has been reported in southern Turkey, Saudi Arabia, and southern India. In the United States, the prevalence of the sickle cell trait among African-Americans is 8 to 10%, and the number of those who are homozygous is greater than 60,000. Hemoglobin-AC occurs in approximately 3% of African-Americans.

The lung is a major target organ in sickle cell disease. Acute pulmonary disease such as bone marrow emboli, fat emboli, pulmonary emboli secondary to deep venous thrombosis, pneumonia, and vaso-occlusive episodes may occur. The frequency of bone marrow and fat emboli is unknown. However, in 1982 Haupt and coworkers (156) found bone marrow emboli at autopsy in 13% of patients with sickle cell disease. Histologic evidence of bone marrow infarction was always associated with the bone marrow emboli (156, 157). Pulmonary emboli secondary to deep venous thrombosis do occur, but probably no more frequently than in patients without sickle cell disease.

Infections are a frequent cause of morbidity and mortality in sickle cell disease, accounting for 60.7% of deaths in patients younger than 3 yr of age. Patients have compromised immune function as a result of functional asplenia and a poorly defined abnormality of their alternative complement pathway. Loss of splenic function occurs in nearly 85% of children with the disease by the age of 6 yr (158-160). Persons with sickle cell disease are particularly susceptible to polysaccharide-encapsulated bacteria (*Streptococcus pneumoniae* and *Hemophilus influenzae*) (158, 161). In general, infections are probably more common in childhood; however, the use of prophylactic penicillin has resulted in a significant reduction in pneumococcal infections (162).

Acute chest syndrome is a term used to describe a clinical picture of fever, cough, chest pain, and pulmonary opacities in a patient with sickle cell disease (163). The syndrome occurs in 15 to 35% of hospitalized patients and is a significant cause of morbidity and mortality. The etiology of acute chest syndrome is rarely identified clinically, but pneumonia and/or vaso-occlusive episodes have been implicated, with early studies suggesting that pneumonia was more common (164, 165). However, more recently, Charache and coworkers (166), Poncz and colleagues (167), and Kirkpatrick and coworkers (168) found pneumonia to be the etiol-

ogy in less than 45, 12, and 21%, respectively, suggesting that vaso-occlusive episodes are the more common cause of the acute chest syndrome. On the basis of review of smoking habits of 69 patients with sickle cell anemia, Young and coworkers (169) found that cigarette smoking was a significant risk factor for the occurrence of the acute chest syndrome.

As the life expectancy of patients with sickle cell disease increases, chronic organ damage, particularly chronic lung disease, may become an increasing cause of morbidity and mortality. Chronic lung disease is defined as sickle cell disease with chronic hypoxemia, pulmonary fibrosis, abnormal pulmonary function tests, and pulmonary hypertension with cor pulmonale. In 1988, Powars and coworkers (170) reported on 28 children and adults with chronic lung disease attributed to sickle cell. They proposed a staging system based on clinical, physiologic, and radiographic criteria. Patients were believed to progress from Stage I (mild disease) to Stage IV (pulmonary hypertension) in a fairly predictable fashion. Abnormal pulmonary function tests were the best marker of early disease. In addition, recurrent episodes of acute chest syndrome were a major risk factor for progression to chronic lung disease (170). However, the role of acute chest syndrome in causing pulmonary function impairment among children is controversial. Pianosi and colleagues (171) conducted pulmonary function studies in 37 children with sickle cell anemia and found mild decreases in FVC and TLC compared with those in control subjects. There was no difference in lung function between patients who had suffered from acute chest syndrome and those who had not.

**Lung cancer.** Currently, all cancers combined are the second leading cause of death among black Americans, surpassed only by cardiovascular diseases (172, 173). Estimates of the number of lung cancer cases among blacks for 1991 ( $n = 20,500$ ) showed that lung cancer now accounts for approximately 25% of all cancers among male blacks and 13% among female blacks.

National data on cancer incidence and mortality in blacks are available from the population-based registries operated by the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute (table 5) (174). These data show that male blacks had consistently higher lung cancer incidence and mortality than do male whites across the 1970s and 1980s. Among female subjects, rates were comparable in blacks and whites, with both groups showing the same rapid increase (table 5) (174). Survival is somewhat lower in blacks than in whites, regardless of stage (174), but the greater mortality of black men from lung cancer primarily reflects their higher incidence (table 5).

The higher incidence and mortality rates in black men have not been directly explained. Two ecologic analyses of population-based incidence data for metropolitan areas show that the black-white gradient in lung cancer occurrence for men is explained by socioeconomic indicators (175, 176). Devesa and Diamond (175) examined the black-white difference using 1969 to 1971 cancer incidence data for metropolitan areas included in the Third National Cancer Survey. The 10% excess in black men was reduced to a 4% excess with adjustment for median educational level from the census tract, and to a 4% deficit with adjustment for family income. A similar analysis was reported by Baquet and Horn (176) based on SEER data for 1978 through 1982 from San Francisco-Oakland, Detroit, and Atlanta. Adjustment for census tract characteristics, including population density, educational level, and income, reduced an approximately 20% excess for both sexes combined to an approximately 5% deficit. These analyses suggest that environmental correlates of being black rather than black race itself are responsible for the excess lung cancer. Cigarette

Mexican, and Native American lineages (200). Although characterization as Hispanic is useful from a demographic standpoint, the term does not reflect sociocultural or socioeconomic characteristics relevant to health nor distinguish persons who are newly arrived in the United States from those who may be generations removed from their original ancestral heritage and homeland.

The difficulty of operationally identifying Hispanics has posed a barrier in the interpretation of health data and the conduct of health studies (201-204). For example, operant definitions for Hispanic have utilized country of birth, Spanish surname, and response to Hispanic origin items on death certificates and questionnaires (202, 203). Routine vital statistics and other health statistics on Hispanics may be affected by misclassification of both the numerators and denominators of rates; numerator and denominator may be differentially affected and rates consequently biased.

The Hispanic population in the United States is now a large demographic group, numbering approximately 22 million, or 9% of the total population (table 1). However, the proportion of Hispanics is much higher in some geographic areas such as parts of the western and southwestern United States. California contains the largest Hispanic population, numbering nearly 7.7 million and constituting 25% or more of the state's population (205). Hispanics also represent one of the fastest growing ethnic groups in the United States. Between 1980 and 1990 their numbers increased by 53%, and their relative proportion in the population increased from 6.5% to 9.0%. Hispanics, like blacks, are younger than whites. In the 1990 Census the median age of Hispanics was 25.5 yr compared with 34.9 yr for non-Hispanic whites. Only 5.2% of Hispanics were 65 yr of age or older compared with 13.9% of non-Hispanic whites (60).

Until 1990, national mortality rates for Hispanics were not available. Previously, death certificate ascertainment of Hispanic origin was limited to 15 selected states (129). In 1990, information on Hispanic origin was indicated on death certificates in 45 states (New York City was excluded from New York State) and the District of Columbia, accounting for 89% of the Hispanic population in the United States (206). However, 1990 mortality rates specific for pulmonary disease have not been published. As for any group in the population, death certificates have imperfect validity for cause of death (207), and probably a limited validity as a source for determining Hispanic origin (6, 43).

In addition to mortality statistics, national and multistate surveys such as NHIS, NHANES, and the Behavioral Risk Factor Survey conducted by the Centers for Disease Control, are another nationwide source of data. The Hispanic Health and Nutrition Examination Survey (HHANES), conducted from 1982 through 1984, focused on the three major U.S. Hispanic populations (Mexican-Americans, Puerto Rican-Americans, and Cuban-Americans) (208). Unlike other national or multistate surveys, the HHANES utilized Spanish-language instruments and interviewers. Apart from the HHANES, however, the various national surveys have not focused specifically on Hispanics, and prevalence estimates for this ethnic group from these data have often been imprecise because of small numbers of Hispanic subjects.

#### Socioeconomic Characteristics

Although the socioeconomic circumstances of Hispanics in the United States vary, the socioeconomic status of the entire group is lower in general than that of non-Hispanic whites (table 4). Median family income for Hispanics is lower, and Hispanics are more than twice as likely to live below the poverty line. Among Hispanics, Puerto Rican-Americans are the most impoverished, with 38% below the poverty line, and Cuban-Americans are the least, with

14% below the poverty line (209). Of all racial and ethnic groups, Hispanics are least likely to have completed high school (table 4).

Fewer Hispanics than non-Hispanic whites have health insurance (210, 211). Whereas 10% of non-Hispanic whites lack health insurance, the corresponding percentages for Hispanics are 37% for Mexican-Americans, 16% for Puerto Rican-Americans, and 20% for Cuban-Americans (210). The relatively low percentage of Puerto Rican-Americans without health coverage primarily reflects a higher rate of participation in state Medicaid programs (210). A lowered likelihood of having a regular source of care is associated with not having health insurance (210).

Hispanics have a lower rate of annual physician visits per year than do non-Hispanic whites (4.4 versus 4.8 visits/yr), but utilization rates vary among the specific Hispanic groups. Mexican-Americans have the lowest rate (3.7 visits/yr), and Puerto Rican-Americans and Cuban-Americans have rates above those of non-Hispanic whites (6.0 and 6.2 visits/yr, respectively) (210). Hispanics are twice as likely as non-Hispanic whites to report use of the emergency room as a source of primary medical care. Hispanics also tend to have longer and more expensive hospitalizations (209).

Occupational exposures pose a significant risk for respiratory disease, and because Hispanics are a major labor force in farm production, particularly in the West and Southwest, hazards of agricultural work are of particular concern in this population. Nationally, about 70% of farm workers are Hispanic (212). The total farm worker labor pool in the United States comprises at least 6.5 million people (213); estimates for the number of migrant and seasonal laborers range as high as 5.0 million (214). California hosts as many as 1.2 million farm workers, of whom roughly a third are migrant workers, indicating that they travel and stay overnight in different locations in the course of their work. California farm workers are among the most ethnically homogenous of the various national farm worker groupings and are approximately 90% Hispanic (214).

Few studies of respiratory health have been conducted among Hispanic farm workers (215). Gamsky and coworkers (216) recently reported on a respiratory health study of 747 California Hispanic farmworkers. The prevalence of current smoking (34% in men, 13% in women) was comparable to that seen in other studies of Hispanic populations, but reported daily cigarette consumption among current smokers (median, 5 for men and 3 for women) was lower. Crude prevalences for smoking-associated symptoms such as chronic cough, chronic phlegm, and persistent wheeze were also low (1.6, 5.1, and 2.8%, respectively). Current smoking, increased age, female sex, and working  $\geq 8$  months per year in agriculture were associated with increased prevalence of respiratory symptoms. Lung function in these workers was slightly higher than in reference white and Hispanic populations, perhaps reflecting the fact that the subjects were a relatively healthy employed population.

#### Smoking

The most important preventable cause of respiratory disease is tobacco use, a risk factor that has received increasing investigation among Hispanic populations (table 6) (74, 217-236). Nationwide data for 1985 suggest that the frequency of current smoking among Hispanic men is comparable to that of non-Hispanic white men (30.9% versus 30.7%), whereas among Hispanic women the frequency of current smoking is lower than in non-Hispanic white women (16.5% versus 25.8%) (217). Other national and local studies that have included both Hispanics and non-Hispanic whites indicate that smoking prevalence among Hispanic men is equal to or greater than that among non-Hispanic white men (218, 223,

TABLE 6  
SELECTED STUDIES OF SMOKING BEHAVIOR IN U.S. HISPANICS

| Study, Date, (Reference No.)                                      | Target Population and Sample Size   | Current Smoking Prevalence (%)      | Cigarette Consumption for Current Smokers |
|---|---|-------------------------------------|---|
| National Health Survey, 1979-1980 (218)                           | U.S. population (n = 429 Mexican-American; n = 15,402 non-Hispanic white)                                 | Mexican-American males, 41.1        | 12.8 cpd                                  |
|   |   | Non-Hispanic White males, 38.4      | 23.8 cpd                                  |
|   |   | Mexican-American females, 19.9      | 9.2 cpd                                   |
|   |   | Non-Hispanic White females, 32.0    | 19.9 cpd                                  |
| Behavioral Risk Factor Survey, 1981-83 (219)                      | U.S. Population (n = 22,236)  | Hispanic males, 31.5                | -   |
|   |   | Non-Hispanic White males, 35.3      | -   |
|   |   | Hispanic females, 15.0              | -   |
|   |   | Non-Hispanic White females, 28.5    | -   |
| Hispanic Health and Nutrition Examination Survey, 1982-1983 (221) | Mexican-American, Cuban-American, and Puerto Rican-American populations 28 to 74 years of age (n = 5,411) | Mexican-American males, 42.5        | 33.8% > 1 ppd                             |
|   |   | Cuban-American males, 39.8          | 64.1% > 1 ppd                             |
|   |   | Puerto Rican-American males, 41.6   | 52.3% > 1 ppd                             |
|   |   | Mexican American females, 23.8      | 18.8% > 1 ppd                             |
|   |   | Cuban-American females, 24.4        | 48.6% > 1 ppd                             |
| Current Population Survey, 1985 (217)                             | U.S. population (n = 114,342)   | Puerto Rican-American females, 30.3 | 35.1% > 1 ppd                             |
|   |   | Hispanic males, 30.9                | -   |
|   |   | Non-Hispanic White males, 30.7      | -   |
|   |   | Hispanic females, 16.5              | -   |
|   |   | Non-Hispanic White females, 25.8    | -   |

Definition of abbreviations: cpd = cigarettes per day; ppd = packs per day.

227, 228, 231, 233). Studies involving national and local samples have shown that Hispanic smokers, both male and female, tend to consume fewer cigarettes per day than do non-Hispanic whites (218, 225, 227, 228, 231, 233). Rather than smoking a pack of cigarettes daily, as typical non-Hispanic white smokers do, Hispanics more often report smoking one-half pack daily or less.

Studies of smoking in Hispanics have uniformly found that women smoke less than men do. The HHANES confirmed this observation, but it found higher smoking rates and heavier daily cigarette consumption among Hispanics than did other large national studies. Age-adjusted smoking rates were high for male Mexican-Americans (42.5%), Cuban-Americans (41.6%), and Puerto Rican-Americans (39.8%). Corresponding rates for women in these three groups were 23.8, 24.4, and 30.3%, respectively (221). Birth-cohort analyses, however, show increasing smoking among more recent birth cohorts of Cuban- and Puerto Rican-American women (220, 222). In the HHANES data, male Mexican-American smokers tended to be lighter smokers, in that they were less likely to smoke 20 or more cigarettes per day than were Puerto Rican-Americans or Cuban-Americans (33.8% versus 52.3 and 64.1%, respectively). The corresponding figure for male whites based on the NHIS study is 37% (221). As a group, Hispanics favor cigarette brands high in tar and nicotine (221).

In other racial and ethnic groups, educational level and income are strong determinants of smoking behavior (68). For minority populations, the degree of assimilation with the majority population may also affect aspects of lifestyle such as smoking. Several recent studies have examined education level, income, and acculturation, one dimension of assimilation, as predictors of smoking in Hispanics.

The HHANES showed that educational level and income were inversely correlated with smoking prevalence among men; an index of acculturation to the non-Hispanic culture was not consistently associated with smoking in Mexican-Americans, Puerto Rican-Americans, and Cuban-Americans. Among women, income was inversely correlated with smoking prevalence; acculturation was positively correlated with smoking prevalence in Mexican-American women only, and education had no clear effect (221).

Similar results were obtained from a telephone survey of San Francisco Hispanics which showed that smoking was increased in more acculturated women (225). In a survey of Puerto Rican women from the Boston area and Hartford, Connecticut, education and religiosity were inversely related to smoking, and the degree of acculturation was positively related to smoking (237). As in other ethnic and racial groups, parental smoking appears to be an important factor in encouraging smoking among offspring (232, 238). In 1,072 Hispanics residing in a small community in New Mexico, cigarette smoking status (never, former, and current) varied with educational attainment, showing less smoking with increasing years of education (239). Cigarette smoking status did not vary in a consistent pattern with reported language preference; language preference also did not affect continued smoking among ever smokers and numbers of cigarettes smoked.

