

# Chapter 2

## Health Disparities in Tobacco Smoking and Smoke Exposure

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### Key Points

- While overall smoking rates have decreased in the USA, disparities related to tobacco smoking by race/ethnicity and socioeconomic status persist.
- Secondhand smoke exposure also differs by race/ethnicity and socioeconomic status, but objective measurement using cotinine levels is complex because nicotine metabolism differs by gender, race/ethnicity, and type of cigarette consumed.
- Reporting aggregate data on racial/ethnic groups, sampling strategies that capture small numbers of disparate groups, and low response rates to national surveys are examples of some of the methodological challenges that influence the study of tobacco-related health disparities.
- Comprehensive tobacco control programs are essential in developing strategies to reduce health disparities in tobacco-related respiratory diseases.

### Introduction

Cigarette smoking rates in the USA have dramatically declined in the past 50 years, and the reduction in cigarette smoking is one of the top public health achievements in the 20th and 21st centuries [1, 2]. Per capita cigarette consumption has declined from 4345 cigarettes in 1963 to 1196 cigarettes in 2012 [2]. However, in the past 10 years, declines in cigarette smoking have slowed among adults [3].

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Approximately 42 million Americans smoke, putting many smokers at risk for tobacco-related and -caused diseases and conditions [4].

Tobacco exposure, including cigarette smoking and secondhand smoke exposure (SHS), is the leading cause of preventable death in the USA [5] and globally [6]. Worldwide, tobacco kills more than six million people annually and in the USA, an estimated 480,000 Americans die each year from tobacco exposure [2]. Cigarette smoking is responsible for one in five deaths in the USA annually [2]. Since 1964, 20 million Americans have died from smoking-attributable diseases [2]. Tobacco affects nearly every organ in the body [7]. Tobacco exposure in utero and among children, adolescents, and adults can increase the risk for adverse reproductive health outcomes, cancer, cardiovascular disease, respiratory disease, hip fractures, sudden infant death syndrome, cataracts, and other conditions [7] (see Table 2.1). There are economic costs as well. Annual indirect costs due to productivity losses are \$150 billion [2] and medical expenses range from \$130 billion to \$176 billion [2].

There are 16 million people in the USA who have at least one tobacco-related serious illness [2], and tobacco is associated with the top three leading causes of death in the USA. Among adults age 35 years and older, 41 % of all smoking attributable deaths are due to cancer [7], 32.7 % are due to cardiovascular disease, and 26.3 % are due to respiratory disease [8]. The three major categories of tobacco-caused deaths are lung cancer ( $n=128,922$ ), ischemic heart disease ( $n=126,005$ ), and chronic obstructive pulmonary disease (COPD) ( $n=92,915$ ). In addition, 49,400 lung- and heart disease-related deaths are due to SHS annually [8].

About 90 % of all lung cancers in the USA are due to tobacco [8], and lung cancer is the leading cause of cancer mortality in the USA. From 2005 to 2011, 5-year survival rates have increased from 11.4 to 17.4 %, but survival rates remain quite low [9]. Lung cancer comprises an estimated 13.3 % of all new cancers and 26.8 % of cancer deaths [9]. Most importantly, lung cancer can nearly be eliminated if tobacco were eliminated. Therefore, the prevention of lung cancer by targeting tobacco exposure has been a primary goal for the U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion [10]. Unfortunately, tobacco-caused respiratory diseases and conditions other than lung cancer have received less attention. Tobacco exposure affects the trachea, bronchi, and the lungs. The primary nonmalignant respiratory diseases caused by tobacco exposure are asthma and COPD, which includes emphysema and chronic bronchitis.

The 1964 Surgeon General's report, *Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service*, was the first document to conclusively state that smoking causes chronic bronchitis [11].

"Cigarette smoking is the most important cause of chronic bronchitis in the USA and increases the risk of dying from chronic bronchitis [11]."

The casual relationship between smoking and COPD was later confirmed in the 1984 Surgeon General's report, *The Health Consequences of Smoking: Smoking and Chronic Obstructive Lung Disease* [12]. Subsequent reports further supported this finding, and additional diseases and conditions have been causally linked to

**Table 2.1** Diseases and conditions causally linked to tobacco exposure

Cancer	Cardiovascular	Respiratory	Other
• Oropharynx	• Stroke	• Chronic obstructive pulmonary	• Oral clefts <sup>a</sup>
• Larynx	• Coronary heart disease	• $\alpha$ 1-antitrypsin deficiency and cutis laxa are genetic causes of COPD <sup>a</sup>	• Reduced fertility in women
• Esophagus	• Atherosclerotic peripheral vascular disease	• Childhood asthma incidence <sup>a</sup> , poor asthma control, exacerbations of asthma in adults, asthma symptoms, wheezing severe enough to be diagnosed as asthma in susceptible children and adolescents	• Erectile dysfunction <sup>a</sup>
• Trachea	• Abdominal aortic aneurysm	• Chronic respiratory symptoms	• Low birth weight and fetal growth
• Acute myeloid leukemia		• Acute respiratory illness	• Ectopic pregnancy <sup>a</sup>
• Stomach		• Pneumonia	• Preterm delivery, still births <sup>a</sup> , and other pregnancy complications
• Pancreas		• Mycobacterium tuberculosis disease <sup>a</sup> and mortality <sup>a</sup>	• Sudden infant death syndrome
• Kidney and ureter		• Reduced lung function	• Periodontitis
• Cervix		• Impaired lung growth	• Diabetes mellitus <sup>a</sup>
• Bladder		• Early onset of lung function decline	• Diminished health status <sup>a</sup>
• Lung and bronchus		• Lower respiratory illnesses	• Hip fractures
• Liver <sup>a</sup>		• Middle ear disease	• Nuclear cataracts
• Colorectal <sup>a</sup>		• Nasal irritation	• Macular degeneration <sup>a</sup>
• All-cause mortality and cancer-specific mortality in cancer patients and survivors <sup>a</sup>			• Low bone density in postmenopausal women
• Second primary cancers <sup>a</sup>			• Peptic ulcer disease in persons with <i>Helicobacter pylori</i>
			• Odor annoyance
			• Rheumatoid arthritis <sup>a</sup>
			• Inflammation and impair immune function <sup>a</sup>
			• Nicotine activates biological pathways through which smoking increases the risk for disease <sup>a</sup>

<sup>a</sup>Causal link reported for the first time in Surgeon General's Report in 2014

Source: The Health Consequences of Smoking—50 years of Progress: A report of the Surgeon General. 2014

**Table 2.2** Causal relationships between tobacco use and exposure and respiratory diseases and conditions

Active smoking	Secondhand smoke exposure
<ul style="list-style-type: none"> <li>• Lung cancer</li> </ul>	<ul style="list-style-type: none"> <li>• Lung cancer in nonsmokers</li> </ul>
<ul style="list-style-type: none"> <li>• Poor asthma control</li> </ul>	<ul style="list-style-type: none"> <li>• Stroke</li> </ul>
<ul style="list-style-type: none"> <li>• Asthma-related symptoms (i.e., wheezing) in childhood and adolescence</li> </ul>	<ul style="list-style-type: none"> <li>• Coronary heart disease morbidity and mortality</li> </ul>
<ul style="list-style-type: none"> <li>• Acute respiratory illnesses, including pneumonia, in persons without underlying smoking-related chronic obstructive lung disease</li> </ul>	<ul style="list-style-type: none"> <li>• Ever having asthma among children of school age</li> </ul>
<ul style="list-style-type: none"> <li>• Exacerbations of asthma in adults</li> </ul>	<ul style="list-style-type: none"> <li>• Lower respiratory illnesses in infants and children</li> </ul>
<ul style="list-style-type: none"> <li>• Chronic obstructive pulmonary disease morbidity and mortality</li> </ul>	<ul style="list-style-type: none"> <li>• Middle ear disease in children, including acute and recurrent otitis media and chronic middle ear effusion</li> </ul>
<ul style="list-style-type: none"> <li>• All major respiratory symptoms among adults, including coughing, phlegm, wheezing, and dyspnea</li> </ul>	<ul style="list-style-type: none"> <li>• Ever having asthma in school age children</li> </ul>
<ul style="list-style-type: none"> <li>• Mycobacterium tuberculosis disease and mortality</li> </ul>	<ul style="list-style-type: none"> <li>• Exposure after birth and lower level of lung function during childhood</li> </ul>
<ul style="list-style-type: none"> <li>• Premature onset of accelerated age-related decline in lung function among adults</li> </ul>	<ul style="list-style-type: none"> <li>• Cough, phlegm, wheeze, and breathlessness among children of school age</li> </ul>
<ul style="list-style-type: none"> <li>• Reduced lung function and impaired lung growth during childhood and adolescence</li> </ul>	<ul style="list-style-type: none"> <li>• Onset of wheeze illnesses in early childhood</li> </ul>
<ul style="list-style-type: none"> <li>• Early onset of decline in lung function during late adolescence and early adulthood</li> </ul>	<ul style="list-style-type: none"> <li>• Maternal smoking and persistent adverse effects on lung function across childhood</li> </ul>
<ul style="list-style-type: none"> <li>• Respiratory symptoms in children and adolescents including coughing, phlegm, wheezing, and dyspnea</li> </ul>	
<ul style="list-style-type: none"> <li>• Asthma-related symptoms (i.e., wheezing) in childhood and adolescence</li> </ul>	
<ul style="list-style-type: none"> <li>• A reduction of lung function in infants of mothers who smoked during pregnancy</li> </ul>	
	<ul style="list-style-type: none"> <li>• Odor annoyance</li> </ul>
	<ul style="list-style-type: none"> <li>• Nasal irritation</li> </ul>

