

Chapter 6

Health Inequalities, General Trends in Mortality and Morbidity, and Associated Factors

Measurement of Inequality of Mortality and Health

Introductory Methodological Notes

All measures of health status are ultimately derived from observations of individuals. At the field level we have such measures as self-assessed health status, report of a specific disease, record of a particular death, or an individual's test on a biomarker, such as blood pressure or serum cholesterol. The observations for individuals are combined and summarized to represent subnational geographic areas, demographic or socioeconomic groups within countries, or national populations. The summary measures, whether they are percentages, averages, or rates, apply to groups. A problem arises when the measures that are based on groups are assumed to represent individuals. The analysis becomes especially problematic when the units analyzed are geographic areas and inferences are being made about individuals from the analysis for these geographic areas.

Individuals differ from one another in their health status, and they tend to be disproportionately clustered in one health status or another. A population described by health-status variables then tends to be characterized by varying degrees of health inequality. There is health inequality among population groups within countries and subnational geographic and residence areas, and among countries and world regions. The basic methods of measuring inequality of the health status of groups within countries are generally the same as the methods used to measure health inequality among whole countries and world regions.

Some simpler measures of health inequality describe single populations independently, which may then be compared with the results of the same group measure for other populations; or are designed to link two populations directly in the computation of the measure. Another group of more complex measures of health inequality links health status or health events with the demographic and socioeconomic characteristics of persons such as age, sex, race, ethnicity, marital status, educational level, and income class, and seeks to describe the extent of the association and even

the causal relation between health status and these characteristics. Most measures of health inequality were developed in order to analyze differences in health status for such socioeconomic subgroups in national populations. The first group of measures, extended to include age, is described in the present chapter, and the second group of measures is described in Chap. 7. It is important to distinguish between measures of inequality in the distribution of a health measure, such as among ages, geographic areas, and general population groups, and measures of the differences in a health measure among various socioeconomic groups in a population for the purpose of determining the degree of causation between them. The latter may involve not only different measures but also causal inferences, value judgments, and differences in interpretation. While most recent research has focused on variations in the health status of socioeconomic groups within countries, in the discussion that follows in this chapter, I focus mainly on variations in health status among countries and among general populations. As is usual in demographic or epidemiological analysis, the conclusions drawn about variations and trends in health inequality among populations may differ depending on the measure used.

Measures of Inequality Within Countries

Measures That Describe Individual Populations

The reader is familiar with the summary measures of central tendency and dispersion that are used for analyzing the mortality and health characteristics of countries and comparing a group of countries with regard to these characteristics. They include the mean (x_m), median (x_{md}), and mode (x_{mo}), the standard deviation (σ) and interquartile range (IQR), and the standardized versions of the latter two measures, namely the coefficient of variation (CV) and the relative interquartile range (RIQR). The mean, median, and mode are used to compare the level of the distributions and the other measures are used to compare the pattern of the distributions.

Formulas for these measures were given in Chaps. 3 and 4; only the measures of dispersion are repeated here. For the interquartile range and the relative interquartile range:

$$\text{IQR} = x_{.25} - x_{.75} \quad (6.1)$$

where $x_{.25}$ and $x_{.75}$ refer to the value of the variable (e.g., age at death) at the first and third quartile, and

$$\text{RIQR} = (\text{IQR} \div x_{md}) * 100 \quad (6.2)$$

where the IQR is divided by the median, or second quartile, so as to adjust for the level of the distribution and “isolate” the degree of dispersion. For the standard deviation and the coefficient of variation,

$$\sigma = \sqrt{\left[\left(\sum x_i^2 \div n \right) - x_m^2 \right]} \quad (6.3)$$

$$CV = (\sigma \div x_m) * 100 \quad (6.4)$$

where, to derive the coefficient of variation, the standard deviation is divided by the mean. (To simplify the computations, the mean deviation ($= \sum_i (x_i - x_m) \div n$) may be used instead of the standard deviation; its standardized form is derived by dividing it by the mean of the distribution.) As noted earlier, the relative interquartile range and the coefficient of variation may be used to evaluate the differences in the shapes of the different distributions. It is important to use relative measures in comparing distributions that have very different levels of mortality or morbidity, such as distributions for two very widely separate dates or for different animal species.

Some measures adjust for the difference in the level of distributions being compared in a different way. Distributions can be “scaled” for comparison of their patterns by dividing or multiplying the elements in each distribution by a representative constant derived from the same distribution, such as its sum, mean, or median. After such scaling, the total, mean, or median of each distribution is reduced to a value of one. The same parameter, i.e., total, mean, or median, must be used for each distribution being compared. An alternative method is to multiply the elements of each distribution by the ratio of the mean or median of one of the distributions (“the standard distribution”) to the mean or median of each of the other distributions. In effect, the “standard” distribution is left unchanged. Now all the distributions being compared have the “same” general level, scaled to the mean or median of the standard distribution.

Scaling is most appropriately applied in comparisons of age distributions of different species, where the lengths of life of the species being compared are often sharply different, e.g., deaths of humans and deaths of mice. Scaling may arguably be applied in comparisons of age distributions of two human population groups with sharply different life expectancies.

Comparing absolute and relative differences of health measures. In this and earlier chapters I describe many summary measures of the health status of population groups, e.g., the percents in categories of self-reported health, mean age at death, age-adjusted death rate, disease prevalence ratio, and life expectancy. In comparing such measures for two or more population groups, there are several choices. First, one can take the absolute difference between the measures. For this purpose one group must be selected as a standard or basis of comparison. One can select as the standard the value for the “leading” or “best” group (e.g., age-adjusted death rate in State A), to be compared with the value for the lagging group (e.g., age-adjusted death rate in State B). The absolute difference between the leading value and the lagging value provides an indication of the distance the lagging group has to go in order to close the gap between the two groups. Alternatively, the leading value may

be a future target value (e.g., life expectancy in 2015) and the other value may be the figure for the current date (e.g., life expectancy in 2005). The absolute difference between the target value and the current value provides an indication of the progress required to reach the target.

Often the choice of the target level or the leading group is arbitrary. The classification system used for the groups being compared (e.g., races) affects the choice of the leading group. For example, the amount of progress in reducing the differences between the age-adjusted death rates of the races would vary by the degree of disaggregation of the races (e.g., Asian-Americans as a group vs. Japanese, Chinese, Koreans, etc.). With the passage of time, the levels achieved by the various groups may change, the identity of the leading group may change, and the form of the computation may have to be changed.

Differences between groups can also be measured in relative terms (although some analysts object to taking ratios of rates in this way). The deviations from the leading value or target value can be expressed as a ratio of the baseline value or leading value:

$$\frac{\text{Target value} - \text{Baseline value}}{\text{Base line value}} * 100 \quad (6.5)$$

For example, given a life expectancy for black males in 2002 of 68.8, and a target value in 2010 of 75.5, the required progress is

$$\frac{75.5 - 68.8}{68.8} * 100 = 9.7\%$$

If the figure for black males in 2002 (68.8) is being compared with the figure for white males (75.1) in 2002, the percent shortfall of the black male figure is 8.4%. Here the base is the white male figure:

$$\frac{68.8 - 75.1}{75.1} * 100 = 8.4\%$$

Another measure, the progress quotient, shows the percent of the targeted change that has been achieved; that is, the difference between the current value and the baseline value is expressed as a proportion of the difference between the targeted value and the baseline value:

$$\frac{\text{Current value} - \text{baseline value}}{\text{Target value} - \text{baseline value}} * 100 \quad (6.6)$$

For example, given a life expectancy for black males in 1998 of 67.6, a current figure in 2002 of 68.8, and a target value in 2010 of 75.5, the progress quotient is:

$$\frac{68.8 - 67.6}{75.5 - 67.6} * 100 = 15\%$$

Normally this measure ranges from 0.0% to 100.0%, the first figure implying no progress and the second, achievement of the target. The result would be negative, however, if the current value moves away from the target. Because of the often small size of the changes, the progress quotient may change irregularly from year to year, and if the target value is only slightly greater than the baseline value, the instability in the measure gives the results an uncertain meaning. The current value may already have exceeded the target value, and in this case the result would exceed 100%.

Absolute and relative measures of differences may lead to different conclusions about changes over time. That is, equal relative progress for different groups does not correspond to equal absolute progress for them. The absolute difference between two groups in some health measure may decline while the relative difference may increase, depending on how the denominator is selected (as in the example below). In the calculation of the relative measure, the base chosen for calculating the percent difference at some date is usually the value for the group taken as the standard but this does not have to be the case. It may be better to use a more neutral and a more stable figure in the denominator, such as the value for all groups combined that are being compared. The percent differences in such a case would vary with the base, resulting in a difference in the measured health of the two populations:

A. Comparing the absolute and relative differences between absolute changes in percents:

$$21.5 - 4.2 = 17.3$$

$$10.8 - 1.9 = 8.9$$

$$\text{Absolute difference} = +8.4 \text{ percentage points}$$

$$\text{Relative difference} = +94\%$$

B. Computing the relative difference between the relative changes in percents:

$$\frac{21.5 - 4.2}{4.2} = +312\% \quad \frac{10.8 - 1.9}{1.9} = +368\%$$

$$\text{Relative difference} = 312/368 = -15\%$$

Using the average for all groups in the denominator:

$$\frac{21.5 - 4.2}{6.8} = +154\% \quad \frac{10.8 - 1.9}{3.2} = +178\%$$

$$\text{Relative difference} = 154/178 = -45\%$$

Here the relative measure reverses the direction of the absolute measure and the shift in the base of the percents changes the measured relative difference from -15% to -45% . Usually in making comparisons of general summary rates or ratios, the rates or ratios are age-adjusted before the comparison in order to eliminate the effect of differences in age composition from distorting the interpretation of the difference between the rates or ratios.

Measures That Link Two Populations

The above measures are computed for single populations, which are then compared with one another in order to describe differences in their levels of health. Some measures directly describe the differences in health between two populations, being computed from data linking the populations. The populations being compared may be segments of the population of countries or whole countries. The three most widely applied measures of health inequality that link two populations are quintile analysis, the index of dissimilarity, and the Gini Ratio, with its graphic counterpart, the Lorenz curve. The index of dissimilarity, the Gini Ratio, and the Lorenz curve are mathematically related to one another and so can be considered a single package of measures of inequality.

Quintile analysis. In quintile analysis, the question posed is, what percent of the aggregate of the health characteristic or percent of the population with the health characteristic is associated with each fifth of the population? As a hypothetical example, the “lowest” quintile of the population 25 years and over may be associated with 24% of the aggregate of the body mass indexes (BMI, a measure of body weight in relation to height) of the members of that population in a given year while the “highest” quintile of that population may be associated with only 16% of the BMI aggregate. In other words, the health indicator BMI is not distributed evenly in the adult population, i.e., 20% of the “weight” for each fifth of the population. One could also use quintile analysis to characterize a population in terms of the distribution of blood pressure, serum cholesterol, or other quantitative health variable that can be ranked numerically, that is, interval variables.

To do a quintile analysis relating to weight, one would rank each individual in the population according to their body mass index, and count off the top 20% of the population, then the 2nd 20% of the population, and then the 3rd, 4th, and 5th 20%. One would then take the percent of the total aggregate of the BMIs for each fifth of the population ranked according to BMIs. The relation of population and aggregate BMIs in percents could then be stated for each fifth of the population for a series of years and represented in a 100% surface chart with each of the four graphed lines in the chart distinguishing each quintile’s shares over time (Fig. 6.1).

Index of dissimilarity. The index of dissimilarity measures the inequality of two distributions with respect to some variable. The formula of the index of dissimilarity is

$$ID = 1/2 \sum (p_i - p_a) \quad (6.7)$$

where p_i and p_a represent the percent of the health variable (e.g., obese persons and diabetic persons) in relation to their sum (all obese persons and all diabetic persons) for each of two distributions representing categories of subnational areas (e.g., census tracts of a city, states) or some demographic or socioeconomic variable (e.g., age classes).

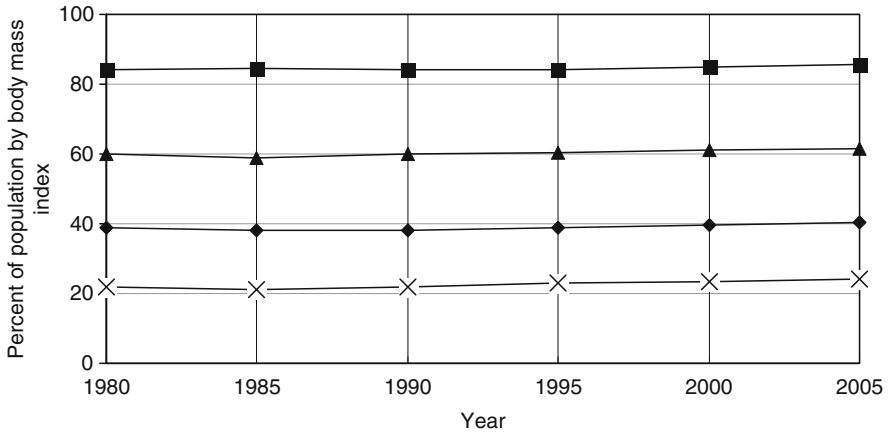


Fig. 6.1 Chart illustrating quintile analysis of body mass index in a population 18 years of age and over (Assumes that the body mass index of the members of a population or a representative sample of them is known every fifth year over 25 years. The population is distributed into fifths according to body mass index, one fifth with the lowest BMIs, one fifth with the next highest BMIs, and so on until the fifth of the population with the highest BMIs. The proportion of the BMIs total corresponding to each fifth of the population is then plotted for each year. These are hypothetical data)

Examples of such distributions that may be compared are numerous: Endogenous deaths by age vs. exogenous deaths by age; overweight persons by age vs. non-overweight persons by age; diabetic persons by age vs. nondiabetic persons by age; HIV/AIDS cases in States vs. single adult males without HIV/AIDS in States; and self-reported health (e.g., poor or fair vs. good, very good, or excellent) for census tracts within a city. The index of dissimilarity in this last example is calculated to determine the degree of inequality in the geographic distribution of healthy vs. unhealthy persons in a city. Indexes of dissimilarity may then be calculated for other cities to compare the degree of geographic concentration of persons with good or bad health in a group of various cities. This measure can be calculated for any variables, including non-quantitative, “non-rankable” attributes, i.e., nominal variables.

To derive the index, the absolute numbers in each distribution (e.g., obese persons, diabetic persons) are converted to percents of their totals, the differences between percents for corresponding categories (e.g., census tracts) are taken, these differences are summed over all areas, and then the sum is reduced by half. Table 6.1 provides an illustration of the calculation of the index of dissimilarity. It compares, with hypothetical data, the distributions of adult obesity and diabetes for the census tracts in a county. The index is low because obesity and diabetes tend to be distributed geographically in a similar fashion.

The index of dissimilarity (ID) varies between zero and one, zero representing perfect equality between the distributions and one representing perfect inequality between them. With perfect equality every geographic area or every age has the

Table 6.1 Calculation of the index of dissimilarity between census tracts, comparing the distribution of the number of diabetes and the number of cases of adult obesity: Hypothetical data

Tract	Cases of	Cases of	Percent of total		Absolute difference (5) (3)–(4)
	diabetes (1)	adult obesity (2)	Diabetes (3)	Adult obesity (4)	
All tracts	562	1760	100.0	100.0	–
A	75	293	13.3	16.6	3.3
B	143	495	25.4	28.1	2.7
C	125	361	22.2	20.5	1.7
D	98	357	17.4	20.3	2.9
E	121	254	21.5	14.4	7.1
Sum (without regard to signs)					17.7
Index of dissimilarity = 1/2 sum					8.8

identical percent for the two variables being compared; for example, the share of obese persons in each county of a state is the same as the share of diabetics in each county of the state, or the proportion of persons in excellent, very good, or good health is the same as the proportion of persons in fair or poor health in every census tract of a county. Perfect inequality requires extreme conditions, such as that all the healthy persons live in one census tract and all the unhealthy persons in other census tracts of the county. The value of ID indicates the amount by which the proportions in the first distribution would have to be shifted upwards for negative differences and downwards for positive differences to make the two distributions equal.

The index of dissimilarity has a number of limitations, such as being affected by the number of categories in the distribution and the choice of classes of the variable being compared (e.g., 5-year age groups vs. 10-year age groups; census tracts vs. health zones), and being limited to comparing only two variables at a time (e.g., healthy vs. unhealthy persons).¹

Gini Ratio and Lorenz curve. The Gini Ratio (Gini index of concentration/Gini coefficient) is a summary measure of the inequality of distributions similar to the

¹We may wish to compare distributions of rates by age (e.g., death rates) rather than absolute numbers (e.g., deaths). The index of dissimilarity may be adapted for this purpose. We simply treat the age-specific rates as absolute numbers in applying the formula; i.e., sum the rates, compute the percent distribution of the rates, and take the differences between the percents at each age. Converting the rates to percents tends to dampen the variation in the original data considerably. In deriving the percent distribution of the rates, the rates are multiplied in effect by the reciprocal of the total of the distribution.

In an alternative handling of age-specific death rates, they may be weighted by population before the sums and percent distributions are calculated. This is to allow for the fact that the rates at the highest ages dominate the distribution. The rates are in effect converted to absolute numbers of deaths by this weighting process. The weighting process will modify the resulting indexes of dissimilarity greatly.

index of dissimilarity and related to it. The Gini ratio like the ID ranges from zero to one. A ratio of one indicates perfect inequality and a ratio of zero indicates perfect equality.

The calculation of the Gini ratio may best be described geometrically. First, calculate the cumulative shares of an aggregate health measure corresponding to cumulative shares of persons. Next, plot the results on a rectangular grid, with persons from 0 to 100 (percent) on the x-axis and with the aggregate health measure from 0 to 100 (percent) on the y-axis.² This line is designated the line of inequality, or Lorenz curve. Third, draw a straight (diagonal) line from (0, 0) to (100, 100), representing a perfectly equal distribution of population and health. The Gini ratio represents the proportion of the total (triangular) area under the diagonal line that lies in the area between the diagonal line and the Lorenz curve (Fig. 6.2). In the

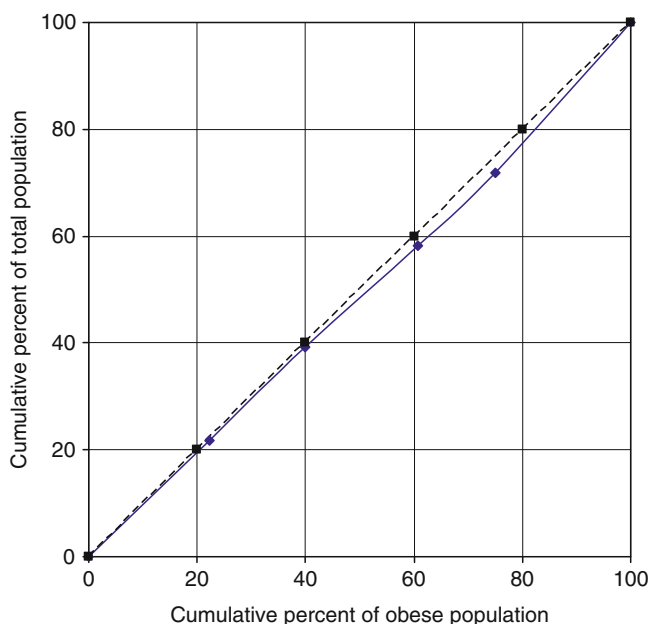


Fig. 6.2 Lorenz curve relating the income distribution of the population 18 years of age and over and the income distribution of the obese population 18 years and over: United States 2006. (The cumulative percent of the population is paired with the cumulative percent of obese persons, ranked according to household income, and plotted on a square grid. A diagonal line is drawn from (0, 0) to (100.0, 100.0), representing an equality of the distribution of population and obese persons according to income. The deviation of the line of inequality from the diagonal line represents the degree of inequality between the general population and obese persons according to income status. The figure shows a minimal degree of inequality) (Source: Adaptation by author of NHIS data for 2006)

²Adapted from Siegel, J. S. (2002). *Applied demography, applications to business, government, law, and public policy* (pp. 26–27), San Diego, CA: Academic Press (Fig. 1.5, p. 27).

figure, if B is assumed to represent the area under the Lorenz curve and A is assumed to represent the area between the diagonal line and the Lorenz curve, the Gini ratio is calculated as the share that A constitutes of (A + B). The maximum vertical distance between the line of equality and the Lorenz curve is equivalent to the index of dissimilarity.

The computing formula for the Gini ratio is,

$$\text{Gini Ratio} = \sum_i P_i Q_{i+1} - \sum_i P_{i+1} Q_i \quad (6.7)$$

where P_i and Q_i represent the cumulative proportions of individuals whose distributions with respect to the health variables are being compared.³ The health variable can be a non-quantitative, nominal variable. Examples of such health variables are healthy persons, age at death, number of cases of a chronic illness, waist-hip ratio, body mass index, and blood pressure. A specific example would be a comparison of the distribution of high blood pressure of the general population and high blood pressure of children 5–14.

Measurement of International Inequality

Inequality of international health refers to the unequal distribution of good and bad health among countries as units. A variety of measures of inequality are used for measuring international health inequality similar to those used for intranational inequality in health and mortality. [Goesling and Firebaugh \(2004\)](#) also suggest that we could develop a world measure of inequality in population health by combining a global measure of inequality in population health within countries and a global measure of inequality in population health between countries or regions. World health inequality (I_w) would then be represented by the sum of the intracountry variation (I_i) and the intercountry variation (I_b):

$$I_w = I_b + I_i \quad (6.8)$$

There are many methods of analysis and many indicators that could be used to represent population health for international comparisons. I follow [Goesling and Firebaugh \(2004\)](#) in selecting life expectancy as the health variable and four measures of health inequality to illustrate the measurement of levels, differences, and trends in international health inequality. Life expectancy is a proxy for many aspects of health and data for it are virtually universally available. The illustration used here defines health inequality as the uneven distribution of life expectancy among world regions considered as units (with China and Japan listed separately).

³An illustration of the computation of the Gini Ratio is given in [Siegel and Swanson \(2004\)](#), Table 6.6, p. 117.

The four measures of health inequality employed here are the Gini coefficient, the Theil index, the mean logarithmic deviation, and the squared coefficient of variation. These measures are usually employed to measure inequality in income and other socioeconomic variables. They can be used to measure inequality for any variables that can be expressed on an interval scale, such as morbidity prevalence ratios, bed-days of activity limitation, and percent dying from a given disease. The formulas for all four indexes, using the notation of Goessling and Firebaugh, are structured as weighted averages of some function of the variable of interest. Here the variable of interest is the life expectancy ratio, defined as the ratio of life expectancy in a particular area (x_i) to the weighted average of life expectancies over all areas ($\sum_i p_i x_i$), the weights being the relative size (or percentage) of the population in each area (p_i):

$$\text{Life expectancy ratio} = r_i = x_i \div \sum_i p_i x_i \tag{6.9}$$

Note that $\sum_i p_i = 1$. In calculating the various inequality indexes, we have to transform the life expectancy ratio into a specified mathematical function of the ratio and then average the transformed values of the life expectancy ratios by the populations in each area. That is:

$$\text{Measure of inequality} = \sum_i p_i f(r_i) \tag{6.10}$$

where r_i is the life expectancy ratio for a country or region and f is some mathematical function of it.

The Gini coefficient is defined as

$$GC = \sum_i p_i r_i (q_i - Q_i) \tag{6.11}$$

where q_i is the proportion of the total population over all areas in which the value of life expectancy is lower than in area i and Q_i is the proportion of the total population over all areas in which the value of life expectancy is higher than in area i .

$$TI = \sum_i p_i r_i \ln(r_i) \tag{6.12}$$

where the symbols r_i and p_i have the same meanings as above and the function of r_i is the product of r_i and the natural logarithm of r_i .

The mean logarithmic deviation is defined as

$$MLD = \sum_i p_i \ln(1/r_i) \tag{6.13}$$

where the logarithm of the reciprocal of r_i is the function of r_i used.

The squared coefficient of variation is defined as

$$CV^2 = \sum_i p_i (r_i - 1)^2 \quad (6.14)$$

where the function of r_i becomes $(r_i - 1)^2$.

If the x_i (i.e., life expectancies) are equal, $r_i = 1$. All of these indexes are standardized at zero when life expectancy is the same for all areas. Each index converges to zero as the life expectancy ratio approaches one ($r_i \rightarrow 1$); in this case there is a greater degree of equality among the areas studied. As the value of the index moves toward 1.0, there is a greater degree of inequality among the areas studied.

Table 6.2 shows the results of evaluating the four formulas for the geographic regions of the world with respect to inequality in longevity in 1980, 1990, and 2000, as calculated by Goesling and Firebaugh. Each measure implies a somewhat different degree of inequality and change in inequality over time. The Gini coefficient gives the least indication of inequality and the other three indexes are rather consistent in showing a major degree of inequality and change in inequality over the last few decades.

The mean logarithmic deviation showed a 21% decline in inequality between 1980 and 1990 and an increase of 26% between 1990 and 2000 (Table 6.2). The other three measures showed similar reversals in direction over the two decades, although the Gini coefficient indicated shifts in inequality from only -13% to $+6\%$. Interregional inequality increased in the 1990–2000 decade mainly because life expectancy declined in sub-Saharan Africa (by 7 percent) as a result of the HIV/AIDS epidemic, and in the Transition Economies (by 2%) as a result of the disorder following the dissolution of the former Soviet Union. Accordingly, while global life expectancy continued to rise during both decades, the global rise in the 1990–2000 decade was small.

