# 5

# Age-Dependent Population Structures

## Introduction

This chapter presents an analysis of the distribution of ages in a population. We begin with a discussion of the aging process itself and then present some data on the age structures of actual populations. We finish with a mathematical description of age structures. Our primary interest is in humans, but the principles we present will apply to practically any mammal and perhaps to other animals as well.

## 5.1 Aging and Death

*The notion of aging is not simple. One must consider that oak trees, and perhaps some animals like tortoises, seem to have unlimited growth potential, that a Pacific salmon mates only once and then ages rapidly, and that humans can reproduce for many years. In each case a different concept of aging may apply.*

*The reason that aging occurs, at least in mammals, is uncertain. The idea that the old must die to make room for the new gene combinations of the young is in considerable doubt. An alternative hypothesis is that organisms must partition their resources between the maintenance of their own bodies and reproduction, and that the optimal partitioning for evolutionary fitness leaves much damage unrepaired. Eventually, the unrepaired damage kills the organism. We present several hypotheses about how and why damage can occur.*

*What is meant by "aging" in an organism?*

We will use a simple definition of aging, or *senescence*:[1] it is a series of changes that accelerate with age and eventually result in the death of an organism. This definition is a loose one because it does not specify the source of the changes—the

[1] There is much argument about definitions in the study of aging, and we wish to avoid being part of the dispute. Our simplification may have the opposite effect!

R.W. Shonkwiler and J. Herod, *Mathematical Biology: An Introduction with Maple and Matlab*, Undergraduate Texts in Mathematics, DOI: 10.1007/978-0-387-70984-0_5,

only requirement is that they accelerate. We will adopt a common approach and not regard predation, injury, and disease caused by parasites, e.g., microorganisms, as causes of aging, even though their incidence may increase with age.

The effect of aging on survival is demonstrated in Figure 5.1.1 for a simple model system of test tubes. Suppose that a laboratory technician buys 1000 test tubes and that 70% of all surviving test tubes are broken each month. Curve (a) of Figure 5.1.1 shows the specific rate of breakage of the tubes—a constant 70% per month.[2] Note that a test tube surviving for three months would have the same chance of breakage in the fourth month as would one at the outset of the experiment (because aging has not occurred). Alternatively, suppose that the test tubes broke more easily as time passed. A tube surviving for three months would have a much greater chance of breakage during the fourth month than would one at the outset of the experiment (because the older one has aged). Curve (b) shows the rate of breakage for these tubes (doubling each month in this example).

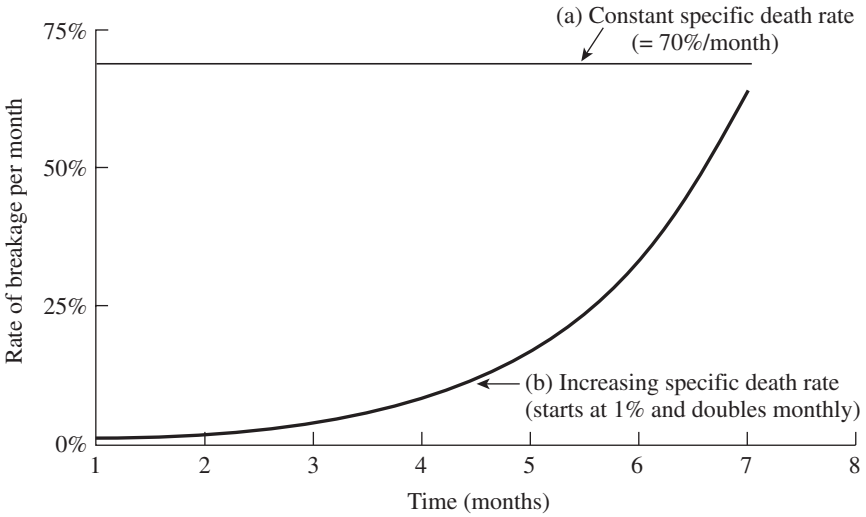


**Fig. 5.1.1.** Death rate, modeled on the breakage of test tubes. Curve (a) is obtained by assuming a specific death (breakage) rate of 70% of survivors per month of test tubes surviving to that point. This is equivalent to assuming that there is no aging, because the probability of death (breakage) is independent of time. The data of curve (b) is obtained by assuming that the specific death rate is 1% of the survivors in the first month and then doubles each month thereafter. This is equivalent to assuming that the test tubes age, because the probability of death (breakage) increases with time.

[2] The specific death (= breakage) rate is the number dying per unit time *among those of a specific age*. This is to be distinguished from the simple death rate, which is the death rate irrespective of age. In this experiment, of course, all the test tubes are of the same age.

Figure 5.1.2 shows survivorship curves for the two cases whose specific death rates are described by Figure 5.1.1. You should compare them to Figures 4.1.2 and 4.1.4, which are survivorship curves for $r$-strategists and $K$-strategists, respectively. It should be clear that $r$-strategists do not show aging (because they are held in check by climatic factors, which should kill a constant fraction of them, regardless of their ages).[3] The situation with regard to $K$-strategists is a bit more complex: Mammals, for instance, are held in check by density-dependent factors. If they live long enough, aging will also reduce their numbers. Both density-dependent factors and aging become more important as time passes. Thus the survivorship curve for a mammalian $K$-strategist should look somewhat like that shown in Figures 4.1.4 and 5.1.2(a).

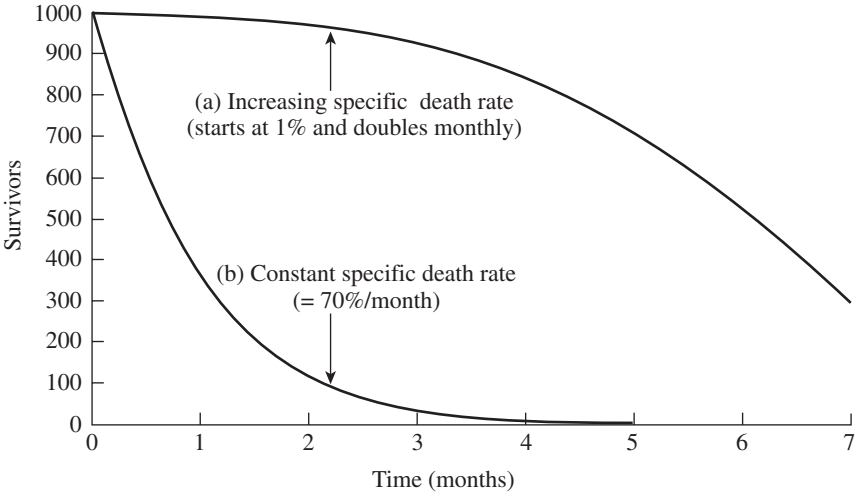


**Fig. 5.1.2.** (a) A survivorship curve for a nonaging system, using the data of Figure 5.1.1(b). (b) A survivorship curve for a system that exhibits aging, using the data of Figure 5.1.1(a). Both curves assume an initial cohort of 1000 test tubes at time $t = 0$. Note the similarity of curves (a) and (b) to Figures 4.1.2 and 4.1.4, which are survivorship curves for $r$-strategists and $K$-strategists, respectively.

