INTRODUCTION

What Are the Big Questions?

Why should human biologists study the demographic characteristics of humans and their close relatives? The primary reason is that demographic rates drive the process of evolution. Three of the four forces of evolution are demographic: mortality and fertility drive natural selection, migration is associated with gene flow, and population size and density influence the rate of genetic drift. Thus, demographic processes are responsible for the genetic structure of human populations considered in detail in Chapters 3 through 5. Demographic rates interact with human genetic architecture as well as environmental factors, which collectively drive the evolution of life history processes such as the rate of senescence, age at menarche, and age at menopause considered in Chapters 11 and 13. The theories underlying the evolution of these aspects of fertility and mortality all involve the interaction of genes with demographic rates (Stearns 1992).

Many of the most important questions in demography are variations on the following: How do human demographic rates—mortality, fertility, and migration rates—vary across populations and over prehistory, history, and the modern age? What are the causes, both proximate and ultimate, of the variation in these rates? What are the consequences of changes in demographic rates? For example, currently many researchers are concerned with population growth (a consequence of fertility rates exceeding mortality rates) and how it affects human health, the environment, local and global economies, conflict and warfare, and so on (see discussion of population growth in Chapter 15). Dramatic disparities in life expectancies among contemporary racial/ethnic groups raise concerns about variation in human health among populations. In recent years, life expectancies have been increasing worldwide, raising questions about the limits of human longevity. What are the
consequences—at the family, local, and national level—of having ever-increasing numbers of elderly individuals? Alternatively, what were the social implications of smaller numbers of elderly in past populations? When and why did a long post-productive life span emerge as a human life history characteristic?

This is the first of two chapters that deal directly with demography. Here we consider what is known about mortality and migration, and Chapter 15 considers fertility and population dynamics. The major issues considered in this chapter are the following:

1. How does the level of mortality vary prehistorically, historically, geographically, and by socioeconomic integration (i.e., hunter-gatherer, agriculturalist, industrial nations)? What are the causes of variation in the level of mortality?
2. How do the age patterns of mortality vary prehistorically, historically, geographically, and by socioeconomic integration? What do we know about the cause of this variation?
3. What are the levels and age patterns of migration, and how do they vary prehistorically, historically, geographically, and by socioeconomic integration?

First, however, we will consider some of the problems with the interpretation of demographic rates, since this affects the conclusions that can and should be drawn from the available data.

DEMOGRAPHIC RATES AND THEIR ESTIMATION

Fundamentally, demography is the study of mortality, fertility, and migration rates (see Box 14.1). Rates by definition incorporate a time element and are typically presented as the number of events (births, deaths, and migrations) per year per 1000 individuals. Demographic rates are often computed separately for each sex and for different age categories given that most rates vary with sex and age; however, aggregate rates are also computed. See Box 14.1 for additional details.

BOX 14.1 DEMOGRAPHIC RATES

Commonly reported demographic rates include measures of mortality, migration, and fertility (Preston et al. 2001). In general, the ideal demographic data consist of longitudinal observations on a population over the period in question with complete reporting of births, deaths, and changes in residence. Typically, however, demographic rates are estimated from cross-sectional data and may not be complete. Demographic rates are usually categorized into two types: crude rates and age-specific rates.

Crude rates refer to the population as a whole and do not account for any differences by age or sex. As described in Chapter 7, the crude mortality rate is defined as

\[ m = \left( \frac{d}{r} \right) \times 1000, \]  

where \( m \) is the crude mortality rate, \( d \) is the number of deaths during a year, and \( r \) is the number of individuals exposed to the risk of dying. It is traditional to present crude rates based on 1000 individuals. If longitudinal data are available, the number of person-years exposed to risk can be calculated exactly (Lee 2003). However, in demographic work, exposure to risk is often based on cross-sectional data and estimated as the "midyear" population, that is, the average of the population at the beginning of the year and at the end of the year. The crude birth, migration, marriage, and divorce rates can be calculated simply by replacing \( d \) with the number of the appropriate vital event and \( r \) with the appropriate exposure to risk.

Most demographic rates change with age and/or sex. Hence, demographic rates are often computed specifically for each age and sex. For example, age-specific mortality rates are computed as

\[ m_x = \frac{d_x}{N_x} \times 1000, \]  

where \( m_x \) is the death rate (aka the central death rate, or force of mortality), \( d_x \) is the number of deaths to individuals in the \( x \)th age category during a year (the value of \( x \) represents the beginning of the age interval), and \( N_x \) is the number of individuals in the \( x \)th age category exposed to the risk of dying during a year. As is the case with crude demographic rates, exposure to risk for age-specific rates is usually based on cross-sectional data and estimated as the midyear population of individuals aged \( x \). Typically, age-specific mortality (death) rates are computed for each sex by 5-year age categories, although 1-year age categories are sometimes used. Since infant mortality is of special importance (see below), the first 5 years of life are often broken down into 0- to 1- and 1- to 5-year categories.

Demographic rates for national populations are generally based on censuses and vital registration systems (formal records of births, deaths, and marriages). International migration is also registered in most contemporary nations, but internal migration is frequently not registered, particularly among the democracies. For most populations, the derivation of internal migration rates is particularly problematic.

Often net migration rates (the sum of in-migration and out-migrations) are computed from censuses, births, and deaths. In particular, the net number of migrants (\( M \)) is computed by rearranging the equation

\[ N_{t+1} = N_t - D + B + M, \]  

where \( N_{t+1} \) is the population at time \( t + 1 \), \( N_t \) is the population at an earlier time \( t \), and \( D \) and \( B \) are deaths and births to the population between times \( t \) and \( t + 1 \). Obviously, any errors in the census or in birth or death registrations will be transferred to migration. But even when migration is registered, migration rates are not necessarily easily interpreted. For example, in Great Britain, internal

(Continued)
migration is registered because of the national medical system, but the variation in medical district sizes and densities makes the resulting migration rates very difficult to interpret.

Estimating demographic rates for small populations is even more difficult because census materials and/or vital registration may be lacking. If everything is lacking, there is no hope. But when some data exist, it may be possible to derive demographic rates indirectly. These processes are discussed in more detail in Boxes 14.2 and 14.3.

The life table is an array of mortality rates by age (and some derived measures of mortality, such as the expectation of life at different ages) for a particular population. It has been an important methodological device for studying mortality for more than 200 years but can be applied to any kind of “duration,” for example, length of life, a marital union, or time between births. As a result, modern life-table analysis, survival analysis, or event history analysis and its extensions have become very important tools for the social sciences. The derivation of a life table is presented in Box 14.2. The interpretation of demographic rates is sometimes difficult, even when the data on which they are based are completely accurate. However, demographic data are often inaccurate, incomplete, and/or biased, which further complicates the problem. Demographic data may be inaccurate because people are missed by the census, births and/or deaths are not recorded, and ages and/or sex are either misestimated or misreported. The problem is compounded when these errors are unequally distributed among segments of the population. For example, inaccurate age estimates may be more frequent among the very old. Furthermore, bias in demographic estimates may occur when demographic rates are estimated “indirectly.” Indirect estimation typically uses a population theory to derive a rate from rates from complete data. However, the use of theories such as stationary population theory to estimate mortality or fertility results in bias if the assumptions underlying the theory are violated. (See Box 14.3 for further discussion.)

Finally, an important simplifying assumption of much demographic research is that the population is biologically and culturally homogenous. Where heterogeneity is known to exist, the subpopulations are usually examined separately. For example, mortality is generally considered separately for males and females. Females tend to live longer than males, at least in most present-day populations. Therefore, when a life table is estimated without distinguishing between the sexes, the younger age categories of the life table are based on about equal proportions of males and females, whereas the older age categories of the life table are based predominantly on females. Consequently, the estimated life table does not accurately represent the age patterns of mortality of either males or females or even a consistent average of males and females. Though the resulting life table still faithfully represents the deaths at each age of the population as a whole, some features of the age patterns of the life table might be the result of heterogeneity and not mortality per se. To complicate matters further, in many cases, subpopulations are not readily identifiable or the sources of heterogeneity are not known or understood. For example, before the 1960s the impact of cigarettes on mortality was not established and therefore not controlled for in life-table analyses. It is highly likely that additional factors will be identified in the future. Population heterogeneity is arguably most problematic for researchers who work with paleodemographic data, as many sources of heterogeneity are simply not detectable using skeletal samples. Until they are identified, the effects of heterogeneity cannot be completely controlled or assessed, and thus even the best empirical demographic data must be interpreted with caution. Wood et al. (1992a) have extensively reviewed the problem of heterogeneity with respect to paleodemography, and several recent paleodemographic studies have incorporated approaches that explicitly address heterogeneity and its effects on population dynamics in the past (e.g., see Usher 2000; Boldsen 2007; DeWitte and Wood 2008; DeWitte and Bekvalac 2010; Kreger 2010; Wilson 2010; and DeWitte 2010a).

In general, demographic data on small nonindustrial, non-Western populations tend to be of poor quality whether ethnographically or archaeologically collected. The problems of census coverage, missing vital events, and misestimating age are generally more severe than in large industrial populations. Skeletal samples are plagued by problems of differential preservation and biases in discovery and recovery of remains; even skeletal samples resulting from catastrophic events that kill many individuals in a short period of time are unlikely to provide unbiased demographic data given evidence that such events affect some categories of people more severely than others (DeWitte and Wood 2008). Furthermore, most published life tables available for these populations have been estimated by using stationary population theory (see Box 14.3), which can introduce serious biases if the population was not in fact stationary (Wood et al. 1992a). However, these small populations may be biologically and culturally more homogenous and have larger populations, reducing but probably not completely eliminating the misleading effects of heterogeneity. In general, small-population demography is particularly challenging. It has been aptly described as an attempt to “infer the unobservable” (Weiss 1969). But since the vast majority of human populations that have ever existed were small and lacking in “proper” demographic records, the attempt is worthwhile.

Demographic data from national European populations (after 1850 AD) are generally considered more accurate, whereas the data from the rest of the world’s nations run the gamut from very good in developed nations to completely lacking in some developing nations. As a consequence of the association of “good” demographic data with European populations, demography (even anthropological demographics) has an intrinsic national, industrial-era European bias. This situation is improving as more data become available from other time periods and regions of the world. However, the tendency is still to use the recent European demographic experience as “demographic analogies” for non-European populations, both contemporary and prehistoric. The rationale is the “uniformitarian” assumption that we are all human and hence should respond in at least roughly similar ways to similar environments (Howell 1976). This is not an unreasonable assumption as stated, because as yet, little convincing evidence indicates that much human variation in demographic rates is the result of genetic differences among populations. The problem with the “uniformitarian” hypothesis is that the extent to which human populations live or have lived in “similar environments” is not always clear. It is highly unlikely that industrial-era Europe is representative of the range of environments (biological and cultural) that human populations have been exposed to.
around the world and over the past 200 millennia. In addition, if we use it uncritically, the uniformitarian hypothesis can become a self-fulfilling prophecy.

In the following sections, we begin with the demography of historic European and contemporary developed nations (national populations) and then extend these patterns as carefully as possible to developing nations and to the contemporary and prehistoric populations typically studied by anthropologists (small populations).

MORTALITY

Demographers divide mortality into two independent phenomena: the level of mortality, commonly measured as an expectation of life, and the changes in mortality with age, that is, the age patterns of mortality. Both of these phenomena are estimated by using life tables (Box 14.2). Expectation of life is the average number of years remaining to an individual of a particular age and can be calculated for all exact ages reported in a life table. However, life expectancy at birth is the most commonly reported measure of the level of mortality. Expectation of life and the level of mortality are inversely related. High expectations of life at birth are associated with low levels of mortality and low expectations of life at birth are associated with high levels of mortality. But in high-mortality situations, life expectancy at birth can be disproportionately influenced by high death rates during early childhood mortality—especially infant mortality, which involves deaths among children less than 1 year of age—which may create a mistaken impression that no one lives very long after reaching adulthood. A life expectancy at birth of 35 years, for example, mostly reflects high mortality among the very young and should not be interpreted as suggesting that few adults live beyond the age of 35.

Box 14.2 Mortality Analysis: The Life Table

The life table is the standard method used for analyzing mortality data but has very wide applications within the social and biomedical sciences. It is useful for measuring the duration of any phenomenon: lives, marriages, birth intervals, hospital stays, and more. Life table analysis is now commonly referred to as hazards analysis or event history analysis to emphasize its broad applicability.

There are two kinds of life tables: actuarial and Kaplan–Meier. The major difference is that actuarial life tables categorize individuals into fixed age categories (usually 5-year age categories in abridged human life tables), whereas Kaplan–Meier life tables account for each individual by his/her exact age at death. The actuarial approach will be presented here because it is the best-known method in human biology. However, it is not necessarily the best procedure for use with small population data. (For a useful introduction to life-table analysis, see Lee 2003 or Preston et al. 2001.)

A typical actuarial life table for a small population is presented in Table 14.1. It represents the female population of the Trio of Surinam in 1-year age categories from birth to age 5 and 5-year age categories for ages 5–80. The last age category is open-ended and includes all individuals older than 80 years. The Trio individuals live in northern South America, in a tropical environment, and practice shifting cultivation.

