WHO BECOMES SICK, INJURED, OR DIES?

Chapter 2

Sickness does not just happen. Rather, discernible patterns are evident in the distribution and frequency of sickness, injury, and death in human populations. Social epidemiology is the study of the distribution of patterns of health and illness in human populations and of the social factors that shape them (Berkman and Kawachi, 2000).

REFOCUSING UPSTREAM: WHY SOCIAL EPIDEMIOLOGY

The wry anecdote on the preceding page reminds us that we, as a society—indeed, as a whole world—spend enormous energy and expense trying to save lives and rescue the sick after they have already fallen ill and suffered physical damage due to preventable causes. In order to “refocus upstream” and address those causes pushing people into the stream of sickness and death, we need to understand how social factors promote sickness and death. All humans die eventually, but many premature deaths are preventable. Some sickness is inevitable, but “refocusing upstream” could greatly reduce the amount and severity of sickness, allowing people to function better and live fuller—not just longer—lives. When people are being pushed into the stream of sickness and death by human social arrangements or identifiable human actions (and inaction), then social epidemiology can identify which social arrangements need to be changed for the well-being of all.

Noticing relationships between specific illnesses and people’s social situation is hardly a new phenomenon: Early Greek and Egyptian writers made such connections (Sigerist, 1960). Popular lore also includes awareness of the linkage between, for example, an occupation and a sickness. Alice in Wonderland’s Mad Hatter was a plausible madman because people had recognized a connection between the occupation of hatmaking and bizarre behavior long before the relationship was traced to the effects of the mercury with which hatmakers regularly worked (Stellman and Daum, 1971: 255). Similarly, long before medicine identified the problem, villagers living by the Niger River in Africa recognized that their proximity to the river was related to the prevalence of a horrible sickness characterized by intense itching and eventual blindness. Often they would move away, abandoning valuable farmlands, when the sickness had affected too large a proportion of the community (Eckholm, 1989).

Before there was a formal discipline to study the social distribution of sickness or death, astute observers noticed the significance of social class and occupational variables. For example, more than 150 years ago, one chronicler noted:

“In Bethnal Green in the year 1839 the average age of deaths in the several classes was as follows: Gentlemen and persons engaged in professions, and their families . . . 45 years; Tradesmen and their families . . . 26 years; Mechanics, servants and labourers, and their families . . . 16 years . . . Although the average age of deaths in the different classes varied somewhat from area to area, the differences between the classes existed in all areas of the country” (cited in Blane, 1986: 113).

Refocusing Upstream: Why Social Epidemiology?

Complex Webs of Causal Factors
Methodological Issues

Changes in Life Expectancy and Other Health Indicators

The Myth of Medical Progress

Morbidity and Mortality in a Global Perspective

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Age
Gender/Sex
Race and Ethnicity
Social Class

During the late eighteenth and early nineteenth centuries, industrialization set the stage for the development of the disciplines of both sociology and epidemiology (Spruit and Kromhout, 1987). The changes that accompanied industrial capitalism created radically different conditions for health and illness:

The rates of smallpox, typhus, typhoid fever, diphtheria and scarlet fever all increased: two cholera epidemics had swept through the warrens of the Great Towns, a third was on its way. ... The reordering of the circumstances of everyday life ... ensued. Industrial capitalism gave rise to novel physical arrangements for work and dwelling (the factory, the company town), created new patterns of economic exploitation (mass displacements from land, urban migration in unprecedented numbers, wage labor). ... Hazardous and fatiguing work, damp cold and stifling living quarters, cheap gin and adulterated foods, demoralization—the legacy of disease bequeathed by early capitalism stems from such an environment. (Sussner et al., 1985: 4)

Epidemiology, as a discipline, evolved from early studies of epidemics among people living in such environments. Like sleuths tracing the path of a suspected criminal, epidemiologists examined clues to the path of a suspected source of a disease, such as a virus in an infection or asbestos fibers in asbestosis. Three main, intertwining aspects: agent, host, and environment. The agent is the condition of the internal environment of the host. The host is the site within which an agent creates a disease. The environment is the external conditions linking the agent to the host (such as unsanitary living conditions; the vector, or the means through which the agent is carried (such as diseased marmots or contaminated drinking water); and the condition of the internal environment of the host (such as relative immunity). To cause disease effectively, an agent must be able to survive in the environment and find a susceptible, fertile host (Johnson and Sargent, 1990). For example, a human body (host) may harbor tubercular bacilli (agent), but if the body's resistance is high, this disease agent cannot produce infection. Thus, disease is not the outcome of just one factor; rather, many factors contribute to the complex web of causation.

Complex Webs of Causal Factors

Epidemiology examines the interaction of complex disease-producing factors: a "web of causation" (MacMahon and Pugh, 1970). This web is composed of three main, intertwining aspects: agent, host, and environment. The agent is the source of a disease, such as a virus in an infection or asbestos fibers in asbestosis. The agent is a necessary but not sufficient cause of disease; disease does not occur without an agent, but the agent alone is not sufficient to produce the disease.

The site within which an agent creates a disease is called the host. The host and agent interact within a biosocial environment, which includes the external conditions linking the agent to the host (such as unsanitary living conditions; the vector, or the means through which the agent is carried (such as diseased marmots or contaminated drinking water); and the condition of the internal environment of the host (such as relative immunity). To cause disease effectively, an agent must be able to survive in the environment and find a susceptible, fertile host (Johnson and Sargent, 1990). For example, a human body (host) may harbor tubercular bacilli (agent), but if the body's resistance is high, this disease agent cannot produce infection. Thus, disease is not the outcome of just one factor; rather, many factors contribute to the complex web of causation.

As the name implies, epidemiology arose from the study of widespread infectious diseases, or epidemics. The maturing field now includes study of the distribution, spread, and causes of noninfectious diseases (for example, heart disease, cancer), as well as of other conditions of ill health or death (for example, violence and abuse) which are not diseases of individual bodies. The epidemiology of these noninfectious sicknesses is more difficult than infectious diseases, however, because they develop more slowly, often over decades. The causal factors are thus

Box 2.1

**Fur Fashions and Plague**

"The outbreak of Manchurian plague at the turn of this century constitutes a well-documented example of the role of living patterns in disease causation. The plague bacillus is widely distributed among the wild rodents of Asia. Manchurian marmots normally harbor this microbe, but they do not suffer from the infection under usual circumstances. Around 1910, a change in women's fashions in Europe suddenly created a large demand for the fur of the Manchurian marmot, and a number of inexperienced Chinese hunters began to hunt this wild rodent. Until then it had been hunted only by Manchurians who had a taboo forbidding them to hunt sick animals. In contrast, the inexperienced Chinese trapped every animal within reach, especially the sickest who were slower and easier to catch. As it turned out, the sick marmots were suffering from plague, and many Chinese hunters contracted the infection from them. When the hunters met in the crowded and ill-ventilated Manchurian inns, those who had caught the microbe spread it to their neighbors, thereby initiating a widespread epidemic of pneumonic plague. A change in women's fashions in Europe thus indirectly caused an epidemic of pneumonic plague in Manchuria.'’

recalling accurately relevant information about his life as much as 40 or 50 years earlier.

Noninfectious diseases also often involve multiple contributing factors rather than a single infectious agent. It is difficult to identify all possible factors and determine their relative weight. For example, the hormones in birth control pills may have been implicated in the development of breast cancer. But determining just how significant a factor they may be is complicated for several reasons. The disease does not develop in clinically observable forms immediately upon use of the pill. Other factors (such as the age of the onset of menstruation) are also involved and, in combination, greatly increase probability of developing the disease. Evaluating the relative importance of these various other factors (such as the woman's leanness, her age at the birth of her first baby, and whether she breastfed her babies) or even whether some possible factors are not being considered is difficult. It is hard to determine, many years after the fact, exactly how much or which type of hormones women received; formulations for early contraceptive pills contained much higher dosages of hormones and in different combinations than more recent formulations. If a woman is now 48, she may have taken several different formulations for many periods of varying length since the age of 18.

Although web of causation stresses multiple causes, this approach has a number of limitations. First, it still assumes disease to be a feature solely of individual bodies, divorced from their sociocultural contexts (Link and Phelan, 1995). Few epidemiological studies consider the "spiders" that "spin the web" (Krieger, 1994), that is, the fundamental social, cultural, and biological factors that interweave agent, host, and environment. Epidemiology needs an ecosocial approach incorporating historical, evolutionary, and sociopolitical determinants of health (Susser and Susser, 1996a, 1996b; see also MacIntyre and Ellaway, 2000).

Epidemiology's quantitative focus on separate, discrete variables is a different approach and level of analysis than those of qualitative studies of health and illness. Epidemiological approaches, alone, tend to treat data out of their social and historical contexts, thus reducing their complexity. Epidemiology should be combined with qualitative approaches (such as ethnography) to refine categories (for example, "race," "ethnicity") and to provide a richer, more textured picture of illness and health (Fenton and Charsley, 2000). In the chapters that follow, we suggest some of the spiders that weave the web of illness causation, for which we must look beyond epidemiological studies. Epidemiological studies, however, are a good starting place to describe general demographic factors linked to patterns of disease and death.

**Methodological Issues**

Epidemiology proceeds by observing statistical correlations between two or more variables pertaining to health, sickness (that is, morbidity) or death (that is, mortality). The data for these statistical procedures are often drawn from records kept for other institutional purposes; researchers must therefore contend with discrepancies or errors created by the original records.

