CHAPTER 2

Determinants of Individual and Population Health

LEARNING OBJECTIVES

Having mastered the materials in this chapter, the student will be able to:

1. Define health and explain different perspectives on defining health, as well as the complexities involved in assessing health status.
2. Explain how a population is defined from different perspectives and by different stakeholders.
3. Explain the difference between the medical care model and the population health model of healthcare delivery, and the tensions between the two.
4. Explain what different determinants of health are, how they interact with one another, and how they affect the overall health of individuals and populations.

CHAPTER OUTLINE

KEY TERMS

Determinants of health
Environmental heterogeneity
Genetic heterogeneity
Health
Mental health
Population
Population health
Race
Socioeconomic determinants of health

2.1 Introduction

The principles of *integrated healthcare delivery*, *patient-centered care*, and *accountable care* require healthcare providers and managers to think in terms of the whole person rather than a case or *episode* of a disease or a medical event in a person's life. Healthcare providers and managers need to move away from the traditional way of thinking about health and health care that narrowly focuses on treating or managing an episode of disease and meeting the medical care needs of individuals. Rather, they must develop a holistic understanding of the word *health* and fully appreciate the context in which disease or sickness occurs. People experience "poor health" not necessarily because they are afflicted with a malady, but because they do not have the circumstances, resources, and education to lead a healthy and meaningful life.

Healthcare managers need to understand why people get sick in the first place and why they do not seek or receive the right care from an appropriate provider in a timely manner—that is, they need to understand the context in which health care is needed, sought, and given. In this regard, *context* means understanding the relationship between health and socioeconomic conditions in which people are born, live, work, and die. They have to appreciate that health of individuals and communities is directly linked to education, income, and occupation. This chapter is designed to help future healthcare mangers attain such an understanding.

2.2 Definition of *Health*

Signed on July 22, 1946, by the representatives of 61 states and formally adopted by the World Health Organization (WHO) on April 7, 1948, the constitution of the WHO defines **health** as "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity." Though officially unchanged since 1948, the definition was expanded in the 1980s by the leadership of the WHO to include "the ability to lead a socially and economically productive life."[1] The idealistic and unattainable nature of this definition has often been criticized by pragmatists who point toward the fact that health is an elastic concept that can only be assessed indirectly by looking for the presence or absence of disease and disability.[2,3] For the same reason, no direct measure exists to assess a person's "state of complete physical, mental, and social well-being." Many in the healthcare field find the WHO's definition of health to be of limited value and raise the question as to who makes a determination of another person's well-being, as a person can fully enjoy a state of well-being that may be considered unhealthy by someone else. An alternative definition offered by Taber's Cyclopedic Medical Dictionary[4] states that health is "A condition in which all functions of the body and mind are normally active." By implication, this definition recognizes that individuals are constantly responding to external stimuli or stressors that result in a person's overall condition being in a state of flux from day to day, or even from hour to hour. Consequently, the health status of a person cannot be viewed as a static condition; rather, it is a dynamic and ever-changing state on a continuum, with optimum physical, mental, and social functionality constituting the starting point of the continuum, and total loss of functionality being the other end. Given the impermanence or transient nature of one's overall condition, Dubos[5] has suggested that the goal of society is not for everyone to achieve "an ideal state of well-being through complete elimination of disease," but to enable everyone in the society to live a reasonably comfortable and rewarding life.

Because it is impossible for anyone to attain a perfect state of physical, mental, and social well-being, and it is equally difficult for scientists to devise methods for appropriately measuring the health status of individuals and communities, the only practical approach is to measure the level of disease, disability, and death as a proxy measure of health. Measurement of disease, disability, and death in a population at different times or in different populations at any given time allows social scientists to make comparative

statements regarding improvement or deterioration in the health status of a population or disparities in the health status of different populations.

2.3 Definition of a Population

A **population** can be defined from a variety of perspectives. Taber's Cyclopedic Medical Dictionary[6] defines *population* as "1. All people, plants, or animals inhabiting a specified area. 2. The group of people from which a research sample is drawn."

From this perspective, the measurement and reporting of the total number of individuals or the number of people in a group, class, or race are tied to the specification of a geographically defined entity such as the world, a country, or a province. Naturally, any geographically defined entity can be, and usually is, further divided into smaller geographic units such as states, provinces, counties, census tracts, and postal codes. Additionally, the population of any geographically defined entity such as a country or province can be further divided into subgroups on the bases of characteristic such as gender, age, color, ethnicity, education, or religion. It is important to note that no population, even at the smallest geographic level, is perfectly homogenous with respect to any given characteristic such as age, gender, or education.

Because no population is static in time or space, in addition to the specification of a geographic area, the characteristics of a population or its subgroups cannot be discussed without the specification of a period, interval, or date. Hence, any discussion of a population necessitates specification of both a geographic boundary and a period or point in time. For example, we could discuss global population at the end of the 20th century, or the total population of blacks in the United States on December 31, 2017. Similarly, from an epidemiologic perspective, we could discuss the demographic characteristics of a population, such as age and gender distribution of whites in the United States, or birth and death rates per 1,000 Hispanics in the United States at the end of 2017.

Attributes such as age, sex, education, average income, or employment rates in a population are known to be associated with health status and use of health services. In population health studies, the basic unit of analysis and comparison of statistics is usually determined by the objectives of the study. For global and national policy initiatives such as disease control and vaccination or screening strategies, comparisons are made at the country level, whereas allocation of resources within a country or state may require analysis and comparison of data at a district or county level. For example, strategies to reduce teen pregnancy or health promotion initiatives to increase physical activity may require comparisons of teen pregnancy rates and levels of physical activity in various groups at the county level.

In the context of health services planning or healthcare delivery, a population may be defined from the perspective of a healthcare provider, insurer, or payer. For example, a hospital administrator may define *service area*, *service population*, or *catchment population* of the hospital in terms of zip codes and relative proportion of clients in those zip codes served by the hospital. Likewise, an insurance company may define population in terms of the "geographic market" it operates in and its "market share" of the privately insured sector of the population. A healthcare planner or economist, on the other hand, might define a population as the number of Medicaid- or Medicare-eligible individuals in a geographic area. In the United States, America's Health Rankings (http://www.americashealthrankings.org/), a joint initiative of the American Public Health Association and United Health, provides useful state-level comparative health information through its annual reports and issue briefs. The County Health Rankings & Roadmap (http://www.countyhealthrankings.org/), a program of the Robert Wood Johnson Foundation, provides state-by-state county-level data on various health indicators.

2.4 What Is Population Health?

Population health is generally considered as the field of study that examines the health status of populations or groups of individuals. However, there is considerable confusion and debate about the definition and scope of the term *population health*. Some believe that the term relates exclusively to an understanding and measurement of factors such as demographic characteristics, lifestyles and behaviors, genetic makeup, and availability of services that affect the health status of the population. Others put it squarely in the realm of the assessment and measurement of health status or health outcomes,[7] and still others take it as the conceptual framework for understanding why some populations are healthier in comparison with others.[8,9] This definition includes understanding the health outcomes and their distribution within a population or a group of individuals.[7] There is growing emphasis on disease prevention and health promotion by policy makers, third-party payers, and agencies such as the Centers for Medicare and Medicaid Services. As a

result, healthcare managers and providers are becoming increasingly aware of the need for a holistic model of healthcare delivery to achieve population health outcomes and community-based goals, such as those listed in Healthy People 2020.

2.5 Population Health Model Versus Medical Care Model

Historically, healthcare systems all over the world were based on a *medical care model* that put a greater premium on reinstating health through treatment and rehabilitation than maintaining and promoting health by focusing on behavioral and socioeconomic determinants of health. The medical care establishment had focused in the past on meeting the needs of the sick rather than keeping people healthy. In most instances, it continues to do so even today. The medical care model neither was designed for nor rewards community-level investigation and management of factors that lead to ill health and death. Even at the individual level, the focus of healthcare systems all over the world remains, preeminently, on treatment and rehabilitation rather than disease prevention and health promotion. Up until the last few years, healthcare systems in most countries, including the United States, had largely remained disengaged from the epidemics of obesity, smoking, substance abuse, and teen pregnancy that ultimately led to high levels of morbidity and mortality both locally and nationally.