The five standard columns of a life table are as follows:

- $q_x$ = the probability that an individual who survives to age $x$ dies within the interval, that is, between age $x$ and $x + 1$ or $x + 5$, depending on the interval;
- $l_x$ = the number (or portion) of individuals born who survive to exact age $x$, that is, to the beginning of the interval;
- $L_x$ = the person-years lived between age $x$ and $x + 1$ or $x + 5$, depending on the interval;
- $T_x$ = the total future person-years lived after exact age $x$; and
- $e_x$ = the expectation of life at age $x$.

It is traditional to assume that the life table represents an imaginary cohort of individuals exposed to a particular mortality schedule. An arbitrary initial size for the imaginary cohort, called the radix ($l_0$) of the life table, is selected. For Table 14.1, 100,000 was chosen. Once we have estimated the $q_x$ column, all the other columns can be computed from it (for technical details, see Preston et al. 2001). The life table provides a detailed picture of how the risk of death changes with age in a population—although, strictly speaking, it does not tell us why those changes occur.

The $q_x$ and $l_x$ columns are related and can be estimated from each other; if $l_x$ is known,

$$q_x = \frac{l_{x+1} - l_{x+n}}{l_x},$$

(14.4)

where $n$ is the length of the interval; for example, for ages 5–9, 0.01161 = (89,257 − 88,220)/89,257. If $q_x$ is known,

$$l_{x+n} = \text{radix} \left( \prod_{x} (1 - q_x) \right),$$

(14.5)

for example, for ages 5–9, 89,257 = 100,000[(1 − 0.0749)(1 − 0.01728)(1 − 0.00823)(1 − 0.00557)(1 − 0.00444)]. The next two columns in the life table, $L_x$ and $T_x$, allow for the calculation of life expectancy. The number of person-years lived within an age interval $L_x$ can be computed from $l_x$:

$$L_x = \frac{n}{2}(l_x + l_{x+n});$$

(14.6)

(Continued)
for example, for ages 5–9, 443,693.0 = (5/2)(89,257 + 88,220). The total number of years lived in a particular age interval and in all older age intervals \( T \), is, in turn, computed from \( L_x \):

\[
T_x = \sum_{x}^x L_x.
\]  

(14.7)

for example, for ages 5–9, 5,459,542 = 443,693.0 + 439,112.4 + 434,238.9 + 427,599.1 + 419,583.0 + 410,583.0 + 405,084.1 + 388,596.1 + 374,732.7 + 357,089.9 + 338,902.2 + 322,552.5 + 264,417.5 + 206,126.7 + 142,540.9 + 118,538.8. Finally, expectation of life at age \( x \), or the average remaining life span for those who survive to age \( x \) can be computed:

\[
e_x = \frac{T_x}{l_x}.
\]  

(14.8)

for example, for ages 5–9, 61.7 = 5,459,542/89,257.

There are several difficulties in estimating \( L_x \) exactly. Equation 14.6 works reasonably well at most ages and assumes that deaths are evenly distributed across the interval. However, during the first year of life, the majority of deaths occur within a few months of birth. Consequently, \( L_0 \) is often estimated as

\[
L_0 = 0.3l_0 + 0.7l_1.
\]  

(14.9)

This equation gives less weight to the number of individuals entering the interval, because many of them will die early in the interval and do not contribute heavily to person-years lived during this earliest age interval.

In Table 14.1, additional corrections suggested by Coale et al. (1983) were used, namely,

\[
L_0 = 0.27491l_0 + 0.72509l_0;
\]  

(14.10)

for example, for age 0, 94,564.0 = [0.27491(100,000)] + [0.72509(92,503)].

Furthermore, the last entry in the \( L_x \) column (\( L_{80} \)) is an open-ended category and difficult to estimate, as there are no individuals surviving at the end of the interval (i.e., the \( l_{80} \) portion of Eq. 14.6 is nonexistent). The best strategy is to carry the life table to an age at which all individuals are dead and then aggregate the results back to age 80. However, this information is often not available. In paleodemography, it is often assumed that

\[
L_{80} = n/2.
\]  

(14.11)

where \( n \) is chosen to be the length of the age category preceding the last open-ended age category; for example, for age 80+, 118,538.8 = (52/2)21,860. In general, this equation underestimates \( L_{80} \) and the columns to the right of \( L_x \). More complex methods for estimating \( L_{80} \) are available.

As was done in Table 14.1, the open-ended category can be estimated by extrapolating \( l_x \) to the end of life by using a mathematical hazard model such as the Gompertz equation, which provides a good model of the general human mortality pattern, between ages 30 and 80, of ever-increasing risk of death with age (Coale et al. 1983; Gage 1989). This procedure is probably more accurate but is still arbitrary.

The first column of the life table may be either \( l_x \) or \( q_x \). National life tables generally approximate \( q_x \) from the age-specific death rate (aka central death rate, or force of mortality) (see Box 14.1),

\[
m_x = \frac{d_x}{N_x},
\]  

(14.12)

without multiplying by 1000. The central death rate differs slightly from \( q_x \), which is the proportion of individuals entering the interval that die in the interval. The \( q_x \) value is always slightly smaller than \( m_x \) because the denominator of \( q_x \) is the number entering the interval, whereas the denominator of \( m_x \) is the number still alive at the middle of the interval (hence the name, central death rate). Provided that deaths are linearly distributed across the age interval,

\[
q_x = \frac{2m}{2 + m}.
\]  

(14.13)

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<th>Age</th>
<th>( q_x )</th>
<th>( l_x )</th>
<th>( L_x )</th>
<th>( T_x )</th>
<th>( e_x )</th>
</tr>
</thead>
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<td>21,860</td>
<td>118,538.8</td>
<td>118,538.8</td>
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</tbody>
</table>

* Adapted from Gage et al. (1964a).
the mortality distribution during childhood (not shown in Fig. 14.1), a hump in the mortality distribution during early adulthood (shown in Fig. 14.1), and a slowing of the rate of increase in mortality among the very oldest individuals (shown in Fig. 14.1).

A bump in the mortality distribution during childhood is often attributed to increased mortality associated with weaning. However, this explanation is not well supported empirically and consequently is not included in Figure 14.1. We are aware of only one study that provides convincing evidence of a bump that is attributable to weaning (Rosetta and O'Quigley 1990). There are no reports of this pattern among national populations with accurate demographic data. Consequently, most demographers believe that mortality declines steadily throughout the first few years of life. When a weaning bump appears in a mortality distribution, it is generally thought to be the result of inaccurate or incomplete data, probably owing to underreporting of neonatal deaths and thus underestimation of mortality at the preweaning ages.

The hump in the mortality distribution during early adulthood is frequently attributed to accidental deaths, particularly among young males who tend to engage in high-risk behaviors. This feature of the human mortality curve is deeply ingrained in the literature of national demography and occurs in some populations, primarily low-mortality western European populations (either living in Europe or elsewhere, such as in the United States and Australia). However, statistical analyses suggest that it is not a consistent or particularly important feature of human mortality (Gage and Mode 1993), even in those populations where it occurs. The importance attributed to this phenomenon by national demographers may be a consequence of the common practice of graphing the age patterns of mortality on a log scale, which is helpful in the visualization of data that cover a wide range of values but also greatly accentuates this feature of the mortality distribution (see Fig. 14.1b).

Finally, evidence is growing that mortality among the oldest old (90 years or older) does not continue to increase at an ever-faster rate (as it does between the ages of 30 and 80) but increases more slowly, or even levels off, at some maximum level of mortality (Perls 1995; Robine et al. 2006). Once again, however, there are potential problems with data: low numbers of individuals survive to these ages, and the data on the elderly are generally of poor quality. Thus, the leveling off of the mortality curve at the oldest ages may be an artifact of bad data (Coale and Kisker 1986; Elo and Preston 1994) since age is misreported more often in the elderly than in younger individuals. Alternatively, the leveling off of mortality may result from the fact that only physiologically exceptional individuals survive to the oldest ages (Vaupel et al. 1979; Economos 1982; Perls 1995; Robine et al. 2006); that is, it might be the result of selection on heterogeneity (variability) in health or vitality within populations: As more frail individuals die off, the remaining population is increasingly dominated by those with lower frailty, and thus, the risk of mortality for the population levels off.

In the remainder of the section on mortality, we present several issues of human biological interest: empirical evidence for variation in the level of mortality among populations and between the sexes, empirical evidence for geographical variation in the age patterns of mortality among national populations and between national populations and small populations, and the causes of variation in the level and age patterns of mortality.

The age pattern of mortality is usually presented as a list of age-specific mortality (death) rates (Box 14.2). A typical human (or for that matter, mammalian) age pattern of mortality is presented in Figure 14.1. Mortality tends to be high in the first few days and weeks after birth but declines rapidly, reaching a low in human populations usually between 5 and 10 years of age. Mortality remains relatively low throughout the next 20 years of life and then begins to accelerate at least until 70 or 80 years of age. The result is a rather asymmetrical U-shaped curve. Other more controversial features of the typical human age pattern of mortality are a bump in

Figure 14.1 Australian mortality 1970–1972. This is a low mortality population that displays a particularly strong "accident hump" during early adulthood. Panel (a) shows the data on linear scale, while panel (b) displays the data on a logarithmic scale (data from Heligman and Pollard 1980).
Variability in the Level of Mortality

The levels of mortality estimated to date by demographers, human biologists, and anthropologists vary widely among human populations, with expectations of life at birth ranging from perhaps less than 20 to more than 80 years (see Fig. 14.2). Many of the estimates at the lower end of this range are taken from paleodemographic studies and may reflect defective data or inappropriate analyses (as we discuss below). But reliable studies have frequently provided estimates of 30 or even a little less. Whatever the actual range, the environmental factors that account for the temporal and geographic variation in the level of mortality are not yet well understood. What is clear, however, is that the modern industrial revolution and the process of economic development are closely associated with dramatic reductions in the level of mortality.

The distributions of expectation of life for prehistoric, preindustrial, contemporary developing, and contemporary developed populations are presented in Figure 14.2. Before the beginnings of the industrial revolution, expectation of life at birth was short, less than 40 years. However, few present-day countries, regardless of their state of economic development, have expectations of life at birth less than 40 years. In general, expectation of life at birth among developing nations ranges from 40 to 70 years, whereas expectations of life greater than 60 years are characteristic of developed populations (Weiss 1973; Behn and Vallin 1982). Clearly, the range of variation in mortality appears to be much greater among contemporary populations than among prehistoric populations.

Table 14.2 presents some expectations of life among prehistoric small populations. The estimates are based on traditional paleodemographic methods that are using skeletal samples assuming stationary population theory and traditional aging techniques. The limitations of assuming a stationary population are discussed in Box 14.3. A theoretical solution to the problem of nonstationarity has been proposed (Wood et al. 2002; Gage 2010), but no applications have been carried out. Traditional paleodemographic aging techniques are known to be biased. The primary problems associated with traditional methods of age estimation are (1) underestimation of older adult ages and (2) age mimicry, whereby estimated ages are biased toward the age distribution of a known-age reference sample that is used as a standard (Boquet-Appel and Masset 1982; Boldsen et al. 2002). A number of
theoretically unbiased methods of aging have also been proposed in response to the criticisms leveled at traditional methods (Boldsen et al. 2002; Hoppe and Vatnæl 2002; Konigsberg and Frankenberg 2002; Müller et al. 2002; Konigsberg and Herrmann 2006; Samworth and Gowland 2007; Weise et al. 2009; Caussinus et al. 2010). However, no particular method has been generally accepted or widely used, and only a few reanalyses of prehistoric life expectancies are currently available (Konigsberg and Frankenberg 2002; Konigsberg and Herrmann 2006; Wilson 2010). In general, the results using new methods of age estimation indicate that expectation of life is underestimated by traditional methods. However, Storey (2007) reported unrealistically high estimates using the new methods. Clearly, this is an active area of research, and consequently the results in Table 14.2 must be considered with considerable caution.

**BOX 14.3 EXPONENTIAL MODELS OF POPULATION GROWTH**

Exponential models of population growth (i.e., models that assume growth rates are constant) are useful in both applied and theoretical research in demography. Here we focus on two reasons to use them: to predict short-term future population growth and composition, and to estimate demographic rates indirectly. Predicting future population levels is an important practical application of demography because this information is used by governments to plan for the future. This kind of application is most commonly used among national populations.

The second application of population models, indirect estimation, is critical to small-population demography because the data on these populations are often incomplete and/or defective, so demographic rates cannot be estimated directly. The models of population growth indicate the functional relationships among mortality, fertility, migration, and the age structure of a population. In principle, if three of these parameters are known, then the fourth can be estimated. Frequently, to simplify the theory, the population is assumed to be closed to migration. Because little is known about migration in small populations (see below), this convention will be followed here.

The three most common models of population growth are stable population theory, stationary population theory, and the so-called variable $r$ (nonstable) population theory. Historically, the most influential theory has been stable population theory. A stable population is one that has age-specific fertility and mortality rates that are constant through time; under these conditions, the "stable" age structure will be defined as

$$N_x = B \exp(-rx) l_x, \quad (14.14)$$

where $N_x$ is the number of individuals of age $x$, $B$ is the number of births, exp is the mathematical constant $e$, $r$ is the **intrinsic rate of increase**, and $l_x$ is the proportion of births that survive to age $x$ (see Box 14.2).