**Statistical Associations**

The existence of a statistical correlation between two variables is not sufficient evidence of a causal link between them. Many statistical relationships are spurious. For example, a nineteenth-century observer noted that the incidence of cholera was inversely correlated with the altitude of a community; low-lying places had higher rates of the disease than did those at higher altitudes. This correlation seemed to confirm a prevalent notion that stagnant air (miasma) caused cholera; accordingly, because their air was supposedly fresher, communities at higher altitudes had less miasma and thus less cholera. Later epidemiological discoveries showed that impure water was actually the vector (means of spreading the agent) for cholera. Because low-lying places were more likely to have both stagnant air and impure water, investigators had been led to a spurious correlation between the disease and miasma (Mausner and Bahn, 1985).

Historically, many epidemiological uses of statistical correlations have been seriously biased by racial or ethnic prejudice. Many late nineteenth- and early twentieth-century U.S. public health measures were justified by reference to epidemics allegedly caused by immigrants. An epidemic of smallpox in San Francisco between 1876 and 1877 was attributed to the immigrant Chinese, although deaths among Chinese San Franciscans accounted for only 77 of the 482 smallpox deaths in that epidemic. The city's public health officer attributed the epidemic to "unscrupulous, lying, and treacherous Chinamen who have disregarded our sanitary laws," merely on the basis of the statistical correlation between the influx of Chinese immigrants in the years prior to the outbreak and the increased morbidity compared to the previous epidemic (Kraut, 1994: 82).

Although epidemiology as a science has become more careful methodologically, similar misuse of epidemiological evidence arose in the early years of the AIDS epidemic, when oversimplified statistical correlations made AIDS appear to be linked to Haitian immigrants. In the early 1980s, the U.S. Centers for Disease Control announced that Haitian immigrants had been classified as a high-risk category for AIDS. A few years later, when it dropped this classification, the director of the Center for Infectious Disease acknowledged the spurious relationship, stating, "The Haitians were the only risk group that were identified because of who they were rather than what they did" (cited in Kraut, 1994: 261).

It is often difficult to determine whether a statistical correlation is the product of an indirect relationship actually explained by intervening variables. For example, epidemiological data from the rural southern regions of the United States may show a statistical correlation between low birth weight and childhood deaths from diarrhea. Does that mean that low birth weight causes subsequent death from diarrheal dehydration? This is a plausible interpretation because birth weight may affect the child's susceptibility to illness, but birth weight may be only a by-product of some other causal factors. One clue is to ask a somewhat oversimplified
question: What kind of children are likely to experience both low birth weight and diarrhea? The profile of children with these conditions suggests several related features: They are likely to be from poor families in which the mother was young and received little or no prenatal care (Stevens, 1988). To establish a direct causative link between low birth weight and death due to diarrhea, it would be necessary to identify and hold constant all other factors. Any causal analysis must take into account multiple factors that may operate at several levels in the web of causality.

**Bases of Data** Epidemiological studies may be based on seriously flawed records. Some statistics come from the records of schools, industries, insurance companies, hospitals, and public health departments. Because these data are kept for various other institutional reasons, they are often of limited accuracy for epidemiological purposes. For example, industrial accident statistics can be distorted by an industry's attempt to give the impression of a low accident rate.

Likewise, death certificates are very inaccurate and unreliable sources of information about the causes of death (Hill and Anderson, 1988, 1991). A Connecticut study found that 29 percent of death certificates inaccurately stated the cause of death, based on autopsy reports and patients' medical records (Kircher et al., 1985). In a further 26 percent of the cases, the autopsy and death certificate gave the same general disease category but attributed death to different specific diseases. More than half of the certificates thus provided seriously flawed data about the cause of death. Similarly, interviewing doctors in a Scottish city, Bloor (1991) found wide variations in how deaths were certified.

The usefulness of data from both medical records and death certificates may also be diminished by judgments made by the medical personnel who keep the records. Deliberate misrepresentations often occur when the cause of death is a stigmatizing sickness, such as alcoholism or AIDS. Such reporting problems make it difficult to assess the scope and actual impact of these important health problems. Political, economic, and ideological considerations also figure into the deliberate underreporting of the incidence of stigmatized diseases.

Death certificates also often state only a final cause of death, rather than the initial or contributing causes. For example, a person who dies of a gunshot wound may be listed as having died of internal bleeding. Although the bleeding is a fact in the case, the gunshot wound does not appear in the mortality statistics. Certain causes of death, such as adverse reactions to prescribed medications, are also systematically underreported (Altman, 1988).

The *International Statistical Classification of Diseases, Injuries and Causes of Death*, revised about every ten years by the National Center for Health Statistics of the United States, specifies standards to be used by attending doctors, coroners, and others in an attempt to produce internationally comparable data. Changes in these guidelines for classification, however, yield different rates of morbidity and mortality. One revision of the manual required coders to disregard medically induced (iatrogenic) causes of death, such as postsurgical bleeding or drug reaction, and instead to record the medical condition that first necessitated the treatment (Bloor et al., 1987). The most recent classification guidelines code asks for the immediate cause of death and a sequential listing of all underlying causes. These new data will likely result in the discovery of comparatively high rates of medically induced conditions leading to death.

Despite recent efforts to change the format of U.S. death certificates to allow for more complete and thorough reporting, the data entered on them remain problematic. Most death certificates are based on doctors' clinical judgments about the cause or causes of death; autopsies are expensive and performed for a decreasing proportion of cases. Whereas in the 1950s, autopsies were performed on about half of all patients who died in a hospital, current rates are only about 5–10 percent, performed disproportionately for forensic (that is, legal) evidence and, thus, hardly a representative sample. Even when an autopsy subsequently contradicts the certificate or adds significant new information (which is the case for 20–40 percent of autopsies), physicians often do not amend it (Brody, 2001).

Even if autopsy findings are included on death certificates, these data may be slanted by the criteria used to select cases for autopsy. Women are less likely to be autopsied than men. In some states, nonwhite deaths are less likely to be investigated than those of whites (Bloor et al., 1987). Social status and/or the perceived social worth of the victim may also influence the thoroughness with which a death is investigated (see, for example, Sudnow, 1967). The cases autopsied are not a representative sample of all deaths.

So-called errors or variations in diagnosis are not random; they are often socially produced. For example, medical personnel's judgments may vary according to the perceived social class of the dead person. In earlier medical reporting, the cause of a professional person's death from heart disease was likely to be labeled "angina," whereas the cause of death of a working-class person with the same condition was usually classified as something else (Marmot et al., 1987). Similarities in diagnoses (and thus morbidity statistics) are influenced by doctors' attitudes toward patients' social characteristics, such as gender, social class, or occupation, as discussed further in Chapters 9 and 10. For example, earlier data indicated that the rate of deaths due to alcoholism in Scotland was about twice those in England and Wales; these data are now in question, because studies show that doctors in England and Wales are much less likely than those in Scotland to attribute death to alcohol-related diseases such as cirrhosis (see Altman, 1988).

Cultural differences in medical practice, even among Western industrial nations and among doctors trained in modern biomedicine, account for some variations in diagnoses. For example, blood pressure readings considered hypertensive in the United States would be considered normal in England (Payer, 1988). Doctor characteristics may also affect the diagnosis or reported cause of death. Because of differences in their training, younger British doctors were less likely than older ones to report stomach cancer as a diagnosis (Bloor et al., 1987). Thus, a falling rate of mortality due to stomach cancer may be at least in part an artifact of differences in doctors' education.
These examples show that many health statistics may be *artifactual*, or produced by the social arrangements by which the statistics themselves are gathered and processed. Such artifactual evidence is misleading and often altogether incorrect. With these limitations in mind, let us examine some results of epidemiological investigation. The cautious use of epidemiological information can give us a broad picture of health.

**CHANGES IN LIFE EXPECTANCY AND OTHER HEALTH INDICATORS**

The twentieth century saw dramatic increases in life span, worldwide—particularly in the richer countries that became economically “developed.” Most of this gain was achieved by reducing “premature” mortality; that is, the average life expectancy rose particularly due to the reduction of deaths among infants, children, and young adults. It is worthwhile to examine and compare such health indicators as infant and child mortality rates, both between countries and over time. These comparative data suggest clues about where to invest efforts and money in order to improve people’s health and to prevent illness and untimely death. The last part of this chapter examines some variables in the United States for such clues.

American males born in 1900 could expect to live 46.3 years, and females, 48.3 years. By 1999, the life expectancy for both sexes had increased, but so had the gap between male and female rates. Males born in 1999 can expect to live 73.9 years; females born that same year could live on the average to the age of 79.4 years (USDHHS, 2001a). Figure 2.1 shows the dramatic increase in life span in the United States by decades from 1900 to 1999.

A general decline of infant mortality and increasing difference in life expectancy between men and women began to emerge around 1920. In 1999, the infant mortality rate for the United States was 7.1 deaths per 1,000 (USDHHS, 2001a). Figure 2.2 shows U.S. patterns of infant mortality between 1920 and 1999. There are also dramatic differences between blacks and whites in both general life expectancy and infant mortality. Infant mortality rates are a crude indicator of a nation’s health status. Relative to other economically developed nations, the United States has a very high infant mortality rate, ranking twenty-sixth among the world’s “developed” nations (USDHHS, 2000: 157). Similarly, the United States has a high under-five child mortality rate, ranking behind all other developed countries in a worldwide comparison, and tying for thirtieth place with Cuba, which has a “developing” economy (UNICEF, 2000).