Principal Trends and Causes of Mortality

Trends in the United States

Age Changes

Life expectancy at birth in the United States has risen by about 9 years in the last half century and by about 30 years since 1900. Life expectancy at birth was 47 years in 1900, 68 in 1950, and 77 in 2000 (Table 6.3; Fig. 6.3). These numbers reflect an annual average absolute gain of 0.3 year and an annual average rate of gain of 0.5% over the century. The two halves of the century can be distinguished by their very different gains in life expectancy. Major shifts also occurred in the age incidence and

Table 6.2 World and regional trends in life expectancy at birth and in health inequality between regions: 1980–2000

Region ^a	(Both sexes combined)				
	Life expectancy			Percent change	
	1980	1990	2000	1980–1990	1990–2000
World	62.5	65.2	66.4	+4.3	+1.9
Western Europe	73.9	76.1	78.0	+3.1	+2.4
Transition economies	68.1	69.3	68.0	+1.8	–1.9
Western offshoots	73.8	75.5	77.4	+2.3	+2.4
Latin America and the Caribbean	64.7	68.0	70.4	+5.1	+3.6
Middle East and North Africa	59.2	65.0	68.5	+9.7	+5.4
Sub-Saharan Africa	47.6	50.0	46.5	+5.0	–7.0
South Asia	53.4	58.3	62.1	+9.1	+6.5
East Asia (excl. China and Japan)	59.7	65.1	68.5	+8.9	+5.2
Japan	76.1	78.8	80.7	+3.6	+2.4
China	66.8	68.9	70.3	+3.0	+2.0
Index of health inequality ^b					
Gini coefficient	.797	.691	.730	–13.3	+5.6
Theil index	.103	.080	.099	–22.3	+23.8
Mean logarithmic deviation	.107	.084	.106	–21.5	+26.2
Squared coefficient of variation	.200	.155	.185	–22.5	+19.4

Source: [World Bank \(2002\)](#). *World Development Indicators Online Database*: www.worldbank.org/data/onlinebs/onlinebases.htm. Secondary source: [Goesling and Firebaugh \(2004\)](#), Table 6.2, p. 137. Copyright © John Wiley & Sons. Reprinted with permission

Transition economies (22 countries): Former member countries of Soviet Union in Asia and socialist economies of Eastern Europe

Western offshoots (4 countries): United States, Australia, Canada, and New Zealand

Latin America and the Caribbean (29 countries)

Middle East and North Africa (20 countries)

Sub-Saharan Africa (45 countries)

South Asia (8 countries)

East Asia (excluding Japan and China): (13 countries)

^aWestern Europe (19 countries)

^bSee text for definitions

the causal pattern of mortality rates between the first half and the second half of the century. Prevention and control of infectious diseases explain much of the increase in life expectancy at birth in the first half of the century, and reductions in the chronic diseases of later life explain much of the increase in the second half of the century. In general, improvements in nutrition, personal hygiene, public sanitation (i.e., water supply and sewage disposal), and housing conditions account for most

Table 6.3 Life expectation at birth and at age 65, for the United States: 1900–1902 to 2005

Year	Life expectation		Increase over preceding date	
	At birth	At age 65	At birth	At age 65
1900–1902 ^a	49.2	11.9	X	X
1939–1941	63.6	12.8	14.4	0.9
1949–1951	68.1	13.8	4.5	1.0
1959–1961	69.9	14.4	1.8	0.6
1969–1971	70.7	15.0	0.8	0.6
1979–1981	73.9	16.5	3.2	1.5
1989–1991	75.4	17.3	1.5	0.8
1999–2001	76.8	17.7	1.4	0.4
2005	77.8	18.7	1.0	1.0
<i>Increase</i>				
1900–1902 to 1949–1951			18.9	1.9
1949–1951 to 1999–2001			8.7	4.0

Source: Based on decennial life tables, 1900–1902 to 1999–2001, and annual life table for 2005 published by the U.S. National Center for Health Statistics or its predecessor agencies

^aOriginal Death Registration States

X Not applicable

of the reduction in death rates during the first half of the century. The greater access to health care, medical advances, and adoption of a healthier lifestyle (e.g., reduced smoking) mainly account for the reductions in death rates during the second half of the century (Fried 2000). An element in this general reduction is the greater survival of people to age 65 in a condition of better health.

As a result of the timing and types of socioeconomic and medical developments during the last century, the distribution of the gain in life expectancy over the century at age 65 was quite different from the distribution of the gain in life expectancy at birth. While life expectancy at birth increased sharply early in the century, life expectancy at age 65 improved mainly after 1950 (Table 6.3; Fig. 6.3). Life expectancy at age 65 increased by 4.1 years during the second half of the twentieth century and only 1.9 years during the first half of the century. These absolute gains seem modest, but the increase of life expectancy at age 65 in relative terms over the whole century (51 percent) was almost as great as the relative increase in life expectancy at birth (56%).

Table 6.4 also illustrates this trend with data on changes in survival ratios and age-bounded life expectancies (i.e., average years lived in an age interval). The average years lived in the interval from age 65 to age 80 increased from 11.1 years to 12.5 years (out of a total of 15 possible years), that is, by 1.4 years, in the half century from 1950 to 2000, whereas in the previous half century the increase in average years lived in this interval amounted to only 1.0 year. In the birth-to-65-year interval, the comparable shifts were in the opposite direction, from 14.3 years in the earlier period to 3.5 years in the later period.

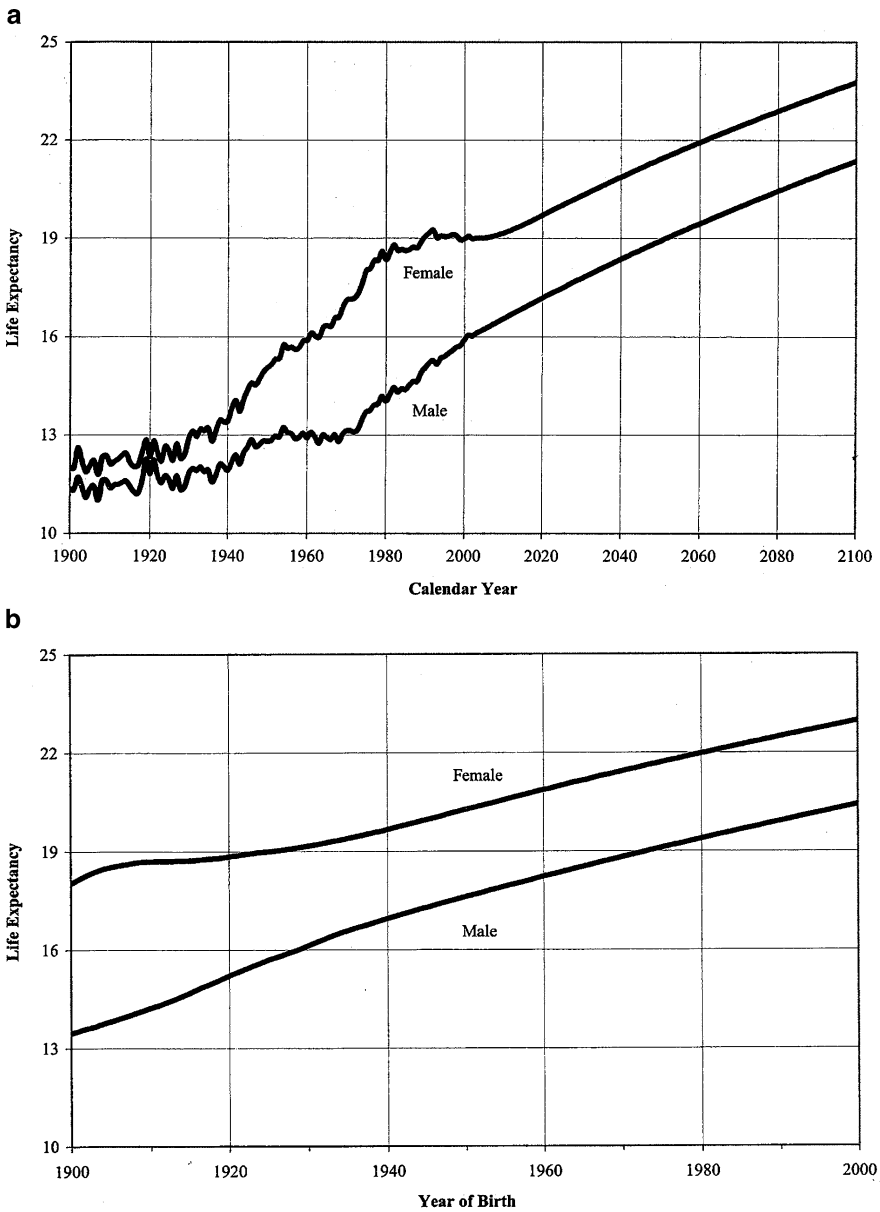


Fig. 6.3 Trend of life expectancy at age 65, by sex: U.S. Social Security Area

The reductions in death rates and the increases in survival ratios at the older ages (ages 65–80) have also increased greatly in relation to the corresponding changes for the younger age groups (ages under 65). Table 6.4 shows survival changes for the

Table 6.4 Percent surviving and age-bounded life expectancies, for the United States: 1900–1902 to 2005

	Percent surviving			Average years lived in interval			
	Birth to 65	Age	Age	Birth to 65	65–80	80–95	95+
		65–80	80–95				
1900–1902 ^a	40.9	3.1	2.5	44.4	10.1	5.2	2.2
1939–1941	60.4	37.9	3.7	55.9	10.6	5.6	2.6
1949–1951	67.6	43.4	5.2	58.7	11.1	6.2	2.5
1959–1961	71.1	47.2	4.5	59.7	11.4	6.3	2.4
1969–1971	71.9	49.1	7.9	60.0	11.5	6.9	3.1
1979–1981	77.1	56.0	11.7	61.1	12.0	7.6	3.3
1989–1991	79.5	59.2	13.3	61.6	12.3	7.0	3.3
2000	82.1	62.1	14.2	62.2	12.5	8.1	3.5
2005	83.1	65.2	17.5	62.3	12.8	8.5	3.6
Increase							
1900–1950	26.7	10.3	2.7	14.3	1.0	1.0	0.3
1950–2000	14.5	18.7	9.0	3.5	1.4	1.9	1.0

Source: Based on decennial life tables, 1900–1902 to 1989–1991, and annual life tables for 2000 and 2005 published by the U.S. National Center for Health Statistics or its predecessor agencies

^aOriginal Death Registration States

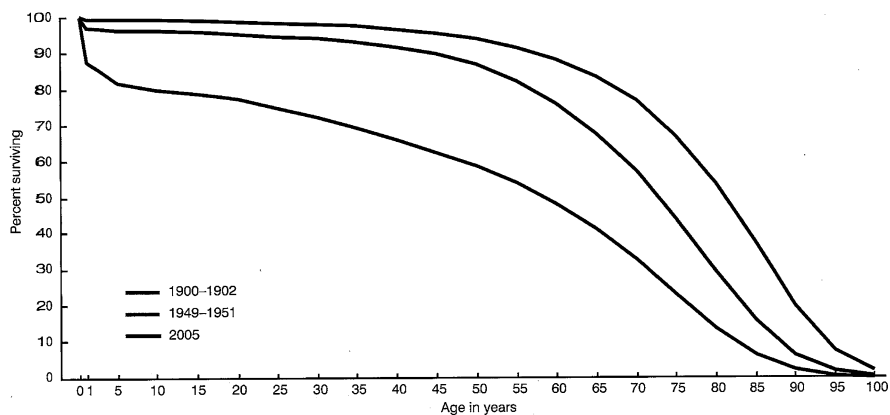


Fig. 6.4 Life table survival curves for the Original Death Registration States, 1900–1902, and the United States, 1949–1951 and 2005 (Source: U.S. National Center for Health Statistics, *National Vital Statistics*)

older and younger age groups during the periods 1900–1950 and 1950–2000. The change at the older ages during the earlier period was far smaller than at the younger ages (10 vs. 27% points), but during the later period the change at the older ages was much greater than at the younger ages (19 vs. 14% points). (See also Fig. 6.4).

Leading Causes of Death

The four leading causes of death currently in the United States, accounting for two-thirds of all deaths, as reported by NCHS, are, in rank order, heart disease, malignant neoplasms (cancer), cerebrovascular disease (stroke), and chronic lower respiratory diseases. The next six leading causes are accidents, diabetes, Alzheimer's disease, influenza and pneumonia, kidney disease, and septicemia (i.e., blood poisoning). (See Table 6.5) A century earlier the top four ranks were held by influenza and pneumonia, tuberculosis, heart disease, and diarrhea in that order, and the next six leading causes included cerebrovascular disease, kidney disease, and malignant neoplasms (Linder and Grove 1947). Two conclusions can be drawn from these lists. First, at a time when the general level of mortality was far higher than it is today, infectious diseases headed the list of causes while today the list is headed by the chronic diseases of later life. Second, both then and now several chronic diseases, particularly heart disease, stroke, kidney disease, and cancer, are in the top ten. Because most of the leading causes of death are endogenous causes now, these same causes figure among the top 10 causes at ages 65 years and over currently (2003):

Cause ^a	Rate ^b
All causes	5023.4
1. Heart diseases	1568.5
2. Malignant neoplasms	1082.7
3 Cerebrovascular diseases	384.6
4. Chronic lower respiratory diseases	303.8
5. Alzheimer's disease	174.9
6. Influenza and pneumonia	160.6
7. Diabetes mellitus	152.9
8. Nephritis, nephrotic syndrome, and nephrosis	96.1
9. Accidents (unintentional injuries)	95.6
10. Septicemia	73.6
All other causes (residual)	928.1

Source: U.S. NCHS (2007)

^aCause of death based on *International Classification of Diseases, Tenth Revision*, (1992)

^bRate per 100,000 population 65 years and over

The combination of heart disease and cerebrovascular disease, the main cardiovascular diseases, accounts for over one-third of all of the deaths currently. This is true even though there has been a tremendous decrease – about 31% – in the death rate from this group of causes between 1970 and 2004 (U.S. NCHS 2007a). The reduction in the death rates from these and some other major chronic diseases of later life in the United States in this period was spectacularly large, unanticipated, and unprecedented. Mainly for this reason, overall mortality at the older ages declined by over one-third between 1970 and 2004. On the other hand, the pace

Table 6.5 Numbers and Rates for the 15 Leading Causes of Death, by Rank, for the Total Population of the United States: 2004

Rank ^a	Cause of death ^b	Code	Number	Percent of total deaths	Death rate ^c	
					Crude	Age-adjusted ^d
X	All causes	X	2,397,615	100.0	816.5	800.8
1	Diseases of heart	I00-I09, I11, I13, I20-I51	652,486	27.2	222.2	217.0
2	Malignant neoplasms	C00-C97	553,888	23.1	188.6	185.8
3	Cerebrovascular diseases	I60-I69	150,074	6.3	51.1	50.0
4	Chronic lower respiratory diseases	J40-J47	121,987	5.1	41.5	41.1
5	Accidents (unintentional injuries)	V01-X59, Y85-Y86	112,012	4.7	38.1	37.7
6	Diabetes mellitus	E10-E14	73,138	3.1	24.9	24.5
7	Alzheimer's disease	G30	65,965	2.8	22.5	21.8
8	Influenza and pneumonia	J10-J18	59,664	2.5	20.3	19.8
9	Nephritis, nephrotic syndrome, and nephrosis	N00-N07, N17-N19, N25-N27	42,480	1.8	14.5	14.2
10	Septicemia	A40-A41	33,373	1.4	11.4	11.2
11	Intentional self-harm (suicide)	X60-X84, Y87.0	32,439	1.4	11.0	10.9
12	Chronic liver disease and cirrhosis	K70, K73-K74	27,013	1.1	9.2	9.0
13	Essential (primary) hypertension and hypertensive renal disease	I10-I12	23,076	1.0	7.9	7.7
14	Parkinson's disease	G20-G21	17,989	0.8	6.1	6.1
15	Assault (homicide)	X85-Y09, Y87.1	17,357	0.7	5.9	5.9
X	All other causes	Residual	414,674	17.3	141.2	X

Source: U.S. NCHS/Miñino et al. (2007a), Table C, p. 8

X Category not applicable

^aRank based on number of deaths^bBased on the Tenth Revision, International Classification of Diseases, 1992^cDeaths per 100,000 population^dBased on the year 2000 standard population, i.e., total population in 2000 in 10-year age groups

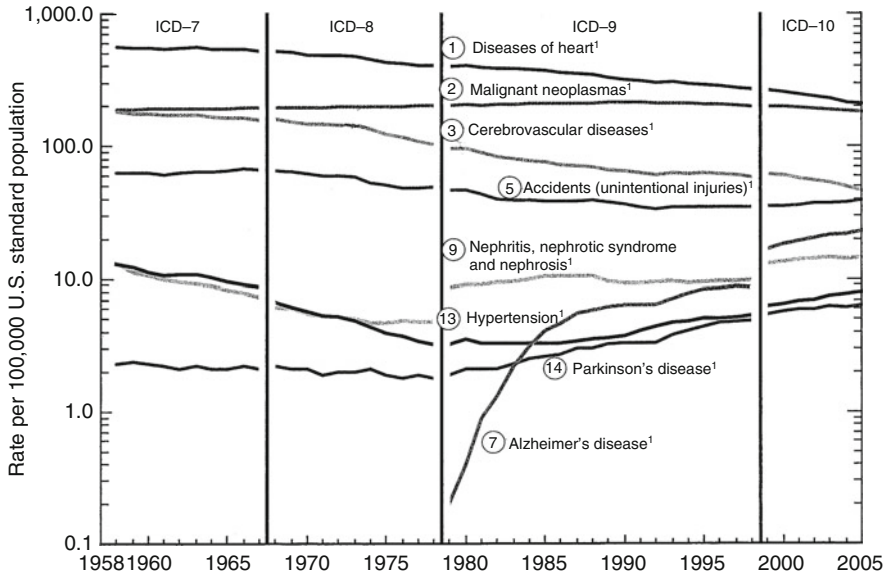


Fig. 6.5 Trend of age-adjusted death rates for selected leading causes of death, for the United States: 1958–2005 (Notes: *Circled numbers* indicate rank of the cause of death. Age-adjusted rates expressed per 100,000 standard population; Source: [U.S. National Center for Health Statistics. \(2008\)](#). Deaths: Final data for 2005. *National Vital Statistics Reports*, 56(10), 2008, Fig. 5, p. 8)

of the decline of the major cardiovascular diseases has been slowing during these decades. The rate of decline in the death rate from these causes was 15% in the 1970s, 13% in the 1980s, and 8% in the 1990s.

During the same 34-year period the death rates for several other leading diseases have been increasing or changing little. The (age-adjusted) death rate from cancer has shown zero net change in this period, first increasing in the 1970s and 1980s and then decreasing in the 1990s (Fig. 6.5). The (age-adjusted) death rates from Alzheimer's disease, chronic obstructive pulmonary disease (COPD), influenza and pneumonia, diabetes, and a few other leading chronic diseases have risen in this period. Diabetes is reaching near-epidemic proportions ([U.S. NCHS 2007a](#)).

On the basis of the anticipated aging of the population, medical developments, lifestyle changes, and other factors, we can expect a shift in the future distribution of deaths by cause in the United States. Even if current cause-specific death rates do not change, expected population changes alone would result in some reordering of the causes. For example, if a hypothetical distribution of deaths by cause was obtained as the product of the middle series of projections of population for 2050 published by the U.S. Census Bureau and recent recorded age-sex-cause-specific death rates, all causes except heart disease, the other cardiovascular diseases, and diabetes would lose ground, relative to these three causes, between 2000 and 2050 ([Sonnenschein and Brody 2005](#)). To determine the shift in the pattern of causes resulting from changes both in age structure and in cause-specific death rates, we

need to combine population projections for 2050 with projections of mortality rates by cause-categories, age, and sex. Such data can be derived from the files of the U.S. Social Security Administration, which prepares its U.S. population projections by making assumptions about future cause-specific mortality. In the industrialized countries a few causes of death now dominate the cause-pattern of mortality. Some notes on these leading causes are set forth below, drawing mainly from the lists of the 15 leading causes of death at each age in the United States published annually by the NCHS (Table 6.5; U.S. NCHS 2007b).

Heart disease. Heart disease is a leading cause of death at every age, but at the older ages this cause is consistently the front-runner. Over one-quarter of the deaths in any year is due to heart disease and one out of two to three persons will eventually die from the condition. Heart disease is often associated with and results from several other chronic diseases such as hypertension, atherosclerosis, and diabetes, but at a more basic level, factors such as stress, unhealthy diet, poor lifestyle, and adverse environments, contribute to it and to the other chronic diseases associated with it.

Cancer. Cancer, the second most common cause of death, is a leading cause at every age, but it afflicts persons in the late middle ages and early old age particularly often. At the most advanced ages the rate slacks off somewhat and is greatly exceeded by the death rate for heart disease. Nearly one-quarter of the deaths in any year is due to cancer and one out of four or five persons will eventually die of this disease. Cancer results from a variety of causes, not a single cause, but all cancers have a common characteristic, the uncontrolled proliferation of cells. Uncontrolled growth may result from a single defective or missing gene or, more commonly, from cumulative mutations that affect numerous genes and that ultimately destroy the integrity and functioning of cells.

Cerebrovascular disease (“stroke”). Stroke is the third most common cause of death. A stroke is caused by the rupture of a blood vessel in the brain as a result of an aneurysm (i.e., a bulge in an artery wall) or by the blockage of a blood vessel in the brain as a result of a blood clot. It may cause paralysis of part of the body, with loss of muscular control, aphasia (i.e., inability to speak), and memory loss. Most stroke victims are over 65 years of age. It is a leading cause of death at nearly all ages (except 1–4), even though its rate is quite low at the ages below 65 (e.g., 5.5 per 100,000 population at ages 35–44). The chance of ever dying from stroke is about 1 out of 10 and about 6% of all deaths are due to stroke.

Chronic lower respiratory diseases. Chronic lower respiratory diseases represent another group of endogenous (or intrinsic) causes of death. They have proven extremely difficult to reduce, if not eliminate. This group of causes of death is also referred to as COPD, or chronic obstructive pulmonary disease, and includes bronchitis, emphysema, asthma, and other chronic lower respiratory diseases. It appears in the list of the 10 leading causes at most ages (excluding the ages 25–44 years). High in infancy, it falls low on the list or disappears in the intermediate ages and then moves up again in later years.

Accidents. Unintentional injuries, or accidents, is one of the 10 leading causes at every age but, with advancing age, its position falls farther and farther back in the list. It still accounts for nearly 5% of all deaths at ages 65 years and over, however. The accident rate has been gradually increasing in the decade 1995–2005 after a period of substantial decrease.

Diabetes. Diabetes is now the sixth leading cause of death in the United States. Diabetes appears in the list of the 10 leading causes as early as ages 20–24, its rate rises rapidly after these ages, and it remains among the leading causes at all higher ages (U.S. NCHS 2007b). Nearly one in three people in the United States will develop diabetes in their lifetimes and one in 50 persons will die from it. Type 1 diabetes results when the body cannot produce insulin, a hormone needed to convert food into energy, and first appears usually among children. Type 2 diabetes, which covers 90% of all cases, results from insulin resistance or deficiency. Levels of diabetes are increasing in the United States and around the world. The greater prevalence of diabetes is associated with the tremendous increase in obesity in the last several decades. A family history of obesity or diabetes, that is, having parents or siblings with these conditions, is a risk factor for diabetes both in childhood and adult life. Diabetes is associated with and is cause of several other chronic conditions, including heart disease, stroke, blindness, and kidney disease.

International Trends

Variations in Life Expectancy

Wide disparities exist among the mortality levels of individual regions and countries, as noted earlier. With a figure of 78 years for life expectancy at birth in 2005, United States was far from first in rank according to this measure of health (Table 6.6). Japan, Australia, Canada, Costa Rica, Israel, and much of Northern, Western, and Southern Europe (e.g., Austria, France, Italy, Spain, Sweden, Netherlands, and Norway) are in front, leading the United States by a year or more. Japan leads the countries of the world in life expectancy with a figure of 82 years and hence surpasses the United States by 4 years. This has been achieved in part by its low levels of heart disease and cerebrovascular disease (Yanagishita and Guralnik 1988).

The five countries with the highest and lowest life expectancies at birth in 2005 are:

Highest		Lowest	
Japan	82	Botswana	35
Iceland	81	Lesotho	35
Sweden	81	Swaziland	35
Australia	80	Zambia	37
Canada	80	Angola	40

Table 6.6 Life expectancy at birth and at age 65, by sex and by rank, for selected countries: 1980 and 2002

Country	Male			Female		
	1980	2002	Rank	1980	2002	Rank
At birth						
Australia	71.0	77.4	6	78.1	82.6	7
Bulgaria	68.5	68.9	34	73.9	75.6	35
Costa Rica	71.9	76.2	12	77.0	81.0	18
Cuba	72.2	74.7	25	NA	79.2	28
Finland	69.2	74.9	23	77.6	81.5	11
Hungary	65.5	68.4	35	72.7	76.7	34
Israel	72.2	77.5	5	75.8	81.4	13
Japan	73.4	78.3	2 ^a	78.8	85.2	1
Norway	72.3	76.4	10	79.2	81.5	11
Russian Fed.	61.4	58.9	37	73.0	72.0	37
Slovakia	66.8	68.9	33	74.3	77.8	33
United States	70.0	74.5	26	78.8	79.9	26
At age 65						
Australia	13.7	17.4	4	17.9	20.8	5
Bulgaria	68.5	68.9	34	12.7	13.1	33
Costa Rica	16.1	17.8	2	18.1	20.5	8
Cuba	NA	16.8	10	NA	19.3	19
Finland	12.5	15.8	22	16.5	19.6	17
Hungary	11.6	13.1	33	14.6	17.0	32
Israel	14.4	17.3	6	15.8	19.7	12
Japan	14.6	18.0	1	17.7	23.0	1
Norway	14.3	16.2	18	18.0	19.7	12
Russian Fed.	11.6	10.9	36	15.6	15.1	37
Slovakia	12.3	13.3	32	15.4	17.0	32
United States	14.1	16.6	13	18.3	19.5	18

Source: [U.S. National Center for Health Statistics \(2006d\)](#), Table 26. For primary sources, see this publication

Note: Ranks are from highest to lowest life expectancy for the most recent year available. Since calculation of life expectancy varies among countries, comparisons among them should be made with caution. Countries with the same life expectancy receive the same rank. The country with the next lower life expectancy is assigned the rank it would have received had the higher-ranked countries not been tied

NA Not available

^aHong Kong holds rank 1

Then, among the highest, come France, Italy, Norway, Spain, and Switzerland, all with a life expectancy of 80, and among the lowest, Sierra Leone with 40, Zimbabwe, with 41, and Afghanistan, Liberia, and Mozambique with 42 ([Population Reference Bureau 2005](#)). The populations of the Newly Independent States (NIS) of the former Soviet Union in Eastern Europe and Asia (i.e., the Economies in Transition) have much lower life expectancies than the countries of Western Europe. In 2000, life expectancy in these countries averaged 68 years in comparison with 78 years in the West (Table 6.2; [World Health Organization 2002](#)).