A population perturbed from its stable age structure—perhaps by war or an **epidemic**—will return to a stable age distribution over time, provided that mortality and fertility remain constant. A population’s age structure will effectively regain its stable configuration in about 70 years after a large change in demographic rates. Consequently, most human populations appear to have stable age structures. One well-known exception to the rule is the United States that has not yet achieved a stable age structure after the baby boom following World War II. Nevertheless, it is this tendency toward stability that makes stable population theory so useful. In any event, after a population has achieved its theoretical stable age distribution, it will grow or decline at a constant rate defined by $r$, the intrinsic rate of increase. Furthermore, each age category will grow or decline at a constant rate defined by $r$.

The importance of Equation 14.14 is that a life table ($l_x$) can be estimated from a census and the growth rate of the population, that is, an enumeration of population size at two points in time. If the population age structure is stable, then a census provides an estimate of $N_x$ and the number of births $B$ within the past year, the observed growth rate of the population provides an estimate of $r$, and an accurate life table can be estimated. An estimate of the distribution of ages at death from an archaeologically recovered skeletal collection and an estimate of the growth rate of the population may also be used to compute a life table (Moore et al. 1975). Similar procedures can be applied to indirectly estimate fertility (Weiss 1975). These methods are particularly useful when births and deaths are not registered or are incomplete and a life table cannot be computed directly from the data. Thus, these methods are suited for populations in many less-developed nations and most of the small populations studied by anthropologists. The necessary assumption is that the age structure of the population is theoretically stable, but human age distributions naturally tend to be close to their stable states, so this is likely a reasonable assumption.

Often, only a census is available for a population, and the growth rate of the population (an estimate of $r$) is not known. In such cases, investigators often assume that the population is stationary (i.e., not growing or declining). Stationary population theory requires two assumptions: that the age structure is stable and that $r = 0.0$. A life table then can be computed from Equation 14.14 and a census ($N_x$ and $B$). The problem is that whereas the assumption of stability is generally reasonable, because populations naturally tend toward stable age distributions, the assumption of no population growth or decline is much less likely to be true. If the population is growing and stationary assumptions are imposed, the increasing proportion of children in the population will create a more youthful age structure and decrease average age at death; mortality will therefore be underestimated; if the population is declining, then mortality will be underestimated. Consequently, mortality estimates based on the stationary population model must be interpreted with great caution. It has been argued that stationary estimates of average age at death are more sensitive to changes in fertility than mortality and thus provide better estimates of the variation in fertility than mortality (Coale 1957; Sattenspiel and Harpending 1983). This argument has even been used to estimate variation in prehistoric levels of fertility (Buikstra et al. 1986). However, the application of stationary population theory to fertility estimation is as prone to error as its application to mortality.

(Continued)
The stable population model has been extended to a nonstable population model (Preston and Coale 1982). The extension consists of simply replacing $r$ with age-specific rates of growth $r_i$. Equation 14.14 thus becomes

$$N_s = B\exp\left(-\sum_i r_i \right).$$

This equation provides a series of indirect methods that can be applied to any population—nonstable, stable, or stationary—provided that estimates of $N_s$ and $r_i$ are available. These values can be estimated from two censuses on the same population at two different points in time (Gage et al. 1984a, b; Gage 1985; Preston et al. 2001). If the data are available, this method provides assumption-free estimates of the level of mortality and fertility and should be preferred over other estimates. Unfortunately, it is infrequently used in anthropological applications.

The sustainable lower limit of human expectation of life is probably not much less than 20 years due to the levels of fertility that would be required to maintain a population under a heavier mortality regime. For example, the two lowest expectations of life in Table 14.2 represent New World populations around the time of European contact. The Mobjridge I and Larson sites, located in South Dakota, are associated with the Plains Arikara and date to AD 1600–1650 and 1750–1785, respectively. The very high mortality rates in these populations may be temporary and represent epidemics associated with contact; for example, it is known from historical records that the Arikara faced a devastating outbreak of smallpox in 1780–1781. However, the high mortality rates may also be the result of defects in the data (e.g., the populations may not be closed to migration because of the disruption of contact). Consequently, the lowest expectations of life are probably not realistic long-term estimates of human mortality.

Furthermore, it is clear that prehistoric estimates of expectation of life at birth will increase over those shown in Table 14.2 when the new unbiased aging techniques are more widely used. But this still may not explain the generally lower expectations of life among prehistoric populations compared with contemporary small and national populations. Only three expectations of life at birth are currently available based on the new methods. Koenigsberg and Frankenbogen (2002) estimated Loisy-en-Brie expectation of life at birth at 25 years, although they arrived at this by artificially increasing the number of infant deaths to account for underenumeration of infants (we will return to this below). Similarly, Kreger (2010) and Strott (2006) both reported estimates of 28 years for Postclassic Cholula and medieval Bavaria, respectively. All of these new estimates still fall within the range of those presented in Table 14.2. Overall, prehistoric life expectancy at birth appears to have fallen within a surprisingly narrow range, perhaps 20–40 years, generally below the range reported for contemporary small and national populations.

Given that mortality levels at the national level generally declined with the industrial revolution (as discussed below), it is commonly assumed that the agricultural revolution (the transition from nomadic hunting and gathering to sedentary agriculture) was associated with changes in mortality. Some maintain that mortality increased, based on evidence of lower mean ages at death and higher frequencies of skeletal pathologies in some skeletal samples from agricultural populations compared with samples from hunter-gatherers (see papers in Cohen and Armelagos 1984 and Cohen 1989); others argue that the evidence might just as well suggest a decrease in mortality, given the problems associated with estimating mortality in paleodemographic samples (Wood et al. 1992b). On the basis of the data presented in Table 14.2, there are few significant differences among these populations. The mean expectations of life are 21.6 years for hunter-gatherers (standard deviation [SD] = 2.1 years), 21.2 years for horticulturalists (SD = 3.9 years), and 24.9 years for agriculturalists (SD = 8.5 years). The mean for hunter-gatherers is between that of horticulturalists and agriculturalists. None of these differences, however, are statistically significant. Again, it is unclear exactly how the new unbiased aging techniques might influence absolute estimates of expectation of life at birth. However, comparisons among populations are less likely to be seriously affected. Consequently, the currently available data suggest that the level of mortality during prehistory did not vary a great deal from one population to another or one economic system to another; this is markedly different from the variation observed in contemporary populations, although it is consistent with predictions from recent theoretical models of preindustrial population dynamics (Wood 1998). These findings suggest that the greatest historical discontinuity in the demography of the human species may not have resulted from the origin and spread of agriculture some 10,000 years ago as some have suggested (e.g., Cohen, others), but rather from the “modern” industrial revolution that began in England in the late 18th century.

Among contemporary nations, expectation of life ranges from about 40 to above 80 years (see Fig. 14.2). Japan currently has the longest life expectancy: 79.1 years for males and 86.4 years for females (United Nations 2005). The current estimate of expectation of life at birth for the world’s population as a whole slightly exceeds 64.3 years for males and 68.7 years for females. Economic development and industrialization have been associated with dramatic declines in the level of mortality and increases in the variation of the level of mortality worldwide. In the 1970s and 1980s, Third World nations typically had expectations of life between 30 and 60 years at birth, whereas the developed nations had expectations of life above 60 years (Weiss 1973; Behn and Vallin 1982). As indicated in Figure 14.2, which includes more recent data, the range of expectations of life at birth among the developing nations is now from 40 to 65 years, with the “break” between the developing and developed countries occurring around 65 years.

Additionally, it has been observed in recent years that a few developing nations have surprisingly low mortality given their low gross domestic products, indicating that the decline in mortality is not strictly a function of economic development. Costa Rica for example has a surprisingly high expectation of life at birth; that is, it is only about a year less than that of the United States. Caldwell (1986) attributed this to dissolving the national army and the implementation of a national health care system in Costa Rica, although he went on to describe other unusual paths developing countries have taken to low mortality. Among developed nations, the
decline in mortality appears to have begun during the early 19th century. In developing countries, the decline in mortality began later, around 1920 (see Fig. 14.3). Mortality appears to have ameliorated worldwide in the developing countries beginning in the 1920s (Baker 1987; Gage et al. 1989), that is, independent of the level of industrial or economic development. The possible causes of this decline will be discussed further below.

Expectation of life at birth among living nonindustrial societies that have been studied ethnographically suggests that the level of mortality is lower among present-day small, nonindustrial populations (Tables 14.3 and 14.4) than among human populations studied archaeologically (Table 14.2). The data presented in Table 14.4 were analyzed using the same methodology as the analysis of the archeological data in Table 14.2, including combining the sexes (Gurven and Kaplan 2007). The data for populations presented in Table 14.3 are not all analyzed using the same methodology but are broken down by sex. Overall, the mortality of living small populations appears more similar to the mortality observed in the contemporary developing nations than to the mortality of socioeconomically similar archaeological populations presented in Table 14.2. With the notable exceptions of the contemporary Yanomama, Hiwi, and Agta, and perhaps of the rural Chinese in the 1920s, expectation of life in contemporary small populations is greater than 30 years (Tables 14.3 and 14.4). The mortality of acculturated small populations is even lower with a mean expectation of life of 37.7 years. The expectations of life of contemporary small populations fall at the upper range of prehistoric expectations of life at birth or above. It appears that either contemporary small populations in Tables 14.3 and 14.4 have lower mortality than the archaeological (prehistoric) populations, or there remain additional large and consistent errors in the data and analyses for the archaeological populations, the ethnographic populations, or both. One possible explanation is that like the developing nations, contemporary small populations also participated in a worldwide decline in the level of mortality observed in the developing countries that began about 1920.

There is no statistically significant variation in the level of mortality by economic system among living small populations listed in Tables 14.3 and 14.4. Hewlett (1991) collected evidence concerning the relative rates of infant and childhood mortality in contemporary hunter-gatherer, horticulturalist, and pastoralist small populations. The data consist of infant mortality (the death rate during the first year of life) and childhood mortality (the death rate during the first 15 years of life). These data suggest that, on average, hunter-gatherer populations have a mean infant mortality rate of about 20.3 per 100 (SD = 8.6), whereas horticulturalists have a mean infant mortality rate of 21.0 (SD = 4.3). The mean childhood mortality rate for hunters–gatherers is 43.4 (SD = 11.1), whereas for horticulturalists and pastoralists, the mean childhood mortality is 38.1% (SD = 10.4). Neither of these differences is statistically significant.

It is generally considered biologically “normal” for women to live longer than males of the same population, at least in large national populations (Lopez and Ruzicka 1983; Waldron 1983; Teriokhin et al. 2004; Preston and Wang 2006). The secular decline in mortality that has occurred over the past few hundred years

<table>
<thead>
<tr>
<th>Hunter–Gatherer Population</th>
<th>$e_{0}$</th>
<th>$e_{15}$</th>
<th>$e_{45}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hadza</td>
<td>34.0</td>
<td>42.5</td>
<td>24.2</td>
</tr>
<tr>
<td>Ache forest</td>
<td>37.0</td>
<td>38.5</td>
<td>21.1</td>
</tr>
<tr>
<td>Hiwi</td>
<td>27.0</td>
<td>32.2</td>
<td>17.9</td>
</tr>
<tr>
<td>!Kung</td>
<td>36.0</td>
<td>38.1</td>
<td>19.7</td>
</tr>
<tr>
<td>Agta</td>
<td>21.0</td>
<td>28.6</td>
<td>13.7</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td><strong>31.0</strong></td>
<td><strong>37.7</strong></td>
<td><strong>20.7</strong></td>
</tr>
<tr>
<td>Forager–Horticulturalist Population</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yanomamo Mucaj</td>
<td>39.0</td>
<td>41.3</td>
<td>23.5</td>
</tr>
<tr>
<td>Yanomamo</td>
<td>21.0</td>
<td>28.3</td>
<td>16.8</td>
</tr>
<tr>
<td>Tsimane</td>
<td>42.0</td>
<td>41.6</td>
<td>20.6</td>
</tr>
<tr>
<td>Guni</td>
<td>30.0</td>
<td>29.6</td>
<td>11.9</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td><strong>33.0</strong></td>
<td><strong>36.4</strong></td>
<td><strong>19.8</strong></td>
</tr>
</tbody>
</table>

| Acculturated Hunter Gathering Population |
| !Kung 1963–1974               | 50.0   | 50.9   | 25.2   |
| Ache reservation             | 50.0   | 52.0   | 27.2   |
| Northern Territory Aborigines| 49.0   | 47.6   | 23.1   |
| Hiwi post-1960               | 28.0   | 41.9   | 23.8   |
| Agta transition              | 28.0   | 35.7   | 14.8   |
| Agta peasant                | 21.0   | 30.6   | 13.4   |
| **Average**                 | **37.7** | **46.4** | **24.6** |

* Adapted from Gurven and Kaplan (2007).
Among contemporary small populations, higher mortality in females than in males appears to be considerably more common. Among the seven populations broken down by sex in Table 14.4, only two (Trio and Yanomama) indicate higher mortality in males than in females. Prehistoric data on sex differentials in mortality measured as an expectation of life at birth are not available because of the difficulties of sexing the skeletal remains of children (clear skeletal sex differences do not appear until puberty). As a result, published paleodemographic life tables typically pool the sexes among pre-adults. Paleodemographic estimates of life expectancies at adulthood (e.g., at age 15) or mean ages at death among adults have revealed several different patterns in the past: lower mortality for women (e.g., Bennett 1973; Nagaoka et al. 2006), lower mortality for men (e.g., Acasdi and Nemeskeri 1970; Angel 1972; Helgeman 1983; Šlaus 2000), and crossovers such that mortality was higher in one sex at early adult ages but lower at older ages (e.g., Owsley and Bass 1979; Högb erg et al. 1987; Wilson 2010). Whether expectation of life at birth was lower for females or males is not clear from these analyses, because the sex differential in mortality during childhood (which generally favors females) is not incorporated in these analyses. A recent analysis of the differences between medieval British men and women in the risks of mortality associated with skeletal pathologies has revealed that previous physiological stress increased the risk of death for men to a greater extent than was true for women, at least in medieval London (DeWitte 2010a). These results might indicate that the basic underlying biological differences that typically benefit females in modern populations might also have favored women in the past; however, it is not clear yet whether these results from the medieval population of London can be generalized to other past populations. What is clear is that the large sex differentials in favor of females that are characteristic of developed national populations today are not universal and may be a relatively recent development. Possible causes will be discussed further below.