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1 The terms “developed” and “underdeveloped” are unfortunately laden with connotations of superiority and inferiority and fail to indicate the extent to which wealthier and more powerful nations have historically prevented other countries from becoming “developed.” Because these are the terms used by the United Nations and other data-gathering agencies, however, we have continued to use them.

2 The infant mortality rate is the number of children who die within the first year, calculated per 1,000 live births.
Along with life expectancy changes have also come changes in the causes of death in Western industrial societies. Despite the recent development of some infectious diseases such as AIDS, there has been a long-term decline in death rates due to infectious diseases and an increase in death rates due to chronic degenerative diseases (McKinlay et al., 1989). In 1900, acute infectious diseases (pneumonia, influenza, and tuberculosis) were the foremost causes of death in the United States. Chronic diseases (heart disease, cancer, cerebrovascular disease, and chronic lower respiratory disease) were the major killers in 1999. In the last two decades of the twentieth century, the death rate from coronary heart disease decreased, but rates of death from cancer and diabetes increased (USDHHS, 2001a).

This shift from infectious to chronic degenerative diseases is part of epidemiological transition, characterizing changes in societies as they become more developed. Nevertheless, infectious disease can also be chronic (as is the case with AIDS) and infectious diseases are still a source of morbidity and mortality in developed societies (Radley, 1994). Some evidence indicates that since 1980, mortality from infectious diseases in the United States has been increasing, after years of steady decline. In 1980, infectious diseases ranked fifth as cause of death; by 1992, they ranked third (Pinner et al., 1996). Possible reasons for these increased rates include the spread of AIDS and diseases such as tuberculosis (of which some strains are resistant to antibiotics), especially in contexts of increased poverty (Pinner et al., 1996; Radley, 1994). Nonetheless, chronic degenerative diseases (including heart disease and cancer) still predominate as causes of death.

The increased incidence of chronic degenerative diseases cannot be attributed simply to the fact that people are living longer. It is true that the longer people live, the more likely they are to develop a chronic disease; however, increased longevity does not account for the fact that, beginning in the 1950s, even younger age groups have shown an increased incidence of these afflictions (Eyer and Sterling, 1977; McMurray and Smith, 2001). This premature development of degenerative sickness may be the result of contemporary dietary, environmental, and social factors. Evidence that the social, biochemical, and physical environment of industrialized societies create new health problems, such as increased rates of cancer, comes from a comparison with preindustrial societies, which have lower incidence of these problems (McMurray and Smith, 2001). Aspects of the way of life in industrialized societies are implicated because the rates of chronic degenerative diseases increase among groups that migrate from agricultural to industrial communities (Janes, 1986).

THE MYTH OF MEDICAL PROGRESS

Many people believe miracles of medical progress have been responsible for the improved health and longevity in Western industrial countries since the turn of the century. Media imagery often contributes to this notion, but much evidence suggests the contribution of medical intervention to increased life expectancy has been rather limited. McKeown (1979) argues that improved nutrition and population control, the control of predators, and improvements in urban dwelling conditions and hygiene have played a much greater role in extending life expectancy than has medical technology. The impact of these various factors on health can be documented through epidemiological studies.

A study of mortality rates in the United States shows that medical intervention (such as inoculations) accounts for only a small percentage of the decline in mortality from infectious diseases during the early part of the twentieth century (Kates, 1996: 57; Wilkinson, 1996: 66–67). Another study (Bunker et al., 1994) calculated that only about five to seven years of the 30 years by which U.S. life expectancy increased in the twentieth century have been due to any form of preventive or therapeutic medical care.

Figure 2.3 shows that, of nine common infectious diseases, only poliomyelitis began to decline significantly after the introduction of the vaccine. All the other major infectious diseases had declined dramatically prior to the introduction of a vaccine or antibiotic (McKinlay and McKinlay, 1977). The near-eradication of smallpox was not due mainly to new technologies (for example, vaccines), but to international surveillance and communities' rapid and concerted response to outbreaks (Baxby, 1995). The decline of tuberculosis likewise preceded the introduction of chemical therapies (Friedman, 1987). Nonmedical factors, such as improved nutrition, clean water, garbage and sewage disposal, and other public health measures, may account for the primary decline in mortality due to many infectious diseases.

The precise contribution of medicine to increased life expectancy, to the reduction of morbidity, and to the quality of life is difficult—if not impossible—to assess. Some have argued that the iatrogenic (that is, medically induced) health risks of modern medicine and its institutions outweigh its benefits (Illich, 1975). This position probably overstates the harmful effects of modern medicine. Nevertheless, any evaluation of the contribution of medical intervention must take into account its iatrogenic consequences, such as unneeded surgery, over- and misprescription of drugs, the negative side effects of medication, and infections transmitted in medical settings.

Without a doubt medicine has made significant contributions to human health. It is effective in treating many acute diseases. Emergency medicine has saved many lives, and technological innovations in medicine since World War I have made it possible for many to survive previously mortal health crises. Medicine has also played a major role in the virtual eradication of some infectious diseases, such as smallpox and polio. The ability of medicine to treat or prevent chronic, degenerative diseases is more limited, however. Furthermore, the benefits from medical progress must be distinguished from social-environmental changes and public health measures that have played a major part in improving people's health. The contributions of public health measures are often devalued by modern medicine, resulting in social policies that allocate the vast majority of health care expenditures...
The Fall in the Standardized Death Rate (per 1,000 Population) for Nine Common Infectious Diseases in Relation to Specific Medical Measures in the United States, 1900–1973. (Sources: John B. McKinlay and Sonja M. McKinlay, “The questionable effect of medical measures on the decline of mortality in the United States in the twentieth century,” Milbank Memorial Fund Quarterly 55, 1977: 422–423.)

MORBIDITY AND MORTALITY IN A GLOBAL PERSPECTIVE

The pattern of high rates of mortality due to infectious diseases that was evident in industrialized societies at the beginning of the twentieth century still characterizes the health situation of many parts of the world. International comparisons corroborate the historical comparisons: Life and health are largely the product of healthy living conditions. When people’s living conditions—nutrition, sanitation, clean water, adequate shelter, safe workplaces—improve, then sickness and “premature” death are greatly reduced. Life expectancies in economically advantaged countries are substantially higher than in the rest of the world.

On average, compared to people in poorer countries, those in economically advantaged countries live more of their life span in relative good health. In 2000, the World Health Organization (WHO) changed its method of calculating longevity, taking the expected overall life longevity and subtracting years “lost” to disability and debilitating diseases (weighted for severity). Thus, for example, Uganda—a desperately poor country, struggling with epidemic AIDS, malaria, and tuberculosis—has the low overall life expectancy at birth of about 42 (World Bank, 2001b), but many people are living with diseases so debilitating that the average “healthy life expectancy” at birth is closer to 32.7 years. In the WHO rankings, all of the countries in the lowest ten were in Sub-Saharan Africa, where overall life expectancy has been dropping dramatically since about 1990, as discussed further in this chapter (WHO, 2000).

By contrast, the top countries for healthy life expectancy were all relatively economically advantaged: Japan (74.5 years), Australia (73.2), France (73.1), Sweden (73.0), Spain (72.8). Not only was their overall average life expectancy greater than lower-ranked countries, but also the years “lost” to debilitating disease or injury were fewer. In this rating system, the United States ranked twenty-fourth (70.0 years)—lower than most other economically advantaged countries (WHO, 2000). Some of the reasons for this low ranking, as compared with many countries with far less per capita income, are discussed further in this chapter and in Chapter 11.

Average life expectancies and mortality rates are somewhat misleading statistics, however, because they do not reflect the variations between subgroups (such as class, gender, and rural-urban differences) within each society. For example, the “healthy life expectancy” rate for Brazil is relatively low (59.1 years), but the southern half (where wealthier citizens are concentrated) has a relatively high rate (WHO, 2000). Extreme disparities between the living conditions of the wealthy and the poor explain most of this difference. The average infant mortality rate for Brazil is 43.2 and the average under-five mortality rate is 116.7. The richest quintile (20 percent) of Brazil’s population, however, experiences only a 15.3 infant mortality rate, while the poorest quintile has a rate of 72.7 (nearly five times as great). The richest quintile has an under-five mortality rate of 18.7, compared to medical research and treatments rather than to preventive programs such as maternal nutrition or workplace safety.
In the nineteenth century, Africa was viewed as a “white man’s grave,” a disease-filled “dark continent.” Many diseases, however, such as smallpox, syphilis, measles, influenza, cholera, and tuberculosis, were introduced to Africa from Europe and Asia, devastating native populations, which had no immunity to diseases to which they had never been exposed. The colonization of Africa and the slave trade created massive sociocultural dislocations and altered patterns of land use. Migration to the cities and the attendant overcrowding, as well as the ecological disruption of the land, generated conditions that bred and spread certain infectious diseases. In epidemiological terms, alterations in the environment made for a high mortality rate and prevalence of infectious diseases. (Chapter 3 discusses further the problem of world hunger.) As of the beginning of the twenty-first century, nearly one-quarter of people in developing countries lacked access to safe drinking water, and approximately half lacked adequate sanitation (World Bank, 2001a). Childhood diseases such as measles are much more likely to result in death where nutrition and sanitation are poor. Indeed, communicable diseases account for 50 percent of all deaths among the poorest quintile of the world population, compared to only 8 percent among the richest quintile (World Bank, 2001b).