*Why do organisms age and die?*

When asking "why" of any biological process as profound as senescence, we should immediately look to the Darwinian model of evolution for enlightenment and seek a positive selective value of aging to a species. A characteristic conferring a positive advantage is called an *adaptation*, and as we shall see, the adaptation we seek may not exist.

A simple adaptive explanation for senescence is that the Darwinian struggle for survival creates new organisms to fit into a changing environment. Thus the previous

[3] This is admittedly an approximation.

generation must die to make space and nutrients available for the new generation. Thomas Kirkwood has made two objections to this hypothesis [1]. The first objection is posed in the question, "How can aging have a positive selective value for a species when it can kill all the members of the species?" Besides, many organisms show the most evident aging only after their reproductive lives have ended. If the organism should show genetically programmed deterioration in its old age, that would have minimal (or no) selective value because the organism's reproductive life would have already ended anyway.

Kirkwood's second objection is that most organisms live in the wild and almost always die from disease and predation. Thus there is no need for selection based on aging in most organisms—they die too soon from other causes.

There is another way to answer the question, "Why do organisms age?"—one that is nonadaptive in that aging does not have a positive selective value. First, recall that in Section 4.1 we discussed how trees can partition each year's energetic resources and physical resources between asexual and sexual reproduction. For a year or two a tree would add thick trunk rings (asexual growth) at the expense of reduced nut production (sexual reproduction). Then for a year or two, the tree would reverse the situation and produce lots of nuts at the expense of vegetative growth. There is a hypothesis about aging that generalizes this situation; it is called the *disposable soma model*.[4]

Kirkwood assumes that the organisms whose aging is of interest to us must partition their finite resources between reproduction and the maintenance of the soma, i.e., the body. In particular, somatic maintenance means the repair of the many insults and injuries that are inflicted on the body by factors like ordinary wear and tear, toxin production, radiation damage, and errors in gene replication and expression. The two needs, reproduction and somatic maintenance, thus compete with one another. If excessive resources are put into somatic maintenance, there will be no reproduction, and the species will die out. If excessive resources are devoted to reproduction, there will be insufficient somatic maintenance, and the species will die out. We thus assume that there is an optimal partitioning of resources between somatic maintenance and reproduction. The disposable soma model postulates that this optimal partitioning is such that some somatic damage must go unrepaired and that the organism eventually dies because of it. Thus the organism has a finite lifetime, one marked by increasing rate of deterioration, i.e., aging.

The disposable soma model is nonadaptive in that aging is a harmful process. It is, however, an essential process because it is a measure of the resources diverted into reproduction. In a way, aging is a side effect, but, of course, it has powerful consequences to the organism.

*Aging of cells can provide insight into organismal aging.*

The death of the only cell comprising an amoeba has consequences that are quite different from those associated with the death of a single skin cell of a person; thus we will have to distinguish between aging in single-celled and multicellular organisms.

[4] "Soma" means "body."

It is fine to study the processes that lead to the death of a cell, but what if that cell is only one of many in an organ of a multicellular organism? To answer this question, we must first understand that cell death is a natural part of the life and development of organisms. Our hands are initially formed with interdigital webbing, perhaps suggesting our aquatic ancestry. This webbing is removed in utero by the death of the cells that comprise it in a process called *apoptosis*. There are many other examples of cell death as a natural consequence of living: our red blood cells live only about three months and our skin cells peel off constantly. Both are quickly replaced, of course.

We can now return to the question of what happens if one, or even a small fraction, of the cells in an organ die. Usually, nothing—we see that it happens all the time. But if that cell dies for a reason connected to the possible deaths of other cells, then the study of the one cell becomes very important. Thus the study of aging in cells can contribute greatly to our knowledge of aging in multicellular organisms.

*How do organisms become damaged?*

Whether we accept Kirkwood's disposable soma model or not, it is clear that our cells age, and we must suggest ways that the relevant damage occurs. Numerous mechanisms have been proposed, but no single one has been adequate, and in the end it may be that several will have to be accepted in concert. Some examples of damage mechanisms that have been proposed are the following:

(a) *Wear and tear*: A cell accumulates "insults," until it dies. Typical insults are the accumulation of wastes and toxins, as well as physical injuries like radiation damage and mechanical injury. These are all well known to cause cell death. Cells have several mechanisms by which insults can be repaired, but it may be that these repair systems themselves are subject to damage by insults.
(b) *Rate of living*: This is the "live fast, die young'' hypothesis. In general, the higher a mammal's basal metabolic rate, the shorter its life span is. Perhaps some internal cellular resource is used up, or wastes accumulate, resulting in cell death.
(c) *Preprogrammed aging*: Our maximum life span is fixed by our genes. While the average life span of humans has increased over the past few decades, the maximum life span seems fixed at 100–110 years. Noncancerous mammalian cell lines in test tube culture seem capable of only a fixed number of divisions. If halfway through that fixed number of divisions, the cells are frozen in liquid nitrogen for ten years and then thawed, they will complete only the remaining half of their allotted divisions.

*Cell reproduction seems to have a rejuvenating effect on cells.*

It is a common observation that cells that reproduce often tend to age more slowly than cells that divide infrequently. This effect is seen in both asexual and sexual reproduction. Cancer cells divide rapidly and will do so in culture forever. Cells of our pancreas divide at a moderate rate, and our pancreas seems to maintain its function well into old age. Brain cells never divide and brain function deteriorates

noticeably in old age. Even single-celled organisms can exhibit this effect: they may show obvious signs of senescence until they reproduce, at which point those signs disappear.

## 5.2 The Age Structure of Populations

*Age-structure diagrams show the frequency distribution of ages in a population. The data for males and females are shown separately. The shape of these diagrams can tell us about the future course of population changes: The existence of a large proportion of young people at any given time implies that there will be large proportions of individuals of childbearing age* 20 *years later and of retirees* 60 *years later. The shapes of age-structure diagrams are also dependent on migration into and out of a population. Comparison of data for males and females can tell us about the inherent differences between the genders and about the society's attitude toward the two genders.*

*Age-structure diagrams are determined by age-specific rates of birth, death, and migration.*

Figure 5.2.1 is a set of age-structure diagrams for the United States for 1955, 1985, 2015 (projected), and 2035 (projected) (see also [2]). They show how the population is, or will be, distributed into age groups. Data are included for males and females.