Source: The Health Consequences of Smoking—50 years of Progress: A report of the Surgeon General, 2014

tobacco exposure. The 2004 Surgeon General's report on *Smoking and Health* [7] confirmed that active smoking and involuntary exposure to tobacco smoke cause multiple preventable respiratory diseases and conditions that affect the trachea, bronchi, and lungs of the respiratory tract (see Table 2.2). Tobacco exposure increases the risk for acute respiratory illnesses, respiratory symptoms, and reduced lung function among children and adults. Data also suggest that tobacco use is

associated with asthma, idiopathic pulmonary fibrosis, bronchiolitis, influenza, Legionnaires' disease [7], and pulmonary hypertension [13]. There is growing evidence to support that respiratory bronchiolitis-interstitial lung disease [14], histiocytosis X [14], smell dysfunction [15, 16], and snoring [17] are related to tobacco exposure, but causal relationships have not yet been confirmed. The Surgeon General's report, *The Health Consequences of Smoking—50 Years of Progress*, is the first to address tuberculosis related to tobacco exposure [2]. Tobacco use and exposure are associated with about 53,795 respiratory disease-related deaths annually [8].

The mechanisms by which tobacco exposure causes and is linked to respiratory diseases and conditions are described in detail in the Surgeon General's report, *How Tobacco Smoke Causes Disease* [13]. In brief, tobacco smoke exposure moves through the mouth to the upper airways and eventually reaches the alveoli [13]. Both harmful soluble gases and particles are deposited in the airways and alveoli [13]. Tobacco use and exposure increase the exposure of the airways and lungs to toxic constituents, and over time, tobacco smoke can reduce the lung defenses to these toxins. Tobacco smoke reduces the clearance rate of particles from the lung, and 60 % of the particles from cigarette smoke are deposited in the lung [13]. Reduced particle clearance is due to the shortening, loss, or discoordination of cilia [12, 18, 19] and possibly changes in airway surface liquid including mucus viscoelasticity [12, 19, 20]. Furthermore, these particles are difficult to clear due to their high numbers, and smokers remove these particles at a slower rate [12]. The amount of particles and gases received from tobacco smoke depends on the nature of the tobacco, puff volume, air drawn in through ventilation holes of cigarettes, and local characteristics within the lung that determine the diffusion of toxic gases and the deposition of particles. The repeated exposure to these gases and particle damage to the mucociliary system increase the risk for bacterial or viral infections [13].

Tobacco-caused and tobacco-related respiratory diseases and conditions affect all smokers, but studies suggest that some racial/ethnic groups and individuals of low socioeconomic status (SES), and the intersection of these groups, suffer disproportionately from respiratory diseases and conditions. Tobacco use has also been linked to disparities in lung and other cancers and cardiovascular disease. There is adequate evidence to say that tobacco causes disparities in cancer among minority racial/ethnic groups [10, 21] and low SES groups [22]. However, it remains unclear if tobacco exposure is a cause of health disparities related to nonmalignant respiratory diseases among minority racial/ethnic groups and low SES groups in the USA.

For example, cigarette smoking is the primary cause of chronic obstructive pulmonary disease (COPD) [2]. Approximately 80–90 % of all COPD deaths are caused by smoking [23]. COPD is associated with an elevated risk of lung cancer and although African Americans have similar COPD prevalence rates as Whites [24], African American men with COPD have a sixfold increased risk for lung cancer compared to Whites [25]. African American men have the highest incidence and death rates of lung cancer in the USA [9]. Disparities in lung cancer between African American and White men and women are largely unexplained by the duration, frequency, and intensity of cigarette smoking [21, 25, 26]. In one study, 94 % of

African American men and 78 % of African American women with lung cancer also had a diagnosis of COPD [27]. These data suggest that it is possible that a respiratory diagnosis can contribute to tobacco-caused disparities in another disease category since African Americans disproportionately suffer from lung cancer incidence and mortality.

COPD can also contribute to deaths from pneumonia, ischemic heart diseases, and heart failure [20, 28–31], and heart disease disproportionately affects minority racial/ethnic groups. Deaths from heart disease, stroke, and hypertension combined are higher among African Americans compared to all other ethnic groups and almost twice that of White adults [32]. Furthermore, SHS increases adverse health outcomes among COPD patients and could adversely affect minority groups who are more likely to be exposed to SHS [33, 34]. Thus, although Whites suffer more adverse health outcomes from COPD [23], COPD increases the risk for other tobacco-caused illnesses that minority groups suffer from disproportionately.

The purpose of this chapter is to (1) provide an overview of populations in the USA who disproportionately experience disparities; (2) review current data on tobacco exposure among these groups; (3) present a framework for examining the problem; (4) discuss gaps in research and methodological challenges; and (5) provide suggestions for future research and practice.

This chapter specifically focuses on disparities in tobacco use and exposure among racial/ethnic minority and low socioeconomic groups for which there have been long-standing disparities. We report on the intersection between gender and race/ethnicity and gender and socioeconomic status (SES) when possible. There is insufficient evidence on tobacco-related health disparities in lesbian, gay, bisexual, and transgender (LGBT) individuals and populations that suffer from mental illnesses, but we report the available data. Recommendations for research and practice are made for all of these populations in the chapter summary.

## **Populations in the USA Who Disproportionately Experience Tobacco-Related Health Disparities**

There are differences in health and indicators of health, but not all differences are health disparities and not all similarities suggest an achievement of equity. For example, smoking prevalence has declined among racial/ethnic groups, and African Americans and Whites have similar smoking rates. In 2013, current smoking was 18.3 % among African American and 19.4 % among White adults [4]. African Americans smoke fewer cigarettes per day on average, have a higher percentage of non-daily smokers, and have later age of onset of smoking compared to Whites [21, 35]. If one were to only examine these indicators, one might assume that there is equity and possibly a slight health advantage to African Americans as compared to Whites.

However, African Americans have disproportionately higher tobacco-caused cancer morbidity and mortality rates and lower survival rates. One might suggest that the lag in lung cancer rates may be due to lag in time related to smoking declines. Yet, historically, cigarettes smoking rates among African American males were not much higher than White males in 1965 and began to decline at the same time. In addition, smoking rates among African American women since 1965 have been similar to rates among White women [36], but African American women have historically had higher lung cancer incidence rates and lower 5-year survival rates than White women. These disparities are largely unexplained using the dose–response model of lung cancer. In this chapter, disparities are examined from a broad perspective, since not one indicator tells the entire story and there are multiple factors that influence the respiratory disease continuum in minority racial/ethnic and low SES groups.

### ***Definition of Tobacco-Related Disparities***

The definition of tobacco-related disparities was derived from the 2002 *National Conference on Tobacco and Health Disparities: Forging a National Research Agenda to Reduce Tobacco Related Health Disparities*, which was a meeting of national stakeholders co-sponsored by the National Cancer Institute, Centers for Disease Control and Prevention, the American Legacy Foundation, the Robert Wood Johnson Foundation, the American Cancer Society, the Campaign for Tobacco-Free Kids, the National African American Tobacco Prevention Network, and the National Latino Council on Alcohol and Tobacco. The definition was created at a time when stakeholders at local, state, and national levels were defining health disparities and seeking to increase the visibility of the need to address disparities within the USA. The consensus statement developed by this group defined tobacco-related disparities as, “differences in patterns, prevention, and treatment of tobacco use; the risk, incidence, morbidity, mortality, and burden of tobacco-related illness that exist among specific population groups in the USA; and related differences in capacity and infrastructure, access to resources, and environmental tobacco or SHS” [37].

This definition was later modified slightly by Fagan and colleagues [38] to capture more details embedded in the patterns of use that impact prevention and treatment: “tobacco-related health disparities are differences in exposure to tobacco, tobacco use initiation, current use, number of cigarettes smoked per day (cpd), quitting/treatment, relapse, and the subsequent consequences among specific groups, and include differences in capacity and infrastructure as well as access to resources”.