Several recent studies of smoking in Hispanics that incorporated validation of self-reported smoking with biologic markers of smoking found that Hispanics underreported smoking. Coultas and coworkers (235) compared self-reports of smoking in a survey of New Mexico Hispanics against levels of two biologic markers, salivary cotinine level and end-tidal carbon monoxide concentration. Variable and substantial proportions of male and female self-reported never and former smokers had levels compatible with active smoking. Correction of the self-reports using the biologic marker data increased the prevalence of current smoking by approximately 4 to 5%. Comparison of self-report to serum cotinine level in a sample of 743 Mexican-American participants in HHANES yielded similar findings with regard to misclassification of smoking status by self-report (236). Of 124 self-reported never smokers, five were classified as smoking by level of serum cotinine; seven of 65 self-reported former smokers were similarly classified as smoking by biochemical classification.

Underreporting the numbers of cigarettes smoked may contribute to the pattern of light smoking documented in southwestern Hispanics. Using data from Mexican-American participants in HHANES, Perez-Stable and coworkers (236) found that approximately 20% of self-reported light smokers (one to nine cigarettes per day) had underreported daily tobacco consumption, as indexed

by level of serum cotinine. This finding suggests that the pattern of light smoking found among Hispanic smokers could partially reflect biased reporting of amount of cigarettes smoked; on the other hand, differences in cotinine metabolism between Hispanics and non-Hispanics or very efficient smoking could also explain the findings. Underreporting may be due to cultural factors leading to a reluctance to disclose health habits and symptoms to outside investigators (202, 204).

#### Respiratory Diseases Limited to Childhood

**Hyaline membrane disease.** Information on the occurrence of hyaline membrane disease in Hispanic newborns is virtually nonexistent. The 1988 Vital Statistics (130) for the United States includes numbers of deaths from the respiratory distress syndrome among Hispanics. However, mortality rates were not provided, making comparison with other ethnic groups impossible.

**Sudden infant death syndrome.** Mortality rates from SIDS are similar among Hispanic infants and among non-Hispanic white infants, but lower than among blacks (83, 84). Black and coworkers (84) compared SIDS mortality rates among Hispanics, non-Hispanic whites, and blacks in Cook County, Illinois, from 1975 to 1980. The overall SIDS mortality rates were 1.2 per 1,000 for Hispanics, 1.3 per 1,000 for non-Hispanic whites, and 5.1 per 1,000 for blacks. Similar results were reported by Grether and Schulman (83) based on analysis of SIDS mortality rates in California from 1978 to 1982.

**Cystic fibrosis.** Among Cystic Fibrosis Centers in the United States, 2.5% of registered patients are Hispanic (88). The clinical spectrum and genotypic patterns in Hispanics compared with those in blacks and whites have not been well characterized. Lemna and coworkers (241) analyzed patterns of mutations on 17 cystic fibrosis chromosomes obtained from Hispanic subjects, and they found a pattern similar to that of non-Hispanic whites.

#### Respiratory Diseases of Childhood and Adulthood

**Tuberculosis.** As previously described, recent epidemiologic data paint a sobering picture of tuberculosis, particularly among Hispanics and blacks (94). The 1990 incidence rate for Hispanics (4.2 per 100,000) was five times the rate among non-Hispanic whites. Although the rate of tuberculosis was higher among other minority groups than among Hispanics, Hispanics experienced the largest percent increase in annual tuberculosis incidence from 1985 to 1990. The increase in total cases for 1990 relative to 1985 was 54.7% for Hispanics and 26.9% for non-Hispanic blacks, whereas non-Hispanic whites experienced a reduction of 7.3% during this period. The largest increases were in the 5- to 14-yr age group (102%) and the 25- to 44-yr age group (76.7%).

Mortality data from 1979 to 1981 for a 15-state reporting area indicate an increased risk for tuberculosis among Hispanics (129). The increase affected Mexican-Americans (Standardized Mortality Ratio [SMR] = 3.75) and Puerto Rican-Americans (SMR = 2.75) but not Cuban-Americans (SMR = 1.00). As among non-Hispanic whites, tuberculosis rates for Hispanics were 2- to 3-fold higher in men than in women. In comparison with U.S. whites and non-Hispanic white New Mexicans, Hispanics in New Mexico have approximately 2-fold increased mortality from tuberculosis, but mortality dropped by nearly 100% across the period 1958 to 1982 (242, 243). Increased mortality from respiratory tuberculosis has also been reported for Spanish-surnamed California residents from 1969 through 1971 (244).

Factors discussed in relationship to tuberculosis in blacks probably contribute to the increase in tuberculosis among Hispanics and other minority populations of the United States (94, 98). In

addition, persons immigrating from areas of high tuberculosis endemicity appear to contribute to the increase in numbers of cases. From 1986 through 1990, the percentage of total cases among foreign-born persons increased from 21.6 to 24.4% (94).

**Pneumonia and influenza.** Pneumonia and influenza represent the sixth leading cause of death among Hispanics, after heart disease, neoplasms, cerebrovascular disease, accidents, and COPD (129). In comparison with non-Hispanic whites, age-adjusted death rates for 1979 to 1981 for pneumonia and influenza were increased among Puerto Rican-Americans (SMR = 1.60), but not among Mexican-Americans (SMR = 1.01) and Cuban-Americans (SMR = 0.66). A similar pattern of mortality was seen for 1986 to 1988 (table 2). Mortality from influenza, pneumonia, and bronchitis was not increased among Spanish-surnamed residents of California from 1969 through 1971 (244). By contrast, New Mexico mortality data for 1958 through 1982 show higher mortality in Hispanic than in non-Hispanic whites, with much greater excess mortality in males; both male and female Hispanics experienced declining mortality from pneumonia and influenza during this period (243).

**Asthma.** Increasing information has become available on the morbidity and mortality associated with asthma among U.S. Hispanic populations. Data from the HHANES (1982 to 1984) and the second NHANES (1976 to 1980) were analyzed by Carter-Pokras and Gergen (245). Among 6-month- to 11-yr-old Puerto Rican children, 20.1% had ever had asthma, the highest cumulative prevalence of any ethnic group. Similarly, the point prevalence of active asthma was highest among Puerto Ricans (11.2%). The corresponding figures for the other groups were 2.7% in Mexican-Americans, 5.2% in Cuban-Americans, 3.3% in non-Hispanic whites, and 5.9% in non-Hispanic blacks. Furthermore, the age of onset tended to be younger for Puerto Rican children (50% prior to 1 yr of age) than for those from the Mexican-American community (33% prior to 1 yr of age). Patterns of health care utilization did not explain the differences in asthma prevalence between Puerto Ricans and Mexican-Americans. An increased prevalence of alpha-1-antitrypsin phenotype variants S and Z has been found among Puerto Rican children (246) and adults (247) with asthma in New York City, although a biologic basis for this association is unclear.

Two population surveys conducted in New Mexico provide estimates of the prevalence of asthma in another group of Hispanics and show lower morbidity in this region in comparison with national data (227, 230). In 1978 to 1979, Samet and coworkers (227) surveyed 1,722 adult residents of Bernalillo County, New Mexico. Asthma was less common in Hispanics than in non-Hispanic whites; the overall age-standardized rates for current, physician-confirmed asthma were 0.9 and 3.3% for male Hispanics and non-Hispanics, respectively, and 2.3 and 4.8% for female Hispanics and non-Hispanics. In a 1984 to 1985 survey in a semirural community near Albuquerque, comparable estimates of the prevalence of asthma in Hispanic adults were obtained (230). Current, physician-diagnosed asthma was more common in male than in female patients, and it increased with age. For male children younger than 6 yr of age the prevalence was 2.8%, and it increased to 8.1% among children 13 to 17 yr of age. The corresponding estimates for female children were 1.2 and 3.6%.

Except for New York City, little data are available on patterns of hospitalization for asthma among Hispanics. Carr and coworkers (124) examined hospitalization rates in New York City residents younger than 35 yr of age for the period 1982 to 1986. The average annual rate of hospitalization was 62.9 per 10,000 among Hispanics, 59.9 per 10,000 among blacks, and 12.2 per 10,000



among non-Hispanic whites. Median family income was inversely related to hospitalization, and was found to be the most important predictor in multivariate statistical analyses.

In a cross-sectional survey to describe morbidity among Hispanic children with asthma, Wood and coworkers (248) enrolled 78 children seen at the University of Texas Health Science Center at San Antonio. Overall, these children were mildly impaired, with a median FEV<sub>1</sub> of approximately 90% of predicted and a mean "illness score" of 24.2 (score of 28 indicates no impairment by illness). However, these data are limited because of low participation (44.5% of eligible children) and the lack of a comparison group.

Mortality rates for asthma vary among the different Hispanic populations, and patterns of mortality from asthma vary in different regions of the country. Mexican-Americans and Cuban-Americans have low rates compared with Puerto Rican-Americans (SMR = 4.63) (129). It is not clear whether this increase reflects diagnostic biases, genetic predisposition, or factors associated with the urban environment (125). Asthma mortality rates for 1969 to 1977 in New Mexico Hispanics were lower than those in non-Hispanic whites, but the numbers of deaths were small (242). In contrast, Carr and coworkers (124) found a higher annual mortality among Hispanic residents (1.3 per 100,000) of New York City than among non-Hispanic whites (0.4 per 100,000).

**COPD.** Among the three major Hispanic groups included in the HHANES, Puerto Rican-Americans (2.9%) demonstrated higher prevalence of reported chronic bronchitis, a surrogate for COPD, than Mexican-Americans (1.7%) or Cuban-Americans (1.7%) (249). Surveys in New Mexico have shown physician-diagnosed chronic bronchitis or emphysema to be less common among Hispanics (227, 230) than among non-Hispanic whites. As assessed by spirometry, chronic airflow obstruction was present in less than 1% of Hispanic adults included in the survey of a New Mexico community (230).

Mortality rates from chronic obstructive lung diseases and allied conditions are reduced among the three major Hispanic population groups when compared with non-Hispanic whites (129) (table 2). Similar reductions in mortality from COPD were noted for both male and female Hispanics in comparison with non-Hispanic whites. However, male Hispanics demonstrated an approximate 2-fold increased mortality from COPD in comparison with female Hispanics. Mortality data for New Mexico have also shown lower mortality in Hispanic men than in non-Hispanic white men; however, from 1958 through 1982, mortality from COPD rose steeply in Hispanic men, from 5.0 per 100,000 in 1958 to 30.1 in 1978 to 1982 (250). During this same time, mortality rates for COPD in Hispanic women also increased, but remained comparable to those in non-Hispanic whites (250).

**Lung cancer.** National and regional vital statistics have shown differing patterns of occurrence of lung cancer in Hispanic populations throughout the United States in comparison with non-Hispanic whites. Since the 1950s, descriptive studies of mortality documented differing patterns of lung cancer in Hispanics and non-Hispanic whites in the western and southwestern United States. In California during the 1950s and 1960s, mortality rates from lung cancer among older Mexican-born women were two to three times the rates for all California women (251, 252). Lung cancer mortality rates for Texas and New Mexico during the 1960s and 1970s showed a similar pattern of age-specific rates (242, 250, 253), although Hispanic women in the West and Southwest have lower overall lung cancer mortality rates than non-Hispanic white women (250, 254-256). In New Mexico (242), Texas (253), California (256, 257), and Colorado (254), mortality and incidence rates have been much lower in Hispanic than in non-Hispanic white

men. The elevated rates of lung cancer mortality in Hispanic women in the region were attributed to a pattern of heavy smoking beginning at an early age by a cohort of women born in Mexico before 1900 (252, 253). Data from a case-control study in New Mexico documented an unusual pattern of smoking hand-rolled cigarettes by older Hispanic women that may also have contributed to this cohort phenomenon (228). As this group has aged out of the population, their impact on lung cancer mortality rates has lessened (218, 223).

Mortality data for the 15 reporting states for 1979 to 1981 indicate that rates for all malignant respiratory diseases, predominantly lung cancer but also including cancers of the larynx, pleura, and other intrathoracic locations, are reduced among the three major Hispanic groups (SMRs ranging from 46 to 53) for both men and women (129). A nationwide analysis limited to foreign-born Cubans, Mexicans, and Puerto Ricans provided similar results for the same period (258). Although comparable reductions in respiratory malignancy mortality for Hispanics in comparison with non-Hispanic whites are found for men and women, Hispanic men are at a 3- to 4-fold increased risk for respiratory cancer in comparison with Hispanic women. Lung cancer incidence rates among Hispanic men (259) and women (260) in Florida during 1982 to 1983 were also reduced in comparison with non-Hispanic whites. Similarly, Mexican and Puerto Rican immigrants in Illinois have reduced lung cancer mortality when compared with non-Hispanic whites (261). Using U.S. Surveillance Epidemiology and End Results (SEER) data from all areas (excluding Puerto Rico) for comparison, Puerto Rican men and women living in Long Island, New York, were found to have slightly lower mortality rates for lung cancer (262). However, lung cancer mortality rates were found to be 3.0 (men) and 4.1 (women) times the rates among residents of Puerto Rico.

The presently observed lower rates of lung cancer in Hispanics appear largely to reflect differences in smoking between Hispanics and non-Hispanic whites. A case-control study of lung cancer cases, 1980 to 1982, was conducted to evaluate the risks of smoking and other factors among New Mexico Hispanics and non-Hispanic whites (228). The study noted comparable risks associated with smoking among Hispanics and non-Hispanic whites, suggesting that the reduced rates in Hispanics were attributable to their lower cigarette consumption and not to some other correlate of Hispanic ethnicity. A mortality study of Mexican-American women in Texas during 1970 to 1979 noted stable lung cancer mortality rates (approximately 30 per 100,000) in the presence of increasing rates for non-Hispanic white women (226). The lower lung cancer rates for Hispanic women were consistent with lower smoking prevalence (18.5% versus 31.6% for non-Hispanic white women).

Some regional data suggest that the low rates of lung cancer in Hispanics are rapidly increasing. New Mexico mortality data for the period 1958 to 1982 indicate increasing lung cancer rates for successive birth cohorts among Hispanics (250). Between 1958 and 1962 and 1978 and 1982, the mortality rate for Hispanic men increased from 10.1 per 100,000 to 28.8 per 100,000; the corresponding change for Hispanic women was from 4.8 to 11.2 per 100,000 (250). However, rates remained below those of the U.S. general population for both Hispanic men and women. Savitz (254) reported doubling of lung cancer incidence rates for those with Spanish surnames residing in the Denver area from 1969 to 1971 through 1979 to 1981.

#### Prediction of Lung Function

Only a few lung function prediction equations for U.S. Hispanics



have been published, and the extent to which they should be applied beyond the source populations is uncertain. The relevance of prediction equations based on studies in Mexico and Central and South America is also uncertain (263, 264).

Hsu and colleagues (187, 265, 266) studied ventilatory function of Mexican-American, black, and non-Hispanic white schoolchildren, 7 to 20 yr of age in Houston, Texas. For spirometric measures, the values were selected from the best of two curves; peak expiratory flow rate was measured with a Wright peak flowmeter until two satisfactory blows were obtained. Predicted values, based on either standing or sitting height, were close for the Mexican-American and non-Hispanic white regression equations.

Coultas and coworkers (267) developed prediction equations for ventilatory function based on a community survey of Hispanics in rural New Mexico; the published equations cover the age ranges 6 through 18 yr and 25 through 80 yr. The subjects had lower pulmonary function than predicted by comparison reference equations from non-Hispanic whites; the lowest 5% of the Hispanic population was placed at 73 to 80% of expected based on equations from non-Hispanic populations.

A study of Hispanic agricultural workers in California found higher FVC and comparable FEV<sub>1</sub> when compared with reference populations of non-Hispanic whites and New Mexico Hispanics (216). These differences may be attributed to the inclusion of employed workers in the California study, whereas the comparison equations were derived from community-based studies and more likely to include less healthy, unemployed persons.

In summary, lung function predictions based on external reference populations that are not exclusively Hispanic tend to overestimate predicted lung function in Hispanics, and they may lead to erroneous classification as abnormal for Hispanics with low-normal pulmonary function. However, the available data are insufficient to merit use of a specific correction factor, as has been advocated for African-Americans.

### Summary

The diverse Hispanic population of the United States is large and rapidly growing. Available data indicate that the respiratory health of Hispanics is variable among the principal subgroups (table 2). For example, morbidity and mortality rates from asthma are highest among Puerto Ricans when compared with Cuban-Americans, Mexican-Americans, and other Hispanics from New Mexico. In general, Hispanics have lower rates than do non-Hispanic whites of malignant and nonmalignant conditions related to tobacco use. Although morbidity and mortality from COPD are more common among non-Hispanic whites than among Hispanics, Puerto Ricans have the highest COPD mortality of all Hispanic groups (table 2).