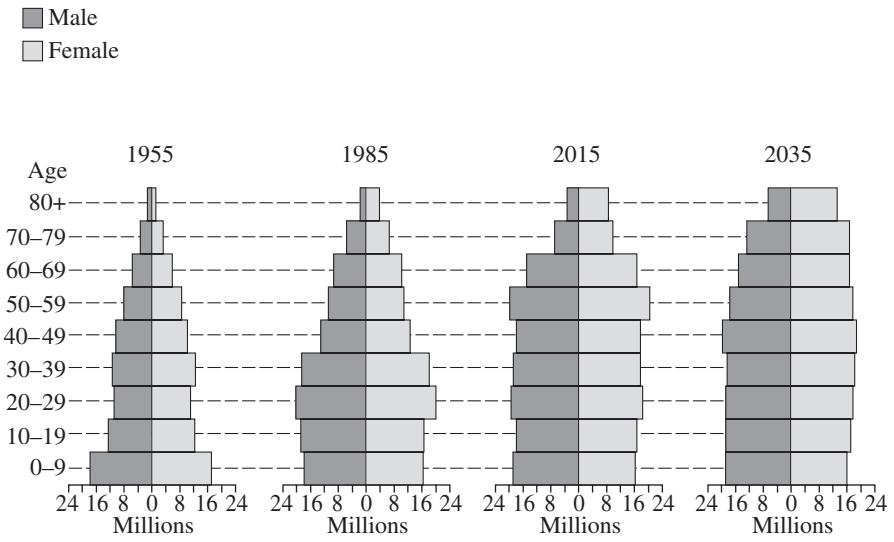


**Fig. 5.2.1.** Past and future (projected) age-structure diagrams for the United States. Note the growing proportion of elderly, compared to young, people. The cohort of "baby boomers" is evident at the base of the 1955 data. That group moves up in the 1985 and 2015 diagrams. (Redrawn from "Age and Sex Composition of the U.S. Population," in *U.S. Population: Charting the Change: Student Chart Book*, Population Reference Bureau, Washington, DC, 1988. Used with permission.)

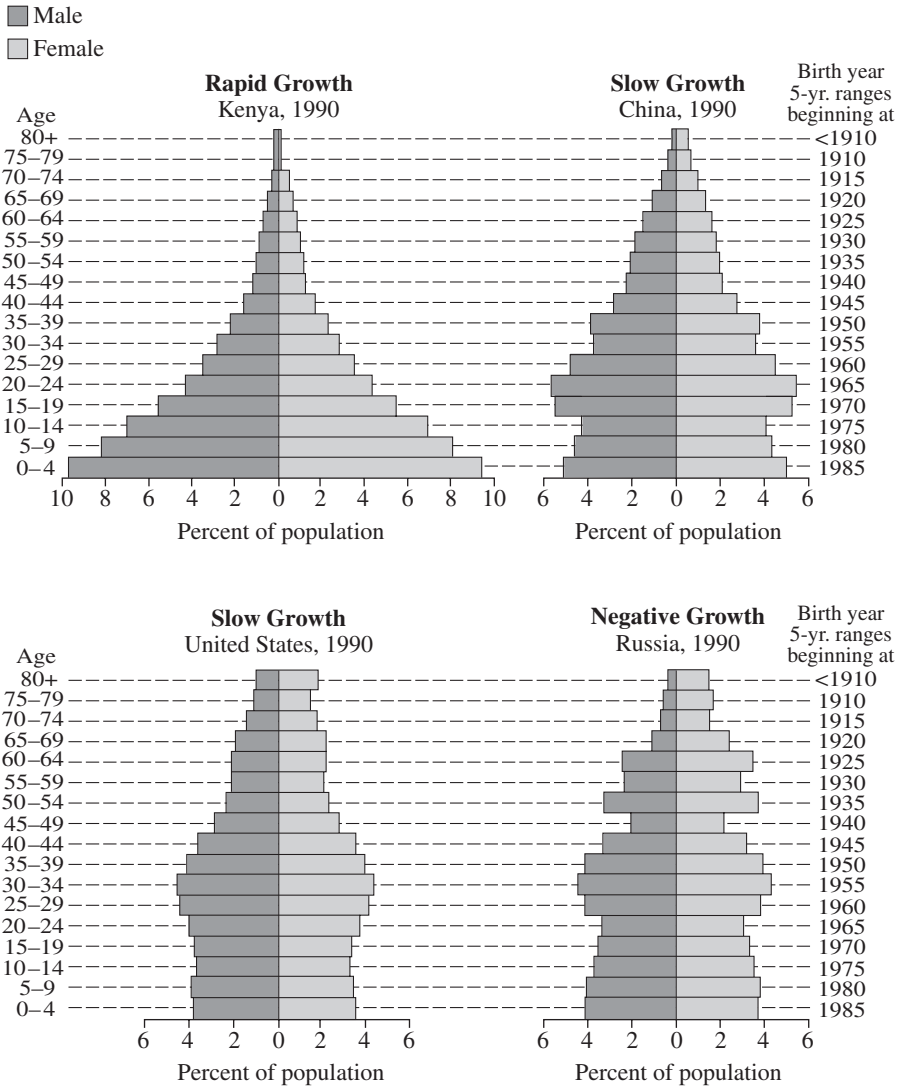


**Fig. 5.2.2.** Age-structure diagram for four countries for 1990. Each is labeled according to its expected future growth rate. For instance, Kenya has a high proportion of young people, so we expect its future growth rate to be high. (Redrawn from "Patterns of Population Change," in *World Population: Toward the Next Century*, Population Reference Bureau, Washington, DC, 1994, p. 5. Used with permission.)

These diagrams can convey a great deal of information. For example, look at the data for 1955 and note the 20–30-year-old *cohort*.[5] There are relatively fewer people

[5] A cohort is a group of people with a common characteristic. Here the characteristic they share is that they were born in the same decade.

in this group because the birth rate went down during the Great Depression. On the other hand, the birth rate went up dramatically after the Second World War, as the 20–40-year-old cohort in 1985 (the "baby boomers'') shows clearly. Both of these cohorts can be followed in the projected data. Note also how the population of elderly people, especially women, is growing.

Figure 5.2.2 shows recent data for four countries—Kenya, China, the United States, and Russia. Future population growth can be estimated by looking at the cohort of young people, i.e., the numbers of people represented by the bottom part of each diagram. In a few decades, these people will be represented by the middle part of age-structure diagrams *and* will be having babies. Thus we can conclude that the population of Russia will remain steady or even decrease, those of the United States and China will grow slowly to moderately, and that of Kenya will grow rapidly.