Similarly wide variations in mortality exist in Latin America. In Latin America as a whole, life expectancy is about 72 years. Haiti has the lowest life expectancy, with 52 years, and Guatemala, Bolivia, and Guyana are at 66 years or less. Cuba, Costa Rica, Chile, Uruguay, Mexico, French Guiana, and Panama have the highest expectancies at 75 years or higher.

The region of greatest concern is sub-Saharan Africa. At its best in 1990 this region lagged considerably behind the other regions of the world (Table 6.2). The HIV/AIDS epidemic of the decades since the 1980s has pushed these countries farther behind the world average. Its life expectancy in 2005 was only 48 and some countries in the region have lost as much as 10 years of their previous progress (e.g., Kenya). In 2005 the gap in life expectancy between sub-Saharan Africa and Japan was 34 years as compared with 29 years in 1980.

In the early nineteenth century the range of life expectancy among countries was small in absolute terms (Goesling and Firebaugh 2004). Life expectancy at birth ranged from the low 20s to the low 40s. Large gains in life expectancy then occurred in areas where life expectancy was highest and, as a result, inequality in life expectancy increased. Inequality in regional life expectancy appears to have peaked between the two World Wars. At its peak, life expectancy was about twice as great in the West as in sub-Saharan Africa, South Asia, and China. Then, in a reversal, inequality sharply declined in the second half of the century, as the countries with the lowest life expectancy made the greatest gains. In recent decades, overall life expectancy in Europe has been increasing, but Eastern Europe has experienced a decline – a sharp decline in some countries. As a result, the gap among countries of the European continent widened in the 1990s after converging in the previous decade. As noted above, the drop in life expectancy in Russia and in other countries of Eastern Europe is a result of the social and economic disorder in these countries following the dissolution of the Soviet Union.

Great inequality in life expectancy among the countries of the world remains, but the variation is much smaller now than a half century ago. The divergence is reflected now in two main clusters, one consisting of the world's poorer countries converging around 45–50 years, and another consisting of the world's richer countries converging around 75–80 years (Goesling and Firebaugh 2004). As the countries of the European Union forge a common identity and establish a common economic union, we may expect to see a further convergence of the mortality levels of these countries. United Nations projections of life expectancy at birth for the major regions of the world for the first half of this century posit a considerable convergence of this measure during this period (Fig. 6.6).

Different combinations of demographic, socioeconomic, and environmental factors account for the rise in life expectancy in different countries during a particular period, even from the same initial level of mortality, and in the same country at different times. With the same apparent influences, mortality changes may vary across countries and across time at different rates. As noted, the declines in mortality that occurred in the LDC in the second half of the twentieth century were more rapid than those experienced in the MDC in the first half of the century.

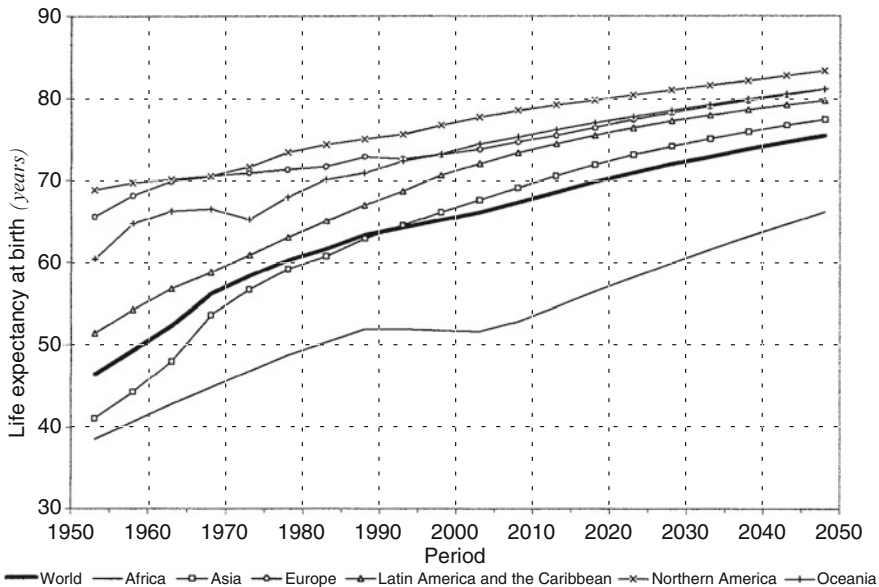


Fig. 6.6 Life expectancy at birth for the world and its major areas: 1950–2050 (Source: United Nations (2007))

The differences in mortality levels among the countries of the world are reflected in even greater variations in infant mortality. Infant mortality at 27 per 1,000 live births for Latin America is still high by the standards of the best countries, but far better than the average achieved by the less developed countries as a group (59) and by sub-Saharan Africa (94). These figures may be compared with the figure of 6.6 per 1,000 live births for the United States and 5.4 for Canada ([Population Reference Bureau 2005](#)).

Leading Causes of Death

Chronic degenerative diseases are no longer afflictions only of the affluent, developed societies, but have become global problems. Chronic diseases, especially the cardiovascular diseases, cancer, diabetes, and the chronic lower respiratory diseases, account for more than half of all deaths in the world ([Yach et al. 2005](#)). These diseases are leading causes of death in both the more developed countries (MDC) and the less developed countries (LDC). In spite of the much greater role of the infectious and parasitic diseases in the latter countries than in the former, the chronic degenerative diseases are now quite common in the LDC and their prevalence there is increasing rapidly. Yach et al. report that about three million deaths occur annually from cardiovascular diseases in both India and China.

Consumer practices and activity patterns have been changing in the LDC. Increasing use of tobacco, consumption of non-nutritious food, and pursuance of

less active lives have been elevating the risk of chronic disease, and hence the incidence of deaths from these conditions. One million tobacco-related deaths occur annually in China and 700,000 such deaths occur annually in India. As explained further below, an unhealthful lifestyle, including use of tobacco, poor diet, and lack of exercise, is a major risk factor contributing to the rise of chronic diseases, both in the LDC and MDC.

Most efforts at reduction of mortality in the world, such as the Millennium Development Goals of the United Nations, focus on selected infectious diseases and child health. However, a World Bank analysis of the most effective means to improve health in Eastern Europe and Central Asia concluded that measures to control cardiovascular diseases would contribute more to life expectancy than would measures implementing the Millennium Development Goals. According to [Yach et al. \(2005\)](#) this finding should probably be generalized to apply to many of the four billion people living in low- and middle-income countries.

Principal Causes and Trends of Morbidity

The principal causes of morbidity in the MDC are the major mental diseases (major depression, bipolar disease, and schizophrenia); the major neurological diseases of later life (Alzheimer's disease and Parkinson's disease); and the leading chronic physical diseases of later life (e.g., heart disease, cancer, cerebrovascular disease, hypertension, chronic lower respiratory disease, diabetes, arthritis, osteoporosis, and hepatitis). Neuropsychiatric (i.e., mental and neurological) disorders are the leading causes of disability from noncommunicable diseases in the world ([World Health Organization 2002, 2003](#); [Lopez et al. 2006](#)). They account for 11.5% of the total disability load or disease burden (i.e., a weighted combination of years of life lost and years of disability). The United States experience follows a similar pattern. In the section below I consider several of these diseases individually with respect to their characteristics, prevalence, and associated conditions.

The principal causes of morbidity and disability in the LDC are HIV/AIDS, malaria, and tuberculosis. I mention these conditions only in passing in this chapter and discuss them more fully in Chap. 11, which treats special issues of health in the LDC.

Mental Diseases

The major mental disorders are schizophrenia, bipolar disorder, and major depression although others, such as post-traumatic stress disorder, autism, and eating disorders, are also common and serious. An estimated 13 million American adults (approximately 1 in 17 persons) have a seriously debilitating mental illness ([Kessler et al. 2005](#)). Perhaps 20 percent of the population of the United States suffers

from some type of mental illness and two-thirds of those afflicted go untreated. According to the [World Health Organization \(2003\)](#), mental health disorders are the leading cause of disability in the United States and Canada, accounting for 30% of all disability-adjusted life years (i.e., years lost due to disability and premature mortality).

The frequency of these disorders and the lack of treatment for most victims in part explains the high suicide rates for youth and all higher ages. In the United States suicide is the eleventh leading cause of death. It is the third leading cause of death among youths 15–24 years of age, following accidents and homicides, with a death rate of 10 per 100,000. The suicide rate rises generally with increasing age. The rate for ages 85 and over is three-quarters larger than the rate for ages 15–24 years of age. Adults 65 and over account for 17% of all deaths from suicide.

The major psychiatric disorders “run in families.” The increased risk for developing the same disease among first-degree relatives varies from 3 to 10 times (autism, 150 times). Mental illnesses are generally now viewed as due to gene defects resulting in chemical imbalances in the brain. Multiple genes, where some are protective and others are pathogenic, and where some interact with other genes and some interact with the environment, appear to be involved.

Major Depression

Major depression is characterized by a feeling of dark gloom, sadness, inability to concentrate, inability to function in a disciplined way, and sleeplessness. About 10% of the U.S. population experiences depression, either chronic or episodic depression, severe enough to seek medical attention. The U.S. National Health Interview Survey reported that in 2004 14% of those 65 years old and over who had Medicare-only health-care coverage experienced feelings of sadness for all, most, or some of the time during the 30 days prior to the interview. This figure compares with 10% of those who had both Medicare and private health insurance. Among adults under 65, 17% of those without health insurance and 8% of those with private insurance reported such feelings. These figures probably greatly understate the facts, but we can say that a substantial share of the population suffers from depression and we can surmise that most of the affected individuals are going untreated (Table 6.7). Major depression ranks at the top of the neuropsychiatric disorders among the causes of disability in the world and is the first-ranked cause of disability in the Americas ([WHO and World Bank 1996, 2003](#)).

Depression can result from a familial tendency, side-effects of certain drugs or physical illnesses, changes in hormone levels, other mental disorders, and emotionally traumatic events, although there may be no apparent cause. To some extent major depression is familial, if not genetic, inasmuch as a person has three times the risk of being afflicted with the condition if a close relative has the illness. Depression increases the risks for a variety of other illnesses, including particularly heart disease, and is a principal cause of suicide.

Table 6.7 Age-adjusted percentages of persons 18 years old and over, in poor or fair health or with selected diseases, by sex and by age, for the United States: 2004

Disease	Sex			Age group				
	Total	Male	Female	18–44	45–64	65–74	75+	85+ ^a
Fair or poor health ^a	26.0 ^b	26.4 ^b	25.7 ^b		19.6 ^c	22.9	28.5 ^d	33.6
Heart disease	11.6	12.5	10.9	4.5	12.3	27.3	37.6	38.5
Coronary	6.4	8.3	4.9	1.1	6.9	18.4	26.1	
Hypertension	22.0	21.9	21.9	7.4	30.9	49.8	55.4	50.5
Stroke	2.6	2.8	2.4	0.5	2.5	6.9	12.4	
Emphysema	1.7	1.9	1.5	0.3	2.0	4.9	6.0	
Asthma ^e	6.7	5.0	8.2	6.4	7.0	7.5	6.6	
Chronic bronchitis	4.2	2.7	5.6	3.2	4.9	6.1	6.3	
Cancer, total	7.0	6.9	7.4	1.9	7.9	18.7	24.7	
Breast cancer	1.2	–	2.2	0.1	1.6	3.0	4.8	
Cervical cancer	1.0	–	1.0	0.9	1.2	0.8	0.9	
Prostate cancer	1.9	1.9	–	–	1.1	5.8	13.0	
Diabetes	7.1	7.6	6.6	2.0	10.1	18.9	16.4	11.0
Ulcers	6.9	7.1	6.8	4.5	8.2	10.9	13.3	
Kidney disease	1.7	1.5	1.9	0.9	1.8	3.4	4.9	
Liver disease	1.3	1.2	1.4	0.8	2.1	1.4	1.7	
Arthritis	21.6	18.5	24.2	8.0	28.8	46.5	55.8	
Chronic joint symptoms	26.8	25.3	28.1	15.7	35.0	44.7	49.8	
Migraines or severe headaches	15.3	9.7	20.2	18.4	15.0	7.1	5.1	
Pain in neck	14.6	12.1	17.0	12.4	18.7	13.9	15.0	
Pain in lower back	27.1	25.0	29.0	23.9	30.8	28.5	32.5	
Hearing trouble	16.4	20.1	13.2	7.7	18.5	31.7	48.9	58.0
Vision trouble	8.8	7.6	10.0	5.1	10.9	14.1	19.9	30.3
Edentulous ^f	7.9	7.7	8.1	1.8	8.5	21.3	30.7	40.2
Feelings of sadness ^g	3.3	2.7	3.9	3.1	3.8	2.5	3.5	
Feelings of hopelessness ^g	2.0	1.5	2.4	1.9	2.5	1.2	1.7	
Feelings of worthlessness ^g	1.8	1.5	2.1	1.6	2.1	1.2	2.5	
Feeling everything is an effort ^g	5.7	5.1	6.3	5.8	5.8	4.4	6.2	

Source: U.S. National Center for Health Statistics 2006a,b

Standard errors are given in the source tables

Blank spaces represent Not Available

– Not applicable or too small to be shown

^aAnnual average, 2000–2003^b65 years and over^c55–64 years of age^d75–84 years of age^eCurrently has asthma^fLacking all natural teeth^gAll or most of the time

Bipolar (Manic-Depressive) Disorder

In bipolar disease a person's mood swings unpredictably between mania (uncontrolled excitement, unrealistic euphoria, and a sense of unlimited power) and depression (uncontrollable gloom). About 2% of the U.S. adult population, or more than 5 million people, suffer from bipolar disorder in the United States. Bipolar disorder has a largely genetic basis, with several susceptible genes being involved. With bipolar disorder there is a 7–10 times increased risk of developing the disease among first-degree relatives. With fraternal twins, if one twin has bipolar disorder, there is a 20% chance the other twin has it. With identical twins, this risk rises to 80%.

Schizophrenia

Schizophrenia is characterized by hallucinations or false sensory perceptions, delusions or false belief systems, disordered thinking, difficulty in functioning in interpersonal and occupational relations, and diminished motivation and drive. Schizophrenia ranks ninth worldwide among all the causes of disability and fourth worldwide among the major neuropsychiatric causes of disability. One percent of a general population tends to have schizophrenia. For persons with a sibling having the condition, the risk goes up to 10%; and for identical twins when one twin has the disease, the probability for the other twin is 50% (Malaspina 2002). The average age of onset of schizophrenia is 18 for men and 25 for women. An inherited genetic defect is one cause of schizophrenia but so also are acquired genetic defects resulting from accumulated mutations of genes in sperm cells, as demonstrated by the higher rate of schizophrenia among children of older fathers. Other contributing factors for schizophrenia include prenatal malnutrition, obstetric complications, and infections of the mother.

Neurological Conditions

Alzheimer's disease and Parkinson's disease are the most common, serious neurodegenerative diseases. They result from the deaths of particular groups of neurons in the brain, but the causes of these changes remain unresolved.

Alzheimer's Disease

Alzheimer's disease (AD) is characterized by progressive memory loss, cognitive deterioration, and dementia. It is a disease of later life and its prevalence rises sharply with advancing age after age 65. It is estimated that about 4.5 million people now have Alzheimer's disease in the United States (Herbert et al. 2003).

Some persons younger than age 65 show cognitive impairment but by ages 65–74 the number begins to rise sharply. About 5% of the population 65–74 years of age, 17% of those 75–84 years of age, and nearly half (46%) of the population 85 years and over suffer from it. The numbers are expected to grow dramatically as the population ages, especially among persons 85 years and over. Herbert et al. have projected that some 13.2 million older Americans (their middle projection series) will have Alzheimer's disease in 2050 unless new ways are found to prevent or treat the disease. Of these, 8.0 million will be 85 years old and over.

A death rate from AD of 2.0 (per 100,000 population) is now reported at ages 55–64. The reported rate rises sharply with advancing age. AD first appears among the 10 leading causes of death at ages 75–84, where it is sixth in order. Up to the early 1980s, AD hardly appeared as a cause of death. Apparently because of the growing number of cases of Alzheimer's disease in the United States and a growing public and medical awareness of the condition, it has become an increasingly popular diagnosis as an underlying cause of death reported on death certificates. Accordingly, the reported number and rate for AD have steadily risen in the United States over the last few decades. In 2006 72,400 persons were reported as dying from AD and only a decade earlier the number was 21,400. Its true rank as a cause of death in relation to the other causes is subject to question and needs further investigation. Calculations made by [Ewbank \(2004\)](#) would place it at a much higher rank – third among all causes over all ages – than the NCHS-reported rank of seventh. On the other hand, some neurologists would question the role of AD as an underlying cause of death.

At the present time the etiology of Alzheimer's disease is unknown. There is evidence that the disease has a genetic basis.⁴ A healthy lifestyle and healthy behaviors, such as a healthy diet, regular exercise, a supportive social network, and intellectual pursuits, may lower one's risk of developing Alzheimer's disease. It has been shown too that the risk factors for heart disease and stroke (i.e., high readings on blood pressure, serum cholesterol, and homocysteine) are also risk factors for Alzheimer's disease. If the risk factors for stroke and AD are the same, common approaches are suggested for the prevention and treatment of vascular cognitive impairment and of Alzheimer's disease.

⁴Three mutated genes – amyloid precursor protein (APP), presenilin I, and presenilin 2—are known causes of inherited early-onset Alzheimer's disease. A gene for late onset AD (i.e., APOE) has been identified; it comes in three forms, conveying different degrees of vulnerability to the disease. The underlying biochemical changes associated with Alzheimer's disease are the accumulation of beta-amyloid peptide (i.e., a gummy protein that accumulates outside of nerve cells as plaque) and hyperphosphorylated tau protein (i.e., neurofibrillary tangles inside brain cells). These proteins build up in and around neurons in the neocortex and hippocampus, parts of the brain that control memory. When these neurons die, individuals lose their capacity to remember and their ability to do everyday tasks. As of now, the diagnosis of Alzheimer's disease is based on vague clinical criteria, such as simple memory tests and tests of efficiency in performing activities of daily living. A diagnosis can be confirmed only by biopsy or autopsy. Testing with brain imaging devices (MRIs, PET scans) and spinal taps are in the early stages.

Parkinson's Disease

Parkinson's disease is a neurodegenerative disease characterized by muscular tremors, impaired motor control, and muscular rigidity. It results from destruction of the neurons in brain cells that produce the neurotransmitter dopamine, but the underlying cause is unknown. These neurons die off in the basal ganglia, an area of the brain that controls body movements. As a result, the person can no longer control the movements of his or her body. It is much less common than Alzheimer's disease and appears 14th on the list of the 15 leading causes of death in the United States. Because it is concentrated among the older ages, its prevalence may be expected to grow rapidly in the next several decades.

Chronic Physical Diseases of Later Life

The chronic physical diseases of later life in combination with the neuropsychiatric illnesses described above account for a considerable share of the morbidity in the United States. According to the National Health Interview Survey, the lethal diseases among them alone account for a substantial share of the total for the chronic physical diseases. It reported that in 2004 12% of the U.S. population 18 years and over had heart disease, 22% had hypertension, and 3% have had a stroke (Table 6.7). Seven percent had some type of cancer and a similar percent had diabetes. Twelve percent had chronic lower respiratory diseases such as emphysema, asthma, and chronic bronchitis. In every case the prevalence ratio for the condition rises with advancing age. For example, for heart disease, at ages 18–44 it is 5%, but at ages 75 and over it is 38%. Over half (55%) of the population 75 years and over has hypertension, one quarter (25%) has some type of cancer, and one-sixth (16%) has diabetes. Many persons have two or more chronic conditions – so-called comorbidities – including most persons 80 years and over. (See Fig. 6.7.)

The prevalence of some of these diseases has been increasing in recent decades in the United States. Among them are diabetes, influenza and pneumonia, and chronic lower respiratory diseases. In particular, the prevalence ratio of diabetes has increased sharply in the 50 years since 1955.

The leading nonlethal chronic physical conditions are arthritis, osteoporosis, urinary incontinence, overactive bladder syndrome, and benign hyperplasia of prostate. Some notes on these conditions are given in the following paragraphs.

Osteoarthritis

Osteoarthritis is a chronic degenerative disorder in which the joints and bones erode because the cartilage separating the bones has deteriorated. It results from excessive wear or abuse, or an accident affecting the joints. Rheumatoid arthritis is a related but distinct disease. It is a chronic autoimmune disorder in which the joints erode

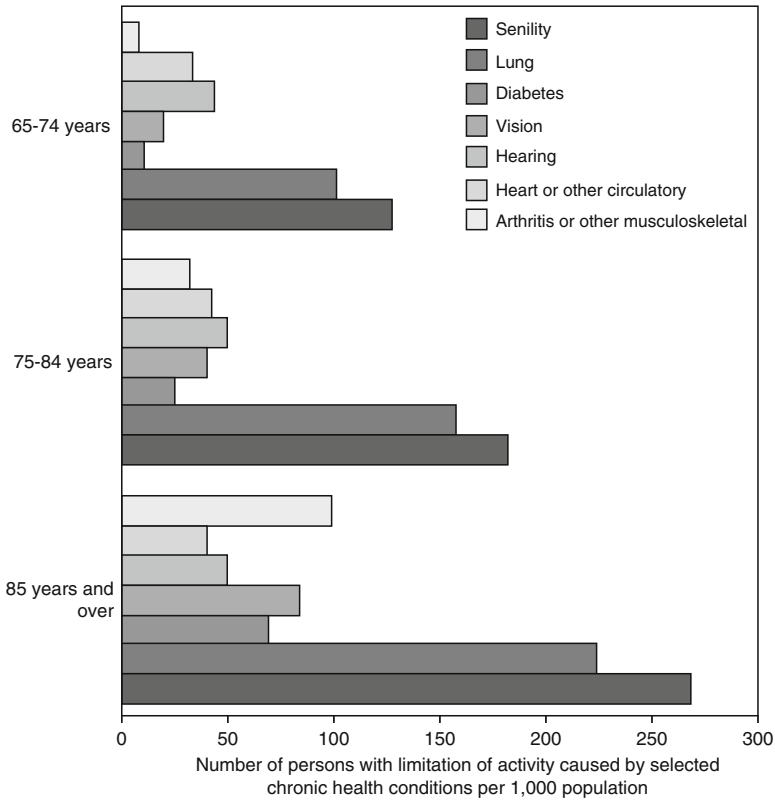


Fig. 6.7 Selected chronic health conditions causing limitation of activity among older adults by age, for the United States: 2003–2004 (Notes: Data are for the civilian noninstitutional population. Persons may report more than one health condition as the cause of their limitation; Source: NCHS (2006d). Primary source: National Health Interview Survey)

as a result of the immune system’s attacking the body. Osteoarthritis is the more common form of arthritis.

While arthritis is not a cause of death, it results in inflammation and severe long-term pain and disability. It is the leading cause of disability. About one in every five persons 18 years and over and half the persons 75 years and over have arthritis (Table 6.7; U.S. NCHS 2006a,b). An estimated 47 million Americans 18 years and over, mostly middle-aged and older, have either osteoarthritis, rheumatoid arthritis, or other diseases of muscles, bones, or joints, such as gout, lupus, or fibromyalgia.

Osteoporosis

Osteoporosis is characterized by bone thinning and bone loss. Bones become more porous and fragile as well as thinner in later life. They continue to grow in later life

but bone loss begins to exceed bone growth; as a result, there is a net loss of bone. The change leaves bones vulnerable to fractures. Hip (femur), wrist (radius), and spinal (collapsed vertebrae) fractures are frequent consequences of osteoporosis. These fractures are painful and can impair one's ability to perform the activities of daily life. Bone loss is more typical for women than men because women have less bone mass at the start and bone loss begins at an earlier age for women. For women, the principal cause of osteoporosis is a deficiency of estrogen, associated with the postmenopausal years. Apart from age-related musculoskeletal changes, poor bone health usually results from lack of exercise, particularly weight-bearing exercise, and from inadequate amounts of calcium and vitamin D.

The combined facts that older women exercise less than older men and that they are also far more likely to be affected by osteoporosis and bone fractures than men are of considerable official concern. A Surgeon General's report on *Bone Health and Osteoporosis* published in 2004 warns that by 2020 half of all Americans over age 50 will be at risk of fracturing a bone because of osteoporosis.

Detection of osteoporosis and determining one's risk of incurring a bone fracture are uncomplicated. Inasmuch as the risk depends on bone mass and density, newer imaging methods can be employed to measure it easily. Examination of the skeleton by computer automated tomography (CAT scan) and other imaging methods can detect bone thinning and loss with minimal error.

Urinary Incontinence

Urinary continence is the unintended loss of urine to a degree that is significant enough to make it difficult to maintain good hygiene and carry on one's regular public activities. Its direct consequences are quite disruptive. It interferes with such daily activities as participation in social events and is responsible for sleep deprivation, mood disorders, and increased risks of mobility impairment and falls. It is a common cause of (personal) disability and hence a common reason for the transfer of older persons from home care to nursing-home care. An estimated 25 million adults in the United States have urinary incontinence.