### Variation in the Age Patterns of Mortality

The age patterns of mortality are thought to vary geographically among national populations and may vary between national populations and small populations. The age patterns are considered to vary independently of the level of mortality because the geographical distribution of age patterns of mortality has been remarkably consistent throughout the historical decline in mortality, at least in Europe (Coale et al. 1983).

Coale et al. (1983) identified four patterns of mortality that they named North, East, South, and West, because they appeared to be characteristic of the age patterns of mortality of northern, eastern, southern, and western Europe, respectively: The North pattern has low infant and post-50 mortality with high mortality at middle adult ages; the East pattern has high infant and post-50 mortality; the South pattern has high mortality below age 5 and high post-65 mortality; and the West pattern does not deviate systematically from the average pattern for all regions combined. Their sample consisted almost entirely of European populations and populations of European ancestry (e.g., in the United States, Canada, and Australia). Only a few truly non-European populations were included (several life tables from Japan and Taiwan). Other non-European populations were excluded because of the low quality of data available. Nevertheless, Coale and Demeny considered West as the “average”

### Table 14.4: Some Expectations of Life among Contemporary Small Populations by Sex

<table>
<thead>
<tr>
<th>Population</th>
<th>$e_0$</th>
<th>$e_x$</th>
<th>Economic Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yanomama(^a)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>17</td>
<td>22</td>
<td>h</td>
</tr>
<tr>
<td>Females</td>
<td>15</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Yanomama(^b)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>21</td>
<td>25</td>
<td>h</td>
</tr>
<tr>
<td>Females</td>
<td>20</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td>Rural China 1920</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males (North)</td>
<td>29</td>
<td>37</td>
<td>a</td>
</tr>
<tr>
<td>Females (North)</td>
<td>20</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td>Males (South)</td>
<td>22</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Females (South)</td>
<td>21</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>I'Kung</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sexes combined</td>
<td>35</td>
<td>42</td>
<td>g</td>
</tr>
<tr>
<td>Trio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>42</td>
<td>43</td>
<td>h</td>
</tr>
<tr>
<td>Females</td>
<td>51</td>
<td>44</td>
<td></td>
</tr>
<tr>
<td>New Guinea(^c)</td>
<td></td>
<td></td>
<td>Mixed</td>
</tr>
<tr>
<td>Males</td>
<td>53</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>51</td>
<td>44</td>
<td></td>
</tr>
<tr>
<td>Cocos Islands(^d)</td>
<td></td>
<td></td>
<td>Mixed</td>
</tr>
<tr>
<td>Males</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Semi Senoi</td>
<td></td>
<td></td>
<td>h</td>
</tr>
<tr>
<td>Males</td>
<td>31</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>32</td>
<td>30</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\) Adapted from Gage et al. (1989).
\(^b\) Estimate by Gage.
\(^c\) Estimate by Noel and Weiss (1975).
\(^d\) Indigenous population as a whole.
\(^e\) Malay peoples, migrated to Cocos in 1820s.

g. hunter-gatherer; h. horticultural; a. agricultural.
human pattern of mortality and the remaining patterns as deviations from that pattern. More recent studies have attempted to extend these analyses to include the age patterns of mortality of non-European populations (United Nations 1982; Gage 1990).

Gage (1990) reported that seven age patterns of mortality (labeled as "clusters" in Fig. 14.4) are required to encompass the range of variation found in a worldwide sample of contemporary national populations. Four of these are similar to the Coale et al. (1983) regional patterns (see Fig. 14.4). The Coale et al. West pattern is equivalent to the average of Gage's (1990) sample and similar to Gage's Cluster 2, whereas East is similar to Cluster 1, South to Cluster 5, and North to Cluster 4. Gage's study identified three new patterns—Clusters 3, 6, and 7—that deviate from the Coale et al. patterns and that are not prevalent in Europe (Table 14.5).

The age patterns from Gage's study also tend to vary geographically (see Table 14.5), although the correlation of age pattern with region is not perfect. Cluster 7 consists predominantly of North American, southern African, and southern South American populations. Cluster 6 is predominantly an Asian pattern of mortality.

Cluster 3 consists largely of central African populations. On the other hand, there is some evidence that life tables cluster by race/ethnicity/culture. For example, Asian life tables from South Africa (where racial/ethnic groups are reported separately) are included in Cluster 6, rather than in Cluster 7 with the life tables for the other races of southern Africa.

Most of the distinct differences among the clusters occur in adult mortality rather than infant or childhood mortality. These differences are contrasted in Figure 14.5. Three patterns have adult mortality that increase at the older ages relative to the
overall average pattern of mortality (see Fig. 14.5a), whereas four patterns have adult mortality that declines at older ages (see Fig. 14.5b). The three patterns with increasing mortality at the oldest ages are European; Cluster 2 is the only European pattern with mortality that declines at the oldest ages, though it is the least extreme of the four patterns shown in Figure 14.5b.

Cluster 3 is the most extreme of the age patterns, with mortality at the oldest ages declining very rapidly compared with the overall average age pattern of mortality (see Figs. 14.4 and 14.5b). It also has the lowest infant and post-60 mortality of any cluster and the highest childhood and young to mid-adult mortality. This age pattern is generally less U-shaped than other age patterns of mortality. Because of the poor quality of demographic data from Africa, this pattern may simply represent errors in the underlying life tables (e.g., underenumeration of population and/or deaths, misstatement of age). However, the African pattern may represent mortality in Africa more closely than do the European patterns of mortality, which are based on “better” data but from a radically different physical, biological, and cultural environment. Interestingly, comparisons of mortality between African-American (black) and European-American (white) populations in the United States show similar differences in the age patterns of mortality. The black pattern is less U-shaped and shows lower mortality among older individuals than the white pattern again suggesting the existence of race/ethnic/cultural variation in mortality patterns. This phenomenon is often referred to as the black-white mortality crossover (Gage 1989). Its causes are currently attributed to bad data (e.g., age misreporting) and/or the effects of heterogeneity (e.g., higher black mortality at the younger ages selects on frailty so that at the older ages, the surviving blacks are more robust than the surviving whites of the same age). The causes and consequences of ethnic diversity in health remains a serious issue (Anderson et al. 2004).

Very little empirical evidence—ethnographic or archaeological—exists concerning the age patterns of mortality characteristic of small populations. This paucity of data occurs because of the difficulties in collecting sufficient data and the potential for error in the data collected. Furthermore, studies of small populations often depend on model life tables, usually the West (average) pattern of mortality, to estimate a life table, and/or the ages of living individuals (e.g., Howell 1979; Gage et al. 1984b). Consequently, western European mortality patterns are often imposed on the data, and independent evidence of the age pattern of mortality is not available.

O'Connor (1995) developed a model life table from prehistoric life tables, based on ages using traditional biased techniques and using the methods of model life-table estimation developed by Gage and Dyke (Gage 1988; Gage and Dyke 1988; Dyke et al. 1993). O'Connor's analysis includes 12 life tables based on age at death distributions. Each of these life tables was estimated assuming stationary conditions. As a result, any deviation from the stationary state will incorporate error into the individual life tables (see Box 14.3). However, if a series of these life tables is combined, the average growth rate should tend toward zero, the stationary condition. This expectation is based on the assumption that, before the industrial revolution, mean population growth rates were very small. In any event, the resulting paleodemographic model life table has a life expectancy of about 20 years at birth.

This age pattern of mortality is compared with Gage's (1990) average national pattern at a life expectancy of 20 years in Figure 14.6. The paleodemographic age pattern indicates very low infant mortality but extraordinarily high adult mortality. A similar age pattern was observed in two medieval European samples in a study that used a new age estimation method and a hazards model approach, rather than the traditional life table (DeWitte 2010b). These results might be indicative of underrepresentation of infant deaths (resulting from biases in burial, preservation, and/or excavation), misestimation of age at death particularly at the older ages because of the use of biased methods, or both.

The new unbiased methods of adult aging may make a significant difference in our understanding of prehistoric mortality patterns among adults. Konigsberg and Herrmann (2006) reported the impact of Bayesian methods on three adult skeletal populations. In two of these populations (Averbuch and Loisy-en-Brie), estimates of adult mortality are significantly reduced by the new techniques and more closely resembles adult age patterns of the mortality characteristic of contemporary small populations and national populations, such as Coale et al. (1983) West mortality. However, in the third example (Indian Knoll), adult mortality remained more or less the same, at relatively high levels Konigsberg and Herrmann (2006) conclude that this latter life table is defective. Kreger (2010) and Strott (2006) reported similar results. If these examples are representative of the effects of the new age estimation techniques, then the very high adult mortality traditionally reported for paleodemographic life tables may be spurious. Additional applications of the new methods will be necessary to determine how the new aging methods influence our understanding of prehistoric mortality.

Gurven and Kaplan (2007), also using the methods of model life table estimation developed by Gage and Dyke (Gage 1988; Gage and Dyke 1988; Dyke et al. 1993), have argued on the basis of the data presented in Table 14.3 that the high adult
mortality of observed paleodemographic life tables is spurious. In particular, they pointed out that the expectation of life at age 45 among contemporary small populations is generally 20 years or more, indicating a long postreproductive life span, as expected among human populations (Table 14.3). The expectation of life at age 45 is much shorter among traditional paleodemographic life tables, approximately 5 years (O’Connor 1995). Gurven and Kaplan’s (2007) argument is based on the assumption that contemporary small populations are a good ethnographic analogy for prehistoric populations. However, this may or may not be the case given that contemporary small populations coexist with large industrial human populations, whereas prehistoric populations did not. As described below, the disease environment of prehistoric populations was likely to have been quite different from that of living small populations.

The issue of adult mortality is of interest because it has implications for the evolution of human life histories (Chapter 11), specifically the evolution of menopause. For example, if the very high adult mortalities of traditional paleodemographic life tables are correct, few individuals survive to postmenopausal ages and menopause would have been rare in the past. This is the case for other organisms, where reproductive senescence is closely associated with the end of life. If survival past reproductive ages were rare in human history, the emergence of menopause might be very recent and an explanation of why a postreproductive life span evolved would not be necessary. Menopause might simply be an artifact of the decline in mortality in recent times. On the other hand, if life span exceeded menopause in prehistoric times, as suggested by some reestimates of prehistoric mortality using the new unbiased age estimators, then evolutionary explanations of human postreproductive life span are needed (see Chapter 11).

Estimated prehistoric infant mortality, on the other hand, appears to be low compared with the age patterns of national populations (Fig. 14.6). It is often assumed that this is the result of underenumeration of infant deaths in the archeological record because of taphonomic processes, methodological factors, or burial practices; small, fragile infant skeletal remains are more likely to be disintegrated over time or less likely to be detected by excavators compared with adult skeletal remains in certain burial contexts and soil conditions, and in some cultures, infants are buried away from the primary cemeteries and thus not recovered (Lewis 2007). Konigsberg and Frankenberg’s (2002) reestimate of Loisy-en-Brie mortality using unbiased aging methods also introduced several extra infant deaths to account for underenumeration. The result is an age pattern of mortality very similar to that observed in national populations. The resulting life table has an expectation of life at birth of 25 years, with 30% mortality in the first year of life and 51% mortality by age 15 years. The uncorrected archaeological data by O’Connor (1995) gave an estimate of infant mortality of 18.6% and 47% mortality by age 15, lower infant mortality, and higher childhood mortality with Konigsberg and Frankenberg’s reestimate of Loisy-en-Brie. The results from these studies raise questions about whether infant mortality is generally underestimated and therefore needs to be corrected.

If we accept the new unbiased adult estimates, and accept the underenumeration of infant deaths, then prehistoric age patterns of mortality may have resembled those observed today. But if that is correct, then the level of mortality among prehistoric populations must have been higher than that observed in historic or contemporary populations, small or national (as argued above). On the other hand, if we accept the new unbiased adult estimates, but do not correct for presumed underenumeration of infant deaths, then prehistoric age patterns would differ significantly from the national average. Qualitatively (if not quantitatively), they would resemble the age patterns of Cluster 3 (see above; Gage 1990). If this was the case, then the expectations of life among prehistoric populations will be closer to the level of mortality observed in contemporary high mortality populations. Clearly, additional research is needed to determine which set of the underlying assumptions to accept. Either way the mortality of contemporary populations does not appear to be an adequate model of prehistoric mortality.