Another factor besides births and deaths can change an age-structure diagram: migration into and out of a population may change the relative numbers of people in one age group. Figure 5.2.3 shows data for Sheridan and Durham Counties, North Carolina, for 1990. Rural areas of the Great Plains have suffered a loss of young people due to emigration, and the data for Sheridan County demonstrate it clearly. On the other hand, Durham County is in the North Carolina Research Triangle, the site

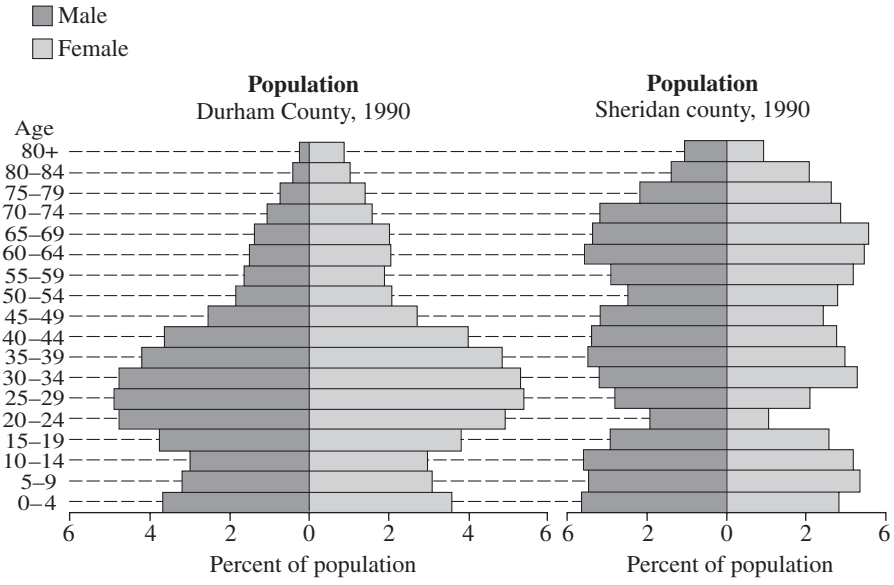


**Fig. 5.2.3.** An age-structure diagram showing the effects of migration. Many young people in the 20–45-year-old age group have moved into Durham County, North Carolina, and many young people in the 20–30-year-old age group have moved out of Sheridan County, North Carolina. (Redrawn from "Age and Sex Profiles of Sheridan and Durham Counties, 1990,'' in "Americans on the Move," *Population Bull.*, **48**-3 (1993), 25 (published by Population Reference Bureau, Washington, DC). Used with permission.)

of several major universities and many research industries. It is therefore a magnet for younger people, and its age-structure diagram reflects that fact.

*Some populations have more men than women.*

We are accustomed to the idea that there are more women than men in our country. That (true) fact can be misleading, however. While the sex ratio at conception is not known, there is evidence that a disproportionate number of female fetuses are spontaneously aborted in the first trimester of pregnancy. On the other hand, in the second and third trimesters, more male than female fetuses are lost. The ratio of sexes at birth in the United States is about 106 males to every 100 females. The specific death rate for males is higher than for women, and by early adolescence the sex ratio is 100:100. You can refer back to Figure 5.2.1 to see the effect of males' higher death rate on the relative numbers of males and females in later life.

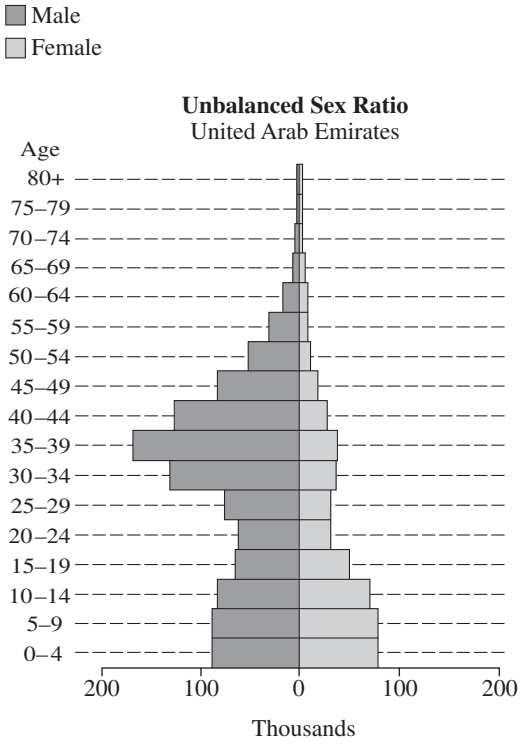


**Fig. 5.2.4.** Age-structure diagram from the United Arab Emirates showing an unbalanced sex ratio. The gender imbalance, males outnumbering females, is due to the importation of males to work in the oil fields: these males are not accompanied by their families. (Redrawn from "Unbalanced Sex Ratio: United Arab Emirates, 1985," in "Population: A Lively Introduction," *Population Bull*, **46**-2 (1991), 25 (published by Population Reference Bureau, Washington, DC). Used with permission.)

The fact that there are more females than males in the United States might lead us to be surprised by the data of Figure 5.2.4, an age-structure diagram for the United Arab Emirates. The unbalanced sex ratio, heavily tilted toward males, arises from immigration: U.A.E. has brought in many men from other countries to work in its oil fields, and the men seldom bring their families.

Another feature of gender ratios can be noted in age-structure diagrams of certain countries. In the late 1980s, the ratio of men to women in advanced countries was about 94:100; in developing countries, it was about 104:100.

## 5.3 Predicting the Age Structure of a Population

*A graph of population size $P$ as a function of age $y$ visually documents the age structure, or profile, of a population. Over time, a population profile can change due to periodic environmental conditions that may be favorable or unfavorable to the population, and to occasional events such as natural diasters and epidemics. For human populations, medical improvements have gradually increased the representation in the higher age brackets.*

*But much greater use can be made of the population density function $P$. With a knowledge of survival rates by age, $\ell(y)$, the trend in $P$ can be predicted. It can be shown that if survival rates are relatively constant over time, then the age structure of a population tends to a fixed profile within which the overall size of the population may nonetheless increase or decrease.*

*Age structure is the distribution of a population by age.*

The age structure of a population can be described by means of a function $P(y)$ giving the size of the population in the $y$th age group for a set of groups covering all possible ages. Table 5.3.1 shows the age distribution of the U.S. population in 1990 refined to 20-year age brackets. Mathematically, it is more common to use one-year age brackets, so that $P(0)$ is the number of newborns less than one year of age, $P(1)$ counts the one-year-olds, and so on. We shall refer to $P(y)$ as the *age density* function. The total size of a population is calculated from its density by summing,

**Table 5.3.1.** U.S. population, 1990.

| Age bracket | Number bracket (in millions) |
|---|---|
| 0–20 | 71.8 |
| 20–40 | 103.4 |
| 40–60 | 60.3 |
| 60–80 | 20.9 |
| 80–100 | .209 |
| 100 | .001 |

$$P = \sum_{n=0}^{\infty} P(n). \tag{5.3.1}$$

The use of infinity as the upper limit of this sum is a simplifying measure; for some age, maybe $y_{\max} = 115$, $P(y) = 0$ for $y > y_{\max}$, so the indicated infinite sum is in reality only from 0 to 115.