Overactive Bladder Syndrome (OBS) and Benign Prostatic Hyperplasia (BPH)

An estimated 17 million Americans, mostly women, suffer from overactive bladder syndrome (OAB). Most are under the age of 55. Many do not treat their ailment, and as a result, limit their activities on the basis of the available bathroom facilities. Benign prostatic hyperplasia (BPH) is a noncancerous enlargement of the prostate gland in men, resulting in frequent and urgent need to urinate, difficulty in starting urination, and a weak urine stream. It is very common in men over the age of 50. Nocturia, the urgent need to urinate during sleep at night, is a frequent consequence of BPH. In disrupting sleep, it contributes to daytime fatigue and stress.

Common Infectious Diseases

Apart from the common childhood infectious diseases, many other infectious diseases are present in the MDC. They include HIV/AIDS, hepatitis, septicemia, and tuberculosis – diseases that are extremely common in the LDC. According to [Hotez \(2010\)](#), several parasitic diseases – toxocariasis, trichomoniasis, Chagas disease, and cysticercosis – flourish in the United States; they are found predominantly among the rural and urban poor. Brief notes on HIV/AIDS and hepatitis are given here and additional material on these and other infectious diseases is given in Chap. 11.

HIV/AIDS

HIV is the disease that is the precursor of the lethal illness AIDS. Some think of HIV as merely a risk factor for disease since many persons appear to live “disease-free” with HIV for many years. About two million persons (or less than 2/3 of one percent of the population) in the United States and around 33 million persons in the world are now infected with HIV/AIDS. In the United States men are more likely to be infected than women, and non-Hispanic blacks are more likely to be HIV-positive than all other race-ethnic groups. In 2006 12,100 persons died of HIV/AIDS in the United States, considerably fewer than a decade earlier, when 31,100 died. Two-thirds of the deaths are at ages 35–54. According to the United Nations, the HIV/AIDS epidemic is responsible for the deaths of three million people in the world each year, with the African continent being particularly stricken.

The basis of acquiring AIDS has been quite different for men and women. In the United States nearly half (48%) of the men with AIDS acquired it through male homosexual contact; 26% acquired it through infected needles; 6% through heterosexual contact not related to intravenous drug use; and 10% through sexual relations with a partner and use of intravenous drugs. Of the women with AIDS, 60% acquired it through intravenous drug use and 22% from unprotected sex with an infected partner. (See U.S. [CDC 1998](#)) The ways of acquiring AIDS in the less developed areas are quite different from the ways it is acquired in the West. In the LDC most cases are acquired by heterosexual contact.

Until recent developments in treatment modalities, nearly all HIV cases resulted in death about a decade or more after infection. Now effective treatment with a battery of retroviral drugs has converted it into a chronic infectious disease that may not end in death for several additional decades after infection. Since HIV/AIDS is a sexually transmitted disease, it has affected patterns of sexual behavior, marriage, family structure, and social dependency (e.g., frequency of orphanhood).

Hepatitis

There are three types of infectious hepatitis – A, B, and C. The various types are genetically distinct and are spread by different microbes. Hepatitis A is spread by an enteric pathogen, hepatitis A virus (HAV); and hepatitis B and hepatitis C are blood-borne, sexually transmitted diseases. Hepatitis A is the most widespread form of the disease (affecting 37% of adults 20–59 in 1988–1994), but hepatitis C (HCV) is of more concern because it has more serious health consequences. Hepatitis C is a potentially lethal virus that hits young men in the prime of life. In 1988–1994, 2.0 to 3.3 million Americans 20–59 years of age were infected by this virus and most did not know it ([U.S. NCHS 2005](#)).

Hepatitis C is spread by use of reusable shared syringes, accidental needle sticks with the blood of infected patients, and receipt of blood from an infected blood bank. About 80% of HIV patients are infected with hepatitis C, 87% of hemophilia patients treated before 1987 are infected, and 10% of dialysis patients are infected. Inoculation programs among former soldiers or the practice of tattooing may have spread the virus. Untreated, it can lead to cirrhosis and death. Current safeguards with blood banks, disposal of used medical paraphernalia, and other protections have reduced the risk to a negligible level in the United States and other Western countries; in 2006 only 7,250 persons were reported as dying of viral hepatitis in the United States. IV drug abuse, however, continues to pose a problem, as does the receipt of blood before 1992. The reported numbers of deaths and the death rate from hepatitis may be artificially low in the United States currently because some of the deaths due to hepatitis may be reported as deaths from cirrhosis.

Epidemiological and Health Transitions

The Epidemiological Transition

Over the course of the last few centuries the western industrial countries have experienced vast changes in the relative contributions of fertility and mortality to their population growth. These changes are collectively termed the demographic transition. They are encapsulated in three stages. In the first stage birth and death rates are high and population growth is low; in the second stage population growth is rapid as death rates fall but fertility remains high; and in the third stage, fertility falls and once again growth is low. These changes occurred largely in the eighteenth, the nineteenth, and the first half of the twentieth centuries. Many of the less developed countries have been moving through a somewhat similar demographic transition, but the transition began more recently, there is considerable variation among these countries in their experience of the stages, and the changes do not always conform to the classic pattern.

Paralleling the primary demographic transition in the more developed countries, but with a substantial lag, a pronounced shift has occurred in the pattern of the causes of mortality and morbidity. It began in the latter part of the nineteenth century. Extending the notion of a demographic transition, Omran used the phrase “epidemiological transition” to characterize the shifts in the cause-of-death and cause-of-morbidity patterns that occurred mainly in the last century. The cause patterns shifted from a predominance of acute, infectious and parasitic diseases to a predominance of chronic, degenerative diseases of later life, and accident-related and “self-imposed” conditions (Omran, 1971, 1977; Siegel 1993; Olshansky et al. 1997). The chronic degenerative diseases of later life are similar to the endogenous (intrinsic) diseases, which, as we may recall from Chap. 3, comprise such diseases as heart disease, cancer, diabetes, cerebrovascular disease, kidney diseases, and emphysema. The shift in concern for the acute infectious diseases to the chronic degenerative diseases has been accompanied by a shift in attention from the transmission of pathogenic agents to concerns, first, about the deterioration in physiological systems of the body and, then, about healthy behavior, healthy lifestyles, and health promotion. A similar change has been occurring in many less developed countries since the third quarter of the twentieth century (Omran 1971; Gribble and Preston 1993).

In Omran’s design, the stage of the predominance of endogenous/chronic-degenerative diseases is really the third stage of the epidemiological transition. The first is the “age of pestilence and famine” during which mortality is high and variable and life expectation is under 30 years. In the second stage, termed the “age of receding pandemics,” mortality falls sharply, life expectancy rises to about 50 years, and population grows briskly. In the last stage, mortality decline has slowed, communicable diseases virtually disappear, and the degenerative diseases rise to prominence.

Health Transitions

Viewing this three-stage model today, it is readily evident that some modifications and extensions are needed. In the MDC we have recently seen a sharp decline in several chronic, degenerative diseases, the resurgence of several infectious diseases, and the emergence of many new infectious diseases. Moreover, there are variations in the application of the Omran model, especially to the LDC. Accordingly, Olshansky and Ault (1986) proposed a fourth stage of the transition, the “age of delayed degenerative diseases,” in which the ages for the onset of mortality from some of the leading degenerative diseases move upward as mortality declines. In this fourth stage of the transition rapid progress has occurred in the United States and other Western countries in the reduction of the rates for several chronic diseases of later life, especially heart disease and cerebrovascular disease. The spectacular and unanticipated declines in these rates that began in the late 1960s have led to notable declines in the overall death rates at the higher ages and an acceleration in the increases in life expectancy at these ages.

We are readily able to document the rapid progress in the reduction of the rates for various causes of death, but documentation of the incidence and prevalence of the corresponding diseases is more difficult. Various national health surveys in the United States have revealed an increase in the prevalence of severe disability in the 1970s and decreases in the more recent decades. Whether these changes in disability levels correspond to delays in the onset of the degenerative diseases or an expansion of the period of morbidity in later life is not clear. One finding that questions U.S. progress in health comes from the Health and Retirement Study. Persons born between 1948 and 1953 (about 55–59 years of age in 2008) reported poorer health than the older cohorts, those born between 1936 and 1941 and 1942 and 1947. Their overall self-assessed health was not only poorer but they reported more difficulties in mobility and physical strength, more psychiatric problems, and higher levels on biomarkers such as blood pressure and cholesterol.

A fifth stage in the epidemiological transition appears to be developing with the resurgence of some of the infectious and parasitic diseases of a prior era, the increased incidence of others, and the emergence of new infectious diseases not known previously in the western world. To facilitate understanding of these infectious agents, Exhibit 6.1 sets forth a simplified classification of pathogenic microbes.

Emergence of New Infectious Diseases, Zoonoses

West Nile virus, ebola virus, monkeypox virus, severe acute respiratory syndrome (SARS), avian flu, and HIV/AIDS figure among the newly emerging diseases. Some of these diseases represent transplants from regions in Africa and Asia, areas where infectious agents have often been transmitted to humans from animals. Diseases that are transmitted to humans from animals are called zoonoses. They use a variety of vector paths and methods to accomplish this transfer.

The methods of transmission, with illustrations of diseases (not necessarily new diseases) transmitted from animals to humans, include:

1. Direct contact: Anthrax
2. Ingestion: Giardiasis
3. Inhalation: Q fever
4. Anthropod (e.g., tick) vector: Rocky Mountain spotted fever
5. Animal (e.g., raccoon) bite: Rabies

Many diseases are transmitted in more than one way. Most cannot be transmitted from human to human, only animal to human. A very general pattern of movement of the diseases from animals to humans consists of their becoming widespread or endemic among feral animals, then their transfer to domestic animals, including those domesticated animals that live indoors and other animals that live among humans (e.g., raccoons, bats), and finally their transfer to humans (Weinberg 2006).

Living microbes:

Prokaryotes: Unicellular microorganisms containing no nuclear membrane

Bacteria: Causes cholera, typhoid fever, tuberculosis, bacterial pneumonia, Lyme's disease, syphilis

Subtypes: Rickettsiae: Causes typhus, Rocky Mountain spotted fever

Ehrlichiae: Tick-born infections causing fever, headaches, and malaise

Archaea: A type of prokaryote that usually thrives on extreme temperatures and is classified as a separate domain in taxonomic systems on the basis of similarities of DNA sequences.

Eukaryotes: Microorganisms whose cells contain a membrane-bound nucleus

Subtype: Protozoa: unicellular organisms such as amoebas.

Non-living microbes:

Prions: Single protein molecule, containing no nucleic acid or DNA or RNA; that is, it lacks genetic material needed to replicate itself. Considered to be the infectious agent in certain diseases of the nervous system, e.g., Creutzfeldt- Jakob disease.

Viruses: Noncellular submicroscopic entities consisting of a core of RNA or DNA surrounded by a protein coat. Causes smallpox, measles, influenza, viral pneumonia, viral hepatitis; dengue, ebola virus, Marburg virus, West Nile virus, monkeypox virus; herpes virus, chickenpox, herpes simplex; human papilloma virus, avian flu. Some distinctive subtypes of viruses are:

Hantavirus: Causes hantavirus infection

Corona virus: Causes SARS

Retrovirus: HIV/AIDS virus: Converts its RNA into DNA by means of the enzyme reverse transcriptase. Causes HIV/AIDS.

Echovirus: Causes gastrointestinal tract diseases

Rotavirus: Causes gastroenteritis

Arbovirus: Causes viral encephalitis and dengue

Arenavirus: Causes meningitis

Rhinovirus: Causes common cold

Exhibit 6.1 Classification of pathogenic microbes, with illustrative associated diseases^a

^aInfections caused by parasites (e.g., cryptosporidiosis, giardiasis, schistosomiasis, onchocerciasis, and malaria) and fungi (e.g., candidiasis) are not included

Another general pattern of transfer is from animals that fly, to terrestrial animals, and finally to people (e.g., SARS corona).⁵

A feature of the current, or fifth, epidemiological stage is the appearance of a new class of infectious pathogens called retroviruses. Among the retroviruses is human

⁵The distribution of zoonoses is affected by geoclimatic conditions (e.g., warming of waters), the availability of avian and aquatic hosts, the presence of anthropod vectors, migration patterns of animals and humans (including recreational and adventure travel), global trade involving the transfer of feed, food, animals (e.g., exotic pets), and inert conveyors, and the general influence of eco- and bio-systems. The risk of acquiring these diseases is increased for immune-compromised individuals who travel in areas where the disease is prevalent, persons who purchase exotic pets, and persons in areas where anthropod vectors abound. The problem of control is likely to become more difficult in the future because of people's pet fads, resistance of the newer bacteria to existing antibiotic drugs, the increase in the number of compromised persons, global warming, the invasion and clearing of previously undeveloped regions, and the increasing international migration of things, animals, and people (Weinberg 2006).

immunodeficiency virus, or HIV, the precursor to AIDS. The HIV/AIDS epidemic has resulted in a rise in the death rates in some regions, particularly sub-Saharan Africa.

Resurgence of Old Infectious Diseases and Rise in Existing Infectious Diseases

Malaria, whooping cough, typhoid, measles, and tuberculosis figure among the reemerging diseases in the industrial countries. At the same time the incidence of some existing infectious diseases, for example syphilis and influenza, has been rising. The syphilis rate rose in the current decade after it had declined to very low levels in the last quarter of the last century. This may reflect a new complacency with respect to protection against sexually transmitted diseases, and/or a phase of a disease cycle wherein immunity has diminished and the pathogenic agent has grown more powerful. The influenza rate has also been sharply rising in the last few decades.

One reason for the resurgence of bacterial infections in the industrial countries is the gradual impotence of the antibiotics that have been used to control them. Overuse of these antibiotics has encouraged the growth of microbes that are resistant to the control of antibiotics – so-called superbugs or superresistant microbes. Two such superbugs are *C. diff. (clostridium difficile)* and MRSA.⁶ Few antibiotics are available to deal with this problem. Since the development of new antibiotics by pharmaceutical firms is not viewed as cost-effective, as compared with other medicinals that are used repetitively by the same individual, the companies are not generally engaged in developing them. Hence, new types of lethal bacterial infections are appearing for which there are no effective control agents.

Implications for the Trend of Mortality and Morbidity

An important implication of the epidemiological transition and the later health transitions, especially the new developments in infectious diseases, the rise in obesity and in diabetes, and the impact of smoking, is that a monotonic downward trend in mortality can no longer be taken for granted, even in the western world. This view of steady progress has been typical of both professional and lay thinking in the last half century, but the reality has been different. First, the historical record reflects pronounced fluctuations in the rate of decline of mortality. As we may recall, life expectancy in the United States remained virtually unchanged between 1954 and 1968, life expectancy among blacks in the United States moved downward in various years during the 1980s, and the rates for several chronic diseases of later life moved upward in the last few decades even while others moved sharply and unexpectedly downward.

⁶MRSA is the abbreviation for Methicillin-Resistant Staphylococcus Aureus, a strain of staphylococcus resistant to nearly all antibiotics and common in hospitals.

Not only have there been a number of major epidemics but they are continuing to occur. The influenza epidemic of 1918–1919 was responsible for the deaths of 40 million people around the world (Golini 2002) and about half a million people in the United States. Lesser influenza epidemics have occurred in the world and the United States since then. Tuberculosis, once considered under control, now kills 1.7 million persons in the world and several hundred persons in the United States each year.

A review of these developments has led to considerable questioning of the original epidemiological-transition model and to the proposal to rename the entire process the “health transition process.” This term would suggest not a further stage in the epidemiological transition but the variety of changes in the pattern of causes of death and sickness that are occurring. For example, the term health transition has been used to refer to the “social, behavioral, cultural, and ideational changes that accompany mortality decline and improved health” (Caldwell and Santow 1989). More specifically, it has been used to refer to the recent interest of demographers in the inequalities in health levels, and the social and economic factors underlying these inequalities, especially how such factors as race and ethnicity, education, income, and marital status are related to health outcomes. The term, health outcomes, is defined broadly in this context to include death and disease, mental health as well as physical health, and wellness as well as sickness.

Summary Note on Developments in Mortality and Morbidity

It is apparent that the nature of mortality and morbidity is vastly different among today’s population than among previous populations, among the old than among the young, and among the more advanced industrial populations than among the less developed countries. In the advanced industrial societies the epidemiological transition has replaced the major infectious diseases, usually acute illnesses of childhood and youth, with the chronic diseases of later life as the principal causes of sickness and death. As a result, mortality in the western countries is almost entirely due to chronic, degenerative diseases. This shift in causal patterns of sickness and death are beginning to characterize the less developed countries as well.

The old are likely to have multiple concurrent chronic diseases, or comorbidities, and with very advanced age, one or more functional limitations, or codysfunctionalities. Comorbidities lead to impaired functionality, decrease in biological homeostasis (i.e., the ability of an organism to maintain equilibrium of its physiological systems), and ultimately to death. Unlike the infectious diseases causing death at younger ages, the multiple morbidities of later life do not run their courses independently of one another. Rather, they are likely to have an interdependent and cause-and-effect relation with one another that intensifies the risks of dysfunctionality and mortality of the affected individuals.

Achieving acceptable levels of health both in the MDC and LDC appears to depend more on successful functioning and stability of the public health and sanitary

systems and the social and economic order than on new medical developments, just as did the shift from the first to the second stage of the demographic and epidemiological transitions. Yet, technological developments and medical advances are a major characteristic of the health systems of the MDC and are expected to grow in importance.

Factors Associated with Mortality and Morbidity

General Overview: Biological, Social, Individual, and Stochastic factors

Many statements have been put forward as to the way the risk factors accounting for diseases and deaths should be apportioned between genetic and other factors. For example, the [National Research Council's, Institute of Medicine \(2000\)](#) concluded, on the basis of available studies, that half of all deaths and the majority of diseases and disabilities in the United States are linked to behavioral and social factors ([Goldman 2002](#)). The National Institute on Aging of the National Institutes of Health announced that, while the relative roles of genetic and environmental factors in exceptional longevity are not known, life span studies indicate that genetic factors account for about 30% and environmental (i.e., nongenetic) factors for about 70% of the variation in longevity ([Hodes 2005](#)). [Hjelmsberg et al. \(2006\)](#) have reported that human family studies suggest that a similarly modest amount of the overall variation in adult life span (approximately 20–30%) can be accounted for by genetic factors.

A genetic component in human longevity can be immediately inferred from the widely different life spans of the different animal species, but the complexity of the issue becomes evident from the considerable variability in longevity within each species, especially humans. This variation implies that nongenetic (i.e., environmental and lifestyle) factors can be quite important as well. The roles of genetic, social (i.e., environmental), individual (i.e., behavioral and lifestyle), and stochastic (i.e., chance) factors in the causation of disease and mortality are complementary and interactive. These factors do not usually act independently but in innumerable complex combinations and variations, with different degrees of influence under different circumstances. Their relative impact differs for different parts of the age cycle, different groups in the population (e.g., sex, race, ethnic, marital, and socioeconomic groups), and different life experiences. Early life experiences affect health and mortality in later life as well as later-life experiences, greatly augmenting the role of environmental and behavioral factors in later life. The genetic influence on human life span over the generations varies with the genetic characteristics of the parents and their antecedents and the tendency of individuals to marry within or outside their racial/ethnic/socioeconomic group (i.e., homogamy/exogamy).

Biological Factors

Age is the leading biological risk factor for disease and death. For this reason the role of age in disease and death is considered often in this and other chapters. Among adults, with advancing age the risks of disease and death rise steadily. Like other ascribed characteristics, age (equivalently, date of birth) is a personal characteristic that does not change or that changes in a completely predetermined way, although the health consequences of age changes are usually modifiable to a substantial extent and they vary greatly from individual to individual. Age has important social dimensions in its relation to health and mortality as well, as when persons of different ages have different degrees of access to the health-care system or when persons at the older ages are treated differently by the health-care system than younger persons. Sex and race, like age, are also ascribed characteristics that have important biological and social dimensions. For the present work, I have grouped sex and race with such social characteristics as marital status and socioeconomic status, for the discussion of group variations in the next chapter.

Various types of biological influences in addition to the compositional factors of age, sex, and race affect morbidity and mortality. First is the influence of biological evolution (the “species” influence). Evolution has imposed some broad protections and restrictions on the processes of development of all humans that affect their health and longevity. At the same time, each family passes along a particular set of genetic characteristics (“family inheritance”) that differentiate the genetic potential of its progeny from that of other families. Some of these familial genetic influences are essentially determinative in their consequences while others have a weaker potential whose expression in the progeny depends on the chance reactions of internal metabolic processes, environmental experiences, and life-style. In practice, it is difficult to distinguish the various types of biological influences in health outcomes but it is useful and important to try to do so.

Evolutionary (or “Species”) Influences

Some evolutionary pressures, such as the increasingly limited role of natural selection after the peak of reproduction (i.e., late teen ages and early twenties), operate throughout the animal kingdom.⁷ The probability of the occurrence of many adverse somatic conditions rises steadily with advancing age, especially after the reproductive period, because evolution does not work as consistently to protect against these risks as it does earlier in life. One could say that these risks are, in effect, “hard-wired” by evolution into the genes of humans. [Carnes et al. \(1996\)](#) and [Carnes and Olshansky \(1993\)](#) have described the stark difference in the course of the mortality rate for females, the effectiveness of natural selection, and the course

⁷This statement has some rare exceptions. They are described as cases of negative senescence.

of cumulative reproductive success. They note that evolution “designed” various genetic mechanisms to increase the likelihood of survival to reproductive age even under conditions of duress, and to maintain health and vigor long enough for the individual to reproduce. Evolution has not, however, designed any genetic program for aging or death in humans. “Aging is not a genetically controlled mechanism. It is an inadvertent byproduct of bodies surviving beyond their warranty period, i.e., the time required for reproduction, nurturing, and grandparenting” (Carnes 2005). Our genes do affect our longevity, however, working singly or in combination, to control (i.e., support or suppress) various metabolic and hormonal processes directly influencing our state of health.

Family Inheritance and Genetic Influences

Some health conditions are purely genetic in origin, being passed on to offspring through the specific genetic characteristics of the mother or father. Genetic diseases of this kind are likely to manifest themselves early in life although there are important exceptions. The stronger the role of genes in a disease, the earlier in life will its effects lead to disability and death. The risk of incurring some diseases and dying from them rises to about age 50 and then declines with increasing age. Cystic fibrosis, genetic dyslipidemic hypertension, and genetically determined cancers of the breast and lung are manifest by age 40 (Manton and Stallard 1994). Two of the genes that carry the mutation for breast cancer, called BRCA1 and BRCA2, account for early-onset cancer and are responsible for 5–10% of breast cancer cases. These genes are also implicated in hereditary ovarian cancer and prostate cancer. Genetically determined breast cancer is thought to be inherited as a dominant trait; that is, only one parent has to transmit it.⁸ Sometimes, genes are inherited as recessive traits, that is, an affected individual has inherited one mutated (or non-functioning) gene for the disorder from each parent.⁹ Cystic fibrosis and Gaucher’s disease type I are recessive traits. There are many illustrations of familial genetic conditions that are passed on to progeny. Some examples of inherited genetic diseases are given in Exhibit 6.2.

Genetic disorders inherited from fathers. Cumulative genetic mutations of male germ cells account for a number of inherited genetic disorders. Male germ cells continually divide and errors accumulate during spermatogenesis as men age. These mutations are then passed on to their children. As a result, older fathers are far more

⁸A woman can inherit a BRCA mutation from her mother or father but she also has a 50% chance of not inheriting it at all.

⁹A person who has a mutation in only one gene of the pair of genes for a given condition inherited from the parents is a carrier. Carriers are healthy persons who do not develop the disorder but are at risk for passing on the mutated gene to their children. If both parents are carriers of the gene for the same condition, there is a 25% chance that their child will be affected with the condition, a 50% chance that the child will be a carrier, and a 25% chance that the child will be neither affected nor a carrier.

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1. Phenylketonuria (PKU) is a rare genetic disease that causes mental retardation, deafness, and seizures. Immediate initiation of dietary therapy virtually eliminates PKU as a cause of mental retardation; continuing dietary therapy before and during pregnancy can prevent the problems associated with maternal PKU, allowing normal development of infants of affected mothers.
 2. Non-classical congenital adrenal hyperplasia (CAH) is the most common single-gene disorder in the population. It causes a broad spectrum of disorders. There is a prenatal treatment that prevents the manifestations of CAH.
 3. Congenital hypothyroidism is a metabolic condition that results from the loss of thyroid function and, if left untreated, causes irreversible mental retardation. Where newborns are screened for this condition, it can be eliminated as a cause of mental retardation.
 4. Fragile X syndrome is the leading cause of inherited mental retardation. Its cause is a mutation in the Fragile X Mental Retardation 1 (FMR) gene found on the X chromosome. The mutation is an inheritance mechanism called a "Atriplet repeat," which gets larger in each generation and, when large enough, disrupts gene function.
 5. Cystinosis is a rare genetic error of metabolism that can cause kidney failure and neurological damage. As a result of an error in the gene for Rett syndrome, infant girls gradually lose their language capabilities, mental functioning, and ability to interact with others. Taking the drug cysteamine bitartrate regularly completely prevents any manifestation of the condition.
 6. Tay-Sachs disease is a fatal hereditary disease in which an enzyme deficiency leads to the accumulation of gangliosides in nervous tissue. It affects young children of eastern European Jewish descent almost exclusively.
 7. Sickle-cell anemia is a hereditary form of chronic anemia caused by a mutant hemoglobin gene that makes the red blood cells become crescent in shape. The disease is characterized by fever, leg ulcers, severe joint pain, and jaundice, and affects black persons almost exclusively.
 8. Hemophilia A is a bleeding disorder in which there is a deficiency of clotting factor VIII. The gene abnormality is sex-linked, that is, inherited through the mother and expressed in the male.
 9. Polycystic kidney disease is a disorder in which many cysts form in both kidneys; as a result, the kidneys enlarge, some kidney tissue is destroyed, and functioning kidney tissue is lost.
 10. Cystic fibrosis is a hereditary disease characterized by impaired respiratory and pancreatic function as a result of the production of abnormal secretions from many exocrine glands (*i.e.*, glands that secrete fluids into a duct). It is a recessive genetic disorder that disproportionately affects white persons as compared with other races.
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Exhibit 6.2 Examples of inherited genetic diseases

likely than younger fathers to have children with inherited genetic disorders. Some of the disorders resulting from inherited genetic mutations associated with paternal age are as follows (*Narsad Research Newsletter* 2002):

Achondroplastic dwarfism: Improper development of cartilage at the ends of the long bones, resulting in a form of congenital dwarfism.