Many Westerners consider Third World countries to be inherently disease ridden, eagerly awaiting the benefits of modern civilization. In reality, the intrusion of so-called civilization from the West has introduced devastating infectious diseases to Third World peoples. Native peoples of the Americas and Polynesia were decimated by diseases introduced by European explorers, sailors, merchants, and colonists (Kraut, 1994). Since Captain Cook’s voyage there in 1778, more native Hawaiians have died of infectious diseases introduced by foreigners than of all other causes combined (Bushnell, 1993).

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In countries such as South Africa, radically different pictures of health exist side by side in the same society. As a whole, South Africa has a highly developed economy. Nevertheless, the policy of apartheid (1950–1993) produced the social and economic domination of a white minority over a nonwhite majority by radical segregation of housing, education, occupations, and legal status. This social arrangement resulted in very different living conditions for whites and nonwhites, and hence in different patterns of morbidity and mortality. The infant mortality for blacks in South Africa is approximately six times higher than that of whites.

One-quarter of black children under 14 years of age are chronically malnourished or undernourished. By contrast, hardly any malnutrition or undernutrition exists among whites, and whites generally die of “diseases of affluence,” which are linked with lifestyles characterized by high-calorie diets and sedentary occupations. Blacks are more likely to die of infectious diseases, such as cholera, tuberculosis, and measles (a disease that is rarely fatal in affluent societies). Thus, in one nation, two very different ways of life produce different health profiles. These differences are a dramatic illustration of how social-structural arrangements (that is, apartheid) can produce different patterns of health, illness, and death (Frankel, 1986).

Another socially produced cause of illness, disability, and death is human violence toward other humans. Assault, homicide, and domestic violence take an enormous toll on the health and well-being of people worldwide. When the person who is killed or maimed by such violence is also economically and/or emotionally the support of a family, the long-term health of many people can be harmed. War and other organized violence harm people indirectly, by destroying or siphoning off valuable resources that people need for their subsistence. Many countries spend a large portion of their gross domestic product (GDP) on military expenditures, rather than social needs. For example, Pakistan bought 40 fighter jets from a French transnational corporation for an amount that could have supplied safe drinking water for two years for the 55 million Pakistanis who lack it, plus essential medicine for the 13 million Pakistanis who lack it, and basic education for 12 million Pakistani children who lack it (cited in Millen and Holtz, 2000).

War and war-related injury and death are particularly devastating to the health and well-being of an entire country. For example, Afghanistan (a desperately poor country that came to most Westerners’ attention only when the United States and its allies pursued their “war on terrorism” on Afghan territory) had experienced more than two decades of warfare, in which U.S.-backed Afghan tribes fought Soviet-supported tribes. Warfare and drought had created conditions of extreme deprivation, causing Afghanistan to suffer the fourth highest rate of under-five mortality in the world (more than 25 percent of all Afghan children die before the age of five) (UNICEF, 2000). Many of those who survived had serious injuries and disabilities, indirectly due to the years of war. An estimated 5–7 million undetonated land mines (many supplied by the United States3) had been left buried in the fields and byways, and many civilians—especially farmers and children—were killed or maimed by them (Bearak, 2001). Long after such active violence ends, the people must struggle with deprivation, injury, and illness brought on by warfare (see Taiapale et al., 2001).

Not only are diseases themselves spatially and socially segregated, but various forms of segregation also reduce people’s ability to know and understand

3The United States has been the world’s foremost arms supplier. Between 1993 and 1994, U.S.-supplied weapons were used on one or both sides in 45 of the 50 largest conflicts; between 1993 and 1997, the U.S. government sold, gave away, or let industry sell $190 billion in weapons to almost every country in the world (cited in Millen and Holtz, 2000: 212).
Threats of biological warfare and bioterrorism have also heightened our awareness of the threat of epidemics of dangerous infectious diseases, such as smallpox. Improperly handled meats, factory emissions of radiation or air pollutants, and chemical contamination of wells and reservoirs by foreign terrorists. Acute febrile diseases (like malaria and dengue fever) have reappeared and spread—often in drug-resistant mutant forms—even in developed countries. Improperly handled meats, factory emissions of radiation or air pollutants, and chemical contamination of wells and reservoirs by foreign terrorists. The same political and economic forces that spurred such globalized “development,” however, have also brought new (and the return of old), globalized sources of infection, disease, and death. By the end of the twentieth century, it was clear that economic globalization had dramatically increased the worldwide epidemiological “web of interconnectedness” (Zielinski Gutiérrez and Kendall, 2000).

The myth of medical progress, together with belief that the world was engaging in economic development and “progress,” led many people in the 1960s and 1970s to believe that infectious diseases were being eradicated. Many proponents of spreading technological development throughout the world argued that, partially in response to improvements in pharmaceutical and agricultural technologies, developing societies would, likewise, pass from conditions of high mortality due to infectious diseases to concern about chronic diseases of aging (see Garrett, 1994: 32).

In the early 1980s, Americans and Europeans began to become aware of a new epidemic (eventually attributed to the Human Immunodeficiency Virus, HIV) in their midst. The HIV/AIDS epidemic has shaken that faith in “progress” and, in many places, completely reversed previous trends toward improvements in life expectancy and health. In the 20 years since what had been an animal virus apparently mutated and began infecting humans, it has killed an estimated 28 million people and has spread rapidly worldwide (WHO, 2001). Other infectious diseases have reached epidemic proportions in recent years, as well. Tuberculosis and acute febrile diseases (like malaria and dengue fever) have reappeared and spread—often in drug-resistant mutant forms—even in developed countries. Threats of biological warfare and bioterrorism have also heightened our awareness of the threat of epidemics of dangerous infectious diseases, such as smallpox.4

4This heightened awareness of our vulnerability to outside sources of infection simultaneously reveals how relaxed external regulation in recent decades has made us vulnerable, as well. In the United States, water, air, and food are far more likely to be dangerously contaminated by internal agents (such as improperly handled meats, factory emissions of radiation or air pollutants, and chemical contamination of wells and reservoirs) than by foreign terrorists.

Epidemiology—especially social epidemiology—has grown more sophisticated as it tries to understand and respond to these urgent health issues worldwide. The epidemiology of HIV/AIDS is a good illustration of how a disease spreads and affects populations differently in different cultural and socioeconomic situations. The history of our understanding of the AIDS epidemic also illustrates many of the blind spots that often prevent effective and rapid response to epidemics and their victims, both in the United States and throughout the world.

In 2000, there were an estimated 3 million deaths worldwide due to AIDS—the highest total since the beginning of the epidemic more than two decades ago. The United Nations estimates that, by the end of 2001, 40 million people worldwide were infected with HIV. Of those infected, more than 96 percent live in developing countries. About 940,000 people in North America (mainly the United States) were living with HIV/AIDS. The infected proportion of the U.S. population is much higher than in other developed countries (about three times the rate in Europe and six times the rate in Australia and New Zealand). It is, nevertheless, much lower a proportion than in severely affected regions, such as the Caribbean (where HIV/AIDS is about four times as prevalent as in North America) and Sub-Saharan Africa (where it is about 15 times as prevalent) (UNAIDS, 2001a).

Table 2.1 shows the distribution of HIV/AIDS as of December 2001. This epidemic is rapidly changing. For example, in 2001, the epidemic was spreading most rapidly in Eastern Europe and Central Asia, where the economies and public health systems collapsed after the Soviet Union dissolved. East Asia and the Pacific also showed dramatic increases in new HIV/AIDS cases, as parts of China and other countries that had been politically and culturally isolated began to experience economic conditions promoting spread of the disease (UNAIDS, 2001a).

The transmission of HIV depends on the geosocial and cultural context in which it is spread. In the early years of the epidemic, in North and South America, Western Europe, Australia and New Zealand, HIV/AIDS affected mainly males. The mode of transmission often involved men having sex with men (MSM), whereas the rate from heterosexual intercourse was relatively low. In these countries, AIDS was concentrated primarily among MSM, who may or may not self-identify as homosexual, contributing to the myth that it is essentially a “gay plague.” This misperception, however, may have initially blinded researchers and the public alike to the breadth of HIV infection. We now know that HIV transmission is the result of sexual (heterosexual as well as homosexual) contact with an infected person, sharing needles and/or syringes with someone who is infected, and blood transfusions with infected blood supplies. Babies born to women with HIV infection may become infected before or during birth or through breastfeeding after birth.

This early misinterpretation illustrates how researchers’ categories for classifying groups can misguide the interpretation of data. These categories are far from neutral. In this case, in the early years of the epidemic, the notion that AIDS was a “gay plague” classified homosexual males as the prime risk group. Thus, some American physicians could not believe intravenous drug users who developed...
AIDS but denied any homosexual contact (Bloor, 1995: 56). Rather than oversimplifying data into “risk groups,” it makes sense to focus on the variable sociocultural, political, and economic contexts in which HIV transmission occurs.