Model life tables are particularly useful and important as an aid for estimating the level of mortality in situations where the demographic data are incomplete or defective. Thus, a model life table can be used to estimate the overall level of mortality from estimates of childhood mortality, which may be easier to obtain. This task could be as simple as selecting the Coale et al. (1983) West model life table with the same childhood mortality as the observed population. Konigsberg and Frankenberg (2002) basically use this approach when they added extra infant deaths to the “Loisy-en-Brie” sample until it matched an average national mortality pattern (Coale et al. 1983; West level 3 females). Variations of this procedure have been used to estimate expectations of life for several small populations (e.g., Howell 1979; Gage et al. 1984b). The assumption is that an appropriate model life table exists and is selected for this purpose; however, in practice, selecting the appropriate model can be problematic. For example, is the West age pattern an appropriate model life table to use in central Africa? Probably not, unless the extreme age patterns of mortality of central Africa are simply and completely a result of poor data and not the effects of different environments. Similarly, is the West age pattern appropriate for use in prehistoric populations, as assumed by Konigsberg and Frankenberg (2002)? Perhaps not; as discussed below, the disease environment is likely to have been radically different for prehistoric populations compared with that for contemporary populations, either national or small.

To resolve these issues, a greater understanding of the causes of variation in human age patterns of mortality is necessary. This area of research has largely been ignored by national demographers. Consequently, little is known about the causes—genetic, environmental, cultural, and behavioral—that influence the age patterns of mortality. But it is likely to be of particular interest to human biologists, given their focus on understanding human biocultural variation (see, e.g., Gage and O’Connor 1994). If we knew what factors influenced the age patterns of mortality and how they did so, we would be in a better position to choose between the alternatives discussed above—(1) prehistoric mortality was very high (clearly higher than observed in any contemporary populations), but the prehistoric age pattern resembled an average human age pattern of mortality, or (2) prehistoric mortality was high (comparable to or higher than observed in any contemporary populations) but the prehistoric age pattern did not resemble an average human age pattern of mortality. In any event, there is a trend in the data reported above of increasing infant mortality and decreasing childhood mortality from the archeological data (corrected for infant underenumeration) to the contemporary hunter-gatherer data, to the contemporary horticultural data, to the national mortality patterns. The age pattern of mortality has become more steeply U-shaped over time; that is, mortality
has become more concentrated, at the youngest ages, and at least in the national life tables, at the older ages as well.

**Proximate and Ultimate Causes of the Variation in Mortality**

In this section, we consider the factors that may account for variation in the level and age patterns of mortality. The section is divided into four parts. In the first section, we consider the variation in the distribution of causes of death from a theoretical and an empirical perspective. Variation in causes of death is clearly a proximate determinant of the human variation in mortality. Proximate causes, however, only raise questions concerning their ultimate causes, so in the second section, we present some potential ultimate determinants of the variation in level of mortality. In the third section, we briefly consider the proximate and ultimate determinants of the variation in age patterns of mortality. Finally, in the fourth section, we look specifically at determinants of early childhood mortality since young children are especially vulnerable to environmental effects on the risk of death, and early childhood mortality is one of the most important components of the human age pattern of mortality in differentiating modern national populations from small preindustrial ones.

Given that **infectious disease** was clearly an important cause of death in past human populations and still is in the modern-day developing world, many researchers have examined variation in the types of diseases present and their **prevalence** in such populations as a proximate cause of interpopulation variation in mortality. Fenner (1970) described hypothetical changes in human infectious disease ecology throughout human prehistory and history on the basis of several simple **epidemiological** principles:

- the characteristics of **host-pathogen interaction** (the mode of transmission of the pathogen and the nature of the human host’s **immune response**);
- the frequency of contact between **hosts** and **pathogens**, as influenced by the host’s population size, density, and patterns of movement; and
- the lifestyle of the host population (nomadic, sedentary, urban).

Given the rise in population associated with the agricultural and the industrial revolutions, the increasingly sedentary lifestyle as a result of the adoption of agriculture, and the acceleration of urbanism with industrial development, Fenner (1970) argued that the types and prevalence of diseases and hence causes of death characteristic of human populations must have changed in predictable ways (Table 14.6).

<table>
<thead>
<tr>
<th>Years BP</th>
<th>Economic Type</th>
<th>Size of Human Communities</th>
<th>Major Infectious Causes of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,000,000</td>
<td>Hunter-gatherer</td>
<td>Scattered nomadic bands &lt;100, mean size about 25</td>
<td>Zoonose: scrub typhus, encephalitis, Rocky Mountain spotted fever, plague, Helminths: trichinosis, trematode, filariasis, and so on</td>
</tr>
<tr>
<td>10,000</td>
<td>Horticulture</td>
<td>Settled villages &lt;300</td>
<td>Zoonose: Helminths: trichinosis+, trematode+, and so on</td>
</tr>
<tr>
<td>5500</td>
<td>Intensive agriculture</td>
<td>A few cities &gt;100,000, mostly villages &lt;300</td>
<td>Bacterial: tuberculosis+, malaria+, dysentery, and diarrhea, Viral: Zoonose: Helminths: sistrosomiasis+</td>
</tr>
<tr>
<td>250</td>
<td>Steam power</td>
<td>Cities &gt;5000, villages 1000</td>
<td>Bacterial: tuberculosis++, dysentery, and diarrhea, Viral: smallpox, measles, rubella, influenza, Zoonose: Helminths: Bacterial: tuberculosis++, dysentery, and diarrhea, Viral: smallpox++, measles++, rubella++, influenza++, colds</td>
</tr>
<tr>
<td>130</td>
<td>Sanitation</td>
<td></td>
<td>Decline of most infectious diseases as causes of death, except polio+, influenza+, and colds+</td>
</tr>
</tbody>
</table>

* Indicates an increase in the cause of death over previous periods.
* Adapted from Fenner (1970).
When introduced into a small or low-density population, these diseases sweep through the population, infecting the susceptible individuals. However, after all the susceptible individuals have been infected, the diseases cannot find new hosts and go extinct. Consequently, Fenner (1970) assumed that many of the common childhood diseases of the historic period may not have been important causes of death in low-density populations such as hunter-gatherers or shifting cultivators (Table 14.6).

Some of these diseases may have evolved as human pathogens only relatively recently. Measles is perhaps the best example, because it cannot be endemic in isolated populations of less than half a million inhabitants. Consequently, these diseases could not have originated as exclusively human pathogens before the advent of large urban populations, which accompanied the origins of intensive agriculture (Fenner 1970). Furthermore, Fenner (1970) argued that an increasingly sedentary lifestyle may have also changed the disease environment, because it brought humans in more direct contact with human waste products and parasites. Thus, the infectious diseases spread by fecal-oral transmission should have increased with the development of sedentary lifestyles. The emergence of urbanism as a result of agriculture and its acceleration with industrialization brought many people into face-to-face contact, hence providing the large, dense populations necessary to support many of the infectious diseases that characterize human populations today (e.g., measles, mumps). "Emerging" diseases, those that are increasing in frequency after being introduced into a new host population, are not a recent development in human affairs; the appearance of human immunodeficiency virus (HIV) and Ebola in modern times is not unprecedented; the Black Death, for example, was an emerging disease of 14th century Europe. Fenner (1970) also argued that many—even most—of these diseases appear to have been originally introduced into human populations by contact with other animals. This may have accelerated with the domestication of animals, although it was presumably common much earlier as a result of hunting.

Fenner’s (1970) theory suggests two hypotheses concerning the variation in mortality: that infectious disease mortality should increase and that infant infectious disease mortality should become more important as populations become denser, increasingly sedentary, and more urbanized. The first hypothesis is based on the assumption that the increase in the number of infectious diseases should cause infectious disease mortality to increase. The second hypothesis is based on the assumption that individuals are likely to be infected at an earlier age if the disease is endemic. This has been an influential theory in human biology. It is an important argument for the idea that health deteriorated with the advent of agriculture (Cohen and Armelagos 1984; Cohen 1989). It is attractive because of its simplicity and because the underlying assumptions seem sensible to most people. Nevertheless, major flaws remain. In particular, it assumes that the infectious diseases do not evolve and do not interact competitively with each other. The theory is based on the characteristics of the "known diseases" of modern human populations. The virulence characteristics of the "known diseases" may have been quite different in the prehistoric period. Recent molecular genetic analysis of known human disease pathogens indicates that they (as a group) may have undergone an intense genetic transformation with the transition to agriculture (Mira et al. 2006). Furthermore, the molecular data indicate that some human diseases are older than predicted under Fenner’s theory and that some human diseases thought to be acquired from domesticated animals, that is, after the advent of agriculture, were actually diseases that domesticated animals acquired from humans (Armelagos and Harper 2005). Furthermore, it is possible that some or even many pathogens adapted to low-density prehistoric human populations could not compete with the pathogens adapted to high-density modern human populations and went extinct. As a result, the known diseases of contemporary human populations may not be good models for the disease environment of prehistoric humans. Lastly, the theory potentially underestimates the importance of zoonoses in determining health and mortality in prehistoric human populations. According to Taylor et al. (2001), of the more than 1400 infectious organisms that are pathogenic to humans, over 60% are zoonoses, and recent research has revealed that the majority of zoonoses that infect humans and nonhuman primates (especially those carried by arthropod vectors like mosquitoes, lice, or fleas) are characterized by low host specificity; that is, they are capable of infecting multiple host species (e.g., Taylor et al. 2001; Pedersen et al. 2005). The majority of recent emerging diseases are associated with zoonotic pathogens that are maintained in multiple nonhuman host species (Taylor et al. 2001). This suggests that prehistoric human populations who engaged in hunting and gathering and thus came into contact with other animals were exposed to a variety of infectious pathogens. Infectious diseases might therefore have been an important determinant of mortality in the past long before the large, dense settlements associated with agriculture existed. The ecology of infectious disease is currently an emerging area of research in biology, and as our understanding of the ecology and evolution of infectious diseases improves, so too should our understanding of human disease in the past.

In any event, the empirical data do not support Fenner’s first hypothesis. The prehistoric data are controversial, but as far as can be determined, mortality changed little over the course of prehistory, even with the introduction of agriculture (Table 14.2). Furthermore, the recent expansion of populations during the historic period has clearly been associated with a decline in mortality, not an increase. Finally, those causes of death that declined most during the historic period are precisely those predicted to increase: the infectious diseases.

Empirical studies have demonstrated that during the historic period, mortality declined and there were changes in the predominant causes of death. In particular, the structure of causes of death shifted from predominantly infectious diseases (e.g., measles, respiratory tuberculosis) to predominantly chronic degenerative diseases (e.g., cardiovascular disease, cancers), a process that has been called the epidemiological transition (Omran 1977; McKeown 2009). An accurate empirical understanding of the epidemiological transition is complicated by competition between causes of death and by secular changes in the reporting and reliability of diagnosis of the various causes of death. Clearly, there has been a change from a predominance of infectious causes of death to a predominance of degenerative causes. It is often assumed that this shift necessarily indicates an increase in the risk of the degenerative diseases as a result of maladaptation to modern lifestyles (largely characterized by overnutrition, reduced exercise, and increased psychosocial stress) (Trowell and Burkitt 1981; Rose 1982; McKeown 1983; Frisancho 1993; Eaton and Eaton 1999; Elton and O’Higgins 2008; Pollard 2008). However, the epidemiological transition could simply be the result of a decline in the infectious causes of death. Everyone
must die; consequently, if the risk associated with infectious diseases declines, then
the proportion of deaths associated with degenerative causes must increase, even if
the risk of the degenerative causes remains constant. Furthermore, observed
“increases” in the risk of the degenerative disease may simply result from improve-
ments over time in diagnosis; that is, deaths caused by degenerative diseases, which
in the past may have been misclassified as “ill-defined” causes of death (e.g., old
age) are increasingly classified into the appropriate degenerative category (e.g.,
cardiovascular disease, cancer, type 2 diabetes).

As a result of these problems, the exact nature of the epidemiological transition
is still a matter of debate. Studies of the epidemiological transition that attempt
to control for improvements in diagnosis tend to conclude that the risks of both
infectious and degenerative causes of death declined (Preston 1976; Gage 1994,
2005). These results are consistent with trends in these same causes of death
that have been observed over the past several decades in developed countries,
during which the reporting of degenerative causes of death is considered to be
accurate (Stallones 1980; Greenberg 1983; US Bureau of the Census 1988;
American Cancer Society 2006). Thus, the transition from predominantly infectious
diseases to predominantly degenerative diseases appears to be the result of the
faster decline of the infectious than the degenerative causes of death. Perhaps mod-
ernization is not as bad for health as some believe. Furthermore, evidence indicates
that the decline in infectious disease mortality was greater for males than for
females, whereas the decline in degenerative disease mortality was greater for
females than for males. The generally higher life expectancy of females in the
modern world may well be a consequence of the greater decline in degenerative
disease mortality among females compared with males (Lopez and Ruzicka 1983;
Gage 1994).

Although the overall combined risk of degenerative causes of death has declined,
one exception is worthy of note. The risk of cancer has clearly increased, and the
increase has been greater in men than in women (Preston 1976). This phenomenon
is largely responsible for the slower decline in degenerative mortality in males than
in females. The difference mostly represents lung cancer and is largely a function of
changes in the frequency and pattern (especially gender differences) in cigarette
smoking. However, the declines in the other degenerative causes of death have more
than compensated for the increased incidence of lung cancer so that the risk of total
degenerative deaths has declined in both sexes (Gage 1994, 2005).

These trends in the sex differentials in the national populations might explain the
unusual sex differences in mortality in favor of males in the contemporary small
populations shown in Table 14.3 and noted earlier. If infectious disease mortality is
relatively low in small populations compared with dense populations, then the sex
differentials might be expected to favor males. However, this notion is contrary to the
anthropological dogma that the degenerative diseases are absent in traditional
small populations.