The *population health model*, on the other hand, focuses on developing a safe and health-friendly built environment promoting healthy nutrition through informative labeling of food products, discouraging the availability and consumption of sugary drinks, promoting the availability of opportunities for a physically active life, and providing preventive medical care in the form of vaccinations, periodic screenings, and lifestyle counseling. In contrast to the medical care model, the population health model is neither commonly understood nor fully appreciated by the practitioners of the medical care model. Though the population health model appeals to common sense, the scientific link between socioeconomic factors (such as poverty or lack of education) and health, despite overwhelming empirical evidence, is often regarded by the practitioner community as unproven or beyond the scope of their influence. Research on the effects of stress and anxiety on cardiovascular, endocrine, and neural systems has shown a definite link between hypertension, heart disease, stroke, diabetes, and other disorders, and socioeconomic factors. The pathophysiologic effects of different gradients of these stresses are also well established.

In contrast to the population health model, the medical care model focuses on disease and injury at the individual level and is reactionary rather than preemptive in nature. It comes into action only after the most distal effects of genetic, behavioral, or socioeconomic determinants have become clinically detectable.

2.6 Link Between Physical and Mental Health

There is ample evidence that **mental health** and physical health are interconnected, and the relationship between the two is bidirectional—that is, both affect each other.[10] For example, chronic physical problems, especially those accompanied by chronic pain and loss of function, can also lead to chronic mental health problems, such as depression. Studies have shown that people living with chronic physical problems experience anxiety and depression at twice the rate of the general population. In fact, nearly 50% of patients with chronic pain have been shown to suffer from depression and are reported to be at greater risk of suicidal ideation and suicidal attempts, and have a higher suicide completion rate than the general population.[11-13] **FIGURE 2.1** shows the prevalence of depression in patients suffering from some common chronic physical disorders. It shows, for example, that 27% of patients with diabetes and more than half of Parkinson's disease patients suffer from depression.

Conversely, mental health disorders increase the likelihood of developing a wide range of physical ailments through psychosomatic pathways. Patients with serious mental disorders are known to be at increased risk of asthma, chronic bronchitis, and chronic obstructive pulmonary disease. Psychiatric problems commonly alter eating and sleeping patterns and affect hormonal balance. Stressful life conditions, traumatic experiences, and lack of social support can lead to poor eating habits, lack of physical activity, and alcohol abuse, thus increasing the risk of both physical and mental disorders. Further, the side effects of medication for psychiatric disorders can also result in weight gain and cardiac arrhythmias.[10] As discussed later in this chapter, similar to the association between socioeconomic factors and physical disorders such as hypertension and diabetes, socioeconomic factors are also known to be associated

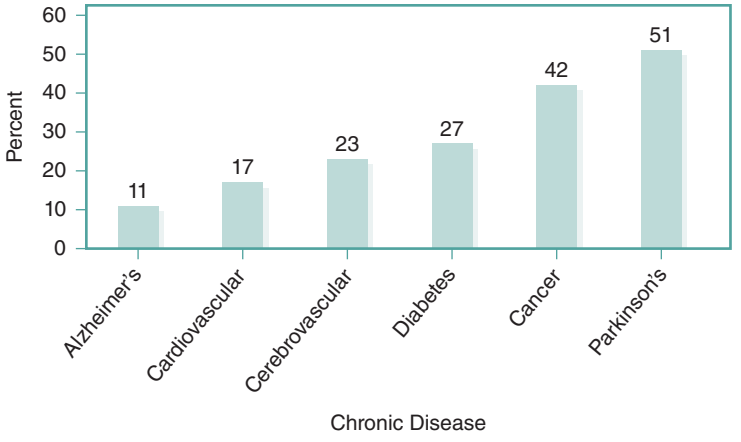

FIGURE 2.1 Prevalence of major depressive disorder in chronic disease.

Reproduced from: Centers for Disease Control and Prevention (CDC), National Center for Chronic Disease Prevention and Health Promotion, Division of Population Health. Mental health and chronic diseases. Issue brief no. 2. October 2012. Accessed on July 21, 2017. https://www.cdc.gov/workplacehealthpromotion/tools-resources/pdfs/issue-brief-no-2-mental-health-and-chronic-disease.pdf

with mental health conditions such as anxiety, panic attacks, and depression.

2.7 Determinants of Health

Health is maintained by the internal regulatory mechanisms of an individual. Therefore, a disease is nothing but the manifestation of a failure of biological functions and internal regulatory mechanisms of the body.[14] A host of variables collectively known as the **determinants of health** can disrupt the internal regulatory mechanisms and biological functions. These variables include both psychological and social factors. As such, determinants of health can be defined as both external and internal factors that directly or indirectly affect the health of individuals and populations. Gradients in these factors and their cumulative or interactive effects ultimately determine whether someone gets sick or remains healthy.[15-17] Over the years, a number of hypotheses have been offered regarding the biologic mechanisms through which various social factors such as education, marital status, social networks, and employment exert influence on the homeostasis or physiologic equilibrium.[16,17]

Historical data have provided convincing evidence that improvements in health and life expectancy observed in Europe in the 18th and 19th centuries were largely the result of rising standards of living and sanitary reforms.[17] In due course, this evidence led to the proposition that health status of a population is closely linked to the physical, social, and economic conditions of the population. Social scientists have investigated pathways through which emotional and psychological states of individuals bring about physiological changes that lead to diseases of different bodily systems.

The term *determinants of health* refers to extrinsic or intrinsic factors that, in a relatively short or long span of time, can affect the health status of individuals. The presence of these factors in varying degrees or their complete absence can make a person sick or can help a sick person recover to normal health. For example, the presence of toxins or pollutants of one kind or another in the air we breathe can make us sick. The nature and degree of the impact of such pollutants on one's health can depend on a variety of factors, including the nature of the agent and the amount present in the air. As an example, consumption of food rich in trans-fatty acids can increase the risk of coronary heart disease or death. However, the risk of coronary heart disease depends on the amount of trans-fatty acids in food consumed every day, as well as the length of time, in terms of month and years, of consuming food containing trans-fatty acids. Conversely, the term *determinants of disease* refers to extrinsic factors, such as microorganisms and chemicals in the environment, or intrinsic factors, such as genetic mutations, that can make us sick. The presence of a certain amount of substances such as minerals, vitamins, fats, and amino acids in our diet is essential for us to remain healthy. Deficiency or excess of these substances over a period of time (chronic deficiency) can make us sick.

A vast body of research conducted over the last few decades provides irrefutable evidence of the effects of genetic, behavioral, socioeconomic, environmental, and healthcare-access-related factors on the health status of individuals throughout the course of one's life, and the interactive or integrative nature of these factors.[18] In the United States since

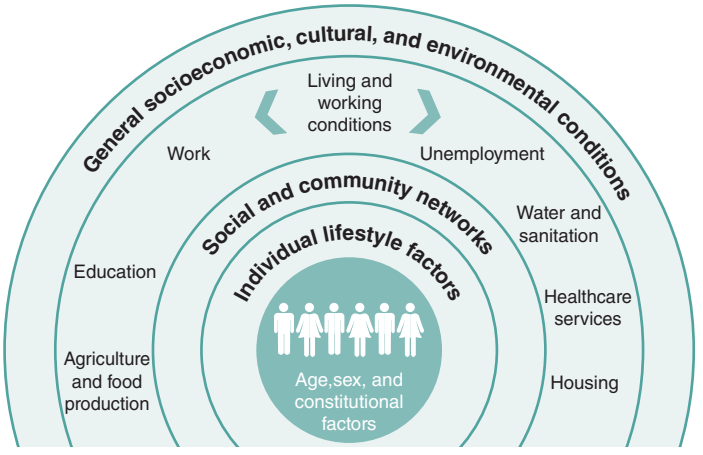

FIGURE 2.2 A guide to thinking about the determinants of population health.

Reprinted from: Dahlgren G, Whitehead M. Levelling up (part 2): a discussion paper on European strategies for tackling social inequities in health, studies on social and economic determinants of population health. WHO Collaborating Centre for Policy Research on Social Determinants of Health. University of Liverpool. Available at: http://www.who.int/social_determinants/resources/leveling_up_part2.pdf. Copyright © 2006.