In this expanded definition, differences in capacity, infrastructure, and access to resources are inclusive of access to care, quality of health care, socioeconomic indicators that impact health care, and psychosocial and environmental resources [38]. These definitions were intended to provide a framework for the scope of research that is needed to understand tobacco-related disparities at different points

along the tobacco-disease continuum, different trajectories that lead to health consequences, and how various social, community, and societal level factors that interact with tobacco use/exposure contribute to the development of or amelioration of tobacco-related disparities.

## ***Populations Who Experience Tobacco-Related Disparities***

In 2018, the nation will celebrate the 20-year anniversary of the publication of the 1998 Surgeon General's Report, *Tobacco Use Behaviors Among U.S. Racial/Ethnic Minority Groups* [21]. This was the first major government report to bring attention to the need to examine tobacco use and disease outcomes in minority racial/ethnic groups in the USA. This report focused on Blacks/African-Americans, Hispanic/Latino Americans (Hispanics/Latinos), American Indians and Alaska Natives (American Indian/Alaska Natives), and Asian, Native Hawaiian, and other Pacific Islander Americans. This chapter defines these groups more inclusively since data are often reported using aggregate racial/ethnic categories. This chapter also recognizes the heterogeneity within each aggregate racial/ethnic group where possible. The aggregate categories include people who come from diverse cultures, nationalities, religions, heritages, and lifestyles.

American Indians and Alaska Natives are people whose ancestors include any of the original peoples of North and South America (including Central America) and who maintain tribal affiliation or community affiliation or attachment with their indigenous group [39]. There are approximately 566 federally recognized tribes [40] and non-federally recognized tribes that have their own culture, beliefs, and practices. We use Blacks/African-Americans to be inclusive of the diverse people who self-identify as Black or African American. This category may include people of US born descent, Caribbean descent, or immigrants from other countries. Hispanic/Latino/Spanish American is an aggregate ethnic category that includes people who self-identify with at least one of these terms, and this identification is consistent with the census terminology as well. Persons who self-identify as Hispanic/Latino/Spanish American often are people from Latin American, South America, or Spain. Asian, Native Hawaiian, or other Pacific Islander Americans is an aggregate category that comprises persons of Asiatic descent and persons of Polynesian, Melanesian, or Micronesian descent. The aggregate grouping is largely based on sample size rather than similarities in origin. Furthermore, the category is somewhat misleading since these social groups convey different disease risks related to tobacco. Some studies have used Asian Americans alone or Native Hawaiian/Pacific Islander alone. Although important to report, because of the population sizes at the national levels, there are often too few data to report out specific Asian groups including Japanese, Chinese, Korean, Vietnamese, Hmong, Filipinos (many of whom will state they are of Hispanic origin), and many other Asian ethnic groups. The Native Hawaiians and Pacific Islanders category includes Native Hawaiians, Samoans, Guamanians, Chamorros, Tahitians, Tongans, Tokelauans,



Chuukese, Palauans, Yapese, Marshallese, Carolinians, Pohnpeians, Kosraeans, Nauruans, Fijians, Guineans, or Solomon Islanders, or other Pacific Islander ethnic groups [41]. Although important to report if available, Native Hawaiians and Pacific Islanders are often not reported in national data due to sample sizes, but these groups also experience disparities. In 2015, the first national survey on Native Hawaiians and Pacific Islanders was released as public data [42].

Thus, the four major minority racial/ethnic groups in the USA (American Indian/Alaska Native, Black/African American, Hispanic/Latino/Spanish American, Asian/Native Hawaiian/Pacific Islander Americans) are aggregate categories with unique ethnolinguistic characteristics; multiple ancestries; different histories of entry to the USA; diverse settlement in the USA; and different evolutions as racial, ethnic, and minority groups. None of these racial/ethnic groups represent biological groups or are necessarily used to describe one’s skin color. Common factors shared by some of these racial/ethnic groups include that they have often suffered from disparities and estimates suggest that these groups will experience population growth in the next 50 years.

Overall, the USA will experience population growth and the total population will increase by 98.1 million between the years 2014 and 2060 [43] (see Table 2.3). Changes in population size are driven by births, deaths, and net international migrations [43]. The U.S. Census Bureau estimates that as the number and proportion of non-Hispanic Whites declines, the number and proportion of minority populations will increase. For example, the White population will decrease from 198 million in 2014 to 182 million in 2060, and the number and proportion of all other racial/ethnic categories will increase [43] (see Table 2.3). In 2014, minority comprised 37.8 % of the US population and in 2060 will comprise 56.4 % of the US population [43]. The actual growth of minority populations will more than double and increase from 116.2 million people in 2012 to 241.3 million by 2060 [44]. The number of

**Table 2.3** Population growth estimates for racial/ethnic aggregate groups in the USA

Race/ethnicity	2014	2060
	% or number	% or number
Total population (in millions)	318,748	416,795
White alone <sup>a</sup>	77.7	68.5
White alone, not Hispanic or Latino	62.6	43.6
Black or African American alone <sup>a</sup>	13.2	14.3
American Indian and Alaska Native alone <sup>a</sup>	1.2	1.3
Asian alone <sup>a</sup>	5.4	9.3
Native Hawaiian and Other Pacific Islander <sup>a</sup>	0.2	0.3
Two or more races	2.5	6.2
Hispanic or Latino <sup>b</sup>	17.4	28.6

Source: Colby S and Ortman JM. Projections of the size and composition of the US population: 2014 to 2060, Current Population Reports, P25-1143, U.S. Census Bureau, Washington, DC 2014

<sup>a</sup>Includes persons reporting only one race

<sup>b</sup>Hispanics may be of any race, so also are included in applicable race categories

Americans of Hispanic ethnicity will more than double by 2060 and Hispanics will experience the largest increase of all racial/ethnic groups (see Table 2.3). In 2014, 48 % of children under age 18 were minority and by 2060, 64.4 % of children in the USA will be minority [43].

As minority racial/ethnic populations grow in the USA, our nation's health is not likely to improve. Minority racial/ethnic groups are over-represented at the bottom end of the socioeconomic ladder. Since 1967, median household income has both increased and decreased among racial/ethnic groups. For example, among all racial/ethnic groups, in 1967 the median household income was \$43,558 and in 2013 was \$51,939. Among Asians and Pacific Islanders, the median income was \$63,214 in 1987 (year data were first collected) and was \$70,571 in 2001 [45]. The racial/ethnic categories were then changed to separate Asians from Pacific Islanders. Among Asians, the median income was \$68,143 in 2002 and \$67,065 in 2013. Data are not reported for Pacific Islanders or Native Americans and Alaska Natives. Among non-Hispanic Whites, the median income was \$51,380 in 1972 and \$58,270 in 2013. Among Hispanics, the median income was \$38,229 in 1972 (year data were first collected) and \$40,963 in 2013. Among African Americans, the median income was \$29,569 in 1972 and \$34,598 in 2013. In 2013, the median household income among Asian Americans was more than double that in African Americans [45].

The poverty rate for all Americans was 14.7 % in 1966 and 14.5 % in 2013 [45]. For the first time since 2006, poverty rates declined from 15 % in 2012 to 14.5 % in 2013, but the number of people in poverty did not significantly change [45]. Furthermore, there have been very small fluctuations in the percent of people in poverty. In 2013, 9.6 % of Whites, 10.5 % of Asians, 10 % of Asian and Pacific Islanders, 27.2 % of African Americans, and 23 % of Hispanics lived in poverty [45]. Aggregate data, like Asian and Pacific Islander, mask some of the differences in poverty among racial/ethnic groups. For example, prior data show that American Indians, Alaska Natives, and Native Hawaiians have higher levels of poverty than Whites. If the data were aggregated with Asians, who have lower levels of poverty, then the data would be misleading. Data from the U.S. National Center for Education Statistics also show that individuals with greater educational attainment were further away from poverty than those with less education, and overall, Asians and Whites have higher educational attainment compared to the other racial/ethnic groups [46].

According to the 2014 National Healthcare Quality and Disparities Report, few disparities were eliminated. For example, advice for cessation services for African Americans decreased. Poor people generally experienced less access and worse quality health care compared to more advantaged people. Disparities in health care quality and outcomes by income and race/ethnicity are large, remained the same, and did not improve substantially through 2012 [47]. Through 2012, most disparities in access to care related to income and race/ethnicity also showed no significant change, neither getting smaller nor larger.