Hispanic women, in general, have reduced risk for respiratory disease when compared with men, which probably relates to a low prevalence of smoking in this group. However, disturbing data from the HHANES and other sources suggest that smoking rates among Hispanics are higher than previously believed, and that Hispanic women are not exhibiting decreasing rates in successive cohorts, leading to convergence of rates between men and women. These data may augur a tragic rise in lung cancer and other tobacco-related diseases in coming years.

### ASIAN/PACIFIC ISLANDERS

The Asian-Pacific Islander population in the United States is characterized by extremely heterogeneous origins, cultures, languages, physical appearances, socioeconomic status, and educational lev-

TABLE 7  
ASIANS AND PACIFIC ISLANDERS IN THE UNITED STATES\*

| Asian Groups            |                  |                       |             |
|-------------------------|------------------|-----------------------|-------------|
| Asian Indian            | Cernan           | Japanese              | Okinawan    |
| Bangladeshi             | Chinese          | Javanese              | Pakistani   |
| Bhutanese               | Filipino         | Korean                | Sikkimese   |
| Borneo                  | Hmong            | Laotian               | Singaporean |
| Burmese                 | Indochinese      | Malayan               | Sri Lanka   |
| Cambodian               | Indonesian       | Maldivian             | Thai        |
| Celebesian              | Iwo Jiman        | Nepali                | Vietnamese  |
| Pacific Islander Groups |                  |                       |             |
| Polynesian              | Micronesian      | Melanesian            |             |
| Hawaiian                | Guamanian        | Fijian                |             |
| Samoa                   | Mariana Islander | New Hebrides Islander |             |
| Tahitian                | Marshallese      | Papua New Guinean     |             |
| Tokelauan               | Palauan          | Solomon Islander      |             |
| Tongan                  | Salpanese        |                       |             |
|                         | Tinian Islander  |                       |             |

\* Source: Asian American Health Forum (269).

els (268). The "Asian" group consists of 28 ethnic groups (table 7) (269), according to categorizations applied by the U.S. Census Bureau (270). Similarly, the "Pacific Islanders" consist of three major ethnic groups (Polynesian, Micronesian, and Melanesian) that originate from more than 10,000 islands (also termed Oceania) spread across 30 million square miles in the central and southern Pacific Ocean and including New Zealand. These three major ethnic groups conveniently reflect regions of origin, but they do not adequately define the 15 population subgroups or their diverse cultural practices. More than 32 primary languages are spoken by the ethnic groups that constitute Asians and Pacific Islanders.

Asians and Pacific Islanders are currently the third largest and the fastest-growing minority group in the United States (table 1). The population of Asians and Pacific Islanders has doubled each decade since 1970. The 1.43 million Asians and Pacific Islanders (0.7% of total U.S. population) in 1970 increased by 144% to 3.50 million (1.5%) in 1980 (270, 271). By 1990, the Asians and Pacific Islanders population increased by 108% to 7.27 million (2.9%), of which 3.56 million were male and 3.72 million were female (270, 272, 273).

The composition of the Asian and Pacific Islanders population in the United States has also rapidly changed because of different waves of immigrants. Prior to the 1960s, Asians came primarily from China, Japan, and the Philippines, resulting in well-established groups, now with several generations of native (U.S.)-born Asians. During the 1970s and 1980s, the Asian influx was from more than 20 countries, with a substantial increase in immigrants from Vietnam, Laos, Cambodia, Thailand, and Indonesia. In 1980, 73% of Asians and Pacific Islanders in the United States were foreign born, most immigrating during the previous decade (274). Immigration continued during the 1980s, with increasing numbers from Asia except for Laos and Cambodia (268). From 1980 to 1984, Asia accounted for 48% of immigrants legally admitted to the United States (274). Approximately 3.8 million Asians and Pacific Islanders immigrated to the United States between 1980 and 1990, with doubling of the Filipino, Vietnamese, Korean, and Asian Indian groups (272). In 1989, more than 15,000 immigrants came from Vietnam, 40,000 from the Philippines and India, 30,000 from China, and 55,000 from Korea (269). In 1990, the largest Asian ethnic populations in the United States were the Chinese (22.6%),

Filipinos (19.3%), Japanese (11.6%), Asian Indians (11.2%), Koreans (11.2%), and Vietnamese (8.4%). Pacific Islanders constituted 15.7% of the Asian-Pacific Islander populations (270).

In 1990, approximately 55.6% of Asians and Pacific Islanders lived in the West, 18.4% in the Northeast, 15.4% in the South, and 10.6% in the Midwest (270). More than 70% of the total Asian and Pacific Islanders population resides in seven states: 39.1% in California, 9.5% in New York, 9.4% in Hawaii, 4.4% in Texas, 3.7% in New Jersey, 3.9% in Illinois, and 2.9% in Washington (270). California has the nation's largest Asian and Pacific Islander population (2.84 million; 125% increase between 1980 and 1990) (270, 272, 273).

Similar to the other minority groups, Asians-Pacific Islanders are younger than whites. In the 1990 Census (60) the median age of Asians-Pacific Islanders was 29.8 yr compared with 34.4 yr for whites. Only 6.2% were 65 yr of age or older compared with 13.9% for whites.

Morbidity and mortality from respiratory and other diseases in Asians and Pacific Islanders are incompletely characterized, and the relevant statistical data continue to be limited in quantity, quality, and recency. Nationwide and regional descriptive surveys have been conducted, but the data are limited and possibly misleading because of the lack of standardized data collection and coding methods, cultural barriers in survey-taking, inadequate sample sizes, consolidation of ethnic groups into one category ("Asian"), and mixing of Asians with non-Asian minorities into non-specific categories of "other race" (275). The national health surveys, e.g., the National Health Interview Survey (NHIS), have included Asians and Pacific Islanders, but the samples have been too small to draw valid conclusions for subgroup comparisons. The states record vital statistics (birth and death certificates) according to their own criteria for classification of race and ethnicity, creating nonuniform databases (6). Asians were misclassified on 21.1% of death certificates in 1986, most often as white decedents; as a consequence, white mortality may be inflated and mortality among nonwhite races, including Asians and Pacific Islanders, reduced (6). Asians and Pacific Islanders are usually considered as a separate racial category (with breakdown of ethnic group and nativity) only in states with a large total Asian population. Thus, the populations of California and Hawaii are the two major sources of mortality data on Asians and Pacific Islanders. Data from California can be considered more representative than Hawaiian data because the former state has a larger, more pluralistic and heterogeneous Asian and Pacific Islander population (276). Nativity is a critical factor because mortality rates consistently range from one and a half to six times higher among foreign-born Asians than among native (U.S.-)born Asians and Pacific Islanders.

#### Socioeconomic Characteristics

A bimodal distribution of socioeconomic status characterizes the Asians and Pacific Islanders in the United States. This pattern follows from the large population of U.S.-born, later-generation Asians and Pacific Islanders at higher socioeconomic levels and the rapidly increasing number of foreign-born immigrants, regardless of ethnicity, at lower socioeconomic levels. The "nativity" factor has been largely ignored, but it is key in the understanding of the socioeconomic and educational (and health) diversity in the Asian and Pacific Islander populations in the United States. For example, the median family income for Asians and Pacific Islanders is higher than that for other minority groups and for whites (table 4) (271). However, these figures may be misleading because of a larger number of wage earners per Asian household and the presence of well-established (U.S.-born) Asian groups.

In 1980, median family income for Asians and Pacific Islanders was \$23,600 (versus \$20,800 for whites), but it ranged from \$12,840 among families of Vietnamese origin to \$27,350 among Japanese-Americans (277). In 1980, approximately 66% of Laotians, 48.7% of Cambodians, 33.5% of Vietnamese, and 25.5% of Samoans, compared with 12.4% of the overall U.S. population, lived below the poverty level (269).

The Asians and Pacific Islanders tend to live in urban and suburban areas (273). The recent influx has stressed basic local and regional services such as schools, social services, and the health care system, and it has created increased competition for jobs and for land use. On the other hand, former mountain tribesmen, the Hmongs of Vietnam, came in large numbers to the San Joaquin Valley of California during the 1980s to farm (278).

The educational status of Asians and Pacific Islanders has also been bimodal. Asians and Pacific Islanders have the highest proportion of high school graduates among the racial and ethnic groups of the United States (table 4). In 1990, more than a third of Asians and Pacific Islanders were college graduates (table 4). However, in 1980, more than 40% of Laotians, Cambodians, Vietnamese, Samoans, and Tongans in the United States did not graduate from high school compared with 34% for the overall U.S. population (269).

#### Smoking

Asian immigrants originate from countries where cigarette consumption has generally increased during the past two decades, especially the developing nations (279, 280). China alone consumes almost 30% of the world's annual total of 1.5 trillion cigarettes (280). Male smoking rates are now higher in developing countries than in many industrialized countries.

Smoking by Asians and Pacific Islanders has been little studied. Although prevalence data suggest that Asians and Pacific Islanders have the lowest current smoking rates (15 to 29%) of the racial groups (68), the national data are limited by small sample sizes and the lack of categorization into ethnic subgroups. Smoking prevalence tends to vary according to age, sex, and ethnic subgroup (281-287). For example, a 1977 NHIS found that 28.7% of Asian and Pacific Islander respondents were smokers (281). A 1979 California survey (284) found an overall smoking prevalence of 20.8% in Asians and Pacific Islanders, substantially lower than in whites (32.7%), blacks (40.1%), and Hispanics (29.0%). Smoking prevalence among Chinese-American men was 26.6% for men younger than 50 yr of age and 35.2% for men  $\geq$  50 yr of age, whereas rates for Japanese-American men in the two age groups were 34.8 and 17.7%, respectively. Smoking prevalence among women in the two ethnic groups ranged from 3.6% among Chinese-American women younger than 50 yr of age to a high of 18.1% among Japanese-American women 50 yr of age and older.

Similar results were found in a survey of California residents conducted in 1990 and 1991 (282). Overall, 23.5% of Asian and Pacific Islander men smoked compared with 24.8% of non-Hispanic white men. The corresponding data for women were 8.9 and 21.8%, respectively. The highest prevalence rates of smoking among the Asian and Pacific Islander groups were 35.8% among Korean men and 14.9% among Japanese women. Overall, Asians and Pacific Islanders were more likely to be light smokers (< 15 cigarettes/day) than were non-Hispanic whites.

In a survey of 215 randomly selected foreign-born Vietnamese adults living in the San Francisco Bay Area (285), 9% of the women were current smokers (compared with 27% of U.S.-born women), and 56% of the men were current smokers (compared with 32% of U.S.-born men). Smoking by men was significantly increased

by residence in the United States for 9 yr or less, limited English proficiency, income below the poverty level, and not knowing that smoking causes cancer. In this same sample of Vietnamese smokers, 88% of male smokers reported smoking high-tar, high-nicotine brands of cigarette, and 54% always smoked soon after awakening in the morning. Most (82%) wanted to quit smoking, but 71% thought that it would be difficult. The high prevalence rate of cigarette smoking for Vietnamese men was confirmed in a larger California-wide telephone survey of 1,011 Vietnamese respondents (286). Newly arrived Southeast Asian immigrants who intended to reside in the Seattle area were surveyed about smoking habits in 1989 (287). The prevalence of smoking was higher among men (42.5%) than among women (5.7%). Of the ethnic groups Laotians had the highest prevalence (51.2%) followed by Vietnamese (41.7%) and Cambodians (32.8%).

#### Respiratory Diseases of Childhood

*Hyaline membrane disease.* The incidence and mortality rates of hyaline membrane disease and of respiratory distress syndrome among Asian and Pacific Islander newborns in the United States is unknown.

*Sudden infant death syndrome.* The limited data on mortality rates from SIDS among Asians and Pacific Islanders and non-Hispanic whites provide conflicting results. Grether and Schulman (83) analyzed SIDS mortality rates in California from 1978 to 1982 and found infant mortality rates of 1.5 per 1,000 Chinese, 1.8 per 1,000 Japanese, 1.1 per 1,000 Filipino, and 0.5 per 1,000 Vietnamese compared with 1.5 per 1,000 white infants. However, in an analysis of SIDS mortality rates from a nationwide sample, Wang and coworkers (288) found higher mortality rates among Chinese (1.46 per 1,000) and Japanese (1.60 per 1,000) infants than among white infants (1.05 per 1,000).

*Cystic fibrosis.* Although data on the occurrence of fibrosis among Asians in the United States are limited, available information suggests that it is rare. The incidence in Hawaiians has been estimated at 1 in 90,000 live births (289). In 1990, 0.2% of patients registered at Cystic Fibrosis Centers in the United States were Asian (88).

#### Respiratory Diseases of Childhood and Adulthood

*Tuberculosis.* General trends indicate high rates of tuberculosis among Asians and Pacific Islanders, but little is known about the associated respiratory morbidity and mortality or their patterns in Asians and Pacific Islanders subgroups. Differences in tuberculosis rates among ethnic groups appear mainly attributable to differences in risks of infection, i.e., largely explicable by the high prevalence and risk of tuberculosis infection in their countries of origin (290). The high rate of tuberculosis infection and disease among Asians and Pacific Islanders is probably related to a complex interplay of cultural, economic, occupational, and nutritional factors, infection with the human immunodeficiency virus (HIV), high prevalence of latent tuberculosis infections, and lack of access to health care and preventive services. Southeast Asians have high rates of diseases such as tuberculosis that are associated with poverty (291). Tuberculosis is by far the most commonly imported infection in Southeast Asian refugees, who are at risk for subsequent reactivation of latent infection.

Overall, effective screening, prophylaxis, and chemotherapy have decreased the incidence of tuberculosis in the United States (93, 290, 292). However, the decline of tuberculosis cases in the United States was broken in the late 1970s by a large influx of Southeast Asian refugees. During 1979 to 1981, Southeast Asian refugees had an overall prevalence of tuberculosis at entry of 1,138

cases per 100,000 (92% pulmonary, 7% extrapulmonary, 1% unreported site) and a 14- to 70-fold higher tuberculosis case rate than the white population (293). In 1985, 22,201 new tuberculosis cases were reported in the United States, and Asians and Pacific Islanders accounted for 11.4% of these new cases (96), resulting in a group case rate of 49.6 per 100,000, the highest for any racial minority group and 8.7 times higher than the rate in the white population (294). Tuberculosis among Asians and Pacific Islanders in 1983 occurred almost entirely (93.6%) among foreign-born persons (countries of origin primarily Laos, Kampuchea, Vietnam, the Philippines, Korea, and People's Republic of China) (294). In 1986, 62% of all reported tuberculosis cases in the United States were among racial and ethnic minorities (including nonwhites and Hispanic whites). This percentage decreased to 51% in 1989, with Asians and Pacific Islanders contributing 12% of all tuberculosis cases. Overall, Asians in the United States have a 9-fold greater risk of developing tuberculosis than does the white population.

Immigrants from areas with a high prevalence of tuberculosis have an increased risk of disease during the first few years after immigration; the risk varies among the different ethnic groups (293-295). The risk is highest in the year immediately after immigration, between 30 and 45%, and by the end of the second year it falls to between 5 and 10%. After the second year, the occurrence of cases gradually declines to less than 5% after 10 yr or more of residency in the United States (294).

The age distributions of tuberculosis cases differ between whites and Asians, who have a higher proportion of cases at younger ages than do whites. In 1985, 44.5% of tuberculosis cases among Asians and Pacific Islanders occurred in those younger than 35 yr of age (294). Among non-Hispanic whites only 14.0% occurred before 35 yr of age.

During the early 1980s, standard tuberculosis skin testing (Mantoux) with commercial purified protein derivative (PPD) showed positive for 38% of Southeast Asians in Denver (296) and for 55% in San Diego (297). The rates of tuberculosis infection were highest in the middle-aged and elderly immigrants and in the Cambodians and Vietnamese (296, 297). Remote tuberculosis infection and nutritional factors may account for negative skin tests in infected immigrants.