Marfan syndrome: A disorder principally affecting the connective tissue, manifested by excessive bone elongation and joint flexibility, and resulting in weakened tissues and abnormalities of the heart, blood vessels, lungs, bones, joints, and eyes.

Neurofibromatosis: Disease characterized by the formation of neurofibromas (*i.e.*, soft fleshy benign growths of nerve tissue), sometimes accompanied by physical deformity (*e.g.*, curved spine, rib deformities).

Osteogenesis imperfecta: Defective formation and development of bony tissue.

Advanced age of the father is an important risk factor for schizophrenia in children. [Malaspina \(2002\)](#) analyzed birth records that showed a strong escalation in the risk of schizophrenia in the child as the age of the father increased. Records of 92,000 pregnant women in Jerusalem were studied, including a linkage to the records of the psychiatric case registry. She found that the risk for children of fathers in their forties was double that for children of fathers under 25 years old, and that the risk was triple for children of fathers over 50. One of every 50 children of fathers over 50 suffered from the condition. Father's age alone accounted for one-fourth of all cases of schizophrenia in the population studied. Several other studies have yielded similar results.

Randomly Acquired Genetic Defects

Many more diseases result from randomly acquired genetic defects. During the long period from fertilization to old age, genetic damage to DNA and somatic cells occur and accumulate as a result of hostile internal and environmental influences and copying errors in the subdivision of the cells. These may manifest themselves as one of the chronic diseases of older age such as colon cancer or heart disease. Further, one's chances of developing such diseases double if one's parents or siblings are affected by these diseases. The risk is even higher for people with two affected family members.

As suggested earlier, most purely genetic defects are expressed by the time of the menopause and are lethal. By the older ages, the genetic conditions determining most health outcomes have been greatly modified by a "lifetime" of environmental and lifestyle influences as well as by internal metabolic, hormonal, and stochastic processes. Hence, it is reasonable to accept the conclusions of the National Institute of Health and others cited earlier that only a modest amount of the overall variation of the adult life span is accounted for by genetic factors.

Genetic Influences at the Very Oldest Ages

Genetics appear to play a different role in health risks at the very oldest ages than at the adult post-reproductive ages, however. Genetic factors may become increasingly important for survival at these ages. On the basis of studies of extinct cohorts of Danish, Swedish, and Finnish twins (20,502 individuals), [Hjelmborg et al. \(2006\)](#) found that having a twin survive to old age substantially and significantly "increased" the chances of reaching the same old age and that this chance is higher for monozygotic (MZ) twins than for dizygotic (DZ) twins. Specifically, the relative risk of reaching age 92 is 4.8 for MZ males and 1.8 for DZ males. Hjelmborg et al. concluded that genetic influences on life span are minimal prior to age 60 but increase thereafter, and interpreted this finding as support for the search for genes affecting longevity in humans, especially at advanced ages. (Further research on this issue is discussed in Chap.13.)

Biological Aging

Biogerontologists differentiate superficial bodily changes that occur with age, such as graying and thinning of the hair and wrinkling of the skin, from those changes that increase the risk of disease, disability, or death. They use the term biological aging to describe all these changes, especially the second group of changes. When such age-related vulnerabilities manifest themselves as disease or disability, biogerontologists call the process senescence. Alternatively, senescence may be defined as the progressive deterioration of bodily functions over time. This loss of function is associated with decreased fecundity (i.e., physiological ability to parent a child) and increased risk of morbidity and mortality as an individual gets older. The rate and progression of the process of somatic deterioration varies greatly from person to person and from bodily organ to bodily organ for any given person, but generally over time every organ of the body is affected. Since individuals differ greatly in the rate and progression of these deleterious processes, a group of persons of the same chronological age may differ greatly in their biological age.

Biogerontologists disagree as to when senescence begins. Some maintain that it begins at birth, others that it begins after the peak reproductive years, and still others that it begins at various other ages before age 45. In any case, the process leads to, or represents, a loss of bodily functions, increasing the probability of death and ending ultimately in death itself.

Aging as “cause” of disease. Of all the factors one can identify as possible contenders for the role of leading “cause” of the chronic diseases of later life, aging comes first. One cannot reflect on these diseases independent of the role of aging. Most biogerontologists maintain, however, that aging is not a disease even though aging is associated with many bodily changes that are considered diseases. They draw a distinction between normal aging and pathological aging, but blur the distinction by identifying numerous age-related conditions that compromise health as part of normal aging. Most “normal” (that is, usual or typical) age-related changes shift gradually with advancing age to pathological levels – that is, levels that are immediate threats to, or limit, functioning.

There is no easy basis for setting a dividing line between normal and pathological age-related somatic changes, even if measures of physiological performance or biomarkers are taken into account. [Ling et al. \(2007\)](#) conclude that state-of-the-art imaging and immunologic and biochemical methods are needed to analyze the process by which a chronic disease (e.g., osteoarthritis) develops and progresses as an individual ages, and to understand how complex the determination of the line between aging and disease is. While an important heuristic and philosophical distinction can be made between normal and pathological aging, it may not be productive to debate when, and even whether, certain gradual degenerative changes should be called normal aging or pathological aging. I discuss this topic in more detail in Chap. 13.

The principal physical changes that gradually occur in humans with age over an adult lifetime have been the object of study by the Baltimore Longitudinal Study

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1. Thoracic area: Lung tissue loses much of its elasticity; muscles of the rib cage shrink. Maximum breathing (vital) capacity diminishes progressively beginning about age 20 and may decline by about two-fifths between the ages of 20 and 70.
 2. Heart and vascular system: Blood vessels accumulate fatty deposits and lose much of their flexibility. With the resulting atherosclerosis, the heart needs to supply more force to propel the blood forward through the less elastic arteries. The heart muscle thickens; maximum oxygen consumption declines by about 10% for men and by about 7.5% for women with each decade in adult life. The heart's maximum pumping rate and the body's ability to extract oxygen from the blood decline.
 3. Gastrointestinal system: Production of digestive enzymes diminishes and tissues lose much of their ability to catabolize and absorb foods properly.
 4. Reproductive system: In women, production of vaginal fluid decreases and sexual tissues atrophy. In men, sperm production decreases and the prostate gland enlarges. Libido in both sexes declines gradually.
 5. Urinary system: Kidneys gradually become less efficient at extracting wastes from the blood; bladder capacity declines. Individuals are at greater risk of urinary incontinence.
 6. Musculoskeletal system: Without adequate exercise muscle mass declines over one-fifth between the ages of 30 and 70. Bone mineral is lost and replaced simultaneously until the loss begins to outstrip replacement around age 35; the loss is especially rapid for women after menopause (unless offset by regular weight-bearing exercise).
 7. Neurological system:

Brain: The brain loses some of the structures (axons) that connect nerve cells (neurons) to each other, and the ability of neurons to function may diminish. The flow of blood and the supply of oxygen diminish. Some types of memory, the ability to shift between tasks, reaction speed, and attention deteriorate. Other types of memory (*e.g.*, expert knowledge and access to cognitive "templates"), automatic functions, and the ability to focus on a task and resist distractions are improved in older age, and the brain's control of the senses is maintained rather well. Senses of vision and hearing: Various aspects of vision begin to decline at later ages: Focusing up close (in the forties) and distinguishing fine details (in the seventies); adjustment for glare, seeing at low levels of illumination, and detecting moving targets (in the fifties). Hearing acuity declines with age: Ability to hear higher frequencies is reduced; and understanding speech is more difficult, especially in situations where there is background noise.

Other senses: Acuity of the other senses – smell, taste, touch, heat, pain, kinesthesia, proprioception – similarly declines in older age. A reduced "sense" of balance results from the aging of the vestibular system (inner ear including the nerve receptors in the semicircular canals). This sense also depends on the proper functioning of the eyes, ears, and the central nervous system.
 8. Metabolic changes: Absorption of food is reduced, weight falls, and the individual tends to lose total body fat. However, fat is redistributed in the body to the hip-waist area and from just beneath the skin of the abdomen to deeper parts of the body. For women the shift is to the thighs and hips; for men the shift is to the abdomen.
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Exhibit 6.3 Principal physical changes with advancing age

of Aging. They are enumerated in Exhibit 6.3. As noted there, every physiological system experiences a major loss of efficiency from youth to old age. Underlying these pervasive system changes is the fact that, on average, by age 75 or 80 the number of cells in the human body has decreased by 30% or more.

Environmental and Ecological Factors

There are many ways to set forth the nongenetic, or social and individual factors, affecting the risk of disease and death. Any attempt to do this on the basis of an assessment of their relative importance, except perhaps in particular cases of disease or death, is fraught with grave difficulties. They work collectively to influence health outcomes. Many are related to one another, some in a temporal or causal sequence. Some have a sort of imperial power, like smoking, obesity, or high blood pressure, to effect negative outcomes. Presumably these conditions could be put at the head of the list. Yet, for making positive gains in longevity, regular exercise comes up shining. Many of us were brought up to observe five valid health rules, prescribed in no particular order: Do not smoke, check your blood pressure, eat properly, get sufficient exercise, and choose your parents carefully. I do not attempt any ranking of the nongenetic risk factors here, only a simple enumeration of them, with brief notes regarding their role in disease in the United States and elsewhere today.

One group of influences affecting health, the environmental factors, are outside the control of the individual except as individuals are part of a group, act collectively, or experience them in common. They include community health programs, air and noise pollution, workplace conditions, food and water quality, exposure to infectious agents and radiation, and availability and quality of health services. I consider these environmental factors in more detail next.

Community Health Programs

Public health risks can be greatly influenced by the efforts of private and public agencies to inform and guide the public in its health choices and practices. Community agencies can sponsor public health information programs that promote and support responsible health choices and that are designed to reduce the burden of preventable disease. Such programs can motivate people, especially young people and older Americans, to make healthful choices regarding diet, nutrition, and exercise; and can help remove the stigma from reporting and treating certain health conditions, especially mental illness and sexually transmitted diseases. Community organizations can set up programs to train individuals to examine themselves for high blood-pressure, breast cancer, sexually transmitted diseases, and skin cancer. They can set up community clinics to carry out these examinations and can sponsor programs of training in prenatal care.

Air and Noise Pollution, Workplace Conditions, and Radiation Exposure

Air pollutants, such as sulphur dioxide, nitrogen dioxide, and mercury, emitted mostly by power plants and automobiles but by other sources as well, contribute to the formation of smog. These emissions react with sunlight to form ground-level

ozone, also part of smog. A correlation has been found between ground-level ozone and death rates in cities. More than 150 million Americans live in areas with officially excessive levels of ozone.

The workplace may expose workers to excessive noise; air pollution, including dust, smoke, chemical fumes and gases, and radiation; hazardous work conditions, with excessive risk of injury; and other health risks, such as occupational stress. The work environment may put the worker's health directly at risk for respiratory and lung diseases, such as asthma, chronic bronchitis, and lung cancer. Work in occupations that involve repetitive, boring tasks, low rewards with low prestige, and minimal control over one's work life is associated with higher rates of musculoskeletal conditions, cardiovascular diseases, and emotional disorders. Persons in manual occupations report poorer health than persons in white-collar work, even allowing for education and income ([Case and Deaton 2003](#)).

Individuals are constantly exposed to various degrees of radiation – in the home, public places, the workplace, and the health center (from medical diagnostic and therapeutic equipment). The radiation absorbed by the body is negligible in most cases (e.g., dental x-ray), but in other cases it is substantial (e.g., chest x-ray, whole body CT scan, and overseas air flight). It is cumulative over an individual's lifetime, and workers who are constantly or frequently exposed to radiation and who fail to take adequate protective measures live with an excessive risk of incurring various types of cancer.

Food and Water Quality, and Exposure to Infectious Diseases

Impure water supplies and sanitation not only are responsible for illness and death among children, but they also stunt the growth of those who survive. Pesticides used in food cultivation may be absorbed by the growing crops, and then have toxic effects on the animals and humans who eat them. The infectious diseases that ravished childhood early in the last century have been largely eradicated, but some old ones and other new ones, such as HIV/AIDS, are creating increased risks for the population. Hospital stays contribute their own health risks because of nosocomial (i.e., hospital-caused) infections as well as common systems errors, including particularly errors in drug treatment and slow response to in-house heart attacks.

Housing Quality

It is well known that the home can be the source of many types of health risks, especially as a common milieu of accidents. These risks may emanate from the physical structure itself, the characteristics of the occupants in relation to the structure, or the surrounding environment. The physical structure may be a toxic environment for the occupants if it contains lead pipes, asbestos in the building materials, or radon in its subterranean areas. The U.S. Annual Housing Survey inquires about signs of rats

or mice, holes in floors, open cracks or holes, exposed wiring, and water leakage. Homes occupied by elderly persons may not be physically adapted for optimum safety to the limitations of its occupants. The lighting may be inadequate, protective safety devices, such as grab bars, may be missing, or the stairs may impose a special risk of falls. The environment in which the house is located may include special health risks such as excessive street or other noise, neighborhood crime and inadequate police protection, and trash, litter, and junk on the streets.

A summary measure useful for relating housing conditions to health is the crowding index. The crowding index is the number of residents in an occupied housing unit divided by the number of rooms in the housing unit. An index of 1.01 persons or more per room is usually considered an indication of crowded housing conditions. The observations may be grouped as less than 0.5 person per room, 0.5–0.99 person per room, 1.0–1.5 persons per room, and 1.51 persons or more per room. The use of this measure is illustrated in a report based on the National Health and Nutrition Examination Survey showing a gradation in the crowding index for persons having one or more of several infectious diseases, including hepatitis A, B, and C, and herpes simplex virus (U.S. NCHS/Ogden et al. 2005).

Quality of Care, Access to Care, and Utilization of Care

Individuals' health risks differ because of differences in their opportunity to access health care and in the quality of their health care. The risks arising from the variations in the quality of health care vary depending on the appropriateness, safety, timeliness, and equity of the health care (U.S. AHRQ 2005). Equitable care is care that does not vary in quality because of the personal characteristics of the patient, such as age, gender, race/ethnicity, and socioeconomic status. To minimize health risks, an individual should be able to gain access to the health care system easily, as suggested by the following indicators: Availability of health insurance and of an ongoing source of care; convenient transportation; no difficulties in scheduling appointments; little problem in getting referrals to specialists, in securing necessary health screening tests, childhood immunizations, and flu vaccinations; and ready access to prenatal care.

For some, the lack of communication and transportation facilities complicates the problem of securing health care when needed. Many persons lack telephone or public transportation services. About 5% of the households in the United States lack public transportation and the same percentage lack telephone service.

The United States is unique among the industrialized countries in lacking a system of universal health insurance. As a result, a massive number of persons do not have health insurance (about 47 million in 2007), and many more millions who have health insurance have coverage that is severely limited or is tenuous because of the conditions of their employment. Moreover, the U.S. health care "system" fails to offer meaningful mental health insurance, dental insurance, and eye-care and hearing-care insurance. Many persons cannot afford to take advantage of the available health services because of the high costs of care. Physicians, particularly

specialists, are scarce in many areas and some refuse to participate in government-sponsored health insurance programs, such as Medicaid and Medicare, because of the inadequate remuneration for their services. The problem of access to health care is discussed in more detail in Chap. 15.

Lifestyle and Behavior

A variety of lifestyle practices and behaviors are associated with specific diseases and are considered to be risk factors for these diseases. The list of behavioral risk factors includes some conditions that could be considered symptoms of disease or diseases, and not merely risk factors. Thus, a clear dividing line between risk factors for disease, symptoms of disease, and disease cannot always be drawn. Consider a few examples. Obesity is a risk factor for several serious diseases, but it is sometimes considered a disease in itself, especially in the extreme form labeled morbid obesity. Hypertension is always considered a disease, even if it is asymptomatic, but it is also considered a risk factor for disease because of its close linkage with, and role as precursor to, other cardiovascular diseases. Persistent pain is a symptom of many diseases. Often, as in the case of arthritis, pain is a symptom of the disease and it is treated as a symptom. Pain may be viewed as a disease in itself, however, if it is unexplained pain or there is no easy modality for treating it.

Accordingly, in this section I commingle lifestyle practices and behaviors that are risk factors for disease with some health conditions that are symptoms of disease and some that are diseases in their own right. Among the lifestyle practices and behaviors examined for their potential health impact are tobacco use, alcohol consumption, sleep, exercise/inactivity, nutrition and diet, immunization history, and social support and stress. Among the symptoms or indicators of disease considered are overweight/obesity, hypertension, high serum cholesterol levels, C-reactive protein and homocysteine levels, premature birth and low birth weight, and sexually transmitted diseases. The reader will recognize some of these items as biomarkers discussed in Chap. 5.

The data collection systems that provide much of the basic data on behavioral risk factors discussed below are the Behavioral Risk Factor Surveillance System (BRFSS), sponsored by the Centers for Disease Control and Prevention (CDC), and the National Health Interview Survey (NHIS) and the National Health and Nutrition Examination Survey (NHANES), sponsored by the National Center for Health Statistics (NCHS). Data on five important health-related behaviors (alcohol use, cigarette smoking, leisure-time physical activity, body-weight status, and sleep), based on the National Health Interview Survey, are given in the NCHS reports, *Health Behavior of Adults: United States, 2002–2004* and *Health, United States, 2006*. Separate reports on some of these risk factors have also been issued. Inasmuch as health behaviors are self-reported in the National Health Interview Survey, it is reasonable to expect underreporting of the unhealthy behaviors.

Tobacco Use

Smoking among U.S. adults 18 years and over fell sharply in the last third of the last century, from 42% in 1965 to 21% in 2004 (U.S. NCHS/Adams and Schoenborn 2006c; NCHS 2006d). On the other hand, there was an increase in youths' smoking through much of the 1990s that has been essentially maintained over the early years of this century. In addition to those who were current cigarette smokers (i.e., smoked every day or some days), 22% of U.S. adults were former smokers. Hence, well over half (57%) of U.S. adults had never smoked.

The shares of both men and women who smoke are still high. The age-adjusted percentages of men and women 18 years and over who smoked in 2004 were 23 and 19 (U.S. NCHS 2006d). The gender gap was much smaller than a generation earlier but is still sizeable. In 1965 51% of men and 34% of women 18 years and over smoked. Clearly a larger decline occurred among men than women. This is reflected indirectly in the slower rates of decline in death rates from lung cancer among women than men.

Cigarette smoking is the single most important preventable cause of premature mortality in the United States. The CDC estimates that about 400,000 deaths in the United States each year are smoking-related; that is, smoking "causes" one in five deaths each year. The validity of this estimate depends on the selection of causes of death imputed to smoking. Some analysts view it as an overestimate. Smoking substantially raises one's risk of developing cancer, various cardiovascular diseases, and various respiratory diseases, and so it has the potential to reduce life expectancy by several years. It is responsible for almost a third of all deaths from cancer. Smoking during pregnancy increases the risk of complications of pregnancy, fetal losses, premature births, low-birth-weight infants, and infant mortality. It is teratogenic; that is, it tends to produce defects in the fetus *in utero*. Young infants exposed to second-hand smoke are at increased risk of Sudden Infant Death Syndrome (SIDS), asthma, and other respiratory conditions.

Illustrative calculations show that smoking takes a tremendous toll in years of expected life. Rogers et al. (2005) calculated life expectancies at age 20 for persons with different smoking histories and different social and lifestyle histories, using data in the NHIS-MCD file for 1990–1997 (Table 6.8). The figures they obtained cover a tremendous range. They indicate essentially that smoking reduces expected life sharply for both men and women, especially for current smokers who have certain other unfavorable characteristics. Current smokers whose lives are characterized by unmarried status, low income, less than some college education, stressful lives, relative physical inactivity, and overweight have only about half the life expectancy of the average U.S. male (54 years) and the average U.S. female (60 years) at age 20. The figures for persons with these characteristics are only 27 and 34.¹⁰

¹⁰Stress, having a parent with only a high-school, or more limited, education, growing up in a one-parent household, drinking alcohol, and having a poor academic record increase the chances of becoming a smoker. These are some of the characteristics that contribute to a relatively low life expectancy of smokers as well as nonsmokers.

Table 6.8 Life expectation at age 20 for men and women according to smoking status and other social and behavioral characteristics: 1990–1997

Smoking status	Average		Best case ^a		Worst case ^b	
	Male	Female	Male	Female	Male	Female
Never smoked	63.4	72.4	75.2	92.4 ^c	37.1	48.1
Current smoker	51.5	53.3	59.8	62.8	27.2	34.0
Former smoker < 1 pack a day	62.4	68.1	73.8	84.8 ^d	36.4	45.5

Source: [Rogers et al. \(2005\)](#). Copyright © John Wiley & Sons. Reprinted with permission

^aMarried, income of \$50,000+, some college education, drinks occasionally, uses seatbelt, does not experience a lot of stress, more active than peers, normal weight

^bNot married, income below \$50,000, no college education, drinks excessively, does not use seatbelts, experiences a lot of stress, less active than peers, weight too low or too high

^cFigure implies a life expectancy at birth of about 111 years

^dFigure implies life expectancy at birth of about 104 years

Consumption of Alcohol

About 55% of U.S. adults were current drinkers in 2004; that is, they had at least 12 drinks in a lifetime and at least one drink in the last year (U.S. [NCHS 2006d](#)). One in six adults were infrequent drinkers (i.e., current drinkers who had fewer than 12 drinks in the past year) and about two in five were regular drinkers (i.e., current drinkers who had 12 or more drinks in the past year). Of current drinkers seven percent were “heavier” drinkers (i.e., more than seven drinks per week for women; more than 14 drinks per week for men). Since 1997 the share of heavier drinkers among current drinkers has fallen only slightly (from 7.9%). About one in three adults were lifetime abstainers.

A more favorable picture is presented by the results of NHANES for 1999–2004 (U.S. [NCHS/Wright et al. 2009](#)). It was reported that 84% of U.S. adults used alcohol moderately or not at all, on the basis of the definition of moderation as 1 drink or less per day for women and 2 drinks or less per day for men. The NHIS figure for 2004 was 76%.

In general, the abuse of alcohol and of a variety of other addictive drugs is considered inimical to health, but research findings differ as to the amount of drugs that would have to be ingested to be harmful. Alcohol consumption in small amounts each day has been found to be helpful in retarding cardiovascular diseases. On the other hand, no one is advised to begin drinking alcohol for this purpose and consumption of several drinks a day is considered unhealthful. Where, as in Russia, excessive alcohol consumption is widespread, it has been found to be a major factor in the recent rise in death rates and the decline in life expectancy ([Nicholson et al. 2004](#)).

Sleep

Humans are “hard-wired” to require about 7–8 h of sleep each night. To secure this amount of sleep, they need to use about the same clock-hours each night. Millions of Americans suffer from sleep problems, sleeping too few hours or irregularly because of overcrowding of the daily schedule, irregular work shifts, a sleep abnormality, genetic influences, a bad lifestyle, and especially, mental illness.

About 63% of the U.S. adult population usually sleeps 7–8 h according to the National Health Interview Survey (U.S. NCHS 2006c). The survey also shows that 29% of the U.S. adult population and 24% of the population 65 years and over usually sleeps 6 h or less each night. There may be a genetic basis for an individual’s sleep requirements. Some persons have a gene mutation that reduces their sleep needs to less than 6 h per night and they recover more quickly from sleep deprivation.

On the basis of such data, the AARP Foundation and ILC-USA (2003) reported that sleeplessness regularly affects about one-third of older Americans. Most persons aged 65 years and over report at least one chronic sleep problem; insomnia occurs frequently after age 70. On the basis of the Alameda County study it was concluded that having less than about 7½ h of sleep per night has an adverse effect on longevity. Cappucchio (2007) maintains that consistently sleeping about 7 h per night is optimal for good health and a sustained failure to get this amount of sleep may predispose to ill health. Other analysts have also reported that lack of adequate sleep is associated with poor health and susceptibility to disease, particularly in older men and women (AARP Foundation and ILC-USA 2003; Miles and Dement 1980).

Lack of sufficient sleep may cause an increased risk of falling, disturbances in hormone production and metabolism, compromised immunity, and destructive changes in the nervous and cardiovascular systems. It may also result in cognitive impairment, high blood pressure, reduced sensory acuity, shortness of attention span, impairment of attention, memory, and comprehension, an increase in reaction time, a depressed mood, and, in extreme cases, symptoms of psychosis. Too little sleep is also associated with weight gain and damage to the body’s ability to regulate blood-sugar levels (increasing the risk for type 2 diabetes).

Experiments with rats indicate that mild, chronic sleep restriction may have long-term destructive effects on neurogenesis (i.e. ability to generate new brain cells) and neural function (Hairston et al. 2005). Hairston et al. maintain that the brain needs sleep more than any other part of the body and lack of sleep undermines the rejuvenating effect of new learning on the brain. On the other hand, too much sleep is associated with depression and cancer-related fatigue.

As with many of the risk factors discussed, a formal measure of adequacy has been developed. The Pittsburgh Sleep Quality Index has been devised for assessing the quantity and quality of sleep among older adults. It consists of 18 questions relating to the seven areas in which sleep problems occur.