The age structure of the United States has gradually evolved over the last half of the twentieth century, as seen in Figure 5.2.1. On the other hand, any of several catastrophes can bring about rapid change to an age structure. We account for these possibilities by regarding the age-density function as dependent on calendar time $t$ as well as age $y$, and in deference to these dual dependencies we write $P(y, t)$. In addition, including the reference to time provides a mechanism for describing births year by year, namely, $P(0, t)$. This is the birth rate of a population in year $t$. If the birth rate is down in some year, say, $t = t_0$, this affects the population in subsequent years as well, as we have seen above. To begin with, the population of one-year-olds cannot exceed the population of newborns in the previous year,

$$P(1, t_0 + 1) \leq P(0, t_0),$$

assuming no immigration into the population, of course. This is generally true for any age bracket; thus under the condition of no immigration,

$$P(y + 1, t + 1) \leq P(y, t) \quad \text{for } y \geq 0 \text{ and for all } t. \tag{5.3.2}$$

While the population in an age bracket cannot increase in the following year, it can decrease due to deaths that occur during the year. Let $\mu(y)$ denote the death rate, or *mortality*, experienced by the population of age $y$. The *death rate* is dimensionless, being the fraction of deaths per individual, or since it is usually a number in the thousandths, it is frequently given as deaths per 1000 individuals. The actual number of deaths that occur among the segment of the population of age $y$ in year $t$ is the product of the death rate and the number of individuals at risk,

$$\mu(y)P(y, t)$$

($\mu$ must be deaths per individual here or $P$ must be population in thousands).

Virtually all natural populations experience very high preadult mortality rates. Insect populations and other unnurtured species ($r$-strategists; cf. Chapter 4) experience death rates similar to that shown in Figure 5.1.1(a). Notice that the newly hatched young suffer the highest mortality rates, with improvement as the animal ages. By contrast, nurtured species ($K$-strategists), such as mammals, experience much lower preadult mortality rates, as seen in Figure 5.1.1(b).

A mortality table for the United States is given in Table 5.3.2. In most species, mortality rates are lowest during the middle adult years.

Returning to (5.3.2), taking deaths into account yields the equality

$$P(y + 1, t + 1) = P(y, t) - \mu(y)P(y, t), \tag{5.3.3}$$

**Table 5.3.2.** U.S. mortality table for 1991. (Source: U.S. Department of Health and Human Services, Hyattsville, MD.)

| Age | Deaths (%) |
|---|---|
| 0–10 | 1.2 |
| 10–20 | .57 |
| 20–30 | 1.2 |
| 30–40 | 1.8 |
| 40–50 | 3.1 |
| 50–60 | 7.2 |
| 60–70 | 16.4 |
| 70+ | 100 |

**Table 5.3.3.** U.S. mortality rates; rates per 1,000 population. (Source: U.S. Department of Health and Human Services, Hyattsville, MD.)

| Year | Average mortality |
|---|---|
| 1920 | 13.0 |
| 1930 | 11.3 |
| 1940 | 10.6 |
| 1950 | 9.6 |
| 1960 | 9.5 |
| 1970 | 9.5 |
| 1980 | 8.6 |
| 1990 | 8.6 |

provided there is no immigration or emigration. But this equation ignores the effect of external events that may play havoc with death rates. For example, due to a catastrophic epidemic, death rates in the youth age groups may be high during the calendar year in which it strikes. On the other hand, the U.S. population has experienced a gradually decreasing death rate over this century as a result of improved medical care (see Table 5.3.3). To account for these and other factors unrelated to age, we must regard $\mu$ as a function of time as well as age. Thus (5.3.3) becomes

$$P(y+1,t+1) = P(y,t) - \mu(y,t)P(y,t) = \ell(y,t)P(y,t), \tag{5.3.4}$$

where $\ell(y,t) = 1 - \mu(y,t)$ is the fraction of the population of age $y$ that will live through year $t$. These factors $\ell(\cdot,\cdot)$ are called *survival rates*.

*In the absence of external events, populations evolve to a stable age distribution.*

While survival rates depend on calendar time in general, here we are interested in predicting the population structure in the absence of external events. Consequently, we will regard $\mu$ (and $\ell$) as a function of age only.

If we know yearly birth rates $P(0,t)$ and age-specific survival rates $\ell(y)$, (5.3.4) allows us to calculate the course of the population through time, including its age

distribution and size. We also need to know the present age distribution, $P(y, 0)$, where we may regard the present time as $t = 0$. Usually the calculation is done for the female population of the species, since birth rates depend largely on the number of females while being somewhat independent of the number of males. The birth rates given will therefore pertain to the birth of females.

We illustrate this calculation for a $K$-strategist, specifically, for the gray seal, whose (female) fecundity and survival rates are given in Table 5.3.4.

**Table 5.3.4.** Gray seal fecundity and survival rates. (Source: D. Brown and P. Rothery, *Models in Biology: Mathematics, Statistics, and Computing*, Wiley, Chirchester, UK, 1993.)

| **Age** | 0 | 1 | 2 | 3 | 4 | 5 | 5+ |
|---|---|---|---|---|---|---|---|
| **Fecundity** | 0 | 0 | 0 | 0 | 0.08 | 0.28 | 0.42 |
| **Survival** | 0.657 | 0.930 | 0.930 | 0.930 | 0.935 | 0.935 | 0 |

To get it started, we make the assumption that the present population has uniform age density. Actually, this assumption about the starting population is not important in the long term, as we will see in the exercises. The key values are the birth and survival rates in the table. Since the survival rate for age 0 is 0.657, from (5.3.4) we have

$$P(1, t + 1) = 0.657P(0, t) \quad \text{for all } t \geq 0.$$

Similarly, for $y = 1, 2, 3$,

$$P(y + 1, t + 1) = 0.930P(y, t) \quad \text{for all } t \geq 0.$$

And for $y = 4, 5$,

$$P(y + 1, t + 1) = 0.935P(y, t) \quad \text{for all } t \geq 0.$$

In this we take $5 + 1$ to be $5+$. Since there is no category beyond "5+," the survival rate $\ell(5+)$ is 0. The birth-rate calculation uses the fecundity entries and is only slightly more complicated,

$$P(0, t + 1) = 0.08P(4, t) + 0.28P(5, t) + 0.42P(5+, t).$$

It is convenient to write the calculation in matrix form. Let $\mathbf{p}(t)$ be the vector whose components are $P(y, t)$,

$$\mathbf{p}(t) = \begin{bmatrix} P(0, t) \\ P(1, t) \\ P(2, t) \\ P(3, t) \\ P(4, t) \\ P(5, t) \\ P(5+, t) \end{bmatrix}.$$