Improvements have been observed in health insurance coverage among adults. From 2000 to 2010, the percentage of adults aged 18–64 who were uninsured increased from 18.7 to 22.3 % [47], whereas from 2010 to 2013, the percentage

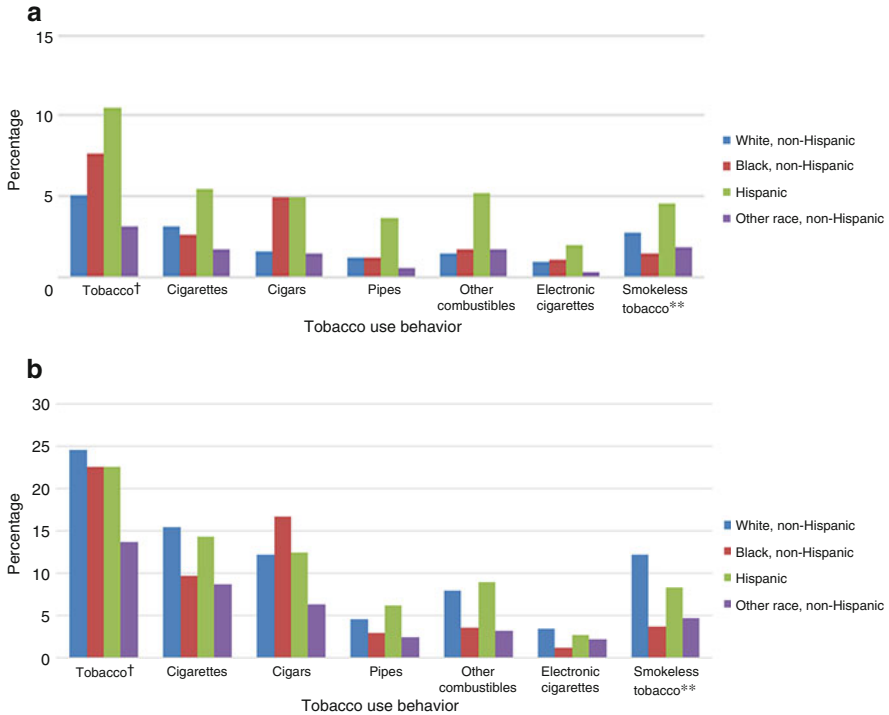
without health insurance decreased to 20.4 %. During the first half of 2014, the percentage without health insurance decreased even further to 15.6 %. Although disparities still exist in insurance coverage and African Americans and Hispanics are less likely to be insured than Whites, uninsured adults decreased from 2013 to 2014 among three racial/ethnic aggregate groups reported. In 2013, 14.5 % of Whites, 24.9 % of African Americans, 40 % of Hispanics reported being insured. In 2014, 11.1 % of Whites, 15.9 % of African Americans, and 33.2 % of Hispanics reported being uninsured. Improvement in insurance coverage is likely due to the 2010 Affordable Care Act, which as part of its implementation established marketplace enrollment in health insurance in 2013. No such declines in the uninsured population were observed among racial/ethnic groups prior the implementation of the Affordable Care Act [47]. It is important to determine whether improvements in health insurance will lead to improvements in preventive care, access to care, and quality care among the poor and minority racial/ethnic groups. As the US population becomes more diverse, it becomes more important to monitor changes in access to care and quality care among racial/ethnic and socioeconomic groups.

## **Tobacco Use Disparities**

Racial/ethnic and SES disparities exist in tobacco use and SHS exposure. Differences in smoking prevalence rates exist by employment status, occupation, income, poverty, and education. SES, race/ethnicity, and gender often interact to increase tobacco-related disparities among these groups. We briefly review tobacco use prevalence rates among racial/ethnic and low SES groups as well as SHS exposure in these groups using the available data.

### ***Tobacco Use Rates Among Young People***

Healthy People 2020 seeks to reduce cigarette smoking among adolescents to 21 % overall and less than 16 % in the past 30 days as a strategy to help reduce tobacco-related and tobacco-caused diseases and conditions in the USA [10]. Significant progress was made in reducing cigarette smoking as a result of the 1998 Master Settlement Agreement (MSA) [48]. The MSA resulted after Attorney Generals from 46 states, five US territories, and the District of Columbia filed a lawsuit against tobacco industry to recover health care-related costs of tobacco use. Five of the largest tobacco industries paid states approximately \$10 billion per year, and the MSA sets standards for the sales and marketing of cigarettes, particularly to young people. Cigarette smoking rates declined among young people after the 1998 Master Settlement Agreement from 2000 to 2009 [31] and then reached a plateau. Recent data show dramatic changes in the use of combustible versus noncombustible tobacco among middle school and high school students [49, 50].



**Fig. 2.1** (a) Percentage of middle school students currently using\* tobacco products, by school level, sex, race/ethnicity, and product type—National Youth Tobacco Survey, United States, 2012. (b) Percentage of high school students currently using\* tobacco products, by school level, sex, race/ethnicity, and product type—National Youth Tobacco Survey, United States, 2012

Among adolescents, tobacco use varies by tobacco product. Combustibles, including cigarettes and cigars, have historically been used more commonly than other tobacco products and to our knowledge, pose a higher risk for respiratory disease than noncombustibles (see Fig. 2.1). In 2012, among middle school students, past 30-day tobacco use rates were highest among Hispanics, followed by African Americans, Whites, and others, respectively. Among high school students, past 30-day tobacco use was highest among Whites, followed by African Americans, Hispanics, and others, respectively. Among middle school students, cigarette use was highest among Hispanics, but among high school students, cigarette use was most prevalent among Whites. The prevalence of cigar use was highest among African Americans, followed by Hispanic, White, and other middle and high school students, respectively [49].

In 2014, a major shift occurred in the use of combustibles and noncombustibles among young people. Past 30-day use rates of electronic cigarettes and hookah increased and surpassed past 30-day use of cigarettes overall. Among high school students, 13.4 % reported electronic cigarette use, 9.4 % reported hookah use, 9.2 %

reported cigarette use, and 8.2 % reported cigar use in the past 30 days. Among middle school students, 3.9 % reported electronic cigarette use, 2.5 % reported hookah use, 2.5 % reported cigarette use, and 1.9 % reported cigar use in the past 30 days [50].

The 2014 data also showed differences in the use of combustibles and noncombustibles by race/ethnicity. Among high school students, 10.8 % of Whites, 4.5 % of African Americans, 8.8 % of Hispanics, and 5.3 % of non-Hispanic others reported cigarette use in the past 30 days. Among middle school students, 2.2 % of Whites, 1.7 % of African Americans, and 3.7 % of Hispanics reported cigarette use in the past 30 days. Data were not reported for “other” race/ethnicity. Among high school students, 8.3 % of Whites, 8.8 % of African Americans, 8.0 % of Hispanics, and 2.6 % others used cigars in the past 30 days. Among middle school students, 1.4 % of Whites, 2.0 % of African Americans, and 2.9 % of Hispanics used cigars in the past 30 days. Data were not reported for “other” race/ethnicity [50].

Noncombustible use increased from 2012 and use rates were largely driven by increases in electronic cigarettes. Among high school students, 15.3 % of Whites, 5.6 % of African Americans, 15.3 % of Hispanics, and 9.4 % of non-Hispanic others reported electronic cigarette use in the past 30 days. Among middle school students, 3.1 % of Whites, 3.8 % of African Americans, and 6.2 % of Hispanics reported electronic cigarette use in the past 30 days. Among high school students, 9.4 % of Whites, 5.6 % of African Americans, 13.0 % of Hispanics, and 6.0 % of non-Hispanic others reported hookah use in the past 30 days. Among middle school students, 1.4 % of Whites and 5.6 % of Hispanics reported hookah in the past 30 days. Data were not reported for African American middle school students and “other” race/ethnicity [50].

The most recent data are not reported by SES indicators. Parental education has often been used as a proxy for SES [51], but the data are difficult to interpret since parental education does not necessarily predict smoking rates among adolescents.

## ***Cigarette Use Among Adults***

Healthy People 2020 seeks to reduce current cigarette smoking among adults aged 18 and over to less than 12 % as a strategy to help reduce tobacco-related and tobacco-caused diseases and conditions in the USA [10]. Smoking rates among adults are slowly declining. In 2013, an estimated 17.8 % of adults smoked cigarettes [4]. The most recent data show that smoking rates among adults are highest among individuals reporting multiple races, followed by American Indians and Alaska Natives, Whites, African Americans, Hispanics, and Asians, respectively (see Table 2.4 and Fig. 2.2). Smoking decreases with educational attainment and is higher among persons in poverty compared to persons not in poverty (see Table 2.4; Figs. 2.3 and 2.4). However, there were no significant changes from 2005 to 2013 in smoking by educational attainment status. Current smoking among persons in

**Table 2.4** Percentage of persons aged >= years who were current cigarette smokers\* by selected characteristics- National Health Interview Survey, United States, 2005 and 2013