Isoniazid (INH)-resistant *Mycobacterium tuberculosis* is common in Asian immigrants. From 1975 to 1980, the prevalence of INH-resistant *Mycobacterium tuberculosis* was as high as 10% in Southeast Asian refugees and about one third of isolates were resistant to at least one antituberculosis drug (298). Resistance to INH and streptomycin was found most frequently, 25 and 22% of specimens tested, respectively. Resistance to ethambutol and rifampin was much less common (3% each), and resistance to rifampin was always associated with INH resistance (298). Currently, the overall prevalence of INH resistance is approximately 5% in the United States, compared with 12% in Hong Kong, 22% in India, 10% in Japan, 14% in Korea, 23% in Taiwan, 14 to 33% in the Philippines, and 44% in Samoa (292).

Drug resistance in the United States varies by region and Asian and Pacific Islander subgroup. From 1977 to 1981, Hawaii had a low prevalence of tuberculosis (2.9 cases per 100), and low levels of primary drug resistance to INH, streptomycin, and para-aminosalicylic acid (PAS) in Asians with tuberculosis and no history of previous antituberculosis therapy (299). In San Francisco, 17% of the Chinese population who developed tuberculosis had an INH-resistant organism. The prevalence of INH-resistant isolates was 15% in Southeast Asian refugees with active tuberculosis living in Seattle (300). A community-based study (301) in Santa Clara County, California, from 1984 to 1986 found a tuberculosis

case rate in Southeast Asians of 100 per 100,000 population per year (more than 17 times the national case rate for non-Hispanic whites). Among foreign-born cases, 61% of the drug-resistant and 43% of the drug-susceptible isolates were in patients of child-bearing age (ages 20 to 40 yr). Immigrant cases accounted for 86% of drug-resistant isolates, and immigrants from Asia had the highest frequencies of resistance (33 to 45%) and a drug-resistant tuberculosis case rate of 30 per 100,000 population per year (more than 15 times the rate of drug-resistant tuberculosis among U.S.-born, non-Hispanic whites). INH resistance was present in 21% of Southeast Asians. Rifampin-resistant tuberculosis was isolated in 5% of Vietnamese immigrants, 5% of Filipinos, and 18% of Koreans. Drug-resistant strains (to one or more drugs) from Southeast Asians were isolated only in the Vietnamese, among whom primary drug resistance was found in 33% of 105 cases who did not have a previous history of tuberculosis, compared with 50% of 20 cases who had a previous history of tuberculosis. The variability in susceptibility patterns of isolates among different immigrant groups and the increased risk of drug-resistant tuberculosis in children born in the United States of foreign-born parents emphasize the need to examine local patterns of drug-resistant tuberculosis.

**Pneumonia and influenza.** In California during the period 1986 to 1987, the overall mortality for pneumonia and influenza among "Asian and Other" was lower than that for whites, but it varied widely for specific ethnic groups (figure 2) (276). Chinese-Americans in California have a proportionate mortality ratio (PMR) of 4.4% for pneumonia and influenza (compared with 4.1% in whites), whereas other ethnic subgroups have lower PMRs than do whites (276). Pneumonia was the most commonly reported pulmonary illness in Japanese-American men in Honolulu, with a prevalence of 4.2 cases per 100 (302).

**Other pulmonary infections.** The etiologic agents for the majority of pulmonary bacterial infections in Southeast Asians do not differ appreciably from organisms indigenous to the American population, except for *Pseudomonas pseudomallei*. Infection with *Pseudomonas pseudomallei* may occur in a higher proportion of Southeast Asians and Americans returning from Southeast Asia

(303). Melioidosis can occur abruptly after a prolonged latent period and commonly presents as diffuse upper lobe involvement with early cavitation (303). This picture may be confused or be associated with pulmonary tuberculosis.

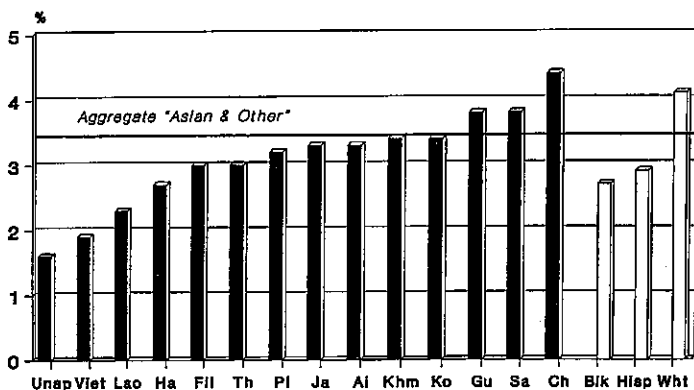
The intestinal parasites that have high incidence and can potentially cause symptomatic pulmonary disease include *Paragonimus westermani*, *Ascaris lumbricoides*, *Strongyloides stercoralis*, and microfilariae. Intestinal parasites have been frequently detected in Southeast Asian refugees, with differences among the ethnic subgroups (296, 297, 304). Each of these organisms has the potential for producing pulmonary disease after a latent period. For example, cases of pulmonary paragonimiasis with hemoptysis, cystic lung lesions, or large pleural effusions in nonimmunocompromised Southeast Asian immigrants have been reported in different areas of the United States (305, 306). The incidence of parasitic pulmonary disease related to hematogenous or contiguous migration through the lung has been relatively low, perhaps because of interruptions in the life cycle of the parasites, hygienic strategies, detection, and effective treatment (303, 307).

As discussed in the section on blacks, race may be an important risk factor for dissemination of coccidioidomycosis. In a group of 230 acutely infected patients from central California (105), fungal dissemination occurred in 18.7%. Among 17 patients of Asian and Pacific Islander heritage, 41.2% developed disseminated disease, compared with 11.2% among whites.

Infection with the human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS) is rare among Asians and Pacific Islanders when compared with whites (308). Asians and Pacific Islanders account for only 1% of all AIDS cases in the United States. However, the number of new cases is rapidly increasing. New cases of AIDS in Asians and Pacific Islanders increased 150% between 1985 and mid-1988, the highest rate of increase among all American racial and ethnic groups.

**Asthma.** The occurrence of asthma among Asians and Pacific Islanders in the United States has primarily been examined in Hawaii. Among 7,498 Japanese-American men recruited between 1965 and 1968 for the Honolulu Heart Program, 3.8% reported ever having bronchial asthma, and 1.7% were currently bothered by asthma (302). The prevalence of self-reported asthma in non-military Asian and Pacific Islander residents of Hawaii was 37.7 per 1,000 (4,256 of 112,938 sampled subjects), with increasing annual rates from 1980 to 1986 and involving primarily (61%) Japanese, Hawaiians, Chinese, and Filipinos (309). The highest prevalence occurred in 6- to 11-yr-old children (66.6 per 1,000), residents of Hawaiian or part-Hawaiian ethnicity (57.4 per 1,000), and those living on the island of Kauai (56.6 per 1,000). Asthma rates varied inversely with family income level. Information on patterns of hospitalization and mortality from asthma are not available for Asians and Pacific Islanders in the United States.

**COPD.** As with asthma, information on morbidity and mortality from COPD among Asians and Pacific Islanders is sparse. In 1965, 6,346 Japanese-American men 46 to 68 yr of age had spirometric testing as part of the Honolulu Heart Program (302). Of these men, 48% were current cigarette smokers, 25% were former smokers, and 27% were never smokers. Airflow obstruction, defined by a Z score (observed FEV<sub>1</sub> minus predicted FEV<sub>1</sub>/standard deviation of numerator) of greater than 1 standard deviation, was found in 11.7%. 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 (310). Because the definition of airflow obstruction used in this investigation did not exclude participants with asthma, the



**Figure 2.** Proportionate mortality ratios (PMR) in Asian-Pacific Islander (API) subgroups in California (1986-1987) for pneumonia and influenza. The aggregate "Asian & Other" horizontal line indicates the mean PMR of all API, which may be misleading in comparisons with other racial groups. Abbreviations: AI = Asian Indian; CH = Chinese; FIL = Filipino; GU = Guamanian; HA = Hawaiian; JA = Japanese; KHM = Cambodian; KO = Korean; LAO = Laotian; PI = Other Pacific Islander; SA = Samoan; TH = Thai; UNSP = Asian-unspecified; VIET = Vietnamese. (Reproduced with permission from the American Asian Health Forum, Inc., 276.)

prevalence estimates probably exaggerate the occurrence of COPD.

A low prevalence of COPD among Asians and Pacific Islanders in Hawaii compared with that among whites is also supported by mortality statistics for COPD in California. During the period 1986 to 1987, overall mortality for COPD among "Asian and Other" in California was lower than among whites, but it varied widely for specific ethnic groups (276).

Serum protease inhibitor (Pi) status is a well-defined, although rare, risk factor for emphysema, and the frequency of Pi phenotypes varies among different racial groups (311, 312). The normal Pi M gene is predominant in all populations. The S gene is very common in Spaniards but absent in Africans and Hindus. The Z gene occurs in most populations studied, but it is rare and perhaps absent in others such as the Japanese, Southeast Asians, Asian Indians, and Micronesians (311, 313). Only Pi M phenotype was found in Japanese-American patients with moderate or severe COPD and healthy control subjects in Honolulu, Hawaii (312). The evidence suggests that, as in other populations, Pi status contributes little to the occurrence of COPD in Asians and Pacific Islanders.

*Sarcoidosis.* The occurrence among Asians and Pacific Islanders of the United States is not known, but the rate, based on incidence and prevalence estimates from the United States and from Asian and European countries (131), is probably low compared with that among blacks and whites. Sarcoidosis appears to be rare among Chinese populations in California, Hawaii, Singapore, Vietnam, and mainland China, but the actual prevalence may be recognized to be higher as awareness increases (314). At the Third International Conference on Sarcoidosis in 1963, Bauer and Lofgren (134) summarized worldwide prevalence estimates for sarcoidosis. In a Japanese survey, the prevalence was 5.6 per 100,000, a low figure compared with that for many European countries, generally 10 to 20 cases per 100,000, but ranging as high as 64 per 100,000 in Sweden. Among Japanese patients with sarcoidosis, prognosis may be associated with specific HLA-DR antigens (315, 316).

*Lung cancer.* The occurrence of lung and other cancers may change after immigration; for many sites, rates have tended to assume a level intermediate between that in the home and host countries (317, 318), suggesting effects of environmental factors such as smoking and dietary habits. Lung cancer is the most common form of malignancy among Asians and Pacific Islanders. Incidence rates for lung cancer in Chinese, Filipinos, and Japanese in the United States from 1978 to 1981 were comparable to or lower than those in whites, except for higher rates in Chinese and Hawaiian women. Incidence rates for Chinese, Japanese, Filipino, and Hawaiian men were 62.6, 45.1, 38.1, and 100.9 per 100,000, respectively. These rates were lower than those in white (81.0) and black men (119.0), but higher than those in Hispanics (34.3) and Native Americans (14.5). In Los Angeles County the proportional incidence ratios (PIR) for lung cancer from 1972 to 1988 among Chinese and Vietnamese men were 102 and 121, respectively, and 123 and 78 among women, respectively (319). Overall, lung cancer rates for Southeast Asian men are 18% higher than for whites. Overall cancer rates and total mortality from all causes of cancer are remarkably low in Chinese, Japanese, and Filipinos compared with rates in other racial/ethnic groups in California (Los Angeles County) (320-322). Data on cancer incidence in Pacific Islander populations in the United States are relatively limited. However, Polynesians, as represented by native Hawaiians, have relatively high cancer incidence rates for all sites combined, including lung cancer, in both sexes (268, 323).

Standardized mortality ratios (SMR) in the United States for lung cancer in foreign-born (issei) and native-born (nisei) Japanese men and women during the years 1968 to 1972 were lower than those in Japan, but issei SMR has increased since 1960 (324). Foreign-born (idai) Chinese men and women in California, Hawaii, and New York City, as well as Chinese men in Taiwan during the period 1968 to 1972 showed high SMRs for lung cancer, presumably because of smoking and other environmental factors (325). Regional risks for cancer vary greatly in China (and to a lesser degree in Japan), so that studies of Chinese migrants must take into account the region of origin (317, 326). The birth origins of U.S. Chinese are diverse, but descendants from Guangdong province in China are predominant (318). Mortality from lung cancer has increased since 1960 among idai men, partly reflecting the recent influx of Hong Kong Chinese and Hong Kong males of all ages. The mortality rates among both idai and erdai (U.S.-born) Chinese women have exceeded those for U.S. white women since 1960.

The 5-yr survival rates for lung cancer from 1973 to 1979 were low for all races, although the rates were relatively high (15%) among Hawaiians, Chinese, and Japanese, regardless of sex (327). For the period 1960 to 1974, the Hawaii Tumor Registry showed that whites with non-small-cell carcinoma had a significantly poorer 5-yr survival rate than did Chinese, Filipino, or Japanese patients after simultaneous adjustment for differences in sex, age at diagnosis, stage, and socioeconomic status (328). The reasons for the ethnic differences in survival rates are unclear.

Rates of lung cancer in Chinese women from China, Hong Kong, Singapore, and the United States are among the world's highest for female sex (326, 329-332), despite low smoking prevalence (323, 326, 330, 331, 333, 334). The age-adjusted lung cancer incidence rates among Japanese, Chinese, and Hawaiian women in Hawaii during the years 1968 to 1978 did not correlate with their respective cigarette smoking exposures (331). Lung cancer risks associated with smoking were greatest for Hawaiian women (OR = 10.5, 95% CI = 6.1-18.3), less for Japanese women (OR = 4.9, 95% CI = 3.2-7.3), and least for Chinese women (OR = 1.8, 95% CI = 0.69-4.8) after adjusting for age and socioeconomic index. Smoking significantly increased lung cancer risk for all histologies (epidermoid and small-cell carcinoma predominated) among Japanese and Hawaiian women. Among Chinese women, epidermoid and small-cell lung cancers were the only histologic types showing significant association with smoking. However, the lung cancer attributable risks for smoking were 79% for Hawaiian women, 44% for Japanese women, and 11% for Chinese women, suggesting that the majority of lung cancer cases in Japanese and, in particular, Chinese women in Hawaii are caused by factors other than cigarette smoking. Furthermore, the records of 452 lung cancer patients in San Francisco from 1972 to 1979 indicated that, of 31 Chinese women with adenocarcinoma or large cell undifferentiated carcinoma, 64.5% had never smoked (333). Of these 31 women, 90% were born in China and 40% were of Cantonese origin. The factors that influence the interethnic and intraethnic differences in risks for lung cancer (specifically, adenocarcinoma) are speculative. Gao and coworkers (335) reported that smoking was a strong risk factor that accounted for only about 25% of all newly diagnosed cases of lung cancer (60% adenocarcinoma) in women in Shanghai, China. A similar increased risk was observed for squamous and oat cell carcinomas in women who were exposed for more than 40 yr to a husband who smoked. Women who used rape seed oil in cooking had a 2.8 relative risk for lung cancer. An association between exposure to indoor coal burning for cooking and lung cancer in nonsmoking women has



also been reported (237, 326). Women reporting tuberculosis and other lung disease had more than a 70% increase in risk of lung cancer after adjustment for cigarette smoking (335). The risk for adenocarcinoma was 2.5-fold greater among those with tuberculosis infection within the previous two decades compared with those without such a history (336).

### Prediction of Lung Function

Few descriptions of normal lung function are available from large Asian and Pacific Islander populations in the United States. Although a number of studies describe predicted values for lung function in various ethnic groups, the majority of results are limited by such problems as small numbers of subjects in a given ethnic subgroup, restricted age range (especially for the elderly), and variable adjustments for occupation, smoking, and respiratory disease.

Differences in lung function between whites and Asians are well established (337), as are differences among Asian ethnic groups in the same area, adjusted for age and height (194, 338-340), and between the same ethnic subgroups in different countries (341, 342). After adjustment for body size, Asians have been consistently shown to have lung volumes lower than those of whites and flow rates, when corrected for lung volume, similar to those of other racial groups (192, 338, 341-343). People of mixed race usually have intermediate values (183). Ventilatory response to carbon dioxide was lower in a small number of native male Chinese than in whites, although their hypoxic ventilatory responses were similar (344).