2008, the Robert Wood Johnson Foundation's Commission to Build a Healthier America has issued a number of reports on how factors outside the healthcare system affect people's prospects to live a long and healthy life. For example, the Commission reported that in 2013, children born to mothers in Arlington and Fairfax Counties, as compared with those born just a few miles away to mothers in Washington, DC, could expect to live 6 or 7 years longer. Even more startling is the finding that average life expectancy for babies born to mothers across neighborhoods in New Orleans can vary by as much as 25 years.

The degree to which various factors, alone or in combination with other variables, influence the health status of an individual is often uncertain and difficult to estimate. However, understanding the role of various determinants is necessary to address disparities in the health status of different populations.[16] Some believe that the effect of isolated social factors accounts for only a small amount of variation in the health status of different individuals.[14] They suggest that the cumulative effect of multiple interactive factors, often referred to as the *allostatic load*, is what triggers the sequence of events that ultimately lead to an adverse health outcome.[19] Based on a review of literature from 1977 to 1993, McGinnis and Foege,[20] in a seminal article, concluded that close to 50% of all deaths in the United States resulted from a few nonmedical factors, of which smoking and diet topped the list. Subsequent assessments by the Institute of Medicine[21] and by the Centers for Disease Control and Prevention not only confirmed the findings reported by McGinnis and Foege but also increased the estimation to 70% of all deaths in the United States and considerably broadened the list of such nonmedical factors.[22]

Tarlov[23] has developed a conceptual framework that classifies various determinant of health into the following five categories: (1) genetic and biologic factors, including age, gender, and race or ethnicity; (2) lifestyles and personal behaviors such as smoking, alcohol use, and sexual behavior; (3) socioeconomic characteristics such as education, income, and social network; (4) physical environment, such as housing, sanitation, and air quality; and (5) degree of access to and quality of health services. **FIGURE 2.2** provides a schematic representation of various determinants of health and their relative importance from core biologic and genetic factors to more peripheral environmental and social factors that directly or indirectly affect health—whether independently or through interaction with other socioenvironmental factors.

2.8 Genetic Makeup as a Determinant of Health

The list of physical and mental disorders that have been linked to a person's genetic makeup is long. [24-26] The 2010 revision of the Nosology and Classification of Genetic Skeletal Disorders by the Nosology Group of the International Skeletal Dysplasia Society listed 456 such conditions in 40 groups on the bases of molecular, biochemical, and/or radiological criteria.[25] It would not be surprising if future research shows that, aside from infectious and nutritional deficiency disorders such as those resulting from the deficiency of vitamins or minerals in diet, most other diseases

have a genetic etiologic component. Even for some of the infectious and deficiency disorders, variance in susceptibility or severity of clinical condition may very well have a genetic explanation.

There is abundant evidence that variations in patients' responses to medication, the rate at which drugs are metabolized, the frequency and severity of side effects, and appropriate dose regimens for different individuals are all related to genetic variations.[27] In fact, between 20% and 95% of variation in metabolism, disposal, and effects of drugs may be the result of genetic variation.[28] For example, some patients with cystic fibrosis, a hereditary lung disorder, have a genetic mutation that allows them to benefit greatly from the drug Kalydeco (ivacaftor), which was developed specifically to target this mutation. Cystic fibrosis patients who do not have this mutation do not respond to the same medication.[29] With the identification of a growing number of oncogenes and the development of drugs that specifically target these genes, cancer care is becoming increasingly customized, personal, and precise. Scientists can now create the genetic profile of a tumor through advanced genomic testing and develop treatment options that are customized for individual patients.

Getting a person's genome sequenced through advanced genomic testing can give useful insights into a person's risk of various diseases and reveal the probability of passing genetic mutations or variants to their children. For example, knowing that a patient carries gene variants that predispose him or her to the risk of sudden death from abnormal heart rhythms such as long QT syndrome can allow preemptive interventional measures. As another example, about 1 in 10,000 to 1 in 15,000 children born in the United States have phenylketonuria (PKU)—a disease caused by an inherited genetic mutation. Untreated PKU can lead to a severe intellectual developmental deficit in children. With early detection through a simple screening test, the disorder can be treated through a strict diet regimen low in phenylalanine, an essential amino acid found in plant proteins.

With the exception of a few diseases, such as Huntington's disease or cystic fibrosis, most genetically linked diseases are not purely determined by the presence or absence of a single genetic mutation or marker.[14] For example, the presence of BRCA1 and BRCA2 mutations is a strong predictor of lifetime risk of breast and ovarian cancer in women. However, empirical evidence shows that only a small proportion of women who develop breast cancer have this mutation. Conversely, not all women with these mutations develop breast cancer—in fact, about 13% to 40% of women with BRCA1 and BRCA2 gene mutations do not develop breast cancer over the course of their lives, and even fewer develop ovarian cancer. Clearly, these mutations are neither a sufficient cause nor the only predictors of breast cancer—that is, there are factors other than the presence of BRCA1 and BRCA2 mutations that predispose women to breast cancer. For example, early onset of menarche, later age of having first child, not breastfeeding, and low fertility are known to increase the risk of breast cancer.[30] In fact, some of these observations now explain why breast cancer was commonly known as the "nuns' disease" or "spinster's disease." Further, there also exist factors that facilitate or suppress the effects or expression of genetic mutations.[14]

The observation that some individuals who are exposed to an environmental, dietary, chemical, or other biological insult develop a disease whereas others do not prompted inquiries into the effect of interactions among genes and the environment. These inquiries and research into DNA repair pathways have shown that there are genetic mutations that can exert a protective effect against risk factors such as carcinogens in tobacco smoke. Most scientists now seem to agree that few diseases are caused purely or exclusively by genes rather than an interaction of genes with the environment that leads to genetic mutations.[31] A number of studies, including some on obesity, sickle cell anemia, and functioning of immune systems, have provided ample evidence to show that the occurrence of diseases and severity of their symptoms are strongly influenced by the interaction of social and genetic factors.[31]

Aside from diseases, such as sickle cell anemia and cystic fibrosis, that result from simple Mendelian inheritance, most genetically linked diseases either occur due to the interaction of multiple genes (gene–gene interaction) or interaction of genes with environmental factors (gene–environment interaction). Variation in the distribution and severity of a disease such as lung cancer results from the combined effect of (1) variation across individuals in exposure to environmental factors—for example, different levels of exposure to tobacco smoke (**environmental heterogeneity**), and (2) variation in the genetic makeup of individuals (**genetic heterogeneity**). The unique combination of the extent of exposure to environmental factors and the personal genetic makeup of an individual (etiologic heterogeneity) not only determines whether a person will develop a disease in the first place, but also affects the level of severity and outcome of the disease.

Genetic variation from one individual to another and within the same individual at different stages of life likely affects all aspects of cellular, biochemical, metabolic, physiologic, and morphologic functions

of the individual and responses to environmental, behavioral, and socioeconomic conditions. Because most diseases have a multifactorial etiology, it is virtually impossible to predict with certainty the likelihood of developing a disease as well as its course, progression, and outcome.[31]

2.9 Socioeconomic Determinants of Health

To better understand the nature and full spectrum of factors that can affect a person or a community's health, it is critically important to raise the question, "Is there truly such a thing as **socioeconomic determinants of health**?" and, if the answer is affirmative, then ask, "How do these factors affect health?"[14] Surveys of self-reported health status by middle-aged individuals in England and the United States have shown a strong negative association between socioeconomic status and diabetes, hypertension, heart disease, myocardial infarction, stroke, lung disease, and cancer in both countries.[32,33] Health disparities were found to be greatest for those at the bottom of socioeconomic status, measured in terms of years of schooling and household income. In both countries, biological markers of disease also showed the same exact patterns of association with the hierarchy of socioeconomic status.[32] Longitudinal studies of the relationship between poor childhood conditions and adult health behaviors and psychosocial characteristics have revealed that men whose parents were poor grew up to have low education levels, hold blue-collar jobs, and demonstrate poor health behaviors.[34] These findings lend further support to the previously observed relationship between health and indicators of social deprivation such as poverty, lack of education, poor nutrition, and certain environmental conditions.