In any event, infectious diseases clearly declined with the rise of population
associated with modernization, at least until recently. This trend is clearly counter
to epidemiological predictions suggested by Fenner (1970) and requires additional
examination of higher-order, that is, ultimate, causes of the decline in mortality. In
particular, we need to ask what factors Fenner left out that might explain the decline
in infectious disease mortality in modern times.

| TABLE 14.7 Declines in Cause of Death in England and Wales, 1850–1971 |
|------------------|-----|-----|-----|-----|
| Cause of Death   | 1850 AD | 1971 AD | Percent Reduction | Percent Reduction prior to 1901 |
| Infectious causes|      |      |     |     |
| Airborne diseases| 7259 | 619  | 40  | 32  |
| Water- and food-borne diseases | 3562 | 35  | 21  | 46  |
| Other conditions | 2144 | 60  | 13  | 35  |
| Total infectious | 12,965 | 714  | 74  | 37  |
| Noninfectious causes | 8891 | 4070 | 26  | 10  |
| Grand total      | 21,856 | 5384 | 100 | 30  |

a Adapted from McKeown (1976).
b Death rates per million population.

Ultimate Determinants of the Variation in Mortality

The classic study of the historical decline in mortality during the 19th and 20th
centuries (McKeown 1976) examined four potential causes, all of them focused on
infectious diseases: the evolution of host–pathogen interactions (coevolution),
the development of modern medicine, the introduction of effective sanitary systems,
and improvements in nutrition. None of these factors appears in Fenner's theory.
McKeown based his identification of the ultimate cause or causes of the mortality
decline on the changes in the empirical structure of cause of death associated with
the decline in mortality in England and Wales from 1860 to 1971 (Table 14.7).

Host–parasite interactions refer to the coevolutionary process by which natural
selection tends to influence the virulence of the pathogen and increases the resis-
tance of the host. McKeown (1976) eliminated this cause of the decline on the basis
that there was insufficient time, less than 200 years, for significant evolution of the
human population and because of the lack of any evidence for the amelioration of
virulence in the pathogens. Scarlet fever is noted as an exception because evidence
shows cyclical changes in virulence of this pathogen; however, the decline in mortality
requires the simultaneous decline in virulence of many pathogens, for which,
McKeown argued there is no compelling evidence. Recent research on the ecology
and evolution of infectious diseases introduced above does not contradict these
findings. However, it is clear that the evolution of virulence is much more complex
than previously thought and can lead to higher rather than lower virulence (Bull
The role of evolution with respect to human infectious disease is a part of McKeown’s
argument and Fenner’s theory that needs to be reconsidered.

McKeown (1976) also argued that sanitation was not responsible for most of the
historic decline in mortality. He defined sanitation as the introduction of effective
measures to eliminate contact between host and pathogen populations. Only 21% of
the overall decline in mortality is attributed to causes of death that can easily be
controlled using sanitary measures, that is, water- and food-borne diseases (Table
14.7). McKeown maintained that sanitary measures are unlikely to have affected
the airborne diseases because of the lack of effective methods of isolation.

Similarly, McKeown (1976) claimed that modern medicine was not responsible
for the decline in mortality. He defined modern medicine as the development of
“effective treatments,” usually drug therapies. However, the decline in mortality and the epidemiological transition generally precede the development of what are considered effective treatments today. For example, the initial decline of respiratory tuberculosis (the largest single component of the decline in mortality in England and Wales) preceded the development of streptomycin, the first effective treatment of respiratory tuberculosis, by at least 100 years (see Fig. 14.7).

McKeown (1976) concluded that the decline in mortality was associated with improvements in nutrition. He based this conclusion on Sherlock Holmes’ principle that if all other possible explanations can be eliminated, the one remaining explanation must be correct. Nutritional status clearly affects immune function, and there is some evidence that nutrition improved during the 19th and 20th centuries (Overton 1996; Fogel 2004). In England, for example, there was rapid growth in agricultural productivity in the 19th century as a result of many factors including improvements in land quality (e.g., draining of marshes), the replacement of many lower-yield crops with higher-yield crops, and mechanization of farming: agricultural output in 1850 was approximately three times higher than it had been in 1700 (Overton 1996). In France, increases in food supplies from 1700 through the 20th century were associated with declining mortality rates (Fogel 2004). A historical (secular) increase in stature (see Chapter 12), which at least partly reflects the concurrent increases in food availability, has accompanied the decline in mortality (Floud et al. 1990; Fogel 2004). Additionally, nutritional availability based on national food balance sheet estimates is closely correlated with the level of mortality (Gage and O’Connor 1994). However, we cannot exclude the possibility that a decline in infectious diseases was a cause of, rather than the result of, improvements in nutritional status, given the synergy between infection and nutrition described below.

It is not clear that McKeown’s (1976) list of causes is complete (Michaud et al. 2001; Ezzati et al. 2004). For example, advances in medical knowledge (e.g., the germ theory of disease [Chapter 9]), coupled with education, may have been responsible for the improvements in primary care and the decline in associated infectious disease mortality (Preston and Haines 1991). Thus, the cause of the decline in mortality during the industrial era remains a matter of debate. If it is not known why mortality declined, it is also not known how to keep new infectious diseases from emerging and old ones from reemerging.

Cause of Death and the Age Patterns of Mortality

The factors that affect human variation in the age patterns of mortality have received less attention than studies of the level of mortality. Preston (1976) showed that variation in the age patterns of mortality are closely correlated with the geographical distribution of cause of death. For example, the deviations of the North pattern of mortality from the West pattern are thought to be the result of the relatively high rates of respiratory tuberculosis found in northern European countries, as tuberculosis is an important cause of variation in mortality during the middle adult ages. Gage and O’Connor (1994) showed that variation in nutritional availability could be responsible for the observed variation in the age patterns of mortality (Gage 1990). In particular, abnormally low-calorie and low-protein diets are associated with the African pattern of mortality. Whether nutrition is a causal or simply an associated phenomenon is not evident from these analyses. Nevertheless, the results suggest that the geographical distribution of causes of death and hence the age patterns of mortality within Europe, for example, might be partially the result of differences in agricultural productivity or even national cuisines.

Changes in population density and endemicity of infectious diseases might be responsible for some changes in the age patterns of mortality, that is, the second prediction suggested by Fenner’s (1970) theory of disease and society. In particular, infant mortality appears to have increased and childhood mortality decreased among small populations when comparing the archeological data with the contemporary hunter–gatherer data and the contemporary horticultural data, as noted earlier. A similar trend occurs with the historical decline in mortality and across contemporary national populations when ranked by level of mortality (Gage 1993, 1994). This trend could be the result of contracting these diseases at earlier ages, as population density increases and more diseases become endemic. If this view is correct, then model life tables based on historical European mortality patterns may not be appropriate for use in low-density prehistoric populations.

Infant and Early Childhood Death

The ultimate causes of the high mortality during infancy (the first year of life) and early childhood (up to age 5) that distinguishes anthropological populations, including prehistoric ones, from modern, industrialized nations are of special interest to human biologists. The difference between past and present is graphically illustrated by childhood mortality rates estimated before, during, and after the 19th- and 20th-century epidemiological transition in England and Wales (Fig. 14.8). Mortality during the first year of life, for example, declined by about 90% during the transition
from the preindustrial to the modern age. Today, infant mortality is almost nonexistent in industrialized nations, whereas in the past, it was one of the largest components of age-specific mortality across the life span (Gage 1993; Bideau et al. 1997).

What were the primary factors contributing to high childhood mortality in human communities of the past and of living anthropological populations? This question is difficult to answer from skeletal data because we only observe children who died in childhood, so we cannot determine what differentiated them from children who survived to die at later ages. In addition, as noted above, the skeletons of a very young child from archaeological sites are often not well preserved. As a first step in answering this question, however, we note that undernutrition is a major predictor of the risk of death among young children in much of the modern rural developing world, which may or may not provide good models of genuinely preindustrial life. In Table 14.8, for example, children under the age of 10 in rural Bangladesh who were moderately to severely undernourished (according to the local distribution of one particular anthropometric index) were also at substantially elevated relative risk of death (R) over an 18-month follow-up period compared with their mildly undernourished counterparts. Shockingly, the juvenile mortality rates observed in most of these populations were actually greater than neonatal mortality in the United States (universally neonatal mortality, or mortality during the first month of life, is the highest component of childhood mortality within a population). Many more examples could have been adduced (for a recent summary of the literature, see Christian 2008), but the general pattern is clear enough. Undernutrition is without a doubt an important influence on the risk of early childhood death in a variety of developing-world farming communities. From all available evidence, the same is almost certain to have been true in the preindustrial past, at least among agriculturalists (Aykroyd 1971; Bengtsson 1999; Fogel 2004).

To understand the significance of inadequate diet for child mortality in the preindustrial world, we need to examine the mechanisms linking the two in more detail.
The first step is to ascertain what preindustrial people actually died of, a question that has been investigated in the field by several anthropologists and other researchers. It is necessary to point out that retrospective diagnosis of the primary cause of any particular death that occurs during field research, as it is based on “verbal autopsy” (a postmortem interview with witnesses), is problematic (Snow and Marsh 1992). This is so principal because the symptoms elicited in interviews are often rather vague and nonspecific (Kalter et al. 1990; Marsh et al. 2003). But if we are content to settle for an extremely crude classification of causes, a strong and consistent pattern emerges: contemporary small populations and rural Third World people mostly die of infectious diseases, which collectively lead other categories of causes by a large margin (Table 14.9). Even in modern developing-world communities with some access to modern health care and with rapidly falling mortality rates, infectious diseases remain the most important class of serious clinical problems in young children (Table 14.10). Historically, the dropping away of deaths by infectious

### TABLE 14.9 Causes of Death (as Percentage of Recorded Deaths at All Ages) in Selected Preindustrial and Developing-World Societies for Three Broad Categories of Causes

<table>
<thead>
<tr>
<th>Causes</th>
<th>'Kung</th>
<th>Aka</th>
<th>Yanomama</th>
<th>Gainj</th>
<th>Developing Countries*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>F</td>
<td>M + F</td>
</tr>
<tr>
<td>Infectious</td>
<td>77</td>
<td>82</td>
<td>91</td>
<td>95</td>
<td>76</td>
</tr>
<tr>
<td></td>
<td>77</td>
<td>82</td>
<td>91</td>
<td>95</td>
<td>76</td>
</tr>
<tr>
<td>Degenerative</td>
<td>8</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Violent and accidental</td>
<td>15</td>
<td>8</td>
<td>9</td>
<td>5</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>8</td>
<td>9</td>
<td>5</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>No. of deaths</td>
<td>342</td>
<td>669</td>
<td>111</td>
<td></td>
<td>44</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10,000+</td>
</tr>
</tbody>
</table>

* Based on data from eight national populations with life expectancy at birth <40 years, similar to the life-table estimates for the other four populations.

From Preston et al. (1972); Howell (1979, p. 69); Wood (1980, p. 122); Melancon (1982, table 3.1); and Hewlett et al. (1986, p. 54).

*The 'Kung (Kalabari Desert) and Aka (Congo Basin) were predominantly hunter-gatherers at the time of study. The Yanomama (Orinoco Basin) and Gainj (Highland New Guinea) were shifting cultivators.

### TABLE 14.10 Frequency of Pediatric Hospital Admissions by Cause in a Birth Cohort of 452 Infants (<1 Year Old at Admission), Velore, South India, 2002

<table>
<thead>
<tr>
<th>Cause of Admission</th>
<th>Frequency of Admission (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory infection</td>
<td>51</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>21</td>
</tr>
<tr>
<td>Other infections</td>
<td>8</td>
</tr>
<tr>
<td>Neonatal conditions</td>
<td>12</td>
</tr>
<tr>
<td>Genetic anomalies</td>
<td>3</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>2</td>
</tr>
<tr>
<td>Others</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
<tr>
<td>Number of admissions</td>
<td>106</td>
</tr>
</tbody>
</table>

*All infections 80%*

From Gladstone et al. (2008).

![Figure 14.9](image_url)  
**Figure 14.9** Annual rates of infectious disease mortality, United States 1900–1996. (Top) Deaths at all ages. (Bottom) Dark line, deaths among children less than 5 years old; light line, infectious disease deaths as a percentage of all deaths among young children. From Armstrong et al. (1999:63–64).

...disease accounted for almost all the decline in childhood mortality during the modern epidemiological transition in the West (Fig. 14.9).