This focus better enables us to understand changing patterns (such as the increasing proportion of women and children) in HIV/AIDS infection in developed countries. The rates of new infection in the gay community had leveled off until the mid- to late-1990s because of dramatic changes in sexual practices. Such changes preceded large-scale government health education campaigns and were due, in part, to social organizing, education, and condom distribution by the gay community (Bloor, 1995). More recently, however, rates of new infection among MSM have increased again, especially among young men. In 2000, 59 percent of reported HIV infections among adolescent males aged 13-19, and 53 percent of cases among men aged 20-24, were transmitted by MSM contact (USCDC, 2002a). At the same time, however, rates of HIV infection among men of color—especially gay men of color—are increasing. The stigma attached to homosexuality in the communities of color may prevent men from being tested and from identifying themselves as gay or bisexual (Altman, 2001). Research among gay and bisexual men indicates that some are now less concerned about risk of infection than in the 1980s, perhaps assuming (incorrectly) that AIDS is no longer difficult to treat and fatal (USCDC, 2002a).

When the AIDS epidemic was first recognized, some epidemiologists referred to groups (such as homosexuals and Haitian Americans) with disproportionately high rates of HIV infection as “risk groups.” The designation of groups as “high risk” is itself problematic. First, no one is at risk of HIV infection because of their membership in a group but because they engage in certain risky practices. Intravenous drug users are at risk, not because they inject drugs, but because they share needles (Bloor, 1995: 29). Second, researchers’ use of broad categories (such as “Hispanic”) to label so-called “high-risk” groups obscures the wide variation within groups. Higher rates of poverty and substance abuse increase the risk of infection in some Hispanic American communities. For example, among Puerto Rican-born Hispanics, high-risk behaviors associated with drug abuse are far more common than MSM as a mode of transmission. By contrast, for those born in Mexico, Cuba, and Central and South America, MSM is the primary mode of transmission (USCDC, 2000). Thus, clearly, an individual’s risk for HIV infection is not a product of being Hispanic. Even “risk groups” identified on the basis of risky behavior (such as prostitution) show considerable variability in HIV incidence. For example, prostitutes in Las Vegas, Nevada (where prostitution is legal and regulated) had none, while prostitutes in Newark, New Jersey had a 55 percent rate of HIV infection (Bloor, 1995). Third, to single out certain groups as “high risk” may contribute to stigmatizing them, leading to discrimination and further poverty, as happened to many Haitian Americans earlier in the epidemic.

Although HIV/AIDS strikes rich and poor alike, new AIDS cases in developed countries are disproportionately among poor people (World Bank, 2001). Thus, it would be a mistake to conclude that the growing rates of infection among certain minority groups are mainly about race or ethnicity, per se. One grassroots organization of AIDS activists, ACT UP (AIDS Coalition to Unleash Power), has shifted the focus of its protests accordingly. First formed in the late 1980s as a coalition of white, middle-class gay and lesbian activists, the organization now emphasizes racism and poverty (both in local and global contexts) as its main AIDS-related targets (Kim, 2001). The growing differential between the situation of the poor and peoples of color, on the one hand, and the rich, generally white, developed world, on the other hand, leads some critics to suggest that the HIV/AIDS epidemic is but one product of a “global apartheid”—an international system of minority rule (comparable to that of whites under the South African policy of apartheid) based on differential access to basic human rights (Booker and Minter, 2001).

The global pattern of HIV/AIDS infection has been a disproportionate burden for the poor—of sickness, death, orphaned children, increased poverty, and the loss of entire generations of prime-age adults. Worldwide, an estimated 96 percent of those infected are in the developing countries (World Bank, 2001). The greatest devastation of HIV infection has been in Sub-Saharan Africa (as Table 2.1 shows). There, the patterns of transmission and of the course of infection are very different from those in Europe and the Americas. In Sub-Saharan Africa, one-third to one-half of all persons infected with HIV/AIDS are women, with a particularly high concentration of cases among prostitutes. This prevalence suggests that, in
that region, heterosexual intercourse is a more common means of transmitting AIDS than other means, such as homosexual contact and intravenous drug use (Ateka, 2001). In Sub-Saharan Africa alone, an estimated 2.2 million children are at risk of HIV infection through mother-to-child transmission (UNAIDS, 2001b).

What cultural and socioeconomic factors account for these differences? Diseases are spread when populations are forced to move, due to violence (such as the 1994 exodus of millions from Rwanda), or natural disasters (such as drought, urbanization, and displacement). Such displacement often separates young men from their families and breaks cultural patterns that previously constrained sexual behaviors. The United Nations (1998) reports that, worldwide, the number of refugees, returnees, and internally displaced persons has increased more than fourfold in less than two decades. Africa accounts for a vastly disproportionate number of those displaced persons (in 1997, 4.3 million refugees, 1.7 million “returnees,” and 16 million internally displaced persons).

In parts of Africa, the practice of migratory labor (residual from the colonial past), together with dire poverty, make prostitution a common practice. Millions of couples are separated for months at a time, while men leave their families to work in the cities or on plantations (Lear, 1996). Men who resist the use of condoms yet have multiple sex partners, transmit the virus from place to place during their travels for work. And when they return to their families, even if they are obviously sick, their wives are not free to refuse to engage in sex (see du Guerny and Sjoberg, 1999; Zielinski Gutierrez, and Kendall, 2000). Women cannot insist their husbands use condoms. One South African schoolteacher explained that, in their culture, “the woman has no power with the man,” because having paid the bride price, he believes “she is supposed to do everything I say” (quoted in McNeil, 2001a).

At the same time, African women who become infected and transmit HIV infection may have few or no alternatives for their subsistence and survival. An estimated 65 percent of AIDS infection in females occurs by age 20, due in part to older infected males taking very young women as partners, to increasingly very young women resorting to prostitution for survival, and to widespread and growing rates of rape in many African cities (Zielinski Gutierrez, and Kendall, 2000). Cultural myths that sex with a virgin will cure AIDS have resulted in dramatic increases in the number of child rapes by HIV-infected men (Swarns, 2002). Rape itself is epidemic in many parts of Africa. One doctor in a rural South African province where there is about a 40 percent chance that a young male rapist is HIV positive explained that it was nearly impossible to obtain anti-AIDS drugs for rape victims, in part because “forced intercourse is not regarded as a hell of a crime by the average rural Zulu; it’s very accepted” (quoted in McNeil, 2001b). Thus, women’s already low status and lack of power renders them more vulnerable to HIV infection (see Crossette, 2001; Schoofs, 1999). Cultural pressures on women define their status as child bearers and mothers (Bassett and Mhloyi, 2001); hence, they are expected to bear children, even when there is a likelihood of transmitting HIV to them. Furthermore, the burden of home care for people with AIDS falls mostly on women, thus increasing their burden of poverty (du Guerny and Sjoberg, 1999).

Poverty is a significant factor in the pervasiveness and intensity of the HIV/AIDS epidemic in Sub-Saharan Africa, as well as in other developing countries. People who are already malnourished, have parasitic and other infections, and lack access to health care, succumb more readily to HIV infection (Stillwagon, 2001). Women are particularly affected in this respect, too, because they constitute 70 percent of those living in extreme poverty in the world (Schoofs, 1999). A combination of domestic, socioeconomic, and cultural factors shape the epidemiology of AIDS in Sub-Saharan Africa differently from the patterns of other countries, especially economically developed countries. Particularly important factors in Sub-Saharan Africa include rapid urbanization, the migrant labor system (a legacy of colonialism), extreme gender inequality, and abject poverty.

Dire poverty is not only a causal factor but also a growing result of the HIV/AIDS epidemic in Sub-Saharan Africa. Victims’ families and the entire economy lose the productivity of younger, skilled workers who die of AIDS. The epidemic is creating a generation of orphans—12.1 million in Sub-Saharan Africa as of 2001 (UNAIDS, 2001b). HIV/AIDS is putting a strain on already limited health care resources (Danzinger, 2000). Lacking money to provide infected patients with expensive antiretroviral drug therapies, hospital staffs are often overwhelmed with the emotional, as well as financial, drain of tending large numbers of inexcorably dying patients (Swarns, 2001).

The epidemiology of HIV/AIDS (and many other diseases) in Africa and other developing countries is, thus, linked with economic globalization—especially the policies of the World Bank, the International Monetary Fund (IMF), the World Trade Organization (WTO), and transnational pharmaceutical corporations. In developed countries like the United States, many HIV-infected persons can obtain the costly treatments that may enable them to live and continue to work, sometimes turning HIV infection into a chronic disease. Transnational pharmaceutical companies control the patents on these drugs and want to protect their worldwide profits.

In 2001, U.N. Secretary General Kofi Annan called on rich countries to allocate $7–10 billion annually to a global fund to address infectious diseases (particularly HIV/AIDS, malaria, and tuberculosis) in developing countries. At the same time, Doctors Without Borders, the Nobel Prize-winning NGO (nongovernmental organization), and other international health activists tried to arrange for large-scale purchase of antiretroviral drug treatments from generic producers, like the Indian manufacturer CIPLA. At meetings of the World Trade Organization, the United States sided with the patent-owning transnational pharmaceutical corporations, which vehemently argued that sales of the generic drugs violated their “intellectual property rights.” If the U.N. AIDS Fund and developing countries’ health agencies are forced to pay the “market” prices of patented drugs, they will be able to help

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3The United States was quick to shirk its fair share of contribution, offering only $200 million for the fund—less than two-tenths of 1 percent of a tax cut given earlier that year to the upper quintiles of American taxpayers (Weisman, 2001). Nine months after the call, donations and pledges had fallen far short of the goal, and the U.S. pledge was particularly low proportionate to its national wealth (Sjoberg, 2002).
only a small portion of the infected population (Rosenberg, 2001; Weissman, 2001). Now that epidemiologists understand how HIV/AIDS is spreading and other researchers have developed treatments that, while not curing, prevent AIDS from killing and debilitating its victims, the world needs the political will to address the causes and effects of this global epidemic. Meanwhile, millions of preventable cases of new infection are developing; millions of needless deaths are occurring.