Then $\mathbf{p}(1)$ is given as the matrix product

$$\mathbf{p}(1) = \begin{bmatrix} 0 & 0 & 0 & 0 & 0.08 & 0.28 & 0.42 \\ 0.657 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0.930 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0.930 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0.930 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0.935 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0.935 & 0 \end{bmatrix} \begin{bmatrix} P(0,0) \\ P(1,0) \\ P(2,0) \\ P(3,0) \\ P(4,0) \\ P(5,0) \\ P(5+,0) \end{bmatrix} = L\mathbf{p}(0). \tag{5.3.5}$$

Denote by $L$ the $7 \times 7$ matrix indicated. The first row reflects the births coming from various age groups and has nonzero terms indicated by them. Except for the first row, the only nonzero terms are the principal subdiagonal entries and those are the survival rates $\ell(y)$. This matrix is called the *Leslie matrix*, and it always has the same form:

$$L = \begin{pmatrix} a_1 & a_2 & a_3 & \cdots & a_n \\ b_1 & 0 & 0 & \cdots & 0 \\ 0 & b_2 & 0 & \cdots & 0 \\ \cdots & \cdots & \cdots & \cdots & 0 \\ 0 & 0 & 0 & \cdots & 0 \end{pmatrix}.$$

To be specific, assume a starting density $\mathbf{p}(0)$. The new density $\mathbf{p}(1)$ in (5.3.5) can be computed by inspection, or by using the computer:

```
  MAPLE (symbolic calculation)
> with(LinearAlgebra):
> el:=Matrix(7,7); # Maple initializes the entries to 0
  # symbolic maple calculations require rational numbers,
  # .08 = 2/25, .28 = 7/25, and so on
> el[1,5]:=2/25: el[1,6]:=7/25: el[1,7]:=21/50: el[2,1]:=657/1000: el[3,2]:=93/100:
> el[4,3]:=93/100: el[5,4]:=93/100: el[6,5]:=935/1000: el[7,6]:=935/1000:
> el;
> evalm(el &* [P0,P1,P2,P3,P4,P5,P6]);
```

Either way, we get

$$\mathbf{p}(1) = \begin{pmatrix} 0.08P(4,0) + 0.28P(5,0) + 0.42P(5+,0), \\ 0.657P(0,0) \\ 0.930P(1,0) \\ 0.930P(2,0) \\ 0.930P(3,0) \\ 0.935P(4,0) \\ 0.935P(5,0) \end{pmatrix}.$$

Furthermore, the population size after one time period is simply the sum of the components of $\mathbf{p}(1)$.

The beauty of this formulation is that advancing to the next year is just another multiplication by $L$. Thus

$$\mathbf{p}(2) = L\mathbf{p}(1) = L^2\mathbf{p}(0), \qquad \mathbf{p}(3) = L\mathbf{p}(2) = L^3\mathbf{p}(0), \qquad \text{etc.}$$

The powers of a Leslie matrix have a special property, which we illustrate. For example, compute $L^{10}$:

```
MAPLE
> el10:=evalf(evalm(el^10)):
> Digits:=2; evalf(evalm(el10)); Digits:=10;

MATLAB
> L=[0 0 0 0 .08 .28 .42; .657 0 0 0 0 0 0; 0 .930 0 0 0 0 0; 0 0 .930 0 0 0 0; 0 0 0 .930 0 0 0;...
     0 0 0 0 .935 0 0; 0 0 0 0 0 .935 0]
> L^(10)
```

The result, accurate to three places, is

$$L^{10} = \begin{pmatrix} 0.0018 & 0.018 & 0.058 & 0.094 & 0.71 & 0 & 0 \\ 0 & 0.0018 & 0.013 & 0.041 & 0.067 & 0.050 & 0 \\ 0 & 0 & 0.0018 & 0.013 & 0.041 & 0.066 & 0.050 \\ 0.11 & 0 & 0 & 0.0018 & 0.013 & 0.031 & 0.033 \\ 0.073 & 0.16 & 0 & 0 & 0.0018 & 0.0063 & 0.0094 \\ 0.021 & 0.10 & 0.16 & 0 & 0 & 0 & 0 \\ 0 & 0.030 & 0.10 & 0.16 & 0 & 0 & 0 \end{pmatrix}. \tag{5.3.6}$$

Remarkably, the power $L^n$ can be easily approximated, as predicted by the Perron–Frobenius theorem [3], as we now describe. Letting $\lambda$ be the largest eigenvalue of $L$ (see Section 2.6) and letting $V$ be the corresponding normalized eigenvector, so $LV = \lambda V$, then

$$L^n p(0) \approx c\lambda^n \mathbf{V},$$

where $c$ is a constant determined by the choice of normalization; see (5.3.7). This approximation improves with increasing $n$. The importance of this result is that the long-range forecast for the population is predictable in form. That is, the ratios between the age classes are independent of the initial distribution and scale as powers of $\lambda$.

The number $\lambda$ is a real, positive eigenvalue of $L$. It can be found rather easily by a computer algebra system. The eigenvector can also be found numerically. It is shown in [4] that the eigenvector has the following simple form:

$$\mathbf{V} = \begin{pmatrix} 1 \\ \frac{b_1}{\lambda} \\ \frac{b_1 b_2}{\lambda^2} \\ \vdots \\ \frac{b_1 b_2 b_3 \cdots b_n}{\lambda^n} \end{pmatrix}. \tag{5.3.7}$$

To illustrate this property of Leslie matrices, we will find $\lambda$, $\mathbf{V}$, and $L^{10}$ for the gray seal example. Other models are explored in the exercises.

```
MAPLE
> vel:=Eigenvectors(fel);
> vals:=vel[1]; lambda:=vals[1] # only one real e-value, should be the first
  # grab the first e-vector and normalize it
> vects:=(Transpose(vel[2]): V:=vects[1]; V:=[seq(V[i]/V[1],i=1..7)]:
> V:=convert(V,Vector[column]);

MATLAB
> [evect,eval]=eig(L)
```

```
> lambda=eval(1)
> pf=evect(:,1)
  % get pf=0.8586 and eigenvector=[-0.3930 -0.3007 … -0.4532],
  % multiply by a constant so leading term is 1
> pf=pf/pf(1)
```