The mechanisms or pathways that link a socioeconomic variable to health outcomes are distinct from those that link other variables such as genetic makeup or quality of care with health outcomes. For example, educational disparities can be linked to gradients in morbidity and mortality through unhealthy lifestyle choices such as smoking, poor nutrition, and obesity. The mechanisms linking income disparity and poverty to gradients in morbidity and mortality, on the other hand, are defined by the ability to afford better housing, food, and clothing, and greater access to health care. Higher income is also associated with positive psychological factors such as a sense of security and control over one's life, decisions, and environment.[31]

Generating convincing evidence of the health effects of socioeconomic and environmental factors is difficult for a number of reasons, including the challenge of bridging the realm of biology with the realm of sociology. It is known that stressful life events such as unemployment, divorce, or death of a loved one set the stage for unhealthy behavioral choices such as smoking and excessive alcohol consumption. However, the neurochemical or neurobiologic pathways through which socioeconomic factors exert influence on the risk of morbidity and mortality are not yet understood. One problem that social scientists encounter is that data regarding stressful life events and socioeconomic variables are gathered at the population level, whereas diseases occur at the individual level.

A number of studies have related endocrine, neural, and physiologic changes in the body to a wide range of stress factors in personal, social, and working environments. More important, these studies have shown that individuals with certain psychosocial and behavioral characteristics, such as introversion, emotional lability, and self-indulgence, are more prone to diseases or illness, including allergies, asthma, and gastrointestinal irritability. In short, such individuals are much more vulnerable to a range of health problems, from reproductive and gynecologic problems to bacterial and viral infections.[35]

Though neuroendocrine and biochemical pathways are not clear, there is compelling evidence of physiologic changes in human body in response to social stressors. Increased heart rate, perspiration, dilatation of blood vessels in the skin and muscles, and changes in the gastrointestinal and urinary systems have been noted during and after stressful events.[17,35,36] The direct and indirect relationships between stressors and health outcomes are complex. The direct effect is in the form of psychophysiologic changes that lead to increased blood lipid levels, abdominal obesity, high blood pressure, insulin resistance, and increased levels of C-reactive protein. These changes lead to increased risk of heart disease, stroke, and diabetes.[36] The indirect effects of a stressful environment or life situation occur in the form of unhealthy coping strategies such as smoking, substance abuse, alcohol dependence, and the secondary effects of mood alterations and insomnia resulting from stress.[37,38]

In two longitudinal studies known as Whitehall I and Whitehall II, which involved studying British civil servants, Marmot and colleagues[39-40] collected extensive longitudinal data from more than 27,000 individuals. Demographic, socioeconomic, and health-related data were collected in multiple phases from British civil servants of different ranks and income levels over a period of more than four decades. Follow-up with participants in the Whitehall II study has continued since 1985. The results of these studies have shown that individuals at the lowest rank and income level

were 3.5 times more likely to die than those at the top. Further, the inverse relationship between social standing and mortality held strong at all levels of comparison. The inverse relationship also remained consistent for specific causes of death, such as heart disease, stroke, suicide, and lung cancer. Moreover, the difference in mortality between different ranks remained even after adjusting for differences in risk factors such as smoking and having high blood pressure and high cholesterol.

Marmot and his colleagues[39,41] discovered that the blood pressure of those at the top of the hierarchy was much lower at home after returning from work, whereas it was higher and remained higher for much longer among those at the lowest ranks.[42] Other studies on the effects of stress factors such as job loss, bankruptcy, social isolation, and discrimination have shown similar negative effects of stress on cardiovascular, endocrine, neural, and immunologic systems and increased risk of death and disease.[43,44] Socioeconomic factors such as income, education, employment, and social support are sometimes called the "upstream" factors because they directly affect "downstream" living conditions, including housing, nutrition, lifestyle, and levels of stress.[7,18] Clearly, a particular socioeconomic variable may not be independently sufficient to affect health outcomes, but in concert with other factors, it can set the stage for better or poorer health outcomes. From a health policy standpoint, it is important to bear in mind the interactive nature of the effects of socioeconomic factors and devise longitudinal interventions that simultaneously target multiple factors.

An interesting research finding is that women of higher socioeconomic standing are at a greater risk of breast cancer. However, the increased risk of breast cancer among women of higher socioeconomic standing is partly explained by reproductive factors, such as age at menarche, age of mother at first childbirth, and low fertility. Additionally, women of higher socioeconomic standing enjoy a much better chance of surviving breast cancer because of earlier detection and greater access to effective treatment.[45-47] It is worth repeating that breast cancer was historically known as the "nuns' disease" because of the known fact that nuns experienced much higher rates of breast cancer than did other women. Now it is understood that pathophysiologic pathways of breast cancer are directly linked to hormonal levels in the body. Hormonal changes related to pregnancy, fertility rates, and lactation confer a degree of protection against breast cancer.[46,47]

2.9.1 Income as a Determinant of Health

Accurate measurement of income is difficult for a variety of reasons, including the challenge of specifying the reporting time frame, sources of income, and units of measurement; determining whether reported income is for an individual or a household; and clarification regarding gross or disposable income.[31] Nonetheless, a large number of studies have documented a positive correlation between income and health status. Poverty affects the health and development of individuals from embryonic stages all the way to old age. This happens through interactive effects of exposure to environmental elements, poor nutrition, inadequate housing, lack of access to sanitation and safe drinking water, lack of access to good education, and lack of access to age-appropriate disease-preventive and curative health services.[31]

Results from the Panel Study of Income Dynamics, the longest running longitudinal U.S. household survey, which began in 1968, have shown a 3.6-fold mortality risk difference between working-age adults in the top posttax family income bracket of >$70,000 (in 1984 dollars) and those in the bottom income category of <$15,000 per year.[48] Similar to the effects of different levels of education attainment on health, the relationship between better health and higher income displays a gradient with successively higher levels of income. This gradient is steepest at lower income levels and plateaus at income levels that are twice the median income.[31] Further, the relationship between income and health is reciprocal in nature—that is, those in better health have better prospects of making more money, and those with higher income can afford, and display, healthier lifestyles.[49] In fact, poor health, one of the most common causes of job loss, results in a spiral of worsening economic deprivation and deteriorating psychosocial health. The hypothesized causal pathways of the income–health relationship are based on the greater ability of more affluent people to buy goods and services and having greater sense of security or peace of mind.[31]

FIGURE 2.3 provides a conceptual model of how parents' income can shape families' options for higher standards of living and children's prospects for better education, employment, and future income, as well as health status during the course of their entire life. The 2014 Robert Wood Johnson Foundation report titled "Time to Act: Investing in the Health of Our Children and Communities" suggests that "parents' income can affect children's chances for health by shaping options for living conditions and educational chances, which in turn shape their income and living conditions as adults." As shown in **FIGURE 2.4**, income gradients have a marked effect on health status within and across racial or ethnic groups. For instance, among blacks, only 6.8% of those with family income equal to or greater than 400% of federal

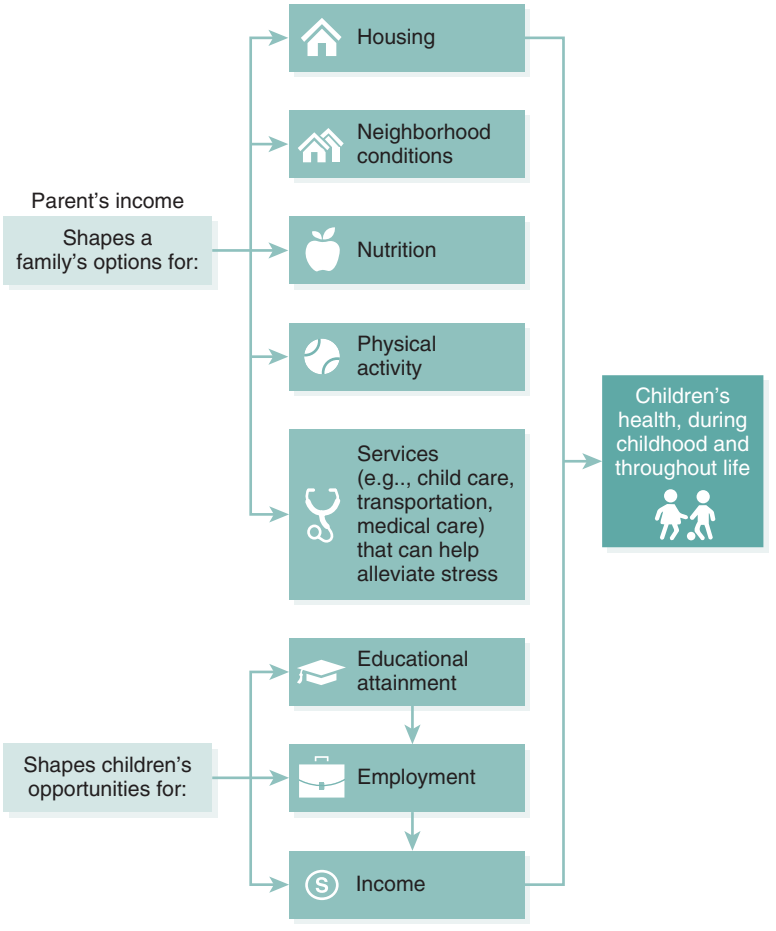

FIGURE 2.3 Parents' income can affect a child's chances for health throughout life.

poverty level in 2010 reported being in poor or fair health, as opposed to 23.9% of those in the family income bracket of less than 100% of the federal poverty limit.