VARIATIONS IN MORTALITY AND MORBIDITY: THE CASE OF THE UNITED STATES

The wide range of mortality (that is, death) and morbidity (injury and disease) within societies is distributed along basic sociological variables such as region, age, gender, ethnicity, and class. Variations in health and death are linked to people's different locations in social space and structure. The following discussion examines major variables in mortality and morbidity for the United States.

Age

As expected in a developed country, U.S. death rates increase with age. Figure 2.4 shows population pyramids for the United States, which indicate, for a given year, the relative numbers of males and females in each age bracket. The data for 1910 take a conventional "pyramid" shape, because the birth rates are balanced by death rates for both sexes, in a somewhat even pattern. The U.S. pattern for 1910 resembles that of many moderately well-off developing countries today. The U.S. pattern for 2000, however, is less conventionally shaped because of decreased (but varying) birth rates and lower death rates, a pattern that characterizes economically developed countries.

This trend, in which fewer people are born and more live past the age of 50, has been called the graying of society because larger proportions of the population survive into old age. Between the Census 1990 and Census 2000, the proportion of people aged 85-years-and-older grew dramatically; however, the growth rate of the proportion of 65-and-older decreased, due to low birth rates in the 1920s and 1930s (U.S. Census Bureau, 2001b). By Census 2020, the older members of the "baby boom" generation will have swelled the ranks of the 65-and-older population. There are socioeconomic consequences to a changing age structure of a society. For instance, in a "graying" society, one can anticipate increased health costs and a greater demand for care (McMurray and Smith, 2001).

Although chronic degenerative diseases have been increasing in all age groups, their rates increase most dramatically for older people (Radley, 1994). Only rates for death due to homicide, accidents, and suicide decline with age; rates of death due to chronic illnesses and disabling conditions increase. Many chronic conditions are not a natural result of aging. For example, the rate of hypertension (high blood pressure) increases with age in U.S. society but not in hunting and gathering societies, such as the nomadic !Kung bushmen of the Kalahari.

Desert in Africa (Schall and Kern, 1981). Similarly, the U.S. rate of death and chronic illnesses, such as chronic lung disease and cancer, associated with tobacco use is higher than in most other countries; aging Japanese, by contrast, are far less likely than their American counterparts to experience these health problems (WHO, 2000).

Although aging seems inevitably to involve increased morbidity, some researchers suggest the possibility of "compressing" morbidity. They argue that although the life span is biologically limited, it may become possible to limit the period when an aging person is infirm (Fries, 1990). The "compression of morbidity" thesis helps to challenge stereotypes about aging (such as the inevitability of deterioration), but it may be overly optimistic about health problems associated with aging.

Although compression of morbidity may be occurring, it pertains mostly to higher socioeconomic groups. Widespread compressed morbidity would require greatly increased social equality (Bury, 2000). One study examined healthy life expectancy by gender and education for U.S. whites and African Americans in 1970, 1980, and 1990. The study found evidence of the compression of morbidity in the last decade among those of higher educational status. Lower-status persons, however, are still experiencing an expansion of morbidity (Crimmins and Saito, 2001). Clearly, in order to achieve widespread compression of morbidity or a significant societal increase in healthy life expectancy, the United States needs a much higher level of social equality.

Gender/Sex

Before the twentieth century, women typically died younger than men. One explanation of the difference is that frequent pregnancies and childbirth involved considerable health risks. Also, under conditions of general material scarcity, women often got what food was left after men and children ate their share. Poor nutrition then compounded women's health difficulties (Shorter, 1982). Today, in some developing countries, male life expectancy is greater than, or the same as that of women (Doyal, 1995). In several countries of North Africa and the Middle East, where the male-female ratio of healthy life expectancy is even or reversed, female mortality rates may be adversely affected by the disadvantaged position of women in these societies (WHO, 2000).

The female advantage in life expectancy became apparent in the United States and Europe by the late nineteenth century (Doyal, 1995). In the early 1900s, women's life expectancy was about two to three years longer, on average, than men's in the richer countries around the world. By 1999, women were living seven to eight years, on average, longer than men in those countries (WHO, 2000). As Figure 2.1 shows, the United States exemplifies this pattern, with the female "advantage" of 2.0 years in 1900 growing to 5.5 years in 1999 (USDHHS, 2001a).

Biological factors may partially account for the higher mortality rates of males, especially early in life (Carpenter, 2000). Higher fetal mortality rates have been reported for males (Harrison et al., 1992). Because the male-female mortality rate difference occurs cross-culturally, it cannot be explained fully by social or cultural factors. Female life expectancy is also higher among many other animal species (Sagan, 1987). This biological advantage for women may come from hormonal differences, although such differences may also produce higher rates of some sicknesses in women (Friedman, 1987). Although biological factors may contribute to differences in men's and women's health and longevity, social factors linked to gender—rather than sex—differences often create, maintain, or exacerbate biological health differences (Bird and Rieker, 1999).

In the United States, men die earlier and have more life-threatening illnesses, but in general women become ill more frequently (Carpenter, 2000). Chronic illnesses are more prevalent among women than men, but they are less life-threatening than those of men (Marks, 1996). Some researchers argue, however, that rates of debilitating chronic degenerative diseases are rising among older women, such that their healthy life expectancy is much lower than their overall life expectancy. Women may live longer, on average, than men, but are likely to spend most of those extra years in conditions of disability and dependency (Litt, 1997).

Women also report more episodes of illness and more contact with physicians (Susser et al., 1985: 71). Women's higher rates of sickness and doctor visits can be partly explained by gynecological or reproductive problems, which men do not have, but the incidence of these problems is not great enough to account for the difference in morbidity rates between men and women. Perhaps women are more willing than men to admit they are sick and/or to visit physicians. If this is true, then lower morbidity rates for men are deceptive, because they are a function of the underreporting of sickness rather than differences in rates of actual disease (see Waldron, 1994). Perhaps women learn more effective coping skills and ways of caring for themselves, but no firm evidence exists for this hypothesis. Gender differences in morbidity may be underestimated, however (Carpenter, 2000). For such conditions as flu and the common cold, men are more likely than women to overstate the severity of their symptoms. Thus rather than being stereotypical "chronic complainers," women may be underreporting their sickness.

Men are more likely than women to engage in high-risk behavior, such as smoking, violence, or unsafe driving (Stillion, 1995). Likewise, risks associated with work roles may be higher for men (Krieger and Fee, 1994; Woods, 1995). The health effects of women's employment outside the home are mixed. There is evidence that women's health benefits from employment, but such work may also force women to carry the stressful "double burden" of paid employment and work at home (Bellaby, 1999). Many women's income is essential for their subsistence and that of their children; paid employment may give them not only greater resources for health and well-being, but also greater control over household resources. Gender-specific barriers to women's good health may be, at root, caused by inequality and relative lack of power. Those women who experience ill effects from paid employment are most likely to be those who are underpaid and overworked, who bear unequal burdens of work in the home, and who experience discrimination in the allocation of household resources (see Doyal, 1995). This
inequality thesis is corroborated by a comparative analysis of men's and women's health in the 50 states, which found that women experience higher mortality and morbidity in states where they have lower levels of political participation and economic autonomy (Kawachi et al., 1999b).

The mortality gap between men and women may be narrowing in recent years. The gap may narrow further as women adopt such high-risk behaviors as smoking and enter workplaces with job stressors and other risks, while men, by contrast, improve their health behaviors (Woods, 1995). Death rates due to lung cancer (a disease caused primarily by cigarette smoking) rose among U.S. women by 600 percent between 1950 and 2000, making lung cancer the leading cause of preventable deaths among women (USCDC, 2001b). Although such biological factors as hormonal differences play some role, sociocultural factors linked with differences between men's and women's roles probably account for much sex-linked variation in mortality and morbidity rates (see Bird and Rieker, 1999).

Race and Ethnicity

Patterns of morbidity and mortality also vary among ethnic and racial groups in a society. Some differences may be explained by hereditary factors that are disproportionately characteristic of certain groups. Genetically transmitted sicknesses include sickle-cell anemia (among blacks and some other groups) and Tay-Sachs syndrome (among persons of Eastern European Jewish descent).

Hereditary factors may also combine with sociocultural factors to produce ethnic differences in morbidity and mortality. For example, Mexican Americans are more likely to have inherent lactose intolerance than Anglo Americans; lactose intolerance combined with social factors (such as higher rates of poverty) produces serious nutritional deficiencies (Schreiber and Homiak, 1981: 283). Similarly, genetic tendencies may be a component factor that, together with environmental factors and changed diet, leads to disproportionately high and increasing rates of obesity and diabetes among Mexican Americans (Guendelman, 1998).