The eigenvalue and eigenvector are given as

$$\lambda \approx 0.8586 \quad \text{and} \quad \mathbf{V} \approx \begin{pmatrix} 1.0 \\ 0.765 \\ 0.829 \\ 0.898 \\ 0.972 \\ 1.06 \\ 1.15 \end{pmatrix}, \tag{5.3.8}$$

which is normalized to have first component equal to 1. The alternative formula (5.3.7) for computing $\mathbf{V}$ can be used to check this result:

MAPLE
```
> chk:=[1,el[2,1]/lambda, el[2,1]*el[3,2]/lambda^2, el[2,1]*el[3,2]*el[4,3]/lambda^3,
        el[2,1]*el[3,2]*el[4,3]*el[5,4]/lambda^4, el[2,1]*el[3,2]*el[4,3]*el[5,4]*el[6,5]/lambda^5,
        el[2,1]*el[3,2]*el[4,3]*el[5,4]*el[6,5]*el[7,6]/lambda^6];
```

MATLAB
```
> V=[1; L(2,1)/lambda; L(2,1)*L(3,2)/lambda^2; L(2,1)*L(3,2)*L(4,3)/lambda^3;...
     L(2,1)*L(3,2)*L(4,3)*L(5,4)/lambda^4; L(2,1)*L(3,2)*L(4,3)*L(5,4)*L(6,5)/lambda^5;...
     L(2,1)*L(3,2)*L(4,3)*L(5,4)*L(6,5)*L(7,6)/lambda^6]
```

Evidently, we get the same vector $\mathbf{V}$ as (5.3.8). Next, we illustrate the approximation of the iterates for this example. Take the intial value to be uniform, say, 1; then make the following calculations:

MAPLE
```
> evalf(evalm(el10 &* [1,1,1,1,1,1,1]));
> evalm(lambda^10*V);
```

MATLAB
```
> p=ones(7,1) % column vector of 1s
> (L^10)*p
> lambda^10*V
```

$$p(0) = \begin{pmatrix} 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \\ 1 \end{pmatrix}, \qquad L^{10}p(0) = \alpha \begin{pmatrix} .24 \\ .17 \\ .17 \\ .19 \\ .25 \\ .28 \\ .29 \end{pmatrix} \approx c\lambda^{10}\mathbf{V} = c \begin{pmatrix} .22 \\ .17 \\ .18 \\ .19 \\ .21 \\ .23 \\ .25 \end{pmatrix}.$$

One implication of this structure is that the total population is stable if $\lambda = 1$, and it increases or decreases depending on the comparative size of $\lambda$ to 1.

*Continuous population densities provide exact population calculations.*

Any table of population densities, such as $P(y, t)$ for $n = 0, 1, \ldots$ as above, will have

limited resolution, in this case one-year brackets. Alternatively, an age distribution can be described with unlimited resolution by a continuous age-density function, which we also denote by $P(y,t)$, such as we have shown in Figures 4.1.2 and 4.1.4.

Given a continuous age density $P(y,t)$, to find the population size in any age group, just integrate. For instance, the number in the group 17.6 to 21.25 is

$$\text{number between age 17.6 and 21.25} = \int_{17.6}^{21.25} P(y,t)dy.$$

This is the area under the density curve between $y = 17.6$ and $y = 21.25$. The total population at time $t$ is

$$P = \int_0^\infty P(y,t)dy,$$

which is the analogue of (5.3.1). For a narrow range of ages at age $y$, for example, $y$ to $y+\Delta y$ with $\Delta y$ small, there is a simpler formula: Population size is approximately given by the product

$$P(y,t)\cdot\Delta y$$

because density is approximately constant over a narrow age bracket.

The variable $y$ in an age density function is a continuous variable. The period of time an individual is exactly 20, for instance, is infinitesimal; so what does $P(20,t)$ mean? In general, $P(y,t)$ is the limit as $\Delta y \to 0$ of the number of individuals in an age bracket of size $\Delta y$ that includes $y$, divided by $\Delta y$,

$$P(y,t) = \lim_{\Delta y\to 0} \frac{\text{population size between } y \text{ and } y+\Delta y}{\Delta y}.$$

As above, the density is generally a function of time as well as age, and it is written $P(y,t)$ to reflect this dependence.

Table 2.7.3 gives the mortality rate for Alabama in 1990. From the table, the death rate for 70-year-olds, i.e., someone between 70.0 and 70.999..., is approximately 40 per 1000 individuals over the course of the year. Over one-half of the year it is approximately 20 per 1000, and over $\Delta t$ fraction of the year the death rate is approximately $\mu(70,1990)\cdot\Delta t$ in deaths per 1000, where $\mu(70,1990)$ is 40. To calculate the actual number of deaths, we must multiply by the population size of the 70-year-olds in thousands. On January 1, 1990, the number of such individuals was $\int_{70}^{71} P(y,1990)dy/1000$. Thus the number of deaths among 70-year-olds over a small fraction $\Delta t$ of time at the beginning of the year 1990 is given by

$$\mu(70,1990)\Delta t \int_{70}^{71} P(y,1990)dy/1000. \tag{5.3.9}$$

A calculation such as (5.3.9) works, provided the death rate is constant over the year and the time interval $\Delta t$ is less than one year. But in general, death rates vary continuously with age. In Figure 5.3.1, we show an exponential fit to the data of Table 2.7.3. The approximate continuously varying death rate is

$$\mu(y,t) = Ae^{by},$$

which is drawn using the methods of Exercise 1 in Section 2.7. This equation assumes that the death rate is independent of time; but as we have seen, it can depend on time as well as age.

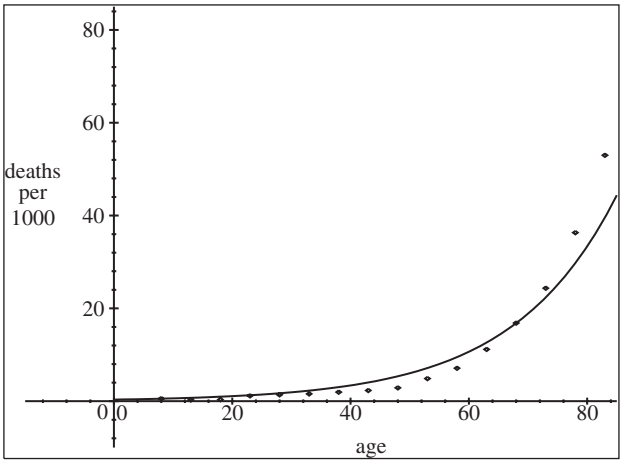


**Fig. 5.3.1.** Least squares fit to the death rate table, Table 2.7.3.

To calculate a number of deaths accurately, we must account for the changing death rate as well as the changing density. The term that calculates the number of deaths to individuals of exact age $y$ at time $t$ over the interval of time $\Delta t$ is