Measurements of ventilatory function correlate better with height than with any other parameter of body size. Standing height is the preferred measurement for all races (345). Regression equations derived from white populations, using standing height as the measure of size, usually overestimate predicted values of lung function compared with measured values in other races (192, 338, 343). In one study (346), TLC was 15 to 20% lower in healthy non-smoking Chinese and Asian Indians than in whites at all ages and heights, accounting for lower values for FVC and FEV<sub>1</sub>. However, the FRC, residual volume (RV), peak expiratory flow rate (PEFR), and FEV<sub>1</sub>/FVC% were very similar among some ethnic groups of Asians and Pacific Islanders (343). Values of FVC and FEV<sub>1</sub> are significantly lower in Micronesian men than in whites and Polynesians, and approximately equal to those in Melanesian and Asiatic men (347). Thus, reference values for pulmonary function derived from white populations based on standing height are generally inadequate for Asians, especially for subjects in the older decades (342).

The immigration and settlement of various groups may eventually influence physiologic and other functional aspects in successive generations. Lung function in discrete, homogeneous groups raised in the same environment is more similar than in the same racial groups raised in different environments (341). The anthropometry and lung function in succeeding generations show a gradation from that of the native population to the population of the new homeland, the extent depending on age and environmental influences (342). Both male and female "Oriental" subjects 20 to 79 yr of age in San Francisco are approximately 3 inches shorter than whites and blacks (348). Elderly Japanese and Chinese Americans in San Francisco also weigh less than elderly Caucasians (349). The lack of racial differences in skinfold thickness (a reflection of the amount and distribution of subcutaneous fat) suggests that the Asian elderly have become acculturated into American culture. Larger heights for Japanese adults who were raised as children on "westernized" diets have been reported (341,

342). As nutrition improves, body height and lung volumes increase (350). Thus, succeeding generations of some ethnic groups may attain lung volumes similar to those of Caucasians (350). As a consequence, generation-specific (in addition to race-specific) reference values for lung function may be necessary for evaluating lung function of individual Asians. Further studies are needed in order to decide whether separate regression equations are needed for the best characterization of "westernized" Asians.

### Summary

The patterns of risk factors and respiratory diseases among Asians-Pacific Islanders are complex because of their marked ethnic diversity. Limited data suggest that the overall prevalence of smoking is low compared with that of whites, but it varies widely among the groups. For example, Laotians and Vietnamese have had the highest smoking prevalences of any group in the United States.

The *Report of the Secretary's Task Force on Black and Minority Health* in 1985 stated that "the Asian Pacific Islander minority, in the aggregate, is healthier than all racial/ethnic groups in the United States, including whites" (281). This statement is further supported by recent mortality statistics for Asian-Pacific Islanders (table 2). Overall, mortality from nonsmoking and smoking-related diseases is low compared with that in whites. However, all data are limited by the lack of ethnic-group-specific information. At present, the health status and needs of Asians-Pacific Islanders are probably greatly underestimated. Although the 1990 Census may provide much needed demographic data about the ethnic groups, the health status of Asians and Pacific Islanders in the United States cannot be adequately determined until a comprehensive, national database is established, e.g., an Asian-American HANES (Health and Nutritional Examination Survey).

### AMERICAN INDIANS/ALASKA NATIVES

American Indians and Alaska Natives, collectively referred to in this discussion as American Indians, comprise many culturally and linguistically distinct groups descended from Asian peoples who migrated across the Bering Strait (351). Approximately 1.3 million American Indians reside in the 33 reservation states in which the Indian Health Service (IHS) has a responsibility to provide services. Within the subset of the self-identified Indian population residing in these 33 states is an additional subset of those who belong to "federally recognized" tribes and consequently are eligible for certain services, including health services, provided by the federal government. The establishment of this federally recognized status with its eligibility for services has a long history and is based upon treaties, legislation, and court actions. Finally, of principal interest to the IHS, within this latter subset of "eligible" American Indians are those residing in 505 counties that lie on or near reservations, and are therefore considered to be most nearly identical to those who receive IHS services. This is the population subset usually considered in compilations of IHS mortality, morbidity, and work-load information. This complicated definition of the population of American Indians under consideration is relevant to the examination of sources and calculations of data for vital and health events discussed below.

In the 1990 Census, approximately 2 million people identified their race as American Indian or Alaska Native (table 1). Of these, approximately 55% resided in the following seven states: Oklahoma (12.9%), California (12.4%), Arizona (10.4%), New Mexico (6.9%), Alaska (4.4%), Washington (4.2%), and North Carolina (4.1%) (29). The median age of American Indians-Alaska Natives



was 26.2 yr compared with 34.4 yr for the U.S. white population. Only 5.8% of these American Indians were 65 yr of age or older compared to 13.9% for the U.S. white population (60).

Prior to the Second World War, the great majority of American Indians eligible for federal health services resided on one of the approximately 400 reservations. Since then, however, a great many have migrated to metropolitan areas, and no more than about one half of this American Indian population now resides in the traditional reservation areas (352). Urban members of federally recognized tribes who return to a reservation receive the services that are provided directly by the IHS. However, they are generally not eligible to receive "contract health services," purchased by the IHS from the private sector, until they have established residency for a period of 180 days.

In analyzing health information, the IHS compares rates and trends of various diseases and conditions for American Indians with those for similar groups in the general U.S. population. Examination of these patterns is essential for program planning, and it also provides an opportunity to examine clinical manifestations of disease processes, comparing American Indians and the rest of the U.S. population. The systematic collection of information by the IHS since 1955 has permitted a further important refinement: the elucidation of differences, sometimes of great magnitude, that exist between different Indian groups, permitting further analyses of trends and disease behaviors. The IHS publishes results of these tabulations each year in "Trends in Indian Health" (353) and in "Regional Differences in Indian Health" (49). Two useful summaries have been written by Hill and Spector (354) and Sievers and Fisher (22). This section updates and extends a recent report in the *American Review of Respiratory Disease* (23) on respiratory diseases of American Indians.

#### Socioeconomic Characteristics

Economic conditions of American Indians living on reservations in 1990 tended to be worse than those for the nation as a whole, based upon 1990 employment data from the Bureau of Indian Affairs (355) and the U.S. Census (29). In 1990, 45% of all American Indians 16 to 64 yr of age living on American Indian reservations were unemployed. The highest unemployment rates for reservations with 5,000 or more residents in this age group were in Chey-

enne River (86%), Rosebud (86%), Bethel Alaska Native Village (80%), and Yakima (80%).

In the 1990 U.S. Census, approximately 15% of male American Indians 16 yr of age and older and 13% of female American Indians were unemployed. The unemployment figures for male blacks, Hispanics, and whites were 13.7, 9.8, and 5.3%, respectively, and the corresponding figures for females were 12.2, 11.2, and 5.0%, respectively. The median household income in 1989 for American Indians was \$20,025 (table 4). On the basis of 1989 household income data, approximately 31% of all American Indians lived below the poverty level. Educational attainment for American Indians is less than for whites.

#### Smoking

Most of the available information on the prevalence of smoking among American Indians was obtained prior to the 1980s in selected populations (356-365), and it may not accurately reflect current smoking patterns nor adequately describe the heterogeneity among regions and tribes (360-362, 365). Recent unpublished data from a survey of adult American Indians for the period 1985 to 1988 show that the prevalence of smoking varies widely among the American Indian tribes, and for some tribes it exceeds the overall prevalence of smoking in the United States (Sugarman and coworkers, personal communication). Among American Indians in the southwestern states, 18.1% of men and 14.7% of women reported current smoking, compared with 48.4% of men and 57.3% of women in the plains states. The impression that smoking is much more prevalent in the northern plains than in the Southwest is generally supported by the experience of IHS physicians. Data from the National Medical Expenditure Survey (363) show that the overall prevalence of smoking among American Indians and Alaska Natives is higher than in the general U.S. population (table 8) (359, 360, 363, 366). The patterns of smoking for the different tribes or regions of the country were not provided in the report of this survey. A similar pattern of smoking was found in the Behavioral Risk Factor Surveillance System conducted from 1987 through 1991 (table 8). In addition, American Indians/Alaska Natives smoked fewer cigarettes than do whites. The high overall prevalence of smoking among American Indians found in recent surveys of adults has also been evident among adolescents. Blum

TABLE 8  
SELECTED SURVEYS OF CIGARETTE SMOKING AMONG AMERICAN INDIANS

| Location/Year/Reference No.                                 | American Indian Population  | Findings   |          |                  |     |
|---|---|--|----------|------------------|-----|
|   |   | Male   | NS       | EX               | S   |
| Navajo, 1977 (359)  | 640 Navajo adults   | Smoking prevalence 13%; 94% of smokers used less than 1 pack/day |          |                  |     |
| Minneapolis, 1982 (360)                                     | 242 urban Indians   | 70% current smokers, 45% of smokers exceeded 1 pack/day          |          |                  |     |
| National Medical Expenditure Survey, 1987 (363)             | Total sample includes about 35,000 with approximately 6,500 American Indians/Alaska Natives (AI/AN) living on or near an Indian Reservation | Male   |          |                  |     |
|   |   | AI/AN  | 40%      | 19%              | 38% |
|   |   | U.S.   | 36       | 27               | 30  |
|   |   | Female   |          |                  |     |
|   | AI/AN   | 54   | 15       | 28               |     |
|   | U.S.  | 52   | 18       | 25               |     |
| Behavioral Risk Factor Surveillance System, 1987-1991 (366) | 3,102 AI/AN, 297,438 whites from 47 states and the District of Columbia   | Male   | % smoked | Mean no. of cigs |     |
|   |   | AI/AN  | 33.4     | 19.4             |     |
|   |   | Whites   | 25.7     | 21.4             |     |
|   |   | Female   |          |                  |     |
|   | AI/AN   | 26.6   | 15.5     |                  |     |
|   | Whites  | 23.0   | 17.7     |                  |     |

Definition of abbreviations: NS = nonsmoker, EX = former smoker, S = current smoker; cigs = cigarettes.

and coworkers (364), in a large sample of American Indian adolescent school students, found that for every grade level after the seventh, female students were more likely to be daily cigarette smokers than were male students, with prevalence increasing from 8.9% in junior high school to 17.8% in high school. The increment for male students was 8.1 and 15.0% across the same grades.

### Respiratory Diseases of Childhood

**Hyaline membrane disease.** For the period 1984 to 1988, overall mortality from the respiratory distress syndrome (ICD 769) among American Indians was similar to that among U.S. All Races (table 3) (6). However, mortality rates vary widely between the IHS Service Areas (table 9) (6), ranging from 0.5 per 100,000 in California to 10.1 per 100,000 in Oklahoma. Because of known variations in recording American Indian deaths in California, Oklahoma, and Portland Areas, the validity of data from these areas is suspect.

**Sudden infant death syndrome.** In 1980 the National Infant Mortality Surveillance program collected infant mortality data from six states (367) and found that American Indian infants had a relative risk of 3.5 for SIDS compared with that for white infants. Similar results were obtained for the period 1976 to 1980 among Alaska Native infants whose mortality rate was 2.9 times that of white Alaskans (368). A 2-fold excess mortality from SIDS was reported by Grether and Schulman (83) among American Indians in California from 1978 to 1982. Among these studies, the variability between groups cannot be consistently explained by differences in low-birth-weight babies or in socioeconomic status. However, these mortality data on SIDS must be interpreted cautiously because of possible problems with coding cause of death (369).

**Cystic fibrosis.** Cystic fibrosis is rare in most groups of American Indians (370, 371). In 1990, only 0.2% of patients registered with Cystic Fibrosis Centers were American Indian or Alaska Native (88). During the decade 1981 to 1990, there were 139 hospital discharges for cystic fibrosis from IHS hospitals nationwide, for an average of 14 per year. Although the overall occurrence of cystic fibrosis is rare among American Indians, its frequency among some southwestern pueblo tribes may be similar to that of whites in the United States (372, 373).

### Respiratory Diseases of Childhood and Adulthood

**Upper respiratory infections: otitis media.** Otitis media is the second leading cause of ambulatory clinic visits for American Indians, accounting for 291,000 visits in Fiscal Year 1990, when it was the leading cause of visits for males. Otitis media is more frequent among American Indians than among the U.S. general population

(22). Female American Indians had approximately the same number of visits for otitis media as do male American Indians, but visits for prenatal care, upper respiratory infections, and diabetes each exceeded the number of visits for otitis media (353). Klein (374) has suggested that genetic factors, in addition to low socioeconomic status, may partially explain the increased incidence of otitis media among American Indians, citing differences in the size and angle of the eustachian tube of American Indians as possible contributors to the increased incidence. Beery and coworkers (375) suggested that the lower resistance in the eustachian tube of Apache Indians compared with that in whites might predispose to the more ready entrance of secretions and microorganisms into the middle ear. Reed and Dunn (376) documented the substantial morbidity of otitis media in a study of 345 Alaska Eskimo children. For 41% the onset occurred during the first year of life, and another 18% developed the disease during the second year; hearing loss was common. Wiet (377), in an analysis of outpatient and operative records at the IHS Phoenix Indian Medical Center for the years 1973 to 1978, found a higher incidence of chronic otitis among Athabaskan Indians than among other American Indians in the Southwest, confirming intergroup differences noted earlier by Zonis (378), Jaffe (379), and Hayman and Kester (380).

**Tuberculosis.** Because of its frequency in American Indians, tuberculosis has been a focus for public health research and intervention (381). Public health methods to control tuberculosis through screening of children and isoniazid (INH) chemoprophylaxis, and hospitalization for treatment of active tuberculosis were investigated on the Navajo Reservation in the 1950s (382, 383). Clinical trials to investigate the efficacy of INH chemoprophylaxis were conducted among Alaskan Natives (384, 385).

Although the incidence of tuberculosis among American Indians has been declining, it remains disproportionately high compared with that among whites. In 1985, 2% of the 22,201 cases of tuberculosis reported to the Centers for Disease Control were from American Indians; the incidence rate of 25.0 per 100,000 population was more than four times that of U.S. whites (386). Of the incident cases of tuberculosis among American Indians, 97% were reported from states with reservations, with 11 states accounting for 82% of the reported cases. The highest incidence was in the Aberdeen Area of South Dakota, where the rate was 30.4 times that for all other races. The tribes in South Dakota accounted for 62% of the reported cases while constituting only 7% of the state's population (386). In Alaska, Alaska Natives accounted for 71% of reported cases but only 14% of the population.

Tuberculosis was the leading cause of death among Ameri-

TABLE 9  
MORTALITY RATES\* FOR SELECTED RESPIRATORY CAUSES BY IHS SERVICE AREA,  
AMERICAN INDIANS AND ALASKA NATIVES, 1984-1988†

| Cause of Death<br>(ICD-9-CM Code)                            | IHS Service Area |          |        |       |         |          |                 |                |        |               |         |          |        |
|--|------------------|----------|--------|-------|---------|----------|-----------------|----------------|--------|---------------|---------|----------|--------|
|  | Total            | Aberdeen | Alaska | Albuq | Bemidji | Billings | Calif-<br>ornia | Nash-<br>ville | Navajo | Okla-<br>homa | Phoenix | Portland | Tucson |
| Tuberculosis, respiratory system<br>(010-012)                | 1.5              | 2.9      | 1.8    | 0.4   | 0.8     | 2.3      | 1.1             | 0.5            | 2.9    | 0.7           | 1.6     | 0.8      | 2.1    |
| Malignant neoplasm of<br>bronchus and lung (162.2-162.9)     | 17.4             | 27.6     | 37.7   | 6.3   | 26.0    | 31.8     | 10.8            | 21.3           | 3.6    | 22.6          | 7.3     | 16.8     | 9.6    |
| Pneumonia, all types (480-486)                               | 19.9             | 29.2     | 22.7   | 17.1  | 23.2    | 30.9     | 9.8             | 16.4           | 20.8   | 17.2          | 24.3    | 12.5     | 33.1   |
| Emphysema (492)  | 1.3              | 2.4      | 0.8    | 0.4   | 3.2     | 3.2      | 1.6             | 1.6            | —      | 1.5           | 0.9     | 2.0      | —      |
| Asthma (493)   | 1.0              | 1.9      | 1.6    | 0.4   | 2.0     | 0.9      | —               | —              | 0.4    | 1.4           | 0.7     | 0.6      | 3.2    |
| Chronic airway obstruction not<br>elsewhere classified (496) | 7.4              | 12.7     | 10.5   | 4.1   | 11.6    | 11.3     | 6.8             | 10.9           | 2.6    | 6.2           | 6.4     | 9.1      | 8.6    |
| Respiratory distress syndrome (769)                          | 1.9              | 4.3      | 4.0    | 2.2   | 2.4     | 2.3      | 0.5             | 0.5            | 1.1    | 10.1          | 1.4     | 2.8      | 3.2    |

\* Mortality rates per 100,000 population.