Patterns of variation are linked indirectly to ethnicity through other factors: nutrition, housing and sanitary living conditions, employment and types of occupation, family patterns, and lifestyle. For example, inner-city black preschoolers have a higher incidence of childhood lead poisoning than their suburban black counterparts, suggesting that the causal factors are not race but living conditions—in this case, substandard, older housing where the crumbling lead paint may be ingested by young children (Melnick and Rouse, 2001). The following discussion gives some of the highlights of U.S. data about racial and ethnic variation in morbidity and mortality.

African Americans constitute by far the largest racial/ethnic minority in the United States. Epidemiological data referring to "nonwhites" are thus mainly describing blacks. The pattern of health of black Americans is very different from that of white Americans. In 1999, black life expectancy was 71.4 years compared to 77.3 years for whites (USDEHIS, 2001a). These rates represent a dramatic improvement since 1920, when life expectancy was 45.3 years for blacks and 54.9 years for whites. Nevertheless, African Americans are at higher risk for almost every health problem compared to other racial or ethnic groups (Dressler and Bindon, 2000).

One major factor in the lower life expectancy for blacks is their high rate of infant mortality. Figure 2.2 shows differences in infant mortality between white and nonwhite Americans between 1920 and 1999. Even though infant mortality has been steadily declining for both groups, the rate for blacks is more than twice that of whites (USCDC, 2002b). Infant mortality is especially high among newborns with low birth weight or very young mothers or both. The rate of live births of babies with low birth weight (less than 2,500 grams) was 12 percent for blacks compared with 6 percent for whites; 38 percent of black mothers did not receive prenatal care until after the first trimester of pregnancy, compared to 20 percent of white mothers (Anderson et al., 1987). These factors are clearly linked with socioeconomic factors. Low birth weight is often the direct result of poor maternal nutrition and inadequate prenatal care. Maternal tobacco and other drug use also contribute to poor infant health among African Americans. Fetal alcohol syndrome is also disproportionately higher among African Americans and Native Americans compared to other ethnic groups (Collins, 1996). Compared to other industrialized countries, the U.S. infant mortality rate is appallingly high, largely due to this country's failure to address the problems of poverty, especially nutrition and health care.

The proportionately higher rates of hypertension and hypertensive heart disease among blacks compared with whites illustrate the complexities of causal factors with which epidemiological studies must contend. Factors accounting for these differences include genetic factors in susceptibility, greater stress due to racism, lower socioeconomic status, less access to good medical care, and a higher rate of obesity among blacks (Krieger, 2000). The genetic susceptibility explanation seems doubtful, because cross-cultural studies found no such patterns among genetically comparable peoples of Africa (Dressler and Bindon, 2000). Whereas American blacks show a rapid increase in blood pressure after about the age of 24, African blacks living in tribal communities do not have increased blood pressure as they age. Their migration to industrialized, urban settings, however, does promote increased blood pressure (Janes, 1986). The lower socioeconomic status of many blacks and other racialized ethnic groups accounts for much of the health inequality (Howard et al., 2000; Smith et al., 2000). Even after taking social class
into consideration, however, some significant differences remain (Crimmins and Saito, 2001; Smaje, 2000). Such factors as stress produced by racism, residential segregation, and inferior quality of these residential spaces, which influence even the more affluent blacks, may explain some of these remaining “racial” differences (Adler et al., 1999; Krieger, 1990, 2000; Schulz et al., 2000; Smaje, 2000; Smith et al., 2000; Williams, 2000).

Hispanics (among them, Mexican Americans, Puerto Ricans, and Cuban Americans) are the fastest growing sector of the U.S. population. Some “Hispanic” groups are at increased risk for diabetes, hypertension, tuberculosis, AIDS, alcoholism, cirrhosis, specific cancers, obesity, and violent deaths. Among subgroups of Hispanics, Puerto Ricans have the poorest health, due mainly to lower socioeconomic status, lower education levels, and an extremely high (60 percent) rate of female-headed households. An epidemiological paradox, however, is that Hispanic women have favorable birth outcomes (that is, rates of low-birthweight babies and of infant mortality that are more similar to Anglo women’s than to blacks’), despite the fact that they are impoverished, poorly educated, and medically underserved (Guendelman, 1998).

Some American ethnic groups comprise individuals whose families came to the United States in different waves of immigration. Comparative studies thus have the potential of identifying which factors in sickness and death are due to the particular living situation and lifestyle of a group in the United States compared with the country of origin. For example, although the rate of tuberculosis among Chinese Americans is higher than among the general population, it is nevertheless lower than in the old country. Similarly, both first- and second-generation Chinese Americans have more coronary heart disease than Asian Chinese; diet and stress are both implicated (Gould-Martin and Ngin, 1998). An epidemiological paradox, however, is that Hispanic women have favorable birth outcomes (that is, rates of low-birthweight babies and of infant mortality that are more similar to Anglo women’s than to blacks’), despite the fact that they are impoverished, poorly educated, and medically underserved (Guendelman, 1998).

In the last three decades of the twentieth century, both the volume and sources of new immigrants to the United States changed dramatically. By the year 2000, only 15 percent of the foreign-born population of the United States had come from Europe; far more, at least one-fourth, had come from Asia (U.S. Census Bureau, 2000a). Thus, the diversity of racial and ethnic minority groups complicates interpreting epidemiological data. For example, in order to understand health-related data for blacks and Asian Americans in Minnesota, we would need to realize that many of them are Somali and Hmong refugees, whose ways of life and illnesses reflect both their native cultures and the traumas of the refugee experience (see Faust et al., 1998).

Demographic transition refers to the changes populations undergo (usually in the course of so-called modernization) as they move from a situation of high mortality and fertility to a stage in which mortality has declined but fertility is still high. The third phase is characterized by low fertility and mortality (Omran, 1971). Native American groups have experienced more decline in mortality than in fertility, and the decline in mortality is largely due to the reduction of epidemic infectious diseases. The resulting rates of morbidity and mortality, at this stage of transition, reflect a shift in causes of death. Between 1950 and 1999, overall mortality rates for Native Americans declined dramatically, especially mortality due to influenza, pneumonia, and certain diseases of early infancy. At the same time, mortality due to accidents, violence, and alcohol-related diseases remained relatively high, and deaths due to diabetes, lung cancer, and chronic respiratory disease have increased (USDHHS, 2001a).

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There thus appears to be considerable racial and ethnic diversity in the United States in rates of mortality and morbidity, but precise figures are not available because national epidemiological data do not differentiate among white ethnic groups or nonwhite ethnic groups (for studies on minority groups’ health and immigrant groups’ health issues, see Hogue et al., 2000; Loue, 1998; Polednak, 1989). Most differences, however, appear to be largely due to social-structural factors, especially class. Nonetheless, purely materialist explanations (that is, those that focus on the impact of different material conditions on health differences) may not always completely explain ethnic differences in such health indicators as infant mortality (Andrews and Jwson, 1993). Cultural factors also influence diet, reproduction, and such health-related behavior as smoking and drinking. Yet cultural explanations of health inequalities should not become deterministic ones that emphasize differences between cultural groups and obscure what humans have in common, while neglecting within-group cultural diversity (Karlsen and Nazroo, 2000).

Social Class

Social class is probably one of the most useful shorthand indicators of a person’s power. Class is usually measured by income, education, or occupation; wealth (or durable income) is more difficult to measure but also very useful (on issues of measurement and indicators, see Lynch and Kaplan, 2000; Robert and House, 2000). Generally, a consistently significant relationship exists between socioeconomic position and health (Link and Phelan, 1995). The lower the social class, the higher the rates of morbidity and mortality. This relationship is not surprising,
considering the important part that our socioeconomic situation plays in the quality of our everyday lives.

Numerous social epidemiological studies have documented, for many countries—both developing and economically well off—that socioeconomic disparities are correlated with differentials in death from a wide range of causes, longevity, and morbidity rates for a wide range of illnesses (Lynch and Kaplan, 2000; Marmot, 2002; Wilkinson, 2000). Inequality affects people's health even before they are born, as reflected in rates of low-birthweight babies, infant mortality, and under-five mortality. These disparities in childhood may continue to affect people's health in adulthood, even if they change socioeconomic position (for an overview of key studies, see Kawachi and Berkman, 2000; Kawachi et al., 1999a; Link and Phelan, 2000; Lynch and Kaplan, 2000; Marmot, 2000; Robert and House, 2000).

Increasingly, researchers are finding that these within-society differences in mortality rates and other health indicators are not attributable mainly to absolute material standards or absolute poverty. Conditions of poverty obviously have a direct relationship with causing disease and death; absolute poverty results in malnutrition, high rates of infection, unsanitary living conditions, and so on. However, the degree of social inequality within the society appears to be a more powerful explanatory factor than the degree of absolute poverty (Wilkinson, 2000).

A major study in England (the Whitehall Study) found pervasive differences by social class (defined by occupation) in morbidity rates for a wide range of diseases (Marmot et al., 1987). A follow-up study 25 years later was able to identify the actual ages at death and cause of mortality for the same subjects. It found the same gradient by social status for most causes of death: Nearly regardless of cause of death, persons in the lowest "class" occupations died younger than persons in the middle classes, and those in the middle classes died younger than persons in the top class (Marmot, 2000).