Characteristic	Men		Women		Total	
	2005 (n = 13,762)	2013 (n = 15,440)	2005 (n = 17,666)	2013 (n = 19,117)	2005 (N = 31,428)	2013 (N = 34,557)
	% (95 % CI)	% (95 % CI)	% (95 % CI)	% (95 % CI)	% (95 % CI)	% (95 % CI)
Overall	23.9 (22.9–24.8)	20.5 (19.5–21.4)	18.1 (17.4–18.9)	15.3 (14.6–16.1)	20.9 (20.3–21.5)	17.8 (17.2–18.4)
Race/Ethnicity <sup>§</sup>						
White	24.0 (22.8–25.2)	21.2 (19.9–22.4)	20.0 (19.1–20.9)	17.8 (16.8–8.8)	21.9 (21.1–22.7)	19.4 (18.6–20.2)
Black	26.7 (23.9–29.5)	21.8 (19.2–24.3)	17.3 (15.6–19.0)	15.4 (13.7–17.0)	21.5 (19.9–23.1)	18.3 (16.8–19.7)
Hispanic	21.1 (19.2–23.0)	17.3 (15.3–19.2)	11.1 (9.8–12.4)	7.0 (6.0–7.9)	16.2 (15.0–17.4)	12.1 (11.0–13.2)
American Indian/ Alaska Native	37.5 (20.7–54.3)	32.1 (20.9–43.3)	26.8 (15.5–38.1)	22.0 (12.2–31.8)	32.0 (22.3–41.7)	26.1 (18.5–33.7)
Asian <sup>¶</sup>	20.6 (15.7–25.5)	15.1 (12.1–18.1)	6.1 (3.7–8.5)	4.8 (3.2–6.5)	13.3 (10.4–16.3)	9.6 (7.9–11.4)
Multiple race	26.1 (16.3–35.9)	29.1 (22.0–36.2)	23.5 (14.8–32.2)	24.8 (18.0–31.5)	24.8 (17.7–31.8)	26.8 (21.9–31.8)
Education**						
0–12 years (no diploma)	29.5 (27.2–31.8)	30.6 (27.7–33.5)	21.9 (20.0–23.7)	18.0 (16.1–20.0)	25.5 (24.0–27.1)	24.2 (22.5–25.9)
8th grade or less	21.0 (17.7–24.3)	21.9 (17.3–26.5)	13.4 (11.1–15.6)	9.2 (6.8–11.6)	17.1 (15.1–19.0)	15.4 (12.8–17.9)
9–11th grade	36.8 (33.3–40.2)	40.0 (36.0–44.0)	29.0 (26.1–31.8)	26.6 (23.2–29.9)	32.6 (30.3–34.9)	33.3 (30.6–35.8)
12th grade, no diploma	30.2 (23.5–36.9)	24.2 (18.3–30.1)	22.2 (16.9–27.5)	15.4 (11.1–19.8)	26.0 (21.8–30.2)	19.7 (16.0–23.5)
GED	47.5 (41.4–53.6)	42.9 (36.4–49.3)	38.8 (33.6–44.0)	39.7 (33.5–45.9)	43.2 (39.0–47.4)	41.4 (36.8–45.9)
High school graduate	28.8 (27.0–30.6)	26.7 (24.6–28.8)	20.7 (19.3–22.2)	17.6 (16.1–19.2)	24.6 (23.5–25.7)	22.0 (20.7–23.3)
Some college, no diploma	26.2 (24.4–28.0)	22.4 (20.4–24.8)	21.1 (19.2–22.9)	19.5 (17.8–21.3)	23.5 (22.1–24.9)	20.9 (19.4–22.3)
Associate degree	26.1 (23.3–28.9)	17.8 (15.5–20.2)	17.1 (15.0–19.3)	17.7 (15.5–20.0)	20.9 (19.2–22.6)	17.8 (16.0–19.6)

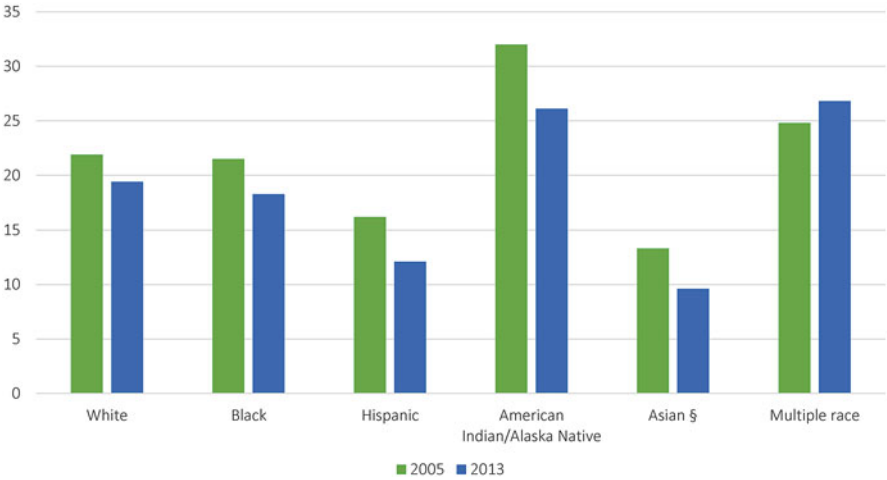
Undergraduate degree	11.9	(10.5–13.3)	10.4	(9.0–11.9)	9.6	(8.3–10.8)	7.9	(6.9–9.0)	10.7	(9.8–11.6)	9.1	(8.3–10.0)
Graduate degree	6.9	(5.3–8.5)	5.7	(4.5–7.0)	7.4	(65.9–8.8)	5.5	(4.1–6.8)	7.1	(6.0–8.3)	5.6	(4.7–6.5)
<i>Poverty status</i> <sup>††</sup>												
At or above poverty level	23.7	(22.6–24.8)	18.7	(17.7–19.7)	17.6	(16.8–18.5)	13.8	(13.0–14.6)	20.6	(19.9–21.3)	16.2	(15.6–16.8)
Below poverty level	34.3	(31.1–37.5)	33.8	(30.7–36.8)	26.9	(24.5–29.3)	25.8	(23.8–27.8)	29.9	(27.9–31.9)	29.2	(27.5–31.0)
Unspecified	21.2	(19.2–23.2)	10.9	(17.2–22.5)	16.1	(14.8–17.4)	12.6	(10.7–14.6)	18.4	(17.2–19.6)	16.0	(14.3–7.7)

*Source:* Jamal A, Agaku IT, O'Connor E, King B, Kenemer JB, Neff L. (2014). Cigarette smoking among adults—United States, 2005–2013. MMWR, 63(47); 1108–1112  
 §Excludes 45 (2005) and 73 (2013) respondents of unknown race. Unless indicated otherwise, all racial/ethnic groups are non-Hispanic; Hispanics can be of any race

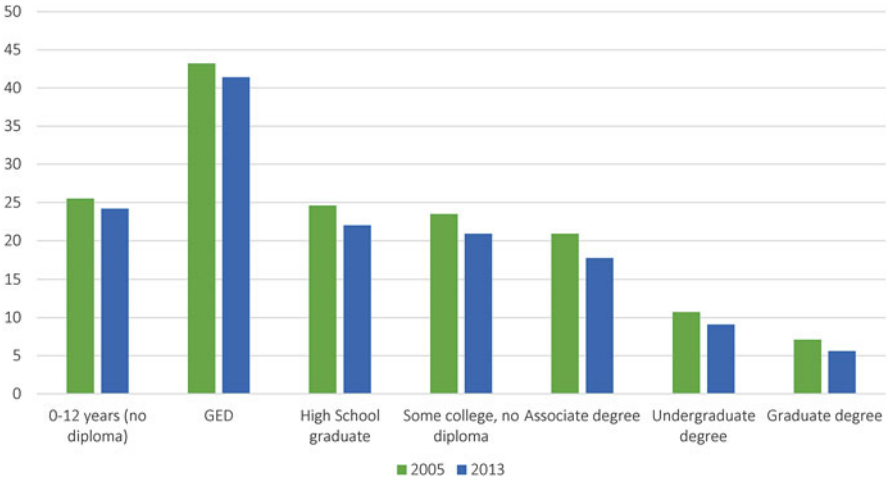
<sup>¶</sup>Does not include Native Hawaiians or Other Pacific Islanders

<sup>\*\*</sup> Among persons aged ≥25 years. Excludes 339 (2005) and 155 (2013) persons whose educational level was unknown

<sup>††</sup>Family income is reported by the family respondent who might or might not be the same as the sample adult respondent from whom smoking information is collected. 2005 estimates are based on reported family income and 2004 poverty thresholds published by the U.S. Census Bureau, and 2013 estimates are based on reported family income and 2012 poverty thresholds published by the U.S. Census Bureau



**Fig. 2.2** Percentage of persons aged  $\geq 18$  years who were current cigarette smokers,\* by race/ethnicity—National Health Interview Survey, United States, 2005 and 2012