† Sources: Indian Health Service (6).

TABLE 10  
TRENDS IN AGE-ADJUSTED MORTALITY RATES\* FOR  
TUBERCULOSIS, PNEUMONIA, AND INFLUENZA AMONG  
AMERICAN INDIANS AND NON-HISPANIC WHITES  
IN NEW MEXICO, 1958-87†

|                     | 1958-1962 | 1983-1987 | Change in rates<br>from 1958 to 1987<br>(%) |
|---------------------|-----------|-----------|---|
| <b>Tuberculosis</b> |           |           |   |
| <b>Males</b>        |           |           |   |
| American Indian     | 40.5      | 11.7      | -71.1                                       |
| Non-Hispanic white  | 9.4       | 1.1       | -88.3                                       |
| <b>Females</b>      |           |           |   |
| American Indian     | 38.0      | 10.2      | -73.2                                       |
| Non-Hispanic white  | 3.7       | 0.6       | -83.8                                       |
| <b>Pneumonia</b>    |           |           |   |
| <b>Males</b>        |           |           |   |
| American Indian     | 88.5      | 51.6      | -41.7                                       |
| Non-Hispanic white  | 34.8      | 34.0      | -2.3  |
| <b>Females</b>      |           |           |   |
| American Indian     | 65.8      | 28.0      | -57.4                                       |
| Non-Hispanic white  | 19.2      | 21.3      | +10.9                                       |
| <b>Influenza</b>    |           |           |   |
| <b>Males</b>        |           |           |   |
| American Indian     | 8.1       | 0.0       | -100.0                                      |
| Non-Hispanic white  | 2.9       | 0.5       | -82.3                                       |
| <b>Females</b>      |           |           |   |
| American Indian     | 4.9       | 0.8       | -85.4                                       |
| Non-Hispanic white  | 2.8       | 0.6       | -78.6                                       |

\* Age-adjusted rates per 100,000.

† Source: Becker et al. (243).

can Indians for many years, and despite reductions in mortality, it remains a common cause of death compared with that in whites (tables 3, 9, and 10) (242, 243, 381, 386, 387). In New Mexico from 1958 to 1987, mortality rates from tuberculosis among American Indians declined approximately 70%, but even so, during the period 1983 to 1987, the rates among men and women were 10.6 and 17.0 times, respectively, the rates among white men and women in the state. High mortality rates from tuberculosis have also been reported for American Indians residing in New York (388) and Arizona (389). Possible explanations for the increased mortality include the high incidence of alcoholism (353) and diabetes (22, 353, 354, 390), and the generally lower socioeconomic conditions prevailing in American Indian communities.

**Pneumonia and influenza.** For many years a high incidence of severe lower respiratory tract infections in American Indian children of the United States and Canada has been recognized by clinicians (391-393). In 1961, a study of Alaskan Natives from four western Alaskan villages showed that 35% had a lower respiratory infection by 1 yr of age, and 15% had a second lower respiratory tract infection by 10 yr of age (391). Houston and coworkers (393) analyzed clinical information from 166 children hospitalized with pneumonia at the University Hospital, Saskatoon, from 1974 to 1976. Overall, the Canadian Indian children had a more severe clinical course than did the white children; chest roentgenograms worsened during the hospitalization in 19.6% of the Indian children and in only 1.6% of the white children. On average, the Indian children's hospital stay was 26.9 days compared with 8.7 days for the white children; 3.9% of the Indian children and none of the white children died.

Pneumonia was the leading cause of respiratory deaths for the period 1984 to 1988 among American Indians (table 3). A similar mortality pattern for pneumonia was found in New Mexico (table 10) (243). Although there was a sharp decline in mortality from

pneumonia among American Indians in New Mexico from 1958 to 1987, mortality from pneumonia remained 31 to 52% higher in American Indian women and men, respectively, than in whites during the years 1983 to 1987. Factors leading to the difference in mortality rates for pneumonia between the American Indian population and the U.S. All Races population have not been explored.

Although the mortality rate from pneumococcal pneumonia has decreased in recent years (unpublished data from IHS 1991), it remains twice that for U.S. All Races, and it is a particular problem among Alaska Natives and American Indians cared for at the IHS Phoenix Indian Medical Center (table 9). Davidson and coworkers (394) reported an incidence rate of invasive pneumococcal disease in Alaska Natives for the period 1986 to 1988 of 74 per 100,000 population, compared with 12 per 100,000 for all other racial groups in Alaska, primarily whites, and a 6-fold increase for children younger than 2 yr of age. Of isolates serotyped, 92% were identical to the serotypes contained in the available pneumococcal vaccine. However, of 114 Alaska Native patients whose records were reviewed, only eight had prediagnostic pneumococcal vaccine. In addition, intermediate levels of penicillin resistance were found in 3.7% of isolates from Alaska Natives, most of them from a single remote location. Similar results concerning invasive pneumococcal disease among Alaska Natives were reported by the same investigators for the period 1980 to 1986 (395). Differences in the distributions of blood types, in particular a low frequency of blood group B among American Indians (396), may also contribute to more severe disease among American Indians than among whites.

Oseasohn and coworkers (397) examined the medical records of all Navajos in one community who presented with clinical findings of pneumonia from November 1971 through October 1973. Most episodes were associated with pneumococci. The 2-yr fatality rate was 2.2%, and all deaths occurred among patients with underlying diseases. The investigators confirmed the greater susceptibility to pneumonia of individuals at both extremes of age and also demonstrated that most of the pneumococcus isolates were of vaccine serotypes. Intermediate levels of penicillin resistance of pneumococci among Navajos (397) were also reported by Tempest and coworkers (398), who found that 14% of pneumococci encountered at the Gallup Indian Medical Center in New Mexico were relatively resistant to penicillin (requiring 0.1 µg/ml or more for inhibition).

The highest incidence for invasive pneumococcal disease has been reported among members of the White Mountain Apache tribe of Eastern Arizona (399). During the period 1983 through 1990, Cortese and coworkers (399) identified 138 cases of invasive pneumococcal disease. Among children younger than 5 yr of age, 39.6% of cases were pneumonia, 29.2% were fever without a source, and 28.1% were otitis media. For children and adults 5 yr of age and older, 78.6% of cases were pneumonia and 9.5% were fever without a source. The overall annual incidence of invasive pneumococcal disease was 207 per 100,000.

According to Gellin and coworkers (400), the incidence of invasive *Hemophilus influenzae* infection in Alaska Native children younger than 5 yr of age is nearly five times that in non-native Alaskans and six times that in the general U.S. population of the same age. Among all Alaska residents 10 yr of age or older from whom *Hemophilus influenzae* was isolated from a normally sterile site from 1980 to 1988, there were 72 cases with 11 fatalities; 23 cases in Alaska Natives (two deaths) and 47 in Alaska whites (eight deaths). Age-adjusted annual incidence rates were 5.4 per 100,000 for Alaska Natives and 2.4 per 100,000 for Alaska whites. Other

American Indian groups have also been found to have a high incidence of *Hemophilus* infections (401–403). Recent developments in immunizations hold considerable promise for decreasing mortality from *Hemophilus* infections (404). However, antibody response to *Hemophilus influenzae* polysaccharide vaccine may be impaired in some American Indian children when compared with white children (405). *Hemophilus influenzae* immune globulin is of particular interest for use in the very young child who might have suboptimal antibody responses to vaccine (406, 407).

Although the major consequences from the high rates of lower respiratory tract infections among American Indians are acute morbidity and mortality, chronic sequelae may result. From 1956 to 1966, Fleshman and coworkers (408) found 100 cases of bronchiectasis among Alaska Natives 16 yr of age or younger; in 68% nontuberculous bronchiectasis was diagnosed, secondary to severe lower respiratory tract infection occurring in infancy or childhood.

**Other pulmonary infections.** As discussed in the section on blacks, increased risk for dissemination of coccidioidomycosis has been found among some minority populations. During the period 1959 to 1972, Sievers (409) found that morbidity and mortality from disseminated coccidioidomycosis were three to five times higher among American Indians living in southern Arizona, an endemic area, than in whites living in the same area. However, in a follow-up study, Sievers and Fisher (410) found that the annual rate of disseminated coccidioidomycosis among Arizona Indians had dropped from 8.9 per 100,000 during the period 1959 to 1969 to 3.9 per 100,000 from 1970 to 1989. The mortality rate from disseminated coccidioidomycosis among the Indian population in 1970 to 1980 was less than the rate among whites living in the same counties, 0.5 per 100,000 and 1.1 per 100,000, respectively. The decline in morbidity and mortality from coccidioidomycosis among these American Indian populations suggests that environmental rather than genetic factors contributed to the earlier excess.

**Asthma.** Early reports, admittedly of small sample size, suggested that asthma was rare among American Indians (411, 412). In New Mexico, from 1969 to 1977, there was only one death from asthma among American Indians (242). However, recent nationwide mortality rates for asthma are similar among American Indians and U.S. All Races (table 3). The similarity of mortality rates for American Indians and U.S. All Races suggests either that the prevalence of asthma among American Indians is comparable to U.S. All Races or that the disease is more severe among American Indians. In addition, differences in definition and diagnosis of asthma might account for some of the differences in rates between American Indians and U.S. All Races.

**COPD.** Little information is available on the occurrence of COPD among American Indians. In 1987, a nationwide sample of approximately 6,500 American Indians and Alaska Natives 19 years of age and older was surveyed about various chronic conditions (413). On the basis of this survey, estimates of the prevalence of "emphysema" were 2.4% for American Indian men and 1.4% for women. The corresponding estimates for men and women in the general U.S. population were 2.7 and 2.3%, respectively.

For the period 1984 to 1988, the mortality rate among American Indians from COPD was approximately one-third the rate for U.S. All Races (table 3). Differences in cigarette smoking appeared to account for much, but not all, of this difference. However, the differences in mortality rates between different American Indian groups (table 9) may be largely accounted for by different rates of smoking. The COPD mortality rate among American Indians in the Southwest, where smoking rates are low, is well below the rates for the American Indians of the great plains and for Alaska

TABLE 11

TRENDS IN AGE-ADJUSTED MORTALITY RATES\* FOR COPD AND LUNG CANCER AMONG AMERICAN INDIANS AND NON-HISPANIC WHITES IN NEW MEXICO, 1958–1982†

|                    | 1958–1962 | 1978–1982 | % Change in rates from 1958 to 1982 (%) |
|--------------------|-----------|-----------|---|
| <b>COPD</b>        |           |           |   |
| <b>Males</b>       |           |           |   |
| American Indian    | 1.4       | 12.2      | + 771.4                                 |
| Non-Hispanic white | 17.0      | 64.5      | + 279.4                                 |
| <b>Females</b>     |           |           |   |
| American Indian    | 1.7       | 0.8       | - 52.9                                  |
| Non-Hispanic white | 1.6       | 22.9      | + 1331.3                                |
| <b>Lung cancer</b> |           |           |   |
| <b>Males</b>       |           |           |   |
| American Indian    | 5.3       | 10.8      | + 103.8                                 |
| Non-Hispanic white | 30.1      | 62.9      | + 109.0                                 |
| <b>Females</b>     |           |           |   |
| American Indian    | 1.6       | 4.2       | + 162.5                                 |
| Non-Hispanic white | 4.5       | 19.9      | + 342.2                                 |

\* Age-adjusted rates per 100,000.

† Source: Samet *et al.* (250).

Natives. Unpublished data from the Alaska Area indicate that the COPD mortality rate from 1979 to 1986 was 31.6 per 100,000 for male Alaska Natives compared with 40.3 per 100,000 for male Alaska whites and 38.3 per 100,000 for U.S. All Races. The mortality rate for female Alaska Natives was 22.3 per 100,000 compared with 34.8 per 100,000 for female Alaska whites and 18.6 per 100,000 for U.S. All Races. Mortality rates for COPD in New Mexico (table 11) (250) reflect the nationwide pattern of low mortality among American Indians compared with that among whites. The increment in COPD mortality among men, comparing the periods 1958 to 1962 with 1978 to 1982, should be interpreted cautiously because of the small numbers of deaths.

**Silicosis.** Because of mining operations on or near American Indian reservations, mining has been a common occupation for some American Indian tribes. Southwestern American Indians, particularly the Navajo, have been a major component of the work force in the uranium mining industry. As a result, silicosis has been a prevalent condition in Navajo men (414). Among 452 American Indian miners in New Mexico who volunteered for screening for mining-related health problems, approximately 8 to 26% had chest roentgenogram findings consistent with silicosis, small rounded opacities with a profusion of 1/0 or greater. The lowest prevalence occurred among miners with 10 yr or fewer of underground mining experience, and the highest prevalence occurred among miners with more than 20 yr of underground mining. Prevalence varied slightly with the type of mining performed, either uranium or other types of mining.

**Lung cancer.** The low overall occurrence of cancer in American Indians compared with that in U.S. whites has been documented in a variety of studies since the early 1900s (242, 250, 415–430) (table 12). In 1926, Hoffman (416) conducted a survey of physicians who provided health care to approximately 100,000 American Indians to assess life insurance risks among American Indians. He identified only 276 cancer deaths in total, and no lung cancer deaths. Subsequent investigations of lung cancer incidence and mortality have confirmed that lung cancer is infrequent among American Indians compared with that among whites (431) (table 12). Among Alaska Natives, the occurrence of lung cancer approaches that for whites, although small numbers limit interpre-

TABLE 12  
LUNG CANCER IN AMERICAN INDIANS (AI) AND ALASKA NATIVES (AN)

| Year/Reference No. | Population   |         | Findings                                      |      |        |
|--------------------|--|---------|---|------|--------|
|                    |  |         | n   | SMR  |        |
| 1949-52, (418)     | 428,225 AI, US white rates used to calculate SMRs                                    | Males   | 34  | 28   |        |
|                    |  | Females | 19  | 85   |        |
| 1950-57, (423)     | 5,897 deaths among AI in the U.S., white rates used to calculate SMRs                | Males   | 127   | 31   |        |
|                    |  | Females | 37  | 66   |        |
| 1960-69, (425)     | AN population, approximately 43,000 in 1960, U.S. white rates used to calculate SMRs | Males   | 28  | 58   |        |
|                    |  | Females | 7   | 90   |        |
| 1967-77, (242)     | 80,225 AI in New Mexico  |         | Annual Incidence of Lung Cancer (per 100,000) |      |        |
|                    |  |         | AI  | H    | Non-HW |
|                    |  | Males   | 10.5  | 25.5 | 75.9   |
|                    |  | Females | 5.0   | 15.7 | 20.8   |

Definition of abbreviations: SMR = standardized mortality ratio (observed mortality rate/expected mortality rate); H = Hispanic; Non-HW = non-Hispanic white.

tation of the patterns of occurrence (table 12). Among the various American Indian groups, mortality rates for cancer of the lung vary widely (250, 420, 432) (table 9). For example, the mortality rate for bronchogenic carcinoma among American Indians of the Southwest is approximately 3 to 6 per 100,000, in contrast to rates in the north central states, which are in the range of 23 to 26 per 100,000 (23). These differences are undoubtedly associated with much higher rates of smoking in the northern regions than in the Southwest (357). Schaefer and coworkers (433) attributed the rise in lung cancer in Canadian Eskimos to an increase in smoking. However, even in the northern regions of the United States, where the rates of smoking among American Indians exceed those of the rest of the population, the rates of deaths from bronchogenic carcinoma are only slightly more than one-half the rate for U.S. All Races.

Although mortality from lung cancer is still uncommon among American Indians (431), the rate may be increasing. From 1958 to 1982, mortality rates for lung cancer in New Mexico increased 104% among male American Indians and 163% among female American Indians (table 11) (250). Changes in smoking patterns may partly explain these increases in lung cancer, but a substantial proportion of the increment in men in New Mexico can be attributed to exposure to radon progeny in uranium mines (361).