But if most deaths of children in the preindustrial past were the direct result of infection, does that mean that undernutrition was unimportant as a cause of death? Not at all, though it may indicate that undernutrition was mostly a contributing cause of death rather than an immediate one. But it was a profoundly important contributing cause. One of the most thoroughly investigated functional effects of undernutrition, in both humans and lab animals, is reduced immunocompetence or the ability of the immune system to defend its owner from contracting or succumbing to infectious disease (for reviews, see Keusch 1998; Woodward 1998; Gershwin et al. 2004). Table 14.11 summarizes elements of the immune system known to be...
TABLE 14.11 Components of the Immune System Known to Be Impaired by Protein-Energy Undernutrition

<table>
<thead>
<tr>
<th>Component</th>
<th>Percent of Time Ill with Diarrhea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mucus production</td>
<td></td>
</tr>
<tr>
<td>Integrity of the intestinal mucosa</td>
<td></td>
</tr>
<tr>
<td>Weight of thymus, spleen, tonsils, and lymph nodes (e.g., nutritional thymectomy)</td>
<td></td>
</tr>
<tr>
<td>Number of T-lymphocytes in the bloodstream</td>
<td></td>
</tr>
<tr>
<td>Number of CD4+ and CD8+ cells in the bloodstream</td>
<td></td>
</tr>
<tr>
<td>Proliferation of T-lymphocytes</td>
<td></td>
</tr>
<tr>
<td>Production of Th1-type cytokines (IL-2, IFN-γ)</td>
<td></td>
</tr>
<tr>
<td>Delayed-type hypersensitivity</td>
<td></td>
</tr>
<tr>
<td>Concentration of secretory IgA in breastmilk, the intestinal mucosa, tears, saliva, and so on</td>
<td></td>
</tr>
<tr>
<td>Concentration of C3 (from complement system) in the bloodstream</td>
<td></td>
</tr>
<tr>
<td>Natural killer cell activity</td>
<td></td>
</tr>
<tr>
<td>Respiratory bursts in neutrophils</td>
<td></td>
</tr>
<tr>
<td>Bacterial killing by neutrophils</td>
<td></td>
</tr>
<tr>
<td>Production of the cytokines TNF, IL-1, and IL-6 by monocytes/macrophages</td>
<td></td>
</tr>
<tr>
<td>Concentration of acute-phase proteins in the bloodstream</td>
<td></td>
</tr>
</tbody>
</table>

Modified from Jackson and Calder (2004, p. 79).

Cytokines (inter- and paracellular messengers): IL, interleukin; IFN, interferon; TNF, tumor necrosis factor; Ig, immunoglobulin (antibody).

TABLE 14.12 The Relationship between Nutritional Status and the Incidence and Duration of Diarheal Disease in 343 Children Ages 6-32 Months in Rural Nigeria

<table>
<thead>
<tr>
<th>Nutritional Status</th>
<th>Number of Children</th>
<th>Diarrhea Attack Rate*</th>
<th>Percent of Time Ill with Diarrhea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight for age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥75%</td>
<td>220</td>
<td>1.25</td>
<td>8.5</td>
</tr>
<tr>
<td>&lt;75%</td>
<td>123</td>
<td>1.52</td>
<td>11.3**</td>
</tr>
<tr>
<td>Height for age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥90%</td>
<td>245</td>
<td>1.37</td>
<td>7.9</td>
</tr>
<tr>
<td>&lt;90%</td>
<td>98</td>
<td>1.45</td>
<td>10.8**</td>
</tr>
<tr>
<td>Weight for height</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥88%</td>
<td>302</td>
<td>1.29</td>
<td>7.6</td>
</tr>
<tr>
<td>&lt;88%</td>
<td>41</td>
<td>1.90*</td>
<td>13.4***</td>
</tr>
</tbody>
</table>

From Tomkins (1981).
* Cases per child in 3 months.
** P < 0.05.
*** P < 0.01.

TABLE 14.13 Recorded Causes of Death (by Percentage) during the Irish Famine of 1846-1849

<table>
<thead>
<tr>
<th>County</th>
<th>Counties</th>
</tr>
</thead>
<tbody>
<tr>
<td>County</td>
<td>Mayo</td>
</tr>
<tr>
<td>Dysentery/diarrhea</td>
<td>28.2</td>
</tr>
<tr>
<td>Unspecified fever</td>
<td>27.5</td>
</tr>
<tr>
<td>Starvation</td>
<td>9.1</td>
</tr>
<tr>
<td>Consumption (tuberculosis)</td>
<td>4.6</td>
</tr>
<tr>
<td>Cholera</td>
<td>2.2</td>
</tr>
<tr>
<td>Dropsy (nutritional edema)</td>
<td>2.0</td>
</tr>
<tr>
<td>Marasmus (severe protein-energy malnutrition)</td>
<td>1.6</td>
</tr>
<tr>
<td>Other (including unknown)</td>
<td>24.8</td>
</tr>
<tr>
<td>Total</td>
<td>100.0</td>
</tr>
</tbody>
</table>


Environment can exert an influence on early childhood mortality, but only if it has some effect on at least one of a small number of more proximate factors intimately related to the risk of death in young children and acting mainly at the level of the household. Central to the whole scheme is the child’s nutritional status as it affects its immunocompetence. But undernutrition sneaks in again through the backdoor during illness itself. If a sick child does not recover, it may die immediately or may first pass through a state known as growth faltering. If the child does experience growth faltering, its risk of dying rather than recovering is substantially increased (Pelletier et al. 1993). Given an ample food supply and good health care, neither of which is guaranteed in many populations particularly in the Third World, catch-up growth might follow growth faltering. Both growth faltering and catch-up growth,
As with mortality, demographers study migration in two ways: the level of migration and the age patterns of migration (Rogers and Castro 1986). The level of migration is particularly difficult to estimate because migration is difficult to define, particularly across cultures (see Box 14.1). In fact, even the Migration and Settlement Study, conducted by the International Institute for Applied Systems Analysis (a cooperative effort to study migration in several European countries), was unable to produce data sufficiently consistent across countries to conduct meaningful comparisons (Rogers and Castro 1986). For our purposes, migration is defined as a permanent or semipermanent change in location to a completely new geographical area. The intention is to separate “mobility” (or circulation or partial displacement migration) associated with nomadism (i.e., the yearly movements of hunter-gatherers and pastoralists within their territories), tourism, commuting (in modern societies), attending professional meetings, or changing residences within a metropolitan area from true migration or total displacement migration (Cavalli-Sforza 1962; Clark 1986). Mobility enables an individual to continue many of his or her activities within the old geographic area, whereas the true migrant transfers most of his or her activities to a new geographical area. In this regard, migration implies a new hunting territory, a new farm, or a new city. The movement of people within an old hunting territory, the movement of slash-and-burn agricultural fields around the same village, and the relocation of a family within an urban area are classified as mobility, not migration.

There is no standard demographic measure of the level of migration. Many studies simply resort to estimating the raw number of people that migrate, or the crude outmigration rate (Davis 1974). However, migration is highly age dependent (as discussed below), so some control for the age structure of the population is desirable. The measure used by the Migration and Settlement Study was the sum of the individual age-specific migration rates (Rogers and Castro 1986). This calculation has been called the “gross migration production rate” because of its similarity to the gross reproduction rate (see Chapter 15).

Like mortality, the age pattern of migration—at least in contemporary national populations—has a characteristic shape (see Fig. 14.11). The risk of migration tends to be high in infants and children, and in young adults 20–30 years of age. The high migration rate in children is thought to be an artifact of families moving, because families with parents in their 20s are likely to have young children. In contemporary national populations, fairly high migration rates also occur at 60–70 years. This peak presumably represents retirees moving to new locations (Clark 1986; Rogers and Castro 1986).

Migration rates do not appear to vary between the sexes in national populations. In present-day Sweden, for example, the rate of migration among females is only slightly higher than that of males (see Fig. 14.11). These principles appear to apply to the United States and are probably true for western Europe. However, this may result from neolocal marriage customs and the industrial sexual division of labor characteristic of contemporary industrial states. Marital migration in strongly matri-local or patrilocal societies must, by definition, be sex biased. Migration for employment purposes may attract one sex more than the other. Thus, sex-specific migration may depend on local employment conditions. More research on migration in small populations is needed.

**Figure 14.10** A framework for studying the major determinants of early childhood mortality in the preindustrial world, where most juvenile deaths are caused by infectious disease. Conceptually, the framework refers to the risk of death in a single child.
Variability in Level of Migration

Difficulties in defining migration consistently across populations make it difficult to conduct meaningful cross-cultural comparisons of the variability in level of migration. Nevertheless, some general trends in migration patterns have occurred with industrial development and within prehistoric and small populations. In this section, we consider the trends in internal migration, invasion (international) migration, and forced migration.

Characteristic changes in the level of internal migration accompany the process of industrial development (Fig. 14.12) (Zelinsky 1971). This mobility transition accompanies the historical decline in mortality (discussed earlier) and the decline in fertility (the demographic transition; see Chapter 15) as well as the epidemiological transition (discussed earlier). With industrial development, rural-to-urban migration increases during the early developing phases, contributing to the growth of cities, followed by a decline in the rural-to-urban flow in the later phases (i.e., in developed nations). Urban-to-urban migration also increases with development, as more of the population is located in cities and migration between cities predominates. Furthermore, circulation for business and vacation increases, apparently because of a decline in the relative costs of transportation. Finally, Zelinsky (1971) predicted that circulation may decline in the future, at least for business purposes, as a result of improved communication.

The developed nations are considered to be in the latest phases of this transition. Although not a part of Zelinsky’s (1971) original description, evidence now shows a reversal of the rural-to-urban migration in the early 1960s. Urban-to-rural migration appears to have exceeded rural-to-urban migration in the early 1970s (Oosterbaan 1980; Jones 1990). This trend may be the result of improvements in transportation communication and economics that reduce the advantages of cities as industrial hubs.

Zelinsky (1971) assumed that migration in small populations and in prehistoric times was generally low. This is not the case. Because of high transport costs in the preindustrial world—reflecting limited methods of transportation and a lack of infrastructure such as permanent roads—migration distances in the remote past were probably quite low, except where water transport was possible (Wood n.d.). Until the mid-19th century, for example, the average marriage distance (the distance between birthplaces of spouses) in Oxfordshire villages in England was 10–13 km (Küchemann et al. 1967), and among the !Kung, it is 51 km (Wijsman and Cavalli-Sforza 1984). However, the rate of circulation migration is high among nomadic hunter–gatherer populations, and it declined prehistorically only with the development of horticulture and particularly intensive agriculture (i.e., the situation as Zelinsky saw it at the advent of the industrial revolution) (Lee and Devore 1968; Fenner 1970). Of course, pastoral populations (who generally coexist with agriculturalists) have maintained high levels of circulation, as well. Additionally, rural-to-rural migration has always occurred, at least with respect to finding mates. Furthermore, there must have been a prehistoric “mobility transition” with the introduction of intensive agriculture and the original emergence of cities with the attendant possibility of rural-to-urban migration (Table 14.6). It has even been argued that mortality was so universally high in preindustrial cities that urban settlements could grow or even survive only because of high rates of net in-migration from the surrounding countryside. This “urban graveyard” or “law of natural urban decrease” hypothesis was first put forward in an influential book by a historical demographer working with records from early modern London (Wrigley 1969). Although the hypothesis has been accepted rather uncritically by more recent authors (e.g., Goose 1986; Barry 1990), it has not been immune to criticism (Flinn
1981; Galley 1998). For example, it has been shown that mortality exceeded fertility in historical London for only a brief period in the 17th and 18th centuries, and that the highest mortality at the time occurred among the rural migrants, not the urban-born segment of the city (Galley 1998, pp. 16–17). A recent paleodemographic study of the city of Cholula in pre-Hispanic Mexico has also suggested that the “urban graveyard” model may not be of general significance (Kreger 2010).

Prehistory is replete with migratory expansions of human populations (Table 14.14) (Cavalli-Sforza et al. 1993). These expansions have been called invasion and demic expansion, and they might be continuing today with modern international migration. The demic expansion of European populations into the rest of the world observed historically is only the latest of these phenomena. Clearly, the data in Table 14.14 indicate that humans—even prehistoric humans—are highly migratory. Of course, prehistoric migrations are underreported, because the evidence for these expansions is mainly inferred from the distribution of genes. Any migrations that did not influence the contemporary distribution of genes have been overlooked (see Chapter 4).

Invasion migration takes two forms: uncontested migration, when a population moves into a region previously unoccupied by human populations, and contested migration, when a population moves into a region occupied by another population. Invasion migration is generally thought to be the result of social, climatological, biological, or technical innovations that increase the rate of population growth in a particular population. The resulting population pressure is relieved by outmigration. An example might be the Viking expansion c. AD 1000, which coincided with the Early Medieval Temperature Optimum in Europe, and the Bantu expansion with the adoption of swidden agriculture. The rate of a population's geographical expansion from a central locus is simply a result of the population growth rate and the rate of migration (Davis 1974; Cavalli-Sforza et al. 1993). There are no direct ethnographic analogies for this process among hunter-gatherers, because most contemporary hunter-gatherer populations are not growing rapidly. In fact, they have been pushed into marginal environmental regions by the more recent expansion of agriculturalists. However, expansions of hunter-gatherer populations out of Africa (first as *Homo erectus* and later as *Homo sapiens sapiens*) to the rest of the world must have occurred prehistorically (Table 14.14). It is assumed that bands continually grow in size and then split apart; there is an increase in the number of bands and displacement of other bands in the area and an expansion of their hunting territories. New linguistic “tribes” might form as expansion continued and exchange of mates and communication between widely separated bands declined.

Horticultural populations have been observed during periods of expansion (Vayda 1969; Chagnon 1974; Early and Peters 1990). As for hunter-gatherers, the basic model is one of local population growth followed by the breaking apart of villages (in the case of horticulturalists) and the concomitant displacement of the surrounding local groups. Vayda (1969) proposed two models on the basis of ethnographic observations. The first is characterized by intratribal warfare, that is, warfare among the villages, including and perhaps particularly among recently split villages (Chagnon 1974). In this case, hostility among the villages provides the motivating force for the dispersal of the population. Examples include the Tiv, the Tupinamba, the Maori (Vayda 1969), and the Yanomama (Chagnon 1974; Early and Peters 1990). The second model is characterized by intratribal peace, relatively higher rates of mobility of people among villages, and the availability of unclaimed cultivable land is maintained by extratribal warfare and conquest. The Ibans of Sarawak have been proposed as an example of this type of expansion. In this case, new villages in virgin territory were often colonized by the younger families. The initial colonization may have been carried out by younger males in advance of the main body of settlers (Vayda 1969).