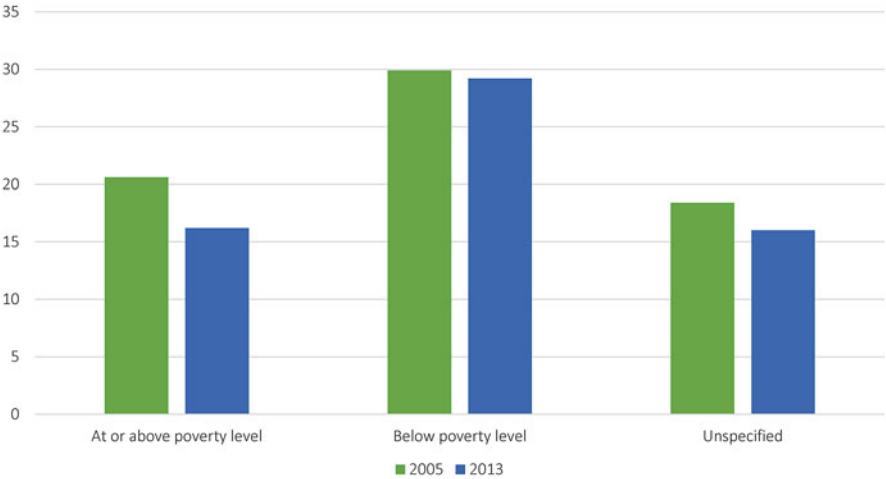


**Fig. 2.3** Percentage of persons aged  $\geq 18$  years who were current cigarette smokers,\* by education—National Health Interview Survey, United States, 2005 and 2012

poverty did not change in the years 2005 and 2013. Smoking rates are lower among women compared to men, but patterns of disparities by race/ethnicity and SES are similar for men and women [4].

For the first time, the Centers for Disease Control and Prevention recently reported cigarette smoking rates by sexual orientation [4]. In 2013, 26.6 % of lesbian, gay, and bisexual (LGB) persons reported current smoking compared to 17.6 % of straight adults. Among males, 26.4 % of LGB compared to 20.3 % of straight





**Fig. 2.4** Percentage of persons aged  $\geq 18$  years who were current cigarette smokers,\* by poverty status—National Health Interview Survey, United States, 2005 and 2012

males smoked; however, these differences were not significant. Significant differences were found among females and 26.7 % of LGB smoked compared to 15 % of straight women. Data on transgender populations, a gender identity category, were not reported.

***Intersection between Race/Ethnicity and SES with Mental Illness***

There are limited data available on tobacco use among the mentally ill but data show that smoking prevalence rates among persons with a mental illness are almost double those of persons without a mental illness [52]. Few studies have reported on smoking among the mentally ill by race/ethnicity and SES, but smoking prevalence rates among the mentally ill reflect the specific disparities observed in the general population [52]. For example, American Indians/Alaska Natives have the highest smoking prevalence rates followed by Whites, and smoking prevalence rates among the mentally ill are highest among Native Americans and Alaska Natives followed by Whites, African Americans, Hispanics and the Asian aggregate groups. Smoking prevalence in general is highest among the least educated. Smoking rates are also highest among the least educated persons who have a mental illness and lowest among the most educated. In addition, smoking rates are also higher among the poor mentally ill compared to mentally ill smokers who are not in poverty [52] (see Table 2.5).

**Table 2.5** Percentage of adults who smoke cigarettes,\* by mental illness status,<sup>†</sup> sex, and selected characteristics—National Survey on Drug Use and Health, United States, 2009–2011

	% of persons with any mental illness who smoke cigarettes ( <i>n</i> = 29,400) %	% of persons with no mental illness who smoke cigarettes ( <i>n</i> = 84,700) %
<i>Race/ethnicity</i> <sup>§</sup>		
White	37.7	22.3
Black	34	22.3
Hispanic	31.6	19.8
American Indian/Alaska Native	54.7	30.5
Asian <sup>¶</sup>	20.6	10.4
Other	40	26.3
<i>Education</i> <sup>**</sup>		
Less than high school graduate	46.6	28.9
High school graduate	40.2	25.2
Some college	38.1	21.6
College graduate	18.7	10.6
<i>Poverty status</i> <sup>††</sup>		
At or above poverty level	33.3	20
Below poverty level	47.9	32.8
Unknown	24.2	19.5
<i>Total</i>	36.1	21.4

Source: CDC (2014). Vital signs: Current smoking among adults aged. Aged ≥18 years with mental illness—United States, 2009–2011. MMWR, February 8, 2013;62(05);81–87

§ Excludes 45 (2005) and 73 (2013) respondents of unknown race. Unless indicated otherwise, all racial/ethnic groups are non-Hispanic; Hispanics can be of any race

¶ Does not include Native Hawaiians or Other Pacific Islanders

\*\* Among persons aged ≥25 years. Excludes 339 (2005) and 155 (2013) persons whose educational level was unknown

†† Family income is reported by the family respondent who might or might not be the same as the sample adult respondent from whom smoking information is collected. 2005 estimates are based on reported family income and 2004 poverty thresholds published by the U.S. Census Bureau, and 2013 estimates are based on reported family income and 2012 poverty thresholds published by the U.S. Census Bureau

## ***Disparities in Secondhand Smoke Exposure***

In 2006, the Surgeon General concluded there is no safe level of exposure to SHS [53]. About 49,000 tobacco-caused deaths each year are due to secondhand smoke (SHS) exposure [8]. SHS is inhaled involuntarily by nonsmokers including children, and the smoke lingers in the air hours after the cigarette has been extinguished [54].

SHS causes several nonmalignant respiratory conditions including nasal irritation, middle ear disease, respiratory symptoms, impaired lung function, lower respiratory illness, and sudden infant death syndrome. Secondhand exposure among

**Table 2.6** Percentage of nonsmokers aged 3 and older with serum cotinine levels 0.05–10 ng/mL, by selected demographic characteristics—National Health and Nutrition Examination Survey, United States, 1999–2012

	% with serum cotinine 0.05–10 ng/mL (95 % CI)	
Characteristic	1999–2000	2011–2012
Total	52.5 (47.1–57.9)	25.3 (22.5–28.1)
<i>Sex</i>		
Male	58.5 (52.1–64.9)	27.7 (24.7–30.6)
Female	47.5 (42.5–52.5)	23.3 (20.4–26.3)
<i>Race/ethnicity</i>		
White, non-Hispanic	49.6 (42.4–56.7)	21.8 (18.6–24.9)
Black, non-Hispanic	74.2 (70.2–78.2)	46.8 (30.8–55.6)
Mexican–American	44.3 (37.4–51.1)	23.9 (16.3–31.4)
<i>Poverty status</i>		
Below poverty level	71.6 (64.8–78.5)	43.2 (37.3–49.0)
At or above poverty level	48.8 (42.8–54.8)	21.2 (18.8–23.6)
Unspecified	53.5 (48.4–58.6)	31.7 (22.8–40.5)
<i>Education aged 25 and older</i>		
≤Grade 11	53.9 (48.7–59.0)	27.6 (23.0–32.2)
High school diploma or equivalent	51.6 (44.5–58.6)	27.5 (21.2–33.7)
Some college or associate degree	48.2 (40.8–55.6)	21.2 (17.5–24.9)
≥College diploma	35.2 (27.5–43.0)	11.8 (9.1–14.4)
<i>Own or rent home</i>		
Own	45.8 (39.3–52.3)	19.0 (16.1–22.0)
Rent	68.1 (61.6–74.6)	36.8 (32.3–41.3)

Source: Homa DM, Neff LJ, King BA, Caraballo RS, Bunnell RE, Babb SD, Garrett BE, Sosnoff CS, Wang L. (2015). Vital Signs: Disparities in Nonsmokers' Exposure to Secondhand Smoke—United States, 1999–2012. *MMWR*, February 6, 64(04);103–108

children is associated with acute respiratory infections, middle ear disease, exacerbated asthma, respiratory symptoms, and decreased lung function [7]. Prior reports have confirmed that some minority racial/ethnic and low socioeconomic groups are disproportionately exposed to SHS. If smoking rates are higher among some minority racial/ethnic groups, then one might hypothesize that secondhand smoke would be higher as well. However, disparities in SHS exposure exist, but do not mirror cigarette smoking rates as noted in Tables 2.4 and 2.6. For example, SHS exposure among African Americans is more than double that of Whites. Moreover, Mexican Americans have higher SHS exposure than Whites, yet cigarette smoking is higher among Whites than Hispanics. These disparities in SHS exposure likely influence children's risk of tobacco-caused respiratory conditions and diseases.

The principal indicator used to determine tobacco smoke exposure in nonsmokers is cotinine, which is the primary metabolite of nicotine [55]. Nicotine is first metabolized to cotinine and cotinine is metabolized to *trans* 3' hydroxycotinine, a process which is almost exclusively mediated by the enzyme cytochrome P450 2A6 (CYP2A6) [56]. Data show that SHS exposure as measured by detectable serum cotinine levels 0.05 to

10 ng/mL has significantly declined overall from 1999/2000 to 2011/2012 [57]. Despite these declines, according to the most recent data, a considerably higher proportion of non-Hispanic African American nonsmokers were exposed to SHS than other groups, and current exposure is double that of non-Hispanic Whites (see Table 2.6).