2.9.2 Education as a Determinant of Health

Educational attainment in terms of the number of years of education has consistently been a good predictor of future health behaviors and health outcomes, including future morbidity and mortality not only of individuals themselves, but their children as well. Increasing levels of infant mortality have been observed with successively lower levels of educational attainment of mothers—that is, a gradient exists in the association of health outcomes and educational attainment.[31] The "totality of evidence" in this regard suggests a direct causal link between education and health outcomes. Part of the evidence comes from "natural experiments" that occurred in the form of United States legislation, passed in different localities at different times, making school education compulsory; as schooling progressively became compulsory, health outcomes improved successively.[50] Randomized trials of preschool education also have been linked with reduced teen pregnancy rates when these children became adolescents and young adults.[51] The hypothesized causal pathways to explain the relationship between higher levels of schooling and future health outcomes include adoption of healthy lifestyles due to awareness, acquisition of health-related knowledge ("health literacy"), and better ability to "navigate the healthcare system."[31]

In a meta-analysis of 47 different studies that examined the health effects of sedentary time independent of all other factors, Biswas et al.[52] found that greater sedentary time was positively associated with increased risk of "all-cause mortality," cardiovascular disease incidence and mortality, cancer incidence and mortality, and incidence of type 2 diabetes. The greatest effect of sedentary time was on increased risk of type 2 diabetes. With regard to increased risk of cancer, specific associations of sedentary time were identified with colorectal, breast, endometrial, and epithelial ovarian cancer.

FIGURE 2.5 shows the effect of different levels of education on life expectancy. According to the data from the U.S. National Center for Health Statistics on which this figure in the Robert Wood Johnson Foundation 2014 report is based, on average, 25-year-old college graduates have a life expectancy 8 or 9 years longer than their counterparts who did not finish high school. Similarly, education is also linked with better health across racial or ethnic groups. As shown in **FIGURE 2.6**, across all racial groups, 42% to 50.7% of those 25- to

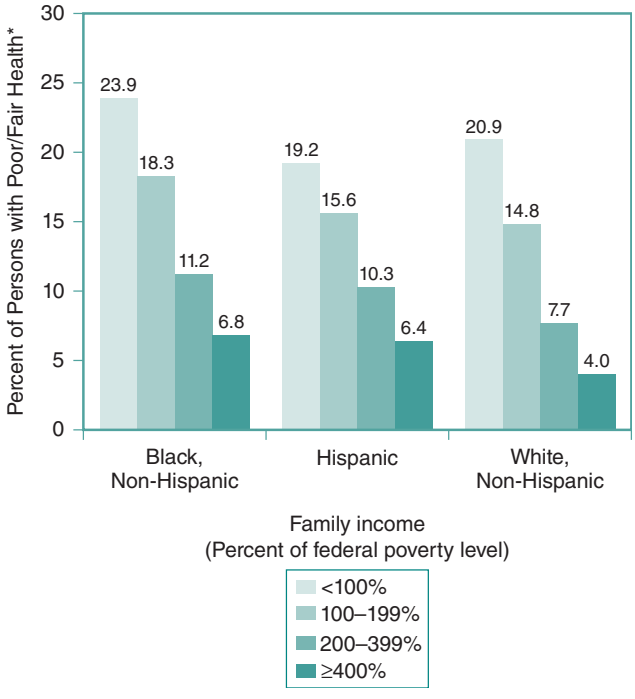

FIGURE 2.4 Income is linked with health across racial and ethnic groups.

* Age-adjusted. Based on self-report and measured as poor, fair, good, very good, or excellent.
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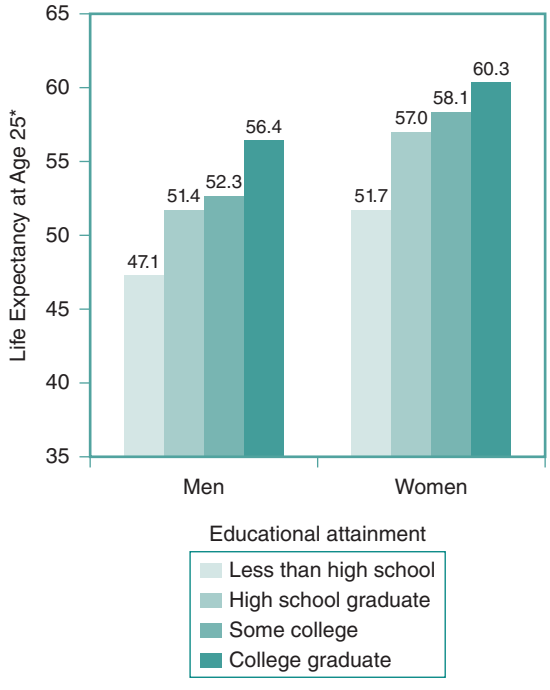

FIGURE 2.5 More education, longer life.

* This chart describes the number of years that adults in different education groups can expect to live beyond age 25. For example, a 25-year-old man with a high school diploma can expect to live 51.4 additional years and reach an age of 76.4 years.
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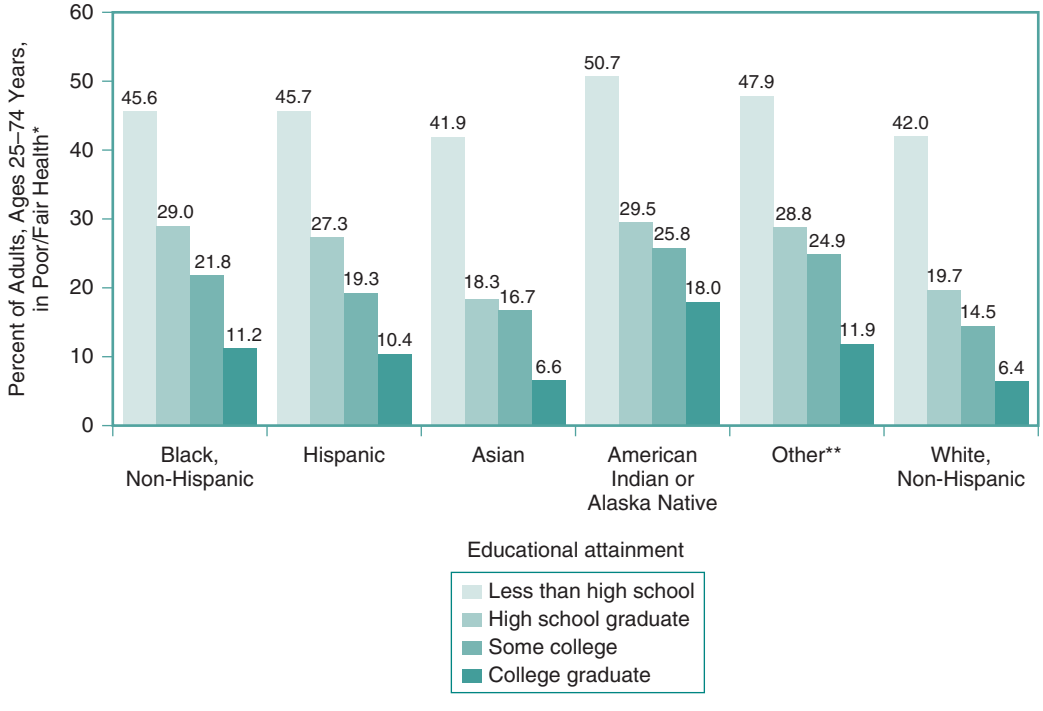

FIGURE 2.6 Education is linked with health across racial or ethnic groups.