The association between uranium mining and cancer of the lung in American Indians was initially described by Archer and coworkers (356) and later confirmed by Gottlieb and Husen (434). Archer and colleagues (356) followed the mortality of 780 American Indian participants, primarily Navajos, in the U.S. Public Health Service Study of Colorado Plateau miners. Eleven lung cancer deaths were observed, with an expected number of 2.6 based on mortality rates for male non-whites of Arizona and New Mexico. Gottlieb and Husen (434) identified 17 patients with lung cancer among Navajo men admitted to the Shiprock Indian Health Service hospital between February 1965 and May 1979, of whom 16 had worked as uranium miners. Samet and coworkers (361) also confirmed the association of uranium mining with lung cancer in Navajos and its importance in this group of people with a very low frequency of cigarette smoking. Of 32 Navajo men with lung

cancer diagnosed between 1969 and 1982, 26 (72%) had been uranium miners, and most were nonsmokers or light smokers. The histologic types of lung cancer found in these miners were similar to the distribution seen in lung cancer cases from the general population (435).

The problem of occupational lung diseases, including lung cancer and silicosis, among uranium miners and in particular among American Indian miners prompted Congress to pass the Radiation Exposure Compensation Act of 1990 (436). The act authorizes compensation to individuals exposed to radiation from employment in an underground uranium mine who develop lung cancer or a nonmalignant respiratory disease, including pulmonary fibrosis, silicosis, or pneumoconiosis.

**Other thoracic cancers.** Mesothelioma, a rare malignancy of the pleura, is strongly associated with exposure to asbestos. Driscoll and coworkers (437) reported on five patients from the same pueblo in whom malignant mesothelioma had been diagnosed between 1970 and 1985. The investigators surveyed family, friends, and one surviving patient to determine the patients' exposures, and found that materials used in silver smithing and for whitening ceremonial leggings and pottery contained asbestos.

#### Prediction of Lung Function

Few studies have been conducted to define normal lung function among American Indians. Gould (180) compared lung function of American Indians, most of whom were from New York, with that of blacks and whites during the Civil War. He reported that the mean thoracic circumference of American Indians exceeded that of whites but with less "play" of the thorax. The average vital capacity of these American Indians was 184.98 cubic inches, compared with 184.69 cubic inches for whites and 163.45 cubic inches for blacks. Beaudry (438) found that Canadian Eskimos had decreased forced expiratory volume and decreased midmaximal flow rates when compared with whites. In contrast, Rode and Shephard (439) found that the forced expiratory volume and the FEV<sub>1</sub> of Canadian Eskimos exceeded the predicted values for whites, with no difference in diffusing capacity in the two groups. Lanese and coworkers (440) found in a small sample of textile mill workers

that the values for lung function of American Indians were between those of blacks and whites. Crapo and coworkers (441) found slight differences between whites and Navajos in measurements of FVC and FEV<sub>1</sub>.

Wall and coworkers (442) performed pulmonary function tests on 94 females and 82 males 7 to 18 yr of age from the Warm Springs Indian Reservation in Oregon; they found that lung volumes and expiratory flow rates in American Indian children were similar to those previously reported for white and Mexican-American children but greater than those for blacks. The investigators caution against assuming that these values are representative of all American Indians of similar age.

### Summary

American Indians, in comparison to the general U.S. population, have increased rates of infectious lung diseases and lower rates of smoking-related diseases. Invasive pneumococcal, *Hemophilus*, and streptococcal infections and tuberculosis are among the most prominent conditions in which there is increased susceptibility. Reasons for increased rates of these infectious lung diseases are not completely known, and they must be attributed in many instances to the high level of risk factors among American Indians, including alcoholism, low socioeconomic status, smoking, and diabetes mellitus.

Limited data suggest that the prevalence of smoking is high among tribes of the plains and northern states and low among American Indians of the Southwest. Although the influence of smoking on lung cancer and COPD is clearly evident in the differences that exist between American Indian groups, the differences between American Indians and the general U.S. population do not appear to be completely explained on this basis. The mortality rates for cancer and COPD appear to be less than those for the general U.S. population, even in American Indians who are heavy smokers.

### IMPLICATIONS FOR PATIENT CARE

This review of disease occurrence and risk factor patterns among different populations of the United States shows many examples of strong differences between the white majority and the minority groups. Clinicians should be aware of these differences as they indicate prior probabilities of diseases among the different population groups. For example, a lung mass that would almost certainly be a lung cancer in a white, male smoker is far less likely to be a lung cancer in a never-smoking male Navajo who has never been a uranium miner. Clinicians should be cognizant of the patterns of diseases and risk factors in their locale, and of the heterogeneity within the groups in their practice; they also need to understand and be sensitive to the social and cultural backgrounds of their patients. Other relevant issues for the pulmonary physician who provides care to minority patients include the prediction of normal lung function, screening for the treatment of tuberculosis, and control of cigarette smoking.

#### Country of Origin

The country of origin and duration of residence in the United States may directly affect a patient's probability of having an endemic infectious disease (443). Information on the endemic diseases worldwide is summarized in the publication by the Centers for Disease Control, *Health Information for International Travel* (444). The country of origin may also determine the pattern of drug resistance of various organisms. For example, *M. tuberculosis* among Hispanic immigrants and persons from southeast Asia is often resistant to conventional drug treatments for tuberculosis (445).

In addition to infectious diseases, exposures to occupational agents and air pollutants in the country of origin may affect risk for chronic respiratory diseases. For example, chronic bronchitis in nonsmokers is more common in Third-World countries than in the developed countries, possibly because of intense exposures to domestic smoke in houses without chimneys (280, 346) or from other forms of air pollution (346, 446).

#### Culture

Communication and understanding between physician and patient, always important if medical management is to be optimal, present a special challenge when physician and patient have different cultural backgrounds (3, 9, 36, 447-449). This discussion of issues that are clinically relevant to respiratory diseases in minorities would be incomplete without a section on relevant cultural characteristics and their potential impact on the clinical encounter. The following paragraphs discuss briefly the cultural aspects of greatest potential impact: language use and knowledge, attitudes, and behaviors related to health and disease. For more detailed descriptions of culture and health and illness, we refer readers to the books written by Harwood (9) and Helman (3).

**Language.** Barriers to health care for many monolingual minority populations in the United States can be overwhelming. In general, most of the health care system in the United States is organized around the assumption of literacy in English (291); and many health facilities do not have bilingual or bicultural professionals on the staff. Minorities may well be deterred from seeking care at such facilities when faced with language and communication barriers (450). When monolingual patients do access the health care system, language differences between the doctors and the patients may have an adverse effect upon the quality of information exchanged during their clinical encounter. The consequences of even a slight misunderstanding can be far-reaching, from an inaccurate diagnosis to failure to follow prescribed treatment. In a study of 96 patients with asthma cared for at a New York City hospital, Manson (451) found that differences in language use between doctors and patients were associated with medication noncompliance (OR = 1.72, 95% CI = 0.69-4.30) and missed office appointments (OR = 1.66, 95% CI = 0.86-3.20).

Although the use of an interpreter may facilitate communication between a doctor and patient, the physician must continue to establish and maintain a rapport with the patient, even while working within the constraints of communicating through a translator, the translator's skills and background, and the translation itself (452). The multitude of languages and dialects, even within a single minority group, may prove challenging for the patient, the health care provider, and the interpreter.

Dialects within the English language can also have adverse consequences on health care delivery. Dialects evolve when groups of people are separated by social class or ethnicity, and the nature and frequency of communication between the groups changes, resulting in the development of different linguistic norms (453). These differences in linguistic norms have been shown to correlate with levels of social stratification in our society (454) and to adversely affect communication between patients from a low socioeconomic background and physicians (455).

Problems in communication between speakers of different languages or dialects can arise not only from the language itself but also from differences in the social norms of communication (453). For example, the choice of vocabulary must fit the social situation and not violate the rules of etiquette; as in knowing when to address a person by his or her title, or last name, or by first name only. Black Americans have frequently said they resent the use of their first names by health care providers (453).



**Health beliefs and behaviors.** Health beliefs and behaviors are tightly linked to race and ethnicity, and their influence is pervasive, affecting risk factor profiles, behavior in seeking health care, and adherence to medical advice (9, 448). For example, awareness of the health effects of cigarette smoking is variable among minority groups (68, 456–458). In a survey of 2,092 adults from St. Louis, Brownson and coworkers (457) found that blacks were less likely than whites to believe that cigarette smoking was a cause of lung cancer and emphysema, and less likely to acknowledge the health benefits of quitting, in a ratio of 82 to 89% of whites.

In a survey of knowledge and attitudes about cancer among 844 Latinos and 510 Anglos who were current members of Kaiser Permanente in San Francisco, Perez-Stable and colleagues (459) found marked differences between the groups. For example, Latinos were less likely to think environmental pollution causes cancer than were Anglos, 83 and 90%, respectively. Furthermore, a higher proportion of Latinos (26%) than Anglos (18%) believed there was little that could be done to prevent cancer.

Of particular relevance to the pulmonologist/critical care specialist is the role of culture in making life-support decisions (460, 461). Although patient autonomy in medical decisions and truth telling about prognosis have become the norm in the United States (461), these standards may not be acceptable to persons from all cultural backgrounds, and they may even be the cause of conflict between the physician and a patient or the patient's family in making life-support decisions. Klessig (460) surveyed 230 people from the Los Angeles area about decisions they would make in response to eight different clinical scenarios concerning the withdrawal of life-support. Response patterns on the acceptability of withdrawing life-support varied widely among the racial and ethnic groups.

General guidelines for improving understanding between patients and physicians from different cultural backgrounds are available (9, 462). In addition, several reviews are available on health beliefs and behaviors for specific groups such as blacks (9, 463), Hispanics (9, 464, 465), Indochinese (9, 18), and Navajos (9).

### Pharmacologic Considerations

**Drug metabolism.** A particular metabolic phenotype may have implications for drug dosage and the occurrence of adverse drug reactions, and possibly for risks for some diseases. For example, some pharmacokinetic differences may be related to phenotypes for hepatic cytochrome P-450 isozymes, which may be measured by oxidation of debrisoquine or sparteine. The ability to oxidize debrisoquine and sparteine is poor in only 0.7% of the native Chinese population but in 8 to 10% of the American population (466). Because debrisoquine metabolism is associated with lung cancer (467), interracial differences in metabolism may contribute to differences in the occurrence of lung cancer. Interethnic or interracial differences in metabolism of other compounds have been reported for only a small number of drugs (466, 468); differences in the metabolism of methylxanthines have not been consistently demonstrated between ethnic groups (469, 470).

The metabolism of isoniazid (INH) is influenced by genetic and environmental factors. Hepatic acetylation is the primary metabolic route determining the rate at which INH is eliminated from the body. Drug acetylation is an autosomal dominant trait, with individuals being either slow, heterozygous rapid, or homozygous rapid acetylators (471). The proportions of rapid and slow acetylators vary among populations (472, 473), and, in general, rapid acetylators are most common among persons of Asiatic origin (70% or greater), including American Indians and Alaska Eskimos (471, 473); intermediate for persons from Latin America (67%); and lowest among American blacks (49 to 58%) and whites (43 to 48%).

The acetylator status of patients with tuberculosis does not have

prognostic significance if INH-containing treatments are administered daily (473). However, it may adversely affect therapeutic response in twice-weekly regimens, especially when the initial daily chemotherapy is given for only a short period and the companion drug during the continuation phase is relatively weak. The adverse effect is even greater in once-weekly regimens, in which rapid acetylators fare considerably worse than do slow acetylators. Therapeutic failures in rapid acetylators occur primarily when INH, in combination with other antituberculosis drugs, is administered to patients with open-cavity tuberculosis (471). However, it is doubtful whether the acetylator phenotype alone makes a significant difference in treatment outcome in most standard INH-containing treatment regimens (473).

Adverse side effects from INH may vary in frequency by acetylator status (473). Studies of INH-induced hepatitis and rapid acetylation have given conflicting results (473). However, acetylator phenotype is probably not an important risk factor for INH-induced hepatitis (473). Peripheral neuropathy from INH is more common among slow acetylators, but it can be readily prevented by pyridoxine.

**Other pharmacologic considerations.** Hereditary abnormalities of red blood cells, including glucose-6-phosphate dehydrogenase deficiency (G6PDD) and hemoglobin E are more common in minority populations of the United States than in whites (474–476). In a survey of 3,159 healthy U.S. subjects, Frischer and coworkers (474) found G6PDD in 10.6% of blacks, 4.1% of Vietnamese, 9.8% of Iranians, and none among European-Americans. The prevalence of G6PDD varies widely among Southeast Asian refugees (475), ranging from 13% among Lowland Laotian (Lao Loum) to 29% among Cambodians (Khmer). Although G6PDD does not cause respiratory disease, it can result in hemolytic anemia with use of sulfonamide-containing antibiotics, which are frequently used to treat respiratory tract infections (477). Hemoglobin E is a benign beta-chain variant that occurs almost exclusively in persons of Asian descent, particularly Southeast Asians (476). Neither the homozygous nor heterozygous states of Hb E are associated with significant clinical illness, except when paired with a beta-thalassemia gene (475, 478).

### Physiologic Considerations

Lung function differences between the various racial and ethnic groups may have direct relevance for the pulmonologist during the diagnostic process and in making management decisions. The determinants of racial differences in lung function are not fully understood. Differences among races cannot be removed by adjustment for a variety of factors, including age, stature, smoking, air pollution, physical activity, and altitude (479). Genetic factors may lead to airways of different sizes or to different elastic recoil during lung growth and maturation (279, 480). Differences in lung volumes begin in childhood (343) and may be due in part to differences in body build or height. Differences in ventilatory lung function are related to differences in lung size at full inspiration (194). The lack of consistent differences in FEV<sub>1</sub>/FVC% and PEFr between racial groups suggests that airway caliber is similar among them and that the larger TLC in Caucasians may result from larger airway or alveolar size or increased inspiratory muscle pressure (343, 350). The size or shape of the chest appears to be another determinant of measurements at TLC (194, 350).

Other predictors of lung function have also been evaluated. The relative sitting height (RST = sitting height × 100/standing height) is the proportion of trunk size to standing height. The RST is characteristic for an ethnic group and a major determinant of ethnic differences in lung volumes (481). However, the use of sitting height as an index of body size in prediction equations reduces

but does not fully eliminate the observed differences between races (183). Biacromial diameter, skinfold thickness, and dynamometry are also statistically significant independent predictors of lung function, after controlling for age and height (192).

The use of racial adjustments in predicting lung function is variable and arbitrary, and the correction factor for adjusting white predicted values for other groups is inadequately defined. Responses from 95 North American respiratory training programs have indicated that 53% did not apply ethnic adjustments to predict lung function (481). Of the 45 programs that used corrections, five programs used population-specific equations and 29 reduced the predicted values for whites by 10 to 15%. Ethnic adjustments varied according to region, with Asians having adjustments only in California and Asian Indians only in New York. There is no clear consensus on whether ethnic adjustments are necessary for predicting lung function, which ethnic groups require adjustment, and how adjustment should be approached such as using either proportional differences or population-specific equations. Corey and coworkers (482) compared spirometric prediction equations for Caucasian and East Indian male sawmill workers and found that Caucasian predicted values for FEV<sub>1</sub> could reliably predict East Indian values over a wide range of heights by multiplying the Caucasian values by 0.8. However, the relationships between the predicted values for FVC and MMF were less consistent, suggesting that use of a single scaling factor may be inadequate for predicting normal spirometry values among different ethnic groups. For each minority population, we have reviewed physiologic differences in pulmonary function, and in the next section we provide guidelines for the interpretation of pulmonary function tests.

*Interpretation of pulmonary function.* The problem of selecting the most appropriate reference population for predicting lung function values has long been recognized (182, 183, 483). The genetic and environmental determinants of lung function may differ between the people living in a particular community and the people from whom spirometric prediction equations were derived. Although many studies have examined the effect of race and ethnicity on lung function level, the consequences of failing to use racial- or ethnic-specific prediction equations for clinical purposes have received little attention.

Differences among published prediction equations for whites have been described (183, 484, 485). Lebowitz and Holberg (486) recently compared the performance of 13 different prediction equations in participants in the Tucson epidemiologic study. This analysis showed substantial variation in the proportion of the population classified as abnormal depending upon the choice of the regression equation and the method used to determine abnormality. Harber and coworkers (487) compared three regression equations in a population of 900 undergoing testing for respiratory disability. The choice of prediction equation changed the percentage of subjects with FEV<sub>1</sub> less than 60% of predicted by only a few percentage points.