Exercise/Inactivity

Every field study seeking to identify the factors contributing to health and longevity concludes with a recommendation for regular exercise suitable to the health condition of the person (e.g., Alameda County, Framingham Heart Study, Baltimore Longitudinal Study). Regular physical activity has been shown to have many positive effects, such as reducing falls, strengthening bones, increasing muscle mass, improving balance, aiding in sleep, enhancing musculoskeletal functioning, reducing stress and anxiety, boosting immunity, and reducing excessive weight and obesity. In addition, regular physical activity improves cardiac output, retards atherosclerosis, lowers blood pressure, and reduces the risk of heart disease and several types of cancers (especially breast and colon cancer), diabetes, and depression. Exercise contributes to one's recovery from cardiovascular illness or cancer. It may even improve cognitive functioning and lower the risk of acquiring Alzheimer's disease. This list suggests that the benefits of exercise may go far beyond its strength- and conditioning effects on health. Exercise seems like the universal health potion.

Exercise is the only intervention for humans for which there is some evidence that disability is prevented or the onset of disability is delayed. It has a positive effect on all the modulatory systems of the body. Exercise increases the level of hormones such as testosterone and DHEA, substantially reduces the levels of inflammation shown by biomarkers, stimulates the production of free radical scavengers, and in some instances improves the function of the autonomic nervous system.¹¹ Of course, an alternative explanation of the role of exercise is that people who exercise may simply be less ill.

In spite of the pervasive public admonitions about the need for regular exercise to maintain and enhance health, less than one out of three adult Americans engaged in regular leisure-time physical activity in 2004 (U.S. [NCHS 2006d](#)). NHANES for 1999–2002 reported that 36% of the civilian noninstitutional population engaged in moderate or vigorous leisure-time physical activity for 150 min or more per week. In addition, one out of three adults engaged in some, but not regular, leisure-time physical activity. The shares of men and women who are regularly active were within a few percentage points of one another in 2004, but as recently as 1998 there was a substantial gap in favor of men. There is a strong inverse relation between age and the percentage of persons who engage in physical activity; for example, 67% of persons 18–24 years old exercise regularly or some of the time and only 38% of persons 75 years and over do so. Older women engage in regular physical exercise much less than older men.

¹¹The biological mechanism(s) explaining the beneficial effects of exercise are not completely known. In the case of some types of cancers (colorectal cancer and breast cancer), exercise may help lower levels of insulin, insulin-like growth factor (IGF), and estrogen, which are associated with tumor growth. On the other hand, exercise may help only these cancers and not others because of their age and growth patterns.

While aerobic exercise is the key element in any exercise program, a complete program has four components: aerobic (cardiovascular) activity, weight (strength) exercises, stretching (flexibility) routines, and balance exercises. These varied activities are intended to counteract the tendencies of muscles, including the heart muscle, to shorten, become less flexible, and weaken with age, of bones to lose calcium, become thinner, and weaken, of the body to put on fat, and of the balance skill to deteriorate.

Nutrition and Diet

Humans need three basic categories of macronutrients, namely carbohydrates, fats, and proteins. A healthful diet calls for a mix of foods from these three categories, although in different shares. It also calls for a variety of micronutrients, that is, vitamins and minerals. The general guidelines for a healthful diet requires eating fruits, vegetables, nuts, whole grains, and low-fat dairy products, and avoiding saturated fats, trans fatty acids (partially hydrogenated oils), and processed sugars.¹² Some types of fat, specifically those from most plants and fish, are healthful foods. Poor diets are associated with a number of unhealthful outcomes, such as hypertension, overweight/obesity, bone-thinning, and high cholesterol, and ultimately with diabetes, atherosclerosis, heart disease, stroke, and some forms of cancer.

NHANES (U.S. NCHS/Wright et al. 2009) reported how well the U.S. adult population was following recommendations on several nutritional standards to reduce CVD risk in 1999–2002. These recommendations are the least likely to be followed of the lifestyle recommendations designed to reduce CVD risk. They relate to consumption of fruit, vegetables, salt, and saturated fat. On the recommendation to eat 2–4 servings of fruit per day, only 16% complied, and on the recommendation to eat 3–5 servings of vegetables per day, only 29% complied. The advice to consume 2,400 mg. or less of sodium per day was followed by only 31%, and 42% followed the recommendation to consume less than 10% of total calories from saturated fat per day. Far greater percentages complied with recommendations on smoking, alcohol use, and checking of blood cholesterol and blood pressure, with physical activity being intermediate.

Some research suggests further that certain foods and vitamin supplements may reduce the risk of Alzheimer's disease. These include fish, vitamins E and C, folates, and vitamins B-6 and B-12. The merit of some of these vitamin supplements is

¹²In 2002 the U.S. Department of Agriculture (USDA) revised its nutritional recommendations to include new “dietary reference intakes” (DRI) for proteins, carbohydrates, fats, and fiber. Its guidelines advise the eating of fruits, vegetables, whole grains, and unsaturated fats, and engaging in daily exercise. The World Health Organization endorsed these recommendations in 2003, adding the admonition to reduce salt consumption. In 2005 the USDA further revised its guidelines to urge limiting caloric intake and engaging in regular exercise, and modified its recommended food pyramid to put new emphasis on whole grains and healthful fats. Its customized food pyramid offers 12 different eating plans depending on age and level of activity.

that they are antioxidants and may therefore offset the deleterious effect of oxygen free radicals. Vitamin supplements should be taken sparingly, however; and they should not be taken as substitutes for a healthful diet. Healthy persons can obtain all their vitamins and minerals from their diet, with few exceptions, such as calcium supplements for those who need to improve their bone structure and folic acid supplements for women of childbearing age.

Malnutrition, undernutrition, overnutrition, and food insufficiency, refer to other nutritional risk factors and problems predisposing to disease. Malnutrition encompasses the other nutritional problems listed. Elderly persons are especially vulnerable to malnutrition for a variety of reasons, among them poor oral health, low income, poor appetite related to illness and medications, and insufficient knowledge of nutrition. There is no standardized test for assessing malnutrition among members of a population although a Mini Nutritional Assessment has been designed to identify older adults who have malnutrition or are at risk of developing malnutrition (DiMaria-Ghalili and Guenter 2008). It consists of 18 questions, including six questions used to screen older persons for their nutritional status and a dozen additional questions used to obtain more comprehensive information. The 18 questions include three anthropometric tests – body mass index, arm circumference, and calf circumference.

Inasmuch as the varieties of malnutrition are particularly common in the less developed areas, they are discussed further in Chap. 11.

Other Risk Factors

Many other risk factors may be grouped under the heading, lifestyle and behavioral factors. Like the other individual factors I have discussed, these are partly, sometimes even largely, under the control of the individual although there are always environmental, genetic, and stochastic influences involved. Overweight and obesity, social support and stress, hypertension and similar biomarkers, premature birth and low birth-weight, sexual practices, and immunization history are discussed in the following paragraphs.

Overweight/Obesity. In the present context obesity is treated as a risk factor predisposing to disease although some analysts treat obesity as a disease as well as a risk factor. Excessive body weight, whether in the extreme form labeled obesity or in the more moderate form labeled overweight, contributes to the risk of sickness and death. Obesity is associated with an increased risk for diabetes, gall bladder disease, some types of cancers (e.g., colon cancer, kidney cancer, postmenopausal breast cancer), and a range of cardiovascular conditions, including hypertension, coronary artery disease, congestive heart failure, and stroke. Obesity is also a contributor to osteoarthritis, chronic fatigue, low back pain, edema (i.e., swelling in feet and ankles), insomnia, indigestion, impotence, and various conditions of the hips and knees. The Framingham Heart Study found a strong link between overweight/obesity and heart failure. This risk increases steadily with increasing body weight; it is 104 percent higher for obese persons than for nonobese persons.

In short, obesity greatly increases the risk of premature death, either directly or indirectly, from a range of causes, and the more severe the obesity the greater the risk. In the United States a few hundred thousand deaths a year can be attributed to causes for which obesity is a major risk factor. [Olshansky et al. \(2005\)](#) have estimated that the elimination of obesity would add about 3 years to life expectancy at birth, that is, about the same number of years as the elimination of all cancers.

Anthropometric measures: Body mass index, waist-hip ratio, waist circumference. The most common measure of obesity is the body mass index (BMI). BMI is defined as the quotient of body weight in kilograms divided by height in meters squared ($\text{wt}_{\text{kg}}/\text{ht}_{\text{m}}^2$). Persons with a body mass index (BMI) of 25 or more are classified as overweight. The increase in the percent of overweight persons in recent decades in the United States has been spectacular. Between 1960–1962 and 2001–2004 the percentage of adults 20–74 years of age with a BMI of 25 or more rose from 45% to 66% ([Fig. 6.8](#) and [Table 6.9](#)). About 74% of persons between the ages of 55 and 74 years were overweight in the more recent period. This figure is 17% points greater than the corresponding figure only a quarter century earlier (57% in 1976–1980). The percent of overweight children aged 6–11 (defined in the note in [Fig. 6.8](#)) nearly quadrupled between 1963–1965 and 1999–2002 (from 4.2% to 16%), and the percent of overweight adolescents aged 12–17 more than tripled between 1966–1970 and 1999–2002 (from 4.6% to 16%).

Obesity is defined as having a BMI equal to or greater than 30 kg/m². The National Heart, Lung, and Blood Institute (NHLBI) has identified three classes of obesity. These correspond to three levels of risk: Class I = $30 \leq \text{BMI} < 35$; Class II = $35 \leq \text{BMI} < 40$; and Class III = $\text{BMI} \geq 40$. Obesity increases the risk for several diseases over an overweight condition, as noted above, and the risks increase further from Class I obesity to Class II and Class III obesity. Obesity is the most common nutritional “disorder” in Western countries. As of 2001–2004, 32% of the population 20–74 was obese (U.S. [NCHS 2006d](#)). Adult women are more likely than adult men to be obese (34% vs. 30%). A substantial percentage, between 11% and 14%, of children are classified as obese ([Troiano and Flegal 1998](#)). Older adults tend to be obese more commonly than younger adults; the percentage at ages 55–64 (38%) is 12% points greater than the percentage at ages 20–34 (26%). Then, apparently because of selectively greater survival of nonobese persons to the older ages, the percentage of obese persons falls to 22% at ages 75 and over.

The prevalence of obesity has risen sharply in recent years. Between 1960–1962 and 2001–2004 the percentage of adults 20–74 years of age who were obese in the United States increased from 13% to 32%, with most of the increase occurring after 1976–1980. The high prevalence of obesity and its rise are not limited to the United States; it is observed in all the more developed countries. In addition, the incidence of obesity is rising rapidly in the less developed countries as they take on the ways of living of the more developed countries.

After age 40, without regular activity, muscle mass is steadily lost and the lost muscle mass is often replaced by fat. The health threat lies with abdominal fat, the fat that collects around the organs in the abdomen, rather than subcutaneous fat, the

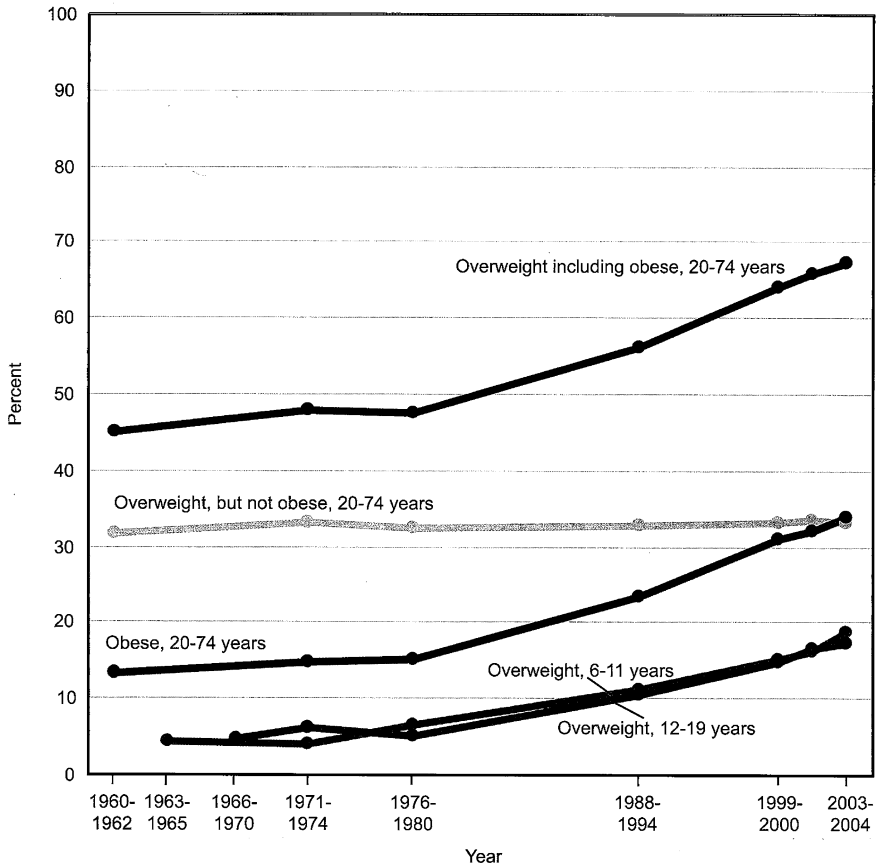


Fig. 6.8 Trend in percent overweight and percent obese for adults and in percent overweight for children: United States, 1960–2004 (Note: Percents for adults are age-adjusted. For adults, overweight including obese, BMI = ≥ 25 ; overweight but not obese, $25 < \text{BMI} < 30$; obese, BMI = ≥ 30 . For children, overweight = BMI at or above the sex- and age-specific 95 percentile BMI cut points from the 2000 CDC Growth Charts, United States. Obese is not defined for children. Points are plotted at the middle years of the periods indicated for the surveys; Source: U.S. National Center for Health Statistics (2006d), Fig. 13, p. 39. Primary source: National Health Examination Survey and National Health and Nutrition Examination Survey)

fact that lies just below the skin of the abdomen. Excess weight around the belly (i.e., abdominal obesity) is common among obese adults. Obesity with visceral, or abdominal, fat, is a particularly risky type of overweight condition because it is a high risk factor for a wide range of cardiovascular diseases and diabetes. The body mass index is not a good measure for this condition since the same BMI may represent very different amounts of abdominal fat and muscle may be distributed very differently in different individuals. Abdominal obesity is more satisfactorily and more simply measured by the waist-hip ratio, and it is indicated by a larger waist

Table 6.9 Mean body mass index of the U.S. population, by age and sex: 1960–1962 and 1999–2002

Age (years)	Male		Female	
	1971–1974	1999–2002	1971–1974	1999–2002
Under 20				
6–11 ^a	16.6	18.2	16.7	18.3
12–17 ^a	20.4	22.5	21.1	22.8
18–19 ^a	23.5	24.5	22.0	25.0
	1960–62	1999–2002	1960–1962	1999–2002
20 and over	NA	27.8	NA	28.1
20–74	25.1	27.9	24.9	28.2
20–39 ^b	24.8	27.0	23.2	27.4
40–59 ^b	25.6	28.6	25.8	28.9
60–74	24.9	28.6	27.2	29.2
75 and over	NA	26.8	NA	26.8

Source: U.S. NCHS (2004). Primary source: National Health Examination Survey and National Health and Nutrition Examination Survey

Note: Overweight but not obese, $25 < \text{BMI} < 30$; Obese, $\text{BMI} \geq 30$; healthy weight, $18.5 < \text{BMI} < 25$

Body mass index is calculated as weight in kilograms divided by height in meters squared
 NA not available

^aAverage of single ages

^bAverage of two 10-year groups

circumference than hip circumference. Waist circumference is intended to reflect the extent of abdominal fat, the “bad” fat, and hip circumference is supposed to reflect the extent of subcutaneous fat, the “good” fat. The standard value for the waist-hip ratio is 0.82, and the point at which the risks for health conditions begin to “take off” are 0.90 for men and 0.85 for women.

An even more direct and simple measure of the extent of abdominal fat is the waist circumference, a measure that is concerned only with abdominal fat since almost all the girth of the waist derives from abdominal fat. Determining waist circumference requires only a measuring tape. Like BMI, waist circumference is a good biomarker for heart disease, but it is a better predictor of diabetes than BMI. It has not become part of the regular medical diagnostic examination, however, perhaps because it does not seem “clinical” enough or because agreement has not been reached as to where the waist measurement should be taken. The principal recommendation is to take the measurement at navel level and to consider the cutoff point for health problems 40 inches for men and 35 inches for women. Recent research suggests, however, using even lower cutoff points, perhaps as low as 35 for men and 33 for women, and using waist circumference and BMI jointly in crossclassified scales. Some researchers prefer to employ as a proxy measure of abdominal fat the sagittal abdominal diameter, the length of a straight line through the body from slightly below the navel to the lower back, but this is not as simple to measure as waist circumference.

Fitness or low weight – which is better? The evidence shows that moderate physical activity as well as intense exercise can achieve weight loss. Because the efforts of many overweight women to reduce their weight by exercise meet with limited success, it is useful to ask, it is better to be fit than thin? The evidence shows that active women, thin or fat, are much less likely to have a heart attack or other cardiac problems than women who do not exercise. Nevertheless, weight does matter. Overweight women are still much more likely to develop diabetes than women who are not overweight, with or without exercise.

Being underweight can also be a health risk. It should be a matter of concern that persons of borderline “healthy” weight have an adequate reserve for the weight loss that accompanies many infectious and other illnesses and major surgery. A low BMI associated with unintentional weight loss is a greater mortality risk to older adults than is obesity with or without intentional weight loss (Locher et al. 2007). The curves of the BMI or the waist-hip ratio in relation to mortality are U-shaped; this reflects the fact that extremely low or extremely high values are “dangerous for one’s health.”

Causes of obesity. Weight gain occurs when the number of calories gained by eating (regardless of the type of food) exceeds the number of calories lost by exercise (of whatever type).¹³ Hence, weight reduction can occur only by reducing the intake of calories from food or increasing physical activity. Our proclivity to eat goes beyond our desire to satisfy our hunger. Eating is a source of pleasure, a way to deal with stress, a “duty” at social events, and a convenient preoccupation to avoid other more serious concerns, given that food is so readily available. For some, eating becomes an addiction. This occurs when the need to eat more than is required for sustenance becomes compulsive behavior, is subject to decreasing rational control, and becomes associated with changes in brain structure. In general, compulsive eating and an addiction to overeating are precipitated by easy access to food and poor lifestyle choices. People who act compulsively do not think about the act; the addiction is converted into a powerful automatic response. Once excess weight has been put on, it becomes extremely difficult to discard it even if a healthy life style and a diet plan are pursued in an effort to eliminate the extra weight.

Genetics and environmental/lifestyle factors both contribute to the onset of obesity, but their relative contributions are difficult to specify. Several genes influence a person’s weight but these genes have not been identified. Obesity tends to run in families but, inasmuch as the family is both a gene-transmitter and an environment for its members, the family connection complicates any attempt to determine the relative role of genetics and environment in the expression of obesity. Adult obesity, common in the postmenopausal years, is associated with obesity in childhood (Ferraro et al. 2003) and lower socioeconomic status, especially

¹³This statement is an oversimplification. There are circumstances where the volume of food eaten is effectively reduced by changes in the absorption rate, as when the immune system is compromised or a person undergoes a major surgical procedure or experiences other bodily trauma.

among women. The degree of obesity is exacerbated by a high-fat diet, alcohol consumption, a variety of medications, and physical inactivity, especially among older persons.

From the view of evolutionary biology, a substantial part of obesity is genetically caused and possibly triggered by hormones. Our body resists our efforts to follow a healthful lifestyle because it is “hard-wired” by evolutionary pressures to put on weight, keep its weight up, resist starvation, and defend itself from premature death (i.e., death prior to reproductive success) resulting from lack of food. Moreover, there is the biological tendency to store fat in the midregion of the body pending a food shortage and obviating the threat of starvation.

Social support and stress. Social support is positively associated (and social isolation is negatively associated) with physical and mental health and can reduce the stress caused by many adverse life events (Seeman et al. 2002). According to a study of the MacArthur Foundation for the Study of Successful Aging, a very strong predictor of well-being among adults is the frequency of visits with friends and the frequency of attendance at meetings of clubs and organizations (Rowe and Kahn 1998). One’s social network can be supportive and protective, and aid in coping with serious illness, death, and other adverse life events.

Social support mollifies the effects of these events by reducing stress. Much research has been done to identify and rank the events that create stress. These events include death of a spouse, death of a child, chronic illness of a child, divorce, loss of a job, a serious accident, the death of a close friend, a major lawsuit against the person, and destruction of one’s house by fire, flood, or hurricane. Other lifetime traumas are an unwanted pregnancy, rape, failing a school grade, divorce of parents, family violence, and witnessing a death. Death of a spouse is one of the most traumatic events; it is an event that removes from the survivor a principal source of social support. Note that some stressful events, such as birth of a child, marriage, and successfully passing a PhD preliminary examination, are potentially positive experiences and may contribute to “eustress” (“good stress”) rather than “distress” (“bad stress”).

A person will experience many major and minor stressful events during the life course. The effect they have on her or his physical and mental well-being will be determined by how she or he deals with them. The effect of stress on health and mortality also depends on the stage of life at which the stress occurs. Stress experienced by some persons early in life may result in their premature illness or death, even before they reach middle or old age, or in long periods of chronic disease and disability prior to death in later life; or it may endow them with a greater resilience or motivation to handle future stressful events. The perception of stress by individuals has an effect similar to the stress itself. How persons view their lives, cope with the demands made on them, and secure and use social support are important. To overcome stress, people at risk for a high level of stress can engage in exercise, Yoga, meditation, prayer, breathing exercises and other relaxation techniques, or other forms of personal stress reduction, as well as seek external social support.

Stress as a pathway to illness. We need a biological explanation of how negative lifestyle risk factors or lack of social support can increase the risk of disease, or how the availability of social support or absence of negative lifestyle risk factors can reduce the risks of disease. Although some lifestyle factors are directly responsible for disease (e.g., smoking, radiation exposure), diseases generally do not result directly from such factors. We need to find a mechanism or mechanisms by which the body is biologically altered and set on the path to pathological change. Stress may serve as that pathway. Stress has been linked to heart disease, stroke, cancer, chronic lower respiratory disease, and depression, anxiety, and anhedonia (i.e., an inability to experience pleasure). It is responsible for episodes of asthma, rheumatoid arthritis, and gastrointestinal illnesses. Other consequences of stress are headache, sleep and appetite problems, difficulty in swallowing, ringing in the ears, an urgent need to urinate, fatigue, and attention difficulties.

One theory connecting stress and a disease reaction maintains that chronic stress accelerates the aging process by shortening the life span of cells (Epel et al. 2004). In the Epel et al. study the cells of the immune systems of women under much stress (i.e., parents with chronically ill children) aged 10 years (i.e., lost life span) more than the corresponding cells of women under little stress (i.e., mothers with healthy children). Three factors in the cells of the immune system of research subjects were measured in this study: Telomeres, which cap the ends of chromosomes and become shorter as cells reproduce; telomerase, an enzyme that replenishes telomeres; and oxidative stress, which damages DNA in telomeres. The telomeres in cells of women under stress had a shorter length, telomerase showed lower activity, and the cells experienced greater oxidative stress.¹⁴

Some researchers suspect that hormones such as cortisol, epinephrine, and norepinephrine, which respond to stress, are responsible for the disease process. Animal and human studies link responses to stressful social relationships to the body's patterns of neuroendocrine regulation (Seeman and McEwen 1996). These patterns are part of the homeostatic regulatory processes of the body, that is, those that maintain its internal equilibrium. Nonsupportive social relationships induce the body's stress reaction, which entails enhanced responses of the hypothalamic-pituitary-adrenal (HPA) axis, the sympathetic nervous system (SNS), and other internal regulatory systems. Chronic neuroendocrine activity leads to such physiological responses as elevations in blood pressure, heart rate, and serum lipids – conditions that add to the risk for a number of chronic pathologies.

Inflammation: A possible unifying theory of major chronic diseases. The concepts relating stress, neuroendocrine regulation, and disease leads to a hypothesis linking inflammation as a physiological factor in the development of the major chronic

¹⁴Telomeres are DNA-protein complexes that cap the ends of chromosomes and promote genetic stability. When a cell divides, part of the DNA in telomeres is eliminated, and after many cell divisions, with the elimination of much of the telomeric DNA, the aged cell stops dividing. Telomerase is an enzyme that replaces a part of the telomeres during each cell division and protects them. Oxidative stress is a destructive metabolic process that contributes to the shortening of telomeres.

diseases. In the search for a unifying biological mechanism to complete the link between social risk factors, physiological responses to risks, and actual disease, many medical researchers point to inflammation as that mechanism. They hypothesize that inflammation is the common underlying cause of the major chronic diseases of later life – the cardiovascular diseases, diabetes, cancer, and Alzheimer’s disease. How does this hypothesis tie these diseases together?

Inflammation is the body’s protective reaction to an attack by disease. It is part of the immune response and hence is a beneficial immediate response to illness and injury. It results from the physical action of white blood cells and the chemicals they produce, including antibodies and cytokines. The characteristics of inflammation may be recalled by their rhyming Latin names – *rubor* (redness), *tumor* (swelling), *calor* (heat), and *dolor* (pain). Normally when the process of inflammation has done its job, neuroendocrine-regulation systems turn it off. According to [Seeman and McEwen \(1996\)](#), however, the stress response may persist, with “inflammatory” results. When the risk factors and stress persist, the neuroendocrine responses and inflammation may continue and the latter become factors themselves in the causation of disease. In this way, Seeman and McEwen hypothesize, excessive activation of the HPA axis and SNS increases the risks for a number of important pathophysiological processes, including hypertension and cardiovascular disease. As a further example, persons with excessive levels of C-reactive protein, a biomarker for inflammation, are more likely than others to develop insulin resistance, a precursor to diabetes. With this condition, cells reject insulin and do not properly metabolize glucose circulating in the blood.