African Americans have similar smoking rates as Whites, so observed differences in SHS exposure among nonsmokers may be due to differences in policy implementation, but could also be related to differences in nicotine metabolism. African American smokers have lower odds of having smoke-free policies in the home compared to non-Hispanic Whites [58]. However, even among children who are not exposed to SHS in the home, non-Hispanic Blacks have significantly higher serum cotinine levels compared to non-Hispanic Whites [59].

Research indicates that nicotine metabolism varies by gender [44] and race/ethnicity [60, 61], and some studies show that menthol also influences the metabolism of nicotine in the liver [62]. Study findings suggest that African Americans have slower rates of nicotine metabolism as indicated by cotinine and the nicotine metabolite ratio, which is highly correlated with rates of nicotine clearance [63] and the CYP2A6 genotype, which is primarily responsible for nicotine metabolism [64, 65]. African American nonsmokers exposed to tobacco smoke may have slower nicotine metabolism like African American smokers.

Nationwide, about 76–88 % of African Americans smokers consume menthol-flavored cigarettes [66–68] compared with 26 % of the Asian/Pacific Islander smokers [49], 28 % of Hispanic smokers, and 22 % of White smokers [66, 67]. Data from Hawaii also show that 78 % of Native Hawaiian/Pacific Islander aggregate category of smokers use menthols [69]. Menthol inhibits the metabolism of nicotine in liver microsomal test systems [70, 71] by slowing oxidative metabolism and glucuronide conjugation [71]. Some studies have demonstrated higher cotinine levels [71, 72] among menthol smokers compared to non-menthol smokers [72, 73]. However, other studies have not shown higher cotinine levels among menthol compared to non-menthol smokers [74, 75]. Because race/ethnicity, gender, menthol, and other factors may influence nicotine metabolism, additional studies are needed to determine how these factors are related to and influence the assessment of SHS exposure and health disparities.

Data show declines in SHS exposure, but still indicate significant differences in SHS exposure by poverty status (see Table 2.6). In 2011–2012, a significantly greater percentage of nonsmokers living in poverty had serum cotinine levels 0.05–10 ng/mL compared to their more economically advantaged counterparts (43.2 % vs. 21.2 %) [57]. In another study, SHS exposure in the home was significantly higher among children and adolescents from families with annual income less than \$20,000 (26.4 %) compared to those earning \$20,000 or more (15.5 %) [59]. To our knowledge, studies have not examined poverty as an environmental factor that influences the metabolism of nicotine to cotinine, but it is likely that the data reflect true differences in SHS exposure among nonsmokers.

While SHS exposure has declined overall, there continues to be differences by educational attainment (see Table 2.6). Among nonsmokers with less than 11 years of education, 27.6 % were exposed to SHS, 27.5 % with a high school education or equivalent, 21.2 % with a college or associates degree, and 11.8 % with a college

degree or more [57]. These data also show significantly higher serum cotinine levels among children from families with lower annual family incomes and lower householder educational levels even in homes where they did not have exposure to SHS in the home [57]. It is possible that children of disadvantage may not only be disproportionately exposed to SHS inside the home, but perhaps outside the home and in other social environments. Data on SHS exposure is also reported at the national level by home ownership, which is another indicator for SES. SHS exposure declined among both homeowners and renters yet remained significantly higher among renters (see Table 2.6).

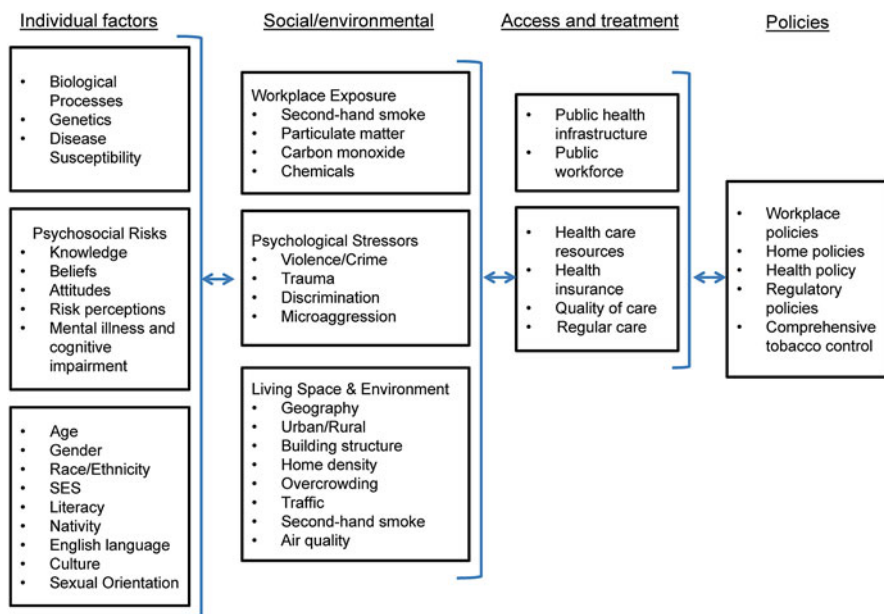
It is clear that SHS exposure is associated with respiratory disease, but whether or not disparities in SHS exposure lead to disparities in respiratory diseases and conditions like middle ear disease, respiratory symptoms, impaired lung function, sudden infant death syndrome, and nasal irritation is not clear. There are genetic variations in CYP2A6, and studies suggest this enzyme can bioactivate tobacco-specific pre-carcinogens including (methyl-nitrosamino)-1-(3pyridyl)-1-butanone (NNK) [75] and *N'*-nitrosonornicotine (NNN) which have been associated with lung cancer [76] in addition to its role in nicotine metabolism. However, it is not clear if CYP2A6 is related to nonmalignant respiratory diseases.

### **Tobacco Causes Respiratory Health Disparities and Populations Impacted by Disparities**

Americans are living longer, and life expectancy has increased for most populations in the USA. In 2012, the life expectancy for all Americans was 79 years according to the World Bank [77]. The USA was only ranked 26th out of 36 countries that are members of the Organisation of Economic Cooperation and Development with respect to life expectancy [78]. Factors such as health care system fragmentation, large uninsured population, socioeconomic conditions, and enormous income inequalities may contribute to relatively modest life expectancy gains in the USA compared to other countries [78]. Notably, respiratory disease is a large contributor to lower life expectancy among Americans, and tobacco use exposure is a major cause of respiratory diseases and conditions in the USA. Other chapters in this book will specifically address health disparities in COPD, asthma, lung cancer, and tuberculosis, which are all related to tobacco exposure with respect to risk and/or exacerbation of disease.

### **Framework for Examining the Problem**

To better understand tobacco-related health disparities among different groups, it is important to have a framework for examining the issue (Fig. 2.5). Asthma is used as an example since it is a chronic respiratory condition associated with smoking as well as individual, social, and environmental factors; access to care and treatment issues; and health policy.



**Fig. 2.5** Framework for examining disparities in tobacco-related respiratory diseases and conditions

Figure 2.5 suggests that there are individual level factors that put smokers and persons exposed to smoke at risk for respiratory diseases and conditions. While research is not well developed in this area, there may be biological processes and differences in disease susceptibility that either increase or decrease the risk for respiratory symptoms among smoking or SHS-exposed asthmatics. Knowledge, attitudes, beliefs, and risk perceptions related to tobacco use may influence personal decisions about smoking or parental decisions to smoke around children, which increases their risk for asthma and exacerbates asthma symptoms. Sociodemographic factors, literacy, nativity, culture, and use of English language may influence help-seeking behaviors related to the treatment of asthma.

Social and environmental factors may influence air quality independent of tobacco smoke exposure, which may further exacerbate asthma. Moreover, social and environmental factors may affect where asthma care is received (e.g., patients may go to emergency rooms to receive care instead of receiving ongoing regular care). Furthermore, policies such as smoke-free multi-unit housing may improve air quality and reduce asthma incidence among children. Persons who rent will benefit since they are disproportionately exposed to SHS exposure. Social and environmental factors and related policies are important to understanding health disparities because minorities are more likely to live in poor environments. For example, African American and Hispanic individuals are more likely than Whites to live in environmental spaces with high levels of air toxins [79]. Minorities are also more

likely to live in communities near freeways and areas with high traffic, which increases their exposure to air toxins [80–82]. To date, the mechanisms by which interactions between environmental exposures and tobacco use affect respiratory disease risk remain poorly understood.