The specific features of social inequality that might account for this strong correlation with morbidity and mortality are hotly debated. Is it due to the "hidden injuries of class," such as humiliation and disrespect? Is, perhaps, race and ethnic inequality an intervening variable that explains most of income inequality? Is the correlation with morbidity and mortality due to psychosocial factors, such as insecurity, stress, or sense of hopelessness or lack of control? How are class-linked health behaviors involved? Is any of the differential due to variations in access to the health care system? Several chapters of this book address these questions further.

How serious is socioeconomic inequality? In the United States and the United Kingdom, socioeconomic disparities have increased (Graham, 2000; Navarro, 2002a). Despite its national rhetoric of equality in the "land of opportunity," since 1980, the United States has seen growing inequality in wealth, income, consumption, and proportional share of tax burden. Of all "wealthy" countries, the United States now has the greatest inequality between its upper and lower classes. In 1973, mean income of the highest income quintile (fifth) of all American households was 10.3 times that of the lowest income fifth, and 2.5 times that of the middle-income quintile. By 1997, the disparity had grown to 13.9 times the income of the lowest income fifth, and 3.3 times that of the middle-income quintile. The incomes of the very wealthy (top 5 percent) American households grew even more dramatically, relative to all others' (U.S. Census Bureau, 2000b). The Congressional Budget Office (CBO) found even more marked increases in inequality of after-tax income, due largely to tax reductions that favored the rich (and this was before the further tax cuts of 2001). The CBO data showed that, as of 1997, the highest income 20 percent of the population, in aggregate, received as much after-tax income as the entire other 80 percent (CBO, 2001).

Social class stratification shows up more dramatically in measures of household wealth (that is, net worth), and wealth is more likely than income alone to be linked with access to power and privilege. In the last two decades, the United States has experienced considerable consolidation of wealth in the hands of its richest citizens. By 1998, the wealthiest 1 percent of all households owned 42.1 percent of all wealth—the greatest concentration of wealth at the top since 1929 when the Great Depression began (Wolff, 2000).

Other economically developed (OECD) countries had much greater equality: Sweden, Finland, Belgium, Norway, Denmark, and Luxembourg all had minuscule levels of inequality, compared to the United States. Other European countries were somewhat more unequal but still considerably less so than the United States. Of OECD countries, only the United Kingdom approached the high level of inequality characterizing the United States, and disparities between high- and low-income groups have continued to grow, although not as fast as in the United States (cited in Navarro, 2002a: 70).

Differences in mortality between socioeconomic groups have generally increased in the United States since the 1960s (Kennedy et al., 1996). In recent decades, similar increases in mortality-rate disparity have been noted between socioeconomic groups in other countries (for reviews of this literature, see Deaton, 2002; Robert and House, 2000). In Scandinavia, long-standing illness is reported more frequently among blue-collar than white-collar groups (Rahkonen et al., 1993). There is a relationship between people's self-assessment of health and their income levels. The higher the income level, the more likely self-assessed health is excellent; the lower a person's income, the more likely health will be self-rated as poor (Blaxter, 1990; Graham, 2000).

These data illustrate that differences that at first appear to be individual are often actually social variations. Although smoking is an individual behavior, for example, those in the lowest socioeconomic positions are almost three times as likely to smoke as those in the highest positions (Marmot et al., 1987), which may partially explain the higher rates of respiratory diseases among the lower classes. Similarly, alcohol consumption is highest among the lower classes, as is the incidence of obesity (Dutton, 1986). Deaths due to accidents also increase as socioeconomic status decreases (Quick, 1991).

The Black report, a famous British study on the relationship between class and health, concluded that lack of personal control over one's life was an important
factor linking low social status with poor health (Black, 1980; see also Gray, 1982). A 1987 update of the Black report not only documented persisting class differences in morbidity and mortality, but also showed a widening gap in health between the upper and lower classes (Townsend, 1990). Similar conclusions can be found in the Acheson Report (Independent Inquiry into Inequalities in Health, 1998).

Syme and Berkman (1976) have argued that this lack of control and other adverse features of lower-class life create a “generalized susceptibility” to disease (see also Blaxter, 1990). The conditions of lower-class life include living and working in more polluted and crowded environments, exposure to higher noise levels and risks of accidents, and inadequate housing and transportation. Medical care for the poor is less accessible and of lower quality than that for higher social classes. Furthermore, health problems of the poor are typically more serious, complex, and difficult to treat (Dutton, 1986).

As the Black report shows, for England, improved access to medical care does not by itself significantly reduce social class differences in health. Scandinavian countries, which provide citizens with excellent access to high-quality health care, nonetheless exhibit some class differences in health. A study of the Scandinavian situation concluded that “health care without social welfare and economic policy will be ineffective in reducing social inequalities in health” (Rahkonen et al., 1993: 77). To seriously reduce the effects of social class on health requires far more basic economic and social reforms (Hurowitz, 1993).

Measuring social class variables is difficult. Occupation is one indicator of social class, but occupational categories are often broad and not neatly tied with a single social stratum. For example, how would the classification scheme distinguish between a farmer who owns 50 acres and cultivates them for family subsistence, and a “farmer” who owns 4,000 acres and hires numerous workers to cultivate them (Susser et al., 1985)? Measures of class using occupation may use the husband’s occupation as a measure of the wife’s socioeconomic status, but such measures do not indicate whether a husband is, indeed, sharing his socioeconomic advantage with his wife and children. As the occupational structure of society changes, older occupational categories may no longer be useful, and many persons may be excluded or mismeasured (Blaxter, 2000). Most researchers currently use some measure of income, education, and/or wealth to identify degrees of socioeconomic inequality.

Despite such definitional problems, studies of many societies document a consistent relationship between social class and health. Rates of mortality and social class are clearly linked, but the relationship between class and specific causes of death is not so consistent, and may change over time. The epidemiology of poliomyelitis also illustrates how the relationship between class and mortality from specific diseases may change over time. In lower-class areas, characterized by overcrowding and poor sanitation, children often developed antibodies and hence immunity to polio after being exposed to the virus early in life. Thus, the poor had lower risk of paralysis and death than the more “sheltered” middle and upper classes. When the polio vaccine was introduced in the mid-1950s, however, this relationship between class and vulnerability to the disease changed. Those upper- and middle-class children who were first vaccinated were the first to benefit. An education campaign eventually resulted in the widespread acceptance of immunization, but pockets of underimmunized persons still exist in the United States, particularly among lower-class minority groups (Susser et al., 1985: 229–231).

Thus, the relationship between specific kinds of morbidity or causes of mortality may not follow the usual relationship with social class and may shift over time. At present, however, there are no major causes of mortality for which death rates are not higher among lower-class than upper-class persons. On the whole, the lower classes bear higher burdens of mortality and morbidity (Blaxter, 1990).

Even when data show a strong correlation between two variables, such as social class and health status, the direction of causality is sometimes difficult to determine. Does variable A produce variable B; does variable B produce A; or are they related in even more complex ways, mutually influencing each other or indirectly influencing each other through some third (intervening) variable? For example, do lower social status and the poor living conditions that accompany it produce poor health, or does poor health lead to downward mobility and lower income, or do both situations interact (Dutton, 1986)? The explanation that poor health results in downward mobility has been called the drift hypothesis, which suggests that those who acquire disabling diseases “drift” down the social ladder in the course of their lives (Lawrence, 1958).

Some evidence hints that serious illness in childhood is related to downward mobility in later life. Boys who were seriously ill are more likely than those who were healthy to gravitate to a social position below that of their fathers (Wilkinson, 1986b). Downward drift may be linked only to specific “diseases” such as schizophrenia (Adler et al., 1999), so the drift hypothesis does not explain satisfactorily most of the relationship between class morbidity and mortality (Graham, 2000; Marmot, 1996; Wilkinson, 1996). While the drift hypothesis may not sufficiently account for the relationship between class and health, it does remind us that health inequalities between individuals are the outcome of “cumulative differential response to adverse material conditions and to behavioral and psychological risks” (Graham, 2000: 15). Young bodies influenced by such adverse conditions may have health problems later in life.

Some of the relationship between social inequality and specific kinds of morbidity and mortality may be more artifactual—a product of the way the variables are measured—than real. An extensive critique of the Black report argues that the correlation between social class and mortality due to chronic degenerative diseases is essentially artifactual (Bloor et al., 1987). For example, the perceived social class of the deceased person influences whether the cause of death was classified as heart disease. The significance of such artifacts in the correlations between social class and health is not clear. Other reviews (compare Graham, 2000; Marmot et al., 1987) consider artifacts to be relatively unimportant in explaining the social class differences in health. They observe that a large number and variety of
studies, done in several societies, indicate a strong relationship among class, health, and death.

SUMMARY

There are observable patterns to the frequency and incidence of mortality and morbidity. As opposed to the clinical-medical model, which focuses on individual bodies and a limited number of causal factors in disease, the epidemiological approach looks at social patterns of morbidity and mortality and the complex “web of causation.” Epidemiological studies show that many patterns of morbidity and mortality are connected with social variables, such as the kind of society, race, ethnicity, age, social class, and gender. These epidemiological variables are also rough indicators of the social distribution of power and of the different power relationships people experience. However, epidemiological studies are limited because of their tendency to decontextualize health and illness, and their purely quantitative orientation. Yet with its broad generalizations, social epidemiology sets the stage for a more refined analysis of the ways a society produces, defines, experiences, and treats sickness and death.