Invasions carried out by societies at the “state” level of socioeconomic integration probably resemble the Iban model, that is, relative internal peace (maintained

| Table 14.14 | List of Some Invasion Migration Events* |

<table>
<thead>
<tr>
<th>Origin of Migrants</th>
<th>Destination of Migrants</th>
<th>Period, BP in 1000 of Years</th>
<th>Contested or Uncontested</th>
<th>Advantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Africa (<em>H. erectus</em>)</td>
<td>Old World</td>
<td>&gt;1000</td>
<td>Uncontested</td>
<td>Not applicable</td>
</tr>
<tr>
<td>Africa (<em>H. sapiens sapiens</em>)</td>
<td>Old and New World</td>
<td>100–30</td>
<td>Contested (Old World)</td>
<td>Advances in tool production</td>
</tr>
<tr>
<td>Middle East</td>
<td>Europe, North Africa, and Southwest Asia</td>
<td>10–5</td>
<td>Contested (by hunter-gatherers)</td>
<td>Horticulture and intensive agriculture</td>
</tr>
<tr>
<td>Central America and Northern Andes</td>
<td>New World</td>
<td>9–2</td>
<td>Contested (by hunter-gatherers)</td>
<td>Horticulture and intensive agriculture</td>
</tr>
<tr>
<td>West Africa including Bantu expansion</td>
<td>Sub-Saharan Africa</td>
<td>4–0.3</td>
<td>Contested (by hunter-gatherers)</td>
<td>Horticulture</td>
</tr>
<tr>
<td>Eurasian Steppes</td>
<td>Eurasia</td>
<td>5–0.3</td>
<td>Contested (by agriculturalists)</td>
<td>Pastoral nomadism (horses and warfare)</td>
</tr>
<tr>
<td>Europe</td>
<td>Much of the world</td>
<td>0.4–0.04</td>
<td>Contested (by agriculturalists and hunter-gatherers)</td>
<td>Advances in transportation, and later industrial development (the demographic transition)</td>
</tr>
<tr>
<td>Developing countries</td>
<td>Developed countries</td>
<td>0.04 to present</td>
<td>Contested (by industrialists)</td>
<td>Industrial development (the demographic transition)</td>
</tr>
</tbody>
</table>

* Adapted from Cavalli-Sforza et al. (1993).
in this case by the state) and warfare against other groups. Some invasions probably took place as explicit acts of conquest. How much migration actually took place when the Mongols under Genghis Khan overran much of Asia and eastern Europe is not known (Davis 1974); perhaps, there was very little. Other invasions may have largely replaced the original populations, such as the European colonization of North America (Weiss 1988). The European invasion of North America and Australia is not dissimilar to the process of Iban expansion described above except that “state” governments may actively promote the process of expansion. It is interesting to point out that the European invasion of underdeveloped areas of the rest of the world during the historic period has recently been replaced by a counterclockwise flow of migrants from the developing countries to the developed countries (e.g., the migration of Hispanic populations in the United States) (Table 14.14).

One question with respect to contested migration is why the invading population replaces the indigenous population in some cases and in other cases does not (Weiss 1988). Modern analogies, such as the European invasion of the New World, are varied. In North America and in Costa Rica, for example, the indigenous populations were largely replaced, whereas in Mexico and much of South America, the indigenous populations remained an important segment of society. Three possible explanations of these differences are the size and density of the indigenous population, the relative differences in technology, and/or the relative differences in the level of sociopolitical integration (e.g., the extent to which indigenous people were integrated into the invading culture). Clearly, where indigenous populations were low, replacement occurred. However, there were also large relative differences in technology and the level of sociocultural integration was high in these locations, so there is no single clear explanation for the variation.

We generally think of migration as voluntary and based on the decisions of individuals or families. However, migration may be forced—that is, not voluntary from the point of view of the migrant. This category would include the forced transport of Africans to the New World, the slave trade within Africa and Europe, deportation of convicts to Australia, and even the capture of wives by Yanomama (Chagnon 1974; Early and Peters 1990). The movement of refugees during periods of war and civil strife is also considered forced migration. This form of migration appears to be increasing with development. Davis (1974) estimated that over the 55-year period from 1913 to 1968, 71.1 million individuals throughout the world were forced to migrate, whereas over the 90-year period from 1840 to 1930 (the height of the European expansion), an estimated 52 million left Europe voluntarily. More recently, the breakup of the Soviet Union and civil strife in Africa, Asia, and Central America have contributed to the volume of people forced to migrate. Forced migration is clearly an important and perhaps increasing component of total migratory flows.

**Variability in the Age Patterns of Migration**

Almost no quantitative data on the age patterns of migration exist, outside of a few developed nations. However, some circumstantial evidence suggests that the age patterns of migration in small populations are qualitatively similar to those plotted in Figure 14.11. With respect to contemporary hunter–gatherers, Fix (1977) reported the highest rates of migration during “childbearing ages” among the Semai Senoi. Similar findings have been reported for horticulturalists such as the Yanomama (Early and Peters 1990) and the Iban (Vayda 1969).

It appears likely that most migration occurs just before or shortly after the beginning of the reproductive career, and may be considered part of a larger “household-formation” strategy. However, adjustment might need to be made for lower ages of marriage that characterize many small populations compared with contemporary industrialized societies. However, forced migration and the pattern of village fissioning along family lines in hunter–gatherer and horticultural populations may not be well represented by Figure 14.11. There are simply no good quantitative data on the age patterns of migration in these situations. Additional research is necessary to determine the extent of this variation.

**Proximate Causes of Migration**

The proximate causes of migration are sometimes divided into causes that push people out of their current area of residence and causes that pull people to new locations. Proximate causes may stem from a new marriage, economic pressures or attractions, or forced displacement. In voluntary migration, the proximate cause is clearly the difference between the conditions in the original location and those at the potential new location, including, perhaps, the availability of potential spouses.

Marital migration occurs in all populations, as individuals move in search of mates or couples marry and set up new households in a new region. In hunter–gatherers, bands are typically exogamous and exchange mates among bands within the same linguistic “tribe” (Service 1962; Lee and DeVore 1968). Horticulturalists may or may not practice village exogamy but tend to practice clan or lineage exogamy. At the same time, mates are usually exchanged within subunits of the same linguistic “tribe” (Service 1962). Among agriculturalists, marital migration occurs among households (or farms) within the agricultural proportion of the population, or within the urban portions of the population. In any event, because this process is conceived of as an exchange of mates, the results do not greatly change the demographic structure of the populations involved. However, this kind of migration may have profound effects on the genetic structure of the population (see Chapter 4).

In general, people migrate voluntarily because they believe that moving will enhance their economic well-being; that is, they believe that the benefits accrued in the future exceed the costs of migration (Sjaastad 1962). In developed countries, individuals and/or families clearly benefit from migrating (DaVanzo 1976; DaVanzo 1978; Grant and Vanderkamp 1980). Whether the same is true of migrants in the developing countries, particularly migrants to developing cities, is less clear. Todaro (1976) has argued that migrants in these areas may not be better off economically because of the high rates of unemployment and/or underemployment that characterize cities in developing countries. Todaro argued that migration may be driven by the perception of being better off. Unfortunately, sufficient economic data are not available to formally test these models on a large scale in the developing countries. However, studies of the biological indicators of well-being, such as faster growth and maturation and lower mortality and fertility, suggest that long-term
migrants from rural to urban areas of the developing countries are generally better off than nonmigrants (Bogin 1988) (see Chapter 12). Similar economic and/or ecological considerations are thought to have driven the invasion migrations (Davis 1974; Cavalli-Sforza et al. 1993).

The reasons for forced migration are more varied. Some forced migration may be economic. Certainly, the slave trade was based on the need (by the enslavers) for a labor force (Davis 1974). Famine is also a frequent cause of forced migration (Bongaarts and Cain 1982; Findley and Salif 1998; Dyson and Ó Gráda 2002). Other forced migration may have political rather than economic causes; this includes the growing ranks of refugees resulting from international and civil strife.

As Davis (1974) pointed out, much of this politically forced migration stems ironically from the principle of "national self-determination" originally proposed by President Woodrow Wilson but carried to its extreme in the form of "ethnic cleansing."

A question that has concerned human biologists is whether migrants (voluntary migrants—the question is moot for forced migrants) are truly better off. This question is difficult to answer because it requires a comparison with how well off the migrant would have been had he or she not migrated. Comparisons with nonmigrants are frequently used as a surrogate measure of the effects of migration on the individual. However, migrants are not a random sample of the individuals who choose to stay; furthermore, the simple fact that a migrant leaves changes the environment in which the nonmigrant is exposed to. The evolutionary hypothesis suggests that migrants should be worse off because they migrated from an environment to which they are presumably better adapted (genetically, developmentally, physiologically, and/or socially) to a different environment, to which they may not be well adapted (Little and Baker 1988). Blood pressure, for example, a risk factor for cardiovascular disease, is generally higher in rural-to-urban migrants than in nonmigrants (Little and Baker 1988). These phenomena, in addition to moving and setting up a new household and finding employment (Sjaastad 1962), are the costs of migrating. As mentioned earlier, however, overall the biological and economic data currently available suggest that the benefits of moving outweigh the costs at least for voluntarily migrants (DaVanzo 1976, 1978; Grant and Vanderkamp 1980; Bogin 1988; Little and Baker 1988; Hummer et al. 2000; Frisbie et al. 2001; Cho et al. 2004). Still it is not clear whether all those who choose to migrate are truly better off. Comparisons of migrants and nonmigrants are based on studies of the migrants that actually succeed at migrating. Some migrants die during the migration process or fail to adapt to new surroundings and either move on or move back to where they came from (Markides and Eschbach 2005). Thus, the apparent improved living conditions of migrants may be an artifact of heterogeneity.

CHAPTER SUMMARY

In this chapter, we have considered some of the problems in estimating demographic rates, the history of variation in human mortality patterns, and human migration. One basic tool for demographic analysis is the life table, an array of mortality rates by age for a particular population. The interpretation of demographic rates is sometimes difficult, even when the data on which they are based are completely accurate. However, demographic data are often inaccurate, incomplete, and/or biased. In general, demographic data on small nonindustrial, non-Western populations tend to be of poor quality whether collected ethnographically or archaeologically.

All human populations show a similar U-shaped age pattern of mortality. Mortality declines from birth to about age 5 to age 10, remains relatively low throughout the next 20 years of life, and then begins to increase at an ever-increasing rate at least until 70 or 80 years of age. Some populations may show additional features—such as a weaning hump or accident hump—under special conditions.

Age patterns of mortality vary among national populations and may vary between national populations and small populations. In general, the age pattern of mortality has become increasingly U-shaped over time, at least at the younger ages. Another apparent change in mortality over time is that the large sex differentials in favor of female characteristic of contemporary national populations are not universal and probably are a relatively recent development.

The level of mortality varies widely among past and living human populations, from expectations of life at birth of less than 15 to more than 80 years. Most estimates of prehistoric life expectancy at birth vary within a surprisingly narrow range, from perhaps 20 to 35 years. The means are similar for hunter-gatherers, horticulturalists, and agriculturalists, indicating that mortality during these times was high and apparently did not vary a great deal from one subsistence type to another. In historic populations prior to the industrial revolution, expectation of life at birth was short—usually less than 40 years. Among contemporary nations, expectation of life ranges from about 40 to more than 80 years. Expectation of life at birth among contemporary small populations, studied ethnographically, suggests that the level of mortality is lower among contemporary small populations than among archaeologically studied small populations. The extent of this reduction in mortality depends on the assumed prehistoric age pattern of mortality. It is possible that the high adult mortalities of traditional prehistoric life tables may be underestimated. This conclusion is based on two reestimated life tables using improved age methods and must be further verified. The unusual age pattern of prehistoric life tables at the younger ages may be the result of underenumeration of infant deaths or may reflect environmental differences between prehistoric, and contemporary small and national populations.

During the historic period, the major causes of death changed from predominantly infectious diseases to predominantly chronic degenerative diseases, a process that has been called the "epidemiological transition." The risks of both infectious and degenerative causes of death declined, and the transition from a predominance of infectious deaths to degenerative deaths appears to be the result of the faster decline of the infectious than degenerative causes of death. The rapid decline in deaths from infectious causes, especially among young children, may have resulted from improvements in agricultural yields and the food supply, but that hypothesis needs to be tested further.

Several kinds of migration were considered: internal migration, invasion migration, and forced migration. Migration rates have changed over time and vary with age. Migration rates tend to be particularly high in young adults between the years of 20 and 30 years of age.
ACKNOWLEDGMENTS

We would like to thank Drs. Kathleen O’Connor, Richard Wilkinson, John Relethford, Alan Swedlund, Anne Buchanan, Patricia Johnson, and Lyle Konigsberg for comments on the original manuscript and suggestions concerning the treatment of migration. We would also like to thank Kiersten Fussell for her help in preparing the manuscript.

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RECOMMENDED READINGS