Blood Pressure, Cholesterol, C-Reactive Protein, and Homocysteine

Hypertension, or high blood pressure, may be considered a disease, a cause of disease, and a risk factor for disease. High blood pressure can be a factor in heart disease, kidney disease, and stroke. According to NHLBI it is a factor in 50% of the heart attacks, two-thirds of the strokes, 90% of the cases of heart failure, and one-quarter of the cases of chronic kidney failure in the United States. High blood pressure compromises circulation and so adds to the risk of cognitive impairment and dementia.

Blood pressure is measured in two phases, diastolic blood pressure (i.e., pressure in the blood vessels between beats, or when the heart relaxes) and systolic blood pressure (i.e., pressure when the heart beats). For diastolic pressure, <80 mmHg is considered healthful and >90 mmHg is considered high. For systolic pressure, <120 mmHg is considered healthful and >140 mmHg is considered high. Blood pressure less than 120/80 mmHg is considered optimal and blood pressure over 140/90 mmHg is considered unhealthy. In 2003 NHLBI issued new guidelines for diagnosing high blood pressure. It set up a new blood pressure category, namely a “prehypertension” range for systolic pressure – 120–140 mmHg. This band covers 22% of American adults (18 years and over), or about 50 million people. A high

systolic pressure in combination with a low diastolic pressure is denominated isolated high blood pressure. This condition was formerly considered benign, but now it is viewed as a risk factor that requires treatment.

Over one-fifth of adult Americans had high blood pressure in 2004 (Table 6.7). The percentages generally move upward with increasing age, especially after age 45. The lifetime risk of developing high blood pressure at age 55 is 90%. Half the population 65–74 years of age has high blood pressure. A peak is reached at ages 75–84, with some decline thereafter. This decline is presumably a result of the selective demise of those having the disease, given that the population is heterogeneous with respect to this risk.

The specific cause of most cases of hypertension is unknown. A small share of the cases is caused by kidney disease, glandular tumors, diabetes, and some drugs (including some medicines for colds and migraine), but most cases are associated with a poor lifestyle, that is, physical inactivity, unhealthy diet, stress, smoking, and ingesting too much salt and alcohol. There is a wide array of medications for treating high blood pressure but, in treating the condition, elimination of the causes and predisposing factors enumerated above is usually considered first.

Research cardiologists differ on the merits of reducing high blood pressure at the ages over 80. [Goodwin \(2003\)](#) concluded from a number of research studies that higher levels of systolic and diastolic blood pressure in persons 80 years old and over are associated with increased survival. He notes that clinical trials of the treatment of hypertension for those 80 years and over result in outcomes favoring placebo over drug therapy and that for persons of this age range, high blood pressure is not associated with premature death. Goodwin cites epidemiological and clinical evidence (e.g., the Framingham study, EPESE) to support his conclusions. He measures success in terms of survival after 5 years. In several studies, both total mortality and cardiovascular mortality were substantially decreased in the treatment group under age 80 but not in the group 80 years and over. These studies also indicate that incidences of heart attack, congestive heart failure, stroke, and even cognitive impairment were reduced as a result of the use of medication even while mortality increased.

Others conclude from the available studies that there is increased survival of persons over age 80 when blood pressure is lowered with medication, or shows an insignificant rise. [Aronow \(2003\)](#) and [Newman \(2003\)](#) present this view, noting also that antihypertensive drug therapy improves the quality of life for persons 80 years and over by reducing adverse health events such as stroke even if it does not significantly lower mortality from all causes. The reason for this specific latter outcome may be that some aged persons in the studies have disproportionately low blood pressure from various health conditions, or from having their blood pressure lowered excessively by the medication, rendering them more vulnerable under conditions of sustained stress. The broader outcome may be a result of demographic selection in that these elderly populations may already have been “cleared” of those with the greatest risks. Thus the issue is whether treatment of hypertension in

persons over 80 is more important for increasing survival or reducing the incidence of several serious health conditions. In sum, there is no consensus about treating hypertension in the very old.¹⁵

Cholesterol. Cholesterol is a lipoprotein (i.e., a fatty organic compound) found in body structures and some foods. Excess cholesterol builds up in arteries in the form of plaque and adds to the risk of heart attack, heart failure, and stroke. Lipoproteins in the blood, measured by cholesterol tests, can be fractionated into high density cholesterol (HDL) and low density cholesterol (LDL), the former being good for cardiovascular health and the latter being bad for it. Levels of lipoproteins increase slightly with advancing age, particularly in women after menopause.

There has been a decline of a few percentage points in the cholesterol levels of the U.S. population in the last few decades. Cholesterol levels among all adults in the United States decreased from 206 mg/dL during the 1988–1994 period to 203 mg/dL during the 1999–2002 period, mainly as a result of the use of statin drugs (Carroll et al. 2005). The drop was accounted for entirely by older adults (i.e., men 60 years and over, women 50 years and over); levels for younger adults did not change. Current levels are still considered too high for good health; a total cholesterol below 200 has been recommended by the National Heart Lung and Blood Institute. Serious questions have been raised, however, about the merits of lowering cholesterol levels after age 80 using statin medications.

A person's genetic makeup affects the rate at which the body makes, uses, and disposes of fats. The statin class of drugs can be used to lower LDL and raise HDL. HDL levels can also be increased by changes in diet, exercise, and weight, and sometimes by doses of the nutrient niacin. Recent research suggests the desirability of more aggressive use of cholesterol-lowering drugs than has been the case, since both men and women at high risk of a heart attack or a stroke benefit from the drugs even if their cholesterol levels are within the healthy range. The recommendation is that high-risk persons – people who smoke, have diabetes or high blood pressure, or have suffered at least one heart attack – should lower their risk by reducing their level of LDL from the previously recommended 129 mg/dL to 100 mg/dL and even 70 mg/dL.

C-reactive protein and homocysteine. Elevated levels of C-reactive protein (CRP) are found in the blood when some disease in the body causes an inflammatory condition. Inflammation in the vascular system is usually indicated by a high level of CRP in the blood, and so the level of CRP/inflammation is now considered an “etiological” factor in heart attacks and strokes. A high level of homocysteine

¹⁵Hajjar (2003) believes that, until studies specifically designed to detect a survival benefit in the very old are conducted, aged patients with high blood pressure should be treated actively for that condition, barring other strong reasons for avoiding such treatment. This may be a prescription designed to protect physicians from the charge of not practicing in accordance with the state of the art. It will be difficult to secure definitive evidence for the advanced aged, however. As explained in Chap. 17, few very old qualify for research protocols at NIH, mainly because the pool of applicants is quite small and their many comorbidities confound the interpretation of research results.

in the blood is another biomarker for vascular inflammation and hence another major risk factor for heart disease and stroke. High levels of homocysteine may promote the formation of blood clots, injure the lining of the arteries, and increase the risk of atheromas (i.e., deposits of lipid-containing plaques on the walls of an artery) and atherosclerosis (i.e., thickening and reduced elasticity of the walls of an artery, resulting from atheromas). Homocysteine levels increase with advancing age, particularly in women after menopause.

Prenatal Care: Low Birth Weight and Preterm/Premature Birth

Low birth weight is defined as a weight of less than 2,500 g at birth. The corresponding population measure is the birth-weight ratio (per 100), defined as the percentage of births in a given year weighing less than 2,500 g. Low birth weight is a factor in infant mortality as well as poor health in later years. It can be prevented by proper prenatal care. Prematurity is usually associated with low birth weight. A preterm or premature infant is an infant born at less than 37 weeks of uterogestation. The population measure is the percent of births with gestational age under 37 weeks. Like low birth weight, prematurity can be prevented by proper prenatal care. Another measure of the general physical condition of newborn infants is the Apgar score. It rates five characteristics of newborn infants 5 min after birth and sums the scores of the five components, A score of less than 7 out of 10 is considered poor or fair. For the United States in 2004 the birth-weight ratio, percent of premature births, and the percent of newborn infants with Apgar scores under 7 were 8.1%, 12. 5%, and 1.5%, respectively (U.S. NCHS/Martin et al. 2006e)

Sexual Behavior

There is ample evidence that regular sexual companionship is conducive to both physical and mental health. This may explain in part the advantage of the married state over the other marital states with respect to longevity and health. On the other hand, having numerous partners and promiscuity constitute risky sexual behavior, associated especially with the risk of incurring sexually transmitted diseases. It was reported in the *New York Times* of March 12, 2008, that three-quarters of American adolescents are sexually active by the age of 19 and one-quarter of teenage girls has a sexually transmitted disease. Sexually transmitted diseases, including the most serious of them, HIV/AIDS, will be discussed further in Chap. 11 because of their widespread prevalence and pervasive consequences in many less developed countries.

Immunizations

I include immunizations as a risk factor to call attention to the fact that failure to secure proper childhood and other immunizations exposes the person to the risk

of incurring many infectious diseases with serious health consequences, including death. The formulation of the germ theory of disease a century and a half ago led to the development of vaccines as treatment modalities. Many types of immunizations are well established as standard procedure in preventive public health. The DTP vaccine, the vaccine used to protect children against diphtheria, tetanus, and pertussis, is one such standard vaccine. New vaccines continue to be developed for old diseases and new ones. For example, a vaccine against *Haemophilus influenzae* type b (Hib) was developed in 1987. Since then, Hib infections have decreased by more than 99%. The incidence of Hib meningitis, previously a leading cause of acquired mental retardation, has also decreased by more than 99%.

A vaccine against *Staphylococcus Aureus*, a major cause of death and infection in hospital patients, became available in 2002. *S. Aureus* causes illnesses ranging from minor skin infections to pneumonia, meningitis, and heart infections, particularly in persons with weakened immune systems. More recently a form of *S. Aureus* resistant to available antibiotics known as MRSA evolved and is contributing to a resurgence of *S. Aureus* in hospitals and in the community. A vaccine for typhoid fever is under development; it shows a 91.5% effectiveness rate in clinical trials. Perhaps most important of all are the current efforts to develop a vaccine for HIV/AIDS; clinical trials for such vaccines are now under way.

The Stochastic (Chance) Factor

Health and longevity are greatly influenced by stochastic processes as well as by genetics, lifestyle, and the environment. Chance is a major cause of individual differences in morbidity and mortality since random events occur at all stages of life, from fetal development and birth to the oldest ages (Finch and Kirkwood 2000). Chance events affect the physiological development of the individual and have potentially positive and negative effects on the individual's growth. As a result, chance plays a major role in determining the cause and timing of the events of sickness and death.

Considerable phenotypical variations (i.e., differences in observed traits) have been found among individuals reared in relatively homogenous conditions, partly because of the stochastic factor. Thereby, identical twins reared together show differences in health and longevity. Persons reared with different lifestyles may experience a common negative health outcome depending on the workings of chance. For example, some people die of lung cancer who do not smoke or work or live in smoke-contaminated environments. People suffer from peripheral vascular disease, coronary artery disease, and abdominal aortic aneurysms even though they have lived a healthful lifestyle and have monitored their health regularly. In sum, chance, interacting with a person's lifestyle, environmental experiences, and genetic predispositions, can account for many unexpected adverse outcomes.

On another level of analysis, the causes of death compete with one another to take the life of an individual. Death is a random event and, to a large extent, the

reported cause of death is arbitrary. As noted in Chap. 3, when individuals die, they usually suffer from an underlying cause (which is typically the reported cause), contributing causes (including the immediate cause and other conditions leading to death), and other health conditions. With such an array of health conditions, it is quite likely that, if they had not died from the reported cause, they would have died shortly thereafter from one of the contributing causes or other associated conditions, or a combination of them. According to [Hayflick \(2001\)](#), the assignment of a conventional cause of death is unrealistic and all intrinsic deaths result from the cumulative “molecular disorder” that characterizes the aging process in persons who live long enough to experience it.

Consider the case of the distinguished biogerontologist, Roy Walford, who died at age 79 in 2004 of amyotrophic lateral sclerosis (“Lou Gehrig’s disease”). He devoted many years to research into and writing about the requisites for a long life. He religiously pursued a healthy lifestyle, which for him mainly involved exercising regularly and eating a very low-calorie nutritious diet. Such “caloric restriction” is known to contribute to an increase in the life span of several species of subhuman animals. In Walford’s case the confluence of genetics and chance were dominant and intervened to take his life prematurely in spite of his efforts to “live right.”

Interrelations of Genetic and Nongenetic Factors

We recognize then that the etiology of sickness and death involves complex interrelations of genetics, environment, lifestyle, and chance. All these factors are always involved and they operate interdependently. Global figures for the contribution of these factors to longevity are usually expressed, however, as a dichotomy between genetic and environmental/lifestyle factors; that is, the structuring of the question neglects the role of chance. Even so, experts disagree as to the relative importance of these influences on human disease. Furthermore, this way of structuring the problem can be misleading in that it conceals a considerable degree of variability. The share contributed by each factor varies not only with the cause of illness or death but also the age, sex, and race of the subject. For example, lifestyle risk factors dominate in the teenage years and youth as direct and relatively independent causes of illness and death (i.e., accidents and other deaths of violence). At the later ages genetic and nongenetic causes play more complex interactive roles. Cumulative chance mutations account for changes in the genes, and environmental “hits” result in numerous changes in them as immunosenescence weakens the body’s ability to ward off internal and external “enemies.”

At the ages under 50, inherited genetic diseases may also be important, although they are uncommon and confined to a select list of conditions. When genetic diseases occur early in life, their expression (usually determined by a single gene) is little influenced by environmental and lifestyle factors. At the older adult ages, however, lifestyle and environmental factors are more likely to be collaborating with genetic forces (usually represented by combinations of genes) in influencing the

cause and timing of illness and death. These are the usual conditions under which the various endogenous/intrinsic illnesses manifest themselves. Through all these developments, chance is always involved and the determination of the time, form, and site of the chronic conditions of later life can be described as an essentially stochastic process.

Environment and behavior can be viewed as dominant over genetics in the causation of many diseases on the ground that they alter gene activity, determining when and how long given genes are active, i.e., when they manufacture new proteins. This can be important because the genes may have multiple subtle abnormalities and, depending on environmental and behavioral influences, may express themselves normally or abnormally. External influences having notable effects on gene expression are stress, exercise, radiation, use of drugs, and diet. For example, neurogenesis, i.e., the creation of new nerve cells, can occur even in adult life as a result of appropriate environmental and behavioral influences.

The presentation of celiac disease illustrates how genetic and environmental forces interact to determine the course of some diseases, even clearly genetic diseases. Celiac disease is a chronic disorder of the gastrointestinal tract characterized by an inability to metabolize gluten and causing chronic inflammation of the intestinal mucosa. Although the disease has its major genetic risk factors (HLA-DQ2 and HLA-DQ8), it requires an environmental trigger, namely, specific peptides present in wheat, rye, and barley, to activate it. Most patients experience complete “remission” from their symptoms after these grains are excluded from their diet.

Earlier in this chapter I cited some estimates of the relative contributions of genetics and environment to the causation of mortality. The National Institute on Aging (Hodes 2005) estimates that two-thirds of the variance in mortality can be accounted for by nongenetic forces and only one-third by genetic forces. Carnes (2005) provides a similar estimate. Finch and Tanzi (1997) also estimate the relative contributions to be about one-third genetic and two-thirds environmental. Vaupel et al. (1998) offered a more nuanced explanation of the role of genetics and environment that reflects the complexity of the relation better:

A frailty model applied to Danish twin data . . . suggests that about 50% of the variation in human life-spans after age 30 can be attributed to survival attributes that are fixed for individuals by the time they are 30; a third to a half of this effect is due to genetic factors and half to two-thirds to nongenetic survival attributes (related to, for example, socioeconomic status or nutritional and disease history). The model suggests that the importance of survival attributes may increase with a person's life expectancy. For persons who at age 30 can expect to live into their 90s, more than 80% of the variation in life-span may be due to factors that are fixed by this age. These calculations were made by I.A. Iachine on the basis of a frailty model described by Yashin and Iachine (1997).

Rowe and Kahn (1998:64–65) maintain that, with advancing age, genes play an increasingly minor role in the promotion of single risk factors, such as hypertension, and even full-blown disease, such as heart disease. Their view is that, with advancing age, the lifestyle factor is of paramount importance in causing these conditions. They maintain further that, inasmuch as lifestyle determines the ultimate impact of genes, lifestyle changes can prevent the presentation of a potential genetic trait. This

view of the role of the lifestyle factor goes beyond any of those previously cited. It is arguably too expansive, since genetics, certain environmental influences, and chance are still primary players in this “dance of power.” Even if such a broad role for lifestyle has limited demonstrable validity, however, the championing of it may have considerable practical merit in serving to induce people to be responsible for their own health and try to be proactive in maintaining it. While the dictum that people are entirely responsible for their own health is patently not true, promoting it can have highly positive consequences. Under a future regime of personal medicine, when the genomic profile of each individual can be determined quickly and cheaply and developments in nanomedicine/nanogenetics will provide expanded understanding of the function of individual genes, this view can be reviewed more critically.

Heritability Index

The measure used for the proportion of an observed disease or physical characteristic attributable to genetic influences is called the heritability index. In an informal sense, the heritability index refers to the degree to which a specific trait is passed on from parent to offspring. It is formally defined as the degree to which the total phenotypic variation within a population is due to genetic variation, that is, the ratio of the genetic variance to the phenotypic variance. The phenotypic variance (V_P) equals the sum of the genetic variance (V_G) and the environmental (i.e., nongenetic) variance (V_E):

$$h^2 = V_G \div V_P = V_G \div (V_G + V_E) \quad (6.15)$$

The heritability index is represented by h^2 . An h^2 of 1 signifies that all phenotypic variance is due to genetic variance. The complement of the heritability index ($1-h^2$) is a measure of the environmental (i.e., nongenetic) effect. Note that in this construct of heritability no allowance appears for stochastic variance, which is assumed to express itself through the other factors. When the index is considered in terms of variations by age and cause of death, a relatively low heritability index would be expected at the younger ages because of the predominance of exogenous (extrinsic) causes and a higher index would be expected at the later ages where endogenous (intrinsic) diseases prevail. Recall, however, the powerful role of lifestyle and environment in modifying these genetic tendencies in later life.

Individual vs. Categorical Risks

The vulnerability to morbidity and mortality of a person varies according to the various demographic/social/economic/biological characteristics of the group to which a person belongs (i.e., categorical risks) as well as according to the unique genetic endowment and personal experience of the members of the group (i.e., individual risks). In groups, however there is heterogeneity, especially in health. Heterogeneity of risk is pervasive in human experience. Age, gender, and race may

be viewed as biological characteristics for which risks vary, that is, characteristics that add to heterogeneity of risk. In Chaps. 3 and 4 the variation of mortality with age was discussed in considerable detail. Children and the elderly are more likely to succumb to various mortality risks (e.g., accidents, starvation, infectious diseases) than persons in the intermediate ages. Through these age-associated effects, among other ways, biological factors contribute to mortality in varying degrees, even of extrinsic mortality. Adding to the heterogeneity of risk of incurring disease in later life are three aspects of family life – socioeconomic conditions of the family when the individual was a child or teenager, life-course influences, especially health experiences, and current socioeconomic conditions of the family, as described below.

Earlier classifications of deaths have tended to assign whole categories of causes either to the endogenous/intrinsic or exogenous/extrinsic classes. For example, all cancers, cardiovascular diseases, kidney diseases, liver diseases, diabetes, congenital malformations and chromosomal abnormalities, and similar categories of causes were classified as endogenous; and all infectious and parasitic diseases, acute lower respiratory infections, and deaths due to violence (i.e., accidents, homicides, and suicides) were classified as exogenous. Bourgeois-Pichat (1952) divided deaths into these two broad classes composed of these broad categories of causes in his original formulation.

Many “endogenous/intrinsic” diseases result, however, from a complex combination of biological and environmental/lifestyle factors or from environmental/lifestyle factors almost wholly. For example, some diseases in the broad categories labeled endogenous/ intrinsic are initiated by external factors such as pathogenic microbes or self-destructive behavior. Accordingly, a narrower and more refined definition of intrinsic deaths than the one described above was employed by Carnes et al. (2006) for their analysis of mortality partitions in biogerontological research. In their classification, several causes of death included in the intrinsic categories listed above were reassigned to extrinsic mortality, either because the principal etiological factor is an infectious agent (e.g., rheumatic heart disease, cervical cancer, infection of the kidney, chronic hepatitis, urinary tract infection), the abuse of a toxic substance (e.g., lung cancer, cirrhosis, drug dependence), or a food deficiency or excess (e.g., nutritional deficiency).

Summary Note

Trying to measure the exact contribution of genetic and nongenetic forces to human mortality is a speculative task, like trying to determine the relative importance of the length and width of a rectangle when the use of the rectangle is unspecified. Genetic and nongenetic factors are inextricably intertwined. Important conditions such as age, sex, and cause of death have to be considered to make this determination meaningful. Moreover, chance plays a significant role in the development and expression of morbidity and mortality – a fact clearly evident in the case of the extrinsic causes of death involving violence, but also, as indicated, in the case of the

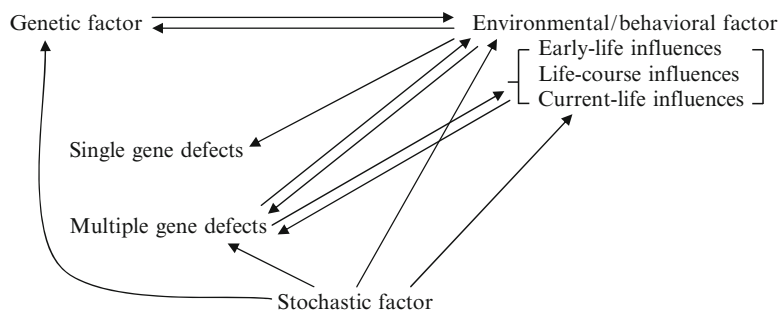


Exhibit 6.4 Schema relating various factors determining health and longevity in later life

endogenous causes of later life (See Exhibit 6.4). As result, the development and the nature, timing, and form of the expression of genetic and nongenetic factors are inevitably subject to stochastic influences.

Some important causes of death have a purely genetic basis that is present as a risk factor at conception (i.e., single-gene hereditary disorders), e.g., Huntington's chorea and sickle-cell anemia. Others involve genetic susceptibility and predisposition, and then triggering by the environment, e.g., Type I diabetes and celiac disease. Still others arise from somatic mutations that accumulate over the life course, e.g., congestive heart disease and prostate cancer, although there may be a genetic basis for these diseases also. Cumulative somatic mutations result from such factors as oxygen free-radical damage, radiation damage, protein cross-linking, reduced levels of heat shock proteins, and a decrease in hormone production (see Chap. 13 and [U.S. NIA 2006](#)). In all of this, chance may play the determinative role as to which genes are adversely affected, how they are affected, and the nature of the adverse effect on other genes and their protein production.

Some endogenous diseases have very large genetic contributions; for others the genetic contribution is small. There is always some genetic contribution if only because of the variation in the vulnerability of persons of different ages to different health conditions.

Early Life Influences on Health and Mortality in Later Life

Among the nongenetic factors that play an important role in the causation of chronic illness and death in later life are one's earlier-life experiences and environment, especially the socioeconomic status of one's family and one's health history in the earlier years. I discussed cohort effects on later-life morbidity and mortality to a limited extent in Chap. 5, and I pursue this theme further in this section. Four different interpretations can be given to the effect of early life experiences on later-life health and longevity of the members of a birth cohort. Much of the recent research has focused on the deleterious effects of ill health in childhood on

health in later life. Some of the research emphasizes the rise in socioeconomic status of the members of a cohort and the improvements in public health and medical knowledge as the members of the cohort grow older. Under these circumstances the members of the cohort tend to enjoy improved health opportunities and improved health conditions in later life as compared with their younger years. Some research has focused on the positive effects of having survived illness in childhood, whether by acquiring immunity to various infectious diseases or by becoming physically and emotionally hardened for survival to later years.¹⁶ Finally, early sickness and death tend to weed out the weaker members of a cohort so that the stronger members constitute a relatively larger share of the survivors in later life – an expression of the concept of heterogeneity of frailty in cohorts.

The life-course perspective is associated with the concept of a chain of risk, which posits that adverse experience in early life modifies the life course, usually in an adverse direction. The opposing concept, called the acquired-immunity concept, posits that early exposure to adverse and stressful experiences reduces the risk of a similar adverse experience in later life. The evidence would appear to argue that the chain-of-risk concept is the more tenable general interpretation of later-life health events than the acquired-immunity concept although both are at play among the individuals of a cohort and serve to explain the health-history of members of the cohort in different degrees. The heterogeneity-of-frailty concept has a more general application in characterizing all cohorts, with evident consequences for the health composition of the cohort in late life.

There is accumulating evidence of the importance of early life experiences in affecting health status in later life, including the experience of having lived both with the risk factors for disease and with actual disease (O’Rand and Hamil-Luker, 2005; Costa and Lahey 2005; Blackwell et al. 2001; Preston et al. 1998; Ferraro et al. 2003; Elo and Preston 1992, 1996; Hayward and Gorman 2004). O’Rand and Hamil-Luker found that early disadvantage and childhood illness increase the risk for a heart attack in later life but that adult experiences may mediate the effects of early disadvantage. Costa and Lahey estimated that at least one-fifth of the increase between 1900 and 1999 in the probability of a 65-year-old surviving to age 85 may be attributable to improved early life conditions. They attribute the remainder of the gain to a host of other changes, mostly public health improvements, such as filtration and chlorination of water, construction of integrated sewage systems, widespread vaccination against childhood diseases, improvements in the milk and food supply, and reductions in environmental pollutants and poisons.