$$P(y,t)\mu(y,t)\Delta t. \tag{5.3.10}$$

The number of deaths among those individuals who are between $y$ and $y + \Delta y$ years old over this same period of time is

$$[P(y,t)\Delta y]\mu(y,t)\Delta t.$$

Suppose we want to do the calculation for those between the ages of $a_1$ to $a_2$ over the calendar time $t_t$ to $t_2$. The approximate answer is given by the double sum of such terms,

$$\sum\sum \mu(y,t)P(y,t)\Delta y\Delta t,$$

over a grid of small rectangles $\Delta y \Delta t$ covering the range of ages and times desired. In the limit as the grid becomes finer, this double sum converges to the double integral

$$\int_{t_1}^{t_2}\int_{a_1}^{a_2} \mu(y,t)P(y,t)dydt. \tag{5.3.11}$$

Return to (5.3.10), which calculates the loss of population, $\Delta P$, in the exact age group $y$ over the time interval $\Delta t$,

$$\Delta P = -\mu(y,t)P(y,t)\Delta t.$$

But by definition, the change in population is

$$\Delta P = P(y+\Delta y, t+\Delta t) - P(y,t).$$

Equate these two expressions for $\Delta P$, incorporating the fact that as time passes, the population ages at the same rate, that is, $\Delta y = \Delta t$. Therefore, we have the continuous analogue of (5.3.3),

$$P(y+\Delta t, t+\Delta t) - P(y,t) = -\mu(y,t)P(y,t)\Delta t.$$

Subtract the term $P(y, t+\Delta t)$ from both sides, transpose $P(y,t)$, and divide by $\Delta t$:

$$\frac{P(y+\Delta t, t+\Delta t) - P(y, t+\Delta t)}{\Delta t} + \frac{P(y, t+\Delta t) - P(y,t)}{\Delta t} = -\mu(y,t)P.$$

Finally, take the limit as $\Delta t \to 0$ to get

$$\frac{\partial P}{\partial y} + \frac{\partial P}{\partial t} = -\mu(y,t)P. \tag{5.3.12}$$

This is referred to as the *Von Foerster equation*. Its solution for $y > t$ is

$$P(y,t) = P(y-t,0)e^{-\int_0^t \mu(y-t+u,u)du},$$

as can be verified by direct substitution.

MAPLE (symbolic, no MATLAB)

```
> P:=(n,t)->h(n-t)*exp(-int(mu(n-t+u,u),u=0..t));
> diff(P(n,t),t)+diff(P(n,t),n)+mu(n,t)*P(n,t);
> simplify(%);
```

This solution does not incorporate new births, however. Just as in the discrete case, we must use experimental data to determine $P(0,t)$ as a function of $P(y,t)$, $y > 0$.

**Exercises/Experiments**

**1.** Consider the following discrete population model based on (5.3.1). Suppose the initial population distribution (year $t = 0$) is given by

$$P(n,0) = (100-n)\cdot(25+n), \quad n = 0, \ldots, 100.$$

Take the birth rate to be 1.9 children per couple per 10 years in the ten-year age bracket from 21 to 30 years of age. Thus over the year $t$, the number of births (number of people aged 0 in year $t+1$) is

$$P(0,t+1) = \frac{1.9}{2}\sum_{i=21}^{30}\frac{P(i,t)}{10}.$$

(Assume that this formulation accounts for the complication of a $\frac{3}{4}$-year gestation period.) Take the death rate for people of age $n$ to be given by the exponential

$$\mu(n) = 0.0524(\exp(0.03n) - 1), \quad n > 0.$$

The problem is to advance the population for three years, keeping track of the total population:

$$\text{Total}(t) = \sum_{n=0}^{100} P(n, t).$$

Does the total population increase? (One can use the Leslie matrix approach or (5.3.3) directly.)

MAPLE

```
> restart;
> for n from 0 to 100 do
    P[n,0]:=(100-n)*(25+n); mu[n]:=.0524*(exp(.03*n)-1);
    od:
> plot([seq([i,P[i,0]],i=0..100)]);
> plot([seq([i,mu[i]],i=0..100)]);
> for t from 1 to 3 do
    P[0,t]:=(1.9/20)*sum(P[i,t-1],i=21..30);
    for k from 1 to 100 do
      P[k,t]:=(1-mu[k-1])*P[k-1,t-1];
    od: od:
> for t from 0 to 3 do
    total[t]:=sum(P[i,t],i=0..100);
> od;
```

MATLAB

```
> n=0:1:100;
> P0=(100-n).*(25+n);
> plot(n,P0);
> P=P0'; % rows=age, columns=time
  % no base 0 indexing so P(n,t) = number aged n-1 in year t-1
> mu=.0524*(exp(.03.*n)-1); % mu(n) applies to age n-1
> plot(n,mu);
> for t=1:3
    total(t)=sum(P(:,t));
    P(1,t+1)=(1.9/20)*sum(P(22:31,t));
    for n=2:101
      P(n,t+1)=(1-mu(n-1))*P(n-1,t);
    end; end;
> total(1) %starting year
> total(4)=sum(P(:,4)) % 3 years later
```

**2.** For the following two Leslie matrices find $\lambda$ and $\mathbf{V}$ as given in (5.3.4). What is the ratio of the ages of associated populations?

$$L_1 = \begin{pmatrix} 1 & \frac{2}{3} \\ \frac{1}{2} & 0 \end{pmatrix}, \qquad L_2 = \begin{pmatrix} 0 & 4 & 3 \\ \frac{1}{2} & 0 & 0 \\ 0 & \frac{1}{4} & 0 \end{pmatrix}.$$

**Questions for Thought and Discussion**

**1.** Draw age-structure diagrams for the three cases of populations whose maximum numbers are young, middle-aged, and elderly people. In each case, draw the age-structure diagram to be expected 30 years later if birth and death rates are equal and constant and if there is no migration.

2. Repeat Question 1 for the situation in which the birth rate is larger than the death rate and there is no migration.
3. Repeat Question 1 for the situation in which the birth and death rates are constant, but there is a short but extensive incoming migration of middle-aged women at the outset.

## References and Suggested Further Reading

[1] Aging:
T. B. L. Kirkwood, The nature and causes of ageing, in D. Evered and J. Whelan, eds., *Research and the Ageing Population*, Ciba Foundation Symposium, Vol. 134, Wiley, Chichester, UK, 1988, 193–202.

[2] Aging in humans:
R. L. Rusting, Why do we age?, *Sci. Amer.*, **267**-6 (1992), 130–141.

[3] Perron–Frobenius theorem:
E. Senata, *Non-Negative Matrices and Markov Chains*, Springer-Verlag, New York, 1973.

[4] Leslie matrices:
H. Anton and C. Rorres, *Elementary Linear Algebra*, Wiley, New York, 1973, 653.