* Age-adjusted. Based on self-report and measured as poor, fair, good, very good, or excellent.
** Defined as any other or more than one racial or ethnic group, including any group with fewer than 3 percent of surveyed adults nationally in 2008–20.
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74-year-olds who had less than a high school education reported being in poor health, whereas only 6.4% to 11.2% of those with a college degree reported being in poor health.

2.9.3 Occupational Status as a Determinant of Health

Occupational status is one of the legs of a three-legged stool called "socioeconomic status"; income and education constitute the other two legs. Broadly, occupational status represents the level of authority, prestige, money, and power not just in the labor market, but in the overall society as well. There are three different aspects to the relevance of occupational status with health status. First, the extent to which an occupation exposes a person to the risk of physical injury, including injury from falls and exposure to heat, cold, or chemical toxins. The second consists of the psychosocial aspects of a person's work environment, including the degree of job security, level of stress, and latitude in decision making. The third aspect of the relationship between occupational status and health relates to prestige and symbols of power that have an impact on the emotional and psychological health of the individual.[31]

A number of theoretical and methodological frameworks have been developed to measure occupational status.[53] For example, one way of classifying occupational status is based on manual versus nonmanual (blue-collar vs. white-collar) work.[54] Historically, blue-collar jobs, or manual work, have been associated with low prestige, power, and money, and greater health hazards. Cross-sectional and longitudinal studies on the conditions of mineworkers, construction workers, and factory workers support this assertion. An alternative approach to the classification of occupational status is Duncan's Socioeconomic Index (SEI).[55,56] Duncan's SEI combines subjective measurements of prestige with objective measurements of income and education. Higher scores on SEI have been linked with lower scores on self-reported physical, mental, and social health.[40] Similar to the effects of income and education on health, the relationship between occupational status and health is also bidirectional—that is, poor health poses a serious hindrance to achieving upward social mobility through attainment of higher occupational status.

Conversely, low occupational status can lead to poor physical, mental, and social health.[31] **FIGURE 2.7** shows differences in life expectancy for

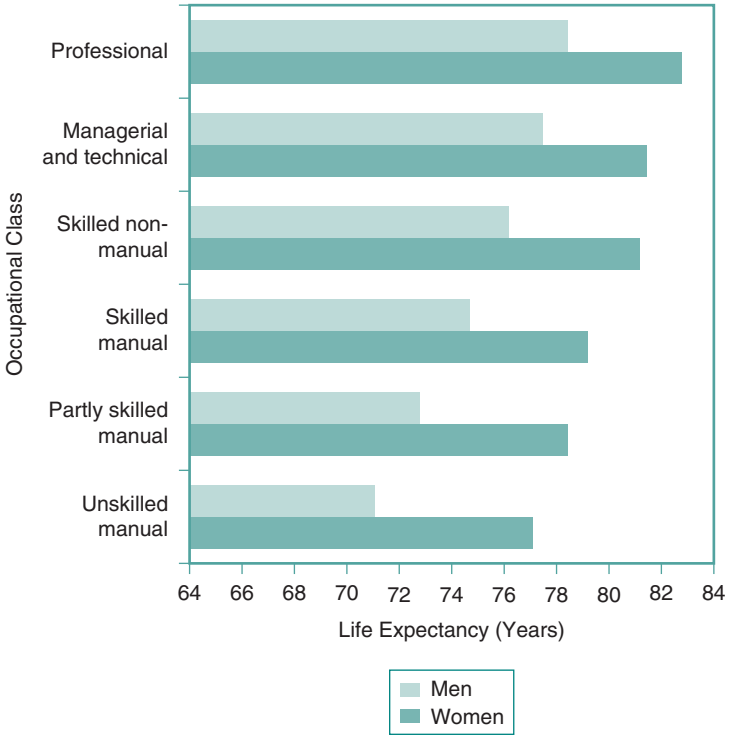

FIGURE 2.7 Occupational class differences in life expectancy, England and Wales, 1997–1999.

Original Source, Donkin A, Goldblatt P, Lynch K. Inequalities in life expectancy by social class 1972–1999. Health Statistics Quarterly. 2002;15:5–15. Secondary Source: World Health Organization: Europe. Social determinants of health: the solid facts. 2nd edition. Wilkinson R, Marmot M. (eds). 2003. http://www.euro.who.int/__data/assets/pdf_file/0005/98438/e81384.pdf © World Health Organization. 2003.

men and women in different occupational classes in England and Wales between 1997 and 1999. Although women in each occupational class have a longer life expectancy than men, both men and women in "unskilled manual" occupations, on average, have a life expectancy 6–8 years shorter than those in the "professional" class. The life expectancy gradient from one occupational class to the next consistently favors those in the successively upper occupational stratum.

2.10 Race as a Determinant of Health

In literature, the terms *race* and *ethnicity* are frequently used interchangeably and capture common geographic origins, ancestry, language, traditions, and cultural norms of a group of people.[18] In social science research, racial categorization has been used to reflect oppression, social inequality, and lack of opportunity for one group as compared with another.[57]

Rather than any evidence of intrinsic differences in biochemical, physiologic, or genetic makeup, the construct of **race** is based on the pigmentation of skin and other physical traits, as well as geographic distribution of people: African, Hispanic, Caucasian, Native American, Asian, Pacific Islander, and so on. Historically, racial categorization was largely used to imply differences in genetic makeup. However, genetic profile data unequivocally show that there is far greater genetic variation within racial groups (particularly among Africans) than exists between different racial groups such as Africans versus Caucasians. "Although race is still used as a label, the original concept of race as a genetically distinct subspecies of humans has been rejected through modern genetic information. . . . It is more appropriate to reconceptualize the old genetics of race into a more accurate genetics of ancestry."[31]

Outside of genetic predisposition to certain diseases, such as sickle cell anemia or thalassemia, the problem with employing race as a determinant of health is that it is a social construct for which we have no knowledge of how or whether, in and of itself, race predisposes a group of people to better or poorer health.[14] The only empirical evidence we have is that of a statistical association between race and disease prevalence or health outcomes such as hypertension or diabetes, or survival rates from various cancers. However, these associations cannot be isolated from, and can be explained on the bases of, gradients in socioeconomic factors. The cumulative effect and hemodynamic responses to the stress of social isolation, unemployment, poverty, and resentment of one's circumstances provide the physiologic bases of gradients in hypertension and diabetes.[19] Passive coping strategies to the stress of social isolation and other socioeconomic stressors also set the stage for poor behavioral choices, including excessive eating, drinking, and use of illicit drugs. The long-term health effects of these choices ultimately lead to obesity, diabetes, heart disease, psychiatric problems, and violence.[58]

Significant disparities in health and health care exist in the U.S. healthcare system between different racial groups. A disparity in health refers to differences in the burden of disease, death, and disability, whereas a disparity in health care relates to differences between groups in terms of access to services, quality of care received, and health outcomes for the same services and procedures. Minority individuals continue to have less access to health care, receive poorer quality care, and have worse health outcomes as compared with the white population. Although some of these disparities are explained by differences in the socioeconomic status of white and minority populations, notable disparities persist within the same socioeconomic strata in the same geographic districts. Black, Hispanic, Native, and Asian Americans, for example, face greater barriers to access, receive fewer services such as coronary artery bypass graft or perinatal care, and have worse outcomes for the same procedures as compared with their white counterparts of the same education and income levels. These disparities cost the nation approximately $35 billion in excess health care, $10 billion in lost productivity, and $200 billion in premature deaths every year.[59] Some of the differences in access and use of services are also linked to a historic distrust in minority population of government and healthcare providers. Designation by the federal government of various groups, such as people of color, the elderly, women, and children, as "priority population" attempts to reduce disparities in both health and health care.

In the chapter entitled *Descriptive Epidemiology*, we provide more information on the epidemiologic importance of genetically linked disorders found more commonly in various groups, such as African Americans or Ashkenazi Jewish women, and the categorization of various racial groups by the U.S. Census Bureau.

CASE STUDY 2.1: Effect of Education on Health

Extracted from: Rogers RG, Everett BG, Zajacova A, Hummer RA. Educational degrees and adult mortality risk in the United States. Biodemography and Social Biology. 2010;56(1):80–99.