Some studies point to the month or season of birth as strongly associated with longevity (e.g., Gavrilov and Gavrilova 2003). Fogel (1993), Steckel (1995), and Komlos (1993) have linked developed height, as a measure of net nutrition and health in infancy and early childhood, with health in later life. Barker (1997) offers

¹⁶In this view, exposure to a moderate level of stress may provide protection against the original stressor and sometimes against other stressors in later life and thereby add to health in later life and longevity. This process is called *hormesis*.

medical evidence connecting nutrition during life *in utero* and in infancy with adult health. He maintains that short-term disturbances in nutrition, especially during fetal life, as well as chronic malnutrition during early life, have long-term negative effects on health that may result in any of several chronic conditions (e.g., hypertension, stroke, coronary artery disease, and adult-onset diabetes). This conclusion has been subject to considerable debate and criticism (e.g., [Ben-Shlomo and Smith 1991](#); [Lundberg 1993](#); [Vågerö and Leon 1994](#); [Järvelin et al. 1998](#); [Leon et al. 1998](#); [Preston et al. 1998](#); [Vaupel et al. 1998](#); [Bengtsson and Lindström 2000, 2003](#)). These other researchers focus on conditions in early childhood as compared to conditions in fetal life. In the mix of early-life adverse conditions, stress and mental disorder may add to the likelihood of later-life adverse circumstances and the intergenerational transmission of social adversity and poorer physical and mental health (e.g., [Wickrama et al. 2005](#)).

The research on early life influences mostly distinguishes the mortality experiences of different members of the same cohort according to their own early health experiences. Other research shows that the early collective life experiences of a cohort are reflected in higher mortality of the cohort in later life as compared with the preceding and following cohorts. [Finch and Crimmins \(2004\)](#) single out inflammatory disease processes that severely affect basic somatic functions as such a precursor to increased death rates in the adult years. On the other hand, [Kannisto et al. \(1997\)](#) found no difference in the later mortality experience of cohorts born during famine and cohorts born before and after the famine.

We can extend the focus on early-life morbidity to include the accumulation of risk through the entire life course, as some authors have done ([Kuh and Ben-Schlomo 1997](#); [Ben-Shlomo and Kuh 2002](#)). They examined the later-life effect not only of fetal and childhood conditions but health conditions over the life span. Environmental and lifestyle influences on health, experienced at different stages of the life course, can affect the development of chronic diseases in different ways through their differential effect on physiological processes at different life stages ([Lynch and Smith 2005](#)).

Many health conditions of later life first make their clinical appearance in childhood, adolescence, or youth. For example, atherosclerosis has been found in many young men who were battle casualties. Most serious mental diseases, e.g., schizophrenia, depression, and schizoaffective disorder, first appear in the teen ages or youth. Some disease-associated conditions, such as being obese, appear first in childhood. [Ferraro et al. \(2003\)](#) have demonstrated the importance of excessive weight in childhood as a risk factor for obesity over the life course, especially between the ages of 45 and 64. This finding does not preclude a role for genetic and other factors as well in the chain of risk linking excess weight in childhood and severe obesity in later life.

On the positive side, good health in one's younger years fosters good health in the later years. If one becomes informed about practices conducive to good health as a child, follows good health practices during those years, and secures the care of health providers as needed, one's chances for good health in later life are greatly

improved. A favorable socioeconomic status in childhood and youth can contribute to good health practices and experiences in childhood, which become a strong foundation for maintaining good health in later life. A favorable socioeconomic status as a child or youth is defined by having parents with greater education, higher income, or a white-collar occupation.

Analysts have long noted the importance of cohort changes in accounting for the improvements in generational health. They report a general tendency for the mortality of infants and children to decline much earlier than for adults (Preston and van de Walle 1978; Kermack et al. 1934). Wilmoth (1988) and Kannisto (1994) maintain, however, that the changes that occur over the life course, such as the rise in the socioeconomic status of members of a cohort and the improvements in public health and medical practice in the community as the members of the cohort grow older, are more important than childhood circumstances in affecting health outcomes in later years.

It is evident that the momentum of cohort succession is superimposed on the role of early life influences on health in later life and may modify them. Given the considerable class mobility in the United States, the generally improving health of the younger population is reflected in improved health in a cohort in later years. This change results in the cohort's having more healthful children who, in turn, become more healthful adults. Thus, upward mobility leads to better health for the families of the upwardly mobile, which they "pass on" to their children by example, education, and provision of better care. These changes have been supported by progress in social and biological medicine and in medical technology. Medical and other human interventions have contributed greatly to the improvement of the health of children and youth as well as adults. The children have benefited particularly from immunizations, water purification, cleaner air, and a greater supply of nutritious food, and the adults have benefited from coronary artery, cardiac, and other surgery, organ transplantation, new diagnostic methods, improved medications for chronic diseases, and similar developments.

In sum, the focus on early life influences can be extended to life-course health-associated experiences, to cover transitions from childhood and youth to adulthood and older age. This perspective broadens the basis of late-life mortality and morbidity beyond the events of early life to include the totality of experiences throughout the life course. We can identify three component periods for the environmental/behavioral factor in later-life health and longevity, namely, early-life influences, midlife transitions, and current adult living conditions. Complementing the environmental/behavioral factor is the genetic factor. As stated, these factors interactively influence one another to determine health and longevity in later life. In addition, these factors are heavily influenced by chance at every stage of the life course. In the case of extrinsic (exogenous) morbidity and mortality, e.g., accidents, the role of the genetic factor is vastly reduced and may be described as secondary, whereas in the case of intrinsic (endogenous) morbidity and mortality, the role of the genetic factor is primary, but not usually determinative. A sketch of these relationships is shown in Exhibit 6.4.

Reclassification of Diseases According to Social/Preventive Causes

The above discussion of the genetic, environmental, lifestyle, and stochastic factors contributing to disease and causes of death included in the WHO international classification provides the background for an alternative grouping of the causes, one that goes beyond the conventional classification and even the classification of the causes as exogenous and endogenous. This alternative classification divides the causes into those that can be considered preventable by the individual and society and those that cannot. It calls for a division of the cause categories commonly classified as endogenous into those caused by social factors such as inactivity, use of tobacco, poor diet, and infectious agents and those caused by purely genetic and biological factors. This reclassification recognizes, for example, the role of tobacco use in causing lung cancer and heart disease, or of a poor diet and inactivity in causing cardiovascular disease.

One such reclassification was carried out by [McGinnis and Foege \(1993\)](#), who reviewed the literature on the etiology of the conventional diseases as listed on death certificates and developed a new distribution of the diseases in the United States for 1990 in terms of social/nonsocial causes. Some would call these social causes the “real” causes of the deaths with which they are associated. According to the recast distribution of deaths derived by McGinnis and Foege, of the 2,148,000 deaths in the United States in 1990, they classified 1,060,000, or about half, as socially caused and hence, preventable. About 19%, or 400,000, were attributed to the use of tobacco, 14%, or 300,000, to poor diets and inactivity, and 5%, or 100,000, to the use of alcohol. Smaller numbers were assigned to microbial agents, toxic agents, firearms, unsafe sexual behavior, motor vehicles, and illicit use of drugs. Recall that the leading “real” cause, tobacco use, has been indicted in the causation of a very wide range of lethal diseases: cancer, especially cancer of the lungs, esophagus, oral cavity, bladder, pancreas, and kidney; the leading cardiovascular diseases; chronic obstructive pulmonary diseases, particularly asthma, bronchitis, and emphysema; prematurity and low birth weight of babies of mothers who smoke; and accidental deaths from cigarettes causing fires.

The obvious implication of this reclassification is that a large share of all deaths is preventable even though it may not be possible to eliminate all “social” deaths entirely. If the preventable deaths were wholly eliminated, life expectancy would be increased by possibly 8 years. A substantial addition to life expectation would be achieved if only the population practiced a more healthful life style. For example, eliminating the effect of smoking would add possibly 2–3 years to life expectancy.

Different analysts would be expected to assign some causes to different categories than McGinnis and Foege did. More than one social cause may be associated with the same health outcome. Multiple causes may contribute synergistically to a particular outcome. For example, poor diet, inactivity, and smoking lead to heart disease and stroke. The analyst has the difficult task of apportioning the conventional underlying causes among the social causes without overlap. Cultural differences in different countries and in different regions of the same country may suggest

assigning different shares of deaths to different social causes. Furthermore, an evolution of thinking in the interpretation of the causes of morbidity and mortality may dictate an allocation of the causes to different social factors.

Obesity is now emerging as a new villain, replacing smoking. The proponents of the view that “obesity is the new smoking scourge” predict that the decline in life expectancy may come to an end within a few decades in the United States when the obese children of today reach adulthood (Olshansky et al. 2005). In the face of a so-called epidemic of obesity in the United States in the 1970–2004 period, the Centers for Disease Control and Prevention (CDC) announced that an estimated 400,000 deaths were due to obesity in 2005. For this estimate, obesity was presumably charged with many of the deaths from cardiovascular disease, cancer, diabetes, and other causes that were previously attributed to tobacco, inactivity, and poor diet.

Critics of this estimate argue that the boundaries of overweight and obesity were set too low, that a moderate degree of overweight is favorable for elderly persons, that obesity is merely a visible marker for other less obvious factors that account for many lethal diseases, and that there is no evidence that the rise in obesity has caused a rise in heart disease or cerebrovascular disease (diseases which, as we may recall, declined sharply in the period 1970–2004). (See Campos 2004; Oliver 2005; Gibbs 2005.) However, the rise in obesity can be associated with the rise in deaths from diabetes between 1988 and 2004. The CDC modified its estimate of deaths due to obesity after it was criticized for overstating its case; its latest estimate of obesity-related deaths is 365,000. A recent study by Flegal et al. (2005), based on all three NHANES surveys, maintains that the evidence is lacking that there is any measurable mortality toll among overweight Americans as a group in relation to people who are of healthy weight.

Limited Effectiveness of Personal Behavior in Retarding Age-Related Health Changes

Many research studies point to the differences in longevity between those who are free from the main risk factors and those who are not, but evidence that risk-factor modification is fully effective in preventing the occurrence or halting the progression of the leading chronic diseases is not strong. Regular exercise, proper eating, avoidance of stress, better sleep practices, cessation of smoking, and moderate consumption of alcohol may aid in slowing the progression of some diseases for some people, but their effectiveness in retarding age-related health conditions is limited. That is because of the continuing and powerful effect of age-related physiological dysregulation of the cells of the body and the important roles of genetics and chance in the etiology of disease. For example, although hyperlipidemia, hypertension, hyperglycemia, and hyperhomocysteinemia have been identified as risk factors for peripheral artery disease and coronary artery disease, efforts to modify these risk factors will not necessarily prevent these disease outcomes or keep them from becoming progressively worse. Millions of Americans

who closely follow the guidelines for a healthy lifestyle still acquire many serious diseases and, having acquired them, cannot stop their inexorable and relentless progression. Risk-factor modification may be more effective in this regard in the earlier ages of the post-reproductive years than in the later ages of life.

Demographic Characteristics of the Disabled and Nursing Home Populations

This chapter concludes with some notes on the demographic characteristics of the disabled and the nursing-home populations.

Disabled Population

According to U.S. American Community Survey (ACS), the disabled population 5 years old and over in the United States numbered an estimated 39.8 million in 2005, or 14.9% of the population 5 years old and over. The survey questions distinguished several types of disability: self-care, sensory, physical, mental, employment, and “go-outside-home” disability (*i.e.*, inability to go alone to shop or visit a doctor’s office). The survey total excludes the disabled population living in group quarters, which encompasses nursing homes, correctional institutions, college dormitories, military quarters, and group homes. In spite of the fact that, according to the 2000 census, all or nearly all residents of nursing homes have at least one disability, adjustment of the data for the omission of the nonhousehold population in the ACS of 2005 raises the original ACS percentage to only about 15.3%. This adjusted ACS figure is well below the 2000 census figure of 19.3%, which includes the nonhousehold population as well as the household population.

As expected, age-specific disability ratios rise with advancing age. For the household population in 2005, the disability ratio is 6% for ages 5–15 years, 12% for ages 16–64 years, and 41% for ages 65 years and over (Table 6.10; Fig. 6.9). (See also Freedman et al. 2004; Lutz and Scherbov 2003.) The adjusted ACS figure for ages 65 years and over for 2005 is 43%, as compared with the reported ACS figure of 41%. The adjustment of the ACS to include the nonhousehold population raises the figure for the percent disabled among the elderly population only modestly. Among the elderly, a limitation in physical functioning (*i.e.*, walking, climbing stairs, reaching, lifting, carrying) is by far the most common type of limitation, affecting 31% of that population. The second most common limitation, affecting some 17% of the elderly population, is “going-outside-the-home” disability.

The disabled elderly make up a substantial share of the disabled population 5 years old and over (one-third), but the great majority of persons with disability limitations are younger than age 65 (two-thirds), and they are mostly persons of

Table 6.10 Disability status of the U.S. population, by age, sex, and type of disability: 2005

Type of disability and age	Total	Male	Female
Population 5 years and over	267,388	130,302	137,086
Without any disability	85.1	85.7	84.6
With one type of disability	6.9	7.1	6.7
With two or more types of disability	8.0	7.2	8.7
Population 5–15 years	44,586	22,811	21,776
With any disability	6.5	8.0	4.8
With a sensory disability	1.2	1.3	1.1
With a physical disability	1.2	1.3	1.1
With a mental disability	5.2	6.8	3.6
With a self-care disability	0.9	1.1	0.7
Population 16–64 years	188,041	92,647	95,394
With any disability	12.1	12.0	12.2
With a sensory disability	2.8	3.3	2.4
With a physical disability	7.2	7.7	6.7
With a mental disability	4.5	4.5	4.5
With a self-care disability	2.0	1.8	2.2
With a go-outside-home disability	3.0	2.6	3.4
With an employment disability	6.8	6.6	7.1
Population 65 years and over	34,761	14,844	19,916
With any disability	40.5	38.2	42.1
With a sensory disability	16.4	18.2	15.1
With a physical disability	30.8	27.4	33.3
With a mental disability	11.5	10.6	12.1
With a self-care disability	9.7	7.8	11.0
With a go-outside-home disability	16.6	12.0	20.0
Population 65–74 years	18,360	8,401	9,959
With any disability	30.3	30.0	30.5
With a self-care disability	5.8	5.0	6.4
Population 75 years and over	16,400	6,443	9,957
With any disability	51.9	49.0	53.8
With a self-care disability	14.0	11.0	15.6

Source: U.S. Census Bureau (2005) American Community Survey. www.census.gov

Note: See text for definition of types of disability

Percents of population in age group, except for absolute population numbers shown for each age group. Data are limited to the household population and exclude the population living in institutions and other group quarters. Data are based on a sample, and are subject to sampling error and to various types of nonsampling error. Numbers are given in thousands

working age.¹⁷ The proportions disabled are higher for females than for males at every age above about age 40 and, accordingly, at the older ages disabled women greatly outnumber disabled men. Both self-reports and performance-based measures

¹⁷According to the 2000 census 9% of the nursing-home population (155,000) is in the age group 16 to 64, the “working ages.” All of these persons may be assumed to have one or more

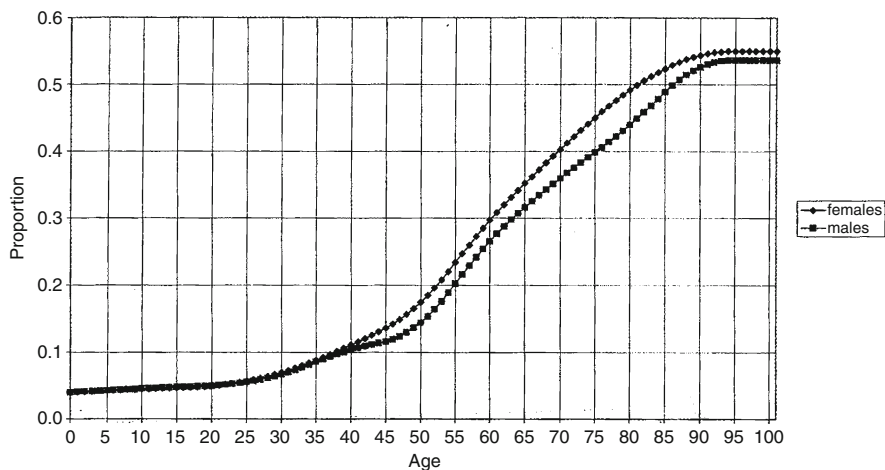


Fig. 6.9 Proportion disabled, graduated to single years of age, for males and females in the European Union-15: 1994–1996 (Source: [Lutz and Scherbov 2003](#)), Figure 2. Reprinted with permission. Primary source: European Community Household Panel (ECHP), 1994 and 1996; data provided by J.-M. Robine to Lutz and Scherbov)

indicate that blacks have significantly higher disability levels than whites, but socioeconomic status accounts for a substantial share of this difference ([Mendes de Leon et al. 2005](#)). Of the persons with disabilities, 91% live in housing units, 2% live in noninstitutional group quarters, and 6% live in institutional facilities.

There is strong evidence that age-specific disability ratios in the United States have been falling in recent decades. During the 1970s the increase in life expectancy was associated with an increase in years of disability of persons aged 65 and over, but during the 1980s and 1990s added years of life were associated with a substantial decrease in the proportion of disabled persons at these ages ([Crimmins 2001, 2004](#); [Manton and Gu 2001](#); [Freedman et al. 2002](#); [Manton et al. 2006](#)). Further, the analysis of Manton et al., based on the National Long-Term Care Survey, shows that the decrease was greater during the 1990s than during the 1980s. The age-standardized figures for the proportion of chronically disabled persons decreased steadily, on a per annum basis, by 0.6% from 1982 to 1984 and by 2.2% from 1999 to 2004/2005. These declines were found to be significant for both persons with less severe disability and persons with more severe disability. Manton et al. estimate that, if the disability ratios of 1982 had not changed since that year, there would have been 9.2 million disabled persons 65 years and over in 1999 rather than the actual 7.0 million (Fig. 6.10). These declines in disability cannot be interpreted to mean that the health of the U.S. population is better now than a few decades

disabilities. About 22% of the noninstitutional group-quarters population (818,000) and over half of the institutional population excluding the nursing-home population (800,000) is disabled. The great majority of these persons are under 65 years of age.

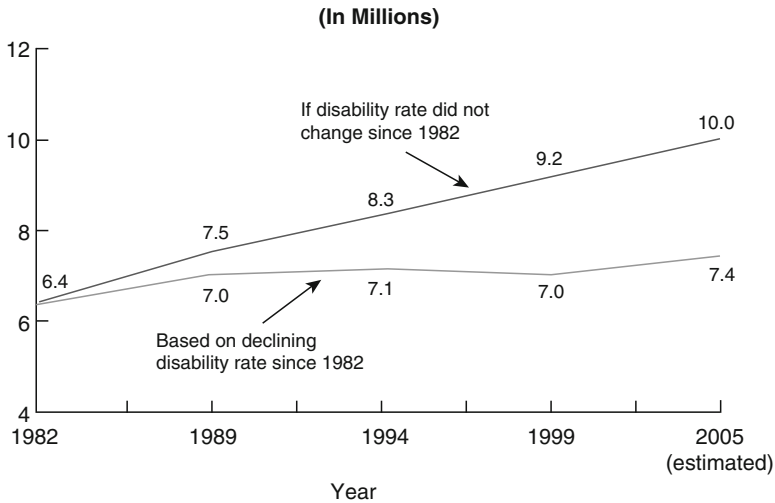


Fig. 6.10 Trend in actual numbers of disabled persons 65 years of age and over and in numbers assuming no change in disability ratios after 1982: United States, 1982 to 2004/2005 (Source: Based on [Manton et al. \(2006\)](#), Copyright © (2006) National Academy of Sciences, U.S.A. Reprinted with permission. Primary source: U.S. National Long-Term Care Survey, 1982–2004/2005)

earlier because alternative definitions of health (e.g., chronic disease prevalence) give contrary results ([Parker and Thorslund 2007](#)).

[Lutz and Scherbov \(2003\)](#) attribute most of the decline in disability ratios in the United States to the rise in educational attainment in the United States. They note that disability ratios tend to be much lower for the more educated segments of the population and that educational levels have been rising for the elderly population. The rise in educational levels at each age have combined with the variations in disability according to educational level and with the declines in disability within each educational level to account for the large decrease in the overall disability ratio at each age among the elderly and in the general disability ratio of the elderly.

About 16% of the population of the European Union was disabled in 1994–1996, according to the European Community Household Panel ([Lutz and Scherbov 2003](#)), but data on trends in disability for the European Union and other regions of the world, such as are available for the United States, are lacking. [Jacobzone et al. \(1998\)](#) reported disability ratios for six European countries at a single date, but not for a series of dates. However, [Egidi \(2003\)](#) reported decreases in the disability ratios of the elderly in selected countries of Europe for the 1980s and 1990s.

These findings have important implications for the outlook of the growth of the disabled population in the United States and Europe. If we combine the expectation of a tremendous aging of the population of the United States and Europe in the next half century (caused in part by the advent at the older ages of the massive number of baby boomers) with the fact that disability ratios increase with advancing age, we should expect a rapid growth in the number of disabled people in the United States and Europe in the next few decades. This expectation, however, is based on

the assumption that disability ratios will remain at present levels. If, as has recently occurred in the United States, disability ratios fall, it is conceivable that the number of disabled persons will not rise and may even fall in the United States (Fig. 6.10).

Sensitivity calculations to evaluate the effect of falling disability ratios have also been carried out for Europe as for the United States. If Europe experiences a decline in disability ratios at each age, such as the United States did, it is possible that population aging in Europe will not result in an increase in the number of persons with disabilities. Lutz and Scherbov (2003) show that, if disability ratios are shifted only two ages up the age scale per decade, the number and proportion of disabled persons will be about the same in 2050 as in 2000. Further, if the disability ratios are shifted three ages up the age scale per decade, the number and proportion of disabled persons will fall by 2050.

Nursing-Home Population

In the United States and many European countries, there has been, and continues to be, a tremendous increase in the number and proportion of persons who live alone and a corresponding decrease in the proportion of persons living in married-couple households and multigenerational households. This is significant because single persons are much more likely to require formal health care than married persons and are, therefore, at greater risk than married persons of entering an institution. Yet, nursing-home residence has been declining in the last several years. According to the National Nursing Home Survey (NNHS), 1,492,000 persons resided in nursing homes in the United States in 2004, while 1,628,000 persons resided in them in 1999.¹⁸ In the two prior decades, from 1977 to 1999, the number had been steadily increasing but its share of the total population remained steady.

The residents of nursing homes in the United States as a group are quite old and are getting older. The NNHS of 2004 reported that over three-quarters of the residents of U.S. nursing homes were 75 years of age or over and nearly half were 85 years or over:

Age group	Percent of all ages		
	2004	1999	1977
Under 65	11.7	9.7	13.0
65–74	11.7	12.0	16.2
75–84	31.4	31.8	36.0
85 and over	45.2	46.5	34.8
All ages	100.0	100.0	100.0

Source: U.S. National Center for Health Statistics/Decker 2004; NCHS (2008)

¹⁸Alternative estimates are provided by the 2000 census and the National Long Term Care Survey. The census figure is 1.72 million, for example.

Yet, many persons under 65 years of age reside in nursing homes; about 12% of the nursing home population, numbering 175,000 persons, was under 65 years of age in 2004. The nursing-home population has been aging, as shown by the sharp increase in the share of residents over age 85 in the two decades prior to 1999. This would be expected, given the aging of the general population during the same period.

The nursing-home population is a largely female population, only one-quarter of the residents being male. The main reason for this imbalance of the sexes is that women have greater longevity than men and, hence, are more likely to survive to the oldest ages, usually as widows. Recall that the chronic illnesses of men tend to be “killers” while the chronic illnesses of women tend to be “disablers.” The widowed status of women also means that they are less affluent and usually cannot afford private paid caregivers. Elderly persons tend to enter institutions for a combination of reasons, including mainly the unavailability of, or inability to afford, private caregivers, in addition to functional impairments and mental infirmity. Whites are overrepresented in nursing homes; blacks constitute 12% and Hispanics only 2% of the residents in nursing homes. Blacks and Hispanics show a great readiness to care for their elders at home and typically lack the means to support institutional care for them.

About one-third to one-half of persons 65 years old and over may expect to enter a nursing home some time in their lives. Two out of three persons will either never enter a nursing home or will stay in one for less than 3 months. Nursing home stays tend to be relatively short in spite of the public’s conception of their duration. Although the mean length of stay of residents from the time of admission, according to the NNHS of 2004, is over 2 years, the distribution is skewed sharply to the right because more than one-quarter of the residents stay more than 3 years. The median length of stay is only a little over a year. The percent distribution of residents of nursing homes by length of stay since the time of admission according to the NNHS of 2004 and a few earlier years is as follows:

Length of stay	Percent		
	2004	1999	1977
Less than 3 months	29.8 ^a	17.8	14.4
3 months to <1 year	14.3 ^b	25.0	22.1
1 year to <3 years	30.3	30.1	32.8
3 years or more	25.6	27.1	30.7

Source: U.S. NCHS/Decker 2004; NCHS 2008.

^aLess than 6 months

^b6 months to less than 1 year

These figures indicate not only that a large share of the residents stay less than 1 year but that the residents were staying for shorter periods in 2004 than in 1999 and 1977. Other pertinent information regarding the trend in the length of stay is provided by the rate of discharge for stays of less than 3 months per 100 nursing-home beds. The rate doubled between 1977 and 1999, rising from 46 in 1977 to 92 in 1999, while the rate of discharge for the other lengths of stay remained steady in this period.

We tend to assume that the entire nursing-home population is disabled in one way or another. The NNHS of 1999 shows that this is almost true. About half can eat on their own but only 6 percent can bathe themselves and only 13% can dress themselves. Two decades earlier the situation was much more favorable, when 30% could dress themselves, 13% could bathe themselves, and two-thirds could eat without assistance. This trend may be accounted for by the aging of the elderly population and the rise of assisted living residences and other alternative ways of providing long-term care. These residential alternatives to nursing homes take care of persons less in need of medical and rehabilitative care.

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