## **Policies to Reduce Tobacco-Related Respiratory Diseases**

Tobacco policies have been implemented primarily to reduce smoking and SHS exposure. For example, clean-indoor air laws and policies in workplaces, restaurants, bars, and other public places; voluntary smoke-free home policies; federal and state tobacco taxes; age of purchase laws; restrictions on advertising and promotion; and youth access restrictions are important components of a comprehensive tobacco control program to reduce tobacco use initiation, increase smoking cessation, and reduce SHS. The Family Smoking Prevention and Tobacco Control Act of 2009 (Public Law 111–31, U.S. Statutes at Large 123) [83] has allowed government, for the first time, to have the authority to regulate a legal but lethal product. Moreover, the Patient Protection and Affordable Care Act (Public Law 111–148, U.S. Statutes at Large 124) [84] passed in 2010 requires insurance companies, including Medicaid, to cover tobacco cessation treatments as a strategy to reduce barriers to access to cessation treatments.

From a societal perspective, it is important to understand not only the impact of tobacco control on trends [85] in smoking and SHS exposure among racial/ethnic and SES minority groups, but also the effects on disease rates as well. For example, the 2014 Surgeon General’s report concluded that there was sufficient evidence to infer that smoke-free laws and policies reduce coronary events in persons younger than 65 years of age. Further investigation is needed to determine if policies can eliminate cardiovascular disease disparities. Lung cancer rates are declining among most racial/ethnic groups [9], but African American males still show the highest incidence and death rates from lung cancer. Further investigation regarding the impact of tobacco policies on respiratory diseases such as lung cancer and COPD is warranted. Understanding how tobacco control programs affect respiratory disease beyond their impact on smoking rates alone will help influence policymaker decisions and governmental strategies to decrease smoking-related health disparities and decrease the overall burden of tobacco.

## **Limitations and Methodological Challenges**

This chapter focuses on disparities in tobacco smoking and smoke exposure among different groups in the USA, examining differences by gender, race/ethnicity, age, and socioeconomic status. However, the evidence available to examine these disparities is limited by several factors. For example, death rates due to smoking-related respiratory

diseases are often reported in aggregate form, and thus it may be difficult to distinguish differences in specific conditions by race/ethnicity or SES. On the other hand, death rates reported in disaggregate form by race/ethnicity may be difficult to generate due to the small sample sizes of many racial/ethnic groups. In general, there is limited information available regarding the relationship between tobacco exposure and respiratory disease and conditions among minority racial/ethnic groups, but there is even less data for Native Americans, Alaska Natives, Native Hawaiians, and Pacific Islanders. While these groups account for less than 2 % of the US population, the limited evidence suggests that disparities exist, and population numbers should not drive the generation of scientific evidence that would facilitate the health of populations, though they may be small in number. In most cases, data for these groups are either not reported or collapsed into a single “other” category. Response rates are also low for national surveys. The National Health Interview Survey, which is used to report adult current smoking annually, had a 61.2 % response rate in 2013 [4]. Non-response can introduce bias and result in under-reporting of smoking rates, particularly among racial/ethnic minority and low SES groups.

We have reported on interactions between gender–race/ethnicity and gender–SES where possible, but these data are often not available for all groups due to small sample sizes. We do not focus on pregnant women as a disparate population in this chapter but believe it is critical to our nation’s health to examine the relationship between tobacco exposure and respiratory illnesses among pregnant women and their children. This review also has limited information on health disparities in LGBT populations since there is little data available at this time. New national data were reported on LGB smoking, but not transgender smoking, for the year 2013. LGB data were not reported by race/ethnicity or SES [4]. Further investigation is warranted on tobacco use and exposure by race/ethnicity, SES, LGBT status, and their associations with tobacco-related disease. Finally, prevalence data on smoking among the mentally ill have recently been reported at the national level, but tobacco-related respiratory diseases and conditions for these groups are not reported at the national level. Thus, our understanding of the impact of smoking among individuals with mental illness relative to the US population at large remains limited.

This report does not focus on smokeless tobacco, cigars of any kind, kreteks, hookahs/waterpipes, pipes, electronic cigarettes/vaporizers, or any other form of tobacco/nicotine, although we report some data for youth. The investigation of how new and emerging products like flavored electronic cigarettes, cigars of any kind, and hookah/waterpipes contribute to respiratory diseases and conditions is critical since the landscape of tobacco use is changing, particularly among young people who may benefit the most from early cessation of these products.



## Directions for Future Research

Approximately 42.1 million Americans smoked in 2013 [4], and we are not likely to reach our Healthy People 2020 goals to reduce cigarette smoking to 12 % among adults. As a result, progress in reducing health disparities for tobacco-related diseases will also likely be delayed. The landscape of tobacco control is changing and has expanded to include more combustibles and also many popular flavored non-combustibles. Such changes may reverse progress made to reduce tobacco use among youth and potentially establish new pathways for disparities.

Moving forward, the power of the Food and Drug Administration Center for Tobacco Products to have a significant impact on tobacco control will depend on their ability to overcome legal and lobbying challenges by the tobacco industry, to circumnavigate the boundaries of operating within the federal government system, and to garner public support for regulatory policies that may benefit public health. The Affordable Care Act may be a game changer for those with the least health care access who are also often those at greatest risk for smoking and tobacco-caused respiratory diseases.

In future research, it is important that we monitor dual and poly-tobacco use and its impact on respiratory diseases and conditions. Young people are more likely than older people to use multiple forms of tobacco along with alcohol, marijuana, and other drugs. About 30 % of young adult cigarette smokers report dual use of tobacco products [86]. Young adults who currently use cigarettes are also at increased risk for electronic cigarette use [87] and are more likely to use flavored little cigars and cigarillos [88–90]. To date, there is limited data regarding the effects of poly-tobacco use on respiratory disease risk and progression. Furthermore, we have limited data on dual and poly use of substances among racial/ethnic minority and low SES groups.

Because national surveys cannot capture sufficient data to report respiratory diseases for Native Hawaiians and Pacific Islanders, Filipinos, Native Americans, Alaska Natives, Asian ethnic, and Hispanic ethnic groups, it is important to collect state and local data that would allow for the accurate reporting of tobacco-related respiratory diseases in these minority groups. These populations are growing, and by 2060, there may be sufficient numbers of different Hispanic ethnic groups to report data at the national level. However, this is unlikely to be the case for other minority groups in the USA. Data can be collected at the national level for LGBT populations and for the mentally ill since there are sufficient numbers of these groups. It is recommended that data on the prevalence and death rates of tobacco-related respiratory diseases should be reported by these factors and by race/ethnicity, poverty status, and gender when possible.

Longitudinal data on tobacco-related respiratory diseases are evolving, but it has been challenging to track how the prevalence and death rates of tobacco-related respiratory diseases are related to tobacco exposure trajectory data among minority racial/ethnic and low SES groups in the USA. In addition to studying prevalence and death rates over time, it is important to examine other health indicators such as hospitalization and quality of life in individuals with tobacco-related diseases.

Furthermore, knowledge, attitudes, and beliefs and health care access and quality may influence disease outcomes related to tobacco exposure among different groups, and therefore warrant further study.

Future research should also examine the differential disease causal pathways of tobacco exposure and increase our understanding of who is at greatest risk for each tobacco-related disease. Tobacco-caused diseases may be consequences of multiple pathways, multiple mechanisms toward those pathways, and the interactions of genes and environmental factors that modulate the activities of the pathways [13]. Understanding these mechanisms can help us better target disease prevention strategies including the implementation of policies that target specific products and product constituents, like nicotine, menthol, and other flavors. A better understanding may also allow us to develop new approaches, such as using biomarkers in early stage disease diagnosis or genetic counseling for smoking cessation programs that specifically seek to eliminate disparities [13].

Further study is needed to understand how socioeconomic status may influence the risk of tobacco-related respiratory diseases and their outcomes and how this intersects with health disparities in racial/ethnic minorities. Cumulative adverse health effects result from living in poverty [91, 92], and poor individuals are more likely to die prematurely than higher income persons [91, 92].

## Summary and Conclusions

In summary, minority racial/ethnic group populations are growing in the USA. It is not expected that the nation will grow healthier with as the population of those who experience health and socioeconomic disparities increases. Understanding how tobacco exposure impacts diseases among these groups is important to the planning of targeted public health initiatives to curtail disease growth with population growth. Forward thinking and planning will also help to reduce health care costs associated with disparities as minority populations in the USA increase in numbers.

Smoking has declined among all racial/ethnic and socioeconomic groups. However, some minority racial/ethnic and low SES groups continue to suffer disproportionately from tobacco use and exposure. These use patterns, however, do not convey disease risk. Lower use of tobacco is not directly associated with lower risk of tobacco-caused chronic conditions. Nor is higher tobacco use associated with higher risk of tobacco-caused illnesses. Tobacco control continues to be a top public health priority, as we know that quitting smoking, reducing the initiation of tobacco use, and eliminating SHS exposure will ultimately reduce tobacco-caused diseases and deaths and improve the quality of life for many Americans. Different strategies may be needed for different groups since declines in smoking despite existing interventions are not equivalent for all groups.

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