Shaffer and coworkers (488) applied prediction equations for the FVC and FEV<sub>1</sub> derived from New Mexico Hispanics and prediction equations derived from non-Hispanic whites to 442 Hispanic patients evaluated at the University of New Mexico Hospital. The classification of the FVC and FEV<sub>1</sub> as normal or abnormal was influenced by the regression equation in approximately 5 to 10% of the subjects; most discrepancies occurred with classification of subjects as normal by the Hispanic equations and abnormal by the external equations derived from non-Hispanic white populations.

The findings from these investigations suggest that the choice

of reference equation may have clinically important implications. This problem could be addressed by developing regression equations for specific ethnic or racial groups in particular areas. However, in many communities the needed technical resources are lacking and inability to access "normal" subjects may pose feasibility constraints. When the development of ethnic- and community-specific regression equations is not feasible, the use of a "scaling" factor may be acceptable to adjust for ethnic and racial differences (183). However, a "scaling" factor is currently available only for blacks, and, therefore, interpretation of pulmonary function tests, particularly those obtained from other minority patients, will require sound clinical judgment to avoid potential errors from rigid interpretation of numerical values.

## IMPLICATIONS FOR PREVENTION

Much of the morbidity and mortality associated with the diseases discussed in this review could be prevented through smoking prevention and cessation, screening and chemoprophylaxis for tuberculosis, and immunization for influenza and pneumococcal pneumonia (489). However, several barriers, especially among minority populations, may limit the effectiveness of these preventive interventions. For example, the concept of prevention may be culturally unfamiliar, particularly to new immigrants, and not readily accepted (449); the language barriers and lack of health insurance that limit access to health care also limit access to preventive services (25, 490). Attempts by individual physicians to promote preventive measures among individual minority patients may be unsuccessful without community-wide interventions that address the minority group as a whole and set the health problems in the broader, social context. This section reviews preventive interventions and specific clinical issues relevant to smoking prevention and cessation, tuberculosis screening and chemoprophylaxis, and immunization for influenza and pneumococcal pneumonia.

*Smoking.* The high prevalence of smoking among some minority groups in the United States demands the attention of clinicians and others concerned with public health to prevent further initiation of smoking and to assist current smokers to quit. At the community level, minorities have been the target of tobacco industry advertising (491); having inadequate knowledge of the health consequences of smoking may increase the influence of the advertising on their initiation of smoking. For example, Hispanics are viewed as a fertile market (492), and those speaking only Spanish may lack access to educational resources that would help them to resist the blandishments of tobacco merchants (493). On the other hand, minority groups are aware of this targeting. Data from a recent tobacco-use survey in California suggests that Hispanics have recognized the potential consequences of tobacco advertising targeted at their community and support public health measures to limit smoking (282). Nearly 60% of Hispanic smokers supported a ban on tobacco advertising, compared with less than 40% of non-Hispanic smokers. Hispanic smokers were also more supportive of measures to restrict access to tobacco for children and for increasing taxes on tobacco products.

Community-wide interventions to prevent smoking initiation and facilitate smoking cessation are needed among all racial and ethnic populations of the United States (494, 495). Components of community-wide interventions have been described in the Healthy People 2000 report (26), and they include (1) mass media campaigns to target high-risk groups, (2) increased excise taxes on tobacco products, (3) increased minimum age for purchase of tobacco products, (4) prohibiting the distribution of tobacco prod-

uct samples to minors, (5) elimination or severe restriction of tobacco product advertising and promotion to which youths are likely to be exposed, (6) restricting the sale of tobacco products through vending machines, (7) enforcing tobacco access laws for minors, and (8) school-based prevention programs.

Although prevention of smoking initiation by children and adolescents among all racial and ethnic groups in the United States is a major public health priority (26), little information is available on the effectiveness of interventions that target minority youth. However, peer influence may be the strongest predictor of smoking initiation among adolescents regardless of racial or ethnic background (238, 496), and thus interventions to limit peer influence are fundamental to any program. In a recent survey of 3,129 Latino adolescents from New York City, Dusenbury and coworkers (496) found that experimental smoking was 8.1 times more likely (95% CI = 6.0–10.9) and current smoking 16.9 times more likely (95% CI = 7.8–36.5) among adolescents with 50 to 100% of their friends smoking than among adolescents with none of their friends smoking.

In addition to interventions to prevent tobacco use, physicians and other health care professionals can be effective in counseling about the health effects of smoking and in assisting smokers to quit (497). Education about the health effects of smoking may be facilitated by materials that are culturally sensitive (493) and provide messages that are appropriate to the patient's preferred language and educational and cultural background. Studies comparing the efficacy of culturally appropriate and standard materials have not been reported (498). In a community intervention to promote smoking cessation among Hispanics in the San Francisco area, Perez-Stable and coworkers (499) developed a Spanish language self-help guide to quitting smoking and evaluated smoking cessation among 431 smokers self-selected from approximately 2,000 candidates who received the guide and who provided demographic and smoking status information. At 14 months, 8.4% had quit, similar to the proportion who quit with other self-help materials (500). However, this study is limited by the low proportion who participated and the lack of a control group. Despite the limitations of this study the guide may help in assisting Spanish-speaking smokers to quit, and it is available from the National Cancer Institute.

**Tuberculosis.** The priorities of tuberculosis control are the identification of infected persons with PPD screening and provision of INH chemoprophylaxis and treatment of active tuberculosis (292, 489, 501). Patients arriving in the United States with an immigrant visa are usually adequately screened on admission. However, screening may not occur among those arriving on student or visitor visas. Among Asian immigrants, their high risk of tuberculosis and young age make them a target group for tuberculin skin test screening and appropriate intervention strategies (93). Immigrants who receive bacille Calmette-Guérin (BCG) vaccination prior to coming to the United States should be evaluated and provided chemoprophylaxis as if they never had BCG, i.e., a positive PPD reaction should be assumed to be positive for tuberculosis infection rather than from BCG inoculation (292, 502).

Because an initial PPD may be negative, repeating the skin test 2 to 3 wk after the initial test may be indicated to increase the sensitivity of the test, an increase termed the "booster phenomenon." Morse (503, 504) reported that 52% of Southeast Asian refugees with initially "nonsignificant" tuberculin tests had "significant" reactions on PPD testing 1 to 3 wk later. This "conversion" phenomenon affects a significant percentage of refugees and continues to occur over time. Its public health significance

is unclear, but it presumably reflects the boosting of a waned, preexisting mycobacterial sensitivity.

Screening for tuberculosis has to be followed by chemoprophylaxis to decrease the risk of active tuberculosis. However, barriers for patients and physicians may limit the success of screening and chemoprophylaxis programs. In a case-control study of 46 cases of active tuberculosis in American Indians from South Dakota, Mori and coworkers (505) found that 75% of the tuberculosis cases could have been prevented if American Thoracic Society guidelines (506) for treatment of tuberculosis infection had been followed. Of 41 cases of active tuberculosis, only 29% had been given INH for prophylaxis, compared with 69% of 42 control subjects with a positive PPD who were selected from the same IHS hospital. In addition, compliance with preventive therapy was found in only 2% of the active tuberculosis cases and in 75% of the control subjects. Patient adherence with medication taking requires frequent encouragement (300) and use of multiple strategies with attention to cultural appropriateness of the strategies employed (507).

Although the effectiveness of INH chemoprophylaxis among persons potentially infected with INH-resistant tuberculosis could be questioned, available evidence does not support this concern. Among refugees from Southeast Asia, INH chemoprophylaxis has a small risk of failure (300), and resistance to rifampin and ethambutol does not yet appear to be a significant clinical problem. Successful screening and preventive efforts in a cohort of 9,328 Southeast Asian refugees were reported in a 5-yr surveillance study from 1980 to 1986 (295). During the 60-month follow-up period, 25 cases of tuberculosis were diagnosed in patients who were free from current disease and tuberculin reactive at time of immigration. The annual incidence of tuberculosis declined sharply from 30.6 to 5.1 cases per 10,000 during the first 24 months after immigration, and it was 4.3 cases per 10,000 at the end of the study. The 5-yr cumulative incidence of tuberculosis was 26.8 cases per 10,000 population, and it was greatest (324.3 cases per 10,000) in tuberculin-positive refugees who had abnormal screening chest radiographs and least (5.0 cases per 10,000) in those with nonsignificant tuberculin reactions. The incidence in Kampucheans (30.6 per 10,000) and Vietnamese (31.7 per 10,000) was higher than in Laotians (18.4 per 10,000). No cases occurred in children, and the cumulative incidence rose progressively with increasing age. Seven (32%) of 22 isolates were resistant to isoniazid (INH); all instances occurred in patients who had received INH preventive therapy. Although Southeast Asians remain a major reservoir of drug-resistant tuberculosis (301), refugees from Southeast Asia who receive currently recommended tuberculosis screening and preventive services are likely to show a downward trend in morbidity.

Selection of an initial treatment regimen for active tuberculosis should also consider the patient's country of origin and the region of the United States where he or she resides. Because of the high incidence of INH resistance among Asians and Pacific Islanders and in certain regions of the United States, initial daily treatment of active tuberculosis, especially for patients younger than 65 yr of age, should include multiple drugs with at least two to which they are likely to be sensitive (508). Patients with multiple-resistant strains, including INH-resistant strains, will require tailored regimens according to their drug sensitivities. INH resistance may reflect primary drug resistance or acquired resistance during therapy (previous or current) that is due in part to poor medication adherence (300, 507) and the easy availability (without prescription) of INH in Southeast Asia, as in most parts of the world. Medi-

cation noncompliance is a major problem among patients cared for at inner-city public hospitals in the United States. Brudney and Dobkin (509) found that 89% of patients discharged from Harlem Hospital with tuberculosis during 9 months of 1988 failed to complete treatment.

All of the tuberculosis chemotherapies are associated with numerous potential complications, and the risks for these complications may vary among the different minority groups. Potential problems with antituberculosis therapy have been noted in Asians and Pacific Islanders. The Asian population has a high incidence of hepatitis carriers. It is prudent to monitor liver function in patients older than 35 yr of age and patients who have preexisting hepatic dysfunction. Recent immigrants with a history of depression, suicidal tendency, or other significant psychologic problems (including extraordinary life and social adjustment stresses) must be carefully monitored to avoid possible overdose (268, 510, 511).

Infection with the human immunodeficiency virus (HIV) is a major factor contributing to the changing epidemiology of tuberculosis in the United States, particularly among minority populations (42). Knowledge about HIV infection varies among the different minority populations, and misconceptions are more prevalent among Hispanic adolescents than among white and black adolescents (512). Targeted educational campaigns are needed to correct these misconceptions and to prevent HIV infection and its associated pulmonary complications (512).

**Pneumonia and influenza.** Although the efficacy of pneumococcal and influenza vaccines has been established, and their administration is recommended by the Advisory Committee on Immunization Practices (109, 513, 514) and the U.S. Preventive Services Task Force (489), their overall use is low in those at high risk for these infections, and particularly among the minority populations of the United States (25, 26). In 1985, the United States Immunization Survey found that 23.6% of whites 65 yr of age and older received an influenza vaccination compared with 13.8% among all other races. The discrepancy between whites and all other races was less apparent among persons 20 to 64 yr of age for whom the vaccine was indicated because of a debilitating condition, 10.5 and 9.1%, respectively. Less frequent administration of these vaccines to minority patients may be partly explained by physicians not recommending the vaccines to their minority patients. In New York City, physicians with 50% or more black and Hispanic patients recommended influenza vaccination to 48% of their older patients compared with 74% recommended by physicians caring for predominately white patients (515).

The persistence of higher mortality from pneumococcal pneumonia and influenza among nonwhites compared with whites in the United States may be partly explained by the lower rates of administration of effective vaccines among the nonwhite populations. Physician recommendation may be a motivating factor for all patients, regardless of race or ethnicity, in a patient's decision to be vaccinated (516).

## RESEARCH NEEDS

The rapid growth of minority populations in the United States, and the high morbidity and mortality from many nonmalignant and malignant respiratory diseases in minority populations, point to an urgent public health problem that needs to be addressed immediately. However, much needed information for this purpose is lacking. This section provides a conceptual framework and overview of the research needs on respiratory diseases in minority populations of the United States, including epidemiologic, clinical, and preventive areas.

The recent establishment of the Association for the Respiratory Health of Minorities (ARHM) should foster research on minority populations. "The ARHM comprises physicians and health care workers dedicated to the promotion of the respiratory health of minorities through the advocacy of programs that (a) educate health care providers and their patients; (b) educate minority communities; (c) conduct basic, clinical, and behavioral research on respiratory diseases that disproportionately impact minority populations; (d) encourage underrepresented minorities to pursue careers in health care and biomedical research."

**Epidemiology.** Although we have reviewed a large body of epidemiologic data in this review, gaps remain in our knowledge of respiratory diseases in minority populations of the United States. These gaps may be partly explained by inadequate inclusion of minorities in epidemiologic investigations. Jones and coworkers (517) selected manuscripts from the *American Journal of Hygiene*, 1921 to 1960, and its successor, the *American Journal of Epidemiology*, 1965 to 1990, and examined the proportion of investigations that included "race" in their analyses. Of 558 studies conducted in the United States from 1921 to 1990, only 34% reported inclusion of nonwhite subjects. In 1990, 44% of 124 U.S. studies reported inclusion of nonwhites. Although these results do not refer specifically to epidemiologic investigations of respiratory diseases, they provide evidence that minorities have probably been underrepresented in these investigations. Failure to include minorities as subjects in health-related research prompted the National Institutes of Health to implement a policy in 1990 requiring researchers to "Include women and minorities in study populations for clinical research, unless compelling scientific or other justification for not including either women or minorities is provided" (518). Similarly, at the nationwide level, future health surveys and vital statistics need to include all minority groups to facilitate comparisons between the different groups.

The epidemiologic information on respiratory health of the minority populations of the United States primarily addresses the occurrence of disease, and only a few analytic studies have been conducted to explain the differences in disease distributions. "Race" is indicative not only of genetic factors but of culture and socioeconomic status (517). Nationwide, overall mortality rates have been associated with income, and the magnitude of income differential within a country is the strongest predictor of overall mortality (519). Those countries with the smallest differentials between rich and poor have the highest average life expectancy. On the basis of these findings, Wilkinson (519) has suggested that the purpose of research should not be ". . . to identify each separate risk factor in an attempt to account for the myriad of separate contributions to the lower class health disadvantage, but to identify points at which it is possible to intervene in the social processes which make almost all the common causes of mortality and morbidity more common in the lower social classes." Defining the causal pathway or pathways among socioeconomic factors, the development of disease, and the disease outcome is a critical need.

Although socioeconomic status is a strong predictor of morbidity and mortality, greater information on the effects of cultural and genetic factors is needed. Investigations into these factors may provide insights into disease susceptibility as it is affected by certain cultural behaviors or genetic characteristics. A greater understanding of specific cultural and genetic characteristics would also enable the development of more precise "racial" definitions for research purposes.

**Clinical.** Limited access to health care is one of the most urgent problems among minority populations of the United States. Although inadequate financial resources may be the major con-

tributor to limited access and will require reform of the health care system for resolution, other factors, including language and health beliefs, that affect access to care can be addressed through other interventions. Studies are needed to evaluate the effects of language discordance between patient and physician, and to determine the effectiveness of methods to remove the language barriers to health care access and adherence to medical advice. Similarly, more information is needed on how patients' health knowledge and beliefs about respiratory disorders affect access to care and adherence to medical advice. This information could provide the basis for implementation and evaluation of educational programs targeting patients and health professionals.

Other clinically relevant issues that need further research include pharmacanthropology (469) and the clinical consequences of physiologic differences in pulmonary function. Kalow (469) described pharmacanthropology as "interethnic comparisons of a pharmacological or toxicological nature." Interethnic differences in drug uptake, metabolism, and excretion may result from cultural practices or from genetic factors.

**Preventive.** Ultimately, the goal of further epidemiologic and clinical research on respiratory diseases in minorities is the prevention of morbidity and mortality. Identification of risk factors, including environmental and cultural factors, through epidemiologic investigations will lead to the development and evaluation of specific interventions. Such interventions must be culturally appropriate, including sensitivity to language preferences and to health beliefs and attitudes. Although further research is needed to identify and ameliorate the risk factors for some respiratory diseases among minority populations, sufficient information is currently available to begin interventions and evaluations on smoking and immunizations and on screening and chemoprophylaxis for tuberculosis.

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