In this 2010 study published in the journal *Biodemography and Social Biology,* Rogers et al. estimated the risk of mortality in U.S. adults aged 25 years or older by educational levels. The authors used the 1997–2002 National Health Interview Survey Linked Mortality Files and Cox proportional hazards modeling statistical analyses to estimate the risk of mortality for individuals with different levels of educational attainment. The study included data on 184,499 individuals 25 years or older who had participated in the National Health Interview Survey. The National Death Index matching criteria showed that 8,994 of these individuals had died since the survey. Based on the date of birth, the survey participants were divided into four cohorts: Good Warriors (born between 1909 and 1928), Lucky Few (born between 1929 and 1945), Baby Boom (born between 1946 and 1964), and Generation X (born between 1965 and 1982). **TABLES 2.1**, and **2.2**, present some of the results of the study. In Tables 2.1 and 2.2, a hazard ratio of 1.0 indicates that there is no relationship between the variable of interest, such as sex, race, or marital status, and the risk of mortality. A hazard ratio of greater than 1.0 suggests a heightened risk of mortality in comparison with the reference category. For example, a hazard ratio of 1.26 would suggest that the specific variable (e.g., having only a bachelor's degree) is associated with a 26% increased risk of mortality during the follow up period as compared with the reference category. Conversely, a hazard ratio of less than 1.0 means a reduced risk of mortality in comparison with the reference group. The statistical models developed in this study controlled for the effects of race/ethnicity on the risk of mortality (Model 1 in Table 2.1 and all models in Table 2.2) as well as the effect of marital status (Model 2 in Table 2.1 and all models in Table 2.2). The authors concluded that in comparison to adults who had a professional degree, those with a bachelor's degree, those with some college education, those with a high school diploma, and those with GED or ≤12 years of schooling were 26%, 65%, 80%, and 95%, respectively, more likely to die during the follow-up period. The heightened risk of mortality with lower educational attainment varied by gender and age cohort.

Questions

Question 1. In Table 2.1, as compared with those with a high school diploma, how much higher or lower is the risk of mortality for those with less than 12 years of education? What about those with a professional degree?

Question 2. In Table 2.1, as compared with Generation X, were the Good Warriors at a higher or lower risk of mortality? Explain your answer.

Question 3. In Table 2.2, as compared with those with a high school diploma, how much higher or lower is the risk of mortality for the Baby Boom and Generation X cohorts with fewer than 12 years of education? What is the risk of mortality for Baby Boom and Generation X individuals with a MA/PhD/professional degree?

TABLE 2.1 Hazard Ratios of Educational Degrees and Mortality Risk, U.S. Adults Aged 25 and Older, 1997–2002

	Model 1		Model 2	
	H.R.	S.E.	H.R.	S.E.
Education (High school diploma; reference category)				
Less than 12	1.21 ***	(0.04)	1.18 ***	(0.04)
Grade 12	1.07	(0.08)	1.09	(0.08)
GED	1.11	(0.08)	1.09	(0.08)
Some college	0.92 *	(0.03)	0.92 *	(0.03)
AA	0.80 ***	(0.04)	0.80 ***	(0.04)
BA	0.70 ***	(0.03)	0.70 ***	(0.03)

MA	0.59 ***	(0.05)	0.59 ***	(0.05)
Prof. degree (MD, DDS, JD, DVM)	0.55 ***	(0.07)	0.56 ***	(0.07)
PhD	0.60 ***	(0.08)	0.61 ***	(0.08)
Sociodemographic				
Male	1.58 ***	(0.04)	1.69 ***	(0.01)
Cohort (Generation X, born 1965–1982; reference category)				
Baby Boom, born 1946–1964	1.14	(0.31)	1.17	(0.30)
Lucky Few, born 1929–1945	1.11	(0.34)	1.12	(0.33)
Good Warriors, born 1909–1928	1.14	(0.36)	1.16	(0.34)
Race/Ethnicity (Non-Hispanic white; reference category)				
Non-Hispanic black	1.34 ***	(0.05)	1.26 ***	(0.05)
Hispanic	0.90	(0.06)	0.89†	(0.06)
Other	1.06	(0.07)	1.05	(0.07)
Marital Status (Married; reference category)				
Widowed			1.26 ***	(0.04)
Divorced/separated			1.48 ***	(0.06)
Never married			1.64 ***	(0.07)

Model 1 controlled for race/ethnicity; Model 2 controlled for both race/ethnicity and marital status.
†p ≤ .10. *p ≤ .05. **p ≤ .01. ***p ≤ .001.
Abbreviations: H.R. = hazard ratio; S.E. = standard error.

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TABLE 2.2 Hazard Ratios of Educational Degrees and Mortality Risk by Cohort and Sex, U.S. Adults Aged 25 and Older, 1997–2002

	Good Warriors (born 1909–1928)		Lucky Few (born 1929–1945)		Baby Boom & Gen X (born 1946–1982)	
	H.R.	S.E.	H.R.	S.E.	H.R.	S.E.
Males						
N	9,988		19,327		56,767	
Died during follow-up	2,495		1,391		764	

(continues)

TABLE 2.2 Hazard Ratios of Educational Degrees and Mortality Risk by Cohort and Sex, U.S. Adults Aged 25 and Older, 1997–2002 *(continued)*

	Good Warriors (born 1909–1928)		Lucky Few (born 1929–1945)		Baby Boom & Gen X (born 1946–1982)	
	H.R.	S.E.	H.R.	S.E.	H.R.	S.E.
Education (High school diplomo used as reference group)						
Less than 12	1.18 **	(0.06)	1.40 ***	(0.10)	1.51 ***	(0.19)
Grade 12	1.15	(0.15)	1.35†	(0.24)	0.97	(0.25)
GED	1.17	(0.17)	1.34 *	(0.18)	1.41 *	(0.25)
Some college	0.95	(0.07)	0.98	(0.09)	0.90	(0.10)
AA	0.80 *	(0.09)	0.96	(0.13)	0.84	(0.12)
BA	0.75 **	(0.07)	0.64 ***	(0.08)	0.46 ***	(0.06)
MA/PhD/Prof. degree	0.69 ***	(0.07)	0.56 ***	(0.10)	0.31 ***	(0.07)
Race/Ethnicity (Non-Hispanic white used as reference group)						
Non-Hispanic black	0.98	(0.07)	1.20 *	(0.12)	1.53 ***	(0.16)
Hispanic	0.76 *	(0.10)	0.89	(0.16)	1.37 *	(0.19)
Other	0.94	(0.14)	0.93	(0.21)	1.59 *	(0.30)
Marital Status (Married used as reference group)						
Widowed	1.19 ***	(0.06)	1.46 **	(0.17)	3.29 ***	(1.07)
Divorced/separated	1.29 *	(0.14)	1.68 ***	(0.13)	1.90 ***	(0.20)
Never married	1.16	(0.13)	2.01 ***	(0.23)	2.44 ***	(0.25)
Females						
N	14,472		21,573		62,372	
Died during follow-up	2,698		1,059		587	
Education (High school diploma used as reference group)						
Less than 12	1.10 *	(0.06)	1.47 ***	(0.13)	1.44 *	(0.23)
Grade 12	1.00	(0.13)	0.87	(0.18)	1.55 *	(0.33)
GED	0.52 *	(0.15)	1.04	(0.24)	1.03	(0.27)

Some college	0.95	(0.07)	0.79 *	(0.09)	1.00	(0.13)
AA	0.85	(0.09)	0.71 *	(0.11)	0.73†	(0.13)
BA	0.89	(0.08)	0.91	(0.12)	0.66 **	(0.13)
MA/PhD/Prof. degree	0.82	(0.11)	0.60 **	(0.11)	0.56 **	(0.12)
Race/Ethnicity (Non-Hispanic white used as reference group)						
Non-Hispanic black	1.14 *	(0.07)	1.47 ***	(0.13)	1.55 ***	(0.17)
Hispanic	0.87	(0.12)	0.56 **	(0.11)	1.02	(0.19)
Other	0.81	(0.16)	1.24	(0.24)	1.09	(0.26)
Marital Status (Married used as reference group)						
Widowed	1.23 ***	(0.06)	1.26 **	(0.10)	1.34	(0.37)
Divorced/separated	1.20†	(0.12)	1.34 **	(0.12)	1.75 ***	(0.19)
Never married	1.28 *	(0.14)	1.64 ***	(0.23)	1.55 **	(0.22)

†$p \leq .10$; *$p \leq .05$; **$p \leq .01$; ***$p \leq .001$.
Abbreviations: H.R. = hazard ratio; S.E. = standard error.

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CASE STUDY 2.2: Effect of Socioeconomic Status on Health

Extracted from: Banks J, Marmot M, Oldfield Z, Smith PJ. Disease and disadvantage in the United States and in England. JAMA. 2006;295(17):2037–2045.

In a 2006 seminal study, Banks et al. examined the relative health status of older individuals in England and the United States in relation to their socioeconomic status. The researchers used 2002 data from the U.S. Health and Retirement Survey (n=4,386) and the English Longitudinal Study of Aging (n=3,681) as representative samples of individuals aged 55 to 64 years in both countries. They supplemented their analysis with samples of individuals aged 40 to 70 years from the 1999–2002 waves of National Health and Nutrition Examination Survey (n=2,097) and the 2003 wave of the Health Survey from England (n=5,526). To ensure integrity of results untainted by health status difference among white, black, and Hispanic populations, the study was limited to only non-Hispanic white populations in the two countries. Age and health behavior–adjusted self-reported prevalence of diabetes, hypertension, heart disease, stroke, myocardial infarction, lung disease, and cancer were used as indicators of health status. These indicators were compared across different education and income groups in the two countries.

Overall, the results showed that the U.S. middle-aged population was less healthy than the comparable English population. Within each country, a clear negative socioeconomic gradient for self-reported health status was observed. Adjusting for risky health behaviors such as smoking, being overweight, being obese, and abusing alcohol, health status disparities were greatest for those at the bottom of the education and income hierarchy. Diabetes was noted to be twice as high in the U.S. population of 55- to 64-year-old individuals as compared with the same-age English population. As only one fifth of the difference in the health status of the two populations was explained by a difference in health-related behaviors of the two populations, the difference in health status was deemed real and unexplained by differences in health-related behaviors. Similarly, average levels of C-reactive protein were 20% higher, and average levels of high-density lipoprotein cholesterol were 14% lower, in the U.S. population of 55- to 64-year-olds.

(continues)

TABLE 2.3 shows the percentage of 55- to 64-year-olds in each education and income category in the two countries who reported being smokers, drinking heavily, and being overweight or obese. **TABLE 2.4** shows that in both countries, even after adjusting for health-related personal behaviors such as smoking, abusing alcohol, and being obese, for all diseases, with the exception of cancer, a steep negative health gradient existed between different educational and income groups, with less educated and low-income individuals being worse off than their more educated and economically well-off counterparts.

Questions

Question 1. Based on the data presented in Table 2.3, across different education and income categories, were there any consistent patterns in the distribution of risk factors in the populations of the two countries? Explain your answer.

Question 2. Based on the data presented in Table 2.3, were middle-income Americans more or less obese than middle-income English 55- to 64-year-olds? How confident can one be in making this assertion? Explain your answer.

Questions 3. Based on the data presented in Table 2.3, were "current smoker" patterns across low-, medium-, and high-education groups different between the English and American 55- to 64-year-olds? Explain your answer.

Question 4. Based on the data presented in Table 2.4, what conclusions can you draw about the occurrence of stroke across education and income categories in the English and American populations?

Question 5. Based on the data presented in Table 2.4, what conclusions can you draw about the occurrence of cancer across education and income categories in the English and American populations?

TABLE 2.3 Prevalence of Self-Reported Risk Factors in England and the United States, Ages 55–64 Years*

Self-Reported Risk Factor	England				United States			
	Low	Medium	High	Total	Low	Medium	High	Total
Years of schooling, percent distribution								
Current smoker	28.6	18.2	13.3	21.9	24.9†	20.5	11.4	20.1
Ever smoked	69.1	64.0	62.6	66.1	64.5‡	65.0	54.8‡	61.9‡
Obese	26.5	20.9	18.6	23.0	33.6‡	34.5‡	24.0‡	31.1‡
Overweight	38.8	42.4	43.2	40.9	38.2	37.8	40.5‡	38.8
Heavy drinker	21.8	32.8	42.2	30.0	10.6‡	13.2‡	21.9‡	14.4‡
Income, percent distribution								
Current smoker	28.6	22.2	15.2	21.9	26.9	21.8	11.6†	20.1
Ever smoked	69.1	65.8	63.4	66.1	66.1	62.6	56.9‡	61.9‡
Obese	25.3	23.2	20.5	23.0	35.6‡	32.9‡	24.8†	31.1‡
Overweight	38.9	41.8	42.1	40.9	35.8	39.0	41.4	38.8
Heavy drinker	22.6	26.2	40.6	29.9	8.7‡	14.3‡	20.2‡	14.1‡

*Extracted from: English data are from first wave of English Longitudinal Survey of Aging, and U.S. data are from the 2002 wave of the Health and Retirement Survey. See Table 2.4 for sample sizes and definitions of income and education groups. All data are weighted.

†P<.01 for comparison of United States and England

‡P<.05 for comparison of United States and England

TABLE 2.4 Adjusted Self-Reported Health by Education and Income in England and the United States, Ages 55–64 Years*

Self-reported Disease	England				United States			
	Low	Medium	High	Total	Low	Medium	High	Total
Years of schooling, percent distribution								
Diabetes	7.7	6.2	7.4	7.2	13.9†	11.9†	10.6‡	12.5†
Hypertension	37.6	32.9	32.5	35.1	46.0†	40.2†	38.0‡	42.4†
All heart disease	12.2	8.3	7.9	10.1	17.1†	14.9†	11.9	15.1†
Myocardial infarction	4.8	4.0	3.3	4.2	6.7‡	4.2	4.3	5.4‡
Stroke	2.7	2.3	1.8	2.3	4.7†	4.1‡	2.0	3.8†
Lung disease	7.7	5.4	4.3	6.2	10.4†	7.9‡	4.4	8.1†
Cancer	4.9	5.3	6.5	5.4	8.8†	9.7†	10.5†	9.5†
Income, percent distribution								
Diabetes	8.1	7.7	6.0	7.2	16.8†	11.4†	9.2†	12.5†
Hypertension	37.9	35.8	31.6	35.1	46.1†	42.8†	38.2†	42.4†
All heart disease	14.3	9.1	6.9	10.1	20.2†	13.1†	12.1†	15.1†
Myocardial infarction	6.7	3.3	2.5	4.2	8.6	4.3	3.3	5.4‡
Stroke	3.5	1.9	1.6	2.3	5.8‡	3.7†	1.8	3.8†
Lung disease	7.6	6.3	4.8	6.2	12.3†	7.0	5.1	8.1†
Cancer	5.7	5.1	5.5	5.4	9.3†	9.8†	9.5†	9.5†

*Ordinary Least Squares regression models adjusted to reflect what health conditions would be if all individuals in both countries had the same level of behavioral risk factors as the average American in that age group. Regression coefficients are country specific.

Extracted from: English data are from the first wave of the English Longitudinal Survey of Aging, and U.S. data are from the 2002 wave of the Health and Retirement Survey. See Table 2.1 for sample sizes and definitions of income and education groups. All data are weighted.

†P<.01 for comparison of United States and England

‡P<.05 for comparison of United States and England

2.11 Summary

There are important philosophic and practical differences in defining health and practical challenges in assessing the health status of individuals, communities, and populations. One area in which there is general agreement is that the health of a person at any given time and over the span of a lifetime is the result of complex interactions among genes, personal behavior, and socioeconomic factors, including education, income, and occupation. Although our genetic makeup increases or decreases the risk of various diseases, few diseases are purely and exclusively the result of inherited genes or genetic mutations. There is increasing evidence that environmental and psychosocial stimuli trigger genetic changes or responses. The environment in which people are born, live, and work plays a large role in determining whether they are healthy or sick. Consistent empirical evidence from all over the world shows that people at the lowest levels of the socioeconomic hierarchy have the poorest health indicators. Adequate access to a medical care system is critical for relief from suffering and restoration of health. However, the availability and appropriate utilization of a comprehensive healthcare system that prevents the occurrence of diseases through education, timely vaccinations, periodic medical screenings, and sustained health promotion through optimal behavior modification and lifestyle choices is far more important. Access to and availability of balanced and nutritious food, good education, adequate housing, nonhazardous work environments, minimally stressful psychosocial conditions, and a supportive social network offer the best prospects for optimal individual, familial, and communal life.

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